Review Article

Intraocular pressure - physiology and implications for anaesthetic management

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Introduction

Over the past two decades the pattern of ophthalmic surgery has changed dramatically. The rapid evolution of technology now permits a level of controlled intraocular manipulation inconceivable 20 years ago.[1]

Key words

EYE: intraocular pressure; ANAESTHESIA: ophthalmic.

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Improved operating microscopes, suture materials and intraocular lenses have revolutionised cataract surgery. Correcting aphakia with intraocular lenses overcomes the problem of unilateral cataract and leads to cataract surgery at an earlier stage of visual handicap. Of the estimated 640,000 cataract operations performed in the United States in 1982, more than 70 per cent involved intraocular lens implantation.[2] Furthermore, 57 per cent of all current intraocular lens implants are posterior chamber lenses. This implant requires a microsurgical dissection of the cataract and is a major shift from traditional cryo extraction.

Intraocular gas tamponade has added a new dimension to conventional retinal detachment surgery. Vitrectomy, a closed intraocular microsurgical procedure, now permits definitive restoration of vision in eyes hitherto considered blind by the ravages of trauma, diabetes and complicated retinal detachment.[3] Such closed intraocular microsurgery is made possible by the development of controlled intraocular infusion, mechanical suction-cutters, intraocular fibre optic illumination, scissors, foreign body forceps and laser endo-photocoagulating probes.

The increased use and safety of general anaesthesia is a major contribution factor to these changing patterns.

Ophthalmic surgery may be simply classified as extraocular, intraocular of mixed (Table I). For a purely extraocular procedure such as strabismus correction, intraocular pressure has no impact on the conduct of surgery. During vitrectomy, a closed intraocular procedure, the ophthalmic surgeon con-

TABLE I Ophthalmic surgery

Extraocular	
Closed	e.g. Strabismus
Intraocular	
Closed	e.g. Vitrectomy
Open	e.g. Intracapsular cataract extraction ± intraocular lens Glaucoma surgery
Mixed	e.g. Extracapsular cataract extraction ± intraocular lens Drainage subretinal fluid ± gas tamponade
Penetrating trauma	

trols intraocular pressure manometrically. This is achieved by a watertight infusion entry site through the pars plana. Additional water-tight pars plana entry sites permit the introduction of illuminating suction and cutting instruments. Minimal choroidal blood flow is required for the safe introduction of all such instruments into the vitreous cavity. Conversely, control of intraocular pressure is crucial for open ophthalmic procedures such as traditional intracapsular cataract extraction or drainage operations for glaucoma. These procedures, and the repair of penetrating eye injuries, present special challenges to the anaesthetist and proper anaesthetic management contributes significantly to a successful surgical outcome. The prime anaesthetic considerations in the mangement of such cases include[4] a stationary eye with extraocular muscle akinesis; airway control with adequate alveolar ventilation; cardiovascular stability with avoidance of stimuli likely to raise central venous pressure; controlled intraocular pressure before, during and after surgery. When the sclera is surgically incised the intraocular pressure approximates atmospheric pressure. Prior to surgical incision a low-normal intraocular pressure is desirable. Sudden decompression of a hypertensive eye may be catastrophic with iris or lens prolapse, vitreous loss or expulsive choroidal haemorrhage.[5]

TABLE II Intraocular pressure – Summary of physiological factors

Aqueous humour fluid dynamics
Production
Drainage
Choroidal blood volume
Autoregulation
Central venous pressure
Chemical – $PaCO_2$ PaO_2
Metabolic – pH
Vitreous humour volume
Osmotic pressure
Extraocular muscle tone
Neurogenic control

This review will outline the physiological factors involved in the regulation of intraocular pressure; the effects of drugs administered during anaesthesia on intraocular pressure; the anaesthetic management of patients for elective intraocular procedures; the anaesthetic management of emergency penetrating eye trauma.

Intraocular pressure physiology

Intraocular pressure is normally 16 ± 5 mmHg, with a value in excess of 25 mmHg considered pathological.[6] Intraocular pressure must be maintained within this normal range to ensure constant corneal curvature and a proper refracting index of the eye. This pressure is determined by the volume of aqueous humour, vitreous and blood within the eye exerting an outward pressure, scleral compliance and extraocular muscle tone exerting inward pressure. Some pressure sensitive feedback control system may be operative to maintain constant intraocular pressure. However, the presence of such a system in the eye or central nervous system is purely speculative. The major controlling influence on intraocular pressure is the dynamic balance between aqueous humour production in the ciliary body and its elimination via the canal of Schlemm.

