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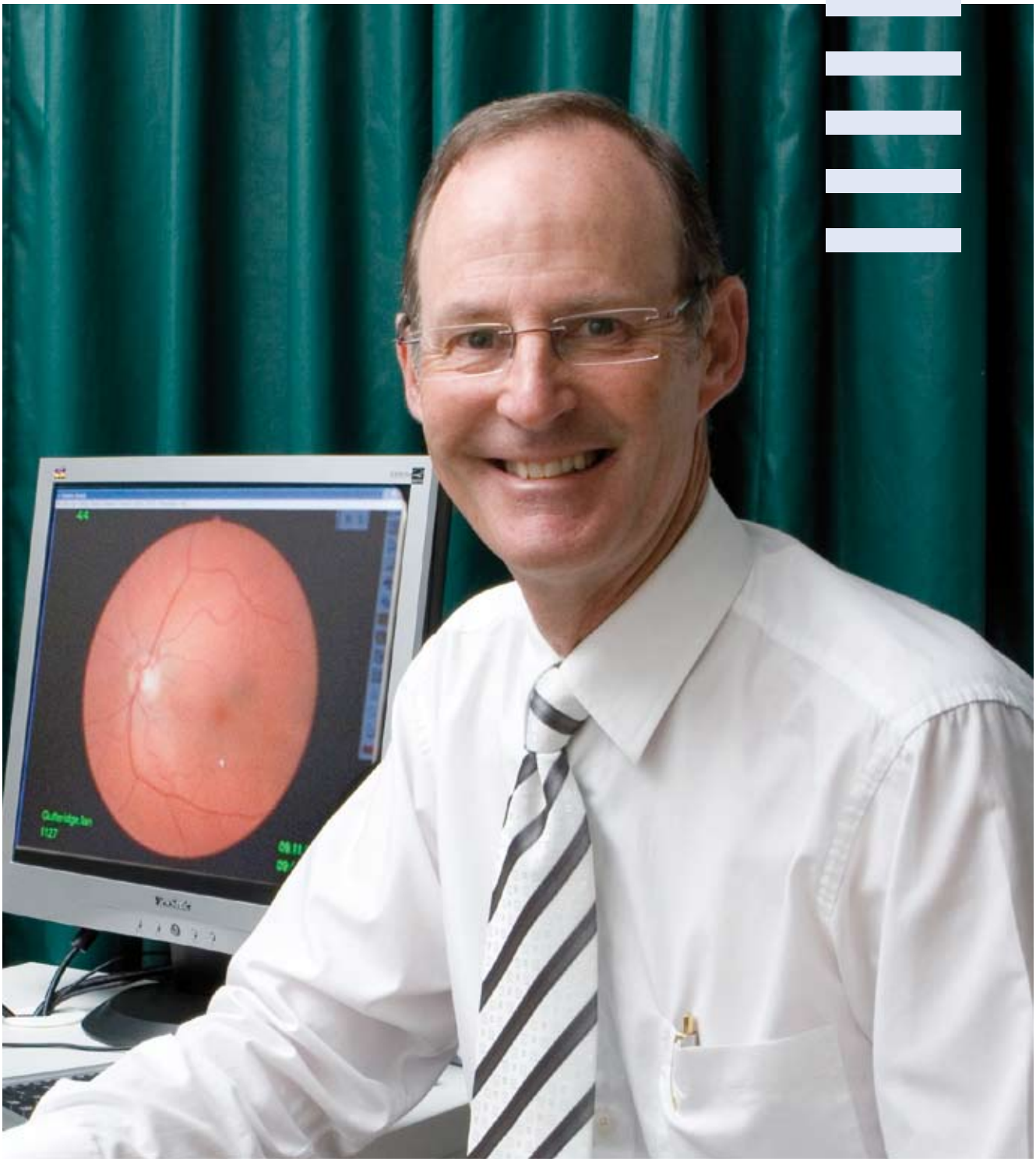

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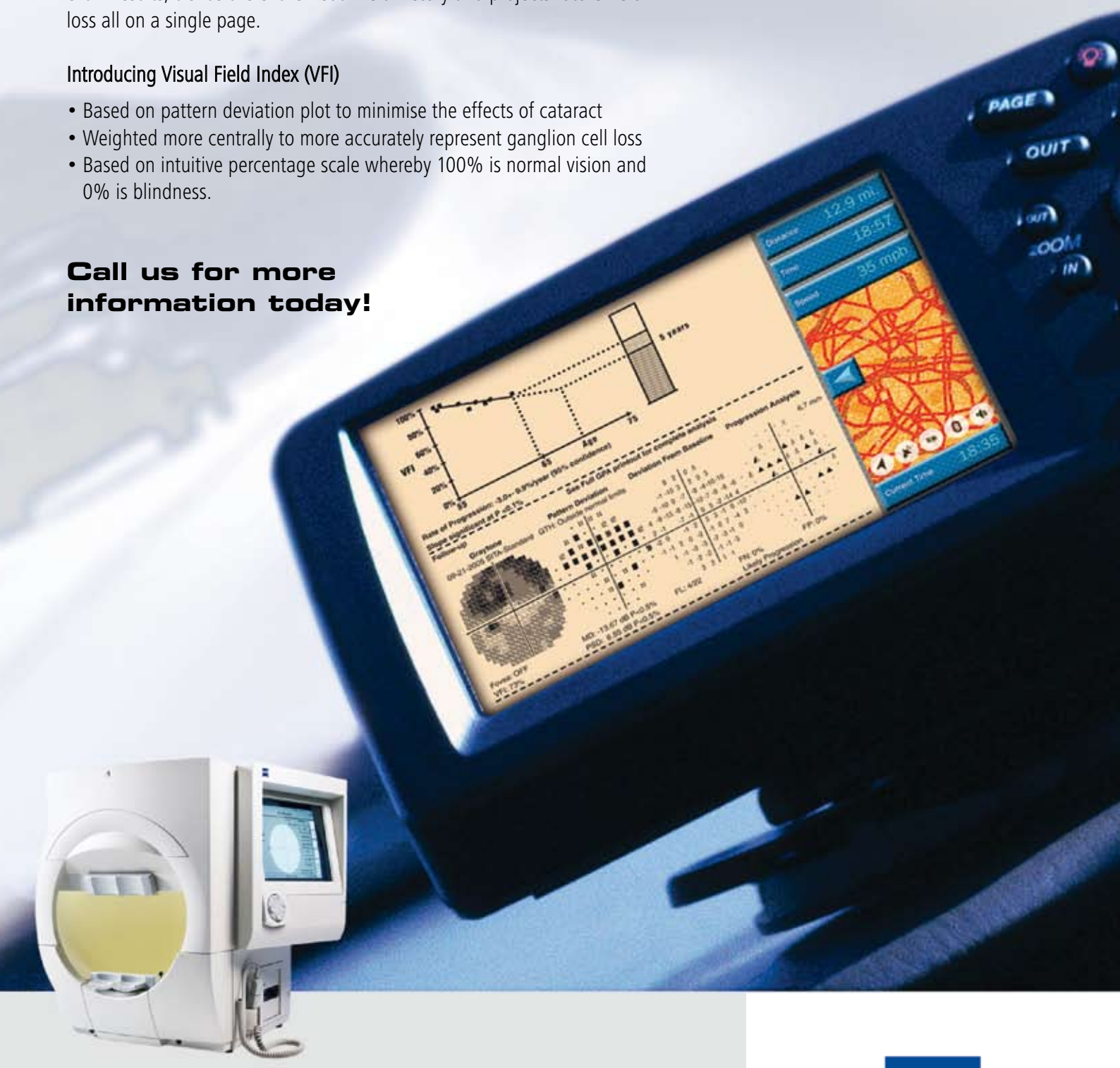

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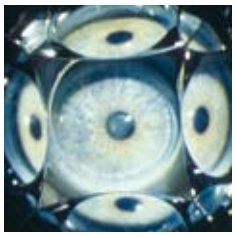
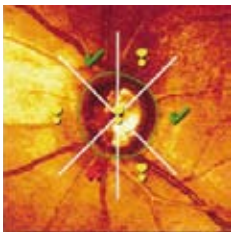
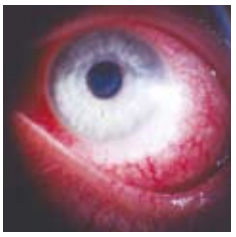
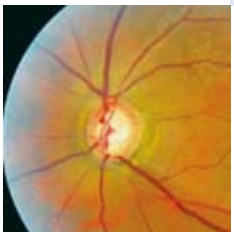
Contents

2 **Take the next step** David Cockburn
4 **Update on angle closure glaucoma** Lance Liu
8 **Children's future is brighter** Wendy Marshman
11 **IOP spiking** Charles McMonnies
12 **Acute anterior uveitis** Ian Gutteridge
14 **The future for medication** Rafael Chan
14 **Clinical tip: administering eye-drops**
15 **Why bother with gonioscopy?** Ian Douglas
16 **Letter from New York** Murray Fingeret
17 **Ocular hypertension** Philip Anderton
18 **Intraocular pressure fluctuation** Bang Bui
20 **A sceptic and his POAG** Richard Vojlay
22 **PBS: we're in for the long haul**
24 **Three-pronged attack** Lee Pepper
25 **What's going on here?** Jeffrey Chibert
25 **VFI regression trend analysis**
26 **Stick to it—patient persistence**
28 **Family matters**
28 **Life goes on: patients' perspective**
30 **Medical treatment** Catherine Green
33 **OCT essential to preserve vision** Randal Lamont
35 **Retinal nerve fibre layer defects** Algis Vingrys
36 **Multidisciplinary approach** Jonathan Crowston
38 **Anti-glaucoma agents table**
40 **Medmont-Star conversion** Mark J Walland

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Optometrists Association Australia
ABN 17 004 622 431

204 Drummond Street
Carlton VIC 3053
Tel (03) 9668 8500
Fax (03) 9663 7478
g.oshry@optometrists.asn.au
www.optometrists.asn.au

COVER
Melbourne optometrist
Ian Gutteridge

Photography
Belinda Strodder

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Go on, take the next step

With perhaps half the cases of normal tension glaucoma undetected, optometrists can rise to the challenge.

Dr David M Cockburn
Sandringham VIC

Guest editorial

Glaucoma was a more simple disease when I was a student well over half a century ago. There were two types of glaucoma, 'chronic simple' and 'angle closure', which were easily defined.

Chronic simple glaucoma was the condition in which the intraocular pressure was greater than a magic number of 21 mmHg and anything below that was normal so diagnosis, if not the disease itself, was indeed simple. Easy, wasn't it? Perhaps that is the reason it was called simple glaucoma.

There could be a problem when a patient presented with an IOP that was exactly 21 mmHg. This could be resolved easily by making a repeat Schiotz tonometer reading, which could be relied on to give a different result. After a second reading leaving 10 grams of metal sitting on the cornea for half a minute, the pressure would happily fall back into the normal range.

In the mid 20th Century, performing tonometry was at the cutting edge of our practice routine because putting drops in eyes was considered by many to be outside optometry's field of practice. In those days the only wind instruments were in symphony orchestras. It was unthinkable in some quarters that an optometrist should so much as touch the eye and heaven forbid they should presume to take a measurement that could determine a diagnosis.

The officially approved optometric technique for estimating IOP was digital tonometry, which involved both the optometrist and the patient closing their eyes, the optometrist placing the two index fingers on the upper lid and alternately palpating whatever structures lay beneath. It was generally held that using this technique, an experienced clinician could differentiate a glass eye from an eye having normal pressure.

Angle closure glaucoma was more of a problem; fortunately rarely seen by optometrists but its lurid textbook description was never forgotten. It was said to present in a patient already effectively blind, with a blood red eye and a well anchored pupil, and in so much diffusely distributed pain it could–and was–occasionally mistaken for bowel obstruction.

The late Dr Ronald Lowe was responsible for popularising the fact that angle closure glaucoma also and more commonly presented in a chronic or 'creeping angle closure' form. He wrote several groundbreaking papers on the topic, noting in one that general medical practitioners detected 75 per cent of these cases while optometrists neglected 25 per cent of theirs.

Visual field analysis became popular in those early days because it added another step toward diagnostic competence. The early, more advanced optometrists copied Professor Bjerrum, the father of perimetry, and installed a black cloth-covered board on the back of the consulting room door. Later, two-metre screens became the norm

where space permitted.

With this equipment and a white-tipped black rod you could easily create by suggestion a scotoma exactly where you expected it should be. This was achieved by waving the wand rapidly over areas where you had no such expectations and then slowing dramatically where the scotoma was expected to lie.

There was considerable doubt about the earliest detection of glaucomatous field loss, some textbooks maintaining that its earliest manifestation was enlargement of the blind spot. This too could be influenced by moving the target from the blind spot to the seeing areas or vice versa. Starting from the blind spot and moving outward tended to produce the larger physiological blind spot, whereas moving from seeing areas into the blind spot produced a smaller one.

Prior to the introduction of perimetry, we relied on the confrontation technique, which sometimes, in skilled hands, could detect a really solid hemianopia. The confrontation method may still be a useful clinical exercise in severe field loss in patients unable to co-operate during more sophisticated examination and possibly to maintain the examiner's digital flexibility.

If all this sounds bizarre in the context of modern glaucoma detection and optometry's contribution, give some thought to the subsequent management of the disease.

Treatment of simple glaucoma was indeed simple. It was treated with pilocarpine; mild and newly diagnosed cases with 0.25%, more severe or unresponsive cases with 0.50% increasing in steps to a maximum of 4.0%, after which unresponsive patients were offered the alternative of prayer or surgery.

Patients rarely complied with medical treatment because the drops caused pain and a tiny pupil, all but eliminating vision in those having any degree of cataract and making night vision for old-fashioned nightly pursuits such as putting out the cat and the milk bottles dangerous to life and limb. Some of my patients confided that they used the dreaded pilocarpine only for a day or two before their next ophthalmology review.

Angle closure glaucoma was usually treated with a combination of surgery and drops and was perhaps more effective than the treatment of open angle glaucoma. The treatment of normal tension glaucoma was not an issue because the condition was simply not recognised, leaving probably up to 40 per cent of open angle glaucoma to run its course.

The evidence suggests that not much has changed, with an educated guess that at least half the cases of normal tension glaucoma remain undetected.

Gonioscopy is the essential clinical procedure for assessment of angle closure or risk of it occurring. Australian optometrists and even some ophthalmologists were slow to adopt this technique.

A member of the Victorian College of Optometry travelled to the USA in 1976 as a visiting professor but had a hidden agenda of learning how to apply this important technique. He was disappointed to find that the major US optometry school at that time did not have anyone with the necessary skills and had never taught these skills.

Undeterred he joined forces with a British optometrist also temporarily working at that school. They took turns at being subject and examiner, mastering the technique by trial and error, changing places as their corneal epithelial damage dictated. They eventually became proficient and even recorded good quality colour photographs, which at that time was an unusual practice.

On his return, gonioscopy was introduced to Australian optometry and a firm friendship cemented by mutually inflicted corneal abrasions still exists between the two optometrists.

Most optometrists in practice today would assume that binocular indirect ophthalmoscopy is a relatively recent addition to our clinical investigative armamentarium. In fact it was practised in at least one Australian private practice more than 50 years ago.

Bausch and Lomb produced a stand-mounted instrument that provided an excellent binocular image. The reason it did not become popular was that pupil dilation was necessary and we were led to believe that this would almost certainly lead to angle closure and permanent vision loss. In 1957 this instrument was converted to a retinal camera by a clinical optometrist. It produced the first fundus photographs taken in Australia.

Another early foray into new territory was the development of an Australia made, binocular indirect ophthalmoscope through the collaboration of optometrist Don Schultz and ophthalmologist Professor Gerrard Crock. One of the first production run of these instruments was in use in private optometric practice in the early 1960s, where it remains today in retirement but good working order.

Much has changed with optometrists providing glaucoma detection with high sensitivity and treatment tailored to the individual needs of patients. Intraocular pressure is generally brought under control with a minimum of patient grief and relatively low cost.

Unfortunately, this does not guarantee protection against vision loss but at least it appears to slow the process to the degree that most patients retain useful and adequate vision until another condition leads to their demise. Neuroprotection and stem cell therapies are the new glaucoma grail; we shall watch the progress of these modalities with a great measure of hope.

> **The officially approved optometric technique for estimating IOP was digital tonometry, which involved both the optometrist and the patient closing their eyes, the optometrist placing the two index fingers on the upper lid and alternately palpating whatever structures lay beneath ... an experienced clinician could differentiate a glass eye from an eye having normal pressure.**

The future of eye care has been strengthened by co-operation between optometry and ophthalmology. Continued and growing co-operation between these professions bodes well for the battle against eye disease. ■

Update on angle

Recent advances have changed the definition, detection and treatment of angle closure glaucoma.

What type of glaucoma does the patient have?

Primary angle closure glaucoma (PACG) is characterised by optic nerve damage where the elevated intraocular pressure (IOP) is due to a blockage of the anterior chamber angle (ACA) by the iris (Figure 1) in the absence of secondary causes. Primary open angle glaucoma (POAG) is where the optic nerve is damaged and there is related raised IOP but no blockage of the ACA by the iris (Figure 2) in the absence of secondary causes.

Epidemiological studies[1] show that both POAG and PACG exist in all glaucoma population studies.

What happens in PACG?

In PACG, the mechanism(s) that cause the blockage of the ACA by the iris can be divided into pupil block, lens-induced and plateau iris, although there is usually a combination of all three (so-called mixed mechanism).

This leads to appositional and/or synechial closure of the angle, which is characterised by the iris touching the trabecular meshwork or iridotrabecular contact (ITC).[2]

Recent evidence shows that the elevated IOP in PACG can also be the result of damage to the trabecular meshwork.[3] Episodic ITC can cause the IOP to fluctuate, which can lead to intermittent symptoms.*

Over time, repeated ITC may lead to permanent damage to the trabecular meshwork and/or synechial closure, leading to a chronic elevation of the IOP, which increases the risk of developing glaucoma. Rarely does the whole angle block off, causing a rapid rise in IOP that can lead to loss of vision over minutes to hours.

Figure 1. Gonioscopy showing angle closure. Photo: Lance Liu

How is glaucoma diagnosed?

Glaucoma is diagnosed on the basis on the appearance of the optic nerve and the presence of other risk factors, such as IOP. The findings of gonioscopy determine the type of glaucoma the patient has. Gonioscopy should be performed at the initial or subsequent consultation and repeated at yearly intervals, as the ACA can narrow over time.

A video entitled 'Is the Angle Open or Closed–A Video Guide to Modern Gonioscopy' can be view at www.apaophth.org in the glaucoma section under education online.

ITC is a common feature of the various mechanisms of angle closure. Traditionally, an occludable angle was thought to occur when there is blockage or ITC of greater than 270 degrees of the ACA.[4] Given that the level of IOP depends on not only the extent of angle closure but also the amount of trabecular meshwork damage, it is thought that you only need more than 90 degrees of ITC for the angle to be at risk of damage.[2] This can cause the IOP to rise, which increases the risk of developing glaucoma.

Many eye carers rely on the Van Herrick measurement[5] or central anterior chamber[6] depth to assess the risk of angle closure. These are not sensitive enough (62-70 per cent) to detect angle closure so this condition may be missed if gonioscopy is not performed–especially in patients with a plateau iris configuration. More is missed by not seeing than not knowing.

Unfortunately, gonioscopy is not routinely performed by all eye carers and recent evidence shows that the appearance of the angle can change in different lighting conditions.[7,8] Angles that look 'open' in light settings can become 'closed' in dark lighting conditions when the pupil is physiologically

closure glaucoma

Dr Lance Liu
MBBS FRANZCO
Preston Eye Clinic, Preston VIC

dilated (Figures 3A and 3B).

Instead of assessing whether the angle is *open*, gonioscopy should now be performed to assess whether the angle is *closed* by looking for ITC. This is most likely to occur when the pupil is physiologically dilated.[9] Gonioscopy should be performed in a completely dark room with a very small slitlamp beam and care should be taken not to shine any light onto the pupil.[10] There are now anterior segment OCT imaging machines that are more sensitive in detecting and confirming ITC.[11,12]

How is PACG classified?

