



Glaucoma in 2026: Beyond Eye Pressure Rethinking what we know about glaucoma

Written by Glaucoma Australia

For many years, glaucoma was explained in relatively simple terms: pressure inside the eye rises, the optic nerve becomes damaged, and vision is gradually lost. While this remains an important part of the story, we now know that glaucoma is much more complex.

Today, researchers understand glaucoma as a group of diseases that cause progressive damage to the optic nerve. This damage involves the loss of specialised nerve cells in the retina, called retinal ganglion cells (RGCs), and the nerve fibres that connect them to the brain.

Eye pressure, or intraocular pressure (IOP), remains the most important risk factor that we can currently modify with treatment. But IOP is not the same thing as glaucoma.

Some people develop glaucoma even when their eye pressure is within the statistically normal range, while others can have relatively high eye pressure without developing noticeable damage to the optic nerve.¹⁻³

This changing understanding is reflected in the latest international guidelines.

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From the CEO



Dear friends and supporters,

Welcome to this special edition of Glaucoma News. Research gives us something incredibly important: the

opportunity to change what is possible for people affected by glaucoma.

Through the Glaucoma Australia Quinlivan Research Fund, we have invested more than \$2 million in Australian glaucoma research, supporting leading researchers to explore innovative approaches to the complex challenges of glaucoma.

Encouraging progress is being made in areas including stem cell therapies, neuroprotection and approaches to repairing or restoring damage to the optic nerve.

These developments demonstrate the value of sustained investment. With persistence, collaboration and rigorous science, promising discoveries can progress from the laboratory towards clinical trials and new treatment options.

In this edition, we take a closer look at these advances and the questions researchers are working to answer. Every project we support is an investment in a better future for people living with glaucoma.

Thank you for being part of that journey. With your continued support, we can help turn promising discoveries into meaningful advances and move closer to a future where glaucoma no longer steals sight.

Gratefully yours,

Adam Check
Chief Executive Officer

Cover Story

Glaucoma in 2026: Beyond Eye Pressure

Continued from page 1

The American Academy of Ophthalmology's 2026 Primary Open-Angle Glaucoma Preferred Practice Pattern continues to place lowering IOP at the centre of glaucoma treatment, while emphasising individualised assessment and preventing further damage to the optic nerve and visual field.¹

The European Glaucoma Society's 6th Edition guidelines, published in 2025, similarly emphasise evidence-based, patient-centred care and incorporate advances in genetics, artificial intelligence, imaging and surgery.²

So, glaucoma research is increasingly asking a bigger question. Instead of simply asking, "How do we lower eye pressure?", researchers are also asking 'Why does one optic nerve remain healthy at a particular pressure while another becomes damaged - and can we identify and protect vulnerable eyes before irreversible vision is lost?'

IOP is still important - but it is not the whole story

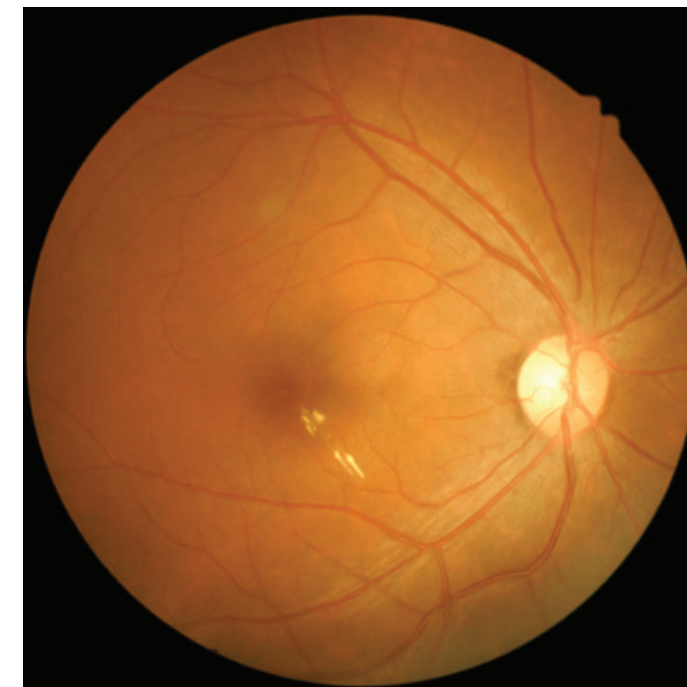
The strongest clinical evidence supports lowering eye pressure (IOP) to slow glaucoma progression. The goal is to reduce each patient's long-term risk of damage, rather than simply reach a target pressure.

