Local Blocks and Regional Anesthesia in the Head and Neck

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KEYWORDS

• Local anesthesia • Pain control • Otolaryngology • Education

KEY POINTS

- There are a wide variety of local anesthetic agents with several different properties, such as onset time and length of action.
- Local and regional anesthetic techniques are often used for laceration repair or in addition to intravenous sedation, but some can be used for chronic pain situations, such as trigeminal neuralgia, migraines, and chronic cough, or in addition to topical anesthesia for intubation procedures.
- Although local and regional anesthesia is often safe, there are several complications that are possible, ranging from issues with the injection (eg, a broken needle or nerve injury) to reactions to the anesthetic agent (eg, allergic reaction, systemic toxicity). These possible reactions are even more important in certain patient populations.

INTRODUCTION

Local anesthesia refers to the application of an anesthetic with the intention to induce the loss of sensation in a particular part of the body, as opposed to general anesthesia where the anesthetic is applied systemically, resulting in loss of consciousness. Otolaryngologists perform a variety of procedures under both general anesthesia and local anesthesia. Because surgeons often inject the local anesthetic, it behooves them to understand the pharmacology of common drugs that they may be using. It is also important to have a firm grasp of facial anatomy so that local anesthetic can be precisely applied and to ensure the desired effect of anesthesia is achieved. This article provides an overview of the pharmacology of local anesthetics (**Table 1**) as well as a brief overview of sensory facial neural anatomy.

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Table 1
Description and qualities of various local anesthetic agents in otolaryngology

Drug	Classification	Onset	Half-Life (h)	Duration (with Epinephrine)	Lipid Solubility	pKa	Maximum Dose, (with Epinephrine) (mg/kg)	Application
Cocaine	Ester	Rapid	1	5–90 min	++	8.6	3	Topical for use in nasal procedures
Benzocaine	Ester	Rapid	—	5–10 min	—	2.5	—	Topical
Tetracaine	Ester	Rapid	—	120–180 min	++++	8.2	1–3 (1.5)	—
Lidocaine	Amide	Rapid	1.6	120 min (240 min)	++	7.8	4.5 (7)	Local infiltration, IV, intrathecal
Bupivacaine	Amide	Intermediate	3.5	4 h (8–12 h)	++++	8.1	2 (3)	—
Ropivacaine	Amide	Intermediate	1.9	3 h (6 h)	++++	8.1	2–3	—
Prilocaine	Amide	Intermediate	1.6	90 min (360 min)	++	7.8	5 (7.5)	Topical (component of Emla cream)

Data from Refs.[2,32–35]

Mechanism of Action

Local anesthetics inhibit the neuronal pain signal of peripheral nerve endings. Local anesthetic molecules reversibly bind to sodium channels, resulting in their inactivation and cessation of nerve signal propagation by blocking cell depolarization.[1] The fewer sodium channels present, the more easily a nerve signal can be blocked, so the smaller the diameter, the more sensitive a nerve is to local anesthesia. Therefore, smaller nerve fibers are generally easier to anesthetize because they require smaller volumes of local anesthetic for complete nerve signal blockade. In general, the sensitivities of differing nerve fibers from most sensitive to least are as follows: rapid-firing autonomic nerves, followed by sensory nerve fibers, then motor nerve fibers. Within sensory nerve fibers, pain fibers are the most sensitive, followed by pressure, and then proprioception signals.[2]

Classification

Local anesthetics can be classified by their chemical components into 2 groups: amides and esters. All local anesthetics are made up of an aromatic lipophilic ring attached to a hydrophilic tertiary amine by an intermediate chain. The intermediate chain is either an amide or an ester, which plays an important role in the chemical properties of the compound. It also plays a role in the metabolism of the drug, with amides being metabolized in the liver and esters metabolized in the plasma.[2] Simply put, a local anesthetic can be easily recognized as belonging to one chemical group versus the other by the name of the compound. Amides have 2 Is in their names, whereas esters have only 1.[3]

Lipid Solubility

This property is closely related to potency of the local anesthetic (although is not the entire picture). The more potent an agent, the more lipid soluble it likely is. Because nerve cell membranes are primarily composed of lipids, the more lipid soluble an agent is, the easier it can enter the cell membrane. However, other properties, such as vasodilation, can play a role in a drug's potency. Furthermore, agents that are highly lipid soluble can be easily sequestered in nearby lipid tissues, which can result in a longer duration of action but a lower potency.[4]

