Local anesthetics

Mechanism of action

Local anesthetics block the conduction of neural messages from the peripheral toward the central nervous system. The primary mechanism of conduction is the sodium channel. These channels are located on the axoplasmic side of the nerve cell and while closed, prevent sodium influx into the cell. This closed channel creates a negative resting potential inside the nerve cell. After a mechanical, chemical, or electrical excitation, these sodium channels open and allow sodium ions into the cell causing depolarization and impulse propagation. Local anesthetic molecules bind to closed sodium channels, preventing activation and cellular depolarization, thus inhibiting propagation of the nerve impulse [1]. This blockade is maintained until the anesthetic wears off, and is displaced from the sodium channel.

Structure of local anesthetics

Local anesthetics consist of an aromatic (hydrophobic) ring structure connected to a tertiary amine (hydrophilic) by an ester or amide linkage. The difference in the linking chain divides the anesthetic into an ester or amide class (Table 1). The hydrophobic ring structure in local anesthetics requires the addition of hydrochloride salt to create a water-soluble injectable medication. In addition to these general features, each local
anesthetic has a different chemical composition that determines the potency, duration, and onset of action.

The potency of an individual anesthetic is determined by the lipid solubility of the compound [2]. Highly lipophilic anesthetic agents can more readily cross the cell membrane and create a more effective blockade of nerve conduction. The duration of a local anesthetic is determined by the plasma protein-binding potential [3]. This correlation is thought to exist because the local anesthetic receptor is also a protein [4]. By binding the receptor for a longer period of time, conduction is blocked, and metabolism of the compound is delayed. Two of the more common anesthetics, procaine and bupivacaine, are excellent examples. Procaine binds loosely to the protein and is a shorter acting agent, whereas bupivacaine binds tightly and is used as a longer acting anesthetic [1]. Finally, the onset of action is determined by the \( pK_a \) of the particular solution. Only the nonionized form of the anesthetic is able to pass easily through the neural membrane, but only the ionized form (cation) can bind within the sodium channel receptor. The \( pK_a \) is the pH at which equal percentages of the drug exists in ionized and nonionized forms. Local anesthetics with \( pK_a \) values closer to the physiologic pH produce higher concentrations of nonionized bases. This creates a high level of available drug passing through the membrane, causing a faster onset of action. Because of the \( pK_a \), local anesthetics are less effective in acidic situations and more effective in an alkaline environment, which speeds the onset of action by creating a more nonionized anesthetic. For example, the onset of action is decreased in anesthetics containing epinephrine, which require an acidic environment to remain stable. Once inside the neuron, the ionized and nonionized molecules reach equilibrium and allow the

<table>
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<tr>
<th>Generic (Trade)</th>
<th>Potency</th>
<th>( pK_a )</th>
<th>Duration</th>
<th>Maximum dose (mg/kg)</th>
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<tr>
<td>AMIDES</td>
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<tr>
<td>Bupivacaine</td>
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<td>8.1</td>
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<td>Dibucaine</td>
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<td>Etidocaine</td>
<td>4</td>
<td>7.7</td>
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<tr>
<td>Lidocaine</td>
<td>2</td>
<td>7.8</td>
<td>2</td>
<td>4.5 (7 with epinephrine)</td>
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<td>Mepivacaine</td>
<td>2</td>
<td>7.6</td>
<td>2</td>
<td>4.5 (7 with epinephrine)</td>
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<td>Prilocaine</td>
<td>2</td>
<td>7.8</td>
<td>2</td>
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<td>Ropivacaine</td>
<td>4</td>
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<td>ESTERS</td>
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<tr>
<td>Chloroprocaine</td>
<td>1</td>
<td>9.0</td>
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<td>12</td>
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<td>Cocaine</td>
<td>2</td>
<td>8.7</td>
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<td>3</td>
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<td>Procaine</td>
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<td>8.9</td>
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<td>Tetracaine</td>
<td>4</td>
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ionized form to bind the sodium channel, inactivate it, and prevent propagation.

Toxicity

Toxicity from local anesthetics predominantly results from actions on the central nervous and cardiovascular systems. The severity of toxicity is related to the potency, dose, systemic absorption, protein binding, metabolism, and excretion. Most toxic reactions are related to inadvertent intra-arterial administration. This possibility of intra-arterial administration makes anesthetizing highly vascular sites more prone to systemic toxicity. The absorption rates for different sites from highest to lowest are: intercostals, intratracheal, epidural/caudal, brachial plexus, mucosal, distal peripheral nerve, subcutaneous. Because of its high vascularity, the dosage for intercostal blocks is one tenth of the maximum of peripheral blocks.

Metabolism and excretion also play a key role in an anesthetic’s toxicity. These mechanisms differ between the ester and amide classes. Ester anesthetics are rapidly metabolized by plasma pseudocholinesterase. The water-soluble metabolite is then excreted by the urine. Patients who have pseudocholinesterase deficiency (eg, sensitivity to succinylcholine, those taking cholinesterase inhibitors, and patients who have myasthenia gravis) have an increased risk for systemic toxicity. The exception in the ester class is cocaine, which undergoes partial hepatic metabolism while the remainder of the molecule is excreted in the urine unchanged [4]. The amide local anesthetics are metabolized by the liver. Although each agent has a different rate of metabolism, the overall rate of the class is much slower than ester anesthetics. This slowed metabolism mandates smaller doses of the agent over time. Patients who have hepatic dysfunction or poor renal flow are predisposed to a higher risk of systemic toxicity. Care must also be taken with topical application of amide anesthetics such as eutectic mixture of local anesthetics (EMLA) and benzocaine. The metabolites of these agents may induce a methemoglobinemia.

Central nervous system toxicity

The central nervous system (CNS) toxic effects of local anesthetics are directly related to lipid solubility [1]. The gap between the desired clinical effect and systemic toxicity narrows as the lipid solubility of the local anesthetic increases. For example, the high lipid solubility of bupivacaine causes it to have a higher risk for toxicity than lidocaine. In addition to lipid solubility, the protein-binding capacity also contributes to CNS toxicity. Products with higher-protein binding capacity have high concentrations of nonionized molecules, allowing rapid crossing of the blood brain barrier and inducing CNS effects.

CNS toxicity can present as a range of symptoms to include lightheadedness, tongue numbness, metallic taste, and restlessness at low levels, to
perioral paresthesias, slurred speech, and excitability or drowsiness at higher levels. Severe toxicity may cause seizures and coma. The etiology of CNS toxicity is unclear, but thought to be due to depression of inhibitory neurons, while leaving the excitatory pathways unopposed [5]. Anesthetic-induced seizures are treated with benzodiazepines, which raise the seizure threshold. A possible complication of using benzodiazepines is the high protein-binding ability of the compound. By administering the drug intravenously, more toxicity could be created by displacing the anesthetic from proteins in the plasma and increasing the free active form [6]. Thiopental 0.5 to 2 mg/kg can be used as an alternate anticonvulsant, but repeat dosing may be needed because of the short duration of action. Careful attention should be given to oxygenation and ventilation because hypoxia, hypercarbia, and acidosis all worsen the toxicity of local anesthetics.

**Cardiac toxicity**

Cardiac toxicity is caused secondary to blockade of the sodium channels in the cardiac conduction system [1]. The increased activity in reentrant pathways and reduction of the refractory period predisposes the heart to ventricular dysrhythmias. High plasma concentrations also cause depression in myocardial contractility. Dilation of smooth muscle may also cause hypotension. The symptoms of cardiac toxicity can vary from palpitations, cardiac dysrhythmias, hyper- or hypotension, and cardiovascular collapse [7]. Cardiac toxicity usually occurs after the stimulant phase of CNS toxicity. Lipophilic agents such as etidocaine and bupivacaine are most likely to cause cardiac toxicity, but it may result from any of the other local anesthetics. Cardiac toxicity is amplified by the use of epinephrine-combined anesthetics, hypoxia, hypercarbia, and acidosis [3].

