Chapter 17

EYE INJURIES

JOHN D. CURRENT, M.D.*

Colonel, Medical Corps, U.S. Army Reserve; formerly, Chief, Anesthesia and Operative Service, Brooke Army Medical Center, Fort Sam Houston, Texas 78234; currently, Associate Professor, Department of Anesthesiology, University of Mississippi Medical Center, 2500 North State Street, Jackson, Mississippi 39216

INTRODUCTION

There are two basic types of eye injuries: penetrating (ie, open) and nonpenetrating. As a rule, nonpenetrating injuries are less severe than penetrating injuries, but either type can lead to blindness. Nonpenetrating eye injuries, in which the globe remains intact, are usually lesser anesthetic-management dilemmas than penetrating eye injuries, in which the globe is disrupted. Nonpenetrating eye injuries include injuries to the eye and orbit and can be as minor as a corneal abrasion or eyelid ecchymosis, or as severe as retinal detachment, vitreous hemorrhage, or optic nerve injury. Penetrating injuries—the major source of serious combat eye injuries—are more common than might be thought, given the small portion of the body surface area that the eyes occupy. In wars of this century, from 3% to 9% of surviving casualties have been treated for eye injuries.[1] The higher-than-expected frequency of eye injuries reflects not only the exposed position of the head in combat but also the vulnerability of the eyes. They are a "soft" target: an injury of the same magnitude in another part of the body might very well be ignored. Finally, the increased use on the modern battlefield of explosive munitions that are designed to produce large numbers of small fragments is an additional factor that predisposes to the unexpectedly high frequency of eye injuries.

Of combat eye casualties in the Vietnam War in whom the mechanism of their eye injury was known, 79% were injured by fragments, with rockets, mortars, mines, and grenades being the most common explosive munitions responsible; bullets were the source of only 6% of eye wounds.[1] This is interesting because in living casualties, bullets caused a much smaller proportion of eye injuries compared with their overall casualty-generating importance during the Vietnam War (ie, bullets were the wounding agents in 30%–50% of living casualties).[2] These percentages would seem to be contradictory, but the reason for the seeming contradiction is simple: compared with a small fragment, a bullet that injures the eye is much more likely to continue on into contiguous organs such as the brain, where it frequently causes a fatal injury.[2] The fragments that cause eye injuries in *living* casualties are, in fact, quite small: 70% of the fragments had a mass of 1 to 100 mg.[1]

Many combat casualties present with corneal foreign bodies, the removal of which rarely requires the participation of an anesthesiologist. In the Vietnam surgical experience, the most common indication (50%) for a major eye operation was the presence of a disrupted globe requiring an enucleation (Figure 17-1). Other common indications for operation were corneal or scleral lacerations (19%), the presence of an intraocular foreign body (15%), and the laceration of the ocular adnexa (11%). Outside the combat zone, the most common indications for a major eye operation in a combat casualty with an eye injury were the presence of an intraocular foreign body (37%) and the need for enucleation (35%). The indication for the latter was severe infection that had destroyed the globe or the threat of sympathetic ophthalmia.[1] To care properly for such patients, the military anesthesiologist must know of the anatomy and physiology of the eye.

ANATOMY AND PHYSIOLOGY OF THE EYE

The eye is divided into the posterior and anterior chambers by the iris and the pupil (Figure 17-2). Aqueous humor fills the anterior and posterior chambers. The aqueous humor is formed primarily by the ciliary processes, with some contribution from the iris. It flows forward through the pupil to the angle formed by the iris and cornea. There, a trabecular meshwork containing the spaces of Fontana (ie, the spatia anguli iridocornealis) is the site of absorption of the aqueous humor. The aqueous humor then flows through the canal of Schlemm (ie, the sinus venosus sclerae), eventually reaching the venous system. Hypercarbia results in the dilation of choroidal arteries.[3] The sclera forms a fairly rigid structure, restricting volume changes in the eye (ie, restricting compliance). The net effect is analogous to changes in intracranial pressure: hypercarbia causes increased intraocular pressure (IOP).[4] Similarly, hyperventilation causes choroidal arterial vasoconstriction with a concomitant decrease in IOP.[5] The surgeon may request hyperventilation by the anesthesiologist in hopes of reducing IOP. However, the effect is quantitatively small. There is only about a 0.1 torr change in IOP for every torr change in the partial pressure of carbon dioxide in the arteries ($Paco_2$), which limits the value of the practice.[6]

Hypoxia also causes choroidal arterial vasodilation and a rise in IOP. Interestingly enough, hypothermia does not increase IOP; on the contrary,

