EFFECTIVENESS OF ARTICAINE VS LIDOCAINE IN DENTAL PROCEDURES.

DR. N TAMILKKUMARAN*, DR. MADHULAXMI.M AND DR.P.U.ABDUL WAHAB

Department Of Oral and Maxillofacial Surgery Saveetha Dental College, Chennai, India

ABSTRACT

Local anesthesia causes a localized sensational loss due to the excitation depression or conduction process inhibition at peripheral nerve. Articaine and lidocaine belongs to amide group of local anesthesia. The aim of this paper is to evaluate the effectiveness of articaine and lidocaine in dental procedures.

KEYWORDS: Articaine, Lidocaine, Local anesthetics, Anaesthesia, Dental procedures

*Corresponding author
INTRODUCTION

Local anesthesia is used in several procedures in dentistry. Local anesthesia has been defined as a loss of sensation in a circumscribed area of the body caused by a depression of excitation in nerve endings or an inhibition of the conduction process in peripheral nerves[1]. The first substance that was used for this function was cocaine, as far back as in 1884. In the year 1903, Braun recommended using adrenaline as a “chemical tourniquet” to extend the duration of local anesthetics. In the year 1904 Einhorn synthesized procaine, an ether anesthesia. In the year 1940’s an innovative group of local anesthetic compounds, the amides, were introduced. The early amide local anesthetic, lidocaine, was synthesized by the Swede chemist Nils Løfgren in the year 1943. Lidocaine revolutionized pain manage in dentistry wide-reaching, as it was both more effective and less allergenic than procaine. In the year 1969, Rusching et al equipped a new drug, articaine. It differs from the earlier amide local anesthetics in that it is a derivative from thiophene, and since of that contained a thiophene ring in its molecule in its place of the usual benzene ring. It was initially named as carticaine, but its generic name was changed to articaine in 1984. It was introduced into the German market in 1969[1].

THE BASIC PROPERTIES OF ARTICAINE AND LIDOCAINE

<table>
<thead>
<tr>
<th>Substance</th>
<th>Articaine</th>
<th>Lidocaine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vd (ss)</td>
<td>7.7 +/- 5.1 L/kg [2]</td>
<td>91 [3]</td>
</tr>
<tr>
<td>pKa</td>
<td>7.8 [1]</td>
<td>7.9 [1]</td>
</tr>
<tr>
<td>Lipid solubility</td>
<td>1.5 [4]</td>
<td>4.0 [4]</td>
</tr>
<tr>
<td>Plasma protein binding</td>
<td>76 % (pH 8.5) [5]</td>
<td>74 % (pH 8.5) [6]</td>
</tr>
</tbody>
</table>

PHARMACOKINETICS

Local anesthetics wedge the feeling of pain by prying with the propagation of peripheral nerve impulses. Both the generation and the conduction of action potentials are withdrawn. Electrophysiological data indicate that local anesthetics do not significantly alter the normal resting potential of the nerve membrane; instead, they impair certain dynamic responses to nerve stimulation [6]. Local anesthetics interfere with nerve conduction by blocking the influence of stimulation on sodium channel permeability. A developing local anesthetic block is characterized by a progressive reduction in the rate and extent of depolarization and a slowing of conduction. Since the onset and rate of repolarization are not greatly affected by local anesthetics, the safety factor for transmission decreases. When depolarization is retarded such that repolarization processes develop before the threshold potential can be reached, nerve conduction fails[3].

PHARMACODYNAMICS

The signs and symptoms are referable to the CNS and cardiovascular system. A comparison between articaine and lidocaine showed that the signs of CNS toxicity after intravenous administration of lidocaine were observed more frequently and at a higher degree of severity when compared with articaine. The cardiovascular parameters did not change [2].

BIOTRANSFORMATION

Normally the metabolism of amide drugs in the liver. Liver function and hepatic perfusion influence the velocity of biotransformation of an amide local anesthetic. Approximately 70% of the dose of injected lidocaine metabolize in patient’s liver. Patients with lower than usual hepatic blood flow (hypotension, congestive heart failure) or poor liver function (cirrhosis) are unable to metabolize amide local anesthetics at a normal rate. This can lead to increased in anesthetic blood levels and potentially
increased toxicity. Significant liver dysfunction (ASA IV-V) or heart failure (ASA IV-V) represents a relative contraindication to the administration of amide local anesthetics [1]. Articaine differs from other amide local anesthetics, in that it has an extra ester linkage (COOCH₃). 90-95% is metabolized in the blood, and only 5-10% in the liver. This feature is clearly demonstrated when you compare the half-life (t₁/₂b) between articaine and lidocaine. The elimination half life for lidocaine is 90 min, versus that for articaine of 20 min[6]. This is the time it takes to reduce the plasma levels of the drug by 50%. The major metabolic product of articaine is articainic acid. It is inactive as a local anesthetic, and systemic toxicity has not been observed [2]. This finding is important because an active metabolite may affect toxicity and may exert undesirable side effects. In comparison, lidocaine has active metabolites. It is metabolised in the liver by the microsomal P450 enzyme system to monoethylglyceine and xylidide; xylidide is a local anesthetic and potentially toxic[1].

