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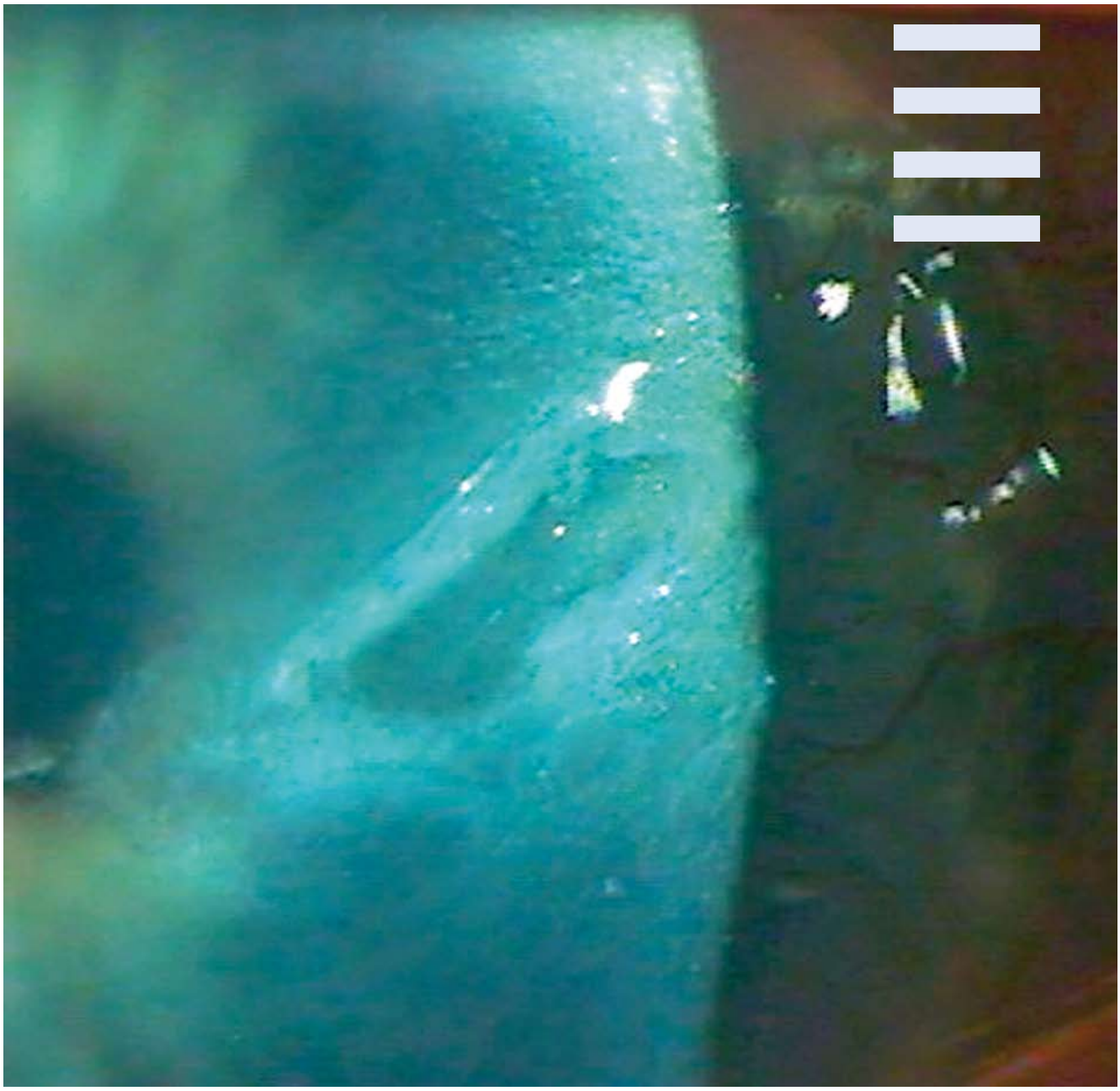
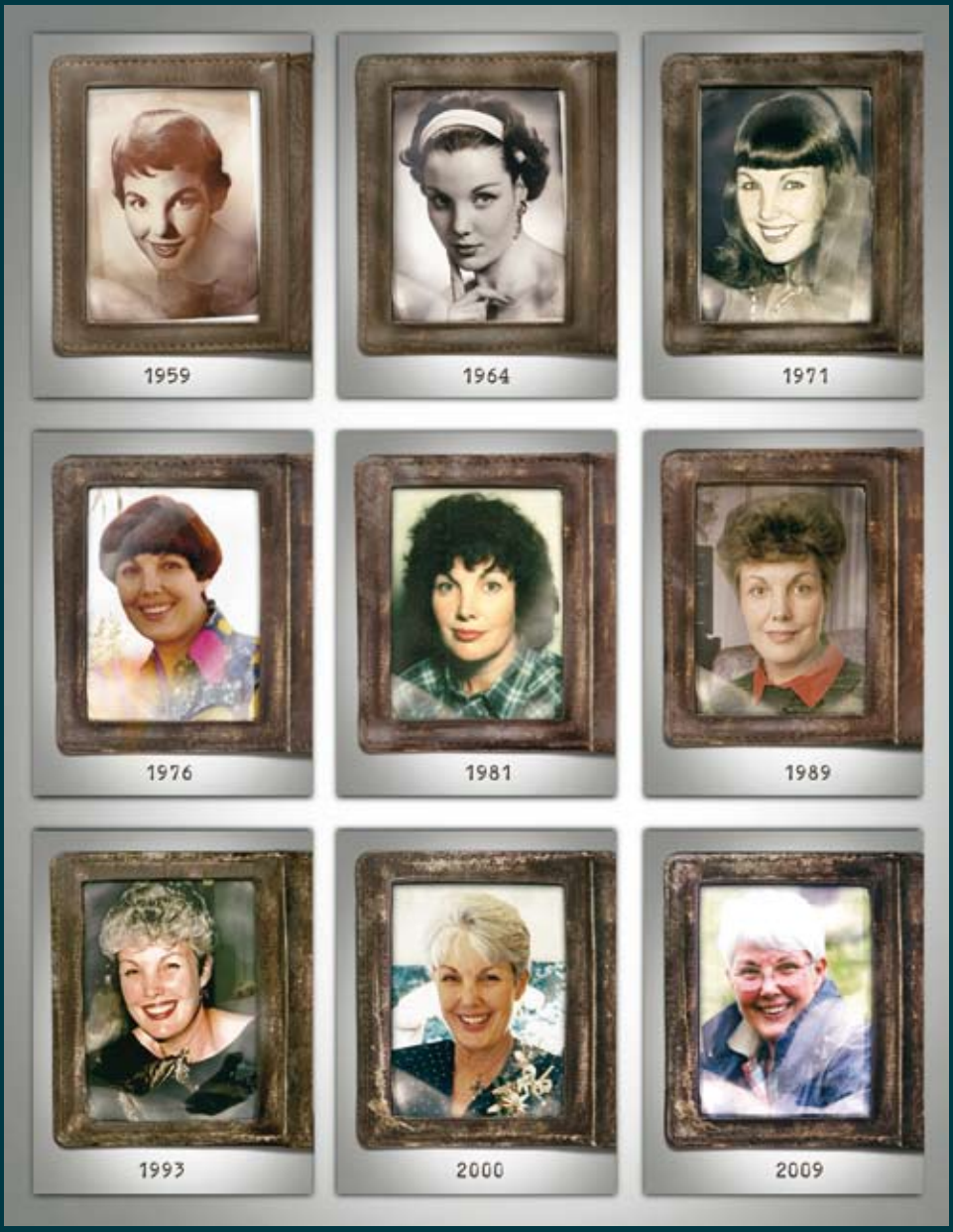
SUPPLEMENT TO AUSTRALIAN OPTOMETRY JUNE 2009



- Chronic, non-infectious uveitis
- Herpes simplex keratitis
- Systemic medications affecting ocular health
- Botulinum toxin
- Treat dry eye with sodium hyaluronate



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

*Low IOP is associated with reduced progression of visual field defect in patients with glaucoma.[4,5] ^Xalatan used as adjunctive therapy.[1]

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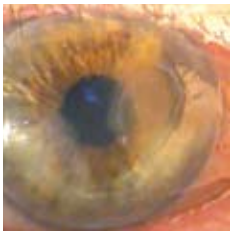
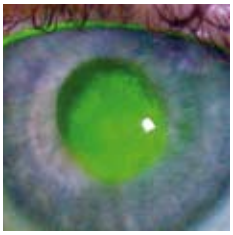
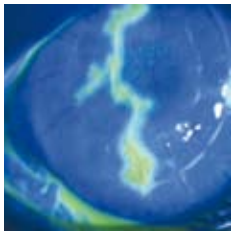
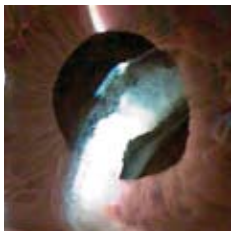
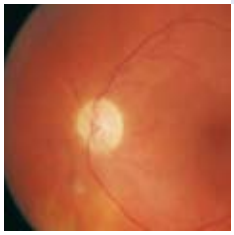

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COVER
Non-healing neurotrophic ulcer requiring antibiotic prophylaxis and topical steroid treatment

All references to pharmaceutical preparations in *Optometry Pharma* are applicable within Australia

Chronic, non-

Suppressing a patient's immunity carries the risk that the complications of systemic therapy will cause a healthy patient with an eye disease to become a sick patient with an eye disease.

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The treatment of chronic, non-infectious uveitis is a difficult task that often involves choosing the least of all evils. Both the disease and its treatment may be complicated by systemic and ophthalmic problems. This review of recent changing trends in the therapeutic approach to this condition is limited to the treatment of uveitis in patients who do not have an active underlying systemic disease such as sarcoidosis or ankylosing spondilitis, and whose main clinical problem is their eye disease. This is a very common clinical scenario.

The prevailing treatment paradigm for significant, chronic, non-infectious uveitis has been based on systemic or orbital (steroid injection) therapy, mainly used for problems in the posterior segment (vitreous haze, macular oedema, retinal and choroidal inflammation). Most specialists in the field have used orbital long-acting steroid injections as a first choice in unilateral disease, and systemic therapy in patients with bilateral disease. Systemic therapy is based on corticosteroids and often on the addition of immune-suppressive drugs, so-called steroid-sparing agents.

While steroids usually have a rapid and dramatic therapeutic effect, immune suppressive drugs are designed to maintain quiescence of the disease, rather than immediately control the uveitis. Their action has a slower onset, often taking several months to kick in, and their independent beneficial effect is hard to measure as they are usually given in conjunction with oral steroids.

Both oral steroids and immune suppressive agents cause many systemic side-effects, some of which are life threatening, and many others are very disruptive to the patients' lifestyle. Examples include weight gain, psychosis, severe systemic infections, osteoporosis, liver and kidney toxicity, abnormal hair growth, hypertension and diabetes. Avoiding such complications requires medical experience and expertise, and even in the most experienced hands they are sometimes inevitable. In addition, some popular immune-suppressive agents are very expensive (for example, Cyclosporine, mycophenolate and biologics such as infliximab).

In the past decade or so, more experience has been accumulating with intraocular (mainly intravitreal) therapy, both in the field of retinal diseases and in uveitis. Steroids and various biological treatments have gained popularity and proved to be extremely efficacious against various pathologies in the posterior segment.

