Chapter 5

ANESTHETIC CARE OF THE TRAUMATIZED EYE

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INTRODUCTION

Ocular injuries account for a significant percentage of combat injuries, even though the eyes account for only 0.27% of the body's total surface area, 0.54% of the body's total frontal silhouette, and 4% of the surface area of the face.[1] The incidence of ocular injury in combat is 20 to 50 times greater than that expected by its surface area alone.[2] This differential is largely the result of the increased exposure of the head and eyes during combat and the ease with which the eye can be injured by such seemingly innocuous mechanisms as windblown foreign bodies. Many of these foreign bodies would be easily stopped by the skin or clothing elsewhere but can incapacitate a soldier if they strike the eye.

The medical literature has shown that the rate of ocular injuries during combat has steadily increased since the American Civil War.[3–8] Ocular injuries accounted for 0.57% of casualties during the American Civil War, 2% of casualties during World War I and World War II, and nearly 3% during the Korean War. The Israeli experience from 1967 until 1982 showed rates that started at 5.6% during the 1967 Arab–Israeli Six-Day War and increased to 6.8% during the war in Lebanon. This parallels the US experience during the Vietnam War, in which 5% to 9% of the casualties were ocular. Finally, in the Persian Gulf War (1991/92), ocular injuries accounted for up to 13% of all war injuries. These rates indicate the need for a significant number of well-trained medical personnel who are experienced with the complexities of the care of the injured eye. This includes not only modern ophthalmic surgical techniques but also the subtleties and intricacies of providing adequate anesthesia.

Interested readers may find additional information on anesthesia for eye trauma in another volume in the Textbooks of Military Medicine series, *Anesthesia and Perioperative Care of the Combat Casualty,* particularly Chapter 17, Eye Injuries.[9]

PREOPERATIVE ASSESSMENT

The preoperative assessment of the patient injured in combat must first identify and treat any life-threatening condition. The algorithms published for Advanced Cardiac Life Support and Advanced Trauma Life Support are extremely effective in identifying and treating such conditions in a systematic way. This is especially important in a patient who has multisystem trauma, a common occurrence on today's battlefield. Patients with ocular trauma often have midfacial trauma as well. Such trauma can significantly complicate airway management and may require early intervention by an anesthesiologist or an otolaryngologist for definitive airway control. A cribriform plate injury with cerebrospinal fluid leak must also be considered when ocular and midface trauma are present. Nasotracheal intubation and the placement of nasogastric tubes should be avoided in patients with cribriform plate injury to prevent inadvertent intracranial placement.

Once the initial ABCs of airway, breathing, and circulation have been stabilized, a more definitive examination of the eye and ocular adnexa can be performed. If the patient has other life-threatening injuries, the ophthalmologist may be able to perform only a cursory examination, with the main goal of determining whether an open (ie, penetrating) globe injury is present or suspected. The most important ocular consideration before the induction of anesthesia is the integrity of the eye. If an ocular examination cannot be performed before life-threatening injuries must be managed, the injured eye should be presumed to have an open globe injury. A metal protective shield should be placed so that no pressure is exerted on the eye, and anesthesia should be induced to minimize the risk of extrusion of the intraocular contents.

The importance of a careful preanesthetic evaluation cannot be overemphasized. The importance of the integrity of the eye has already been discussed; other important considerations include the presence of preexisting disease, prior anesthesia and surgery, current medications used chronically or given in the posttrauma time frame, drug allergies, and physical examination. Most patients who sustain injury in combat are young and otherwise healthy without underlying disease; however, cardiovascular and pulmonary dysfunction can occur with multisystem trauma or exposure to chemical or biological warfare agents.

A common anesthetic problem in acute trauma is a full stomach. Combat-injured patients should all be treated as if they have full stomachs. In the setting of ocular trauma in which an open globe is verified or suspected, mechanical gastric emptying with nasogastric or orogastric tubes should be avoided because placement of the tubes may evoke coughing and vomiting, which could result in ex-

pulsion of the intraocular contents. Neutralization of gastric acid should be undertaken in all patients to minimize the risk of aspiration pneumonitis. Sodium citrate 0.3 molar (30–40 mL, administered orally) given 30 minutes before induction can raise the gastric contents' pH but at the expense of a small increase in gastric volume. Cimetidine (300 mg, orally, or 150 mg, intramuscularly) or ranitidine (150 mg, orally, or 50 mg, intramuscularly) given 90 minutes before induction can also provide some protection. Additionally, these agents do not appear to affect the intraocular pressure (IOP) of the closed eye.[10] Metoclopramide (10 mg, intravenously) may reduce the volume of the stomach contents by promoting gastric emptying. However, preliminary data demonstrate this drug's tendency to raise IOP[11]; it should, therefore, be used with caution in open globe injuries.

TOPICAL ANESTHESIA

Topical anesthesia is commonly used during routine ophthalmic examinations and procedures. It allows a more complete examination, especially when corneal epithelial defects are present or when such procedures as lacrimal probing and irrigation, nasal examination, and forced duction testing are used to obtain more detailed information concerning the anatomy or physiology of the injured area. To add to patient comfort, topical anesthesia is also useful before facial preparation. If an open globe is suspected and topical anesthetic is required, care should be taken to minimize the amount used and to ensure that the drops are sterile.

Several topical anesthetic agents are available for use during ophthalmic examinations and procedures (Table 5-1). Proparacaine is an ester preparation available in 0.5% solution. It is quickly absorbed through the corneal epithelium because of its high lipid solubility and thus causes less discomfort than a more hydrophilic solution such as lidocaine.[12] Long-term use can result in corneal epithelial toxicity and delayed corneal healing. Local-

TABLE 5-1

COMMONLY USED TOPICAL AND LOCAL ANESTHETICS IN OPHTHALMIC SURGERY

Kind of Agent	Concentration	Max. Dose	Onset	Duration	Comments
Topical:					
Proparacaine	0.5%	—	Seconds	10–20 min	Any topical anesthetic can cause superficial punctate keratitis[1]
Tetracaine	0.5%	—	Seconds	10–20 min	
Lidocaine	1%–4%	—	Seconds	10–20 min	
Regional:					
Lidocaine	1%–2%	500 mg[1]	4–6 min	40–60 min	Least painful on injection[2]
Mepivacaine	1%–2%	500 mg[1]	3–5 min	2–3 h	Duration of action greater without epinephrine[3]
Bupivacaine	0.25%–0.75%	23 mL of 0.75% solution[2]	5–11 min	3–12 h	Most painful on injection[2]
Adjuvant:					
Epinephrine	1:100,000 to 1:200,000	—	—	—	Increases duration of action of all except mepivacaine[3]
Hyaluronidase	150 U per vial	Standard dose is 150 U/10 mL of local	—	—	Can decrease duration of action of local[2]
Sodium Bicarbonate	8.4% (1 meq/mL)	Standard dose is 1 mL in 10 mL of local	—	—	Can decrease pain on injection[2]

1. Medical Economics Data. *Physicians' Desk Reference for Ophthalmology*. 22nd ed. Montvale, NJ: Medical Economics Co; 1994: 9.
2. Bilyk JR, Sutula FC. Anesthesia for ophthalmic plastic surgery. In: Stewart WB, ed. *Surgery of the Eyelid, Orbit, and Lacrimal System*. Vol 1. San Francisco, Calif: American Academy of Ophthalmology; 1993: 33.
3. Everett WG, Vey EK, Finlay JW. Duration of oculomotor akinesia of injectable anesthestics. *Trans Am Acad Ophthalmol*. 1961;65:308.

ized allergic reactions have also been reported; if a reaction occurs, tetracaine, another ester preparation in 0.5% solution, can be substituted.[13] However, tetracaine is more toxic to the epithelium and has significantly more systemic toxicity. Fatalities with excessive topical use have been reported.[14]

