

Quality Resource Guide

Local Anesthetics

Author Acknowledgement

Clarence Trummel, DDS PhD

Emeritus Professor
School of Dental Medicine
University of Connecticut
Farmington, Connecticut

Dr. Trummel has no relevant relationships to disclose.

Educational Objectives

Following this unit of instruction, the learner should be able to:

1. Understand the relevant pharmacologic and clinical features of currently available local anesthetics used in dentistry.
2. Understand the rationale for using vasoconstrictors together with local anesthetics.
3. Understand the mechanisms by which local anesthetics and vasoconstrictors can produce adverse reactions and how such reactions can be avoided or minimized.
4. Appreciate the need to assess patient's risk for adverse reactions to local anesthetics and vasoconstrictors.
5. Be familiar with the factors that determine choice of a local anesthetic preparation and its route of administration in a particular patient.
6. Be familiar with the use and limitations of topical anesthetics in the oral cavity.

MetLife designates this activity for **1.0 continuing education credits** for the review of this Quality Resource Guide and successful completion of the post test.

The following commentary highlights fundamental and commonly accepted practices on the subject matter. The information is intended as a general overview and is for educational purposes only. This information does not constitute legal advice, which can only be provided by an attorney.

© Metropolitan Life Insurance Company, New York, NY. All materials subject to this copyright may be photocopied for the noncommercial purpose of scientific or educational advancement.

Originally published March 2005. Updated and revised July 2009, June 2012, June 2015 and May 2018. Expiration date: May 2021.

The content of this Guide is subject to change as new scientific information becomes available.

ADA CERP® | Continuing Education Recognition Program

MetLife is an ADA CERP Recognized Provider.

Accepted Program Provider FAGD/MAGD Credit **11/01/16 - 12/31/20**.

ADA CERP is a service of the American Dental Association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors, nor does it imply acceptance of credit hours by boards of dentistry.

Address comments or questions to:

DentalQuality@metlife.com

MetLife Dental Continuing Education
501 US Hwy 22, Area 3D-309B
Bridgewater, NJ 08807

Cancellation/Refund Policy:

Any participant who is not 100% satisfied with this course can request a full refund by contacting us.

Concerns or complaints about a CE provider may be directed to the provider or to ADA CERP at www.ada.org/goto/cerp.

Introduction

The use of local anesthetics to block pain safely and effectively is arguably the most essential component of modern dental care. The discovery of these drugs transformed dentistry from a dreaded and usually avoided ordeal to an experience that most patients readily tolerate. The oral health enjoyed by so many individuals today owes much to this important group of drugs.

The objective of this article is to review the properties, indications, adverse effects, and methods of delivery of local anesthetics currently used in dentistry. Practitioners must be well versed in all of these aspects in order to safely and effectively deliver anesthesia as well as provide sufficient information to patients so that they may give informed consent to treatment.

It should be noted that this review deals only with local anesthetics and does not address safety issues that arise when local anesthetics are used in conjunction with drugs for control of anxiety.

Chemical and Pharmacologic Properties

Local anesthetics are defined as drugs capable of producing absence of sensation by blocking nerve conduction. The extent of anesthesia is largely determined by the means of delivery. Topical application to skin or mucosa produces anesthesia only in the immediately adjacent tissue by blocking free nerve endings. Injection of the anesthetic into tissue will block pain in tissue at the immediate site as well as in distal structures innervated by the nerves traversing the site. Two other properties of local anesthetics are implicit by definition: there is no alteration of consciousness, i.e., the block is locally mediated at the site of drug administration; and the block is entirely reversible.

Most local anesthetics in use today are weakly basic tertiary or secondary amines. They have in common a structure consisting of a hydrophilic part (the substituted amine) and a lipophilic part. These parts are joined by an intermediate linkage that is either an ester or an amide, as shown in Figure 1. R₁ represents the lipophilic part and R₂ and R₃ are various substituents on the terminal amine.

The relative nature of these groups determines important chemical and biological properties such as solubility, stability, and anesthetic potency and toxicity. **Table 1** summarizes these characteristics for representative ester and amide anesthetics.

The nature of the intermediate linkage of a local anesthetic molecule determines the mode by which these drugs are biotransformed in the body. Ester-linked agents are largely inactivated

by plasma esterases, and the cleavage products eliminated in the urine. In contrast, most amides are degraded in the liver by both hydrolytic and oxidative reactions, and these various metabolites are excreted in bile and urine. Liver function thus becomes an important determinant of how rapidly amide local anesthetics are inactivated. An exception is the amide articaine, which is largely inactivated by a plasma carboxylesterase; less than 10% of the drug is metabolized in the liver.

