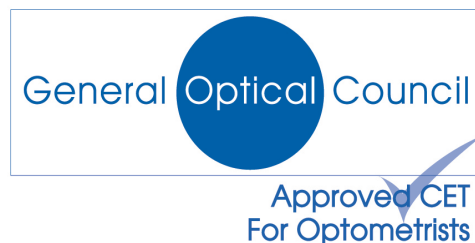


# Glaucoma detection for the domiciliary optometrist.

## Paper Two: Management

C 17915 (O)

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GOC Registered Optometrists

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## Medical Management of Glaucoma

The first of these two articles discussed the detection of glaucoma by domiciliary optometrists. This article will discuss the treatment options for glaucoma with specific emphasis on the medical treatments available and common side-effects of these medications.

### The role of IOP in glaucoma

Ocular hypertension (OHT) is a major risk factor for the development of glaucoma<sup>1</sup>. The majority of patients with ocular hypertension, however, do not develop glaucoma. There is also a distinct group of glaucoma patients in whom the IOP is always within the statistically normal range and yet they display glaucomatous changes to the optic nerve and visual field (normal tension glaucoma). We know from landmark glaucoma studies such as OHTS, EMGT, CNTG, and AGIS<sup>2-5</sup> that lowering the IOP is the only strategy known to either reduce the risk of conversion from OHT to COAG, or to prevent or slow the rate of progression in established glaucoma. Lowering the IOP can be achieved medically (with topical hypotensive eye drops), by laser procedures such as argon laser trabeculoplasty (ALT) or selective laser trabeculoplasty (SLT), or surgically by trabeculectomy. In the UK, the majority of glaucoma patients are managed medically. A discussion of laser and surgical treatments is beyond the scope of this article, but interested readers are advised to look at a very good review by Vernon<sup>6</sup>.

### Intra-ocular pressure and aqueous humour dynamics

The intra-ocular pressure is determined by the relative balance between aqueous humour production and drainage. Aqueous is a colourless fluid that is essential for supplying nutrients to the avascular cornea and lens, and for the removal of metabolic waste products. It also gives the eyeball its turgidity. Aqueous is constantly being produced by the ciliary processes of the ciliary body. The ciliary processes have a rich, highly permeable, capillary vascular supply from which aqueous is derived. Aqueous production is predominantly an active secretory process, which employs a number of enzymatic systems including carbonic anhydrase and  $\text{Na}^+/\text{K}^+$  ATPase. These systems help drive bicarbonate and sodium ions respectively into the posterior chamber, thus creating an osmotic gradient to encourage the movement of water out from the plasma in the capillaries. This is believed to be predominantly driven by the sympathetic division of the autonomic nervous system (ANS). Although the mechanism is not fully understood, it is likely that  $\beta_2$ -adrenergic stimulation increases aqueous

production by the ciliary epithelium. Aqueous passes from the posterior chamber around the equator of the lens, through the pupil, and into the anterior chamber. Thermal convection currents circulate the aqueous within the anterior chamber.

Aqueous drainage occurs via two routes:

#### *The conventional pathway:*

through the trabecular meshwork into Schlemm's canal, then through the episcleral venous plexus. This pathway is pressure dependent, with greater outflow facility at higher intra-ocular pressure. This accounts for ~75-90% of aqueous outflow.

#### *The unconventional pathway:*

a pressure independent pathway whereby aqueous drains or seeps into the iris root and passes between ciliary muscle fibre bundles into the supraciliary and suprachoroidal spaces.

### The autonomic nervous system and the eye.

Some of the medical compounds used in glaucoma management exert their actions by modulating the activity of the autonomic nervous system. Noradrenaline is the neurotransmitter released from postganglionic neurones in the sympathetic division of the ANS, whilst acetylcholine is the neurotransmitter released from postganglionic neurones in the parasympathetic division of the ANS. Some glaucoma drugs will mimic the effects of these neurotransmitters (agonists) whilst others will actively compete against these neurotransmitters (antagonists).

Postganglionic parasympathetic nerve fibres innervate the iris sphincter muscle and the ciliary body. Acetylcholine will stimulate muscarinic receptors, which will increase aqueous outflow via contraction of the ciliary body.