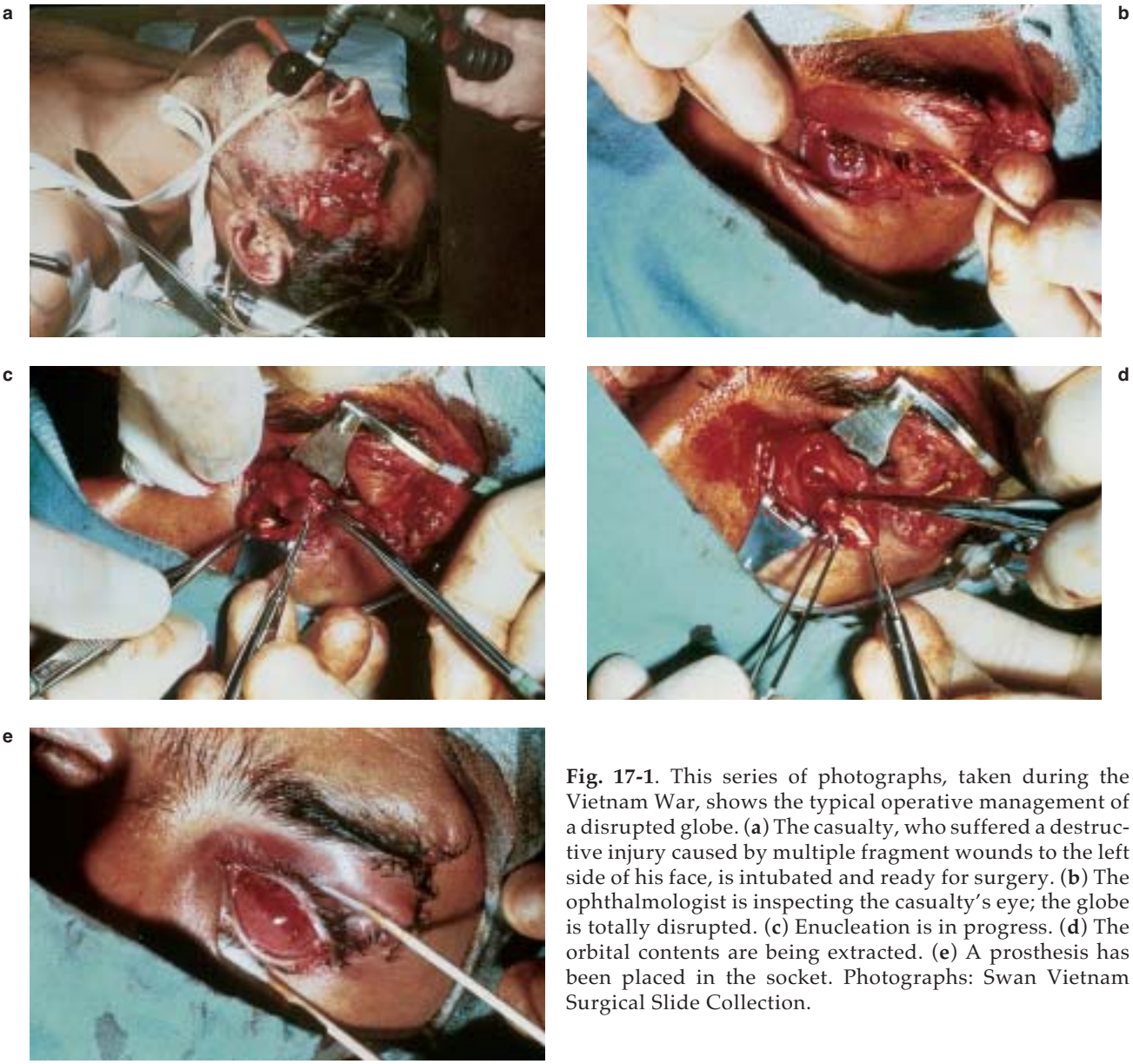

Fig. 17-1. This series of photographs, taken during the Vietnam War, shows the typical operative management of a disrupted globe. (**a**) The casualty, who suffered a destructive injury caused by multiple fragment wounds to the left side of his face, is intubated and ready for surgery. (**b**) The ophthalmologist is inspecting the casualty's eye; the globe is totally disrupted. (**c**) Enucleation is in progress. (**d**) The orbital contents are being extracted. (**e**) A prosthesis has been placed in the socket. Photographs: Swan Vietnam Surgical Slide Collection.

hypothermia reduces the production of aqueous humor and also causes vasoconstriction, which result in a lowered IOP.[7] Increasing venous pressure may profoundly increase IOP. This occurs by impeding outflow of aqueous humor through the canal of Schlemm, compression of the eye by episcleral veins, and dilation of choroidal vessels. Any straining, coughing, vomiting, and so forth during laryngoscopy or emergence from anesthesia may result in dramatic increases in IOP via this mechanism.[8] Increases of 30 to 40 torr have been recorded. External compression of the eye (eg, pressure from a mask or the increased tone of extraocular muscles) may increase IOP. This is particularly hazardous in the case of an open eye injury because the potential exists for loss of eye contents. However, when the globe is intact, the ophthalmologist may use several minutes of eye compression to reduce IOP and make the globe less rigid. This likely occurs owing to expression of aqueous humor from the anterior chamber.

