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Current concepts

Glaucoma: diagnosis and management

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Summary

Glaucoma is the third most prevalent cause of global blindness, accounting for over 5 million blind. It is common in Western Countries; the estimated prevalence of primary open angle glaucoma rises from a total prevalence of 1.1% to approximately 3% of our population as it ages. Ethnicity affects both the risk of developing glaucoma and the outcome. It is an expensive disease both to detect and to treat. Recent scientific advances include elucidation of the genetic mechanism behind the disease and the study of haemodynamic and biochemical co-factors in the development of glaucomatous optic neuropathy, particularly in relation to the pathogenesis of normal tension glaucoma. Several new types of topical medication have recently been developed for use in glaucoma; both the impact of these therapies and their cost effectiveness remain to be evaluated.

There are widely differing regimes which effectively treat glaucoma; some ophthalmologists prefer early surgical intervention whilst others reserve surgery for relatively advanced disease. All methods of current treatment rely on the reduction of intra-ocular pressure; as yet there is no medication which has been definitively proven to be either neuroprotective or to influence favourably optic nerve perfusion. Despite this, most sufferers of glaucoma are able to lead lives of quality. Increased community awareness of glaucoma, and earlier detection of the condition, will doubtless result in decreased morbidity due to glaucoma.

Keywords: glaucoma

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Glaucoma is a major cause of visual loss. Current terminology may be confusing, and older definitions of the disease have often put undue stress on the role of raised intra-ocular pressure (IOP) in the pathogenesis of open-angle glaucoma. Recent scientific studies have revealed the situation to be more complex and have shown that other factors, both vascular and biochemical, are probably involved in the pathogenesis of glaucomatous optic neuropathy.

In glaucomatous optic neuropathy there is optic disc cupping and atrophy and apoptosis of retinal ganglion cells and their axons, and possibly other retinal elements, leading to irreversible visual field loss (figure 1). The IOP is usually elevated. Glaucoma can thus be considered a generic name for a group of diseases causing optic neuropathy (disc cupping) and visual field loss, usually but not always in the presence of raised IOP. Other factors, such as optic nerve head perfusion, are concomitantly responsible for optic neuropathy in adult glaucoma. Some authors postulate a disturbance of the mechanism of autoregulation of blood flow to the optic nerve whilst other authors describe glaucomatous optic neuropathy as an intrinsic optic neuropathy due to localised vascular disease and to other biochemical mechanisms which have yet to be fully elucidated.[1]

The most common type of glaucoma in Western societies is primary open-angle glaucoma (POAG).[2] This review will emphasise the diagnosis, morbidity and treatment of POAG.

Prevalence, ethnicity and socio-economic importance

Glaucoma is a major cause of both blindness and of lesser degrees of visual impairment. (The WHO definition of blindness is vision less than 3/60 in the better eye with best available spectacle correction.) Glaucoma and its management has an enormous impact in our society in terms of patient morbidity, loss of productivity, number of ophthalmic consultations and health costs.[2–4] Worldwide, glaucoma is reckoned to be the third leading cause of blindness with an estimated five million blind from the disease (table).[2]

Shared care of glaucoma by ophthalmologists and other health professionals within a hospital setting has been postulated by numerous learned bodies as a potential mechanism of reducing the high cost of glaucoma management.[2–4] For example, the Royal College of Ophthalmologists, as cited by Hume, stated in 1994 that "patients with POAG comprise approximately 25% of the general ophthalmic workload and half this number as currently diagnosed can be regarded as stable and suitable for a shared care scheme."[3]

The prevalence of POAG has been estimated as 1.1–3% of the Western population in various surveys. Although these surveys used differing diagnostic criteria, the inescapable conclusion is that POAG is an increasingly common disease in our ageing population.[4–9] The prevalence of POAG is thought to be higher in men than in women although some studies do not support this.[8] A recent study from the Netherlands found an overall prevalence of POAG of 1.1% (95% confidence interval (CI): 1.09–1.11). Age-specific prevalence figures increased from 0.2% (95% CI: 0.16–0.24) in the age group 55–59 years to 3.3% (95% CI: 2.57–4.04) in the age group 85–89 years. Men had a more than three times greater risk of having POAG than women (odds ratio 3.6). In 8.8% of the eyes (2.9% of patients), visual acuity was 6/60 or less due to POAG.[7]