The conventional classification of PACG was based on the presence or absence of symptoms and IOP; divided into latent angle closure glaucoma, intermittent or sub-acute angle closure glaucoma, acute angle closure glaucoma and chronic angle closure glaucoma. However, there are problems with this classification in that each sub-type implies the presence of glaucoma (of which the first three are not glaucoma but can lead to it) and symptoms are usually present in only 25 to 33 per cent of cases.

Given that the ACA narrows over time, a new classification of PACG has been developed by ISGEO[13] that follows the natural disease process and is based on the appearance of the ACA and optic nerve. A patient with a blockage of the angle or ITC is classified as:

- primary angle closure suspect (PACS)—the patient is at risk of developing damage to the trabecular meshwork
- primary angle closure (PAC)—the patient has developed damage to the trabecular meshwork that causes the IOP to rise or fluctuate
- primary angle closure glaucoma (PACG)—the patient has developed the glaucoma secondary to the rise in IOP.

What happens if the patient is a PACS or has PAC?

A PACS patient has demonstrable ITC with a normal IOP and optic nerve. It has been shown that 20-25 per cent of these patients will develop elevated IOP or damage to the drainage system (PAC) over five or six years.[14-15] A patient with PAC has demonstrable ITC with raised IOP or damage to the drainage angle but a normal optic nerve. It has been shown that up to 28 per cent of these patients will go on and develop PACG over a five-year period.[16] There are ongoing studies looking at how to manage these patients.

There are two management options available to these patients. One may elect to observe the patient by taking baseline optic disc photos. The patient needs to be reviewed annually with eye examination and repeat gonioscopy as the angles narrow over time. Alternatively, one may perform a prophylactic laser iridotomy to stabilise the IOP by opening up the drainage angle (that is, treating ITC) and reduce further damage to the trabecular meshwork. It has a high success rate with minimal complications such as worsening of the IOP or blurring of vision.

Although it is commonly taught that a prophylactic laser iridotomy is performed only to reduce the risk of patients developing 'acute angle closure glaucoma', it can also reduce the risk of developing PACG. The more risk factors the PACS patient has of developing angle closure glaucoma, or if the patient has PAC, the more one is likely to intervene.

Continued page 6

Figure 2. Gonioscopy showing an open angle. Photo: Lance Liu

Update on angle closure

From page 5

These risk factors include:

- demographic risk factors such as age, female, Asian origin
- family history of glaucoma
- medical conditions that require frequent pupil dilation such as diabetes
- poor or delayed access to ophthalmic care

and/or the presence or development of:

- intermittent symptoms of raised IOP*
- elevated IOP or signs of previous elevated IOP
- damage to the trabecular meshwork, for example, patchy pigmentation or PAS
- glaucomatous optic nerve damage.

* Sixty per cent of those with intermittent symptoms will develop PACG.[17] These include headaches, blurred vision or halos or rainbow colours around lights. The patients' interpretations of these symptoms can vary but are clinically important in the presence of ITC.

What happens if the patient has PACG?

Patients diagnosed with PACG have damage to the optic nerve resulting from raised IOP from a blockage of and/or damage to the ACA. Initially, the IOP needs to be lowered with IOP lowering medications, then a laser iridotomy is performed to stabilise the IOP by opening and reducing further damage to the trabecular meshwork, but it does not usually lower the IOP. Finally, a visual field test and optic nerve imaging need to be performed to assess the degree of damage and to be used as a baseline for assessing the efficacy of ongoing treatment.

What happens if the laser iridotomy fails to open the angle?

It is a common misperception that the angle 'automatically' opens following the laser iridotomy. PACG is usually a multimechanism disease and ITC can still persist or recur in the presence of a patent laser iridotomy. Gonioscopy must be performed after the laser treatment to determine if ITC has been abolished (that is, has the angle 'opened') or if there is undiagnosed PAS. It also needs to be repeated at yearly intervals as ITC can recur. Depending on the stage of the disease or glaucoma, the options available to treat persistent ITC are pilocarpine, laser peripheral iridoplasty, early cataract surgery or observation.

If the IOP level is too high despite maximal tolerated medical therapy (usually where there is severe damage to the drainage angle, for example, extensive synechial closure) one may opt for glaucoma filtering surgery. Each option has its own advantages and disadvantages, depending on the degree of invasiveness, long-term results, potential complications and the patient's eye, health and wishes. Long-term studies are underway to look at the long-term effectiveness of each option.

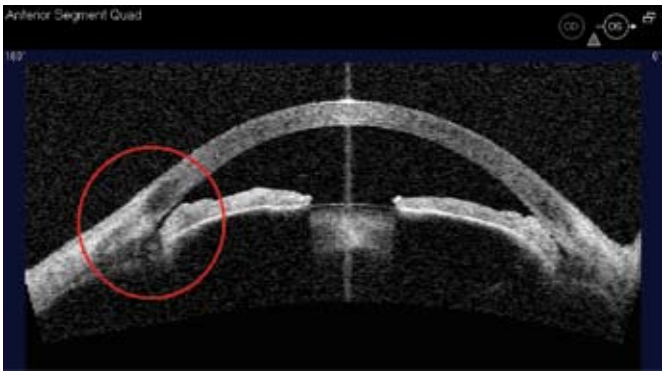

Figure 3A. Anterior segment OCT images of the angle in light conditions showing a narrow but open angle. Photo: Lance Liu

Where does acute angle closure glaucoma (AACG) fit into the new ACG classification?

AACG is now termed acute primary angle closure (APAC) and is an emergency eye condition. It is caused by a sudden rise in IOP due to a complete blockage of the ACA. This can occur out of the blue or on a background of intermittent symptoms.* The longer the symptoms are present, the greater the chance of developing severe damage to the optic nerve (glaucoma) and the trabecular meshwork.

How is AACG/APAC diagnosed and managed?

AACG or APAC is a clinical diagnosis. The patient usually experiences a sudden onset of headache, loss of vision and/or nausea and vomiting. An examination of the eye reveals a cloudy cornea, a mid-dilated pupil, shallow anterior chamber and the IOP is very high (usually 50 mmHg or more). There is usually a poor view of the fundus.

First, one must ascertain if there are medical conditions such as asthma or allergies to sulphur containing medications that may exclude certain treatment options. Then the principles of managing APAC are to:

- lower the IOP
- determine and treat the cause of the blockage of the ACA.

Most start with medical treatment to lower the IOP by administering a STAT dose of:

- timolol 0.5% if there is no history of asthma or COAD and
- brimonidine and
- oral dose of acetazolamide 500 mg, if there is no allergy to sulphur containing medications.

Pilocarpine is not useful in this situation as the pupil does not contract when the iris is ischaemic in the setting of the high IOP. Brinzolamide or dorzolamide does not usually add to the IOP lowering in the presence of systemic acetazolamide. However, it may be used as an alternative to acetazolamide if the patient is unable to take the latter medication orally. The IOP should be checked one hour after medical treatment was initiated. During this time, arrangements need to be made for the patient to be seen by

an ophthalmologist or at a tertiary referral centre on the same day.

Once the IOP is lowered, the cornea may be clear enough to allow gonioscopy to be performed to confirm the diagnosis of APAC and exclude causes such as rubeotic glaucoma, which can give a similar clinical picture. In the case of APAC, the ophthalmologist will then perform a laser iridotomy or a laser peripheral iridoplasty to open the drainage angle.

After the eye settles down, gonioscopy needs to be repeated to confirm whether the angle has opened. The patient may still require IOP lowering treatment, depending on the amount of damage to the trabecular meshwork and optic nerve. A laser iridotomy is performed in the fellow eye as 50-75 per cent of patients develop APAC in this eye over 5 years.[18,19] It is important to follow up these patients long term as the angle can still narrow over time and cause further damage to the trabecular meshwork.

If there is a contraindication to any component of the medical treatment of APAC, it is best not to use the medications in question. Use the medical treatments that are not contraindicated and urgently transfer the patient to an ophthalmologist. A laser iridotomy or laser peripheral iridoplasty can be used to lower the IOP if it remains high, and by opening up the drainage angle. If the IOP remains high despite medical or laser treatment, an urgent glaucoma filtering operation or trabeculectomy is usually performed. This creates an alternate pathway for the fluid to leave the eye, which drains to the subconjunctival tissue.

What does it mean for the patient?

Routinely, each part of the eye is examined, from the front to the back, to determine if there is a problem. As most of patients with glaucoma exhibit no early symptoms, from a glaucoma perspective, the eye examination is performed to exclude that the patient does not have glaucoma and that the patient is not at risk of developing glaucoma.

This includes taking a detailed clinical and family history of eye problems and performing an examination to look for conditions that are associated with glaucoma, for example, pseudoexfoliation, pigment dispersion or occludable angles (ITC).

Over time, the drainage angle narrows that can lead to PACG so intervention at an earlier stage may reduce the risk of the latter occurring. If ITC is demonstrated in an otherwise normal eye, one may elect to just observe but if the patient has a number of risk factors for developing PACG, a laser iridotomy needs to be considered to reduce the risk of developing glaucoma (acute or chronic). If the IOP is elevated in association with ITC, laser treatment may hopefully stabilise the IOP and/or the glaucoma. In either case, the patient still needs to be followed at regular intervals as the laser treatment is not a cure for this condition.

The problem lies in the detection of angle closure. Gonioscopy needs to be performed to see whether the angles are closed by looking for ITC. It needs to be performed in all patients and repeated at regular or yearly intervals. Both angle closure and open angle glaucoma exist in our population and are associated with a number of risk factors, of which IOP and angle closure or ITC are modifiable. We await the results of further long-term studies regarding this interesting disease.

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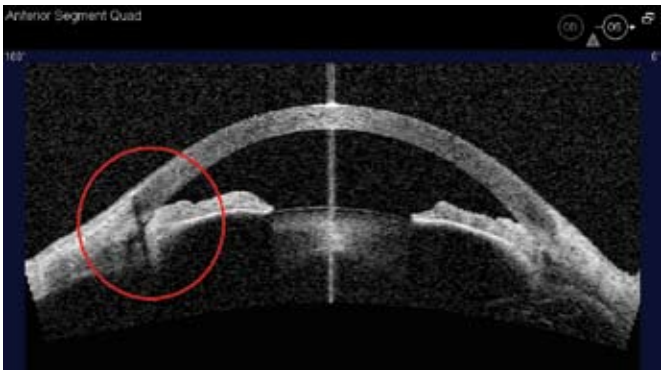

Figure 3B. Anterior segment OCT images of the angle in dark conditions of the same patient showing the 'open' angle has 'closed', characterised by iridotrabecular contact. Photo: Lance Liu

Children's future

Every child should have a dilated examination to perform retinoscopy and fundal examination at their initial visit to an ophthalmologist or optometrist, regardless of the assumed diagnosis.

In 1939 J Ringland Anderson wrote: 'The future of children with hydrophthalmia (primary infantile glaucoma) was bleak ... with little hope of preserving sufficient sight to permit the earning of a living.'

Today the future of children with 'hydrophthalmia' or primary infantile glaucoma is much improved and this changed with the introduction of microsurgical techniques and the operating microscope in the 1960s. Better topical medications that aid the lowering of intraocular pressure (IOP) have also assured the continued vision in these children and those with secondary glaucomas.

Incidence and classification of the paediatric glaucomas

In the paediatric age group, glaucoma is a heterogenous group of disorders and there are several classification systems. Probably the best is that suggested by DeLuise and Anderson in 1983[1] as is divides the disease into primary and secondary disorders, and as the secondary infantile glaucomas are associated with different variables they are further classified according to a disease process. This system circumvented the need to differentiate between confusing syndromes that had been grouped on the basis of superficial characteristics (Table right).

Primary paediatric glaucoma is rare with an incidence of approximately one in 10,000 in Australia and is usually manifest at birth or prior to the age of three years. It is less common than primary open angle glaucoma in adults but the most common form of glaucoma in children. It is usually a sporadic occurrence but there are reported cases of recessive and autosomal dominant inheritance.

The incidence is increased when founder effect or a high rate of consanguinity is found in a population. The 'founder effect' is a gene mutation that occurs in high frequency in a specific population due to that gene mutation being present in a single or small number of ancestors.[2]

> **The infant glaucomatous cup is different from that of the adult. It is typically round rather than oval, steep-walled and central. It tends to enlarge circumferentially, which is thought to be due to stretching of the scleral canal.**

Primary paediatric glaucoma is due to an isolated maldevelopment of the trabecular meshwork and not associated with any other ocular disease that could raise IOP. It is usually bilateral, although the initial presentation may be asymmetric. In one-third of cases a significant rise in IOP may occur in only one eye.

Diagnosis

Presentation is often the classic triad of epiphora, photophobia and blepharospasm. In young children, elevated pressure causes enlargement of the globe, as the corneal and scleral collagen has not hardened sufficiently to prevent expansion with raised IOP. In the early phases, this may lead to intermittent clouding of the cornea but with further distension and breaks in Descemet's membrane (Haab's striae), corneal oedema persists and the full picture of congenital glaucoma with 'buphthalmos' is present.

The normal neonatal cornea has a diameter of 10 to 10.5 mm and increases a further 0.5 to 1.00 mm in the first year of life. A diameter greater than 12 mm in the first year of life is highly suggestive of glaucoma. To make the diagnosis of congenital glaucoma often requires an examination under anaesthetic (EUA) to elucidate the type of glaucoma and examine the eye fully so that a treatment plan can be made.

At the examination under anaesthetic the following features should be observed.

1. **Corneal clarity and horizontal corneal diameter**
2. **Refraction**

Determination of increasing myopic shift in refractive error may be a clue to increasing eye size due to undetected changes in IOP.

3. **Intraocular pressure measurement**

All anaesthetics alter the IOP of the patient. This needs to be taken into account when assessing the pressure measurement, as does the means by which this measurement is made. Previously, the Perkins hand-held tonometer was the only available portable device and its accuracy can

is brighter

Dr Wendy Marshman
MBBS MD FRANZCO
Malvern VIC

Primary infantile glaucoma
(congenital glaucoma, trabeculodysgenesis)

Secondary infantile glaucoma

Associated with mesodermal neural crest dysgenesis

1. Iridocorneotrabeculodysgenesis
   - Axenfeld's anomaly
   - Rieger's anomaly
   - Peter's anomaly
   - Systemic hypoplastic mesodermal dysgenesis (Marfan's syndrome)
   - Systemic hyperplastic mesodermal dysgenesis (Weill-Marchasani syndrome)
2. Iridotrabeculodysgenesis (aniridia)

Associated with phakomatoses and hamartomas

1. Neurofibromatosis
2. Encephalotrigeminal angiomatosis (Sturge-Weber syndrome)
3. Angiomatosis retinae (von Hippel-Lindau syndrome)
4. Oculodermal melanocytosis (Naevus of Ota)

Associated with metabolic disease

1. Oculocerebrorenal syndrome (Lowe's syndrome)
2. Homocystinuria

Associated with inflammatory disease

1. Maternal Rubella syndrome
2. Herpes simplex iridocyclitis

Associated with mitotic disease

1. Juvenile Granulomatosis
2. Retinoblasoma

Associated with other congenital disease

1. Trisomy 13-15 (Patau's syndrome)
2. Rubinstein-Taybi syndrome
3. Persistent hyperplastic primary vitreous

DeLuise-Anderson (1983) classification of congenital and infantile glaucoma

be operator dependent. The invention of the Tonopen or electronic tonometer was less operator dependent but it does tend to over- and under-estimate pressures. The recent invention of the Icare tonometer (Tiolat Oy, Finland) appears to have circumvented most of these problems by producing a machine with accuracy similar to that of a Goldmann tonometer, which is not operator dependent. It is also well tolerated by most infants when performed awake, as the readings do not require local anaesthetic. This machine alone has decreased the incidence of repeated EUAs in the paediatric glaucoma population as IOP can often be measured in the consulting rooms.

4. Slitlamp examination

This is typically performed with a hand-held slitlamp (Kowa).

5. Gonioscopy

Evaluation of the anterior chamber angle is obligatory for the accurate diagnosis of the developmental glaucomas.

6. Ophthalmoscopy

Evaluation of the optic disc is essential and should be performed after dilatation of the pupil.

The infant glaucomatous cup has a configuration different from that of the adult. It is typically round rather than oval, steep-walled and central. It tends to enlarge circumferentially and this is thought to be due to stretching of the scleral canal. A decrease in cupping can occur in hours to days after the pressure is controlled in infants and children and again, this is thought to be due to decreasing the stress on the optic canal.

Continued page 10

Children's future is brighter

From page 9

7. Ultrasound

Repeatedly measuring the axial length of the eye is another way of monitoring progression of glaucoma as related to ocular growth. Similarly, with the control of pressure, studies have documented a decrease in axial length by up to 0.8 mm.[3]

8. Management

The mainstay of treatment in primary congenital glaucoma is surgical and the treatment of choice is usually a goniotomy (that is, incision into the trabecular meshwork under gonioscopic view), provided the cornea is clear enough to enable this view. If the cornea is opaque, a trabeculotomy can be performed via Schlemm's canal. For the secondary glaucomas, adult surgical techniques are more commonly performed, for example, trabeculectomy with adjunctive antimetabolites such as 5 FU and mitomycin, as well as drainage implant devices or ciliary body ablation with laser or cryotherapy. Unfortunately, there are complications with these techniques that are more prevalent in the paediatric age group, such as hypotony after surgery, as the wall of the paediatric eye is not as firm as the adult eye, as previously discussed, and the healing response of paediatric eyes is abundant, which makes filtering techniques harder to maintain. The choice of technique depends in part on the type of glaucoma, IOP and age of patient, as well as the history of previous treatments.

Medical therapy of paediatric glaucoma

There is a role for medical therapy in children with glaucoma, either as a short-term measure to lower pressure and help maintain corneal clarity while awaiting surgery or as an adjunct to surgery. The use of glaucoma medications in children does require caution.

- **Beta-blockers**

As with adults, these are contraindicated in children with asthma and in younger infants with a history of apnoeic episodes or cardiac disease. The use of 0.25% versus 0.5% is often recommended as well as a once daily dosing rather than twice, to decrease the plasma levels of drug and thus the potential for side-effects.

- **Carbonic anhydrase inhibitors**

Systemic and topical carbonic anhydrase inhibitors have been used in children and the potential for metabolic acidosis needs to be remembered, especially in smaller infants. There has also been a report of growth suppression in children on oral medications.[4] The more common problem with acetazolamide and children is that is may induce nocturnal enuresis (bed-wetting).

- **Prostaglandin related drugs**

These have been used for more than 10 years in children and the side-effects such as ocular irritation, lash growth and iris pigmentation as occurs in adult users are well documented. These groups of drugs appear to be well-tolerated in children, with few side-effects although their efficacy is variable.

- **Alpha-2 agonists**

This group of drugs has the potential for significant and life-threatening side-effects in children. The somnolence described rarely in adults with Brimondine can induce apnoeic episodes in children under the age of six years and the drugs are not recommended for children under this age. Poorly rousable states and even a coma-like episode have been reported.[5]

The use of Iopodine appears to be better tolerated in younger children and is probably relatively safe in children over the age of eight months. The first dose should be given under supervision and the child observed for at least four hours post dose to ensure that the potential for apnoea and decreased conscious state does not exist.

- **Cholinergic drugs**

The use of pilocarpine has a limited role after goniotomy and may be of use in aphakic glaucoma as an adjunct to other treatments. These drops are well tolerated in children; their major disadvantage is the frequency of the dose interval, that is, every six to eight hours as in adults, and the lesser effect on IOP lowering. The induced myopia may have a profound effect on vision.

We also need to remember that when the IOP is controlled, there will still be an ongoing need for visual rehabilitation in these children. Their refractive errors need correcting and amblyopia treated, and their ongoing surveillance will be life-long.

The differential diagnosis when a child presents with a watering eye includes nasolacrimal duct obstruction, megalocornea and Congenital Hereditary Endothelial Dystrophy (CHED) as well as glaucoma. I advocate that any child should have a dilated examination at their initial visit to an eye care specialist (ophthalmologist or optometrist) to perform retinoscopy and fundal examination, regardless of the assumed diagnosis as, to quote the late John Colvin, 'There are more mistakes made in medicine by not looking than not knowing'.

