Primary open-angle glaucoma (POAG) is sometimes called the ‘thief of sight’. There is no pain or discomfort, and vision loss is so gradual that people often do not notice it.

POAG cannot be cured: it requires ongoing treatment for the remainder of a patient’s life. Blindness from POAG also cannot be reversed, but it can be prevented if the disease is diagnosed early and treated.

Effectively addressing POAG therefore requires the careful involvement of many different people, including health workers, the patient, non-clinical staff, and health planners.

As eye care practitioners, we must do more than merely diagnose and treat people with POAG; we must gain the trust of patients and show them that we are there to help.

It is important to understand the fears people have about surgery, which is often the most certain way to preserve the sight of people with POAG. Many people dread the words ‘surgery’ or ‘operation’ but will also struggle to afford eye drops or come to the eye clinic for follow-up visits (see the case study on page 71). Unless we address their fears and misconceptions, they will certainly lose their sight.

Patients themselves, and their carers, are extremely important in the successful management of glaucoma, whether by surgery or medical treatment. They must come to the clinic for surgery and follow-up and instil any eye medication needed. Patients also can help to prevent blindness from POAG in their loved ones by encouraging their first-degree relatives (parents, siblings, and children) to come for an eye examination.

It can be very helpful to find out what the community knows and thinks about POAG (page 44). This knowledge enables us to provide appropriate information to patients and their families and ensures that we are effective in raising the public’s awareness of the condition (page 46).

For patients on medical treatment, time is needed to explain the crucial role patients or their carers have to play, as well as all the practicalities (pages 77–79):

• what happens when they are not used
• how and when to instil eye drops
• where to get eye drops, where to keep them, and how much they cost
• how to recognise when their vision is deteriorating
• how to recognise fake drugs.

People who have been diagnosed with POAG will have a lot of new information to absorb, and they may struggle to come to terms with being told that they may lose their sight or – more frequently – with the fact that the sight they have already lost cannot be restored. Patients also have different ways of dealing emotionally with a diagnosis of POAG. They may postpone taking any action, go into denial, or may seek help from other providers, some of whom may have harmful practices.

We should do our best to understand how our patients feel and what they may...
EDITORIAL Continued

be experiencing. We can use these insights to help them deal with the despair and despondency of having vision loss, for example by emphasising the residual vision which can be enhanced (with the help of low vision services and/or community-based rehabilitation) rather than the loss which cannot be retrieved. Strong links with low vision services and community-based rehabilitation is therefore an essential component of glaucoma care.

POAG is very similar to other non-communicable diseases, such as high blood pressure and diabetes, where the disease causes damage before there are any signs or symptoms to observe. As with these conditions, regular measurements have to be made, patients must adhere to advice about the use of medications. Close relatives also may be at risk and have to be examined. We may be able to learn a lot from our colleagues about how they manage these conditions and how we can apply their experience to the management of POAG. For example, patients who are struggling to accept a diagnosis of POAG would find tapping into the strengths of a POAG patient support group to be of great help.

We must give our patients enough of our time, and if our own time is limited, then we should arrange for them to speak with a trained counsellor. This will be someone who understands the condition and the treatments available but who also knows how to listen to patients and help them make the best decisions for their eye health and future vision.

Although the medical and surgical treatment of POAG is the responsibility of ophthalmic staff at the tertiary or secondary level, people working in primary health care have a major role to play in the counselling of patients and their relatives who are also at risk. After diagnosis, they can help to provide support to patients regarding low vision services and the use of medications.

Mid-level personnel with good training and supervision can help by taking regular measurements (intra-ocular pressure and visual fields), capturing and transferring information and images, and making timely referral of patients according to agreed clinical guidelines and protocols.

Optometrists have a special role to play: routine checks for POAG within the presbyopia age group will help with early diagnosis and referral. They could provide

**Facts about primary open-angle glaucoma**

- Worldwide, 45 million people were estimated to have POAG in 2010
- A total of 4.5 million (10%) were estimated to be blind as a result
- Relative to population size, there are up to four times more cases of POAG in people of African origin than in other ethnic groups.

regular monitoring of people who are at risk of developing POAG.

Non-clinical eye care staff also have a very important role to play in glaucoma care and it is important that they are well informed (see panel below).

• Receptionists are the first contact for people with POAG. They may have more time to talk with patients. They also speak the same kind of ‘non-medical’ language as patients, and may therefore have a greater influence over the opinions of patients. If they are informed, less harm is done.

• Records staff must understand why it is so important to have well-kept and accurate records, as these will help to reveal the trend in the progression of POAG.

• Technicians are responsible for maintaining and servicing eye care equipment essential to the provision of glaucoma care. Poorly calibrated or non-functioning tonometers can result in inaccurate test results, which means that patients will receive the wrong care. Tonometers must be calibrated regularly and every user must know how to do this (page 65). A surgical instrument set that is not complete or well maintained can also contribute to poor surgical outcomes. We should encourage entrepreneurial attempts to make technology affordable, including image capture and transfer for diagnosis as well as laser treatment. Eventually, there will be technology to help with early diagnosis of glaucoma, which will also allow patients to self-diagnose, keep accurate records, and track disease progression.

• We need pharmacists who are willing to stock the right glaucoma drugs and know where to procure them. If pharmacists have comprehensive knowledge of what impact glaucoma has on patients, families, and society, they can advocate for and prioritise glaucoma medications for inclusion in essential drugs packages.

Fear of job loss and the perceived stigma of vision loss contributes to the secrecy of some glaucoma patients. We must engage with employers and encourage them to help to reduce the stress for people with glaucoma. They can do so by providing insurance coverage for health checks and treatment, and by offering some assurance of continued employment (even if a change of roles is necessary to suit the person’s reduced vision).

Health planners may find that a strategic approach similar to that used for non-communicable diseases such as diabetes and hypertension may have benefits. This might include conducting surveys, making drugs affordable, and advocating for the inclusion of glaucoma in policies relating to non-communicable diseases and universal coverage.

When POAG is truly made everyone’s business, it will be possible to achieve control and reduce its devastating impact.

What non-clinical staff should know

Susan Lewallen  
Co-director, Kilimanjaro Centre for Community Ophthalmology,  
www.kcco.net

Understanding a few simple facts about POAG will help non-clinical members of the eye care team to work in tune with the clinical staff and to understand what patients are facing.

In particular, it is useful for them to understand how POAG differs from cataract (page 62).

• There are different types of glaucoma so diagnosis and treatment is more difficult than for cataract.

• People with POAG are often unaware that they have the condition until significant vision has been lost.

• Visual loss from glaucoma cannot be reversed; treatment aims to stop or slow the visual loss.

• Because vision lost from POAG cannot be restored, early detection is critical.

• Accurate diagnosis of POAG in the early stages can be difficult, even for highly-trained ophthalmologists.

• Treatment may involve surgery or eye drops.

• Eye drops, if used, will have to be used regularly and for the remainder of the patient’s life.

• Surgery will not restore vision; it stops or slows down the further loss of vision.

• By comparison to cataract surgery, patients need much closer follow-up after glaucoma surgery.

• ‘IOP’ in the patient record refers to the pressure in the eye, or the ‘intra-ocular pressure’.

• IOP is important in diagnosis and management of glaucoma, as the aim of treatment is to lower the IOP.

• POAG is a chronic condition, like high blood pressure or diabetes. Patients will need care for the rest of their lives and keeping accurate records is vital.

The patient’s perspective

Aruna’s story

When I was first diagnosed it hit me hard. I thought – “Oh no, it cannot happen to me.” I was really quite distressed; I didn’t know anything about glaucoma – all I knew was that I could go blind. I’m quite an independent person and I would hate to be dependent on anyone. The thought was very scary. I do have follow-up appointments at the hospital, but I think they should be more frequent than they are. Even at the eye clinic, the staff write down notes, but don’t tell you the information. My daughter, who is a doctor, helped me to find out about glaucoma and the importance of keeping up with my treatment. Having the right information made me feel much better and gives me the independence to manage my condition well.

John’s story

I was diagnosed with ocular hypertension, which then developed into glaucoma. The environment of the hospital and the way health professionals communicate with patients can affect our ability to live with the condition. Staff should be open, explain what is going on and how medicines work, so that it takes the worry out of the situation. One of the great difficulties with glaucoma is that I have no idea how it is progressing. I have to rely on what I am told, so I want to ask questions to understand. After all, if I wasn’t worried I wouldn’t ask! I visit two hospitals for visual field tests. In one, the test is done with no explanation while in the other the nurse will take time to go through the results. This is much better. It means I can see the difference for myself and can ask sensible questions of my ophthalmologist during my appointment.

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www.glaucoma-association.com
Mohammed Abdullah is the head of the ophthalmology department at Abubakar Tafawa Balewa University Teaching hospital in Bauchi, Bauchi State, Nigeria, and is studying for a PhD at the International Centre for Eye Health, London School of Hygiene and Tropical Medicine. His field research, conducted in Bauchi, is aimed at understanding how people’s awareness of glaucoma relates to the severity of glaucoma they have when they arrive at the hospital as well as their ability to manage the disease. He spoke to Fatima Kyari.

In general, how advanced is the glaucoma in patients presenting at your hospital?
Most of our patients present with advanced disease. The usual presentation is with one eye blind and the other with moderately advanced disease. We think this is because they are not aware of glaucoma and do not understand how important it is to seek help.

How did you find out what people thought about glaucoma?
We met with a number of patients’ relatives and traditional healers and conducted a series of interviews and focus group discussions. The term ‘glaucoma’ was not known by many people. Those who had heard the term had different ideas about what it meant. There is no local word for the disease. Some knew it as ‘black blindness’, as opposed to ‘white blindness’ (cataract).

Different people had different ideas about the causes of glaucoma. Some thought it was caused by excessive crying, excessive anger, or excessive exposure to firewood smoke. Others related it to black flies or confused it with river blindness. Some understood it as hypertension of the eye. Very few people related it to death of the nerve of the eye.

What prevents people from coming forward early enough?
The main reason is the lack of symptoms. This, together with poor awareness of the existence of the disease, means that people wait until the eye is blind or there is substantial loss of vision before they come.

Also, people in this area know about cataract and tend to wait until the cataract is mature before they come for an operation. So those experiencing some sight loss often assume they have cataract and by the time they come it is too late to save their sight.

People also waste a lot of time seeking alternative or traditional medication. This is because, in many communities, traditional healers are more easily available and accessible. They come to the patient’s house and offer their services, which the patient is often unable to refuse because the healers are recommended by influential relatives and they may initially offer free service or service on credit.

Treatment provided by traditional healers can sometimes cause physical harm, but perhaps the most serious consequence is that this will cause a delay in patients seeking and receiving the correct medical treatment. The consequence of this delay is very often frustration for the health worker, grief for the family, and irreversible blindness for the patient.

How can we encourage people to come forward early enough?
I think the key to getting patients to treatment sooner is to create awareness, emphasising the lack of initial symptoms and urging the people most at risk to go for a check-up. These are people with first degree relatives with blindness or glaucoma, people wearing glasses for myopia, those on long-term steroid treatment, and all adults over 40 years with diabetes, hypertension, or a history of trauma to the eye.

Radio and television can make a big difference. Every time we talk about any disease on TV or radio, our clinics are full of people the next day who either think they have the condition or want to be screened for it. So these kinds of programmes, done often enough, will reach more and more people in the community and will reduce the level of ignorance about the disease in the community.

What form of treatment is most suitable in your area: medication or surgery?
The cost of eye drops is a major barrier against adherence to treatment in our patients. The cost of one month’s treatment with the more effective drugs is more than the minimum wage in the country. Although there are cheaper generic medications on the market, they are not really available here.

We tend to recommend surgery as it is a single procedure. When successful, it maintains the pressure for a long time. However, surgery has its problems too, as...
the rate of failure of the filter is high in African patients and therefore regular follow-up is important. Another problem is that some doctors are reluctant to operate on patients if they have advanced glaucoma because of the risk of wiping out the remaining vision. If a patient does lose their vision after surgery, she or he may attribute that to the surgery, not the severity of the disease. Cases like these may give a clinic and the doctor a bad reputation and drive away patients.

Laser treatment is a good option where available. It is non-invasive, affords reasonable control over a long period of time, and can be repeated. Lately we have been offering diode laser cyclo-photo-ablation – one of the various laser procedures available for glaucoma – and our patients are accepting this more and more as an alternative to surgery and eye drops. The outcome of treatment has been good so far in the group of patients we are following.

How difficult is it to ensure that people come for treatment and follow-up?

Distance to glaucoma clinics is one of the reasons that stops patients coming to hospital. One way to remove this barrier would be to establish outreach clinics in communities, staffed by nurses and allied eye care personnel able to recognise glaucoma and refer patients.

When we investigated why some people did not come for follow-up visits, many patients we spoke to said they were put off by the unsympathetic attitudes of health care workers. They also complained about the long waiting times before being seen, clinic workers’ inability to retrieve their records, and queue-jumping by some patients.

How can we improve patients’ experience?

We must ensure that clinics are organised and that we treat patients on a strictly first-come, first-served basis. We must improve our record-keeping and filing systems, and reduce waiting times (see Community Eye Health Journal Issue 78: ‘Putting patients at the centre of eye care’ and Community Eye Health Journal Issue 74: ‘Ten years to VISION 2020: Why information matters’).

Our communication with patients must also improve. Many patients come to the hospital and receive treatment without knowing what they are being treated for. Health workers must explain patients’ diagnoses to them, say what glaucoma is all about, how it will impact on their sight and life, and discuss the management options with the patients (see page 54).

Other factors that are barriers to treatment – such as the availability of facilities and drugs for treatment, the distance to such facilities, and availability and cost of drugs – must also be addressed.

For those who are able to afford eye drops, what are the challenges?

Patients sometimes stop using their eye drops because there is usually no improvement in vision and no relief of symptoms (as they had no other symptoms to start with). Therefore, there is little to motivate them to persist with their treatment. Continuing deterioration in vision is an issue even in patients who continue to use their medication as prescribed. When this happens, patients feel that their treatment has failed and they visit traditional healers instead of coming back to the clinic to find out about alternatives, such as surgery.

Traditional healers are often allowed to advertise on television and on local radio, and actively encourage patients to stop treatment. By the time patients come back to us, they are irreversibly blind, with no light perception, after having tried and lost faith in the traditional healers. They come back to us broke, despondent, and desperate for anything to restore their sight.

What can eye care practitioners do to help?

To ensure patients continue with their treatment, we must ensure that they know the following:

1. Their vision may not improve even when the treatment is effective.
2. There may be slight deterioration in their vision over time even when on medication as the treatment is not a cure but a control to delay blindness for as long as possible.
3. Patients should continue with their treatment even if the eye is completely blind. Doing so will prevent them from having a painful blind eye or a discoloured eye from corneal decompensation.

It is important to use strategies that motivate, empower, and commit the patient to change their behavior and improve their control of the glaucoma. We are exploring the use of motivational interviewing, administered by trained counsellors, to achieve this.

Gender and glaucoma

Evidence suggests that women account for about:

• 55% of people with open-angle glaucoma
• 70% of people with angle-closure glaucoma.

There are very few studies on service provision.

• In Tanzania, women account for a smaller proportion of people receiving surgical intervention for glaucoma.

• In Malawi, women who are suspected of having glaucoma are less likely to be referred to secondary facilities for assessment.

What can we do?

• Hospitals should be monitoring the use of medical and surgical services by men and women separately. If you note more men receiving services than women, you should investigate.

• Screening programmes are rare; where they exist, findings for men and women should be assessed separately. Use the findings to revise the programme.

• Qualitative research is needed in a number of settings to understand any differences in how men and women with glaucoma seek help, whether medical or surgical. Use this information to develop gender-specific educational materials and programmes.

• In many settings, coming to hospital or clinic is difficult for women. Programmes should aim to create a more dynamic network to enable follow-up.

Further reading

Abeba T Giorgis
Consultant senior ophthalmologist and glaucoma specialist, Department of Ophthalmology, School of Medicine, Addis Ababa University, Addis Ababa, Ethiopia.
Email: abebatgiorgis@yahoo.com

In Ethiopia, glaucoma is the fifth most common cause of blindness and the disease caused irreversible blindness in an estimated 62,000 people in 2006.1

Due to the nature of the disease, an inadequate and inaccessible eye care service, and a very poor level of public awareness, glaucoma patients tend to come for help after they have become either unilaterally or bilaterally blind.

Even among some health professionals in Ethiopia, awareness and understanding of glaucoma is low. There are many instances of parents being told that their child does not have an eye problem when in fact they are suffering from congenital glaucoma, and I have seen many people with acute angle-closure glaucoma who have been treated for conjunctivitis!

