INTRODUCTION

In all areas of anesthesia, safety and efficiency are valued goals, and in developing countries additional challenges due to shortages of anesthetic drugs, supplies and monitoring equipment may be present. Caudal epidural anesthesia in developing countries, can in combination with general anesthesia or alone provide safe, reliable and efficient analgesia and / or anesthesia for both high risk and general pediatric surgical patients ***(Silvani et al., 2006)***.

These techniques can be easily learnt and may be modified to extend analgesia into the postoperative period (with the addition of opioids or continuous techniques) or replace general anesthesia in circumstances where either the equipment or general anesthetic techniques are not available. The following manuscript will describe the pharmacological and physiologic basis of caudal epidural anesthesia, techniques for administration, monitoring, and specific modifying techniques of caudal epidural anesthesia and a discussion of complications and contraindications to caudal epidural anesthesia in pediatric patients ***(Silvani et al., 2006)***.

There are many parameters used to evaluate the effectiveness of caudal anesthesia which applied for postoperative analgesia, two of those parameters are MBP and HR. Generally, at the beginning of a surgical procedure, a 15-

20% or more increase of these two parameters compared to baseline is considered to be an insufficient block. Effects of local anesthetics on MBP and HR values are similar and no significant difference was found ***(Ivani et al., 2005)***.

AIM OF THE WORK

This study is mainly directed to update the knowledge about caudal anesthesia in pediatric lower limb procedures, and to highlight its effect on reducing intraoperative anesthetic requirements and postoperative analgesia.

ANATOMY OF CAUDAL CANAL

The caudal (sacral) canal extends from the upper border of sacral bone (in relation to lumbar epidural space) to the sacral hiatus. Whole of this canal is enclosed in sacral bone ***(Miller et al., 2010)***.

Sacrum

The sacrum (figure 1) is a triangular bone that articulates with the fifth lumbar vertebra, the coccyx and the ilia. The dorsal roof consists of the fused laminae of the five sacral vertebrae and is convex dorsally. In the midline is a median crest which represents the sacral spinous processes. Lateral to this is the intermediate sacral crest with a row of four tubercles which represent the articular processes. The S5 processes are remnants and form the cornua, which provide the main landmarks for indentifying the sacral hiatus. The hiatus is covered by the sacro-coccygeal membrane. The canal contains areolar connective tissue, fat, sacral nerves, lymphatics, the filum terminale and a rich venous plexus ***(Weber and Wulf, 2003)***.

