Local Anesthetics and Peripheral Nerve Blocks in the Emergency Department

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Local anesthetics work by blocking the conduction of neural messages. Sodium is mostly an extracellular ion. Closed sodium channels located on the axoplasmic side of the nerve cell prevent its influx into the cell. In this resting state the electrical potential inside a nerve cell is negative in reference to the outside of the cell. This is the resting potential of the nerve cell. After mechanical, chemical, or electrical excitation the sodium channels open and allow sodium ions to move into the cell causing depolarization and propagation of the nerve impulse. In the inactive state, the sodium channels are susceptible to the action of local anesthetic molecules that bind to the channels, causing them to remain inactive and prevent subsequent depolarization [1]. Further conduction by the nerve is blocked until the local anesthetic is displaced from the neural membrane.

Structure of local anesthetics

Local anesthetics are weak bases that require the addition of a hydrochloride salt to be water soluble, facilitating their injection. They usually have an aromatic (hydrophobic) ring structure connected to a tertiary amine (hydrophilic) by an intermediate chain that includes an ester or amide linkage. The chemical composition of local anesthetics determines their potency, duration, and onset of action while the nature of the intermediate chain establishes the two classes of local anesthetics: esters and amides (Table 1). Lipid solubility determines potency [2]. The more lipophilic a local anesthetic is, the more easily it penetrates the nerve cell membranes, resulting in more effective blockade of the neural signal. The plasma protein binding

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potential determines the duration of action of the local anesthetic molecule [3], presumably because the local anesthetic receptor is also a protein [4]. Those with more protein binding remain associated with the neural membrane for a longer period of time—making it unavailable for metabolism and clearance. For example, procaine is poorly protein bound, and its duration of neural blockade is relatively short. On the other hand, bupivacaine is highly protein bound, and consequently, has a long duration of action [1]. Last, pKₐ determines the speed of onset of neural blockade. The pKₐ is the pH at which equal percentages of the drug exist in the ionized and nonionized forms. Local anesthetics are weak bases. They therefore tend to become positively charged as the pH of the local milieu declines. Only the nonionized base is able to pass easily through the neural membrane. Local anesthetics with pKₐ values closer to the physiologic pH produce higher concentrations of this nonionized base. This equates to a more rapid onset of action. This is why the local anesthetics are less effective in an acidic environment, and why alkalinization speeds onset of action and increases duration. Furthermore, epinephrine is unstable in alkaline environments. Commercially prepared solutions containing it are therefore made more acidic, further increasing the time of onset. Once inside the nerve cell, the nonionized base reaches equilibrium with its ionized form. It is this ionized form (cation) that actually binds to the receptor within the sodium channel and inactivates it.

Toxicity

All local anesthetics have a similar toxic profile predominantly affecting the central nervous and cardiovascular systems. Toxicity is related to the
potency of the local anesthetic, total dose delivered, systemic absorption, protein binding, and metabolism and excretion. Of note, most toxic reactions are either dose related or due to inadvertent intraarterial administration.

The rate of systemic absorption of local anesthetics is proportional to the vascularity of the injection site. The absorption rates from highest to lowest are: intercostal > intratracheal > epidural/caudal > brachial plexus > mucosal > distal peripheral nerve > subcutaneous. For this reason, the recommended local anesthetic dose for intercostal blocks is one tenth the maximum for peripheral blocks. Absorption of local anesthetic is slower for those that are highly tissue bound.

The metabolism and excretion of local anesthetics differ between esters and amides. Ester local anesthetics are quickly metabolized by plasma pseudocholinesterase, and the water-soluble metabolites are excreted in the urine. Patients with pseudocholinesterase deficiency (eg, sensitivity to succinylcholine, patients taking cholinesterase inhibitors, and patients with myasthenia gravis) are at an increased risk for systemic toxicity. Of note, cocaine undergoes partial hepatic metabolism and the remainder is excreted into the urine unchanged [4]. Amide local anesthetics are metabolized in the liver. Although the rate of metabolism depends on the agent, the overall time for metabolism is slower than for ester hydrolysis. Consequently, there is a smaller allowance for cumulative dosing over time with amide anesthetics when compared with the esters. Furthermore, decreases in hepatic function or liver blood flow will reduce the metabolic rate and predispose to systemic toxicity. Metabolites of prilocaine (o-toluidine derivatives), a component of EMLA cream, may accumulate after high doses of the drug, and can convert hemoglobin to methemoglobin. Benzocaine may also cause methemoglobinemia.

Central nervous system toxicity

The central nervous system (CNS) toxic effects of local anesthetics are directly related to lipid solubility [1]. As the lipid solubility of a local anesthetic increases, the gap between the wanted clinical effect and systemic toxicity narrows. This is why bupivacaine has more risk for systemic toxicity than lidocaine. Furthermore, local anesthetics that exhibit strong protein binding cross the blood–brain barrier more effectively as they remain in the nonionic state more often.

Symptoms of CNS toxicity range from lightheadedness, tongue numbness, metallic taste, and restlessness at low levels, to perioral paresthesias, slurred speech, and excitability or drowsiness at higher levels. Seizures and coma are manifestations of severe toxicity. The CNS effects are believed to be due to the depression of inhibitory neurons, leaving the excitatory pathways unopposed [5]. Treatment of local anesthetic-induced seizures is with benzodiazepines, which raise the seizure threshold. One caveat to this treatment is benzodiazepines, which, like certain local anesthetics, are highly
protein bound in the plasma. Intravenous administration of benzodiazepine can compound toxicity by displacing the anesthetic from plasma proteins, contributing to the free, active form [6]. An alternate anticonvulsant is thiopental 0.5 to 2 mg/kg. However, the duration of action is shorter with thiopental, and it may require repeat dosing should the seizures reoccur. Careful attention should be given to oxygenation and ventilation as hypoxia, hypercarbia, and acidosis worsen the toxicity of local anesthetics.

**Cardiac toxicity**

Cardiac toxicity occurs secondary to blockade of the sodium channels in the cardiac conduction system [1]. The resultant increased activity in reentrant pathways and reduction of the refractory period predisposes the heart to ventricular dysrhythmias. At higher plasma concentrations, myocardial contractility is depressed. Smooth muscle dilation may cause hypotension. Symptoms of cardiac toxicity include palpitations, cardiac dysrhythmias, hyper- or hypotension and cardiovascular collapse [7]. It usually occurs after the stimulant phase of CNS toxicity. Cardiac toxicity may result from any of the local anesthetics; however, it is more likely in the more lipophilic agents such as etidocaine and bupivacaine. Cardiac toxicity is worsened by the use of epinephrine [3], hypoxia, hypercarbia, and acidosis.