Protein Binding

This property determines the duration of action of a compound. The higher affinity for protein binding, the longer duration of action y/because of a firmer bond to the sodium channels. However, like potency, duration of action is also affected by the solubility of an agent as well as vasodilation or vasoconstriction.[2]

Diffusibility

This property is related to the speed of action of the local anesthetic, with faster-onset medications being diffused more quickly through tissues.[3]

Ionization

Most local anesthetics are weak bases and therefore exist partly in an ionized state at physiologic pH.[4] The nonionized state (or uncharged form) is the form able to cross the cell membrane. The amount of nonionized local anesthetic that exists in a solution is based on the pH of the environment and the pKa of the compound. The higher the pKa, the more ionized form exists. Therefore, the lower the pKa, the faster the onset of action of the drug. For anesthetics with a larger pKa, the onset of action can be increased by using a higher concentration of the drug.[4] In addition, as the pH of the

solution decreases, the equilibrium shifts toward the ionized form of the compound and this decreases the onset of action of the drug. This process explains why it can be difficult to anesthetize an infected area, because the pH is often decreased in infection.[2]

Vasoconstriction

With the exception of cocaine, all local anesthetics have vasodilatory properties to a degree, with lidocaine having the greatest vasodilatory properties of the local anesthetics.[4] Vasodilation is caused by the direct relaxation of the peripheral arteriolar smooth muscle fibers.[3] Vasoconstrictive agents, such as epinephrine, are often added to local anesthetics to increase the duration of action of the drug by delaying absorption and metabolism.[2]

Facial anatomy

The sensory innervation of the face predominantly comes from the trigeminal nerve (cranial nerve V) and its 3 main branches: the ophthalmic nerve (V1), the maxillary nerve (V2), and the mandibular nerve (V3). In general, sensory innervation to the upper third of the face is performed by branches of V1, the middle third of the face by branches of V2, and the lower third by branches of V3. Sensory innervation of the auricle stems from multiple cranial nerves, including the mandibular branch of trigeminal, as well as facial, glossopharyngeal, and cervical spinal nerves. Sensory innervation of the neck comes from the cervical plexus (ventral rami from C1–C4)[5] as well as the vagal nerve (cranial nerve X), specifically the superior laryngeal nerve branch. Some specific terminal nerve branches and their functions are discussed next.

Facial Anatomy

Nerve anatomy of the upper third of the face[5–8]

- Branches of V1
  - Ophthalmic: cutaneous nose, supraorbital region, forehead
  - Supraorbital: upper eye lid, forehead, scalp (to lambdoid suture)
  - Supratrochlear: conjunctiva, upper eye lid, glabella and midline forehead, anterior scalp
  - Infratrochlear: bridge of nose, medial aspect of eyelid below brow
  - Nasociliary: frontal sinus, anterior and posterior ethmoid sinuses, sphenoid sinus, anterior nasal septum, lateral nasal wall, skin of nasal tip, cornea, iris, ciliary body

Nerve anatomy of the middle third of the face

- Branches of V2
  - Zygomaticofacial: skin overlying cheek
  - Zygomaticotemporal: skin over side of lateral forehead and temples
  - Infraorbital: lower eyelid, lateral nares, upper lip
  - Nasopalatine: anterior portion of hard palate behind incisors
  - Greater and lesser palatine: hard and soft palate, lateral nasal wall
  - Superior alveolar: maxillary teeth, nasal floor, gums
  - Sphenopalatine: orbit, nose, buccal mucosa, palate, paranasal sinuses

Nerve anatomy of the lower third of the face

- Branches of V3
  - Buccal: cheek and oral mucosa and gingiva
  - Glossopharyngeal: posterior tongue and pharynx
  - Inferior alveolar: mandibular teeth
  - Lingual: anterior two-thirds of tongue

- Mental: cutaneous chin, lower lip, and side of mandible
- Auriculotemporal: outer ear and temporal region

