

**COMPARISON OF ANALGESIC EFFICACY OF CAUDAL
DEXMEDETOMIDINE VS CAUDAL TRAMADOL WITH ROPIVACAINE IN
PEDIATRIC INFRAUMBILICAL SURGERIES**

DISSERTATION SUBMITTED TO THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

M.D. BRANCH – X

**Register No.: 201720306
(ANAESTHESIOLOGY)**



**DEPARTMENT OF ANAESTHESIOLOGY
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MAY-2020

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PROTOCOL TITLE: COMPARISON OF ANALGESIC EFFICACY OF CAUDAL DEXMEDETOMIDINE VS CAUDAL TRAMADOL WITH ROPIVACAINE IN PEDIATRIC INFRAUMBILICAL SURGERIES
PRINCIPAL INVESTIGATOR: Dr.C.RAMPRAKASH, MBBS,
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Dear Dr.C.RAMPRAKASH, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 16.03.2018


THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form
2. Study Protocol
3. Department Research Committee Approval
4. Patient Information Document and Consent Form in English and Vernacular Language
5. Investigator's Brochure
6. Proposed Methods for Patient Accrual Proposed
7. Curriculum Vitae of The Principal Investigator
8. Insurance /Compensation Policy
9. Investigator's Agreement with Sponsor
10. Investigator's Undertaking
11. DCGI/DGFT approval
12. Clinical Trial Agreement (CTA)
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)
14. Clinical Trials Registry-India (CTRI) Registration

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

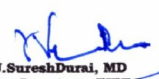
1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
5. The TIREC will monitor The study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
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 - f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
 - g. Any deviation/violation/waiver in The protocol must be informed.

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INTRODUCTION

Pain is perhaps the most dreadful symptom of disease and man has attempted to discover methods to get relieved from pain. It is an highly unpleasant sensation that only can be experienced but cannot be expressed particularly in children age group. The utmost important thing is that they can feel varying intensities of pain from similar type of tissue damage and they can feel pain without injury or with apparent injury. Children usually depend on their parents or care takers for their health well being. In addition children generally lack communicative ability.

The various techniques of allevating pain have remarkable side effects prohibiting their usage in children for example., narcotics - due to respiratory depression, the other analgesics which are usually avoided for sometime after general anaesthesia because of the fear of vomiting and aspiration, the objection to the use of needles in the case of analgesics which are parenterally administered. Regional anaesthetic method significantly reduces postoperative pain and decreases analgesic requirements. Caudal route is being selected for this study as this is one of the simpler and safer technique in Pediatric surgeries with a remarkable success rate.

Caudal block is usually performed after the induction of General anaesthesia and is used as an additional effect to Intraoperative anaesthesia which is more comfortable for surgeons and Postoperative pain relief for children undergoing

surgical procedures below umbilicus. In order to decrease the analgesic requirements and to increase the quality of block, various additives such as Clonidine, Neostigmine, Fentanyl, etc with Local anaesthetic have been studied during Caudal epidural blockade in single shot technique.

Ropivacaine is a local anaesthetic belonging to Amide group which is long acting with regards to duration of action. It has widely been used for Pediatric caudal block since it provides adequate pain relief with less duration of motor blockade. There is similarity in structure between Ropivacaine and bupivacaine. However, various studies suggests that Ropivacaine being less cardiotoxic than bupivacaine thus making it suitable agent for Caudal block.

Dexmedetomidine is an α_2 adrenergic receptor agonist with higher affinity for the receptor when compared to clonidine which is a partial agonist.

A striking feature of this drug is that it has greater selectivity for α_2A adrenergic receptors with affinity ratio ($\alpha_2 : \alpha_1$) being 1600:1. This feature accounts for the hypnotic and analgesic effects of Dexmedetomidine.

Tramadol, a synthetic 4- phenyl piperidine analogue of Codeine is a racemic mixture of two enantiomers , both of which contribute to analgesic activity through different mechanisms enhancing inhibitory effects on pain transmission in the spinal cord.

Objective of our study is to find out the analgesic efficacy of Dexmedetomidine and Tramadol when used as an anaesthetic adjuvant with

Ropivacaine for Caudal epidural anaesthesia in children undergoing below umbilicus surgeries.

AIM

PRIMARY OBJECTIVE: To compare the duration of analgesia of Ropivacaine with Dexmedetomidine and Tramadol administered caudally in Pediatric Infraumbilical surgeries.

SECONDARY OBJECTIVE: To assess the duration of motor blockade, postoperative sedation and incidence of any adverse effects.

REVIEW OF LITERATURE

A.M.El-Hennawy et al studied the analgesic and adverse effects of Caudal Dexmedetomidine added as adjuvant to Bupivacaine in children posted for lower abdominal surgeries and he found that dexmedetomidine added to bupivacaine increased the analgesic duration.

Mausumi neogi et al compared dexmedetomidine and clonidine as an anaesthetic adjuvant to ropivacaine for caudal epidural analgesia and he found that both the adjuvants added to ropivacaine increased the duration and quality of block.

Saadawy et al did a study on the analgesic effects of dexmedetomidine added to bupivacaine in Caudal epidural analgesia in pediatric patients and they showed that the above combination significantly increased the duration of analgesia with no significant adverse events.

G.Ivani et al. did a study to compare the postoperative analgesic efficacy of 0.2% plain Ropivacaine and 0.1% ropivacaine with Clonidine 2µg/kg added and they showed that drug combination of clonidine and ropivacaine significantly increased the duration of postoperative analgesia than the plain drug administered.

Obayah et al. studied the postoperative analgesic efficacy of dexmedetomidine added to bupivacaine in pediatric patients undergoing repair

of cleft palate with greater palatine nerve block and found that this significantly prolonged the analgesic duration without side effects.

P.A.Lonnqvist et al did a study on the pharmacokinetic properties of Ropivacaine administered caudally in children posted for surgery below the level of umbilicus and showed that Caudal Ropivacaine resulted in adequate relief of postoperative pain without significant motor blockade.

Alparslan Turan et al evaluated the efficacy of analgesia of Neostigmine added to Caudal Ropivacaine in children posted for genitourinary surgery and concluded that the above mentioned drug combination provides significant pain relief in the postoperative period.

Prosser DP et al evaluated the efficacy of postoperative analgesia of Tramadol added to Caudal Bupivacaine in children posted for Hypospadias corrective surgery and concluded that Tramadol failed to improve the duration of analgesia with slow onset of action too.

