Glaucoma affects millions of people worldwide, but it is difficult to diagnose and manage.

Glaucoma is a major cause of irreversible blindness worldwide, and it also causes substantial disability before patients become blind. Glaucoma is difficult to detect and diagnose, and it is highly undertreated globally. In most surveys carried out in high-income countries, over 50% of people found to have glaucoma had not been diagnosed and are therefore not receiving treatment, rising to over 90% in low- and/or middle-income countries. This is because glaucoma is mostly asymptomatic until relatively late in the disease, so patients do not notice that there is a problem. In many low- and/or middle-income settings, as many as 35% of people diagnosed with glaucoma already have severe sight loss: they presented too late to benefit from interventions that may have preserved their vision.

Our aim in this issue of the Community Eye Health Journal is to provide practical articles that will help clinicians facing the challenge of providing care for glaucoma patients. Topics include glaucoma detection and diagnosis, gonioscopy (a vital examination technique), the latest guidelines for open-angle glaucoma management, and tips for managing neovascular glaucoma and the painful, blind eye. We also look at the importance of counselling and how low vision support can benefit patients with vision loss due to glaucoma.

Glaucoma is a huge topic, and this issue covers care for adults only, concentrating on open-angle disease. Many useful resources are listed on page 27 and additional articles are available only on our website and smartphone app.

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About this issue

Glaucoma affects millions of people worldwide, but it is difficult to diagnose and manage. In this issue, we have included practical articles to help clinicians facing the challenge of providing care to glaucoma patients. Topics include glaucoma detection and diagnosis, gonioscopy (a vital examination technique), the latest guidelines for open-angle glaucoma management, and tips for managing neovascular glaucoma and the painful, blind eye. We also look at the importance of counselling and how low vision support can benefit patients with vision loss due to glaucoma.

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Defining and diagnosing glaucoma: a focus on blindness prevention

A diagnostic approach that focuses on patients with definite (or clinical) glaucoma optimises the likelihood of preventing visual disability due to this potentially blinding condition.

The term glaucoma refers to a group of diseases that affect the optic nerve and could potentially lead to irreversible visual loss. Glaucomatous optic neuropathy is the hallmark of all types of glaucoma. It is characterised by deformation of the optic nerve (see Figure 1, page 4), which manifests as diffuse or focal narrowing of the neuroretinal rim and peripapillary retinal nerve fibre layer loss. The type of glaucoma, the severity of the disease, and the risk of blindness can be assessed by carrying out gonioscopy, slit lamp examination, visual field tests, and intraocular pressure (IOP) measurement.

Definitions of glaucoma

The definition of glaucoma in adults has changed over the years due to changes in our understanding of how glaucoma affects the eye, the technology available, and the

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reasons why a particular definition was constructed. A current clinical definition of glaucoma is: “A characteristic pattern of glaucomatous optic neuropathy (e.g., narrowing of the neuroretinal rim) with corresponding visual field defects.”

Other changes related to glaucoma might be present in some patients before a clinical diagnosis is made. These may include:

- Thinning of the retinal nerve fibre layer or ganglion cell layer, which can be visualised using optical coherence tomography (OCT)
- Other functional changes such as reduced contrast sensitivity and electrophysiological abnormalities.

However, if clinically significant glaucomatous damage has occurred, then optic disc changes are normally visible, and it is very unusual that glaucoma would be diagnosed from visual field or OCT changes without disc features.

In this article, we want to propose an approach to diagnosing adults with glaucoma that is focused on preventing visual disability. With this approach, we distinguish between two groups of patients:

- Patients with definite (or unequivocal) glaucoma: those who have definite signs of glaucomatous optic neuropathy. They are at imminent risk of visual loss, usually need treatment, and must be monitored.
- Patients with suspected (or equivocal) glaucoma: those with possible signs of glaucomatous optic neuropathy. They are not at immediate risk of visual loss, at least in the short term, and usually do not need treatment, but can/must be followed up and monitored (depending on the patient).

We propose that attention is focused on patients with definite glaucoma as they are at greater risk of blindness and are likely to require monitoring and treatment.

Essential investigations
The following investigations are needed in order to determine whether a patient has definite glaucoma requiring treatment, has suspected glaucoma and also for ongoing monitoring.

Visual acuity with best correction. Although visual acuity only deteriorates at the last stages of glaucoma, measuring it on all visits is important to assess the overall visual function and rule-out other diseases.

Anterior segment examination using a slit lamp. This will help in the detection of secondary types of glaucoma such as pseudoexfoliation, uveitic or pigmentary glaucoma.

Anterior chamber angle assessment (including gonioscopy). This will determine whether the patient has open-angle or angle-closure glaucoma.

Examination of the optic nerve head. This is done using a slit lamp (binocularly) and a 90D or similar lens. Perform a dilated examination to rule out other retinal diseases and to make it easier to examine the optic nerve. Do this on the initial visit and yearly or if any clinical parameter (decreased visual acuity, new onset metamorphopsia etc.) changes significantly.

Visual field testing. Carry out static, automated perimetry, commonly performed using a Humphrey visual field machine.

Tonometry. Use Goldmann’s applanation tonometer.
Optional investigations
These additional investigations or tests can be performed if needed and if the equipment is available.

Measuring central corneal thickness. This can improve the accuracy of measurements using Goldmann’s applanation tonometer because this method overestimates IOP in patients with thick corneas and underestimates it in those with thin corneas. However, nomograms to ‘correct’ the IOP tend to be inaccurate at an individual level and are not recommended. A thin central cornea might influence the decision if the target IOP is reached in a patient with progressive disease, as the actual IOP is likely to be higher than measured.

Optical coherence tomography (OCT). This can help in the examination of the optic nerve and retina. The most frequently used metrics are the average thickness of the circumpapilar retinal nerve fibre and ganglion cell layer. However, each innovation cycle produces a different generation of devices with incompatible measurements, so the results cannot be compared for long-term follow-up assessments.

Corneal hysteresis. This non-contact tonometry technique also assesses the corneal biomechanical response and may prove to be helpful for glaucoma assessment.

Characteristics of definite glaucoma
After an initial or single examination
The characteristics of a definite or unequivocal diagnosis of glaucoma and/or where intervention might be needed, after an initial or single examination include:

1. Focal complete loss of the neuroretinal rim. The disc damage likelihood scale (DDLS) is a good way of grading the optic disc.
2. DDLS stage ≥ 6.
3. Cup-to-disc ratio > 0.8.
4. Focal narrowing of the neuroretinal rim, with a corresponding visual field defect. It is important to identify if the sector of the optic nerve that is affected corresponds to the location of the VF defect. Representations of the structure-function relationship, such as the Garway-Heath map, help clinicians identify if the damaged sector of the neuroretinal rim is affecting the corresponding visual field locations (Figure 2).
5. IOP > 35 mm Hg is not diagnostic of glaucoma, but almost all patients with this IOP level need IOP-lowering interventions. A high IOP in the absence of disc damage may be seen in patients with a secondary glaucoma or primary angle closure disease. Over-estimation of the IOP should be considered, e.g., a non-contact tonometer used or a very thick cornea. It is recommended to repeat the measurement before initiating treatment.

Figure 1 Different forms of glaucomatous optic neuropathy. A Enlarged CDR. B Focal superior disc rim narrowing. C Diffuse loss of the inferior disc rim. D Inferotemporal thinning of the disc rim and retinal nerve fibre layer defect.
After consecutive examinations
On follow-up examination, the following signs of progression would confirm an unequivocal or definite diagnosis of glaucoma:

1. Progression of a visual field defect: that corresponds to a location of narrow neuroretinal rim or retinal nerve fibre layer loss.
2. Progression of glaucomatous optic neuropathy:
   - Enlargement of the vertical cup-disc ratio > 0.2
   - Increase in DDLS > 2 stages
   - Increased narrowing of the neuroretinal rim (change in a sector of the neuroretinal rim (NNR) from narrow to complete loss or from a homogeneous neuroretinal rim to a narrow sector)
   - Significant expansion of a retinal nerve fibre layer defect on OCT
   - Change in the course of vessels due to changes of the optic nerve head
   - The detection of a single new disc haemorrhage usually should not be considered sufficient to diagnose glaucoma or progression, however, they do increase the risk of developing glaucoma and visual field deterioration, particularly if they appear repeatedly.

There are other factors which need to be considered when deciding on and planning the treatment of a person with glaucoma.

Patients with confirmed glaucoma, i.e., those who are most in need of treatment, can be diagnosed using a slit lamp, tonometer and visual fields alone. Clinicians who do not have access to other instruments, such as OCT, can be reassured that their patients are receiving good care so long as they receive a good clinical examination and visual field analysis.

When to suspect glaucoma
The identification of only one of the following characteristics is not enough to confirm the diagnosis of glaucoma: See Figure 1 for examples.

- Cup-to-disc ratio > 0.7 (this value applies to all disc sizes, but in small discs, a smaller cup-to-disc ratio represents greater damage than in larger discs)
- Diffuse narrowing of the neuroretinal rim
- DDLS stage ≥ 4
- Disc haemorrhages
- Retinal nerve fibre layer defects
- IOP > 24 mm Hg increases the risk for glaucoma, especially in thin corneas
- Abnormal visual field defects (remember that all the diseases that affect the visual pathway from dry eye affecting the cornea, cataracts affecting the lens, retinal changes, to cerebral strokes affecting the posterior visual pathway, can affect the visual field results)
- A reduction in the thickness of OCT parameters.

It is important to remember that an OCT scan that flags in red the thickness of the retinal nerve fibre layer, ganglion cell layer, or optic nerve head rim of a patient is not enough to diagnose glaucoma. These parameters are compared to a normative dataset that underrepresent many ethnic and age groups. Similarly, an OCT scan with all parameters in green is not enough to rule out glaucomatous optic neuropathy and should not be considered equivalent to a ‘normal’ optic nerve. OCT changes which, in reality, represent early glaucomatous changes, typically involve only the superior or infero-temporal neuroretinal rim or macular ganglion cell layer. These changes should usually correspond with optic disc changes seen clinically and/or with visual field changes (Figure 2).

