



Local anesthesia in dentistry - Clinical Considerations

Sharmraaj Subramaniam,

Prasanna Neelakantan*

Undergraduate Clinic 6,
Saveetha Dental College and
Hospitals, Saveetha University,
162, PH Road,
Velappanchavadi,
Chennai - 600077, India

Corresponding Authors:

Prasanna Neelakantan

Email:

prasanna_neelakantan@yahoo.com

Abstract:

Local anaesthesia is commonly employed prior to most dental procedures. It is imperative to understand the mechanisms by which local anaesthetics work, so that their efficacy can be improved for painless dental care. Local anaesthesia also has major clinical implications in that it can precipitate emergencies in patients with an underlying systemic disease. It is imperative that a dentist have a thorough knowledge of the considerations one must take when administering local anaesthesia in patients with systemic diseases. This review discusses the various methods of enhancing the efficacy of local anaesthetics and the precautionary measures one must take when administering local anaesthesia in medically compromised patients.

Keywords: local anaesthesia, allergy, biotransformation, diabetes mellitus, hypertension, congestive heart failure, Magic Wand, Comfort Control Syringe

Introduction

Most dental procedures are performed under local anesthesia. Local anesthetics have a great efficacy and safety in dental practice. Their use is so routine, and adverse effects are very uncommon. The main compositions of this anesthesia are lignocaine (anesthetic), adrenaline (vasoconstrictor), methyl paraben (agent), sodium metabisulphate (fungicide) and water.

Local anesthetics (LA) interrupt neural conduction by inhibiting the influx of sodium ions through channels or iontophores within neuronal membranes. These channels exist in a resting state, where sodium ions are denied entry. When the neuron is stimulated, sodium ions diffuse into the cell, initiating depolarization. Following this sudden change in membrane voltage, further influx is denied while active transport mechanisms return sodium ions to the exterior. Then repolarization, the channel assumes its normal

resting state. An appreciation of these sodium channel states helps to explain the preferential sensitivity of local anesthetics for various classes of neuronal fibers. Local anesthetics have greater affinity for receptors within sodium channels during their activated and inactivated states than when they are in their resting states (1,2). Therefore, neural fibers having more rapid firing rates are most susceptible to local anesthetic action.

Chemistry

The basic chemical structure of a local anesthetic molecule consists of three parts (3):

- A Lipophilic group- an aromatic group, usually an unsaturated benzene ring.
- An Intermediate bond- a hydrocarbon connecting chain, either an ester (-CO-) or amide (-HNC-) linkage. The intermediate bond determines the classification of local anesthetic.
- A Hydrophilic group- a tertiary amine and proton acceptor.

