International Journal of Health Sciences and Research
Vol.13; Issue: 1; January 2023
Website: www.ijhsr.org
Review Article *ISSN: 2249-9571*

Local Anaesthesia in Periodontics - A Brief Review

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DOI: https://doi.org/10.52403/ijhsr.20230111

ABSTRACT

Pain is an unpleasant sensory and emotional experience of varying degrees often caused by biological, social and psychological factors. Local anesthetics are amphipathic molecules that when administered in sufficient concentration, inhibit pain transmission. As the administration of local anesthetics is inevitable in routine dental treatments, in-depth knowledge about local anesthesia, its effects on the body and how the body reacts to the drug is of paramount importance. This review article highlights the key aspects of local anesthesia including its role in periodontal therapy.

Keywords: Dental pain, Local Anesthesia, LA in Periodontics, LA techniques, Lidocaine, Epinephrine.

INTRODUCTION

Local anesthesia forms the basis of many routine dental procedures. It is defined as a loss of sensation in a circumscribed area of the body caused by depression of excitation in nerve endings or an inhibition of the conduction process in peripheral nerves [1]. The local anaesthetic solutions are injected at the surface of the nerves which in turn temporarily inhibits the transmission of nerve impulses resulting in profound anesthesia in a localized area without altering the consciousness. Hall in 1884, introduced cocaine as the first local anaesthetic agent in dentistry [2]. Procaine was discovered as a synthetic substitute that eventually led to the development of esters and amide types of LA but the search is still set to develop an effective LA with the least adverse effects.

PHARMACOLOGY OF LOCAL ANAESTHESIA

The structure of local anesthesia includes – a lipophilic aromatic ring, an intermediate ester or amide linkage and a tertiary amine, all of which contribute to a distinct property. The potency of the local anaesthetics largely relies on its lipid solubility, which in turn is responsible for the rapid diffusion of the LA molecule through the nerve membrane. Although greater lipid solubility slows down the onset of action, the number of LA molecules that are converted to its tertiary, lipid soluble variant when exposed to a pH of 7.4 (physiological pH), majorly determines the time of onset of local anesthesia [3]. These tertiary molecules get converted to a quaternary form once they reach the neuron, resulting in neural blockade. The local anaesthetic molecules have larger affinity for the circulating plasma proteins and are less likely to diffuse from the site of action. Hence, the duration of action of local anesthesia directly depends on the percentage of protein binding of the LA [4]. Proximity to the nerve fibers also influences the duration of action of LA and thus vasoconstrictors are added to combat the vasodilatation property of local anaesthetics, resulting in delayed

absorption and prolonged effect of anesthesia [1].

METABOLISM AND ELIMINATION OF LA MOLECULE

The Amide type of local anaesthetics is acted upon by the hepatic microsomal enzymes and are bio-transformed into water soluble metabolites in the liver, which is then subsequently excreted via the kidney. Esters are hydrolyzed in the bloodstream by Pseudo choline esterase and get converted to Para amino butyric acid (PABA) which can cause allergic reactions and thus usage of ester type of LA is limited to topical usage except for benzocaine. An amide type of LA called Articaine consists of an ester side chain and hydrolysis of this ester side chain inactivates the drug and eliminates it in a similar mechanism as ester type of local anaesthetics.

MECHANISM OF LOCAL ANAESTHESIA

Calcium ions regulate the movement of sodium ions through the nerve membrane and are pivotal for nerve membrane depolarization. Local anaesthetics act as competitive antagonists of the calcium ions for the receptor on the nerve membrane and eventually blocks the sodium channel. LA molecules selectively inhibit the permeability of the ion channels to sodium, insignificantly decreases the conductance of potassium, together resulting in conduction blockade. This nerve block produced by the local anaesthetics is called as the nondepolarizing nerve block [1]. The sequence of conduction blockade is described in *Figure 1*.