It is always important to exclude non-glaucomatous causes of an enlarged cup-to-disc ratio and loss of neuroretinal rim or retinal nerve fibre layers.

Glaucoma classification
After clinical or definite glaucoma is diagnosed, the two main questions for the clinician are:

1. Is this glaucoma primary or secondary?
   
   It is important to rule out secondary glaucomas which might well carry a high risk of glaucoma blindness. These include those which could have causative treatment if identified appropriately (e.g., neovascular glaucoma) and those associated with medical conditions (e.g., uvetic or increased episcleral pressure glaucoma) and which may even potentially be life threatening (e.g., rheumatological diseases and cavernous-carotid fistulas).

2. Is the anterior chamber angle open or closed?
   
   Gonioscopy is critical.

The management plan of a patient with glaucoma differs significantly depending on whether the iridotrabecular angle (where aqueous drains out of the eye) is open, narrow or closed and it is vitally important to determine this with gonioscopy.

When a patient is diagnosed with definite glaucoma, it is the responsibility of all eye care providers to advise patients and family about the increased risk of glaucoma in first-degree relatives.

Figure 2 Map representing the relationship between Standard Automated Perimetry visual field sectors and sections of the peripapillary OCT scan circle. This map is based on the work of Garway-Heath et al and shows the correspondence between areas of the visual field and peripapillary retinal nerve fibre layer due to the anatomical configuration of the retinal nerve fibre bundles.
Gonioscopy skills and techniques

All glaucoma patients must undergo a thorough gonioscopy examination as part of their ophthalmic assessment.

Gonioscopy is a technique of viewing the iridocorneal angle: the area between the iris and cornea where the trabecular meshwork is located and where aqueous humour drains out of the eye. Gonioscopy lenses are needed to view the angle, as light from this area would not otherwise reach the observer.

Gonioscopy allows the identification of structures of the anterior chamber angle and an estimation of the angle width; it is also necessary during any procedures affecting the angle, such as laser or surgery.

Anything which impedes drainage through the trabecular meshwork can cause an increase in the intraocular pressure. It is therefore critical that all potential and newly diagnosed glaucoma patients undergo a thorough gonioscopy examination as part of their ophthalmic assessment so that the mechanism of raised intraocular pressure can be established.

In this article, we will focus on a basic gonioscopy technique for the diagnosis of primary and secondary angle-closure glaucoma and for use in angle procedures.

**Structures of the iridocorneal angle**

From anterior (towards the cornea) to posterior (towards the iris), the structures seen are:

1. **Schwalbe’s line.** Demarcates Descemet’s membrane from the anterior trabeculum. It can be located by identifying the corneal wedge (Figure 1).

Gonioscopy allows the identification of structures of the anterior chamber angle and an estimation of the angle width; it is also necessary during any procedures affecting the angle, such as laser or surgery.

2. **Non-pigmented trabecular meshwork.** A pale area adjacent to Schwalbe’s line which does not drain aqueous humour.

3. **Pigmented trabecular meshwork.** Brown/pigmented area where aqueous humour drains from the eye; it is critical to identify whether or not it is visible on gonioscopy.

4. **Scleral spur.** A narrow, dense, whitish band posterior to the trabeculum; a consistent landmark in all eyes.

5. **Ciliary body.** A dull, brown band posterior to the scleral spur.

Figure 1 Two photographs (A and B) and two drawings (C and D) showing the structures seen on gonioscopy of an open angle. B shows a patient with pigment dispersion where the angle is densely pigmented, especially the pigmented trabecular meshwork. Some patients may have very little pigment present (hypopigmented angle) and identifying the different structures can be challenging. The bottom left image shows a cross-section of the corresponding image on the bottom right. The corneal wedge is shown where the reflections from the inner and outer aspects of the cornea meet, showing the position of Schwalbe’s line, helpful in hypopigmented angles.

Key

1. Schwalbe’s line
2. Non-pigmented trabecular meshwork
3. Pigmented trabecular network
4. Scleral spur
5. Ciliary body band
6. Iris
7. Corneal wedge
Gonioscopy lenses

**Direct** gonioscopy lenses (Figure 2a), such as the Swan-Jacobs lens, act as prisms and provide a direct, panoramic view of the angle. They are used for surgical procedures on the angle, with the patient lying on their back in the operating theatre.

**Indirect** gonioscopy lenses (Figure 2b), such as the Goldmann and Magnaview goniolenses (see the panel at the end of the article), combine a prism and a mirror to provide a reflected image of the opposite angle. Gonioscopy is carried out at the slit lamp, with the patient in a sitting position.

Gonioscopy examination technique

Some excellent videos and tutorials are freely accessible at www.gonioscopy.org

- Ensure minimal lighting in the room and a short (1 mm) slit beam to avoid artifactual opening of the angle (bright illumination will cause pupil constriction and opening of the angle).
- Instil topical anaesthesia and explain the procedure to the patient.
- Instruct the patient to keep both eyes open as this results in less squeezing of the eye to be examined.
- For less experienced practitioners, we suggest using an indirect gonioscopy lens with coupling gel as a more stable view is gained.
- Apply a coupling gel to the lens (e.g., carbomer gel).
- Instruct the patient to look up.
- Insert the inferior rim of the lens onto the surface of the eye and then quickly apply the rest of the lens rim to the globe (Figure 3).
- To make insertion easier, the forefinger of the hand inserting the lens can be used to pull the lower lid down and if necessary, the thumb of the other hand to elevate the upper lid.
- Once the lens is in, ask the patient to look straight ahead.
- View the inferior angle through the superior mirror, and vice versa.
- Rotate the lens to view the nasal and temporal angles. In order to best visualise the trabecular meshwork and other angle structures, the slit beam should be at right angles to the mirror and the light offset by 30–60 degrees.

**Assessing the angle width and status**

First establish whether the angle is open or closed. Iridotrabecular contact (ITC) is present when it is not possible to visualise the pigmented trabecular meshwork without manipulation. A good method of locating trabecular meshwork is to identify Schwalbe’s line (see Figure 1) and then move posteriorly.

“A good method of locating trabecular meshwork is to identify Schwalbe’s line (see Figure 1) and then move posteriorly.”

Continues overleaf

Figure 2a Direct gonioscopy.

Figure 2b Indirect gonioscopy.

Figure 3 Insertion of an indirect gonioscopy lens using coupling gel.
...It is important to consider the whole clinical situation.

Asking the patient to move the eye in the direction of the mirror, indenting the cornea, or increasing the light can all help to open the angle and visualise more posterior structures. These manoeuvres can help to differentiate between appositional angle closure as compared to synechial closure. However, grading of the angle width (how open the angle is) should be performed in dim light, with the eye in the primary position and without indentation.

There are a number of different grading systems for angle assessment (see references). In practice, a modified Shaffer grading scheme (Figure 4) is commonly used for grading the angle width. The visibility of angle structures is used:

- **Grade 0.** No angle structures visible.
- **Grade 1.** Schwalbe’s line visible (i.e., the angle is essentially closed as aqueous humour is not able to drain).
- **Grade 2.** Pigmented trabeculum visible (aqueous able to drain but the angle is relatively narrow).
- **Grade 3.** Scleral spur visible.
- **Grade 4.** Ciliary body visible (angle is wide open).

Each quadrant (superior, inferior, nasal and temporal) should be graded. Although this is a quick and easy system it is also helpful to also estimate the angle width in degrees (Figure 5) as it provides more information on risk of future closure.

When assessing whether angle closure is present, or if the patient is at high risk of angle closure, it is important to consider the whole clinical situation (clinical history, symptoms, other examination findings, and so on). For practical purposes, if the angle width is greater than 25 degrees and scleral spur is visible all the way around the angle, there is likely to be a low risk of angle closure. If pigmented trabecular meshwork cannot be seen, i.e, there is iridotrabecular contact for more than two quadrants (over half) of the angle on gonioscopy, there is likely to be a high risk of angle closure. In this situation, intervention to open the angle may be warranted, depending on the presence of other abnormalities such as raised intraocular pressure or risk factors for acute angle closure.

**Indirect gonioscopy lenses**

Goldmann and Magnaview lenses are indirect gonioscopy lenses, or goniolenses (Figure 6), which require the use of a coupling gel to fill the gap between the lens and the cornea and give a stable, undistorted view of the angle structures and configuration. These lenses have either one or two mirrors through which the observer views the angle. The Magnaview lens is larger than all the Goldmann lenses, giving more magnification and allowing a more detailed view of the angle. However, it may be difficult to insert if patients have a small palpebral aperture. It can be used for delivering selective laser trabeculoplasty.

The Zeiss, Posner and Sussman lenses are indirect gonioscopy lenses which allow a rapid view of the entire angle without the need for a coupling gel. They can be used for indentation gonioscopy – pressure is applied on the cornea with the lens, this can help to open up the angle so that further structures can be identified (such as with plateau iris syndrome). If peripheral anterior synechiae are present the angle will not open further even with indentation. The view of the angle is not as stable or clear as that with the Goldmann or Magnaview. Inadvertent indentation can result in corneal striations and distortion of the view as well as accidental opening up of the angle and misclassification of a closed angle as open.

**Further reading**

- University of Iowa Health Care. www.gonioscopy.org
- Eyewiki. https://eyewiki.aao.org/gonioscopy
Management of chronic open-angle glaucoma

The purpose of glaucoma care is to preserve the quality of life and livelihood of a person with glaucoma, which includes maintaining their visual function while minimising the side-effects and complications of treatment. In order to deliver such patient-centred care, a glaucoma care system which can provide long-term, affordable, sustainable, and equitable care needs to be in place.

The objective of glaucoma treatment

A person with open-angle glaucoma is at risk of irreversible blindness. The objective of treatment is to minimise this risk, usually by lowering the intraocular pressure (IOP) so that an individual upper threshold IOP (also known as their target IOP) is not exceeded. However, we must weigh the expected long-term benefit of preserving vision against side-effects, complications, and the long-term cost of treatment – all of which can affect quality of life and the person’s livelihood.

