CHAPTER 4 **DISEASES OF THE ANTERIOR SEGMENT OF THE EYE**

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Introduction

In the previous section concerning the sclera, cornea, conjunctiva, lids and lacrimal apparatus, we found that the common pathologic processes were those one might expect in an exposed area of the body surface, chiefly trauma and exogenous infection. The nature of the common abnormalities of the structures currently under consideration is somewhat less obvious, consisting chiefly of:

A. Anterior uveitis
B. Cataract
C. Glaucoma

As in the previous section, the following categories will be added :
D. Neoplasia
E. Miscellaneous

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A. ANTERIOR UVEITIS

Anterior uveitis includes inflammation of the iris (iritis), the ciliary body (cyclitis) or both (iridocyclitis). In practice iritis and iridocyclitis are used almost synonymously, since inflammation of either of these structures usually involves the other. Although purulent bacterial infections of these structures do occur in association with penetrating wounds, in the usual endogenous iridocyclitis no causative micro-organisms can be demonstrated and the aetiology of the inflammation is obscure. Iridocyclitis probably represents an antigen-antibody reaction of unknown aetiology.

Pathologically the iris and ciliary body show infiltration with lymphocytes and plasma cells. The uveal and episcleral blood vessels become dilated and show perivascular cuffing with round cells. The blood-aqueous barrier is broken down, with accumulation of protein and inflammatory cells in the aqueous humour. These are detectable clinically with the slit-lamp, the protein-containing aqueous taking on a slightly turbid appearance ('flare') and the cell clumps becoming visible as points of reflected light when a narrow, sharply focused beam of bright light passing through the cornea and anterior chamber is examined with a high power microscope. These findings are the most reliable evidence of iridocyclitis, differentiating it reliably from conjunctivitis

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Inflammatory cells are seen on the posterior surface of the inferior half of the cornea (keratic precipitates or K.P.), where they are deposited by the thermal circulation of aqueous. Additional signs are slight decrease in size of the pupil, due to spasm of the sphincter muscle, and injection of the episcleral vessels adjacent to the limbus (ciliary flush). The patient usually notes mild to moderate aching in the eye or frontal region, photophobia, and at times, blurring of vision.

Two main types of anterior uveitis are seen:

1. The acute (non-granulomatous) type is common and usually unilateral. Its onset is abrupt, with fairly severe pain, redness, and photophobia. Spontaneous remissions are common and the condition is often recurrent. The prognosis for vision, with treatment, is generally good. The characteristic inflammatory cells are lymphocytes and plasma cells, seen as punctate KP with the slit lamp. The aetiology of acute iritis is often not discovered. However, iritis can be associated with ankylosing spondylitis, Reiter's disease, psoriasis, Still's Disease and toxoplasmosis infection.

2. The chronic (granulomatous) type is less common but frequently bilateral. It has a more insidious onset, less pronounced ciliary flush, a more intractable course, and is more likely to produce permanent impairment of vision. Characteristic inflammatory cells are epithelioid cells, which occur in nests surrounded by lymphocytes. The KP are larger and 'greasy' looking ('mutton-fat KP'). Chronic granulomatous uveitis may be caused by tuberculosis, leprosy, sarcoidosis and juvenile rheumatoid arthritis. Cataract and glaucoma frequently occur as complications of granulomatous iridocyclitis.

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A special type of granulomatous uveitis, 'sympathetic ophthalmia', occasionally follows penetrating wounds of the eye. It usually involves the choroid in addition to the iris and ciliary body. The uninjured eye becomes inflamed also, both eyes running a chronic intractable course not infrequently leading to blindness. The aetiology is obscure, perhaps being related to an abnormal immune response to uveal pigment. Inflammation in the second, uninjured (sympathising) eye can be prevented by early enucleation of the injured eye. Enucleation of severely injured eyes with little hope of useful vision is usually carried out to avoid the risk of sympathetic ophthalmia. This is particularly urgent if the injured eye shows persistent inflammation.

