

# Medical co-management of glaucoma

Greater emphasis is being put on suitably trained optometrists to play a greater role in managing glaucoma. **Aachal Kotecha** and **Alexander Spratt** introduce the topical medication commonly prescribed to patients. **Module C7948**, two CET points suitable for optometrists, additional supply optometrists and supplementary prescribers

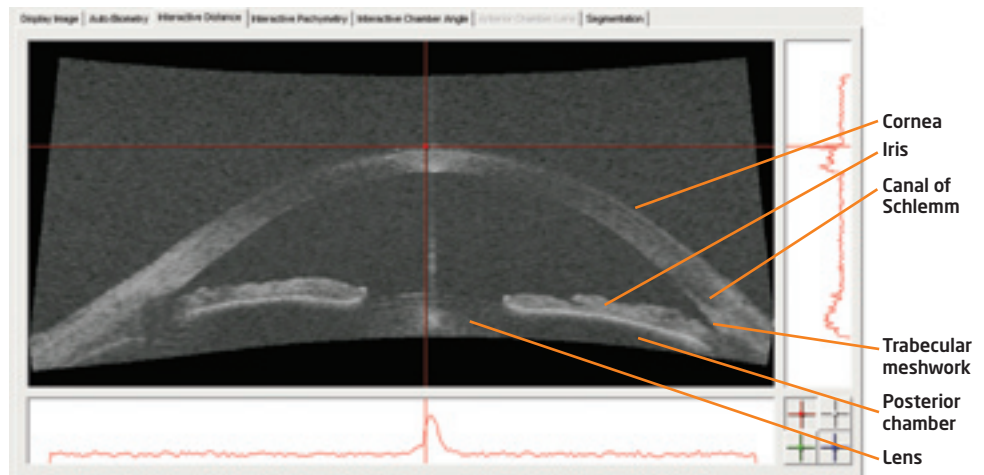
It is estimated that within our ageing population, the number of glaucoma sufferers will increase by a third over the next 20 years.<sup>1</sup> Coping with this extra demand would require a significant expansion of hospital eye department services, and concerns about financial and staffing issues within the NHS have sparked a push for non-medical personnel to become involved in the clinical care of glaucoma patients. In the UK, undergraduate optometric training already provides the basic skills required for the detection of glaucoma.<sup>2</sup>

The Department of Health and the National Eye Care Steering Group have identified glaucoma as one of the four pathways for greater involvement of optometrists in providing primary care co-management.<sup>3</sup> In recent times, optometrist-led glaucoma clinics have developed within hospital eye departments as have shared-care schemes between community optometrists and local eye departments.<sup>4,5</sup> To participate in such schemes optometrists undergo extensive further training and examination by supervisory consultant ophthalmologists to prove their clinical competency in making safe decisions about which glaucoma patients are stable and which merit re-referral back to hospital care.

The Department of Health has plans to extend further the role of suitably trained optometrists to include the right to independently prescribe a small range of eye drops and, as part of a shared-care scheme, to prescribe a larger range of topical medications, possibly including those used to treat glaucoma. The aim of this article is to introduce the topical medication commonly prescribed in the management of glaucoma.

## Glaucoma

Glaucoma is the leading cause of irreversible blindness in the developed world.<sup>6</sup> Although the vast majority of patients with glaucoma do not go blind, many lose useful vision and it



**Figure 1**  
Anterior eye  
anatomy  
(OCT image)

accounts for an estimated 12 per cent of all cases of blind registration in the UK. The majority of glaucoma cases are the result of primary open-angle glaucoma (POAG); its prevalence in Caucasian populations is estimated at between 1.1 per cent and 2.2 per cent of the adult population, increasing with advancing age.

Glaucoma can be defined as a progressive optic neuropathy showing characteristic morphological changes of the optic nerve head and retinal nerve fibre layer in the absence of other ocular disease and congenital abnormalities.<sup>7</sup> A major risk factor for the development and progression of POAG is raised intraocular pressure (IOP) and reduction of IOP remains the mainstay of glaucoma treatment. Recent studies have shown that treated glaucoma patients with a 'target IOP' set in the low teens have the lowest rate of progression. This IOP reduction is achieved through either medical or surgical means.

## Aqueous humour dynamics

To understand the fundamentals of the medical management of POAG, it is necessary to be familiar with the dynamics of aqueous production and outflow. The role of aqueous humour is to bathe the crystalline lens and corneal

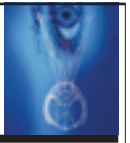
endothelium in a nutrient medium, to remove the by-products of metabolism and to maintain a level of IOP that keeps the eye inflated.