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IOP spiking and baropathic eye diseases

Charles W McMonnies
Adjunct Professor
School of Optometry and
Vision Science UNSW

Progressive axial myopia and glaucoma have been classified as baropathic diseases.[1] **A recent study of the effects of IOP spikes in keratoconus indicates that this form of keratectasia is also a baropathic disease.**[2] **In each of these diseases, a critical region of the sclera or cornea is susceptible to distending IOP forces.**

The posterior sclera yields in axial myopia; the lamina cribrosa in glaucoma; the corneal apex in keratoconus, and some other part of the cornea in other thinning diseases, including post-LASIK keratectasia.

Relentless field losses are difficult to explain when patients are fully compliant with their glaucoma treatment and show a significant reduction in IOP at follow-up visits. One suggestion has been that IOP spikes may be sufficient to progress these field losses.[1] The wide range of spiking mechanisms include eye rubbing, tear wiping, make-up removal, compresses for lid hygiene and to treat Meibomian gland dysfunction, pillow contact with lids during sleep, wearing various types of sleep masks, squinting to reduce blur or in response to glare, strenuous muscular effort and wearing swimming goggles.

Considering the wide range of potential spiking mechanisms, clinical tonometry may indicate the low point in a range of everyday IOP. The potential contribution to baropathic eye disease progression from IOP spikes depends on their size, duration, frequency, the number of years the spikes have been occurring and the number of different spiking activities to which an individual is exposed.

There are numerous mechanisms for axial myopia but ultimately it is IOP that distends the sclera and the weakened posterior pole yields.[3] High pressure is not necessary if the posterior pole is susceptible enough to distending forces. Consequently, progressive axially myopic eyes may continue to lengthen under normal IOP levels. However, IOP spiking may be a significant contributing factor and accelerate axial lengthening.

Eye rubbing is significantly associated with keratoconus development.[4] The IOP spike due to rubbing appears to be a causal factor. If the rubbing is sufficiently severe, frequent or long-lasting, the cornea may be wounded and tenderised by the mechanical trauma, and become more susceptible to IOP rubbing-related spikes.

People who develop keratoconus in the absence of a chronic habit of abnormal rubbing may have a genetic basis for having corneas that are more susceptible to IOP. If their cornea is weak enough, normal IOP distending forces may be sufficient to promote an ectasia. As is the case for progressive axial myopia and glaucoma, all keratoconus may progress more rapidly when there is significant exposure to IOP spiking.

Many IOP spiking activities are less likely to be encountered in older patients but increased age is not necessarily an indication of reduced exposure to IOP spiking activities. A case was reported of a 61-year-old man with glaucoma and continuing field losses despite having his IOP stabilised at 10 mmHg with medication.[5] He subsequently reported a daily routine that included 20 minutes of yoga headstands and strenuous exercises. He had IOP of 40 mmHg when he assumed a headstand posture and large IOP spikes occurred when he made strenuous muscular efforts. His fields remained stable when he gave up these activities.

Wearing swimming goggles was found to spike IOP by 4.5 mmHg[6] although IOP assessments in that study were done with subjects in a conventional sitting posture. Recumbent body positions, strenuous muscular effort and deep breathing can raise IOP independently of one another. When combined with wearing swimming goggles, IOP spiking might be more significant. A patient who swims laps regularly may be at risk of significant exposure to IOP spiking.

Patients who have or are at risk of developing baropathic diseases may benefit from counselling on the need to avoid IOP spiking activities. Assessing spiking risk can be facilitated using a survey structured instrument, available on request from c.mcmonnies@unsw.edu.au.

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Acute anterior

Associate Professor
Ian Gutteridge
MScOptom FAAO
Department of Optometry
and Vision Science,
and Private practice

'Atropine or homatropine?' is a frequent discussion point, bordering on a dilemma in managing a patient with acute anterior uveitis (AAU).

Cycloplegia and mydriasis have two important goals in AAU. One arm of the treatment is symptomatic: relief of pain and photophobia caused by iris and ciliary body spasm, which all agents will satisfactorily achieve. Patients are very thankful for this. The second is the breaking of fresh posterior synechiae through mydriasis. The benefit of this, although clinically more important, is not so obvious to the patient.

Posterior synechiae

Posterior synechiae risk trapping aqueous in the posterior chamber, mainly when there is extensive or complete posterior synechiae. Iris bombe and secondary angle closure can end in complicated uveitic. Even if fresh posterior synechiae are not present at first examination, if they develop, it would be preferable that they form in the dilated pupil position. Fortunately, as long as aqueous can flow past old adhesions, the synechiae do not cause much difficulty when a patient needs cataract surgery in the future.

Posterior synechaie form quickly in AAU. The double layer of the iris pigment epithelium both disintegrates and proliferates. Together with fibrin and protein in the aqueous, this forms the substrate for adhesion to the lens.

Initially adhesion is soft and tenuous but soon becomes rigid. As soon as AAU is diagnosed with the slitlamp, and as part of the work-up, the pupil should be dilated with tropicamide–in the consulting room. Posterior segment and vitreous inflammation must be considered early also, and viewing is made possible with the dilated pupil.

Synechiae tend to form when the pupil is small. Miosis is caused by the release of Substance P in inflammation and injury–especially during cataract surgery–with subsequent stimulation of the iris sphincter. Substance P is a neuropeptide acting as a neurotransmitter and a neuromodulator. It is also involved in transmitting information about damaged peripheral tissue to the central nervous system as part of the sensation of pain. Mydriatics must be applied regularly to counter the ongoing effect of Substance P and the break-down of mydriatics in the inflamed eye.

Which is the best agent?

Influential practitioners and experts often have a strong preference for one agent and prescribe this in most cases. What are the real arguments behind choosing from atropine, homatropine, cyclopentolate, tropicamide or phenylephrine? We know about their relative strength and length of action in the normal eye (Table right). Do they behave differently in the inflamed eye?

Important clinical questions can guide the choice of a mydriatic agent.[1]

- How severe is the anterior chamber reaction/inflammation ?
- What damage/iris adhesion already exists (present/previous episode)?
- Have new posterior synechiae formed?
- Are new posterior synechiae likely to form?
- How quickly will the inflammation be controlled by steroid use?
- Is the mydriatic strong enough to prevent inflammatory miosis redeveloping?
- How quickly will that mydriatic break down, lose effect–that is, how long would the treatment effect last?
- What preparations are available?
- How compliant will the patient be?

Your answer to these clinical questions should influence your decision. Most debate is about the choice of atropine or homatropine. Atropine has about 10 times the potency of homatropine, is a better cycloplegic, has some capacity to stabilise the blood aqueous barrier and causes more reduction in thickness of the lens.

However, it causes a more fixed pupil (risking synechial formation in the dilated position) and has more potential for systemic side-effects (presumably greater with more lengthy use).

It would be reasonable to use atropine in a moderate to severe case with posterior synechiae. In milder AAU without new synechiae, homatropine is a more logical option because some pupil movement may be possible. Of course, switching from atropine to homatropine or even cyclopentolate after control of inflammation is entirely logical.

Considerable effort must be put on this synechia to break it. The first line of attack is sufficient mydriasis, that is, using clinical judgement based on the questions above to choose an agent that is sufficiently powerful. A synechia may not break straight away.

A powerful corticosteroid should also have some effect through dissolving fibrin through fibrinolysis. This may occur over the first few days of treatment but the judgement must be made to also use a sufficiently potent mydriatic. If in doubt, one should use a stronger agent.[2] Effective steroid treatment becomes the main factor in preventing new synechiae forming in the dilated position.

Once the anterior chamber inflammation has been adequately controlled, synechiae are unlikely to form. Generally two weeks of control permits stopping the mydriatic. One measure of the absence of inflammation

uveitis

is the pupil remaining dilated without any mydriatic (as in the normal eye). Another approach is to change from a strong mydriatic to a weaker one after that initial control.

Consider a case of AAU with Grade 2-3 cells seen four or five days after onset of symptoms, with a moderate amount of inflammatory debris on the anterior lens surface.

For the record, my preference—or prejudgement—is initially the combination of Pred Forte (prednisolone actetate 1% and phenylephrine 0.12%) q1h and atropine 0.5-1.0% qid. This offers the twin mydriatic effect plus the most potent anti-inflammatory effect.

However, the clinician must consider the features of the individual case and then exercise clinical judgement according to the principles discussed here.

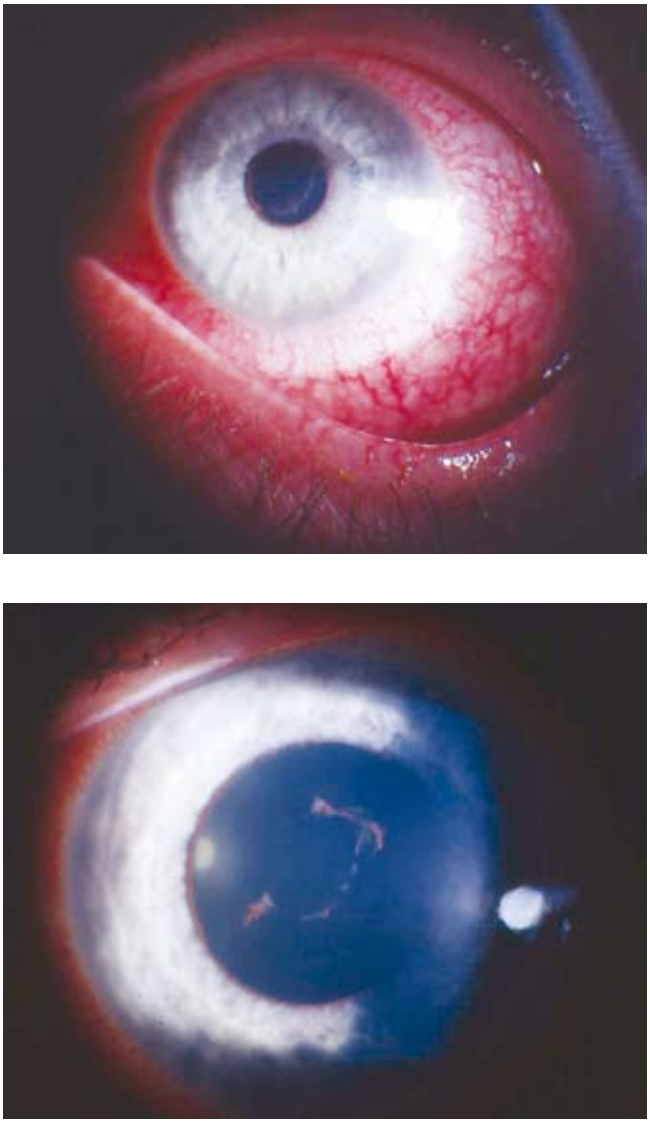

Figure 1. Patient with moderate AAU and developing posterior synechiae on Day 1 (top) before dilation and (bottom) 30 minutes after dilation with one drop of tropicamide

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Agent	Maximum dilation (min)	Recovery (days) normal eye	Proprietary name	Conc.	Bottle size
Atropine sulfate	30-40	7-10	Atropt	0.5% 1.0%	15 ml 15 ml
			Atropine	1.0%	Minim
Homatropine hydrobromide	40-60	1-3	Isopto-homatropine	2.0% 5.0%	15 ml 15 ml
Cyclopentate hydrochloride	20-45	1	Cyclogyl	1.0%	15 ml
Tropicamide	20-35	0.25	Mydriacyl	0.5% 1.0%	15 ml 15 ml
			Tropicamide	0.5% 1.0%	Minim Minim
Phenylephrine hydrochloride	60	0.3-0.4	Phenylephrine	2.5% 10.0%	Minim Minim

Mydriatic agents in Australia 2008 and some of their properties[2,3]

The future for medication

Rafael Chan
Alcon, Forth Worth USA

Current treatment for glaucoma includes topical pharmaceutical therapies, laser trabeculoplasty, and various surgical options, with trabeculectomy being the most common.

As we look to the future of glaucoma treatment, several trends emerge.

- The launch of fixed combinations within the pharmaceutical arena show great promise as they simplify treatment for glaucoma patients. Fixed combination products allow a patient to take one drop a day rather than three drops if the patient was taking the medications separately. This simplification of therapy helps with patient compliance.
- The development of benzalkonium chloride (BAK)-free pharmaceutical alternatives is providing physicians with additional options to treat glaucoma. Long-term exposure of BAK to the ocular surface has been shown to exacerbate symptoms of dry eye, which in an ageing population can be problematic.
- The development of sustained delivery systems for pharmaceutical therapy will be the ideal way to improve patient compliance. Compliance is a major concern for glaucoma patients who live with an asymptomatic disease for the rest of their lives. Sustained delivery systems that could last three to six months would ease the burden of daily drop therapy.

The future of glaucoma treatment is promising as new products and technologies allow for easier and more successful treatment. ■

Combination lowers IOP extra 20%

Dorzolamide hydrochloride when added to travoprost is an excellent and safe adjunctive agent that can lower intraocular pressures further.

A study evaluating changes in intraocular pressure after dorzolamide hydrochloride was administered to patients already using travoprost found that the agent can lower intraocular pressure by an additional 20 per cent.

Dorzolamide hydrochloride is a topical instilled carbonic anhydrase inhibitor used primarily as an adjunctive glaucoma therapy. After absorption through the cornea and stroma, dorzolamide hydrochloride inhibits carbonic anhydrase in the ciliary process. The result is a reduction in aqueous humour production and subsequent intraocular pressure decline.

The study also found that dorzolamide hydrochloride significantly shortens the retinal arteriovenous passage (AVP) time. This is the time it takes for blood to pass from the retinal artery to the vein for the affected hemisphere. The shortened AVP time may show that dorzolamide hydrochloride is a good medication for slowing glaucoma field progression.

Optometry 2008; 79: 501-504 ■

Clinical tip

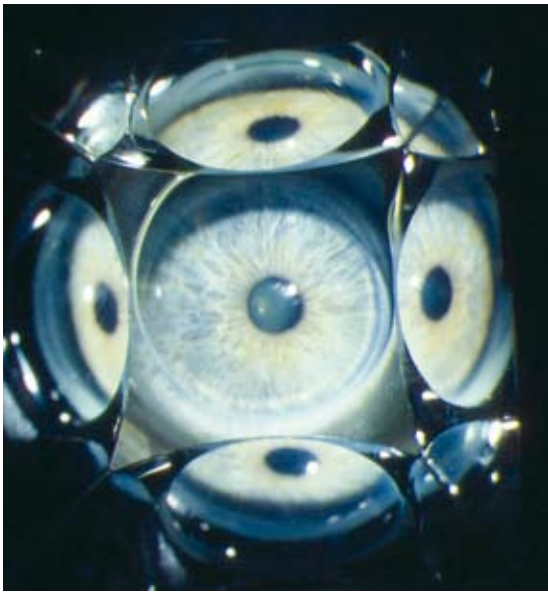
Administering eye-drops

Elderly, disabled or injured patients can find it difficult to administer eye-drops. Optometrists Paul Brand, Allan Ared and Michael Hare share their clinical tips on how this task can be made easier and more effective.

- To solve aiming problems in a patient with tremours, get them to tilt their head back about 70 degrees and place the bottle across the bridge of their nose. With the tip pointing toward the eye requiring drops, they should tilt the tip of the bottle toward the inner corner of the eye and squeeze.
- Dry eye sufferers can benefit from being prescribed TheraTears, as the angle of the applicator allows the patient to look straight ahead and still correctly administer the drops.
- Getting patients to lie down to administer anti-glaucoma or anti-inflammatory drops can be effective as these need to be instilled in the lower conjunctival sac. Having somebody else administer the drops may also be helpful.
- Goggles are available from pharmacies, which help contain the drops in the patient's eyes rather than have them spill onto their cheeks.
- Advising the patient (or helper) to aim for the pouch in the lower lid and instil the drops there provides them with a good-sized target area. Ensure the patient fixates on a target when administering drops. ■

Why bother with gonioscopy?

Ian Douglas
BsScOptom
Hampton VIC



Gonioscopy allows us to view the anterior chamber angle of the eye. It enables us to:

- more accurately diagnosis glaucoma type
- assess the risk of angle closure glaucoma
- assess the risks associated with pupil dilation
- assess the risk of glaucoma in congenital abnormalities
- assess angle status following trauma
- assess angle status with diabetes and retinal vascular changes
- investigate possible cause of unresponsive or recurrent uveitis

In clinical optometric practice the most commonly used gonioscopy lenses are the four-mirror or the Goldmann style three-mirror.

With the four-mirror lens (Zeiss, Posner, Sussman, Volk) all parts of the angle can be viewed with minimal rotation of the lens, as all four mirrors are inclined at the same angle.

There is no need for a viscous fluid between the lens and the cornea, as the tear film or saline is adequate lubrication. Following gonioscopy with such lubrication, the retinal view is unaffected and ophthalmoscopy can be done with minimal impact on the clarity of the image.

A commonly used alternative is the three-mirror lens (Goldmann, Ocular instruments), which has only one mirror aligned to allow the angle structures to be viewed. To view all different parts of the angle it is necessary to rotate the lens on the eye. A more viscous fluid such as single dose Celluvisc is required between the lens and the cornea. The view with this lens is more magnified at 0.93x compared to 0.8x for the four-mirror lens.

A lens called the Magna View is also available. It is a single mirror goniolens with the greatest magnification of any goniolens at 1.3x. This significantly improves the clarity and detail of the angle structures visible. Like the three-mirror lens it requires a viscous fluid between the lens and the eye.

Gonioscopy is typically conducted within a comprehensive examination as required by the presenting signs and symptoms of the patient. The patient has one drop of topical anesthesia[1] as would be used for applanation tonometry. Although in the past a lid speculum has been used to control the lids, I find it is rarely required.

The patient is positioned in the slitlamp and the gonioprism placed on the cornea with lubricating fluid as required. It is important to support the prism but not to exert pressure on the cornea as this will create folds in Descemet's membrane and affect the clarity of the image. Initially the prism should be parallel to the plane of the iris.

When I examine the angle I imagine myself sitting on the edge of the iris with my feet resting on the front surface of the crystalline lens, looking across the plane of the iris toward the angle.

It is best to assess the lower angle first (superior mirror position) as this is usually the widest and allows the best view of the angle structures. The structures that are visible are recorded and their relative appearance with regard to pigment, psuedoexfoliative material et cetera are noted. The superior, temporal and nasal angles are then examined and results recorded.

If the structures of the angle are not visible but the angle still appears open, it is often possible to indent the gonioprism toward the angle of interest and 'look' down into the angle structures.

Drainage of the aqueous at the angle occurs via the posterior trabeculum so when visible this gives an indication of the effectiveness of the angle at providing adequate drainage. Other than recording the visible structures, there are other grading systems that have been developed; the most common are those developed by Shaffer[2] and Spaeth.[3]

The Victorian College of Optometry in Melbourne runs a practical workshop for learning or enhancing skills in the technique of gonioscopy. These courses deal with the basics and more advanced aspects of the technique. They are usually run twice a year in June and November; to enrol contact the college.

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Letter from New York

MURRAY FINGERET writes about new medications and diagnostic tools used in the United States

Murray Fingeret, OD FAAO, is Clinical Professor, SUNY College of Optometry; and Chief, Optometry Section, Department of Veterans Administration, New York Harbor Health Care System

It is worth taking note of changes that are occurring in the management of glaucoma.

Therapy is being driven by the prostaglandin (PGs) class of drugs, which are more efficacious and safer than other drug classes. Historically, as drugs became more potent, safety was compromised. PGs have become the most commonly prescribed primary agent for the therapy of ocular hypertension and glaucoma. Most importantly, PGs efficacy and safety have simplified glaucoma care, leading to more optometrists becoming comfortable in the therapy of ocular hypertension (OTHN) and open angle glaucoma (OAG). The number of optometrists involved in the management of glaucoma is increasing monthly, as seen by statistics of new providers prescribing glaucoma agents.