Bupivacaine is notorious for cardiac toxicity, often associated with accidental intravascular injection. A range of effects including hypotension, atrioventricular block, and dysrhythmias has occurred. The proposed reason for its increased cardiotoxicity when compared with other agents includes its higher degree of depolarization changes and high degree of protein binding.

Management of cardiovascular collapse from local anesthetic toxicity should follow advanced cardiac life support (ACLS) guidelines, with a few notable exceptions. First, as lidocaine is a local anesthetic, its use should be avoided. Animal models suggest increased benefit with high-dose epinephrine to treat bupivacaine-induced cardiac toxicity, although this is controversial. Bretylium, 5 mg/kg repeated up to 30 mg/kg, can occasionally convert ventricular tachyarrhythmias to normal sinus rhythm, or at least facilitate electrical cardioversion. Unfortunately, the effects of Bretylium may take 30 minutes to occur, so prolonged resuscitation is needed [8].

**Allergic reactions**

True allergy to local anesthetics is rare. A careful history is paramount to discriminate the true allergic reactions. Many times what the patient reports as an allergy was actually an uncomfortable drug effect, vagal reaction, or intraarterial injection of the local anesthetic or epinephrine. True allergic reactions will usually produce some form of skin or upper airway involvement. When an allergic reaction does occur it is usually due to the
metabolite \textit{para}-aminobenzoic acid (PABA) in ester anesthetics and the preservative methylparaben (MPB) in amide anesthetics. If a true allergy is suspected, based on history or documentation, a preservative-free agent from the other class should be used. Most true allergic reactions are due to the esters. Therefore, if in doubt as to the class of local anesthetic that caused the previous allergic reaction, it is safest to administer a preservative-free amide local anesthetic (e.g., cardiac lidocaine). The risk of a hypersensitivity reaction to this agent is extremely low.

Alternatively, agents such as saline with benzyl alcohol, benzyl alcohol with epinephrine or 0.5\% to 1\% diphenhydramine may be used. Benzyl alcohol is the preferred agent, as initial studies suggest it is the most effective alternative \cite{9}. Diphenhydramine has been shown to cause severe pain on injection, prolonged analgesia, and prolonged rebound hyperesthesia \cite{10}. Furthermore, diphenhydramine carries the additional risk of local irritation and necrosis of the skin when used in the fingertips or areas supplied by the end arteries \cite{11}.

**Topical anesthetics**

\textit{Overview}

Topical anesthetics began to be used in the latter half of the 19th century with the advent of cocaine. It took almost another century for more effective and safer topical anesthetic agents to become available. A laceration repair no longer requires a painful injection for anesthetic delivery. An injection may be avoided altogether, or performed painlessly through an area treated with a topical anesthetic. Additionally, topical anesthesia avoids the wound edge distortion that subcutaneous infiltration may produce. Topical anesthesia can be divided into three main categories of use: mucosal membranes, nonintact skin, and intact skin.

**Topical anesthetics for mucosal membranes**

Cocaine and lidocaine are two agents that may used to numb the mucosa of the nose, mouth, and pharynx. Cocaine is a unique anesthetic in that it has intrinsic vasoconstrictor properties, making it useful both intranasally and during surgery to reduce blood loss. Cocaine in a 4\% solution provides rapid and effective anesthesia for approximately 45 minutes. The maximum dose given should be less than 3 mg/kg. Unfortunately, cocaine has some disadvantages. Due to its potential for coronary artery vasoconstriction, hypertension, and tachycardia it should not be used in patients with known or suspected coronary artery disease. In addition to concerns of systemic toxicity, cocaine’s expense and federal regulatory issues make it a somewhat unattractive agent.
Lidocaine provides adequate anesthesia in this region when used in the 1% to 4% concentration. Although it does not have any intrinsic vasoconstrictor properties, epinephrine or phenylephrine can be added for this effect. Topical application of lidocaine results in a high level of absorption, so caution must be used to avoid exceeding the maximum weight-based dose. To minimize error, it is best to calculate the maximum dose based (5 mg/kg of lidocaine without epinephrine or phenylephrine, 7 mg/kg of lidocaine with epinephrine or phenylephrine) before administering the anesthetic. To determine the amount of anesthetic in a given volume, simply multiply the concentration of the anesthetic by 10 to determine the milligrams of anesthetic in 1 mL of solution. For example, 2 mL of 4% viscous lidocaine contains 80 mg of lidocaine. More dilute solutions may be used in situations requiring high volumes.

Several alternatives are available for applying local anesthetic to mucous membranes. An atomizer can be used to create a fine mist. Lidocaine jelly can be rubbed between the cheek and gums. Cotton swabs or pledgets can be soaked in the solution and introduced into the nose to either numb the entire mucosal surface or a selective area. A vaporizer, such as one might use for albuterol, is effective in anesthetizing the entire oropharynx, often including the vocal cords [12]. The measured amount of lidocaine is placed into the vaporizer and the patient then inhales the mist produced.

Topical anesthetics for intact skin

There are three choices available for topical anesthesia to intact skin: Eutectic mixture of local anesthetics (EMLA), ELA-Max, and iontophoretic preparations. EMLA contains a mixture of 2.5% lidocaine and 2.5% prilocaine in a 1:1 ratio by weight. Each gram of cream contains 25 mg of lidocaine and 25 mg of prilocaine, purified water, carboxypolymethylene (a thickening agent), and sodium hydroxide (to adjust the pH to 9.4). The suggested dose is 2.5 g on 20 to 25 cm² of skin, with a maximum dosage of 2 g on 10 cm² of skin. The Federal Drug Administration (FDA) approved EMLA cream in 1992 for use on intact skin; however, more recent studies suggest EMLA might be effective and safe for use in lacerations [13]. Further studies are needed before EMLA can be recommended for nonintact skin, due to its high cost (approximately $5.00 per application) and prolonged onset of action.

EMLA has a slow onset of action, and takes at least 45 to 60 minutes to achieve anesthesia. EMLA should be applied to intact skin for at least 1 hour but not longer than 2 hours before the planned procedure. Once it has been applied, it should be covered with an occlusive dressing. Studies have shown anesthetic depth of 3 mm after a 60-minute application and 5 mm after a 120-minute application [14]. Anesthesia lasts for 1 to 2 hours. Advantages of EMLA are its ease and comfort of use and benign safety profile. Complications of EMLA include blanching of skin, redness at the
application site, and contact dermatitis. In infants less than 3 months of age there is a theoretical risk of methemoglobinemia due to inadequate levels of methemoglobin reductase. For this reason EMLA should not be used in patients less than 3 months old. However, at least one meta-analysis has demonstrated no increased risk of methemoglobinemia when EMLA cream was used in neonates. Although toxicity from lidocaine and prilocaine is possible, it should not occur if EMLA is used as directed. It can be used in the emergency department for venipuncture, arterial puncture, lumbar puncture, and arthrocentesis, yet its slow onset of action makes it impractical for the large majority of emergency medicine patients.

