OPHTHALMOLOGY
CONTENTS

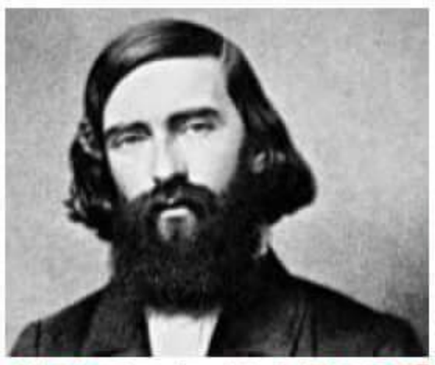
BASICS OF OPHTHALMOLOGY

→ Father of Modern medicine → WILLIAM OSLER

→ Father of Ophthalmology → ALBRECHT VON GRAEFE

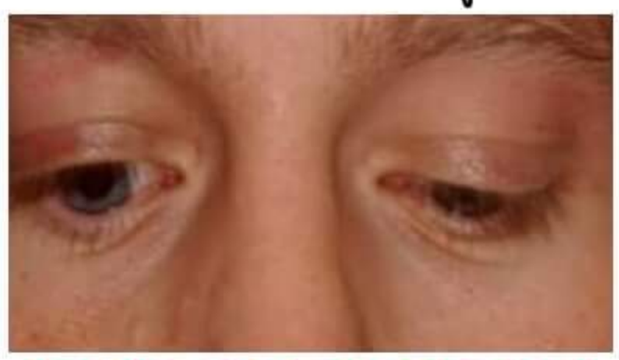
VON GRAEFE'S SIGN → Lid lag in hyperthyroidism

PSEUDO VON GRAEFE SIGN → Seen in abberant 3rd nerve regenerat^{n}



ALBRECHT VON GRAEFE

VON GRAEFE'S SIGN



PSEUDO VON GRAEFE SIGN

ANATOMY

ORBIT

→ Volume of each orbit → 30 ml

→ Volume of eyeball / globe → 6 ml

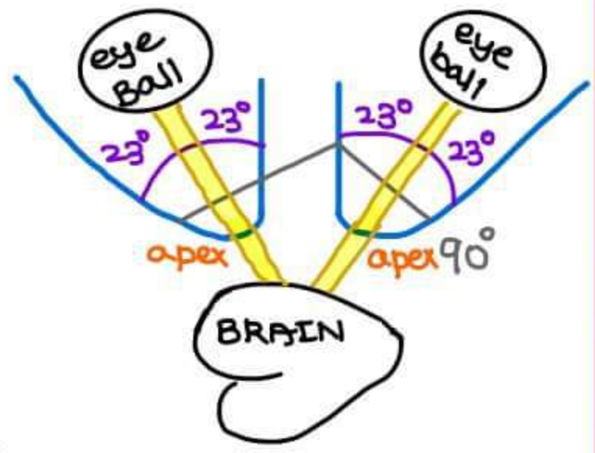
→ globe suspended by 7 attachments [6 muscles & 1 nerve] in orbit

→ Angle b/w lateral walls → 90°

medial walls are parallel to each other

Angle b/w medial & lateral walls → 45°

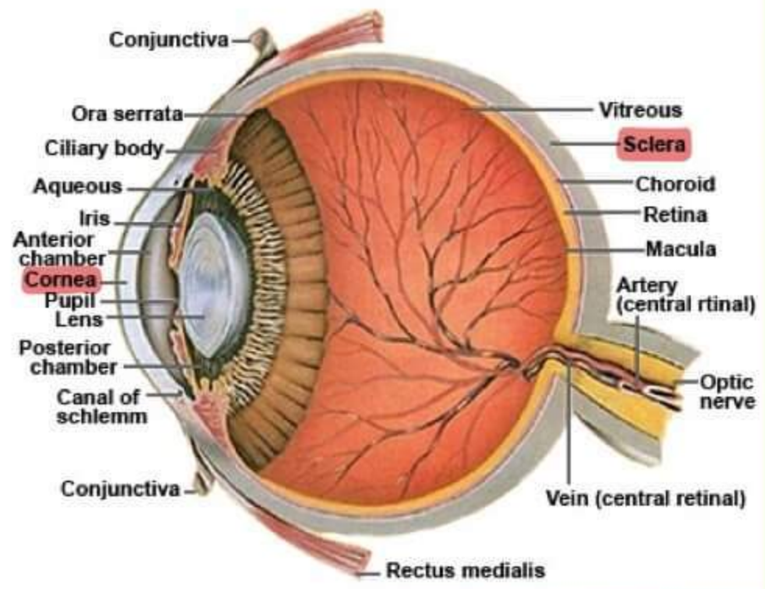
→ Optic nerve bifurcates this angle of 23° of each & attaches to brain by passing through apex on one side & attaches to eye ball on another side



EYE BALL

→ OUTER MOST LAYER consists of

- anterior 1/6 th → CORNEA
  clear & transparent
- posterior 5/6 th → SCLERA
  Opaque & white
- Junction b/w two → LIMBUS



CORNEA

KERATOMETRY

→ measures the cornea curvature

→ Indications → Dx of Keratoconus

For prescribing contact lenses

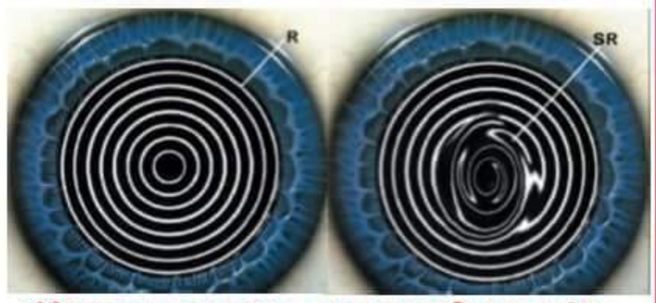
KERATOCONUS

→ Instruments used

Placido's disc

- on normal cornea, there is no distorth of reflected pattern
- on Keratoconus, distortion of reflected pattern is present

PLACIDO'S DISC



NORMAL CORNEA KERATO CONUS

LIMBAL INJURY

→ limbal injury leads to loss of transparency of cornea

→ mc form of injury → chemical injuries

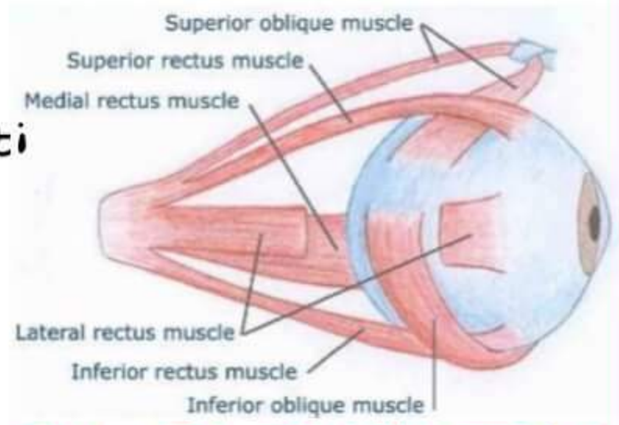
→ limbal injuries can be corrected by limbal cell transplant from other eye.

LIMBAL STEM CELL DEFICIENCY

SAME EYE AFTER LIMBAL TRANSPLANT

SCLERA

→ Thinnest & weakest part → posterior to insertⁿ of recti



Insertion of ocular muscles

MIDDLE LAYER OF EYE BALL → UVEA [≅ Grape]

UVEA

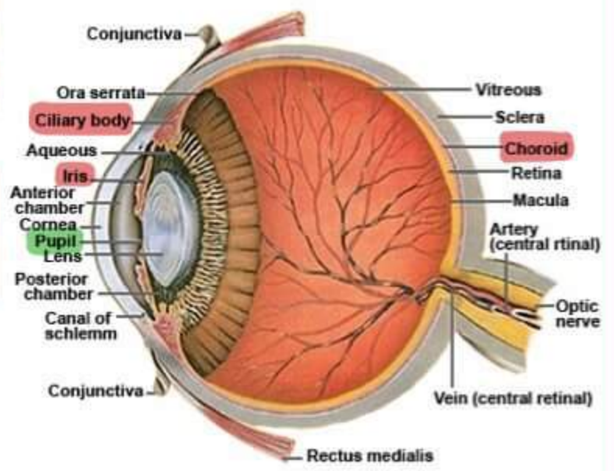
→ consists of

① IRIS

② CILIARY BODY

③ CHOROID

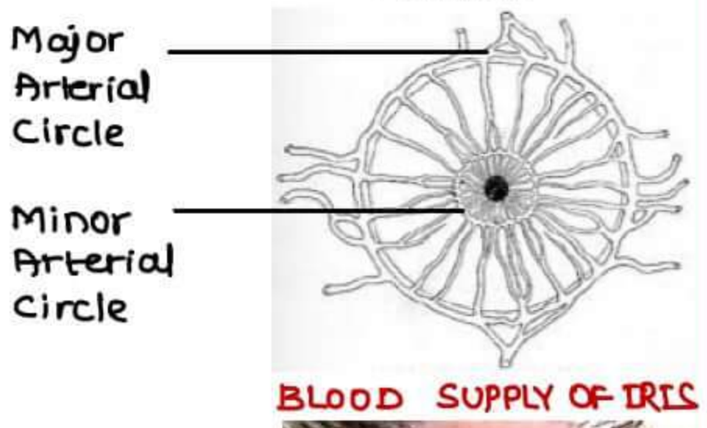
COLLARETTE



→ COLLARETTE

→ Pupil surrounded by collar like structure

→ minor arterial circle lies here



BLOOD SUPPLY OF IRIS

→ Major arterial circle lies on Root of Iris

→ Injury to iris may lead to hyphema

HYPHEMIA ↳

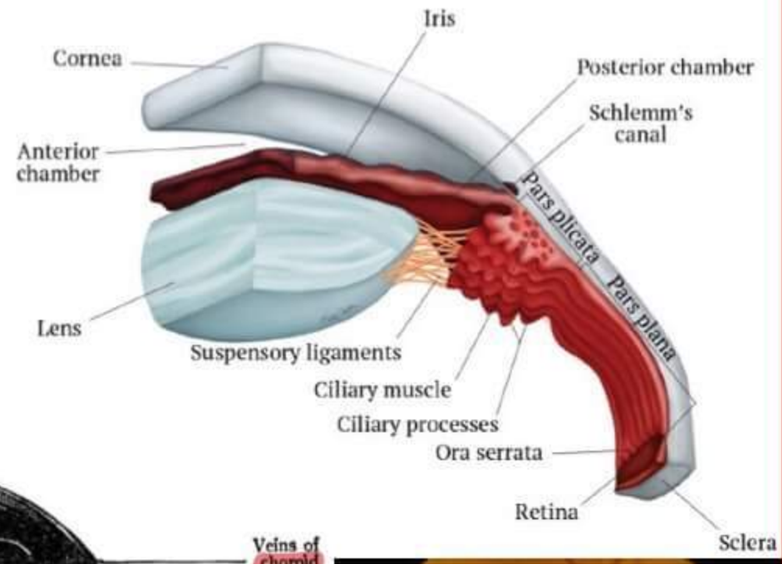
→ Main source of bleeding in case of hyphemia is → Major arterial circle

CILIARY BODY

→ PARS PLICATA → Anterior half ⅔ folds

PARS PLANA → Posterior half ⅓ out folds

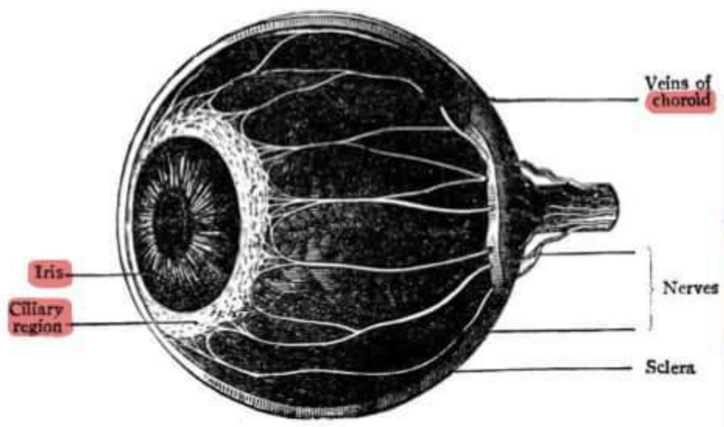
CILIARY MUSCLE → enclosed by pars plicata & pars plana

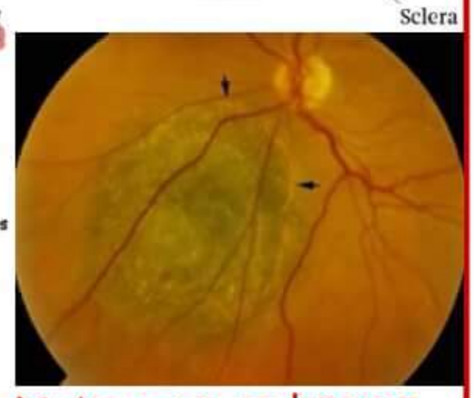


→ Functions

① Aqueous humor production

② Provide Accommodation





Malignant melanoma

CHOROID

→ Looks like chorion

→ most vascular part of eyeball

→ includes 85% of blood supply to eye ball

→ VORTEX VEINS – drains uvea [majority comes from choroid]

MALIGNANT MELANOMA OF CHOROID → vortex vein invasion seen

– distinguished from benign conditⁿ by orange pigmentatⁿ

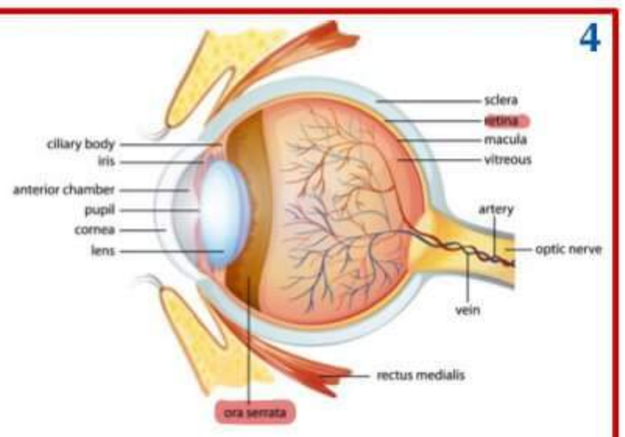
– mc malignant tumor of eye in adults

– mc malignant tumor of eye in Children → RETINOBLASTOMA

INNER MOST LAYER OF EYE BALL

RETINA

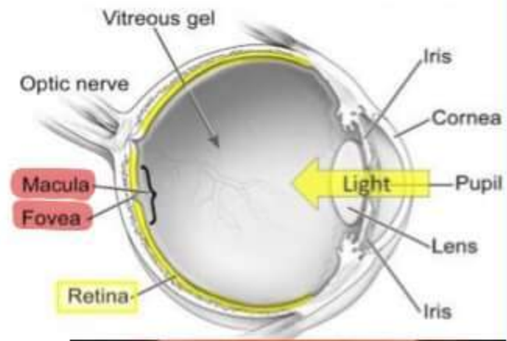
→ nervous layer of eye



PERIPHERAL RETINA / ORA SERRATA

→ attached at the junction of Ciliary body & Choroid

→ PILOCARPINE can cause Retinal Detachment

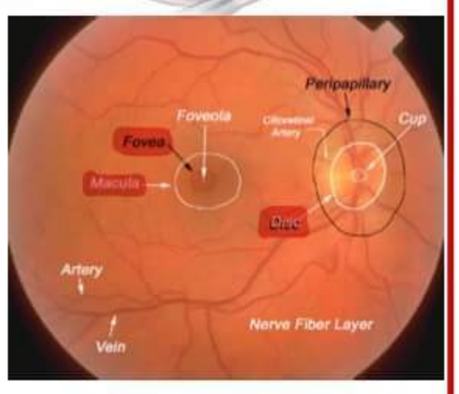


CENTRAL RETINA

→ centre of retina occupied by → MACULA

→ centre of Macula occupied by → FOVEA
most sensitive structure to light

→ Optic disc
- → present close to fovea
- → begining of optic nerve
- → diameter → 1.5 mm [fovea diameter → 1.5mm]
- → distance between optic disc & fovea → 3-4 mm



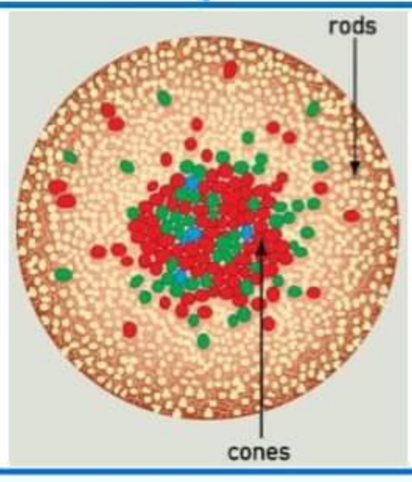
MICROSCOPIC STRUCTURE OF RETINA

① NEURONS → 5 types
② NEUROGLIA [supporting cells] → 3 types
} 8 types

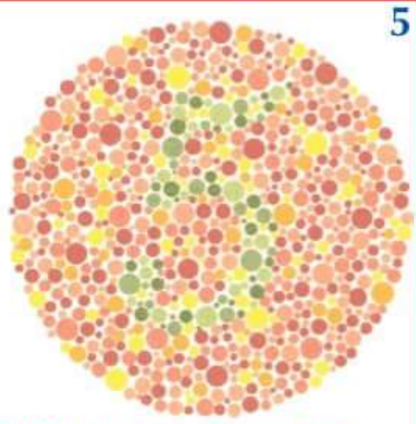
NEURON TYPES - 5

① PHOTO RECEPTORS [RODS / CONES] [1st order neuron]
② GANGLION CELLS [3rd order neuron]
③ BIPOLAR CELLS [2nd Order neuron]
④ AMACRINE CELLS
⑤ HORIZONTAL CELLS

RODS	CONES
→ Peripherally located	→ centrally located
→ 120 millions in number	→ 6 millions in number
→ 1 type	→ Red, Green, Blue types of cones ⊕
→ Responsible for – Peripheral vision – Black & White vision – NIGHT VISION	→ Responsible for – Central vision – Color vision – Day light vision



COLOUR BLINDNESS [DALTONISM]


ISHIHARA CHART

→ colour vision is tested by ISHIHARA CHART

→
- ① TRICHROMATS → all 3 cone types ⊕
- ② DICHROMATS → 2 cone types ⊕
  - → Red colourblind → Red cones ⊖nt
  - → Green colourblind → Green cones ⊖nt
  - → Blue colourblind → Blue cones ⊖nt
- ③ MONOCHROMATS → 1 cone type ⊕

→ mc colourblindness → Red Green colourblindness [90%]

ANTERIOR CHAMBER [AC]

→ depth
- → measured from centre of cornea to centre of pupil
- → 3 mm
- → SHALLOW AC → Women Elderly children [WEC]
- → Deep AC → young males

→ volume → 0.25 to 0.3 mL

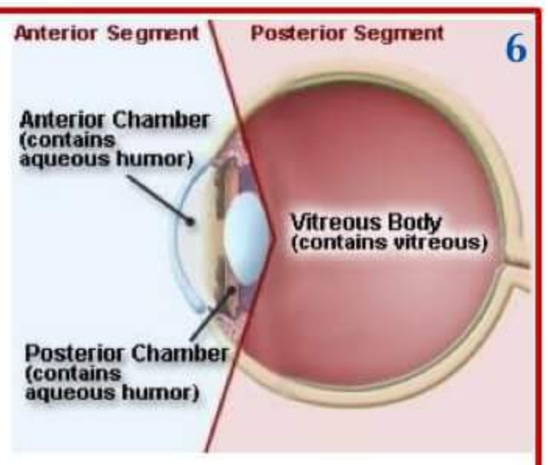
→ Filled i AQUEOUS HUMOR
- → provides nutrition
  - Avascular structures [Cornea, Lens] completely rely on it

→ CORNEA NUTRITION SUPPLY
- @ Glucose → completely supplied by aqueous humor
- ⓑ O_2 supplied by
  - (i) Air
  - (ii) Tear film
  - (iii) Aqueous humor

Contact lens blocks O_2 supply mainly from tear film
- → Leads to corneal ulcers
  - mainly d/t Pseudomonas [emergency condition]
  - avoid wearing contact lens while sleeping

→ LENS NUTRITION SUPPLY
- → Glucose & O_2 entirely supplied by aqueous humor
- → In case of Aqueous humor shut down d/t Ciliary body injury Lens can survive
  - → Reason → glucose metabolism of Lens is mainly anaerobically [85%]
    - 5% metabolised by HMP pathway
    - 5% metabolised by Kreb cycle
    - 5% metabolised by Sorbitol pathway

CRYSTALLINE LENS



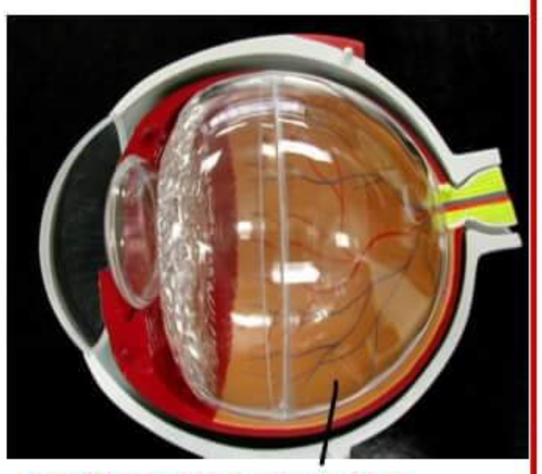
- → Lies in patellar fossa
- → composition
  - 65% → Water
  - 35% → proteins [90% → crystalline proteins]
- → contains
  1. Nucleus
  2. cortex
  3. capsule
     - → thinnest part of capsule → Posterior capsule
     - → LEC's [lens Epithelial cells] → only present under anterior capsule
- → Lens suspended by ZONULES / SUSPENSORY LIGAMENTS from ciliary body
- → IRIS LENS DIAPHRAGM includes
  1. Iris
  2. Ciliary body
  3. zonules
  4. Lens

  } moves together


ZONULES OF THE EYE

POSTERIOR CHAMBER [PC]

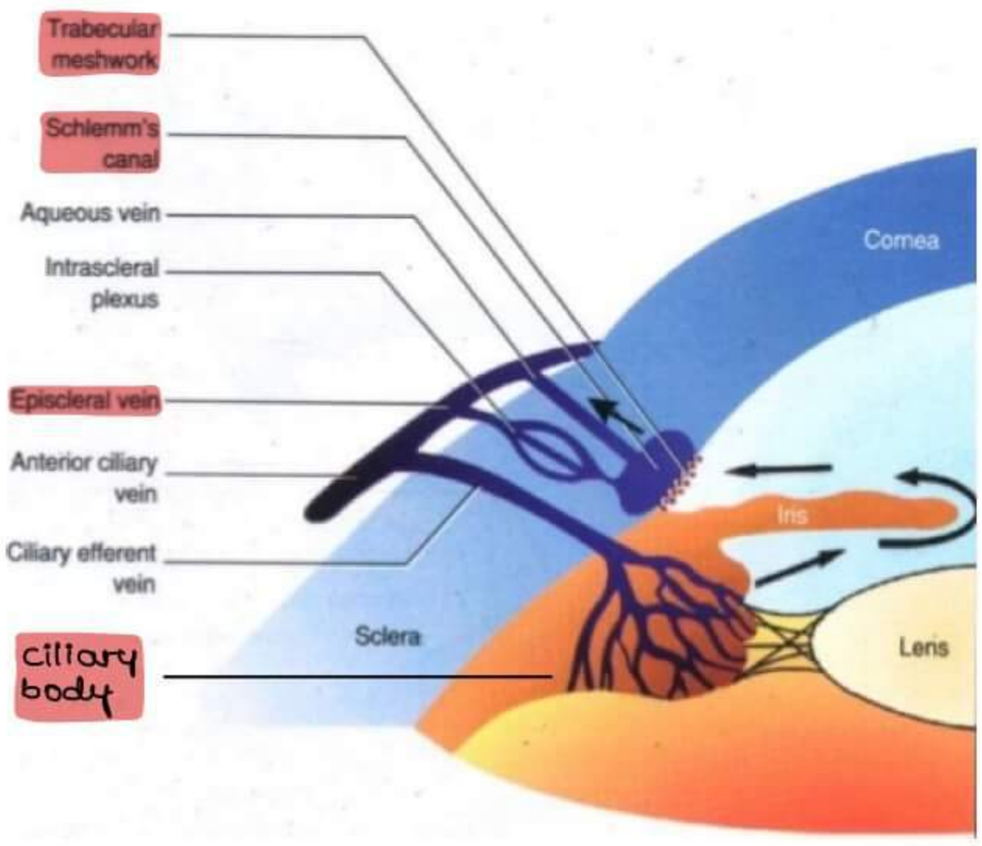
- → Space blw the iris & lens
- → contains aqueous humor
- → AC & PC connected t each other by pupil


VITREOUS HUMOR

VITREOUS CAVITY

- → Space blw posterior surface of lens & Retina
- → volume → 4 ml
- → contains VITREOUS HUMOR [4ml - 70% of eye ball]
  - → single largest structure inside the eye
- → strongest attachment → vitreous base

AQUEOUS HUMOR DYNAMICS



PARS PLICATA [CB]
↓
POSTERIOR CHAMBER
↓
PUPIL
↓
ANTERIOR CHAMBER
↓
ANGLE
↓
TRABECULAR MESHWORK
↓
SCHLEMM'S CANAL
↓
EPISCLERAL VEIN

ANGLE CLOSURE GLAUCOMA → Angle blocked
OPEN ANGLE GLAUCOMA → Trabecular meshwork [mc] or Schlemm's canal blocked

ANTERIOR SEGMENT & POSTERIOR SEGMENT

→ Lens divides eye ball into anterior segment & posterior segment

→ ANTERIOR SEGMENT → Structures anterior to lens [including lens]

POSTERIOR SEGMENT → structures posterior to lens

AQUEOUS HUMOR	VITREOUS HUMOR
→ Aqueous = H_2O	→ Vitreous = Glass
→ solution	→ Gel
→ provides nutrition	→ provides shock absorption
→ produced at 2.5 µl/min	→ produced at the time of birth
→ entry through limbus	→ entry through Pars plana only
→ composition ≅ Blood plasma ↓ few exceptions	→ composition → 98% H_2O → Hyaluronic acid → type II collagen

AQUEOUS HUMOR

→ site of production → from non pigmented epithelium of PARS PLICATA

→ MECHANISM OF PRODUCTION

① SECRETION
- → requires active energy
- → contributes to 70% of production

② ULTRA FILTRATION } contributes to 30% of production

③ DIFFUSION]

→ produced at a rate of 2.5µL/min

→ NEVER STOPS
- → Slows down while sleeping
- → ↑ses during early morning
  - → Highest IOP present at 7-8 AM

→ OUT FLOW FACILITY [C]
- → volume of aqueous flowing out of eye every minute
- → C = 0.2 µL/min/mm of Hg [2µL/min, if IOP is 10mm of Hg]
- → depends on IOP

→ BLOOD PLASMA VS AQUEOUS HUMOR

BLOOD	AQUEOUS humor
→ ↑ Glucose	→ 80% of blood Glucose
→ ↑ Protein	→ almost protein free
	→ ↑ Ascorbate
	→ ↑ Lactate

→ AQUEOUS HUMOR CIRCULATION / DYNAMICS [Refer page no. 6]

OPTICS

→ only curved surfaces can bend light
→ CURVATURE ∝ BENDING

POWER

→ Power of eye → + 60 Diopters [exactly 58.6 D]
Power of Cornea → 43 D [70%.]
Power of Lens → 19 D [30%.]

→ ⊕ indicates convergence
⊖ indicates Divergence

→ Power of Eye contributed by
① Anterior surface of cornea [maximum contribution]
② Posterior surface of cornea
③ Anterior surface of Lens
④ Posterior surface of Lens

→ AP [ANTERO-POSTERIOR] DIAMETER / AXIAL LENGTH
→ Line joining the centre of cornea to centre of retina
→ Ⓝ → 24 mm [in adults]
→ 16 mm [at birth]
→ b/w 18-21 yrs eye ball stops growing

→ Distant vision → 6/6

NEAR VISION

→ Optimal near vision → N_6
→ Near vision depends on
① ACCOMMODATION
② CONVERGENCE
③ MIOSIS

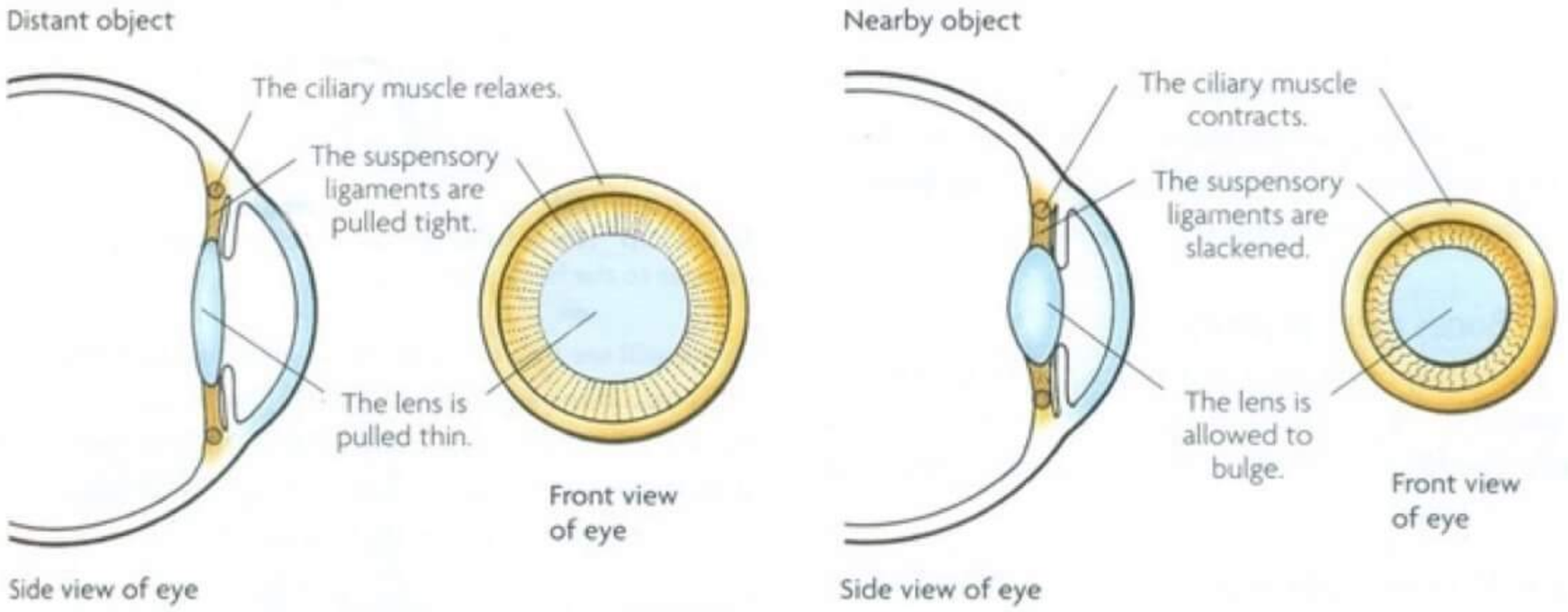
ACCOMMODATION

→ CHANGE OF SHAPE OF LENS to ↑ power of the lens to look at nearer objects

→ minimum distance for light rays to become parallel → 6 m
→ Nearer the Object, rays are more divergent
→ Distant object, rays are parallel

→ ↑ curvature of lens → ↑ power of Lens [19D + additional 16D = 35D]
→ rays of light can be focussed on retina

MECHANISM OF ACCOMMODATION



→ Lens changes shape by

CILIARY MUSCLES → ZONULES → LENS


CONVERGENCE


MIOSIS

→ **CILIARY MUSCLES ACTION** 1/α **ZONULAR ACTION**

Ciliary muscle contraction → zonular relaxation

Ciliary muscle relaxation → zonular contraction

→ **CILIARY MUSCLES ACTION CONTROLLED BY CLARITY OF VISION**

Clear vision → CM relaxatⁿ → zonlular contractⁿ → Thin lens [Distant vision]

Blurred vision → CM contractⁿ → zonular relaxatⁿ → Spherical lens [Near vision]

CCC - ciliary muscle contracts for close vision

CONVERGENCE → Two eyes comes closer together by medial recti

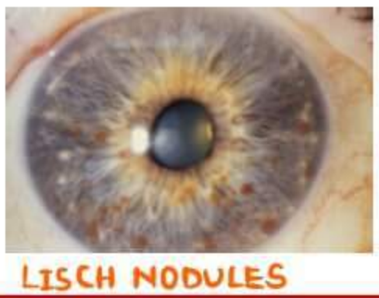
MIOSIS → constriction of pupil

TRIPLE REACTION / NEAR REACTION / ACCOMMODATION REACTION [N_6]

Accommodatⁿ, Convergence & miosis work together for near vision

MOST COMMON OCULAR MANIFESTATIONS OF SYSTEMIC DISEASE

NEUROFIBROMATOSIS TYPE 1	→	LISCH NODULES
NEUROFIBROMATOSIS TYPE 2	→	Posterior Subcapsular cataract
STURGE WEBER SYNDROME	→	Glaucoma [particularly if angioma present in upperlid]
VON HIPPEL LINDAU SYNDROME	→	Retinal Angioma
TUBEROUS SCLEROSIS	→	Retinal Phakoma
RHEUMATOID ARTHRITIS	→	Dry eyes
SLE	→	Dry eyes
WEGENER'S GRANULOMATOSIS	→	Peripheral corneal ulcers
THYROID EYE DISEASE	→	Lid retraction
BEHCET'S DISEASE	→	Anterior Uveitis
MC cause of LOSS OF vision in NF1	→	BIL OPTIC NERVE GLIOMA


LISCH NODULES


STURGE WEBER SYNDROME


RETINAL ANGIOMA


PERIPHERAL CORNEAL ULCER


LID RETRACTⁿ

ORBIT & ADNEXA

ORBIT

→ COMPOSED OF

① Superior wall

② Inferior wall → mcly fractured in blow out # [dlt poor support by maxillary sinus]

③ Medial wall → Thinnest wall [infections enters through this wall]

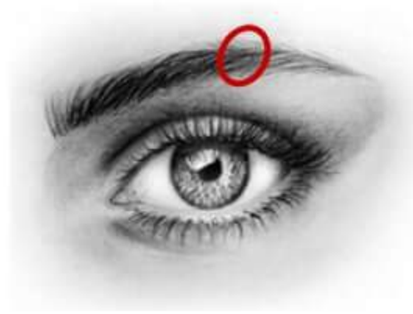
④ Lateral wall → Thickest & Strongest wall

BLOWOUT #

TRAUMATIC OPTIC NEUROPATHY

→ Optical canal lies just behind the Lateral 1/3rd & medial 2/3rd of eye brow

→ This is the commonest site of injury that causes traumatic optic neuropathy



ORBITAL CELLULITIS

→ infection of Orbit behind orbital septum

mc cause of U/L proptosis in a child	→	Orbital cellulitis
mc cause of B/L proptosis in a child	→	Neuroblastoma metastasis
mc cause of U/L proptosis in an adult	}	Thyroid Eye Disease [TED] / Thyroid related Ophthalmopathy [TRO] / Grave's Ophthalmopathy [GO]
mc cause of B/L proptosis in an adult	}	

→ mc cause of Orbital Cellulitis → Ethmoidal sinusitis

→ mc organism causing Orbital Cellulitis → Staph. aureus

→ CLINICAL FEATURES

→ proptosis
→ chemosis [conjunctival edema] } TRIAD
→ Restrictⁿ of Ocular movements

ORBITAL CELLULITIS

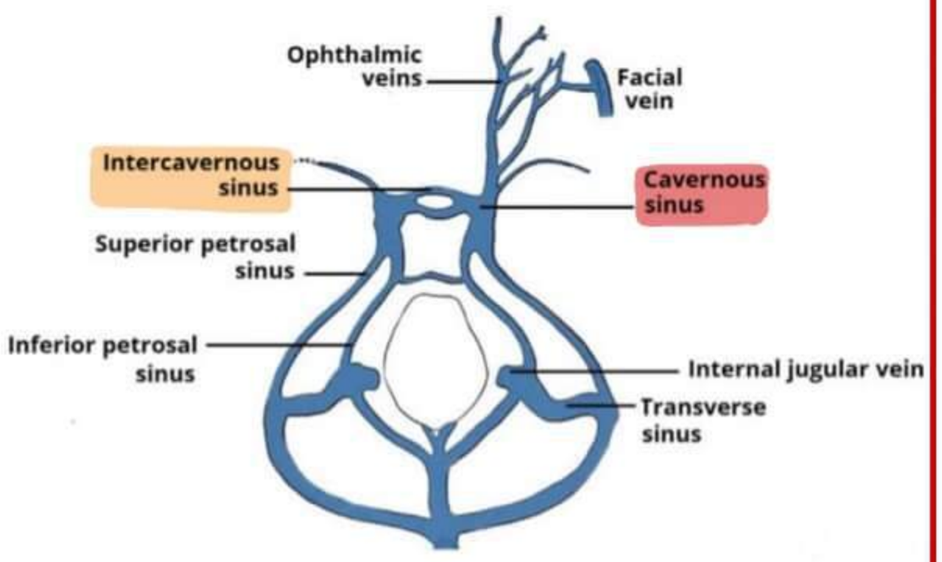
→ TREATMENT → DOC → IV VANCOMYCIN

→ most serious complication → CAVERNOUS SINUS THROMBOSIS

CAVERNOUS SINUS THROMBOSIS

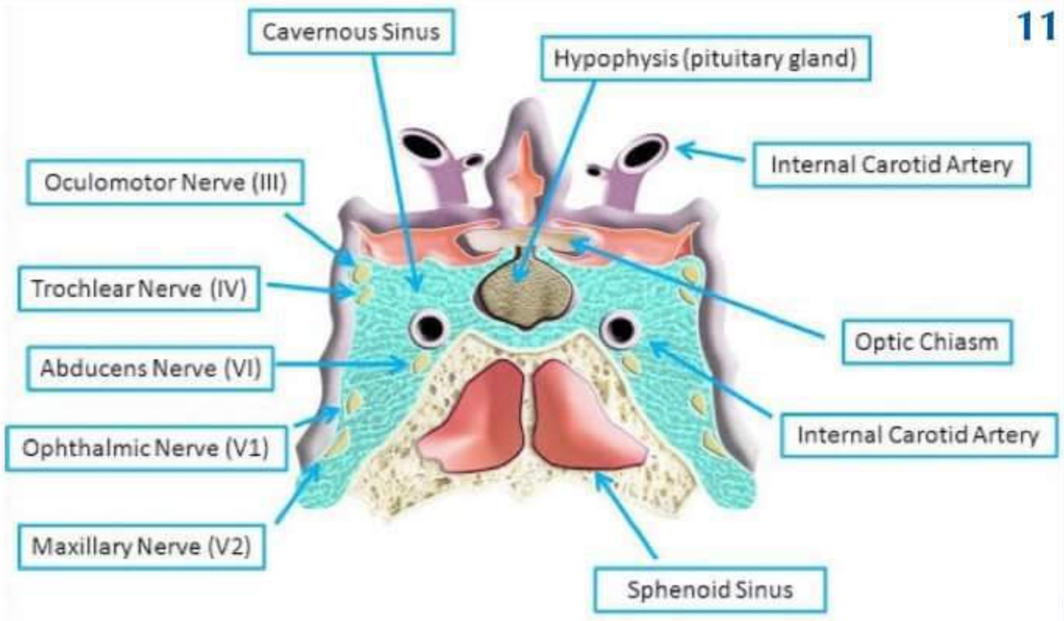
→ CAVERNOUS SINUSES

→ present in middle cranial fossa just behind the Orbit
→ connected by intercavernous sinus
→ intercavernous sinus have no valves
→ infectⁿ can transmits from each other very easily



→ **STRUCTURES OF CAVERNOUS SINUS**

- → Oculomotor nerve [III]
- → Trochlear nerve [IV]
- → Ophthalmic nerve [V_1]
- → Maxillary nerve [V_2]
- → Abducens nerve [VI]
- → Internal carotid Artery
- → Pituitary gland



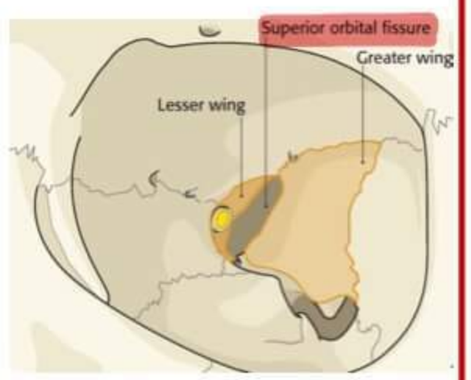
→ **CLINICAL FEATURES**

- → Proptosis , chemosis , Extra ocular muscle paralysis
- → Earliest sign → 6th nerve palsy
- → CNS SYMPTOMS → Disorientation, drowsiness , coma
- → High fever & rigors, chills


CAVERNOUS SINUS THROMBOSIS

→ **TREATMENT**

- → IV Antibiotics
- → IV Heparin
- → Steroids



DIFFERENTIAL DIAGNOSIS

① SOFS → Superior Orbital fissure Syndrome
② CSS → Cavernous sinus Syndrome
③ OAS → Orbital Apex Syndrome
→ Loss of vision dlt optic nerve involvement is distinguishes OAS from other two

ORBITAL TUMORS

→ mc primary orbital tumor in children → Dermoid cyst
— Most common location in Supero temporal area
→ mc malignant primary orbital tumor of children → Rhabdomyo Sarcoma


RHABDOMYOSARCOMA

→ mc Secondary tumors of orbit in children → Metastasis from Neuroblastoma > Ewing's sarcoma > Wilm's tumor > chloromas


RACOON SIGN IN NEUROBLASTOMA

→ mc primary orbital tumor in adults → Cavernous Hemangioma
→ mc Secondary orbital tumor in adults → Metastasis from Breast > Lungs > Prostate


ENOPHTHALMOS

Q 65yrs female ī ptosis , enophthalmos , loss of ocular movements . Dx ?
A Breast cancer