## Aqueous production

Aqueous humour is produced by the ciliary body, the ring of tissue extending from the *ora serrata* posteriorly to the iris root anteriorly. The ciliary body is composed of loosely arranged collagen fibres, blood vessels and nerves, interwoven with the predominating smooth muscle, the ciliary muscle. Anatomically there are two distinct areas of the ciliary body: the anterior *pars plicata* and the posterior *pars plana*. The anterior *pars plicata* comprises approximately 70 radially arranged major ciliary processes which project into the posterior chamber. These ciliary processes have three main components:

- A double layered epithelium lining the processes
- A highly vascularised and fenestrated capillary core
- The stroma, composed of mucopolysaccharide ground substance and collagen, that separates the capillary network from the epithelium.

Aqueous humour is derived from the blood plasma of the capillaries in the ciliary processes via three mechanisms:



diffusion, ultrafiltration and active transport (secretion). Most aqueous production is from the latter mechanism which occurs as a result of the active transport of sodium ions across the ciliary epithelium into the posterior chamber. Thus primary aqueous enters the posterior chamber where its composition is altered by the iris, ciliary body and crystalline lens, before passing through the pupil into the anterior chamber. It leaves the anterior chamber via the iridocorneal angle.

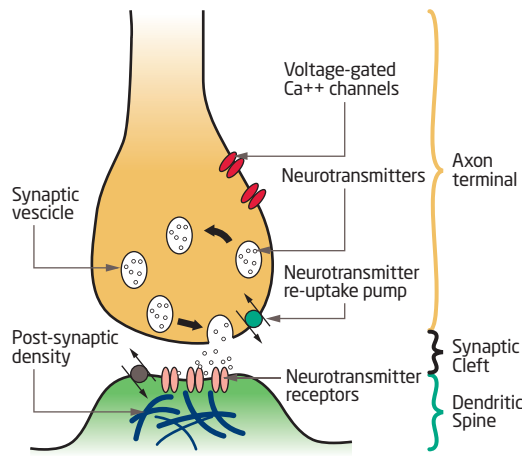
**Aqueous outflow**

The majority of aqueous leaves the eye through the trabecular meshwork (Figure 1), percolating into Schlemm’s canal and from there into the collector channels, finally draining into the episcleral veins. The rate of outflow is determined by the hydrostatic pressure head and resistance of the meshwork to flow. This is often called the ‘conventional’ outflow pathway and accounts for approximately 90 per cent of the outflow process. A small amount of aqueous exits the eye by passing through the interstitial spaces of ciliary muscle and choroid, or the suprachoroidal space and out of the eye through the sclera or the perivascular spaces surrounding the emissarial channels in the sclera. This ‘unconventional’ or uveoscleral outflow pathway is pressure-independent.

**Pharmacological principles of ophthalmic drugs**

Many of the drugs used in the medical management of glaucoma exert their actions by modulating the activity of the autonomic nervous system (ANS). The ANS regulates involuntary actions within the body, mainly the control of smooth muscle, cardiac muscle and exocrine glands. It is divided into the sympathetic and the parasympathetic nervous systems and differs from the somatic (voluntary) nervous system by possessing a synapse outside of the central nervous system (CNS).

The parasympathetic nerve supply to the eye originates from the oculomotor nucleus in the CNS, travelling via the oculomotor nerve to synapse in the ciliary ganglion. Post-ganglionic parasympathetic nerve fibres then enter the eye as the short ciliary nerves to innervate the ciliary muscle and iris sphincter pupillae muscle. Other parasympathetic nerve fibres enter the orbit with the trigeminal nerve to innervate the lacrimal gland. The sympathetic nerve supply of the eye comes from the cervical and upper thoracic segments of the spinal cord, synapsing



**Figure 2 Principle of neurohumoral transmission**

at the superior cervical ganglion. Some post-ganglionic sympathetic nerve fibres pass through the ciliary ganglion, entering the eye as short ciliary nerves to allow ocular vasoconstriction, others bypass the ciliary ganglion and enter the eye as long ciliary nerves innervating the iris dilator pupillae muscle. Another

branch of sympathetic nerves travel via the oculomotor nerve to Müller’s muscle, the smooth muscle component of levator palpebrae superioris.

**Neurohumoral transmission**

A synapse is a region of close proximity and communication between two neurones or a neurone and an effector cell. At this site there is a small gap called the synaptic cleft. On arrival at the synaptic junction, the nerve impulse from the pre-synaptic nerve causes a release of chemicals (or neurotransmitters) into the synaptic cleft. These bind with receptors on the post-synaptic membrane, resulting in either a propagation of the nerve impulse along the second neurone, or an action within the effector cell (Figure 2). This is the principle of neurohumoral transmission.