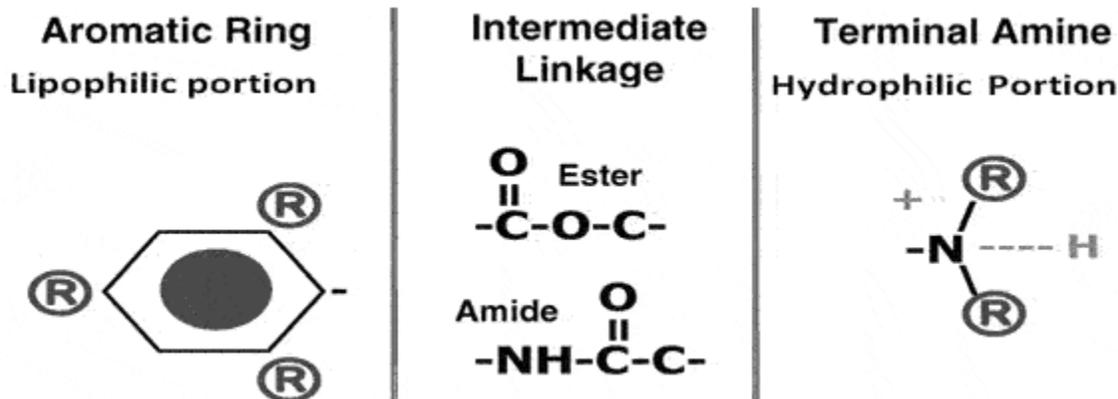


Figure 1: Chemistry of local anaesthetics

Time of onset

Rapid diffusion through cell membranes and increased potency of LA are due to the greater lipid solubility. For local anesthetics, this increases the action for anesthesia in isolated fibers during in vitro studies, although other factors also play a role in this factor. For example, vasodilating properties may promote systemic absorption. High lipid solubility may impede dispersion throughout tissue fluids and also spreads in neighboring adipose tissues or myelin sheaths. In either case, fewer numbers of molecules reach the neuronal membrane and onset is delayed. Therefore, unlike in vitro studies of isolated fibers, greater lipid solubility generally slows the onset of anesthesia in the clinical setting. Injecting higher concentrations that allow a greater number of molecules to reach the membrane and increases onset can offset this influence (3).

Biotransformation and Excretion of Local Anesthetics

The metabolism of local anesthetics always defers in two ways; either its ester or amide in composition. Examples of esters are cocaine, chlorprocaine, procaine and tetracaine. Examples of amides are bupivacaine, lidocaine, ropivacaine, etidocaine and mepivacaine. Amide local anesthetics are metabolized primarily by

microsomal P-450 enzymes in the liver (N-dealkylation and hydroxylation)

Ester local anesthetics undergo massive hydrolysis in the plasma by pseudocholinesterase. Ester hydrolysis is rapid, resulting in water soluble metabolites which are excreted in the urine. An exception is cocaine. In addition to ester hydrolysis cocaine is partially metabolized in the liver (N-methylation). Patients with pseudocholinesterase deficiency are at risk for toxicity (genetic or liver disease). This happens due to the slowing of metabolism ester local anesthetic. Procaine and benzocaine are metabolized to p-aminobenzoic acid (PABA), which can cause allergic reactions. Benzocaine may result in methemoglobinemia (4). Prilocaine metabolites include o-toluidine derivatives, which can accumulate after large doses (>10 mg/kg), resulting in the conversion of hemoglobin to methemoglobinemia. The excretion of amide local anesthetics occurs in the kidneys. Less than 5% of the unchanged medication is excreted by the kidneys (2).

Local anaesthesia in patients with systemic disorders

Diabetes mellitus

The action of vasoconstrictors directly opposes that of insulin. Epinephrine increases

gluconeogenesis and glycogen breakdown in the liver, leading to hyperglycemia. An increased hyperglycemic response to epinephrine has been observed in insulin-dependent diabetics. This risk varies, but the diabetic's level of blood glucose control is an important determining factor. As a result, well-controlled diabetics better tolerate vasoconstrictors and have fewer episodes of hyperglycemia than poorly controlled diabetics.

Studies have shown that the amounts of epinephrine contained in one to three cartridges of local anesthetic (0.018 to 0.054 mg) may be enough to significantly increase the risk of complications (ketoacidosis, hyperglycemia) in patients with unstable diabetes, and so should be avoided until their condition is brought under glycemic control (5). Patients with well-controlled diabetes, on the other hand, can generally receive vasoconstrictor-containing anesthetics without special precautions

Cardiovascular disease

Hypertension

Although vasoconstrictors may precipitate significant elevations in blood pressure, numerous studies have shown that the use of one to two cartridges of 2% lidocaine with 1:100,000 epinephrine (0.018 to 0.036 mg of epinephrine) is of little significance in most patients with hypertension (6). By improving the level of anesthesia, vasoconstrictors lower the risk of endogenous catecholamine release that may result from inadequate pain control (7). For patients with advanced disease, however, special precautions are required. Elective dental care should be avoided in the following situations (8).

