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The use of regional anesthesia for head and neck, and more specifically, maxillofacial procedures has a long history in anesthesiology. Indeed, the introduction of local anesthetics such as cocaine and procaine revolutionized much of these procedures as early as the late 1800s, when Koller began using cocaine as an anesthetic. Today, a working knowledge of the pharmacology of local anesthetics and of head and neck anatomy is all that one needs to safely and effectively utilize regional anesthetic techniques for intraoperative maxillofacial surgery and for acute postoperative pain management strategies.

Essentials of Regional Anesthesia

Regional anesthesia can be described as either central or neuraxial regional anesthesia including spinal and epidural anesthesia and peripheral nerve anesthesia. The use of regional anesthetic techniques has evolved from their use as a sole anesthetic, under which a surgical procedure could be performed, to a way of providing intraoperative anesthesia as one component of a balanced anesthetic and as but one strategy for postoperative analgesia. An evolution has occurred from relying on surface landmarks to the increased use of nerve stimulation and direct visualization with ultrasound technology that has certainly improved patient comfort and perhaps safety and efficacy of peripheral nerve block techniques. The role of these newer technologies in OMFS procedures is less well defined, however.

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Indications

Instituting regional anesthesia is dependent on a number of conditions: the patient's considerations including comorbidities, anatomy, and procedural consent, and the surgical considerations including the planned surgical procedure and technique, the surgeon's request, and lastly the availability of necessary resources. Although the use of regional anesthetic techniques is not without risk, it offers a very viable alternative to general anesthesia or "deep" monitored anesthetic care (MAC) that risks aspiration and apnea/hypoxemia. The disadvantages of peripheral nerve blocks include local anesthetic toxicity, permanent nerve damage and paresthesias, and potential damage of surrounding structures (depending on the block performed, this may include the eye, other nerves, or even brain injury).

When planning to perform head and neck procedures under regional anesthesia, it is critical that the anesthetic and surgical teams have a clear understanding of surgical and anesthetic expectations. Due to the proximity of the surgical site to the airway, patients must remain cooperative, breathe spontaneously, and, because of the risk of fire in oxygen-enriched areas, require minimal supplemental oxygen and airway support. Therefore, the surgical team must be aware that heavy sedation will not be considered a safe option since a major goal for anesthesiologists in these cases is to limit oxygen supplementation. If the surgical team is not confident that they will be able to accomplish the planned surgery given these conditions, then general anesthesia with a protected airway should be employed to reduce the risk of fire and respiratory compromise.

Patient Education and Selection

As with all cases performed under regional anesthesia as the primary technique, patient education, motivation, and selection are critical. This is especially important in

maxillofacial surgery given the proximity of the surgical site to the face and airway. Preoperatively, patients must be educated and have a clear understanding of the surgical procedure and a clear understanding of the anesthetic expectations. Patients presenting for and requesting regional anesthesia for their surgery must understand that minimal sedation will be employed to assure their cooperation, airway maintenance, and oxygenation. With this knowledge, many patients may opt for general anesthesia. Patients should be mature and generally free of anxiety disorders or claustrophobia to cooperate. The ability to communicate with the patient intraoperatively is also critical. There should be no language barrier between providers and patients undergoing head and neck surgery under regional anesthesia. If these conditions do not exist, the option to perform the procedure exclusively under a regional anesthetic must be questioned. It should be kept in mind that in such a situation use of both a general anesthetic supplemented with a regional block is a viable alternative and affords the benefit to reduce intraoperative anesthetic exposure will be providing postoperative analgesia.

Contraindications

Most contraindications to peripheral nerve blockade using local anesthetics are relative. Local infections or tumors at the sight of the injection are absolute contraindications and although extremely rare hypersensitivity to local anesthetics would preclude their use. Bleeding disorders, bloodstream infections, and preexisting neuropathy are relative contraindications. Patient refusal is of course an absolute contraindication.

In adult patients, neuraxial or peripheral regional anesthesia is generally not instituted after the induction of general anesthesia for fear of undetected neuronal injury. However, most procedural regional anesthesia for maxillofacial surgery is essentially a “field block” and can be performed safely even after the induction of general anesthesia.

Premedication

Premedication with small doses of a benzodiazepine and/or opioid helps reduce anxiety and raise the pain and seizure (in the case of benzodiazepines) thresholds. Intravenous midazolam 1–2 mg for most adults is a safe and effective dose. Fentanyl 25–50 mcg IV is similarly effective. In general, only light sedation is desirable so the patient can report paresthesias during the performance of regional nerve blockade. While placing the block supplemental oxygen should generally be administered to all patients via nasal cannula or facemask to reduce the incidence of hypoxemia following even light sedation.

Local Anesthetics

Overview

Local anesthetic agents are a group of heterogeneous drugs that reversibly block transmission of nerve conduction to diminish or abolish sympathetic, sensory, and or motor function. The injection of local anesthetics near nerves is most often used in regional anesthesia either as a stand-alone technique or in conjunction with monitored anesthesia care or general anesthesia. Local anesthetics can also be infiltrated and applied topically to mucous membranes and skin to provide anesthesia. Local anesthetics have wide-ranging application in maxillofacial surgery, from minor office procedures to more invasive open surgeries. Application of local anesthetic appears to decrease postoperative pain in common head and neck procedures. Of course, local anesthesia is also used successfully for regional anesthesia of the head and neck whereby entire surgeries may be performed without general anesthesia and postoperative pain relief may be effectively achieved.

Pharmacology

Local anesthetics are sodium channel blocking drugs that decrease neural transmission of nociceptive signals. Normally, following activation by excitatory signals, activation of voltage-gated sodium channels leads to an action potential that travels down the axon. Impulses are then expedited down the axon by hopping from successive nodes of Ranvier (saltatory conduction). Local anesthetics function by binding to the inner aspect of neuronal sodium channels in order to inhibit this impulse propagation. Local anesthetics preferentially block small, myelinated, and high-frequency firing nerves. In general, blockade of sympathetic nerve fibers occurs first, sequentially followed by pain and temperature, touch, and then motor fibers. Due to the presence of sodium channels, the central nervous and cardiovascular systems are the sites where local anesthetic toxicity will predominate.

Structurally, local anesthetics are composed of an aromatic ring and an amine group, linked centrally by either an ester or amide bond (Table 5.1). This central portion is used to classify the two groups, either esters or amides. Esters are generally metabolized to p-aminobenzoic acid (PABA) by plasma cholinesterase, have a short half-life in the plasma (<1 min), and are more likely to elicit (rare) allergic reactions (due to PABA). Cocaine is a unique ester local that undergoes extensive liver metabolism. Amides are degraded in the liver and have a half-life of 2–3 h. All local anesthetics are weak bases with a pKa of 7–9, existing mainly in the cationic form at physiologic pH.

Table 5.1 Physiochemical and pharmacological properties of commonly used local anesthetic agents for subcutaneous infiltration

Local anesthetic	Pka	Onset	Duration	Maximum dose ^a (mg/kg)
<i>Esters</i>				
Procaine	8.9	Slow	Short	11 (13)
Chlorprocaine	8.5	Fast	Short	10 (12)
Tetracaine	8.4	Slow	Long	1.5 (2)
<i>Amides</i>				
Prilocaine	7.9	Fast	Moderate	5 (7)
Lidocaine	7.9	Fast	Moderate	5 (7)
Mepivacaine	7.6	Fast	Moderate	3 (5)
Bupivacaine	8.1	Moderate	Long	2 (2.5)
Ropivacaine	8.1	Fast	Long	2 (2.5)
Etidocaine	7.7	Slow	Long	4

^aDose in parenthesis is the maximum dose in mg/kg with the addition of epinephrine

Local anesthetics must first cross the cell membrane in the uncharged state before binding to the inner pore of the sodium channel (Fig. 5.1). With the exception of chlorprocaine, local anesthetics with a pKa closer to physiologic pH are more likely to exist in the unprotonated, more hydrophobic form and therefore have a faster onset of action. In general the speed of onset of local anesthetics is therefore inversely related to the pKa. Adding sodium bicarbonate to a local anesthetic solution in order to increase the unprotonated fraction may speed the rate of onset.