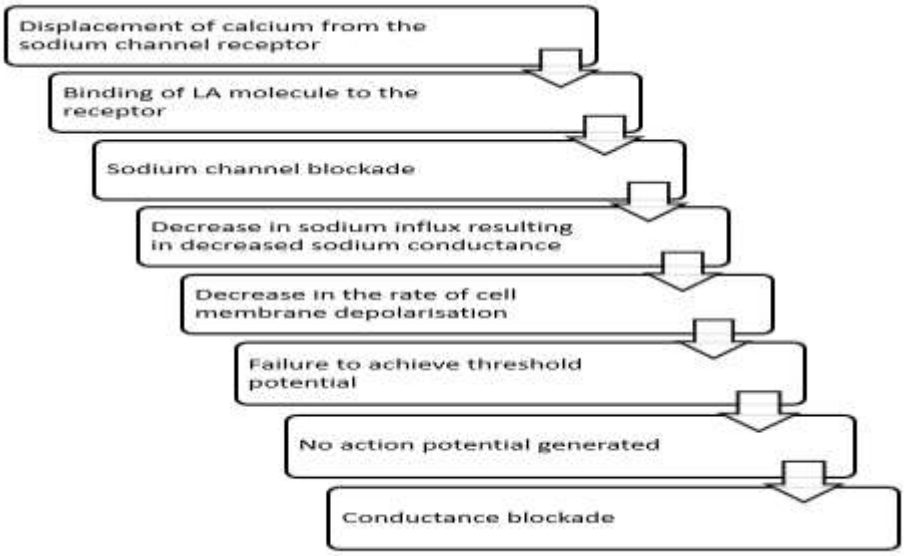

FIGURE 1 - MECHANISM OF ACTION OF LOCAL ANAESTHESIA [1,3]

CONTRAINDICATIONS:

A) Relative Contraindication:

The drug should be administered only after careful analysis of the risk benefit ratio in conditions where an alternative drug is not available. Malignant hyperpyrexia (Succinylcholine, Halothane, Lidocaine, Mepivacaine), Atypical plasma cholinesterase (Succinylcholine, Ester type LA), Idiopathic or congenital methemoglobinemia (Prilocaine) are the relative contraindications of LA.

B) Absolute Contraindication:

Under no circumstance, the drug should be administered as it is toxic to the patient as in case of Allergy to LA.

CLASSIFICATION:

1) Based on the biological site and mode of action:

Class A: Agents like biotoxin (Tetrodotoxin and Saxitoxin) act at the receptor site on the external surface of the nerve membrane.

Class B: Agents like Quaternary ammonium analogs of lidocaine and scorpion venom acts at the receptor site on the internal surface of the nerve membrane.

Class C: Agents like Benzocaine, act by the receptor independent physico-chemical mechanism.

Class D: Agents like Lidocaine, Articaine, Prilocaine, Mepivacaine act by combination of receptor and receptor independent mechanisms.

2) Based on the presence or absence of vasoconstrictors they can be classified into Plain local anaesthetic solution and Vasopressor containing local anaesthetic solution

3) Surface anaesthetics are classified into soluble and insoluble anaesthetics. Soluble surface anaesthetics include Cocaine, Tetracaine. Benoxinate, Lignocaine whereas insoluble surface anaesthetics include Benzocaine, Oxethazine [5].

4) Based on the potency and duration of action, Local anaesthetics are classified as low potency and short duration of action (Procaine, Chloroprocaine); Intermediate potency and duration (Prilocaine, Lidocaine, Mepivacaine); High potency and long duration (Tetracaine, Etidocaine, Bupivacaine) [6].

5) Based on the chemical structure:

Ester type of local anaesthetic possesses an ester linkage between the benzene ring and the intermediate chain, whereas the amide type of local anaesthetics contains an amide linkage between them [5]. Classification is described as a flowchart in *Figure 2.*

