CHAPTER 36

Anesthesia for Ophthalmic Surgery

KEY CONCEPTS

1. Any factor that increases intraocular pressure in the setting of an open globe may cause drainage of aqueous or extrusion of vitreous through the wound. The latter is a serious complication that can permanently worsen vision.
2. Succinylcholine increases intraocular pressure by 5–10 mm Hg for 5–10 min after administration, principally through prolonged contracture of the extraocular muscles. However, in studies of hundreds of patients with open eye injuries, no patient experienced extrusion of ocular contents after administration of succinylcholine.
3. Traction on extraocular muscles, pressure on the eyeball, administration of a retrobulbar block, and trauma to the eye can elicit a wide variety of cardiac dysrhythmias ranging from bradycardia and ventricular ectopy to sinus arrest or ventricular fibrillation.
4. Complications involving the intraocular expansion of gas bubbles injected by the ophthalmologist can be avoided by discontinuing nitrous oxide at least 15 min prior to the injection of air or sulfur hexafluoride, or by avoiding the use of nitrous oxide entirely.
5. Medications applied topically to the mucosa are absorbed systemically at a rate intermediate between absorption following intravenous and subcutaneous injection (the toxic subcutaneous dose of phenylephrine is 10 mg).
6. Echothiophate is an irreversible cholinesterase inhibitor used in the treatment of glaucoma. Topical application leads to systemic absorption and a reduction in plasma cholinesterase activity. Because succinylcholine is metabolized by this enzyme, echothiophate will prolong its duration of action.
7. The key to inducing anesthesia in a patient with an open eye injury is controlling intraocular pressure with a smooth induction. Coughing and gagging during intubation is avoided by first achieving a deep level of anesthesia and profound paralysis.
8. The postretrobulbar block apnea syndrome is probably due to injection of local anesthetic into the optic nerve sheath, with spread into the cerebrospinal fluid.
9. Regardless of the technique employed for intravenous sedation, ventilation and oxygenation must be monitored, and equipment to provide positive-pressure ventilation must be immediately available.

Ophthalmic surgery poses unique problems, including regulation of intraocular pressure, control of intraocular gas expansion, prevention of the oculocardiac reflex and management of its consequences, management of systemic effects of ophthalmic drugs, and frequent utilization of only mild to moderate sedation. A thorough understanding of potentially complicating issues, in addition to the mastery of general, regional, local, and sedation anesthesia techniques for ophthalmic surgery, will favorably influence perioperative outcome in these cases.

INTRAOCULAR PRESSURE DYNAMICS

Physiology of Intraocular Pressure

The eye can be considered a hollow sphere with a rigid wall. If the contents of the sphere increase, the **intraocular pressure** (normal: 12–20 mm Hg) must rise. For example, glaucoma is caused by an obstruction to aqueous humor outflow. Similarly, intraocular pressure will rise if the volume of blood within the globe is increased. A rise in venous pressure will increase intraocular pressure by decreasing aqueous drainage and increasing choroidal blood volume. Extreme changes in arterial blood pressure and ventilation can also affect intraocular pressure (Table 36–1). Any event that alters these parameters (eg, laryngoscopy, intubation, airway obstruction, coughing, Trendelenburg position) can affect intraocular pressure.

Alternatively, decreasing the size of the globe without a proportional change in the volume of its contents will increase intraocular pressure. Pressure on the eye from a tightly fitted mask, improper prone positioning, or retrobulbar hemorrhage can lead to a marked increase in intraocular pressure.

Intraocular pressure helps to maintain the shape, and therefore the optical properties, of the eye. Temporary variations in pressure are usually well tolerated in normal eyes. For example, blinking raises intraocular pressure by 5 mm Hg, and squinting (forced contraction of the orbicularis oculi muscles) may increase intraocular pressure greater than 50 mm Hg. However, even transient episodes of increased intraocular pressure in patients with underlying low ophthalmic artery pressure (eg, deliberate hypotension, arteriosclerotic involvement of the retinal artery) may jeopardize retinal perfusion and cause retinal ischemia.

TABLE 36–1 The effect of cardiac and respiratory variables on intraocular pressure (IOP).[1]

Variable	Effect on IOP
Central venous pressure	
Increase	↑↑↑
Decrease	↓↓↓
Arterial blood pressure	
Increase	↑
Decrease	↓
$Paco_2$	
Increase (hypoventilation)	↑↑
Decrease (hyperventilation)	↓↓
Pao_2	
Increase	0
Decrease	↑

[1] ↓, decrease (mild, moderate, marked); ↑, increase (mild, moderate, marked); 0, no effect.

When the globe is open by surgical incision (Table 36–2) or traumatic perforation, intraocular pressure approaches atmospheric pressure. Any factor that increases intraocular pressure in the setting of an open globe may cause drainage of aqueous or extrusion of vitreous through the wound. The

TABLE 36–2 Open-eye surgical procedures.

