Tintinalli, Judith E., Kelen, Gabor D., Stapczynski, J. Stephan
Emergency Medicine: A Comprehensive Study Guide, 6th Edition

37

Local and Regional Anesthesia

Eric Higginbotham

Robert J. Vissers

Local anesthesia, as it is known today, is the result of the discovery of suitable drugs and delivery methods. Cocaine was first isolated in Europe between 1859 and 1860 and introduced into clinical practice about 25 years later. Simultaneously, the advent of a precise syringe that allowed the delivery of an exact amount of a pharmaceutical agent was being developed concomitantly in Scotland and Lyon, France.1 Together these discoveries led to the invention of local anesthesia.

Impure cocaine's toxic and addictive effects were rapidly noticed as its use spread throughout Europe, resulting in many deaths among patients and addicted medical staff. Newer, safer agents were developed in the late 19th and 20th centuries, the ester local anesthetics (e.g., tropocaine, eucaine, benzocaine, and tetracaine) and the amide local anesthetics (e.g., procaine, lidocaine, mepivacaine, prilocaine, and bupivacaine). Overall, these drugs are less toxic than cocaine but still possess central nervous system (CNS) and cardiovascular (CV) system toxicity.

Local and regional anesthetics are essential tools in the ED for procedure- and trauma-related pain management. Local anesthetic (LA) techniques provide short-term pain relief by (1) infiltration or application of LA directly into the area to be anesthetized, (2) infiltration of LA into the region of peripheral nerves supplying the area, or (3) infusion of LA into the venous system of an extremity. Specific skills and anatomic knowledge for regional anesthesia techniques are necessary for the effectiveness of LA procedures, and knowledge of the dosages, actions, and toxicity of the LA medications are required for safe use of these agents.

LOCAL ANESTHETICS

Local Anesthetic Agents

Almost all LA agents are synthetic drugs derived from cocaine (except diphenhydramine and benzyl alcohol, which are discussed separately). The chemical structure includes hydrophilic and hydrophobic regions and a linkage (amide or ester) region. Local anesthetics are weak bases supplied as a salt (usually HCl) in an acidic pH solution to increase stability, solubility, and shelf life. The most commonly used pharmacologic agents for local infiltrative and regional anesthesia are the amide agents, with lidocaine and bupivacaine being most often used. Lidocaine has a shorter duration of action than does bupivacaine, but it possesses a lower toxicity profile. Mepivacaine, with an onset of action

close to that of lidocaine, with a longer duration of action and devoid of bupivacaine toxicity, is becoming an increasingly recognized alternative.2 Tetracaine, an ester LA, is frequently used in topical anesthetic preparations. Ester LAs are hydrolyzed by cholinesterase enzymes in plasma. Amide LAs are metabolized by hepatic microsomal enzymes. The metabolism rates of the amide LA (prilocaine > lidocaine > mepivacaine > bupivacaine) are slower overall than those of ester LA, creating the potential for sustained plasma levels and cumulative effects of amide LA. Maximum doses and duration of action of various anesthetics are shown in Table 37-1.

TABLE 37-1 Local Anesthetic Doses for Infiltration

				Maximum Dose*			
				Without Epinephrine		With Epinephrin	
Drug	Concentration for Infiltration (%)†	Onset (min)	Duration (min)	mg/kg	Total mg	mg/kg	T m
Esters							
Procaine	1	2–5	15–45	7	500	9	6(
Amides							
Lidocaine‡	1	2–5	30–60	4.5	300	7	5(
Bupivacaine#	0.25	3–7	90–360	2	175	3	2:

Mepivacaine	1	3–7	90–180	5	400	7	5(
Others							
Diphenhydramine	0.5–1	<2	20–30	1.25–1.7	90	§	
Benzyl alcohol	0.9	2–5	Unknown	**	**	**	

*Maximum dose based on a 70-kg patient.

†Percentage of solution defined as the number of grams per deciliter: 0.25% = 5 2.5 mg/mL, 0.5% = 5 5 mg/mL, a 1.0% = 5 10 mg/mL.

‡Lidocaine dose can be repeated in 2 to 4 h.

#Bupivacaine can be repeated every 4 to 6 h (maximum daily dose, 400 mg per d).

§Not given with epinephrine.

**See text.

The anesthetic action of LA is produced when the drug molecules occupy enough sodium channels within the axon to interrupt activity and temporarily stop conduction. Local anesthetics bind to receptor sites on the voltage-gated sodium channels (a four-subunit transmembrane protein) in the neuronal membrane and impair or block sodium influx. As LAs increasingly occupy sodium channels, there is a decrease in the rate and degree of depolarization and repolarization, a decrease in conduction velocity, and a prolongation of the refractory period of the neural action potential.3

The pharmacologic activity of the LA agents is based on several of their biochemical properties; lipid solubility, pK_a, and protein binding are the key characteristics. Potency is related to lipid solubility; LAs that are more lipid-soluble easily penetrate the nerve cell wall and result in higher intracellular concentrations and thus more blockade. The pK_a, the pH at which 50 percent of the drug exists in the basic or nonionized state, determines the

speed of onset of conduction block. The base form is more lipid-soluble than the ionized form. Therefore, the lower the pK_a for a specific LA, the more uncharged form of the drug is present at tissue pH, and the faster it traverses lipid layers to the

axoplasm. Protein binding is the affinity an LA has for the sodium channel, and duration of action is related to this affinity; the longer the LA binds to the sodium channel and effects conduction block, the longer the duration of the anesthesia.

Addition of epinephrine (usually in a concentration of 1:100,000, or 10 = µg/mL) provides a longer duration of anesthesia, promotes wound hemostasis, and slows systemic absorption, thereby decreasing the potential for toxicity and allowing a greater volume of agent to be used for extensive laceration repair.4 Epinephrine can increase the pain of infiltration, because it lowers the overall pH of the solution. Epinephrine acts through vasoconstriction to achieve these actions (“chemical tourniquet”). Because of this vasoconstriction, epinephrine has traditionally been avoided in an end-arterial field (e.g., digits, pinna, nose, and penis), although a recent comprehensive review found no cases of digital ischemia since the advent of commercial lidocaine with epinephrine (introduced in 1948).5 If epinephrine is inadvertently injected into an end-arteriole field, 1 in. of topical nitroglycerin paste applied to the affected area will quickly restore perfusion. Blood pressure should be checked to make sure hypotension does not occur, thought it is unlikely. Vasospasm and ischemia from end-arteriole or arterial injection can also be reversed with local or intravascular injection of phentolamine 1.5 to 5 mg. The main side effect of phentolamine, hypotension, is unlikely in this setting.

Toxicity

Toxicity of LA is related to the potency (i.e., lipid solubility) and duration of action (i.e., protein binding) as expressed in the target organ. Toxicity correlates with the amount of nonionized and unbound LA present. It is also related to the total dose of LA and the rate of plasma increase of the LA, with more rapid accumulation being more toxic. The relative absorption of LA is dependent on the vascularity of the site. The absorption therefore, from most rapid to slowest, is: intercostal/intratracheal > epidural/caudal > brachial plexus > mucosal > distal peripheral nerve > subcutaneous. Caution should be exercised with intercostal blocks: the recommended LA dose for intercostal blocks is one-tenth maximum for peripheral blocks. Serious adverse reactions, including systemic toxic reactions, are more frequently encountered with the use of amide rather than with the use of ester LA agents, due in part to the slower amide metabolism. However, patients with atypical plasma cholinesterase may be prone to systemic toxicity from ester LA. The systemic toxicity of LA is enhanced by hypercarbia, hypoxemia, and acidosis. Systemic toxicity is usually the result of rapid inadvertent intravenous injection or delivery of an excessive total dose of LA. Serious systemic toxicity primarily involves the CNS and CV system (Table 37-2).