Landmark trials show that IOP reduction can delay glaucoma development in people with ocular hypertension and slow progression in those with established glaucoma.^{1,4}

However, the relationship between eye pressure and glaucoma is not straightforward.

Some people develop progressive glaucoma at pressures that fall within the statistically normal range. Others have elevated eye pressure for many years without developing detectable glaucoma. Research suggests that retinal ganglion cells differ in their vulnerability, meaning that the same level of eye pressure can have very different effects in different eyes.^{3,5}

This suggests that IOP is a major driver of glaucoma, but it is not the complete explanation. Researchers are investigating many other factors, including the physical properties of the optic nerve and surrounding tissues, blood supply, mitochondrial function, oxidative stress, inflammation, the transport of nutrients along nerve fibres and differences in how resilient individual nerve cells are.^{3,5,7}



The optic nerve is therefore increasingly viewed as a living, dynamic environment - not simply a structure that is damaged when pressure becomes too high.

How the optic nerve responds to pressure

One important area of research focuses on a structure called the lamina cribrosa. The lamina cribrosa is a supportive, mesh-like layer of tissue at the back of the eye. The nerve fibres of the retinal ganglion cells pass through it as they leave the eye and form the optic nerve.

Eye pressure creates mechanical forces within this part of the eye. But how those forces affect the nerve fibres may depend on the structure and physical properties of the surrounding tissues. This could help explain why two people with similar eye pressures can have very different outcomes.

Researchers are investigating whether differences in the lamina cribrosa, the tissue surrounding the optic nerve and other parts of the optic nerve head influence how mechanical forces are transmitted to vulnerable nerve fibres.^{6,7}

This is still an active area of research rather than something routinely used to guide treatment. But it represents an important shift in thinking.

In the future, understanding glaucoma may involve knowing not only how much pressure is in the eye, but how an individual eye responds to that pressure.

Glaucoma and the health of nerve cells

Another important development is the growing recognition of glaucoma as a neurodegenerative disease.

This means that the disease involves changes within nerve cells themselves. Retinal ganglion cells do not simply disappear. Before they die, they can undergo changes involving their metabolism, mitochondria - the structures that provide energy to cells - nerve-fibre transport, inflammation and other processes.^{3,5}

A 2025 systematic review described retinal ganglion-cell degeneration as the central process underlying glaucomatous optic-nerve damage. It highlighted the way mechanical stress, reduced blood supply, metabolic problems and inflammation can interact to contribute to nerve-cell injury.⁵

The growing interest in neuroprotection.

Neuroprotection aims to keep nerve cells alive and functioning, rather than simply reducing the pressure that may be contributing to their injury.

Researchers are investigating potential treatments that target mitochondrial dysfunction, oxidative stress, inflammation, nerve-fibre transport and other mechanisms involved in cell survival.^{5,8-10}

Recent laboratory studies, for example, have investigated pathways involving CaMKII,

neuroinflammation and mitochondrial function. Other research has examined AIBP-related mechanisms and the role of the immune system in the retina.^{8,9}

