



# Anesthesia-Related Complications of Periocular Surgery

# 5

Sathyadeepak Ramesh and Jonathan A. Hoenig

## Introduction: Why Do Surgeons Need to Know About Anesthesia?

Periocular surgery is now routinely performed on an outpatient basis, either with local anesthetic, intravenous sedation, or general anesthesia. Cases can even be performed without an anesthesiologist in the office – with local anesthetic and oral sedation. While patients and surgeons alike benefit from the efficiency and convenience of surgery in the ambulatory setting, intraoperative anesthesia-related complications can be devastating when unrecognized or improperly treated. The “captain of the ship” doctrine, while not necessarily legally defensible in many states [1], nevertheless places the surgeon at the center of any liability arising from an adverse event in the operating room even when not related to negligence on the surgeon’s part. Moreover, the increasing prevalence of certified registered nurse anesthetists (CRNAs), who may even practice independently depending on location, muddies the waters of medicolegal responsibility should such an event occur [2]. Most importantly, prompt recognition of anesthetic-related complications can save lives, and the periocular surgeon has a responsibility to learn about the potential adverse effects of and treatments for these complications. Medically appropriate anesthesia leads to smoother surgery, an improved patient experience, and better outcomes.

---

S. Ramesh (✉)

Wills Eye Hospital, Thomas Jefferson University, Philadelphia, PA, USA

e-mail: [info@deepakrameshmd.com](mailto:info@deepakrameshmd.com)

J. A. Hoenig

Division of Orbital and Ophthalmic Plastic Surgery, Jules Stein Eye Institute,  
Los Angeles, CA, USA

Private Practice, Beverly Hills, CA, USA

© Springer Nature Switzerland AG 2020

M. E. Hartstein et al. (eds.), *Avoiding and Managing Complications in Cosmetic Oculofacial Surgery*, [https://doi.org/10.1007/978-3-030-51152-4\\_5](https://doi.org/10.1007/978-3-030-51152-4_5)

## Risks of Local Anesthetic

Local anesthetic can be associated with several complications. Direct damage from injection into vital structures (vessels, nerves, and the globe itself) can lead to immediate (hemorrhage, vascular occlusion) or late (extraocular muscle fibrosis) complications. Care must be taken at all times to ensure that sharp objects are not directed toward the globe while injection, particularly in the patient who is not under general anesthetic. Sudden sneezing or coughing due to intravenous anesthetic can lead to inadvertent globe injury. Furthermore, hematoma can distort tissues and weaken action of muscles such as the levator, complicating surgical planning during ptosis surgery and blepharoplasty. Finally, patients metabolize local anesthetic at different rates and can even be unresponsive to certain types of anesthetic. Short-lived or inadequate anesthesia can lead to patient dissatisfaction and a compromised surgical result.

Local anesthetic systemic toxicity (LAST) can be a devastating phenomenon that can suddenly lead to potentially lethal complications. Local anesthetics act primarily through blockade of voltage-gated sodium channels, preventing action potentials from being generated in affected nerves [3]. These sodium channels are also present in the central nervous system and cardiac tissue, as well as all other tissues, such that increased concentrations can cause unwanted side effects. Local anesthetics within the same class have different pharmacokinetics, including binding affinity, diffusion coefficient, lipophilicity, plasma protein binding, and molecular weight; these factors account for varying toxicity among these drugs. Moreover, physiologic states that induce increased blood flow to the brain (e.g., hypercapnia) or heart (e.g., metabolic acidosis) can increase the free fraction of drug and worsen toxicity. Coadministration of epinephrine with its subsequent vasoconstriction can delay systemic absorption of the drug, allowing for increased doses to be given.

Early clinical suspicion and diagnosis are paramount to prompt treatment. Prodromal symptoms include a metallic taste in the mouth, dizziness, tinnitus, auditory/visual disturbances, and perioral numbness; frank toxicity can lead to seizures, cardiac dysrhythmias, and even cardiac arrest [4]. These effects are primarily dose dependent (Table 5.1), although idiosyncrasies exist – bupivacaine, in particular, unbinds more slowly from sodium channels than other anesthetics (including lidocaine and ropivacaine), leading to an increased fraction of bound receptors and greater risk of toxicity without prodromal symptoms [4]. In fact, cardiac dysrhythmias from bupivacaine can occur as the first presenting sign, prior to any prodrome