The parasympathetic and sympathetic nervous systems are often referred to as the cholinergic and adrenergic pathways respectively. These names refer to the neurotransmitter released from the pre-synaptic neurone to the effector cell – acetylcholine in

**TABLE 1 Summary of ANS receptor sites and actions**

Receptor	Distribution in the eye	Effect on aqueous dynamics	Action on other smooth muscle
<b>Parasympathetic/ cholinergic:</b>			
Muscarinic	Ciliary muscle Sphincter pupillae	Increase in aqueous outflow	Stimulation of sweat glands Inhibition of salivary glands
<b>Sympathetic/ adrenergic:</b>			
Alpha-1	Dilator muscle. Ciliary, retinal & choroïdal blood vessels	Reduction in aqueous formation	Vasoconstriction → blood pressure rises Inhibition of salivary glands
Alpha-2	Non-pigmented ciliary epithelium Ciliary muscle (NB: alpha-2 receptors are on the pre-synaptic terminal. Stimulation inhibits release of neurotransmitter)	Reduction in aqueous formation	Vasoconstriction Inhibition of salivary glands Centrally determined hypotension and sedation
Beta-1	Ciliary, retinal & choroïdal blood vessels	Promotes aqueous secretion	Present on cardiac muscle → increase in heart rate (tachycardia)
Beta-2	Non-pigmented ciliary epithelium Ciliary, retinal & choroïdal blood vessels Trabecular meshwork	Promotes aqueous secretion  Relaxes meshwork and increases outflow facility	Present on cardiac muscle → tachycardia  Present in tracheal and bronchial musculature → relaxation



the parasympathetic system and nor-adrenaline in the sympathetic system.

Some ophthalmic drugs potentiate the effect of the natural neurotransmitter (agonists) by either mimicking the action of the neurotransmitter ('direct' action), or inhibiting the re-uptake/breakdown of the neurotransmitter ('indirect' action). There are also drugs that competitively reduce the effect of a neurotransmitter (antagonists) by combining with the post-synaptic receptors to block the action of the neurotransmitter.

### Receptors in the eye

The distribution and actions of the post-ganglionic parasympathetic and sympathetic receptors on the eye are summarised in Table 1.

### Principal aims of treating glaucoma

Reduction of IOP by a third has been proven to reduce the rate of progression of glaucomatous optic neuropathy.<sup>8-10</sup> Therefore the aim of treatment is to find the simplest and safest means of lowering the IOP to a satisfactory level. This is achieved by acting either to reduce aqueous production or to increase aqueous outflow. The ideal topical medication for the treatment of glaucoma would be:

- Highly effective, offering long-term IOP control
- Well-tolerated with minimal topical and systemic side-effects
- Simple to use – ideally requiring one drop a day for minimal patient inconvenience, thereby maximising compliance
- Cost-effective.

The human tear volume is approximately 7µl with a tear turnover rate of approximately 1µl per minute. The use of topical drugs in the eye doubles this rate, spontaneous tearflow causing complete washout of medication from the conjunctival cul-de-sac within five minutes. Once a drop has been instilled into the eye, only 20 per cent manages to enter the eye, the rest draining through the nasolacrimal duct. Drop availability is increased to 35 per cent when the lacrimal punctum is occluded following drug instillation. The maximum bioavailability of a topical ophthalmic drug occurs when a drop volume of 20µl is administered.

It is worth noting that, following nasolacrimal drainage of an eye drop, substantial systemic absorption takes place through the highly vascularised nasal mucosa and via pulmonary absorption of inhaled drug particles. The relevance of this will be covered

**TABLE 2**

**Topical beta-blockers available**

Generic name	Brand	Concentrations	Cardio-Selectivity	Dose
Timolol maleate	Timoptol	0.25%, 0.5%	None	<i>Od, Bd</i>
Timolol maleate	Nyogel	0.1%	None	<i>Od</i>
Levobunolol hydrochloride	Betagan	0.5%	None	<i>Od, Bd</i>
Carteolol hydrochloride	Teoptic	1%, 2%	None	<i>Od, Bd</i>
Metipranolol minims (non-preserved)	Metipranolol	0.1%	None	<i>Od, Bd</i>
Betaxolol	Betoptic	0.25%	Beta-1	<i>Bd</i>

in more detail in the next section.

### Commonly encountered ocular hypotensive medications

#### Cholinergic agents/parasympathomimetics

Miotics, such as pilocarpine, are long established medications that have been used in ophthalmology for almost 100 years. Their mode of action is increasing aqueous outflow by contraction of the longitudinal muscle of the ciliary body. Contraction of this muscle pulls on the scleral spur posteriorly and internally, thus opening up the trabecular meshwork.

