GLAUCOMA

Dr Suresh Kumar
Associate Professor
Department of Ophthalmology
GMCH-32

What is Glaucoma?

• Glaucoma is a progressive optic neuropathy with characteristic appearance of the optic disc and specific pattern of visual field defects, irrespective of IOP level.

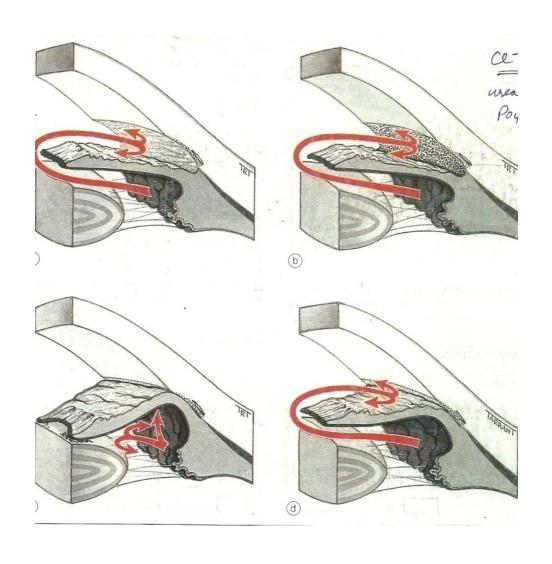
Physiology of aqueous production

CiliaryBody: Ant Pars plicata (2mm wide)
Post Pars plana (4mm wide)

Aqueous humor is actively secreated by non pigmented epithelium of ciliary processes

Trabecular Meshwork: Uveal meshwork
Corneoscleral meshwork
Endothelial(juxtacanalicular)meshwok

Pathogenesis of Sec. Glaucoma



Outflow of aqueous

 Posterior chamber - via pupil - Anterior chamber - exits eye by 2 routes :

- Trabecular (conventional) route: 90%

- Uveoscleral (unconventional) route: 10%

Factors determining IOP

Rate of aqueous secretion

 Rate of aqueous outflow - difference between the IOP and episcleral venous pressure

Normal range of IOP 11-21mm Hg

Investigations

- Visual acuity and refractive state
- Slit-lamp biomicroscopy (optic disc; 90D)
- Goldmann Applanation tonometry
- Gonioscopy
- Perimetry
- OCT

Tonometry

- Indentation tonometry: Schiotz
- Applanation tonometry:
 - Goldmann
 - Perkin
 - Mackey Marg
 - Tonopen
- Non contact tonometry: Airpuff
 Pulsair 2000 Keeler

 Applaanation tonometry based on Imbert-Fick principle: For an ideal, dry, thin walled sphere, pressure inside sphere (P) equals force needed to flatten its surface(F) divided by area of flattening (A) - 3.06mm Goldmann tonometry

Angle of anterior chamber

 Formed by root of iris, anterior most part of ciliary body, scleral spur, trabecular meshwork and schwalbe line.

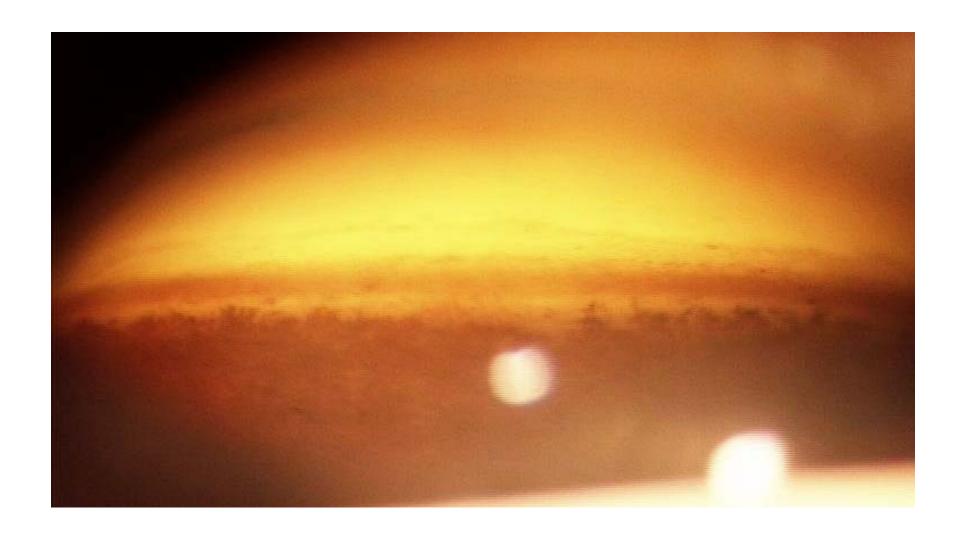
 Aqueous humor fills (0.25ml) of anterior chamber and (0.06ml) of posterior chamber

