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Local anesthetics in dentistry: An update review

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Abstract
Introduction: Pain management in dentistry, where local anesthetics are crucial, is fundamental to ensure patient comfort and treatment success by performing virtually painless procedures.
Objective: To analyze the literature on lidocaine, mepivacaine and articaine anesthetics, particularly their chemical characteristics, mechanism of action, pharmacokinetics and administration.
Methodology: An electronic search was performed through PubMed, Google Scholar and Scopus, using the terms: anesthesia, pain management, dentistry, mepivacaine, lidocaine, articaine.
Results: Lidocaine continues to be the "gold standard" in dental local anesthesia due to its rapid onset of action and adequate duration. It is used in a variety of procedures, from conservative treatments to complex surgeries. Mepivacaine is the third most commonly used in dentistry, after articaine and lidocaine. It has a short action and rapid onset, with less vasodilatation than lidocaine, which prolongs its effect even without vasoconstrictor. It is used in local infiltration, nerve block and epidural anesthesia. Articaine is safe and widely used in dental surgery, both for local infiltration and peripheral nerve block. It is also applied in epidural, ocular, spinal and regional intravenous anesthesia.
Conclusions: The choice of dental anesthetic should be based on a comprehensive approach that considers the patient's health profile (medical history, allergies and contraindications), the characteristics of the procedure, drug interactions and adverse effects, prioritizing the balance between efficacy and tolerability. This personalized strategy minimizes risks and optimizes clinical outcomes.

Keywords: Anesthesia, pain management, mepivacaine, lidocaine, articaine

1. Introduction
Pain in dental treatment is the main cause of dental stress, anxiety and dental phobia, especially in children, but effective pain management can transform these negative experiences into positive ones, reducing anxiety and improving treatment acceptance [1].
Local anesthesia is essential in dentistry to reversibly inhibit nerve impulses and control pain during surgical, periodontal, endodontic and restorative procedures, allowing painless treatments [2]. Local anesthesia is the transient loss of painful sensation in a specific area of the body, caused by inhibition of peripheral nerve conductivity by drugs applied mainly by infiltrative blockade in dentistry [3]. In the United States, about 300 million anesthetic cartridges are used annually, and worldwide this figure is in the millions. However, contrary to their clinical benefits, some adverse effects may occur, particularly in combination with vasoconstrictors [4].
Local anesthetics are fundamental in dentistry because of their ability to selectively block nerve fibers, providing precise pain control and improving the patient experience by reducing anxiety. However, despite advances, challenges remain in the efficacy and duration of analgesia. Their mechanism of action is to inhibit action potentials by binding to sodium channels, preventing neuronal depolarization and blocking pain transmission. This makes them essential in dental and medical procedures. Chemically, they are divided into aminoamides (lidocaine, mepivacaine, bupivacaine), which are more commonly used due to their lower toxicity and longer duration, and aminoesters (benzocaine, cocaine derivatives), which are more rapidly metabolized.

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Articaine stands out for its unique structure (thiophene ring and ester group hydrolyzable by plasma esterases), which gives it distinct pharmacokinetic properties [5].

Articaine is very popular internationally, especially in Germany, where 50% of its formulations use epinephrine 1:200,000 and the rest 1:100,000. However, in the UK and USA, lidocaine remains the most common local anesthetic. Despite these differences, epinephrine is the most widely used vasoconstrictor globally in local anesthetics [7].

In this work we analyzed the literature on certain relevant aspects of the anesthetics lidocaine, mepivacaine and articaine, such as their chemical characteristics, mechanism of action, pharmacokinetics and administration.

2. Methodology

Information from articles published in PubMed, Scopus and Google Scholar was analyzed with emphasis on the last 5 years. The quality of the articles was evaluated based on the standard guidelines, i.e., identification, review, choice, and inclusion. The quality of the review was assessed using the measurement instrument for evaluating systemic reviews. The search was performed using Boolean logical operators AND, OR and NOT. It was realized with the words “lidocaine”, “mepivacaine”, “articaine”, along with the following terms: “physicochemical characteristics”, “mechanism of action”, “pharmacokinetics and pharmacodynamics” and “administration”.

