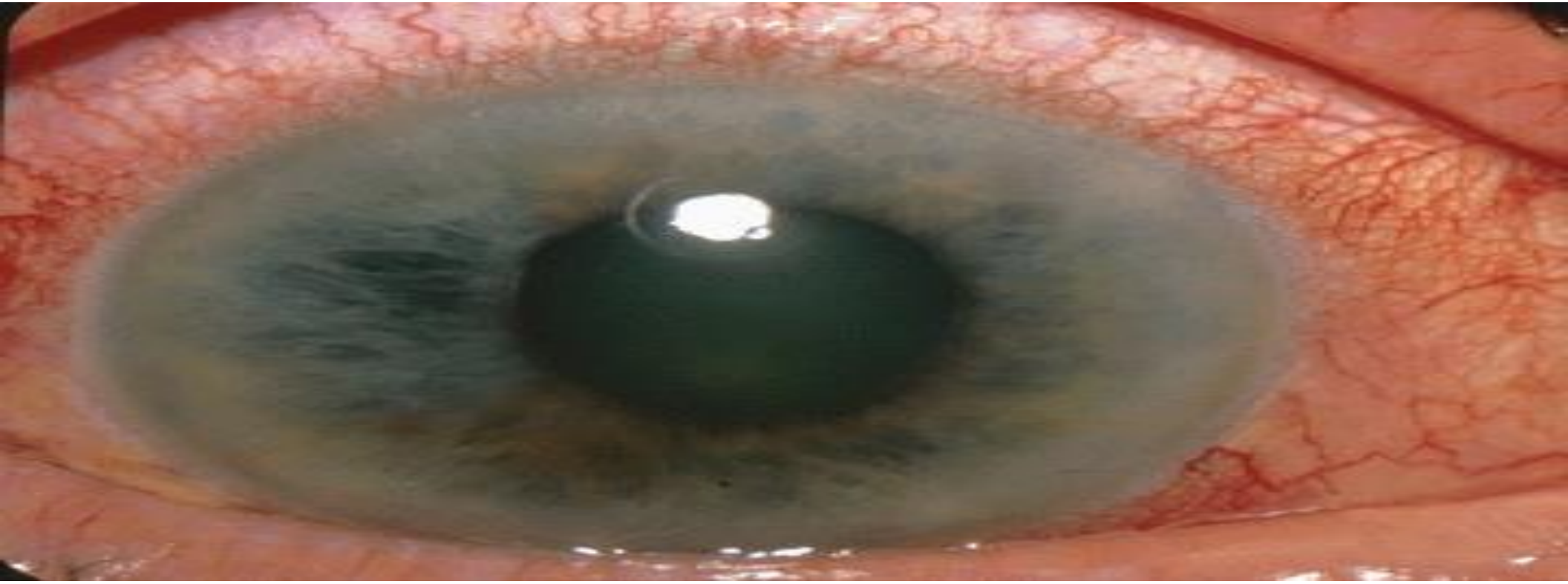


GLAUCOMA

Done by : Mohammad Qaddoura , Asma'a Alrfo'o , Haneen Al-Tarawneh , Sadeen Mohammad , Ibtihal Al-Khawaldeh

Introduction

- The glaucomas are a group of diseases causing damage to the optic nerve (**optic neuropathy**) by the effects of **raised ocular pressure** on the optic nerve head.
- Independent **ischemia** of the optic nerve head may also be important.
- Axon loss results in **visual field defects** and a **loss of visual acuity** if the central visual field is involved.

pathophysiology

- The intraocular pressure level is determined by a **balance between production and removal of aqueous humor .**

- Aqueous is actively secreted into the posterior chamber by the **ciliary processes**, by a combination of **active transport and ultrafiltration**. It then passes through the pupil into the anterior chamber and leaves the eye, predominantly, via **the trabecular meshwork, Schlemm 's canal and the episcleral veins to reach the bloodstream (the conventional pathway) .** A small but important proportion of the aqueous **(4%) drains across the ciliary body into the supra - choroidal space and is absorbed into the venous circulation (the uveoscleral pathway)**

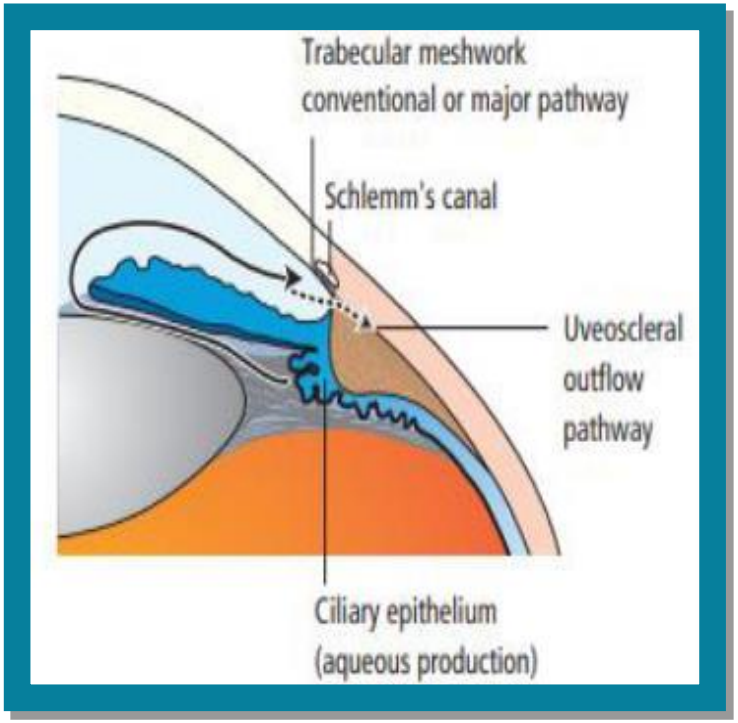
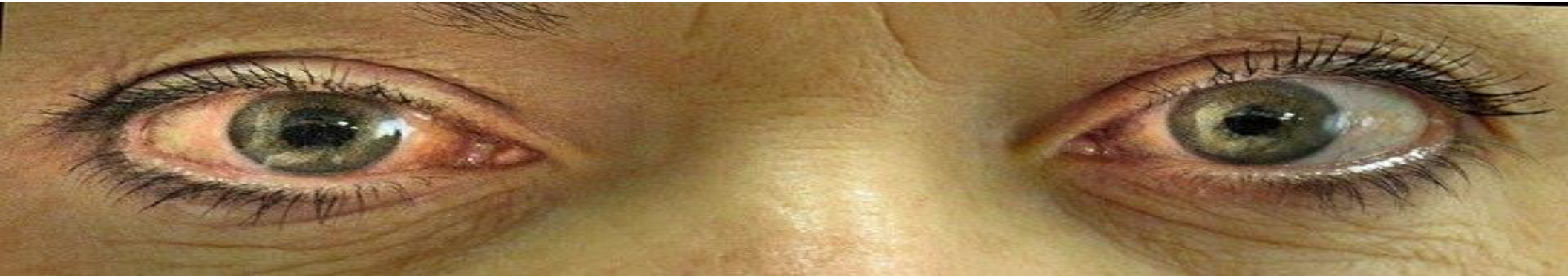


Diagram of the drainage angle , showing routes taken by aqueous from production to absorption

Two theories have been advanced to explain how elevated IOP, acting at the nerve head , damages the optic nerve fibers



theory 1

Raised intraocular pressure causes **mechanical** damage to the axons.

theory 2

Raised intraocular pressure causes **ischemia** of the nerve axons by reducing blood flow at the nerve head. The pathophysiology of glaucoma is probably **multifactorial** and both mechanisms are

Classification of the glaucomas

1 Primary glaucoma:
- Chronic open angle.
- Acute and chronic closed angle.

2 Congenital glaucoma:
- Primary.
- Secondary to maternal rubella infection.
- Secondary to inherited ocular disorders (e.g. aniridia – absence of the iris).

3 Secondary glaucoma (causes):
- Trauma.
- Ocular surgery.
- Associated with other ocular disease (e.g. uveitis).
- Raised episcleral venous pressure.
- Steroid induced.

classification

The mechanism by which aqueous drainage is reduced provides a means to classify the glaucoma.

Classification of the primary glaucoma is based on whether or not the **peripheral iris** is:

- **clear** of the trabecular meshwork (**open angle glaucoma**);
- **covering** the meshwork (**closed angle glaucoma**).

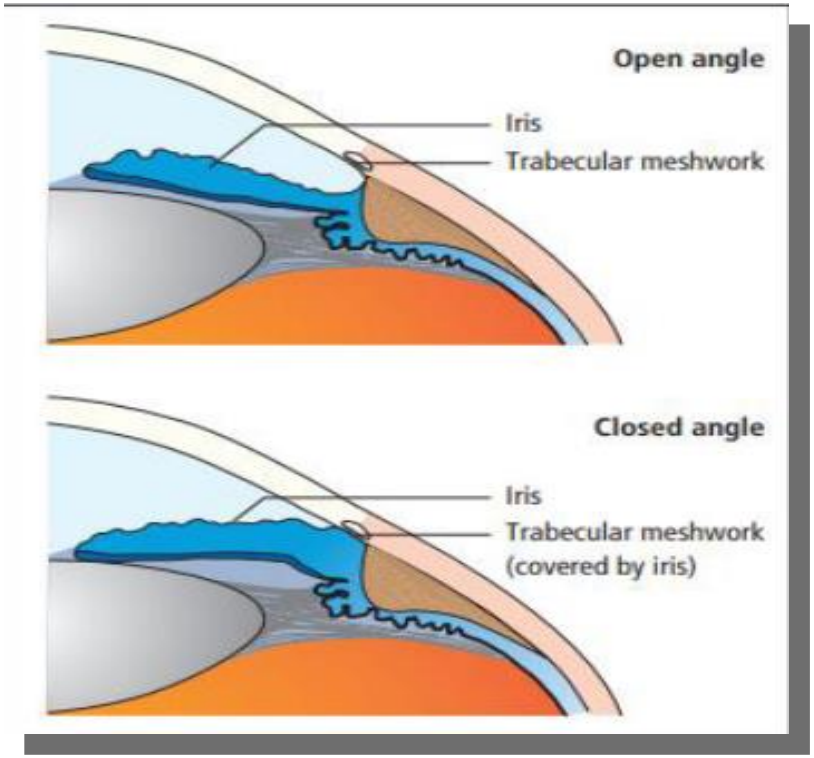
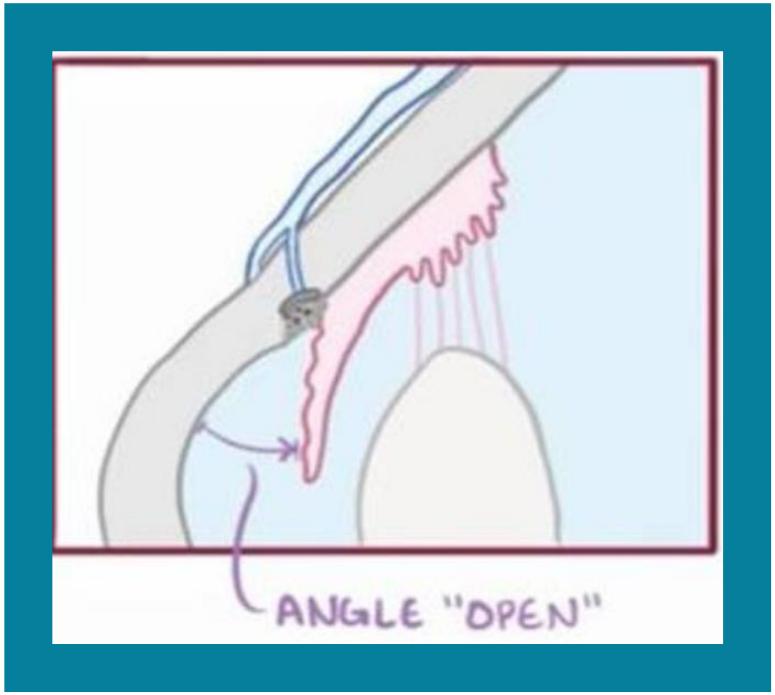


