الزَّرَق

Introduction

- Glaucoma is an optic neuropathy associated with characteristic damage to the optic nerve head (cupping) and the visual field (nerve fibre bundle defects).

nerve imaging shows structural damage

shows functional damage

- It is a blinding disease where first the peripheral visual field becomes constricted, followed by loss of central visual acuity

- Glaucoma, if defined with either field or nerve criteria, has a prevalence of 5.6%

- If defined with both field and nerve criteria, it has a prevalence of 2.4% → still common → screening is needed

- The appearance of the optic nerve head and visual fields are the major factors for a diagnosis of glaucoma

Risk Factors

- Age ↑
- Ethnicity
- Family History
- ☆☆ Intraocular pressure (IOP) is the most important risk factor
- Trauma
- Eye surgery
- Drugs
- Refractive errors

Non-IOP dependent risk factors

- Systemic Vascular Dysregulation
  - Raynaud's, Prinzmetal Angina, Migraine
- Nocturnal Hypotension → related to perfusion of the nerve
- Sleep Apnea

Intraocular Pressure IOP

- An elevated eye pressure is neither necessary nor sufficient to make the diagnosis:
  - in "normal tension glaucoma", the patient is never found to have a pressure over the normal limits
  - in "ocular hypertension" the patient has high eye pressures but no signs of optic nerve or visual field damage

→ Ironically !

⇒ treatment still will be to lower the tension even if it's in the normal limits

→ some will eventually will develop glaucoma

+ ↑ IOP is the most important risk factor because it's the only modifiable one

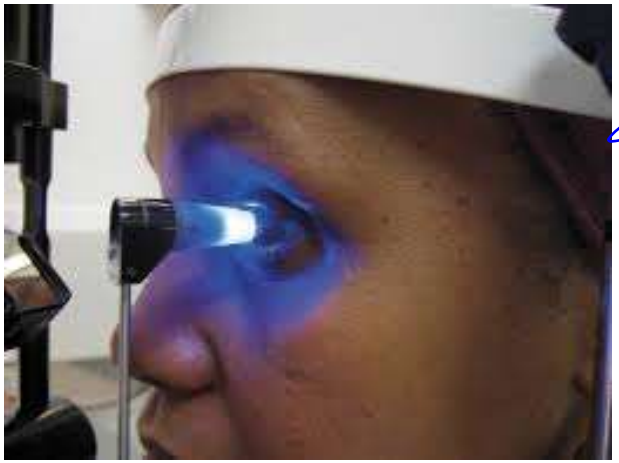
- The level of the intraocular pressure is the main risk factor, and is important in the monitoring of treatment even if it's not necessary to make the diagnosis
- Gonioscopy is of major importance in the classification of the glaucoma type
- The extent of damage to the optic nerve and visual field determines the stage of the glaucoma

Basic Principles-IOP

- Intraocular pressure (IOP) represents the equilibrium between the rigidity of the cornea and sclera, and the outward pressure of the ocular contents
- As the vitreous is of fixed volume, the most important variable is the amount of aqueous humour, which varies with respect to production and drainage.
- The pathology of elevated intraocular pressure is due to inadequacies of aqueous outflow rather than production

Mostly the problem is here

- The normal mean IOP is 15.5 mm Hg
- Range is 10-21 mm Hg
- Diurnal variations exist ↑ in morning, ↓ in night (perfusion is also lower so the eye is more vulnerable to risks during night more than day)
- Many ways to measure it but standard is Goldmann Applanation Tonometry -GAT



Basic Principles- Aqueous Humor

- The volume of the aqueous humour in the anterior segment is 0.25 cc or 250 µL
- One quarter of this is in the posterior chamber and three quarters in the anterior chamber
- The ciliary body produces 2.5 µL per minute, with complete turnover of the aqueous in about 100 minutes

Aqueous Humor

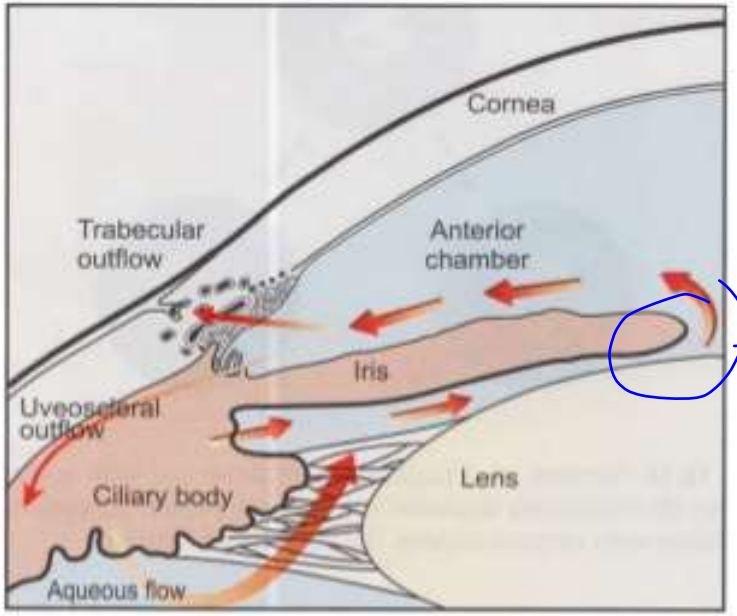
- It is produced by the non pigmented epithelium of the ciliary processes

- Produced by :
  - Ultrafiltration
  - Active secretion

Aqueous Humor Pathway

- Aqueous humor passes from the posterior chamber between the iris and the lens through the pupil into the anterior chamber

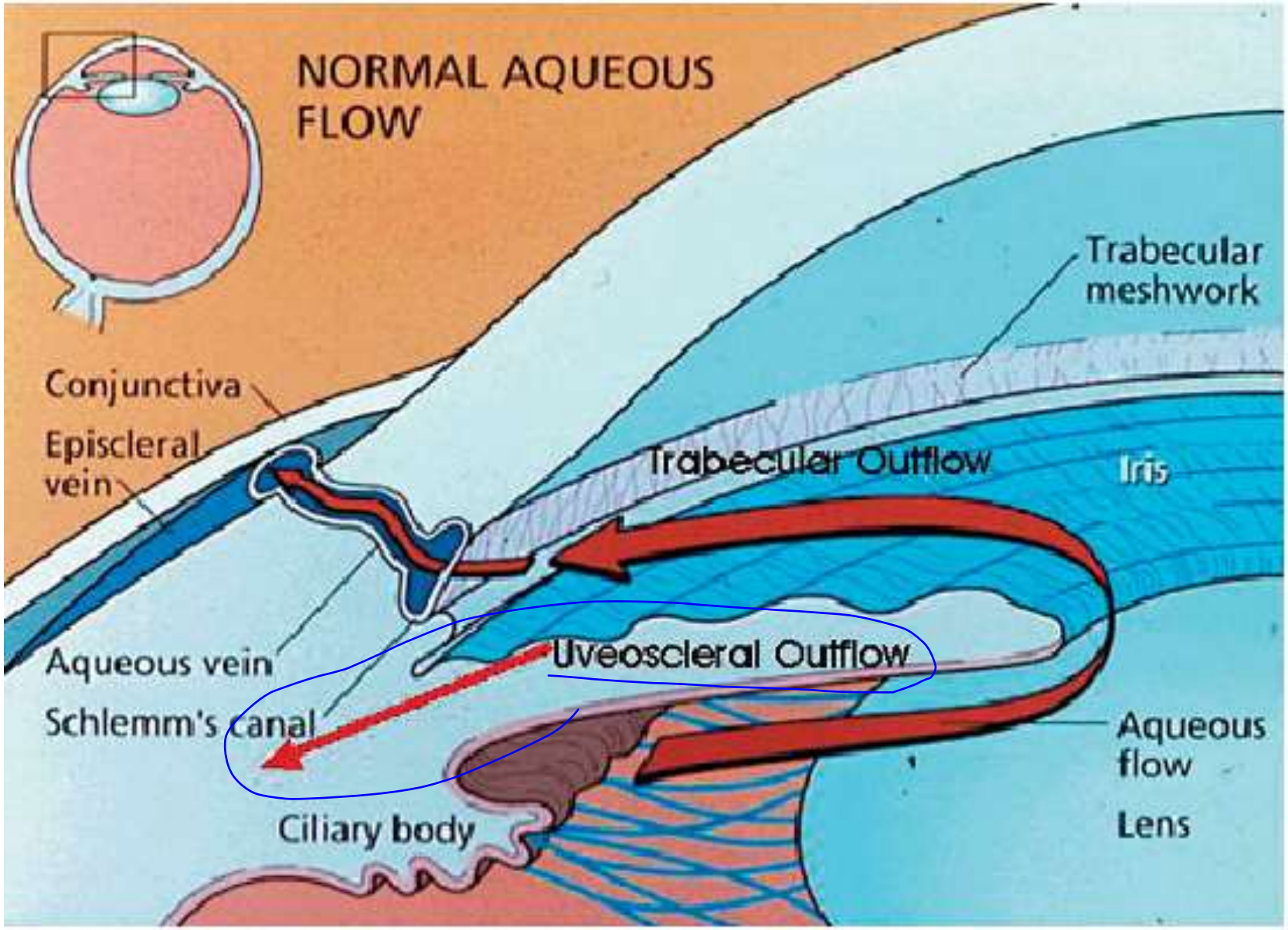
AQUEOUS HUMOUR DYNAMICS

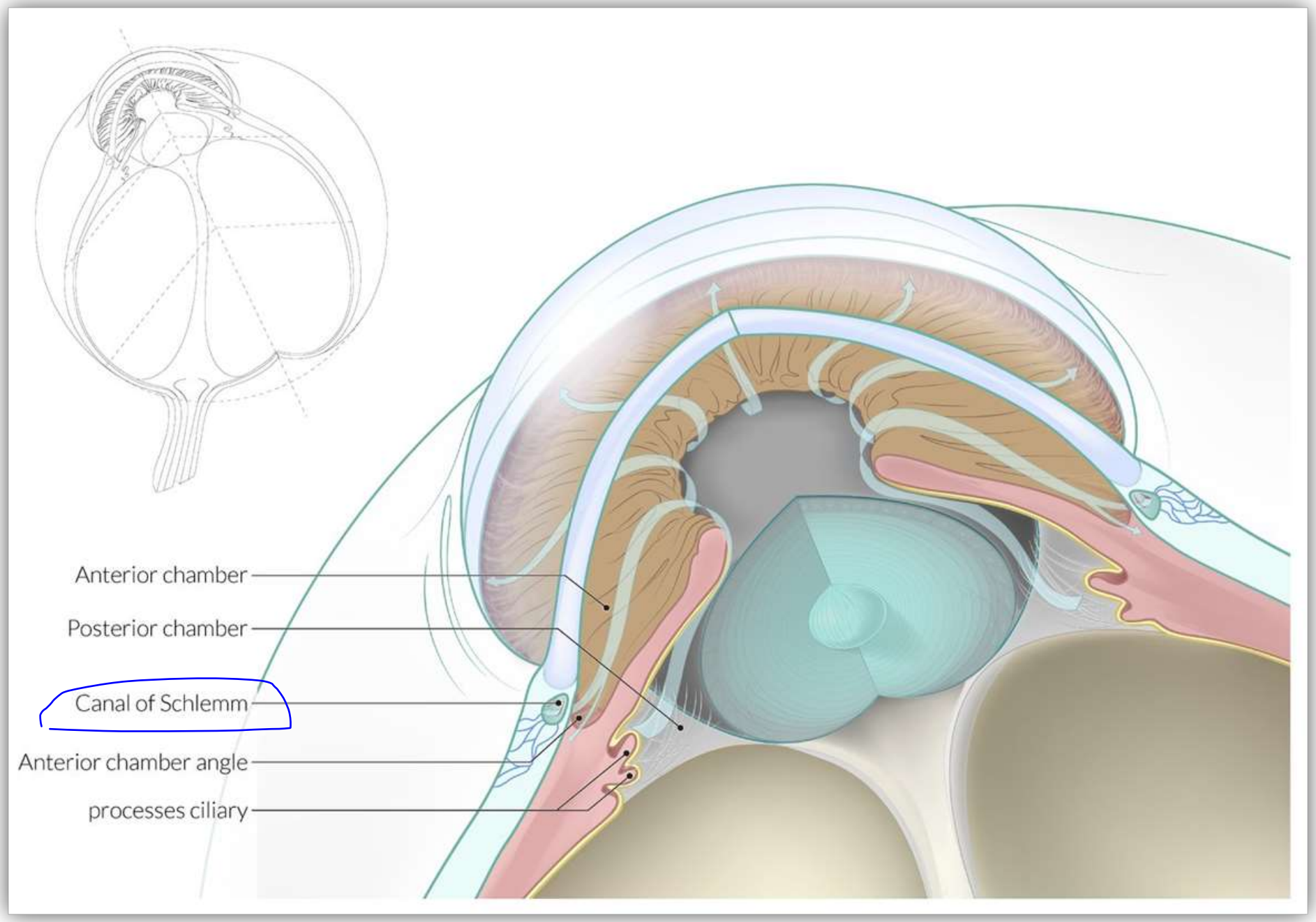


If this angle was closed → pupillary block glaucoma

Aqueous Humor Pathway

- It drains through 2 pathways:
  - Conventional (80-90%): trabecular meshwork, Canal of Schlemm, aqueous veins and episcleral veins
  - Uveoscleral (10-20%): Face of the ciliary body and iris to the supraciliary/suprachoroidal space.