3. Results

In clinical dental practice, the most commonly used infiltrative formulations of local anesthetics are lidocaine (2%), mepivacaine (3%) and articaine (4%), commonly available with and without vasoconstrictor such as epinephrine, which prolongs the duration of anesthesia and improves hemostasis.

3.1 Lidocaine

Lidocaine is the most widely used and safest local anesthetic, discovered in 1942 by Nils Lofgren and Bengt Lundquist. It remains the “gold standard” in dentistry because of its efficacy and safe profile. Although new alternatives have emerged, its rapid onset of action and adequate duration maintain it as the preferred choice in procedures ranging from conservative treatments to complex surgeries [6].

3.1.1 Physicochemical Characteristics

Lidocaine is a class Ib (Vaughan-Williams) drug, used as a local anesthetic and antiarrhythmic. Its molecular formula is $C_{14}H_{22}N_2O$, with a structure combining an aliphatic chain, an aromatic ring and an amide group, derived from the condensation of N, N-diethylglycine and 2,6-dimethylalanine. It appears as a white crystalline powder, soluble in water and with a bitter taste [8].

3.1.2 Mechanism Of Action

Lidocaine acts as an anesthetic by blocking voltage-dependent sodium (Na+) channels, reducing their peak current and accelerating the inactivation of the neuronal action potential, which suppresses pain transmission. Its mechanism involves binding to a high-affinity site on the channel, prolonging the recovery period after inactivation and thus inhibiting pathological electrical activity. In addition, it interferes with potassium (K+) channels, contributing to the rapid inhibition of postsynaptic excitability [9].

3.1.3 Pharmacokinetics and Pharmacodynamics

Lidocaine stands out for its rapid anesthetic action due to the fact that 25% of its molecules remain non-ionized under physiological conditions (pH 7.4), favored by its low pKa (7.7), which facilitates its diffusion towards nerve cells in comparison with anesthetics of higher pKa. Its absorption is fast (3-5 min), although influenced by factors such as injection site, concentration, dose and patient's condition.

It has moderate binding to plasma proteins (65%), which gives it an intermediate duration, and its lower liposolubility limits its potency, with a volume of distribution of 0.7-1.5 L/kg. It is metabolized in the liver by CYP1A2 and CYP3A4, generating both active (monoethylglycylcylcylixilide and glycylcylcylxylidine) and inactive metabolites. Its half-life is 1.5-2 hours, and it is eliminated mainly by the renal route (90%) [10].

3.1.4 Administration

In Mexico, lidocaine is used in dental anesthesia in three formats: 10% topical spray, 2% infiltration cartridges (simple) and 2% with epinephrine (1:100,000). The maximum safe dose is 4.5 mg/kg (300 mg in adults) without epinephrine and 7 mg/kg (500 mg in adults) with epinephrine. It is safe in pregnant women, infants and renal patients without adjustment. However, it is contraindicated in allergy sufferers (although severe reactions are rare) and may cause methemoglobinemia in anemic patients, especially when combined with other anesthetics. It interacts with nitrates, nitric oxide, dapsone, sulfonamides, chloroquine, pentobarbital and phenytoin [11].

3.2 Mepivacaine

Mepivacaine, an aminoamide local anesthetic introduced in 1960, is pharmacologically distinct from lidocaine due to its unique molecular structure. This peculiarity gives it distinct pharmacokinetic and pharmacodynamic properties, resulting in an anesthetic profile with a characteristic onset and duration, which has significant clinical consequences [12].

Mepivacaine is a fast-acting local anesthetic (2-3 minutes in maxillary infiltrations, 5-8 minutes in dental blocks) and moderate duration (60-90 minutes). In inflamed tissues (low pH), it ionizes faster, facilitating its diffusion and enhancing its anesthetic effect, even surpassing lidocaine in these conditions [13].