TABLE 37-2 Toxicity of Local Anesthetics

	Toxic Effects
Central nervous system	Mild: visual disturbance, tongue numbness, lightheadedness, apprehension, restlessness Moderate: perioral paresthesia, muscle twitching, slurred speech, excitability, drowsiness Severe: seizures, cardiorespiratory depression, coma
Cardiovascular system	Palpitations, vasodilation, hypertension, ventricular dysrhythmias (particularly bupivacaine), myocardial depression, hypotension, bradycardia, cardiovascular collapse
Respiratory system	Hypoventilation, respiratory arrest
Allergy	Amides (rare) Esters (more common) associated with para-aminobenzoic acid metabolite
Methemoglobinemia	Cyanosis, dyspnea, dizziness, lethargy; children more susceptible

CENTRAL NERVOUS SYSTEM TOXICITY

Central nervous system toxicity is due to conduction block that occurs within CNS structures. The CNS toxic effects are directly related to lipid solubility; therefore, the agents that are the most effective LAs also carry the greatest CNS toxicity. The more

protein bound the agent is, the narrower the gap is between the initial milder symptoms of CNS toxicity and the more catastrophic cardiovascular reactions to LA. Toxicity is due to a combination of central excitatory and depressant activities of the LA, ranging from perioral tingling and numbness to confusion, seizure, and coma. Vigilance for the subtle symptoms of CNS toxicity is important, because the therapeutic-to-toxic ratios are often narrow with these agents. Seizure, although ominous, should be viewed as a warning for impending ventricular arrhythmias and cardiovascular collapse. Because toxicity of the LA agents is potentiated by hypoxia, hypercarbia,

P.266

and acidosis, seizures with resultant compromise or loss of the airway can hasten life-threatening CV toxicity.3

CARDIOVASCULAR TOXICITY

All LAs have a dose-dependent CV toxic effect that is mediated through sodium channel blockade within the heart. This blockade results in two main types of CV toxicity: myocardial depression and dysrhythmias. All LAs have CV toxicity, although the drugs with the highest lipid solubility have the highest incidence of significant CV toxicity (e.g., bupivacaine). In addition, bupivacaine has a “fast-in, slow-out” kinetic pattern that results in accumulation of the drug within the conduction system that increases as heart rate increases. These pharmacologic properties make bupivacaine particularly cardiotoxic and, for this reason, is contraindicated for intravenous regional anesthesia. When the primary cardiac conduction system is blocked, there is increased activity in re-entrant pathways that predisposes the heart to ventricular arrhythmias. Ventricular fibrillation or tachycardia from LA toxicity can be refractory to treatment due to the degree of sodium channel blockade. Pregnancy has been noted to worsen the toxicity of LA, bupivacaine in particular, which is probably mediated through progesterone and the adverse effects of pregnancy on venous return.

METHEMOGLOBINEMIA

Prilocaine and benzocaine can cause the oxidation of the ferric form of hemoglobin to the ferrous form, creating methemoglobin. When the methemoglobin concentration exceeds 1.5 g/dL, visible cyanosis results. This cyanosis is usually benign, but when oxygen transport needs to be preserved or signs of hypoxia occur, specific treatment may be necessary (see Chap. 189).

ALLERGIC REACTIONS

True allergic reactions to local anesthetics are rare and usually due to the metabolite para-aminobenzoic acid, in the case of ester anesthetics, or the preservative methylparaben, which is structurally similar to para-aminobenzoic acid, in the case of amide anesthetics. Esters are more commonly associated with allergic reactions than are amides. If a true allergy is suspected based on history or documentation, the optimal approach is to use a preservative-free agent from the other class. Diphenhydramine (0.5 to 1.0%) and benzyl alcohol are alternative anesthetic choices in patients allergic to either amide and/or ester-type anesthetics.

MANAGEMENT

Management of systemic LA toxicity follows standard advanced life-support protocols (ensure a patent airway, oxygen, and ventilatory and circulatory support), immediate discontinuation of the administration of LA and prompt treatment of the CNS and CV complications. Correcting hypoxia, hypercarbia, and metabolic acidosis is paramount, because these conditions enhance the toxicity of LA. Incremental doses of intravenous benzodiazepines are usually effective to control seizures, and standard protocols should be followed for the management of ventricular arrhythmias. Arrhythmias refractory to standard advanced cardiac life-support protocols have met with anecdotal success with the use of bretylium and cardiopulmonary bypass.

Specific Agents

AMIDE LOCAL ANESTHETICS

Lidocaine

Lidocaine is an amide anesthetic available in 0.5, 1.0, and 2% concentrations for injection. It is the most commonly used anesthetic in the ED, because of its excellent efficacy and low toxicity profile. Lidocaine has a rapid onset of action and intermediate duration. It has a pK_a of 7.9, with a pH in commercial preparations of 5.0 to 7.0. Lidocaine with epinephrine has a pH within 3.3 to 5.5 (necessitating bicarbonate buffering to decrease pain during infiltration). The elimination half-life is 45 to 60 min. Metabolism is predominantly hepatic, with high plasma levels occurring due to hepatic failure and decreased hepatic blood flow (e.g., congestive cardiac failure). Caution should be exercised if these conditions exist.

Bupivacaine

Bupivacaine is an amide anesthetic that is highly protein bound. The concentration appropriate for local anesthetic use is 0.25%. It has a pK_a of 8.1 and in commercial preparations has a pH of 3.3 to 5.5. Alkalinization with bicarbonate can cause precipitation at a pH of 6.8 or higher. Some studies suggest that its injection is more painful, possibly owing to the lower pH. It has a slow onset, long duration, and a higher CV toxicity potential. The duration of action is 4 to 6 h. Bupivacaine is preferred for prolonged procedures (such as ingrown toenail removal), when longer postprocedural analgesia is desired, and for peripheral nerve regional blocks. Bupivacaine is contraindicated for intravenous regional blocks due to its cardiac toxicity, from which fatalities have been reported. Bupivacaine also should be used very cautiously in any condition affecting hepatic metabolism.

Mepivacaine

Mepivacaine is an amide anesthetic, with a pK_a of 7.6 and a pH of 4.5 to 6.8 in commercial preparations. It has a rapid onset, intermediate duration, and intermediate toxicity. Toxicity, which was once thought to approximate the level of bupivacaine, has been for the most part repudiated.2 Local infiltration with a 1% concentration has been associated with anesthesia

of 1.5 to 3 h in duration. Redosing for longer procedures does not cause tachyphylaxis, and cumulative toxicity is low.3

Prilocaine

Prilocaine, an amide LA with pK_a of 7.9, has a lower cardiac toxicity profile than does lidocaine or bupivacaine, with similar anesthetic potency. It has rapid onset and intermediate duration. After intravenous injection, its CNS toxicity is less than that of lidocaine due to a lower blood level, because of differences in its distribution and peripheral uptake. It is also broken down by amidases in the liver more rapidly than is lidocaine, resulting in a shorter duration of toxic effects. Prilocaine may lead to methemoglobinemia after a large intravenous bolus (or total dose >600 mg) due to the oxidative properties of one of its metabolites, *o*-toluidine. Clinical uses include infiltration with up to a 1% solution with an anesthetic duration of 60 to 120 min. Due to its lower toxicity, a 0.25 to 0.5% concentration is commonly used in Australia for intravenous regional arm blocks. Prilocaine and lidocaine are the active agents in a cream consisting of an eutectic mixture of LAs (EMLA) for topical use on intact skin.

ESTER LOCAL ANESTHETICS

Procaine

Procaine is a short-acting ester LA, with a pK_a of 8.9. Commercially prepared solutions have a pH of 5.5 to 6.0. Procaine has a slow onset and very short blood half-life (approximately 20 s) owing to the rapid hydrolysis by plasma cholinesterase; hence, little toxicity is associated with this agent. Procaine can be used for patients who are allergic to the amide anesthetics (e.g., lidocaine).

Tetracaine

Tetracaine is an ester local anesthetic, with a pK_a of 8.6. In liquid form, the pH can vary from 4.5 to 6.5. It has a slow onset, short plasma half-life (2.5 to 4 min), and long duration of action. Injectable tetracaine 0.2% is used most commonly in spinal anesthesia. In addition, it is used for topical anesthesia of the eye (0.5%) mucous membranes (2%), and skin (4% gel or cream). When applied to the larynx, anesthesia of the airway will persist for a very long duration, with absent airway reflexes. The potential for rapid uptake from mucous membranes does exist with the risk for systemic toxicity. The toxic dose is believed to be approximately 2 mg/kg.

ALTERNATIVE AGENTS

Diphenhydramine 0.5 to 1.0% and benzyl alcohol 0.9% are alternative anesthetic choices in patients with true allergies to the amides and/or ester-type anesthetics.6,7 Although

diphenhydramine is an effective local anesthetic, its injection is more painful than lidocaine and can cause tissue irritation and even skin necrosis.6 Thus, its role for local anesthesia is extremely specific and limited to those patients who have true allergies to ester or amide

anesthetics, which are quite rare. Benzyl alcohol 0.9% with epinephrine (1:100,000) is as effective as lidocaine and superior to diphenhydramine as an LA.6,7 However, the duration of action is short, and about 30 percent of patients need additional anesthetic injection during the procedure.7

LOCAL ANESTHETIC INFILTRATION

The most common usage of LA in the ED is local infiltration for wound repair and invasive procedures. Local infiltration has a rapid onset and low risk of systemic toxicity. Infiltration can be into the wound margins or as a field block in a “diamond-shaped wheal” of LA surrounding the wound or procedure site. Lidocaine is the drug of choice for LA infiltration for brief procedures. Bupivacaine or mepivacaine is preferable for longer procedures due to their longer duration of action, but with due caution regarding the increased toxicity. Epinephrine can be added to enhance safety and efficacy, and bicarbonate can be added to minimize pain of infiltration. The maximal dosages commonly cited apply to infiltration of LA, in contrast to the lower maximal dosages for most regional procedures (Table 37-1 and Table 37-3).

