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## REVIEW ARTICLES

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### PREVENTION AND MANAGEMENT OF COMPLICATIONS OF REGIONAL ORBITAL ANESTHESIA

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#### Abstract

The majority of ophthalmic procedures are performed under regional anesthesia, but the proximity of important structures such as the blood vessels, optic nerve and the brainstem lead to increased risks associated with these blocks. The most serious of these complications is brainstem anesthesia. As the number of outpatient freestanding surgical centers increase, the significance of these potential complications is expected to increase from orbital blocks for ocular surgical procedures such as cataract removal and vitrectomy. An understanding of these complications, which may sometimes be life-threatening, are thus vital to the anesthesia practitioner. Procedural improvements include a close evaluation of the precise anatomy of the region, with particular attention to injection sites, depth of injection, position of the globe, and techniques to avoid nerve damage and accidental injection into surrounding structures, including blood vessels, globe and cerebrospinal fluid. This literature review emphasizes the importance of the prevention, recognition and management of these complications, which includes the extremely serious complication of brainstem anesthesia.

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#### Introduction

The majority of ophthalmic surgeries utilize either topical anesthesia or regional anesthesia by the injection of local anesthetic (LA) around or behind the eye, either by ophthalmologists or anesthesiologists. Over the past three decades, anesthesiologists have become increasingly involved in eye blocks that were previously performed by surgeons. The three most common techniques to supply anesthesia include retrobulbar, peribulbar, and topical anesthesia (TA). The proximity of the targeted nerves to vital structures predisposes retrobulbar and peribulbar techniques in particular to inherent potential complications.

One rare, but life-threatening complication is brainstem anesthesia and is associated primarily with retrobulbar injection. It is estimated to accompany anywhere between 0.01% to 0.79% of

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regional ophthalmologic blocks and the consequences can be deadly<sup>1,2,3</sup>. Symptoms range from drowsiness and slurred speech to respiratory depression and hemodynamic instability<sup>4</sup>. It is important for anesthesia providers to be cognizant of the potential complications of LA injections for orbital anesthesia, to recognize the signs and symptoms particular to possible adverse events, and to be knowledgeable of the preventive measures and the management of these complications.

In this review, we discuss the anatomical considerations, LA pharmacological considerations, regional anesthetic techniques, and potential complications, which include hemorrhage, blindness, cardiopulmonary arrest and brain stem anesthesia.

## Anatomic Considerations

Ophthalmic surgery is challenging on many fronts. Immobility and analgesia are paramount to surgical success. Basic knowledge of orbital anatomy is important for effective analgesia, successful surgery, and reduction of potential complications.

### *Orbit Anatomy*

It is important to be intimately familiar with the anatomy of the orbit in order to prevent and treat the complications that can arise from the administration of LAs for eye surgeries. With the exception of the inferior oblique, all of the six extraocular muscles (lateral rectus, medial rectus, superior rectus, inferior rectus, superior oblique, and inferior oblique) form a cone in the bony orbit, through which several sensory and motor innervations make their passage, including the first branch of the trigeminal nerve (ophthalmic nerve) through the ciliary ganglion, which supplies sensory innervation to the globe. Motor innervation is supplied by the oculomotor nerve, trochlear nerve, abducens nerve, and the temporal division of the facial nerve. The branches of the ophthalmic division of the trigeminal nerve along with most of the motor nerves to the orbit travel behind the globe within a space for which LA can be deposited (except for the temporal division of the facial nerve). It should be noted that the cone is anatomically incomplete and that there is no structure which links all of the extrinsic ocular

