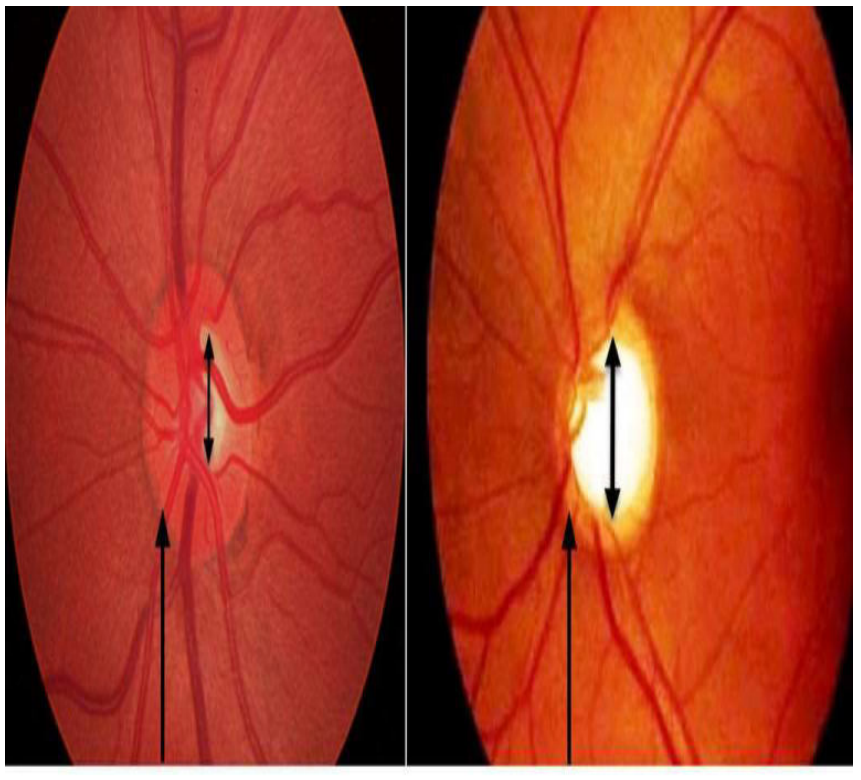
Primary open angle glaucoma

Acknowledgement

- Kanski's Clinical Ophthalmology (8th Edition).
- Becker- Schaffer's Diagnosis and therapy of The Glaucomas (8th Edition).
- Comprehensive Ophthalmology (A.K.Khurana) (7th Edition).

Learning Objectives

- **At the end of this class the students shall be able to :**
- Define primary open angle glaucoma(POAG).
- Comprehend the pathophysiology and risk factors of POAG.
- Understand the clinical features of POAG.
- Understand the fundamentals of managing primary open angle glaucoma



Normal optic nerve head Glaucomatous cupping

Question

Glaucoma is defined as:

- **a.** a group of diseases that have in common a characteristic optic neuropathy associated with increased intraocular pressure.
- **b.** a group of diseases that have in common a characteristic optic neuropathy with associated visual function loss.
- **c.** a group of diseases that have in common high intraocular pressure with or without optic neuropathy.
- **d.** a group of diseases that have in common a characteristic optic neuropathy with poor visual acuity.

Definition of POAG

- Chronic, progressive optic neuropathy characterised by morphological changes at the optic disc and retinal nerve fibre layer leading to characteristic visual field changes, in the absence of other ocular diseases or congenital anomalies (with or without a raised IOP).

Etiopathogenesis

- Multifactorial aetiology
- Risk factors include:

➢ Elevated Intra Ocular Pressure(IOP)

(More than 21 mm Hg)

➢ Optic disc cupping

➢ Increasing Age : More common in 5th to 7th decades

➢ Race: More common and severe in Black population

Etiopathogenesis

➢ Heredity/ Family History: Risk of about 10% in siblings; 4% in off springs

➢ Diabetes

➢ Systemic Hypertension

➢ Myopia

➢ Thin central corneas

➢ Steroid usage

➢ ??Migraine, Cigarette smoking

Pathophysiology of POAG

- Decrease in aqueous outflow facility due to increased resistance to outflow leads to rise in IOP
- Two theories of axonal loss in optic disc
- 1. Mechanical: Distortion of lamina cribrosa leading to impaired axoplasmic flow

2. Vascular: Optic disc ischaemia with defective autoregulation of blood vessels

FORMATION OF AQUEOUS HUMOR

CILIARY PROCESSES

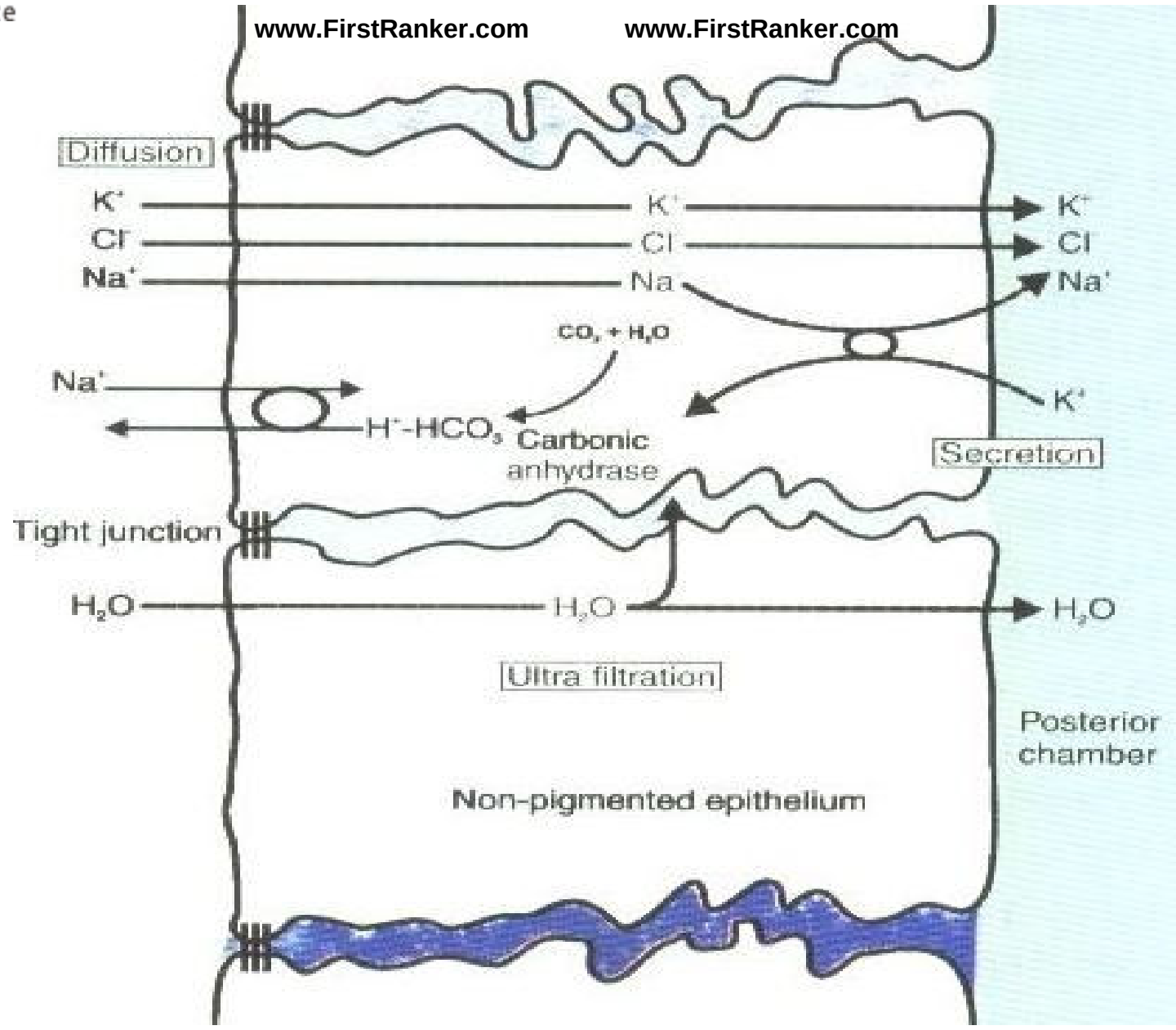
-approx. 70-80 radial folds in the pars plicata which form the site of aqueous production.

-Zonular fibers attach primarily in the valleys of the ciliary processes and also along the pars plana

DIFFUSION

SECRETION
(80-90%)

ULTRA-
FILTRATION



FORMATION PROCESSES

Formation of aqueous humor

- Diffusion and ultrafiltration are both passive mechanisms so no active cellular participation occurs.
- Active secretion is an active process.

- Rate of formation of aqueous humor in a healthy human eye is-

2 - 3 microlitres/minute

Table 1. Constituents of Human Aqueous Humor*

Constituent (µmol/mL)	Anterior Chamber Aqueous	Plasma
Ascorbate	1.06	0.04
Bicarbonate	22.0	26.0
Calcium	2.5	4.9
Chloride	131.0	107.0
Glucose	2.8	5.9
Lactate	4.5	1.9
Magnesium	1.2	1.2
Phosphate	0.6	1.1
Potassium	22.0	26.0
Sodium	152.0	148.0
Urea	6.1	7.3
Protein (gm/dL)	0.024	7.0
pH	7.21	7.4

Differences between aqueous humor & plasma

	AQUEOUS	PLASMA
-Marked deficit of proteins	0.024 gm/dl	7.0 gm/dl
-Marked excess of Ascorbate	1.06 micromol/ml	0.04 micromol/ml
-Excess of Lactate	4.5 micromol/ml	1.9 micromol/ml
-Excess of Chloride & certain amino acids		

Functions of aqueous humor

*Maintaining IOP :

-important for early ocular development & maintaining global integrity throughout life.

*Serves as a vascular system for the avascular structures of the eye: cornea, lens & TM.

- by providing substrates & nutrients & removing metabolites.