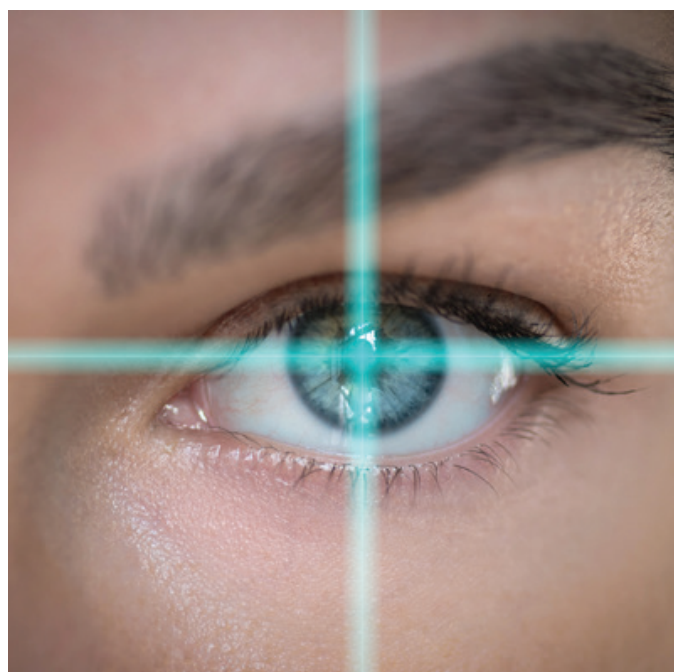
However, it is important to distinguish promising research findings from treatments that are already proven to work in patients.

As of 2026, neuroprotection has not replaced IOP reduction as the established treatment for slowing glaucoma. Many potential neuroprotective and neuroregenerative treatments remain in laboratory or early clinical development and still need well-designed clinical trials and long-term safety data.¹⁰

Could we detect glaucoma earlier?

Glaucoma is a complex biological disease assessed using a limited number of clinical measurements. Eye pressure is an important risk factor but does not directly indicate nerve-cell health.

Optical coherence tomography (OCT) has transformed care by precisely measuring changes in the retinal nerve fibre layer and ganglion cells. Visual-field testing assesses functional vision, while optic-nerve imaging provides additional information on structural change.



But researchers are now asking an even more challenging question: Can we identify the biological process of glaucoma before significant, irreversible damage occurs?

Scientists are investigating potential biomarkers – measurable biological or imaging changes that could help predict who is at risk and how their disease may progress.^{5,11} Reliable biomarkers could eventually help answer questions that today's tests cannot always answer with confidence:

- Which person with suspected glaucoma is most likely to develop the disease?
- Which patient is likely to progress quickly?
- Is a patient who appears stable actually experiencing early biological changes?
- Why is one optic nerve more vulnerable to a particular level of eye pressure?
- Which treatment is most likely to prevent progression for an individual patient?

Finding better answers could move glaucoma care towards predictive medicine, where treatment decisions are increasingly based on an individual's future risk rather than simply on whether a test result is currently abnormal.

Artificial intelligence: from diagnosis to prediction

Artificial intelligence (AI) is becoming an increasingly important part of glaucoma research.



AI systems can analyse retinal photographs, OCT scans and other clinical information very quickly. Researchers are now exploring whether AI can do more than identify glaucoma – including predicting which people may develop the disease, which patients may progress and who may eventually require surgery.¹¹⁻¹⁴

A 2025 systematic review of 44 studies found promising results for AI in predicting glaucoma development among people with suspected disease and in the general population, as well as

predicting progression in people who already have glaucoma.¹¹

Other studies have explored multimodal AI, combining OCT scans with electronic medical records to predict clinically important outcomes.¹²

However, research accuracy is only the first step. AI systems must be shown to perform reliably across different populations, imaging equipment and healthcare settings-and to improve patient outcomes.

The European Glaucoma Society recognises AI as part of the evolving glaucoma landscape.² For now, AI is more likely to support than replace ophthalmologists, particularly by detecting subtle changes, estimating progression risk and integrating long-term patient data.

The changing role of laser

The way glaucoma is treated is also evolving. For many years, eye drops were often the first treatment offered for open-angle glaucoma. Increasing evidence has strengthened the role of selective laser trabeculoplasty (SLT) as another first-line option for suitable patients.

The 6th European Glaucoma Society guidelines recognise SLT as a first-line treatment for appropriate people with open-angle glaucoma or ocular hypertension, alongside medication.²

The potential benefit of laser is not simply that it can lower eye pressure. For some patients, reducing the need for daily eye drops can make glaucoma treatment easier to manage over the long term.