TABLE 37-3 Suggested Volumes for Regional Nerve Blocks in Adults (Weight >40 kg)

	Total Volume
Location	**Lidocaine 1% or Bupivacaine 0.25%**
Median nerve	3–5 mL
Ulnar nerve	5–7 mL
Radial nerve	5–10 mL

Digital	3–4 mL
Metacarpal	2–3 mL
Posterior tibial	3–7 mL
Sural nerve	3–5 mL
Saphenous nerve	3–5 mL
Superficial peroneal nerve	3–5 mL
Deep peroneal nerve	3–5 mL
Great toe	4–6 mL
Lesser toes	2–3 mL

Forehead	3–6 mL
Inferior alveolar nerve	1–2 mL
Mental nerve	1–2 mL (intraoral), 2–4 mL (extraoral)
Infraorbital	2–3 mL
Femoral nerve	10–20 mL
Intercostal nerve (per segment)	3–5 mL

Additives and Adjuncts to Minimize Pain of Infiltration

Many variables influence the degree of pain experienced with LA infiltration. The depth and rate of injection and the temperature and buffering of the agent are considerations in the attempt to make infiltration painless. Slow infiltration of the anesthetic (30 s/mL) with a 27- or 30-gauge needle will decrease the pain as compared with rapid injection with a larger needle, probably due to less rapid distention of local tissue.

Numerous clinical studies have demonstrated attenuation of pain on infiltration with buffered lidocaine.8 Buffered lidocaine is prepared by mixing a solution of 9 mL of lidocaine 1% (with or with- out epinephrine) with 1 mL of sodium bicarbonate 8.4% (1 mEq/mL). The resultant mixture can be prepared ahead of time and stored for future use. Bicarbonate can cause precipitation of bupivacaine and should not be added to this anesthetic unless it can be used immediately. It is prepared by mixing a solution of 29 mL of bupivacaine 0.25%

with 1 mL of sodium bicarbonate 8.4% (1 mEq/mL). The exact mechanisms by which buffering of lidocaine reduces pain is not clear. Buffering increases the pH of these acidically packaged anesthetics, bringing the mixture's pH closer to the drug's pK_a, resulting in a higher percentage of the uncharged species. The higher percentage of uncharged molecules allows faster diffusion into the nerve fiber, resulting in more rapid sensory nerve blockade. Buffering to a higher pH also may reduce direct tissue irritation by reducing the acidity of the agent. Because buffered lidocaine undergoes biodegradation at room temperature, its shelf life is limited; studies suggest that efficacy is maintained from 7 to 30 days.

Studies evaluating the efficacy of warm lidocaine (anywhere from 37°C to 42°C) to decrease injection pain have yielded equivocal results.8 It is postulated that warming the anesthetic causes faster diffusion into tissues and reduces or avoids stimulation of cold receptors, thereby increasing the rate of onset of neuronal block. Lidocaine can be warmed in dry heat (blanket warmers) or in temperature-regulated water baths at 37°C. From a practical standpoint, anesthetics should be administered at least at room temperature. Unlike buffered lidocaine, heated lidocaine undergoes no chemical denaturation, so there are no limitations on shelf life.

Injection through the margins of a wound rather than through intact skin surrounding it greatly reduces the pain associated with infiltration.9 Use of long needles reduces the number of puncture sites. To prevent breakage, no more than two thirds of the needle should be inserted. The smallest appropriate syringe allows for greater control over the volume and rate of injection.

The nuances of local wound infiltration should be mentioned and in most ways is more important than the agent selected in obtaining a desirable outcome. If the caregiver attempts to establish rapport and allay fears early in the encounter, anxious and uncooperative patients can be calmed and much of their evaluation completed before anesthetic infiltration has occurred. The neurologic examination should be demonstrated on an uninvolved, uninjured area first, to reassure the patient that the examiner will not inflict pain. Distraction techniques are beneficial, including the use of music, skin pressure, or pinching. The needle should be hidden from view of the patient, if possible. The analogy of a bee sting or insect bite should be avoided; most patients are terrified of insect bites, and for most children that has been the most painful experience in their memory. Although unstudied, a topical anesthetic application followed by infiltration may be less painful and better tolerated in selected patients.

To summarize, the least painful method of infiltration is deep, slow injection through the wound margins using a warm, buffered solution with the use of a 27- or 30-gauge needle and a concerted effort to minimize the anxiety surrounding the process.

TOPICAL ANESTHETICS

Topical anesthetics can be used to reduce the discomfort of local procedures and may eliminate or decrease the need for local infiltrative injection.10 When compared with local infiltration, topical anesthetics can be applied painlessly, do not distort wound edges, and may provide good hemostasis if the formulation includes a vasoconstrictive agent. In

general, topical anesthetics work better on the head and neck than on the extremities, have a slower onset, and are less efficacious than injectable LAs. There a number of available formulations in common use: TAC (tetracaine, adrenaline, and cocaine), LET (lidocaine, epinephrine, and tetracaine), and EMLA (lidocaine and prilocaine).

P.268

Tetracaine, Adrenaline, and Cocaine

The TAC (original formulation: tetracaine 0.5%, adrenaline 0.05%, and cocaine 11.8%) solution has been largely replaced by other mixtures that are less likely to cause toxicity, cost less, and do not have the regulatory issues associated with a controlled substance. The TAC solution has been associated with seizures, respiratory arrest, and, rarely, death in children due to inadvertent systemic absorption of cocaine. Systemic absorption was most common when used on mucosal surfaces or in large amounts. Other agents such as LET have been found to be equally efficacious and have a more desirable safety profile.11

Lidocaine, Epinephrine, and Tetracaine

The LET (lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5%) solution should be prepared in single-use 5-mL vials.10 It may be applied to the wound directly as drops from a syringe, and then the remainder of the solution is soaked into a cotton ball or gauze, which is then held against the wound. The individual applying the LET should use gloves in order to prevent topical absorption to the caregiver. The LET solution should be applied for at least 20 to 30 min to achieve adequate anesthesia.11 Contact with mucous membranes, fingers and toes, the ear pinna, the penis, and the tip of the nose should be avoided. It also can be used in a gel form by adding 150 mg of methylcellulose to 3 mL of LET solution. The gel may be applied directly to the wound with a cotton applicator and irrigated off after 20 min. The gel form is more likely to remain in the wound and may be associated with a higher rate of complete anesthesia.

Eutectic Mixture of Local Anesthetics (EMLA)

The EMLA cream contains lidocaine 2.5% and prilocaine 2.5%.10,12 The EMLA cream has been shown to be effective in relieving the pain associated with venipuncture, port access, arterial puncture, lumbar puncture, and superficial skin procedures. It has been investigated as an anesthetic for open wounds; however, the available preparation is not sterile, and its use is presently recommended only for application on intact skin. Systemic reactions are rare; however, the prilocaine may cause methemoglobinemia, particularly in children younger than 3 months. The EMLA cream should be used sparingly and with caution in infants younger than 3 months (1 g maximum dose for 1 h) and avoided in patients predisposed to methemoglobinemia. EMLA cream in adults is applied by squeezing a dollop of about 2g directly onto the skin and covering the area with an occlusive dressing; the cream layer should be left thick and not massaged or rubbed into the skin. The depth of anesthesia is related to the duration of contact, with satisfactory analgesia achieved at 1 h, peak effect at 2 h, and duration for 1 h after removal.

Lidocaine

Lidocaine is available in solution, ointment, cream, and jelly preparations in concentrations from 1 to 30%. It is commonly used to facilitate the placement of urinary catheters, nasogastric tubes, and the passage of oral and nasal fiberoptic scopes. Two percent viscous lidocaine in an oral solution has been used to provide temporary relief of stomatitis, but the patient must be cautioned to use the solution sparingly (not to exceed 1 teaspoon every 3 h) and expectorate the excess anesthetic. Lidocaine 4% solution applied intranasally with a disposable atomizer has been shown to reduce the discomfort of nasogastric tube placement, even in addition to lidocaine jelly.13 Plasma levels after topical mucosal applications are similar to those associated with intravenous use, and doses should not exceed 4.5 mg/kg.

