GLAUCOMA Part 2

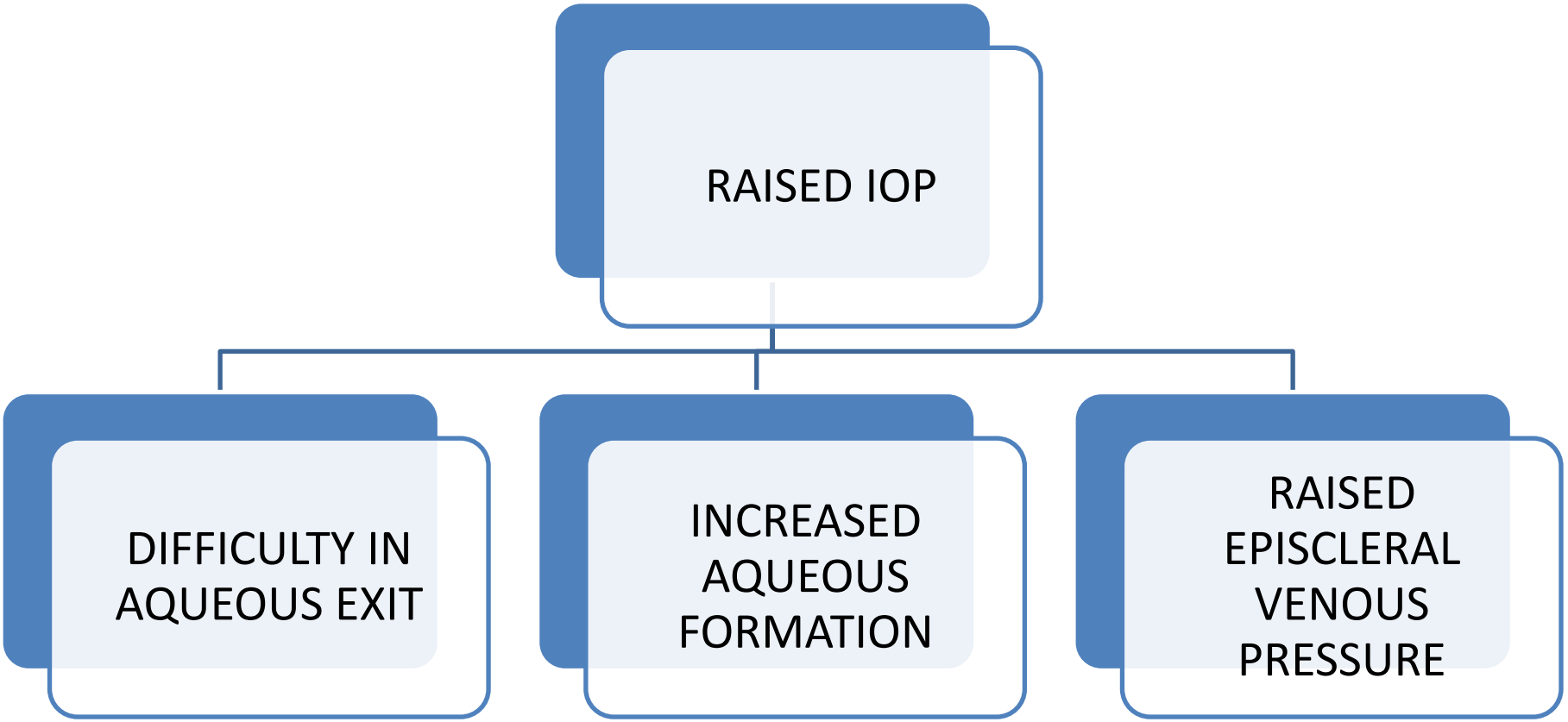
Topics covered

- Definition of glaucoma
- Classification of glaucoma
- Congenital glaucoma
- Primary open angle glaucoma

DEFINITION

Glaucoma is a chronic, progressive optic neuropathy caused by a group of ocular conditions which lead to damage of the optic nerve with loss of visual function.

Most common risk factor- Raised IOP



Factors to be considered in classification of Glaucoma

1. Congenital/Acquired (acc to time of onset)
2. Acute/Chronic (acc to duration)
3. Primary/Secondary (acc to associations)
4. Open angle/Close angle (acc to gonioscopy)

Classification

Clinico-etiological classification

(A) Congenital/ Developmental Glaucomas

1. Primary Congenital Glaucoma – without associated anomalies
2. Developmental Glaucoma (with associated anomalies)

(B) Primary Adult Glaucomas

1. Primary Open Angle Glaucoma (POAG)
2. Primary Angle Closure Glaucoma (PACG)
3. Primary Mixed mechanism Glaucoma

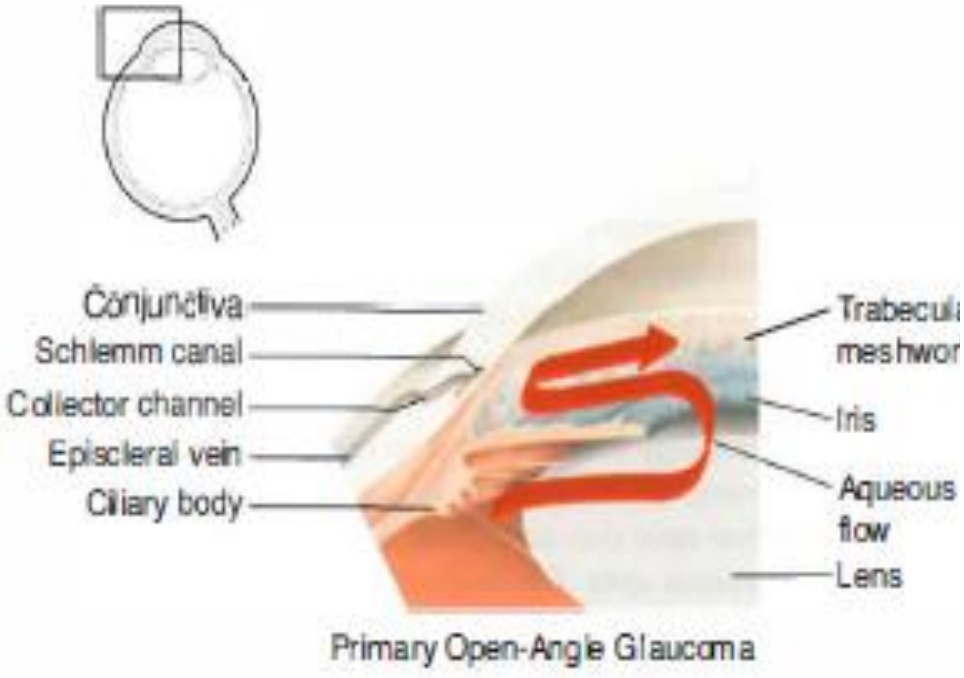
(C) Secondary Glaucomas

1. Secondary Open Angle Glaucoma
2. Secondary Angle Closure Glaucoma

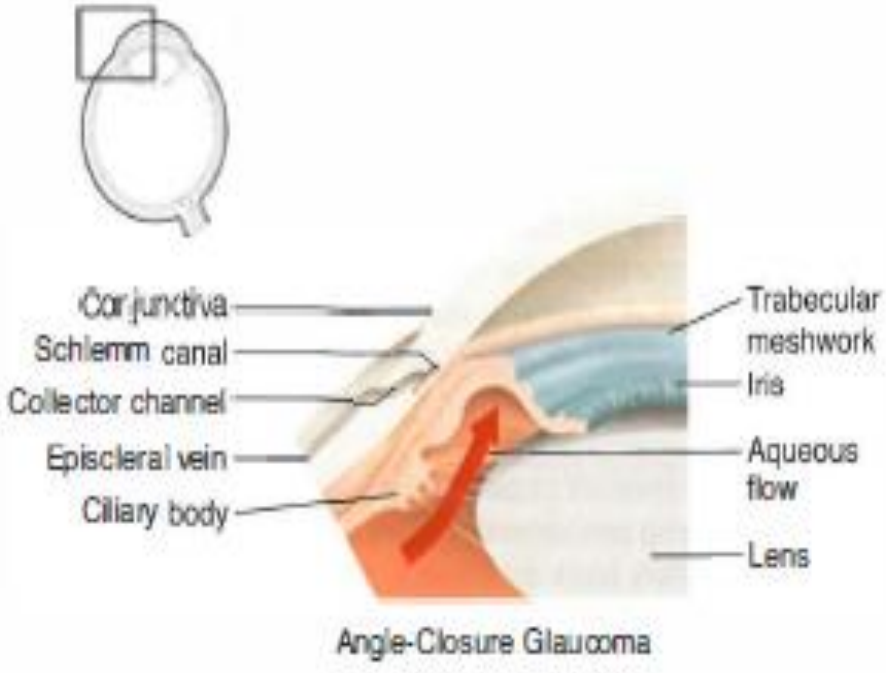
Classification of the Glaucomas

- Open angle
- Angle closure

- Primary
- Secondary



Primary Open-Angle Glaucoma



Angle-Closure Glaucoma

Open-angle glaucomas

Primary open-angle glaucoma (POAG)	**Not associated with known ocular or systemic disorders that cause increased resistance to aqueous outflow or damage to optic nerve; usually associated with elevated I O P**
Normal-tension glaucoma (NTG)	Considered in continuum of POAG; when I O P is not elevated but glaucomatous optic disc and visual field defects
Juvenile open-angle glaucoma (JOAG)	Terminology often used when open-angle glaucoma diagnosed at young age (typically 4-35 years of age)