Functions of aqueous humor

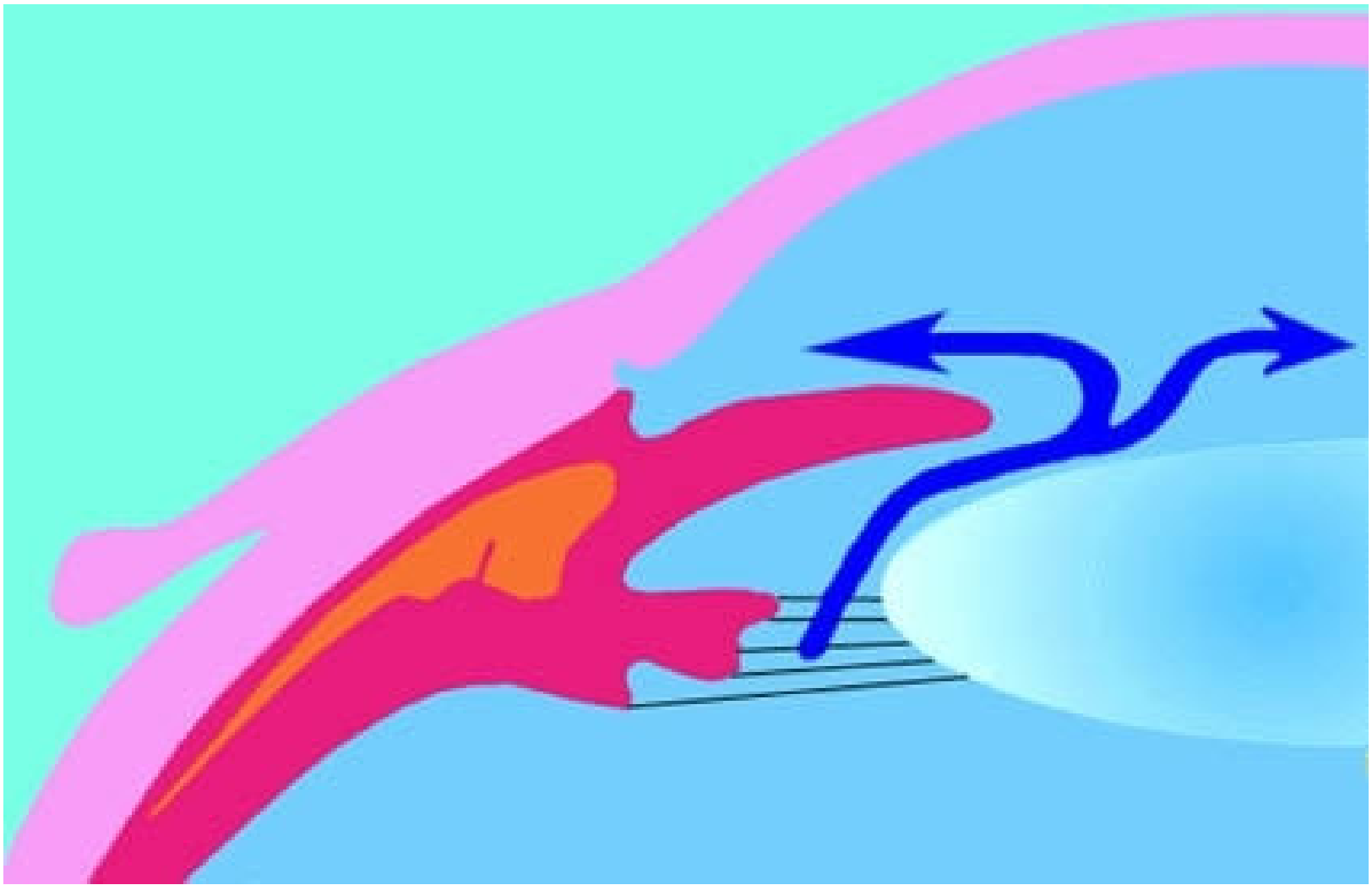
*Delivering high concentration of Ascorbate:

- scavenges free radicals & protects against UV rays & other radiations.

*Local paracrine signaling & immune responses.

*Colourless & transparent medium as part of eye's optical system.

Aqueous humor outflow



Video of aqueous humor outflow

Major amount of aqueous humor leaves the eye by

↓

BULK FLUID FLOW

↓

i.e. fluid flows along normal pressure gradient through non-energy dependent process

Ciliary processes

↓

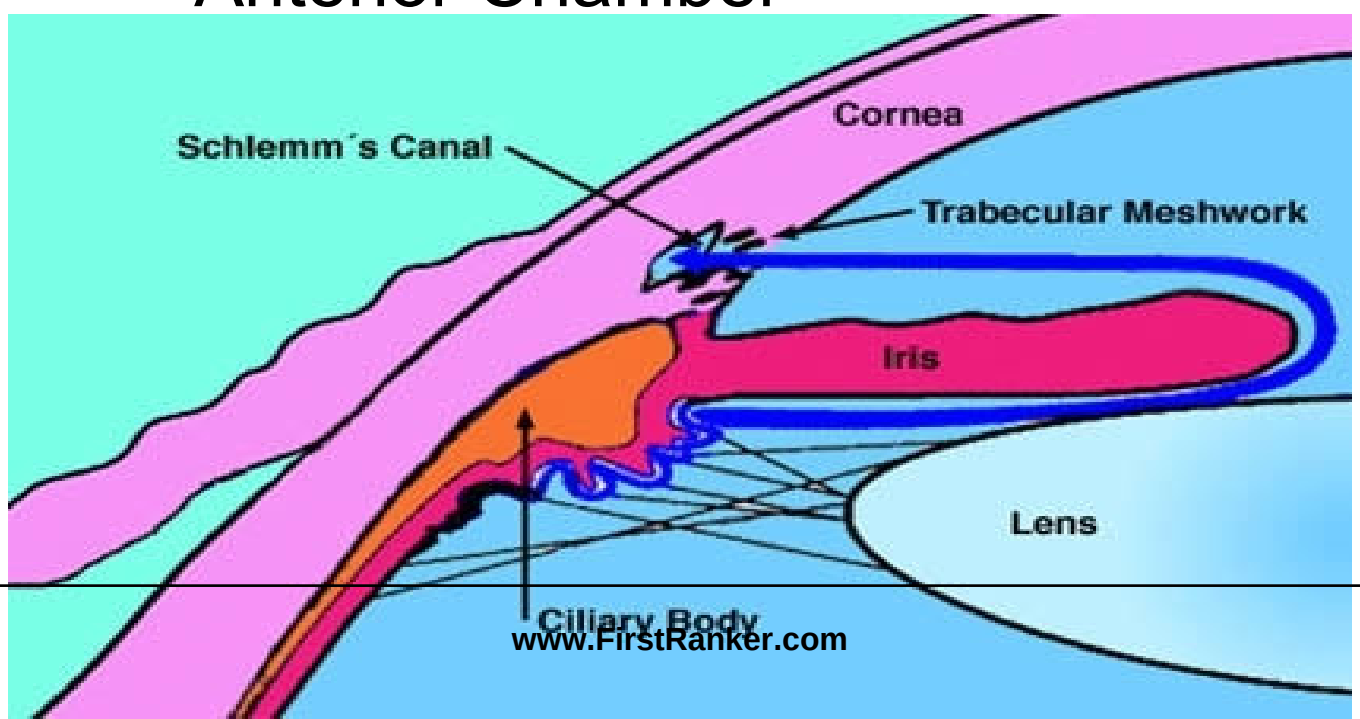
Aqueous Humor in PC

↓

through pupil

↓

Anterior Chamber



Trabeculo-canalicular outflow

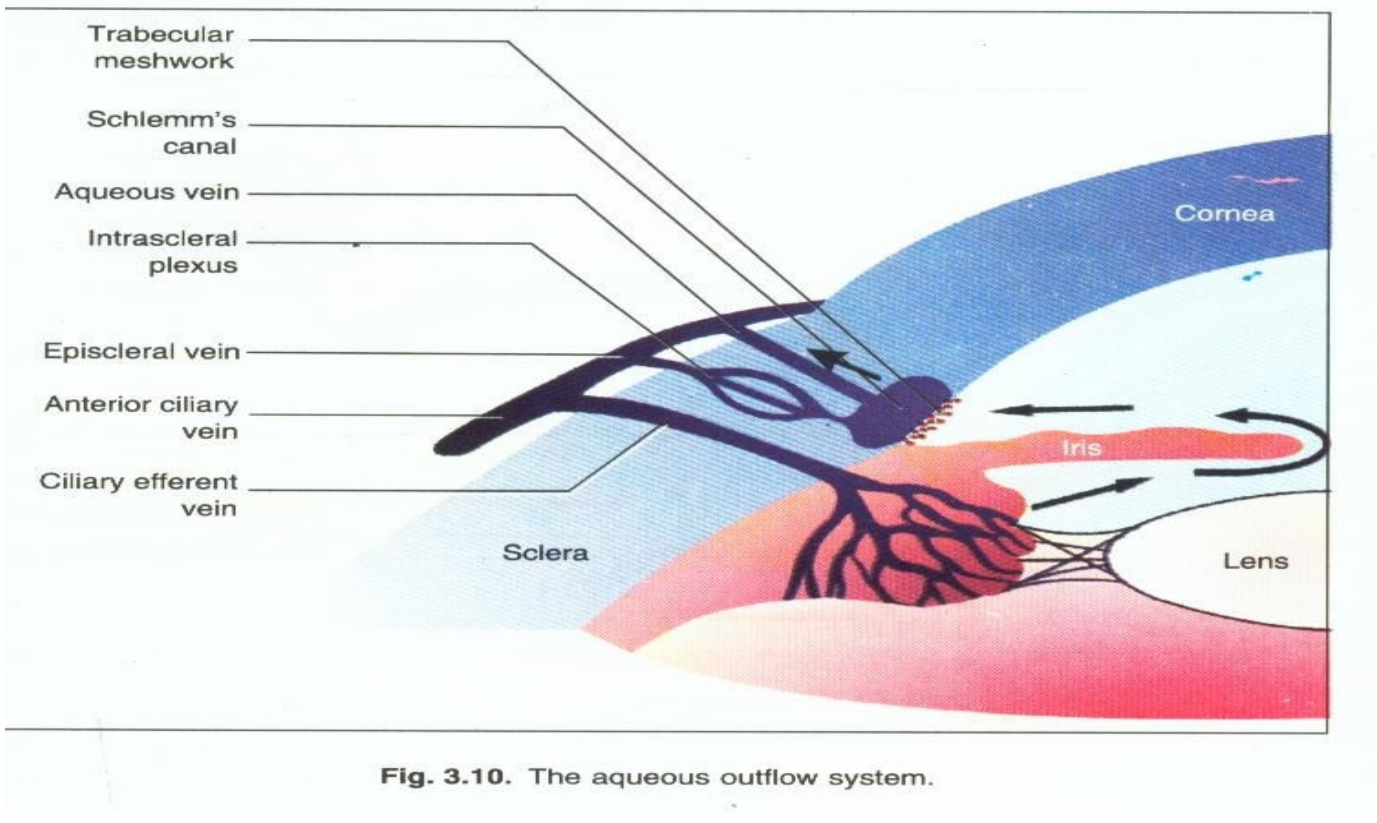


Fig. 3.10. The aqueous outflow system.