As with seizure toxicity, bupivacaine is often associated with increased levels and incidence in cardiac toxicity. A range of effects including hypotension, atrioventricular block, and dysrhythmias has occurred. High degrees of protein-binding and depolarization changes are the proposed reasons for increased toxicity.

Advanced cardiac life support is used to manage cardiovascular collapse for most local anesthetic injections. One exception to advanced cardiac life support guidelines is the use of lidocaine as an antiarrhythmic, because it is also a local anesthetic. Although controversial, high doses of epinephrine have also been effective in bupivacaine-induced cardiac toxicity. Bretylium given at 5 mg/kg and repeated up to 30 mg/kg can occasionally convert ventricular tachyarrhythmias to normal sinus rhythms, or at least facilitate electrical cardioversion. Unfortunately effects from Bretylium may take 30 minutes to occur, so prolonged resuscitation is needed [8].

**Allergic reactions**

True allergies to local anesthetics are rarely seen. True allergic reactions will usually produce some form of skin or upper airway involvement.
Careful history of the event can allow a physician to discriminate true allergic reactions to anesthetics. Many times the patient will report an uncomfortable drug effect, vagal reaction, or intra-arterial injection of anesthetic as an allergic reaction. True reactions are usually due to the metabolite para-aminobenzoic acid in ester anesthetics and the preservative methylparaben in amide anesthetics. In cases whereby a true allergy is suspected from initial history, a preservative-free agent from the other class should be used. The ester class is responsible for most true allergic reactions. Therefore, if the previous allergic reaction source is in doubt, it is safest to administer a preservative-free amide local anesthetic (eg, cardiac lidocaine). The risk of a hypersensitivity reaction to this agent is extremely low.

Other alternatives for patients who have had prior reactions include benzyl alcohol and diphenhydramine. Benzyl alcohol with saline or epinephrine is the most effective alternative [9]. Diphenhydramine has been shown to cause prolonged analgesia, prolonged rebound hyperesthesia, and severe pain on injection [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 [11].

Topical anesthetics

Overview

Topical agents have been used since the latter half of the nineteenth century with the advent of cocaine. A century later, there are many safer anesthetic agents that have become available. Repairing lacerations no longer requires a painful injection for anesthetic delivery. In some cases injections may be avoided all together, or performed without pain, through an area treated with a topical anesthetic. In addition to decreasing pain for the patient, topical anesthetics avoid wound edge distortion, which may be produced by subcutaneous infiltrates. There are three main categories of topical anesthetics, based on their location of use: mucosal membranes, non-intact skin, and intact skin.

Topical anesthetics for mucosal membranes

The mucosa of the nose, mouth, and pharynx may be numbed with cocaine or lidocaine. Cocaine is a unique anesthetic in that it also has vasoconstrictive properties, which makes it useful to decrease pain and blood loss. A 4% cocaine solution provides rapid and effective anesthesia for approximately 45 minutes. The maximum dose given should be less than 3 mg/kg. Disadvantages to cocaine are its potential for coronary artery vasoconstriction, hypertension, and tachycardia. Because of these factors, it should not be used in patients who have known or suspected coronary artery disease. Federal regulatory issues and its excessive cost make it a somewhat unattractive agent.

Lidocaine in 1% to 4% concentration is an excellent alternative to cocaine on mucosal surfaces. Although it does not have intrinsic vasoconstrictive
properties, additives such as phenylephrine or epinephrine can be added for this effect. Caution must be used to avoid exceeding the maximum weight-based dose because topical application results in a high level of absorption. Calculating the maximum dose before administration can help minimize error. To determine the amount of anesthetic in a given volume, simply multiply the concentration by 10 to determine the milligrams in each 1 mL of solution. For example, 4 mL of 4% viscous lidocaine contains 160 mg of lidocaine. More dilute solutions may be used in situations requiring high volumes.

Application of topical anesthetic to mucous membranes can be accomplished in various methods. Atomizers can be used to create a fine mist for absorption. Lidocaine jelly can be rubbed directly into the mucosa. Cotton swabs or pledgets can be soaked in solution and introduced into the nose to anesthetize an entire surface or selective area. Vaporizers, such as those used for albuterol, can also be used to effectively anesthetize the entire oropharynx, including the vocal cords [12].

**Topical anesthetics for intact skin**

The three choices available for topical anesthesia to intact skin are: EMLA, LMX-4 (formerly 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 (thickening agent), and sodium hydroxide (to adjust the pH to 9.4). Initial US Food and Drug Administration (FDA) approval for EMLA cream was for use on intact skin only; however, more recent studies suggest EMLA might be effective and safe for use in open wounds [13,14]. Further studies are needed before EMLA can be recommended for nonintact skin. Because of its high cost (approximately $5.00 per application) and prolonged onset of action, it is not often used in the emergency department (ED).

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. Because of its slow onset of action (45–60 minutes) it should be applied to the intact skin for at least 1 hour, but no longer than 2 hours before the planned procedure. Once applied, it should be covered with an occlusive dressing. The anesthetic penetrates 3 mm in 60 minutes, and 5 mm after a 120-minute application [15]. The effect of the anesthetic will last for 1 to 2 hours. EMLA improves patient comfort during procedures and has a benign profile. Minor complications of EMLA include blanching of the skin, redness at the application site, and contact dermatitis. There is a theoretic risk of methemoglobinemia in infants less than 3 months old because of their low levels of methemoglobin reductase. There is one metanalysis that demonstrated no increased risk of methemoglobinemia when used in neonates, but in general it is not recommended in patients under 3 months old. Although there is a theoretic risk of lidocaine and prilocaine toxicity with EMLA, it should not occur if the product is used as directed. Its application can be advantageous in venipuncture, arterial
puncture, lumbar puncture, and arthrocentesis, but its slow onset of action makes it impractical for the large majority of emergency medicine patients.

LMX-4 contains 4% lidocaine cream in a liposomal matrix and is FDA-approved for pain relief from minor cuts and abrasions. Liposomes enhance the action of lidocaine by facilitating the rate and extent of its absorption, allowing it to provide adequate anesthesia in only 30 minutes [16,17]. Unlike EMLA, LMX-4 does not require an occlusive dressing. In limited trials it seems to be a safe topical anesthetic, but some trials have shown little or no improvement over EMLA cream in a 30-minute period [18]. It also does not contain prilocaine, which has been recently implicated in EMLA-induced methemoglobinemia in older children. Thus LMX-4 theoretically should not induce methemoglobinemia—even in young infants.

Lidocaine iontophoresis is another method of dermal anesthesia for intact skin. Iontophoresis allows the introduction of soluble, positively charged lidocaine hydrochloride into the skin by using small external electrical current. Studies have shown this technique to be safe and efficacious [19] with an insignificant systemic absorption of lidocaine [20–22]. Lidocaine iontophoresis achieves anesthesia in 10 to 20 minutes, making it a more rapid alternative to EMLA cream, but some studies suggest EMLA produces better analgesia, which should be borne in mind when selecting for patients [23]. Undesirable effects of this method include the sensation of electrical current flowing through the skin, temporary erythema, blanching, itching, and urticaria at the application site. Iontophoresis devices cost approximately $400, and the one-time use disposable electrodes are $6 to $7.50 each, making this slightly more expensive than EMLA. This cost could theoretically be made up in faster ED turnover.