- patients with blood pressure greater than or equal to 180/110 (Stage III hypertension)

- patients who have hypertensive symptoms

Hypertensive symptoms include occipital headache, failing vision, ringing in the ears, dizziness, weakness, and tingling of the hands and feet (6). If emergency dental treatment is necessary, medical consultation is required and vasoconstrictor amounts should be limited to one to two cartridges of 1:100,000 solution (0.018 to 0.036 mg of epinephrine) (9). In patients with blood pressure of 160-179/100-109 (Stage II hypertension), epinephrine should be limited to three cartridges (0.054 mg) (8). The use of retraction cord with epinephrine and intraligamentary and intra-bony injections should be avoided in these patients (6).

Ischemic heart disease (Angina pectoris & Myocardial infarction)

Ischemic heart disease results from oxygen deprivation to the heart because of reduced blood supply to a portion of the myocardium. Its causes include atherosclerosis, embolism, coronary artery spasm and congenital abnormalities. Management of these patients involves the use of nitrate drugs, beta-adrenergic blockers, calcium channel blockers, platelet aggregation inhibitors, thrombolytic drugs, and revascularization procedures (coronary angioplasty, coronary stents, or coronary artery bypass graft (CABG) (6).

In the presence of ischemic heart disease, elective dental treatment is contraindicated in the following situations (9):

- patients with unstable angina
- recent myocardial infarction (less than six months)
- recent CABG surgery (less than three months)

If emergency dental treatment is necessary, medical consultation is required and treatment should be aimed mainly at eliminating pain. Epinephrine dosages should be limited to one to two cartridges of 1:100,000 solution (0.018 to 0.036 mg of epinephrine) (9). Similarly, in patients with stable angina, vasoconstrictors should be limited to one to two cartridges (9).

Cardiac arrhythmia

Disturbances in the normal rhythm of the heartbeat can result from cardiovascular diseases, pulmonary disorders, abnormalities in the autonomic nervous system, systemic diseases, drug-related reactions, or electrolyte imbalances (6). Symptoms can include palpitations, dizziness, syncope, chest pain and difficulty breathing. Medical consultation is required for patients with an existing arrhythmia or conditions that predispose to an arrhythmia. Vasoconstrictors are contraindicated in the following (9):

- patients with severe arrhythmias
- patients with an arrhythmia refractory to treatment

In addition, intraligamentary or intraosseous injections should be avoided in these patients (6).

Congestive heart failure

Congestive heart failure (CHF) is the result of the heart's inability to function effectively as a pump. It is the end-stage manifestation of numerous cardiovascular diseases: systemic and pulmonary hypertension, coronary, valvular and congenital heart diseases, cardiomyopathy, infective endocarditis, and endocrine disorders (6). Signs and symptoms include rapid, shallow breathing followed by periods of apnea (Cheyne-Stokes respirations), heart murmur,

arrhythmia, jaundice, fever, cough, distended neck veins, edema, ascites, cyanosis, weight gain, and clubbing of fingers. Elective dental treatment is contraindicated in patients with uncontrolled or untreated CHF, characterized by a marked limitation in physical activities and by the presence of symptoms while at rest (6, 9). Routine dental treatment is acceptable for patients with well-controlled CHF, although vasoconstrictor dosages should be limited to amounts contained in two cartridges of anesthetic (0.036 mg of epinephrine) (9).

Some medications prescribed for the management of CHF have potential interactions with vasoconstrictors. Digoxin, prescribed to increase the heart's contractile force, has a narrow therapeutic index and may precipitate a cardiac arrhythmia when used concurrently with vasoconstrictors. In patients taking nitroglycerin and other vasodilators, the diminished effects of vasoconstrictors can shorten the anesthetic's duration of action (6). Medical consultation and monitoring of vital signs are necessary in the dental treatment of these patients

Methods of enhancing effects of local anaesthesia

Warming

There is evidence in the medical literature that warming local anesthetics to body temperature (37°C) reduces the pain during injection (10,11). Unfortunately these reports are not consistent. Overheating the local anesthetic solution can lead to discomfort for the patient and destruction of a heat-labile vasoconstrictor over a period of time. It has been demonstrated that, after the warmed glass cartridge is removed solution is forced through a fine metal needle; it has cooled nearly to room temperature (12). Cartridge

warmers do not appear to be particularly beneficial.