Inflammation of the iris may cause the pigment epithelium of the iris to become adherent to the anterior surface of the lens at one or more points, forming posterior synechiae. If the entire pupillary margin becomes adherent, impeding the free flow of aqueous humour through the pupil, the pupil is said to be secluded.

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Iris bombé or ballooning forward of the iris from increased pressure in the posterior chamber may develop. The anterior edge of the iris is pushed against the trabecular meshwork, impeding aqueous outflow and thereby causing elevation of intraocular pressure. Adhesions between the iris and the peripheral cornea or trabecular meshwork are called peripheral anterior synechiae.

The treatment of iridocyclitis is non-specific if no definite aetiology is found for which treatment is available. Topical corticosteroids are used to reduce the inflammatory response and a mydriatic is used to relax the iris sphincter and put the ciliary muscle at rest. The resultant pupillary dilatation helps to prevent the development of posterior synechiae. Complications such as secondary glaucoma, cataract, corneal oedema and choroiditis all need specific attention.

B. CATARACT

Although cataract was correctly described as an opacification of the lens by an Indian surgeon, Susruta, many centuries before the birth of Christ, in western history cataract was considered, until the middle of the 17th century, to be an opaque humour that flowed down into the space between the iris and the lens. (The lens was considered to lie in the centre of the eye and to be the seat of vision). This incorrect notion is still alive in the minds of many patients, who require, prior to cataract extraction, a very careful explanation of the true nature of cataract, the impossibility of removal of the opacity without removing the lens, and the optical disadvantages of aphakia (absence of the lens), if disappointment is to be avoided post-operatively.

Types of cataract:

1. Nuclear cataract, or nuclear sclerosis is an exaggeration of the physiological process of increasing rigidity of the lens. With increasing sclerosis, the density (refractive index) of the lens nucleus increases. The increased refractive power produces a lenticular myopia which is initially correctable with glasses. Eventually diminution in acuity occurs as the sclerosis progresses.

2. Cortical cataract. Cortical fibres appear to be more easily damaged than those of the nucleus. This presumably is due to the accumulation of products of defective lens metabolism. The fibres lose fluid and shrink, the fluid accumulating between the fibres causing vacuoles and clefts. Eventually, often years later, coagulation of the lens proteins occurs with loss of transparency. Typically, cortical opacities assume a spoke-like configuration beginning near the lens equator. Not infrequently, peripheral cortical opacities become very extensive, obstructing adequate examination of the peripheral retina, while a small central area remains clear, allowing retention of nearly normal vision. When opacification of the central area occurs, profound visual loss results. The typical history given by patients with cataracts is gradual, painless, progressive loss of vision.

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3. Posterior subcapsular cataract. Under the influence of certain stimuli, eg. steroids and trauma, some of the lens epithelial cells at the equator migrate posteriorly beneath the capsule to the posterior pole of the lens. The result is a coarse, granular, rosette-shaped cataract in the axial portion of the posterior lens.

Stages of cataract formation:

1. Incipient
Myopia may occur due to the increased refractive power of the lens.

2. Immature
Typical complaints include gradual, painless loss of vision in bright light, 'haloes' seen around lights, and uniocular diplopia. This stage may last from a few months to many years.

Nuclear and posterior subcapsular cataracts never become 'mature' so that all of the lens is opaque, but cortical cataracts may evolve further, passing through stages of 'intumescence', 'maturity' and 'hypermaturity'.

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As the lens approaches maturity, it has a tendency to imbibe excess water and to become intumescent (swollen). As the lens swells, it pushes the iris forward and the anterior chamber becomes shallow.

3. Mature
When the lens is completely opaque the cataract is 'mature'.

4. Hypermature
The opaque cortical proteins begin to liquefy.

Causes of cataract:

1. Senile cataract
This is the most common form of cataract. Asymptomatic peripheral lens opacities occur in 65% of people aged 51-60 years. Fortunately, opacities sufficient to cause visual symptoms are much less common, but senile cataract is probably the commonest cause of impaired vision in the so-called developed countries.