The discussion of IOP has so far emphasized mechanical and vascular determinants. However, the normal mechanism for control of IOP involves the balance between production and drainage of the aqueous humor. Most pharmacological means to control IOP affect this balance. Normal IOP is 10 to 22 torr; IOP is considered abnormal if it exceeds 25

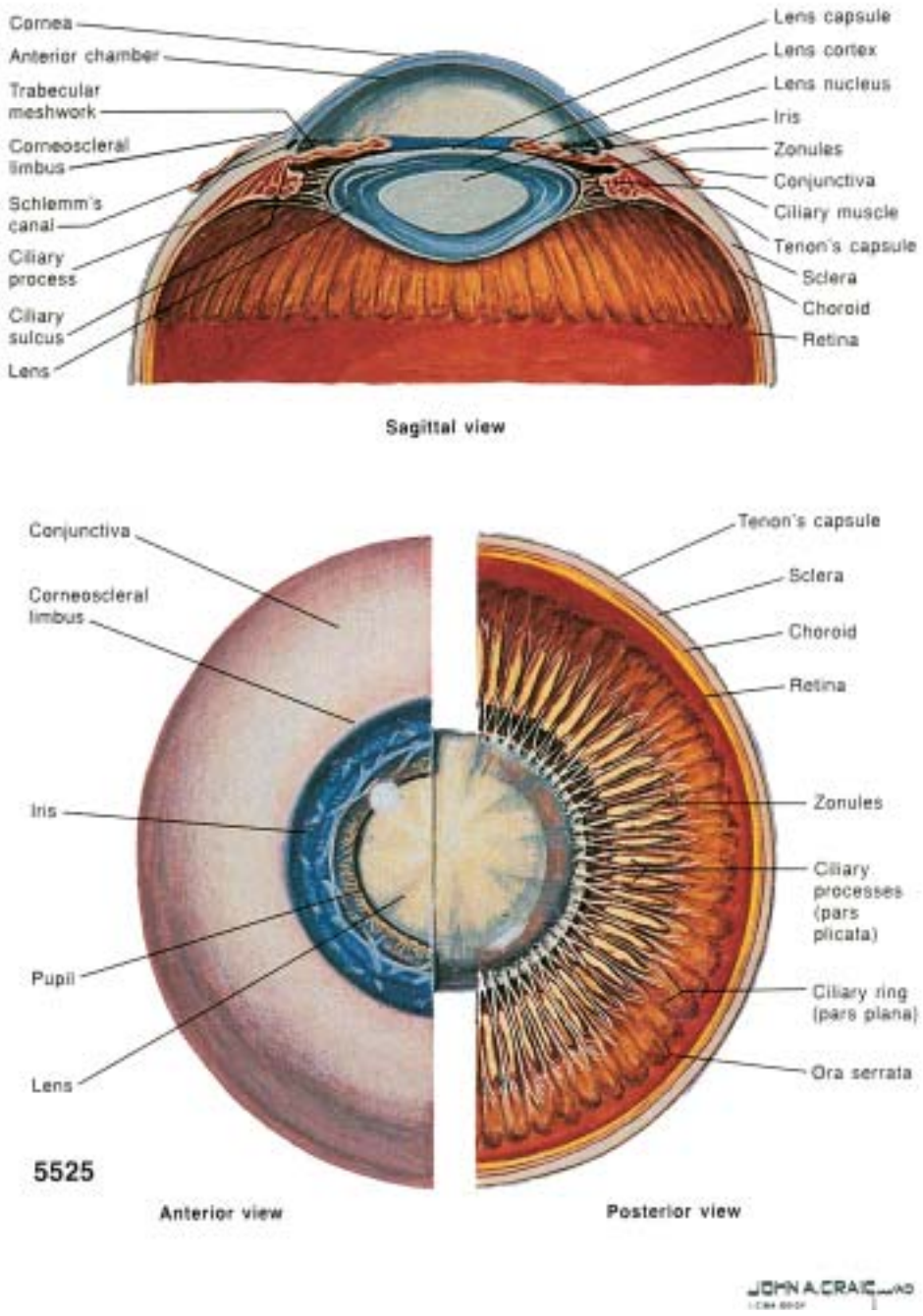

Fig. 17-2. Anterior segment of the eye. Reprinted with permission from Paton D, Craig JA. Management of cataracts. *Clinical Symposia*. 1990;42(4):6.

torr. The method of measurement may influence the IOP determined. Indentation (ie, Schiötz) tonometry may cause expression of aqueous humor and a reduction of IOP with repeated determinations. Older studies using indentation tonometry for measuring the effects of drugs and anesthetic agents on IOP have been called into question because of this. The more accepted and accurate method for determination of IOP is applanation tonometry, particularly if repeated measurements are necessary.[7]

EFFECT OF DRUGS ON INTRAOCULAR PRESSURE

Miotic agents such as echothiophate (Phospholine Iodide, manufactured by Wyeth-Ayerst Laboratories, Philadelphia, Pa.) and acetylcholine are used to lower IOP. The constriction of the pupil tightens the iris, stretching the trabecular meshwork of the spaces of Fontana. The net result is a facilitation of drainage of the aqueous humor and lowering of IOP. Aqueous humor production may be decreased with ophthalmic preparations of epinephrine, timolol, and acetazolamide. The production of aqueous humor is an active process using the carbonic anhydrase enzyme system. Therefore, acetazolamide can reduce IOP by reducing the production of aqueous humor by 50%. Acute lowering of IOP may be achieved through the use of osmotic agents. These drugs act to increase plasma oncotic pressure, which diminishes aqueous production, albeit transiently. Mannitol is the drug most commonly used, although urea and glycerol have also been used.[3] They all require catheter drainage of the urinary bladder intraoperatively. Furthermore, glycerol, given orally, increases the volume of stomach contents and adds a potential risk for regurgitation and aspiration under general anesthesia.