NORMAL AQUEOUS FLOW
Trabecular meshwork
Conjunctiva
Episcleral vein
Trabecular Outflow
Iris
Aqueous vein
Uveoscleral Outflow
Schlemm's canal
Aqueous flow
Ciliary body
Lens

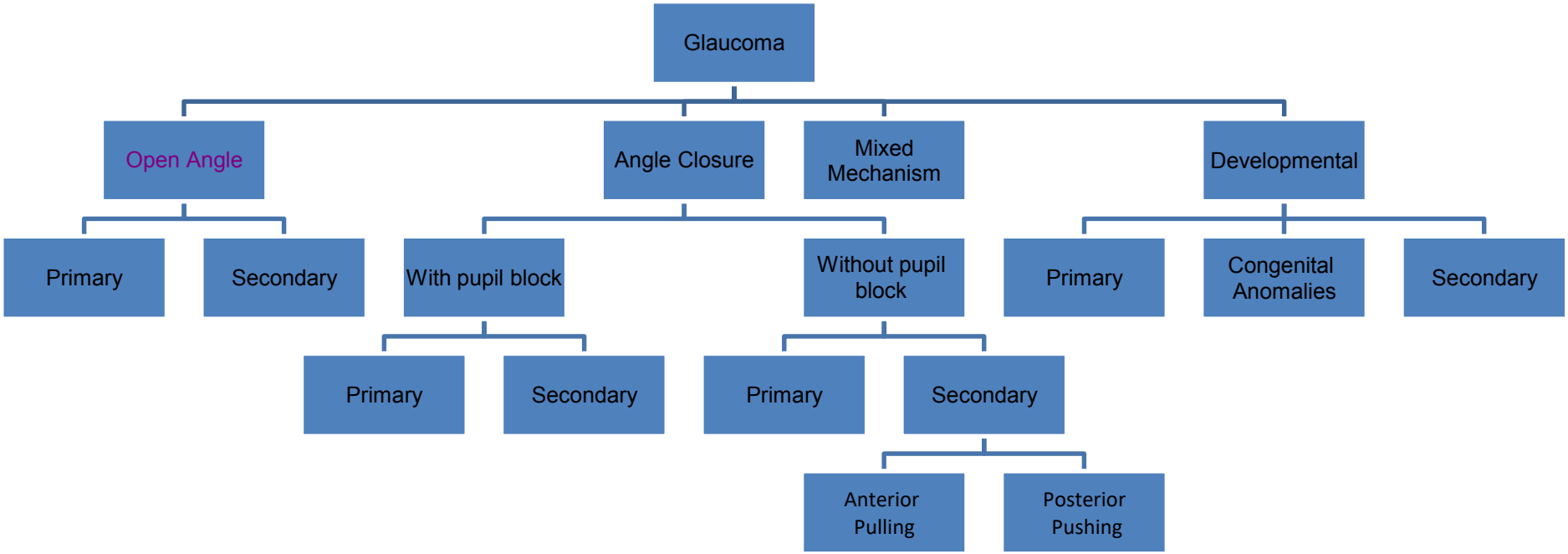

Anterior chamber
Posterior chamber
Canal of Schlemm
Anterior chamber angle
processes ciliary

Classification

- Glaucoma is not a single disease, but a large number of similar conditions with factors in common.
- It is usually classified on the basis of the anatomy of the anterior chamber angle as open or closed, and each type has primary and secondary sub-categories.

angle between Iridocorneal angle ←

Glaucoma Classification



Open Angle Glaucoma

- Primary Open Angle Glaucoma POAG:
  - Idiopathic increase in outflow resistance → most common type
- Secondary Open Angle Glaucoma SOAG:
  - Clogging of trabecular meshwork(TM) → as if after trauma and TM is filled with blood
  - Increased episcleral venous pressure EVP
  - Scarring of TM
  - Increased TM resistance due to medications (steroids)
    ↳ most common secondary type

POAG

- Most prevalent type
- Female = male
- More common in myopes
- Asymptomatic till late in the disease
- IOP 20-40 mmHg

TM resistance is higher in myopes (inherint structur changes)

⇒ even after lasik surgeries the risk is still there (the surgery only corrects the refractive error)

→ and still asymptomatic → the optic nerve is deteriorating

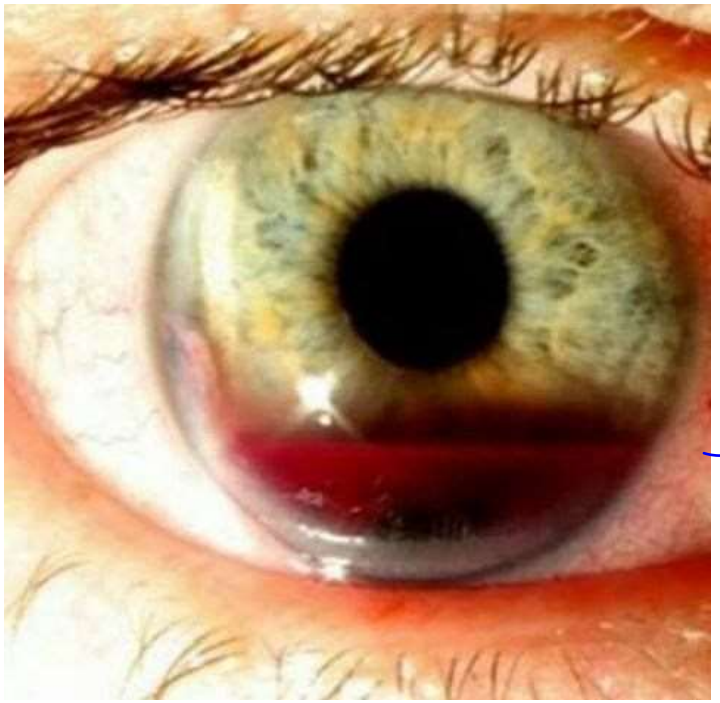
only becomes symptomatic at late stages (when it's too late)

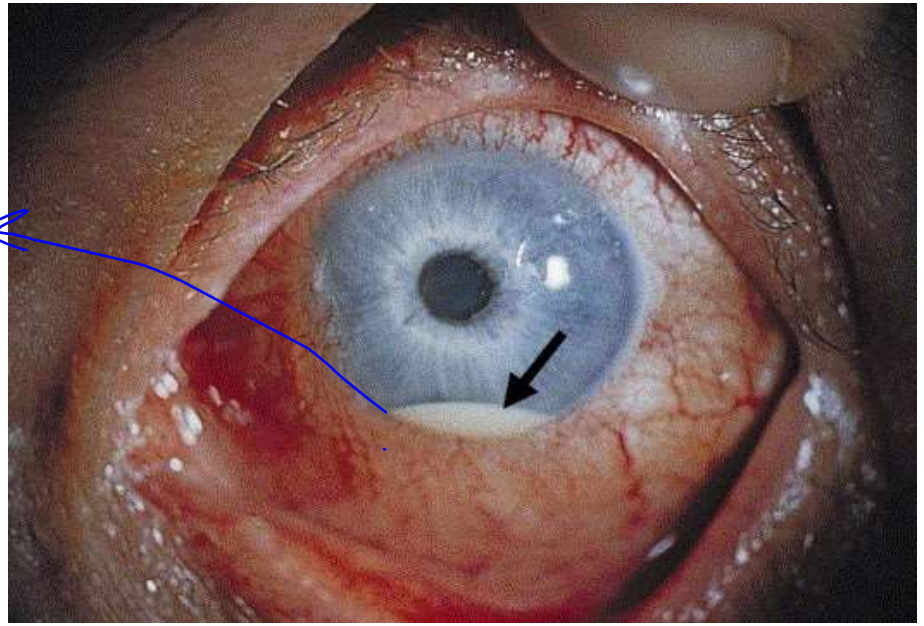
⇒ thus this disease is screenable disease

SOAG

- Clogging:
  - RBCs: Hyphema
  - WBCs: Uveitis
  - Pigment: pigment dispersion syndrome, melanoma → shedding of Melanocytes may close the angle
  - Proteins: Pseudoexfoliation syndrome
    lens proteins

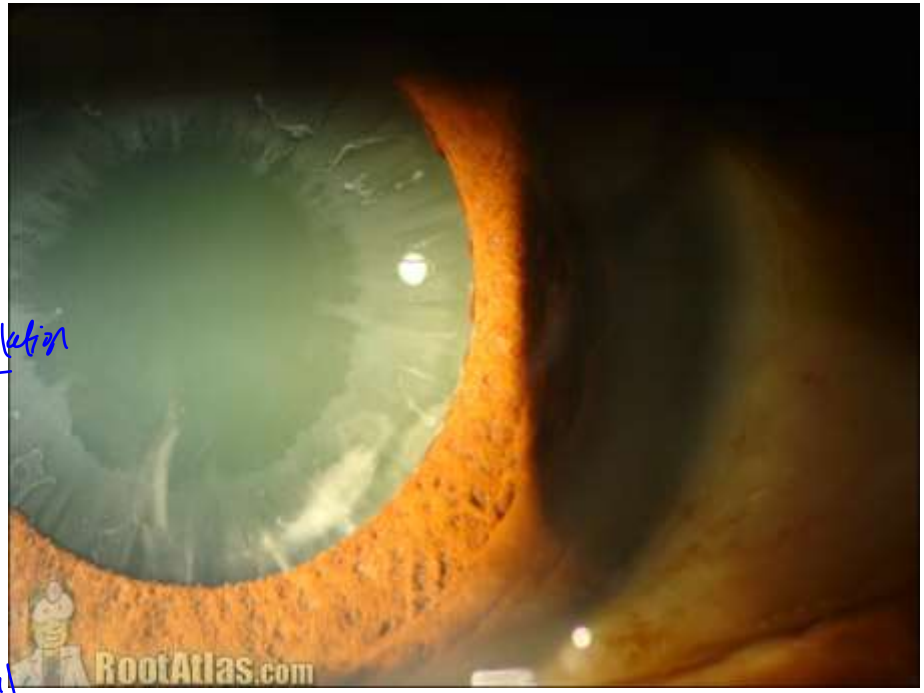
☆ The angle itself is open but there's something covering it (think of it as pseudoclosing)





pus covering

blood covering angle



pseudoexfoliation syndrome

↳ pigment dispersion syndrome (melanoma) due to

SOAG

- Increased EVP:
  - Carotid cavernous fistula
  - Sturg Weber Syndrome
  - SVC obstruction
- Scarring:
  - Angle recession (trauma)

Closed Angle Glaucoma

Most common type is acute closed angle glaucoma

- Anatomic features predisposing to angle closure: shallow anterior chamber (e.g., hyperopia, short eye)
- Advanced age (>60 years).
- Female gender
- Inuit and Asian ethnicity
- Eye injury with scarring and adhesions
- Rubeosis iridis
- Drugs: Sulfonamides, TCA, MAOi, antihistamines
- Mydriasis
  I. Drug-induced: mydriatics
  II. Darkness
  III. Stress/fear response

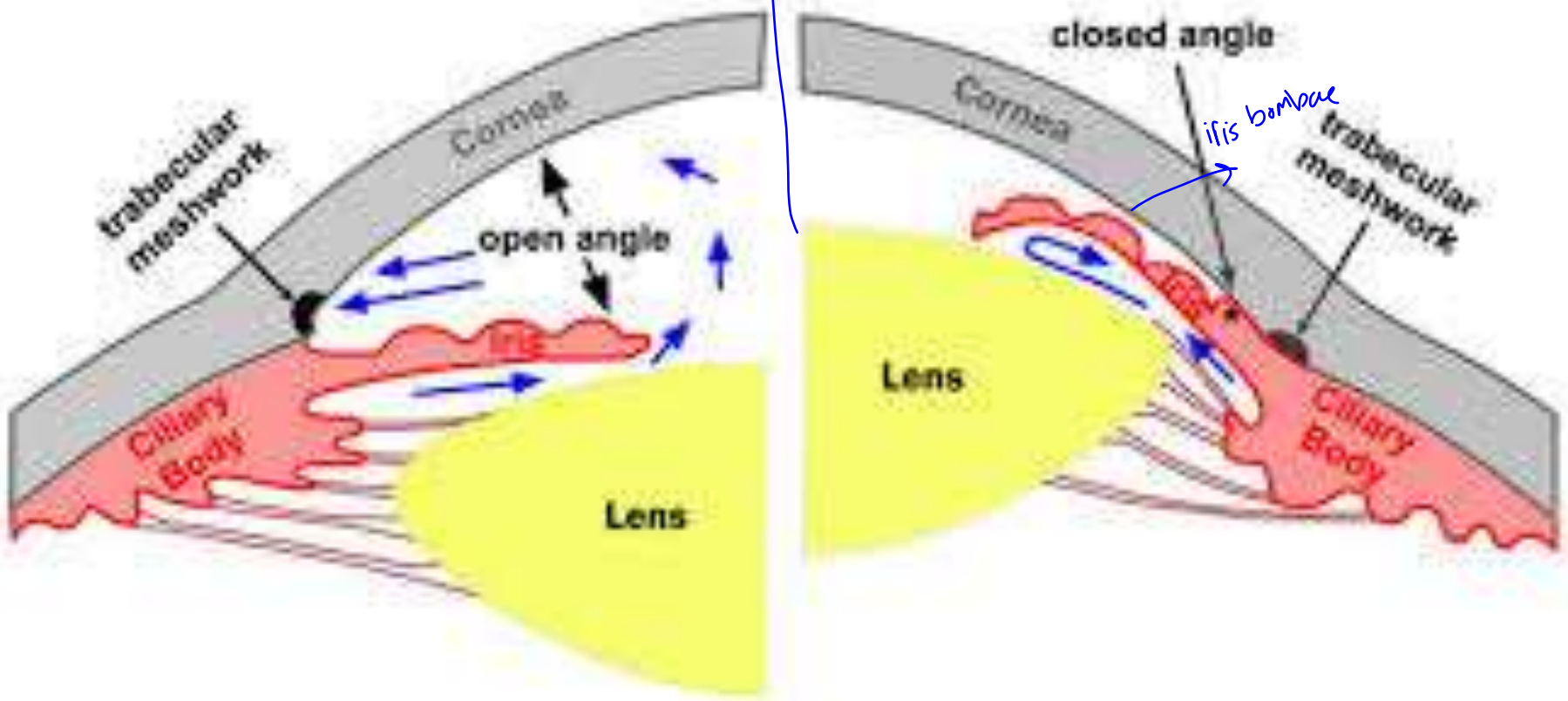
Pathophysiology

- Blocked trabecular meshwork → decreased drainage of aqueous humor from the eye → sudden ↑ in IOP

