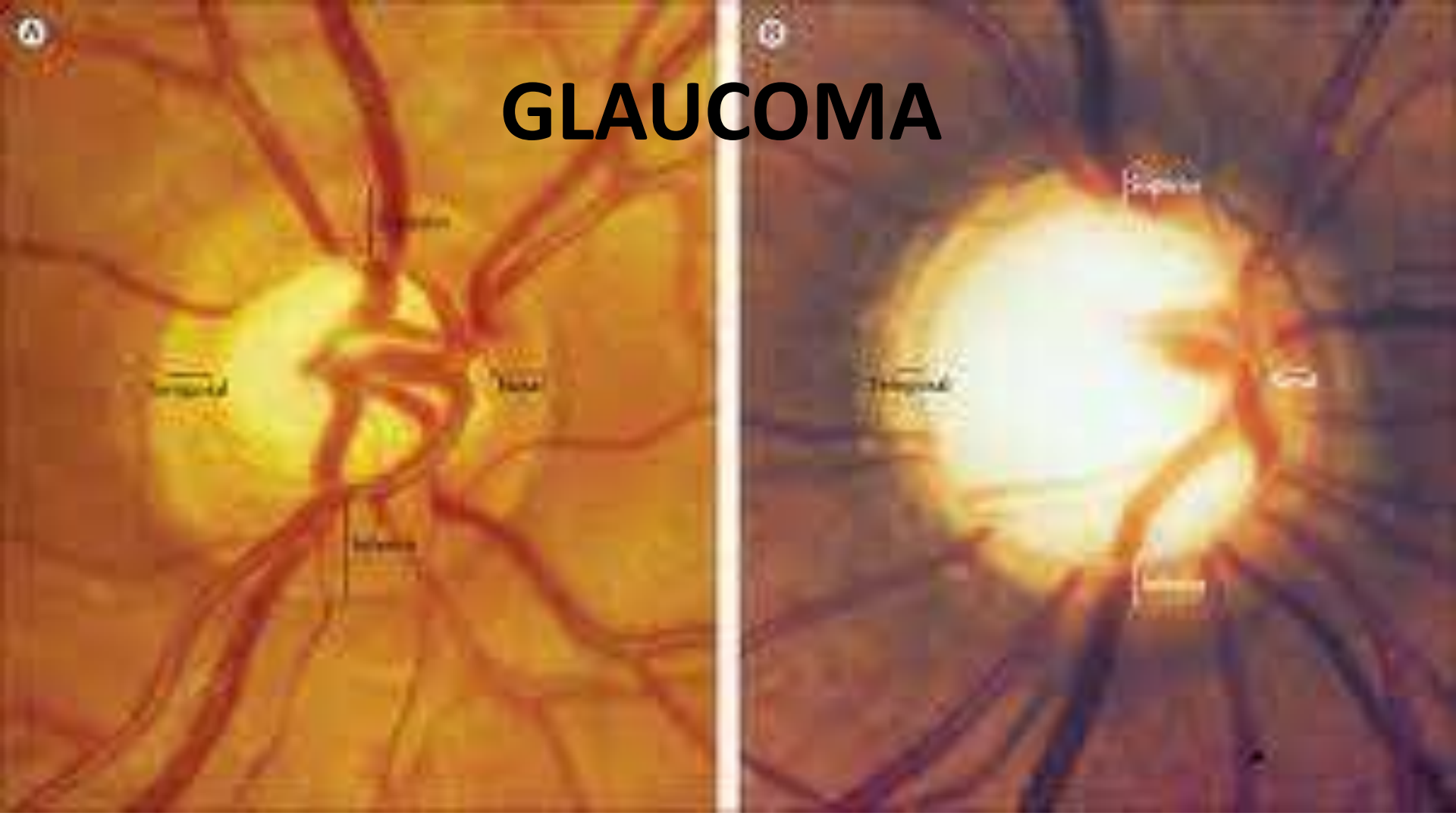
GLAUCOMA



Dr Smita Pawar
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Aqueous humour

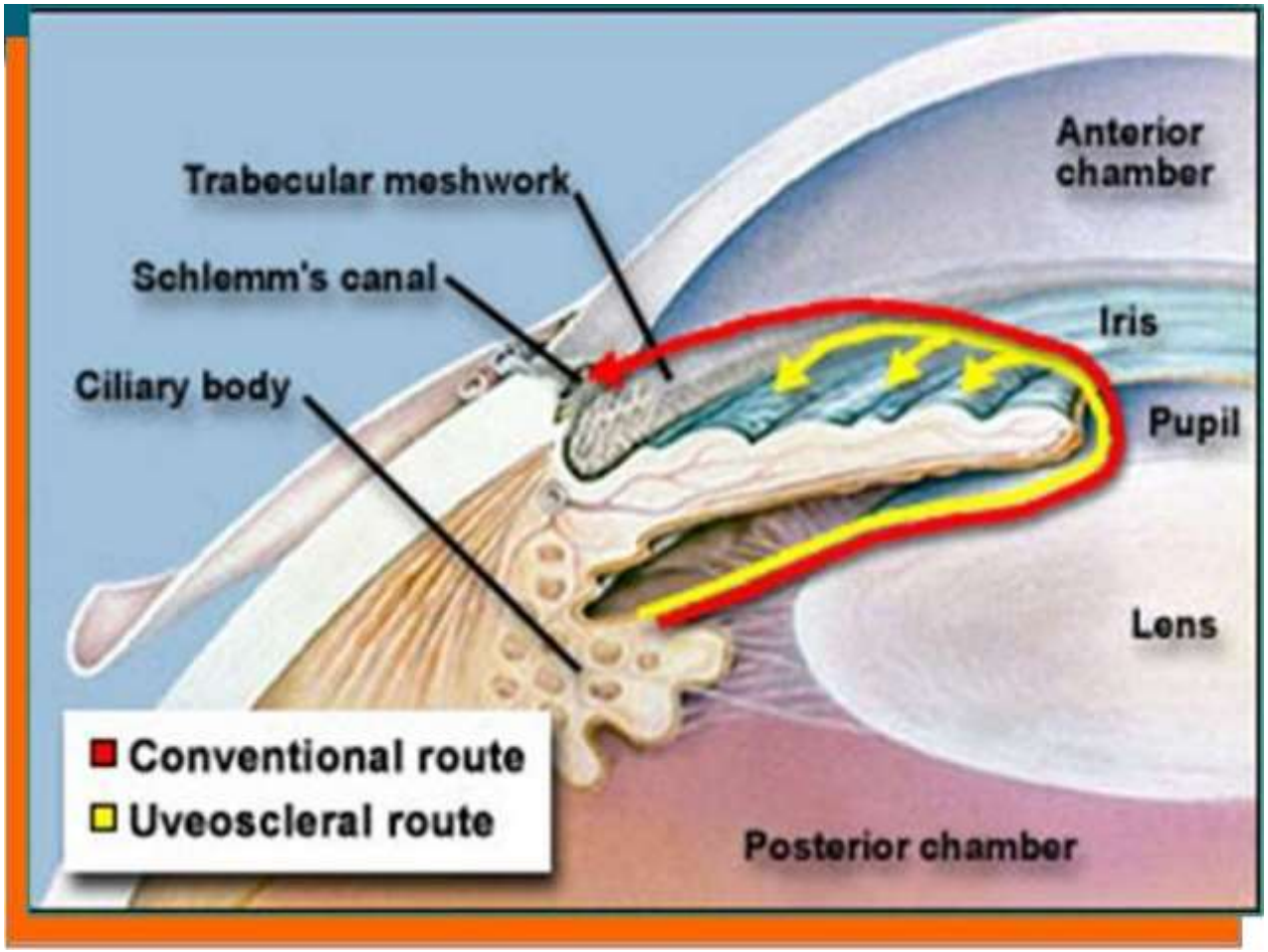
Formation

- *Active secretion-pressure independent but energy dependent 70%*
- Ultrafiltration-20% pressure dependent
- Diffusion-10%
- Composition-similar to plasma except high concentration of ascorbate,pyruvate and lactate.low conc of protein,urea,glucose

function

- Transparency
- Intraocular pressure
- Nutrition and removal of metabolites from avascular cornea and lens
- Ant chamber-0.25ml
- Post chamber-0.06ml
- Normal IOP-16±2.5mm of Hg