Choosing a therapy plan

An individual therapy plan is based on a detailed history, visual acuity, and examination of general and glaucoma-related structural and functional details and any changes in these (visual field, disc damage likelihood scale, etc.).

The key result from the history and examination is the rate of progression of the glaucomatous damage. This has to be determined regularly, for each eye separately, and can be divided broadly into three groups:

Group 1. Probably no progression, or only a low rate of progression.

Group 2. Insufficient information to determine the rate of progression.

Group 3. A high rate of progression of vision loss which will probably lead to vision impairment during the patient’s lifetime and might have an impact on her or his quality of life and daily activities.

If the rate of progression is low, monitoring can continue, either by only observing the eye or continuing with the same treatment (Group 1). If this was the patient’s first assessment or if there is not enough information from previous examinations available, the risk for progression can be estimated (Group 2). An increased risk of glaucoma progression to visual loss is associated with advanced disease on presentation, high intraocular pressure, older age, certain ethnic groups, disc haemorrhages, and thin central corneal thickness, among others.1,2

Choosing a therapy plan

If there is an estimated high risk of progression (Group 2), or if there is actual evidence of a high rate of progression (Group 3), an escalation of treatment is indicated. However, it is important to review the current treatment before escalating the therapy;

Key points

- The aim of patient-centred glaucoma care is to preserve and promote quality of life and livelihood
- The objective of treatment is to minimise the risk of irreversible vision loss
- The individual therapy plan is determined by the rate of progression of glaucoma
- Available treatments should be tailored to the person with glaucoma
- Available treatments should be tailored to the person with glaucoma
- A single measurement of a high intraocular pressure (IOP) alone should not usually trigger a change of the plan
- Refer patients for low vision care, rehabilitation, and counselling as needed.
e.g., first checking whether the patient was able to purchase the prescribed eye drops and whether they have actually been taking the treatment.

**Lowering IOP**

Lowering IOP prevents or delays the onset or progression of glaucoma. However, there is no specific IOP threshold, formula, or percentage reduction which applies to all patients. Instead, it is recommended to set and subsequently adapt an individual target IOP. This can be defined as the IOP that slows down the rate of progression of the glaucomatous damage enough to maintain the patient’s quality of life and livelihood during their lifetime.\(^{1,3}\)

This definition contains three elements which need to be considered:
- **Intraocular pressure**
- **Rate of progression of the glaucomatous damage**
- **Quality of life and livelihood.**

Analysis of the advanced glaucoma intervention study (AGIS) showed that participants with IOP <18 mmHg at 100% of visits showed no visual field progression.\(^{4,5}\) However, high-quality prospective data comparing different target IOP levels are not currently available; as such, the trade-off between risks and benefits associated with different thresholds is unclear.\(^{1}\) Target pressure should therefore be individualised and may need adjustment over time.\(^{1}\)

A single measurement of a high intraocular pressure alone should not trigger a change of management and needs to be put into the context of the other examination results and the history, including self-reported adherence. IOP may also fluctuate within hours or days so that several measurements might provide a better picture of the general level of IOP in an eye. Sometimes a repeat examination on the same day or a repeat follow-up visit within a few weeks might be helpful to decide on the next step, e.g., an escalation of treatment. This also depends on the level of urgency, which can be high for eyes with severe visual field loss and a high rate of progression.

There are several treatment options available to reduce IOP. They can be divided in three groups: medical treatment (usually eye drops but may include oral or intravenous medication, e.g., acetazolamide), laser and surgery. Current cost-effective examples are timolol eye drops, selective laser trabeculoplasty, and trabeculectomy. Other eye drops are only available at considerably higher cost and may not be affordable for some patients in an LMIC context.\(^{6}\)

Some examples are given below, but these will vary depending on the local or regional glaucoma care system.

**Medical treatment**

Medication (a conservative treatment) can reduce IOP by decreasing aqueous production (Table 1a) or enhancing aqueous outflow (Table 1b). Osmotic agents are not mentioned as they are not for long-term use.

### Table 1a Efficacy and side effects of glaucoma medication to decrease aqueous production

<table>
<thead>
<tr>
<th>Drug</th>
<th>Efficacy</th>
<th>Side effects (selection)</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-Blockers (e.g., timolol)</td>
<td>+++</td>
<td>Bronchospasm, bradycardia, depression</td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors (systemic) (e.g., acetazolamide)</td>
<td>+++</td>
<td>Metallic taste, electrolyte imbalance</td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors (topical) (e.g., dorzolamide)</td>
<td>++</td>
<td>Stinging, burning, headache</td>
</tr>
<tr>
<td>α2-adrenergic agonists (e.g., brimonidine)</td>
<td>++(+</td>
<td>Toxic reaction of external eye, dry mouth. Contraindicated in children</td>
</tr>
</tbody>
</table>

### Table 1b Efficacy and side effects of glaucoma medication to enhance aqueous outflow

<table>
<thead>
<tr>
<th>Drug</th>
<th>Efficacy</th>
<th>Side effects (selection)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostaglandin analogues (e.g. latanoprost)</td>
<td>+++(+)</td>
<td>Eyelash growth, periorbital fat atrophy, increased iris pigmentation</td>
</tr>
<tr>
<td>Rho-kinase inhibitors (e.g. netarsudil)</td>
<td>++(+)</td>
<td>Conjunctival hyperaemia, headache</td>
</tr>
<tr>
<td>Cholinergic agonists (e.g., pilocarpine)</td>
<td>++(+)</td>
<td>Headache, dim vision</td>
</tr>
</tbody>
</table>

**Laser treatment**

Laser treatment can decrease aqueous production by partial destruction of the ciliary body epithelium, which produces aqueous (Table 2a) or by increasing aqueous outflow through the trabecular meshwork (Table 2b).

### Table 2a Laser treatment to decrease aqueous production

<table>
<thead>
<tr>
<th>Laser</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transscleral cyclophotoagulation (TSCPC)</td>
<td>Typically, diode laser (810 nm) is used. Risk of irreversible hypotony. Therefore, fractional treatment is common.(^{7})</td>
</tr>
<tr>
<td>Endoscopic cyclophotoagulation</td>
<td>Similar to TSCPC, with a better complications profile, but more invasive.</td>
</tr>
<tr>
<td>Micropulse transscleral cyclophotoagulation (MP-TSCPC)</td>
<td>Diode laser (810 nm) with short bursts instead of continuous delivery of laser energy to reduce destruction of adjacent non-ciliary tissue. Might also enhance uveoscleral outflow.(^{8})</td>
</tr>
</tbody>
</table>

### Table 2b Laser treatment to enhance aqueous outflow

<table>
<thead>
<tr>
<th>Laser</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argon laser trabeculoplasty (ALT)</td>
<td>Initial treatment with argon laser trabeculoplasty was at least as efficacious as initial treatment with topical medication (GLT). Risk of scarring of the trabecular meshwork and peripheral anterior synechiae formation.</td>
</tr>
<tr>
<td>Selective laser trabeculoplasty (SLT)</td>
<td>532 nm frequency-doubled Q-switched Nd:YAG laser. Similar efficacy as ALT (LiGHT, KiGIP SLT trials) but less side effects and repeatable.(^{9,10})</td>
</tr>
<tr>
<td>Micropulse laser trabeculoplasty (MLT)</td>
<td>Using 810 nm, 532 nm or 577 nm lasers. Possibly similar efficacy as SLT</td>
</tr>
</tbody>
</table>

---

**CHRONIC OPEN-ANGLE GLAUCOMA Continued**
Surgery
There are several surgical options to reduce intraocular pressure, including a selection of minimally invasive options. ‘Ab externo’ refers to a surgical approach from outside the eye, often involving a conjunctival dissection and scleral incision. ‘Ab interno’ refers to a surgical approach from inside the eye, usually through the anterior chamber, with a corneal incision.

There are three main categories of glaucoma surgery, each with a different purpose:
1. To enhance aqueous outflow into the sub-Tenon space
2. To enhance aqueous outflow through the trabecular meshwork
3. To enhance aqueous outflow through the suprachoroidal space.

1. Surgery to enhance aqueous outflow into the sub-Tenon space
Ab externo approach:
- **Trabeculectomy.** The gold standard, low-cost procedure to create a guarded fistula between the anterior chamber and sub-Tenon space, requires adherence to follow-up. The Moorfields safer technique (i.e., using releasable sutures), is also suitable in low-resource settings.11
- **Goniotomy.** Typically used for childhood glaucoma. The trabecular meshwork is incised under direct gonioscopic visualisation using a goniotomy knife (e.g. 25-gauge needle on a syringe).
- **Iridectomy.** Improving aqueous flow from the posterior to the anterior chamber.

Ab interno approach:
- **iStent.** A 360 μm stent with a central lumen of 80 μm implanted into the trabecular meshwork.
- **Hydrus.** A permanent, 8 mm long, slightly curved microstent to dilate Schlemm’s canal.
- **Goniootomy.** Typically used for childhood glaucoma. The trabecular meshwork is incised under direct gonioscopic visualisation using a goniotomy knife (e.g. 25-gauge needle on a syringe).
- **Kahook Dual Blade or Trabectome.** Disposable ab interno trabeculectomy devices to remove parts of the trabecular meshwork.

Evidence for selective laser trabecuoplasty
The LiGHT trial in the UK10 showed that selective laser trabecuoplasty (SLT) as first-line treatment of ocular hypertension and primary open-angle glaucoma was safe, cost-effective and resulted in the same quality of life (after 3 years) compared to eye drops.