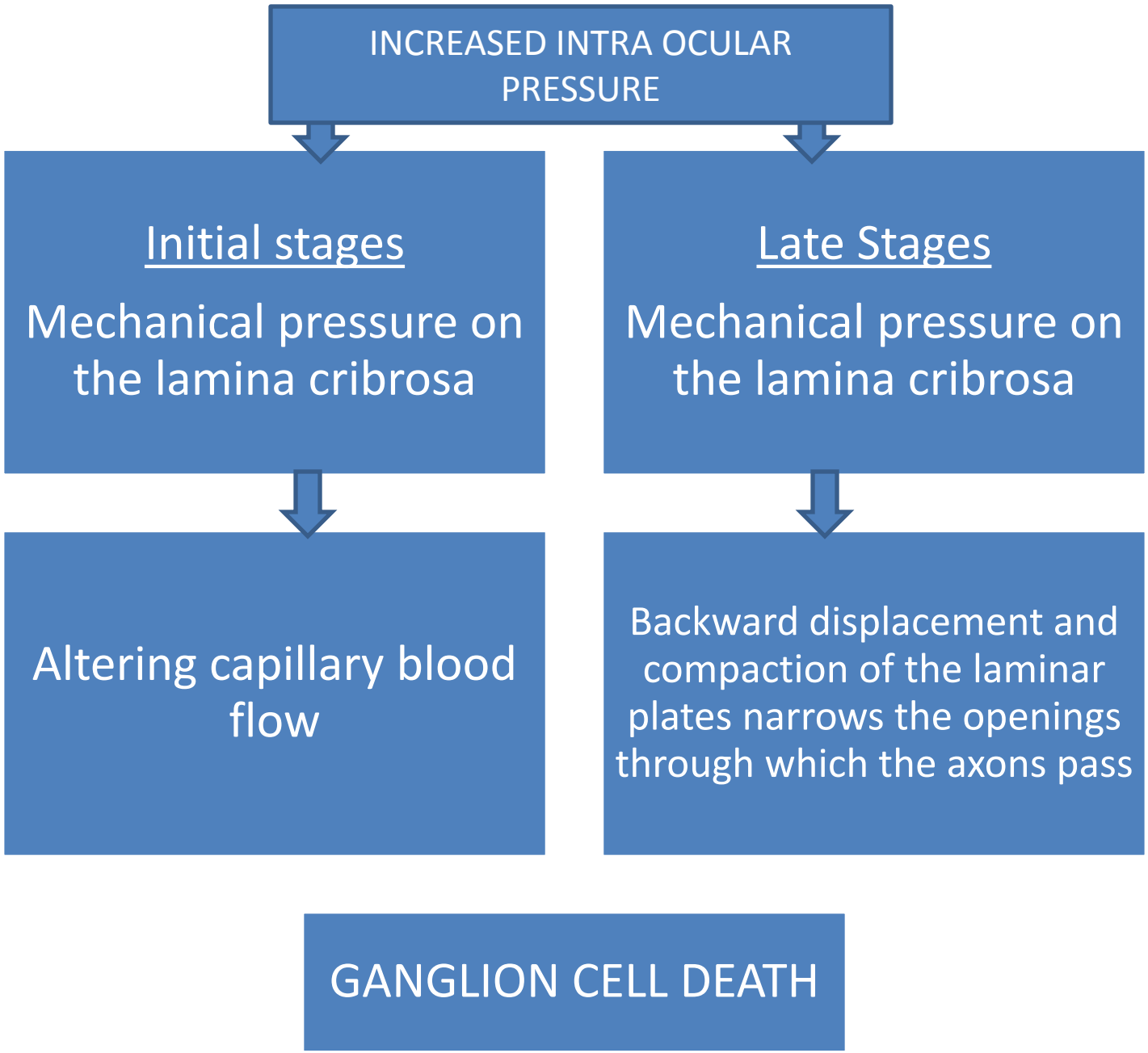
Ocular hypertension	Normal optic disc and visual field associated with elevated I O P
Glaucoma suspect	Suspicious optic disc or visual field regardless of I O P
Secondary open angle glaucoma	Increased resistance to trabecular meshwork outflow associated with other conditions (eg, pigmentary glaucoma, phacolytic glaucoma, steroid-induced glaucoma, exfoliation syndrome, angle-recession glaucoma) Increased post-trabecular resistance to outflow secondary to elevated episcleral venous pressure (eg, carotid cavernous sinus fistula)

PATHOGENESIS

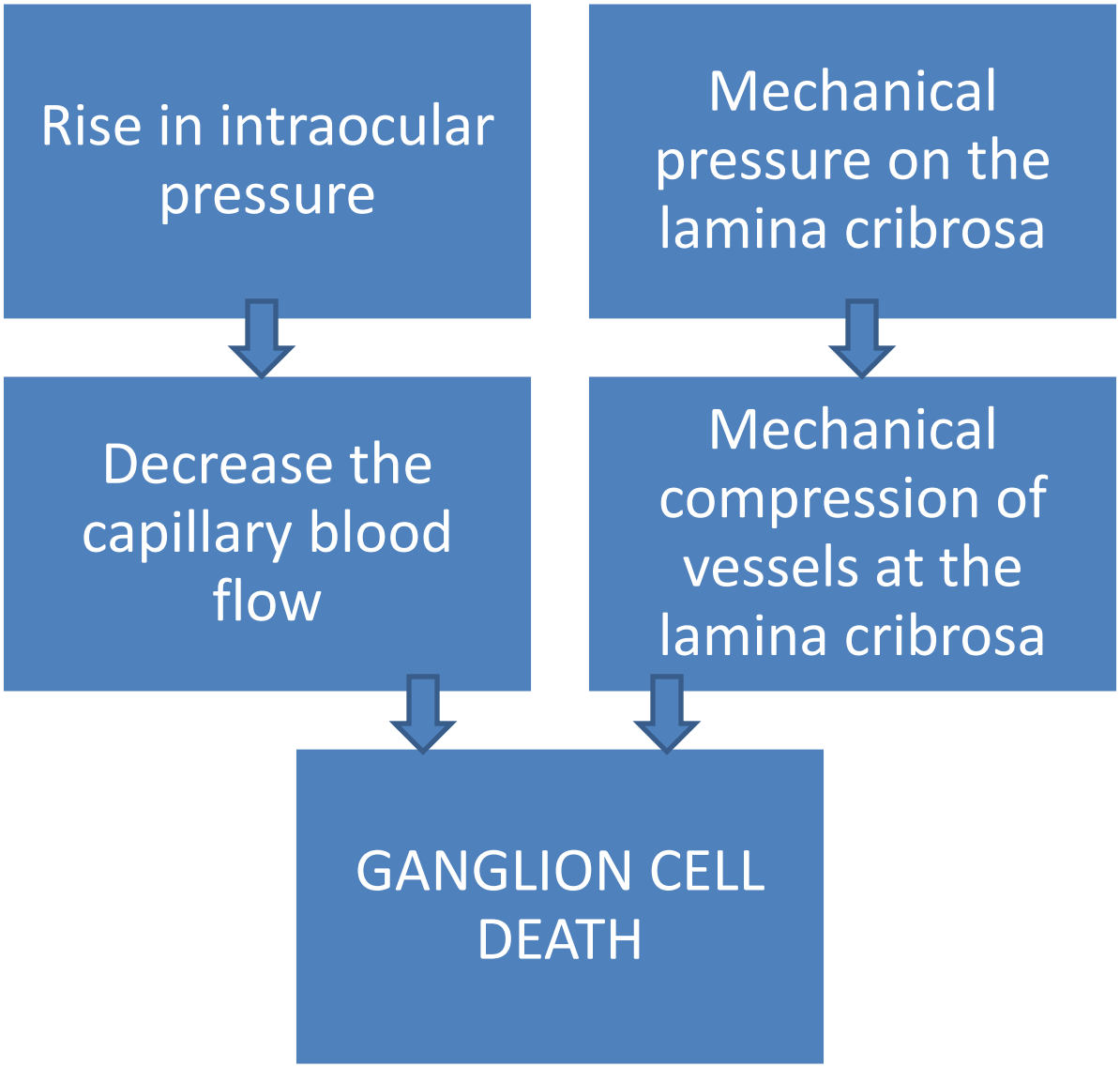
- MECHANICAL changes due to the rise of intraocular pressure and

- VASCULAR perfusion of the optic nerve head.

