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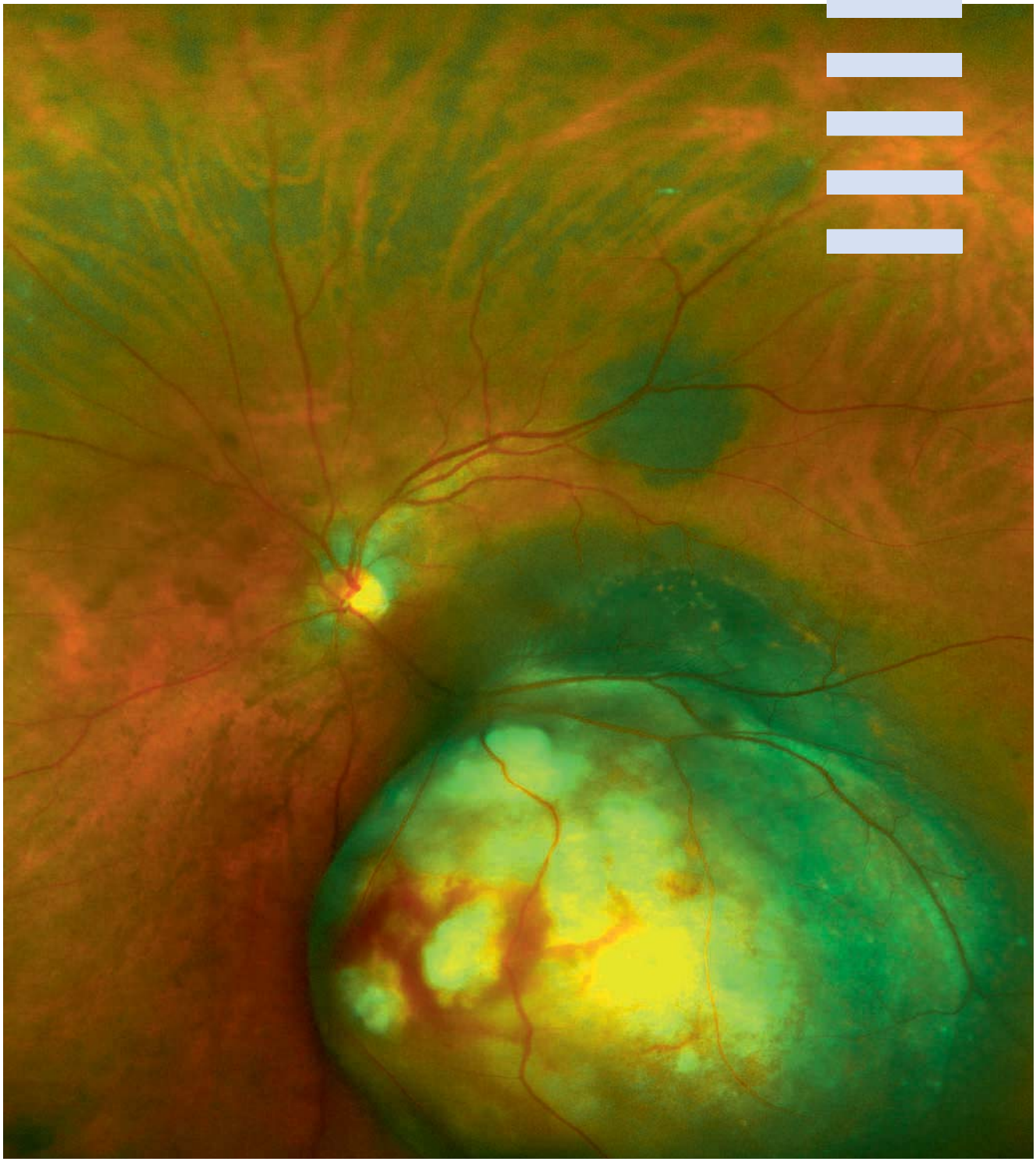

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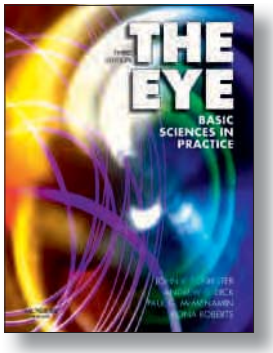

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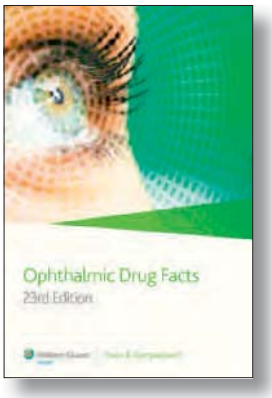

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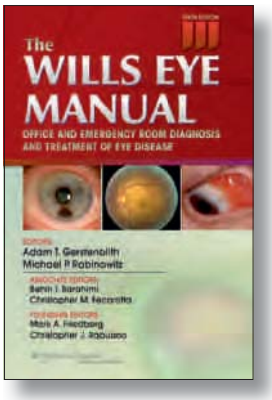

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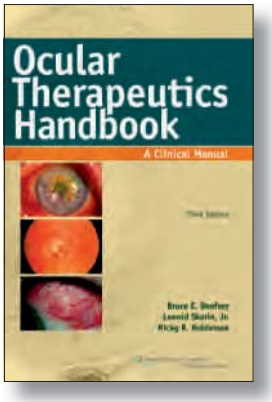

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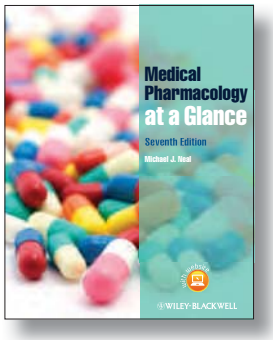

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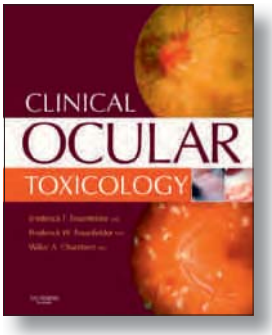

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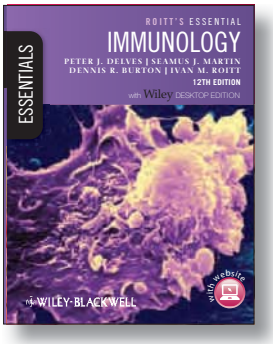

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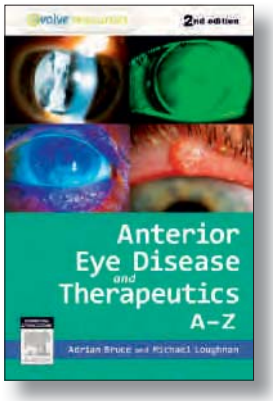

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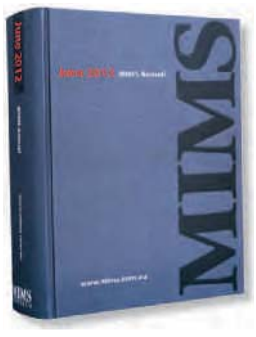

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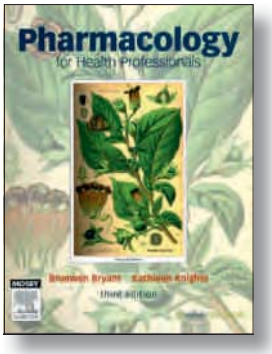

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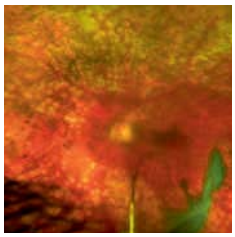
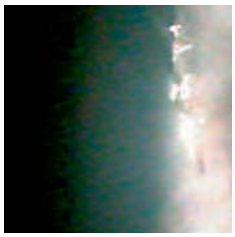
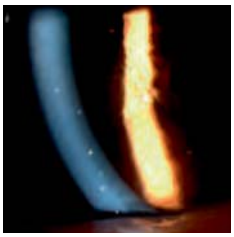
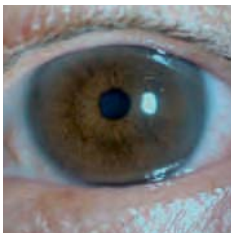


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Perplexing puzzle of

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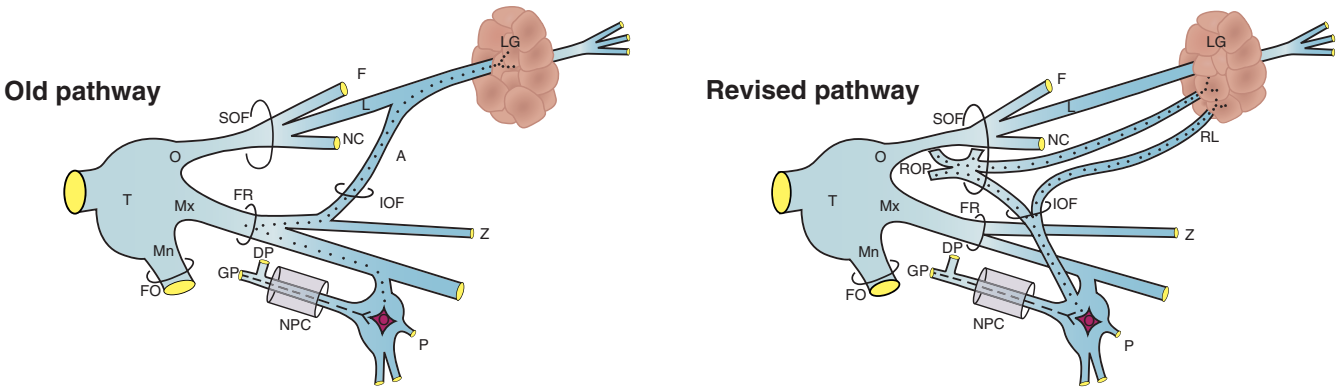
The term dry eye syndrome (DES) is used for a collection of ocular surface conditions that share common signs and symptoms, but may result from a variety of underlying causes. It is a very common condition, especially in older individuals.[1] During the past two decades, a large body of research has expanded our understanding of the underlying pathophysiology of various forms of dry eye.

The report of the National Eye Institute/Industry workshop on clinical trials in dry eyes, published in 1995, increased our awareness of aqueous deficient versus evaporative dry eye, and provided clinicians with strategies for determining the causes of dry eye.[2] The 2007 report of the International Dry Eye Workshop expanded on the 1995 workshop and provided new information and a better understanding of dry eye states.[3]

In 2011, the report of the Meibomian Gland Dysfunction Study provided us with a much better understanding of what may be the most common cause of DES.[4] All of these studies, plus a plethora of other dry eye related research and publications, have vastly improved our ability to identify and treat DES.

Diagnosing and managing DES presenting as a bilateral condition is challenging enough, but when it affects only one eye or is significantly asymmetric, the underlying cause may be a serious neurologic or systemic condition. Ferreting out the cause of asymmetric dry eye necessitates a detailed case history of previous trauma, as well as systemic disease and medications, and requires an understanding of neurologic and hormonal control of tear production.

Stern described the lacrimal functional unit, which is composed of the ocular surface (cornea, conjunctiva, accessory lacrimal glands and meibomian glands), the main lacrimal gland and afferent/efferent innervation that interconnects these structures.[4] Damage to these neural circuits interrupts normal regulation of tear production and causes dry eye disease.[5] Herpes simplex keratitis is an example of a



CN VII parasympathetic innervation of the lacrimal gland
Dashed line: preganglionic pathway. Dotted line: postganglionic pathway.

A: anastomosing branch
DP: deep petrosal nerve
F: frontal nerve
FO: foramen ovale
FR: foramen rotundum
GP: greater petrosal nerve
IOF: inferior orbital fissure
L: lacrimal nerve
LG: lacrimal gland
Mn: mandibular nerve
Mx: maxillary nerve
NPC: nerve of pterygoid canal
NC: nasociliary nerve
O: ophthalmic nerve
P: pterygopalatine ganglion
RL: ramus lacrimale
ROP: retro-orbital plexus
SOF: superior orbital fissure
T: trigeminal ganglion
Z: zygomatic nerve

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asymmetric dry eye

Determining the underlying causes of dry eye requires more than a detailed knowledge of the patient's case history. You also need an understanding of neurologic and hormonal control of tear production.

condition that damages corneal nerves and markedly alters the function of the lacrimal functional unit.[6]

The afferent portion of the 'unit' begins with corneal sensory nerves derived from the ophthalmic branch of the trigeminal nerve (cranial nerve V); corneal sensation is critical in preventing injury through the blink reflex and reflex tearing. It is also crucial to the maintenance of the corneal surface. Loss of sensory innervation (neurotrophic keratitis) leads to punctate keratitis and epithelial loss.[5] Lacrimation is innervated by the facial nerve. Parasympathetic fibres exit the pons and then travel to the pterygopalatine ganglion where they synapse. Post-synaptic fibres travel to the acini of the main and accessory lacrimal glands.[7] Loss of efferent innervation to lacrimal glands dramatically affects the ocular surface.

Toshida and colleagues reported that preganglionic parasympathetic denervation in rabbits resulted in rose bengal staining of the conjunctiva, corneal fluorescein staining, increased blink rate, decreased tear film break-up time, decreased goblet cell density and a 26 per cent reduction in tear flow.[8]

Bell's palsy

Bell's palsy is an idiopathic acute peripheral-nerve palsy affecting the facial nerve (cranial nerve VII), which innervates the muscles of facial expression as well as the lacrimal glands. Bell's palsy presents as unilateral weakness or complete paralysis of all the facial muscles, including paresis of the orbicularis, which results in incomplete closure of the lids with exposure keratitis. Because the facial nerve also supplies parasympathetic fibres to stimulate secretion by the lacrimal and salivary glands, Bell's palsy can produce unilateral dryness. This may be obscured by excessive tearing resulting from lid laxity and subsequent loss of apposition to the puncta to the globe.[9] The neurologic evaluation of facial nerve palsy should include the ability to blink, the presence of Bell's phenomenon and corneal sensation.[10]

The pathophysiology of Bell's palsy is unknown. The herpes viruses including herpes simplex 1, herpes zoster and the Epstein-Barr virus have been targeted as potential triggers, and clinicians have used anti-herpetic medications to treat new cases. Recent studies call this practice into question.