Gonioscopy

 Biomicroscopic visualisation of angle of anterior chamber using goniolens

Types of Goniolens

- Direct: Koeppe goniolens Swan - Jacob
- Indirect: Goldmann three mirror
 Zeiss goniolens
 Posner and Sussman



Angle structures

Root of iris

Ciliary body

Scleral spur

Trabecular meshwork schlemms canal Schwalbe line

Grading of angle width

SHAFFER GRADING SYSTEM

Grade 0 - closed angle(0)

Slit angle

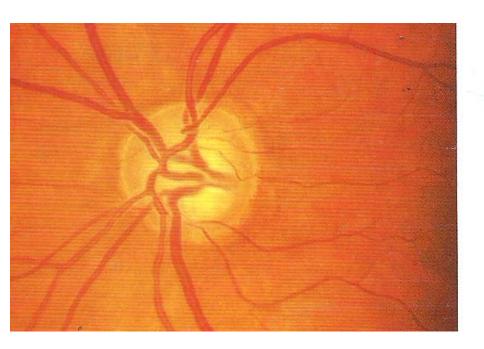
Grade 1 - Schwalbe line(10)

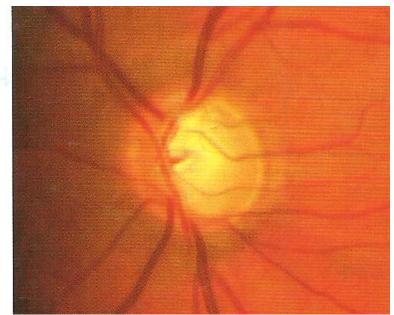
Grade 2 - Trabecular meshwork(20)

Grade 3 - Scleral spur (25-35)

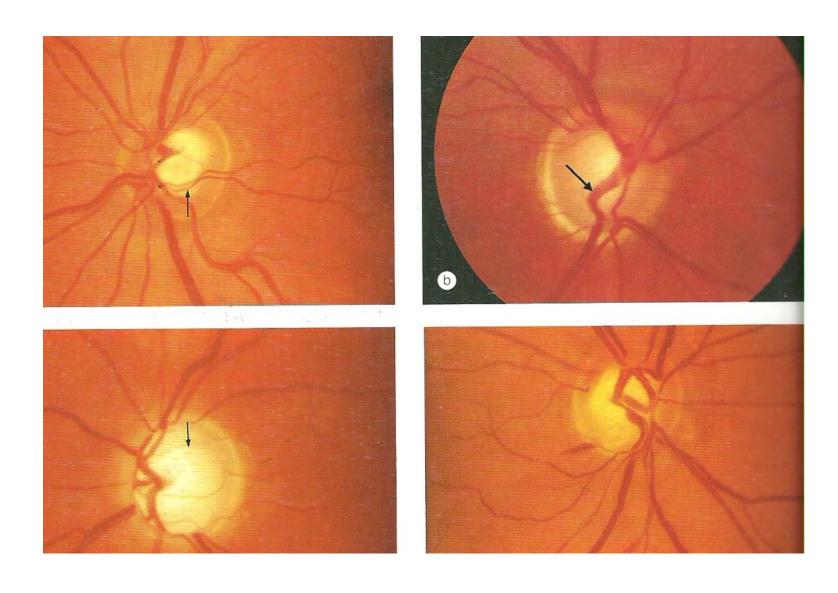
Grade 4 - Ciliary body(35-45)