Several minor diurnal fluctuations are observed in normal intraocular pressure recordings. A diurnal variation of 2–3 mmHg is observed with the higher recordings noted in early morning.[7] This increase may reflect circadian adrenocortical steroid secretion, pupillary dilation during sleep, carbon dioxide retention, an immobile eye and the recumbent position. A deep inspiration may reduce intraocular pressure by up to 5 mmHg, while blood pressure oscillation, changes in body position and plasma osmotic pressure changes account for the minor fluctuations observed.[8]

The three major categories of factors affecting intraocular pressure during surgery are (Table II):

1 Aqueous humour fluid dynamics
2 Choroidal blood volume
3 Vitreous volume and extraocular muscle tone

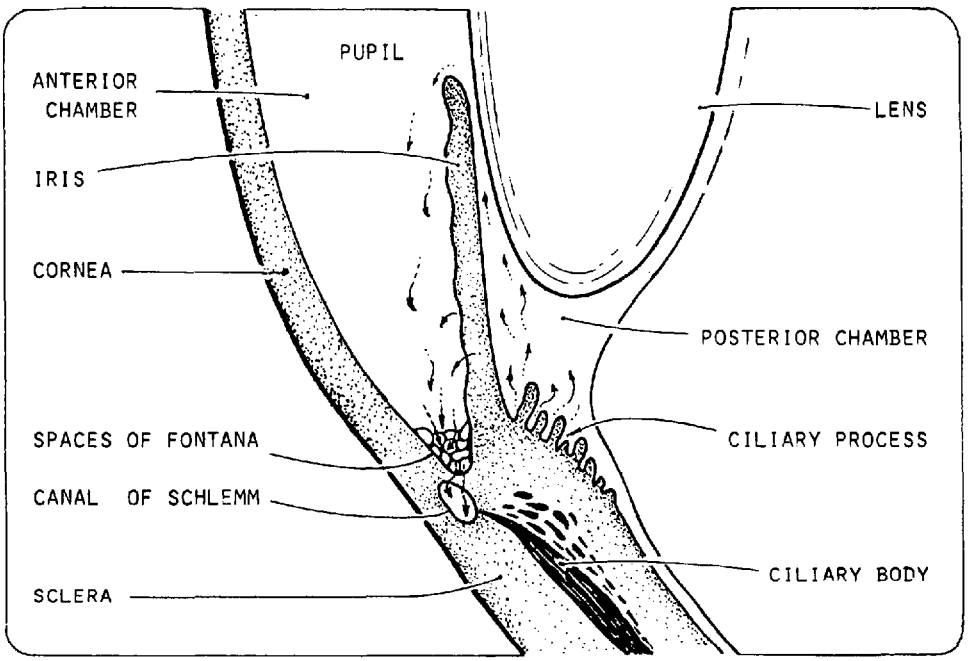

FIGURE 1 Illustration of sites of formation, circulation and drainage of aqueous humour.

Aqueous humour fluid dynamics

The balance between aqueous humour production and drainage is the primary physiological control mechanism regulating intraocular pressure. IOP rise may be associated with increased aqueous production, impaired drainage or both.

Aqueous humour is a clear fluid with a pH 7.1–7.2, a viscosity of 1.025–1.040 relative to water and low protein, urea and glucose content. Aqueous occupies the anterior and posterior chambers of the eye – the total volume is approximately 0.3 ml with 0.25 ml in the anterior chamber and the remaining 0.05 ml in the posterior chamber.

Two-thirds of the aqueous is formed in the posterior chamber by the epithelial cells of the ciliary body[8] in an active secretory process utilizing carbonic anhydrase and cytochrome oxidase (Figure 1). The remaining one-third is formed in the anterior chamber by simple filtration through the anterior surface of the iris. The aqueous produced in the posterior chamber circulates through the pupil, enters the anterior chamber, bathes the lens and cornea and exits via the trabecular meshwork in the angle of the anterior chamber between the peripheral cornea and iris. Having passed through Fontana's trabecular spaces, the aqueous enters the venous system following its passage through the canal of Schlemm. The fluid enters the aqueous veins in the episcleral tissue and drains into the orbital venous system and eventually the aqueous reaches the cavernous sinus via the superior and inferior ophthalmic veins (Figure 2). Ultimately, via the internal and external jugular venous ystems, the aqueous drains into the right atrium.

Interference with aqueous drainage through Fontana's spaces has a major impact on intraocular pressure. The outflow of aqueous through the canal of Schlemm is governed by Hagen-Poiseuille's Law[9] (Table III). The most important factor regulating aqueous outflow is the cross-sectional area of Fontana's space. When the space narrows the resistance to flow increases and a marked rise in intraocular pressure follows. By contracting the ciliary muscles and opening the trabecular mesh-

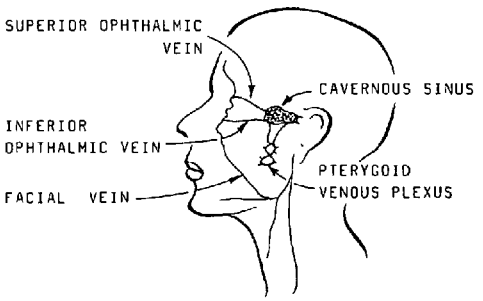

FIGURE 2 Aqueous and venous drainage of the eye.