Figure 1

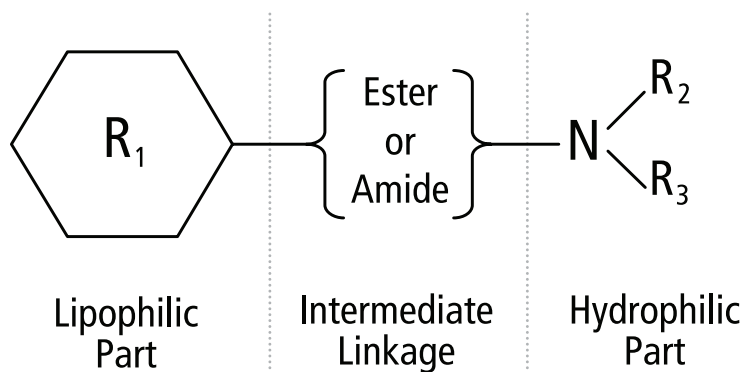


Table 1 - Chemical and Biological Characteristics of Local Anesthetics

Drug	Type	Lipid Solubility	Potency*	Topical Activity
Procaine	Ester	Low	Low	None
Tetracaine	Ester	High	High	High
Lidocaine	Amide	Medium	Medium	Intermediate
Mepivacaine	Amide	Medium	Medium	Low
Articaine	Amide	Medium	Medium	Low
Prilocaine	Amide	Medium	Medium	Low
Bupivacaine	Amide	High	High	N.D.

* Yagiella, JA. Local Anesthetics. In Yagiella, JA., Dowd, FJ., Johnson, B., Mariotti, A. and Neidle, EA. Pharmacology and Therapeutics for Dentistry, 6th ed., St. Louis: Elsevier Mosby, 2011.

N.D. = Not Determined

Since local anesthetics are weak bases, they exist in solution in two forms, charged (or ionized) and uncharged (or unionized). Consequently, the pH of the ambient fluid environment is a major determinant of the relative proportions of these two forms. When a local anesthetic is injected into inflamed tissue, where the pH may be as low as 5, a lesser proportion of the drug will exist in the uncharged form. As a result, the effectiveness of the drug in such an environment may be markedly reduced, since only the uncharged, or lipophilic, form can pass through nerve cell membranes to reach the site of action. In addition, the inflammatory process separately produces various substances that increase neuronal activity and inhibit local anesthetic activity.

Local anesthetics block nerve conduction by preventing depolarization of the neuron and thus preventing the propagation of an action potential. In brief, the uncharged local anesthetic passes into the neuron membrane, becomes protonated, i.e., charged, and binds to receptors within sodium channels. This binding prevents the inward flux of sodium in response to an action potential, which in turn prevents depolarization of the neuronal membrane and subsequent propagation of the action potential, i.e., nerve block. As the local anesthetic adjacent to the nerve is cleared and the intracellular concentration decreases, normal function of the sodium channels is restored and conduction resumes.

Within peripheral nerves, there is considerable variation according to the size and type of nerve fibers. Small-diameter fibers mediating pain and temperature sensation are blocked before larger myelinated nerves responsible for other sensory input and motor function.

Two important factors in the clinical application of local anesthesia are the onset and duration of anesthesia. These are primarily a function of the chemical nature of the drug and its effects on blood flow at the site of injection. Immediately following deposition of the anesthetic into tissue, the uncharged form can diffuse into an adjacent nerve to block conduction. The rate at which this occurs is determined by the amount of drug injected, the fraction in the uncharged form at tissue pH and

the inherent lipid solubility of this form. Thus, local anesthetics with greater lipid solubility and a lower pKa have proportionately faster onset due to more rapid uptake into the neuron. Highly lipid-soluble agents will also have longer duration of action due to greater binding and more prolonged retention within the neuron. At the same time the drug moves into cells, it also diffuses into adjacent small vessels and is removed from the site. The rate of clearance depends upon the inherent vascularity of the tissue and the degree to which the anesthetic may increase blood flow by vasodilation. Most local anesthetics used in dentistry cause some degree of vasodilation, increasing blood flow and more rapid clearance from the site. This not only shortens the duration of nerve block but also leads to a higher peak blood level and an increased risk of systemic toxicity.

The addition of a vasoconstrictor to a local anesthetic solution is a well-established strategy to increase duration of anesthesia and reduce systemic toxicity.¹ Both parameters are achieved by virtue of reduced blood flow in the area of injection; slower absorption means the drug enters the circulation more slowly, thus remaining at the site longer and decreasing the total dose required. Although vasoconstrictors have their own potential local and systemic adverse effects, the contribution to safe and effective local anesthesia normally justifies the risk. Epinephrine has been used for a century and remains the vasoconstrictor of choice. Other vasoconstrictors have been used (e.g., norepinephrine, phenylephrine and levonordefrin), but none are as efficacious.