There is no single best treatment for everyone. Disease severity, target IOP, eye characteristics, access, cost, preferences, adherence and previous treatment response should all guide decisions. The focus is on effective, sustainable and individualised treatment-not simply maximising pressure reduction.

Glaucoma treatment is usually lifelong.

Daily eye drops can be difficult to administer, especially for people with reduced vision or hand problems, while frequent dosing and eye-surface irritation can make treatment burdensome. Adherence is therefore not simply a behavioural issue but also a treatment-design challenge.

Laser treatment and longer-acting drug-delivery approaches may reduce the need for daily drops in selected patients, supporting the growing emphasis on patient-centred care and shared decision-making.² A treatment that works extremely well in theory but is difficult for a particular person to use consistently may not be the best treatment for that individual.

Why monitoring change over time matters

A single eye-pressure reading, OCT scan or visual-field test is only a snapshot. Glaucoma is a disease of change over time. The important question is not simply whether one measurement is abnormal.

It is whether the optic nerve is progressively losing tissue or function, how quickly that loss is occurring and whether the rate of change could threaten a person's vision over their lifetime. This makes long-term monitoring increasingly important.

Future glaucoma care is likely to make greater use of progression modelling, combining repeated OCT measurements, visual fields, eye-pressure history, treatments and individual risk factors.

AI could be particularly useful here because predicting progression requires many different pieces of information to be considered together over time.

Genetics and personalised glaucoma care

Glaucoma also has an important genetic component. Family history has long been recognised as a risk factor, and genetic research is increasingly showing that inherited differences can influence a person's susceptibility to glaucoma.

The challenge is turning these discoveries into useful information for individual patients. At present, most genetic findings are not precise enough to determine treatment on their own. A more likely future direction is to combine genetic information with other factors, including age, family history, eye pressure, optic-nerve structure, OCT results and changes over time.

This could eventually help distinguish between patients who have similar eye pressures but very different risks of developing vision loss.

An especially interesting area of research is the discovery that retinal ganglion cells are not all biologically identical. Different types of these nerve cells appear to have different levels of vulnerability and resilience following glaucoma-related or experimental injury.

Recent molecular and spatial studies have begun to identify groups of nerve cells that appear to be relatively resistant to glaucoma-related damage.¹⁵

This raises an exciting possibility: if we can understand why some nerve cells survive while neighbouring cells are lost, could we use those natural protective mechanisms to develop new treatments?

This is an area where glaucoma research increasingly overlaps with neuroscience and regenerative medicine.

Could we one day repair the optic nerve?

Neuroprotection aims to preserve nerve cells that are still alive. Neuroregeneration goes a step further: it aims to restore nerve cells or their connections after injury.

This is an enormous scientific challenge. For the optic nerve to recover, retinal ganglion cells would need to survive and potentially regrow their long nerve fibres over a considerable distance, while also reconnecting correctly with the brain.

Researchers are investigating gene therapy, cellular signalling, axon regeneration and other approaches to neural repair.^{10,16}

However, neuroregeneration remains a long-term research goal rather than an established treatment. This distinction is important. Once retinal ganglion cells and their nerve fibres have been permanently lost, current treatments cannot reliably bring them back. For now, the most effective strategy remains preventing further damage and preserving the vision a person still has.

Where is glaucoma research heading?

The future of glaucoma research is increasingly bringing together many different fields. Ophthalmology is intersecting with neuroscience, biomechanics, genetics, vascular biology, molecular biology, advanced imaging and artificial intelligence.

The long-term goal is a more complete picture of each person's glaucoma - one that considers much more than eye pressure.

This could make glaucoma treatment increasingly personalised. For example, a person with ocular hypertension and a healthy, stable

optic nerve may ultimately be managed very differently from someone with a relatively modest eye pressure but rapidly progressing retinal ganglion-cell loss.

That distinction is already part of modern glaucoma care. Future research aims to make it more precise.