In a large clinical trial, Sullivan and colleagues treated subjects with recent onset (less than 72 hours) Bell's palsy using one of three systemic regimens: prednisolone, acyclovir or a combination of the two. At three months, 83 per cent of the prednisolone group recovered facial function compared with 71.2 per cent in the acyclovir group. After nine months, 94.4 per cent of the prednisolone group had recovered compared with a recovery rate of only 85.4 per cent for the acyclovir group. There was no significant difference in recovery rate between patients with no treatment compared with the acyclovir group.[11]

Because of the ocular manifestations of Bell's palsy, the patient may present to the optometrist's practice before seeing any other health-care provider. It is crucial to triage these individuals immediately, because a Swedish study showed that elderly individuals with Bell's palsy who were treated with steroids within 48 hours of onset had a much better prognosis than those for whom treatment was delayed.[12]

Treatment of the ocular disease focuses on prevention of corneal damage until the condition resolves and includes therapy with artificial tears and bland ointment to prevent drying of the cornea, and in severe cases, use of a moisture chamber. It is essential that the eye is closed during sleep, so patching may be necessary. In severe cases, tarsorrhaphy may be helpful and can be reversed once the condition resolves.[13,14]

Reduced sensation

Diminished corneal sensation, a common cause of unilateral dry eye, can be caused by a wide variety of conditions.[15] Reduced sensation can be confirmed with aesthesiometry. The Cochet-Bonnet Aesthesiometer uses mechanical pressure to evaluate and quantify corneal sensitivity.[16] A nylon monofilament enclosed in a tubular housing can be adjusted from 5 mm to 60 mm in length. Increasing the length of the filament decreases mechanical pressure transmitted to the cornea. In clinical practice we can screen for reduced corneal sensitivity by touching the apex of the cornea with a cotton wisp or non-flavoured dental floss.[16]

Neurotrophic keratopathy

Neurotrophic keratopathy (NK) is a degenerative corneal condition caused by impaired corneal sensation. The basic underlying pathophysiology is denervation of the trigeminal nerve (cranial nerve V) resulting in unilateral dry eye and accompanying deterioration of the ocular surface.[17] Conditions associated with NK include herpes simplex, herpes zoster, leprosy, lattice and granular corneal dystrophies and

Continued page 4

Perplexing puzzle of asymmetric dry eye

From page 3

Stage 1.	Corneal irregularity, dry spots, punctate keratopathy, superficial vascularisation, stromal scarring and epithelial hyperplasia[9,10]
Stage 2.	Epithelial defect, usually located in superior cornea, an area of loose epithelium, stromal oedema
Stage 3.	Corneal ulceration, stromal melting and perforation

Table 1. The Mackie classification system for neurotrophic keratopathy

refractive/penetrating ocular surgery.

Any neurosurgical procedure that impinges on and/or damages the trigeminal nerve has the potential to cause corneal anaesthesia.[18] The Mackie classification of neurotropic keratopathy is used to delineate the severity of the condition (Table 1).

Several studies have demonstrated the vital role innervation plays in maintaining the health of epithelial cells. Araki and colleagues reported alterations in the epithelial surface and adherence between cells after denervation of rabbit corneas. Spontaneous epithelial breakdown was widespread; 83 per cent of the corneas showed persistent epithelial defects.[19] Epithelial breakdown in neurotrophic keratopathy is believed to be secondary to the loss of neurotransmitters such as acetylcholine, catecholamines and substance.[20]

Management and treatment

Management of the monocular dry eye secondary to corneal denervation can be very challenging. Conventional therapies for milder cases include non-preserved artificial tears, bland ointments and gels. For more severe presentations, lateral tarsorophy and amniotic membrane transplantation may be indicated.[18] Lambiase and colleagues reported good results in treating refractory neurotrophic ulcers with nerve growth factor.[17]

Reynolds and Kabat reported the case of a 46-year-old female who suffered from NK secondary to herpes simplex keratitis. Previous treatment with a bandage contact lens, topical antibiotic and artificial tears was unsatisfactory. After four weeks of treatment with of BID Restasis[a] (0.05 per cent cyclosporine ophthalmic emulsion; Allergan, Irvine, California), the patient was able to discontinue the bandage contact lenses and antibiotic.[18]

- Structural lesions in the ear or parotid gland, for example, cholesteatoma, salivary tumours
- Guillain-Barré syndrome
- Lyme disease
- Otitis media
- Ramsay Hunt syndrome (herpes zoster in the facial nerve distribution)
- Sarcoidosis
- Some influenza vaccines

Table 2. Causes of peripheral neuropathy

Unilateral dry eye can result from a broad range of conditions, many of which we are not able to address in this short article. Some are relatively benign, while others have the potential for vision loss. Optometric education encompasses ocular and neurological anatomy and physiology and serves as excellent preparation for appreciating the fundamental processes that maintain the ocular surface and facilitate tear production. Optometrists are well prepared to meet the challenge of the unravelling the cause of unilateral dry eye.

• William D Townsend OD FAAO is a graduate of the University of Houston College of Optometry and practises in a multi-location setting. He served for 11 years as a consultant at the VA Medical Centre in Amarillo, Texas. He is an adjunct professor and preceptor for senior University of Houston College of Optometry externs who rotate through his practice. He conducts research in pharmaceutical agents and contact lens materials and solutions and ocular surface disease. Dr Townsend is a fellow of the American Academy of Optometry and president of the Ocular Surface Society of Optometry.

a. Restasis is commercially unavailable in Australia but topical Cyclosporin eye-drops can be prescribed through a compounding pharmacy.

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The dry eye and systemic disease connection

When managing dry eye, keep in mind all the systemic disorders and medications that have ocular side-effects.

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Gadsden AL, USA

Part of every ophthalmic work-up includes a comprehensive history that incorporates a review of systems and noting all the medications prescribed.

There are many systemic diseases that can lead to dry eye. Although some individuals may have ocular surface disease intrinsically, numerous systemic diseases include an ocular component that manifests as dry eye. Knowing the systemic cause of the ocular surface disease can aid us in the management of dry eye disease.

The most common associations between systemic diseases and dry eye are autoimmune disorders such as Sjögren's syndrome and rheumatoid arthritis.[1] Skin disorders such as rosacea are also likely to have a dry eye component, that is, evaporative dry eye or meibomian gland dysfunction.[2] Finally, some systemic medications can lead to dry eye.

Systemic diseases associated with dry eye

Rheumatoid arthritis is a chronic inflammatory disease that affects about 384,000 Australians. Roughly two per cent of the population have been diagnosed with rheumatoid arthritis by a doctor.[3] More than 90 per cent of people with rheumatoid arthritis have dry eye.[4] Up to 31 per cent of patients with rheumatoid arthritis have a co-existing Sjögren's syndrome with dry eye.[4] Sjögren's syndrome is one of the most prevalent autoimmune disorders affecting as many as 0.5 per cent of Australians.[5]

Sjögren's syndrome has its own complexities: its pathogenesis is obscure, it presents with both dry eyes and dry mouth, and it can present as primary or secondary Sjögren's syndrome. Dry eye syndrome is the most common ocular feature of systemic lupus erythematosus and is often associated with secondary Sjögren's syndrome.[6] Thyroid eye disease is a common systemic disease associated with dry eye due to thyroid hormone imbalance and exophthalmos-related corneal exposure.[7]

One of the most common ocular manifestations of diabetes is dry eye disease.[8] More than half of patients who have diabetes experience dry eye symptoms (54.3 per cent in one study), such as burning and foreign body sensation, and suffer from ocular dryness.[9] Reflex tearing has been demonstrated to be significantly decreased in insulin-dependent diabetic patients.[10]

Systemic medications

Numerous systemic medications can have ocular side-effects producing dry eye, including anticholinergic drugs, that is: antidepressant, antipsychotic, anti-Parkinson's disease and antihistamine drugs. Any systemic medication that dries mucosal surfaces or slows the activity of mucosal surfaces may produce dry eye.

If a medication causes dry mouth, it will also cause dry eye. Some of the leading causes of dry eye disease from systemic medications include antihypertensives, beta-blockers,[11] cholesterol-lowering medications,[12] anticoagulants/aspirin therapy (which may also aggravate the condition, as they are secreted in the tear film), genitourinary medications such as hormone therapy (both oestrogen and androgen),[13] psychogenic medications[14] and dermatology treatments (such as isotretinoin).[15]

Almost 75 per cent of patients taking glaucoma medications have some signs and symptoms of dry eye. This is a direct result of the preservatives in the medications, the side-effects of the active ingredients, and the overall age group typically affected by glaucoma.[16] Instillation of any eye medications, especially when they are instilled frequently (that is, more than four drops a day), may prevent the normal maintenance of the tear film and cause dry eye symptoms.

Dry eye and ocular surface disease are major concerns in today's optometric practice. It is incumbent on every optometrist to research the role of systemic diseases and systemic medications in their patients with dry eye disease.

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Optic disc oedema:

Case report

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The University of Melbourne EyeCare Neuro-ophthalmology referral clinic
Department of Optometry and Vision Sciences
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History

A 22-year-old woman was seen at the University of Melbourne EyeCare Neuro-ophthalmology clinic, complaining of transient visual loss in both eyes that lasted for 10-20 seconds. She reported severe headaches daily with occasional double vision when directing her eyes at extreme right and left gazes. The patient mentioned that whenever she bent down, she could hear her 'pulse'. All these symptoms began three weeks prior to her visit to the clinic. The patient was not on any medications and was in good general health. She reported weight gain of 15 kg over the previous year. A family member reported that the patient was undergoing high levels of personal stress.

Examination findings

Uncorrected visual acuities were R and L 6/4.8 with +1.00 DS R and L. Cover test showed 8 PD esophoria at distance (similar in R and L gaze) and orthophoria at near. Ocular motility revealed bilateral abduction deficit in extreme gaze consistent with bilateral sixth nerve palsy. Colour vision was normal (Ishihara plate) in both eyes. Pupil reactions were equal with no relative afferent pupillary defect. Blood pressure was 114/77 mmHg. The patient's height was 168 cm and weight was 105 kg.

Dilated fundus examination (Figures 1A and 1B) showed moderate bilateral disc oedema (L > R) in both eyes with clean fundi. Parts of the vessels overlying the discs were obscured. The veins were dilated (AV 1:3) and tortuous. Both maculae were normal.

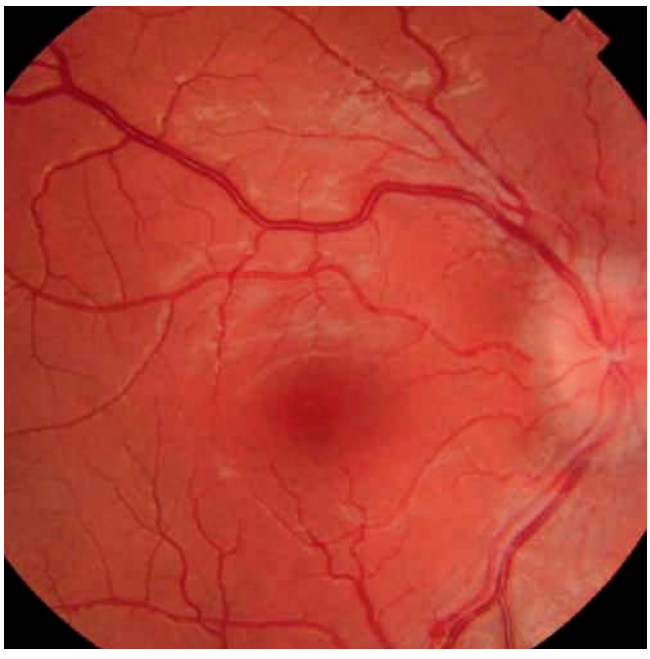


Figure 1A. Fundus of the patient: right

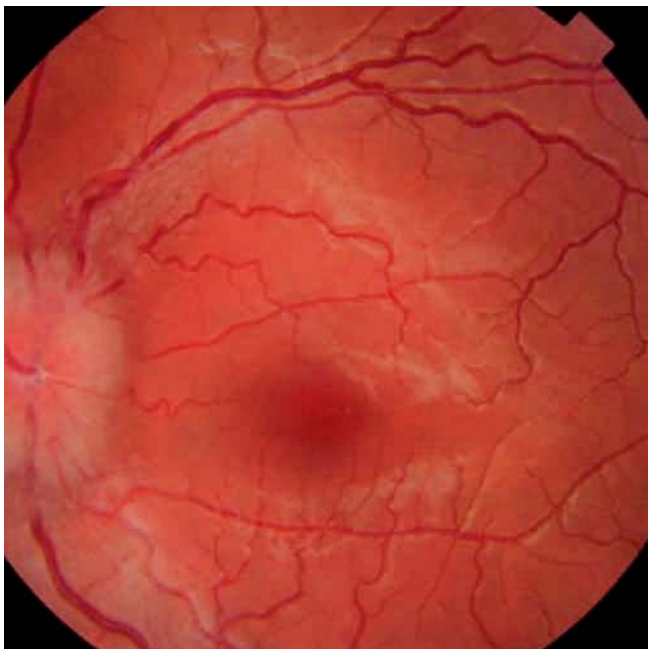


Figure 1B. Fundus of the patient: left

a diagnosis of exclusion

It is critical to eliminate malignant hypertension and intracranial mass/abnormality.

Differential diagnoses of disc oedema

Bilateral pseudopapilloedema: the most common causes are disc drusen[1] and high hypermetropia.

- Refractive state rejects high hypermetropia (+1.00 DS) and drusen are not visible. The drusen might be buried but the venous congestion (AV = 1:3) suggests that is not likely and the concurrent presence of neurologic and systemic symptoms mandate investigation for other causes of disc oedema.

Malignant hypertension with papilloedema[2]

- The normal blood pressure (114/77 mmHg) and no evidence of other signs of hypertensive retinopathy rejects this possibility.

Optic neuritis with disc swelling[3]

- Vision loss typically over hours to days and visual acuity often worse than in this patient. Presents with orbital pain on eye movement, acquired loss of colour vision, relative afferent pupillary defect and visual field defect.
- Presence of neurologic and systemic symptoms mandate investigation for other causes.

Orbital nerve tumour or intracranial tumour/abnormality[4]

- Orbital. Typically unilateral disc swelling or very asymmetric if bilateral with mass that crosses chiasm. Proptosis and restriction of ocular motility often present. It is highly unlikely but a CT/MRI scan is needed to rule this out.
- Intracranial. The papilloedema, systemic and neurologic symptoms are consistent with an intracranial mass/lesion, or with cerebral venous sinus thrombosis. This diagnosis is not rejected at present and there is a need for scans to do so.

Idiopathic intracranial hypertension[5]

- The patient's clinical signs and symptoms fit this diagnosis. Further work-up is required to dismiss the possibility of intracranial mass.

Further investigations and results

Visual field results

A central (22 degree) neurological visual field test (Figures 2A and 2B) was performed on both eyes. Both visual fields showed an enlarged blind spot typical for papilloedema.

Imaging/cerebrospinal fluid (CSF) analysis

- CT: no abnormalities detected
- MRI/MRV: no abnormalities detected
- Lumbar puncture: normal CSF constituents with mildly increased opening pressure (27 mmHg). Note the lumbar puncture was performed at a time when the patient's symptoms had spontaneously improved considerably.

Conclusion

The diagnosis of idiopathic intracranial hypertension is one of exclusion, in this case of malignant hypertension and intracranial mass/abnormality. A weight loss strategy (low calorie diet and exercise) is to be aggressively pursued. The patient is reviewed in four weeks. The provisional prognosis is good as the patient still retains normal visual acuities, pupils, colour vision and no presence of significant visual field defects.