For example, a single intravitreal injection of 4 mg of long-acting steroid (triamcinolone) produces a rapid effect on cystoid macular oedema, roughly equivalent to that of a total of 4,000 mg systemic steroid (prednisolone) given systemically over two to three months, but without any systemic side-effects. A vial of the drug costs a few dollars. It is important to mention that periocular (orbital) steroid injections can provide beneficial effects similar to those of intraocular injections in most but not all patients. For patients who fail to improve with periocular steroids, intravitreal administration is almost always more effective and reverses the oedema. In fact, some uveitis specialists believe that if a patient's posterior uveitis does not improve significantly with intravitreal steroids, it is unlikely to improve

infectious uveitis

with any other medical therapy.

Despite its extremely high efficacy, intravitreal (and, on a lower scale, peri-ocular) steroid treatment carries a high risk of ocular complications. The most common complication is increased intraocular pressure, with or without glaucoma. It is usually manageable medically and requires filtration surgery in a sizeable minority of patients. Other complications include cataract, retinal detachment and endophthalmitis. Most of the ocular complications are manageable medically or surgically.

In summary, systemic toxicity is traded off for ocular complications, most importantly glaucoma.

The other shortfall of triamcinolone injections is the duration of their effect, three months on average. Patients may have initially a favourable response but often require repeated injections as the effect wears off. This potentially increases the risk of surgical complications such as retinal detachment and endophthalmitis. The long-term safety profile of multiple intravitreal injections is yet unknown. This is mainly of concern in young patients, who may have to live with uveitis for decades.

Several drug delivery systems have been introduced in the past decade, releasing steroids and other drugs from a small, surgically implanted pellet. The pellet is usually inserted through the pars plana and either left in the vitreous cavity or sutured to the sclera. The longest-acting system available at present releases a steroid slowly and steadily over two years. The current cost of this device in the USA is close to AU$30,000. Results from clinical trials have shown remarkable efficacy in suppressing uveitis and improving vision for from two to three years.

It is hard to compare this treatment to the very effective and much cheaper option of repeated intravitreal steroid injections: the two have not been compared head to head. The side-effects are similar, with 40 per cent of implant patients requiring glaucoma surgery. A multi-central, randomised controlled trial called the MUST trial is being conducted, comparing this implant to systemic 'traditional' therapy. The study is sponsored by the National Eye Institute in the USA. In Australia, The Ocular Immunology Clinic at the Royal Victorian Eye and Ear Hospital in Melbourne is participating in the MUST trial.

Chronic uveitis will continue to be a therapeutic challenge in the foreseeable future. While various treatments are available to modify and suppress the patient's immunity, they all carry various systemic risks and their efficacy is not always well established.

Local treatment with steroids and perhaps other molecules in the future offers unquestionable efficacy in most patients but the ocular side-effect, mainly glaucoma, may require aggressive therapy. More patients with chronic uveitis will probably develop surgical glaucoma with the availability and rising popularity of local steroid injections and implants. While far from perfect, this treatment philosophy may avoid the unfortunate scenario in which a healthy patient with an eye disease becomes a sick patient with an eye disease, due to complications of systemic therapy.

Steroids and various biological treatments have gained popularity and proved to be extremely efficacious against various pathologies in the posterior segment.

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Generic versus brand-name products

Oils ain't oils. Excipients, the therapeutically inactive components of a medication, can affect the bioavailability of the active ingredient in many ways.

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Generic drugs comprise a large percentage of prescriptions for drugs today. Generics can be offered at a greatly reduced price compared to their brand-name counterparts; reduced cost to the consumer leads to increased accessibility, and studies show this leads to improved adherence to therapy.[1]

Generic medications are less expensive because the cost of manufacturing is less, and multiple manufacturers compete, which drives down prices. While generics benefit the patient financially, they are not without controversy, especially in the case of ophthalmic generics.

When a new drug comes on the market, regulations are in place to protect the intellectual property rights of the manufacturer that invested large sums of money into research and development of that drug.[2] After a certain period, other manufacturers can use the technology without the financial burden of conducting expensive clinical trials.

Regulations ensure that manufacturers establish bioequivalence to the original medication.[2] This is supposed to ensure the therapeutic action of the generic form will be the same as the branded product. Companies save a lot of money by foregoing clinical trials, which is one way they are able to offer generics at such a reduced price.

Generic drugs differ from their brand-name form in that they have excipients different from the original drug.[3] Excipients are the therapeutically inactive components of a medication. They are of interest because they affect the bioavailability of the active ingredient.[3]

Bioavailability refers to the percentage of active ingredient that is absorbed and delivered to the site of action.[3] Excipients in topical ophthalmic solutions include buffers, antimicrobial agents, antioxidants, salts and detergents. The Table (left) lists some commonly used excipients.

Excipients can influence bioavailability of the active ingredient in a medication in multiple ways: by altering pH, viscosity, surfactants and osmotics. The pH of a drug solution will determine if the active ingredient is in the ionised or non-ionised form.[3] The non-ionised form will be more lipid-soluble and cross cell membranes more readily but

Benzalkonium	Phenylmercuric acetate and nitrate
Benzethonium	Polyoxypropylene-polyoxyethelenediol
Boric acid	Polysorbate 80
Chlorbutanol	Propylparaben
Creatine	Sodium bisulfite
Ethylenediaminetetraacetic acid (EDTA)	Sodium carbonate
Glycerine	Sodium citrate
Hydrochloric acid	Sodium nitrate
Mannitol	Sodium phosphate
Methylcellulose	Sodium sulfite
Methylparaben	Tyloxapol
Oxine sulfate	Zinc sulfate
Phenylethyl alcohol	

Excipients frequently added to ophthalmic solutions

the ionised form is more stable with a longer shelf-life.[3] The final pH of a drug solution is a balance between bioavailability and shelf-life of the drug, so bioavailability of the active ingredient will be reliant on the pH the manufacturer decides to have.

Viscosity of a drug is influenced by adding substances such as hydroxypropyl methylcellulose and polyvinyl alcohol.[3] Studies support that increased viscosity improves drug-corneal contact time, thus increasing bioavailability.[3] Surfactants or detergents are added to ophthalmic solutions for several reasons: they increase the solubility of the drug by making it more hydrophobic, they act as preservatives and are said to enhance penetration of the drug in ocular tissues.[3]

Benzalkonium chloride is a frequently used detergent in ophthalmic solutions. Osmotics are important additives that help match the tonicity of the ophthalmic solution to that of natural tears, 0.9% sodium chloride.[3] If tonicity of the medication is too far off from the natural tears, the patient will experience reflex tearing which washes the drug away, thereby decreasing bioavailability. Ocular discomfort alone may lead the patient to discontinue therapy if they are not willing to endure the irritation.

All of these factors must be considered when dealing with multiple brands of the same medication as they all will influence the bioavailability of the drug. Decreased bioavailability translates into the drug being less effective clinically.

One example of this occurred with the topical ophthalmic suspension prednisolone acetate 1%. In this case, the particle size of generic prednisolone acetate 1% was larger than the name brand (Pred Forte).[4] In ophthalmic suspensions, particle size may be the most important factor of formulation that determines bioavailability of the active ingredient.[4] Larger particles will settle out and cluster together, resulting in reduced bioavailability of the drug.[4] The clinical implications of this would be reduced clinical efficacy of the generic drug with larger particle size.

Concerns have been raised about other products as well, including a generic form of 1% diclofenac sodium ophthalmic solution. In this case, an increase rate of corneal toxicity, ranging from superficial punctate keratopathy to corneal melting, was associated with the introduction of generic diclofenac.[5]

The generic form of diclofenac was different from the name-brand in preservatives and inactive ingredients, and was recalled after cases of toxicity were reported.[5] A study later reported that the generic was recalled on anecdotal evidence and the cases of corneal melt could have been caused by coexistent factors and not simply drug toxicity.[6]

The prescribing dilemma for practitioners choosing from multiple forms of the same drug is considering cost to the patient and clinical effectiveness or bioavailability. Generic non-equivalence is not frequently documented. Regulating agencies that monitor the development of generics work to ensure bioequivalence so the practitioner can comfortably prescribe any form of the drug.

In the case of ophthalmic drugs, cases of generic non-equivalence have been reported but with controversial results in some cases. It is important for the practitioner to understand how generics differ from their name brand counterparts, especially considering the non-active ingredients that influence bioavailability of the drug.

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Herpes simplex keratitis

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Herpes simplex (HSV) is a truly remarkable virus. It is a double-stranded DNA virus and is the most ubiquitous communicable virus in humans. It is also the most common single micro-organism to infect the cornea, affecting about one in 650 people at some point in their lives. Humans are the only known natural reservoir of the virus. The virus enters the body through the skin or a mucous membrane and after resolution of the primary infection, frequently leads to latent infection with the virus resident in the local nerve ganglion cell as well as the nerve axon.

There are two serotypes: HSV-1, which mainly causes infections above the waist, and HSV-2, which mainly causes infections below the waist and is the primary cause of genital herpes. There is some cross-protection provided by immunity to one serotype against the other. The most common site of HSV infection is on the lips with about one-third of the human population having recurrent herpetic labialis/dermatitis (cold sores). A history of cold sores around the lips may be protective against herpes simplex keratitis (HSK) as it may lead to immunity.

HSK is common and relatively complex, with both primary and secondary forms of the disease and multiple manifestations of the secondary disease. HSK is more common in men than women (2:1) and does not demonstrate any known seasonal variation in frequency of attacks.

About one in 10 people with a history of HSK will have a repeat attack in any given year and about one in 10 with HSK develop stromal keratitis, the form most commonly associated with loss of vision. The disease is usually unilateral with only about two per cent of affected people having bilateral disease; ocular atopy appears to be a particular risk factor for the development of bilateral disease.

The rationale and treatment protocols for treating HSK were greatly enhanced by the Herpetic Eye Disease Studies (HEDS),[1,2] a series of prospective multicentre randomised clinical trials performed in the USA in the early 1990s. These looked at a number of treatment issues in HSK including trigger factors, the need for anti-virals and corticosteroids, as well as the use of oral anti-viral agents as prophylaxis.

Primary ocular HSV

The primary HSV ocular disease is often not identified because the episode is frequently mild and similar to other viral forms of conjunctivitis. The primary infection most commonly occurs in infants, who catch the disease from direct contact with family members or friends, or in teenagers.

The primary ocular infection may be so mild as to be asymptomatic or may lead to a slightly red, watery eye with minimal discomfort. Sometimes this is associated with typical cold sore type vesicles along the lid margin (blepharoconjunctivitis). The infection is usually unilateral. Examination reveals a typical follicular conjunctivitis, sometimes with associated small, grouped punctate epithelial erosions, even on occasion microdendrites. It typically resolves fully

Layer involved	Name of disease	Primary cause	Treatment
Epithelium	Dendritic ulcers	Viral replication	Debridement and anti-virals
	Geographic ulcers	Viral replication	Debridement and anti-virals
	Metaherpetic ulcers (also termed an indolent ulcer)	Neurotrophic/Toxicity	Unpreserved lubricants
Stroma	Stromal necrotic	Immune response & low level viral replication	Topical steroids with antiviral prophylaxis
Stroma & endothelium	Disciform (also termed keratouveitis)	Immune response	Topical steroids with antiviral prophylaxis

Table 1. Secondary herpes simplex keratitis

without any scarring or loss of vision.