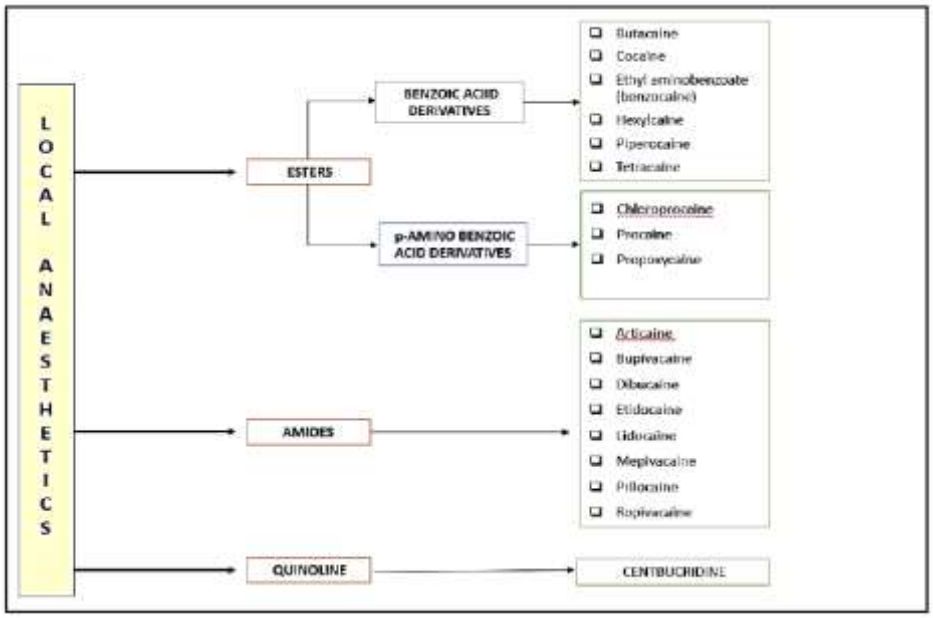

FIGURE 2 - CLASSIFICATION OF LOCAL ANAESTHESIA [1].

COMPOSITION OF LA:

The major composition of local anesthesia includes the local anaesthetic drug such as Lidocaine hydrochloride for blockade of nerve transmission. This drug is the only stable component and can be subjected to autoclave or heat without getting deteriorated. Vasoconstrictors like Adrenaline are added to LA for safety, enhanced depth and duration of action. Sodium metabisulphite is an antioxidant that prevents the oxidation of vasoconstrictors by oxygen and is always included in LA with a vasoconstrictor. This even cause

allergic manifestations and burning sensation that is attributed to the administration of the old local anaesthetic solution. Sodium chloride is added to provide isotonicity to the solution and distilled water is added to make up the volume of the liquid. Methylparaben 0.1% was used as an antibacterial agent until the Food and Drug Administration mandated its removal in 1984 owing due its allergic manifestations. The pH for plain LA is 6.5 whereas with vasoconstrictor the Ph ranges from 3.5 to 4.5 [1].

ROLE OF VASOCONSTRICTORS IN LA:

All local anaesthetic drugs produce vasodilatation but the degree may vary among each other. One exclusion is cocaine, which initially produces vasodilatation followed by intense vasoconstriction. This vasodilatation property of LA increases its absorption into the blood thereby compromising the duration and quality of anesthesia achieved [1]. Vasopressors are drugs that provide constriction of blood vessels by activating alpha-1 adrenergic receptors [3]. Thus, a vasoconstrictor is added to LA, to combat its vasodilatation effects. The effects of vasoconstrictors when added to a local anaesthetic is mentioned in *Figure 3.* The vasoconstrictors fall under 2 major categories: The catecholamines such as Epinephrine, Nor-epinephrine, Levonordefrin, Isoproterenol, Dopamine and the Non-catecholamines like Amphetamine, Methamphetamine, Hydroxy amphetamine, Ephedrine, Phenylephrine, Metaraminol, Mephentermine. The two vasoconstrictors commonly used are Epinephrine and Levonordefrin. Epinephrine remains the most effective and most used vasoconstrictor in medicine and dentistry. They are available in various concentrations like 1:1,00,000 and 1:2,00,000 concentrations. Even slight concentrations of epinephrine increase the cardiac stimulation, and is thus contraindicated in patients with hyperthyroidism and underlying cardiac disease. Levonordefrin produces peripheral vasoconstriction similar to norepinephrine and is thus not widely used in LA. Felypressin, a synthetic analogue of anti-diuretic hormone is also used as a vasoconstrictor in local anaesthetics. The maximum dosage of local anaesthetics with and without vasoconstrictors is mentioned in *Figure 4.*

FIGURE 3 - ROLE OF VASOCONSTRICTOR IN LA [1,3]

Agent	Onset	Duration	Maximum dose	Maximum Epinephrine dose
Bupivacaine	5–10 min	200 min + (up to 540 min w/ epinephrine)	2.5mg/kg	3mg/kg
Lidocaine	<2 min	30–60 min (longer w/epinephrine)	3mg/kg	5mg/kg
Articaine	2–3 min	180–360 min	7mg/kg	7mg/kg
Mepivacaine	3–5 min	45–90 min	5–6mg/kg	5mg/kg
Prilocaine	5 min	30–90 min	5mg/kg	7mg/kg
Ropivacaine	5–15 min	200 min +	3mg/kg	3mg/kg
Procaine	10–20 min	40 min	7mg/kg	Not applicable

FIGURE 4: MAXIMUM DOSAGE OF VARIOUS LA DRUGS [7].