Diagram showing the difference between open and closed angle glaucoma . Outflow resistance is increased in each case . In open angle glaucoma the obstruction is due to structural changes in the trabecular meshwork . In closed angle glaucoma the peripheral iris blocks the meshwork

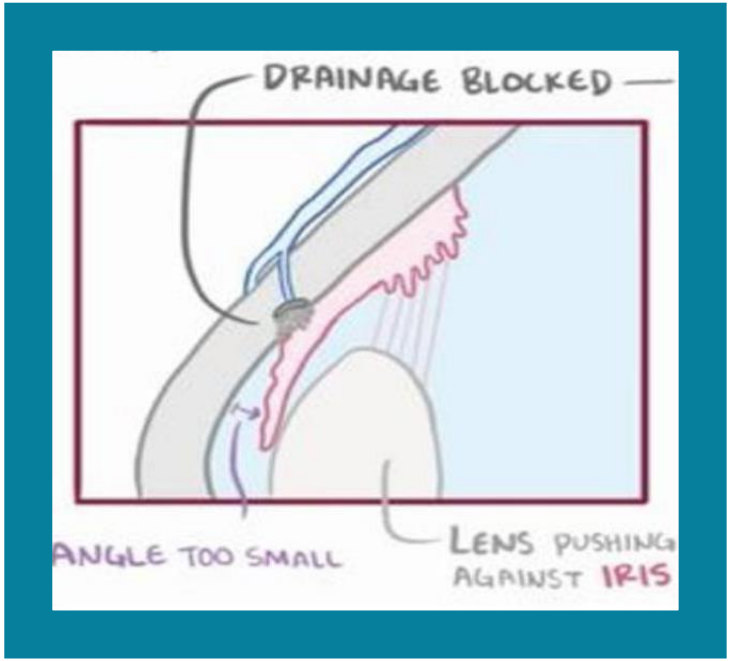
Primary glaucoma

N.B. Angle is the iridocorneal angle



Open angle glaucoma

The iris is clear of the trabecular meshwork



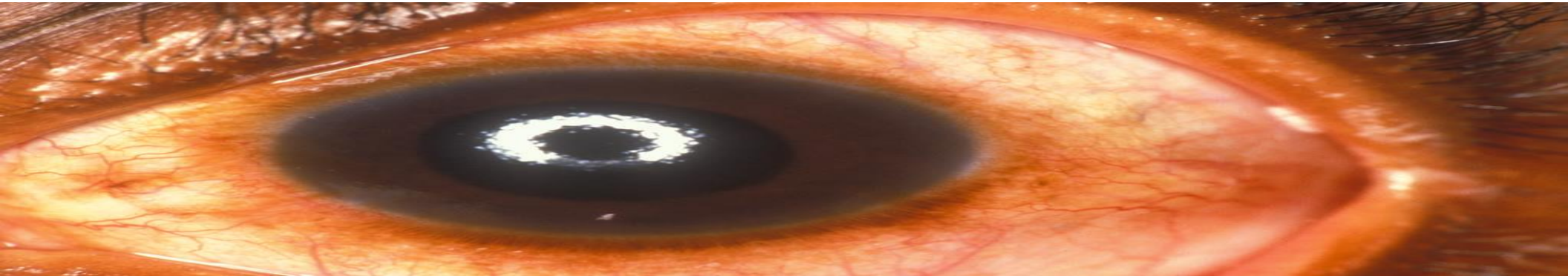
Closed "narrow" angle glaucoma

The iris is covering the trabecular meshwork

Normal tension glaucoma

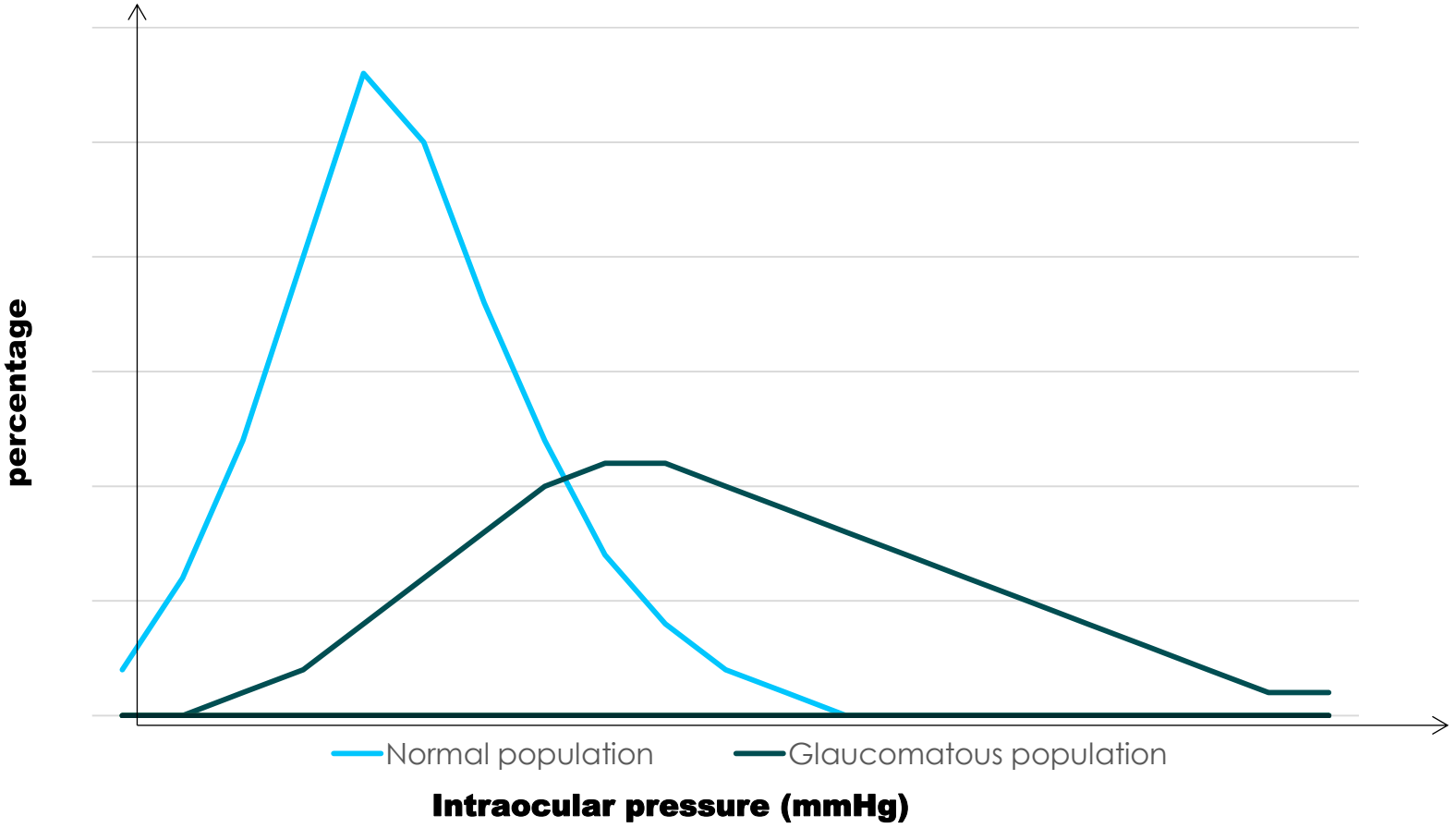
Normal tension glaucoma, considered to lie at one end of the spectrum of **chronic open angle glaucoma**, can be particularly difficult to treat, although in those with progressive field loss, lowering intraocular pressure may be beneficial. Some patients appear to have non - progressive visual field defects and may require no treatment.

Normal tension glaucoma



- A form of glaucoma also exists in which glaucomatous field loss and cupping of the optic disc occurs even though the intraocular pressure is not raised (**normal tension or low tension glaucoma**).
- It is thought that the optic nerve head in these patients is unusually susceptible to the intraocular pressure and/ or has an **intrinsically low blood flow** . Conversely, intraocular pressure may be raised without evidence of visual damage or pathological optic disc cupping (**ocular hypertension**).
- These subjects may represent the extreme end of the normal range of intraocular pressure; however, a small proportion (about 1% per year) will subsequently develop glaucoma.

The distribution of IOP in a normal and glaucomatous population



- 1 Primary open angle glaucoma
- A special contact lens applied to the cornea (a gonioscopy lens) provides a view of the iridocorneal angle with the slit lamp. In open angle glaucoma the trabecular meshwork appears normal on gonioscopy but functionally, it offers an increased resistance to the outflow of aqueous. This results in an elevated ocular pressure. The causes of outflow obstruction include:
- thickening of the trabecular lamellae, which reduces pore size;
- reduction in the number of lining trabecular cells;
- increased extracellular material in the trabecular meshwork spaces.

Chronic open angle glaucoma

- Epidemiology
- Chronic open angle glaucoma affects 1 in 200 of the population over the age of 40, affecting males and females equally. The prevalence increases with age to nearly 10% in the over-80 population. There may be a family history although the mode of inheritance is often unclear.

- History
- The symptoms of glaucoma depend on the rate at which the intraocular pressure rises. Chronic open angle glaucoma is associated with a slow rise in pressure and is symptomless until the patient becomes aware of a visual deficit or the diagnosis is made by chance. Many patients are diagnosed when the signs of glaucoma are detected by an optometrist.