Treatment of primary HSV conjunctivitis or blepharoconjunctivitis is with topical acyclovir ointment (Zovirax), x5 daily for one week. The patient can be reviewed at one week if the episode has not resolved fully but the condition is typically self-limiting. The risk of the rare complication of HSV encephalitis exists if the patient is immunocompromised, such as with HIV/AIDS. Treatment with oral antiviral agents is indicated, such as famcyclovir (Famvir) 250 mg tds orally for one week or intravenously if the infection is severe.

If primary HSV ocular disease is suspected, it is worth sending off a swab to the local laboratory for an HSV polymerase chain reaction (PCR). This is a very straightforward test with good sensitivity and specificity. The swabs are readily available from local pathology laboratories and store at room temperature in the clinic for a long time. The specimen is taken by gently rubbing the dry swab along the inferior fornix of the affected eye. A positive result may help diagnosis of secondary HSK at a later date.

Patients should be advised of the risk of recurrent infections following the primary disease and asked to represent promptly for review if they develop a red or sore eye. Recurrences can occur at any stage following the primary infection but the initial episode of HSV reactivation typically occurs in the first several years following the primary infection.

Secondary ocular HSV

Secondary HSK refers to ocular infections resulting from reactivation of viral activity, or of an immune reaction to the virus or viral particles. Recurrent viral activity with viral multiplication may occur following a 'trigger factor'.[3] The most common 'trigger factors' appear to be exposure to abnormally high levels of UV and an acute febrile illness such as influenza. High UV exposure commonly occurs in Summer at the beach or in Winter when skiing. Patients with multiple recurrences of HSK should especially be advised to wear protective eyewear, such as wrap-around sunglasses or skiing goggles, when in high light level situations.

Secondary HSK is usually classified on the basis of the part of the cornea that is involved (Table opposite). There are several other rare forms of HSK, which I have left out from this classification for simplicity.

While this is a very useful classification system for helping to make the diagnosis for treatment, it is more useful to classify the disease on the basis of the primary problem (pathophysiology) being one of viral replication or an immune response to viral antigens (Table). After an active viral infection, residual viral proteins remain strewn throughout the cornea. These act as foreign antigens; it is the reaction of the body's immune system to these antigens that causes so-called 'immune based' HSK. It is the same mechanism that causes most of the delayed onset keratitis following herpes zoster ophthalmicus (HZO).

Dendritic (Figure 1) and geographic ulcers are caused by active viral replication; while disciform and stromal necrotic disease is primarily from an immune response. Metaherpetic ulcers (Figures 2A and 2B) are non-healing defects caused by a poor ocular surface usually resulting from a combination of a digested, scarred, insensitive (neurotrophic) cornea coupled with toxicity from topical medication (medicamentosa).

As with all HSK diseases, a dendritic ulcer causes slight to moderate ocular surface discomfort; it does not cause significant pain. A dendritiform ulcer with a history of a very painful eye is probably a healing epithelial defect.

The typical signs of a dendrite include not only its characteristic shape but also the typical staining pattern. As the edges of the lesion are compromised by the growing virus they freely leak fluorescene. If you instil fluorescene and look a minute or two later, you can observe that the fluorescein has leaked under the edges of the lesion, causing a smudge-like stain. Decreased sensation around the area, as seen with gentle stroking of the area with a wisp of cotton from a cotton bud, is an interesting although usually not particularly helpful sign. It is more marked with stromal disease and metaherpetic ulcers.

If there is any doubt about the cause of the lesion, a HSV PCR swab can be taken as described above for primary HSV infections. This is obtained by rolling the swab along the edges of a suspected lesion.

Lesions caused by active viral replication, dendritic and geographic ulcers, are treated with anti-virals, usually with gentle debridement of the edges of the lesion with an anaesthetic-soaked cotton bud and administration of acyclovir ointment (Zovirax) x5 daily for one week. Topical corticosteroids will make these infections worse and can turn a dendrite into a deep geographic ulcer.

Immune-based disease is treated with topical steroids. Depending on the level of inflammation, the initial treatment is either prednisolone sodium acetate (Pred Forte) or flurometholone acetate 1% (Flarex), both usually x4 daily for a few weeks. When a good clinical response is noted the steroids can be tapered slowly. Initially this may entail changing a stronger steroid such as Pred Forte for a weaker one such as Flarex, and later even Flarex for flurometholone (FML). Then the number of drops per day is gradually decreased, usually by one drop a day every month, going down from x4 to x3 a day after one month and continuing the tapering until they are ceased.

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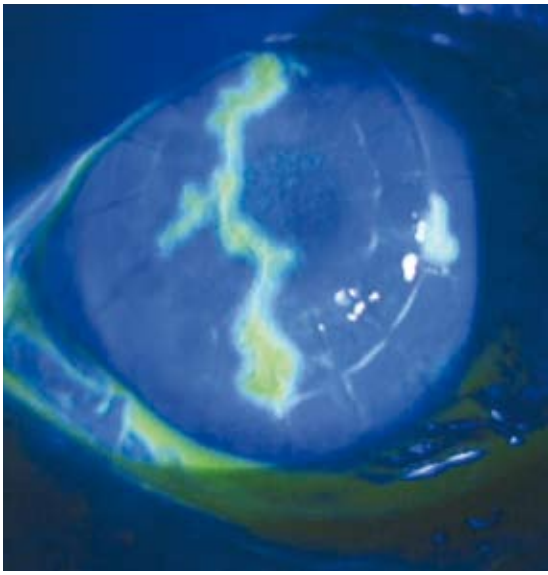

Figure 1. Two dendrites, one across the whole length of the graft and a smaller one on the host near the graft-host junction. The multiple dendrites and extensive nature of the central one is probably secondary to the use of topical steroids for the corneal graft. Although fluorescene was instilled into the eye less than one minute before the photograph was taken, there is already considerable leakage of fluorescene from the ulcer to the surrounding epithelium.

Herpes simplex keratitis

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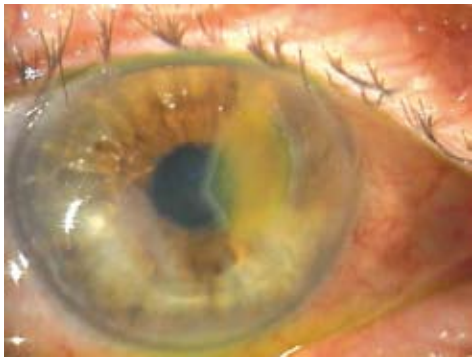

Figure 2A. A large metaherpetic (indolent) ulcer. Note the grey and rounded edge to the ulcer. Although fluorescein was instilled into the eye some time before the photograph was taken, there is no leakage of fluorescene from the ulcer to the surrounding epithelium.

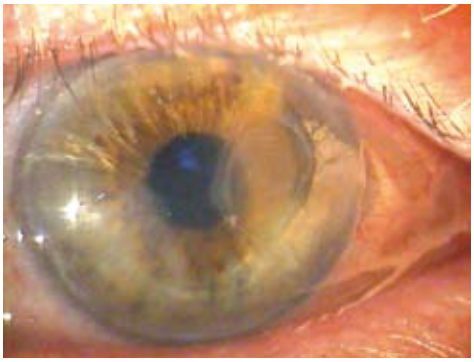

Figure 2B. The same patient as in Figure 2A one month after stopping anti-virals and use of unpreserved lubricants. Note how the epithelium has healed around the whole edge of the ulcer.

As always, monitoring for steroid-related side-effects needs to be carried out regularly. During treatment with topical steroids the patient should be covered with a prophylactic dose of acyclovir ointment, x2 daily. This can usually be stopped when the steroid is decreased to only once a day or less.

One of the outcomes of the HEDS[4] series of trials has been the use of oral acyclovir as prophylaxis to prevent the recurrence of secondary disease. The acyclovir prevention trial (APT) arm of the HEDS found an approximate 50 per cent reduction in recurrences of most forms of HSK when patients took acyclovir tablets, 400 mg orally twice daily. This indication is not covered by the pharmaceutical benefits scheme in Australia and is very expensive for patients, costing about $120 per month, unless it is subsidised and dispensed by a public hospital.

Common guidelines for commencing oral acyclovir are: two or more recurrences of sight-threatening HSK within one year, HSK occurring in an only sighted eye and HSK on a corneal graft. The study found that the acyclovir is effective only while the patient is taking the drug. It does not have any longer-term benefit. For ongoing protection the patient needs to continue on the acyclovir. Practically, patients usually continue the medication for one to two years or longer, especially in an only eye.

Diagnosis and differential diagnoses

The main differential diagnosis for a dendrite is a healing epithelial defect, either from trauma or recurrent corneal erosion syndrome (RCES). Typical findings seen with a dendritic and geographic ulcer and how they differ from other epithelial defects are listed above. *Acanthamoeba keratitis* is an extremely uncommon differential diagnosis.

Stromal disease usually presents with a focal infiltrate with an overlying epithelial defect. There is often a smear of intra-stromal inflammation surrounding the lesion and frequently evidence of previous infections with scars and small divets on the cornea from tissue loss. These are often adjacent to the active lesion. The lesion can mimic other stromal viral infections such as HZO, adenoviral keratitis (which typically has multiple non-ulcerated lesions), infections such as bacterial keratitis and other infectious keratitis such as *Acanthamoeba keratitis* and fungal keratitis.

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Responsibilities of a pharmacist

A step-by-step guide for pharmacists helps to eliminate errors in the dispensing process

Reducing medication risks for patients and ensuring that all legal requirements are met make standardised protocols necessary for pharmacists when they dispense prescriptions.

Pharmaceutical Defence Limited (PDL) has developed a set of guidelines as a resource for all Australian pharmacists. The Guide to Good Dispensing is a step-by-step process designed to reduce the possibility of dispensing errors and to save pharmacists time and money.

Under the guidelines, pharmacists are advised to check the patient's details—their name, contact information, Medicare number and health characteristics—and the details of their prescription, including the practitioner's signature and S4 and S8 requirements.

The pharmacist should then review the patient's medication profile to ensure consistency of treatment, identify possible interactions with other medications and investigate any instances of medication misuse, such as the patient presenting to the pharmacist for prescription repeats more frequently than has been recommended.

After selecting the required drug and its dosage, the pharmacist should label the medication. This process involves checking label information such as the expiry date and the drug and dosage against the original copy of the prescription, and that the label does not obscure important information on the manufacturer's label.

Scanning the label's barcode to bring up the medication details on computer, the pharmacist should again check that these details match those listed on the original prescription.

When finalising the prescription, the pharmacist must ensure that it is accompanied by any necessary repeat forms and consumer medication information, placed in a container with all items visible and kept out of reach of the public.

When the patient comes to collect their prescription, the pharmacist should consider whether they require counselling and verify that they are the correct person receiving it. A final check of the medication details against the original copy of the prescription is recommended.

A spokeswoman for PDL says the guidelines—developed about 10 years ago—are popular with the thousands of pharmacists across Australia. 'The guidelines are printed in our annual report each year and we often get requests for them, especially from hospital pharmacists,' she said.

Pharmaceutical Society of Australia director of professional services, Grant Martin, says that while the process for dispensing scripts remains the same, differences exist when processing private and Pharmaceutical Benefits Scheme (PBS) scripts.