Nerve anatomy of the auricle
- Auriculotemporal (branch of V3): superior pinna and temporal region
- Facial: ear canal, ear drum, conchal bowl
- Greater auricular (from C2–C3): inferior and posterior pinna, mastoid process, and parotid gland
- Lesser occipital (from C2): posterior medial surface of ear and back of neck

Nerve anatomy of the neck
- Greater occipital (from C2): posterior neck
- Superior laryngeal (branch of vagus): sensation of larynx above vocal cords and supraglottic mucosa
- Supraclavicular (from C3–C4): anterolateral shoulder and clavicle
- Transverse cervical (from C2–C3): neck around sternocleidomastoid muscle

LOCAL ANESTHETIC BLOCKS

There are a wide variety of local anesthetic blocks available. This article provides information on some that are more often used and ones that can used for more chronic conditions, such as glossopharyngeal and trigeminal neuralgia, neurogenic cough, or migraines. These anesthetic blocks have been divided based on their anatomic locations.

Upper Third of the Face

Supraorbital/supratrochlear block

Anatomy The supraorbital and supratrochlear nerves are terminal branches of the frontal nerve, which is a branch of the ophthalmic nerve (V1). The supraorbital nerve exits the skull at the supraorbital notch located at the midpupillary line. The supratrochlear nerve is on average 1.05 cm medial to the supraorbital nerve at its exit point from the supraorbital foramen.[9]

Nerve function Sensation to most of the forehead, with the supratrochlear providing more medial coverage and components of medial nasal dorsum and upper medial eyelid.

Reasons for regional block:
- Laceration repair
- Adjunct to surgery involving the upper third of the face in the setting of intravenous (IV) sedation
- Postoperative pain control
- Migraines and chronic headaches[10]

How to Injection is usually completed with either lidocaine or bupivacaine, with bupivacaine providing longer-lasting results. A 27-gauge needle is commonly used. In order to complete this nerve block, the supraorbital notch is palpated at the midpupillary line. The injection is carried in a lateral to medial direction, and a small amount of anesthetic (1–3 mL) is applied in a plane deep to the muscle. Ensure that there is no blood return before injection and that the foramen has not been entered. In order to apply adequate anesthesia to the supratrochlear nerve, injection must be carried medially to provide anesthesia to the nerve.

Zygomaticotemporal nerve block

Anatomy The zygomatic nerve is a terminal branch of the maxillary division of the trigeminal nerve (V2). The zygomatic nerve has 2 branches, the zygomaticotemporal and

the zygomaticofacial. The zygomaticotemporal nerve exits the cranium along the lateral orbital rim and is usually anywhere from the level of the lateral canthus to 1 cm inferior.

Nerve function Sensation to small area of the lateral forehead, anterior to hair line and above the zygoma.

Reasons for regional block:
- Used in conjunction with supraorbital nerve block for complete forehead anesthesia
- Laceration repair
- Adjunct to surgery in setting of IV sedation

How to The important aspect in this injection is to access the area behind the lateral orbital rim. In order to accomplish this, start by palpating the zygomaticofrontal suture. Slide the forefinger of the noninjecting hand into the depression just posterior and inferior to the suture. From here, use a 27-gauge 38-mm (1.5 inch) needle and place the needle just posterior to the finger. Slide the needle anteriorly to the area just behind the concave surface of the lateral orbital rim. Inject 1 to 3 mL of local anesthetic into this area.

Midthird of the Face

Dorsal nasal nerve block

Anatomy The dorsal, or external, nasal nerve is a branch of the anterior ethmoidal nerve. The anterior ethmoidal nerve is a division of the ophthalmic nerve (V1). The external nasal nerve ends up exiting the skull base at the junction between the bony and cartilaginous junction of the nose.

Nerve function Sensation to nasal dorsum, nasal alae, and nasal vestibule.

Reasons for regional block:
- Postoperative pain after nasal surgery[11]
- External nasal neuralgia[12]
- Adjunct for nasal surgery

How to A 27-gauge needle is used to inject the bony cartilaginous junction in the sub–superficial musculoaponeurotic system plane. The injection should be lateral to the midline (~5 mm). Approximately 1 to 2 mL of local anesthetic can be used.

Infraorbital nerve block

Anatomy The infraorbital nerve is a terminal branch of the maxillary nerve (V2). This nerve exits the skull at the infraorbital foramen. The foramen is approximately 0.5 to 1.0 cm below the midpupillary line.