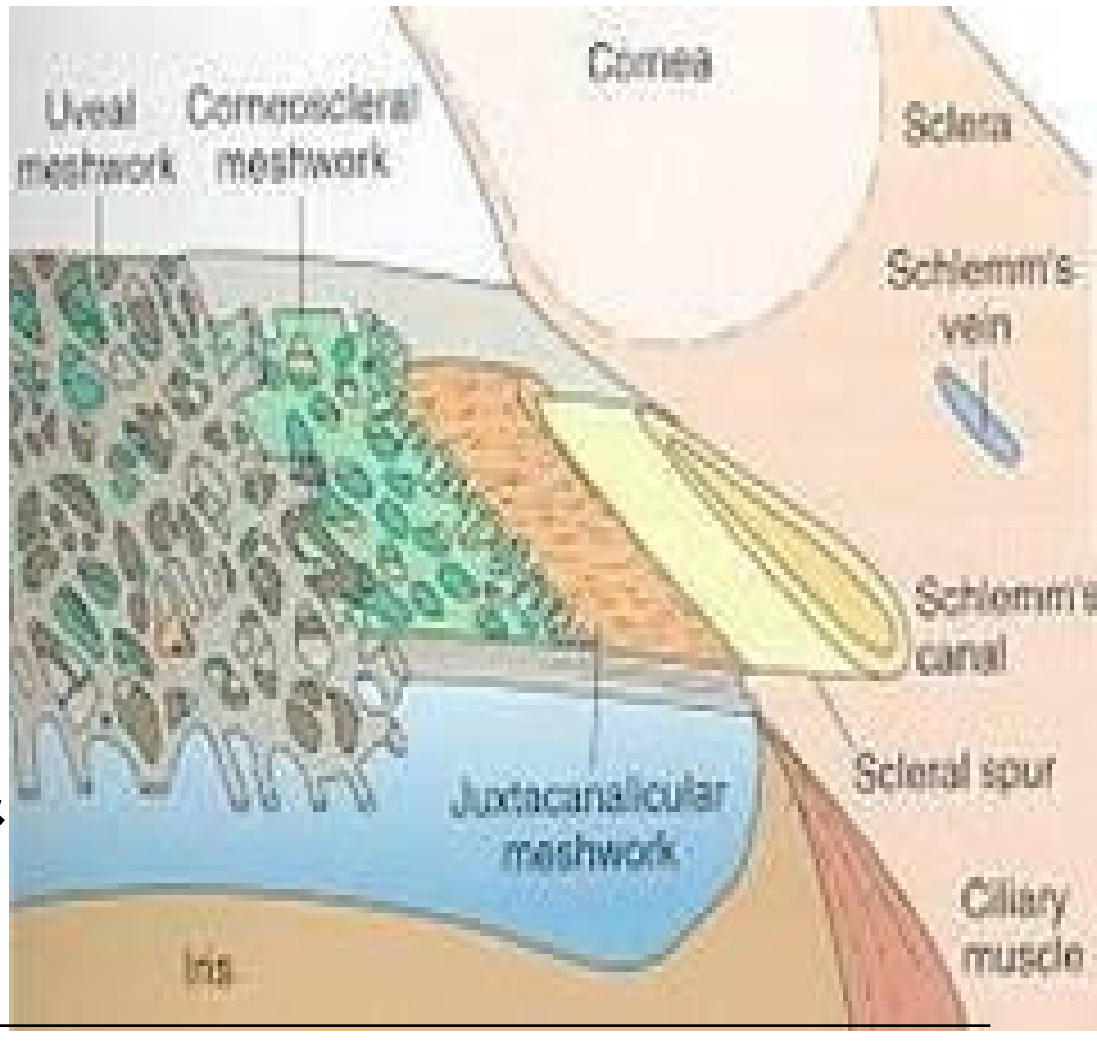
*It is the main outlet for aqueous from the AC

*70-90% of total aqueous is drained by this route

TRABECULAR MESHWORK

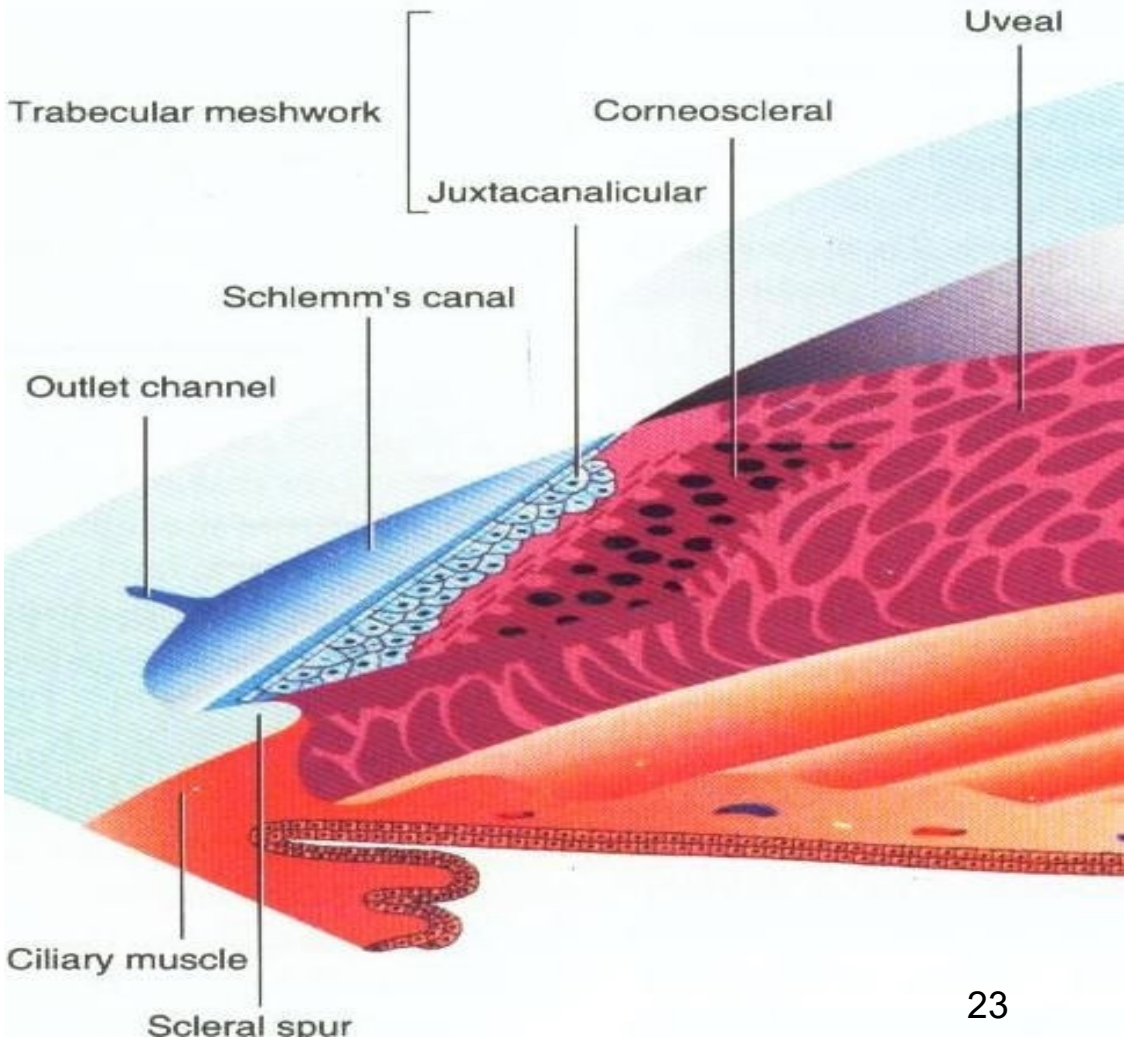
-A sponge work of connective tissue beams arranged as super-imposed perforated sheets.

- Extracellular spaces contain hydrophilic glycosaminoglycans & collagen.



JUXTACANALICULAR (ENDOTHELIAL) MESHWORK

- Outermost portion of TM which mainly offers the normal resistance to aqueous outflow
- Connects the corneoscleral meshwork with schlemm's canal



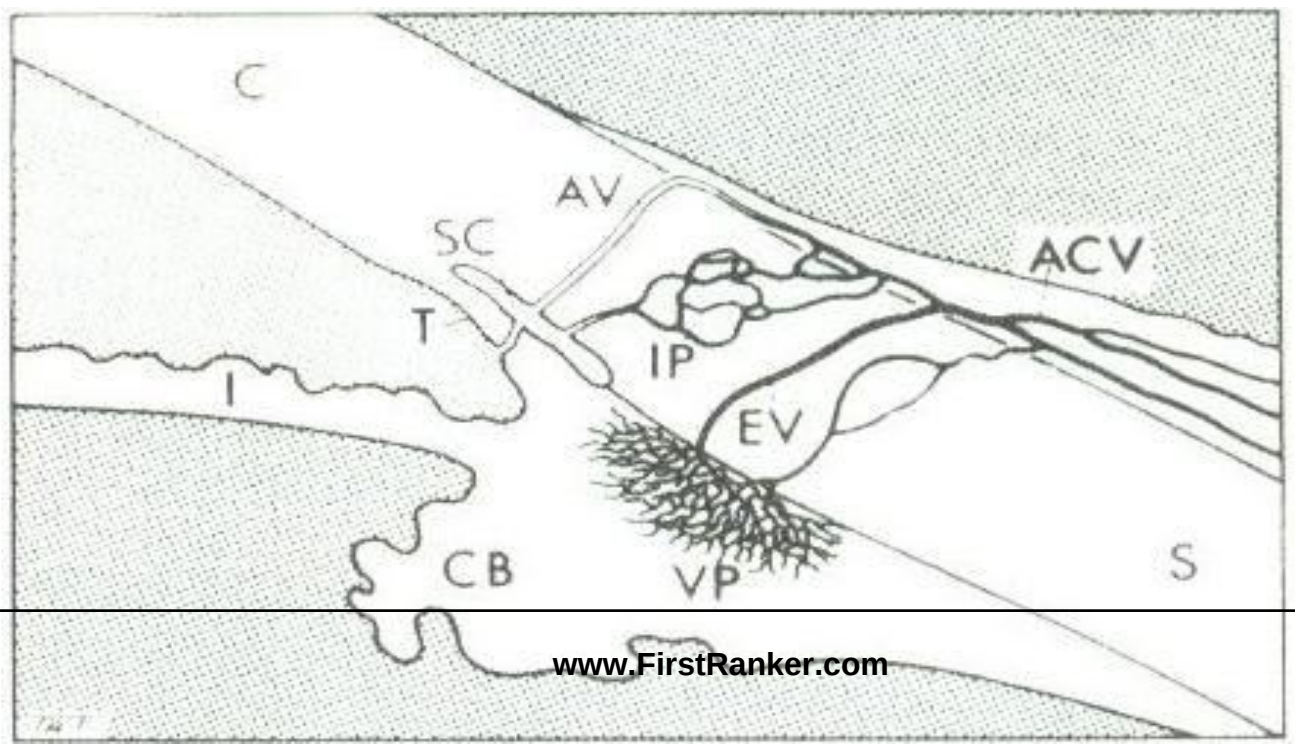
- Veins from the anterior part of ciliary body form the Ciliary venous plexus

⇩

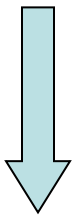
Anterior ciliary veins & Episcleral veins