An Australian study, the Blue Mountains Eye Study, which provided detailed age and sex-specific prevalence rates for open-angle glaucoma and ocular hypertension in an older population, found a prevalence of 3.0% for POAG. Ocular hypertension, defined as an IOP in either eye of more than 21 mmHg, without matching disc and field changes, was present in 3.7% of this population (95% CI: 3.1–4.3), but there was no significant age-related increase in prevalence of ocular hypertension.[8]

Ethnicity affects both the chance of an individual developing glaucoma and the prognosis of his or her disease. The Barbados Eye Study highlighted the public health importance of POAG in the Afro-Caribbean region and has impli-

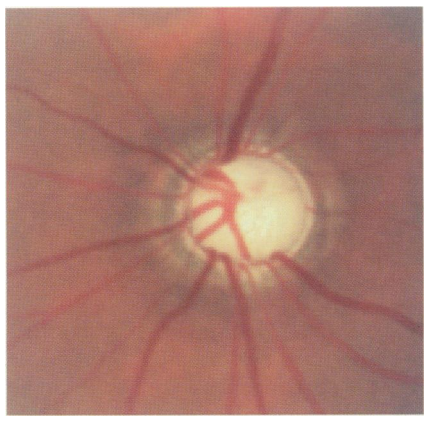


Figure 1 Advanced cupping of the optic disc due to primary open angle glaucoma. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

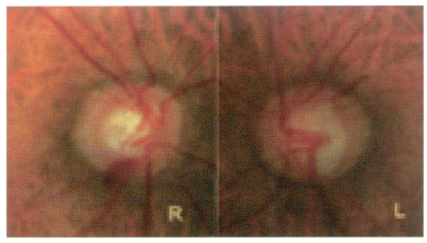


Figure 2 Optic disc cupping and asymmetry in open angle glaucoma. Note also disc haemorrhage on right optic disc. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

Optic disc changes in glaucoma

- notching of neuroretinal rim
- pallor
- splinter haemorrhage
- progressive enlargement of the disc cup
- vertical elongation of the disc cup
- asymmetry (between left and right eyes)
- nasal displacement of central retinal vessels, baring of lamina cribrosa

Box 1

Table Causes of world blindness[2]

Causes of blindness	*Number blind (millions)*	*Geographical distribution*
Cataract	16–38	world-wide
Trachoma	5.5	Asia, Africa
Glaucoma	5.0	World-wide
Diabetic retinopathy	2.5	world-wide
Xerophthalmia	1.0	Asia, Africa, Latin America
Age-related macular degeneration	1.0	Europe, North America
Oncocerciasis	0.5	West Africa, Latin America
Injuries	0.5	world-wide
Leprosy	0.25	Asia, Africa, Latin America
Others	3.0	world-wide

cations for other populations (including local populations in the UK).The prevalence of POAG by self-reported race was 7.0% in black, 3.3% in mixed-race, and 0.8% (1/133) in white or other participants. In black and mixed-race participants, the prevalence reached 12% at age 60 years and older and was higher in men (8.3%) than in women (5.7%), with an age-adjusted male:female ratio of 1.4. Among participants 50 years old or older, one in 11 had POAG, and prevalence increased to one in six at age 70 years or older.[10]

Glaucoma is considered to be under-diagnosed in the UK. Glaucoma screening of all people over the age of 40 years would be worthwhile if one were prepared to pay approximately £375 to detect a new case.[4]

Diagnosis of glaucoma

The diagnosis of glaucoma is based upon three main measurements, IOP measurement, optic disc examination and visual field testing (perimetry).[4 11] Examination of the anterior chamber angle with a gonioscopic contact lens (gonioscopy) is also important in determining the aetiology of glaucoma, for example, in differentiating between chronic angle closure glaucoma and POAG.[11 12]