Alpha-1,  $\alpha_2$ ,  $\beta_1$ , and  $\beta_2$ -receptors are found within the postganglionic sympathetic system:

- **$\alpha_1$ -receptors** are found in the iris dilator muscle and in the smooth muscle of the ciliary, retinal and choroidal blood vessels. Stimulation of these receptors tends to cause smooth muscle contraction.
- **$\alpha_2$ -receptors** are found mainly on the presynaptic nerve terminal, and stimulation inhibits further release of noradrenaline.
- **$\beta_1$ -receptors** are predominantly found in the heart, stimulation leading to increased heart rate (tachycardia). They are also distributed in the smooth muscle of the ciliary, retinal and choroidal blood vessels.

- **$\beta_2$ -receptors** are distributed throughout the non-pigmented ciliary epithelium as well as the smooth muscle of ciliary, retinal and choroidal blood vessels. They are also found in the tracheal and bronchial muscles, stimulation leading to relaxation and dilation.

## Medical treatment options

Intra-ocular pressure reduction can be achieved either by reducing aqueous production and /or by increasing aqueous outflow. There is currently a choice of five topical hypotensive drug groups (**Table 1**):

Drug Group	Beta-blockers	PGAs	CAIs	Sympathomimetics	Parasympathomimetics
Drug Name	Timolol* (Timoptol, Timoptol-LA, Nyogel) Levobunolol* (Betagan) Carteolol (Teoptic) Betaxolol* (Betoptic) Metipranolol	Latanaprost (Xalatan) Bimatoprost (Lumigan) Travoprost (Travatan) Tafluprost (Saflutan)	Dorzolamide* (Trusopt) Brinzolamide (Azopt)	Brimonidine* (Alphagan)  Nb. Dipivefrine (Propine) discontinued	Pilocarpine Pilogel
Dose	Twice daily <sup>§</sup>	Once daily (night)	Two/three times daily	Twice daily	Four times daily <sup>§</sup>

Table 1: Topical hypotensive drugs available in the UK

### Table Notes:

PGAs=prostaglandin analogues; CAIs=carbonic anhydrase inhibitors.

\*denotes non-proprietary availability.

Names in brackets indicate brand names.

Different strengths and formulations exist, please refer to the British National Formulary (BNF) for full information.

Blue indicates unpreserved option available.

<sup>§</sup>except Timoptol-LA and Nyogel which are once daily

Prostaglandin analogues and beta-blockers are licensed for first-line use, the remainder for second-line use only. Fixed-combination drops are also available (**Table 2**).

Brand name	Beta-blocker	PGA	CAI	$\alpha_2$ -adrenergic agonist	Dose
Xalacom	Timolol 0.5%	Latanoprost			Once daily
DuoTrav	Timolol 0.5%	Travoprost			Once daily
Ganfort	Timolol 0.5%	Bimatoprost			Once daily
Cosopt	Timolol 0.5%		Dorzolamide		Twice daily
Azarga	Timolol 0.5%		Brinzolamide		Twice daily
Combigan	Timolol 0.5%			Brimonidine	Twice daily

Table 2: Fixed-combination topical hypotensive eye drops

**Aqueous suppressants**

Beta-blockers and CAIs reduce aqueous production, albeit by different pharmacological mechanisms. In the 1970s the development of timolol established beta-blockers as an effective treatment for OHT and COAG. It became the benchmark against which all other topical hypotensive treatments were compared. There are a number of different timolol preparations available as well as a number of other topical beta-blockers (Table 1). With the exception of betaxolol, they are all non-selective and block both  $\beta_1$  and  $\beta_2$  receptors. It is universally agreed that beta-blockers reduce aqueous humour production by competitive blockade of noradrenaline at the  $\beta_2$ -receptors on the ciliary epithelium<sup>7</sup>, although curiously betaxolol is a relatively selective  $\beta_1$ -receptor blocker. They have little or no effect on aqueous outflow. Because of its cardioselectivity, betaxolol may be a more appropriate choice for people with asthma or chronic obstructive pulmonary disease (COPD). In the author’s experience, however, betaxolol is not used due to its weaker IOP-lowering effect compared to timolol. Beta-blockers are potent medicines and have the ability to cause significant systemic side-effects (Table 3). They should be avoided in patients with a history of asthma, COPD, sinus bradycardia (slow heart rate), atrio-ventricular heart block or cardiac failure. If a patient is diabetic, they should be advised to monitor their blood sugars closely as beta-blockers have the ability to mask the signs and symptoms of low blood sugar, which may put them at risk of hypoglycaemic shock. Beta-blockers are lipophilic and may cross the blood-brain barrier into the central nervous system (CNS). This may give rise to CNS disturbances ( although these are quite rare. Beta-blockers cause few ocular side-effects and are well tolerated by the majority of patients who use them.