3.2.1 Physicochemical Characteristics

Mepivacaine is a medium-acting local anesthetic, whose chemical structure derives from a piperidinacarboxamide formed by the union of N-methylpipecolic acid and 2,6-dimethylaniline via an amide bond. With a solubility of 7000 mg/L at 23°C and a pKa of 7.7, it exhibits rapid onset and prolonged duration of anesthetic effect, maintaining anesthetic block for 60 to 90 min [12,14]. Marketed as mepivacaine hydrochloride in dental formulations, it stands out for its high potency, long half-life and lower allergenic effect compared to other amide-type local anesthetics, surpassing them in efficacy [15].

3.2.2 Mechanism of Action

Mepivacaine is a local anesthetic that blocks voltage-dependent sodium channels in neuronal membranes, inhibiting depolarization and action potential propagation, affecting both sensory fibers (producing analgesia) and motor and autonomic fibers (causing anesthesia and neuromuscular blockade). When acting on a nerve trunk, it can cause loss of

painful sensation and, in some cases, motor loss in the innervated area, depending on the type of fiber affected, its action being completely reversible [16]. In clinical studies, mepivacaine has been shown to induce QRS complex shortening, QTc interval shortening and ventricular arrhythmias, although the underlying cellular mechanisms are not fully understood. *In vitro* studies in myocardiocytes suggest that it affects contraction and conduction by interacting with sodium and calcium channels, while animal research proposes that blockade of Na+ channels could transiently reduce Ca2+, generating a negative inotropic effect; however, further studies are required to confirm these mechanisms and their role in mepivacaine cardiotoxicity [17].

3.2.3 Pharmacokinetics And Pharmacodynamics

Mepivacaine is used in local and regional anesthesia, and its systemic absorption affects the cardiovascular and central nervous systems, although, at normal therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility and peripheral vascular resistance are minimal. This local anesthetic binds approximately 75% to plasma proteins, and its binding percentage increases as its plasma concentration decreases [12].

Mepivacaine is metabolized mainly in the liver, with only a small fraction excreted unchanged by the renal route. In patients with cardiovascular disease, the maximum recommended dose is 6.6 mg/kg in adults and 4.4 mg/kg in other cases, with an absolute limit of 300 mg, being considered the least vasodilator local anesthetic [18]. Like articaine, it has low toxicity, with an LD50 greater than 1000 mg/kg, and its adverse effects are usually mild, such as dizziness and nausea, related to the central nervous system [19].

3.2.4 Administration

It was introduced in dentistry as a 2% solution with levonordefrin (epinephrine) and in 1961 at 3% without vasoconstrictor, standing out for its slight vasodilatation that prolongs its effect without vasoconstrictor. It is the third most used anesthetic in dentistry, after articaine and lidocaine, available at 3% without vasoconstrictor or 2% with vasoconstrictor, with a weighted dose of 6.6-7 mg/kg [20]. Together with its derivatives (ropivacaine and bupivacaine), it is widely used in regional anesthesia for lower limb orthopedic surgeries, as well as in oral and maxillofacial procedures [21].

It is indicated for infiltration, nerve block and epidural anesthesia, with similar characteristics to lidocaine, such as short action and rapid onset, but with a milder vasodilation that prolongs its duration even without vasoconstrictor [22], which makes it suitable for patients with hyperthyroidism due to its low vasodilator effect [18]. It has an anesthetic duration of 20 to 40 min for pulpal anesthesia and 2 to 3 h for soft tissue anesthesia, mepivacaine has even been reported to guarantee deep and prolonged anesthesia in pulpal treatments [31]. After administration, a portion of the drug is absorbed systemically and can reach plasma concentrations that, in some cases, can produce adverse effects at the cardiovascular level, such as hypotension and bradycardia, and at the central nervous system level, such as dizziness, drowsiness and, in more severe cases, convulsions [15].

3.3 Articaine

Articaine was first synthesized in Germany in 1969, and its first clinical studies began in 1971 by Winther and Nathalang. Approved in 1976 as carticaine hydrochloride, it demonstrated greater effectiveness than 2% lidocaine with vasoconstrictor 1:200,000, achieving a deep blockade in all dental organs except mandibular molars. In 1989 it was renamed articaine, and in 2000 the FDA approved its 4% formulation with epinephrine 1:100,000, marketed as Septocaine® by Septodont®.

