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Glaucoma and how to protect your sight: an expert's guide

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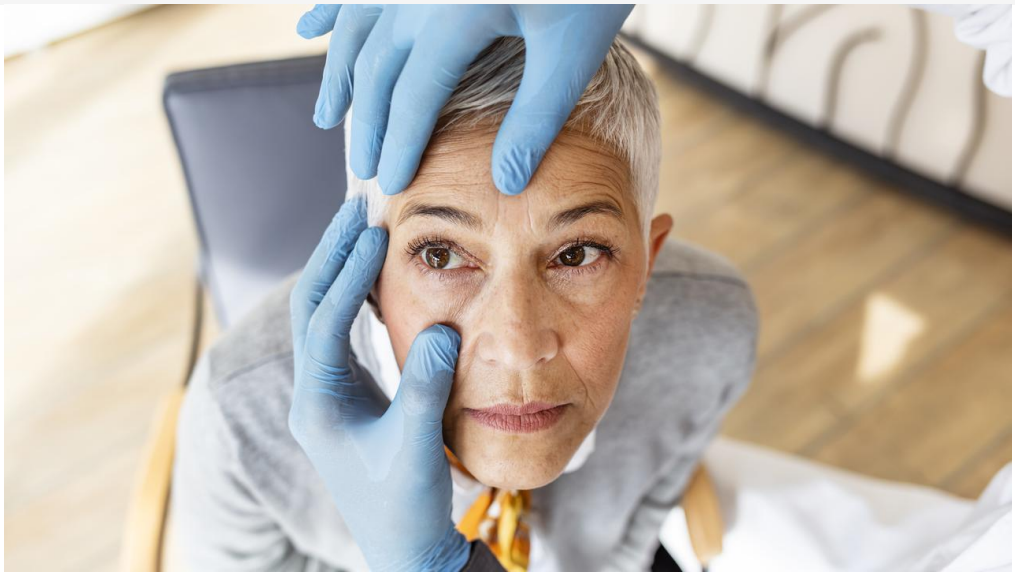

Too many Australians are unaware they have this debilitating eye disease. Picture: Supplied.

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"I heard Wayne had to give up driving because he is blind from glaucoma, but I saw him at the cafe reading a newspaper. I'm struggling to make sense of that: what is glaucoma?"

When most people think about their eyesight, they focus (literally) on what is directly in front of them. To see something off to one side, they turn their head and/or eyes.

If, instead of turning to look at the object, you were simply aware of it in your peripheral vision, then you are using your visual field. This side vision is what is damaged by glaucoma.

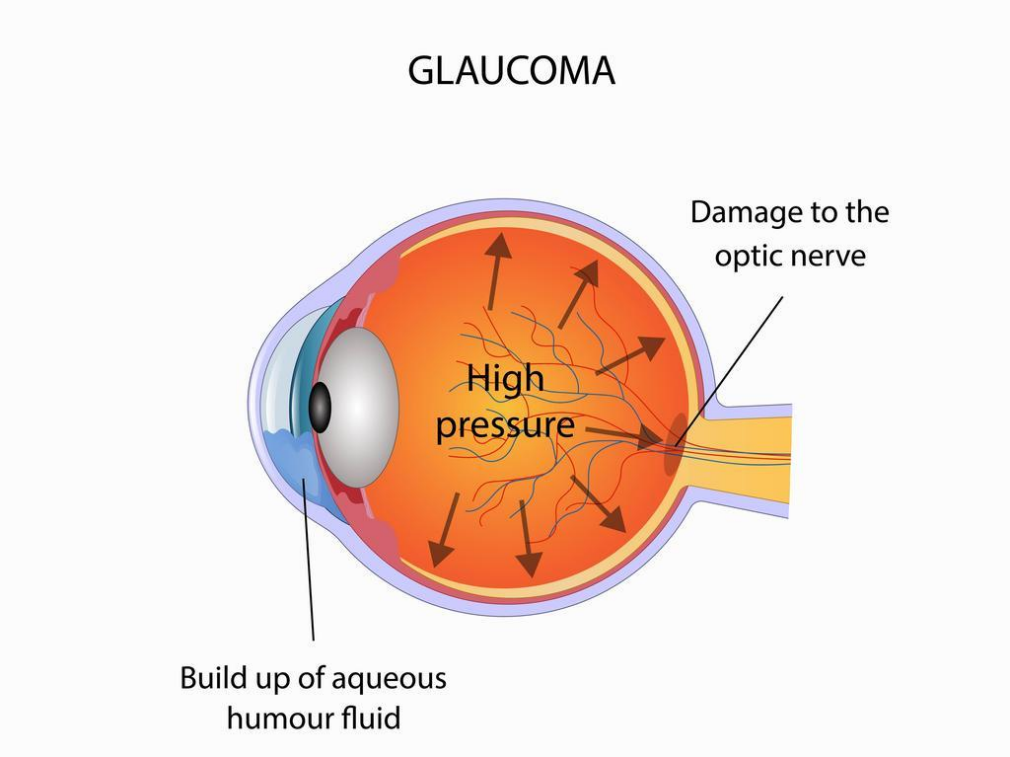
It is not sufficient for Wayne to be able to come to an intersection and to look each way and see nothing coming before crossing. He must also be able to see things he doesn't know he needs to look at (like a child chasing a ball on to the road), which will initially only register in his side vision.