Cataract extraction
Corneal laceration repair
Corneal transplant (penetrating keratoplasty)
Peripheral iridectomy
Removal of foreign body
Ruptured globe repair
Secondary intraocular lens implantation
Trabeculectomy (and other filtering procedures)
Vitrectomy (anterior and posterior)
Wound leak repair

TABLE 36–3 The effect of anesthetic agents on intraocular pressure (IOP).[1]

Drug	Effect on IOP
Inhaled anesthetics	
Volatile agents	↓↓
Nitrous oxide	↓
Intravenous anesthetics	
Propofol	↓↓
Benzodiazepines	↓↓
Ketamine	?
Opioids	↓
Muscle relaxants	
Succinylcholine	↑↑
Nondepolarizers	0/↓

[1]↓, decrease (mild, moderate); ↑, increase (mild, moderate); 0↓, no change or mild decrease; ?, conflicting reports.

latter is a serious complication that can permanently worsen vision.

Effect of Anesthetic Drugs on Intraocular Pressure

Most anesthetic drugs either lower intraocular pressure or have no effect (Table 36–3). Inhalational anesthetics decrease intraocular pressure in proportion to the depth of anesthesia. The decrease has multiple causes: a drop in blood pressure reduces choroidal volume, relaxation of the extraocular muscles lowers wall tension, and pupillary constriction facilitates aqueous outflow. Intravenous anesthetics also decrease intraocular pressure, with the exception of ketamine, which usually raises arterial blood pressure and does not relax extraocular muscles.

Topically administered anticholinergic drugs result in pupillary dilation (mydriasis), which may precipitate or worsen angle-closure glaucoma. Systemically administered atropine or glycopyrrolate for premedication are not associated with intraocular hypertension, even in patients with glaucoma.

2 Succinylcholine increases intraocular pressure by 5–10 mm Hg for 5–10 min after administration, principally through prolonged contracture of the extraocular muscles. However, in studies of hundreds of patients with open eye injuries, no patient experienced extrusion of ocular contents after administration of succinylcholine. Unlike other skeletal muscle, extraocular muscles contain myocytes with multiple neuromuscular junctions, and repeated depolarization of these cells by succinylcholine causes the prolonged contracture. The resulting increase in intraocular pressure may have several effects. It will cause spurious measurements of intraocular pressure during examinations under anesthesia in glaucoma patients, potentially leading to unnecessary surgery. Lastly, prolonged contracture of the extraocular muscles may result in an abnormal forced duction test, a maneuver utilized in strabismus surgery to evaluate the cause of extraocular muscle imbalance and determine the type of surgical correction. Nondepolarizing neuromuscular blockers (NMBs) do not increase intraocular pressure.

THE OCULOCARDIAC REFLEX

3 Traction on extraocular muscles, pressure on the eyeball, administration of a retrobulbar block, and trauma to the eye can elicit a wide variety of cardiac dysrhythmias ranging from bradycardia and ventricular ectopy to sinus arrest or ventricular fibrillation. This reflex consists of a trigeminal (V1) afferent and a vagal efferent pathway. The **oculocardiac reflex** is most commonly encountered in pediatric patients undergoing strabismus surgery, although it can be evoked in all age groups and during a variety of ocular procedures, including cataract extraction, enucleation, and retinal detachment repair. In awake patients, the oculocardiac reflex may be accompanied by nausea.

Routine prophylaxis for the oculocardiac reflex is controversial. Anticholinergic medication is often helpful in preventing the oculocardiac reflex, and intravenous atropine or glycopyrrolate immediately prior to surgery is more effective than intramuscular premedication. However, anticholinergic medication should be administered with caution to any patient who has, or may have, coronary artery disease, because of the potential for increase in heart rate sufficient to induce myocardial ischemia. Ventricular tachycardia and ventricular fibrillation following administration of anticholinergic medication has also been reported. Retrobulbar blockade or deep inhalational anesthesia may also be of value in preempting the oculocardiac reflex, although

administration of a retrobulbar block may itself initiate the oculocardiac reflex.

Management of the oculocardiac reflex when it occurs includes: (1) immediate notification of the surgeon and temporary cessation of surgical stimulation until heart rate increases; (2) confirmation of adequate ventilation, oxygenation, and depth of anesthesia; (3) administration of intravenous atropine (10 mcg/kg) if bradycardia persists; and (4) in recalcitrant episodes, infiltration of the rectus muscles with local anesthetic. The reflex eventually fatigues (self-extinguishes) with repeated traction on the extraocular muscles.

INTRAOCULAR GAS EXPANSION

A gas bubble may be injected by the ophthalmologist into the posterior chamber during vitreous surgery. Intravitreal air injection will tend to flatten a detached retina and allow anatomically correct healing. The air bubble is absorbed within 5 days by gradual diffusion through adjacent tissue into the bloodstream. The bubble will increase in size if nitrous oxide is administered, because nitrous oxide is 35 times more soluble than nitrogen in blood (see Chapter 8). Thus, it tends to diffuse into an air bubble more rapidly than nitrogen (the major component of air) is absorbed by the bloodstream. If the bubble expands after the eye is closed, intraocular pressure will rise.