Potency of local anesthetics is related to their lipid solubility. Protein binding (to α -1 acid glycoprotein), peripheral vascular effects, and lipid solubility determine duration of action. Epinephrine (1:200,000) can be added to local anesthetic solutions to prolong the duration of action by causing local vasoconstriction and decreasing systemic absorption. Use of epinephrine is best avoided in very peripheral nerve blocks, patients with heart disease, and uncontrolled hypertension. Refer to for pharmacological properties of individual local anesthetic agents.

Toxicity

Central Nervous System

When two different local anesthetics are used, the maximum safe dose should be assumed to be additive and not independent. The most likely adverse effects of local anesthetics are due to their initial excitatory (seizures) and ultimately depressive (coma) effects on the central nervous system. Patients generally will first complain of lightheadedness or dizziness, followed by tinnitus and/or perioral numbness. They may then become agitated which could progress to tonic-clonic seizures. If left untreated reversible coma (general anesthesia) will follow. It is essential to have immediate access to resuscitation and intubation equipment in addition to anticonvulsant medications (midazolam 0.03–0.06 mg/kg

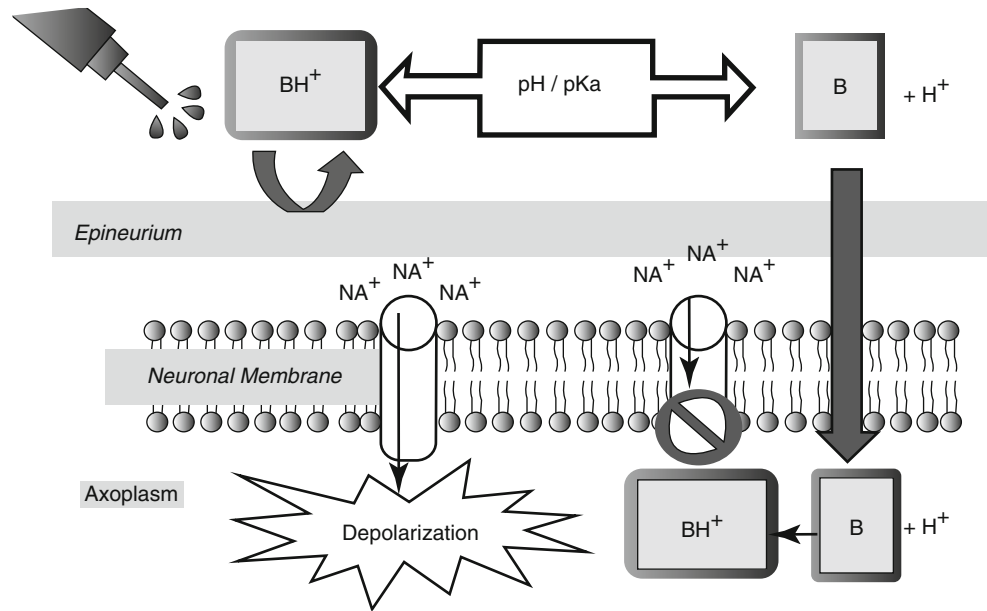
or propofol 0.5–1 mg/kg) when performing nerve blocks. This is especially true in head and neck blocks where delivery of local anesthetics directly to the central nervous system can be incredibly efficient. Hyperventilation may help lessen this toxicity by avoiding acidosis and hypercarbia, which increases cerebral blood flow and decreases local anesthetic binding to serum proteins.

Cardiovascular System

Local anesthetics are class 1 antiarrhythmic agents that have depressant effects on cardiac conduction, hence the use of lidocaine to treat excitatory arrhythmias. Excessive inhibition of these channels, however, can lead to prolonged conduction and decreased inotropy. Local anesthetic overdose also leads to arteriolar dilation and hypotension. The exception to this trend is cocaine, which inhibits reuptake of norepinephrine and dopamine, leading to hypertension, increased inotropy, tachyarrhythmias, and coronary spasm. Therefore, cocaine should be used cautiously in patients with cardiac disease as it can precipitate myocardial ischemia and even cerebrovascular accidents.

Bupivacaine has increased affinity for sodium channels and is more likely to produce severe cardiotoxic effects. Refractory ventricular tachycardia or fibrillation from bupivacaine is particularly resistant to treatment and is often fatal. The use of 20 % intralipid solution has been found to be effective as a rescue agent in malignant dysrhythmias and cardiovascular collapse from bupivacaine that is unresponsive to traditional resuscitation protocols. Ropivacaine is a stereoisomer of bupivacaine that has reduced affinity for cardiac sodium channels and is accordingly less cardiotoxic (although still more cardiotoxic than lidocaine). With all local anesthetics, aspiration prior to injection decreases the risk of intravascular injection. Figure 5.2 below outlines the American Society of Regional Anesthesia and Pain Medicine Guidelines for the treatment of local anesthetic toxicity (LAST).

Fig. 5.1 Diagram of mechanism of local anesthetic action. Local anesthetic exists as both a charged (BH^+) and uncharged (B) basic structure. The proportion of each is determined by the pK_a of the anesthetic and the pH of the environment. Once the uncharged form reaches the intracellular environment, it is protonated and is able to block the sodium channel



- Get Help
- Initial Focus
 - Airway management: ventilate with 100% oxygen
 - Seizure suppression: benzodiazepines are preferred; AVOID propofol in patients having signs of cardiovascular instability
 - Alert the nearest facility having cardiopulmonary bypass capability
- Management of Cardiac Arrhythmias
 - Basic and Advanced Cardiac Life Support (ACLS) will require adjustment of medications and perhaps prolonged effort
 - AVOID vasopressin, calcium channel blockers, beta blockers, or local anesthetic
 - REDUCE individual epinephrine doses to <1 mcg/kg
- Lipid Emulsion (20%) Therapy (values in parenthesis are for 70kg patient)
 - Bolus 1.5 mL/kg (lean body mass) intravenously over 1 minute (~100mL)
 - Continuous infusion 0.25 mL/kg/min (~18 mL/min: adjust by roller clamp)
 - Repeat bolus once or twice for persistent cardiovascular collapse
 - Double the infusion rate to 0.5 mL/kg min if blood pressure remains low
 - Continue infusion for at least 10 minutes after attaining circulatory stability
 - Recommended upper limit: Approximately 10 mL/kg lipid emulsion over the first 30 minutes
- Post LAST events at www.lipidrescue.org and report use of lipid to www.lipidregistry.org

Fig. 5.2 American Society of Regional Anesthesia and Pain Medicine Guidelines for the treatment of local anesthetic toxicity

Hematologic System

Some local anesthetics can cause the development of methemoglobinemia, a condition characterized by excessive methemoglobin. The iron moiety of methemoglobin is oxidized (ferric [Fe³⁺] rather than ferrous [Fe²⁺]). Oxygen delivery is compromised resulting in tissue hypoxia. Patients report feeling short of breath, appear cyanotic, and have a pulse oximeter reading that approaches 85 %. Their blood will classically look “chocolate brown.” Intravenous methylene blue (a reducing agent) 1–1.5 mg/kg intravenous is the treatment.