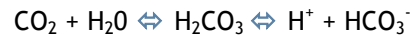
Cardiovascular	Pulmonary	Central Nervous System
Bradycardia Hypotension Arrhythmia Syncope Heart failure Myocardial infarction Death	Tracheal/bronchial muscle constriction Shortness of breath Airway obstruction/asthma Pulmonary failure	Depression Fatigue Lethargy Hallucinations

Table 3 - systemic side effects of beta-blockers

**Carbonic Anhydrase Inhibitors**

These topical hypotensive agents reduce aqueous humour production by inhibiting the action of the enzyme carbonic anhydrase. This enzyme, found in the ciliary epithelium, catalyses the reversible reaction of carbon

dioxide and water into carbonic acid, which dissociates into bicarbonate and hydrogen ions:



Bicarbonate ions are actively secreted from the ciliary epithelium into the posterior chamber, creating an osmotic gradient, which leads to fluid movement and aqueous humour production. Blockade of the enzyme carbonic anhydrase will therefore prevent the formation of this osmotic gradient and consequently aqueous humour production.

Carbonic anhydrase inhibitors are available in both topical and systemic formats. The systemic CAI acetazolamide (Diamox) may be given orally in tablet form or by intravenous injection. It is used as an adjunct to other glaucoma treatments to bring about either a rapid lowering of IOP e.g. in acute angle-closure, or in situations where topical treatments alone produce an insufficient lowering of IOP. It is frequently associated with adverse side effects such as tingling in the fingers and toes, taste disturbance, headache, fatigue, gastrointestinal disturbance and kidney stone formation.

Topical CAIs include dorzolamide (Trusopt) and brinzolamide (Azopt). Both have a comparatively weak IOP lowering effect compared to beta-blockers and PGAs, hence their licence for second-line use only. Systemic side effects (Table 4) are relatively uncommon with topical CAIs, but a transient metallic taste is frequently reported due to drainage into the nasopharynx. Local side effects are common. These medicines are derived from sulphonamides (a group of antibiotics which are rarely used now) and are therefore contraindicated in patients with known allergy to this class of medication.

Ocular side-effects	Systemic side-effects
Stinging Allergy Dryness/foreign body sensation Blurred vision Conjunctival hyperaemia	Metallic taste Urticaria Headache Bowel disturbance

Table 4 - ocular and systemic side effects of CAIs

**Adrenergic-agonists**

Alpha<sub>2</sub>-adrenergic agonists reduce aqueous production and increase aqueous outflow. Brimonidine 0.2% (Alphagan or non-proprietary) and apraclonidine (Iopidine 0.5% and 1%) are currently the only  $\alpha_2$ -agonists in the UK. Brimonidine is also available in combination with Timolol 0.5% as Combigan.

Brimonidine is a selective  $\alpha_2$ -agonist and a potent hypotensive agent. It is used for the treatment of ocular hypertension and COAG, and is especially effective as an adjunct to other topical hypotensive agents. Apraclonidine has both  $\alpha_1$ - and  $\alpha_2$ -adrenergic activity. Its main use is in the prevention of IOP-spikes following anterior segment laser procedures such as argon laser trabeculoplasty (ALT) and laser peripheral iridotomy (LPI), both of which can be associated with a post-laser rise in IOP.

Brimonidine reduces aqueous humour production via its action on the pre-synaptic nerve terminal, inhibiting the release of noradrenaline and thus preventing further aqueous production. Apraclonidine is much less selective and may further reduce IOP production by vasoconstriction of the uveal vascular supply via its activity on  $\alpha_1$ -adrenergic receptors. Brimonidine has the additional feature of increasing aqueous outflow via the uveoscleral drainage route<sup>8</sup>.