ELA-Max is a recent addition to the topical anesthetics. It contains 4% lidocaine cream in a liposomal matrix and is FDA approved for the temporary relief of pain resulting from minor cuts and abrasions. Liposomes enhance the action of the lidocaine by facilitating the rate and extent of its absorption. Consequently, ELA-Max achieves adequate anesthesia in only 30 minutes, as opposed to EMLA’s 60-minute onset of action [15,16]. An occlusive dressing does not need to be applied over ELA-Max, but it may be beneficial in preventing active children from displacing the ELA-Max. In limited trials it appears to be a safe topical anesthetic. ELA-Max does not contain prilocaine or its metabolites that have recently been implicated in EMLA-induced methemoglobinemia in older children. Thus, ELA-Max theoretically should not increase the risk of methemoglobinemia—even in young infants.

Another method for dermal anesthesia of intact skin is lidocaine iontophoresis. Iontophoresis allows the introduction of soluble, positively charged lidocaine hydrochloride into the skin by the use of a small external electrical current. In recent studies, this technique appears to be safe and efficacious [17], with an insignificant systemic absorption of lidocaine [18–20]. Lidocaine iontophoresis achieves anesthesia in 10 to 20 minutes, making it a more rapid alternative to EMLA cream. Undesirable effects of this delivery method include the sensation of electrical current flowing through the skin, and temporary erythema, blanching, itching, and urticaria at the application site. Another disadvantage of this technique is only one site can be anesthetized at one time. Last, an iontophoresis device costs approximately $400, and the one-time use disposable electrodes are $6 to $7.50 each, making this slightly more expensive than EMLA. However, the time saved by using iontophoresis may outweigh the slightly higher costs of its use.

Topical anesthetics for nonintact skin

Two primary choices exist to painlessly provide anesthesia over nonintact skin: TAC and LET. TAC was introduced in 1980, and is composed of tetracaine 0.5%, adrenaline 0.05%, and cocaine 11.8%. Five to 10 mL of TAC is applied to an open wound and covered with gauze. The gauze should be held in place for 10 to 20 minutes with either tape or a gloved hand to
prevent inadvertent absorption of the medication by a well-intentioned parent. TAC works best on the scalp and face, but it can be used on extremities. However, due to its adrenaline content, it should not be used in areas with end arteries. Use of TAC on or near mucous membranes should be avoided to prevent toxicity from the systemic absorption of tetracaine and cocaine. There are significant concerns over the use of TAC due to reported cases of respiratory arrest, seizures, and death in children due to its improper application leading to increased systemic absorption [21]. Other drawbacks to TAC include its high cost, potential for abuse and the regulatory issues involved with dispensing and administering a cocaine-containing medication. These problems have led most emergency departments to abandon its use.

LET (lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5%) is an excellent alternative agent to TAC. LET is as effective as TAC [22–24], possesses a superior safety record, is more cost effective, and does not contain a controlled substance. LET comes in both a solution and gel form. They are equally efficacious [25], although the gel may be easier to apply with less run off. One to 3 mL of LET should be applied directly to the wound and then covered for 15 to 30 minutes. Like TAC, LET should not be applied directly to or near mucous membranes, the pinna of the ear, the nose, penis, fingers, or toes.

**Local anesthetics used for subcutaneous infiltration**

**General**

Local anesthetics are classified into two groups: amides and esters (Table 1). Certain key differences exist between the two broad classes, especially with regard to metabolism and allergic potential. Esters are hydrolyzed in the plasma by pseudocholinesterase, a much more rapid process than the hepatic metabolism of the amides. The slower metabolism of the amides increases the possibility of toxicity, especially in a patient with liver dysfunction. The breakdown of the ester compounds by pseudocholinesterase produces para-aminobenzoic acid, a potentially allergenic substance. Because metabolism of the amide compounds does not result in this byproduct, most allergic reactions to the local anesthetics are due to the esters.

**Lidocaine**

Lidocaine (Xylocaine) is the most commonly used local anesthetic for intradermal infiltration. This is most likely due to its excellent clinical efficacy and good safety record. It is prepared commercially as a 1.0 to 2.0% solution with a pH of 6.5 in solution and a pKₐ of 7.9. Onset of action usually begins 4 to 7 minutes after injection. The maximum dose is 3 to 5 mg/kg, up to 300 mg in a single dose. The addition of epinephrine prolongs its duration of action and decreases its systemic absorption, allowing a maximal dose of 7 mg/kg. A 1% solution without epinephrine has an average duration of action of
approximately 1.5 hours. The duration is extended to approximately 3.5 hours with epinephrine. Traditionally, it was taught that epinephrine should never be used in the hands or feet due to the risk of ischemia from significant constriction of end arterioles. However, a few citations in the recent literature suggest that, with careful screening, epinephrine may be used in the hands and feet on a limited basis [26,27]. Given the preponderance of data that states otherwise, it would be prudent to avoid using epinephrine in the digits, and to use it in the hands and feet only very selectively until more data is available [28]. To reverse digital artery vasospasm due to intentional or inadvertent local injection of epinephrine locally inject 2 mg of phentolamine.

**Bupivacaine**

Bupivacaine (Marcaine) is a very potent local anesthetic with an anesthesia equivalent to lidocaine. Due to its high pKₐ and protein binding, its onset of action may take up to 20 minutes, with duration of action of 3 to 6 hours. It is clinically available as either a 0.25% or 0.5% solution. Maximal doses of bupivacaine are 2 to 2.5 mg/kg without epinephrine and 3 to 3.5 mg/kg with epinephrine. A dose of bupivacaine may be repeated every 3 hours, but the total dose in a 24-hour period should not exceed 400 mg. Although bupivacaine is well suited for longer procedures, it should be noted that the risk of systemic toxicity with bupivacaine is much higher than most other local anesthetics due to its high potency and protein binding. The toxic potential of bupivacaine has led to the development of newer agents such as ropivacaine and levobupivacaine.

**Ropivacaine (Naropin)**

Ropivacaine is a new amide local anesthetic approved by the FDA in 1996. It is very similar to bupivacaine in terms of potency, onset time, and duration of action, but it is 70% less likely to cause the cardiotoxic effects associated with bupivacaine. It does have some intrinsic vasoconstrictive properties, so it probably should not be used on end arterial areas. It costs eight times more than bupivacaine [29].

**Levobupivacaine**

Levobupivacaine is the S-isomer of bupivacaine. This pure S-enantiomer has fewer cardiovascular and CNS side effects when compared with its racemic cousin bupivacaine. The potency of the two drugs is similar. It costs five times more than bupivacaine [30].