Nerve function Sensation to midface, including cheek, upper lips, and lower eyelid.

Reasons for regional block:
- Reduce emergence agitation following nasal surgery[13]
- Laceration repair of the midface
- Adjunct to surgery in setting of IV sedation
- Possible pain reduction following cleft lip repair[14]
- Treat refractory trigeminal neuralgia isolated to the midface[15]

How to There are 2 approaches available. The first is through an intraoral approach. Through this approach, 1 hand palpates the inferior orbital rim and retracts the cheek, which should expose the gingivobuccal sulcus adjacent to the second

premolar. A 27-gauge needle is inserted just lateral into the buccal mucosa from the gingivobuccal sulcus and is then carried to approximately 0.5 to 1.0 cm below the infraorbital rim at the midpupillary line. Inject approximately 3 to 5 mL of anesthetic in this area. Care must be taken to prevent anesthetic from entering into the orbit. The other approach is through an external approach. The infraorbital foramen is identified below the inferior orbital rim at the midpupillary line. A 27-gauge needle is used to inject through the skin to the area of the infraorbital rim, and is carried below the muscle. There must be no intraluminal injection because the facial artery is in close proximity.

LOWER THIRD FACIAL BLOCKS

Mental Nerve Block

Anatomy

The mental nerve is the terminal branch of the mandibular division of the trigeminal nerve (V3). This nerve exits the mental foramen between the first and second premolars, roughly 1 cm below the gum line. This nerve should sit in the midpupillary line and be in the same plane as the supraorbital notch and the infraorbital foramen.

Nerve function

Sensation to the lower lip and chin.

Reasons for regional block:

- Laceration repair
- Lower lip procedures
- Treat trigeminal neuralgia isolated to the lower lip[15]

How to

This block is completed intraorally. The lower lip of the side of the injection is retracted, making the mucosa taut at the gingivobuccal sulcus. The first and second mandibular premolars are identified. A 27-gauge needle is inserted approximately 1 cm below the gum line at a 45° angle until bony contact is made. Once this is reached, withdraw the needle slightly and inject approximately 2 to 3 mL of local anesthetic.

NECK REGIONAL BLOCKS

Glossopharyngeal Nerve Block

Anatomy

The glossopharyngeal nerve originates from the medulla and exits the skull base at the jugular foramen. This nerve then descends with the vagus nerve posterior and medial to the styloid process in the parapharyngeal space. Numerous branches exist after the glossopharyngeal nerve has exited the skull base.

Nerve function

Provides motor, special sensory, and sensory information. From a sensory standpoint, it provides somatic information along pharyngeal walls, base of tongue, anterior surface of epiglottis, ear, and tympanic membrane.

Reasons for regional block:

- Treat glossopharyngeal neuralgia[16]
- Reduce posttonsillectomy pain[17]
- Adjunct during upper esophageal or awake airway procedures[18]
- Eagle syndrome[19]

How to

There are both external and intraoral approaches. There are several different ways to approach the external injections, but most use ultrasonography guidance to identify the large blood vessels in the area. The details of the procedure are too in depth for the overall goal of this article, but suffice to say that the injections come with great risk. These risks include bleeding (secondary to location adjacent to the internal jugular vein and carotid) as well as the risk of anesthesia to the vagus nerve (leading to progressive hoarseness, and, if completed bilaterally, bilateral cord paralysis). Intraoral approaches focus more on blocking distal branches of the glossopharyngeal nerve and not necessarily targeting the main trunk. Often, steroids can be used in addition to local anesthetic to provide symptom improvement. From an intraoral standpoint, there are 2 primary techniques. In the anterior tonsillar pillar approach, the tongue is retracted away from the anterior tonsillar pillar. The palatoglossal fold is identified at the base where it transitions to the tongue. From here, a spinal needle is used and 1 to 2 mL of anesthetic is applied roughly 0.5 cm deep in this area.[20,21] The posterior tonsillar pillar approach is similar but targets an injection through the posterior pillar at the base of tongue. This approach is tougher because the tongue has to be retracted further and gagging often occurs.