Wayne's driving vision assessment would include both reading the letters on a vision chart and performing a visual field test – usually computerised – to assess his peripheral vision.

Wayne must pass both tests to be visually fit to drive: so he may be able to read the paper but still fail because his peripheral vision is constricted by glaucoma.

What is glaucoma?

Glaucoma is a condition where the pressure inside the eye (intra-ocular pressure, or IOP) damages the optic nerve at the back of the eye.



A cross-sectional representation of an eye with glaucoma. Picture: iStock

The optic nerve is like an electrical or fibre-optic cable, connecting the eye to the brain via a network of neurons (nerve fibres). Like a fibre-optic cable, if the tough outer coating of the optic nerve is stripped back a million or so tiny, fragile filaments would be revealed.

Each of these filaments accounts for a small area of vision: if that filament is damaged, then that spot in the vision of the eye is damaged or even lost.

If the pressure damage to the optic nerve is allowed to continue, these fine fibres are progressively weakened and the visual field can slowly be lost. This will characteristically take years to a decade or more.

Because the vision loss is generally so slow and affects peripheral vision – to which less attention is paid – glaucoma has been called the “sneak thief of sight”.

How widespread is it?

In Australia, an estimated 300,000 to 380,000 people are affected by glaucoma, its prevalence increasing with age. About half don’t know they have the condition.

Early defects in vision can be subtle and not noticed, but crucially cannot be repaired. Early detection and timely treatment are the best way to stop long-term blindness.

A formal eye assessment by age 40 is sensible, and being aware of a family history, especially a first-degree relative, may encourage an even earlier baseline assessment.

Other risk factors include increased IOP; African or Asian ancestry; long or short sightedness; a history of eye trauma or surgery; and prolonged use of medications such as steroids.

Optometrists and ophthalmologists are usually equipped to undertake appropriate assessments (and also oversee the formal driving reviews in most cases). Diagnosis of glaucoma is difficult for the GP, as they generally don't have the equipment.

Nevertheless, awareness of symptoms, risk factors, medication interactions, general health status and patient compliance with treatment all mean that your GP is a crucial member of the referral and management team.

Is glaucoma just one disease?

The ultimate reason for glaucomatous vision loss is damage to the optic nerve. The historical adherence to the idea that this occurred only above a pressure of 21mmHg has been abandoned.

The pressure that matters is the IOP at which nerve damage will occur in this particular eye. There is no "normal" IOP. The IOP in any eye is a balance between fluid (aqueous humour) inflow versus outflow.

There are numerous disease mechanisms by which the IOP rises to an unacceptable level.

Glaucoma is divided into various types on this basis.

Primary open-angle glaucoma (POAG) is the most common form of glaucoma in Australia. It is characterised by a structurally "open" anterior chamber angle, with progressive optic nerve damage leading to loss of peripheral vision in a characteristic pattern.

A subtype of POAG is recognised, which occurs at lower levels of IOP, and is associated with vascular risk factors such as migraine, Raynaud's phenomenon (white fingers and toes in cold conditions), low blood pressure and obstructive sleep apnoea.


Various types of glaucoma can be uncovered through testing. Picture: iStock

In contrast to POAG, primary angle-closure glaucoma (PACG) has physical narrowing or closure of the anterior chamber angle, resulting in limited outflow of the aqueous humour.

It may occur as a chronic, painless condition with gradual optic nerve damage, but is also recognised sometimes to present suddenly. Angle closure glaucoma is more common in Asian populations.

Acute angle-closure is a relatively sudden onset of severe eye pain, headache, blurred vision, haloes around lights, nausea and vomiting. Examination findings may include a red eye, a steamy cornea (the very front “window” of the eye), a mid-size pupil which does not change with light exposure, and markedly elevated IOP. Not all patients with acute angle closure have glaucoma (PACG), but all need urgent, same-day referral to an ophthalmologist.