**Mepivacaine**

Mepivacaine (Carbocaine) is structurally similar to lidocaine. Its onset of action is similar to lidocaine, but its duration of action is much longer
(3 hours). Epinephrine increases the duration of action by 20% to 30%. It is prepared for local infiltration as a 0.5% or 1.0% solution. Despite its quick onset and longer duration of action, mepivacaine’s reputation for being more toxic than lidocaine has limited its clinical use.

Reducing the pain of injection of local anesthetics

Many techniques may be employed to lessen the degree of pain from the injection of a local anesthetic. The use of a small needle (27–30 gauge) and slow administration of the anesthetic (30 seconds per mL) [31] in a proximal to distal direction tend to lessen the pain of injection. Infiltration of local anesthetics inside the cut wound edge is less painful than injection through intact skin [32].

Many studies have shown that buffering of lidocaine reduces the pain of infiltration [33–37]. To make a buffered lidocaine solution, add sodium bicarbonate (44 mEq/50 mL) to lidocaine in a 1:10 ration (1 mL of bicarbonate is added to 10 mL of lidocaine). Buffering of lidocaine decreases its shelf life to 7 days because buffered lidocaine undergoes biodegradation at room temperature. The limiting factor in the addition of sodium bicarbonate to local anesthetic solutions is the tendency for the more lipid-soluble agents to precipitate. For example, mixing sodium bicarbonate and bupivacaine in a 1:10 ratio causes precipitation. Therefore, bupivacaine is buffered in a 1:50 ratio (0.1 mL of bicarbonate is added to 5 mL of bupivacaine).

The literature is equivocal on whether warming an anesthetic will reduce the pain of administration. What is reasonable is to ensure the anesthetic is at least room temperature. Lidocaine can be warmed in either dry heat, such as a blanket warmer, or in temperature-regulated water baths at 37°C. If the lidocaine has not been buffered, it will not suffer degradation in shelf life with heating.

Addition of vasoconstrictors to local anesthetics

The addition of a vasoconstrictor, such as epinephrine, offers several advantages to most local anesthetics. First, a vasoconstrictor will decrease local blood flow, allowing for better hemostasis in the surgical field. This is beneficial, as local anesthetics, with the exceptions of cocaine and ropivacaine, are vasodilators by a direct action on smooth muscle vessel walls causing relaxation. Vasoconstriction also decreases the rate of systemic absorption of the local anesthetic. This allows an increased maximum dose of local anesthetic to be administered while reducing the risk of systemic toxicity. Last, by decreasing local blood flow metabolism and redistribution of the local anesthetic, the duration of action is prolonged.

Commercially prepared solutions of lidocaine or bupivacaine with epinephrine in concentrations of 1:100,000 (10 μg/mL epinephrine) to
1:200,000 (5 μg/mL epinephrine) are available and widely used for subcutaneous infiltration. These preparations are acidified to a pH of 3 to 4.5 to stabilize the epinephrine component. Therefore, these solutions will have a slower onset of action and be associated with more pain on injection.

**Regional anesthesia**

*General indications and contraindications*

The general indication for regional anesthesia is pain, in an area amenable to a blockade of a specific peripheral nerve (or group of nerves) in a clinical situation that nerve block provides an advantage over other techniques. In reality, the route of anesthesia chosen is a decision made between the physician and the patient. It often involves a variety of factors including patient preference, physician comfort with the techniques involved, and the time constraints of a busy emergency department. As always, the risks and benefits of the proposed procedure(s) should be explained to the patient, who may then make a decision based on informed consent. Indications for the individual nerve blocks are contained within their respective sections below.

Contraindications to the specific regional anesthetic techniques are discussed in their sections below. In general, nerve blocks are contraindicated in uncooperative patients and patients who are unable to communicate severe pain on injection (e.g., those patients suffering from dementia or psychosis and children). Severe pain is often an indicator of intraneural injection, which may produce ischemic nerve injury. Infection directly overlying the injection site, distortion of the anatomical landmarks, and allergy to the local anesthetic being used are also contraindications [38].

*Advantages*

In the emergency department a variety of methods exist to reduce the discomfort of painful conditions procedures. Peripheral nerve blocks are just one of these techniques. Considered as a group, they have multiple benefits over local infiltration anesthesia. Often less local anesthetic may be used leading to a lower risk of systemic toxicity. They are often less painful to perform than subcutaneous injection and result in less anxiety for the patient. In regions with great cosmetic significance (such as the lip and face), a peripheral nerve block avoids the tissue distortion produced by directly injecting a local anesthetic into the wound margins.

*Disadvantages*

Regional anesthesia requires a high degree of patient cooperation, especially in areas where success is guided by a patient’s ability to detect subtle paresthesias. There is a risk of systemic toxicity (approximately 7.5 per
10,000), especially due to inadvertent intravascular injection, and a small risk of peripheral nerve damage (1.9 per 10,000) [39]. Peripheral nerve blockade is a “blind” technique, as it is most often performed in the emergency department without the benefit of a nerve stimulator. This results in a risk of failure that is probably closely related to the clinical experience of the practitioner performing the block. For this reason, the patient should be informed before the procedure of a possible need for additional anesthesia.

**General techniques**

Due to the risk of systemic toxicity, the majority of peripheral nerve blocks should be performed in an area with adequate monitoring and resuscitation equipment. Immediate toxicity is often due to accidental intravascular injection of the local anesthetic. Careful aspiration of the syringe before injection and the use of the “immobile needle” technique (intravenous tubing between the needle and syringe allows the operator to use both hands for needle stabilization and anatomic palpation while an assistant aspirates and injects the syringe) may reduce this risk. Delayed toxicity occurs when an excessive amount of local anesthetic is absorbed into the systemic circulation. The timing of this event depends on the anesthetic agent used and the area being injected. A high index of suspicion is required to diagnose the first signs of both immediate and delayed toxicity.

The patient may be premedicated with a systemic benzodiazepine or opioid to reduce the anxiety and pain of injection. However, the sedation must be light to facilitate patient cooperation.

The skin should be prepared in an antiseptic fashion before needle insertion. Povidone-iodine (Betadine) is one acceptable agent. Preparation of the oral mucosa before an intraoral procedure is discussed below.

The majority of the procedures described herein are “blind” techniques, and knowledge of the local anatomy must be combined with patient cooperation to facilitate a successful block. When a needle comes into contact with a peripheral nerve, it produces a paresthesia in that nerve’s sensory distribution. For a successful block to occur, the local anesthetic must be injected in proximity to the nerve (perineural) but not directly within it (intraneural), as intraneural injection may produce ischemic nerve injury secondary to the high pressures it produces. The intensity of the pain and its duration serve as clues to the injection site of the anesthetic agent. Perineural injection causes a brief increase in the paresthesia, whereas intraneural injection produces an intense and prolonged pain. If intraneural positioning is suspected, the injection should be terminated immediately and the needle repositioned [38].