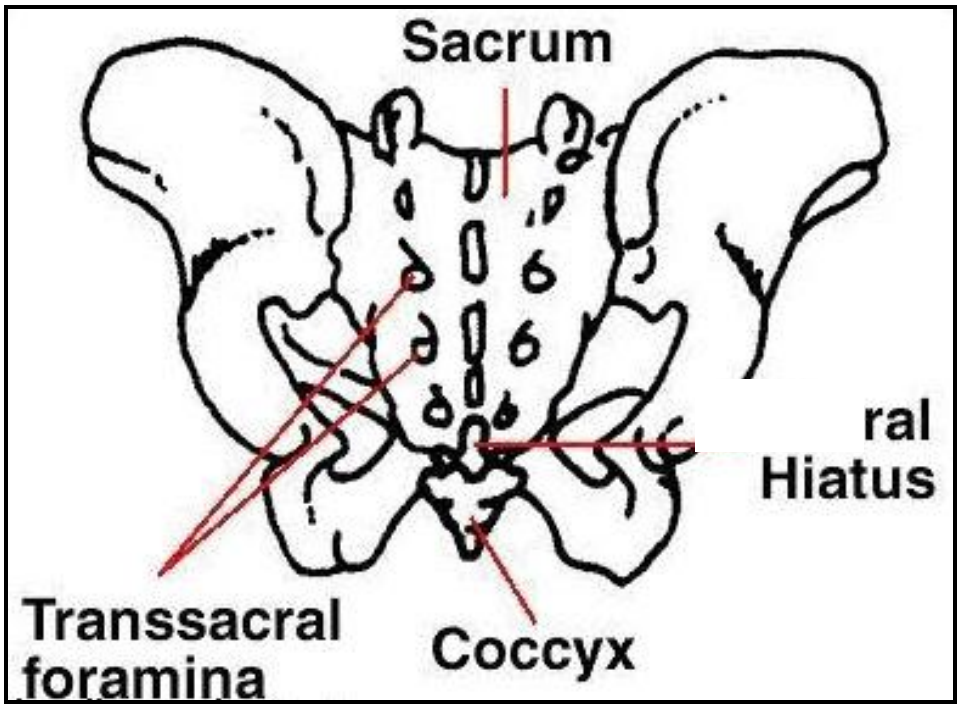

Figure (1): Anatomy of sacrum ***(Weber and Wulf, 2003).***

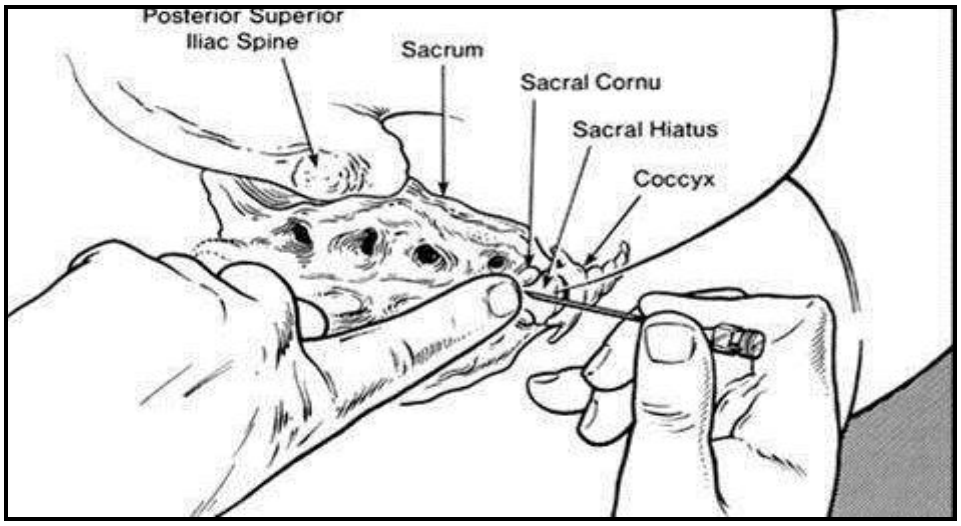

Figure (2): The caudal epidural space is the lowest portion of the epidural system and is entered through the sacral hiatus ***(Weber and Wulf, 2003)***.

Coccyx

Coccyx, otherwise called tail bone gets attached superiorly to sacrum & inferiorly to anococcygeeal ligament. Coccyx is actually made up of 3 to 5 vestigeal remnants of vertebrae. The inferior portion of coccyx is mobile & prone for fractures. Coccyx curves anteriorly & superiorly. Ganglion impar is situated at the junction of sacrum & coccyx ***(Chen et al., 2004)***.

Sacral (caudal) canal

This canal has a total volume of 30-35ml in adults after the contents have been evacuated (cadaver). The spinal cord ends at the lower border of L_1 vertebra, subsequently the sacral & coccygeal nerves pass to sacral canal as cauda equina. The dura ends at S_2 level & the pia continues as filum terminale to get attached to coccyx. The sacral nerve roots (upper four) anterior rami exit through anterior sacral openings & posterior rami through posterior sacral openings. As mentioned earlier, the fifth sacral nerve & coccygeal nerves traverse the canal & pierce through sacrococcygeal membrane to come out. The vertebral venous plexus continues into sacral canal, they are more concentrated towards anterior surface. The remainder of the sacral canal is filled with adipose tissue, which is subject to an age-related decrease in its density. This change may be responsible for the transition from the predictable spread of local anesthetics administered for caudal anesthesia in children to the limited and unpredictable segmental spread seen in adults. The

sacral hiatus is covered only by skin, a subcutaneous fatty layer and the sacrococcygeal membrane. The most distal portion of the dural sac and the sacral hiatus usually terminates between levels S1 and S3. ***(Figure 3)***. Anomalies in the position of the dural sac reflection can result in unintended dural puncture ***(Uemura and Yamashita, 1992)***.

