CONTROL OF THE EYE DURING GENERAL ANAESTHESIA FOR INTRAOCULAR SURGERY

K. B. HOLLOWAY

"There are nine and sixty ways of constructing tribal lays,
And - every - single - one - of - them - is - right."

Rudyard Kipling

THE PROBLEM
Interest in general anaesthesia for intraocular surgery is relatively recent. Undoubtedly, the small area involved may be anaesthetized and immobilized by retro-ocular and facial nerve blocks performed by the ophthalmologist. Also, general anaesthesia may produce problems in the many elderly patients and especially in those with concomitant systemic disease. Further, straining, coughing and vomiting associated with general anaesthesia cause serious increases in intraocular pressure (i.o.p.) which may be disastrous.

As ophthalmology advances the operative procedures become more complex, take longer to perform and are more demanding on both the surgeon and the patient. The patient's ability to co-operate may be overstretched. Whilst local anaesthesia is satisfactory for most standard procedures in skilled hands, in the complex cases the range of techniques for controlling intraocular conditions available with general anaesthesia comes into its own.

The problem is thus the control of the intraocular contents during general anaesthesia. In most intraocular operations the surgical approach is via the anterior chamber and inevitably aqueous will be lost through the incision. If at this time i.o.p. is great, the intraocular contents move towards the incision. The iris, the lens or vitreous may prolapse either immediately or when the surgeon attempts to move the lens. With a high i.o.p. during posterior chamber surgery the vitreous may present first.

Sudden release of pressure in a hard eye may produce rupture of the short ciliary blood vessels at the back of the eye, leading to a calamitous expulsive haemorrhage (Ruiz and Salmons, 1976).

If the eye has already been opened i.o.p. is effectively zero, but any factor which might otherwise have increased the pressure in an intact eye may result in intraocular contents moving towards the incision or being lost through it.

A closed eye will normally withstand a short increase in i.o.p. without trouble, although if extraocular surgery, for example correction of a squint, is being performed there will be more bleeding.

When glaucoma is present there may already be impairment of the blood supply to the optic nerve, and increase of i.o.p. may cause further loss of visual field and acuity.

Intraocular pressure can be measured easily and accurately in most cases, but to assume that i.o.p. is synonymous with "intraocular operating conditions" is a misleading oversimplification.

There are four variable components which may be controlled: pressure from extraocular structures, aqueous volume, choroidal volume and vitreous volume.

Measurement of these is much more difficult than measuring i.o.p., and is impossible in clinical practice. Nevertheless, an understanding of the mechanisms involved in a change of i.o.p. will greatly facilitate the production of safe operating conditions within the eye.

For example, it is possible to produce a low aqueous volume but a high choroidal blood flow. Measurement of i.o.p. might produce an apparently satisfactory reading but following corneal section, after having lost the small volume of aqueous, the ophthalmologist would be faced with a visibly bulging iris, lens and vitreous.

Anaesthetic drugs
In many early studies of the effect of anaesthetic drugs on i.o.p. the conditions in which measurements were taken were not fully standardized and the mechanisms producing the measured effect were not explained. This is understandable, since the minimum


© Macmillan Journals Ltd 1980
monitoring required would include arterial and central venous pressure, heart rate, $P_{aO_2}$, $P_{aCO_2}$ and acid-base state; the value of i.o.p. readings if these are not controlled is limited.

Many general anaesthetic agents have been shown to reduce i.o.p., nitrous oxide being a notable exception. Kornblueth and others (1959) showed that thiopentone, cyclopropane, diethyl ether and divinyl ether all caused a reduction.

Magora and Collins (1961) added chloroform, trichloroethylene and halothane to the list, and showed that the decrease in i.o.p. was proportional to the inspired concentration of the agent. They suggested that all potent inhalation anaesthetics would have a similar effect.

The mechanism is not known, but may be related to the relaxation of extraocular muscle tone and to the depression of ocular centres in the diencephalon, mid-brain and hypothalamus (Von Sallman et al., 1956).

In a carefully controlled trial of patients receiving artificial ventilation with halothane in a nitrous oxide–oxygen mixture Al-Abrak and Samuel (1974a) showed a marked reduction in i.o.p. which was closely related to halothane concentration, but independent of the decrease in arterial pressure.