muscles. The apex of the cone is the site of entry of the optic nerve into the orbit. The four rectus muscles (superior, medial, inferior and lateral) insert near the equator of the globe, which is suspended in the anterior part of the orbit and is the base of the retrobulbar cone. These four rectus muscles stem from the annulus of Zinn, a fibrous ring at the orbital apex that surrounds the optic foramen and the medial aspect of the superior orbital fissure. The muscle cone divides the orbit into the extraconal, conal, and intraconal compartments<sup>5</sup>. With the exception of the trochlear nerve, the motor supply to the extraocular muscles also passes through this muscular cone. It is, therefore, highly likely that the administration of LA inside the muscular cone should provide intense operative akinesia and anesthesia as well as post-operative analgesia for ocular surgeries<sup>6,7</sup>. However, this practice is associated with risks. Many other structures are located in this muscular cone including the meningeal sheaths of the optic nerve, the optic nerve and the blood supply to the orbit. Perforation of these structures can lead to injury, risk of toxicity to the nerve and hemorrhage by damage to the blood vessels supplying the orbit<sup>8</sup>. Additionally, injection of LA can lead to spread from the orbital apex via the submeningeal pathways to the central nervous system<sup>9</sup>. Intraconal space (inside the cone formed by the rectus muscles) is the location for retrobulbar injection. It is anatomically different than extraconal space, which is the site for peribulbar injection. These techniques will be discussed in greater detail later in the manuscript.

### *Transmitting Nerves and Vessels from the Orbits*

The nerves and vessels that innervate the optic foramen and medial portion of the superior orbital fissure extend through the annulus of Zinn in order to supply structures in the muscle cone. The three cranial nerves that supply the motor innervations to the extraocular muscles are the trochlear nerve (cranial nerve IV), the abducens nerve (cranial nerve VI) and the oculomotor nerve (cranial nerve III). The oculomotor nerve supplies the medial, superior and inferior rectus, the inferior oblique, and the levator palpebrae superioris muscles. The abducens nerve supplies the lateral rectus muscle and the trochlear nerve supplies the superior

oblique muscle. The zygomatic nerve, a branch of the maxillary nerve in the floor of the orbit splits into the zygomaticotemporal and zygomaticofacial branches. The anterior and posterior ethmoidal foramina are the exit points for the ethmoidal nerves and vessels as they leave the orbit<sup>7</sup>.

As previously mentioned, three of the most common techniques for ophthalmological procedures include retrobulbar block, peribulbar blocks, and TA. Though not suitable for open globe surgery, these blocks are adequate for many different types of surgery. Significant growth in topical anesthesia for certain lens and anterior chamber procedures has become accepted in clinical practice and has evolved in technique over the past two decades.

### **i. Retrobulbar Block**

Since its first description by Knapp in 1884, the retrobulbar block has commonly been utilized for intraocular surgeries<sup>10</sup>. Anesthetic injection into the muscular cone by the retrobulbar block gives retrobulbar anesthesia (RBA). The purpose of the block is to anesthetize the cornea, the ciliary body, the ciliary ganglion located between the ophthalmic artery and the optic nerve all of which are supplied by the ophthalmic cranial nerves (II, III, & VI).

Atkinson refined the retrobulbar block in 1936<sup>11</sup> with a technique that involves having the patient look in and up, allowing the inferior oblique muscle and the fascia between the lateral and inferior rectus muscles to be out of the way of the needle tip<sup>12</sup>. However, CT analysis of cadaveric orbits has revealed that this technique leads to stretching of nerves and the conformation of the optic nerve, ophthalmic artery, and superior orbital vein to be precariously close to the needle tip, increasing the potential risk of accidental puncture and systemic anesthetic toxicity<sup>13</sup>. The current recommendation to increase safety is to position the eye either into a primary gaze or an infranasal position<sup>14</sup>.

The current technique of RBA involves advancing the needle into the temporal lower lid, through the orbital septum, and then following the orbit toward the muscle cone and orbital apex. The needle is aspirated after entering the muscle cone space. After determination via aspiration that the needle is not in the vasculature, LA is administered<sup>15</sup>. In addition, some practitioners will administer a separate facial

nerve block to achieve eyelid akinesia.

The anesthetic solution is usually a 2-6 mL mixture of 0.75% bupivacaine (or 0.5% ropivacaine) and 2% lidocaine that is injected with a 25-gauge, 3-cm needle, aiming toward the orbital muscle cone<sup>16</sup>. Epinephrine is sometimes added to reduce hemorrhage and to extend the duration of action of the LA, while hyaluronidase is sometimes used as another adjunct, which acts by increasing the potency of the bupivacaine (or ropivacaine)<sup>17</sup> and expediting its onset<sup>18</sup>.