THYROID EYE DISEASE

→ aka THYROID RELATED OPHTHALMOPATHY
GRAVE'S OPHTHALMOPATHY

→ 5 times more common in female

→ Age group → 40 - 60 yrs

→ NOSPECS CLASSIFICATION

N → NO SIGNS, NO SYMPTOMS

O → ONLY SIGNS, NO SYMPTOMS
- mc presentat^{n} → Lid retraction
- 2nd mc presentat^{n} → Lid Lag


LID LAG

S → SOFT TISSUE SIGNS
- Periorbital lid edema
- caruncle edema
- injection over lateral rectus insertion


EXOPHTHALMOS

P → PROPTOSIS / EXOPHTHALMOS
- dlt deposit^{n} of mucopolysaccharides
- muscle belly is enlarged but no tendon enlargement [elicited radiologically]

E → EXTRA OCULAR MUSCLE RESTRICTION
- EOM involvement sequence
  - I → Inferior rectus
  - aM → Medial rectus
  - So → Superior rectus
  - Lucky → Lateral rectus

C → CORNEAL INVOLVEMENT
→ corneal exposure keratopathy → corneal ulcer

S → SIGHT LOSS due to optic nerve compression

ADNEXA

EYE LIDS

→ Muscles opening eye lid
① LPS [Levator Palpabrae Superioris]
② Muller's muscle [doesn't elevate >2mm]
③ Frontalis muscle

→ muscle closing eye lid
① Orbicularis oculi


ORBICULARIS IN ACTION

PTOSIS

→ Upper eye lid covers 2 mm of upper cornea } Normal
Lower eye lid at limbus

→ Drooping of upper eye lid below 2 mm → PTOSIS

CONGENITAL PTOSIS → from birth to 1 yr

ACQUIRED PTOSIS

① APONEUROTIC / INVOLUTION / SENILE PTOSIS
- → present in old people
- → aponeurosis of LPS is damaged

② NEUROGENIC PTOSIS
- → 3rd nerve palsy [3rd nerve supplies LPS]
- → Horner's syndrome

③ MYOGENIC PTOSIS → Myasthenia gravis / myotonic dystrophy

④ MECHANICAL PTOSIS → d/t inflammation or tumors


CONGENITAL PTOSIS


SENILE PTOSIS


3rd NERVE PALSY


MYOGENIC PTOSIS

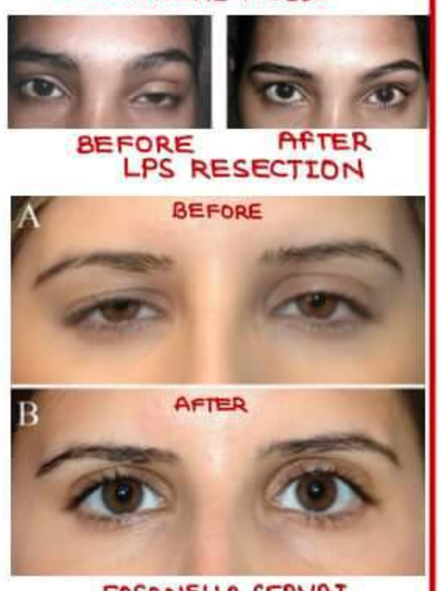

MECHANICAL PTOSIS

TREATMENT

SURGICAL TREATMENT

① LPS RESECTION [80% of ptosis is d/t LPS involvement]

② FASANELLA SERVAT SURGERY
- → 10-15% of ptosis is d/t muller's muscle involvement
- → Horner's syndrome is d/t muller's muscle involvement



BEFORE AFTER LPS RESECTION

FASANELLA SERVAT

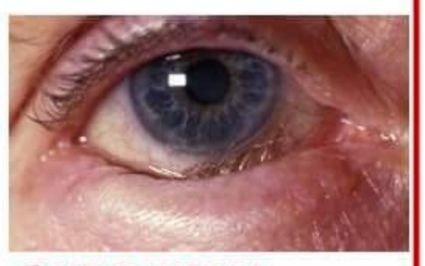
ENTROPION

→ Inward rolling of eye lids → ENTROPION
Inward rolling of eye lashes → TRICHIASIS

→ TYPES
- ① CONGENITAL
- ② CICATRICIAL
- ③ INVOLUTIONAL [mc]
- ④ SPASTIC

→ CLINICAL FEATURES
- → Foreign body sensation
- → redness of eye

→ TREATMENT
- → JONES RETRACTOR PLICATION F/b WEIS PROCEDURE


ENTROPION

ECTROPION

→ outward rolling of eye lids

→ TYPES
- ① CICATRICIAL
- ② INVOLUTIONAL [mc]
- ③ MECHANICAL
- ④ PARALYTIC

→ TREATMENT
- ① LATERAL STRIP PROCEDURE
- ② RETRACTOR REINSERTION


ECTROPION

LENS

BLINDNESS

mc cause of blindness in world	→	cataract
→ 70% of blindness in india	}	contributed by cataract
50% of blindness in World	}	
2nd mcc of blindness in the world	→	Glaucoma
3rd mcc of blindness in the world	→	ARMD
mc infectious cause of blindness	→	Trachoma
2nd mc infectious cause of blindness	→	Onchocercosis [River blindness]
mcc of blindness in children	→	Vit A deficiency
mcc of ocular morbidity	→	Refractive errors

→ According to NPCB [National Programme For control of Blindness],

$\frac{6}{6}$ – $\frac{6}{18}$	→	Normal vision
$\frac{6}{18}$ – $\frac{6}{60}$	→	Low vision
$< \frac{6}{60}$	→	Blind [changed now]
< 3/60 ī best possible correction in better eye	}	Blind [New definition]
$< \frac{6}{60}$	→	ECONOMIC BLINDNESS
$< \frac{3}{60}$	→	SOCIAL BLINDNESS
$< \frac{1}{60}$	→	MANIFEST BLINDNESS
No Light perception	→	ABSOLUTE BLINDNESS

→ According to WHO,

< 3/60 ī best possible correction in better eye	}	BLIND

CATARACT

→ Opacification of lens or capsule

CLASSIFICATION BASED ON CAUSE

① AGE RELATED CATARACT
- → previous term → Senile cataract
- → age group → 50 - 60 yrs
- → mc cause of cataract [80%]
- → most important risk factor → UV light exposure
  - − Sunglasses provide protection

② CONGENITAL / DEVELOPMENTAL CATARACT

③ TRAUMATIC CATARACT

④ COMPLICATED CATARACT

⑤ METABOLIC CATARACT

⑥ HEAT CATARACT

⑦ RADIATION CATARACT

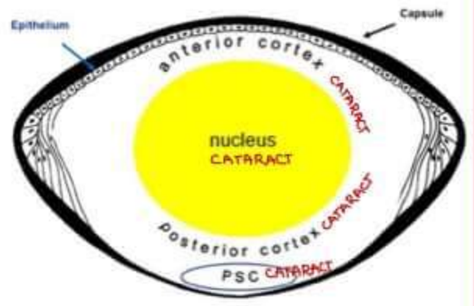
⑧ DRUG INDUCED CATARACT

CLASSIFICATION BASED ON MORPHOLOGY

① NUCLEAR CATARACT

② CORTICAL CATARACT

③ POSTERIOR SUB CAPSULAR CATARACT



NUCLEAR CATARACT

→ CLINICAL FEATURES
- → Hemeralopia [Day blindness dlt constrictn of pupil]
- → Decreased distant vision [dlt index myopia] [distant > near]
- → Second sight
  - → recovery of near vision
  - → presbyopia resolves itself as nuclear cataract ↑ses refractive index


NUCLEAR CATARACT

CORTICAL CATARACT / CUNIEFORM CATARACT

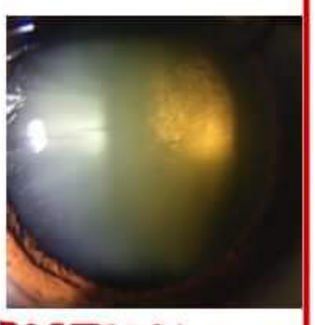
→ CLINICAL FEATURES
- → Nyctalopia [Night blindedness]
- → Cunieform cataract [Wedge shaped cataract]


CORTICAL CATARACT

POSTERIOR SUB CAPSULAR CATARACT / CUPULIFORM CATARACT

→ CLINICAL FEATURES
- → Cause maximum visual handicap
- → have maximum glare [dlt they are closest to nodal point]

NODAL POINT
- → lies close to the posterior capsule
- → light rays passing through nodal point donot bend


POSTERIOR SUB CAPSULAR CATARACT

→ more posterior the cataract, more drop in vision
→ aka CUPULIFORM CATARACT [cup shaped cataract]
→ ↓sed near vision [Both visions are affected (near > distant)]
→ dlt miosis in near vision

CONGENITAL / DEVELOPMENTAL CATARACT

→ FOVEAL FIXATION
→ critical period for development of fixatⁿ → 2-4 months
→ fovea collects 80 - 90% of rays after fixatⁿ
→ Fovea fixatⁿ occurs dlt stimulatⁿ by light rays upto 2-4 months
→ foveal fixatn do not happen here

ⓐ ZONULAR / LAMELLAR / RIDER'S CATARACT

→ mc cataract causing loss of vision in a child
→ Affects certain zone of the lens } ZONULAR / LAMELLAR
Trianglar spokes are seen } RIDER'S CATARACT

RIDER'S CATARACT


BLUE DOT CATARACT

ⓑ PUNCTATE / BLUE DOT CATARACT

→ mc congenital cataract in children
→ no loss of vision

ⓒ CORALLIFORM CATARCT

→ coral like
→ no profound loss of vision


CORALLIFORM

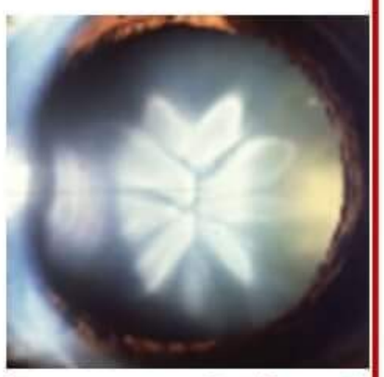
ⓓ CONGENITAL RUBELLA SYNDROME

→ TRIAD
→ Salt & pepper retinopathy [mc ophthalmic finding]
→ Nuclear cataract
→ Glaucoma


RUBELLA CATARACT

TRAUMATIC CATARACT | ROSETTE CATARACT

→ mostly by blunt trauma [concussion]
→ petal shaped [mostly 10 in number]
→ posterior cortical cataract


ROSETTE CATARACT

COMPLICATED CATARACT

→ CAUSED BY DISEASES OF EYE } Uveitis [mc], Retinitis pigmentosa
Glaucoma, High myopia

→ mostly posterior sub capsular cataract
→ BREAD CRUMB APPEARANCE
→ shows polychromatic lusture


Polychromatic lusture

METABOLIC CATARACT

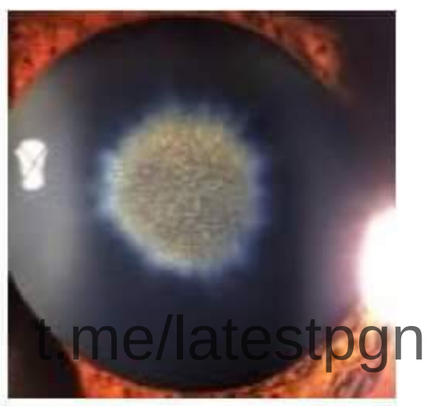
① DIABETIC MELLITUS

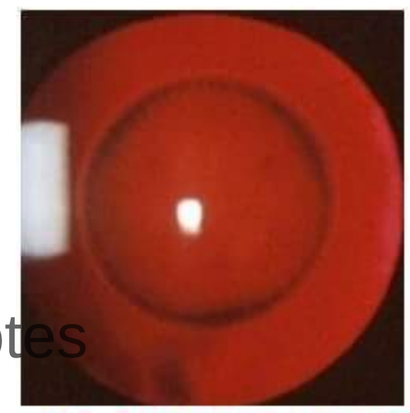
Early senile	True diabetic / Snow flake
→ early 40's	→ young
→ NIDDM / Type 2 DM	→ IDDM / Type 1 DM
→ no particular shape	→ Snow Flake / Snow Storm like
→ chronic onset	→ Only cataract & acute onset

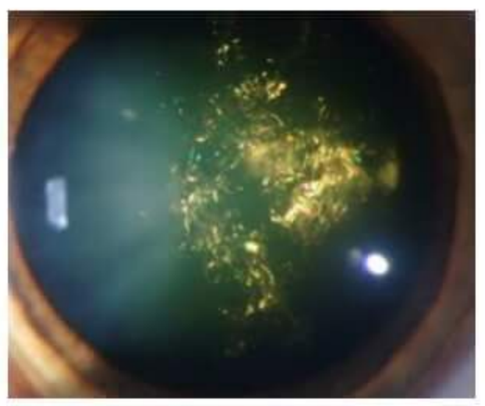
② WILSON'S DISEASE → Sun flower cataract
③ GALACTOSEMIA → Oil droplet cataract
④ MYOTONIC DYSTROPHY → Christmas tree cataract
⑤ FABRY'S DISEASE → Propeller / Spoke like cataract
⑥ ATOPIC DERMATITIS → Shield cataract
Anterior sub capsular cataract

→ Keyser – Fleisher ring → characteristic of Wilson's disease


SNOW FLAKE CATARACT


SUN FLOWER CATARACT


OIL DROPLET CATARACT


CHRISTMAS TREE CATARACT

HEAT CATARACT

→ True exfoliation cataract
→ Glass blower's cataract
→ Iron worker's cataract

POSTERIOR SUB CAPSULAR CATARACT – CAUSED BY
→ Steroids
→ Complicated cataract
→ Radiation induced cataract

RADIATION CATARACT

→ Ionising radiatⁿ causes cataract
  → mcly by x rays
→ posterior sub capsular cataract
→ most sensitive to radiation → Lens

DRUG INDUCED CATARACT

→ Systemic steroids (mc cause) → posterior subcapsular cataract
→ Chlorpromazine → causes stellate cataract
→ Chloroquine
→ Amiodarone
→ Busulphan
→ Penicillamine
→ Gold
→ Ecothiophate [strong miotic]


Chlorpromazine Cataract

SENILE CATARACT

STAGES OF SENILE CATARACT

① INCIPIENT
② IMMATURE
③ MATURE
④ HYPERMATURE

INCIPIENT CATARACT

→ early cataract
→ CLINICAL FEATURES
  → Loss of contrast sensitivity
  → Diplopia [mild]
  → Loss of vision [mild]

IMMATURE CATARACT

→ partially opaque
→ yellowish grey
→ iris shadow seen


IMMATURE CATARACT

MATURE CATARACT

→ totally opaque
→ White
→ often intumescent [swell i water]
→ can cause glaucoma


MATURE CATARACT

HYPER MATURE CATARACT

→ Lens shrinks
→ capsule crumples
→ Phacodonesis [rocking movement of lens] +nt
→ Subluxation / dislocation occurs
→ TYPES


HYPERMATURE CATARACT

MORGAGNIAN CATARACT	SCLEROTIC CATARACT
→ cortical cataract → hypermature → cortex liquifies Nucleus falls into it  MORGAGNIAN CATARACT	→ nuclear cataract → hypermature → CATARACTA BRUNESCENT → brown in colour → and hardest cataract → CATARACTA NIGRA → black in color → hardest cataract  C. brunescent  C. nigra

SYMPTOMS OF CATARACT

- → Slow painless loss of vision
- → DIPLOPIA / POLYOPIA
- → COLOURED HALOS [always abnormal]

SLIT LAMP EXAMINATION

- → can be examined from eye lids & eye lashes to anterior 1/3rd of vitreous
- → aids in the diagnosis of cataract

SLOW PAINLESS LOSS OF VISION

caused by

- → Cataract
- → Refractive errors
- → Open angle glaucoma
- → ARMD
- → Diabetic retinopathy

TREATMENT

① GLASSES

② SURGERY

→ Indicatⁿ depends on visual Handicap

— If there is no problem ĩ daily activities, surgery not indicated

Q A 30 yr male ĩ cataract, better management option ?

A. Glasses

B. Surgery

→ ĩ surgical mx, accommodatⁿ is lost → near vision compromised

SURGERY

① ICCE

② ECCE ĩ IOL

③ PHACOEMULSIFICATION

④ SICS

ICCE [INTRA CAPSULAR CATARACT EXTRACTION]

- → Lens + capsule removed
- → Leads to aphakia
  - → high powered convex lens prescribed
    - → For each 1D, convex lens ↑ses magnificatⁿ by 2%.
    - → For each 1D, concave lens minimises by 2%.
    - → leads to DIPLOPIA
- → only indication → Subluxated / dislocated cataract
- → APHAKIA

CLINICAL FEATURES

- → Deep anterior chamber
- → Jet black pupil
- → Iridodonesis
- → Diplopia
- → JACK IN BOX SCOTOMA
- → Loss of visual field
- → Pin cushion defect

ICCE

SUBLUXATED CATARACT

JET BLACK PUPIL

ECCE [ī PCIOL]

→ EXTRA CAPSULAR CATARACT EXTRACTION ī POSTERIOR CHAMBER IOC

→ Lens removed, capsule left in situ

→ Anterior capsulotomy done

→ cataract lens removed

→ Artificial Intra ocular lens placed in posterior chamber

→ 6/18 vision restored [compared to finger counting at 1 mtr in ICCE]

→ Leads to PSEUDOPHAKIA

→ IOL

① PMMA [Poly Methyl Meth Acrylate] IOL

→ can transmits about 4 times the normal light

→ monofocal IOL

→ mcly used

→ Patient has to wear glasses for near vision

UNIFOCAL IOL

MULTIFOCAL IOL

② MULTI FOCAL IOL

→ Expensive

→ have more side effects

→ used in selective patients who don't want to wear glasses

TORIC IOL

③ TORIC IOL

→ indicated for patients with astigmatism

→ implanted according to corneal markings

corneal marking

④ ACCOMADATIVE IOL

→ moves back & forth & provides pseudo-accommodation

Accommodative IOL

PHACO EMULSIFICATION

→ stitchless

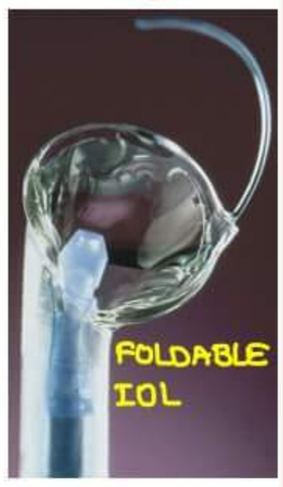
→ INCISION

→ less than 3 mm in size

→ valvular/ multiplanar incision

→ self - sealing

→ Scleral / corneal tunnel incision



→ Lens emulsification by ultrasonic Frequency [40,000/s]

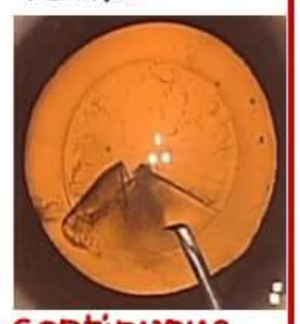
→ FOLDABLE IOL → Silicone / Acrylic IOL is used

→ less time consuming ; 6/6 vision restored

→ costly

→ STEPS OF PHACO EMULSIFICATION

① 3mm INCISION
② ANTERIOR CAPSULOTOMY → continuous curvilinear capsulorhexis
③ HYDRO DISSECTION → to seperate cortex from capsule
④ HYDRO DELINEATION → to seperati epi & endonucleus
⑤ PHACO EMULSIFICATION
⑥ IRRIGATION & ASPIRATⁿ
⑦ FOLDABLE IOL INSERGION


continuous curvilinear incision

SMALL INCISION CATARACT SURGERY [SICS] VS PHACO EMULSIFICATION

SICS	PHACO EMULSIFICATION
→ 6mm incision	→ 3 mm incision
→ manual method	→ machine is used
→ PMMA IOL	→ foldable IOL
→ sutureless	→ sutureless
→ 6/6 vision restored	→ 6/6 vision restored
→ Economical	→ Costly

FLACS [FEMTOSECOND LASER ASSISTED CATARACT SURGERY]

→ FEMTOSECOND LASER ADVANTAGES
① Perfect Incision
② Perfect capsulorhexis
③ Less energy delivered
[Less traumatised]

→ costly


FEMTOSECOND LASER


FLACS INCISION


CAPSULORHEXIS

POST OP COMPLICATIONS

SECONDARY / AFTER CATARACT / POSTERIOR CAPSULAR OPACIFICATION

→ mc [30 - 40%]
→ Younger the age → faster the occurance
→ 50 - 75 yrs patients develops in 6-8 months
20 - 25 yrs patients develops in 3 months
1 yr child develops in 3 weeks

→ Trauma to LECs [LENS EPITHELIAL CELLS]
↓
stimulates mitosis
↓
Proliferation of cells
↓
migrates & deposits at the centre of posterior capsule
↓
Slow & painless loss of vision following cataract surgery

→ NO after cataract in ICCE

→ TYPES

① ELSHNIG'S PEARLS [more common]

② SOMMERING'S RING

SOMMERING'S RING ELSHNIG'S PEARLS

→ TREATMENT

ND YAG LASER CAPSULOTOMY

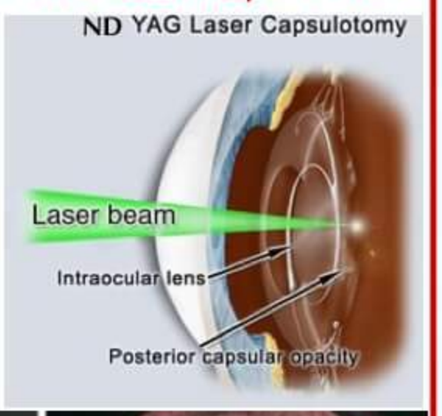
→ components of ND YAG LASER

ND → Neo Dymium

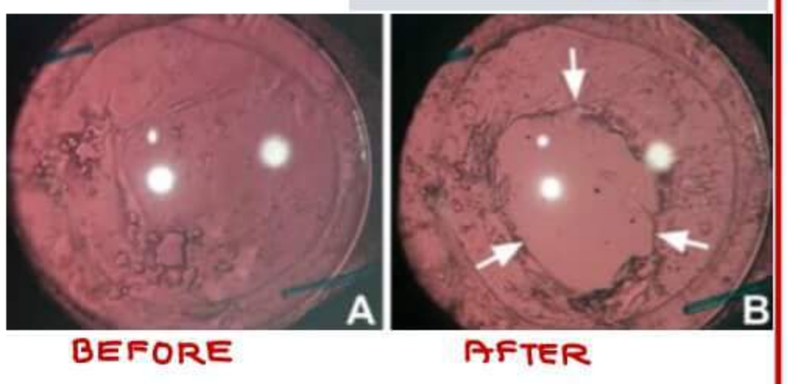
Y → Yttrium

A → Aluminium

G → Gornet [mineral]



→ Wavelength → 1064 nm



BEFORE AFTER

CYSTOID MACULAR EDEMA

→ causes slow painless vision

→ can occur in 1st month

→ IRVINE GASS SYNDROME → CME after cataract surgery

CME

Q $\frac{6}{6} \xrightarrow{\text{4 Weeks}} \frac{6}{18}$, Slow painless loss of vision, D_x ?

Ⓐ Secondary cataract

Ⓑ CME

ENDOPHTHALMITIS

→ infection of vitreous cavity [surgical emergency]

EARLY ONSET ENDOPHTHALMITIS	LATE ONSET ENDOPHTHALMITIS
→ < 6 wks [usually 2-3 days]	→ > 6 wks
→ CAUSES	→ CAUSES
– Staph. epidermidis [mc]	– Propionibacterium acnes [mc]
– Staph. aureus	

ENDOPHTHALMITIS

→ CLINICAL FEATURES

→ Immense pain

→ Marked congesion

→ dramatic loss of vision [< 72 hrs, from 6/6 vision to hand movement +]

→ TREATMENT

→ DOC : Intravitreal VANCOMYCIN [for Gram +ive organisms]

Intravitreal CEFTAZIDIME [for Gram -ive organisms]

→ maximum Macular toxicity → GENTAMYCIN

→ INTRACAMERAL [into anterior chamber] route can also tried

→ Most important PRECAUTION → Eye lid & Eye lash cleansing ℃ Povidone Iodine

POSTERIOR CAPSULAR RUPTURE


ACIOL


ACIOL

→ IOL is placed in Anterior chamber in this case
  → complication of ACIOL UGH SYNDROME
    - U → Uveitis
    - G → Glaucoma
    - H → Hyphema

→ In case of shallow anterior chamber [elderly], SCLERAL FIXED IOL used


SCLERAL FIXED IOL

Rx OF a ruptured posterior capsule	→	ACIOL
Rx OF APhakia	→	PCIOL

PEDIATRIC CATARACT

→ TIMING OF SURGERY depends on
  → LATERALITY
    - Bilateral cataract → 2-4 months [ideally at 8 wks]
      → after foveal fixation
    - Unilateral cataract → ASAP [ideally at 6 wks]
      → prone for AMBLYOPIA / LAZY EYE SYNDROME

AMBLYOPIA / LAZY EYE SYNDROME

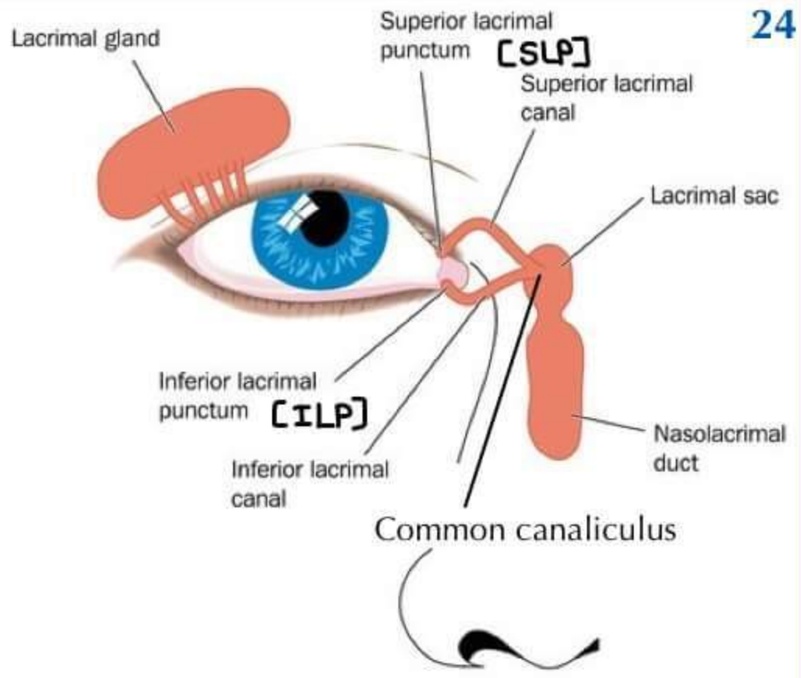

OCCLUSION Rx

→ Loss of vision in a child [< 8yrs]
→ d/t prolonged visual deprivation
→ Visual deprivation may be due to
→ cataract, squint, refractive error etc
→ MCC of amblyopia → Squint
→ DIAGNOSIS
  - Ⓐ 5yr child, $\frac{6}{60}$ + 3DS → $\frac{6}{6}$ → HYPERMETROPIA [Refractive error]
  - Ⓑ 5yr child, $\frac{6}{60}$ + 3DS → $\frac{6}{60}$ → AMBLYOPIA

→ Upper age limit to Rx amblyopia → 8-10 yrs
→ Ⓡx → OCCLUSION OF GOOD EYE [For 6-8hrs/day in 6 days/week]

cataract ↓ max visual handicap	→	Posterior Sub capsular cataract
cataract ↓ highest possibility of capsular rupture	→	Posterior polar cataract
IOL ↓ least incidence of secondary cataract	→	Hydrophobic acrylic IOL
cataract ↓ max fluctuation of vision	→	Intumescent cataract

LACRIMAL APPARATUS

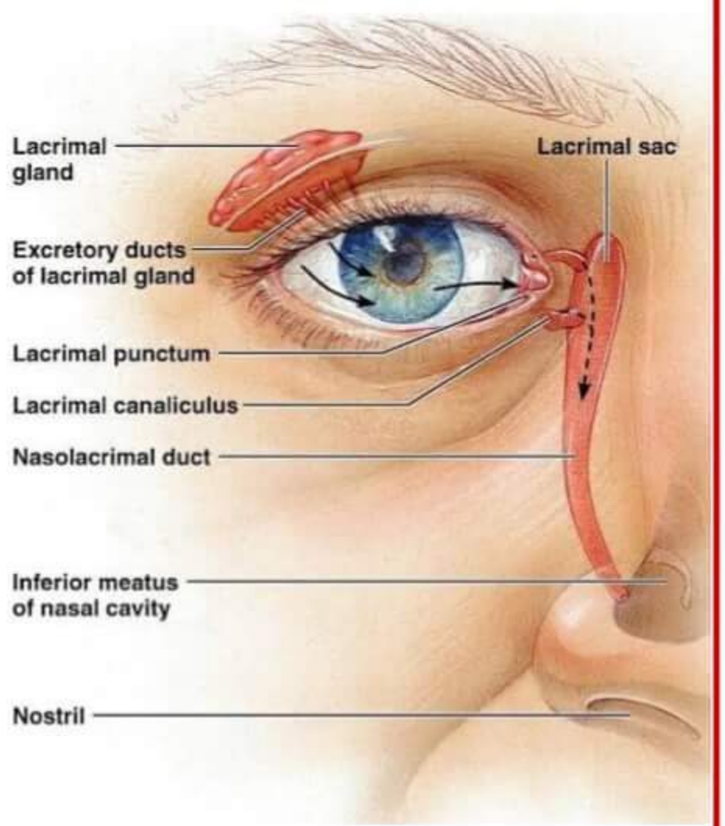


→ BASIC STRUCTURES

① LACRIMAL GLAND
② LACRIMAL SAC
③ NASO LACRIMAL DUCT [opens into inferior meatus of nose]

→ Upper canaliculus [UC] → 10 mm
Lower canaliculus [LC] → 10 mm
Lacrimal sac [LS] → 12 mm
Naso lacrimal duct [NLD] → 15 mm

→ 75 - 80% of tears drained out through inferior punctum



EPIPHORA

→ Excessive watering of eyes

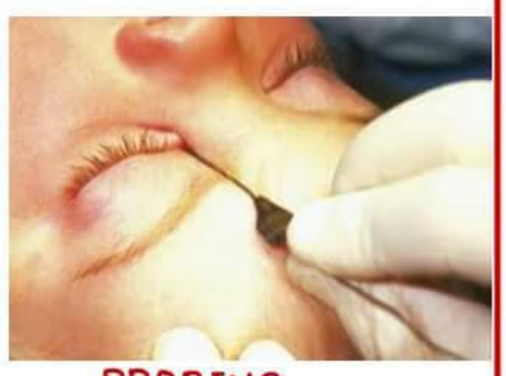
→ CAUSE

① Excessive lacrimation
② Blockage of lacrimal apparatus [more common]

→ BLOCKAGE OF LACRIMAL APPARATUS

→ COMMON SITES OF BLOCKAGE

① Nasolacrimal duct [mc]
② common canaliculus
③ upper| lower canaliculus


PROBING

→ Site of block determined by

① PROBING
② SYRINGING


SYRINGING

① PROBING

@ SOFT STOP → stoppage of probe d/t non canalizatn of valve

→ soft stop can occur due to

- inferior canaliculi blockage [When passed through ILP]
- superior canaliculi blockage [When passed through SLP]
- common canaliculi blockage

→ canaliculi blockage distinguished by length of the probe

- < 10mm → lower| upper canaliculi block
  > 10mm → common canalicular block

ⓑ HARD STOP → d/t blockage of Naso lacrimal duct

② SYRINGING ī H_2O

→ IF H_2O rushes out through inferior punctum
When injected through inferior punctum
} Lower canalicular block

→ IF H_2O rushes out through superior punctum
When injected through superior punctum
} Upper canalicular block

→ IF H_2O rushes out through one punctum
When injected through other punctum
} common canalicular block

→ IF H_2O rushes out through one punctum
When injected through other punctum
– ī a latency
ī mucus flakes [contents of Lac. sac]
} Nasolacrimal duct block

→ R/ PROTOCOL FOR CONGENITAL NLD OBSTRUCTION

→ incidence at birth	→ 6%
→ From birth till 1 yr of age	→ CRIGLER SAC MASSAGE
→ After 1 yr of Age	→ PROBING
→ After 3 yrs of Age	→ DACRYO CYSTO RHINOSTOMY [DCR]

UVEITIS

→ UVEA = GRAPE

→

COMPONENTS		INFLAMMATION
Iris	→	Iritis
Ciliary body	→	Cyclitis
Choroid	→	Choroiditis
ALL	→	Panuveitis

CLASSIFICATION

① ① ANTERIOR UVEITIS [mc; 70%]

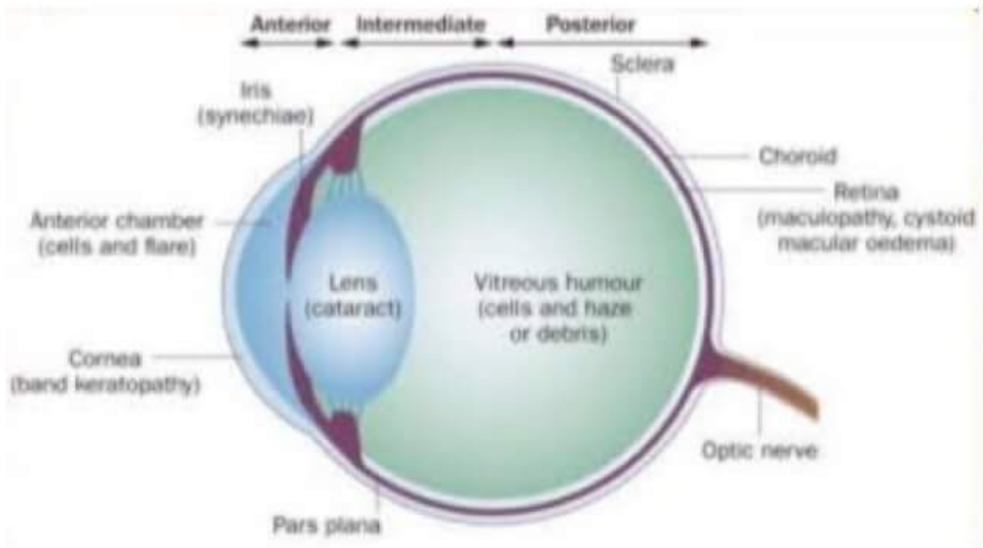
→ Iritis

→ Iridocyclitis [only pars plicata] [more common]

② INTERMEDIATE UVEITIS

→ Pars Planitis

→ Vitritis



③ POSTERIOR UVEITIS

→ choroiditis

④ PAN UVEITIS

→ Sympathetic Ophthalmitis

→ Vogt Koyanagi Harada Syndrome

Ⅱ ACUTE UVEITIS → Acute onset

CHRONIC UVEITIS → > 3 months

Ⅲ GRANULOMATOUS UVEITIS → TB, Leprosy, Syphilis, Sarcoidosis etc

NON GRANULOMATOUS UVEITIS


ANKYLOSING SPONDYLITIS UVEITIS

ANTERIOR UVEITIS

CAUSES

① IDIOPATHIC [mc, 50%]

② HLA B 27 SPONDYLO ARTHROPATHIES

ⓐ Ankylosing Spondylitis [2nd mc] → common in young males

ⓑ Inflammatory Bowel disease

ⓒ Psoriatic Arthritis

ⓓ Reiter's Syndrome [Reactive Arthritis]

→ Conjunctivitis, Uveitis, Arthritis


JRA UVEITIS

③ JUVENILE ARTHRITIS [JRA]

→ Pauciarticular, ANA positive, RF negative

→ Contra indication for IOL implantation [as eye is inflammed]

PRESENTATION

SYMPTOMS

→ Sudden red eye ī pain & loss of vision

SIGNS

① CIRCUMCILIARY CONGESION

→ bluish red

→ Radial

CIRCUM CILIARY CONGESION

② CELLS

→ WBC, neutrophils present in AC

→ elicited by slit lamp examinatⁿ

→ earliest sign

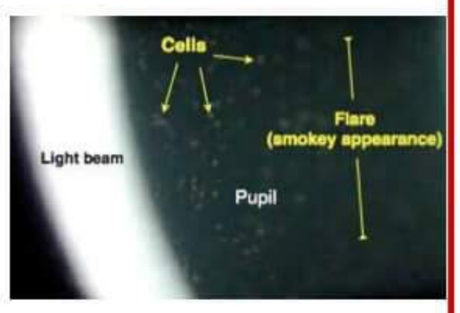
→ hallmark of activity

CELLS

③ FLARE

→ protein depositⁿ in aqueous humor

→ presents in severe uveitis



blood vessels get inflammed

↓

Serum protein leaks out & mixes ī aqueous humor

↓

TURBID AQUEOUS HUMOR

↓

Splits the light

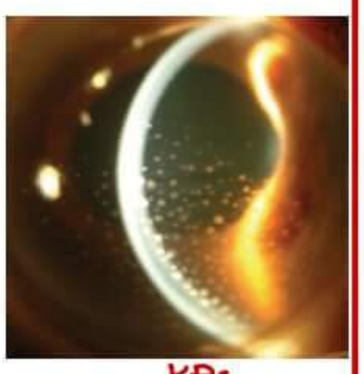
↓

FLARE

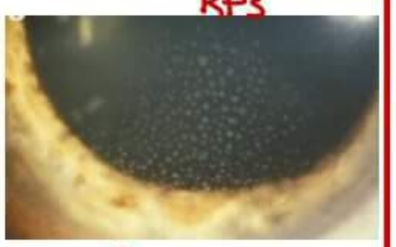
④ KERATIC PRECIPITATES [KP]

→ neutrophils & lymphocytes on cornea

→ ARLT'S TRIANGLE → Triangular area in the inferior part of cornea where KPs preferentially attaches



KPs



ARLT'S TRIANGE

→ DIFFUSE KPs → KPs attaches all over the cornea

→ Seen in

Herpetic uveitis

FUCh's Heterochromic Iridocyclitis

→ MUTTON FAT KPs → Look likes mutton fat

→ Seen in Granulomatous Uveitis

TB, Leprosy, Syphilis, Sarcoidosis

MUTTON FAT KPs

⑤ IRIS NODULES

ⓐ KOEPPE'S NODULES → present on pupillary margin

ⓑ BUSACCA'S NODULES → present on surface of Iris

→ Seen in Granulomatous diseases

KOEPPE'S NODULES

BUSACCA'S NODULES

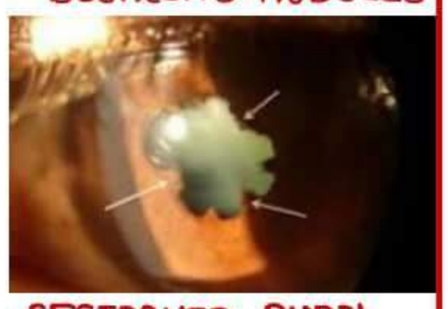
⑥ SYNECHIAE

ⓐ ANTERIOR SYNECHIA → may cause glaucoma

ⓑ POSTERIOR SYNECHIA → may cause cataract

FESTOONED PUPIL

- Small irregular pupil
- Sign of posterior synechiae



FESTOONED PUPIL

⑦ MIOSIS

⑧ LOW IOP initially

⑨ HYPOPYON → collection of pus in AC

→ feature of Severe Uveitis

HYPOPYON

INTERMEDIATE UVEITIS

CAUSES

① IDIOPATHIC [mc]

② SARCOIDOSIS [2nd mc]

③ MULTIPLE SCLEROSIS

INTERMEDIATE UVEITIS

CLINICAL FEATURES

① FLOATERS / MUSCAE

→ black spots floating infront of eye

→ looks like house flies → Musca

→ dlt neutrophils in vitreous cavity

→ seen in

1 normal people

2 vitreo retinal disorders

ⓐ LOSS OF VISION → commonest cause → CME

③ SNOW BALLS & SNOW BANK APPEARANCE

SNOW BALLS & SNOW BANK APPEARANCE

POSTERIOR UVEITIS [CHOROIDITIS]

CAUSES

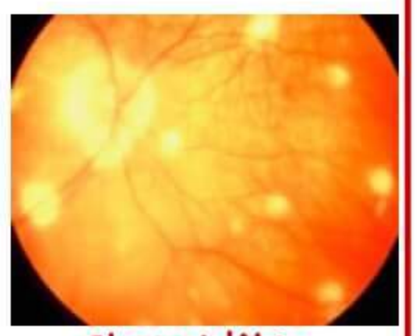
INFECTIONS	IMMUNE DISORDERS
→ Toxoplasmosis [mc]	→ Sarcoidosis [mc immune]
→ TB [2nd mc]	→ PAN
→ Toxocariasis , Herpes	→ Scleroderma
→ CMV , HIV	

CLINICAL FEATURES

- Presents as
  - CHORIORETINITIS
  - VASCULITIS
  - VITRITIS

- O/E
  - Creamy yellow patches seen → Choroiditis
  - Chorioretinitis
  - vitritis
  - vasculitis
  - HEAD LIGHT IN FOG APPEARANCE seen
    - Chorioretinitis + Vitritis
      - Chorioretinitis → Head light
        Vitritis → Fog

    - occurs in Toxoplasmosis


Choroiditis


Headlight in fog

TREATMENT

① ANTERIOR UVEITIS

@ TOPICAL STEROIDS

- DOC
- S/E
  - Topical steroids → Glaucoma
    Systemic steroids → cataract

- Topical steroids
  - BETA METHASONE
  - DEXA METHASONE [causes max glaucoma]
  - PREDNISOLONE
  - FLUOROMETHOLONE [causes min. glaucoma] [SOFT STEROIDS]

- Glaucoma occurance depends on AIP [Anti inflam. Potency]
  AIP ∝ Glaucoma causation

TOPICAL STEROIDS

@ CYCLOPLEGICS

- ATROPINE → 14 days action [most potent]
  HOMATROPINE → 3 days action
  CYCLOPENTOLATE → 1 day action
  TROPICAMIDE → 6 hrs action [least potent]

- relaxes ciliary spasm [reduces pain]
  - DOC for Acute anterior uveitis
- Dilates the pupil, breaks synechiae
- Reduces vascularity


posterior synechiae breakage by cycloplegic

→ HOMATROPINE is preferred drug for R_4

→ ATROPINE OINTMENT is preferred in children

→ they require strong cycloplegic action

→ When we use DROPS,

→ 20% absorbed in cornea

→ 80% absorbed in systemic circulation

– leads to Atropine toxicity in children

Subconjunctival inj.