work, miotics may improve aqueous drainage and reduce intraocular pressure.[10]

Choroidal blood volume

The choroid is a highly vascular area in which there are extensive anastomoses between the anterior ciliary arteries and the long and short posterior ciliary arteries. The choriocapillaris has the ability to locally auto-regulate so that choroidal blood flow remains constant through a range of perfusion pressures.[11] A sudden increase in systolic blood pressure produces a transient acute rise in IOP until the aqueous outflow accommodates the rise. Moderate decreases in arterial pressure have little effect on IOP but below mean pressures of 90 mmHg marked reductions in IOP occur.[12,13] At systolic pressures of 50–60 mmHg in primates intraocular pressure approaches zero, in part due to reduced choroidal blood volume and cessation of aqueous production (Figure 3). Choroidal blood volume may vary considerably. During surgery, increased choroidal blood volume may displace the posterior vitreous, forcing it forward into the surgical field when the anterior chamber is open or dramatically increasing intraocular pressure when the globe is intact.

TABLE III Factors affecting aqueous drainage (Hagen-Poiseuille Law)

$$Q = \frac{(P_{IOP} - Pv) \times r^4}{8 \times n \times L}$$

Q = Aqueous flow ml/unit time
P_{IOP} = Intraocular pressure
Pv = Venous pressure
r = Radius of Fontana's space
n = Aqueous viscosity
L = Length of Fontana's space

The venous drainage from the iris, ciliary body and choroid enters the four vortex veins which pass through the sclera behind the equator and joins the venous plexus of the orbit en route to the cavernous sinus. Obstruction of central venous return or increased central venous pressure associated with coughing, vomiting, Valsalva manoeuvre or straining on an endotracheal tube cause an immediate sharp rise in choroidal blood volume and intraocular pressure. By rapidly increasing central venous pressure, a cough may raise intraocular pressure by 34–40 mmHg.[14] The choroidal blood volume is sensitive to changes in arterial PCO_2.[15] Using $Xenon^{-133}$ clearance in baboon studies, Wilson and Stang[16] noted a linear increase in choroidal blood volume when $PaCO_2$ increased from 4.5 to 9.1 KPa. The effects of hyper- and hypocapnia have been well described in dogs,[17] monkeys[18] and man.[19] Respiratory acidosis, by increasing choroidal blood volume and aqueous production and decreasing aqueous drainage, produces a linear relationship with intraocular pressure. Respiratory alkalosis lowers intraocular pressure by reducing choroidal blood volume; by reducing blood flow to the ciliary processes; by inhibiting carbonic anhydrase and by decreasing ultra filtration of aqueous humour. Changes of pH due to metabolic acidosis and alkalosis have opposite effects on intraocular pressure compared to the corresponding respiratory pH changes. Metabolic acidosis of diabetic,[20] exercise[21] or drug-induced[22] origin is associated with a reduction in intraocular pressure while metabolic alkalosis secondary to sodium bicarbonate treatment increases IOP. The choroidal circulation is sensitive to arterial PaO_2 changes. Hyperbaric oxygen tensions[23] are associated with profound choroidal vasoconstriction and a reduction in IOP. Hypoxaemia may induce choroidal vasodilatation and elevate IOP. Hypothermia,[8] despite increasing aqueous viscosity, may induce significant reductions in intraocular pressure due to decreased aqueous production and associated vasoconstriction.

Extraocular muscle tone and vitreous humour volume

The central nervous system,[6] directly through its

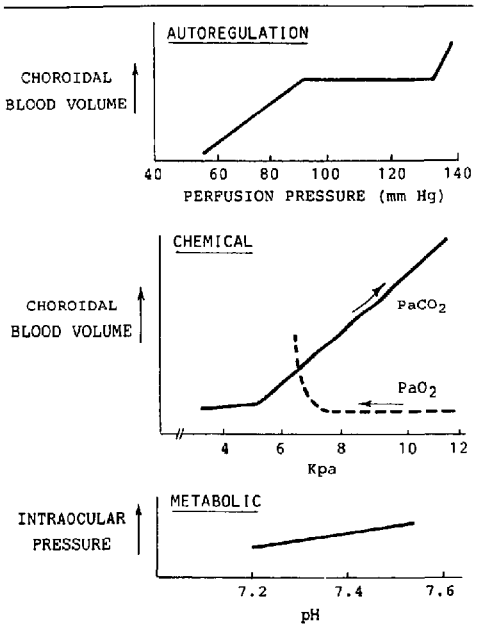

FIGURE 3 Factors influencing choroidal blood volume and intraocular pressure.

