Anesthetic Efficacy of Inferior Alveolar Nerve Block in Patients with Irreversible Pulpits after Premedication with Two Types of Analgesics

(An In Vivo study)

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Dedication

This work is dedicated to

My father & mother, to whom I owe everything I ever did and will achieve

My sweet wife, for continuous support

My dear brothers

My lovely kid, for being the joy of my life

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INTRODUCTION

Control of pain is of vital importance in dentistry. Local anesthesia is the primary method used in dentistry to control patient pain inside dental clinic. The inferior alveolar nerve block (IAN) is the most commonly used block in dentistry. However, anesthetizing mandibular teeth with IAN has been regarded as one of the most techniqually difficult local anesthesia injections. It was suggested that the success rate of IAN is 75–90% in patients with un inflamed pulp tissue (1). However, local anesthetics are generally much less effective when administered to patients with inflamed tissue (2). Therefore, it will be of great advantage to improve the success rate of IAN in endodontics.

Many theories have been hypothesized to explain the failure of local anesthetics. One of these theories is the prostaglandin-induced sensitization of peripheral nociceptors (3). Based on this theory one might conclude that since Ibuprofen or acetaminophen reduce the amount of prostaglandins, the pretreatment with these drugs may increase the effectiveness of local anesthetics.

Therefore conducting a study to compare and evaluate the effect of preoperative oral medication of

ibuprofen or combination of ibuprofen and acetaminophen on anesthesia efficacy of inferior alveolar nerve block in patients with irreversible pulpits will be of value.

In this part of thesis we will see a general over view about local anesthetics mechanism of action and properties. Lidocaine which is the local anesthetic solution used in the study will be discussed. Ibuprofen and acetaminophen with focusing on their combinations will be summarized. The review will then be focused on mandibular anesthesia technique, prevalence of failure, hypotheses for local anesthetic failure. Finally, therapeutic approaches for managing local anesthetic failures will be disscused.

LOCAL ANESTHETICS

Local anesthesia has been defined as a loss of sensation in an area of the body caused by a depression of excitation in nerve endings or an inhibition of the conduction process in peripheral nerves (4). Local anesthetic solutions have been utilized in clinical dentistry to eliminate pain associated with invasive procedures as early as the 19th century (5). An important requirement prior to initiating endodontic or operative dental treatment is the ability to achieve and maintain profound anesthesia. Local anesthetics are correctly considered to be the most important drugs used in clinical dentistry.

Mechanism of Action of Local Anesthetics

The primary action of a local anesthetic is the interference with the excitation-conduction process of nerve fibers (6). A nerve fiber has the capability to respond to a stimulus by excitation and to propagate this stimulus along the nerve fiber to its point of termination (7). This conduction of the stimulus is temporarily blocked by the action of the local anesthetic (6).

The electrophysiological properties of the neuronal membrane rely on both the permeability of the membrane to specific electrolytes and to the concentration of these electrolytes in the cytoplasmic and extracellular fluid (6). A nerve cell membrane is fully permeable to potassium and chloride ions in its resting state and relatively impermeable to proteins, amino acids, and sodium ions (4, 8). As a result of this selective permeability, cations (+) including sodium ions are concentrated extracellularly and anions (-), potassium ions, are concentrated intracellularly. The permeability of the nerve cell membrane along with the cytoplasmic and extracellular electrolyte concentrations combine to determine the electrophysiologic properties of the nerve cell membrane. The electrochemical gradient set up between the inside of the nerve membrane and the

outside results in an electrical potential of approximately -70 to -90 mV across the cell membrane (9). Stimulating the nerve results in increased sodium permeability through a transitory widening of the transmembrane channels. This widening allows sodium ions to rapidly diffuse to the interior of the cell resulting in depolarization of the neural cell membrane to a firing threshold of approximately -50 to60 mV. Upon reaching the firing threshold, sodium permeability increases remarkably and a rapid influx of sodium ions occurs across the cell membrane. At the end of the depolarization phase, the electrical potential is actually reversed across the membrane to approximately +40 mV (4, 8).

Once depolarization is complete, the permeability of the nerve membrane to sodium ions decreases and the high permeability to potassium is restored. The resulting movement of the sodium ions out, and the potassium ions in, by passive diffusion, restores the normal resting potential of the nerve cell membrane. When the refractory potential is achieved, there is excess of sodium ions intracellularly and of potassium ions extracellularly (9). The ‘sodium pump’ actively transports the excess sodium ions out of the cell. This process is energy dependent, the

source being adenosine triphosphate (ATP), which is oxidatively metabolized to provide the necessary energy (4, 9). Once the normal ionic gradient is restored, the nerve is again in its resting state. This repolarization process takes approximately 0.7 msec, after which the nerve cell membrane re-achieves its normal resting potential of approximately -90 mV (4).

The exact mechanism of action of local anesthetics is not known. The presently accepted theory on the action of local anesthetics is that they prevent depolarization by blocking the transmembrane sodium channels (4, 8). This is believed to be accomplished by either of the following mechanisms: the specific receptor mechanism and/or the membrane expansion mechanism (4, 8).

The specific receptor theory is based on four proposed binding sites within the sodium channel, to which local anesthetic molecules can attach. Molecules may bind to the inner mouth of the channel pore resulting in a tonic block. Binding to a second site deeper within the pore will result in a use-dependent block (10). The other two proposed sites are located at the gate of the sodium channel. Only the charged, or ionized, forms of the local anesthetic can bind to these sites and this form is unable to cross the nerve

membrane (9,10). The unionized form of the local anesthetic diffuses across the membrane thus establishing a chemical equilibrium. Once the unionized form of the local anesthetic has entered the cell, approximately 75% of it is converted to the ionized form. It is this ionized form of the anesthetic molecule that is capable of binding to the receptor sites resulting in decreased membrane permeability to sodium and leading to a prevention of the firing of the membrane (4,9).

The second theory, known as the membrane expansion theory, states that the anesthetic agent acts by penetrating the nerve membrane, resulting in an expansion of the membrane and a decrease in the diameter of the sodium channel, thereby preventing sodium permeability (4,9). This theory offers an explanation for the action of anesthetics that do not exist in the ionized form such as benzocaine (4).

“However, molecular research has demonstrated the existence of at least nine subtypes of voltage-gated sodium channels (VGSCs) that differ in their expression pattern, biophysical properties, and roles in mediating peripheral pain. These channels have a clear clinical relevance.(11) The broad class of VGSCs can be divided into channels

that are blocked by a toxin (tetrodotoxin [TTX]) and those that are resistant to the toxin (TTX-R). Most TTX-R channels are found primarily on nociceptors (e.g., Nav 1.8 and Nav 1.9). (12)These channels also are relatively resistant to local anesthetics (13) and are sensitized by prostaglandins.The presence of TTX-R sodium channels may explain why local anesthetics are less effective when injected into patients with odontalgia. Moreover, the sensitization of these channels by prostaglandins suggests that rapid-acting nonsteroidal anti-inflammatory drugs (NSAIDs) may be useful as a pretreatment to enhance the efficacy of local anesthetics in patients with odontogenic pain. (14)" (15)

Factors affecting anesthetic properties

The properties of local anesthetics are dependent on its lipid solubility, protein binding capacity, pKa, pH, tissue diffusibility, and intrinsic vasodilating properties (6).

The potency of the anesthetic compound is primarily determined by its ability to penetrate the nerve cell membrane, which is directly related to its lipid solubility. Highly lipid soluble anesthetic compounds can easily penetrate the nerve membrane thereby requiring lower concentrations to produce adequate anesthesia (9,16,17).