General anesthetic agents, particularly barbiturates and potent inhalational agents have consistently been shown[9–11] to reduce IOP. In humans, intravenous administration of etomidate has been shown[12] to consistently and significantly lower the IOP. Intravenous lidocaine has been shown[13] to reduce the IOP response to laryngoscopy and intubation. Ketamine, on the other hand, may increase the IOP or leave it unchanged. An intravenous bolus dose of ketamine has been found[14] to result in a 37% increase in IOP, with the maximum increase occurring 15 minutes after the injection. Other researchers[15,16] have presented data that suggest that heavily premedicated patients do not experience increases in IOP with ketamine. However, blepharospasm, nystagmus, or laryngospasm may make ketamine undesirable for ophthalmic surgery.

Succinylcholine has been found[17] to increase IOP. This increase is not temporally related to muscle fasciculations[18]; indeed, pretreatment with acetazolamide or propranolol have been reported[19] to prevent a rise in IOP after succinylcholine without preventing fasciculations. Increases in IOP with succinylcholine have been reported[20–24] after adequate pretreatment doses of nondepolarizing muscle relaxants. Attempts to use small, "self-taming" doses of succinylcholine have themselves resulted in elevation of IOP.[25] Furthermore, pretreatment with lidocaine does not prevent increases of IOP following succinylcholine administration.[26]

In contrast, nondepolarizing muscle relaxants consistently reduce—or at worst, do not change—IOP.[27–31] Intubating doses of atracurium and vecuronium do not raise IOP, but the intubation itself may.[32] The increase-in-IOP response to laryngoscopy and intubation in children was prevented by the addition of intravenous lidocaine (1.5 mg/kg) to the standard induction sequence of thiopental and pancuronium.[33]

ANESTHETIC MANAGEMENT OF THE CASUALTY WITH AN OPEN EYE INJURY

Anesthetic management may play an important role in the outcome of ophthalmic surgery. The surgeon requires a motionless eye and a bloodless field. Any coughing, bucking, straining, or vomiting by a patient with an open eye injury may result in hemorrhage and loss of vitreous or vision.[8] Eye injuries in which the globe is fully perforated require especially careful anesthetic management to avoid extrusion of intraocular contents and resultant permanent disability. The anesthesiologist must certainly avoid applying external pressure to the eye, such as during application of the face mask. It is also helpful to the surgeon to have the endotracheal tube out of the way, using a downward-directed tube such as a RAE (named for its developers: Ring, Adair, and Elwyn; manufactured by Mallinckrodt Critical Care, Great Falls, N.Y.) or an armored tube. Of simultaneous concern to the military anesthesiologist is the likelihood that the casualty will have a full stomach. If time and the patient's medical condition permits, aspiration prophylaxis should be considered. Oral or intramuscular histamine 2 blocking agents may be administered to the patient, preferably 1.5 to 2 hours prior to the procedure. Ranitidine 150 mg, administered orally, or 50 mg, administered intramuscularly (or alternatively, cimetidine 300 mg, administered orally, or 150 mg, administered intramuscularly), should increase the pH of the stomach contents. Famotidine 20 mg, administered orally or intravenously, will also reduce gastric volume and increase pH. Reglan 10 mg, administrated orally or

intravenously, may reduce the volume of stomach contents by promotion of gastric emptying. Administration of a nonparticulate antacid (ie, Bicitra, manufactured by Baker Norton Pharmaceuticals, Inc, Miami, Fla., 30 mL, administered orally) will effectively increase the pH of stomach contents although at the expense of a small increase in gastric volume.

Intubation and Induction

Retrobulbar block is contraindicated due to the risk of external pressure causing extrusion of eye contents. Indeed, retrobulbar block itself may be complicated by global perforation.[34] Similarly, awake intubation is contraindicated due to the real risk of coughing during topical anesthesia or airway insertion. There is a general agreement that the best approach is to perform endotracheal intubation with a rapid-sequence induction of anesthesia and cricoid pressure.[35] Care must be taken in the application of cricoid pressure to avoid both occluding venous return from the head and, possibly, increasing the IOP. The controversy surrounds the choice of muscle relaxant selected to facilitate intubation. The thiopental-pancuronium induction sequence is the standard against which other techniques must be compared.[36] If hemodynamic stability permits, a relatively large dose of thiopental may lessen the likelihood of any patient reaction to laryngoscopy or intubation. Administration of pancuronium 0.15 mg/kg should allow intubation within 90 seconds. The patient should be counseled regarding the possibilities that the relaxation could outlast the procedure and therefore that postoperative intubation and ventilation might be required. The likelihood of tachycardia may make this technique undesirable in patients with coronary artery disease.[37]

Variations on this nondepolarizing sequence include (*a*) large-dose atracurium or vecuronium,[38] (*b*) Metubine-pancuronium combination (Metubine Iodide, manufactured by Dista Products Co., Indianapolis, Ind.), and (*c*) the priming principle. Researchers[39] who studied onset time and intubating conditions using atracurium 1.5 mg/kg and vecuronium 0.25 mg/kg found onset time of 95% twitch depression to average 56 and 64 seconds, respectively. Intubating conditions at 60 seconds were somewhat better with vecuronium, but were satisfactory with either regimen. They noted that 40% of the patients receiving that dose of atracurium suffered hypotension and tachycardia.