Table 2 - Dosages of Local Anesthetics Used in Dentistry

Product	Amount of Drug Per Cartridge ^{†*}	Maximum Dose (mg/LB)	Maximum Total Dose (mg) [†]	Maximum No. Cartridges [§]	Epinephrine Total Dose (µg)
Mepivacaine 3%	Mepivacaine 54 mg	3	400	7.5	-
Mepivacaine 2% with levonordefrin 1:20,000	Mepivacaine 36 mg Levonorefrin 90 mcg	3	400	11	-
Lidocaine 2%	Lidocaine 36mg	2	300	8	-
Lidocaine 2% with epinephrine 1:50,000	Lidocaine 36 mg Epinephrine 36 mcg	3.2	500	5.5	198
Lidocaine 2% with epinephrine 1:100,000	Lidocaine 36 mg Epinephrine 18 mcg	3.2	500	11	198
Prilocaine 4%	Prilocaine 72 mg	4	600	8	-
Prilocaine 4% with epinephrine 1:200,000	Prilocaine 72 mg Epinephrine 9 mcg	4	600	8	72
Articaine 4% with epinephrine 1:100,000	Articaine 70 mg Epinephrine 18 mcg	3.2	500	7	119
Articaine 4% with epinephrine 1:200,000	Articaine 70 mg Epinephrine 9 mcg	3.2	500	7	63
Bupivacaine 0.5% with epinephrine 1:200,000	Bupivacaine 18 mg Epinephrine 9 mcg	Not Established	90	5	45

[†]Standard dental cartridge containing 1.8 ml, except articaine which contains 1.7 ml per cartridge.

* Amount of vasoconstrictor given as micrograms (mcg; 1 milligram = 1000 micrograms) to avoid fractional values.

[†] For Individuals greater than 150 lb.; if less than 150 lb., calculate dose based on weight.

[§] Based on maximum dose of anesthetic agent or maximum dose of epinephrine (200 mcg), whichever is lower.

The optimal concentration of epinephrine in local anesthetic solutions has long been debated. A concentration of 5 to 10 mcg/ml (or 1:200,000 to 1:100,000) provides a useful extension of the duration of action of such commonly used amide anesthetics as lidocaine, articaine and prilocaine with low risk of adverse effects. The additional benefit gained by higher concentrations of epinephrine, e.g., 20 mcg/ml (or 1:50,000) is seldom justified. Although a vasoconstrictor only marginally increases the duration of intrinsically long-acting agents, e.g., bupivacaine, epinephrine is still utilized with such agents to reduce the risk of toxicity.

In addition to increasing the duration of anesthesia, the slower rate of removal of drug from the site of injection resulting from a vasoconstrictor may also contribute to the safety of local anesthetics in two ways. First, by prolonging the period of anesthesia, a lower total dose of drug may suffice to complete the operative procedure. Second, slower removal from the site of injection decreases both the rate of rise of drug concentration in plasma as well as the peak concentration reached. This may allow the administration of somewhat greater total doses of anesthetic should it be necessary in extensive or lengthy procedures. In the interest of safety, however, the maximal doses of local anesthetics approved by the Food and Drug Administration with the concurrence of the ADA (**Table 2**) are, with the exception of lidocaine, independent of the presence of a vasoconstrictor.

Side Effects and Toxicity

The use of local anesthetics is enormous, and these drugs are among the most widely used in the world. It is indeed fortunate that the therapeutic importance of currently available local anesthetics is matched by an impressive record of safety. However, local anesthetics and the often present vasoconstrictors can cause serious adverse effects on the brain and cardiovascular system that may have a fatal outcome. It is unfortunate that the acknowledged safety of local anesthetics together with their daily routine use may lead the unwary practitioner to complacency and errors in drug selection or administration with adverse outcomes.

The most common reactions encountered in the delivery of local anesthesia occur in anxious patients and are psychogenic in nature, *i.e.*, a consequence of the administration of the drug rather than the drug itself. Psychogenic responses are usually manifested by nervousness and agitation, sweating, pallor, hyperventilation or fainting (vasodepressor syncope). It is essential for both prevention and management that the practitioner be able to distinguish these reactions from those resulting from the response to the injected drug or drugs themselves.

Localized reactions to local anesthetics at the site of injection are uncommon and may be difficult to distinguish from tissue injury resulting from needle penetration. However, prolonged neurosensory disturbances can occur following local anesthetic injection, suggesting a neurotoxic effect of the local anesthetic itself. A recent study indicated that trigeminal neurosensory disturbances following mandibular blocks were highly associated with the use of articaine 4%, compared to lidocaine, mepivacaine and prilocaine. Whether this finding was related to the chemical nature of these agents or a concentration effect is unknown.

Serious adverse systemic reactions to local anesthetics are also uncommon. The majority are due to either direct toxicity or allergic reactions.^{2,3} Toxic reactions are by definition due to an overdose of drug, and should be largely preventable.

Systemic toxicity occurs when the level of local anesthetic in plasma exceeds a certain level. Although all the injected dose ultimately appears in blood, several factors determine whether the level reached will be toxic. The most important of these are the total drug dose injected and the rate of uptake from the site of injection into blood.

These two parameters are inversely related; the slow uptake of a large dose may be less dangerous than rapid uptake of a smaller dose. Obviously, intravascular injection of a local anesthetic solution is equivalent to immediate transfer of the drug from the site into blood. Since most amide anesthetics (articaine is an exception) are largely metabolized in the liver, toxic levels of such agents may be

reached at lower doses of drug in patients with severely impaired liver function, and downward adjustment of dosage should be considered.