NM Solanki et al compared the duration of postoperative analgesia of Tramadol over Fentanyl added to Caudal Bupivacaine in pediatric patients and they concluded that Tramadol with bupivacaine significantly improved the quality of caudal block than bupivacaine with fentanyl.

HISTORY

1901 - "Regional analgesia" was coined by Harvey Cushing

SICARD & CATHELIN described epidural injection through sacral hiatus.

1920 - Zweifel was able to analyse 4200 caudal epidural injections recorded in literature.

1933 - Cambel M.F. first described sacral epidural block in children and infants.

1957 - Another milestone was synthesis of bupivacaine by Ekenstein et al

1963 - L.J. Tulivuo first used Bupivacaine clinically

1974 - Kay B used caudal block for post operative pain relief in children. Jean enthuse sicard and Fernard cathelin independently introduced cocaine through the sacral hiatus in 1901.

ANATOMICAL CONSIDERATION

The Anatomy of Sacrum :

The Sacrum is a large , triangular , flattened bone formed by joining of all five sacral vertebrae. It forms the posterosuperior part of the pelvis. It articulates with the hip bone on the either side at the sacroiliac joint. Sacrum has a base, an apex and four surfaces being Pelvic, Dorsal, Right and left lateral. The pelvic surface is smooth and concave. The dorsal surface is irregular and convex. The lateral surface is irregular and partly articular.

The sacrum is divided by rows of foramina into

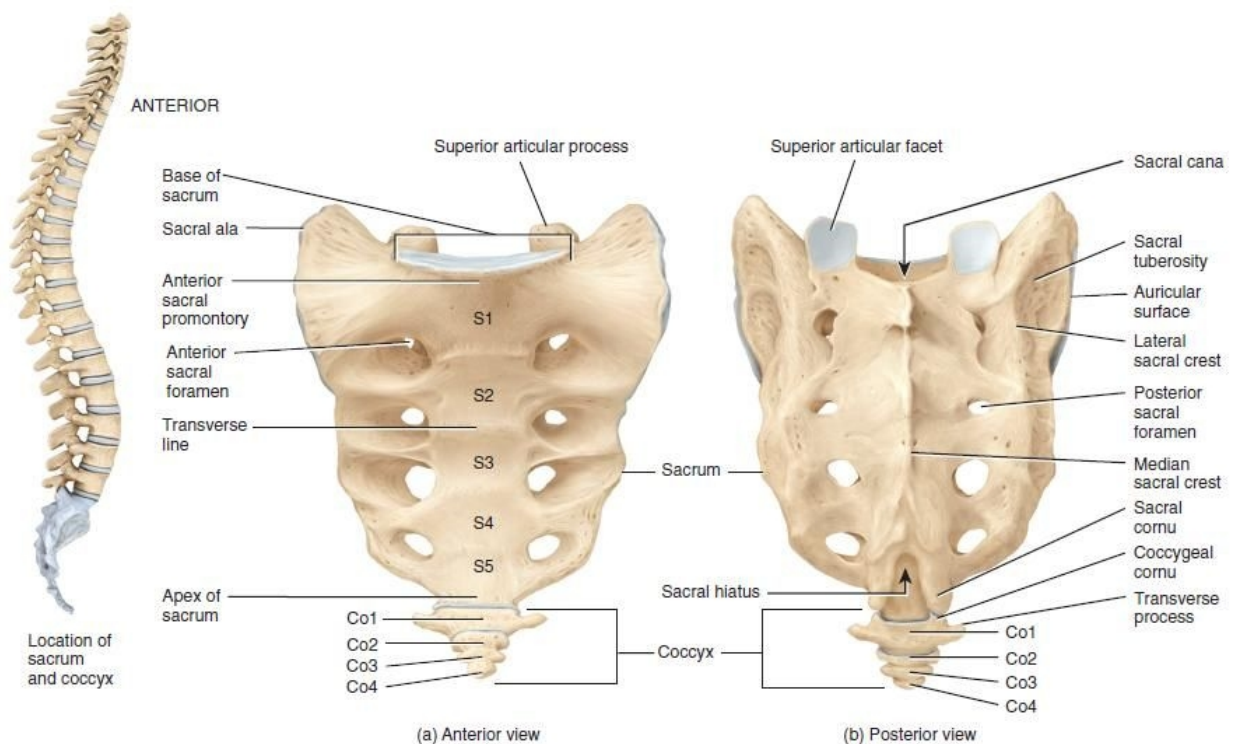
1. Median portion , traversed by sacral canal.
2. A pair of lateral masses formed by fusion of the transverse processes and the costal elements.

Base: It is formed by the upper surface of 1st sacral vertebra. The body is lumbar type. It articulates with 5th lumbar vertebra at the Lumbosacral joint. The projecting anterior margin is called the Sacral promontory. The surface slopes at an angle of 30 degrees. The base of the lateral masses forms a broad sloping surface spreading fan wise called the Ala of the sacrum.

Apex: It is formed by the inferior surface of the fifth sacral vertebra.

Pelvic surface: The median area is marked by four transverse ridges which indicate the line of fusion of the bodies of the five sacral vertebrae. These ridges end on the either side at the four sacral foramina which communicates with the sacral canal through the Intervertebral foramina.

Dorsal surface: In the median plane, it is marked by the Median Sacral crest which bears 3-4 spinous tubercles which represents the fused spines of the upper 4 sacral vertebrae. Below the 4th tubercle, there is an inverted U shaped opening in the posterior wall of the sacrum called the SACRAL HIATUS. It results due to failure of the lamina of the 5th sacral vertebra to meet posteriorly. The inferior articular processes of the 5th sacral vertebra are free and forms the SACRAL CORNUA which projects downwards at the sides of sacral hiatus.



Lateral surface: It is formed by the fused transverse processes and the costal elements of the sacral vertebrae. The upper wider part bears an L shaped auricular surface anteriorly, and a rough deeply fitted area posteriorly. The auricular surface is formed by the costal elements. The posterior pitted area is

formed by the transverse processes. The abrupt medial bend at the lower end of the lateral surface is called the Inferior lateral angle of the sacrum.

SACRAL CANAL: It is formed by the sacral vertebral foramina and is triangular in cross section. The upper end of the canal appears oblique but actually it is directed upwards in anatomical position. Inferiorly, the canal opens at the sacral hiatus, laterally it communicates through the intervertebral foramina with the pelvic and dorsal sacral foramina. The sacral canal contains the spinal meninges.