LOCAL ANAESTHETIC TECHNIQUES:

Supra-Periosteal Injection:

This technique is employed mainly to anaesthetize the maxillary anterior teeth. The target site is the height of the muco-buccal fold on to the apex of the tooth to be treated. The needle is penetrated and approximately 1-1.5 ml of the solution is deposited slowly following aspiration.

Posterior Superior Alveolar Nerve Block:

This technique is confined to the upper molar region along with the attachment apparatus supporting the teeth apart from the mesiobuccal root of the maxillary first molar [8]. The needle is positioned at 45 degrees to mesiodistal and buccolingual planes at the height of mucobuccal fold, distal to the zygomatic process of the maxilla. 1ml of the solution is slowly deposited following aspiration [1,10].

Middle Superior Alveolar Nerve Block:

This technique is mainly used to anaesthetize the mesiobuccal root of the maxillary first molar along with the maxillary premolars. The needle is penetrated to about 5mm at the height of mucobuccal fold laterally to the maxillary second premolar and the solution is slowly deposited following aspiration [11,12].

Anterior Superior Alveolar Nerve Block:

This technique is confined to the anterior region from the canine to the midline. The target site is at the height of muco-buccal fold of the maxillary canine. The needle is penetrated and 1ml of the solution is slowly deposited after aspiration[12].

Greater Palatine Nerve Block:

This technique anaesthetizes the distal most portion of the hard palate to the distal part of the canine. The needle is then penetrated at the soft depression found posterior to the hard palate, located distal to the second molar where 0.5ml of the solution is slowly deposited following aspiration [12].

Nasopalatine Nerve Block:

This technique is confined to the anterior region of the maxilla. The target site includes the nasopalatine foramen at the incisive papilla, posterior to the maxillary central incisor region where the needle is penetrated less than 5mm and 0.25ml of the solution is slowly deposited following aspiration [12]. Blanching can be appreciated in the nasopalatine area where the needle puncture is given

Inferior Alveolar Nerve Block:

Anesthesia is confined to the mandibular teeth region involving the lower molars, the attachment apparatus, skin and sub-cutaneous tissues of the chin and lower lip of the ipsilateral side. The target site is located 1.5mm above the mandibular occlusal plane. The needle is penetrated at a point between the pterygomandibular raphae and deep temporalis till it hits the bone approximately at 25mm [13]. 1.5ml solution is deposited after aspiration, and the needle

is advanced back to find the lingual nerve where the remaining solution is deposited. Thus, obtaining the lingual nerve block at about 10mm of penetration [14,15].

Buccal Nerve Block:
The needle is injected distal to the third molar region and 0.2ml of the solution is slowly deposited following aspiration to anaesthetize the buccal soft tissues. [16].

Gow Gates Technique/ Open Mouth Technique:
Anesthesia is confined to the inferior alveolar, buccal and lingual nerves [17,18]. The target site is the lower part of lateral pterygoid muscle where the condyle is palpated with the finger of one hand and the cheek is retracted with the thumb of other hand. The needle is penetrated to a depth of 25-30mm lateral to the condylar head where it hits the bone inferiorly and laterally and the solution is then slowly deposited following aspiration.

Vazirani Akinosi Technique / Closed Mouth Technique:
Here, the Anesthesia confined to inferior alveolar, buccal, lingual and mylohyoid nerves [19]. The target site is the maxillary occlusal plane away from the mandibular ramus. The needle must be placed opposite to the mesial aspect of the maxillary 2nd molar and the solution is deposited slowly following aspiration

Mental/Incisive Nerve Block:
Anesthesia is confined to the premolar and anterior region bilaterally [20]. The target site is at the height of mucobuccal fold anterior to the mental foramen between the root apices of premolars.

Intra-Pulpal Technique:
Anesthesia is confined within the pulp of endodontically treated teeth [20,21]. The needle is directed inside the pulp chamber through the access opening entering the root canal, where 0.2-0.3ml of the solution is directly deposited.

Intra-Septal Technique:
Anesthesia is confined to the osseous, soft tissues and achieve homeostasis for scaling and root planning procedures and flap procedures [20]. The needle is placed at 45 degrees to the long axis of the tooth and 90 degrees to the gingiva and is penetrated 2mm apical to the gingival margin at interdental region. The solution is deposited initially in the soft tissue followed by further advancement of the needle to hit the bone and reaches the interdental septum where 0.2-0.4ml of solution is deposited.

