Supplement to
AUSTRALIAN OPTOMETRY

Special issue GLAUCOMA

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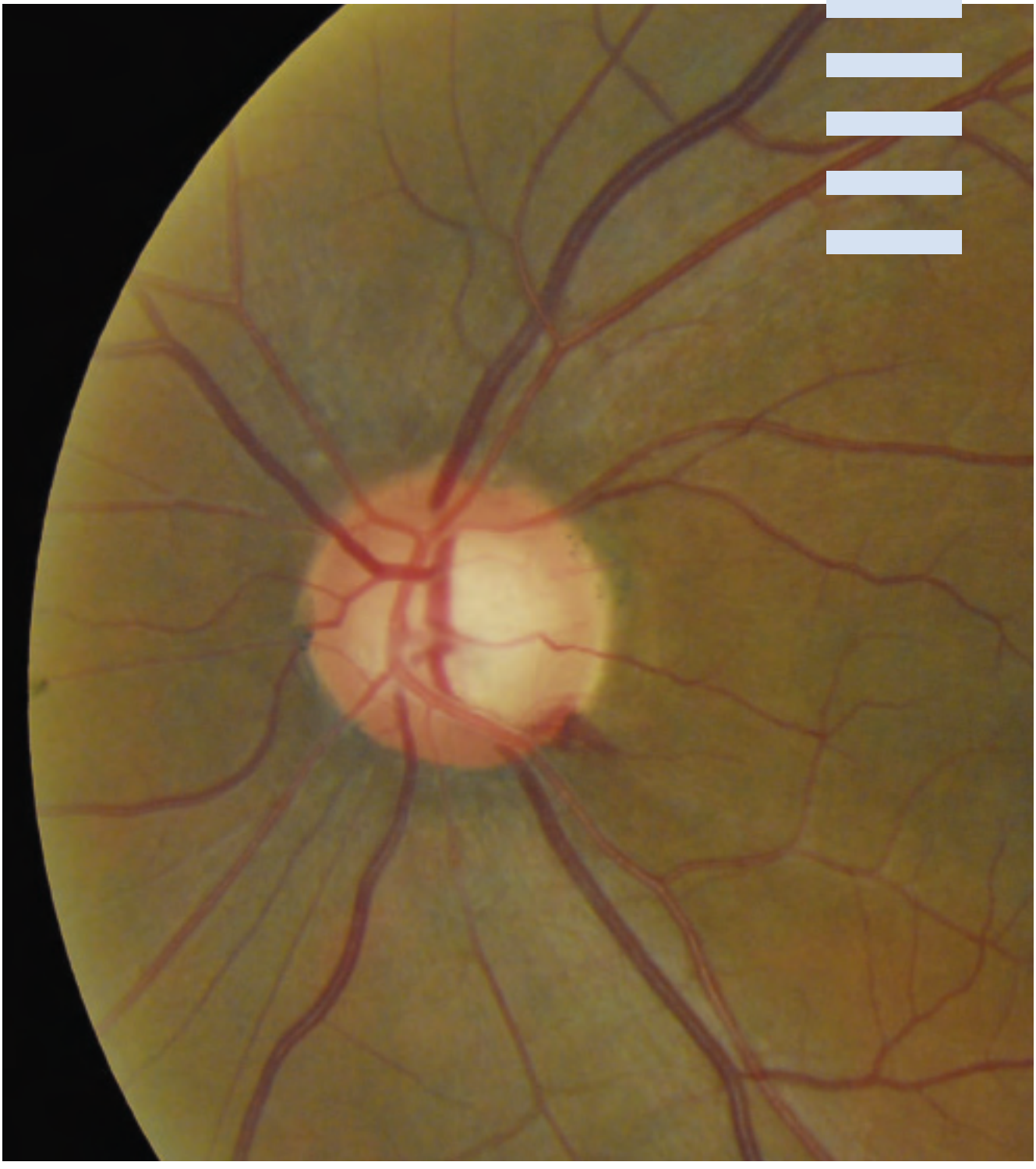
JUNE 2011



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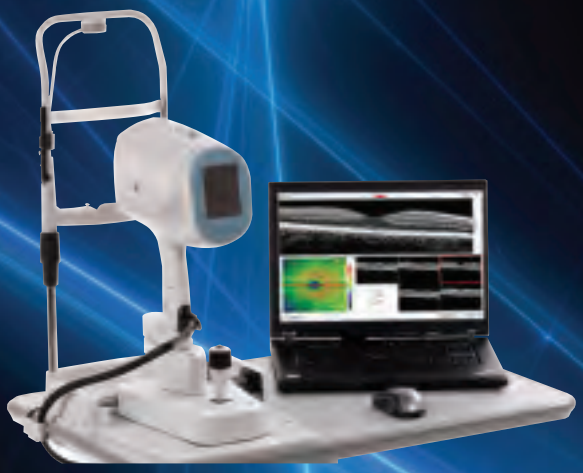

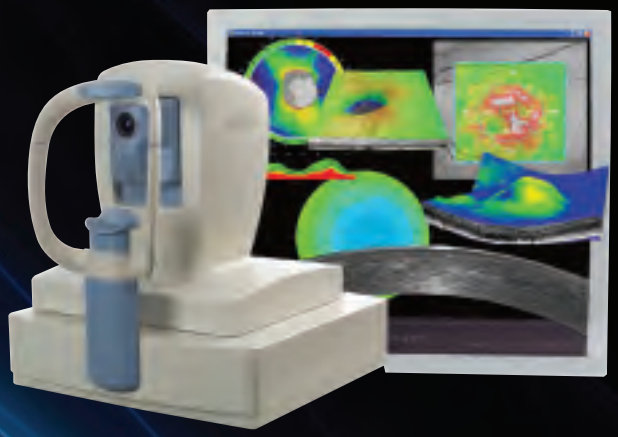

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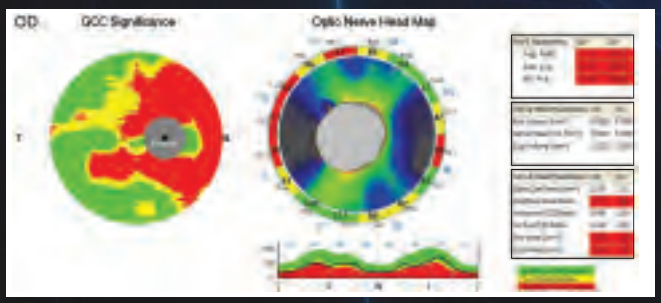


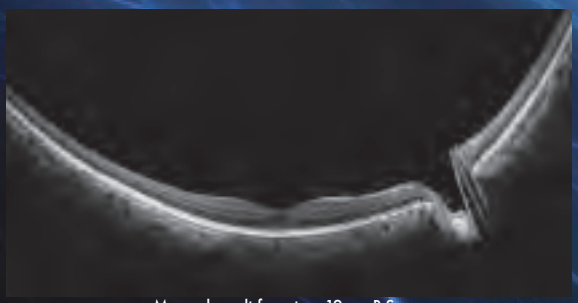

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AUSTRALIAN OPTOMETRY pharma JUNE 2011

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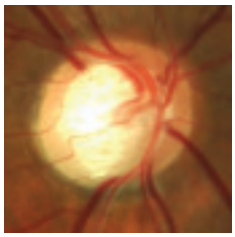
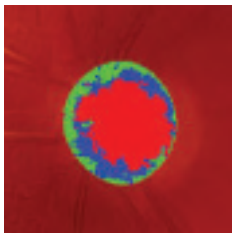
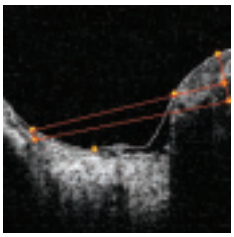
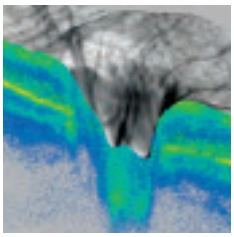
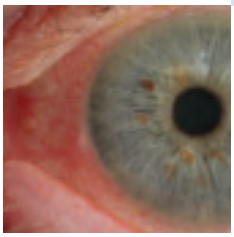

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# An approach to the diagnosis of a glaucoma

**The diagnosis of glaucoma is usually made after a comprehensive clinical eye examination that includes biomicroscopy, applanation tonometry, gonioscopy and dilated stereoscopic evaluation of the optic disc and fundus. Further testing including automated perimetery is performed on those suspected to have glaucoma after such an examination. Automated perimetry confirms functional damage and provides a baseline for follow-up.**

Imaging techniques are not essential for the diagnosis but may have a role to play in follow-up. A comprehensive eye examination for every clinic patient can achieve the objective of detecting all potentially sight threatening disease including glaucoma.

The eye-care practitioner should aim to detect all potentially serious ophthalmic pathology, including glaucoma. Screening for glaucoma using imaging or visual fields prior to a clinical examination can have too many false positives and negatives and is not recommended.

## Introduction

Glaucoma affects around three per cent of the Australian population over the age of 50 years; of those affected, 50 per cent do not know that they have the disease.[1] This article on the evaluation of primary adult glaucomas is targeted at eye-care providers working in Australia with a strategy for prevention of blindness by case detection of established disease. The piece is adapted from an article originally published in the *Indian Journal of Ophthalmology* and has been revised and updated for presentation here.[2] The detection of most ocular pathology including glaucoma is the purview of all eye-care providers.[3, 4]

The patient usually does not present to an eye-care provider with a diagnosis of glaucoma. The presenting complaints may be trivial but it is responsibility of the examining clinician to rule out all potentially serious ocular pathology, including glaucoma. The diagnosis of glaucoma at a treatable stage can be achieved by a clinical examination using basic instrumentation that should be available in every clinician's office.

Glaucoma is a chronic optic neuropathy with typical structural damage to the optic disc, usually leading to correlating functional changes in the visual field.[5-7] 'Raised' intraocular pressure is a causal risk factor for glaucoma, the only one that can be treated and does raise suspicion of glaucoma but it is neither sufficient nor necessary for the diagnosis.[5-8]

It is also important to remember that glaucoma is usually asymptomatic until the late stages, at which time the prognosis is poor. The diagnosis of end stage glaucoma is straightforward and can be made by a trainee using an ophthalmoscope. It is desirable to detect the disease at a stage where the diagnosis is possible, yet intervention can still alter the course of the disease and change the prognosis. Diagnosis at an even earlier stage (pre-perimetric glaucoma) is ideal but far more difficult than and not as critical as in established disease. As early diagnosis comes with implications of 'labelling' and life-long treatment,[3,9] it is best confirmed by an experienced ophthalmic examiner using the experience and tools at their disposal. The diagnostic importance of concepts such as pretest probability, sensitivity, specificity and likelihood ratios of symptoms, signs and tests and how they can be used to confirm a diagnosis are basic to any test and are dealt with elsewhere.[10,11]

## History

Primary open angle glaucoma (POAG) and primary angle closure disease (PACD) are usually asymptomatic. A history of frequent changes of reading spectacles, while suspicious, is not sensitive or specific enough for clinical use. A family history of the disease increases the risk of glaucoma up to eightfold and mandates a careful examination.[12-15] A comprehensive eye examination is recommended for all family members of a glaucoma patient. Myopes are at higher risk for POAG and hypermetropes are at higher risk for PACD.[16,17]

A directed history helps rules out secondary causes for glaucoma such as steroid use (especially topical), trauma, uveitis, sleep apnoea, severe blood loss and intracranial disease. It is wise to enquire about the use of systemic medications that may influence glaucoma management. For example a topical beta blocker may not add significantly to the IOP lowering effect for someone already on a systemic beta-blocker. As another example topical alpha-2 agonists are contraindicated in a patient on monoamine oxidase inhibitors.

## Examination

A comprehensive eye examination is recommended as routine for all ophthalmic patients. Such an eye examination helps detect not just glaucoma but other potentially blinding ocular pathology. Such a comprehensive eye examination comprises:

- Vision and refraction
- External examination and assessment of ocular motility
- Examination of the pupil with special attention to the presence of a relative afferent pupillary defect
- Slitlamp biomicroscopy
- Intraocular pressure measurement, preferably using applanation tonometry
- Gonioscopy to examine the angle of the eye
- A dilated examination of the optic disc and retina
- Visual fields: if glaucoma is suspected, automated perimetry is performed to detect functional defects in the visual field.

The clinical diagnosis of glaucoma is usually based on a combination of intraocular pressure (IOP), gonioscopy, optic disc and visual field examination, and these steps should always be carried out as a part of the comprehensive eye examination, not in isolation.

The prevalence of glaucoma is high enough and the implications serious enough to suggest that ALL patients seen in an eye-care professional's clinic undergo the comprehensive eye examination as well as

# the assessment suspect

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'directed' investigations.

In some instances a diagnosis may not be possible at the initial examination; in suspects and early disease it may be necessary to repeat the entire examination after a period of observation. We briefly describe the essential components of a comprehensive eye examination.[18]

**External examination**
May detect subtle signs of facial hemangioma or dilated episcleral veins, which indicate a secondary cause. Ciliary conjunctival congestion suggests sinister intraocular pathology including acute angle closure.

**Ocular motility**
Detection of amblyopia or sensory exotropia may be the reason for a less aggressive management plan.

**Examination of the pupil**
As glaucoma is usually an asymmetric disease, a relative afferent pupillary defect is an important diagnostic clue and a possible prognostic factor as well. A dilated pupil may be a sign of acute angle closure.

**Slitlamp examination**
Is performed both before and after dilatation to detect signs of pseudo-exfoliation, pigment dispersion, uveitis or trauma. Pigment liberation following dilatation is very suggestive of pseudo-exfoliation (or active pigment dispersion) and directs the search for subtle signs of disease like the early 'brown' stage of pseudoexfoliation (Figure 1). Corneal oedema detected during such an examination may underestimate IOP measurement.

Posterior synechiae may explain pupillary distortion. Corneal endothelial or iris pathology may direct the search towards a secondary cause.

**Intraocular pressure (IOP)**
The IOP is measured at every visit. The current gold standard is the Goldmann applanation tonometer attached to the slitlamp; the hand-held Perkins instrument can be used. too. It is important to remember that the Goldmann applanation tonometer, like any other measurement device, may be subject to errors; measurements must be carefully performed to avoid erroneous readings. The measurement is also affected by corneal thickness. A structurally thick cornea overestimates the IOP while a thin cornea underestimates the measurement.

**We recommend a central corneal thickness (CCT) measurement for all ocular hypertensives and those suspected to have 'normal tension' glaucoma, certainly before subjecting the latter to a neurological 'massage'.[19] On the other hand, we should avoid the tendency to consider the CCT-corrected IOP to be 'accurate', as all the nomograms remain to be validated and issues such as corneal hysteresis may be as or more important. There are other sources of error that cannot be accounted for by the CCT.**

Statistically, an IOP measured in the recommended manner, which after 'correction' for corneal thickness is raised beyond two standard deviations of the population mean, is suspicious. The two standard deviations value varies between populations but > 22 mmHg is a reasonable cut-off for most populations.[20-23] The tonopen, ICARE and air puff tonometers have a place in busy clinics but all abnormal values should be repeated and then confirmed by Goldmann applanation. The Pascal tonometer can provide a closer estimate of the intracameral IOP and seems to have the least variability.

As with any measurement and especially in the absence of other signs of glaucoma, we should not rely on a single reading.[10,11] A measurement obtained after dilatation may increase the 'yield'.

If the disc is suspicious, other signs of the disease are present or the patient is considered high risk, for example, by virtue of a family history, the IOP measurements

**Continued page 4**

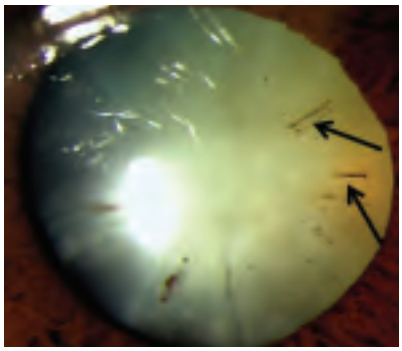

**Figure 1**

**Figure 2**

From page 3

should be repeated. This is necessary not just to detect raised IOP but also to obtain a baseline for treatment.

In the presence of disc and field changes, if the IOP is 'normal' or 'low', multiple readings should be obtained during different times of the day–in come cases, even at night time–if facilities are available. Such multiple readings could be considered before initiating any expensive or invasive investigations to explain the disc and field changes. The principle of multiple readings, preferably obtained at different times of the day applies even after treatment is initiated. Obtaining diurnal curves is difficult; the recent use of the water drinking test to predict the peak IOP and IOP fluctuation requires further study. So-called normal tension glaucoma patients do not routinely require neurological imaging simply by virtue of their IOP; by contrast, any pallor of the optic disc rim and/or disc-field–that is, structure-function–mismatch mandates imaging, irrespective of the IOP.

## Gonioscopy

POAG is a diagnosis of exclusion. The name itself indicates that the signs of glaucoma must be present with an open angle, in the absence of other causes. The visualisation of an open angle is especially important in regions of the world where PACD is common. Australia is a migrant nation that comprises several ethnic groups that are at risk for PACD; and PACD including acute angle closure does occur in the Caucasian population, too. Detection of PACD in the early stages presents an excellent opportunity for prevention using a simple laser iridotomy.[24]

Goniosocopy is used to examine the angle of the anterior chamber and is best performed using an 'indentation' type of gonioscope. (Figure 2). A four-mirror indentation gonioscope is the better choice; the lack of need for coupling fluid also makes the goal of routine gonioscopy easier. In difficult patients and where the four-mirror indentation gonioscope is not available, 'manipulation' with a two-mirror gonioscope to try and achieve indentation is an acceptable option. The features of an open angle are shown in Figure 3.

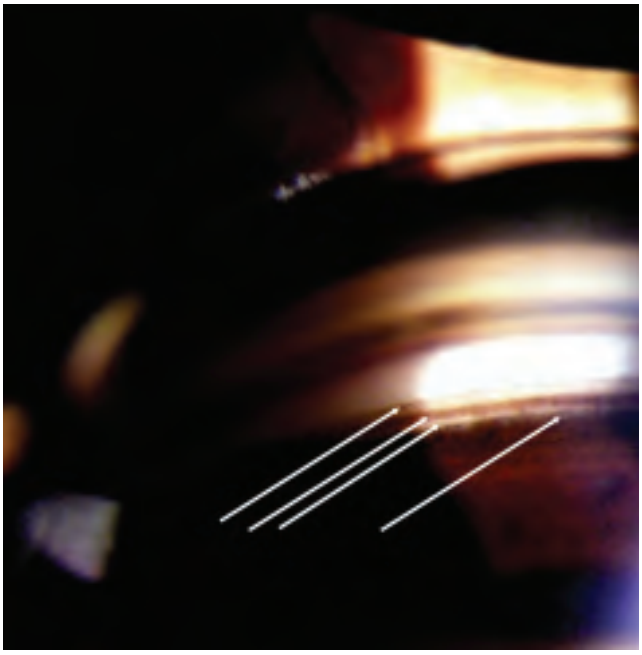

**Figure 3**

**Figure 4**

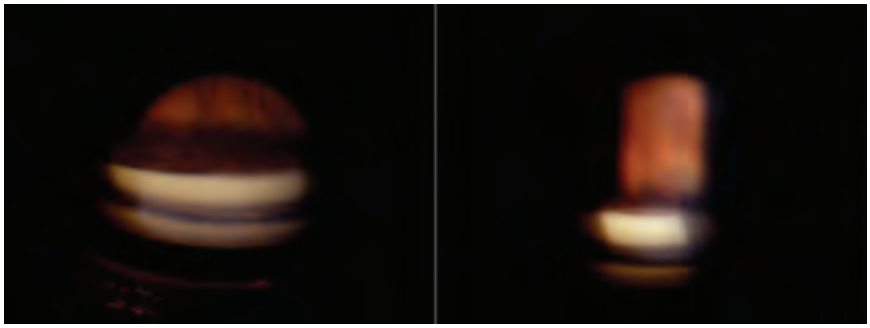

**Figure 5**

When determining if an angle is open or closed, the testing conditions are critical. If the examination is done in a bright room with a long slit beam that impinges on and constricts the pupil, and/or with some pressure applied by the gonioscope, many angles will 'open'. Figure 4 shows the same angle, open with bright illumination but closed in dim illumination.

The ideal gonioscopic testing conditions include: dim room illumination, minimal intensity of the slitlamp illumination, a low slit beam height such that light does not impinge on the pupil and no pressure on the eye with the gonioscope. It is necessary to wait for 30-45 seconds for the pupil to dilate before deciding if the angle is open.

- If under these conditions the posterior trabecular meshwork (PTM) is not seen, the patient is asked to look towards the mirror in order to obtain an 'over the (iris) hill view' of the angle. If > 180 degrees of the PTM is seen with such an 'over the hill view' under the specified testing conditions, without any pressure on the eye, the angle is considered open (Figure 5). If not, the patient is considered a primary angle closure suspect. (The current clinical classification

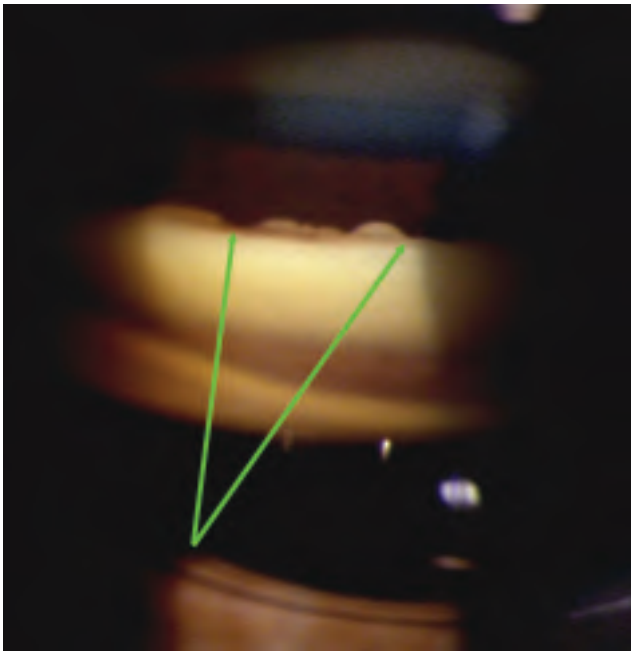

**Figure 6**

uses 270 degrees of PTM non visibility, but data for progression is available only for 180, hence our use of the latter.) In the next step, the illumination and slight beam height are increased to constrict the pupil, and 'indentation' is performed with the gonioscope to look for other signs of pathology in the angle. These may include peripheral anterior synechiae (PAS) (Figure 6), a consequence of angle closure or inflammation, signs of PXE, trauma, old haemorrhage, inflammation or new vessels. The authors recently encountered a rare case where trabecular precipitates were the only evidence of inflammation in a patient being treated as POAG;[25] investigations led to the diagnosis of sarcoidosis.

If other signs are absent AND the angles are open under the conditions described above, then in the presence of disc and/or field changes we consider a diagnosis of POAG.

Gonioscopy is not a once only examination. A patient with POAG can develop an angle closure component over time. Gonioscopy should be repeated at least annually if the signs of the disease change, and also after interventions like iridotomy or trabeculectomy.

## Role of van Herick tests and angle imaging

The van Herick test has been suggested as a screening test for angle closure. The sensitivity and specificity of this test are such that a negative test does not rule out angle closure and a positive test still requires gonioscopy.[3,26] The presence of a positive van Herrick AND a raised IOP is highly specific and almost pathognomonic of closure but gonioscopy is still required for management.[3,26] Accordingly if the philosophy is one of case detection and management, the van Herrick test does not really help.

Angle imaging techniques such as the ultrasound bio-microsope (UBM) and anterior segment OCT (ASOCT) are adjunctive tools in the management of PACD, are subject to the general rules guiding such tests and have not replaced the gonioscope. While the ASOCT is easier to use than the UBM, both instruments examine only a few sections of the angle. False positives of PACD are common and most signs in the angle (blood, black balls, blotchy pigment) can be missed. Unless a scanning section has serendipitously passed through an area of peripheral anterior synechia, even this important pathology can be missed. At the moment the ASOCT can at best identify eyes that may harbour such angle pathology but the detection of most such pathology requires a gonioscope.

Available data for progression of PACD that currently guides management were obtained using a gonioscope and this data cannot be extrapolated to imaging. There is currently no evidence to base management decisions on imaging alone. It is possible that imaging for PACD will have a role to play in documenting progression and the effect of intervention but at the moment they have not replaced gonioscopy and are not necessary for routine clinical use.

## Optic disc and NFL examination

A magnified, stereoscopic examination of the optic disc using a 60-90 D lens, or a contact lens with the slitlamp is the ideal method of examining the optic disc and nerve fibre layer (NFL).[18] Retinal examination also requires an indirect ophthalmoscope but this alone is not good enough to comment on the optic disc.

**In experienced hands the direct ophthalmoscope can provide valuable information. Stereo-photographs are considered the current gold standard for documentation and monitoring of the optic disc. There is considerable disagreement even between experts for evaluating progression using stereo-photographs and this is likely to be greater with standard disc photographs. Stereo-photographs detect disc haemorrhages more often than routine clinical examinations. It is possible that the same may be true for standard photographs but the data is not available. Finally, while stereo-photographs are still considered the gold standard, optic disc findings should at least be documented with a drawing or imaging for comparison with future examinations.**

The structural changes in the optic disc in glaucoma are numerous; the diagnosis is based on a combination of signs.[27]

- The most commonly used sign for the diagnosis of glaucomatous damage is an 'increased' cup to disc ratio (CDR). Generally, an arbitrary statistical cut off of 0.7:1 is considered to be suspicious, more so if the cup is vertically oriented. The CDR can be fallacious and should not be used in isolation. The reason is that about one million plus axons exit the eye through the optic disc; they form the 'neuro-retinal' rim of the optic disc. Think of the cup as the 'space' that is left over after these axons have been 'accommodated' in the disc. The size of the optic disc varies considerably; the 'space left over', that is the cup, has to vary with the size. Accordingly, a small disc may not be entitled to any cup; and a large disc is entitled to a very large cup, beyond the 0.7:1 cut-off (Figure 7) .

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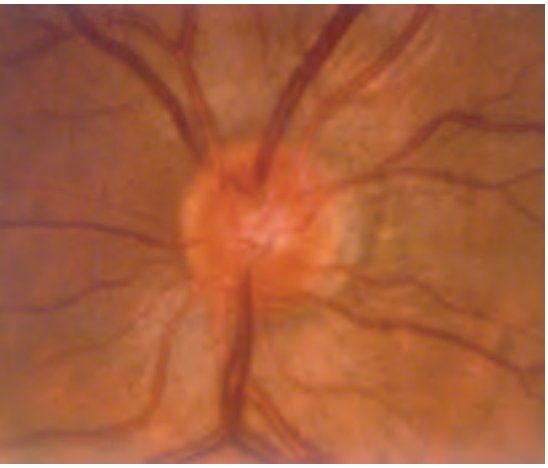

Figure 7A

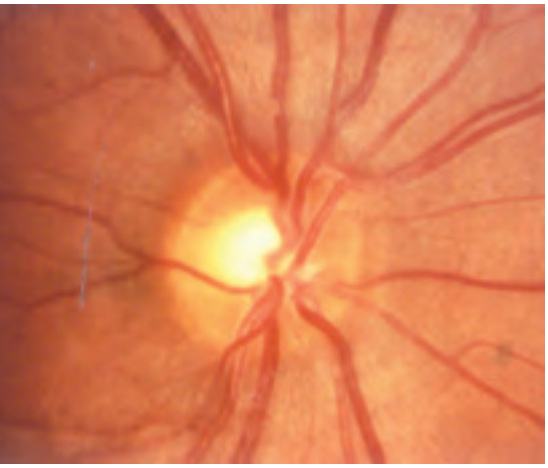

Figure 7B

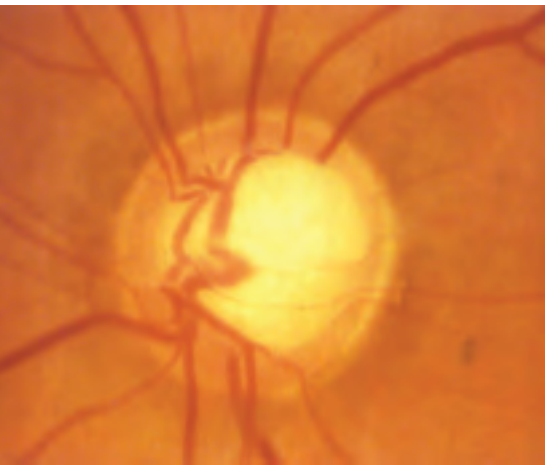

Figure 7C

The cup disc ratio can be useful, but only if it is related to the size of the disc. The size of the disc can be easily estimated on the slitlamp with a 60 D lens. A narrow slit beam height is adjusted vertically until it just encompasses the margins of the optic disc and the height is read directly off the graticule: 90 D and 78 D lenses require a conversion factor (x1.3 and x1.1 respectively) (Figure 8).

It is not as important to obtain an actual measurement as it is to get a feel for whether a disc is small (vertical diameter <1.5 mm), medium (1.5-2 mm), or large (>2 mm). As with any other examination, that becomes possible only after examining and measuring a large number of discs. The question we ask is: 'Is this disc physiologically allowed to have this sort of cup?' A small cup, like 0.3, usually considered to be in the normal range, may not be physiological in a small disc; on the other hand, a large cup may by physiological in a large disc. In other words, a small cup may be abnormal in a small disc and a large cup may be normal for a large disc.

The CDR is also useful in two other situations. If, after accounting for a difference in size of the two discs, the CDR in the two eyes differs by more than > 0.2, that is suspicious for glaucomatous damage. A loss of rim tissue (increase in the cup) over time without rim atrophy is pathognomonic of glaucoma.

It is important to remember that the cup and the CDR are only a surrogate for the tissue that we really want to examine: the neuro retinal rim (NRR). Changes in the neuro retinal rim suggest pathology.

Rather than the usual cup to disc ratio, we prefer to document the RIM to DISC ratio in the superior, superotemporal, inferotemporal, inferior and nasal areas of the disc. A rim to disc ratio of under 0.1:1 should be considered pathology until proved otherwise. Rim to disc ratio also allows better monitoring.[28,29]

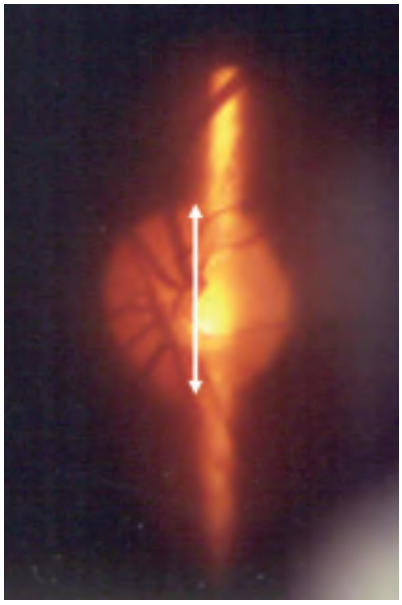

Figure 8

## Changes in the neuro retinal rim

**Pattern**

The NRR is usually thickest inferiorly, followed by superior, nasal and temporal (Figure 9). This 'ISNT' rule holds true in about 80 per cent of normals. While that is not specific enough and there may be normal variations, any change in this pattern should be considered suspicious[27] (Figure 10). If the inferior rim is thinner than the superior, that could suggest pathology. Certainly, an inferior or superior rim that is equal to or thinner than the temporal rim is highly suspicious. The temporal rim should be the thinnest.

Localised narrowing of the inferior or superior rim that does not extend to the rim is also suspicious. A rim that extends to the edge of the disc for a clock hour is called a notch. A notch is characteristic of glaucoma and usually correlates with a functional field defect (Figure 11) .

A flame-shaped haemorrhage that touches the neuroretinal rim is specific but not sensitive for glaucoma (Figure 12).

Pallor of the rim is NOT a sign of glaucoma. Pallor of the rim outside the area of loss or out of proportion to the 'cupping' suggests other neurological causes and needs to be investigated.

Peripapillary choroidal atrophy is a soft sign of glaucomatous damage. It is significant if associated with other signs or if it increases in size.