Superior Laryngeal Nerve Block

Anatomy

The vagus nerve exits the skull base at the jugular foramen and runs the course of the neck in the carotid space. There are several branches of the vagus nerve, including the superior laryngeal and inferior laryngeal nerves. The superior laryngeal nerve splits into 2 components: an external and internal branch. The internal branch of the superior laryngeal nerve enters the larynx through the thyrohyoid membrane.

Nerve function

The internal branch of the superior laryngeal nerve provides sensory information from the larynx. The external branch provides motor control to the cricothyroid muscle.

Reasons for regional block:

- Treatment of neurogenic cough[22,23]
- Adjunct during awake fiberoptic intubation and laryngeal procedures[24–27]

How to

There are several techniques in completing the superior laryngeal nerve block. Ultrasonography can be used to aid in identification of the thyroid cartilage and the hyoid bone, especially in patients with difficult external neck anatomy. This technique can be completed with the patient supine or sitting upright. Usually a combination of local anesthetic and steroid is used. One hand grasps the larynx and displaces the hyoid to the side of the injection. The space between the thyroid cartilage and hyoid bone represents the thyrohyoid membrane. The greater cornu and superior tubercle of the thyroid cartilage can indicate roughly the location of the internal branch. The carotid can be used as a posterior border. The injection can be completed from an anterior to posterior direction, which is thought to provide the most coverage. However, approaching posteriorly allows greater security, knowing that the carotid is posterior to the injection point. The use of a 27-gauge needle and approximately 1 to 2 mL of topical anesthetic mixed with steroid is sufficient. Bilateral injections can be done for fiberoptic intubations, but, for cough in the outpatient setting, unilateral injections should be completed. A trigger point can be palpated and can be used for injection location.[23]

POTENTIAL COMPLICATIONS OF LOCAL AND REGIONAL BLOCKS

Administration of local anesthesia is generally safe and effective, but there are several potential complications associated with the use of local anesthetics.

Overdose

It is imperative to calculate maximum dosages of various local anesthetics to prevent overdose, especially in pediatric patients, because dosages are calculated in milligrams per kilogram. A list of commonly used local anesthetics with their associated maximum dosages is provided in **Table 2**. The addition of epinephrine increases the maximum dose allowable for most local anesthetics. Symptoms associated with an overdose are the same as those seen in systemic toxicity, which is discussed in further detail later.

Intravascular Injection

Even when low doses of local anesthetics are used, injection intravascularly can lead to rapid onset of systemic toxicity. When injecting local anesthetics, the syringe should be drawn back after the needle is inserted to confirm it is not located intravascularly. During negative aspiration, the presence of a flash of blood should prompt repositioning instead of injection. In situations where the anatomy is difficult or there are large vessels in close proximity to the injection site, ultrasonography guidance may be helpful to confirm location of the needle.

Allergic Reactions

Allergic reactions to local anesthetics are very rare, especially with amides, in which the presentation of an allergic reaction should prompt consideration of an overdose or intravascular injection.[28] Esters are more allergenic because of their para-aminobenzoic acid (PABA) structure. Patients with a PABA allergy or atypical pseudocholinesterase activity are at higher risk. Tetracaine, specifically, has been shown to have a higher incidence of allergic reactions.

In some cases, patients are allergic to the preservative in local anesthetics, such as methylparaben in amides (which is similar to PABA), or sodium bisulfite, which is commonly found in epinephrine.[28] In addition, when local anesthesia is administered in the setting of latex glove use, consider a potential latex allergy instead. True allergic reactions are either type I or type IV hypersensitivity, with type IV being more common. In patients with a suspected allergy, patch testing can be performed to confirm the diagnosis.

Table 2
Maximum doses for local anesthetic agents

Anesthetic	Maximum Dose (mg/kg)
Lidocaine	5
Lidocaine with epinephrine	7
Mepivacaine	4.4
Mepivacaine with epinephrine	6.6
Articaine	7
Bupivacaine	1.3
Prilocaine	2.7

Symptoms associated with allergic reaction to local anesthetics include itching, edema, hives, nasal congestion and rhinorrhea, wheezing, shortness of breath, and headache. Severe allergic reactions can lead to toxic eruptions, bronchospasm, and anaphylaxis. For mild reactions, treatment consists of an oral antihistamine and/or hydrocortisone cream. In more severe cases, the use of an epinephrine pen, oxygen supplementation, and urgent transfer to a monitored setting is recommended.