⇩

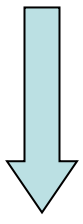
communicate with Schlemm's canal



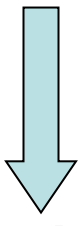
Schlemm's Canal



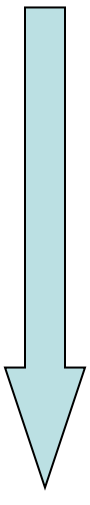
20-30 Collector channels



Intra-scleral venous plexus



Aqueous Vein



Episcleral venous plexus

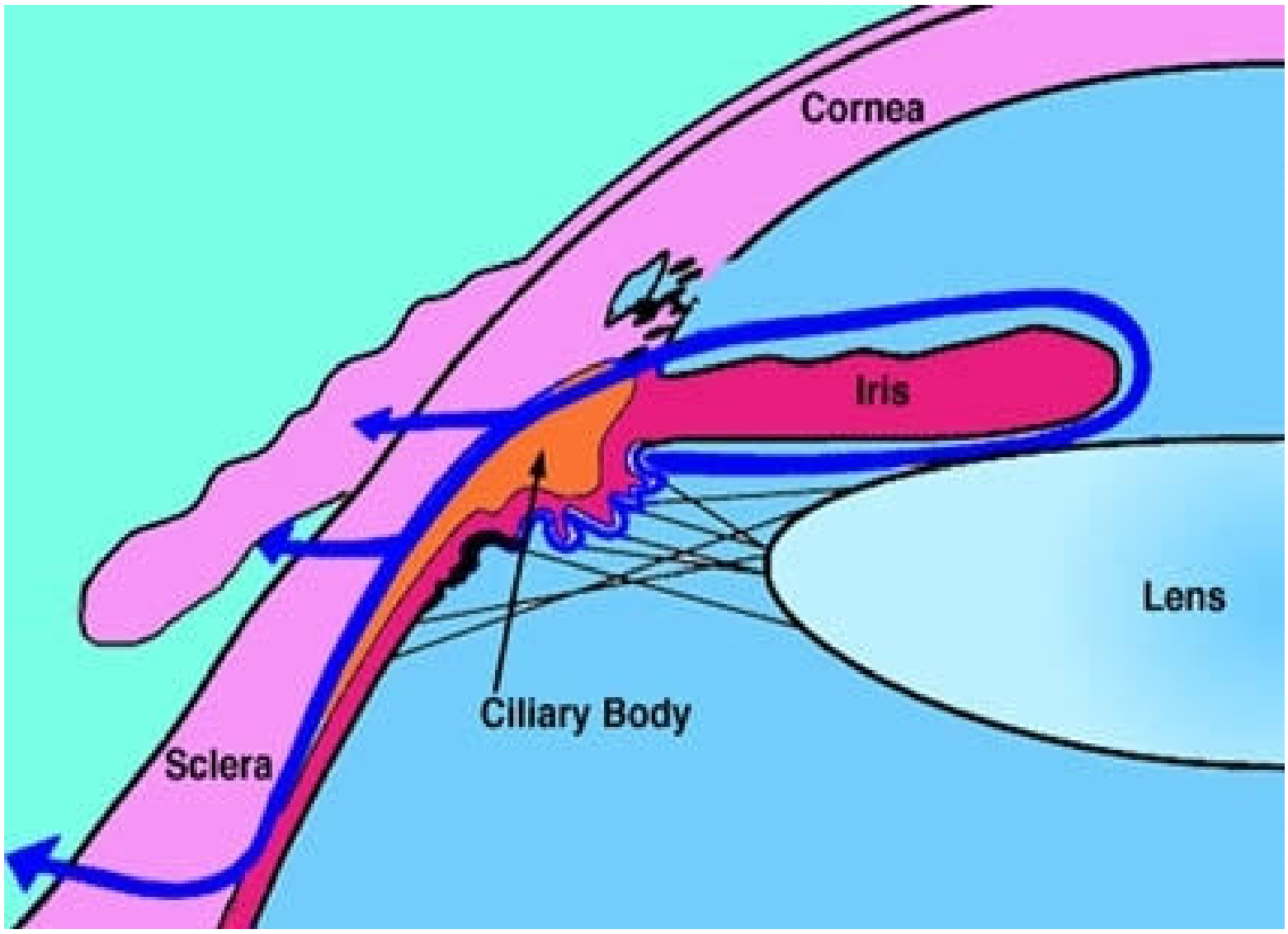
& Anterior Ciliary vein

25

UNCONVENTIONAL OUTFLOW

*responsible for 10-25% of total aqueous outflow

UVEO-SCLERAL OUTFLOW



Trans-corneal outflow

- Aqueous humor from anterior chamber goes into tear film through cornea.
- Very little aqueous passes through this pathway.
- Total volume of fluid transferred is limited by high hydraulic resistance of the cornea.

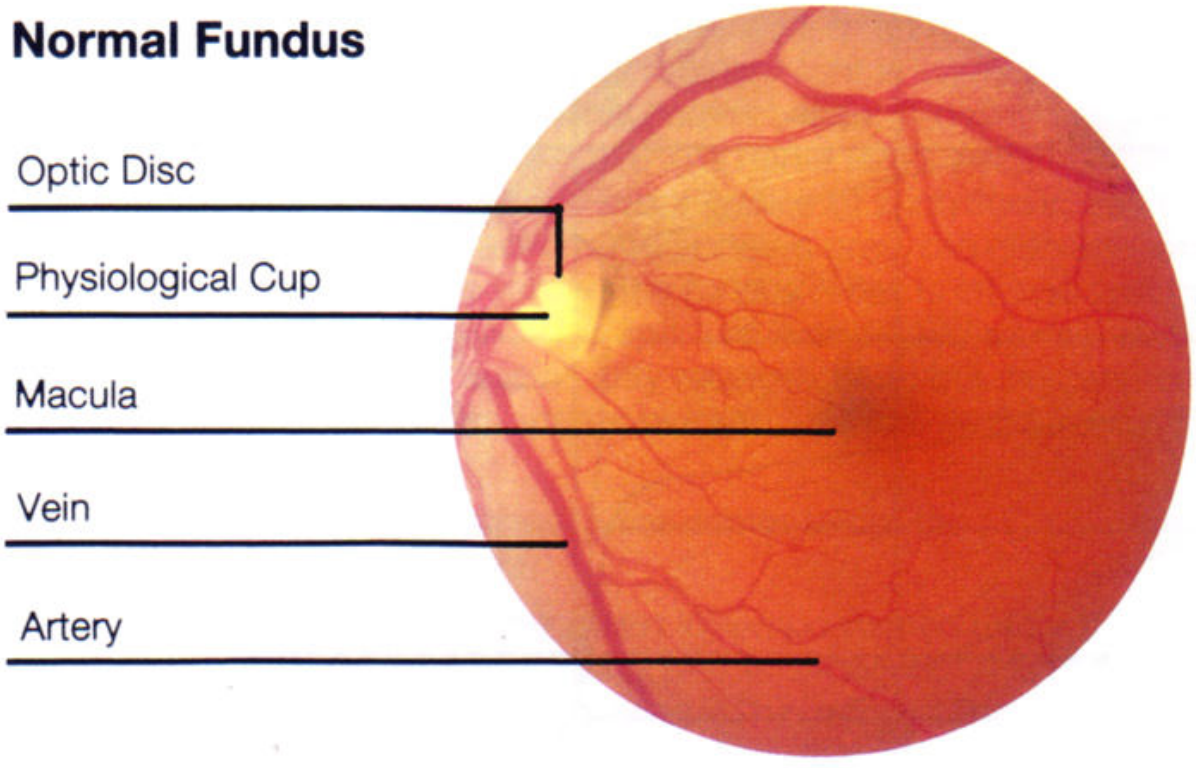
Clinical features of POAG

Symptoms

- Usually asymptomatic in early cases
- Mild headache and eye ache
- Frequent changes in presbyopic glasses
- Delayed dark adaptation
- Loss of peripheral vision
- Loss of central vision(late cases)

Signs of POAG

- Normal anterior segment
- Pupil reaction to light may be sluggish(in advanced cases only)
- Elevated IOP(More than 21 mm Hg) with diurnal variation more than 5-8 mmHg
- Optic disc changes (Progressive, asymmetric)
- Visual field defects



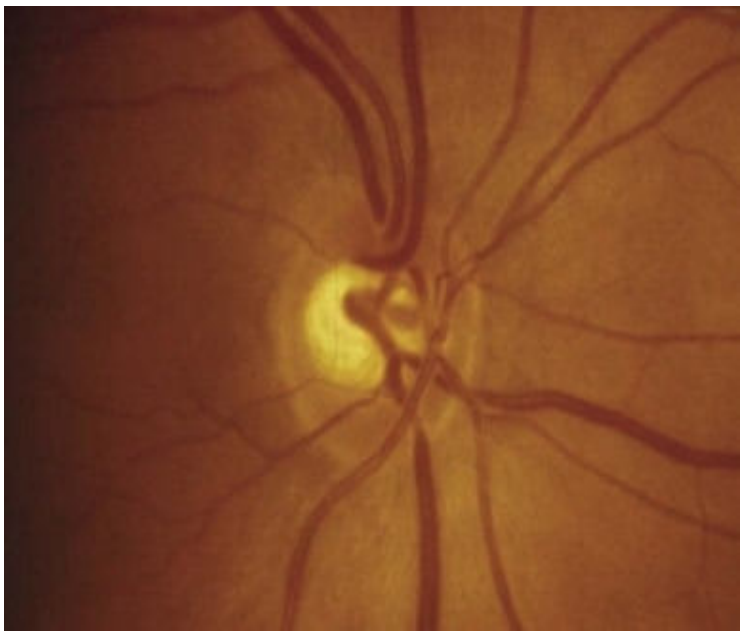
Optic disc changes in glaucoma

- Early changes
  - Retinal nerve fibre layer atrophy
  - Vertically oval cup
  - Asymmetry of the cups(More than 0.2 difference)
  - Large cup(CD more than 0.6)
  - Splinter haemorrhages