2. Metabolic cataract

- Diabetes mellitus
- Hypoparathyroidism - produces primary cortical opacities which do not progress if the metabolic imbalance is corrected.
- Hurler's disease. (A mucopolysaccharide storage disease).
- Galactosaemia - produces cortical opacities which are reversible in the early stages. Urine of neonates with cataracts should be tested for reducing substances.

3. Cataracts associated with systemic disorders

- Mongolism - gives tiny punctate opacities in cortex
- Dystrophia myotonica
- Dermatitides (e.g. atrophic dermatitis, psoriasis, eczema.)

4. Cataracts complicata - associated with other ocular disorders

- High myopia (more than 8-10 dioptres).
- Glaucoma, especially after glaucoma surgery.
- Retinitis pigmentosa.
- Chronic uveitis
- Chronic retinal detachment
- Intraocular tumour e.g. malignant melanoma

5. Drug-induced cataract

POST SUBCAPSULAR

- Steroids - related to the dose and the time over which drug is administered. Produce posterior subcapsular cataracts in the axial centre of the lens.

ANT SUBCAPSULAR

- Phospholine iodide - an antiglaucoma drug which may cause anterior subcapsular opacities.

6. Traumatic cataract

- Radiation. Frequently a complication of x-ray therapy around the eye.
- Electricity - lightning
- Blunt trauma
- Penetrating injuries

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7. Congenital cataract

- Autosomal dominant cataract is the most commonly occurring congenital cataract.
- Rubella

NUCLEAR

  - lenses may be totally opaque with fluid cortex at birth or may show only nuclear cataract.
  - frequently associated with congenital glaucoma, microphthalmos, congenital nystagmus, rubella pigmentary retinopathy, and other systemic malformations.

- There are many other types of congenital cataracts which will not be considered here. The majority are genetically determined e.g. trisomy 13-15.

Treatment of cataract

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The treatment of cataract is surgical removal of the lens.

Indications for cataract extraction in an adult are:

1. Visual - The most important criterion. Extraction is indicated if the patient is unable to function adequately because of cataract formation.

   A 50 year old business executive will be greatly handicapped by a distance vision of 6/12 and a near vision of only headlines, even with his bifocals.

By contrast, an 80 year old woman with 6/18 vision for distance and near will have sufficient vision to watch TV, to knit and shop. In the former patient cataract extraction may be indicated, while in the latter patient it may be better deferred. Even when relatively good visual acuity is maintained, there may be significant 'glare' symptoms caused by disorganised diffraction patterns arising from irregular lens opacities.

The optical correction of aphakia is usually managed by the implantation of a perspex intraocular lens at the time of surgery. This has become a routine procedure in all age groups except children. It has allowed much easier and quicker visual rehabilitation after cataract surgery.

Intraocular lenses most closely resemble the optics of the physiological lens. Previously, aphakia was most commonly corrected by the use of 'aphakic' (highly positive) spectacles. These were difficult to tolerate due to their weight, their limited visual fields, and peripheral distortion. In adults having surgery for a unilateral cataract, an intraocular lens allows binocularity, whereas spectacle correction of the aphakia would not allow binocularity, on account of the difference in size of the retinal image between the phakic eye and the aphakic eye. This difference can be as great as 30% when the aphakic eye is corrected with a spectacle lens making fusion of the two images impossible. Alternatively a contact lens can be used which gives a difference in image size of 3% allowing fusion of the two images.

2. Medical - The presence of a cataract may be causing an adverse effect on the eye. This may be a direct effect as in phacolytic glaucoma or angle closure glaucoma, or an indirect effect by preventing adequate visualisation of the fundus as in cases of diabetic retinopathy or retinal detachment.