**Wrist blocks**

For many injuries to the hand, local infiltration or a digital block may be more effective than a wrist block. Wrist blocks have a variable and
possibly slow time to onset and can be time-consuming to perform if all three nerves must be blocked. Examples of situations that are amenable to a wrist block include: “road rash,” thermal burns requiring debridement, hydrofluoric acid burns, injury to more than one finger, and lacerations to the palm.

**Radial nerve**

To begin, identify the radial artery and radial styloid. At the level of the styloid inject 3 mL of local anesthetic just lateral to the radial artery. From this point proceed dorsally and subcutaneously inject local anesthetic along the dorsum of the wrist until the dorsal midline is reached. Approximately 5 mL of local anesthetic is required [40].

**Ulnar nerve**

The ulnar nerve lies deep to the flexor carpi ulnaris tendon at the level of the proximal palmar crease. It runs into the wrist alongside the ulnar artery. The flexor carpi ulnaris tendon is found just proximal to the pisiform bone when the wrist is flexed against resistance. At the proximal palmar crease the artery and nerve both run on the radial side of the tendon, but the nerve lies deep to the artery. This makes the volar approach difficult. The lateral approach is therefore recommended [41]. A 25-gauge needle is inserted at the proximal palmar crease on the ulnar side of the wrist. It is advanced 1.0 to 1.5 cm in a horizontal plane under the flexor carpi ulnaris. Three to 5 mL of local anesthetic is injected. Cutaneous nerves branch off the ulnar nerve, wrap around the wrist, and supply the dorsum of the hand. These may be blocked by injecting 5 to 6 mL of local anesthetic subcutaneously from the lateral border of the flexor carpi ulnaris tendon to the dorsal midline. The same injection site may be used, which may minimize the pain of injection [41].

**Median nerve**

The median nerve is located between the flexor carpi radialis tendon and palmaris longus tendon at the level of the proximal volar crease. In 20% of the population no palmaris longus tendon is present. In these patients the median nerve is found 1 cm ulnar to the flexor carpi radialis tendon. Insert a 25-gauge needle vertically through the skin and penetrate the deep fascia of the flexor retinaculum at a depth of approximately 1 cm. A slight “pop” should be appreciated and a paresthesia elicited. Inject 5 to 7 mL of local anesthetic just radial to the palmaris longus tendon. If the deep fascia has been penetrated a cutaneous wheal should not develop. It is better to begin injecting the local anesthetic too deep and continue injecting as the needle is withdrawn than to inject too superficially, as the retinaculum acts as a barrier to superficially injected anesthetic [41].
Hand

Digital nerve

Four digital nerves course into each digit. The two dorsal nerves run alongside the phalanx at the 2 and 10 o’clock positions, while the two palmar nerves occupy the 4 and 8 o’clock positions. The palmar nerves supply sensation to the volar aspects of the fingers, while the dorsal digital nerves supply the dorsal aspect of the digits. The palmar nerves of the middle three fingers also supply sensation to the dorsum of the fingertips, including the nailbed. Therefore, in the middle three fingers only the palmar digital nerves must be blocked to provide anesthesia to the area distal to the DIP joint. In the thumb and fifth finger all four nerves must be blocked to obtain adequate anesthesia of the fingertip and nailbed [41].

A variety of approaches may be used to perform a digital block. A few of them will be described here. When an option exists, the dorsal approach may be preferred to the volar approach as the dorsal skin is thinner and less painful to inject.

A 3.75-cm 25-gauge or 27-gauge needle is inserted at the web space, lateral to the bone and just distal to the knuckle. A subcutaneous wheal of 0.5 mL to 1.0 mL of local anesthetic is injected at this point. This will anesthetize the dorsal digital nerve and injection site. The needle is advanced lateral to the bone until the palmar skin begins to tent. At this point the needle is withdrawn 1 mm and 0.5 mL to 1.5 mL of local anesthetic is injected. This is repeated on the opposite side of the digit so that all four nerves are blocked [42].

A variation of this technique may cause less pain to the patient. One side of the digit is injected as described above, but rather than withdrawing the needle, the clinician redirects it across the dorsum of the digit to anesthetize the opposite side. The needle is then withdrawn, and may be reinserted into the area that was previously anesthetized. The block then proceeds as usual.

As noted above, the palmar approach may be more painful than dorsal needle insertion. However, in the middle three fingers, where only the volar nerves must be blocked for most common fingertip injuries, it is a useful technique. The needle is inserted over the center of the metacarpal head and local anesthetic is injected while the needle is advanced to the bone. The needle is then withdrawn 3 to 4 mm and angled to the left and right to block both volar nerves. Four to 5 mL of local anesthetic should be injected in total. A variation of this technique can be used in children to provide anesthesia for the most common fingertip injuries. The finger is pinched just distal to the proximal finger crease such that the skin overlying the finger crease is tented. A needle is inserted here and 0.5 to 1.0 mL of local anesthetic is injected subcutaneously. This single injection will anesthetize both volar digital nerves [41].

As discussed above, some clinicians perform digital blocks with epinephrine-containing solutions. At this time, most emergency medicine
authorities advise against this practice. If epinephrine is accidentally injected into a digit, phentolamine may be used to reverse the alpha-agonism.

**Metacarpal and intrathecal blocks**

At least one study has suggested that a digital block provides more rapid and consistent anesthesia than a metacarpal block [43]. The transthecal technique is probably equivalent to the traditional digital block [44]. These techniques are therefore not discussed here.

**Bier block (intravenous regional anesthesia)**

Intravenous regional anesthesia (known as a Bier block) has been shown in numerous studies to be a safe and effective form of regional anesthesia for procedures involving the upper or lower extremities [45–48]. It is a reliable method to achieve consistent anesthesia, a bloodless field, and muscle relaxation. No deaths have ever been directly linked with its use. The procedure may be used in any patient who is able to cooperate. Contra-indications include an allergy to the local anesthetic being used, uncontrolled hypertension, severe peripheral vascular disease, and soft tissue damage in the proximal extremity.

The procedure should be explained to the patient and an intravenous line placed in the unaffected extremity as a “backup” for resuscitation; 0.5% lidocaine without epinephrine is used. This can either be purchased as such or made by diluting 1% lidocaine in equal parts sterile saline. The usual dose is 3 mg/kg (recall there are 10 mg of lidocaine in 1 mL of 1% lidocaine), but a dose of 1.5 mg/kg is nearly as efficacious, and should be used as the starting point in the emergency department [49]. More lidocaine may always be given if needed. The lidocaine solution should be premixed in a 50-mL syringe.

Other anesthetic agents have no advantage over lidocaine, and should not be used. Bupivacaine should never be used due to the risk for severe cardiovascular and neurologic sequelae.