Topical anesthetics for nonintact skin

TAC (contains tetracaine 0.5%, adrenaline 0.05%, and cocaine 11.8%) and LET (lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5%) are the two primary choices that exist to painlessly provide anesthesia over nonintact skin. Five to 10 mL of TAC is applied to the open wound and securely covered with gauze for 10 to 20 minutes. If securing the gauze with a hand, be sure to use gloves to prevent inadvertent absorption of the medication. TAC can be used anywhere on the body with the exception of the mucous membranes. This will prevent toxicity from the systemic absorption of the agents. It works best on injuries to the face and scalp. There are significant concerns with the use of TAC because of reported cases of respiratory arrest, seizures, and death in children due to its improper application [24]. TAC is expensive, has potential for drug abuse, and also has the regulatory issues involved with dispensing and administering a cocaine-containing medication. These problems have led to most EDs abandoning its use.

An excellent alternative to TAC is LET. Studies have shown LET to be safer, cheaper, and as effective as TAC [25–27]. LET comes in a solution or
gel form, which are equally efficacious [28]. However, the gel form may be easier to use because of less run off. To apply LET, place 1 to 3 mL of the agent directly to the wound and leave covered for 15 to 30 minutes. Like TAC, LET should not be applied directly to or near the mucous membranes, the pinna of the ear, the nose, penis, fingers, or toes.

Topical anesthetics have also been effective in relieving pain in burn victims. Some studies have shown a possible healing benefit, with improved blood flow to the affected area. In these studies, application of EMLA has been used within normal limits and has not been associated with systemic toxicity [29,30]. Although treatment of burn relief has been studied, no studies could be found that evaluate the use of topical agents in severe abrasions or “road rash” injuries. Although the mechanism is different, similar tissue damage and pain is expected, and it is not unreasonable to treat these wounds with topical agents, as long as they are applied within dosing recommendations.

Local anesthetics used for subcutaneous infiltration

General

Local anesthetics are classified into either amides or esters (see Table 1). The key differences in these two groups include allergic potential and metabolism. Esters are hydrolyzed in the plasma by pseudocholinesterase, a much more rapid process than the hepatic metabolism of the amides. Due to a slower metabolism, the amides have an increase in possible toxicity, especially in patients who have liver dysfunction. Breakdown of ester compounds by pseudocholinesterase produces para-aminobenzoic acid, a potentially allergenic substance. Because amide metabolism does not create this byproduct, most allergic reactions to local anesthetics are due to esters.

Lidocaine

The most commonly used local anesthetic for intradermal infiltration in the ED is lidocaine. It is effective and has a good safety record. It is available commercially as a 1.0% to 2.0% solution with a pH of 6.5 and a pK_a of 7.9. The onset of action is 4 to 7 minutes after injection. The maximum dose is 3 to 5 mg/kg, up to 300 mg in a single dose. This dose increases with the addition of epinephrine to 7 mg/kg of anesthetic. Solutions without epinephrine provide analgesia for approximately 1.5 hours, whereas those with epinephrine last approximately 3.5 hours. Traditional teaching is that epinephrine should never be used in the hands or feet because of the risk of ischemia from constriction of end arterioles. However, recent literature suggests that with careful screening, epinephrine may be used when performing digital blocks [31–33]. In instances of prolonged ischemia as a result of the injection, arterial vasospasm may be reduced by locally injecting 2 mg of phentolamine near the site or applying topical nitroglycerine.
Bupivacaine

Bupivacaine is a potent local anesthetic whose anesthesia is equivalent to lidocaine. Its high protein binding and $pK_a$ cause its onset of action to occur at 10 to 15 minutes, and its duration of action to be 3 to 6 hours. It is available as either a 0.25% or 0.5% solution. The maximum doses of bupivacaine are 2.0 to 2.5 mg/kg without epinephrine, and 3.0 to 3.5 mg/kg with epinephrine. The dose can be repeated every 3 hours, with the total dose given in 24 hours not to exceed 400 mg. Although bupivacaine provides long patient comfort, the risk of systemic toxicity with bupivacaine is much higher than most other local anesthetics secondary to its high potency and protein-binding properties. This toxic potential has led to the development of newer agents such as ropivacaine and levobupivacaine.

Ropivacaine

Ropivacaine is an amide local anesthetic approved by the FDA in 1996. It is similar to bupivacaine in terms of potency, onset time, and duration of action, but it is 70% less likely to cause the cardio-toxic effects associated with bupivacaine. This agent does have some intrinsic vasoconstrictive properties, so it probably should not be used on end arterial areas. The cost in comparison to bupivacaine is much higher [34].

Levobupivacaine

Levobupivacaine is the S-isomer form of bupivacaine. The pure S-enantiomer has fewer cardiovascular and CNS side effects when compared with regular bupivacaine. Although the potency of the two drugs is similar, the cost of five times more than bupivacaine hinders its use in most EDs [35].

Mepivacaine

Mepivacaine is structurally similar to lidocaine. Its onset of action is similar, but its duration of action is much longer (3 hours). By adding epinephrine, the duration of action is increased by 20% to 30%. Local infiltration solutions come in 0.5% or 1.0%. Mepivacaine does have a reputation for being more toxic than lidocaine, which has limited its clinical use.

Reducing the pain of injection of local anesthetics

There are many techniques that can be used to decrease the pain of injection for local anesthetics. Slow administration in a proximal to distal direction, along with a small needle (27–30 gauge) can decrease the pain of injection [36]. Injecting into tissue that has been exposed by the wound causes less pain than injection through intact skin [37].

Many studies have shown that buffering lidocaine can reduce the pain of infiltration [38–42]. Buffered solutions can be made by adding sodium bicarbonate (44 mEq/50 mL) to lidocaine in a 1:10 ratio (1 mL of bicarbonate is added to 10 mL of lidocaine). However, buffering lidocaine causes an
increased rate of biodegradation at room temperature and decreases its shelf life by 7 days. The limiting factor for addition of sodium bicarbonate to local anesthetic is the tendency of more lipid-soluble agents to precipitate. For example, a highly lipid-soluble agent like bupivacaine must be buffered in a 1:50 ratio to avoid precipitation.

Warming of the anesthetic solution has also been shown to reduce pain of administration. However, other studies have shown it to have little or no effect. A reasonable compromise is to ensure that the agent is at least used at room temperature. Lidocaine can be warmed in either dry heat, such as a blanket warmer, or in a temperature-regulated water bath at 37°C. If lidocaine has not been buffered, warming will not shorten shelf life.

**Addition of vasoconstrictors to local anesthetics**

Adding a vasoconstrictive agent such as epinephrine offers several potential advantages to local agents. Epinephrine can improve a physician’s ability for exploration and closure because of increased hemostasis in the surgical field. This effect is especially beneficial because most anesthetics cause smooth muscle relaxation and vasodilation. By constricting the blood vessels, the rate of systemic absorption is decreased. This allows an increase in the maximum dose of anesthetic, reduces systemic toxicity, prevents redistribution of the anesthetic, and prolongs the duration of action.

Commercially prepared solutions of lidocaine and bupivacaine containing epinephrine (1:100,000 to 1:200,000) are available and widely used for subcutaneous infiltration in EDs. These preparations are acidified to a pH of 3 to 4.5 to stabilize the epinephrine component. Because of this acidification, they have a slower onset of action and can be associated with more pain on injection than single agents.