The Metubine-pancuronium combination takes advantage of the synergistic effect of these two drugs to reduce the amount of each necessary to achieve rapid relaxation. Pretreatment with Metubine 0.05 mg/kg followed by pancuronium 0.06 mg/kg gives adequate intubation conditions nearly as rapidly as the larger-dose pancuronium technique but does not produce so prolonged a period of paralysis. Other researchers[40] pretreated patients with metocurine 0.03 mg/kg and 3 minutes later administered pancuronium 0.08 mg/kg. They found that 95% depression of twitch height occurred in 70 seconds and all patients had good-to-excellent intubating conditions. However, these patients' recovery to 25% twitch height required 100 minutes.

The use of the priming principle has been advocated for patients with open eye injuries.[41] A defasciculating dose of an intermediate-duration nondepolarizer is followed 3 to 5 minutes later with a larger dose, 1.5- to 2-fold larger than the usual intubating dose.[36] This gives a fairly rapid onset of muscle relaxation but not as rapid as that produced by succinylcholine.

One group of researchers[42] found that additional thiopental improved intubating conditions when using the priming principle with atracurium. Furthermore, other researchers[43] found that priming with atracurium did not significantly improve intubating conditions, and recommended that it not be done. This may reflect the importance of deep anesthesia in securing favorable intubating conditions without increasing IOP.[42] The duration of the blockade is much shorter than that obtained with pancuronium, although postoperative ventilation could be necessary following short procedures. At recommended doses, atracurium may occasionally result in adverse hemodynamic changes. Therefore, I suggest the use of vecuronium 0.01 mg/kg as a priming dose, followed in 3 to 5 minutes with 0.2 mg/kg. There should be virtually no hemodynamic consequences from the vecuronium.

Whether succinylcholine should be used in the patient with open eye injuries remains an issue of debate. Although some researchers[44] have reported considerable experience with the use of a pretreatment dose of curare followed by succinylcholine in the patient with open eye injuries with neither loss of vitreous nor aspiration, others[45] consider this

approach to be controversial. Nevertheless, investigators[46] reporting in 1990 suggest that the rapid-sequence induction technique modifies the effect of succinylcholine on IOP resulting in no loss of vitreous. They believe that both (*a*) scrupulous attention to adequate depth of anesthesia with thiopental and (*b*) waiting until complete paralysis is instituted with succinylcholine before laryngoscopy and intubation permit safe intubation, as had been demonstrated[44] in 1985. Adequate depth of anesthesia avoids the likelihood of coughing, straining, and grimace, which are likely to be more problematic than the effects of succinylcholine in the anesthetized patient.[46]

A large dose of thiopental should be used in the hemodynamically stable patient, assuring adequate depth of anesthesia and minimizing any response to laryngoscopy and intubation. The addition of alfentanil 20 μg/kg has been found useful in reducing the response to tracheal intubation.[47] Awake intubation is inadvisable in patients with open eye injuries because any cough or straining could result in loss of ocular contents. Similarly, retrobulbar block is contraindicated because the block itself may increase the IOP due to extrinsic pressure on the globe. Furthermore, blepharospasm, grimace, or perforation of the globe may occur,[48] and the operator may not be able to minimize hemorrhage by applying manual pressure.

Patients with severe hypovolemia from acute blood loss may not tolerate standard anesthetic-induction techniques. Severe hypotension or cardiac arrest may occur if the patient is given a hypnotic induction such as ketamine, which causes cardiac depression, or thiopental, which causes sympathetic and cardiac depression. The safest induction technique for these patients will generally be scopolamine and a muscle relaxant alone.

No single induction technique is suitable for all trauma situations. Often, the medical officer will be confronted with a combination of injuries and conditions that make choosing the best anesthetic-induction technique difficult. Examples would be a patient with a head injury and a full stomach, a patient with an open eye injury and a displaced mandibular fracture, and an uncooperative patient with bleeding and possible cervical spine injury. In situations such as these, the medical officer must decide which conditions are of overriding importance and select the most appropriate airway-management technique and anesthetic plan for those particular conditions.

Emergence

Although modern ophthalmic wound closure can withstand bucking and straining by the patient, it seems best to avoid these on awakening to minimize the risk of vitreous loss.[3] Deep extubation may be considered in the patient known to have an empty stomach but this is not the usual circumstance, as all battlefield casualties are assumed to have a full stomach. Intravenous lidocaine may be helpful in minimizing the patient's reaction to the endotracheal tube. A dose of 1.5 mg/kg should be used; this seldom causes prolonged emergence. Other measures that may be considered include emptying the stomach by gastric tube while the patient is still paralyzed and anesthetized, and using an antiemetic such as droperidol.