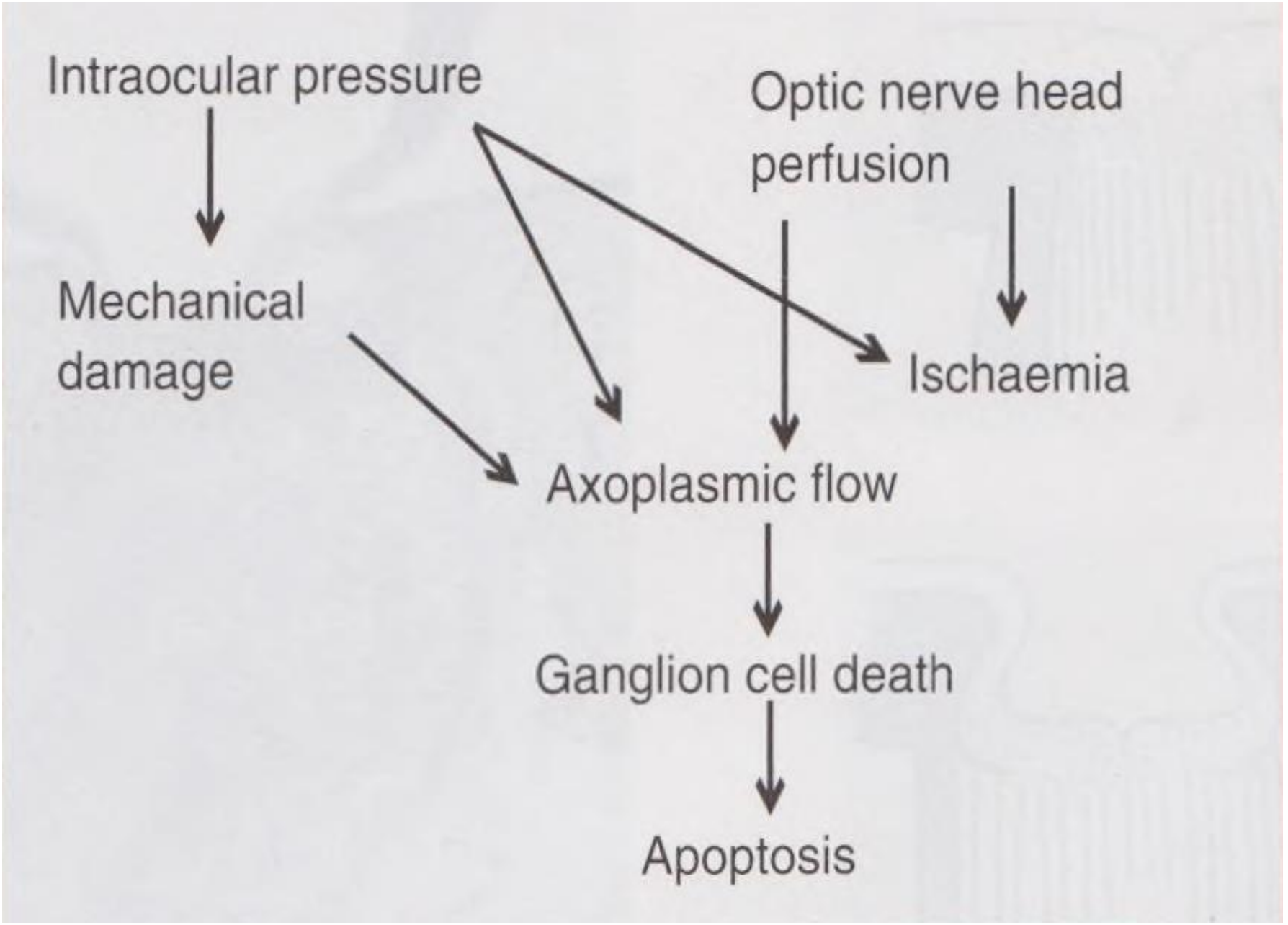
MECHANICAL


INCREASED INTRA OCULAR PRESSURE
Initial stages
Mechanical pressure on the lamina cribrosa
Late Stages
Mechanical pressure on the lamina cribrosa
Altering capillary blood flow
Backward displacement and compaction of the laminar plates narrows the openings through which the axons pass
GANGLION CELL DEATH

VASCULAR


Rise in intraocular pressure
Mechanical pressure on the lamina cribrosa
Decrease the capillary blood flow
Mechanical compression of vessels at the lamina cribrosa
GANGLION CELL DEATH

PATHOGENESIS


Intraocular pressure
Optic nerve head perfusion
Mechanical damage
Ischaemia
Axoplasmic flow
Ganglion cell death
Apoptosis

Diagnosis

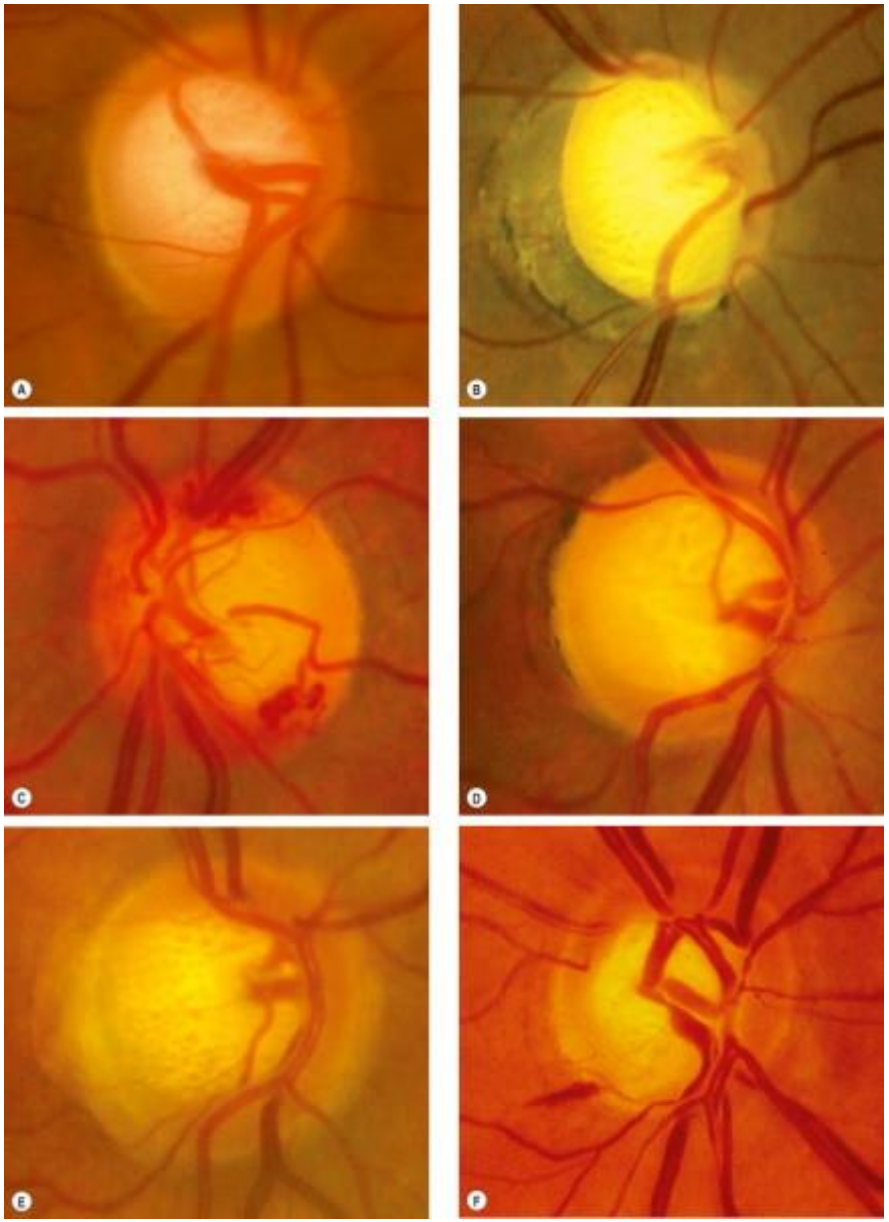
Combination of clinical signs—characteristic changes in

- (A) The optic nerve head
- (B) Abnormalities in the visual field
- (C) Rise in intraocular pressure

- The type of glaucoma is determined by the status of the anterior chamber angle as determined by Gonioscopy

(A)Optic nerve head changes

- Asymmetry of CDR >0.2
- A localized notch or thinning of NRR.
- Enlarged CDR >0.5 in vertical axis
- Superficial disc hemorrhages
- Shift of vessels to nasal side
- Bayonetting
- Peripapillary atrophy (β&α Zones)
- Lamellar-dot sign



Non-specific signs of glaucomatous damage. **(A)** Inferior baring of circumlinear blood vessels; **(B)** inferior bayoneting; **(C)** collaterals; **(D)** loss of nasal neuroretinal rim; **(E)** lamellar dots; **(F)** disc haemorrhage

(B) Glaucomatous Visual field abnormalities

- Initially observed in Bjerrum area, 10- 25° from fixation
- Correlate with abnormalities seen on optic nerve head
- Field defects:
    1. Paracentral scotomas
    2. Nasal step (Earliest)
    3. Siedel scotoma
    4. Arcuate scotoma
    5. Double arcuate or ring scotoma
    6. End-stage or near total defect with only a residual temporal island of vision

(C) Intraocular Pressure

- IOP > 21mmHg on more than one occasion
- Circadian Variation > 8mmHg
- Asymmetry in IOP between 2 eyes of more than 5 mmHg

PRIMARY OPEN ANGLE GLAUCOMA (POAG)

- K/a Chronic simple glaucoma
- Most prevalent of all glaucoma
- Affects both sexes equally

POAG

CLINICAL FEATURES

- An IOP >21 mmHg
- Glaucomatous optic nerve damage
- An open normal appearing anterior chamber angle
- Characteristic visual field loss
- Absence of signs of secondary glaucoma or a non-glaucomatous cause for the optic neuropathy

GENETICS

- Mutations at 15 loci in the human genome..***GLC1A to GLC1O***
- 4 susceptible genes have been identified
  - **MYOC gene** (chromosome 1q21-q31), coding for the glycoprotein myocilin that is found in the trabecular meshwork and other ocular tissues
  - **OPTN gene** on chromosome 10p, which codes for optineurin
  - **WDR36 gene** on chromosome 5q22
  - **NTF4 gene** on chromosome 19q13.3.