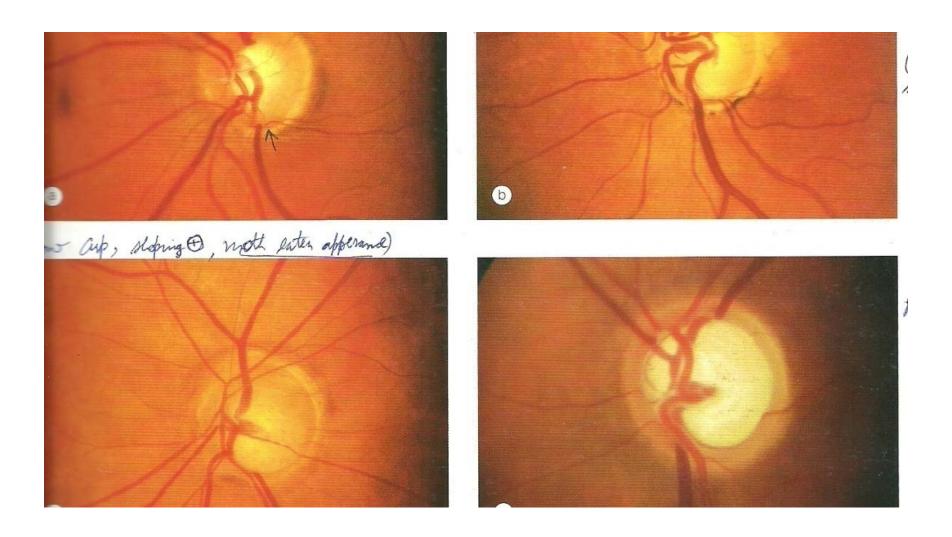


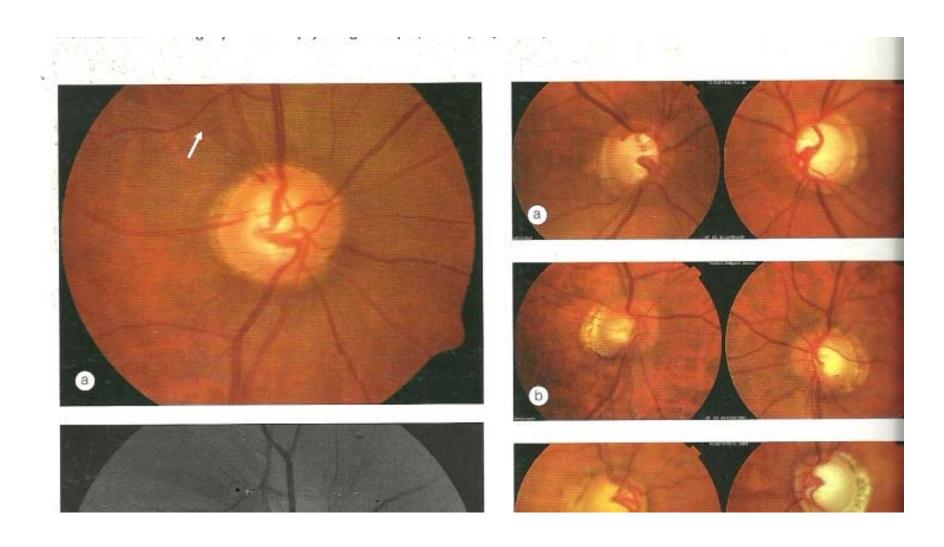




Optic disc signs







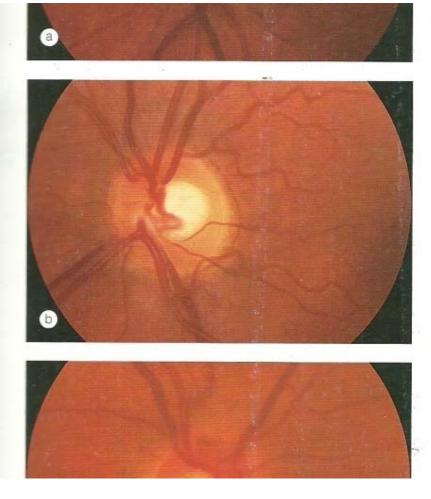




Fig. 13.6 Cup-disc ratio (C = cup; arrowheads = edge of optic disc) (Courtesy of | Salmon)

50% damage. Approximately half of all ocular hypertensive eyes that convert to POAG exhibit progression of parapapillary atrophic changes

Optic nerve head

Optic disc damage is superimposed upon physiological cupping present prior to the onset of raised IOP. If an eye with a small cup develops glaucoma (the cup will increase in size) but during the early stages its dimensions may still be smaller than that of a large physiological cup. An estimation of cup size alone is therefore of limited value in the diagnosis of early glaucoma, unless it is found to be increasing. Glaucomatous cups are usually larger than physiological cups, although a large cup is not necessarily pathological) Assessment of the thickness/symmetry and colour of the MRK

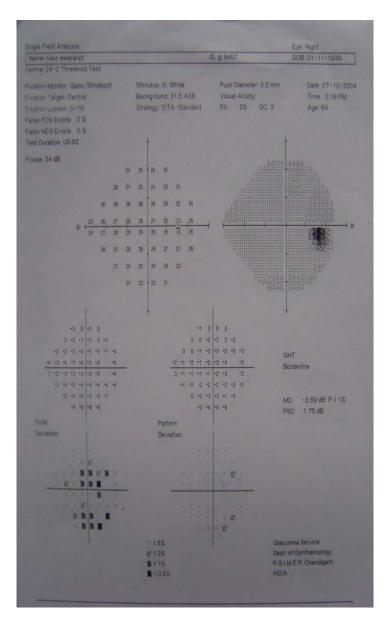
Imaging techniques

Perimertry

Heidelberg retinal tomograph

Optical cohererce tomography

Scanning laser polarimetry



PRIMARY OPEN ANGLE GLAUCOMA

POAG

- · Bilateral, not always symmetrical
- Adult onset > 40 years
- IOP > 21mmHg
- Open angle on gonioscopy
- · Glaucomatous optic nerve head damage
- Visual field loss
- Most common 1 in 100 prevelance