New diagnostic instruments are allowing ODs to become more comfortable in glaucoma care. Digital photography has simplified the procedure of taking a photograph, allowing documentation of the optic nerve and retinal nerve fibre layer that was not available 15 years ago. Imaging instruments are evolving that are analogous to second opinions in the analysis of the optic nerve and retinal nerve fibre layer (RNFL). Combined with better software to detect visual field progression along with the recognition of the role of newer visual field technologies to detect early loss (FDT, SITA SWAP), ODs now have tools to diagnose as well as monitor glaucoma that were simply on our wish list just a few years ago.

Another new and important tool is the Ocular Hypertension Risk Calculator (http://ohts.wustl.edu/risk/calculator.html). Until recently, there was little consensus on how to manage the individual with ocular hypertension. Some clinicians may treat when the IOP exceeds 25 mmHg; others would wait until the 30 mmHg mark was breeched, while others would treat only if damage consistent with glaucoma was discovered.

The Ocular Hypertension Treatment Study provided data to show that in addition to IOP, corneal thickness, age, cup-disc ratio and visual field status should be considered. These five areas can be placed in a statistical model with the risk of an ocular hypertensive individual converting to glaucoma damage in five years calculated.

This validated model is not a guess and we then understand which of our patients is at significant risk of developing glaucoma, allowing proactive therapy. Others with a small risk can be followed over time. Currently, therapy is considered when the risk exceeds 15 per cent in five years but this number is not a cook book as other factors such as age, health and family history need to be considered.

The diagnosis and management of glaucoma is evolving quickly, due to the introduction of new medications and diagnostic tools. These tools are being embraced by optometrists so that they are more comfortable in glaucoma management. ■

When does ocular hypertension become glaucoma?

Dr Philip Anderton
BOptom PhD
Manilla NSW

Research in clinical and fundamental sciences is yet to discover the reason for ganglion cells dying in glaucomatous eyes.

Modern medicine is founded on the idea that a diseased organ is different from a normal organ, and that a skilled practitioner can identify the presence of disease through a process of diagnosis. To identify the presence of glaucoma in an otherwise normal eye is perhaps one of the most difficult diagnoses, not only within the spectrum of eye disease but for any human condition.

The critical diagnosis of glaucoma is made when a practitioner finds evidence of ganglion cell death (optic neuropathy) as evidenced by morphological changes to the optic nerve head, loss of retinal ganglion cell axons in the retinal nerve fibre layer, and the key appearance of characteristic depressions and scotomata in the patient's visual field.

Despite years of intensive research in both the clinical and fundamental sciences, there are few clear answers to the question of why ganglion cells die in glaucomatous eyes. A classic theory has been that high pressure in the eye causes changes at the optic nerve head, possibly damaging ganglion cell axons as they pass from retinal nerve fibre layer to optic nerve. Ganglion cell death might occur through a process of retrograde degeneration from damaged axon to ganglion cell soma.

This raises the question of just how important high intraocular pressure is as a cause of glaucoma. Do all eyes with high intraocular pressure ultimately develop glaucoma? More importantly, does lowering the abnormally-high IOP with topical hypotensive drops reduce the risk of such an eye developing glaucoma?

The presence of an abnormally high intraocular pressure is not diagnostic for glaucoma; however, it is now recognised as a significant risk factor for the later development of primary open angle glaucoma.

Between 1994 and 2004 a long-term, randomised, controlled multi-centre clinical trial, The Ocular Hypertension Treatment Study (OHTS), was conducted in the USA to attempt to answer this question. This study was conducted in multiple centres on a large number of patients with relatively high intraocular pressure (between 24 and 32 mmHg), but no other signs or symptoms of glaucoma.

They were divided into two groups: one group received no treatment, the other group was treated with topical hypotensive drops (average reduction in IOP was 22 per cent). Over a seven-year period, subjects in both groups were observed closely at six-monthly intervals for signs or symptoms indicating that their ocular hypertension had converted to glaucoma.

Useful summaries of this study and its outcomes are available at the National Eye Institute (NEI) Clinical Trials website.[1] The most significant finding was that treatment can halve the risk of conversion of OHT to glaucoma in the African-American population. However, in terms of the general population, the remarkable outcome[2] was that the risk of conversion to glaucoma in the untreated group over the first four years was low (five per cent) and not very different from the risk of conversion in the treated group (3.5 per cent).

Interestingly, in one six-month interval of the study (five years, 60 months; Figure 4 in Kass et al, 2004) there was a significant spike in the number of conversions to glaucoma in the untreated group (25 individuals, as opposed to 1-10 individuals per six months in the preceding five years and subsequent two years). Without this outlying data, the final difference between treated and untreated groups over the seven years would have been significantly reduced.

Over the seven years of the study about 13 per cent (one in eight) of those who were not treated developed open-angle glaucoma. In the treated group, only five per cent (one in 20) of ocular hypertensives developed open angle glaucoma. In other words, the decision to apply hypotensive drops to an eye with ocular hypertensive reduced the risk of it developing glaucoma from 13 per cent to five per cent

Given the cost and inconvenience of prescribing daily topical glaucoma medication, there are significant issues that practitioners and patients need to address in each individual case before making any decision to prescribe prophylactic glaucoma medication for ocular hypertensives.

Unfortunately, the OHTS study did not address the diversity that we see in patients at risk of glaucoma or with early glaucoma. In many patients, a therapeutic reduction in IOP appears to halt the progress of the disease and we find minimal or no changes between visits. In other patients, the changes appear to proceed rapidly, with significant erosions in optic disc morphology, nerve fibre layer and visual fields evident at each visit.

Continued page 18

When does ocular hypertension become glaucoma?

From page 17

These are the patients who need special attention and close collaboration with other practitioners to ensure that all available therapeutic options, including surgery, are made available as soon as possible after their diagnosis.

As primary eye-care practitioners, what can optometrists take from the OHTS studies? The most important outcome is the recognition that a person who has high intraocular pressure does not necessarily have glaucoma and, indeed, may have a relatively low risk of developing glaucoma.

The critical process in the examination of an ocular hypertensive is to ensure that *all* of the other risk factors for glaucoma are addressed with their subjective and objective examinations. Is there a family history of glaucoma? Do they have a history of diabetes and/or cardiovascular disease? Are there any significant morphological features of their optic disc and peripapillary retina? Do they need a visual field examination? Should they be examined every six months to rule out conversion from ocular hypertension to glaucoma?

In the end, each of these decisions can be made only on an individual basis with each patient.

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Intra pres

Although elevated intraocular pressure (IOP) has clearly been identified as a risk factor for glaucoma development (Gordon et al 2002; Miglior et al 2005) and progression (Heijl et al 2002; Leske et al 2003), many apparent clinical inconsistencies call into question our understanding of IOP.

For example, only a small proportion of those with high IOP develop glaucoma. On the other hand the majority of those with high IOP never show visual field deficits (Tielsch 1996). In addition, glaucoma progression is equally likely at all IOPs (Chauhan 1995) and can occur despite successful IOP lowering.

To explain such inconsistencies other characteristics of IOP have been considered, including the mean IOP, the peak IOP, the IOP time integral and the degree of IOP fluctuation. In particular undetected IOP fluctuations could account for many of the above clinical inconsistencies.

Sources of IOP fluctuation

It is well known that IOP undergoes variation, which can occur in the ultra-short-term (occur in seconds to minutes, such as from pulse beats, squinting, eye rubbing or Valsalva manoeuvre), the short-term (occur over hours to days, such as posture or circadian variation) and the long-term (occur over months or years, such as from ageing or response to drugs) as defined by the World Glaucoma Association Consensus Meeting on IOP (Medeiros et al 2007).

It is not surprising that studies demonstrate that single IOP measurements during clinical consultation during office hours miss the true IOP peak in many individuals (Collaer et al 2005; Hughes et al 2003). The question arises: are such IOP important in glaucoma development and progression?

Short-term IOP fluctuation and glaucoma development

A circadian rhythm exists that produces the lowest IOP in the night and a peak in the morning just after waking (David et al 1992; Drance 1963; Hughes, Spry et al 2003; Liu et al 2003). Such circadian fluctuations are normally 2–4 mmHg, and can be due to changes in aqueous production, outflow efficacy or some as yet to be identified mechanism. Nocturnal IOP measurement must take into account an increase of up to 5 mmHg due to a change in posture from upright to supine (Liu et al 1998; Liu et al 1999).

Assessment of IOP curves suggests that there is greater fluctuation during the diurnal period in patients with glaucoma (Liu, Zhang et al 2003). Other studies show that the degree of diurnal IOP fluctuation is related to the level of IOP, indicating that diurnal fluctuation itself is not an independent risk factor for the progression of glaucoma (Bengtsson and Heijl 2005; Sacca et al 1998).

Epidemiological studies have evaluated the role of IOP fluctuations as a risk factor for glaucoma progression. Visual field prognosis was found to be better in those with smaller IOP fluctuation (Bergea et al 1999). Gonzalez et al (1996) show that ocular hypertensive patients who went on to develop visual filed defect were more likely to show IOP fluctuation.

Asrani et al (2000) found that diurnal IOP fluctuation, as measured by home self-tonometry, was an independent risk factor for glaucoma progression after adjusting for office IOP, age, race, gender and severity of VF loss at baseline. However, Liu et al (2003) found that circadian IOP fluctuations were similar in untreated glaucoma patients and control subjects.

Long-term IOP fluctuation and glaucoma development

Long-term variation occur over months or years and includes both short-term variation as well as the effects of ageing and disease processes on IOP. Like short-term fluctuations the association between long-term IOP fluctuation and glaucoma development remains contentious. Dayanir et al (2008) report that ocular hypertensive eyes (8/33) that developed frequency doubling technology

Clinical practice awaits a more complete understanding of the role of IOP fluctuation in glaucoma

ocular sure fluctuation

Dr Bang Bui
BOptom PhD

perimetry defects had greater visit-to-visit IOP fluctuation (5 vs. 2 mmHg) than those OHT eyes without field defects.

However, Bengtsson et al (2005) showed in treated ocular hypertensive patients (Mälmo Ocular Hypertension Study) over a period of 10 years that visit-to-visit (every three months) IOP fluctuation was not associated with increased risk of glaucoma development.

This is consistent with recent data from the European Glaucoma Prevention Study in ocular hypertensive patients, which reported no relationship between visit-to-visit IOP fluctuation and the risk of developing glaucoma (Miglior et al (2007).

Similarly, Medeiros et al (2008) report no association between IOP fluctuation and the risk of conversion to glaucoma in 256 eyes with untreated ocular hypertension. On average, the difference in IOP fluctuation between ocular hypertensive patients who developed glaucoma and those who did not was 0.4 mmHg.

Long-term IOP fluctuation and glaucoma progression

Although long-term IOP fluctuation seems to be less important as a risk factor for the development of glaucoma, it may play a larger role in glaucoma progression.

In particular, by reanalysing data from the Advanced Glaucoma Intervention Study Nouri-Mahdavi et al (2004) found that larger IOP variation from visit-to-visit had a stronger association with glaucoma progression than the mean IOP. Each 1 mm Hg increase in IOP fluctuation was associated with a 31 per cent increase in the risk of visual field progression.

Lee et al (2007) in a retrospective study of patient charts found a strong association between long-term IOP fluctuation and visual field progression. Likewise, in a review of patients who had undergone glaucoma surgery, those with larger long-term IOP fluctuation (> 2 mmHg) were at greater risk of visual field progression (Hong et al (2007).

Collaer et al (2005) investigated sequential office measurements over a single day and found that in patients with normal-tension glaucoma there was a significant relationship between visual field deterioration and the range and peak of IOP.

More recently the authors of the Early Manifest Glaucoma Trial (Bengtsson et al 2007; Leske et al 2007) report that visit-to-visit IOP fluctuation was not an independent risk factor for visual field progression, whereas mean IOP was significantly correlated over the eight years of data analysed. This was found to be the case for both treated and untreated patients.

Bengtsson et al (2007) suggest that one reason for the difference between the EMGT and AGIS outcomes may reflect the IOP measurements included in the analysis. In particular, the EMGT data include only IOP measurements taken up to the date of progression, whereas the AGIS analysis included measurements taken after progression when treatments were changed, which could lead to greater IOP reduction and thus larger IOP variability.

A recent reanalysis of the AGIS data by Caprioli et al (2008) to only include IOP measurements up to visual field progression showed that IOP fluctuation was associated with visual field progression only in those with low mean IOP and not those with high mean IOP.

These data raise the possibility of a 'higher-than-endurable' IOP (Orzalesi et al 2008) or pressure variation that invade a 'damage zone' (Caprioli and Coleman 2008) that is likely to vary between individuals.

Ocular perfusion pressure fluctuation

While the role of IOP fluctuation remains to be more fully assessed, the idea of fluctuations in and out of a damage zone might be pertinent to ocular perfusion pressure (OPP, difference between blood pressure and IOP), which describes the force that drives blood into the ocular tissues, and like IOP, OPP following a circadian rhythm.

Importantly, in some individuals diurnal IOP peaks can coincide with blood pressure troughs and, together with postural increases in IOP, can lead to a substantial reduction in OPP (Liu et al 2003).

Previous studies provide evidence that those with low OPP are at greater risk of glaucoma (Anderson 2003; Sehi et al 2005; Tielsch et al 1995). Indeed, reductions in diastolic blood pressure have been correlated with visual field progression in normal tension glaucoma, particularly in patients over treated for systemic hypertension (Choi et al 2006; Choi et al 2007).

Taken together, at present there is no compelling evidence that IOP fluctuation, whether short- or long-term, is useful in predicting those who will develop glaucoma or show visual field deterioration. While it is important to question our current thinking about IOP measurement and concepts of target pressure, changes in clinical practice guidelines await a more complete understanding of the role of IOP fluctuation in glaucoma.

References available on request from the author, email bvb@unimelb.edu.au.

A sceptic and his POAG

Case report

Associate Professor Richard Vojlay
BScOptom DipOcTherap DipHumanities(Music)

DB, a 55-year-old male, presented for an examination in April 2008. For a few years he had been satisfied wearing ready-made spectacles. He had no problems with his distance vision and no other significant symptoms.

This was his first eye examination although I had been seeing his family for many years. His wife, who has been a patient of mine for 24 years, made an appointment for DB after a casual discussion about his eye health. DB was a diabetic suspect, hypercholesterolaemic and a well-controlled hypertensive yet had no significant ocular history.

Examination results:

Vision: R 6/8 L 6/6-2

Refraction: R -0.25 VA: 6/5-2, L -0.25/ -0.50x75 VA: 6/5-, Add: +2.25 (N4)

Slitlamp: deep anterior chambers R + L

Gonioscopy: angles open 360 deg R + L

Dilated pupil fundus examinations: 90 D: C/D R 0.5, L 0.8.

The right disc was normal and passed the ISNT rule; the left disc had an inferior notch and the ISNT rule was not respected (Figures 1 and 2).

The right nerve fibre layer was normal. There was an inferior nerve fibre layer defect in the left eye (Figures 3 and 4).

There was some pigmentary irregularity surrounding the maculae, L > R.

BIO: No other abnormality in either fundii.

Tonometry:

R 16 mmHg L 18 mmHg (Perkins 17.04.08: 6.45 pm)

R 20 mmHg L 21 mmHg (Perkins 07.05.08: 1.25 pm)

R 21 mmHg L 24 mmHg (Perkins 21.05.08: 4.10 pm)

Pachymetry: R 537 µm L 521 µm

Medmont perimetry: (glaucoma threshold) R early inferior nasal step, L substantial superior arcuate defect (Figures 5 and 6).

DB was advised that he had open-angle glaucoma and was referred to an ophthalmologist for initial management and treatment.

The ophthalmologist noted a small rim of beta peripapillary atrophy on the left disc and otherwise confirmed the clinical findings that were recorded. SWAP perimetry confirmed the small early nasal step in the right eye and the superior arcuate defect in the left eye.

Figure 1. Right optic nerve, normal

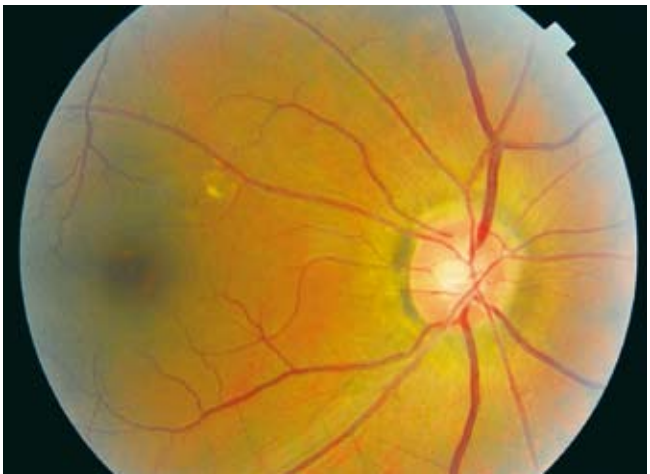


Figure 2. Left optic nerve showing inferior notch

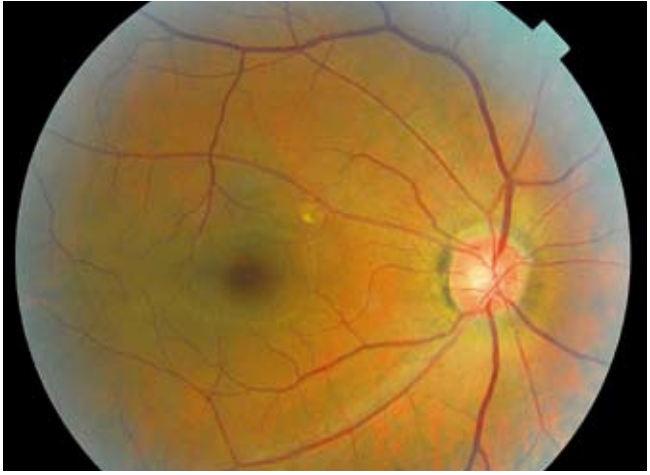


Figure 3. Right fundus showing normal nerve fibre layer

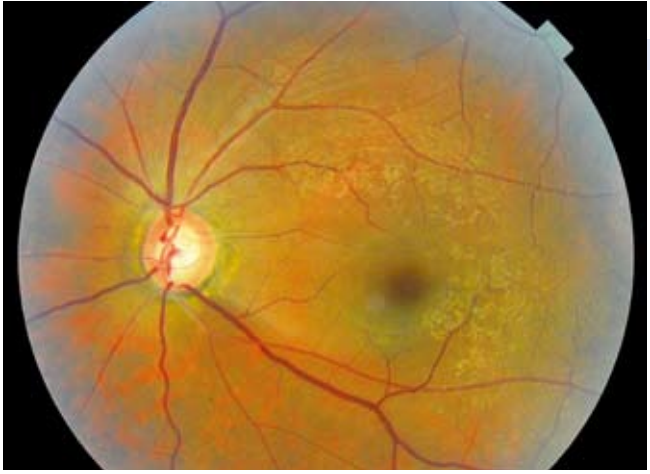


Figure 4. Left fundus showing inferior nerve fibre layer defect

According to results from the Heidelberg Retinal Tomography (HRT), the right disc was normal but the inferior sector of the left disc was abnormal and several other sectors were borderline (Figure 7).

The patient was advised that he had primary open-angle glaucoma and prescribed with the treatment of Lumigan nocte for both eyes. Three weeks after commencing treatment his IOP was 13 mmHg in both his right and left eye.

The significant reduction in his IOP was pleasing and the patient was scheduled for repeat visual fields and IOP measurements in one month.

This case highlights the need to be assertive in the face of a sceptical public that at times feels that a comprehensive eye examination, including dilation and retinal photography, may be unnecessary.