Symptoms and signs of chronic open angle glaucoma

- Symptomless in its early stages.
- A white eye and clear cornea.
- Raised intraocular pressure.
- Visual field defect.
- Cupped optic disc.

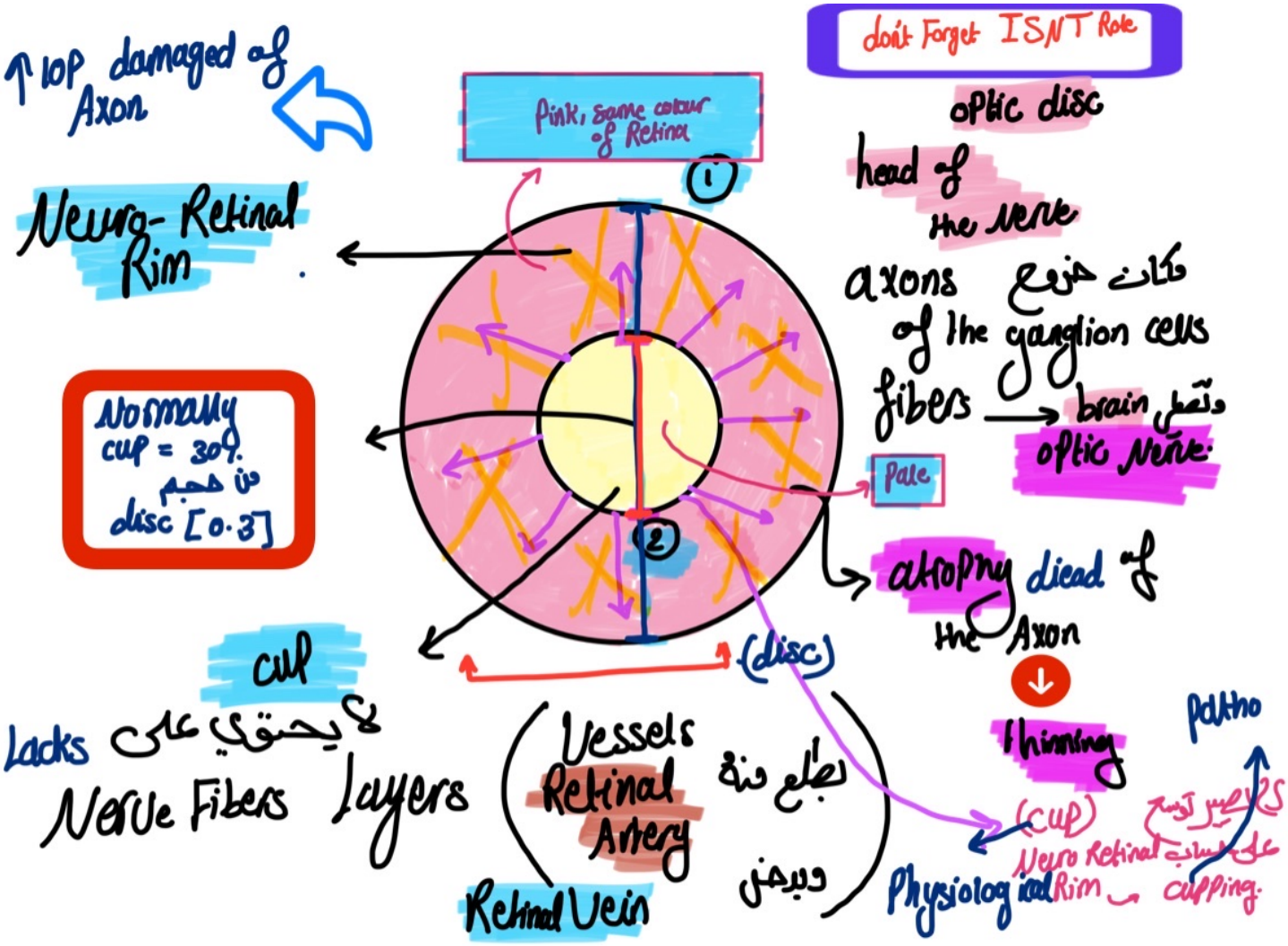
Examination

In patients with chronic open angle glaucoma the eyes are white and the corneas are clear. Assessment of a glaucoma suspect requires a full slit - lamp examination and involves:

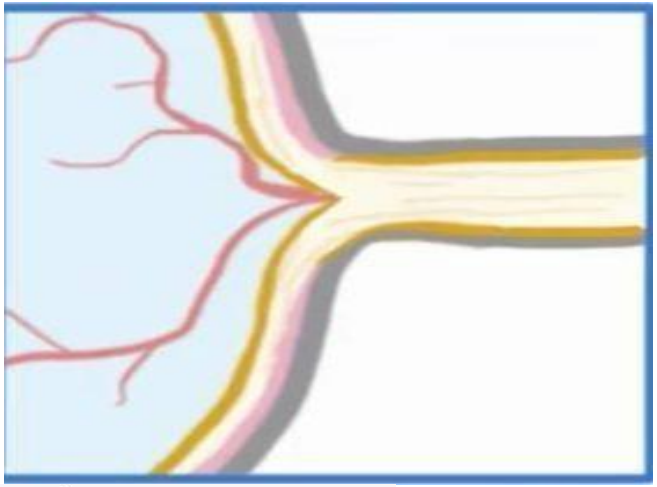
• Measurement of ocular pressure with a tonometer. The mean, normal **pressure is 15.5 mmHg**. The limits are defined as 2 standard deviations above and below the mean **(11 – 21 mmHg).** In chronic open angle glaucoma on presentation, the pressure is typically in the **22 – 40 mmHg** range. In angle closure glaucoma it rises **above 60 mmHg**.

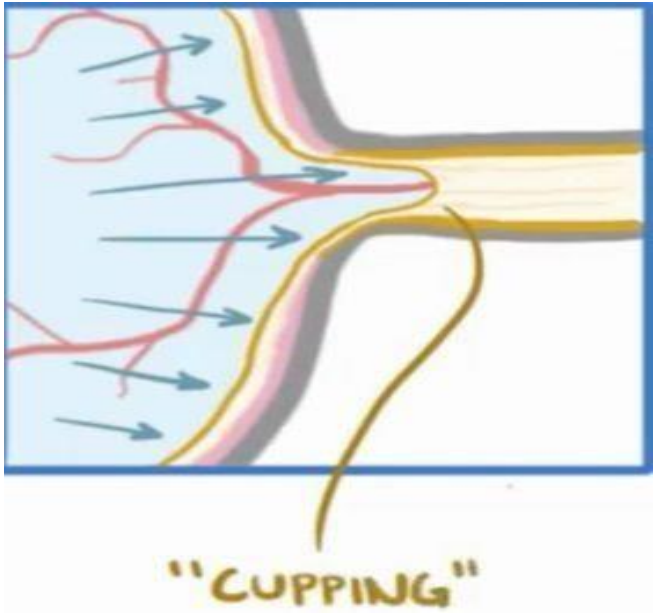
• Measurement of the thickness of the cornea with a pachymeter. The measured value of the intraocular pressure must be adjusted according to the corneal thickness .

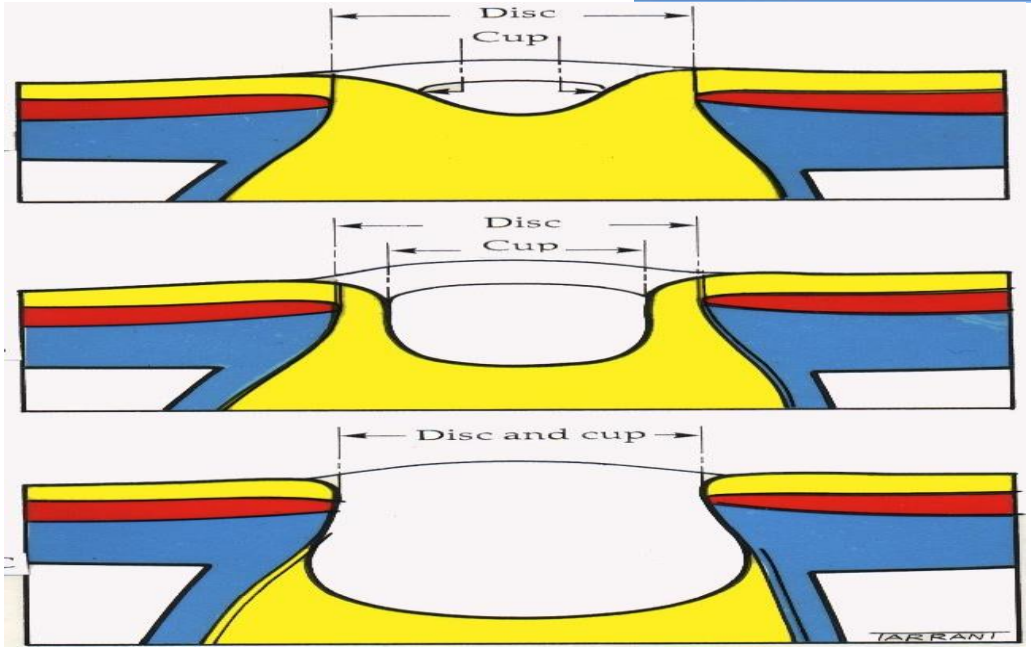
• Examination of the iridocorneal angle by **gonioscopy**, to confirm that an open angle is present.