The ultimate goal is to save sight. Despite the enormous progress made in glaucoma research, one fundamental challenge remains: we currently cannot reliably reverse established glaucomatous nerve-cell loss. That makes early detection, prevention and preservation incredibly important.

The next major breakthrough in glaucoma may not simply be another improvement in lowering eye pressure. It could be a treatment that directly protects retinal ganglion cells, makes them more resistant to injury, changes the biological environment around the optic nerve or, ultimately, helps damaged nerve cells regenerate.

This may sound like an ambitious goal but the research is moving in that direction from:

- pressure control towards protecting nerve cells;
- individual snapshots towards understanding change over time;
- population averages towards personalised risk;
- detecting established damage towards predicting vulnerability; and
- simply slowing disease towards the long-term possibility of repairing the visual system.

Perhaps the most important change in our understanding of glaucoma is that it is not simply a disease of high eye pressure.

The vulnerability of an individual’s optic nerve and nerve cells, along with other biological and environmental factors, also plays an important role in determining how the disease develops and progresses.

This evolving understanding highlights the need to look beyond IOP alone and consider the individual patient’s risk of disease and progression.

It also reinforces the importance of research into the mechanisms that make the optic nerve vulnerable, with the ultimate goal of developing treatments that can protect nerve cells and preserve vision. As our understanding of glaucoma continues to evolve, so too must our approach to its detection, monitoring and treatment.

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Research News

Researchers Find ‘Hidden’ Eye Cleanup System

Written by Mivision

New research from Canada has identified what appears to be a previously unknown waste drainage system at the back of the eye.

The discovery gives researchers new opportunities to harness this system to treat retinal diseases associated with buildup of fluid, waste, and inflammatory debris in the eye, including glaucoma and age-related macular degeneration.

Researchers from the University of British Columbia (UBC) and the University of Toronto said the hidden circulatory pathway, called the posterior ocular lymphatic outflow, or POLO pathway, provides a route for fluid and waste to exit the eye and enter the body’s lymphatic system.¹

... the team was “surprised to see such a direct route for fluid to leave the eye and connect with the lymphatic system. “The retina is one of the most metabolically active parts of the body, constantly generating byproducts that need to be cleared,” said Professor Neeru Gupta from UBC.²

“This discovery helps explain how the eye flushes this waste and promises to transform how we think about and treat a range of eye conditions.

“This gives us a whole new framework for understanding these diseases and a potential target for therapeutics,” Prof Gupta said. “The question now is: How can we enhance or exploit this cleanup system to treat or even prevent disease?”

To uncover the POLO pathway, Prof Gupta and Professor Yücel, from the University of Toronto, and their team introduced fluorescent tracer molecules into a thin space at the back of the eye in mice, then used advanced imaging to track their movement in real time.

This allowed them to identify small lymphatic vessels in the choroid, a thin layer beneath the retina. Fluid drained from the back of the eye into surrounding orbital tissue and, within minutes, reached nearby lymph nodes that link the eye to the broader lymphatic system.

Prof Yücel said the team was “surprised to see such a direct route for fluid to leave the eye and connect with the lymphatic system”.

“It suggests the back of the eye has an active clearance pathway, which could play an important role in removing fluid, proteins and inflammatory material that build up in disease.”

Further research is needed to understand how the lymphatic pathway operates in humans and how it could be targeted with therapeutics.

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Solving the complex puzzle of glaucoma

Written by Insight

Early intervention is just one of many jigsaw pieces to ponder for eyecare professionals approaching glaucoma, says Australian Society of Ophthalmologists vice-president Dr Justin Sherwin.

Glaucoma remains one of the most challenging causes of irreversible vision loss, not because effective treatments are lacking, but because the disease is typically silent until damage is well established.

As populations age and patients live longer with early disease, the opportunity and responsibility to intervene earlier has never been greater.

The emphasis in glaucoma care is therefore shifting from late intervention to earlier identification of risk and more thoughtful prevention. This does not imply eliminating glaucoma entirely, but rather delaying progression and preserving meaningful vision.