Rate of injection

The rate at which a drug is injected is very important factor in prevention of overdose and for the comfort of patient. Lidocaine frequently administered (15s or less) of 36mg produces an overdose reaction. Slow administration over a period of 60 seconds is recommended for optimal reduction of pain during injection (13).

Buffering

Local anesthesia solutions are in 2 forms namely the tertiary and quaternary form. The quaternary form is predominantly in a solution with a low pH. The clinical relevance of this is that it is the tertiary form that is lipid soluble and have diffuses across nerve membrane (which is lipid rich) gets converted into the quaternary form and blocks nerve conduction. Commercially available local anesthetic solution with a vasoconstrictor will have a pH about 3.5. At this pH a very small percentage of tertiary form is available which reduces the speed of onset of anesthesia. However considering the normal pH of human tissue (7.4) this solution of lower pH is buffered and hence the percentage of tertiary form increases (14).

Increasing the pH of local anesthesia solution has three advantages. They are more comfortable to patients, increase the speed of onset of local anesthesia and increase the tertiary form which is a more profound anesthesia. This process of buffering can be done by adding sodium bicarbonate in local anesthesia solution immediately before injection. Researches demonstrated a controversial finding that buffering is the main reason of causing failure is lack of knowledge of the exact pH of

sodium bicarbonate (15,16). If the pH of solution exceeds 7.6, a solid precipitate will be formed.

Magic Wand

It is a computer controlled injection. The flow is controlled by a computer so its guaranteed to be slow and steady thus being comfortable for patients. A tube connects to a pen like hand piece. A foot pedal is used by operator to start the computer. Operator can focus on holding in right position. All holders, tubes and hand pieces are single use disposables. Advantages are operator can prevent needle stick injuries and comfortable for patients injection such as in highly elastic area like the hard palate.

Intraosseous injections

An intraosseous injection is used to anesthetize teeth in a quadrant when conventional block and infiltration injections are ineffective. Initially a round bur is used to provide entry in to septal bone and intraosseous anesthesia is given. Now specialized devises are used like the Stabident System (Fairtax Dental Inc) which is a slow speed hand piece driven perforation and a solid 27 gauge wire is beveled and activated. The X tip (Dentsply) anaesthetizes and delivers in 2 ways namely drill and a guided sleeve. The drill leads the guide sleeve through a cortical bone until it's separated and it's withdrawn. The guide sleeve is removed after the intraosseous injection is given. It's given at 2mm apical to the intersection line drawn horizontally to gingival margin. It is preferable administered distal to tooth treated where it's given and not around the area of the mental foramen. (0.6 to 1.2ml) (17).

Novel Devices

Many patients feel that a good dentist should be able to administer profound local anesthesia comfortably. However, the fear and anxiety associated with local anesthetic administration

can be challenging for the practitioner. There have been novel devices developed in this area that may aid in patient comfort. The Single-Tooth Anesthesia System is a computerized local anesthesia delivery system controlled by a foot pedal. It works by delivering local anesthetic at a constant pressure and controlled volume, regardless of the resistance in the tissues. A clinical study indicated that 48 of 50 dentists who volunteered to receive palatal injections with this system experienced a significant decrease in the level of discomfort, compared to the use of a traditional syringe for the identical injection. The operators also experienced reduced stress levels during administration of the palatal injection (18). This device is especially popular because of its efficacy in comfortably administering "single tooth anesthesia" into the PDL space. An additional difference of this system is that, compared to traditional methods of performing a PDL injection, which involve high pressure and low volume, it is a high-volume, low pressure technique that leads to increased patient comfort.