↑ IOP damaged of Axon
Pink, same colour of Retina
doñt Forget ISNT Rule
optic disc
head of the nerve
Neuro-Retinal Rim
axons of the ganglion cells
fibers → brain
optic Nerve
Normally cup = 30% من حجم disc [0.3]
Pale
atrophy dead of the Axon
cup
(disc)
Lacks يحتوي على
Nerve Fibers layers
Vessels
Retinal Artery
Retinal Vein
Thinning
Patho
(CUP)
Physiological Rim
Neuro Retinal
cupping

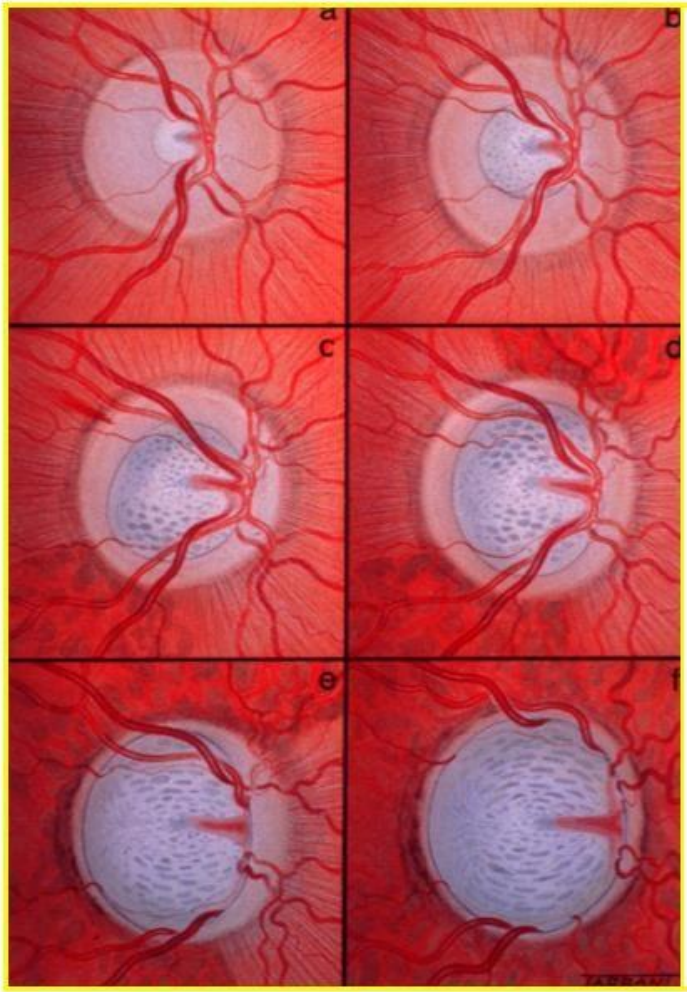
• Examination of the **optic disc** and determination of whether it is pathologically cupped . Cupping is a normal feature of the optic disc . The disc is assessed by estimating the ratio of the vertical height of the cup to that of the disc as a whole (**the cup:disc ratio**). In the normal eye **the cup:disc ratio is usually no greater than 0.4**. There is, however, a considerable range (0 – 0.8), and the size of the cup is related to the size of the disc. **It is greater in bigger discs and less in smaller discs**. In chronic glaucoma, axons leaving the optic nerve head die. The central cup expands and the outer rim of nerve fibres (**neuroretinal rim**) becomes **thinner**. The nerve head becomes atrophic. The vertical cup:disc ratio becomes greater than 0.4 and the cup **deepens**. If the cup is deep but the cup:disc ratio is lower than 0.4, then chronic glaucoma is unlikely, unless the disc is very small. **Notching** of the rim, implying focal axonal loss, may also be a sign of glaucomatous damage. Defects in the nerve fibre layer of the retina may also be apparent and determine the location and area of field loss.




"CUPPING"


Disc
Cup
Disc
Cup
Disc and cup
TARRANT

Progression of glaucomatous cupping



a. Normal (c:d ratio 0.2)

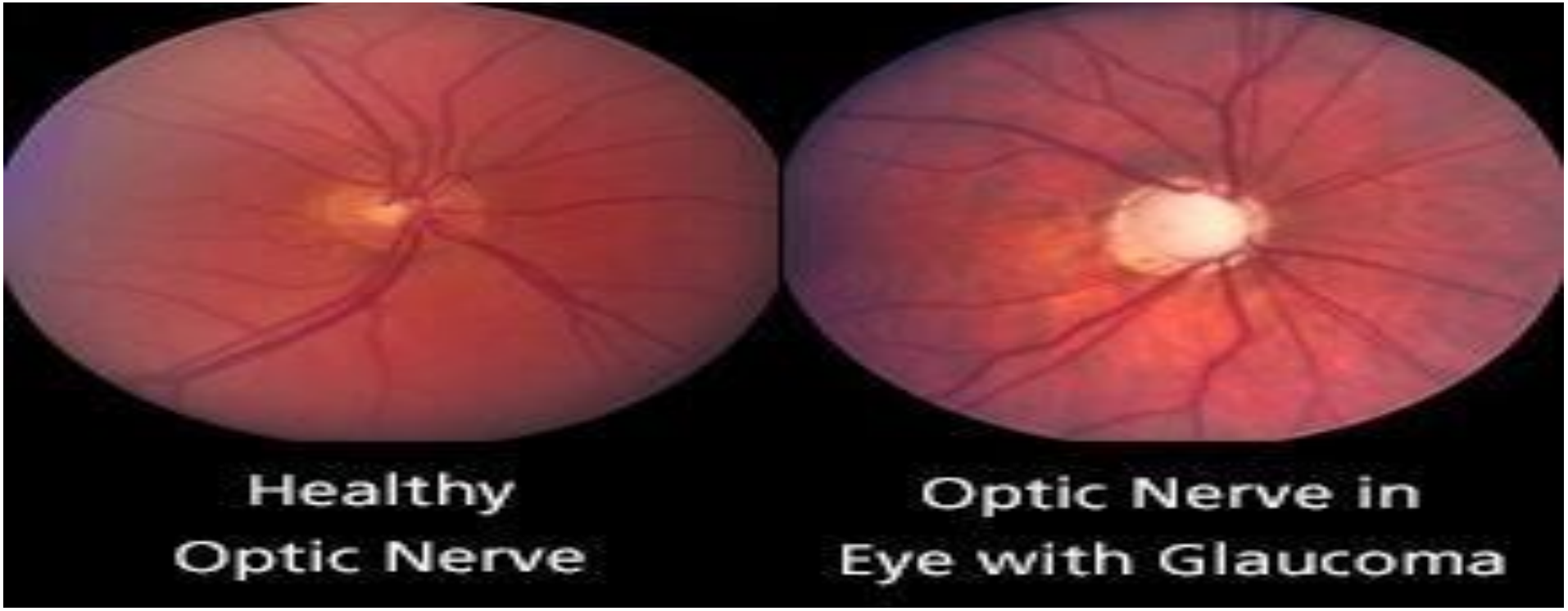
b. Concentric enlargement (c:d ratio 0.5)

c. Inferior expansion with retinal nerve fibre loss

d. Superior expansion with retinal nerve fibre loss

e. Advanced cupping with nasal displacement of vessels

f. Total cupping with loss of all retinal nerve fibres


Healthy
Optic Nerve
Optic Nerve in
Eye with Glaucoma

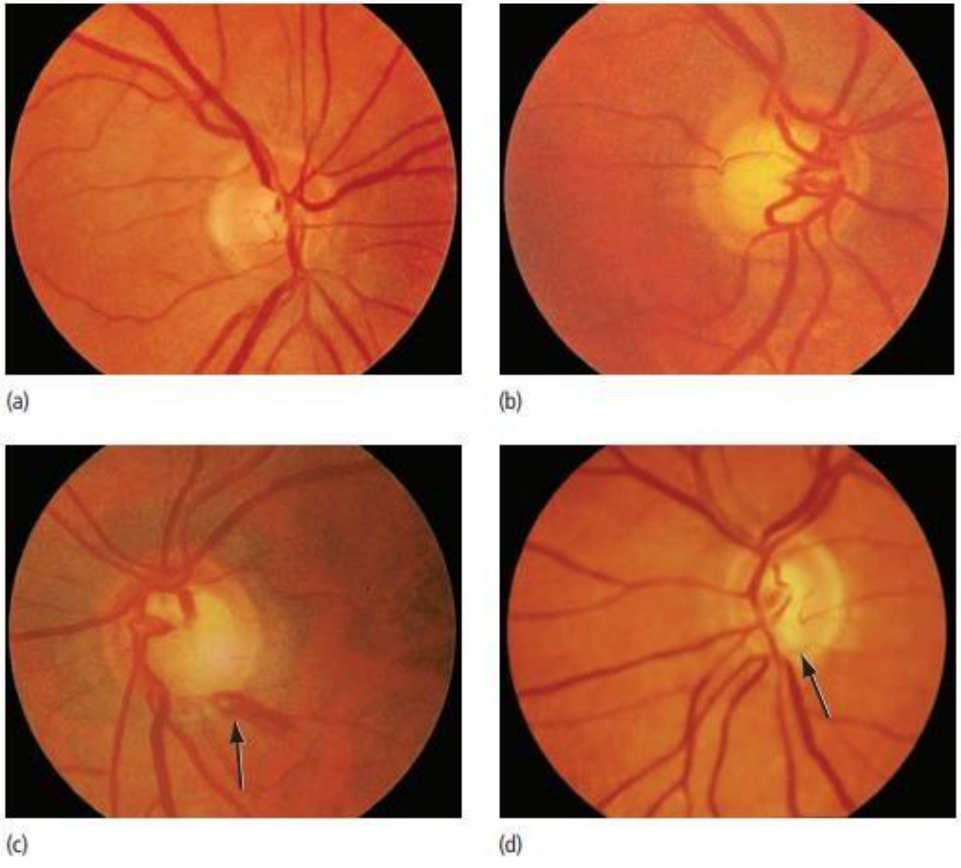
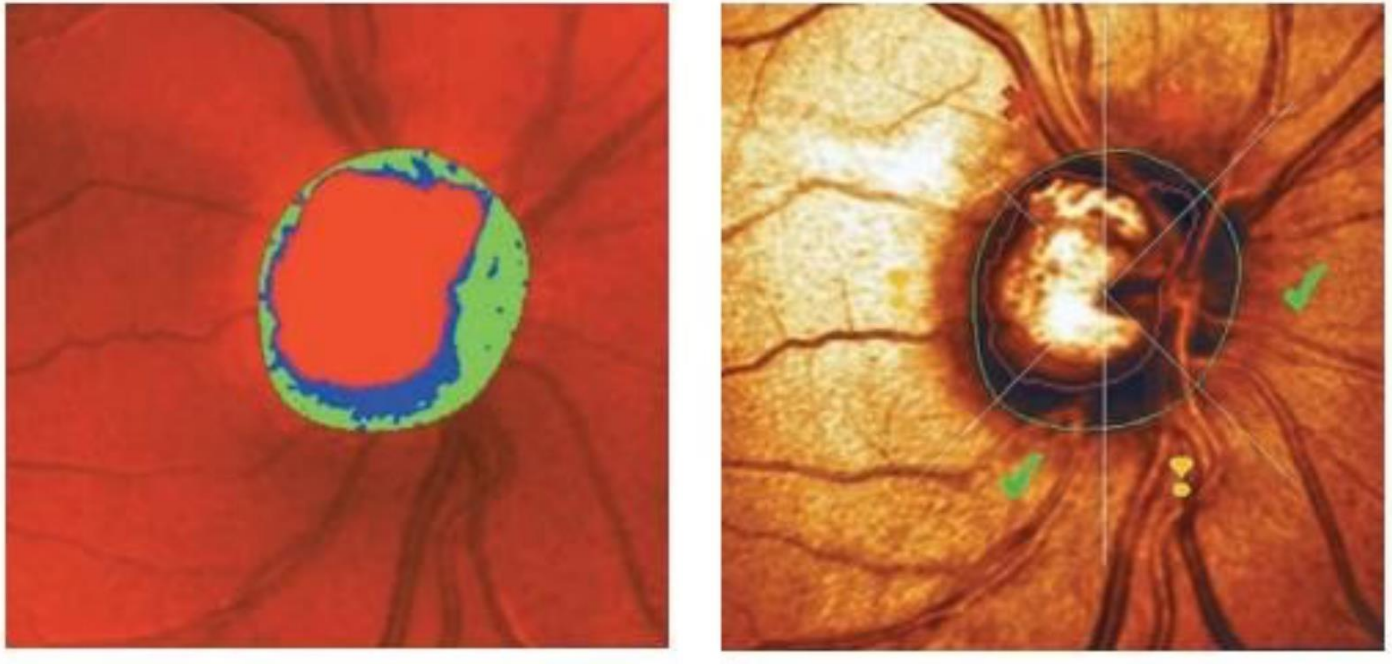


Figure 10.5 Comparison of (a) a normal optic disc, (b) a glaucomatous optic disc. (c) A disc haemorrhage (arrowed) is a feature of patients with normal tension glaucoma. (d) A glaucomatous notch (arrowed) in the disc.