➢ ***Among them MYOC is the most frequently mutated gene in POAG***

RISK FACTORS

- IOP
- Age
- Race
- Family history
- Diabetes Mellitus
- Myopia
- Vascular disease

Clinical features

- Usually asymptomatic until a significant visual field loss has occurred
- Eye ache, headache, colored haloes
- Delayed dark adaptation time
- Frequent changes of presbyopic (near) glasses
- Raised IOP & fluctuations in IOP
- Visual field changes consistent with glaucomatous optic nerve defects
- Defects in the nerve fibre layer, extending from the optic nerve head in an arc from
  the superior and inferior poles of the disc.
  Best appreciated with a red-free light in dilated fundus examination

DIAGNOSIS

- **Primary open angle glaucoma (POAG)**
  - IOP(>21 mm of Hg) associated with definite glaucomatous optic disc cupping and visual field changes
- **Ocular hypertension or glaucoma suspect**
  - IOP constantly more than 21 mm of Hg but no optic disc or visual field changes
- **Normal tension glaucoma (NTG)**
  - Typical glaucomatous disc cupping with or without visual field changes is associated with an intraocular pressure constantly below 21 mm of Hg

MANAGEMENT

- Optic Nerve damage is irreversible
- Early detection is required
- Glaucoma screening >40 yrs of age specially for those with family history, diabetes, myopia
- Good baseline information and close follow-up throughout life

Base line information

- History: Ocular, Systemic, Family history, History of medication
- Pupillary reaction
- Slit lamp bio-microscopy:

✓ Anterior segment to rule out 2° causes- shallow anterior chamber, pseudoexfoliation syndrome, inflammation

✓ Fundus evaluation to rule out lesions which can cause visual field defects

✓ Applanation tonometry and Diurnal variation test

- CCT > 520µm: false high IOP

  < 520µm: false low IOP

- Gonioscopy

- Perimetry: Automated static threshold perimetry

- Provocative Tests: Water drinking test

TREATMENT

- MEDICAL MANAGEMENT
- LASER
- SURGICAL

Principle of treatment

- Usually start with MEDICAL THERAPY.
- Before starting the treatment - Assess each eye individually, inform patients
- Start treatment in worse eye first
- Set TARGET PRESSURE

Target IOP depends upon

- IOP at which damage has occurred
- Severity of Visual Field damage
- Rate of progression of damage
- Age and Life Expectancy

TARGET IOP

- Level at which damage doesn't develop or already existing damage doesn't progress
- Progression may be slow & it may take 3-5 yrs to find a safe IOP level for an individual

- Target IOP must be updated during follow ups by monitoring progression of structural & visual field abnormalities

- Target IOP must be </=25% of the untreated level

- Target IOP should be lower especially if the patient has
  - Advanced glaucoma
  - Several Risk factors
  - Long life expectancy
  - Aggressive glaucoma
- Central corneal thickness(CCT): Patients with thin corneas having been identified as a major risk factor for patients with ocular hypertension

- In most cases, reducing the IOP by 20% to 30% from baseline is recommended
- Target IOP in
  - **Middle to high teens** (mm Hg) for eyes with minimal damage (e.g., early neural rim thinning without visual field loss),
  - **Low to middle teens for eyes with moderate damage** (e.g., cupping to the disc margin in one quadrant with early field loss)
  - **High single digits to low teens for eyes with advanced damage** (e.g., extensive cupping and field loss).

ANTI-GLAUCOMA DRUGS

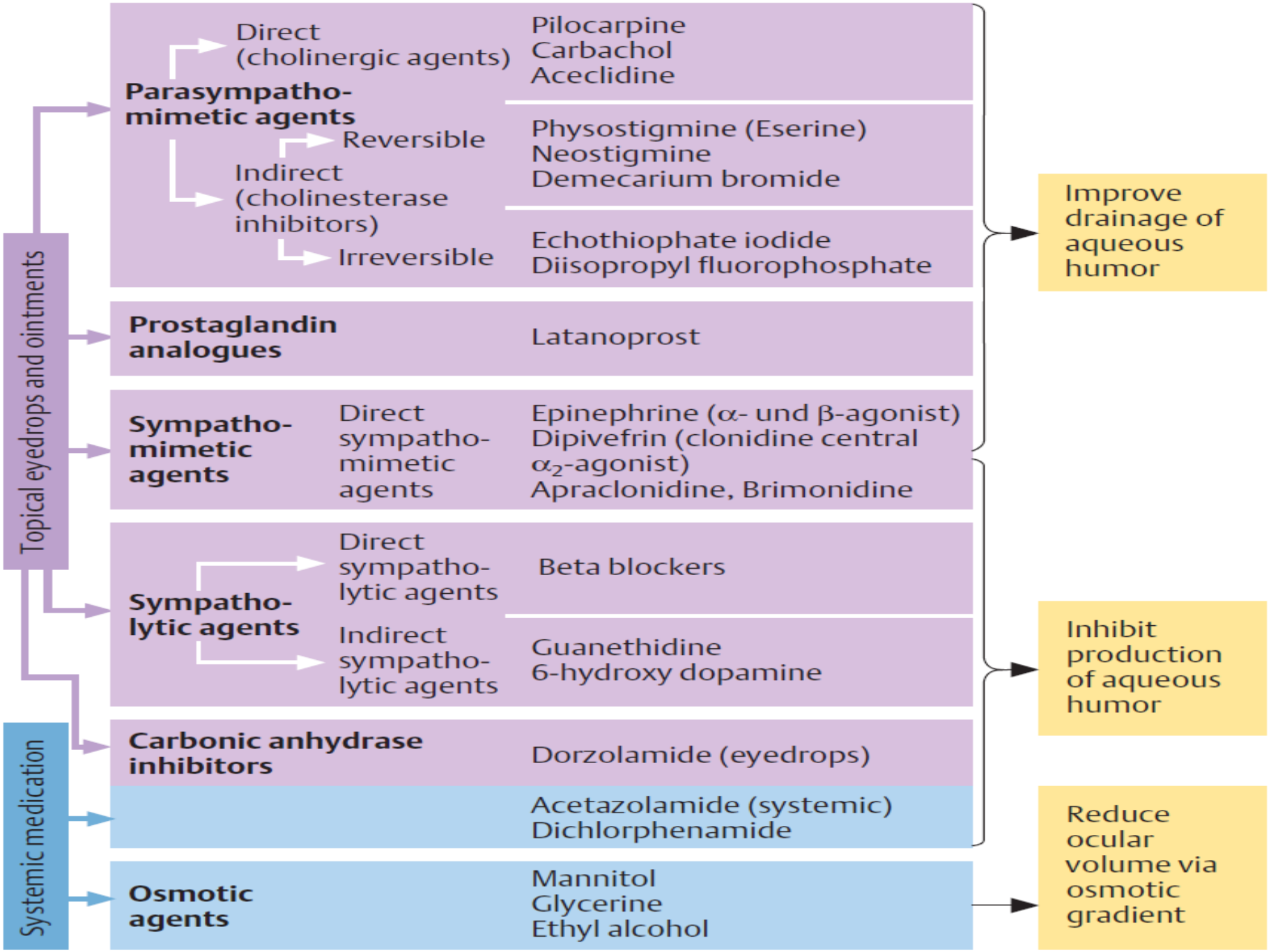
Classification

- A. Parasympathomimetic drugs (Miotics)
- B. Sympathomimetic drugs (Adrenergic agonists)
- C. β-blockers
- D. Carbonic anhydrase inhibitors
- E. Hyperosmotic agents
- F. Prostaglandins
- G. Calcium channel blockers

Classification

- **Drugs decreasing AQUEOUS PRODUCTION**
  - Beta-blockers
  - Alpha-2-agonists
  - Carbonic Anhydrase Inhibitors
- **Drugs increasing TRABECULAR OUTFLOW**
  - Parasympathomimetics
  - Non selective agonists
  - Prostamides
- **Drugs increasing UVEOSCLERAL OUTFLOW**
  - Alpha-2-agonists
  - Prostaglandin Analogues & Prostamides

Options in medical treatment of glaucoma.