In 2007, a group of volunteers made up of physicians and glaucoma patients set up the Glaucoma Group, with the aim of increasing public awareness of glaucoma. The group is supported by the Ophthalmological Society of Ethiopia (OSE) in collaboration with the Department of Ophthalmology, Addis Ababa University, and the Ministry of Health.

The mass media has been the key to the success of the group’s activities as it is a means of reaching millions of people. The Glaucoma Group has worked with the media on documentaries, discussion programmes, and short ‘advertising’ type messages on both television and radio.

The first glaucoma message was broadcast on television in 2008 and was paid for by Light for the World. There were also programmes and educational messages on national and regional radio stations. We approached the media through the OSE and through Menelik Hospital, where the Department of Ophthalmology is based. The people working in the media were interested in broadcasting health-related programmes and were very helpful.

The Glaucoma Group produced posters, banners, brochures, leaflets, a book about glaucoma translated into Amharic, caps, and T-shirts, which were given to glaucoma patients and their families.

Here are a few examples of the key messages the group communicated through the media at different times.

• ‘Did you know there is an eye disease called ‘glaucoma’ (related to high eye pressure) which is different from trachoma?’ (this was the first message, in 2008).
• ‘Did you know that glaucoma could steal your sight?’
• ‘You have a high chance (likelihood) of developing glaucoma if you have parents, sisters, or brothers living with glaucoma.’
• ‘If you have glaucoma, encourage your family to be checked for glaucoma to prevent your loved ones from losing vision due to glaucoma blindness.’
• ‘Using steroid eye drops for a long time increases your risk of glaucoma and cataract.’

Every year, during World Glaucoma Week, trained nurses also provided glaucoma education to patients, relatives, and carers at ophthalmic centres in Addis Ababa, Jimma, and Gondar. The OSE approached drug distributors to fund these activities. The translation of the glaucoma book to Amharic, and the printing costs, were paid for by the author, Josef Flammer of Basel Eye Hospital in Switzerland.

Since these public awareness activities began, most ophthalmologists practising in Ethiopia have noticed an increase in the number of people who come to be examined for glaucoma. Many patients now ask ophthalmologists and ophthalmic nurses whether they are being checked for glaucoma, even if they have come for some other reason (such as getting a prescription for reading spectacles). It has also become quite common to see siblings and children of glaucoma patients coming forward to be examined.

The level of glaucoma awareness among ophthalmic patients at the same tertiary eye care centres has increased from 4% in 2006 to 28% in 2011 (research not yet published). The proportion of patients coming for follow-up appointments, and the numbers who accept medical and surgical treatment, have also increased. Eye care providers have become more aware of glaucoma, some as a result of seeing or hearing something in the mass media, and others because of the questions patients ask them.

Raising public awareness of glaucoma is a key means of addressing this devastating eye condition. In our experience, using the mass media is the easiest and most effective way to do so.

Reference

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The UK National Institute for Health and Clinical Excellence (NICE) published guidelines on the diagnosis and management of open-angle glaucoma in 2009 (www.nice.org.uk/CG85). These are intended to set standards for practice in the UK’s National Health Service (NHS).

As these guidelines are the only strictly evidence-based glaucoma guidelines available, however, they are a resource that can be accessed and used globally. To produce evidence-based guidelines, questions are asked about which diagnostic tests to use, or which treatments to offer to particular patients. High quality evidence is then systematically sought to answer those questions. When none is found, the consensus of the guideline development group is sought. When important gaps in the evidence base are found, recommendations for research are made.

Why were the guidelines needed?
The process of developing guidelines is costly in terms of human resources, time, and money, so there has to be a good reason to produce them. To put it mildly, glaucoma care in the NHS was far from ideal; there were wide variations in practice and standards and, inevitably, in outcomes. Over-diagnosis and missed diagnoses were also widespread, as were over-treatment and under-treatment. Every year, about 1,500 people with glaucoma are registered as blind in the UK. This is despite the fact that medication is available to preserve existing vision and delay or prevent the progression to blindness for most patients.

Limitations of the guidelines
It is important to remember that guidance is guidance, not a rigid protocol, and it is not comprehensive. It is ‘applicable to 80% of cases, 80% of the time’. In addition, as mentioned in other articles in this issue, eye drops are not always a feasible form of treatment in low- and middle-income countries, and therefore the guidelines may not be as widely applicable outside of the United Kingdom or Europe.

The NICE quality standards for glaucoma
In addition to providing guidance to clinicians, it was also important to set standards for the delivery of glaucoma care (http://guidance.nice.org.uk/QS7). This is applicable to the information systems, referral pathways, communication, staff management, etc. needed to provide patients with consistently high levels of care. These standards, although not easily achievable in poorly resourced settings, do give us some ideas about what needs to be in place in our eye care or health care systems if we want to reduce avoidable blindness from glaucoma. Primarily, what is needed is a reasoned and coordinated approach:

‘The quality standard for glaucoma requires that services should be commissioned from and coordinated across all relevant agencies encompassing the whole glaucoma care pathway, including primary, secondary and social care. An integrated approach to provision of services is fundamental to the delivery of high quality care to people with glaucoma. A local register of glaucoma-related conditions, organised according to diagnosis, could be used to facilitate such integration.’

Those most at risk of blindness from glaucoma are those in the most deprived circumstances, including material and educational poverty. For example, in the Caribbean, wealthy people with glaucoma can afford diagnosis and treatment while those on average incomes will have little chance of saving their sight.

The huge challenge in poorer countries and emerging economies is to put in place the requirements for the ‘whole glaucoma pathway’. This is why preventing glaucoma blindness requires VISION 2020 programmes to achieve their highest potential by offering integrated and fully equipped services from primary to tertiary care. Thus the NICE standards are a remote ideal towards which all programmes should strive; the UK National Health Service (NHS) itself still has a long way to go. For example, having local and/or national registers of glaucoma would help with the monitoring of standards, but this requires major infrastructural development. It would require a database that is secure in terms of information governance but also technically sustainable and fully, regularly, and automatically backed up. It must be able to maintain individual records over the 30-year natural history of the disease. However, basic beginnings can make an enormous difference. For example, on the island of Dominica, every person keeps an exercise book containing all their patient records; this is a useful solution when there are few resources.
Detecting possible glaucoma with only limited equipment: a crucial first step

Glaucoma causes irreversible visual loss and must be detected as early as possible. It is therefore vital that all health care professionals are aware of glaucoma when they encounter patients who are most at risk. This includes people who are over 40 years of age and those who have first-degree relatives (siblings, parents or grown-up children) with glaucoma. People are also at higher risk if they wear spectacles for distance vision, if they have had an eye injury in the past, and if they complain about a gradual loss of vision.

There is no single test which can detect glaucoma; the key is to perform a basic eye assessment and to combine all findings to identify people who would benefit from a more comprehensive examination.

In some clinics, particularly at primary level, there is often only limited equipment available. However, by conducting the basic eye assessment described in this article, it will be possible to pick up important clues that suggest a patient is at high risk of glaucoma.

1 Take a history

Early glaucoma usually has no symptoms and the patient will be unaware of the problem. In advanced glaucoma, the person may describe a slow onset of visual loss and/or increasing difficulty avoiding obstacles, despite apparently still seeing well. This is because central vision is frequently preserved.

Ask every patient you see whether they have experienced such gradual loss of vision. Remember that many patients with loss of peripheral vision will not notice it until their glaucoma has become very advanced, and therefore difficult to treat. The visual field loss in each eye may also not be the same, allowing the person’s brain to compose a complete image of the world by combining the two images from each eye (page 66). Some form of visual field testing for each eye separately is therefore advisable, such as confrontational field testing (see opposite and on page 69). You could also ask patients to close one eye at a time and tell you if they notice any differences in what they can see.

Primary open-angle glaucoma has a significant inheritable component and tends to be more common and more severe in people of African origin. A family history of the disease should raise suspicion of glaucoma, and everyone with a parent, brother, sister, or grown-up child with glaucoma should be referred for a comprehensive examination.

In Asia, angle-closure glaucoma is more common. Acute angle-closure is not only very painful but also can be associated with loss of vision and vomiting. Sometimes patients will have had episodes in the past which resolved, and so it is important to ask if they have ever experienced eye pain and headache which came on suddenly, with loss of vision or haloes around lights. Anyone with symptoms which suggest angle-closure glaucoma must be referred urgently for assessment to avoid permanent loss of sight.

2 Observe the patient and talk to relatives

A person with advanced glaucoma may look and move differently. They may have obvious difficulty navigating and avoiding obstacles, especially in unfamiliar places.

People with extensive loss of visual field move slowly and carefully, often holding onto solid objects, such as the back of a chair, until they are certain that their next step is safe. People with advanced glaucoma with only a central island of preserved vision may have a staring gaze. Those with only limited residual peripheral vision may not look directly at your face when talking to you, but will look to the side, or tilt their head. They may find it difficult to see where the visual acuity chart is, for example, and may move their eyes or head around until the chart moves into their restricted field of view.

Relatives may report that the person finds it more and more difficult to move around, or takes a long time to find...
things, such as a piece of fruit on the table, even when it is quite obvious to others where the object is.

All these findings also can be caused by other diseases; however, everyone with such functional visual loss must be referred for a comprehensive eye examination.

3 Test visual acuity
This should always be tested with spectacles on if applicable.
A 1 m Snellen chart, or newspaper headlines, can be used at a distance of 6 m. Another option is to ask the patient to count fingers at different distances. The examiner can use his or her own visual acuity as a reference (assuming it is normal). Visual acuity is always tested for each eye independently.

In glaucoma, central vision, which is what is used for testing visual acuity, can be preserved until the disease is advanced. It is, therefore, vital to realise that normal visual acuity does not exclude glaucoma.

Also, loss of visual acuity can be due to many eye conditions, including refractive error and cataract. As a general rule, everyone with loss of visual acuity in one or both eyes must be referred so that the cause can be determined.

4 Confrontational visual field test
Advanced glaucoma is characterised by visual field defects (although not all visual field defects are due to glaucoma). Confrontational visual field testing (see page 69) can pick up marked visual field defects, particularly in patients whose visual acuity is still good. In confrontational visual field testing the examiner compares the patient’s visual field with her or his own.

5 Assess digital intraocular pressure
Intraocular pressure (IOP) can be estimated by palpating the eye (Figure 1). Ask the patient to close her eyes and look down. Put the tips of both index fingers onto the closed upper eyelid. Keeping both fingertips in contact with the eyelid, apply gentle pressure through the closed upper eyelid, first gently pressing on the eye with the right index finger and then with the left, then with the right again (see picture opposite). A normal eye should feel a bit like a tomato that is just ripe: not solid, nor very soft. It is important to compare the two eyes with each other.

Although experience is needed to assess the intraocular pressure in this way and the method is not very accurate, a very high IOP may be detected as the eye feels abnormally hard and solid. 

End-stage glaucoma
is a useful test if you suspect glaucoma from the history and from visual acuity testing. Every person in whom a high IOP is suspected should be referred for further examination.

6 Use a torch
A simple hand-held torch can provide useful information about the appearance of the pupil, pupillary reactions to light, and the anatomy of the front of the eye.

Pupil appearance
Use the torch to examine the front of the eye and look closely at the shape of the pupil and its response to light. An irregular, poorly responding pupil may suggest the presence of posterior synechiae from uveitis or an old injury, which can both lead to secondary glaucoma. This is sufficient reason to refer the patient for a more thorough eye examination.

Exclude cataract
If the patient has loss of visual acuity in one or both eyes, and the pupil remains black, then cataract is probably not the cause of the vision loss: there must be another reason, such as glaucoma.

Relative afferent pupillary defect
Although glaucoma usually affects both eyes, it is sometimes asymmetric. This can be detected with the relative afferent pupillary defect (RAPD) test – see article on page 58. As other diseases of the retina or optic nerve can also cause a RAPD (e.g. optic atrophy), all patients with RAPD should always be referred for further assessment. Remember: a RAPD is not only caused by a cataract.

7 Use a direct ophthalmoscope
Examination of the optic nerve head (ONH) is a vital part of the assessment for glaucoma, as this disease is characterised by changes in the ONH, which are subtle in the early stages of glaucoma. The ONH can be examined with a direct ophthalmoscope, preferably through a dilated pupil in a dimmed room. See the article on page 55 for guidance on how to examine the optic nerve head.

NOTE: Examination of the ONH with a direct ophthalmoscope can be challenging, for example if the pupil does not dilate well. If patients have a first degree relative with glaucoma, and particularly if they are over 40 or seem to have a visual field defect, you must refer them for further investigation even if you are unable to thoroughly assess the optic disc.

7 Educate and inform about glaucoma
After you have referred your patients, and treatment has been initiated, they may come back to you for possible lifelong follow-up. It is very important to talk to them repeatedly about the irreversible course of the disease, the need for lifelong control of their intraocular pressure, and the importance of regular follow-up visits. Many people are afraid of eye surgery, particularly when the ophthalmologist cannot promise them improved vision.

‘Glucoma is challenging for eye care providers since it is not possible to improve vision’

Talk to patients about the benefits of surgery and, if possible, introduce them to other patients who have had successful glaucoma surgery.

Glucoma is challenging for eye care providers since it is not possible to improve the patient’s vision. A lot of effort has to be invested in keeping the visual status stable in order to avoid blindness. Good communication and listening skills are critical if the patient is to understand their condition and adhere to a long-term treatment and follow-up plan.

If possible, patients with vision loss should be referred to an optometry or refractive error service to ensure they are making the best use of their vision. Some patients may require low vision support or community-based rehabilitation, so ensure that you are aware of these services and can make an appropriate referral.

Reference
The next step: detailed assessment of an adult glaucoma patient

Introduction

The glaucomas are a group of progressive optic neuropathies associated with characteristic structural changes at the optic nerve head (cupping) and corresponding visual field defects. The main modifiable risk factor for glaucomatous optic neuropathy is increased intraocular pressure (IOP). The aims of assessment are:

• quantification of the level of glaucoma damage and functional impairment.

Although there are many possible causes of glaucoma (e.g. trauma, inflammation, previous surgery, or an inherited tendency), it is usually possible to identify the mechanism of elevated IOP by careful history taking, anterior segment examination, and optic disc assessment using a slit lamp.

After assessment, the clinician can group the glaucomatous optic neuropathies into three main categories: primary open-angle glaucoma (POAG), primary angle-closure glaucoma (ACG), and secondary glaucomas (including pseudoexfoliation, pigmentary, uveitic, lens-induced, neovascular, steroid-induced and traumatic).

As a group, the glaucomas are chronic, life-long diseases, and it is therefore essential to collect and record clinical data accurately so that patients with progressive disease or who develop other ocular pathology can be identified at an early stage.

History

Taking a careful history helps in two ways:

1. Identification of risk factors for glaucoma and glaucoma progression.
2. Identification of medical and social factors critical to optimum glaucoma management.

Risk factors for glaucoma

• High IOP
• Age
• Ethnicity (African: POAG, Asian: ACG)
• Positive family history of glaucoma
• Refractive status (myopia and hypermetropia)
• Previous ocular trauma
• Previous intraocular inflammation
• Previous ocular surgery
• Steroid usage.

Risk factors for disease progression

• Family history of glaucoma blindness
• Severe visual loss at presentation
• Previous history of high IOPs.

Medical factors in glaucoma management

• Contra-indications to medications. For example, topical beta-blocker therapy (such as Timolol) is contra-indicated in people with asthma, chronic pulmonary obstructive disease and chronic heart failure.

Van Herick’s technique, step by step

The depth of the anterior chamber measured at the temporal limbus is a good indicator for the risk of angle closure. Van Herick’s technique involves using a slit lamp to estimate the depth of the anterior chamber at the temporal limbus by comparing it with the peripheral thickness of the cornea at this point.

The technique should be performed in a standardised way so that results can be compared at different points in time or between patients.

The steps (Figure 1)

1. Explain to the patient what you are going to do.
2. Dim the lights in the room.
3. Turn the illumination column of the slit lamp to the temporal side, away from the visual axis, by 60°. Some slit lamps can look at this angle (Figure 1).
4. Shine the slit lamp beam from the side at the peripheral part of the cornea and iris (the limbus), where the anterior chamber and iris are just visible. The light must be perpendicular to the temporal limbus, as close as possible to the limbus.
5. View the anterior chamber from the nasal side.
6. Compare the depth of the anterior chamber with the peripheral corneal thickness (Figure 2).

If the depth of the temporal limbal chamber is less than a quarter of the peripheral corneal thickness, then there is a high likelihood (around 84%) that the person has an occludable angle in that eye. If the thickness of the temporal limbal chamber is less than 5% of the depth of the chamber, the likelihood that it is angle-closure glaucoma increases to around 91%.