Local Anesthetic Selection

Although regional anesthesia of the head and neck generally does not require the use of large volumes of local anesthesia, toxicity must be considered in light of the agents' individual characteristics such as onset duration and their use in close proximity to major blood vessels. Good surgical anesthesia is obtained only when local anesthetic is injected close to the nerve(s) to be blocked. Common local anesthetic solutions that are used for surgical anesthesia include lidocaine 1.5–2 %, mepivacaine 2 %, bupivacaine 0.5 %, levobupivacaine 0.5 %, or ropivacaine 0.5 %. In general more dilute solutions are recommended for postoperative analgesia due to the decreased motor blockade; since motor blockade may be less of a concern with head and neck surgery, the authors recommend using more concentrated solutions for improved efficacy and duration of action.

Utility as Topical Anesthetics

Besides use by injection in regional nerve blocks and tissue infiltration, local anesthetic agents can also be used topically. Local anesthetic sprays are available for application to the esophagus and larynx for endoscopic procedures. Several agents can be sprayed into the larynx and trachea prior to intubation to reduce the incidence of coughing during emergence from anesthesia (i.e., laryngotracheal anesthesia) and decrease the sympathetic response to tracheal intubation. The cricothyroid membrane may also be punctured with a small needle for the direct application of local anesthetic to the larynx and trachea. Local anesthetics can be delivered via nebulizer for endoscopy or awake intubation.

Eutectic mixture of local anesthetics (EMLA) is a cream composed of 2.5 % lidocaine and 2.5 % prilocaine

that may be applied to intact skin as a local anesthetic. Use is limited by onset time (requires at least 30 min) and the risk of methemoglobinemia.

Applications in Maxillofacial Surgery

Anatomy

Understanding the sensory innervation of the head and neck is critical in planning and conducting regional anesthesia for the head and neck (Table 5.2).

The majority of the sensory input of the face, head, and neck is supplied by the trigeminal nerve (CN V) and the cervical plexus (C2, C3, and C4). Whereas the sensory innervation of the nasal cavity is predominately from the V1 and V2 branches of CN V, the sensory innervation of the tongue is complex and comes from both trigeminal (anterior 2/3) and the glossopharyngeal (CN IX) (posterior 1/3). In general, the use of regional anesthesia (and analgesia) for maxillofacial procedures can be divided by anatomical distribution of the trigeminal nerve and the cervical plexus. After originating at the gasserian ganglion, the trigeminal nerve branches into the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves – each providing targets for regional anesthetics. For maxillofacial procedures, V2 and V3 nerves will be targeted more commonly. The superficial portions of the cervical plexus (C2–C4) provide sensation to the posterior region of the head and neck (Figs. 5.3 and 5.4) (see Chap. 2).

Ophthalmic Branch (V1)

The ophthalmic division of the trigeminal nerve divides into the supraorbital, supratrochlear, and nasociliary nerves. The nasociliary branch innervates the sinuses and mucous membrane of the nasal cavity. Regional anesthesia of the ophthalmic division of the trigeminal nerve can be utilized in ophthalmologic, neurologic, and frontal sinus surgery. Ocular injury is a significant risk during blocks of these nerves. For maxillofacial procedures, these nerves will generally not be targeted as commonly as blocks of V2 and V3.

Table 5.2 Sensory innervation of the head and neck

Trigeminal nerve (CN5-V1 V2 V3)	Face, head, tongue
Glossopharyngeal nerve (CN9)	Tongue
Cervical plexus (C2 C3 C4)	Head, mandible, neck

Fig. 5.3 Terminal branches of the trigeminal nerve

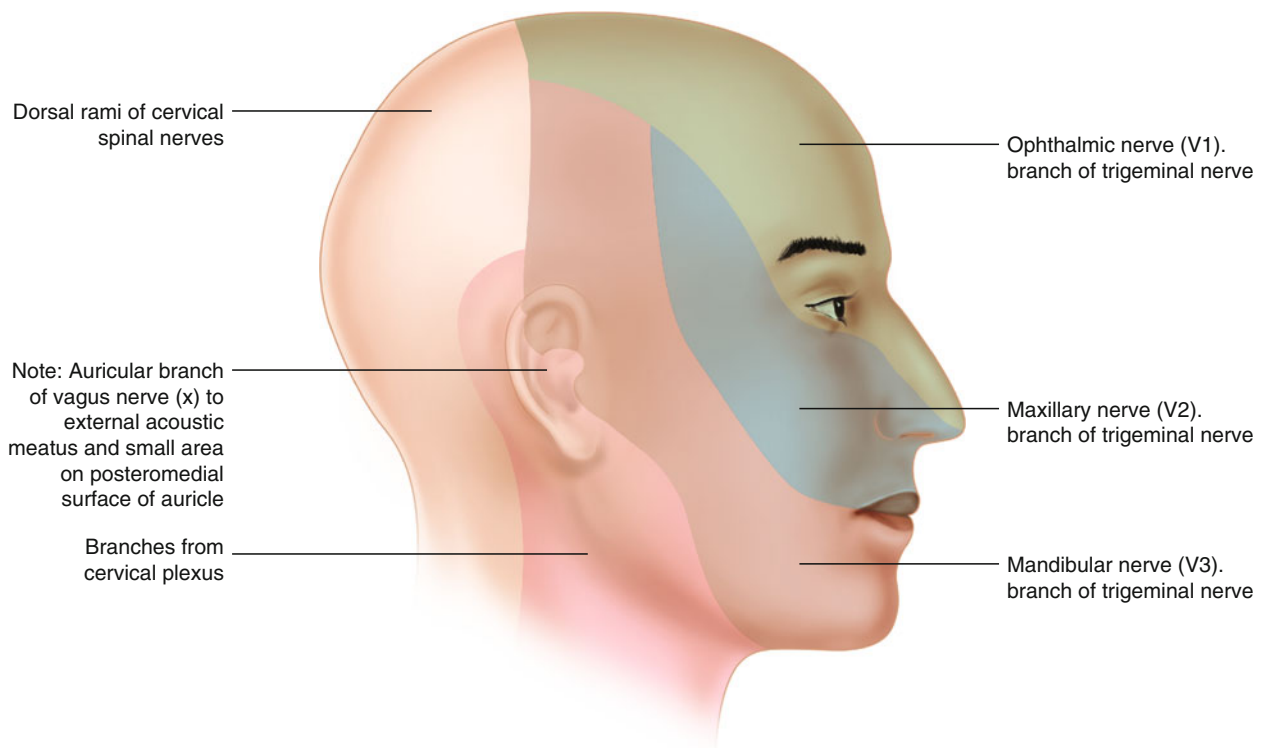
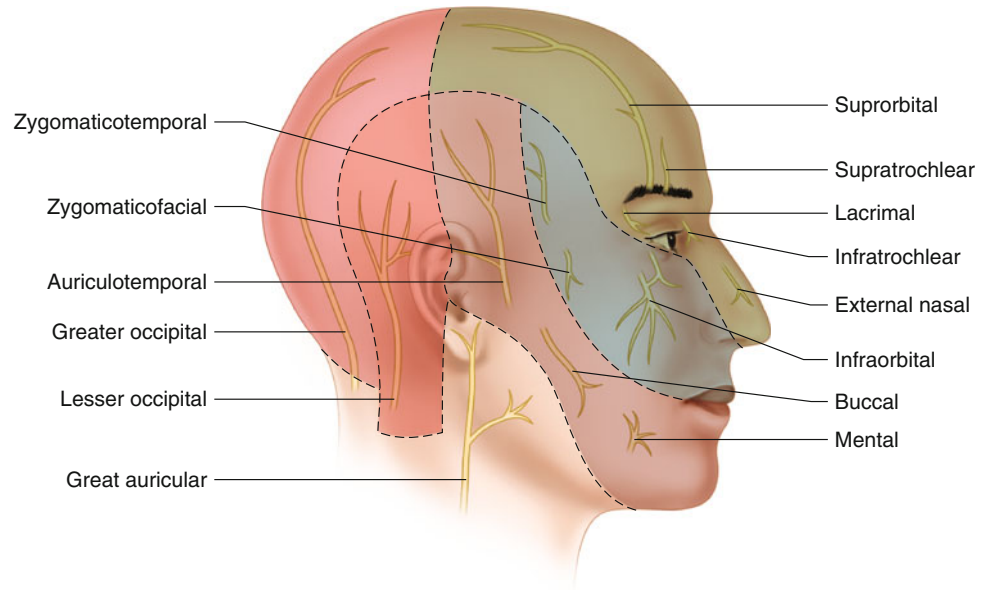


Fig. 5.4 Supraorbital and supratrochlear nerve block distribution

The Frontal Nerve Block

The so-called frontal nerve block can be used to block both the supraorbital and supratrochlear nerves as well as the infratrochlear nerve, which exits a foramen below the trochlea and provides sensation to the medial upper eyelid,

canthus, medial nasal skin, conjunctiva, and lacrimal apparatus (Figs. 5.5 and 5.6). When injecting this area, use the free hand to palpate the orbit and prevent inadvertent injection into the globe. One can block all three nerves by injecting 2–4 ml of local anesthetic solution from the central brow proceeding to the medial brow.