The anesthetic ELA-Max is a 4% liposomal lidocaine cream.12 The lidocaine is encapsulated in liposomes, which enhances the rate and amount of absorption in intact skin and resists rapid metabolism, thereby prolonging its effects. Compared with EMLA, the onset is approximately twice as fast and does not require occlusion.12 Similar to EMAL, ELA-Max should be applied in a thick layer (like cake frosting) and should not be rubbed into the skin. It is not recommended for mucosal application.

Other Topical Anesthetic Agents

A number of topical anesthetic combinations have been studied, mainly in comparison with TAC, and have demonstrated different degrees of efficacy. These preparations include prilocaine, tetracaine, or bupivacaine in combination with a vasoconstrictor, such as phenyl-ephrine. It is unclear whether any of these have significant advantages over LET.

Benzocaine is available as a 20% liquid, gel, or spray formulation for mucosal anesthesia. It is used to relieve the pain from oral ulcers, wounds, and inflammation and to facilitate the passage of oral nasogastric tubes and endoscopy. Benzocaine rarely may precipitate methemoglobinemia. A topical combination of antipyrine and benzocaine is used to temporarily relieve the pain of otitis media or externa.

Iontophoresis is a method of delivering a topical anesthetic with a mild electric current. The device is bulky and may cause an uncomfortable electrical sensation. Presently, it has limited use in the emergency setting.

Ethyl Chloride

Ethyl chloride is not an LA but rather a skin refrigerant or vapo-coolant delivered by a spray. Upon contact with the skin, it vaporizes, causing a transient drop in temperature to = 20°C, temporarily freezing it, and causing anesthesia through nerve desensitization. It is useful for venipuncture, injections, and incisions of small abscesses.

Ethyl chloride is applied by inverting the bottle at a distance of 10 to 30 cm from the skin and spraying for 3 to 7 s. The affected area will turn white. Anesthesia is present for only 30 to 60 s. Prolonged spraying could cause chemical frostbite and skin ulceration, and it should not be used on mucosal surfaces. Ethyl chloride should not be inhaled, because it may cause general anesthetic and opioid effects. It is also flammable.

REGIONAL ANESTHETIC PROCEDURES

Regional anesthetic procedures performed in the ED include peripheral nerve blocks, hematoma blocks, and intravenous (Bier's) blocks. In addition to their utility during invasive procedures, these techniques can minimize opiate use and decrease the need for procedural sedation or general anesthesia. The safety and effectiveness of LA for regional anesthetic procedures depends on proper dosage, correct technique, adequate precautions, and preparations to deal with emergencies, if they arise. Regional procedures with LA should be employed only by clinicians who are well-versed in diagnosis and management of LA dose-related toxicity and other acute emergencies that might result from inadvertent systemic absorption of the LA or intra-arterial injection of an epinephrine-containing LA solution. The setting should have resuscitative measures immediately available, if necessary. LA should be administered in the lowest dosage that results in an effective block. Epinephrine can be added to some blocks to enhance the duration, quality, efficacy, reliability, and safety of a block. However, epinephrine is contraindicated in nerve blocks in end-arterial areas except, perhaps, digital nerve blocks.

Peripheral Nerve Blocks

Peripheral nerve blocks are advantageous in the ED environment, particularly for procedures on the digits, hand, or foot. They require less total LA medication, and the site of drug delivery often is less painful

than that for local infiltration.14 It is imperative to document neurovascular status before application of the block, to prevent masking a primary traumatic neurovascular injury. During needle insertion, if the patient reports paresthesia in the nerve distribution, that sensation can be taken as a sign of proper localization for LA injection. The onset of anesthesia with peripheral nerve blocks is more delayed than by direct infiltration and may be up to 15 min. Lidocaine 1% or bupivacaine 0.25% can be used to achieve peripheral nerve blockade (see Table 37-3). The duration depends on the agent used and the amount of drug injected. Complications of nerve blocks include nerve injury and systemic LA toxicity. During injection, severe pain suggests that contact has been made with the nerve. In this circumstance, the needle must be withdrawn and repositioned before anesthetic is injected. Intravascular injection can result in limb and systemic toxicity. Local anesthetic dose exceeding the maximum may result in systemic toxicity, primarily cardiac and CNS. To minimize the likelihood of intravascular injection, the plunger on the needle syringe apparatus should be drawn back in all nerve block procedures before infiltration; if blood is withdrawn, the needle should be repositioned. Total doses of the agent should be calculated in all cases and doubled-checked against the concentration to prevent inadvertent overdosing of a specific LA available in more than one concentration (see Table 37-1).

Wrist Blocks

MEDIAN NERVE

The median nerve provides sensation to the lateral two thirds of the palm of the hand,

palmar surfaces of the lateral three and one half digits, and their fingertips. The palmar branches of the median digital nerves extend dorsally over the digit to supply the dorsum of the thumb, the index finger, the middle finger, and lateral half of the ring finger distal to the proximal interphalangeal joint and including the nail and the nail bed (Figure 37-1). The median nerve enters the hand through the carpal tunnel, deep to the flexor retinaculum, between the tendons of the flexor digitorum superficialis and the flexor carpi radialis.

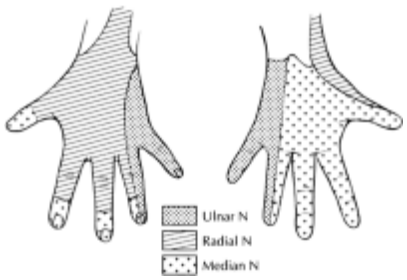


FIG. 37-1. Sensory distribution of the major nerves of the hand.

For lacerations of the hand, regional blocks at the wrist are performed at the level of proximal volar skin crease (Figure 37-2). The median nerve is anesthetized by inserting a 27-gauge needle perpendicular to the skin between the tendons of the palmaris longus and flexor carpi radialis muscles at the midpoint of the distal volar crease and injecting 3 to 5 mL of the LA solution with epinephrine (1:100,000).14

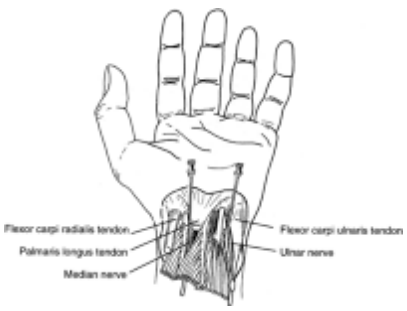


FIG. 37-2. Palmar view of the left hand demonstrating sites for median and ulnar nerve blocks at the wrist.

ULNAR NERVE

The ulnar nerve can be blocked at the elbow or the wrist to provide anesthesia to the medial aspect of the hand and the small finger, including its nail and nail bed (see Figure 37-1). Just proximal to the wrist, the ulnar nerve gives off a palmar cutaneous branch,

which passes superficially to the flexor retinaculum and palmar aponeurosis to supply the skin of the ulnar side of the palm. It also gives off a dorsal cutaneous branch that supplies the ulnar half of the dorsum of the hand, the small finger, and the ulnar half of the ring finger. The ulnar nerve ends by dividing into a superficial and a deep branch. The superficial branch supplies cutaneous fibers to the anterior surfaces of the small finger and the ulnar half of the ring finger. In the small finger, the dorsal digital nerve extends to the tip of the digit.

A regional block of the ulnar nerve at the wrist is accomplished by passing a 27-gauge needle between the ulnar artery and the flexor carpi ulnaris at the level of the proximal volar skin crease (see Figure 37-2). Alternatively, the needle can be inserted underneath the flexor carpi ulnaris on the ulnar side of the wrist and directed toward the radial side. Once the needle is positioned and especially if the patient reports paresthesia, 5 to 7 mL of LA solution with epinephrine (1:100,000) is injected slowly.14

RADIAL NERVE

The radial nerve provides sensation to the lateral two thirds of the dorsum of the hand; the proximal aspect of the dorsum of the thumb, index, and long finger; and the lateral aspect of the dorsum of the ring finger, excluding the nails and nail beds of these digits (see Figure 37-1). The superficial branch of the radial nerve is the direct continuation of the radial nerve along the anterolateral side of the forearm and is entirely sensory.