A. Primary : the chamber angle is narrowed due to the peripheral iris obstructing the TM.

B. Secondary :
   A. Scarring: PAS or PS
   B. Lens luxation/ large cataracts
   C. Rubeosis iridis (neovascular glaucoma)

Open vs Closed Angle

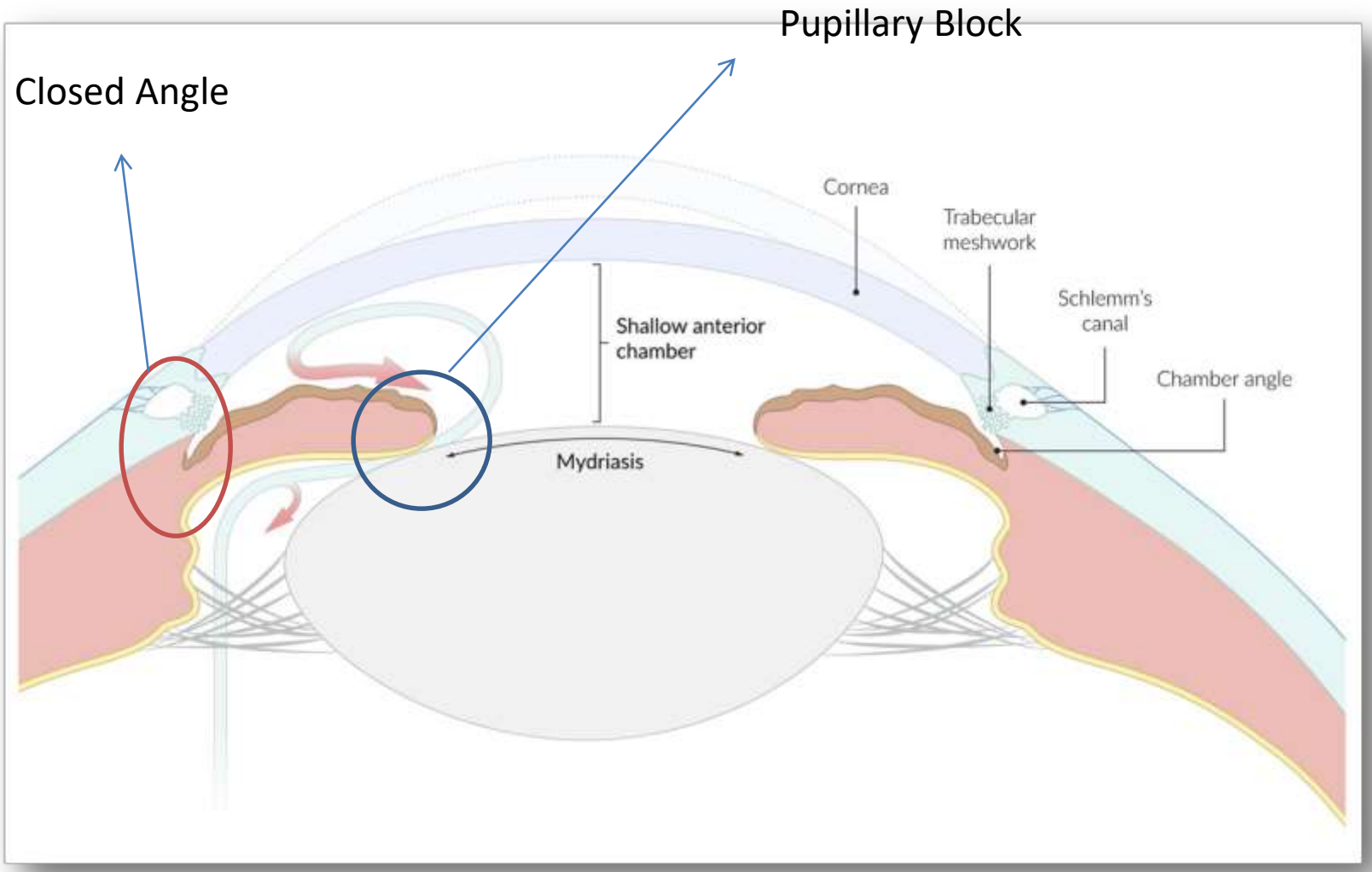


↓ management of acute closed angle glaucoma is never surgical

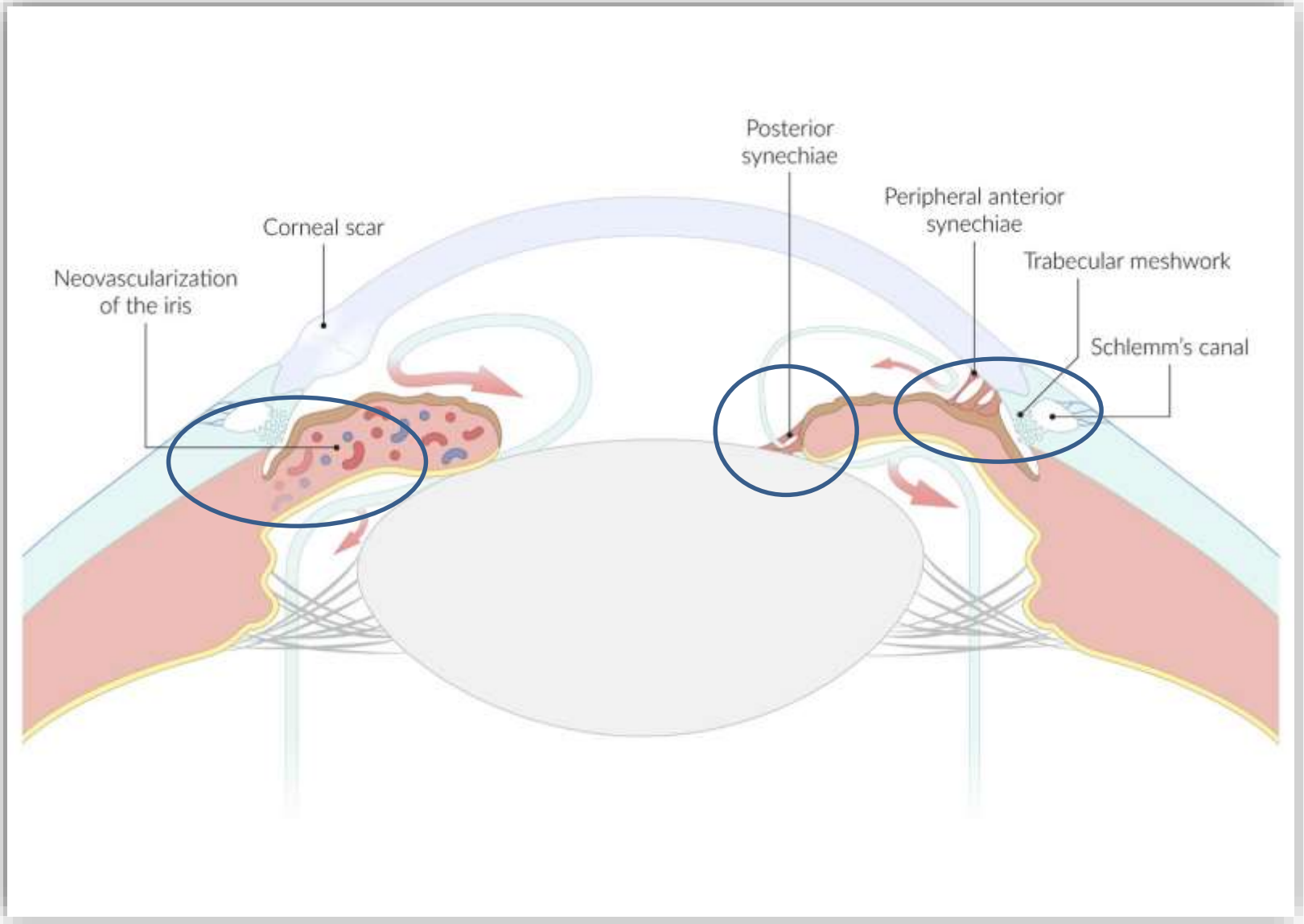
it is medical ⇒ mannitol (takes water from vitreous humor) ⇒ then laser I/I. (do it for both eyes)

contraindication; anuric patients + uncontrolled DM

Acute Angle Closure with pupillary Block



Chronic Angle Closure-NVG/ Uveitis



Clinical Presentation of Acute Angle Closure Attack

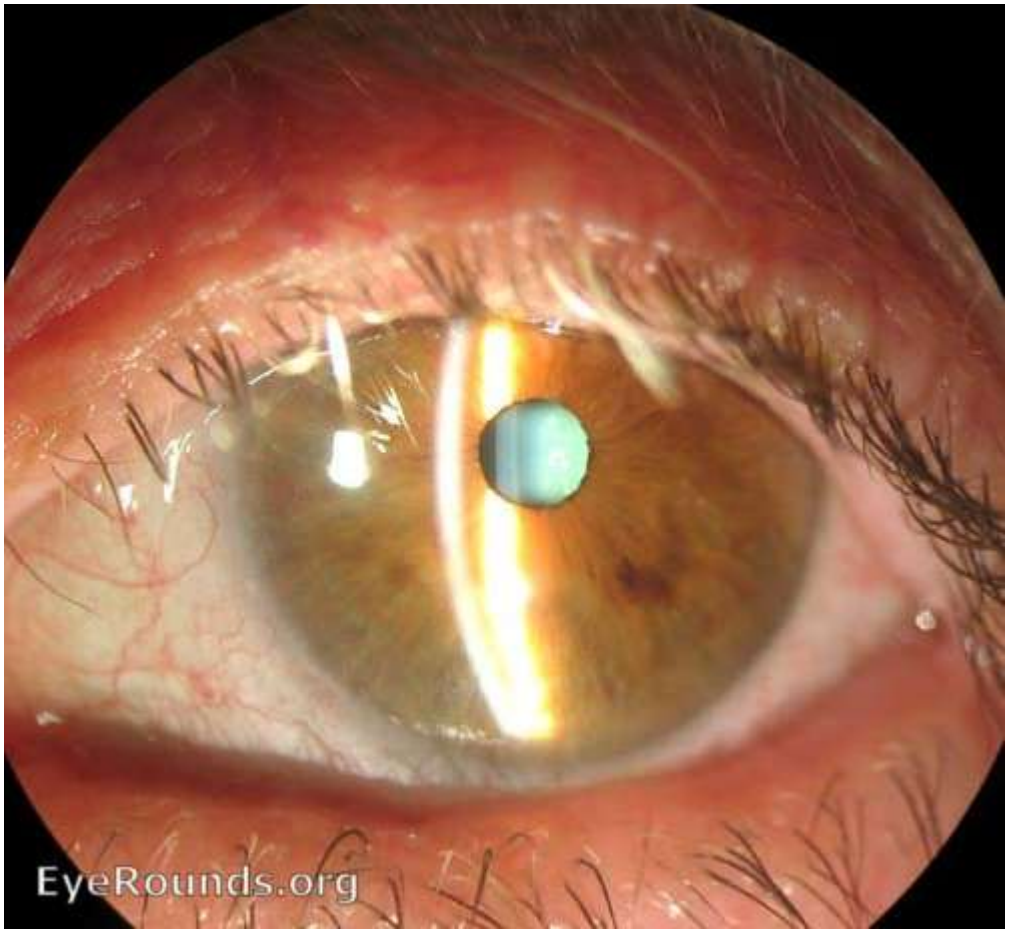
- Sudden onset of symptoms
- Severely painful eye (hard on palpation), redness
- Photophobia and excessive tearing
- Headache, nausea and vomiting
- Blurred vision and halos seen around lights
- Complications: irreversible damage of the optic nerve

Diagnosis- Physical Examination

- Decreased visual acuity
- Non reactive, fixed oval pupil
- Cloudy edematous cornea
- Shallow anterior chamber
- Closed angle on gonioscopy
- IOP > 40 mm Hg