3. Cosmetic - This is a rare indication if leucocoria (white pupil) is distressing.

Indications for cataract extraction in congenital cataracts:

Congenital cataract surgery is indicated whenever the cataracts are bilateral. Unilateral congenital cataract responds poorly to surgery and the eye often is or becomes amblyopic. There is a high incidence of retinal detachment occurring 15 to 30 years after operation for congenital cataract.

TECHNIQUES OF CATARACT EXTRACTION

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1. IN ADULTS

The most common method in use is the extracapsular cataract extraction. A circular opening is made in the anterior capsule of the lens and the 'nucleus' of the lens is expressed from the eye; the remaining cortical fibres are peeled off the posterior capsule by gentle suction leaving an intact capsular bag into which the posterior chamber intraocular lens can be inserted.

In the previously common intracapsular method of extraction the lens and capsule are removed in total, having dissolved the zonule with the enzyme alpha chymotrypsin. An anterior chamber intraocular lens can then be implanted with its feet embedded in the angle between the cornea and the iris.

The extracapsular method is safe and produces excellent results. Post-operative opacification of the posterior capsule occurs in 10% of eyes in 3-5 years. This can be overcome by using a YAG laser to create an opening in the thickened membrane (capsulotomy).

2. IN CHILDREN

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Because of the tendency of the vitreous to be attached to the posterior capsule of the lens and the greater strength of the zonule in the child, intracapsular cataract extraction is not performed.

These cataracts are soft and can be removed with a fine suction cutting needle which is introduced within the cataract via a stab incision at the corneal limbus. This technique is called lensectomy.

Dislocation of the lens

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Dislocation of the lens may result from trauma or congenital defects in the zonule. Marfan's syndrome is the most commonly associated condition. Homocystinuria is associated with subluxated (partially dislocated) lenses. This defect of amino acid metabolism was first recognised in Northern Ireland. Spontaneous dislocation may occur when the lens has developed a hypermature cataract. Partial dislocation often remains stable for many years, but frequently induces considerable astigmatism. A completely dislocated lens may partially enter the anterior chamber, blocking the flow of aqueous through the pupil and leading to glaucoma by obstructing the forward flow of aqueous into the anterior chamber. If the lens completely dislocates into the anterior chamber, an acute closed angle glaucoma results.

C. GLAUCOMA

The word 'glaucoma' appeared in the writings of Hippocrates describing blindness coming on in advancing years, associated with a greenish or bluish appearance of the pupil. The association of glaucoma with increased intraocular pressure was not known until the 18th century. The complete clinical picture of glaucoma consists of increased intraocular pressure, excavation and degeneration of the optic disc and typical nerve fibre bundle damage, producing arcuate defects in the field of vision.

There are four general categories of glaucoma:

1. Primary angle closure glaucoma
2. Primary open angle glaucoma
3. Congenital glaucoma
4. Secondary glaucoma

Primary glaucomas result from an obstruction to aqueous outflow at the angle of the anterior chamber resulting in elevation of intraocular pressure. Both eyes are usually involved although the disease in one eye may precede that in the other by several years.

1. Primary angle closure glaucoma

a) Mechanism

This condition is dependent on an anatomically narrow anterior chamber angle which can best be appreciated clinically by gonioscopic examination. Hypermetropic (farsighted) eyes are especially prone to angle closure glaucoma. With increasing age, there is a slight increase in size of the lens and in small eyes, the already narrow chamber angle becomes increasingly narrow. When the anterior chamber is shallow and the angle is narrow, pupillary dilation either physiological e.g. watching TV in dim light, or dilatation with mydriatic eye drops, may lead to mechanical obstruction of the trabecular meshwork by the iris, with subsequent rise in intraocular pressure. This is called acute angle closure glaucoma.