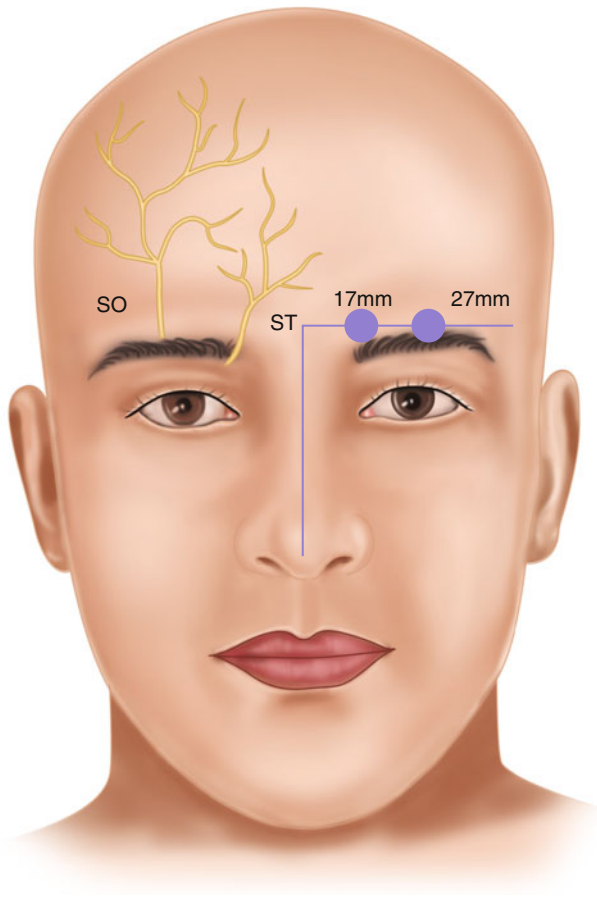


Fig. 5.5 Frontal nerve block. The frontal nerve exits at the superior orbital rim approximately 27 mm lateral to the glabellar midline. This supraorbital notch is readily palpable in most patients. The supratrochlear nerve exits approximately 17 mm from the glabellar midline and supplies sensation to the middle portion of the forehead. *SO* supraorbital nerve, *ST* supratrochlear nerve

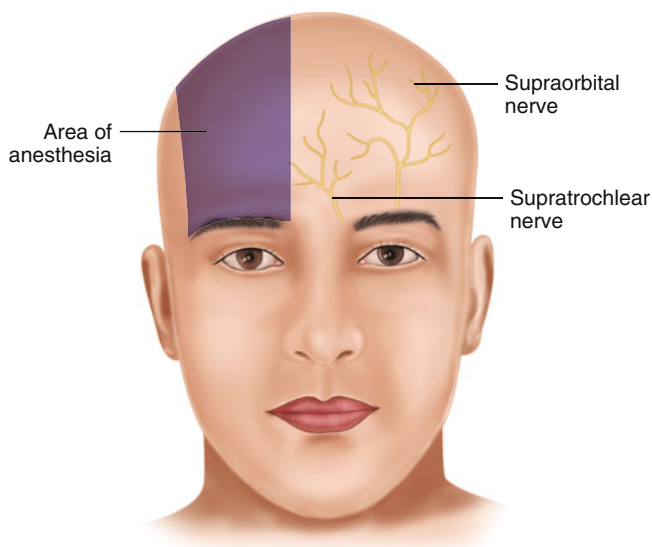


Fig. 5.6 Sensory innervation of the forehead

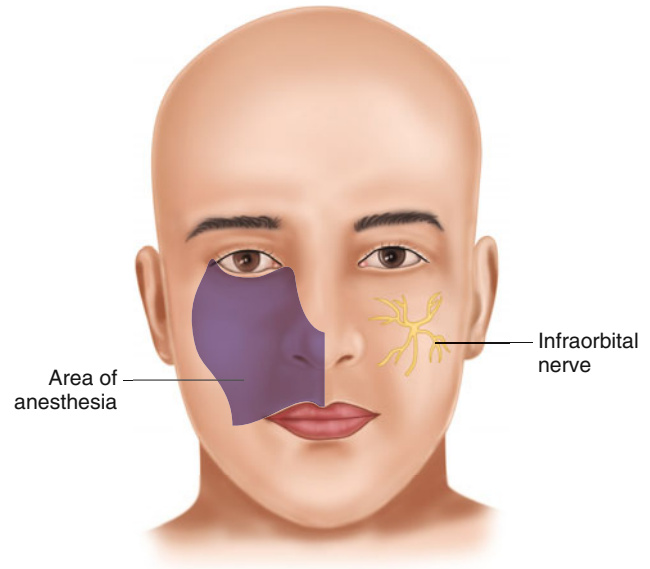


Fig. 5.7 Infraorbital nerve block distribution

Maxillary Branch (V2)

The maxillary division (V2) becomes the infraorbital nerve (IFON) and pterygopalatine branches from V2 travel to the pterygopalatine ganglion or sphenopalatine ganglion (SPG) and the postganglionic fibers form the greater and lesser palatine nerves. The IFON provides innervation to the palate, teeth (5–8 from the right and 9–12 from the left), gums, nasal mucosa, lower eyelid, and upper lip. In combination with the nasociliary branch of the ophthalmic division, the infraorbital nerve also provides sensation to the sinuses and nasal mucosa. The SPG supplies the lacrimal gland, paranasal sinuses, the mucosa of the nasal cavity and pharynx, the gingiva, and the mucous membrane of the hard palate.

The Infraorbital Nerve Block

The infraorbital nerve is a relatively simple nerve to locate and block using regional anesthesia techniques. The infraorbital nerve provides sensation to the cheek, upper lip, eyelid, and lateral aspect of the nose (Fig. 5.7) and has been used successfully for a variety of these structures procedures. Bilateral or unilateral blocks may be used for surgery of the upper lip, nasal fracture reductions, and dental work on maxillary teeth. The infraorbital nerve exits the infraorbital foramen approximately 1 cm below the inferior orbital ridge, palpable with a finger, along a vertical line from the medial limbus of the eye. Three approaches are possible in order to block the infraorbital nerves: a transcutaneous (direct), intra- or transoral, or an intra- or trans-nasal approach.