Sulfur hexafluoride is an inert gas that is less soluble in blood than is nitrogen—and much less soluble than nitrous oxide. Its longer duration of action (up to 10 days) compared with an air bubble can provide a therapeutic advantage. The bubble size doubles within 24 hr after injection, because nitrogen from inhaled air enters the bubble more rapidly than the sulfur hexafluoride diffuses into the bloodstream. Even so, unless high volumes of pure sulfur hexafluoride are injected, the slow bubble expansion does not typically raise intraocular pressure. If the patient is breathing nitrous oxide, however, the bubble will rapidly increase in size and may lead to intraocular hypertension. A 70% inspired nitrous oxide concentration will almost triple the size of a 1-mL bubble and may double the pressure in a closed eye within 30 min. Subsequent discontinuation of nitrous oxide will lead to reabsorption of the bubble, which has become a mixture of nitrous oxide and sulfur hexafluoride. The consequent fall in intraocular pressure may precipitate another retinal detachment.

4 Complications involving the intraocular expansion of gas bubbles can be avoided by discontinuing nitrous oxide at least 15 min prior to the injection of air or sulfur hexafluoride, or by avoiding the use of nitrous oxide entirely. The amount of time required to eliminate nitrous oxide from the blood will depend on several factors, including fresh gas flow rate and adequacy of alveolar ventilation. Depth of anesthesia should be maintained by substituting other anesthetic agents. Nitrous oxide should be avoided until the bubble is absorbed (5 days after air and 10 days after sulfur hexafluoride injection). Many ophthalmologists routinely request that nitrous oxide not be used in their patients.

SYSTEMIC EFFECTS OF OPHTHALMIC DRUGS

Topically applied eye drops are systemically absorbed by vessels in the conjunctival sac and the nasolacrimal duct mucosa (see Case Discussion, Chapter 13). One drop (typically, approximately 1/20 mL) of 10% phenylephrine contains approximately 5 mg of drug. Compare this dose with the intravenous dose of phenylephrine (0.05–0.1 mg) used to treat an adult

5 patient with acute hypotension. Medications applied topically to mucosa are absorbed systemically at a rate intermediate between absorption following intravenous and subcutaneous injection (the toxic subcutaneous dose of phenylephrine is 10 mg). Children and the elderly are at particular risk of the toxic effects of topically applied medications and should receive at most a 2.5% phenylephrine solution (**Table 36–4**). Coincidentally, these patients are most apt to require eye surgery.

6 Echothiophate is an irreversible cholinesterase inhibitor used in the treatment of glaucoma. Topical application leads to systemic absorption and a reduction in plasma cholinesterase activity. **Because succinylcholine is metabolized by this enzyme, echothiophate will prolong its duration of action.** Paralysis usually does not exceed 20–30 min, however, and postoperative apnea is

TABLE 36–4 Systemic effects of ophthalmic medications.

Drug	Mechanism of Action	Effect
Acetylcholine	Cholinergic agonist (miosis)	Bronchospasm, bradycardia, hypotension
Acetazolamide	Carbonic anhydrase inhibitor (decreases IOP[1])	Diuresis, hypokalemic metabolic acidosis
Atropine	Anticholinergic (mydriasis)	Central anticholinergic syndrome[2]
Cyclopentolate	Anticholinergic (mydriasis)	Disorientation, psychosis, convulsions
Echothiophate	Cholinesterase inhibitor (miosis, decreases IOP)	Prolongation of succinylcholine and mivacurium paralysis, bronchospasm
Epinephrine	Sympathetic agonist (mydriasis, decreases IOP)	Hypertension, bradycardia, tachycardia, headache
Phenylephrine	α-Adrenergic agonist (mydriasis, vasoconstriction)	Hypertension, tachycardia, dysrhythmias
Scopolamine	Anticholinergic (mydriasis, vasoconstriction)	Central anticholinergic syndrome[2]
Timolol	β-Adrenergic blocking agent (decreases IOP)	Bradycardia, asthma, congestive heart failure

[1]IOP, intraocular pressure.
[2]See Case Discussion, Chapter 13.

unlikely. The inhibition of cholinesterase activity lasts for 3–7 weeks after discontinuation of echothiophate drops. Muscarinic side effects of echothiophate, such as bradycardia during induction, can be prevented with intravenous anticholinergic drugs (eg, atropine, glycopyrrolate).

Epinephrine eye drops can cause hypertension, tachycardia, and ventricular dysrhythmias; the dysrhythmogenic effects are potentiated by halothane. Direct instillation of epinephrine into the anterior chamber of the eye has not been associated with cardiovascular toxicity.

Timolol, a nonselective β-adrenergic antagonist, reduces intraocular pressure by decreasing production of aqueous humor. Topically-applied timolol eye drops, commonly used to treat glaucoma, will often result in reduced heart rate. In rare cases, it has been associated with atropine-resistant bradycardia, hypotension, and bronchospasm during general anesthesia.

General Anesthesia for Ophthalmic Surgery

The choice between general and local anesthesia should be made jointly by the patient, anesthesiologist, and surgeon. Patients may refuse to consider local anesthesia due to fear of being awake during the operation, fear of the eye block procedure, or unpleasant recall of a previous eye block or local eye procedure. General anesthesia is indicated in children and uncooperative patients, as even small head movements can prove disastrous during microsurgery.

PREMEDICATION

Patients undergoing eye surgery may be apprehensive, particularly if they have undergone multiple procedures or there is a possibility of permanent blindness. However, premedication must be administered with caution and only after careful consideration of the patient's medical status. Adult patients are often elderly, with myriad systemic illnesses, such as hypertension, diabetes mellitus, and coronary artery disease. Pediatric patients may have associated congenital disorders.