Systemic and local ocular side effects are associated with the use of these medicines and are summarized in [Table 5](#).

Ocular side-effects	Systemic side-effects
Burning/stinging	Dry mouth
Allergy	Headache
Blurred vision	Fatigue
Conjunctival hyperaemia	Drowsiness

Table 5 - Side effects of  $\alpha_2$ -adrenergic agonists

## Medications that increase aqueous outflow

### Prostaglandin analogues

The prostaglandins (PGAs) arrived in the mid-1990s and have now become the mainstay of modern glaucoma management, principally due to their effectiveness, once-daily dosing, tolerability and low incidence of systemic side-effects.

PGAs are potent hypotensive agents, reducing IOP by 25%-35% with a single daily dose<sup>9</sup>. There are currently four PGA eye drops available in the UK: latanaprost (Xalatan 0.005%), bimatoprost (Lumigan 0.03% and 0.01%), travoprost (Travatan) and a preservative-free preparation tafluprost (Saflutan). PGAs work by enlarging the spaces between ciliary muscle fibres and by “softening” the extracellular matrix surrounding the muscle fibre bundles by reducing the density of collagen and laminin. These changes increase the permeability of the ciliary body and consequently increase aqueous

outflow. Both latanaprost and travoprost achieve their effect by acting on the prostaglandin-FP2 $\alpha$  receptor class. There is some debate about the exact mechanism by which bimatoprost achieves its pressure-lowering effect, some studies indicating that it increases trabecular outflow facility as well as uveoscleral<sup>10</sup>.

PGAs are generally well tolerated by patients. Side-effects tend to be mild and local. Serious side-effects are infrequent and systemic ones rare. Local side-effects include stinging, foreign body sensation and conjunctival hyperaemia, which is most prevalent with bimatoprost and least with latanoprost. Other ocular side-effects include irreversible iris hyperpigmentation, darkening and lengthening of the eyelashes ([Fig1](#)), distichiasis, and (reversible) periocular hyperpigmentation. Cystoid macular oedema may occur in aphakic patients or pseudophakes where the posterior capsule has been ruptured. PGAs may also exacerbate anterior uveitis. Systemic side-effects are rarely encountered. They are well tolerated by patients with asthma, although reports of wheeziness and asthma have been reported in post-marketing surveillance<sup>11</sup>.

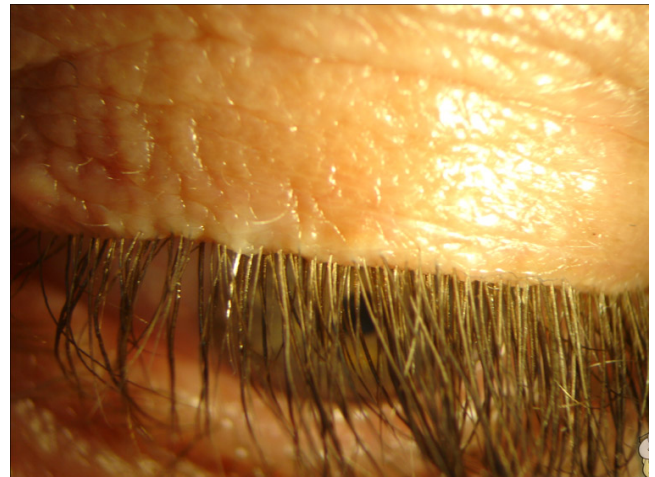


Fig 1: Long, lush eyelash growth that can occur with prostaglandin analogue drops like Xalatan and Travatan.

### Parasympathomimetics

Parasympathomimetics (or cholinergic agonists) are the oldest known medical antiglaucoma treatment. As the name implies, these drugs mimic the effects of acetylcholine, the neurotransmitter released from postganglionic parasympathetic neurones. Parasympathomimetic drugs used in the treatment of glaucoma are applied topically because of their effect on the ciliary body and iris.

The only parasympathetic agent available in the UK is pilocarpine, which is available in a number of

preparations including unpreserved as Minims Pilocarpine 2% and in long-lasting gel form as Pilogel 4%. The typical dose regime is four-times daily, with the exception of Pilogel, which is only used at bedtime.