Cocaine is another ester derivative that not only provides excellent topical anesthesia but also causes vasoconstriction by preventing the reuptake of norepinephrine.[15] This added property of cocaine makes it an excellent agent when examination or manipulation of the nasal mucosa is required. Before lacrimal drainage surgery, the nasal cavity is usually packed with neurosurgical cottonoids soaked in 4% cocaine solution. Care must be taken to place the cottonoids directly against the nasal mucosa and in the location of the anterior middle meatus where the nasal mucosa will be opened. Cocaine is toxic to the corneal and conjunctival epithelium, and its use in the eye is usually limited to detecting the sympathetic dysfunction of Horner's syndrome. A drop or two of the 2% solution can confirm the diagnosis of Horner's syndrome when less dilation is noted on the affected side. When cocaine is used, the vital signs must be monitored closely because hypertension, tachycardia, and ventricular dysrhythmias can occur. Concomitant use of other systemic agents, such as monoamine oxidase inhibitors and tricyclic antidepressants, can potentiate this effect and require extra vigilance.[16] Additionally, because cocaine is detoxified by plasma and liver cholinesterases, persons with cholinesterase deficiencies are at risk for sudden death from the use of cocaine.[17]

Lidocaine is an amide preparation that recently has been used more frequently as a topical anesthetic agent. In the past it was used mostly as a local injectable anesthetic agent. However, the 4% solution used topically can provide enough anesthesia to perform cataract surgery or allow for a much more comfortable examination. It is particularly helpful when performing forced duction testing and when probing and irrigating the nasolacrimal system. A pledget can be fashioned from the tip of a cotton-tipped applicator and soaked in a 2% or 4% lidocaine solution. It can then be placed either over the muscle insertion or over the lacrimal punctum for several minutes before the procedure is performed.

EMLA Cream (an emulsion of lidocaine 2.5% and prilocaine 2.5%, mfg by AstraZeneca LP, Wilmington, Del) is a topical agent that may be an alternative to local anesthesia of the skin. It is not suitable for use in the eye but can be used in the periocular region if care is taken to avoid contact with the ocular surface. EMLA Cream is particularly useful for providing anesthesia before venipuncture. It can also be used for superficial surgical procedures, such as removal of superficial foreign bodies or laser skin resurfacing, but anesthesia can only be achieved to a depth of approximately 5 mm. EMLA Cream has a relatively long onset of action and should be applied 1 hour before the planned procedure.[17]

LOCAL ANESTHESIA

All local anesthetics inhibit sodium ion influx across neuronal cell membranes and produce a blockade of the nerve impulse. However, not all neuronal functions are affected by local anesthetics in equal fashion. The rates of blockade of the components of a peripheral nerve occur at different speeds with loss of sympathetic function first, followed by a pin-prick sensation, touch, temperature, and finally motor function. The reason for this differential blockade is not totally clear but may have to do with small or nonmyelinated fibers being affected more quickly than large or myelinated fibers.[17]

Local anesthetics are benzoic acid derivatives and can be separated into two different groups on the basis of whether there is an amide or ester link between the lipophilic head and the hydrophilic tail.[17] Amides and esters differ in several respects. Amides are metabolized in the liver, whereas esters require a plasma pseudocholinesterase for breakdown. Amides cause fewer allergic reactions than esters do, but are more toxic.[15] Allergic cross-reactions between the ester and amide groups do not occur, so if an individual is allergic to one group of agents, it may be possible that the other group can be used safely. Commonly used agents that are members of the ester group are tetracaine, cocaine, and procaine. The amide group includes lidocaine, mepivacaine, and bupivacaine.

Specific Local Anesthetic Agents

Lidocaine is the most commonly used local anesthetic agent and is available in 1% and 2% solutions (see Table 5-1). It produces the least pain on injection and has a rapid onset of action and a mod-

erate duration of action.[18] Mepivacaine is available in 1%, 2%, and 3% solutions and, like lidocaine, has a rapid onset of action and moderate duration. Unlike lidocaine, however, it has no topical activity.[19] Bupivacaine is a more potent agent and is more toxic, probably because of its increased lipid solubility. It is available in 0.5% and 0.75% solutions and has a delayed onset of action and longer duration of action. One of the shorter-acting agents can be combined with bupivacaine to facilitate rapid onset of action and prolonged duration. This is commonly done when performing retrobulbar blocks, when postoperative analgesia is necessary, or when a longer procedure is anticipated.

Adjuvant Agents

Several adjuvant agents (see Table 5-1) can be added to any of the local anesthetics. One commonly added agent is epinephrine, which has several beneficial effects, including

- prolonging the duration of anesthesia,
- minimizing the peak level of local anesthetic in the blood,
- increasing the intensity of the blockade, and
- reducing surgical bleeding.

Epinephrine concentrations from 1:100,000 to 1:400,000 are available. One study[20] showed that decreasing the concentration from 1:200,000 to 1:400,000 caused the same vasoconstrictive result locally. Using lower concentrations might minimize the potential for systemic toxicity, including tachycardia, hypertension, and dysrhythmia. Epinephrine should be used cautiously in patients with unstable angina, malignant dysrhythmias, uncontrolled hypertension, or hyperthyroidism, and in patients taking monoamine oxidase inhibitors and tricyclic antidepressants, which can enhance the effects of catecholamines. To prevent tissue necrosis, epinephrine should also be used with caution in areas with poor collateral blood flow.

Hyaluronidase is another adjuvant often added to local anesthetics. Hyaluronic acid inhibits the diffusion of foreign substances in interstitial spaces. Hyaluronidase, on the other hand, depolymerizes hyaluronic acid and facilitates the spread of local anesthetics,[17] which can hasten their onset of action. However, hyaluronidase significantly shortens the duration of action and may cause diffusion of the anesthetic agent into undesirable locations. If a local anesthetic agent with hyaluronidase is used in the upper lid, the drug may diffuse into the levator muscle, paralyzing it and making intraoperative adjustment of lid height difficult. If hyaluronidase is used, 150 units (U) can be added to 10 mL of local anesthetic.

Sodium bicarbonate can also be added to local anesthetics to raise the pH and increase the concentration of nonionized free base. In theory, these actions increase the rate of diffusion and speed the onset of action.[17] Raising the pH has also been shown to decrease the pain of injection when 1 mL of 1 mEq/mL sodium bicarbonate solution is added to 10 mL of local anesthetic, for a final concentration of 0.1 mEq/mL.[21]

Adverse Reactions to Local Anesthetic Agents

Local anesthetic agents present a continuum of toxic effects as systemic blood concentrations increase. The earliest signs of toxicity may include numbness of the tongue, lightheadedness, visual disturbances, and muscle twitching. Further progression results in central nervous system (CNS) signs such as unconsciousness, convulsions, and coma. Finally, the cardiovascular system collapses, with respiratory arrest and refractory dysrhythmias.

Treatment of local anesthetic toxicity is mainly supportive, with the immediate administration of oxygen and the use of an agent to stop seizure activity. The first item recommended is succinylcholine to facilitate ventilation. Then, either a barbiturate or benzodiazepine can be given, as tolerated by the cardiovascular system, to reduce CNS metabolic demands. To treat cardiovascular effects, high-dose epinephrine may be needed to support the heart rate and blood pressure. Atropine may be needed to treat bradycardia, and ventricular dysrhythmias should be treated with an agent like bretylium tosylate instead of lidocaine.[17]

The best treatment for toxic reactions is prevention. The toxic dose of the agent being used must be known. For 2% lidocaine without epinephrine, the maximum recommended dose is 15 mL, and for 2% lidocaine with epinephrine, it is 25 mL. The maximum recommended dose of 0.75% bupivacaine is 23 mL.[12] Other preventive measures include using meticulous technique to avoid intravascular administration and assessing for individual risk factors (eg, liver failure, pseudocholinesterase deficiencies) that could slow the metabolism of the agent.

TECHNIQUES FOR ANESTHETIZING THE OCULAR ADNEXA

Several techniques are available to adequately anesthetize the ocular adnexa after injury. These techniques are usually a combination of subcutaneous regional infiltration, field block, and nerve block. When any of these techniques are used, we must remember that a sharp needle tip is close to the globe. It is advisable to place a plastic globe protector over the eye when possible to reduce the risk of inadvertent penetration. A globe protector also decreases the discomfort often reported from the bright operating room lights.

Subcutaneous Regional Infiltration

Subcutaneous regional infiltration is easily administered because it requires the least amount of knowledge of neuroanatomy. When this method is used, the surgeon must remember that the motor and sensory nerves run deep to the orbicularis muscle; therefore, the anesthetic must be injected in this plane. Injection in this plane also facilitates surgical dissection, because hydraulic dissection has already occurred.