Although mechanical obstruction of the angle by iris with pupillary dilatation is a factor in this type of glaucoma, a relative pupillary block is equally important. With the lens situated more anteriorly than normal, the area of the posterior surface of the iris that is in contact with the anterior lens capsule around the pupil is greater than normal, resulting in an increased resistance to the forward

flow of aqueous through to the pupil. This is termed 'pupillary block'. The pressure in the posterior chamber builds up in relation to the pressure in the anterior chamber and the periphery of the iris is ballooned forward as in iris bombé. This narrows the anterior chamber angle further, finally obstructing it altogether when the anterior surface of the iris comes into contact with the trabecular meshwork.

b) Clinical picture

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This is a medical emergency. It most commonly affects middle-aged or elderly patients.

Symptoms:

The eye is red, painful and vision is markedly reduced. Nausea, vomiting and prostration may occur and can mimic gastrointestinal disease.

Signs:

Visual acuity is reduced. The eye shows marked ciliary injection and conjunctival hyperaemia with corneal oedema. The intraocular pressure is markedly raised (>50 mm Hg) and the eye feels stony hard. The pupil is mid-dilated, unreactive and often vertically elongated.

c) Treatment of angle closure glaucoma

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The intraocular pressure is initially lowered using intravenous acetazolamide (Diamox) and, if necessary, an osmotic agent such as intravenous mannitol or oral glycerol. Once the pressure has been reduced, 4% Pilocarpine drops are instilled frequently to constrict the pupil and draw the iris away from the trabecular meshwork, which reopens the angle. Pilocarpine will not work until the pressure has been reduced to about 35 mm Hg, which will reverse the iris ischaemia and allow the constrictor muscle to recover. Because the other eye is also at risk it is essential to constrict the other pupil with pilocarpine.

Once the eye has been stabilised on Pilocarpine drops, the definitive treatment is peripheral iridectomy. This prevents relative pupil block and may either be done surgically or by laser. Iridectomy is also done in the fellow eye because the eventual risk of acute angle closure in this eye, if untreated, is very high.

If the angle closure is not reversed and the pressure reduced rapidly, vision will be permanently damaged. Extended closure of the angle leads to adhesions called peripheral anterior synechiae which prevent its reopening and in this case trabeculectomy will be required to maintain normal pressures.

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2. Primary open angle glaucoma

a) Mechanism

This is the most common form of glaucoma, affecting 0.5% of the population over 40 years of age. Chronic elevation of intraocular pressure is painless, and usually ranges from 20 to 40 mm Hg. Most commonly the patient has no symptoms, usually becoming aware of field loss only very late, often when most of the field in at least one eye has been extinguished.

The angle is open and appears normal to gonioscopic and histological examination. The facility of aqueous outflow is decreased due to an abnormality in the outflow channels. Evidence points to the portion of trabecular meshwork closest to Schlemm's canal being defective. The nerve fibres at the optic disc are nourished by capillaries arising largely from the short posterior ciliary arteries. These capillaries give the normal disc its healthy pink colour. If the intraocular pressure is elevated, blood flow in these capillaries is impaired, the disc becomes ischaemic and the nerve fibres atrophy.

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B
(stereo pair)

Direct mechanical trauma is also possible. The atrophy causes expansion of the normal physiological cup and pallor of the disc. Atrophy of the nerve fibres produces a characteristic loss of field of vision called a nerve fibre bundle defect. This typical field defect arises at the blind spot and extends in an arc above or below (or both) the fixation point ending in a sharp horizontal line nasally (nasal step). This gradually progresses until only a small central island remains. Eventually this also is lost. This progression is often so slow and insidious in chronic open angle glaucoma that the patient is unaware of the visual field loss until very late, which is why chronic simple glaucoma must be detected in early stages and treatment instituted. Testing of Intraocular

pressure should be a routine part of every general physical examination.