The superficial rami of the radial nerve can be blocked by raising a subcutaneous wheal with 5 to 10 mL of LA solution with epinephrine (1:100,000), beginning at the level of the tendon of the extensor carpi radialis and extending around the dorsum of the wrist to the styloid process, a distance of about 6 to 8 cm (Figure 37-3).14

FIG. 37-3. Radial view of the left hand demonstrating location for radial nerve block at the wrist.

Digital Blocks

DIGITAL NERVE BLOCK

The digital nerve block provides excellent anesthesia for fingers and has a more rapid onset than the metacarpal block. It is commonly used for laceration repair, incision and drainage of paronychia, and finger or toenail removal or repair. Preparation with EMLA or ethyl chloride can minimize the pain of injection for these blocks. Epinephrine traditionally has been avoided for digital blocks, although there is no evidence to support this practice.5 Contraindications to performing this block are any compromise to the digits' blood supply. Complications are few, but large volumes of anesthetic can result in a compartment syndrome. Each digit is supplied by a palmar (volar) and a dorsal digital nerve on each side of the digit, superficial to the digital arteries (Figure 37-4A).

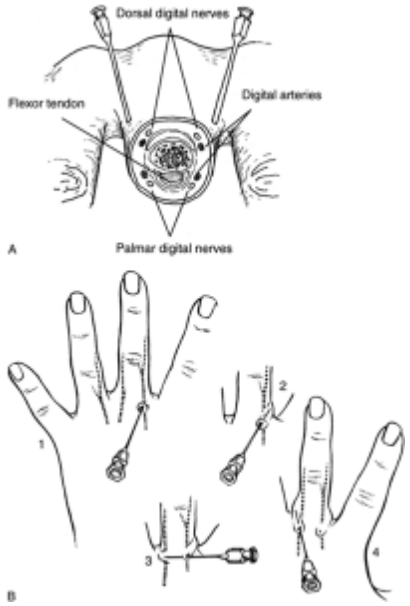


FIG. 37-4. A. Digit cross-section showing location of digital nerves. **B**. Needle positions for digital nerve block. **1**. Initial injection to block dorsal digital nerve. **2**. Needle is advanced to block palmar digital nerve on same side. **3**. Needle is directed across dorsum of digit to block dorsal digital nerve on opposite side. **4**. Needle is inserted to complete block of palmar digital nerve on opposite side of initial injection.

A 27- or 30-gauge needle is inserted through the skin into one side of the extensor tendon of the affected finger just proximal to the web (see Figure 37-4B, 1). After aspirating, approximately 1 mL of LA solution is injected superficially into the subcutaneous tissue lying on the dorsal surface of the extensor tendon to block the dorsal digital nerve. The needle is then advanced toward the palm until its tip is palpable beneath the volar skin at the base of the finger, just distal to the web (see Figure 37-4B, 2). After aspirating, another 1 mL of the anesthetic solution is injected to block the palmar digital nerve. Before removing the needle, it is redirected across the extensor tendon to the opposite side of the finger, and approximately 1 mL of the anesthetic solution is injected into the tissue overlying the other dorsal digital nerve (see Figure 37-4B, 3). Five minutes later, the needle is reintroduced in the anesthetized skin on the opposite side of the finger and advanced to block the palmar digital nerve on that side of the digit (see Figure 37-4B, 4). The total volume of the anesthetic agent should not exceed 4 mL.

METACARPAL BLOCK

Metacarpal blocks can be used to anesthetize the index, long, ring, or small finger, although digital blocks are preferred. The block is performed on each side of the affected

finger by inserting a 27-gauge needle at a 90-degree angle to the dorsum of the hand approximately 1 cm proximal to the metacarpophalangeal joint midway between each metacarpal bone. The needle is then advanced at a 90-degree angle to the skin until its tip is at the level of the lateral volar surface of the metacarpal head or until resistance of the palmar aponeurosis is detected. After aspirating, 3 mL of LA solution is injected slowly.14

Toe Blocks

Digital nerve blocks of the toe are used for laceration repair and minor surgical procedures of the toes. Epinephrine is avoided as an adjunct to lidocaine, because of the largely theoretical risk of irreversible ischemic injury. For the lesser toes, a 27- or 30-gauge needle should be introduced through the skin on the dorsal aspect of the base of the midpoint of the involved toe (Figure 37-5B). The needle should be angled around the bone until it induces blanching of the skin on the plantar surface. As the needle is withdrawn, approximately 1.5 mL of LA solution is injected. Before the needle is withdrawn completely from the skin, it should be redirected to the opposite side of the injured toe to inject the LA agent in a similar manner. The total volume of the injected LA agent should not exceed 3 mL.

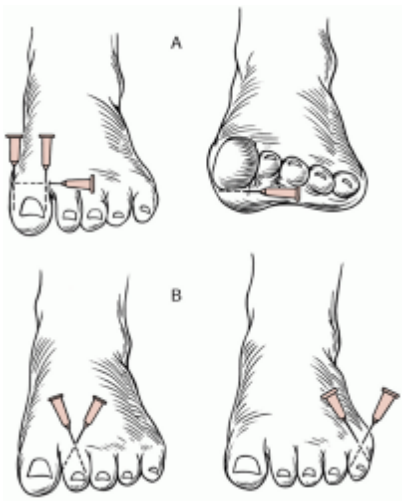


FIG. 37-5. A. Regional block of the great toe using collar infiltraton technique. **B**. Digital nerve block of the lesser toes.

For the hallux (great toe), a modified collar (ring) block is used (see Figure 37-5A) because of the frequency of occurrence of accessory nerves over the dorsum of the great toe. The 27-gauge needle is inserted through the skin on the dorsolateral aspect of the base of the toe until it blanches the plantar skin. As the needle is withdrawn, 1.5 mL of LA solution is injected into the tissues. Before the needle is removed completely from the skin, the needle is passed under the skin on the dorsal aspect of the toe, and 1.5 mL of LA solution is injected as the needle is withdrawn from the skin. The needle is then introduced through the anesthetized skin on the dorsomedial aspect of the toe and advanced until it produces blanching of the plantar skin, at which time the needle is withdrawn and 1.5 mL of LA solution is injected. To complete the collar block, the needle is inserted at the plantar and medial aspect and advanced to the lateral side until it blanches the skin, and then withdrawn as an additional 1.5 mL of LA solution is injected. Approximately 6 mL of LA

solution is needed to anesthetize the hallux.15

Foot Blocks

These regional nerve blocks are used for anesthesia of surgical procedures of the foot. There are five nerves that supply sensation to the foot. Most foot blocks involve a block of at least two nerves; it is unusual in the ED setting to need to block the whole foot. The sole of the foot is a commonly injured area, and local infiltration directly into the sole is extremely painful, difficult to perform effectively, and is not recommended. Regional nerve blocks are the preferred LA technique. Buffered lidocaine 1% and bupivacaine 0.25% are the LA agents of choice. Epinephrine is contraindicated, and these blocks should not be used in patients with peripheral vascular disease or traumatic circulatory compromise. All peripheral nerves involved in blocks of the foot

P.271

are branches of the sciatic nerve, except for the saphenous nerve (a branch of the femoral nerve; Figure 37-6).

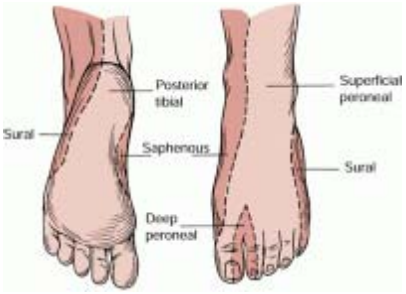


FIG. 37-6. Sensory innervation of the foot and ankle.

The sensory innervations of the plantar surface of the foot are primarily the two main branches of the tibial nerve (posterior tibial and sural nerves) and a small contribution from the saphenous nerve medially. The sensory innervations of the dorsum of the foot are predominantly from the two main branches of the common peroneal nerve (the superficial and deep peroneal nerves), with contribution from the sural nerve laterally and the saphenous nerve medially. The saphenous nerve is the only branch of the femoral nerve below the knee. It becomes subcutaneous at the medial side of the knee joint and then follows the saphenous vein to a site anterior to the medial malleolus. It provides sensory innervation to the skin over the medial malleolus and extends to the skin over the medial side of the foot to the base of the great toe. The superficial peroneal nerve becomes the dorsal digital nerves. It descends toward the ankle in the lateral compartment, entering the ankle just lateral to the extensor digitorum longus, and provides the cutaneous supply to the dorsum of the foot and all five toes, except for the adjacent sides of the first and second toes (deep peroneal nerve) and lateral side of the fifth toe (sural nerve; see Figure 37-6). It also supplies the peroneus longus and brevis muscles. The sensory supply of the

deep peroneal nerve is limited to the 1-cm area of web space between the first and second toes. Thus, blocks of the deep peroneal nerve are not reasonable or practical to perform.15

The three nerves that supply the sole of the foot are the posterior tibial nerve (most of the sole and heel), the sural nerve (posterolateral sole), and the saphenous nerve (small area, medially over the arch; see Figure 37-6). The posterior tibial nerve is located along the medial aspect of the ankle, lying between the medial malleolus and the Achilles tendon, just posterior and slightly deeper than the posterior tibial artery (Figure 37-7A) and gives off the three terminal branches, the medial calcaneal branch and the medial and lateral plantar nerves. The sural nerve travels with the short saphenous vein, posterior and inferior to the lateral malleolus; it terminates as the dorsal lateral cutaneous nerve. The saphenous nerve follows the great saphenous vein to the medial malleolus.