Drainage –trabecular and uveoscleral outflow



Trabecular or conventional (90%) flow

Trabecular meshwork

Uveal

Corneoscleral

Juxtacannalicular

Schlemms canal

Collector channels

aqueous veins and

episcleral veins

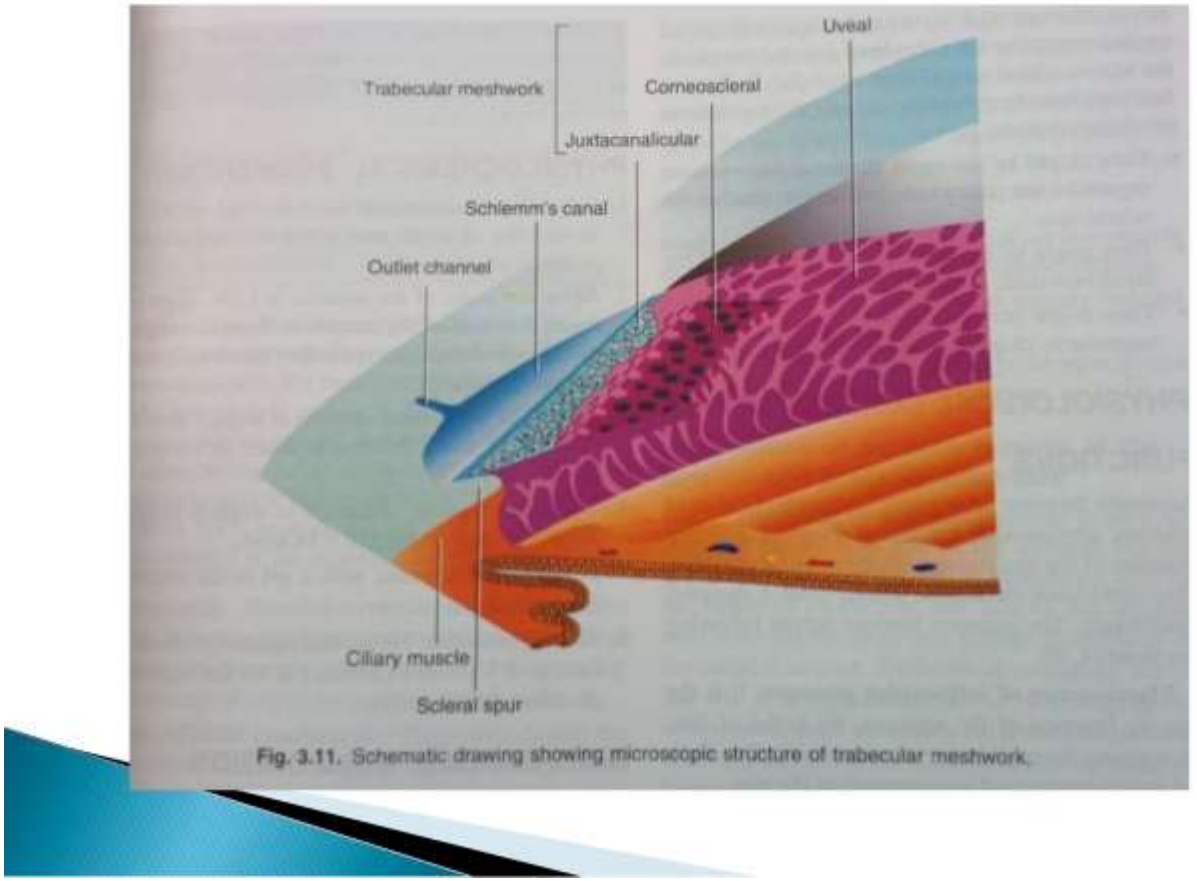
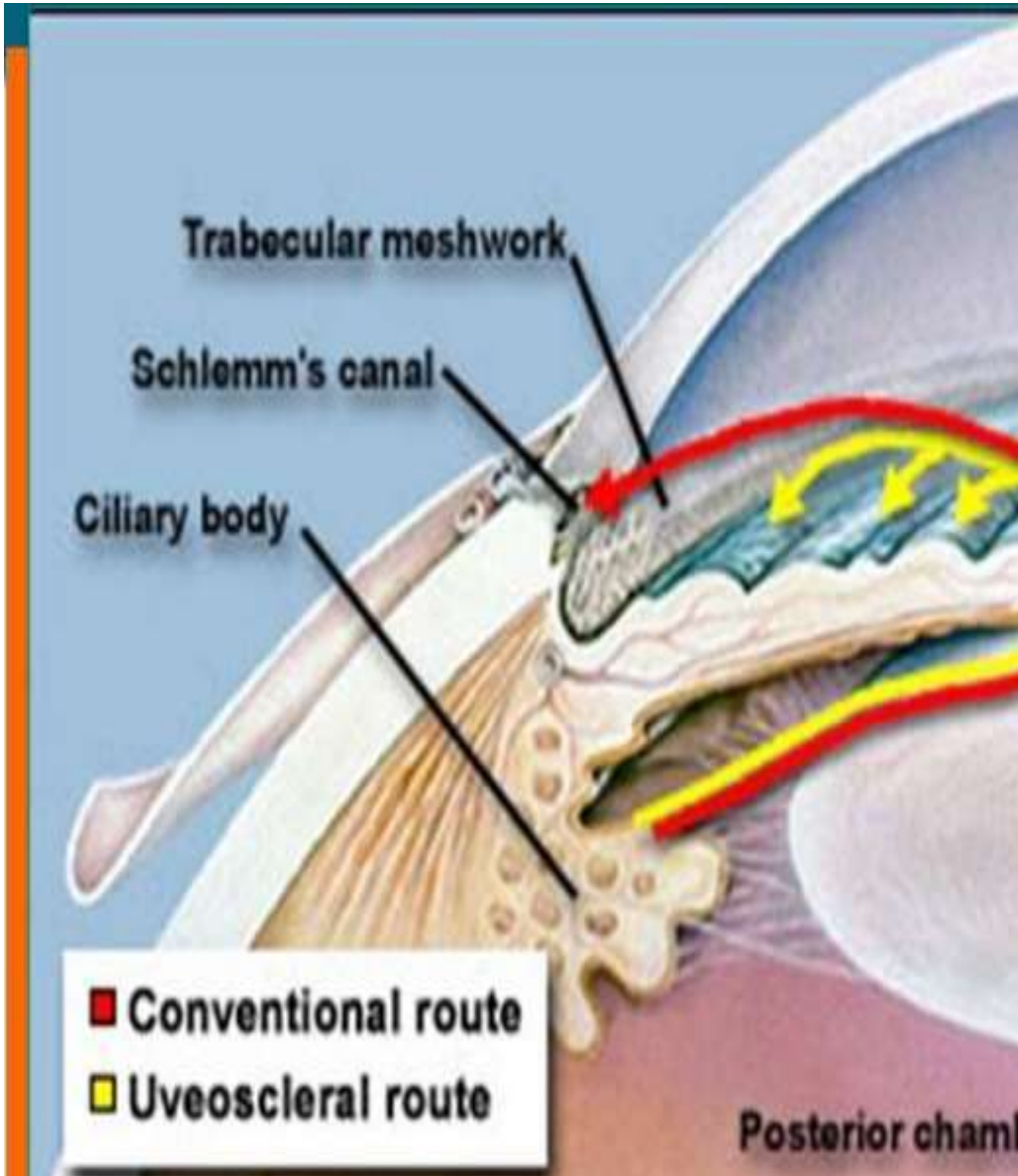


Fig. 3.11. Schematic drawing showing microscopic structure of trabecular meshwork.

Uveoscleral (unconventional)outflow

- Ciliary body to choroid
- suprachoidal space,
- episcleral tissue.
- 10%aqueous outflow



Factors influencing IOP

- Local factors
- Rate of aqueous formation
- Outflow resistance
- Pupillary dilatation
- Episcleral venous pressure

- General factors
- Hereditary
- Age
- Sex
- B P
- Osmotic pressure
- Diurnal variation±5mm
- Postural variation
- Drugs-anesthesia
- Mechanical pressure on globe

IOP measurement

Manometry

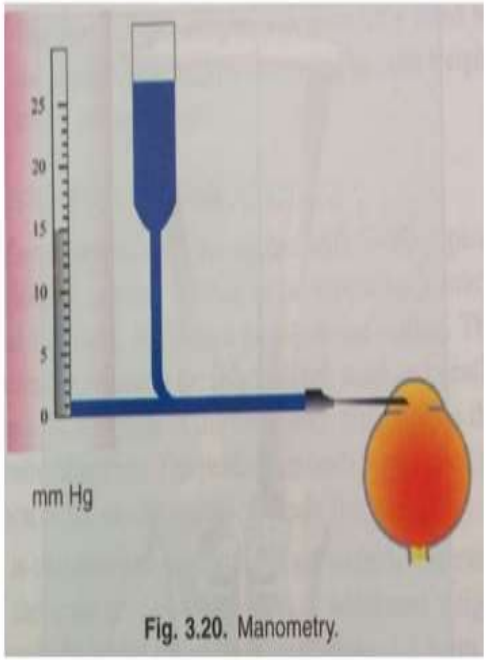


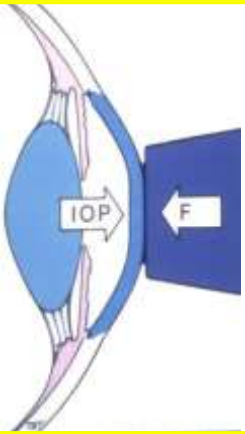
Fig. 3.20. Manometry.