Although miotics have a good safety record and offer significant IOP lowering effects, the inconvenience of a four times a day dosing regimen, the visual disturbance caused by significant pupil miosis and common headaches from ciliary muscle spasm mean that they are no longer routinely used to manage chronic open-angle glaucoma.

#### Beta-adrenergic antagonists (beta-blockers)

Since the introduction of Timolol in the early 1970s, beta-blockers have remained a popular choice of treatment in the management of glaucoma. The precise mechanism by which beta-blockers lower IOP remains unknown, but their use results in a reduced aqueous formation. Research suggests aqueous humour formation is mediated by tonic sympathetic stimulation.

Beta-blockers are usually instilled once or twice a day and are licensed for use as monotherapy or in conjunction with other drugs. As aqueous production is naturally reduced at night, many feel a simple once-a-day morning instillation of the drug to be

as effective at IOP reduction as a twice daily regimen.

#### Long-term control

Despite the ability to produce a 20-40 per cent IOP reduction, beta-blockers, efficacy diminishes over time in up to a fifth of individuals prescribed the drug. This reduction in long-term efficacy has been termed 'long-term drift' or 'tachyphylaxis' and should be remembered when following up a patient on β-blocker monotherapy.

#### Side-effects and contra-indications

In humans, there are two main beta-adrenergic receptors, β<sub>1</sub> and β<sub>2</sub>. The former predominate in the heart while the latter are found in the lungs. Pharmacological blocking of beta-receptors in these tissues can therefore result in a reduced heart rate and reduced air entry to the lungs. Most topical beta-blockers are non-selective – they block both types of receptor. Only betaxolol claims to be a cardio-selective beta-1 blocker, theoretically avoiding the undesirable respiratory side-effects.

Systemic absorption of ophthalmic drugs following topical instillation represents a real problem in the case of topical beta-blockers. Nasal mucosal absorption avoids early drug metabolism by the liver and leads to blood concentrations sufficient to cause haemodynamic impairment. Cardiovascular side-effects associated with the use of topical beta-blockers include systemic hypotension, syncope, impotence and, in extreme cases, myocardial infarction. Studies have found an increase in the number of falls in elderly glaucoma patients using topical beta-blockers.

Using a weaker concentration of the drug will cause fewer side-effects.



**Figure 3** Topical alpha-agonist allergy (courtesy of Keith Barton, Moorfields Eye Hospital)

Although 0.1 per cent and 0.25 per cent concentrations of timolol are as effective at reducing IOP as the 0.5 per cent concentration, the higher concentration seems to be much more widely prescribed. Betaxolol can be useful in patients in whom non-selective beta-blockers are contraindicated but it is not as effective at reducing IOP as timolol and may still result in adverse pulmonary side-effects in susceptible individuals. Never forget that the use of beta-blockers is contraindicated in patients with asthma or a history of chronic obstructive airways disease.<sup>11</sup>

Shared-care/co-management perspective – when to refer back to the primary ophthalmic care provider

- Evidence of long-term drift of IOP control
- Suspicious symptoms such as shortness of breath or newly prescribed inhalers (suggesting patient having respiratory side-effects)
- Interactions with systemic medication, such as an enhanced hypotensive effect if patient is using ACE inhibitors or calcium channel blockers for high blood pressure.

**Alpha-adrenergic agonists (alpha-agonists)**

Alpha-agonists reduce aqueous production and improve its outflow through the trabecular meshwork. They have

**TABLE 3**

Some reported effects of alpha-agonists used in glaucoma treatment

	Apraclonidine hydrochloride (Iopidine)	Brimonidine tartrate (Alphagan)
Concentrations available	0.5 %, 1 %	0.2 %
Tachyphylaxis	Yes	No
Blurry vision	3 %	17.5 %
Conjunctival hyperaemia	13 %	3 % to 17 %
Conjunctival follicles	Yes	4 % to 13 %
Lid retraction	45 %	Not stated
Ocular allergy	Up to 36 %	Up to 13 %
Foreign body sensation	3 %	Up to 17%
'Burning'	Yes	6 %
Crosses the blood-brain barrier?	No	Yes
Dry mouth	10 % to 19 %	30 %
Fatigue	1 %	4 % to 13 %
Headache	1 %	4 % to 13 %

been used to treat glaucoma since the early 20th century, originally with the use of topical adrenaline. However, the numerous ocular and systemic side-effects of topical adrenaline resulted in it being phased out following the arrival of topical beta-blockers.