Field and Nerve Blocks

Successful administration of field and nerve blocks requires much more detailed understanding of the neuroanatomy of the ocular adnexa. A field block is defined as infiltration of anesthetic into tissue to block neural transmission and provide anesthesia to distal tissue. Nerve blocks, in contrast, involve the injection of anesthesia directly around a nerve to provide anesthesia to an area supplied by that nerve. These two techniques often meld into one in the ocular adnexa because the nerves are often in close proximity to each other and because there is significant redundancy of sensory innervation.[12]

Neuroanatomy for Ocular Adnexal Blocks

The sensory nerve supply to the ocular adnexa is provided by the first two branches of the trigeminal nerve (Figure 5-1). The first branch, known as the ophthalmic division, enters the orbit via the superior orbital fissure and has three branches: the

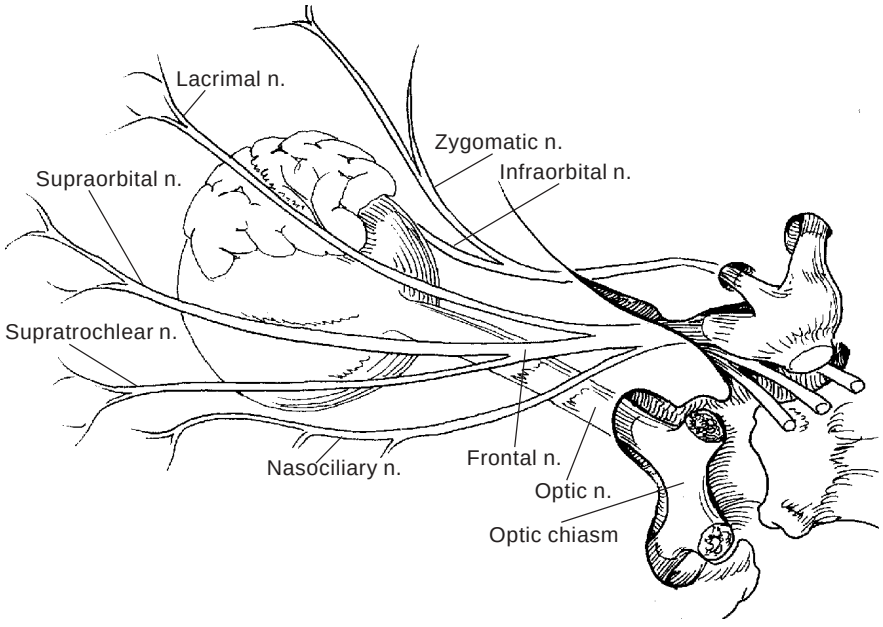

Fig. 5-1. This drawing illustrates the courses of the branches of the trigeminal nerve. It also shows the course of the optic nerve and the position of the optic chiasm. Fully understanding the anatomy of the sensory supply to the orbit and orbital adnexa will allow the development of a standardized approach to local anesthetic and infiltrative blocks. Drawing prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

lacrimal, the frontal, and the nasociliary.[22] The lacrimal nerve supplies sensation to the lacrimal gland and to the skin of the lid and periorbital region superolaterally. The frontal nerve runs forward in the roof of the orbit just under the periorbita. It divides into the supraorbital and supratrochlear nerves, which supply sensation to the skin and deeper tissues of the lid and periorbital regions in the superonasal and frontal areas. The nasociliary nerve gives off sensory fibers to the ciliary ganglion and then passes above the optic nerve, where long ciliary nerves branch to the globe. It continues forward superiorly and nasally in close proximity to the ophthalmic artery, dividing into the anterior ethmoidal nerve, the posterior ethmoidal nerve (often not present), and the infratrochlear nerve. The ethmoidal nerves supply sensation to the nasal mucosa. The infratrochlear nerve supplies sensation to the side and tip of the nose and the lacrimal sac and canaliculi.[22]

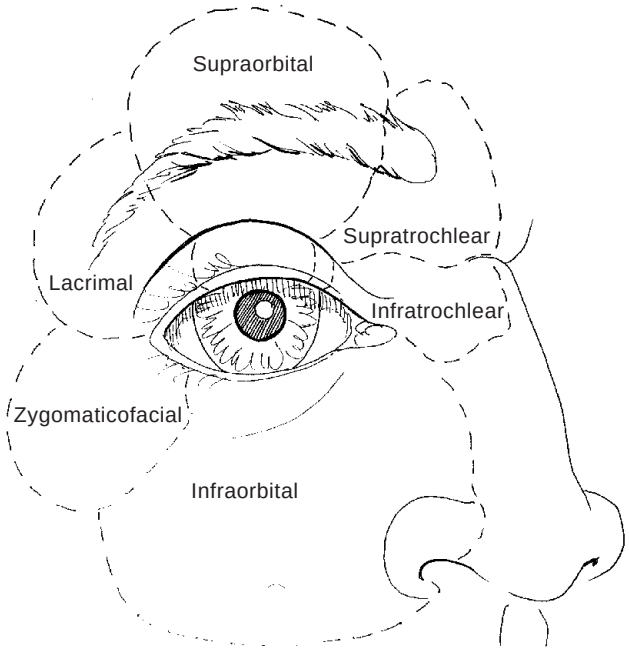

Fig. 5-2. This drawing illustrates the occular adnexal sensory innervation. Each sensory nerve is responsible for providing sensation to a certain area (seen above), although there is some overlap in innervation. An understanding of this nerve supply is the basis for the local anesthetic techniques and nerve blocks utilized. Drawing prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

The maxillary nerve is the second division of the trigeminal nerve and courses forward through the foramen rotundum. The zygomatic nerve is a branch that subdivides into the zygomaticofacial and zygomaticotemporal nerves.[22] These sensory branches exit through foramina in the lateral wall of the orbit and supply sensation to the skin of the lateral orbit. The infraorbital nerve is the terminal branch; it runs along the orbital floor, supplying sensation to skin of the lower lid, the upper lip, and some teeth (Figure 5-2).

Fully understanding the anatomical relationships of these nerves as they exit the orbit allows for successful anesthetic infiltration. Four of the nerves are palpable as they leave the orbit: the supraorbital, the infraorbital, the supratrochlear, and the infratrochlear.[23] The supraorbital foramen is usually located 2.7 cm lateral to the midline of the glabella, and the supratrochlear and infratrochlear nerves are 1.7 cm lateral to the midline. The infraorbital nerve exits approximately 1 cm below the inferior orbital rim in a vertical line drawn from the supraorbital notch.[23] To effectively block these nerves' sensory distribution, a few milliliters of local anesthetic can be infiltrated around their points of exit from the orbit. When injecting the local anesthetic, anesthesia providers should always aspirate before injecting to minimize the risk of an intravascular injection, because these nerves also run with blood vessels (Figure 5-3).

The motor nerve supply to the eyelids and ocular adnexa is from the seventh cranial nerve—the facial nerve—which exits the stylomastoid foramen behind the ramus of the jaw and then courses through the parotid gland. It divides into five branches: temporal, zygomatic, buccal, mandibular, and cervical. The temporal and zygomatic branches innervate the orbicularis muscle of the upper and lower lid respectively, allowing for full lid closure. Blocking lid closure can allow complete examination in a patient with excessive blepharospasm and can facilitate safer surgery by minimizing squeezing, which can increase IOP.[24]

Block Techniques for the Seventh Nerve Supply

Several techniques can be used to block the seventh cranial nerve supply to the orbicularis muscle (Figure 5-4). The modified van Lint method is performed by placing a needle approximately 1 cm lateral to the lateral orbital rim. The needle is directed in the suborbicularis plane, and anesthetic agent is injected perpendicularly to the skull above the pe-