INDUCTION

The choice of induction technique for eye surgery usually depends more on the patient's other medical problems than on the patient's eye disease or the specific operation contemplated. One exception is the patient with a ruptured globe. The key to inducing anesthesia in a patient with an open

eye injury is controlling intraocular pressure with a smooth induction. Specifically, coughing during intubation must be avoided by achieving a deep level of anesthesia and profound paralysis. The intraocular pressure response to laryngoscopy and endotracheal intubation can be moderated by prior administration of intravenous lidocaine (1.5 mg/kg) or an opioid (eg, remifentanil 0.5–1 mcg/kg or alfentanil 20 mcg/kg). A nondepolarizing muscle relaxant or succinylcholine may be used. Despite theoretical concerns, succinylcholine has not been shown to increase the likelihood of vitreous loss with open eye injuries. Many patients with open globe injuries have full stomachs and require a rapid-sequence induction technique because of the risk of aspiration (see Case Discussion below).

MONITORING & MAINTENANCE

Eye surgery necessitates positioning the anesthesia provider away from the patient's airway, making close monitoring of pulse oximetry and the capnograph particularly important. Endotracheal tube kinking, breathing circuit disconnection, and unintentional extubation may be more likely because of the surgeon working near the airway. Kinking and obstruction can be minimized by using a wire-reinforced or preformed oral RAE® (Ring-Adair-Elwyn) endotracheal tube (see **Figure 36–1**). The possibility of arrhythmias caused by the oculocardiac reflex increases the importance of constantly scrutinizing the electrocardiogram (ECG) and making sure that the pulse tone is audible. In contrast to most other types of pediatric surgery, infant body temperature may rise during ophthalmic surgery because of head-to-toe draping and insignificant body surface exposure. End-tidal CO_2 analysis helps to differentiate this phenomenon from malignant hyperthermia.

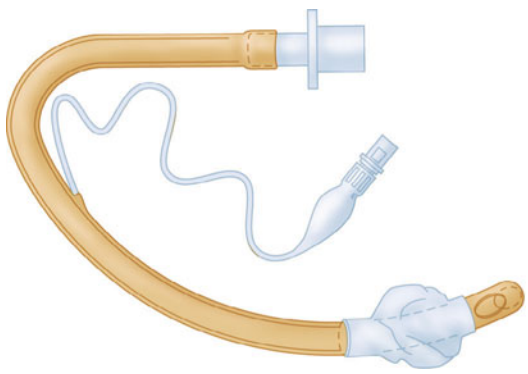

FIGURE 36–1 An oral RAE™ endotracheal tube has a preformed right-angle bend at the level of the teeth so that it exits the mouth away from the surgical field during ophthlamic or nasal surgery.

The pain and stress evoked by eye surgery are considerably less than during a major intraabdominal or intrathoracic procedure. A lighter level of anesthesia would be satisfactory if the consequences of patient movement were not so potentially catastrophic. The lack of cardiovascular stimulation inherent in most eye procedures combined with the need for adequate anesthetic depth can result in hypotension in elderly individuals. This problem is usually avoided by ensuring adequate intravenous hydration and administering small doses of ephedrine or phenylephrine. The practice of substituting muscle relaxation with nondepolarizing muscle relaxants for sufficient depth of anesthesia requires constant attention to the level of neuromuscular blockade to avoid patient movement, injury to the eye, and a malpractice claim.

Emesis caused by vagal stimulation is a common postoperative problem following eye surgery, particularly with strabismus repair. The Valsalva effect and the increase in central venous pressure that accompany vomiting can be detrimental to the surgical result and will increase the risk of aspiration. Intraoperative intravenous administration of a 5-HT_3 antagonist (eg, ondansetron) decreases the incidence of postoperative nausea and vomiting (PONV). Dexamethasone (8–10 mg in adults) should also be considered for patients with a strong history of PONV.

EXTUBATION & EMERGENCE

A smooth emergence from general anesthesia is very important in order to minimize the risk of postoperative wound dehiscence. Coughing or gagging due to stimulus from the endotracheal tube can be minimized by extubating the patient at a moderately deep level of anesthesia. As the end of the surgical procedure approaches, muscle relaxation is reversed, and spontaneous respiration is allowed to return. Anesthetic agents may be continued during gentle suction of the airway. Nitrous oxide, if used, is then discontinued, and intravenous lidocaine (1.5 mg/kg) can be given to blunt cough reflexes temporarily. Extubation proceeds 1–2 min after the lidocaine

administration and during spontaneous respiration with 100% oxygen. Proper airway maintenance is crucial until the patient's cough and swallowing reflexes return. Obviously, this technique is not appropriate in patients at increased risk of aspiration (see Case Discussion at end of chapter).

Severe discomfort is unusual following eye surgery. Scleral buckling procedures, enucleation, and ruptured globe repair are the most painful operations. Modest incremental doses of intravenous opioid (eg, fentanyl 25 mcg or hydromorphone 0.25 mg for an adult) usually provide sufficient analgesia. The surgeon should be alerted if severe pain is noted following emergence from general anesthesia, as it may signal intraocular hypertension, corneal abrasion, or other surgical complications.