1. Lam BL, Morais CG Jr, Pasol J. Drusen of the optic disc. *Curr Neurol Neurosci Rep* 2008; 8: 5: 404-408.
2. Tajunisah I, Patel DK. Malignant Hypertension with Papilledema. *J Emerg Med* 2011 Aug 3. [Epub ahead of print]
3. Protti, A, Spreafico, C, Frigerio, R et al. Optic neuritis: diagnostic criteria application in clinical practice. *Neurol Sci* 2004; s296-s297.
4. Eggers H, Jakobiec FA, Jones IS. Tumors of the optic nerve. *Doc Ophthalmol* 1976; 41: 1: 43-128.
5. Friedman DI, Jacobson DM. Idiopathic intracranial hypertension. *J Neuroophthalmol* 2004; 24: 2: 138-45.

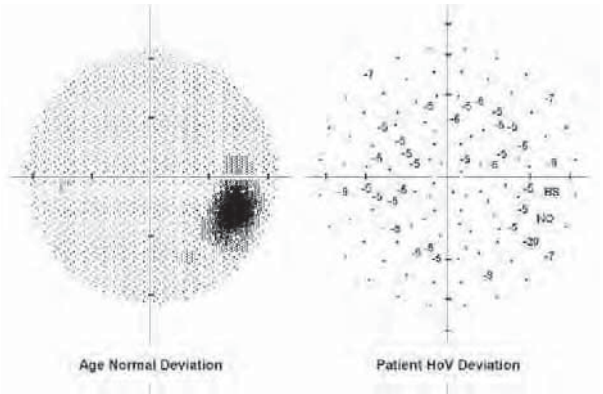


Figure 2A. Visual field results: right

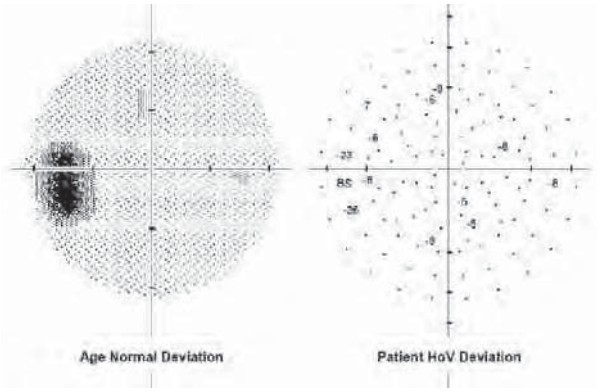


Figure 2B. Visual field results: left

Analysis of retinal pathologies in the context of the entire retina

New imaging technology enables you to document retinal findings with a wide field of view without compromising detail.

Mary Travis
MOptom
Vision Eye Institute
Melbourne VIC

When Vision Eye Institute in the Melbourne suburb of Coburg acquired the new ultra-widefield (UWF) imaging technology from Optos, both the optometry and ophthalmology segments of this practice recognised the clinical benefits of this new device. In this article, I share my impression as a clinical optometrist of ultra-widefield imaging, using two medical retina case studies to highlight the impressive features of the Optomap system.

The ultra-widefield Optomap images of the retina can provide a new perspective on the diagnosis, management and documentation of retinal conditions. No imaging system can replace a thorough history and appropriate clinical examination of an optometry patient; the stereopsis and clarity of images provided by fundus lenses cannot be replaced. However, the ability for optometrists to use the ultra-widefield images to document retinal findings with such a wide field of view without compromising detail, and the ability to share these images with patients, GPs and ophthalmologists, is unique and a great advance in ophthalmic imaging technology.

The scanning laser ophthalmoscope uses two wavelengths of light to produce high resolution colour images through an undilated pupil, even in the presence of a cataract that would sacrifice image quality in many other systems.

The high resolution images taken with this technology can supplement the regular posterior eye clinical examination that is usually done through dilated pupils.

As the name suggests, one of the chief advantages of the system is its ability to capture an ultra-widefield image extending up to 200 degrees without compromising resolution. This allows for easy integration of posterior pole and mid-peripheral findings to aid in the overall assessment of retinal conditions.

Case report 1

Diabetic retinopathy with CSME

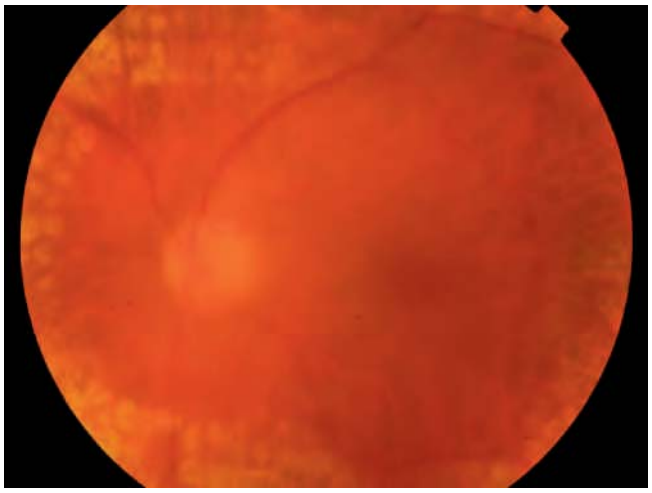


Figure 1. Colour fundus photography of the left eye of a 72-year-old female with diabetes, illustrating dense cataract prior to cataract surgery

This patient is a 72-year-old female with a seven-year history of non-insulin-dependent diabetes mellitus, who is currently not using insulin. The presenting HbA1c was extremely elevated at 14.6 but blood pressure (132/66) and blood lipid profile were well controlled.

At initial presentation in September 2011, the right eye had severe clinically-significant macula oedema (CSME) and severe non-proliferative retinopathy, while the left eye displayed proliferative diabetic retinopathy. During follow-up, steps have been taken by the patient, treating general practitioner and endocrinologist to improve diabetic control.

Both eyes underwent extensive pan-retinal photocoagulation during follow-up, and bilateral intravitreal injections of both bevacizumab (Avastin) (two in each eye) and triamcinolone (one in each eye). As expected, there was progression of pre-existing cataract, which not only reduced visual function (VA R 6/36 and L 6/24) but also made further retinal photocoagulation impossible to perform as the retina could not be visualised clearly.

Figure 1 shows a colour fundus image of the left eye with our previous retinal camera, illustrating the dense nuclear sclerotic and anterior cortical spoke cataract precluding a meaningful view of the retina.

Figure 2 is the Optomap image of the same eye, illustrating the fine retinal details available with this image to complement the wide-angle views. The wedge-shaped shadows in the right-hand side of the image are the spokes of the stereotypical cortical cataract. The white opacity in the vitreous in the inferior fundus is remaining triamcinolone from a previous intravitreal injection.

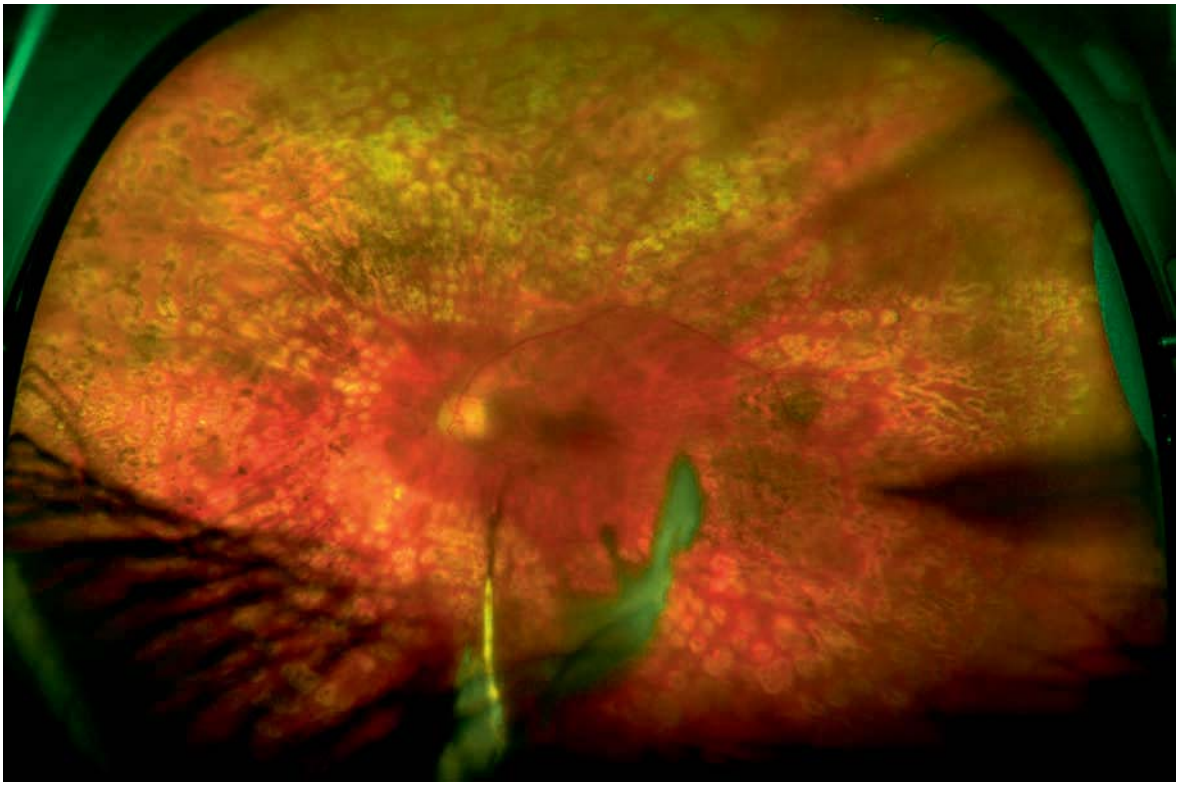


Figure 2. Optomap UWF colour fundus image of same eye in Figure 1 prior to cataract surgery, showing extensive panretinal photocoagulation and remnant triamcinolone in the vitreous. Artefactual images of eyelashes are sometimes evident in the inferior section of the image.

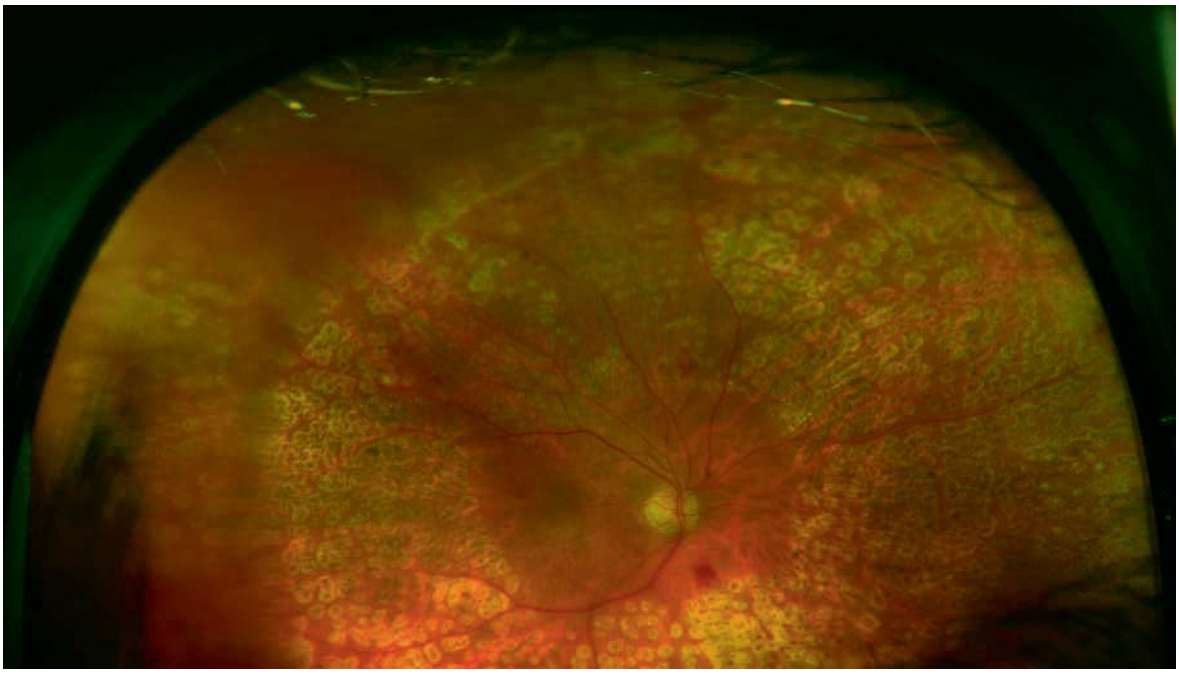


Figure3. Cropped Optomap UWF colour fundus image of the right eye of the same 72-year-old female following cataract surgery

Images: orthoptists Lucinda Tomaino and Marian Saeed

Figure 3 is a cropped Optomap colour fundus image of the right eye of the same patient, which has undergone cataract surgery and maintained best corrected VA of 6/9. The slight blur in the left-hand side of the image is due to the view of the retina being obtained through the lens capsule outside of the capsulorexis.

This case illustrates the level of retinal detail provided by the UWF imaging device even in the presence of significant cataract, a degree of detail that is not sacrificed in the imaging of the mid- and far-peripheral retina. There is a significant saving of valuable patient contact time because the mid- and far-peripheral retina can be recorded in a single image.

Continued page 10

Case report 2

AMD with CRNVM

This patient is a 68-year-old male who initially presented in February 2009 with a superior temporal branch retinal vein occlusion in the right eye accompanied by cystoid macula oedema (CME). The cause of this event is unattributed, as cardiovascular parameters (blood pressure, lipid profile and cardiac function) have always been normal and/or well controlled.

The right eye underwent sector pan retinal photocoagulation (PRP) and intravitreal injections of bevacizumab (Avastin) to treat the CME, maintaining a best corrected VA of R 6/12- at his last review. The right eye had a subsequent combined vitrectomy with peeling of the internal limiting membrane to remove an epiretinal membrane, and cataract surgery in December 2010.

In May 2012, the patient developed further vision loss in the right eye, from post-operative 6/12 to 6/24. Fundus fluorescein angiogram confirmed a chorioretinal neovascular membrane (CRNVM) associated with AMD in the right eye, requiring to date two injections of ranibizumab (Lucentis). Figure 4 is a traditional fundus colour image of the right eye focusing on the posterior pole, illustrating the macula haemorrhages associated with recent onset AMD.

Figure 5 is an UWF colour fundus image of the same eye taken one week after Figure 4, which accounts for the slight difference in macular haemorrhages. The sclerotic vessels in the superior temporal retina extend to the optic nerve, and the sector PRP in the mid-peripheral retina is clearly visible. This image has been cropped to reduce field of view to enhance detail in print, but the field of view is still much broader than the traditional fundus photography. In addition, the Optos software magnifier tool has been used to produce an image of resolution and detail comparable to that of traditional fundus photography.