(a) (b)

Figure 10.6 A scanning laser ophthalmoscope (Heidelberg) picture of the optic nerve head. The thin green circle in (b) outlines the optic nerve head, allowing the machine to calculate the area of the cup (red in (a)) and neuroretinal rim in different sectors of the disc.

Treatment

Treatment is aimed at **reducing intraocular pressure**. The level to which the pressure must be lowered varies from patient to patient and is that which minimizes further glaucomatous visual loss. This requires careful monitoring in the outpatient clinic. Three modalities of treatment are available:

1. medical treatment;
2. laser treatment;
3. surgical treatment.

1. Medical treatment

Topical drugs commonly used in the treatment of glaucoma are listed in Table 10.1 .

- **prostaglandin analogues** are becoming the first - line treatment. They act by **increasing the passage of aqueous through the uveoscleral pathway**.
- **Topical adrenergic beta - blockers** may further reduce the pressure by **suppressing aqueous secretion**. **Non - selective beta - blockers** carry the risk of precipitating asthma through their beta - 2 blocking action, following systemic absorption, or they may exacerbate an existing heart block through their beta - 1 action.

Beta - 1 selective beta - blockers may have fewer systemic side effects, but must still be used with caution in those with **respiratory disease**, particularly **asthma**, which may be exacerbated even by the small residual beta - 2 activity.

- **Pilocarpine** may occasionally be used in the treatment of chronic open angle glaucoma. If intraocular pressure remains elevated the choice lies between:

• adding additional medical treatment;

• laser treatment;

• surgical drainage procedures.

Table 10.1 Examples and mode of action of drugs used in the treatment of glaucoma. Side effects occur with variable frequency. Systemic effects are due to systemic absorption of the drug.

Drug	Action	Side effects
Topical agents		
Beta-blockers (timolol, carteolol, levobunolol, metipranolol, betaxolol-selective)	Decrease secretion	Exacerbate asthma and chronic airway disease Hypotension, bradycardia, heart block
Parasympathomimetic (pilocarpine)	Increase outflow	Visual blurring in the young Darkening of the visual world due to pupillary constriction Initially, headache due to ciliary spasm
Sympathomimetic (adrenaline, dipivefrine)	Increase outflow Decrease secretion	Redness of the eye Headache palpitations
Alpha-2 agonists (apraclonidine, brimonidine)	Increase outflow through the uveoscleral pathway Decrease secretion	Redness of the eye Fatigue, drowsiness
Carbonic anhydrase inhibitors (dorzolamide, brinzolamide)	Decrease secretion	Stinging Unpleasant taste Headache
Prostaglandin analogues (latanoprost, travaprost, bimatoprost, tafluprost, unoprostone)	Increase outflow through the uveoscleral pathway	Increased pigmentation of the iris and periocular skin Lengthening and darkening of the lashes, conjunctival hyperaemia Rarely, macular oedema, uveitis
Systemic agents		
Carbonic anhydrase inhibitors (acetazolamide)	Decrease secretion	Tingling in limbs Depression, sleepiness Renal stones Stevens–Johnson syndrome

2.Laser trabeculoplasty

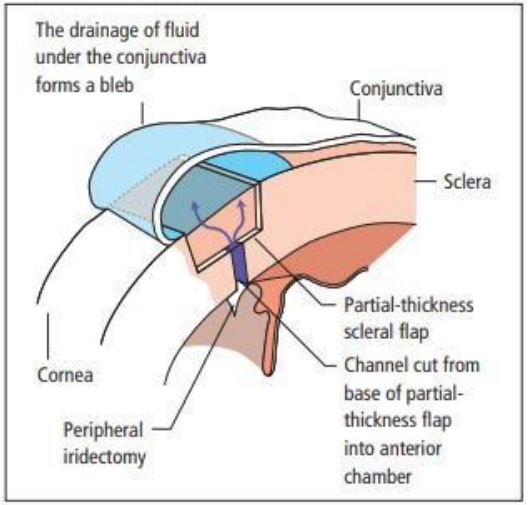
This involves placing a series of laser burns (**50 µ m** wide) in the trabecular meshwork, to improve aqueous outflow. Whilst effective initially, the intraocular pressure may slowly increase. In the UK there is an increasing tendency to proceed to early drainage surgery.

3. Surgical treatment

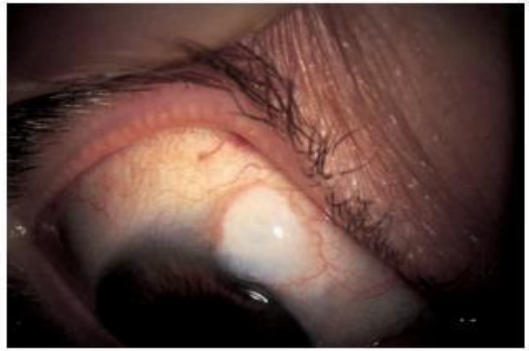
Drainage surgery (**trabeculectomy**) relies on the creation of a **fistula** between the **anterior chamber and the subconjunctival space**. Aqueous humor leaves the anterior chamber, via a bleb of conjunctiva, into the subconjunctival space. The operation usually achieves a substantial reduction in intraocular pressure. It is performed increasingly early in the treatment of glaucoma. Complications of surgery include:

- shallowing of the anterior chamber in the immediate postoperative period risking damage to the lens and cornea;
- intraocular infection;
- possibly accelerated cataract development;
- failure to reduce intraocular pressure adequately.
- an excessively low pressure (hypotony) which may cause macular oedema.

Evidence suggests that some topical medications, particularly those containing **sympathomimetic** agents or **preservatives**, may decrease the success of surgery by causing increased postoperative subconjunctival scarring, resulting in **a non - functional drainage** channel. In patients particularly prone to scarring, antimetabolite drugs (5 - fluorouracil and mitomycin) may be used at the time of surgery to **prevent subconjunctival fibrosis**..



(a)



(b)

Figure 10.8 (a) Diagram showing a section through a trabeculectomy. An incision is made in the conjunctiva, which is dissected and reflected to expose bare sclera. A partial-thickness scleral flap is then fashioned. Just anterior to the scleral spur a small opening (termed a *sclerostomy*) is made into the anterior chamber to create a low-resistance channel for aqueous. The iris is excised in the region of the sclerostomy (*iridectomy*) to prevent it moving forward and blocking the opening. The partial-thickness flap is loosely sutured back into place. The conjunctiva is tightly sutured. Aqueous can now leak through the sclerostomy, around and through the scleral flap and underneath the conjunctiva, where it forms a bleb. (b) The appearance of a trabeculectomy bleb.

Primary angle closure glaucoma

Epidemiology Primary angle **closure** glaucoma

- affects 1 in 1000 subjects over 40 years old
- **females** more commonly affected than males.
- **small eyes** (i.e. often **hypermetropic**) which therefore have **shallow anterior chambers**.

Normal physiology:

In the normal eye, the point of contact between the pupil margin and the lens causes resistance to aqueous humor flow from the posterior chamber into the anterior chamber.
This is called relative pupil block, producing a pressure drop between the two chambers.

Initiation of angle closure :

During pupil dilation, the peripheral iris bunches up in the angle.

This increases resistance and the pressure gradient bows the iris forward, closing the drainage angle.

As a result, aqueous can no longer pass through the trabecular meshwork, and intraocular pressure rises abruptly.

Structural consequence:

The peripheral iris contact leads to the formation of peripheral anterior synechiae (PAS).

These adhesions consolidate and make the obstruction permanent.

- Corneal effects:
- The stagnant aqueous circulation deprives the cornea and posterior cornea of oxygen and nutrition.
- This causes failure of endothelial pumping leading to :
- massive corneal edema and clouding.
- Raised intraocular pressure amplifies these effects and results in severe fall in vision

- Prodromal (warning) episodes:

- Before acute attack, patients may have subacute episodes with transient pressure rises.
- Symptoms include headache and colored haloes around bright lights.
- The haloes (or rainbows) occur because of mild corneal epithelial edema, which separates basal epithelial cells so they act like a diffraction grating.
- #Therefore, any patient with headaches should be asked about the presence of rainbows around lights, as this is a key sign of prodromal attacks.

Clinical presentation :

- **pain in eye** (due to ischemic tissue damage).
- **loss of vision** and There is watering of the eye .
- **blurring of vision** and seeing **colored rainbows around lights**.
- systemically unwell **: nausea and referred abdominal pain.**

photophobic (Due to inflammation of iris & ciliary spasm)

Examination

visual acuity is reduced, the eye red, the cornea cloudy and the pupil oval, fixed and dilated .