neurogenic control of extraocular muscle tone and indirectly through hormonal and haemodynamic effects, exerts a central controlling influence over intraocular pressure, so the eye can respond to the needs of the moment. Von Sallman and Lowenstein,[24] in elaborate stereotaxic experiments, isolated numerous points in the cat diencephalon where electrical stimulation produced variations in intraocular pressure. Some responses appeared neurovascular in nature while other eye pressure responses seemed predominantly mediated by contractions of smooth or striated extraocular muscles. Stimulation of a ventral zone in the hypothalamus produced effects analogous to a diffuse sympathetic nervous system discharge. Intraocular pressure changes coincided with changes in systemic blood pressure. General anaesthesia lowers IOP, partly by depressing these centres. Intraocular pressure may rise markedly following pressure on the eye, contraction or contracture of the extraocular muscles, contraction of the orbicularis oculi muscle, eyelid closure and venous congestion of the orbital veins.

The vitreous is an unstable gel, consisting mainly of water with a fine fibrillar supporting structure. The volume of vitreous and its pressure effect may be reduced by dehydrating the vitreous.[25] Urea 30 per cent solution in water, $1.5\ g \cdot kg^{-1}$ of 20 per cent mannitol IV infusion or oral glycerol $2\ g \cdot kg^{-1}$ may produce a dramatic reduction in intraocular pressure and relief of symptoms in acute angle closure glaucoma.

Retinal detachment surgery involves procedures affecting intraocular volume, often utilizing a synthetic silicone strap or sponge to produce a localized or encircling scleral indentation. Internal tamponade of the retinal break may be achieved by injecting the expandable gas sulphur hexafluoride into the vitreous or alternatively the vitreous substitute silicone oil may be injected. Because of the blood gas partition coefficient differences, the administration of nitrous oxide may enhance the internal tamponade effect of sulphur hexafluoride during surgery only to be followed by a marked drop in intraocular pressure and volume on discontinuing nitrous oxide. SF_6, with an Ostwald solubility coefficient of 0.0044, is exceedingly insoluble in water.

Nitrous oxide is 117 times more soluble than SF_6. When SF_6 is injected into the vitreous cavity, the more soluble nitrogen and nitrous oxide dissolved in the surrounding blood and tissues diffuses into the bubble faster than SF_6 diffuses out. Stinson and Donlon,[26] using a mathematical model, predicted a rapid almost threefold increase in volume of the injected bubble if 70 per cent nitrous oxide is administered following intravitreal injection, with only minor increases if nitrous oxide is discontinued prior to intravitreal injection. Wolf *et al.*[27] noted significant intraocular pressure increases when nitrous oxide was added following SF_6 intravitreal injection.

Measurement of intraocular pressure

Intraocular pressure may be measured manometrically or tonometrically. Direct or manometric measurement involves cannulation of the anterior chamber and its use is confined to experimental studies. Indirect or tonometric measurements are employed in clinical studies.[28]

Applanation tonometry is based on the principle that the pressure in a fluid-filled sphere is equal to the force required to flatten part of the boundary membrane. The tonometer is in contact with the tear film and the area of applanation is visualized by

addition of fluorescein dye to the tear film. The Goldman tonometer flattens an area of 3.06 mm in diameter, displaces little aqueous and produces minimal changes in IOP. The Perkins applanation tonometer[29] is a portable hand held instrument incorporating the Goldman head and is the most commonly used instrument for measuring IOP during anaesthesia.

Schiotz[30] indentation tonometry is a simple indirect method of measuring intraocular pressure. The technique involves the use of weighted vertical plunger which indents the cornea. The extent of indentation for a given weight is inversely proportional to the intraocular pressure. A Friedenwald nomogram is used to convert pointer movement on a scale to mmHg. The Schiotz tonometer is less reliable because of wide variations in scleral rigidity and repeated measurements may displace aqueous and produce lower IOP readings.

A more recent development in indirect tonometry is the pneumatic or air tonometer[31] in which a jet of air at constant pressure from a fixed distance indents the cornea and a photocell measures the reflection of a light beam from the cornea. The air tonometer is not used extensively in anaesthesia–intraocular pressure studies because of the high cost and the patient's upright position.

Cooper *et al.*[32] have developed a method of non-invasive passive radiotelemetry to measure continuously intraocular pressure. An applanation transensor is mounted in the lower part of the scleral haptic so that it applanates the inferior sclera under the lower lid. This technique has not been employed extensively in clinical practice.

Anaesthetic drug effects on intraocular pressure

Central nervous system depressants, general anaesthetic agents and narcotics

Drugs administered to patients during anaesthesia may affect intraocular pressure directly through action on the central diencephalic control centres, through facilitation or inhibition of aqueous production and drainage, through relaxation or contraction of extraocular and orbicularis oculi muscles or indirectly through their effects on the cardiovascular or respiratory systems. Earlier studies of anaesthetic drug effects on intraocular pressure were characterized by failure of the authors to standardize study methods. The factors affecting intraocular pressure such as $PaCO_2$, systolic blood pressure and central venous pressure were not maintained constant while different patient populations and measurement techniques were used.