In a similar study the same authors (Al-Abrak and Samuel, 1973) had shown an increase associated with an increase in central venous pressure on commencing the administration of trichloroethylene.

Adams, Freedman and Henville (1979) and Adams, Freedman and Dart (1979) have used halothane and trichloroethylene in patients who received normocapnic artificial ventilation for cataract surgery. Greater decreases in i.o.p. were produced with halothane (almost 50%) compared with trichloroethylene (14–20%). Central venous pressure was not measured.

Runciman and others (1978) compared the effects of administering MAC equivalents of enflurane and halothane and found that the decrease in i.o.p. was greater with enflurane.

Thiopentone has been shown to reduce i.o.p. in both normal and glaucomatous eyes (De Roeth and Schwarz, 1956; Joshi and Bruce, 1975).

Peuler, Glass and Arens (1975) found no effect on i.o.p. after administering ketamine to adults, and Yoshikawa and Murai (1971) showed a small increase in children. However, Ausinch and others (1976) produced a 25% reduction in i.o.p. by administering ketamine i.m. to children.

Etomidate was shown by Oji and Holdcroft (1979) to produce a marked decrease in i.o.p. even in small doses, in spite of muscle movements and pain on injection.

Opiates cause a moderate reduction in i.o.p., but many anaesthetists avoid them because of their emetic effects. It is fortunate that pain is not often a serious problem after intraocular surgery.

**Pressure from extraocular structures**

Lid retractors may press on the eye and cause bulging of the contents in spite of the anaesthetist's best efforts. The eyelids should be retracted with sutures, or malleable stick-on disposable retractors, arranged to pull them away from the globe. Rarely canthotomy may be necessary.

In eye surgery non-depolarizing muscle relaxants are mainly used to facilitate controlled ventilation of the lungs but these drugs will also relax the extraocular muscles, and might be expected to decrease i.o.p. Al-Abrak and Samuel (1974b) found little change in i.o.p. after pancuronium, but a marked decrease with tubocurarine, associated with circulatory effects. Pancuronium and alcuronium were both found to produce no significant change by George and others (1979).

Hartley and Fiddler (1977) used fazadinium with thiopentone to aid rapid tracheal intubation in emergency cases; there was no increase in i.o.p., a finding that has been confirmed by Couch, Eltrincham and Maguaran (1979).

In intraocular surgery the selection of a non-depolarizing relaxant will depend on its speed of onset and the desired effects on the circulation.

Suxamethonium has been shown to cause an increase in i.o.p. of 7–12 mm Hg (Taylor, Mulcahy and Nightingale, 1968; Pandey, Badola and Kumar, 1972). The increase is rapid in onset, maximal in 1–2 min and with a duration of 5–6 min.

It is generally agreed that suxamethonium may be used safely if the eyeball is intact, as the i.o.p. effect will have worn off by the time an incision is made, but that it should not normally be used after the incision.

The suxamethonium-induced increase in i.o.p. is thought to be largely a result of an increase in tone of the extraocular muscles, which have at least two types of fibres: tonic fibres, which receive multiple innervation by small diameter nerve fibres, do not respond to single stimuli, but react to tetanic stimulation by producing a slow sustained contraction; phasic fibres are innervated by single large-diameter fibres, and respond to single stimuli with a rapid twitch (Wislicki, 1976, 1977).
Joshi and Bruce (1975) showed that, following induction of anaesthesia with thiopentone, which caused a decrease in i.o.p., suxamethonium 0.5 mg kg\(^{-1}\) caused an increase, while a dose of 1 mg kg\(^{-1}\) did not.

Extraocular muscles are unusually resistant to the effect of suxamethonium. Small doses, adequate to produce relaxation of other muscles, increase the tone of extraocular muscles by an effect on tension of tonic fibres. Larger doses affect tension of phasic fibres also, and produce some relaxation; this may be important in clinical practice.

The increase in muscle tension caused by suxamethonium may be abolished by pre-treatment with hexafluorenium, but this is not clinically useful (Katz, Eakins and Lord, 1968).

Attempts have been made to block the increase in i.o.p. associated with suxamethonium by pre-treatment with small amounts of non-depolarizing relaxants; pancuronium (Bowen, McGrand and Palmer, 1976), tubocurarine and gallamine (Bowen, McGrand and Hamilton, 1978) have all been tried and found ineffective.