The Kilimanjaro Glaucoma Intervention Programme (KiGIP) SLT trial compared SLT and Timolol eye drops (with standardised counselling) in patients with moderate and advanced glaucoma in Tanzania.11 After one year, SLT treatment was successful in 60.7% of eyes, and Timolol eye drops were successful in 31.3% of eyes. In the SLT group, approximately one third of eyes required one repeat session of SLT; in the Timolol group, a similar proportion needed one repeat session of counselling. Safety, acceptability, vision-related quality of life, and preservation of visual acuity were comparable in both groups after one year. Eye care units in the region using a not-for-profit eye care service model would need to treat around 500 eyes per year with SLT to cover the cost of the procedure, charging an amount similar to one year’s supply of timolol eye drops.

Focus on the patient, not just the eye
The variety of treatment options available makes it much easier to find an approach to suit the individual patient. There are many factors to consider, including those related to the individual and the health system; see the online version of this article (bit.ly/CEHJpoag) for more detail. It is just as important to ensure that people with glaucoma receive counselling to support their compliance with treatment and quality of life, and to refer them for low vision services and rehabilitation as needed – see the rest of this issue for more information.

References
Counselling in a glaucoma care service

Counselling can improve the eye health and quality of life of patients with glaucoma.

Glaucoma patients often think that their condition is synonymous with blindness and disability, leaving them feeling worried and vulnerable. They may also develop mental health issues such as depression or anxiety. Unfortunately glaucoma patients may have mental health issues even if these concerns are addressed. The glaucoma care team must offer patients balanced information that will help them to understand their options, regain hope for their future, and take practical action to protect their eyes and vision. This support – known as counselling – will help to improve patient’s quality of life.

Who should counsel glaucoma patients?

In a typical, busy clinic, some aspects of counselling can be delivered by examining clinicians, supported by other members of the glaucoma care team. However, it is preferable to give patients access to a dedicated counsellor (who may also offer counselling about other diseases and postoperative care).

Ideally, the glaucoma care team should train someone to take on this role. The person must be approachable, be skilled at talking to patients and their families, and must understand glaucoma and its treatment. Nurses, social welfare, or community health workers could be good candidates for training as glaucoma counsellors.

It is vital that clinical personnel on the glaucoma care team are in close contact with the person responsible for counselling and that they share any relevant clinical information (about the patient) with the counsellor. This enables the counsellor to answer any questions the patients may have, and/or to ask the clinicians for more information if anything is unclear.

The purpose of counselling

Counselling can be helpful in several ways, including:

- To help patients understand their condition and accept their prognosis; this may sometimes include coming to accept that the vision they have lost cannot be restored.
- To find out what patients need or want to do and refer them to other services that may help (e.g. low vision or rehabilitation).
- To provide information and help them make a decision about treatment, such as surgery.
- To improve patients’ compliance with their medical treatment (e.g. regularly instilling eye drops in the correct way).

Focusing on the patient and their needs

A list of information can be helpful and complimentary to counselling and is not necessarily the opposite of counselling. Counselling is, by its nature, patient-centred: the counsellor is focused on the patient, what they know and understand, what they want to do, and what they need in order to have good eye health and to live as well as they possibly can.

Patients’ family members and carers should always be included in counselling and it is important to listen to them. Ask them questions and find out how they intend to help the patient to achieve her or his goals. Another reason for including family members is that suffering from vision impairment or blindness without a visible cause such as a white pupil (as in cataract) can lead to alternative explanations, e.g. laziness or curses, and this can result in the patient being stigmatised by others. Red eyes from eye drops can be mistaken for an alcohol problem, for example. Constant and expensive treatment and visits to the glaucoma care team, seemingly without improvement, may be difficult for the patient to explain.

It may be necessary to provide the same information several times, particularly shortly after diagnosis, when an asymptomatic patient may be in denial. We have to be careful, however, as providing the same information, in the same way, may not have the desired effect – patients may stop listening if the information seems over-familiar. Finding out how patients have used the information in their lives, or how it applies to them, makes for a better approach (see Table 1).

Where possible, avoid giving information that sounds like a command. For example, instead of telling a patient to “use your medication or you will go blind”, ask them to tell you what they think the consequences of not adhering to treatment. Listen carefully, then follow up by asking what they think...
they can do to avoid these consequences. After you have listened to the patient, reflect back to them what they have said ("So, what I heard you say is that…"). Then you can explain further or correct any misunderstanding.

Explaining the disease

Take care not to overload patients with facts – only give information that they can understand and apply in practice. Start by asking patients what ideas they have about glaucoma and about the subject of the counselling session (e.g., their own prognosis, acceptance of a surgical procedure, or compliance with treatment), and go on from there.

Glaucoma also has a genetic component, and it is important to discuss this carefully and with great sensitivity. Knowing that glaucoma can occur in many family members is helpful as patients can encourage their relatives to come for screening. However, if glaucoma is seen as a condition that will inevitably lead to blindness and disability, there is a risk of stigma which can lead to family difficulties. For example, it could cause problems in a relationship if there are fears about passing the condition on to children. Therefore, the counsellor must be tactful and guide the discussion towards the identification of first-degree relatives, in particular.

Table 1

<table>
<thead>
<tr>
<th>What to ask patients</th>
<th>What to tell them (adapt this depending on what they have told you)</th>
<th>Why this is important</th>
</tr>
</thead>
<tbody>
<tr>
<td>Can you tell me what you know about glaucoma?</td>
<td>Glaucoma typically develops because of increased eye pressure, often because of a reduced outflow from the eye. Reducing the eye pressure can slow down the progression.</td>
<td>When a patient is diagnosed with glaucoma, it often has a negative impact on their quality of life. It is important to explain the causes, how it progresses, and the patient’s individual prognosis so that they are not unnecessarily anxious but will also take any treatment seriously.</td>
</tr>
<tr>
<td>What symptoms made you come to the hospital? Do you know other ways that glaucoma patients can present?</td>
<td>Glaucoma starts very gradually but will get worse over time – it is a progressive disease. In the early stages there may be no symptoms until there is more damage. Vision impairment, which occurs late in the disease, may be the first symptom that brings most people to hospital, but some can recognise a reduction (constriction) in their visual field and come early. Other symptoms may include dizziness, aches and pain in the eyes, problems with colour vision, proneness to accidents (from stumbling on objects when walking), haloes (rainbow colours) around point light sources, etc.</td>
<td>Explaining to patients what the symptoms are may help them to educate others, so that people with similar symptoms may be more likely to report to hospital early. It can also help patients to talk to their relatives and friends about their specific needs, for example the need to remove obstacles at home.</td>
</tr>
<tr>
<td>What do you know about the differences between cataract and glaucoma?</td>
<td>Surgery for cataract (‘white’ blindness) involves replacing the opaque lens (often visible to the patient) with a clear lens, and vision is restored. With glaucoma (‘black blindness’), treatment (including surgery) stops the vision from getting worse. Vision that is lost cannot come back.</td>
<td>Glaucoma and cataract are often confused and facts about cataract might be more widely known in some regions. This article and a poster from a previous edition of this journal, can be used to explain the difference to patients. &lt;a rel=&quot;noopener&quot; href=&quot;https://www.cehjournal.org/article/what-is-wrong-with-my-vision-and-what-can-i-do/&quot; target=&quot;_blank&quot;&gt;www.cehjournal.org/article/what-is-wrong-with-my-vision-and-what-can-i-do/&lt;/a&gt;</td>
</tr>
<tr>
<td>What, in your opinion is the aim of treatment for glaucoma?</td>
<td>Treatment of glaucoma does not improve vision and sometimes progression can only be slowed down. But with regular visits and treatment, vision can often be preserved. Follow-up and treatment must be pursued life-long.</td>
<td>Many patients expect an improvement of their vision after treatment. Without explanations, they might assume that the condition is treated with a single bottle of eye drops or may stop if a few months of treatment has not improved their sight.</td>
</tr>
<tr>
<td>Is someone else in your family having a similar problem?</td>
<td>Glaucoma may be more common in some families because the disease may be inherited. However, this does not mean that everyone will inevitably have it. This is why it is important for you to advise your first-degree relatives to find time and come to the hospital to check if they have glaucoma. If they come early on, we can treat them before they lose any of their sight.</td>
<td>A positive family history might help the patient to accept the diagnosis and family members can motivate each other to seek counselling and care. Often, a positive family history will help in reducing stigma associated with the disease as other family members have a better understanding of the problem. It also helps the family as a group to reinforce the individual’s management of the disease. It is important to ensure that patients and family members understand what can be done to prevent glaucoma from resulting in vision impairment.</td>
</tr>
<tr>
<td>Do you sometimes see patterns, objects, or people that you know do not exist, or which other people don’t see?</td>
<td>This is known as Charles-Bonnet syndrome, and people who have very advanced glaucoma can be affected. You are seeing these images because of the damage to the nerve at the back of your eyes. You may notice that the images are smaller than you would normally expect; this is typical of the syndrome, and it really is nothing to be concerned about. If you want, I will explain this to your carers and loved ones.</td>
<td>Symptoms of Charles-Bonnet syndrome are caused by cortical stimulation without visual input which leads to visual hallucinations (not auditory, nor olfactory) in people with advanced and end-stage glaucoma. Patients understand that the images are not real and often do not report them because they fear mental illness and possible stigmatisation. The symptoms can also be misinterpreted by those around the patients.</td>
</tr>
</tbody>
</table>

Top tips

- Develop a rapport with the person. There should be a relationship of trust – a therapeutic relationship. Listening is vital. Allow the patient to express themselves in their own words. This is also a way of finding out how they understand the situation, and where there may be gaps in their knowledge.
- Explain the disease to them in terms that are easy to understand (see From the Field panel for an example).
- Listen to the patient. Try to understand how they see their disease and its treatment, as well as their interaction with the health system.
- Try and determine what their ‘soft spots’ are – what matters to them? For example, their children, their job, or looking after an older relative? Explaining how adhering to treatment would benefit them and the people or things they care about, may be helpful motivation for them to take action or change their behaviour.
- You may have given them some bad news about vision that is already lost, and which may be lost in future. You must give them time and space to go through the various stages of grief: denial, anger, bargaining, depression and then acceptance. Only when they accept this can they take action.
Encouraging compliance with medical treatment

When discussing eye medication, it is important to ask the patient how often they use their medication and to invite them to demonstrate how they instil it. You can then show them the correct way to do it.