Injected, Cloudy cornea, oval pupil

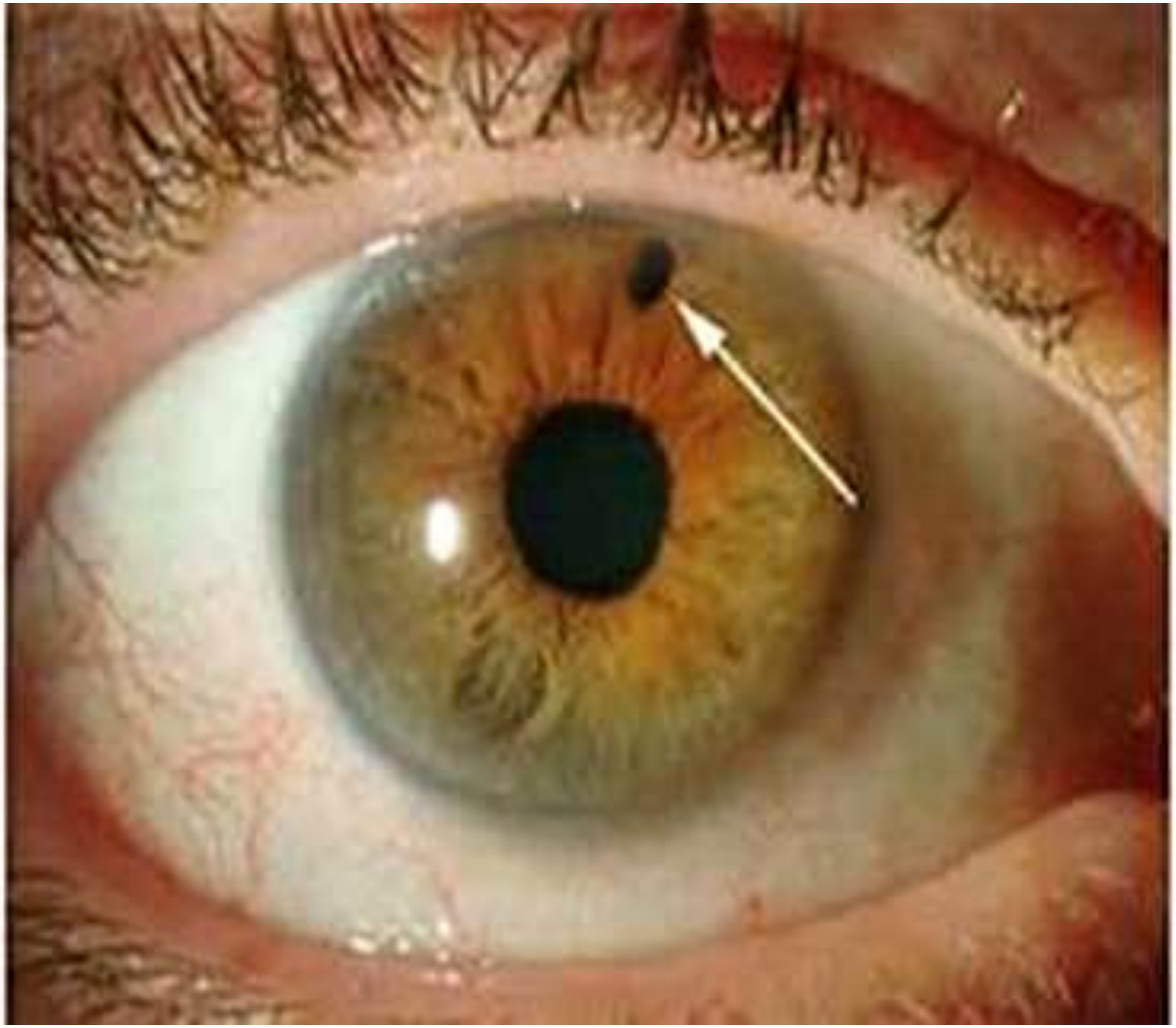
Shallow A/C

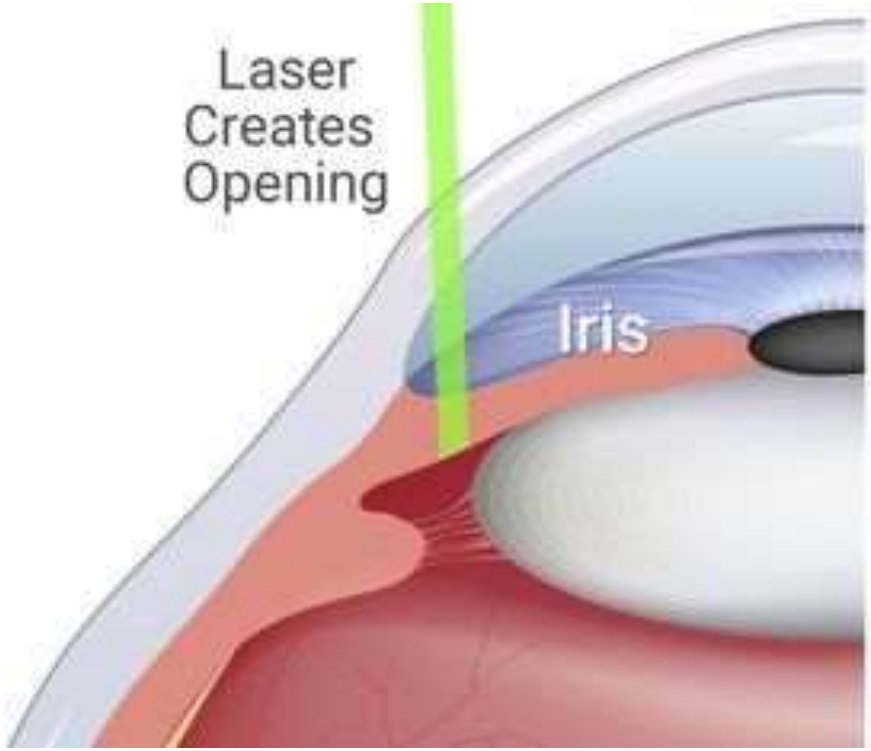


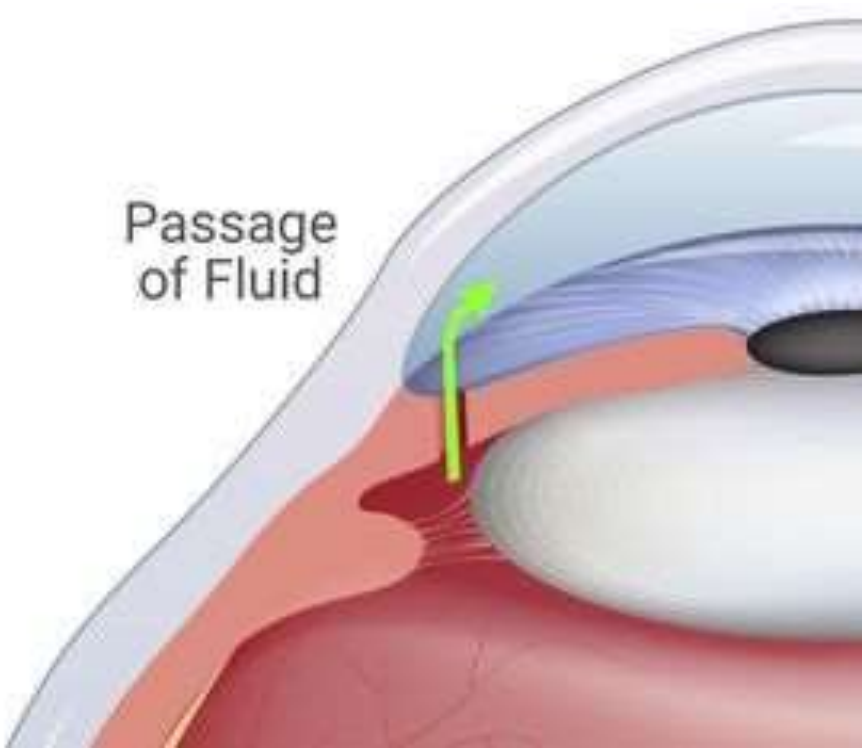
Management outline

- Lower IOP:
  - Systemically : IV Acetazolamide/ Mannitol
    oral Acetazolamide
  - Topical Eye drops: B blockers, α agonists, Carbonic anhydrase inhibitors, pilocarpine
- Break the angle closure cycle:
  - YAG laser Iridotomy/ Surgical iridectomy
- Examine second eye and treat prophylactically

YAG Laser Iridotomy




Laser
Creates
Opening
Iris

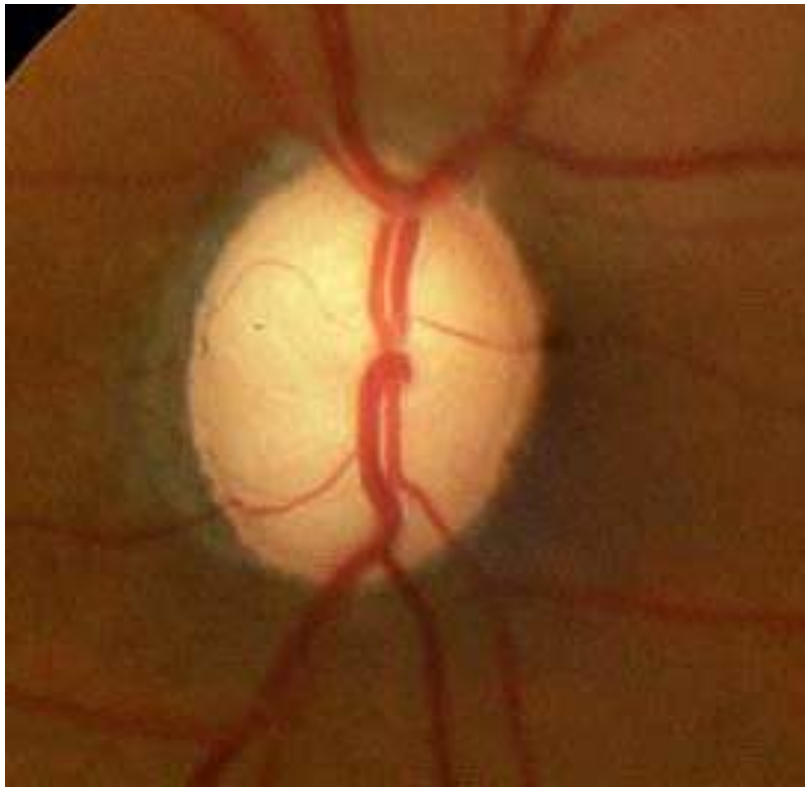

Passage
of Fluid

Glaucoma Diagnosis: Tips and Tools

- History
- Physical Examination
- Special Tests

Tip One

- Every patient has glaucoma until proven otherwise

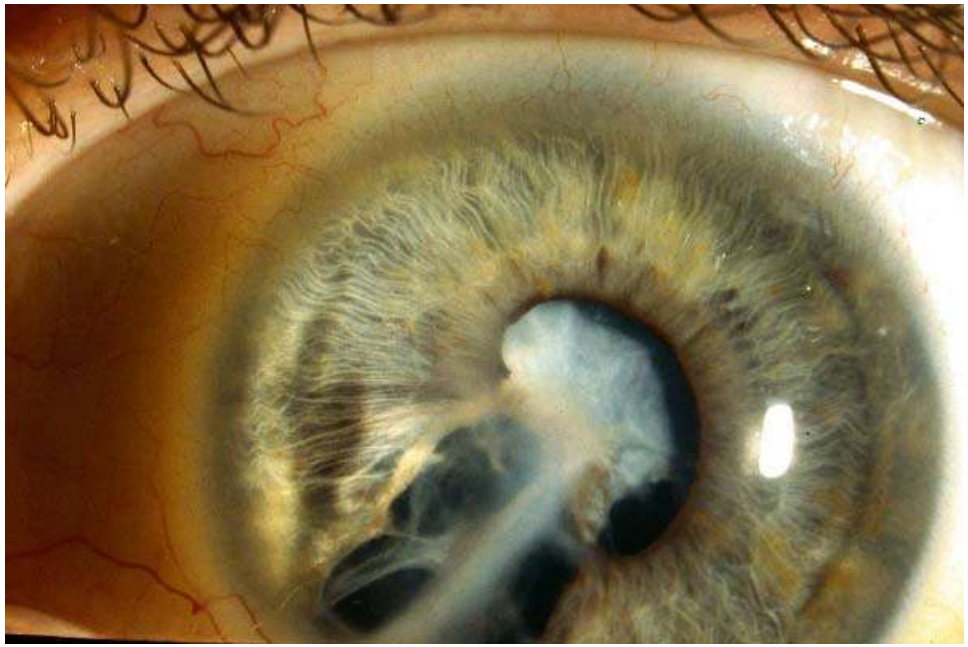


What are glaucoma risk factors?

- History
  - Family history
  - Race
  - Age
  - Refractive Error
  - Diabetes
  - Vasospasm