### **ii. Peribulbar anesthesia (PBA)**

RBA was the predominant regional eye block for much of the last century<sup>19</sup> but has recently given way to the similarly efficacious pericone block, the peribulbar anesthesia block (PBA), which theoretically leads to fewer complications because it does not involve introducing a needle into the muscular cone, which is close to numerous vulnerable structures<sup>20</sup>. However, it should be noted that RBA is still being practiced all over the world.

PBA was first reported by Davis and Mandel in 1985<sup>21</sup>. With peribulbar block the local anesthetic is deposited within the orbit but not in the muscular cone. Widespread use of the phacoemulsification technique has changed anesthesia requirements in which total akinesia is no longer essential. The block consists of two injections: a superior injection above the medial canthus nasally and an inferior injection given at the junction of the outer two thirds and the inner one third of the inferior rim of the orbit. A common local anesthetic mixture used is 5 cc of 2% lidocaine with 1: 200,000 epinephrine, along with 5 cc of 0.75% bupivacaine (or 0.5% ropivacaine) for the block.

### **iii. Topical Anesthesia (TA)**

LA drops are effective for many uncomplicated ophthalmologic procedures. It is estimated that approximately half of all cataract procedures and 90% of those utilizing phacoemulsification are performed with TA. Limitations include lack of akinesia and short duration of action. Whenever phacoemulsification is not employed, total akinesia is required making TA largely inadequate. Intraoperative comfort is diminished with TA and the use of lidocaine jelly instead of eye drops has been demonstrated to enhance the quality of analgesia. Specific patients, who are on anticoagulants

including daily aspirin or NSAIDs, would benefit from TA to minimize the risk of bleeding.

### Choice of Local Anesthetic

LAs are the key agents used to achieve neural blockade in regional anesthesia, including orbital blocks. The basic structure of LAs consists of an aromatic end and an amine end linked to each other through an ester or and amide chain. These molecules act primarily via targets on neuronal sodium channels thereby blocking the conduction of action potentials along the course of nerves. The susceptibility of a nerve to blockade by LAs depends on characteristics of the individual drug (e.g. potency, pKa, buffering), characteristics of the tissue (e.g. presence of local inflammation and acidosis), and characteristics of individual nerve fibers (e.g. diameter, myelination, activity). LAs tend to block small nerve fibers both earlier and at lower concentrations than large nerve fibers, and blockade resolves in the reverse order. For example, blockade of 0.15 $\mu$ m C-fibers (post-ganglionic autonomic, pressure, dull pain, and temperature) occurs before 1.5 $\mu$ m A $\delta$  fibers (sharp pain), and blockade of 15 $\mu$ m A $\alpha$  (motor) is the last to occur. In addition to blocking sodium channels on sensory and motor fibers, at high doses or after inadvertent intravascular injection, LAs can block sodium channels in the brain and heart, potentially resulting in morbidity or mortality, including loss of consciousness, seizures, myocardial depression, and even cardiac arrest.

LAs can be broadly divided into two categories: those with an amide linking chain and those with an ester linking chain. Ester LAs can generally be identified as those with only one "i" in their names, and they include cocaine, chlorprocaine, and tetracaine. Amide LAs can generally be identified as those with two "i's." Amides are more commonly available and tend to be utilized for orbital regional techniques, and include lidocaine, mepivacaine, bupivacaine, and ropivacaine. Allergy to ester LAs is much more common than amide LAs, due to the metabolism of ester LAs to the common allergen para-aminobenzoic acid (PABA). Amide LAs can generally be safely used in patients with a history of allergy to ester LAs. Ester LAs are primarily metabolized by pseudocholinesterase,

whereas amide LAs undergo hepatic metabolism.

Lidocaine is an intermediate-duration amide LA with significant potency, fast onset, good tissue penetration, and low cardiac toxicity. The concentration of lidocaine used for regional anesthesia ranges from 1-2%, with orbital techniques typically employing a 2% preparation, and a single injection can provide up to approximately 6 hours of analgesia. As with many regional techniques, lidocaine is often used in combination with a long-acting LA, such as bupivacaine in order to achieve both rapid and enduring analgesia for orbital anesthesia.