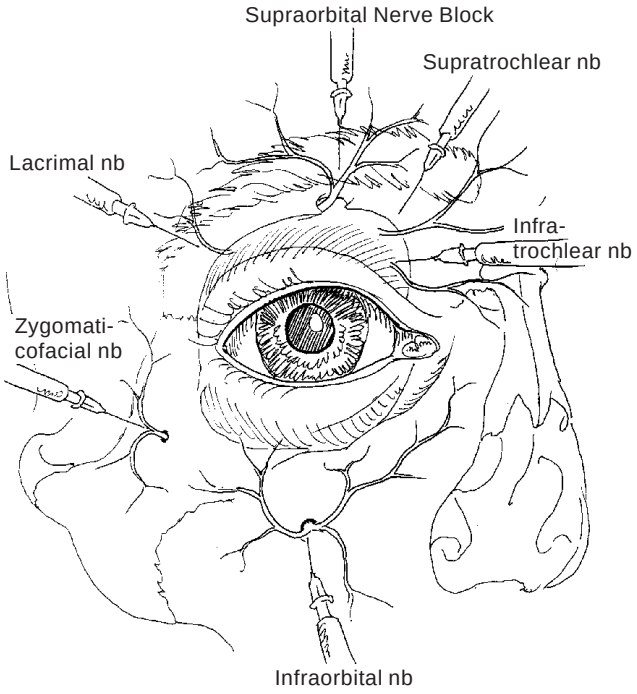

Fig. 5-3. This drawing illustrates the locations that would be used to administer the various ocular adnexal nerve blocks. When administering the blocks it is important to remember that each nerve is also accompanied by vasculature. To avoid intravascular injections of local anesthetic, one should pull back on the syringe before injection to ensure that blood return does not occur and that the needle is not within a blood vessel. Drawing prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

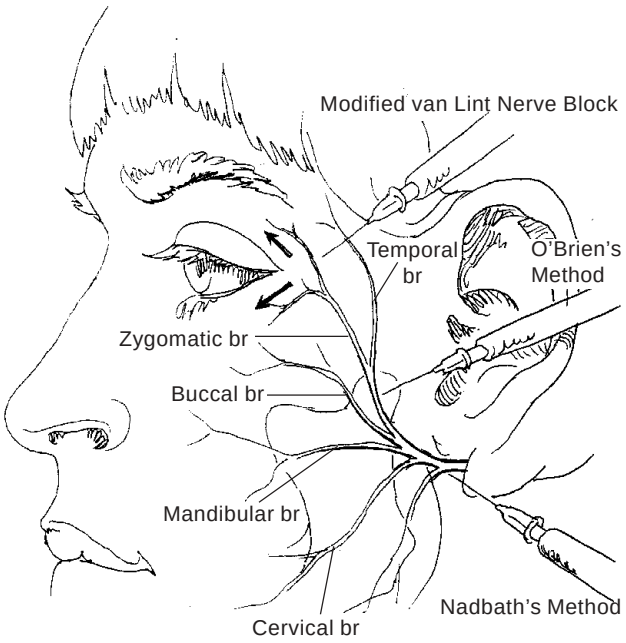

Fig. 5-4. The drawing illustrates both (1) the course of the branches of the seventh cranial nerve, which provides the motor function for the face and for eyelid closure, and (2) the location of the various seventh nerve blocks. The modified van Lint block is the most distal nerve block and causes only eyelid closure weakness. O'Brien's method and Nadbath's method are more proximal and cause increaced facial weakness in addition to eyelid weakness. Drawing prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

riosteum. The needle is then directed in a cephalad and caudad direction while more anesthetic is injected to block the terminal branches of the seventh nerve and avoid blocking other facial muscles. The disadvantages of this method include pain on injection and the possibility of bruising and swelling of the eyelids.

O'Brien's method involves injecting local anesthetic agent just anterior to the tragus of the ear, below the posterior portion of the zygomatic process, and directly over the condyloid process of the mandible. A short needle is used; it goes straight inward until the bony condyloid process is felt, usually 1 cm deep. Two to six milliliters of local anesthetic is injected.[25] The disadvantage of this block is that an incomplete block or a failed block is possible because of variations in the course of the branches of the seventh nerve.

The Nadbath method results in complete hemifacial akinesia because the injection is given over the main trunk of the seventh nerve. The needle is inserted behind the posterior border of the ramus of the mandible in front of the mastoid process. Having the patient open his or her mouth widely can help identify this space. A short needle is advanced in an anterocephalad direction. Three to five milliliters of local anesthetic is injected.[25] Potential complications of this block include hoarseness, dysphagia, pooling of secretions, laryngospasm, respiratory distress, and agitation. These effects are believed to be due to the close proximity of other cranial nerves, including the vagus and glossopharyngeal nerves.[24] This block is usually not needed in the setting of ocular trauma management because paralysis of the lower facial muscles is usually not necessary.

Retrobulbar Block

The retrobulbar block has been used successfully for more than 80 years in intraocular surgery. It can effectively cause globe akinesia and anesthesia by

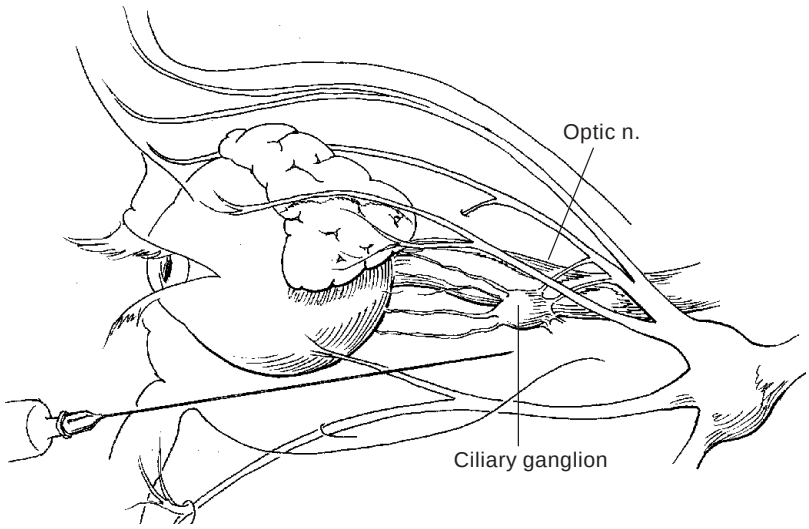

Fig. 5-5. This drawing illustrates the course of the retrobulbar needle during a retrobulbar block. It also illustrates the final position of the needle within the intraconal space. This block will effectively paralyze all the extraocular muscles that receive their nerve supply from within the intraconal space. The only muscle spared is the superior oblique muscle, as it receives its nerve supply from outside the intraconal space. Care must be taken while performing this block not to damage the eye or the optic nerve. Drawing prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

blocking the sensory and motor nerve supply of the intraconal space. However, in the trauma setting it must *only* be used when there is assurance that the globe is not ruptured. If such a block is performed in the setting of an open globe, the pressure used to inject the anesthetic into the orbit can cause extrusion of the intraocular contents.

The retrobulbar block is performed by inserting a 25- or 27-gauge, 1.5-in. needle through the skin of the lower lid directly above the orbital floor at the border of the lateral limbus. The needle is advanced until it has passed the equator of the globe, then it is directed upward and medially to place the tip within the muscle cone (Figure 5-5). Approximately 4 mL of local anesthetic is injected. A 50/50 mixture of 2% lidocaine without epinephrine and 0.75% bupivacaine is commonly used. Often 15 to 20 U of hyaluronidase per milliliter of local anesthetic is added to facilitate diffusion of the local.

The classic Atkinson method called for the patient to look upward and medially while the block was being given. This method has fallen out of favor because several studies have shown that such an eye position places the optic nerve closer to the path of the needle.[25] Most ophthalmologists now have the patient look straight ahead.

Several complications have been reported after retrobulbar anesthesia. Although rare, serious complications, including blindness and death, have occurred. This method should be used only if there is no risk of an open globe injury. If an open globe is even suspected, then general anesthesia should be used. The complications of retrobulbar anesthesia include direct injection of anesthesia into the sheath of the optic nerve, causing blindness from anesthetic toxicity or direct trauma to the nerve. Direct injection into the optic nerve sheath can also cause brainstem anesthesia with respiratory collapse if the anesthetic agent travels to the CNS. Retrobulbar hemorrhages have also been reported that can lead to an orbital compartment syndrome with increased IOP and optic nerve compression. Finally, globe perforation has been reported with resultant retinal detachment and blindness from anesthetic toxicity to the retina.[24]

Several techniques—including peribulbar and parabulbar anesthesia—have been developed since the mid to late 1970s to minimize such complications. In peribulbar anesthesia, an injection is given below the globe in a fashion similar to the technique for retrobulbar anesthesia, except that the needle is not directed upward and inward and more anesthetic agent is injected. This injection is sometimes supplemented with another injection above the globe and below the supraorbital notch. Parabulbar anesthesia is given in the sub-Tenon's fascia plane with a blunt cannula. Unfortunately, complication rates have not diminished significantly, and these alternative blocks do not provide the same level of akinesia as the retrobulbar block does.