INTRA-OCULAR PRESSURE (IOP)

The aqueous humour is produced by non-pigmented epithelium of the ciliary processes of the ciliary body. It flows through the pupil from the posterior chamber to the anterior chamber and leaves the eye via the trabecular meshwork, Schlemm's canal and episcleral veins.[11 13] IOP is determined by the rate of aqueous production, the rate of aqueous outflow and the episcleral venous pressure. Many factors may affect the IOP, including age, systemic blood pressure, genetic factors and topical or systemic medications.[11 13]

Factors that affect the level of recorded IOP include the time of day, season, respiration and method of measurement. Recent studies have also indicated that IOP can be lowered by regular aerobic exercise.[13]

The measurement of IOP (tonometry)

The measurement of IOP (tonometry) can be performed using one of several instruments. Commonly used devices are the Goldmann applanation tonometer (used in conjunction with a slit lamp biomicroscope), the Perkins tonometer (a hand-held applanation tonometer) and the non-contact air-puff tonometer (particularly useful for population screening in optometry/optician practice).[11] There are inaccuracies inherent in all methods of tonometry that may contribute to the varying IOP measurements seen in individual patients.[11]

The normal range of IOP and the concept of ocular hypertension

The mean IOP is 16 mmHg; the arbitrary 'normal' range is 10–21 mmHg. IOP must be evaluated in a clinical context.[11–13] There is no clearly defined level of safe IOP; some individuals may develop optic nerve damage with an IOP of 12 mmHg whilst others may not develop damage with an IOP of 30 mmHg.[11–13]

Elevation of IOP is regarded as an important risk factor for the development of glaucoma. However, it is only a risk factor and is present in about 80% of patients with POAG (so-called 'high tension' glaucoma in contrast to 'normal tension' glaucoma).[14]

Ocular hypertension can be defined as IOP greater than 21 mmHg where the optic disc and visual field are normal.[8] (The Baltimore Eye Survey found 6.6% of people had an IOP over 22 mmHg in one or both eyes.[15])

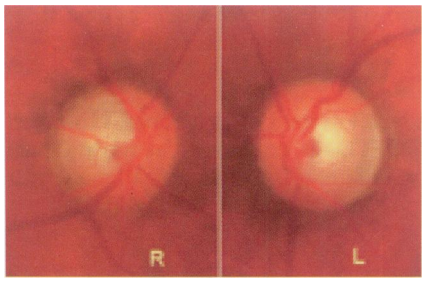


Figure 3 Optic disc cupping in normal tension glaucoma. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

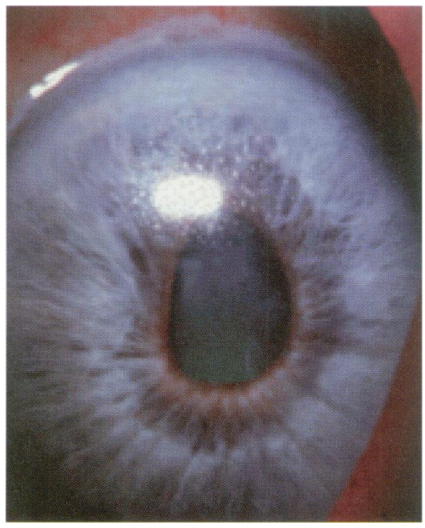


Figure 4 Angle closure glaucoma. Note corneal oedema, shallow anterior chamber, ovoid, mid-dilated pupil, red, congested eye. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

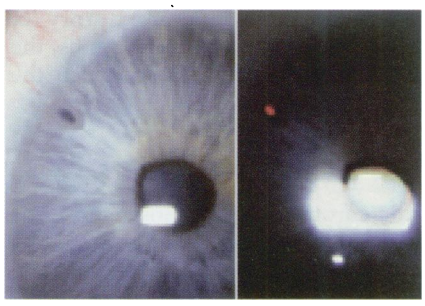


Figure 5 Laser iridotomy. Angle block is relieved by providing a communication between the anterior and posterior chambers of the eye. The patency of the iridotomy is demonstrated by slit-lamp assessment. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