Subtenon's injectn

② INTERMEDIATE UVEITIS

@ STEROID INJECTIONS

→ TRIAMCINOLONE

→ only injectable steroid in the eye

→ by Subconjunctival route or

by Subtenon's route [better but more risky]

③ POSTERIOR UVEITIS

@ ANTI BIOTICS for infections

→ DOC for Toxoplasmosis during pregnancy → SPIRAMYCIN

→ HAART Therapy for HIV

→ GANCYCLOVIR for CMV

ⓑ SYSTEMIC STEROIDS

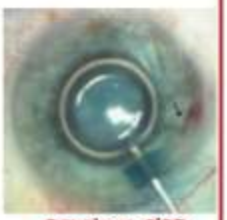
→ not to give for > 3 months

ANTERIOR UVEITIS	INTERMEDIATE UVEITIS	POSTERIOR UVEITIS
→ Iridocyclitis	→ Pars planitis Vitritis	→ Chorioretinitis
→ Idiopathic	→ Idiopathic	→ Toxoplasmosis
→ Pain ✓ Redness ✓ Loss of vision ✓	→ Pain × Redness × Loss of vision ✓ Muscae/floaters	→ Pain × Redness × Loss of vision ✓
→ Cells	→ Snow balls & Snow banks	→ Toxoplasmosis → Headlight in fog viritis, vasculitis, chorioretinitis
→ Topical steroids Cycloplegics	→ Inj. Triamcinolone	→ systemic steroids anti microbials

FUCHS HETEROCHROMATIC IRIDOCYCLITIS [FHI]


Fuchs heterochromatic iridocyclitis

- → usually U/L uveitis
- → seen in young age
- → Idiopathic
- → TRIAD
  - Heterochromia
  - cataract
  - Diffuse KPs
- → Low grade inflammation
  - → No remarkable symptoms until cataract formation
    - – Presents as young patient ↓ U/L cataract


Amslers sign

- → O/E
  - → Heterchromia
  - → cataract
  - → Diffuse KP s
  - → AMSLER'S SIGN → Paracentesis [entering AC], causes Hyphema
  - → NO posterior synachiae
  - → Uveitis ↑ max. predisposition to glaucoma → FHI
    → Herpetic Uveitis

SARCOIDOSIS

- → can cause
  - Anterior uveitis [mc]
  - Posterior uveitis
  - Intermediate uveitis
  - Pan uveitis


Mutton fat KPs


Candle wax drippings

- → 2nd mc presentation → Uveitis
  mc presentation → B/L lymphadenopathy
- → more common in colored races
  more common in 20-50 yr females
- → CLINICAL FEATURES
  1. MUTTON FAT KPs
  2. KOPPE'S | BUSACCA'S NODULES
  3. B/L UVEITIS
  4. VITREOUS INFILTRATION → SNOWBALL / STRING OF PEARL APPEARANCE
  5. CANDLE WAX DRIPPINGS [VASCULITIS]

SYMPATHETIC OPHTHALMITIS (PAN UVEITIS)

→ B/L granulomatous uveitis

→ caused by TRAUMA TO ONE EYE

→ Injured eye → Exciting eye

→ Other eye → Sympathising eye

→ caused by

→ penetrating / perforating injury

→ metallic foreign body

→ injury to ciliary body

– Dangerous Area of eye → Ciliary body

– causes max. sympathetic Ophthalmitis

→ CLINICAL FEATURES

→ presents at 2 weeks – 2 months after injury

→ DALEN FUCH'S NODULES on choroid seen

→ mcly seen in children

→ earliest symptom → Photophobia in good eye

→ Accommodation failure in good eye

Dalen Fuch's Nodules

→ Earliest sign → Retrolental flare [vitreous flare]

Mutton fat KPs

Hypotony

→ TREATMENT

INJURED EYE	NORMAL EYE	MANAGEMENT
PL → –ive	$\frac{6}{6}$ vision	→ ENUCLEATION of Injured eye
$\frac{6}{60}$ vision	$\frac{6}{6}$ vision	→ REPAIR of Injured eye
PL → –ive	progressed sympathetic Ophthalmitis	→ High dose SYSTEMIC STEROIDS for sympathetic eye

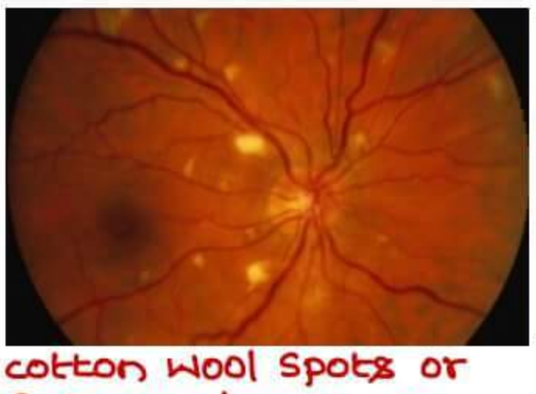
OCULAR HIV

→ mc Ocular manifestatn

→ Cotton wool spots or

→ Soft exudates or

→ HIV retinopathy



cotton Wool Spots or Soft exudates or HIV Retinopathy

Pizza pie appearance

→ Commonest occular infection → CMV retinitis

→ crumbled cheese and tomato ketchup / pizza pie appearance

→ Brush fire appearance

→ mc ocular tumor → Kaposi sarcoma > Lymphoma

Kaposi's Sarcoma

→ mc systemic infection in HIV } TUBERCULOSIS
mc cause of death in HIV }

→ commonest cause of soft exudates → Diabetes | HTN Retinopathy

→ commonest ocular SIE OF HAART Therapy → IMMUNE RECOVERY UVEITIS (anterior | intermediate)

2 2 2 2

OPPORTUNISTIC INFECTIONS OF HIV

1 TUBERCULOSIS } 2 Bacterial
2. SYPHILIS }

3 CRYPTOCOCCUS } 2 fungal
4 CANDIDA }

5 HERPES ZOSTER } 2 viral [most common]
6 CMV }

7 PNEUMOCYSTIS } 2 parasitic
8 TOXOPLOSMOSIS }

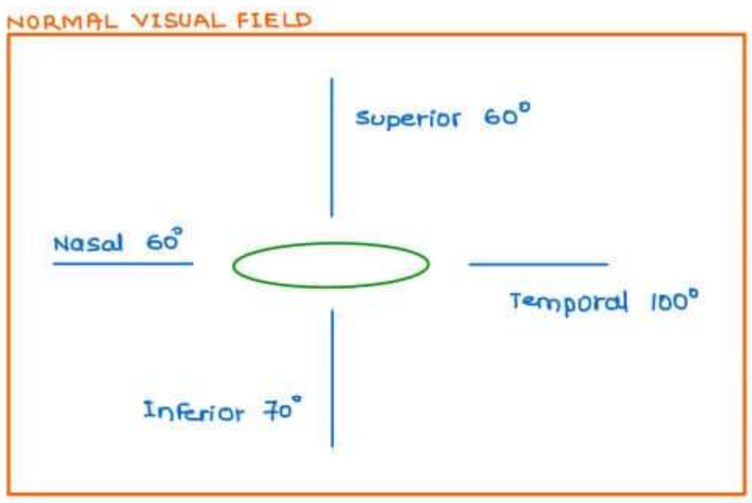
GLAUCOMA

→ 2nd mcc of blindness in the world

CLASSIC TRIAD OF GLAUCOMA [Atleast 2 out of 3 → GLAUCOMA]

1 ↑ IOP [> 22 mm of Hg]
2 Visual field Defects
3 Optic Disc changes

VISUAL FIELD DEFECTS



visual field contract^{n} is present in Glaucoma

OCULAR HTN

↑ IOP
Ⓝ visual field
Ⓝ optic disc

LOW TENSION GLAUCOMA

Ⓝ IOP
visual field defect
optical disc changes ⊕

PRIMARY

→ cause unknown
→ more common

SECONDARY

→ causes
- Uveitis
- Neovascular
- Lens induced
- Trauma

PRIMARY GLAUCOMA

1 Childhood
- congenital → from birth till 3yrs , Buphthalmos
- Juvenile → >3yrs of Age , normal eyes

2 Adult
- Open Angle Glaucoma [OAG] } differentiated by GONIOSCOPE
- Angle closure Glaucoma [ACG] } [measure the angle]

CONGENITAL GLAUCOMA

→ From birth till 3yrs
→ Buphthalmos [Large eye]
→ BARKAN'S MEMBRANE → blocks aqueous flow
→ AR [autosomal recessive]
→ also consanguineous marriage

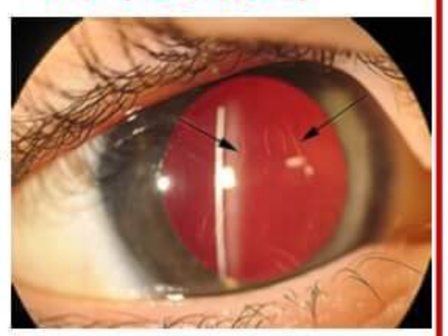
→ CLASSICAL TRIAD

1 Lacrimation
2 Photophobia
3 Blepharospasm

→ Signs

→ Large eye
→ Hazy cornea d/t corneal edema
→ ↑ IOC overcomes the endothelial pump resistance
→ HAAB'S STRIAE

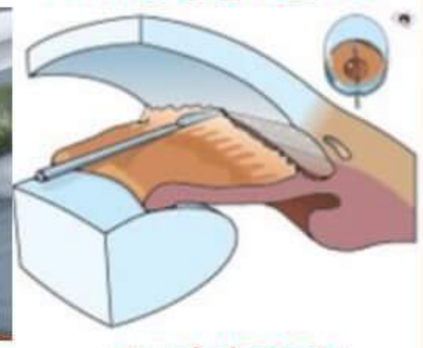

HAZY CORNEA


HAAB'S STRIAE

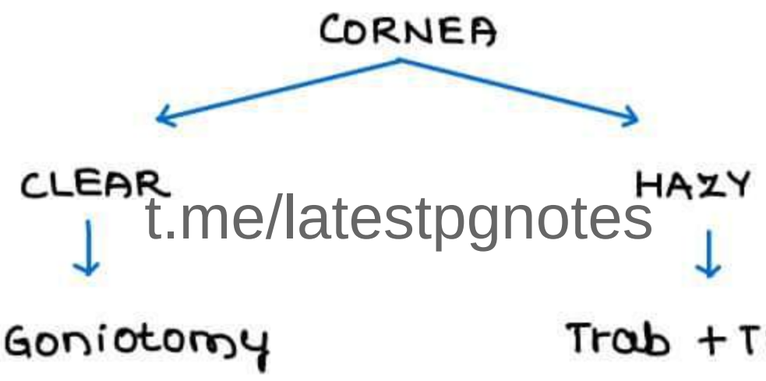
→ TREATMENT

1. GONIOTOMY
   → safer but rare
   → Gonioscopy is d/t Hazy cornea

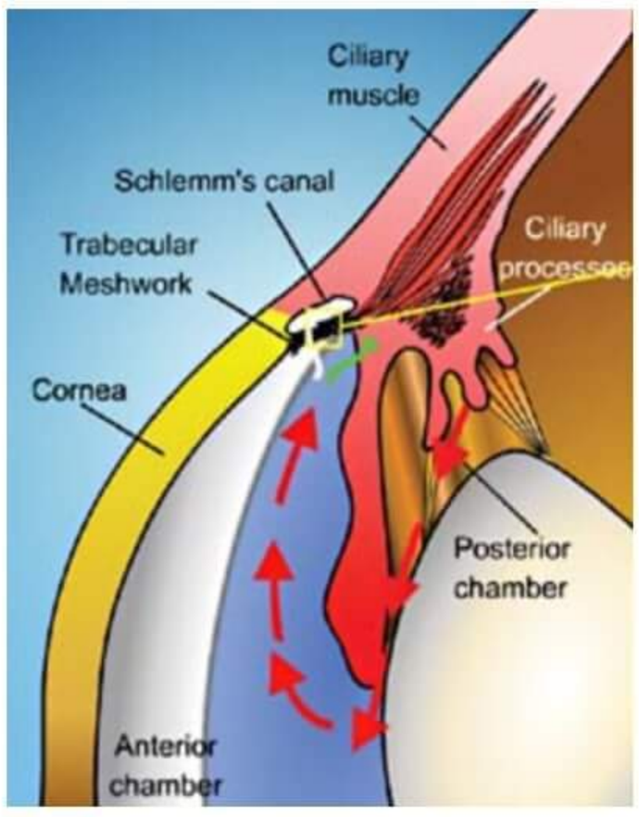

Trabeculotomy


Goniotomy

2. TRABECULOTOMY
3. TRABECULOTOMY + TRABECULOTOMY [TRAB + TRAB]



AQUEOUS HUMOR DYNAMICS



OPEN ANGLE GLAUCOMA

→ 3-4 times more common
→ Glaucoma ≅ OAG
→ more dangerous

ANGLE CLOSURE GLAUCOMA

→ more painful

ANGLE CLOSURE GLAUCOMA

More common in

→ Middle aged ['coz of growing lens] Women [d/t shallow AC]

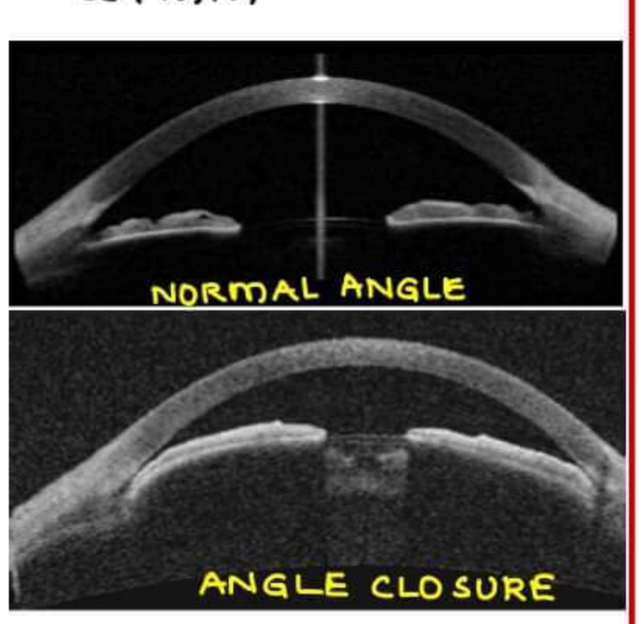
→ Hypermetropia

→ Emotionally unstable women

Hypermetropia → condition where rays focus behind the lens

→ mc cause → Axial hypermetropia [small eye ball]

N Axial length → 24 mm



→ PUPILLARY BLOCK d/t mid dilated pupil

Diameter of pupil

Normal	→	3-4 mm
Fully dilated	→	9-10 mm
mid dilated	→	6-8 mm

→ In 30 min, IOP changes from 15 mm → 60 mm

[4 times the Ⓝ pressure]

→ Severe pain
→ Loss of vision
→ Vomiting

} Acute Angle closure glaucoma / Acute congestive glaucoma

SYMPTOMS

→ occurs in late nights

→ Severe pain

→ colored halos

→ sudden drop in vision

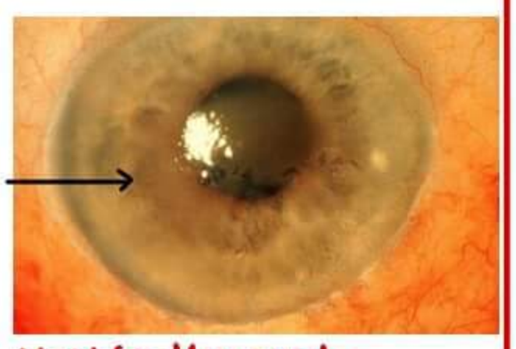
} d/t corneal edema

SIGNS

→ Stony hard eye

→ Steamy cornea

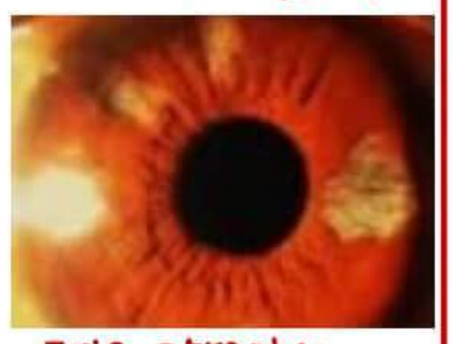
→ vertically oval, mid dilated, non reacting pupil



vertically oval, mid dilated, non-reacting pupil

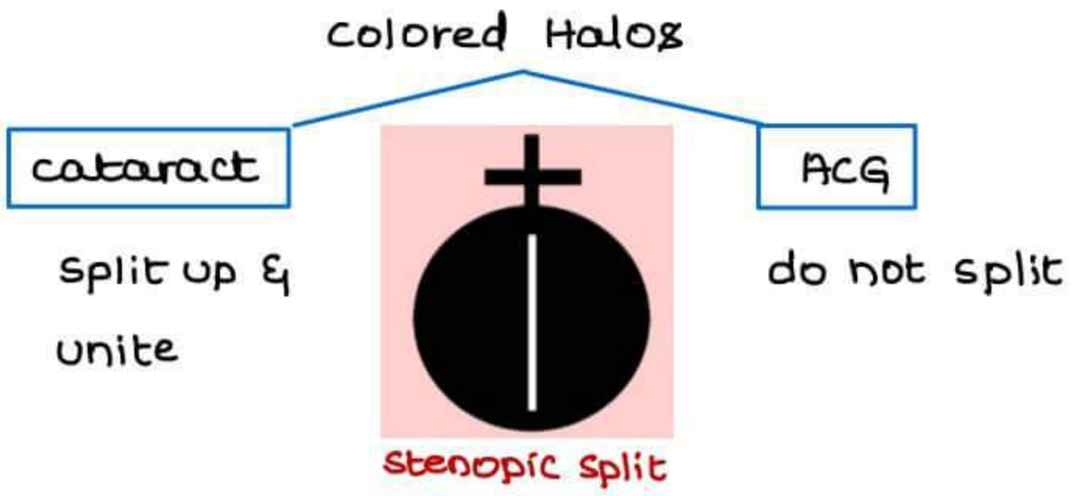
→ VOGT'S TRIAD → tells us about the past attack

1. Iris atrophy
2. Peripheral anterior synechiae
3. Glaucomaflecken


Iris atrophy


Glaucomaflecken

FINCHAM'S TEST



Colored halo

OPEN ANGLE GLAUCOMA | SILENT THIEF OF SIGHT

RISK FACTORS

1. Coloured Races
2. thin corneas
3. myopics
4. elderly
5. Trabecular fibrosis
6. myocillin gene
7. Optineurin gene

FIBROSIS OF TRABECULAR MESHWORK → TRABECULAR OUTFLOW BLOCKED

→ very slow painless loss of vision [20-30yrs]
→ Ⓝ aqueous production → 2.5 µL/min
→ 'C' value Ⓝ value → 0.2 µL/min/mm of Hg
→ Above 2 values will ↑ dlt → trabecular blockade

SYMPTOMS

→ PAINLESS [dlt very slow onset]
→ NO CORNEAL EDEMA
→ NO COLORED HALOS
→ NO LOSS of VISION

CAN BE DIAGNOSED EARLIER BY

→ Progressive rise of IOP + nt [may get unnoticed]
>40yrs, measurement of IOP to be done every year

→ PERIMETRY should be done

BY THE TIME OF PRESENTATION

→ TUNNEL VISION + nt [END STAGE GLAUCOMA]
→ aka SILENT THIEF OF SIGHT [slowly progressive in nature]


Tunnel vision

ONLY SYMPTOM → FREQUENT CHANGE OF PRESBYOPIC GLASSES
every 6-8 months [Ⓝ frequency - 3yrs]

→ ↑'d DARK ADAPTATION TIME


Normal cornea
Keratoconus
Vision in Keratoconus

FREQUENT CHANGE OF PRESBYOPIC GLASSES → OAG
FREQUENT CHANGE OF DISTANT VISION GLASSES → CATARACT
FREQUENT CHANGE OF GLASSESS IN YOUNG → KERATOCONUS > PATHOLOGICAL MYOPIA

ACG	OAG
→ Female predominance	→ No gender predisposit^{n}
→ 45 years	→ Elderly
→ Hypermetropic	→ myopic
→ Sudden, painful	→ Slow, painful
→ colored halos	→ No symptoms
→ pupillary Block	→ Trabecular fibrosis


Humphrey's Perimeter

VISUAL FIELD [Superior & Nasal → 60°; inferior → 70°; temporal → 100°]

→ area of space visible to human eye ☐ out moving the eye from a central target

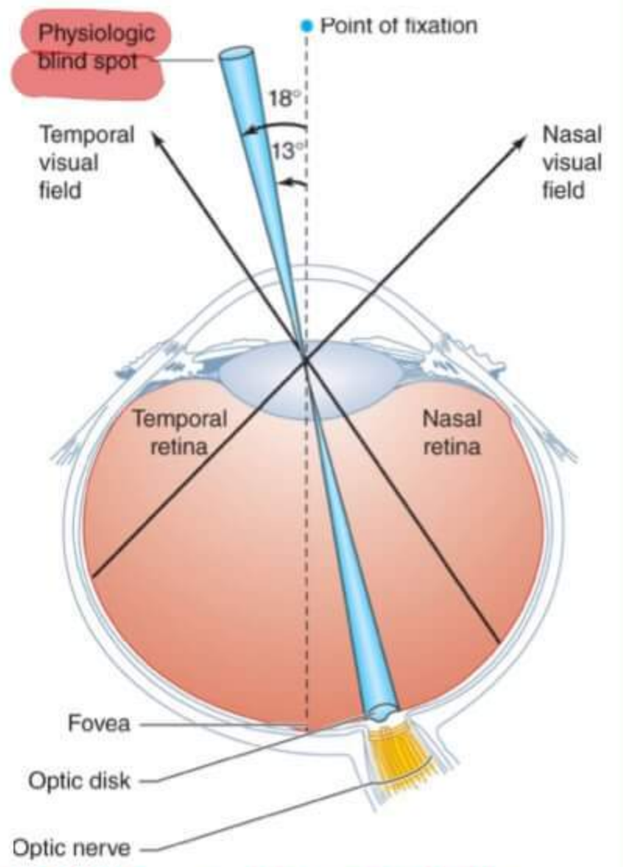
→ measured by Perimeter [HUMPHREY'S PERIMETER (Gold Standard)]

VISUAL FIELD DEFECTS

→ dlt optic nerve damage

→ 2 Kinds of visual field defects

1. dlt Glaucoma
2. dlt Neurophthalmologic conditions
   - ↳ Optic neuritis
   - ↳ Retro bulbar neuritis
   - ↳ Anterior ischemic neuropathie etc



RETINA & VISUAL FIELD

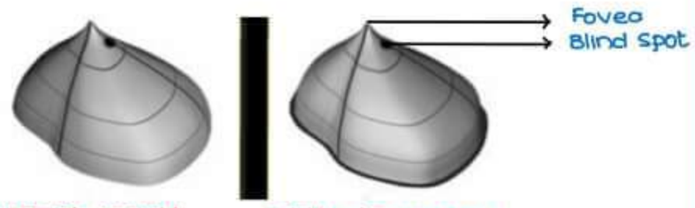
NORMAL VISUAL FIELD

SUPERIOR RETINA CONTROLS INFERIOR FIELD

INFERIOR RETINA CONTROLS SUPERIOR FIELD

NASAL RETINA CONTROLS TEMPORAL FIELD

TEMPORAL RETINA CONTROLS NASAL FIELD



NORMAL VISUAL FIELD — EARLY CONTRACTN

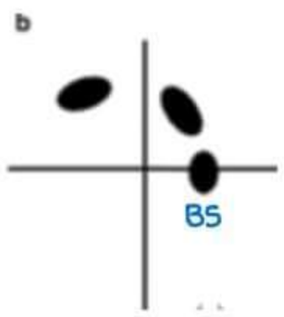
Physiological Blind spot is Located in Temporal visual field as optic disc lies in Nasal retina

VISUAL FIELD DEFECTS IN GLAUCOMA

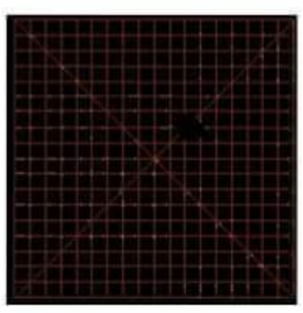
1 PARA CENTRAL SCOTOMA

→ Earliest VFD

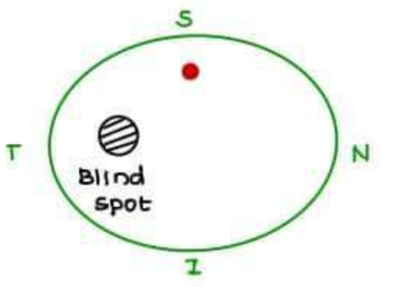
→ Localized loss of vision surrounded by normal vision



PARA CENTRAL SCOTOMA


PARA CENTRAL SCOTOMA


AUTO CORRECTION



PARA CENTRAL SCOTOMA

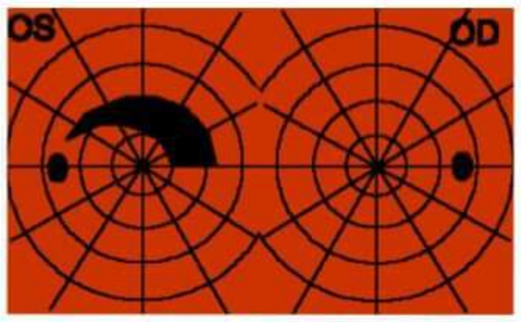
2 BJERRUM SCOTOMA → Scotoma in Bjerrum's area

→ BJERRUM'S AREA → An extension of blind spot in the shape of an arc in the central 30°

→ occupied by the arcuate nerve fibres

→ these are the 1st attacked nerve fibres in glaucoma

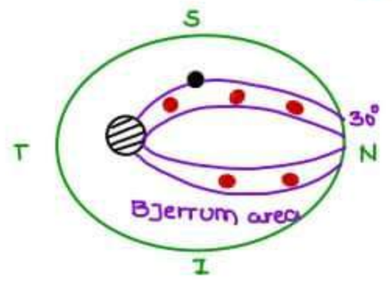
→ characteristic field defect of glaucoma



BJERRUM'S SCOTOMA

BJERRUM'S SCOTOMA

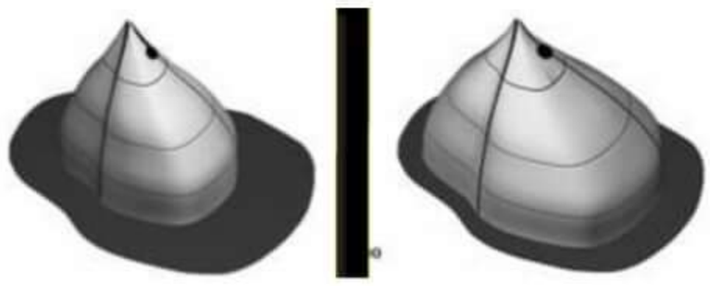
AUTO CORRECTION



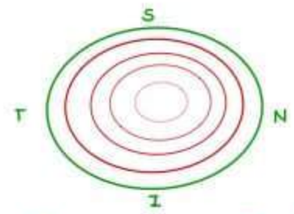
BJERRUM SCOTOMA

3. GENERALISED CONSTRICTION OF FIELD / CONCENTRIC CONTRACTION OF ISOPTERS

→ constrictⁿ visual field in all directions

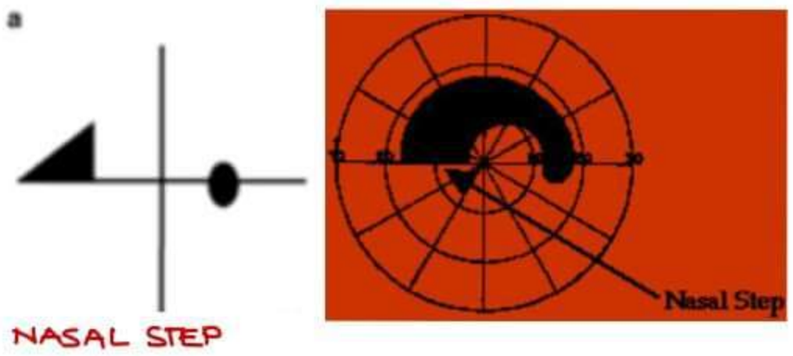


GENERALIZED CONTRACTION OF ISOPTERS

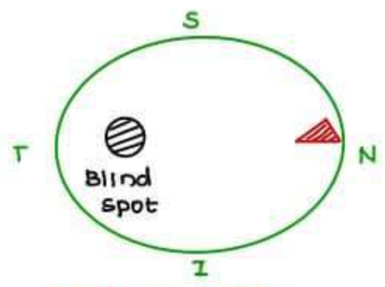


GENERALIZED CONSTRICTⁿ

4. NASAL STEP → characteristic field defect of glaucoma



NASAL STEP

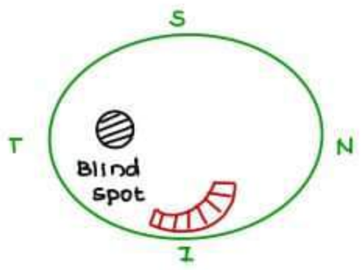


NASAL STEP

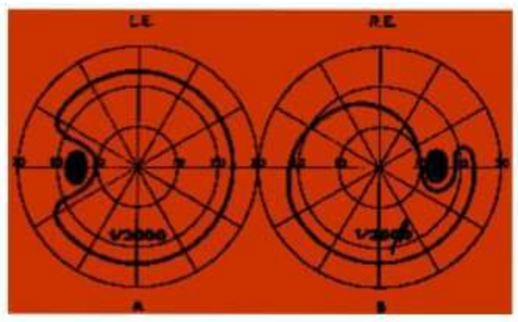
5 ARCUATE SCOTOMA → Scotoma in shape of an arc

6 BARING OF BLIND SPOT

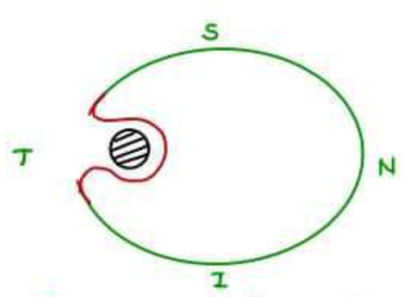
ARCUATE SCOTOMA



ARCUATE SCOTOMA

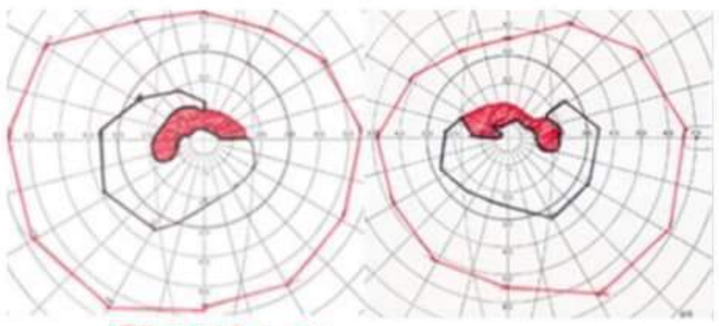


BARING OF BLIND SPOT

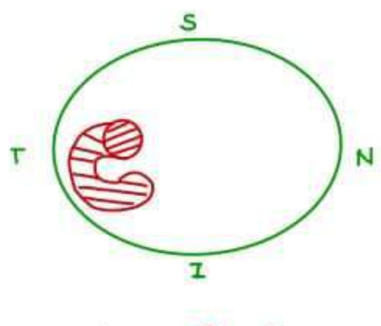


BARING OF BLIND SPOT

7 SEIDEL' SCOTOMA → Sickle shaped extension of the blind spot



SEIDEL'S SCOTOMA



SEIDEL'S SCOTOMA

1. NO SEQUENCE in particular in formation of visual field defects
   - → mostly the earliest is Paracentral Scotoma
   - → RULE OF THUMB → Earliest VFD in glaucoma is always begin from close to center [paracentral > Bjerrum > Generalised constrictⁿ]

2. CHARACTERISTICS
   - → Nasal Fields are first destroyed & Temporal vision is last to be destroyed
   - → most scotomas are shaped [Arc]
   - → follow horizontal meridian

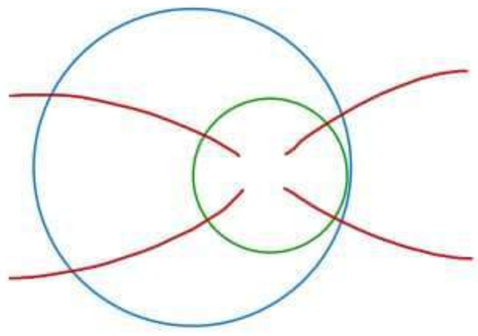
3 NEGATIVE SCOTOMAS

→ Glaucoma scotomas are negative scotomas
  ↳ can't observed dlt cortical filling in
  ↳ Example – Blind spot
  ↳ measured by Humphrey's perimeter


Positive scotoma

→ Positive scotomas are seen in Retinal disorders
  ↳ Patient can observe the scotoma

OPTIC DISC

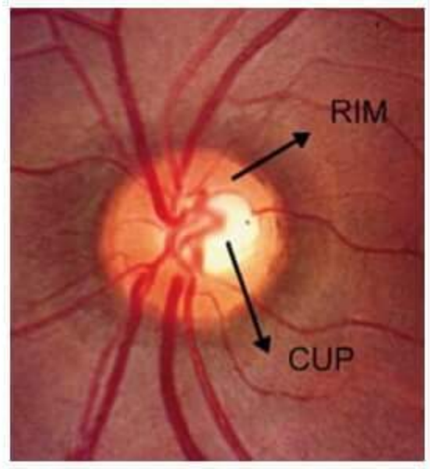


CUP → allow blood vessels to enter & exit the eye ; No function perse

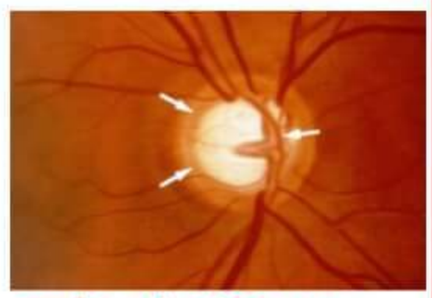
NRR
→ contains neurons of Optic Nerve
Each optic nerve contains 1.2 M neurons
In Glaucoma, NRR surface area gradually decreased

→ CUP DISC RATIO [CDR]

$$CDR = \frac{\text{Area of CUP}}{\text{Area of DISC}}$$



Normal optic Disc


Optical Disc in glaucoma

  ↳ Normal CDR = 0.3 [Range → 0.3 - 0.6]
  ↳ CDR → ≥ 0.7 → Glaucoma
  ↳ CDR indicates the NRR damage

NORMAL OPTIC DISC

→ clear margin
→ color of NRR → Reddish pink color
→ OD → 0.3 to 0.6
→ Distribution of vessels
  ↳ ② Central Retinal Artery → Relatively narrow
  ↳ ② Central Retinal vein → more dilated
  → PULSATES

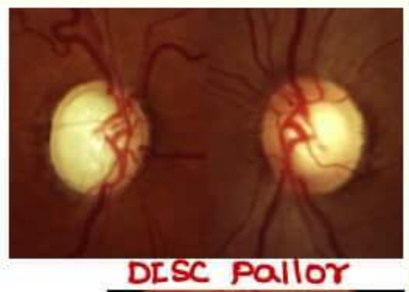
  ↳ 1 artery & 1 vein → nasal
  ↳ 1 artery & 1 vein → Temporal

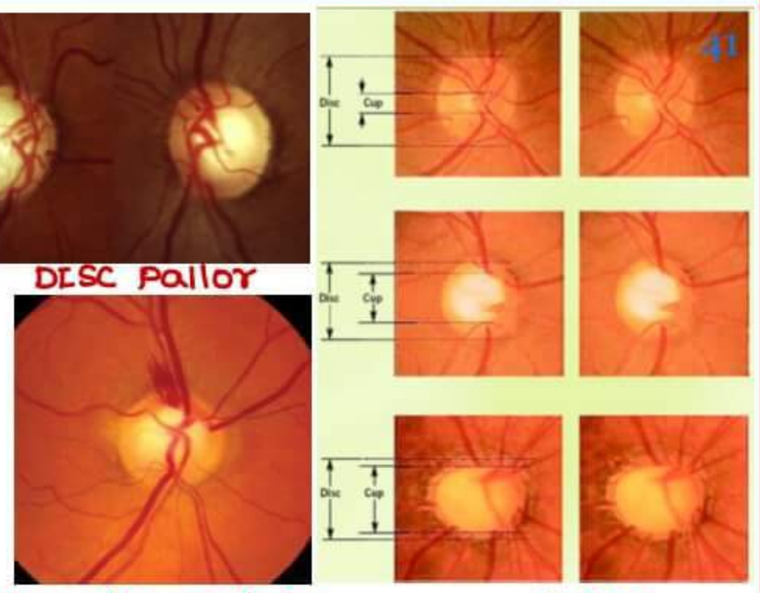
ONLY PULSATILE VEINS IN THE BODY
→ JUGULAR VEIN
→ CENTRAL RETINAL VEIN

↳ IF central Retinal artery pulsatis, then
  1. IOP → very high (or)
  2. BP → very low
  } Emergency conditions

OPTIC DISC CHANGES IN GLAUCOMA

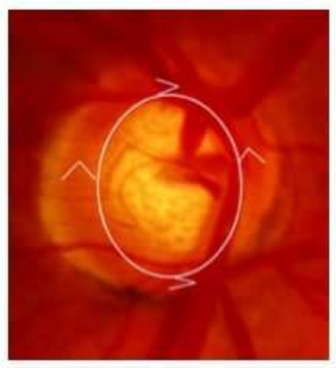
1. ↑ CDR
2. DISC PALLOR
3. SPLINTER HAEMORRHAGE
4. SIGN OF NASALIZATION [apparent shift]
5. LAMINAR DOT SIGN → ↑ no. of dots
   dlt more exposed Lamina Cribrosa

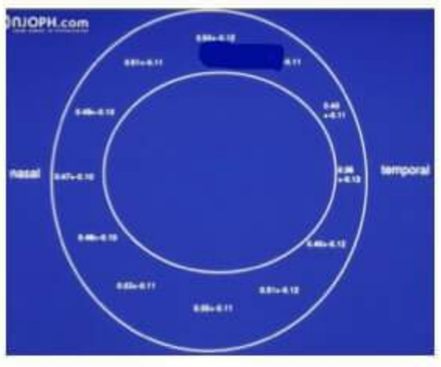

DISC Pallor



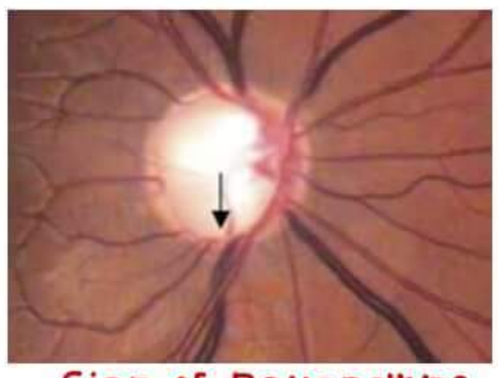
↑ CDR

Splinter Haemorrhage

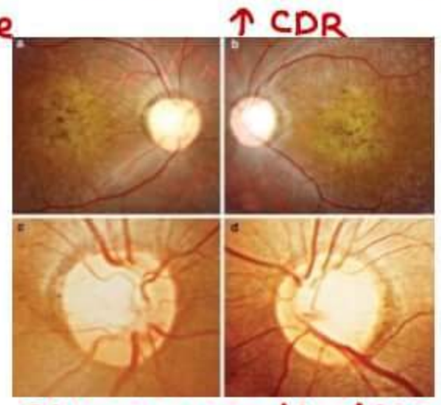

Vertically oval cup



ISNT RULE

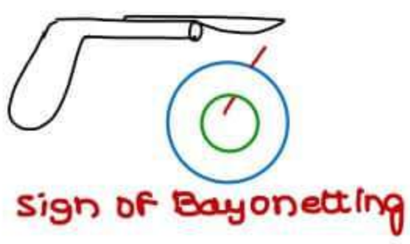

Sign of Bayonetting

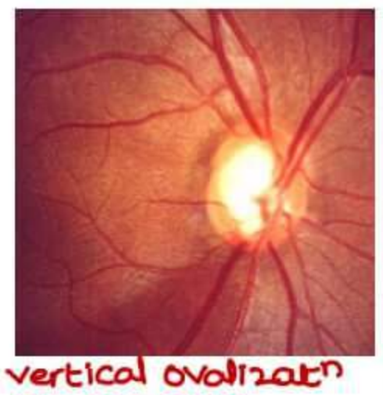

Laminar Dot Sign


Sign of nasalization

6. SIGN of BAYONETTING
   apparent discontinuity of blood vessel
7. ISNT RULE [Inf. Sup. nasal & Temporal]
   Sequence of Width decreases
   Inferior → Widest
   Superior
   Nasal
   Temporal → Narrowest

ISNT Rule broken in glaucoma
[dlt vertical ovalization of the cup]


Sign of Bayonetting


vertical ovalizatn of the cup


Glaucomatous Optic atrophy

8. GLAUCOMATOUS OPTIC ATROPHY

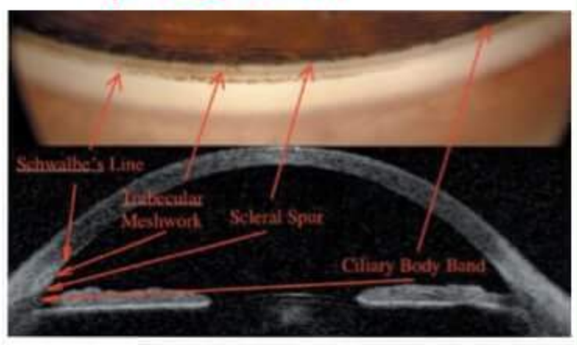
GONIOSCOPY

→ Differentiates OAG & ACG
  ↳ Main mx modalities
    → OAG → Medical
    → ACG → Surgical