Several theories exist as to how alpha-agonists exert their IOP-lowering effects. It is thought that stimulation of the alpha-2 receptors on the pre-synaptic nerve ending prevents it from releasing its neurotransmitter nor-adrenaline. This in turn reduces aqueous production, primarily by causing vasoconstriction of the capillary core within the ciliary processes. The main alpha-agonist topical preparations available today are apraclonidine hydrochloride and brimonidine tartrate. These are chiefly alpha-2 agonists, although some reports suggest that apraclonidine also has an effect on alpha-1 receptors. Brimonidine is

thought to exert its hypotensive effect though reduction of aqueous production and also by increasing outflow through the uveoscleral pathway.

Long-term control

Some reports have shown a reduced long-term efficacy with apraclonidine but not with brimonidine.

Side-effects and contraindications

Table 3 lists some of the ocular and systemic side-effects of topical apraclonidine and brimonidine. The most significant ocular side-effect of apraclonidine is a follicular conjunctivitis with or without associated contact dermatitis, making it of limited use as long-term therapy (Figure 3). However, apraclonidine is used immediately after anterior segment laser treatment to prevent IOP spikes and is occasionally used as the long-term treatment of POAG in patients for whom other

topical medications have failed and in whom surgery is contraindicated.

Apraclonidine and brimonidine are both derived from the systemic antihypertensive drug clonidine, which was found to decrease IOP by reducing aqueous production. Apraclonidine does not cross the blood-brain barrier and so has no systemic hypotensive effects. Dry nose and dry mouth are the most common non-ocular side-effects. Brimonidine does cross the blood-brain barrier and can cause mild systemic hypotension and lethargy but causes fewer local side-effects than apraclonidine.

Shared-care/co-management perspective – when to refer back to the primary ophthalmic care provider

- A delayed hypersensitivity reaction to brimonidine tartrate eye drops resembles a viral follicular conjunctivitis with or without a periocular contact dermatitis and can occur many months after initially prescribing the drug. A patient exhibiting these signs/symptoms should be referred back to the ophthalmologist for alternative treatment

- Systemic drug interactions – alpha-adrenergic agonists are contraindicated in patients using tricyclic or monoamine oxidase inhibitor antidepressants. The effect of interactions with these drugs have not been extensively studied but it is possible that tricyclic antidepressants may blunt the hypotensive effect of brimonidine.

## Topical carbonic anhydrase inhibitors

Carbonic anhydrase catalyses the conversion of carbon dioxide to bicarbonate ions. Inhibition of this reaction in the ciliary body results in reduced active transport of sodium ions across the ciliary epithelium and thus reduced formation of aqueous humour.

Systemic carbonic anhydrase inhibitors (CAIs) include acetazolamide and methazolamide. These are used orally or intravenously to treat acute IOP rises. Their serious side-effects, including renal impairment and potential to cause fatal haematological disorders prevent them from being employed in the treatment of chronic glaucoma.

Two topical CAIs are available – dorzolamide hydrochloride 2 per cent (Trusopt) and brinzolamide 1 per cent (Azopt). These are less effective than timolol at reducing IOP and as such are used as an adjunctive therapy rather than first-line treatment. Although their maximum hypotensive effect is achieved with a three times daily

**Figure 4**  
Topical prostaglandin induced eyelash growth and skin pigmentation (courtesy of eyetext.net)



**TABLE 4**

Currently available prostaglandin analogues in the UK

Latanoprost (Xalatan)	Travoprost (Travatan)	Bimatoprost (Lumigan)	
Concentration	0.005%	0.004%	0.03 %
Ester or amide?	Isopropylester	Isopropylester	Ethylamide
Dose	Od (usually nocte)	Od (usually nocte)	Od (usually nocte)
Reported IOP reduction	30% average (max report 49%)	30% average (max report 55%)	30% average
Tachyphylaxis	No	No	No

dosage, to keep in line with beta-blocker agents and promote compliance with treatment they are often prescribed for twice daily usage.

Long-term control

Although systemic CAIs reduce aqueous production by up to 30 per cent, the topical preparations are much less effective, reducing production by approximately 18 per cent. There are no reports of tachyphylaxis occurring with the topical preparations.

Side-effects and contraindications

Carbonic anhydrase is distributed ubiquitously in the human body. It is extremely important in the red blood cells for the transport of CO<sub>2</sub>, and carbonic acid (H<sub>2</sub>CO<sub>3</sub>) is part of the major buffer system in the human body.

In the eye, the corneal endothelial pump requires carbonic anhydrase to maintain corneal dehydration and transparency. Studies have shown that in patients with endothelial gutatta or other endothelial pathology, irreversible corneal oedema can occur with topical dorzolamide. This has not been shown to be the case with normal, non-pathological corneas. Thus, topical CAIs are avoided in patients who have compromised corneal endothelium, such as in those with Fuch's dystrophy or post-intraocular surgery.