Regional Anesthesia for Ophthalmic Surgery

Options for local anesthesia for eye surgery include topical application of local anesthetic or placement of a **retrobulbar block** or the more commonly utilized **peribulbar or sub-Tenon's (episcleral) block.** All of these techniques are most commonly combined with intravenous sedation. Local anesthesia is preferred to general anesthesia for eye surgery because local anesthesia involves less physiologic trespass and is less likely to be associated with PONV. However, eye block procedures have potential complications and may not provide adequate akinesia or analgesia of the eye. Some patients may be unable to lie perfectly still for the duration of the surgery. For these reasons, appropriate equipment and qualified personnel required to treat the complications of local anesthesia and to induce general anesthesia must be readily available.

RETROBULBAR BLOCKADE

In this technique, local anesthetic is injected behind the eye into the cone formed by the extraocular muscles (Figure 36–2), and a facial nerve block is utilized to prevent blinking (Figure 36–3). A blunt-tipped 25-gauge needle penetrates the lower lid at the junction of the middle and lateral

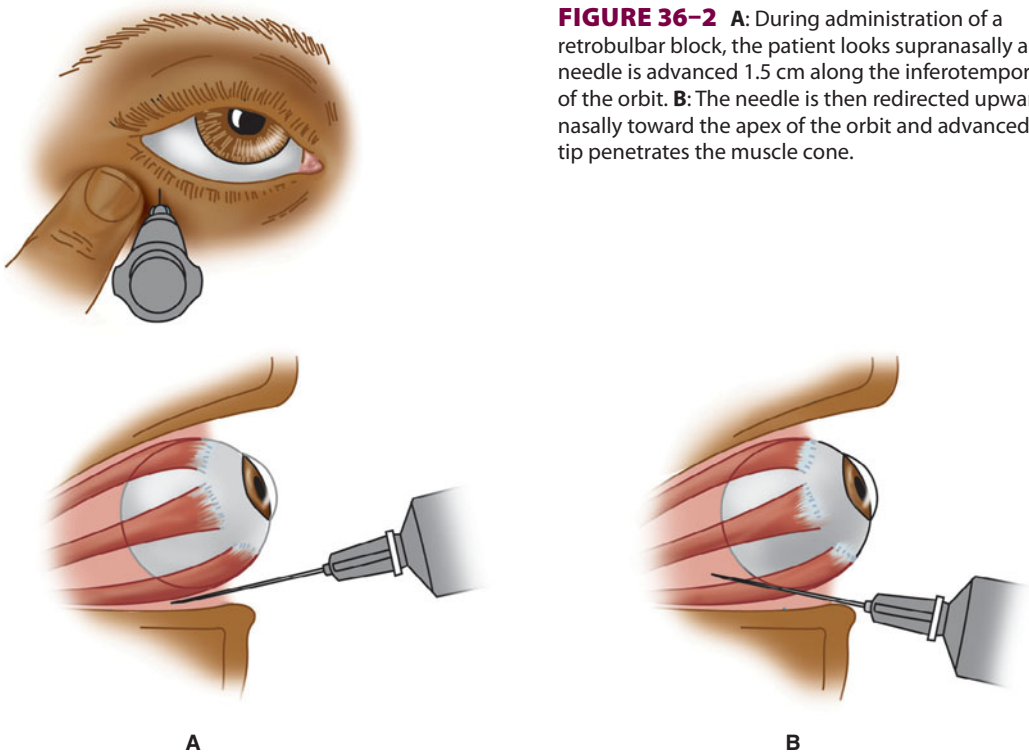

FIGURE 36–2 **A**: During administration of a retrobulbar block, the patient looks supranasally as a needle is advanced 1.5 cm along the inferotemporal wall of the orbit. **B**: The needle is then redirected upward and nasally toward the apex of the orbit and advanced until its tip penetrates the muscle cone.

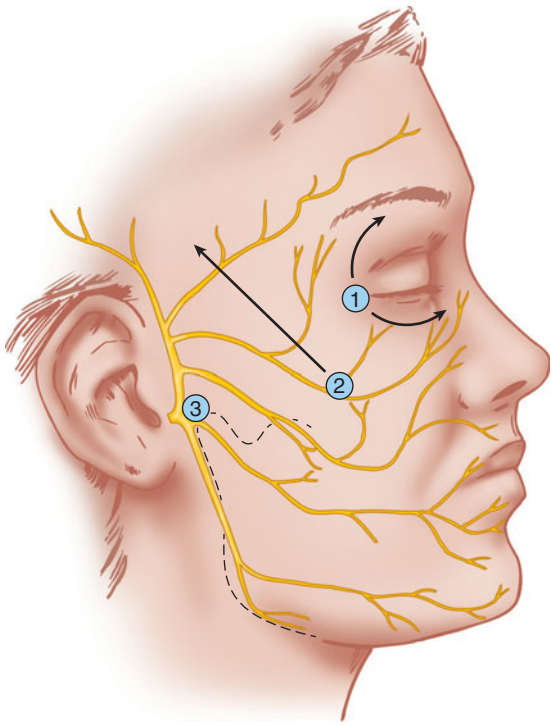

FIGURE 36–3 Facial nerve block techniques: van Lint (1), Atkinson (2), and O'Brien (3).