Adams and Barnett (1966) showed that section of the extraocular muscles and canthotomy do not completely prevent the increase in i.o.p. caused by suxamethonium, and produced evidence that there is an increase in choroidal blood flow resulting in a greater blood volume in the eye which is partly responsible for the increase.

Carballo (1965) found that acetazolamide 500 mg administered i.v. before induction of anaesthesia prevented the effect of suxamethonium on i.o.p.

If the increase is entirely an effect on the muscles it would be difficult to suggest a mechanism for the protective effect of acetazolamide, which is normally administered to reduce the production of aqueous humour. However, Wilson, Strang and MacKenzie demonstrated in primates that i.v. acetazolamide produced an increase in choroidal blood flow to two or three times the normal value, lasting 50 min.

Thus acetazolamide may merely mask that part of the suxamethonium-induced increase in i.o.p. which is a result of choroidal vasodilatation, by pre-dilating it.

**Choroidal volume.**

The choroid consists largely of blood vessels and the volume of blood contained within them may vary widely. This variation may be caused either by the state of vasoconstriction or vasodilatation of the choroid itself, or by distant factors such as changes in intrathoracic pressure affecting venous drainage. As choroidal blood volume is not readily measurable, choroidal thickness and i.o.p. are used as indicators.

Changes in arterial pressure have little effect on i.o.p. within the physiological range. A sudden increase in arterial pressure causes a small increase in i.o.p. because of a slight increase in choroidal volume. In an eye with normal drainage i.o.p. returns rapidly to normal even if the arterial hypertension is sustained, because of loss of aqueous, but the eye would be unpleasant to operate on.

Moderate decreases in arterial pressure also have little effect on i.o.p., but at values of systolic pressure less than 85–90 mm Hg marked reductions in i.o.p. occur (Adams and Barnett, 1966; Schroeder and Linssen, 1972). At systolic pressures of 50–60 mm Hg i.o.p. rapidly approaches zero. The reduction in i.o.p. is partly the result of a decrease in the choroidal blood volume, but also probably of failure of aqueous production. Controlled arterial hypotension is the most effective method of producing and maintaining a very small i.o.p. (in the region of 0–3 mm Hg) should it prove necessary.

An increase of central venous pressure has a far more serious effect on i.o.p. than have increases in arterial pressure. Obstruction of central venous return may be caused by coughing, straining on a tracheal tube, vomiting or a Valsalva manoeuvre. This causes an immediate increase in choroidal blood volume. If the globe is intact there will be an additional slower effect as aqueous outflow is reduced by back pressure on the veins which drain the canal of Schlemm.

A cough can produce an i.o.p. of 40 mm Hg or more. This will return to normal 30 s after the cause of the increase has ceased, but if the eye is open damage may already have occurred.

The choroidal blood flow, like the cerebral circulation, is dependent on arterial P\(\text{CO}_2\). Wilson, Strang and MacKenzie (1977) measured choroidal blood flow in primates by the xenon-133 clearance technique and found a linear increase in flow of 0.5% per kPa increase of P\(\text{CO}_2\) from 4.5 to 9.1 kPa.

Increasing the arterial P\(\text{CO}_2\) has been shown to cause an increase of i.o.p. (Samuel and Beaugié, 1974; Wilson et al., 1974), while a reduction below normal causes a decrease (MacDiarmid and Holloway, 1976).

The increase in choroidal blood flow following i.v. amiphenazole has already been mentioned.

The choroidal circulation is affected by arterial P\(\text{O}_2\). Saltzman and others (1965) studied the effects of breathing oxygen at pressures of 1–3.7 atmospheres.
absolute. Profound choroidal vasoconstriction, which correlated with the increase in $P_{a_4}$ occurred within 3 min. The vasoconstriction was much more marked than that associated with hyperventilation, but reversed in 1 min on breathing air. This effect does not appear to have been used to facilitate intraocular surgery, presumably because of the scarcity of hyperbaric operating facilities or the inconvenience of using them.

**Vitreous volume**

The vitreous is an unstable gel with a fine fibrillar supporting structure and consisting mainly of water. The inert appearance is deceptive, there being a continuous turnover of the water content.