MONITORED ANESTHESIA CARE

Monitored anesthesia care (MAC) involves the use of intravenous sedation and analgesia with noninvasive monitoring during a surgical case also involving local anesthetics. MAC has several advantages over straight local anesthesia and general anesthesia. Two major advantages over local anesthesia are the following[26]:

1. MAC's ability to reduce the anxiety of the patient by providing some amnesia and by providing for noninvasive monitoring, which allows the surgeons to concentrate solely on the task at hand while the anesthesiologist delivers the sedation and monitors vital signs; and
2. conversion to general anesthesia is readily available if needed.

The major advantages of MAC over general anesthesia are the following[26]:

1. it is less stressful to the normal body physiology and
2. recovery time is shorter.

Ideally, MAC is used when the patient has had nothing to eat for at least 6 hours. In the trauma setting, a full stomach is assumed so the same techniques described in the preoperative management section to minimize gastric contents should be used if MAC is to be employed.

The most common sedatives used for MAC (Exhibit 5-1) include benzodiazepines (eg, midazolam), narcotics (eg, fentanyl), and other agents (eg, propofol). Benzodiazepines act primarily on the CNS and produce sedation, retrograde amnesia, anxiolysis, and muscle relaxation. These drugs are metabolized in the liver and must be used with care in patients with underlying liver disease. Hypotension and respiratory depression may occur, especially when benzodiazepines are used in combination with narcotics.[27] Midazolam is a widely used benzodiazepine because of its short half-life and because it produces little irritation at the injection site. When administered intravenously, sedation occurs within 3 to 5 minutes. Its peak sedation is seen in about 30 minutes and lasts for approximately 2 hours. It is usually given in small doses starting at 1 mg because the respiratory depressant effects may not manifest immediately.[28] Flumazenil, a newly available benzodiazepine antagonist, can be used to reverse the sedation and respiratory depression caused by agents like midazolam. The recommended dose is 0.2 mg/min, intravenously, with a maximum dose of 3 mg within an hour.[12] After administration of flumazenil, patients should be monitored for at least 2 hours because of the risk of resedation.

Often, a benzodiazepine is given in conjunction with a narcotic such as fentanyl. The main effect sought with the narcotic is analgesia. Narcotics, however, also cause respiratory depression and decreased gastrointestinal motility and nausea. Vital signs must be closely monitored when narcotics are used with or without other agents. Fentanyl is short-acting, with onset of action within 2 minutes. If respiratory depression occurs, assisted ventilation may be required with naloxone. Naloxone is an effective opioid antagonist that is routinely used in doses of 0.1 to 0.2 mg, intravenously, to reverse opioid-induced sedation and respiratory depression.[12] Naloxone can produce its own cardiovascular side effects,

EXHIBIT 5-1

RECOMMENDED DOSAGES OF SELECTED SEDATIVES AND ANALGESICS

Oral
- Diazepam 0.2 mg/kg
- Lorazepam 1–4 mg
- Midazolam 0.3–0.8 mg/kg

Intramuscular
- Diazepam 0.2 mg/kg
- Midazolam 0.07–0.30 mg/kg
- Methohexital 8–10 mg/kg
- Meperidine 1–2 mg/kg
- Morphine Sulfate 0.1–0.2 mg/kg

Intravenous
- Fentanyl 0.5–2.0 μg/kg
- Meperidine 0.5–1.5 mg/kg
- Morphine sulfate 0.05–0.10 mg/kg
- Methohexital 50–1,000 μg/kg load, 15–50 μg/kg/min infusion
- Midazolam 50–100 μg/kg load, 0.6–2.0 μg/kg/min infusion
- Propofol 250–1,000 μg/kg load, 25–75 μg/kg/min infusion

including hypertension, hypotension, acute pulmonary edema, and dysrhythmia. Additionally, patients must be monitored for resedation.

A newer, short-acting sedative–hypnotic that has been found to be extremely useful for MAC is propofol. After injection, hypnosis is usually induced with only one pass through the CNS and takes effect within 2 minutes. Patients are usually wide awake several minutes after a single bolus is given.[28] Propofol can also be given as a continuous drip to maintain sedation throughout a procedure. The usual dosage for MAC is a slow infusion of 0.5 mg/kg over 3 to 5 minutes followed by 1.5 to 4.5 mg/kg/h if continued sedation is desired. The major adverse side effects of propofol include respiratory depression and hypotension. Vital signs must be monitored closely during its use, and ventilatory support should be immediately available.

GENERAL ANESTHESIA

General endotracheal anesthesia is commonly used to provide adequate and safe anesthesia for a patient with multisystem trauma. It is also the only technique available for patients with open globe injuries or presumed globe rupture. The objectives in a patient with an open globe injury include overall patient safety, maintenance of decreased extraocular muscle tone, avoidance of elevation of intraocular volume, and avoidance of external pressure on the eye.[10] As has been discussed above, all trauma patients must be treated as full-stomach encounters and should be pretreated as outlined.

Induction of Anesthesia

The induction of anesthesia, the most critical period of anesthetic management, is encountered after pretreatment. A rapid-sequence induction technique is usually chosen to minimize the risk of aspiration. Nevertheless, each step must be evaluated for its effect on ocular pressure to minimize the risk of extruding the intraocular contents. Several basic techniques are widely accepted to minimize risk during such inductions. The debate continues, however, over the selection and use of neuromuscular blocking agents for facilitating intubation. Widely accepted techniques include the following[29]:

- preoxygenation with care to avoid pressure on the eye from the face mask,
- cricoid pressure during intubation to prevent regurgitation,
- establishment of a deep enough level of anesthesia prior to laryngoscopy to prevent coughing and sudden increases in arterial blood pressure, and
- controlled ventilation after intubation to avoid hypercapnia-associated increases in IOP.

Which neuromuscular blocking agent should be used during induction? The controversy centers mostly on the transient increase in IOP noted with the use of depolarizing agents such as succinylcholine. This effect is due to the initial contraction of the extraocular muscles, which can increase IOP about 8 mm Hg.[10] More-recent clinical studies have challenged the aversion to using depolarizing agents in open globes.[30] Two major disadvantages should be kept in mind when using a very fast-acting agent such as succinylcholine[10]:

1. If a nondepolarizing agent, with its delayed onset, is used, then coughing and straining may occur if intubation is attempted before complete blockade is achieved.
2. Conversely, if enough time is left to ensure complete blockade, then the airway is left unprotected in a situation in which the casualty may well have a full stomach.

Many techniques have been developed to overcome these inherent problems. The techniques include[29]

1. using large doses of nondepolarizing agent to hasten onset,
2. using a priming dose of nondepolarizing agent followed by a larger dose, and
3. pretreatment with a nondepolarizing agent followed by a barbiturate–succinylcholine sequence.

All three techniques have met with variable results, and globe rupture should no longer be considered an absolute contraindication to the use of succinylcholine. The specific muscle relaxant and technique to be used should be decided by the anesthesiologist on a case-by-case basis.

Maintaining Anesthesia

Once the induction is complete, maintenance of adequate anesthesia becomes the next priority. Ex-

traocular muscle tone must be kept to a minimum to keep IOP low, and straining and bucking that can increase choroidal congestion must be minimized by deep anesthesia. Deep anesthesia can usually be achieved with an inhalational agent, narcotics, or muscle relaxants titrated to an appropriate response on the neuromuscular function monitor.[29]

Inhalational Agents

Several inhalational agents are available, including the halogenated hydrocarbons (sevoflurane, desflurane, halothane, enflurane, and isoflurane) and nitrous oxide. Often, nitrous oxide is combined with one of the other agents to decrease their relative concentrations and minimize side effects.[10] Each has its own advantages and disadvantages; the choice of which agent to use is best left to the anesthesiologist. Other factors that must be continually assessed during anesthesia maintenance are the effects of the anesthetic agents and other adjuvant medications on IOP.