**Table 5.1** Recommended local anesthetic doses

Local anesthetic	Maximum recommended dose (mg/kg)	Maximum adult dose (mg)
Bupivacaine	2 (2 with epinephrine)	150
Cocaine	1.5	1.5
Lidocaine	4.5 (7 with epinephrine)	200 (500 with epinephrine)
Prilocaine	6 (8 with epinephrine)	400 (600 with epinephrine)
Ropivacaine	3	225

Adapted from Butterworth et al. [5]

or central nervous system symptoms. Ropivacaine is the pure S(−) enantiomer of bupivacaine, which binds much less avidly to sodium channels than the R(+) enantiomer [5] and potentially exhibits decreased cardiotoxicity; however, given reduced potency, higher doses are typically injected which may negate this benefit. Liposomal bupivacaine (Exparel, Pacira Biosciences, Parsippany, NJ) allows slow dissociation of the anesthetic which may improve its safety profile, although this has not been tested.

Timing of these reactions can be immediate (from direct, inadvertent intravascular injections) to delayed (due to slow absorption from the infiltrated tissue into the intravascular space), with plasma concentrations sometimes reaching their peak hours after initial infiltration and, in some cases, after discharge from the surgery center [6]. In particular, patients in the extremes of age should be monitored closely due to decreased lean muscle mass, hypoalbuminemia, and impaired cardiorenal function leading to decreased drug metabolism.

Treatment is directed at the specific adverse effect of the toxicity. Standard cardiopulmonary resuscitation guidelines should be followed for cardiac dysrhythmias, hypoventilation, and central nervous system depression or seizure activity with key exceptions – epinephrine should be given at a dose of <1 μg/kg, and vasopressin, calcium channel blockers, beta adrenergic receptor blockers, and other local anesthetics (e.g., lidocaine) are to be avoided. Airway maintenance is key as hypercapnia can exacerbate local anesthetic toxicity [7]. Seizures should be treated with benzodiazepines. Most importantly, intravenous lipid infusion [8] should be administered at a dose of 1.5 mL/kg, followed by a continuous infusion of 0.25 mL/kg/min for a maximum of 10 mL/kg in 30 minutes [7]. Adults can be treated with a simplified regimen of 100 mL over 2–3 minutes followed by 200–250 mL of the 20% emulsion over 15–20 minutes [9], with a maximum of 12 mL/kg or 1 L to be given. Lipid emulsion creates a potential space in which the local anesthetic rapidly accumulates due to its lipophilicity and also exhibits cardioprotective effects [10] which may ameliorate ischemia-related or reperfusion-induced injury. While propofol does contain lipid, it is only a 10% emulsion, and any benefits are outweighed by an overdose of the propofol itself; as such, propofol itself cannot be used to treat LAST.

---

## Risks of Systemic Anesthetics

Whether a patient is undergoing conscious sedation or general anesthesia, inhalational and/or intravenous anesthetics can cause complications that can be inconvenient or even catastrophic. The distinction of monitored anesthesia care (MAC) versus general anesthesia (GA) is subtle and has to do with the level of sedation of the patient rather than the device used to secure the airway (laryngeal mask airway (LMA), endotracheal tube (ETT), versus no device (with nasal cannula)). A patient with a nasal cannula and deep intravenous sedation, who is not responsive to external stimuli yet still breathes spontaneously, is still considered to have undergone general anesthesia. In comparison, patients undergoing conscious sedation (or MAC) can have varying levels of responsiveness and can even be awoken

completely for portions of the procedure (e.g., ptosis surgery). Patients undergoing any type of sedation incur risks associated with the medicines administered (commonly inhalational anesthetics, opiates, propofol, and benzodiazepines), the airway, or postoperative nausea and vomiting. A full treatise on best practice for anesthesia is out of the scope of this book, but we will focus on selected points that are relevant for the periocular surgeon.

Malignant hyperthermia is a rare but potentially life-threatening condition that occurs due to an inherited skeletal muscle disorder. In response to certain inhalational anesthetics or succinylcholine, susceptible individuals experience sustained muscular contraction that leads to hyperthermia, cardiac dysrhythmias, and, eventually, death. The earliest signs include elevated end-tidal CO<sub>2</sub> and tachypnea; clinical suspicion should remain high in the young patient who is overbreathing the ventilator, and prompt recognition and treatment with discontinuation of the anesthetic and infusion of intravenous dantrolene (2.5 mg/kg loading dose, and then 1 mg/kg until the patient improves) can save the patient's life. All surgical areas where inhalational or intravenous anesthetic is administered should have a malignant hyperthermia treatment cart, even if the surgical area is within the plastic surgeon's office.