Acute angle closure glaucoma can be extremely painful and distressing, but rapid relief may be obtained by dehydrating the vitreous. Initially, urea was used for this purpose, 1–2 g/kg body weight given i.v. as a 30% solution in water. It is very effective in decreasing i.o.p. but because many of the patients are elderly with co-existing hypertension or renal disease, it has largely been superseded by mannitol.

Mannitol is given in doses of up to 1.5 g kg⁻¹ as an i.v. infusion of 20% solution and will produce relief within 40 min lasting a few hours. It may then be decided to proceed with a surgical drainage procedure and the anaesthetist will be presented with a rather dehydrated patient who is experiencing a marked diuresis and who will require urethral catheterization if rendered unconscious.

The oral administration of glycerol up to 2 g kg⁻¹ may be used either alone or to enhance the effect of mannitol on the vitreous volume. If a patient with acute glaucoma is presented for general anaesthesia, this should always be enquired about because of the risk of regurgitation.

If mannitol is used to reduce i.o.p. for elective intraocular surgery under anaesthesia, administration should commence 45 min before operation and the patient must be catheterized when unconscious; this may be a disadvantage, particularly in elderly men who may be developing prostatic obstruction of the urethra.

A more rapid response can be obtained by an i.v. bolus injection of 1 g kg⁻¹ of a 50% aqueous solution of sucrose. I.o.p. will decrease significantly in 5 min, accompanied by an increase in arterial pressure and heart rate. Thus the effect is rapid enough to be of use if the eye is tense during the surgical procedure, but it is essential first to determine the underlying cause of the trouble.

Diuresis is not a problem with sucrose and routine bladder catheterization is not necessary.

**MANAGEMENT OF PATIENTS UNDERGOING INTRAOCULAR SURGERY**

With so many ways of influencing i.o.p. available to the anaesthetist, it is not surprising that a variety of anaesthetic techniques has been employed for intraocular surgery. If the method used is based on an understanding of the mechanisms of i.o.p. changes a satisfactory result is likely, and in difficult cases the anaesthetist may take some of the credit for the success of the operation.

Conversely, if the anaesthetic management is unsatisfactory, this will be apparent to the surgeon and some responsibility for operative complications must be taken.

The same anaesthetic technique will not be appropriate for all intraocular cases. The writer works in a unit where a high proportion of the patients are secondary referrals from other ophthalmologists, and encounters a wide range of difficulties presented and of the amount of control of intraocular conditions required. The use of a standard technique for all cases would result in some patients receiving an unnecessarily severe physiological insult, or some eyes being at risk, or both. It is no longer true to say “when an eye is to be opened the intraocular pressure should be as low as possible” (Duncalf and Rhodes, 1963), since what is possible may be excessively drastic. The method of anaesthesia also must be adapted to match the age and general physical state of the patient.

Intracapsular extraction of a cataract may be taken as the typical intraocular operation in which a large opening is made in the eye.

The basic essential for all intraocular surgery is a meticulously smooth, flawless anaesthetic technique, with avoidance of coughing, straining or vomiting and adequate and unimpeded ventilation of the lungs. Most patients with uncomplicated cataract may be anaesthetized quite simply using spontaneous ventilation with halothane. Premedication should not include drugs likely to produce ventilatory depression or vomiting. Promethazine and atropine, or diazepam and atropine, perhaps combined with droperidol in the more robust patients, are satisfactory.

The decrease in i.o.p. following thiopentone induction may compensate for the increase caused by suxamethonium, and a large dose of the latter drug may produce less of an increase than a small one (Joshi and Bruce, 1975).
Spraying the pharynx, larynx and trachea with 4% lignocaine may help to minimize the increase in i.o.p. which often accompanies tracheal intubation. The tracheal tube should be well lubricated with 5% lignocaine ointment; if there is any doubt about the tube being too long it should be shortened to avoid irritating the carina. If bronchial suction is necessary, this should be done very gently at this stage, remembering that it will not be possible to do this during the operation without inconvenience to the operating team and risk to the eye. The level of anaesthesia should then be deepened with halothane and nitrous oxide in at least 35% oxygen as rapidly as the patient will tolerate.