The Comfort Control Syringe

This syringe is an electronic preprogrammed anesthesia delivery device that uses a 2-stage delivery rate. The rate of injection varies based on the injection technique chosen. It begins with a slow rate; the flow then increases to a preprogrammed technique-specific rate selected by the dentist. The operation of this syringe (initiation and termination of the injection, controlled aspiration and flow rate) is controlled by a button on the hand piece. A disposable cartridge sheath is required for each patient, but a standard dental needle and anesthetic cartridge can be used with this device.

References

- 1) Berde CB, Strichartz GR. Local anesthetics. In: Miller RD, Eriksson LI, Fleisher LA, et al, eds. *Miller's Anesthesia*. 7th ed. Philadelphia, Pa: Elsevier, Churchill Livingstone; 2009.
- 2) Katzung BG, White PF. Local anesthetics. In: Katzung BG, Masters SB, Trevor AJ, ed. *Basic and Clinical Pharmacology*. 11th ed. New York, NY: McGraw-Hill Companies Inc; 2009.
- 3) Heavner JE. Pharmacology of local anesthetics. In D.E. Longnecker et al (eds) *Anesthesiology*. New York: McGraw-Hill Medical; 2008.
- 4) Joyce JA. A pathway toward safer anesthesia: stereochemical advances. *AANA Journal* 2002; 70, 63-67.
- 5) Perusse R, Goulet JP, Turcotte JY. Contraindications to vasoconstrictors in dentistry: Part II Hyperthyroidism, diabetes, sulfite sensitivity, cortico dependent asthma, and pheochromocytoma. *Oral Surg Oral Med Oral Pathol* 1992 ; 74:687-91.
- 6) Little JW, Fallace DA, Miller CS, Rhodus NL. Dental management of the medically compromised patient. 6th ed. St. Louis: Mosby; 2002.
- 7) Niwa H, Sugimura M, Satoh Y, Tanimoto A. Cardiovascular response to epinephrine-containing local anesthesia in patients with cardiovascular disease. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001;92:610-63.
- 8) Glick M. New guidelines for prevention, detection, evaluation and treatment of high blood pressure. *J Am Dent Assoc* 1998 ;129:1588-94.
- 9) Budenz AW. Local anesthetics and medically complex patients. *J Calif Dent Assoc* 2000;28:611-9.
- 10) Martin S, Jones J, Wynn B. Does warming local anesthetic reduce the pain of subcutaneous injection? *Am J Emerg Med*. 1996;14:10-12.
- 11) Bainbridge LC. Comparison of room temperature and body temperature local

anesthetic solutions. BrJ Plast Surg. 1991;44:147-148.

- 12) Malamed SF. Handbook of Local Anesthesia. 4th ed. St Louis:The CVMosby Co;1997.
- 13) Malgodi, MH, Munson ES, Embro, MJ: Relation to etidocaine & bupivacaine toxicity to rate of infusion in rhesus monkeys Br J Anaesth 49, 1997, 121-125.
- 14) Bjerneroth G. Alkaline buffers for correction of metabolic acidosis during cardiopulmonary resuscitation with focusonTribonat. Resuscitation.1998; 37:161-171.
- 15) Catchlove RFH.The influence of CO2 and pH on local anesthetic action. J Pharmacol ExpTher.1972;181:298-309.
- 16) Scarfone RJ, Jasani M, Gracely EJ. Pain of local anesthetics: rate of administration and buffering. Ann Emerg Med. 1998;31:36-40.
- 17) Moore PA,CuddyMA,Cooke MR, Sokolowski CJ. Periodontal ligament and intraosseous anesthetic injection techniques: alternatives to mandibular nerve blocks. J Am Dent Assoc. 2011; 142:13S-18S.
- 18) Hochman M, Chiarello D, Hochman CB, et al.Computerized local anesthetic delivery vs. traditional syringe. Subjective pain response.NYStateDentJ.1997; 64:24-29.

Article History:

Date of Submission: 22-04-2013

Date of Acceptance: 29-04-2013

Conflict of Interest: NIL

Source of Support: NONE