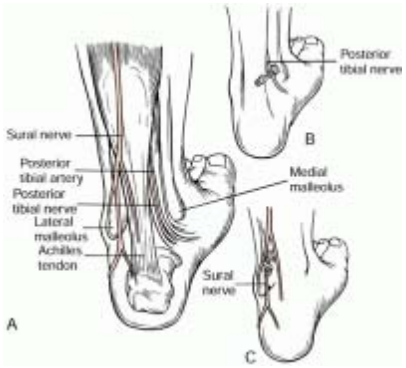


FIG. 37-7. Posterior view of the left foot demonstrating sites for posterior tibial and sural nerve blocks.

POSTERIOR TIBIAL NERVE BLOCK

A posterior tibial nerve block is best performed with the patient in the prone position. The posterior tibial nerve is blocked by injecting the LA solution between the posterior tibial artery and Achilles tendon, at the level of the upper border of the medial malleolus (see Figure 37-7B).15 Careful aspiration is necessary to ensure no inadvertent intravascular access. Paresthesia upon needle insertion is a sign of correct placement, and adequate block is achieved with 3 to 5 mL of LA solution. If no paresthesia is encountered, then 5 to 7 mL of LA solution should be injected as the needle is withdrawn. Onset of anesthesia should occur within about 5 to 10 min if paresthesia has been elicited and in about 30 min if it has not.

SURAL NERVE BLOCK

The sural nerve is blocked between the lateral malleolus and the Achilles tendon (see Figure 37-7). It is superficial, lying just anterior to the short saphenous vein, and is blocked by superficially injecting 3 to 5 mL of LA solution in a fanlike distribution just lateral to the Achilles tendon 1 cm above the lateral malleolus.15

SAPHENOUS NERVE BLOCK

The saphenous nerve lies superficially between the medial malleolus and the anterior tibial tendon (Figure 37-8) and is blocked anteriorly by infiltration of 3 to 5 mL of LA between these landmarks as the needle is withdrawn.15

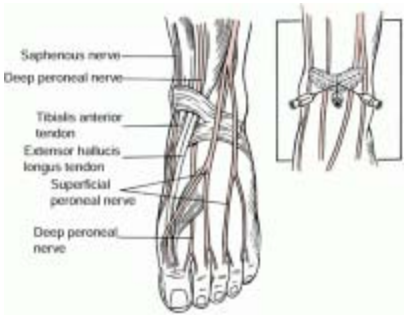


FIG. 37-8. Anterior view of the left foot demonstrating sites for superficial peroneal, saphenous, and deep peroneal nerve blocks.

P.272

PERONEAL NERVE BLOCKS

Regional block of the dorsum of the foot has fewer ED applications than do blocks for the sole of the foot, because direct infiltration of the dorsum is more easily performed. The superficial peroneal nerve primarily supplies the sensory innervation of the dorsum of the foot, with contributions from the deep peroneal nerve over the first web space, the sural nerve laterally extending to the lateral malleolus, and the saphenous nerve extending medially over the arch and the medial malleolus (see Figure 37-8).

Peroneal nerve block is best performed with the patient in the supine position. With a 30-gauge needle, a small wheal of LA is raised just above the level of the talocrural joint in the midline anteriorly, between the extensor digitorum longus and the extensor hallucis longus. The superficial peroneal nerve is then blocked by infiltration of 3 to 5 mL of buffered LA solution in a large wheal extending from this point just superior to the talocrural joint at the anterior border of the tibia to the lateral malleolus (see Figure 37-8).15 The deep peroneal nerve can be blocked by infiltrating 5 mL of buffered LA solution between the tendons of the tibialis anterior and the extensor hallucis longus just above the talocrural joint.15 However, the area supplied by the deep peroneal nerve is so small that a digital block or local infiltration would achieve the same effect.

Facial and Oral Blocks

Facial blocks are ideal anesthesia techniques for commonly injured areas such as the forehead, chin, lips, nose, tongue, and ear, where local infiltration is often not possible, extremely painful, or results in tissue distortion or potential tissue necrosis. These blocks, similar to foot blocks, often require blockade of more than one nerve to provide for

adequate regional anesthesia. For all intraoral routes of infiltration, a small amount of 2% viscous lidocaine should be applied to the mucosa before injection. The needle should not be inserted to its full length during an intraoral procedure nor should the direction of the needle be changed while it is deep in tissue; if inadvertent breakage occurs, retrieval may be difficult. Injection should be slow to minimize pain, and careful aspiration should occur before any injection.16,17 For percutaneous routes of infiltration, topical EMLA cream, topical ELA-Max cream, or refrigerant sprays should be applied before injection.

FOREHEAD

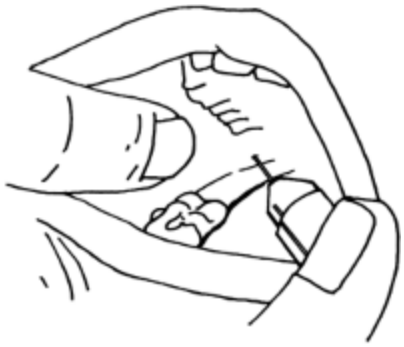
The sensory innervation of the forehead (anterior aspect from eyebrows extending posteriorly to the lambdoid suture) is supplied by the lateral and medial branches of the frontal (or supraorbital) nerve, the supratrochlear nerve, and fibers from the ophthalmic branch of the trigeminal nerve (Figure 37-9). Regional nerve block of the forehead can be achieved by infiltration of 3 to 6 mL of LA solution with a 27-gauge needle into the skin immediately above the full length of the eyebrow.16,17

FIG. 37-9. Regional block of the frontal nerve above the eyebrow.

LOWER LIP AND CHIN

Direct infiltration to the lip is very painful and causes tissue distortion, which can interfere with the quality of the repair of lacerations. The skin of the chin and lower lip is supplied by the mental nerve (branch of the inferior alveolar nerve). A block of the inferior alveolar nerve as it enters the mandibular foramen, medial and just behind the anterior border of the ramus of the mandible, is performed by the intraoral route (Figure 37-10). Block of the mental nerve can be performed by an intraoral or extraoral percutaneous route (Figure 37-11). The mental foramen is located at the mucosal reflection of the lower lip and gum, inferior to the second premolar (tooth number 20 or 29; see Chap. 242).

FIG. 37-10. Intraoral approach for inferior alveolar and lingual nerve blocks.



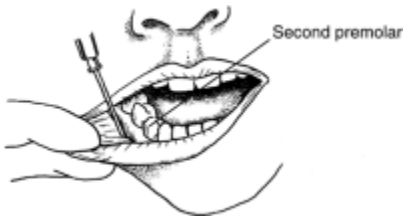


FIG. 37-11. Intraoral approach for mental nerve block.

The landmark for the inferior alveolar nerve block is the medial surface of the mandibular ramus posterior to and 1 cm above the occlusal surface of the third molar. The physician first identifies the anterior border of the ramus and the oblique alveolar ridge by palpation with the tip of the thumb. The buccal tissues then can be pulled laterally away from the mandible by the pad of the thumb, with the index finger placed externally behind the ramus to stabilize the mandible. The mucosal injection site, 1 cm above the occlusal surface of the third molar, should be anesthetized with a topical agent. A 27-gauge needle is directed from the other side of the mouth, typically between the opposite first and second premolars, and inserted slowly until the needle point touches the bony surface of the medial surface of the ramus (see Figure 37-10). The needle is withdrawn slightly, and 1 to 2 mL of LA solution (with or without epinephrine) is infiltrated.16

For the intraoral approach to the mental nerve, the lip is retracted with the thumb and index finger, and topical anesthetic is applied to the mucosa (see Figure 37-11). A 27-gauge needle is inserted at the mucosal junction of the lower lip and gum beneath the second premolar, and 1 to 2 mL of LA solution (with or without epinephrine) is infiltrated while taking care that the needle is not introduced into the mental foramen to avoid neural injury. For the extraoral percutaneous

approach, the mental foramen is located, and percutaneous infiltration of 2 to 4 mL of LA

solution close to that location is done.16,17 The intraoral approach is less painful than the percutaneous approach.