one-third of the orbit (usually 0.5 cm medial to the lateral canthus). Awake patients are instructed to stare supranasally as the needle is advanced 3.5 cm toward the apex of the muscle cone. Commonly, patients undergoing such eye blocks will receive a brief period of deep sedation during the block (using such agents as etomidate, propofol, and remifentanil). After aspiration to preclude intravascular injection, 2–5 mL of local anesthetic is injected, and the needle is removed. Choice of local anesthetic varies, but lidocaine 2% or bupivacaine 0.75% are most common. Ropivacaine may be used instead of bupivacaine. Addition of epinephrine (1:200,000 or 1:400,000) may reduce bleeding and prolong the anesthesia. Hyaluronidase (3–7 U/mL), a hydrolyzer of connective tissue polysaccharides, is frequently added to enhance the retrobulbar spread of the local anesthetic. A successful retrobulbar block is accompanied by anesthesia, akinesia, and abolishment of the oculocephalic reflex (ie, a blocked eye does not move during head turning).

Complications of retrobulbar injection of local anesthetics include retrobulbar hemorrhage, perforation of the globe, optic nerve atrophy, intravascular injection with resultant convulsions, oculocardiac reflex, trigeminal nerve block, respiratory arrest, and, rarely, acute neurogenic pulmonary edema. Forceful injection of local anesthetic into the ophthalmic artery causes retrograde flow toward the brain and may result in an instantaneous seizure. 8 The postretrobulbar block apnea syndrome is probably due to injection of local anesthetic into the optic nerve sheath, with spread into the cerebrospinal fluid. The central nervous system is exposed to high concentrations of local anesthetic, leading to mental status changes that may include unconsciousness. Apnea occurs within 20 min and resolves within an hour. Treatment is supportive, with positive-pressure ventilation to prevent hypoxia, bradycardia, and cardiac arrest. Adequacy of ventilation must be constantly monitored in patients who have received retrobulbar anesthesia.

Retrobulbar injection is usually not performed in patients with bleeding disorders because of the risk of retrobulbar hemorrhage, extreme myopia because the elongated globe increases the risk of perforation, or an open eye injury because the pressure from injecting fluid behind the eye may cause extrusion of intraocular contents through the wound.

PERIBULBAR BLOCKADE

In contrast to retrobulbar blockade, in the peribulbar blockade technique, the needle does not penetrate the cone formed by the extraocular muscles. Advantages of the peribulbar technique include less risk of penetration of the globe, optic nerve, and artery, and less pain on injection. Disadvantages include a slower onset and an increased likelihood of ecchymosis. Both techniques will have equal success at producing akinesia of the eye.

The peribulbar block is performed with the patient supine and looking directly ahead (or possibly under a brief period of deep sedation). After topical anesthesia of the conjunctiva, one or two

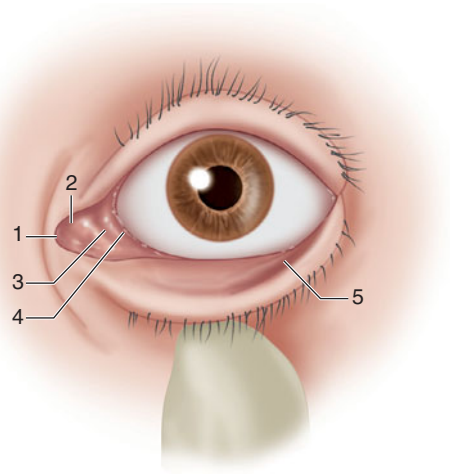

FIGURE 36–4 Anatomic landmarks for the introduction of a needle or catheter in most frequently employed eye blocks: (1) medial canthus peribulbar anesthesia, (2) lacrimal caruncle, (3) semilunaris fold of the conjunctiva, (4) medial canthus episcleral anesthesia, and (5) inferior and temporal peribulbar anesthesia.

transconjunctival injections are given (Figure 36–4). As the eyelid is retracted, an inferotemporal injection is given halfway between the lateral canthus and the lateral limbus. The needle is advanced under the globe, parallel to the orbital floor; when it passes the equator of the eye, it is directed slightly medial (20°) and cephalad (10°), and 5 mL of local anesthetic is injected. To ensure akinesia, a second 5-mL injection may be given through the conjunctiva on the nasal side, medial to the caruncle, and directed straight back parallel to the medial orbital wall, pointing slightly cephalad (20°).

Sub-Tenon's (Episcleral) Block

Tenon's fascia surrounds the globe and extraocular muscles. Local anesthetic injected beneath it into the episcleral space spreads circularly around the sclera and to the extraocular muscle sheaths (Figure 36–4). A special blunt 25-mm or 19-gauge curved cannula is used for a sub-Tenon block. After topical anesthesia, the conjunctiva is lifted along with Tenon's fascia in the inferonasal quadrant with forceps. A small nick is then made with blunt-tipped scissors, which are then slid underneath to create a path in Tenon's fascia that follows the contour of the globe and extends past the equator. While the eye is still fixed with forceps, the cannula is inserted, and 3–4 mL of local anesthetic are injected. Complications with the sub-Tenon blocks are significantly less than with retrobulbar and peribulbar techniques. Globe perforation, hemorrhage, cellulitis, permanent visual loss, and local anesthetic spread into cerebrospinal fluid have been reported.

