

Intraocular pressure

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Key points

Intraocular pressure (IOP) is normally regulated by changes in the volume of the aqueous humour.

Acute increases in IOP are caused by increases in episcleral venous pressure, determined by CVP.

Avoidance of coughing, straining and vomiting is important in preventing acute increases in IOP.

Prevention of the hypertensive response to intubation and extubation is important if control of IOP is required.

Acetazolamide can be used to reduce an acutely raised IOP.

The tissue pressure of the intraocular contents is called the intraocular pressure (IOP). The normal range for IOP is 10–20 mm Hg and is maintained at this level throughout life and between the sexes, though there is some diurnal and seasonal variation. Control of IOP within the correct physiological range is necessary to maintain the anatomical conditions necessary for optimal refraction and thus vision.

The importance of IOP for anaesthetists is that:

- (i) patients with acutely or chronically raised IOP may present for corrective surgery;
- (ii) patients with chronically raised IOP present for non-ophthalmic surgery;
- (iii) patients present with open globes following penetrating eye injuries;
- (iv) several drugs and procedures used in anaesthesia affect the IOP.

An acutely raised IOP may cause expulsion of the globe contents through a surgical or traumatic opening, or may lead to retinal artery occlusion and retinal ischaemia. In the chronic setting, raised IOP may cause nerve damage at the head of the optic nerve leading to visual field loss. This may be due to a direct effect of the raised pressure upon the nerves, or the effect of chronic under-perfusion of the nerve head.

This article will focus on the factors that determine the level of IOP, its regulation, how acute elevation of IOP can be prevented and its measurement. The effect of anaesthetic drugs on the eye and IOP has been discussed previously in this journal.¹

Determinants of intraocular pressure

The orbital globe is essentially a non-compliant sphere within a rigid box. Therefore, IOP can be influenced by a change in volume of the contents of the orbit or by external pressure (Table 1).

Normal regulation of IOP occurs chiefly through the regulation of the volume of the aqueous humour in the anterior chamber of the

eye. This will compensate to some degree for an increase in pressure due to expansion of other orbital elements; however, this may take as long as 15–30 min to occur. The vitreous humour in the posterior chamber has a relatively fixed volume and is not involved in IOP regulation.

Aqueous humour dynamics

Aqueous humour is produced in the ciliary bodies to supply oxygen and glucose to the avascular lens and cornea. Production is predominantly by active secretion mechanisms (80%); the Na⁺K⁺ATPase enzyme creating an osmotic gradient for the passage of water into the posterior chamber. This pathway is independent of IOP, though production may be reduced by a fall in the blood flow to the ciliary body. A more minor pathway for humour production (20%) is through ultrafiltration of the plasma. The rate of filtration is influenced by the blood pressure in the ciliary body capillaries, plasma oncotic pressure and IOP.

The aqueous humour produced flows from the posterior chamber over the lens surface, through the iris and into the anterior chamber where it is removed by two mechanisms. The bulk of resorption occurs through the trabecular network and canal of Schlemm in the angle between the cornea and the iris. Aqueous humour passes through the progressively smaller pores that make up the trabecular network and through the cells lining the wall of the canal. The canal communicates directly with the episcleral veins and absorption through this route is thus dependent upon the gradient of the IOP to episcleral venous pressure. Around 20% of resorption occurs through the uveoscleral route, which is the reverse of ultrafiltration, relying on the pressure gradient from the anterior chamber (IOP) to the interstitium of the sclera (Fig. 1).

The implications of the above are:

- (i) a rise in IOP will be compensated to some degree by an increased rate of aqueous humour drainage;

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Table 1 Anatomical and pathological features of the orbit that influence intraocular pressure

Intraglobal	
Aqueous humour volume	
Blood volume	
Foreign bodies	
Sulphur hexafluoride or carbon octafluoride bubble	
Tumours	
Haemorrhage	
Vitreous humour volume	
Scleral rigidity	
Extraglobal	
Anaesthetic regional blocks	
Extraocular compression devices	
Honan balloon	
Extraocular muscle tone	
Scleral strapping (for retinal detachment)	
Retrobulbar or peribulbar	
Haematoma	
Abscess	
Tumour	
Face mask	
Prone positioning	

- (ii) aqueous humour production is largely constant. When the capacity of the trabecular drainage system is reduced (e.g. glaucoma) or the episcleral venous pressure is raised (e.g. a rise in central venous pressure), IOP will rise.

Blood volume

The blood supply to the vitreous chamber in the posterior section of the eye is formed from the retinal arteries and veins on the surface of the retina, the choroidal arteries and veins and their chorioplexus lying beneath. A change in the volume of blood in the eyeball would lead to a change in IOP and a pressure wave of 1 mm Hg amplitude is seen with arterial pulsation.