Further reading

There are two types of indirect goniolenses. The **Goldmann lens** needs a coupling fluid. When indenting, the patient has to look towards the mirror. It gives clear views of 360° of angle with rotation. The **four-mirror goniolens** does not require a coupling fluid. Indentation can be performed in primary gaze, but the lens is unstable on the cornea. Corneal folds therefore develop easily and may reduce the clarity of angle structures.

1. Gonioscopy needs to be done in a dark room with a short slit lamp beam. Shining the beam directly into the pupil should be avoided, as this may change the angle configuration, changing a narrow angle to an open configuration.
2. The mirror is placed at 12 o’clock in order to visualise the inferior angle, which is usually more open.
3. Angles are best graded from anterior to posterior. An anterior landmark which serves as a starting point is Schwalbe’s line (Figure 3): the end of Descemet’s membrane between the corneal endothelium and trabecular meshwork. It can be located with the optical corneal wedge: if a narrow slit beam is tilted showing the cornea in full thickness, the reflections from the anterior and posterior surfaces of the cornea meet at Schwalbe’s line.
4. The goniolens is then rotated to view 360° of the angle.
5. If the iris has a convex configuration and obscures angle structures then the patient can be asked to look towards the mirror.
6. Indentation is helpful if the angle is narrow or closed. If the angle is closed by adhesions, it will not open on indentation (synechial closure). If the angle is closed only by apposition, it will be forced open and reveal the recess on indentation.

## Visual acuity

Accurate measurement of visual acuity (VA) is critical in glaucoma. Distance VA is normal in most patients with glaucoma unless the disease is advanced. Rule out refractive errors if VA is reduced. Reduced best-corrected visual acuity in mild or moderate glaucoma should alert the clinician to an alternative co-pathology (such as cataract, central retinal vein occlusion, retinal detachment, or diabetic retinopathy). Longitudinal analysis of distance visual acuity over time allows the clinician to detect disease progression, cataract, and other problems.

## Visual fields

Testing visual fields to confrontation with a red target (see page 68) can detect significant visual field defects. Simple measures such as tangent screen testing can be effective. Accurate assessment of visual field defects requires visual field perimetry: manual (Goldmann) or automated (Humphrey) perimetry techniques give detailed visual field data. The results of these are dependent on the experience and skill of the person doing the tests, however.

## Slit lamp examination of the anterior segment

A systematic examination of the anterior segment ensures that all important clinical signs are observed.

Gonioscopy is very helpful, however, if a gonioscope is not available, the depth of the limbal anterior chamber can be estimated by Van Herick’s test (see the panel opposite). See Table 1 overleaf for a standardised glaucoma assessment tool.

## Gonioscopy

The anterior chamber angle drains most of the aqueous fluid, hence it is essential to posterior. An anterior landmark which serves as a starting point is Schwalbe’s line (Figure 3): the end of Descemet’s membrane between the corneal endothelium and trabecular meshwork. It can be located with the optical corneal wedge: if a narrow slit beam is tilted showing the cornea in full thickness, the reflections from the anterior and posterior surfaces of the cornea meet at Schwalbe’s line.

Figure 3. Cross-section of the chamber angle. Abbreviation: TM: trabecular meshwork

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Figure 4. Open chamber angle of an African patient viewed with a gonioscope

Schwalbe’s line
Trabecular meshwork
Scleral spur
Ciliary body band

### diseases
- heart block and bradycardia
- Physical difficulty in instillation of eye drops, for example in people with severe hand arthritis, injuries, or poor vision.

### Social factors in glaucoma management
- Ability to afford long-term medication
- Access to a pharmacy to obtain repeat drug prescriptions
- Social and family support
- Contact with other health care workers or traditional healers
- Visual requirements for daily activities
- Level of social deprivation
- Geographical isolation with respect to medical services
- Age or life expectancy.
# GLAUCOMA ASSESSMENT

Continued

Table 1. Standardised glaucoma assessment tool. Abbreviations: XFG: pseudoexfoliation glaucoma; PG: pigmentary glaucoma; UG: uveitic glaucoma; LG: lens-induced glaucoma; NG: neovascular glaucoma; TG: traumatic glaucoma; OSD: ocular surface disease.

<table>
<thead>
<tr>
<th>What to examine?</th>
<th>Why?</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right eye</strong></td>
<td><strong>Left eye</strong></td>
</tr>
<tr>
<td><strong>Visual acuity:</strong></td>
<td>If a reduced visual acuity does not improve after refraction, it is commonly due to cataract or very advanced glaucoma. Alternative or co-pathology must be ruled out</td>
</tr>
<tr>
<td>Uncorrected</td>
<td></td>
</tr>
<tr>
<td>Pinhole</td>
<td></td>
</tr>
<tr>
<td>With correction</td>
<td></td>
</tr>
<tr>
<td>RAPD</td>
<td>Asymmetric glaucoma, previous trauma or inflammation of the anterior segment causing posterior synechiae.</td>
</tr>
<tr>
<td>Anisocoria</td>
<td></td>
</tr>
<tr>
<td>Pupils</td>
<td></td>
</tr>
<tr>
<td>Lid margin</td>
<td>Blepharitis, dry eye as part of OSD</td>
</tr>
<tr>
<td>Conjunctiva</td>
<td><strong>Papillae, Follicles, Inflammation, Oedema, cicatrical disease, vernal keratoconjunctivitis, Bitot’s spot, pterygium, conjunctival growth causing ocular surface disease</strong></td>
</tr>
<tr>
<td>Cornea</td>
<td>Oedema, scars (TG?), infiltrates, keratic precipitates (UG), central vertically distributed pigment deposits on the endothelium (Krukenberg spindle) (PG)</td>
</tr>
<tr>
<td>Anterior chamber</td>
<td>Depth (van Herick), cells (inflammation) (UG), hyphema, vitreous (TG)</td>
</tr>
<tr>
<td>Iris</td>
<td>Transillumination defects (PG), white flake-like material on the pupillary border, absent pupillary ruff, poor dilatation (XFG), pigment dispersion (PG, XFG), heterochromia, iris nodules, posterior synechiae, atrophy, anisocoria (UG), Neovascularisations of the iris (UG, NG)</td>
</tr>
<tr>
<td>Gonioscopy 1° position</td>
<td>Increased trabecular pigmentation (XFG; PG), debris and peripheral anterior synechiae (UG), fine white protein deposits (LG), trabecular neovascular membrane or fine neovascularisations (NG), angle recession, ghost cells, retained foreign body, cyclodialysis cleft (TG). Angle closure.</td>
</tr>
<tr>
<td>Lens</td>
<td>Luxation (TG), irido-phacodonesis (XFG), large lens, hypermature cataract (LG)</td>
</tr>
<tr>
<td>Optic nerve head</td>
<td>Vertical cup-disc ratio, position of vessels, macular degeneration, diabetic retinopathy, retinal detachment, hypertensive retinopathy</td>
</tr>
<tr>
<td>Macula</td>
<td></td>
</tr>
<tr>
<td>Periphery</td>
<td></td>
</tr>
</tbody>
</table>
to assess it in cases of suspected glaucoma. Gonioscopy contributes answers to two questions:

1. What type of glaucoma is it?
2. What is the risk of angle closure?

See the panel on page 51 for practical instructions. The morphology of the chamber angle can be classified using several systems. For example, the Shaffer classification grades morphology from 4 to 0, where:

- Grade 4: ciliary body band visible, angle wide open
- Grade 3: scleral spur can be identified
- Grade 2: trabecular meshwork is visible, angle closure is possible but not very likely
- Grade 1: only Schwalbe’s line visible, high risk for angle closure
- Grade 0: angle closure due to iridocorneal contact.

Other important signs are peripheral anterior synechiae, which occur when the peripheral iris adheres to the trabecular meshwork. Peripheral anterior synechiae should not be confused with iris processes, which usually do not cross the scleral spur. Other features to look for are iris or angle neovascularisation, angle recession, cleft, and pigmentation. The angle should be documented in four quadrants. If there is angle closure, additional manoeuvres such as asking the patient to look towards the mirror or indentation will give additional information (e.g. the presence of a plateau iris configuration or synechial closure).

**Tonometry**

Accurate IOP measurement together with optic disc assessment is the backbone of diagnosis and management of glaucoma. IOP can be measured with applanation tonometry; this is still the gold standard, but it is difficult to get accurate readings unless the examiner is experienced. The applanation tonometer also needs to be calibrated regularly. Other instruments for measuring IOP include the Schiotz tonometer, the tonopen, and the non-contact ‘airpuff’ tonometer. Rebound tonometry may be also an alternative if applanation tonometry is not available, and is very useful in children or at mobile clinics. Normal IOP is below 21 mmHg. However, be aware that patients who return for follow-up visits may remember to use their eye drops just before they come to the clinic, so that the assessed IOP appears to be controlled. This means that the optic disc and visual fields must also be assessed; do not rely on IOP alone.

**Ophthalmoscopy of the optic disc**

Glaucmatous changes to the optic nerve head are central to diagnosing glaucoma and its progression. See page 55 for a detailed guide to identifying a glaucomatous optic nerve head.

**Summary**

Identifying and documenting the cause of glaucoma, as well as the resulting structural changes and functional loss, are key steps in assessing a patient with glaucoma. They allow the clinician to determine if there are any specific modifiable factors and provide information on the severity of the disease to guide the management decisions.

**References**


**Further reading**


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Conversations with glaucoma patients

Hannah Faal
Chairperson: Africa Vision Research Institute, Durban, South Africa.

With a chronic eye condition such as glaucoma, patients and their carers also become ‘eye care workers’. Without their acceptance of surgery or active participation in their own medical treatment, you will not be able to preserve their sight!

Assuming that you have taken into consideration the patient’s personal and financial circumstances (see pages 71–72), other reasons that patients may not follow medical advice include:
- having a poor understanding of the benefits of treatment
- the occurrence of side effects that had not been discussed with the patient
- poor communication or lack of trust between the patient and his or her health care provider.

So it is important to make time to talk and to listen to patients and their carers as often as needed and to respond honestly to any fears and concerns.

‘ICE’ is an easy acronym to help you remember the key areas to cover when talking with your patients. It stands for ideas, concerns, and expectations:

- **Ideas (beliefs)**: what are patients’ ideas about their condition? Knowing these means you can build on what they know already, and/or (gently) correct any incorrect or harmful ideas and beliefs they may have. Ask questions such as: ‘What do you think might be happening?’
- **Concerns**: what are their concerns about the condition, signs, symptoms, treatments, etc. Know what they are and address each one.
- **Expectations**: what are patients’ expectations of staff, or of that particular visit, admission to hospital, or operation?

The lists below give suggestions for what patients and their carers may wish to know. If your clinic is very busy, it may be a good idea to train a nurse counsellor who will be able to spend time talking with patients and carers about the different concerns and questions they may have.

**General**
Talk to patients and carers about:

- their specific condition, its lifelong implications, and the likelihood they will keep their sight, provided they accept surgery and/or adhere to treatment
- the reasons you decided to offer them this particular type of treatment, as well as the risks and benefits
- opportunities for referral: community-based rehabilitation, patient groups, or low vision clinics
- requirements to drive legally.

**Medical treatment**
Talk to patients and carers about:

- the importance of their role in their own treatment
- how to apply eye drops
- possible side effects
- what the treatment will involve, how it will help them, and what sort of improvements they might expect
- what they should do if they have a bad reaction to the eye drops, forget to use the eye drops, or use too much.

**Surgical treatment**
Talk to patients and carers about:

- how long they will be in the hospital
- how much it will cost
- whether they will have any pain during and/or after the operation
- how they can expect their vision to change after the operation
- where they have to go, what day and time, and what they should expect. If possible, call the clinic and make the appointment while the patient is present.

**Monitoring/follow-up**
Talk to patients and carers about:

- how often they have to come back to hospital
- which tests they will need, and what will happen at each visit
- how to recognise when their vision is getting worse, and what they should do.

**FROM THE FIELD**

How I talk to patients with primary open-angle glaucoma

Fatima Kyari is an ophthalmologist in the Department of Ophthalmology, University of Abuja, Nigeria.

She is studying for a PhD at the International Centre for Eye Health, London School of Hygiene and Tropical Medicine, London UK.

Patients, especially those who are newly diagnosed with primary open-angle glaucoma (POAG), have many questions. Consultation times are short, so it can be useful to give patients some generic information about glaucoma in written form.

Even within the short consultation time, however, my usual practice with newly-diagnosed patients is to explain:

1. Basic information about glaucoma; i.e. the pressure within the eye is too high, which damages the nerve at the back of the eye without causing any pain. They will experience a gradual loss of vision if no treatment is given.
2. How we can stop or delay vision loss – a brief explanation of treatment. Options: medicines, surgery, laser
3. The patient’s visual prognosis in terms of treatment and adherence to medications, etc. (see above).

Then I ask the counsellor, if there is one, to explain further.

I would recommend that all busy glaucoma clinics employ a glaucoma counsellor.

Where I work, it seems that most patients would rather hear the initial explanation from the doctor. They are then better able to understand and relate to any further explanation from someone who has more time to counsel or motivate them.

**References**

All types of glaucoma involve glaucomatous optic neuropathy. The key to detection and management of glaucoma is understanding how to examine the optic nerve head (ONH).

This article addresses the following issues:

• how to examine the ONH
• normal characteristics of the ONH
• characteristics of a glaucomatous ONH
• how to tell if the glaucomatous optic neuropathy is getting worse

The ONH can be examined using a direct ophthalmoscope, an indirect ophthalmoscope, or a posterior pole lens with a slit lamp.

Many types of health professional can assess the ONH accurately after having appropriate training. Dilating the pupil makes this easier and will improve the accuracy of the examination, regardless of which instrument is used. Where the equipment is available, more sophisticated techniques such as scanning laser polarimetry, confocal scanning laser ophthalmoscopy, and ocular coherence tomography can also be used to complement the clinical examination of the ONH and provide quantitative measurements.

The time available to view the ONH is often short as the examination is uncomfortable for the patient. It is therefore essential that the examiner has a strategy for making the observations needed to distinguish a glaucomatous ONH from a normal ONH.

Before you start, you should first be able to recognise the characteristics of both a normal and a glaucomatous ONH, and be able to look for additional signs that could indicate a glaucomatous ONH.

Characteristics of the normal ONH (Figure 1)
The ONH, or optic disc, is a round/oval ‘plughole’ down which more than a million retinal nerve fibres descend through a sieve-like sheet known as the lamina cribrosa. The retinal nerve fibres are then bundled together behind the eye to form the optic nerve which then continues towards the brain.

The retinal nerve fibres are spread unevenly across the surface of the retina in a thin layer which has a ‘feathery’ appearance, best seen immediately above and below the disc (Figure 2).

As the nerve fibres approach the edge of the disc they pour over the scleral ring (which marks the edge of the disc) and then down its inner surface. The dense packing of nerve fibres just inside the scleral ring is visualised as the neuroretinal rim. The cup is the area central to the neuroretinal rim. The cup edge (where it meets the neuroretinal rim) is best seen by the bend in small and medium-sized blood vessels as they leave, or descend into, the cup.

Most normal discs are more vertically oval and their cup more horizontally oval.

In addition, most (but not all) normal ONHs obey the ‘ISNT’ rule: the inferior (lower) rim is usually thicker than the superior (upper) rim, which is thicker than the nasal rim (inner, nearest the nose). The temporal rim (outer, nearest the temple) is the thinnest.

Figure 2: Normal optic nerve head of a young African patient.
Characteristics of a glaucomatous ONH

- Generalised/focal enlargement of the cup. (Note that the cup always appears smaller when viewed monoscopically than in stereo)
- Disc haemorrhage (within one disc diameter of ONH) (Figure 3)
- Thinning of neuroretinal rim (usually at the superior and inferior poles) (e.g., Figures 4 and 5)
- Asymmetry of cupping between patient’s eyes
- Loss of nerve fibre layer (Figure 6).

Additional signs which should heighten suspicion of a glaucomatous ONH

- Cup/disc ratio (CDR) ≥ 0.7. A measurement of CDR alone is insufficient and may be misleading as small discs will have smaller cups and hence a smaller CDR. It is important, therefore, to document disc size by measuring the vertical height of the disc. In most populations, only 5% of people with no glaucoma will have a CDR of ≥ 0.7.
- Rim does not obey the ISNT rule
- Presence of parapapillary atrophy (more common in glaucomatous eyes).