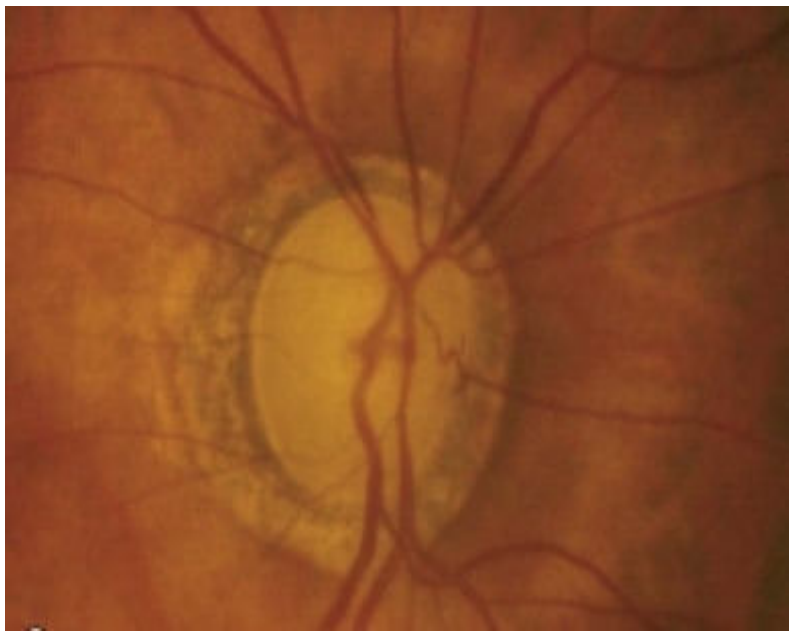
Advanced glaucomatous disc changes

- Marked cupping (More than 0.7)
- Thinning of NRR (Neuroretinal rim)
- Lamellar dot sign
- Vascular alterations
  - Nasal shifting of retinal vessels
  - Bayonetting sign(convoluted path due to NRR loss)
  - Baring of circumlinear vessels and overpass vessels
- Glaucomatous optic atrophy

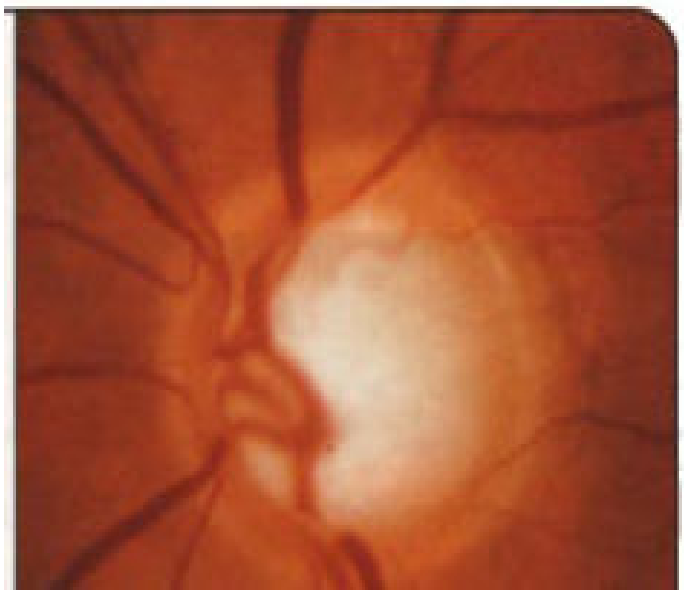
Normal Optic Disc



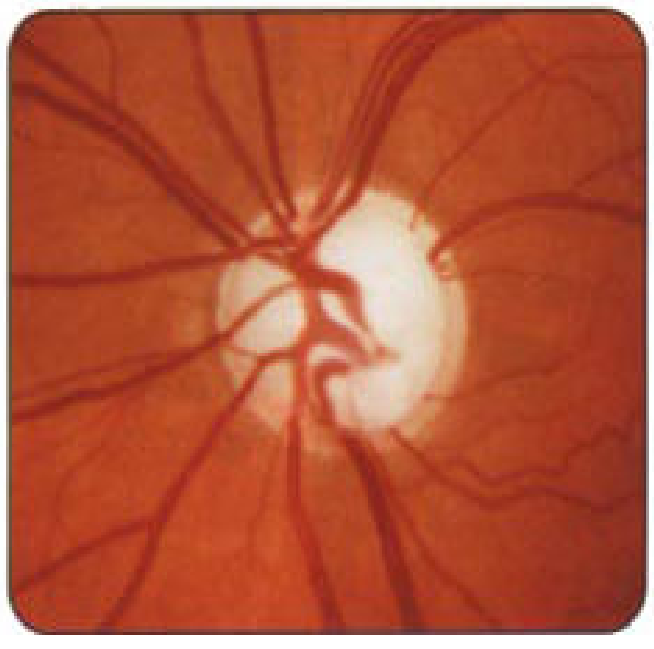
Glaucomatous optic disc



Glaucomatous optic disc



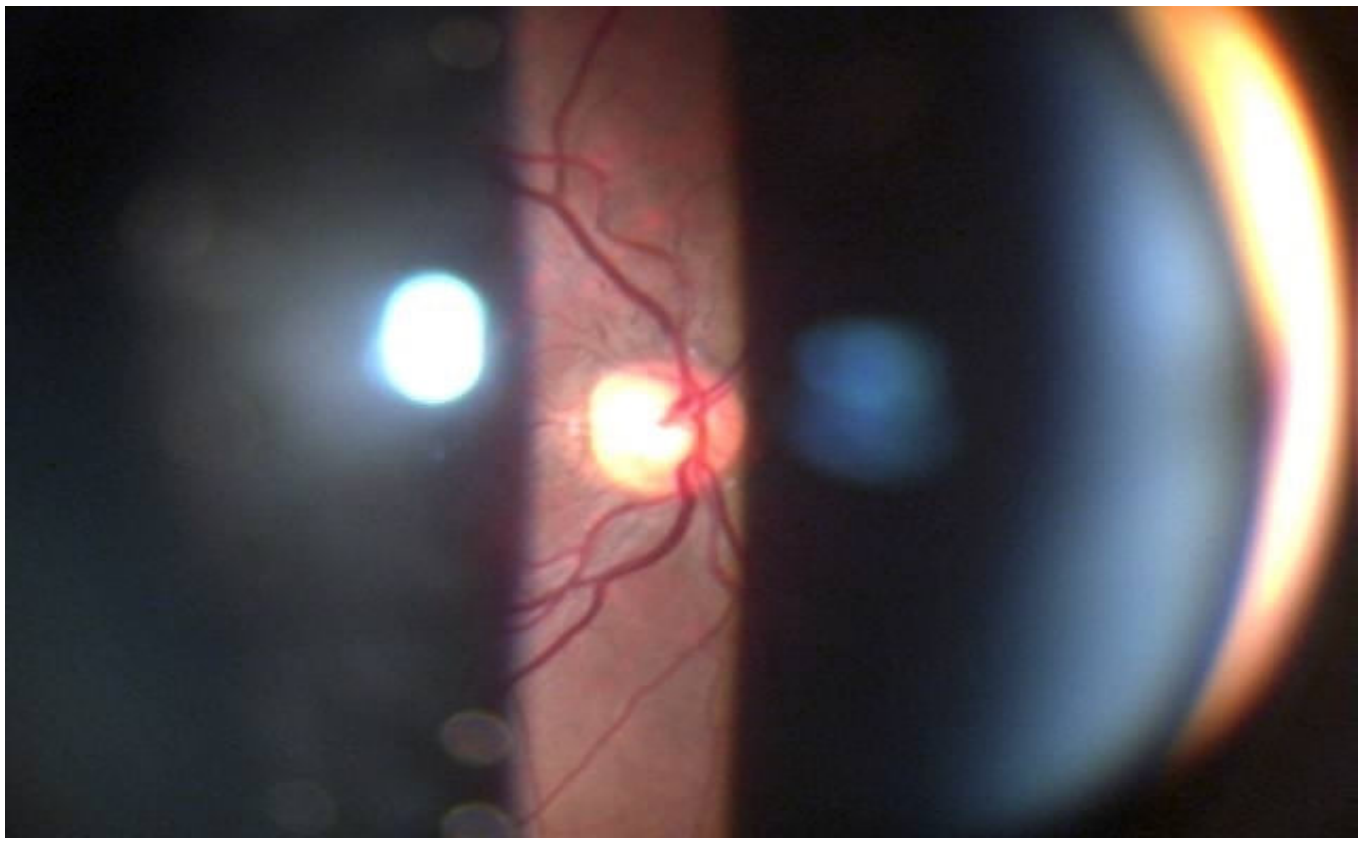
Is this a normal or glaucomatous disc ?



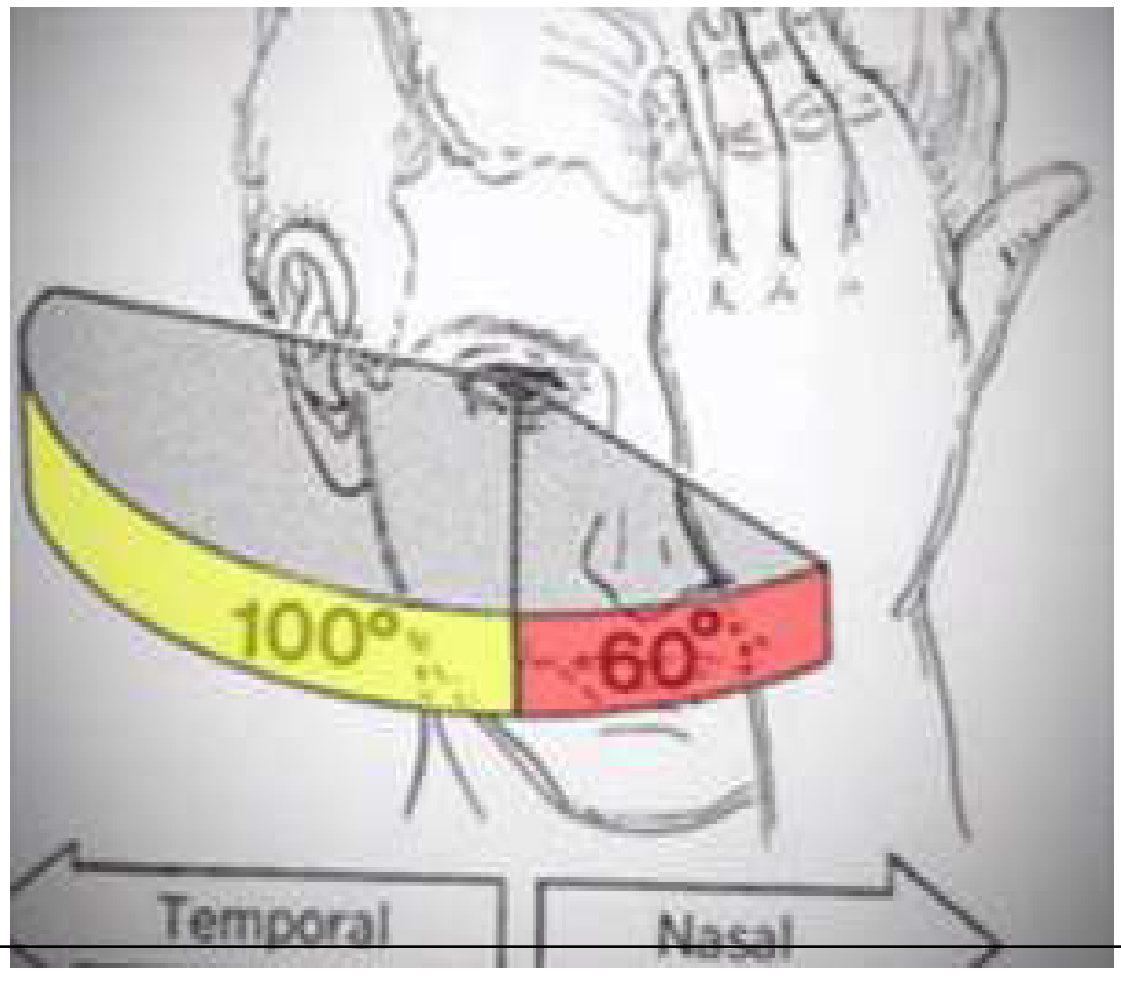
Recording and documenting disc changes

- Serial drawings (10 square grid) after seeing fundus by ophthalmoscopy/slit lamp with +90D/+78D lens
- Disc photography
- HRT(Heidelberg retinal tomography)
- OCT (Optical coherence tomography)
- NFA(Nerve fibre analyser)