The filum terminale and the subdural and subarachnoid spaces end at the level of the 2nd sacral vertebra. Therefore the lower sacral nerves and the filum terminale pierce the dura and arachnoid at this S2 level.

ATTACHMENTS ON THE SACRUM:

1. The anterior and posterior longitudinal ligaments.
2. Ligamentum flavum
3. Ventral sacroiliac ligament
4. Piriformis
5. Interosseous ligament.
6. Gluteus maximus.
7. Sacrotuberous and sacrospinous ligaments.
8. Lateral coccygeal ligament.

STRUCTURES TRANSMITTED THROUGH THE FORAMINA

1. The pelvic sacral foramina transmits the Ventral rami of upper four sacral nerves and the lateral sacral arteries.
2. The dorsal sacral foramina transmit the dorsal rami of the upper four sacral nerves.
3. The 5th sacral nerve, the filum terminale and the coccygeal nerves emerge at the sacral hiatus.

CAUDAL ANAESTHESIA

Characteristics of equipment:

Reliability of the technique and the incidence of complications largely depend on the characteristics of the needle used. The four important characteristics of the needle

- Bevel
- Internal and external diameter
- Its length
- Presence of a stylet

Sharp bevelled Needle:

Advantage: Traverse easily through the tissues

Disadvantages:

1. Characteristic “give way” when sacrococcygeal membrane is punctured may not be clearly felt with sharp needles.
2. Sharp needles have long bevel advanced further into the epidural space so that it lies entirely within it.
3. Cartilaginous sacrum can be easily traversed by a sharp and long bevelled needle leading to rectal puncture or iliac vessel puncture.

Straight tipped needle with a bevel of 45 – 60 degree is ideal.

Diameter:

Small needles may bend & break during procedure. Thin needles may “give way”. Puncturing cartilaginous structures give rise to inadvertant

intraosseous injection which produces effect similar to I.V. Injection. It may enter pelvic viscera and cause damage. 21 to 23 Gauge is ideal because it is rigid and large enough to allow reflux of blood or cerebrospinal fluid.

Length:

Proximity of the dural sac makes it dangerous to use very long needles. Distance from the skin to the epidural space is almost always less than 20mm even in adults. So it is not advisable to use a needle longer than 30 mm. If needle with a stylet is used, it prevents the formation of an epidermoid tumour due to skin tag. Epidural needle with 20 to 22 gauges are employed when one intends to use an epidural catheter via caudal route to achieve anaesthesia at higher level after radiographic conformation.

Factors determining the quality of caudal block:

- Intensity of block achieved by type and concentration of local anaesthetic.
- Height of block which depends on the volume injected

Methods for determination of the volume of Local anaesthetic:

Formula based on weight or age:

Armitage formula - most commonly employed and easier to calculate

- ✓ Sacral dose - 0.5 ml per kg
- ✓ Lumbosacral - 1 ml per kg
- ✓ Thoracolumbar - 1.25 ml per kg

Schlute – Steinberg formula: Applicable for children with age from 8 to 12 years.

The volume of local anaesthetic is calculated as 0.1 ml per segment to be blocked per years of age. In children age group less than 7 years the formula goes by Required ml of local anaesthetic = $0.65 \times \text{number of spinal segments intended to block} \times \text{weight of the child in kilogram}$

Formula proposed by Spiegel:

Optimal volume of local anaesthetic solution (ml) is calculated as $4 + (\text{Distance between the sacral hiatus and spinous process of 7th cervical vertebra} - 15) / 2$

Takasaki's formula: This is yet another formula used for calculation of caudal dosage of local anaesthetic solution. It goes by the 0.056 ml per kilogram body weight per segment to be blocked.

The formula proposed by Armitage holds excellent results and being easier to calculate and apply practically. Hence in our clinical study we had used the same for formulating the dose of local anaesthetic to be given caudally.

Patient position: There are three positions possible for performing caudal block
Three positions are available for caudal anesthesia;

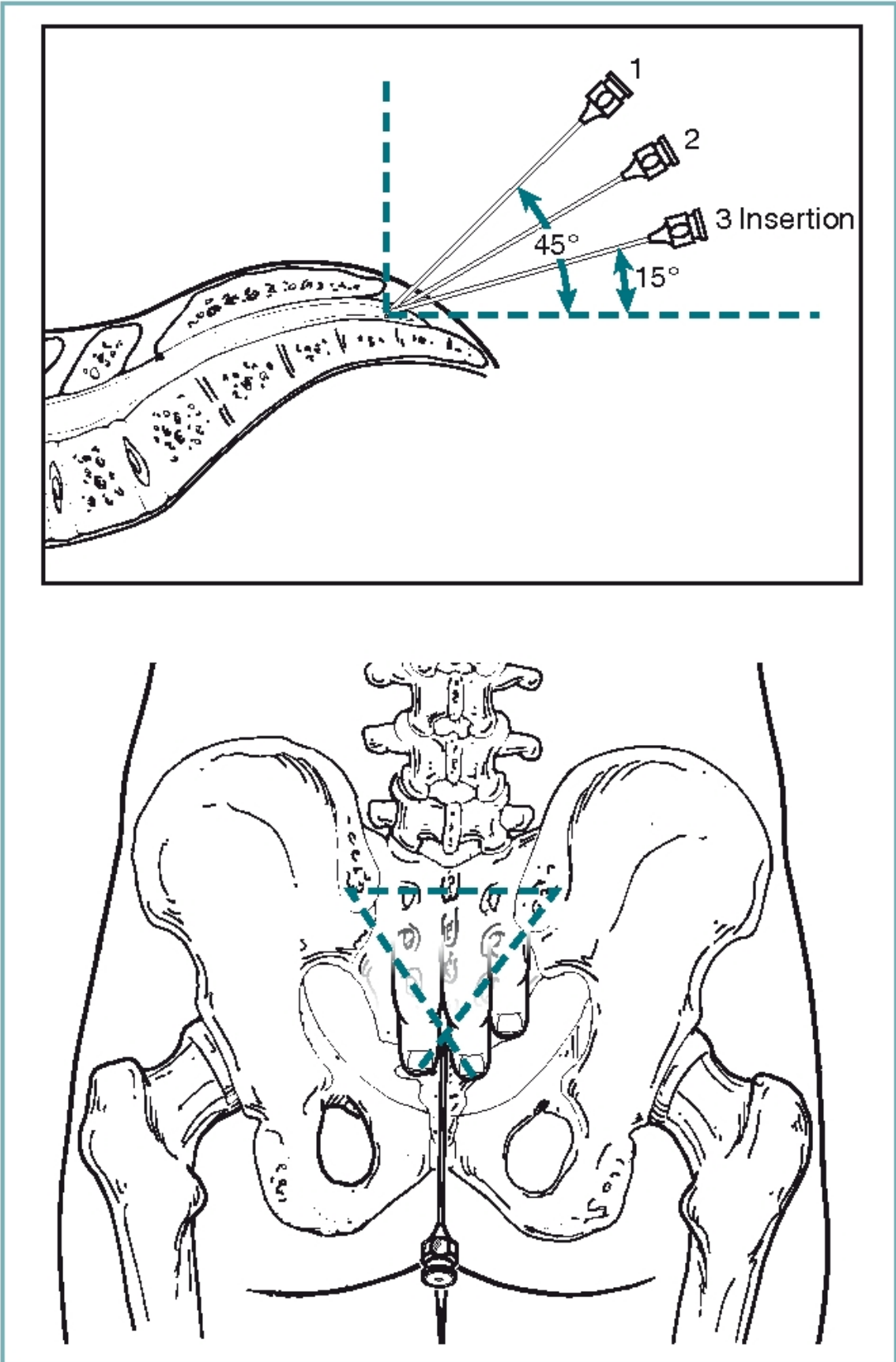
1. Prone - Mostly carried out in adult age group.
2. Left lateral - Mostly employed in children since the bony landmarks are visualized clearly
3. Knee-chest - Very rare in use.