The earliest human studies on the effects of general anaesthesia on intraocular pressure[33] noted a dose-dependent reduction in intraocular pressure when diethyl ether, cyclopropane or vinyl ether was used. Subsequently chloroform, trichlorethylene and halothane were reported[34] to produce a comparable reduction in intraocular pressure.

In 1974, Al-Abrak and Samuel[35] attempted to standardize the anaesthetic technique, the physiological variables and the measurement methods for intraocular pressure studies and most subsequent investigations have followed these guidelines. A marked reduction in intraoocular pressure which was closely related to the inspired halothane concentration but independent of the decrease in arterial pressure, was noted. A reduction of 50 per cent from preoperative intraocular pressure was reported when a nitrous oxide:oxygen, 0.5 per cent halothane mixture was administered using controlled ventilation and normocapnic anaesthesia.[36] Trichloroethylene[37] when used for cataract patients in association with a normocapnic controlled ventilation technique was associated with a higher intraocular pressure than when halothane was used. Enflurane one per cent in a nitrous oxide:oxygen mixture in controlled ventilation, normocapnic situations consistently reduced intraocular pressure by approximately 35 per cent.[38] Runciman *et al.*[39] compared the effects of equal MAC equivalents of enflurane and halothane and found an IOP reduction of 40 per cent with enflurane compared with 14 per cent for halothane. Isoflurane significantly decreased intraocular pressure in unsedated children.[40]

The central nervous system depressants in general have been thought to lower intraocular pressure. Sedative doses of barbiturates lower IOP and thiopentone has been shown to reduce IOP in normal[41] and glaucomatous eyes.[42] Etomidate,[43] despite its tendency to produce pain and muscle movement on administration, is associated with a marked decrease in IOP whether given as a bolus dose or as total intravenous anaesthesia. Ketamine is a notable exception to the general tendency of general anaesthetic agents to lower intraocular pressure. In earlier studies, when given IV or IM to children, ketamine increased intraocular pres-

sure.[44] In more recent studies, ketamine when given following diazepam–meperidine premedication produced no change in intraocular pressure in adult patients[45] and a 25 per cent reduction in IOP when administered IM to children.[46] However, the associated nystagmus and blepharospasm makes ketamine an unsuitable induction agent for use in ophthalmic surgery. Neuroleptanalgesia, combining fentanyl and droperidol for induction of anaesthesia, was associated with a 12 per cent reduction in intraocular pressure in normocapnic patients.[47] Presbitero *et al*,[48] using Innovar neuroleptanaesthesia, noted an insignificant increase in intraocular pressure during stage I anaesthesia, followed by a significant intraocular pressure reduction during stages II and III.

The succinylcholine controversy

As early as 1955 Lincoff[50] reported both a transient but significant rise in intraocular pressure when succinylcholine was given intravenously to facilitate intubation and vitreous loss followed the administration of succinylcholine in the presence of an open eye. In a series of volunteers given succinylcholine without anaesthesia, intraocular pressure rose 18 mmHg.[51] Following induction of anaesthesia with thiopentone and succinylcholine, intraocular pressure increased within the first minute, with a peak rise of 6–8 mmHg between 2–4 minutes and by six minutes intraocular pressure had returned to control values.[52] Tracheal intubation following succinylcholine exaggerated the rise in intraocular pressure but did not prolong it. The intraocular pressure effect of succinylcholine was found by Joshi and Bruce to be dependent on the timing of the administration of succinylcholine and thiopentone. A 1.0 $mg \cdot kg^{-1}$dose given immediately following 3 $mg \cdot kg^{-1}$ thiopentone was associated with a reduced intraocular pressure while a 1.0 $mg \cdot kg^{-1}$ dose given two minutes after 3 $mg \cdot kg^{-1}$ thiopentone maintained constant intraocular pressure. Cook[53] found no succinylcholine dose–response effect when he observed similar increases in intraocular pressure when succinylcholine 1 $mg \cdot kg^{-1}$ and 2.5 $mg \cdot kg^{-1}$ were given following thiopentone 3 $mg \cdot kg^{-1}$.