Unfortunately, the retina, like central nervous tissue, demonstrates no regeneration once damaged. It follows that once visual field has been lost, it cannot be recovered. All treatment can possibly do is prevent further field loss.

b) Treatment of open angle glaucoma

The aim of treatment is to lower the intraocular pressure and prevent further damage. Treatment is required indefinitely but cannot reverse existing field loss. Initial treatment is medical, by the use of pressure-lowering eye drops which may be used singly or in combinations; when these are inadequate to control the pressure or prevent the progression of field loss, trabeculectomy is performed.

Timolol or a similar B-blocking eye drop is the usual first line of treatment. It is used twice daily and will reduce intraocular pressure by up to 10 mm Hg. It is contraindicated in patients susceptible to wheezing or heart block.

If this is insufficient then topical adrenaline (Eppy, Simplene) or a precursor (Propine) has an additive effect in reducing intraocular pressure. It may be used alone in patients who cannot use Timolol, but is less effective. Because it dilates the pupil it can precipitate angle closure in eyes with narrow angles. It is also used twice a day.

Pilocarpine 1% to 4% in drop form may be used in combination with the above or alone. It is a parasympathomimetic drug which constricts the pupil and causes contraction of the ciliary muscle. This is thought to pull on the scleral spur and open up the trabecular meshwork. It has to be used four times a day and the pupillary constriction and accommodation spasm may cause problems, particularly in patients who also have cataracts.

Acetazolamide (Diamox) is an oral carbonic anhydrase inhibitor which is very effective in reducing intraocular pressure by reducing aqueous secretion. It has unpleasant side-effects such as nausea and tingling of the fingers, and can produce renal calculi. It is very useful for short-term reduction of intraocular pressure, but is rarely used long term, except in patients who are unable to undergo trabeculectomy.

When medical treatment is inadequate to control intraocular pressure and prevent progression of field loss, a trabeculectomy is performed. In this operation the pressure is reduced by surgically

forming a new drainage channel from the anterior chamber to a bleb formed below the conjunctiva posterior to the superior limbus.

c) Prevention

Because chronic glaucoma is often asymptomatic and occurs in about 10% of first degree relatives of sufferers, it is important to screen those with a family history of glaucoma by examining the optic discs and measuring the intraocular pressures.

3. Congenital glaucoma

a) Mechanism

Congenital glaucoma is a condition found in infants in which there has been incomplete cleavage of the angle embryologically. Fine embryonic tissue covers the trabecular meshwork obstructing outflow of aqueous. Under the influence of increased intraocular pressure there is progressive enlargement of the infant's eye called buphthalmos which means ox eye.

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b) Clinical picture

Photophobia and excessive lacrimation are prominent symptoms. The cornea becomes hazy due to oedema.

c) Treatment

Treatment of congenital glaucoma is surgical. The obstructing tissue is cut (goniotomy) under microscopic control allowing aqueous access to the trabecular meshwork. This procedure must be done early if a high degree of success is to be achieved.

4. Secondary glaucoma

a) Mechanism

This results from obstruction of aqueous outflow secondary to:

i) Lens changes

- Intumescent cataract which swells pushing the iris forward and causing angle closure.
- Hypermature cataract in which lens material leaks through the capsule and is phagocytosed by mononuclear cells which plug up the pores in the trabecular meshwork (phacolytic glaucoma).

- subluxation or dislocation of the lens through the pupil into the anterior chamber producing pupillary block.

ii) Uveitis leading to obstruction of the trabecular pores, with cellular debris.

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iii) Rubeosis iridis - newly formed blood vessels developing on the anterior surface of the iris and continuing over the trabecular meshwork (indicating severe ocular ischaemia e.g. in diabetes mellitus).

iv) Angle recession - contusions to the eye producing separation of the ciliary body from the scleral spur. Mechanical damage to the trabecular meshwork results. Rarely glaucoma may appear after several years.