Pilocarpine stimulates the muscarinic receptors of the ciliary body longitudinal muscle fibres. Ciliary body contraction puts traction on the scleral spur (the anterior insertion of the ciliary body) and the trabecular meshwork, preventing the canal of Schlemm from collapse. This increases aqueous outflow facility through the trabecular route. Pilocarpine reduces outflow through the uveoscleral route by obliterating the spaces between muscle fibre bundles and so the net IOP reduction represents a predominance of the trabecular outflow over uveoscleral outflow<sup>12</sup>. Pilocarpine also stimulates pupil miosis because of its effect on the iris sphincter muscle. This effect has no influence on reducing the IOP and is merely an undesirable side effect of the drug.

Pilocarpine's main use is in the treatment of acute-angle closure and to prepare an eye for laser peripheral iridotomy (LPI). In these cases, the effect of pupil miosis tightens the iris and pulls the peripheral iris away from the trabecular meshwork. If the IOP is especially high, the iris sphincter becomes ischaemic and unresponsive to cholinergic stimulation. Other topical and systemic medications are thus required to reduce the IOP and clear any corneal oedema before miosis can successfully be achieved.

Pilocarpine is no longer used in modern open-angle glaucoma management, principally because of the undesirable side-effects of pupil miosis, which can cause reduced vision particularly in elderly patients with cataract; ciliary spasm, which may cause eye-, brow- and head-ache; induced myopia in phakic patients, and the high-dosage regime of four-times daily.

## The role of the optometrist in patient management

Optometrists have a key role in the management of patients using glaucoma medications. This relates specifically to the areas of adherence to treatment and identifying potential adverse reactions to treatment, both ocular and systemic.

### Adherence

Adherence is defined as:

*“the regular use and correct administration of medication as prescribed by health-care professionals.”<sup>13</sup>*

This denotes an active participation by the patient to achieve a common therapeutic goal for both them and the healthcare practitioner. Persistence describes the period of time when there is consistent use of the prescribed treatment. Glaucoma is predominantly an asymptomatic, chronic condition in which poor adherence and persistence are factors in disease progression. Estimates suggest a level of adherence and persistence of 15-58% 3 years after the initiation of therapy<sup>14</sup>.

There are many factors that influence adherence and persistence including the capacity of the patient to follow their treatment plan. It is thought that as many as 64% of people in care homes have some form of dementia<sup>15</sup>. These individuals are invariably elderly and may have other conditions that make it difficult for them to instil their eye drops e.g. arthritis. Often the responsibility for instilling the eye drops will fall on the carers at the home. The optometrist should enquire about a patient's medications, and specifically enquire as to how and when any eye drops are used. Patients will often have a drugs chart detailing their medications, so it can be helpful to see this to determine which eye drops are being used. It can also be helpful to ask the carers to check that the drops are being used as advised. One should also enquire as to when the patient last attended for a glaucoma follow-up and when the next one is scheduled for. Unfortunately, follow-up appointments can often be cancelled and delayed for numerous reasons, so that a 6 month follow-up becomes 9 months and so on. Sometimes patients are lost to follow-up, either due to periods of hospitalisation, moving home, or other life events. If it appears that a patient has been lost to follow-up, the optometrist should write to the patient's GP to request that the patient is reviewed again at the local eye department.

### Side-effects

As discussed earlier, all of the available medical treatments have the potential to cause systemic and ocular side effects, of which the beta-blockers have the greatest potential to cause serious systemic side effects. Optometrists should, therefore, enquire specifically about problems with breathing, wheeziness, tightness of the chest, episodes of fainting (syncope) and extreme tiredness (all of which can be symptoms of pulmonary or cardiac compromise) especially if the patient has recently started taking these drops. If there is any doubt it would be quite reasonable to advise the patient to stop taking the beta-blocker eye drops and write a letter to the GP.

In the author's experience, adverse ocular reactions are most likely to occur with the adrenergic-agonist

brimonidine, the CAI Azopt, and the PGAs bimatoprost and travoprost.