Patients' ability to instil medication correctly may be affected by several factors, e.g., arthritis of the hands, and it is important to suggest adaptations that can help them, or to train family members to do it for them. Family members can also remind the patient when it's time to instil their eye medication, which will help to improve their adherence to medication regimes and improve the effectiveness of their treatment.

The PDF of this article (www.cehjournal.org/article/instilling-your-own-eye-drops) can be printed and given to patients as a guide.

Low vision and rehabilitation

If the patient has vision impairment, refer them for low vision services – low vision services can provide equipment and training to help them make the best use of the vision available to them. Patients may also benefit from rehabilitation services, where available, and can learn skills such as reading Braille or using a keyboard, typewriter, or other adaptive technologies.

Daily living

A counsellor can also support the patient to adapt to their condition and improve their circumstances. Instead of listing the different environmental modifications they can make, it is better to find about their present condition and how they can adapt, depending on their needs. What do they struggle with, and what do they want to be able to do? For example, finding their way from their home to a friend's house, or taking care of their physical appearance (personal grooming).

This discussion should be about concrete ideas that the patient can carry out with what they have available. You can make suggestions, but it is also important to elicit from the patient and their family members or care what they can to achieve that change.

Patients may benefit from joining support groups for people with glaucoma. These are often organised by patients for patients and their relatives, and sometimes they are facilitated by eye care providers such as nurses. Here patients can learn more and share their own experience with others. They can also have a positive impact on someone by, for instance, sharing techniques used in maintaining adherence to medication, procurement of medication, or adapting their environment and lifestyle. Find out what groups are available in your area and encourage patients to join them.

Peer mentoring is another useful strategy. This is where the patient is connected to another person with a similar condition to provide guidance towards developing self-help or personal grooming skills, for example.

Low vision care for patients with glaucoma: there is more you can do!

Some patients with glaucoma will experience permanent vision loss. The correct low vision advice and support will enable them to carry out their daily activities with greater ease and comfort.

People with glaucoma have specific vision-related problems that will affect their daily activities in various ways (Table 1). Learning about patients' needs and challenges is the first step in offering them useful advice on suitable interventions.

First ask questions (and observe) what your clients need and what they now find difficult when performing their daily activities. Use different tests (clinical and functional) to assess distance and near visual acuity, visual field, contrast sensitivity, and light sensitivity. The CEHj article 'When someone has low vision' lists useful methods. Check what support there is at home, at work, at school and in the community. This will help you to advise them on training and interventions. For example, if someone needs to be guided at night when walking to the local shop, it would be helpful to train the client and a family member or friend in a safe way of guiding. Remember to correct presbyopia in older patients before starting other interventions.

What interventions can help?

Here are some ideas you can suggest to your client.

- Sunglasses can help to reduce the effects of glare and improve contrast. Try a few different coloured lenses to find the ones that work best (see Case study 1, opposite).
- Improve lighting. Consider quantity, type, and direction. E.g., try a reading lamp with a flexible arm in a position that avoids creating glare.
- Ensure enough ambient lighting in dimly lit rooms and prevent large differences in lighting levels.
- Reduce glare by closing curtains or changing position so that you have less excess light.
- Add contrasting strips to steps. Line the borders of the garden with bricks painted white.
- Remove clutter and dispose of little-used items in your kitchen.
- Always carry a torch with you.
- Move closer to the TV.
- Use felt-tipped pens, which are bolder and easier to see.
- Enlarge the labels on your medication or colour code them.
Provide (or advise the client to undergo) compensatory visual field training to enable people with visual field defects to improve navigation and avoid obstacles. For example: “Pause regularly when walking and move your head slowly up and down, then from left to right, to scan the area in front of you. For example, if you scan the area before crossing the street, you may notice a car parked on the street corner which you did not see when looking straight ahead. Then you can avoid bumping into it.”

Refer the client to appropriate peer support groups and to counselling services; these can be of real benefit to them.

Other interventions that some (but not all) people need:

- Orientation and mobility training to learn to walk using a white cane, for people who have lost all their vision or have very low visual acuity.
- Magnifiers, after refraction, correction of presbyopia and prescription of glasses has been done (see Case study 2). These can include optical magnifiers (often only low to medium magnification is possible due to the limited visual field) and smartphone apps that magnify. A handheld video magnifier can offer a significantly larger field of view at a given level of magnification and contrast can be enhanced.
- Text-to-speech software, e.g., the free application ‘Non-visual Desktop Access’ (NVDA).
- Reverse telescopes: objects look smaller so that more information fits into a small field of vision (only people with a good distance acuity will benefit).

### Table 1

<table>
<thead>
<tr>
<th>Vision-related problems</th>
<th>Examples of daily activities affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of peripheral (side) vision</td>
<td>Detecting people or objects only when they appear in the central visual field; bumping or stumbling into objects</td>
</tr>
<tr>
<td>Paracentral scotomas or blurred parts</td>
<td>Reading is often slow and words may be missed</td>
</tr>
<tr>
<td>Reduced ability to adapt from light to dark and dark to light</td>
<td>Moving around during sudden changes in lighting is more difficult</td>
</tr>
<tr>
<td>Trouble seeing in the dark</td>
<td>Orientation at dusk or at night is limited</td>
</tr>
<tr>
<td>Reduced (distance) visual acuity</td>
<td>Unable to recognise people in the distance (but may still see small details)</td>
</tr>
<tr>
<td>Light sensitivity (especially to glare)</td>
<td>Working outside when the sun shines is difficult; cannot see well inside with glare from window light</td>
</tr>
<tr>
<td>Impaired contrast sensitivity</td>
<td>Unsteady navigating on uneven terrain; tripping on steps; limited interpretation of facial expressions</td>
</tr>
</tbody>
</table>

Assess for and correct presbyopia in older glaucoma patients before starting other low vision interventions. INDIA

References
3. RNIB. Advice on guiding a blind or partially sighted person. www.rnib.org.uk/advice/guiding-blind-or-partially-sighted-person
Neovascular glaucoma: prevention and treatment

Patients with diabetic retinopathy and retinal vein occlusion are at risk of developing neovascular glaucoma, a blinding and painful condition. Early detection and prompt treatment is vital.

Neovascular glaucoma (NVG) is a devastating type of glaucoma caused when new and abnormal blood vessels block the trabecular meshwork (the tissue that drains fluid out of the eye). The formation of new blood vessels is most frequently caused by diabetic retinopathy (DR) and retinal vein occlusion. However, other atypical causes also need to be considered (see panel).

Presentation

In the early stages of this condition, patients present with symptoms of the underlying disease (diabetic retinopathy is the most common cause) such as blurred vision, floaters, or complete vision loss due to a bleed in the back of the eye (retinal or vitreous haemorrhage). As the disease progresses and the intraocular pressure (IOP) increases, patients can develop severe pain in the eye, headache, a red eye, nausea, or vomiting.

On examination, blood vessels can be seen around the pupil (rubeosis iridis); see Figure 1. Large vessels can be detected using a torch, but smaller vessels can be difficult to detect in the early stages, even when using a slit lamp. In order to detect rubeosis iridis early, it is therefore important to examine the iris before dilation.

The anterior chamber angle is open in the early stages of neovascular glaucoma. However, as the new vessels grow, peripheral anterior synechiae (adhesions of iris to the cornea) develop and these can close the angle, resulting in further increases in IOP. The pupil also tends to be less reactive to light and eventually progresses to a fixed, dilated pupil with abnormal curling of the more pigmented layer of the iris around the pupil margin (ectropion uveae). The new blood vessels are particularly fragile and sometimes bleed spontaneously in the anterior chamber (hyphaema).

Natural history

Disease processes in the eye (DR, retinal vein occlusion, or others) trigger the production of vasoproliferative factors such as vascular endothelial growth factor (VEGF), which in turn promotes the formation of new, fragile blood vessels that are prone to leaking or bleeding. The new blood vessels (Figure 1) appear at the pupil margin and/or anterior chamber angle. Initially, the IOP is normal as the new vessels only partially block the angle; not enough to obstruct outflow and increase the IOP.

As the vessels continue to grow, aqueous outflow is reduced and the IOP increases, although the angle remains open. Eventually, contractile cells are formed and they cause the iris to adhere to the inner part of the cornea in the periphery of the iris (peripheral anterior synechiae), eventually completely obstructing the trabecular meshwork and inducing ectropion uveae at the pupillary border. The anatomical blockage of the trabecular meshwork progresses to a complete closure of the angle (the angle is drawn together like a zipper), and the IOP increases to very high levels. As a result, the corneal endothelial cells cannot maintain the cornea’s transparency and corneal oedema appears, visible as a hazy cornea. The breakdown of the blood-aqueous barrier produces anterior chamber flare and inflammation is seen clinically as ciliary injection and anterior chamber cells.

The patient will experience pain, headache, nausea, and vomiting over days to weeks. The pronounced increase in intraocular pressure also damages the optic nerve, with progressive sight loss in the affected eye. In some cases, the ciliary body becomes progressively ischaemic to the point that it no longer produces aqueous humour and some eyes can then progress to phthisis bulbi (a shrunken, non-functioning eye).

In many patients with diabetes, the severity of the neovascular glaucoma may not mirror the severity of the diabetic disease seen on examination of the eye. For instance, many patients with proliferative DR may have normal IOP and no anterior segment neovascularisation. On the other hand, patients with normal visual acuity may have proliferative DR and severe neovascular glaucoma with pain as the first clinical sign that they...
have diabetic eye disease. Therefore, the clinicians who assess patients with diabetes or other retinal vascular diseases should be familiar with neovascular glaucoma and examine patients to assess for neovascularisation of the anterior segment. Patients with neovascular glaucoma should be referred urgently to ophthalmologists trained in treating glaucoma and the underlying retinal causes.

**Detection**
When examining a patient with, or suspected to have, neovascular glaucoma, ask yourself the following questions.