TONGUE

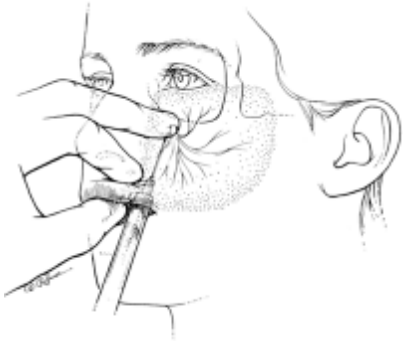
Direct infiltration into the sensate tongue is painful and poorly effective, and a regional block is preferred. The lingual nerve provides sensory innervation to the anterior two thirds of the tongue, the floor of the mouth, and gums. It lies in close proximity to the inferior alveolar nerve at the entrance to the mandibular foramen. The lingual nerve can be blocked by the intraoral route similarly to that for the inferior alveolar nerve (see Figure 37-10).

From an intraoral approach, the vertical ridge of the anterior border of the ramus of the mandible is identified by palpation with the index finger. After the mucosal injection site is anesthetized topically, the procedure for an inferior alveolar nerve block (same as above) is followed. Infiltration of the LA solution as the needle is withdrawn will anesthetize the lingual nerve. Alternatively, the lingual nerve can be anesthetized by injecting 2 to 3 mL of LA solution into the lateral floor of the mouth adjacent to the premolar teeth.16

CHEEK, LOWER EYELID, UPPER LIP, AND LATERAL ASPECT OF THE NOSE

The infraorbital nerve supplies sensory innervation to the cheek, lower eyelid, upper lip, and lateral aspect of the side of the nose. A regional block of the infraorbital nerve can be performed by an intraoral approach (Figure 37-12) or extraoral percutaneous approach. The duration of action is more prolonged with the intraoral approach. To identify the infraorbital foramen, the midpoint of the lower margin of the orbit is palpated; approximately 1 cm inferior to this point, the infraorbital nerve exits the infraorbital foramen. For the intraoral approach, a palpating finger is positioned over the infraorbital foramen (see Figure 37-12). The cheek is retracted cephalad with the thumb and index finger, and a 27-gauge needle with syringe, held in the other hand, is directed through the mucosa at the reflection of the upper gum opposite and parallel to the long axis of the upper premolar tooth. The needle is then advanced until it is palpated near the infraorbital foramen, approximately a depth of 2.5 cm.16,17 Caution should be used so as not to introduce the needle directly into the infraorbital foramen, to avoid neural injury. Also, caution should be used so as not to direct the needle too far superiorly or posteriorly, to avoid inadvertently entering the orbit. The syringe should be aspirated to ensure that the facial artery and vein are avoided. Then 2 to 3 mL of LA solution is instilled adjacent to the foramen. The extraoral (percutaneous) approach uses the same landmarks for identification of the infraorbital foramen. Epinephrine is best avoided due to the proximity of the facial artery.16

FIG. 37-12. Intraoral approach for infraorbital nerve block.



NOSE

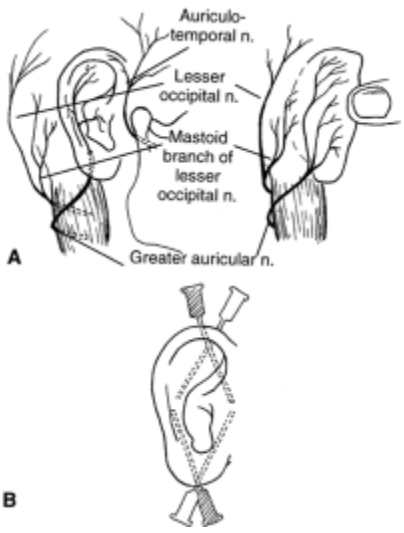
The ophthalmic and maxillary branches of the trigeminal nerve also supply the sensory supply of the nose; block of the infraorbital nerve alone will not provide adequate anesthesia of the nose. The mucosal surface of the nose can be anesthetized by topical application of LA spray or gel. The ophthalmic branch of the trigeminal nerve (infratrochlear and external nasal nerves) provides sensation to the majority of the external nose in the midline. These nerves can be blocked by percutaneous infiltration of LA at the sites of their emergence from bony foramina. The remaining aspects of the nose are supplied by the maxillary branch of the trigeminal nerve, the infraorbital nerve for the lateral aspect (see above for intraoral and extraoral block technique), and the posterior nasal and nasopalatine nerves for the septum and inferior midline. The posterior nasal and nasopalatine nerves are best approached intraorally in the midline from the mucosal surface of the reflection of the upper lip.

EAR

The sensory innervation to the external ear is supplied anteriorly by the auriculotemporal nerve (mandibular branch of the trigeminal nerve) and posteriorly by the greater auricular nerve and the mastoid branch of the lesser occipital nerve (branches of the cervical plexus; Figure 37-13A). Direct infiltration of the pinna should be avoided due to the risk of tissue necrosis. Regional block of the ear is

achieved by infiltration of LA solution via a 27- or 30-gauge needle at the base of the ear from an inferior and superior direction, anteriorly and posteriorly (see Figure 37-13B).

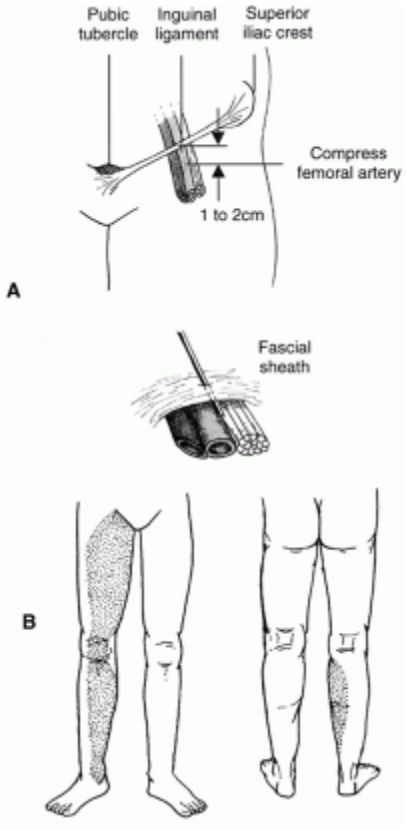
FIG. 37-13. A. Sensory nerve supply to the auricle. **B**. Technique for regional anesthesia of the auricle.



Femoral Nerve Block

Femoral nerve block is an effective technique for relieving pain of a femoral shaft fracture and is useful in the multiple trauma patient when minimizing opiates is important. The femoral nerve is lateral to the femoral artery at the inguinal ligament and innervates the anterior thigh, the periosteum of the femur, and the knee joint (Figure 37-14). Bupivacaine 0.25 to 0.5% is suggested as the preferred LA agent, because of its longer duration of action. A sterile field is prepared over and surrounding the femoral triangle. The femoral artery is located midway between the anterior superior iliac crest and the pubic tubercle. The femoral artery is compressed 1 to 2 cm below the inguinal ligament with the nondominant hand. A wheal of LA is raised in the skin and subcutaneous tissues lateral to the artery; the needle is then introduced at an angle 45 to 60 degrees to the skin lateral and parallel to the femoral artery and directed cephalad. A double loss of resistance, or “pop,” is felt as the needle traverses the fascia overlying the femoral nerve. Ten to 20 mL of LA without epinephrine is slowly injected. Onset of anesthesia is 10 to 20 min, and the duration is 3 to 8 h.

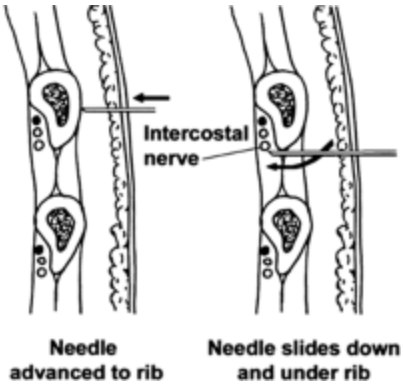
FIG. 37-14. A. Location for femoral nerve block. **B**. Sensory innervation for the femoral nerve (shaded area).