INTERACTIONS WITH OTHER DRUGS

Many of the ophthalmic agents introduced into the eye may be absorbed systemically, resulting in cardiovascular or metabolic effects. Timolol is a β-adrenergic blocking agent used in the treatment of glaucoma. The use of this agent may cause systemic beta-blockade, with bronchospasm or bradycardia possible. It is important that the military trauma anesthesiologist be aware of this fact, but, in most cases, timolol certainly does not need to be discontinued preoperatively. The ophthalmologist may apply phenylephrine eyedrops to dilate the pupil perioperatively. Concentrations as high as 10% are available; however, occasional systemic manifestations occur (hypertension or bradycardia or both). Caution in use is advised. Perhaps a diplomatic request to the surgeon to use the lowest effective concentration may be made. An ophthalmic drug very interesting to the anesthesiologist is the miotic agent Phospholine Iodide. This long-acting pseudocholinesterase inhibitor may still have systemic effects up to 6 weeks after discontinuation.[37] This drug prolongs the effects of succinylcholine, trimetaphan, and ester local anesthetics, all of which depend on pseudocholinesterase for termination of action.

PERIOPERATIVE OPHTHALMIC COMPLICATIONS

Corneal Abrasion

The most common perioperative ophthalmic complication is corneal abrasion, to which two factors contribute: basal tear production is greatly reduced, and the protective eyelid closure reflex is lost under general anesthesia. The corneal abrasion will often occur in the inferior one third of the globe, which is the area exposed when the eyelids are allowed to remain partially open. Preventive measures to avoid corneal abrasions include closure of the eyelids and instillation of some type of artificial tears. Many techniques have been suggested to keep the eyelids closed, including taping, suturing, or patching the eyelids. Aqueous artificial tear solutions have to be reapplied during long procedures, as they are not long retained in the eye. For this reason, bland ointments are more commonly used, although there may be a small likelihood of allergic reactions to preservatives used in these preparations.[6]

Acute Angle-Closure Glaucoma

In postoperative patients, it may be important to distinguish the painful eye associated with a corneal abrasion from that occurring with acute glaucoma. The corneal abrasion often is accompanied by pain aggravated by blinking and rapid eye movement, tearing, and the sensation of a foreign body. An area of dullness may be seen on examination. In acute glaucoma, the cornea may be dull and reddened, and the pupil may be dilated. The patient may complain of headache and pain around the eye. A loss of vision may be recorded.

There are two types of glaucoma: open angle and closed angle. Both have the common feature of abnormally elevated IOP. In closed-angle glaucoma, the iris is bulged or folded forward, blocking the aqueous flow into the trabecular meshwork. An acutely swollen lens or pupillary dilation may result from this aqueous humor obstruction. Anesthetic care of such patients includes instilling miotic agents,[8] which constrict the pupil and promote aqueous drainage; avoiding venous congestion (eg, keep the patient's head up, do not allow the patient to perform the Valsalva maneuver, and prevent the patient from bucking or coughing)[19]; and carefully managing intravenous fluid administration to prevent overhydration. Neither atropine nor glycopyrrolate in the usual premedication dosages have been found to worsen IOP with either type of glaucoma; nor have they been implicated when used in combination with anticholinesterases for reversal of neuromuscular blockade.[8] However, scopolamine has been found to have a greater mydriatic effect than atropine and should not be used in patients with glaucoma. This effect of scopolamine may be more problematic in white than in black patients.[49] Open-angle glaucoma is associated with scarring or endothelial thickening of the trabecular meshwork, resulting in obstruction to aqueous humor outflow. Anesthetic considerations are the same as with closed-angle glaucoma with the addition of avoiding hypotension, because these patients are prone to thrombosis of the retinal artery.

Supraorbital Nerve Compression

Another potential postoperative complication is supraorbital nerve compression. This may occur due to improper positioning of the patient or incorrect placement of the face mask or mask straps during anesthesia. The patient may complain of periorbital numbness and edema may be noted. Fortunately, this problem usually resolves without treatment, although it may require some weeks.

Retinal Artery Thrombosis

A much worse complication is retinal artery thrombosis. This complication is more likely to occur in the patient with elevated IOP (eg, in a patient with glaucoma). Retinal artery thrombosis has also been associated with the use of deliberate hypotension.[6] It is most important to ensure that no pressure is applied to the eye of the anesthetized patient, such as occurs in the prone position.

Oculocardiac Reflex

An oculocardiac reflex has been described and is occasionally seen in patients who are undergoing eye surgery. This reflex is elicited by traction on the extraocular muscles, pressure on the globe, pinching of the conjunctiva, or placement of a retrobulbar block. The afferent limb of this reflex involves the ciliary nerves and ganglion, the ophthalmic branch of the trigeminal nerve, the trigeminal ganglion, and, finally, the main sensory nucleus of the trigeminal nerve near the fourth ventricle.[6] The efferent impulses are conducted down the vagus nerve, resulting in bradycardia or arrhythmias such as

junctional rhythm, atrioventricular block, ventricular bigeminy, or even cardiac standstill. This reflex is seen more frequently in children than in adults. Furthermore, hypercarbia or hypoxemia may increase the likelihood or severity of the response to the eliciting stimulus. Usual premedication doses of atropine given intramuscularly are of no value in preventing this reflex. However, intravenously administered atropine or glycopyrrolate, given prior to the application of a stimulus, modifies or prevents the response. Routine use is controversial in the adult and is seldom done. Rather, most anesthesiologists take advantage of the easy fatigability of this reflex in the management of bradycardia or arrhythmias resulting therefrom. Asking the surgeon to halt manipulations long enough for the reflex to diminish and then proceeding is feasible. Adequacy of oxygenation and ventilation should be checked. If the reflex recurs or is severe, then intravenous atropine may be given.[37]