LOCAL COMPLICATIONS:

Needle Fracture:
Needle breakage is a rare complication. In most cases, breakage has occurred with 30-gauge needles and during inferior alveolar nerve block. The broken needle can be managed by removing it with the hemostat. If it is inaccessible, it can be removed with the help of CT scan by operating the patient under general anesthesia [21].

Pain On Injection:
The temperate of the solution, rate of injection administration, usage of blunt needles, needles with barbs, aggressive insertion, damaging soft tissues, blood vessels, nerves or periosteum. This can be prevented by using topical anaesthetics and by using a smaller gauge needle [1].

Soft Tissue Injury:
Soft tissue injury most commonly occurs in younger children, children with special needs or disabled patients. These injuries include injury to the tongue, lips and gums that is due to accidental biting while eating or while having hot food. This can be prevented by using short-acting local anesthesia, placing a cotton roll between the teeth and lips and warning the patient or guardian about eating, drinking hot fluids and biting on the tissues to test for anesthesia [22].

Facial Nerve Paralysis:
Improper technique of LA administration especially in case of IANB will insert the needle more posteriorly into the capsule of the parotid gland resulting in facial paralysis. The patient will have signs such as difficulty in closing the eye, winking and blinking, absence of wrinkles in the forehead and drooping of the corner of mouth. The duration of paralysis persists until the action of drug resolves [23].

Trismus:
Trauma to the muscles or infratemporal fossa when administering LA is one of the common causes of trismus. Multiple needle penetrations, Hemorrhage and excess volume of LA administered are some of the causes [1,22].

Hematoma:
Occurs as a result of venous or arterial laceration, intra-arterial blood pressure due to effusion of blood into extravascular spaces. Hematoma leads to pain and trismus. Hematoma can be prevented by aspirating prior to the injection, keeping the number of penetrations minimal and by using a short needle [22].

Edema:
Edema can be due to trauma during injection, infection, allergy, hemorrhage or injection of irritating solution. Edema is minimal if it is due to traumatic injection or injection of an irritating solution. The management of edema is cause-specific. Analgesics are prescribed to reduce pain. In case of allergy induced edema, intramuscular and oral histamine blocker administration done [21].

Ophthalmologic Complications:
The most common complications are diplopia, paralysis or weakening of eye muscles ptosis and mydriasis [22].

SYSTEMIC COMPLICATIONS
Systemic Toxicity
Management of local anaesthetic systemic toxicity includes activating emergency medical services, ensuring adequate oxygenation, treating seizures if present, treating bradycardia or hypotension with an intravenous vasopressor, monitoring ventricular fibrillation and treating as appropriate [24].

Allergy:
Various allergic reactions are urticarial, erythema, itching, angioedema and respiratory distress. This can be avoided by doing a skin prick test before injecting LA. Management of anaphylaxis includes emergency medical services, removing the anaphylaxis inciting agent, ensuring airway latency and administering epinephrine [21].

Psychogenic Reactions:
Presyncope and syncope are the most common medical emergencies that occur during dental procedures. Clinical manifestations are variations in heart rate and blood pressure, pallor, nausea, vomiting and dyspnea. For psychogenic reactions to be prevented, stress reduction protocols should be followed before administering local anesthetic injections is important [24].

Methemoglobinemia:
Exposure to some local anesthetics can precipitate a change in the iron atom in the hemoglobin, specifically from a ferrous state to a ferric state to create a molecule called methemoglobin. An abnormal amount of methemoglobin is produced. In methemoglobinemia, hemoglobin can carry oxygen but it cannot release it effectively to the body tissues leading to headache, dizziness, fatigue, dyspnea and tachycardia are seen. Management includes administration of supplemental oxygen immediately [21].

LA IN PERIODONTICS:
Periodontal procedures majorly involve manipulation of the soft tissue, hemorrhage and postoperative pain. Hence, a longer acting local anesthesia with vasoconstrictor would be of better choice [1]. Crout et al, suggested that vasoconstrictors of higher

concentrations like 2% lidocaine with 1:1,00,000 epinephrine can be used for better hemostasis during periodontal surgical procedures [24]. A study done by Linden et al in 1986, stated that when bupivacaine was administered during periodontal procedures, it resulted in decreased postoperative pain and decreased usage of analgesics, due to its prolonged anesthesia when compared to lidocaine [25]. The presence of inflammation and infection lowers the tissue pH thereby altering the quality of anesthesia. Hence in case of inflammation prefer regional nerve block anesthesia as it could be administered away from the site of inflammation or infection. Depositing a slightly larger volume in this case will provide more base molecules to diffuse into the nerve sheath and thus will not impair the anesthetic effect [26]. Apart from the conventional techniques like the posterior superior alveolar nerve block, the inferior alveolar nerve block, infiltration and lingual nerve block, alternative techniques for LA administration in periodontal procedures are slowly being adapted into practice.