→ PREREQUISITES → 1 CLEAR CORNEA
  → 2 NORMAL PUPIL


GONIOSCOPY

→ STRUCTURES LIE IN THE NORMAL ANGLE from cornea to Iris



1. SCHWALBE'S LINE [represents the terminatn of Descem-ent's membrane on to cornea]
2. TRABECULAR MESHWORK [Band]
3. SCLERAL SPUR [Line]
4. CILLIARY BODY BAND

GRADING

GRADE IV

→ 40°

→ all 4 structures can be seen

→ usually do not close [no ACG]

GRADE III

→ 30°

→ ciliary band can not be seen , all other 3 structures seen

→ usually do not close [NO ACG]

GRADE II

→ 20°

→ Schwalbe's line & Trabecular meshwork seen

→ Scleral spur & ciliary band not seen

→ can progress to ACG

GRADE I

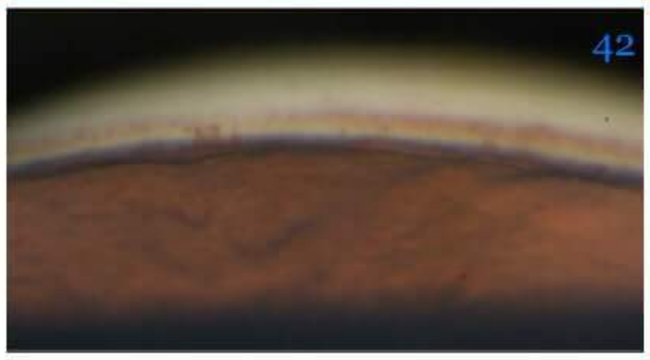
→ only structure seen is → Schwalbe's line

→ high risk for ACG

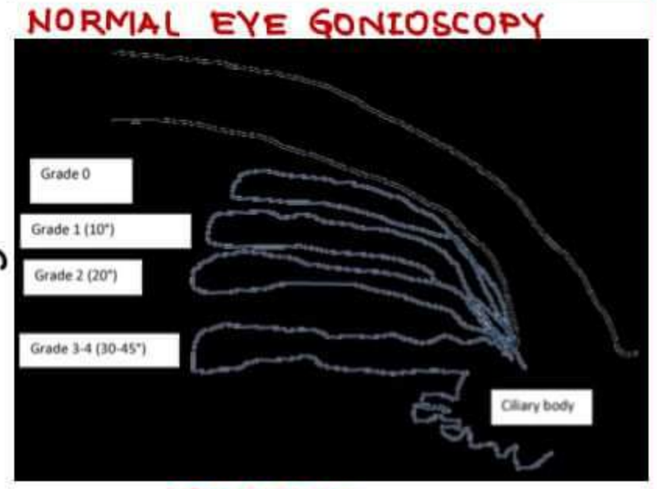
GRADE 0

→ ACG occur right now

→ Schwalbe's line also not seen



NORMAL EYE GONIOSCOPY



GRADING

MANAGEMENT

OAG

MEDICAL MANAGEMENT

↑ OUT FLOW

1. PILOCARPINE
2. PG ANALOGUES

↓ PRODUCTION

β blockers

carbonic anhydrase Inhibitors

SURGICAL MANAGEMENT

1. ALT [ARGON LASER TRABCULOPLASTY]
2. TRABECULECTOMY

ANTI GLAUCOMA DRUGS

1 CHOLINERGIC AGONIST [PILOCARPINE]

→ ↑ Trabecular outflow

→ S|E

1 Uveitis

2 ciliary spasm

→ Ocular pain + nt

→ loss of distant vision

3 Pseudomyopia

4 Punctal stenosis

5 Retinal detachment

2 β BLOCKERS

→ ↓ Production

→ NON - SELECTIVE

TIMOLOL

LEVO BUNOLOL

→ SELECTIVE

BETAXOLOL

→ SIE

1. C/I in Bronchial Asthma, COPD
2. C/I in Arrythmias
3. Dry eyes
4. Depression
5. Nasolacrimal Duct Block

3 EPINEPHRINE

→ ↑ Trabecular outflow } DUAL MECHANISM
↓ Production }

→ SIE

1 SYSTEMIC
→ Sweating
Palpitatⁿ, tachycardia, HTN
nervousness, Tremors

2 OCULAR
→ CME in aphakia
Pupil dilatatⁿ

→ C/I in ACG, HTN

DIPIVEFRINE

→ prodrug of epinephrine
→ only intraocular S/E
→ C/I in ACG

4. CARBONIC ANHYDRASE INHIBITORS

→ ↓ Production

→ SYSTEMIC → ACETAZOLAMIDE

TOPICAL → DORZOLAMIDE & BRINZOLAMIDE

→ ACETAZOLAMIDE

S/E → C/I in Sulfa Allergy [contains sulfa group]
→ hypokalemia
→ Acidosis
→ chronic Renal failure
→ Hepatic failure [C/I]

→ DORAZOLAMIDE & BRINZOLAMIDE
  → safest
  → DOC in children
  → SIE → corneal decompensation [↑ corneal edema]

5 α_2 AGONISTS

→ BRIMONIDINE & APRACLONIDINE

→ ↓ production
  ↑ uveoscleral outflow
} DUAL MECHANISM

→ BRIMONIDINE
  SIE → CI children [dlt apnea & death]
  → drowsiness & depression
  → NEUROPROTECTION

→ APRACLONIDINE
  SIE → Tachyphylaxis
  → Blepharoconjunctivitis [maximum]

6 PROSTAGLANDIN [$PGF_{2\alpha}$] ANALOGUES

→ ↑ uveoscleral outflow
→ LATANOPROST
  BIMATOPROST
  UNOPROST

IRIS HYPERCHROMIA

→ DOC for OAG & Normo Tension Glaucoma
→ most potent anti glaucoma drugs
→ SIE → Uveitis, CME,
  IRIS HYPERCHROMIA [irreversible]
  Blepharoconjunctivitis
  Trichomegaly [common ∈ BREMATOPROST]


Before Rx


After Rx


TRICHOMEGALY

7 HYPEROSMOTICS

→ Vitreous humor contains → 98% of water
→ These drugs shrinks the vitreous volume

→ MANNITOL
  → Given IV
  → Fastest acting antiglaucoma drug [∈ in 20 min]
  → DOC for Acute ACG
  → SIE → Decompensation in case of CHF


Mannitol

→ GLYCEROL
  → oral syrup
  → C/I in DM

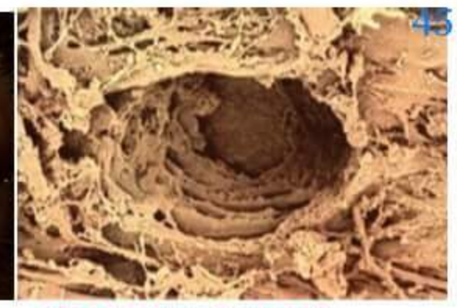
Glycerol

SURGICAL MANAGEMENT

ALT [ARGON LASER TRABECULOPLASTY]

TRABECULECTOMY

TRABECULECTOMY



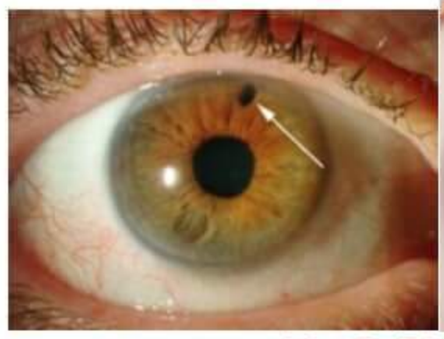
TRABECULOPLASTY

ACG MANAGEMENT

PERIPHERAL IRIDECTOMY

ND - YAG LASER IRIDOTOMY [Better]

→ other use → Rx of secondary cataract



LASER IRIDOTOMY

PI/LI are done in opposite eye [prophylactic]

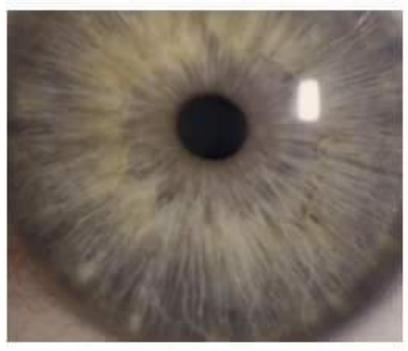
ACG Mx ALGORITHM

1. ↓ IOP → Mannitol [DOC]
   Acetazolamide
   Glycerol
   } systemic drugs

2. PROPHYLACTIC PI|LI OF SECOND EYE
3. LI OF ATTACKED EYE

SECONDARY GLAUCOMA

PIGMENTARY GLAUCOMA

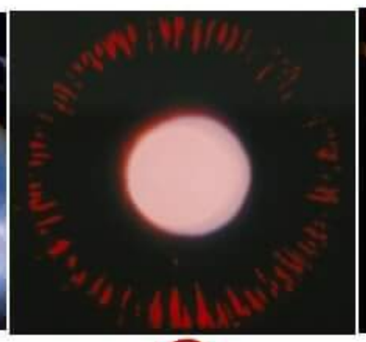


→ SEEN IN
  → young
    White Males
    Myopes

→ Concave Iris → touches zonules → Disperssion & Settlement of pigments on Iris
→ Exercised Induced glaucoma
→ DOC → MIOTICS [PILOCARPINE]

→ CLASSIC TRIAD
  1 Krukenberg spindle
  2 Radial transillumination iris defects
  3 Pigmentatⁿ of trabecular meshwork

③



②

①

→ Treatment → Pilocarpine or similar miotics

PSEUDO EXFOLIATION GLAUCOMA

→ MC secondary glaucoma worldwide
→ MC cause OF U/L glaucoma
→ Elderly Females, Scandinavian
→ Depositⁿ OF fibrillar material on TM → resistance to aqueous outflow → Glaucoma

→ SIGNS

↳ 3 RING / TARGET SIGN on anterior lens capsule

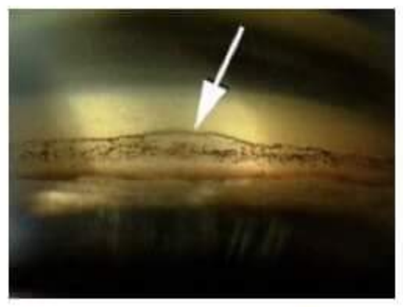
↳ pseudo exfoliat^{n} flakes on pupillary margin

↳ MOTH EATEN TRANSILLUMINAT^{n} DEFECT , close to pupillary margin

↳ SAMPADLESI'S LINE [line of pigment anterior to schwalbe's line]

3 ring | target sign

moth eaten defect



Sampaolesi's line

→ TREATMENT

→ done same as Open angle glaucoma [OAG]

→ Aqueous suppressants [BROMONIDINE]

→ But progression of glaucoma is much higher & faster than OAG

→ it is DIFFerent from True exfoliation [HEAT CATARACT]

MALIGNANT GLAUCOMA / POSTERIOR AQUEOUS MISDIRECT^{n} SYNDROME / CILIARY BLOCK GLAUCOMA

→ 2° glaucoma occuring post intraocular surgery characterised by

1. Shallow AC
2. elevated IOP
3. absence of pupillary block

→ Expansion of vitreous volume dlt sequestrat^{n} of aqueous posteriorly i resulting anterior shift in Lens Iris diaphragm

PATHOLOGY

→ For certain reasons the Aqueous humor goes posteriorly to Vitreous humor, instead of going anteriorly → POSTERIOR AQUEOUS MISDIRECT^{n} SYNDROME

→ creates POCKETS OF AQUEOUS HUMOR inside vitreous cavity

→ Expanded vitreous volume → Iris & Lens pushed upwards

→ AC becomes completely flat or shallow & the angle closes

→ ↑ IOP

→ MC in eyes with H/O angle closure undergoing glaucoma fitting Surgery

DIAGNOSIS → Shallow AC , absence of Pupillary block [Patent PI]

TREATMENT

1. CYCLOPLEGICS [ATROPINE]

↓

Paralyse the ciliary muscles

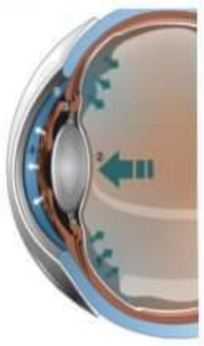
↓

zonular contraction

↓

Pulls the lens backwards

↓



↓

Deepening the AC

↓

Aqueous moves forward

↓

↓ IOP

Anti - Glaucoma drugs - miotics contraindicated

2. VITRECTOMY [Definitive treatment]

HYPERTENSIVE UVEITIS

→ Uveitis leading secondary glaucoma [not dlt HTN]

→ DOC → ATROPINE > STEROID

IN SECONDARY GLAUCOMA , WE TREAT THE PRIMARY CAUSE

NEOVASCULAR GLAUCOMA

→ Highest O_2 consumption in eye → Retina

↓

Hypoxia

↓

VEGF [vascular Endothelial Growth factor]

↓

NEOVASCULARIZATN

→ NEOVASCULARIZATN

↓

Retina

↓

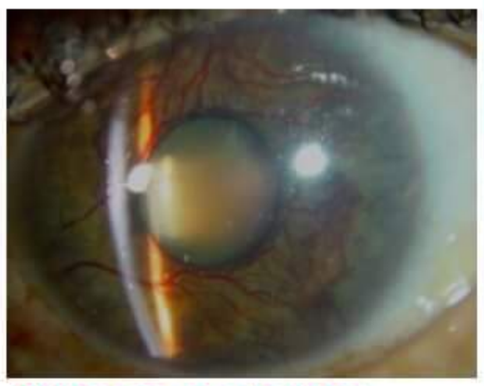
Vitreous

↓

IRIS [RUBEOSIS IRIDIS] [NEOVASCULAR IRIS (NVI)]

↓

NEOVASCULAR GLAUCOMA



RUBEOSIS IRIDIS

→ CAUSES

1 DM [mc, 30%]

2 CRVO

3 OCULAR ISCHEMIC DISEASE

→ Secondary angle closure glaucoma

→ Zipper - like closure

→ EARLY GLAUCOMA Mx

→ Stop neovascularizatn

→ ↓ IOP

→ STOP NEOVASCULARIZATION

1 ANTI - VEGF DRUGS

→ BEVACIZUMAB [AVASTIN]

→ RANIBIZUMAB [LUCENTIS]


Through pars plana

2 PAN RETINAL PHOTOCOAGULATION [except macula] For hypoxia


Pan Retinal coagulat

→ ↓ IOP

→ Latanoprost & pilocarpine are cI

→ Trabeculectomy

→ LATE GLAUCOMA

ABSOLUTE GLAUCOMA [patient start loosing vision]

→ IOP → 70 -80 mm Hg

→ can lead to glaucomatous optic atrophy

→ Pain +nt

→ TREATMENT

CYCLODESTRUCTION

→ Diode Laser cyclophotocoagulation [DLCP]

→ Cyclocryotherapy [temp of probe → -80°C]


ABSOLUTE GLAUCOMA

REFRACTION

EMMETROPIA → condition where the rays of light focus on the retina

AMMETROPIA

1. MYOPIA
2. HYPERMETROPIA / HYPEROPIA
3. ASTIGMATISM

MYOPIA / SHORTSIGHTEDNESS

→ condition where the rays of light focus infront of Retina

→ Eyes half closed → Myopic look

→ CAUSES

1. Axial Myopia
2. Curvature myopia
3. Index myopia

AXIAL MYOPIA

→ mc cause of myopia

→ axial length is longer than normal

↳ (N) → 24mm

↳ eye ball is longer than normal

→ For each 1mm of extra length of eye ball, 3D myopia occurs

Q Axial length of a patient who wears −21 D glasses

A

21/3 = 7 ⇒ 24 + 7 = 31 mm

→ Myopics wear the glasses close to the eyes

→ Hypermetropes wear convex lenses lower down on the nose

Q Hypermetropes wearing glasses & reading news paper. The glasses pushed down the nose. will the vision improve or worsen

A improves

CURVATURE MYOPIA

→ curvature of cornea is more

→ Ex: Keratoconus

INDEX MYOPIA

→ refractive index increases

→ Ex: Nuclear cataract

MYOPES ARE SHORTSIGTED PEOPLE (can't see long distance objects)

↳ Long distance rays are parallel & focus infront of retina

↳ Short distance rays are divergent rays, will focus on retina

Rx by CONCAVE / DIVERGENT / MINUS LENS

↳ Limitation → MINIFICATION OF IMAGES

↳ each 1D, minifies by 2%

HYPERMETROPIA / HYPEROPIA / LONGSIGHTEDNESS

→ rays of light focus behind the retina

→ CAUSES

1. Axial hypermetropia
2. Curvature hypermetropia
3. Index hypermetropia

AXIAL HYPERMETROPIA

→ mcc of hypermetropia

→ Axial length is smaller than the normal

→ For each 1 mm of shortening, cause 3 D of hypermetropia

CURVATURE HYPERMETROPIA

→ cornea is comparatively flat

→ Ex: congenital causes (rare)

INDEX HYPERMETROPIA → refractive index decreases

Q Refractive error of a new born child ?

Ⓐ All new borns are hypermetropic

Ⓝ refractive error at birth → 2-3 D of Hypermetropia

LONG SIGHTEDNESS

→ neither rays from Long distance nor the rays from short distance will focus on retina

→ But one can accomodates upto 16 D

↳ by this mechanism, rays from Long distance will focus on retina

↳ so, hypermetropes do not require convex lenses upto certain limit (out any visual disturbances

↳ but dlt excessive accomodation, ASTHENOPIA (eye strain) is the main problem in these patients

→ develops headaches & eye aches after 5-6 hrs of studying

→ uncorrected hypermetropes, as they are converging (synk-inesis) every time, will develop CONVERGENT SQUINT.

↳ also known as ACCOMMODATIVE SQUINT

→ Rx by CONVEX / CONVERGENT / PLUS LENS

↳ Limitation → Magnification

↳ every 1D, magnifies by 2%.

→ UNCORRECTED MYOPE, has very poor ciliary muscles (no accom-modation), may develop DIVERGENT SQUINT

MYOPIA	HYPERMETROPIA
→ rays of light focus infront of retina	→ rays of light focus behind the retina
→ can't able to seen distant vision	→ presents ī Asthenopia
→ corrected by concave lenses ↳ wear close to the eyes ↳ cause minificatⁿ	→ corrected by convex lenses ↳ wear away from the eye ↳ cause magnificatⁿ
→ poor accommodatⁿ	→ strong accommodatⁿ
→ uncorrected myopes develop DIVERGENT SQUINT	→ uncorrected hypermetropes develop CONVERGENT SQUINT

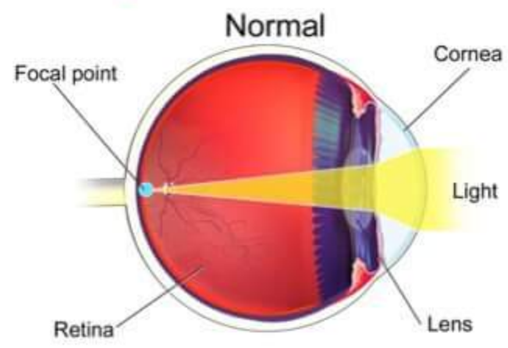
Together these are called as 'SPHERICAL ERRORS OF REFRACTION'

↳ Lenses used for correction are Spherical Lenses

↳ prescriptⁿ should include 's' (SPHERE)

→ Eg: − 2D S for myopes

\+ 2D S for hypermetropes



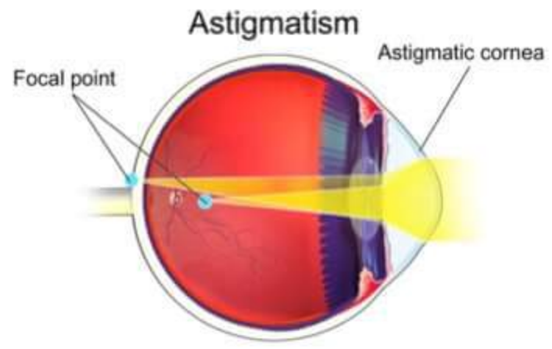
ASTIGMATISM

→ condition where there are 2 different foci are present

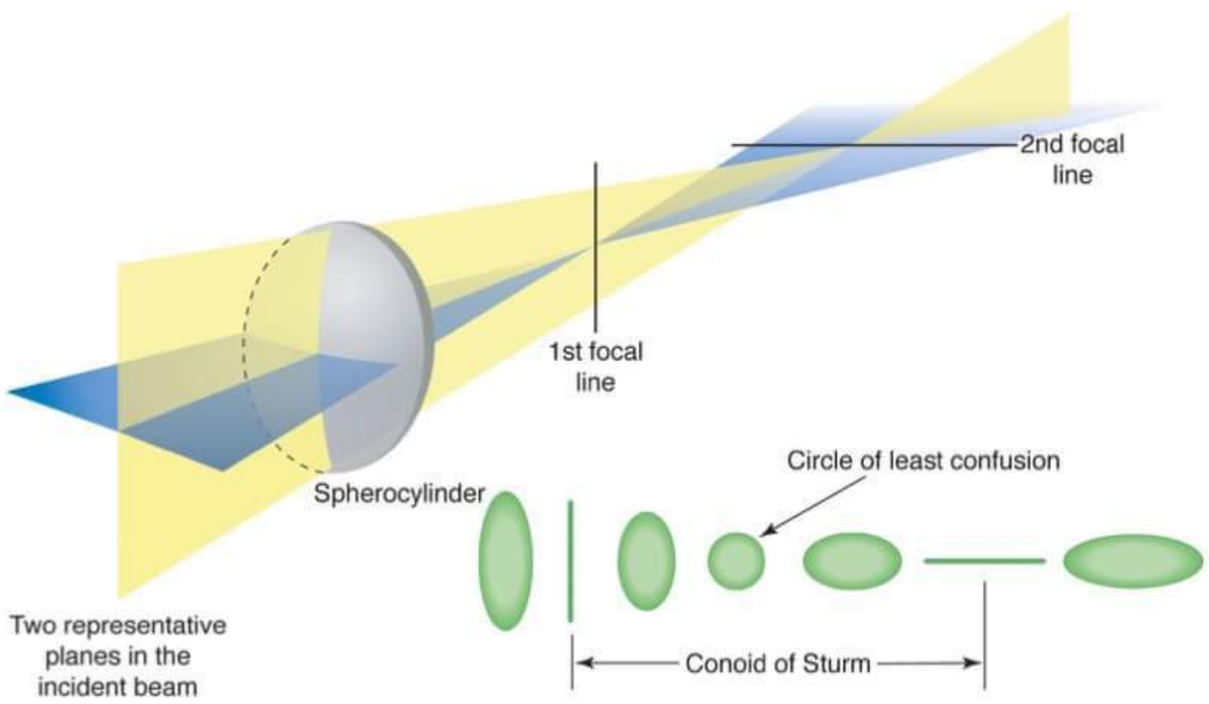
→ periphery of cornea is steeper than centre

↳ rays from periphery focus infront of the retina

↳ rays from centre focus behind the retina



Astigmatic cornea distorts the focal point of light in front of and/or behind the retina



STURM'S CONOID

↳ Interval blw the 2 foci

↳ CIRCLE OF LEAST CONFUSION

→ centre of Sturm's conoid

→ has the best visual acuity

→ This is the aim to place the retina

→ ASTIGMATISM IS THE MC REFRACTIVE ERROR

CORRECTION

→ done by CYLINDRICAL | TORIC LENS

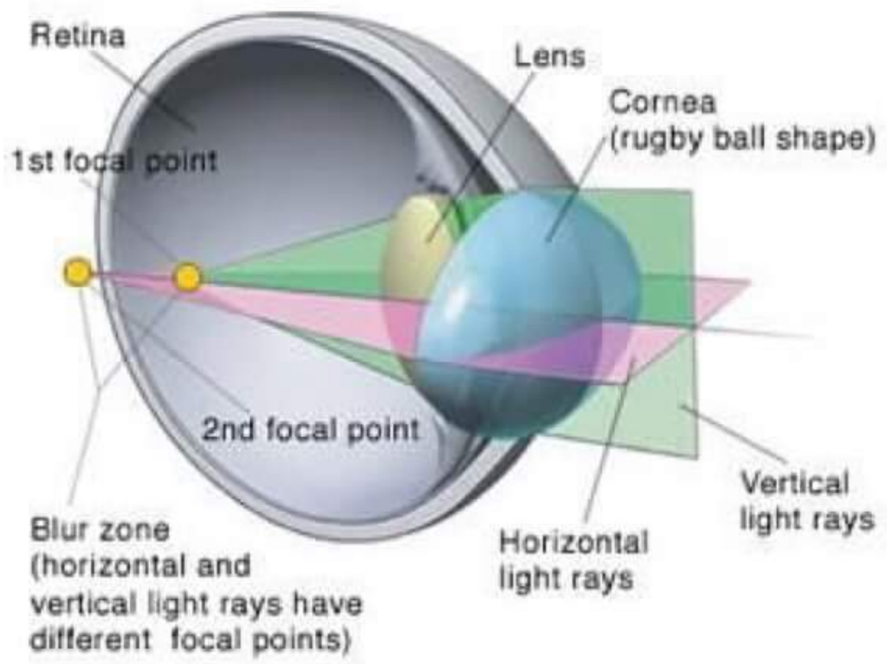
→ prescriptⁿ should be

± 2DC x 90°

C = cylindrical

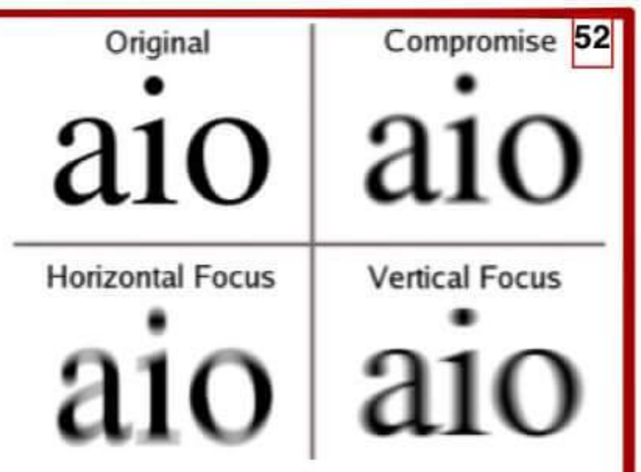
90° = indicates axis (1-180°)

CROSS SECTION OF ASTIGMATIC EYE



CASE

A → Myopic
B → Hyper metropic
C → Astigmatic

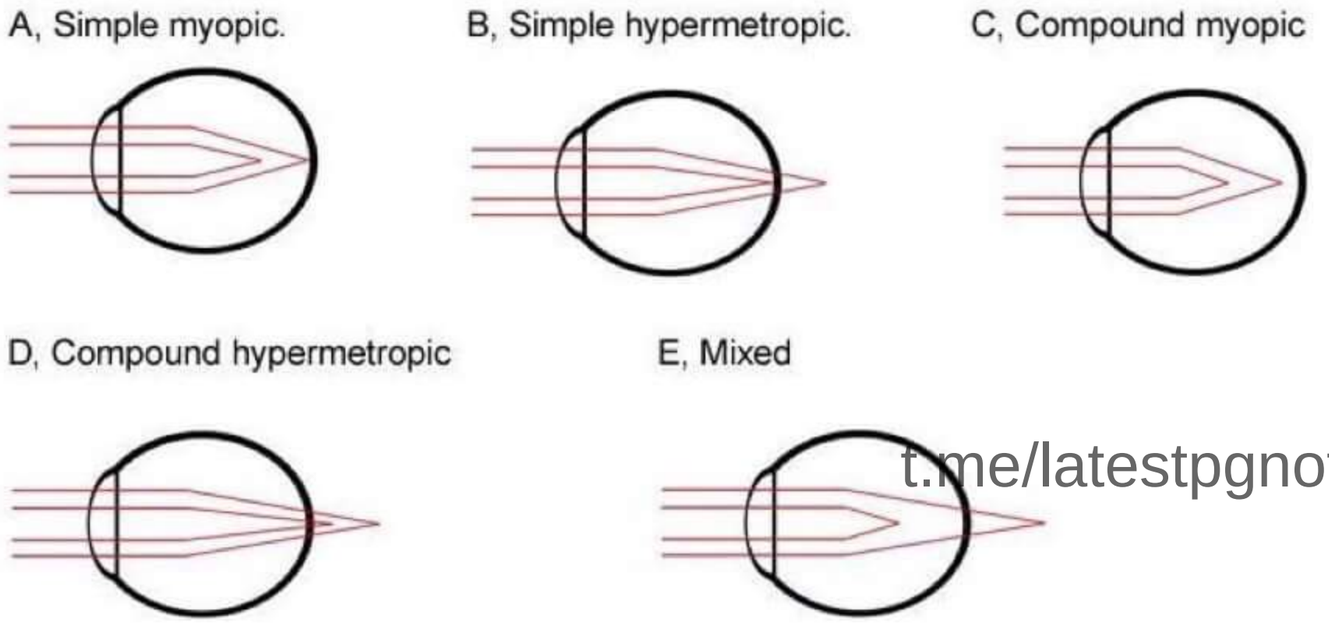


Ask them to Study letter 'A' on Snellen chart

A → Myopic → not able to study
B → Hyper metropic → able to study but will have headache, eye ache
C → Astigmatic → half the letter is clear & other half is hazy & will have headache & eye ache (max Asthenopia)

Minor amount of Astigmatism would create problems similar to those created by larger amount of hypermetropia
↳ ASTIGMATISM WILL HAVE MAXIMUM ASTHENOPIA

TYPES OF ASTIGMATISM

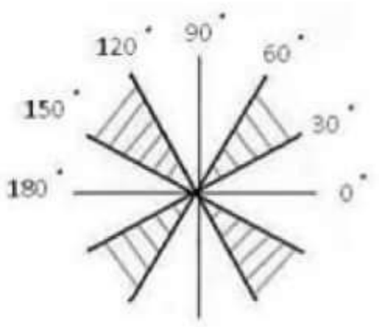


TYPES BASED OF SOURCE OF CONTRIBUTION

1. CORNEAL (mc & Significant)
2. LENTICULAR
3. RETINAL

CORNEAL ASTIGMATISM

1. Regular Astigmatism (common)
2. Oblique Astigmatism (less common)
3. Irregular Astigmatism
   → caused by irregular corneal surface
      ↳ corneal scar
      ↳ Keratoconus
   → difficult to treat

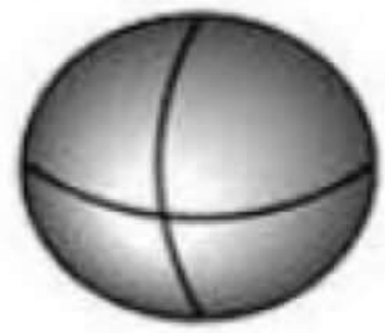


Oblique astigmatism (shaded)
Regular astigmatism

AXIS of ASTIGMATISM → (1-180°)
Oblique Astigmatism → 30°-60° or 120°-150°
Regular Astigmatism → outside the limits of Oblique astigmatism

REGULAR ASTIGMATISM

1. WITH THE RULE (WTR) → common ; Seen in young (<50yrs)
2. AGAINST THE RULE (ATR) → Seen in older (>50 yrs)



WTR Astigmatism

ATR Astigmatism

WTR

- → common
- → Seen in young (<50yrs)
- → has better visual acuity
- → depends on corneal shape
- → The vertical curvature > The horizontal curvature

ATR

- → Seen in elderly (>50 yrs)
- → Horizontal curvature > vertical curvature

WTR Astigmatism Corrected by → $+ C \times 90^\circ$ (or) $- C \times 180^\circ$

ATR Astigmatism corrected by → $- C \times 90^\circ$ (or) $+ C \times 180^\circ$

Q $-2DS / -3C \times 90^\circ$

A compound myopic / Regular / ATR Astigmatism

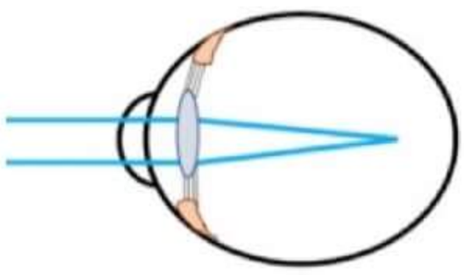
Q $+3DS / +2C \times 90^\circ$

A compound hypermetropic / Regular / WTR Astigmatism

Q $+5DS / -7C \times 180^\circ$

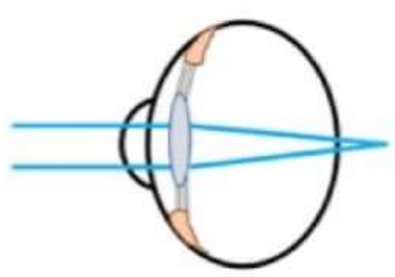
A Mixed / Regular / WTR Astigmatism

MYOPIA



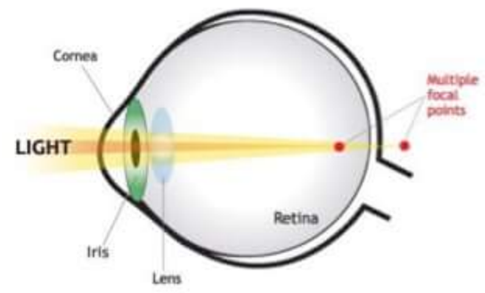
- → visual problem
- → R/ by concave lenses

HYPERMETROPIA



- → Asthenopia
- → R/ by convex lenses

ASTIGMATISM

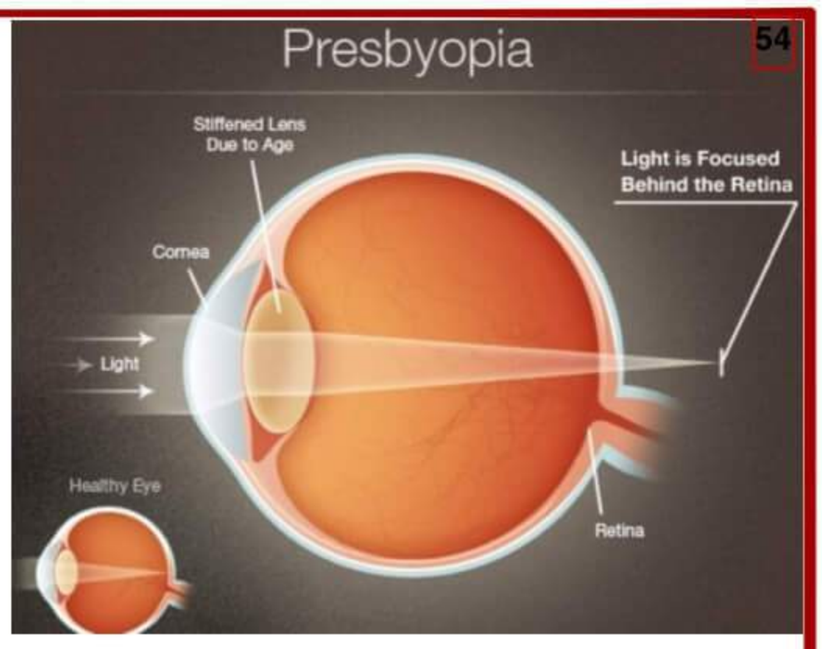


- → maximum asthenopia
- → R/ by Toric lenses

PRESBYOPIA

- → loss of accommodat^n ↓ age [40 - 45 yrs]
- → Physiological phenomenon
  - ↳ Normally, Lens power → 19 D
    Lens can able to accommodate additional 16 D of power
  - ↳ ↓ Age the accommodat^n power (ability to become spherical) decreases dlt
    1. hardening of lens dlt calcificat^n / mineralisat^n
    2. capsule becomes more fibrous
    3. Ciliary muscles become weaker
- → Difficulty in seeing near object

→ corrected by CONVEX LENS



DIFFERENTIATING FEATURES FROM HYPEROPIA

PRESBYOPIA

→ 40 - 45 yrs
→ difficulty for near vision (wears glasses)
→ distant vision is normal (no need of glasses)

HYPEROPIA

→ Wears glasses all the time

DARK ROOM PROCEDURES

→ In Dark rooms, there is
1. Less reflection from cornea
2. Pupil dilates more

1. RETINOSCOPY → done ī Retinoscope
2. DISTANT DIRECT OPHTHALMOSCOPY (DDO) } done ī direct ophthalmoscope
3. DIRECT OPHTHALMOSCOPY (DO) }
4. INDIRECT OPHTHALMOSCOPY → done ī Indirect ophthalmoscope

RETINOSCOPY

→ patient ī 6/60 vision

→ Check for
1. Optical Error
   a. Myopia
   b. Hypermetropia
   c. Astigmatism

2. Organic Error
   a. Glaucoma
   b. cataract
   c. Retinal detachment etc

PIN HOLE TEST

→ a completely opaque plastic disc ī a central hole [pin size ≅ 1mm diameter]
→ on applying pin hole test,
if vision improves → Optical Error
if vision do not improves → Organic Error

→ PIN HOLE allows only single Ray of Light.
Single ray of light passes through nodal point of eye (no distractn)

→ Pin hole test differentiates the optical & organic error
But it doesn't differentiates the type of optical Error

RETINOSCOPY

→ differentiate blw the types of refractive Errors

→ done ĩ RETINOSCOPE

↳ MISNOMER → checks the power, not the retina

↳ has 3 Parts

1. Head
2. neck
3. Body

Retinoscope

↳ contains a light source

↳ performed at 1 metre

↳ aka STREAK RETINOSCOPE

→ as reflected light from the retina appears like a streak of light.

→ reflected light appears at pupillary margin

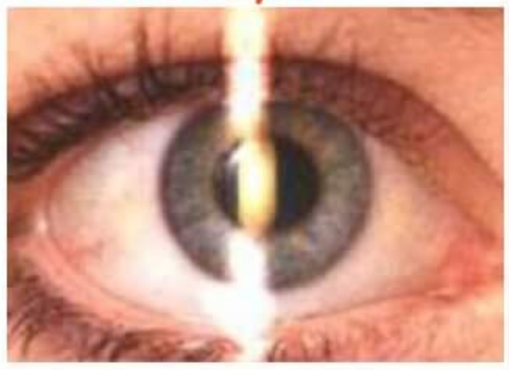
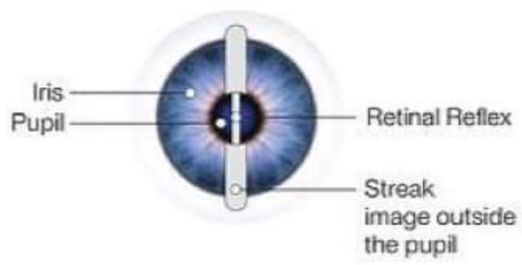


Fig. 1 A hand held instrument called a retinoscope projects a beam of light into the eye during a retinoscopy.