“Alphagan allergy” is fairly unmistakable; the patient will often complain of very sore, red, angry-looking eyes, which make the patient feel utterly miserable. There will often be a follicular reaction in the inferior tarsal conjunctiva. The lid margins may display blepharitis-changes including thickening and telangiectasia. Although referred to as an allergy, this is actually a delayed hypersensitivity reaction, and it is not uncommon for patients to have been using brimonidine for months or even years when the reaction develops.

The CAI brinzolamide (Azopt) can frequently be a cause of irritation to patients. This eye drop is a creamy white suspension, which can leave a dry white residue along the eyelids and lashes. Not only is this unsightly, but also it can cause blepharitis and contact dermatitis. If seen, it is worth advising the patient or their carer to gently wipe this residue away with a wet face cloth or moistened cotton-wool ball/pad.

Bimatoprost and travoprost frequently cause symptoms of red-eyes, foreign body sensation, and periorbital skin

changes. If forewarned, most patients can accept these changes, but many can find it difficult to tolerate them. Again, if one suspects an intolerance to these medications, it would be wise to inform the GP.

### Summary

Optometrists are increasingly likely to encounter patients taking glaucoma medications. All of the glaucoma medications currently available have the potential to cause undesirable side effects, ranging from benign to potentially life-threatening. Elderly patients and in particular those with dementia may have difficulty adhering to their glaucoma treatment. As optometrists we are uniquely placed to ensure these vulnerable patients are receiving their glaucoma medication, reinforce the importance of adherence to patients and their carers, enquire about when their next glaucoma follow-up is due, and importantly be able to spot when undesirable side effects are occurring. If one suspects that a patient is suffering ill-effects from their glaucoma medication, the optometrist should notify the patient's GP so that treatment can be reviewed.

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## Multiple Choice Questions

**Question 1: With respect to IOP, which of the following statements is true?**

- A. High IOP always leads to glaucoma
- B. People with normal IOP do not develop glaucoma
- C. Results from landmark glaucoma studies show that reducing IOP can reduce the risk of developing glaucoma, and prevent or slow the rate of progression in established glaucoma
- D. IOP is mainly controlled by surgery

**Question 2: Which of the following statements about aqueous humour is true?**

- A. Aqueous humour production is a sympathetic,  $\beta$ -mediated, active secretory process
- B. Aqueous humour is produced by the choroid
- C. Aqueous humour drainage occurs only through the trabecular meshwork
- D. Aqueous humour is a physiologically unimportant metabolic by product

**Question 3: With respect to topical hypotensive drugs, which of the following statements is true?**

- A. Brinzolamide is a first-line treatment for glaucoma
- B. Beta-blockers frequently cause local side-effects but rarely cause systemic problems
- C. Prostaglandin analogues are the mainstay of modern glaucoma therapy
- D. All hypotensive eye drops reduce IOP by reducing aqueous synthesis

**Question 4: Which of the following statements is false?**

- A. The development of timolol in the 1970s established beta-blockers as effective IOP-lowering drugs

- B. Prostaglandin analogues are recommended by NICE as a first-line treatment for glaucoma
- C. Carbonic anhydrase inhibitors have a relatively weak IOP-lowering effect when compared to prostaglandin analogues
- D. Pilocarpine lowers IOP by causing pupil miosis

**Question 5: With respect to side effects of topical hypotensive drugs, which of the following statements is true?**

- A. Topical hypotensive eye drops rarely cause side-effects
- B. Brimonidine can cause a delayed hypersensitivity reaction, even after several years of use
- C. Prostaglandin analogues are not suitable for people with a history of cardiovascular or respiratory disease
- D. Beta-blockers can cause increased heart-rate, hypertension, and bronchial muscle dilation

**Question 6: Which of the following statements best describes the role of the optometrist in patient management?**

- A. The patient is being looked after by the ophthalmologists so optometrists have no role to play
- B. Optometrists should recommend changing to a different eye drop when they find the IOP to be raised
- C. The optometrist is unlikely to see many patients using topical hypotensive eye drops
- D. Optometrists are ideally placed to check adherence to treatment, identify potential side-effects to treatment, and to alert the patient's GP and/or ophthalmologist when they are concerned about the possibility of undesirable side-effects

To receive your CET point for this article, complete the  
Multiple Choice Questions  
A pass mark of 66% (4 out of 6 correct answers) must be achieved  
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