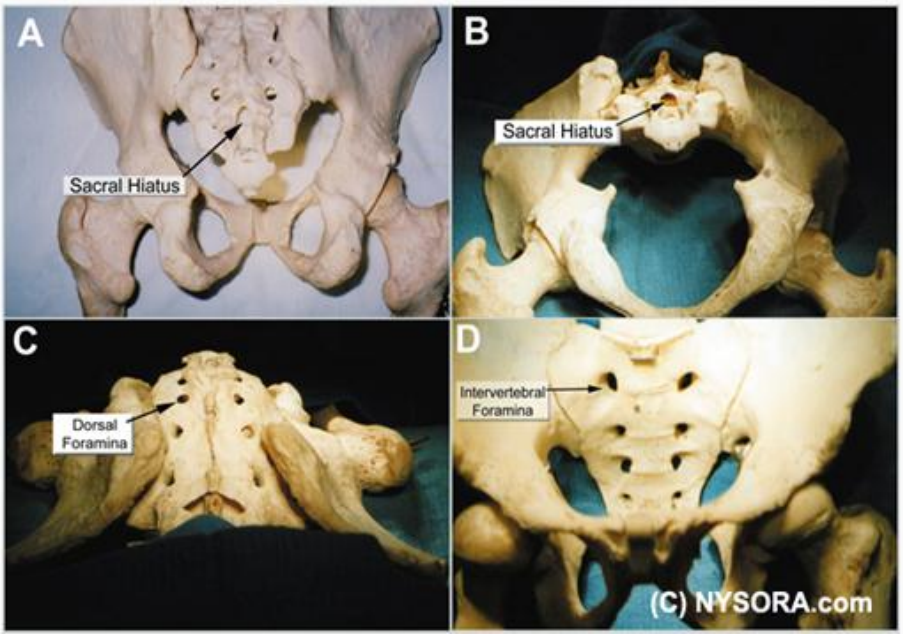

Figure (3): A: Skeletal model demonstrating the sacral hiatus and its relationship to the coccyx and sacrum. The fifth inferior articular processes project caudally and flank the sacral hiatus as sacral cornuae. **B:** Skeletal specimen viewed from inferior to the sacral hiatus. The hiatus is seen as the oval shaped opening at the 12 o' clock position in the photograph. **C:** Skeletal specimen of the sacrum viewed from craniad to caudad demonstrating the five dorsal foramina, situated bilaterally. **D:** Skeletal specimen of the sacrum demonstrating the ventral sacral surface. Note the five bilateral intervertebral foramina, paired on either side of the midline, defined by the retention screws used to hold the specimen together ***(Candido and Winnie, 2013)***.

The lowest margin of the filum terminale emerges at the sacral hiatus and traverses the dorsal surface of the fifth sacral vertebra and the sacrococcygeal joint to reach the coccyx. The fifth spinal nerves also emerge through the hiatus medial to the sacral cornua. The sacral canal contains the epidural venous plexus, which generally terminates at S4, but which may continue more caudally. Most of these vessels are concentrated in the anteriolateral portion of the canal. The remainder of the sacral canal is filled with adipose tissue, which is subject to an age-related decrease in its density. This change may be responsible for the transition from the predictable spread of local anesthetics administered for caudal anesthesia in children to the limited and unpredictable segmental spread seen in adults ***(Candido and Winnie, 2013)***.