1. Is the diagnosis neovascular glaucoma, or another type of secondary glaucoma?
2. What is the underlying disease that caused the neovascular glaucoma? The cause is often DR or retinal vein occlusion, but also rule out atypical causes.
3. How high is the IOP? It can be considered severe when the patient develops signs or symptoms of very high IOP (corneal oedema, ocular pain, or headaches).
4. Is the angle opened or closed? See article on gonioscopy within this issue of the journal.
5. What is the visual potential? It can be roughly estimated based on the severity of the macular damage caused by the underlying disease and the damage to the optic nerve caused by glaucoma.
6. What is the life expectancy of the patient?
7. Is the patient in pain or comfortable?
8. Are there underlying systemic diseases that require an urgent referral, such as renal failure or cancer?

Involve the other health care providers who are required to treat the patient (primary care doctor, nephrologist, cardiologist, neurologist, or nutritionist). Advise patients and carers to avoid the overuse of non-steroidal anti-inflammatory agents (NSAIDs) for pain relief due to the risk of gastrointestinal bleeding.

**Treatment**

Treatment is challenging and requires close collaboration between different health professionals. Treatment will often involve a combination of medical treatment as well as laser or surgical treatment (see Table 1). When the angle is still open, early treatment with pan-retinal photocoagulation and intravitreal anti-VEGF is needed. When the angle is still open, early treatment with pan-retinal photocoagulation and intravitreal anti-VEGF will also be needed. It should be emphasised that neovascular glaucoma secondary to DR may be asymmetrical, but tends to be bilateral. The treatment of neovascular glaucoma in the worst eye should not distract clinicians from treating the better eye with pan-retinal photocoagulation.

### Table 1: Treatment of neovascular glaucoma according to clinical manifestations

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>IOP</th>
<th>Treatment to control IOP</th>
<th>Treatment to control neovascularization</th>
</tr>
</thead>
<tbody>
<tr>
<td>New blood vessels (rubeosis iridis) on the iris</td>
<td>Normal</td>
<td>No</td>
<td>Pan-retinal photocoagulation, retinal cryotherapy, or intravitreal anti-VEGF</td>
</tr>
<tr>
<td>or anterior chamber angle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rubeosis iridis with an open angle</td>
<td>High</td>
<td>IOP-lowering drops, if no improvement surgery</td>
<td></td>
</tr>
<tr>
<td>Rubeosis iridis with a closed angle</td>
<td>High</td>
<td>Surgery</td>
<td></td>
</tr>
<tr>
<td>Severe neovascular glaucoma (with severe pain or IOP</td>
<td>Very high</td>
<td>Urgent surgery to lower the IOP, such as a drainage device or a cyclodestructive procedure. Intravitreal anti-VEGF, pan-retinal photocoagulation or retinal cryotherapy will also be needed.</td>
<td></td>
</tr>
<tr>
<td>&gt; 40 mmHg at presentation)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-stage (blind) neovascular glaucoma (no perception</td>
<td>Low to very high</td>
<td>Usually for pain control only, e.g., steroids and cycloplegic eye drops, as well as laser or surgery to lower the IOP.</td>
<td></td>
</tr>
<tr>
<td>of light)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**How to prevent neovascular glaucoma**

All health care providers (doctors, nurses, technicians, nutritionists, health visitors, etc.) should encourage patients to actively control any pre-existing conditions, such as diabetes or hypertension.

1. Encourage patients with diabetes to control their blood sugar levels. Elevated blood sugar levels encourage the formation of the abnormal blood vessels.
2. Advise all patients to have annual eye examinations (more frequent examinations may be required).
3. Promptly treat severe or proliferative DR with pan-retinal photocoagulation (PRP).
4. In patients with retinal vein occlusion, carry out slit lamp examination and gonioscopy to monitor the anterior segment closely (monthly if ischaemic). Look out for iris or anterior chamber angle neovascularisation during the first 3–6 months.
5. If regular anti-VEGF intravitreal injections are used to treat DR or retinal vein occlusion, monitor the patient’s eye closely for 3–6 months after treatment is stopped.
6. Monitor the severity of DR closely after cataract surgery, particularly if there was a posterior capsule rupture during surgery.
Managing the painful blind eye

Although definitions vary widely, a working definition for a painful blind eye is one with a visual acuity of counting fingers or worse that has no realistic probability of recovering function and is accompanied by ongoing chronic pain and ongoing discomfort which has lasted for at least four weeks.

Causes of the painful, blind eye

A painful, blind eye may result from any disease that causes blindness or a phthisical (shrunken, scarred, and non-functioning) eye. Acute causes include chemical or physical trauma, and chronic conditions include corneal decompensation and advanced and intractable glaucoma, especially neovascular glaucoma.

Understanding eye pain

Mechanical, temperature, irritant and inflammation stimuli in the eye are detected by specialised nerve terminals of the trigeminal nerve (called nociceptors) which send signals to the brain in response to injury or damage in the eye tissues. These nerve terminals are part of the peripheral nervous system, whereas the brain and spinal cord form part of the central nervous system.

The signals are interpreted by the brain as different levels of discomfort, including pain, burning, or stinging, and the response can include increased tearing, blinking, protective movements, and verbal expressions. There are two types of eye pain:

- **Physiological (normal) pain** resulting from damage to eye tissues. Inflammation (e.g., from uveitis) can also cause increased sensitisation of the nociceptors, resulting in persistent pain that is exaggerated in comparison to any tissue damage.1,2
- **Neuropathic pain** resulting from damage to the nociceptors or the other structures involved in detecting, transmitting and processing pain signals between the peripheral nervous system and the brain. This abnormal signalling response can cause sensations of discomfort and pain in response to non-painful stimuli.1,2

How to manage a painful, blind eye

There are very few evidence-based approaches for managing the painful blind eye. Caregivers should try to differentiate between physiological pain and neuropathic pain, but this can be challenging.

Physiological pain. Pain that responds to anaesthetic eye drops, such as proparacaine hydrochloride, or steroid drops, implies that this is physiological pain originating in the peripheral nerves. Physiological pain can be reduced by treating the underlying cause of the pain and/or by reducing inflammation.

Neuropathic pain. Eyes that don’t respond to anaesthetic drops, or steroid drops in the case of ocular inflammation, suggest a neuropathic type of pain which can be very difficult to treat. Systemic, or even psychological, interventions may be required (ideally via a specialised pain management clinic, although these are not widely available).

Whatever the cause of the pain, the most important management aim is to reduce, or help the patient cope with, the pain. Possible methods of management are outlined below. Alternatives that can be safely performed in the clinical office without the need for a surgical room are classified as “non-invasive” in this article.

Treatment of the underlying cause is important, it is also essential that the patient understands the irreversible loss of visual function; this will help when discussing some of the management options, particularly the more invasive types of treatment.

Figure 1 Neovascular glaucoma often results in a painful blind eye. This eye has a collapsed anterior chamber with angle closure resulting from neovascular glaucoma secondary to proliferative diabetic retinopathy.
Non-invasive treatments
The first line of treatment usually consists of topical eye drops that may include lubricants, anti-inflammatory drugs (both steroidal and non-steroidal) as well as immunomodulators.

Eye drops containing atropine (a cycloplegic) are commonly used to reduce possible ciliary spasm. A combination of pressure-lowering, steroid, and cycloplegic drops used in the long term can help to reduce pain for patients with a painful blind eye resulting from neovascular glaucoma.

Steroid drops, lubricants and, in some cases, therapeutic contact lenses are useful for patients with corneal issues, for example, a painful blind eye after multiple failed corneal transplants.

Retrobulbar injection of absolute (100%) alcohol to destroy the sensory ciliary nerves has also been used as a pain management option.2

Systemic drugs, including antidepressants and anticonvulsants, can be used for some types of neuropathic corneal pain. These would be given in collaboration with a family doctor or at a pain clinic.

Invasive treatment
Where a high intraocular pressure is likely contributing to the pain, transscleral and endocyclophotocoagulation, high-intensity focused ultrasound (ultrasound cycloplasty),3 and cyclocryotherapy4 can be used. Destruction of the ciliary body and reduced aqueous production is the common mechanism of action and they offer the advantage of repeated treatments. Cyclocryotherapy is appropriate where vision has been lost completely.

Where the cause of the pain is a damaged or non-healing corneal surface, procedures such as an amniotic membrane graft or conjunctival advancement (Gunderson) flap will bring relief.

Surgical correction and even implantation of electrode devices have been used for neuropathic ocular surface-related disorders.

Evisceration (removal of the eyeball contents while leaving the sclera, the outer layer of the eyeball behind) and enucleation (complete removal of the eyeball) are the final alternatives that can grant definitive relief.4

Conclusion
An understanding of the underlying mechanism, and awareness of the different treatment options, will help direct the best approach for individual patients and achieve sustained pain relief.

Table 1 Summary

<table>
<thead>
<tr>
<th>Common clinical presentations</th>
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<tr>
<td>- Following trauma (e.g., mechanical, chemical, thermal)</td>
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<td>- Severe glaucoma, especially neovascular glaucoma (common causes include diabetic retinopathy and retinal vein occlusion)</td>
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<td>- Endophthalmitis</td>
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<td>- Microbial keratitis</td>
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<td>- Uveitis</td>
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<th>Non-invasive treatment options</th>
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<tr>
<td>- Drops to lower the intraocular pressure (if it is elevated)</td>
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<tr>
<td>- Steroid eye drops</td>
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<td>- Oral analgesics including NSAIDs or paracetamol</td>
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<tr>
<td>- Therapeutic (bandage) contact lens for corneal surface damage</td>
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<tr>
<td>- Retrobulbar injection of alcohol or chlorpromazine</td>
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<tr>
<td>- Oral neuropathic pain medication (e.g., amitriptyline or gabapentin)</td>
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<tr>
<th>Invasive treatment options</th>
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<tr>
<td>To lower an elevated intraocular pressure:</td>
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<tr>
<td>- Transscleral or endocyclophotocoagulation</td>
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<tr>
<td>- Ultrasonic cycloplasty</td>
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<tr>
<td>- Cyclocryotherapy (when vision has been lost completely)</td>
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<tr>
<td>Corneal protection surgery</td>
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<tr>
<td>- Amniotic membrane graft</td>
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<tr>
<td>- Conjunctival advancement flap</td>
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<tr>
<td>As a last resort: eye removal</td>
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<td>- Evisceration</td>
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<td>- Enucleation</td>
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References
Minimum requirements for a glaucoma programme

A successful glaucoma health care programme must provide a timely diagnosis as well as life-long monitoring and treatment of glaucoma.