View of optic disc by 90D lens examination



Field of vision



Visual field defects in glaucoma

- Arcuate nerve fibres in the superior and inferior temporal portions of the optic disc:

  Most sensitive to damage
- Macular fibres : Most resistant to damage

CENTRAL VISION IS PRESERVED TILL THE LAST IN GLAUCOMA

Progression of field defects

- Isopter contraction: Generalised field constriction
- Baring of blind spot : Non specific

  (Exclusion of blind spot from central field)
- Paracentral scotoma: Wing shaped and occurs above or below the blind spot in the Bjerrum's area(10-25 degrees from fixation)

  Is the earliest clinically significant defect

Progression of field defects

- Seidel's scotoma: sickle shaped
  Due to joining of blind spot and paracentral scotoma
- Bjerrum's/Arcuate scotoma:
  Extension of Seidel's scotoma to reach the horizontal line.
- Double arcuate/ring scotoma

Progression of field defects

- Roenne's central nasal step:
  Sharp right angled defect at the horizontal meridian when arcuate scotomas run in different arcs
- Peripheral field defects
- Advanced defects
  Residual Tubular vision
  Temporal island of vision

Quantification of visual field defects

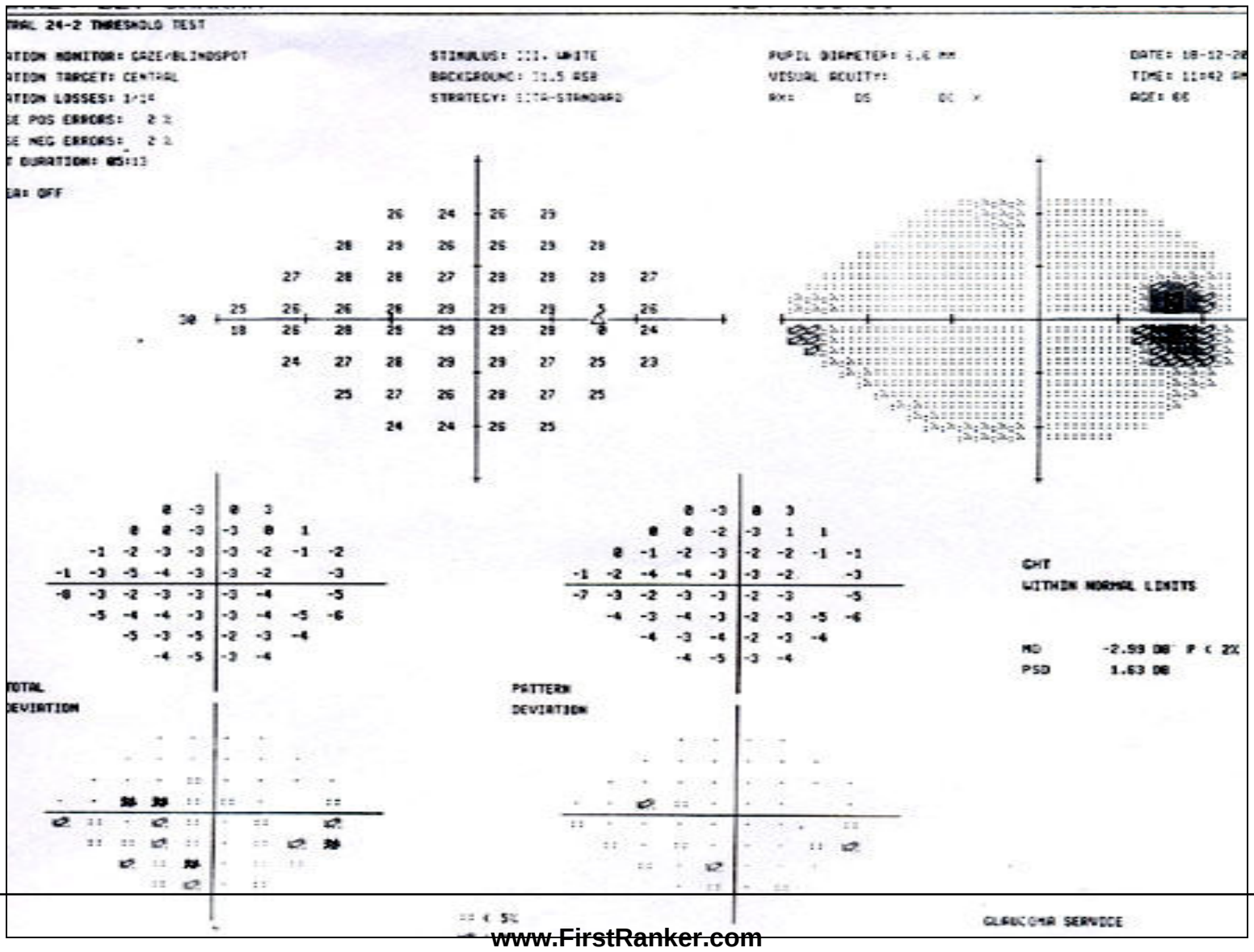
- Visual field analyzer

  Kinetic perimeter

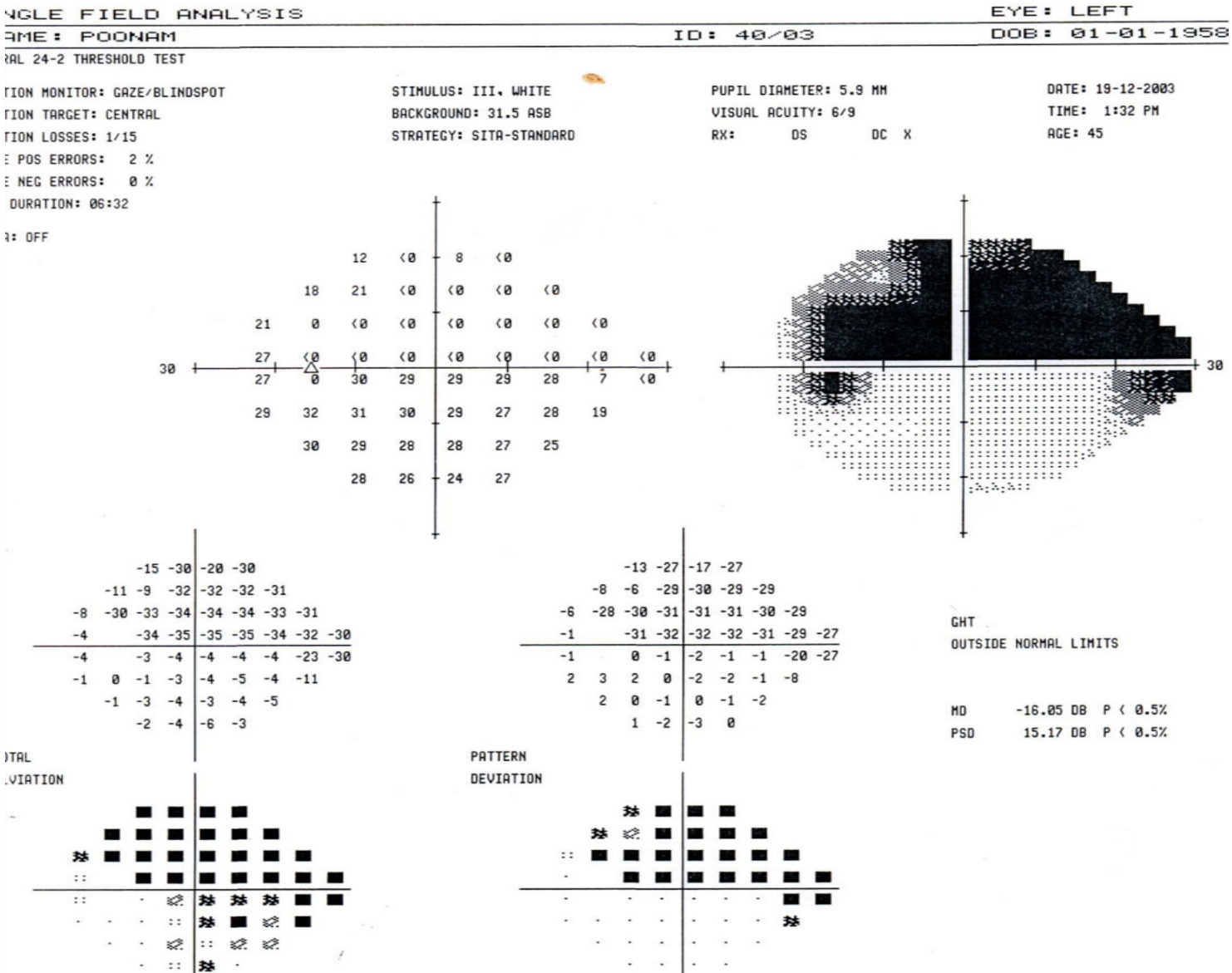
  Static perimeter (automated)