Airway-related complications, whether they are due to laryngospasm, pharyngeal obstruction, a misplaced tube, or hypoventilation/apnea, lead to downstream hypoxia and ischemic brain damage. Obstruction is common in patients with diagnosed or undiagnosed sleep apnea. Laryngospasm is particularly common in the pediatric population [11]. Respiratory depression or central apnea can result from excess dosages of sedative medicines. A reactive airway (e.g., from recent infection) can lead to coughing and straining during surgery. Aspiration risk can be reduced by adhering to published *nil per os* (NPO) guidelines [12]; in the event an LMA or nasal cannula is used, the airway is unprotected from gastric contents, and strict adherence to published guidelines is mandatory for elective surgery. The surgeon must be prepared to deal intraoperatively with these complications by helping the anesthesia staff secure the airway. This may include providing a sterile jaw thrust/chin lift, allowing an oral airway or LMA/ETT to be placed, or deferring cautery so that more concentrated oxygen can be delivered.

Cardiovascular complications including dysrhythmias and hypotension/hypertension can also occur. Continuous monitoring is necessary when any patient undergoes sedation, and staff should be trained in the relevant cardiovascular resuscitation protocols. It is crucial for the surgeon to recognize that it is *impossible* to attend to both the patient's vital signs and the surgical procedure at hand. As such, there should always be a dedicated staff member to continuously evaluate the patient's level of sedation, oxygenation, and other vital signs at all times – this can be an anesthesiologist, nurse anesthetist, or the circulating room nurse. All tools necessary for resuscitation (including airway management, drugs, and defibrillator) should be readily available and routinely checked for function. Staff should be trained and up to date on best practices in advanced cardiac life support. Most importantly, dysrhythmias and cardiovascular anomalies should be detected in an early state that obviates the need for dramatic interventions such as defibrillation.

Thorough communication with an attentive anesthetist is key to preventing disasters before they occur.

Each drug has a specific side effect profile that includes both dose-related and idiosyncratic reactions [13]. Benzodiazepines provide rapid anxiolysis and amnesia, which is often desirable for the initial injection of local anesthetic. However, in combination with opioids, this can cause respiratory depression or apnea. An atypical reaction to benzodiazepines can cause the patient to exhibit *increased* agitation or even delirium. Opioids can provide significant analgesia with some sedation and antitussive effect, at the risk of increased respiratory depression. Fentanyl in particular can cause chest wall rigidity in an idiosyncratic reaction that makes ventilation ineffective; reversal of the opioid with naloxone is key to treating this reaction. Propofol provides excellent sedation with ease of titration, although synergistically causes respiratory depression in combination with opioids and benzodiazepines. Dexmedetomidine avoids these risks of respiratory depression, although there is a higher risk of bradycardia, hypotension, and prolonged recovery due to a delayed peak effect. Any of these medications may cause a prolonged emergence from anesthesia, and staff in the recovery area should be available for continuous monitoring of sedated patients. Ketamine provides amnesia and analgesia without less risk of respiratory depression; however, a dissociative state may develop that confounds typical sedative effects, and hypersalivation may exacerbate a reactive airway. Adequate local anesthetic is key to allowing the patient to remain comfortable under sedation; in our practice, we are able to perform facelifts with oral benzodiazepines and local anesthetic, if the patient so desires. A thorough evaluation of the patient and thoughtful discussion with the anesthesia staff about the duration of surgery and level of cooperation necessary will allow the best cocktail of medicines to be given for each patient.

---

## Fire Risk

Assessment of intraoperative fire risk is critical for the periocular surgeon given the proximity to the airway and common use of unsecured methods to deliver oxygen (e.g., nasal cannula). The “fire triad” describes the elements necessary for a fire to occur – an ignition source (cautery), oxidizer (intraoperative oxygen delivery), and fuel (hair, gauze, preparatory solutions, drapes, etc.) [14]. While most fires are quickly arrested, the consequences can be no less catastrophic, and an operating room fire is a “never” event.

Monopolar cautery and handheld battery-powered cautery are the most common ignition sources, and patient skin/hair and surgical gauze were the most common fuels [14]. Alcohol-based preparations also carry a higher risk of fire, and standard practice is to clean the face with 10% iodine solution; if chlorhexidine is required, then a 3-minute drying time is recommended with care to ensure no pooling of solution.