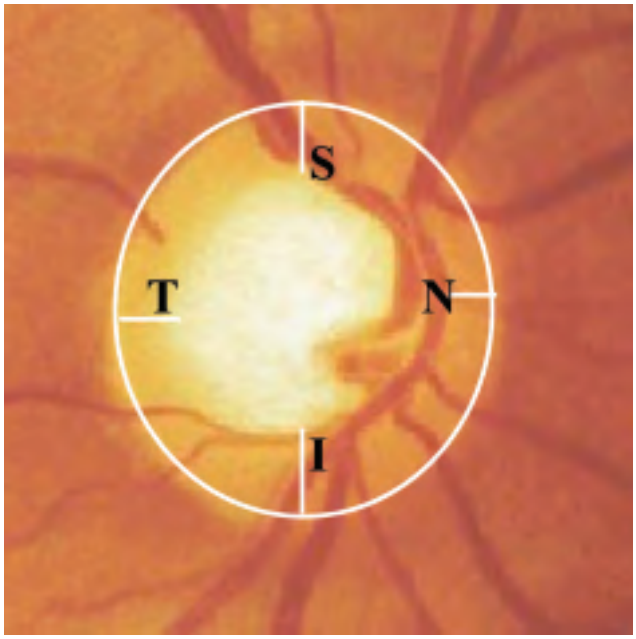


Figure 9

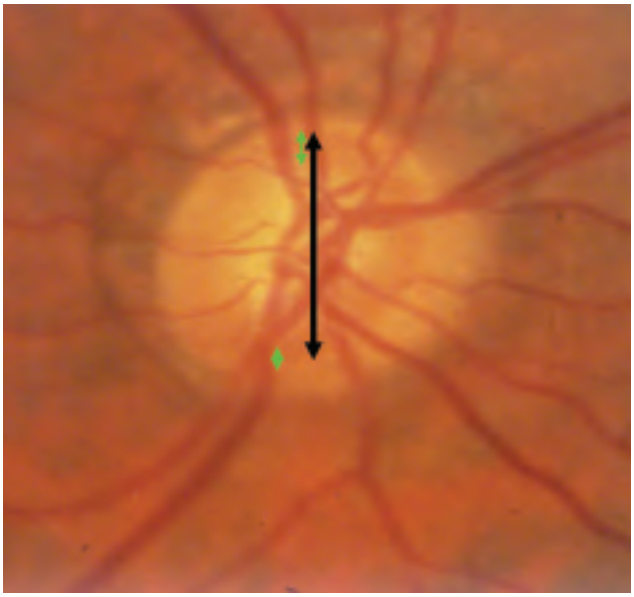

Figure 10

**Nerve fibre layer defect (NFLD)**

The gold standard for the examination of the NFL is red free photography but it can be examined clinically using the green filter on the slitlamp or ophthalmoscope. It is sometimes clearly seen on indirect ophthalmoscopy too, both with and without the green filter.

The normal arcuate nerve fibre layer is seen as fine bright striations. Viewed from the superior to the inferior arcuate area the NFL has a bright, dark, bright pattern, the 'dark' being the region between the disc and macula (Figure 13). The inferior arcuate NFL has a larger area and is more clearly seen than the superior arcuate NFL; this is consistent with the NRR thickness.

A localised NFLD appears as a dark wedge that follows the pattern of the nerve fibre layer and widens towards the periphery (Figure 14). Such a defect should be wider than an arteriole, touch the edge of the disc and increase in width towards the periphery. NFL defects have a strong predictive value for future functional changes. The specificity is very high but the sensitivity is poor. The defects are a definite sign of pathology but can occur in diseases other than glaucoma, too. Diffuse NFLDs are more difficult to detect. The normal bright, dark, bright pattern is lost. The pattern takes on a dark, dark, dark apearance. Better visibility of the superior NFL compared to the inferior is also suspicious.

The diagnosis of glaucomatous changes in the optic nerve is usually based on a combination of the above signs.[27-29] For example in a disc with a notch as well as a NFLD, the combined specificity is high enough to rule in glaucoma. Similarly, in a disc with a thinning of the rim as well as an optic disc haemorrhage, the specificity is again high enough to rule in glaucoma. On the other hand, the sensitivity of individual signs is not high enough to rule out glaucoma unless most or all the signs are absent.

Jost Jonas usually teaches three rules for optic disc examination in glaucoma diagnosis (Jonas JB, personal communication). Until proved otherwise:

- all glaucoma suspects have nerve fibre layer defects
- all glaucoma suspects have optic nerve haemorrhage
- all myopes have glaucoma (myopes are at higher risk for glaucoma).

**Continued page 8**

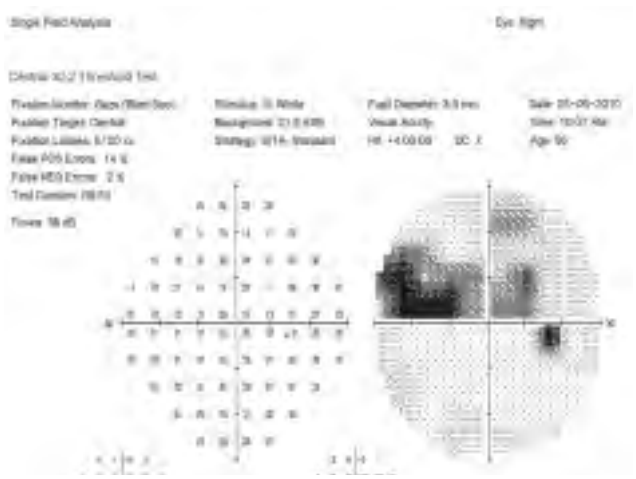

Figure 11A

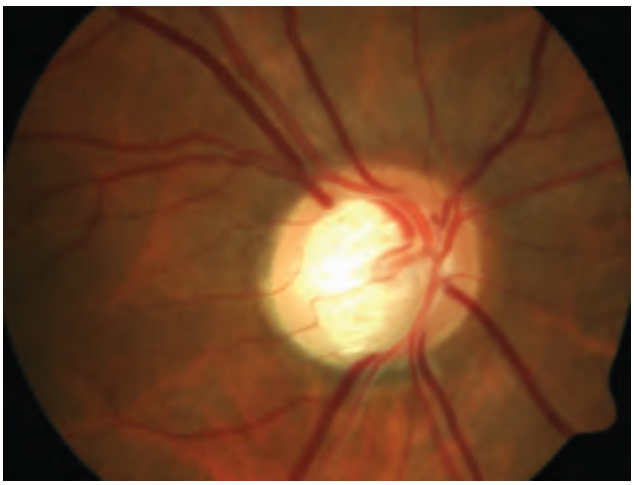

Figure 11B

From page 7

As we are serious about detecting glaucoma at a stage when we can prevent visual disability, we have added the following rule:

- Unless proved otherwise, ALL optic discs have glaucomatous changes (and all angles are closed).

The rule emphasises that to detect glaucoma, we must have a high index of suspicion and examine ALL patients carefully.

The optic disc should be examined at every visit. Depending on the course of the disease, documentation should be performed every six to 12 months.

## Imaging techniques for examining optic disc

The optic nerve and or nerve fibre layer imaging techniques include the Heidelberg Retinal Tomograph (HRT III), Optical Coherence Tomography (OCT) and the Nerve Fiber Layer Analyzer (GDx VCC). The World Glaucoma Association consensus on imaging states that these instruments lack the sensitivity and specificity for routine clinical use but that in the hands of experts, they may provide valuable clinical information.[4] These instruments can help corroborate our suspicions and help support our diagnosis but are not required for routine clinical diagnosis. If we have to make a choice between automated perimetery or one of the imaging techniques for diagnosis, we suggest the automated perimeter. We do feel that the imaging devices may have a potential for documenting and detecting change and have a major role to play in this important area.[30,31] The clinical application for such data for diagnosis and progression requires a basic knowledge of how to apply the published sensitivity, specificity and likelihood ratios.[10]

**Their use for screening and in lieu of clinical examination is not based on clinical principle or evidence.**

## Visual field

Glaucoma is a potentially blinding disease because it causes functional defects in the visual field. In the presence of a repeatable defect that correlates with the disc and NFL changes, the diagnosis and management decisions become clearer. The detection of field defects and their progression (or stability) is therefore extremely important in glaucoma management. Like any other test, a visual field is obtained ONLY if there is a suspicion of disease. A field 'defect' in a person whose examination is normal is likely to be a false positive. If there is no suspicion of disease, do not obtain fields or any other test, including imaging.

- The current gold standard for examination of the visual fields is full threshold automated perimetry. Automated perimetry has a learning curve and it is best not to rely on the first two fields. It is best to use the same model of perimeter for the same patient; a perimeter that has a validated progression program like the visual field index is desirable.[32,33]
- The perimetry printout is analysed systematically in zones.[34] A field with an early glaucomatous defect is shown in Figure 15.
- The field defects in glaucoma are usually localised. A localised defect will show up in both the total deviation and pattern deviation plots. A generalised depression is more characteristic of anterior segment related causes, like cataract affecting the visual field. In such cases the defects are limited to the total deviation plot (Figure 16).

**A warning: visual fields like any other test must NEVER be interpreted in isolation.**

- The field should usually correlate with structural changes in the optic disc and NFL.
- If there is definite structural damage but the field is normal, repeat the visual field.
- If there is a visual field defect but no correlating structural damage, examine the disc again. If there is still no correlating structural damage, re-examine the disc using a contact lens to obtain a good stereoscopic view.

While the gold standard for perimetry is a full threshold examination, in the presence of other signs of glaucoma the presence of a repeatable defect in the 20-1 screening mode of a frequency doubling perimeter (FDP) (Figure 17) is sufficient evidence for a visual field defect due to glaucoma.[35-36] While it is not in the remit of this article, we feel that the demonstration of a repeatable functional defect is usually required before incisional surgery is considered. This can be done even with a Bjerrum screen but an automated simple machine like the FDP is more likely to be used. The FDP is also capable of a full threshold test but has no follow-up capability.

The initial evaluation of a patient may or may not lead to a confirmed diagnosis or a decision to treat. Follow-up of suspects

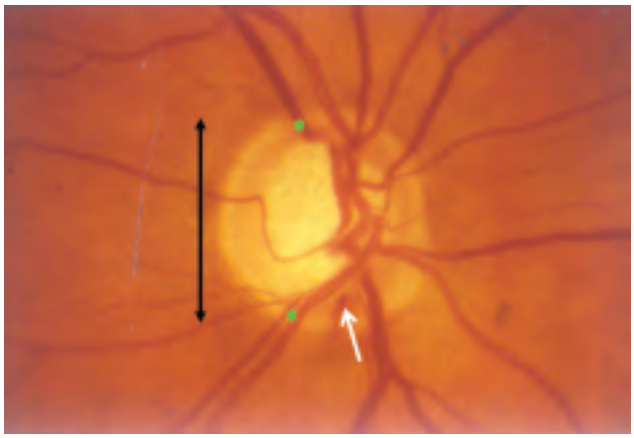

Figure 12

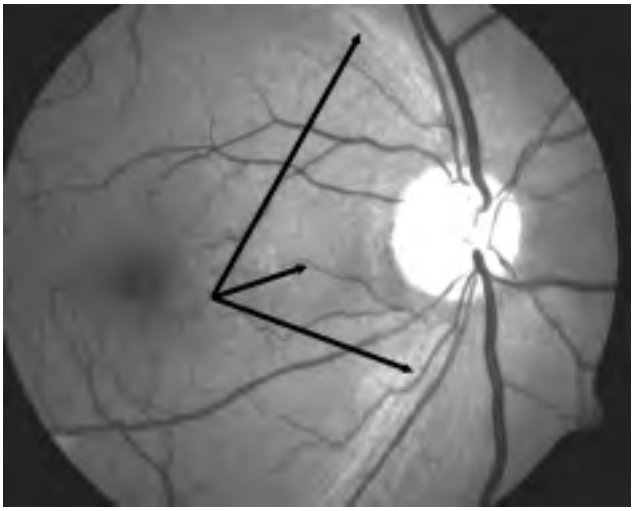

Figure 13

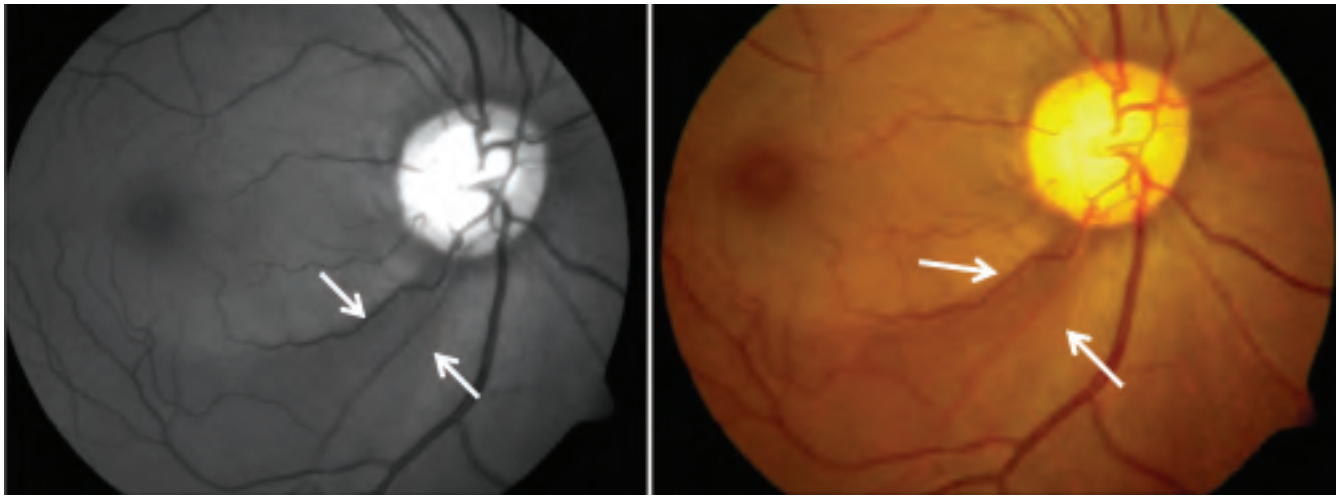

**Figure 14**

and patients at appropriate intervals for detection of progression based on optic disc examination, imaging and serial visual fields is crucial to further decision-making. It is important to obtain baseline documentation of the optic disc and visual fields early in the course of the disease. Baseline fields should exclude the learning curve.

## When do we suspect glaucoma?

- Family history of glaucoma
- Raised IOP (more than 22 mmHg)
- Non-visibility of the trabecular meshwork on goniosocpy
- A 'suspicious' optic disc (anything that looks out of the ordinary or outside the normal range or a rim to disc ratio < 0.1:1)
- Retinal nerve fibre layer defect
- Optic disc haemorrhage
- High myopia
- The prevalence of glaucoma after the age of 60 years is high enough (five per cent) to suspect it on ALL persons older than this age.
- If we want to detect glaucoma early and prevent blindness, ALL those who present to an eye-care professional are glaucoma suspects and should AT LEAST undergo a comprehensive eye examination. This will not only detect glaucoma but also most potentially blinding conditions. Ancilliary testing is useful only if indicated after such an examination.

The work-up of a glaucoma suspect is shown in Figures 18 and 19.

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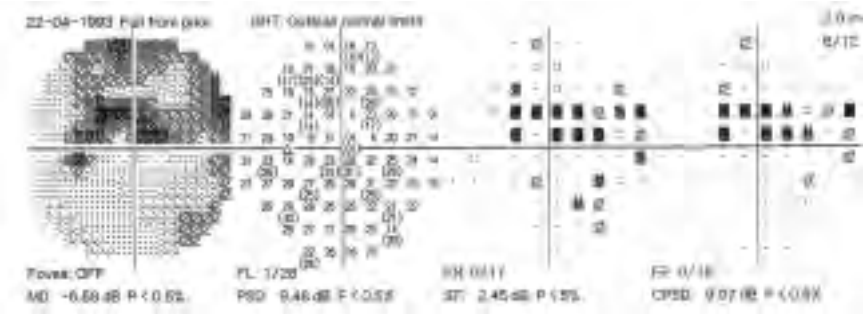

**Figure 15**

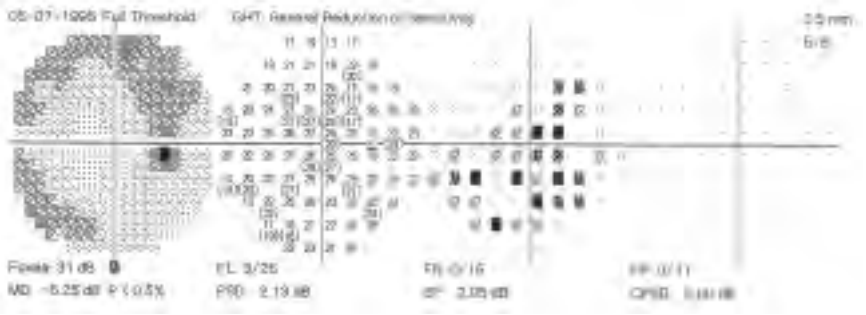

**Figure 16**

From page 9


## Summary

The diagnosis of established glaucoma at a stage where treatment can prevent blindness involves the strategy of case detection. The eye-care practitioner should aim to detect all potentially serious ophthalmic pathology, including glaucoma. This requires a comprehensive eye examination including slitlamp, IOP, gonioscopy and a dilated disc and fundus examination on all clinic patients. Automated perimetry should be obtained for all suspects and programs like a visual field index (VFI) should be used for follow-up. FDP is an alternative to confirm the presence of glaucomatous visual field defects. Screening for glaucoma using imaging or visual fields prior to a clinical examination can have too many false positives and negatives and is not recommended.



### Acknowledgement

The authors are grateful to the *Indian Journal of Ophthalmology* for permission to reproduce photographs and text.


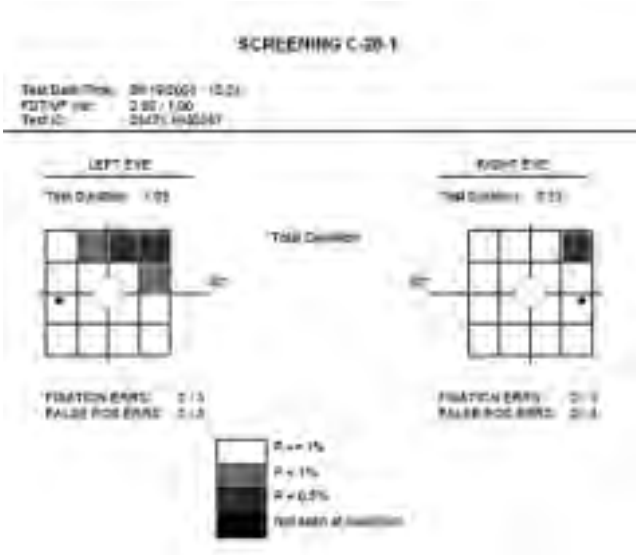


**Figure 17**

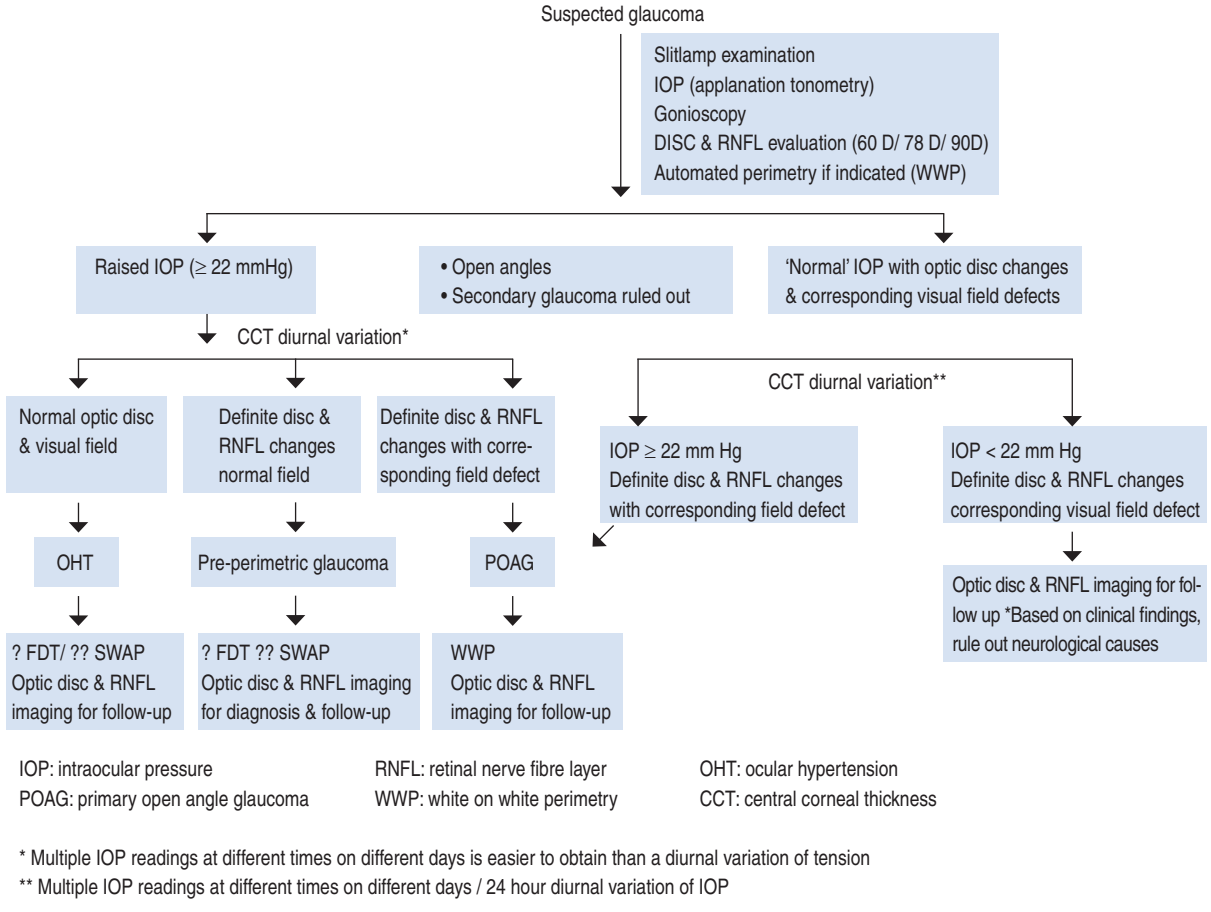


**Figure 18**

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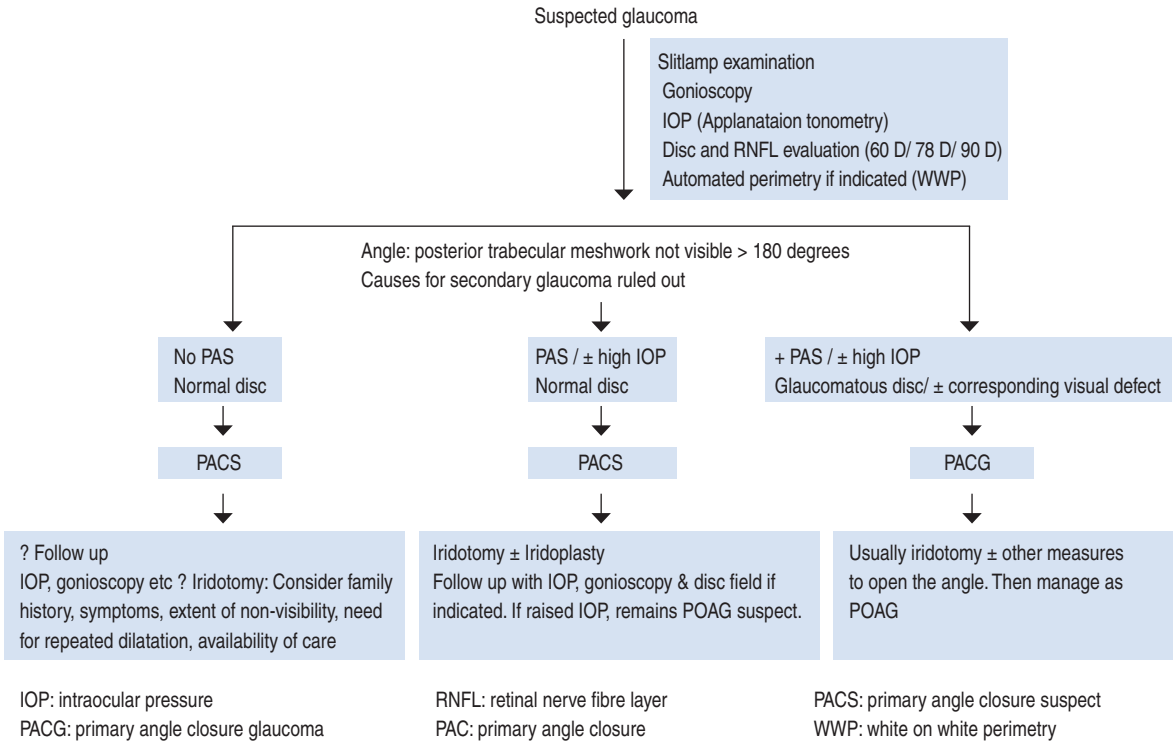


**Figure 19**

# The long march of the

**David M Cockburn**
DSc MScOptom FAAO

**The 3,000-year-old network of tracks and rough roads that formed the Silk Road became the first permanent land trade link between the eastern and western world. Across it flowed merchants, soldiers, nomads and refugees of diverse ethnic origin and speaking numerous languages. The Silk Road carried much more than silk; along it flowed slaves, precious goods and produce, a human tide that with its ebb and flow carried and scattered its genome carelessly in passing, or through formation of permanent relationships with people along the road. These genetic packages in turn carried the blueprints for specific disease risks, both outward and homeward. Among the many were the genetic markers for Behcet's disease and glaucoma in its several forms.**

Glaucoma is a serious problem in all parts of the globe, affecting all races with a current world prevalence that approximates 67 million people affected[1] and is expected to rise with increases in longevity and world population. Glaucoma is the cause of 12 per cent of the world's blindness and considerable loss of vision short of blindness.[2] We know a great deal about the prevalence and morphology of glaucoma in the western world but about our near neighbours, most Australians at least know very little.

World-wide prevalence of primary open angle glaucoma (POAG) is about two per cent. In western countries, primary open angle glaucoma is about five times more prevalent than primary angle closure glaucoma (PACG), in contrast to most Asian countries where PACG is reported to be as or more common than POAG.[3]

We must use these prevalence estimates with caution. The symptoms of acute PACG are usually painful, with rapid loss of vision causing sufferers to be well aware of the need for treatment. Even the sub-acute form of PACG can have most unpleasant and frightening symptoms. On the other hand, POAG is almost symptomless until the visual field is extensively damaged, so that people in low-income areas of Asia with this form of the disease are unlikely to present for diagnosis and treatment. This may cause selection bias toward overestimating the POAG relative prevalence, especially when derived from hospital attendance-based reports, and possibly although to a lesser extent in population-based surveys.

Differences in relative proportions of POAG and PACG have implications for the routine investigations that optometrists and ophthalmologists employ. We tend to search diligently for the conditions we believe to be common, allowing the uncommon to emerge unsought. Migration to Australia has increased the variability of its population genome to the point where 60 per cent of Australians have a mixed ethnic genome and 20 per cent have at least four ethnically different ancestors.[4] This migration lately includes a large and growing proportion of people of Asian descent. Mixed race marriages are no longer unusual in Australia, so the rapidly changing demographic of the Australian population presents new challenges that will be accelerated by the fact that migration has outstripped natural births in Australia and the demographics of glaucoma risk may be about to change. We should be aware.

At about three per cent, POAG presently is the most common form of glaucoma in Australians of Anglo-Saxon descent.[5] Angle closure glaucoma is relatively uncommon, having a prevalence of about 0.4 per cent of all glaucoma cases in European/Anglo-Saxon people. In terms of worldwide prevalence, PACG affects a staggering 15 million people.[6] In people of Asian descent, PACG is generally the more common, although varying in prevalence in different parts of Asia. These figures are very likely to be distorted by differences in definition, diagnostic methods and selection of samples.

It is estimated that 3.9 million people worldwide will be blinded due to POAG by 2010 and that this figure will rise to 5.3 million by 2020.[7] The majority of those affected will be living in Asia simply because of its high population density. The presentation of PACG and its precursers is just as variable as that of open angle glaucoma, but has one clearly defined risk factor consisting of a narrow or occludable anterior chamber angle. Narrowness of the angle is an anatomical feature of the eyes of people of Asian descent. What is the extent

# glaucoma and Silk Road

Glaucoma can manifest in our Asian neighbours in various ways and for different reasons

of the problem in our near neighbours who now provide a significant proportion of our visitors and migrants?

## Glaucoma in Asian neighbours

About 60 per cent of all world blindness occurs in Asian countries.[8] Although many Asian countries have optometry and ophthalmology schools and services that match the most advanced in the world, access to these services is variable and remains difficult for the vast majority of Asian people, especially those living in rural areas, remote from good quality care. There is also the problem of affordability of these services, regardless of whether they are met privately or financed by the state, in which case they are in competition for resources with other perhaps more damaging diseases and of course other essential state projects. This article describes the relative prevalence of forms of glaucoma in a few of our neighbours, selected for proximity, sources of immigration or the presence of interesting features.

## Mongolia

Mongolia is entirely land-locked, bordered to the south by China with its northern border shared with Russia. It has a population of about 5.8 million. In the 13th Century its empire dominated all of China and its legendary hordes cascaded over most of Asia well into eastern Europe as far as Poland. It was the important major terminal of the Silk Road with its traders and warriers extending their cultural, religious and genetic influence along its considerable lengh. As a consequence, much of the population of present day Asia and western Europe has a genome, part of which is derived from the Mongol people. A narrow anterior chamber angle is a feature of the Mongolian ocular anatomy, carrying with it a relatively high risk of angle closure glaucoma.[9] The prevalence of chronic and acute PACG in Mongolian people is at least equal to and possibly higher than in the Inuit people, their cousins of the cold northern polar regions.

Health care in Mongolia has improved considerably in the past two decades. It now has a ratio of 3.9 physicians to 1,000 people, with women forming more than 80 per cent of the largely Russian-trained physicians. Glaucoma accounts for around 35 per cent of the blindness in Mongolia and the angle closure form causes the great majority of these cases.[10] Information regarding ophthalmology and optometry services is sparse.

## China

In Western and African communities, POAG is five times more common than PACG but these figures are reversed in China,[11] where it is estimated that there will be 15.7 million Chinese people with PACG in 2010. Angle closure glaucoma is the most common form, with an estimate as high as 72 per cent of all glaucomas. It is responsible for 94 per cent of glaucoma-related bilateral blindness in China.[12]

In one population-based glaucoma screening there were 13 new cases detected, comprising 11 cases of angle closure and one each of open angle and secondary glaucoma.[13] In addition, it is estimated that more than 90 million people in China already have significant angle closure although not yet complete, and a further 28 million are at risk by having 'occludable' angles.[11]

China's health services have undergone remarkable changes in the past decades but many rural areas still lack access to modern eye-care services. Some optometry and ophthalmology schools are of very high standard, as for example the Wenzhou School of Optometry and Ophthalmology that has 913 students; however, there is considerable diversity in optometry training programs from the comprehensive curriculum of the Wenzhou school, to short courses that equip graduates only for dispensing.[14] This diversity translates into a considerable variability of eye services in different regions of China.

In Australia, optometrists need to be specially vigilent in detecting angle closure and occludable anterior chamber angles in patients of Chinese origin. Gonioscopy should be a high priority in optometric teaching and clinical practice as it is the crucial diagnostic skill.

## India

India has a huge population of 1.15 billion people, second in size only to China and expected to outstrip that nation by 2030. There has been a three-fold increase in population in India since 1947, coinciding with a rapid period of economic development. There were 11,286 migrants from India to Australia in 2010, an increase of 20 per cent over the previous year, making India our third-largest source of migrants behind Britain and New Zealand.