Testing more than once is required before final interpretation

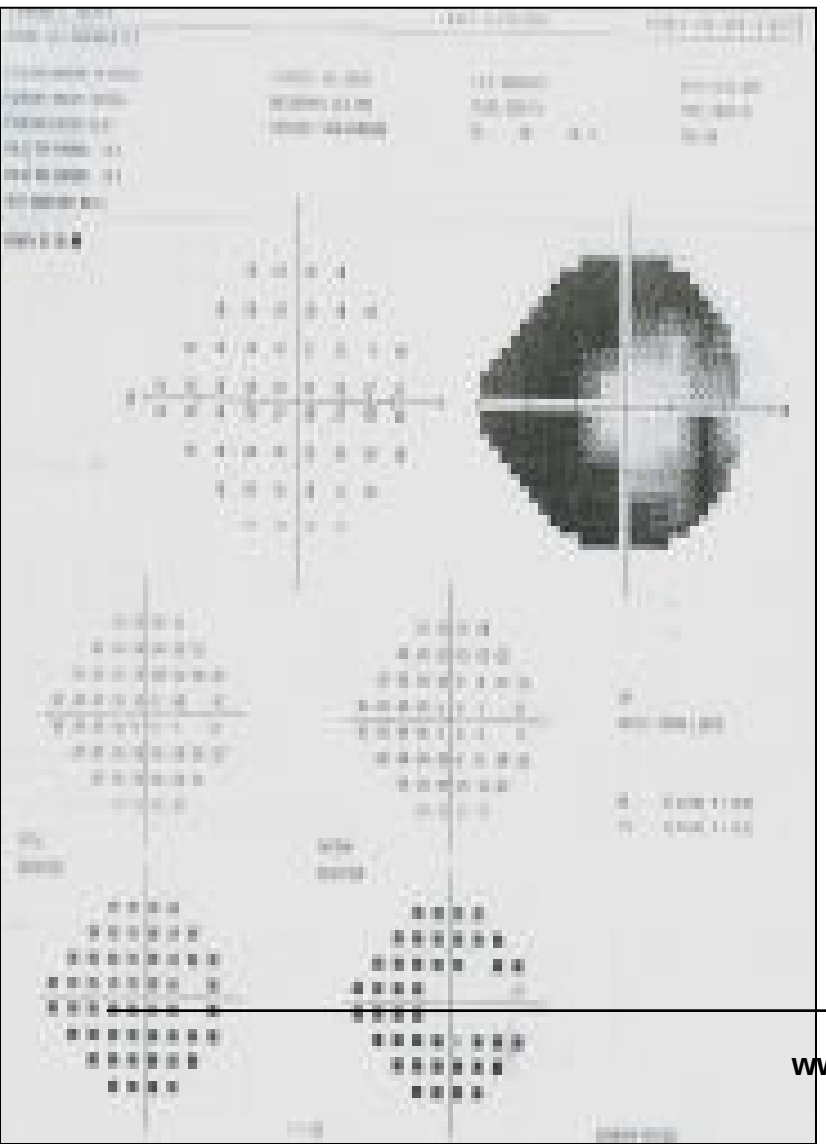
Enlarged blind spot

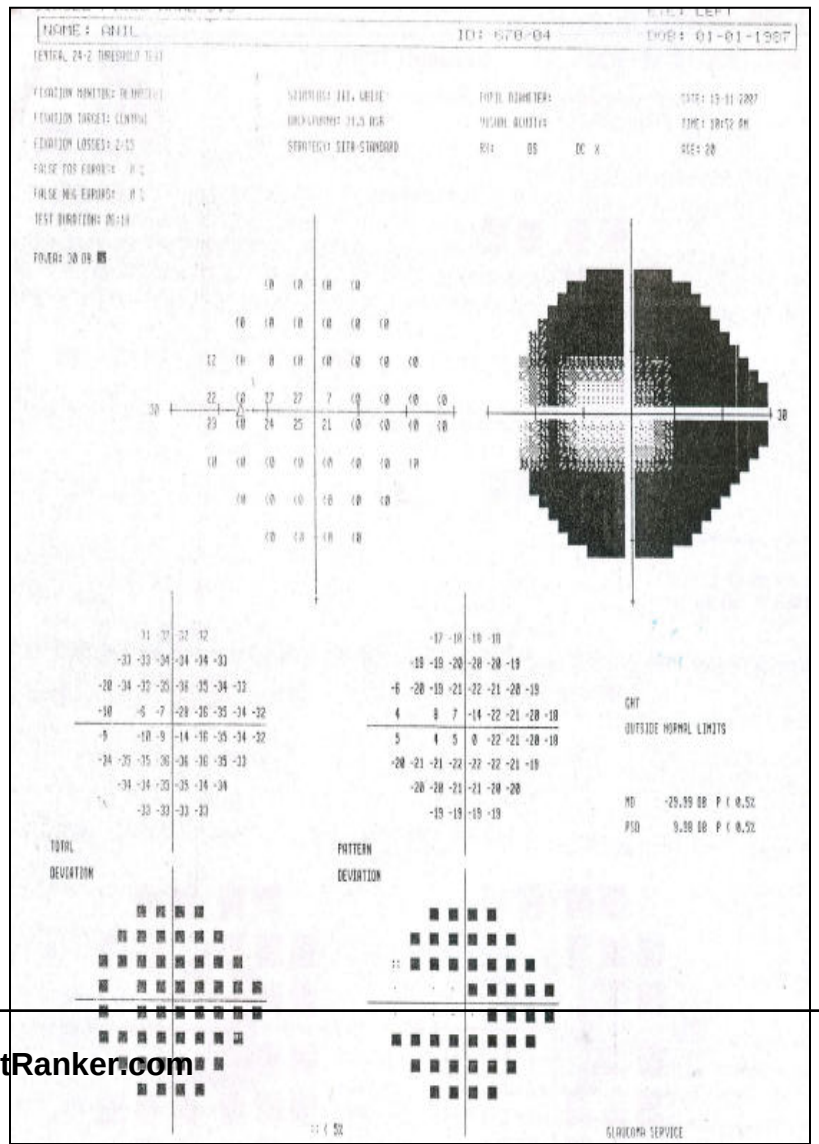


Superior arcuate scotoma

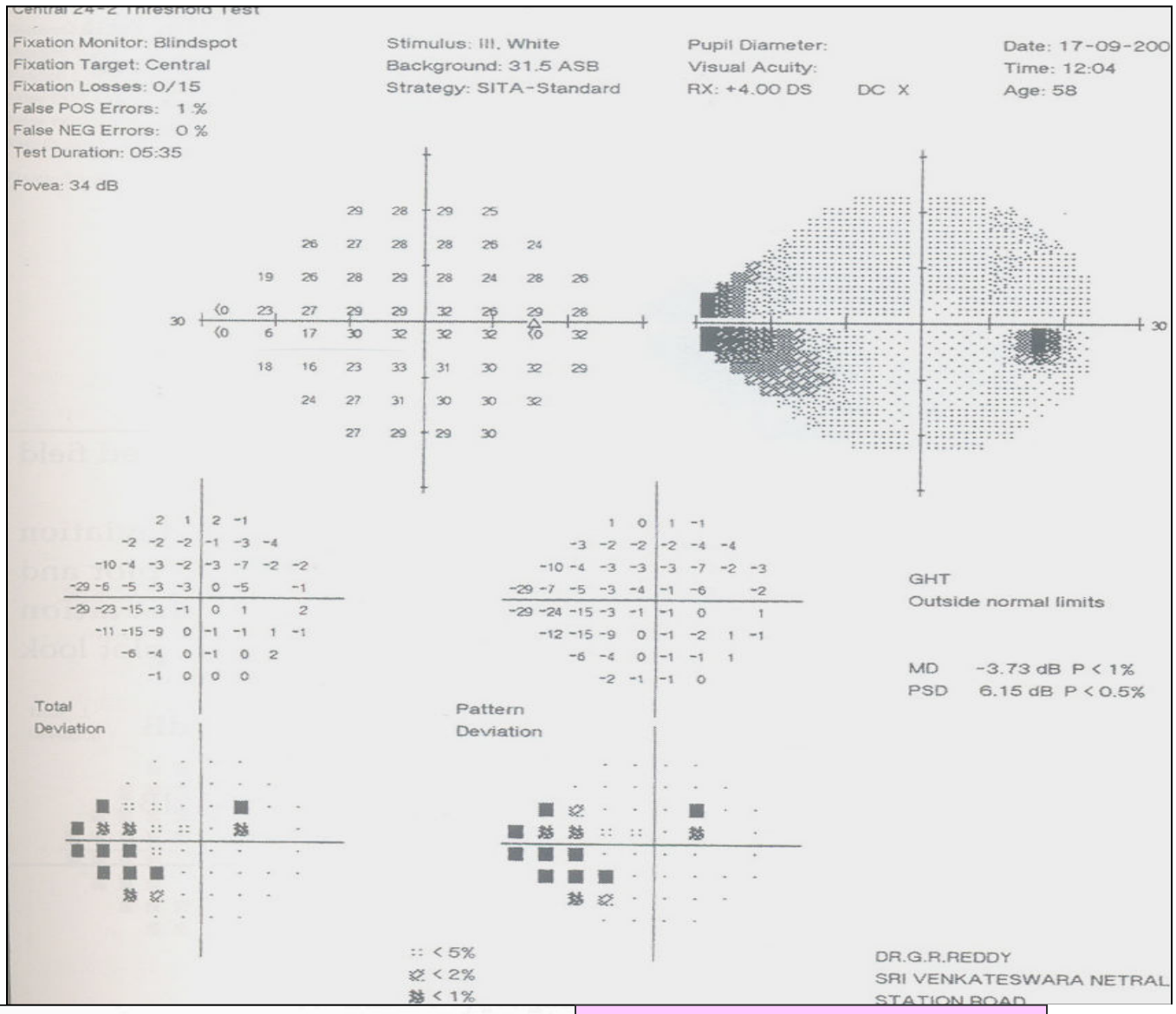


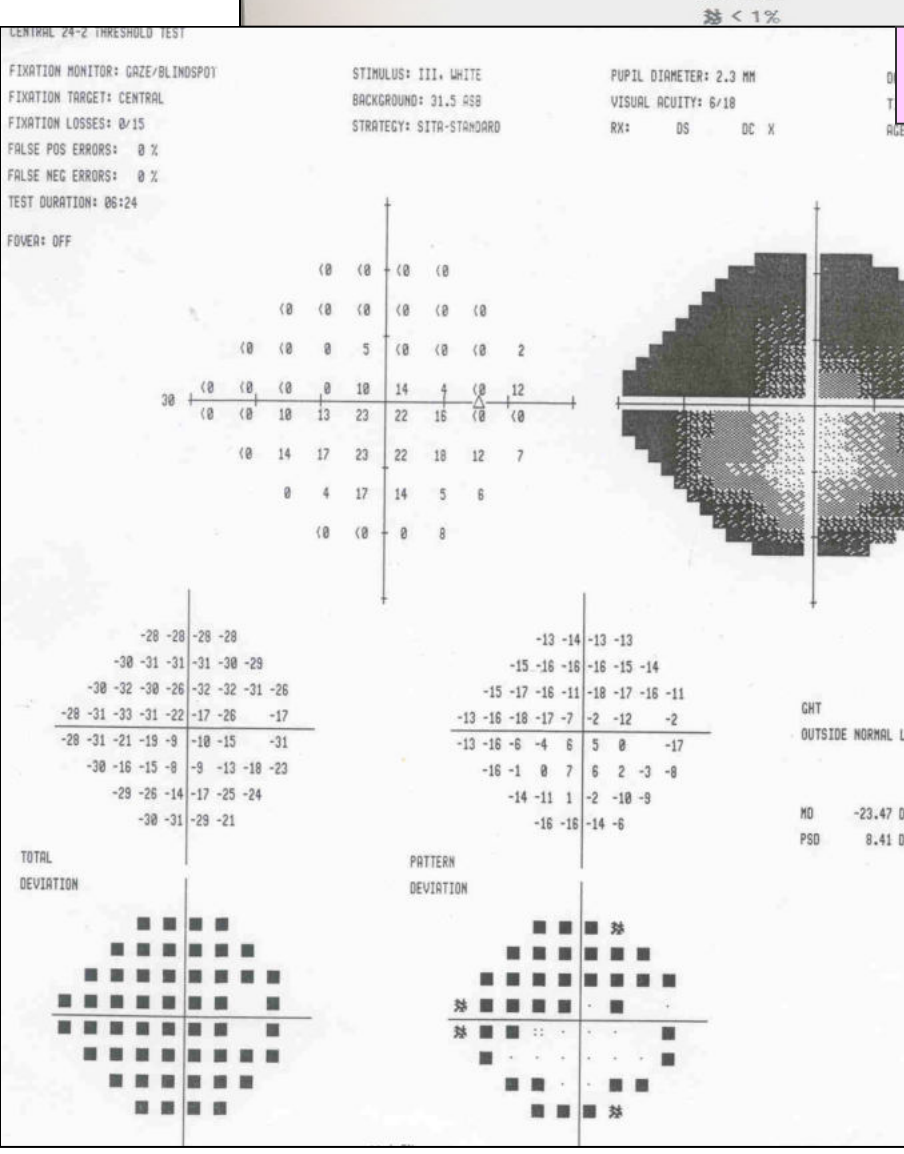
Bjerrum's scotoma



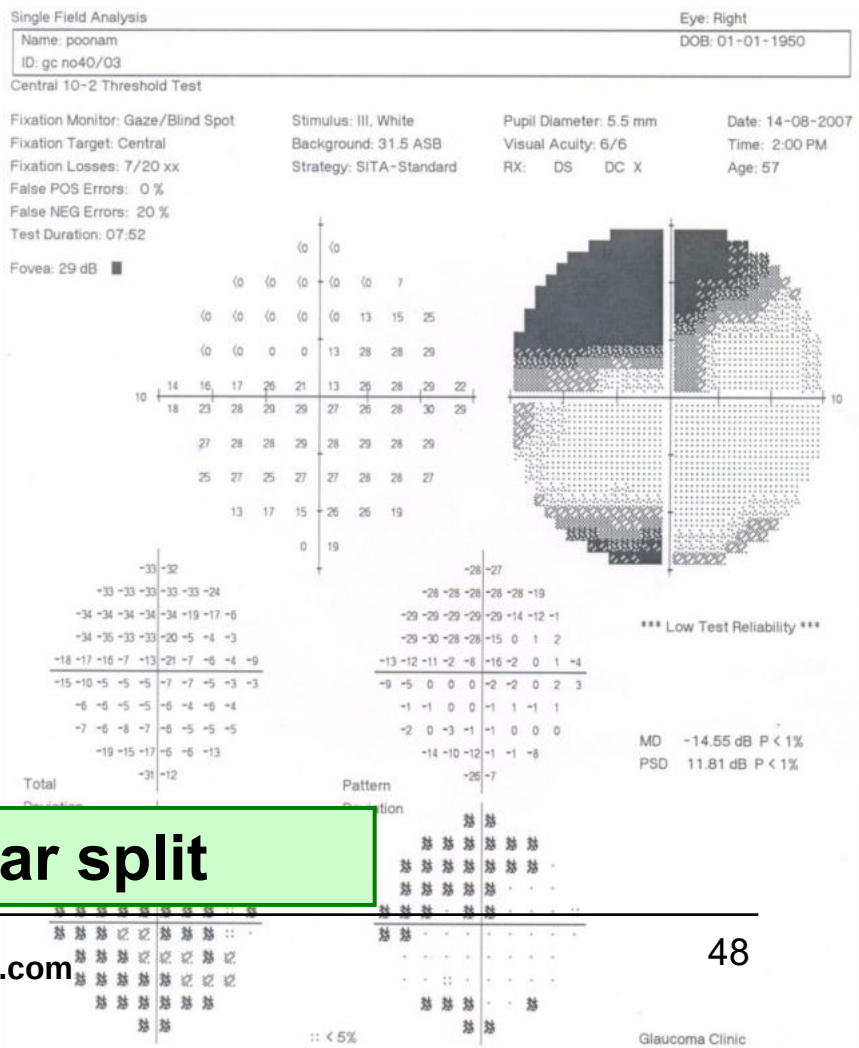


Roenne's nasal step



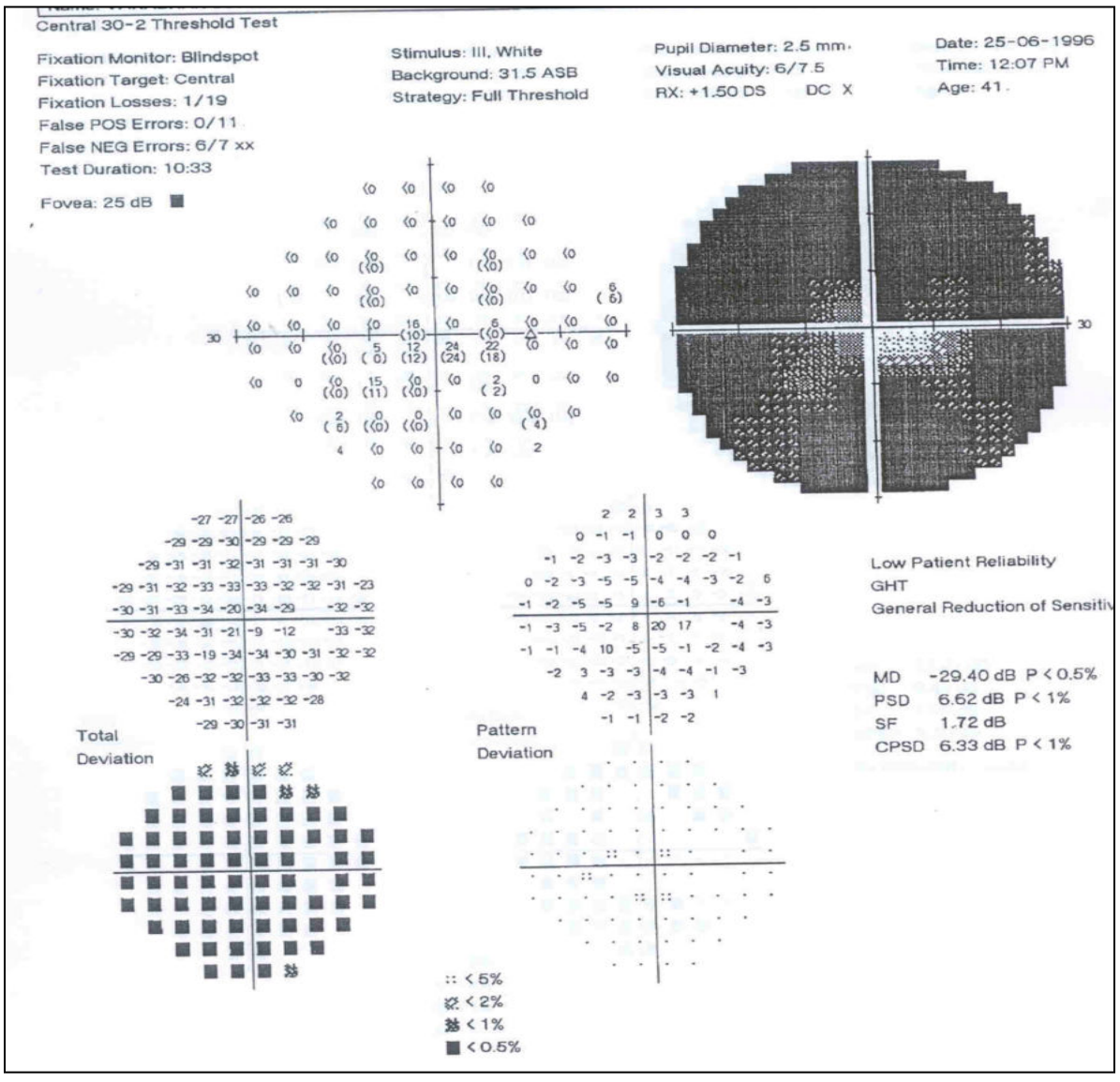


Double arcuate



10-2- Advanced VFD , macular split

Advanced glaucoma



Diagnostic work up/Investigations

- Tonometry
- Goniscopy: Open angles
- Perimetry: To detect visual field defects
- Slit lamp examination: To rule out causes of secondary open angle glaucoma
- Fundus examination to document optic disc changes
- Diurnal variation testing
- Provocative testing: Water drinking test

Diagnosis

- POAG: Raised IOP(More than 21 mm Hg), glaucomatous optic disc cupping, visual field changes.
- Ocular hypertension/glaucoma suspect: Raised IOP
- NTG(Normal tension glaucoma): Glaucomatous optic disc cupping with or without visual field changes with normal IOP

Management of POAG

- Therapeutic choices

❑ Medical therapy

❑ Argon/Diode Laser Trabeculoplasty

❑ Filtration surgery

Basic principles of therapy

- Make a correct diagnosis
- Set a target IOP
- Start with a single drug to lower IOP
- Switch to another group of drugs if needed
- Control IOP on minimal medications
- Monitor therapy and reset target IOP whenever needed

Topical drugs used for POAG therapy

- Prostaglandin/Prostamides

  Latanoprost, Bimatoprost, Travoprost
- Beta blockers

  Timolol maleate, Betaxolol
- Carbonic anhydrase inhibitors

  Dorzolamide, Brinzolamide
- Sympathomimetics