To maintain a low venous pressure, the operating table should be placed in 10–15° foot-down tilt, and if the expiratory valve has a resistance of more than 1 cm H$_2$O fully open, the spring should be shortened.

An adequate depth of anaesthesia is essential, not only to prevent any patient reaction, but also because the effect of halothane on i.o.p. is proportional to dose (Al-Abrak and Samuel, 1974a). Initial halothane concentrations of 2–5% will usually be necessary and in most cases at least 1.5% will be needed until the lens has been extracted and the first suture tied. A controllable reduction in arterial pressure may be considered desirable in many cases, but excessive circulatory depression is a risk in frailer patients if the anaesthetist is not vigilant.

Rapid breathing may cause movement of the operating field under the microscope, and may be controlled by titration with pentazocine i.v., care being taken to avoid significant ventilatory depression.

It used to be the practice of the writer to maintain deep anaesthesia until after extubation to prevent coughing at this time, with resultant strain on the suture line and the risk of iris prolapse or worse. This is no longer done, because although the patient does not cough immediately he is likely to do so within a short time. The view is now taken that, with microtechnique, a “soft” eye and plenty of time for suturing, the surgeon must be responsible for making the eye watertight.

The technique described above is unlikely to provide satisfactory operating conditions if there is any impairment of the efficiency of spontaneous ventilation as a result of respiratory disease, obesity or skeletal malformation. For such cases the technique of controlled ventilation with 0.5% halothane described by Adams, Freedman and Henville (1979) would be satisfactory. These authors used normocapnic ventilation to avoid circulatory depression during anaesthesia and postoperative hypoventilation. Gallamine was chosen for myoneural blockade because of its short action and relative lack of side-effects. Pancuronium or alcuronium could be used instead (George et al., 1979).

Satisfactory small values of i.o.p. were obtained for standard intercapsular cataract extraction. When trichloroethylene was substituted for halothane, the resulting i.o.p. decrease was less (Adams, Freedman and Dart, 1979). For most elderly patients with uncomplicated cataract this technique will soften the eye sufficiently for a safe lens extraction.

If the iris and lens bulge forward after the eye is opened, there is usually a simple explanation, such as difficulty with ventilation, straining on the tracheal tube, too light anaesthesia, pressure on the neck, or pressure on the eye from retractors. If no such cause is found, administration of 50% sucrose i.v. will shrink the vitreous and control bulging within a few minutes. The effect will last long enough to allow lens extraction and insertion of corneal sutures in safety.

Cataract extraction may be made more hazardous by various complicating factors. In younger patients the attachments of the lens to the zonule are stronger and it may also be adherent to the vitreous face. Patients with diabetes mellitus often have a weak lens capsule which ruptures more easily, a pupil which will not dilate adequately and vitreous which degenerates, becoming more watery and thus more easily lost.

Vitreous degeneration may also follow uveitis or trauma, and often accompanies myopia. Adhesions within the eye may occur after uveitis, trauma or previous detachment or other operation.

There may be co-existing glaucoma, and it is becoming more common to perform combined operations, such as cataract extraction accompanied by trabeculectomy.

Preoperative dislocation of the lens is particularly troublesome if it is posterior and there is vitreous in front of it. This can occur simply as a complication of cataract, or part of the syndrome in homocysteinuria or Marfan’s disease.

If the patient has only one saveable eye this is an added burden of responsibility upon the operating team, even in the absence of other complications. In many of these cases the risk to the eye will be less if a greater degree of control of intraocular conditions is used than has so far been described. Since this will involve greater physiological disturbance, the degree of assistance required from the anaesthetist should be discussed with the surgeon. It is fortunate that most
of the more difficult problems occur in the younger patients.

Hyperventilation of the lungs is relatively safe and the resulting choroidal vasoconstriction reduces the i.o.p. and decreases bleeding. Excessive hyperventilation is not necessary and may cause a troublesome increase in central venous pressure. $Pa_{CO_2}$ 4 kPa or slightly less should be aimed at, and may be conveniently monitored with an end-tidal carbon dioxide sampler. Central venous pressure is controlled by a foot-down tilt of 10-15°, a short but not abrupt inspiratory phase from the ventilator, and the use of a negative end-expiratory phase of $-10 \text{ cm H}_2\text{O}$.