In India, the acute and chronic forms of angle closure glaucoma are over 60 per cent of all primary glaucomas and are responsible for a large proportion of glaucoma related blindness.[15] Very high standard ophthalmology services are increasingly available in India; however, they are not accessible to the majority of the

**Continued page 14**

# Glaucoma and the Silk Road

**From page 13**

population due to economic circumstances, distribution of these services and problems associated with adherence to treatment.[16]

## Pakistan

Pakistan has a population of 170 million with high racial diversity that differs considerably from that of India. It includes significant numbers of people of Caucasian, Aryan, African and Mongol heritage, which suggests that there is also likely to be variation in the prevalence of glaucoma and its type in different communities.[17] Life expectancy in Pakistan is low in comparison with western countries, being 63 years for females and 62 for males; a factor that may skew glaucoma statistics because of the greater likelyhood of onset of POAG in later life and the relatively benign late appearance of symptoms of that form.

A study of Pakistani hospital-attending patients having glaucoma found that 55.3 per cent of these patients had PACG and 44.7 per cent had POAG. The authors also noted that 38 per cent of those people previously diagnosed with POAG were in fact suffering from PACG.[18] Secondary glaucoma is a major cause of blindness in Pakistan and accounts for 35 per cent of glaucoma hospital admissions. These findings are in contrast with figures on glaucoma surgery carried out in all 18 tertiary care hospitals in Pakistan that indicated that 37.6 per cent were for POAG, 35 per cent secondary glaucoma, and 18.2 per cent for PACG.[19] This strange anomaly invites attention.

Another glaucoma clinic survey[20] carried out in Pakistan suggests that normal tension glaucoma may be a problem among these people, with the finding that 22 per cent of the open angle glaucoma cases diagnosed were due to this form of the disease. This is a similar prevalence to that found in many western countries and in Japan, although much higher than found in other Asian countries. Is it possible that definition and sampling bias may play a large covert part in these interesting differences?

## Indonesia

Indonesia, our nearest neighbour, has 14 main categories of ethnic origins and 88 sub-categories spread over 17,508 inhabited islands. This must make it one of the world's most ethnically diverse nations and as a consequence bring a wide variation of genetically related disease risk factors. For these reasons, statistics relating to prevalence of glaucoma in a single area should be generalised to the Indonesian nation with caution, even when a large cohort is screened.

Glaucoma is stated to be the second major cause of blindness in Indonesia, based chiefly on hospital records with overall glaucoma prevalence estimates varying between 0.4 per cent and 1.6 per cent. Together, acute and chronic PACG are stated to be the most common presenting form of glaucoma in Indonesia. The onset of PACG tends to occur in Indonesians at a younger age than in Caucasians; the prevalence rises with increasing age in a fashion similar to that of patients in western societies.[21] The availability of medical care for Indonesians varies for reasons of affordability and geographical remoteness, which may also distort the figures relating to the prevalence of the glaucomas and the classification of the disease.

In summary, the impact of glaucoma in Indonesia is of concern, with PACG posing the most urgent target for diagnosis and management, particularly in areas more remote from high quality eye services. The overall and relative prevalence of POAG and number of people having risk factors for both forms of glaucoma are probably underestimated.

## Japan

Glaucoma is a leading cause of visual impairment in Japan where it has a prevalence of five per cent for all forms combined. There is an expected increase of 23 per cent during the next 20 years, in line with a predicted increase in an already average long life expectancy. The prevalence of PACG is estimated to be 0.6 per cent, a figure for PACG well below that of most other Asian countries and more similar to European findings.[22] It is tempting to ascribe the low prevalence of PACG to Japan's lengthy isoation from the Asian mainland since the last Ice Age about 11,000 BC, which would have mostly prevented the introduction of the Mogol genome. Perhaps the Kamikasi, or divine wind, that saved Japan from the Mogol invasion in the latter part of the 13th Century also saved the Japanese people from introduction of the genetic markers for PACG. This of course conflicts with the historic long march along the Silk Road of Behcet's disease that is believed to have had its origin in a variation in a Japanese human leucoscyte antigen. This rather nasty condition that includes painful and destructive uveitis reached Mediterranian countries where it has relatively high prevalence today and is seen also in Australians with Mediterranian forebearers.

An important finding in Japan is that 92 per cent of the people with POAG aged ≥ 40 had IOP less than 21 mmHg. The average pressure in the group was 15.4 mmHg. Tonometry clearly has very poor sensitivity for open angle glaucoma in Japanese people. Just to complicate the diagnosis further, a study[23] found no significant difference in central corneal thickness in Japanese subjects with glaucoma compared to non-glaucomatous Japanese.

Despite having universal access to high quality medical services, there remains a relatively high rate of undetected glaucoma among Japanese citizens.[24] This fact emphasises a need to improve detection of early glaucoma in Japan with normal tension glaucoma being a target of special importance. The simple solution would be to train the people performing refraction to detect glaucoma and other eye diseases with good sensitivity or, in other words, to create a modern standard of optometric education and independent practice in Japan.

## Nepal

Nepal also lies on the ancient Silk Road linking India, Burma and China that has delivered a highly diverse ethnic population that now approximates 28 million people. Its public sector spending on health is very low by Western standards,[25] making modern medical services unavailable to the vast majority of Nepalese. Numerous private sector medical schools have opened recently and although numbers of doctors graduating have increased, there continues to be a doctor shortage in rural areas.[26] There are 150 ophthalmologists and approximately 300 'eye care assistants' in Nepal, including some optomerists trained in Western countries. An optometry school now provides a four-year course similar to those of Western countries.

There is very little data available for the prevalence of glaucoma in Nepal. A survey[27] conducted in 1980-1981 found that glaucoma accounted for 3.2 per cent of all bilateral blindness and the number of cases of sub-acute angle closure and early to moderately advanced open angle glaucoma is conceivably much greater

than those who are bilaterally blind from the disease. Contrary to most other Asian based studies, open angle glaucoma was more commonly diagnosed than the angle closure form. This interesting finding may be the result of the high degree of ethnic diversity of the country. A study protocol has been arranged to perhaps provide a more definitive estimate of the prevalence and type of glaucoma in Nepal.[28]

## Vietnam

Vietnam has China, Laos and Cambodia as border countries. It has a population of 82 million comprising 54 different ethnic groups. Vietnam is the second largest south-east Asian populaton after Indonesia, squeezed into an area similar in size to Italy. It has one of the the highest population densities in the world.

Health services are rapidly improving, with modern medical management available in larger cities, but accessability remains a problem in rural areas and for the poor. The relatively low per capita annual health care expenditure is primarily aimed at disease eradication such as HIV-AIDS and treatment of disease in younger people. Information relating to the prevalence of eye disease in Vietnam is scarce.

As in many Asian countries, cataract is the leading cause of blindness at 50 per cent and with secondary glaucoma next at 16 per cent followed by PACG at five per cent.[29] It is estimated that 607,814 Vietnamese people have glaucoma of some form, with the high proporton of PACG being similar to most other Asian countries.[30]

## Conclusion

Our Asian neighbours have glaucoma with approximately the same prevalence as in Australia but with important differences. The prevalence of PACG and risk factors for angle closure appear to be higher in most Asian communities with the possible exception of Nepal and Japan. In people of Japanese and Pakistani heritage, the prevalence of normal tension glaucoma is much higher, with PACG having roughly the same low prevalence as in Western countries. The statistics for the prevalence of POAG and normal tension glaucoma in Asia may be unreliable because of the lack of notable symptoms and slow progress that may be cut short by the present reduced life expectancy of many Asian communities. Australian immigration and tourist policies ensure that more Asian people will visit us and become citizens of Australia, so our clinical management should reflect the differences in glaucoma presentation in these people. The human traffic of the Silk Road has been replaced by jumbo jets and its commerce by container ships, but the potpourri of our genetic code continues to change.

The problem of glaucoma in Asia could be greatly relieved by creating more optometrists having similar training to those of the Western world, thereby improving early diagnosis opportunities and facilitating rapid referral for treatment. In Australia, optometrists should bear in mind the special risk factors that can lead to angle closure in patients of Asian heritage. Gonioscopy is an essential technique for this investigation.

> **Asian people will visit us and become citizens of Australia, so our clinical management should reflect the differences in glaucoma presentation in these people.**


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# Fixed combination drops for glaucoma

**Dr Catherine Green**
MBChB FRANZCO MMedSc

**Glaucoma is a progressive optic neuropathy for which the only currently proven treatment is lowering of the intraocular pressure (IOP).**[1] **The aims of treatment are to preserve the patient's quality of life, preserve visual function and minimise the side-effects of treatment.**

The IOP should be reduced to a level at which no further optic nerve damage occurs. This is called the target IOP and it needs to be individualised for each patient, taking into account the IOP level at which optic nerve damage occurred, the extent and rate of progression of the disease, the patient's age and expected life span, family history, and the costs and risks of treatment.

Treatment modalities for glaucoma include topical medication (IOP lowering eye-drops), laser therapy and incisional surgery. For many patients topical treatment is sufficient to control their IOP, but many will require more than one medication to achieve a sufficiently low pressure. In the Ocular Hypertension Treatment Study, 40 per cent of patients were being treated with two or more medications after five years, and nine per cent were using three or more medications.[2] This was to achieve a modest IOP reduction target of 20 per cent; the percentage would probably have been higher with more aggressive treatment goals.

The challenges for the patient being treated for glaucoma are that, as the disease is usually asymptomatic, there is little perceived therapeutic effect from the medication, as well as the cost and the inconvenience of using a medication over the long term. Compliance and adherence with the treatment regimen are important factors affecting its success.[3]

Factors affecting compliance include:

- number of medications
- number of doses per day
- visual acuity
- the patient's knowledge about glaucoma
- tolerability of the medication.

Patel and Spaeth[3] found that only 49 per cent of patients were compliant when they were required to use one medication a day; this dropped to just 32 per cent for those prescribed two medications.

Besides a decrease in the percentage of patients who are compliant when multiple drops are prescribed, the efficacy of the treatment is reduced by the 'wash-out effect' that occurs when drops are dosed without sufficient time between different medications. Eye-care practitioners should advise patients to wait at least five minutes between doses. Chrai, Makoid at al[4] demonstrated that if a patient waits only 30 seconds between eye-drops, up to 45 per cent of the first medication can be washed out by the second. With an interval of two minutes, 17 per cent of the first medication is washed out, but after five minutes there is no loss of the first drop.

In the past few years, numerous fixed combination antiglaucoma drops have become available; these contain two medications in a single bottle. The fixed combination products currently available in Australia are listed in Table 1.

## Advantages

Fixed combinations offer several advantages for patients:

- increased convenience of dosing because of the need for fewer bottles and for some drops, less frequent dosing
- potential for increased adherence and persistence with medication[5]
- no wash-out effect
- reduced exposure to preservatives
- cost savings through not having to purchase multiple drops.

## Efficacy

For the fixed combinations to have been approved for use in Australia, studies needed to demonstrate that they were at least as effective as the concomitant use of the component drugs. A thorough overview of the results of comparative studies is included in a review article by Higginbotham.[6]

## Disadvantages

The disadvantages of fixed combinations include:

| Proprietary name | Contents | Dosing |
|---|---|---|
| Azarga | timolol 0.5% and brinzolamide 1% | twice a day |
| Cosopt | timolol 0.5% and dorzolamide 2% | twice a day |
| Combigan | timolol 0.5% and brimonidine 0.2% | twice a day |
| DuoTrav | timolol 0.5% and travoprost 0.004% | once a day |
| Ganfort | timolol 0.5% and bimatoprost 0.003% | once a day |
| Xalacom | timolol 0.5% and latanoprost 0.005% | once a day |

**Table 1. Fixed combination drops available in Australia**

The benefits are simplicity, convenience, adherence and potential savings in cost

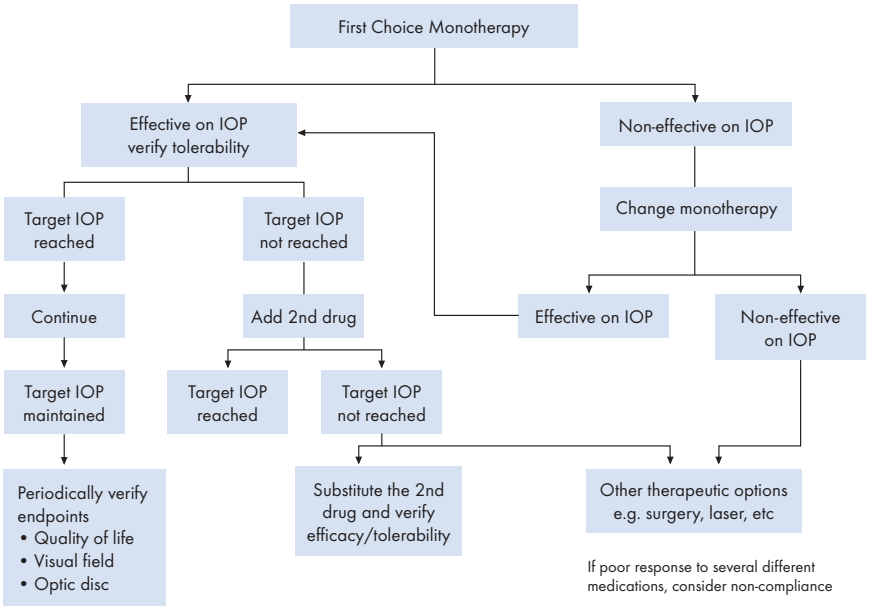


**Figure 1. Treatment algorithm: European Glaucoma Guidelines[7]**

- it is not possible to change a drug concentration or dosing schedule for one component medication independently of the other
- if there is an adverse reaction, it may be more difficult to identify the culprit component
- there may be a temptation to use a fixed combination as first-line treatment, without a trial of components individually
- there is a potential to 'double up'—the inadvertent prescribing of one of the component drugs in addition to the fixed combination.

## When to use fixed combinations

The guidelines for initiating medical therapy should be adhered to (Figure 1).

The aim of treatment should be to achieve the patient's target IOP with the fewest and best tolerated medications. If a patient has responded to a drug that has been introduced but the IOP is not at target, another drop may be added. If the two drugs to be used are available in a fixed combination, consider prescribing the medication in this form. Fixed combinations should also be considered in patients who have been using multiple medications for some time, but would benefit from rationalisation of their treatment regimen to improve convenience, compliance and adherence. As with any change in medication, review of the patient should be arranged to check efficacy and tolerability and to reinforce compliance.

## Side-effects

The side-effects of the fixed combinations are the same as those of their individual components. Prescribers should be aware that all the fixed combinations available in Australia at present contain 0.5% timolol, and should be alert to the contraindications and side-effects of topical beta blockers including shortness of breath, bradycardia and arrhythmias, fatigue, depression and erectile dysfunction. For some fixed combinations including Combigan[8] and Ganfort,[9] the fixed combinations showed improved tolerability compared with individual components, with reduced hyperaemia and allergy rates in some patients.

## Conclusion

Fixed combination glaucoma drops offer several advantages for patients on glaucoma treatment including simplification of the treatment regimen, increased convenience and improved adherence, as well as potential cost savings. Eye-care practitioners prescribing fixed combinations need to be familiar with the component medications of any fixed combination they prescribe and be vigilant for side-effects of the individual components. Treatment decisions in glaucoma management need to be implemented in a logical, step-wise fashion and the response to treatment changes assessed. When patients are on several medications or have had multiple changes to their treatment, it is important to check that the patient is using drops as prescribed and that there has not been an inadvertent duplication of medication.


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# Imaging at your

**Michael Yapp**
Principal Staff Optometrist
**David Pye**
Associate Professor
Centre for Eye Health

## Case report

**A 53-year-old Asian female was referred to the Centre for Eye Health (CFEH) for a glaucoma assessment as a result of a significant family history of glaucoma, including father, paternal aunt and paternal uncle. Her ocular history also included blunt trauma to both eyes from a car accident 15 years earlier resulting in an inferior orbital wall blow-out fracture to the right eye, which was surgically repaired, and a permanent ecchymosis of the left superior eyelid. The patient's glaucoma risk profile also included myopia, with the left eye having a significantly higher refractive error than the right eye.**

Monitored closely over previous years, her Humphrey visual fields were consistently clear; however, a recent Matrix FDT showed a possible inferior nasal arcuate defect in the right eye and a superior nasal step in the left eye, prompting referral to CFEH for further investigation.

On examination by clinical staff at the centre, the patient's pupil reactions were normal, and gonioscopy showed angles open to the ciliary body in all sectors and inferior angle recession in both eyes. Intraocular pressures (IOP) were 16.5 mmHg in the right eye and 16 mmHg in the left eye at 11 am. Pentacam imaging showed regular corneal topography, and pachymetry in the centre of the pupil was 516 µm in the right eye and 527 µm in the left eye.

A repeat Matrix assessment (Figure 1) was essentially clear for the right eye but, consistent with the previous result, showed a superior nasal step in the left eye.

Funduscopy, HRT3 Moorfields regression analysis, GDx Pro and Cirrus optical coherence topography (OCT) scans were performed (Figures 2, 3, 4 and 5). The results collectively showed:

- glial tissue inferiorly on the right optic nerve head, with mild beta peripapillary atrophy (PPA) temporal to the left eye.
- a notch in the inferior neuro-retinal rim (NRR) of the left eye, with an associated thinning of the retinal nerve fibre layer (RNFL).
- a sloped and relatively thin superior NRR in both eyes, with a relative reduction of the superior RNFL in the right eye.

The structural and functional assessments correlated well, suggesting glaucomatous damage to the left neural tissue with a probable angle recession component.

It is thought that IOP elevation with angle recession is secondary to trabecular meshwork damage, rather than as a direct result

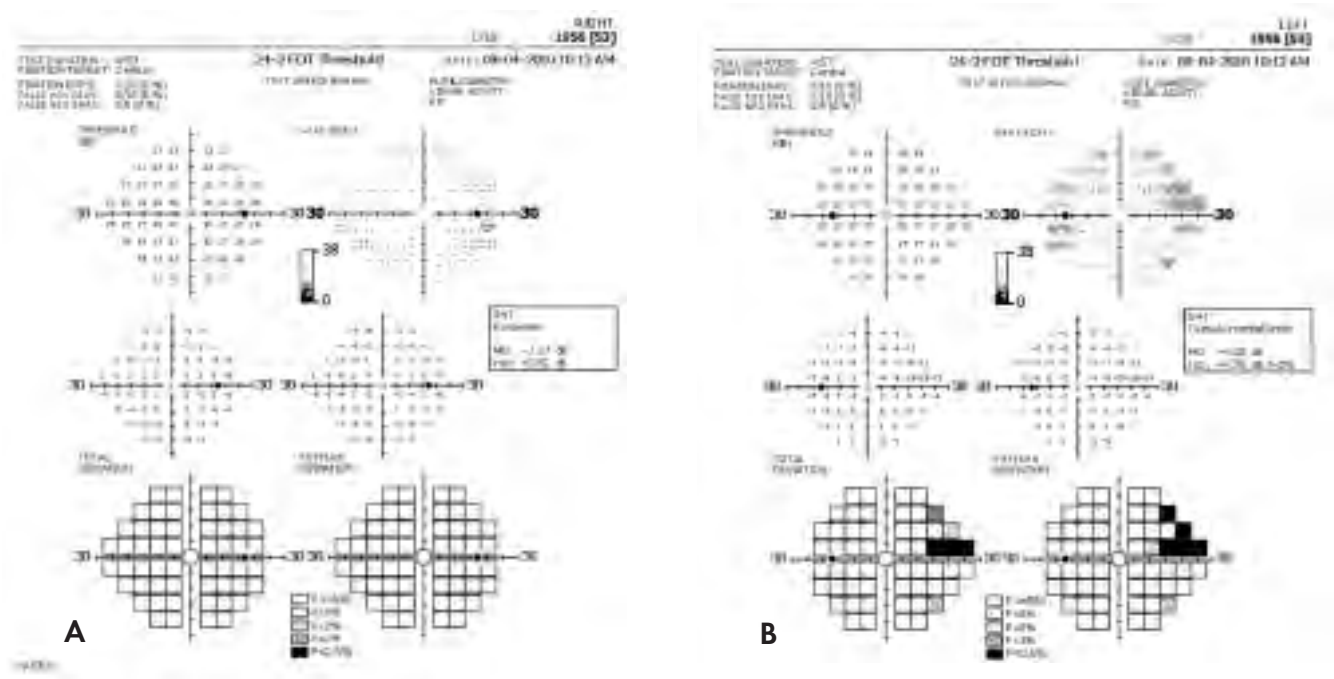


**Figure 1. Matrix 24-2 test results. A: right eye, B: left eye**

# service

The cause of glaucoma remains elusive but imaging devices can assist diagnosis and management

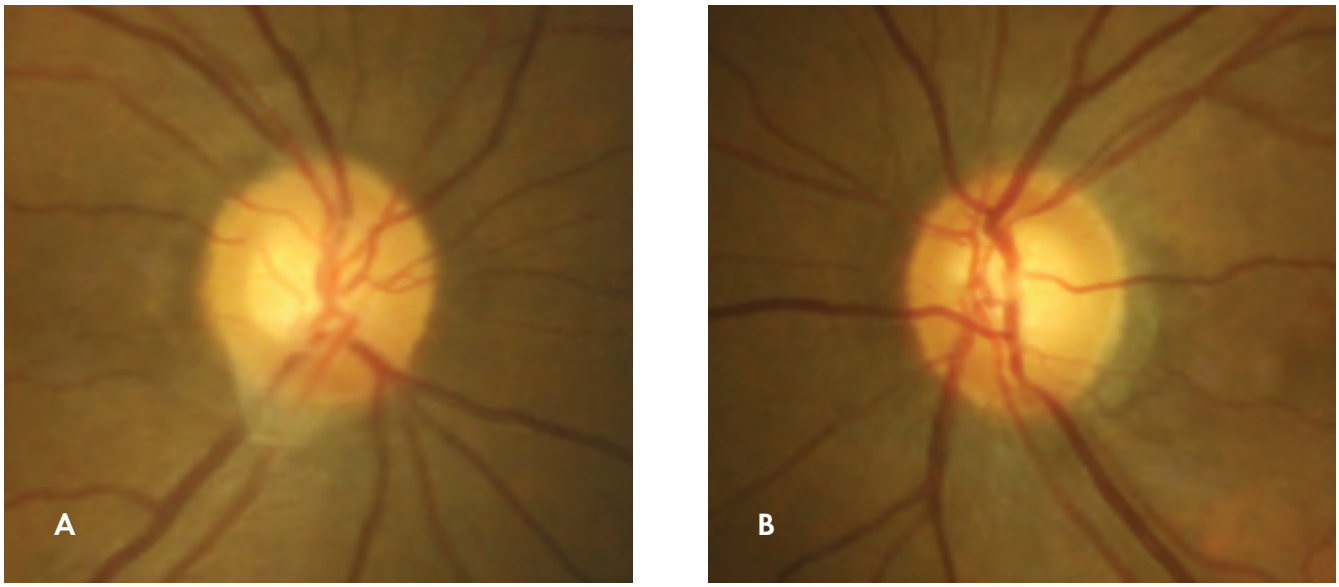


**Figure 2. Optic nerve head photographs. A: right eye, B: left eye**

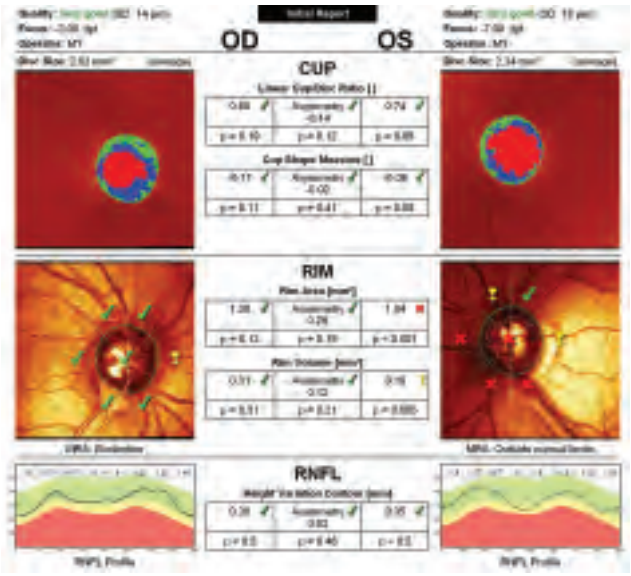


**Figure 3. HRT3 results**

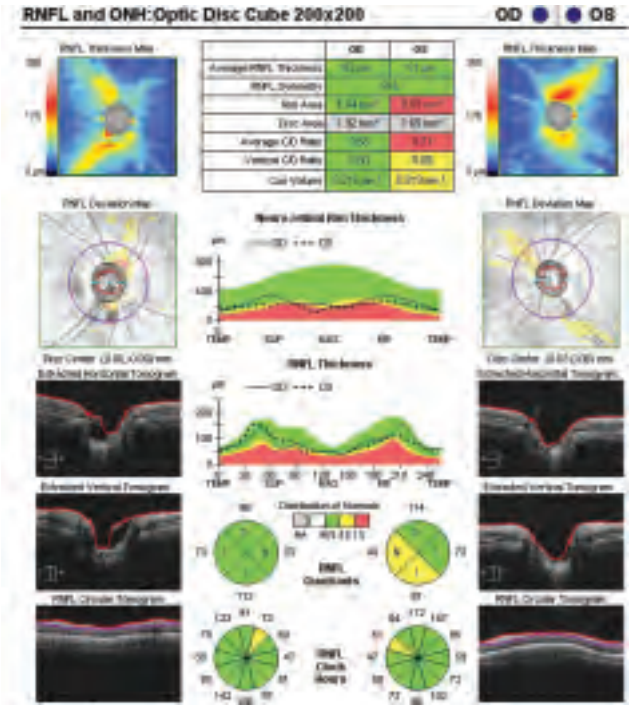


**Figure 4. Zeiss Cirrus OCT results**

# Imaging at your service

**From page 19**

of the recession itself.[1] About six to nine per cent of patients develop glaucoma within 10 years of developing angle recession[1] and the risk is proportionate to the extent of angle recession.

The patient was referred for ophthalmological assessment and selective laser trabeculoplasty (SLT) was subsequently performed on the left eye. SLT uses a double frequency Nd:YAG laser and a green wavelength light (532 nm). The pulse duration is limited to three nanoseconds and fixed at 400 µm in diameter.[2] The most commonly accepted theory is that the SLT mechanism releases cytokines, stimulating macrophage recruitment, to remodel the extracellular matrix of the trabecular meshwork.[3]

The SLT technique has been shown to cause minimal mechanical damage to the trabecular meshwork, as opposed to coagulative necrosis resulting from argon laser trabeculoplasty (ALT).[4]

Studies have shown that ALT treatment for angle recession glaucoma is ineffective,[5] although no studies on the effectiveness of SLT in angle recession were found. Given this particular patient's historical risk profile, it is likely that the angle recession is only one component of her optic neuropathy. A recent 12-month retrospective study showed that SLT effectively controlled IOP in normal tension glaucoma.[6] As a result and given the patient's relatively young age, SLT was selected in an attempt to minimise her long-term dependence on topical medication. The treating ophthalmologist reported a 25 per cent reduction in IOP after performing SLT on the left eye.

The patient returned to CFEH for repeat imaging 12 months after the SLT and subsequent follow-up. IOPs measured 16 mmHg in the right eye and 15 mmHg in the left eye. The GDx Pro, Cirrus OCT and topographical change analysis available with the HRT3 all require a minimum of at least three examinations to produce a change analysis so it is difficult to distinguish instrument variability from real change at a second visit. However, with the HRT3, stereometric parameter analysis can be calculated after two visits and in this case the results suggested a slight worsening in the right eye but a slight improvement in the left eye (Figure 6). While this result is most likely to be due to inter-test reliability of the instrument, there is published evidence of reversed cupping after reduced IOP.[7,8,9] A third assessment will be needed to further document and establish any changes.

## Conclusion

Glaucoma is a complex condition with multiple aetiologies and associations. A definitive cause is proving elusive for the condition in general, and with individual cases such as this. While stereoscopic evaluation of the optic nerve is still considered the gold standard for assessing the neuro-retinal rim, imaging devices such as those available at CFEH can provide additional information to assist with the initial diagnosis and for monitoring subsequent changes.


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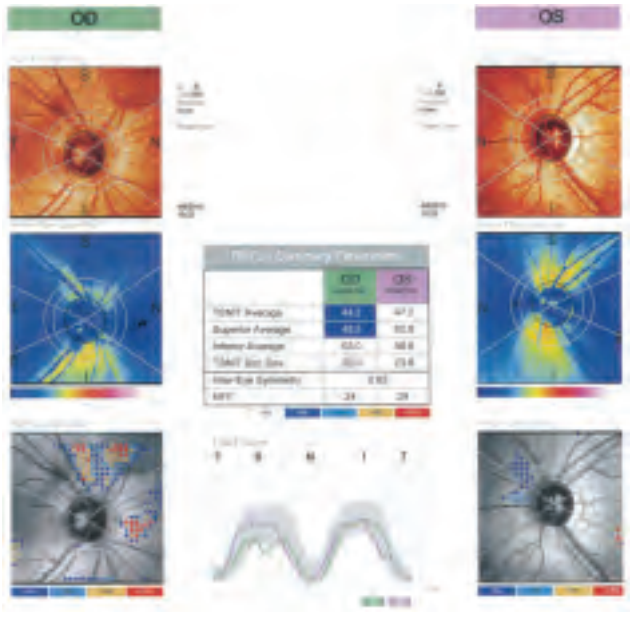


**Figure 5. GDx PRO results**

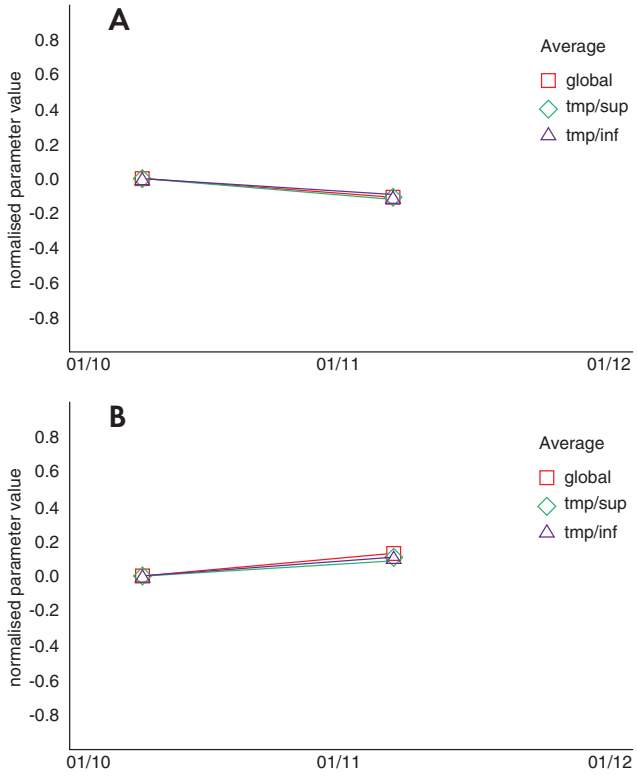


**Figure 6. HRT trend analysis. A: right eye, B: left eye**

# “The good news is we can now do more about glaucoma’s low rate of diagnosis.”

**To access these services, visit cfeh.com.au or call 1300 421 960**

*Michael Yapp, principal staff optometrist, utilises the different imaging modalities available at Centre for Eye Health (CFEH) to assist optometrists with cases where the differential diagnosis is unclear.*

“Glaucoma is a challenging condition. Multiple aetiologies mean that a wide range of information is required before a differential diagnosis can be made. At CFEH we assist optometrists in making fully-informed decisions about which patients can be monitored, and which require a referral for specialist care. It’s not surprising that more than 50% of referrals to the Centre are for glaucoma related testing and assessment.

“In a busy optometric practice it can be difficult to stay abreast of the latest developments in ocular pathology. Our regular continuing professional development events are designed to improve how conditions such as glaucoma are detected and managed in the community.”

## *Early Detection Saves Sight*

CFEH is a referral service aimed at early detection and monitoring of eye disease to prevent vision loss. Patients remain under the care of the referring practitioner and services are provided free of charge.