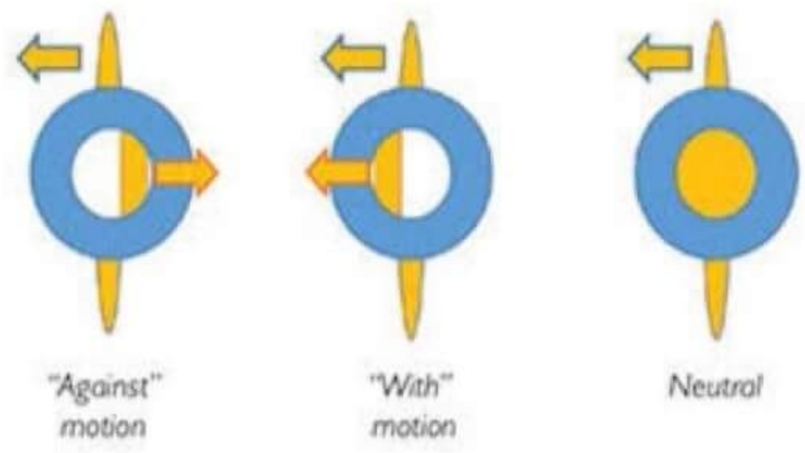
→ REFLECTION OF LIGHT ĩ RETINOSCOPE

1. Against / Opposite movement
   ↳ Myopia > −1



2. With / Same movement
   ↳ myopia < −1 (or)
   ↳ Emmetropia = 0 (or)
   ↳ Hypermetropia = + value

3. NO Movement
   ↳ myopia = −1



CALCULATION OF POWER OF LENS

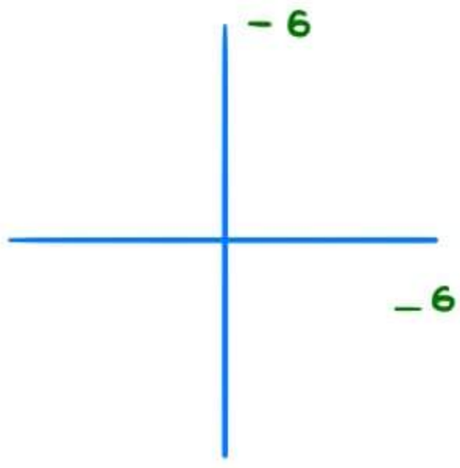
EXAMPLE

→ on Retinoscopy, if Retinal reflex has Opposite movement ĩ vertical movement of retinoscope

↳ it indicates power > −1

↳ Adjustment ĩ trial set done until the light fills the entire pupil ĩout any further movement → NEUTRAL

↳ Assume at −6D, neutral movement achieved



POWER CROSS

→ Same procedure done ĩ Retinoscope ĩ horizontal movement

↳ Assume at −6D, neutral movement achieved

→ Both values are plotted on 'POWER CROSS'

→ It indicates −6DS at 1 meter

TRIAL SET

→ To calculate the power at infinity → subtract the power that have been introduced into the eye by standing at 1m

$$P = \frac{1}{\text{Focal length}}$$

P = 1/1 = 1 D

Power at Infinity → −6 −1 = −7DS

Q Retinoscopy is −5D at 50 cm

A. P = 1/f = 2/1 = 2

Power at Infinity → −5 −2 = −7 DS

DISTANT DIRECT OPHTHALMOSCOPY (DDO)

→ done ī direct ophthalmoscope

→ contains light source & viewing window

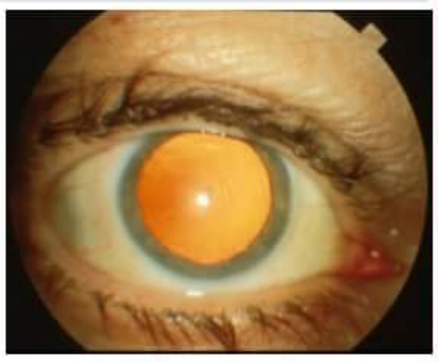
→ Reflected light is circular framed by pupillary window

→ distance at which DDO performed is → 25 cm

→ Normally 'RED GLOW' is seen

→ INTERPRETATIONS

1. Red Glow → healthy Retina
2. Grey Glow → Retinal detachment
3. NO Glow → Vitreous Haemorrhage
4. Black spot in center → cataract or corneal opacity



Normal Red Reflex

METHOD OF PARALLAX

DIFFERENTIATING cataract & corneal opacity

→ Ask the patient to look up,

in case of corneal opacity, blackspot moves up

in case of cataract, black spot moves down

→ Ask the patient to look down,

in case of corneal opacity, blackspot moves down

in case of cataract, black spot moves up

SAME SIDED MOVEMENT	→	CORNEAL OPACITY / SCAR
OPPOSITE SIDED MOVEMENT	→	CATARACT / LENTICULAR OPACITY

→ Gold Standard for Locating Subluxated lens → DDO

DIRECT OPHTHALMOSCOPY (DO)

→ done ī direct ophthalmoscope

→ distance at which DO performed at → VERY CLOSE BUT NOT FIXED

→ AIM → to See the 3 basic structures of Retina

↳ Optic disc

↳ macula

↳ fovea

→ Optical quality

↳ virtual, erect, magnified image

↳ 15 times magnified than the normal

→ 6° - 10° of retina can be visualized

INDIRECT OPHTHALMOSCOPY

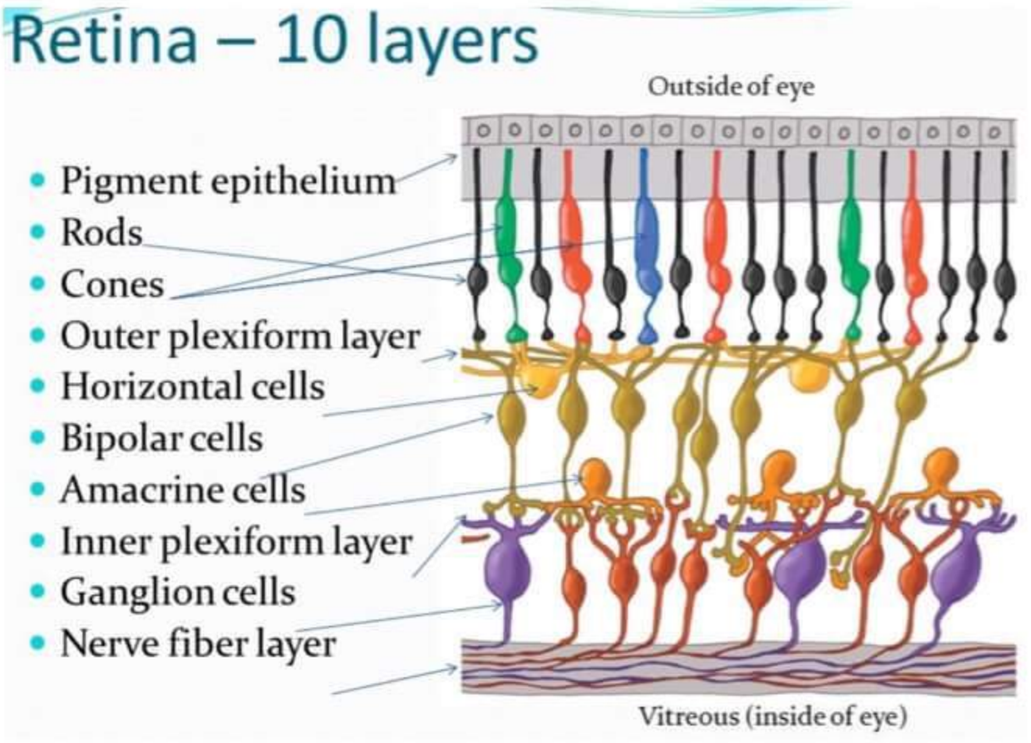
- → done i indirect ophthalmoscope
- → Position of patient → Lying down
- → distance of doctor → Arm length of the doctor
- → Power of holding lens → 20 D
- → Structures visualized
  - ↳ can be seen uptil Pars plana (farthest structure)
  - ↳ Pars plicata can't be seen

- → Quality of Image
  - ↳ Real Inverted Magnified Image
  - ↳ magnification → 3 to 5 times

TEN LAYERS OF RETINA

1st 9 Layers → NEURO SENSORY LAYERS [NSL]

10th Layer → RETINAL PIGMENT EPITHELIUM [RPE]



EXCEPTION → FOVEA, MACULA do not have 10 layers

BLOOD SUPPLY OF RETINA

DUAL BLOOD SUPPLY

→ Inner 2/3 rd → CRA [Central Retinal Artery]

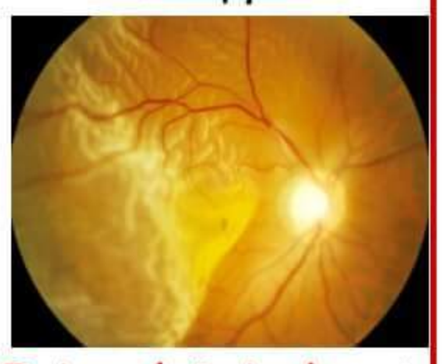
→ Outer 1/3 rd → PCA [Posterior Ciliary artery] [also supplies choroid]

WATERSHED LAYER → OPL [Outer plexiform layer] [Junct^{n} of 2 blood Supplies]

RETINAL DETACHMENT

→ Seperat^{n} of all 9 layers from RPE

RETINOSCHISIS → intra Neuro sensory layer Splitting



Retinal Detachment
[greenish glow]

PRESENTAT^{n}

→ Sudden painless loss of vision

→ FLOATERS / MUSCAE → Sign of vitreo retinal disorders

→ Flashes of lightning → PHOTOPSIAE

→ CURTAIN like sensat^{n} in front of eye

Shaffer's Sign

ON EXAMINATION

SLIT LAMP EXAMINATION

SHAFFER'S SIGN → TOBACCO DUSTING in vitreous

CLASSIFICATION

RHEGMATOGENOUS [mc] → Break in the retina

EXUDATIVE

TRACTIONAL

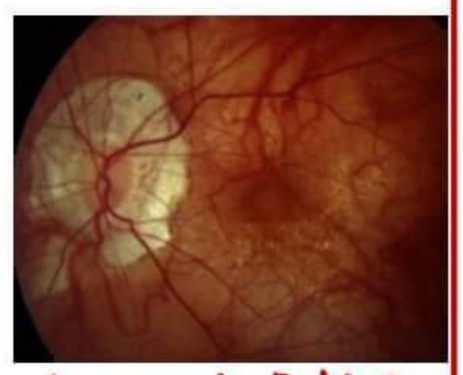
RHEGMATOGENOUS DETACHMENT

RISK FACTORS

1 Myopia
2 Post cataract surgery
3 Trauma

MYOPIA

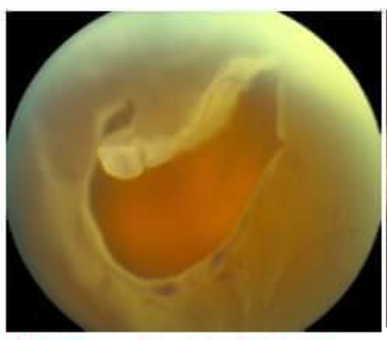
→ Long Eye Ball
↓
Strech on Retina
↓
Become Thinner

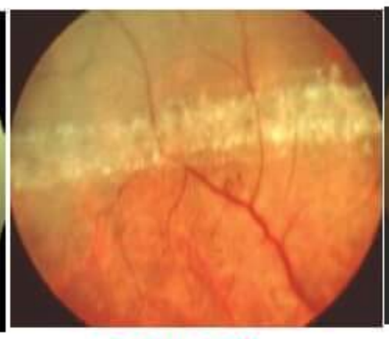

High myopic Retina


High myopia

→ Myopia α Detachment
→ > - 8D → High chance
> - 12D → very High chance

→ LESIONS CAUSING DETACHMENT
1. HOLES
2. LATTICES
3 HORSE SHOE TEAR

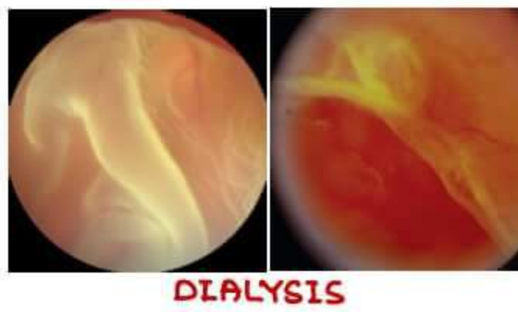

Horse Shoe Tear


Lattices


Holes

→ max detachment takes place → at Supero Temporal quadrant [40%]

APHAKIA → Tamponade effect of Lens is lost
TRAUMA → DIALYSIS [separatⁿ from root]


DIALYSIS


APHAKIA

WATERMARKS IN CHRONIC RD

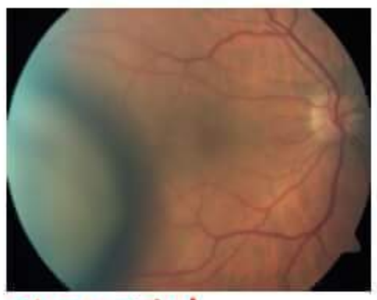
→ Hyperpigmented lines in retina caused by depositⁿ of pigmented epithelium in chronic RD
→ sign of long standing Retinal Detachment

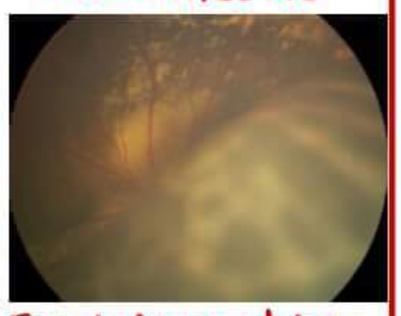

Watermarks

EXUDATIVE RETINAL DETACHMENT

→ No hole, no break, no lattice
→ CAUSES

1 MALIGNANT MELANOMA OF CHOROID
→ commonest tumor of eye in adult
→ commonest tumor of eye in children
→ Retinoblastoma


Choroidal melanoma


Exudative detachment


Exudative detachment dlt PIH

2 PREGNANCY INDUCED HTN / GESTATIONAL HTN
→ BP > 140|90 after 20 wks of POG

3 CHOROIDITIS

4 MALIGNANT HTN [HTN + Papilledema]

5 VKH SYNDROME [VOGT KOYANAGI HARADA SYNDROME]

→ BIL granulomatous Pan Uveitis

→ Seen in Japan

→ PRESENTATION

→ Young lady i Viral fever

→ N → NEURDLOGICAL SYMPTOMS

Headache

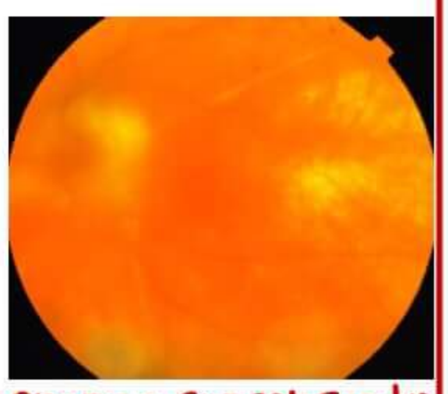
Neck stiffness

Auditory Symptoms [mc]

→ Tinnitus

→ Vertigo

→ Deafness



Orange sunset Fundus

O → OPHTHALMIC SYMPTOMS

→ BIL Granulomatous Panuveitis

→ BIL Exudative Retinal detachment

→ ORANGE SUN SET FUNDUS

D → DERMATOLOGICAL SYMPTOMS

→ Poliosis [whitening of eye lashes]

→ Vitiligo

→ alopecia

TRACTIONAL RETINAL DETACHMENT

CAUSES

1 DM [mc]

2 PROLIFERATIVE VITREORETINOPATHY

3 SICKLE CELL ANEMIA

4 RETINOPATHY OF PREMATURITY

→ Slow painless loss of vision

Tractional Retinal Detachment

RETINAL DETACHMENT TREATMENT

→ RPE provides nourishment for NSL

→ In 48-72 hrs, Photo receptors [rods & cones] starts dying

RHEGMATOGENOUS DETACHMENT

1 DRAIN THE FLUID

2 Close holes, lattices & tears by PHOTOCOAGULATION by laser beam [60-80°c]

→ causes chorioRetinal adhesn when NSL & RPE in apposition

3 In some cases, TAMPONADE on NSL is required

Tamponade by

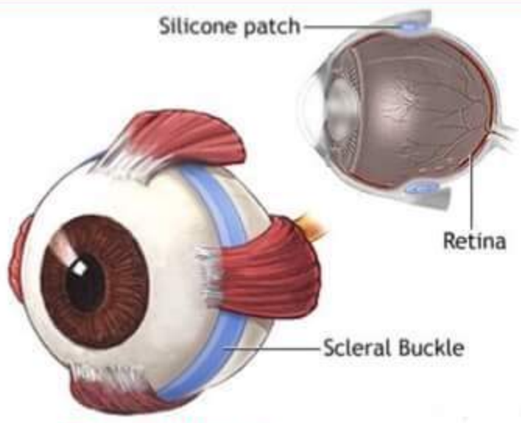
→ Inj SILICON OIL through Pars Plana

EXPANSILE GASES

Air

SF6 [Sulfur Hexa Fluoride]

C_3F_8 [Perfluoro Propane]



Buckling

Gas Bubble

4 BUCKLING [Silicon patch on Retina]

EXUDATIVE DETACHMENT → Treat underlying cause

TRACTIONAL DETACHMENT → VITRECTOMY

DIABETIC RETINOPATHY

→ Diabetic capital of the world [WHO] → India [63 Million diabetics]

RISK FACTORS

→ Duration → most important prognostic factor

→ Poor control of Blood glucose

→ Hypertension

→ Pregnancy

TIMING FOR CHECKUP

→ 5yrs from detectⁿ → Type 1/IDDM

→ Immediately from detectⁿ → Type 2/ NIDDM

THEN ONCE EVERY YEAR for Both

CLINICAL FEATURES

SYMPTOMS

1. BLURRED VISION
2. **FLUCTUATING VISION**
3. IMPAIRED COLOR VISION
4. FLOATERS

SIGNS

→ Earliest manifestation → Micro aneurysms

→ Hallmark → Neovascularizatⁿ

PATHOLOGY

1 OCCLUSION

2 LEAKAGE

OCCLUSION

→ dlt Thromboembolisms

→ Diabetics suffer 5 times more MI attacks than Ⓝ people

occlusion

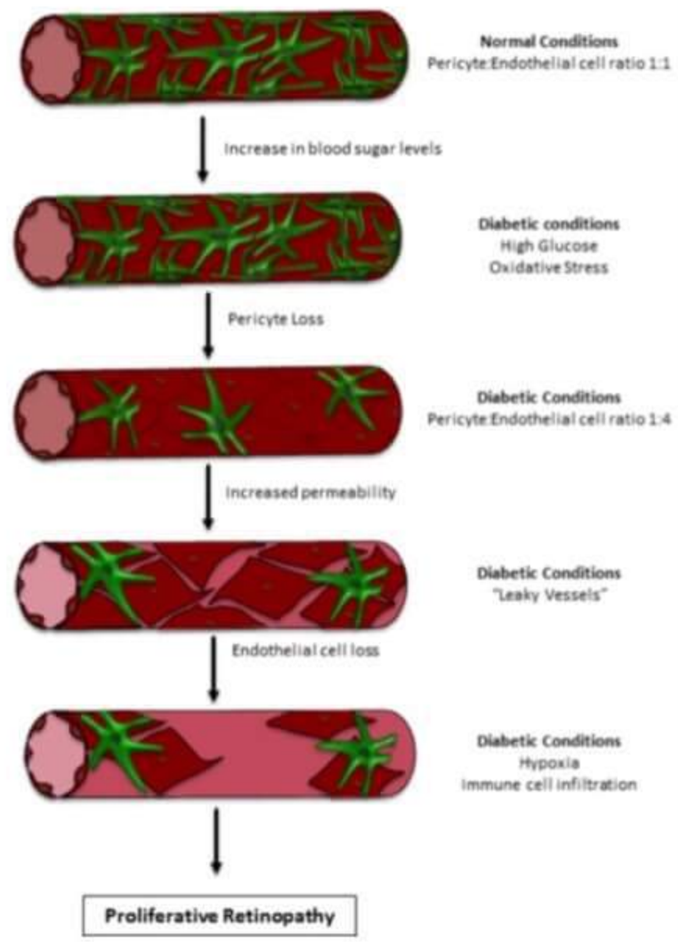
OCCLUSION
↓
HYpoxia
↓
VEGF
↓
NEOVASCULARIZATⁿ

LEAKAGE

→ dlt PERICYTE NECROSIS [also seen in TAKAYASU'S ARTERITIS]

Normal Pericyte : Endothelium ratio → 1:1

Qn Pericyte necrosis, P : E ratio → 1:4

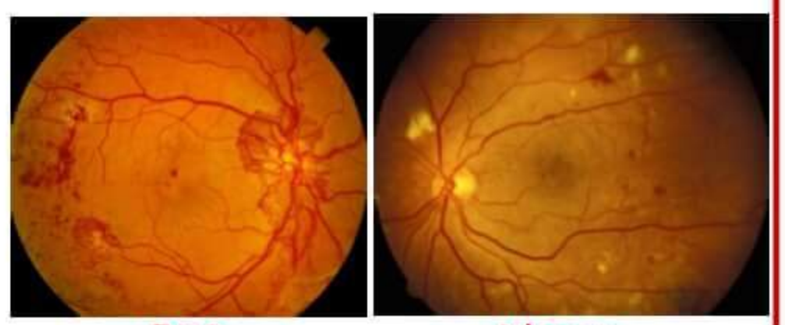


Pressure
↓
Ballooning out
↓
MicroAneurysms
↓
LEAKAGE

CLASSIFICATION

1 NPDR [Non Proliferative Diabetic Retinopathy] [<20yr]

2 PDR [Proliferative Diabetic Retinopathy] [>20yrs]



PDR

NPDR

ⓘ NPDR [NON PROLIFERATIVE DIABETIC RETINOPATHY]

1. MICROANEURYSMS [earliest manifestatⁿ]
2. DOT & BLOT HAEMORRHAGES
3. HARD EXUDATES
   - → Lipid deposits on retina dlt leakage
   - → present in outerplexiform layer
4. SOFT EXUDATES / COTTON WOOL SPOTS
   - → Axoplasmic debris dlt occlusion
   - → present in Nerve fibre layer

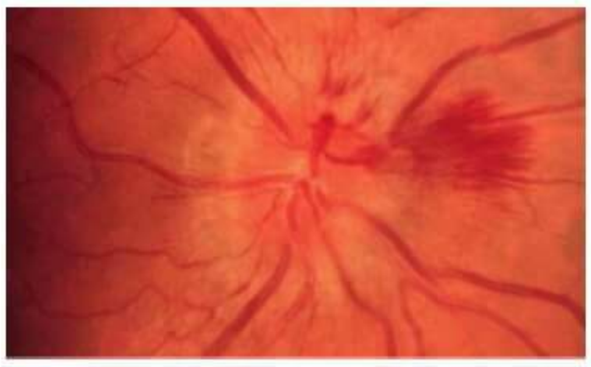
Dot & Blot Haemorrhages

microaneurysms

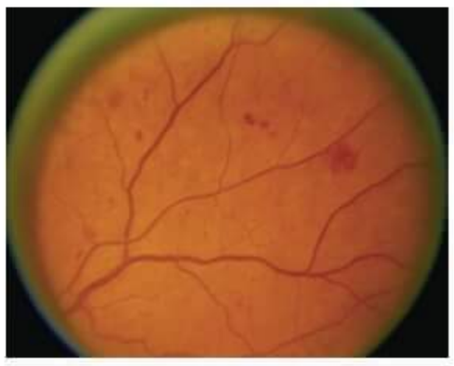
soft exudates

hard exudates

SUPERFICIAL HAEMORRHAGES	DEEP HAEMORRHAGES
Common in Hypertensive Retinopathy	Common in Diabetic retinopathy
FLAME SHAPED Haemorrhages	Dot & Blot Haemorrhages
present in Nerve fibre layer	present in Inner nuclear layer



Flame shaped Haemorrhage



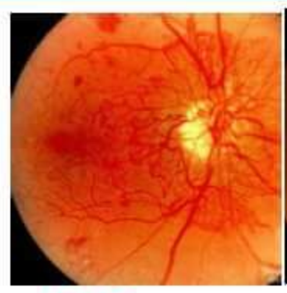
Dot & Blot Haemorrhag

	LOCATION
Micro Aneurysms	Inner nuclear Layer
Hard Exudates	outer plexiform layer
Soft Exudates	Nerve fibre layer
Flamed Shaped Haemorrhages	Nerve fibre layer
Dot & Blot Haemorrhages	Inner nuclear layer

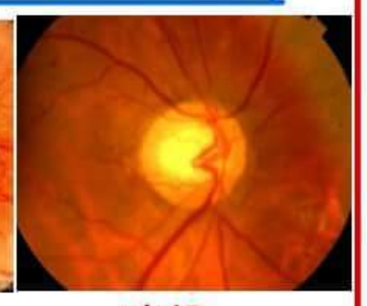
⑪ PDR [PROLIFERATIVE DIABETIC RETINOPATHY]

1 NVD → Neo vascularization of Disc

2 NVE → Neo vascularization elsewhere



NVE



NVD

VISUAL LOSS IN DR

① NPDR

1. MACULAR EDEMA → CSmE [clinically significant Macular Edema]

Macular edema in 500μ radius from fovea

CSME

② PDR

1 VITREOUS HAEMORRHAGE

2 TRACTIONAL RD

3 NEOVASCULAR GLAUCOMA

Commonest cause of visual loss overall → Macular Edema

Commonest cause of visual loss in NPDR → Macular Edema

Commonest cause of visual loss in PDR → Vitreous Haemorrhage

VITREOUS HAEMMORRHAGE

CLINICAL FEATURES

→ FLOATERS

→ SUDDEN PAINLESS LOSS OF VISION

VITREOUS HAEMORRHAGE	RETINAL DETACHMENT
Floater ⊕	Floater ⊕
Sudden painless loss of vision ⊕	Sudden painless loss of vision ⊕
Flashes ⊖	Flashes ⊕

CAUSES

1 DIABETIC RETINOPATHY [mcc]

2 TRAUMA [mcc in young]

3 EALES DISEASE

→ Periphlebitis retinae

→ young males

→ Spontaneous vitreous Haemorrhage

→ Recurrent

→ a/w TB

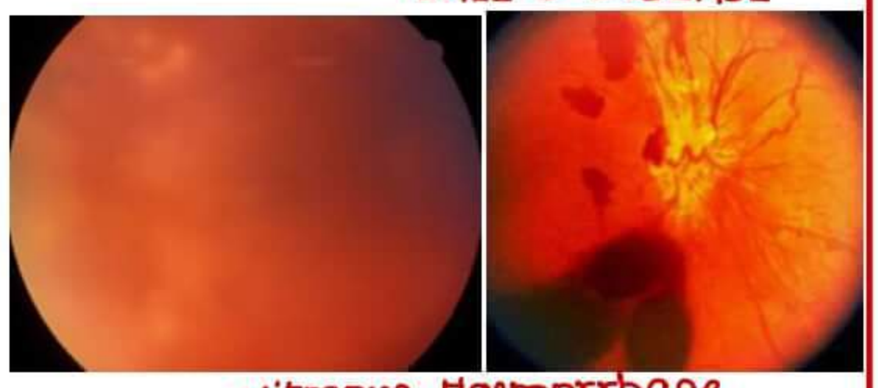
→ STAGES

1 INFLAMMATION OF VEINS

2 OCCLUSION / ISCHEMIA

3 NEOVASCULARIZATION

EALE'S DISEASE



vitreous Haemorrhage

→ TREATMENT

1 Systemic steroid

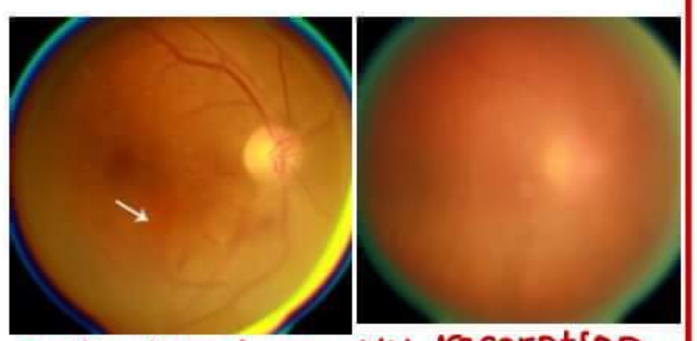
2 ATT

TREATMENT

1 BED REST for atleast 3 months

2 TREAT UNDERLYING CAUSE

3 VITRECTOMY [after 3 months]



post vitrectomy

VH resorption to some extent

DIABETIC RETINOPATHY TREATMENT

NPDR

DIABETIC MACULAR EDEMA

1 LASER PHOTOCOAGULATION

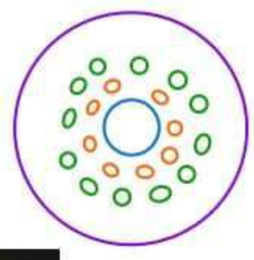
→ DF - ND YAG LASER [DF - Double frequency]

→ wavelength → 1064/2 → 532 nm

→ Green laser [applied peripherally]

→ 60-80°C heat used [absorbs edema]

Diabetic Macular edema



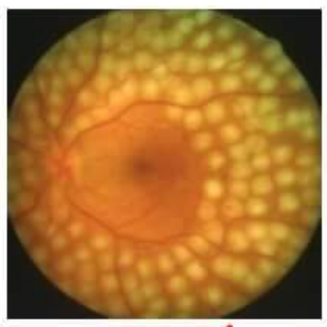
Optic Disc
Laser
Edema
Fovea

Focal laser

2 ANTI VEGF DRUGS

BEVACIZUMAB

RANIBIZUMAB



Pan retinal coagulation

3 INTRA VITREAL STEROIDS

TRIAMCINOLONE

OZURDEX [Implantable Dexamethasone]

PDR

1 RETINAL PHOTOCOAGULATION

→ Focal

→ 60-80°c seal blood vessels

→ Recurrence is common

2 PAN RETINAL COAGULATION

→ Entire retina except macula

→ Hypoxia eliminated

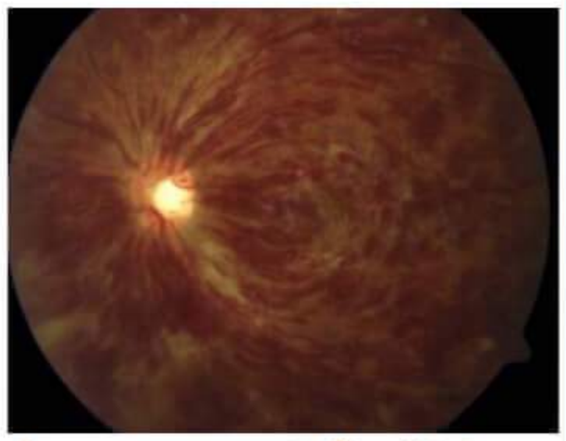
CRVO (CENTRAL RETINAL VENOUS OCCLUSION)

CAUSES

1. older people (>60yr)
2. uncontrolled HTN
3. uncontrolled DM
4. Young women taking OCPs

PATHOGENESIS

→ central Retinal artery & vein share common adventitious sheath
Thickened artery compresses Retinal vein in the common adventitious sheath close to the lamina cribrosa


SPLASH TOMATO FUNDUS

→ causes sudden painless loss of vision
  ↳ sudden dimming of lights
  ↳ sudden drop in voltage (partial loss)

→ OIE
  → Entire retina is full of flame shaped Haemorrhages
    ↳ TOMATO KETHUP / SPLASH TOMATO / BLOOD & THUNDER FUNDUS
  → soft exudates
  → veins are dilated & tortuous
  → disc edema

→ CRVO
↓
HYPOXIA
↓
VEGF
↓
NEOVASCULARIZATN
↓
NEOVASCULAR GLAUCOMA [90/100 Day Glaucoma]

→ mcc of loss of vision → Macular edema

→ TYPES
1. ISCHEMIC CRVO (25%)
2. NON ISCHEMIC CRVO (75%)

TREATMENT

1. control BP & Diabetes
2. Control IOP ± anti-glaucoma drugs
3. Wait & Watch till the haemorrhage absorbs (upto 6 months)
4. Macular edema Rx by
   ↳ Intra vitreal anti VEGF drugs
   Intra vitreal steroids (IVTA - Intravitreal Triamcinelone Acetonide (or OZURDEX)
5. Neovascular Glaucoma Rx by Anti VEGF DRUGS
   ↳ BEVACIZUMAB
   RANIBIZUMAB

CRAO (CENTRAL RETINAL ARTERIAL OCCLUSION)

RISK FACTORS

1. Older people
2. Uncontrolled HTN
3. Uncontrolled Dm

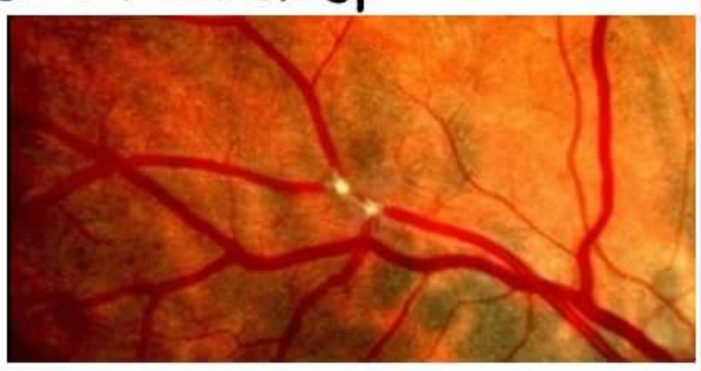
PATHOGENESIS

Carotid Atherosclerosis → Embolus → CRAO

SITES

→ Narrowest Site of CRA , Where it pierces the dural sheath of optic nerve

→ Posterior to lamina cribrosa



Hollenhorst plaque

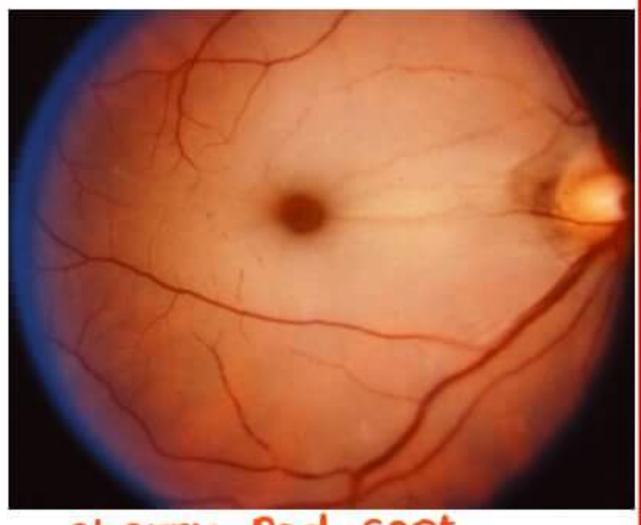
→ Embolism is the mc cause
  ↳ HOLLENHORST PLAQUE (cholesterol plaque)

CLINICAL FEATURES

→ Sudden painless loss of vision
  ↳ Complete switching off of lights (complete loss)

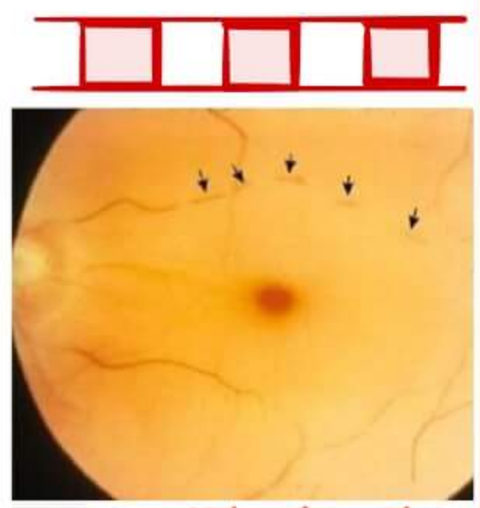
O/E

→ Cherry Red Spot from fovea
  Surrounded by white retina



cherry Red Spot

CHERRY RED SPOT also seen in			
Cherry	→	CRAO	Acute UIL
Trees	→	Trauma	
Never	→	Neimann pick	Chronic BIL
Grow	→	Gaucher's	
Tall in	→	Tay Sach's	
Sand	→	Sandhoff	



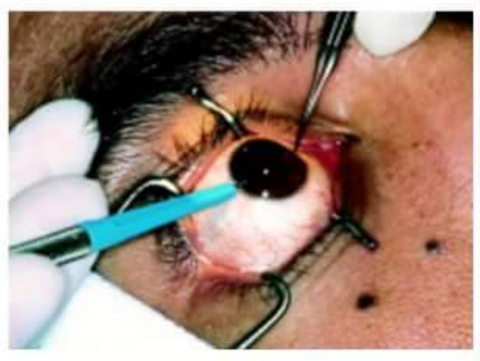
cattle truck appearance

→ CATTLE - TRUCK | BOX CAR APPEARANCE
  ↳ discontinuity in blood column
  ↳ slow & jerky movement of blood column

→ RETINAL ISCHEMIC TIME → 90 minutes (complete recovery can be done)
→ Partial Recovery done by → 240 minutes
→ Irreversible → after 24 hrs

TREATMENT

1. Ocular Massage
2. Immediate lowering OF IOP by Mannitol, Acetazolamide
3. Paracentesis → Aspiration of aqueous from AC
4. Carbogen → CO_2 (5%) + O_2 (95%) inhalation
5. IV Heparin , Tissue plasminogen activator
  ↳ may cause haemorrhage

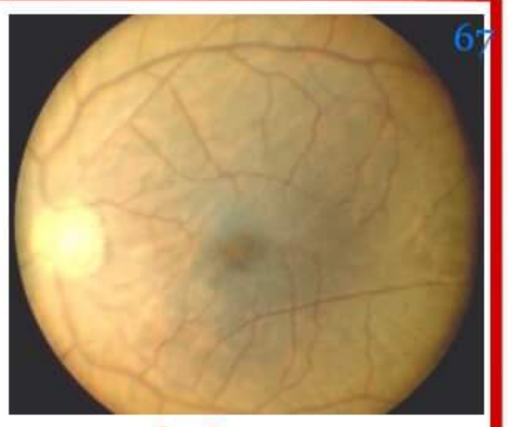


Paracentesis

Ocular Perfusion Pressure = Mean Blood Pressure − IOP

CYSTOID MACULAR EDEMA

→ outer plexiform layer has maximum edema
→ causes slow painless loss of vision



CME

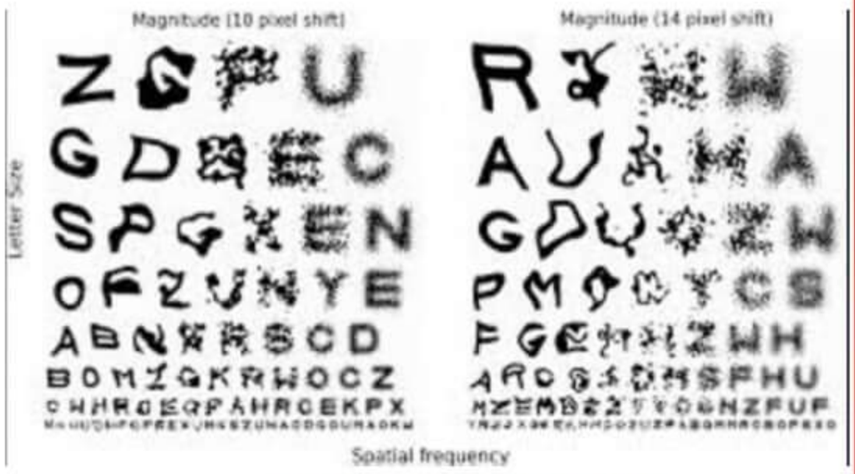
CAUSES

- **D**iabetes
- **E**pinephrine
- **P**ars planitis
- **R**etinitis pigmentosa
- **I**rvin gas syndrome [CME after cataract S_x]
- **V**ascular occlusions [CRVO]
- **E_2** Prostaglandins
- **N**icotinic Acid
- **S**urgical [post S_x]
  - ↳ Retinal detachment S_x
  - ↳ Glaucoma S_x
  - ↳ Keratoplasty

→ more common causes
  - ↳ DM
  - ↳ Irvin Gas Syndrome

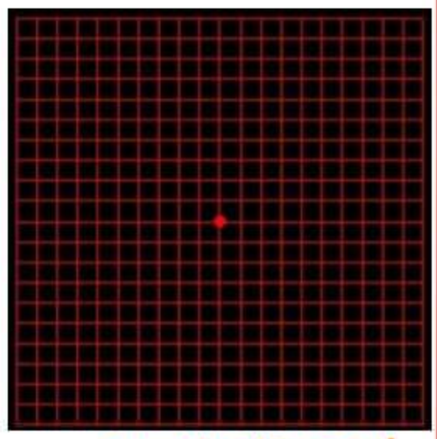
CIF

→ Slow painless loss of vision
→ METAMORPHOPSIA
  - ↳ distortn of shape
  - ↳ occurs in all macular disorders
  - ↳ FORMS
    - a. MICROPSIA → Objects are small & distorted
    - b. MACROPSIA → Objects are large & distorted



Metamorphopsia

  - ↳ Diagnosed by AMSLER'S GRID
    - → Give the patient Amsler's Grid & ask him to trace it
    - → The tracing is distorted
    - → can pick very small amounts of metamorphopsia



Amsler'S Grid

→ Best instrument to examine Macula → + 90 D lens

+ 90 D

INVESTIGATIONS

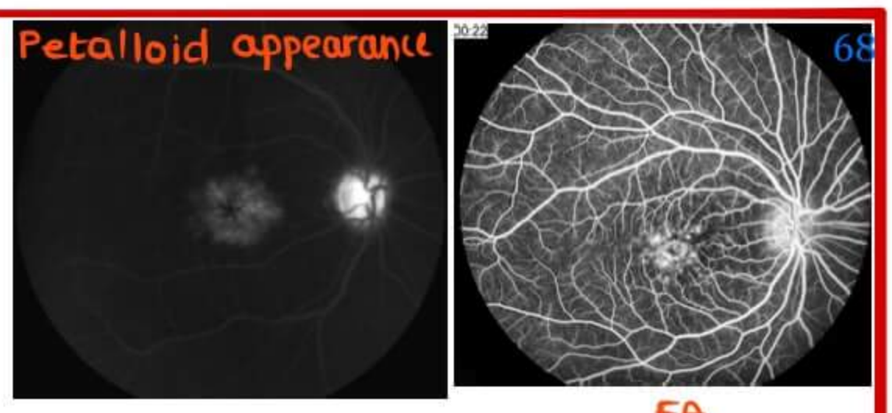
→ can be used in any retinal disorders

1. Fluorescein Angiography
2. OCT

ANGIOGRAPHY OF EYE
  - ↳ Dyes used
    - FA → For retina
    - ICG → For Choroid (Indocyanine Green)

FLUORESCEIN ANGIOGRAPHY

→ Dye used → Sodium fluorescein
→ injected into Ante - cubital vein
↳ reaches retina approx. in 11 sec



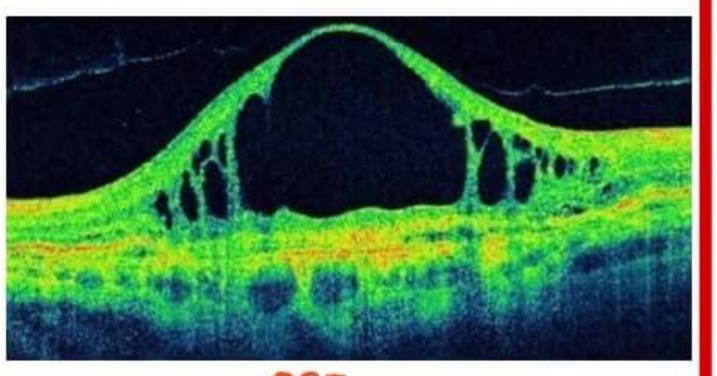
FA

→ In case of no pathology → excreted through urine
→ In case of pathology → Dye leaks into Surrounding Retina
→ leak is recorded by FUNDUS CAMERA
→ pattern of leak tells us the pathology
↳ CME → PETALLOID APPEARANCE (Confirms CME)

→ LIMITATIONS → invasive & may cause allergy

OCT [OPTICAL COHERENCE TOMOGRAPHY]

→ Gold standard investigat^{n} for all macular disorders
→ non invasive
→ cross section of Retina can be studied

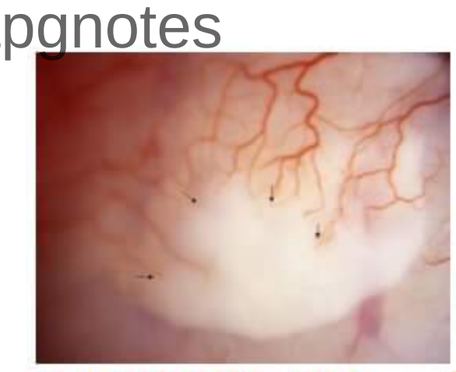

OCT

ICG [Indocyanine Green]

→ Indications
1. Polypoidal choroidal vasculopathy (PCV) → Best Indicat^{n}
2. Occult CNVM [Choroid Neo vascular membrane] → mc Indicat^{n}