To locate the nerve, the infraorbital foramen is first palpated approximately 2–3 cm from the midline of the face and

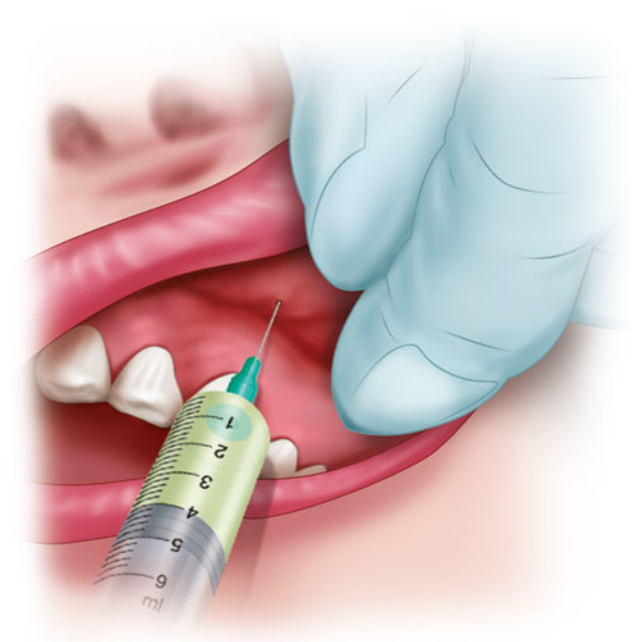


Fig. 5.8 Transoral approach to the infraorbital nerve block

inferior to the orbit. The infraorbital notch can be palpated with a fingertip rolled over the inferior orbital rim. In the direct approach, the needle is placed 0.5–1 cm inferior to the foramen and directed toward the nerve in order to prevent direct passage of the needle through the foramen and into the orbit and globe of the eye. Indeed, ocular injury has been reported and is a potential and severe complication of these blocks.

One to three milliliters of local anesthetic is injected after a negative aspiration for blood or vitreous humor. In the transoral approach, the needle is inserted into the superior buccal groove (between the gums and lip) and directed superolaterally until the tip of the needle is positioned midway between the nasal labial fold and the infraorbital foramen (Fig. 5.8) (We recommend leaving the finger over the foramen for the reasons mentioned previously.)

The trans-nasal approach may be used to block the infraorbital nerve. For the trans-nasal technique, the index finger of the nondominant hand is placed over the infraorbital foramen. During the trans-nasal approach, a 1.5-in. 25-gauge needle is inserted through the nares and tunneled under the skin toward the foramen (Fig. 5.9). After negative aspiration, injection occurs when the tip of the needle is located midway between the nasal labial fold and the infraorbital foramen delineated by the procedures finger. The fingertip is maintained over the foramen throughout the procedure to identify the nerve origin and to prevent the needle from entering the foramen and potentially causing ocular injury. A total of 2 ml of 0.5 % bupivacaine or 0.75 % ropivacaine is injected. This technique should not be performed in patients with pathology such as neoplasms or AV malformations involving the nasal cavity



Fig. 5.9 Trans-nasal approach to the infraorbital nerve block

such as the septum and nasal vestibule. In such cases we perform the procedure using the transoral approach.

The Sphenopalatine Ganglion (Greater Palatine Nerve) Block

Located within the pterygopalatine fossa under a thin layer of connective tissue and mucous membrane, the SPG is traditionally accessed either trans-nasally or transorally. The sphenopalatine ganglion (SPG) originates from the maxillary branch of the trigeminal nerve (V2) and provides sensory innervation to the lateral wall of the nasal cavity and inferoposterior portions of the nasal septum. The greater palatine nerve gives off branches to innervate the inferior nasal cavity mucosa and then exits through the greater palatine foramen to provide sensation to the hard palate. The lesser palatine nerve also provides branches to the nasal cavity mucosa and then travels through the lesser palatine foramen to innervate the soft palate and tonsils.

The sphenopalatine ganglion or pterygopalatine ganglion supplies the lacrimal gland, paranasal sinuses, the mucosa of the nasal cavity and pharynx, the gingiva, and the mucous membranes of the hard palate. The ganglion may be blocked via the greater palatine foramen approach. SPG blockade is currently indicated in the treatment of a variety of painful conditions including cluster headaches, acute migraines, atypical facial pain, and trigeminal neuralgia as well as an analgesic adjunct for major sinonasal and palatal surgeries.



Fig. 5.10 Mac 3-assisted SPG blockade. The SPG block is performed with the practitioner at the head of the bed using gentle anterior pressure to displace the tongue and visualize the hard palate

The trans-nasal approach to the SPG involves use of a cotton-tipped applicator soaked in local anesthetic that is then topically applied through the nasal cavity into the posterior pharynx. This approach is difficult, however, when dealing with inflamed and enlarged nasal mucosa, as is the case with many patients undergoing ESS. The transoral approach requires identification of the greater palatine foramen with subsequent injection of a local anesthetic into the pterygopalatine canal and is considered to be more technically challenging due to difficulty in identification of the appropriate anatomy within the hard palate. The approach involves use of finger palpation to identify the greater palatine foramen, which is located posteromedial to the second or third maxillary molar and anteromedial to the maxillary tuberosity and pterygoid hamulus. Identification simply by finger palpation, however, is not always consistent and varies greatly depending on individual anatomy. A transoral approach using direct visualization with the help of a Macintosh 3 blade or a headlamp with “loops” may also be used as a technique for SPG blockade.

When used in conjunction with a general anesthetic, the block is performed after anesthetic induction and placement of the endotracheal tube (ETT) but prior to surgical incision. With the patient supine, and with neck extension, a Macintosh 3 blade is used in similar fashion as in direct laryngoscopy but without advancing to the vallecula in order to displace the tongue anteriorly (Figs. 5.10 and 5.11). This provides direct exposure to a well-lit hard palate without disrupting the ETT.

The greater palatine foramen is then visualized by identifying a shallow groove located approximately 0.5–1 cm medial to the space between the second and third maxillary

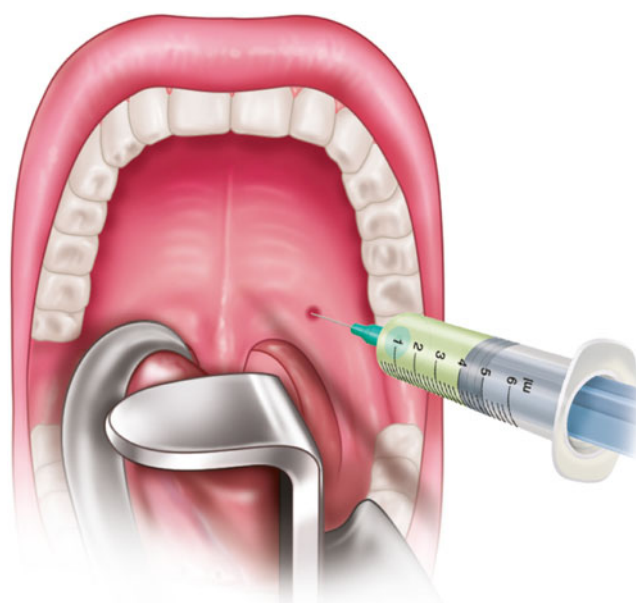


Fig. 5.11 Sphenopalatine ganglion blockade with blanching of hard palate from epinephrine-containing local solution. Correct placement of the SPG block is confirmed by resistance to local anesthetic injection and by blanching of the hard palate due to maxillary artery vasoconstriction with epinephrine

molars. A 25-gauge, 1.5-in. needle bent at a 90° angle 1.5 cm from the tip is used to limit the depth of entrance into the pterygopalatine canal and avoid the theoretical risk of intraorbital penetration. The needle tip, once in the foramen, should feel as though it has “fallen into a space.” The needle should then be advanced no further than the length of the bent portion. If the foramen is not easily visualized and accessed in this manner, the tip of the needle is used to probe through the mucosa in the expected anatomic location until the needle slips easily into the canal, located approximately where the hard palate adjoins the mucosa. We use 1–1.5 ml of 1 % lidocaine with epinephrine 1:100,000 bilaterally for a total of 2–3 ml injected with aspiration throughout needle insertion to avoid intravascular injection. Resistance met during injection of the local anesthetic verifies correct placement of the block within the shallow pterygopalatine canal rather than the nasopharynx. Appropriate placement is further confirmed by blanching of the hard palate on the side that is blocked. The vasoconstriction is due to the spread of the local anesthetic into the pterygopalatine fossa, where the vasoconstricting agent in the anesthetic (i.e., epinephrine) acts on the internal maxillary artery and its terminal branches. This region is highly vascular and significant systemic absorption is minimized by use of epinephrine. In addition, the added vasoconstriction will be helpful in creating a “bloodless” surgical field. Used in combination with general anesthesia, this nerve block helps to provide both local anesthesia and vasoconstriction for the endoscopic sinus surgeon.