Figure 4: Normal optic disc (a) and glaucomatous optic nerve heads of two patients with different severities of glaucoma

a) Normal optic disc
Vertical cup/disc ratio = 0.2

b) Moderate glaucoma (left eye)
Vertical cup/disc ratio = 0.7 with a notch at 1 o’clock. The curve of the vein at 5 o’clock suggests a reduced rim thickness. There is a wedge defect in the retinal nerve fibre layer between 12 and 2 o’clock. There is moderate nasal displacement of the central retinal vessels.

c) Advanced glaucoma (right eye)
Cup/disc ratio = 0.99. The disc (virtually all cup) is pale. No retinal nerve fibre layer is visible. There is significant nasal displacement of the central retinal vessels and parapapillary atrophy.
The hallmark of glaucomatous optic neuropathy is excavation of the neuroretinal rim. Advanced glaucomatous ONH can result in a pale optic disc, but disc pallor should also raise suspicion of another cause such as optic atrophy. A colour difference should not be used to distinguish the cup edge; change in direction of the blood vessels is a more reliable indicator (Figure 5).

The optic disc abnormality should correlate with the visual field defect. Where this is not the case, further investigations (e.g. CT/MRI scan) may be indicated.

The size of the cup always appears smaller when viewed monoscopically rather than stereoscopically.

**Strategy: distinguishing a glaucomatous ONH from a normal ONH**

1. Dilate pupils, if possible and safe to do so.
2. Identify the disc edge and cup edge, and identify the rim.
3. Does the rim thickness obey the ISNT rule?
4. Is there a haemorrhage?
5. Measure the vertical height of the ONH.
6. Estimate the vertical CDR.
7. Examine the retinal nerve fibre layer (using green light). *This may only be possible with a slit lamp and posterior pole lens.*
8. Draw an annotated diagram of the ONH.

**Is the glaucomatous optic neuropathy worsening or progressing? (Figure 7)**

The appearance of any of the features of a glaucomatous ONH, or the exacerbation of these features compared to a previous record, is indicative of a progression/worsening of the disease.

Disc haemorrhages may be present for two weeks to three months and are an important prognostic sign of progression. An accurate record requires careful observation and a detailed drawing, and photographic documentation (preferably stereophotography) is highly recommended.

Other imaging devices offer progression analyses, but these are not a surrogate for a detailed clinical examination.

Progressive worsening of the visual fields should correlate with structural changes at the ONH.

**Further reading**


**Pitfalls and pearls**

- The hallmark of glaucomatous optic neuropathy is excavation of the neuroretinal rim.
- Advanced glaucomatous ONH can result in a pale optic disc, but disc pallor should also raise suspicion of another cause such as optic atrophy.
- A colour difference should not be used to distinguish the cup edge; change in direction of the blood vessels is a more reliable indicator (Figure 5).
- The optic disc abnormality should correlate with the visual field defect. Where this is not the case, further investigations (e.g. CT/MRI scan) may be indicated.
- The size of the cup always appears smaller when viewed monoscopically rather than stereoscopically.

**Figure 5. (left) Glaucomatous optic nerve head of a patient with pseudoexfoliation glaucoma (PXFG).** The demarcation of the cup by the blood vessels differs from the margin between the pallor of the base of the cup and the surrounding pinker colour between this and the disc edge. Focussing on the colour difference is misleading. One should judge the edge of the rim by the change in direction of the small and medium-sized vessels which, in this case, indicates a thinner rim than might be suspected by the colour difference.

**Figure 6. Glaucomatous optic neuropathy: focal enlargement of cup (notch) and nerve fibre layer defect**

**Figure 7: An example of progression of glaucomatous optic neuropathy (left eye) over seven years.**

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How to test for a relative afferent pupillary defect (RAPD)

The ‘swinging light test’ is used to detect a relative afferent pupil defect (RAPD): a means of detecting differences between the two eyes in how they respond to a light shone in one eye at a time. The test can be very useful for detecting unilateral or asymmetrical disease of the retina or optic nerve (but only optic nerve disease that occurs in front of the optic chiasm).

The physiological basis of the RAPD test is that, in healthy eyes, the reaction of the pupils in the right and left eyes are linked. In other words, a bright light shone into one eye leads to an equal constriction of both pupils. When the light source is taken away, the pupils of both eyes enlarge equally. This is called the consensual light reflex.

To understand how the pupils react to light, it is important to understand the light reflex pathway (Figure 1). This pathway has two parts.

1. The afferent part of the pathway (red) refers to the nerve impulse/message sent from the pupil to the brain along the optic nerve when a light is shone in that eye.
2. The efferent part of the pathway (blue) is the impulse/message that is sent from the mid-brain back to both pupils via the ciliary ganglion and the third cranial nerve (the oculomotor nerve), causing both pupils to constrict, even though only one eye is being stimulated by the light.

A positive RAPD means there are differences between the two eyes in the afferent pathway due to retinal or optic nerve disease. If the light used is sufficiently bright, even a dense cataract or corneal scar will not give a RAPD as long as the retina and optic nerve are healthy. Indeed, the test can be used to assess the health of the retina and optic nerve behind a dense cataract, for example.

In glaucoma, if other tests of visual function (e.g. visual fields) are not possible, detecting a RAPD can be very useful as it indicates that there is more optic nerve damage in one eye than in the other, even if the visual acuity in both eyes is equal.

NOTE: If the glaucomatous damage is equal in the two eyes, there will be no RAPD, however severe the damage is.

The swinging light test
In a normal swinging light test (i.e. there is no RAPD) the pupils of both eyes constrict equally regardless of which eye is stimulated by the light (Figure 2). In an abnormal swinging-light test (i.e. there is a RAPD) there is less pupil constriction in the eye with the retinal or optic nerve disease (Figure 3).

Steps
• Use a bright torch which can be focussed to give a narrow, even beam of light. Perform the test in a semi-darkened room. If the room is too dark it will be difficult to observe the pupil responses, particularly in heavily pigmented eyes.
• Ask the patient to look at a distant object, and to keep looking at it. Use a Snellen chart, or a picture. This is to prevent the near-pupil response (a constriction in pupil size when moving focus from a distant to a near object). While performing the test, take care not to get in the way of the fixation target.
• Move the whole torch deliberately from side to side so that the beam of light is directed directly into each eye. Do not swing the beam from side to side around a central axis (e.g. by holding it in front of the person’s nose) as this can also stimulate the near response.
• Keep the light source at the same distance from each eye to ensure that the light stimulus is equally bright in both.
• Keep the beam of light steadily on the first eye for at least 3 seconds. This allows the pupil size to stabilise. Note whether the pupil of the eye being illuminated reacts briskly and constricts fully to the light. Also note what happens to the pupil of the other eye: does it also constrict briskly?
• Move the light quickly to shine in the other eye. Again, hold the light steady for 3 seconds. Note whether the pupil being illuminated stays the same size, or whether it gets bigger. Note also what happens to the other eye.
• As there is a lot to look at, repeat the test, observing what happens to the pupils of both eyes when one and then the other eye is illuminated.

When the test is performed on someone with unilateral or asymmetrical retinal or optic nerve disease, a RAPD should be present (Figure 3). The following happens:

• When the light is shone into the eye with the retinal or optic nerve disease, the pupils of both eyes will constrict, but not fully. This is because of a problem with the afferent pathway.
• When the light is shone into the other, normal (less abnormal) eye, both pupils will constrict further. This is because the afferent pathway of this eye is intact, or less damaged than that of the other eye.
• When the light is shone back into the abnormal eye, both pupils will get larger, even the pupil in the normal eye.
• It doesn’t matter whether you start with the eye you think has the greater problem or the healthier eye: as long as the light is switched from one eye to the other and back again the signs should become apparent.

Sometimes the RAPD is obvious, as the pupil in the (most) affected eye very obviously gets larger when that eye is illuminated. But the signs can be more subtle (see Table 1).

Specific situations
Hippus
Normal pupils, particularly those of young people, sometimes show slight fluctuation in size (of less than 1 mm) even when the light shining into the eye is constant. This is called hippus and it can make eliciting a RAPD more difficult.

Non-reactive pupils
A RAPD can still be detected even if one pupil cannot change size (i.e. it is fixed), because of trauma, posterior synchiae or because dilating or constricting eye drops have been used (Figure 4). Having
established that the pupil of one eye does not change size, regardless of which eye has the light shone into it, concentrate on the eye where the pupil is reactive. Note what happens to the reacting pupil when the light is shone into each eye in turn. Figure 4 shows what happens when the eye with the afferent pathway defect is also the eye with the fixed pupil. If the (more) normal eye is the one with the fixed pupil then, as the light moves from this eye to the other eye, the reacting pupil will dilate.

Asymmetric refractive errors and/or amblyopia
These occur when the vision is poor but the eye itself is normal, and are not associated with a RAPD.

Maculopathy
Unless very severe, this not usually associated with a RAPD and in eyes where the macular damage is sufficient to result in an RAPD, the grade is rarely more than 1–2+ (Table 1). Extensive retinal damage, major retinal vascular occlusion, or retinal detachment, by contrast, can lead to a high-grade RAPD.

Causes of RAPDs
Common causes of unilateral optic nerve disorders that can be associated with a RAPD include ischaemic optic neuropathy, optic neuritis, optic nerve compression (orbital tumours or dysthyroid eye disease), trauma, and asymmetric glaucoma. Less common such causes include infective, infiltrative, carcinomatous, or radiation optic neuropathy. A RAPD is an extremely important localising clinical sign that can be detected by a simple, quick, non-invasive clinical test, provided that the test is performed carefully and correctly.

Table 1. The grading of a RAPD in the swinging light test

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amaurotic</td>
<td>This is seen when one eye has no perception of light. The pupil of this eye only constricts when light is shone into the other eye. When the light is shone back into the eye with no perception of light the pupil rapidly enlarges against the light.</td>
</tr>
<tr>
<td>3–4+</td>
<td>The pupil enlarges as soon as the light is swung from the normal eye into the abnormal eye.</td>
</tr>
<tr>
<td>1–2+</td>
<td>The pupil enlarges, but only after a short delay, after the light is swung from the normal eye into the abnormal eye.</td>
</tr>
<tr>
<td>Subtle/trace</td>
<td>Sometimes the pupils of both eyes can enlarge in the short time interval between shining the light in the normal eye and the abnormal eye. If this happens, the pupil of the abnormal eye may constrict a little bit before enlarging.</td>
</tr>
</tbody>
</table>

References

Figure 2. Swinging-light test – normal (no RAPD)
Illumination of either eye induces normal and equal pupil responses in both eyes (consensual responses).

Figure 3. Swinging-light test – left RAPD
Illumination of the (more) normal right eye causes both pupils to constrict. When the light is moved to the (more) abnormal left eye (e.g. with optic neuropathy), both pupils dilate (constrict less), the left pupil dilating despite the light being shone directly at it. Returning the light to the (relatively) normal right eye results in constriction of both pupils again.

Figure 4. Swinging-light test: left RAPD + non-reactive left pupil
Illumination of the relatively normal right eye causes only right pupil constriction. When the light is moved to the abnormal left eye (e.g. fixed pupil and optic neuropathy), the right pupil dilates (constricts less). Returning the light to the right eye results in constriction of the right pupil again. In this situation it is only necessary to observe the eye with the reactive pupil in order to identify an RAPD.

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How to measure intraocular pressure: applanation tonometry

Sue Stevens  
Former Nurse Advisor to the Community Eye Health Journal; International Centre for Eye Health, London School of Hygiene and Tropical Medicine, London, UK.

Clare Gilbert  
Co-Director: International Centre for Eye Health, and Chief Medical Advisor: Sightsavers, UK.

Nick Astbury  
Consultant ophthalmic surgeon (part-time): Norfolk and Norwich University Hospital NHS Trust.

Unless there is a contraindication (e.g. trauma or corneal ulcer), all adults attending an eye unit should have their intraocular pressure (IOP) measured. Many people with glaucoma have no symptoms and do not know they have the condition. All children who have had cataract surgery should also have their IOP measured at every follow-up visit, if possible. Finding glaucoma early allows treatment to be given which will preserve sight. Although elevated IOP is not the only sign of glaucoma, measuring it is simple and quick to do. Applanation tonometry, using a Goldmann tonometer at a slit lamp, is the preferred method (the ‘gold standard’).

Equipment
- Goldmann tonometer
- Applanation prism
- Disinfectant: isopropyl alcohol 70% or sodium hypochlorite 1%
- Local anaesthetic drops
- Fluorescein strips
- Clean cotton wool or gauze swabs.

Preparation
1. Ensure the prism has been disinfected with isopropyl alcohol 70% or sodium hypochlorite 1%. The prism must be rinsed in sterile water and wiped dry with a clean swab. **WARNING:** residue of the disinfectant may cause a caustic burn on the cornea.
2. Check that the gradation marked ‘0’ on the measuring prism is aligned with the white marker point on the tonometer head.
3. Check that the calibrated dial of the tonometer is set around 10 mmHg.
4. Ensure that the patient is sitting comfortably at the slit lamp: at the correct height, with chin on the rest and forehead against the headband.
5. Set the magnification of the slit lamp at ×10.

Method
6. Instill the local anaesthetic drops and then the fluorescein. Only a very small amount of fluorescein is needed.
7. For measuring the IOP in the right eye, make sure the slit beam is shining onto the tonometer head from the patient’s right side; for the left eye, the beam should come from the patient’s left side.
8. Move the filters so that the blue filter is used to produce a blue beam.
9. Make sure the beam of light is as wide as possible, and that the light is as bright as possible. This makes visualising the fluorescein semi-circles easier (with the slit diaphragm fully open).
10. Ask the patient to look straight ahead, open both eyes wide, and keep perfectly still.

11. With the thumb, gently hold up the patient’s top eyelid, taking care not to put any pressure on the eye.
12. Direct the blue light from the slit lamp onto the prism head.
13. Make sure that the tonometer head is perpendicular to the eye.
14. Move the tonometer forward slowly until the prism rests gently on the centre of the patient’s cornea.
15. With the other hand, turn the calibrated dial on the tonometer forward until the two fluorescein semi-circles in the prism head are seen to meet and form a horizontal ‘S’ shape. The correct end point is when the inner edges of the two fluorescein semi-circle images just touch – see Figure 1.
16. Note the reading on the dial and record it in the notes.
17. Withdraw the prism from the corneal surface and wipe its tip with a clean, dry swab.
18. Repeat the procedure for the other eye.
19. Wipe the prism with a clean, dry swab and replace the prism in the receptacle with just its tip touching the disinfectant.

Figure 1. Applanation tonometry semi-circles viewed through the Goldmann prism

| High intraocular pressure will result in this image. Turn the calibrated dial on the tonometer backwards to reach the accurate end point. |
| Low intraocular pressure will result in this image. Turn the calibrated dial on the tonometer forwards to reach the accurate end point. |
| This is the correct end point – the inner edges of the semi-circles are just touching. This will give an accurate reading of intraocular pressure. |
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What is wrong with my vision, and what can I do?

<table>
<thead>
<tr>
<th>No eye disease</th>
<th>Glaucoma or ‘black blindness’</th>
<th>Cataract or ‘white blindness’</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal vision</td>
<td>Seeing only what is in front, not what is above, below, or on either side</td>
<td>Seeing things as if looking through smoke, mist, or clouds</td>
</tr>
</tbody>
</table>

### Appearance of the eye to others
- Normal vision
- Normal black appearance of pupil
- White appearance of pupil

### Glaucoma
- What can I notice/feel?
  - Nothing initially, not even pain. You may often bump into things, or fall over objects on the ground, because you are losing the outer edges of your vision.
- Who is at risk?
  - People who are 40 years of age or older
  - People with a relative who has glaucoma – a parent, sister or brother, or older child
  - People who wear spectacles to see distant objects, who have had an eye injury before, or who use steroid eye drops.