Management guidelines for POAG

FIRST LINE

PROSTAGLANDIN ANALOGUES

BETA BLOCKERS

SECOND LINE

PROSTAGLANDIN ANALOGUES

ALPHA AGONISTS

TOPICAL CARBONIC ANHYDRASE INHIBITORS

THIRD LINE

ALPHA AGONISTS

SYSTEMIC CARBONIC ANHYDRASE INHIBITORS

PILOCARPINE

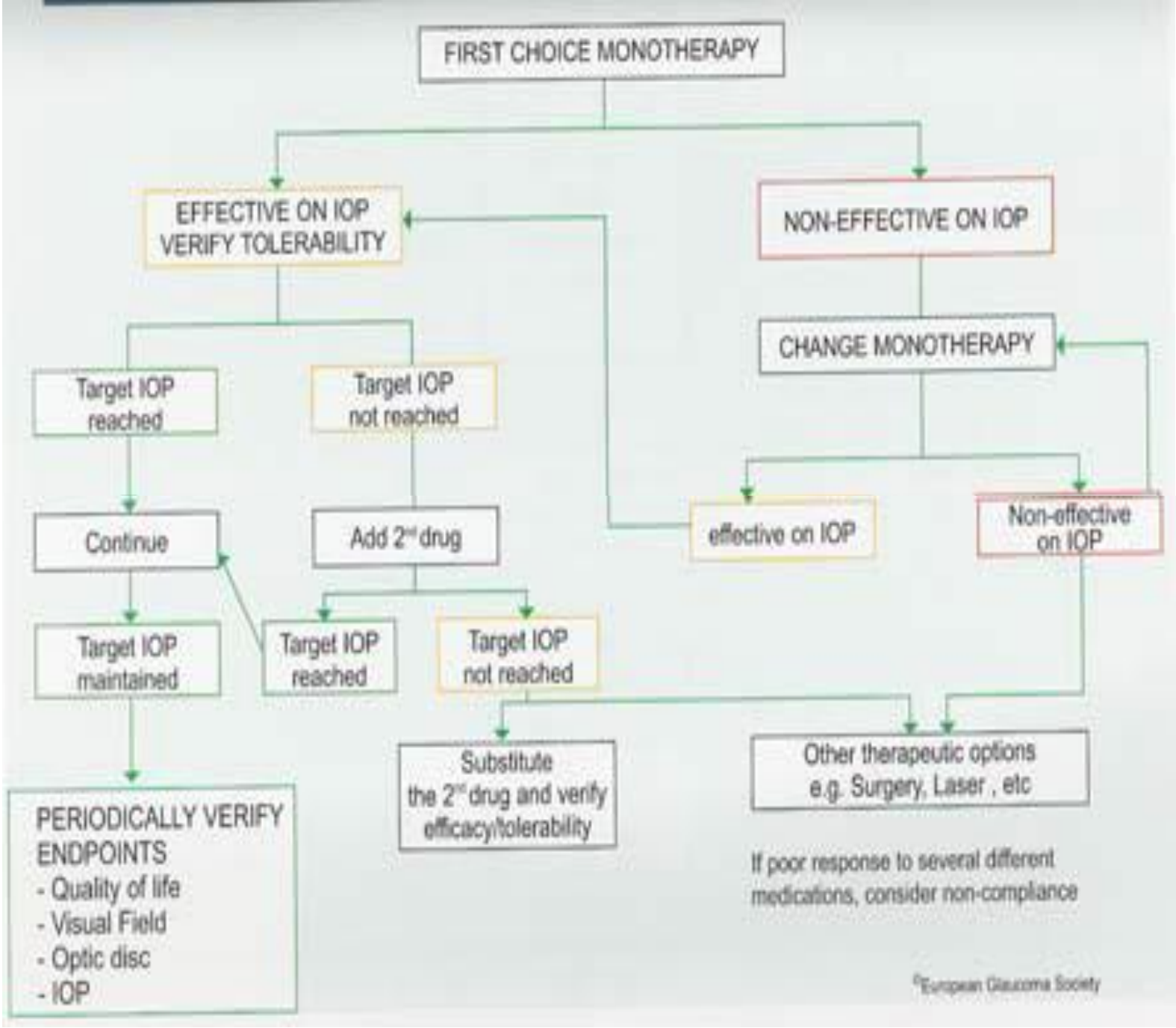
TABLE 10-24-1 -- DRUGS USED TO MANAGE GLAUCOMA

Drug	Example	Mechanism of Action	Efficacy	Side Effects
β-Blockers				
Nonselective	Timolol Levobunolol Carteolol Metipranolol	Decreased aqueous production (? Daytime only)	+ + +	Pulmonary - bronchoconstriction Cardiovascular - bradycardia/heart block Exacerbation of CCF Depression Impotent Death
Selective	Betaxolol		+ +	Impotent Death
Adrenergic agents	Epinephrine Dipivefrin	Outflow enhancement	+ (+)	External eye - toxic reaction
α-Adrenergic agents	Apraclonidine	Decreased aqueous production	+ + (+)	External eye - toxic reaction
	Brimonidine	Uveoscleral outflow increase with brimonidine		Lethargy Dry mouth

Miotics	Carbachol Pilocarpine Echothiophate	Increased tonographic outflow	+ + +	Eye ache Headache Dim vision
Carbonic anhydrase inhibitors				
Oral	Acetazolamide	Decreased aqueous production	+ + + +	Malaise
	Methazolamide			Depression Weight loss Kidney stones
Topical	Dorzolamide		+ +	Risk of rare aplastic anemia - never reported
				Metallic taste
				Eye irritation
Lipids (prostaglandin analogs, prostamides decosanoids)	Latanoprost Travoprost Bimatoprost	Enhanced outflow	+ + + +	Iris color change Hyperemia Periocular skin pigmentation
	Unoprostone		+ +	Eyelash growth

Rationale for drugs Combinations

- Do not combine drugs of same pharmalogical group
- More than two drugs usually not recommended. Increasing the number of topical medications increases the incidence of adverse effects and decreases patient compliance
- If first line of drugs is not effective or tachyphylaxis occurs-change drug rather than adding another drug


FIRST CHOICE MONOTHERAPY
EFFECTIVE ON IOP
VERIFY TOLERABILITY
NON-EFFECTIVE ON IOP
CHANGE MONOTHERAPY
Target IOP reached
Target IOP not reached
Continue
Add 2nd drug
effective on IOP
Non-effective on IOP
Target IOP maintained
Target IOP reached
Target IOP not reached
Substitute the 2nd drug and verify efficacy/tolerability
Other therapeutic options e.g. Surgery, Laser , etc
PERIODICALLY VERIFY ENDPOINTS
- Quality of life
- Visual Field
- Optic disc
- IOP
If poor response to several different medications, consider non-compliance
©European Glaucoma Society

Prostaglandin analogues

- Mechanism of action: Increases uveoscleral outflow
- Indication : First line therapy
- Preparations :

1. Latanoprost (0.005%) - PG F2-α analogue, OD dosage, additive effect with timolol.
2. Bimatoprost (0.03%) - decreases outflow resistance, OD dosage.
3. Travoprost (0.04%) – PG F2-α analogue, OD dosage.
4. Unoprostive isopropyl (0.12%) – dolosanoid related structure similar to PG F2-α, BD dosage.

Side effects Prostaglandin analogues

- Systemic

1. Upper respiratory tract symptoms (flu like)
2. Headache and precipitation of migraine in susceptible individuals
3. Muscle and joint pains
4. Skin rash

⦿ Ocular Side Effects

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

β–adrenergic antagonists

- **Mechanism of action** – Block β receptors in ciliary processes decreases aqueous production
- **Indication** – Used as first line therapy for patients who cant use PG analogues.
- **Preparations**

1. Timolol maleate(0.25%,0.5%) – non selective β blocker, BD dosage

   "short term escape" and "long term drift"
2. Betaxolol(0.25%,0.5%) – cardioselctive (β1) blocker, BD dosage, useful in asthmatics,

   less effective than timolol.
3. Levobunolol(0.5%)–Non selective β blocker
4. Carteolol(1%, 2%) – lesser incidence of bradycardia
5. Metipranolol(0.1%, 0.3%, 0.6%)

Side effects of β–adrenergic antagonists

- Systemic

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

- Ocular

1. Conjunctival hyperaemia
2. Superficial punctate keratopathy
3. Corneal anaesthesia

Contraindications β–adrenergic antagonists

1. Bronchial asthma
2. Chronic obstructive pulmonary disease
3. Heart blocks
4. Congestive heart failure
5. Cardiomyopathy

Parasympathomimetics

- Classification

1. Agonists (direct acting) – Pilocarpine

2. Cholinesterase Inhibitors (Indirect acting) :

- Reversible – Physostigmine
- Irreversible – Echothiophate iodide, Demecarium, Diisopropyl-fluoro-phosphate

3. Dual action - e.g. Carbachol

- Mechanism of action Increase Trabecular outflow
- Preparations

  a) Pilocarpine e/d(1%,2%,4%)BD-QID dosage

  b) Ocuserts (pilo-20,pilo-40)

  c) Pilocarpine gel(4%)HS

2. Carbachol(0.75%,1.5%,3%)BD-TDS dosage, may be useful in pilocarpine sensitivity
3. Echothiophate iodide(0.125%)OD-BD dosage, intense miosis, more GI side effects
4. Demecarium bromide(0.125%,0.25%)
5. Physostigmine(0.5%)

Side effects of Parasympathomimetics

- Systemic

1. Increased salivation, increased gastric, abdominal cramps, diarrhoea
2. Increased sweating, anxiety
3. Bradycardia

- Ocular

1. Miosis leading to decrease visual acuity in cases of posterior polar cataracts, impairment of night vision
2. Brow ache, head ache
3. Myopia
4. Keratitis
5. Iritis, iris cyst, posterior synechiae
6. Lenticular opacities
7. Retinal detachment

Carbonic anhydrase inhibitors

- Mechanism of action

  Decreases aqueous humor production

- Indications :

  Systemic CAI useful as short term therapy, especially in acute cases

  Topical CAI used as second line or third line agents for lowering IOP

⦿ Preparations

Oral

1. Acetazolamide tablet (diamox 250mg, IV diamox 5-10mg/kg)BD-TDS dosage
2. Dichlorphenamide(50mg) BD-TDS dosage
3. Methazolamide(50 mg) BD-TDS dosage

Topical

1. Dorzolamide(2%) BD-TDS dosage, additive effect with timolol
2. Brinzolamide (1%)BD-TDS dosage, less stinging sensation

Side effects of CAI

- Systemic

1. Paraesthesias, numbmness, lethargy, depression, malaise
2. Metabolic acidosis, hypokalemia, increased serum urate 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