The extraocular muscles contain unique histological structures with different physiological responses to acetylcholine.[54] In 1963 Hess and Pilar[55] demonstrated morphologically that the extraocular muscle of the cat had two types of muscle fibres that differed from other types of skeletal muscles. The Felderstruktur, with small grape-like nerve endings, respond with a slow tonic contraction when exposed to acetylcholine or depolarizing muscle relaxants. The Fibrillenstruktur, in contrast, with its plaque-like nerve endings, responds to acetylcholine and depolarizing relaxants by producing a twitch response.[56] Succinylcholine in increasing doses causes an increase in extraocular muscle tension by its effect on the tonic system. The twitch system is unaffected by low doses, increased by larger doses and inhibited by very large doses.[57] Section of the recti muscles fails to prevent a rise in IOP following succinylcholine administration. Part of the succinylcholine-induced rise in intraocular pressure may be due to distortion of the globe with axial shortening, choridal vascular dilatation or relaxation of orbital smooth muscle.[58]

Many techniques have been advocated during the past two decades to prevent the rise in intraocular pressure following succinylcholine administration. No reported method to date has been shown to consistently prevent the intraocular hypertensive response to intravenous succinylcholine administration. Carballo[59] advocated the preoperative administration of 500 mg IV acetazolamide, a carbonic anhydrase inhibitor. Subsequently such acetazolamide pretreatment has been shown to dramatically increase choroidal blood flow[16] so, by predilating the choroidal vessels, this pretreatment may mask the succinylcholine induced rise in IOP secondary to choroidal vasodilation. Contradictory claims have been made for many pretreatment regimes. Miller,[60] employing indentation tonometry, reported that d-tubocurarine and gallamine pretreatment regimes inhibited succinylcholine-induced rise in IOP. Myers[61] and Bowen,[62] using the more sensitive applanation tonometry techniques, were unable to confirm the efficacy of such pretreatment. Verma,[63] utilizing indentation tonometry, claimed that a 10 mg "self-taming" dose of succinylcholine prevented a subsequent rise in intraocular pressure when the full intubating dose of succinylcholine was administered. Myers[64] subsequently in a controlled study using applanation tonometry refuted Verma's claim. Cunningham[65,66] claimed that 0.1 $mg \cdot kg^{-1}$ diazepam pretreatment diminished the subsequent rise in intraocular pressure following succinylcholine and tracheal intubation while

Fennick[67] and Cook reported that 0.05 mg·kg^{-1} diazepam pretreatment failed to prevent succinylcholine-induced rise in IOP. Intravenous lidocaine 1–2 mg·kg^{-1} pretreatment may attenuate the haemodynamic response to laryngoscopy[68,69] but such pretreatment has been shown to be ineffective in preventing the rise in intraocular pressure following succinylcholine and/or intubation.[70]

The non-depolarizing muscle relaxants are associated with a reduction in intraocular pressure. D-tubocurarine may be associated with the greatest reduction in IOP[71] not only due to its relaxation of extraocular muscle tone but also due to its hypotensive and ganglion-blocking effects. Pancuronium in awake and anaesthetized patients[72] is associated with a reduction in intraocular pressure while metocurine and metocurine-pancuronium combination[73] are associated with a reduction in IOP below control values. The new non-depolarizing relaxants atracurium[74] and vecuronium have no significant effects on intraocular pressure. Maharaj *et al.*[75] studied the effects of atracurium 0.45 mg·kg^{-1} and pancuronium 0.1 mg·kg^{-1} administration following thiopentone induction and intubation without neuromuscular blockade in patients requiring surgery for ocular trauma. While no significant change in intraocular pressure was noted with either agent, greater cardiovascular stability followed atracurium administration.

Anaesthetic management of elective intraocular surgery

Modern ophthalmological surgery involves complex, delicate microscopic intraocular manipulation. Satisfactory surgical conditions demand a stationary eye, low–normal intraocular pressure, adequate depth of anaesthesia without carbon dioxide elevation, avoidance of episodes of coughing, straining, vomiting or other stimuli likely to raise central venous pressure, avoidance of the oculo-cardiac reflex, smooth induction and emergence from anaesthesia and minimal bleeding. The patient population presenting for such surgery is usually elderly with frequently associated cardiovascular, respiratory and metabolic disorders. Prior to the 1970's the majority of patients presenting for ocular surgery were managed with either local anaesthesia or local anaesthesia with sedation. Local anaesthesia involves facial and infra-orbital nerve block and retrobulbar block with the attendant hazards of retrobulbar haemorrhage and proptosis. Local anaesthesia and sedation with a variety of tranquillizers, narcotics and hypnotics may be the most unsatisfactory technique[76] because of the unpredictable pharmacologic effect in this age group; the hazards of respiratory depression and airway obstruction; the inconvenience of patient restlessness and confusion and the prolonged recovery time.

While available data[77] have not shown a major difference in complications such as vitreous loss and iris prolapse between local and general anaesthesia and while local anaesthesia has proved itself a safe technique for patients with cardiovascular disease,[78] interest in and utilization of general anaesthesia for intraocular surgery has increased dramatically over the past two decades.