### Cataract
- What can I notice/feel?
  - Gradual clouding of the vision until the vision has almost gone. No pain. Usually in both eyes.
- Who is at risk?
  - People who are 40 years of age or older
  - People with a previous eye injury
  - People with diabetes
  - People who use steroid eye drops or tablets.
| **Can other family members be affected?** | Yes! Glaucoma can run in the family | Cataract does not run in the family, but other older family members may also develop cataract |
| **How urgently do I need to get help?** | Very urgently! | As soon as possible |
| **What happens if I wait a long time before being treated?** | You may lose your sight, and you will be unable to get it back | It is very likely that you will regain vision |
| **What treatment options do I have?** | Vision can be preserved by lowering eye pressure with eye drops, an operation, laser treatment, or a combination of these options | Vision can be restored with a cataract operation and an artificial lens implant |
| **What long-term treatment may I need?** | Continued use of eye drops or monitoring after the operation or laser treatment at an eye clinic | Usually none. You may need to wear spectacles after the operation |
| **What are the costs?** | Medical treatment: the lifetime cost of eye drops An operation or laser treatment: the cost of an operation and possibly some medication afterwards | One-time cost of the cataract operation |
| **What are the risks of treatment?** | Very few side effects of eye drops. Surgery can have complications, but these can be managed at the eye clinic | Highly successful operation with very few or no complications |
| **What will happen if I stick to my treatment and/or say yes to an operation?** | Your vision will be preserved – you will still be able to see as you did before It will take a lot longer before you go blind, if you go blind at all | Your vision will be better than before |
| **What happens if I do not accept treatment?** | Your vision will gradually worsen and eventually you will become completely blind. This vision is lost forever and can never come back! | Your vision will gradually worsen until you become completely blind. But, at any time, accepting a cataract operation will restore your sight |
| **Will traditional medicine help me?** | No. Delay in obtaining the correct treatment means you are likely to lose even more vision | No. Traditional treatment, known as ‘couching’ (pushing a needle into the eye), can have very serious complications and is not recommended. Eye medication from traditional healers cannot restore sight |
| **What can I do?** | Report to the nearest eye clinic urgently to be examined. If you think a relative may have glaucoma advise them to do the same | Report to the nearest eye clinic to be examined. If you know someone who may have cataracts advise them to do the same |
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How to verify the calibration of Goldmann tonometers

A tonometer is an instrument for measuring the intraocular pressure (IOP), the fluid pressure inside the eye. It is an important test in the evaluation of patients with glaucoma, as damage to the optic nerve is more likely to occur in patients with high IOP. Most tonometers are calibrated to measure pressure in millimeters of mercury (mmHg).

Goldmann tonometry is considered to be the gold standard test for IOP and is the most widely accepted method. A disinfected prism is mounted on the tonometer head and placed against the cornea. The force applied to the tonometer head is then adjusted using a dial connected to a variable tension spring until the pressure in the eye can be determined from the force applied (see page 60 for a detailed description of how to use a tonometer).

Tonometers in busy clinics have been shown to lose accuracy within months of purchase or calibration by the manufacturer. They are more likely to deviate into the positive range, resulting in higher IOP measurements.

It is essential that all eye units develop protocols for calibration checks. Ideally, tonometers should be checked for calibration errors on a monthly basis by individuals who can be held responsible for ensuring their accuracy.

Procedure

The following is the suggested user-level calibration verification procedure for a Goldmann tonometer. Calibration is done at dial positions 0, 2, and 6 (equivalent to 0, 20, and 60 mmHg, respectively).

Please note that this procedure is only intended to verify the accuracy of the instrument. If the tonometer is inaccurate at any of these dial positions, it should be returned to the manufacturer for recalibration.

Before you start

- Insert the prism in the prism holder on the tonometer head and place the tonometer on the slit lamp.

Calibration at dial position 0

- At dial position 0, the feeler arm should be in free movement. If the dial is turned backwards a small distance (to the equivalent of position -0.05), the arm should fall towards the examiner. If the dial is turned forwards a small way (to the equivalent of position +0.05), the arm should fall towards the patient (Figure 1)
  - If the arm does not respond in the above way, the tonometer is inaccurate at dial position 0.

Preparing to calibrate at dial positions 2 and 6

- To check dial positions 2 and 6, the check weight is used. The weight is in the shape of a bar and is normally found in the case with the tonometer prisms or in the drawer of the slit lamp. There are five line markings engraved on the weight. The middle marking represents 0, with 2 on either side of 0 and 6 towards the edges (Figure 2)
  - Line up the adjustable holder with the lines representing either the ‘2’ or the ‘6’ mark on the weight. With the longer end of the bar facing the examiner, slide it into the axis on the side of the tonometer and push it all the way in (Figure 2).

Calibration at dial position 2

- Check that the adjustable holder is lined up with the line representing the 2 mark (Figure 2) and that the longer end of the bar facing the examiner.
- Set the dial to position 2. As with dial position 0, the feeler arm should be in free movement. If the dial is turned backwards a small way (to the equivalent of position +1.95), the arm should fall towards the examiner. If the dial is turned forwards a small way (to the equivalent of position +2.05) the arm should fall towards the patient (Figure 3)

Calibration at dial position 6

- Check that the adjustable holder is lined up with the line representing the ‘6’ mark (Figure 2) and that the longer end of the bar is facing the examiner.
- Set the dial to position 6. If the dial is turned backwards a small way (to the equivalent of position +5.95), the arm should fall towards the examiner. If the dial is turned forwards a small way (to the equivalent of position +6.05) the arm should fall towards the patient (Figure 4)
  - If the arm does not respond in the above way, the tonometer is inaccurate at dial position 6.

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Visual field testing for glaucoma – a practical guide

Part 1 Understanding visual field testing

Examining visual fields is an integral part of a full ophthalmic evaluation. Several methods for assessing visual field loss are available, and the choice of which to use depends on the patient’s age, health, visual acuity, ability to concentrate, and socio-economic status. Available techniques can test the full field (including confrontation, tangent screen, Goldmann perimetry and automated perimetry), or assess just the central field of vision, such as the Amsler Grid (Figure 1).

If undertaking or interpreting visual field tests, it is a good idea to undergo the tests yourself. You will probably find that undergoing a visual field test can be difficult, requiring an understanding of what is required, an ability to perform tasks quickly, and high levels of concentration. Some methods require patients to press a buzzer when they see a target, and this requires quick reflexes and nimble hands. Undergoing the experience yourself will enable you to see how it is possible for patients to be slow and to make mistakes; this will help you to be patient and realise that you may need to explain the test several times. It will also give you greater insight when interpreting the test results, and can be very useful for teaching others how to explain the process to patients.

This article focuses on some of the more practical aspects of visual field testing, with an emphasis on assessing glaucoma.

Key facts about visual field testing

1. Manual and/or automated visual field testing is subjective: it is totally dependent on the co-operation and responses of the patient. Poor results that are difficult to interpret are often due to the fact that the patient either did not understand what was required, or did understand but was unable to respond.

2. Abnormalities in the visual field are a sign of damage anywhere in the visual system from the retina through to the brain’s visual cortex. Visual field defects are, therefore, not limited to glaucoma. It is very important to examine the retina and optic disc carefully to assess whether or not a visual field defect matches the appearance of the disc and retina, or fits with other clinical signs. One should be very wary of the person with extensive field loss, which seems genuine, where examination of the retina and optic disc are normal. This person may have a neurological condition (e.g. a brain tumour) or they may have had a stroke and not have glaucoma at all.

3. The combination of good visual acuities with full visual fields leads to excellent functional vision and both are very important. Loss of visual acuity can be very disabling, but so too can extensive loss of peripheral visual field. Loss of visual field, particularly the lower field, makes walking around difficult and slow, and people can easily lose confidence. Even in advanced glaucoma, when only a small area of peripheral visual field remains, health care workers should do all they can to preserve this remnant of vision, which can be very important for the patient’s independence and dignity.

4. For diagnostic purposes, it is important to test each eye separately. This is because non-congruous defects in each eye (e.g. a superior defect in the visual field of the right eye and an inferior defect in the left eye) could be missed when testing both eyes together as the normal areas of field in one eye overlap the defects in the other eye. This leads to a normal binocular field of vision when both eyes are open (see Figure 1). This is clearly a good situation as far as the patient is concerned, but means that further extensive loss of field can occur without the person being aware of it. So, test the field of vision in each eye separately – at every visit!

5. Early glaucomatous visual field defects are subtle and easily missed. Even with modern automated and sensitive visual field analysers, glaucomatous visual field loss is not evident until at least 30% of the retinal ganglion cell axons that make up the optic nerve have been lost. Progression of visual field loss in untreated glaucoma can be quite slow, and signs of deteriorating disease can therefore be missed quite easily.

6. With all forms of visual field testing, record the patient’s name or identification number, eye tested, visual acuity, the date, pupil size, whether the pupil was dilated, whether the upper lid had to be taped up, and/or if any corrective lenses were used. In addition, comment on patient cooperation, fixation, and perceived reliability in performing the test.

7. There are two major types of perimetry. Kinetic perimetry involves the detection of moving targets and static perimetry involves the detection of a stationary target. Static testing in general is superior to kinetic perimetry in detecting slopes and scotomata.
Visual field loss can be diffuse (as with cataract or corneal opacification), but more commonly there are isolated defects. The visual field defects associated with glaucoma appear to be fairly non-specific, although typical loss fits with the arrangement of the retinal ganglion cell axons within the retinal nerve fibre layer of the retina.

**New technologies and the future**

Other technologies are available and being developed in the hope that formal visual field testing will become easier, more reliable, more affordable, and more widespread – using equipment that can detect glaucoma earlier than standard perimetry. These emerging technologies include:

- short-wavelength (blue–yellow) automated perimetry (SWAP)
- frequency doubling technology (FDT) perimetry
- motion displacement perimetry (MDP).

Over the next few years, it is likely that there will be a wider range of affordable visual field testing equipment available. This is good news since it means that glaucoma detection as well as management can be greatly improved.

Training in visual field testing should be an integral part of ophthalmology residency programmes, as well as of training programmes for mid-level eye-care workers, so that awareness and expertise in glaucoma care become an integral part of the work of eye care workers everywhere.

Careful detection of visual field defects can be diagnostic of many ophthalmic and/or neurological conditions, including glaucoma. In glaucoma – as well as other conditions – it is vital to repeat visual field testing to track any changes over time.

**Part 2 Features of glaucomatous visual field defects**

Visual field loss can be diffuse (as with cataract or corneal opacification), but more commonly there are isolated defects. The visual field defects associated with glaucoma appear to be fairly non-specific, although typical loss fits with the arrangement of the retinal ganglion cell axons within the retinal nerve fibre layer of the retina.

Relatively specific glaucomatous visual field defects (see Figure 3 for examples) include:

1. a nasal step defect obeying the horizontal meridian
2. a temporal wedge defect
3. the classic arcuate defect, which is a comma-shaped extension of the blind spot
4. a paracentral defect 10–20° from the blind spot
5. an arcuate defect with peripheral breakthrough
6. generalised constriction (tunnel vision)
7. temporal-sparing severe visual field loss
8. total loss of field.

Ideally, the same method of testing should be used for baseline and subsequent follow-up. In glaucoma, if visual field loss is progressive, it may mean that control of intraocular pressure (IOP) is inadequate. Be aware, though, that patients may remember to use their eye drops just before they come to the clinic, so that their IOP may appear to be controlled. It is far better to monitor control by assessment of the optic disc and visual fields than to rely on IOP alone.

---

*Figure 2. A normal visual field (left eye)*

*Figure 3. Glaucmatous field defects in left eyes*
The differential diagnosis for visual field loss similar to that seen with glaucoma includes:
- optic nerve head drusen
- retrobulbar optic neuritis
- tilted optic nerve
- anterior ischaemic optic neuritis
- neurological field defects (especially bitemporal homonymous hemianopia)
- other rare optic nerve disorders
- focal retinal disease
- visual field artefacts.

Some non-glaucomatous visual field defects are shown in Figure 4.

**Figure 4. Non-glaucomatous bilateral visual field defects**

![Visual field defects](image)

The first thing you should do is to ask if the patient has noticed a visual field defect. The problem with this approach alone is that early (or even moderate) visual field defects often go unnoticed, particularly if only one eye is affected.

Useful questions to ask are:
- Have you noticed if any part of your vision is missing in either eye?
- Have you noticed any gaps in your vision?
- If you close each eye in turn, does what you see differ from one eye to the other?

In addition, it is essential to enquire about past ophthalmic and medical history, concentrating on family history, dietary/drug/smoking/alcohol history, and whether there are any additional ophthalmic or neurological symptoms.

Visual field testing using confrontation only takes a few minutes, and should be mastered by all eye care workers, since it can provide a lot of useful information. Initially it is best to try and detect gross (absolute) defects. Position yourself in front of the patient, facing her/him with your face level with that of her/his, at a distance of about a metre. A comparison of examiner and patient fields is made, the assumption being that you, as the examiner, have normal visual fields (this is another reason why you should undergo visual field testing yourself).

First test the binocular visual field and then test each eye separately. A defect is detected by the absence of a patient response to the showing of a target, when the target is visible to you.

### Part 3 Confrontation testing

**Testing to confrontation with both eyes open**

1. Ask the patient to stare directly and steadily into your eyes. Staring can cause embarrassment or awkwardness, so allow the patient to rest and try again if they find it difficult to look at you so directly. Check that the patient can look steadily at your eyes while you look steadily at theirs. Ask the patient whether any part of your face is missing or indistinct.

2. Check the patient’s left hemi-field by making a fist with your right hand and holding it in their left hemi-field, at eye level, just to the right of your face. Making sure that the patient is still holding your gaze, raise one to four fingers and ask how many fingers can be seen. To test the upper and lower
quadrants, move your hand up and to the right, and down and to the right, repeating the test at various points. This simple finger-counting test is particularly useful for detecting visual field loss due to neurological problems (such as strokes), but is only useful for patients with glaucoma when the visual field loss is severe.

3 To test the patient’s right hemi-field and upper and lower quadrants, repeat the finger-counting test using your left hand, starting just to the left of your face and moving up and left and then down and left.

4 A useful, additional test to perform in patients with a suspected homonymous hemianopsia (i.e. loss of either the right or left field of vision in both eyes, often from a stroke) is to test for sensory inattention. Hold both hands up and wiggle the fingers of the right hand, followed by those of the left hand in each hemi-field. If the patient sees the moving fingers, then wiggle one finger of each hand at the same time – if the patient can only see movement on one side then they may have a subtle hemianopia.

**Testing each eye to confrontation**

1 Ask the patient to cover their own eye with the palm of their hand (not their fingers, as it is easy to peep between fingers). Remember that you should close your eyes in turn too, so that you are comparing the field in your right eye with the field of the patient’s left eye, for example (Figure 5).

2 Do the finger counting test first (static testing). Be sure to test on both the left and the right for **each eye tested**.

3 Next, bring your target finger from the far periphery in towards the central region (kinetic testing). Ask the patient to say when they first see the target. Repeat from several different directions, ensuring that the full 360° for each eye is tested. The examiner should remember to perform kinetic testing at a speed appropriate for the patient’s responses.

4 Next, test the peripheral field with a white-headed neurological pin (beyond a central 30° radius) and the central field with a red-headed neurological pin (within a 30° radius). Testing with neurological pin targets gives much more accurate results than testing with fingers, and can detect earlier visual field loss. Red-headed neurological pins are also useful for assessing the size of the blind spot (e.g., with papilloedema), again by comparing the size of your blind spot with that of the patient’s. In addition, red-headed neurological pins can be used to test for red-desaturation in early optic nerve disease.

**Part 4 Other visual field tests**

This article does not cover all the details relating to the use of automated devices and users fortunate to have access to such devices should read and carefully adhere to the manufacturer’s instruction manuals. Learning how to fully interpret standard automated perimetry (SAP) visual field printouts is also beyond the scope of this article and the reader is advised to source this information from an appropriate textbook. Readers with internet access can visit the Community Eye Health Journal website (www.cehjournal.org) for an additional online-only article about glaucoma and visual fields.

**Amsler chart testing**

A printed grid (Amsler chart) can be used to detect subtle central defects (uncommon in patients with glaucoma) as well as paracentral defects (fairly common in patients with glaucoma – especially those with normal tension glaucoma). Test one eye at a time, correcting for any near refractive errors. Patients should hold the chart at a comfortable reading distance from their uncovered eye, and stare at the central spot of the grid. Ask them to identify and then point out any areas where the grid is missing or distorted. Missing areas may suggest paracentral glaucomatous visual field loss, whereas distortion is more common with macular disorders (Figure 6).

**Figure 5. Testing visual fields to confrontation. The examiner’s left eye is closed, so he can compare the field of his right eye with the field of the patient’s left eye. TANZANIA**

**Figure 6. Amsler grid, when viewed by someone with normal central vision (a) and by people with a problem with their central visual field (b and c)**

a) Normal central field

b) Small scotoma (defect within a field of vision) below central fixation, with surrounding distortion

c) Large scotoma encroaching on central fixation with some distortion

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**Continues overleaf**

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**Tangent screen testing (campimetry)**

Tangent screen testing uses a flat testing surface and is useful for testing the central visual field, but for testing beyond 30° its value is limited (Figure 7). The tangent screen is usually a black felt screen mounted on a wall and testing is performed while the patient is sitting down. The screen should be well illuminated and appropriate for the specific type of test and target used. Most screens have circular white stitching or markings every 5° from a central fixation spot, up to 30°. The screen also has radial markings around the fixation point that start at the 180° meridian and are usually spaced 22.5° apart.