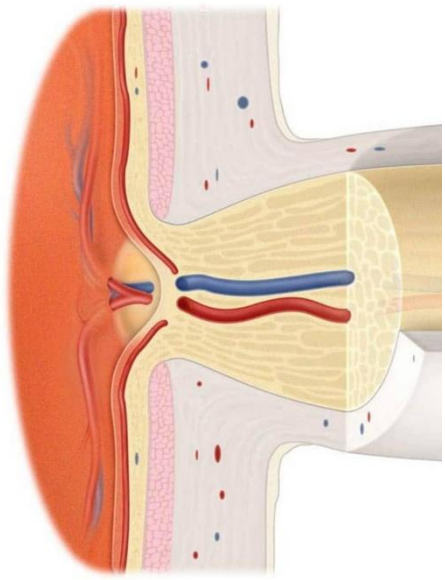
There are other, less common reasons for glaucoma to develop, as a consequence of other diseases (secondary glaucomas). Congenital glaucoma must be recognised early in a baby but is relatively rare.

It is important to note that – with the exception of acute angle closure – glaucoma most often produces no pain, nor any redness or inflammation. The lack of these symptoms – as well as early vision loss being asymptomatic – can be falsely reassuring.

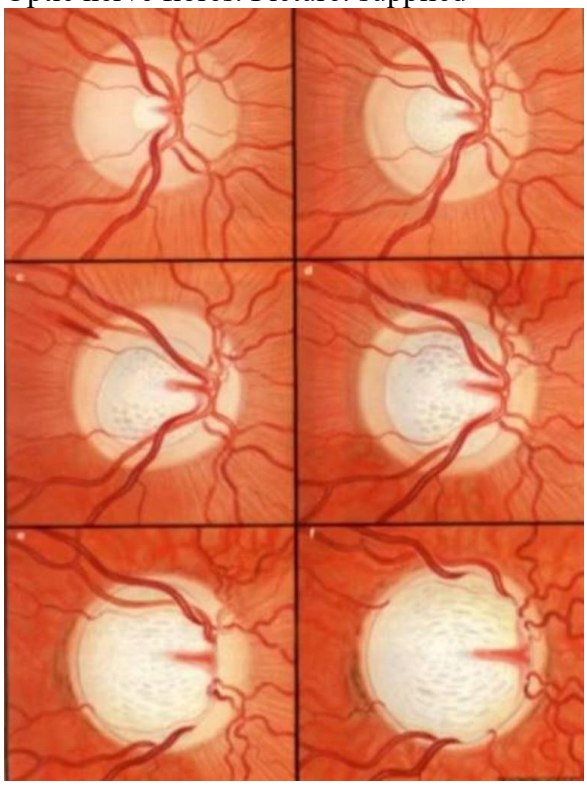
Some medications may produce an increase in IOP as a side effect, most prominently steroids (cortisone, prednisolone) and this can be via any route into the body (e.g. tablets, inhalers, skin cream, eye drops). Other medications – which will often carry product warnings for glaucoma – include some antidepressant or anti-anxiety medications and some antihistamines.

How is glaucoma diagnosed?

The optometrist or ophthalmologist will use a combination of structural and functional tests. These will include looking clinically at the optic nerve, the beginning of which (optic disc) can be seen by looking into the back of the eye; IOP measurement; assessment of the drainage; and then usually computerised imaging of the optic nerve and component nerve fibre layer as well as visual field testing.