Broken Needle

Rarely, during injection of local anesthetic, the needle breaks off into the tissue. To avoid this complication, avoid using a short needle and burying it to the hub when injecting.[28] In addition, avoiding bending the needle before injection decreases the potential for breakage. Needles that break off should be removed if possible, because, over time, they can migrate and cause damage to vessels or other structures.

Nerve Injury

When local anesthesia is administered close to a named nerve, there is potential for nerve injury. The mechanism for injury can be related to direct trauma from the needle, epineural hemorrhage, or to a neurotoxic effect from the anesthesia. Nerve injury can lead to persistent numbness, burning, or motor deficits.

Methemoglobinemia

Hemoglobin is a protein in the blood that contains ferrous iron. Ferrous iron is adept at releasing oxygen into the tissues as the blood circulates. In cases of oxidative stress, the heme group of the hemoglobin molecule is oxidized to the ferric state, converting hemoglobin to methemoglobin. Because methemoglobin molecules have a limited ability to release bound oxygen to tissues, high levels of methemoglobin in the blood lead to hypoxia. Hypoxia is manifested by a chocolate-brown appearance of arterial blood, in contrast with its typical bright-red hue. Patients being monitored with pulse oximetry show a discrepancy between saturation level and a measured arterial oxygen partial pressure.[29]

Certain local anesthetics can prompt oxidation of the heme group to create methemoglobin. Most cases in the literature involve benzocaine, but prilocaine, lidocaine, and tetracaine have also been implicated.[29] Patients with congenital methemoglobinemia, caused by NADH deficiency, are at higher risk for exacerbation with concurrent use of local anesthetics. In addition, the use of local anesthetics in the setting of other oxidative drugs can prompt methemoglobin production. Methemoglobinemia can cause seizures, respiratory compromise, shock, and coma. In severe cases, hypoxic encephalopathy, myocardial infarction, and even death can occur. Treatment involves the use of methylene blue infusion, which converts to leukomethylene blue, a reducing agent that converts methemoglobin to deoxyhemoglobin.[29] Admission and observation in a monitored setting is recommended, because, in some cases, a rebound effect occurs 4 to 12 hours after the initial successful methylene blue infusion. Patients with glucose-6-phosphate dehydrogenase deficiency typically cannot convert methylene blue to leukomethylene blue, so ascorbic acid is recommended instead.

Systemic Toxicity

Systemic toxicity related to local anesthetics is estimated to occur in 2.5 per 10,000 blockades.[30] The 2 organ systems typically affected by toxicity are the cardiac system and central nervous system (CNS). Risk factors include large doses, rapid absorption, and intravenous injection. There is an inverse relationship between toxicity and speed of injection, with faster injections decreasing the blood level threshold for symptoms.[31]

In cases of systemic toxicity, patients should be admitted to a monitored setting. Resuscitation and monitoring should last until the local anesthetic is completely metabolized.

CNS toxicity typically manifests first, because doses needed to cause CNS symptoms are lower than doses needed to cause cardiac symptoms. Local anesthetics block inhibitory pathways in the cerebral cortex, causing unopposed excitatory activity,[31] which manifests as dizziness, trouble concentrating, confusion, tinnitus, and circumoral numbness. Severe reactions can progress to seizures and respiratory arrest. In patients taking other CNS-suppressing drugs, the excitatory phase may be silent, progressing directly to seizures and respiratory depression. Treatment of CNS toxicity includes securing the airway; providing supplemental oxygen; and the use of benzodiazepines, barbiturates, or propofol to decrease excitatory neurologic activity and seizure risk.[31]

Cardiac toxicity occurs when local anesthetics bind to sodium. Because cardiac cells rely on sodium-initiated depolarization, local anesthetics can affect conduction and contraction, leading to arrhythmias and decreased cardiac output.[30] Small amounts of local anesthetics create vasoconstriction and increased sympathetic activity, which cause a small increase in cardiac output, heart rate, and blood pressure. As toxicity occurs, peripheral vasodilation occurs, leading to hypotension and decreased cardiac output. Profound hypotension, arrhythmias, and cardiac arrest can follow. Treatment options for cardiac toxicity include vasopressin for hypotension and amiodarone for arrhythmias. Intravenous intralipid can absorb circulating local anesthetic and help decrease cardiovascular effects. Use caution with epinephrine, because it can exacerbate arrhythmias. Cardiopulmonary bypass may be used in the most severe situations.