Several factors affect the blood flow and therefore blood volume of the eye. These are the same factors which affect cerebral blood flow (Fig. 2). Whilst the retinal artery will only dilate

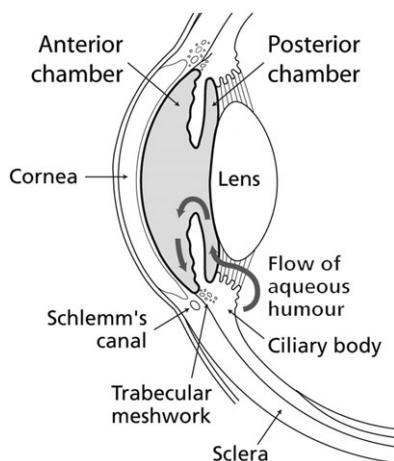


Fig 1. Production and flow of aqueous humour in the eye.

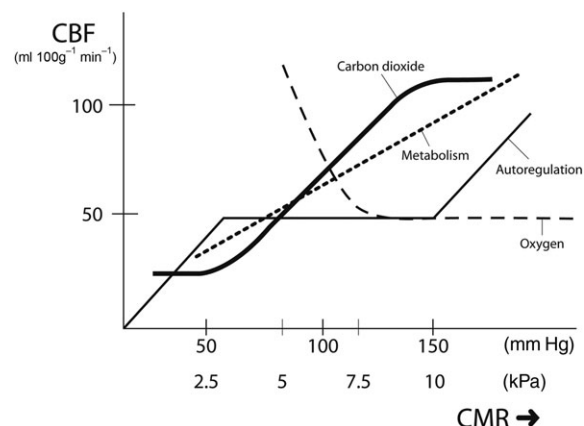


Fig 2. Factors affecting cerebral blood flow.

if the blood pressure is significantly raised, the choroidal vessels do not have myogenic autoregulation and will dilate in response to a raised perfusion pressure. Vasodilatation occurs with hypoxaemia, hypercarbia, and an increase in metabolic rate. However, these effects are small compared with the effect that a raised venous pressure has on blood volume. The normal venous pressure within the globe is only just above IOP (15 mm Hg), so if episcleral venous pressure outside the globe rises, the pressure gradient draining the choroidal venous plexuses falls and blood pools within the orbit.

Foreign bodies

Addition of sulphur hexafluoride or carbon octafluoride to the vitreous chamber following vitreoretinal surgery may increase IOP. Expansion of this gas due to an increase in altitude, a lowering of ambient pressure, or exposure to nitrous oxide (which will diffuse into the bubble faster than inert insoluble gasses will leave it) will cause an increase in IOP. Carbon octafluoride may remain in the globe for 70 days after insertion and there have been numerous accounts of visual loss following inadvertent use of nitrous oxide during this period.² It has been proposed that patients with intraocular gas bubbles should wear a warning bracelet until the ophthalmologist has confirmed absorption of the bubble.

Other intraglobal masses

Intraorbital and extraorbital bleeds are usually of insufficient volume to lead to raised IOP. However, debris or blood in the anterior chamber may lead to a blockage of aqueous humour drainage and an acute glaucoma.

Extraglobal

Anaesthetic blocks

The introduction of several millilitres of local anaesthetic into the orbit would be expected to lead to a rise in IOP. Indeed, a rise in

IOP has been demonstrated following peribulbar anaesthesia.³ However, a reduction in IOP has been shown following sub-Tenon blocks, possibly due to a reduction in muscle tone.⁴

Ocular compression devices

The role of ocular compression devices such as the Honan balloon is contentious. The application of such a device may improve surgical conditions by a reduction in chemosis, lid swelling, and bleeding. It may also aid spread of the local anaesthetic. However, the value of the balloon in the reduction of IOP has been questioned due to an initial rise in pressure whilst the balloon is *in situ*.⁵

Extraocular muscle tone

Contraction of the extraocular muscles leads to an increase in IOP. This is evidenced by the increase in IOP observed when eye movement is opposed by traction by the surgeon. Forceful contraction of the orbicularis oculi can increase IOP to >50 mm Hg; even normal blinking increases IOP by 10 mm Hg.⁶

It has previously been proposed that succinylcholine raises IOP by its depolarization and contraction of the extraocular muscles. However, the rise in IOP has been demonstrated experimentally even when the muscle insertions have been released.⁷

Avoiding a raised intraocular pressure

Of all the factors detailed, the most important in determining IOP acutely is the episcleral venous pressure, which is determined by CVP. A raised episcleral venous pressure would lead to vitreous chamber venous engorgement and a reduction in aqueous humour drainage, both of which will raise IOP. Therefore, the use of the reverse Trendelenberg position and the avoidance of venous congestion caused by neck positioning or tube ties around the neck are important factors in controlling IOP.