A pneumatic tourniquet is applied over cotton padding proximal to the pathology. A double-cuff pneumatic system is ideal. A standard blood pressure cuff should not be used, as it is prone to failure under prolonged high pressures.

The tourniquet is inflated and a 20-gauge catheter is inserted in a superficial vein, at least 10 cm distal to the tourniquet. Some studies show a higher success rate with more distal catheter placement. The tourniquet is now deflated and the extremity exsanguinated by either elevating it or wrapping it with an elastic bandage. Elevation is less painful to the patient, but must be done for at least 3 minutes and may be somewhat less effective than wrapping. As some authorities do not believe exsanguination is essential, one should be careful to avoid causing the patient unnecessary pain with the wrapping. The wrapping is done in a distal-to-proximal direction with the extremity elevated, being careful not to dislodge the previously placed catheter. With
the arm still elevated, the pneumatic cuff is inflated to 250 mmHg (or 50 mmHg above the systolic pressure in a child). The extremity is lowered and the wrap, if used, is removed.

The lidocaine solution is slowly injected into the catheter at the predetermined dose. The patient will begin to note paresthesias or warmth in 3 to 5 minutes. It begins at the fingertips and progresses to the elbow to complete the anesthesia in 10 to 20 minutes. Muscle relaxation follows. If adequate anesthesia is not achieved in 15 minutes, more lidocaine may be infused, but the 3 mg/kg limit should never be exceeded. Another option is to inject 10 to 20 mL of saline solution. Once adequate anesthesia is achieved, the catheter is removed, the site is tightly taped to prevent leakage of the local anesthetic, and the procedure is performed [45].

A double-cuff tourniquet system may be used to alleviate tourniquet discomfort, which many patients experience after 20 to 30 minutes. In this system there is both a proximal and distal cuff. The proximal cuff is inflated at the beginning of the procedure and the anesthetic solution infused. Anesthesia will be obtained under the distal cuff. When the patient complains of pain under the proximal cuff, the distal cuff may be inflated. Be careful to not deflate the proximal tourniquet until the distal cuff is adequately inflated. Most patients are only able to endure approximately 1 hour of tourniquet time, limiting this block to procedures that can be performed in less than this amount of time [50].

At the completion of the procedure, a careful sequence must be followed to avoid potential lidocaine toxicity. If the procedure was completed in less than 30 minutes, the lidocaine may not have achieved adequate tissue fixation, and deflation of the cuff may produce a high peak plasma level of lidocaine. Therefore, the clinician should wait a full 30 minutes before attempting to deflate the tourniquet. At this point, the tourniquet is deflated for 5 seconds and reinflated for 1 to 2 minutes. This cycle is repeated three to four times and the patient observed for 20 minutes before being discharged [45].

Severe complications are rare, and include seizures and cardiovascular problems. High blood levels of lidocaine may be avoided by cautiously preventing a large intravascular bolus from reaching the systemic circulation. This may be accomplished by not deflating the cuff until at least 30 minutes have elapsed, not using blood pressure cuffs as tourniquets, never exceeding the 3 mg/kg dosage recommendation, and avoiding the placement of the catheter proximal to the tourniquet.

Femoral nerve block

The femoral nerve block has been used in the treatment of femoral shaft fractures for over 50 years. It has been shown to be effective for hip fractures as well [51]. Some studies suggest that patients with femoral neck fractures prefer this block to opioid analgesia [52]. Complications are rare, but careful aspiration of the syringe must be performed before injection of the local
anesthetic, given the proximity of the femoral vein and artery [53]. A careful neurovascular exam must be performed both before and after the procedure.

To perform this block, the patient is placed in a supine position. The skin overlying the femoral triangle is prepared in the usual fashion. The femoral artery is palpated 1 to 2 cm distal to the inguinal ligament, and a subcutaneous wheal of local anesthetic is injected 1 to 2 cm lateral to this point. The nondominant hand is kept on the femoral artery throughout the remainder of the procedure. A 1.25-cm 22-gauge needle is attached to an extension tube setup and a 20-mL syringe. It is inserted just lateral to the artery at a 90° angle, and is advanced until a paresthesia is produced or the needle pulsates laterally. If a paresthesia is elicited, the needle is assumed to be in close proximity to the nerve and 10 to 20 mL of local anesthetic is injected. If no paresthesia is elicited, 10 to 20 mL of local anesthetic is injected in a fan-like distribution lateral to the artery in an attempt to anesthetize the nerve [41].

3 in 1 block (inguinal perivascular block)

The 3-in-1 block may be used to block the femoral, obturator, and lateral femoral cutaneous nerves with a single injection. The femoral nerve runs down the thigh in a fascial sheath that is continuous with a nerve sheath that contains all three nerves more proximally. If a large amount of local anesthetic is injected into this sheath it will track proximally and block all three nerves. This block demands injection of the local anesthetic into the nerve sheath. Although 20 to 30 mL of local anesthetic is being injected, the nondominant hand must be used to apply firm pressure distal to the injection site both during and for 5 minutes after the injection. It may take up to 30 minutes for this block to reach its peak efficacy [41].

Ankle blocks

Five peripheral nerve branches supply sensation to the foot. The superficial peroneal, deep peroneal, and saphenous nerves are anterior and supply sensation to the dorsum of the foot. At the level of the ankle, the superficial peroneal nerve consists of several branches, all of which lie superficially between the lateral malleolus and extensor hallucis longus tendon. These branches provide sensory supply to a large part of the dorsum of the foot. At the ankle, the deep peroneal nerve runs under the extensor hallucis longus tendon. It supplies the sensation for the web space between the first and second toes. The saphenous nerve parallels the saphenous vein between the medial malleolus and tibialis anterior tendon. It supplies sensation to the medial foot, around the arch. The posterior tibial and sural nerves are posterior and supply sensation to the volar aspect of the foot. The sural nerve lies superficially between the lateral malleolus and Achilles tendon. It supplies the lateral aspect of the foot, both volar and dorsal. The posterior tibial nerve lies deep and posterior to the posterior tibial artery.
between the medial malleolus and Achilles tendon. It provides sensation to most of the volar surface of the foot and toes [41].

**Posterior tibial nerve**

With the patient prone, and the foot off the end of the bed in slight dorsiflexion, the posterior tibial artery is located just posterior to the medial malleolus [54]. A point is selected 0.5 to 1.0 cm superior to this. A 3.75-cm 25-gauge needle is inserted here, just posterior to the artery, and directed at a 45° angle to the mediolateral plane. When 0.5 to 1.0 cm deep, the needle is moved side to side in an attempt to produce a paresthesia. If one is produced, 3 to 5 mL of local anesthetic is injected after aspiration. If a paresthesia is not produced, the needle is advanced further until it contacts the posterior border of the tibia. It is withdrawn 1 mm, and 5 to 7 mL of local anesthetic are injected while the needle is withdrawn another 1 cm [41].