Anatomical landmarks :

Classically hiatus is described as the inferior apex of an equilateral triangle formed by joining the two posterior superior iliac spine and the tip of coccyx. Intergluteal fold is not an ideal landmark because it will not always correspond to the midline. Left forefinger placed in coccyx tip, hiatus corresponds to the second crease of finger. Palpation of this membrane gives a characteristic feel of a membrane under tension similar to that of a fontanelle, the point of puncture is at the midpoint of this triangular space.

Technique :

- Landmark area to be painted with appropriate antiseptics.
- Place the drape which are sterile over the site of injection
- Skin to be punctured with appropriate sized needle at an angle of 90 degrees to the sacrococcygeal membrane.
- A give away sensation will be felt once the needle pierces the membrane.
- Later advance the needle to 30 degrees angulation to the skin.
- The needle to be advanced by 2 mms so that the bevel of the needle lies in the sacral canal.



Confirmation of caudal epidural space :

A)Whoosh test :

This is the commonly employed test. The person performing block must inject air around 2 ml through the needle into the space. An another person with the stethoscope has to auscultate above the site of injection. If a whoosh sound is heard then it indicates correct placement of needle in the caudal epidural space

B)Swoosh test :

This is similar to the above mentioned test with the exception being injection of local anaesthetic solution through the needle resulting in Swoosh sound instead of air. The test carries 91 percent Sensitivity and 100 percent specific.

Injection of Drug :

- Aspirate gently and after confirming negative for blood and Cerebrospinal fluid, inject the drug slowly over a period of 1 - 2 mins.
- Aspirate frequently when injecting the drus.
- The patient vitals like pulse rate, electrocardiogram, blood pressure and Oxygen saturation to be monitored frequently.
- If the drug is given in a fast manner, it may result in high spread of the drug culminating in respiratory distress. Occasionally there may be rise in the intracerebral pressure too. Alternatively, if the drug is given slowly, it may result in patchy block or inadequate level of the block.

Indications :

Below umbilicus surgeries , torsion testis, surgery for hernia with either obstruction or strangulation.

Elective :

Generally carried out along with either Intravenous sedation or General anaesthesia.

- Repair of various types of hernia and Hydrocele.
- Urogenital procedures
- Lower limb Orthopedic procedures
- Circumcision

Contraindications :

- Infection at the site of injection
- Malformed sacrum
- Inflammation of the meninges
- Condition associated with raised intracranial pressure.
- Neural tube disorder - a relative contraindication

Complications :**Problems of Inadvertant position of the Needle:**

1. Injecting the drug in the subcutaneous plane.
2. Sacral foramen may be punctured accidentally.
3. Injury to the blood vessels.

4. Subarachnoid injection of the drug.
5. Injecting the drug into the bony cavity or rectal administration.

Complications due to errors of injection :

Intravascular injection

Since epidural veins are valveless, injection immediately followed by convulsions, arrhythmias, hypotension, respiratory depression.

Subarachnoid space injection

- Lead to total spinal
- Total caudal injection
- Total analgesia even along cranial nerve distribution
- Rarely ? Subdural injection

Hemodynamic problems:

Rare in children below 8 years in the absence of intravenous or subarachnoid injection.

1.Complete or partial failure of the block :

- Complete failure of block
- More common > 7 years old.
- Success rate increases / failure rate decreases with experience, but the failure rate will never be zero even in experienced hands.

2.Lateralization occurs in 1 in 1000 cases

When caudal is performed in lateral decubitus, 50% have a level of anesthesia 2 dermatomes higher on the dependent side. Slow injection difference may be more than 4 dermatomes. May be due to the presence of a complete plica mediana dorsalis

3.Unanesthetized dermatomes

L5, S1, - Large size

4. Inappropriate height of the anaesthetic block

Neurologic complications :

1.Urinary retention :

More common if narcotics given via caudal route first act of micturition may be delayed but not troublesome.

2.Loss of consciousness : Due to very rapid injection of a large volume

3.Nerve lesions : Rarest complication

4.Poor Psychological tolerance :

Persistent motor block can cause anxiety relieved by simple assurance

- Vomiting
- Epidural infection / meningitis
- Shivering.