Coughing, straining, and vomiting can lead to an increase in IOP of 30–40 mm Hg. Laryngoscopy and intubation lead to a rise of 10–20 mm Hg and this may be prevented by avoiding the hypertensive response to intubation and extubation. This may be achieved by using a laryngeal mask, propofol, deep extubation or by covering intubation with lidocaine, clonidine, β -blockers or high-dose opioids.

Inadvertent external pressure on the eye is an important and largely avoidable cause of postoperative blindness. Prone and lateral positionings both carry an increased risk, though visual loss may be multifactorial in some cases.⁸ For example, visual loss may be due to a combination of ischaemic optic neuropathy caused by hypotension, and raised IOP due to the venous engorgement that both accompany prone positioning.

It is worth emphasizing that patients with a chronically raised IOP due to a reduced capacity for aqueous humour drainage (chronic open angle glaucoma) have a reduced capacity to compensate for an acute rise and are therefore at an increased risk of having a marked rise in IOP.

Reducing intraocular pressure

Pharmacological treatments to reduce the IOP are shown in Table 2. Systemic absorption of topical glaucoma medications such as the sympathomimetic drugs or β -adrenoceptor antagonists can have rapid and profound effects on the cardiovascular system due to the lack of first-pass liver metabolism when absorbed via the conjunctiva or nasal mucosa.

If there is an acute elevation of IOP during surgery, detected by either protrusion of the orbital contents or by palpation of the globe, the IOP may be rapidly reduced by the use of intravenous acetazolamide or possibly mannitol. A head-up tilt, prevention of venous congestion and mild hypocapnia may be used in addition, if appropriate.

Effect of general anaesthesia on intraocular pressure

The effect of anaesthetic drugs on IOP has been reviewed in this journal.¹ In summary, all induction agents (apart from ketamine) and all inhalational anaesthetic agents reduce IOP. This fall in IOP is independent of their effect on blood pressure, central venous pressure and extraocular muscle tone and is more likely to be a direct action on central control mechanisms. Opioids have no direct effect on IOP, but attenuate the elevation in pressure due to intubation. Non-depolarizing muscle relaxants have a minimal effect on IOP. Succinylcholine leads to an increase in IOP of up to 10 mm Hg for 10 min but, as has been debated frequently, it is also the drug of choice to provide rapid, short acting and ideal intubation conditions in an emergency situation where there is a risk of aspiration. The balance of airway risks *vs* eye risks should be weighed up in each individual case.

General anaesthesia enables easier manipulation of physiological factors important in the control of IOP, such as Pa_{O_2} and Pa_{CO_2} . Anaesthesia for a patient with an open eye should include consideration of the following factors:

- (i) a smooth induction with muscle relaxation (as determined by the risk of losing the airway);

Table 2 Therapeutic intraocular pressure reduction

Intravenous	
Acetazolamide	Carbonic anhydrase inhibition leads to a reduction in aqueous humour production
Mannitol	Osmotic diuretic dehydrates the vitreous chamber
Topical	
Parasympathomimetics	
Cholinergic and anticholinesterase medication contract the ciliary body and increase aqueous humour drainage through the trabecular network	
Sympathomimetics	
Epinephrine reduces aqueous humour production and increases drainage, possibly through ciliary body vasoconstriction and adenylate cyclase inhibition	
β -adrenoceptor antagonists	
Timolol reduces aqueous humour production through adenylate cyclase inhibition	
Prostaglandin analogues	
Increase aqueous humour drainage via uveoscleral route	

- (ii) intubation or LMA placement with care to avoid coughing and the hypertensive response to intubation;
- (iii) ventilation to control Pa_{O_2} and Pa_{CO_2} ;
- (iv) head up tilt with no obstruction to venous drainage by the tube tie;
- (v) smooth extubation with consideration of changing an endotracheal tube to a LMA prior to reversal to minimize the risk of coughing;
- (vi) meticulous avoidance of postoperative nausea and vomiting.

Measurement of intraocular pressure

The gold standard for measurement of IOP is the Goldmann tonometer. This is an applanation tonometer, measuring the force necessary to flatten the area of the surface of the cornea. However, this requires use of a slit lamp, fluorescein dye and topical anaesthesia to the cornea. A portable version is available (Perkins) but more commonly seen is the 'air-puff' tonometer. This device forces a burst of air onto the cornea until it is flat and reflects an emitted light beam. No topical anaesthesia is required.

An alternative method is indentation tonometry, relying on the measurement of the indentation of the cornea by a known weight. However, this is subject to error by variations in the rigidity and thickness of the sclera. An advantage of the use of indentation tonometry is

that, because the application of the weight will lead to an increase in IOP, the capacity for a compensatory increase in aqueous humour drainage can be measured by using a single prolonged measurement.

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Please see multiple choice questions 18–21