Special Populations

- Cardiac transplant patients: because the heart is denervated, these patients can be more sensitive to epinephrine.[28]
- β-Blockers, amiodarone, and digoxin: there is an increased risk of arrhythmia with the use of bupivacaine in patients on these medications.
- Sulfonamides, chloroquine, dapsone, phenobarbital, and metoclopramide: these medications increase the risk of methemoglobinemia when used in conjunction with local anesthetics.
- Pregnancy: mepivacaine, bupivacaine, and articaine are considered pregnancy category C.[28]
- Porphyria: avoid the use of local anesthetics during a porphyric crisis. It is difficult to differentiate between neurologic sequelae of porphyria and the anesthetic.

SUMMARY

There are a wide variety of local anesthetic agents available. The selection of which to use depends largely on the goal of the regional anesthetic as well as any potential patient comorbidities. Various local anesthetic blocks are described in this article. Although they are often used for laceration repair or local procedures, some can be used for more chronic conditions (eg, neurogenic cough, migraines, or even trigeminal neuralgia). They can often be used as an adjunct to surgical procedures as well (either providing additional benefit in conjunction with IV medications or to aid fiberoptic intubations). Although the use of local anesthetics is generally safe, it is important to be mindful of potential complications and certain patient populations that may be at high risk for certain local anesthetics.

DISCLOSURE

The authors have nothing to disclose.

REFERENCES

1. Thiagarajan B. Local anesthesia of nose and nasal cavity- a review. Global Journal of Otolaryngology 2017;4(4):001–3.
2. Becker DE, Reed KL. Local anesthetics: review of pharmacological considerations. Anesth Prog 2012;59(2):90–103.
3. McLeod IK. Local anesthetics. Medscape 2019. Available at: https://emedicine.medscape.com/article/873879-overview. Accessed October 15, 2019.
4. Eappen S, Datta S. Pharmacology of local anesthetics. Seminars in Anesthesia, Perioperative Medicine and Pain 1998;17(1):10–7.
5. Burnett G, Demaria S, Levine AI. Regional anesthesia and acute pain management. Otolaryngol Clin North Am 2019. https://doi.org/10.1016/j.otc.2019.08.013.
6. Moskovitz JB, Sabatino F. Regional nerve blocks of the face. Emerg Med Clin North Am 2013;31(2):517–27.
7. Szymanski A, Geiger Z. Anatomy, head and neck, ear. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2019. Available at: https://www.ncbi.nlm.nih.gov/books/NBK470359/. Accessed June 17, 2020.
8. Waxenbaum JA, Bordoni B. Anatomy, head and neck, cervical nerves. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2019. Available at: https://www.ncbi.nlm.nih.gov/books/NBK538136/. Accessed June 17, 2020.
9. Konofaos P, Soto-Miranda MA, Halen JV, et al. Supratrochlear and supraorbital nerves. Ophthalmic Plast Reconstr Surg 2013;29(5):403–8.
10. Miller S, Lagrata S, Matharu M. Multiple cranial nerve blocks for the transitional treatment of chronic headaches. Cephalalgia 2019;39(12):1488–99.
11. Ibrahim M, Elnabtity AM, Keera A. Efficacy of external nasal nerve block following nasal surgery. Anaesthesist 2018;67(3):188–97.
12. García-Moreno H, Aledo-Serrano Á, Gimeno-Hernández J, et al. External nasal neuralgia: a neuropathic pain within the territory of the external nasal nerve. Headache 2015;55(9):1259–62.
13. Choi H, Jung SH, Hong JM, et al. Effects of bilateral infraorbital and infratrochlear nerve block on emergence agitation after septorhinoplasty: a randomized controlled trial. J Clin Med 2019;8(6):769.
14. Feriani G, Hatanaka E, Torloni MR, et al. Infraorbital nerve block for postoperative pain following cleft lip repair in children. Cochrane Database Syst Rev 2016. https://doi.org/10.1002/14651858.cd011131.pub2.
15. Perloff MD, Chung JS. Urgent care peripheral nerve blocks for refractory trigeminal neuralgia. Am J Emerg Med 2018;36(11):2058–60.
16. Liu Q, Zhong Q, Tang G, et al. Ultrasound-guided glossopharyngeal nerve block via the styloid process for glossopharyngeal neuralgia: a retrospective study. J Pain Res 2019;12:2503–10.
17. Ahmed SA, Omara AF. The effect of glossopharyngeal nerve block on post-tonsillectomy pain of children; randomized controlled trial. Anesth Pain Med 2019;9(2):e90854.
18. Ortega Ramírez M, Linares Segovia B, García Cuevas MA, et al. Glossopharyngeal nerve block versus lidocaine spray to improve tolerance in upper gastrointestinal endoscopy. Gastroenterol Res Pract 2013;2013(2013):4.
19. Maher T, Shankar H. Ultrasound-guided peristyloid steroid injection for eagle syndrome. Pain Pract 2017;17(4):554–7.