Intercostal Block

Intercostal block is valuable for the management of pain after chest trauma (typically rib fracture) or discomfort from a chest tube. It is a simple block; however, caution should be exercised due to the rapid and high systemic absorption of LA at this site. Contraindications are local soft tissue disease and contralateral pneumothorax. Complications include pneumothorax and systemic toxicity. The intercostal nerves run anteriorly within the neurovascular bundle along the inferior portion of the rib. Addition of epinephrine enhances the safety of this block. The LA dosage range for an adult is 3 to 5 mL per segment (pediatric dosage, 1 to 3 mL). To perform this procedure, the landmarks are the palpable intercostal spaces and the midaxillary line. The intercostal space is identified by palpation. On the upper margin of this space, the inferior border of the upper rib is palpated. At the mid or posterior axillary line, the needle is inserted and advanced at a 90-degree angle until the rib is reached. The needle is withdrawn slightly and redirected caudally to the inferior aspect of this upper rib (Figure 37-15).

FIG. 37-15. Intercostal nerve block.



HEMATOMA BLOCKS

Hematoma blocks are a simple, quick, and effective mode of regional anesthesia for isolated closed fracture reduction. Although hematoma blocks are safe, the anesthesia that they provide is not as efficacious as the intravenous regional (Bier's) block.18 The hematoma block is a useful alternative when the intravenous block is contraindicated.

Superficial anesthesia is obtained with local infiltration or other technique such as EMLA applied to the skin over the fracture site. An analgesic dose of opiate also can be given, if deemed appropriate. The fracture site is identified and, using sterile technique, the hematoma is aspirated with a 10-mL syringe and a 20- to 22-gauge needle. Lidocaine 1% without epinephrine is infiltrated to a dose of 3 to 10 mL into the fracture cavity and around the periosteum. The block is effective within 5 to 10 min, with several hours' duration.

INTRAVENOUS REGIONAL BLOCK (BIER'S BLOCK)

The intravenous regional block is an anesthetic procedure involving intravenous infusion of LA distal to an inflated pneumatic tourniquet.19 It is useful for fracture reductions, large laceration repairs, and foreign body removal. Duration of regional anesthesia is 30 to 60 min. In addition to routine monitoring and resuscitation equipment, the procedure requires a double-cuff tourniquet with a constant pressure gas source. A standard blood pressure cuff is not acceptable and can result in catastrophic systemic leakage of LA. Contraindications include peripheral vascular disease, Raynaud syndrome, sickle cell disease, cardiac conduction abnormalities, hypertension, cellulitis, and children

younger than 5 years. The need for patients to have nothing by mouth for 4 h before the procedure also may limit the usefulness of the technique. Intravenous regional blocks are used most commonly for upper extremity procedures. Less frequently, this technique has been applied to the lower limb in children.

Lidocaine (3 mg/kg or 0.6 mL/kg of 0.5% solution) without epinephrine is used. In Australia and the United Kingdom, prilocaine (3 mg/kg or 0.6 mL/kg of 0.5% solution) is used. Increased efficacy has been shown with the addition of intravenous ketorolac (60 mg) or fentanyl (1 µg/kg) to the lidocaine.20 Bupivacaine is absolutely contraindicated due to

cardiac toxicity. A “minidose” block with lidocaine (1 to 1.5 mg/kg) has been used effectively in children.

Vital signs and limb neurologic status and perfusion should be monitored and recorded. Intravenous access should be established in both upper limbs, distal to the fracture site on the affected limb. Standard resuscitation equipment and medications should be available. A small dose of intravenous opiate may allay apprehension and the pain of the cuff, which usually occurs after 15 to 30 min. The injured extremity is elevated, and an Esmarch bandage may be applied (distal to proximal) to exsanguinate the limb. Protective padding is then applied to the upper arm to minimize cuff discomfort. Pneumatic double cuffs are then positioned over the padding on the upper arm. The proximal cuff is inflated to 50 to 100 mm Hg above systolic pressure. A venous tourniquet may be positioned just proximal to the fracture site to contain the intravenous local anesthetic to this site. The lidocaine (or prilocaine) 0.5% solution is injected slowly over 2 min into the intravenous cannula in the affected limb. Mottling of the affected limb should occur within 2 to 5 min, followed by anesthesia and paresis of the limb, usually within 10 to 20 min. After 10 min, the distal cuff should be inflated (the area under this cuff should now be anesthetized); once the distal cuff is securely inflated, the proximal cuff can be deflated to minimize cuff pain. The distal cuff should not be deflated until at least 30 min has elapsed from the time of LA injection for tissue binding of the LA agent to occur and thus to minimize the potential for toxicity. Loss of tourniquet pressure before this time will result in a systemic bolus of LA and potential systemic cardiac and CNS toxicity. After completion of the procedure, deflation of the distal cuff should be cycled to prevent the bolus infusion of the LA into the systemic circulation. The cuff is deflated for 5 s, followed by inflation for 1 to 2 min. This cycling action should be repeated three to four times.

REFERENCES

1. Ruetsch YA, Böni T, Borgeat A: From cocaine to ropivacaine: The history of local anesthetic drugs. *Curr Topics Med Chem* 1:175, 2001.

2. Tagariello V, Caporuscio A, De Tommaso O: Mepivacaine: Update on an evergreen local anaesthetic. *Minerva Anesthesiol* 67(suppl 1):5, 2001.

3. Tetzlaff JE: The pharmacology of local anesthetics. *Anesthesiol Clin North Am* 18:217, 2000.

4. Metaxotos NG, Asplund O, Hayes M: The efficacy of bupivacaine with adrenaline in reducing pain and bleeding associated with breast reduction: A prospective trial. *Br J Plast Surg* 52;290, 1999.

5. Denkler K: A comprehensive review of epinephrine in the finger: To do or not to do. *Plast Reconstr Surg* 18:114, 2001.

6. Bartfield JM, Jandreau SW, Raccio-Robak N: A randomized trial of diphenhydramine vs. benzyl alcohol with epinephrine as an alternative to lidocaine local anesthesia. *Ann Emerg Med* 32:650, 1998.

7. Bartfield JM, May-Wheeling HE, Raccio-Robak N, Lai SY: Benzyl alcohol with epinephrine as an alternative to lidocaine with epinephrine. *J Emerg Med* 21:375, 2001.

8. Howell JM, Chisholm CD: Wound care. *Emerg Med Clin North Am* 15:417, 1997.

9. Knapp JF: Updates in wound management for the pediatrician. *Pediatr Clin North Am* 46:1201,1999.

10. Singer AJ, Stark MJ: EMLA versus LET for pretreating lacerations. A randomized trial. *Acad Emerg Med* 8:223, 2001.

11. Schilling CG, Bank DE, Borchert BA, et al: Tetracaine, epinephrine (adrenalin), and cocaine (TAC) versus lidocaine, epinephrine, and tetracaine (LET) for anesthesia of lacerations in children. *Ann Emerg Med* 25:203, 1995.

12. Eichenfield LF, Funk A, Fallon-Friedlander S, et al: A clinical study to evaluate the efficacy of ELA-Max (4% liposomal lidocaine) as compared with eutectic mixture of local anesthetics cream for pain reduction of venipuncture in children. *Pediatrics* 109:1093, 2002.

13. Wolfe TR, Fosnocht DE, Linscott MS: Atomized lidocaine as a topical anesthesia for nasogastric tube placement: A randomized, double-blind, placebo-controlled trial. *Ann Emerg Med* 35:421, 2000.

14. Gerancher JC: Upper extremity nerve blocks. *Anesthesiol Clin North Am* 18:297, 2000.

15. Dilger JA: Lower extremity nerve blocks. *Anesthesiol Clin North Am* 18: 319, 2000.

16. Simpson S: Regional nerve blocks. Part 2—The face and scalp. *Aust Fam Phys* 30:565, 2001.

17. Eaton JS, Grekin RC: Regional anesthesia of the face. *Dermatol Surg* 27:1006,

2001.

18. Furia JP, Alioto RJ, Marquardt JD: The efficacy and safety of the hematoma block for fracture reduction in closed, isolated fractures. *Orthopedics* 20:423, 1997.

19. Simpson S: Regional nerve blocks. Part 5—Bier's block (intravenous regional anesthesia). *Aust Fam Phys* 30:875, 2001.

20. Reuben SS, Steinberg RB, Kreitzer JM, Duprat KM: Intravenous regional anesthesia using lidocaine and ketorolac. *Anesth Analg* 81:110, 1995.