With the exception of strabismus correction and some retinal detachment procedures, opthalmic surgery is associated with little patient discomfort. Narcotic premedication, with its potential emetic effects, is seldom necessary and is particularly unsuitable in the context of intraocular surgery. Premedication should be chosen for its sedative, hypnotic, amnesic and anti-emetic effects. A reasonable choice would include a benzodiazepine for its sedative-hypnotic effect; a butyrophenone for its anti-emetic effects; or the phenothiazine promethazine or the antihistamine hydroxyzine for their sedative and anti-emetic effects. The inclusion of the anti-cholinergic agents atropine, scopolamine or glycopyrrolate in the premedication regime for their anti-sialogogue and oculo-cardiac reflex prophylaxis is highly controversial.[79,80] Atropine, given systematically in the usual premedicating dose, has no effect on pupil size or intraocular pressure in normal eyes[81] and glaucoma patients.[82] The current recommended practice[83] is to administer atropine 0.01 mg·kg^{-1} body weight intravenously to children prior to surgery involving the extraocular muscles while adults may best be given atropine intravenously to treat an established oculocardiac reflex.[84]

Close monitoring of patients undergoing intraocular surgery is mandatory because of the lack of access to the patient's airway, patient draping and the position of the anaesthetist at the patient's side. Standard basic monitoring for intraocular surgery includes an exposed patient's hand to check colour, capillary filling and radial pulse palpation; an

intravenous line and blood pressure cuff on the arm accessible to the anaesthetist; electrocardiographic and temperature monitoring; neuromuscular function monitoring to ensure a 90–95 per cent twitch suppression level during surgery and continuous end-tidal CO_2 analysis. A 10–15 degree head up tilt promotes venous drainage and reduces intraocular pressure during intraocular surgery. Hvidberg *et al.*[85] noted simultaneous and parallel reductions in central venous pressure and intraocular pressure on changing from Trendelenburg to head-up positions. The use of an atropine/neostigmine combination has minimal effects on pupil size and intraocular pressure.[86] Techniques[36,38] utilizing normocapnic general anaesthesia and controlled ventilation, offer the benefits of low–normal intraocular pressure, cardiovascular stability and an early return to spontaneous ventilation at the end of surgery.

Duncalf in 1963[87] originally suggested that when the eye is to be surgically opened the intraocular pressure should be as low as possible. An excessive reduction in intraocular pressure due to high inspired concentration of volatile agent or hypotension has been reported[88] to cause technical difficulties during intra-capsular cataract extraction which may make anterior chamber lens prosthesis implantation impossible.

A technique of spontaneous ventilation with nitrous oxide–oxygen and halothane has been popular[25] in years past because spontaneous respiration is maintained and prompt recovery is observed. This technique has many drawbacks. To ensure adequate depth of anaesthesia, to prevent patient movement and to ensure a reduction in intraocular pressure initial halothane concentrations of 2–5 per cent followed by maintenance inspired concentrations of 1–1.5 per cent may be required. These high inspired halothane concentrations may eventually produce tachypnoea and shallow tidal volumes resulting in respiratory acidosis and profound hypotension, especially in elderly patients. Normocapnic anaesthesia with controlled ventilation is becoming more popular of late. Gallamine[89] has proved to be a suitable neuromuscular relaxant because of its short duration of action while pancuronium and alcuronium[90] have also proved satisfactory. A thiopentone–succinylcholine induction sequence for elective intraocular surgery has a long established safety record. A two-year retrospective review of 2,217 consecutive cataract extraction[91] performed under general anaesthesia with thiopentone as induction agent and succinylcholine as muscle relaxant revealed no major differences in intraoperative and postoperative complications compared to a group of 561 similar patients operated under local anaesthesia over the same time period.

Anaesthetic management for emergency repair of penetrating eye injuries

The emergency repair of a penetrating anterior chamber injury, in the presence of a full stomach, presents the anaesthetist with a challenging problem. The anaesthetic technique chosen must balance the risk of aspiration of gastric contents with the risk of blindness in the event of increased intraocular pressure and extrusion of ocular contents secondary to coughing, vomiting or drug-effect. Preoperative aspiration pneumonia prophylaxis[92,93] may include gastric acid neutralisation with 0.3 m sodium citrate;[94] the administration of H_2 receptor antagonists[95] cimetidine or ranitidine to increase gastric pH[96] and reduce gastric acid production; metoclopramide administration to increase peristalsis and promote gastric emptying;[97] and intravenous droperidol as an antiemetic prophylaxis.[98]

Because the non-depolarizing relaxant pancuronium $0.15\,mg{\cdot}kg^{-1}$ has been shown to lower intraocular pressure, a barbiturate, non-depolarizing relaxant[99] technique using preoxygenation and cricoid pressure is often advocated as the technique of choice for an emergency repair of a penetrating eye injury.[100] This technique, although widely accepted, has many associated drawbacks. High-dose pancuronium followed by endotracheal intubation may increase heart rate, blood pressure and sympathetic tone[101] and in children intraocular pressure increased to the same extent during laryngoscopy and intubation as when an intravenous bolus of succinylcholine was given prior to laryngoscopy.[102] Pretreatment with intravenous lidocaine $1.5\,mg{\cdot}kg^{-1}$ immediately prior to pancuronium[103] may significantly attenuate the rise in intraocular pressure following laryngoscopy and endotracheal intubation.

