The Use of Lidocaine and Prilocaine in Dental Surgery. A Review

Shahrooz Shafaifard¹, Ramooz Hussain Khan¹*

The purpose of the present article is to carry out a review of the literature on two local anesthetic agents, which are the most commonly used drugs in dental procedures. It would provide a clear concept about the local anesthetics, their dosages and exact usage in dental surgeries.

Keywords: local anesthetics; lidocaine; prilocaine; dental surgery

For more than 100 years, local anesthetics have been used in dental surgery. The appearance of local anesthetics with the advancement of nerve blockade techniques heralded a new era of patient convenience while permitting more extensive and invasive dental procedures. It has been estimated that more than 300 million cartridges are administered by dentists in the United States every year [1].

The first local anesthetic agent to be widely used in dentistry was cocaine. Neiman recognized the anesthetic effect of cocaine when he noted that “it benumbs the nerves of the tongue, depriving it of feeling and taste” [2]. Halsted introduced numerous regional nerve block techniques and many of them are still fundamental to dental practice [3]. Although cocaine’s introduction in dental surgery had been a great achievement, it had major drawbacks, such as high tendency for addiction and a short duration of action. Because of its short duration of action, multiple injections were needed to get the desired effect and thus a large dose had to be utilized. So the potential for systemic toxicity increased. An alternative technique was to apply a tourniquet near the surgery site to curtail its systemic absorption.

Chemical tourniquet with epinephrine was reported by Heinrich Braun in 1903 [4]. Epinephrine added to the cocaine solution produced localized vasoconstriction to slowing its vascular uptake and thus reducing the total cocaine dose and preventing its systemic absorption [5]. This is attributed to the vasoconstrictor effect of epinephrine which helps in keeping the local anesthetic cocaine at its site of injection thus preventing its systemic absorption.

Alfred Einhorn discovered an ester-based synthetic local anesthetic and named it procaine in 1905 [6]. It was accepted as a safe local anesthetic immediately after its introduction. Lidocaine was synthesized by Nilsofgren in 1943 [7]. Lidocaine was more potent and less allergenic than procaine.

It is an amide based local anesthetic agent. Several other amide-based local anesthetics were subsequently developed for use in dental surgery: mepivacaine, prilocaine, bupivacaine, etidocaine and articaine.

Amide based agents have advantages compared to ester based agents, specially their low rate of allergenicity. Although, investigations show that most of allergic reaction reports are of psychogenic origin [8-9]. The para-aminobenzoic acid derivative in ester-based agents may contribute as the true allergic factor. In contrast to amide-based agents, an allergy to one ester rules out use of another ester. Nowadays, the amide based agents have led to complete replacement of the ester-based anesthetics in dental procedures. However a variety of local anesthetic agents enable dentists to choose an anesthetic that has specific properties such as its time of onset and duration, hemostatic control and degree of cardiac side effects that are appropriate for each individual patient and for each specific dental procedure.

A number of factors influence the duration and depth of anesthesia [1]:

1- Individual variation in response to the drug administered
2- Accuracy in administration of the drug
3- Tissue condition at the site of drug deposition (vascularity, pH)
4- Anatomical variation
5- Type of injection administered (Infiltration or nerve block)

U.S. Food and Drug Administration (FDA) has recommended particular local anesthetic agent for special patient populations. Lidocaine and prilocaine have the best FDA ranking [10-11]. 2% lidocaine with epinephrine 1/100000 may be preferred for children.

Lidocaine

Lidocaine hydrochloride injection, contains lidocaine hydrochloride, which is chemically designated as acetamide, 2-(diethylamino)-N-(2,6-dimethylphenyl)-, monohydrochloride and has the molecular weight 270.8 (Figure 1).