Target pressure – a key therapeutic concept

A useful clinical, therapeutic concept is that each eye treated for glaucoma has a target pressure, this is based upon a general assessment of each individual patient's disease burden.[12 14]

OPTIC DISC EXAMINATION

The optic disc and nerve fibre layer are examined for signs of glaucoma damage. The slit-lamp biomicroscope offers a far superior view to the direct ophthalmoscope due to stereo-visualisation, favourable illumination and high magnification.[11] The normal optic disc is round or slightly oval. It contains a central physiological cup which is devoid of optic nerve fibres and of a different colour. The ratio of optic cup diameter to optic disc diameter (cup to disc ratio, CDR) is usually 0.3. A CDR of greater than 0.5 is seen in 6% of the normal population.[11]

Progressive enlargement of the optic disc cups, documented by periodic follow-up, is an important sign of uncontrolled glaucoma and progressive disease (figure 2). Temporal notching of the neuroretinal tissue of the optic disc is also characteristic (box 1). Atrophy of the retinal nerve fibre layer may be detectable using the green (red-free) light of the slit-lamp biomicroscope.[11]

Other conditions which may mimic glaucomatous optic neuropathy include a large physiological cup and congenital optic disc abnormalities such as coloboma.[11] A so-called splinter haemorrhage located at the optic disc margin may be seen in glaucoma, more commonly in normal tension glaucoma.[16]

Recent scientific work has been devoted to determining the role of optic disc perfusion in glaucoma. Most authorities now agree that there are abnormalities of the ocular micro-circulation which contribute to the pathogenesis of the disease. The precise significance of these abnormalities is not understood and there are some inconsistencies between the results of the major research centres. There are as yet no drugs available which have been consistently shown to aid optic nerve perfusion or which have a neuroprotective function *in vivo*.[17–21]

VISUAL FIELD EXAMINATION (PERIMETRY)

Visual field examination is important in glaucoma management in order to detect any deficits of the patient's central and peripheral vision and to quantify any progressive changes seen with repeated follow-up.[11 16] Several perimeter machines are available, They include the Humphrey, the Goldmann, the Dicon, the Henson and the Octopus.[22 23]

The machines basically test the patient's ability to detect a target stimulus within a spatially defined field. Testing conditions that can be varied include the number, duration and light intensity of the target stimulus and the position of the target.[16 22 23] Factors that can affect the result include patient fatigue or lack of concentration, the spectacle frame, miosis (from pilocarpine drops), and opacities of the ocular media (such as cataract). It is therefore extremely important to differentiate between these artefacts and actual visual field changes.[16 22 23]

The two basic types of perimetry used are static and kinetic perimetry.[16 22 23] Static perimetry is the basis of computerised threshold perimetry; a stationary target is presented and its intensity varied to determine retinal sensitivity. Computerised perimetry also allows for hard disc storage of visual fields and for serial statistical analysis. In kinetic (isopter) perimetry a moving target of differing sizes is presented and is moved from the non-seeing to the seeing 'island of vision' and a threshold determined.

Common types of glaucoma

PRIMARY OPEN ANGLE GLAUCOMA

The most common type of glaucoma in Western societies is primary open-angle glaucoma (POAG). Considered simplistically, the resistance of the trabecular meshwork decreases, typically as an ageing or involutional change, leading to increased IOP. Histologic features include accelerated loss of trabeculocytes and hyalinisation of the trabecular meshwork.