A disadvantage of campimetry is that no permanent record is generated, however, it can provide a convenient and quick way to obtain an impression of a field defect in a more formal manner than is achieved by confrontation testing.

**Goldmann perimetry**

The Goldmann visual field analyser or perimeter (Figure 8) is a device that enables kinetic visual field testing, is more standardised than tangent screen testing campimetry), and generates a permanent record of the visual field, making it more sensitive, reproducible, and better for detecting change over time. Many of the practical principles of Goldmann perimetry are similar to those relating to campimetry (and are not, therefore, repeated in this section).

The Goldmann perimeter consists of an illuminated hemispheric bowl upon which target spots of light are shone and moved from non-seeing regions to seeing regions. For kinetic testing, the examiner is able to move the target where they choose throughout a test, observe that the patient’s eye is fixating on the fixation spot, communicate with the patient and document boundaries (isopters) between seeing and non-seeing regions to produce an exact drawing of visual fields. For static testing, the test target can be projected statically at a single location and the brightness increased until the patient responds that the target has been seen. The Goldmann device allows control of the luminance of both the background and stimulus targets and it is important that the device is calibrated on a regular basis to ensure that individual visual field plots can be compared with others; see the device calibration instructions. Target size is classified by Roman numerals (I–V), gross intensity of the target by Arabic numerals (1–4) and fine intensity by letters (a–e). Many different target stimuli can be selected (varying in size and luminance) and the II14e target with a diameter of 0.05° and area of 4 mm² has subsequently become a standard target for many types of automated perimeter (see below).

**Standard automated perimetry**

Automated visual field analysers have been developed, but these are expensive and not yet available in all departments throughout the world. However, research has shown that glaucomatous visual field loss is best detected and is managed with high reliability when automated perimetry is performed. SAP machines are highly technical and use intelligent computer software. It is hoped, therefore, that SAP will be used more widely with time.

SAP testing may be performed as a threshold or suprathreshold analysis. With suprathreshold analysis, the intensity of the stimulus target is not reduced to the level of detection/non-detection. A threshold analysis is more sensitive, but takes longer and is more susceptible to detecting artefacts (changes in the data that are due to the testing process itself, and not as a result of actual problems with the visual field).

SAP analysis provides the following:

- reliability indices (test duration, fixation losses, false positive and false negative error scores)
- a pictorial grey-scale plot of the visual field
- a plot of raw data sensitivities for each test spot
- global indices (in dB) indicating how the height and shape of the patient’s hill of vision deviates from normal. Mean deviation gives the average difference between the patient’s overall visual field sensitivity compared to a normal, age-corrected, reference field. Pattern standard deviation gives the standard deviation of the tested spot deviations from normal, thereby providing a measure of the degree to which the shape of a patient’s field differs from normal
- a total deviation plot, together with a probability map indicating the likelihood for each missed point that it is abnormal
- a pattern standard deviation plot, together with its probability map
- analyses of change in visual field sensitivity with time
- results of the glaucoma hemi-field test – a relatively crude test for detecting glaucoma, based on the assumption that glaucoma frequently affects the superior hemi-field more than the inferior hemi-field or vice versa.

The key feature of a glaucomatous visual field defect is an abnormality on the pattern standard deviation plot, which also shows on the total deviation plot. A field defect on the total deviation plot, in the absence of a defect on the pattern standard deviation plot, can be due to glaucoma (diffuse field loss), but is more likely to be due to media opacity (e.g. cataract).

A field defect that appears more extensive on the total deviation plot than on the pattern standard deviation plot may indicate co-morbidity (e.g. cataract and glaucoma).

**Figure 9. A standard automated perimetry printout for someone with advanced primary open-angle glaucoma**
Managing a patient with open-angle glaucoma: a case study

**Case presentation**

Mr AA is a 48-year-old shop attendant who presented at the eye unit of a teaching hospital with a history of gradual, painless vision loss. His presenting (unaided) visual acuity was counting fingers at 1 metre in the right eye and 6/60 in the left eye. Both corneas were clear, and the pupils had a slow reaction to light. There was a right relative afferent pupillary defect (RAPD). The right eye had a nuclear sclerotic cataract which precluded a good view of the optic nerve head, and a vertical cup:disc ratio (VCDR) of about 0.9, barely visible through the dilated pupil with the binocular indirect ophthalmoscope. The left eye VCDR was 0.8. Intraocular pressure (IOP) was 32 mmHg (right eye) and 30 mmHg (left eye) by applanation tonometry. Visual field tests (standard automated perimetry [SAP]) could not be carried out.

**How would the panel manage Mr AA?**

Most of the panelists mentioned the importance of talking to Mr AA about glaucoma and what his treatment options were. Some mentioned asking a nurse counsellor to talk to the patient. The next important issue to be addressed was the setting of a target IOP in the lower teens, and discussing this target with the patient.

There was general agreement that the initial control of IOP should be by medical treatment, while preparing for surgery on the right eye. First choice was a combination of a beta-blocker and a prostaglandin analogue (PGA). A second option was a combination of a beta-blocker and an alpha-agonist. The panel mentioned the need to bear in mind the cost and availability of the drugs.

All panelists agreed that the right eye should be treated first, and firmly recommended a combined procedure: cataract with posterior chamber intraocular lens (PCIOl), and trabeculectomy with adjunctive antimetabolite therapy. The reasons were both clinical and patient related:

“A trabeculectomy alone may give better IOP control, but will likely worsen vision and, depending on the techniques available and how the bleb turns out, going back to take out the cataract could create inflammation and/or directly compromise the bleb and worsen IOP control.”

“Cataract surgery alone is out of the picture, since a serious IOP spike could wipe out remaining visual field and adequate IOP control is not likely to be achieved.”

“The patient will better understand the benefit of surgery [and therefore be more likely to attend further appointments] if he can be offered some visual improvement.”

Depending on the centre and available facilities, the suggested approaches for surgery on the right eye were:

- phacoemulsification with PCIOl and trabeculectomy
- small incision cataract surgery (SICS) with PCIOl and trabeculectomy at a separate site
- extra-capsular cataract extraction (ECCE) with PCIOl and trabeculectomy.

Adjunct therapy could be with:

- beta irradiation applied with a strontium plaque
- mitomycin C (MMC)
- 5-fluorouracil (5FU).

There is some evidence to support using separate sites rather than the same site in combined phacoemulsification and trabeculectomy surgery.

The choice of treatment for the left eye was not so uniform across the panel. Having initiated medical treatment for IOP control, a top choice was to perform a trabeculectomy with adjunct 5FU or MMC. However, some panelists said they would only offer surgery if there was inadequate IOP control with medications; others would also offer laser treatment as an option.

Both eyes would also have refraction, and the patient would be given spectacles if needed.

**Additional comments from panelists**

“Patients are becoming more informed and are like more informed and are likely to seek more information and ask for more choices, regardless of their literacy or socioeconomic levels. Therefore, counselling needs to be more comprehensive, to include the biological situation of the eye and whole body, the patient’s psychological perceptions, their social and economic situation, as well as their religious beliefs.”

“The role of counsellors cannot be overemphasised, as they will take more time to explain to the patient the pros and cons of staying away or declining surgery.”

“The nurse counsellor could keep a register with the patient’s mobile phone number. She could SMS (text) or phone him if he defaults on follow-up.”

“When the mode of treatment is certain and options are limited, like in the case of the right eye, then be firm to recommend that to the patient.”

Continues overleaf ➤
Mr AA was diagnosed with glaucoma and cataract at his initial presentation. At that time he was told he had advanced eye disease and needed to have surgery to preserve his vision. He asked whether the operation would make him see better. He was frankly informed that it would only preserve the vision he had at that time in the left eye; and that, if the cataract was causing much of the poor vision in the right eye, his vision in that eye would improve after cataract surgery.

Medical treatment with eye drops (xalatan and timolol) was recommended, and Mr AA was given one month to make a decision about surgery. He was told to get the prescribed medications in the meantime and to start using them.

Mr AA did not return until six months later. He said that he had bought one bottle each of the eye drops, but could not buy more because they were expensive. He decided not to come back to the clinic because he was sure the doctor would be angry with him. At that stage he decided to see a traditional healer on the recommendation of a close family friend.

When this did not work, Mr AA went to a different eye clinic near his home where he was told he had cataract and needed to go to hospital for surgery. This brought him back to the same eye unit, where he was told he had been away. However, he came back to the clinic because he was sure the person accompanying him had been away. However, he came prepared to have surgery and was admitted for surgery immediately so as not to lose him.

The standard surgery usually offered at the hospital is manual-scrice incision sutureless cataract surgery. Mr AA was initially offered right ECCE and PCIOL, because combined SICS and trabeculectomy can be more difficult to perform. However, the final decision was to offer SICS with PCIOL at a temporal site, and simultaneous trabeculectomy with MMC at a more nasal position. The decision to use MMC was to prevent bleb scarring.

Mr AA’s immediate post-operative unaided visual acuity in the operated eye was 6/60. He was also informed about the importance of adherence to prescribed medication and follow-up after the operation. Mr AA returned for his 1-month follow-up appointment and had a post-operative review of the right eye. His unaided visual acuity was 6/60; the bleb was draining and was not cystic; the IOP was 12 mmHg and he was pleased with his improved visual function.

There was some discussion about what to do about the left eye and he was asked to bring his first-degree relatives to the next appointment, so that they could be screened for glaucoma.

Mr AA underwent refraction of the left eye and had a corrected visual acuity of 6/18. IOP was controlled with timolol and xalatan (which Mr AA was able to buy using some of the funds he had set aside for the operation). However, because he expressed concern about not being able to afford life-long medication, left eye trabeculectomy with MMC was subsequently performed.

We are grateful to our reviewers, Clare Gilbert, Richard Wormald, and Nick Astbury for their contributions.

‘The uptake of glaucoma surgery still seems very low in Africa’

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Final comments by the panellists

“An interesting case and very real in our setting. Mr AA highlights the problem that we all experience: non-compliance with topical medication and failure to return for regular follow-up.”

“The ophthalmologist made very reasonable decisions in the light of the prevailing circumstances.”

“Even challenging situations can lead to success, as seen in this case, at least in the short term.”

“Surgery is definitely the right approach in the management of this patient; otherwise the next time he returns his visual acuity may be further reduced.”

“The uptake of glaucoma surgery still seems very low in Africa. However, we should realise that, for many of our patients, surgery should be the first line of treatment. Nevertheless, there will still be patients who will adamantly refuse surgery, and for whom we would need to consider laser treatment, if available.”

“This case underscores the role of advocacy for universal health care to cover potentially blinding conditions such as glaucoma, as well as the need for greater public education and awareness. These are issues which the ophthalmologist cannot handle alone but which require engagement with government and other community development sectors.”
A successful trabeculectomy is a stable surgical fistula. The one thing required to keep a fistula patent (open) is flow. The principal and most challenging complication is scarring; however, other complications may occur, as outlined in this article.

Prevention is vastly preferable to cure, so take every precaution you can to avoid complications. This involves careful case selection and, before surgery, optimisation of the operating environment. In addition, patients should have been counselled prior to surgery so their expectations match their post-operative experience. Nevertheless, every surgeon – even with the most meticulous attention to correct surgical approach and technique – will at some stage encounter most of the complications mentioned here.

Active interventions to avoid complications are therefore common, occurring in about half of post-operative patients at some stage.

Please note, if you have to go back to theatre with someone within four weeks of surgery, I would recommend a low threshold to general anaesthesia if at all possible. The eye is already inflamed from the first operation (and the complications). This can make the field more tricky; local anaesthetic does not work so well since it is rapidly washed away even with the use of adrenaline. In addition, the patient is all the more anxious due to the need for repeat surgery (as are you).

General anaesthesia offers a much better environment for both patient and surgeon. In addition, the operation is much faster; a ‘quick extra suture’ can be less than quick under local anaesthesia.

**Scarring**

Scarring is the number one complication of trabeculectomy surgery; it takes up the majority of time in my own outpatient clinics. Management of this risk factor can improve your success rates in trabeculectomy surgery by 10% or more if you identify the patients most at risk and manage them intensively.

An appreciation of the natural history of fistula formation in trabeculectomy surgery is helpful in planning a management strategy. Immediately post-operatively, all that stands between a good result and a flat anterior chamber are your flap sutures; hence the importance of care at the time of surgery. In the weeks immediately following the procedure, scar tissue forms and offers resistance to outflow. The rate at which this tissue forms depends on ethnicity, use of antimetabolites at the time of surgery, and the external ocular environment (past medical therapy and previous trachoma, blepharitis, and conjunctival inflammation). There is often a natural peak of resistance to outflow which then resolves with remodelling. Increases in pressure are therefore not abnormal a few weeks after a trabeculectomy. The timing of this peak is very population dependent. In many African ethnic groups, it is within three to six weeks; in Caucasians it is usually about six to nine weeks post-operatively; people from Asian population groups often fall somewhere between these two. This pressure ‘hump’ may last a few weeks and (provided it does not go too high and all else looks good) observation is sufficient, since the outcome is a good, draining bleb.

The best way to prevent or control scarring is frequent – a minimum of weekly – reviews. Early signs of scarring must be noticed and actively dealt with. Mechanically re-establishing flow is the first priority. This can be achieved by massage, removing releasable sutures, dividing fixed sutures (by laser or needling) and needling of the bleb as a very last resort. Removing releasable sutures is the simplest and quickest approach by far, so give serious consideration to routine use of these sutures in your surgery.

My own personal observation is that clinicians are not usually aggressive enough in dealing with early scarring. For example, if patients have had combined trabeculectomy and cataract surgery, then the intraocular pressure (IOP) will drop naturally; however, flow needs to be established for the trabeculectomy to
function. Massage techniques are outlined in the panel below, so do not give up easily!

Once you have some healing in place to offer resistance (generally a few weeks post-operatively) it is perfectly sensible to enlist the help of your patient in massaging their own eye if necessary. They can maintain flow by regular massage throughout the day whenever they put eye drops in, or even more often if you teach them carefully.

In all of this, please recognise that there is a ‘window of opportunity’ and be sensitive to the passing of this window, so you do not persist longer than appropriate. I cannot give exact timings, since some patients remain sensitive to massage even one year post-surgery, whilst others have such an intense scarring response that massage may be useless after four weeks! One final point to recognise is that poor surgical technique can result in blebs that require permanent massage. If the arms of the trabeculectomy flap do not reach to the site of the sclerostomy, then this can have the effect of creating a valve only opened with pressure posteriorly. In this instance, re-exploration and revision is the only option to achieve a satisfactory long-term result.

Steroids are the next major post-operative tool to prevent scarring. Intensive topical eye drops should be preservative free formulations if at all possible. Sub-conjunctival administration at the bleb site may, in my view, be extremely helpful. I tend to prefer administration of sub-conjunctival steroids at each post-operative visit. Finally, depot-steroids, when available, should always be considered in patients who are unable or unreliable in taking their drops regularly. I personally administer these to the orbital floor rather than at the bleb site. Insufficient topical steroid therapy is, in my view, one of the principal causes of bleb scarring in patients who are subsequently referred to me for management.

There is evidence that intensive use of sub-conjunctival 5FU is helpful; however, this has potential side effects. The panel on page 75 details a technique for 5FU administration. Others have reported use of other sub-conjunctival medication such as mitomycin C and anti-VEGF agents. I do not have personal experience of these.

**Conjunctival leak**

Clearly, if there is a button-hole at a vital site, the conjunctiva is retracting, or the sutures are too loose. This is surgical error and needs repair. I personally like suturing the conjunctiva very precisely and carefully with at least four interrupted sutures to the limbus. There may still be a leak post-operatively during the first week or so. We have demonstrated such minor leaks to be of no consequence to final outcome. Larger leaks or persistent leaks are obviously more serious. There are two scenarios to consider, based on the IOP:

1. If the leak is combined with hypotony (low IOP) and/or a shallow anterior chamber, then this needs careful observation and surgeons should not hesitate to re-operate to correct it.
2. If the IOP is not too low and the anterior chamber is deep, then a more conservative approach can be considered for a period to see if matters resolve naturally. In this circumstance, if there is preferential anterior drainage through the leak, then sometimes releasing posterior sutures to encourage posterior drainage can solve the problem.

A long-term leak is not desirable since it is a track for infection and may be associated with instability of the anterior chamber. Repairing such a leak should be done with care and time since the track may be epithelialised. The conjunctiva should be taken down and then secured with numerous interrupted sutures. I personally make a one-third thickness groove at the limbus and use mattress sutures to secure the conjunctiva into the groove. If such repeat surgery is required,

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**Massaging techniques**

A trabeculectomy is a guarded fistula. Pushing on the guard will not achieve drainage! You need to ‘fishmouth’ the posterior opening by applying pressure to the sclera, just behind the posterior end of the scleral flap. This means the patient needs to be looking down as far as possible so you are able to apply the pressure in the correct place. Be careful not to stress the conjunctival sutures; use a slight downwards motion towards the cornea.