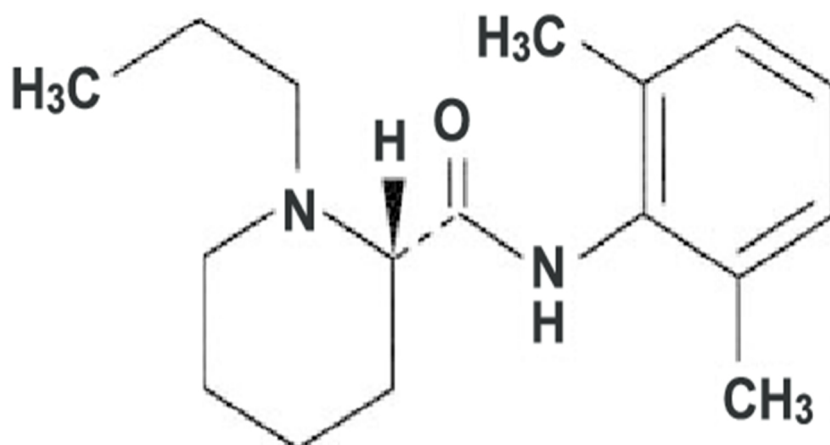
APPLIED PHARMACOLOGY

PHARMACOLOGY OF ROPIVACAINE

Ropivacaine is a S enantiomer of bupivacaine. It is a long-acting amide local anaesthetic agent and it is the first produced pure enantiomer.

STRUCTURE:

Substitution of propyl for the butyl group in the piperidine ring's tertiary nitrogen atom. This confers less lipid solubility and greater sensory motor separation.



Molecular formula: C₁₇H₂₆N₂O

Molecular weight: 274.41

Ropivacaine has a levorotatory counterclockwise rotation. S- enantiomers of local anaesthetic, have different affinity for different ion channels of sodium, potassium and calcium when compared to R⁺ (dextrorotatory clockwise rotation). This causes significant reduction in central nervous system and cardiac toxicity.

Less lipid solubility of ropivacaine favors blockade of C fibers over A fibers. So at higher concentration it has potent sensory and motor block. At lower concentration more sensory blockade than motor blockade. This differential blockade is used in labour analgesia.

MECHANISM OF ACTION:

Ropivacaine acts by causing a reversible inhibition of sodium ion influx, thereby blocking the conduction of impulses in nerve fibers. This action is potentiated by, an additional dose-dependent inhibition of potassium channels.

Ropivacaine is less lipophilic than bupivacaine. This property makes it to be less likely to penetrate through the largely myelinated motor fibres.

Therefore, it has selective action on the pain transmitting A and C nerves, rather than A β fibres, which plays a role in the motor function.

PHARMACODYNAMICS:

The lower lipophilicity of ropivacaine versus bupivacaine correlated with the lesser cardiodepressant effects of both ropivacaine isomers than of the bupivacaine isomers.

The side effects manifest first as CNS symptoms which occurs before the cardiotoxic effects. In CVS it affects conduction time, QRS duration and contractility. It has an additional advantage of inhibiting platelet aggregation, at concentrations of 3.75 and 1.88 mg/ml. It also has an antibacterial activity.

Physiochemical properties:

- pKa : 8.1
- Solubility in water at 25°C : 53.8g/L
- Volume of distribution : 59L
- Protein binding : 94%(α 1-acid glycoprotein)
- onset of action : 10 to 25 min after epidural administration,
- Duration of action : 240- 480 minutes
- T_{1/2} : 111 min
- Clearance : 0.72L/min
- Partition coefficient : 1150
- Maximum dosage : 3 mg/kg

PHARMACOKINETICS:

ABSORPTION AND DISTRIBUTION:

The plasma concentration of ropivacaine depends on

- The total dose of the drug used
- Route by which drug is been administered
- Haemodynamic condition of the patient
- Vascularity of the site where the drug is been administered.

Ropivacaine is 2-3 times less lipid soluble than bupivacaine. So it has lesser penetration to myelinated A fibers and lesser motor blockade. Clinically

significant vasoconstriction is present which is not potentiated by addition of epinephrine.

METABOLISM:

Ropivacaine is metabolised in the liver, by aromatic hydroxylation to 3'-hydroxy-ropivacaine by cytochrome P450 (CYP) 1A2 and N-dealkylation to 2',6'-pipecoloxylidide by CYP3A4.

EXCRETION:

The kidney is the main excretory organ for ropivacaine. It accounts for 86% of the excretion of the drug in urine. It has less lipid soluble and volume of distribution than bupivacaine. So peak plasma level is twice as high as bupivacaine. Its elimination half life is shorter, with a greater clearance, when compared to bupivacaine. It has similar potency with respect to intensity of sensory anesthesia.

Ropivacaine also has a delayed onset of action, shorter duration and less intense motor blockade when compared to that of bupivacaine.

Ropivacaine readily crosses the placenta during epidural anesthesia for caesarean section. There is complete equilibrium of the free fraction of ropivacaine in maternal and foetal circulation. But the total plasma concentration of ropivacaine is seemed to be lower in the foetal circulation when compared to the maternal circulation.

TOXICITY

CNS EFFECTS:

- CNS excitation – nervousness, numbness and tingling circumorally, tinnitus, tremor, lightheadedness dizziness, blurred vision and finally seizures
- This is followed by CNS depression like, drowsiness and loss of consciousness.
- Respiratory depression and apnoea

CVS EFFECTS

- Hypotension
- Bradycardia
- Arrhythmia
- Cardiac arrest- Intravenous lipid emulsion can be used to treat cardiotoxic side effects.

PHARMACOLOGY OF DEXMEDETOMIDINE:

The α_2 -adrenergic receptor agonists have sedative, anxiolytic, hypnotic, analgesic, and sympatholytic effects. The potential for use in anesthesia was recognized in patients who were treated with clonidine. Soon thereafter, a reduction of the minimum alveolar concentration of halothane by clonidine was described. Dexmedetomidine is a more selective α_2 -agonist with a selectivity ratio for the α_2 receptor compared with the α_1 receptor of 1600:1, as compared

with a ratio of 220:1 for clonidine. It was introduced in clinical practice in the United States in 1999 and was approved by the FDA only as a short-term (<24 hours) sedative for mechanically ventilated adult patients in the ICU. Dexmedetomidine is used for prolonged sedation and anxiolysis in the ICU, as well as outside the ICU in various settings, including sedation and adjunct analgesia in the operating room and sedation in diagnostic and procedure units, as well as for other applications such as withdrawal or detoxification amelioration in adult and pediatric patients.

CHARACTERISTICS:

Dexmedetomidine is the S-enantiomer of medetomidine, a substance that has been used for sedation and analgesia in veterinary medicine for many years. It shows a high ratio of specificity for the α_2 receptor (α_2/α_1 1600:1) compared with clonidine (α_2/α_1 220:1), thus making it a complete α_2 -agonist. The pKa is 7.1. Dexmedetomidine belongs to the imidazole subclass of α_2 -receptor agonists, similar to clonidine, It is freely soluble in water and is available as a clear isotonic solution containing 100 $\mu\text{g}/\text{mL}$ and 9 mg sodium chloride per milliliter of water.