'There is slightly more administration for PBS scripts, with differences in the coding of scripts for payment, and the fact they are then forwarded to the government,' he said.

Martin says that although it is not documented in the PDL guidelines, communication between pharmacists and the practitioner who wrote the script may be necessary. 'The pharmacist may detect interaction with other medications, want to discuss the dosage or frequency of the medication with the prescriber, or simply has problems with the legibility of the script.' ■

Electronic prescriptions on trial

An Australia-wide platform for electronic prescriptions has been launched with the aim of improving dispensing accuracy, reducing pharmacists' workload and informing practitioners about whether a patient's medication has been dispensed.

The eRx Script Exchange system was introduced in April and is being trialled by a small number of general practitioners and medical specialists.

The system, which interfaces with existing prescribing and dispensing software, involves patients receiving a paper prescription from their practitioner imprinted with an eRx Script Exchange barcode. An electronic version is sent in an encrypted format to the system 'hub'.

Patients can present their prescription at any pharmacy in Australia, with the pharmacist scanning the barcode to download the electronic prescription from the hub into their dispensing software.

Graham Cunningham, chairman of eRx Script Exchange, hopes the system will be incorporated into significantly more medical practices across Australia within the next 12 months.

He says that although the system is targeting general practitioners and medical specialists, there is no reason that optometrists cannot access it in the future.

Do we need to know

Tony Gibson
MScOptom

Case reports

The optometrist as a primary provider has a crucial role in the health care team. Patients present seeking solutions for a variety of symptoms. Many signs and symptoms have an underlying aetiology related to both systemic conditions and the medication used to treat them. A complete history should alert the optometrist to the possibility of interactions between systemic and ocular health and can be crucial to the management of patient outcomes.

Often patients do not recognise that systemic medication may affect their eyes so it is important to question patients on the history of their general health and all medications they have taken.

More optometrists are receiving referrals from GPs and medical practitioners should be encouraged to include a history of health issues related to the referred patient as well as a list of medications including dosages.

Potential ocular side-effects of commonly prescribed medications include eye movement and lid control, pupil reactions, accommodative dysfunction, pigmentary changes, tear film stability, ocular surface irritation, intraocular pressure changes, cataract, visual field effects, colour vision effects, and retinal and optic nerve pathology.

Examination of these signs is an essential part of a thorough eye consultation and may be the first occasion that such effects have been discovered. Alerting a patient and their medical practitioner to the presence of such ocular effects may improve treatment options that can avoid potentially serious immediate and long-term ocular morbidity.

At the conclusion of the examination, the optometrist must decide on a likely diagnosis and appropriate treatment for the patient's symptoms and signs. Reference matrices that link systemic diseases and medications with potential ocular complications and adverse reactions are a valuable guide to assist clinical decision making. Two useful references are www.aoa.org/x7346.xml and www.academy.org.uk/pharmacy.

These four typical case studies demonstrate the importance of a good history of systemic medications.

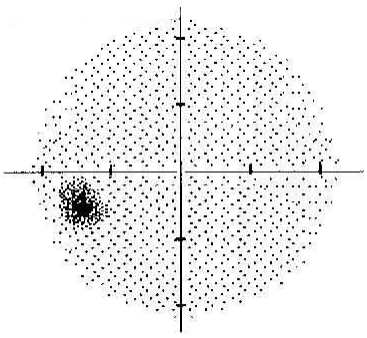


Figure 1. Case 2, LE

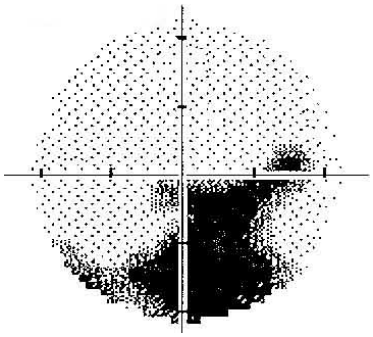


Figure 2. Case 2, RE

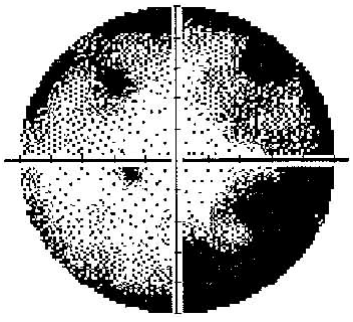


Figure 3. Case 3, LE

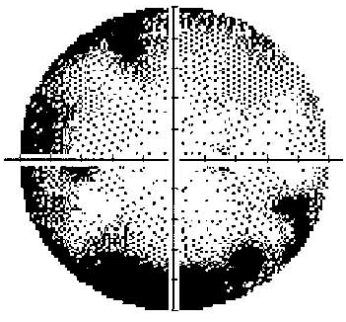


Figure 4. Case 3, RE

IOP steroid response

Prednisolone is a commonly prescribed immunosuppressant steroid for controlling inflammatory responses and is used both systemically and topically. Raised intraocular pressure is a potential ocular side-effect that occurs in seven to 10 per cent of topical users but can also occur in higher dose long-term systemic users.

Case 1. Mr SM

A 74-year-old male with variable intraocular pressure readings over several years underwent bilateral cataract surgery. Both eyes were complicated by IOP spikes immediately post-surgery. The patient recovered well with oral Diamox and temporary Xalatan post-surgery and is a suspected steroid responder.

More recently he was treated with oral Prednisolone to manage a skin rash and his IOP rose from baseline 15-18 mmHg to 24-26 mmHg. After a discussion with his GP, the Prednisolone was withdrawn for a trial period and his IOP returned to base levels. He is strongly suspected to be a steroid responder and may need topical treatment for his IOP if he is again required to take oral steroid in the future.

patients' medications?

Case 2. Mrs AB

A 67-year-old woman was treated intermittently over many years with Prednisolone when her long-term asthma was severe. Her IOP had been monitored for many years and had never been raised. An arcuate field loss and optic nerve match was discovered at a routine examination and she is now comanaged on topical Xalatan. It was considered that she had asymmetrical low tension glaucoma and her target ideal IOP may be lower than in other patients. Her steroid use may have exacerbated her moderate IOP rise and contributed to the asymptomatic onset of her low tension glaucoma. Her field loss has reduced since she has been treated. (Figures 1 and 2)

Acquired retinal pigmentary degeneration with Vigabatrin

GABA (gamma-amino butyric acid) is a neurotransmitter and thought to be at inadequate low levels in epileptic patients. Vigabatrin (Sabril) provides an agonistic GABA effect and is very effective in relieving symptoms but it may accumulate in the retina, where it is thought to destabilise metabolic rates, particularly in rod receptors, and accelerate their death.

The clinical signs that manifest are similar to those of retinitis pigmentosa and may be dose related but appear after months or years of treatment, often without symptoms. These include peripheral pigmentary degeneration, narrowed retinal vessels, optic neuropathy, permanent peripheral visual field loss and reduced night vision. Baseline fields and regular follow-up assessments are recommended before starting therapy.

Case 3. Mr KP

A 44-year-old male with long-term severe epilepsy was managed in a neurology outpatient clinic and treated with Vigabatrin (Sabril). Regular full-field and fundus examinations detected peripheral retinal pigmentary changes and field losses, which have remained stable. He was taken off Vigabatrin and may proceed to temporal lobe surgery. (Figures 3 and 4)

Case 4. Mr OS

An 80-year-old male with long-term epilepsy was treated with Rivotril and Tegretol, and for a four-year period with Vigabatrin. Peripheral pigmentary changes, arteriolar narrowing and a flat waxy optic nerve were noted during a subsequent routine examination. Full-field testing demonstrated a dense peripheral loss corresponding with the retinal pigmentary changes. He subsequently had bilateral cataract surgery and developed some R capsular opacity, which was treated with a capsulotomy.

The fundus images (Figures 5 and 6) were taken before the R capsulotomy and there is some central blur in the R image. The full-field results are shown in Figures 7 and 8. A report to his GP and neurologist alerted them to the problem and the drug was withdrawn.

Vigabatrin has been replaced by anticonvulsant drugs with fewer side-effects. ■

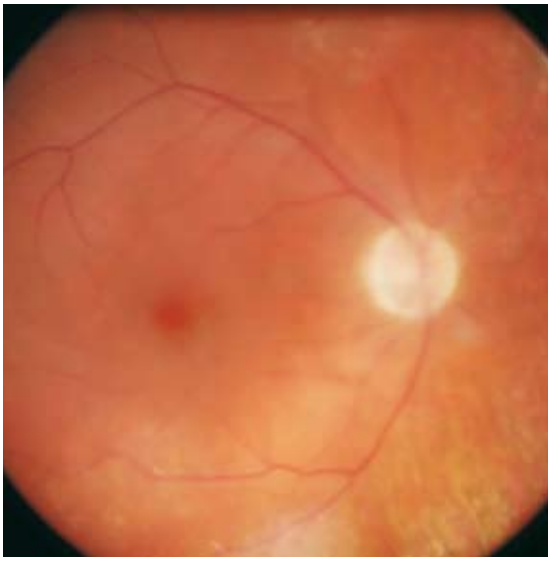


Figure 5. Case 4, RE

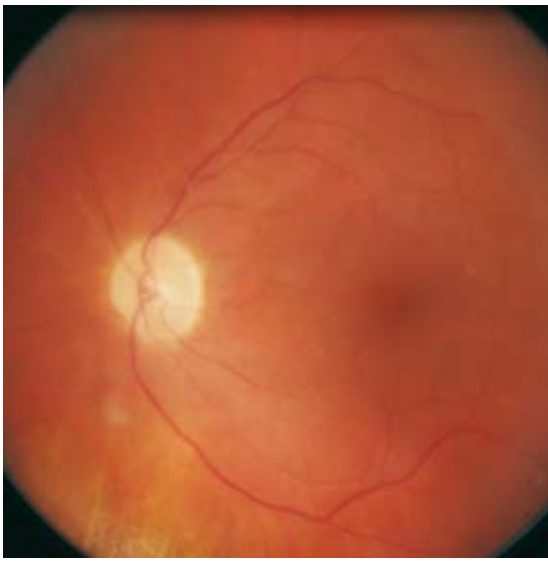


Figure 6. Case 4, LE

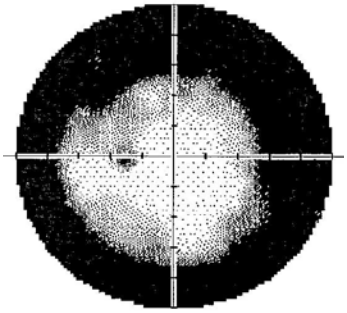


Figure 7. Case 4, LE

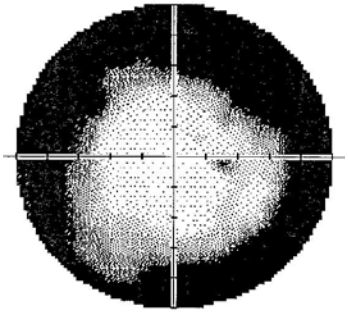


Figure 8. Case 4, RE

Botulinum toxin

can cause drooping eye lids,

Dr Andrew Atkins
MB BS FRACO
Bayside Eye Specialists

Although it was first purified in 1928, botulinum toxin has become popular since the mid-1980s as a cosmetic agent by virtue of its effective muscle relaxant action that diminishes the appearance of facial wrinkles. The disappearance of especially deep wrinkles is dramatic and often cosmetically satisfying. Botulinum toxin is widely used by medical professionals in the fields of ophthalmology, plastic surgery, dermatology and general practice.