Bupivacaine was the first long-acting amide LA created. The concentration of bupivacaine used for regional nerve blockade ranges from 0.25-0.75%, and a single injection can provide up to approximately 16-24 hours of analgesia. It is more hydrophobic than lidocaine and has a slower onset. Bupivacaine is highly protein-bound which allows for a longer duration; however, this also contributes to the potential for cardiotoxicity. Due to its narrow therapeutic index, bupivacaine has been replaced in many locations by ropivacaine.

Levobupivacaine is the levorotatory enantiomer of bupivacaine. Commercial bupivacaine is a racemic mixture of both enantiomers (R and S). Levobupivacaine is approximately equivalent to its racemic bupivacaine with respect to onset, duration and dosing in regional anesthesia. However, cardiac and CNS toxicity of levobupivacaine is approximately 35% less than racemic bupivacaine.

Exparel™ is a bupivacaine liposome injectable suspension (1.3% 266 mg/20 mL or 13.3 mg/mL). DepoFoam, which is a multivesicular liposome, consists of tiny 10-30 microns in diameter lipid-based particles which contain discrete water-filled chambers of bupivacaine dispersed through a lipid matrix. This novel preparation allows for increased duration of efficacy and pain relief up to 72 hours after injection. This drug preparation recently came to market in 2012 and to date, there are no clinical trials for this agent in orbital anesthesia.

Ropivacaine is a long-acting amide local anesthetic derived from mepivacaine and is a structural analog of bupivacaine. Ropivacaine differs from bupivacaine in that it exists as a pure S enantiomer, and it demonstrates significantly reduced cardiac and CNS

toxicity. Concentrations of ropivacaine ranging from 0.2-1% are used for regional anesthesia, and a single injection can provide up to approximately 16 hours of analgesia.

Mixtures of lidocaine with a long-acting local anesthetic such as bupivacaine or ropivacaine are commonly used for regional anesthesia techniques. This combination does achieve a quicker onset of analgesia; however, the plasma levels of the long-acting LA are lower than when using only long-acting LAs.

### Additives to LAs

Vasoconstrictors such as epinephrine, typically administered for orbital regional blocks, or phenylephrine are often added to LAs to improve the duration and quality of neural blockade. Vasoconstriction slows the rate of systemic absorption, which can allow for the administration of higher doses of LAs before encountering toxicity. The addition of epinephrine to LAs can also serve as a marker of intravascular injection. If LA with epinephrine is injected intravascularly, the patient will demonstrate signs of tachycardia, hypertension, or T-wave peaking on EKG. Epinephrine may also improve the quality of neuraxial LA blockade independent of vasoconstriction through  $\alpha$ -2 adrenergic activity.

### Toxicity of LAs

The primary concern in the administration of LAs is the risk of systemic LA absorption resulting in CNS and cardiac toxicity. This can occur from inadvertent intravascular injection of LA or from systemic absorption of LA. The risk of systemic toxicity depends on the LA agent and the site of administration. For example, even a very small dose of lidocaine can rapidly cause unconsciousness and seizure if inadvertently injected into an artery. Even without intravascular injection, rates of absorption and risk of systemic toxicity vary by location of administration. Toxicity also varies with specific LA agents. The potent, lipophilic, long-acting amide agent bupivacaine presents a much higher risk of cardiotoxicity than any of the other commercially available agents.

### CNS Toxicity

At sufficient doses or after intravascular injection, LAs cause CNS stimulation. Early symptoms can include lightheadedness, metallic taste, tinnitus, and circumoral numbness. This can progress to restlessness, unconsciousness, and tonic clonic seizures. At higher doses, CNS depression occurs which can result in respiratory failure and death.

### Cardiovascular Toxicity

LAs can additionally cause cardiovascular toxicity, though this generally occurs at doses higher than those causing CNS symptoms. LAs act on the myocardium, decreasing electrical excitability, conduction rate, and contractile force. On rare occasions LAs have caused cardiovascular collapse and death without preceding CNS symptoms. This may be due to either an action on the pacemaker or the sudden onset of ventricular fibrillation. Ventricular tachycardia and fibrillation are relatively uncommon consequences of local anesthetics but can occur, particularly with bupivacaine. Bupivacaine associated cardiotoxicity is very resistant to resuscitation and defibrillation. In addition to ACLS, treatment should include rapid administration of lipid emulsion.