Ocular History

- Trauma
- Laser
- Surgery
- Other

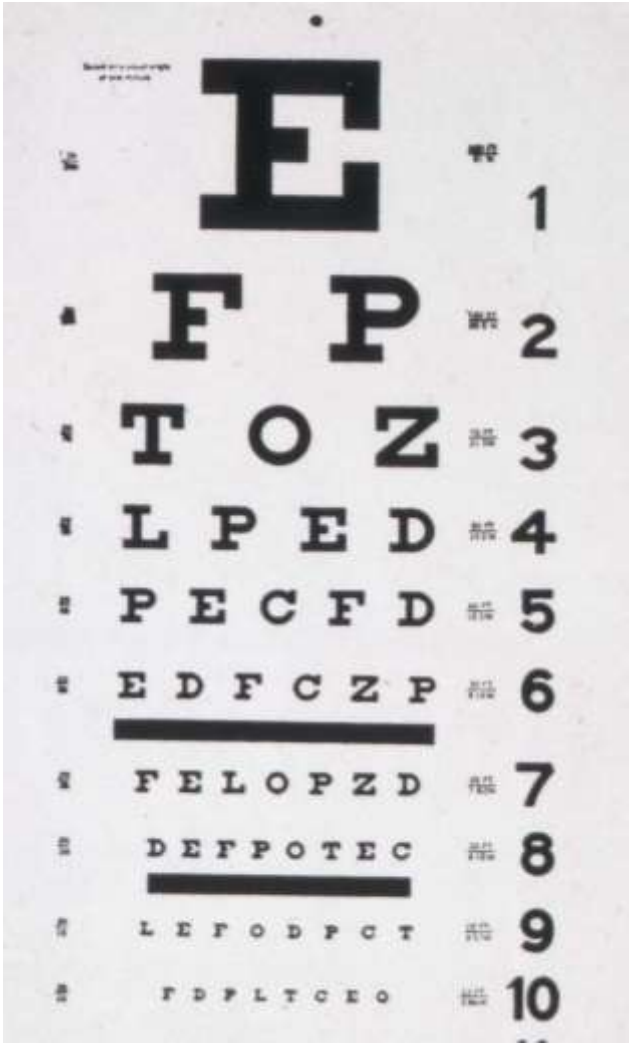


Medical History

- Diabetes
- Hypertension
- Asthma
- Other
- Known drug allergies/reaction

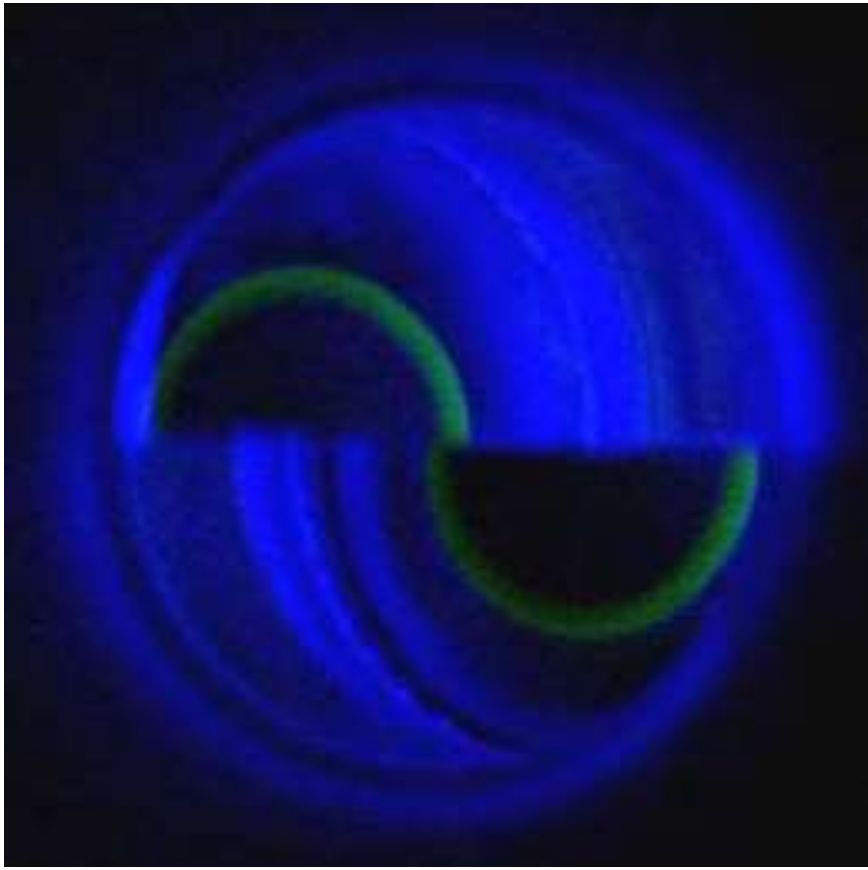
Vision

- BSCVA
- State refraction – myope vs. hyperope
- RAPD



Tip Two

- Intraocular pressure is neither necessary nor sufficient for the diagnosis of glaucoma
- Intraocular pressure is, however, the most important risk factor



IOP

- The higher the pressure the higher the risk
- Goldmann technique preferred
- Tonopen, etc. if necessary

What else is important?

- Slit lamp examination
  - Classify type based on angle structures
  - Look for signs of secondary glaucoma
- Optic nerve head examination
  - Stage disease based on ONH damage
- Visual field examination
  - Stage disease based on VF damage

Slit lamp examination

- Lids/lacrimal
- Conjunctiva/
- sclera
- Cornea
- Lens

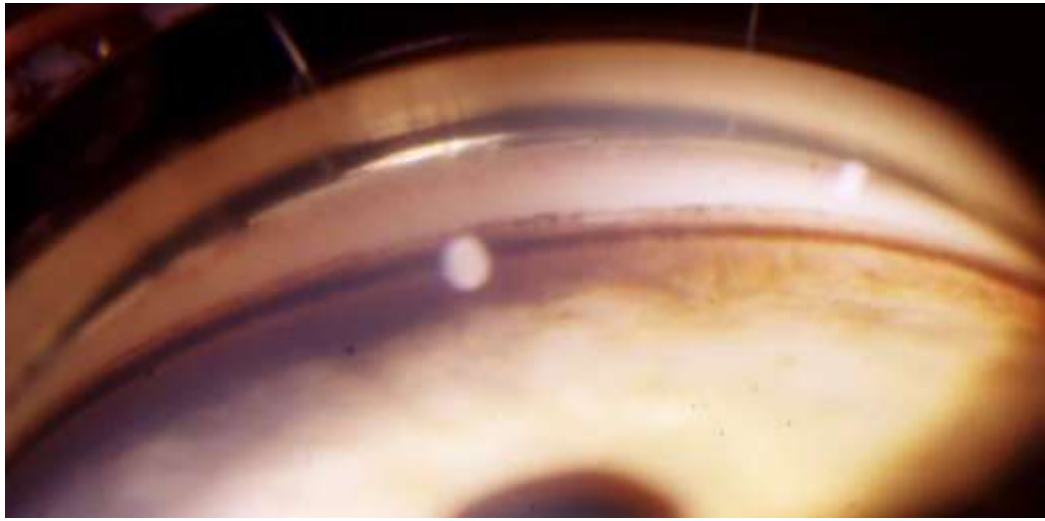
- Other
  - PXE
  - PDG
  - NVI
  - Iritis
  - PI

Gonioscopy

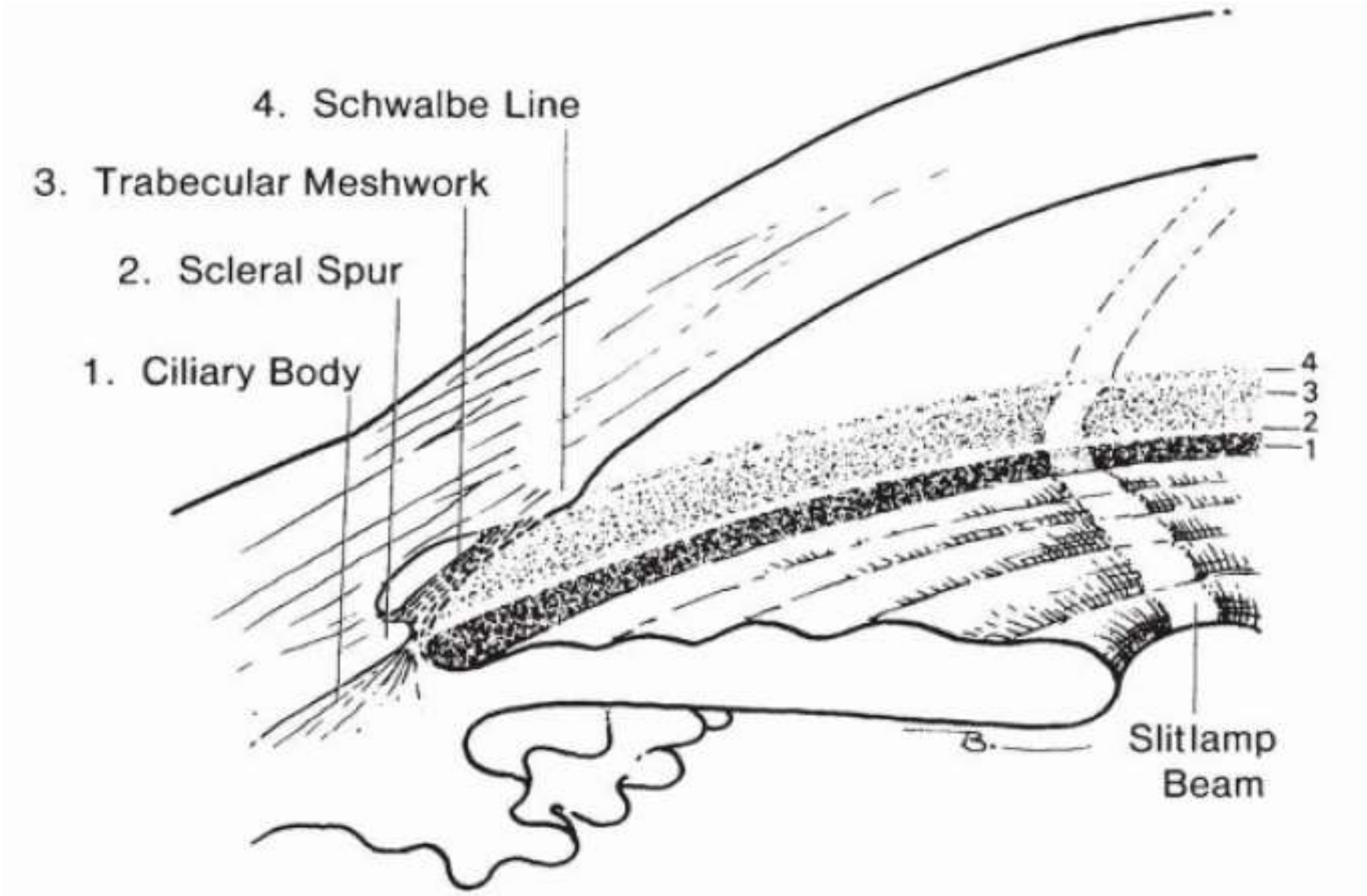
shows you the structures of the angle

- Always performed on any patient where glaucoma is a possibility

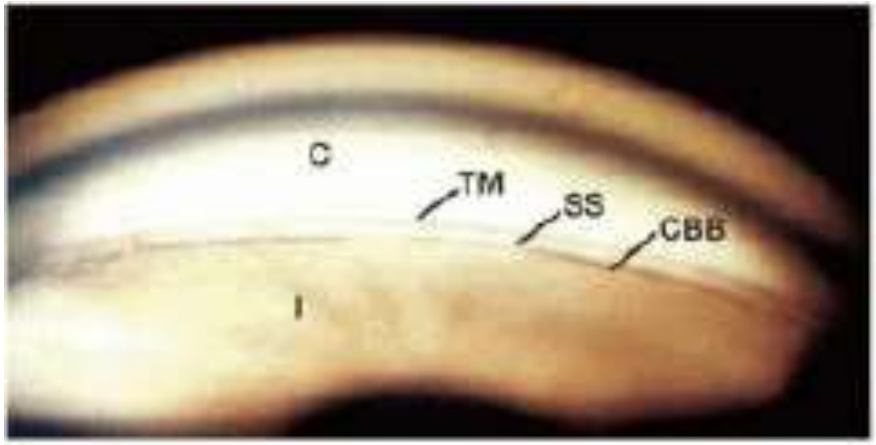
- Classify into open vs. narrow vs. closed angle



What is shown in gonioscopy



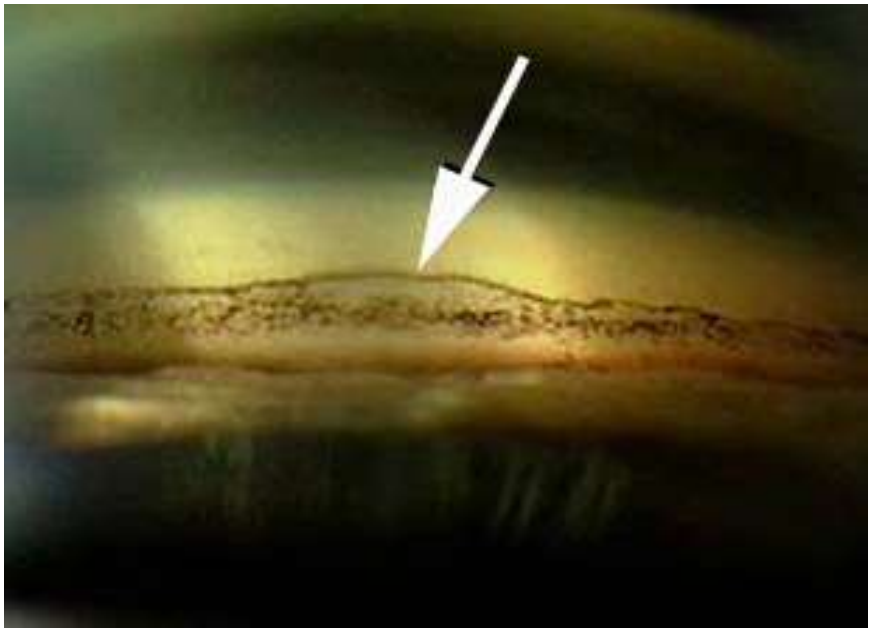
Pigment dispersion syndrome (open angle)