As well as established cosmetic uses, medical therapeutic uses include hemifacial spasm and essential blepharospasm. Injection into extraocular muscles can be performed where temporary treatment of strabismus is desired. Deep injections into neck muscles can relieve neck spasm, and in high doses it can be used to treat muscle spasticity in cerebral palsy and head trauma victims.

Hemifacial spasm before botulinum toxin treatment

Relaxed hemifacial spasm after botulinum toxin treatment

There have been some reports of success in using botulinum toxin to treat migraine, tension headaches and the neuralgia that sometimes debilitates individuals after an episode of herpes zoster ophthalmicus.

Initial cosmetic uses were to treat the glabellar and frontal frown lines. The wrinkle lines at the outer angle of the eyelids (crow's feet) can also be effectively treated but great care must be taken to avoid side-effects. Injected into the lower lids, excessive drooping can occur, which is cosmetically undesirable and may lead to watery eyes.

I am often asked about the possible use of Botulinum toxin to treat wrinkles around the mouth. These are generally best treated with 'fillers', which are synthetic products (such as collagen) used to replace diminished tissue volume. Negative side-effects of botulinum toxin injections around the mouth include the inability to smile normally and drooling while eating.

Botulinum toxin is a protein that is synthetically produced from bacteria in a way similar to the production of penicillin.In Australia, it is marketed in two forms, Botox or Dysport. It must be refrigerated prior to use to prevent denaturing of its protein structure. It is reconstituted with saline and each small injection contains a carefully measured number of units. A small number of units are injected at each site. A muscle of relatively larger bulk such as the glabellar muscle requires a higher dosage unit than the smaller facial and eyelid muscles.

The toxin works by blocking the transmission at the neuromuscular junction. It consists of two polypeptide chains (a heavy chain and a light chain) linked by a disulfide bond. The light chain exerts the effect at the neuromuscular junction by attacking the fusion proteins (SNAP-25, syntaxin or stnaptobrevin) preventing vesicles from anchoring to the membrane to release the neurotransmitter acetylcholine. By inhibiting acetylcholine release, the toxin interferes

Injecting a muscle relaxant can diminish the appearance of facial wrinkles by blocking the transmission at the neuromuscular junction, but it may come at a cost.

headache and burning

with nerve impulses and causes flaccid (sagging) paralysis of muscles as opposed to the spastic paralysis seen in tetanus.

The onset of action is two to five days and the individual is unable to contract the affected muscles, preventing the appearance of the undesirable wrinkles. In general, the length of action varies from patient to patient but in my practice it is usually from three to six months. The action is temporary due to the formation of new sites of effective transmission of the neurotransmitter at the neuromuscular junction.

A very fine needle is used to inject the botulinum toxin and a single treatment may involve between four and 20 injections. There is minimal discomfort and no anaesthetic is required. It is not necessary for the patient to be accompanied to the consultation and they are certainly capable of driving themselves after the treatment.

During a patient's first treatment, the nature of botulinum toxin is clearly explained, including the time for onset of action and possible side-effects. Most people who present for this treatment, especially cosmetic patients, are very well educated on the topic prior to my discussion.

Occasionally minor bruising can occur at the site of the injections and this is minimised by applying pressure immediately after injecting. I emphasise that every patient responds slightly differently to the treatment and over time dosage is individually tailored. I have found it helpful to offer a free 'top-up' service after visits to ensure patient satisfaction.

Less common side-effects include headaches, pain, burning, swelling or redness at the injection site. The induced muscle weakness may be excessive for a short time, resulting in eyelid droopiness. This may last for up to three weeks. ■

Example of 'crow's feet' before botulinum toxin treatment

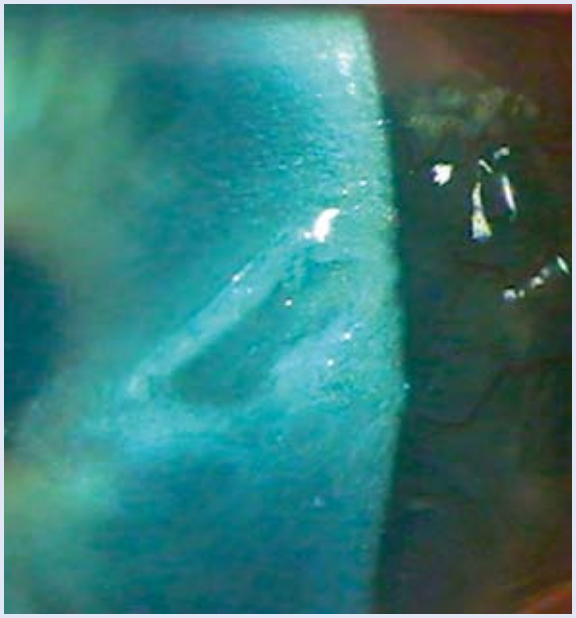
Example of 'crow's feet' after botulinum toxin treatment

Glabellar skin folds before botulinum toxin treatment

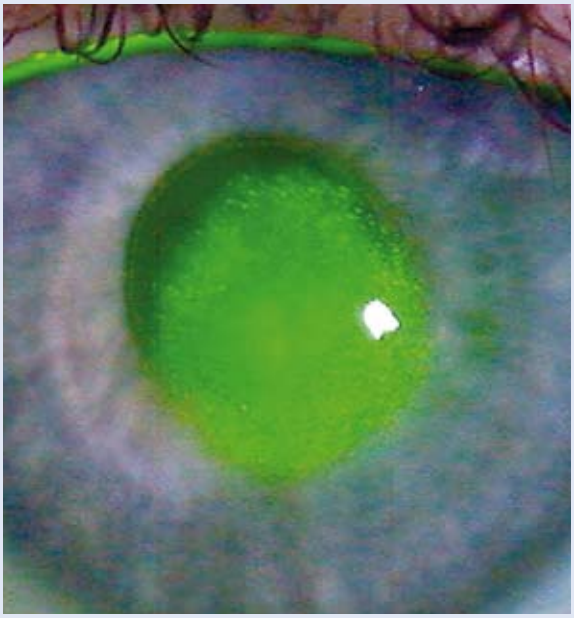
Glabellar skin folds after botulinum toxin treatment

photo clinics

Gary Page
BAppSc(Optom)
DipAppSc(Orth)
GradCert(Ocular Therapeutics)



Persistent neurotrophic ulcer in an elderly patient. Management included prophylactic Tobrex qid and Prednefrin forte qid to minimise tissue destruction caused by the intrinsic inflammatory response.



Epithelial oedema secondary to acute hydrops in a keratoconic patient, stained with NaFl

Drug interaction alerts inadequate

General medical practitioners and pharmacists have good reason to complain about the relevance and accuracy of drug interaction alerts in prescribing and dispensing software, according to a study published in *Medical Journal of Australia*.[1]

The study compared information in alerts with that found in a range of reference sources, and investigated variations between systems, including sensitivities and specificity.

There are many gaps in the information considered necessary for decision-making including clinical effects and management advice. Deficiencies in drug interaction alerts have the potential to impede the quality use of medications in terms of individual patient management.

The relative low specificity rate suggested some software systems contain inappropriate and unhelpful information, such as inappropriate alerts for interactions with topical products and failure to differentiate between drugs in a class. The inundation of irrelevant alerts can desensitise users.

The deficiencies in the quality of alerts may be the fault of publishers as much as the software vendors. The drug interaction alerts incorporated into software systems are usually generated from published reference sources.

A panel of experts urges publishers to ensure provision of accurate, useful, up-to-date information, and for vendors to implement the information appropriately.

The government has identified the need for national standards for decision support in clinical software in its National e-Health Strategy. National Prescribing Service CEO Dr Lynn Weekes said that establishing national standards would make it easier for software vendors to improve the quality of their systems and enable greater consistency between systems.

It is important for users to be aware of the deficiencies and refer to alternative sources.

1. *MJA* 2009; 190: 251-254

As the only therapeutically endorsed optometrist in Maffra, Anna Chan was valued as an employee and a key member of the community, writes **Gary Oshry**

Local community values endorsement

When Anna Chan undertook her therapeutic training course in 2006, she was practising full-time in Maffra, a rural town 220 kilometres east of Melbourne. The Gippsland ranges were not the only obstacle Chan had to overcome.

It took Chan two years to complete the course because of delays in organising the rotations at the Royal Victorian Eye and Ear Hospital. 'Even though we had done the didactic component a year earlier, not being able to do the hospital visits made it difficult for us to get the clinical rotation completed,' she said.

Many residents of Maffra and the surrounding areas have been appreciative of Chan's persistence. At the end of 2007 she became the town's only therapeutically endorsed optometrist, working at John Cronin Optometrist, the only optometric practice in a town of 4,500 residents where access to ophthalmology services is limited.

'It is handy to have a pad in hand; it makes life much easier for my patients. There were numerous instances when I would send the patient to the GP merely to get a prescription. Male patients in particular often consider attending a GP to be too hard and won't bother. Now I can send my patients directly to the pharmacy,' says Chan.

'Being able to treat and monitor uveitis is especially rewarding. Without the endorsement I may have had to send some of these cases to the Eye and Ear Hospital in Melbourne, then without a follow-up you don't really know the result.'

Having a therapeutic endorsement gives Chan greater control to monitor the compliance of her patients. For cases of foreign body removal, which is very common in Maffra, she says that personally handing over the prescription gives her a greater assurance that the patient will comply with her recommendation to use the antibiotic eye-drops needed to prevent the possibility of infection.

Although she worked in a busy practice supported by only two optometrists, Chan says her employer John Cronin was very understanding and accommodating of the demands of the course. 'For private practice visits I could take the morning off and be back in the afternoon,' she says. 'Many of the lectures were over the weekend when the practice was closed, so I only had to take Fridays in leave. For those practising in Melbourne, I imagine it would be harder not to work on Saturdays.

'You can get by as an optometrist without the endorsement but it opens more doors, makes the day far more interesting and offers you the opportunity to manage more complicated cases.'

Chan practised in Maffra for one year after gaining her endorsement and now works in Diamond Creek on Melbourne's north-eastern fringe. ■

> **Male patients in particular often consider attending a GP to be too hard and won't bother. Now I can send my patients directly to the pharmacy.**
>
> Anna Chan

Treat dry eye with sodium

David Ng
BOptom

Sodium hyaluronate is a naturally occurring glycosaminoglycan widely distributed in the skin, connective tissue and synovial fluid. In the eye, sodium hyaluronate is found in the vitreous, the aqueous humour and in the connective tissue of the anterior chamber angle. Because of its viscoelastic properties, sodium hyaluronate has been used in intraocular surgery for more than 20 years. In recent years, eye-drops containing sodium hyaluronate have been available for the treatment of dry eye symptoms (Figure opposite).

> **When the eye blinks, the high shear force causes the sodium hyaluronate molecules to align and spread easily over the ocular surface**

Medical uses

Because sodium hyaluronate is found naturally in many tissues of the body, it has been used extensively in biomedical applications targeting these tissues. The first sodium hyaluronate biomedical product, Healon (Abbott Medical Optics, previously named Advanced Medical Optics, Santa Ana, USA) was developed in the 1970s and used in ophthalmic surgical procedures to maintain deep anterior chamber, which facilitates manipulation inside the eye with reduced trauma to the corneal endothelium and other ocular tissues.