Inhalational anesthetics all cause dose-related decreases in IOP.[31] The exact mechanisms are unknown, but possible etiologies include depression of a CNS control center, reduction of aqueous humor production, enhancement of aqueous humor outflow, and relaxation of the extraocular muscles.[31]

There is, however, one situation in which nitrous oxide must be used with caution. To facilitate retinal detachment repair, an intraocular gas bubble may be injected as a tamponade for retinal tears. Agents used include sulfur hexafloride and octafluoropropane. These agents are relatively insoluble and slowly increase in size over several days. Nitrous oxide can also fill the vitreous cavity intraoperatively. Nevertheless, its solubility coefficient allows for a more rapid increase in volume and a more rapid liberation from the vitreous. This phenomenon presents two possible problems if nitrous oxide is used concomitantly with one of the other agents:

1. an inadequate fill of the tamponading agent, because the nitrous oxide fills some of the space in the vitreous cavity; when the nitrous oxide is then liberated postoperatively, an inadequate tamponade can result; and
2. an intraoperative rise in IOP, which results from the rapid expansion of the nitrous oxide bubble filling the vitreous cavity, in addition to the other agent used.

To prevent these problems, it is recommended that if nitrous oxide is used, it should be turned off at least 15 minutes before the gas is injected into the eye. Furthermore, if a reoperation is required after intraocular gas injection, then nitrous oxide should be avoided for 5 days after an injection of air and for 10 days following the injection of sulfur hexafluoride.[32]

Anesthetics and Intraocular Pressure

Other agents influence IOP, too. The CNS depressants (eg, barbiturates, neuroleptics, opioids, tranquilizers, hypnotics, propofol) all seem to lower IOP. Ketamine, on the other hand, appears to be able to increase the pressure; one study,[31] using indentation tonometry, showed a rise in pressure, although subsequent studies have not always corroborated this finding. Nonetheless, ketamine should be used with caution if an open globe is present or suspected because of its possible role in increasing pressure and because it has also been shown to induce nystagmus and blepharospasm.[31]

Hypertonic solutions (eg, mannitol, dextran, urea, sorbitol), when administered intravenously, all increase plasma oncotic pressure and, thereby, decrease IOP. These agents may produce acute intravascular volume overload, which can place a heavy workload on the heart and kidneys. Hypertension, prolonged diuresis, and dilution of plasma sodium may result. Acetazolamide is a carbonic anhydrase inhibitor; its administration interferes with the sodium pump. The resultant decrease in aqueous humor formation leads to decreased IOP. Its action is not limited to the eye, and systemic effects include loss of sodium, potassium, and water secondary to renal tubular effects. Such electrolyte imbalances can then increase the risk of cardiac dysrhythmias during general anesthesia.[31]

Medications are not the only factors that influence IOP. Ventilatory status and temperature can also affect the pressure within the eye. Hyperventilation decreases IOP, whereas asphyxia, increased levels of carbon dioxide, and hypoventilation have all been shown to increase IOP.[31] Hypothermia decreases IOP secondary to decreased aqueous humor formation from vasoconstriction and the subsequent decrease in ocular blood flow.[31]

Ramifications of Ophthalmic Interventions for Anesthesia Care

The preceding discussion illustrates how actions taken by anesthesia providers can alter the work

environment of the ophthalmologist by influencing IOP. The reverse can also be true when surgical maneuvers or medications given by the ophthalmologist force changes in the anesthetic care provided. One common occurrence is the initiation of the oculocardiac reflex, which is triggered by pressure on the globe or manipulation of the extraocular muscles. This can lead to bradycardia or other serious dysrhythmias, including junctional rhythm, atrioventricular blockade, premature ventricular contractions, ventricular tachycardia, and asystole. This reflex has its afferent limb along the trigeminal nerve and its efferent limb along the vagus nerve. The reflex may appear with the use of any anesthetic technique; however, it is more prevalent with hypoxemia, hypercarbia, and inappropriate anesthetic depth.[31]

Retrobulbar blocks may decrease the incidence of the oculocardiac reflex; however, the administration of the block itself will occasionally *cause* the reflex.[33] If ocular manipulation causes bradycardia or another dysrhythmia, the first step is to have the surgeon stop the surgical maneuver. The patient's anesthetic depth and ventilatory status are then evaluated. The heart rate will usually return to normal within 20 seconds after these measures are instituted. Repeated manipulation of the eye has been shown to decrease the recurrence of the reflex secondary to fatigue of the reflex arc at the level of the cardioinhibitory center.[33] If, however, the initial dysrhythmia was significant or if the reflex continues to recur, the treatment of choice is intravenous atropine. Additionally, careful monitoring of the intraoperative electrocardiogram must be maintained throughout the surgical case.

Several medications that the ophthalmologist administers have the potential for undesirable systemic effects and deleterious anesthetic implications. Topical ophthalmic preparations can be significantly absorbed through the conjunctiva or nasal mucosa after drainage through the nasolacrimal duct. To minimize this, patients can be instructed to occlude the nasolacrimal sac with pressure on the inner canthus of the eye after the administration of a drop. The anesthesiologist must continually monitor for undesirable effects and potential drug interactions.

Acetylcholine is a medication commonly used intraocularly to produce miosis after the lens is removed. The local use of this drug may occasionally result in bradycardia, increased salivation, increased bronchial secretions, bronchospasm, and hypotension. If these occur and require treatment, they can be reversed with intravenous atropine.[31] Echothiophate is a long-acting anticholinesterase and is used to treat chronic glaucoma. When used for more than 1 month, it can decrease plasma pseudocholinesterase activity by 95%, and its effects can last for 4 to 6 weeks even after cessation of the drug.[31] Both succinylcholine and ester local anesthetics are metabolized by plasma pseudocholinesterases, so if either of these is used, ophthalmologists should expect a longer-than-normal duration of action. This phenomenon can lead to prolonged apnea even with usual doses of succinylcholine.

Phenylephrine is commonly used to dilate the pupil. It has α-adrenergic effects that can cause hypertension, headache, tachycardia, and myocardial ischemia. These side effects are rare if the 2.5% solution is used, but they occur more commonly when the 10% solution is used. Caution should be used in the elderly and those with preexisting coronary artery disease to avoid problems. The topical β-blockers are medications commonly used to treat patients with glaucoma. They are contraindicated in patients who have obstructive pulmonary disease, congestive heart failure, preexisting bradycardia, and greater than first-degree heart block.

Cocaine, which was discussed earlier as a topical anesthetic, must be used with care during general anesthesia. Acetazolamide and mannitol, discussed earlier in regard to their being administered by the anesthesiologist, are also frequently used by ophthalmologists to lower IOP. If they are given intraoperatively, the same precautions that were discussed previously must be used to avoid problems.

Anesthetic Emergence

The importance of the anesthetic induction has already been discussed. Another important stage in the care of the traumatized eye is the anesthetic emergence. During emergence, the patient's IOP is likely to increase. Although the adverse consequences of this stage on the repaired eye are less important than for the open globe, coughing, straining, and vomiting during this stage can cause intraocular or orbital bleeding that can jeopardize the results of the surgery. Lidocaine (1.5 mg/kg, intravenously) may attenuate these responses to emergence and extubation.[10]

Shivering after anesthesia can increase IOP and should be treated by warming the patient.[34] Another method for reducing shivering is to administer a small dose of meperidine or hydroxyzine. Methods to minimize postoperative nausea and vomiting are discussed later in this chapter. During transporta-

tion to the recovery room, patients should be kept in a head-up position to facilitate venous drainage from the eye and the orbit. Postoperative hypertension may develop in some patients, owing to anxiety, pain, or urinary retention, and should be treated promptly to prevent straining and elevated eye pressure. Finally, patients who are either blind or bilaterally patched may need psychological support from the recovery room staff and physicians involved in the case.

ANESTHESIA COMPLICATIONS

Major risks of general anesthesia include cardiovascular collapse, allergic or anaphylactic reactions, and malignant hyperthermia. The first two can also occur after the administration of local anesthesia or during MAC. Luckily, such complications are rare, especially if an adequate preoperative assessment is performed. There are also a number of ocular complications from general anesthesia, including corneal abrasions, hemorrhagic retinopathy, retinal ischemia, and periocular nerve compression. Each is discussed here, with an emphasis on prevention.