Other local side-effects of topical CAIs include a stinging, burning sensation on instillation and a bitter taste.

Shared-care/co-management perspective – when to refer back to the primary ophthalmic care provider

- Loss of corneal transparency
- Low patient compliance due to discomfort on instillation
- There are few reported drug interactions with topical CAIs.

Prostaglandin analogues

The prostaglandin analogues are a newer class of highly effective topical ocular hypotensive drugs. Thanks to their potent IOP-lowering effects and once daily dosing regime they have become the commonest first-line treatment in the UK. Prostaglandins occur naturally in the body and have a wide variety of actions including, but not limited to, muscular contraction and mediation of inflammation. Naturally occurring prostaglandin F<sub>2α</sub> is known to lower IOP but at the expense of increased ocular inflammation. As a result, a modified molecule was synthesised to produce a compound with a more favourable adverse effect profile.

Topical prostaglandin analogues usually come in the form of an isopropyl ester or ethyl amide. The ester is hydrolysed within the cornea to free acid in



**TABLE 5**  
Fixed combination preparations

Drug combination	Brand name
Dorzolamide 2%/timolol 0.5%	Cosopt
Brimonidine 0.2%/timolol 0.5%	Combigan
Latanoprost 0.005%/timolol 0.5%	Xalacom
Bimatoprost 0.03%/timolol 0.5%	Ganfort
Travoprost 0.004%/timolol 0.5%	Duotrav

the anterior chamber which binds to the prostaglandin  $F_{2\alpha}$  receptors (or FP receptors) within ciliary muscle. Its main mechanism of action is thought to be in the remodelling of the extracellular matrix of ciliary muscle, resulting in a widening of connective tissue filled spaces and allowing increased uveoscleral outflow. The ethylamide does not bind to the FP receptors. It is thought not only to enhance uveoscleral outflow but also to promote outflow through the trabecular meshwork.

Long term efficacy

There is no reported tachyphylaxis with topical prostaglandin analogues. Initial concerns about the cost of latanoprost have lessened as the arrival of other compounds has made the market more competitive.

A recent meta-analysis of commonly prescribed topical ocular hypertensives used in the management of POAG confirmed that prostaglandin analogues and topical beta-blockers were the most effective intraocular pressure-reducing agents.<sup>12</sup>

Side-effects and contraindications

In clinical trials the safety profile of prostaglandin analogues is promising with no serious systemic side-effects to date, although some patients have reported muscle and joint pains, migraine and flu-like symptoms. There

are several notable ocular side-effects including conjunctival hyperaemia, hypertrichosis and an increase in iris, eyelash and periocular skin pigmentation (Figure 4). In patients who have had cataract surgery or uveitis, there is also an increased risk of developing cystoid macular oedema.

The main contraindication to using these medications is the theoretical risk of inducing abortion in women of childbearing age.

Fixed-combination drugs

Monotherapy frequently fails to achieve a satisfactory IOP reduction in the glaucoma patient. The European Glaucoma Society guidelines suggest that if the treatment does not appear to be working, replacement monotherapy should be attempted before adding a second drug to a patient's treatment regimen. Polypharmacy should be avoided if possible as compliance is likely to suffer (see below).

However, there are cases in which one drug is inadequate to lower a patient's IOP to a desirable level, and a further eye drop is then required. The ocular hypotensive effect of prostaglandin analogues may be effectively supplemented by concomitant use of other topical hypotensive agents. Use of beta-blocker preparations with brimonidine and prostaglandin analogues have been shown to be more effective at IOP lowering than the use of one drug alone.<sup>13-15</sup>

These findings have led pharmaceutical companies to develop fixed combination eye drops containing two therapeutic agents in a single bottle. These combined preparations have many advantages, particularly the potential for improved patient compliance. However, it is important to note that although all of the fixed combinations contain timolol 0.5 per cent, none give any suggestion as to their beta-

blocker component in their proprietary names. As we become increasingly familiar with these products it remains of the utmost importance that we remember to exclude contraindications to beta-blocker use and are alert to beta-blocker-induced side-effects when seeing patients using combination therapy drugs.<sup>16</sup>

Side-effects and contraindications

The side-effects and contraindications of fixed combination preparations are the same as their individual components.