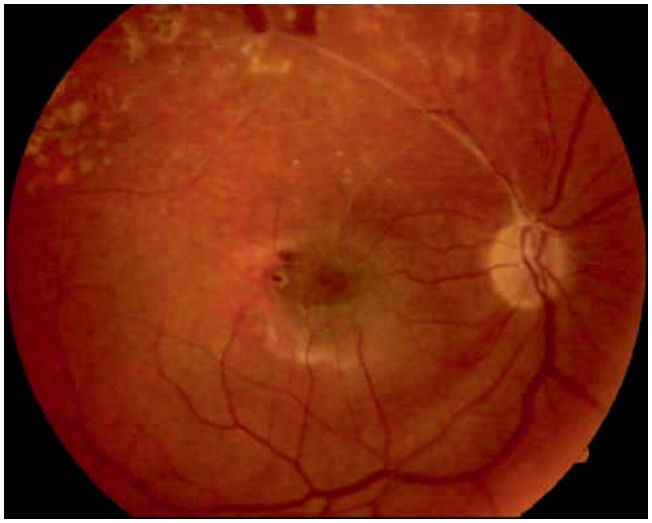


Figure 4. Colour fundus photography of a 68-year-old male with sector PRP due to a superior-temporal branch retinal vein occlusion with new macular haemorrhages due to recent onset CRNVM associated with AMD

This case shows that ultra-widefield imaging not only provides an ultra-wide view of the retina for comprehensive documentation purposes, but also preserves image quality at the posterior pole. This allows an integrated analysis of retinal pathology, whether central or peripheral, in the context of the status of the entire retina.

Summary

As an optometrist practising within an ophthalmology practice, this system is invaluable in documentation of current findings. The ultra-widefield images enhance our ability to gain a global overview of the retina without compromising resolution, and facilitate easy and insightful planning of future treatment strategy. This system would be an excellent tool for an optometrist wanting to further their knowledge and understanding of posterior segment conditions, those affecting both the posterior pole and periphery. Because of the clarity of the images, the ability to resolve fine detail using the magnifier function and the enhanced field of view to 200 degrees, the system would be an invaluable asset to a busy, broad-scope optometric practice. ■

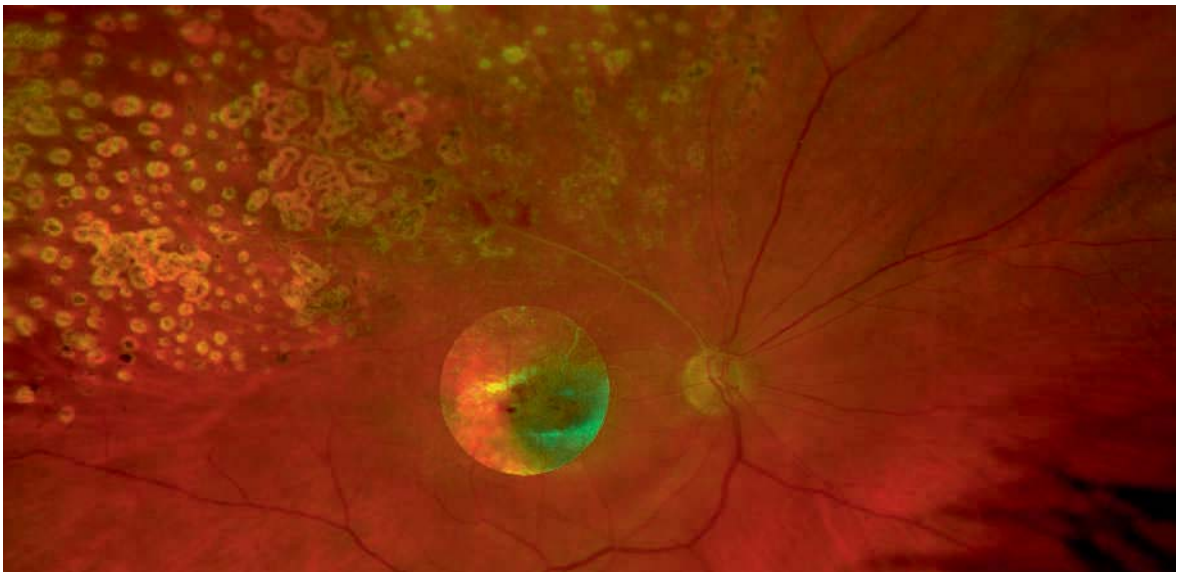


Figure 5. Cropped Optomap UWF colour fundus image of the same eye as Figure 4, illustrating the improved resolution available with the Optos software magnifier tool. The area covering the central macula has been magnified.

Clinical QUIZ

How would you treat and rehabilitate an angled flap laceration and mild corneal oedema?

Traumatic injury treatment

Dr Robert Holloway
BScOptom PGCertOcTher
Holloway Vision Centre
Wangaratta VIC

JD, a nine-year-old boy, presented to the local emergency department following an injury to his right eye. He had been struck in the eye by a length of stick that had broken on impact with the ground and ricocheted upward. He was advised to consult our office the next day. He was in moderate pain, photophobic with a red, watery eye.

His clinical presentation was:

- best acuity R 6/9 L 6/6
- angled flap laceration running vertically through the temporal cornea
- Seidel's sign negative
- corneal thickness R 650 µm L 605 µm, indicating some corneal oedema.

(Figures 1-3)

How would you manage this patient?

ANSWER PAGE 16

Figure 1. Slitlamp image at presentation

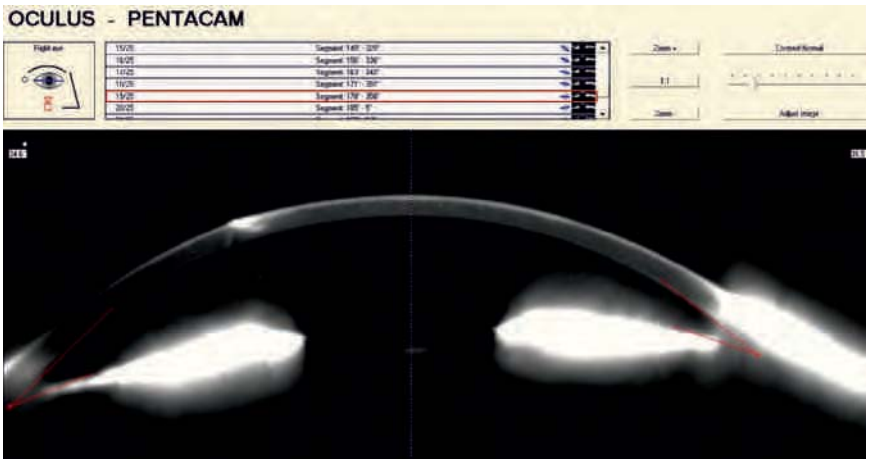


Figure 2. Pentacam image at presentation

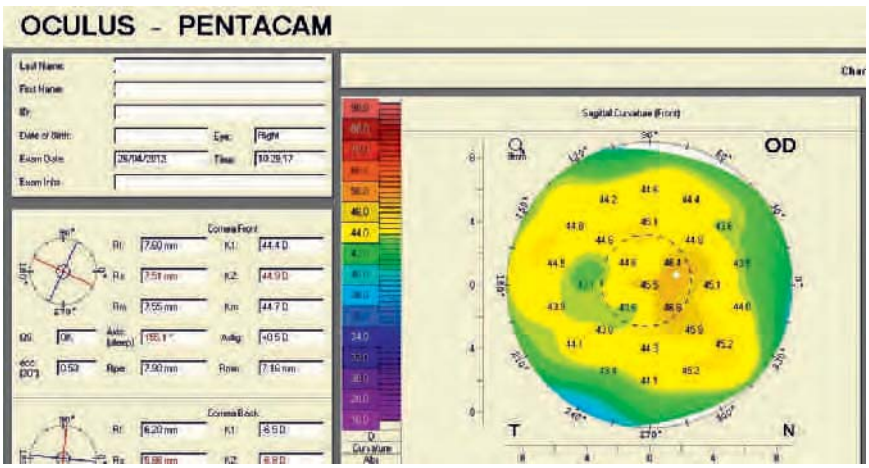


Figure 3. Topography at presentation

Combined refractive and when planning

Dr Noel Alpins
FRANZCO FRCOphth FACS
George Stamatelatos
BScOptom
NewVision Clinics, Melbourne

This method of vector planning draws information from refraction and topography to minimise post-operative astigmatism.

Obtaining the best possible visual outcome without spectacles is the goal of both surgeon and patient after laser vision correction. With the introduction of topographers that can measure the front and back of the cornea, as well as corneal aberrometry, Scheimpflug imaging and whole-of-eye wavefront aberrometry, we have come a long way toward consistently achieving excellent visual outcomes. One final area left to conquer is the optimal treatment of astigmatism, where the two modes of measuring astigmatic treatment parameters (refractive and corneal) often differ significantly from each other.

The laser vision correction procedure involves changing the shape of the cornea to apply the refractive shape of the spectacles to it. This is straightforward for a purely spherical refraction with an aspheric adjustment but can become complex when dealing with myopic, hyperopic and mixed astigmatism corrections.

In most cases, the magnitude and direction of the astigmatism measured in the spectacles differs from that measured on the cornea. When quantified vectorially, this difference is known as the ocular residual astigmatism (ORA). It is calculated as the vectorial difference between the astigmatism on the cornea and that in the manifest refraction (corneal plane) and is expressed in dioptres (D) and degrees (Figures 1A, 1B, 1C).[1,2]

The ORA also quantifies the amount of astigmatism that cannot be corrected, regardless of how good the outcomes of the laser vision correction procedure are. Software programs are available that calculate the ORA (Figure 2) from simulated keratometry parameters exported directly from topography and refractive cylinder at the corneal plane. The values for ORA can be conveniently used directly from the analytical outputs generated within most topography devices today.

It is of utmost importance that the surgeon considers this corneal refractive difference pre-operatively when advising patients of laser vision correction suitability and when planning the treatment to achieve the best possible visual outcome.

If the ORA is high in magnitude (>1.00 D), then the patient may be advised against having the procedure, because a high amount of astigmatism will remain post-operatively. This is particularly the case if the amount of spherical correction is relatively small. The surgeon must consider how to deal with this high ORA.

Treatment that uses refractive parameters alone will leave all the ORA on the cornea post-operatively. In about seven per cent of cases, this amount can be worse than the pre-operative corneal astigmatism,[1] potentially leading to increased aberrations and associated visual symptoms, particularly under mesopic conditions. Treatment using the corneal parameters alone would leave

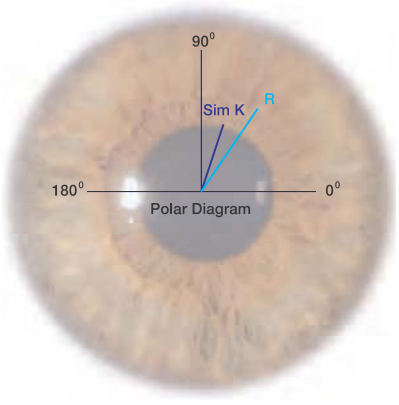
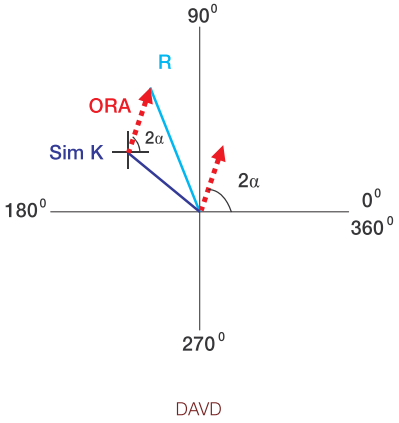


Figure 1A. Polar diagram of refractive cylinder (corneal plane) at positive axis and simulated keratometry (Sim K)



DAVD

Figure 1B. Double angle vector diagram showing a 'doubling' of the positive cylinder axes without a change in the astigmatic magnitudes. The ORA is the vectorial difference between Sim K and the refractive cylinder (corneal plane).

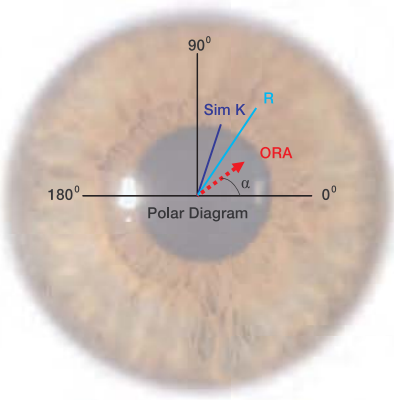


Figure 1C. Polar diagram displaying the ORA as it would appear on the eye

corneal parameters laser vision correction

all the ORA in the spectacle correction (manifest refraction). However, combining both the corneal and refractive astigmatism in the treatment plan deals with leaving the minimum amount of overall astigmatism post-operatively.

This approach is known as vector planning.[3,4,a] It considers both corneal and refractive modes to reduce corneal aberrations and optimise astigmatic outcomes.

Vector planning: keratoconus and wavefront-guided treatments

Two studies applying vector planning have shown the benefits of combining corneal and refractive parameters in the laser vision correction treatment plan.[5,6] The first was a retrospective study of 45 eyes with forme fruste and mild keratoconus patients who had surface ablation laser surgery.[5] All photo astigmatic refractive keratectomy (PARK) treatments were optimised to leave minimum remaining corneal astigmatism favouring a bias to a with-the-rule orientation. Post-operative results at 12 months showed that on average for every eye in the group, the corneal cylinder was reduced by an additional 0.58 D, compared to results that would have been attained by treating refractive values alone when predicted targeted outcomes were calculated pre-operatively.

This benefit of reduced corneal astigmatism was achievable without compromising the refractive outcome. In fact, the refractive outcome was enhanced with a better than predicted refractive astigmatism reduction. No problems or adverse signs were detected, such as increase in corneal irregularity and curvature or progression of ectasia resulting in a reduction of uncorrected or best-corrected visual acuity.

In the second study, 21 eyes of 14 patients were distributed into two groups in a prospective double masked study.[6] One group was treated relying on wavefront parameters alone, the other by using wavefront parameters combined with topography values (WF&VP), following the vector planning approach.

A comparison between the two groups showed a trend to greater correction of corneal astigmatism in the WF&VP combined group with better visual outcomes in low contrast mesopic conditions, greater reduction in horizontal coma and greater potential for improvement in BCVA.

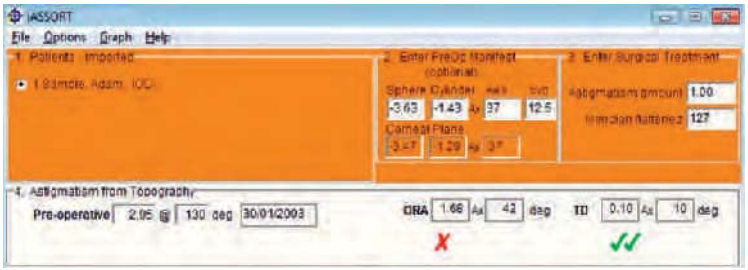


Figure 2. The iASSORT software program for corneal astigmatism analysis. Simulated keratometry parameters are exported from the topography system (in this example: 2.95 D @ 130) and are compared to the refractive astigmatism (-1.29 D x 37 at the corneal plane) to calculate the ocular residual astigmatism (ORA) which in this case is 1.68 D Ax 42. This is high in magnitude as displayed by the red cross which indicates that the ORA magnitude lies outside the normal range of 0.00 D to 1.00 D.