TREATMENT

1. TOPICAL STEROIDS [SIE → Glaucoma]
2. TOPICAL NSAIDS
↳ BREMFENAC / NEPAFENAC
3. Subtenon Inj. of TRIAMCINELONE


Subtenon's Inj

Topical NSAIDs Topical Steroid

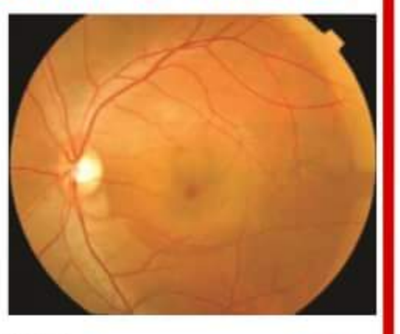
BERLIN'S EDEMA / COMMOTIO RETINAE

→ Post traumatic macular edema
→ H/O blunt trauma ⊕ nt
→ OIE → cherry Red Spot ⊕
→ R/OC → Systemic Steroids


Berlin's edema

CSR (CENTRAL SEROUS RETINOPATHY)

→ Fluid collected under macula in both CSR & CME
→ Difference b/w CSR & CME
↳ CSR → edema present b/w Neurosensory layer & RPE
↳ CME → edema present in outer plexiform layer


CSR

→ occurs in young male executive type A personality
→ causes Sudden painless loss of vision
→ Metamorphopsia + nt
↳ quantified by Amsler's grid
→ positive Scotoma in centre


positive Scotoma

→ FA shows INK BLOTS & SMOKE STACKS (confirms CSR)

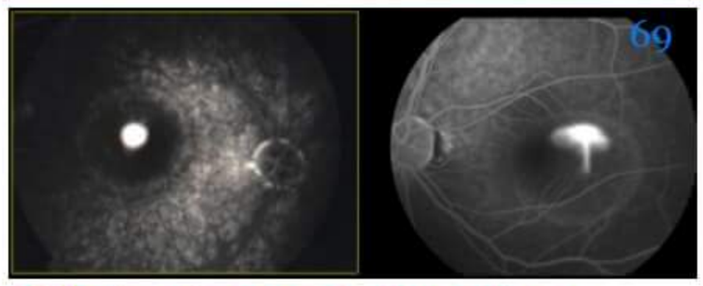
→ Rx → Recover Spontaneously (↓ in 3 months)

→ Supportive therapy

↳ Reassure

↳ anxiolytics

↳ Placebos



Smoke stacks & Ink Blots

MACULAR HOLE

→ RETINAL BREAK, commonly involving the FOVEA

→ MC CAUSE → Idiopathic

→ RISK FACTOR → old age, females, myopia, trauma

→ SYMPTOMS → Centra visual loss, metamorphosia, central scotoma

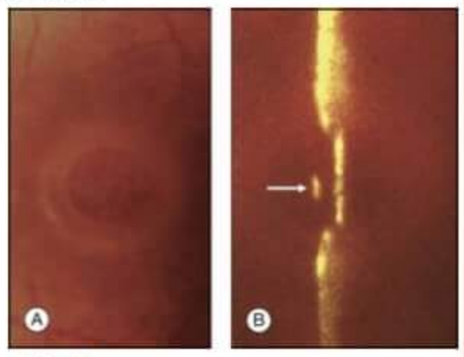
→ LIPOFUSCIN colour spot / ring ⊕

→ WATZKEE ALLEN SIGN ⊕

→ TREATMENT

1. Spontaneous closure occurs [conservative management]
2. vitrectomy ± tamponade
3. Ocripalmin injection in vitreous


Lipofscin spotl ring



Watzke Allen Sign

ROP [RETINOPATHY OF PREMATURITY]

→ occurs in Premature children

→ and mcc of loss of vision in children (1st → vit. A deficiency)

RISK FACTORS

1. Prematurity (< 32 weeks)
2. Poor birth weight (< 1500 gms)
3. Excess O_2 Supplementation

PATHOGENESIS

Full Term Normal child's retina is completely vascular

Pre term child's retina vascularity not complete, extend only upto ora serrata

↳ at birth, child is exposed to hyperoxia

↳ hyperoxia causes vasoconstriction

↳ leads to vaso obliteration

↳ peripheral retina do not develop blood vessels

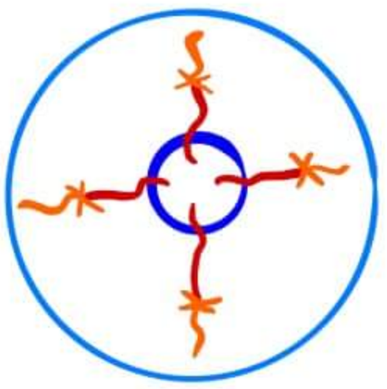
↳ leads to hypoxia

↳ VEGF produced

↳ Neovasularization occurs at vascular & Avascular junct^{n} of retina

↳ New blood vessels contract [dlt fibrous tissue] & pull the Retina from RPE

↳ Loss of vision



LOSS OF VISION is not dlt ROP perse but is dlt TRACTIONAL RETINAL DETACHMENT

OXYGEN SATURATION TARGETS

→ full term healthy baby → 95 - 100 %

→ Pre term baby → 85 - 93 %

SCREENING RECOMMENDATIONS

→ According to AAP (American Academy of Pediatrics) → 4 - 6 Weeks

→ According to IAP (Indian Academy of Pediatrics) → 3 - 4 weeks

STAGES OF ROP

STAGE 1 : DEMARCATION LINE [line where the normal & abnormal vessels meet)

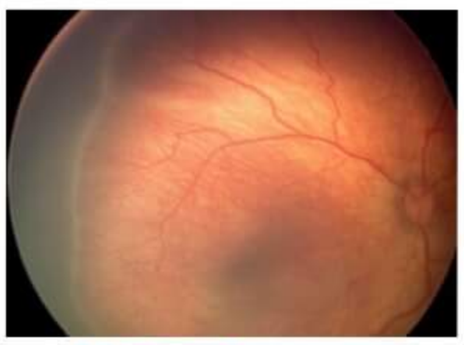
STAGE 2 : DEMARCATION RIDGE [Ridge that rises up from the retina as a result of growth of the abnormal vessels]

STAGE 3 : RIDGE ī EXTRA RETINAL FIBROVASCULAR PROLIFERATION [The ridge grows from the spread of the abnormal vessels & extends into the vitreous]

STAGE 4 : SUB TOTAL RETINAL DETACHMENT [partial detachment of Retina]

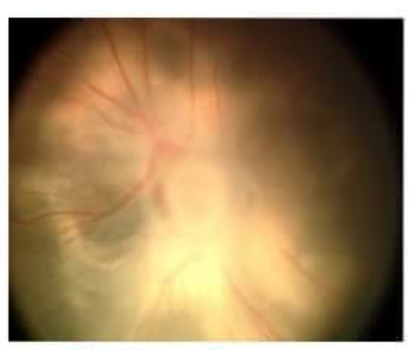
STAGE 5 : TOTAL RETINAL DETACHMENT

Stage 1



Stage 2

Stage 3



Stage 5

TREATMENT

→ 85% ROP regress → No Rx required

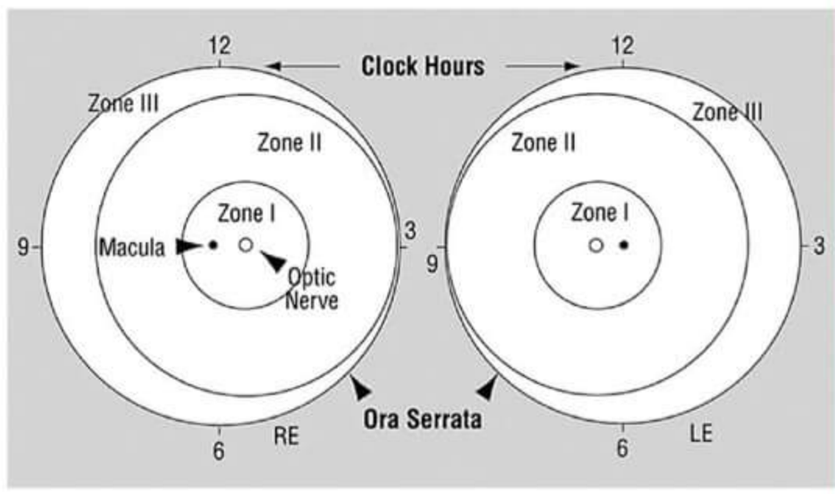
→ 15% ROP progress to Threshold ROP → Rx required

→ THRESHOLD ROP →

↳ 50% children will become blind if not treated

↳ CRITERIA

1. ZONES



zone 1 → central zone

zone 2 → surrounds zone 1

zone 3 → Temporal crescent zone

Created by

Taking optic disc as centre of retina

circle is drawn ī radius equal to twice the distance blw Fovea & optic disc gives ZONE 1

ZONE 2 → drawing the circle ī double the diameter of zone 1, which is meeting the nasal ora serrata keeping optic disc as centre

ZONE 3 → the temporal crescent that is left

ZONE 1 & 2 are more dangerous zones

2. EXTENT → 5 continuous clock hours of Fibrovascular proliferatn or & non continuous clock hours

3. STAGE 3

4. PLUS DISEASE

→ aggressive variety of ROP

→ veins congested & arteries are dilated

↳ All 4 criteria should require to qualify for Threshold ROP

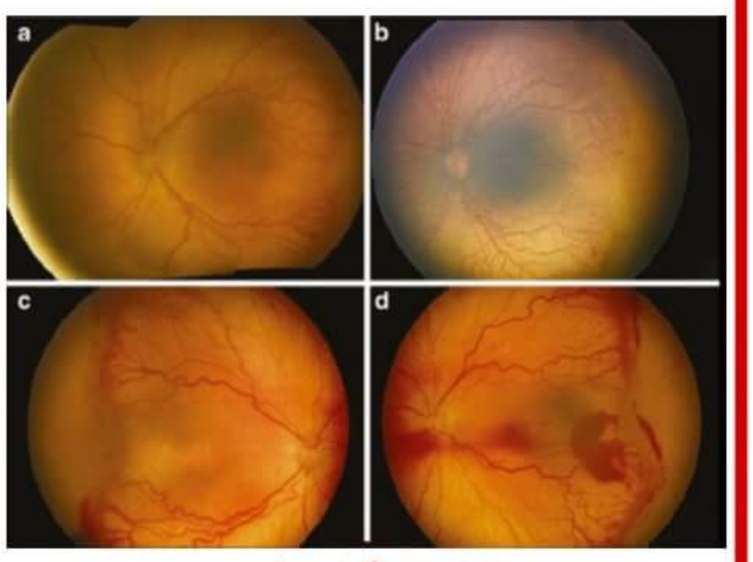
↳ TREATMENT

1. ABLATION → removal of hypoxic tissue by
   a. cryo
   b. Laser Photo coagulation

2. ANTI VEGF DRUGS → BEVACIZUMAB

PRE THRESHOLD ROP

TYPE 1 ROP → > 15% can develop blindness
TYPE 2 ROP → < 15% can develop blindness

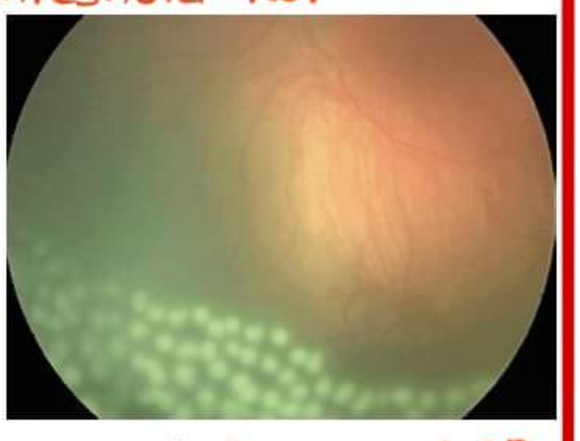


Threshold ROP

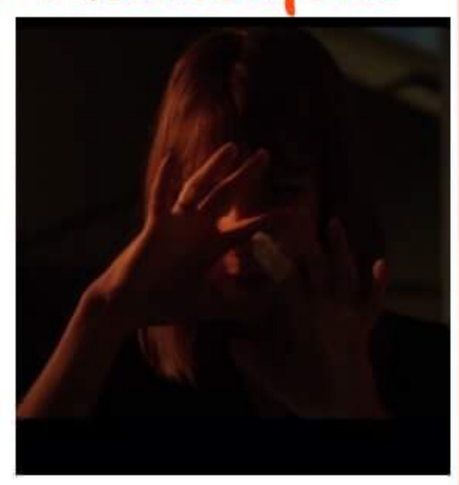
TYPE 1 ROP
→ ZONE 1 / Any Stage 1 plus disease
→ needs to be treated

TYPE 2 ROP → do not require Rx

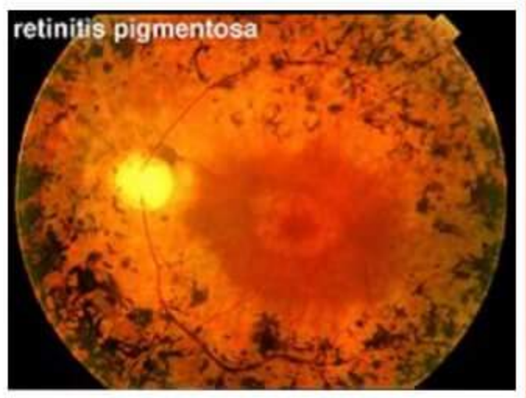
LASER PHOTO COAGULATION
→ Double frequency ND YAG layer is used


Laser Photocoagulatn

RETINITIS PIGMENTOSA
→ dlt Apoptosis of Rods
→ Autosomal Recessive (mc)
Autosomal dominant /
x - linked (worst prognosis)



→ RISK FACTOR → consanguinous marriages
→ CF
→ Nyctalopia
→ Ring scotoma ⊕nt
→ progress to tubular vision (constriction of visual fields)



→ O/E
↳ TRIAD
→ pale waxy disc
→ Arteriolar attenuation
→ BONE SPICULE PIGMENTATⁿ (Characteristic)

↳ Gold standard for Dx → ERG
→ measures the potential difference of Neuro sensory layer
→ Normal ERG
↳ negative 'a' - wave
↳ positive 'b' - wave
↳ small 'c' - wave (sometimes)
↳ a-wave → measures the potential difference of photoreceptors

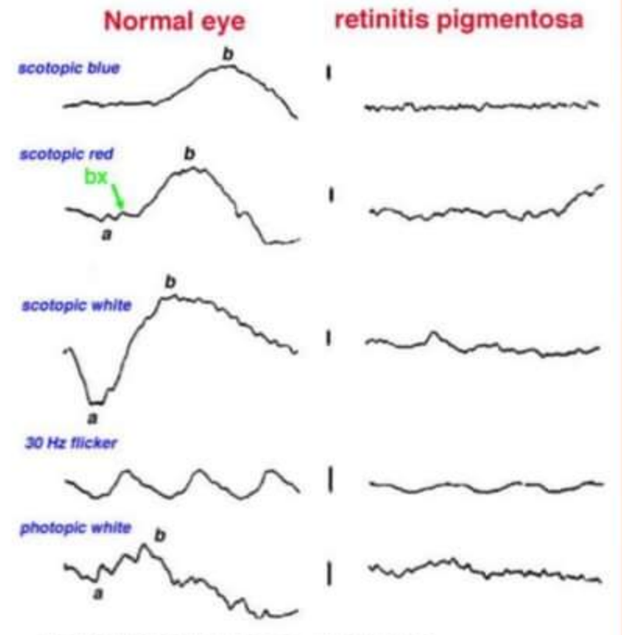


Fig. 13. ERG recordings in a normal patient and one with retinitis pigmentosa.

ERG - ElectroRetinogram

↳ b-wave → measures the potential difference of Muller's cells (neuroglia) & bipolar cells (neuronal cells)

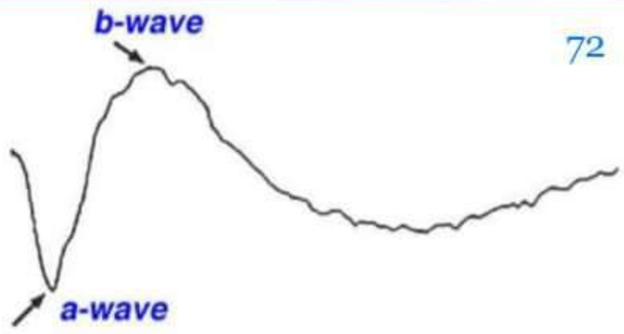


Fig.1 The biphasic waveform of the ERG of a normal patient.

→ in Retinitis Pigmentosa,
↳ ERG becomes FLAT

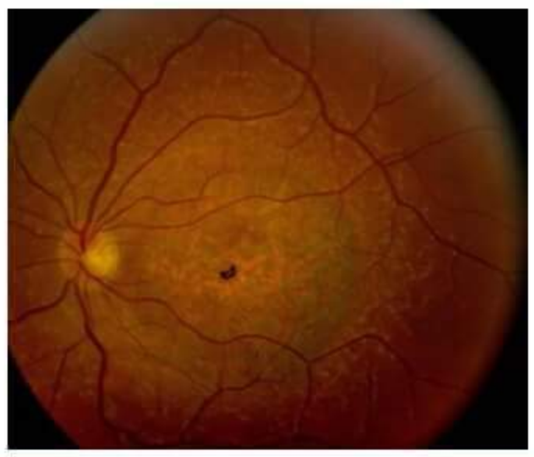
TREATMENT

→ 15000 IU/ day of Vit A in palmitate form for every Day
↳ not toxic
↳ can't be given during pregnancy
↳ doesn't cure RP
→ usually becomes blind by 40 yrs but 7 yrs of vision can be retained by vit A

RETINITIS PIGMENTOSA SYNDROMES

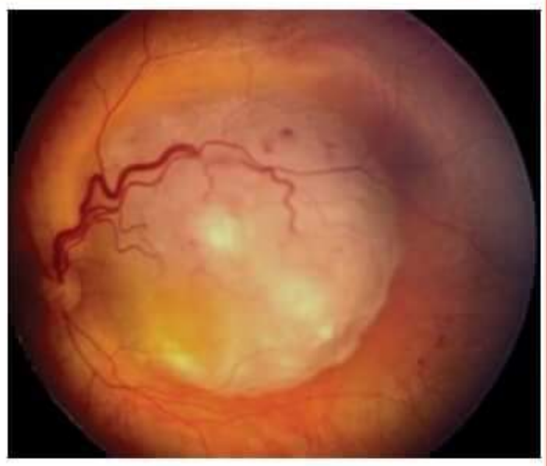
→ BASSEN KORNZWEIG SYNDROME
→ LAURENCE MOON SYNDROME
→ BARDET - BEIDL SYNDROME
→ USHER SYNDROME (mc syndrome a/w RP)
→ REFSUM SYNDROME

INHERITED JUVENILE MACULAR DYSTROPHY

BEST DISEASE	STARGARDT'S DISEASE
→ Autosomal Dominant → 6 yrs → no central loss → Scrumbled egg appearance → ERG → Normal → EOG → abnormal 	→ Autosomal Recessive → < 21 yrs → central loss of vision → Beaten Bronze appearance → ERG → Normal → EOG → Normal → 'Dark choroid' on FA (Diagnostic) 

RETINOBLASTOMA

→ mc ocular tumor of childhood
→ Commonest age of presentatn → 18 months
→ 40% are Heriditary
↳ mutation takes place in all the cells
↳ aka GERMLINE RETINOBLASTOMA
↳ B/L
↳ multifocal
↳ earlier presentatn

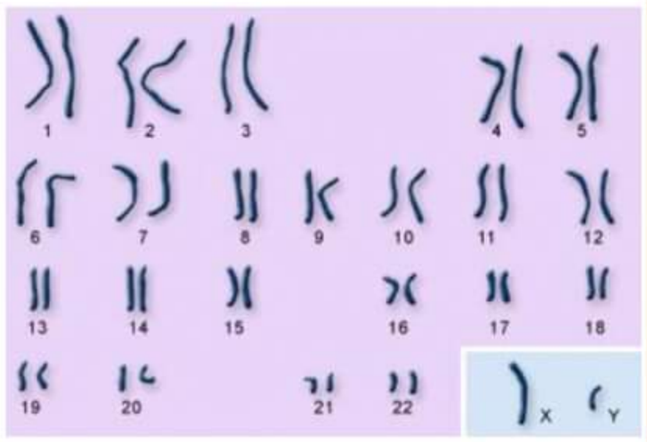


→ 60% are non - heriditary
  ↳ mutation takes place in Somatic cells
  ↳ UIL
  ↳ Later presentation

→ 30% are BIL
  70% are UIL

GENETICS

→ RB 1 Gene
  ↳ 1st tumor suppressor gene discovered
  ↳ located on 13q[14] (long arm of 13 chromosome on 14 band)
    → p = short arm = petit
    → q = long arm



RB 1 Gene

  ↳ Follows KNUDSON'S 2 HIT HYPOTHESIS

CLINICAL FEATURES

→ Leucocoria (Amaurotic cat's eye pupil) (mc)
  ↳ White pupil
→ Strabismus (2nd mc)
→ Glaucoma → mc → Neo vascular glaucoma (3rd mc)
  ↳ Pseudohypopyon (collectn of canar cells)
→ Loss of vision
→ Hetero chromia
→ Hyphemia
→ uniocular mydriasis

Leucocoria

D/D OF LEUCOCORIA

1. Retinoblastoma
2. Congenital cataract
3. Persistent Hyperplastic primary vitreous (PHPV)
   ↳ most difficult to d/d from Retinoblastoma
   ↳ PHPV

FATES OF VITREOUS

BEFORE BIRTH		AFTER BIRTH
Primary vitreous	→	Disappears (retention → PHPV)
Secondary vitreous	→	Normal
Tertiary vitreous	→	Zonules

   ↳ D/D of PHPV from RB

PHPV
1. always U/L
2. Only seen in small eye (microphthalmos)

4. Coat's disease
   ↳ Boys
   ↳ 1st decade of life (7-8 yrs)

Coat's Disease

5. ROP
6. Toxacariasis
7. Endophthalmitis
8. Retinal detachment
9. Norri's disease
10. coloboma

DIAGNOSIS

→ Primarily , clinical diagnosis
→ Intra ocular calcification on CT scan / x-ray / USG of eye
  ↳ mc cause of intraocular calcification → RB
→ FLEXNER WINTERSTEINER ROSETTE
  ↳ hallmark OF RB

Flexner wintersteiner Rosette

MANAGEMENT

→ Primary Aim → save life
  Secondary Aim → save eye
  Tertiary Aim → save vision

INTERNATIONAL CLASSIFICATION OF RB			
STAGE A	→	< 3 mm ;	Good prognosis
STAGE B	→	> 3 mm ;	prognosis is not good Macular location Minor Sub-retinal fluid
STAGE C	→	Localized Seeds , 1 in 3 mm , sub retinal or vitreous	
	→	moderate prognosis	
STAGE D	→	Diffuse seeds , more than 3 mm , Sub retinal or vitreous	
	→	unfavourable prognosis	
STAGE E	→	Massive Retinoblastoma more than 50% of eye ball , 1 sec - ondary glaucoma , hemorrhage	
	→	unfavourable prognosis	

TREATMENT

1. FOCAL
   → cryotherapy
   → Laser photocoagulation
   → transpupillary thermotherapy

2. SYSTEMIC
   → chemotherapy
     ↳ intravenous , improved success rates > 95%.
       → VEC - 6 cycles [VINCRISTINE / ETOPOSIDE / CARBOPLATIN]
       → S/E → deafness

   ↳ Intravitreal → DOC → MELPHALAN
   ↳ Intra arterial
     → into ophthalmic artery
     → MELPHALAN , TOPOTECAN , CARBOPLATIN

3. ENUCLEATION
   - → advanced intra ocular RB ī NVI (neovascularisat^{n} OF Iris)
   - → Secondary glaucoma
   - → AC invasion
   - → 75% OF vitreous volume involvement
   - → hyphema
   - → necrotic tumors ī Orbital inflammation

CAUSE OF DEATH

1. Metastasis
   - ↳ occurs ī in 1 year
   - ↳ mc mode of metastasis → via optic nerve → CNS
2. Intra cranial malignancy
   - ↳ Pinealoblastoma
   - ↳ TRILATERAL RETINOBLASTOMA → BIL Retinoblastoma + Pinealoblastoma
3. Secondary tumors
   - ↳ osteosarcoma OF femur (mc secondary tumor)

HYPERTENSIVE RETINOPATHY

→ occurs at → >140/90 mm Hg Blood pressure

COMPONENTS

1. Retinopathy
2. Choroidopathy
3. Neuropathy

RETINOPATHY

1. ACUTE → dlt vasospasm
1. CHRONIC → dlt arteriosclerosis


Gunn's sign


Bonnet's sign


Salus sign

CLASSIFICATION

MODIFIED SCHEIE'S

GRADE 0 → No changes
GRADE 1 → Barely detectable arterial narrowing
GRADE 2 → Obvious arterial narrowing ī focal irregularities
GRADE 3 → Grade 2 ⊕ Retinal Hemorrhages / exudates
GRADE 4 → Grade 3 ⊕ disc swelling

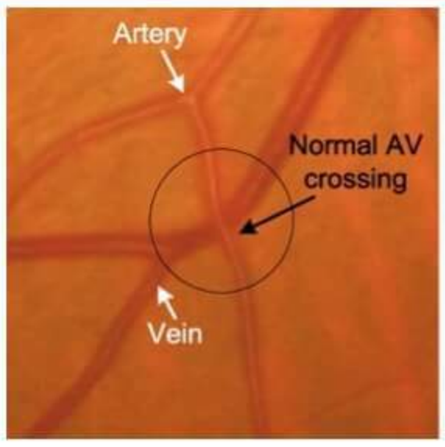
GRADE 3

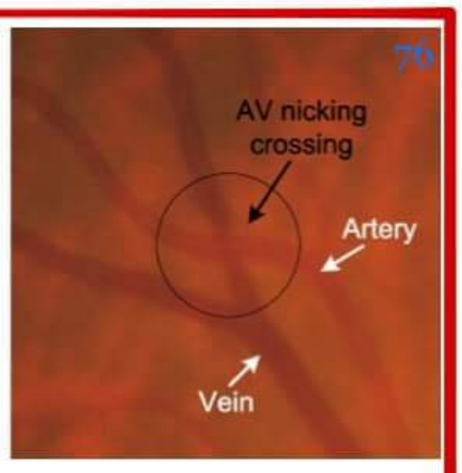
→ Flame - Shaped Haemorrhages on Nerve fiber Layer of Retina
→ soft exudates / cotton wool spots (collect^{n} of axoplasmic debris on retina)

AV CROSSINGS

→ mostly arteries cross over the veins (75% of times)
→ veins cross over the arteries (25% of times)
→ In HTN, Arteriosclerosis occurs, which compress the veins
→ max. AV crossings present in the Supero Temporal quadrant
→ BRVO (Branched Retinal venous occlusions) maximally present at Supero Temporal quadrant

→ Normal AV Ratio → 2 : 3
→ on HTN retinopathy, AV Ratio changes
↳ AV Nipping / Nicking occurs





FOCAL SIGNS OF GRADE 2

GUNN'S SIGN → Tappering of veins on either side of crossing

BONNET'S SIGN → Banking of vein distal to AV crossing

SALUS SIGN → deflectn of vein after AV crossing

ACCELERATED HTN → HTN which leads to end organ damage

MALIGNANT HTN → Accelerated HTN + Disc edema

Malignant HTN

COPPER WIRING & SILVER WIRING

→ indicates the colour of Blood column
→ seen in advanced Hypertensive Retinopathy

copper & Silver Wiring

ACUTE CHOROIDOPATHY

→ ELSCHNING'S SPOTS → hyperpigmented spots surrounded by hypopigmentatn

→ SIEGRIST STREAKS → linear hyperpigmentations Follow choroidal arteries

Elschning's Spots

Siegrist streaks

ACUTE OPTIC NEUROPATHY → contains optic disc edema

OPTIC NEURITIS

TYPES

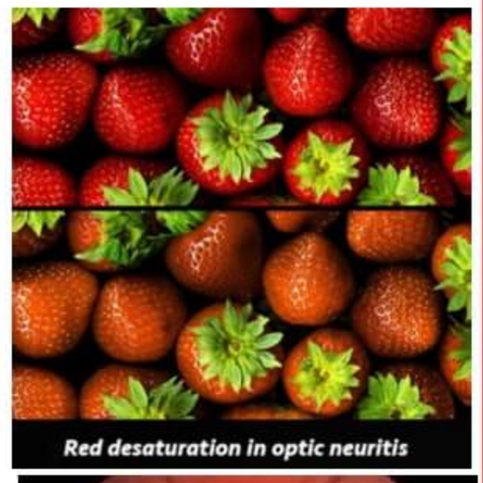
1. OPTIC NEURITIS / PAPILLITIS (40%)
2. RETRO BULBAR NEURITIS (RBN) (60%)

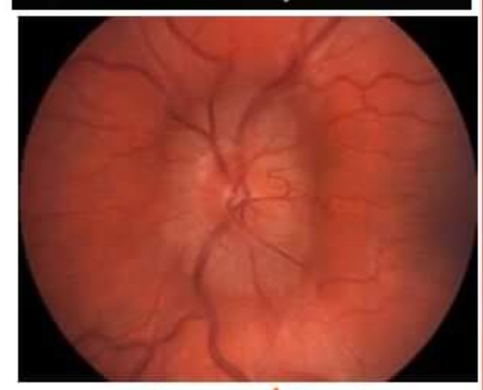
→ optic neuritis → inflammatⁿ of ON anteriorly close to optic disc
→ RBN → inflammatⁿ of ON posteriorly away from optic disc

→ mcc of optic neuritis → Multiple sclerosis
→ mc ocular manifestatⁿ of multiple sclerosis → optic neuritis
↳ IOC → MRI

CIF

→ sudden painful loss of vision
↳ pain intensifies ↑ ocular movement
→ colour vision desaturatⁿ (particulary red & green)
↳ bright colours appear pale
→ abnormal pupillary Reaction /
RAPD (Relative afferent pupillary Defect) /
Marcus Gunn pupil
→ Hallmark → Disc edema


Red desaturation in optic neuritis


Disc edema

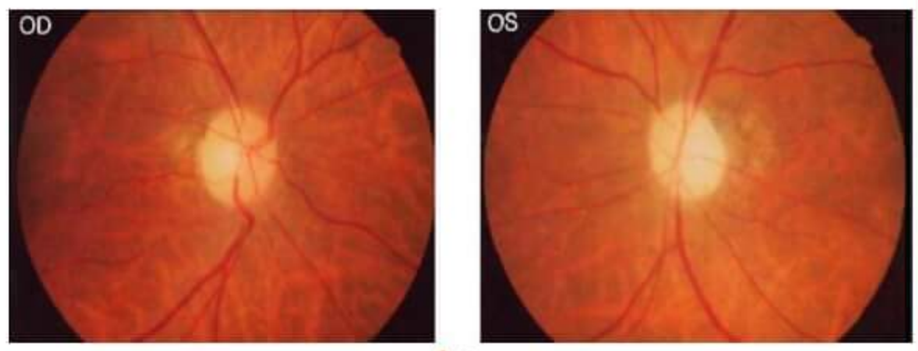
IN RETRO BULBAR NEURITIS, DISC EDEMA IS ABSENT
PATIENT SEES NOTHING & DOCTOR SEES NOTHING

VISUAL FIELD DEFECT

→ central scotoma (mc)
↳ centrocecal scotoma
→ involves centre & blind spot
→ seen in TOXIC AMBLYOPIA
↳ pale atrophic disc found o/E
↳ mc dlt Tobacco (cigars / pipes)
↳ 2nd mcc → Alcohol
↳ slow painless loss of vision (10-20 yrs)
↳ Rᵧ → Stop the drugs
→ inj. hydroxy cobala-mine


central scotoma

centrocecal scotoma



Toxic Amblyopia

TREATMENT OF ON / RBN

→ IVMP (IV Methyl Prednisolone)

ON / RBN	TOXIC AMBLYOPIA
→ sudden painful loss of vision	→ slow painless loss of vision
→ mcc → multiple sclerosis	→ mcc → Tobacco, Alcohol
→ ON → Disc edema	→ B/L pale atrophic discs
→ central scotoma	→ centrocecal scotoma
→ Rᵧ by IVMP	→ Rᵧ by Inj. Hydroxy cobalamine

ALTITUDINAL HEMIANOPIA

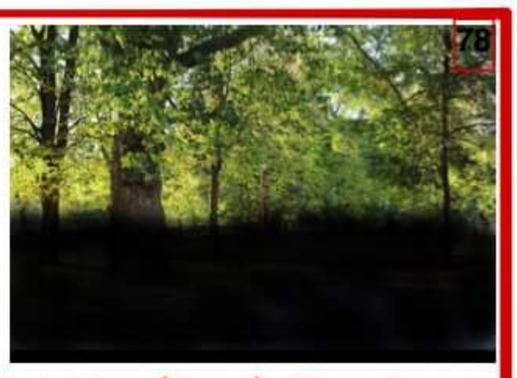
→ superior / inferior half of visual field is lost

→ seen in AION (Anterior ischemic Optic neuropathy)

↳ common disorder

↳ seen in older people ī HTN

↳ optic disc is pale



Altitudinal Hemi-anopia in AION

PUPILLARY REACTIONS

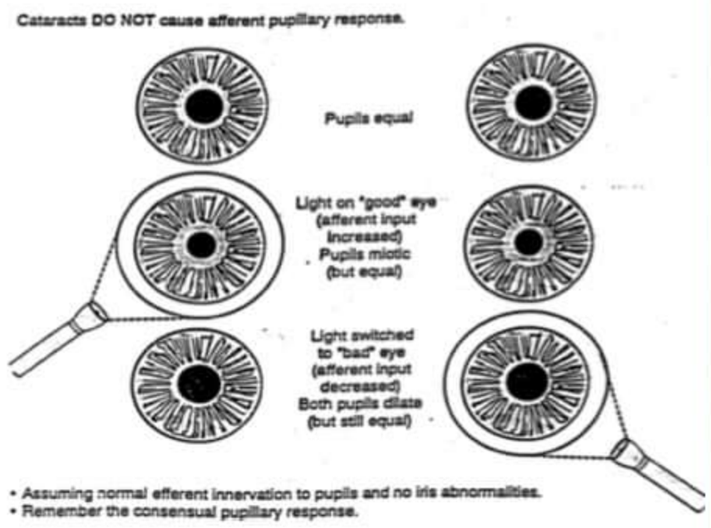
TESTS FOR NORMAL PUPILS

1. LIGHT REACTION
   - a. DIRECT
   - b. CONSENSUAL

   } Parallel to each other

LIGHT REFLEX

2. TRIPLE REACTION / NEAR / ACCOMMODATION REACTION
   - a. Accommodation
   - b. Convergence
   - c. constriction



ABNORMAL PUPILS

MARCUS GUNN PUPIL

→ Tested by SWINGING TORCH LIGHT TEST

↳ On swinging the light to abnormal pupil, it dilates

↳ seen in Optic neuritis

ARGYLL ROBERTSON PUPIL

1. BIL
2. Constricted
3. Irregular pupil

Argyll Robertson pupil

→ seen in neuro syphilis

→ obeys Light -ive & Accommodatⁿ +ve Rule

Argyll	Accommodatⁿ	Pupillary
Robertson	Reflex	Reflex
Pupil	Present	Absent

→ aka PROSTITUTE PUPIL → behaves like a Prostitute

↳ Accommodates but not react

ADIE'S PUPIL

→ seen in young women

→ Seen after viral fever

↳ One pupil dilates & other remains normal

↳ Virus attacks Ciliary ganglion → CN III affected

→ parasympathetic constrictor fibres of pupil damaged

→ unopposed sympathetic dilatatⁿ of pupil occurs

→ Obeys 'Light negative & Accommodatⁿ positive' Rule

ARGYLL ROBERTSON PUPIL	ADIE'S PUPIL
→ constricted pupil	→ Dilated pupil
→ B/L	→ U/L

ADIE'S PUPIL

ADIE'S TEST

→ Confirmed by PILOCARPINE CHALLENGE TEST
  ↳ 0.125% pilocarpine solution used
  ↳ normal pupil do not constrict
  ↳ abnormal pupil will constrict
    ↳ dlt denervation supersensitivity

HORNER'S SYNDROME

→ **O**culo
  Sympathetic
  Paralysis

HORNER'S PUPIL

→ Ptosis
→ Miosis
→ Anhydrosis
→ Enophthalmos
  → not a part of Horner's syndrome
  → not a true Enophthalmos
  → Normally,
    Lower eye lid present at limbus (dlt Inferior tarsal muscle)
    Upper eye lid covers 1-2 mm (dlt muller's muscle)
    Both muscles are supplied by sympathetic system

congenital Horner

→ On Horner's syndrome, oculo sympathetic paralysis occurs
  → Muller's muscle affected → Ptosis (mild)
  → Inferior tarsal musle affected → Lower Lid risesup (Inverse Ptosis)
  → appears to Enophthalmos (PSEUDO ENOPHTHALMOS)

→ TYPES

CONGENITAL	AQUIRED
→ Ptosis miosis Anhydrosis Enophthalmos HETEROCHROMIA (different coloured Irises)	→ Ptosis miosis Anhydrosis Enophthalmos
→ mcc → Birth trauma	→ mcc → Pancoast tumor

Pancoast tumor

→ confirmed by COCAINE TEST
  ↳ Normal eye → 10 mm dilatatⁿ with in 1 hr
    Horner's pupil → No dilatation

NYSTAGMUS

- → involuntary oscillatory to & fro movement of the eyes
- → PENDULAR → Phase of equal velocity
- → JERK → Phase of unequal velocity
- → DIRECTION → direction of fast component, pathological movement is the slow one
- → TRAJECTORY → Horizontal, vertical, rotatory
- → CONJUGATE → Both eyes same movement
- → NULL ZONE → gaze in which intensity is minimal

PHYSIOLOGICAL
1. optokinetic
2. vestibular
3. End - point

INFANTILE
1. congenital
2. manifest - latent

ACQUIRED
1. Gaze Evoked
2. Vestibular
3. Inter nuclear ophthalmoplegia
4. See - saw
5. Down - beat

PHYSIOLOGICAL

- → Optokinetic → looking at moving object
- → vestibular → When cold / warm saline stimulates endolymph
- → End point → goes to extreme point & comes back
- → Specific localising Nystagmus

CONGENITAL

- → present at birth or appear later shortly
- → usually horizontal, pendular or jerk
- → abolished in sleep
- → UNIPLANAR → Plane remains unchanged in all gazes

LATENT

- → Seen only when one eye is covered
- → vision decreases dlt nystagmus
- → When each eye tested separately improved when both uncovered

DOWN BEAT NYSTAGMUS

- → Fast phase down, while eye in primary position of rest
- → PATHOLOGY
  - ↳ Arnold chiari malformation [most important]
  - ↳ platybasia

UPBEAT NYSTAGMUS

- → Fast phase up, eyes in primary position of rest
- → PATHOLOGY
  - ↳ cerebellar degeneration [most important]
  - ↳ multiple sclerosis
  - ↳ Brainstorm stroke

VESTIBULAR NYSTAGMUS

- → dlt vestibular end organ dysfunction
- → Mixed directional, usually horizontal – torsional
- → Suppressed by visual fixation
- → a/w vertigo, tinnitus, deafness
- → ETIOLOGY → Labyrinth, Neuritis, Meniere's disease
- → COWS → cold opposite, warm same
  - → cold water when passed into ear it stimulates the endolymph & produce nystagmus in the same direction

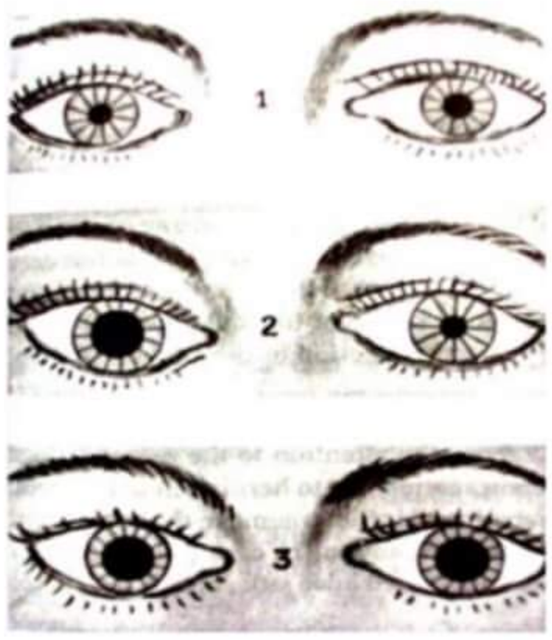
OPTOKINETIC NYSTAGMUS [OKN]

- → Image stabilization when viewing a constantly moving visual field
- → Asymmetric OKN is abnormal OKN, When target is moving in one direction compared to opposite
- → COGAN'S DICTUM
  - → it states that Homonymous hemianopia + asymmetric OKN – parietal lobe lesion, probably mass lesion
  - → Homogenous Hemianopia + Symmetric OKN – occipital lobe lesion, vascular lesion

HUTCHINSON PUPIL

Hutchinson's pupil

- Seen in case of cerebral compression (extra dural Haemorrhage)
- Consists of 3 stages



Pupil of the side of injury contract due to irritation of occulomotor nerve Pupil on other side -normal

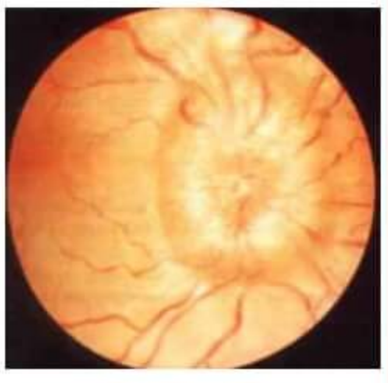
pupil of the injured side becomes dilated due to paralysis of occulomotor nerve Pupil of other side contracts

Pupils of both sides dilated, no reaction to light

SIDE OF TRAUMA

NORMAL SIDE

PAPILLOEDEMA



- → Disc edema i raised ICT
- → Normal ICT → 50 – 180 mm of water
- → Papilloedema
  - ICT in adults → > 250 mm of water
  - ICT in children → > 200 mm of water

PATHOLOGY → ORTHOGRADE AXOPLASMIC STASIS OF OPTIC NERVE

CAUSES

1. LCSOL → Intra cranial Space Occupying Lesions of posterior fossa EXCEPT tumors of medulla
   - → more posterior the tumor, more the papillodema