Risk factors and associations

- Age: older patients >40 years
- Race: black people more than white
- Family history and inheritance:
 - Multifactoral inheritance
 - Ist degree relatives and siblings(10%)
 - Myopia and Diabetes
 - Retinal diseases: CRVO, Rheg RD, RP

Steroid responsiveness

 Normal population divided into 3 Groups IOP response to 6wk course of topical betamethasone

> High responders(>30mmHg) 5% Moderate responders(22-30mmHg) 35%

Non responders (no change) 60%

Pathogensis of glaucoma

- Ischaemic theory
- Direct mechanical theory
 - -Increased ressistance to aqueous outflow in trabecular meshwork
 - -Apoptosis of retinal ganglion cells
 - -Preterminal event is calcium influx into cell body and increase in intracellular nitric oxide

Symptoms

- Insidious and asymptomatic
- Mild headache and eyeache
- Frequent change of presbyopic glasses
- Delayed dark adaptation

Signs

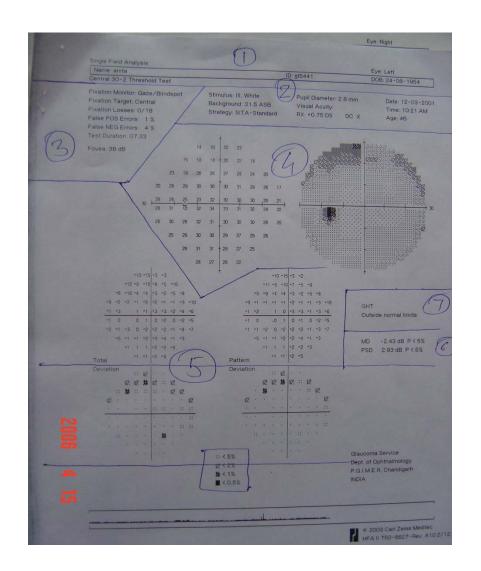
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Raised IOP
Diurnal fluctuation of IOP(DVT)
   Variation of 6-8 mmHg
   Asymmetry of 5mmHg in 2 eyes
Optic disc changes
   Vertically oval cup
   Asymmetry of cups 0.2 bn 2 eyes
   Large cup
   Splinter haemorrhages
   Pallar of NRR
   atrophy of retinal nerve fibre layer
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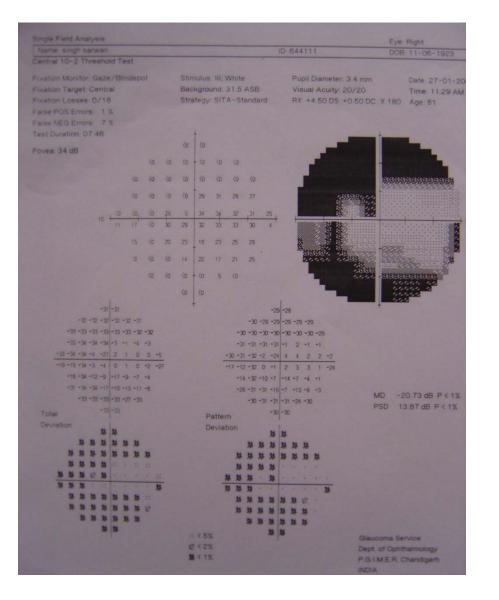
Advanced changes

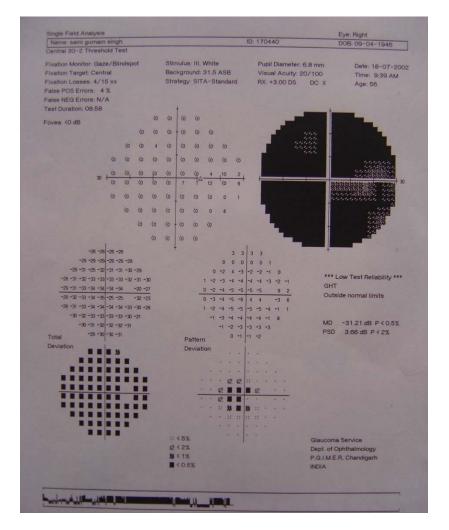
Marked cupping (0.7-0.9)
Thinning of NRR
Bayonetting sign
Lamellar dot sign
Pulsations of retinal arterioles
Glaucomatous optic atrophy

Visual field changes

Isopter contraction
Baring of blind spot
Wing shaped paracentral scotoma
Seidel's scotoma
Arcuate or Bjerrum's scotoma
Ring or double arcuate scotoma
Roenne's central nasal step
Tubular vision







Gonioscopy
 Normal open angle

Grading of glaucomatous damage

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Mild damage: early visual field defects
        MD <-6dB mild cupping
Moderate damage: definite arcuate scotoma MD <-12dB mod
                                   moderate
  thinning of NRR
Severe damage: extensive VF loss
        MD >-12dB
        Marked cupping
End-stage disease: small residual field
        Minimal residual NRR
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Treatment