Increased IOP may cause glaucomatous damage by a mechanical and/or ischaemic effect upon the optic nerve. There may be direct mechanical compression of optic nerve fibres due to the increased IOP. As noted, increased IOP may also interrupt the blood supply of the optic nerve fibres and there may also be an intrinsic mechanism associated with glaucoma responsible for nerve fibre damage.[1 17–21]

Glaucomatous optic neuropathy usually has an insidious onset. It is often bilateral; however, it may also be highly asymmetrical. It is often more prevalent in Afro-Caribbean individuals. It is about five times more prevalent in individuals whose close relatives have POAG. Corticosteroid eye drops may cause glau-

Causes of secondary open-angle glaucoma

- pseudo-exfoliative glaucoma
- pigmentary glaucoma
- topical corticosteroids
- phacolytic glaucoma

Box 2

Factors to consider in glaucoma management

- initial IOP
- life expectancy
- ethnicity
- extent of optic nerve damage
- compliance

Box 3

Glaucoma: treatment options

- medication
- laser trabeculoplasty
- filtration and other surgery

Box 4

Commonly prescribed eye drops for glaucoma

Beta-blockers
- timolol (Timoptol)
- levobunolol (Betagan)
- betaxalol (Betoptic)
- carteolol (Teoptic)

Other medications
- brimonidine (Alphagan)
- dorzolamide (Trusopt)
- latanaprost (Xalatan)
- pilocarpine
- dipivefrin (Propine)

Box 5

Administering eye drops

If more than one type of eye drop is being prescribed the patient should be instructed to wait 5 minutes between the instillation of eye drops in order to maximise ocular absorption. The eyelids should be gently closed after instillation. Patients may also find administration easier in front of a mirror.

Box 6

comatous damage by producing an elevation of IOP as a side-effect. This side-effect, known as steroid response, is more common in patients with POAG.[11]

NORMAL TENSION GLAUCOMA

Elevated IOP is a major risk factor for the development of glaucoma. However, 20% of patients do not have an elevated IOP. This is referred to as normal tension glaucoma (figure 3). It is more common in females. Optic nerve damage and visual field loss occur in normal tension glaucoma, despite a 'normal' IOP.[24]

ANGLE CLOSURE GLAUCOMA: AN OPHTHALMIC EMERGENCY

In primary angle closure glaucoma, the pupil is anatomically apposed to the anterior surface of the crystalline lens, thereby restricting flow of aqueous from the posterior chamber to the anterior chamber (figure 4). The peripheral iris becomes apposed to the anterior chamber angle, thereby decreasing outflow of aqueous through the trabecular meshwork and causing increased IOP. Acute angle closure glaucoma is an ophthalmic emergency. The treatment involves urgent reduction of IOP with medication followed by laser iridotomy (figure 5). Patients with a unilaterally painful red eye should be referred for urgent ophthalmic assessment.[11]

LESS COMMON TYPES OF CHRONIC GLAUCOMA

Secondary open-angle glaucoma

In secondary open angle glaucoma decreased outflow of the aqueous humour results from other conditions such as corticosteroid administration, pigmentary or phacolytic glaucoma, or neovascularisation of the iris (rubeotic glaucoma) (box 2).[11]

Paediatric glaucoma

Paediatric glaucoma can be congenital or develop during infancy or later childhood. It is most commonly primary (ie, isolated). Secondary glaucoma can be associated with ocular conditions (such as aniridia, trauma or ocular tumour) or with systemic conditions (such as rubella). A neonate or young child with glaucoma may present with epiphora, photophobia, blepharospasm, enlargement of the cornea or of the globe. Bupthalmos, enlargement of the globe, is not uncommon. IOP can be measured in infants and young children using a Perkins tonometer or Tono-Pen. In older children a Goldmann applanation tonometer can be used. Many types of paediatric glaucoma are genetically determined.[25-28]

Risk factors

Other factors which may contribute to the development of glaucoma include diabetes, cardiovascular disease, myopia and a positive family history of glaucoma. There may be at least two primary open-angle glaucoma genes, the GLC1A gene on chromosome 1q and other genes located elsewhere in the genome.[6 25-28]