The issue of patient compliance

Glaucoma is a chronic condition requiring, in the majority of cases, lifelong topical therapy. Reports have suggested that up to 75 per cent of patients are non-compliant with their prescribed treatment regimen. Reasons for this include poor memory, unwanted side-effects of the drug, impaired manual dexterity and confusion at the complex treatment regimes.<sup>17</sup> In addition, poor compliance may be more likely in those that have a poor understanding of their disease. Unlike conditions such as cataract or age-related macular degeneration, glaucoma does not have an obvious detrimental effect on visual acuity until the late stages of the disease. Accordingly, patients often remain relatively asymptomatic until they have advanced glaucoma and find it difficult to accept the importance of good compliance in maintaining their visual status quo.

Improving compliance poses a real challenge to the clinician. Simplifying the treatment regime by using once-a-day treatment or combinations drops, or providing gadgets to help arthritic patients successfully instil their drops can help. Patients reporting good compliance may not actually be getting the drops in due to poor



instillation technique. Time taken instructing patients how to self-instill drops effectively may be as useful as the medication itself. In cases where there is 'wilful' poor compliance, patient education regarding their condition and the real-life consequences of untreated glaucoma, such as the potential loss of their driving licence, may encourage patients to use their treatment more regularly. Patients may stop their eye drops due to their numerous side-effects, so it is important to enquire about these at every visit and whether they have started any new systemic medications with the potential for adverse interactions.

### The future of optometric co-management

The success or failure of shared-care will depend entirely upon the optometric community and ophthalmologists managing to communicate effectively with one another. Only in doing so can we hope to work together, yet physically apart, to provide the complete care our patients are right to expect. Working hard to learn new tricks and having the best intentions will never be enough: team-players only need apply.

Shared-care presents many new opportunities; the possibility of replacing the guessing games of the past with informative and constructive two-way dialogue is one of the most exciting. Agreeing clear guidelines as to which patients should be re-referred back to hospital will form a part of the communication puzzle – and both sides adhering to them will build trust.

Finally, as we recognise that some of the 'who' and 'where' parts of health-care provision in the UK are changing, let us also acknowledge that the patients, their physical and their psychological needs have not. Change is difficult at any time; the advancing age and sight-threatening diagnosis which are the burden of most glaucoma patients will not serve to make the transition away from traditional hospital-based, doctor-provided care any easier. When in difficulty apply the old medical rule 'First, do no harm'. It is as good a starting place as any and a universally understood sentiment. ●

### References

- 1 Tuck MW and Crick RP. The projected increase in glaucoma due to an ageing population. *Ophthalmic Physiol Opt*, 2003; 23; 2: 175-9.
- 2 Bell RW and O'Brien C. Accuracy of referral to a glaucoma clinic. *Ophthalmic Physiol Opt*, 1997; 17; 1: 7-11.
- 3 Department of Health National Eyecare

## MULTIPLE-CHOICE QUESTIONS - take part at [opticianonline.net](http://opticianonline.net)

### 1 Which of these statements about the autonomic nervous system is incorrect?

- A It is part of the vertebrate nervous system that controls involuntary action
- B It possesses a synapse within the central nervous system
- C It is divided into the adrenergic and cholinergic nervous systems
- D The parasympathetic fibres synapse close to the neuro-effector organ

### 2 Which of the following statements about the parasympathetic innervation to the eye is incorrect?

- A The oculomotor nerve parasympathetic fibres enter the eye via the short ciliary nerves
- B The oculomotor nerve parasympathetic fibres originate from the Edinger Westphal nucleus
- C Fibres innervating the ciliary muscle and lacrimal gland synapse in the ciliary ganglion
- D Stimulation results in miosis, accommodation and lacrimal gland secretion

### 3 Which of these statements on adrenergic innervation to the eye is correct?

- A Stimulation of the beta-2 receptors results in vasodilation
- B Preganglionic nerves originate in the superior cervical ganglion
- C Stimulation results in dilation and ptosis
- D Nerve fibres enter the cranium with the external carotid artery

### 4 Mydriasis occurs with:

- A Pilocarpine
- B Carbachol
- C Apraclonidine
- D Methazolamide

### 5 Timolol exerts its ocular hypotensive effect by:

- A Inhibition of the production of bicarbonate ions
- B Acting on the ciliary processes to reduce aqueous formation
- C Increasing uveoscleral outflow
- D Increasing aqueous outflow due to contraction of the ciliary muscle

### 6 Which of the following statements about prostaglandin analogues is incorrect?

- A They are usually in the form of an isopropyl ester or ethyl amide
- B They cause an increase in uveoscleral outflow
- C Prolonged use results in increased pigmentation of periocular skin and iris
- D They are hydrolysed into a free acid in the anterior chamber

### 7 Which of the following statements about topical beta-blockers is correct?

- A They are always applied once daily
- B They can be absorbed systemically and so are contraindicated in patients with obstructive airways disease
- C They do not affect the blood-brain barrier
- D Long-term use may result in ptosis

### 8 Which of the following statements about topical alpha-agonists is correct?

- A They are the most efficacious ocular hypotensive agent
- B Their primary mode of action is by increasing outflow facility
- C They can be administered safely in patients with Fuch's endothelial dystrophy
- D They are a poor adjunctive choice with beta-antagonist agents

### 9 Which of the following agents lowers IOP by increasing aqueous outflow?

- A Travoprost
- B Betagan
- C Lopidine
- D Trusopt

### 10 A patient on multiple therapy attends the clinic complaining of a dry mouth. Which of the following is likely to be causing the symptoms?