METABOLISM AND PHARMACOKINETICS:

Dexmedetomidine undergoes almost complete biotransformation with very little unchanged dexmedetomidine excreted in urine and feces. Biotransformation involves both direct glucuronidation and cytochrome P450-mediated metabolism.

The major metabolic pathways of dexmedetomidine are direct *N*-glucuronidation to inactive metabolites, hydroxylation (mediated primarily by CYP2A6), and *N*-methylation. Dexmedetomidine is 94% protein bound, and its concentration ratio between whole blood and plasma is 0.66. Dexmedetomidine has effects on cardiovascular variables, potentially causing bradycardia, transient hypertension or hypotension, and may alter its own pharmacokinetics. With large doses, marked vasoconstriction occurs and probably reduces the drug's volumes of distribution. In essence, dexmedetomidine displays nonlinear pharmacokinetics. In subjects with varying degrees of hepatic impairment clearance values for dexmedetomidine are slower than in healthy subjects. The mean clearance values for patients with mild, moderate, and severe hepatic impairment are 74%, 64%, and 53% of those observed in the normal healthy subjects, respectively. The pharmacokinetics of dexmedetomidine is not influenced by renal impairment (creatinine clearance <30 mL/minute) or age. In patients with severe renal disease, the sedative effect may be stronger as a result of a lower degree of plasma protein binding. Clearance is a function of height. The elimination half-life of dexmedetomidine is 2 to 3 hours, with a context-sensitive half-time ranging from 4 minutes after a 10-minute infusion to 250 minutes after an 8-hour infusion. No clinically relevant cytochrome P450-mediated drug interaction has been found.

PHARMACOLOGY:

Dexmedetomidine acts nonselectively on various subtypes of membrane-bound G protein-coupled α_2 -adrenoreceptors. Intracellular pathways include inhibition of adenylate cyclase and modulation of calcium and potassium ion channels. Three subtypes of α_2 adrenoreceptors have been described in humans: α_2A , α_2B , and α_2C . The α_2A adrenoreceptors are primarily distributed in the periphery, whereas α_2B and α_2C are in the brain and spinal cord. Postsynaptic α_2 adrenoreceptors located in peripheral blood vessels produce vasoconstriction, whereas presynaptic α_2 adrenoreceptors inhibit the release of norepinephrine and potentially attenuate the vasoconstriction. The overall response to α_2 -adrenoreceptor agonists is related to the stimulation of α_2 adrenoreceptors located in the CNS and spinal cord. These receptors are involved in the sympatholysis, sedation, and antinociceptive effects of α_2 adrenoreceptors. The α_2 -agonists have the advantage that their effects are readily reversible by α_2 -adrenergic antagonists (e.g., atipamezole)

Effects on the Central Nervous System:

Sedation: The α_2 -agonists produce their sedative-hypnotic effect by an action on α_2 receptors in the locus coeruleus and by an analgesic action at α_2 receptors within the locus coeruleus and within the spinal cord. Dexmedetomidine produces a decrease in activity of the projections of the locus coeruleus to the ventrolateral preoptic nucleus. As a result, GABA and galanin release in the tuberomammillary nucleus is increased, producing a decrease in histamine

release in cortical and subcortical projections. The α_2 -agonists inhibit ion conductance through L-type or P-type calcium channels and facilitate conductance through voltage-gated calcium-activated potassium channels. Dexmedetomidine induces sedation through different receptors than the sedative drugs propofol and benzodiazepines, which exert their action through the GABA system. The sedative effect of dexmedetomidine acts through the endogenous sleep-promoting pathways, thus generating natural sleep patterns and having the ability to follow commands and cooperate while being tracheally intubated. Undisturbed, patients were noted to fall asleep momentarily. This characteristic allows for “daily wake-up” tests to be done in a safe fashion. This critical test, in which ventilated patients in the ICU are taken off all sedatives to assess their mental status and titrate sedation, shortens their ventilated and ICU length of stay. The number of patients experiencing delirium in the ICU is significantly lower when dexmedetomidine is used for sedation. Patients have been described as being very easy to wake up.

Analgesia: The analgesic effect of the α_2 -agonists is mediated through stimulation of the α_{2C} and α_{2A} receptor in the dorsal horn, thus directly suppressing pain transmission by reducing the release of pronociceptive transmitters, substance P and glutamate, and hyperpolarization of interneurons. Systemic use of dexmedetomidine has an opioid-sparing effect during surgery and postoperatively.

This effect is advantageous in patients who are prone to postoperative apnea or hypoventilation, such as patients undergoing bariatric surgical procedures. In the postoperative ICU setting, narcotic requirements are reduced by 50% when patients are receiving a dexmedetomidine infusion compared with placebo. During general anesthesia, dexmedetomidine reduces the MAC of inhaled anesthetics. Like clonidine, dexmedetomidine is frequently used as an adjuvant in central or peripheral neural blockade. When it is administered caudally, 1 µg/kg as an adjuvant to bupivacaine 0.25% 1 mL/kg, in children undergoing inguinal hernia repair, response to hernial sac traction is reduced, and postoperative analgesia is prolonged.

Central Nervous System Effects: The CNS protective effects are not well defined. In animal models of incomplete cerebral ischemia and reperfusion, dexmedetomidine reduced cerebral necrosis and improved neurologic outcome. The prevalent idea is that dexmedetomidine reduces the intracerebral catecholamine outflow during injury. The neuroprotection may be attributed to modulation of proapoptotic and antiapoptotic proteins. In addition, the reduction of the excitatory neurotransmitter glutamate during injury may explain some of the protective effects. In patients undergoing transsphenoidal hypophysectomy, dexmedetomidine had no effect on lumbar cerebral spinal fluid pressure. The decrease in Cerebral blood flow was not accompanied by a reduction in CRMO₂. Dexmedetomidine has been used in neurosurgical procedures involving neurophysiologic monitoring. Cortical evoked potential

amplitudes and latencies were minimally affected when dexmedetomidine was used intraoperatively. It may also be suitable as an anesthetic adjunct during surgical treatment of seizures because the epileptiform activity of seizure foci was not reduced by dexmedetomidine.