- Ocular

1. Induced myopia, blurred vision
2. Stinging sensation
3. Conjunctivitis, keratitis

Ocular side effects are seen with topical agents

Adrenergic agonists

- Mechanism of action

  Increases aqueous outflow by both α and β receptor stimulation,

  Decreases aqueous production due to stimulation of α receptors

⦿ Classification and preparations

Non selective(α and β receptor stimulation)

1. Epinephrine(0.5%,1%,2%), BD dosage
2. Dipivefrine (0.1%) – prodrug of epinephrine, increased corneal pe netration

α_2 adrenergic agonists

⦿ Apraclonidine (0.5%,1%)BD-TDS dosage, used prophylactically for prevention of IOP elevation following laser trabeculoplasty, YAG laser iridotomy and posterior capsulotomy

⦿ Clonidine (0.125%,0.25%) BD dosage, centrally acting anti-hypertensive agent

⦿ Brimonidine(0.2%) BD-TDS dosage

Clinically, nonselective adrenergic agents have been replaced by α_2 adrenergic agonists because of their improved efficacy and side effect profile

Side effects of Adrenergic Agonists

- Systemic

1. Hypertension(nonselective agents), hypotension(α_2 adrenergic agonists)
2. Headache, fatigue, syncope
3. Anxiety, insomnia, depression

- Ocular

1. Eyelid retraction, lid edema, dermatitis
2. Conjunctival hyperemia, irritation, allergy, follicular conjunctivitis
3. Mydriasis
4. Cystoid macular edema in aphakics

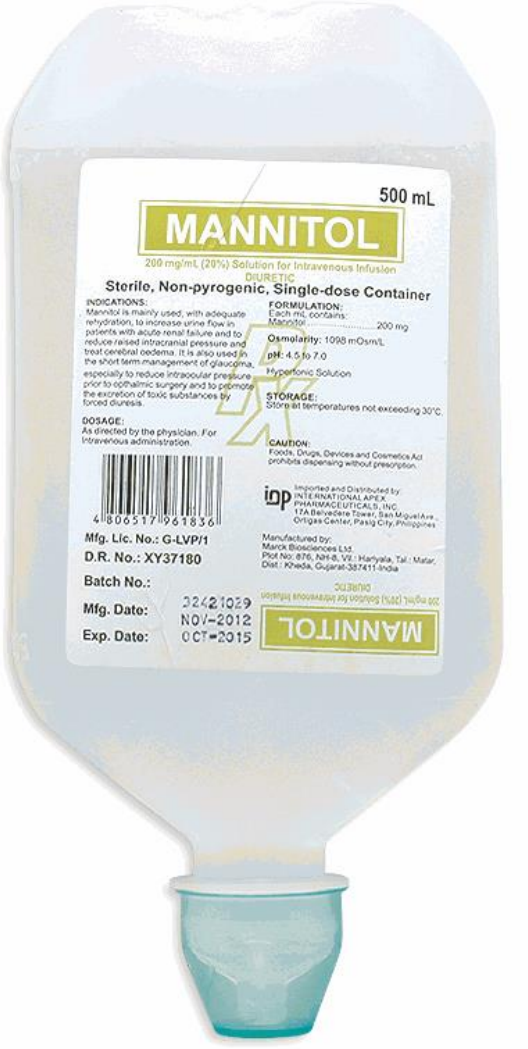
Hyperosmotic agents

- Mechanism of action

  Plasma tonicity increases, osmotic gradient dehydrates the vitreous

- Indications

  To control acute episodes of elevated IOP



Preparations

1. Mannitol IV(20% solution 1-2g/kg over 20-30 minutes), to be used cautiously in hypertensives
2. Glycerol oral (50% solution1-1.5g/kg, mixed with equal amount of water or lime juice), to be used cautiously in diabetics as it is metabolised to glucose
3. Urea IV – not recommended for routine use
4. Isosorbide – metabolically inert

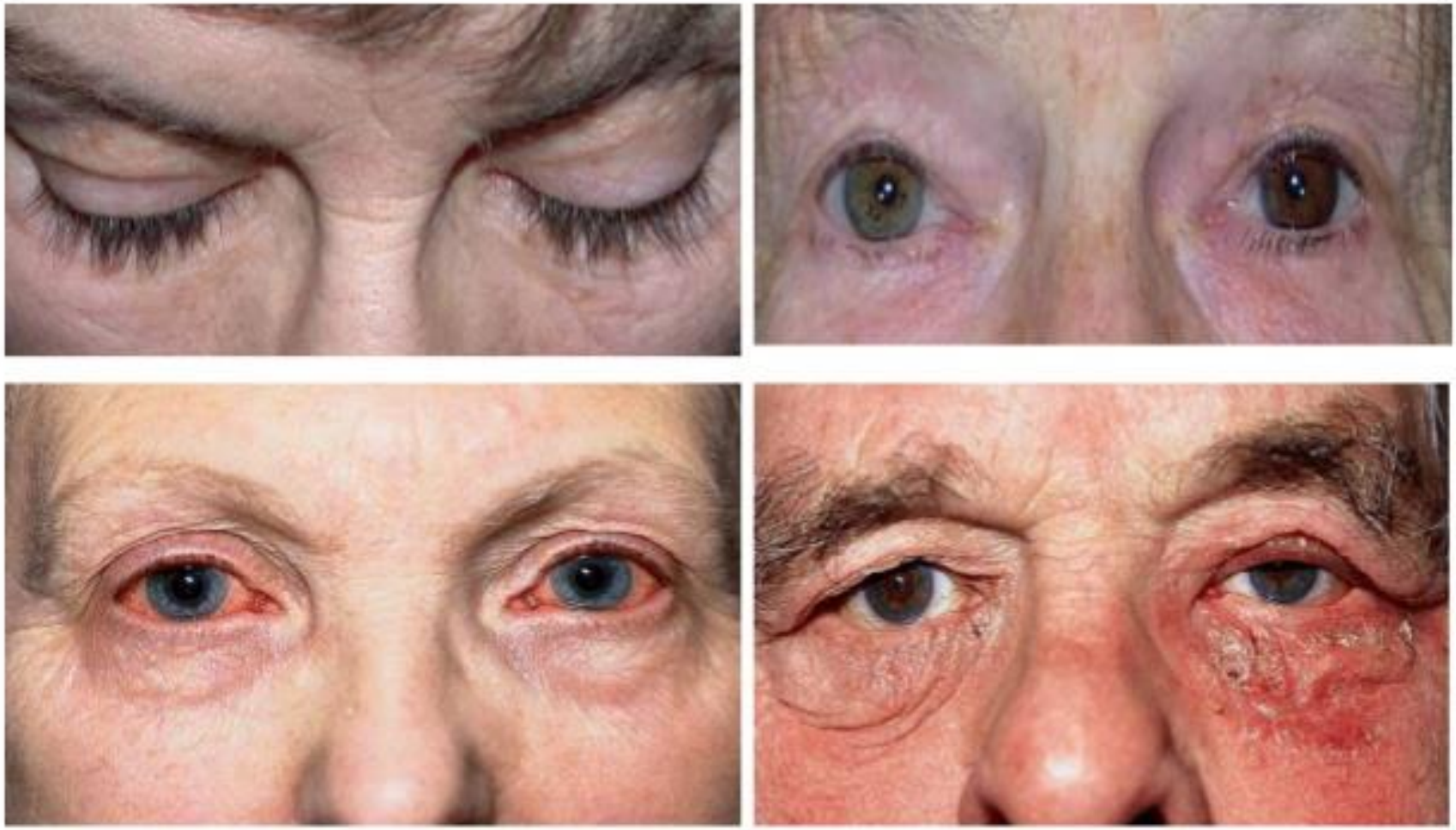
Side effects

- Systemic

1. Expansion of blood volume, congestive heart failure
2. Nausea, vomiting, diarrhoea
3. Electrolyte disturbances
4. Renal failure

- Ocular

1. Rebound increase in IOP
2. Aqueous flare



Side effects of topical medication. (A) Lengthening and hyperpigmentation of lashes with prostaglandin analogue treatment; (B) monocular prostaglandin analogue treatment – darkening of left iris and eyelid skin; (C) allergic conjunctivitis due to brimonidine; (D) blepharoconjunctivitis due to topical carbonic anhydrase inhibitors

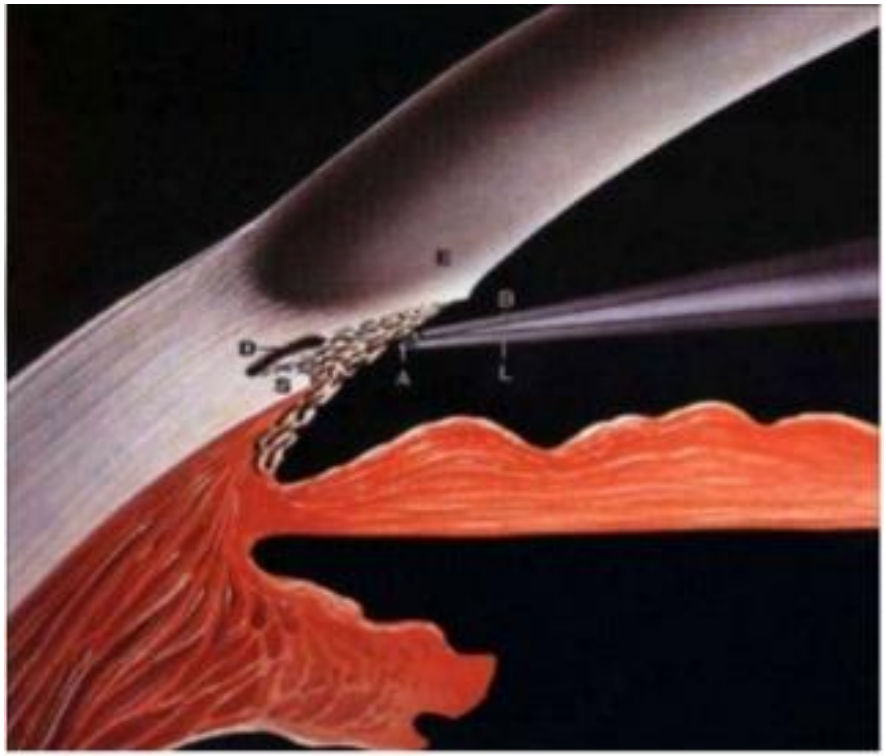
LASERS IN POAG

- Outflow Enhancement
    - Laser Trabeculoplasty
- Inflow reduction
    - Cyclophoto-coagulation (in end stage disease)