Allergic Reactions

Allergic reactions may occur from any of the anesthetic agents discussed so far. For local anesthetics, such reactions are typically characterized by pruritus, urticaria, and edema at the site of the injection. Coughing and wheezing may also be present, but if the cardiovascular system is maintained, the reaction can usually be treated with either oral or intramuscular diphenhydramine. The oral dose for an adult is 50 mg, and the intramuscular dose for an adult is 10 to 50 mg, depending on the severity of the reaction. Patients treated in this manner should be observed for at least 6 hours to ensure that their status does not worsen.[12]

If the allergic symptoms occur with cardiovascular collapse, the reaction is considered to be anaphylaxis and requires immediate care. Treatment includes immediate cessation of the drug, volume expansion with intravenous normal saline, and intramuscular or intravenous epinephrine. Oxygen and intravenous aminophylline are administered to reduce the effects of bronchospasm.[12] Dysrhythmias are possible; their treatment is guided by the standards found in Advanced Cardiac Life Support protocols.[35]

Malignant Hyperthermia

A rare defect in muscle metabolism, malignant hyperthermia results in more generation of heat than the body can dissipate. It is inherited in an autosomal-dominant fashion with incomplete penetrance.[36] During the preoperative assessment, questions regarding family history of death under anesthesia should be raised. If a history exists and the cause is unclear, the clinician should be alerted to the possibility of malignant hyperthermia as the cause. Malignant hyperthermia is more common in children, in the Midwest, and in patients with a history of strabismus. It is incited most commonly by a combination of the use of succinylcholine and halogenated anesthetics.[36] Amide local anesthetic agents are usually safe in this setting.

Malignant hyperthermia is best managed by anticipation and prevention. Its earliest signs may be subtle and include tachycardia, darkening of the blood on the operative field, and masseter muscle rigidity. This can be followed by sweating, increased temperature, dramatic oxygen consumption, increased carbon dioxide production, muscle rigidity, cardiac dysrhythmias, unstable blood pressure, and death.[36] Treatment consists of immediate cessation of the agent presumed to be causing the problem and the administration of 100% oxygen. The patient may need to be cooled by gastric or rectal lavage with ice or ice water. Electrolyte imbalance, especially acidosis, is treated as needed. Intravenous dantrolene (2–10 mg/kg) is administered to stabilize calcium outflow from the sarcoplasmic reticulum, and procainamide is given to stabilize cardiac dysrhythmias.[12] If the preoperative assessment uncovers a possibility of malignant hyperthermia, then screening tests, including resting serum creatinine phosphokinase and muscle biopsy, can be ordered. Because these tests sometimes produce false positives or negatives, it is extremely important to carefully observe the patient during all phases of anesthesia.

Ocular Complications of Anesthesia

One of the most common ophthalmic complications of anesthesia is corneal abrasion. This can occur from direct trauma to the cornea from the anesthesia mask or surgical drapes, or can be the result

of chemical injury from the surgical skin preparation solution. Prevention is the best management. The eyes should be closed during skin preparation. During the surgical procedure, the eye not being operated on should be taped closed with or without a bland petroleum-based ophthalmic ointment being applied. If a patient awakens from general anesthesia complaining of pain, tearing, foreign body sensation, or photophobia, a corneal abrasion should be suspected. If confirmed on examination, prophylactic antibiotic ointment should be applied until healing has occurred.

Hemorrhagic retinopathy can occur in otherwise healthy patients during turbulent emergence from anesthesia or if protracted vomiting occurs after anesthesia.[37] It is commonly called Valsalva retinopathy and is related to increased intrathoracic pressure that is transmitted to the ocular vasculature, leading to intraocular bleeding. The bleeding is usually from the venous side and is found in front of the retina beneath the internal limiting membrane. Visual loss is noted if the hemorrhage is in front of the macula or if the hemorrhage breaks through the internal limiting membrane and causes a vitreous hemorrhage. Luckily, most of these hemorrhages are self-limiting and resolve completely in several weeks. Intervention is rarely required unless a vitreous hemorrhage does not clear, and a vitrectomy is needed.

Retinal ischemia and infarction also may result from direct ocular trauma secondary to pressure on the globe. The pressure can be from an ill-fitting anesthesia mask or from excessive force on the globe during surgical manipulation. Care must also be taken if the patient is positioned prone to make sure that external pressure is not being placed on the eye. External pressure on the eye is even more dangerous if systemic hypotension is present.[37] Finally, retinal infarction can occur from emboli during cardiac or vascular surgery. Such emboli can also lead to optic nerve damage as well.

Periorbital nerve compression is most likely secondary to poor positioning of the patient in the prone or jackknife position or excessive pressure from the face mask. Care must be taken to prevent excessive pressure on the orbital rims and to ensure adequate padding. The supraorbital, supratrochlear, and infraorbital nerves are at risk for this complication, which can lead to postoperative numbness and swelling. These symptoms usually resolve without intervention, but several weeks may be needed for full recovery.

POSTOPERATIVE PAIN AND NAUSEA MANAGEMENT

The goal of postoperative analgesia is to maximize pain control while minimizing the side effects of the analgesic agent. The most common side effects of analgesia are sedation, nausea, and vomiting. Most ophthalmic procedures do not lead to significant amounts of pain. However, in the patient with multisystem trauma, severe pain may be caused by the repair of injuries in other areas than the head and neck region. For these areas, the use of strong narcotics (intravenous or intramuscular) may be necessary. If surgery of only the head and neck is performed, often acetaminophen alone or acetaminophen combined with an oral narcotic is enough.

Ice-Cold Compresses

One easy postoperative pain control method that is often overlooked is the liberal use of ice. Cold (ice) compresses not only decrease pain, they also minimize bleeding and swelling by causing vasoconstriction, which prevents the egress of transudate.[12] Packs of crushed ice are better than ice cubes because crushed ice conforms better to the shape of the body. Ice-cold compresses should be used for 10 minutes four times a day for the first 48 to 72 hours.

Narcotics

Narcotics are commonly used analgesics and can be used orally, intramuscularly, or intravenously, depending on the severity of the pain. Oral opioids have a slower onset of action but provide longer pain relief. In addition to their analgesic properties, however, opioids have several other effects that ophthalmologists should keep in mind, including decreased gastrointestinal motility, respiratory depression, orthostatic hypotension, pupillary miosis, nausea, and vomiting.[12]

Commonly used narcotics include morphine, meperidine, codeine, and oxycodone. Morphine is usually reserved for severe pain when it is used either intramuscularly or intravenously. The usual dose is 2 to 10 mg every 4 to 6 hours as needed. Meperidine is also usually reserved for severe pain and can be given in doses of 50 to 100 mg, intramuscularly, every 4 to 6 hours as needed. If the pain is moderate, oral doses of codeine or oxycodone can

TABLE 5-2

ANTIEMETICS FOR NAUSEA AND VOMITING AFTER OPHTHALMIC SURGERY

Antiemetic	Dose (mg)	Duration (h)	Drug Category and Side Effects
Metoclopramide	10	1–2	Dopamine antagonist Extrapyramidal reactions Abdominal cramping
Droperidol	0.625–2.500	3–6	Dopamine antagonist Extrapyramidal reactions Sedation Dysphoria
Ondansetron	1–8 (4)	4	5-HT3 antagonist Headache Dizziness Muscle pains
Dolasetron	12.5	8	5-HT3 antagonist ECG interval changes (PR, QRS) Headache Dizziness Muscle pain
Prochlorperazine	5–10	3–4	Phenothiazine Extrapyramidal reactions Neuroleptic malignant syndrome Sedation Hypotension
Perphenazine	1–5	6–24	Phenothiazine Extrapyramidal reactions Neuroleptic malignant syndrome Sedation Hypotension
Promethazine	12.5–50.0	4–6	Phenothiazine Extrapyramidal reactions Neuroleptic malignant syndrome Sedation (less so) Hypotension
Chlorpromazine	12.5–50.0, administered *slowly*	2–4	Phenothiazine Hypotension Sedation Extrapyramidal reactions Neuroleptic malignant syndrome

Sources: (1) Davidson JK, Eckhardt WF, Perese DA. *Clinical Anesthesia Procedures of the Massachusetts General Hospital,* 4th ed. Boston, Mass: Little, Brown; 1993. (2) Miller RD. *Anesthesia.* 4th ed. New York, NY: Churchill Livingstone; 1994. (3) Medical Economics Data. *Physicians' Desk Reference*. Montvale, NY: Medical Economics Co; 1998.

be used, usually in combination with acetaminophen. The effectiveness of codeine and acetaminophen combined is greater than if each agent were used alone.[12] The usual dose of codeine is 30 to 60 mg every 4 to 6 hours, and the usual dose of oxycodone is 5 to 10 mg every 4 to 6 hours as needed. If the pain is mild, acetaminophen alone may be all that is needed. The usual dose of acetaminophen is 325 to 650 mg, orally, every 4 to 6 hours as needed.