In the late 1980s, sodium hyaluronate was approved for the management of osteoarthritis (for example, Hyalgan, Fidia SpA, Italy). When injected into the knee, sodium hyaluronate acts as a lubricant and shock absorber to relieve pain caused by osteoarthritis. In the 1990s, sodium hyaluronate was first used as an adhesion barrier to reduce internal scarring after open surgery in the abdomen or pelvis (Seprafilm, Genzyme, USA). Together with carboxymethylcellulose, the biopolymer reduces adhesions between the abdominal wall and the underlying viscera, and between the uterus and surrounding structures.

In the past decade, FDA approved sodium hyaluronate injections for filling soft tissue defects (Restylane, Q-Med AB, Sweden; Juvederm, Allergan, USA). It is believed that sodium hyaluronate can help replace the lost hyaluronic acid and smooth wrinkles and folds.

Around the same time, a number of eye-drops containing sodium hyaluronate were commercially available on the market. In 2004, Advanced Medical Optics launched a contact lens rewetter called Blink Contacts, which contains 0.15% sodium hyaluronate for the management of contact lens associated dry eye, and in 2008 launched Blink Intensive Tears containing 0.2% sodium hyaluronate for the relief of dry eye symptoms.

Managing dry eye

The viscoelastic properties of sodium hyaluronate make it particularly useful for dry eye patients. Between blinks, the long chains of the polymer intertwine, providing viscosity that retards evaporation and drainage from the eye. When the eye blinks, the high shear force causes the sodium hyaluronate molecules to align and spread easily over the ocular surface.

Studies demonstrated the subjective and objective improvements of sodium hyaluronate in dry eye patients.[1-5] Early in 1982, Polack and McNiece used a 0.1%

hyaluronate

The first biomedical preparations containing sodium hyaluronate were developed more than 30 years ago. Only in recent years has it been used as a novel treatment for dry eye.

dilution of Healon in patients with severe dry eye syndrome and found that it could effectively relieve symptoms including pain and photophobia.[1] Later, Mengher and colleagues from the Nuffield Laboratory of Ophthalmology, University of Oxford, reported that 0.1% sodium hyaluronate could significantly increase non-invasive tear break-up time of dry eye patients. The symptoms of grittiness and burning were also significantly alleviated.[2]

More recently, Condon and colleagues demonstrated an improvement in Schirmer's score with the application of 0.1% sodium hyaluronate in patients with keratoconjunctivitis sicca and Sjögren's syndrome,[3] and Prabhasawat and co-workers showed a significant improvement in non-invasive tear break-up time with 0.18% sodium hyaluronate compared to hydroxypropyl-methycellulose in patients with tear dysfunction due to oil defect.[5]

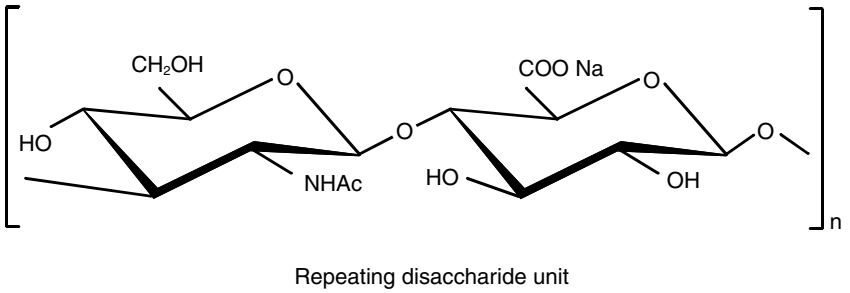
It has been suggested that the effect of sodium hyaluronate on eyes can last for more than 60 minutes.[1,2,5,6] Polack and McNiece evaluated the presence and persistence of sodium hyaluronate on the corneal surface using fluorescein and found that sodium hyaluronate lasted for at least one hour in most patients.[1]

In a study using transmission electron microscopy, Hazlett and Barrett found that sodium hyaluronate was detectable at the corneal surface of mice 60 minutes after its application and appeared to contain electron dense filamentous-like substances, which are associated with corneal surface cell microvilli.[6]

Other studies showed that the relief of symptoms after instillation of sodium hyaluronate lasted from between 60 and 90 minutes.[2,5]

Sodium hyaluronate may also play a role in protecting the corneal epithelium. A number of studies showed that sodium hyaluronate could promote corneal epithelial cell migration and reduce ocular surface damage.[7-9] Clinical observations suggested that sodium hyaluronate may reduce fluorescein and Rose Bengal staining in patients with dry eye.[3,4,10,11]

Sodium hyaluronate may be more effective in treating dry eye symptoms and the introduction of eye-drops containing sodium hyaluronate provides optometrists with a new option apart from traditional tear formulas.



Molecular structure of sodium hyaluronate

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Briefs

Drug education

People can gain a better understanding of their medicines through an online tool developed by the National Prescribing Service (NPS).

The resource, launched on 12 March, is known as the NPS Medicine Name Finder and is available to consumers and health professionals.

Visitors to the site can enter the brand name of a medicine into the online tool's search field. The name of the medicine's active ingredient will appear; entering the active ingredient will bring up the medication's brand name.

Consumers are then prompted to record their medication details in a downloadable medicines list, or print this information to discuss with their health professional.

Updated monthly, the resource contains information about medications listed on the Pharmaceutical Benefits Scheme. Information on over-the-counter and herbal therapies is not included.

The NPS Medicine Name Finder is available at www.nps.org.au/medicine_name_finder.

Eye-drops in cataract trial unsuccessful

***In vivo* trials of a new therapy to prevent the progression of cataract have failed to replicate the success of this treatment in *in vitro* studies.**

The efficacy of calpain inhibitor CAT0059 in preventing the degradation of lens proteins, leading to the development of cataract, was analysed in sheep lenses.

An eye-drop solution and ointment formulation of CAT0059 were used during the trial, and applied *in vivo* daily to the eyes of lambs with cataract, over a 67- and 97-day period, respectively.

The ointment formulation significantly slowed the rate of cataract progression during the first month of treatment but later observations proved its effect unsustainable. The eye-drop treatment showed no ability to slow the rate of cataract progression.

1. Lee H, Morton J, Robertson L et al. Evaluation of a novel calpain inhibitor as a treatment for cataract. *Clin Exp Ophthalmol* 2008; 36: 852-860.

Genetics not a factor in steroid response

A study[1] assessing a possible link between a patient's genetic make-up and their ocular response to steroids has found no conclusive evidence to support this theory.

Researchers were attempting to determine why some patients experienced a spike in intraocular pressure following injections of intravitreal triamcinolone acetonide (IVTA) to treat retinal diseases.

Despite suspecting a genetic determinant for this, no statistically significant correlation was found between several polymorphisms analysed and the level of IOP elevation following IVTA.

Researchers said the exact reason for patients' response to IVTA remained unclear.

One of the authors, Elizabeth Fini, said in a presentation at Hawaiian Eye 2009 that certain polymorphisms identified in the study could lead to new outflow mechanisms and act as drug targets for lowering IOP.

Dr Fini's presentation included information about studies highlighting the capability of genetics to predict a patient's response to IOP-lowering medication.

1. Gerzenstein S, Pletcher M, Cervino A et al. Glucocorticoid receptor polymorphisms and intraocular pressure response to intravitreal triamcinolone acetonide. *Ophthalmic Genetics* 2008; 29: 4: 166-170.

Contact lens drug delivery

Drug delivery and IOP monitoring through contact lenses are the latest developments in glaucoma management to help patients who do not adhere to treatment.

The contact lens uses sustained time-release doses of glaucoma medication, and performs continuous IOP checks and telemetry, according to a report in *Primary Care Optometry News* in January 2009.

The US Food and Drug Administration is trialling the drug delivery system of the lens for its safety and efficiency.

The technology used is The Punctum Plug Delivery System (QLT Inc, Vancouver) and the Triggerfish Continuous IOP Monitoring system (Sensimed, Lausanne, Switzerland).

QLT conducted its own clinical trial of the plug with 61 people who received different dosages of latanoprost over 12 weeks. Mean IOP reduction was 20 per cent among patients who completed the study and across the three dosage groups (3.5 µg, 14 µg and 21 µg).

Further studies from the company aim to explore the effectiveness of higher doses of latanoprost, a drug which is also the active ingredient of a new product claiming to enhance eyelash growth.

Investigators of the study said that the plug's retention rate needed to improve to keep it in place more than 90 per cent of the time to make it more effective.

Ocular nutrition series launched

***American journal Review of Optometry* has launched a year-long series of articles focusing on the impact of vitamins and nutrition on ocular health.**

Beginning in February 2009 and entitled 'Ocular Nutrition from A to Z', the series provides information to raise awareness and understanding of nutrition relating to eye health.

A website has been launched with links to articles published within the series, related literature on ocular health and nutrition, and information on research studies.

The website is available at http://www.ocularnutritionatoz.com.

Practitioners can receive monthly emails advising them of the latest information about vitamins, supplements and nutrition.

The series is supported by Bausch and Lomb.

Taking on the French resistance

A multimillion euro public health campaign in France has yielded a significant decline in the prescribing of oral antibiotic drugs

Nicole Leong
BScOptom PGCertOculTher FVCO

Recent articles in the French media have reported that the consumption of oral antibiotic drugs in France has declined markedly. Authorities attribute this decline to the launch by the Ministry of Health in 2002 of a public awareness campaign regarding the prudent use of antibiotics.

Prior to 2002, France consumed the most antibiotics and had one of the highest rates of bacterial resistance in Europe. Before 2002, 37 per cent of French patients with a throat ailment requested a prescription for antibiotics, compared to the current figure of 23 per cent. Among French parents of sick children, 45 per cent asked for antibiotics for their child before the campaign but now 25 per cent request a prescription for antibiotics.

These recent reports suggest that a further outcome from the campaign has been the drop in bacterial resistance to antibiotics. The percentage of *Pneumococcus* resistant to penicillin was 47 per cent in 2001, compared to 34.5 per cent in 2005; and in hospitals, golden staph resistant to methicillin has dropped from 33.4 per cent in 2001 to 26.7 per cent in 2006.

The public health campaign, which is estimated to have cost 500 million euros (about AU$1 billion), focused on three specific areas.

1. To increase public awareness with the catchphrase 'Les antibiotiques, c'est pas automatique' (Antibiotics, they're not automatic). The broadcast of television advertisements and radio messages are aimed to 'déconditionné' (decondition) the public of its beliefs that minor ailments, which are often viral in nature, require a prescription for antibiotics.

2. To provide doctors with easy access to rapid diagnostic tests for throat ailments, which determine a viral or bacterial origin. This allows doctors to explain to patients why they did not need antibiotics if their illness was viral in nature.

3. To target those doctors who were heavy prescribers of antibiotics and educate them on the appropriate use of these drugs. This was achieved by the organisation of 'grand rounds' type seminars at which doctors could discuss clinical cases and compare methods of practice; clinical workshops to train doctors in the use of the diagnostic tests and mail-outs to 60,000 general practitioners. The Ministry of Health also co-ordinated visits to each doctor's practice to remind them of the recommendations of the national plan.