The disadvantages of this pattern of ventilation, such as intrapulmonary gas trapping, and increased shunt must be recognized, but significant upset is rare in practice.

Where necessary, for example when several complications occur together in the same eye, intraocular conditions may be further improved by substituting tubocurarine for gallamine, pancuronium or alcuronium, and by increasing the concentration of halothane administered (Al-Abrak and Samuel, 1974a, b). The combination of a high tissue concentration of halothane, hypocapnoea and circulatory depression will produce a very soft eye. To view an apparently easy and uncomplicated cataract extraction in a young diabetic person with myopia and glaucoma in the only functioning eye, is rewarding.

The increase in choroidal blood flow following i.v. acetazolamide has been mentioned and this should not be used as a preoperative measure to decrease the i.o.p. in cataract surgery complicated by glaucoma. Reducing the quantity of aqueous in the anterior chamber may make the initial incision safer, but thereafter the bulging choroid may push the intraocular contents forward. In these circumstances sucrose may be unsatisfactory and the best way to control the eye is to initiate controlled arterial hypertension with trimetaphan or sodium nitroprusside.

If intraocular cataract extraction is to be followed by the insertion of a lens implant, it is important that the anterior vitreous face should be well back and unbroken. There is a good case for adding the preoperative administration of mannitol to a hyperventilation technique. The need for bladder catheterization may be acceptable when a compact vitreous with a well-defined and receding face is produced.

Many intraocular operations are performed in which the incision of the globe is very small, and there is not the same risk of extrusion of intraocular contents, particularly vitreous, as occurs in intra-capsular cataract extraction. These include the drainage procedures for the treatment of glaucoma, such as peripheral iridectomy, iridencleisis and trabeculectomy, and a growing list of others, such as needling, phako-emulsification and vitrectomy. The risk of sudden decompression of a hard eye is still present and it may be difficult or impossible to perform a satisfactory drainage procedure if the iris is protruding into the incision.

Although very small values of i.o.p. are not necessary, the anaesthetic requirements for these cases are the same as those for cataract extraction and similar techniques can be used. Replacement of the cornea with a full thickness graft also requires control of i.o.p. but this must not be excessive. If the eye is made too soft, accurate suturing of the graft becomes more difficult.

Retinal detachment surgery is "extraocular", but a reduction in i.o.p. makes the application of indenting plombs and bands easier and the release of subretinal fluid safer. It is also associated with less bleeding.

Ophthalmoscopy at the end of a detachment procedure sometimes reveals that an equatorial band has been applied too tightly. The retinal reflex is pale and the blood supply to the eye is jeopardized. It is usual to remove the band and replace it, which is a time-consuming procedure and risks that the band will be too loose. If aqueous drainage is adequate and the ophthalmologist agrees, this problem can be managed differently by giving 50% sucrose i.v. The simultaneous decrease in i.o.p. and increase in arterial pressure so produced will restore the blood flow through the eye; by the time sucrose effect has worn off the eye will have adjusted its own internal volumes.

**Intraocular tumours**

Until recently an intraocular tumour has been an indication for removal of the eye unless the condition could be treated by radiotherapy. Excision of the tumour with preservation of the eye is often possible now and the writer has anaesthetized 40 patients for this procedure. Their age has ranged from 23 to 74 yr, but most have been nearer the young end of the scale, only two being older than 60 yr.

The most demanding cases are patients with choroidal melanoma since the surgery is delicate and prolonged and the tumour is extremely vascular. After incising the conjunctiva and detaching appropriate extraocular muscles from the globe, a square flap of the outer layers of the sclera over the tumour
ANAESTHESIA FOR INTRAOCULAR SURGERY

Anaesthesia is induced with thiopentone, the trachea is intubated after injection of tubocurarine and the lungs hyperventilated with 0.5% halothane in 50% nitrous oxide in oxygen with a negative expiratory phase of \(-10\) cm H\(_2\)O; the patient is placed in the foot-down position. During the period of 20-30 min when the choroid is being cut and the tumour pulled from the vitreous a systolic pressure of 45-50 mm Hg is the objective; this is produced by increasing the halothane concentration. In elderly patients pressures 10 mm Hg greater will be adequate.