**Regional anesthesia**

**General indications and contraindications**

Regional anesthesia is indicated in areas that are amenable to the blockade of a specific peripheral nerve (or nerve group) and provides a possible advantage over other techniques. As always, the method of anesthesia chosen is a decision made between the physician and the patient. The factors influencing the decision may include patient preference, physician comfort with a particular technique, and time constraints in busy EDs. The risks and benefits of each proposed procedure(s) must be explained to the patients, so they may consent after making a fully informed decision. Multiple nerve blocks are discussed in the following section, and indications for each block are explained within.

Generally, nerve blocks are contraindicated in uncooperative patients and in those who are unable to communicate severe pain on injection. Severe pain with injection is a good indicator of an intraneural injection, which
may cause ischemic nerve injury. Other general contraindications include infection over the site, distortion of anatomic landmarks, and an allergy to the local anesthetic being used [43]. Contraindications to specific procedures are discussed (see later discussion).

Advantages

Emergency physicians have multiple methods of reducing discomfort and pain during procedures. Peripheral nerve blocks have multiple benefits over local infiltration during anesthesia. First, lower levels of anesthetic may be used to produce a greater effect and avoid risks of systemic toxicity. Second, regional blocks are often less painful to perform than subcutaneous injections and help result in less anxiety for the patient. Lastly, regions of injury that have great cosmetic significance (such as the lip and face) can be distorted by local injection and hinder the physician’s ability to approximate wound edges properly. By using a regional block in these injuries, tissue distortion is avoided, and a less edematous field for repair is created.

Disadvantages

Patient selection in regional anesthesia is important, because success is guided by high levels of patient cooperation to detect subtle paresthesias during the procedure. There is a risk of systemic toxicity (approximately 7.5/10,000), most of which is due to inadvertent intravascular injection, and a small risk of peripheral nerve damage (1.9/10,000) [44]. Failure of a nerve block is also a risk, because the procedure is usually performed without a stimulator in the ED. This risk of failure is likely related to the clinical experience of the physician, but requires the patient be informed that further anesthesia may be necessary.

General techniques

Peripheral nerve blocks should be performed in an area with adequate monitoring and resuscitation equipment because of the risk of systemic toxicity. Accidental intravascular injection of the anesthetic is responsible for immediate toxicity. Multiple techniques can be used in attempts to prevent vascular injection. Careful aspiration of the syringe before injection is generally used, and the use of an “immobile needle” technique can also be useful. This technique is performed by connecting a syringe to the needle by a piece of flexible tubing. This flexible tubing allows the operating physician to use both hands for needle localization and stabilization while an assistant aspirates and injects the agent with a syringe. Delays in toxicity may occur when excessive amounts of local anesthetic are absorbed into the systemic circulation. The different properties of each anesthetic agent will alter the timing of delayed toxicity. A high index of suspicion is needed to diagnose the fist signs of both immediate and delayed toxicity.
Patients who have severe pain or anxiety before a procedure may be premedicated with a systemic benzodiazepine or opioid to reduce anxiety and pain of injection. However, sedation must be light to facilitate adequate patient cooperation with the procedure. Skin preparation should involve antiseptic fashion before needle insertion. Providone–iodine is a traditional agent, but there are now multiple agents that are acceptable.

Knowledge of local anatomy and adequate patient cooperation are required for a successful block. Most procedures are performed with “blind” techniques, which rely on production of paresthesia in the nerve’s sensory distribution when contacted by the needle. Successful blocks require local anesthetic to be injected in proximity to the nerve (perineural) but not directly within it (intraneural). Intraneural injection can cause elevated pressures in the sheath and may produce ischemia. Pain intensity and duration serve as clues to the injection site of the agent. Perineural injections cause a brief increase in the paresthesia, whereas intraneural injections produce an intense and prolonged pain. Immediate termination of the injection is indicated if intraneural positioning is suspected, and the needle should be repositioned for adequate anesthesia [38].

**Wrist blocks**

Although effective, wrist blocks have a slow time or onset and can be time consuming if a total block is required. For many injuries to the hand, digital blocks or local infiltration could be more effective. In certain situations wrist blocks are indicated and seem to be the best choice. Examples of the injuries amenable to a wrist block include: road rash, thermal burns requiring debridment, hydrofluoric acid burns, injury to more than one finger, and lacerations to the palm.

**Radial nerve**

Landmarks for the radial nerve include the radial artery and the radial styloid. Once these landmarks are located, inject 3 mL of local anesthetic at the level of the styloid just lateral to the radial artery. After this initial injection, proceed dorsally with subcutaneous injections of the anesthetic along the wrist until the dorsal midline is reached. Approximately 5 mL of local anesthetic is required for this procedure [45].

**Ulnar nerve**

The ulnar nerve runs into the wrist alongside the ulnar artery, lying just below the flexor carpi ulnaris tendon at the level of the proximal crease. The flexor carpi ulnaris tendon can be localized just proximal to the pisiform bone when the wrist is flexed against resistance. At the palmar crease, the nerve lies deep to the ulnar artery, which makes the volar approach difficult. A lateral approach to the nerve is recommended, which allows access to the nerve without risk of arterial puncture [46]. A 25-gauge needle is inserted in
the ulnar side of the wrist at the proximal palmar crease. The needle is advanced in a horizontal plane 1.0 to 1.5 cm underneath the flexor carpi ulnaris. Three to 5 mL of local anesthetic is required for ulnar nerve block. Cutaneous nerve branches of the ulnar nerve wrap around the wrist to supply the dorsum of the hand. These cutaneous nerves 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 [41].

Median nerve

The median nerve can be blocked at the proximal volar crease, between the flexor carpi radialis tendon and palmaris longus tendon. In 20% of the population, there is no palmaris longus tendon at present. In the absence of this landmark, the median nerve can be found 1 cm in the ulnar direction from the flexor carpi radialis tendon. A 25-gauge needle is inserted vertically through the skin to a depth of approximately 1 cm. At this depth, a “pop” should be appreciated and a paresthesia elicited as the physician penetrates the deep fascia of the flexor retinaculum. During injection, no skin wheel should appear if the deep fascia has been penetrated. It is better to begin the injection too deep and proceed with injection as withdrawing than to inject too superficially. The retinaculum is an effective barrier to anesthetic if it is not penetrated [41].

Hand

Digital nerve

Each digit contains four digital nerves for sensation. The two dorsal nerves run at the 2- and 10-o’clock positions, whereas the palmar nerves lie at the 4- and 8-o’clock positions. Both palmar nerves supply sensation for the volar aspect, whereas dorsal sensation is supplied by the dorsal digital nerves. In addition to the palmar surface, the palmar nerves of the middle three digits supply sensation to the nailbed and dorsum of the fingertip. Because of this distribution, only palmar digital nerves require blockade to provide anesthesia to the area distal to the distal interpharyngeal joint. All four nerves must be blocked in the thumb and fifth finger for adequate anesthesia [41].

There are various approaches that may be used in performing digital blocks. The dorsal approach is preferred over the volar approach because the dorsal skin is thinner and less painful to inject. A small 25- or 27-gauge needle is inserted at the web space distal to the knuckle and just lateral to the bone. A small subcutaneous wheal of 0.5 mL to 1.0 mL of anesthetic is injected at this point. The needle is then advanced lateral to the bone until tenting of the palmar skin is noted. Once tenting is seen, the needle is withdrawn 1 mm and injected with 0.5 mL to 1.5 mL of local anesthetic. The same procedure is repeated on the opposite side of the digit so that all four nerves are blocked [47].

In an effort to cause less pain to the patient, the physician may alter the technique in the following manner. One side of the digit is injected as
described in the previous procedure, but instead of withdrawing the needle, it is redirected across the dorsum of the digit to anesthetize the opposite side. The needle is then withdrawn and inserted into the area that was previously anesthetized. The block is then finished as described in the previous paragraph.