Just as local anesthetics suppress conduction in peripheral nerves, these agents similarly impair function in other excitable tissues. These include the cardiovascular and central nervous systems. The latter is more sensitive to the effects of local anesthetics than the former and will generally manifest signs and symptoms of toxicity first. The initial symptoms are tinnitus, blurred vision, dizziness, tongue paresthesias and circumoral numbness. With further rise in drug level, agitation, excitement, nausea, tremor and ultimately convulsions occur. If the drug level rises still higher, convulsions will cease, followed by unconsciousness and respiratory depression. Respiratory and cardiac arrest will occur with further increase in drug levels. It is important to note that previously existing hypoxia increases local anesthetic toxicity by lowering the plasma level that causes convulsions. In a similar dose-related fashion, these drugs also cause peripheral vasodilation and depress conduction in the myocardium both directly and through central nervous system depression, and may lead to circulatory failure and cardiac arrest.

A most important factor in reducing the risk of local anesthetic toxicity is determination of dosage according to body weight. Dosing guidelines based on weight have been established by clinical trial and experience for approved agents used in dental anesthesia (with the exception of bupivacaine) and should be followed. There are also recommended maximum total doses for each agent that should not be exceeded during a single dental treatment visit. For example, the recommended dose of prilocaine is 4 mg/lb body weight and the maximum total dose is 600 mg. This means that for patients weighing 50, 100, 150 or 200 pounds, the maximum doses would be 200, 400, 600 and 800 mg, respectively. Dosing guidelines specific for a particular agent should be applied to pediatric patients as well, unless otherwise specified by the manufacturer. For example, the recommended dose of articaine for patients over 12 years of age is 3.2 mg/lb.,

but for patients 4 to 12 years of age, is 2.3 mg/lb.; the dosage of articaine for patients less than 4 years of age has not been established. Practitioners should be familiar with and follow carefully the dosing guidelines packaged with every unit container of local anesthetic cartridges.

Strategies to minimize the peak plasma level of local anesthetics and reduce the risk of toxicity are straightforward and can be summarized as follows:

- Use the least toxic drug in the lowest dose that will yield the desired quality and duration of anesthesia.
- Use a preparation with a vasoconstrictor (unless contraindicated).
- Use good technique to deliver the solution in close proximity to the intended site of action and thereby minimize the amount of drug required.
- Avoid intravascular injection; aspirate before and several times during injection; aspirate if needle is repositioned; avoid 30 gauge needles in sites where aspiration is likely to be positive.
- Inject slowly - at least one minute to deliver the contents of a 1.8-ml cartridge; never inject a large volume rapidly.
- Use recommended dosage based on body weight (see **Table 2**).
- Do not exceed the recommended maximum dose (see **Table 2**).
- Consider reduction of dosage in elderly patients or those with serious liver disease.

Although uncommon, both anaphylactic and delayed-type allergic reactions to local anesthetics can occur. Most are associated with ester-type agents, such as procaine, tetracaine or benzocaine.

True hypersensitivity to amide-type agents is very rare, and there is no cross-reactivity between amide- and ester-type agents.² Patient reports of “Novacain allergy” are more frequent than true cases for several reasons. Patients may misinterpret vasodepressor syncope that occurs during or following local anesthetic administration as an allergic reaction. They may similarly misinterpret symptoms caused by anxiety or a vasoconstrictor, such as dizziness, tachycardia or palpitations,

as due to the anesthetic. Some patients have a true allergic hypersensitivity to sulfites added to local anesthetic solutions to prevent oxidation of epinephrine or levonordefrin, and may experience an allergic reaction that is wrongly attributed to the anesthetic itself. Another potentially antigenic substance, methylparaben, was formerly added as an antimicrobial to local anesthetic solutions but is no longer present in solutions designed for single use, such as dental cartridges. Finally, most cartridges have latex components, either the diaphragm, plunger or both, creating the possibility for reaction in a latex-sensitive patient. However, there are no reports of allergic reactions attributable to latex in dental anesthetic cartridges.

In spite of the extremely low incidence of true allergic hypersensitivity to amide-type anesthetics, practitioners must carefully evaluate all patient assertions of allergy before administration. Although skin testing is available, it does not rule out a possible reaction and in itself may risk a serious reaction. Assessment of possible local anesthetic allergy relies largely on careful analysis of the patient history. The patient’s description of signs, symptoms and duration of a past reaction to local anesthesia administration may provide clues. For example, did the reaction occur during injection or immediately after and consist of a brief episode of “shakiness” and tachycardia that was self-limiting and required no treatment? Did unconsciousness result, and if so for how long? Was there itching or flushing of skin, urticaria, wheezing or shortness of breath? Was the reaction treated and how; if so, when did it resolve? Does the patient have other known allergies, e.g., latex? Although certainty may not be reached, such queries will usually allow the clinician to judge the risk of administering local anesthesia. If there is reasonable doubt, anesthesia should be withheld and the patient should be referred for testing or for treatment in a medical setting.