Tonometry

- It is an indirect method of measuring IOP using a specialised instrument called tonometer.
- There are two types of tonometry:

1. Indentation or Impression tonometry
2. Applanation tonometry

Tonometers



Goldmann

Contact applanation

Perkins

Portable contact applanation

Schiotz

Contact indentation

Air-puff

Non-contact indentation

Pulsair 2000 (Keeler)

Portable non-contact applanation

Tono-Pen

portable contact applanation

Glaucoma

- Chronic progressive optic neuropathy caused by a group of ocular conditions which lead to damage to the optic nerve with loss of visual function.
- **Ocular hypertension-**↑IOP without glaucomatous damage.
- **Low tension or Normotensive glaucoma**-low or normal IOP with glaucomatous disc or field changes.

PATHOGENESIS OF GLAUCOMA

- **Ischemic Theory**
- **Mechanical Theory**
- **Apoptosis Theory**
- **Immune Theory-** Autoantibodies, Paraproteinemia, Antiglutathion S Transferase Antibodies

Diagnosis

- Optic nerve head changes
- Rise in intraocular pressure
- Visual field changes

Investigations

- SLE for ant segment examination for diagnosis of secondary glaucoma
- Disc changes
- Gonioscopy
- Tonometry
- Diurnal variation
- Provocative test
- Anterior segment OCT
- UBM
- Perimetry
- Nerve fibre layer analyser by scanning laser polarimetry

Provocative test

- **FOR POAG** -Water drinking test-fasting patient baseline IOP –I Lit water -IOP at every 15 min for 1 hour.max rise after 15 to 30 min and normal in 60 minutes.>8 mm rise pathologic

- **FOR PACG-**

- Prone-Dark room test –eyes open >8MM significant

- Mydriatic test –weak mydriatic to keep pupil in mid dilated position. >8 mm positive.

Classification of glaucoma

A. Congenital and developmental (without associated anomalies)

Developmental glaucoma (with associated anomalies)

B. Primary adult glaucoma

Primary open angle glaucoma(POAG)

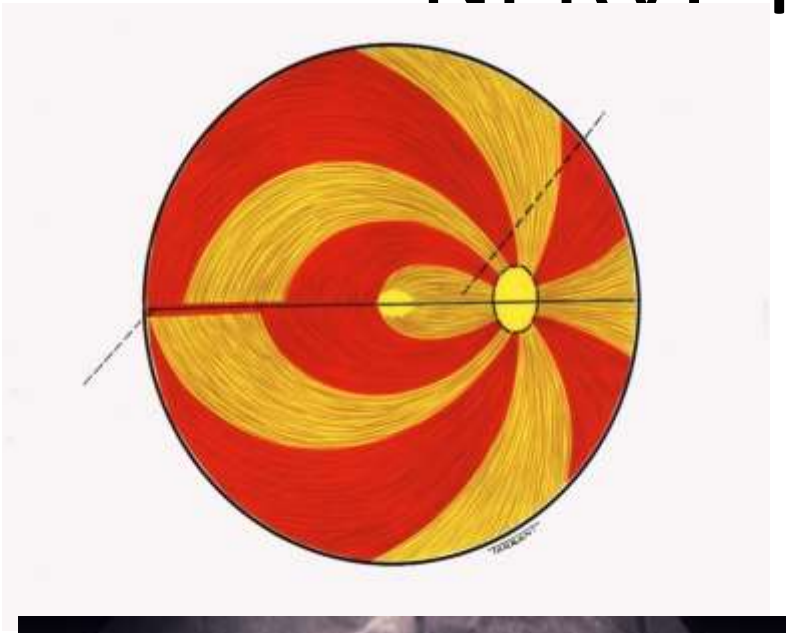
Primary angle closure glaucoma(PACG)