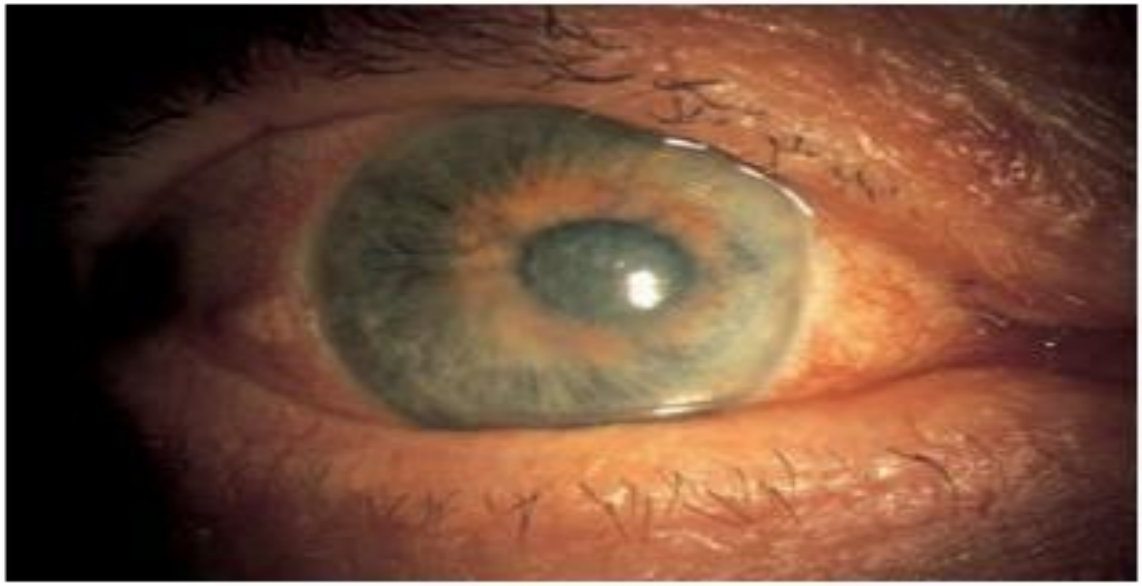


Figure 10.9 The appearance of the eye in angle closure glaucoma. Note the cloudy cornea and dilated pupil.

Treatment:

Immediate goal:

The acute and dramatic rise in intraocular pressure must be **urgently countered to prevent permanent vision damage.**

Medical therapy (initial management):

Acetazolamide: administered intravenously, then continued orally.
Topical pilocarpine: constricts the pupil and **draws the peripheral iris out of the angle.**
Topical beta-blockers: together with acetazolamide, they **reduce aqueous secretion** and lower intraocular pressure. These combined measures often succeed in breaking the acute attack.

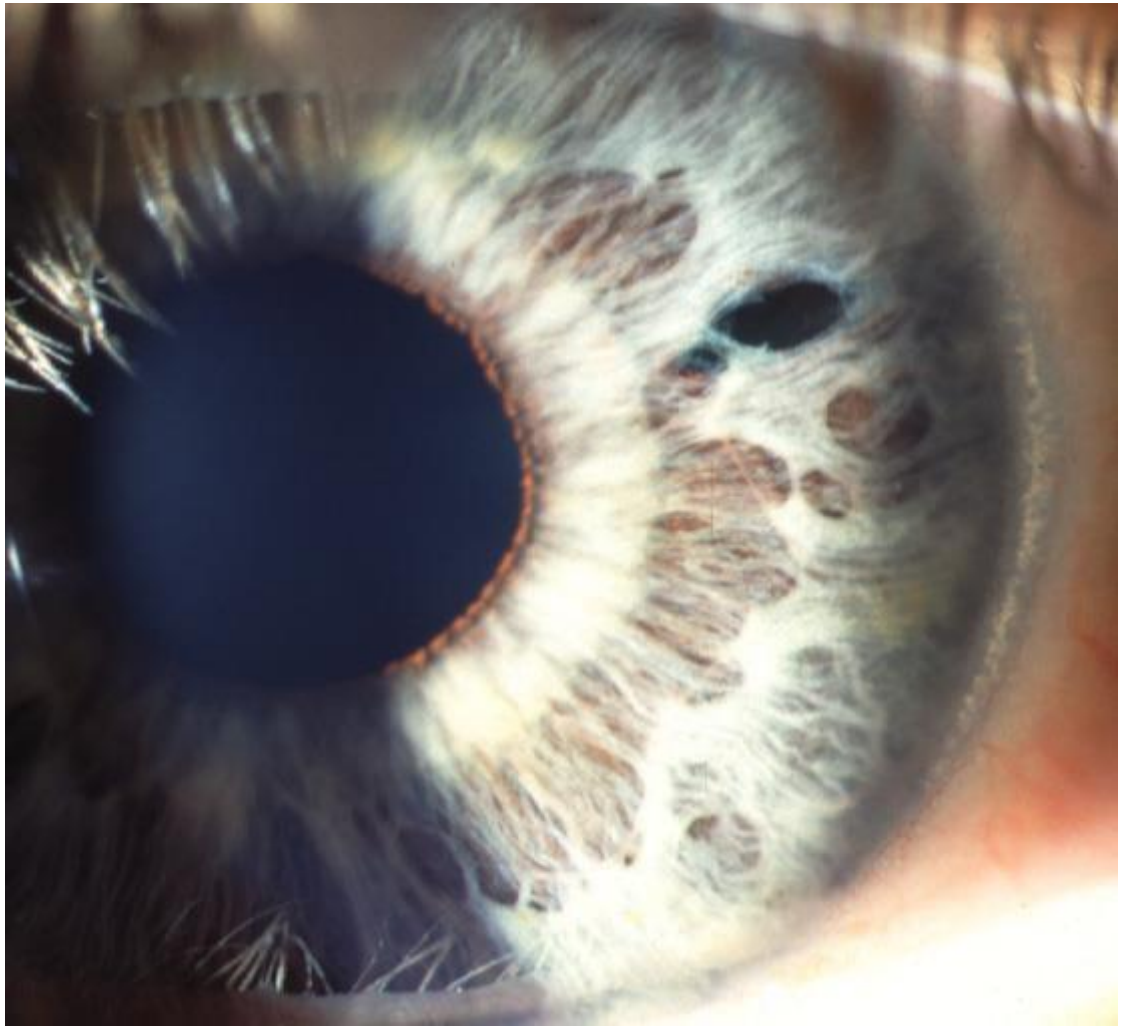
Treatment:

Definitive management:

YAG laser or surgary
Requires creation of a small hole in the peripheral iris (iridotomy or iridectomy) to prevent further attacks ,This provides an alternative pathway for aqueous humor to flow from the posterior to the anterior chamber, bypassing the pupil and thus reducing the pressure gradient.

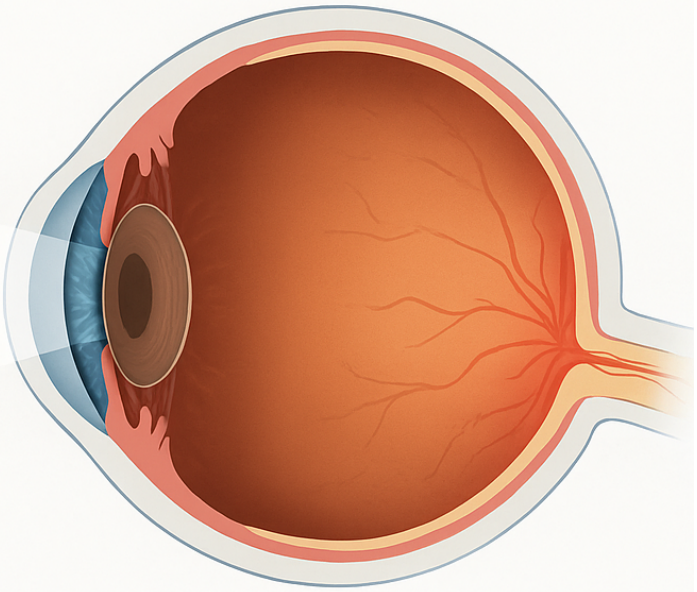
 # peripheral anterior synechiae (PAS): lowering IOP may be more difficult, and sometimes trabeculectomy or other glaucoma surgery is needed.

 # In patients with cataract Lens extraction with intraocular lens implantation may also help to open the iridocorneal angle.



Secondary Glaucoma – Causes

- **Lens-related**
  - Pseudoexfoliation syndrome
  - Cataract
- **Iris causes**
  - Pigment dispersion syndrome
  - Uveitis
  - Rubeosis iridis
- **Trauma**
  - Hyphema
  - Angle recession
- **Drug-induced**
  - Steroids



- **Raised episcleral venous pressure**
  - Carotid–cavernous sinus fistula
  - Sturge–Weber syndrome

3.Secondary glaucoma

the rise of intraocular pressure is usually due to **trabecular meshwork obstruction**. The trabecular meshwork may be blocked by:

- blood (hyphema), following blunt trauma;
- inflammatory cells (uveitis);
- pigment from the iris (pigment dispersion syndrome);
- deposition in the trabecular meshwork of material produced by the epithelium of the lens, iris and ciliary body (pseudo exfoliative glaucoma);
- drugs increasing the resistance of the meshwork (steroid - induced glaucoma).

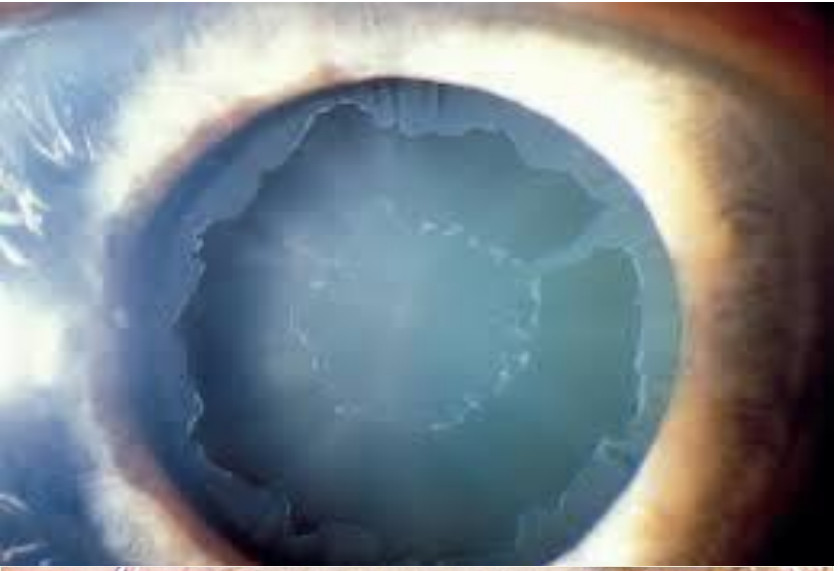
Secondary glaucoma may also result from :