Differential diagnosis

Conditions that can mimic glaucoma include carotid artery stenosis and optic nerve compressive lesions. The need for neuro-ophthalmic evaluation should be considered particularly for patients with atypical features (such as unilateral optic neuropathy).[11 16]

Management of glaucoma

It can often be very difficult to distinguish the patient with early glaucoma. Complex issues in the management of glaucoma include both the indications for treatment and the type of treatment.[12] These difficulties lead to the dilemma of determining which patients should be treated. Patients that do not have glaucoma may be subjected to the inconvenience, expense and possible toxicity of treatment. The unfortunate corollary is that treatment may be withheld from patients who have or may develop glaucoma.[12]

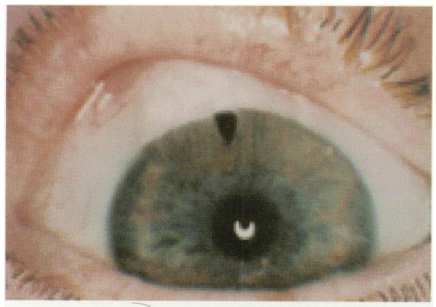
The ultimate goal in glaucoma management is the preservation of vision. Factors to be considered include the initial IOP, the patient's life expectancy and the degree of optic nerve damage.[11]

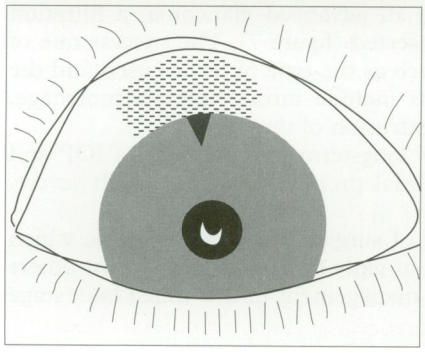
A target IOP should be determined. This represents the IOP aimed for following therapy. For example, a patient with advanced glaucomatous optic neuropathy may require a target IOP of 12 mmHg. In another patient, with an initial IOP of 45 mmHg, who has less optic disc damage, vision may be preserved with a target IOP of 23 mmHg.

Pupil mydriasis by general practitioners, physicians and optometrists

- non-ophthalmologists may perform pupil dilation (mydriasis) provided patients are informed of the risks and benefits of the procedure
- tropicamide 0.5%, which lasts 4–6 hours, is the preferred mydriatic
- the risks of provoking angle closure glaucoma are small, even in patients who are known to have POAG or ocular hypertension
- special care should be taken with elderly patients, especially elderly women with cataracts (women are more prone to primary angle closure glaucoma)
- patients should be instructed not to drive home and should be encouraged to re-attend or seek ophthalmic care if they develop a red or painful eye

Box 7





Figures 6 Trabeculectomy, the principal form of surgery used to treat open-angle glaucoma. The anterior chamber is drained via a filtration bleb to the subconjunctival space. An ostium is made in the inner sclera while a fistula covered by a flap is fashioned in the outer sclera, thereby providing an alternative drainage site for aqueous humour. Note also the peripheral iridectomy. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

A patient with a long life expectancy may require more frequent follow-up and more aggressive therapy. More aggressive therapy may be similarly required for patients with extensive optic nerve damage.

Compliance is a very important issue in glaucoma management.[11 12] Patients may be have no symptoms from glaucoma. Medication may cause various side-effects. It may be difficult to comply with treatment regimes, for example, forgetting to administer midday doses of eye drops or falling asleep before taking the evening dose.[3]

TOPICAL MEDICATIONS

Eye drops (and gel preparations) are the most common form of therapy used to treat glaucoma. In addition, they are often prescribed to prevent glaucoma in patients who are regarded as being glaucoma suspects. The choice of medication depends particularly on how well the eye drop reduces the IOP and how well the medication is tolerated. Many possible ocular and systemic side-effects can occur. The ideal drug to treat glaucoma would be a substance which lowers IOP, facilitates blood flow to the retina and prevents ischaemic neuronal cell death.