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v) Intraocular tumour - retinoblastoma and malignant melanoma of ciliary body and iris.

vi) 'Steroid responders' - two or more weeks of topical corticosteroid treatment produces marked (10mm Hg) elevation of IOP in about 4% of unselected individuals and a small but definite rise of intraocular pressure in a further 30% of such individuals. Patients with a family history of glaucoma are particularly susceptible to glaucoma following use of local corticosteroids. Glaucomatous field loss can result and pressures must therefore be measured in all patients receiving such drops for longer than two weeks.

vii) Pigment dispersion syndrome - pigment shed from the iris is deposited on the trabecular meshwork

viii) Pseudoexfoliation - a protein, similar in structure to amyloid is deposited on the trabecular meshwork.

DIFFERENTIAL DIAGNOSIS OF THE RED EYE

	ACUTE CONJUNCTIVITIS	ACUTE IRITIS	ACUTE GLAUCOMA (closed angle)	ACUTE KERATITIS
ONSET	gradual	gradual	SUDDEN	gradual
VISION	unaffected unless mucus or secretion forms on surface of cornea	slightly reduced (6/12 - 6/9)	MARKEDLY REDUCED (6/60 or less)	reduced, especially if axial
DISCHARGE	MUCOPURULENT	lacrimation	lacrimation	watery
PAIN	burning & itching foreign body sensation	moderately severe	SEVERE	SEVERE
CONSTITUTIONAL SIGNS	absent	slight	NAUSEA & VOMITING PROSTRATION	absent
INJECTION	conjunctival	CILIARY	CILIARY	CONJUNCTIVAL & CILIARY
CORNEA	clear or SPK	clear + /-keratic precipitates	OEDEMATOUS	FB abrasion or ulcer
IRIS	normal	POSTERIOR SYNECHIAE ATROPHY	CONGESTED DISPLACED FORWARDS	normal
PUPIL SIZE	normal	CONTRACTED, IRREGULAR	DILATED, OVAL	normal
PUPIL LIGHT REFLEX	normal	POOR	POOR or ABSENT	normal
AC DEPTH	normal	normal	SHALLOW	normal
AQUEOUS (SLIT LAMP)	clear	FLARE & CELLS	flare & few cells	few cells
INTRAOCULAR PRESSURE	normal	low or normal	INCREASED	normal

D. NEOPLASIA OF THE IRIS AND CILIARY BODY

1. Iris

Benign pigmented lesions of the iris are often seen.

a) Iris freckles are observed clinically as small flat (less than 2mm) yellow, brown, or black circumscribed lesions at the anterior surface of the iris stroma. They are composed of closely arranged melanocytes.

b) Iris naevi are larger benign lesions which may involve any part of the iris stroma. They are circumscribed and slightly elevated lesions. The pupil margin is usually not distorted. Naevi may undergo malignant change (malignant melanoma).

Malignant lesions of the iris are uncommon

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c) Melanomata of the iris may be removed by iridectomy with retention of good vision.

2. Ciliary body

The only common tumour of the ciliary body is malignant melanoma. They are often only visible on wide dilatation of the pupil. These tumours are more malignant than iris melanomas and frequently can only be removed by enucleation (removal of the entire eye).

E. MISCELLANEOUS

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1. Rubeosis iridis

Several pathologic conditions characterised by a disturbance of the vascular supply to the eye (e.g. central retinal vein occlusion, diabetes) lead to the formation of new vessels and thin transparent fibrous tissue on the anterior surface of the iris. This condition is called rubeosis iridis. Rubeosis may also follow long-standing uveitis and complicate intraocular tumours. The fibro-vascular membrane tends to shrink, and as it does so, part of the posterior pigment epithelium of the iris is pulled through the pupil and on to the anterior surface of the iris (ectropion uvea). The new vessels and fibrous tissue grow across the iris and over the trabecular meshwork, impairing aqueous outflow and leading to elevated intraocular pressure (glaucoma). This variety of glaucoma is difficult to treat. Often the only means is by destruction of the

ciliary body, thus decreasing aqueous production (cyclocryotherapy) using a cryoprobe at -70°C.