In 2006, the FDA approved 4% articaine with epinephrine 1:200,000, and studies showed that this concentration is essential to achieve deep anesthesia, being comparable in pulpal effectiveness with both dilutions of epinephrine [32]. This anesthetic is considered safe, rapidly metabolized, with an inactive metabolite that reduces the risk of systemic toxicity due to overexposure; however, it can cause paresthesia more frequently, either temporarily or permanently [23].

Recent studies highlight that articaine combined with epinephrine is a highly effective local anesthetic in dentistry, surpassing lidocaine in depth and duration of anesthesia, which makes it a preferred option for pain management in dental procedures in adults, children over 4 years of age, the elderly and even patients with systemic diseases, renal or hepatic insufficiency. Its efficacy has been proven in various contexts, including cases of infection and in surgical procedures such as lower molar extraction, where it demonstrates a high success rate in anesthetic blockade and pain control [24].

3.3.1 Physicochemical Characteristics

Articaine, a local anesthetic of the aminoamide group, is distinguished by containing in its molecular structure a thiophene ring instead of the benzene present in other similar anesthetics; this particularity increases its liposolubility, thus improving its tissue absorption and making it unique in its class [25]. Numerous studies highlight that articaine outperforms lidocaine in efficacy due to its chemical differences and pharmacological properties, offering advantages such as greater liposolubility, greater anesthetic potency, faster onset of action, prolonged duration of anesthetic effect, and excellent diffusion in bone tissue [26].

3.3.2 Mechanism of action

Articaine hydrochloride reversibly blocks nerve conduction by binding to neuronal membrane sodium channels, inhibiting electrical excitation and reducing nerve impulse propagation and depolarization rate. Local anesthetics alter the function of these channels, preventing the transmission of action potentials: in their non-ionized form, they cross the cell membrane, ionize in the cytoplasm and bind to the sodium channels, keeping them inactive and preventing depolarization. In addition, non-ionized molecules can act directly on the membrane, disrupting the function of ion channels and suppressing pain sensitivity [27].

3.3.3 Pharmacokinetics and Pharmacodynamics

Articaine is inactivated mainly (90%) by rapid hydrolysis in serum by esterases, transforming into (inactive) articaineic acid, which is then excreted by the kidneys as glucuronide; slower hepatic metabolism also contributes to its biotransformation. Articaineic acid has a longer serum half-life (64 min) than articaine (20-40 min), contrasting with lidocaine (90 min), which reduces the risk of toxicity in repeated infiltrations due to its rapid elimination [25,28].

3.3.4 Administration

The recommended dose of articaine is 7 mg/kg for adults and 5 mg/kg for children 4 to 12 years of age. Although local anesthetics, including articaine, can cause adverse reactions such as dizziness, disorientation, tremor, convulsions, hypotension, and respiratory depression, articaine is considered relatively safe because of its rapid metabolism, which generates an inactive metabolite, thus reducing the risk of systemic toxicity and overdose, even with repeated infiltrations [24].

Articaine has been shown to be safe and effective for local anesthesia or peripheral nerve blockade in dentistry, with medical applications such as epidural, ocular, spinal, and regional nerve blockade, as well as intravenous administration for regional anesthesia, although its most widespread use is in dental surgery [30].

Articaine has been associated with an increased risk of paresthesia due to its 4% concentration, considered more neurotoxic than other local anesthetics, although these claims are controversial, since studies show that it does not require repeated infiltrations during surgery and has a success rate of 90%, higher than 81% of lidocaine [26,29].

4. Conclusions

Adequate pain management by means of anesthesia is essential in dentistry, since it guarantees comfortable and safe treatments, transforming previously painful procedures into tolerable experiences. Local anesthetics, by blocking nerve impulses, allow interventions with minimal pain, but their use involves risks such as allergic reactions, systemic toxicity (dizziness, convulsions) or local complications (hematomas, infections), so they should be administered by trained professionals following strict protocols. The choice of the optimal anesthetic depends on the type of intervention, the patient's health profile (age, comorbidities, medications) and the pharmacological properties of the drug.

Conflict of Interest:
Not available

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5. References

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