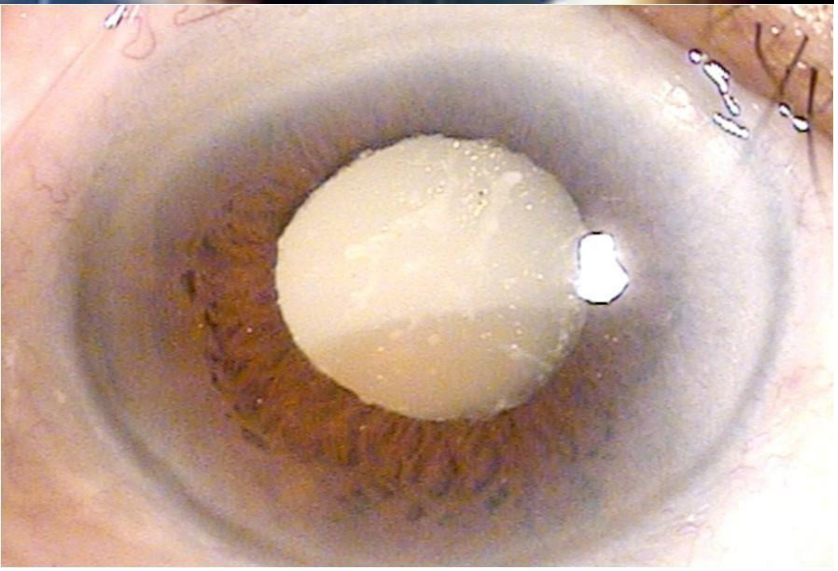
- blunt trauma to the eye causing damage to the drainage angle (**angle recession**).
- **rubeosis iridis :** Abnormal iris blood vessels may obstruct the angle and cause the iris to adhere to the peripheral cornea, closing the angle This may accompany **proliferative diabetic retinopathy or central retinal vein occlusion** due to the forward diffusion of Vaso proliferative factors such as vascular endothelial growth factor (VEGF), from the ischemic retina .
- A large choroidal **melanoma** may push the iris forward, approximating it to the peripheral cornea and causing an acute attack of angle closure glaucoma.
- A **cataract** may swell, pushing the iris forward and close the drainage angle.

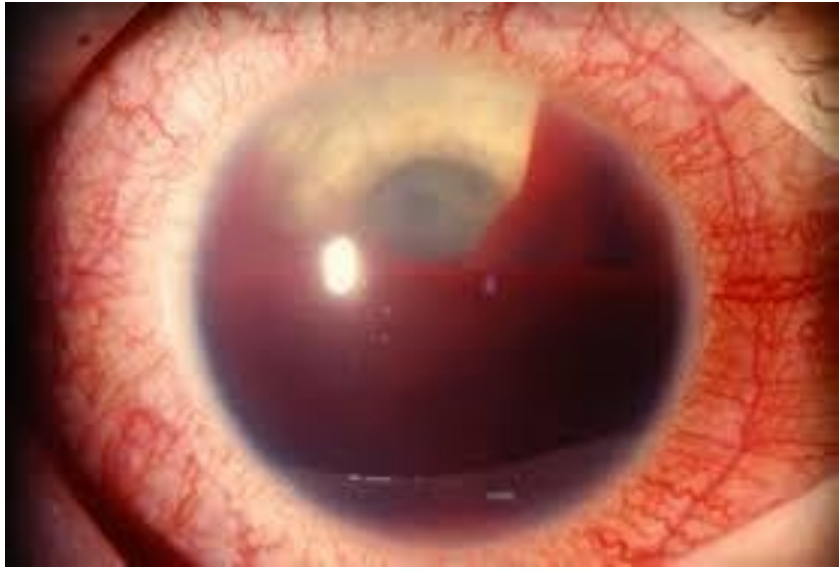
- **Uveitis** may cause the iris to adhere to the trabecular meshwork.

- **Raised episcleral venous pressure** is an unusual cause of glaucoma but may be seen in

1. **caroticocavernous sinus fistula** , where a connection between the carotid artery or its meningeal branches and the cavernous sinus causes a marked elevation in orbital venous pressure.
2. **Sturge – Weber syndrome**







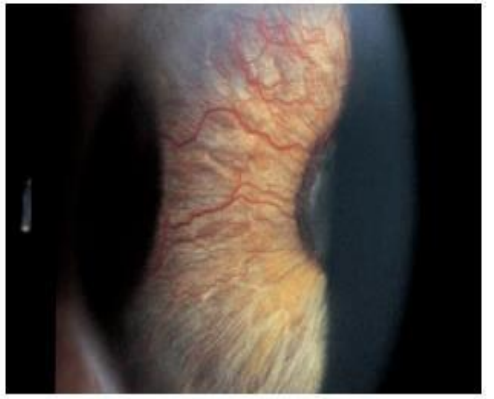
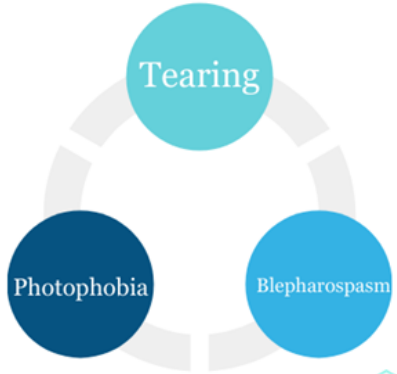


Figure 10.4 The appearance of the rubeotic iris. Note the irregular pattern of the new blood vessels on the surface.

Congenital glaucoma

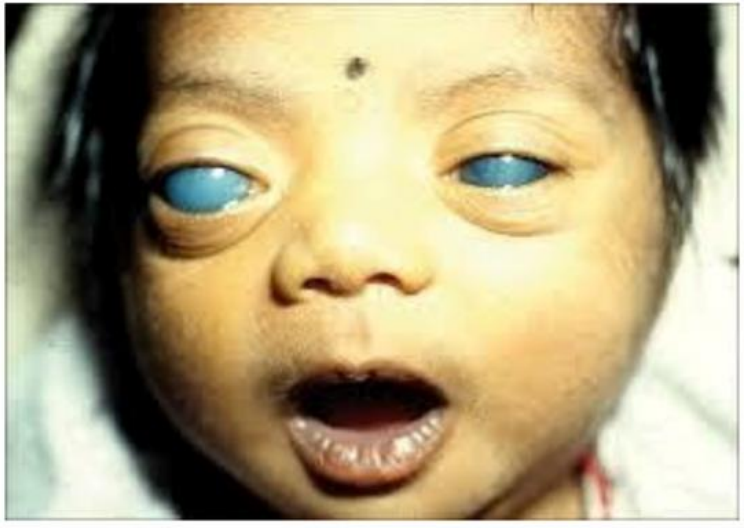
- The cause of congenital glaucoma remains uncertain. One view is that the anterior chamber angle is **developmentally** abnormal and covered with a membrane which increases the outflow resistance.
- Rare but serious cause of childhood blindness.
- This covers a diverse range of disease. It may present at birth or within the first year.

Congenital glaucoma



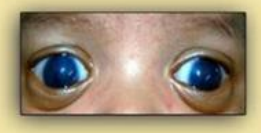
- Symptoms and signs include:
  - excessive tearing, photophobia and blepharospasm;
  - an increased corneal diameter and enlargement of the globe (**buphthalmos**), resulting in progressive myopia;
  - a cloudy cornea due to epithelial and stromal oedema; splits in Descemet ' s membrane.

- Congenital glaucoma is usually treated **surgically**. An incision is made into the trabecular meshwork (**goniotomy**) to increase aqueous drainage, or a direct passage between Schlemm ' s canal and the anterior chamber is created (**trabeculotomy**).



Congenital Glaucoma

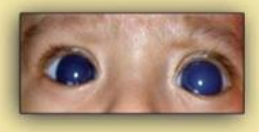
Congenital glaucoma is a rare condition that is caused by incorrect development of the eye's drainage system before birth.



Blue coloured cornea

Buphthalmos

Photophobia



Watering

Blepharospasm

Hiding face

ARAVIND EYE CARE SYSTEM

Prognosis of the glaucoma

The goal of treatment in glaucoma is to **stop or reduce the rate of visual damage**. It may be that control of intraocular pressure alone is not the only factor that needs to be addressed in the management of glaucoma. The possible role of optic nerve ischemia has been discussed, and there is interest in developing **neuroprotective drugs**. Reducing intraocular pressure is thus currently the mainstay of treatment. Some patients will continue to develop visual loss despite a large decrease in intraocular pressure. Nonetheless vigorous lowering of intraocular pressure, even when it does not prevent continued visual loss, appears to significantly reduce the rate of progression. If the diagnosis is made late, when there is already significant visual damage, the eye is more likely to become blind despite treatment .If intraocular pressure remains controlled following acute treatment of angle closure glaucoma, progressive visual damage is unlikely. The same applies to the secondary glaucoma, if treatment of the underlying cause results in a reduction of intraocular pressure into the normal range.

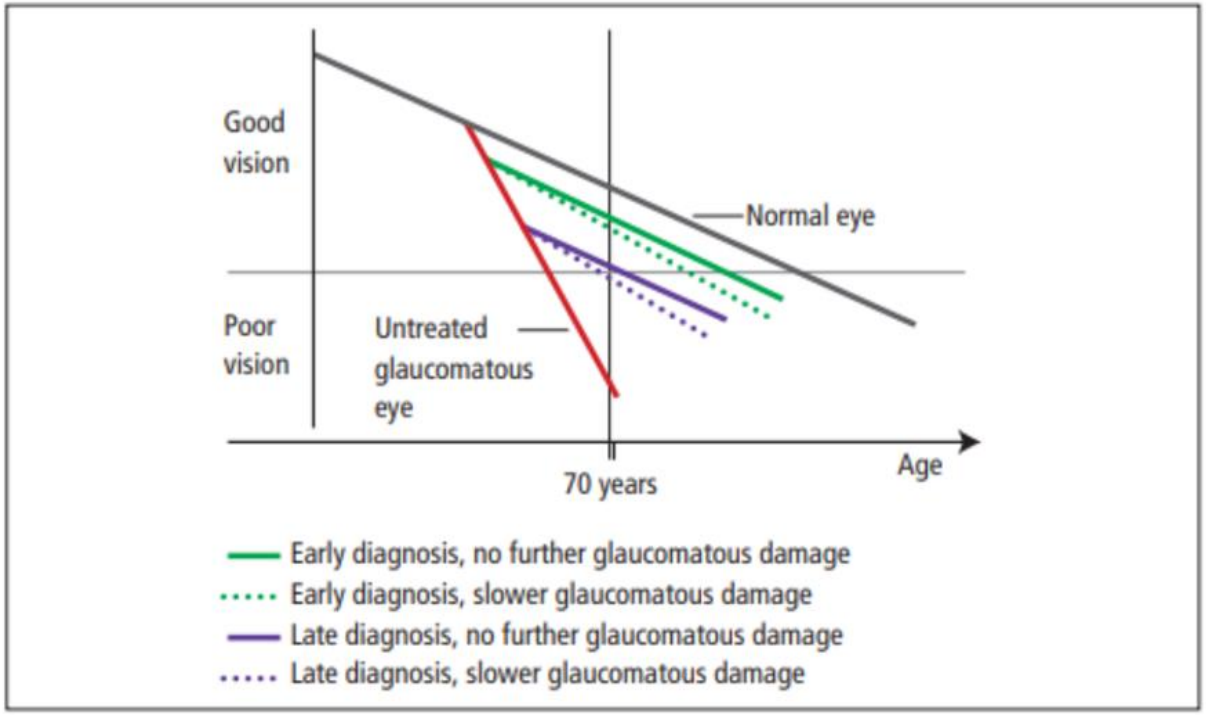


Figure 10.10 All eyes suffer a gradual loss of neurones with aging but death normally precedes a visually significant decline. In glaucoma this loss is speeded up and visually significant loss may occur during life (red line). Early diagnosis of the condition with lowering of intraocular pressure results in future age-related neuronal loss only (green line parallel to the normal eye). Even if there is some continued glaucomatous damage the rate is slowed and the patient is unlikely to suffer visual loss during their lifetime (interrupted green line). If the diagnosis is made late (purple lines), arresting the glaucoma completely may still result in visual loss during the patient's lifetime. This emphasizes the need for early diagnosis.