It should be noted that this technique has been associated with complications such as intravascular injection, infraorbital nerve injury, and transient diplopia. It should be avoided in cases where pathology may involve the pterygopalatine fossa since inadvertent trauma to the tumor may occur. An alternative option is to infiltrate the region overlying the foramen and not inject into the foramen itself.

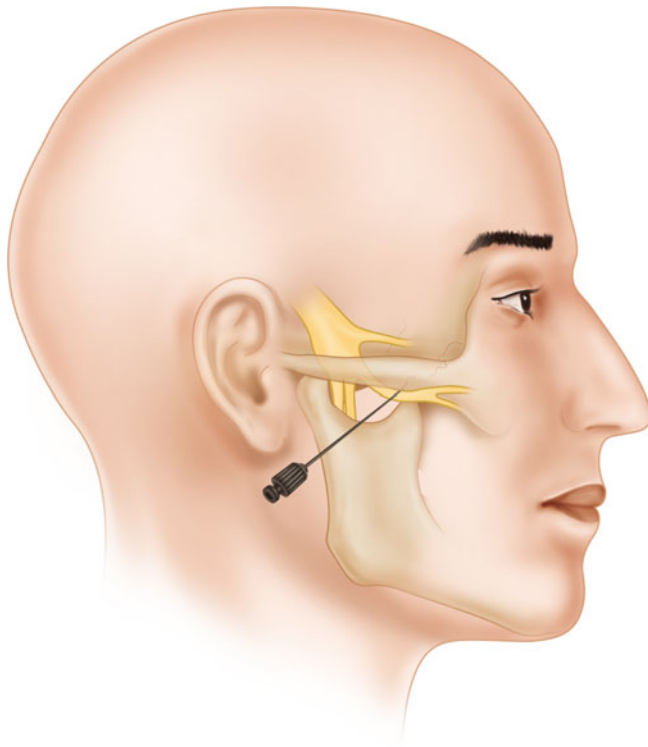


Fig. 5.12 Maxillary nerve block

The Total Maxillary Nerve Block

It is sometimes necessary to block all the divisions of maxillary nerve for extensive surgical procedures. In these cases, the extraoral maxillary nerve block can be used. Under strict aseptic technique, the zygomatic process is located and the depression in its inferior surface is marked. A 1.25-in., 25-gauge needle is inserted perpendicular to the sagittal plane until it touches the lateral pterygoid plate (usually around 3–4 cm deep). The needle is withdrawn and redirected cephalad and anteriorly. After aspiration, 2–3 ml of the local anesthetic solution is injected slowly (Fig. 5.12).

Mandibular Branch (V3)

The mandibular branch (V3) is the largest of the three trigeminal branches and supplies both motor and sensation to the face. V3 exits the skull from the foramen ovale and enters the infratemporal fossa. There it divides into anterior (predominately motor; mastication) and posterior divisions (predominately sensory) (Fig. 5.13). The posterior division divides into three branches: the auriculotemporal nerve, lingual nerve, and inferior alveolar nerve. The auriculotemporal nerve provides sensation from the preauricular area, the lingual nerve provides sensation from the anterior two-thirds of the tongue, and the inferior alveolar nerve provides sensation of the mandibular teeth, buccal mucosa, gingiva, and mandible. The mental nerve, a terminal branch of the inferior alveolar nerve, emerges from the mental foramen and together with input from the facial nerve provides sensory input from the skin of the chin and the mucous membrane of the lower lip. Blockade of the mandibular nerves prior to bilateral man-

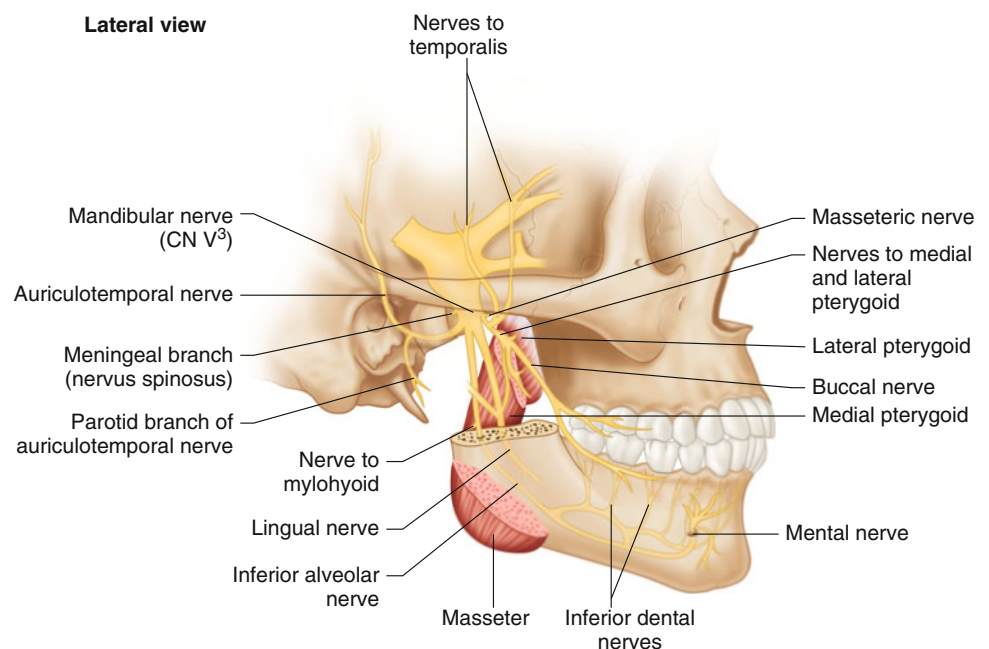


Fig. 5.13 Sensory braches of V3

dibular osteotomy has been shown to effectively decrease total perioperative opioid consumption. A computed tomography (CT)-guided approach to the foramen ovale to block the mandibular nerve in a patient with mandibular deformity caused by segmental mandibulectomy has been successfully performed as an approach to chronic pain therapy.

The Auriculotemporal Nerve Block

The auriculotemporal nerve (ATN) crosses the superior portion of the parotid gland and ascends behind the temporomandibular joint (TMJ), supplying sensation to the skin of the auricle, external auditory meatus, tympanic membrane, temporal region, and temporomandibular joint. Although the sensory innervation of the external ear and surrounding structures is complex and procedures on the ear would require anesthesia of branches from the trigeminal nerve, the vagus, and the cervical plexus, surgery on the TMJ can generally be accomplished under ATN block.

To perform the ATN block, the patient is supine with the head turned to the contralateral side. The periauricular area is sterilized. To identify the location of the ATN, the temporal artery is palpated as it crosses the zygomatic arch. Using a 25–27 gauge needle 2–4 ml of 0.5 % bupivacaine is infiltrated in this area (Fig. 5.14).

The Inferior Alveolar Nerve Block

The inferior alveolar branch of the mandibular nerve descends in the region between the lateral aspect of the sphenomandibular ligament and the medial aspect of the ramus of the mandible. It travels along with, but lateral and posterior to, the lingual nerve. The nerve travels along with the inferior alveolar artery and vein within the mandibular canal and divides into the mental and incisive nerve branches at the mental foramen. The lingual nerve occupies the pterygomandibular space between the medial aspect of the mandible and the lateral aspect of the pterygoid muscle. It then travels deep to the pterygomandibular raphe and terminates deep to the sublingual gland. The lingual nerve provides sensory innervation to the anterior two-thirds of the tongue, mucosa of the floor of the mouth, and lingual gingiva. Due to the proximity of the lingual and inferior alveolar nerves (IAN), both nerves can and will be blocked simultaneously during an IAN block.