Other European countries have also implemented similar public awareness campaigns. The United Kingdom conducted two Winter public awareness campaigns in 2004 and 2005, and reduced its antibiotic consumption by 5.8 per cent. ■

Letter from New Zealand

The New Zealand Government introduced legislation to allow a new group of designated prescribers but it had in mind nurses, not optometrists. **LESLEY FREDERIKSON**, national director of the New Zealand Association of Optometrists, recalls that optometry threw a spanner in the works when it too called for the right to prescribe therapeutic pharmaceutical agents.

When the New Zealand Government put forward a Medicines Amendment Bill in December 1998 to establish a class of 'designated prescriber' that previously did not exist, it seemed like a golden opportunity for the NZAO. The bill lumbered its way through all the usual legislative steps and was passed into law the following year.

We sought further information from the Ministry of Health only to have our hopes dashed. There were no 'applicable requirements' relating to competency, qualifications or training specified in or imposed under the regulations. In fact, no-one had even considered that the change would affect anyone other than nurses.

Undeterred, we developed an application for optometrist prescribing authority and submitted it to the Minister of Health in May 2000. A couple of months later, it was sent back to us with the advice that it could not be accepted as a formal application until we had consulted widely on the proposal.

Armed with a list of 40 organisations that the Ministry of Health had suggested it would be important to consult, we published a discussion document and sent it out with a request for comment in August 2000.

> **Since the development of the Auckland undergraduate therapeutic course for optometry, the research and clinical interests of the departments of optometry and ophthalmology at University of Auckland have become more closely aligned.**

There were some interesting replies but mainly the responses were positive and this gave us a good indication that other groups could see the advantages of optometrists being able to treat the conditions that they diagnosed.

The New Zealand Medical Association noted that it was opposed to the extension of independent prescribing rights to any additional categories of health professional other than medical practitioners. This was bracketed with the curious statement that the NZMA believed that effective, efficient and safe prescribing could exist only when the health professional had the education, training and experience to diagnose and develop comprehensive care programs that were inclusive of prescribing.

As it was the first time that optometry had undertaken this process in New Zealand, we decided to append all the consultation responses in their entirety as an appendix to our application for independent prescribing rights. When the application was resubmitted in February 2002, we addressed some of the issues that were raised as part of the consultation process but in substance the application was pretty much the same as version 1.

This was when optometry hit the first major obstacle in its bid to access prescribing rights.

The application was made jointly in the names of the NZAO and the Department of Optometry and Vision Science, University of Auckland. The Terms of Reference for the New Prescribers Advisory Committee stated one of the objectives of the committee was to establish generic criteria that any health professional group must meet in preparing an application for prescribing rights. However, the committee returned the application, stating that it would accept applications only from the registration bodies

for health professions and not the professions themselves.

In May 2002, we were permitted to submit an application for optometrist independent prescribing rights from the NZAO and the Department of Optometry and Vision Science, with the support of the Optometrists and Dispensing Opticians Board.

We waited nervously until the New Prescribers Advisory Committee released its findings in August 2002. The committee recommended that the Minister of Health:

- Agree that optometrists be granted limited independent prescribing authority and that they have access to the open Pharmaceutical Schedule (PHARMAC)[1] but be limited to those medicines relevant to a defined scope of practice
- Agree that the applicant undertake additional clarification of the scope of practice for optometrists regarding who can prescribe where and for whom
- Agree that the Opticians Board further develop the register of optometrists with prescribing authority
- Agree that the applicants work with experts to further progress the indicative list of medicines
- Agree that the applicants develop clinical guidelines to ensure that prescribing in an open environment has adequate clinical safety mechanisms.

The Minister of Health at the time, Annette King, wrote to the NZAO in November 2002 with the news that she had agreed to extend prescribing rights to optometrists for the therapeutic medicines. These were limited to topical ocular anti-infective preparations, topical anti-inflammatory preparations, other anti-inflammatory preparations including non-steroidal anti-inflammatory drugs, cycloplegics, preparations for tear deficiency, mydriatics and other eye preparations.

The minister deferred prescribing authority for glaucoma preparations until further investigation of the pharmacology courses was undertaken as the New Prescribers Advisory Committee had identified concerns about the ability of optometrists to identify contraindications for use of certain glaucoma treatments based on other pre-existing medical conditions.

Despite the inclusion of comprehensive education relating to diagnosis, management and treatment of glaucoma in both the undergraduate and postgraduate therapeutics course, optometrists in New Zealand were not given independent prescribing rights for glaucoma medication but they were granted access to all other topical ophthalmic medicines, including steroids.

The NZAO concluded that there was more to the glaucoma issue than just concern about patient harm. In any event in October 2004, following graduation of the first cohort of students from the postgraduate therapeutics program at the University of Auckland, the Minister of Health displayed great satisfaction in presenting certificates to the first optometric prescribers.

New Zealand Minister of Health Annette King (centre) presented certificates to the first optometric therapeutic graduates in 2004

The Auckland program has been instrumental in developing good working and teaching relations between optometry and ophthalmology in New Zealand with students having clinical placements in public hospitals and in private ophthalmology clinics.

The program now includes education on emergency care and patient revival skills; Professor Charles McGhee of the Ophthalmology Department, University of Auckland, and his ophthalmology colleagues contribute to the clinical teaching and the examinations of therapeutic competence.

Since the development of the Auckland undergraduate therapeutic course for optometry, the research and clinical interests of the departments of optometry and ophthalmology at University of Auckland have become more closely aligned. More recently we have seen the development of the New Zealand National Eye Centre involving ophthalmology and optometry as the two principal partners.

In the end PHARMAC agreed to review the methods of patient access to subsidies to base them on patient and clinical attributes rather than on prescriber status. The section on eye preparations was completed first and from 1 October 2007 all prescribers were treated equally in terms of patient access to subsidised medicines.

The number of therapeutically endorsed optometrists continues to rise and by June 2008 there were 176 optometrist prescribers spread around New Zealand. This represents 37 per cent of the estimated full-time equivalent optometric workforce; a proportion that is increasing with each crop of new graduates.

The University of Auckland graduated 52 new optometrists from the class of 2008 which pushed the number of therapeutically endorsed optometrists to more than 200 for 2009. The postgraduate therapeutics program is now into its fifth course and among established practitioners demand remains high for places.

1. The New Zealand Pharmaceutical Schedule is equivalent to the Pharmaceutical Benefit Scheme of Australia

Reality check for schizophrenic sufferers

Schizophrenia medication clozapine may be associated with ocular pigmentation, according to a case report published in the *Medical Journal of Australia*.[1]

A 55-year-old woman who had been prescribed long-term, high dose clozapine and medications for other conditions, presented to a hospital eye clinic with progressively worsening vision.

Examination revealed she was suffering several ocular complications, including corneal and retinal pigmentation, an epiretinal membrane, macular atrophy and cataracts. She also demonstrated reduced cone function.

A diagnosis of presumed clozapine-related ocular pigmentation was made. The authors of the report said that this was the first instance that they knew of pigmentation associated with clozapine.

They said that ocular pigmentation was a well-documented side-effect of schizophrenia medication chlorpromazine, with clozapine recommended as a substitute for patients experiencing pigmentation from long-term chlorpromazine use.

Following the diagnosis the patient's medication dosage was reduced but she showed no improvement in vision and no change in the level of pigmentation during an examination six months later.

The authors said that none of the patient's other medications was known to cause ocular pigmentation.

'Our patient had significant irreversible loss of vision ... These changes should be considered as possible side-effects of clozapine, particularly if it is given in high doses,' they said.

'If further similar cases become evident, patients on long-term clozapine therapy should be considered for regular ophthalmological review.'

1. Borovik A, Bosch M, Watson S. Ocular pigmentation associated with clozapine. *MJA* 2009; 190: 4: 210-211

Discuss glaucoma around the family table

Melbourne glaucoma specialist Mark Walland says glaucoma could be more effectively identified if all Australians over 40 years of age had eye examinations and patients revealed their diagnosis to first-degree relatives.

Speaking to *Optometry Pharma*, Walland said that although they were not obligated, it was desirable for family members to be more open with relatives about their glaucoma diagnosis, given the disease's genetic or partially inherited characteristics.

Walland makes these points in an editorial in the *Medical Journal of Australia* in March 2008, in which he writes how it is beneficial for optometrists to be involved in the screening process.

'Optometrists are technologically equipped and trained to detect and diagnose eye disease, are frequently primary eye-care practitioners, and are numerous,' Walland writes. 'The recent move by a vanguard of optometrists into therapeutics and comanagement remains politically vexed. Nonetheless, medical practitioners must accept and encourage the major contribution to glaucoma detection that optometrists make.'

Walland describes measuring only intraocular pressure to diagnose glaucoma as 'an inadequate standard of care', and says that while elevated IOP is frequently associated with glaucoma, it is not the sole indicator of the disease.

He says that there are other factors involved in the diagnosis of glaucoma that should not be overlooked by eye health care providers, and that with glaucoma being the commonest preventable cause of blindness in Australia, asking for a family history, assessing the optic disc and referring patients with positive findings represent opportunities to diminish its impact.

Walland refers to the Baltimore Eye Survey as an example of when IOP measurements can be misleading or even irrelevant when diagnosing glaucoma.

This study found that 50 per cent of newly-diagnosed patients had a 'normal' IOP on a single tonometry measurement, and that in one-third to one-half of open-angle glaucoma cases, the patient's IOP was considered to be in the normal range.

In addition to these cases that the authors described as 'normal pressure glaucoma', Walland points out that patients with ocular hypertension are another example of when certain IOP measurements and the presence of glaucoma do not correlate.

He does not dismiss IOP measurements as being unnecessary when screening for glaucoma; he says that IOP remains 'the single biggest risk factor for glaucoma, and its reduction is the cornerstone of all current treatment'.