Effects on the Respiratory System: In spontaneously breathing volunteers, dexmedetomidine at concentrations producing significant sedation reduced minute ventilation, but with no change in arterial oxygenation, pH, or the slope in the carbon dioxide ventilatory response curve.

Effects on the Cardiovascular System: The most commonly reported hemodynamic adverse reactions associated with dexmedetomidine were hypotension, hypertension, and bradycardia. The initial increase in arterial blood pressure is probably caused by the vasoconstrictive effects of dexmedetomidine when stimulating peripheral α_2 receptors. The incidence of hypotension and bradycardia may be related to the administration of a large IV “loading” dose. Omitting the loading dose or not giving more than 0.4 $\mu\text{g}/\text{kg}$ reduces the incidence of hypotension or makes it less pronounced. Giving the loading dose over 20 minutes also minimizes the transient hypertension. In several studies after IM and IV administration, dexmedetomidine caused, in a small percentage of patients, profound bradycardia (<40 beats/ minute) and occasionally sinus arrest or pause. Generally, these episodes resolved spontaneously or were readily treated without adverse outcome by anticholinergics. No rebound effects have been found when discontinuing a

dexmedetomidine infusion, even when it was given for more than 24 hours. Because clonidine and dexmedetomidine have shown to reduce perioperative oxygen consumption and blunt the sympathetic response to surgery, cardiac outcome may be improved. However, more studies are needed to determine whether dexmedetomidine is beneficial in decreasing the risk of myocardial ischemia.

USES

Dexmedetomidine has been approved as a short-term sedative for adult intubated patients in the ICU. Given its well-documented beneficial effects of anxiolysis, sedation, analgesia, and sympatholysis with minimal respiratory depression, it also has been used in various other clinical situations. It is well used as a sedative during radiologic or invasive procedures, in adults as well as in children.

As a premedicant, dexmedetomidine, at IV doses of 0.33 to 0.67 $\mu\text{g}/\text{kg}$ given 15 minutes before the surgical procedure, seems efficacious, while minimizing the cardiovascular side effects of hypotension and bradycardia. Dexmedetomidine has high bioavailability when administered nasally or buccally. This improves better compliance and absorption in younger children. A dose of 3 to 4 $\mu\text{g}/\text{kg}$ 1 hour preoperatively is safe and effective. The average infusion rate of dexmedetomidine intraoperatively to maintain a Bispectral index value of 70 to 80 was 0.7 $\mu\text{g}/\text{kg}/\text{minute}$. Sedation was more prolonged after termination of the infusion, as was recovery of arterial blood pressure.

Dexmedetomidine can also produce profound sedation, and it has been used as a total IV anesthetic. This characteristic, combined with the cooperative status of the patient at a lighter sedative level, and its analgesic effect with sparing of respiratory function, makes the drug suitable as hypnotic agent during surgical procedures such as awake craniotomy, deep brain stimulation, surgical procedures near speech areas, or awake carotid endarterectomies, with fewer fluctuations from the desired sedation level and more stable hemodynamics. The opioid-sparing effects are advantageous in the performance of bariatric surgery in patients who are prone to postoperative respiratory depression. Dexmedetomidine can be employed for addiction treatment; it has been described for use in rapid opioid detoxification, cocaine withdrawal, and iatrogenically induced benzodiazepine and opioid tolerance after prolonged sedation. Dexmedetomidine may produce dry mouth secondary to a decrease in salivation. Combined with the sparing effect on respiratory function, this effect is beneficial for the facilitation of awake fiberoptic intubation.

Furthermore, dexmedetomidine decreases intraocular pressure and decreases the shivering threshold.

Intensive Care Unit: For sedation in the ICU, loading doses of 0.5 to 1 $\mu\text{g}/\text{kg}$ have been used. Omitting the bolus dose or giving the lower dose has been associated with fewer episodes of severe bradycardia and other hemodynamic perturbations. Infusion rates of 0.1 to 1 $\mu\text{g}/\text{kg}/\text{hour}$ are generally needed to maintain adequate sedation. The unique characteristics of dexmedetomidine

(i.e., providing adequate sedation with minimal respiratory depression) make this selective α_2 -adrenoceptor agonist very useful when weaning patients from the ventilator.

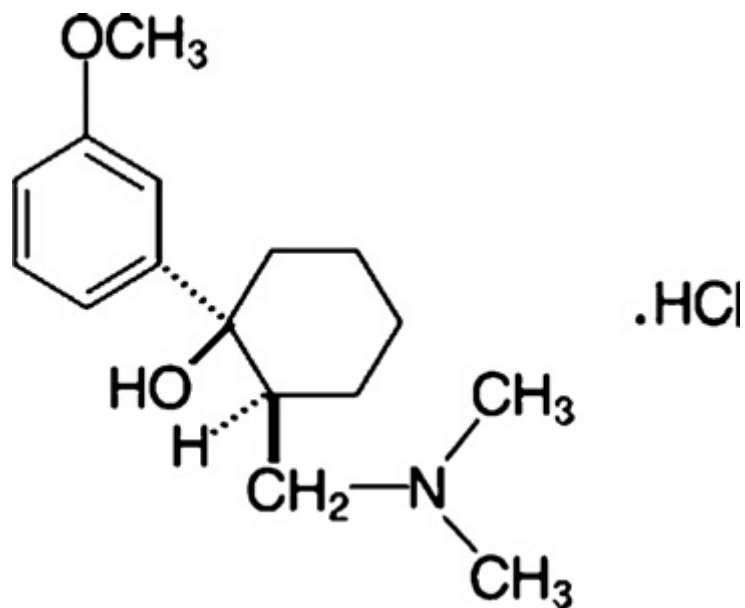
PHARMACOLOGY OF TRAMADOL

Tramadol hydrochloride is a narcotic drug introduced in 1971 in Germany and is now available throughout the world. Tramadol hydrochloride is a synthetic 4-phenyl-piperidine analogue of codeine with a dual mechanism of action.

Tramadol hydrochloride is a centrally acting synthetic codeine analogue that is a weak opioid receptor agonist. Its affinity for μ opioid receptor is modest while that for κ and δ is weak. Its affinity for the opioid receptor is only 1/6000 that of morphine.

Unlike other opioids, it inhibits reuptake of nor-adrenaline and 5-hydroxytryptamine and thus activates monoaminergic spinal inhibition of pain. Its analgesic action is only partially reversed by opioid antagonist naloxone.