The inferior alveolar nerve block is one of the most common nerve blocks for maxillofacial and dental procedures and is routinely used for surgical procedures involving the mandible, all mandibular teeth, the floor of the mouth, the anterior two-thirds of the tongue, the gingivae on the lingual and labial surface of the mandible, and the mucosa and skin of the lower lip and chin. IAN blocks are the most common

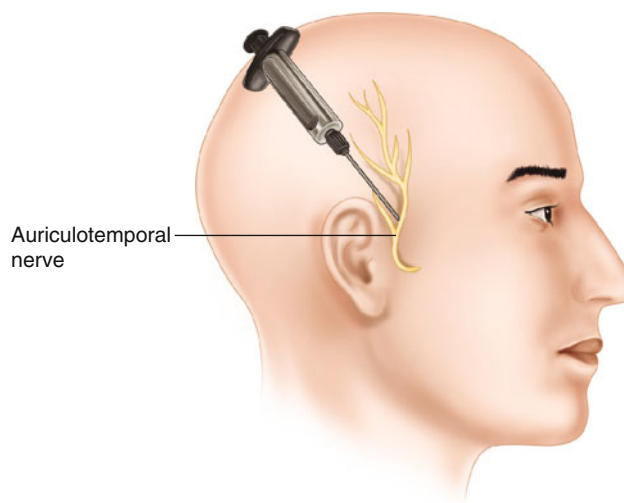


Fig. 5.14 Auriculotemporal nerve blockade

blocks for maxillofacial procedures, yet they carry a high failure rate (10–15 %) compared to maxillary blocks due to nerve root inaccessibility. This block may not provide complete anesthesia and may require supplemental nerve blocks (e.g., the buccal nerve) to increase success. The limited success rate of the standard inferior alveolar nerve block (IANB) has led to the development of alternative approaches to mandibular anesthesia. Although a detailed description of these procedures exceeds the scope of this chapter, the Gow-Gates technique requires that the patient be able to open their mouth widely, while the Akinosi-Vazirani closed-mouth technique (as the name implies) can be used with patients with limited mouth opening, TMJ disorders, or trismus. Both techniques are considered reliable alternatives to the traditional IAN block. Nerve stimulation techniques have also been attempted to decrease the failure rate of these blocks without considerable success. If successful, the IAN block simultaneously blocks the mental, incisive, and lingual nerves.

The target for the IAN block is the mandibular nerve as it travels on the medial aspect of the ramus, prior to its entry into the mandibular foramen. A 25-gauge long needle (1.5–2 in.) in length is used for this technique. The patient is positioned supine and with the mouth open maximally, the coronoid notch and the pterygomandibular raphe are identified. Retracting the cheek, the needle is placed at the injection site from the contralateral premolar region (Fig. 5.15a, b). Local anesthesia can be deposited at the injection site. The needle is advanced until the mandible contacted at approximately 25–35 mm of depth. Once the mandible is contacted, withdraw the needle one millimeter and redirect the needle slightly posterior. After confirming a negative aspirate for blood (a positive aspiration is encountered in approximately 10–15 %), 4 ml of 0.5 % bupivacaine is injected. While withdrawing the needle, an additional 1 ml of bupivacaine should be injected to assure that the lingual nerve is blocked.

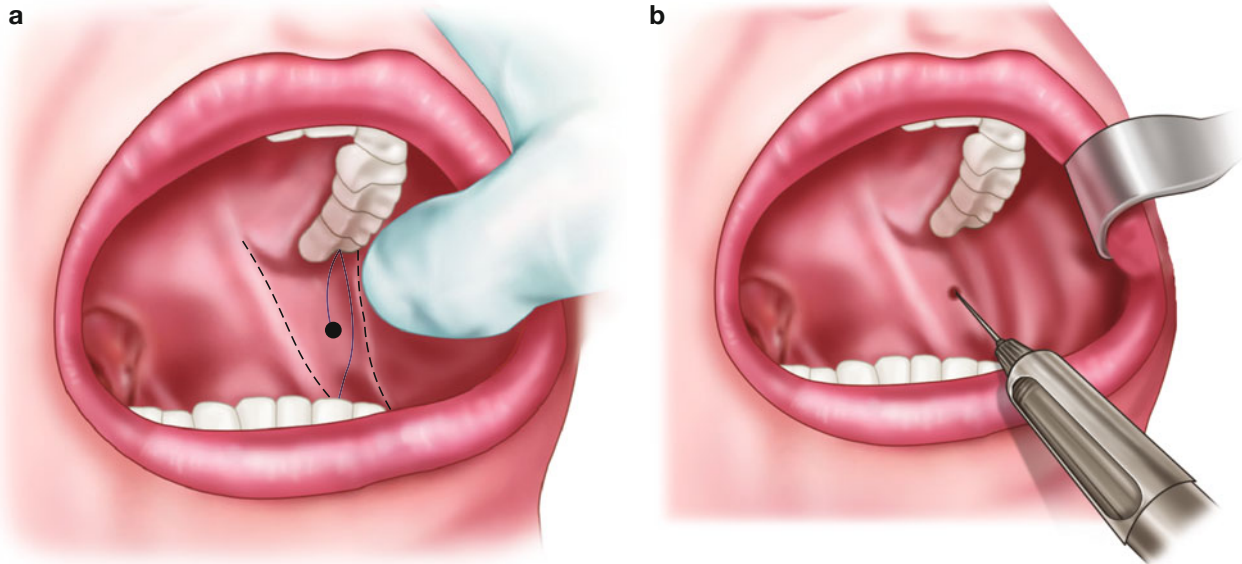


Fig. 5.15 Inferior alveolar nerve blockade. (a) The cheek is retracted to reveal the appropriate injection site. (b) Injection of local anesthesia using a long 25-gauge needle and retractor

The Incisive and Mental Nerve Blocks

Both the mental and incisive nerves are terminal branches of the inferior alveolar nerve and will be blocked with a successful IAN block. One can however block these nerves individually. The incisive nerve provides sensory innervation to the mandibular anterior teeth. The mental nerve emerges from the mental foramen and provides sensory innervation to the skin of the chin and lower lip.

The mental nerve can be blocked either from the external skin or intraorally. To perform the block externally, the mental foramen can be palpated midway between the upper and lower borders of the mandible directly below a vertical line from the pupils (Fig. 5.16a, b). Since the canal of the mental foramen angles medially and inferiorly, a 25-gauge needle is inserted 0.5 cm in depth and placed lateral and superior to the foramen. 2 ml of 0.5% bupivacaine is injected after a negative aspiration. The intraoral approach is accomplished by placing a 25-gauge needle between the two premolar teeth immediately below the tooth root. 2–3 ml of anesthetic is injected after a negative aspiration.