Vector planning: a case study

The ASSORT program (Alpins Statistical System for Ophthalmic Refractive surgery Techniques) developed at NewVision Clinics in Melbourne uses vector planning and analysis in a paradigm that maximally reduces overall astigmatism. It includes the option to favour a bias to with-the-rule astigmatism.

Using the example referred to in Figure 2, the ASSORT laser treatment module in Figure 3 displays the second order spherocylindrical wavefront refraction as measured using aberrometry to be -3.63 DS/-1.43 DC x 37 at vertex distance of 12.5 mm.

The topographic data of the same astigmatic eye (Figure 2) displays a simulated keratometry value of 2.95 D of astigmatism at the steepest meridian of 130 degrees. The amount of uncorrectable astigmatism in this patient's eye is 1.68 D Ax 42 (ORA). The distribution between corneal and refractive astigmatism targets is reflected in the 'Emphasis' bar of the ASSORT user interface (Figure 3) where 100 per cent indicates a goal of completely eliminating refractive astigmatism and 0 per cent shows the emphasis on completely reducing topographic astigmatism through treatment.

If we treat conventionally, that is with 100 per cent reduction of refractive astigmatism as shown in Figure 3, all of this (ocular) residual astigmatism will remain on the cornea to neutralise the internal ocular aberrations quantified by the ORA. This decision results in a 'Target' of 1.68 D at a meridian of 132 degrees. The meridian is 90 degrees away from the ORA axis; this is because the corneal astigmatism fully neutralises the internal (non-corneal) error, resulting in targeting zero astigmatism in the post-operative refraction (shown as the light blue 'Target').

The target induced astigmatism vector (TIA) is the astigmatic treatment[7]– that is the amount of astigmatism that we attempt to correct, which in this example, using conventional treatment based on 100 per cent refraction, is 1.29 D Ax 37.

At the other extreme in the planning process spectrum, if we treat this eye emphasising the complete reduction of topographic astigmatism values alone and sphericise the cornea (Figure 4) 1.68 D of the unavoidable ORA will theoretically remain in the post-operative refraction in conjunction with this spherical cornea to neutralise the internal aberrations.

However, by taking an optimised view of

Continued page 14

a. Web site: http://en.wikipedia.org/wiki/Alpins_method_of_astigmatism_analysis

Refractive and corneal parameters

From page 13

the situation and incorporating a proportion of each of the corneal and refractive astigmatism parameters measured into the overall treatment, we shift the 'emphasis' for astigmatism reduction 'to the left', thereby increasing the preference for complete corneal astigmatism correction. This results in the maximum ablation of treatment being more closely aligned to the principal corneal meridian. There is less 'off axis' effect to the corneal astigmatism, with more astigmatism reduction and less torque and meridian shift.[8]

Figure 5 shows the optimal treatment as determined from the surgical emphasis graph (Figure 6) which displays a linear relationship between the corneal versus refractive astigmatism emphasis and the orientation of the target astigmatism.[1] Figure 6 is based on the notion that with-the-rule astigmatism is favourable and against-the-rule astigmatism is relatively unfavourable. More astigmatism is reduced when its orientation is not favourable—which may also include oblique astigmatism. The placement of the emphasis percentage is a decision that is determined by the surgeon.

In this example, the meridian of the target topography is 132 degrees. As it lies 42 degrees from a favourable with-the-rule orientation of 90 degrees, 42/90 (47 per cent) can preferentially be apportioned to a topography-based goal of zero astigmatism. By the same process, the targeted refractive astigmatism power axis of 42 degrees is 48 degrees away from 90 degrees and therefore places 53 per cent emphasis on the correction of refractive astigmatism. The TIA here is greater than before at 2.07 D Ax 39. There are also other paradigms that may be applied to determine the apportionment of the ORA neutralisation depending on patient parameters—these include using the minimum TIA magnitude, the proportion of corneal to refractive astigmatism pre-operatively should remain the same post-operatively and splitting the ORA magnitude in half—50 per cent emphasis to corneal astigmatism and 50 per cent to refractive astigmatism.

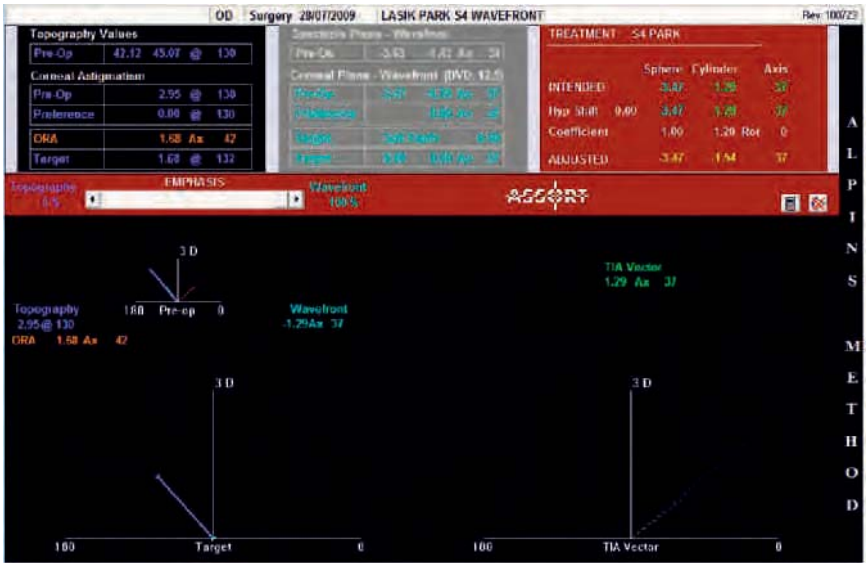


Figure 3. The ASSORT treatment screen displays both the wavefront refraction and the corneal astigmatism by topography. The emphasis shown here is that of conventional treatment with 0 per cent topography/100 per cent wavefront refraction, leaving all the ORA on the cornea as displayed by the 'Target = 1.68 Ax 132'.

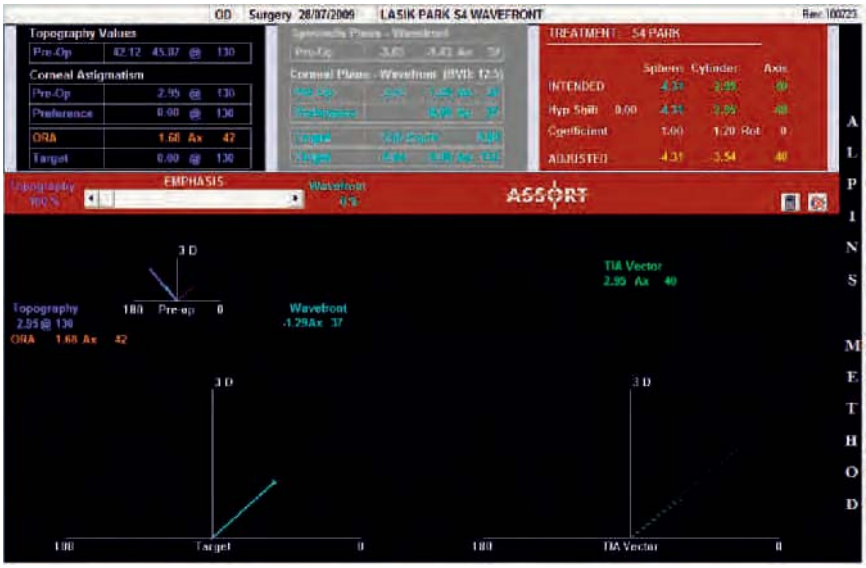


Figure 4. The emphasis shown here relates to topography-guided treatment where all the ORA (1.68 D) remains in the manifest refraction (100 per cent topography/ 0 per cent wavefront refraction), shown as the 'Target = 0.84/-1.68 x 132'

The overriding challenge is how to approach corneal sphericity when the orientation of the target corneal astigmatism (post-operatively) becomes increasingly unfavourable. It is a surgeon's decision which is guided by the orientation that is more favourable than others. For most astigmatism treatment, the emphasis of 60 per cent refraction (rather than 100 per cent conventional) is a safe choice that gains a reduction in remaining corneal astigmatism of 40 per cent without compromising the refractive outcome. The spherical equivalent targeted is zero where any refractive cylinder remains so that a minimum, if any, refractive error remaining is not evident on manifest refraction resulting in spectacle-free vision.

The patient's ORA is still 1.68 D but is now apportioned between the refraction and the corneal parameters. The corneal astigmatism targeted is 53 per cent of 1.68 D (0.89 D); the left-over 47 per cent of the astigmatism (-0.79 DC) is placed refractively as a spherical equivalent of zero (+0.39 DS/-0.79 DC Ax 132). This remaining refractive astigmatism with a spherical equivalent of zero may not be fully perceptually evident to the patient, as shown in a previous study.[5] The goal of spectacle freedom is

achieved and the patient enjoys less corneal astigmatism and clear spectacle-free vision as a result.

It is important to highlight that regardless of the percentage chosen on the 'emphasis' bar, the maximal treatment enables the surgeon to post-operatively target the minimum amount of astigmatism overall, equal to the ORA. If the combined magnitude of the remaining astigmatism (corneal plus refractive) is greater than the initial ORA, the surgery then fails to achieve the maximum astigmatism treatment; the patient is not on the maximum treatment 'emphasis' line and so is either under- or over-treated, leaving an excess amount of astigmatism remaining in the optical system of the eye and its correction.[1]

As technology advances with future possibilities, every point that is measurable on the cornea by topography together with wavefront aberrometry could be 'vector planned', resulting in an ultimate ablation profile and the minimum possible astigmatism remaining for each individual situation.

Summary

Treatments based completely on either topographic or refractive data alone in astigmatic patients with a high ORA leave excess remaining astigmatism overall post-operatively, compared with treatment using vector planning.

Wavefront-guided laser refractive surgery has certainly been of benefit in correcting the internal higher order aberrations (HOAs) of the eye on the cornea. However, the correction of HOAs in cases where there is a high ORA needs to be more fully explored to increase overall patient satisfaction.

Topographically-guided lasers provide comprehensive mapping in situations such as highly irregular corneas where manifest and wavefront refractions may be inadequate to provide a smoother corneal surface.

Using the Alpins method of vector planning to combine information from the refraction (manifest or wavefront) and the topography, we can help minimise astigmatism remaining on the cornea and consequently in the optical system of the eye, and optimise visual outcomes, particularly in low light and reduced contrast environments.

Applying a shift in emphasis toward a greater reduction in remaining corneal astigmatism produces a maximal reduction of astigmatism together with the benefits of reduced corneal aberrations associated with reduced corneal astigmatism.

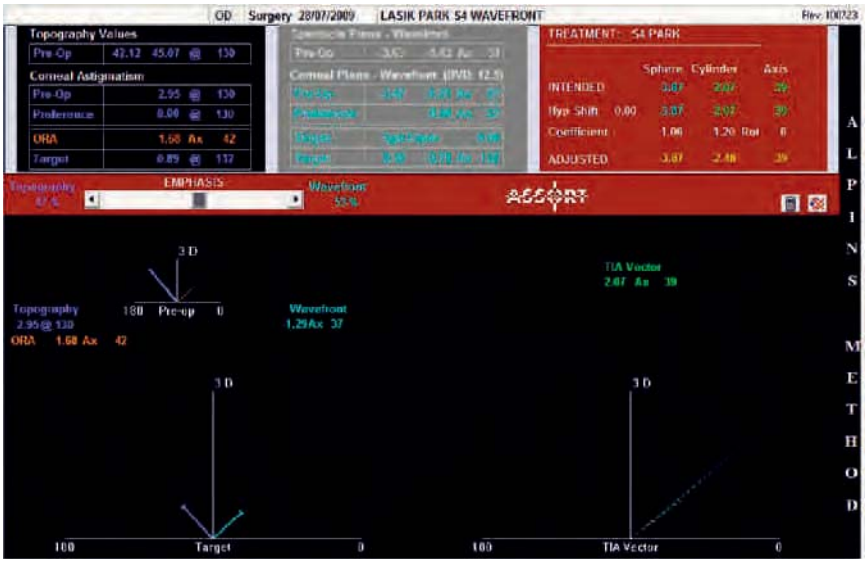


Figure 5. ASSORT Treatment Planning. This figure shows how the ORA of 1.68 D Ax 42 is apportioned 47 per cent of the ORA to eliminate the topography astigmatism and 53 per cent is apportioned to the refractive cylinder. The ORA is neutralised by an equivalent 0.89 D at the cornea and -0.79 DC at the spectacle refraction, but it is at an orientation 90 degrees away from 132 degrees.

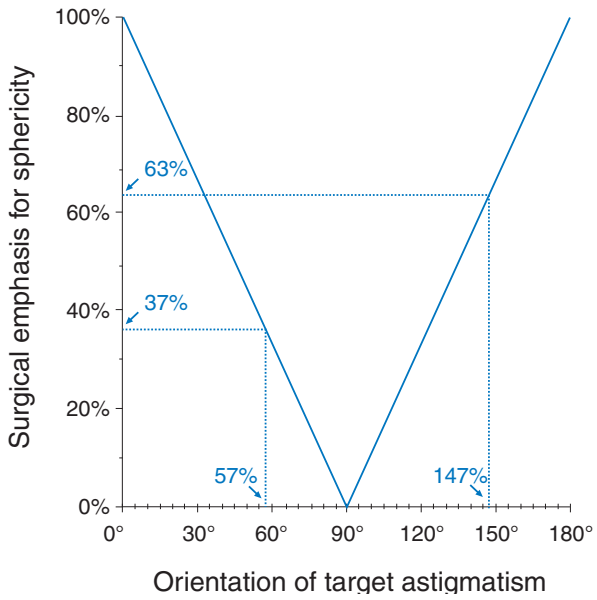


Figure 6. Linear relationship of surgical emphasis for corneal sphericity versus orientation of target topography, based on the notion that with-the-rule astigmatism is favourable and against-the-rule is unfavourable. This graph displays that if the targeted corneal astigmatism is at 90 degrees, all the ORA will be corrected on the cornea and the refractive cylinder target would be zero.

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Clinical QUIZ

ANSWER

Traumatic injury treatment

From page 11

The cornea was examined for any signs of foreign matter and to confirm that no perforation had occurred. A magnified image from the Pentacam showed the depth of the laceration to be about 70 per cent of the corneal thickness with some generalised oedema.

A bandage silicone hydrogel contact lens was placed on the eye to help smooth the corneal flap. Chloramphenicol drops q2h were prescribed with a 24-hour review.

24 hours later

- vision 6/7.5 L 6/6
- eye was feeling significantly better but still watery
- slitlamp examination showed the flap had sealed with no NaFl staining. Pentacam showed a slight decrease in generalised corneal oedema.