I frequently mention this case to my patients to reinforce the importance of regular reviews even when there are no obvious symptoms. ■

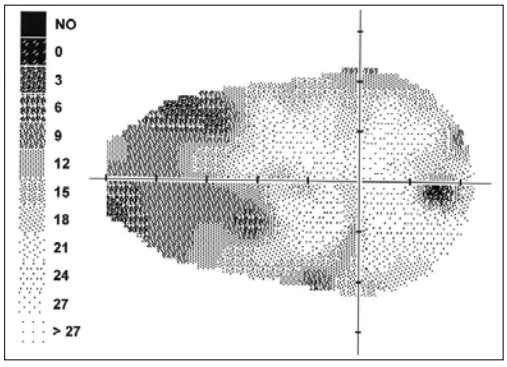


Figure 5. Medmont visual field, right eye: glaucoma threshold—early inferior nasal step

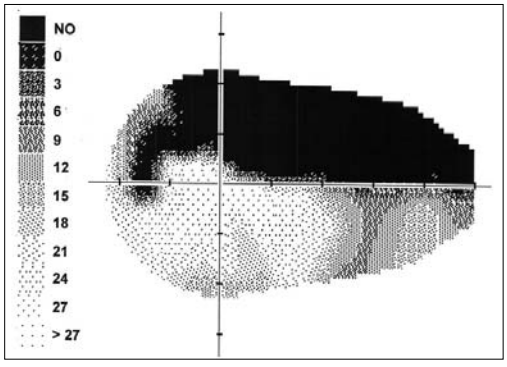


Figure 6. Medmont visual field, left eye: glaucoma threshold—substantial superior arcuate defect

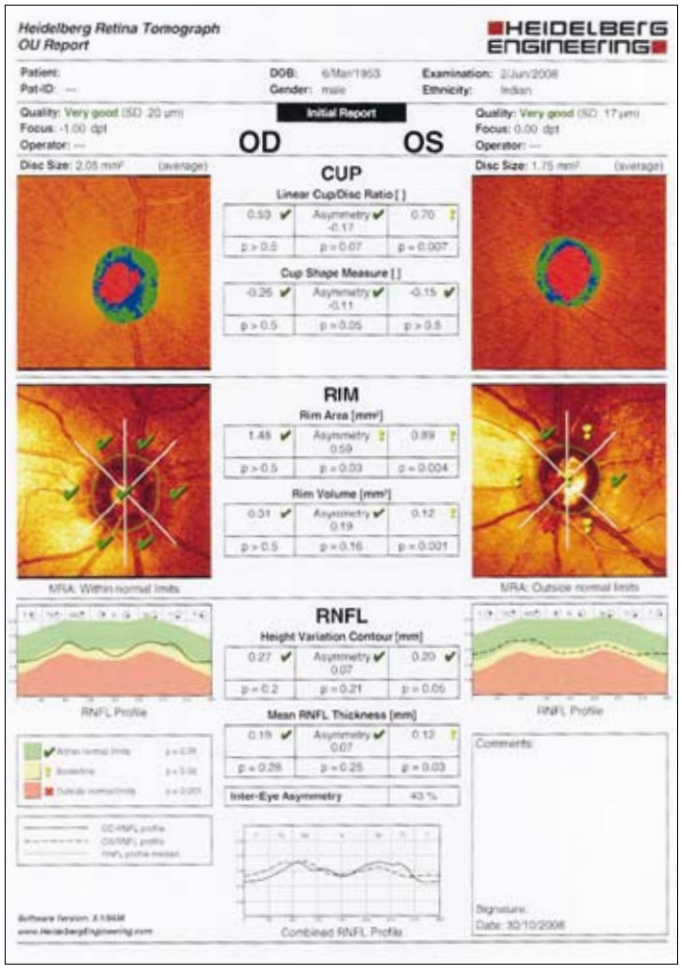


Figure 7. HRT, right eye: normal, left eye, inferior sector: abnormal, other sectors: borderline

PBS: we're in

The association is tenacious as it strives to have more drugs available on the PBS when prescribed by optometrists so patients can benefit from more convenient and lower cost eye care. **GARY OSHRY** reports.

The inclusion of almost the entire list of anti-glaucoma medicines on the PBS when prescribed by optometrists is still under consideration by the Health Minister after the recommendation was put forward by the Pharmaceutical Benefits Advisory Committee (PBAC) but, after seven years of negotiations, the Department of Health and Ageing has finally agreed to include the common anti-inflammatory fluorometholone acetate on the PBS when prescribed by optometrists.

Patients who will benefit most from the inclusion of drugs on the PBS schedule when prescribed by optometrists are in rural and remote areas, where ophthalmic care may not be readily accessible, and the elderly, who are more likely to suffer conditions requiring ocular medicines. Attending another practitioner simply to obtain a PBS benefit may be a financial imposition and require unnecessary effort.

Optometrists play an important role in the management of glaucoma and eye care in general, says the national executive director of Optometrists Association, Joe Chakman. In more than eight years of prescribing ocular medications, optometrists have never exceeded the bounds of accepted professional limits. Complaints about optometrists' clinical and professional behaviour are extremely rare.

'We have been negotiating for PBS benefits almost since therapeutic legislation went through in Victoria. We briefed health ministers on many occasions and have seen countless people in the health department and made many submissions,' says Chakman.

In its most recent submission to the PBAC the association reiterated that without PBS benefits for many optometrists' prescriptions, the pressure remains for optometrists to refer patients to medical practitioners for consultations that serve no purpose other than for the patient to obtain a PBS benefit.

As a result of the inclusion of fluorometholone acetate and if the PBAC recommendation for inclusions of the majority of glaucoma drugs is approved, the cost to the PBS will remain the same as no additional prescriptions will be dispensed but there will be lower Medicare expenditure as additional consultations with medical practitioners will be unnecessary.

Fluorometholone acetate

About one in every nine prescriptions written by optometrists is for fluorometholone acetate (flarex)-, making it one of the most common medicines prescribed by optometrists in Australia. Fluorometholone in its alcohol form is listed for prescription by optometrists under the PBS but, until now, fluorometholone with an acetate base was not.

The inclusion of flarex on the PBS schedule for optometrists will be of most benefit to people in lower socio-economic groups who pay a much lower fee for the medication when it attracts a PBS subsidy. The PBS cost of the drug to non-concession patients is similar to the non-PBS cost.

In its submission the association proposed that flarex was less likely to cause side-effects in optometric practice because optometrists used this medicine only in short-term therapy.

The risk of optometrists inappropriately prescribing flarex in an active herpetic ulcer, fungal infection or when there is a danger of increasing IOP is minimal as they use routinely high magnification slitlamp biomicroscopy to examine the eye and have the necessary expertise to diagnose anterior eye disease.

Anti-glaucoma medicines

If the PBAC proposal is accepted, the inclusion on the PBS of anti-glaucoma medications written by optometrists will be significant as these drugs are probably

for the long haul

more important to the community than the PBS benefits for any other class of medicines prescribed by optometrists. Anti-glaucoma medications are expensive and must be taken regularly for the remainder of the patient's life so the cumulative cost to the patient is very high.

The majority of glaucoma prescriptions written by optometrists are written in rural and outer metropolitan areas where ophthalmic care may not be readily accessible, according to a survey conducted by Optometrists Association.

In the past optometrists often felt uncomfortable referring to GPs for a prescription those patients who wanted to obtain PBS benefits for their glaucoma medications, because GPs usually lacked the instrumentation and experience necessary to adequately care for patients with glaucoma.

The PBAC advised the association in September 2006 that in principle it supported the inclusion of the anti-glaucoma products, provided that an adequate shared-care responsibility model was developed. The PBAC sought further clarification of the comanagement arrangement between optometrists and ophthalmologists within the PBS context.

'Since 2006, New South Wales, South Australia, the ACT and the Northern Territory have amended legislation to permit optometrists to prescribe anti-glaucoma medications, in line with legislation that existed in Victoria,' says Chakman. 'We put to the PBAC that PBS should be available under the rules that apply in the jurisdiction in which they are prescribed.'

Almost the entire list of approved glaucoma drugs are under consideration to be accepted on the PBS schedule for optometrists subject to shared-care arrangements that promote quality use of medicine. The omissions from the proposed list include carbachol and dipivefrine, which have been dropped from the PBS, and apraclonidine, which is available on PBS only under limited circumstances.

According to Chakman, it is imperative that the majority of glaucoma drugs be accepted. 'Patients should not be discriminated against based on their type of glaucoma or other health conditions,' says Chakman. 'In addition, ophthalmologists don't want patients coming through for routine monitoring. It is expensive for the patient and ophthalmologists don't have the time.'

Where to from here?

The states and territories have legislated to permit optometrists to prescribe topical ocular medicines because they believe this policy improves access to eye care, lowers costs to the government, lowers costs to patients and assists in achieving appropriate usage of available skills.

> **We have been negotiating for PBS benefits almost since therapeutic legislation went through in Victoria. We briefed health ministers on many occasions and have seen countless people in the health department and made many submissions.**
>
> Joe Chakman

The absence of PBS benefits when optometrists prescribe ocular medicines undermines these objectives by penalising patients who obtain a prescription from an optometrist. Chakman says that the association will continue making submissions to the PBAC until the list of drugs is complete.

'With each drug approved by the PBS when prescribed by optometrists, hopefully more optometrists will be encouraged to become therapeutically endorsed,' says Chakman. 'Optometrists are always looking to take on a greater role in the care of the patient.' ■

Three-pronged attack

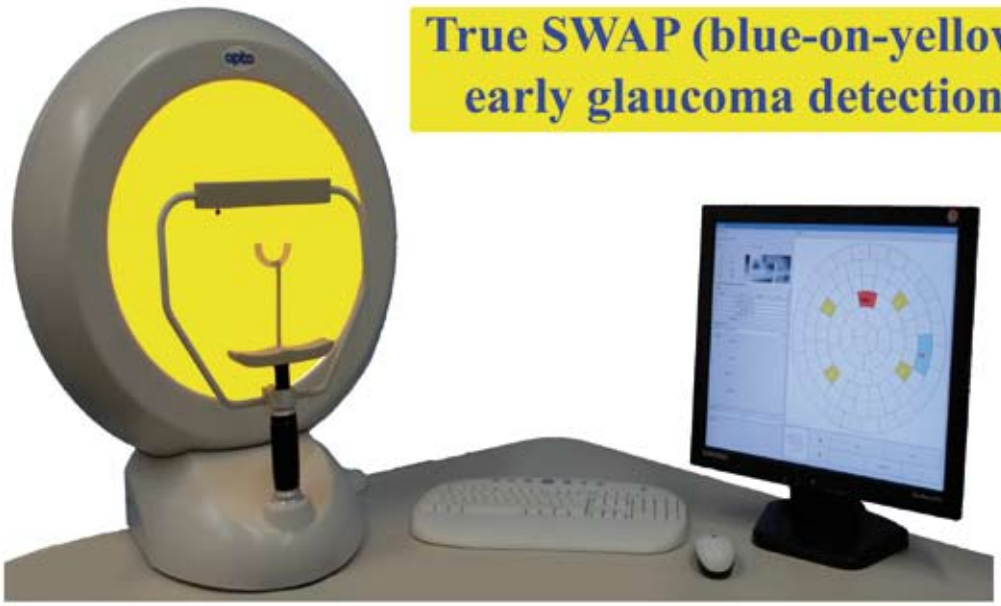
Lee Pepper
BOptom
Personal services manager
Optical Manufacturers

The management of glaucoma in mainstream optometric practice is based on a three-pronged attack: assessment of the posterior pole with emphasis on the optic nerve head, monitoring of the patient's visual fields and measurement of intraocular pressure (IOP). All three tests must be conducted at each visit to properly assess the patient's glaucoma status.

Digital retinal photography

All optometrists understand the importance of assessing the optic nerve head in every patient at risk of glaucoma. Historically, this was a difficult task for practitioners who made observations with direct and indirect ophthalmoscopy, and recorded their observations with drawings and estimates of sizes and ratios. Even the best manual recording may vary from optometrist to optometrist, or from visit to visit by the same optometrist.

Digital retinal photography has been a godsend for nerve head assessment and monitoring. A high-resolution image is a quantum leap over previous attempts to record the optic disc. The exact size and location of the edge of the cup, nerve head vasculature, colour and so on are clearly visible in a digital image, which can be saved for future comparison. The software



in some retinal cameras will allow two images taken on different occasions to be viewed side by side, or superimposed to identify fine changes. Stereoscopic analysis is also possible by the latest cameras using this software.

Perimetry

Visual fields have been easier to compare since the development of automated perimeters. Dark cloaks with Bjerrum targets and tangent screens were replaced by more reliable perimeters that quantified the results, which could be printed and compared from visit to visit.

Newer software eliminates the necessity to compare point by point decibel values, allowing the software to mathematically compare two or more consecutive fields. The practitioner can select the field loss sensitivity and present the results in various formats such as grey scale or numeric, or as a bebie curve. White-on-white perimetry has been supplemented by techniques more specific to detecting early glaucoma, such as blue-on-yellow (SWAP) perimetry.

Tonometry/pachymetry

IOP is still an important determinant of glaucoma risk but now that corneal rigidity is acknowledged as a major factor in determining pressure, the use of tonometry should be qualified in the context of pachymetry. Tonometry and pachymetry can be performed separately on different instruments or by four-in-one instruments that combine them with autorefraction and keratometry.

Although the correlation of corneal thickness with corneal rigidity, and in turn with IOP, is still the subject of much research, we now have a better idea of true IOP.

With these three techniques, we can be more confident of knowing when to refer and when to keep monitoring a glaucoma suspect. ■

What's going on here?

Clinical conundrum

Jeffrey Chibert
BScOptom PGD OcTher FVCO

BM, a 57-year-old male, was diagnosed in Russia with primary open angle glaucoma (POAG) in 2005. He was prescribed Betoptic and Azopt twice daily in the right eye.

In 2008 BM presented for examination in Australia.

Examination results:
R 25 mmHg L 20 mmHg
Corrected VA: 6/6 R + L
C/D R 0.7, C/D L 0.6
Neural-retinal rim thin temporally in both eyes
Pseudoexfoliation (PXF) in both eyes
Perimetry: R inferior extension of blind spot, L superior paracentral loss
Diagnosis: PXF glaucoma R + L

After trialling several different drops, BM began Azopt in both eyes twice daily and Travoprost in both eyes once a day. The treatment was able to control his IOP to R 16 mmHg L 14 mmHg.

The patient went back to Russia for three weeks. On his return he began complaining of bilateral blurred vision. The condition had been occurring for a few weeks.

Examination results from follow-up visit:
R 28 mmHg L 18 mmHg
VA: 6/9 R + L
Pupils dilated and fixed

What is a possible explanation for this patient's presentation?

The answer is on page 36

VFI regression trend analysis

When following glaucoma progression using visual fields, distinguishing true progression from normal variation can be difficult. Software used in the Humphrey Field Analyser known as Guided Progression Analysis (GPA) overcomes this problem by comparing patient tests to a database of stable glaucoma patients. Each of the comparative patient tests on the database is conducted once a week for four weeks to quantify the actual normal variation.

GPA then compares each field in a series to a pair of baseline fields and highlights points that have progressed significantly more than normal variation. The progression criteria are based on the Early Manifest Glaucoma Trial and, to avoid the confounding effects of advancing cataract and cataract surgery, the analysis is based on the Pattern Deviation Plot.

A new index called Visual Field Index (VFI) has been added to GPA to look at the rate of progression using regression trend analysis. Based on an intuitive percentage scale and with low sensitivity to cataract, VFI is weighted more centrally to better reflect ganglion cell loss and better represent advancing glaucoma.

Results are graphed against age and extrapolated five years into the future so that the practitioner can take current rate of progression and the patients' life expectancy into account when making treatment decisions.

The data can be used to educate patients on treatment compliance and to monitor the change in the rate of progression following the initiation of a new treatment regime. ■

Stick to it

Practitioners have a responsibility to ensure that patients understand the importance of using their medication as directed, especially in the early stages of treatment. **MATT TROLLOPE** investigates.

Two recent studies have indicated that patients do not always persist with their glaucoma medication regimes.

This news may surprise many people, given glaucoma affects a patient for life and medication is the only way for them to stave off permanent vision loss. With all glaucoma patients made aware of the disease's potential progression and informed by their specialist about the importance of adhering to their medication regime, it may be hard to understand why patients do not administer their drops as required to preserve their vision.

Rait and Adena, authors of a study published in the American journal *Ophthalmology*, hypothesised several reasons for a glaucoma patient not adhering to their medication plan.

These include the patient ignoring symptoms, refusing to accept that failing to take the medication would result in vision loss, being unable to afford the eye drops or not persisting while travelling.

Dr Lance Liu of Melbourne's Preston Eye Clinic suggests persistence could decrease because patients are forgetful, lack dexterity when it comes to applying drops or find the dosing regimen inconvenient.

He is not surprised that many patients fail to adhere to a management regimen.

'Unfortunately, glaucoma is an asymptomatic disease and unless the patient has lost vision, taking drops for something they're not aware of and that does not seem to improve any symptoms is a hard thing to do,' he said.

'It's the same with conditions such as high blood pressure. The only time that people realise they've got a problem is when they get their eye pressure measured.'

Friedman, Hahn, Gelb, Tan, Shah, Kim, Zimmerman and Quigley, authors of a study published in *Clinical and Experimental Ophthalmology*, proposed that non-compliance could occur due to other factors, such as the medication causing adverse side-effects, or the patient having a shorter time-frame in which their drops needed to be resupplied.

Working out the percentage of patients who do not adhere to their drop regimen is an inexact science. Dr Liu estimates that between 10 and 15 per cent of his patients have forgotten to take their drops at some stage.

According to Rait and Adena, patient persistency declined by half between three and 12 months after starting supply.

Melbourne-based glaucoma specialist Dr Mark Walland said that in his experience, persistency rates are not that low.

'I would not say the rate is as high as 50 per cent, but I think this mostly relates to poor patient education,' he said.

Poor education would mean that patients cannot always be blamed for their low adherence.

Both studies suggest that at times it is the doctor who is responsible for the patient's habits. Rait and Adena indicated patient persistency could decrease if patients failed to receive a reminder to visit their specialist, who would reinforce the importance of persisting.

Dr Walland says patients need a firm and clear explanation from their doctor about why adhering to their medication regime is crucial.

'Simply saying "Here, use these drops" is an inadequate explanation,' he said.

'The discussion should be graded depending on the severity of the disease. In cases of ocular hypertension it is reasonable to describe taking the drops as an insurance policy for vision, while with severe glaucomatous loss it is appropriate to warn the patient that they are at risk of blindness if they don't persist, with greater firmness on patients deemed likely to be recalcitrant.

'Written material can also be provided, given we know that not everything that is said during a consultation is retained by the patient. Reinforcement is also needed during subsequent visits, and also feedback from the patient on the success or otherwise of treatment.'

Dr Walland says he is astounded whenever patients tell him that their previous doctor had never told them their intraocular pressure reading.

Rait and Adena suggested more 'educational efforts in the office' were needed to help doctors improve their communication skills and strategies to increase their patients' compliance rates.

Dr Liu agrees with this assessment. 'I think there are many factors that make the patient take their drops. It comes from the doctor, the literature and many other sources, such as the family and partners,' he said.

'I think there's always room for (doctor) improvement, the problem is whether it's practical for the doctor, because they always seem to be pushed for time. Some of the pharmaceutical companies are now sending out literature reinforcing the importance of taking their drops.'

Among these companies is Pfizer, which in June 2006 launched its glaucoma patient support program called Eye Comply.

Pfizer Australia's ophthalmology team leader, Brett Elliott, says that Eye Comply was developed because the literature on compliance in asymptomatic conditions, such as glaucoma, indicated that providing patients with ongoing support and educational resources for their disease was beneficial.

'Patients can learn a great deal about

their condition without coping with the stress of having just been diagnosed,' he said.

'They learn to take greater ownership of that condition when it comes to accepting the need for chronic treatment, and develop a better appreciation for the importance of remaining compliant with the therapy that has been prescribed by their doctor.'

Current evidence suggests that good compliance habits are developed in the early phases of treatment, and once a patient develops a routine for taking their medication, this behaviour is most likely to continue long-term.

Eye Comply aims to assist newly-diagnosed glaucoma patients in developing positive compliance habits from the commencement of therapy by addressing key reasons why patients can become poorly compliant, such as forgetfulness, confusion regarding dosing and an inability to correctly instil eye-drops.

Pfizer has also partnered with Glaucoma Australia, the leading patient advocacy group for glaucoma sufferers, to ensure newly-diagnosed glaucoma patients receive as much support as they require.