- Baseline evaluation
- Achive target pressure
- Monitoring optic nerve and visual field THERAPUTIC CHOICE

Medical therapy
Argon or diode laser trabeculoplasty
Filteration surgery

Single drug therapy

Prostaglandin analogues

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Latanoprost (0.005%)
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Bimatoprost (0.03%)

Travoprost (0.004%)

Topical beta blockers

Timolol maleate (0.25,0.5%)

Betaxolol (0.5%) Carteolol (1%,2%)

Levobunolol (0.5%) Metipranolol (0.1%)

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    Alpha-2 agonist
        Brimonidine (Alphagan 0.2%)
        Apraclonidine (0.5%, 1%)
        Miotics
        Pilocarpine (1%,2%,3%,4%)
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Topical carbonic anhydrase inhibitors Dorzolamide (2%) Brinzolamide (1%)

Combined topical drugs

- Xalacom
- Travocom
- Cosopt
- Timpilo
- Combigan

Systemic carbonic anhydrase inhibiters

- Acetazolamide 250-1000mg
- Methazolamide 50-100mg
- Hyperosmotic agents
 Glycerol (50%)
 Mannitol (20%)
 Isosorbide



Argon or diode laser trabeculoplasty

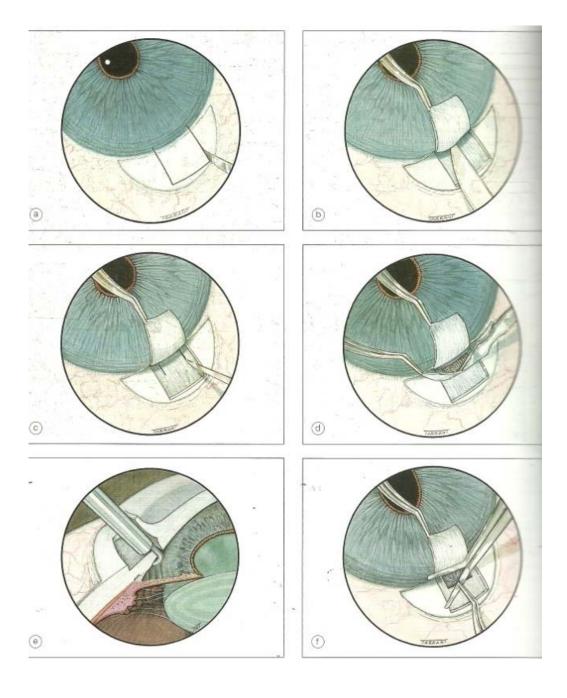
Indications

Avoidance of polypharmacy Avoidance of surgery Non compliant patient

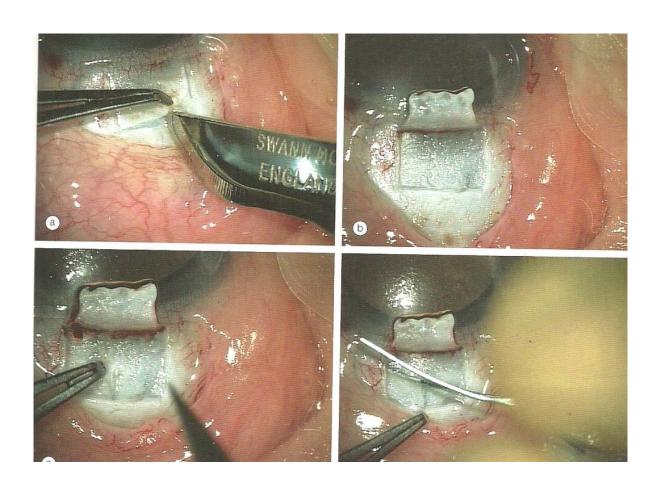
Trabeculectomy

Indications

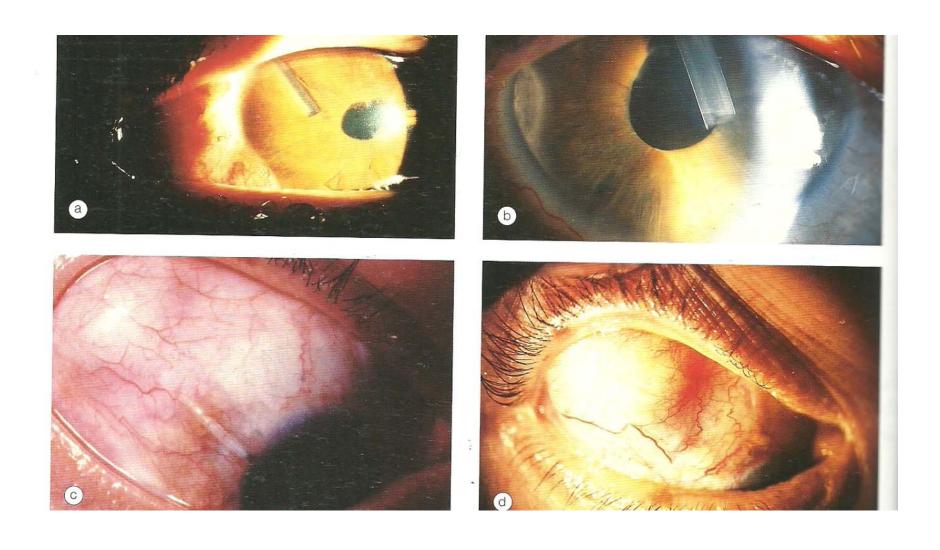
Failed medical therapy
Failed laser trabeculoplasty
Advanced disease
Unsuitability for laser therapy

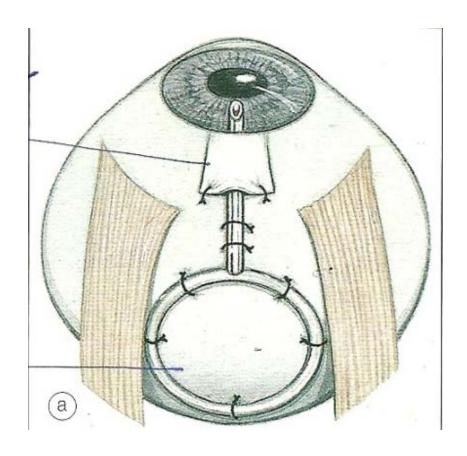


Trabeculotomy

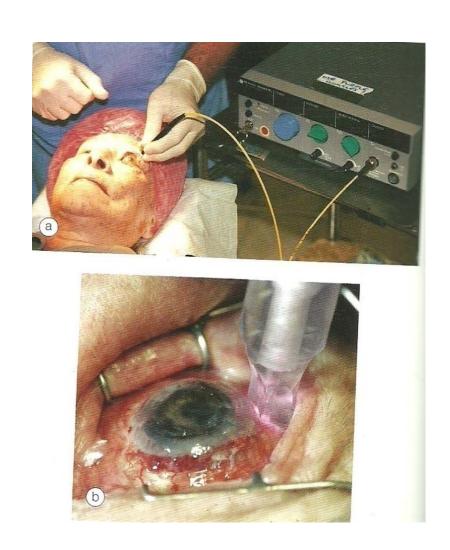


Drainage devices (Tube shunts)