Glaucoma is a chronic non-communicable disease. A glaucoma care service can be most effective when it considers every aspect of the care pathway: from educating the patient who did not even know about glaucoma, to the patient receiving treatment and then maintaining compliance with treatment. The authors have previously developed a conceptual framework for the glaucoma care pathway (Figure 1). The conceptual framework for an optimal glaucoma care pathway considers engaging patients in glaucoma care from the community to the hospital and imagines that patients would take certain steps to avoid blindness. It takes into consideration important details about patients’ experiences such as awareness, uptake of health care services, and engaging with their glaucoma care. Understanding the patient’s journey will aid developing improved patient interaction processes that help promote earlier diagnosis as well as uptake of, and compliance with, treatment for glaucoma – with the aim of preventing vision loss and blindness from the disease.

The six stages needed for good glaucoma service delivery are:
1. Raising awareness
2. Access to care and earlier detection of glaucoma
3. Reaching/making a diagnosis
4. Accepting and choosing treatment
5. Compliance with treatment
6. Follow-up and monitoring to detect and treat disease progression.

All of these must be part of an integrated eye care set-up and not delivered in isolation. There is generally poor awareness of glaucoma as a potentially blinding eye condition. Raising awareness among the general public involves a sustained effort to educate the public and boost their knowledge about glaucoma. We suggest providing targeted and appropriate messages via:
- Mass media (e.g., radio, TV, newspapers, information leaflets, magazines, etc.)
- Social media
- Health promotional activities, such as screening for non-communicable diseases
- Special awareness sessions during global events such as World Glaucoma Week or World Sight Day
- Interactive forums, both online and in the community, where patients can learn about their condition and ask questions.

With better health education and awareness, patients can develop the confidence to accept and adhere to treatment.

### Figure 1 A conceptual framework for the glaucoma care pathway

- **Natural history**
  - **Gradual visual loss and progression to blindness**
  - **How people get into the care pathway**
  - **What keeps patients in the care pathway**
  - **Optimal care pathway**
    - Knowing, access to eye care, accepting treatment, maintaining treatment

- **Why patients drop out of the care pathway**
  - **Not knowing glaucoma**
    - Not knowing the disease
    - Not understanding the possibility of further vision loss
    - Offered alternate non-medical therapy
  - **Not getting a diagnosis**
    - Not getting a diagnosis
    - Not knowing where to find care
    - Far distance from hospital
    - Lack of access to information
    - Hospitals not adequately equipped
    - Late diagnosis
  - **Not accessing on-going care**
    - High cost of care
    - Not understanding the treatment
    - Unrealistic medical instructions
    - Multiple opinions in different hospitals
    - Loss of economic productivity

- **Current situation**
  - Late diagnosis; treatment non-compliance; failed or delayed follow-up

FDR = First-degree relative; TEM = Traditional eye medicine
To improve patients’ access to care, community and primary health care workers can be trained to identify patients with, or at risk of, glaucoma. They should also be involved in increasing awareness among patients and monitoring their compliance with treatment and follow-up. Patients at risk include older adults, first-degree relatives of patients with glaucoma, and people with high intraocular pressure (IOP). It is useful to have a family screening database (or software) to monitor first-degree relatives.

At primary level, a technician can be trained to screen for glaucoma. At the L V Prasad Eye Institute in India, at primary or vision centre level, the Van Herrick test – along with applanation tonometry readings and non-mydriatic images of optic discs – are transmitted to the telemedicine centre for glaucoma detection. Additionally, follow-up care and compliance with therapy are monitored at this level.

The toolkit for glaucoma management in Africa describes the glaucoma care team according to competencies. Depending on their level of competency, members of the eye care team should be able to identify those at risk of vision loss due to glaucoma, provide care for patients with diagnosed and stable glaucoma, initiate treatment, and continue appropriate care for patients with glaucoma in order to prevent vision loss.

Other approaches to earlier detection include systematic population screening and opportunistic case-finding.

Secondary and tertiary eye care facilities should be adequately strengthened to enable them to reach a diagnosis of glaucoma. This requires appropriate equipment, trained, skilled personnel, and good information and management systems. Basic examination includes assessment of the optic nerve head, indentation gonioscopy for anterior chamber angle examination, measurement of central corneal thickness, visual field assessment, and optic disc imaging.

For details of equipment – refer to the IAPB essential list for glaucoma which also categorises the equipment as ‘essential’ or ‘desirable.’

Once a diagnosis of glaucoma is made, the patient and care provider are encouraged to ensure that treatment starts as soon as possible and continues for as long as needed. The choice of treatment should be based on the risk that a patient’s vision loss will progress. Consider the following:

- Stage of disease
- Sociodemographic and economic profile of the patient
- Family history of glaucoma or vision loss
- Systemic and ocular co-morbidity.

To enable maintaining treatment and keeping the patient in the care system, the following are important:

- Service responsiveness. This includes a pleasant hospital experience.
- Counselling. Inform the patient about the natural history of the disease, the irreversible vision loss it causes, the available interventions and purpose of treatment, and the need for long-term follow-up (including hospital visits).
- Cost of care and affordability, opportunity costs and loss of economic productivity should also be discussed.

Patient participation in their care. The knowledge shared during counselling empowers patients to choose the most appropriate treatment through a shared decision process with the health care provider. Patient forums are also useful to encourage patients’ representation and contribute to how they engage in care. For example, through glaucoma patients’ groups and feedback, they can discuss individual concerns and suggest how the clinic/counselling spaces are organised. Patient groups may also provide peer support, e.g. patients can talk about obtaining medicines and taking them.

Follow-up care is required for monitoring and optimising treatment in response to the progression of the disease. Active mechanisms for contacting patients for follow-up include the use of:

- Clear follow-up instructions and provision of appointment dates
- Keeping patients contact details for texting/calling for reminders
- Glaucoma ambassadors – volunteers within the community who encourage patients in their own care.
Minimally invasive glaucoma surgery (MIGS) has emerged in the past few years as a relevant therapeutic option for glaucoma. Intraocular pressure (IOP) reduction is still the only proven treatment to halt glaucoma progression. Intraocular pressure (IOP) reduction is still the only proven treatment to halt glaucoma progression. This has been traditionally achieved by both nonsurgical means (topical medications or laser therapy) and surgical means (trabeculectomy or glaucoma drainage devices). None of these methods are ideal: compliance is the main issue for medications and surgical complications are common. The high safety profile of MIGS allows it to be used earlier than conventional types of glaucoma surgery within a glaucoma treatment plan, and is typically combined with cataract surgery in patients with mild to moderate primary open-angle glaucoma (POAG).

MIGS usually involves the use of a small device that is inserted or placed through a clear corneal incision approached from inside the eye (ab interno).

Minimally invasive glaucoma surgery (MIGS) devices can be helpful in managing intraocular pressure in the early stages of glaucoma, thereby reducing patients’ reliance on medication. However, the IOP reduction tends to be small and the devices are expensive.
There are a few key points to bear in mind when considering use of MIGS devices in areas of the world with limited resources for health care."

The benefit is that MIGS tends to be relatively safe and low risk. However, the IOP reduction tends to be small and there is no good evidence for their utility in low- and/or middle-income countries, where patients might be diagnosed with glaucoma at a very advanced stage.

Currently, there are many choices for the glaucoma surgeon where MIGS devices are concerned. They can be divided according to their site of action or placement: Schlemm’s canal, suprachoroidal, and subconjunctival.

1. Schlemm’s canal devices

Trabectome, ELT (excimer laser trabeculotomy), iStent, iStent inject, Hydrus, and KDB (Kahook dual blade)

Schlemm’s canal devices are inserted through an ab interno method with the assistance of a gonioscopic lens, aiming to increase aqueous humor outflow through the conventional pathway. Therefore, the potential effect on aqueous outflow is influenced by the resistance provided by the episcleral venous pressure (Figure 2).

The most common procedures include the removal of trabecular tissue (Trabectome, ELT, KDB) or the implantation of a small device (iStent, iStent inject, Hydrus).

Among the products currently available, randomised clinical trial data associated the Hydrus with greater eye drop-free glaucoma control and IOP lowering than the iStent; however, these effect sizes were small. 3,4

2. Suprachoroidal devices

CyPass and iStent Supra

Unlike the Schlemm’s canal devices, in which aqueous outflow could be affected by episcleral venous pressure, the suprachoroidal space is a potential space that confers minimal resistance to aqueous outflow. It allows aqueous to traverse the sclera directly via the intercellular spaces between ciliary muscle fibres and loose connective tissue of the suprachoroidal space.

At present, there are no suprachoroidal devices clinically available, given that the CyPass MicroStent, despite receiving FDA approval in 2016, was withdrawn from the market after results from a post-marketing study showing accelerated endothelial cells loss. 5 The iStent Supra is still undergoing investigation.

3. Subconjunctival devices

XEN-45 and PreserFlo Microshunt

The subconjunctival space, despite not being part of the physiological outflow pathway, is the drainage pathway most familiar to glaucoma surgeons as it is used in conventional glaucoma surgery. Just like the suprachoroidal space, the subconjunctival space is a potential site which is not limited by the episcleral...
venous pressure; however, aqueous drainage can be compromised by fibrosis and scarring.6

The XEN-45 gel stent is a biocompatible, hydrophilic tube made from porcine gelatin cross-linked with glutaraldehyde. It has been implanted using various techniques (ab-externo/ab-interno, with or without conjunctival peritomy).