Long-term therapy with topical medication may cause histological changes to the conjunctiva. These secondary changes may be responsible for the subsequent failure of trabeculectomy surgery.[29 30]

Beta-Blockers

Side-effects include asthma, bradycardia, reduction in exercise tolerance, altered mentation, impotence, drowsiness and fatigue. A topical beta-blocker is usually the first line therapy in patients over the age of 40 years unless there are systemic contraindications such as asthma or bradycardia.[11 12 31–33]

Timolol (Timoptol) Timoptol is a non-specific beta-blocker prescribed twice daily. Timolol LA was designed for once-daily administration. It appears to be very similar in efficacy to twice-daily administration of Timoptol 0.25% drops.

Levobunolol (Betagan) Once-daily administration may be effective in about 70% of patients, thereby improving compliance and safety. In view of the diurnal variation in IOP with the peak occurring in the morning, instillation of the eye drop after awakening is advisable.

Betaxolol (Betoptic) This is a selective beta-blocker with similar but less severe side-effects to those seen with other beta-blockers. A favourable effect on the ocular circulation and also a possible neuroprotective action is claimed by some authors for betaxalol, however, it does not reduce IOP as effectively as the non-specific beta-blockers.

Carteolol (Teoptic) Carteolol 1% is a non-selective beta-blocker with intrinsic sympathetomimetic activity.

Other medications

Brimonidine (Alphagan) This agent is an alpha 2-agonist. It appears to be as effective as Timolol in controlling glaucoma but does not cause a significant reduction in heart rate. Side-effects include dry mouth (10% of patients), topical allergy and external eye disease (seen in 10% of patients in a 1-year trial), drowsiness, psychic depression and fatigue.[34]

Dorzolamide 2% (Trusopt) Dorzolamide is a topical carbonic anhydrase inhibitor. It has an additive effect in IOP reduction when combined with beta-blockers. It often requires three times a day instillation whether prescribed alone or in combination. Side-effects include ocular burning and stinging, a metallic taste, blurred vision from corneal epithelial toxicity. It lacks, however, the profound side-effects of systemic carbonic anhydrase inhibitors.[31]

Latanaprost (Xalatan) Latanaprost is a prostaglandin analogue. It is effective with once-daily instillation; it may be as effective as timolol. It is said to be efficacious in the treatment of normal tension glaucoma.[24] Iris pigmentation (iris suntan syndrome) can occur as a side-effect in 15% of patients at 1 year. It is most common in hazel irides, and uncommon in pale blue irides. The change in iris colour occurs slowly and is irreversible. Ocular irritation and redness also occurs in a third of patients.[24]

Pilocarpine Side-effects are common and include brow ache, blurred vision (from spasm of accommodation) and decreased night vision (from miosis). Muscarinic agonists derived from the *pilocarpus* plant have been in use in ophthalmology for over a century and are still highly efficacious in the treatment of both acute and chronic glaucoma.[11 12 33]

Surgical procedures for glaucoma

- trabeculectomy (with or without antimetabolites)
- filtration devices
- combined procedure (glaucoma/cataract surgery)
- goniotomy/trabeculotomy (paediatric glaucoma)
- peripheral iridectomy (surgical)
- cyclodiode or YAG laser cyclodestruction
- cyclodestruction (cyclocryotherapy)

Box 8

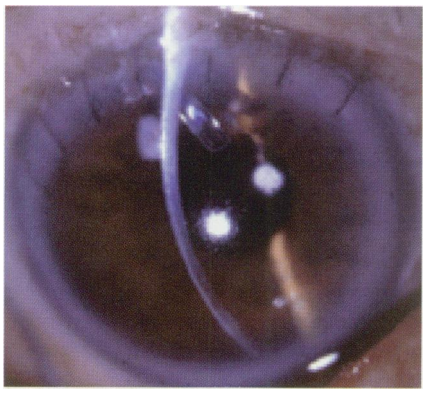


Figure 7 Filtration tube in anterior chamber of eye with advanced glaucoma. Note also the previous cataract surgery. Illustration courtesy of Dr Robert Harvey, from CD ROM *Practical ophthalmology*, Palmtrees Publishing, 1998