To be enrolled in Eye Comply, patients and doctors need to provide written consent and, once enrolled, patients receive a series of educational mailers over a 12-week period.

Mr Elliott says there has been a very strong response to the program in Australia.

The bulk of literature, doctor education and programs such as Pfizer's have focused on the reasons patients do not persist and how to combat this.

Friedman and colleagues went in a different direction, exploring whether patients were more likely to persist with certain types of eye-drops over others.

It was found that patient re-supply rates of prostaglandins and dorzolamide-timolol combination drops were the highest of all drops surveyed in the study, leading the authors to say that 'persistency should be taken into account when selecting the most appropriate eye-drop to treat glaucoma and ocular hypertension.'

Dr Walland says studies looking at persistence tend to assume that all medications are an equally-valid choice for prescription.

'Such studies cannot take account of and are not designed to look at the clinical rationale and indications for prescription of certain medications,' he said.

'In the case of glaucoma drops, questions need to be asked. Is the medication first line monotherapy? Is it adjunctive treatment in a complex treatment protocol? Is the medication a switch or additional agent in the face of worsening disease? Is the addition of a medication necessary, given we also know that compliance declines significantly once more than two bottles of drops are required per day?

'Drug choices are ultimately made on the two issues that influence persistence: efficacy and tolerability.'

How do doctors go about ensuring that patients adhere to their medication regime, once the most suitable medication for their condition is selected after considering all compliance factors?

Dr Liu says each patient is different. 'Some patients say "Oh, I guess I'll get around to it" while others will be really conscientious and do everything,' he said.

'You can talk to some patients until you're blue in the face, and you can give them more information, but as the old proverb goes, you can lead a horse to water but you can't make it drink.'

Dr Liu says doctors themselves had to demonstrate persistence when it comes to reinforcing this importance.

'When a patient is diagnosed with glaucoma, it can take three or four visits to their specialist for the patient to accept the fact that taking medication is what they have to do for the rest of their lives.' ■

Current evidence suggests that good compliance habits are developed in the early phases of treatment, and once a patient develops a routine for taking their medication, this behaviour is most likely to continue long-term.

Brett Elliott, Pfizer

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Family matters

The diagnosis of glaucoma is often missed because of lack of history-taking or insufficient clinical examination.

This is the advice of three ophthalmologists who say that knowledge of primary open angle glaucoma (POAG) among the public and health professionals is poor. Their paper has been published in the *Medical Journal of Australia*.

Using the examples of two patients, the authors stress that eye-care practitioners should investigate a patient's family history of POAG, and that POAG patients should alert all first-degree relatives of their condition so that relatives receive regular and adequate glaucoma screening.

They say that measuring intraocular pressure (IOP) alone is an inadequate method of screening for POAG, and that examination of the optic disc is essential. The diagnosis of POAG was missed in both patients due to insufficient clinical examination.

One patient had his IOP monitored for four years by his brother–an optician–via non-contact tonometry because his parents had suffered glaucoma-related vision loss. His IOP never exceeded 22 mmHg during this time. On examination following referral from a general practitioner, open anterior chamber angles and a cupped myopic optic nerve head were discovered, and visual field testing revealed a large central defect in the right eye. Applanation tonometry showed elevated IOP in both eyes.

The second patient, whose mother and sister had gone blind from glaucoma, 25 years earlier had visited an ophthalmologist who detected no signs of glaucoma. The patient claimed that he had never been told of the importance of having his eyes examined regularly for glaucoma. On examination, it was discovered that he had significantly elevated IOP, completely cupped optic nerve heads and anterior chamber angles in both eyes. Visual field examination showed a complete lack of vision in his right eye and a temporal island of vision in his left eye.

The authors said that determining IOP remained an important aspect of screening for glaucoma, as the disease would have been detected in the second patient had his IOP been measured.

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Life goes on:

John Hunt
Gunnadah NSW

It came as a complete surprise to John Hunt when he was diagnosed with glaucoma. 'Glaucoma is a word I had heard but new very little about. It was a tremendous shock when I was diagnosed with it.'

Hunt is now 79 years old and has been retired for many years. Five years ago he visited his optometrist to request a higher power prescription after experiencing difficulty filling out bank forms. He had 70 per cent vision in one eye and 30 per cent in the other. After being examined and diagnosed with glaucoma by the optometrist, Hunt was referred to an ophthalmologist in Tamworth where the diagnosis was confirmed and treatment initiated.

Glaucoma has not had a major impact on Hunt's lifestyle although he says he is now more cautious when driving. The glare at night is a problem and he encounters difficulty with his vision when adjusting from darkness to brightness.

When Hunt learned that the disease was hereditary he immediately called everyone in his family to urge them to get regular eye examinations. 'I found out both my uncle and brother have had glaucoma for many years. If I had known they had it, I would have gone for a check-up earlier,' he says. 'Things may have turned out differently.'

Hunt drives to Tamworth, which is about 80 kilometres east of Gunnadah, three times a year to see his ophthalmologist. At each visit the doctor reinforces the importance of compliance when administering eye drops. 'I use Xalatan once a day, one drop in each eye,' says Hunt. 'It was difficult at first so my wife used to put them in for me. Now I am used to it.'

Over the past five years Hunt's condition has stabilised.

Iain Craig
Coffs Harbour NSW

Iain Craig, a 57-year-old draftsman, is grateful he wears spectacles. If it were not for the regular eye examinations by his optometrist Alan Burrow, he is convinced the glaucoma would have gone undetected for many years. 'Glaucoma is not affecting my eyesight,' says Craig. 'In fact, the most severe symptoms of the disease presented in my good eye.'

His IOP was normal but the cupping was a concern so Craig was referred to an ophthalmologist.

The ophthalmologist did not initially prescribe medication, preferring to monitor Craig's progress for a few months. After six months, tests confirmed that his cupping had worsened and IOP was increasing.

'The doctor said that I was a prime candidate for glaucoma but I still went for another opinion to make absolutely sure,' says Craig. The second diagnosis confirmed his fears.

Craig visits Burrow every 12 months to have his glaucoma monitored. He uses Azopt and Lumigan but he sometimes finds it hard to adjust to the medication. He says using drops makes his eyes sore and itchy, especially when it is windy or when he is working on the computer for extended periods.

'There is a lack of awareness about glaucoma in the wider community,' says Craig. 'I had seen the signs in the practice a hundred times but had never taken any notice.'

patients' perspective

'Glaucoma is a word I had heard but knew very little about. It was a tremendous shock when I was diagnosed with it.'

Gloria Sills
Gunnadah NSW

Sills was 60 years old and working as an accountant in the tranquil town of Gunnadah in the heart of the Namoi Valley, NSW, when a consultation with a visiting optometrist changed her life forever. Not only did she have early signs of cataracts, she also had glaucoma.

She was referred to an ophthalmologist in Sydney. She says her eyesight was too important to risk seeing anyone but the best and as her daughter was a student in Sydney she had somewhere to stay.

Fifteen years later, Sills still takes the train to Sydney two or three times a year for a consultation with her ophthalmologist. As she is a pensioner, flying is beyond her means. Her daughter relocated to Brisbane so Sills now stays in a hostel or nursing home when in Sydney.

The ophthalmologist operated on her cataracts and glaucoma at the same time. The glaucoma surgery was not successful and her peripheral vision is very poor. She has changed ophthalmologists in Sydney several times.

Sills is strict about compliance. She administers three doses of the drop in the morning and again at night. 'In the morning putting in drops is fine although time-consuming; I need to wait 10 minutes between each drop,' she says. 'Evenings are a hassle because I am usually going out and it's inconvenient. I see my optometrist Tim Duffy on a regular basis to get my pressure reading and to make sure everything is on track. He is very prudent and I am confident of his ability.'

For Sill, living with glaucoma means living in fear. Her independence is paramount. She lives by herself and relies on her car to get around. 'I once had breast cancer,' she says. 'I'd prefer to have my cancer back than go blind. As long as I can see the sky and horizon beyond, I'll be OK.'

Her 41-year-old daughter has been diagnosed with glaucoma.

Kaye Power
Traralgon VIC

Kaye Power remembers vividly the moment 15 year ago that she realised something was wrong. She says, 'I woke up one morning and turned to my husband and asked, "Do you see ash floating in the air?"'

Power says she had 'felt the build-up of fluid' in her forehead but thought the discomfort did not warrant the need for a check-up.

The optometrist diagnosed glaucoma after detecting an IOP of 49 mmHg in her left eye; her right eye was normal. She was referred to an ophthalmologist who initiated treatment.

Power attributes the onset of the disease to stress. At the time she and her husband operated a motel in the Latrobe Valley which went 'belly up' as the economy was in crisis.

Power says a 'concentrated eye-drop' was prescribed to lower her IOP to a manageable range of between 16 and 20 mmHg. Once her condition stabilised she was put on a milder dose.

Five years after being diagnosed with glaucoma, Power found out she had cataracts. She says that the surgery that followed left her with corneal scarring. Power is now 77 years old and is unable to use a computer or read a newspaper, and can read only books in large print.

John Newton
Coffs Harbour NSW

John Newton, 73, had type 2 diabetes when he was diagnosed with glaucoma. Eight years ago he was driving up the coast when his vision became distorted. 'Every tail light looked like that of a Ferrari, a circle with a block dot,' he says.

His IOP was normal at between 12 and 13 mmHg. He was prescribed Tenopt and Xalatan which stabilised the glaucoma. After eight years the ophthalmologist changed his medication to Azopt morning and night and Xalacom.

'I have a system for compliance,' says Newton. 'Every morning when I wake up I go to the "you know where", take a shower and pop the pills in a tray beside my bed; then I take my drops.

'Every evening before bed I have a glass of water from the fridge and take out the Xalatan. These new drops don't need to be refrigerated, which has thrown me off my routine completely.

Santo Mercuri
Kew VIC

Santo Mercuri was diagnosed with glaucoma prior to having his cataracts removed in 2005. He was not prescribed eye-drops for glaucoma but was having regular follow-up appointments with his ophthalmologist who was monitoring his eyes post-cataract surgery.

Last year, Mercuri cancelled one of the appointments to attend to other more urgent health matters. Neither the ophthalmologist's office nor Mercuri's daughter, who attends his medical consultations, followed up the cancelled appointment.

Earlier this year, Mercuri was having difficulty seeing and thought he needed new spectacles. The optometrist detected that the glaucoma had progressed and referred him to an ophthalmologist. 'The optometrist said that these days there is no need for elderly people to lose eyesight but because my father never complained, we didn't do anything about it. Now he has lost vision in one eye,' Mercuri's daughter says.

Mercuri, who is 94 years old, says his glaucoma has not affected his lifestyle dramatically. 'I don't work in the garden any more because I'm not feeling well, not because of my eyesight,' he says.

Medical treatment

Dr Catherine Green
MBChB, FRANZCO, MMedSc

While glaucoma nerve damage may not be only the result of elevated intraocular pressure (IOP), lowering of IOP is currently the only proven method of treatment of glaucoma that prevents progression of the disease.[1,2] **The normal range of IOP is 10-20 mmHg and the main aim of treatment is to lower IOP to a level at which no further visual field loss occurs. This level, referred to as target IOP, should be assessed individually for each patient.**

In many patients, simply lowering the IOP to a level below 20 mmHg will not be sufficient to prevent further visual field loss. Patients at high risk of progression or those with advanced disease will require IOPs at the lower end of the normal IOP range, that is, closer to 10 mmHg. In addition, controlling IOP in patients with high risk ocular hypertension may delay the onset of early glaucomatous damage.[3]

Each patient should be followed long term with regular evaluation of IOP, disc appearance and field-testing to detect progressive glaucomatous damage. When setting the target IOP the following factors should be taken into account:[4]

- IOP level at which damage occurred
- extent of damage and rate of progression
- central corneal thickness
- patient age and expected lifespan
- family history
- race
- cost and risk of treatment (or lack of treatment).

The treatment regimen should be reviewed at each visit and adjusted if the disease is progressing or if the patient is experiencing adverse effects. When therapy is being chosen for a patient, the following factors should be considered:

- drug efficacy
- side-effects of the treatment
- inconvenience of administration
- compliance of the patient with the treatment
- cost.

The ideal treatment is one that achieves the target pressure and arrests the progression of visual deterioration with minimum side-effects and at low cost. In most patients, medical therapy is used as the initial treatment, either as monotherapy or in combination. The choice of treatment may vary during the course of the patient's disease.

Glaucoma drugs reduce IOP by causing a decrease in aqueous production or by increasing outflow of aqueous from the eye. Aqueous drainage occurs via two routes, the trabecular meshwork and uveoscleral outflow.

Drugs that act on outflow may influence one or both of the two mechanisms.

Drugs may be administered topically in the form of eye-drops, or systemically.

Principles of medical therapy

The goal of treatment is to achieve the desired therapeutic response with the least amount of medication, inconvenience and side-effects. In most patients, unless there is a contraindication, treatment is initiated with a prostaglandin analogue. Advantages of these agents include good efficacy and high tolerability with few, if any systemic side-effects. The patient should be reviewed after several weeks of treatment and the response to treatment measured.

If the target pressure has been achieved and the patient is tolerating the medication, the treatment is continued. If there has been an inadequate response to the first line agent, the medication should be switched to another agent rather than adding a second medication. In some patients, switching to one of the other prostaglandins may result in a satisfactory therapeutic response.

If the drug has achieved a satisfactory pressure lowering effect but the target IOP has not been reached, it is recommended that a second agent from another class be added. The choice of second line agent is determined by patient factors including medical conditions. This process is continued until the target IOP is reached. If the target is not achieved on maximum tolerated medical treatment–currently thought of as three agents–laser treatment or surgery should be considered.[5]

Compliance

Glaucoma is a chronic disease with few symptoms. The success of treatment is dependent on the patient taking his or her medication regularly. Studies have shown that compliance with glaucoma medications is considerably less than presumed by treating practitioners.[6] In patients who are progressing despite medical treatment, compliance should be reviewed prior to changing management.

Compliance can be improved by establishing good communication between the patient and treating practitioner and informing the patient about the disease and its management. The number of medications and frequency of administration should be kept to a minimum.

For those who require more than one agent, the introduction of fixed combination medications has been helpful in reducing the number of bottles the patient needs to use. The patient should be taught how to instil drops correctly and to allow an appropriate interval of at least five minutes between different drops to reduce the possibility of diluting the medication.

PROSTAGLADIN ANALOGUES

- **Latanoprost, travaprost, bimatoprost**

Mechanism of action: increase uveoscleral outflow, possibly due to relaxation of the ciliary muscle and creation of dilated spaces between ciliary muscle bundles.

There may also be alteration in the metabolism of the extracellular matrix that surrounds the ciliary muscle cells. Because uveoscleral flow is independent of the episcleral venous system, it is possible to obtain low IOPs (9-11 mmHg). Studies have demonstrated that these agents can lower IOP by 25-30 per cent. The three agents have comparable efficacy but some patients may respond better to one agent than another.[7,8] Latanoprost has fewer local side-effects than travaprost and bimatoprost.[8]

Dose: once daily, usually in the evening.

Side-effects: systemically well tolerated.

Common local effects include conjunctival hyperaemia, discomfort and blurred vision.

Prostaglandins mediate inflammation and may precipitate intraocular inflammation in predisposed eyes. They should be used with particular caution in pseudophakic or aphakic eyes in which the posterior capsule is not intact as macular oedema has been described in this setting. Exacerbation or reactivation of underlying herpetic disease has also been reported. Increased iris pigmentation may occur, probably due to increased production of melanin within iris melanosomes. This effect does not reverse on withdrawal of the drug but has not been associated with any other long-term sequelae. Patients with blue irides are least likely to be affected by the colour change; it is most noticeable in patients with hazel or green eyes. Increased eyelash growth and hyperpigmentation of periocular skin also occur.

BETA BLOCKERS

- **Timolol, betaxolol**

Mechanism of action: aqueous suppression.

Side-effects: contraindications include severe chronic obstructive airways disease, asthma, heart block, bradycardia and congestive cardiac failure. Patients without a prior history of these conditions may develop symptoms after commencing treatment. Other side-effects include lethargy, mood changes, erectile dysfunction and reduced libido.

The elderly are more at risk from side-effects. Patients may not associate their side-effects with the eye-drops they are using and should be asked specifically about any symptoms they may be experiencing. Betaxolol is a relatively cardioselective beta-1 blocking agent that may cause fewer bronchospastic side-effects but its efficacy is slightly reduced compared with non-selective beta blockers.

ALPHA ADRENERGIC AGONISTS

Mechanism of action: stimulate the alpha-2 receptors in the eye, resulting in reduction in aqueous production by the ciliary epithelium and increase in uveoscleral outflow.

- **Brimonidine**

Brimonidine is a highly selective alpha-2 agonist that exhibits favourable IOP lowering effects when compared with other agents including beta blockers. The recommended dosage is one drop twice daily, although some studies have found improved IOP control with three times daily dosage.[9]

Side-effects: dry mouth, headache, lethargy and drowsiness.

Brimonidine crosses the blood-brain barrier. It is contraindicated in infants as it may cause respiratory depression and apnoea and should be used with caution in children.[10] Its use may be limited by the development of allergic reactions to the drug. This was reported to be 11.5 per cent at one year in one study[11] but may be as high as 25.7 per cent with prolonged use.[12] Alphagan P contains brimonidine purite and has a reduced incidence of allergic conjunctivitis a reduction of 41 per cent.[13] This formulation is not routinely available in Australia but can be prescribed in special cases via the Special Access Scheme.

Brimonidine has a negligible effect on pulmonary function, heart rate or blood pressure and can be used safely in patients with contraindications to beta blockers.

Drug interactions: should not be used in patients on monoamine oxidase inhibitors and tricyclic antidepressants.

- **Apraclonidine**

Its long-term use in the treatment of POAG has been limited by a high incidence of local allergic reactions, and tachyphylaxis.

It remains a useful drug for use in the short-term (less than one month) and is widely used in the prevention of post-laser procedure (for example, Nd:YAG capsulotomy, peripheral iridotomy) pressure spikes. The recommended dosage is three times daily.

CHOLINERGIC AGONISTS

- **Pilocarpine, carbachol**

The use of pilocarpine has declined since the introduction of newer drugs but it remains an effective treatment and is relatively inexpensive. Carbachol and ecothiopentate are no longer used in the routine treatment of glaucoma in Australia and are not discussed here.

Mechanism of action: direct acting cholinergic agonist; reduces IOP by causing contraction of ciliary muscle. This results in tightening of the trabecular meshwork, increasing the outflow of the aqueous humour.

Formulation and dose: available in 1%, 2%, 3%, 4% and 6%; 2% and 4% are most commonly used. For an optimal therapeutic effect, the dosage is four times a day.

Pilocarpine gel and Ocuserts allow less frequent administration and reduce some of the side-effects but these formulations are not available in Australia.

Side-effects: miosis, brow ache, myopic shift. The miosis may interfere with vision in dim light, especially in patients with lens opacities. Retinal detachments can occur in susceptible patients, for example, high myopes, especially with the higher concentration preparations. Systemic side-effects are rare but include diarrhoea, abdominal cramps, increased salivation, bronchospasm and enuresis.

Prolonged use has been associated with subconjunctival fibroblast activation, which may affect the outcome of glaucoma filtering surgery. Permanent miosis may also occur with prolonged use; this may cause difficulties when the patient undergoes cataract surgery.

Continued page 32

Medical treatment

From page 31

CARBONIC ANHYDRASE INHIBITORS

- **Dorzolamide, brinzolamide**

Mechanism of action: inhibition of the enzyme carbonic anhydrase, resulting in decreased aqueous production.
Dose: dorzolamide: three times a day; can be dosed twice a day if used as an adjunctive treatment. Brinzolamide: twice daily.
Side-effects: Bitter taste after drop instillation. Ocular discomfort and allergy. These drugs may be associated with idiosyncratic bone marrow suppression and sulphonamide allergy.