The PreserFlo Microshunt is implanted through and ab-externo approach requiring conjunctival dissection. Despite this fact, is has been classified by the FDA as a MIGS device (Figure 3).

Both devices are ‘bleb-forming’: designed to limit or prevent clinically significant postoperative hypotony. On the other hand, this may lead to significant scarring and device failure, the risk of which can be minimised by using antimetabolites and aggressive topical anti-inflammatory therapy in the postoperative period.

Discussion

The overall modest reduction in IOP and generally favorable safety profile of Schlemm’s canal devices make it a welcome option for patients with mild or moderate glaucoma who would like to reduce their medication burden. Suprachoroidal and subconjunctival devices offer the potential of greater IOP reduction. There are no commercially available suprachoroidal devices and they are also potentially associated with unpredictable IOP spikes and hypotony. Subconjunctival devices may fail as a consequence of subconjunctival fibrosis or result in bleb-related complications.

There are a few key points to bear in mind when considering use of MIGS devices in areas of the world with limited resources for health care. Patients may present with very advanced glaucoma, and MIGS devices are likely to be less effective in these group of patients. Also, trials to date have been limited to patients with early to moderate disease.

Conventional glaucoma surgery is still the gold standard for surgical management of glaucoma, and no MIGS device has been compared head-to-head with trabeculectomy or aqueous shunt in a randomised controlled trial.

Finally, MIGS devices are relatively expensive and therefore less likely to be a practical option in countries with limited resources. Some glaucoma drainage devices cost as little as US $50, compared to US $400 or more for any MIGS device; this also doesn’t take into account the extra cost of surgical goniolenses or the steep learning curve/training required for this type of surgery.

More prospective randomised trials, with longer follow-up periods, are required to further evaluate the efficacy and safety of this rapidly evolving field of glaucoma treatment. Further comparative studies between devices would also be helpful to evaluate their relative efficacy.

References


The global challenge of glaucoma

Glaucoma is a major cause of irreversible blindness worldwide. It also results in substantial disability, even before people become blind from it, but remains under-treated globally. In most surveys carried out in high-income countries, over 50% of people found to have glaucoma had not been diagnosed and are therefore not receiving treatment. In low-income and/or middle-income countries (LMICs), this rises to over 90%. This high percentage is because glaucoma is mostly asymptomatic until relatively late in the disease. In LMICs, as many as 35% of people diagnosed with glaucoma are already blind as a result – it is too late for them to benefit from effective interventions that would have prevented vision loss.

Whereas cataract has a one-stop solution (cataract surgery), glaucoma requires more complex management strategies because of its chronic nature and complexity. In the absence of simple and affordable diagnostic and treatment solutions, the global eye health community has not prioritised glaucoma; for example, when VISION 2020 was being developed more than 20 years ago. There are several crucial issues.

First, there is a need to provide effective treatments that prevent glaucoma progression and, maybe someday, restore visual function to those with glaucomatous damage. Lowering intraocular pressure (IOP) slows, and in some cases stops, glaucoma progression, but doing so safely and effectively remains a challenge. The current treatment is often long-term topical eye drops, but poor compliance and ongoing costs are major challenges in low-resource settings. Laser trabeculoplasty, which can be administered in a single session, is an effective strategy that has shown effectiveness in such settings. Unfortunately, it rarely provides lifetime control of IOP. Although there is hope that in the future more effective surgical or laser approaches will provide safe and sustained pressure lowering, more work needs to be done.

Second, individuals need to be monitored to determine whether their glaucoma is progressing so that treatment can be adjusted as needed. Challenges for more remote and resource-limited populations, but home-based monitoring using off-the-shelf technology might become available in the near future. The growth of vision centres in India and elsewhere, staffed by mid-level ophthalmic personnel and supported remotely by ophthalmologists, is an example of how to provide ongoing monitoring and care for people living in remote settings.

Third, affordable and effective screening approaches are needed to enable identification of individuals at risk of sight loss. Major advances in the automated grading of optic disc photographs have led to highly accurate glaucoma diagnoses on the basis of a single photo. Widespread use of screening using fundus imaging, with artificial intelligence-assisted grading, could allow glaucoma to be diagnosed alongside the other major causes of blindness at low cost. Implementation studies are needed to determine how and where to apply these new tools.

Innovation in glaucoma detection and management could catalyse a new care model in which earlier detection and effective long-term IOP lowering, combined with remote monitoring, can prevent unnecessary blindness worldwide. To reach this goal, the global eye care community must include glaucoma in eye care planning, recognising that the patient is a central partner in its management. Many important research questions remain unresolved and require substantial investment and a concerted global effort to answer.
Test your knowledge: glaucoma

Test your understanding of the concepts covered in this issue and reflect on what you have learnt.

We hope that you will also discuss the questions with your colleagues and other members of the eye care team, perhaps in a journal club. To view the activities online, please visit: www.cehjournal.org

Tick ALL that are TRUE

Question 1
Detecting and diagnosing glaucoma, including optic nerve head examination and gonioscopy?

☐ a. Optical coherence tomography (OCT) is essential for making a definite (unequivocal) diagnosis of glaucoma.

☐ b. Gonioscopy is an optional part of the examination of glaucoma patients.

☐ c. Loss of the superior part of the visual field (such as an arcuate scotoma) from glaucoma would usually be associated with thinning/notching of the superior part of the optic disc.

☐ d. Many patients with glaucoma are asymptomatic and may present with irreversible vision loss in one eye.

Question 2
Neovascular glaucoma and managing the painful blind eye

☐ a. Treatment for a painful blind eye due to neovascular glaucoma may include injection of 100% alcohol into the eye to destroy the sensory ciliary nerves.

☐ b. Non-invasive treatment options for a painful, blind eye may include atropine eye drops, steroid eye drops, and oral analgesics such as paracetamol.

☐ c. The most common causes of neovascular glaucoma are diabetic retinopathy and retinal vein occlusions.

☐ d. Severe neovascular glaucoma is an urgent/emergency eye condition any may require intravitreal injection, pan-retinal photocoagulation (PRP) laser, and glaucoma surgery.

Question 3
Managing chronic open-angle glaucoma and running a glaucoma programme

☐ a. All glaucoma patients require treatment which may include drops, laser, or surgery.

☐ b. The purpose of glaucoma care is to preserve the quality of life and livelihood of a person with glaucoma, which includes maintaining their visual function while minimising the side-effects and complications of treatment.

☐ c. Minimally invasive glaucoma surgery is safe and effective at reducing intraocular pressure and is replacing more conventional types of glaucoma surgery (for example trabeculectomy).

☐ d. Selective laser trabeculoplasty has good evidence for its use to treat glaucoma in low- and high-income settings.

ANSWERS

1. a. False. Glaucoma is diagnosed by observing changes in the optic disc, not changes in the visual field.

2. c. False. Glaucoma is not always associated with hypertension.

3. b. False. Glaucoma is not always associated with diabetes.

4. c. True. Selective laser trabeculoplasty has good evidence for its use to treat glaucoma in low- and high-income settings.
PICTURE QUIZ

Select ALL that are TRUE

Question 1
The following features can be seen in the optic disc photo
☐ a. Swelling of the optic disc
☐ b. An extremely thin optic disc rim
☐ c. Parapapillary atrophy
☐ d. Nasal displacement of the central retinal vessels

Question 2
Characteristics of glaucomatous optic disc include:
☐ a. Disc haemorrhages
☐ b. Thinning of the optic disc rim, usually nasally
☐ c. Generalised/focal enlargement of the cup

Question 3
Appropriate next steps in this patient would include:
☐ a. A full ocular examination of both eyes including visual acuity (individual eyes), intraocular pressure measurements, examining the optic disc in the other eye and looking for secondary causes of glaucoma, such as uveitis or trauma
☐ b. Discharging the patient if both eyes have poor vision from advanced glaucoma as nothing more can be done
☐ c. Advising family members to be screened for glaucoma

ANSWERS

1. Answer: b, c and d. There is no optic disc swelling. This disc shows advanced glaucomatous cupping.

2. a and c. Thinning of the optic disc rim is characteristic of glaucoma, but this usually occurs at the superior and inferior poles (see article “The optic nerve head in glaucoma”).

3. a and c. It is very important to do a full examination of both eyes. If the intraocular pressures are high they should be treated urgently. It is very important to stop further glaucomatous damage and visual field loss. If the patient has already developed significant visual field loss they should be referred to peer support groups, counselling services and family to plan how to deal with the implications for the patient and the family. The patient should be made aware of the risks and pressures involved with glaucoma.

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Guidelines

ICO Guidelines for Glaucoma Eye Care
www.icoph.org/glaucoma

The European Glaucoma Society guidelines
Most recent can be accessed at www.eugs.org/eng/guidelines.asp

www.aao.org/preferred-practice-pattern/primary-open-angle-glaucoma-ppp

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Next issue

The next issue is on the theme Technologies in Eye Care
What is glaucoma?

- It refers to a group of eye conditions in which nerve fibres in the optic nerve where they leave the eye are damaged, resulting in irreversible vision loss.
- Damage to the optic nerve in glaucoma is associated with high pressure in the eye.
- It is usually painless, and vision loss is unnoticed until it is quite advanced. Therefore, regular eye health check-ups are essential to pick up glaucoma early.

Essential investigations to diagnose glaucoma

- Visual acuity test to assess overall visual function and to rule out other eye diseases.
- Anterior segment examination using a slit lamp.
- Anterior chamber angle assessment using gonioscopy lenses.
- Tonometry to measure pressure in the eye.

Running a successful glaucoma program

- Spread awareness about glaucoma in the community and enhance access to early detection and care of glaucoma.
- Train primary health workers to identify patients with or at risk of glaucoma.
- Strengthen secondary and tertiary care facilities to enable them in reaching the diagnosis of glaucoma.
- Establish referral mechanisms and facilities to prescribe low vision devices and for visual rehabilitation.