FACIAL NERVE BLOCK

A facial nerve block prevents squinting of the eyelids during surgery and allows placement of a lid speculum. There are several techniques of facial nerve block: van Lint, Atkinson, and O'Brien (Figure 36–3). The major complication of these blocks is subcutaneous hemorrhage. Another procedure, Nadbath's technique, blocks the facial nerve as it exits the stylomastoid foramen under the external auditory canal, in close proximity to the vagus and glossopharyngeal nerves. This block is not recommended because it has been associated with vocal cord paralysis, laryngospasm, dysphagia, and respiratory distress.

TOPICAL ANESTHESIA OF THE EYE

Simple topical local anesthetic techniques have evolved for anterior chamber (eg, cataract) and glaucoma operations, and, increasingly, the trend has been to eliminate local anesthetic injections entirely. A typical regimen for topical local anesthesia consists of application of 0.5% proparacaine (also known as proxymetacaine chlorhydrate) local anesthetic drops, repeated at 5-min intervals for five applications, followed by topical application of a local anesthetic gel (lidocaine chlorhydrate plus 2% methyl-cellulose) with a cotton swab to the inferior and superior conjunctival sacs. Ophthalmic 0.5% tetracaine may also be utilized. Topical anesthesia is not appropriate for posterior chamber surgery (eg, retinal detachment repair with a buckle), and it works best for faster surgeons with a gentle surgical technique that does not require akinesia of the eye.

INTRAVENOUS SEDATION

Many techniques of intravenous sedation are available for eye surgery, and the particular drug used is less important than the dose. Deep sedation, although sometimes used during placement of ophthalmic nerve blocks, is almost never used intraoperatively because of the risks of apnea, aspiration, and unintentional patient movement during surgery. An intraoperative light sedation regimen that includes midazolam (1–2 mg), with or without fentanyl (25–50 mcg) or sufentanil (2.5–5 mcg), is recommended. Doses vary considerably among patients, but should be administered in small increments. Concomitant use of more than one type of drug (benzodiazepine, hypnotic, and opioid) potentiates the effects of other agents, and doses must be reduced accordingly.

Administration of eye blocks can be quite uncomfortable, and many anesthesia providers will administer small incremental doses of etomidate or propofol to produce a brief state of unconsciousness during the regional block. Some will substitute a bolus of opioid (remifentanil 0.1–0.5 mcg/kg or alfentanil 375–500 mcg) to produce a brief period of intense analgesia during the eye block procedure.

9 Administration of an antiemetic should be considered if an opioid is used. Regardless of the technique employed, ventilation and oxygenation must be monitored, and equipment to provide positive-pressure ventilation must be immediately available.

CASE DISCUSSION

An Approach to a Patient with an Open Eye & a Full Stomach

A 12-year-old boy arrives at the emergency room after being shot in the eye with a pellet gun. A brief examination by the ophthalmologist reveals intraocular contents presenting at the wound. The boy is scheduled for emergency repair of the ruptured globe.

What should be stressed in the preoperative evaluation of this patient?

Aside from taking a routine history and performing a physical examination, the time of last oral intake before or after the injury should be established as accurately as possible. The patient must be considered to have a full stomach if the injury occurred within 8 hr after the last meal, even if the patient did not eat for several hours after the injury: gastric emptying is delayed by the pain and anxiety that follow trauma.

What is the significance of a full stomach in a patient with an open globe injury?

Managing patients who have sustained penetrating eye injuries provides a challenge to anesthesia providers because of the need to develop an anesthetic plan that is consistent with at least two conflicting objectives: (1) prevent further damage to the eye by avoiding increases in intraocular pressure and (2) prevent pulmonary aspiration in a patient with a full stomach.

Many of the common strategies used to achieve these objectives are in direct conflict with one another, however (Tables 36–5 and 36–6). For example, although regional anesthesia (eg, retrobulbar block) minimizes the risk of aspiration pneumonia, it is relatively contraindicated in patients with penetrating eye injuries because injecting local anesthetic behind the globe increases intraocular pressure and may lead to expulsion of intraocular contents. Therefore, these patients require general anesthesia—despite the increased risk of aspiration pneumonia.

TABLE 36–5 Strategies to prevent increases in intraocular pressure (IOP).

Avoid direct pressure on the globe
Patch eye with Fox shield
No retrobulbar or peribulbar injections
Careful face mask technique
Avoid increases in central venous pressure
Prevent coughing during induction and intubation
Ensure a deep level of anesthesia and relaxation prior to laryngoscopy[1]
Avoid head-down positions
Extubate under deep anesthesia[1]
Avoid pharmacological agents that increase IOP

[1]These strategies are not recommended in patients with full stomachs.

TABLE 36–6 Strategies to prevent aspiration pneumonia.

Regional anesthesia with minimal sedation[1]
Premedication
Metoclopramide
Histamine H_2-receptor antagonists
Nonparticulate antacids
Evacuation of gastric contents
Nasogastric tube[1]
Rapid-sequence induction
Cricoid pressure
Rapid induction with rapid onset of paralysis
Avoidance of positive-pressure ventilation via mask
Intubation as soon as possible
Extubation awake

[1]These strategies are not recommended for patients with penetrating eye injuries.

What preoperative preparation should be considered in this patient?