FIXED COMBINATIONS

There are currently four preparations of fixed combination medications available for use in Australia. The advantages of fixed combinations include improved ease of dosing allowing improved compliance and less exposure to preservatives. It should be noted that they all contain timolol 0.5% and the treating practitioner and patient should be aware of the potential systemic effects of beta blockers.[14]

FIXED COMBINATIONS

Medication	Combination of timolol and
Xalacom	Latanoprost
DuoTrav	Travaprost
Cosopt	Dorzolamide
Combigan	Brimonidine

SYSTEMIC GLAUCOMA THERAPY

Systemic IOP lowering agents have significant systemic side-effects.

Their use is usually restricted to the management of acute, severe episodes of elevated IOP in patients whose glaucoma cannot be controlled by alternative therapy alone. They are often used in the short term to control the IOP prior to definitive management, for example, surgery. Cautious use is recommended, particularly in the elderly.

The carbonic anhydrase inhibitor acetazolamide is the most commonly used systemic agent. It may be given orally or intravenously in either a 250 mg or 500 mg dose.
Mechanism of action: reduced aqueous production by direct inhibition of carbonic anhydrase in the ciliary body.
Side-effects: usually dose related. Paraesthesia of fingers and toes, lassitude, anorexia, diarrhoea and abdominal discomfort are relatively common. May cause electrolyte imbalances, metabolic acidosis and precipitate renal stones. Although rare, aplastic anaemia, thrombocytopenia and agranulocytosis have been reported.

OSMOTIC AGENTS

- **Glycerol (orally), mannitol (intravenously)**

These agents are used to control acute episodes of elevated IOP in patients in whom acetazolamide has been ineffective or is contraindicated. They are used for short-term IOP lowering, for example, prior to surgery, and should be used with caution owing to their sever side-effects.

Mechanisms of action: lower IOP by increasing the blood osmolarity. This creates an osmotic gradient between the blood and vitreous humour, drawing water from the vitreous cavity and a reduction in IOP.

Side-effects: headaches, urinary retention, confusion, backache, acute congestive cardiac failure, myocardial infarction and renal failure. Subdural and subarachnoid haemorrhages have been reported.

The treatment of glaucoma patients can be complex and needs to be individualised. It is important that the aim of treatment (preserving visual function) be balanced with preserving the patient's quality of life. Today, effective and well-tolerated treatments are available in formulations that may enhance compliance with medical therapy.

It is vital that treating practitioners reassess each patient at every visit for possible side-effects and drug interactions. Patient safety is enhanced by good communication between patients, general practitioners, eye-care professionals and specialists.

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OCT essential to preserve vision

Case report

Randal Lamont
BScOptom

Patient name: Mrs EC, DOB 02.07.1951.

History: Patient presented complaining of dots in front of her L eye.

Ocular history: EC is myopic Rx R -7.75/-1.0 x 60 L -7.75/-1.50 x 40. She has worn soft contact lenses since the early 1980s.

General health: Mild hypertension. No treatment initiated, reviewed regularly.

Family history: Nil.

Examination: Dilated examination found a small haemorrhage on the inferior rim of the left disc. Visual acuities were 6/7.5 R & L. IOP was measured as R 18 mmHg L 17 mmHg. Pachymetry was R 534 µm L 524 µm.

Gonioscopy: showed both angles open 360 deg R + L.

Optic nerves: the right disc shows cupping 0.7 with some inferior notching. There is some peripillary atrophy evident. In the left eye the cup/disc ratio is 0.6. Small haemorrhage on the inferior rim of the left disc.

OCT: The first graph (OD) in each chart shows a marked inferior nerve fibre layer loss. There is a slight superior loss in both eyes. That last graph in each chart shows the marked assymmetry of the NFL between the eyes.

Visual fields: The right visual field shows a marked superior defect that follows the midline. In the left eye, shows a possible arcuate defect developing.

Patient was referred to an ophthalmologist who confirmed the findings, including intraocular pressures of R 18 mmHg L 18 mmHg.

The diagnosis of normal tension glaucoma was most likely with the possible differential diagnoses of:

- branch vein occlusion
- myopic disc degeneration
- glaucoma
- anterior ischemic optic neuropathy
- posterior vitreous detachment.

The diagnosis of normal tension glaucoma was made even though the field defect was not typical. The diagnosis was made by the disc appearance and the borderline pressures relative to pachymetry. The patient was also referred to his GP for a full general health check.

Treatment: Xalatan ou 1 drop at night.

With treatment the IOP went down to 14 mmHg ou but the patient did not tolerate the drug due to persistent soreness and injection. This in turn resulted in contact lens intolerance.

Continued page 34

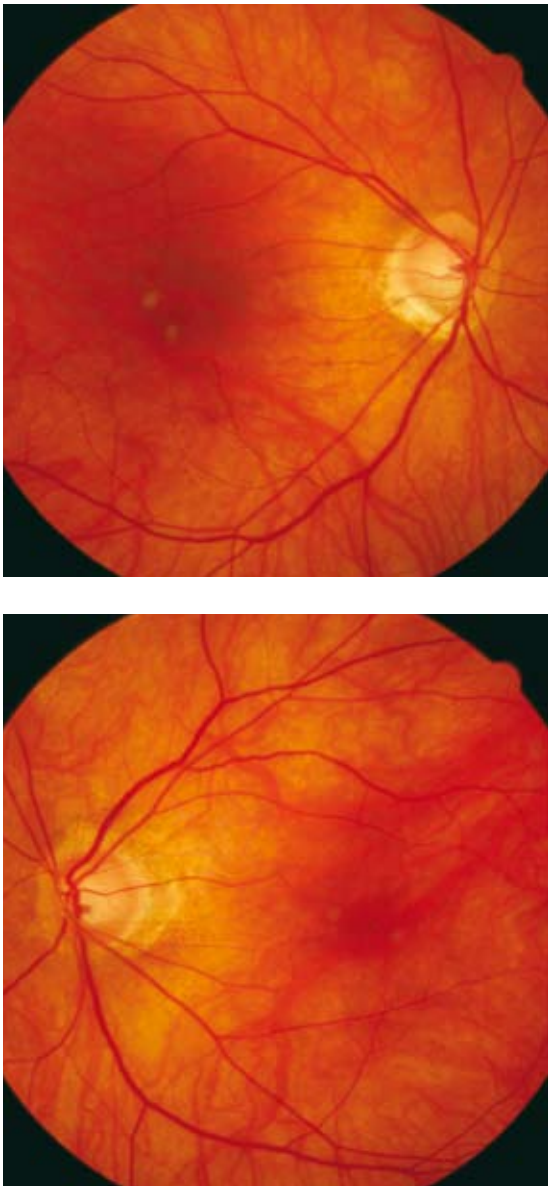


Figure 1. Right disc (top) shows cupping 0.7 with some inferior notching. There is some peripillary atrophy evident. The small haemorrhage has resolved. In the left eye the cup/disc ratio is 0.6.

OCT essential to preserve vision

From page 33

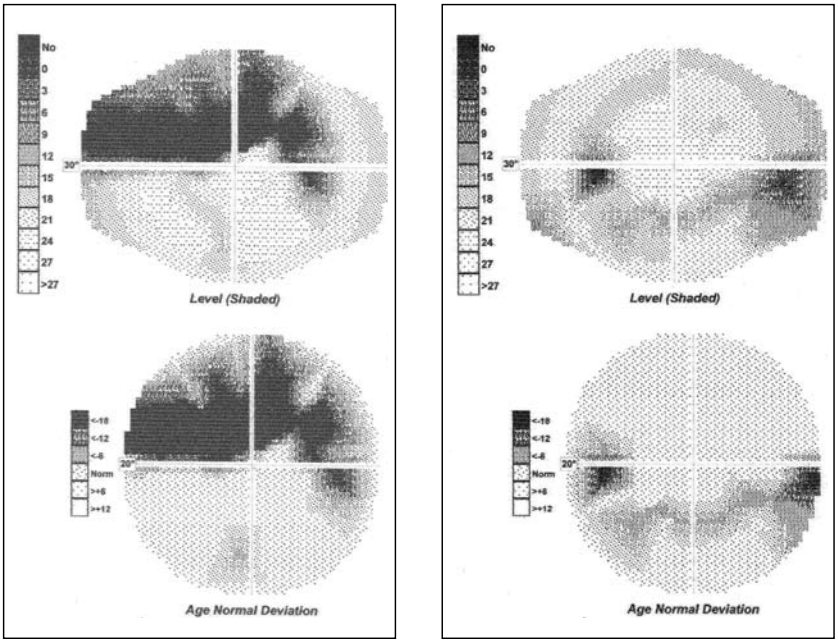
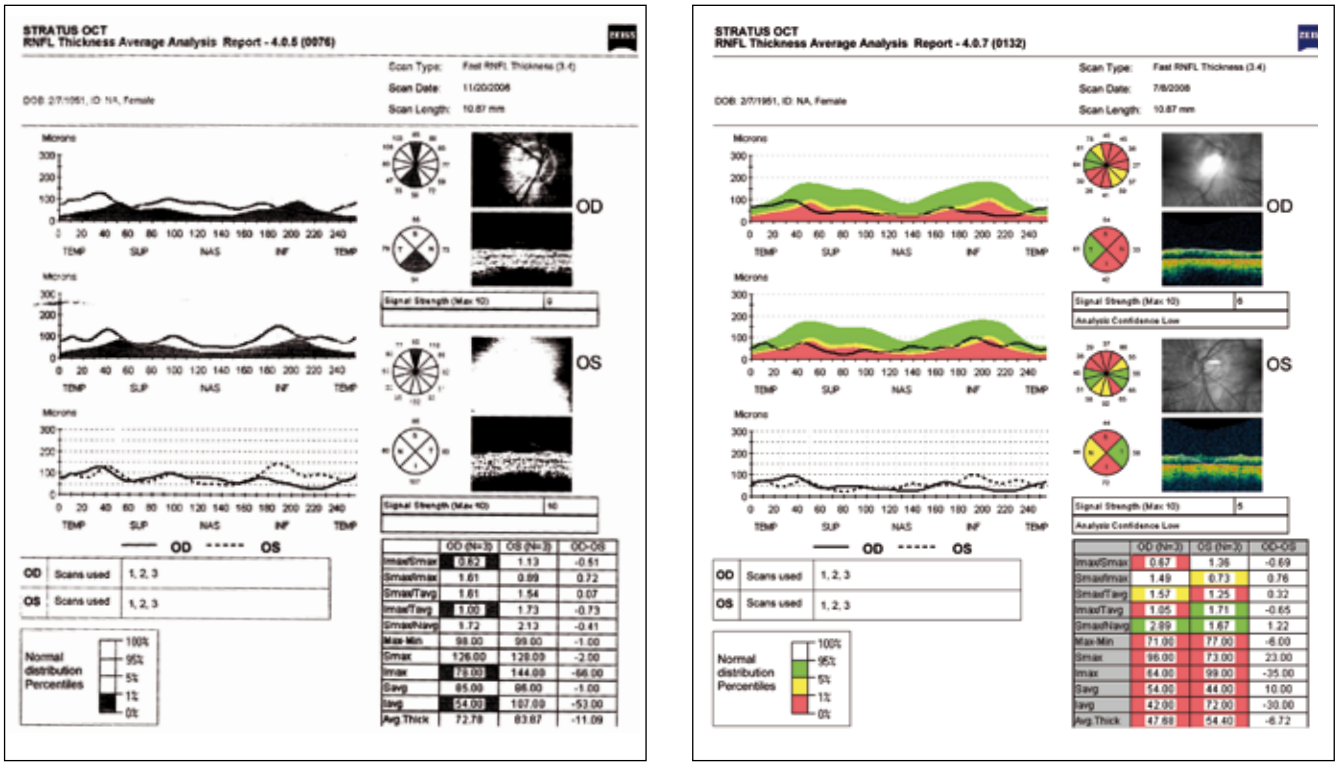


Figure 2. The right visual field (left) shows a marked superior defect that follows the midline. The left eye (right) shows a possible arcuate defect developing.



The first graph (OD) in each chart shows a marked inferior nerve fibre layer loss. There is a slight superior loss in both eyes. The last graph in each chart shows the marked assymmetry of the NFL between the eyes.

Subsequently she was trialled with Travatan but had similar problems. The patient stopped the drops for a while to wear the lenses (about six weeks) and her pressures increased to 19 mmHg R + L.

Patient was then prescribed Timoptol XE 0.5% 1 drop at night R + L. At her last review the pressures were R 11 mmHg L 14 mmHg. She has no problems with this drug and can still wear her contact lenses.

Visual fields are shown and these have not changed in the past few years. The R field does not show a typical glaucoma loss. The defect seems more an altitudinal defect which is more in line with an episode on non-arteritic anterior ischemic optic neuropathy.

The OCT results show a loss of the inferior nerve fibre layer in the right eye. The asymmetry shows up well on the bottom graph.

When viewing the table in the OCT results the bottom three rows are important as they are:

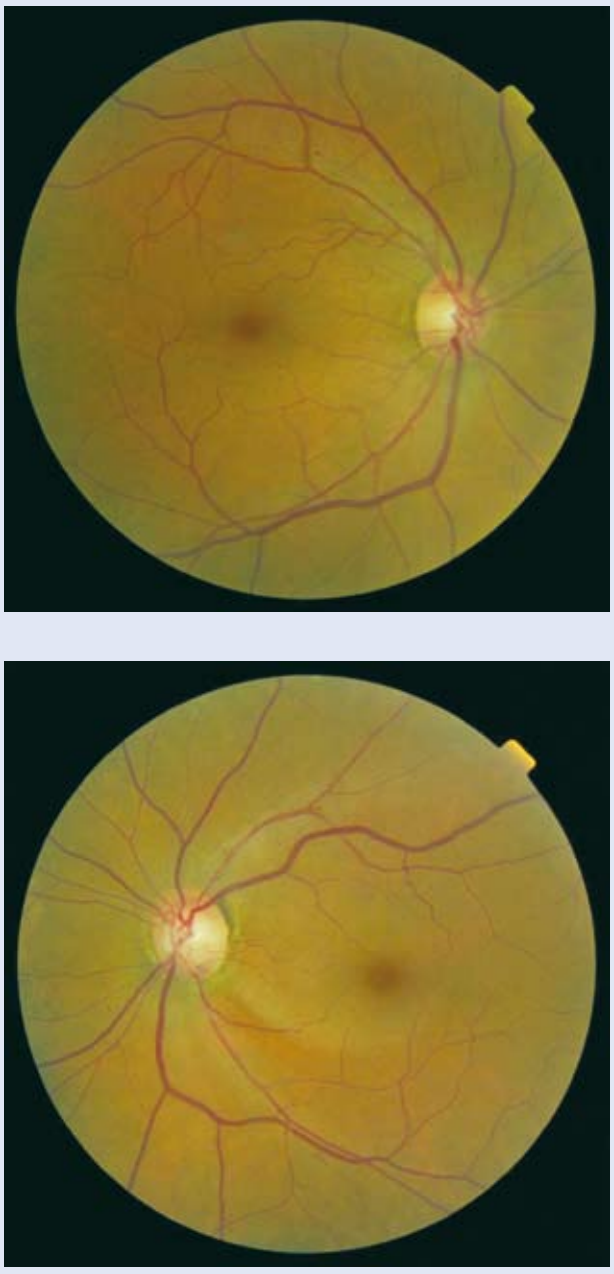
- The average RNFL thickness–this provides an overall assessment of RNFL health. The current guidelines in glaucoma assessment are that above 80 microns is normal, 70-79 borderline, 60-69 early glaucoma, 50-59 moderate glaucoma, 40-49 advanced glaucoma.
- Inferior RNFL average. Normal is 138 µm, early glaucoma is 104 µm. In this case the reading is 54.
- Superior RNFL average. Normal is 142.7 µm, early glaucoma is 104.8 µm.

As the visual fields have shown to be stable, the current treatment has been maintained with six-monthly reviews. This case highlights the difficulties in diagnosing and treating normal tension glaucoma.

Would the patient have had a better outcome had OCT been done routinely years before? ■

Retinal nerve fibre layer defects

Professor Algis Vingrys
BScOptom PhD FAAO



The right (top) and left (bottom) eyes of a 56-year-old woman with a family history of glaucoma. Her pressures are 22 mmHg R and L and she shows normal retinal nerve fibre layer (RNFL) in the RE with three localised defects in the left (5 and 6 o'clock and macula bundle). Her visual fields on HFA 24-2 were normal at this stage. Photo: Algis Vingrys

Clinical conundrum

Answer

Before leaving for Russia, BM had his prescription for drops filled at a different pharmacy from the one he usually visited. When BM presented for examination I realised that he was using the wrong drops, which was affecting his visual acuity.

He was using Atopt 1% (Atropine) instead of Azopt. This caused his pupils to dilate and fix and raised his IOP to R 28 mmHg L 18 mmHg. Once he changed his drops back to Azopt 1% and Travapost, his IOP returned to R 16 mmHg L 16 mmHg.

Educating patients to check that their medication has been dispensed correctly is important. Practitioners should consider asking patients to bring their medications with them to the consultation to ensure they are correct and instructions are being followed. This clinical conundrum demonstrates the consequences when insufficient attention is paid to medication labels.

Multidisciplinary approach

Professor Jonathan Crowston
Head of glaucoma department RVEEH

The Royal Victorian Eye and Ear Hospital (RVEEH) is modelling a multidisciplinary approach to eye care through one of its outpatient clinics. The Glaucoma Monitoring Clinic is in its second year and is a collaborative service involving ophthalmologists, optometrists, orthoptists and ophthalmic nurses.

The clinic, led by Dr Cathy Green, is additional to existing glaucoma clinics. It caters for patients with complex or advanced glaucoma who require follow-up in a tertiary referral setting but have relatively stable disease. Benefits include reduced waiting times for appointments and a stratification of patients to allow targeted clinical care based on the patient's condition.

Every patient is first assessed by a member of the eye-care team and undergoes visual field testing if required. This assessment includes gonioscopy and a dilated fundus examination. Once relevant tests have been performed, the case is discussed with a glaucoma specialist who reviews the clinical findings and outlines a management plan.

This approach provides an excellent opportunity for training and comment for both practitioner and patient. The clinic begins with a teaching session that supports discussion of evidence-based practice, interesting cases and evaluation techniques.

A recent quality assurance survey conducted by RVEEH's administrative staff on the patients attending any glaucoma clinic revealed that several key factors scored more favourably from attendances to the monitoring clinic. More than 80 per cent of patients preferred the monitoring clinic due to the shorter waiting times, consolidation of examinations to one room and amount of information received.

This information reflects the interactive approach between treating consultant, clinician and patient that encourages a more detailed and open discussion of the patient's status.

The program provides an excellent opportunity for improving skills in monitoring glaucoma within the eye-care community. ■

Depression

Glaucoma drops can induce depression, according to a case study published in the online version of the *Medical Journal of Australia*.

The authors referred to the clinical record of a 70-year-old patient who developed depressive symptoms within days of beginning prostaglandin analogue (travoprost) and beta-blocker (timolol) combination drop therapy for worsening glaucoma.

The patient was prescribed venlafaxine for his depression and ceased glaucoma drop therapy, and his symptoms—which included fatigue, sleep disturbances, poor concentration and loss of libido and appetite—improved significantly.

The patient had experienced worse depression requiring hospitalisation and electroconvulsive therapy 11 years earlier when his glaucoma was diagnosed, for which the beta-blocker betaxolol was prescribed.

According to the case report, the patient's depression in this instance was several months later linked to the commencement of betaxolol therapy, and within 48 hours of ceasing treatment he felt more energetic, alert and alive.

MJA 2008; 189: 7: 406-407

Latanoprost enhanced

Italian research has found that the IOP-lowering effects of glaucoma medication latanoprost are enhanced when combined with ketorolac, a non-steroidal, anti-inflammatory drug.

According to the study's authors, participants who were administered ketorolac either orally or topically in addition to their latanoprost therapy experienced a 'marked decrease in IOP ... which remained still significant eight hours later'.

Current Eye Research 2008: 33: 5 and 6: 477-482

News briefs

OAG patients at greater risk of ocular surface disease

A significant proportion of patients with open-angle glaucoma and ocular hypertension have symptoms of ocular surface disease, a study has found.