When dealing with fingertip injuries on the middle three digits, only blockade of the volar nerve is required for adequate sedation. Although the dorsal approach is less painful, decreased amount of anesthetic make this a useful technique. The needle is inserted over the center of the metacarpal head on the volar side, and local anesthetic is injected while the needle is advanced to the bone. Once at the bone, the needle is withdrawn 3 to 4 mm, then angled to each side of the digit for complete volar nerve block. A total injection of 4 to 5 mL of agent is all that is required for the blockade. When dealing with fingertip injuries in children, a different version of the volar block is effective. The finger of the child is pinched just distal to the proximal finger crease, so that minor skin tenting is formed. A needle is then inserted into the skin, and 0.5 to 1.0 mL of local anesthetic is injected subcutaneously. This single injection will anesthetize both volar digital nerves in children.

Most emergency medicine authorities advise against using epinephrine-containing solutions during digital blockade. However, studies and case reports of vasoconstriction are limited. If epinephrine is used and accidentally injected into a digit, phentolamine may be of benefit in reversing 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, but others are mixed [48,49]. The transthecal technique is probably equivalent to the traditional digital block [50]. These techniques are therefore not discussed here.

Bier block (intravenous regional anesthesia)

The Bier block is a form of intravenous regional anesthesia and has been shown in numerous studies to be a safe and effective form of regional anesthesia for upper and lower extremities [51–54]. Its favorable traits are the ability to achieve consistent anesthesia, a bloodless field, and muscle relaxation. There have been no deaths directly linked with its use. The procedure can be used in any patient who is able to cooperate. Contraindications include an allergy to anesthetic, uncontrolled hypertension, severe peripheral vascular disease, and soft tissue damage in the proximal extremity.

Place an intravenous line (IV) in the unaffected extremity in case resuscitation is required. The anesthetic to be used is 0.5% lidocaine without epinephrine. Other anesthetics have no advantage over lidocaine and should not be used. Bupivacaine should never be used because of its risk for severe
cardiovascular and neurologic sequelae. The lidocaine can be purchased commercially or made by diluting 1% lidocaine in equal parts with sterile saline. The lidocaine solution should be premixed in a 50-mL syringe. The recommended dose is 3 mg/kg, but a dose of 1.5 mg/kg is nearly as efficacious and should be used as a starting point in the ED [55]. If needed, more lidocaine can always be given up to the recommended dose.

A pneumatic tourniquet is applied over cotton padding proximal to the pathology. A double-cuff pneumatic system is ideal. The tourniquet is inflated and a 20-gauge catheter is placed in a superficial vein, greater than 10 cm distal to the tourniquet. Some studies have shown a higher success rate with more distal catheter placement. After the IV is placed, the cuff is deflated and the extremity is exsanguinated by either elevating it or wrapping it with an elastic bandage. Manual exsanguinations accomplished by wrapping an elastic bandage from the distal to the proximal of the extremity is more painful than elevation, but is faster and more effective. Care must be taken not to dislodge the intravenous catheter. Some authorities do not feel that exsanguination is essential and that simple elevation of the arm is adequate for the procedure. After wrapping the extremity, the arm is elevated and the pneumatic cuff is inflated to 250 mm Hg (or 50 mm Hg above the systolic pressure for children). The extremity is then lowered and the wrap, if used, is removed.

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

The double-cuff tourniquet is used to alleviate tourniquet discomfort, which patients may experience after 20 to 30 minutes. The system consists of a proximal and distal cuff, in which the proximal cuff is inflated before infusion of anesthesia. Anesthesia will be obtained under the distal cuff. When the patient complains of pain under the proximal cuff, the distal cuff may be inflated. Do not deflate the proximal cuff until after the distal cuff is fully inflated. Most patients are only able to endure approximately 1 hour of tourniquet time, limiting this block to procedures that can be completed in that amount of time [56].

After the procedure is complete, a careful sequence must be followed to avoid potential lidocaine toxicity. If the procedure takes 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, a full 30 minutes should be complete before deflation of the tourniquet. At this time, 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 is observed for 20 minutes before discharge [45].

Severe complications are rare and include seizures and cardiovascular problems. By cautiously preventing a large intravascular bolus from reaching the systemic circulation by following the deflation procedure, high blood levels of lidocaine may be avoided. Other measures to avoid systemic circulation include waiting 30 minutes before deflation, using double cuff tourniquet rather than blood pressure cuffs, never exceeding the 3-mg/kg dosage, and avoiding placement of the catheter proximal to the tourniquet.

**Femoral nerve block**

Pain from femoral shaft fractures has been treated with femoral nerve blocks for over 50 years. Hip fractures have also been responsive to pain control with femoral blocks [57]. There is also evidence that patients who have femoral neck fractures prefer femoral blocks to opioid analgesia [58]. Proximity of the femoral vein and artery create high potential for intravascular injection, but careful aspiration of the syringe before injection greatly reduces this risk [59]. A careful neurovascular examination must be performed both before and after the procedure.

The block is performed with the patient in a supine position. Antiseptic technique is used to prepare the skin over the femoral triangle. Palpate the femoral artery 1 to 2 cm distal to the inguinal ligament. Once localized, move 1 to 2 cm laterally and inject a subcutaneous wheal of local anesthetic. In order to sustain landmarks, 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 a 20 mL syringe using an extension tube technique. The needle is inserted just laterally 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 agent is injected in a fan-like distribution lateral to the artery in an attempt to anesthetize the nerve. Careful aspiration of the needle is required to avoid intravascular injection [41].

**3 in 1 block (inguinal perivascular block)**

The 3-in-1 block is used to block the femoral, obturator, and lateral femoral cutaneous nerves with a single injection. The nerve sheath that surrounds the femoral nerve travels proximally and becomes continuous with a wider nerve sheath that contains all three nerves. By injecting large amounts of local anesthetic in to the femoral nerve sheath, fluid will track proximally and block all three nerves. The femoral nerve injection site is used, while 20 to 30 mL of local anesthetic is injected. During the injection, the nondominant hand must be used to apply firm pressure distal to the injection site, and continuous pressure must be applied for 5 minutes after the injection. It may take up to 30 minutes for this block to reach its peak efficacy [41].
Ankle blocks

The foot is supplied by five peripheral nerve branches. The superficial peroneal, deep peroneal, and saphenous nerves course anteriorly and supply sensation to the dorsum of the foot. Multiple branches of the superficial peroneal nerve lie close to the surface between the lateral malleolus and extensor hallucis longus tendon. These branches provide sensory supply to most of the dorsum of the foot. The deep peroneal nerve supplies the web space between the first and second toes, and runs into the ankle under the extensor hallucis longus tendon. The saphenous nerve runs parallel with 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

The patient is placed in the prone position, with the foot hanging off the end of the bed in slight dorsiflexion. In this position, the posterior tibial artery is located just posterior to the medial malleolus [60]. 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. Advance the needle to a 0.5 to 1.0 cm depth, and then move from side to side in an attempt to produce a paresthesia. Once a paresthesia is induced, aspirate and then inject 3 to 5 mL of local anesthetic. In cases whereby no paresthesia can be elicited, advance the needle further until it contacts the posterior border of the tibia. After contact is made, withdraw the needle 1 mm and inject 5 to 7 mL of local anesthetic while withdrawing the needle another 1 cm [41].

Sural nerve

Place the patient in the same prone position as with the posterior tibial nerve block. The sural nerve lies superficially and is easily blocked using a subcutaneous wheel. The nerve is found 1 cm above the lateral malleolus, between the Achilles tendon and lateral malleolus. At this site, a skin wheel of 3 to 5 mL is produced to block this superficial nerve [54].