Flarex drops were introduced q4h to assist in reducing the inflammation in combination with the choloramphenicol q4h with a one-week review.

One week later

- vision 6/7.5+ 6/6
- Pentacam shows mild oedema only around wound. Silicone hydrogel contact lens removed, antibiotics stopped, continue with Flarex for one week qid. Review one month.

One month later

- no symptoms remaining, vision is 6/6 R&L
- slitlamp examination shows laceration scar and all other findings normal
- Pentacam shows more regular cornea and no oedema. Patient discharged.

(Figures 4-6)

Discussion

Significant vision loss from an eye injury was narrowly averted and the visual rehabilitation assisted by the use of a bandage contact lens with prophylactic antibiotics. Corticosteroids have helped reduce the severity of his scarring and assisted in reducing the discomfort associated with corneal oedema and inflammation.

Optometrists have the skills, equipment and knowledge to treat these traumatic injuries. Practitioners should develop their competency and confidence in dealing with ocular trauma to increase their scope of service and provide further opportunities to enhance their profession. ■

Figure 4. Slitlamp image at one month

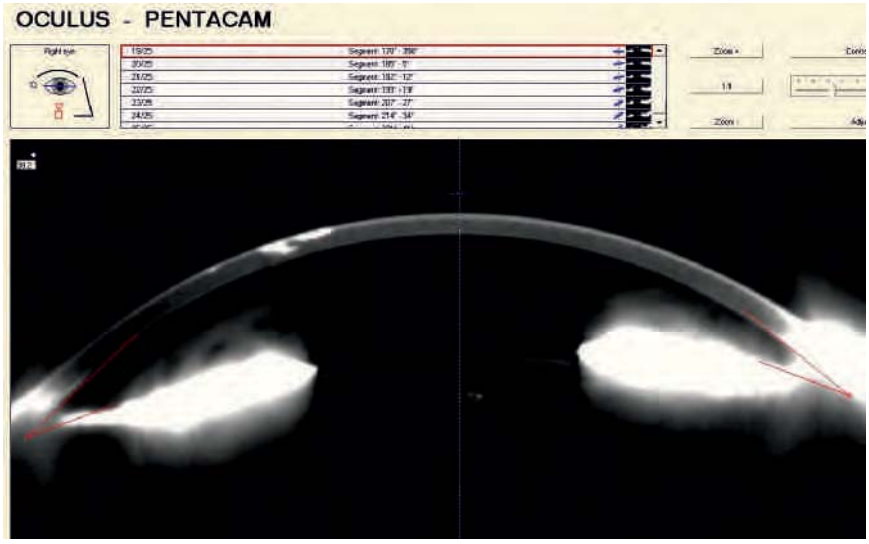

Figure 5. Pentacam image at one month

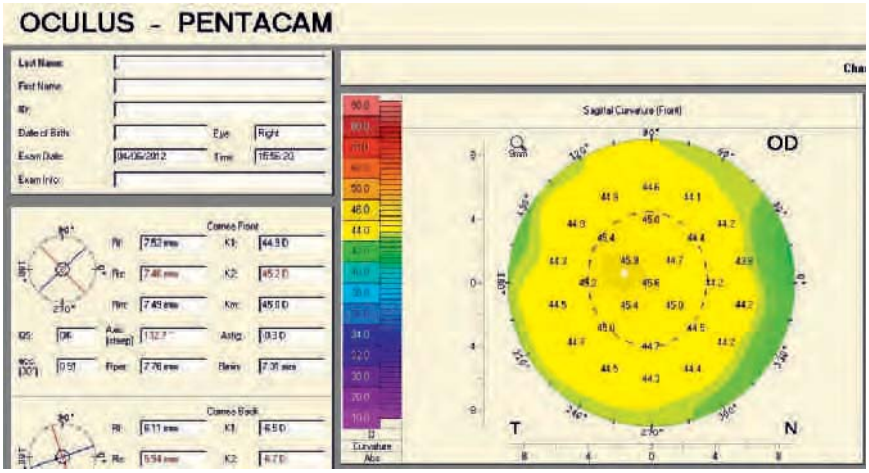

Figure 6. Topography at one month

Nutrition in primary eye care

Make the connection between between nutrition and ocular health for your patients by discussing their dietary choices and stressing 'prevention'.

Dr Jeffrey Anshel
OD FAAO
President, Ocular Nutrition Society USA

In the past 20 years, the diagnosis, treatment and management of ocular diseases have progressed at an unprecedented rate. Even as our ability to treat eye pathologies has increased, preventive measures like attention to dietary intake and micronutrient supplementation are still lagging.

All of this is occurring against the backdrop of a seminal moment in the practice of primary eye care. The World Health Organization has identified eye health as a significant health area and has stated that as the population ages, eye health will become an increasingly important issue in both the eye-care sector and society as a whole.[1]

Researchers across the globe are confirming the vital roles diet and lifestyle play in the protection against ocular diseases. For example, in a recent study into the relationships between diet, smoking and physical activity and the subsequent prevalence of age-related macular degeneration (AMD), researchers from the Department of Ophthalmology and Visual Sciences at the University of Wisconsin, USA found that those who exhibited a combination of three healthy behaviours (healthy diet, physical activity and not smoking) had 71 per cent lower odds for AMD compared with those who smoked, did not eat well and did not exercise.[2]

Public awareness

Nobody disputes the idea that proper nutrition is the vital to good health, but surprisingly few people make the connection between proper nutrition and ocular health. The evidence is growing in support of the role nutrients like zinc, vitamins C and E, lutein, Omega-3 fatty acids and zeaxanthin play in promoting health in the ageing eyes, but most in the public remain uninformed of the connection.

The Ocular Nutrition Society of America conducted the 'Eye on the Boomer' survey of 1001 men and women between the ages of 45 and 65 years. The survey revealed that although members of the 'baby boom' generation were generally aware of the importance of diet and exercise in prevention of heart disease and cancer, most were unaware that they could also affect eye health and vision.[3]

Although three-quarters of those surveyed took supplements to protect their joints, bones or heart, only 18 per cent said they took supplements to support their eye health.[3] In essence, the survey revealed that although ocular health is a significant issue, it is under-appreciated. People are concerned about their eyes but they don't know the simple steps they need to take to care for them.

Gradually, optometrists and ophthalmologists are finding that discussions about nutrition have a valid place in our efforts to treat eye disease. While conventional medications have their place, an increasing number of eye care health providers have chosen to integrate nutrition into their practices, which can serve to support their treatments.

Misconceptions

There are four general misconceptions regarding vitamins and minerals for patient care.

1. They are completely safe. While that is mostly true, they can be abused and cause dangerous effects if not taken appropriately.
2. They are ineffective. The effectiveness of nutrients is much more subtle and long-term than it is with drugs but they are effective.
3. They are all the same. Not true—especially when a multiple vitamin is considered. The type, form and amount of each nutrient can make a major difference in how it works.
4. More is better. This is the most common misconception; just because a certain amount is effective, it does not mean that 10 times that amount is 10 times more effective.

Assess nutrition intake

To successfully evaluate patients for nutrient deficiencies, it would be helpful to assess their nutrient intake. That sounds like a daunting task and one best left for nutritionists but a good sense of a patient's basic diet can be ascertained by asking just a few general questions.

- How many servings of fruits and vegetables do you eat daily?
- How many times a week do you eat fish, and what kind?
- Do you eat baked goods?
- Do you take a full spectrum multi-vitamin/mineral supplement?
- Do you limit the portion of food that you eat at each meal?

You should review products by companies that specialise in supplements for eye-care needs. Their products should have a valid scientific rationale available for anyone to review, as well as a website that puts science first before price and marketing.

Learning about nutrition can be a life-long proposition but can be extremely beneficial for you and your patients. 'Prevention' should be the byword of every consultation. In the final analysis, the onus is on the primary eye care provider to provide the public with the information they need to make informed dietary choices and to take the precautions necessary to ensure the health of their eyes.

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Abstracts

Dr Laura Downie
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Cataract affects sleep time and quality

Advanced lenticular nuclear sclerosis has been reported to be associated with an earlier bed-time and lower quality of sleep in late adulthood.

Clinical interviews and comprehensive ophthalmological examinations were performed on 378 patients over 59 years of age. The severity of cataract was graded overall (lenticular opacity) and for nuclear changes (lenticular nucleosclerosis,) using the Lens Opacities Classification System-III. Pittsburgh Sleep Quality Index was performed to determine sleep profiles.

Lenticular nucleosclerosis was associated with earlier bed-times and a lower quality of sleep. The authors concluded that stimulus by appropriate light to the eye is important for the physiologic regulation of sleep.

Aging Clin Exp Res 2012; July 6.

Retinal vascular geometry in Asian persons with diabetes and retinopathy

Retinal vascular parameters have been shown to be influenced by diabetes and the presence or absence of retinopathy in an older Asian cohort.

Retinal photographs from 2,735 participants of a population-based survey of Asian Malay persons aged 40-80 years were analysed. Specific retinal vascular parameters (tortuosity, branching angle, fractile dimension and calibre) were measured using a semi-automated computer-based program. Diabetes was defined as: random plasma glucose ≥ 11.1 mmol/litre, the use of diabetes medication, or physician-diagnosed diabetes. Retinopathy signs were graded from photographs using the modified Airlie House classification system.

Persons with diabetes (n = 594) were more likely to have straighter (less tortuous) arterioles and wider arteriolar and venular calibre than those without diabetes (n = 2141). Among subjects with diabetes, those with retinopathy had wider venular calibre than those without retinopathy. Among non-diabetic subjects, those with retinopathy had more tortuous vessels than those without retinopathy.

The findings suggest that subtle alterations in retinal vascular architecture are influenced by diabetes.

J Diabetes Sci Technol 2012; 6: 3: 595-605.

Reduced corneal endothelial cell density in pseudo-exfoliation syndrome

Corneal endothelial cell density has been found to be lower in Chinese patients with pseudo-exfoliation syndrome (PXS).

The medical records of 16 patients (n = 20 eyes) with pseudo-exfoliation syndrome who presented to the Qingdao University Medical College, China between July 2008 and June 2010 were retrospectively reviewed; 13 of these eyes also had glaucoma. Controls were the left eyes of 20 patients with bilateral age-related cataracts but no other ocular disease.

Specular microscopy was performed to analyse corneal endothelial cell density and morphology. Cell density, coefficient of variation in cell size and percentage of hexagonal cells in the corneal endothelium were evaluated.

The mean corneal endothelial cell density was significantly lower in PXS eyes (2298 ± 239 cells/mm^2) than control eyes (2652 ± 18 cells/mm^2); no significant differences were observed for any other of the measured parameters.

It was concluded that corneal endothelial cell density is decreased in Chinese patients with PXS; the presence of glaucoma was not related to endothelial cell changes.

Int J Ophthalmol 2012; 5: 2: 186-189.

Predictors of outcome in fungal keratitis

The severity of a fungal keratitis ulcer at presentation has been shown to be highly predictive of the visual outcome.

Data from 120 patients were collected during a prospective, randomised, controlled, double-masked clinical trial of treatment for fungal keratitis. Clinical features at presentation and demographics were collected at patient enrolment. Clinical outcomes were three-month visual acuity, infiltrate/scar size, time for re-epithelialisation and corneal perforation.

Significant predictors for worse three-month visual acuity were older age, worse presentation visual acuity, larger infiltrate size at presentation and a pigmented ulcer.

The authors identified that the clinical presentation characteristics of baseline acuity and infiltrate size can provide information to clinicians about prognosis, and may help guide management and treatment decisions. The prevention of corneal ulcer was deemed to remain important, as it is difficult to change the course of the ulcer once it has begun.

Eye (Lond) 2012; Jun 29; Epub ahead of print.

Vision-related quality of life in corneal graft patients

A study conducted in Hong Kong has found that corneal transplant recipients have a decreased vision-related quality of life.

The aim of the study was to assess vision-related quality of life of corneal transplant recipients using the National Eye Institute Visual Function Questionnaire-25 (NEI VFQ-25) and to identify the socio-demographic factors that associate with patients' self-assessment of perceived visual function. Thirty patients who received corneal transplants were included in this prospective, observational, cross-sectional study. Socio-demographic and clinical data, including age, sex, systemic health status, employment status, visual acuity, need for corneal transplantation, laterality of the graft and follow-up period were collected.

Patients who received bilateral corneal grafts were significantly less able socio-economically than those with a unilateral graft. Those who became unemployed or retired after transplantation were also significantly less able in both visual functioning and socio-emotional status.

It was concluded that apart from anatomical success and visual acuity, ophthalmologists should consider other aspects of visual outcome; those requiring bilateral grafts were advised to require more careful pre-operative consideration.

Eye (Lond) 2012; Jun 29; Epub ahead of print.

Correlation between clinical macular changes and retinal function in AMD

Steady state thresholds (14 Hz flicker and blue colour) and clinical signs in age-related macular degeneration (AMD) have been

demonstrated to be significantly correlated, suggesting that functional tests may be an effective tool for monitoring the progression of AMD in addition to clinical grading.

A total of 357 patients with visual acuity better than 20/60 in the study eye were recruited, including 64 participants with normal fundi and 293 patients with AMD, sub-classified into 12 groups based on the International Classification and Grading System.

Visual function was assessed using two steady-state tests (achromatic 14 Hz flicker and isoluminant blue colour) and two adaptation measurements (Cone Photo-Stress Recovery Rate and Rod Dark Adaptation Recovery Rate).

Steady-state measurements (flicker and blue colour) were observed to decline gradually along the entire hierarchy of fundus changes; flicker was found to be able to detect significant functional change as early as in the intermediate drusen group.

Invest Ophthalmol Vis Sci 2012; June 19; Epub ahead of print.

Association between dry eye and migraine

An increased frequency of dry eye disease has been shown to be present in patients with migraine.

This observational comparative study consisted of 33 migraine patients referred from neurology clinics and 33 control subjects referred from ophthalmology outpatient clinics–without systemic or ocular disease or a history of headache.

Significant differences in dry eye scores (tear break-up time, Schirmer test, lissamine green staining, ocular surface disease index scoring) were found between the patients with migraine and controls.

Cornea 2012; June 15; Epub ahead of print.

Impaired saccadic eye movements in primary open angle glaucoma

POAG alters saccadic programming and execution particularly in the case of moving targets.

The eye movements of eight POAG patients and four healthy age-matched controls were recorded. Four of the patients with POAG had documented visual field scotoma and four had no identifiable scotoma on standard visual field testing. Eye movements were monitored as observers watched static and kinetic targets; the gain, latency and velocity-peak latency of the saccades were recorded and analysed.

In POAG with abnormal visual fields, saccades to static targets were delayed and accuracy was reduced compared with controls. In POAG with normal visual fields, the latency and accuracy of the saccades for kinetic targets were impaired compared with controls.

J Glaucoma 2012; June 14; Epub ahead of print.

Corneal biomechanics are altered in diabetes

Corneal hysteresis and corneal resistance are significantly higher in diabetic patients with poor glucose control when compared with healthy subjects and patients with well-controlled diabetes.