Considerable variability occurs in sacral hiatus anatomy among individuals of seemingly similar backgrounds, race, and stature. As individuals age, the overlying ligaments and the cornua thicken significantly. The hiatal margins often defy recognition by even skilled fingertips. The practical problems related to caudal anesthesia are mainly attributable to wide anatomic variations in size, shape, and orientation of the sacrum ***(Candido and Winnie, 2013)***.

The sacral foramina afford anatomic passages that permit the spread of injected solutions such as local anesthetics and adjuvants. The posterior sacral foramina are essentially sealed by the multifidus and sacrospinalis muscles, but the anterior

foramina are unobstructed by muscles and ligaments, permitting ready progress of solutions through them ***(Candido and Winnie, 2013)***.

The sacral curvature also varies substantially. This variability tends to be more pronounced in males than in females. The clinical significance of this finding is that a noncurving epidural needle will more likely pass easily into the canal of females than males. The angle between the axis of the lumbar canal and the sacral canal varies between 7 and 70 degrees in subjects with marked lordosis. The clinical implication of this finding is that the cephalad flow of caudally injected solutions may be more limited in lordotic patients with exaggerated lumbosacral angles than in those with flatter lumbosacral angles, in whom the axes of the lumbar and sacral canals are more closely aligned ***(Candido and Winnie, 2013)***.

The sacral canal contains:

1. The terminal part of the **dural sac**, ending between S1 and S3.
2. The five sacral nerves and coccygeal nerves making up the **cauda equina**. The sacral epidural veins generally end at S4, but may extend throughout the canal. They are at risk from catheter or needle puncture.
3. The **filum terminale** - the final part of the spinal cord which does not contain nerves. This exits through the sacral hiatus and is attached to the back of the coccyx.

4. **Epidural fat**, the character of which changes from a loose texture in children to a more fibrous close-meshed texture in adults. It is this difference that gives rise to the predictability of caudal local anesthetic spread in children and its unpredictability in adults.

Even though caudal epidural block (CEB) has a wide range of clinical applications, it is sometimes hard to determine the anatomical location of the sacral hiatus and the caudal epidural space, especially in adults. The determination of the landmarks by the clinician enables the sacral hiatus to be ascertained and may increase the success rate of CEB ***(Morgan et al., 2002)***.