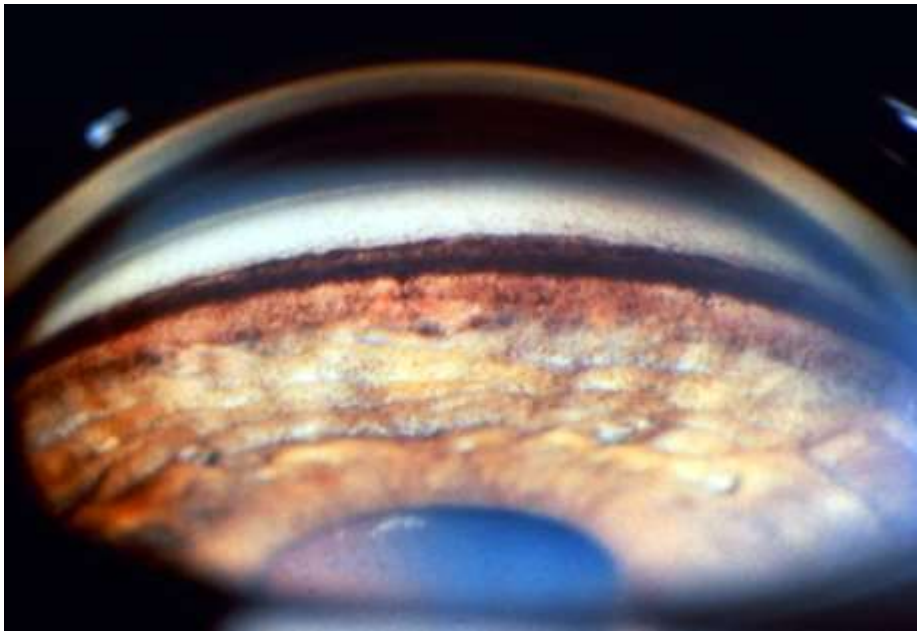


A

B

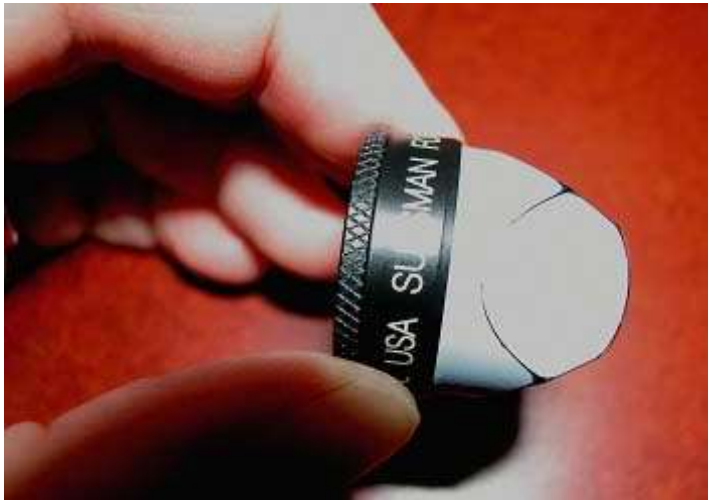
Gonioscopy- Look for secondaries





Tool One

- Four mirror lens excellent for compression gonioscopy
  - This differentiates between appositional and synechial closure
- Three mirror lens also fine



Gonioscopy lenses/mirrors

Tool Two

- Corneal thickness is becoming more and more important in glaucoma diagnosis
- Pachymetry is not, however, currently a part of the standard of care
- Prior LASIK will result in very thin central cornea

lasik for myopia → central thickness is affected
lasik for hyperopia → peripheral thickness is affected

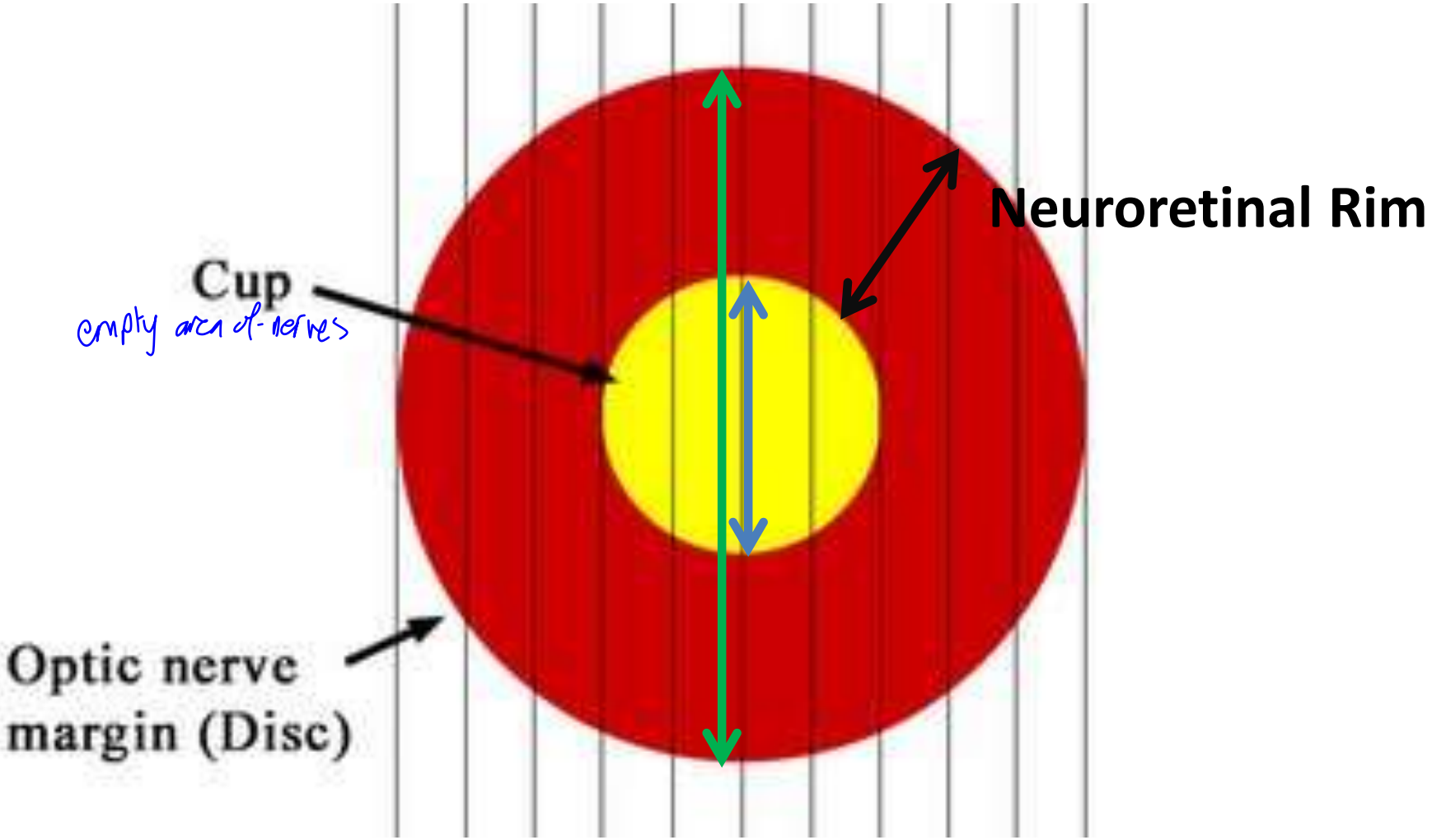
- Thin cornea can give a falsely low IOP reading
  ↳ risk factor for glaucoma (as it's underestimated)
- Thick cornea can give falsely high IOP

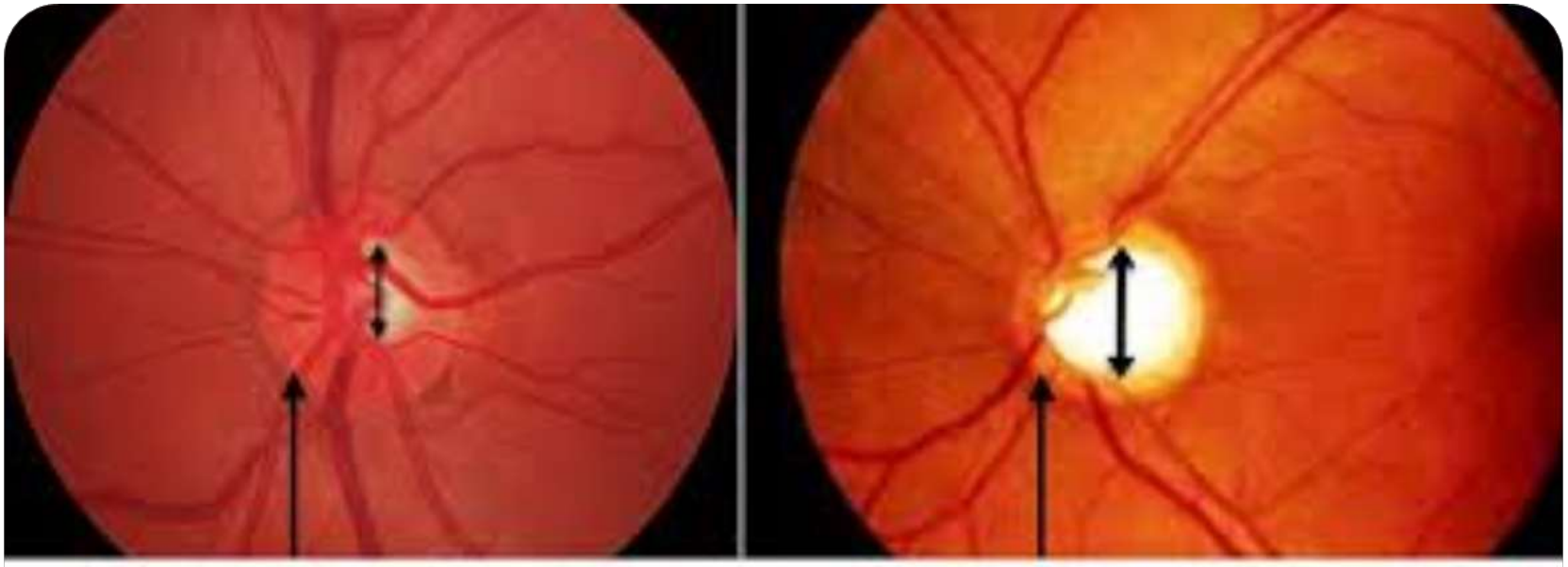
Optic Nerve & Retina

- State C:D ratio
- Note other findings:
  - Thin rim
  - Notch
  - Drance hemorrhage
  - Peri papillary atrophy, α or β
- State relevant retinal findings:
  - AMD, etc