Cervical Plexus

The cervical plexus is comprised of the ventral rami of spinal nerves C1 through C4. Although the plexus is deep to the sternocleidomastoid (SCM), nerves formed from the plexus emerge at the midway point of the sternocleidomastoid and

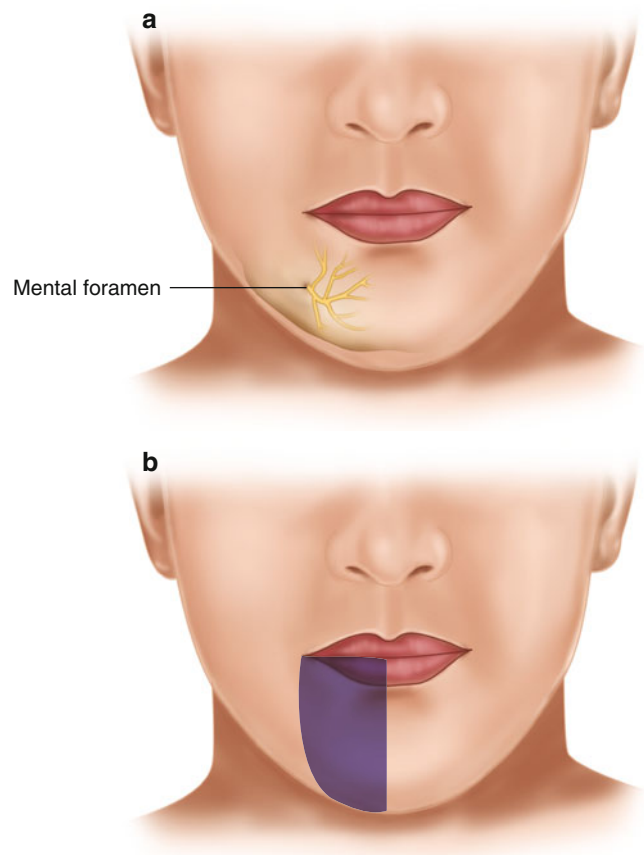


Fig. 5.16 Site for direct mental nerve block (a) and accompanying distribution of anesthesia (b)

supply sensory input from the back of the head and neck. Anesthetizing the nerves as they emerge from behind the (SCM) is known as a superficial cervical plexus block (SCPB). The plexus can also be blocked as the rami exit the vertebral column. This is known as a deep cervical plexus block (DCPB), which is essentially a cervical paravertebral block. Regional blockade of the plexus can provide surgical anesthesia for procedures of the anterior (bilateral block will be required) and lateral neck, jawline, posterior auricular region, and the back of the head including otoplasty, cochlear implant, thyroidectomy, parathyroidectomy, tympanomastoid surgery, and carotid endarterectomy. The use of SCPB and DCPB for postoperative analgesia has also been demonstrated in several studies, while others have dismissed its usefulness. Although many clinicians employ the use of both the deep and superficial block in their practice, bilateral DCPB is contraindicated since bilateral phrenic nerve blockade will result. Care should also be used in patients with significant pulmonary disease who might not tolerate even a unilateral phrenic nerve block. In a review of 69 papers that included nearly 10,000 patients undergoing cervical plexus block for CEA, it was determined that the anesthetic quality was no different when superficial block was used alone compared to a combination of deep and superficial. More importantly the group that had the combined technique required a conversion to general anesthesia and had more complications than those patients where only the superficial block was employed. This might be explained by the increased incidents of phrenic nerve block and the increased potential of intravascular and intrathecal injections. Some no longer employ the use of deep cervical plexus block for either intraoperative anesthesia or postoperative analgesia. For completeness we will describe both blocks below.

The Deep Cervical Block (Cervical Paravertebral Block)

Although our group no longer advocates the use of the deep block because the literature suggests the risks outweigh the benefits, the block, if performed, must only be done unilaterally and used with caution with patients with significant pulmonary disease. The patient is positioned supine with their head turned to the contralateral side at approximately 45–60°. After confirming the surgical site, the following landmarks are identified: the mastoid process and Chassaignac's tubercle (CT) of cervical vertebrae C6 (at the level of the cricoid cartilage). Using a marker, a line is drawn from the mastoid to CT. With a ruler, 2, 4, and 6 cm distances from the mastoid are marked along this line; they

represent the position of the transverse processes of C2, C3, and C4 respectively. Once the landmarks are identified and demarcated, the skin is prepped and draped sterilely. The DCB can be accomplished using three individual injections of 5 ml of local anesthesia (0.5 % bupivacaine, .75 % ropivacaine, or 2 % lidocaine) or a single injection at C4 with 10–15 ml of local anesthesia. Regardless of technique, the needle position and injection technique are the same. A 21-gauge needle with flexible tubing attached to a syringe is employed. The needle is advanced perpendicular to the skin at the injection points. The transverse process should be contacted at approximately 1.5–2 cm in depth. After contact the needle should be withdrawn 1–2 mm, a negative aspiration confirmed and the local injected. This is repeated at each transverse process. If the transverse process is not contacted, the needle should be withdrawn to the skin, redirected 10–15° caudad, and advanced. If failure to make contact still occurs, the reconfirmation of landmarks must be considered. The needle should never be redirected cephalad as this may increase the risk of an intrathecal or intravascular (vertebral artery) injection.

The Superficial Cervical Plexus Block

Superficial cervical blocks can be accomplished bilaterally, unilaterally, while the patient is awake or under general anesthesia. Because the block is accomplished with the deposit of very superficial local anesthesia, the change of traumatizing a major structure (i.e., pneumothorax intraneural injection) or an inadvertent intrathecal or carotid injecting is rare. The patient positioning, landmarks, and site preparation are the same as for the DCB describe above. For this block, it is critical to identify the posterior boarder of the sternocleidomastoid. This is done with the patient awake regardless if the plan is to place the block after the induction of anesthesia. Having the patient lift their head against the practitioner's opposing hand positioned on the patient's fore head easily identifies the contracted muscle. A marker is used to demarcate this border. The mastoid and the level of Chassaignac's tubercle (CT) are also identified. The midway point between the mastoid and CT is marked along the muscle boarder and is where the nerves of the cervical plexus emerge. Ten to 15 ml of local anesthesia placed superficially along this boarder establishes the block (Fig. 5.17).

Although some describe inserting the needle at the midway point and fanning the needle caudad toward the level of CT and cephalad toward the mastoid process, maintaining a superficial needle is important because brachial plexus injury can occur at this level. We recommend alternatively placing the needle at the level of CT and staying very superficial,

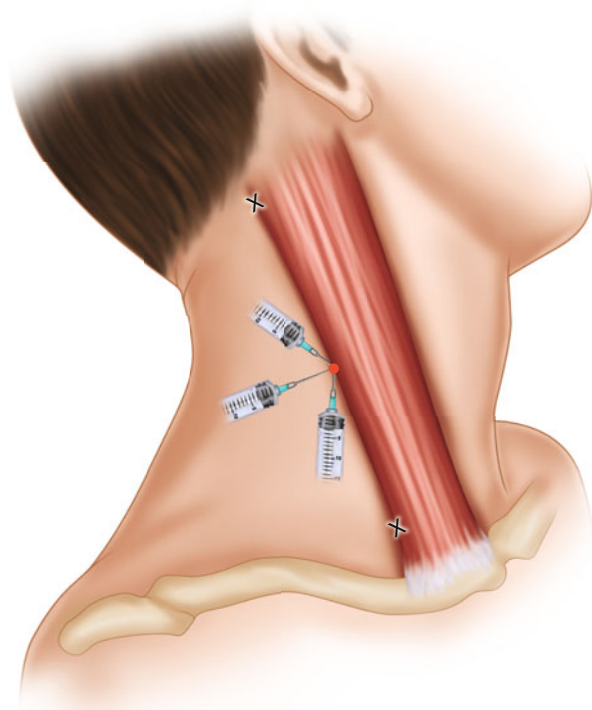


Fig. 5.17 Landmarks and needle fanning for superficial cervical plexus blockade

injecting local anesthesia creating a skin wheal as the needle is advanced toward the mastoid process.

Conclusions

Regional anesthesia for patients undergoing maxillofacial surgery and procedures provides excellent intraoperative surgical conditions as a sole technique or as a component of a balanced anesthetic. Additionally patients benefit from the use of regional anesthetic techniques for acute postoperative pain management. To employ regional techniques for maxillofacial procedures requires a working knowledge of the pharmacodynamics and kinetics of local anesthetics and in depth knowledge of head and neck anatomy.

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