LASER TRABECULOPLASTY

INDICATIONS

- Uncontrolled glaucoma despite maximal tolerated medical therapy particularly in elderly



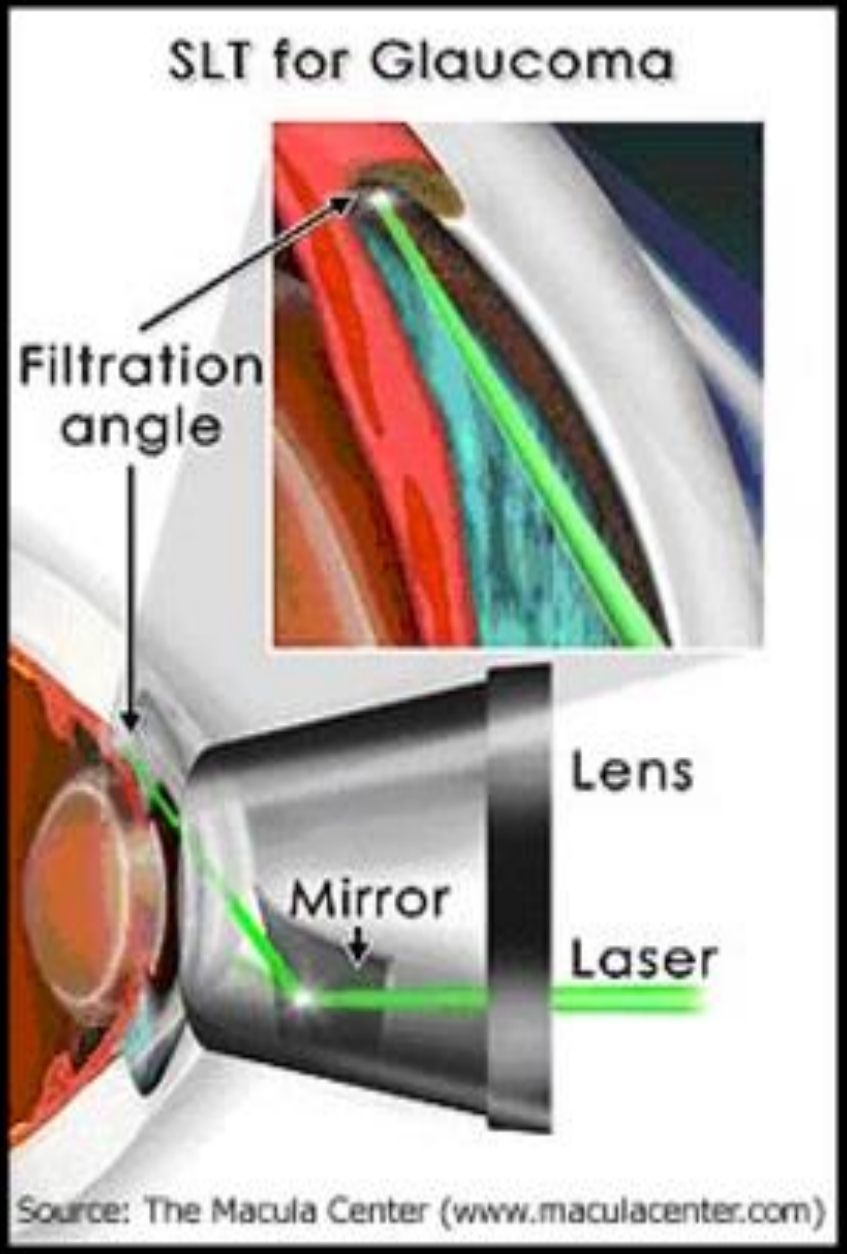
- Avoidance of poly-pharmacy (>2 drugs)
- Avoidance of surgery
- Poor compliance
- Mechanism of action- Increases outflow facility by causing shrinkage of trabecular meshwork

- Technique

  40- 50 spots on the anterior half of trabecular meshwork over 180° using a gonioloens

- Complications

1. Acute rise of IOP
2. Uveitis, haemorrhage, PAS



Surgical management

⦿ Indications

1. Uncontrolled glaucoma despite maximal medical therapy(3 drugs) and laser trabeculoplasty
2. Failure of medical therapy and /or laser trabeculoplasty
3. Non-compliance to medical therapy and unavailability of laser trabeculoplasty
4. Advanced disease requiring a very low target pressure may benefit from early surgery
5. Primary line of treatment

SURGERY IN POAG

- Penetrating filtration surgeries

  TRABECULECTOMY

- Non-penetrating glaucoma surgery (NPGS)

  Deep Sclerectomy

  Viscocanalostomy

NEUROPROTECTION

- Anti Oxidants
- Calcium Channel Blockade
- Glutamate Blockade
- Anti Apoptosis Agents
- Neurotrophins
- Heat Shock Proteins
- Nitric Acid Synthase Protection

CONGENITAL GLAUCOMA

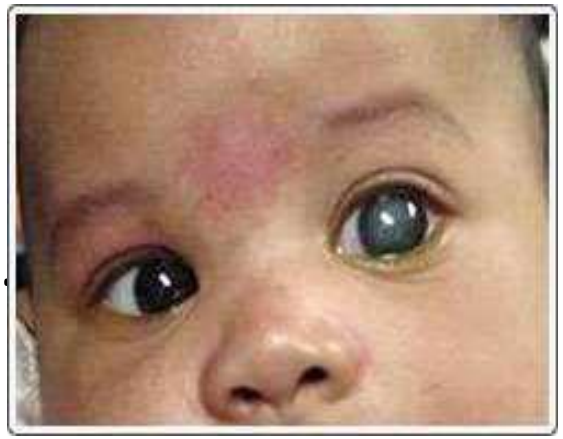
- **<u>Isolated congenital glaucoma</u>**

  1. Isolated mal development of the trabecular meshwork

  2. No other developmental ocular anomalies or ocular diseases that can raise IOP.

  3. The glaucoma exists at birth, and usually before birth.

  4. Recognized with in first month



- **<u>Infantile glaucoma</u>**

  1. Synonymous with congenital glaucoma.

  2. 1 month to 3 years

- **<u>Juvenile glaucoma</u>**

  1. Primary glaucoma occurring later in child hood or early adulthood

  2. 3 years to 35 years

Epidemiology & Demographics

- Incidence – 1 in 10-15,000 live birth
- 75% cases Bilateral,
- 65% are Male
- 75 % present in 1st year of life

Genetics

- Most of cases are Sporadic.
- Transmission pattern is Autosomal recessive.
- Many cases shows Polygenic transmission
- Two genetic loci GLC3A, CYP1B1 gene on it and GLC3C, LTBP2 gene on it have been identified

PATHOGENESIS

- Primary congenital glaucoma is due to failure or abnormal development of the trabecular meshwork .
- Maldevelopment of trabeculum including the iridotrabecular junction ***(trabeculodysgenesis)*** is responsible for impaired aqueous outflow resulting in raised IOP.
- Trabeculodysgenesis is characterized by absence of the angle recess with ***iris having a flat or concave direct insertion*** into the surface.
- The iris may not completely separate from the cornea that the angle remains closed by ***persistent embryonic tissue.***