Lidocaine is amid-based local anesthetic agent and it was twice as potent and twice as toxic as procaine, producing a greater depth of anesthesia with a longer duration over a larger area than a comparable volume of procaine.
Three formulations are available:
1. Lidocaine 2% (plain)
2. Lidocaine 2% + 1/100000 epinephrine
3. Lidocaine 2% + 1/50000 epinephrine

Plain form of lidocaine has a soft tissue anesthetic duration of one to two hours and limited use for most dental surgeries because of a pulpal duration of only five to ten minutes (Table 1). Epinephrine formulations have a pulpal duration of 1 to 1.5 hours and soft tissue range of three to five hours. Hemostasis advantages may be seen by 1/50000 epinephrine concentration. However, it has not significant advantage for pulpal anesthesia duration. Susceptibility to toxic side effects in any given patient depends on several factors, such as site of administration, speed of injection and the presence of a vasoconstrictor. But the maximum recommended dose for lidocaine is 7 mg/kg [10,12-13].

Prilocaine is slightly less potent and less toxic agent than lidocaine. Introduced in 1960 it was named prilocaine. It is chemically designated as propanamide, N-(2-methyl-phenyl)-2-(propylamino)-, monohydrochloride (Figure 2).

Figure 1- Lidocaine hydrochloride structural formula

Figure 2- Prilocaine structural formula

Table 1- Duration of action of lidocaine and prilocaine solutions [12, 13, 17, 18]

<table>
<thead>
<tr>
<th>Formulation</th>
<th>Duration of action (min)</th>
<th>Duration of action (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maxillary infiltration</td>
<td>inferior alveolar block</td>
</tr>
<tr>
<td>Lidocaine 2% with</td>
<td></td>
<td></td>
</tr>
<tr>
<td>epinephrine 1:50,000</td>
<td>60</td>
<td>170</td>
</tr>
<tr>
<td>or 1:100,000</td>
<td>60</td>
<td>85</td>
</tr>
<tr>
<td>Prilocaine 4% with</td>
<td></td>
<td></td>
</tr>
<tr>
<td>epinephrine 1:200,000</td>
<td>40</td>
<td>140</td>
</tr>
<tr>
<td>or 1:100,000</td>
<td>40</td>
<td>60</td>
</tr>
<tr>
<td>Prilocaine 4% plain</td>
<td>20</td>
<td>105</td>
</tr>
<tr>
<td>or 1:100,000</td>
<td>20</td>
<td>55</td>
</tr>
</tbody>
</table>

As compared with lidocaine, it produces less tissue vasodilatation and can be used reliably in plain formulation for short duration procedures.

Prilocaine is available in two formulations:
1. Prilocaine 4% (plain)
2. Prilocaine 4% + epinephrine 1/200000

Pulpal duration of plain form is 40-60 minutes and with soft tissue an anesthesis of two to three hours (Table 1). In comparison with other local anaesthetic agents, anaesthetic duration of plain prilocaine is more dependent upon the type of injection. Infiltration injections provide 5-10 minutes of pulp anaesthesia with due attention to nerve block injections which extend from 40-60 minutes. The epinephrine containing formulation provides pulp anaesthesia for 1 to 1.5 hours and it has longer soft tissue duration of 3-8 hours [1]. An additional advantage is the decrease in cardiac side effects due to the lower vasoconstrictor concentration. Recommended maximum doses of this local anesthetic agent is 8 mg/kg. But the high concentration solutions will reach toxic levels with fewer injections than is the case for the other drugs.

Some relative contraindications have been mentioned in papers [1]:
1. A patient history of methemoglobinemia
2. Anaemia
3. Cardiac or respiratory failure due to hypoxia

Nerve paresthesia may occur particularly during inferior alveolar and lingual nerve block injections [14-15]. In reviews it has been described that the chemical toxicity may be the cause of paresthesia induced by this agent [16]. Most of these reactions are transient and resolve within 8 weeks, but they may become permanent.

Precautions for prilocaine usage are as under:
1. Dose reduction to the absolute minimum amount required for effective anaesthesia
2. The use of slow,atraumatic injection technique with repeated aspirations

As a rule, biotransformation of amides occurs primarily in the liver but prilocaine is also metabolized in the plasma and kidney, and one of its metabolites may lead to methemoglobinemia. Methemoglobinemia may occur as an uncommon side effect with prilocaine, articaine and topical anaesthetic benzocaine. Excess of metabolites of these drugs induce a cyanotic state that does not respond to 100% oxygen. However, the range of symptoms may appear proportionally to the methemoglobin levels.

References