Centre for Eye Health

*An initiative of Guide Dogs NSW/ACT and The University of New South Wales*

# Posner Schloss
## Glaucomatocyclitic

## Case report

**Posner Schlossman Syndrome (PSS), otherwise known as glaucomatocyclitic crisis, is characterised by episodes of elevated IOP secondary to low-grade idiopathic iritis. Its hallmark is a rise in IOP that is out of proportion to the degree of inflammation.**

PI, a 74-year-old white male, presented for review. He had a history of hazy vision in the left eye of 12 hours duration. BCVA was RE 6/5- and LE 6/9= and a left relative afferent pupillary defect was present. Ophthalmoscopy revealed a normal right disc and advanced left disc pallor and cupping. Low-grade left corneal oedema was obvious with slitlamp examination. Only mild conjunctival injection was present.

Investigation of the anterior chamber revealed minimal aqueous flare and small keratic precipitates primarily in the inferior half of the endothelium. Due to corneal oedema it was difficult to view the angles with gonioscopy; however, they appeared open and no posterior or anterior synechiae were present. Applanation tonometry was RE 18 and LE 55 mmHg.

The diagnosis was made relatively easy by the existence of open angles, increased IOP, low-grade uveitis and the fact that he had a 20-year history of PSS. He was currently instilling timolol 0.5% twice daily to the left eye for secondary open angle glaucoma.

He was prescribed flarex drops hourly to the left eye with diamox tablets 500 mg stat and advised to continue instilling timolol twice daily. At review six hours later IOP was RE 11 and LE 45 mmHg. He took another 250 mg diamox and was scheduled for review the next morning.

At the subsequent review IOP was RE 9 and LE 22 mmHg and the anti-inflammatory drops were tapered over the next few weeks. His IOP eventually stabilised at RE 14 and LE 13 mmHg. Computerised perimetry, GDx, OCT and retinal photography were performed once his eye was quiet and comfortable.

He had a long history of PSS with the attacks initially occurring annually. As he has aged the episodes have become less frequent; however, due to the nerve head damage from each attack he sometimes has difficulty determining when he is having an attack. He can usually diagnose an episode by lighting a candle in a darkened room and looking for haloes secondary to corneal oedema. The attacks have caused secondary open angle glaucoma due to damage to his trabecular meshwork.

His IOP is well controlled with timolol between episodes and he has been previously prescribed diamox for use during attacks. Diamox was prescribed to lower his IOP as quickly as possible to minimise permanent damage to the nerve head and trabecular meshwork during each attack.

PSS patients have signs of anterior inflammation that are characteristically minimal with faint flare, rare cells, and only a few keratic precipitates that are small, flat, non-pigmented, and concentrated over the inferior half of the endothelium. The acute stage with rise in IOP can last up to a few weeks or as little as a day. The rise in IOP is more related to the duration of iritis rather than the degree and the condition is not associated with posterior synechiae. Depending on the IOP, corneal oedema may develop with subsequent haloes around lights.

Posner Schlossman Syndrome has two stages, iritis and increased IOP.

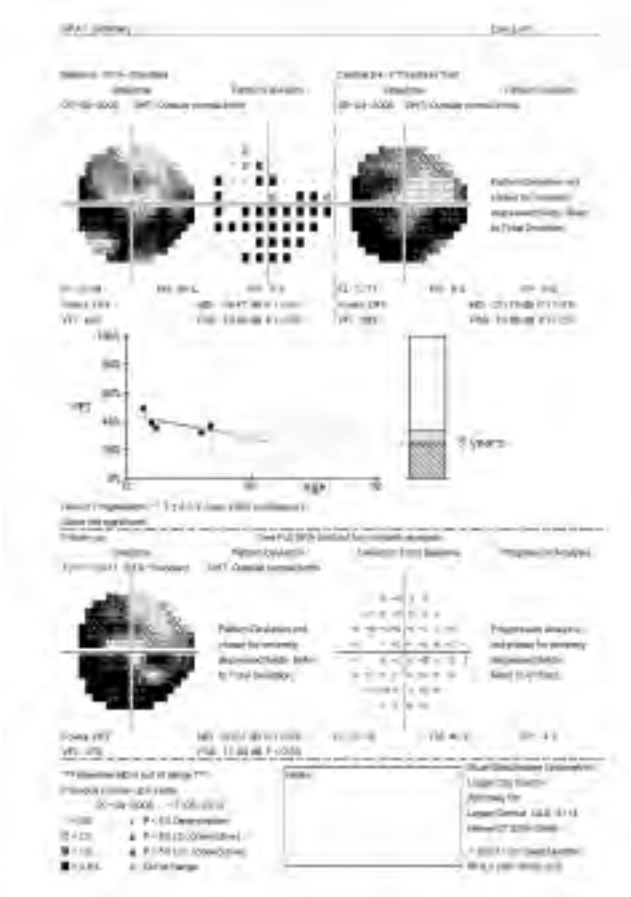

**Figure 1. Computerised perimetry revealing the visual field defect in the left eye from glaucoma secondary to Posner Schlossman Syndrome. The right visual field is normal.**

# man Syndrome crisis

**Stuart Macfarlane**
DipAppSc GradCertOcTherap

The aetiology of the iritis is unclear but has been proposed to be related to:

- abnormal vascular process
- autonomic defect
- allergic condition
- variation of developmental glaucoma
- cytomegalovirus (CMV) infection
- HSV infection.

There may be associations with allergic conditions and gastrointestinal diseases, most notably peptic ulcer disease.[1] Prostaglandins, particularly prostaglandin E, have been found in higher concentration in the aqueous humour of patients during acute attacks. These levels return to normal between episodes.[2]

Currently the most accepted explanation is that HSV is linked to PSS.[3] The trabecular meshwork is innervated by the trigeminal nerve and is a conduit for HSV. It has been proposed that HSV causes an inflammation of the trabecular meshwork, impeding aqueous outflow and increasing IOP. Both PSS and recurrent HSK may present with fine keratic precipitates, increased IOP and stress-induced unilateral attacks.

The rise in IOP is secondary to the iritis and the mechanism is thought to be from a reduction in outflow from inflammation of the trabecular meshwork. Gonioscopy reveals normal open angles without abnormal pigmentation.[4]

PSS is uncommonly bilateral and typically presents between the second and fourth decade and is rarely encountered after the age of 60 years.[5] It is more common in males.

The following diagnoses need to be excluded.

Heterochromia may indicate Fuchs' heterochromic uveitis. Other uveitic glaucomas, which deserve consideration include phacolytic glaucoma (hypermature cataract and acute glaucoma), herpetic uveitis (HSV), acute angle closure glaucoma, iridocyclitis, chorioretinitis with anterior uveitis and secondary glaucoma with anterior uveitis from retinal detachment.[6]

Gonioscopy needs to be performed to rule out angle-closure glaucoma or angle recession glaucoma. In a PSS glaucomatocyclitic crisis, the angles remain open.

Primary treatment is to resolve the inflam-

**Continued page 24**


**Figure 2. GDxVcc nerve fibre analysis showing the structural changes of the left nerve fibre layer. The NFL is 29 in the right eye and 98 in the left eye. An NFL reading below 30 is acceptable.**

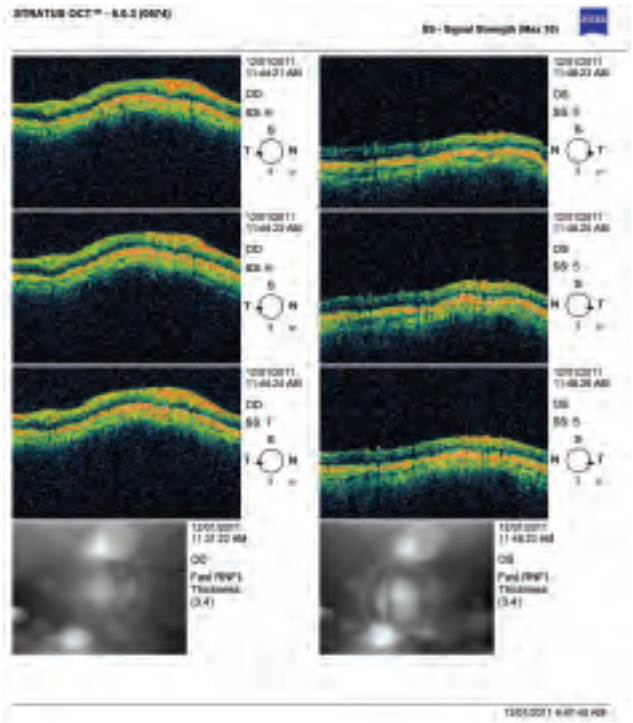

**Figure 3. OCT retinal nerve fibre layer analysis confirming the reduced thickness of the left nerve fibre layer. The NFL is below average thickness in almost all meridians.**

# Posner Schlossman Syndrome

**From page 23**

mation, which will subsequently lead to an increase in aqueous outflow and consequently a reduction in IOP. In recalcitrant cases diamox 250 mg qid may be required.

Some practitioners believe 1% apraclonidine can be prescribed during exacerbations of PSS. Four hours after instillation of 1% apraclonidine, IOP dropped 50.3 per cent during acute PSS in one study.[7]

The disease usually runs its course with topical steroids not required between the acute stages. Prophylactic anti-inflammatory therapy is somewhat controversial and most practitioners do not feel that it prevents recurrences.[4]

PSS may also be associated with POAG. In one study, 26 per cent of PSS cases were diagnosed as having developed glaucoma as a result of repeated attacks.[8] Posner Schlossman patients may also be more commonly steroid responders, which is why flarex is generally initially prescribed, as it has less potential to cause a steroid response.

It can be concluded that surgery should be restricted to those cases with severe and disabling symptoms or to cases with progressive optic neuropathy with visual field loss, when the syndrome is associated with glaucoma.[9]


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# Getting to

**Dr Mile Brujic**
Premier Vision Group USA

**Glaucoma is a chronic condition that affects from about 1.86 per cent to 8.5 per cent of the population.[1,2] Significant risk factors for the development of glaucoma include increasing age, family history and high intraocular pressures.[3]**

Once diagnosed with glaucoma, many of our patients will be on medications for their lifetime. For many of our glaucoma patients, they may even be on multiple medications to help control their intraocular pressures. From a patient perspective, multiple medications with multiple dosing schedules can become logistically difficult to keep track of and at times can decrease compliance with their medical regimen. Additionally, as the number of drops a patient uses increases, there is additional exposure to preservatives that over time can adversely affect the ocular surface.[4]

With glaucoma management, we have been able to effectively minimise preservative exposure with the introduction of prostaglandin analogues that are dosed one drop in the evening. These drops can often replace multiple medications or the use of a single medication that is required to be used multiple times a day. Minimising exposure to the preservatives in the multi-dose bottles is beneficial for a number of reasons. In addition to minimising exposure to preservatives, it is easier for patients to comply with a single dosing regimen than multiple dosing a day.

We need to keep in mind that our glaucoma patients will be treated for the rest of their lives. We need to be increasingly aware of the effects that the preservatives in our multi-dose bottles will have on the ocular surface. This is a cumulative effect that results from repeated exposure to the preservative over time.

## Closer look at preservatives

Benzalkonium chloride (BAK) is an anionic detergent that is the most commonly used preservative in eye care to preserve our multi-dose bottles. BAK is remarkably effective in eradicating microbes from multi-dose bottles, which is why its use has been embraced by manufacturers of ocular medications, but there are also effects to the ocular surface that can be measured after being repeatedly exposed to BAK.[5,6]

Patients who are being treated for glaucoma are typically elderly and will have a decreased basal tear function in addition to being more likely to have ocular surface disease.[7] In a recent study, 59 per cent of patients using glaucoma medications experienced dry eye symptoms.[8]

Numerous studies have demonstrated the effects of various preservatives to the ocular surface. An *in vitro* study looked at human corneal epithelial cells that were cultured and exposed to various preservatives. The cells were then stained to assess their viability. When cells were exposed to a prostaglandin analogue preserved with BAK, there was significantly decreased viability of the cells. When human corneal epithelial cells were then exposed to a prostaglandin analogue preserved with SofZia, an alternative to BAK, the cell viability and size was equal to the control.[9]

In a study involving an animal model, rabbit eyes were exposed to a prostaglandin analogue for three minutes. Cell size was then assessed using confocal microscopy. Corneal epithelial cells in which integrity is compromised will demonstrate morphological changes showing a decreased size

# the surface of glaucoma management

of the cells when viewed with confocal microscopy. Exposure was to a prostaglandin containing BAK and the other with an alternative preservative. Cell size was significantly decreased in the eyes being exposed to BAK containing prostaglandins and was unaffected by the alternative preservative.[10]

As opposed to being exposed to each of the solutions for three minutes, rabbit eyes were then dosed with one drop every minute for three minutes. After the dosing, confocal microscopy was again performed to determine the cell size and viability. Although the methodology was significantly different, the results were virtually identical. Researchers concluded that these superficial cell changes were the result of relatively high concentrations of BAK exposure to the ocular surface.[10]

In one study, patients were randomised into two groups; dosed with either a preservative free carteolol or carteolol preserved with BAK. They were then crossed over in a double-masked fashion to the other group. Tear film break-up time (TBUT), a common test to assess the integrity of the tear, was then tested on these patients. The patients who were dosed with carteolol containing BAK had a TBUT of 6.1 seconds whereas the group that was dosed with preservative-free timolol had a TBUT of 7.9 seconds three hours after dosing. These results were statistically significant and demonstrate some of the potential effects that BAK has on the tear film.[11]

Large-scale studies have attempted to assess the differences that exist in ocular signs and symptoms in patients using glaucoma medications that are preserved versus those that are non-preserved. In one study looking at 4,107 patients using glaucoma medications, those using preserved medications had a higher prevalence of symptoms than patients using preservative-free drops. Symptoms included discomfort on instillation (43% versus 17%), symptoms between instillations such as burning-stinging (40% versus 22%), foreign body sensation (31% versus 14%), dry eye sensation (23% versus 14%), tearing (21% versus 14%), and eyelid itching (18% versus 10%). Additionally, those patients using preserved drops had an increased incidence of corneal, bulbar and palpebral conjunctival signs.[12]

In another study that enrolled 9,658 patients, researchers assessed ocular signs and symptoms in those patients using both preserved and preservative-free drops. Again, similar to the previous study, both ocular signs and symptoms were more prevalent in patients using preserved medications. A significant decrease of all ocular symptoms and signs was observed in patients in whom the preserved eye-drops were diminished in number or who were switched to preservative-free drops.[13]

## Alternative preservatives

In attempts to minimise both the dosing schedule in addition to ocular surface exposure to BAK, industry has taken a great interest in the quest for alternative preservatives used to maintain sterility of multi-dose bottles but also be gentle on the ocular surface. Purite is one example and is the preservative used in Alphagan P, an alpha adrenergic agonist.[14] Additionally, SofZia is the preservative used in Travatan Z (available in Canada and the USA), a prostaglandin analogue.[15]

Polyquad is another preservative strategically positioned in a number of ophthalmic products. It has been used in eye-care products for over a decade with a remarkable safety profile. It is currently used in contact lens care solutions and additionally in dry-eye products. Anecdotally, I have recommended Systane Ultra preserved with polyquad for patients with dry eye who have done remarkably well but with a complete return of their dry eye symptoms when inadvertently switching to a store brand artificial tear preserved with BAK.

Currently studies are being performed on a new solution for glaucoma management, which is travaprost preserved with polyquad. This will be a welcome addition for glaucoma patients as for many it will eliminate repeated exposure to BAK.

## Summary

Glaucoma therapy has a number of challenges, one of which is maintaining a healthy ocular surface in light of long-term medical therapy. With the advent of new medications and preservatives that will be more delicate to the ocular surface, we are certain to improve patient outcomes.


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# Abstracts

**Dr Laura Downie**
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**IOP measurement discrepancies**

IOP measurements in the first eye, whether right or left, have been demonstrated to be consistently higher than IOP measured in the fellow eye. Healthy volunteers (n = 115) were randomised into two groups. The first group underwent three sets of two IOP measurements commencing with the right eye (right eye twice, left eye twice, right eye twice) using a calibrated Goldmann applanation tonometer. The second group underwent the same procedure starting with the left eye. After two weeks the order of IOP measurements was reversed between groups. The results revealed that IOPs measured higher in first eyes compared with fellow eyes. The authors hypothesised that this effect may be partially due to ocular squeezing. Taking multiple IOP measurements in a randomised order was recommended to improve accuracy.

*Arch Ophthalmol* 2011; 129: 3: 276-281

**Optic nerve sheath diameter in NTG**

Patients with normal-tension glaucoma (NGT) have been found to have an increased optic nerve sheath diameter (ONSD). CT scans of the orbit were conducted in subjects with established NTG (n = 18) and a group of gender- and age-matched healthy controls (n = 17). ONSDs were measured in axial sections using a standardised technique. ONSD was significantly increased in NTG patients. While this finding is generally associated with increased intra-cranial pressure, the authors suggest that in NTG these findings may reflect optic nerve sheath compartmentalisation and/or thinning of the optic nerve sheath.

*Br J Ophthalmol* 2011 (Epub ahead of print)

**Si-H punctal plugs an adjunct to glaucoma therapy**

Punctal occlusion has been shown to impart a clinically significant decrease in intraocular pressure (IOP) when used as an adjunctive therapy to topical travoprost 0.004% for patients with open angle glaucoma (OAG) or ocular hypertension (OH). Patients using travoprost for OAG or OH (n = 13) were treated with superior and inferior punctal plugs in one eye. IOPs were measured after one month. There were no significant differences in mean IOP for untreated eyes. In treated eyes, a mean reduction in IOP of 6.82 per cent was observed post-occlusion. The authors suggest that punctal occlusion may be a viable additional therapy option for patients using prostaglandin analogues for the management of OAG and/or OH.

*Clin Exp Optom* 2011 (Epub ahead of print)

**Brass and woodwind musicians at higher risk of glaucoma**

Professional brass and woodwind musicians have been found to undergo temporary and dramatic fluctuations in intraocular pressures (IOP) when playing their instruments, which increases their risk of glaucoma. This study investigated changes in IOP and blood pressure (BP) in subjects when playing brass and woodwind instruments. Tones of low, middle and high frequencies were analysed. Brass players showed significant IOP and BP elevations for high and middle frequency tones. For example, high tone brought on an IOP of 16.6 ± 3.5 mmHg to 23.3 ± 8.9 mmHg. Woodwind players showed the same effect at high frequencies only. For example, oboe players had IOPs of 17 ± 2.9 mmHg to 21 ± 4.4 mmHg. Playing a typical piece of music for 10 minutes was associated with significant temporary elevations in IOP. These findings indicate that professional brass and woodwind players are at increased risk of developing glaucoma and should be carefully monitored for development of the disease.

*Graefes Arch Clin Exp Ophthalmol* 2011 (Epub ahead of print)

**Topical anti-glaucoma treatment may precipitate meibomian gland disease**

A preliminary case series has reported a higher than expected incidence of patients requiring surgical intervention for chalazia when using topic prostaglandin analogues (TPAs). A retrospective study of 43 patients presenting for incision and curettage of chalazion over a two-year period was performed. Eight patients (19 per cent) were found to be using TPAs at the time of surgery. No patient had a history of eyelid margin disease before commencing TPA treatment. The authors postulate that TPAs may contribute to the formation of chalazion by acting directly to stimulate meibomian gland secretion.

*Ophthal Plast Reconstruct Surg* 2010; Dec 20 (Epub ahead of print)

**Comorbidities and glaucoma**

A large study conducted in Taiwan has demonstrated that patients with open-angle glaucoma (OAG) commonly have other serious medical problems. Data were collected retrospectively from a study group comprising more than 75,000 glaucoma patients. More than half of these patients were found to have hypertension, 30.5 per cent had hyperlipidemia and 30.2 per cent had diabetes. OAG patients had a greater tendency to have 28 of the 31 comorbidities, with more than a 50 per cent greater likelihood of having hypertension, hyperlipidemia, systemic lupus erythematosus, diabetes, hypothyroidism, fluid and electrolyte disorders, depression and psychosis. As a result of their more advanced age, many glaucoma patients are at an increased risk of chronic conditions and comorbidities, which highlights the need for thorough systemic evaluation in these patients.

*Ophthalmology* 2010; 117: 11: 2088-2095

**Long-term latanoprost reduces central corneal thickness**

Long-term use of topical latanoprost has been found to decrease central corneal thickness (CCT) in patients with normal-tension glaucoma (NTG). Established NTG patients (n = 128) were retrospectively compared with a control group consisting of glaucoma suspects with suspicious discs but normal visual fields (n = 38). Patients with newly diagnosed NTG who were prescribed latanoprost 0.005% monotherapy once daily and who had not previously been treated with topical glaucoma medications, were followed for two years or more. A significant reduction in the mean CCT was observed in the latanoprost group but not in the control group. Practitioners should therefore be aware of potential longitudinal CCT variations that may arise and affect proper IOP targeting and management in NTG.

*J Ocul Pharmacol Ther* 2011: 27: 1: 73-76

**Continued page 28**

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# Abstracts

**From page 26**

**CRVO and glaucoma link**

Patients with unilateral central retinal vein occlusion (CRVO) have been shown to have reduced retinal nerve fibre layer (RNFL) thickness in their fellow eyes. An observational cross-sectional study was performed with patients having had a unilateral CRVO (n = 79) and age-matched controls (n = 71). OCT was used to measure RNFL thickness parameters in the unaffected eyes of CRVO patients and randomly selected eyes in controls. Reduced RNFL thickness in the fellow eye of the CRVO patients was most pronounced in the inferior- and superior-temporal sectors. The authors suggest that these findings indicated that CRVO and glaucoma may share systemic risk factors that reflect a common pathogenic mechanism.

*Ophthalmology* 2010; Nov 3 (Epub ahead of print)

**Tonometers: head to head**

Intraocular pressure (IOP) measurements have been shown to differ between three major sub-types of tonometers: the Goldmann applanation tonometer (GAT), a non-contact tonometer (NCT) and TonoPen XL. A total of 508 subjects comprising glaucoma patients, ocular hypertensives and ocular normotensives with varying central corneal thicknesses (CCTs) were examined. IOPs were measured by the three different tonometers. IOP measurements with the NCT were consistently higher than those made by GAT. Both NCT/GAT and TonoPen/GAT differences were significantly associated with age, CCT, IOP and glaucoma diagnosis. It was concluded that IOP measurement differences between the three instruments were affected by CCT and IOP, and to a lesser extent age and type of glaucoma.

*Curr Eye Res* 2011: 36; 4; 295-300. ■

# Stereoscopic plays

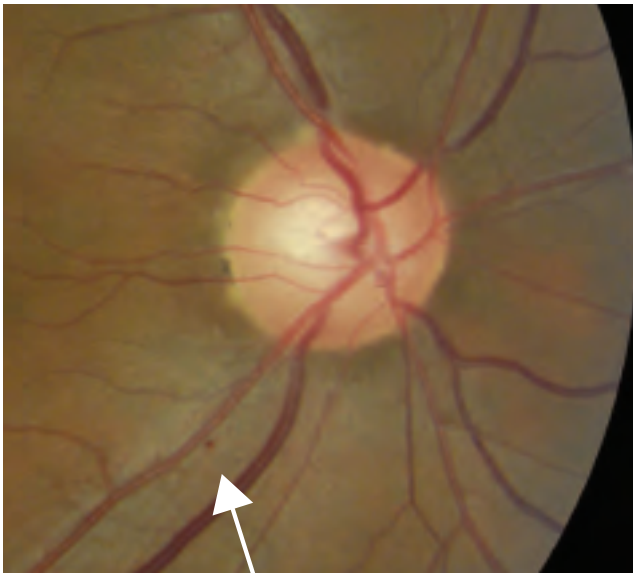

**Figure 1. Small nerve fibre layer haemorrhage (arrow) along infero-temporal vascular arcade**

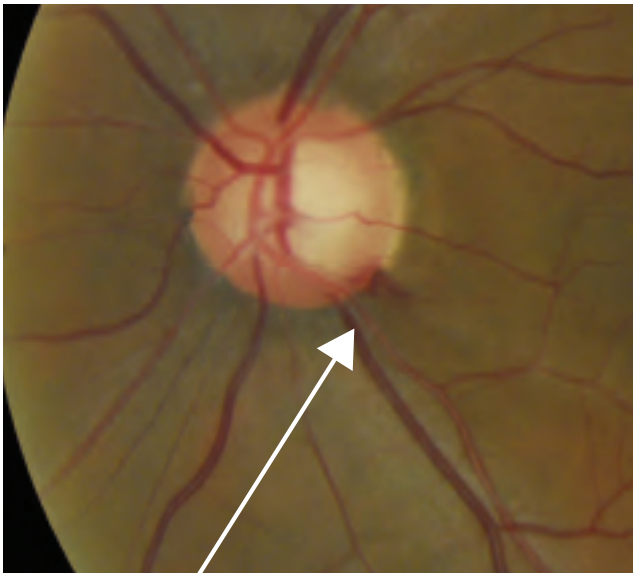

**Figure 2. Drance haemorrhage (arrow)**

# assessment important role

Case report

**Dr Laura Downie**
PhD(Melb) BOptom PGCertOcTher DipMus(Prac) AMusA

**A 49-year-old male of east-Asian descent attended for a routine, initial eye examination. He reported excellent general health and did not have a known family history of eye disease. Unaided vision was measured at R 6/5, L 6/5.**

Slitlamp examination did not reveal any abnormalities. Intraocular pressures were R 13 mmHg, L 13 mmHg with Perkins tonometry at 10.00 am. Central-corneal thicknesses were R 559 µM, L 560 µM. Gonioscopic evaluation of the anterior chamber showed open all angles in all quadrants to Grade 3 in both eyes.

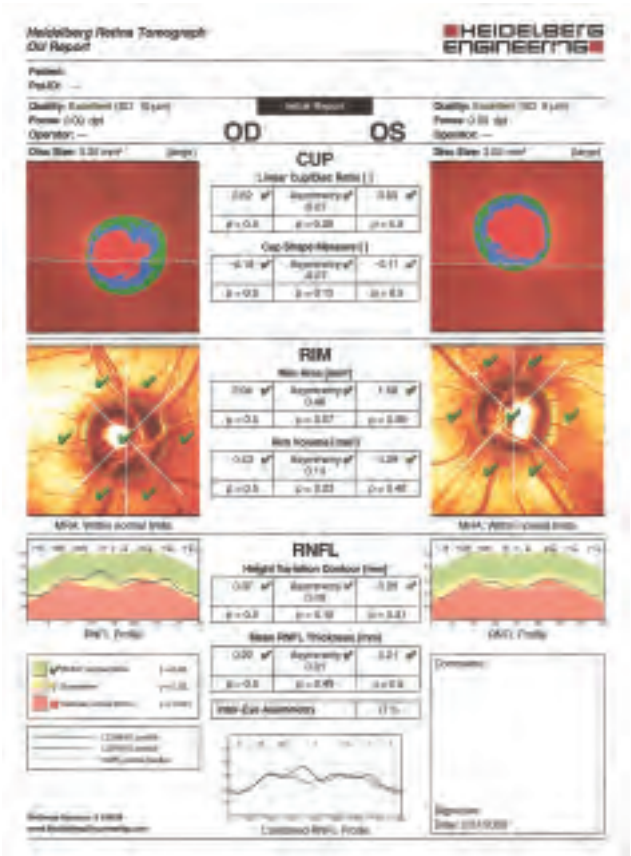


**Figure 3. Retinal tomography report**

Dilated ocular fundus examination revealed changes to the left optic nerve that were consistent with glaucomatous optic neuropathy. Optic nerve head sizes were estimated at R 2.05 mm, L 2.0 mm with cup-to-disc ratios of R 0.50, L 0.75. The right nerve appeared healthy, without any peripapillary atrophy, notching or nerve fibre layer abnormalities. A small nerve fibre layer haemorrhage was evident along the infero-temporal vascular arcade (Figure 1). The left optic nerve demonstrated a marked infero-temporal notch in the neuro-retinal rim which was associated with a Drance haemorrhage and corresponding nerve fibre layer loss; the rim also did not respect the ISNT principle (Figure 2). Mild pigment changes were noted in the para-macular and macula of the right and left eyes, respectively.

Humphrey short-wavelength automated perimetry (SWAP) with a Central 24-2 Threshold Test did not reveal any visual field loss in either eye. Heidelberg retinal tomography indicated that all optic nerve parameters were within normal limits in each eye (Figure 3).

This is an interesting glaucoma case presentation owing to the presence of asymmetric cupping, a localised neuro-retinal rim notch and associated rim haemorrhage, in the absence of any measurable visual field anomaly or quantifiable rim defect on optic nerve tomography. Following referral for an ophthalmologic opinion, the patient was commenced on Lumigan (Bimatoprost 0.3 mg/mL) eye-drops in each eye (nocte), with an initial target intraocular pressure below 10 mmHg.

The case highlights the importance of accurate stereoscopic assessment of the optic nerve by the practitioner to identify optic disc signs that are indicative of glaucomatous optic neuropathy. ■

# When it isn't

**Dr Joseph Sowka**
OD FAAO Diplomate
Nova Southeastern University
College of Optometry

**Glaucoma is a complex family of diseases where the level of intraocular pressure, combined with risk factors as well as other yet unknown features, causes a characteristic optic neuropathy with associated ganglion cell and nerve fibre layer loss with specific types of visual field damage. In most cases, correctly diagnosing glaucoma is very challenging. When making the diagnosis of glaucoma, it is essential to consider the disc appearance, nerve fibre layer structure, visual field function and associated risk factors.**

It is essential not only to take into account all of these factors when examining patients suspected of having glaucoma, but also to be acutely aware of the imposters. There are other conditions such as compressive tumours of the anterior visual pathway that may give a clinical picture similar to that seen in glaucoma. It is crucial to be able to correctly diagnose glaucoma and to differentiate it from other neurological masqueraders, and have these other conditions appropriately addressed to enhance the patient's visual outcome.

When trying to differentiate glaucoma from other neuropathies such as compressive tumours, there are two rules to always remember.

**Rule 1.** Pallor in excess of cupping indicates something other than glaucoma. That is, when the rim tissue is pale, suspect some cause other than or in addition to glaucoma. Rim pallor in glaucoma is very rare and is the exception, not the rule. Disc pallor can be accepted as part of glaucoma only when other potential causes have been eliminated.

**Rule 2.** Nothing notches a nerve like glaucoma. Focal damage to the neuroretinal rim is very specific to glaucoma. Tumours do not notch a nerve, nor do inflammations, infections, ischaemia or the like. The following cases illustrate how glaucoma differs from other optic neuropathies and when to consider that it might not be glaucoma.

## Case 1

A 56-year-old Nigerian man presented for consultation for glaucoma. He had been diagnosed with glaucoma six years earlier in Nigeria but had never received treatment. His best corrected visual acuity was 20/30 (6/8) OD and Light Perception OS. His pupils were equal and reactive with a left relative afferent defect. Intraocular pressure (IOP) was 30 mmHg OD and 23 mmHg OS. Biomicroscopy revealed no abnormalities and gonioscopy demonstrated normal open anterior chamber angles. Dilated fundus examination revealed optic disc cupping of 0.75/0.75 OD and 0.8/0.8 OS. There was distinct damage to the left neuroretinal rim. Nerve fibre layer analysis via scanning laser polarimetry was abnormal in the left eye. Most notably, the remaining neuroretinal rim OS was pale in comparison to the fellow eye (Figure 1). He clearly had optic atrophy OS. His acuity of light perception was not consistent with optic disc cupping of 0.8/0.8, if glaucoma was the sole cause.