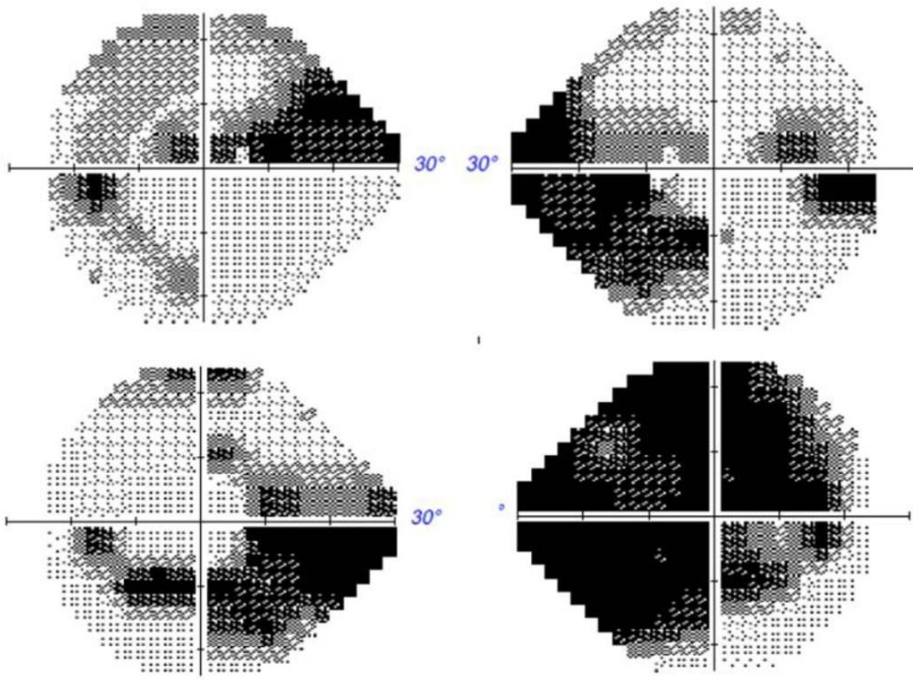


Optic nerve fibres. Picture: supplied



Optic nerve damage – described as ‘cupping’ – seen to be worsening through this sequence. Picture: Supplied

Visual field testing requires significant patient input as it tests the perception of fine light spots in various locations of the peripheral vision and the patient is required to respond by pushing a button when these are perceived.



Examples of a visual field test, where patients are asked to identify dots in their peripheral vision. Picture: Supplied

The patient generally does not have segments of "black vision" as displayed in computerised field testing: this is merely to demonstrate the area of loss to the clinician. In early field loss, there may even be a "filling-in" effect by the brain – much as performed by current AI photo-editing – which puts in place in the area of deficit what the brain expects to see.

How is glaucoma managed?

Almost all glaucoma treatment is directed to lowering the IOP to slow or stop further damage. However, the critical parameter is not the IOP but whether the lowering of IOP has stopped the optic nerve damage and stabilised the visual field: if the IOP is lowered, but the field is continuing to deteriorate, the IOP needs to be lower still (remembering that there is a "floor" below which IOP should not go, given that the eye does need a certain level of internal pressure to maintain its shape and function).

As IOP is the pre-eminent risk factor, sometimes treatment of the IOP will be recommended before glaucoma has developed (ocular hypertension).

Glaucoma is not able to be "cured", but can often be controlled. This means that ongoing monitoring is required, using a varying combination of the initial diagnostic tests, to judge whether there is any evidence of progressive damage.

This monitoring and treatment will often be undertaken in a collaboration between an optometrist and ophthalmologist, and after a time it may be possible – having seen whether

the individual's optic nerve has stabilised with treatment – to estimate the target or safe, pressure for the eye.

Treatments

Treatments fall broadly into three categories: medication (eyedrops), laser or surgery. Many optometrists are now able to prescribe glaucoma medications, but laser and surgical approaches remain the purview of ophthalmologists.

Historically, eyedrop use has been the mainstay of glaucoma therapy. There are numerous classes of medication available following an explosion of new therapies in the 1990s.

First-line medication choice would now most often be from the class of prostaglandins, which have the virtue of once-daily dosing and only rare systemic (general body) side effects; although they have a range of curious side effects local to the eye (one of which may include eyelash darkening and growth, which is not always unpopular).

Treatment will generally be long-term, often lifelong, because of the otherwise progressive nature of glaucoma.

More recent work has supported a greater use of laser in improving aqueous humour drainage and reducing IOP.