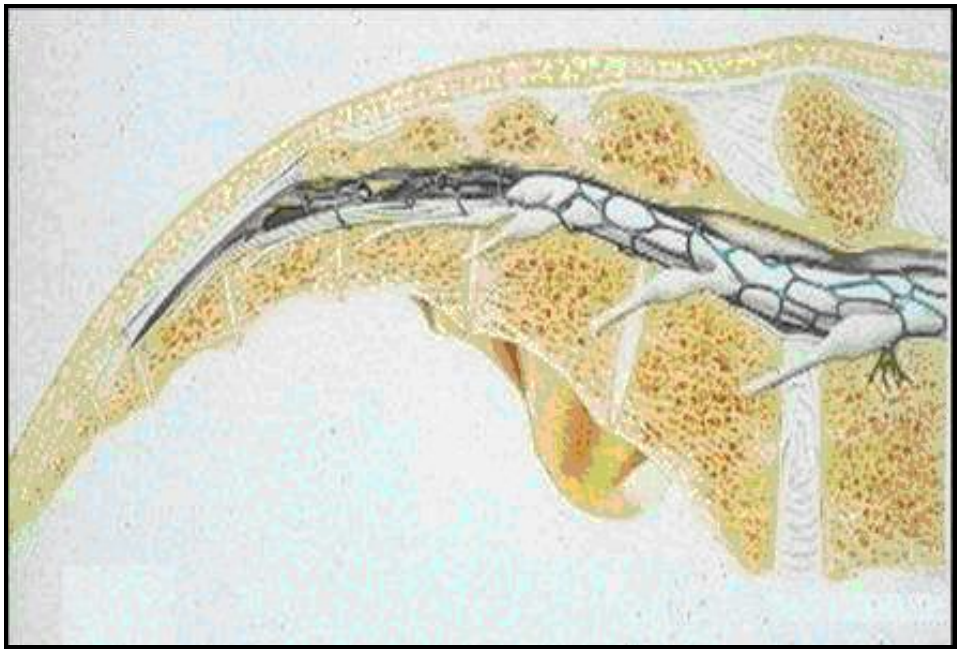

Figure (4): The sacrum is cartilaginous in infants and children which can allow for inadvertent intra-osseous injection ***(Soliman, 2003).***

PHARMACOLOGY OF LOCAL ANESTHETIC DRUGS

Local anesthetics interrupt neural conduction by inhibiting the influx of sodium ions through channels or ionophores within neuronal membranes. Normally these channels exist in a resting state, during which sodium ions are denied entry. When the neuron is stimulated, the channel assumes an activated or open state, in which sodium ions diffuse into the cell, initiating depolarization. Following this sudden change in membrane voltage, the sodium channel assumes an inactivated state, during which further influx is denied while active transport mechanisms return sodium ions to the exterior. Following this repolarization, the channel assumes its normal resting state. An appreciation of these sodium channel states helps to explain the preferential sensitivity of local anesthetics for various classes of neuronal fibers ***(Becker and Reed, 2012)***.

Local anesthetics have greater affinity for receptors within sodium channels during their activated and inactivated states than when they are in their resting states. Therefore, neural fibers having more rapid firing rates are most susceptible to local anesthetic action. Also, smaller fibers are generally more susceptible, because a given volume of local anesthetic solution can more readily block the requisite number of sodium channels for impulse transmission to be entirely interrupted. For these reasons the tiny, rapid-firing autonomic fibers are most sensitive,

followed by sensory fibers and finally somatic motor fibers. The anesthesiologist blocking mixed spinal nerves is acutely aware of these differential sensitivities. As patients recover from spinal anesthesia they first regain voluntary motor function, then sensation returns, and finally they can micturate (autonomic control). The dentist is generally spared this consideration because the trigeminal nerve branches anesthetized for dental procedures are comprised only of small, rapid-firing sensory fibers. However, the many classes of sensory fibers also vary in their diameters and firing rates. For example, pain fibers are more sensitive than those carrying pressure and proprioception. A patient may remain disturbed by a sense of pressure despite complete anesthesia of pain fibers ***(Berde and Strichartz, 2009)***.

General properties of local anesthetics:

The molecular structure of all local anesthetics consists of 3 components: lipophilic aromatic ring, intermediate ester or amide linkage, and tertiary amine. Each of these components contributes distinct clinical properties to the molecule (figure 5) ***(Becker and Reed, 2012)***.

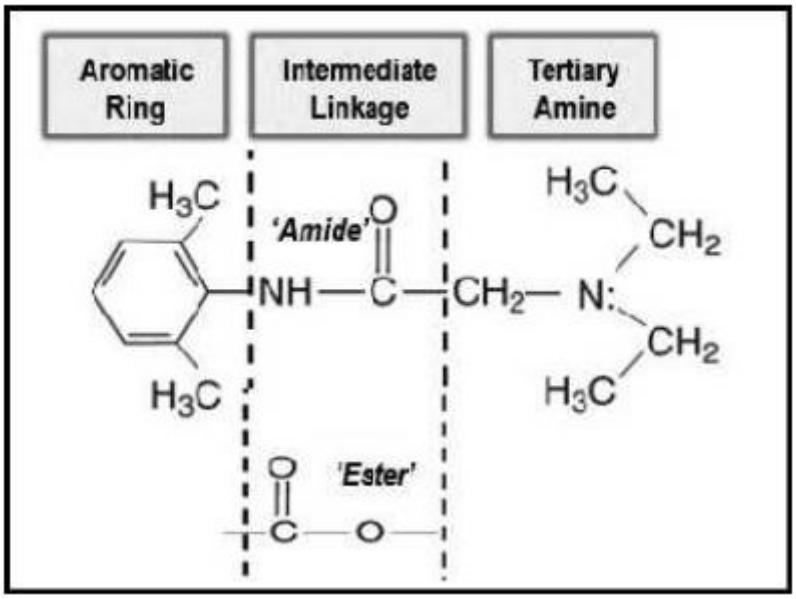

Figure (5): Local anesthetic structure.