At this point, he was diagnosed with glaucoma as one potential condition and suspected of having an additional neuropathy as well. Threshold perimetry was performed, revealing a bitemporal visual field defect respecting the vertical hemianopic line OD and crossing over to involve fixation OS (Figure 2). This was consistent with a chiasmal tumour. Magnetic resonance imaging (MRI) confirmed the tumour. This patient had a tumour as well as glaucoma. The key diagnostic finding was disc pallor

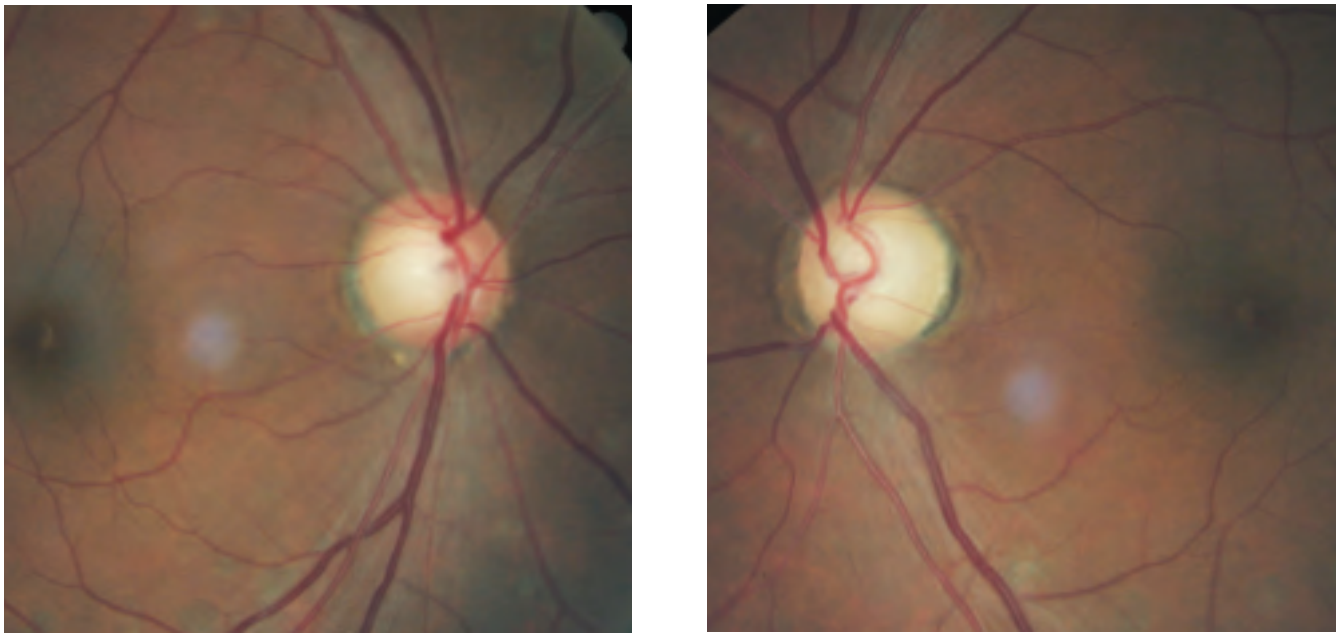

**Figure 1. Relative disc pallor OS compared to OD**

# glaucoma

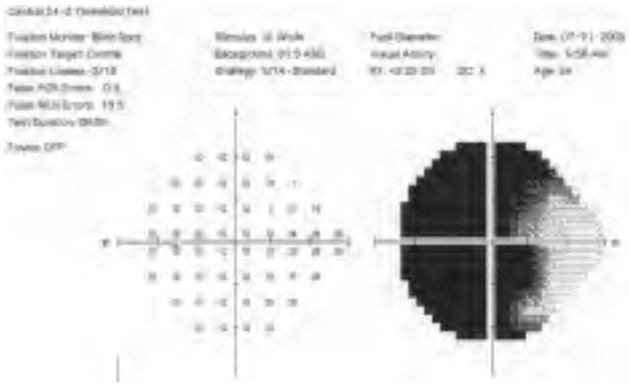

**Figure 2. Bitemporal visual field defect in patient 1 due to chiasmal tumour**

and visual acuity loss much worse than would be expected from glaucoma.

## Case 2

A 53-year-old woman was referred for suspicion of glaucoma. She had no visual or ocular complaints. Best corrected visual acuity was 20/20 (6/6) in each eye. Pupils were reactive and there was a relative afferent defect in the left eye. Colour vision testing was normal. There were no biomicroscopic abnormalities and anterior chamber angles were open gonioscopically. Intraocular pressure was 30 mmHg OD and 32 mmHg OS.

Threshold perimetry revealed a full and normal field OD and a dense inferior arcuate defect OS, consistent with glaucoma (Figure 3). Dilated examination showed concern. Her right optic disc was pink, distinct and normal. Her left nerve, while demonstrating notching of the neuroretinal rim superior temporally, was also pale in this region (Figure 4). Although the acuity and color vision were normal, and the IOP was clearly in a glaucomatous range, the unilaterality and the presence of OS disc pallor raised the suspicion of an optic nerve compressive tumour. The patient underwent MRI of the orbits and chiasm both with and without contrast, which turned out to be normal. Despite the suspicious disc appearance, the patient has nothing more than glaucoma and continues to do well under topical glaucoma therapy.

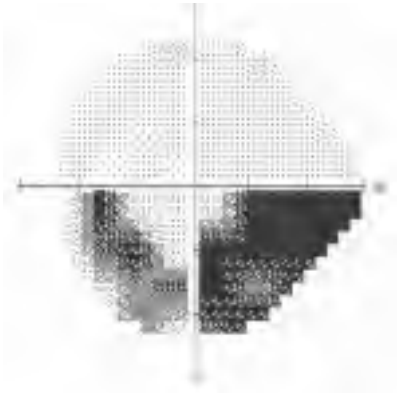

**Figure 3. Inferior arcuate defect consistent with glaucoma**

## Case 3

A 70-year-old man with known advanced glaucoma transferred his care. Through careful titration of topical medication, he achieved an acceptable IOP of 17 mmHg OU. He then had a car accident with concussion and loss of consciousness. Some time afterwards, he developed gaze-induced amaurosis fugax; that is, when he looked in a particular direction of gaze, he would report that his vision would 'black out'. This is highly suggestive of a retro-orbital tumour. In such an instance, when a patient looks into a certain direction, there is an excursion of the optic nerve within the optic canal. If a tumour is situated in the optic canal behind the globe, the optic nerve would touch the tumourous mass, interrupting axoplasmic flow, and vision would black out.

His primary-care physician referred the patient to a neuro-ophthalmologist who performed a complete evaluation and suspected a retro-orbital tumour and ordered an MRI. The MRI was normal with no evidence of a retro-orbital tumour. The neuro-ophthalmologist dismissed the patient back to my care, suggesting that the patient's gaze-induced vision loss was likely to be 'psychological', which did not sit well with the patient.

Examining the patient, neither optic disc manifested any degree of pallor (Figure 5), which meant that Rule 1 had not been violated. He had severely notched optic disc rims in each eye, so Rule 2 was being obeyed. His damaged nerves completely matched his advanced visual field loss, so I had no reason to suspect anything other than glaucoma being present. When discussing the vision loss with the patient, he remarked that it occurred daily. I asked him specifically which tasks prompted the black-outs and he identified shaving. I asked him to approach the mirror on the wall and demonstrate his shaving technique. He mimicked raising his chin up and turning his head to the left while he looked right to shave and at that point, he remarked that his vision suddenly was dark.

The explanation was clear. As he moved his chin up and looked eccentrically, he was looking into his glaucomatous field and his nose was blocking the normal part of the visual field of his other eye that usually served to compensate. His task of shaving only made him more aware of his glaucomatous field loss. Despite a history strongly consistent with a retro-orbital tumour, there was no disc pallor, the nerves were notched in a glaucomatous manner, and the vision and visual field were entirely consistent with the disc appearance. In his case, he had only glaucoma.

In 1998, David Greenfield and associates published a seminal paper discussing which patients with glaucoma needed neuro-imaging.[1] In their retrospective analysis, they compared glaucoma patients who had normal intraocular pressure and had undergone neuro-imaging as part of their evaluation (due to the normal IOP) with patients in whom compressive lesions were diagnosed. They found that none of the patients diagnosed with normal tension glaucoma had neuroradiologic evidence of a mass lesion in the anterior visual pathway.

**Continued page 32**

# When it isn't glaucoma

**From page 31**

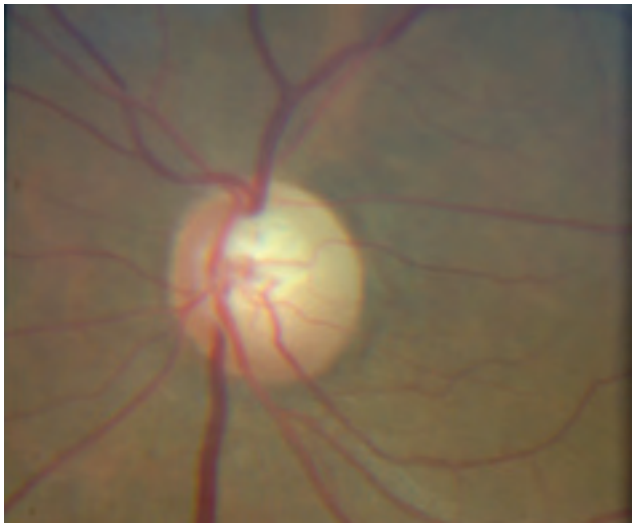

**Figure 4. Glaucoma patient with superior rim notching and pallor**

They found that when compared to the control group with mass lesion, those patients with glaucoma were older, had better visual acuity, greater vertical loss of neuroretinal rim, more frequent disc haemorrhages, less neuroretinal rim pallor and more nerve fibre bundle defects aligned along the horizontal midline. In contrast, those patients with mass lesions of the anterior visual pathway typically had visual acuity less than 20/40 (6/12), vertically aligned visual fields defects, optic disc pallor in excess of cupping, and were younger than 50 years.

Their conclusion was that younger age, lower levels of visual acuity, vertically aligned visual field defects, and neuroretinal rim pallor were more indicative of a compressive mass lesion than of glaucoma.

In the first case, the patient had disc pallor and unexplained vision loss. His visual field defect was vertically oriented and not arcuate in nature. This all indicated something in addition to glaucoma. In his case, he had a chiasmal tumour.

The second patient had unilateral disc pallor but no acuity loss or dyschromatopsia, elevated IOP, notching of the rim, and a corresponding glaucomatous arcuate visual field defect. The pallor obligated an investigation with neuro-imaging, which was ultimately normal. By elimination of other potential causes, the patient is left with a diagnosis of glaucoma.

The final patient had a history strongly suggestive of an orbital tumour, but his disc appearance was strongly indicative of glaucoma and matched well his visual fields. When questioning the patient carefully, it was easy to ascribe his vision loss to his awareness of glaucomatous damage.

It is important to remember the differences between glaucoma and compressive lesions of the anterior visual pathway. To help differentiate these conditions, I created the 'Ode to a Cupped Disc' to help colleagues remember the rules of notching and pallor.

### Ode to a cupped disc

Oh, to have a cupped disc pink,
That my friend hath a glaucomatous stink.
But to have a cupped disc pale,
Call this glaucoma and you will fail.
Disc and field damage that is one sided,
Simply cannot be abided.
It might be trauma or ischaemia, or meningioma.
But if the rim is cut always remember,
Nothing notches a nerve like glaucoma.

1. Greenfield DS, Siatkowski RM, Glaser JS et al. The cupped disc: Who needs neuroimaging? *Ophthalmology* 1998; 105: 1866-1874.

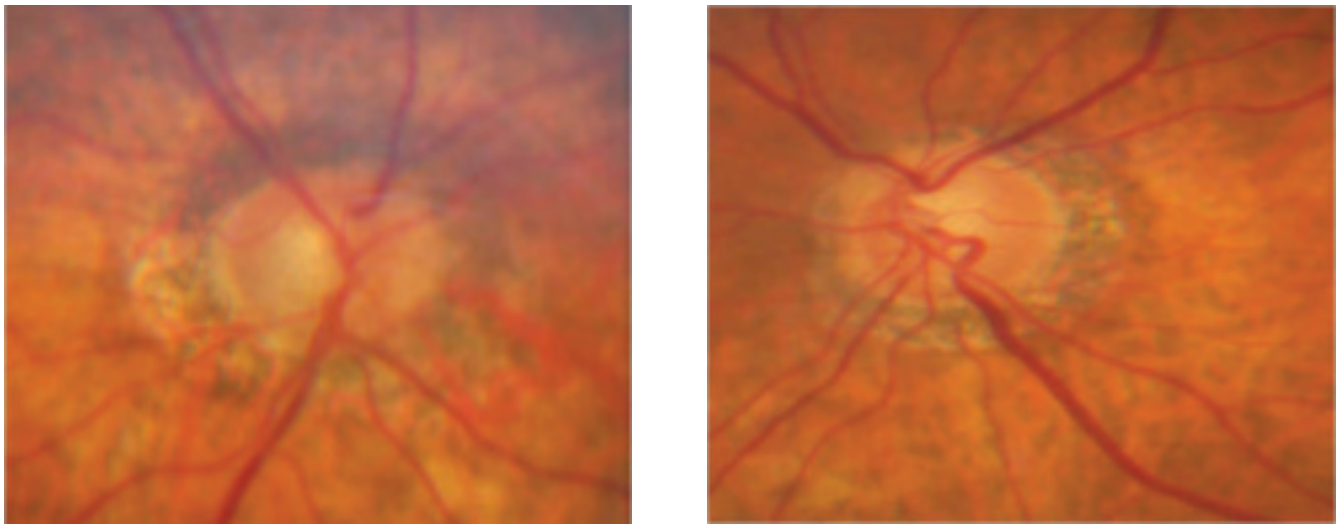

**Figure 5. Glaucomatous nerves with neuroretinal rim notching and no disc pallor**

# Comorbidity

## Patients can have more than one condition that needs your attention

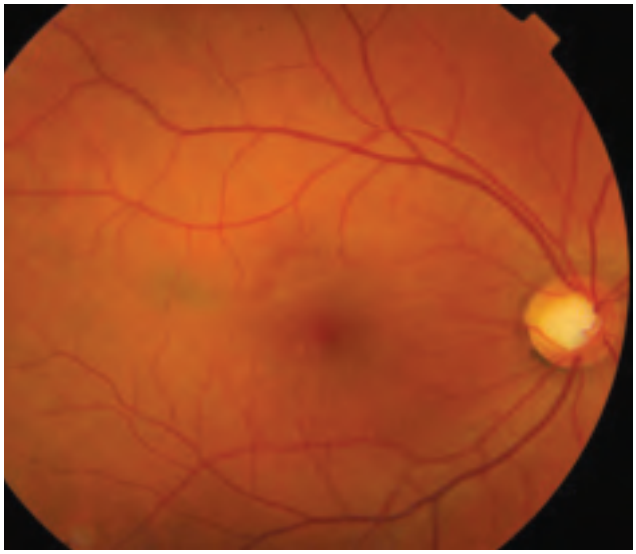

**Figure 1. Colour fundus photo of the right eye March 2009. There is mild NPDR, C/D 0.7 and healthy NRR.**

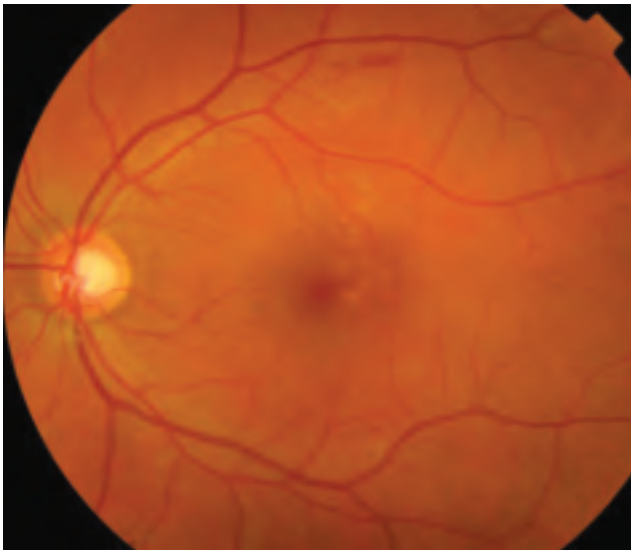

**Figure 2. Colour fundus photo of the left eye March 2009. There is mild NPDR, C/D 0.5, query inferior thinning. Note the retinal pigment epithelial (RPE) changes visible temporally are a photographic artifact, although this appearance is not dissimilar from the appearance of RPE three to six months post argon laser.**

**Mary Travis**
MOptom
Vision Eye Institute, Footscray

**As the scope and complexity of optometric practice continues to evolve, optometrists are increasingly becoming involved in the care of patients with complex medical histories. Refreshing and expanding both undergraduate level knowledge of optometry and complementary clinical capabilities, through formal continuing professional education and intrinsic clinical curiosity, can help to guide career-sustaining professional development.**

This principle applies to all areas of practice in optometry from paediatric optometry to low vision care, from contact lens practice to the broad generalist skills of your friendly neighbourhood optometrist. This principle perpetuates our profession's tradition of a commitment to high standards of clinical care and professional development.

A wise but not so old ophthalmologist once said to me, 'Optometrists spend their lives obsessing over a 2.5 degree change in the axis of astigmatism.' This is perhaps true at times but it is this innate attention to detail that can be easily adapted to the care of patients with multiple medical problems, such as the increasing but unfortunate demographic who have diabetes or glaucoma. Applying too much attention to detail and omitting an overall assessment of the broader clinical picture of your patient can also be problematic.

## Case report

Mr AC is a 45-year-old with mild astigmatism who was referred by his local GP in June 2009 to Vision Eye Institute in Footscray regarding a recent diagnosis of non-insulin dependent diabetes mellitus type 2 (NIDDM). Thankfully before he exhibited a vaso-vagal reaction to tonometry and literally fell off our patient chair, he mentioned

**Continued page 34**

| Date | Record | R-VA | L-VA | R IOP | L IOP | HbA1c | BP | lipids | R disc | L disc | R retina |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 06.01.2009 | MT | 6/5- | 6/6p | 18 | 18 | 8.6 | OK | OK | 0.7 | 0.5 | mild NPDR |
| 22.09.2009 | NJ | 6/5 p | 6/6- | 18 | 18 | 6.2 | 116/77 | OK | 0.7 | 0.5 | CSME/Hex |
| 02.11.2009 | NJ | | | | | | | | 0.7 | 0.5 | |
| 10.11.2009 | NJ | | | | | | | | 0.7 | 0.5 | improved |
| 09.03.2010 | NJ | 6/6+ | 6/9- | | | | | | 0.7 | 0.5 | mild NPDR |
| 25.03.2010 | NJ | | | CCT 542 | CCT 531 | | | | 0.7 | 0.5 | thin inf NRR |
| 16.08.2010 | NJ | 6/5p | 6/6p | 18 | 18 | | | | 0.7 | 0.5 | mild NPDR |
| 10.11.2010 | MT | 6/5 p | 6/6- | 17 | 18 | 6.4 | 138/85 | OK | 0.7 | 0.5 | thin inf NRR |
| 28.03.2011 | MT | 6/6= | 6/5= | 14 | 12 | 4.2 | OK | OK | 0.7 | 0.5 | mild NPDR |

**Table 1. Summary of clinical findings: Mr AC**

# Comorbidity

**From page 33**

a family history of glaucoma. His mother had developed glaucoma with an onset in her seventh decade, may also have had glaucoma surgery in her seventh decade, but definitely did not have NIDDM.

Table 1 summarises his clinical treatment over the course of the past two years.

The points of interest are:

- In this case there is a clear indication for visual fields: young NIDDM patient with family history of glaucoma and 0.2 C/D asymmetry
- Good clinical record-keeping and serial clinical data (fundus photography, OCTs, GDx, HRT et cetera) allow you to retrace your clinical steps when reviewing glaucoma patients. In this patient there have been no optic disc changes since the first appointment in 2009.
- Remember intra- and inter-optometrist variations can also affect C/D ratios. (Direct ophthalmoscopy versus 90 D fundus lens, degree of contrast in fundus photos. For example, a brighter photo will reveal more subtle NRR or nerve fiber layer changes but too much light will also disguise changes, dimming due to cataract can confound interpretation of disc photos.)
- Analysis of Mr AC's diabetic parameters over the two years shows improvement in diabetic control (HbA1c), which has stabilised the retinal findings. In this case the fundus photo of the left eye from March 2011 is absolutely normal aside from some faint laser scars.
- There is little support in clinical research for an anecdotal observation in clinical practice that some patients with NIDDM and IDDM experience worsening of their retinopathy with sudden improvements in diabetic control. This has been noted in young IDDM patients who are switched to insulin pumps or NIDDM patients who make drastic lifestyle changes in diet and exercise over a relatively short time, such as a few months. The level of diabetic retinopathy for Mr AC worsened between March and September 2009 as the HbA1c improved, but then the retinopathy steadily improved to reflect improved control and lifestyle (diet and exercise).

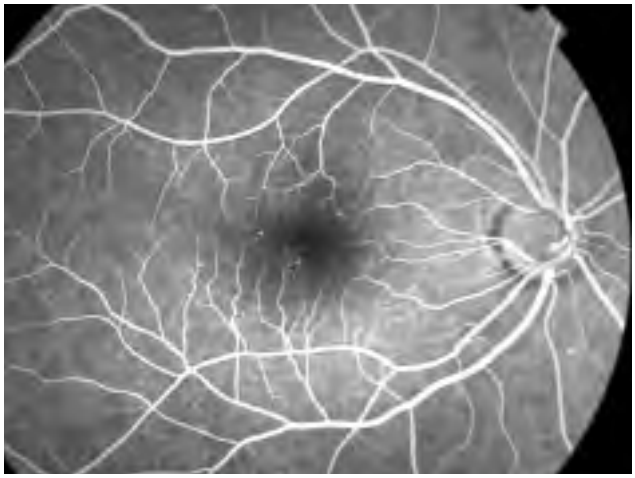

**Figure 3. Fundus fluorescein angiography of the right eye. This image clearly illustrates the architecture of the macular capillary network in the early-mid phase, demonstrating the propensity for an increased population or retinal microaneurysms to be present with angiography.**

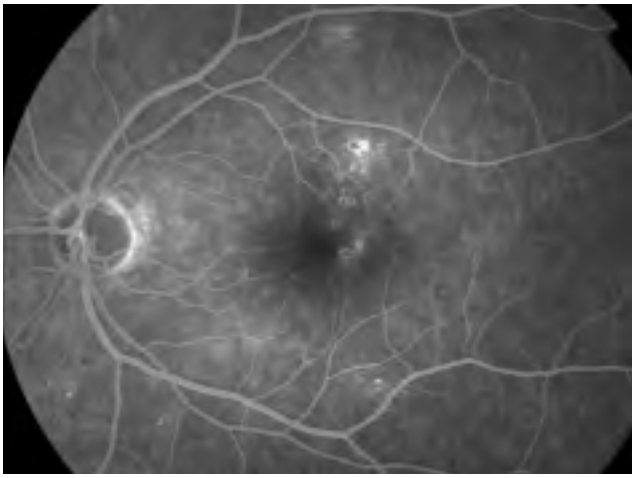

**Figure 4. Fundus fluorescein angiography of the left eye that demonstrates leakage from the microaneurysms, especially those close to the macular. Angiogram findings suggest that focal argon laser would be required in both eyes, due to the presence of leaking microaneurysms in the macular region, however clinically only the left eye subsequently underwent treatment**

| L retina | R OCT | L OCT | Comment |
|---|---|---|---|
| mild NPDR | | | R 4/12 - no glaucoma VF OK |
| CSME/Hex | CT 288 (Stratus) | | FFA/subsequent focal laser |
| | | | L focal laser |
| | no fluid | no fluid | WAIT re R focal laser |
| few ma temporal to macula | no fluid | no fluid | WATCH |
| | | | FFA - no leakage |
| mild NPDR | no fluid | no fluid | needs glaucoma review |
| no DR - RPE drop-out re focal laser | | | R 6/12 – VF OK |

- All patients diagnosed with chronic and potentially progressive disease will vary in their levels of motivation and compliance throughout their treatment. Optometrists are well placed at a primary eye care level to build close and ongoing relationships with patients to monitor their condition and give clinical advice that can help motivate patients to take better care of both their ocular and general health.
- The need to communicate regularly with your patient's general practitioner is crucial for the best overall care of patients with chronic medical diseases such as diabetes or glaucoma. GPs can provide useful contextual data (HbA1c, BP, lipid profile) for monitoring diabetic patients with respect to UKPDS and ACCORD/ACCORD eye study recommendations. Communication with the complete clinical team is also helpful, as there are often multiple practitioners involved, such as a diabetes educator and endocrinologist.
- Multifactorial longitudinal patient data analysis is a complex skill that requires repetition and reinforcement to develop. Useful data such as a C/D ratio from five or 10 years previously can easily be overlooked but we optometrists with a grey hair grading of trace to 3+ are proud of these complex data analysis skills.

| Code | Explanation |
|---|---|
| BP | blood pressure |
| CT | central macular thickness (microns) |
| CCT | central corneal thickness (microns) |
| CSME | clinically significant macula oedema |
| FFA | fundus fluorescein angiogram |
| HbA1c | % glycosolated haemoglobin |
| Hex | hard exudates |
| inf | inferior |
| ma | microaneurysms |
| MT | author |
| NJ | Dr Nandor Jaross |
| NPDR | non-proliferative diabetic retinopathy |
| NRR | neuroretinal rim |
| p | part line of Snellen acuity |
| R | review |

**Acknowledgements**
I thank Mr AC, Dr Nandor Jaross and the orthoptic team at Vision Eye Institute Coburg and Footscray for clinical images.

# Technology 1, Glaucoma 0

**Lee Pepper**
BOptom
Optical Manufacturers

**When I graduated from university in the 1980s, glaucoma was one of those eye diseases we thought we understood. If the pressures were high, the patient had glaucoma and we sent them off to the ophthalmologist, right? Pachymetry was called pachometry and of interest only to contact lens researchers, and terms like SWAP, frequency doubling and flicker perimetry had not been uttered by anyone.**

A few years down the track and it has become obvious how naive we were about the disease then. As I sat in a glaucoma lecture at AVC recently, absorbing the concept of an optometrist diagnosing and treating glaucoma without the need for ophthalmological participation, I was left pondering how far we have come in the treatment of the disease.

There is no excuse for today's optometrist to allow glaucoma to take hold of our patients' vision. Now that we as practitioners benefit from state of the art technology and a better understanding of glaucoma, it is clear that actual scotomata happen only late in the piece.

Traditional perimetry is now far less critical for early detection than other tests, which are now well within the domain of optometrists who practise with care of the patient's vision as the ultimate concern.

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# Benefit of optical

**Dr Alan Burrow**
DipOptom FBOA HD DCLP
DOrth FBCO MSc(Med)
GradCertOcularTher
GCBA

**Glaucoma is one of the major causes of irreversible blindness worldwide**[1] **with primary open angle glaucoma being the most common form in Australia.**[2]

As primary eye-care practitioners, optometrists are at the forefront of glaucoma detection and increasingly involved with glaucoma treatment. Diagnosis is difficult in the early stages as no single definitive test is currently available; therefore, assessment of numerous risk factors is frequently required. Glaucoma is often diagnosed primarily on optic nerve head changes and visual field defects in the presence of an above average intraocular pressure;[3] however, numerous studies since the early 1970s have demonstrated a greater prevalence of normal tension glaucoma.[4] Confirmation of the diagnosis is still dependent on characteristic progression of visual field loss.[3]

While a significantly elevated intraocular pressure may be the first diagnostic indicator, suspicion is more commonly aroused by the characteristic cupping of the disc. Unfortunately, physiological cupping is extremely common and difficult to differentiate from glaucomatous cupping in the early stages. Visual field defects may not be evident until significant irreversible nerve fibre loss has occurred.[5]

Ophthalmic practitioners need to consider a range of risk factors when making a provisional diagnosis. These include disc appearance, intraocular pressure, corneal thickness, visual field defects, pigmentary dispersion and pseudo-exfoliation syndromes as well as hereditary factors.

Traditional methods of optic nerve assessment are subjective and require significant clinical expertise. While visual field analysis is an important tool, it is also subjective and frequently difficult to perform accurately, particularly on patients with a limited attention span. Visual field responses are variable and visual field defects frequently do not occur until the disease is fairly advanced.[6]

Spectral domain optical coherence tomography (OCT) on the other hand provides an objective and repeatable method of assessing the optic nerve head, retinal nerve fibre layer and ganglion cell complex (GCC), which have been shown to undergo changes in glaucoma.[6,7] In some cases OCT

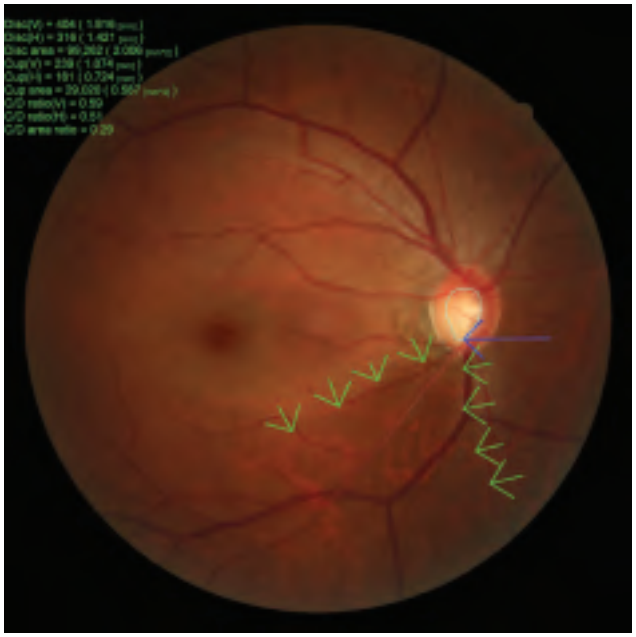

**Figure 1. Right eye illustrating a neural notch (blue arrow) and the limits of the nerve fibre drop-out (green arrows)**

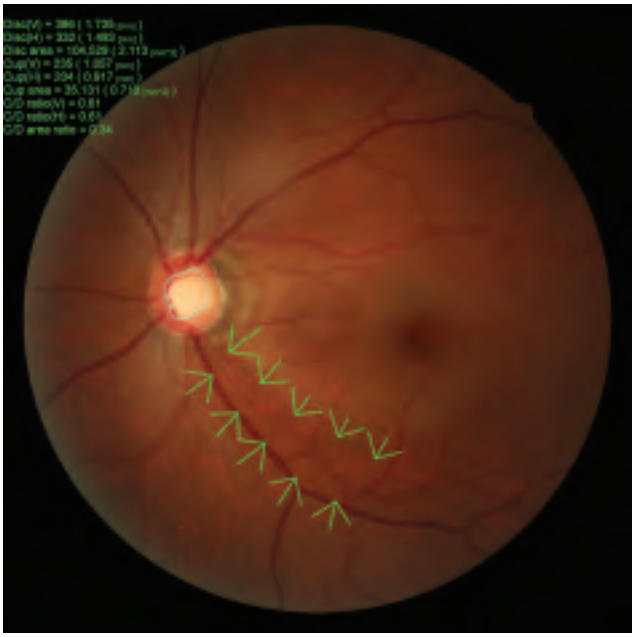

**Figure 2. Left eye illustrating peripapillary atrophy and the limits of the nerve fibre drop-out (green arrows)**

# coherence tomography

## in detection and monitoring of glaucoma

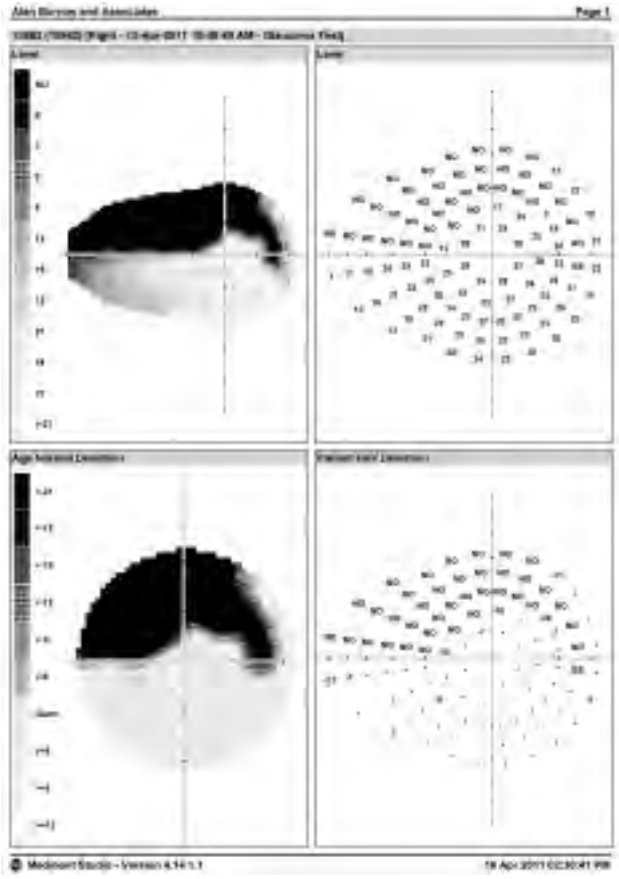

Figure 3

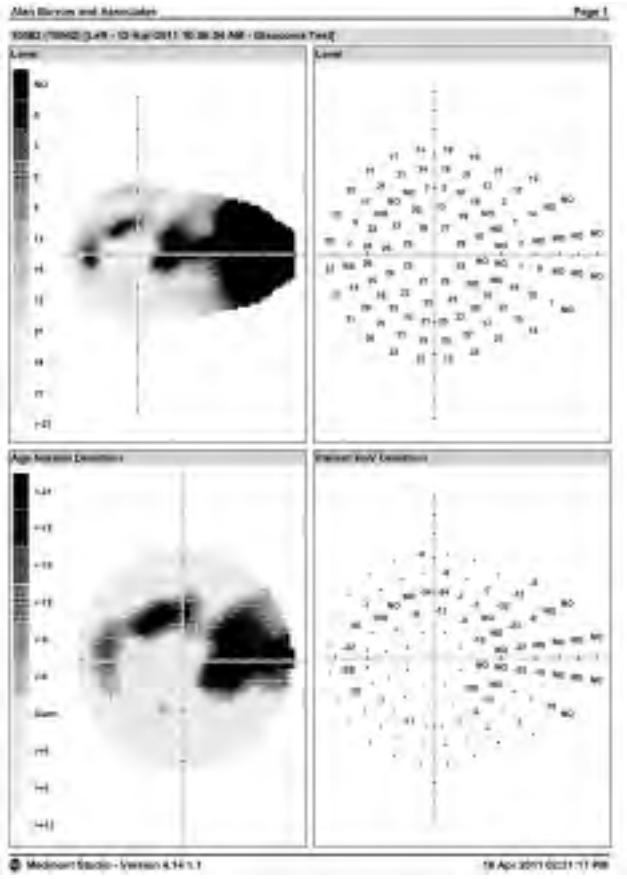

Figure 4

changes may precede visual field loss.[8] This technique is therefore a valuable additional tool in the early diagnosis and ongoing monitoring of the disease.