Supplemental oxygen delivery is the most common oxidizer, although a significant percentage of fires were reported to occur without any high-flow oxygen

delivered. As such, the surgeon must be aware that reducing oxygen delivery is important (ideally <30%) but not sufficient to eliminate the risk of fire while using cautery. Open draping of the face also allows rapid diffusion of concentrated pockets of oxygen, both reducing risk of ignition and limiting severity of fires should they occur. Should the patient require continuous high-flow oxygen delivery and concurrent cautery use, a sealed gas delivery device (e.g., LMA or ETT) should be considered.

Finally, battery-operated cautery devices have a disproportionately higher risk of ignition compared to other devices, and caution should be exercised. Bipolar cautery likely has the least risk of ignition, although the risk is not nil. Regardless of the type of cautery used, continuous communication between the surgeon and anesthesia staff is vital to reducing the risk of operating room fire.

---

## Miscellaneous Risks

Loss of intravenous access can lead to inadequate anesthesia and an awake patient. Inability to treat dysrhythmias, drug reactions, or other cardiovascular abnormalities can lead to complications. Infiltrated needles can also lead to a compartment syndrome, which, if not quickly diagnosed, can lead to loss of function in the downstream appendages. Equipment failure, while thankfully rare, can complicate procedures. Backups of all relevant equipment must be available on site. Procedures must be in place for power outages, fires, or natural disasters. Protocols for dealing with these are well established by the certifying agencies for surgical centers. Care must be taken to ensure that the surgical facility, be it the office, outpatient surgical center, or hospital, is in compliance with these requirements.

---

## Conclusion

Potential intraoperative complications during periocular surgery are numerous, and it is impossible to predict or account for each possibility. However, good patient outcomes depend on prompt recognition and appropriate treatment. Thorough preoperative and intraoperative planning between the surgeon, nurses, and anesthesia staff is critical in providing safe and effective anesthesia for the patient's periocular procedure.

---

## References

1. Murphy E. "Captain of the ship" doctrine continues to take on water. *AORN J.* 2001;74(4):525–8.
2. Weber P. Ophthalmologists' liability for the actions of CRNAs. 2012;(3). <https://docs.google.com/viewerng/viewer?url=http://www.omic.com/wp-content/uploads/2012/12/Digest-Summer-2012.pdf>. Accessed: 12/1/2019.
3. Hille B. Local anesthetics: hydrophilic and hydrophobic pathways for the drug-receptor reaction. *J Gen Physiol.* 1977;69(4):497–515.

4. Gitman M, Fettiplace MR, Weinberg GL, Neal JM, Barrington MJ. Local anesthetic systemic toxicity: a narrative literature review and clinical update on prevention, diagnosis, and management. *Plast Reconstr Surg*. 2019;144(3):783–95.
5. Butterworth JF. Models and mechanisms of local anesthetic cardiac toxicity: a review. *Reg Anesth Pain Med*. 2010;35(2):167–76.
6. Nordström H, Stånge K. Plasma lidocaine levels and risks after liposuction with tumescent anaesthesia. *Acta Anaesthesiol Scand*. 2005;49(10):1487–90.
7. Neal JM, Bernardis CM, Butterworth JF, et al. ASRA practice advisory on local anesthetic systemic toxicity. *Reg Anesth Pain Med*. 2010;35(2):152–61.
8. Weinberg GL, VadeBoncouer T, Ramaraju GA, Garcia-Amaro MF, Cwik MJ. Pretreatment or resuscitation with a lipid infusion shifts the dose-response to bupivacaine-induced asystole in rats. *Anesthesiology*. 1998;88(4):1071–5.
9. Neal JM, Woodward CM, Harrison TK. The American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2017 version. *Reg Anesth Pain Med*. 2018;43(2):150–3.
10. Fettiplace MR, Kowal K, Ripper R, et al. Insulin signaling in bupivacaine-induced cardiac toxicity: sensitization during recovery and potentiation by lipid emulsion. *Anesthesiology*. 2016;124(2):428–42.
11. Hampson-Evans D, Morgan P, Farrar M. Pediatric laryngospasm. *Paediatr Anaesth*. 2008;18(4):303–7.
12. Presta MV, Bhavani SS, Abdelmalak BB. Nil per os guidelines: what is changing, what is not, and what should? *Minerva Anesthesiol*. 2018;84(12):1413–9.
13. Nekhendzy V, Ramaiah VK. Prevention of perioperative and anesthesia-related complications in facial cosmetic surgery. *Facial Plast Surg Clin North Am*. 2013;21(4):559–77.
14. Maamari RN, Custer PL. Operating room fires in oculoplastic surgery. *Ophthal Plast Reconstr Surg*. 2018;34(2):114–22.