Self-massage by the patient should be taught using either one finger or two fingers: one from each hand. Different patients prefer different techniques. Get the patient to practice in front of you. Measure the IOP before and after the massage so you can tell patients what they have achieved.
then there may well be a more vigorous scarring response, which should be addressed as above. Whilst the leak is present, the use of prophylactic antibiotics should be considered.

**Hypotony**

Early post-operative hypotony may be seen in uveitic eyes, high myopes, and others with thin sclera where leakage through suture tracks is unavoidable. In such instances, it is usually a matter of ‘tiding over’ until the leaks seal and/or the ciliary body perks up with the post-operative steroids. ‘Tiding over’ may involve regular observation, rest (no coughing, bending, lifting or straining), and wearing a shield at night.

Should there be choroidal detachment, anterior chamber shallowing, or a threat of hypotony maculopathy, then viscoelastic or gas (isovolumetric SF6 or C3F8) may be used in the anterior chamber. This can be given at the slit lamp after anaesthesia and application of povidone iodine. If the hypotony is due to frank over-drainage, and especially if the anterior chamber is shallow/flat, then this is a surgical error and generally requires repeat surgical repair. It is better to face facts and do this early (on the first or second day after the operation) rather than late. Please remember that leaving an eye hypotonous increases the risk of suprachoroidal haemorrhage.

**Choroidal detachments**

Choroidal detachments are almost always associated with hypotony, and should be evaluated from the point of prevention of hypotony. Intervention should be undertaken to prevent or resolve kissing detachments since these can be extremely destructive. Consideration of drainage or other secondary procedures are only appropriate in the extremely rare instance of exudative detachments unresponsive to medical therapy.

**Infection**

Infection is, fortunately, rare. Again prevention is the key. Leaks should be observed closely, proud suture ends trimmed or removed, and any surgical intervention covered with povidone iodine (or similar) with meticulous ‘no touch’ technique. Should infection occur, admit the patient and administer intra-vitreal and intensive topical antibiotic therapy according to local protocols. Treat any local environmental predisposing factors.

**Hyphaema**

This is most usual on day one, and settles quite rapidly. If it does not, then there is likely to be a bleeding diathesis, either pharmacological or pathological. Treat the bleeding diathesis first and await resolution in the eye if at all possible. In the extremely unlikely event of an eight-ball hyphaema, evacuation of the clot may be necessary.

If available, administer tissue plasminogen activator into the anterior chamber half an hour prior to clot evacuation. This will minimise the risk of anterior segment trauma due to clot adhesion to intraocular structures.

Tranexamic acid (unless systemically contraindicated) can be given by mouth post-operatively to try to decrease re-bleeding.

**Other complications**

Bleb dysaesthesia or a diffuse ‘fish eye’ bleb can persist and require bleb revision to resolve it.

Malignant glaucoma should be anticipated as a risk in short eyes (axial length <20mm, or shallow anterior chambers) and prophylactic atropine 1% prescribed pre-operatively and once daily for a minimum of three weeks post-operatively.

Malignant glaucoma is most likely in the scenario of over-drainage resulting in forward rotation of the ciliary body.

Eyes in which administration of atropine has been unsuccessful require urgent surgical intervention, which is beyond the scope of this article.

I hope the above is of some help in management strategies to improve our surgical results from this fundamental operation to prevent blindness from glaucoma. There are many other potential complications of trabeculectomy during the first three months post-operatively that space does not permit me to explore.

Please remember: prevention is better than cure, and: ‘first do no harm’!

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**Reference**


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**Sub-conjunctival 5FU administration**

Adequate anaesthetic is essential. Get the patient to look down and administer the anaesthetic drops to the upper bulbar conjunctiva (the Bells’ phenomenon ensures the rest of the eye receives anaesthetic). Allow the anaesthetic time to act. Other tips include using a cotton bud soaked in anaesthetic and lodging it under the upper lid for a period or using sub-conjunctival lignocaine and waiting an adequate time for it to disperse and work.

Enter the conjunctiva to the side and behind the scleral flap. Never inject into a bleb cyst; the forces dictate that the injection will enter the anterior chamber using the line of least resistance. (Should the 5FU enter the anterior chamber, go straight to theatre and wash the chamber out). Inject slowly; the stretch receptors produce the most discomfort, so you want to try to avoid this; and, in addition, it gives the 5FU a chance to dissipate.

Once you have completed the injection do not withdraw the needle immediately but rather hold for another minute or so if possible. This gives the rest of the fluid a chance to dissipate, and prevents it leaking directly back out of your needle track onto the surface of the eye.

5FU toxicity largely comes from 5FU leaked onto the surface of the eye so it is vital to prevent this. After withdrawal of the needle, wash the eye with saline to remove any leaked 5FU.

You can check there is no 5FU by administering a drop of tetracaine (amethocaine). The pH of these anaesthetic drops is acidic (around 5) and the pH of 5FU alkaline (around 9). When the two fluids meet, they result in a white precipitate that is visible and shows the 5FU has leaked onto the ocular surface (Figure 3).

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**Figure 3. 5-Flourouracil and amethocaine precipitate in the lachrymal lake, indicating that 5FU has leaked onto the ocular surface**

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The patient needed the trabeculectomy and you have done a great surgical job, but the pressure is now above the pre-operative level and you are feeling a failure. What are you going to do? What follows is a personal suggestion of questions to ask in developing a strategy.

**How long is it since the operation?**
If within three months of the operation, then routine post-operative management should be employed. During this period elevated intraocular pressure (IOP) develops in some patients. This occurs as early as four weeks in some African ethnic groups but is more typically at about six to eight weeks post-operatively. It is part of the wound remodelling process and resolves with good subsequent operative results. The difficulty lies in telling it apart from frank scarring and failure which has to be addressed with every tool available (page 73).

After three months, it is safe to say you have a fully-developed failure on your hands!

**Is there residual function in the trabeculectomy?**
It is always crucial to remember the purpose of the trabeculectomy, namely to prevent ongoing glaucomatous optic nerve damage. Occasionally the recorded IOP post-trabeculectomy differs little from the pre-operative pressure and yet progression of glaucoma is halted. Nothing need be done in this case! All-day pressure readings post-trabeculectomy frequently show remarkably stable pressures, which is generally not the case when glaucoma is progressing. This may partly explain the apparent paradox.

Another option is to simply restart ocular hypotensive therapy. This is frequently sufficient, as evidenced by the ‘partial success’ figures in all trabeculectomy surgical trial outcomes. To optimise the topical therapy, remember to carefully explore the past records for the drug group that was best tolerated and most effective prior to the procedure.

‘As with any procedure, the results in your hands are what matter, not the minutiae of the technique compared to someone else’

**Needling for flap edge scarring:**
- entry away from sclerostomy
- motion to open flap posteriorly

**Posterior pressure after needling for flow – use flat end of needle to avoid perforation**

**Is the environment working against success?**
The environment may be local concurrent ocular disease such as uveitis, inflamed conjunctiva from allergy or blepharitis, lid pathology, or past ocular surgery. All of these should be treated first or a suitable strategy developed to minimise any subsequent procedure being at risk of failure from this secondary pathology.

The adverse environment may equally include external factors. Poor social situations for post-operative care include living alone and being unable to instil eye drops, or personal well-being having lower priority than child care and other responsibilities. Facilitate an improved environment wherever possible. Ask relatives to administer therapy. Help the patient to find a more satisfactory post-operative management environment. Consider depot-steroid use rather than intensive eye drop regimens. Consider review times that match the other time constraints for the patient.

**Is the trabeculectomy amenable to ‘resuscitation’?**
This involves careful examination with gonioscopy to ensure the sclerostomy is patent and free from internal obstruction. The conjunctival mobility, inflammation, and vascularisation should be noted. When examining the bleb, the most frequent site of failure is either scarring at the edge of the scleral flap with flat overlying tissues, or else encapsulation of the trabeculectomy flap with a raised profile.

Needling is not an exact science, but reports of outcomes suggest that greater success is achieved with a lower IOP immediately after needling, performing a course of needlings (i.e. more than one if required), and use of sub-conjunctival anti-scarring mediations.

**If needling is appropriate, how am I going to do it?**
As with any procedure, the results in your hands are what matter, not the minutiae of the technique compared to someone else. You want minimum complications and maximum success!

A good operative field is vital. Use of adequate anaesthesia (I use topical anaesthetic followed by sub-conjunctival lignocaine 2%), a speculum, vascular constriction (I use phenylephrine because it is handy in the clinic), and povidone iodine (or a similar preparation) make a world of difference.

Needling at the slit lamp has the advantage of immediate assessment of pressure effect and easy review at regular intervals after the procedure (more difficult when done in the middle of a busy operating list). Needling in the operating theatre allows you to proceed immediately to a more complex surgical intervention if necessary. Needles of size 25–30 g have been reported, as have MVR blades; some practitioners report being more adventurous in their choices. (I use a 30 g needle at the slit lamp and micro MVR in theatre.)

Most practitioners enter the conjunctiva at a distance from the area of scarring to be perforated and use a ‘slicing’ action to open a good-sized drainage channel (Figure 1). Plan your approach carefully and only work on the area of obstruction to flow. The most common complication when
needing is haemorrhage – either sub-conjunctivally or into the anterior chamber. If your view is obscured, then you should stop and try another time.

A hyphaema requires the patient to be reassured, as their vision will be affected: you should wait until you are sure the active bleeding has stopped. Let the patient rest for 30–60 minutes then check for ongoing haemorrhage and a pressure rise, since the blood can sometime obstruct drainage completely. Once stable, manage the patient as for a hyphaema post-trabeculectomy: discharge the patient and review within one week as appropriate.

The other post-needling complication is hypotony. I personally have only had to take one patient back to theatre for this to date. If the anterior chamber has significantly shallowed, let the patient rest and see if it reforms spontaneously. If it does, then manage the patient as for a low pressure following trabeculectomy. If it does not, then introducing viscoelastic or gas to the anterior chamber is your best option, with regular review as appropriate. Case reports exist of infection and mis-placed needles, but these are fortunately rare (hopefully because appropriate care has been taken by clinicians).

Prophylactic topical antibiotics are used by most practitioners. Pre- and post-operative steroids remain a mainstay of therapy to prevent recurrent scarring.

Sub-conjunctival steroid and 5-fluorouracil (5FU) are the most common antisearing preparations. Be extremely careful that the drugs do not enter the anterior chamber. If they do, wash out in theatre immediately. See page 75 for tips on administration of 5FU. Mitomycin C is being used more frequently, and bevacizumab are amongst the many additional agents that have been reported, with varying success.

If needing is not appropriate or has failed, what subsequent procedure is required?

This depends on all the above factors and what is possible in your unit. Cycloideciliary body ablation, repeat trabeculectomy at a second site, formal revision of the existing trabeculectomy, and drainage tube implantation are the most common options.

References
bottles. There is no evidence that fixed combinations have better outcomes than using individual drugs. However, using fixed combinations is more convenient, reduces the amount of preservatives that enter the eye, and may make it more likely that patients will continue with their treatment (known as adherence or compliance).

It is not advisable to use two or more combinations in an eye. As mentioned before, if a single combination does not work, NICE guidelines recommend offering surgery to the patient.

**Side effects**

Each drug has different side effects, so prescribers and patients are advised to read inserted leaflets carefully.

- Pregnant women should avoid prostaglandin analogues (which can cause uterine contractions) and carbonic anhydrase inhibitors (which have teratogenic effects).

**How to avoid fake glaucoma drugs: top tips**

- Buy drugs from registered pharmacies
- Look out for the national drug administration/agency licensing number in your country.
- Check the manufacturing and expiry dates of drugs and be sure that these have not been altered on the packet.
- Many companies now have holograms of their logos on the packet, look for that.
- Some drug companies provide a means for patients to check the authenticity of their medicines. For example, many drug companies in Nigeria put a unique code or number on each box or bottle. Patients can SMS this free of charge to the phone number provided and the drug company will confirm whether the drug is registered and therefore genuine. This facility should be used where available
- Do not buy drugs from hawkers. Apart from raising doubts about the drug’s authenticity, hawkers will not be able to store the drugs in the correct conditions. Poor storage, heat, and sunshine will decrease the potency.
- Never accept any drug package without a company label from the manufacturer (some people peel off the label and write the dosage on the bottle to hide the identity of the medication).
- If not sure of your medication, bring it to the hospital for it to be checked and confirmed.
- Be careful with expensive imported brands from big, well-known drug companies; they are more likely to be fake than locally produced drugs from smaller companies
- Ideally, eye care facilities should stock genuine drugs in good quantities and at reasonable prices. This will help ensure that patients have access to the medicines they need from a trusted source.

**Useful hints**

1. Determine a target IOP before starting treatment. IOP with initial single-drug therapy should be reduced by at least 20% from baseline. IOP reduction of less than 10% should be considered as a non-response.
2. The treatment goal should include stable optic nerve and nerve fibre layer status, as well as stable visual fields.
3. Switching drugs within the prostaglandin analogue class may, upon occasion, provide greater lowering of IOP.
4. Pilocarpine is useful in pigmented glaucoma (PG) and pseudoxefoliation glaucoma (PXG), as it reduces iris movements. It may therefore reduce deposition of exfoliation material or pigment in the trabecular meshwork.
5. Topical carbonic anhydrase inhibitors (CAIs) and systemic CAIs are poorly additive with respect to lowering IOP.
6. Numerous studies have demonstrated neuroprotection in experimental models of glaucoma or optic nerve injury, but good evidence demonstrating neuroprotection in clinical studies is lacking.
7. There is insufficient evidence for neuroprotection by alpha-2 adrenergic agonists in humans.

**Patient’s adherence to treatment may be encouraged and monitored by:**

- Educating and counselling the patient
- Training personnel to teach patients and their carers
- Explaining the possible side effects of each drug
- Teaching the patient to record the drugs used and instilled (page 79)
- Checking the patient’s drugs at each clinic visit
- Prescribing combination drug preparations, where available, rather than many single preparations
- Giving advice to patients on how to instil eyedrops, particularly if they have any physical impairments, including visual impairment.

**Further reading**


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Instilling your own eye drops

• If you have just been prescribed eye drops, make sure someone has shown you how to instil them. Do not leave the eye clinic until you know what to do. Make sure you have had time to practice under supervision.
• Keep this handout safe for future reference.
• Instilling your own eye drops is not easy at first, but your skill will develop as you practice. You will find a technique that works well for you but there are some points that are very important (shown in bold).
• If, after much practice, you are still struggling, ask a family member or carer to instil the eye drops for you.
• You may think it will help to use a mirror and some people may even advise this. In fact, the use of a mirror only complicates matters and can even create a dangerous technique. Avoid the use of a mirror!
• Eye drops are dispensed in various containers: a bottle (plastic or glass) with a removable dropper and combined cap; a plastic bottle with dropper attached and removable cap; or a glass bottle with plastic pipette attachment and removable cap. Whatever type is given to you, do not touch the part from which the drop falls.
• Before instilling eye drops, wash your hands thoroughly, and afterwards too.

How to instil your own eye drops

1 Sit or lie down with your head supported. As your skill develops you may eventually manage to instil your eye drops while standing.
2 Use your dominant hand to hold the bottle/dropper/pipette
3 With the index finger of your other hand, hold a clean piece of tissue or cotton wool, and gently pull down the lower eyelid to form a ‘pocket’.
4 Hold the bottle/dropper/pipette between your thumb and forefinger, and place the ‘heel’ of your hand (where the wrist meets the hand) on your cheek. This will help to steady shaky hands.
5 Make sure there is a distance of about an inch (2.5 cm) between your eye and the end of the bottle/dropper/pipette. Be careful – the tip must not touch any part of the eye or eyelids.
6 Look up or to the side. Do not look directly at the bottle/dropper/pipette.
7 Squeeze the bottle/dropper/pipette – allow one drop to fall into the lid pocket.
8 Slowly let go of the lower lid. Gently close your eyes; try not to shut them tightly as this will squeeze the drop out of your eye.
9 Dab your closed eye with the tissue or cotton wool to remove any excess.
10 Put gentle pressure on the inside corner of your eye and count to 60, very slowly. This prevents the medicine from draining out of your eye before it is absorbed.