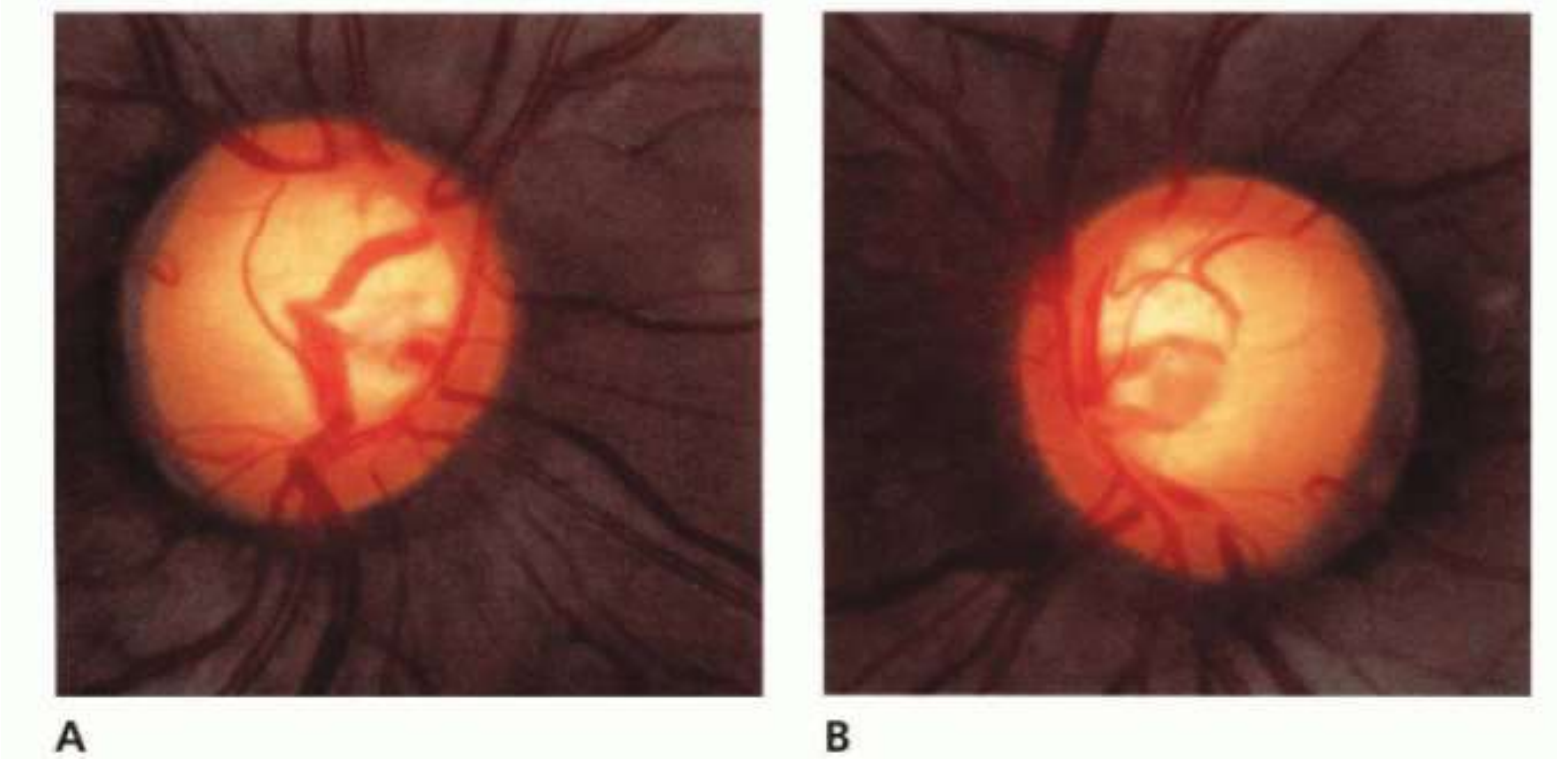
Vertical **Cup** to **Disc** Ratio

↳ area of nerve fibers

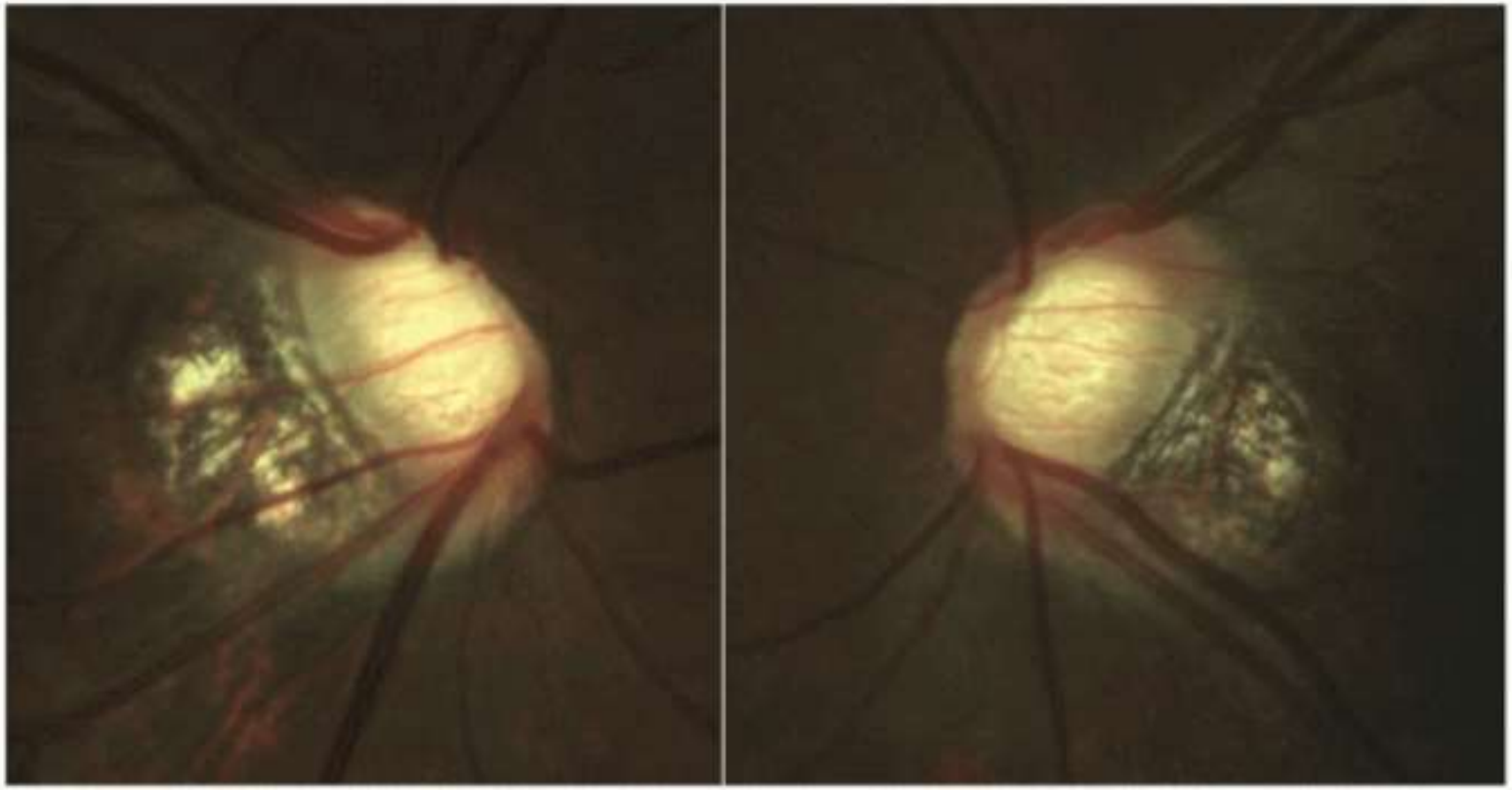



Normal optic nerve head
Glaucomatous cupping

Variants of Normal

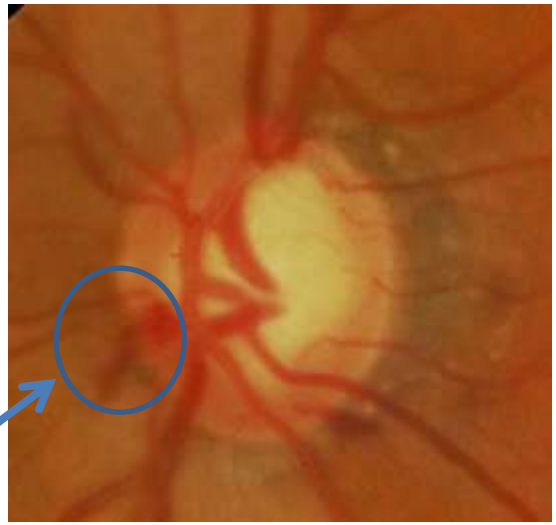


Challenging Nerves

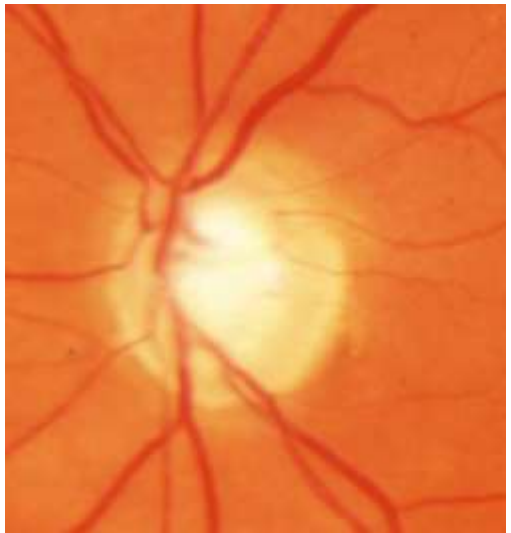


Optic Nerve Head Examination

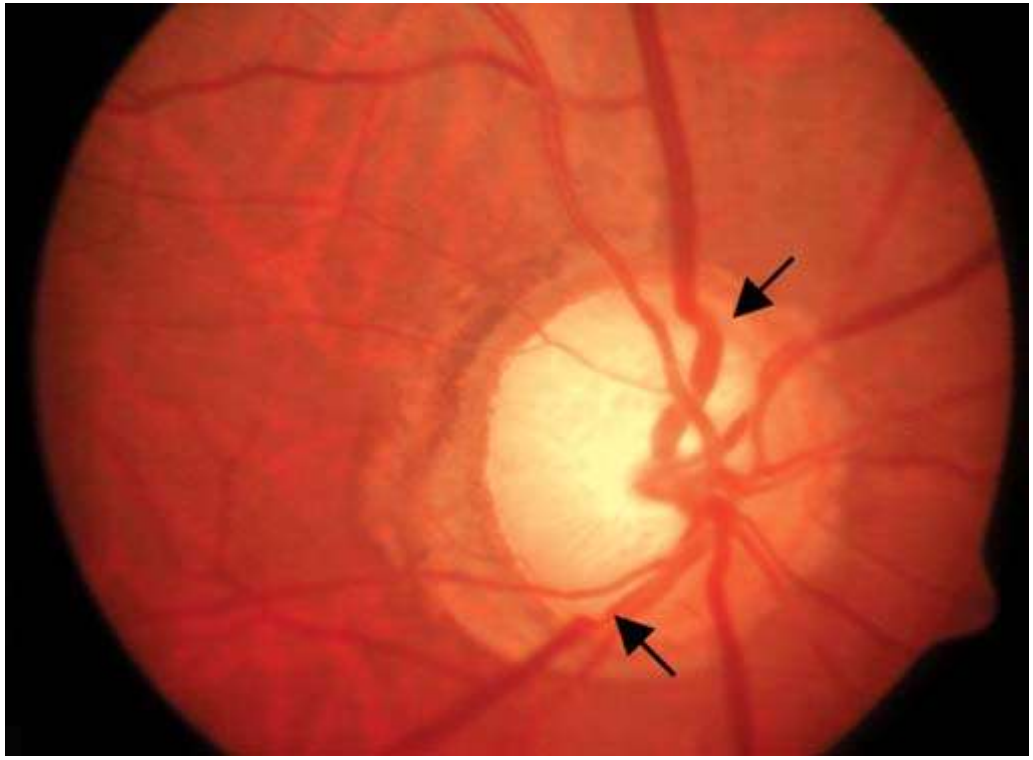
- Look for:
  - Cupping
  - Asymmetry
  - Notching
  - Hemorrhages
  - ISNT rule



(disk hemorrhage on nerve → sign of glaucoma)



Inferior, Superior, Nasal, Temporal
↳ order of fibers affected by glaucoma

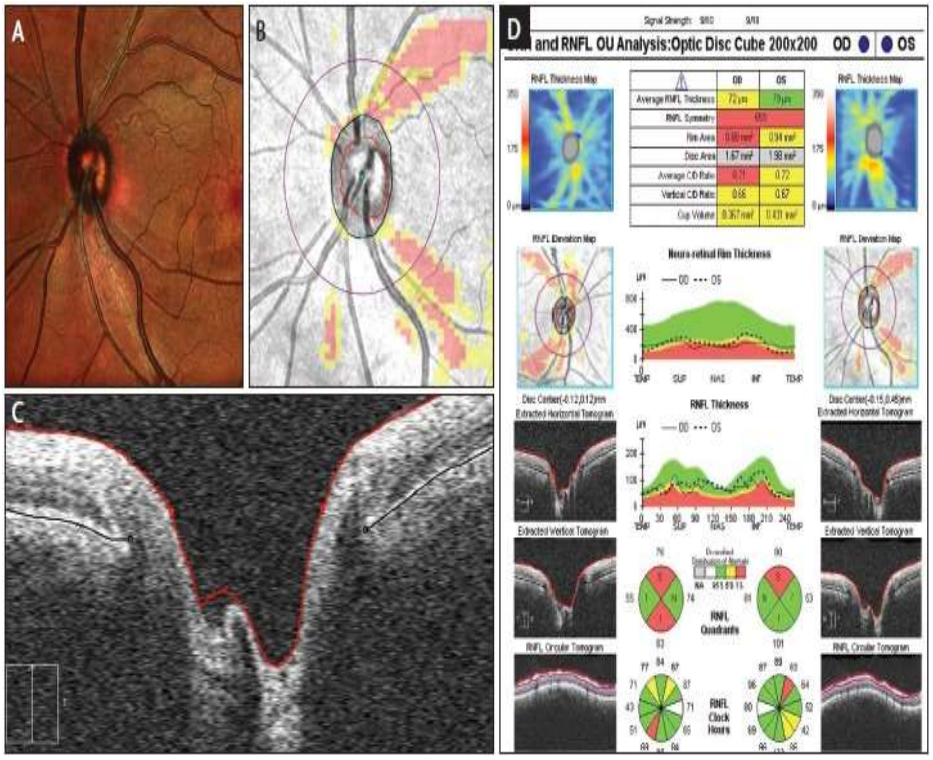


Tool Three

- Stereoscopic viewing at the slit lamp with a 66D or 78D lens and a dilated pupil

Tool Four –
Optic Nerve Head Imaging

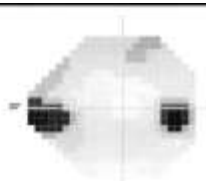
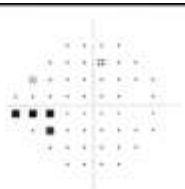
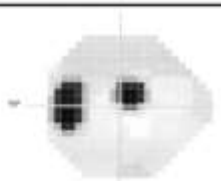
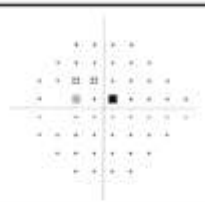
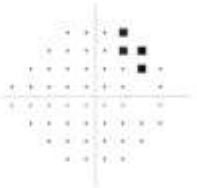
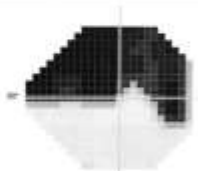
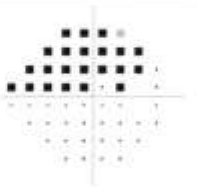
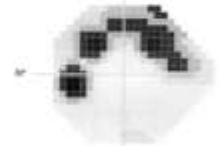
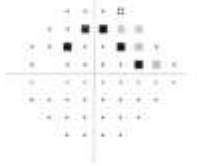
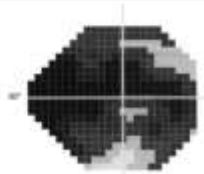
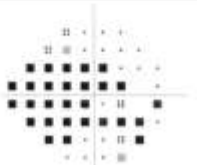
- Computer aided imaging of optic nerve and/or nerve fibre layer
  - Confocal scanning laser ophthalmoscopy / HRT
  - Optical coherence tomography/OCT
  - Polarimetry / GDx
- These are all commercially available – and costly!