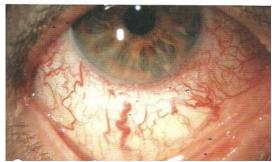


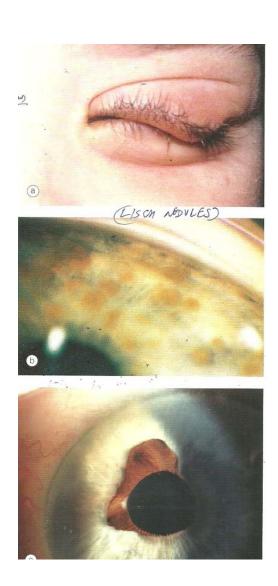
Diode Laser Cyclophotocoagulation



SWS; NF







Direct gonioscopy

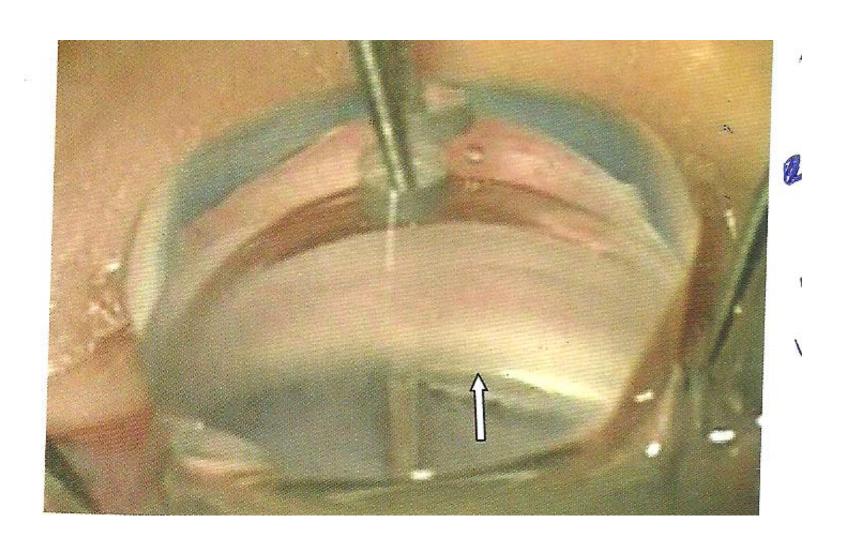




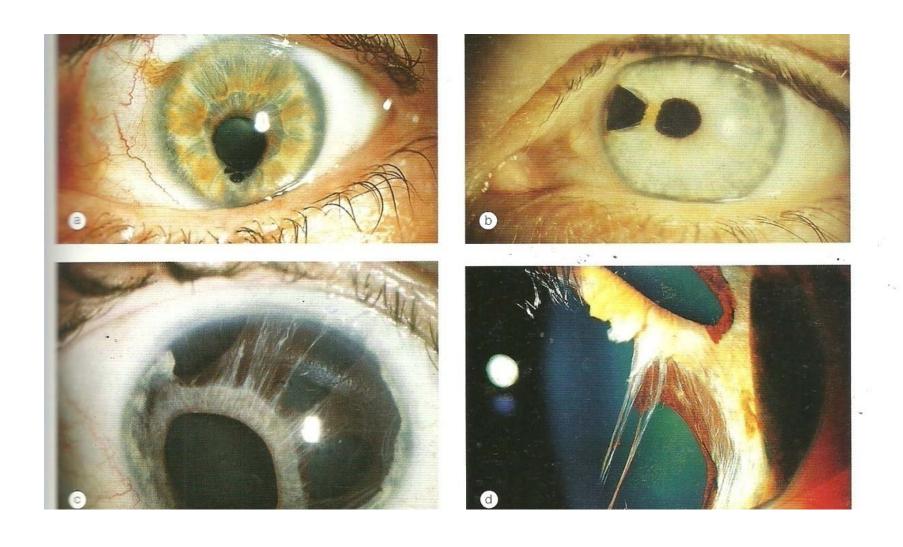




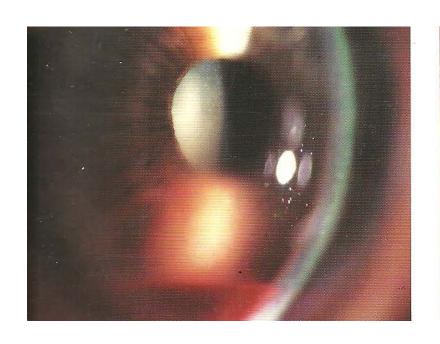
Fig. 13.41
Glaucoma in intraocular tumours. (a) Angle invasion by a solid iris melanoma; (b) melanoma cells infiltrating the trabeculum; (c) melanomalytic glaucoma; (d) angle closure by a large ciliary body melanoma (Courtesy of R Curtis – figs a and c; J Harry – fig. b)