PDL Injection Technique:

Also known as the Intra-ligamentary technique, this is commonly used for single tooth anesthesia for a shorter duration especially when the mandibular nerve blocks fail to work [27]. This involves forcing the local anaesthetic solution into the PDL ligament space with high pressure so that it reaches the medullary cancellous bone. It requires a very small quantity of local anaesthetic solution (0.2 ml) and thus systemic toxicity is less. However, postoperative tenderness, one or two days after the administration and unpleasantness during deposition are its disadvantages [28-30].

Intraosseous Technique:

Masselink in 1910, introduced the intra osseous injection technique wherein he advocated the deposition of local anaesthetic solution, directly into the root tip via a drilled hole approximately 5 mm apical to the buccal papilla [31]. Most often used for single tooth anesthesia in the mandible. Stabident system and the X-Tip dental anesthesia system were introduced for this technique [32]. Gross periodontal infection, presence of fistula or any increased risk of cortical plate perforations are some of its contraindications. When administered with vasopressor, short term palpitations were reported and hence plain local anaesthetic solution whenever possible is more recommended when employing this technique. Pain during perforation and separation of perforator from the drug administering system are its technical difficulties [32].

AMSA Technique:

Friedman and Coachman in 1998, introduced a technique called the Anterior middle superior alveolar never block (AMSA technique) that anesthetizes the anterior superior alveolar nerve, the middle superior alveolar nerve and the sub-neural dental nerve plexus. As stated by Holtzclaw et al, this is a single injection technique that provides a wider area of anesthesia and better hemostasis, but slow rate of administration of 3-4 minutes and operator fatigue are considered its disadvantages [33].

ADVANCEMENTS OVER THE YEARS [34]:

Computer Controlled Devices:

To alleviate the fear and anxiety towards local anesthesia and to provide a more comfortable means of LA administration, many devices were developed. In the mid 1990's computerised system called as Computer- Controlled Local Anaesthetic Delivery (CCLAD) was introduced which was later marketed as The Wand and Compudent, which can be held like a pen and thus improves the tactile sensitivity for the practitioner and also enhances the quality of LA administration. Later in the year 2006, Single tooth anesthesia system (STA) was introduced to decrease the tissue damage and to improve patient comfort.

Comfort controlled syringe involves the use of an electronic device that delivers local anesthesia when the buttons in its handpiece are operated by the practitioner. Quick sleeper, Sleeper one, Vibraject, Syringe micro vibrator are few of the other local anesthesia administering devices

Intra Nasal Local Anesthesia:
Cocaine and tetracaine are commonly used anaesthetics in this route. Trials are taking place to prove the effectiveness of spraying a combination of tetracaine and oxymetazoline into the nares in providing pulpal anesthesia of the maxillary anterior teeth [16].

Ultrasonic Guided IAN Blocks [4]:
Studies on beta mode ultrasound to directly visualize the inferior alveolar nerve and canal and intra-oral ultrasound to guide the inferior alveolar never block is under research.

Needless Local Anesthesia introduced by Madajet and Syrijet using the piston pressure expulsion principle also resulted in good soft and hard tissue anesthesia.

Local Anaesthetics:
Ghavimi et al in 2015, stated that Centbucridine, a quinolone derivative is 5-6 times more potent than lidocaine and does not possess any major adverse effects [35].

CONCLUSION

A thorough knowledge of the composition, metabolism of the drug and its adverse effects is crucial as local anesthesia has become an inevitable part of dentistry. Further researches have to be done to develop more painless and risk free LA agents and techniques, that can be adapted to provide less anxious environment for the patients pertaining to dental treatments.

Declaration by Authors

Ethical Approval: Not Applicable
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Srivainavi Arulmari, Ashwini Athul, Ashwath Balachandran et al. Local anaesthesia in periodontics - a brief review. Int J Health Sci Res. 2023; 13(1):79-89. DOI: *https://doi.org/10.52403/ijhsr.20230111*

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