The goal of preoperative preparation is to minimize the risk of aspiration pneumonia by decreasing gastric volume and acidity (see Case Discussion, Chapter 17). Aspiration in patients with eye injuries is prevented by proper selection of pharmacological agents and anesthetic techniques. Evacuation of gastric contents with a nasogastric tube may lead to coughing, retching, and other responses that can dramatically increase intraocular pressure.

Metoclopramide increases lower esophageal sphincter tone, speeds gastric emptying, lowers gastric fluid volume, and exerts an antiemetic effect. It should be given intravenously (10 mg) as soon as possible and repeated every 2–4 hr until surgery.

Ranitidine (50 mg intravenously), cimetidine (300 mg intravenously), and famotidine (20 mg intravenously) are H_2-histamine–receptor antagonists that inhibit gastric acid secretion. Because they have no effect on the pH of gastric secretions present in the stomach prior to their administration, they have limited value in patients presenting for emergency surgery.

Unlike H_2-receptor antagonists, antacids have an immediate effect. Unfortunately, they increase intragastric volume. Nonparticulate antacids (preparations of sodium citrate, potassium citrate, and citric acid) lose effectiveness within 30–60 min and should be given immediately prior to induction (15–30 mL orally).

Which induction agents are recommended in patients with penetrating eye injuries?

The ideal induction agent for patients with full stomachs would provide a rapid onset of action in order to minimize the risk of regurgitation. Ketamine, propofol, and etomidate have essentially equally rapid onsets of action (ie, one-arm-to-brain circulation time).

Furthermore, the ideal induction agent would not increase the risk of ocular expulsion by raising intraocular pressure. (In fact, most intravenous induction agents lower intraocular pressure.) Although investigations of the effects of ketamine on intraocular pressure have provided conflicting results, ketamine is not recommended in penetrating eye injuries, owing to the high rate of blepharospasm and nystagmus.

Although etomidate may prove valuable in some patients with cardiac disease, it is associated with an incidence of myoclonus ranging from 10% to 60%. An episode of severe myoclonus may have contributed to complete retinal detachment and vitreous prolapse in one patient with an open globe injury and limited cardiovascular reserve.

Propofol has a rapid onset of action and decreases intraocular pressure; however, it does not entirely prevent the hypertensive response to laryngoscopy and intubation or entirely prevent the increase in intraocular pressure that accompanies laryngoscopy and intubation. Prior administration of fentanyl (1–3 mcg/kg), remifentanil (0.5–1 mcg/kg), alfentanil (20 mcg/kg), esmolol (0.5–1 mg/kg), or lidocaine (1.5 mg/kg) attenuates this response with varying degrees of success.

How does the choice of muscle relaxant differ between these patients and other patients at risk of aspiration?

The choice of muscle relaxant in patients with penetrating eye injuries has been controversial. Succinylcholine definitely increases intraocular

pressure. Although there is conflicting research, it is probably most prudent to conclude that this rise in pressure is not consistently and reliably prevented by pretreatment with a nondepolarizing agent, self-taming doses of succinylcholine, or lidocaine. Contradictory findings by various investigators using different regimens are probably due to differences in doses and timing of the pretreatment drugs.

Some anesthesiologists argue that the relatively small and transient rise in intraocular pressure caused by succinylcholine is insignificant when compared with changes caused by laryngoscopy and intubation. They claim that a slight rise in intraocular pressure is a small price to pay for two distinct advantages that succinylcholine offers: a rapid onset of action that decreases the risk of aspiration, and profound muscle relaxation that decreases the chance of a Valsalva response during intubation. Furthermore, advocates of succinylcholine usually point to the lack of case reports documenting further eye injury when succinylcholine has been used and to publications documenting safe use of succinylcholine with open eye injuries.

Nondepolarizing muscle relaxants do not increase intraocular pressure. Regardless of the muscle relaxant chosen, intubation should not be attempted until a level of paralysis is achieved that will definitely prevent coughing on the endotracheal tube.

How do induction strategies vary in pediatric patients without an intravenous line?

A hysterical child with a penetrating eye injury and a full stomach provides an anesthetic challenge for which there is no perfect solution. Once again, the dilemma is due to the need to avoid increases in intraocular pressure yet minimize the risk of aspiration. For example, screaming and crying can lead to tremendous increases in intraocular pressure. Attempting to sedate children with rectal suppositories or intramuscular injections, however, often heightens their state of agitation and may worsen the eye injury. Similarly, although preoperative sedation may increase the risk of aspiration by obtunding airway reflexes, it is often necessary for establishing an intravenous line for a rapid-sequence induction. Although difficult to achieve, an ideal strategy would be to administer enough sedation painlessly to allow the placement of an intravenous line, yet maintain a level of consciousness adequate to protect airway reflexes. However, the most prudent strategy is to do everything reasonable to avoid aspiration—even at the cost of further eye damage.

Are there special considerations during extubation and emergence?

Patients at risk of aspiration during induction are also at risk during extubation and emergence. Therefore, extubation must be delayed until the patient is awake and has intact airway reflexes (eg, spontaneous swallowing and coughing on the endotracheal tube). Deep extubation increases the risk of vomiting and aspiration. Intraoperative administration of antiemetic medication and nasogastric or orogastric tube suctioning may decrease the incidence of emesis during emergence, but they do not guarantee an empty stomach.

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