Antinausea and Antiemetic Agents

Postoperative nausea and vomiting can be caused by several different factors. One cause is the ocu-

logastric reflex, whereby ocular manipulation, especially of the extraocular muscles, causes nausea and vomiting. This reflex is especially common after surgery for strabismus. Other causes of nausea and vomiting are the use of preoperative or intraoperative narcotics or their use in the postoperative period for analgesia. The detrimental effects of vomiting on IOP have already been discussed. For these reasons, the use of prophylactic antiemetics is important (Table 5-2). Metoclopramide is an H_1 (histamine) receptor and dopamine antagonist and can help, intraoperatively and postoperatively, decrease the incidence of nausea and vomiting. In addition to its antiemetic effect, metoclopramide also increases the tone of the lower esophageal sphincter and speeds gastric emptying, minimizing the risk of aspiration. The disadvantages of using metoclopramide include its side effect of dystonia, which occurs in 2% of the patients receiving it intravenously.[12] Drowsiness and anxiety can also occur. Metoclopramide is not available as a suppository, and, when used orally, it is less predictable and less effective secondary to metabolism in the liver before it becomes available systemically. Trimethobenzamide is another benzamide antiemetic in the same family as metoclopramide. It is available in suppository form, which is the major difference between it and metoclopramide.

Dopaminergic antagonists of the phenothiazine group include prochlorperazine and promethazine. Both are effective agents to manage postoperative nausea and vomiting. Neither drug increases the tone of the lower esophageal sphincter; thus, they do not influence the risk of aspiration.[12] Both agents have fairly high rates of dystonic reactions, however, and can cause hypotension if used parenterally. They can also cause pupillary dilation and should be used with care in patients with narrow-angle glaucoma.[12]

Droperidol is a butyrophenone with neuroleptic properties. It is an active antagonist at the dopamine receptor and is very useful in preventing nausea and vomiting, even in small doses. Droperidol may result in dyskinesia, restlessness, dysphoria, and hypotension.[38] To limit theses side effects, the lowest effective doses should be used, especially when the goal is prophylaxis.

Ondansetron selectively blocks serotonin receptors with little or no effect on dopamine receptors.[39] It is an effective antiemetic in the postoperative period. It is very expensive and, therefore, is not recommended for routine prophylaxis. Ondansetron is usually reserved for patients with a history of postoperative nausea and vomiting and for those undergoing procedures that often cause nausea and vomiting. Side effects other than pain on intravenous injection are rare, and the drug does not appear to cause sedation, extrapyramidal signs, or respiratory depression.[39]

SUMMARY

Ocular injuries account for a significant and growing percentage of combat injuries. To effectively care for these injuries, highly skilled eye surgery teams, well-versed in the most modern microsurgical techniques, are required. Anesthesia providers must be comfortable with the subtleties of administering anesthesia for ocular trauma, especially when an open globe is suspected or confirmed. The first step to providing such care is a thorough preoperative assessment. If there are ocular injuries that require anesthesia for further evaluation or definitive care, the optimal method must be chosen for the particular situation. The anesthetic choices include topical, local, MAC, and general endotracheal anesthesia.

Topical anesthesia is commonly used during the complete ophthalmic evaluation to determine IOPs, examine the nasolacrimal system and the nose, and perform forced duction testing. Commonly used topical agents include amide agents (eg, lidocaine) and ester agents (eg, proparacaine, tetracaine, and cocaine).

Local anesthesia is often used to repair ocular adnexal injuries, as well as soft-tissue facial injuries. Lidocaine and bupivacaine are amide local anesthetics that are often used in combination because the lidocaine has a quicker onset of action and the bupivacaine has a longer duration of action. Several adjuvant agents can be added to these local anesthetics to enhance their effectiveness. Epinephrine reduces bleeding and lengthens the duration of the neural blockade, but it can cause hypertension and tachycardia and must be used with caution in patients susceptible to these side effects.

There are several techniques available to adequately anesthetize the ocular adnexa. These techniques are usually a combination of subcutaneous regional infiltration, field block, and nerve block. Regional infiltration is the easiest of the techniques because it requires the least knowledge of the neuroanatomy of the ocular adnexa; field and nerve blocks, on the other hand, require a detailed understanding. Successful anesthesia can be achieved with blocks of the supraorbital, supratrochlear,

infratrochlear, infraorbital, and lacrimal nerves.

In cases where more than local anesthesia is required, MAC can be used. MAC involves the use of intravenous sedation and analgesia with noninvasive monitoring in combination with local anesthetics. The most commonly used sedatives for MAC include benzodiazepines, narcotics, and propofol.

If a patient has multisystem trauma or if an open globe injury is present or suspected, general endotracheal anesthesia is usually required. Controversy exists about which neuromuscular blocking agents should be used during induction. Succinylcholine reportedly increases IOP and could cause extrusion of the intraocular contents, but it is not absolutely contraindicated. The decision to use succinylcholine must be made by the anesthesiologist on a case-by-case basis. Once induction is complete, extraocular muscle tone, straining, and bucking must be kept to a minimum as anesthesia is maintained, usually with one of the inhalational halogenated hydrocarbons and nitrous oxide.

During maintenance anesthesia, the effects of the anesthetic agents and other adjuvant medications on IOP must be continually assessed. All inhalational anesthetics cause dose-related decreases in IOP. One must also be vigilant for the oculocardiac reflex. It is usually the result of extraocular muscle manipulation and can result in bradycardia or asystole. To block or reverse the reflex, intravenous atropine should be used.

Emergence from anesthesia must also be handled smoothly to prevent uncontrolled increases in IOP. Intravenous lidocaine can be helpful to prevent coughing and straining during extubation. Keeping the patient warm can minimize shivering, which has also been shown to increase IOP.

There are several major risks of general anesthesia. Allergic reactions range from mild symptoms to full-blown anaphylaxis. Treatment includes cessation of use of the inciting agent, volume expansion, and the administration of oxygen and intravenous epinephrine. Malignant hyperthermia is another major risk. Its early presentation can be subtle and may include tachycardia and darkening of the blood on the surgical field. This phase can be followed by elevated body temperature, unstable blood pressure, and death. Treatment consists of immediate cessation of the inciting agent, the administration of oxygen and intravenous dantrolene, and correction of electrolyte imbalances.

General anesthesia can cause ophthalmic complications, as well. Corneal abrasions can occur from the anesthesia mask or from the surgical preparation solution. Hemorrhagic retinopathy can be the result of a turbulent emergence or postoperative vomiting. Retinal ischemia can result from external pressure on the globe. Finally, periorbital nerve compression can result from poor patient positioning or excessive pressure from the anesthesia mask.

Postoperative pain and nausea management is the final stage in successful anesthesia care. The goal is to maximize pain control while minimizing side effects. Commonly used narcotic analgesics include morphine, meperidine, codeine, and oxycodone. These agents may be administered orally, intramuscularly, or intravenously.

The oculogastric reflex can lead to vomiting, especially after extraocular muscle manipulation. Prophylactic antiemetics minimize the risk of vomiting, which can elevate IOP. Metoclopramide, a widely used antiemetic, increases the tone of the lower esophageal sphincter and speeds gastric emptying.

When ocular trauma is identified on the battlefield, the eye care team must be ready to act. Ophthalmologists and anesthesiologists must be willing to work together to optimize the care provided. Only through attention to detail and adherence to the techniques described in this chapter can risk to the injured eye be minimized before, during, and after the repair.

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