Therefore, the choice of LA can play a large role in determining the risk of toxicity. Of the commonly used LAs, lidocaine and bupivacaine, it is clear that the latter has the greater toxic potential. Of note is that lidocaine has a relatively short duration compared to bupivacaine and accidental intraocular injections of preservative free lidocaine has not been found to cause permanent neuroretinal damage but has been seen to temporarily paralyze the pupil in mydriasis. Permanent visual damage has been attributed to the retinal injury from the needle penetration and increased intraocular pressure causes by the volume of injected LA<sup>xx</sup>. Bupivacaine, by virtue of its high lipid solubility is highly potent but severe cardiotoxicity can occur with inadvertent intravascular administration<sup>xx</sup>.

Like all LA toxicity, signs of bupivacaine toxicity start with central nervous system (CNS) excitation including dizziness, tremors, and nervousness followed by CNS and cardiovascular depression,

including hypotension, respiratory depression, and cardiac arrhythmias and/or arrest<sup>24</sup>. The toxic potential of LAs makes it essential that LA orbital blocks are administered with extreme perioperative vigilance and complications are dealt with promptly and effectively.

Strict sterile technique including antibiotic skin preparation, sterile gloves, hat, and mask should be used for all regional blocks. Monitoring patients for regional anesthesia is additionally important, as patients may require procedural sedation, and complications such as LA toxicity, nerve injury, vascular puncture and bleeding can occur. Standard monitoring should include pulse oximetry, electrocardiography, and blood pressure monitoring. Some centers use capnography to ensure adequate ventilation, but pulse oximetry and frequent verbal communication with the patient are often adequate.

Resuscitation equipment should also be available, including a self-inflating bag-valve-mask, oxygen source with face-mask, suction, intravenous access, laryngoscope, and endotracheal tubes. Resuscitation medications such as vasopressors and lipid emulsion should also be readily available.

## Complications

Complications from regional anesthesia in ophthalmic surgery could be seen immediately after the administration of the LA orbital block or could take up to 40-60 minutes for symptoms to become clear. It is, therefore, recommended to observe the patient for up to an hour after a block. Complications from retrobulbar injection include trauma to adjacent structures, retrobulbar hemorrhage, oculocardiac reflex, misplaced injections, intra-arterial injections, subarachnoid injection, extraocular muscle damage, optic nerve damage, globe perforation, and brainstem anesthesia. Complications from peribulbar anesthesia include trauma to adjacent structures, peribulbar hemorrhage, central retinal artery occlusion, oculocardiac reflex, toxic injury to rectus muscles, and globe perforation.

## Brainstem Anesthesia

It is imperative that the healthcare practitioner is cognizant of and able to recognize the symptoms of

brainstem anesthesia and promptly provide treatment because, left untreated, the associated cardiovascular and systemic sequelae can be life-threatening. Vigilant perioperative monitoring, with assistance from the anesthesia care team, is essential<sup>25</sup>. Although the incidence of brainstem anesthesia following RBA is only between 1:500 and 1:350, a wide range of symptoms include aphasia, apnea, bradycardia, tachycardia, cyanosis, impaired hearing, cardiac arrest, confusion, diaphoresis, dilatation of the contralateral pupil, drowsiness, dysphagia, facial paralysis, gaze palsy, hypertension, loss of consciousness, nausea and vomiting, seizures and shivering. It is advisable to observe the patient for at least 15 minutes post-injection to detect and to manage these complications promptly<sup>26,27</sup>.

It is estimated that the incidence of brainstem anesthesia complicating eye block anesthesia is one in 350 to 500 patients. A variety of interventions have been utilized depending on the symptoms including reassurance, use of vasoconstrictors or vasodilators, ventilation and endotracheal intubation<sup>28</sup>.

Two possible etiologies have been postulated for the development of brainstem anesthesia. First, simian studies have revealed that reversal of blood flow from the ophthalmic artery towards the anterior cerebral artery or internal carotid artery occurs upon accidental arterial anesthetic injection into the ophthalmic artery. LA solution may then reach the brain causing the symptoms of brainstem anesthesia<sup>29,30</sup>. The second possible etiology postulated is that inadvertent injection of the anesthetic solution into the dura mater sheath of the optic nerve or intrathecally through the optic foramen reaches the cerebrospinal fluid compartment causing the symptoms of brainstem anesthesia<sup>31,32</sup>.