2. PSEUDO TUMOR CEREBRI → Diagnosis of exclusion
   → aka IIH (Idiopathic Intracranial Hypertension)
   → young fat females ī unexplained headaches
   → Pulsatile tinnitus ⊕nt

3. MENINGITIS
4. SUB ARACHNOID / INTRA CEREBRAL HAEMORRHAGE → Sudden papilloedema

CLINICAL FEATURES

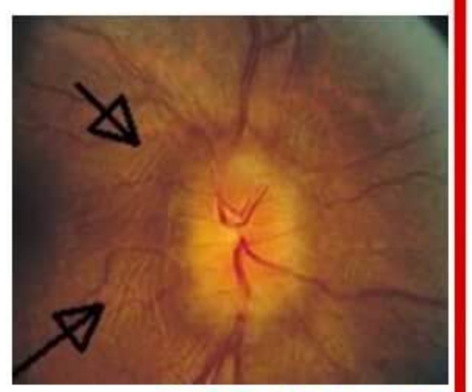
1. Headache
   → occipital headaches
   → Throbbing, pulsating
   → changes ī changing posture
   → worsens ī straining, coughing, Sneezing, valsalva procedure

2. Projectile vomiting → no prior nausea
3. Amaurosis fugax → Sudden Transient temporary loss of vision / Transient Visual Obscuration
4. Disc edema
5. 6th nerve palsy (false localising sign)

PAPILLOEDEMA VS OPTIC NEURITIS

PAPILLOEDEMA

1. BIL Except FOSTER KENNEDY SYNDROME
2. Excessive disc edema
3. PATON'S LINES → circumferential lines around optic disc
4. Loss of venous pulsations



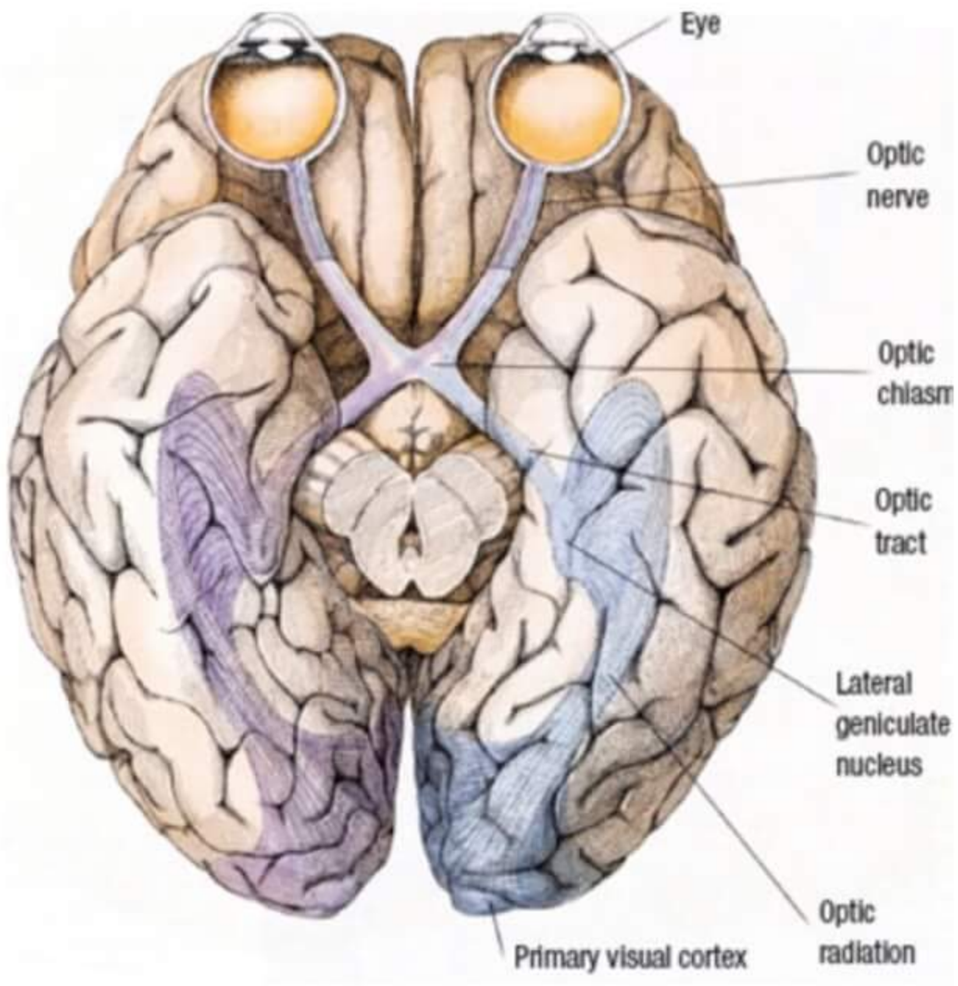
PATON'S LINES

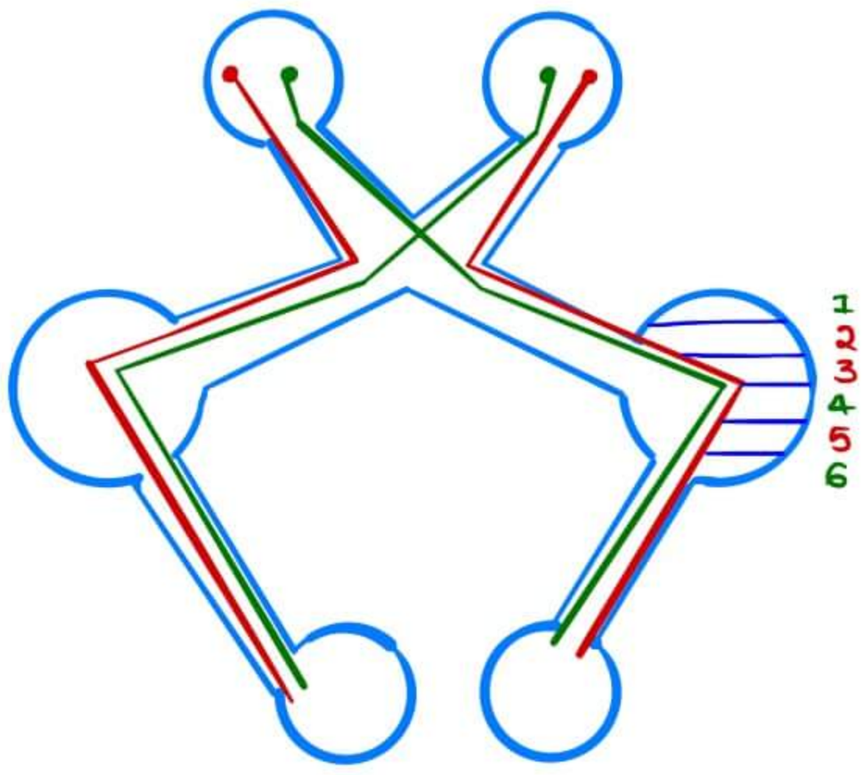
FOSTER KENNEDY SYNDROME

→ Seen in frontal lobe tumors
→ Ipsilateral optic atrophy d/t tumor
→ C/L Disc edema d/t ↑ ICT

VISUAL FIELD DEFECT → Enlargement of blind Spot (mc)

VISUAL PATHWAY





→ Temporal fibres join the Geniculate body in 2,3 & 5 layers
→ Opposite nasal fibres join the Geniculate body in 1,4,6 layers

VISUAL FIELD DEFECTS

CONVENTION FOLLOWED BY DOCTORS
↳ Field infront OF Left eye OF doctor → Left visual field
↳ Field infront OF Right eye OF doctor → Right visual field

BITEMPORAL HEMIANOPIA

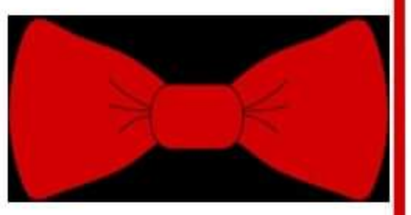
→ Nasal fibres gets damaged
↳ caused by Optic chiasmal lesion → Pituitary adenoma (mc)

BINASAL HEMIANOPIA

→ Both the temporal fibres are damaged [Rarely seen]

RIGHT SIDED HOMONYMOUS HEMIANOPIA

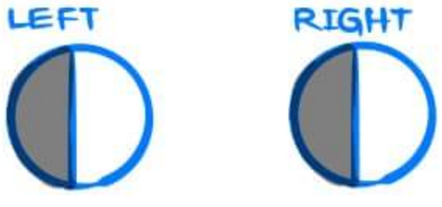
→ Homonymous = corresponds to each other
→ In Left eye → Lt. temporal fibres are damaged
In Right eye → Rt. nasal fibres are damaged
→ Seen in Left Optic tract lesion [mc]
[Any post chiasmal lesion on Left side can cause Rt. Homonymous Hemianopia]
→ FOR ALL HOMONYMOUS HEMIANOPIA, LESION WILL BE IN OPPOSITE OPTIC TRACT



BOW TIE OPTIC ATROPHY

→ Seen in Optic tract lesion
→ dlt Retrograde degeneration [identified after 4-6 weeks after the lesion]
→ elicited in optic disc
↳ temporal & nasal poles of disc are damaged
↳ Superior & inferior poles of disc are undamaged

BOW TIE OPTIC ATROPHY



LEFT HOMONYMOUS HEMI ANOPIA

→ Seen in Right optic tract lesion

(Lt) Homonymous Hemianopia

LEFT MONOCULAR BLINDNESS

- → IN ALL MONOCULAR FIELD DEFECTS , I/L PRE CHIASMATIC LESION WILL BE PRESENT
- → Left monocular blindness is dlt lt prechiasmatic lesion
- → Same side lesion
- → B/L FIELD DEFECTS ARE DUE TO C/L CHIASMATIC OR POST CHIASMATIC LESIONS

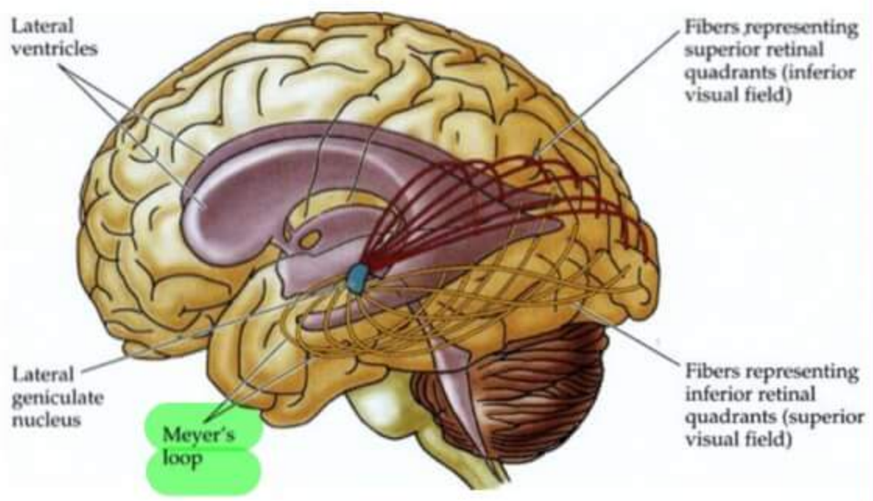
RIGHT HOMONYMOUS SUPERIOR QUADRANTANOPIA / Rt PIE IN THE SKY

- → Inferior fibres are damaged
- → Inferior fibres pass through temporal lobe
- → Left Temporal Lobe lesion causes it
- → ALL PIE IN THE SKY ARE SEEN IN C/L TEMPORAL LOBE LESIONS

RIGHT HOMONYMOUS INFERIOR QUADRANTANOPIA / Rt PIE ON THE FLOOR

- → Superior fibres are damaged
- → Superior fibres pass through parietal lobe
- → Lt parietal lobe lesion causes it
- → ALL PIE ON THE FLOOR ARE SEEN IN C/L PARIETAL LOBE LESIONS


macular sparing



MACULAR SPARING

- → central 5-10° is spared
- → Lesion is present in Right occipital lobe
  - ↳ occipital lobe which represent macula has DUAL BLOOD SUPPLY
    - → Posterior cerebral Artery → damaged
    - → middle cerebral Artery → not damaged → macula spared
  - ↳ Macula represented by LARGE AREA OF occipital Lobe
    - → Extensive lesions needed to damage whole macula

1.	Disc / Optic Nerve lesions	Same sided monocular Blindness
2.	Chiasmal lesions	Bitemporal Hemianopia
3.	Optic tract lesions (mc)	Opposite homonymous hemianopia CIL Bow tie Optic atrophy
4	Geniculate body lesions	Key hole Field defect / Sector anopia
5.	Optic radiation lesion	
	Meyer's Loop (Temporal lobe)	Pie in Sky
	Baum's loop (parietal lobe)	
6.	occipital lobe lesion	Macular Sparing

GAZE CENTERS

→ GAZE = Both eyes moving together

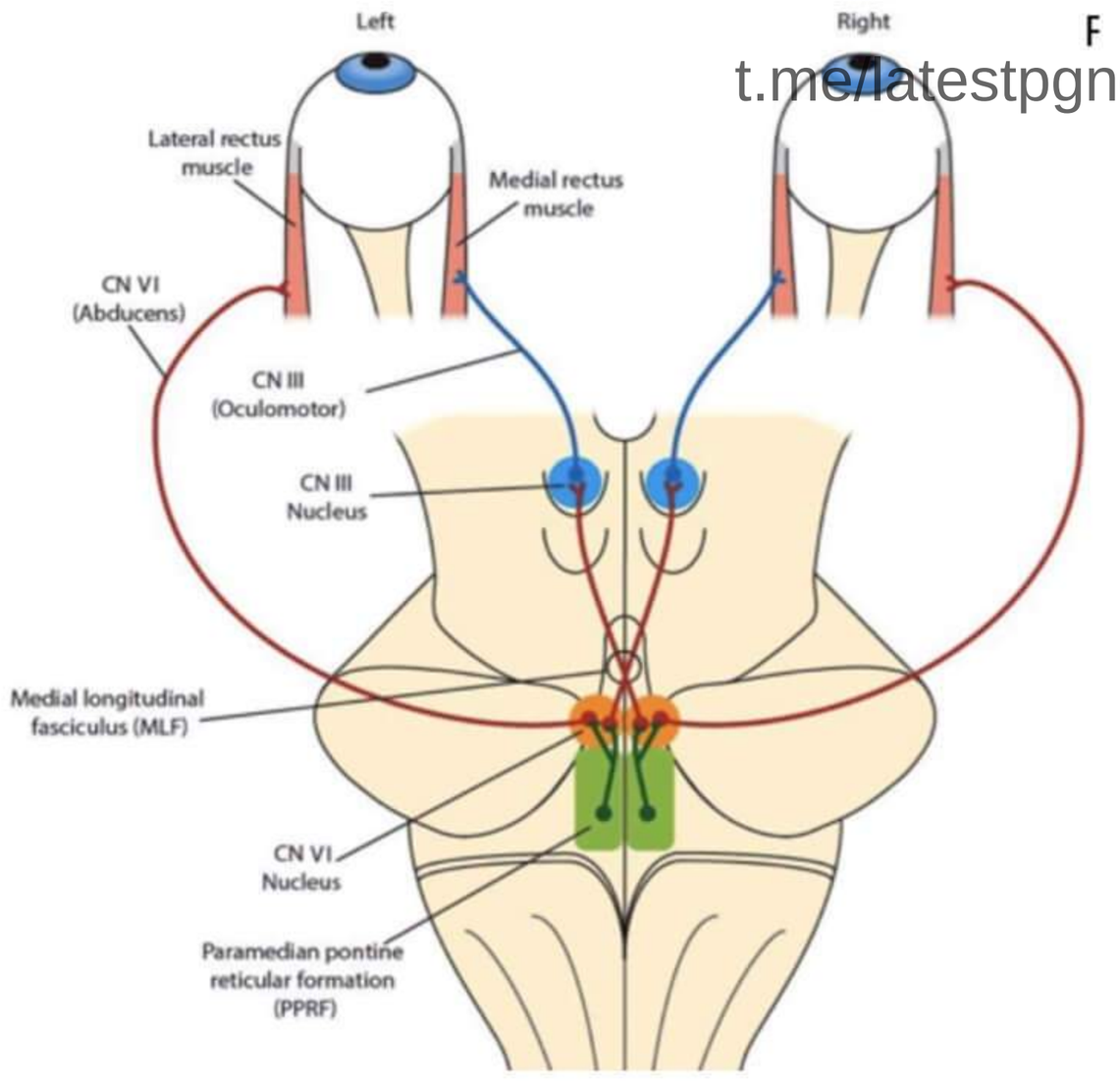
→ Gazes are controlled by certain centres

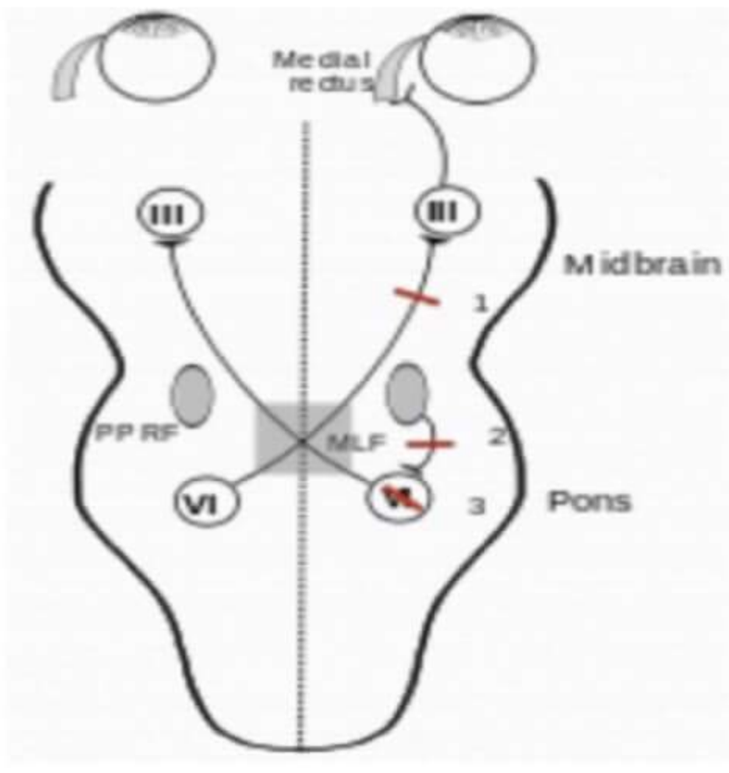
CENTRE FOR HORIZONTAL GAZE [abduction, adduction, Left & right gazes]

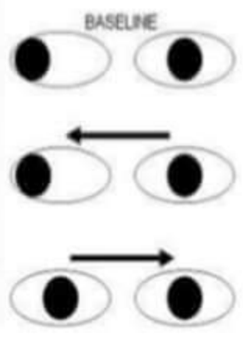
→ PARAMEDIAN PONTINE RETICULAR FORMATION [PPRF]

CENTRES FOR VERTICAL GAZE [Elevation, Depression]

1. Rostral Interstitial nucleus of MLF [Medial longitudinal fasciculus] IMF
2. Interstitial nucleus of cajal
3. Posterior commissure







HORIZONTAL GAZE PALSY

→ dlt PPRF Paralysis

→ Eg : if Right PPRF is damaged, the eye can not move to right side

INTER NUCLEAR OPHTHALMOPLEGIA

- → dlt lesion at MLF
- → INO is Ipsilateral adduction nystagmus ī contralateral abduction nystagmus

ONE AND HALF SYNDROME

- → Lesion is dlt combinat^{n} of MLF ī PPRF of the same side
- → Loss of all horizontal movements except contralateral adduction, which shows abduction nystagmus

EIGHT AND A HALF SYNDROME

- → 7 th Nerve palsy ī one and half syndrome of same side

OCULAR MOVEMENT

10 MUSCLES

- ③ INTRA OCULAR MUSCLES [IOM]
- ⑦ EXTRA OCULAR MUSCLES [EOM]

③ INTRA OCULAR MUSCLES [IOM]

1. Ciliary muscle [supplied by CN III]
2. Sphincter pupillae
3. Dilator pupillae

⑦ EXTRA OCULAR MUSCLES [EOM]

① Muscles belong to eyelid
1. Levator Palpabrae Superioris [LPS]

⑥ Muscles belong to Eye ball

④ Recti
1. Superior rectus [SR]
2. Inferior rectus [IR]
3. medial rectus [mR]
4. Lateral rectus [LR]

② Oblique
1. Superior oblique [SO]
2. Inferior Oblique [IO]

MUSCLES OF EYELID

OPEN THE EYELID

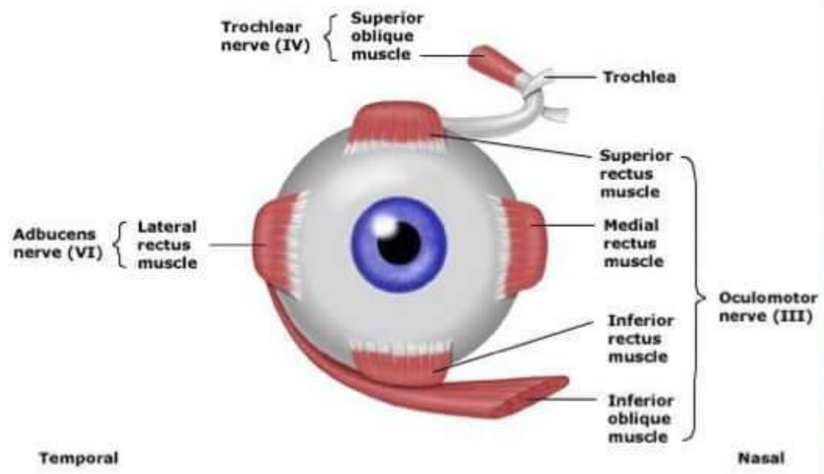
1. LPS → most important
2. Muller's muscle
3. Frontalis muscle

} not part of EOM

CLOSES THE EYELID → Orbicularis muscle

NERVE SUPPLY

LR 6
SO 4
REST ALL by CN III
LPS also by CN III



ACTION OF MUSCLES

DUCTION → One eye moves
VERSION → Both eyes are moved

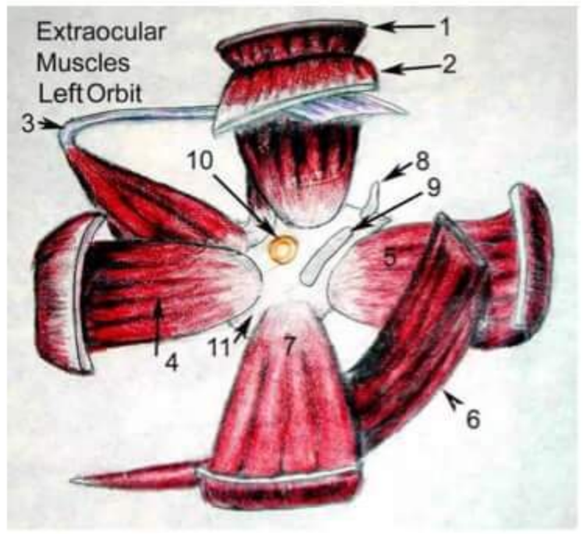
DUCTION

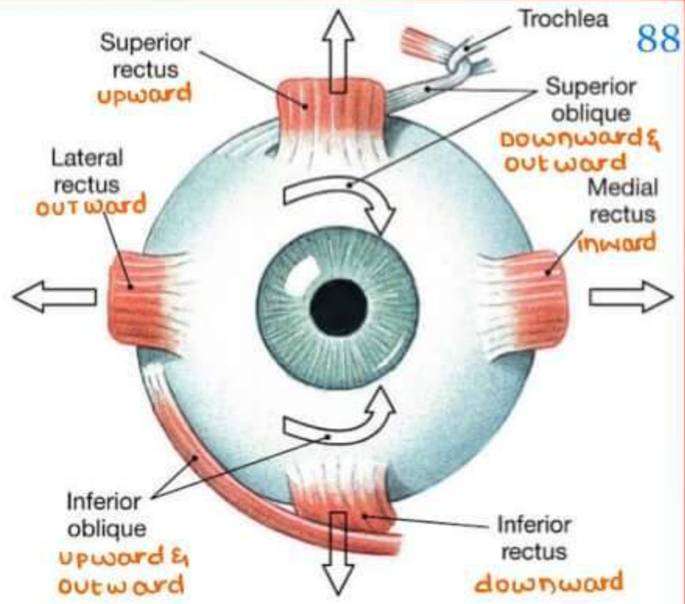
1. Elevation → moving up
2. Depression → moving down
3. Abduction → moving away
4. Adduction → moving inwards
5. Intorsion → rotating inwards
6. Extorsion → rotating outwards

ACTIONS OF MUSCLES

MEDIAL RECTUS → Adduction

LATERL RECTUS → Abduction





SIN RAD

Superior muscles cause Intorsion

Inferior muscles cause Extorsion

Rectus muscles cause Adduction

Oblique muscles cause Abduction

SUPERIOR RECTUS	INFERIOR RECTUS	SUPERIOR OBLIQUE	INFERIOR OBLIQUE
Intorsion	Extorsion	Intorsion	Extortion
Adduction	Adduction	Abduction	Abduction
Elevation	Depression	Depression	Elevation

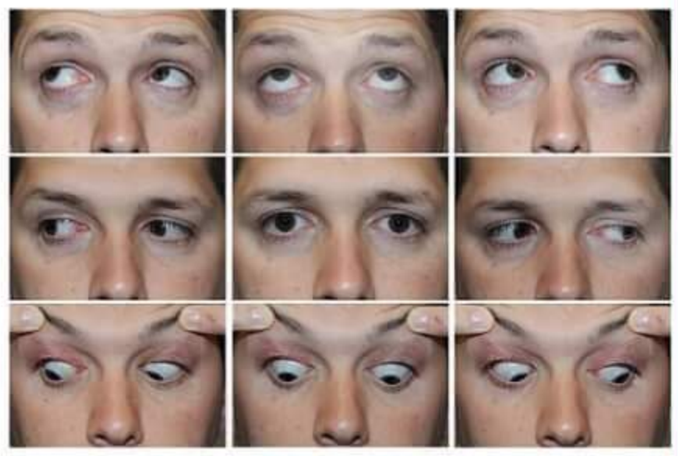
	ELEVATION	ABDUCTION
Agonist	SR	LR
Antagonist	IR	MR
Synergist	IO	SO, IO

Superior Rectus 1° Action = Elevation

Inferior Rectus 1° Action = Depression

Superior oblique 1° Action = Intorsion

Inferior Oblique 1° Action = Extorsion



cardinal Gazes

9 CARDINAL GAZES [Both Eyes]

1 Primary [Straight]

4 Secondary [Up, down, Left, right]

4 Tertiary [Upright, Upleft, Down right, Down Left]

YOKE MUSCLES

→ pair of muscles from both eyes, when they contract moves the eyeball in the same direction

[R] LR + [L] MR

[L] LR + [R] MR

[R] SR + [L] IO

[L] IR + [R] SO

→ Laevoversion → [L] LR + [R] MR

→ Dextroversion → [R] LR + [L] MR

→ Laevo Elevation → [L] SR + [R] IO

→ Dextro Depression → [R] IR + [L] SO

Leavo = Left

Dextro = Right

SQUINT/STRABISMUS

Q Involuntary closure of one eye after entering into bright sunlight. Dx ?
A SQUINT

SQUINT / STRABISMUS → 2 eyes are not parallel
ORTHOPHORIA → 2 eyes are parallel
HETEROPHORIA = SQUINT

TYPES

TROPIA
Manifest squint (visible)

PHORIA
Latent squint (not visible)
↳ too small
↳ Binocular balance

→ When binocular balance suspended, Phoria converts into Tropia

SUBTYPES OF TROPIAS

1. EXOTROPIA → one or both eyes are diverted outwards
2. ESOTROPIA → one or both eyes are inwards
3. HYPERTROPIA → one eye is above the other eye
4. HYPOTROPIA → one eye is below the other eye


Rt. Exotropia


Lt. Esotropia


Rt. hypertropia

SUBTYPES OF PHORIAS

1. EXOPHORIA
2. ESOPHORIA
3. HYPERPHORIA
4. HYPOPHORIA

} no Right & left demarcation


HIRSCHBERG TEST

9 CARDINAL GAZES

1 Primary Gaze
4 Secondary Gazes [up, down, Right, left]
4 Tertiary Gazes [up & right, up & left, down & right, down & left]

TESTS

HIRSCHBERG TEST (for Tropias)
→ Flash a torch into forehead (Glabella) of patient, ask the patient to look at the light
↳ ORTHOPHORIC → Reflectⁿ of light is exactly at the centre of the pupil
↳ HETEROPHORIC → Reflectⁿ of light is not at the centre of pupil

CALCULATION OF SQUINT

→ measure the distance between centre of pupil & where reflection is present

→ Each 1 mm decentration = 7° Squint

1° = 2 prism diopters

= 14 prism diopters

Q Lt. eye decentration of 3 mm to temporal

A. 3 x 7 = 21°

21 x 2 = 42 prism Diopter

= 42 prism Diopter ESOTROPIA

↳ reflectⁿ is outwards

↳ eye movement is inwards

SQUINT IS ALWAYS OPPOSITE TO THE DIRECTION OF REFLECTION

COVER UNCOVER TEST (For phorias)

PROCEDURE

→ Flash the torch on to fore head of patient & ask her to see the light

→ Cover one OF the eye

→ Resting eye will go into it's original position

→ Remove the cover & ask her to see the light again

→ In EXOPHORIA → eye ball moves from Out to IN

→ In ESOPHORIA → eye ball moves from in to out

→ In ORTHOPHORIA → No movement of eye ball

→ No need of Treatment for Phorias most of the times

ESOPHORIA

TROPIA

COMITANT (Together)

→ angle blw the 2 eyes remains the same in all 9 gazes

→ NO functional problem

comitant squint

INCOMITANT SQUINT (angle blw the eyes changes i every gaze)

PARALYTIC SQUINT

→ paralysis of nerve (3,4,6 CN8)

→ paralysis of NMJ → Myasthenia gravis

→ common causes

→ uncontrolled DM

→ uncontrolled Blood pressure

→ Rt. 6th Nerve Paralysis

↳ Rt. lateral rectus paralysed

↳ unopposed medial rectus

↳ presents i massive diplopia

Paralytic squint

INCOMITANT SQUINT

→ angle blw the eyes changes i every gaze, which causes
1. Diplopia
2. Abnormal Head Posture (AHP)
3. vertigo
4. Disorientation

RESTRICTIVE SQUINT

→ dlt Restriction of muscle movement
→ Seen in Thyroid Related ophthalmopathy [TRO] / Thyroid Eye disease [TED]
↳ 1st muscle to get restricted → Inferior rectus
↳ order of muscle Restriction
I → Inferior Rectus
M → Medial Rectus
So → Superior Rectus
Lucky → Lateral Rectus


Restrictive squint

INCOMITANT SQUINT

DIPLOPIA

CLASSIFICATION

HORIZONTAL	VERTICAL	OBLIQUE
one of the horizontal muscles paralysed	one of the vertical muscles paralysed	one of the oblique muscles paralysed

MONOCULAR	BINOCULAR
→ seen by one eye → causes cataract (mcc) Aphakia Subluxated lens Keratoconus	→ seen by two eyes → dlt squint (mcc of binocular diplopia)

ABNORMAL HEAD POSTURE (AHP)

→ compensatory gesture to reduce diplopia
↳ FACE TURN (indicates horizontal muscle palsy)
↳ Chin elevation / depression (indicates vertical muscle palsy)
↳ Head tilt (indicates oblique muscle palsy)

PARALYTIC SQUINT VS RESTRICTIVE SQUINT

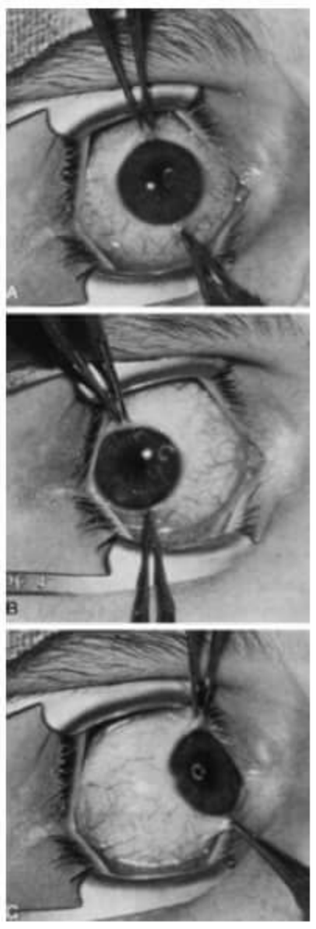
CASE STUDY

POSSIBILITIES

Rt Lateral Rectus palsy (paralytic squint)

Rt medial Rectus Restriction (Restrictive Squint)

FDT [FORCED DUCTION TEST]



1. Local anesthetic drops used to paralyse the sensations
2. ī a forceps, superiorly & inferiorly Examiner pull & pushes the eye ball

In paralytic squint →	Eye ball can be moved
In Restrictive squint →	Eye ball can't be moved

+ive (Restrictive Squint)

-ive (paralytic Squint)

PARALYTIC SQUINT

COMMON CAUSES

1. uncontrolled diabetes
2. uncontrolled HTN

DUE TO

1. Paralysis of Nerve
   a. 3rd CN palsy
   b. 4th CN Palsy
   c. 6th CN Palsy
2. Paralysis of NMJ
   → Myasthenia gravis

Accommodative ESOmetropia (uncorrected hypermetropia)

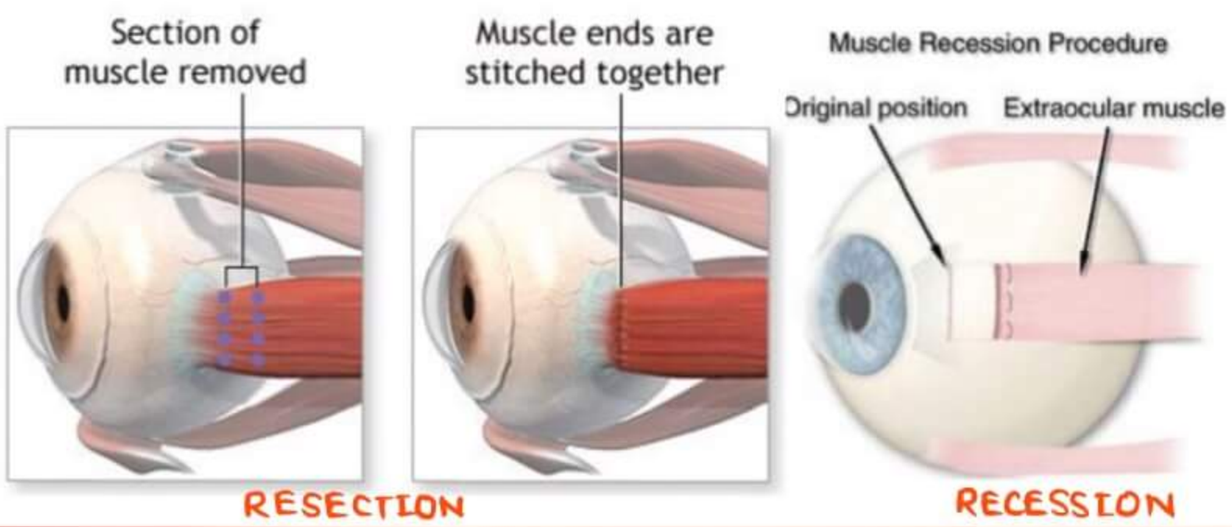
After correction

COMITANT SQUINT TYPES

1. Accommodative Squint
   → dlt uncorrected Refractive error
   ↳ uncorrected myopics develop Divergent Squint
   ↳ uncorrected hypermetropics develop convergent squint
   ↳ Treated by prescribing glasses

Non Accommodative Squint

2. NON ACCOMMODATIVE SQUINT
   → dlt high fever, post trauma, chronic disorders etc
   → do not improve ī glasses
   → Rx by surgery
   → Surgery (Recession and/or Resection)
   ↳ aims at making one muscle weaker → by Recession
   ↳ making opposite muscle stronger → by Resection



RESECTION

RECESSION

In Recession,
Muscle is cut from the surface of the eye and reattached further back from the front of the eye

INCOMITANT SQUINT

PARALYTIC SQUINT

TREATMENT

1. correct underlying disorder (BP & sugar)
2. wait & watch for 6 months (for the recovery of muscle)
3. correct the Diplopia ASAP
   a. Patching
   b. Prismatic Glass
   c. Botulinum Toxin into opposite muscle
      ↳ now both muscles are paralyzed
      ↳ Temporary measure

Patching

Prism

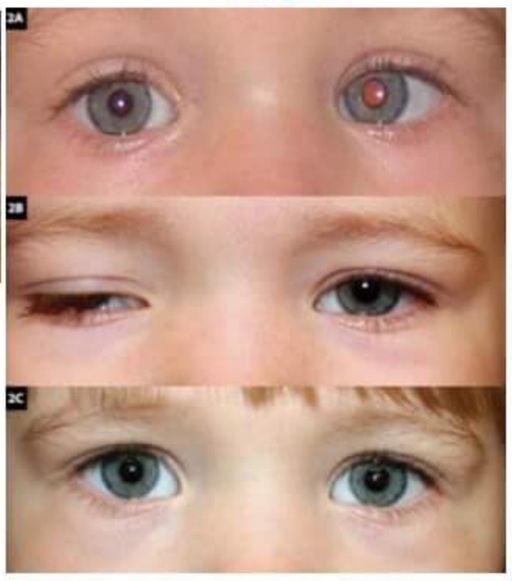
CLINICAL FEATURES

PARALYSIS OF NERVES

3 RD NERVE PALSY

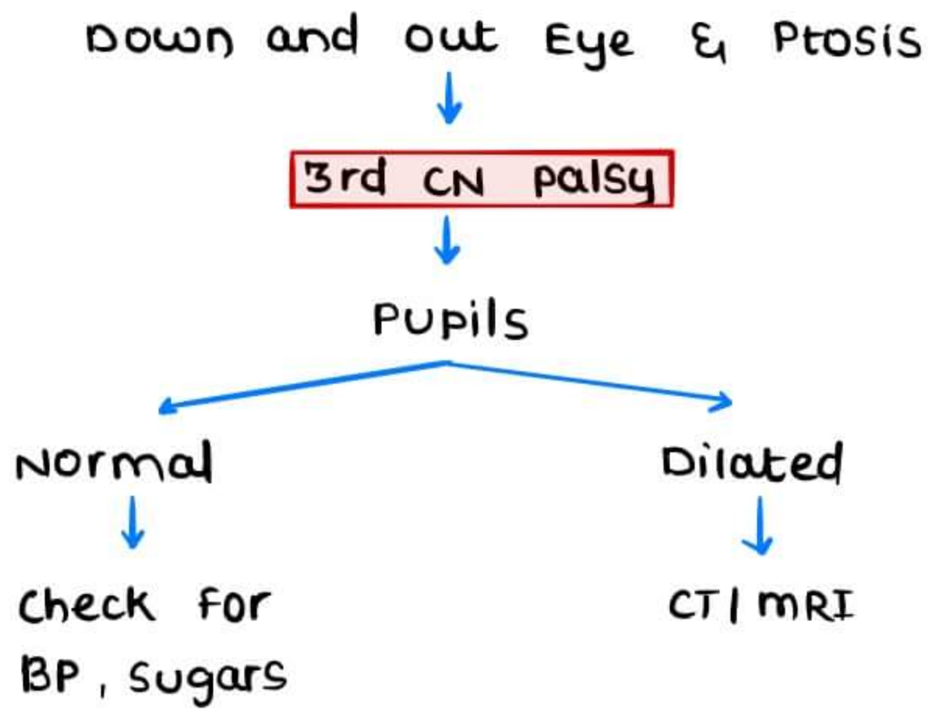
→ LR_6 , SO_4 are not supplied by 3rd nerve
   ↳ DOWN & OUT
→ Ptosis (as LPS also paralysed)
→ common causes → uncontrolled BP / HTN
→ TYPES
   1. Medical → by uncontrolled BP/HTN
      → Pupil Sparing (vascular insult doesn't involve superficial parasympathetic fibres)
   2. Surgical → by tumors or aneurysms
      → pupil involving (dlt compression of superficial parasympathetic fibres by tumor)

3rd nerve palsy
(Down & Out Eye)



Post Botax Alignment

→ DIAGNOSTIC FLOW CHART



4TH CN PALSY

→ diplopia in downward & inward gaze (reading, walking down stairs)
→ mc cause in children → congenital
→ mc cause in adults → Trauma
→ UP & IN Eye
→ Head tilt on opposite Shoulder (dlt Superior Oblique palsy)

4TH CN Palsy

→ ANATOMICAL PECULIARITIES OF 4ℏ CN

1. Longest Intra cranial nerve [75mm long]
2. Thinnest CN
3. 1st to rupture in closed head injury (Trivial injury)
4. Only CN to cross over to Opposite side
5. Only CN coming out from dorsal surface of brain

6ℏ CN PALSY

→ Lateral rectus paralysed } Eye In
→ unopposed medial rectus } Face out
→ common causes → ↑ ICT
↳ 6ℏ CN palsy may be a sign of ↑ ICN
↳ mc nerve damaged in ↑ ICT → 6ℏ CN


6ℏ CN Palsy

PARALYSIS OF NMJ [Neuro muscular junction]

MYASTHENIA GRAVIS

→ Seen in old men & young women
→ 1st ocular muscle to be paralysed → LPS
→ Fluctuating ptosis } ↑ by the end of the day
Diplopia }

→ Auto antibodies against Receptors
→ only skeletal muscles are affected
→ ciliary muscles & pupillary muscles are spared
↳ Accommodatn & pupil are normal
↳ helpful in differentiating it from 3rd CN Palsy

→ DOC → PYRIDOSTIGMINE

CONJUNCTIVITIS

NOTE - CONJUNCTIVA → 112 page

→ inflammation of conjunctiva

CLINICAL FEATURES

→ Bright red eye
→ no pain
→ Discharge
① Purulent discharge → Bacterial etiology
② Serous discharge → Viral etiology
③ Mucoid discharge → Allergic etiology