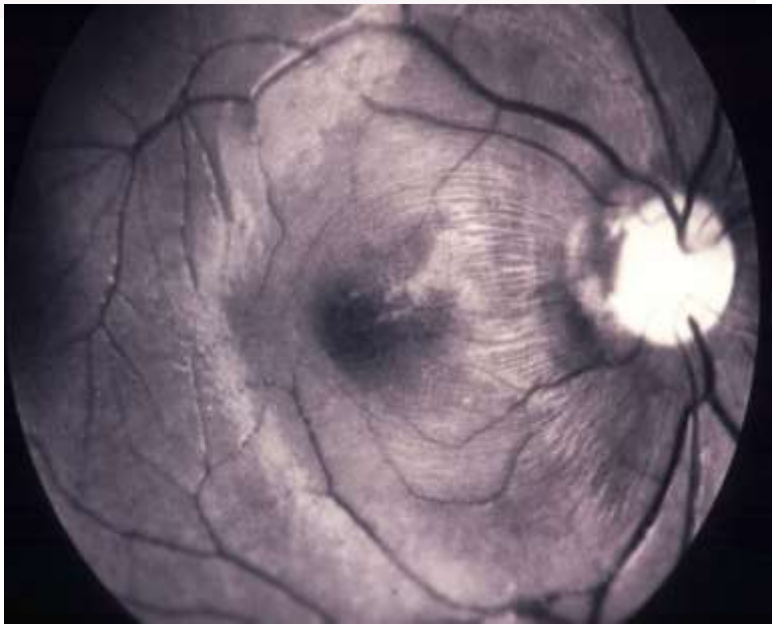
Primary mixed mechanism glaucoma

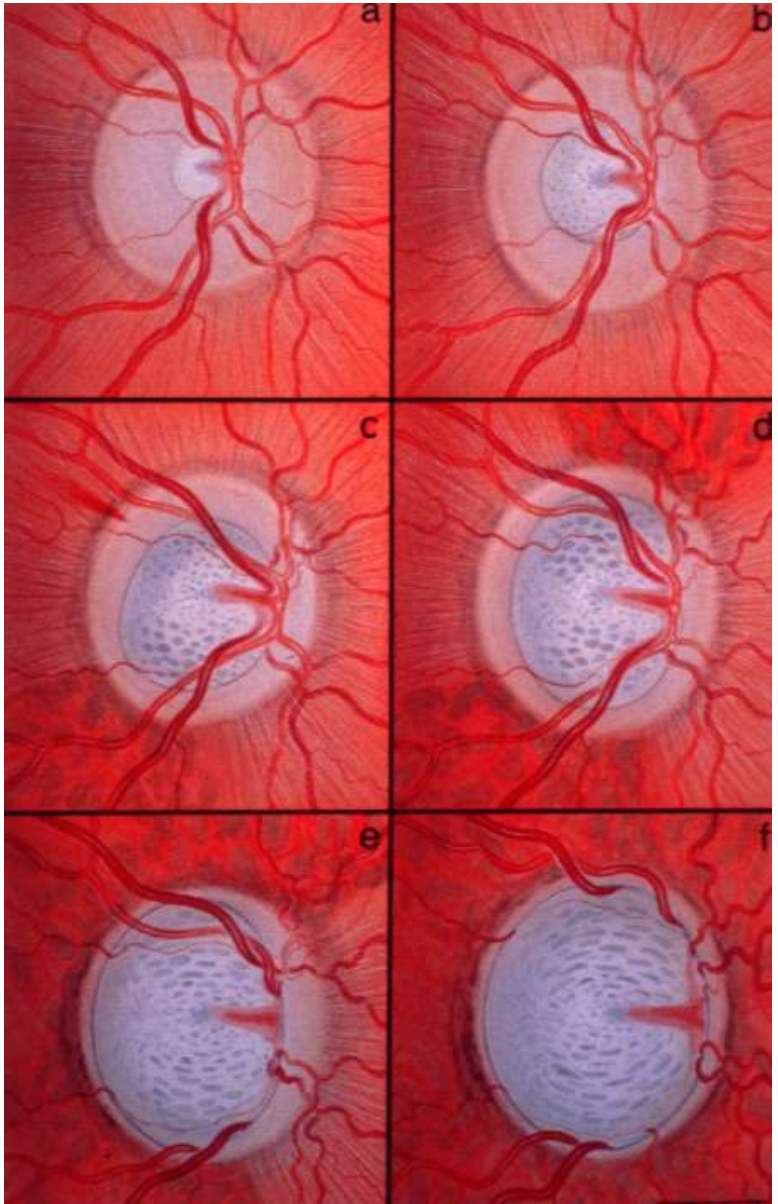
C. Secondary glaucoma

Secondary Glaucoma

NERVE FIBRE LAYER



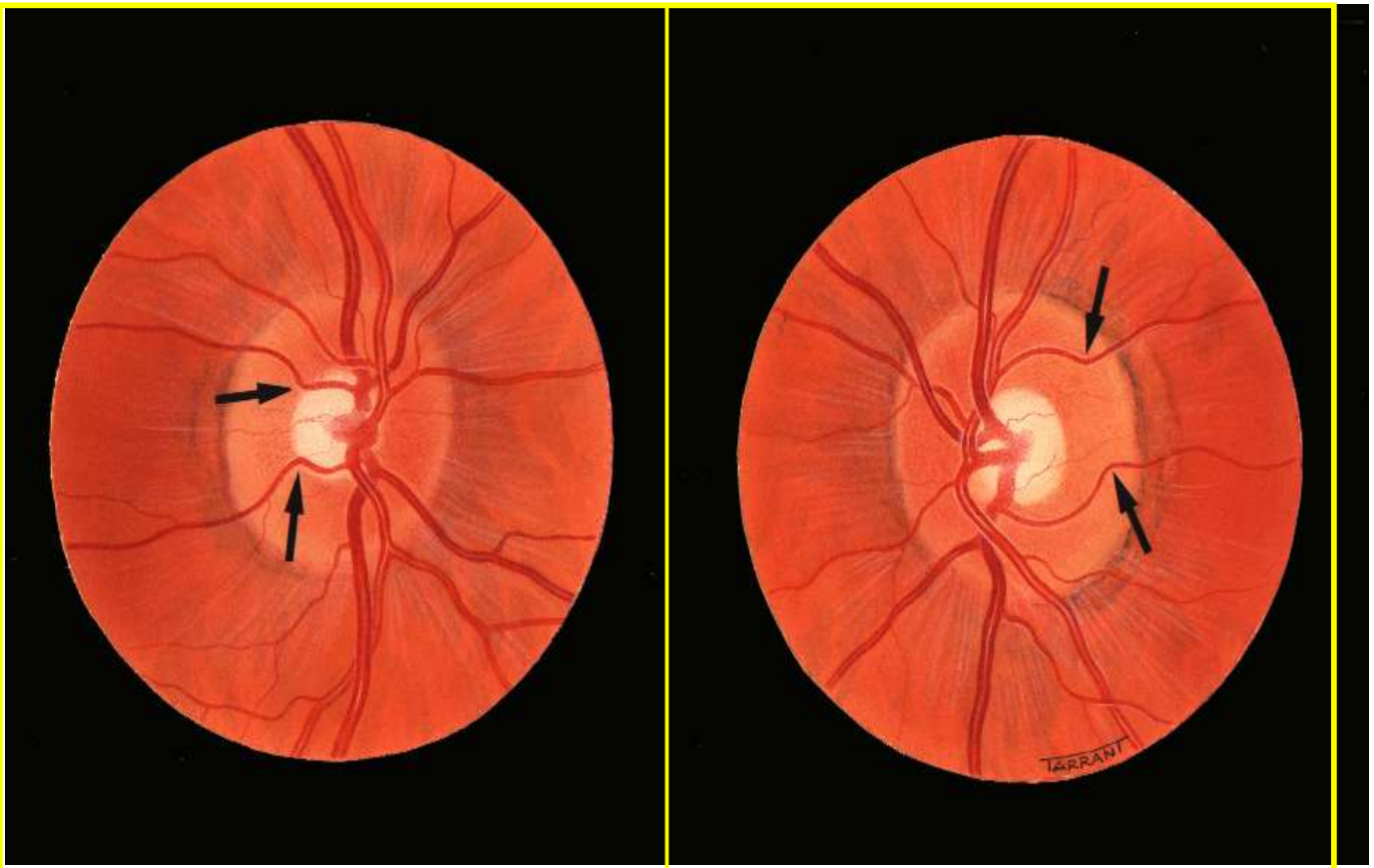




Pallor and cupping

Pallor - maximal area of colour contrast

Cupping - bending of small blood vessels crossing disc



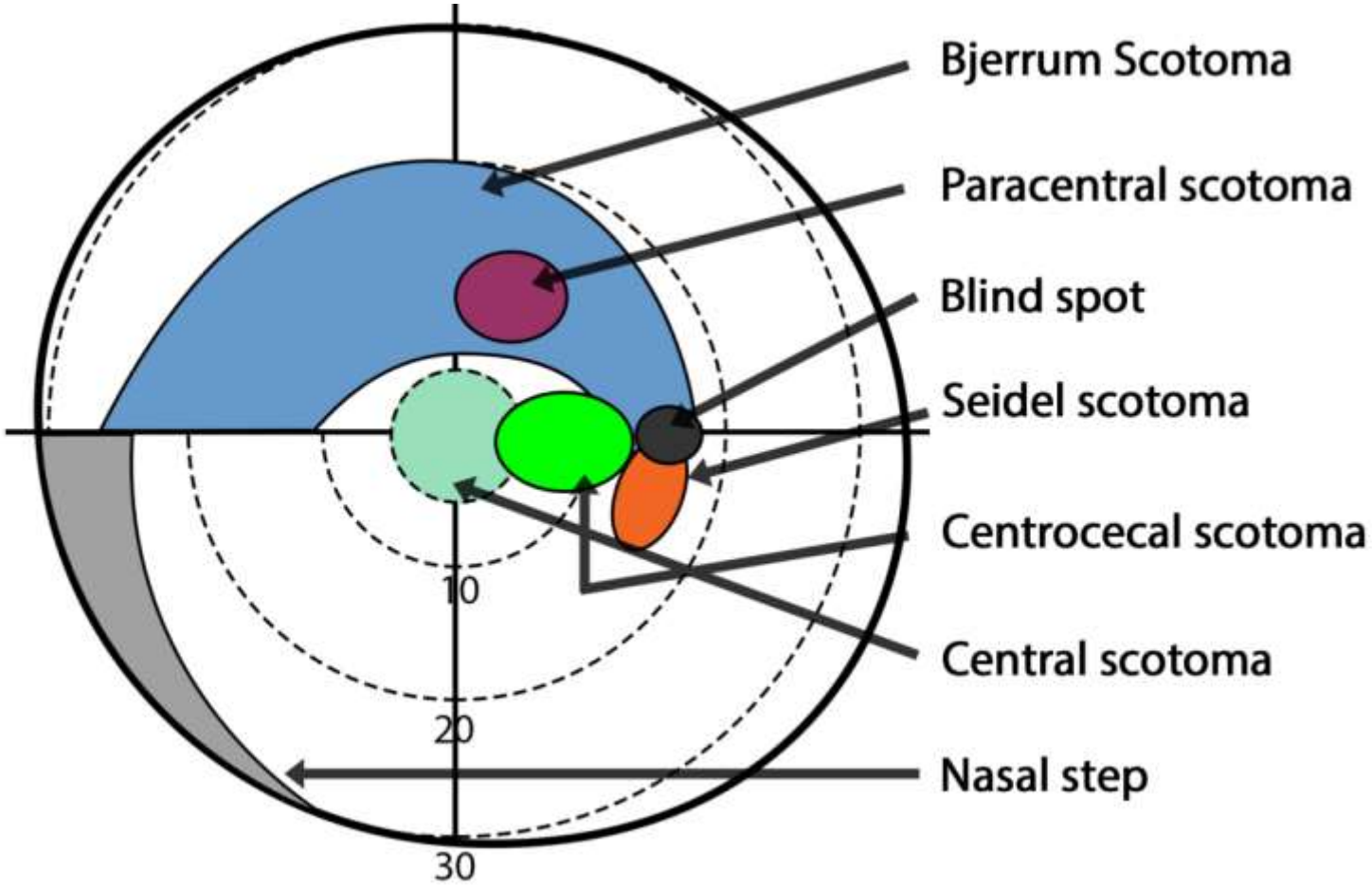
Cupping and pallor correspond

Cupping is greater than pallor

DISC CHANGES

- Cup disc ratio
- Vertical cup
- Bayonetting sig
- Neuroretinal layer thinning, notching
- Pulsation of retinal arterioles
- Lamellar dot sign
- Nerve fibre layer atrophy
- Splinter haemorrhage at or near disc margin
- Parapapillary atrophy
- Nasal shifting of blood vessels

Field changes



Field changes

- Bjerrum's area-10 to 25 degree from fixation.
- Isopter contraction-generalised constriction of central or peripheral field .
- Baring of blind spot-reduced sensitivity around blind spot.(not specific for glaucoma)
- Siedel's scotoma –paracentral scotoma joining blind spot.
- Para central scotoma
- Arcuate or bjerrum's scotoma
- Ring or double arcuate scotoma
- Ronne's nasal step
- Tubular vision