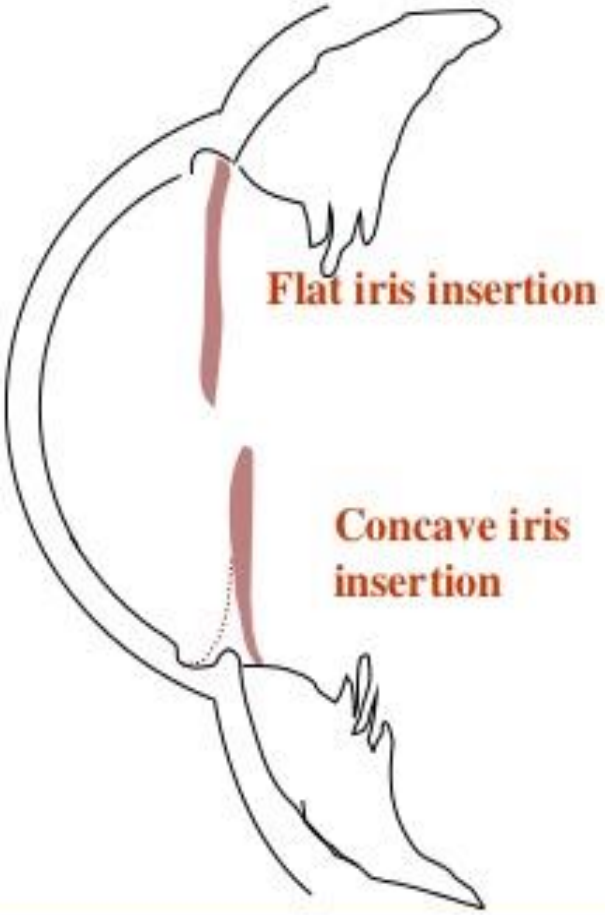
Trabeculo-dysgenesis

Flat iris insertion

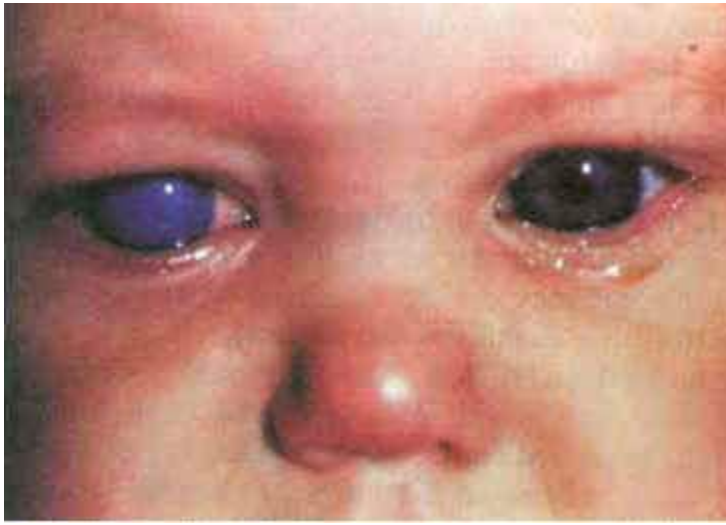
- 1. Iris inserts ant to scleral spur /on TM.
- 2. Obscures view of ciliary body.
- 3. Surface of TM - stippled / orange peel appearance.
- 4. Peripheral anterior iris stroma –thinned.
- Central stroma appears normal

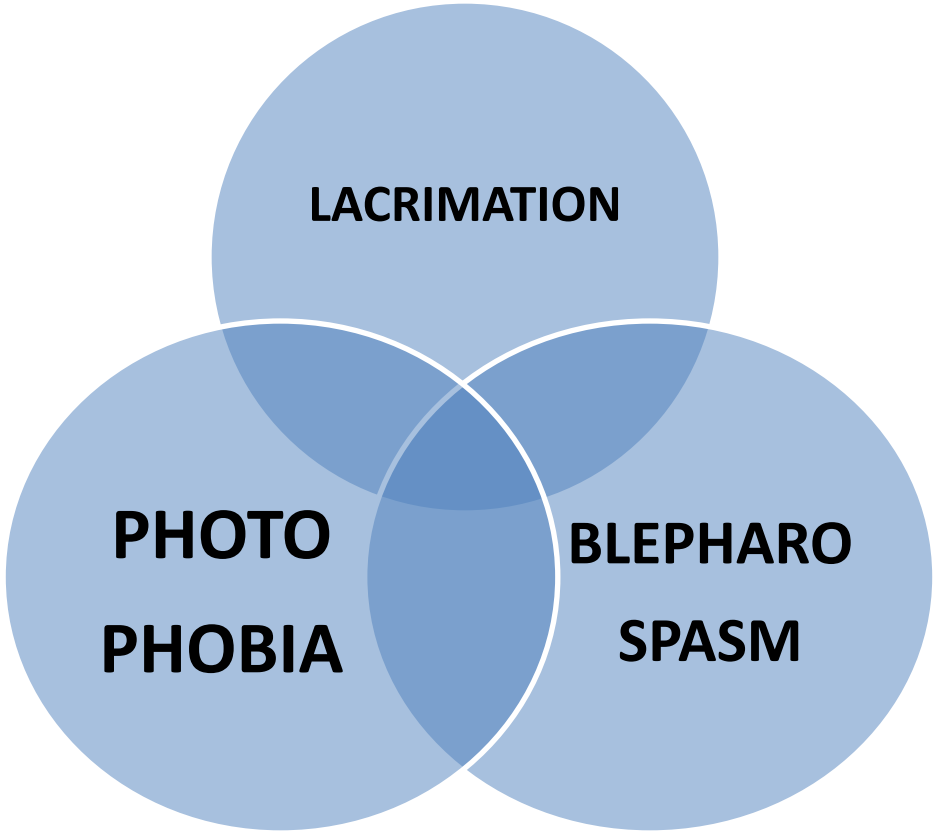
Concave iris insertion

- 1. Plane of Iris insertion normal
- 2. Anterior iris stroma sweeps over TM ending short of schwalbe's lineprobably confused as Barkan's membrane?


Flat iris insertion
Concave iris insertion

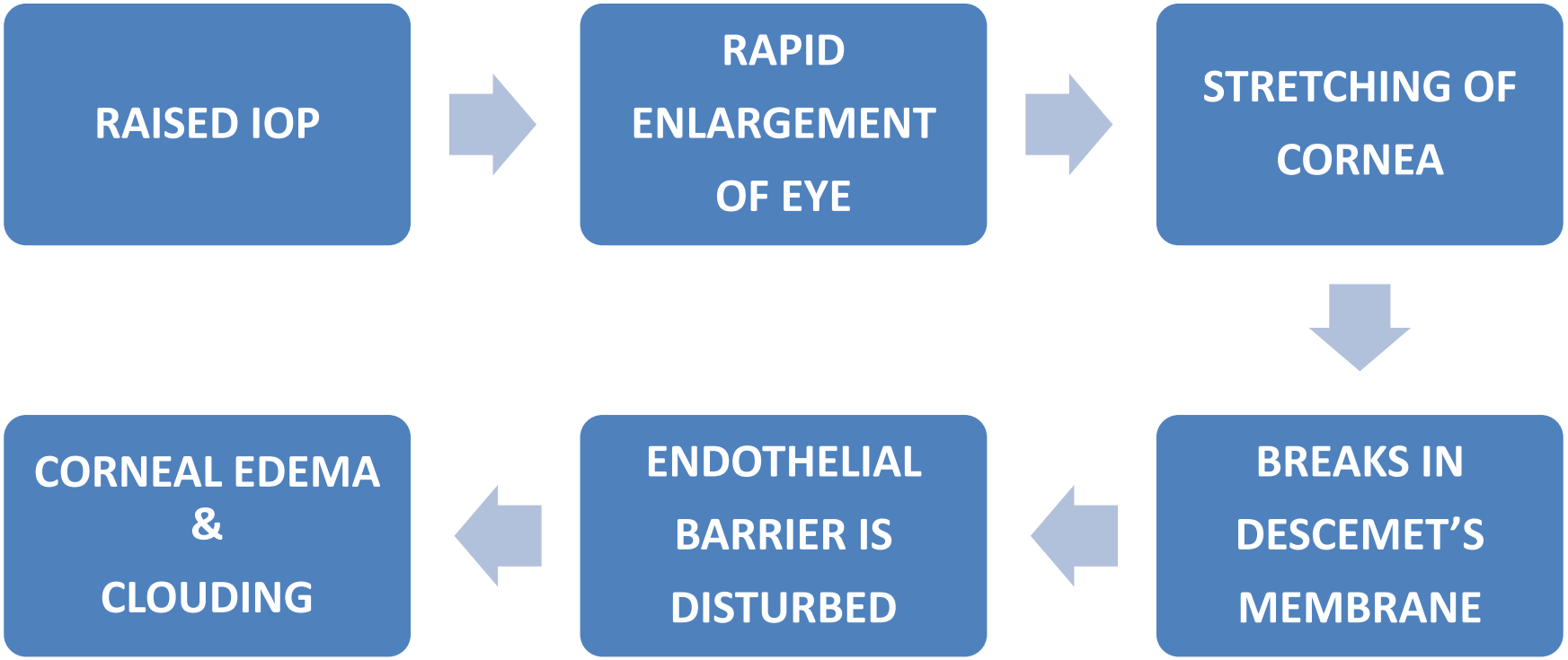
CLINICAL FEATURES





CLASSICAL TRIAD

WHAT HAPPENS



EVALUATION UNDER GA

- 1. Tonometry (After Induction And Before Intubation)
- 2. External Examination
- 3. Anterior Segment Examination
- 4. Corneal Diameter Measurement
- 5. Gonioscopy
- 6. Fundus Examination
- 7. CCT (Omit if edema present)
- 8. Ultrasound (Axial Length Measurement / B Scan)
- 9. Optic Nerve Photography
- 10. Refraction

Developmental glaucoma

- DEFINITION: Glaucoma associated with developmental anomalies of the eye present at birth.

- **Primary developmental glaucoma:** Resulting from maldevelopment of the aqueous outflow system.

- **Secondary developmental glaucoma:** Resulting from damage to the aqueous outflow system due to maldevelopment of some other portion of the eye,

  e.g., Angle closure due to pupillary block in a small eye, or

  An eye with microspherophakia or

  Dislocated lens; or

  As a forward shift of the lens-iris diaphragm in persistent hyperplastic primary vitreous or retinopathy of prematurity

Developmental glaucoma(with asso. anomalies)

- Glaucoma assoc. with iridocorneal dysgenesis
- Glaucoma assoc. with aniridia
- Glaucoma assoc. with ectopia lentis syndromes
- Glaucoma assoc. with phakomatosis
- Miscellaneous conditions


THANK
YOU!