

Ultrasound Guided Transversus Abdominis Plane (TAP) Block versus Caudal Block in Pediatrics Undergoing Inguinal Hernia Repair

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List of Abbreviations

AAG	Alpha 1 acid glycoprotein
ACLS	Advanced cardiovascular life support
ASRA	American society of regional anesthesia
CNS	Central nervous system
CSF	Cerebrospinal fluid
CVS	Cardiovascular system
Fe+2	Ferrous iron
Fe+3	Ferric iron
G	Gauge
GA	General anesthesia
H	Hour
Hb.....	Hemoglobin
IM	Intramuscular
IV	Intravenous
K+	Potassium
kg.....	Kilogram
LA	Local Anesthetics
ml	Milliliter
mm	Millimeter
n.....	Number
Na+	Sodium
NaHCO3	Sodium bicarbonate
NSAID	Non steroidal anti-inflammatory drugs
PACU	Post anesthesia care unit
PDPH	Postduralpuncture headache
SD	Standard deviation
TAP	TransversusAbdominis Plane
US	Ultrasound

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Introduction

Although general anesthesia (GA) is the commonly used technique in children, regional anesthesia is used as an adjuvant for intraoperative and postoperative pain relief (*Ahmed and Rayan, 2016*).

Caudal analgesia is the most common regional technique performed in children. It has been used for many years as an adjuvant to GA and to provide postoperative analgesia for subumbilical procedures (*Ahmed and Rayan, 2016*).

However in recent times, there is a trend toward the use of peripheral nerve blockade wherever applicable, given the lower incidences of adverse effects when w with neuroaxial techniques. Furthermore there may be specific anatomic variations or abnormalities which preclude the use of caudal blockade (*Kanojia and Ahuja, 2015*).

Of the various peripheral nerve block techniques available, the TAP block is a rapidly evolving peripheral nerve block technique that provides effective analgesia during the postoperative period following abdominal surgeries. The intra and postoperative analgesic efficacy of TAP block has been successfully described in adult patients undergoing abdominal surgeries such as colonic resection, total abdominal hysterectomy and appendectomy (*Kanojia and Ahuja, 2015*).

Few studies on children have been done by some authors who concluded that the use of TAP block is a good

alternative in pediatric patients for postoperative pain management in lower abdominal and infraumbilical surgeries. It can be performed using a landmark technique through the lumbar triangle or with ultrasound (US) guidance. Local anesthetic (LA) is injected between the transverses abdominis and the internal oblique muscles (*Sahin et al., 2013*).

Aim of The Work

The aim of the study is to compare the effectiveness and safety of US guided TAP block versus caudal block as a part of multimodal analgesia in children undergoing inguinal hernia repair.

Local Anesthetics

Introduction:

Local anesthetics (LA) are drugs that block conduction of electrical impulses in excitable tissues. These tissues include the nerve cells and myocytes (both cardiac and skeletal muscles). Analgesia and anesthesia occur as a result of the blockage of electrical impulses (*Mumba et al., 2017*).

Nerve Conduction:

The resting membrane potential of a nerve cell is in the range of -60 to -70 mV. At rest, neurons are more permeable to potassium (K^+) ions due to the presence of K^+ leak channels. This explains why the resting neuronal membrane potential is closer to the equilibrium potential of K^+ of -80 mV. The intracellular milieu of the nerve cell is negatively charged relative to the extracellular. Upon excitation of the nerve fibres, the electrical impulse propagates along the axon as a result of changes occurring in the adjacent membrane alternating from negative to positive values of about $+50$ mV due to rapid influx of Na^+ ions. At an electrical potential of $+50$ mV, there is rapid efflux of K^+ ions in an attempt to maintain electrical neutrality of the cell. To restore the resting membrane potential, the Na^+/K^+ ATPase pumps Na^+ extracellularly, while the opposite happens to the K^+ . The conduction of impulses along nerve fibers occurs as small brief, localized spikes of depolarization on the surface of the cell

membrane. Impulses travel in one direction as the axonal membrane that has just undergone depolarization remains in the refractory state until the resting potential is restored by the Na^+/K^+ ATPase pumps (*Mumba et al., 2017*).

Because the rapid influx of Na^+ ions occurs in response to a change in the transmembrane potential, Na^+ channels in the nerve are characterized as “voltage gated.” These channels are protein structures with three subunits that penetrate the full depth of the membrane bilayer and are in communication with both the extracellular surface of the nerve membrane and the axoplasm (interior) of the nerve. LA prevent the generation and conduction of nerve impulses by binding to the α subunit of the Na^+ channel and preventing the influx of Na^+ into the cell, halting the transmission of the advancing wave of depolarization down the length of the nerve (*Gadsden, 2013*).

A resting nerve is less sensitive to a local anesthetic than a nerve that is repeatedly stimulated. A higher frequency of stimulation and a more positive membrane potential cause a greater degree of transmission block. These frequency- and voltage- dependent effects of local anesthetics occur because repeated depolarization increases the chance that a local anesthetic molecule will encounter a Na^+ channel that is in the activated, or open, form-as opposed to the resting form-which has a much greater affinity for LA. In general, the rate of dissociation from the receptor site in the pore of the Na^+ channel is

critical for the frequency dependence of LA action (*Gadsden, 2013*).

Pharmacology of local anesthetics:

Structure-activity relationship of local anaesthetics:

Local anesthetics consist of a hydrophilic amine and a lipophilic aromatic ring connected by an intermediate chain. The structural bond in the intermediate chain determines whether the local anesthetic will be classified as an ester or an amide, (Fig 1). Furthermore, the bond in the intermediate chain determines the pathway of metabolism of the compound. Ester local anesthetics are metabolised by plasma pseudocholinesterases, whereas the amides are metabolised in the liver by the cytochrome family of enzymes (*Mumba et al., 2017*).