**Sural nerve**

The sural nerve lies superficially, and is easily blocked by subcutaneously injecting 3 to 5 mL of local anesthetic between the Achilles tendon and lateral malleolus at a level 1 cm above the lateral malleolus. The patient should be positioned as for the posterior tibial nerve block [54].

**Superficial peroneal nerves**

The patient should be placed in a supine position. These nerves are superficial and are blocked by depositing 4 to 10 mL of local anesthetic subcutaneously between the extensor hallucis longus tendon and the lateral malleolus [41]. Alternatively, a wheal of 0.5 to 1.0 mL of local anesthetic may be deposited midway between the anterior tibial edge and the lateral malleolus [54].

**Deep peroneal nerve**

The deep peroneal nerve is blocked 1 cm above the base of the medial malleolus with the patient supine. The anterior tibial tendon is palpated while the patient dorsiflexes the foot. The extensor hallucis longus tendon is palpated by having the patient dorsiflex the great toe. The needle is placed between these two tendons and a SQ wheal of local anesthetic is deposited. The needle is then advanced under the extensor hallucis longus tendon (about 30° laterally) until it contacts the tibia. This occurs at a depth of less than 1 cm. At this point the needle is withdrawn 1 mm and 1 mL of local anesthetic is injected [41,54].

**Saphenous nerve**

The saphenous nerve is superficial, and is blocked by subcutaneously injecting 3 to 5 mL of local anesthetic between the medial malleolus and anterior tibial tendon.
**Digital toe blocks**

Digital toe blocks in the foot are more efficacious and comfortable for the patient than local infiltration of local anesthetic into the toes. Due to the fibrous septa and limited subcutaneous space, local infiltration here may be painful, ineffective, and causative of local tissue ischemia.

Two volar and two dorsal digital nerves supply each toe. The dorsal digital nerves are branches of the deep and superficial peroneal nerves, while the volar digital nerves branch from the posterior tibial and sural nerves. In the toes, the nerves lie close to the bone at the 2, 4, 8, and 10 o’clock positions. More proximally, they parallel the tendons and are further from the bone [41].

Multiple sites for injection may be considered. The metatarsal approach is performed by inserting the needle dorsally between the metatarsal bones and injecting 1 mL of local anesthetic in a subcutaneous wheal. The needle is advanced until it tents the volar skin, and 2 mL of local anesthetic are deposited as the needle is withdrawn. The needle is then redirected laterally and this procedure repeated. Due to sensory overlap, this procedure must be performed in two or three adjacent spaces for one toe to be blocked. It is difficult to achieve consistent block of the digital nerves using this approach as the nerves do not lie in a predictable location at this level of the foot.

The recommended approach to achieve a digital block of the toe is the web space block. A 27-gauge 3.75-cm needle is inserted at the lateral edge of the bone just proximal to the base of the toe. A subcutaneous wheal is made by injecting 0.5 to 1.0 mL of local anesthetic, and then the needle is advanced until it is seen to almost exit the volar skin; 0.5 to 1.0 mL of local anesthetic is injected and the needle is withdrawn while simultaneously injecting another 0.5 mL of local anesthetic. This is repeated on the opposite side of the toe. Variations such as those used in the finger may be used to minimize patient discomfort and repetitive needle sticks. If the block is performed more distally, in the toe itself, the technique is the same but less local anesthetic is used due to the risk of vascular compromise in such a small subcutaneous space [41].

**Intercostal block**

An intercostal nerve block provides pain relief over the cutaneous area supplied by the nerve blocked. It is commonly employed in the emergency department for patients with broken or contused ribs. Although theoretically advantageous in that it may decrease hypoventilation, atelectasis, and pneumonia, there are no good studies that show it to be more effective than oral analgesics in the outpatient setting [41]. It is contraindicated in patients with flail chest or an overlying skin infection.

Pneumothorax occurs in up to 1.4% of individual intercostal nerves blocked, with an increased incidence in patients with underlying lung disease. When this fact is combined with a less than desirable success rate and duration
of relief far less than what is required, it is important to thoroughly counsel the patient beforehand. A trial of oral analgesics may be indicated first [55].

An understanding of the anatomy involved is essential to obtaining a successful block. Each thoracic nerve exits the spinal column at the intervertebral foramen. The posterior cutaneous branch immediately branches off and supplies the paraspinal area. The intercostal nerve runs along the subcostal groove with the vein and artery. At the midaxillary line it gives off the lateral cutaneous branches. These nerves provide the sensory supply to the anterior and posterior lateral chest wall, and must be anesthetized to obtain adequate pain relief.

It should be noted that posteriorly only a thin layer of fascia separates the nerve from the pleura. However, at the posterior axillary line, the internal intercostal muscle lies between the nerve and the pleura. Because most rib fractures occur in the anterior or lateral portion of the ribs, the clinician can take advantage of this anatomical advantage by performing most blocks at the posterior axillary line [41].

The injured rib is palpated and followed posteriorly with the patient sitting upright and leaning forward on a Mayo stand [56]. The overlying area is prepared in a sterile manner. The index finger of the nondominant hand retracts the skin at the inferior border of the rib superiorly and over the rib. A 25-gauge needle, 3.75 cm long, on a 10-mL syringe is then directed by the dominant hand to puncture the skin at the tip of this finger. The needle should be directed at an 80° angle pointing cephalad. It is advanced until it contacts the rib. Skin retraction is now released, which will move the needle perpendicular to the chest wall and to the inferior edge of the rib. The syringe is switched to the nondominant hand. The palm is rested against the chest wall and the middle finger walks the needle off the inferior edge of the rib. The needle is slowly advanced 3 mm and aspirated. Two to 5 mL of local anesthetic is deposited while the needle is slowly moved in and out 1 mm. This maneuver ensures the compartment between the internal and external intercostal muscles (which contains the nerve) is penetrated. Due to innervation from overlapping nerves, this technique is then repeated on the two ribs above and below.

The patient should be observed for up to 30 minutes after the procedure. If no symptoms suspicious of a pneumothorax develop, he or she may be discharged home with return precautions. Symptomatic patients should have a chest radiograph taken, and those with pneumothoraces treated in the usual fashion [41].

Facial and oral blocks

General recommendations

Bupivacaine is probably the best local anesthetic for ED procedures in this area due to its prolonged duration of action when compared with lidocaine. Epinephrine should be added (when not contraindicated) when
dental blocks are performed due to the high vascularity of the oral cavity. One exception is the maxillary supraperiosteal injection. Lidocaine with epinephrine provides the most prolonged anesthesia for this procedure [57].