One randomly chosen eye of 35 healthy subjects and 31 patients with diabetes were examined. Patients with diabetes were sub-divided into groups with HbA1c<7 per cent (n =14) and HbA1c>7 per cent (n = 17). Corneal hysteresis and corneal resistance factor were measured using the ocular response analyser; central corneal thickness using ultrasound pachymetry and intraocular pressure using Goldmann tonometry.

Both corneal hysteresis and corneal resistance were correlated to HbA1c, suggesting that the biomechanical properties of the cornea are altered depending on the level of glucose control.

Acta Ophthalmol 2012; June 13; Epub ahead of print. ■

Eye disease rising in baby boomers

There are more adults with eye disease than ever before, according to a report released in the United States.

Titled 'Vision Problems in the US', the report contains a searchable database in which eye disease prevalence rates are broken down by state, age, sex and race.

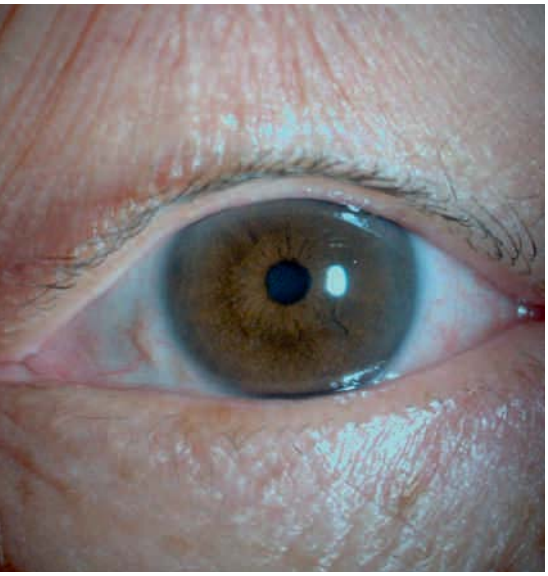
In its report, Prevent Blindness America and the National Eye Institute said that when compared with an earlier version of the study in 2000, data showed that age-related eye diseases were generally rising.

In 2010, it was estimated that more than 7.5 million people aged 40 years and older had diabetic retinopathy, which had increased from four million in 2000. Estimated data show that the number of people age 50 years and older with late age-related macular degeneration has increased 25 per cent. The number of people age 40 and older with cataracts had increased 19 per cent since 2000; the number of people age 40 or older with open-angle glaucoma had increased 22 per cent since 2000.

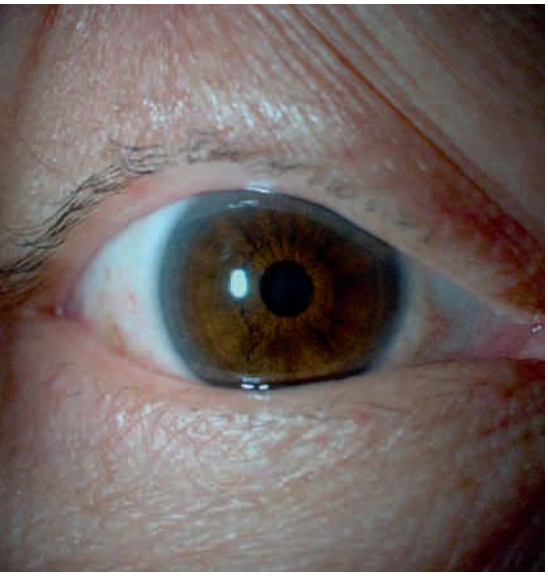
The cause of the increase in these four common eye diseases could be attributed to the ageing 'baby boomer' population, the CEO of Prevent Blindness America, Hugh Parry, said. He pointed out that the dramatic spike in diabetic retinopathy cases was a concerning consequence of the diabetes epidemic that the United States was facing.

The study's primary author, Dr David Friedman, said frequent examinations to detect these conditions early were more important than ever. He suggested that the number of people with eye diseases would far out-number the people graduating from programs to treat them and he called for a reassessment of how better, more efficient care could be provided.

Online report: www.preventblindness.org. ■



Left eye: light colour iris with beaten-metal appearance



Right eye: normal iris

Fuchs heterochromic is often

Case report

Dr William Trinh
BOptom OD
Sydney NSW

A 57-year-old Asian male presented to our practice complaining of persistent left eye discomfort despite using a variety of over-the-counter eye-drops for the previous two months.

His ocular history included refractive amblyopia in the left eye. His current medical history was unremarkable but a small brain tumour had been removed a year before.

On examination, his best corrected vision was R +2.00 DS 6/6 and L + 5.75 DS 6/7.5. Intraocular pressures were R 11 L 8. mmHg.

Slitlamp examination revealed a slightly lighter brown iris in the left compared to the right, with a beaten-metal appearance. Several fine keratic precipitates with cells were found in the left anterior chamber.

Differential diagnosis

Posner-Schlossman Syndrome, which has similar presentations of mild anterior chamber reaction with elevated intraocular pressures but without iris discoloration.

Diagnosis

The patient was diagnosed with Fuchs heterochromic iridocyclitis (FHI).

Management

The patient was symptomatic of his iritis even without elevated intraocular pressure. Clinically, a decision was made to initiate steroid eye-drops to suppress the inflammation. Prednefrin Forte 1% was prescribed qid

Bisphosphonates risk for uveitis

Patients taking oral bisphosphonates for the first time could be at a higher risk of developing scleritis and uveitis.

A large, retrospective cohort study analysed the incidence rate of scleritis and uveitis in 10,827 first-time bisphosphonate users and found rates of 29/10,000 person-years for uveitis and 63/10,000 person-years for scleritis.

In contrast, the incidence rate among 923,320 non-users was 20/10,000 person-years for uveitis and 36/10,000 for scleritis.

Bisphosphonates are a group of drugs that prevent the loss of bone mass and are commonly used to treat osteoporosis and similar diseases.

The researchers said that the study was the first to quantify the risk of uveitis and scleritis with bisphosphonates and the importance of the findings were of particular importance to ocular health practitioners.

They noted that previously reported cases of adverse ocular events were primarily associated with two types of bisphosphonates, alendronate and risedronate, so the next step was to assess whether the risk of uveitis and scleritis varied with individual bisphosphonates.

Canadian Medical Association Journal 2012. Published online. ■

iridocyclitis underdiagnosed

Tell-tale iris discoloration from atrophy was the key.

for the left eye. At one week follow-up, cells and KP were resolved and the patient was asymptomatic. The steroid eye-drops were then tapered over one week.

Discussion

Ninety per cent of FHI is presented as a unilateral condition[1] due to the lack of symptoms,[2] minimal signs of inflammation and discolouration of iris from iris atrophy in early stage of condition. FHI is often underdiagnosed.

Studies also show that two to 17 per cent of patients with anterior uveitis have FHI[3,4] and FHI eventually develops posterior capsular cataracts and some 15.7 per cent[5] develop secondary glaucoma. The degenerative change and the inflammation of trabecular meshwork have been suggested as a possible cause of the secondary glaucoma. It is important that clinicians are highly suspicious of FHI during the routine slitlamp examination when encountering the discoloration of iris and mild iritis.

The patient should also be well educated on the nature of the condition so that when FHI recurs, the condition can be managed quickly, efficiently and without undergoing extensive and expensive systemic work-up when the patient presents to different eye-care practitioners.

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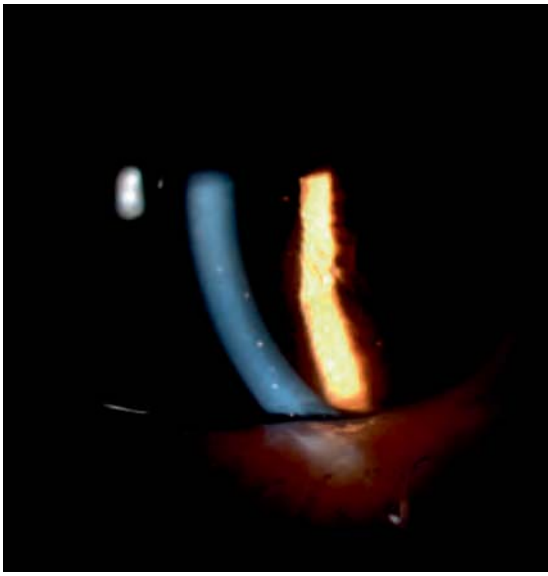
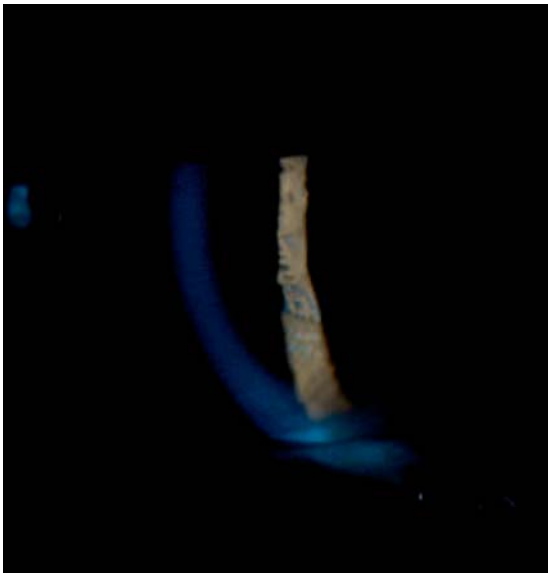


Left eye day 1: with keratic precipitates



Left eye day 7: keratic precipitates resolved by Prednisolone Forte eye-drops treatment

Glaucoma worse in winter

Seasonal weather variations may influence intraocular pressure and visual field sensitivity, particularly in patients with early glaucoma.

Researchers from the Devers Eye Institute in Oregon, USA, analysed data from the Ocular Hypertension Treatment Study and found IOP and visual field sensitivity peaked during winter months, leading researchers to suggest that factors not related to glaucoma could be hampering the detection of disease progression and IOP changes.

Data were collected from 33,873 visits made by 1,636 participants over a median of 12.5 years from 22 participating clinics. The clinics, all based in the United States, were divided into six geographic regions, which were determined to be similar based on size and timing of seasonal changes in precipitation, temperature and sunlight hours.

The researchers found the effect was five times larger in the northern states such as Michigan and Minnesota, compared to the southern states such as Florida and Texas.

The size of variation ranged from 0.14 mmHg to 0.39 mmHg. In five of the six regions, statistically significant seasonal variations in visual sensitivity occurred, ranging from 0.04 dB to 0.13 dB.

Crocodile tears

Botulinum toxin A shuts off the waterworks with no side-effects.

Joel P Causey
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Pacific University College of Optometry, Forest Grove OR
Dr Leonid Skorin Jr
OD DO MS FAAO FAOCO
Mayo Clinic Health System
Albert Lea MN, USA

Crocodile tears refers to tears wept in an insincere display of emotion. The term originated from a description of crocodiles tearing while eating their prey but it is more likely to describe the flicker of their nictitating membrane. Crocodile tears syndrome (gustatory hyperlacrimation) is a consequence of aberrant regeneration following damage to the facial nerve (cranial nerve VII). This subsequent pathologic misdirection of salivary efferent parasympathetic fibres results in the over-activation of the lacrimal gland and inappropriate tearing in response to the stimulation of gustatory taste buds.

The lacrimal gland produces emotional and reflex tearing but not maintenance tears such as those secreted by the accessory lacrimal glands of Wolfring and Krause. Neural input to the lacrimal gland originates from the facial nucleus housed in the reticular formation of the pons.[1] The larger facial motor root relays efferent motor signals to the facial muscles.[1] The smaller root contains both afferent sensory fibres and efferent secretomotor fibres. The efferent parasympathetic fibres carry signals to the salivary glands and main lacrimal gland.[1,2] Both motor and sensory fibres course through the internal acoustic foramen before entering the geniculate ganglion.[3,4] It is here that the parasympathetic lacrimal fibres join the greater superficial petrosal nerve. The gustatory fibres branch shortly after but the facial motor neurons continue through the stylomastoid foramen, exiting the skull to innervate the facial muscles.[2,3,5] This tight, tortuous route of cranial nerve VII is prone to damage.

Implicated aetiologies in the paralysis of cranial nerve VII include compression by trauma and inflammation. Facial nerve paralysis can be idiopathic and in the absence of central nervous system disease is referred to as Bell's palsy. Bell's palsy causes ipsilateral hemifacial paralysis, affecting the upper and lower facial muscles innervated by cranial nerve VII.[6] The muscles of facial expression lose voluntary control and result in a flaccid, hypotonus appearance. The damage occurs at the level of the lower motor neurons, blocking both ipsilateral and contralateral facial nerve fibres. During this time, adequate patient education along with dry eye therapy to prevent keratopathy is the best management. Although Bell's palsy is self-limiting, the resolution timeline is variable and the paralysis may not be resolved for months.

Following the resolution of the hemifacial paralysis, adverse consequences remain in 10 to 20 per cent of cases.[7] Permanent changes to the normal neural pathway may occur. Aberrant regeneration or synkinesis is the misdirection of nerve fibres. Facial nerve synkinesis develops at the level of the greater superficial petrosal nerve where collateral axonal branches of the efferent salivary fibres sprout and begin to re-innervate the lacrimal gland.[3] The gustatory afferent arc is redirected to the lacrimal gland, resulting in tearing whenever the taste buds are stimulated. This acquired gustolacrimal reflex may develop weeks to months following resolution of the paralysis.[3]

Case report

A 79-year-old white male presented to the clinic with complaints of right-sided, unilateral eyelid spasms and epiphora. The patient stated that the excessive tearing occurred while eating, yawning and talking. He also found reading difficult as the tearing blurred his vision. The uncontrollable, hyperlacrimation was debilitating and made social events unenjoyable. These symptoms reduced his quality of life.

The patient had a history of unilateral Bell's palsy on his right side. The episode of paralysis had been resolved prior to his visit to the eye clinic. During the course of examination, forceful involuntary hemifacial spasms occurred frequently. Biomicroscopy evaluation of the anterior and posterior segments was unremarkable. Besides unilateral epiphora and hemifacial spasms, the examination revealed no other secondary complications from the resolved paralysis. No residual ectropion, lagophthalmos or signs of corneal dryness were noted in his right eye. Corneal surface evaluation with sodium fluorescein revealed neither positive

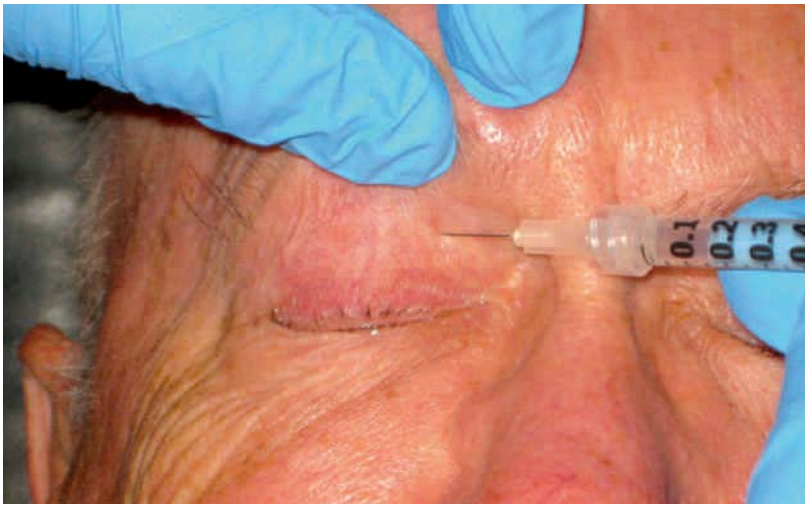

Figure 1. Subcutaneous injection of botulinum toxin A into the orbicularis oculi

syndrome

staining patterns nor punctate epithelial erosions.