## Case report

A 46-year-old Japanese-born female who had worn soft contact lenses for five years reported for a routine optometric assessment. She had been experiencing intermittent blurring of near vision with her contact lenses and required back-up spectacles. No symptoms indicative of visual field loss had been experienced.

The patient was in good general health with no diagnosed diabetes or hypertension. She was not a migraine sufferer nor had she been exposed to cortisone therapy but a maternal aunt was reported to have been diagnosed with glaucoma.

The refraction was right -225/-025 x 175 6/5 and left -175/-025 x 170 6/5. The external ocular examination was unremarkable with normal pupil responses. The fundi were assessed with the slitlamp, a super field lens and stereo photography which demonstrated bilateral tilted disks with some peripapillary atrophy inferotemporally in the right eye (Figure 1) and a temporal crescent in the left (Figure 2).

The disc margins and cups were measured using Nidek Navis software. The parameters are displayed in the upper left-hand corner of the photographs. The right vertical cup/disc ratio was 0.59 with an inferior notch indicated by the blue arrow. The left vertical cup/disc ratio was 0.61. No disc haemorrhages were noted in either eye

**Continued page 38**

# Benefit of OCT

**From page 37**

and there were no signs of pigment dispersion or pseudo-exfoliation syndromes. Both eyes exhibited localised nerve fibre layer drop-out as can be seen in Figures 1 and 2.

The intraocular pressures were R 11 and L 12. The patient had been examined four years previously when similar pressures had been recorded. Pachymetry recorded thinner than average corneal thicknesses with an average central corneal reading (standard deviation) of right 470 µ (2.4) and left 481 µ (5.5).

The visual fields, which were assessed using the Medmont visual field analyser, demonstrated an arcuate type scotoma in both eyes (Figures 3 and 4). The visual field loss was consistent with the nerve fibre layer drop-out, both being more marked in the right eye.

The OCT records numerous parameters relevant to glaucoma including optic disc and cup ratios and sizes, the retinal nerve fibre layer thickness (RNFL) and the GCC layer thickness.

The OCT scans of the optic nerve and retinal nerve fibre layer are shown in Figures 5 and 6. The retinal nerve fibre layer map is the middle image on the right. The optic disc is represented by a circle with the disc being dark grey and the cup light grey. The colour-coded thicknesses of the RNFL are shown by the legend on the right of the map. Surrounding the RNFL map are 16 sectors representing the RNFL thickness at each location. These sectors are colour-coded based on the normative database with green being within normal limits, yellow borderline or suspicious and red outside normal range.

At the bottom right is the TSNIT graph which plots the RNFL thickness profile around the optic disc starting temporally then superiorly, nasally, inferiorly and back to temporally. The patient's nerve fibre thickness is shown by the black line with the green band representing the normal range, the yellow borderline and the red outside normal range. The optic disc parameters are displayed in the box in the lower left corner and include areas, volumes and cup/disc ratios.

In both the right and left eyes the nerve fibre layer thickness dips into the red area on the TSNIT and the retinal nerve fibre layer map in the inferotemporal segments, indicating that the thickness of the layers is outside the normal range with a probability of $p < 1$ per cent. This is consistent with the nerve fibre drop-out and the scotomas recorded on the visual field.

The optic disc parameters are displayed in the table on the left. The RTVue GCC scan consists of 15 vertical line scans covering a 7 mm square region centring 1 mm temporal to the fovea. This scan quantifies the thicknesses of the layers affected by glaucoma.

The GCC maps are displayed in Figures 7 and 8. The thickness map is displayed in the upper right corner with a significance map in the lower right corner. This map shows the deviation from the expected norm with the colour-code indicating the probability of abnormality. The yellow areas are borderline while the red indicates a high probability of abnormality. Red and yellow areas that are smaller than the grey zone surrounding the fovea are not uncommon in normal subjects. The large yellow and red zones for this patient are highly suggestive of glaucoma.

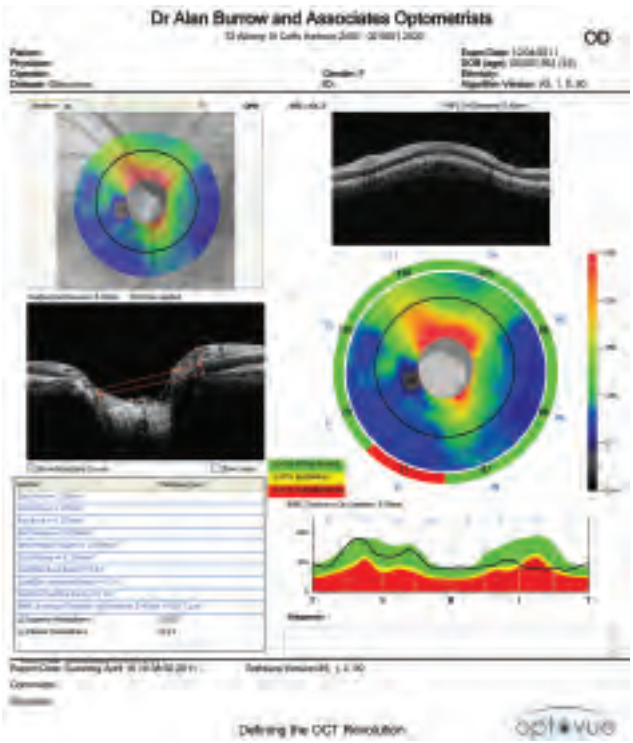


**Figure 5**

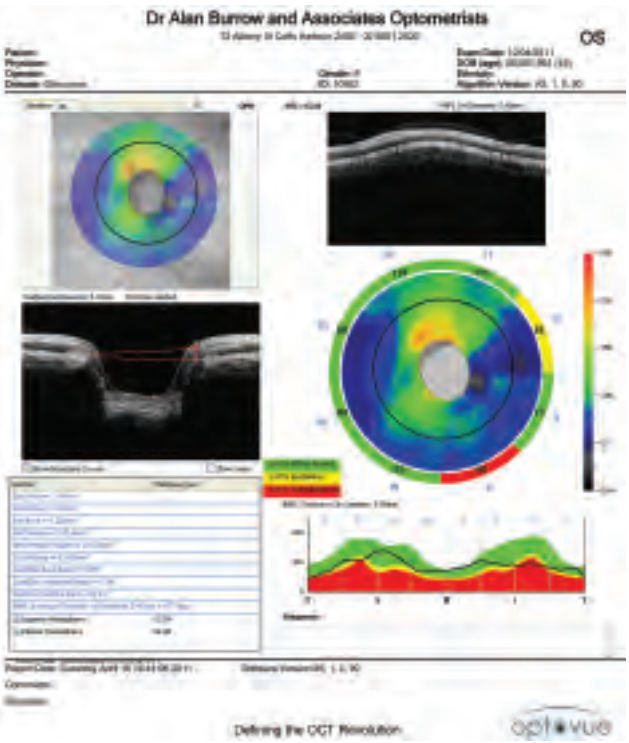


**Figure 6**

## Discussion

This patient displayed a significant number of glaucoma indicators including the increased cup/disc ratios, nerve fibre drop-out, typical visual field scotomas and the notch in the right neuroretinal rim. The corneal thicknesses of right 471 µ and left 481 µ are significantly thinner than the average of 542 µ which represent a risk factor, as does the positive family history.[9] The intraocular pressures are low even when adjusted for the reduced corneal thickness; however, glaucoma with normal intraocular pressures is not uncommon.[4] Peripapillary atrophy, which is considered a risk factor in normal tension glaucoma, is present in both eyes but appears more marked in the left.[10] Both eyes exhibited clear nerve fibre layer drop-out which, while suggestive of glaucoma, can be caused by other ocular conditions.[10,11]

Even though the patient had a low degree of myopia, this has been identified as a significant risk factor.[10,12] There were no signs of Drance-type haemorrhages, pseudo-exfoliation or pigment dispersion syndromes and the patient had not been exposed to steroids.

The RNFL and GCC maps are consistent with the nerve fibre layer drop-out and visual field scotomas, and provide further evidence of glaucoma.

Treatment was appropriate because of the clinical features of this case and the patient's age. Therapeutic treatment consisted of prescribing Xalatan eye-drops and subsequently upgrading to Alphagan bid and Duotrav nocte, which reduced the pressures to R 9 and L 10.

## Conclusion

This patient was alert, providing accurate visual field analyses with definite arcuate scotomas. There was also a significant number of other indicators for commencing treatment without the evidence of the OCT scans; however, frequently visual fields can be highly variable with other signs of glaucoma being far less definitive. In such cases the OCT provides an excellent objective method of assessment that can be invaluable in the early detection and progress evaluation of glaucoma. It should be emphasised that the OCT needs to be viewed in conjunction with other signs and risk factors of glaucoma.


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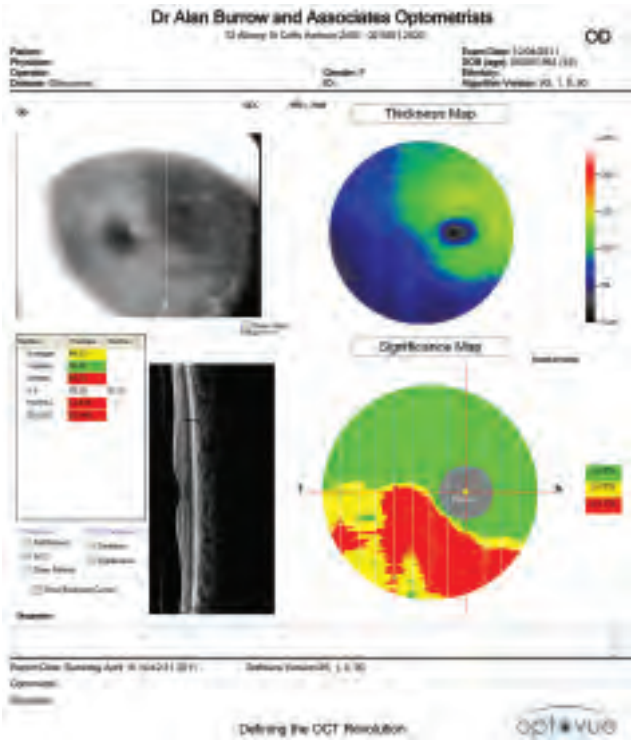


**Figure 7**

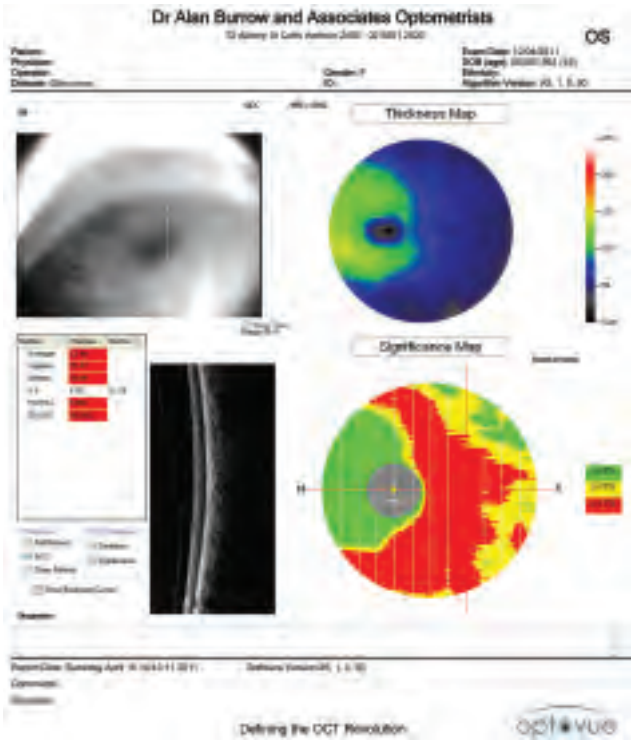


**Figure 8**

# Normal tension

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Clinicians should regularly review the effectiveness of their treatment and adjust their management as the disease progresses

## Case report

**This report presents the case of a 61-year-old female with normal tension glaucoma. She had advanced glaucomatous optic neuropathy with visual field loss and IOP always below 21 mmHg. Her glaucoma was well-controlled with medical therapy (Xalacom and Azopt drops).**

A 61-year-old Mauritian female (Mrs M) presented to the optometry clinic for a routine examination in January 2004. Her previous examination had been three years earlier and there were no ocular or visual complaints. She had mild myopia. There was a family history of glaucoma, with an aunt and an uncle on her mother's side affected. She reported low blood pressure and poor circulation, but was otherwise in good general health. She was not taking any medication.

**January 2004**

At the initial presentation, cup-disc ratios were R 0.65 L 0.55 and the neuroretinal rims followed the ISNT rule. Gonioscopy showed that anterior chamber angles were narrow but open and there were no peripheral anterior synechiae. IOP was R 19 mmHg L 18 mmHg. Visual acuities were R 6/6 L 6/6. HFA 24-2 threshold visual fields showed right eye early inferior nasal step as well as superior losses. The left visual field was within normal limits (Figure 1). Review was scheduled for one month but the patient did not attend.

**August 2005**

After an extended overseas trip, the patient returned more than one year later, complaining of reduced vision in the right eye. Visual acuity had dropped to R 6/18 L 6/7.5. Anterior eye examination showed moderate right nuclear sclerosis and posterior subcapsular cataract. There was progression in disc damage with cup-disc ratios now R 0.75 L 0.75. IOP was R 21 mmHg L 21 mmHg. Mrs M was referred for cataract surgery with a plan to monitor her optic discs, IOP and visual fields post-operatively. However, after the right eye cataract surgery in December 2005, Mrs M was lost to follow-up again.

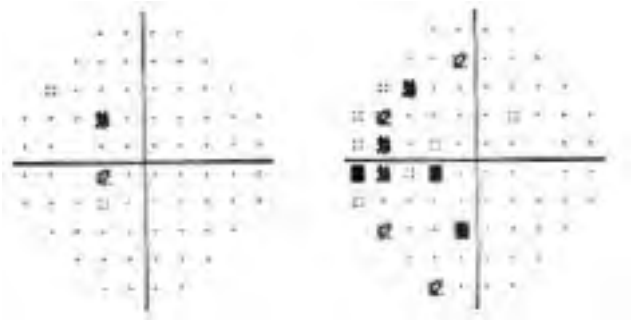

**Figure 1. January 2004**

**March 2009**

Mrs M returned to the optometry clinic and advised us that she had been prescribed Xalatan nocte R&L by a private ophthalmologist in 2007 with repeat prescriptions from her GP. Visual acuities were R 6/7.5 L 6/7.5. Optic discs showed further progression in disc damage with C:D R 0.8 L 0.9 and left inferior baring and notching as well as left inferior nerve fibre layer loss (Figures 2 and 3). There was progression in visual field loss compared to her first visit. The right nasal step had increased to an inferior arcuate defect. The left eye had developed a superior arcuate defect and inferior paracentral defect (Figure 4). On Xalatan monotherapy IOPs were R 17 mmHg L 18 mmHg. This was not a significant pressure reduction compared to her maximum pressures of R&L 21 mmHg. With IOP in the high teens there was demonstrated progression in the optic nerves and visual fields. Mrs M was no longer under private ophthalmological care. She was referred to public ophthalmology for assessment and management with recommendation to either change the prostaglandin or add a beta-blocker.

**May 2009**

The ophthalmologist agreed with the disc assessment: C:D R 0.8 L 0.9 with a left inferior notch. IOPs were R 18 mmHg L 21 mmHg, which were too high for the disc appearance. Compliance with the treatment regime was

# glaucoma

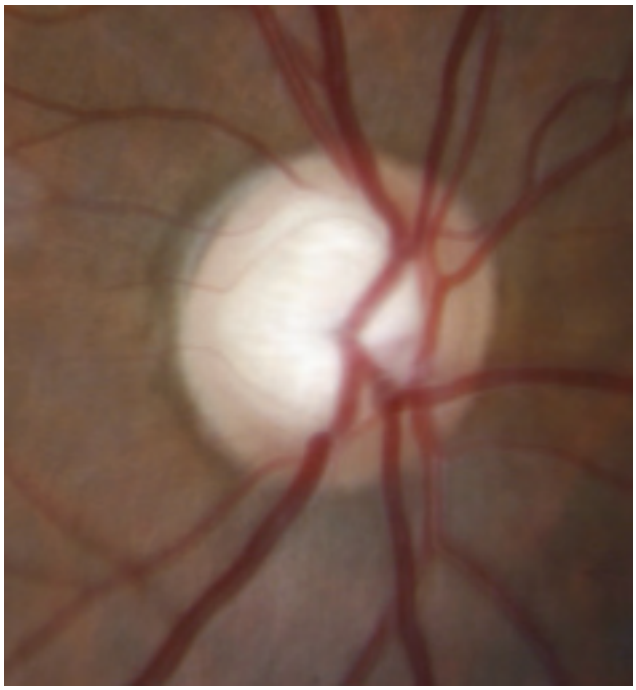

**Figure 2. Right optic disc in March 2009; C:D 0.8 with superior and inferior neuroretinal rim thinning**

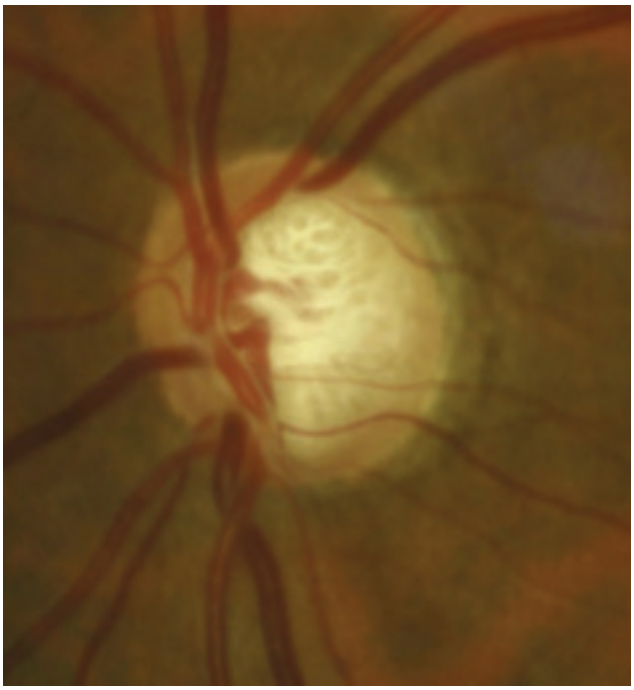

**Figure 3. Left optic disc in March 2009; C:D 0.9 with inferior notch in neuroretinal rim and inferior nerve fibre layer loss**

confirmed. Non-compliance was eliminated as a cause of progression. Mrs M's treatment was changed from Xalatan (latanoprost) to Xalacom (latanoprost and timolol) nocte R&L.

**July 2009**

Mrs M had no complaints and reported no breathing problems with the addition of the beta-blocker. Xalacom was effective in lowering the IOPs to R 14 mmHg L 14 mmHg. Pachymetry revealed thin corneas: R 489 µm L 486 µm. Visual fields showed the right inferior defect extending centrally. The left inferior paracentral defect was stable (Figure 5).

**September 2009**

Progression in optic disc damage was observed again. Cup-disc ratios were now R 0.9 L 0.95 with a left inferior notch. IOPs were R 14 mmHg L 16 mmHg. A target pressure in the low teens was set because of the advanced glaucomatous optic neuropathy. A lower pressure was needed for the left eye, so Alphagan (brimonidine) bd was added for the left eye and Mrs M was advised to also continue Xalacom nocte R&L.

**November 2009**

Optic nerves were stable and IOPs were at target (R&L 13 mmHg) with Xalacom nocte R&L and Alphagan bd L. Mrs M reported occasional itchy eyes.

**April 2010**

Mrs M returned to the optometry clinic for new spectacles. She had stopped Alphagan a couple of weeks prior to this visit because she had developed a red and sore left eye. She had a mild follicular reaction that was typical of an Alphagan allergy. IOP was R&L 14 mmHg, which was still acceptable even on Xalacom alone.

**June 2010**

At the next ophthalmologic review, IOPs were borderline acceptable at R&L 15 mmHg. Optic nerves showed no change. Azopt (brinzolomide) bd was prescribed for both eyes. Mrs M was advised to continue Xalacom nocte R&L as well.

**August 2010**

Visual fields were fairly stable with a right inferior defect and left superior/inferior paracentral defects (Figure 6). IOPs were R 12 mmHg L 11 mmHg, which showed good control with the three medications (latanoprost, timolol and brinzolomide). Optic nerves showed stable advanced glaucomatous optic neuropathy.

**December 2010**

IOPs were still at target (R&L 12 mmHg) and optic nerves showed no progression. Four-monthly reviews were recommended as long as the glaucoma was still stable. Mrs M found it difficult to travel to her ophthalmology reviews so comanagement was arranged with the optometry clinic.

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# Normal tension glaucoma

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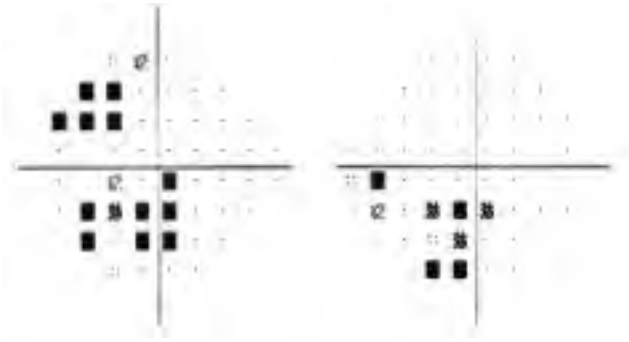

**Figure 4. March 2009**

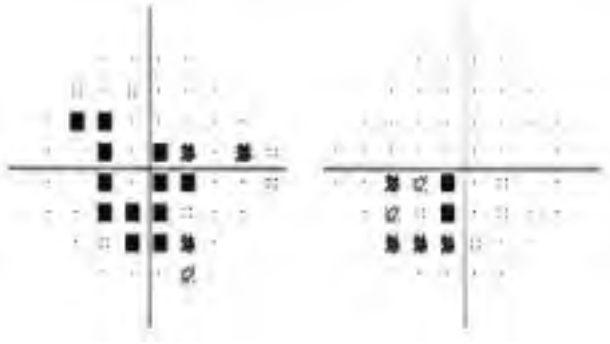

**Figure 5. July 2009**

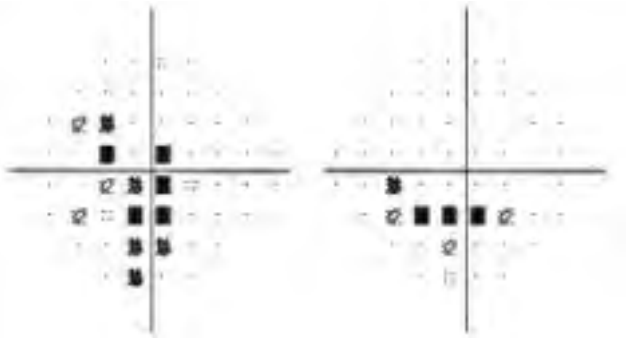

**Figure 6. August 2010**

## Diagnosis

The diagnosis of NTG for this patient was made on the basis of:

- optic nerve appearance characteristic of glaucomatous optic neuropathy: enlarged cup-disc ratio and documented thinning of the inferior and superior NRRs over time
- glaucomatous visual field defects consistent with the optic nerve head appearances: paracentral scotoma, arcuate scotoma, nasal step
- open anterior chamber angles on gonioscopy with no peripheral anterior synechiae
- IOPs less than 21 mmHg.

## Discussion

NTG is characterised by optic nerve changes and progressive loss of peripheral vision with IOPs below 21 mmHg. The Collaborative Normal Tension Glaucoma Study (CNTGS)[1,2] found that patients who were at risk of NTG progression were female, those with history of migraine (many of whom were female) and those with disc haemorrhages. There was also increased risk of progression with increasing age. All of these risk factors applied to this patient, except for disc haemorrhages.

Disc haemorrhages were never noted for Mrs M, although it is possible that a disc haemorrhage occurred and resolved during a period between reviews. There is a higher prevalence of disc haemorrhage in eyes with NTG than with other types of glaucoma or normal eyes.[3] Eyes with a disc haemorrhage showed significantly greater progression in visual field defects and in changes in optic nerve head contour for POAG, NTG and OHT.[4]

Central corneal thickness is significantly reduced in patients with normal tension glaucoma compared with patients with primary open angle glaucoma or healthy patients.[5] This patient had thin corneas (R 489 µm L 486 µm), which increased the risk for glaucoma progression.

## Management

The objective of NTG treatment is to reduce IOP to prevent further damage to the optic nerve and subsequent loss of visual field.

The CNTGS showed that IOP is involved in the pathogenic process in NTG and that treatment to reduce IOP is beneficial in patients who are at risk of disease progression. Lowering IOP by at least 30 per cent will slow the progression of glaucomatous visual field defects compared to untreated NTG.

Mrs M's treatment and target pressure were continually reviewed in the presence of documented progression of optic nerve damage and visual field loss. Target pressure was initially 14 mmHg which is 30 per cent below the highest IOP measurement (21 mmHg), in line with recommendations from the CNTGS. IOPs were on target with Xalacom and Alphagan for about 12 months. After Alphagan was ceased due to allergy, IOPs were borderline acceptable (15 mmHg) on Xalacom alone. The addition of Azopt gave better IOP control. IOPs were now 11-12 mmHg, which was consistent with NHMRC guidelines.


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What are your options when things do not go according to plan?

# Pathways for managing the complex glaucoma patient

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**The management of glaucoma is relatively straightforward in the majority of cases. Once a diagnosis has been made, a target pressure set and treatment initiated, most patients will have a steady and predictable course. Management consists of regular monitoring to ensure that their target pressures are appropriate for their stage of disease and that there are no clinical signs of progression of either optic nerve damage or visual field loss.**

What happens when things do not go to plan and either target pressures are not met or the patient shows progression? What are the management options when either the medication is not successful at lowering pressure or the patient does not tolerate the medication due to persistent side-effects, or when the patient is on maximal topical medications and the intraocular pressure is still too high? These are the patients who usually go on to have a surgical intervention to lower their intraocular pressure.

Non-penetrating glaucoma surgery and selective laser trabeculoplasty are perhaps exceptions as they can be performed as first line instead of medical management.

This article looks at surgical options that currently exist and from a clinical standpoint, factors that influence the selection of the most appropriate intervention for patients who fail medical management.

## Selective laser trabeculoplasty

Mr DW is a 53-year-old male with a past history of asthma and multiple drug allergies. He has a mother with advanced glaucoma. He is found on routine examination to have elevated intraocular pressure to 25 OU, a nasal step in the right visual field and a superior arcuate defect in the left visual field. His OCT shows peripapillary nerve fibre layer thinning and his HRT is abnormal in most sectors.

Traditional management consists of the commencement of pressure lowering medication such as one of the prostaglandin analogues; however, given this patient's history of medication intolerance and multiple medication allergies this approach may not be ideal. Due to the advanced glaucoma, it is highly likely that this may prove to be insufficient, requiring additional topical medication to be added, such as a carbonic anhydrase inhibitor like dorzolamide (the choice of additional medication is limited by his history of asthma). This approach is entirely reasonable and entirely contemporary. A more clinically reasonable approach—and one for which there is increasing evidence—is selective laser trabeculoplasty (SLT).[1,2,3,4]

SLT is a relatively non-invasive procedure that is performed in the consulting room. It takes only several minutes to complete and by and large is painless. There can be some minor discomfort for a day or so due to a low grade anterior uveitis but this is uncommon.

There are no long-term sequelae and recent studies have shown that unlike argon laser trabeculoplasty (ALT) it is repeatable as it does not cause any thermal injury. The pressure lowering effect is not permanent and with time, the patient will require a repeat application. Recent studies have demonstrated that its efficacy and duration of action are the same with repeat application.[5]

SLT overcomes the issues of compliance and the treatment is effective in greater than 80 per cent of individuals.[6,7] The effect of treatment is about equivalent to a single topical medication with a pressure lowering effect of 20 to 30 per cent but it some cases it may be much greater.[7]

The duration of effectiveness ranges from one to five years before it needs to be repeated.

If target pressure is not achieved with a single application, then additional SLT can be performed, which has been shown to be additive.[1]

The greatest response will be seen in patients with pseudoexfoliation or pigmentary glaucoma but it is also effective in POAG.[8]

A recent study has shown that it can be effective in NTG but the pressure lowering effect is not as great, averaging about a 20 per cent reduction and lasting only one to two years.[9]

Patients who are commenced on topical medication and then are switched to SLT have a poorer outcome than a comparable group who are treated with SLT as a primary treatment.[10]

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# Pathways for managing the complex glaucoma patient

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Patients who are already on topical medications but have not reached target intraocular pressure and show signs of disease progression, or in whom there is concern that they may progress, can have SLT instead of the addition of a second agent. In some cases, the SLT effect may exceed that of their topical medication alone, in which case medication can be discontinued. More often than not, this additional lowering effect is beneficial and the patient is kept on the topical agent in addition to their SLT.

## Cataract surgery to lower IOP

Mr FF is 78 years old and has a history of POAG. He is on a prostaglandin analogue and a fixed combination agent. His intraocular pressure is 20 in each eye and he has significant visual field loss in each eye. Recently he complained of difficulty with his vision, which an examination revealed was due to cataract formation. He has a BCVA of 6/9 right and 6/12 left.

Therapeutic options for his inadequate pressure control include the addition of a third agent such as an alpha agonist, changing him to a non-fixed combination such as a beta-blocker and carbonic anhydrase separately, SLT or trabeculectomy. A combined trabeculectomy and cataract procedure would also be a reasonable option.

Given that he had a symptomatic cataract, it was decided to proceed to cataract surgery alone.

Post-operatively his vision improved to 6/6 best corrected and his intraocular pressure fell by 20 per cent to 16 OU.

Cataract surgery has been demonstrated to be an effective means of lowering intraocular pressure.[11,12] The presumed mechanism of lowering is not well understood. The effect is more pronounced in patients with acute or chronic angle closure and in patients with higher pre-operative pressures. In patients in whom there is cataract formation who are deemed high risk for trabeculectomy and who require only a modest fall in pressure, cataract surgery may be a preferred option.