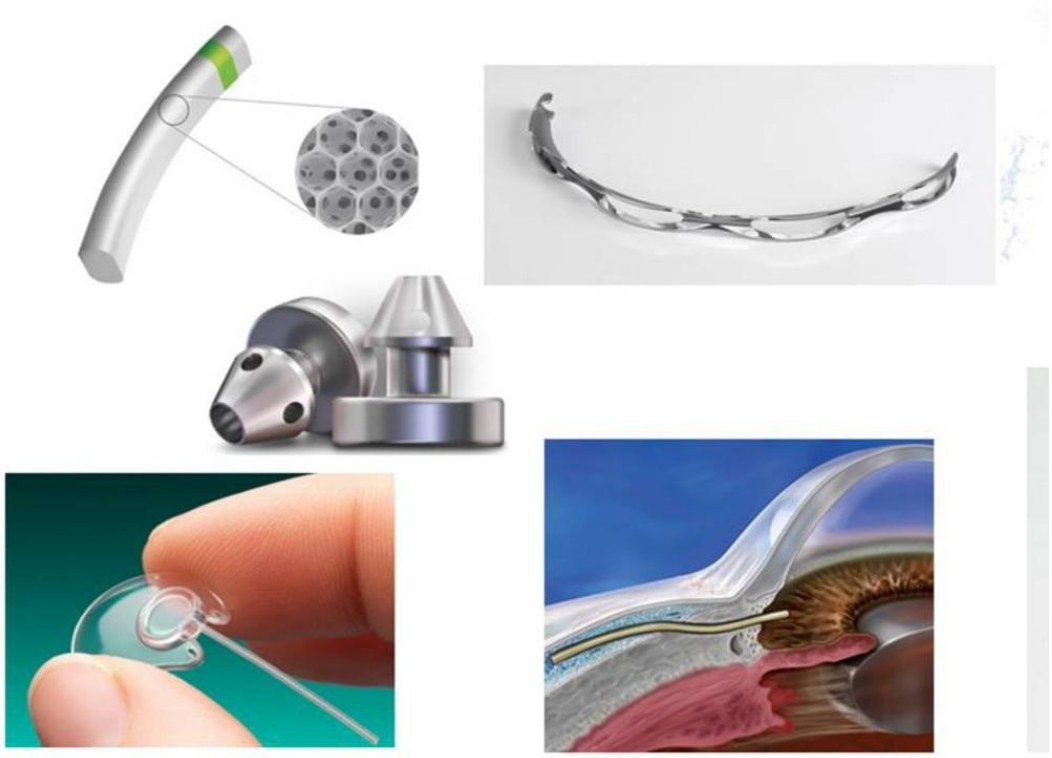
Increasing evidence supports laser as an effective alternative to eyedrop medications in selected patients with open-angle glaucoma or ocular hypertension, as well as an additional treatment when further IOP reduction is required.

Angle closure cases may need laser iridotomy, and cataract or lens extraction surgery may also have a place in treatment for these patients.

Surgical interventions may be considered when glaucoma progresses despite maximal tolerated other therapies, or when substantial IOP reduction is required. All the operations aim to improve fluid drainage from the eye in order to lower the IOP. None are able to restore vision: as with all present glaucoma treatments, they aim to limit further damage.

Trabeculectomy is the long-established operation which remains the gold-standard and uses the eye's own tissues to create a new outflow pathway.

Newer operations include stents and different artificial drainage implant devices, which have varying degrees of effectiveness and which are selected and tailored to the individual eye's disease severity, progression rate and desired target pressure. Some of these tend to be deployed in association with cataract surgery.



A variety of glaucoma surgery stents and drainage implants. Picture: Supplied

As a rule of thumb, the more involved or intrusive the surgery, the greater the likelihood of disease control and freedom from glaucoma eye drops after surgery; but also the greater the risk of complications (all of which should be explained prior to any operation). Almost all operations will require the use of antibiotic and anti-inflammatory eyedrops for a period after the surgery.

There is growing advocacy for earlier deployment of surgery – particularly stents – in glaucoma management, but this should be tempered by the ongoing need for research to show the ability of newer techniques to stabilise the visual field rather than just incrementally lower the IOP. There is also a need to understand better how long the effects will last. Cost-benefit analysis of some of the newer devices is still needed.

Patients will frequently ask about the role of exercise as a benefit or a risk in established glaucoma. Moderate aerobic exercise is generally safe and can be recommended; sustained straining ought to be approached with caution (for example, weightlifting), as are prolonged head-down postures (such as “downward dog” in yoga). Tight swimming goggles not resting on the bony rim of the eye socket should be avoided.

A popular hope is that marijuana – which has been documented to lower IOP – might be useful as a glaucoma treatment. Because the duration of IOP effect is short-lived, present formulations in a sufficient dosing schedule to achieve sustained IOP lowering would impair general functions too greatly to be recommended, but research is ongoing.

The future

Glaucoma is not an inevitably blinding disease, and with early detection and timely management, significant vision loss can be prevented in most patients.

Australian researchers are at the forefront of developments in glaucoma, in explaining disease risk, development and processes, but also in the development of surgical devices and treatments.

For example, a Melbourne-based study through the Centre for Eye Research Australia looking at potential benefits of Vitamin B3 in preserving optic nerve function has generated such interest that an international collaborative trial is now under way seeking to confirm the results.

And researchers at Flinders University in Adelaide have succeeded in developing a risk analysis model based around genetic screening and have now commercialised this as a test that individuals may request to try to establish their long-term risk of glaucoma development.

More information and support can be found at Glaucoma Australia (glaucoma.org.au) *and Vision Australia (visionaustralia.org).*

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