A 27-gauge or larger needle should be used to facilitate aspiration and prevent intravascular injection of the local anesthetic. Aspirating syringes, which contain thumb rings or finger grips, are recommended, as they facilitate aspiration. Topical anesthetics should be used in the oral cavity before needle insertion. The area should be completely dried with gauze and then painted with anesthetic applied to a cotton-tipped applicator. Low concentrations of anesthetic (eg, 2% viscous lidocaine) are ineffective. Agents such as 20% benzocaine or 5% to 10% lidocaine must be used. Benzocaine has the advantages of rapid onset (30 seconds), brief duration (5 to 15 minutes), and low systemic absorption. In addition to the modalities described above, distraction techniques such as shaking the lip may decrease the pain of injection [58].

**Supraperiosteal**

A supraperiosteal block is useful for providing anesthesia to a single tooth. The area to be injected should be prepared as previously described. The mucous membrane is then pulled to expose the mucobuccal fold. For maxillary teeth the mucosa is pulled downward and out, while for the mandibular teeth it is pulled upward and out. The needle should enter the mucobuccal fold with the bevel facing the bone. One to 2 mL of local anesthetic is deposited at the apex of the tooth, close to the periosteum. Complete anesthesia may take 5 to 10 minutes. Because the nerve to be anesthetized lies inside the cortex of bone, this technique may fail if the local anesthetic is placed too far from the periosteum or the root [58].

**Anterior superior alveolar nerve**

This block provides anesthesia to the maxillary canine, the central and lateral incisors, and one half of the upper lip. The area should be prepared as previously discussed. The needle is inserted with the bevel facing bone superior to the apex of the canine and directed into the canine fossa. Two milliliters of local anesthetic are injected [59].

**Middle superior alveolar nerve**

Blocking this nerve will provide anesthesia to the maxillary premolars and part of the first molar. The needle is inserted with the bevel facing bone superior to the apex of the second premolar tooth. Two milliliters of local anesthetic are injected [57].

**Posterior superior alveolar nerve**

This block will provide anesthesia to the maxillary molars. The first maxillary molar sometimes must be separately anesthetized with a middle superior alveolar nerve block to gain complete anesthesia.
The area should be prepared as previously discussed. The patient’s mouth should be half-open. The physician should retract the cheek laterally and insert the needle in the mucosal reflection just distal to the distal buccal root of the upper second molar. The needle is advanced along the curvature of the maxillary tuberosity to a depth of 2 to 2.5 cm. Two to 3 mL of local anesthetic is then injected after aspiration, being careful to avoid injection into the pterygoid plexus [57].

Infraorbital nerve

When local anesthetic is injected adjacent to the infraorbital foramen, it anesthetizes the middle and superior alveolar nerves and the infraorbital nerve. Therefore, this block will provide anesthesia to the intraoral areas previously discussed as well as the skin of the lower eyelid, nose, and upper lip. The infraorbital foramen is located on the inferior border of the infraorbital ridge on a vertical line with the pupil. The intraoral approach is preferred to the extraoral variation, as one study showed it provided a longer duration of anesthesia [60].

The intraoral approach is as follows. While holding one finger over the inferior border of the infraorbital rim a needle is inserted in the labial mucosa opposite the apex of the first premolar tooth approximately 0.5 cm from the buccal surface. The needle is advanced in a plane parallel to the axis of the tooth until it is palpated near the foramen at a depth of approximately 1.5 cm. Two to 3 mL of local anesthetic is deposited near, but not within, the foramen [58,61].

Inferior alveolar nerve block

This block will provide anesthesia to the ipsilateral mandibular teeth and the lower lip and chin via the mental nerve. The area should be prepared in the usual fashion, and the physician should stand on the side opposite to the one being injected. The syringe should be help parallel to the occlusal surfaces of the teeth and angled such that it overlies the first and second premolars on the opposite side of the mandible. A 25-gauge needle may be bent 30° to facilitate this angle. The coronoid process of the mandible is palpated with the non-dominant hand and the cheek retracted laterally. A triangle is visualized in the mucosa posterior to the molars. The needle is inserted in this triangle 1 cm above the occlusal surface of the molars. It is advanced until it contacts the mandibular bone, which is the posterior surface of the mandibular sulcus. The needle is withdrawn slightly and 1 to 2 mL of local anesthetic is injected after aspiration (3 to 4 mL if the needle is placed suboptimally). The bone must be contacted. If it is not it may indicate an approach that was too posterior and directed toward the parotid gland. If the local anesthetic is deposited too far posterior into the parotid gland the facial nerve may be anesthetized. This may produce a temporary Bell’s palsy that will resolve when the local anesthetic wears off. If more local anesthetic is deposited while the needle is withdrawn, the lingual nerve may be anesthetized. The long buccal nerve may be blocked.
as well to provide anesthesia of the mucosa opposite the second and third molars. After the needle is withdrawn, it is reinserted into the buccal mucosa opposite the second molar; 0.5 mL of local anesthetic is injected [58,62].

**Mental nerve block**

The mental nerve is a continuation of the inferior alveolar nerve. It emerges from the mental foramen inferior to the second premolar tooth (in children the foramen lies between the first and second primary molars), and innervates the skin and mucosa of the ipsilateral lip with some midline crossover. For this reason, both mental nerves must be blocked for midline lip anesthesia.

If topical anesthesia is used, the intraoral approach is generally less painful than the extraoral approach [63]. The mental foramen is located approximately 1 cm inferior and anterior to the second premolar (just medial to the pupil along a vertical line). It should be palpated before this block is attempted. The area is prepared in the usual fashion, and the needle is inserted in the lower mucobuccal fold at a 45° angle. One to 3 mL of local anesthetic is injected into the area around the mental foramen [58,64].

**Ophthalmic nerve block**

This technique will provide anesthesia of the forehead and scalp as far posteriorly as the lambdoid suture. The lateral and medial branches of the supraorbital, supratrochlear, and infratrochlear nerves all emerge from the superior aspect of the orbit. The supraorbital notch is along the supraorbital rim directly above the pupil. The supraorbital nerves emerge from it. The supratrochlear nerve emerges 0.5 to 1.0 cm medial to this, and the infratrochlear nerve can be found on the most medial portion of the supraorbital rim.

To perform the block 1 to 3 mL of local anesthetic are deposited adjacent to the supraorbital notch. If this does not produce an adequate block, local anesthetic may be infiltrated subcutaneously along the entire supraorbital rim by injecting 5 mL of local anesthetic superior to the entire length of the eyebrow. A finger should be placed just below the supraorbital rim to reduce the risk of eyelid swelling [58,65].

**Summary**

The emergency physician has a variety of options for providing effective pain relief. A solid understanding of the local anesthetic agents and regional anesthetic techniques is an essential component of every emergency physician’s analgesia armamentarium.

**References**