## Trabeculectomy

Mrs FW is a 66-year-old with a long history of PXF glaucoma. She had been reasonably managed for many years with topical medication and more recently had undergone SLT to her right eye. A period of good control was followed by increasing right intraocular pressure despite maximum topical medication. Oral Diamox had been added to her regime but she developed nausea and significant eye irritation to the drops.

Her right intraocular pressure has increased to 28 compared with 14 for her left eye. The right optic nerve is beginning to show glaucomatous damage with inferior rim thinning and enlargement of the cup. An additional application of SLT was trialled but this failed to bring down her very high intraocular pressure.

This patient is a classic example of glaucoma progression despite initiation of conservative medical management, and requires surgical intervention. A decision was made to surgically intervene with a mitomicin C trabeculectomy.

Trabeculectomy, despite newer procedures such as non-penetrating surgery, remains the gold standard for glaucoma surgery. This is primarily due to its relative safety, predictability and success rates of 80 to 90 per cent in lowering intraocular pressure.[13,14,15] When compared to other glaucoma surgical interventions it is still the most effective in long-term pressure control.[13,14,15]

Trabeculectomy is otherwise known as a guarded filtration procedure. It involves opening the conjunctiva, creating a partial thickness scleral flap or trapdoor adjacent to the limbus and then performing a full-thickness sclerostomy that results in a communication between the anterior chamber and the area under the flap.

The scleral flap is then sutured down sufficiently to allow a controlled amount of flow of aqueous from the anterior chamber. The conjunctiva is closed over the flap. Aqueous that leaves the anterior chamber collects under the conjunctiva, forming the familiar bleb.

The success or failure of trabeculectomy depends on a host of factors that include surgical expertise, regardless of whether it is performed de novo or as a repeated procedure, and whether there has been prior use of topical medication. Young patients tend to have a higher rate of surgical failure due to the combined effect of a strong healing response and their longer expected duration. Black race is also a poor prognostic factor with higher failure rates. Patients who have not been previously treated with topical medication or had their conjunctiva interfered with surgically have the best prognosis because both prior surgery and chronic medication lead to conjunctival fibrosis. Medication use also leads to chronic low level inflammation.

The body's own healing tendencies work against the success of the procedure and it is for this reason that topical 5 fluorouracyl (5FU) or mitomycin C (MMC) are applied to the surgical site at the time of surgery. Despite this there can still be a tendency for healing to occur, requiring further post-operative subconjunctival applications of 5FU.

Possible complications following this kind of surgery may include hypotony and bleb leak, which are more common if an antimetabolite has been used, particularly MMC. A thin bleb wall can develop over time with consequent leakage. This exposes the patient to post-operative blebitis and endophthalmitis. Blebs can also become large and can overhang, causing ongoing irritation.

The immediate post-operative period is tremendously important and careful observation and intervention during this period can mean the difference between success and failure.

Commonly, releasable sutures are placed in the scleral flap, allowing it to be sutured tight initially to minimise the risk of post-operative hypotony. These can be either selectively removed or lasered if a lower IOP is required. Long term, the result can be good with effective IOP-lowering into the single digits. Trabeculectomies can last for many years with patients still having effective control 15 years later. On the other hand, they can fail early with only several months or several years of good pressure control. They can sometimes fail in the first few weeks or not work at all.

Trabeculectomy surgery is not ideal in patients with uveitic glaucoma as the presence of the uveitis will often lead to scarring and failure of filtration. Patients with rubeotic glaucoma similarly are not ideal candidates for trabeculectomy, because the presence of the abnormal vessels in the trabecular meshwork leads to PAS formation with closure of the sclerostomy. During surgery the vessels in the angle will bleed during the procedure.

Normal pressure glaucoma patients are a unique group by virtue of their already low

intraocular pressure. Trabeculectomy can lower intraocular pressure into the single digits but this is difficult to achieve. Patients who have an already low pressure but show signs of progression may not necessarily benefit from further intraocular pressure lowering as other, non-pressure related mechanisms may be responsible for their glaucomatous progression.

## Tube surgery

Mrs HM is 35 years old. She has a long history of recurrent uveitis, each episode with an associated high IOP. Following each episode she has remained with an elevated pressure requiring topical medication. She presented with a recurrence in her left eye with an elevated pressure to 42.

Examination reveals two to three + cells with associated flare and keratic precipitates. There are posterior synechiae present and a gonioscopy examination also shows posterior anterior synechiae (PAS) to be also present.

She is commenced on Pred Forte hourly along with homatropine tds. For IOP control she is commenced on Combigan.

On review her inflammation shows signs of improvement but her IOP remains at 38. The left optic nerve is also showing signs of glaucomatous damage with visible loss of the neuroretinal rim and a deepening of the cup.

Her medications are changed to Lumigan, Cosopt and Alphagan. These also fail to have an impact on her intraocular pressure, which hovers around 32.

In this situation, surgery is clearly indicated as there are signs of optic nerve damage in the face of a poorly controlled intraocular pressure despite almost maximal medical therapy. The question of which procedure is less clear because of the uveitis.

Trabeculectomy would have been the ideal choice except that it will almost certainly eventually fail due to the inflammation causing wound healing. The alternative option is to perform a tube procedure such as a Molteno. The problem is that there can be a delay in the tube opening and thus lowering pressure, and it is a more challenging procedure. A decision is made to perform a trabeculectomy anyway as immediate pressure control is required. At a later date, a tube procedure would then be performed.

Tube surgery has been performed since 1988 when Molteno implants were first used. Tube surgery has traditionally been reserved for patients who had previously undergone conventional glaucoma surgery (trabeculectomy) which had subsequently failed. It is also the procedure of first choice in neovascular glaucoma where a trabeculectomy will almost certainly fail. It is also more likely to be used in patients with ICE syndrome.

Tube surgery consists of implanting a tube into the anterior chamber, which drains aqueous to the area under the conjunctiva much like a trabeculectomy. The tube enters the anterior chamber either under a scleral flap or is protected by the placement of donor sclera tissue (or cornea) over the top of the tube. Tube surgery is successful because a tube is less likely to fibrose like a sclerostomy as it is made of stiff plastic tube.

There are essentially four types of tube devices that fall into two groups. The first group is of those that contain valves such as the Ahmed (developed by Dr Ike Ahmed at the University of Toronto), or the Krupin device (developed by Theodore Krupin at the Northwestern University Medical School in Chicago). The second group is of those that are not valved and consist of just a tube with a plate such as a Molteno implant (developed by Dr Tony Molteno in Dunedin, New Zealand) or the Baerveldt glaucoma implant.

Valved devices were developed because of the tendency for hypotony to occur in the immediate post-operative period with the tube devices; however, there are techniques used in tube devices that prevent post-operative hypotony but at the cost of delayed onset of the pressure lowering effect.

This type of surgery is much more involved than a trabeculectomy with much longer surgical times. There are also potential complications that do not exist in trabeculectomy, such as the increased risk of corneal decompensation due to the presence of the tube in the anterior chamber. There is also the risk of inducing diplopia secondary to the presence of the plate, which is placed between the intraocular muscles.

Despite this, the tube-versus-trabeculectomy study (TVT)[16] showed that a tube procedure is almost as good as a trabeculectomy in its ability to lower intraocular pressure and is associated with less post-operative complications despite its greater difficulty to perform. It also found that there was a lower likelihood of failure than trabeculectomy and as such, it can be considered early in the management of glaucoma.

A newer non-valved device is the Optonol Ex-PRESS shunt which promised to solve some of the difficulties associated with tube surgery. This shunt creates a communication between the supraciliary space and the anterior chamber, potentially avoiding some of the complications associated with trabeculectomy. In practice, there have been problems associated with this and now surgeons who implant them do so under a scleral flap just like a conventional trabeculectomy. No significant differences in outcomes have been found between conventional trabeculectomy and Ex-PRESS shunt implant.

## Cyclodestructive procedures

Mrs MS is 95 years old. She has a history of glaucoma which has never been successfully controlled with topical medication. Medication initially consisted of timolol to which Trusopt was added. ALT was tried but was unsuccessful.

She was referred for a second opinion. At that review her left optic nerve was noted to be an almost total cup and her vision had dropped to 6/24 despite clear optical media.

Alphagan was added and then changed to Combigan. Unfortunately, significant irritation in the form of hyperaemia and lid excoriation necessitated that the Combigan be discontinued. Eventually Diamox was commenced, which lowered the IOP but was associated with nausea. On last review her vision had begun to again be affected, dropping to 6/48.

Under normal circumstances, a trabeculectomy would have been performed but due to her advanced age and advanced

**Continued page 46**

> **The management of glaucoma is relatively straightforward in the majority of cases. Once a diagnosis has been made, a target pressure set and treatment initiated, most patients will have a steady and predictable course.**

# Pathways for managing the complex glaucoma patient

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glaucoma, it was felt that the optic nerve would not survive the stress of the surgery.

A left cyclodestructive procedure in the form of cyclodiode laser was performed. Post-operatively her left IOP fell to 5 and eventually settled at 8. Her Diamox was discontinued and all the remaining topical medications were also able to be discontinued.

Cyclodestructive procedures are usually reserved for patients who have poor visual potential, who are elderly or frail and unable to undergo conventional surgery, who have advanced glaucoma with potential loss of vision secondary to surgery, who have blind painful eye, who suffer refractory glaucoma when other procedures have failed; and in glaucoma with a high failure rate, such as following retinal detachment surgery with silicone oil, or neovascular glaucoma.

There are three types of cyclodestructive procedures: cyclocryotherapy, cyclophotocoagulation (cyclodiode) and endoscopic cyclodiode.

- **Cyclocryotherapy**

Cyclocryotherapy was first performed by Bietti in 1950 and consists of freezing the ciliary body using a hand-held probe. Usually three quadrants were frozen leaving a fourth untouched to minimise the risk of anterior segment necrosis.

It worked by literally destroying the ciliary body.

Although effective, it resulted in a multitude of complications that included prolonged uveitis, hypotony and phthisis in more than 30 per cent of cases, choroidal detachment, cataract and many others. For these reasons, cyclocryotherapy has been largely abandoned in favour of the newer cyclodiode.

- **Cyclodiode**

This consists of a hand-held laser probe operating at a wavelength of 810 nm.

Usually from 16 to 18 applications of 1500-2500 mw power are applied to the ciliary body transclerally. The cilary body absorbs the laser energy and is destroyed by it. The sclera does not absorb the laser energy and is unharmed. This procedure is done in an operating theatre with a peribulbar block anaesthetic.

The complications are similar to those of cyclocryotherapy but much less severe. Most commonly, they are uveitis, loss of best corrected vision and hypotony with phthisis if over-applied.

A recent study[17] has looked at the use of cyclodiode as an alternative to trabeculectomy or tube surgery in patients with good visual potential. It has found that the amount of visual loss is no greater than that seen in patients who have undergone the more conventional procedures. It may be a good alternative, given that it is technically easier to perform and carries fewer complications.

- **Endoscopic cyclophotocoagulation**

Endoscopic cyclophotocoagulation is less commonly performed as it is more invasive.

It requires a small probe to be inserted into the posterior chamber via an incision made at the limbus. Viscoelastic material is introduced into the eye to allow the probe to be inserted into the posterior chamber. The main advantage of this approach is that it can more selectively treat the ciliary body and thus avoid some of the complications of the transcleral approach. As it requires entry into the eye, it carries all the risks of intraocular surgery such as endophthalmitis, choroidal haemorrhage and retinal detachment. For these reasons it is uncommonly performed and probably best reserved for patients undergoing intraocular procedures for other reasons.

## Non-penetrating surgery

Mr PS is a 45-year-old male with a history of POAG diagnosed at age 42 years. He is a high myope and has had stable disease for several years on a single agent. More recently intraocular pressures began to rise necessitating the addition of a second and even a third agent.

Eventually SLT was performed but intraocular pressures remained in the high 20s.

What are the therapeutic options?

A trabeculectomy would be the next logical step and would be entirely reasonable. As he is young a trabeculectomy has a greater risk of failure and even if successful may still fail with time. He is a high myope and therefore has a greater risk of complications such as choroidal effusions and antimetabolite complications owing to his probably thin sclera. A decision was made to perform non-penetrating glaucoma surgery (NPGS).

NPGS was developed primarily to address the safety issues associated with conventional glaucoma surgery. It works by enhancing the natural outflow of aqueous through conventional outflow channels and reducing the resistance which is encountered in the inner wall of Schlemm's canal and the juxtacannalicular trabecular meshwork.

NPGS currently take two forms: deep sclerectomy and viscocanalostomy. Neither is routinely performed in this country and very few Australian ophthalmologists have even attempted them. This is due to the combination of a very steep learning curve and a percption of being less useful.

## What is NPGS?

The essential feature of NPGS is that there is no direct communication between the anterior chamber and the subconjunctival space like there is in a trabeculectomy or tube surgery. Aqueous still has to filter through the trabecular meshwork and the inner wall of Schlemm's canal and so there is a natural barrier preventing infection and hypotony.

Deep sclerectomy like a conventional trabeculectomy involves taking down conjunctiva and creating a scleral flap to about 30 per cent depth. A deep sclerectomy is then created under the initial flap, which consists of a second flap slightly smaller than the first. At the anterior end of the flap, Schlemm's canal is unroofed and the inner wall of Schlemm's canal is then stripped of its juxtacanalicular trabeculum and endothelium. Finally, the deep scleral flap is removed, a collagen implant is sutured to the scleral bed and the superficial flap is sutured closed along with the conjunctiva.

Viscocanalostomy is a variant of deep sclerectomy in which a high viscosity sodium hyaluronate (Healon GV) is injected in the canal of Schlemm to increase filtration via this route. Aqueous humour is able to pass through the trabeculo-Descemet's membrane window into the scleral bed and can diffuse into the uveoscleral outflow system adjacent to it.

## Indications for NPGS

Penetrating glaucoma surgery (trabeculectomy and tube surgery) and non-penetrating glaucoma surgery (deep sclerectomy and viscocanalostomy) each has its benefits and downsides.

Trabeculectomy carries with it an increased rate of cataract formation. In an elderly patient with pre-existing lens opacity

this is less of an issue, particularly given that cataract surgery is a proven and effective approach to lowering intraocular pressure. In a younger patient, this is undesirable.

NPGS carries a much lower risk of cataract formation and is an ideal choice in a younger age group.

Younger patients with glaucoma who are treated with topical medications will have decades of exposure to medications that will consequently lead to conjunctival scarring which in turn will lead to a greater risk of subsequent failure of surgery, should it be required. Thus a compelling argument can be made to intervene early in these patients with NPGS avoiding topical medication use.

High myopes are also good candidates for NPGS as they have large globes and are at greater risk of choroidal effusions following trabeculectomy. This risk is virtually non-existent with NPGS.

Patients with aphakic glaucoma are also ideal for NPGS as trabeculectomy requires a surgical peripheral iridotomy to be performed as part of the procedure. This increases the risk of vitreous prolapse, through the iridotomy with consequent obstruction of aqueous outflow through the sclerostomy. As no iridotomy or, indeed, sclerostomy is performed in NPGS, this risk is eliminated.

Finally, both pigmentary glaucoma and pseudoexfoliative glaucoma patients benefit from NPGS as there is an opportunity to remove pigment or PXF material from the canal of Schlemm at the time of surgery.

With time, NPGS may come to dominate glaucoma surgery but is not likely to replace it, especially in difficult cases with very high pressures. The steep learning curve will mean that it will not replace trabeculectomy any time soon.

## Summary

Once a diagnosis of glaucoma has been established, the initial management in the majority of cases is relatively straightforward. SLT and non-penetrating glaucoma surgery can be offered as first line treatments as an alternative to topical medication or as a second line if medication fails. In patients in whom glaucoma is either difficult to control or medical therapy has clearly failed, it is important to consider glaucoma surgery.

There are numerous surgical options available to the glaucoma surgeon faced with these cases and ultimately the choice of procedure is largely determined by the particular clinical circumstances of the patient.


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# Technology 1, Glaucoma 0

**From page 35**

Three tests are essential components in glaucoma monitoring.

- Recent developments in OCT make it possible to monitor subtle changes in not only the nerve fibre layer but also more importantly the thickness of the ganglion cell layer, which is the retinal layer to suffer first from decreased nourishment.
- Instruments that perform non-contact tonometry and pachymetry now afford optometrists these essential measurements with accuracy but without anaesthetic.
- Perimetry still has a place in early detection now that blue on yellow (SWAP) field screeners are available as a more sensitive alternative to more familiar white-on-white methods.

In the current environment of increased demands on our profession, the ability to use technology in providing comprehensive care of the glaucoma suspects among our patients is an excellent means to differentiate the service you offer.

Early detection and management of glaucoma is possible like never before for practitioners who invest in the latest technology and want to practise at the cutting edge to achieve optimal patient outcomes.

# OCT's role

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Product manager
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Carl Zeiss Australia

**Optical coherence tomography (OCT) produces high-resolution, real-time, three-dimensional images of ocular structures *in situ*. It was first commercialised by Carl Zeiss in 1995. Today with the current spectral domain systems, its uses are vast.**

One application of OCT is in the realm of glaucoma. OCT is an ideal technology with which to image and quantify relevant anatomy related to glaucoma such as retinal nerve fibre layer, optic nerve head, ganglion cell layer, central corneal thickness and irido-corneal angle. Discussion of all of these parameters is too lengthy to be covered in this article, so it is restricted to retinal nerve fibre layer and optic nerve head. It is important to note that different OCTs handle these measurements in different ways, but this article will use the Cirrus HD-OCT to illustrate each example.

## Retinal nerve fibre layer

There are a few ways to measure retinal nerve fibre layer (RNFL) using OCT, including simply taking a circle scan around the optic nerve head or scanning a whole cube of data over the optic nerve head. The Cirrus HD-OCT, for example, does both.

When interpreting the RNFL circle scan, it is important to note two things. First, it has been shown in the literature that the inferior portion of the circle is the most sensitive in picking up early glaucomatous RNFL defects and this has been known since early studies on the Stratus OCT.[1,2] However, placement of the circle can be critical and an incorrectly placed circle can affect RNFL measurements and longitudinal reliability.[3] The Cirrus HD-OCT, for example, avoids this problem by taking a cube scan of the entire six-by-six millimetre area and using software to automatically place the measurement circle retrospectively, thus achieving accurate and repeatable measurements with standard deviations of 1.3 to 1.9 microns[4,5] (Figure 1).

Importantly, a recent journal article has shown that almost 20 per cent of nerve fibre layer defects do not involve the conventional measurement circle, and would be missed if a cube deviation map were not used.[6] The cube gives important qualitative data to reveal the distribution of an RNFL defect and this should not be ignored. The RNFL Thickness Map and Deviation from Normal Map on the Cirrus HD-OCT are designed for this purpose. Both are colour-coded, the former to show the distribution of RNFL thickness and the latter to determine whether any region is thinning significantly compared to an age-matched normative database (Figure 1).

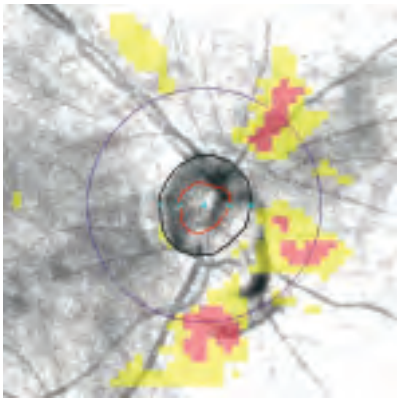

**Figure 1. The purple measurement circle is placed automatically by the software post-capture; the red and yellow super-pixels show areas with significantly thinning RNFL**

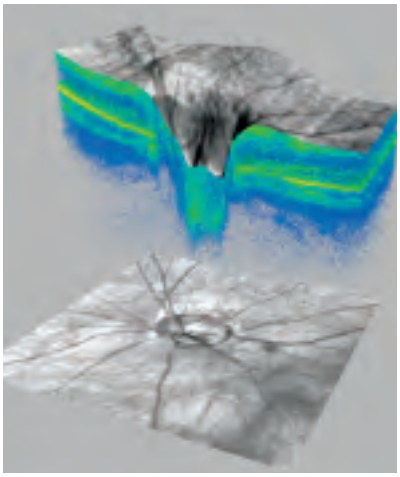

**Figure 2. 3-D visualisation of the optic nerve head**

## Optic nerve head

Although the optic nerve head can be observed using the ophthalmoscope or fundus photography, OCT can add additional information that cannot be gathered by other means. First, OCT can automatically calculate the disc and cup margin. The Cirrus HD-OCT, for example, defines the disc margin by the termination of Bruch's membrane and the cup margin by measuring the amount of neuro-retinal tissue in the optic nerve. From these landmarks, indices such as neuro-retinal rim volume and cup/disc ratios can be measured. It is here that OCT can bring additional information to the table, especially for tilted discs. When observing a significantly tilted optic nerve head via ophthalmoscope or fundus photography, the clinician has a foreshortened view and cannot see the true dimensions. Because it possesses a three-dimensional data set, the Cirrus HD-OCT, for example, measures optic nerve head parameters in the same plane as the disc, thus measuring more realistic dimensions.

Additionally, it is noteworthy that disc area has a more significant effect on the normative data than age. The Cirrus HD-OCT takes this into account by matching its normative data not only to age but to disc area as well (Figure 2).

## Progression

The progression of glaucoma is arguably the most important parameter to measure. It is important that an OCT registers each scan so that it is measuring the same anatomy, point-for-point each time. It is also important to note what is defined as significant change. The Cirrus HD-OCT has a specific program for this purpose call Guided Progression Analysis (GPA). Of note, GPA takes into account its own test-retest variability and flags change only if the thinning of the RNFL is beyond that test-retest variability, thus minimising the risk of false positives. It also shows the rate of change by applying a linear regression analysis to the progression plot. In this way, the program not only flags if change is happening but also reveals the rate of change (Figure 3).

## Structure/function relationships

OCT also offers the possibility to compare the structure and function relationship of glaucoma by comparing the OCT parameters to the patient's visual field. This can be done manually but the Cirrus HD-OCT has taken it one step further by combining its raw data with that of the Humphrey Field Analyzer (HFA), creating a separate report that neither instrument alone can produce (Figure 4). This enhances the understanding of the complex relationship between the two on a single report created within the FORUM Eye Care Data Management system.

These are but a few aspects of OCT relating to glaucoma that can be used in conjunction with an overall glaucoma examination to assist clinicians to make informed clinical decisions for their patients.


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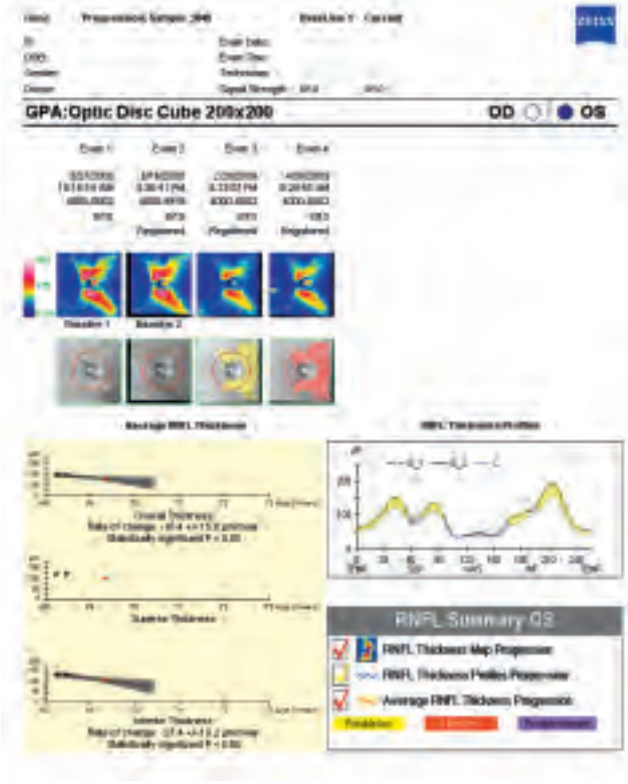


**Figure 3. Guided Progression Analysis report**

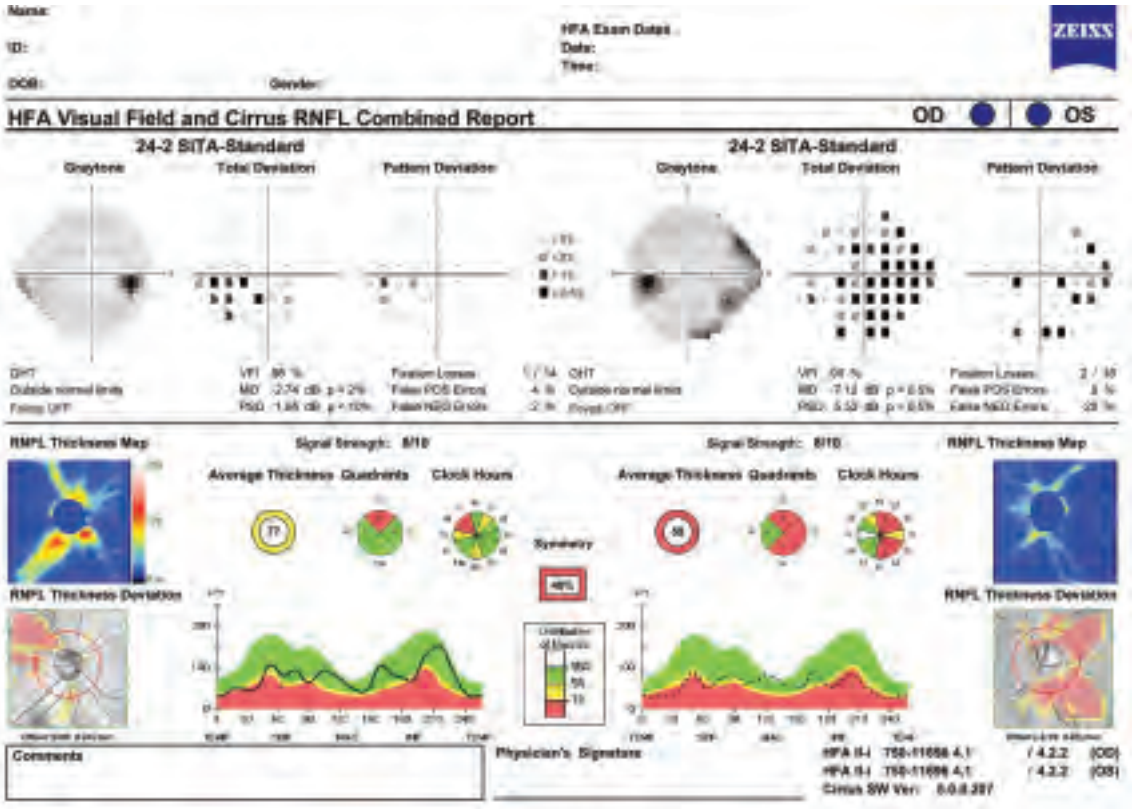


**Figure 4. HFA Visual Field and Cirrus RNFL Combined Report**

# Structural analyses of with

**Robin Lanesman**
BOC Instruments

**Glaucoma is a progressing optic neuropathy characterised by loss of retinal ganglion cells and their axons. These structural changes commence well in advance of functional vision loss and up to 50 per cent of ganglion cell loss may occur before detectable visual field loss.[1] OCT technology provides micron-scale quantification and monitoring of the layers associated with the ganglion cells and peripapillary RNFL, and morphology of the optic disc including the cup and neuro-retinal rim. Earliest glaucoma detection induces timely treatment to avert, slow or delay further damage.**

## Ganglion cell complex analysis

The macular region contains over 50 per cent of retinal ganglion cells and is an ideal region to evaluate early cell loss and monitor damage over time. The inner retinal layers collectively termed the Ganglion Cell Complex (GCC) comprise the nerve fibre layer (cell axons), ganglion layer (cell body) and inner-plexiform layer (cell dendrites). Each of these layers is affected in glaucoma, becoming thinner as the ganglion cells die out from glaucoma with loss of their axons and dendrites. Diagnosis of glaucoma is improved by concentrating on the inner retinal layers as the outer retinal layers are virtually unaffected by glaucoma.

The Optovue RTVue GCC scan analyses the thickness of these layers against an extensive ethnic grouped and age matched normative database to provide three colour-coded maps: Thickness Map, Deviation Map showing percentage of GCC loss and a Significance Map showing significance from normal. As patients may not necessarily have good target fixation, the GCC scan may be off-centre; however, the RTVue provides automatic alignment of the whole scan in horizontal and vertical directions to recentre on the fovea pit for accurate normative database comparison and progression analysis.

Significant GCC loss may present in a non-glaucomatous eye with high myopia and stretched retina although thinning is typically diffuse with no arcuate pattern. Numerous conditions also causing axonal degeneration and GCC loss include non-glaucomatous optic neuropathy, ONH drusen or neurodegenerative disease such as multiple sclerosis.

## Optic nerve head analysis

The Optovue RTVue ONH map provides thickness mapping of the RNFL automatically centred to the geometric centre of the disc. Software algorithms determine the disc margins from the termination points of the RPE and Bruch's membrane from the acquired 3-D disc scan.

RTVue software provides separate

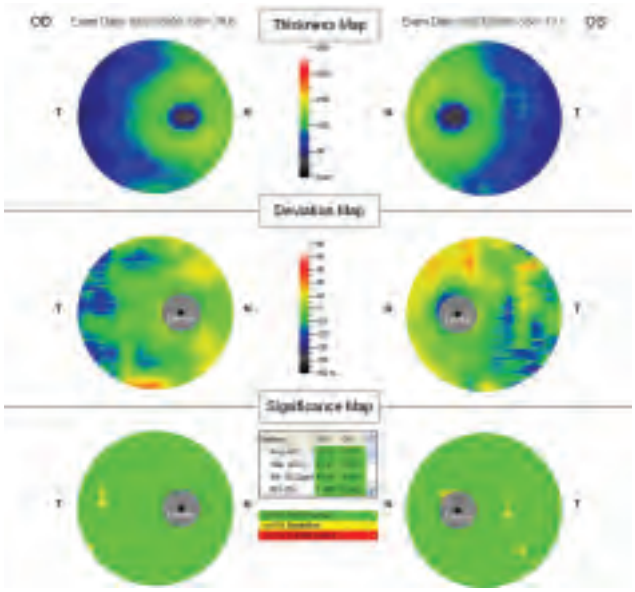


**Figure 1. RTVue GCC OU Report: GCC Thickness Maps, Deviation Maps and Significance Maps for normal GCC thickness. GCC parameters colour coded in green.**

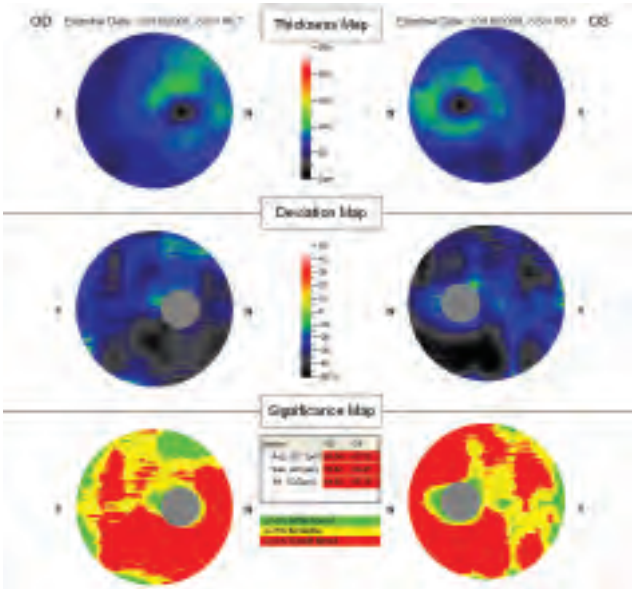


**Figure 2. RTVue GCC OU Report: glaucoma patient. Deviation maps show up to 50 per cent GCC loss (black areas) and large red areas in the GCC significance map reveal extensive damage. GCC parameters colour coded in red.**

# optic neuropathy Fourier domain OCT

normative databases derived from eight ethnic groups. The normative range of each ethnic specific database is narrower than the combined normative database with all individuals, providing increased accuracy when using the appropriate ethnic specific database. Significant correlations were found for age and RNFL thickness as well as age and various optic disc parameters (C/D ratio, cup area and rim area). Each database automatically adjusts the normative range and cut-offs to account for both age and optic disc size effects.