At its core, glaucoma is a chronic optic neuropathy characterised by retinal ganglion cell loss. Age, genetics, and intraocular pressure (IOP) remain the dominant determinants of risk, yet it has long been clear that IOP alone does not tell the whole story.

Many individuals with elevated IOP never develop glaucoma, while others progress despite pressures within the statistically normal range. Prevention therefore requires a broader view that integrates risk stratification, lifestyle considerations, and early structural detection.

Lowering IOP remains the only proven disease-modifying strategy. The Ocular Hypertension Treatment Study confirmed that early treatment reduces the risk of conversion to glaucoma in selected patients, particularly when factors

such as thinner central corneal thickness and optic nerve vulnerability are present. In practice, this supports a more individualised approach: not every patient with ocular hypertension requires treatment, but those at higher risk benefit from earlier intervention. Topical therapy remains effective, though adherence is often imperfect in an asymptomatic disease.

Increasingly, selective laser trabeculoplasty (SLT) is being used earlier, offering durable pressure reduction without the burden of daily drops and without relying on long-term adherence in patients who feel entirely well. For preventive care, this shift reflects both emerging evidence and pragmatism.

Beyond IOP, several lifestyle and systemic factors appear to influence risk and progression.

Regular aerobic exercise has been associated with modest reductions in IOP and improved vascular health. Smoking cessation and management of systemic cardiovascular risk factors are sensible, particularly given the role of vascular dysregulation in some forms of glaucoma, including normal-tension disease.

Diet has also attracted attention. Observational studies suggest that diets rich in leafy green vegetables, antioxidants, and omega-3 fatty acids are associated with lower glaucoma risk. While causality cannot be assumed, such dietary patterns align with broader health advice and carry little downside.

Vitamin B3 (nicotinamide) has emerged as an area of interest. Experimental work demonstrates improved mitochondrial resilience and retinal ganglion cell survival, and early human data are encouraging.

However, large-scale clinical trials are still evolving. At present, nicotinamide should be viewed as a promising adjunct rather than established preventive therapy, and clinicians should be cautious not to overstate its role or substitute it for proven IOP-lowering strategies.

Patients frequently ask about marijuana. While cannabinoids can transiently reduce IOP, the effect is short-lived, inconsistent, and impractical, requiring frequent dosing with systemic and psychoactive side effects. Current evidence does not support marijuana as a safe or effective strategy.

Other lifestyle considerations are increasingly recognised. Sleep quality and nocturnal physiology may be relevant, particularly in progressive or normal-tension glaucoma.

Nocturnal IOP elevation, side-sleeping with the affected eye dependent, and untreated obstructive sleep apnoea have all been associated with disease progression. Systemic blood pressure also requires balance, as both hypertension and excessive nocturnal hypotension may compromise optic nerve perfusion.

Certain physical activities warrant discussion in advanced disease. Sustained inverted yoga positions, heavy breath-holding, and repeated Valsalva manoeuvres can cause significant transient IOP elevations. Advising moderation rather than avoidance is usually appropriate.

Rapid ingestion of large fluid volumes may also cause short-term pressure spikes, and simple advice to spread fluid intake can be helpful.

Early detection remains central to prevention. Visual field testing is essential, but structural damage often precedes functional loss.

Optical coherence tomography (OCT) has

therefore become fundamental in contemporary glaucoma care. Increasing emphasis is being placed on macular ganglion cell analysis, where damage may be detected earlier and more reproducibly than visual field change.



Longitudinal OCT monitoring, particularly of the ganglion cell layer, allows progression to be identified at a stage when intervention is most effective, often before functional loss becomes apparent to the patient.

Glaucoma prevention is no longer defined by a single pressure reading or a reactive treatment model. It requires thoughtful risk stratification, attention to systemic and lifestyle factors, and intelligent use of modern imaging.

By identifying vulnerability earlier and intervening appropriately, clinicians can meaningfully reduce the long-term burden of glaucoma and preserve vision across a lifetime rather than responding only once damage is done. ●

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