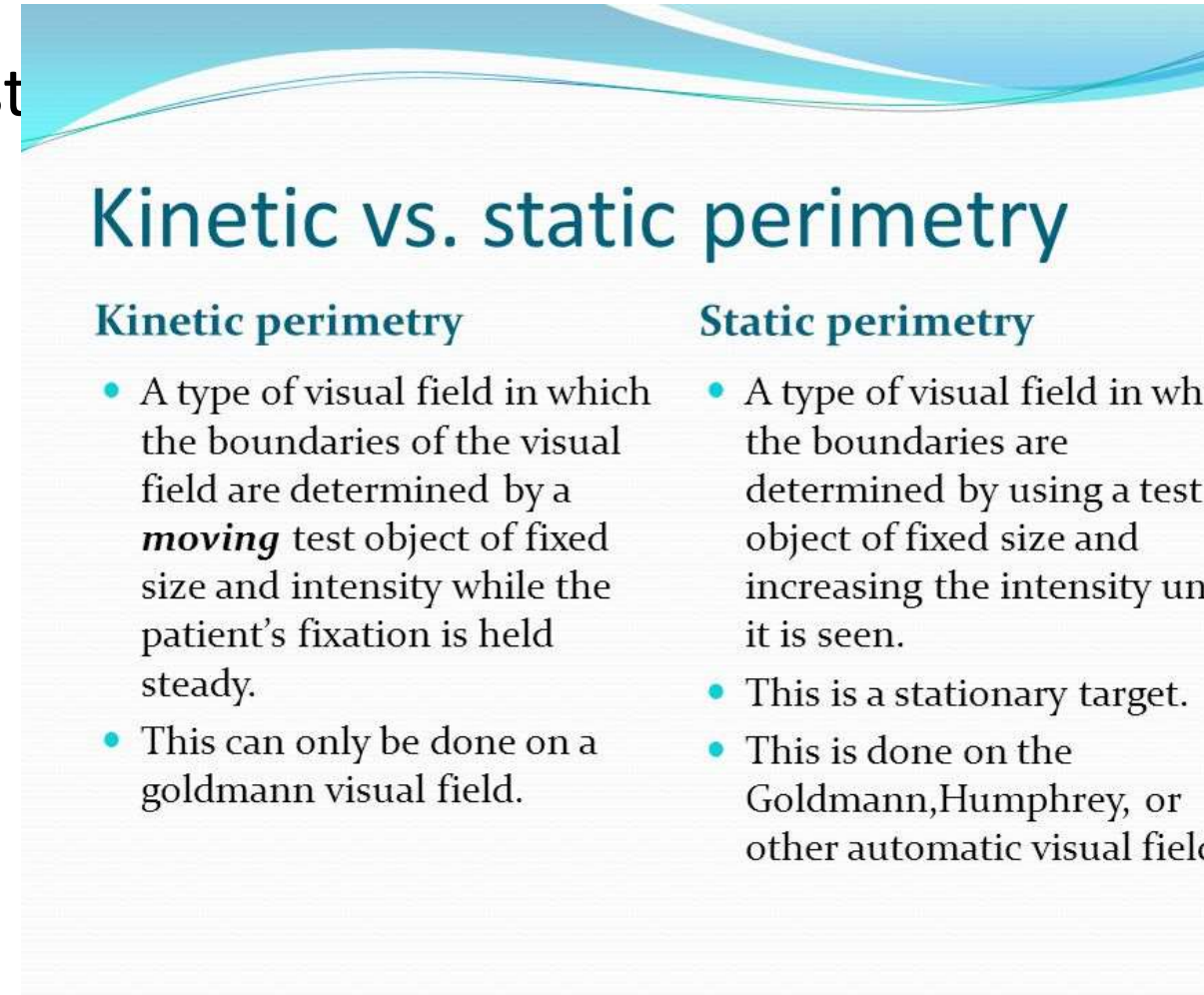
Field defect

Field charting

- Confrontation test
- Perimetry

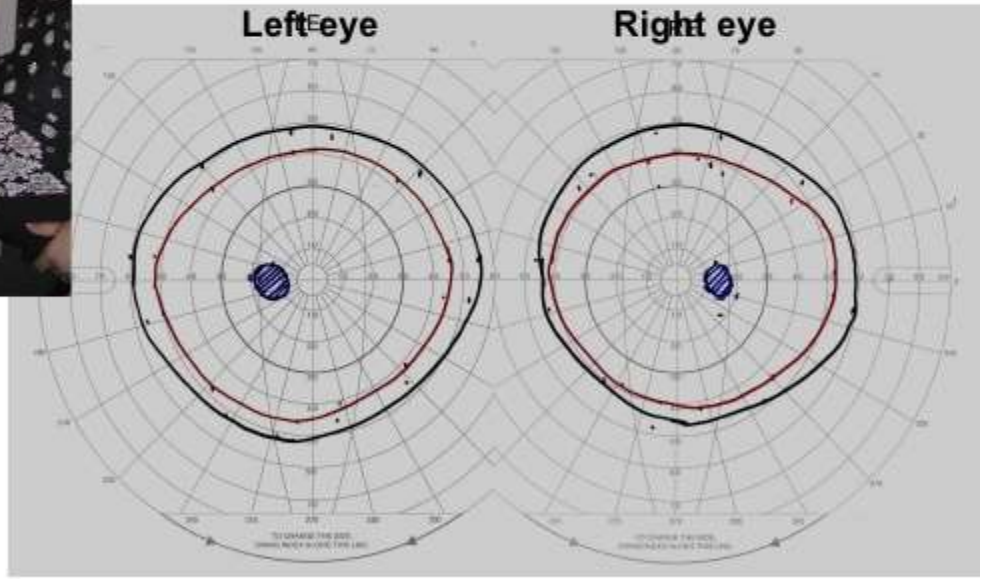
  A) Static

  B) Kinetic



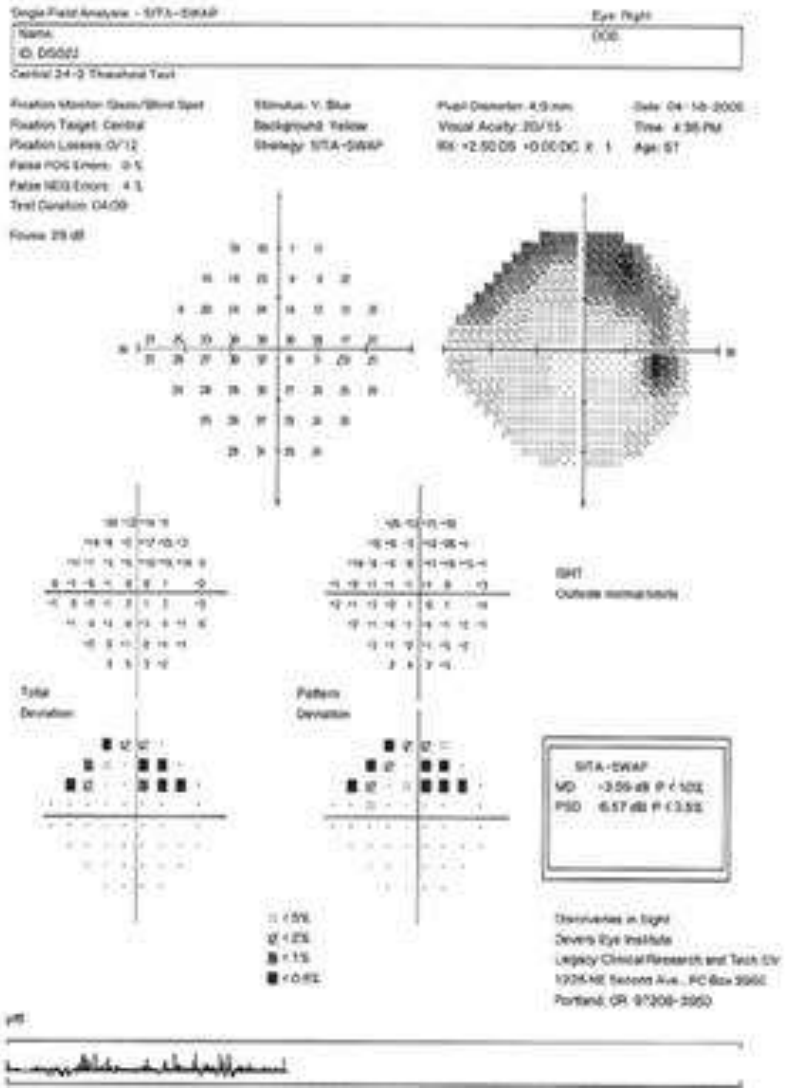
Goldmann perimetry

Light moved from periphery to center of hemisphere
Child signals detection of light with tap on buzzer