Superficial peroneal nerves

Place the patient in a supine position. The superficial nerves are located between the extensor hallucis longus tendon and the lateral malleolus [41]. Blockade of the nerves can be achieved by placing 4 to 10 mL of local anesthetic subcutaneously at this location. An alternative method consists of
placing a wheal of 0.5 to 1.0 mL of local anesthetic midway between the anterior tibial edge and the lateral malleolus [54].

**Deep peroneal nerve**

With the patient in the supine position, and the foot dorsiflexed, palpate the anterior tibial tendon above the medial malleolus. The extensor hallucis longus tendon is then palpated by having the patient dorsiflex the great toe. The needle is inserted 1 cm above the base of the medial malleolus between these two tendons. A subcutaneous wheal of local anesthetic is deposited, and the needle is then advanced under the extensor hallucis longus tendon (approximately 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 located superficially 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 nerve blocks in the foot are more efficacious and comfortable for the patient than attempts with local infiltration. Discomfort with local infiltration is thought to be secondary to the fibrous septa and limited subcutaneous space. In certain instances, these factors cause the block to be ineffective and increase the likelihood of local ischemia.

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

There are multiple injection sites that may be considered for digital blocks. The metatarsal approach is performed by inserting the needle dorsally between metatarsal bones and injecting 1 mL of local anesthetic in a subcutaneous wheal. The needle is then slowly advanced until tenting of the volar surface is noted. At that time 2 mL of local anesthetic is deposited as the needle is being withdrawn. Before being removed from the skin, the needle is then redirected laterally and the procedure is repeated. To fully block one digit, this procedure must be performed in two or three adjacent spaces because of sensory overlap of the nerves. Consistent blocks of the digital nerves are difficult to achieve with this technique, because the nerves do not lie in a predictable location.

The recommended and most commonly used approach for a digital block of the toe is the web space block. A 3.75-cm 27-gauge needle is inserted at
the lateral edge of the bone just proximal to the base of the toe. A subcutaneous wheal is created by injecting 0.5 to 1.0 mL of local anesthetic. The needle is then advanced until tenting of the volar skin is visualized. While stationary, inject 0.5 to 1.0 mL of local anesthetic. Another 0.5 mL of local anesthetic is then injected while simultaneously withdrawing the needle. The procedure is then repeated on the opposite side of the toe. As discussed with digital blocks of the finger, variations may be used to minimize patient discomfort and multiple needle sticks. In cases whereby only the distal portions of the toe must be anesthetized, the block can be performed further away from the web space. In these situations, it is important to decrease the amount of local anesthetic due to risking vascular compromise in such a small subcutaneous space [41].

*Intercostal block*

Intercostal nerve blocks provide cutaneous relief over the affected area. They are commonly used in the ED to provide analgesia for broken or contused ribs. In theory, the advantages of eliminating pain with inspiration may decrease hypoventilation, atelectasis, and pneumonia, but there are no good studies that show it to be more effective than oral analgesics in outpatient settings [41]. Intercostal blocks are contraindicated in patients who have flail chest or infection over the site of injection.

There is a small chance of creating a pneumothorax with intercostals blocks. The rate is reported as high as 1.4%, and is increased in patients who have underlying lung disease. The questionable relief and inconsistent duration of the block, along with a small chance for pneumothorax, require thorough counseling of the patient beforehand. A trial of oral analgesics may be indicated first [61].

Successful blocks require a thorough understanding of the involved anatomy. Each thoracic nerve exits the spinal column at the intervertebral foramen. Immediately after exiting the foramen, the posterior cutaneous branch splits off to supply the paraspinal area. The intercostals nerve runs along the subcostal groove with the vein and artery. As the nerve reaches the midaxillary line, it gives off lateral cutaneous branches. Blocking these lateral cutaneous nerves will provide relief to the anterior and posterior lateral chest wall.

Performing the block in the posterior midaxillary line is advantageous because of several factors. In the posterior region, only a thin layer of fascia separates the nerve from the pleura, creating greater potential for pneumothorax. In the posterior midaxillary line, the internal intercostal muscles lie between the nerve and pleura creating a small buffer. Because most rib fractures occur in the anterior or lateral portion of the ribs, most blocks can be performed in the posterior midaxillary line and still provide adequate relief [41].

The patient is placed sitting in an upright position and leaning forward on a Mayo stand. The injured rib is then palpated and followed posteriorly to the posterior midaxillary line [62]. The overlying area is then prepared in a sterile
manner. Retract the skin at the inferior border of the rib superiorly with the index finger of the nondominant hand. A 3.75-cm 25-gauge needle on a 10-mL syringe is then inserted by the dominant hand into the skin at the tip of the finger. The needle is directed cephalad at an 80° angle. The needle is then advanced until it contacts the rib. Release skin traction with the dominant hand, which will move the needle perpendicular to the chest wall and to the inferior edge of the rib. At this time, the syringe is switched to the nondominant hand. The palm is rested against the chest wall and the middle finger is used to walk the needle off the inferior edge of the rib. Slowly advance the needle 3 mm, aspirate, and inject 2 to 5 mL of local anesthetic while slowly moving the needle in and out 1 mm. This in and out movement ensures penetration of the compartment between the internal and external intercostals muscles where the nerve lies. The same technique is then repeated on the two ribs above and below the initial injection to ensure blockage of overlapping nerves.

The patient should then be observed for 30 minutes after the procedure for signs and symptoms of a pneumothorax. If the patient is stable after this time, they may be discharged home with return precautions. Symptomatic patients should receive a chest radiograph and treated with the standard of care in instances of pneumothorax [41].

**Facial and oral blocks**

**General recommendations**

The best local anesthetic for ED procedures in this area is bupivacaine. The prolonged duration of action makes it superior to lidocaine in these cases. Epinephrine should be used (when not contraindicated) for dental blocks, because of the high vascularity of the oral cavity. One exception to the use of bupivacaine is performance of the maxillary supraperiosteal injection. Lidocaine with epinephrine provides the most prolonged anesthesia for this procedure [63].

Because of the high vascularity involved, a 27-gauge or larger needle should be used to facilitate aspiration and prevent intravascular injection of anesthetic. Needles specifically designed with a thumb ring or finger grip help improve aspiration technique. Multiple techniques may be used to decrease the pain of injection in this sensitive area. Topical anesthetics can be placed on a thoroughly dried area by using a cotton tip applicator. Low concentrations are ineffective, but agents such as 20% benzocaine or 5% to 10% lidocaine are effective. Benzocaine is highly preferred because of its rapid onset of action (30 seconds), brief duration (5–15 minutes), and low systemic absorption. Distraction techniques, such as shaking the lip during injection, can also be used in concordance with topical anesthetics to decrease pain [64].

**Supraperiosteal**

Supraperiosteal blocks provide anesthesia to a single tooth. Preparation of the oral mucosa is used before injection. The mucous membrane is then
pulled in the necessary direction to expose the mucobuccal fold. The needle is inserted into the mucobuccal fold with the bevel facing the bone. Deposit 1 to 2 mL of local anesthetic at the apex of the tooth, close to the periosteum. Complete anesthesia may take 5 to 10 minutes. Because the nerve runs inside the cortex of the bone, this procedure may fail if the local anesthetic is placed too far from the periosteum or the root.

**Anterior superior alveolar nerve**

By performing this block, anesthesia is provided to the maxillary canine, the central and lateral incisors, and one half of the upper lip. General preparation is the same as previously described. The needle is inserted with the bevel facing bone superior to the apex of the canine and directed into the canine fossa. After insertion, 2 mL of local anesthetic are injected.