The study involved 101 patients aged 18 years or older. They completed an Ocular Surface Disease Index questionnaire and were evaluated by Schirmer testing and corneal and conjunctival lissamine green staining, and for tear break-up time.

It was found that 59 per cent of patients had dry eye symptoms in at least one eye.

Schirmer testing showed that 61 per cent experienced decreased tear production in at least one eye, while corneal and conjunctival lissamine green staining revealed positive results in 22 per cent.

Measurement of tear break-up time revealed abnormal tear quality in 79 per cent of patients.

Twenty-seven per cent of patients suffered severe dry eye symptoms in at least one eye and 35 per cent demonstrated a severe tear-production deficiency.

It was also found that for every extra eye-drop containing benzalkonium chloride a patient was taking, their risk of showing abnormal results on the lissamine green staining test increased significantly.

The study, entitled the 'Prevalence of ocular surface disease among glaucoma patients', was published in the *Journal of Glaucoma* (2008; 17: 5 350-355).

Medication works to delay POAG

Using topical ocular hypotensive medication is a safe and effective method in delaying or preventing the onset of POAG in patients with high IOP.

Results from the ocular hypertension treatment study (OHTS) revealed that medication is effective in reducing the incidence of glaucomatous visual field loss and optic nerve deterioration in patients with elevated IOP of between 24 and 32 mmHg.

The OHTS is a landmark study that provides evidence that practitioners should be considering the initiation of treatment for patients who are at moderate to high risk for developing POAG.

The mean IOP reduction in medicated patients in the trial was 22.5 per cent, higher than the target reduction of 20 per cent. During the course of the trial 87 per cent of the medicated participants achieved the IOP target in both eyes and an additional seven per cent did so in one eye. The use of all commercially available topical ocular hypotensive medication prescribed singularly or in combination allowed a high proportion of participants to reach their target IOP.

It is not necessary for all patients with elevated IOP to be treated with topical ocular hypotensive medication. The study outlines factors practitioners should consider before initiating treatment such as: the burden of long-term treatment on the patient, including possible side-effects, costs and inconveniences; the patient's risk of developing POAG; the likelihood that the medication will be effective; and the patient's health-status and life expectancy.

Arch Ophthalmol 2002; 120: 701-713

Race not a key factor

A European study conducted entirely on Caucasians has indicated that the impact of descent or race as a predictive factor for development of open angle glaucoma (OAG) among ocular hypertension patients may not be as important as other ocular or systemic factors.

The results of the European Glaucoma Prevention Study (EGPS) are strongly consistent with and generally replicate the results of the Ocular Hypertension Treatment Study (OHTS), an identical American study conducted on patients from Caucasian, African American and Hispanic backgrounds.

The collaborative results from the two studies will be used to refine a model that estimates the risk of developing OAG in a five-year period in individuals with an IOP between 22 and 31 mmHg. The model will more accurately identify ocular hypertension patients who may warrant treatment with ocular hypotensive medications.

This is significant as ocular hypertension has been recognised as the most important risk factor for the development of OAG and is the only factor that can be influenced by medication or surgery.

The findings of the EGPS confirm that older age, higher IOP, larger vertical or horizontal cup-to-disc ratio, greater pattern standard deviation, and lesser central corneal thickness at baseline are significant predictors of the onset of OAG.

Ophthalmology 2007: 114: 3-9

Three drugs have similar effect

There is no significant difference in efficacy among latanoprost, bimatoprost and travoprost in patients with open-angle glaucoma or ocular hypertension, according to a 12-week study published in the *American Journal of Ophthalmology*.

This is the first randomised, controlled trial simultaneously comparing the IOP-lowering efficacy and safety of these three topical prostaglandin analogues.

Although IOP measurements were significantly reduced from baseline in each of the three treatments, the study revealed lower rates of ocular side effects in latanoprost compared with bimatoprost and travoprost. Significantly fewer latanoprost-treated patients reported eye redness. After three and six months of therapy, hyperaemia rates in patients treated with latanoprost were less than half of those treated with bimatoprost.

Am J Ophthalmol 2003; 135: 688-703

Anti-glaucoma agents prescribed by optometrists

Agent, dose	Product(s)	Conc	Indications	IOP reduction	Precautions/Contraindications	Other details
ALPHA AGONISTS						
Apraclonidine Hydrochloride Dose: bid/tid	**Iopidine**	0.50%	Short-term IOP reduction in patients already on maximally tolerated glaucoma medication	22-28%	MAO inhibitors, systemic sympathomimetics, tricyclic antidepressants. Impaired renal and hepatic function	Neuroprotective? Tachyphylaxis often occurs within 2/12. Possible side effects: GIT problems, bradycardia, HA
Brimonidine Tartrate Dose: bid/tid	**Alphagan** **Enidin** **Combigan** (with timolol 0.5%)	0.20% 0.20% 0.20%	Patient with cardiac or pulmonary problems. Often indicated as an adjunctive treatment .	22-28%	Hypertension, ischaemic disease, MAO inhibitors, depression	Neuroprotective? Side effects: HA, weakness
BETA BLOCKERS						
Betaxolol Hydrochloride Dose: bid	**Betoptic** **Betoptic S** **Betoquin**	0.50% 0.25% 0.50%	Primary treatment where less significant reductions in IOP are required or used as an adjunctive treatment	17-25%	Severe COPD, cardiac disease, systemic β-blocking agents, myasthenia gravis	Neuroprotective? Side effects: blurred vision, depression, impotence
Timolol maleate Dose: qd, bid	**Tenopt** **Timoptol** **Timoptol-XE** **Nyogel** Timolol combinations: **Combigan** (with brimonidine 0.2%) **Cosopt** (with dorzolamide 2%) **Xalacom** (with latanoprost 0.005%) **DuoTrav** (with travoprost 0.004%)	0.25, 0.5% 0.25, 0.5% 0.25, 0.5% 0.10% 0.5% 0.5% 0.5% 0.5%	Primary or adjunctive treatment of POAG	25-29%	Asthma, severe COPD, cardiac disease, systemic β-blocking agents	Tachyphylaxis may occur after 1 year of treatment. Side effects as above. Store gels upside down to prevent bubbles.

CARBONIC ANHYDRASE INHIBITORS						
Dorzolamide Hydrochloride Dose: bid/tid	**Trusopt** **Cosopt** (with timolol 0.5%)	2.0% 2.0%	Primary or adjunctive treatment of POAG. (Used with β-blockers & PG analogues)	15-24%	Sulphonamide hypersensitivity, impaired renal function.	Side effects: ocular irritation, bitter taste, HA, bronchospasm, acidosis
Brinzolamide Dose: bid	**Azopt** **BrinzoQuin**	1.0% 1.0%	As above, useful for patients with LTG and those who cannot tolerate β-blockers	15-19%	As above	Side effects: uncommon but include bitter taste, acidosis
MUSCARINIC AGONISTS						
Carbachol Dose: bid,tid,qid	**Isopto Carbachol**	1.5% & 3.0%	Non-preferred treatment of chronic open angle glaucoma, acute ACG	15-29%	Conditions where miosis is undesirable (eg iritis, risk of retinal detachment), cardiac failure, asthma, hyperthyroidism, Parkinson's disease. Concurrent prostaglandin analogues	Side effects are common: ocular irritation, accommodative spasm, HA, nausea, fatigue, hypotension. Compliance may be difficult when qid dosing is required.
Pilocarpine hydrochloride Dose: bid,tid,qid	**Pilopt** **P.V. Carpine**	0.5,1,2,3,4,6% 0.5,1,2,3,4,6%	Chronic open angle glaucoma, useful for pseudo-exfoliation and pigment dispersion	15-29%	As above. Additionally: corneal abrasion (as this can increase systemic absorption)	As above
Pilocarpine nitrate Dose: bid,tid,qid	**Pilocarpine Nitrate Minims**	1,2,4 %	Combination therapy with β-blocking agents and α-adrenergic agonists. Acute closed angle glaucoma.		As above	
PROSTAGLANDIN ANALOGUES						
Bimatoprost Dose: qd	**Lumigan**	0.03%	Primary or adjunctive treatment of POAG	25-35%	Hx of HSV keratitis as drug may stimulate recurrence, active infection or inflammation of the eye, unilateral treatment	Conjunctival hyperaemia, eyelash growth/ thickening HA, weakness and abnormal liver function
Latanoprost Dose: qd, pm	**Xalatan** **Xalacom** (with timolol 0.5%)	0.005% 0.005%	Primary or adjunctive treatment of POAG	25-35%	As above	As above
Travoprost Dose: qd, pm	**Travatan** **DuoTrav** (with timolol 0.5%)	0.004% 0.004%	Primary or adjunctive treatment of POAG	25-35%	As above	As above

Disclaimer: This table serves to summarise the major details of the anti-glaucoma medications available to Australian optometrists. The clinical details provided are non-exhaustive and may not be applicable to all patients.

Medmont-Star conversion

Can the Medmont Automated Perimeter be used with the STAR II Glaucoma Risk Calculator?

Dr Mark J Walland
FRANZCO FRACS

The Scoring Tool for Assessing Risk (STAR) and STAR II calculators were developed based on the Ocular Hypertension Treatment Study (OHTS) results. These tools allow the risk factors of age, baseline IOP, central corneal thickness (CCT), Humphrey Field Analyzer (HFA) Pattern Standard Deviation (PSD), and vertical cup-to-disc ratio to be processed to give a five-year risk estimate of glaucoma developing for those patients who are ocular hypertensive.

While glaucoma can also clearly occur in patients who have 'normal' IOP, the calculator cannot be used to assess glaucoma risk in normotensive subjects.

The STAR II calculator is an upgraded version of the initial STAR calculator and has been validated against the control group from the European Glaucoma Prevention Study, which has also resulted in diabetes mellitus being deleted as a factor from the newer instrument. The STAR and STAR II calculators are supported by Pfizer Inc.

The problem for many optometrists and ophthalmologists in Australia is that the visual field data required to use STAR II must be the PSD figure from the HFA. Pattern Defect data from the Medmont Automated Perimeter (MAP), for example, is not equivalent and cannot be used directly. Fortunately, work exists that allows the derivation of a PSD-equivalent for the MAP.

Landers and colleagues have published two papers comparing the performance of the MAP and HFA on an empirical basis with sequential testing in more than 60 subjects. This has allowed derivation of a polynomial formula that can be used to convert Medmont PD figures to HFA PSD-equivalents (Table). The Table provides a conversion using the formula across the relevant range of values for input into the STAR II calculator and is useful in the clinical setting.

The PSD inputs in STAR II are limited to 0.50-3.00 dB. Greater pattern defects are more likely to be glaucoma than OHT, and the calculator is validated only for glaucoma development, not for glaucoma progression. The Table is also clearly applicable only to Medmont users; another conversion has been published in Switzerland, for example, for the Octopus perimeter, which is more favoured in Europe. ■

Humphrey Field Analyzer	Medmont Automated Perimeter
PSD	**PD**
0.50	1.09
0.60	1.30
0.70	1.52
0.80	1.73
0.90	1.94
1.00	2.15
1.10	2.36
1.20	2.57
1.30	2.78
1.40	2.99
1.50	3.20
1.60	3.40
1.70	3.61
1.80	3.81
1.90	4.01
2.00	4.21
2.10	4.41
2.20	4.61
2.30	4.81
2.40	5.01
2.50	5.21
2.60	5.40
2.70	5.60
2.80	5.79
2.90	5.98
3.00	6.17

Conversion of Humphrey Field Analyzer Pattern Standard Deviation and Medmont Automated Perimeter Pattern Defect using the Landers formula in the PSD range 0.5-3.0 dB for the STAR II calculator

Pfizer Ophthalmics

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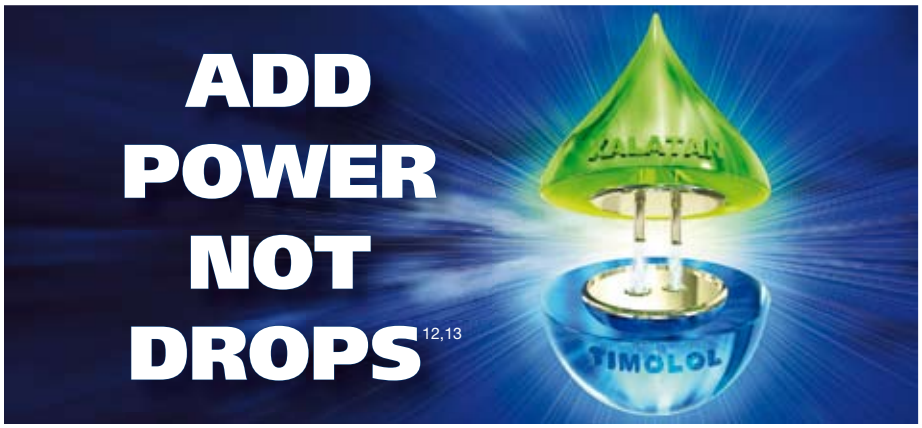
Efficacy[1-4]
+
Tolerability[1-3,7-11*]
=
FIRST LINE
Xalatan®
latanoprost



THE POWER OF ONE.[12,13]
Once Daily
Xalacom™
latanoprost/timolol maleate

Xal-Ease™
Complementary

*Refers to ocular hyperaemia compared to other prostaglandins and systemic adverse events compared to timolol.

PBS Information: This drug is listed on the PBS for the treatment of Open Angle Glaucoma and Ocular hypertension.

Before prescribing, please refer to Approved Product Information. Full Approved PI is available on request from Pfizer. MINIMUM PRODUCT INFORMATION. XALATAN® (Latanoprost 50 micrograms/mL) Eye Drops INDICATIONS Reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension. CONTRAINDICATIONS Hypersensitivity to ingredients. PRECAUTIONS Change in eye colour due to increased iris pigmentation, heterochromia; eyelid skin darkening; changes in eyelashes and vellus hair; macular oedema; other types of glaucoma; pseudophakia; aphakia; contact lenses. Severe or brittle asthma. Pregnancy category B3, lactation. Children. Interactions: other prostaglandins, thiomersal. Blurring of vision. ADVERSE EFFECTS Increased iris pigmentation; eye irritation (burning, grittiness, itching, stinging and foreign body sensation); eyelash and vellus hair changes (darkening, thickening, lengthening, increased number); mild to moderate conjunctival hyperaemia; transient punctate epithelial erosions; blepharitis; eye pain; conjunctivitis; vision blurred; eyelid oedema, macular oedema. Muscle/joint pain; dizziness; headache; localised skin reaction on the eyelids; skin rash. Uncommonly: keratitis; non-specific chest pain; Others, see full PI. DOSAGE AND ADMINISTRATION One eye drop in the affected eye(s) once daily. Other eye drops at least 5 minutes apart. REFER TO PRODUCT INFORMATION BEFORE PRESCRIBING The full disclosure Product Information is available on request from Pfizer Australia Pty Ltd. NAME AND ADDRESS OF THE SPONSOR Pfizer Australia Pty Ltd ABN 50 008 422 348, 38-42 Wharf Road, West Ryde, NSW 2114. Full PI approved by the TGA on 4 February 2003, last amended 20 November 2006. PBS dispensed price, September 2007: $36.65. References: 1. Parrish RK *et al. Am J Ophthalmol* 2003;135:688-703. 2. Gandolfi S *et al. Advances in Therapy* 2001;18(3):110-121. 3. Netland PA *et al. Am J Ophthalmol* 2001;132:472-484. 4. Hedman K *et al. Surv Ophthalmol* 2002;47(Suppl 1):S65-S76. 5. Reardon G *et al. Eur J of Ophthalmol* 2003;13(Suppl 4):S44-S52. 6. Stewart WC *et al. Rev of Ophthalmol* 2002;9(4). Accessed via URL http://www.revophth.com/index.asp?page=1_83.htm. 7. Noecker RS *et al. Am J Ophthalmol* 2003;135:55-63. 8. Watson P *et al. Ophthalmology* 1996;103:126-137. 9. Konstas AGP *et al. Am J Ophthalmol* 1999;128:15-20. 10. Mishima HK *et al. Arch Ophthalmol* 1996;114:929-932. 11. Alm A *et al. Ophthalmology* 1995;102:1743-1752. ®Registered trademark of Pfizer Inc. Pfizer Medical Information 1800 675 229. 04/08 PFXXA7518-B/FC

PBS Information: Restricted benefit:
This product is listed on the PBS for the reduction of elevated intra-ocular pressure in patients with OH or POAG who are not adequately controlled with timolol maleate 5mg (base) per mL (0.5%) eye drops or latanoprost eye drops.

Before prescribing, please refer to Approved Product Information. Full Approved PI is available on request from Pfizer. MINIMUM PRODUCT INFORMATION. XALACOM® Eye Drops (latanoprost 50µg/mL and timolol 5mg/mL). INDICATIONS. Reduction of IOP in open-angle glaucoma and ocular hypertension, if insufficient response to other medications. Not for initial therapy. CONTRAINDICATIONS. Reactive airway disease including bronchial asthma (and history), or severe COPD. Sinus bradycardia, second or third degree atrioventricular block, overt cardiac failure, or cardiogenic shock. Hypersensitivity to ingredients. PRECAUTIONS. Beta-blocker systemic effects: cardiovascular/respiratory reactions; consider gradual withdrawal prior to major surgery; anaphylactic reactions; caution in hypoglycaemia, diabetes, hyperthyroidism, myasthenia gravis; concomitant beta-blocker or prostaglandin not recommended. Ocular: change in eye colour due to increased iris pigmentation, heterochromia; eyelid skin darkening; changes in eyelashes and vellus hair; macular oedema; caution in other types of glaucoma, pseudophakia, aphakia, filtration. Contact lenses. Pregnancy category C, do not use; lactation. Children. Interactions: additive effects with other drugs; thiomersal. Blurring of vision. ADVERSE EFFECTS. Ocular: eye irritation, hyperaemia, abnormal vision, visual field defect, increased iris pigmentation, eyelash and vellus hair changes, corneal oedema and erosions. Systemic: serious respiratory and cardiovascular events (worsening of angina pectoris, pulmonary oedema), anaphylaxis. Others, see full PI. DOSAGE AND ADMINISTRATION. One eye drop in the affected eye(s) once daily. Other eye drops at least 5 minutes apart. REFER TO PRODUCT INFORMATION BEFORE PRESCRIBING The full disclosure Product Information is available on request from Pfizer Australia Pty Ltd. NAME AND ADDRESS OF THE SPONSOR Pfizer Australia Pty Ltd, ABN 50 008 422 348, 38-42 Wharf Road, West Ryde, NSW 2114. Full PI approved by the TGA on 25 November 2002, last amended on 20 November 2006. PBS dispensed price, September 2007: $43.50. References: 12. Higginbotham EJ *et al. Arch Ophthalmol* 2002; 120: 915–22. 13. Konstas AGP *et al. Arch Ophthalmol* 2005; 123: 898–902. www.pfizer.com.au ®Registered trademark of Pfizer Inc. Pfizer Medical Information 1800 675 229. 11/08 XALAT00060

eye drops - olopatadine

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PBS Information: This product is not listed on the PBS.

Please review Approved Product Information before prescribing. Full Product Information is available on request from Alcon Laboratories (Australia) Pty Ltd. PATANOL® (olopatadine) 0.1% Eye Drops Abridged Product Information. Use: Treatment of signs and symptoms of seasonal allergic conjunctivitis for up to 14 weeks. **Contraindications:** Hypersensitivity. **Precautions:** Not for injection or oral ingestion, pregnancy (Category B1), lactation, children below 3 years of age. Caution should be taken when driving or operating machinery if blurred vision is experienced. **Adverse Reactions:** Headaches, asthenia, blurred vision, burning or stinging, cold syndrome, dry eye, foreign body sensation, hyperaemia, hypersensitivity, keratitis, lid oedema, nausea, pharyngitis, pruritus, rhinitis, sinusitis and taste perversion. **Dosage:** One to two drops of PATANOL Eye Drops in the affected eye(s) twice daily. ® Registered trademark. Alcon Laboratories (Australia) Pty Ltd. ABN 88 000 740 830, 10/25 Frenchs Forest Road East, Frenchs Forest, NSW 2086. POPH 1455 **References: 1.** PATANOL Approved Product Information.