SUMMARY

Penetrating missiles in the form of small fragments are the most common cause of eye injuries in combat casualties who require the services of military trauma anesthesiologists. Not only does the missile itself directly destroy ocular tissue, but the wound tract into the globe also serves as an opening through which intraocular contents may be extruded, causing irreversible injury. This secondary complication can occur when IOP is acutely elevated by coughing, bucking, or straining, and its occurrence has also been reported during intubation and emergence from anesthesia. IOP may also be altered pharmacologically. Miotic agents, barbiturates, inhalational anesthetic agents, and nondepolarizing muscle relaxants all decrease IOP, while an increase follows the use of succinylcholine.

The great majority of combat casualties with eye injuries will require general endotracheal anesthesia; for this, the thiopental-pancuronium induction sequence is the standard of choice, as it minimizes the risk of an acute increase in IOP. Bucking and straining during extubation should be avoided, but deep extubation is not usually possible because each battlefield casualty is assumed to have a full stomach.

Military trauma anesthesiologists need to be aware of the potential for such perioperative complications as corneal abrasions, acute angle-closure glaucoma, supraorbital nerve compression, retinal artery thrombosis, and the oculocardiac reflex. Careful planning and attention to detail by the military anesthesiologist will prevent or reduce loss of vision and promote the rapid return to duty of soldiers with eye injuries.

REFERENCES

1. LaPiana F. Colonel, Medical Corps, US Army, Walter Reed Army Medical Center, Washington, DC. Unpublished analysis prepared for the Center of Military History, US Department of the Army, Washington, DC, 1985.

2. Bellamy RF. Colonel, Medical Corps, US Army, Walter Reed Army Medical Center, Washington, DC. Unpublished analysis of Wound Data and Munitions Effectiveness Team database. Personal communication, June 1994.

3. Holloway KB. Control of the eye during general anaesthesia for intraocular surgery. *Br J Anaesth*. 1980;52:671–679.

4. Samuel JR, Beaugie A. Effect of carbon dioxide on the intraocular pressure in man during general anaesthesia. *Br J Ophthal*. 1974;58:62–67.

5. Adams AK, Barnett KC. Anaesthesia and intraocular pressure. *Anaesthesia*. 1966;21:202–210.

6. McCammon RL. Anesthetic considerations in the presence of ocular disease. In: *American Society of Anesthesiologists Refresher Courses in Anesthesiology*. Philadelphia, Pa: JB Lippincott; 1980: 127–137.

7. Cunningham AJ, Barry P. Intraocular pressure—physiology and implications for anaesthetic management. *Can Anaesth Soc J*. 1986;33:195–208.

8. Adams AK, Jones RM. Anaesthesia for eye surgery: General considerations. *Br J Anaesth*. 1980;52:663–669.

9. Schreuder M, Linssen GH. Intra-ocular pressure and anaesthesia. *Anaesthesia*. 1972;27:165–170.

10. Runciman JC, Bowen-Wright RM, Welsh NH, Downing JW. Intra-ocular pressure changes during halothane and enflurane anaesthesia. *Br J Anaesth*. 1978;50:371–374.

11. Magora F, Collins VJ. The influence of general anesthetic agents on intraocular pressure in man. *Arch Ophthalmol*. 1961;66:806–811.

12. Oji EO, Holdcroft A. The ocular effects of etomidate. *Anaesthesia*. 1979;34:245–249.

13. Lerman J, Kiskis AA. Lidocaine attenuates the intraocular pressure response to rapid intubation in children. *Can Anaesth Soc J*. 1985;32:339–345.

14. Yoshikawa K, Murai Y. The effect of ketamine on intraocular pressure in children. *Anesth Analg*. 1971;50:199–202.

15. Peuler M, Glass DD, Arens JF. Ketamine and intraocular pressure. *Anesthesiology*. 1975;43:575–578.

16. Ausinsch B, Rayburn RL, Munson ES, Levy NS. Ketamine and intraocular pressure in children. *Anesth Analg*. 1976;55:773–775.

17. Katz RL, Eakins KE. The actions of neuromuscular blocking agents on extraocular muscle and intraocular pressure. *Proc R Soc Med*. 1969;62:1217–1220.

18. Pandey K, Badola RP, Kumar S. Time course of intraocular hypertension produced by suxamethonium. *Br J Anaesth*. 1972;44:191–195.

19. Carballo AS. Succinylcholine and acetazolamide (Diamox) in anaesthesia for ocular surgery. *Can Anaesth Soc J*. 1965;12:486–498.