Tool Five – Visual Field Examination

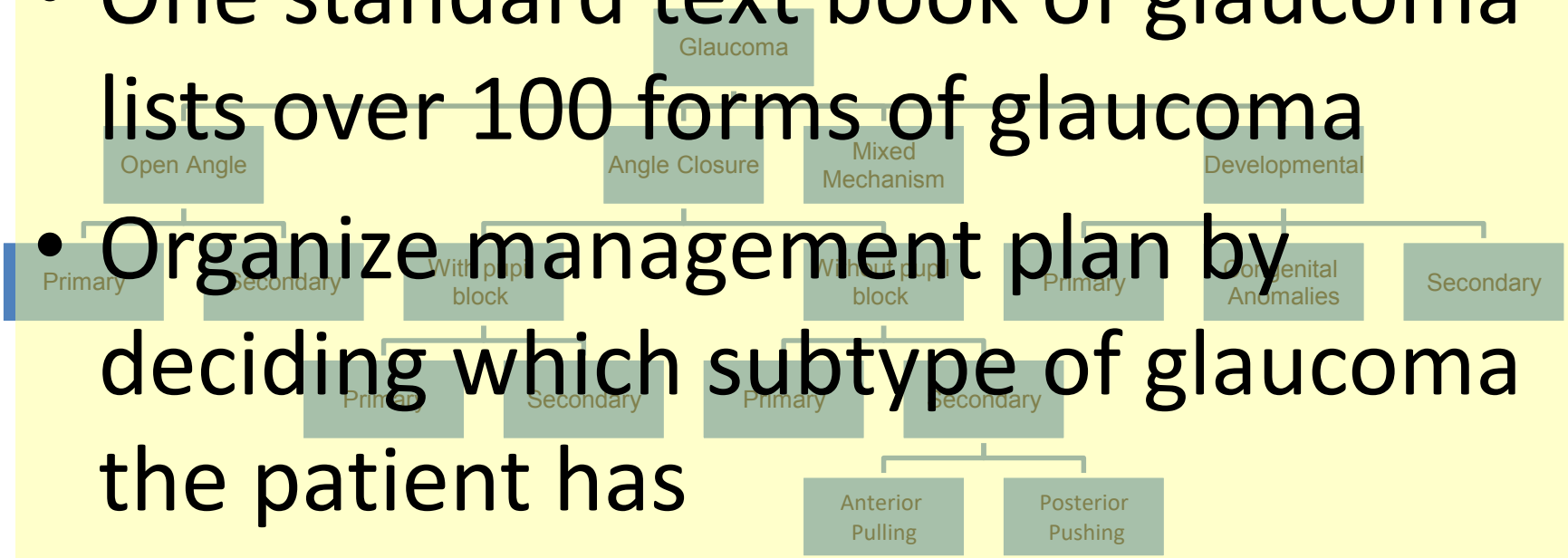
- Assess functional damage prior to patients perception of field loss

- Assess patient's performance in relation to age matched normal database (Statpack, SITA, Octopus)

Nasal Step		
Paracentral		
Temporal Wedge		
Altitudinal		
Arcuate		
Advanced		
		Probability Symbols P < 5% P < 2% P < 1% P < 0.5%

Tip Three

- One standard text book of glaucoma lists over 100 forms of glaucoma
- Organize management plan by deciding which subtype of glaucoma the patient has



Treatment

- Optic nerve damage in glaucoma is irreversible
- Treatment is aimed at maintaining the residual optic nerve function
- Most modifiable risk factor is IOP
- Lowering IOP Increases the chances of slowing down or stopping nerve damage.

Treatment Modalities

- Medical
- Laser
- Surgical

Medical Treatment

- Prostaglandin analogues (PGAs):
  - Once daily
  - Increases uveoscleral outflow
  - Proinflammation- causing hyperemia

↳ work on uveoscleral outflow

(contraindication & it induces labour and in uveitic glaucoma (it's proinflammatory)

* eye drops do reach the systemic circulation

Side effects

Ocular

- Conjunctival hyperaemia
- Eyelash lengthening, thickening, hyperpigmentation
- Irreversible iris hyperpigmentation
- Periorbital fat loss
- deepening of the upper lid sulcus
- Hyperpigmentation of periocular skin – Common but reversible

- B blockers:
  - Twice daily
  - Decrease aqueous production
  - Contraindicated in patients with bradycardia/ heart block/ asthma

- α_2 agonists :
  - Aqueous Suppressant
  - Neuroprotective
  - Can cause severe allergic reactions/ contact dermatitis

Bromodiridine → safe in kids

→ can cause severe hypotension

- Carbonic Anhydrase Inhibitors
  - Systemic (Acetazolamide)
  - Topical
  - Sulfonamide derivative/ watch out for allergy
  - Aqueous suppressant

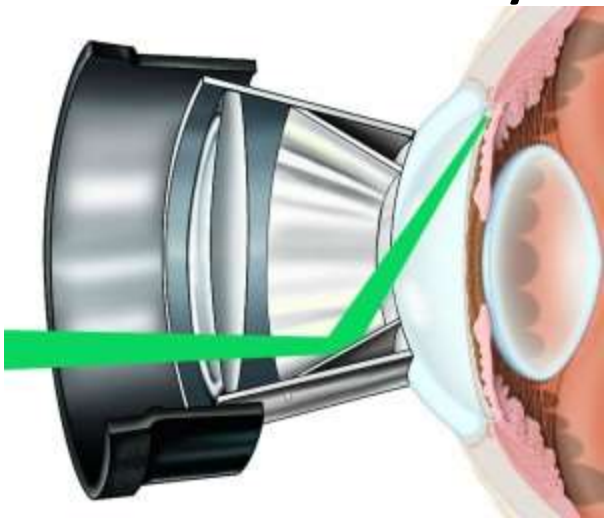
- Parasympathomimetic / Cholinergic agonists
  - Pilocarpine
  - Increase conventional pathway outflow
  - Cause miosis, myopic shift
  - May increase retinal detachment risk

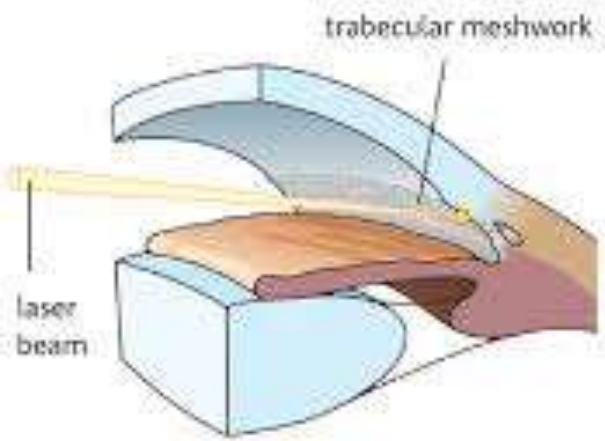
Laser Treatment

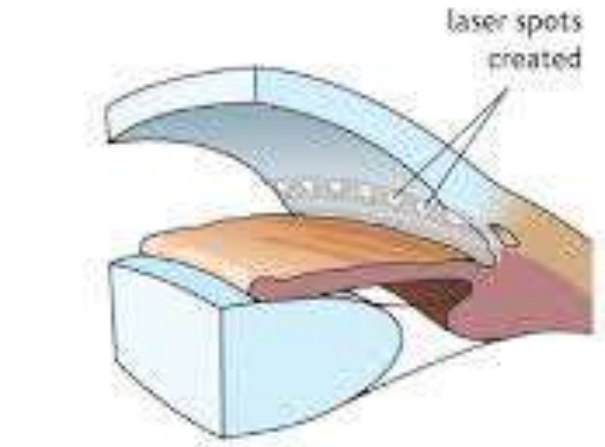
- Increase Outflow Facility
  - Trabeculoplasty
  - Iridotomy
- Decrease Aqueous production
  - Cyclodiode laser

Trabeculoplasty

- Series of laser burns at the TM to increase outflow facility

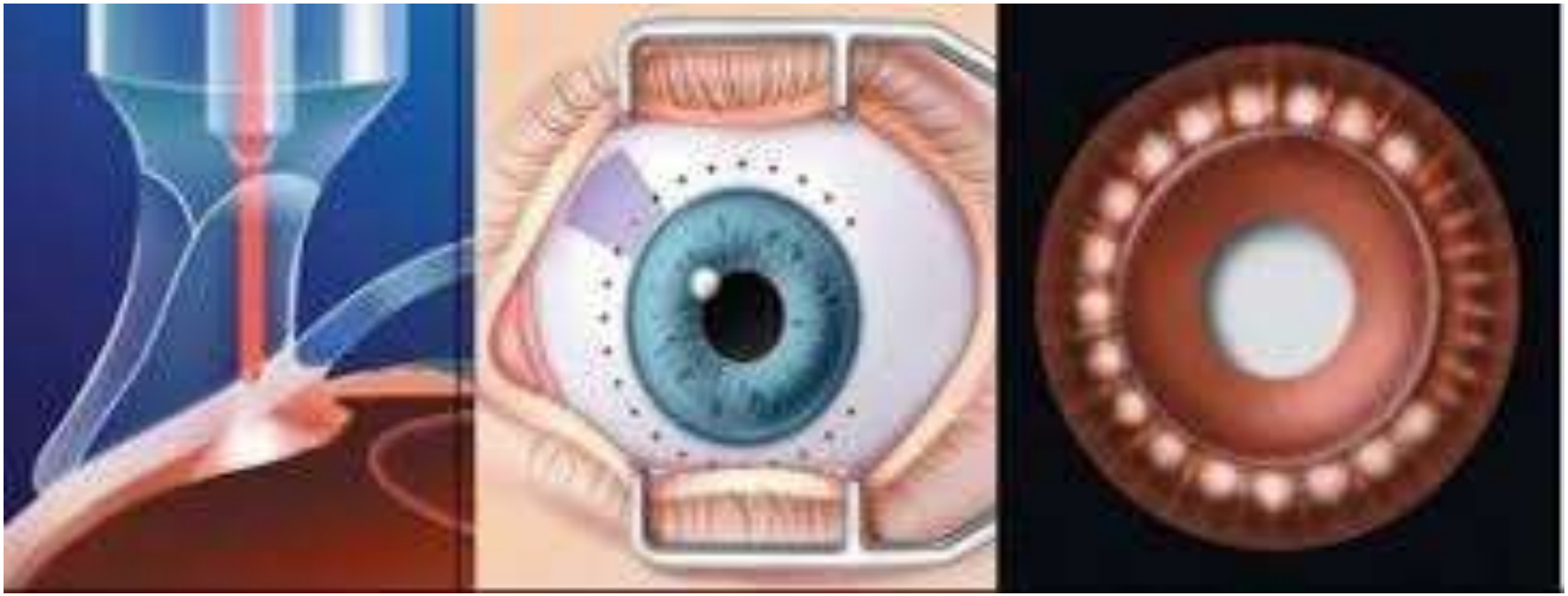






Cyclodiode

- Transscleral Ciliary body Ablation to decrease aqueous production

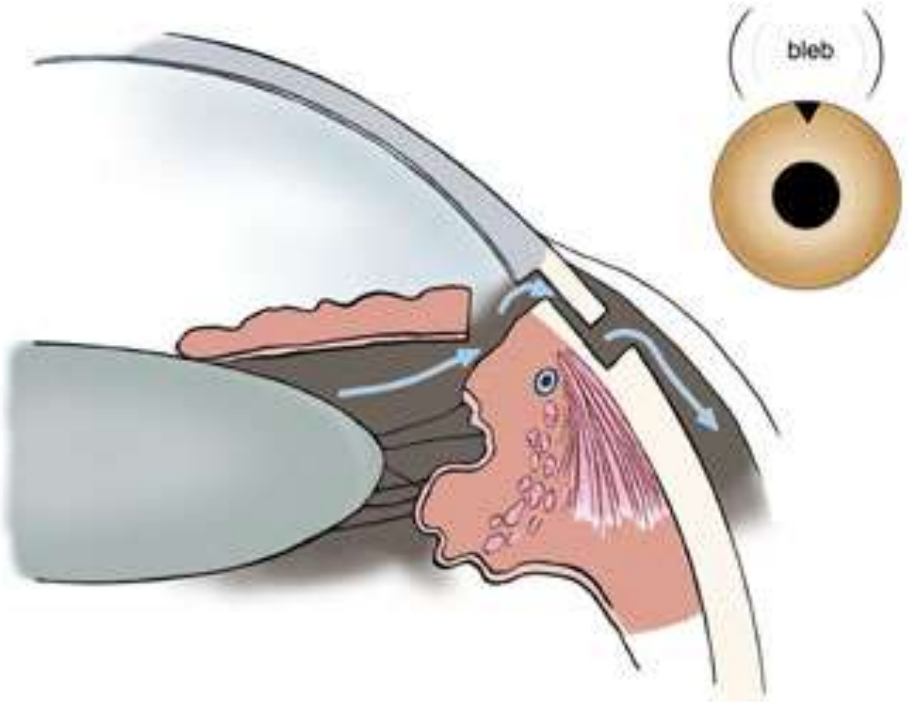


Surgery

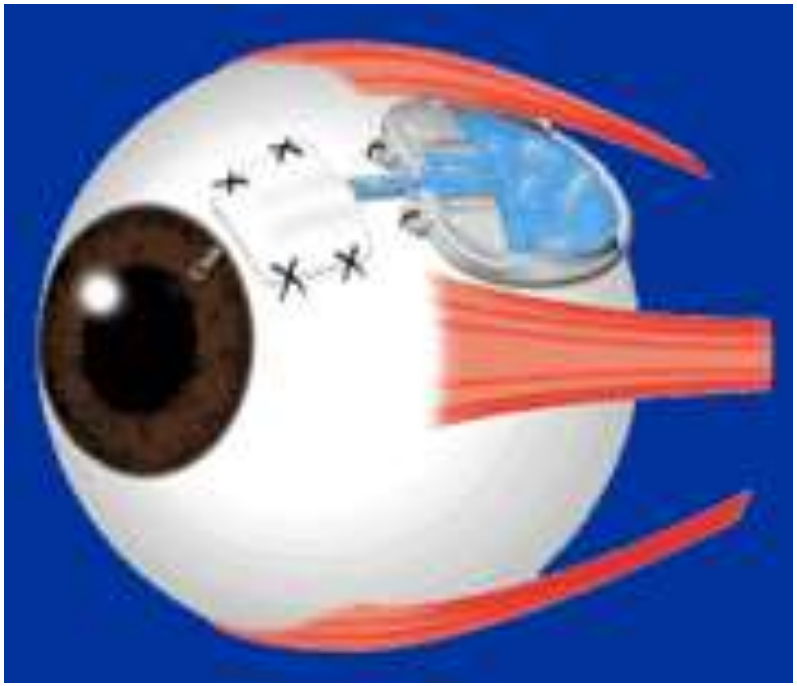
- Trabeculectomy
- Glaucoma Drainage Devices

Trabeculectomy

- A fistula between the anterior chamber and the subtenon space



Glaucoma Drainage Devices



Summary

- **All** patients have glaucoma until proven otherwise
- **Risk** assessment is based on IOP, other risk factors
- **Classify** based on gonioscopy and other anterior segment findings
- **Stage** the disease based on optic nerve and field changes

+ in normal tension glaucoma ⇒ the lower the better, lower the pressure as part of trt.

The End