**Middle superior alveolar nerve**

This nerve provides sensation 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 mL of local anesthetic are injected.

**Posterior superior alveolar nerve**

This block will provide anesthesia to the maxillary molars. In some instances to gain complete anesthesia, the first maxillary molar must be separately anesthetized.

The patient’s mouth should be half open and the cheek retracted laterally. The needle is inserted in the mucosal reflection just distal to the distal bucal 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. At this point, the needle is aspirated, and 2 to 3 mL of local anesthetic is then injected, being careful to avoid injection into the pterygoid plexus.

**Infraorbital nerve**

Infraorbital blockade is extremely useful in repairing cosmetic lacerations that would become distorted with local infiltration. This block provides 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 in line with the pupil and on the inferior border of the infraorbital ridge. The intraoral approach is preferred to the extraoral variation, because it provides a longer duration of anesthesia.

The block is performed by holding one finger over the inferior border of the infraorbital rim and inserting the needle in the labial mucosa opposite the apex of the first premolar tooth approximately 0.5 cm from the bucal surface. The needle is advanced superiorly until it is palpated near the foramen at a depth of approximately 1.5 cm. At this point, 2 to 3 mL of local anesthetic is deposited near, but not within, the foramen.
**Inferior alveolar nerve block**

The inferior alveolar nerve provides sensation to the ipsilateral mandibular teeth and the lower lip and chin. The buccal mucosa is prepared in the usual fashion, and the physician should position him/herself on the side of the patient opposite of the one being injected. Hold the syringe parallel to the occlusal surfaces of the teeth and angle it so that it overlies the first and second premolars on the opposite side of the mandible. If needed, a 25-gauge needle may be bent to 30° to facilitate this angle. Use the non-dominant hand to palpate the coronoid process of the mandible. At this point, a triangle should be visualized in the mucosa posterior to the molars. The needle is then inserted into this triangle approximately 1 cm above the occlusal surface of the molars. Advance the needle until contact is made with the mandibular bone, which creates the posterior surface of the mandibular sulcus. Ensuring the needle comes in contact with the bone will prevent posterior injection of anesthetic into the parotid gland. If this occurs, the seventh cranial nerve will be anesthetized and will create a temporary Bells palsy. After location is verified, inject 1 to 2 mL of local anesthetic after aspiration. Up to 3 to 4 mL can be used as necessary if the needle is placed suboptimally. After the needle is withdrawn, it is reinserted into the buccal mucosa opposite the second molar, and 0.5 mL of local anesthetic is injected [58,68].

**Mental nerve block**

The mental nerve is a continuation of the inferior alveolar nerve and innervates the skin and mucosa of the ipsilateral lip, with mild midline crossover. For this reason, both mental nerves must be blocked when midline lip anesthesia is required.

The mental nerve emerges from the mental foramen 1 cm inferior and anterior to the second premolar tooth. In children, the foramen lies between the first and second primary molars. Internal approach to the nerve is generally less painful than an external approach if a topical anesthetic is used. Palpate the mental foramen in the described location; then insert the needle in the lower mucobuccal fold at a 45° angle. At this time, 1 to 3 mL of local anesthetic is injected into the area around the mental foramen [58,69].

**Ophthalmic nerve block**

The ophthalmic nerve provides sensation to the forehead and scalp as far posteriorly as the lambdoid suture. The nerve consists of lateral and medial branches of the supraorbital, supratrochlear, and infratrochlear nerves. The nerves all emerge for the superior aspect of the orbit. The supraorbital nerve emerges from the supraorbital notch, which is found on the supraorbital rim directly above the pupil. The supratrochlear nerve is located 0.5 to 1.0 cm medial to the foramen, and the infratrochlear nerve is found on the most medial portion of the supraorbital rim.
The block is performed by placing 1 to 3 mL of local anesthetic subcutaneously adjacent to the supraorbital notch. If this initial injection does not provide adequate anesthesia, 5 mL of local anesthesia can be injected subcutaneously along the supraorbital rim the entire length of the eyebrow. Eyelid swelling can be reduced by firmly placing a finger just below the supraorbital rim [58,70].

**Procedural sedation and analgesia in wound management**

Most wounds in the ED may be adequately anesthetized with local and regional techniques as previously described. Larger, multiple, and more extensive wounds are generally addressed in an operative setting; however, procedural sedation and anesthesia (PSA) does have a limited role in certain clinical scenarios. PSA may be utilized as a primary means of decreasing pain and discomfort during assessment, cleansing, and definitive repair of more painful and extensive wounds. PSA agents may also be given to ease the pain of local anesthetics in areas that are particularly painful to inject (ears, nose, genitalia), or as an adjunct to local anesthetics when they alone cannot completely anesthetize a given wound. Examples of wounds seen in the ED that may not be adequately anesthetized with local or regional anesthetics include burns, deep puncture wounds requiring debridement or packing and diffuse, and superficial abrasions with foreign bodies (such as seen with road rash). PSA as a primary or adjunctive method for analgesia may be appropriate at times, and the practicing emergency physician should be comfortable with several agents that can be used in this role. This section provides a brief overview of agents that may be useful in this setting, but the reader should refer to a more in-depth review of PSA for more specific recommendations on dosages, adverse effects, and medication profiles.

**General concepts**

Selecting an appropriate agent depends on several factors. Adequate analgesia will be a primary goal in most scenarios, although sedation, anxiolysis, or amnesia may also be necessary because some patients have a significant aversion or fear of injections. Sedation may be required (eg, a terrified and struggling pediatric patient with wounds to the face) to provide motion control during local injections. In these instances, a short period of sedation (enough to inject the wounds) may be all that is necessary. The length of time for the management of a wound should be considered and the proper agent should be chosen that will provide adequate analgesia while allowing timely discharge from the ED if that is a goal.

Intravenous delivery of any PSA agent is the most reliable and effective method. This provides quicker peak levels and allows the provider to more accurately titrate the agent when necessary. Intramuscular (IM) methods provide less reliable absorption rate and peak effects. IM delivery may be
occasionally considered when IV access is not available or easily obtainable, or the act of obtaining IV might cause more anxiety and discomfort for a given patient. Ketamine, which is discussed later, is an exception because it can reliably and quickly be delivered IM. Oral and transmucosal (rectal and intra-nasal) routes can be excellent choices, especially in the pediatric population. Inhaled nitrous oxide is a good analgesic/sedative choice that does not require injection or an IV; however, most EDs do not carry this because of difficulty maintaining the required delivery and scavenging systems. Regardless of the route of administration, proper monitoring of blood pressure, pulse oximetry, and ventilatory status of the patient should be maintained until the patient’s mental status and vital signs have returned to their preprocedural state.

**Opioid analgesics**

Opioids, in general, are a good choice for PSA in wound management. They provide analgesia as well as some level of sedation. They differ in potency and half-lives, but generally all function to elevate pain thresholds and may be used in conjunction with other agents. Respiratory depression, nausea, and hypotension are potential adverse effects, but serious effects may be reversed with opioid antagonists. Care must be taken when using with sedatives, because synergism may occur that worsens the adverse effects such as respiratory depression and hypotension. Patients who have significant pain can be given IM analgesics upon arrival, but monitoring of the respiratory status is required with larger doses because there may be a delay to peak effect using this route.