Anesthetic potency:

Local anesthetics vary in their potency, allowing for concentrations that range typically from 0.5 to 4%.This is largely the result of differences in lipid solubility, which enhances diffusion through nerve sheaths and neural membranes. This property is determined by the aromatic ring and its substitutions, along with those added to the tertiary amine. For example, bupivacaine is more lipid soluble and potent than articaine, allowing it to be formulated as a 0.5%concentration (5mg/mL) rather than a 4% concentration (40mg/mL) ***(Becker and Reed, 2012)***.

Time for onset:

Greater lipid solubility of a drug not only enhances potency but also enables more rapid diffusion through cell

membranes. For local anesthetics, this hastens the onset for anesthesia in isolated fibers during in vitro studies, but it must be appreciated that other factors come into play clinically. For example, inherent vasodilating properties may promote systemic absorption before the anesthetic reaches the nerve membrane.

High lipid solubility may impede dispersion throughout tissue fluids and also fosters sequestration in neighboring adipose tissues or myelin sheaths. In either case, fewer numbers of molecules reach the neuronal membrane and onset is delayed. Therefore, unlike in vitro studies of isolated fibers, greater lipid solubility generally slows the onset of anesthesia in the clinical setting. Injecting higher concentrations that allow a greater number of molecules to reach the membrane and hasten onset can offset this influence. Although bupivacaine and articaine are both highly lipid soluble, the 4% concentration of articaine provides for a much faster onset ***(Becker and Reed, 2012)***.

Despite myriad factors that influence the quantity of local anesthetic reaching the nerve fibers, the most important factor that determines the onset of anesthesia is the proportion of these molecules that exist in a lipid-soluble rather than a water-soluble state. The terminal amine may exist in a tertiary form (3 bonds) that is lipid soluble, or as a quaternary form (4 bonds) that is positively charged and renders the molecule water soluble. For the local anesthetic base to be stable in solution, it is formulated as a hydrochloride salt. As such, the molecules exist in a quaternary, water-soluble state at the time of injection and are

unable to penetrate the neuron. Therefore, the time for onset of local anesthesia is directly related to the proportion of molecules that convert to the tertiary, lipid-soluble structure when exposed to physiologic pH (7.4). This proportion is determined by the ionization constant (pKa) for the anesthetic and is calculated using the Henderson-Hasselbalch equation:

log(cationic form=uncharged form)~pKa{pH

In simpler terms, if a local anesthetic were to have a pKa of 7.4 and to be injected into tissues having a physiologic pH of 7.4, 50% of the molecules would exist in the quaternary (cationic) form and 50% would exist in the tertiary (uncharged) form; only half the molecules would be lipid soluble and able to penetrate the neuron. Unfortunately, the pKa for all local anesthetics is greater than 7.4 (physiologic pH), and therefore a greater proportion of the molecules exist in the quaternary, water-soluble form when injected into normal tissue. The clinical caveat is that the higher the pKa for a local anesthetic, the fewer molecules are available in their lipid-soluble form. This will delay onset. Furthermore, the acidic environment associated with inflamed tissues lowers their pH well below 7.4 and favors the quaternary, water-soluble configuration even further. This has been suggested as one explanation for difficulty when attempting to anesthetize inflamed or infected tissues. In these situations, for example, bupivacaine (pKa 8.1) would be less desirable than mepivacaine (pKa 7.6) ***(Becker and Reed, 2012)***.