The onset time of neuromuscular blockade with high dose pancuronium is relatively slow and a time interval varying from 75 seconds to 2.5 minutes[104] may elapse while the airway is unprotected. Doses

of atracurium and vecuronium that depress twitch response less than 100 per cent of control have onset times ranging from four to eight minutes while most studies[105] indicate that endotracheal intubation cannot be accomplished in less than two minutes following the administration of large doses of vecuronium $0.1\ mg \cdot kg^{-1}$ or atracurium 0.5 $mg \cdot kg^{-1}$.

By administering small subparalysing "priming" doses of nondepolarizing neuromuscular blocking agents several minutes prior to intubating dose, Doherty *et al.*[106] reported a more rapid onset of action of pancuronium while Schwartz *et al.*[107] and Mehta *et al.*[108] reported similar facilitation of rapid endotracheal intubation with divided doses of vecuronium and atracurium. However, if intubation is attempted prematurely, a sudden sharp rise in intraocular pressure may follow associated straining and coughing.

A thiopentone and pancuronium "crash induction" technique is associated with a longer delay between loss of consciousness and intubation and a significantly increased incidence of muscle responses to cricoid pressure, laryngoscopy and intubation[109] in comparison with a thiopentone–succinylcholine technique.[110] The prolonged duration of action associated with high dose pancuronium may necessitate postoperative ventilation.

For emergency open-eye ocular surgery, in the presence of a full stomach, succinylcholine offers the advantages of rapid onset of relaxation, smooth intubating conditions and short duration of action. Following pretreatment with non-depolarizing relaxants,[111] diazepam[65] $0.1\ mg \cdot kg^{-1}$ or hexafluorenium[112] and in combination with sodium thiopentone $3–5\ mg \cdot kg^{-1}$, succinylcholine produces only minor rises in intraocular pressure above control values. Although the efficacy of this technique has been often challenged, there have been no published case reports of loss of intraocular contents associated with the pretreatment-barbiturate-succinylcholine technique when used for induction of anaesthesia.[113] Libonati *et al.*[114] recently reviewed the Wills Eye Hospital 1982 experience of succinylcholine administration to 63 patients presenting for repair of open eye injuries following non-depolarizing relaxant pretreatment. In no instance was any globe content extrusion associated with succinylcholine administration. Moreover, during the past decade at that institution, there were no cases of gastric aspiration or expulsion of intraocular contents in any emergency procedure employing non-depolarizing pretreatment and succinylcholine for endotracheal intubation. Following surgery and reversal of residual neuromuscular blockade, awake extubation in a head-down, lateral position may minimize the risk of aspiration of gastric contents.

Summary

The major factors controlling intraocular pressure during surgery are the dynamic balance between aqueous humour production in the ciliary body and its elimination via the canal of Schlemm; the autoregulation and chemical control of choridal blood volume; the extraocular muscle tone and vitreous humour volume. Prior to surgical incision of the anterior chamber in open intraocular procedures, a low-normal intraocular pressure is mandatory to avoid the hazards of iris or lens prolapse and vitreous loss associated with sudden decompression. In general, the central nervous system depressant drugs, hypnotics, narcotics, major tranquillizers, volatile anaesthetic agents are associated with a reduction in intraocular pressure, with the exception of ketamine and possibly trichloroethylene.

The mechanism of action of anaesthetic agents in reducing intraocular pressure may involve a direct effect on central diencephalic control centres, reduction of aqueous production, facilitation of aqueous drainage or relaxation of extraocular muscle tone. Succinylcholine administration is associated with a significant rise in intraocular pressure, with a peak increase between two to four minutes following administration and a return to base line values after six minutes. The intraocular hypertensive effect may be due to a tonic contraction of the extraocular muscles, choroidal vascular dilatation or relaxation of orbital smooth muscle. Despite many claims to the contrary, no reported method to date has been shown to consistently prevent the intraocular hypertensive response to intravenous succinylcholine administration.

Because the non-depolarizing relaxants are associated with a reduced intraocular pressure, a barbiturate-non-depolarizing relaxant technique utilizing preoxygenation and cricoid pressure has evolved as the most commonly employed induction technique for the emergency repair of a penetrating eye injury. The alternative non-depolarizing relaxant pretreat-

ment–barbiturate-succinylcholine technique may offer the advantages of more rapid onset of relaxation with only minor increases in intraocular pressure and in a carefully controlled rapid sequence induction technique may be the most acceptable method of handling emergency penetrating eye injuries.

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