**Morphine**

Morphine is a longer acting opioid with peak effect in approximately 15 to 20 minutes and duration of action of approximately 1 to 2 hours when given IV. Onset of action with IM delivery of morphine is more variable with delayed onset (5–20 minutes) and peak onset of approximately 60 minutes. The terminal half-life of morphine is 1.5 to 2 hours and duration of analgesia approximately 3 to 4 hours. Because it is poorly lipid soluble, morphine does not quickly enter the cerebrospinal fluid and is therefore poorly titratable. This makes morphine a poor choice for PSA in the ED. As an adjunct to local anesthesia, it may be used for a patient who has a painful wound early in the ED course, but the operator must allow adequate time to pass before beginning the procedure. This can allow the provider time to prepare for a local or regional block while instituting analgesia and sedation for the procedure. It is a good choice for painful injuries and wounds that should require extended analgesia even after ED wound management.

**Fentanyl**

Fentanyl is approximately 100 times more potent than morphine but is lipid soluble and therefore reaches peak cerebrospinal fluid concentrations
quickly within a few minutes. This makes it desirable as a PSA agent because it is easily titratable. Fentanyl also has a shorter duration of action (30–40 minutes) than morphine, which theoretically allows a more timely discharge of patients. It has much less histamine release than morphine, resulting in less bronchospasm and hypotension than morphine. Nausea, vomiting, and hypotension are possible but rare. A reasonable starting dose for fentanyl is 1 to 2 μg/kg IV, especially if used with a sedative or for an adjunct to local anesthetic. A typical cumulative dose of fentanyl for PSA is approximately 2 to 5 μg/kg, titrating boluses of 1 μg/kg at a time to achieve the desired effect. Chest wall rigidity is a rare complication seen with rapid administration of IV fentanyl in doses much higher than used with PSA (>5 μg/kg bolus) and should not be of concern.

**Ketamine**

Ketamine is an excellent choice for PSA in painful ED wound management. It is a dissociative anesthetic and provides analgesia with amnesia and sedation. This produces an “awake” patient that is dissociated from external stimuli in a trance-like state, but whose respiratory drive is maintained. It has an outstanding side effect profile and only rarely causes respiratory depression, as with other analgesics or sedatives. Most often this is seen when IV opioids or benzodiazepines are added to ketamine as adjuncts. Most respiratory difficulties are related to positioning and increased airway secretions; therefore, suctioning and repositioning of the airway usually suffice to address airway difficulties. Laryngospasm is an infrequent complication that can usually be managed with bag valve mask ventilation, and severe laryngospasm requiring paralysis and intubation has only rarely been reported [71].

Ketamine may be effectively delivered IM at doses of 2 to 5 mg/kg, depending on the depth of desired sedation and length of sedation, with approximate onset of action within 5 minutes and duration of 20 to 30 minutes. IV administration allows for repeated titration and onset of action of 1 minute with duration of 15 to 20 minutes. Doses range from 0.5 to 2 mg/kg IV, with repeat doses of 0.5 mg/kg infused as needed. Return to baseline consciousness generally occurs in 15 to 20 minutes after the last IV dose, unless larger doses are employed. Oral ketamine at doses of 10 mg/kg may also be employed in patients in whom IV access is not obtained, and seems to allow for better sedation than oral midazolam [72]. Because ketamine is a sialagogue, glycopyrrolate or atropine are often used to decrease secretions, although this has not been definitively proven in clinical trials. Concomitant administration of a benzodiazepine may decrease the incidence of the emergence phenomenon (hallucinations, bizarre movements, agitation) in adults, but has not been shown to be effective in children [73,74].

Ketamine would be a good choice in several clinical scenarios in ED wound management. Repeat boluses of 0.5 mg/kg ketamine IV, or 2 mg/kg
IM may allow for adequate analgesia and a cooperative semialert patient. This state would be ideal for certain painful procedures such as burn/wound debridement or follow-up wound care (painful packing changes). Ketamine generally provides adequate analgesia and sedation if delivered alone, but low-dose ketamine may be effective with IV opioids for opioid-resistant pain [75]. No definitive human data exist to clarify if opioids provide additional benefit to low-dose ketamine in opioid naïve patients without increasing side effects, but this combination may be useful in performing painful procedures such as burn debridement in the ED.

Sedatives/anxiolytics/amnestic

Sedatives alone do not provide analgesia, which is the primary desired characteristic sought in an agent used for wound management. Sedatives may be employed with analgesics to decrease anxiety or impart amnesia so that the painful manipulation of a wound is not recalled by the patient. When used alone, sedatives usually require a significant depth of sedation for painful procedures. Often the risk of deeper sedation for this purpose is not appropriate because better options exist. In addition, sedated patients may still perceive the pain and purposefully withdraw or move, making wound management difficult.

Low-dose sedatives may be added to opioids for certain painful procedures, although this may increase the incidence of side effects through synergism, and close monitoring is again warranted.

Benzodiazepines

Midazolam is a short acting benzodiazepine with an onset of 2 to 3 minutes and duration of 30 to 60 minutes, depending on the dose. Intravenous doses range from 0.02 to 0.04 mg/kg and may be titrated based on patient response, making midazolam the preferred benzodiazepine in ED PSA. It is often combined with fentanyl to achieve the desired effect, because both possess a quick onset and short duration. Case reports of death from this combination of agents exist, but it carries a level B recommendation in clinical policy consensus despite the concerns for significant respiratory depression [76,77].

Barbiturates

Barbiturates are also sedatives that provide no analgesic effects. Ultra-short-acting agents, such as methohexital, can produce significant and brief sedation for painful procedures. Hypotension and respiratory depression are frequently seen at doses that impart deep sedation in patients (1–2 mg/kg IV of methohexital or thiopental). Barbiturates are not routinely combined with opioids for PSA, and their short duration may be suitable only as an adjunct for local anesthetics or brief painful wound manipulation.
**Etomidate**

Etomidate produces brief sedation, anxiolysis, and amnesia but without analgesia. Unlike barbiturates, it does not affect hemodynamic stability. Respiratory depression is a dose-related side effect and myoclonus, seen in approximately 10% of patients, may initially prevent motion control until it subsides. Nausea and vomiting occurs occasionally at the usual dosage of etomidate for PSA (0.1–0.15 mg/kg IV) [78]. Evidence for use of etomidate as a PSA agent in the ED in adults and children is mounting, but currently etomidate has a level C recommendation in a clinical policy consensus [79]. Drug characteristics also limit its usefulness in wound management and, like barbiturates, may be primarily suitable as an adjunct for local anesthetics or brief painful wound manipulation.

**Propofol**

Propofol is also a pure sedative without analgesic properties with brief onset and duration (less than 1 minute onset and several minutes duration). A usual PSA dose for propofol is 0.75 to 1 mg/kg and then 0.5 mg bolus every 1 to 2 minutes, or given as a continuous infusion of 25 to 75 μg/kg/min. Propofol produces amnesia, sedation, and hypnosis, but no analgesic effects. It produces less nausea and vomiting due to its antiemetic effects but has a high incidence of respiratory depression, apnea, and hypotension. Fortunately, most of these effects are brief and, with proper monitoring of the airway and blood pressure, have not been shown to cause significant complications in limited studies [80]. It may be used in combination with fentanyl to provide adequate analgesia in addition to sedation for the management of painful wound manipulation. In pediatric patients, the fentanyl–propofol combination currently carries a level B recommendation, and propofol alone has a level C recommendation in a national consensus clinical policy [79].

**Summary**

The practicing emergency physician should be comfortable in his or her ability to alleviate pain when managing patients’ wounds in the ED. This can be effectively and safely accomplished through a firm understanding of regional and local anesthetic techniques, procedural sedation and analgesia, and the possible side effects and toxicities of any agents that are employed for this purpose.

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