20. Saliba DL, Mccutchen TA, Laxton MJ, et al. Reliable block of the gag reflex in one minute or less. J Clin Anesth 2009;21(6):463.
21. Isbir CA. Treatment of a patient with glossopharyngeal neuralgia by the anterior tonsillar pillar method. Case Rep Neurol 2011;3(1):27–31.
22. Dhillon VK. Superior laryngeal nerve block for neurogenic cough: A case series. Laryngoscope Investig Otolaryngol 2019;4(4):410–3.
23. Simpson CB, Tibbetts KM, Loochtan MJ, et al. Treatment of chronic neurogenic cough with in-office superior laryngeal nerve block. Laryngoscope 2018;128(8): 1898–903.
24. Ramkumar R, Arora S, Bhatia N, et al. Ultrasound guided superior laryngeal nerve block as an adjuvant to generalanesthesia during endoscopic laryngeal surgery: A prospective, randomized, double-blind trial. Am J Otolaryngol 2019; 40(1):30–5.
25. Mathur PR, Jain N, Kumar A, et al. Comparison between lignocaine nebulization and airway nerve block for awake fiberoptic bronchoscopy-guided nasotracheal intubation: a single-blind randomized prospective study. Korean J Anesthesiol 2018;71(2):120–6.
26. Chatrath V, Sharan R, Jain P, et al. The efficacy of combined regional nerve blocks in awake orotracheal fiberoptic intubation. Anesth Essays Res 2016; 10(2):255.
27. Sawka A, Tang R, Vaghadia H. Sonographically guided superior laryngeal nerve block during awake fiberoptic intubation. A A Case Rep 2015;4(8):107–10.
28. Ogle OE, Mahjoubi G. Local anesthesia: agents, techniques, and complications. Dent Clin North Am 2012;56(1):133–48.
29. Guay J. Methemoglobinemia related to local anesthetics: a summary of 242 episodes. Anesth Analg 2009;108(3):837–45.
30. Dickerson DM, Apfelbaum JL. Local anesthetic systemic toxicity. Aesthet Surg J 2014;34(7):1111–9.
31. Dewaele S, Santos AC. Toxicity of local anesthetics. In: Hadzic A, editor. Peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd edition. New York: McGraw-Hill; 2012.
32. Mumba JM, Kabambi FK, Ngaka CT. Pharmacology of local anaesthetics and commonly used recipes in clinical practice. In: Erbay RH, editor. Current topics in anesthesiology. IntechOpen; 2017. Available at: https://www.intechopen.com/books/current-topics-in-anesthesiology/pharmacology-of-local-anaesthetics-and-commonly-used-recipes-in-clinical-practice.
33. El-Boghdadly K, Pawa A, Chin KJ. Local anesthetic systemic toxicity: current perspectives. Local Reg Anesth 2018;11:35–44.
34. Rosenberg PH, Veering BT, Urmey WF. Maximum recommended doses of local anesthetics: a multifactorial concept. Reg Anesth Pain Med 2004;29(6):564–75.
35. Heavner JE. Local anesthetics. Curr Opin Anaesthesiol 2007;20(4):336–42.