A few more top tips

<table>
<thead>
<tr>
<th>General</th>
<th>Glaucoma patients</th>
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<tr>
<td>• Store eye drops in a cold place, if possible in a refrigerator. It easier to feel a cold drop going in; this will reassure you that your technique is good.</td>
<td>• Instil your drops at regular intervals throughout the day. This is vital in controlling the intraocular pressure.</td>
</tr>
<tr>
<td>• If you struggle to hold a small bottle/dropper/pipette, wrap something like a folded piece of tissue around it</td>
<td>• Create a form or ‘tick sheet’ you can fill in when you have taken your drops (see Table 1).</td>
</tr>
<tr>
<td>• Wait at least 5 minutes between inserting different types of eye drops.</td>
<td>• It is important to learn to instil your own drops. Medication for glaucoma is usually needed for the long term.</td>
</tr>
<tr>
<td>• Instil eye drops first, then eye ointment (if prescribed).</td>
<td>• Practice makes perfect!</td>
</tr>
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Table 1. Sample patient record of eye drops instilled, for someone who has to instil eye drops four times a day.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Eye (left eye, right eye, both eyes?)</th>
<th>Breakfast/sunrise</th>
<th>Lunch/midday</th>
<th>Evening/sunset</th>
<th>Bedtime</th>
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How communities can control trachoma without a big budget

Stephanie Ogden
WASH/NTD Coordinator, International Trachoma Initiative, Children Without Worms and Emory Center for Global Safe Water
Email: sogden@taskforce.org

Paul Emerson
Director, Trachoma Control Programme,
The Carter Center, Atlanta, USA
Email: Paul.Emerson@emory.edu

Trachoma is an eye infection that affects an estimated 325 million people in Africa, Asia, and the Americas, and is the world’s leading cause of preventable blindness. Infection occurs most readily in children, causing itching, redness, and irritation in the eyes and eyelids, and infected ocular discharge. Repeated infections in childhood lead to the formation of scar tissue which culminates in the inversion of the eyelids and eyelashes in adulthood, and ultimately, blindness.

Blindness from trachoma is preventable in every community in the world – right now. The World Health Organization (WHO) and its partners have set 2020 as the target to eliminate blinding trachoma globally, and communities can do this individually by breaking the cycle of infection and re-infection.

Trachoma control programmes have two major thrusts: providing surgery to patients at immediate risk of blindness from trachomatous trichiasis (see Commn Eye Health J 2012;25(78):38) and preventing transmission of the bacteria so that subsequent generations do not progress to the chronic, blinding stages of the disease.

The WHO endorses and promotes the SAFE strategy for trachoma control. While the S (surgery) of SAFE can repair inturmed eyelids and prevent damage to the cornea, the A, F and E components can stop transmission of the disease – through treatment of current infections with antibiotics, facial cleanliness, and environmental change that encompasses the provision of water and sanitation. Many country programmes benefit from the generous donation of Zithromax® from Pfizer Inc. to address the A component, and donors provide funds for distribution. However, the F and E aspects of SAFE typically don’t benefit from the same level of support, despite increasing evidence that halting trachoma transmission may not be possible without these vital activities that are often considered to be either too difficult or too expensive to implement.

The key to trachoma prevention is not complex infrastructure, but rather individual and household behaviour that prioritises and acts to ensure that faces are clean, that all household members dispose of their faeces in a safe way, and that households are free of material that attracts flies.

Facial cleanliness, hygiene promotion, and access to water and sanitation should be thought of as the cornerstones of trachoma prevention – to which antibiotics (the A component) can be added. The importance of the F component is two-fold: first, washing children’s faces ensures that infectious eye and nose discharge that can be spread to others is washed away. Secondly, removing mucus, traces of food, and other material from children’s faces decreases their attractiveness to eye-seeking flies that can carry the bacteria from one child’s face to another.

The E component, access to water and sanitation, changes the environment from one that favours transmission of trachoma to one which reduces it – and simultaneously contributes to the Millennium Development Goals, which call on each country to reduce by half the population of those without access to safe water and sanitation between 1990 and 2015. The E component also includes both household and wider environmental sanitation. In a practical sense, this means that all households should have access to a latrine (and that they use it), and that households and communities engage in proper waste management that limits the amount of human and animal faeces, food scraps, and excess moisture that attract flies.

Past implementation of the E component has largely focused on encouraging household sanitation and decreasing open defecation. The trachoma vector Musca sorbens is a fly that breeds in human faeces left on the ground out of the direct sun. However, it is not able to breed inside of latrines, where temperatures and moisture are typically too high and oxygen concentrations too low. Ensuring that households have access to and use a latrine for defecation helps to decrease the number of breeding grounds, and results in a reduced adult fly population within the community at large. Similarly, ensuring that communities engage in proper waste disposal, and that households and their adjacent compounds, as well as community areas such as schools, clinics, and streets are swept clean of animal waste and food scraps helps to decrease attractants to flies and reduce overall fly population.

Ensuring facial cleanliness and environmental sanitation is not just about expensive water projects. Facial and hand hygiene (with commercial or locally made soap if available) can be accomplished with small amounts of water used sparingly. Mothers of families have shown us that 1 litre of water can be sufficient to ensure that several children’s faces are kept clean throughout the day. Programmes can celebrate existing positive hygiene practices in the communities, promote them as being achievable, and train community health agents to replicate them.

There are many steps to prevent trachoma that require little cost to households or to the wider community. Preventing trachoma starts with simple tasks.
Tips for community health promoters

Here’s what you can do to help prevent trachoma:

- Promote face washing among families – especially children. Even small amounts of water can be used to clean children’s faces throughout the day so that flies are not attracted to them.
- Teach families that sharing towels or clothes can put their loved ones at risk of infection if someone has trachoma. The bacterium that causes trachoma is easily spread through towels, bed sheets, clothes and wash cloths. These should be washed with soap to kill trachoma-causing bacteria.
- Ensure that trachoma prevention and hygiene education are taught in primary schools.
- Encourage every household in your community to maintain access to a latrine:
  - educate community members of the danger of open defecation. Even one open defecation site in the community may put all community members at risk for trachoma
  - encourage community-wide cooperation and vigilance to encourage community wide sanitation
  - encourage households without a latrine to build, use, and maintain one
  - encourage households with a latrine to allow neighbours to use it until they have built their own
  - conduct a community household sanitation survey. Share this data with local health clinics, government offices or non-governmental organisations (NGOs) to help them understand the magnitude of the community’s sanitation challenge.
- Encourage families to dispose of children’s faeces safely. Faeces must be thrown into the latrine or buried to prevent flies from breeding in it. Children’s faeces left in the open carry the same risk of spreading trachoma as open defecation.
- Congratulate families who sweep their compounds every day to keep them clean, and celebrate them as community role models. Clean compounds look good and decrease the number of flies that can carry trachoma from one face to another.
- Organise women’s groups, village trachoma control committees, or school health and hygiene clubs to actively promote trachoma prevention.
- Make connections with local NGOs that work in water, sanitation and hygiene. Help them to target WASH improvements in communities where trachoma is highly prevalent.

Case study: Village in Mali makes soap to fight trachoma

Safia is a trachoma community health volunteer in Mali. She lives in the village of Sokoura which is 180 km from Mopti, the regional capital. Safia received training in how trachoma is transmitted, how it can be prevented, and the devastating effects that it can have in later life in a national training programme. She is a vibrant community member involved in soap production through a microfinance initiative supported by the programme and also provides regular, individual health education to women in her community. We spoke with her at home just after she had finished preparing a batch of local soap for sale in the market and asked about her involvement with the trachoma control programme.

“Once a year the national trachoma control programme organises a campaign to distribute medicine for trachoma. I work with my neighbours in the community to prevent trachoma transmission twelve months of the year. During my training, I was moved by the thought that I could help my community protect themselves from blindness, and I wanted to do what I can for my friends. I was already a member of the womens’ group, so it is easy for me to discuss new ideas and think about new ways of doing things. The womens’ groups have organised themselves to conduct village cleaning from time to time. We feel proud that the public

Women in a Mali village got a loan to produce and sell soap locally

‘I work with my neighbors in the community to prevent trachoma transmission’

Places are clean like our own compounds. “One of the best ideas that was given to us was microfinance for soap production. We make a loan of 5,000 CFA (approximately US $11) for the purchase of materials and the recipient pays back a total of 5,250 CFA from her profits. The soap production is very popular because it allows women to have soap for their own family for free and gives a little financial independence, so that we can buy treats for our children. The increased availability of soap should help everyone improve their hygiene and make laundry day easier.”

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IAPB 9th General Assembly

The 9th General Assembly (9GA) of the International Agency for the Prevention of Blindness (IAPB) was a resounding success, with over 1,400 people attending the five-day meeting in Hyderabad, India.

The assembly was superbly organised and was characterised by a spirit of learning and collaboration, not only during presentations and panel discussions, but also during chance meetings in corridors and around the tea and coffee tables.

We asked a few leaders in international non-governmental organisations what key messages they took home with them.

Bob McMullan, incoming President of IAPB: “We need to be very disciplined and prioritise because funds are limited. Working in a noble cause increases the obligation to be efficient and effective.”

Serge Resnikoff, Organisation for the Prevention of Blindness: “WHO is leading the movement towards universal health coverage; it will be up to all of us to ensure that, in our country, eye care is on the agenda when this is being discussed.”

Lesley Podesta, Fred Hollows Foundation. “There are so many unheralded women making a real difference in some of the most difficult environments and communities. We need to actively make efforts to bring more women into leadership roles over the next few years.”

Allen Foster, CBM. “Avoidable blindness is highest in neglected communities. In order to achieve the goal of VISION 2020 we need to focus our time, effort, and limited resources on improving eye care for these neglected communities.”

Brien Holden, Brien Holden Vision Institute: “We have to get down to the business of blindness prevention. We must become much more effective at bringing all the stakeholders together to focus on eliminating the problem. The corporate social responsibility panel at the 9GA showed that this is possible.”

Richard le Mesurier, IAPB Western Pacific: “There needs to be a more pro-active focus on primary eye care. There are also various areas of neglect: Francophone Africa is lagging behind, and more work is needed to address refractive error and get spectacles to the poor.”

For more information about the general assembly, including presentations, videos and photographs, please visit: www.iapb.org/9th-general-assembly

GLOSSARY

Glaucoma glossary

**Anterior chamber.** The part of the eye between the cornea and iris, filled with aqueous humour.

**Aqueous humour.** A clear fluid continually produced by the ciliary processes. It contributes to the maintenance of intraocular pressure. The fluid leaves the eye through the sieve-like trabecular meshwork and Schlemm’s canal to reach deep veins in the sclera.

**Bleb.** A ‘blister’ of tissue overlying the site of glaucoma drainage surgery, from where aqueous escapes from the eye.

**Central vision.** The detailed vision in the centre of a person’s gaze for which the macular area of the retina is used.

**Glaucoma.** A group of complex eye diseases characterised by optic nerve damage resulting in loss of vision with typical visual field defects, and, usually, with raised intraocular pressure.

**Intra-ocular pressure (IOP).** The pressure inside the eye that results from the combined production and drainage of aqueous humour, measured in millimetres of mercury (mmHg). Normal IOP ranges between 12 and 22 mmHg.

**Laser trabecuoplasty.** A surgical procedure to deliver a series of laser burns to the trabecular meshwork to improve the outflow of aqueous humour in open-angle glaucoma.

**Optic nerve.** The nerve tract that transmits visual information from the retina to the brain.

**Perimetry.** A test that produces a map of the field of vision to plot visual field defects.

**Peripheral vision.** The top, sides, and bottom areas of vision. These may be the first areas of vision affected by glaucoma.

**Shunt.** An artificial drainage device surgically implanted in the eye to lower intraocular pressure.

**Trabecular meshwork.** A meshwork of connective tissue located at the angle of the anterior chamber of the eye and through which the aqueous humour drains.

**Trabeculectomy.** A well-established surgical treatment for glaucoma, in which a small, covered, drainage hole is created in the sclera to allow a controlled outflow of aqueous. An augmented trabeculectomy involves the local application to the trabeculectomy site of an agent that inhibits scarring.

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Test your knowledge and understanding

This page is designed to test your understanding of the concepts covered in this issue and to give you an opportunity to reflect on what you have learnt.

Diagnose This: quiz 1

The photograph on the left shows the left optic nerve of one of your patients. Which of the four visual field tests (above, right) would best match this photograph: A, B, C, or D?

Diagnose This: quiz 2

Which of the gonioscopic photographs above would represent a normal anatomic finding: A, B, C, or D?

Diagnose This: quiz 3

A patient with primary open-angle glaucoma underwent trabeculectomy. On the first post-operative day, the visual acuity corrected to 20/80, the bleb was almost flat, the anterior chamber shallow, and the intraocular pressure was 1 mmHg. What is the most likely problem?

- Aqueous misdirection (malignant or ciliary-block glaucoma)
- Ciliary body shutdown
- Early failure of bleb with scarring at episcleral surface
- Bleb leak

Quiz 2

The most likely problem is bleb leak.

The left optic nerve depicted in the photograph has an inferotemporal thinning of the optic nerve rim and notch formation from the 5 o’clock to 6 o’clock position. The superior visual field loss in Figure a would match this optic nerve damage. The visual field in Figure B would require an optic nerve with advanced damage of the superior and inferior neural-retinal rim. The visual field in Figure C is normal. The visual field in Figure D would be found in a patient with a defect in the superior portion of the optic nerve.

Quiz 3

The support vector machine in Figure A is normal. The visual field in Figure B shows a narrow nasal step. The visual field in Figure C shows a central scotoma. The visual field in Figure D shows a peripheral defect.
Useful resources

Online resources
RAPD test video
http://tinyurl.com/RAPDvideo

Gonioscopy teaching videos
www.gonioscopy.org (requires good internet access)

Online guide to visual fields
http://tinyurl.com/visualfieldguide

Moorfields trabeculectomy guide
Khaw PT, Shah P, Zeven T.
Trabeculectomy: The Moorfields Safe Surgery System.
http://tinyurl.com/TRABguide

General
A patient’s guide to glaucoma. Free online book about glaucoma. Aimed at patients, but provides a useful overview.
http://tinyurl.com/GLAUCbook

Previous issues
Community Eye Health J 2006;19(59): What’s new in glaucoma treatment?
Community Eye Health J 2010;23(73) Equipment for eye care
Community Eye Health J 2010;23(74): Ten years to VISION 2020: Why information matters
Community Eye Health J 2011;24(76): Instruments and consumables

Notices

Have your say: uveitis
Uveitis is one of the themes we are currently planning. Have you had a useful or interesting experience you would like to share with others? Do you have any questions you would like to ask our experts? Write to: the Editor, International Centre for Eye Health, London School of Hygiene and Tropical Medicine, London WC1E 7HT, UK. Email: editor@cehjournal.org
Word limit: 350 words or fewer.
Photographs are welcome but please obtain written permission from patients.

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Courses
Community Eye Health Institute, University of Cape Town, South Africa
For information about postgraduate diploma (PGDip) in community eye health in 2013, or Masters in Public Health (Community Eye Health) in 2013, contact: Zanele Magwa, Community Eye Health Institute, University of Cape Town, Private Bag 3, Rondebosch 7700, South Africa. Tel: +27 21 404 7735. Email: ntombizanele.magwa@uct.ac.za

International Centre for Eye Health
MSC in Public Health for Eye Care from September 2013 to September 2014, or part-time over two years. Apply before April 2013. For scholarships and application details, write to: Registry, LSTHM, Keppel Street, London WC1E 7HT, UK. Tel: +44 20 7299 4646 or visit www.lshtm.ac.uk/prospectus/masters/mscphec.html

Kilimanjaro Centre for Community Ophthalmology (KCCO), Tanzania
For information on courses, contact Genes Mng’anya, KCCO, Good Samaritan Foundation, PO Box 2254 Moshi, Tanzania. Tel: +255 27 275 3547. Email: genes@kcco.net or visit www.kcco.net

Lions SightFirst Eye Hospital, Nairobi, Kenya
Small incision cataract surgery for ophthalmologists wishing to upgrade from ECCE. Duration: 1 month. Courses run every month. Cost: US $1,000 for tuition and US $500–700 for accommodation and meals. Write to: The Training Coordinator, Lions Medical Training Centre, Lions SightFirst Eye Hospital, PO Box 6676-00800, Nairobi, Kenya. Tel: +254 20 418 32 39. Email: training@lionsloresho.org

Lions Aravind Institute of Community Ophthalmology
To apply, write to: Prof V Srinivasan, LAICO, 72 Kuruvikaran Salai, Gandhi Nagar, Madurai 625 020, Tamil Nadu, India. Email: v.srinivasan@aravind.org

Lance Bellers

Next issue

The next issue of the Community Eye Health Journal will be on Disability