**DISTRIBUTION**

Local anesthetics are distributed throughout the body to all tissues once it enters the bloodstream. The level of a local anesthetic drug in the blood from which it is distributed to certain target tissues/organs has a significant bearing on the potential toxicity of the drug. The blood level of the drug is influenced by the following factors:

- Rate at which the drug is absorbed into the cardiovascular system
- Rate of distribution of the drug is better in healthy patient
- Elimination of the drug through metabolic and/or excretory pathways.

**FACTORS AFFECTING LOCAL ANESTHETIC ACTION**

The dissociation variable (pKa) affects the onset of action. Lesser the pKa means that more uncharged base molecules are present to diffuse throughout the nerve sheath; thus onset time is decreased. Lipid solubility affects the anesthetic effectiveness. Increased lipid solubility permits the anesthetic to penetrate the nerve membrane, which itself is 90% lipid[1] more easily. Articaine differs from all other amide local anesthetics in that it is derived from thiophene. As a product, the articaine molecule does not have a benzene ring like the others but instead contains a thiophene ring. This renders the molecule more lipid soluble and therefore better able to cross lipid barriers like the nerve membrane [6]. Protein binding affects the duration. Increased protein binding allows anesthetic cations to be more firmly attached to proteins located at receptor sites. Thus the duration of action is increased. Vasodilator activity affects both the anesthetic potency and the duration. Superior vasodilator activity leads to increased blood flow to a region, which leads to a rapid removal of anesthetic molecules from the injection site. This will decrease both the anesthetic potency, and duration. Both articaine and lidocaine as basic solutions without a vasoconstrictor added would be ineffective and more toxic because of their vasodilator activity. In order to improve both the interval and safety, the addition of a vasoconstrictor like adrenaline as a supplementary is commonly seen in most preparations.

**DISCUSSION**

The properties very important for diffusion are molecular configuration and lipid solubility which is expressed by partition coefficient. Oertel et al (1997) reported a partition coefficient for articaine of 52, similar to that of lidocaine (N-octanol/buffer, 37°C) [2]. Borchard (1978) found that the partition coefficient for lidocaine was 2.9 (N-Heptan/buffer pH 7.4) and that for articaine was 32 (Octanol-1/buffer pH 7.35) [8]. Articaine contains a thiophene ring instead of benzene like lidocaine. This gives the molecule better diffusion properties compared with lidocaine. Malamed et al (2001) compared the efficacy of 4% articaine with adrenaline 1:100000 with 2% lidocaine with adrenaline 1:100000 using a total of 882 subjective investigators assessed via a
visual analog scale. The volumes and duration of anesthesia were as listed in table below. They concluded that there were no significant differences between subjects receiving articaine and those receiving lidocaine, either for subjects or investigator ratings. This findings is similar to that obtained by Vehetalo & al (1993) [9]

<table>
<thead>
<tr>
<th>Parameter</th>
<th>4% articaine with adrenaline 1:100000</th>
<th>2% lidocaine with adrenaline 1:100000</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>Simple 675</td>
<td>Complex 207</td>
</tr>
<tr>
<td></td>
<td>Simple 338</td>
<td>Complex 104</td>
</tr>
<tr>
<td>Mean volume +/- SEM, ml</td>
<td>2.5 +/- 0.07</td>
<td>4.2 +/- 0.15</td>
</tr>
<tr>
<td></td>
<td>2.6 +/- 0.09</td>
<td>4.5 +/- 0.21</td>
</tr>
<tr>
<td>Mean dose +/- SEM (mg/kg)</td>
<td>1.48 +/- 0.042</td>
<td>2.36 +/- 0.094</td>
</tr>
<tr>
<td></td>
<td>0.80 +/- 0.031</td>
<td>1.26 +/- 0.065</td>
</tr>
<tr>
<td>Range, min</td>
<td>0 - 217</td>
<td>1 - 215</td>
</tr>
<tr>
<td></td>
<td>0 - 220</td>
<td>1 - 171</td>
</tr>
</tbody>
</table>

Another important issue is the concentration of adrenaline. The effectiveness of 4% articaine with either 1:100000 or 1:200000 adrenaline for inferior alveolar nerve block are the same (Tofoli et al 2003) [10]. Therefore 1:200000 dilution of adrenaline is the recommend concentration for dental procedures (Jacob 1989), except for those procedure (e.g surgical interventions) that requires a larger degree of hemostasis. For these purposes the recommended concentration is 1:50000 (Buckley et al 1984) [11] or 1:80000 as used in Scandinavia.

CONCLUSION

In conclusion, considering the efficacy, biology and safety profile of the two drugs, Articaine is proven to be better compared to that of Lidocaine for performing dental procedures.

REFERENCES