Although epinephrine and other vasoconstrictors are not allergenic, they can, as noted above, cause adverse systemic effects. These include tremors, sweating, excitement, tachycardia, palpitation, rapid increase in blood pressure and

disturbances in cardiac rhythm. The magnitude of these reactions depends upon the peak blood level of the vasoconstrictor. At lower levels, effects are mild, transient and generally inconsequential, but at higher doses, the risk of cerebral hemorrhage or ventricular dysrhythmias increases sharply, particular in individuals with existing cardiovascular disease. Just as for local anesthetics, epinephrine overdose usually results from inadvertent intravascular injection or injection of a large dose over a very short period of time. For healthy adults, the total dose of epinephrine administered during a treatment session should not exceed 200 mcg (0.2 mg or ~11 cartridges of solution containing epinephrine 1:100,000). In the absence of specific guidelines, epinephrine should be used in lesser amounts or not at all in patients with a history of cardiovascular disease, particularly stroke and dysrhythmias. This vasoconstrictor should also be limited or avoided in patients with uncontrolled hyperthyroidism who may have increased sensitivity to catecholamines.

Selection of Local Anesthetics

Owing to the greater efficacy and lesser antigenicity of amide-type local anesthetics, the amino-esters are seldom utilized today. There are currently five amides supplied in single-use cartridges for dental use. Selection of these depends primarily on two factors: duration of anesthesia required and the patient’s medical status. As described above, duration is determined by the lipid solubility of the drug, concentration, total volume of solution given, presence of a vasoconstrictor and type of injection, infiltration versus nerve block (**Table 3**).

Lidocaine was the first amide introduced into dentistry and is still the most widely employed. It has a rapid onset of action, and when combined with epinephrine provides anesthesia of sufficient duration for most dental or soft tissue surgical procedures. Although lidocaine is available without epinephrine, its use in this form should be limited to situations where short duration of anesthesia is particularly important. Like all amide-type agents, lidocaine is inactivated in the liver and should be used cautiously in patients with impaired liver function.

Mepivacaine has been used for local anesthesia in dentistry for many years. It has a shorter duration of action than lidocaine with epinephrine, particularly in the absence of a vasoconstrictor, and has been advocated for brief procedures. The vasoconstrictor most commonly used with mepivacaine is levonordefrin (Neocobefrin™), a synthetic alpha-adrenergic amine. Levonordefrin is less potent than epinephrine and a much higher concentration is required for adequate vasoconstriction.

Articaine was approved in 2000 in the U.S. for local anesthesia. Initial reports that articaine penetrated alveolar bone to a greater extent and diffused more widely than other agents have not been substantiated. Its duration of action and other clinical properties are similar to lidocaine, and articaine offers no particular advantage. It is available only with epinephrine. Use in children under 4 years of age is not recommended.

The local anesthetic features of prilocaine are also similar to lidocaine. An advantage is lesser vasodilation, which permits use of a lower concentration of epinephrine. A drawback is a dose-related formation of methemoglobin, which reduces oxygen binding and transport and can lead to cyanosis and respiratory distress. Although this effect is ordinarily not clinically significant at recommended dosages, prilocaine should be avoided in patients with existing methemoglobinemia, severe anemia, chronic hypoxia related to cardiac or pulmonary disease, or patients taking drugs with oxidant properties, e.g., nitrites, sulfonamides or acetaminophen.

Bupivacaine is distinguished from the other amide-type agents by its considerably greater lipid solubility. This property makes bupivacaine more potent (and toxic) and gives it a much longer duration of action. Soft tissue anesthesia up to 4 hours following infiltration and up to 8 hours following nerve block may be achieved with bupivacaine. However, pulpal anesthesia in the maxilla may be relatively short after supraperiosteal infiltration. The greater duration of anesthesia makes bupivacaine useful for longer procedures, particularly surgical. Bupivacaine is the only anesthetic used in dentistry for which dosage on the basis of body weight has

Table 3 - Duration of Action Profiles of Dental Local Anesthetics*

Category	Duration [†]	Preparations
Short	Infiltration: up to 30 minutes Nerve block: up to 2 hours	• Lidocaine 2% plain • Mepivacaine 3% plain • Prilocaine 4% plain
Intermediate	Infiltration: up to 2.5 hours Nerve block: up to 3.5 hours	• Articaine 4% with 1:100,000 or 1:200,000 epinephrine • Lidocaine 2% with 1:50,000 or 100,000 epinephrine • Mepivacaine 2% with 1:20,000 levonordefrin • Prilocaine 4% plain or with 1:200,000 epinephrine
Long	Infiltration: Up to 4 hours Nerve block: Up to 8 hours	• Bupivacaine 0.5% with 1:200,000 epinephrine
* Duration of local anesthesia depends upon multiple factors. In addition to type (supraperiosteal infiltration or nerve block) and quality of injection, anatomic deviations, variations in patient response to the anesthetic agent, local tissue environment, dose, and rate of clearance from the site of injection will influence duration. † Estimated period of soft tissue anesthesia; duration of pulpal anesthesia is typically less.		

not been established. However, a total dose of 90 mg should not be exceeded in adults. Use in children is not recommended.

In mid-2003, local anesthetic manufacturers participating in the ADA Seal program agreed to a system for color-coding and uniform contents labeling of anesthetic cartridges (**Table 4**). This welcome change makes recognition easier and reduces chances of errors in anesthesia administration.