Since the symptoms of brainstem anesthesia often appear minutes after injection, it is thought that perforation of the meningeal sheath of the optic nerve triggers a diffusion mechanism, which is responsible for the symptoms<sup>33</sup>. This theory was verified by monitored injection of contrast material into the optic nerve dural sheath in cadavers. The material was seen to diffuse through the ophthalmic artery into the subdural space and chiasm, ultimately reaching the respiratory center<sup>34</sup>.

Brainstem anesthesia's hemodynamic and respiratory manifestations are due to blockade of the

cranial nerves by caudal flow, specifically the vagus (X) and the glossopharyngeal (IX) in contrast to the cephalad flow of LA drugs, seen in high lumbar spinal anesthesia, thoracic sympathetic blockade (hypotension, bradycardia) and apnea due to thoracic intercostal paralysis. The parasympathic blockade of the vagus nerve leads to a period of tachycardia and hypertension that is prolonged by abolition of the regulation of the carotid sinus reflex caused by the blockade of the glossopharyngeal nerve. Severe apnea is usually the result, when blunting of diaphragmatic respiration occurs<sup>35</sup>.

There have been reports of other cranial nerves being affected, including a case of immediate contralateral amaurosis, which may be caused by optic nerve injection, or cortical stroke by ventricular emboli<sup>36</sup>. Transient bilateral hearing loss following RBA has also been seen<sup>37</sup>.

### Globe Perforation

Inadvertent globe perforations by regional eye blocks has a reported incidence of 0.9 in 10,000 RBA and 1 in 16,000 PBA<sup>38</sup>. Intraglobal hemorrhage can lead to globe rupture. It must be remembered that a highly myopic eye with an axial length exceeding 26 mm is a contraindication for regional ophthalmologic anesthesia<sup>39</sup>. There is a twenty-fold increase in incidence of globe perforation in highly myopic individuals leading to an incidence to approximately 1 in 500 in these patients. Vitrectomies have had to be performed because on severe intraocular hemorrhage or retinal detachments. Less severe bleeds may be undetected in up to a third of cases<sup>40,41</sup>.

### Prevention and management of complications

1. Vigilance and prompt treatment are keys to the management of the complications arising from the administration of local anesthetic regional blocks for eye surgeries. Informed consent should be obtained from all patients before the administration of LA regional orbital blocks<sup>40</sup>.

2. Ventilatory support and intensive care facilities should be immediately available especially in cases where brainstem anesthesia is suspected.

Brain stem anesthesia can occur as a complication of a regional block involving any cranial nerve though most commonly it has been discussed in setting of ophthalmologic surgeries. Nique et al described a case of brain stem anesthesia in a patient who presented with dysarthria and hypertension following the administration of V2-V3 blocks in the diagnosis of trigeminal neuralgia. Rapid progression to unconsciousness and apnea followed. Endotracheal intubation with ventilatory support was needed to regain hemodynamic stability<sup>41</sup>. Endotracheal intubation and Intensive Care Unit admission was needed in other cases involving anesthetic blocks for ocular surgeries such as cataract and trabeculectomies with seizures and signs of brainstem anesthesia to prevent catastrophic outcomes<sup>42,43</sup>.

3. Examination of the fundus after administration of peribulbar or retrobulbar blocks by indirect ophthalmoscope. Globe perforation is a serious complication with the potential for vision loss. It is possible for globe penetration or perforation to go undetected most commonly in cataract removal often until the completion of the procedure. For this reason and in order to decrease the morbidity of the its complication, it is recommended that immediately following the administration of peribubar or retrobulbar blocks, the fundus be checked with an indirect ophthalmoscope to ensure that the retinal vessels are patent and that the sclera has not been inadvertently penetrated<sup>44</sup>. The presence of Hyptony or dark red reflex could indicate the presence of a potential ocular globe penetration<sup>45</sup>. To avoid the risk of hemorrhage from the choroidal and retinal blood vessels, and the risk of retinal detachment, it is recommended that ocular surgery be cancelled if globe penetration is suspected. Treatments of globe perforation include retinopexy of the perforation site, photocoagulation and retinal repair. If the cataract permits indirect ophthalmoscopy of the penetration site, arrangements should be made for retinopexy of the perforated site<sup>1</sup>.