The signal strength of the scans was found to be correlated with age where older individuals tended to have weaker signal strengths due to reduced media clarity; however, analysis of normative data shows that signal strength has virtually no effect on measured RNFL thickness, which means that a weaker scan signal does not affect RNFL thickness measurement.[2]

The RNFL thickness map shows if a nerve fibre bundle pattern is typical or atypical. RNFL sector analysis may present with one or more sectors outside normal limits in a non-glaucomatous eye if a nerve fibre bundle is split or has an atypical pattern.

Mapping and analysis of both ONH & GCC scans may be viewed together in a combined comprehensive OU report. This report shows RNFL thickness maps, sector RNFL analysis, RNFL parameters, Optic Disc Parameters, TSNIT Graph, TSNIT Symmetry Analysis, GCC Significance Map and GCC parameters.

## GCC or RNFL: earliest indicator

Studies have shown that GCC thickness shows excellent diagnostic validity,[3] is highly correlated with pRNFL thickness and significantly correlated with visual field defects.[4] Clinical evidence advocates that GCC parameters provide greater accuracy and indicative of earlier structural damage for detection of early glaucoma compared to RNFL parameters.[5] This supports an established theory that axonal flow of brain derived neurotrophin factor (BDNF), critical for maintenance of health of ganglion cells, is restricted at the lamina cribrosa resulting in ganglion cell death and later followed by loss of retinal nerve fibre.[6]

## Progression analysis

For reliable monitoring and management of glaucoma, OCT measurements must be reproducible, the images must be accurately registered to each other and a statistical test must be implemented to differentiate true change from normal measurement variability. Studies performed with the RTVue show excellent reproducibility.[7] The RTVue registers ONH scans to each other based on matching vessel patterns to align scans to each other vertically, horizontally and rotationally, and a Statistic Image Mapping (SIM) test adapted from neuro-imaging is applied for detecting true change.

**Continued page 52**

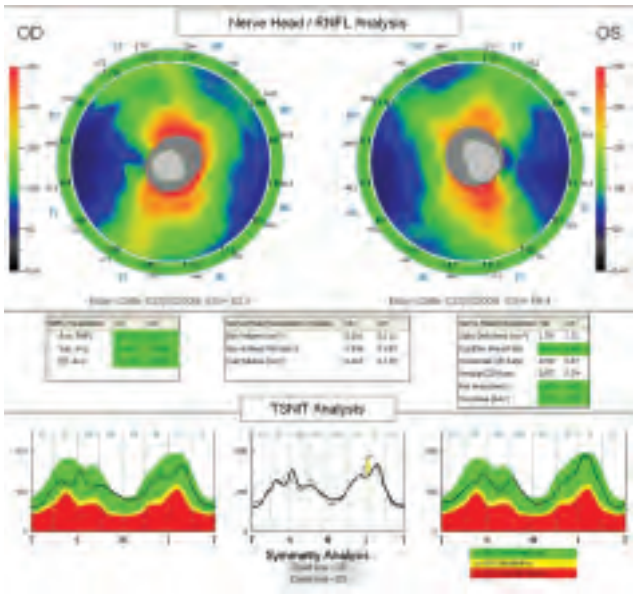


**Figure 3. RTVue ONH OU Report: healthy RNFL. Normal RNFL Map showing thickness and pattern of RNFL bundles. All pRNFL sectors in green and TSNIT Graphs well within green zone. RNFL and cup/disc parameters colour coded in green.**

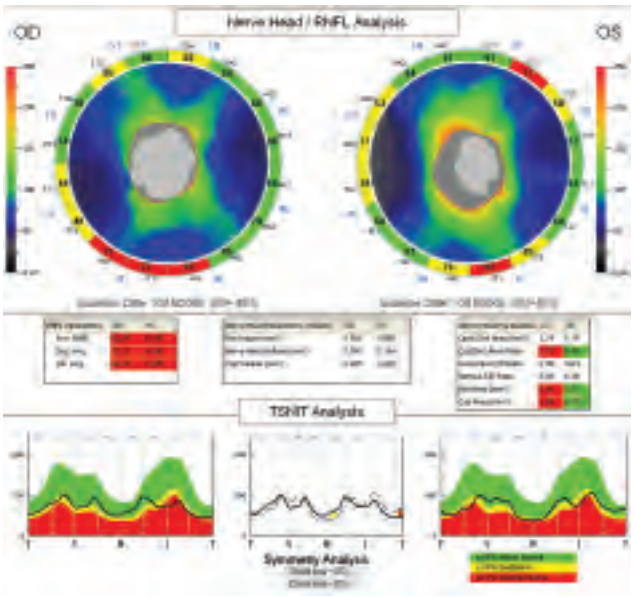


**Figure 4. RTVue ONH OU Report: glaucoma. RNFL colour coded map shows RNFL loss and sector analysis shows significant loss. TSNIT Graphs dip into red zone. RNFL parameters colour coded in red. Large cup RE (light grey area) and cup/disc parameters for RE colour coded in red.**

# Structural analyses of optic neuropathy

**From page 51**

GCC Significance Map: probability map indicating statistical significance of GCC loss; colour coded where green is normal GCC thickness, yellow is border line, and red is outside normal limits

TSNIT Graph: RNFL thickness profile at 3.45 mm around centre of optic disc (black line), superimposed on colour coded normative database

RNFL Thickness Map: colour coded where brighter colors (red and orange) represent thicker areas and cooler colors (blue and green) represent thinner areas

16 Sector RNFL Analysis: local RNFL thickness values compared to the normative database and colour coded; normal (green), yellow (borderline) and red (abnormal)

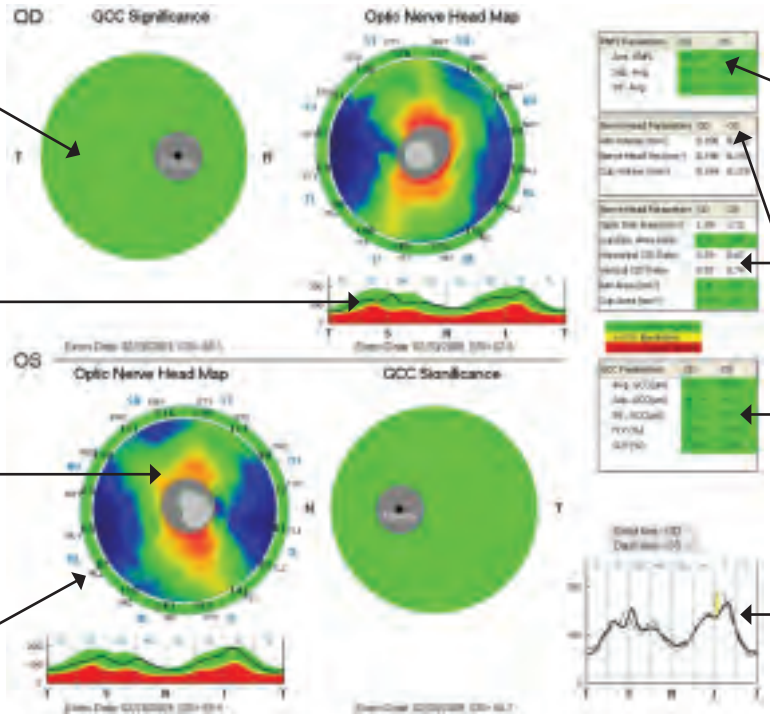


**Figure 5. Combined RNFL and GCC OU Report: normal eye**

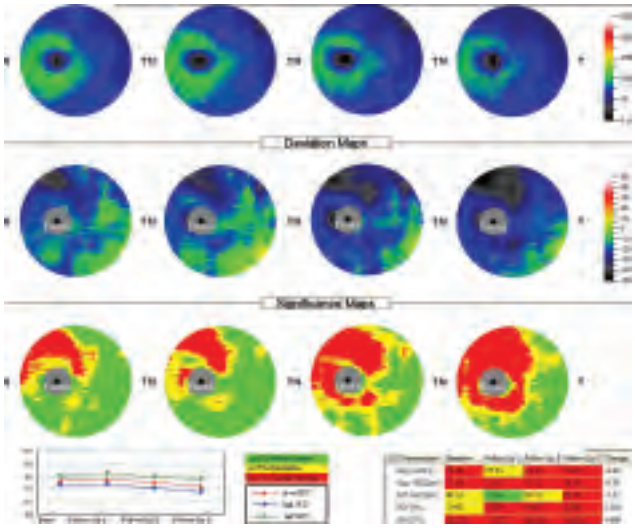

**Figure 6. GCC Progression Analysis: graph shows average, superior and inferior GCC changes over time**

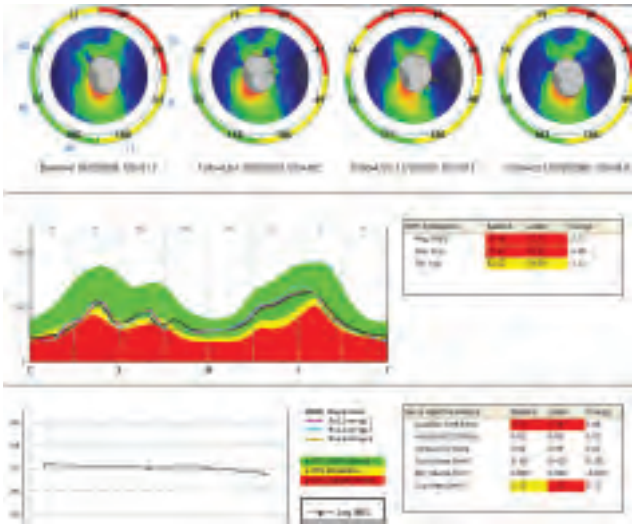

**Figure 7. ONH Progression Analysis: graph shows RNFL change over time**


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Pfizer Ophthalmics

Photos by dave.tozer.com

# A good example of glaucoma

**In June-July 1996 I did bilateral PRK for a 57-year-old woman. She was myopic with refraction R -4.00 = 6/5 L -4.00 = 6/5. Intraocular pressures were Goldmann 20 R and L.**

In those days we assessed corneal topography with the EyeSys. This instrument derived a topographic map from anterior surface Placido reflections. We did not routinely measure corneal thickness.

I recorded her optic discs as normal and did not make any assessment of cupping. I targeted slight residual myopia and she was thrilled with her outcome.

December 1996: Unaided acuity was R 6/9, L 6/6, binocular 6/5. IOP were 12 R and L. Refraction was R -1.00 = 6/ L -0.5 = 6/5.

## Case report

July 1997: Vision was the same. IOP were 18 R and L.

June 1998: Vision the same, IOP 18 right and left, optic discs 'normal'.

March 2000: Vision the same, IOP 18 R and L, optic discs healthy with small central cups. Patient was advised to return in one year for routine examination.

Patient did not attend until October 2004. She was very happy with her vision, unaided 6/6, 6/5 but IOP R 24, L 28, central corneal pachymetry R 511, L 513. Optic disc cupping R 0.8, L 0.5. Humphrey visual fields R normal, L infero-nasal defect.

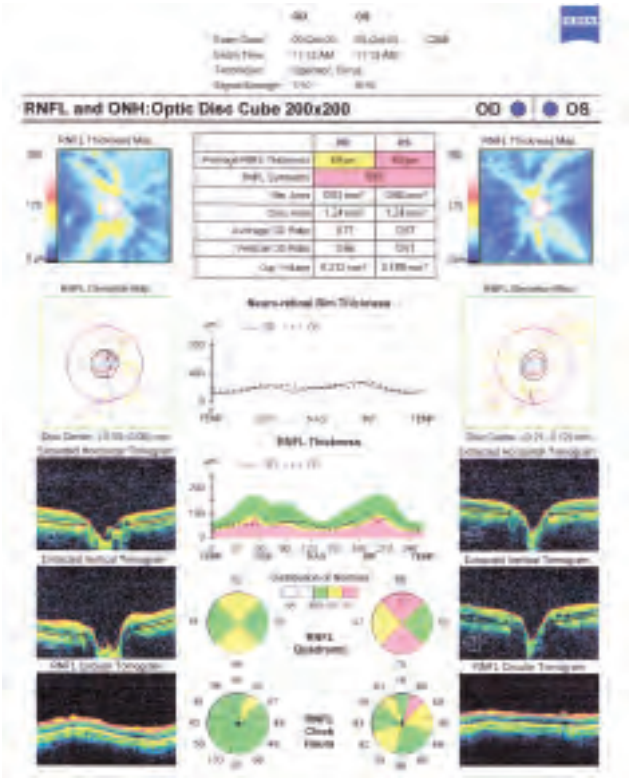
Diagnosis, primary open angle glaucoma. Treatment commenced, Xalatan at night both eyes.

Over the ensuing year pressures were 17 to 19 R and L.

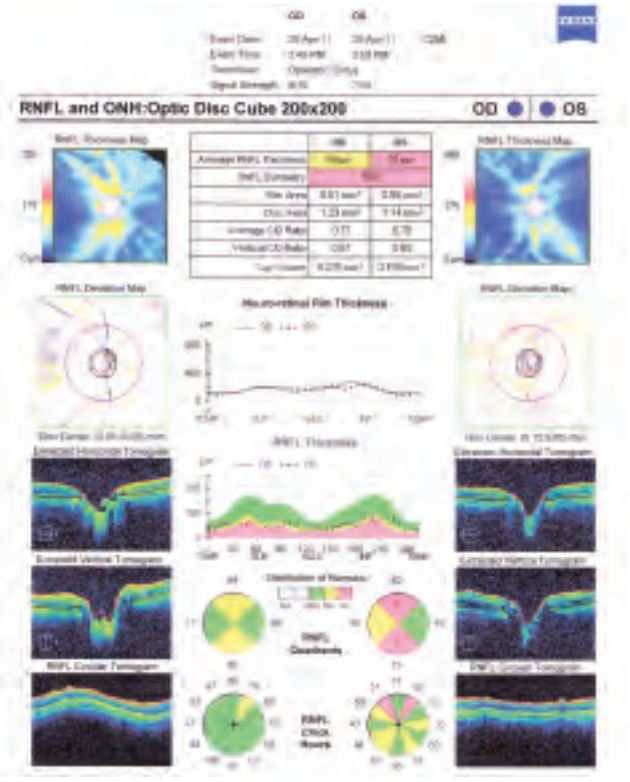
Nyogel was added in 2006 attaining lower pressures of about 16.

I switched to Xalacom in 2007 and pressures remained in range 16 to 18 until January 2009.

At that visit pressures were R 19, L 24. I did two sessions of selective laser trabeculo-



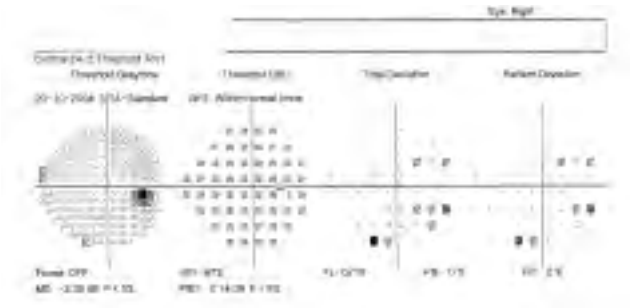

OCT 2009



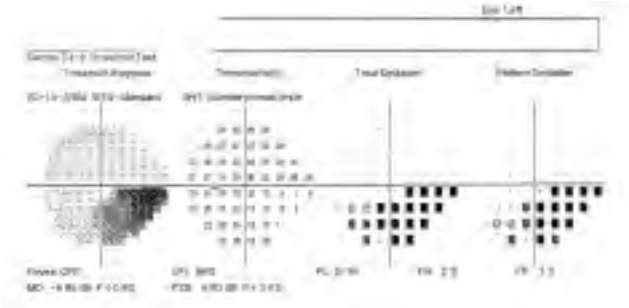

OCT 2011

# long-term management

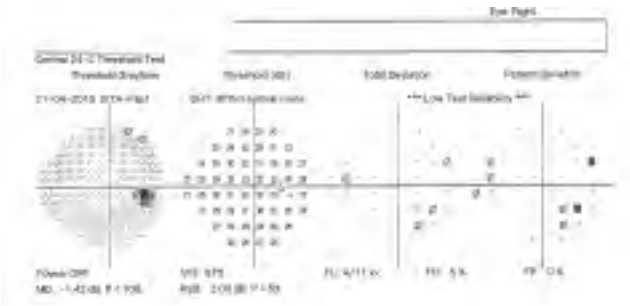
**Dr Richard Smith**
Director Sydney Eye Specialist Centre
MBBS BSc DO FRCS FRACS FRANZCO



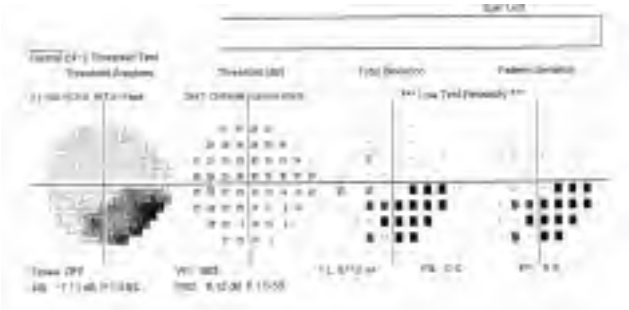
**Visual field, right 2004**



**Visual field, left 2004**



**Visual field, right 2010**



**Visual field, left 2010**

plasty to the left eye and pressures remained around 18 in each eye until November 2010 when they were 22 R and L.

I added Azopt twice daily and continued Xalacom.

By now she is 73 years old and vision is declining due to cataracts, unaided 6/9, 6/9 poor quality vision.

April 2011: bilateral cataract surgery scheduled for May 2011.

She has had normal gonioscopy and her visual fields have not worsened since glaucoma was diagnosed in 2004. Zeiss Cirrus OCT scans in October 2009 and April 2011 were almost identical, showing substantial nerve fibre layer thinning on the left consistent with her left field defect and borderline thinning on the right consistent with her normal right field.

It is likely that cataract surgery will bring some reduction in her pressures although this is not always the case.

## Points to consider about this case

- Periodic routine examination is important, particularly over the age of 45 years. Glaucoma is usually a silent condition and in my opinion is the main reason for periodic examination. Most other conditions give rise to symptoms that may lead a person to seek help.
- Beware of glaucoma in people who have had corneal laser surgery, that is, LASIK and PRK. They have thinner corneas and show false low pressures. It is desirable to measure central pachymetry as well as pressures and we now do this routinely.
- Our most important signs in diagnosing and monitoring glaucoma are optic disc appearances, visual fields, OCT or other imaging of discs and gonioscopy. OCT is proving very helpful.
- Despite the late diagnosis in this case, management has been successful in preventing progression. It is a good example of long-term glaucoma management. ■

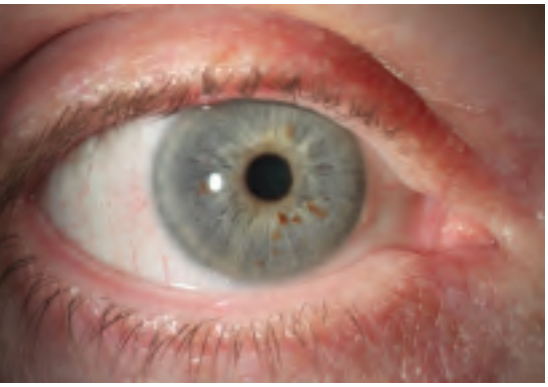


Figure 1. Normal appearance of the right eye

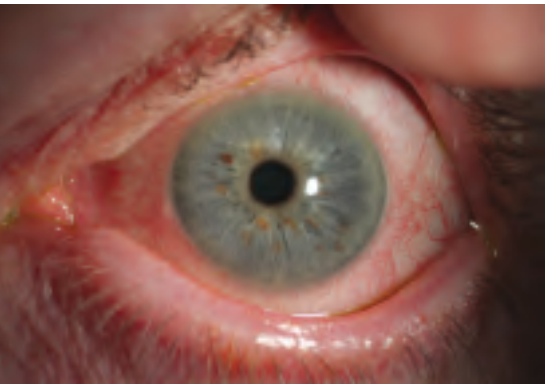


Figure 2. Widespread conjunctival redness on the left eye

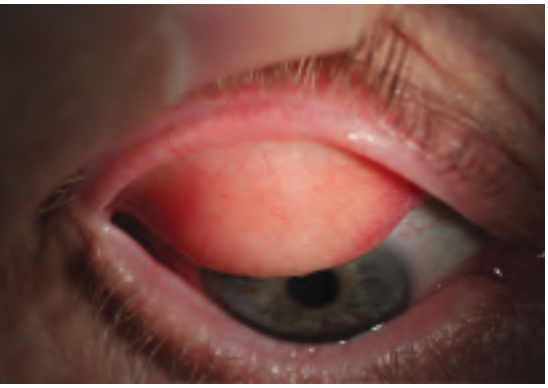


Figure 3. Upper tarsal conjunctiva right eye

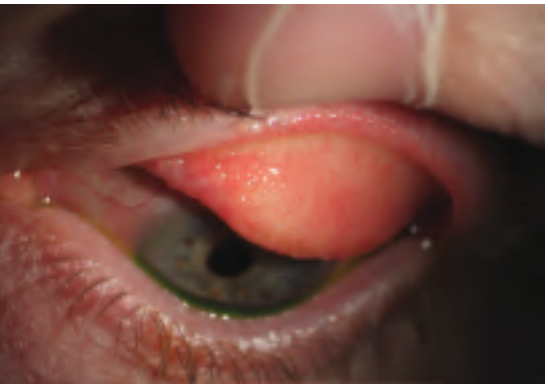


Figure 4. Upper tarsal conjunctiva left eye

# Allergic conjunctivitis secondary to Combigan eye-drops

**Gavin J O'Callaghan**
MOptom FAAO FCCLSA
Norwood SA

**Patient IR (DOB 12.01.1952 male) attended for assessment of a recently developed red left eye. He had an ophthalmic history significant for primary open angle glaucoma with treatment by his ophthalmologist with selective laser trabeculoplasty .**

This had brought his intraocular pressure under control (measured R 12 L 11 mmHg on the day of presentation). In addition, he had been prescribed Combigan eye-drops for the left eye only as a neuroprotective agent.[1]

On examination he had a normal gross appearance on the right side (Figure 1), contrasting with widespread bulbar conjunctival redness on the left (Figure 2). His anterior chamber was quiet, intraocular pressures were as noted above and cornea was clear. His right upper lid eversion showed a normal tarsal conjunctiva (Figure 3), whereas the tarsal conjunctivae on the left showed a follicular response with associated redness (Figures 4 and 5).

He was diagnosed with allergic conjunctivitis secondary to the medication and referred back to his ophthalmologist for review and to consider alternative glaucoma medications.

Follow-up from the ophthalmologist indicates that he was switched to Betoptic eye-drops for that eye, as it also has been claimed to have neuroprotective properties.[2]

Allergic conjunctivitis secondary to one of the agents (brimonidine tartrate) has been noted in the literature as a reasonably common side-effect of this drug, although reduced in the combined preparation this patient was using.[3]

Optometrists reviewing glaucoma patients should be alert to early signs of allergic conjunctivitis or other ocular surface changes secondary to topical glaucoma therapy.

1. Saylor M, McLoon LK, Harrison AR, Lee MS. Experimental and clinical evidence for brimonidine as an optic nerve and retinal neuroprotective agent: an evidence-based review. *Arch Ophthalmol* 2009 Apr; 127: 4: 402-406.
2. Osborne NN, DeSantis L, Bae JH, Ugarte M, Wood JP, Nash MS, Chidlow G. Topically applied betaxolol attenuates NMDA-induced toxicity to ganglion cells and the effects of ischaemia to the retina. *Experimental Eye Research* 1999; 69: 3: 331-342.
3. Sherwood MB, Craven ER, Chou C et al; for Combigan Study Groups I and II. Twice-daily 0.2% brimonidine-0.5% timolol fixed-combination therapy vs. monotherapy with timolol or brimonidine in patients with glaucoma or ocular hypertension: a 12-month randomized trial. *Arch Ophthalmol* 2006; 124: 1230-1238.

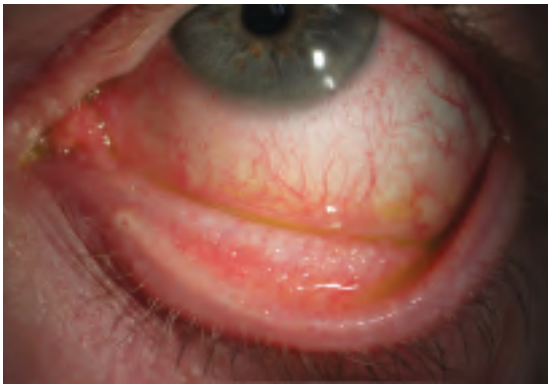
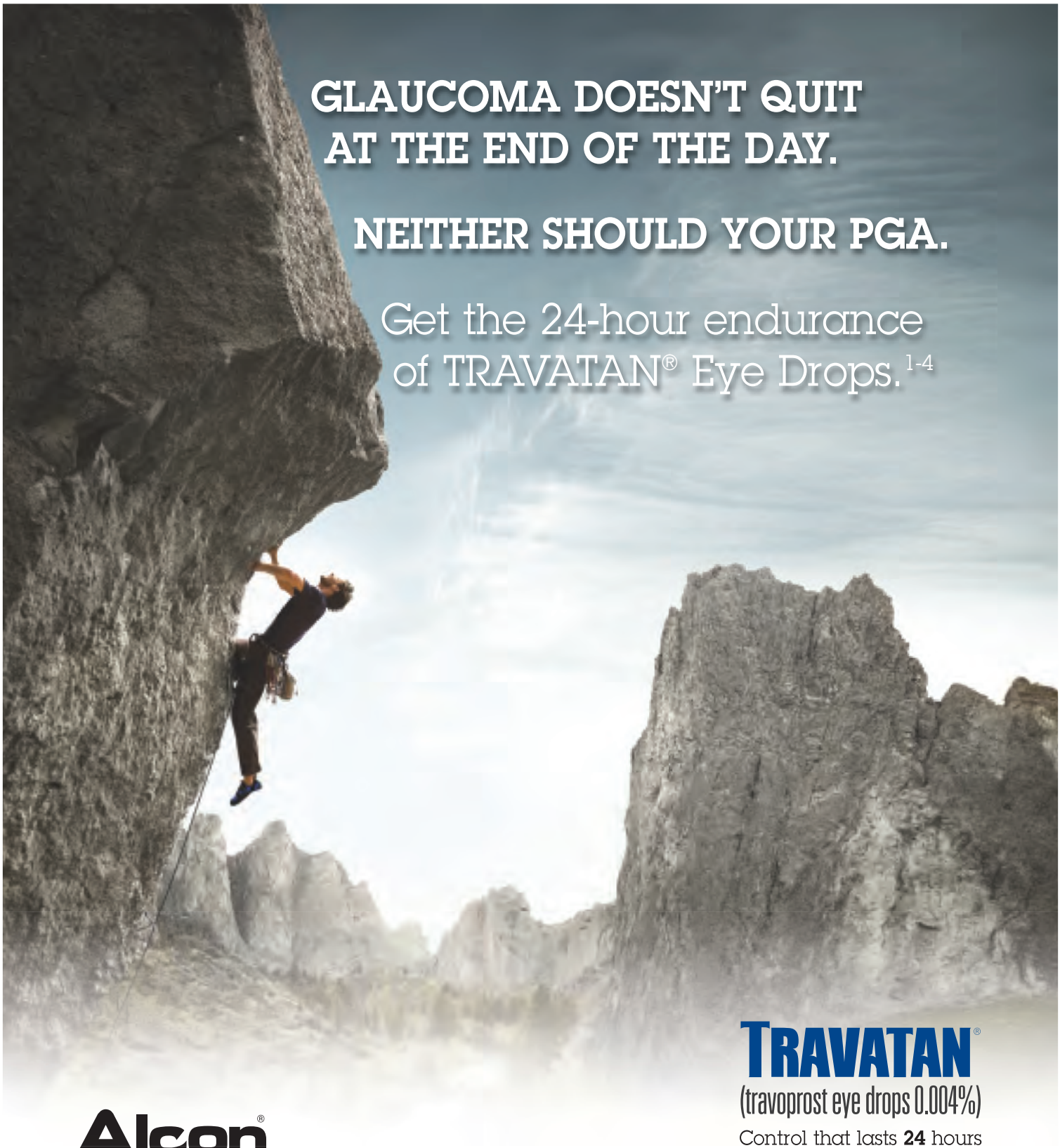


Figure 5. Lower tarsal conjunctiva left eye

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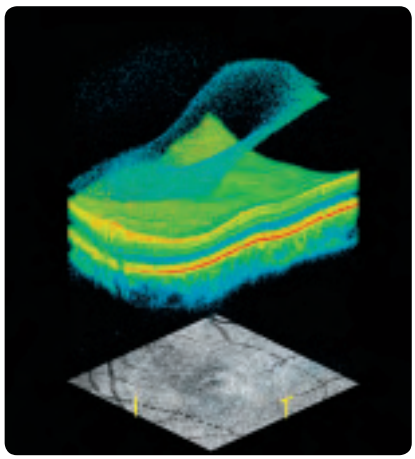
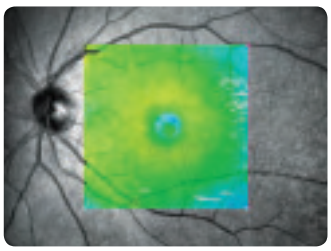
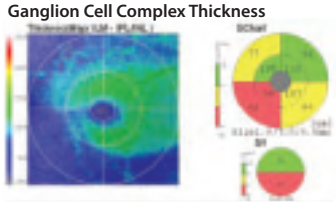
Vitreous Traction 3D Volume



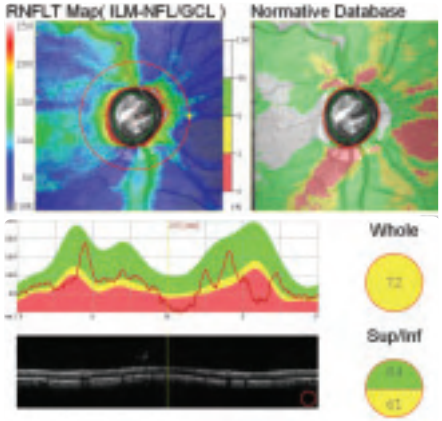
Vitreous Traction Thickness Map



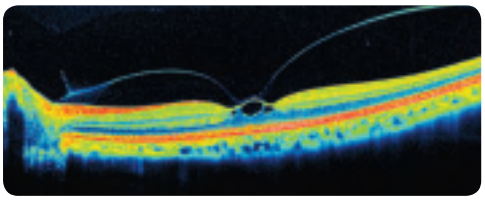
Ganglion Cell Complex Thickness



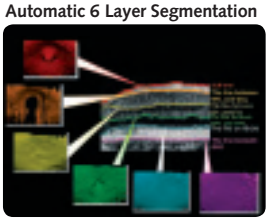
RNFLT Map( ILM-NFL/GCL ) Normative Database



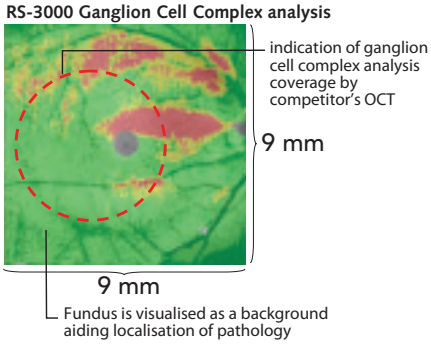

Vitreous Traction B-Scan



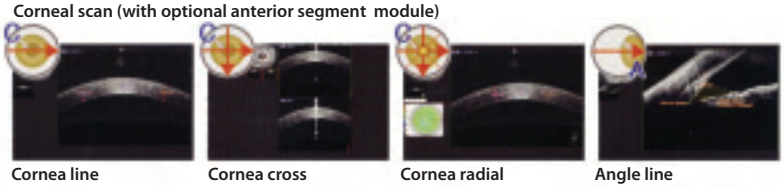
Automatic 6 Layer Segmentation



RS-3000 Ganglion Cell Complex analysis




Corneal scan (with optional anterior segment module)



Cornea line | Cornea cross | Cornea radial | Angle line

- Optional Anterior Segment OCT module with market leading 8mm scan length

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