EPIDEMIC KERATO CONJUNCTIVITIS

→ caused by Adeno virus 8,19, 37
→ highly infectious
→ aka ① PINK EYE
② MADRAS EYE
③ SHIPYARD EYE


PINK EYE

ACUTE HEMORRHAGIC CONJUNCTIVITIS [AHC]


AHC

→ caused by Picarna virus
  → Enterovirus 70 [more common]
  → Coxsackie virus

→ highly infectious
→ Entero virus aka APOLLO VIRUS XI
  → discovered on 1969
  → disease caused is → APOLLO DISEASE

OPHTHALMIA NEONATORUM

→ occurs in neonates [First 28 Days]
Tears do not form till 1st 28 days
Tears in 1st month of birth → infectious

→ RED EYE IN
  → 1st day → chemical conjunctivitis
  → 2nd - 3rd day → Gonococcal conjunctivitis [most dangerous]
  → > 1 week → Chlamydia [mc cause]

  → Only conjunctivitis that causes blindness → Gonococcal conjunctivitis
    → can perforate cornea & causes blindness
    → CREDE'S METHOD
      → instilling 1% $AgNO_3$ as soon as child birth
      → chemoprophylactic against gonococcus
      → but it is toxic → causes chemical conjunctivitis
  → DOC for Prophylaxis → AZITHROMYCIN

KERATO CONJUNCTIVITIS

TRACHOMA / EGYPSIAN OPHTHALMIA

→ caused by chlamydia trachomatis - A, B, C
→ CHLAMYDIA TRACHOMATIS
  A, B, C → causes Trachoma
  D to K → causes Inclusion conjunctivitis
  L_1, L_2, L_3 → causes Lympho Granuloma Venereum [LGV]

→ commonest infective cause of blindness [1.4%]
→ Geographical distribution
  North Africa → Egypt, Libia, Tunisia, Algeria, Ethiopia, Somalia
  South Asia → India, Pakisthan, Bangladesh, Srilanka
  Middle East → Arab countries

Regions i high distribution in India [Endemic Trachoma]

North India

- → Punjab
- → Haryana
- → Uttar pradesh
- → Uttaranchal
- → Rajasthan
- → Gujarat

→ CLINICAL FEATURES

- → Lacrimation
- → Photophobia
- → Red eyes
- → Hall mark → SAGD GRAIN FOLLICLES
- → HERBET'S PITS Seen
- → ARLT'S LINE on upper palpebral conjunctiva seen
  - → ARLT'S TRIANGLE seen in Anterior uveitis
- → PANNUS seen [non specific sign]


Sago grain follicles


Herbet pits


Arlt's line


Pannus

→ SAFE STRATEGY by WHO

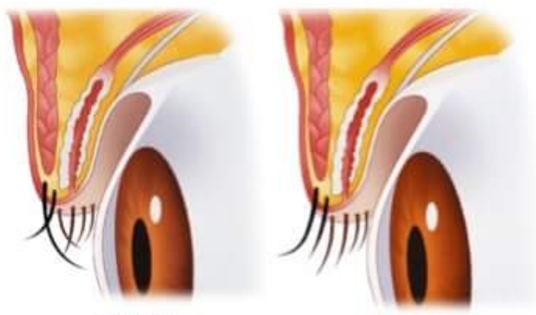
- S → Surgery for Trichiasis
- A → Antibiotics
  - → oral → AZITHROMYCIN [Doc]
  - → Topical → TETRACYCLINE , ERYTHROMYCIN
- F → Facial Hygiene
- E → Environmental sanitation

→ BLANKET / MASS THERAPY by WHO for Endemic areas

- → intermittent therapy
- → 1% Tetracycline ointment OD × 10 days continuously / 1m × 6 months
- → 1% TETRACYCLINE ointment BD × 5 days continuously / 1 month × 6 months

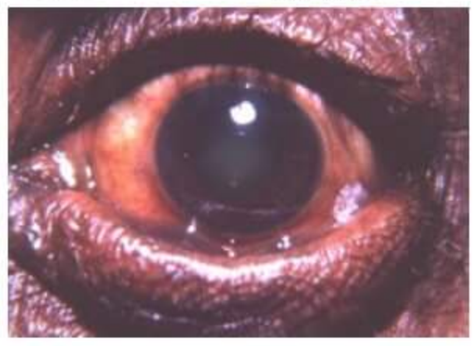
→ mc affected → children

2nd mc affected → Women



OTHER FEATURES

- → TRICHIASIS → posterior misdirectⁿ of eye lashes
- → TYLOSIS → Thickening of eye lid margin
- → MADAROSIS → Loss of eye lashes
- → ENTROPION → inward turning of eye lid margin
- → CORNEAL OPACITIES
  - → NEBULA → most superficial [max. discomfort]
  - → MACULA → half thickness
  - → LEUCOMA → Full thickness [max. loss of vision]


TYLOSIS


ENTROPION

WHO GRADING → FISTO CLASSIFICATION

I → F → > 5 Follicles in upper palpebral conjunctiva

II → I → Intense inflammation [max infectivity]

III → S → Scaring [healing started]

IV → T → Trichiasis

V → O → corneal Opacities

Nebula

GRADE I

GRADE II

GRADE III

GRADE IV

GRADE V

→ VISION 2020

→ Aim → 2020 by 2020 → $\frac{20\text{ feet}}{20\text{ feet}} = \frac{6\text{ metres}}{6\text{ metres}}$ by 2020

→ By eliminating

① Cataract → by Surgery

② Trachoma → by SAFE Strategy

③ Childhood blindness → by Vitamin A supplementation

④ Refractive error → by Spectacle correction

⑤ Oncocerciasis → not a problem in India

In India, We consider instead of Oncocerciasis

⑥ GLAUCOMA

⑦ DIABETIC RETINOPATHY

→ CSR → Cataract Surgical Rate

→ no. of cataract surgeries performed per million/year

→ GET 2020 → Global Eradication of Trachoma by 2020

SPRING CATARRH / VERNAL CATARRH / VERNAL KERATOCONJUNCTIVITIS [VKC]

→ Misnomer → Occurs in Summer, not in Spring [from April to October]

→ occurs in children

→ Allergic conjunctivitis

→ Severe itching present

→ COBBLE STONE PAPILLAE seen → Hallmark

→ Ropy discharge present

→ HORNER TRANTAS SPOTS seen

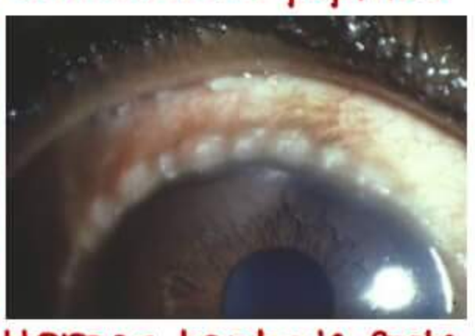
→ SHIELD ULCER seen

→ MAXWEL - LYON'S SIGN seen

→ on eversion of eye lid, pseudo - membrane formed by atmospheric heat

Cobble stone papillae

Shield ulcer



Horner tantra's Spots

→ TREATMENT

→ DOC → SODIUM CROMOGLYCOLATE [Mast cell stabilizer]

→ OLOPATADINE

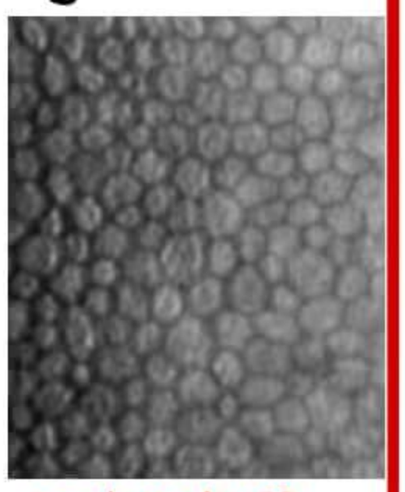
CORNEA

LAYERS [OUTER TO INNER]

1. Epithelium
2. Bowman's membrane
3. Stroma [Thickest, 90%]
4. Descemet's membrane
5. Endothelium

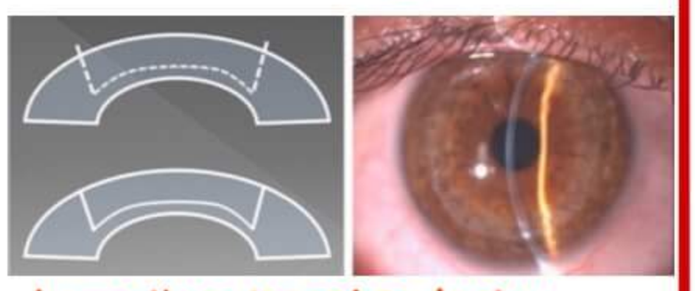
NEW LAYER → Pre descemet's layer / DUA'S Layer
↳ present blw stroma & descemet's membrane

→ corneal scar/ opacity results from damage to Bowman's membrane
→ Bowman's membrane can't regenerate
→ Descemet's membrane
↳ Only fungal infections can penetrate intact descemet's membrane
→ ENDOTHELIUM
↳ most important layer responsible for corneal transparency
↳ Single layer of cells
↳ active endothelial pumps pumps out aqueous humor
↳ irreparable → leads to irreversible corneal edema
↳ Specular microscope is used to count Endothelium cells
→ Average count → 3000 cells / mm^2
→ Every year → 0.5% of loss
→ critical density → < 500 cells / mm^2
→ For corneal donatn → >2000 cells / mm^2


Endothelium

< 500 cells / mm^3
↓
Irreversible corneal edema
↓
↓ vision
↓
Rx by corneal transplantatn
[keratoplasty]


Penetrating Keratoplasty


Lamellar Keratoplasty

KERATOPLASTY

PENETRATING KERATOPLASTY (PK)
Full thickness keratoplasty all 5 layers taken from donar

LAMELLAR KERATOPLASTY (LK)
partial thickness keratoplasty only outer layers taken from donar recipient retains his own endothelium Descemet's membrane Stroma ±
Donar provides Epithelium Bowman's membrane stroma ±

CORNEAL TRANSPLANT IS THE MOST SUCCESSFUL ORGAN TRANSPLANT IN THE BODY [cornea is avascular]
↳ LK is more successful than PK
↳ max. rejection is from Endothelium
↳ LK is Technically more demanding

INDICATIONS

PK [PENETRATING KERATOPLASTY]

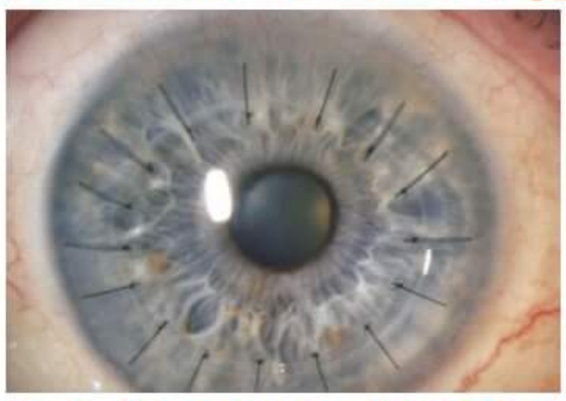
1. Pseudophakic Bullous Keratopathy [PBK] [mc -worldwide]
2. Non healing ulcer
3. corneal scar [mc indicatn in India]
4. Corneal Dystrophy
5. Keratoconus [mc indicatn in America]
6. Chemical injuries

Pseudophakic bullous Keratopathy

CORNEAL DONATIONS

→ Live person can't donate
→ 'PLEDGE' THE CORNEA → consent signed for corneal donatn
→ only 60,000 corneas are collected in India last year
→ India requires 3 - 3.5 lakh corneas
→ Inform spouse before pledging cornea
→ HLA matching not required
→ to be donated ī in 6 hrs after death
→ preserved in MK (Mc carey Kaufmann) media (preserves for 96 hrs)
→ contra Indications
  ↳ HIV
  ↳ Hepatitis B
  ↳ Septicemia
  ↳ Rabies
  ↳ Prions
  ↳ Retinoblastoma
  ↳ Metastatic Brain Tumor
  ↳ Leukemias
  ↳ Lymphomas
  ↳ Head & neck cancers

PK ī continuous sutures



PK ī interrupted sutures

CORNEAL ULCERS

→ break in corneal epithelium ī underlying necrosis of cells
→ Hypopyon formed
  ↳ dlt accumulatn of white blood cells (1st line of defence)
  ↳ steroids c/I
→ Earliest symptom → Photophobia
→ Pain
  ↳ most important symptom
  ↳ extremely painful (highest density of nerve endings present)
→ TYPES
  1. Bacterial
  2. Fungal
  3. Viral
  4. Acanthamoeba

ACANTHAMOEBA ULCER

→ PREDISPOSING FACTOR
  ↳ contact lens wearer
    → mc organism attacking contact lens wearer → Pseudomonas > Acanthamoeba
    → Acanthamoeba attaks contact lens that are washed ĩ water


Acanthamoeba ulcer

→ Ring shaped ulcer
→ pain out of proportion
→ forms Pseudodendrites

BACTERIAL KERATITIS

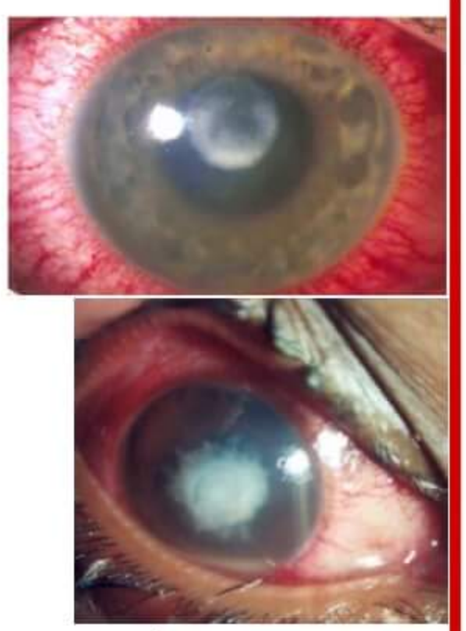
→ mcc in worldwide → Staphylococcus aureus
→ mc in India → Pneumococcus
→ ULCUS SERPENS, HYPOPYON CORNEAL ULER } Pneumococcal ulcer
→ PREDISPOSING FACTORS
  1. Foreign body in eye
  2. Slept ĩ contact lenses
  3. Trauma


Bacterial keratitis

→ Bacteria penetrating Intact Corneal Epithelium
  1. Corynebacterium
  2. Gonococcus
  3. Meningococcus
  4. Hemophilus
  5. Listeria
  6. Shigella

FUNGAL KERATITIS

→ No symptoms
→ Signs morethan symptoms
→ causes
  ↳ Aspergillus
  ↳ Fusarium
→ Slow growing
→ Predisposing factors
  1. Prolonged topical steroids
  2. Organic matter injury


Fungal Keratitis

→ Finger like projections
→ Feathery margins
→ satellite lesions
→ Hypopyon
  ↳ only non-sterile hypopyon
  ↳ non-mobile


Satellite lesions

VIRAL KERATITIS

→ caused by HSV [Herpes Simplex Virus]
  ↳ Epithelial Keratitis
  ↳ Stromal Keratitis


Dendritic Keratitis

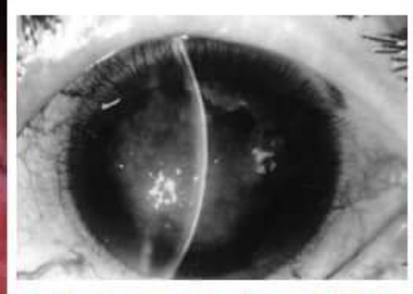
→ EPITHELIAL KERATITIS → DENDRITIC ULCER → GEOGRAPHICAL ULCER
  ↳ loss of corneal sensatⁿ

→ NEUROPARALYTIC KERATITIS
  ↳ seen in 7th CN palsy
  ↳ facial nerve supplies orbicularis (only muscle that closes the eye)
  ↳ causes Exposure Keratitis

→ NEUROTROPHIC KERATITIS
  ↳ 5th CN palsy
  ↳ 5th CN carries corneal sensations
  ↳ Foreign body on cornea causes ulcer

→ STROMAL / DISCIFORM KERATITIS
  ↳ forms disc of edema
  ↳ Herpetic stromal ulcer
  ↳ immunologic reaction
  ↳ DOC → Steroids


Hutchinson Rule


Disciform Keratitis

→ True dendrites are formed

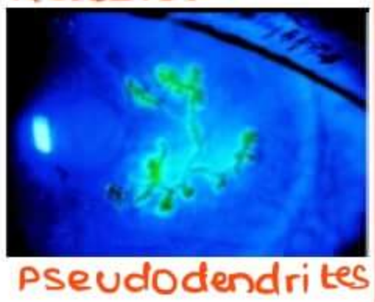
HERPES ZOSTER / SHINGLES

→ caused by varicella zoster
→ causes Chicken pox
→ reactivates from dormant state during immuno compromised states
→ zoster in young patient → sign of HIV
→ HUTCHINSON'S RULE
  → predicts Herpes zoster Ophthalmicus
  → vesicle on the tip of nose indicates the infectⁿ of eye too.
    ↳ Both the tip of the nose & eye are supplied by same branch of nasociliary nerve

→ Rx should be started ī in 72 hrs
  Failure of which leads to POST HERPETIC NEURALGIA
  ↳ most painful condition


Nummular Keratitis

→ Nummular Keratitis seen in Herpes zoster
→ Pseudodendrites are seen


Pseudodendrites

PSEUDODENDRIES SEEN IN
↳ Herpes zoster
↳ contact lens wearers
↳ Acanthamoeba

TREATMENT

CORNEAL ULCERS

1. Bacterial → Topical 4th Generatn Flouroquinolones (DOC)
   - ↳ moxifloxacin
   - ↳ Gatifloxacin
2. Fungal → Topical Natamycin (DOC)
3. viral → Topical Acyclovir / famacyclovir / valacyclovir
4. Acanthamoeba → PHMB (Poly Hexa Methylene Biguanide]

PAIN

CYCLOPLEGICS

1. Homatropine
2. Atropine

NON HEALING ULCER

CAUSES

1. uncontrolled DM (mc)
2. Foreign Body Incarceration
3. wrong diagnosis

TREATMENT FOR IMPENDING PERFORATION → Keratoplasty

TREATMENT FOR PERFORATED CORNEAL ULCER

1. Keratoplasty
2. For < 2mm perforations → CYANOACRYLATE GLUE (Biological glue)

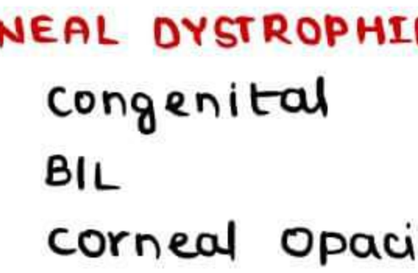
CORNEAL DYSTROPHIES

- → Congenital
- → BIL
- → Corneal Opacity
- → mostly do not progress
- → not alw any systemic conditions

TYPES

1. Epithelial 2. Stromal 3. Endothelial

STROMAL DISTROPHIES

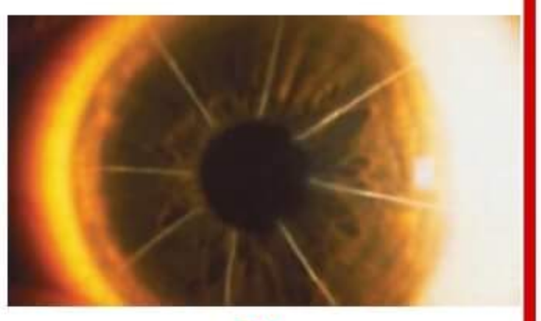
GRANULAR	LATTICE	MACULAR
AD	AD	ar
		
Bread crumbl popcorn like	line like corneal opacities	Discrete white → Diffuse opacities
Limbus is clear	Limbus is clear	Limbus is not clear
clear spaces + nt	clear spaces + nt	No clear spaces
Rx by Keratoplasty	Rx by Keratoplasty	Rx by Keratoplasty

Macular Dystrophy requires maximum Keratoplasty

REFRACTIVE SURGERY

PRINCIPLE → corneal curvature changed

- In Myopia → cornea is flattened
- In Hypermetropia → cornea is steepened
- In Astigmatism → Both done



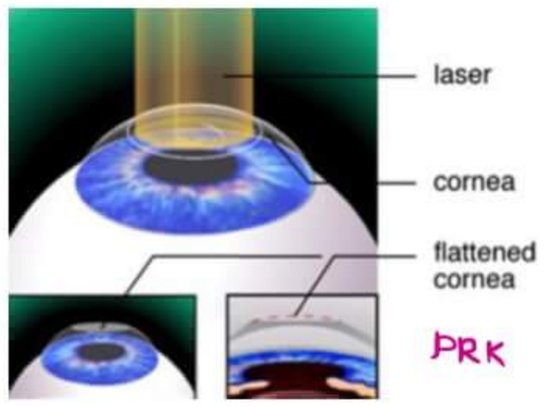
RK

RK (RADIAL KERATOTOMY)

- → Oldest refractive Sx
- → upto 3DS can be corrected
- → radial cuts are placed upto 90% of depth → cornea flattened
- → not recommended now (unwanted hypermetropia occurs)

PRK (PHOTO REFRACTIVE KERATECTOMY)

- → Excimer Laser used to ablate / remove tissue
  - ↳ Wave length → 193 nm
- → Upto −5DS can be corrected
- → DISADVANTAGES
  - → painful [post op pain +nt]
  - → Corneal scar occurs if Bowman's membrane is damaged



PRK

LASIK [LASER ASSISTED INSITU KERATOMILEUSIS]

- → Gold Standard
- → Myopia → − 10DS can be corrected
  hypermetropia → + 5DS can be corrected
  Astigmatism → ± 3.5DC can be corrected

- → Excimer Laser is used
- → PROCEDURE
  - ↳ Keratome cuts the cornea below the Bowman's membrane, in Stroma & creates a FLAP [attached to cornea one side]
  - ↳ corneal Stroma exposed by reflecting back the flap
  - ↳ corneal flattening done by Excimer laser & flap placed back
- → Not painful [epithelium not involved]
- → No corneal scarring [Bowmen's membrane not involved]
- → PACHYMETRY → measures corneal thickness
  - → Single most important factor to decide on LASIK
    - ↳ enough corneal thickness is required
- → ABSOLUTE C/I → Keratoconus

KERATOCONUS

- → conical cornea & very thin at the apex
  conical cornea & Normal thickness → ECTASIA
- → mostly seen in females
- → manifests at 20−30 yrs
- → HIO Frequent change of glasses

Keratoconus

1. MUNSON'S SIGN → Indentath on Lower Lid by Keratoconus while looking down

2. VOGT'S STRIAE → Vertical scratches in stroma of cornea

[VOGT'S TRIAD is Seen in Acute Angle closure Glaucoma]

3. FLEISCHER'S RING → Epithelial Iron ring surrounds the base of the cone

VOGT'S Striae

MUNSON'S SIGN

Kayser Fleischer ring

Fleischer ring

[KAYSER FLEISCHER RING → copper deposit on descemet's membrane
→ seen in Wilson's disease]

TREATMENT

1. Glasses
2. Contact lenses
   - ↳ Soft contact lenses should not be used
   - ↳ Semi soft contact lenses are prescribed

3. Corneal Hydrops
   - ↳ occurs in Keratoconus
   - ↳ Descemet's membrane perforates & aqueous humor enters corneal stroma
   - ↳ ī time hydrops heals ī central scarring
   - ↳ Rx by KERATOPLASTY

4. C_3R (CXR)
   - ↳ Corneal collagen crosslinking ī Riboflavin
   - ↳ New modality of Rx
   - ↳ Stops the progression & increases the strength of cornea ī covalent bonding (done by UV irradiation along ī Riboflavin)

EPISCLERITIS & DEVASTING SCLERITIS

EPISCLERITIS

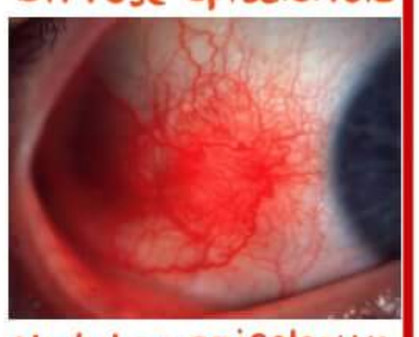
→ Benign, self limited inflammation of episclera

→ TYPES
   1. DIFFUSE
   2. NODULAR

→ DIFFUSE EPISCLERITIS
   - ↳ diffuse in distribution
   - ↳ Sectorial inflammatn

→ NODULAR EPISCLERITIS
   - ↳ Sectorial inflammatn & diffuse
   - ↳ nodule present

Diffuse episcleritis



Nodular episcleritis

→ CAUSE → Idiopathic

→ TREATMENT
  ↳ Reassurance
  ↳ Topical NSAIDs

SCLERITIS

→ Painful inflammatⁿ of sclera often alw Systemic disorders

→ TYPES
  1. ANTERIOR → Inflammatⁿ present anterior to insertion of rectus muscles
  2. POSTERIOR → Inflammatⁿ present posterior to insertion of rectus muscles

→ ANTERIOR SCLERITIS
  ↳ more common
  ↳ diffuse
  ↳ nodular
  ↳ necrotising ī inflammatⁿ
  ↳ necrotising ẗout inflammatⁿ


Anterior scleritis

→ RISK FACTORS
  ↳ Systemic Autoimmune disorders
  ↳ mc associatⁿ is Rheumatoid arthritis
  ↳ more in women in their 50's & 60's
  ↳ Pain
    → severe boring (characteristic)
    → exacerbated by movements
    → worse at night

  ↳ Redness & globe tenderness
  ↳ Characteristic violet blue colour ẗ scleral edema & dilated vessels
  ↳ Phenylephrine drops can not blanch vessels

→ TREATMENT
  1. Oral NSAIDS
  2. Systemic steroids
  3. Immuno modulatory drugs [methotrexate/ cyclosporine/ cyclophosphamide]

NECROTISING SCLERITIS

→ more severe & destructive form
→ may lead to loss of eye dlt perforatⁿ
→ Intense pain, out of proportⁿ
→ White avascular areas surrounded by inflammed sclera
→ Rᵩ by Systemic immunotherapy & scleral grafting

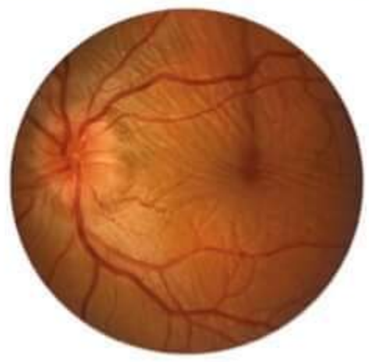

Necrotising scleritis

SCLEROMALACIA PERFORANS

→ necrotising scleritis ẗout inflammatⁿ (minimal signs & painless)
→ alw long standing Rheumatoid arthritis
→ sclera thins, dark uveal tissue visible
→ staphloma develops if IOP elevated
→ Eyes rupture ẗ minimal trauma


Scleromalacia perforans

POSTERIOR SCLERITIS



→ difficult to diagnose

→ pain, tenderness
proptosis
visual loss & Restricted motility

→ Choroidal folds, exudative retinal detachment, papilledema, Angle closure glaucoma

→ B scan usG → T Sign

→ Rx by Systemic steroids, cyclophosphomide

EPISCLERITS	SCLERITIS
→ no pain	→ painful
→ Bright red in colour	→ violet blue in colour
→ no scleral edema	→ Scleral edema ⊕
→ no a/w systemic disorders	→ a/w RA, SLE etc
→ by 10% Phenylephrine drops vessels undergo blanching	→ by 10% Phenylephrine drops vessels do not blanch

TRAUMA

OCULAR TRAUMA

MECHANICAL TRAUMA

→ MECHANISM

Mechanical Trauma
↓
Antero posterior compression of Eye Ball
↓
Equitorial Expansion of Eye Ball

→ ANTERO POSTERIOR STRUCTURES OF EYE BALL

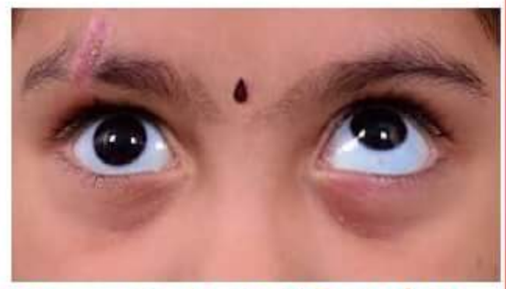
1. Orbit
2. Sclera
3. cornea
4. Anterior chamber
5. Iris
6. Ciliary Body
7. Choroid
8. Lens
9. Vitreous
10. Retina
11. Optic nerve

SUB CONJUNCTIVAL HAEMORRHAGE

↳ no loss of vision
↳ no pain
↳ Rx by Reassurance (10-15 days resolves)
↳ may mask
1. Conjunctival laceration
→ seal by itself if < 1cm
2. Scleral rupture

Sub conjunctival Haemorrhage

BLOW OUT FRACTURE OF ORBIT


Blow out # of orbit

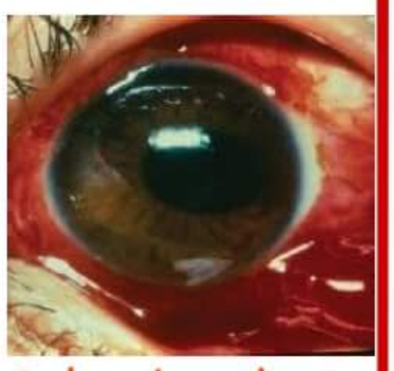
→ mc # of Orbit
→ mcly involves → Inferior wall
  ↳ medial wall is thinnest but it is supported by Ethmoidal sinus (honey coomb structure by trabeculae gives support)
  ↳ inferior wall supported maxillary sinus [hollowed out sinus, not much support]

→ Entrapment of inferior rectus in inferior wall # → Not able to look up
→ FEATURES
  1. Enophthalmous
  2. Diplopia
  3. Infra orbital nerve anesthesia

→ OCULO CARDIAC REFLEX
  1. Bradycardia
  2. Nausea
  3. vomiting

  ↳ seen in children

SCERAL RUPTURE


scleral rupture


Seidel's Test


corneal perforat^{n}


hyphema


Black Ball hyphema

→ most dangerous
→ immediate loss of vision → ↓ IOP
→ dlt trauma by blunt object
→ often by contre coup injury
→ Inside out mechanism of trauma [↑IOP]
→ Tissue Extrusion occurs
→ peeking of pupil indicates scleral rupture
→ SEIDEL'S TEST
  ↳ tells us about the site of rupture
  ↳ tear film ī fluorescein dye under cobalt blue illumination
  ↳ aqueous leakage can be elicited

CORNEAL PERFORATION

→ most commonly by metallic injuries
→ If it is healthy, then immediately Suturing done after repositioning of healthy iris

HYPHEMA

→ Blood in the Anterior chamber → ↑ IOP
→ CLASSIFICATION

GRADE 0 → micro hyphema
GRADE 1 → blood fills upto 1/3 rd of Anterior chamber
GRADE 2 → blood fill up from > 1/3 rd to 1/2 of AC
GRADE 3 → blood fill up from > 1/2 to almost full
GRADE 4 → Total hyphema → Full & red in colour
Black Ball / B-Ball hyphema → Full & black in colour

→ Black Ball hyphema
  ↳ indicates the compromise in circulation & Lack of oxygenation
  ↳ have higher chances of developing glaucoma

→ MANAGEMENT
  1. Irrigation of AC
  2. Surgical
    ↳ Indication → IOP → > 50 mm x 5 days

→ complication
  → Rebleeding
    ↳ mc time period → 2-5 days of initial hyphema
    ↳ a/w ↑ IOP

ANGLE RECESSION GLAUCOMA

→ PATHOGENESIS

Mechanical trauma (Blunt trauma)
↓
Anterior face of ciliary body rupture
↓
separatn blw longitudinal & circular fibres of ciliary body [evident on gonioscopy as "WIDENING OF CILIARY BODY BAND"]
↓
ANGLE RECESSION
↓
ANGLE RECESSION GLAUCOMA (20-50% of angle recession)
  ↳ not dlt angle recession perse
  ↳ dlt force causing damage to trabecular meshwork & leading to trabecular meshwork fibrosis
    → causes obstructn to trabecular outflow → ↑ IOP
  ↳ Diagnosed by Gonioscopy


Angle Recession Glaucoma

→ if > 180° Angle recession → more chances of developing Glaucoma

VOSSIOUS RING

→ imprint of the pupil on the anterior lens capsule
→ diameter is slightly smaller than pupil (dlt pupillary constrictn during trauma)


VOSSIOUS RING

SPHINCTERIC TEARS

→ traumatic mydriasis
→ presented ī glare
→ irreversible changes
→ advised to wear glasses


Sphincteric tears

IRIDODIALYSIS

→ Dialysis = separation from root
→ converts pupil into 'D' shaped pupil


Iridodialysis

SYMPATHETIC OPHTHALMITIS

→ CB injuries cause highest amount of sympathetic ophthalmitis
  ↳ Dangerous area of the eye → Ciliary Body
→ DALEN - FUCHS NODULES are seen
  ↳ Lymphocytic infiltrations on choroid


Sympathetic ophthalmitis

CHOROIDAL RUPTURE

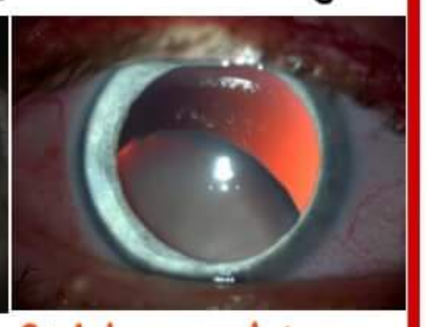
→ common
→ Rupture involves
  RPE
  choroid
  Bruch's membrane
→ crescent / curvilinear yellowish white rupture concentric to optic disc margin


Choroidal rupture

SUBLUXATED LENS

→ partial displacement of lens
→ either anterior into AC
  posterior into vitreous


Rosette cataract


Subluxated lens

ROSETTE CATARACT → petal like, present in posterior part of lens

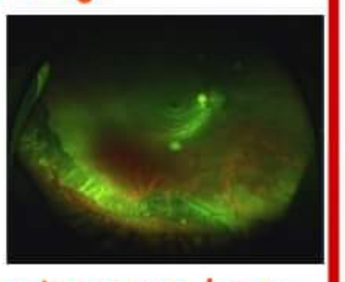
VITREOUS HAEMMORRHAGE

→ sudden painless loss of vision
→ mcc in young people → trauma
→ mcc overall → diabetic Retinopathy
→ Hallmark / pathognomic → Vitreous Base Avulsion [BUCKET HANDLE TEAR]


Vitreous Haemorrhage

→ ATTACHMENTS OF VITREOUS
  ↳ macula
  ↳ optic disc margin
  ↳ Blood vessels of retina
  ↳ vitreous base (Strongest attachment)


Retinal detachment


Vitreous base avulsion

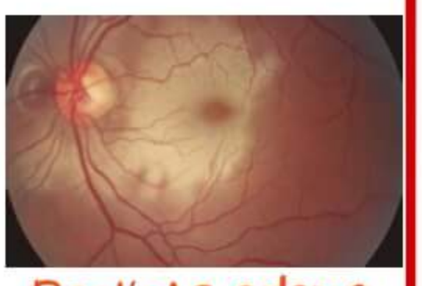
RETINAL DIALYSIS

→ Single most common retinal finding in ocular trauma
→ mc site → Inferotemporal retina > Supero nasal retina
→ can lead to Retinal detachment


Retinal dialysis

BERLIN'S EDEMA

→ causes creamy whitish patch around the macula dt intracellular edema ī cherry Red Spot
→ post traumatic Macular edema
→ aka COMMOTIO RETINAE
→ Rx by Systemic steroids


Berlin's edema

TRAUMATIC OPTIC NEUROPATHY

Q Which part of ON is mc damaged in TON?

A PARTS OF ON (optic nerve)

1. Intra ocular
2. Intra orbital
3. Intra canalicular [mc injured]
4. Intra cranial


Traumatic optic neuropathy

→ occurs after bike accidents

→ occurs in injury to the lateral 1/3rd & medial 2/3rd of eyebrow [indirect trauma]

OPTIC NERVE AVULSION

→ optic nerve get separated from sclera & retracted back into dura

→ immediate loss of vision occurs


Optic nerve avulsn

CHEMICAL INJURIES

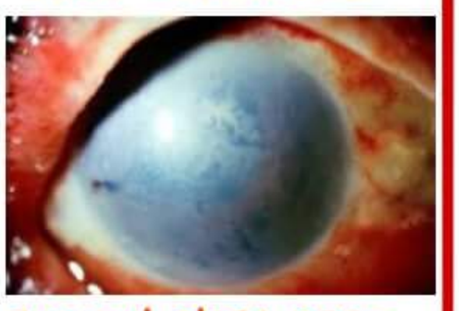
TRUE OCULAR EMERGENCIES IN OPHTHALMOLOGY

1. Chemical Injuries
2. CRAO


Total cicatrizatn

CHEMICAL INJURIES

1. ACID INJURIES
2. ALKALI INJURIES


Ground glass appearance of acid burn

ACID BURNS

→ Less destructive than alkalies
  ↳ acid coagulate & precipitate proteins on contact
  ↳ creating barrier preventing deeper penetration

  ↳ EXCEPTION → Hydrofluoric Acid, fluoride ion penetrates deep into tissues & cause severe injury

→ damaged by Acids, pH < 4

→ Ground glass appearance

→ Other Acids → Sulphuric Acid, hydrochloric Acid

ALKALI BURNS

→ Alkalies are Lipophilic
  ↳ penetrate membranes through saponification of membrane lipids
  ↳ Hydroxyl ions denature collagen matrix of cornea
  ↳ Tissue undergo liquifactive necrosis, triggers release of proteolytic enzymes, cascading damage

→ Alkalies can reach AC in 15 seconds

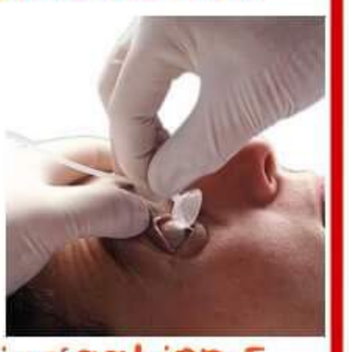
→ Ex: Ammonia, Calcium hydroxide
  ↳ seen in cement, Lime


alkali burn

DUA CLASSIFICATION (2001)

GRADE	PROGNOSIS	Clock hours of Limbal involvement	conjunctival involvement	Analog scale
I	very good	0	0%	0 | 0%
II	Good	⩽ 3	<30%	0.1-3 | 1-29.9%
III	Good	> 3-6	>30-50%	3.1-6 | 31-50%
IV	Good to Gaurded	>6-9	>50-75%	6.1-9 | 51-75%
V	Gaurded to poor	> 9-12	75-<100%	9.1-11.9 | 75.1-99.9%
VI	very poor	12	100%	12 | 100%

Limbal ischemia



irrigation t morgan lens

MANAGEMENT

EMERGENCY MANAGMENT

→ Irrigation ī H_2O
- ↳ principle → dilution of alkali
- ↳ minimum of 30 minutes irrigation recommended
- ↳ MORGAN LENS can be used for irrigatⁿ
- ↳ done till the pH of conjunctival surface reaches to 7.0 to 7.2
- ↳ single most important procedure to save the eye

→ Removal of Residual chemical debris by double eversion technique

RECOMMENDED TREATMENT

→ Based upon grade of injury

→ Topical antibiotic → Fluoroquinolone
- ↳ GRADE I - III → Erythromycin
- ↳ GRADE IV → moxifloxacin, Gatifloxacin

→ Cycloplegic → Homatropine / Atropine

→ Topical steroids → Prednisolone acetate
- ↳ for first 10 days to control inflammatⁿ
- ↳ tapered by Day 14, to minimise corneal melting

→ Doxycycline inhibits MMP (Matrix metallo proteinases)

→ Anti glaucoma drugs

→ Topical 10% / oral high dose vitamin C (2gm/ Day)

→ Tear supplements

MECHANISMS OF GLAUCOMA

→ most important preventable complicatⁿ, occurs in 75%, mi for visual outcomes

→ Acute rise
- ↳ dlt collagen shrinkage & contractⁿ
- ↳ ↑ in uveal & episcleral Flow

→ Long term
- ↳ dlt Trabeculitis
  - PAS (peripheral anterior Synachiae)
  - Steroid induced

CONJUNCTIVA

PARTS

① BULBAR CONJUNCTIVA → covers the sclera but not the cornea
② PALPEBRA CONJUNCTIVA → covers the upper & lower eye lids
③ FORNICEAL CONJUNCTIVA → Junction of bulbar & palpebral conjunctiva

→ Contains GOBLET CELLS
  → Secrets MUCUS
  → mucin stabilizes the tear film
  → max. no. present at infero-nasal conjuctiva
  → deficiency leads to Dry eye


conjunctival xerosis


Bitot's Spot


corneal xerosis


keratomalacia [X3B]


corneal scarring


xerophthalmic fundus

WHO CLASSIFICATION OF VITAMIN A DEFICIENCY [XEROPHTHALMIA]

TABLE 2. WORLD HEALTH ORGANIZATION RE-CLASSIFICATION OF XEROPHTHALMIA SIGNS

Classification	Ocular Signs
XN	Night blindness
X1A	Conjunctival xerosis
X1B	Bitot's spots
X2	Corneal xerosis
X3A	Corneal ulceration-keratomalacia involving one-third or less of the cornea
X3B	Corneal ulceration-keratomalacia involving one-half or more of the cornea
XS	Corneal scar
XF	Xerophthalmic fundus

→ upto X2 [corneal xerosis], we can reverse it vitamin A
→ TREATMENT
  → VITAMIN A SUPPLEMENTATION
    → 2,00,000 IU for children above 1 year
    → at 0, 1, 14 days intervals
    → [0 → day of presentation]
    → 14 th day dose is for liver supplementation

CONJUNCTIVAL REACTIONS

① FOLLICLES
  → collections of Lymphocytes
  → Seen in → Viral infections
    → Chlamydeal infections [Trachoma]
    → Toxic conditions [BRIMONIDINE THERAPY]


Follicles

② PAPILLAE
  → elevation of conjunctiva ± central vascular core
  → Seen in → Allergic conditions [Vernal catarrh]
    FB reaction


PAPILLAE