Support societies for glaucoma patients and for the visually disabled in UK

International Glaucoma Association
King's College Hospital,
Denmark Hill, London SE5 9RS
Tel 0171 737 3265

Royal National Institute for the Blind
224 Great Portland Street,
London W1N 6AA
Tel 0171 388 1266
Handout on facilities available 0345–023153

Partially Sighted Society
PO Box 322, Queens Rd,
Doncaster DN1 2NX
Tel 01302 323132 or 0171 372 1551

Box 9

Dipivefrin (Propine) This is a pro-drug, converted to adrenaline inside the eye. Ocular side effects are common, including redness and stinging, and chronic conjunctival folliculosis. If a patient is already using a topical beta-blocker then adding propine does not usually significantly lower the IOP further.[11]

ARGON LASER TRABECULOPLASTY

This treatment involves the application of laser energy (usually argon green) to the trabecular meshwork, thereby improving the rate of outflow of aqueous humour. Several weeks may be required before the effects become evident. Complications include transient IOP elevation in the first few hours postoperatively and permanent IOP elevation. The IOP is usually reduced by 25% and 80% of patients show an initial beneficial effect.

One study yielded a success rate for argon laser trabeculoplasty of 46% at 3-year follow-up. In Afro-Caribbean patients the benefit is typically lost earlier.[11 35] Argon laser trabeculoplasty is less successful following cataract surgery and retreatment is generally associated with a low success rate.[11 35] Argon laser trabeculoplasty is not useful in certain types of glaucoma, for example, paediatric glaucoma.[11]

GLAUCOMA SURGERY

Usually topical medication is the initial type of therapy. There is increasing evidence, however, particularly from studies undertaken at Moorfields Eye Hospital, that initial therapy in the form of surgery may have significant advantages over medical therapy, even when the patient presents with early glaucoma.[29 30]

Trabeculectomy

The most common surgical procedure for glaucoma is trabeculectomy (figure 6). In trabeculectomy an ostium is made in the inner sclera, a fistula covered by a flap fashioned in the outer sclera, thereby providing an alternative drainage site for aqueous humour.[11 29 30 36 37]

Trabeculectomy lowers the IOP more consistently than anti-glaucoma eye drops or laser trabeculoplasty. While some authorities consider performing trabeculectomy as primary therapy, glaucoma surgery may also be associated with complications. The trabeculectomy fails usually because of scarring around the scleral flap or due to closure of the internal ostium.[29]

Cataracts may progress rapidly after trabeculectomy. Age 61 years or older, exfoliative glaucoma, postoperative hypotony, and IOP peaks were identified as risk factors for accelerated cataract progression after trabeculectomy.[36] Microtrabeculectomy, is a variant of the trabeculectomy procedure which is currently gaining popularity.[37]

Topical antimetabolites, such as 5-fluorouracil and mitomycin, can be used intra-operatively or immediately postoperatively in order to increase the success rate of the trabeculectomy procedure. Antimetabolite use, however, may increase the risk of complications such as ocular hypotony, wound leakage and infection.[38 39]

Filtration devices/contact diode laser transscleral cyclophotocoagulation (TSCPC)

Where trabeculectomy is likely to fail or in advanced glaucoma, a filtration device, such as a Molteno tube, can be inserted (figure 7). The success rate of this type of surgery depends on factors such as the type of device used and the type of glaucoma. Possible complications include intra-ocular haemorrhage, strabismus, tube blockage, infection and extrusion of the device.[11]

Contact diode laser TSCPC also yields long-term improvement of IOP and preservation of visual acuity in a substantial proportion of eyes with severe, medically uncontrolled, glaucoma.[40]

In conclusion, there are a wide variety of surgical therapies available which effectively treat POAG, some authors favour early trabeculectomy whilst others are more conservative, favouring medical management until a much later stage of the disease.[29 30 41]

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