20. Donlon JV. Anesthesia factors affecting intraocular pressure. *Anesthesiol Rev*. 1981;8:13–18.

21. Cook JH. The effect of suxamethonium on intraocular pressure. *Anaesthesia*. 1981;36:359–365.

22. Meyers EF, Krupin T, Johnson M, Zink H. Failure of nondepolarizing neuromuscular blockers to inhibit succinylcholine-induced increased intraocular pressure. *Anesthesiology*. 1978;48:149–151.

23. Bowen DJ, McGrand JC, Hamilton AG. Intraocular pressures after suxamethonium and endotracheal intubation. *Anaesthesia*. 1978;33:518–522.

24. Bowen DJ, McGrand JC, Palmer RJ. Intraocular pressures after suxamethonium and endotracheal intubation in patients pretreated with pancuronium. *Br J Anaesth*. 1976;48:1201–1205.

25. Meyers EF, Singer P, Otto A. A controlled study of the effect of succinylcholine self-taming on intraocular pressure. *Anesthesiology*. 1980;53:72–74.

26. Smith B, Babinski M, Leano N. The effect of lidocaine on succinylcholine-induced rise in intraocular pressure. *Can Anaesth Soc J*. 1979;26:482–483.

27. Tattersall MP, Manus NJ, Jackson DM. The effect of atracurium or fazadinium on intra-ocular pressure. *Anaesthesia*. 1985;40:805–807.

28. Balamoutsos NG, Tsakona H, Kanakoudes PS, Iliadelis E, Georgiades CG. Alcuronium and intraocular pressure. *Anesth Analg*. 1983;62:521–523.

29. Couch JA, Eltringham RJ, Magauran DM. The effect of thiopentone and fazadinium on intraocular pressure. *Anaesthesia*. 1979;34:586–590.

30. George R, Nursingh A, Downing JW, Welsh NH. Non-depolarizing neuromuscular blockers and the eye: A study of intraocular pressure. *Br J Anaesth*. 1979;51:789–792.

31. Maharaj RJ, Humphrey D, Kaplan N, et al. Effects of atracurium on intraocular pressure. *Br J Anaesth*. 1984;56:459–462.

32. Schneider MJ, Stirt JA, Finholt DA. Atracurium, vecuronium and intraocular pressure in humans. *Anesth Analg*. 1986;65:877–882.

33. Lerman J, Kiskis AA. Lidocaine attenuates the intraocular pressure response to rapid intubation in children. *Can Anaesth Soc J*. 1985;32:339–345.

34. Ramsay RC, Knobloch WH. Ocular perforation following retrobulbar anesthesia for retinal detachment surgery. *Am J Ophthalmol*. 1978;86:61–64.

35. Barr AM, Thornley BA. Thiopentone and suxamethonium crash induction. *Anaesthesia*. 1976;31:23–29.

36. Barr AM, Thornley BA. Thiopentone and pancuronium crash induction. *Anaesthesia*. 1978;33:25–31.

37. McGoldrick KE. Current concepts in anesthesia for ophthalmic surgery. *Anesth Rev*. 1980;7:7–16.

38. Abbott MA, Samuel JR. The control of intra-ocular pressure during the induction of anaesthesia for emergency eye surgery. *Anaesthesia*. 1987;42:1008–1012.

39. Lennon RL, Olson RA, Gronert GA. Atracurium or vecuronium for rapid sequence endotracheal intubation. *Anesthesiology*. 1986;64:510–513.

40. Mehta MP, Choi WW, Gergis SD, Sokoll MD, Adolphson AJ. Facilitation of rapid endotracheal intubations with divided doses of nondepolarizing neuromuscular blocking drugs. *Anesthesiology*. 1985;62:392–395.

41. Schwarz S, Ilias W, Lackner F, Mayrhofer O, Foldes FF. Rapid tracheal intubation with vecuronium: The priming principle. *Anesthesiology*. 1985;62:388–391.

42. Naguib M, Abdulatif M, Gyasi HK, Absgood GH. Priming with atracurium: Improving intubating conditions with additional doses of thiopental. *Anesth Analg*. 1986;65:1295–1299.

43. Sosis M, Larijani G, Marr AT. Priming with atracurium. *Anesth Analg*. 1987;66:329–332.

44. Libonati MM, Leahy JJ, Ellison N. The use of succinylcholine in open eye surgery. *Anesthesiology*. 1985;62:637–640.

45. Weiner MJ, Olk RJ, Meyers EF. Anesthesia for open eye surgery. *Anesthesiology*. 1986;65:109–110.

46. Calobrisi BL, Lebowitz P. Muscle relaxants and the open globe. *Int Anesthesiol Clin*. 1990;28:83–87.

47. Morton NS, Hamilton WFD. Alfentanil in an anaesthetic technique for penetrating eye injuries. *Anaesthesia*. 1986;41:1148–1151.

48. Zaturansky B, Hyams S. Perforation of the globe during the injection of local anesthesia. *Ophthalmic Surg*. 1987;18:585–588.

49. Garde JF, Aston R, Endler GC, Sison OS. Racial mydriatic response to belladonna premedication. *Anesth Analg*. 1978;57:572–576.