PBS List of Medicines for Optometrists 14 May 2009

Antiglaucoma preparations

Antiglaucoma preparation	Product	Max qty	Repeats
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 mg/mL, 3 mL	Lumigan	1	5
Brimonidine eye drops containing brimonidine tartrate 2 mg/mL, 5 mL	Alphagan Enidin	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
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	1	5
Pilocarpine eye drops containing pilocarpine hydrochloride 10 mg/mL, 15 mL	Isopto Carpine Pilopt PV Carpine	1	5
Pilocarpine eye drops containing pilocarpine hydrochloride 20 mg/mL, 15 mL	Isopto Carpine Pilopt PV Carpine	1	5
Pilocarpine eye drops containing pilocarpine hydrochloride 40 mg/mL, 15 mL	Isopto Carpine Pilopt PV Carpine	1	5
Pilocarpine eye drops containing pilocarpine hydrochloride 60 mg/mL, 15 mL	Pilopt PV 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

PBS List of Medicines for Optometrists 14 May 2009

	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		1	2
	Chloromycetin		1	2
Chloramphenicol eye ointment 10 mg/g (1%), 4 g	Chlorsig		1	0
	Chloromycetin		1	0
Sulfacetamide Sodium eye drops 100 mg per mL (10%), 15 mL	Bleph-10		1	2
Anti-inflammatory agents		Unrestricted		
Fluorometholone eye-drops 1mg/mL (0.1%), 5mL	Flucon		1	0
	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	Ocufen		1	0
Hydrocortisone Acetate eye ointment 5 mg/g (0.5%), 5 g	Hycor		1	0
Hydrocortisone Acetate eye ointment 10 mg/g (1%), 5 g	Hycor		1	0
Anti-allergy agents		Restricted:		
Sodium cromoglycate eye-drops 20 mg/mL (2%), 10 mL	Cromolux	Vernal kerato-conjunctivitis	1	5
	Opticrom		1	5
Topical ocular lubricants		Restricted:		
Carbomer 980 ocular lubricating gel 2 mg/g (0.2%), 10 g	Geltears	Severe dry eye inc Sjogren's synd	1	5
	PAA		1	5
	Viscotears Liquid Gel		1	5
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	Isopto Tears		1	5
	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
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL	PVA Tears		1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL	PVA Forte		1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL	Liquifilm Tears		1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL	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 unit dose ocular lubricants		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 980 eye-drops 2 mg per (0.2%) , single dose units 0.6 mL, 30	Viscotears		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
Hypromellose with Dextran eye-drops 3-1 mg/mL (0.3-0.1%), single 0.4 mL, 28	Bion Tears		3	5
Tamarindus indica seed polysaccharide eye-drops 10 mg/mL, 0.5 mL, 20	Visine Professional		3	5
Polyethylene glycol 400 with Propylene glycol drops 4 mg-3 mg/mL (0.4-0.3%); single dose units 0.7 mL, 28	Systane		3	5
Topical ocular lubricant ointments		Unrestricted		
Paraffin compound eye ointment 3.5 g	Polyvisc		2	5
Paraffin pack containing 2 tubes compound eye ointment 3.5 g	Polyvisc (2 pack)		1	5
Paraffin compound eye ointment 3.5 g	Duratears		2	5
Paraffin pack containing 2 tubes compound eye ointment 3.5 g	Ircal (2 pack)		1	5
	Lacri-Lube (2 pack)		1	5

Controlled substances that may be used or prescribed by optometrists

Ocular Medicine	Vic	Tas	Qld	NSW & ACT	NT	SA	WA*	PBS Optometry	PBS Listed
Anti-infectives									
Chloramphenicol	✓	✓	✓	✓	✓	✓	–	✓	✓
Ciprofloxacin	✓	✓	✓	–	✓	✓	–	–	✓
Framycetin	✓	✓	✓	✓	✓	✓	–	◆	✓
Gentamicin sulfate	✓	✓	✓	–	✓	✓	–	–	✓
Ofloxacin	✓	✓	✓	–	✓	✓	–	–	✓
Sulfacetamide	✓	✓	✓	✓	✓	✓	✓	✓	✓
Tetracycline	✓	✓	✓	✓	✓	✓	–	N/L	N/L
Tobramycin	✓	✓	✓	–	✓	✓	–	–	✓
Aciclovir	✓	✓	✓	–	✓	✓	–	✓	✓
Anti-inflammatories									
Dexamethasone	✓	✓	◆	–	✓	✓	–	–	✓
Fluorometholone	✓	✓	✓	✓	✓	✓	–	✓	✓
Fluorometholone acetate	✓	✓	✓	✓	✓	✓	–	✓	✓
Hydrocortisone	✓	✓	✓	✓	✓	✓	–	✓	✓
Prednisolone	✓	✓	◆	–	✓	✓	–	–	✓
Diclofenac	✓	✓	✓	✓	✓	✓	–	N/L	N/L
Flurbiprofen	✓	✓	✓	✓	✓	✓	–	✓	✓
Ketorolac	✓	✓	✓	✓	✓	✓	–	N/L	N/L
Decongestants & anti-allergics									
Ketotifen	✓	✓	✓	✓	✓	✓	✓	N/L	N/L
Levocabastine	✓	✓	✓	✓	✓	✓	✓	N/L	N/L
Lodoxamide	✓	✓	✓	✓	✓	✓	✓	N/L	N/L
Olopatadine	✓	✓	✓	✓	✓	✓	–	N/L	N/L
Sodium cromoglycate	✓	✓	✓	✓	✓	✓	✓	✓	✓
Anti-glaucoma preparations									
Apraclonidine	✓	✓	◆	✓	✓	✓	–	–	✓
Betaxolol	✓	✓	◆	✓	✓	✓	–	✓	✓
Bimatoprost	✓	✓	◆	✓	✓	✓	–	✓	✓
Brimonidine	✓	✓	◆	✓	✓	✓	–	✓	✓
Brinzolamide	✓	✓	◆	✓	✓	✓	–	✓	✓
Dorzolamide	✓	✓	◆	✓	✓	✓	–	✓	✓
Latanoprost	✓	✓	◆	✓	✓	✓	–	✓	✓
Pilocarpine	✓	✓	◆	✓	✓	✓	–	✓	✓
Timolol	✓	✓	◆	✓	✓	✓	–	✓	✓
Travoprost	✓	✓	◆	✓	✓	✓	–	✓	✓
Timolol+Bimatoprost	✓	✓	◆	✓	✓	✓	–	◆	N/L
Timolol+Brimonidine	✓	✓	◆	✓	✓	✓	–	✓	✓
Timolol+Dorzolamide	✓	✓	◆	✓	✓	✓	–	✓	✓
Timolol+Latanoprost	✓	✓	◆	✓	✓	✓	–	✓	✓
Timolol+Travoprost	✓	✓	◆	✓	✓	✓	–	✓	✓
Mydriatics & cycloplegics									
Atropine	✓	✓	✓	✓	✓	✓	–	–	✓
Cyclopentolate	✓	D	✓	✓	✓	✓	D	N/L	N/L
Homatropine	✓	D	✓	✓	✓	✓	–	–	✓
Pilocarpine	✓	✓	✓	–	✓	✓	–	–	✓
Phenylephrine	✓	✓	✓	✓	✓	✓	–	N/L	N/L
Tropicamide	✓	D	✓	✓	✓	✓	D	N/L	N/L
Local anaesthetics									
Amethocaine	✓	D	✓	✓	✓	✓	–	N/L	N/L
Lignocaine	✓	D	–	–	✓	✓	–	N/L	N/L
Oxybuprocaine	✓	D	✓	✓	✓	✓	D	N/L	N/L
Proxymetacaine	✓	D	✓	✓	✓	✓	D	N/L	N/L

◆ The use of these medicines by optometrists is currently being considered
* Optometrists in Western Australia do not have access to the PBS
D Diagnostic use only
N/L Substance is not listed under the PBS

FORESIGHT

[the ability to see into the future and be alert to the signs ahead]

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RANIBIZUMAB
Improving vision. Restoring hope.[2,3,5]

PBS Dispensed Price: $1975.93. Please refer to the Product Information before prescribing. Product Information is available from Novartis Pharmaceuticals Australia Pty Limited or visit www.novartis.com.au. For further information please contact Medical Information & Communication on 1800 671 203. **Indication:** Treatment of neovascular (wet) age-related macular degeneration (AMD). 0.5 mg or 0.3 mg is recommended to be administered by intravitreal injection once a month. **Dosage and administration:** Recommended dose is 0.5 mg (0.05 mL) or 0.3 mg (0.03 mL) given monthly. Interval between doses should not be shorter than 1 month. Treatment might be reduced to one injection every 3 months after the first three injections but, compared to continued monthly doses, dosing every 3 months may lead to an approximate 5-letter (1-line) loss of visual acuity benefit, on average, over the following 9 months. Patients should be evaluated regularly. Must be administered by a qualified ophthalmologist using aseptic techniques. Broad-spectrum topical microbicide and anaesthetic should be administered prior to injection. Patient should self-administer antimicrobial drops four times daily for 3 days before and after each injection. Not recommended in children and adolescents. **Contraindications:** Hypersensitivity to product components, active or suspected ocular or periocular infections, active intraocular inflammation. **Precautions:** Intravitreal injections have been associated with endophthalmitis, intraocular inflammation, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. Proper aseptic injection techniques must be used. Monitor patients during the week following injection to permit early treatment if an infection occurs. Intraocular pressure and perfusion of the optic nerve head must be monitored and managed appropriately. Safety and efficacy of administration to both eyes concurrently have not been studied. There is a *potential* risk of arterial thromboembolic events following intravitreal use of VEGF inhibitors. *A numerically higher stroke rate was observed in patients treated with ranibizumab 0.5mg compared to ranibizumab 0.3mg or control, however, the differences were not statistically significant. Patients with known risk factors for stroke, including history of prior stroke or transient ischaemic attack, should be carefully evaluated by their physicians as to whether Lucentis treatment is appropriate and the benefit outweighs the potential risk.* As with all therapeutic proteins, there is a potential for immunogenicity with Lucentis. No formal interaction studies have been performed. Should not be used during pregnancy unless clearly needed; use of effective contraception recommended for women of childbearing potential; breastfeeding not recommended. Patients who experience temporary visual disturbances following treatment must not drive or use machines until these subside. **Side effects:** Very common: Conjunctival haemorrhage, eye pain, vitreous floaters, retinal haemorrhage, *intraocular pressure increased,* vitreous detachment, intraocular inflammation, eye irritation, cataract, foreign body sensation in eyes, lacrimation increased, visual disturbance, blepharitis, subretinal fibrosis, ocular hyperaemia, visual acuity blurred/decreased, dry eye, vitritis, eye pruritis, *nasopharyngitis,* headache, *arthralgia.* Common: Ocular discomfort, eyelid oedema, *eyelid pain,* conjunctival hyperaemia, posterior capsule opacification, punctate keratitis, corneal abrasion, anterior chamber flare, *injection site haemorrhage,* eye haemorrhage, retinal exudates, injection site reactions, conjunctivitis, *conjunctivitis allergic, eye discharge, photopsia,* photophobia, maculopathy, detachment of the retinal pigment epithelium retinal degeneration, retinal disorder, retinal detachment, retinal tear, *retinal pigment epithelium tear,* vitreous haemorrhage, vitreous disorder, uveitis, iritis, iridocyclitis, *cataract subcapsular, influenza, anaemia, anxiety, stroke, cough, nausea, allergic reactions (rash, urticaria, pruritis, erythema).* Uncommon: Keratopathy, *iris adhesions, corneal deposits,* dellen, corneal striae, *injection site pain, injection site irritation, abnormal sensation in eye, hyphema,* cataract nuclear, angle closure glaucoma, endophthalmitis, eyelid irritation, blindness, corneal oedema, hypopyon. Rare but serious adverse reactions related to intravitreal injections include endophthalmitis, rhegmatogenous retinal detachment, retinal tear and iatrogenic traumatic cataract. **Please note changes to Product Information in italics.* **1.** Bressler NM. J Am Board Fam Pract 2002;15:142-152. **2.** Rosenfeld PJ, et al. N Engl J Med. 2006;355:1419-1431. **3.** Brown DM, et al. N Engl J Med. 2006;355:1432-1444. **4.** LUCENTIS Approved Product Information. **5.** Chang TS, et al. Arch Ophthalmol. 2007;125:1460-1469. **6.** Attebo K, et al. Ophthalmol. 1996, 103: 357-364. Novartis Pharmaceuticals Australia Pty Limited, ABN 18 004 244 160. 54 Waterloo Road, North Ryde NSW 2113. ® Novartis Pharmaceuticals Australia Pty Limited. NVO_LUC65_11/2008. Bluedesk LUC3C.

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