Alternative Delivery Methods

Most dental anesthesia is achieved using either infiltration or block injections in soft tissue. These techniques are straightforward, easily mastered and when used correctly are safe and effective. However, the need to deliver an anesthetic by a different route occasionally arises. For example, it may be desirable to anesthetize a single mandibular tooth without numbing the tongue, lip and other soft tissues. There are also instances, e.g., infection, where usual injection techniques fail to produce sufficient anesthesia, and an alternate or “rescue” method is required.

There are several alternatives to soft tissue injections with which practitioners should be familiar. These include the intraosseous and periodontal ligament (intraligamentary) injections. Detailed

descriptions of these techniques can be found in textbooks on local anesthesia⁴, and are described only briefly here. Intraosseous injection requires perforation of the cortical bone on the lateral aspect of an interdental septum. A needle is then inserted through this hole and a local anesthetic injected into the cancellous bone. Diffusion of the drug from the site of injection can produce pulpal and surgical anesthesia of the adjacent teeth and gingiva. The onset is usually achieved with seconds and may last up to 30 minutes or more. In the periodontal ligament injection, the needle is advanced vertically into the gingival sulcus on a proximal surface until the tip is wedged tightly in the periodontal ligament space. A small amount of anesthetic solution is then injected and pulpal anesthesia is usually achieved almost instantaneously. The drug reaches the apical area either by diffusion through the periodontal ligament, the adjacent cancellous bone or both.

Although intraosseous injection can be accomplished with a standard round bur and 27-gauge needle, disposable units consisting of a bone-perforating bur and a size-matched needle are available (e.g., Stabident System™; X-Tip System™). For the intraligamentary injection, special syringes capable of generating high injection pressures are available but offer little advantage over a 27-gauge needle on a standard aspirating syringe.

The intraligamentary injection technique is generally effective, safe and well tolerated by patients. In addition to allowing pulpal and soft tissue anesthesia of single teeth, only a small volume of solution is required, which is desirable when the total dose of anesthetic or vasoconstrictor is a concern in medically compromised patients. However, this injection technique may be problematic on posterior teeth due to the difficulty in attaining proper angulation of the needle. Post-injection tenderness of teeth is an occasional complaint.

The intraosseous technique is more problematic than intraligamentary injection. The cortical bone must first be perforated and care taken not to damage adjacent roots. Depending on the system used, difficulty may be encountered in locating the bone perforation for insertion of the needle or leakage may occur during injection. It has also been frequently reported that local anesthesia via intraosseous injection is associated with transient tachycardia in the majority of subjects.⁵ This effect is apparently due to rapid clearance of the vasoconstrictor from the vascular cancellous bone. For this reason, intraosseous injections should utilize anesthetic preparations without a vasoconstrictor in patients at risk of adverse reactions to such agents.

Computer-Assisted Delivery Devices

In recent years, several semi-automated systems for dental administration of local anesthetics have been introduced; the first of these was the CompuDent (formerly Wand; Milestone Scientific, Deerfield, IL). Using standard dental cartridges, these devices deliver anesthetic solution through a needle attached to a pen-like handpiece. The solution is driven by a pump that compensates for tissue resistance and provides a selectable and controlled rate of injection. A foot pedal allows the operator to activate the injection of solution or to aspirate. Several advantages are claimed, particularly pain-free and more accurate injections.

Several studies of these devices have been reported, and were recently reviewed.⁶ The studies thus far provide no compelling support for the claim that computer-assisted local anesthetic delivery provides significantly greater patient comfort, improved

predictability of anesthesia, or safety compared to conventional syringe techniques. The systems are relatively expensive and require a disposable handpiece and tubing for each use. Practitioners must consider whether the modest advantages of this approach to the safe and effective use of local anesthesia in their practice justify the cost.

Topical Anesthetics

Local anesthetic agents with sufficiently high lipid solubility can produce a modest but clinically useful level of anesthesia when applied topically to oral mucosa. Although this effect extends only a few millimeters beneath the surface, it can minimize the pain associated with needle puncture and minor gingival procedures such as superficial scaling. Agents useful for topical anesthesia include lidocaine, benzocaine and tetracaine.

The efficacy of topical anesthesia is dependent on the amount of drug that penetrates into the tissue subjacent to the site of application. To enhance effectiveness, the concentration of anesthetic is increased (e.g., lidocaine is typically used at 5% and benzocaine at 20%). In addition, anesthetics for topical use are typically supplied in a gel that adheres to mucosa and helps retain the drug at the site. An application period of at least one minute is necessary to achieve a worthwhile effect. During this period, the site should be kept dry and undisturbed but this is difficult to achieve in some areas of the mouth, because of salivary pooling and tissue movement. Liquid preparations of topical anesthetics are even more difficult to keep localized and undiluted at the site of application.