Although the pulsation of the central artery of the retina can be seen through the vitreous after the tumour has been excised, there will be almost no bleeding from the choroid when it is cut, and this is essential to the operation. Meticulous monitoring of the patient's cardiovascular state is essential and the author places greater emphasis on evidence of good peripheral perfusion than on pressure measurement.

After the tumour has been removed and scleral suturing has commenced the inspired halothane concentration is decreased or discontinued and the arterial pressure allowed to increase slowly. Since closure takes some time it will rarely be necessary to take any active steps to restore the arterial pressure to normal.

Tumours of the ciliary body are much less vascular than those of the choroid and the smallest values of arterial pressure described above will not be needed. Tumours of the iris can usually be managed in the same way as a difficult cataract operation.

The results in terms of eradicating the tumour while preserving sight have been very worthwhile, and the experience gained is allowing larger tumours to be excised than was at first thought possible.

**Penetrating eye injuries**

Penetrating eye injuries cause particular concern to the anaesthetist since, added to the problem of anaesthetizing a patient whose stomach may not be empty, there is the risk that anything which increases i.o.p. may result in extrusion of ocular contents and loss of sight.

Procedures used in other emergency cases have serious disadvantages. Emptying the stomach by gastric tube is obviously contraindicated because of the straining and spluttering inevitably caused. Suxamethonium is an essential part of the usual "crash" induction technique, but will have an effect on i.o.p. Cricoid pressure applied to prevent regurgitation may well block the venous drainage of the eye.

Other injuries, perhaps of a more life-threatening kind may co-exist, and a balanced judgement of priorities will have to be made. With regard to the eye, a smooth induction of anaesthesia and intubation of the trachea are the essentials. Thereafter maintenance of anaesthesia can be by any technique normally used for elective intraocular surgery.

The suggestion is often heard that it would be satisfactory to employ an inhalation induction with halothane, enflurane or cyclopropane, deepening the anaesthesia to allow insertion of the tracheal tube. This may be possible sometimes, but experience suggests that the depth of anaesthesia required can easily be mistaken, and this technique is not recommended to an inexperienced anaesthetist in such a case. Furthermore, if the patient does cough or regurgitate the resulting increase in i.o.p. is likely to
be far greater than that produced by suxamethonium, and may also last longer.

Thus it is wiser, especially if other indications for a rapid induction are present, to use suxamethonium, using a large dose after a thiopentone induction to minimize the increase in i.o.p.

The writer prefers to use one of the more rapidly acting non-depolarizing relaxants. Fazadinium is suitable and alcuronium and pancuronium are only slightly less rapid. If the patient is able he is asked to preoxygenate himself for 5 min, since he is less likely to press the mask on his own eye. An indwelling needle is placed and, after atropine, a generous paralysing dose of the relaxant is injected. As the arm holding the mask begins to weaken, it is gently removed by an assistant as a small dose of thiopentone is rapidly injected. After a short pause smooth tracheal intubation can be performed and the patient ventilated in the usual way. This method should not be used if intubation is likely to be very difficult.

Patients with eye conditions such as glaucoma or detachment of the retina frequently require repeated surgical procedures at short intervals and the problem of repeated halothane anaesthesia will arise.

Halothane is such a useful aid in intraocular surgery that this is a serious drawback and possible alternatives also have disadvantages.

Trichloroethylene is much less satisfactory because it is associated with tachypnoea, cardiac arrhythmia and, in some cases, prolonged recovery.

Enflurane can often be an effective substitute, but the manufacturers are unsure about the possibility of cross-sensitivity with halothane or other halogenated hydrocarbons. The central nervous stimulating effect of enflurane is a contraindication to the use of hyperventilation.

Neuromuscular block-controlled paralysis, ventilation and the supplementation of nitrous oxide and oxygen mixtures with a narcotic and neuroleptanalgesia are a possible alternative to halothane, although difficulties may occur in elderly patients in whom metabolism and excretion of depressant drugs may be depressed. Development of reliable methods of measuring anaesthetic depth would help with these techniques, but in complicated cases hyperventilation and hypotension are more likely to be needed.

The writer's present practice is to use halothane for all cases for intraocular surgery where management is likely to present more problems than usual, whether or not it has previously been administered.

No doubt sufficient information will eventually be available to justify or condemn this practice.

REFERENCES