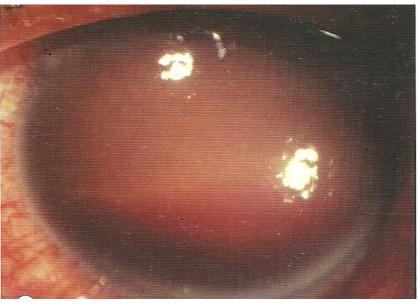


AR Anomaly



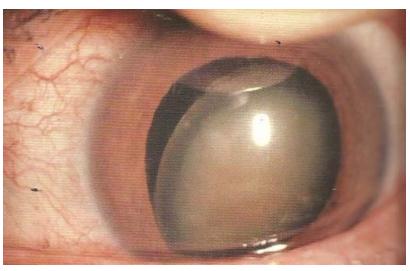
Traumatic Glaucoma: Hyphaema

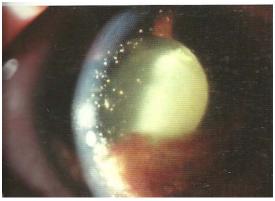




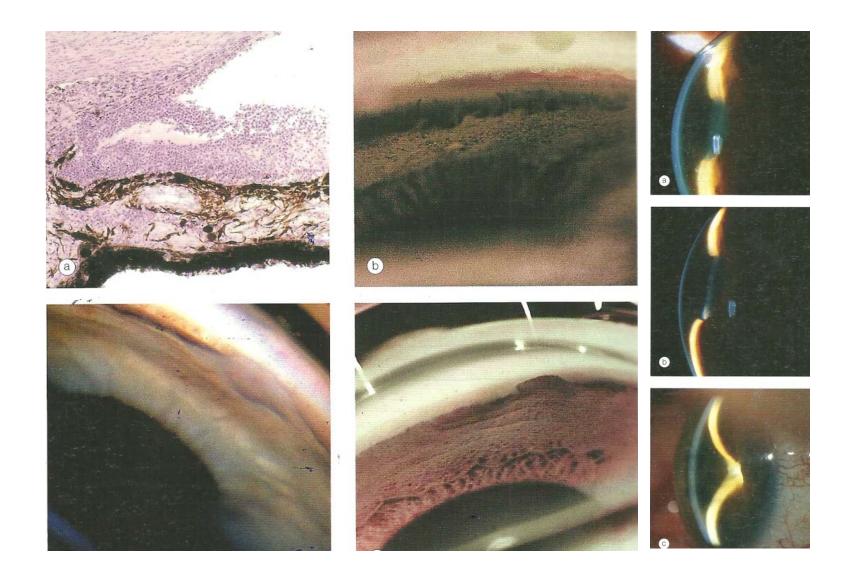
Lens Induced Glaucoma



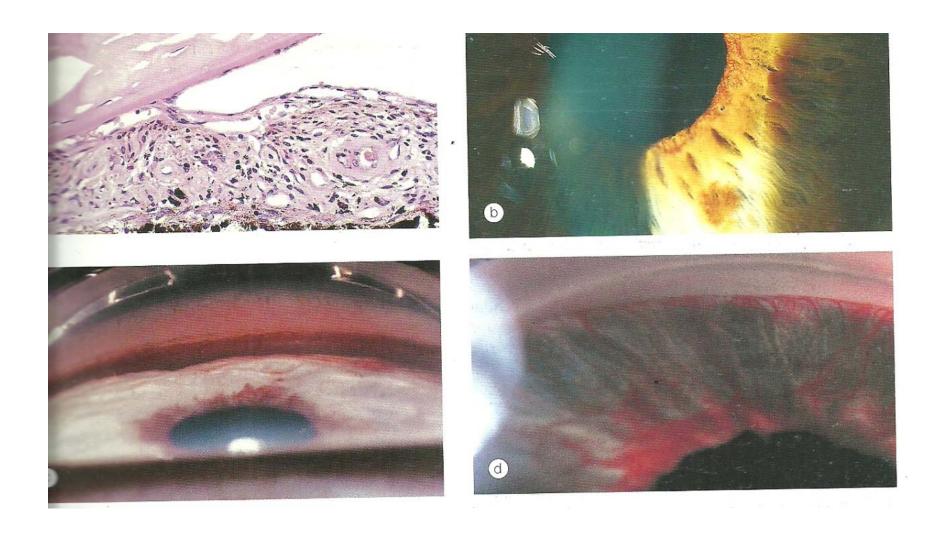




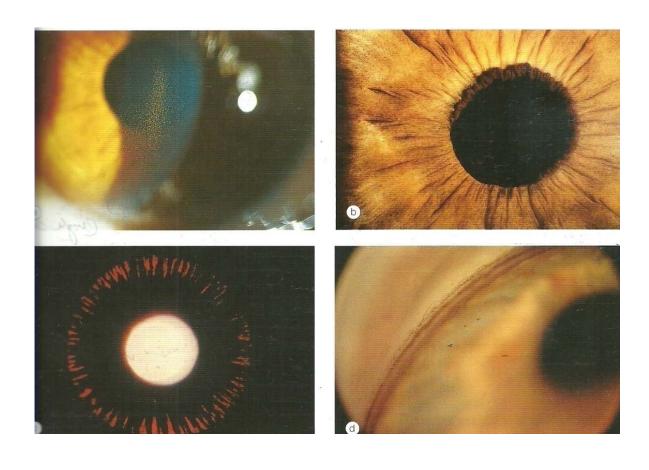
SACG



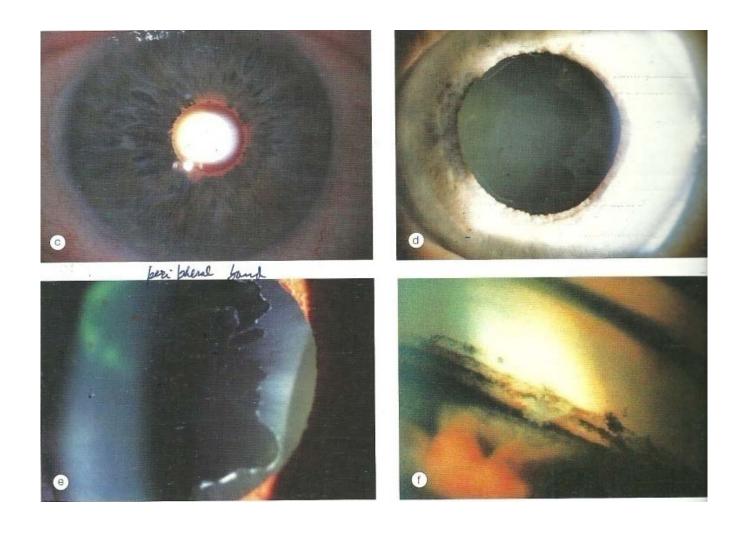
Neovascularisation



Pigment Dispersion



Pseudoexfoliation



Angle Closure Glaucoma