An alternative to gel or liquid preparations is a delivery system consisting of a patch containing 46 mg of lidocaine (DentiPatch™). The patch adheres to dry (but not wet) mucosa and slowly releases the drug for absorption by the underlying tissue. A minimum period of five to ten minutes is recommended to achieve a maximal effect. Although this system is an effective means for topical anesthesia prior to injection, it offers no particular advantage over gel preparations, is more expensive, time-consuming and limited to sites that can be kept dry during application.

Table 4 - Dental Anesthetic Cartridge Color Codes

Product	Color
Lidocaine 2% with Epinephrine 1:100,000	 RED 185
Lidocaine 2% with Epinephrine 1:50,000	 GREEN 347
Lidocaine Plain	 L BLUE 279
Mepivacaine 2% with Levonordefrin 1:20,000	 BROWN 471
Mepivacaine 3%	 TAN 466
Prilocaine 4% with Epinephrine 1:200,000	 YELLOW 108
Prilocaine 4%	 BLACK
Bupivacaine 1.5% with Epinephrine	 BLUE 300
Articaine 4% with Epinephrine 1:100,000	 GOLD 871
Articaine 4% with Epinephrine 1:200,000	 SILVER 877C

Effective June 21, 2003, all local anesthetic cartridges produced by participants in the American Dental Association Seal of Acceptance Program must be color-coded as indicated above. The product is identified by a 3-millimeter colored band placed 15 mm from the stopper end of the cartridge. Labeling of contents is done in durable black letters.

A topical anesthetic system (Oraqix™) designed to reduce pain associated with subgingival scaling and obviate the need for injection was recently introduced. Oraqix is a eutectic mixture of lidocaine/prilocaine (2.5%/2.5%) in a liquid vehicle that gels when placed in a periodontal pocket. It is delivered to the pocket via a blunt canula attached to a cartridge containing 1.7 ml of gel. Anesthesia in the adjacent gingiva occurs within one minute and may persist for approximately twenty minutes. Several studies have demonstrated that this intrasulcular delivery system yielded anesthesia during subgingival instrumentation which was equivalent to that obtained by local infiltration.⁷ The maximum dose of Oraqix is five cartridges per treatment session. Spray devices are often

used to deliver topically effective anesthetics to the posterior palatal mucosa to reduce a hyperactive gag reflex in impression procedures. Some of these are aerosols containing 20% benzocaine (Hurricane™) or 10% lidocaine (Xylocaine™).

Correct use of intraoral topical anesthetics carries few risks. However, just as for administration by injection, the minimal amount of drug should be used. Rapid uptake of local anesthetics, especially such agents as tetracaine, can occur at areas of abraded or ulcerated mucosa, creating the potential for systemic toxicity. Aerosol delivery systems should be used sparingly, since large amounts of drug can be delivered in relatively short period of application. Topical anesthetics should

be avoided in patients with a history of allergic reactions to the particular agent.

In recent years, topical anesthetic formulations prepared by so-called “compounding pharmacies” have been aggressively promoted to practitioners and patients. These concoctions contain typically high concentrations of local anesthetics including lidocaine, prilocaine, benzocaine and prilocaine, and are not approved by the FDA. Toxic reactions and several deaths directly related to such preparations used in non-dental procedures have been reported. FDA-approved products for topical anesthesia are readily available and properly labeled, and dentists are well advised to use only such products.

References

1. Ogle OE, Majoubi G. Local Anesthesia: Agents, Techniques and Complications. *Dent Clin North Amer.* 2012(1):133-148.
2. Hillerup SF, Jensen RH, Ersboll BK. Trigeminal nerve injury associated with injection of local anesthetics. Needle lesion or neurotoxicity? *J Am Dent Assoc.* 2011;142:531-539.
3. Moore PA, Hersch EV. Local anesthetics: Pharmacology and toxicity. *Dent Clin North Amer.* 2010(4):587-599.
4. Malamed SF. *Handbook of Local Anesthesia*, 6th ed. St. Louis: Mosby, 2013.
5. Replogle K, Reader A, Nist R, Beck M, Weaver J, Meyers WJ. Cardiovascular effects of intraosseous injection of 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine. *J Am Dent Assoc* 1999;130:649-57.
6. Blanton PL, Jeske AH. Dental local anesthetics: alternative delivery methods. *J Am Dent Assoc.* 2003;134:228-34.
7. Magnusson I, Jeffcoat M, Donaldson D, Otterbom IL, Hendriksson J. Quantification and analysis of pain in nonsurgical scaling and/or root planing. *J Amer Dent Assoc* 2004; 135:1747-54.

POST-TEST

Internet Users: This page is intended to assist you in fast and accurate testing when completing the “Online Exam.” We suggest reviewing the questions and then circling your answers on this page prior to completing the online exam.

(1.0 CE Credit Contact Hour) Please circle the correct answer. 70% equals passing grade.