Visual fields of a normal 4 year old

Automated perimetry



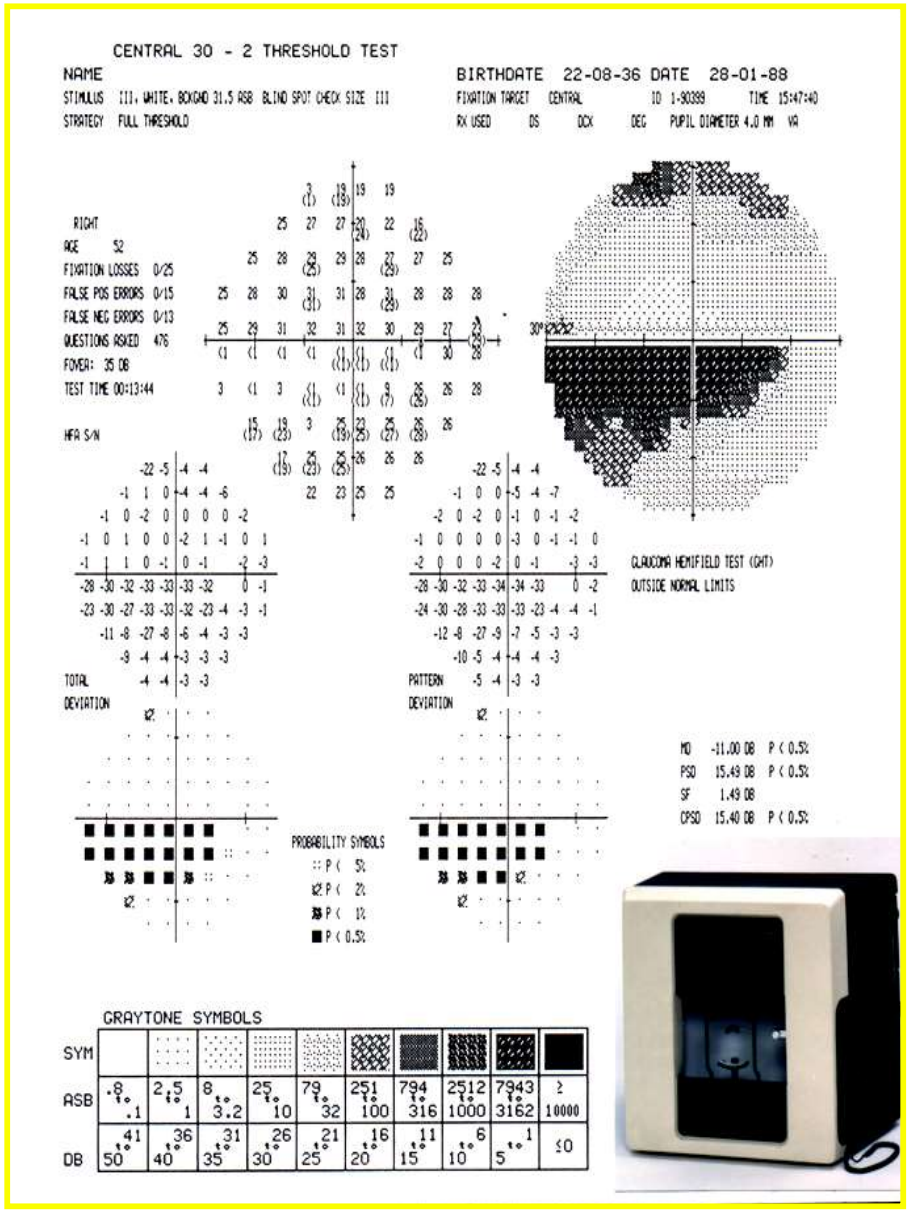
- MD
- CPSD
- GHT
- Single field test not reliable

Anderson criteria

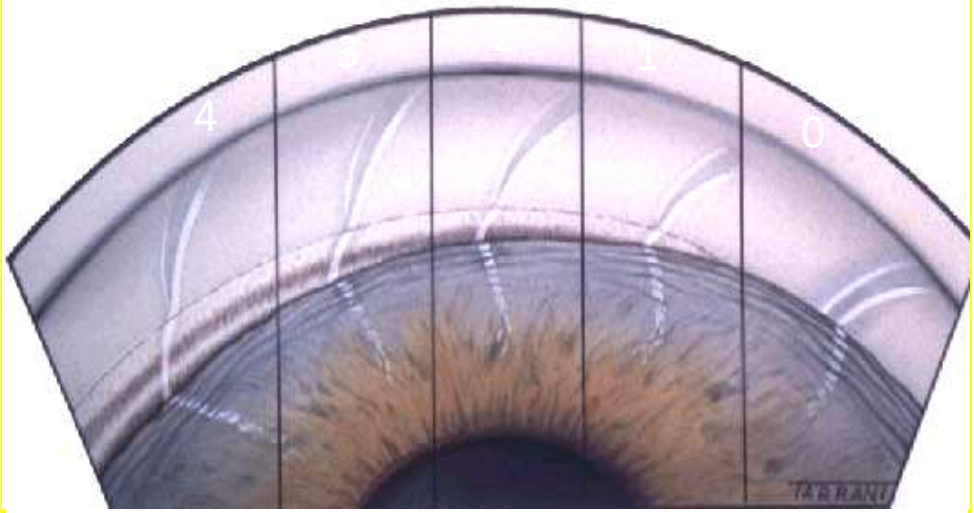
- Glaucomatous field loss is significant in static perimetry if

1. GHT is abnormal on two consecutive occasion.
2. 3 contiguous non edge points on pattern deviation plot within bjerrums area have a probability of <5 % than normal population and out of which one point has probability <1%.
3. PSD have probability <5% on 2 consecutive tests.

Humphrey Perimetry



Shaffer grading of angle width



Grade 4 (35-45 °)

- Ciliary body easily visible

Grade 3 (25-35 °)

- At least scleral spur visible

Grade 2 (20 °)

- Only trabeculum visible
- Angle closure possible but unlikely

Grade 1 (10 °)

- Only Schwalbe line and perhaps top of trabeculum visible
- High risk of angle closure

Grade 0 (0 °)

- Iridocorneal contact present
- Apex of corneal wedge not visible
- Use indentation gonioscopy

Gonioscopy

Direct Gonioscopy- Koppe lens 50 D convex lens

Indirect Gonioscopy -With indentation
Without indentation

Types of glaucoma

- **Congenital and developmental**
- A)with associated anamolies
- B)without associated anamolies
- **Adult glaucoma**
- Primary Open angle glaucoma
- Primary Angle closure glaucoma
- Primary mixed mechanism glaucoma
- Secondary glaucoma

Congenital and developmental glaucoma

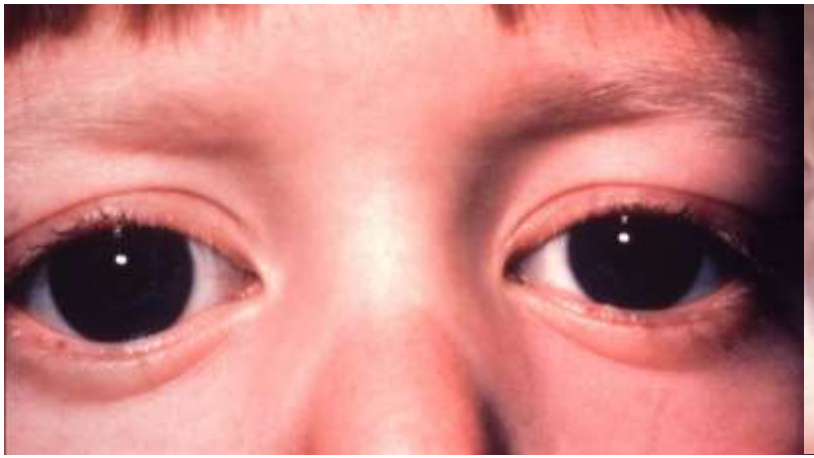
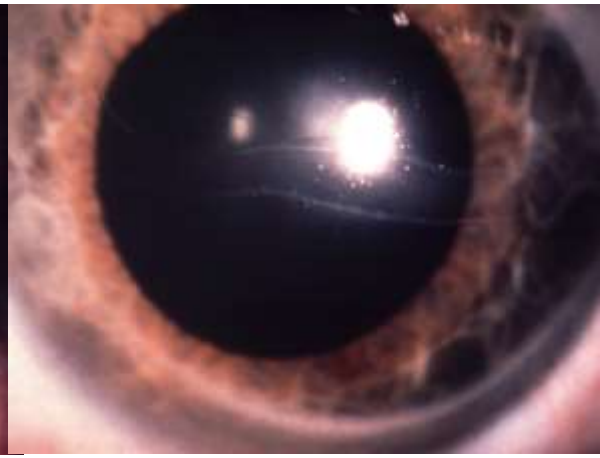
- **True congenital** -IOP raised during intrauterine life so child is born with ocular enlargement.(40%)
- **Infantile**-50%, prior to third b day.
- **Juvenile**-10%. 3 to 16 yrs of life.
- Buphthalmos(bull like eyes)

- M>F, 1 in 10000 births, Sporadic mostly

- **Pathophysiology-** trabecular dysgenesis

Clinical features

- Symptoms-watering, photophobia, eye rubbing, blepharospasm.
- Signs-cornea-enlargement , oedema, descmets breaks(Habbs striae)
- Sclera-thinned appears blue
- Deep A C
- Axial myopia and Anisometropic amblyopia .