4. Positioning can play a large role in the prevention of complications. Correct positioning consists of the globe being in the primary or in the slightly downward and outward position, and directing the needle toward the inferior section of the orbital fissure, and not cross the mid-sagittal plane with

the needle. This adjustment has the disadvantage of increasing the risk of incomplete nerve blockage<sup>46</sup>.

5. Modification of techniques to decrease complications have been developed to improve the effectiveness of the block and to decrease the incidence of accidental punctures. The first such technique, developed by Gills and Lloyd, is to apply a lidocaine post anesthetic injection to reduce site burning, and then to apply pressure for 60 minutes. Another method is to place an additional injection into the subcutaneous space as the first injection is being withdrawn to increase the flow of LAs towards the lateral canthus, thereby increasing the paralysis of the orbicularis oculi muscles<sup>47</sup>.

6. It is also thought that the use of the appropriate needle type may reduce adverse events. It is recommended that needles used should be less than 35 mm long. The use of blunted needles is also encouraged after making a skin wheal with the local anesthetic at the site of injection. Though blunted needles can cause more injection site pain, they may also be associated with less inadvertent perforations<sup>48</sup>.

7. The site of injection also plays an important role. Currently PBA is often the preferred alternative to RBA as it is considered theoretically safer than RBA. The needle only advances until it is parallel to the floor or roof of the orbit and does not enter inside the muscle cone. This results in an even distribution of anesthesia between the lower and the upper peribulbar spaces<sup>49</sup>. It is seen that PBA achieves better lid akinesia though it has a greater chance of having incomplete ocular akinesia due to uncertain local anesthetic flow compared to RBA. Other disadvantages of the PBA also exist. Some studies have shown that the risk of globe perforation remains unaltered<sup>50</sup> and PBA has also been linked to an increased risk of neurogenic pulmonary edema<sup>51</sup>. Different mechanisms exist for the development of acute neurogenic pulmonary edema and subsequent respiratory distress after peribulbar block<sup>52,53,54</sup>. Acute neurogenic pulmonary edema occurs primarily due to changes in Starling's forces, acute rise in hydrostatic pressures<sup>55</sup> and alteration in the permeability between the alveolar-capillary interfaces<sup>56</sup>.

8. Lastly, the type of anesthesia plays an important role. It is recommended to consider topical or general anesthesia in high risk cases. This almost

entirely eliminates the risks associated with both RBA and PBA. Although not possible in all kinds of ophthalmologic procedures, topical anesthesia may be employed in some ocular procedures<sup>57</sup>. However, since pain sensations may not be completely eliminated with topical anesthesia the need exists for the use of RBA or PBA<sup>58</sup>. If RBA or PBA is to be employed, ropivacaine is far less toxic than bupivacaine and substitution should be considered. Bupivacaine mediated pathophysiological processes, including cardiovascular collapse from inadvertent intravascular injection, are, in general, extremely difficult to treat.

## Conclusion

As an increasing number of ophthalmologic surgeries are performed in freestanding surgical centers where full anesthesia care may not be available, it is vital for health care providers to be cognizant of the anesthetic concerns and risks and complications with the administration of regional anesthesia for ophthalmologic surgeries. The complications resulting from the administration of LAs are numerous including confusion, diaphoresis, nausea and vomiting, aphasia, apnea, bradycardia, tachycardia, cyanosis, impaired hearing, cardiac arrest, confusion, dilatation of the contralateral pupil, dysphagia, drowsiness, facial paralysis, gaze palsy, hypertension, loss of consciousness, shivering, seizures, and cardiovascular collapse.

Loss of vision and even death can occur if complications are not promptly recognized and treated. The proximity of the eye to vital structures makes the administration of regional anesthesia for ocular surgery particularly risky. Hence, every attempt must be made to prevent the life-threatening and devastating consequences of the complications of LA blocks. Health care providers should be familiar with the types of anesthesia available, the inherent toxicities of the LAs being used, positioning of the eye, the type of needles, site of injection and techniques of injection. Recognition of complications should be quick and with immediate treatment of complications as appropriate. Facilities for advanced surgical procedures, ventilatory support, and intensive care should be immediately available.

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