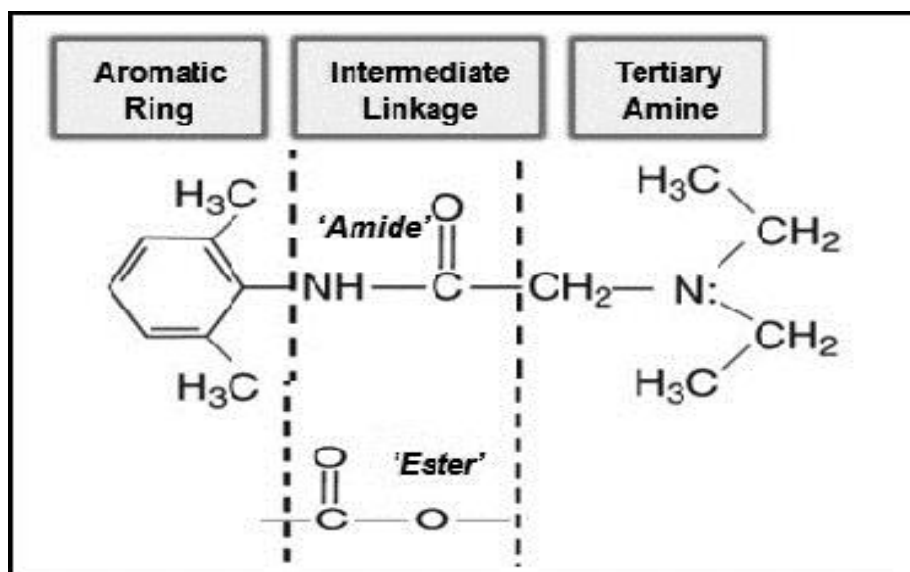


Figure 1:Structure of local anesthetics (*Becker and Reed, 2012*)

Mechanism of action of local anesthetics:

LA block the transmission of nerve impulses by reversibly blocking the fast voltage-gated Na channels, thereby inducing analgesia and anesthesia. Physico-chemically, LA are weak bases that are formulated in an acidic milieu, hence containing a larger proportion of the drug in the ionised state. However, it is the unionised fraction that is able to cross the lipid bilayer neuronal membrane and block the voltage-gated Na⁺ channels from the inside of the axoplasm. This blockade renders the Na⁺ channel inactive, and hence, no further conduction of impulses occurs (*Mumba et al., 2017*).

Determinants of physiological activities of local anesthetics:

The activity of LA is influenced by a number of factors. These include the pH of the surrounding tissue, the lipid solubility of the LA, pKa, the bond in the intermediate chain and its length and the protein binding of the particular LA in question. Details of how each of these factors influence the activity of LA is discussed below:

1. pKa:

The pKa is the pH at which the number of ionised and unionised fractions of the drug is in equilibrium (the pH at which 50% of the drug is ionized and 50% is present as base). The pKa of the LA is related to pH and the concentrations of the cationic and base forms by the Henderson-Hasselbalch equation: $\text{pH} = \text{pKa} + \log \left(\frac{[\text{base}]}{[\text{cation}]} \right)$ (*Mumba et al., 2017*).

The pKa generally correlates with the speed of onset of action of most amide LA drugs; The lower the pKa, the

more the unionised fraction is present for any given pH and hence the faster the onset of action (*Gadsden, 2013*).

2. pH:

The lower the pH, the less the potency because in acidic conditions the ionised fraction predominates, there is less of the unionised fraction, and there is less of the LA available to cross the lipid bilayer and block the voltage gated Na⁺ channels. This explains why LA does not have much efficacy in reducing pain in infected tissues like abscesses in which the pH of such tissues is much lower than the physiological pH of 7.4 (*Gadsden, 2013*).

For this reason, sodium bicarbonate (NaHCO₃) is often added to LA. This increases the amount of drug in the base form, which slightly shortens the onset time. Obviously, the limiting factor for pH adjustment is the solubility of the base form of the drug. Unfortunately, only small changes in pH can be achieved by the addition of bicarbonate because of the limited solubility of the base. As such, only small decreases in onset time are realized. For instance, with the alkalinisation of bupivacaine, an increase in the amount of base in solution is limited by the minimal solubility of free base in solution. For each LA, there is a pH at which the amount of base in solution is maximal (a saturated solution). Further increases in pH result in precipitation of the drug and do not produce an additional shortening of onset time (*Gadsden, 2013*).

3. Lipid solubility:

The more lipid soluble the local anaesthetic is, the higher the potency, the faster the onset of action and the longer the duration of action. This is because there are more drug molecules able to cross the lipid bilayer of the neuronal membrane and create a 'depot' of the drug from within the axoplasm (*Mumba et al., 2017*).

4. Intermediate chain:

The longer the intermediate chain, the more potent the local anaesthetic. Bupivacaine has a longer intermediate chain compared to lidocaine. Bupivacaine is three to four times more potent than lidocaine (*Mumba et al., 2017*).

5. Protein binding:

LA are in large part bound to plasma and tissue proteins. However, they are pharmacologically active only in the free, unbound state. The most important binding proteins for LA in plasma are albumin and alpha1-acid glycoprotein (AAG). The binding to AAG is characterized as high-affinity but low-capacity binding; hence LA bind to AAG preferentially compared with albumin. However, binding to AAG is easily saturated with clinically achieved blood levels of LA. Once AAG saturation occurs, any additional binding is to albumin. Albumin can bind LA drugs in plasma in concentrations many times greater than those clinically achieved (*Gadsden, 2103*).

LA with higher degrees of protein binding have longer duration of action, bupivacaine