Examination under G A

Corneal diameter, gonioscopy, fundus
IOP

Treatment-goniotomy,trabeculotomy
trabeculectomy

POAG

- Chronic simple glaucoma
- Most common,M=F
- IOP>21mm,open angle, deep A C, characterstic field loss, absence of signs of secondary glaucoma
- Risk factors-age, race, family history, IOP,D M, Myopia.

Clinical features

- Asymptomatic till progressed
- Headache, eyeache, coloured haloes
- Delayed dark adaptation
- Frequent change of presbyopic glasses
- Raised IOP and fluctuations in IOP
- Diurnal variation > 8 mm OF Hg
- Asymmetry between 2 eyes >5 mm Hg
- Gonioscopy, water drinking test,SLE,disc changes,perimetry
- Central corneal thickness->550 false high IOP,<540 false low IOP

Primary angle closure glaucoma

- Predisposing Anatomical factors-
- Hypermetropic eyes, small cornea, swollen lens, plateu iris,

PRIMARY ANGLE CLOSURE GLAUCOMA STAGES

- Primary angle closure glaucoma suspect or latent primary angle closure glaucoma
- Sub acute (intermittent) Primary angle closure glaucoma
- Acute primary angle closure glaucoma
- Chronic Primary angle closure glaucoma
- Absolute glaucoma

- **PRIMARY ANGLE CLOSURE GLAUCOMA SUSPECT**
- no signs except schaffers grade 2 or less on gonioscopy
- No PAS OR ITC(Iridotrabecular contact)
- Eyes normal

PRIMARY ANGLE CLOSURE

- ITC in 3 or more quadrant with raised IOP/PAS but without neuropathy
- **PRIMARY ANGLE CLOSURE GLAUCOMA**
- Above conditions and optic neuropathy

Medical treatment

- Mono-therapy, combination of 2 or 3 drugs.
- Set target pressure depending upon age, field changes .for mild to moderate loss aim IOP within 16 to 18 mm of Hg and for severe loss 12 to 14 mm of Hg so that no progressive damage.
- Monitoring with tonometry, perimetry and clinical findings.
- Systemic CIA for short term

Classification of antiglaucoma drugs

Drugs decreasing aqueous production

Beta blockers

Alpha agonists

CAI

Drugs increasing trabecular outflow

Parasympathomimetics

Non selective agonist

Prostamides

Drugs increasing uveoscleral outflow

Alpha 2 agonists

PG AND PM

Medical treatment

P G analogue are the most effective drugs as monotherapy(35-40%)

B blockers (25-30%)

Alpha agonists ,topical CIA,miotics (20 -25%) from baseline

TARGET IOP

- Age
- Vascular perfusion of Optic nerve head, disc changes
- Visual Field changes
- Other predisposing factors like Myopia, Smoking, Central Corneal Thickness etc.
- Progression of Glaucoma

Prostaglandin side affects

- Ocular Side Effects
  1. Conjunctival hyperaemia and foreign body sensation
  2. Eyelash lengthening, thickening, hyperpigmentation, increa in number
  3. Iris hyperpigmentation
  4. Increase in severity and recurrence of herpetic keratitis
  5. Anterior uveitis
  6. Cystoid macular edema

Beta- blocker side effects

- Systemic
  1. Cardiovascular effects – bradycardia, arrhythmia heart failure, syncope
  2. Respiratory reactions – bronchospasm and airwa obstruction, especially in asthmatics
  3. CNS effects – depression, anxiety, confusion, drowsiness, disorientation
  4. Others – nausea, diarrhoea, decreased libido, sk rashes, alopecia

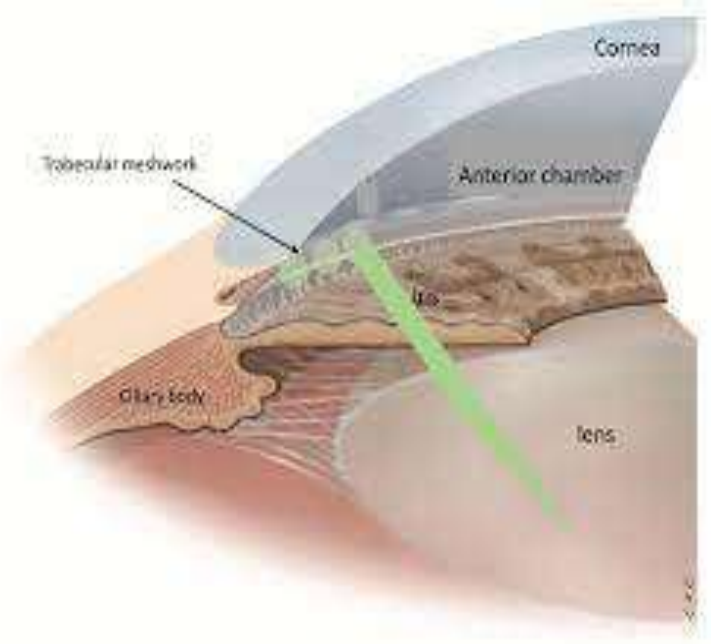
Carbonic anhydrase inhibiters

- Systemic side affects:
  1. Paraesthesias, numbmness, lethargy, depression, malaise
  2. Metabolic acidosis, hypokalemia, increased serum urat level
  3. Urinary frequency
  4. Anorexia, cramps, flatulence, weight loss, diarrhoea
  5. Sulfonamide related – blood dyscrasias, renal calculi, steven-Johnson syndrome

Topical agents are less likely to induce systemic side effects

Lasers In Glaucoma

- Uncontrolled glaucoma despite maximal tolerated medical therapy especially in elderly
- Poor compliance with drugs
- Avoidance of polypharmacy
- Unfit for surgical treatment



- **Outflow enhancement**-

  For POAG-(ALT /DLT/ SLT)

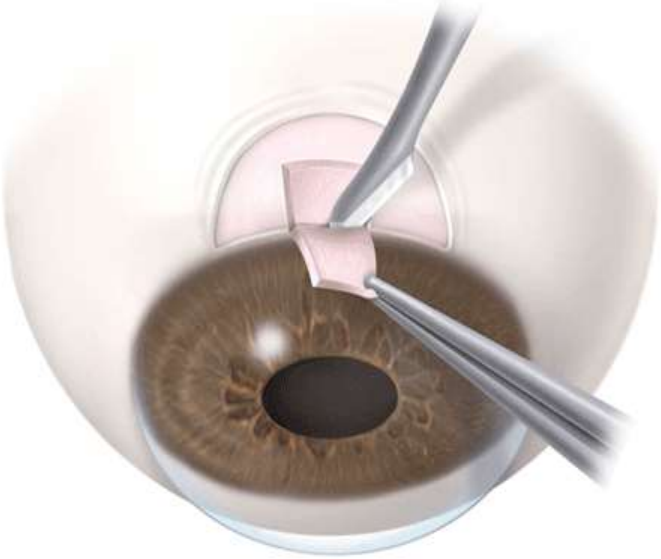
  For ACG- Nd YAG Laser iridotomy

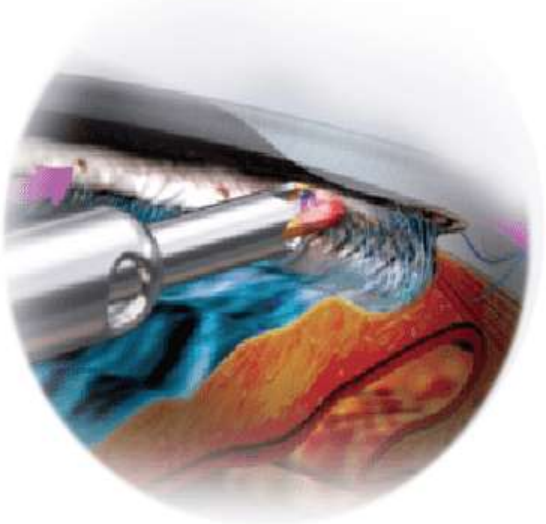
- **Inflow reduction**-cyclophotocoagulation in end stage disease.