History

- Usually asymptomatic because the rise in IOP is slow and chronic
- Patients will present with progressive loss of vision or Visual field defects .
- age : after 40
- Family history: disease type and course in family
- past ophthalmic history
- medical and drug history

History

- CC:

1. Visual blurring
2. Headache
3. Colored haloes rings around lights
4. Others

Examination

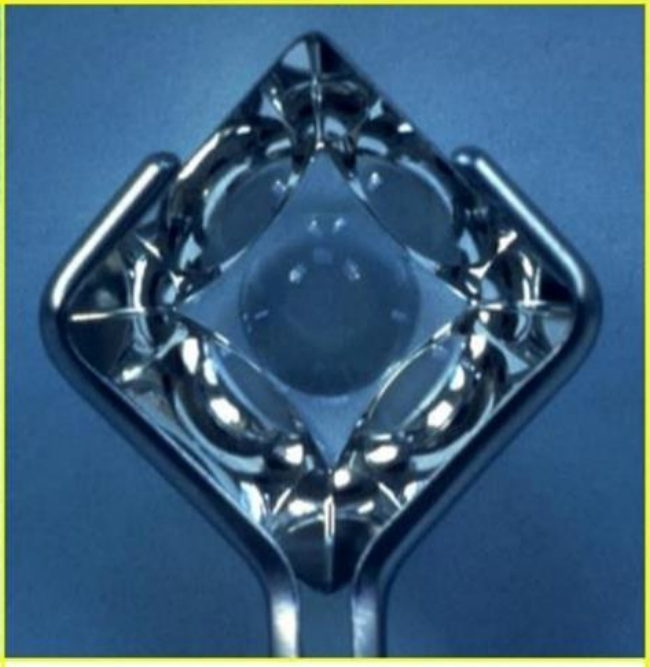
- Decreased vision .
- Red eye
- Cloudy cornea
- Shallow Anterior chamber
- Oval fixed mid dilated pupil
- Cells and flare in AC
- Iris bowing
- Hyperemic optic disc
- High IOP

Examination

1. Visual acuity
2. IOP measurement
3. Anterior segment examination by slit lamp
4. Fundoscopy to check optic nerve
5. Visual field

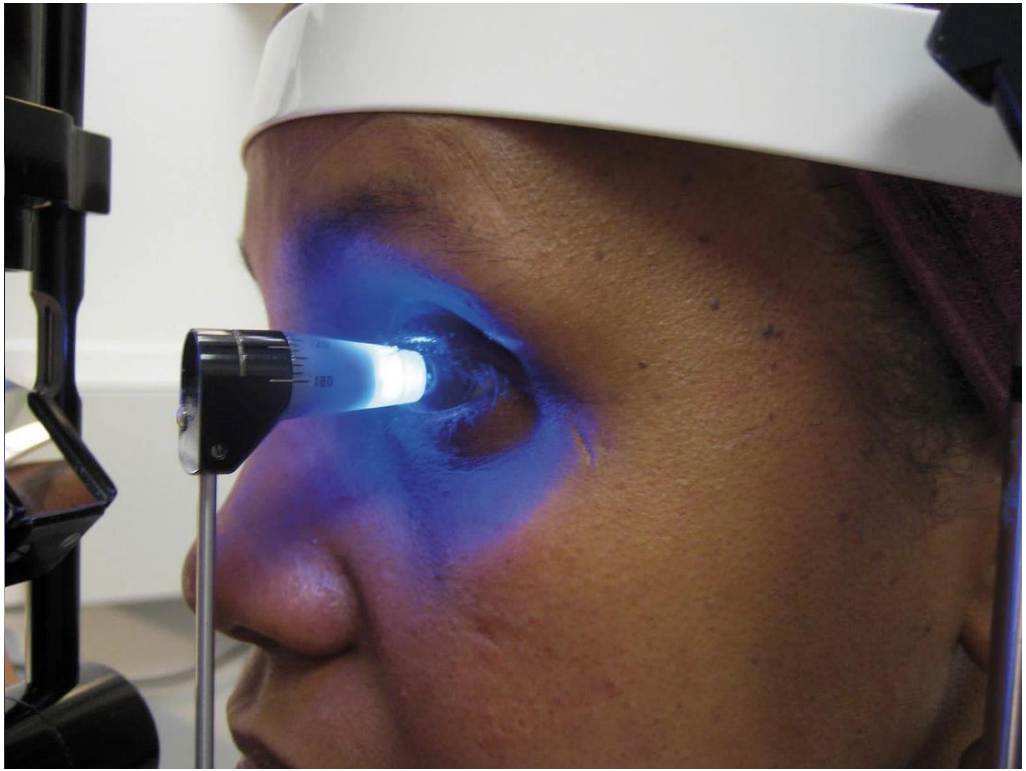
Goniolenses

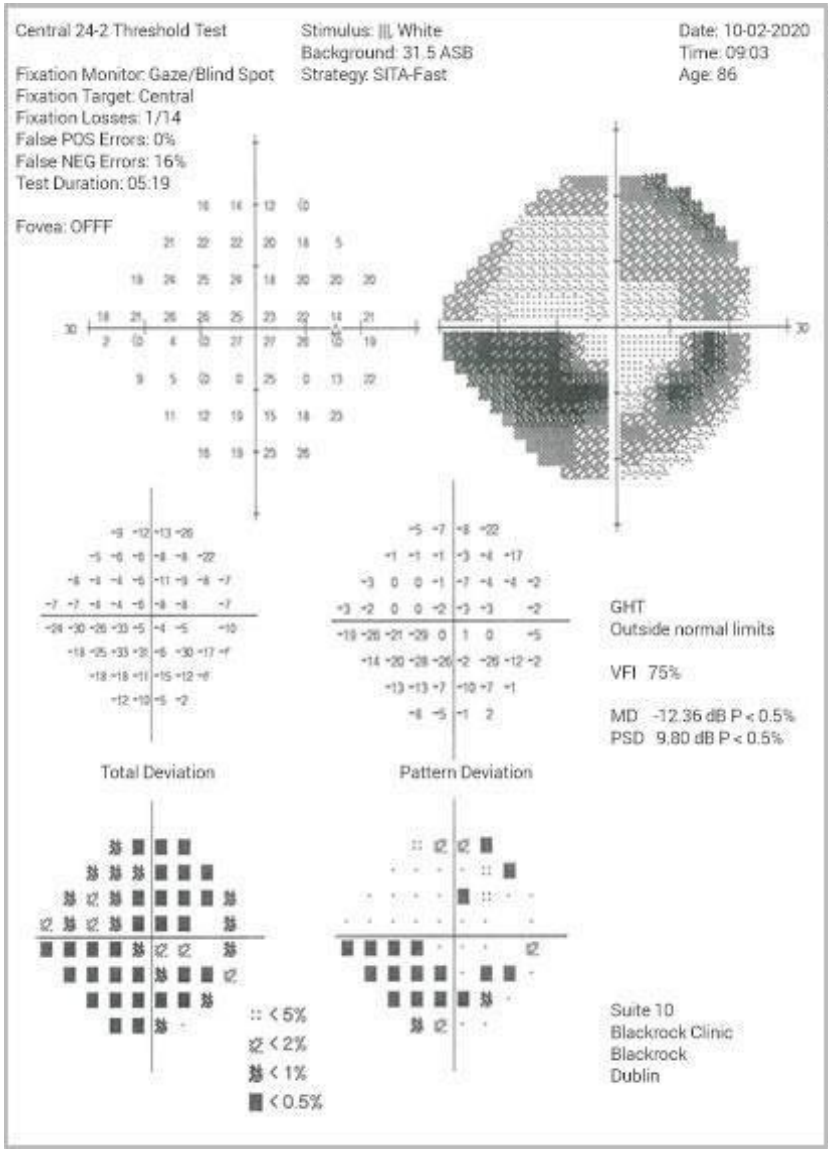
Goldmann S Zeiss



- Single or triple mirror
- Contact surface diameter 12 mm
- Coupling substance required
- Suitable for ALT
- Not suitable for indentation gonioscopy

- Four mirror
- Contact surface diameter 9 mm
- Coupling substance not required
- Not suitable for ALT
- Suitable for indentation gonioscopy




Central 24-2 Threshold Test
Stimulus: III White
Background: 31.5 ASB
Strategy: SITA-Fast
Date: 10-02-2020
Time: 09:03
Age: 86
Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 1/14
False POS Errors: 0%
False NEG Errors: 16%
Test Duration: 05:19
Fovea: OFFF
GHT
Outside normal limits
VFI 75%
MD -12.36 dB P < 0.5%
PSD 9.80 dB P < 0.5%
Total Deviation
Pattern Deviation
:: < 5%
< 2%
< 1%
< 0.5%
Suite 10
Blackrock Clinic
Blackrock
Dublin

KEY POINTS

- Glaucoma is an optic neuropathy caused by an elevation of intraocular pressure.
- Glaucomatous optic neuropathy is distinguished from that due to other causes by an enlarged optic cup.
- Primary glaucoma is classified according to whether the trabecular meshwork is obstructed by the peripheral iris (angle closure) or not (open angle glaucoma).
- Treatment of glaucoma relies on lowering ocular pressure to reduce or prevent further visual damage.
- Ocular pressure can be reduced with topical and systemic medications, laser treatment and surgery.
- Beware patients who are acutely ill, with a red eye; they may have acute angle closure glaucoma.

PAEDIATRIC OPHTHALMOLOGY

- Congenital glaucoma may present with an enlarged eye and epiphora.