2. Defects in the iris

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a) Congenital coloboma - absence of a sector of iris which results from failure of closure of the fetal fissure and is thus placed inferiorly.

b) Surgical 'colobomata' (iridectomies) performed to facilitate removal of the lens during cataract surgery.

EFFECTS OF DRUGS ON THE IRIS AND CILIARY BODY

A. PARASYMPATHOLYTIC DRUGS

1. These drugs result in mydriasis (dilatation of the pupil) and cycloplegia (paralysis of the ciliary muscle).

2. Mechanism of action is competitive inhibition of acetylcholine at the receptor site.

3. Uses in diagnosis:

a) Mydriasis allows better examination of lens, vitreous and fundus.

b) Cycloplegia simplifies accurate testing of the refractive error. If accommodation is active, especially in children, refractive error will vary from moment to moment with the amount of accommodation exerted. If accommodation is temporarily paralysed by cycloplegia an accurate refraction can be performed.

4. Therapeutic uses

Mainly used in iridocyclitis. Parasympatholytics are used to put the sphincter muscle of the iris and the ciliary muscle at rest (by paralysing them). This reduces pain and has a remarkable anti-inflammatory and/or decongestant effect. Posterior synechiae are broken as the pupil margin is pressed less firmly against the lens or even becomes separated from it when the pupil is fully dilated.

Comparison of duration of action of several commonly used parasympatholytic drugs

Drug	Time to Maximum Effect	Duration of maximum effect	Patient able to read	Accommodation normal
Atropine	1-2hrs	8-48hrs	3-4 days	10-14 days
Homatropine	40mins	50mins	6hrs	36hrs
Cyclopent-olate (Mydrilate)	25mins	50mins	3hrs	18hrs
Tropicamide (Mydriacyl)	20mins	15mins	45mins	4hrs

For diagnostic purposes, Mydrilate is the drug of choice, while for therapeutic purposes, Atropine and Homatropine are sometimes preferred. For fundus examination only (when mydriasis without cycloplegia is desired), sympathomimetic drugs can be used. Phenylephrine is the most commonly used. Peak of action is at about 45 minutes and duration of maximum effect 30-45 minutes. The effects of phenylephrine can be reversed by Thymoxamine.

B. PARASYMPATHOMIMETIC DRUGS

1. These drugs result in miosis (constriction of the pupil) and cyclotonia (spasm of the ciliary muscle).

2. Mechanism of action:

   a) Choline esters - acetylcholine and its derivatives have their effect by direct chemical action on the effector muscle.

      - Acetylcholine is rapidly destroyed by cholinesterase and is very short-acting. It is used primarily to constrict the pupil immediately after cataract extraction by instilling it into the anterior chamber (Miochol).

      - Carbachol is immune to cholinesterase and is thus long-acting. It is a powerful miotic and cyclotonic drug.

   b) Anticholinesterases
   Here the mechanism of action is to potentiate the normal action of acetylcholine at the nerve endings

by inhibiting the normal hydrolysing action of the enzyme cholinesterase on acetylcholine.

- Short-acting group (Eserine, Physostigmine, Prostigmine) temporarily and reversibly inhibit cholinesterase. They have a duration of 4-8 hours.

- Long-acting group (Phospholine iodide) bind cholinesterase permanently and thus have a duration of action of 12-48 hours because new enzyme must be manufactured.

c) Pilocarpine
The mechanism of action is direct stimulation of the ciliary (and sphincter) muscle fibres. Duration of action is about 2-4 hours.

3. Therapeutic uses

a) They are employed to reduce intraocular pressure in acute angle closure glaucoma. Also used in open angle glaucoma in combination with Beta-blockers.

b) Rarely used in accommodative esotropia (convergent squint) because of their ability to stimulate accommodation without affecting convergence. Thus, the patient can see clearly for near without exerting accommodation, but N.B. Phospholine iodide may cause anterior subcapsular lens opacities.