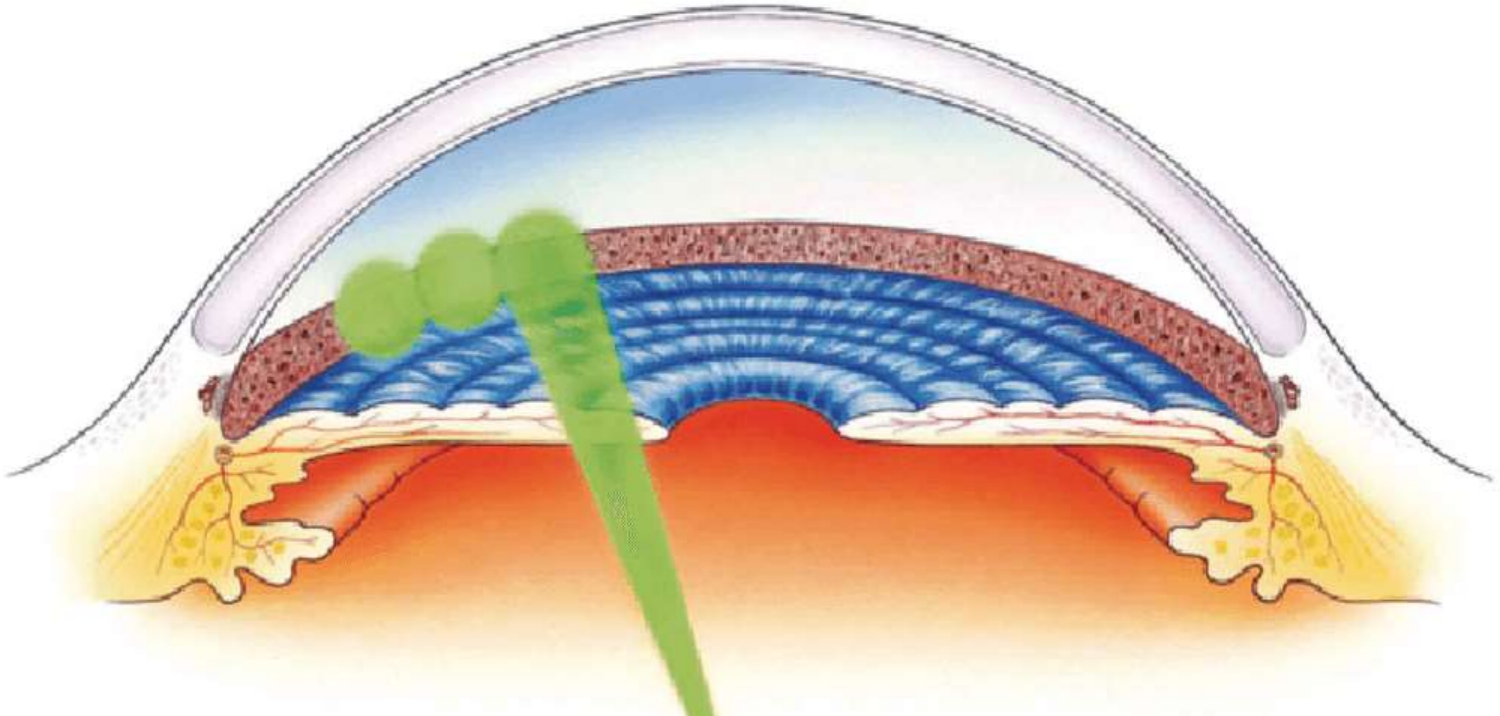
- **Argon or diode laser trabeculoplasty (ALT OR DLT)**.for POAG reduction of IOP by 8 to 10 mm of Hg.50 spots for 180 * ant to trabecular meshwork.
- **Selective laser trabeculoplasty (SLT)-**NdYAG double frequency -532 nm , 400 Micron spot size (fixed), 3 nanosec (fixed), upto 360*, 04 to 1.0 mJ energy for bubble formation.

- ***MLT-MicroPulse laser trabeculoplasty** (MLT) uses 532/577/810nm repetitive, low-energy laser pulses that are separated by brief rest periods. This “micropulsing” allows the trabecular meshwork to cool between laser pulses to prevent tissue damage.

- *SLT absorbed by melanin pigment only so need not to be precise location , minimal thermal damage , activates macrophages







SURGERY

AB EXTERNO –

A)Penetrating filteration surgery - Trabeculectomy

B)Non penetrating filteration surgery

Deep sclerectomy

Viscocanalostomy

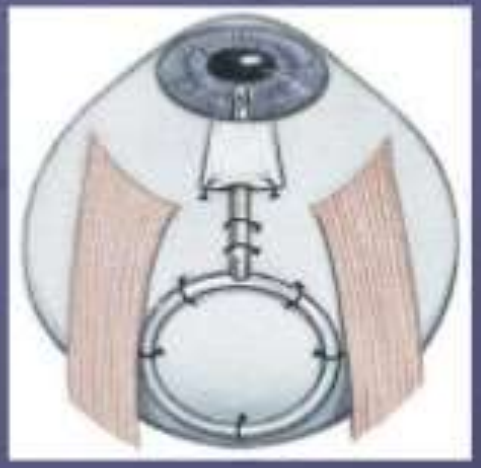
Canaloplasty

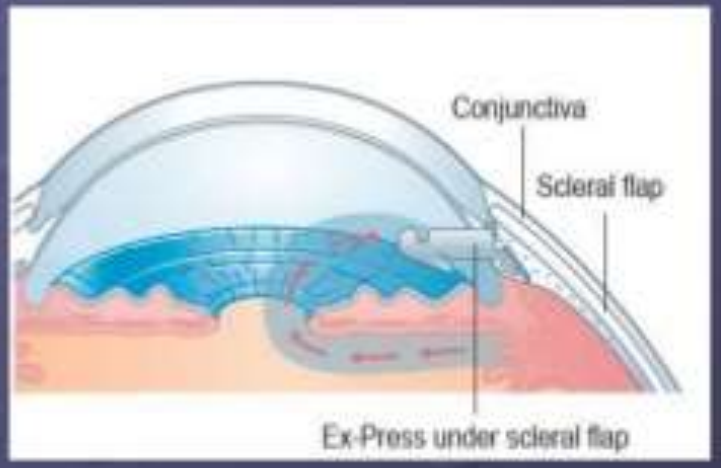
Trabeculectomy

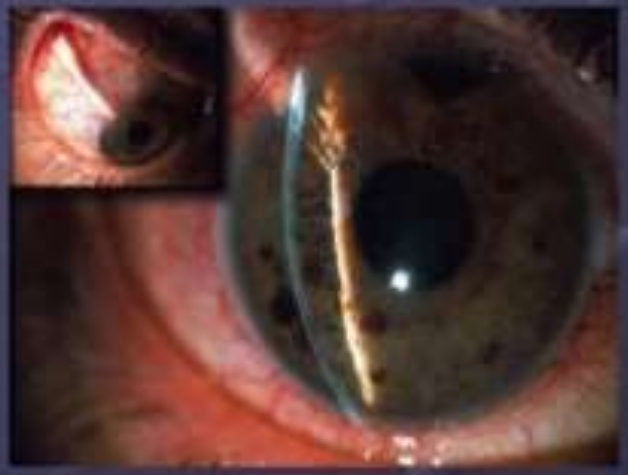
- Indications:
  - **Failure of conservative therapy** to achieve adequate IOP control.
  - **Avoidance of excessive polypharmacy**
  - **Progressive deterioration despite seemingly adequate IOP control** (including poor compliance with medical treatment).
  - **Patient preference**

DRAINAGE SHUNTS

- Shunts using episcleral explants
- Glaucoma Drainage Devices(GDD)= creates communication between AC and sub tenon space

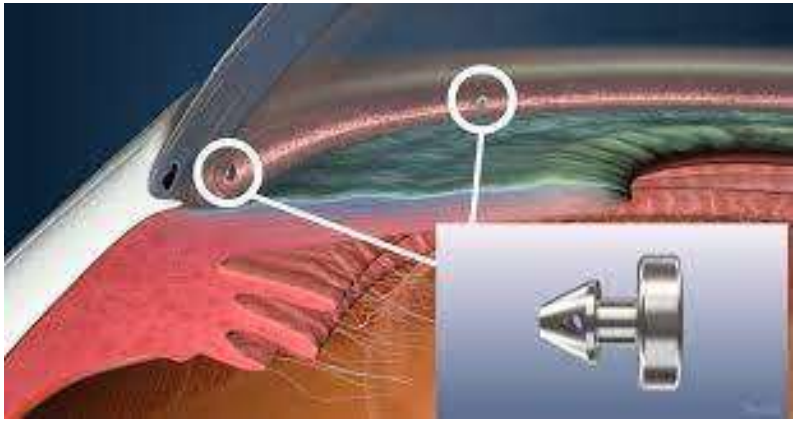






AB INTERNO

I-stent supra- in Suprachoroidal space



I-stent inject- in Canal of Schlemn