  Brimonidine, Apraclonidine
- Parasympathomimetics

  Pilocarpine

Systemic drugs used for POAG therapy

- Used rarely, for short term control of IOP
- Oral carbonic anhydrase inhibitors
  Acetazolamide, Methazolamide

Laser treatment

- Indications
  Target IOP not achieved with medical therapy
  Non compliance of medical therapy

Argon/ Diode Laser Trabeculoplasty (ALT)
Selective Laser Trabeculoplasty (SLT)

Surgical therapy

- Indications

❖ Target IOP not achieved with maximal tolerated medical therapy and laser trabeculoplasty

❖ Non compliance of medical therapy

❖ Non availability of laser therapy

❖ Advanced glaucoma

Surgical therapy

- Filtration surgery : Trabeculectomy
- Modified trabeculectomy :

  Use of antifibrotic agents

  Mitomycin/5FU
- Aqueous drainage devices:

  Ahmed glaucoma valve

In cases with no/poor visual potential:

Cycloablative therapy with laser/cryotherapy

Conclusion

- Primary open angle glaucoma is a progressive optic neuropathy with characteristic optic disc and visual field changes.
- Increased resistance to aqueous outflow leads to rise in IOP.
- Aim of management is to reduce IOP to minimize damage to optic disc and resultant visual field defects.

Question

Which of the following is not a risk factor for the development of primary open angle glaucoma?

a. positive family history.

b. advanced age.

c. increased IOP.

d. increased corneal thickness.

Question

You have been referred a case of open angle glaucoma. Which of the following would be an important point in diagnosing the case?

a. Shallow anterior chamber

b. Optic disc cupping

c. Narrow angle

d. Visual acuity and refractive error

THANK YOU