- A Latanoprost
- B Carteolol
- C Brimonidine
- D Dorzolamide

### 11 A healthy 78-year-old bilateral pseudophake who has been on topical beta-blockers for 7 years attends the clinic with IOPs of R 18 mmHg and L 19 mmHg. The target IOP required is in the low-mid teens. What is the least favourable course of action (assuming cost no issue)?

- A Stop beta-blocker and switch to latanoprost *od nocte*
- B Addition of brimonidine to treatment regimen
- C Check drop technique and compliance
- D Addition of Ganfort

### 12 A healthy 40-year-old woman has just been diagnosed with POAG. Her highest known IOPs are R 24mmHg L 23mmHg and based on her disc appearance and visual fields at presentation it has been decided that her target IOP should be in the mid-teens. Of these treatment regimens, which is the best first-line treatment?

- A Duotrav *od*
- B Betagan *bd*
- C Nyogel *od*
- D Brimonidine *tds*

Successful participation counts as two credits towards the GOC CET scheme and one towards the Association of Optometrists Ireland's scheme. **Deadline for response is December 27**





Steering Group 2004.

**4** Spry PG, Spencer IC, Sparrow JM, *et al.* The Bristol Shared Care Glaucoma Study: reliability of community optometric and hospital eye service test measures. *Br J Ophthalmol*, 1999; 83; 6: 707-12.

**5** Henson DB, Spencer AF, Harper R, and Cadman EJ. Community refinement of glaucoma referrals. *Eye*, 2003; 17; 1: 21-6.

**6** Resnikoff S, Pascolini D, Etya'ale D, *et al.* Global data on visual impairment in the year 2002. *Bulletin of the World Health Organization*, 2004; 82; 11: 844-851.

**7** European Glaucoma Society Terminology and Guidelines for Glaucoma. 2003, DOGMA Srl: Savona, Italy.

**8** Heijl A, Leske MC, Bengtsson B, Hyman L, and Hussein M. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. *Arch Ophthalmol*, 2002; 120; 10: 1268-79.

**9** Kass MA and Gordon MO. Intraocular pressure and visual field progression in open-angle glaucoma. *Am J Ophthalmol*, 2000; 130; 4: 490-1.

**10** Kass MA, Heuer DK, Higginbotham EJ, *et al.* The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. *Arch Ophthalmol*, 2002; 120; 6: 701-13; discussion 829-30.

**11** Diggory P, Cassels-Brown A, Vail A, Abbey LM, and Hillman JS. Avoiding unsuspected respiratory side-effects of topical timolol with cardioselective or sympathomimetic agents. *Lancet*, 1995; 345; 8965: 1604-6.

**12** van der Valk R, Webers CA, Schouten JS, *et al.* Intraocular pressure-lowering effects of all commonly used glaucoma drugs: a meta-analysis of randomized clinical trials. *Ophthalmology*, 2005; 112; 7: 1177-85.

**13** Simmons ST and Earl ML. Three-month comparison of brimonidine and latanoprost as adjunctive therapy in glaucoma and ocular hypertension patients uncontrolled on beta-blockers: tolerance and peak intraocular pressure lowering. *Ophthalmology*, 2002; 109; 2: 307-14; discussion 314-5.

**14** O'Connor DJ, Martone JF, and Mead A. Additive intraocular pressure lowering effect of various medications with latanoprost. *Am J Ophthalmol*, 2002; 133; 6: 836-7.

**15** Konstas AG, Karabatsas CH, Lallos N, *et al.* 24-hour intraocular pressures with brimonidine purite versus dorzolamide added to latanoprost in primary open-angle glaucoma

subjects. *Ophthalmology*, 2005; 112; 4: 603-8.

**16** Spratt A, Ogunbowale L, Wormald R, and Franks W. What's in a name? New glaucoma drugs. *Lancet*, 2006; 368; 9538: 826-7.

**17** Winfield AJ, Jessiman D, Williams A, and Esakowitz L. A study of the causes of non-compliance by patients prescribed eyedrops. *Br J Ophthalmol*, 1990; 74; 8: 477-80.



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