1. Which of the following characteristics of a local anesthetic is the most important determinant of its potency and duration of activity?
 - a. vasoconstriction
 - b. lipid solubility
 - c. molecular size
 - d. chemical class (ester or amide)
2. Local anesthetics may be less effective in areas of inflammation because:
 - a. low tissue pH increases the proportion of anesthetic in the uncharged (lipophilic) form.
 - b. low tissue pH decreases the proportion of anesthetic in the uncharged (lipophilic) form.
 - c. blood flow is increased.
 - d. the anesthetic is rapidly degraded.
3. Which of the following local anesthetics should be avoided in patients with severe anemia or chronic hypoxia related to cardiac or pulmonary disease?
 - a. articaine
 - b. lidocaine
 - c. mepivacaine
 - d. prilocaine
4. The maximum safe dose of a local anesthetic in a particular individual:
 - a. is independent of the individual's systemic status.
 - b. varies by type of injection.
 - c. should be based on age.
 - d. should be based on body weight.
5. Which of the following statements regarding allergic reactions to dental local anesthetics is not true?
 - a. Allergy to local anesthetic anesthetics can be reliably determined by skin testing.
 - b. Non-drug components in dental local anesthetic solutions, e.g., sulfites, may be allergenic.
 - c. Allergic hypersensitivity to ester-type anesthetic agents is more common than to amide-type agents.
 - d. Patients may misinterpret responses to epinephrine present in dental anesthetic solutions as an allergic reaction to the anesthetic agent.
6. As the blood level of a local anesthetic increases, the first sign of toxicity would likely be:
 - a. unconsciousness.
 - b. numbness and tingling of the perioral area and tongue (on non-injected area), drowsiness and dizziness.
 - c. bronchospasm.
 - d. nausea and vomiting.
7. The maximum allowable dose of epinephrine administered with dental local anesthetics in a healthy adult at one visit should not exceed:
 - a. 100 mcg
 - b. 200 mcg
 - c. 500 mcg
 - d. 1,000 mcg
8. A patient with severely impaired liver function presents for caries excavation of tooth #27. To minimize the dose of local anesthetic agent necessary to achieve pulpal anesthesia in this tooth, which of the following techniques would be appropriate?
 1. periapical infiltration, tooth #27
 2. periodontal ligament injection, tooth #27
 3. right inferior alveolar and long buccal nerve blocks
 - a. 1
 - b. 2
 - c. 3
 - d. 1 or 2
 - e. 1 or 3
9. Addition of a vasoconstrictor to a local anesthetic solution results in a:
 - a. doubling of the maximum allowable total dose of the anesthetic.
 - b. more profound anesthesia.
 - c. slower rate of uptake of anesthetic from the injection site.
 - d. tighter binding of the anesthetic to nerve fibers.
10. Which of the following preparations would produce the longest duration of anesthesia?
 - a. articaine 4% with epinephrine 1:100,000
 - b. prilocaine 4% with epinephrine 1:200,000
 - c. bupivacaine 0.5% with epinephrine 1:200,000
 - d. lidocaine 2% with epinephrine 1:50,000

Registration/Certification Information (Necessary for proper certification)

Name (Last, First, Middle Initial): _____

PLEASE PRINT CLEARLY

Street Address: _____ Suite/Apt. Number _____

City: _____ State: _____ Zip: _____

Telephone: _____ Fax: _____

Date of Birth: _____ Email: _____

State(s) of Licensure: _____ License Number(s): _____

Preferred Dentist Program ID Number: _____ ☐ Check Box If Not A PDP Member

AGD Mastership: ☐ Yes ☐ No

AGD Fellowship: ☐ Yes ☐ No Date: _____

Please Check One: ☐ General Practitioner ☐ Specialist ☐ Dental Hygienist ☐ Other

FOR
OFFICE
USE
ONLY

Evaluation - Local Anesthetics 5th Edition

Providing dentists with the opportunity for continuing dental education is an essential part of MetLife's commitment to helping dentists improve the oral health of their patients through education. You can help in this effort by providing feedback regarding the continuing education offering you have just completed.

Please respond to the statements below by checking the appropriate box, using the scale on the right.

1 = POOR

5 = Excellent

	1	2	3	4	5	
1. How well did this course meet its stated educational objectives?	☐	☐	☐	☐	☐	
2. How would you rate the quality of the content?	☐	☐	☐	☐	☐	
3. Please rate the effectiveness of the author.	☐	☐	☐	☐	☐	
4. Please rate the written materials and visual aids used.	☐	☐	☐	☐	☐	
5. The use of evidence-based dentistry on the topic when applicable.	☐	☐	☐	☐	☐	☐ N/A
6. How relevant was the course material to your practice?	☐	☐	☐	☐	☐	
7. The extent to which the course enhanced your current knowledge or skill?	☐	☐	☐	☐	☐	
8. The level to which your personal objectives were satisfied.	☐	☐	☐	☐	☐	
9. Please rate the administrative arrangements for this course.	☐	☐	☐	☐	☐	

10. How likely are you to recommend MetLife's CE program to a friend or colleague? (please circle one number below:)

10 9 8 7 6 5 4 3 2 1 0
extremely likely neutral not likely at all

What is the primary reason for your 0-10 recommendation rating above?

11. Please identify future topics that you would like to see:

Thank you for your time and feedback.



To complete the program traditionally, please mail your post test and registration/evaluation form to:
MetLife Dental Quality Initiatives Program | 501 US Highway 22 | Bridgewater, NJ 08807