We proposed the benefits of therapeutic botulinum toxin A (Botox) injections for the treatment of both the hyperlacrimation and eyelid spasm. The patient received one dose of 2.5 units/0.1 mL via subcutaneous injection in both the lateral and medial aspect of the right upper eyelid (Figure 1). The lacrimal gland was identified under direct visualisation and an injection of 2.5 units/0.1 mL was administrated through the conjunctiva into the gland (Figure 2). The patient tolerated the injections well.

We followed up with the patient twice over the telephone after two and six weeks. The patient reported partial resolution of the unilateral epiphora and near complete resolution of the facial spasm. The frequency and severity of tearing had decreased. He reported no side-effects and was satisfied with the treatment results. An increased unit concentration injected into the lacrimal gland may be warranted at his return visit.

Botulinum toxin A

Commercially available pharmaceutical botulinum toxin type A (Botox) is formulated as a lyophilised powder and packaged in a glass vial. Prior to administration, it must be reconstituted with sterile saline. Care must be taken when preparing the solution not to denature the protein and diminish the toxin's biological activity. This can be avoided by slowly introducing the saline into the vial. The freeze-dried powder dissolves without requiring shaking. The suggested, prepared concentration of 2.5 units/0.1 mL per injection site is preferred for initial or conservative treatment.

Botulinum toxin is a neurotoxin produced by the gram-positive *Clostridium botulinum*. The toxin's mechanism of action works at the neuromuscular junction through inhibiting the release of acetylcholine.[8] It appears to have the same mechanism of action at the neuroglandular junction. The toxin is internalised in the presynaptic axonal terminal via receptor-mediated endocytosis.[8] Here it attaches to the vesicle containing acetylcholine; inhibiting it from binding to the nerve membrane and releasing its contents.[8]

The average onset-of-action is reported at three to seven days and maximal effectiveness peaks around 21 days. The duration varies between patients and use but generally lasts between three to four months with some accounts of up to six months.[8,9] At this time new nerve terminals have sprouted and re-synapsed to re-establish the motor endplate.[9]

Contraindications for botulinum toxin include concurrent neuromuscular disorders such as Lambert-Eaton syndrome and myasthenia gravis.[8] Adverse ocular side-effects are typically temporary and localised to the area of injection. The possible side-effects include blepharoptosis, diplopia, ophthalmoplegia, reduced blinking frequency, ectropion and/or lagopthalmos with subsequent corneal exposure keratopathy.[10] There have been no proven systemic side-effects from periocular botulinum toxin injections.[10] Other therapeutic uses of botulinum toxin include: benign essential blepharospasm, hemifacial spasm, myokymia, strabismus, facial frown lines, cervical dystonia, chronic migraine headaches, hyperhidrosis and urinary incontinence.

The first reported account of relief of gustatory hyperlacrimation with botulinum toxin was in 1998 by Boroojerdi and colleagues.[3,11,12] Since then, numerous cases have been reported and shown success. In most cases, the results are the complete or near-complete resolution of the tearing. Both transcutaneous and transconjunctival routes of administration have been applied. Transconjuctival injection into the lacrimal gland under direct visualisation reduces the risk of inducing eyelid ptosis and/or muscle paralysis.

Each patient responds uniquely to the toxin. A successful dose for one patient may not be appropriate for another. Most commonly, 2.5 to 15.0 units/0.1 mL are used in a single dose treatment. Once the single unit dosing that achieves resolution is determined, no additional benefit for either increased duration or decrease of symptoms can be gained by using higher concentrations of the toxin.

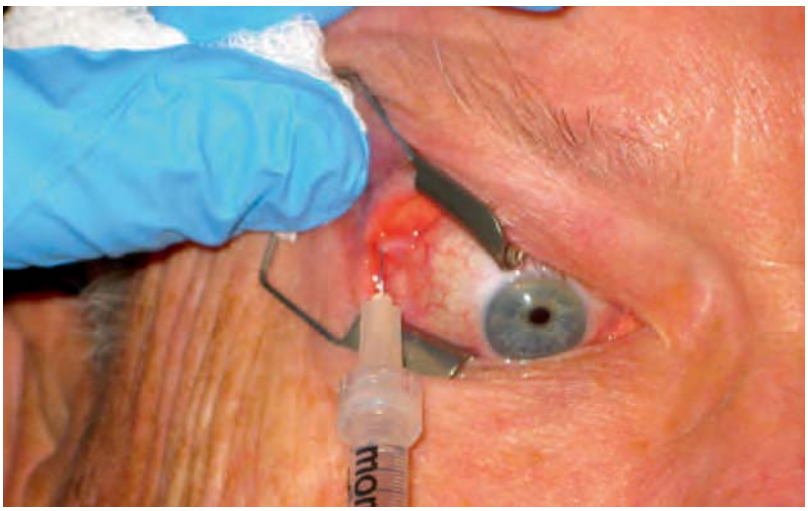

Figure 2. Botulinum toxin A transconjunctival injection into the lacrimal gland, with eyelid speculum to increase lid aperture for better visualisation of the gland

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PBS list of medicines for optometrists

1 August 2012

	Product	Max qty	Repeats
Antiglaucoma preparations			
Betaxolol eye-drops, suspension, 2.5 mg (as hydrochloride)/mL, 5 mL	Betoptic S	1	5
Betaxolol eye-drops, solution, 5 mg (as hydrochloride)/mL, 5 mL	Betoptic, BetoQuin	1	5
Bimatoprost eye-drops 300 micrograms/mL, 3 mL	Lumigan	1	5
Bimatoprost with timolol eye-drops containing 300 micrograms bimatoprost with timolol 5 mg (as maleate)/mL, 3 mL	Ganfort 0.3/5	1	5
Brimonidine eye-drops containing brimonidine tartrate 2 mg/mL, 5 mL	Alphagan, Enidin	1	5
Brimonidine Tartrate eye drops 1.5 mg per mL (0.15%), 5 mL	Alphagan P 1.5	1	5
Brimonidine with timolol eye-drops containing brimonidine tartrate 2 mg with timolol 5 mg (as maleate)/mL, 5 mL	Combigan	1	5
Brinzolamide eye-drops 10 mg/mL, 5 mL	Azopt, BrinzoQuin	1	5
Brinzolamide with timolol eye-drops containing brinzolamide 10mg/mL with timolol 5mg (as maleate)/mL, 5mL	Azarga	1	5
Dorzolamide eye-drops 20 mg (as hydrochloride)/mL, 5 mL	Trusopt	1	5
Dorzolamide with timolol eye-drops containing dorzolamide 20 mg (as hydrochloride) with timolol 5 mg (as maleate)/mL, 5 mL	Cosopt	1	5
Latanoprost eye-drops 50 micrograms/mL, 2.5 mL	Xalatan	1	5
Latanoprost with timolol eye-drops 50 micrograms latanoprost with timolol 5 mg (as maleate)/mL, 2.5 mL	Xalacom, Latanocom	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 10 mg/mL, 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 20 mg/mL, 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 40 mg/mL, 15 mL	Isopto Carpine	1	5
Timolol eye-drops 2.5 mg (as maleate)/mL, 5 mL	Tenopt, Timoptol	1	5
Timolol eye-drops 5 mg (as maleate)/mL, 5 mL	Tenopt, Timoptol	1	5
Timolol eye-drops (gellan gum solution) 2.5 mg (as maleate)/mL, 2.5 mL	Timoptol XE	1	5
Timolol eye-drops (gellan gum solution) 5 mg (as maleate)/mL, 2.5 mL	Timoptol XE	1	5
Timolol eye gel 1 mg (as maleate)/g, 5 g	Nyogel	1	5
Travoprost eye-drops 40 micrograms/mL, 2.5 mL	Travatan	1	5
Travoprost with timolol eye-drops 40 micrograms travoprost with timolol 5 mg (as maleate)/mL, 2.5 mL	Duotrav	1	5

By writing a PBS prescription for an antiglaucoma agent, the prescriber is certifying that the criteria set out in the PBS guidelines are satisfied

	Product	Restriction	Max qty	Repeats
Anti-viral eye preparations		Restricted:		
Aciclovir eye ointment 30 mg per g (3%), 4.5 g	Zovirax	Herpes simplex keratitis	1	0
Antibiotics		Unrestricted		
Chloramphenicol eye-drops 5 mg/mL (0.5%), 10 mL	Chlorsig, Chloromycetin		1	2
Chloramphenicol eye ointment 10 mg/g (1%), 4 g	Chlorsig, Chloromycetin		1	0
Ciprofloxacin eye-drops 3 mg per mL (0.3%), 5 mL	CiloQuin, Ciloxan	Authority required: bacterial keratitis	2	0
Framycetin sulfate eye-drops 5 mg/mL (0.5%), 8 mL	Soframycin		1	2
Gentamicin sulfate eye-drops 3 mg/mL (0.3%), 5 mL	Genoptic	Restricted: Suspected pseudomonal eye infection	1	2
Ofloxacin eye-drops 3 mg per mL (0.3%), 5 mL	Ocuflox	Authority required: bacterial keratitis	2	0
Tobramycin eye-drops 3 mg per mL (0.3%), 5 mL	Tobrex	Restricted: Suspected pseudomonal eye infection	1	2
Tobramycin eye ointment 3 mg per g (0.3%), 3.5 g	Tobrex	Restricted: Suspected pseudomonal eye infection	1	0
Anti-inflammatory agents				
Dexamethasone eye-drops 1 mg / mL (0.1%), 5 mL	Maxidex	Applications for increased maximum quantities and/or repeats will not be authorised.	1	0
Fluorometholone eye-drops 1mg/mL (0.1%), 5 mL	Flucon, FML Liquifilm		1	0
Fluorometholone acetate eye-drops 1 mg/mL (0.1%), 5 mL	Flarex		1	0
Flurbiprofen sodium eye-drops 300 µg/mL (0.03%) single dose units 0.4 mL, 5 mL	Ocufen		1	0
Hydrocortisone acetate eye ointment 10 mg/g (1%), 5 g	Hycor		1	0
Prednisolone acetate with phenylephrine hydrochloride eye-drops 10 mg-1.2 mg per mL (1%-0.12%), 10 mL	Prednefrin Forte	Restriction: Uveitis. Applications for increased maximum quantities and/or repeats will not be authorised.	1	0
Anti-allergy agents		Restricted:		
Sodium cromoglycate eye-drops 20 mg/mL (2%), 10 mL	Cromolux	Vernal keratoconjunctivitis	1	5
	Opticrom		1	5

Continued

	Product	Restriction	Max qty	Repeats
Tear supplements		Restricted:		
Carbomer eye gel 2 mg/g (0.2%), 10 g	Geltears	Severe dry eye including	1	5
	PAA	Sjögren's syndrome	1	5
	Viscotears		1	5
Carmellose sodium with glycerin eye-drops 5 mg-9 mg per mL (0.5%-0.9%), 15 mL	Optive		1	3
Carmellose sodium eye-drops 10 mg/mL (1%), 15 mL	Refresh Liquigel		1	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), 15 ml	Refresh Tears plus		1	5
Hypromellose eye-drops 3 mg/mL (0.3%), 15 mL (contains sodium perborate)	In a Wink Moist'ing		1	5
	Genteal		1	5
Hypromellose eye-drops 5 mg/mL (0.5%), 15 mL	Methopt		1	5
Hypromellose with carbomer 980 ocular lubricating gel 3 mg-2 mg/g (0.3-0.2%), 10 g	HPMC PAA		1	5
	Genteal gel		1	5
Hypromellose with dextran eye-drops 3 mg-1 mg/mL (0.3%-0.1%), 15 mL	Poly-Tears		1	5
	Tears Naturale		1	5
Polyethylene glycol 400 with propylene glycol drops 4 mg-3 mg/mL (0.4-0.3%); 15 mL	Systane		1	5
Polyethylene glycol 400 eye drops 2.5 mg per mL (0.25%), 15 mL	Blink Intensive Tears		1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL	PVA Tears, Liquifilm Tears		1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL	PVA Forte, Liquifilm Forte		1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL (contains sodium chorite/hydrogen peroxide as preservative)	Vistil		1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL (contains sodium chorite/hydrogen peroxide as preservative)	Vistil Forte		1	5
Unpreserved tear supplements		Authority required:		
Carbomer 974 ocular lubricating gel 3 mg/g (0.3%), single dose units 0.5 g, 30	Poly Gel	Severe dry eye syndrome in patients sensitive to preservatives in multi-dose eye-drops	3	5
Carbomer eye-gel 2 mg per (0.2%) , single dose units 0.6 mL, 30	Viscotears Gel PF	As above	3	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), single dose units 0.4 mL, 30	Cellufresh		3	5
Carmellose sodium eye-drops 10 mg/mL (1%) , single dose unit 0.4 mL, 30	Celluvisc		3	5
Carmellose sodium eye-drops 2.5 mg/mL (0.25%), single dose units, 0.6 mL, 24	TheraTears		4	5
Carmellose sodium ocular lubricating gel 10 mg/mL (1%), single dose 0.6 mL, 28	TheraTears		3	5
Carmellose Sodium with Glycerin eye drops 5 mg-9 mg per mL (0.5%-0.9%), single dose units 0.4 mL, 30	Optive		3	5
Hypromellose with dextran eye-drops 3-1 mg/mL (0.3-0.1%), single 0.4 mL, 28	Bion Tears		3	5
Polyethylene glycol 400 with propylene glycol drops 4 mg-3 mg/mL (0.4-0.3%); single dose units 0.8 mL, 28	Systane		2	5
Polyethylene glycol 400 eye-drops 2.5 mg per mL (0.25%), single dose units 0.4 mL, 20	Blink Intensive Tears		5	5
Soy lecithin eye spray 10 mg/mL (1%), 10 mL	Tears again		2	5
Topical ocular lubricant ointments		Unrestricted		
Paraffin compound eye ointment 3.5 g	Polyvisc, Duratears		2	5
Paraffin pack containing 2 tubes compound eye ointment 3.5 g	Polyvisc (2 pack), Ircal (2 pack), Lacri-Lube (2 pack)		1	5

OPTOMETRISTS
ASSOCIATION AUSTRALIA