It is a racemic mixture of 2 enantiomers (+) tramadol and (–) tramadol and has chemical structure: (1R, 2R)-2-[(dimethylamino)methyl]-1-(3-methoxyphenyl)cyclohexanol hydrochloride.



Molecular formula : C 16 H 25 NO 2 HCl

Characteristics:

- White to off white crystalline
- odourless
- Soluble in water and ethanol
- Characteristic unpleasant taste which is mildly bitter
- Molecular weight-299.84
- pH 6-6.8
- pKa value is 9.3 at 293 K

Available as capsules, drops, suppositories and injections. Solution for injection contains 50 mg/ml of tramadol hydrochloride in an adequate sodium acetate buffered solution without preservative.

MECHANISM OF ACTION:

The antinociceptive action of tramadol hydrochloride is mediated by two components:

1. Opioid pathway: Tramadol interacts with μ , κ and δ receptors where it exhibits purely agonist effects. It has moderate affinity for μ receptors and weak affinity for δ and κ receptors. The duration of antinociceptive is relatively long being comparable to morphine and longer than that of codeine and dextropropoxyphene. The opioid component of analgesia is reversed by naloxone.

2. Non-opioid pathway: Tramadol hydrochloride inhibits the reuptake of nor-adrenaline and 5-hydroxy tryptamine (serotonin) and activates monoaminergic spinal inhibition of pain by elevating the pain threshold. It is also 5 HT_{2C} receptor antagonist and Nicotinic ,M₁ and M₃ Muscarinic receptor antagonist, NMDA antagonist and GABA_A receptor inhibitor

PHARMACOKINETICS:

Absorption : The bioavailability following oral administration of the drug is 68 to 100%.It is 100% available when administered intramuscularly

Distribution: The drug is 20 % protein bound in the plasma. 80% of the administered dose crosses the placenta

Metabolism: 85% of the administered dose is metabolized by demethylation in liver via cytochrome P450. The metabolite O- desmethyl tramadol is active and has 200 times μ affinity of that of tramadol. It binds to μ opioid receptor and exerts its effects on GABAergic transmission.

Excretion: Excretion is 90% through the kidneys. Remaining is excreted in the faeces. The $T_{1/2}$ is 5 hours. The elimination half-life is 5-6 hours. The elimination half life is doubled in patients with hepatic or renal impairment. So it is not recommended in patients with end stage renal failure.

PAIN PHYSIOLOGY

Pain is a complex phenomenon that includes both sensory-discriminative and motivational-affective components. The sensory discriminative component of pain depends on ascending projections of tracts (including the spinothalamic and trigeminothalamic tracts) to the cerebral cortex. Sensory processing at these higher levels results in the perception of the quality of pain (pricking, burning, aching), the location of the painful stimulus, and the intensity of the pain. The motivational affective responses to painful stimuli include attention and arousal, somatic and autonomic reflexes, endocrine responses, and emotional changes. These account collectively for the unpleasant nature of painful stimuli. The definition of pain as proposed by the International Association for the Study of Pain emphasizes the complex nature of pain as a physical, emotional, and psychological condition. It is recognized that pain does not necessarily correlate

with the degree of tissue damage that is present. Failure to appreciate the complex factors that affect the experience of pain and reliance entirely on physical examination findings and laboratory tests may lead to misunderstanding and inadequate treatment of pain.

Oversimplified anatomic concepts predispose to simplistic therapeutic interventions, such as neurectomy or rhizotomy, that may intensify pain or create new and often more distressing pain. The nociceptive system is highly complex and adaptable. Sensitivity of most of its components can be reset by a variety of physiologic and pathologic conditions. Innovative medications are being developed that target the causes of pain by actions on pain transduction, transmission, interpretation, and modulation in both the peripheral nervous system and the central nervous system.

NEUROBIOLOGY OF PAIN:

The experience of pain involves a series of complex neurophysiologic processes, collectively termed *nociception*, with four distinct components: transduction, transmission, modulation, and perception. Transduction is the process by which a noxious stimulus (e.g., heat, cold, mechanical distortion) is converted to an electrical impulse in sensory nerve endings. Transmission is the conduction of these electrical impulses to the CNS with the major connections for these nerves being in the dorsal horn of the spinal cord and thalamus with projections to the cingulate, insular, and somatosensory cortices. Modulation of pain is the process of altering pain transmission. It is likely that both inhibitory

and excitatory mechanisms modulate pain (nociceptive) impulse transmission in the PNS and CNS. Pain perception is thought to be mediated through the thalamus acting as the central relay station for incoming pain signals and the primary somatosensory cortex serving for discrimination of specific sensory experiences. Pain may occur in the absence of the occurrence of these four steps. For example, pain from trigeminal neuralgia occurs in the absence of transduction of a chemical stimulus at a nociceptor reflecting axonal discharges initiated at the site of a compressed or demyelinated nerve. Modulation of pain impulses may not occur if specific nervous system tracts are injured. For example, phantom limb pain occurs in the absence of nociception or nociceptors (pain receptors).

GATE THEORY: The gate control theory of pain was first proposed by Ronald Melzack and Patrick Wall in 1965 to illustrate the neuronal network underlying pain modulation (a neurologic “gate”) in the spinal dorsal horn. According to this theory, painful information is projected to the supraspinal brain regions if the gate is open, whereas painful stimulus is not felt if the gate is closed by the simultaneous inhibitory impulses. Here is a commonly used example to describe how this neuronal network modulates pain transmission. Usually, rubbing the skin of painful area seems to somehow relieve the pain associated with a bumped elbow. In this case, rubbing the skin activates large-diameter myelinated afferents ($A\beta$), which are “faster” than $A\delta$ fibers or C fibers conveying painful information.

These A β fibers deliver information about pressure and touch to the dorsal horn and override some of the pain messages (“closes the gate”) carried by the A δ and C fibers by activating the inhibitory interneurons in the dorsal horn. This hypothesis provided a practical theoretical basis for some approaches such as massage, transcutaneous nerve stimulation, and acupuncture to effectively treat pain in clinical patients.

PAIN ASSESSMENT

“Pain is a unique, highly subjective multidimensional experience encompassing many sensory & affective components”. Pain assessment is the most important and critical component of pain management. Assessment and management are interrelated. If pain can be assessed accurately, adequate and appropriate management can be implemented.

Assessing pain in children is an ever challenging as well as a difficult task, mainly because so far no reliable method of assessing and measuring child’s pain is available.

Various methods available are,

- Physiological measures
- Self reporting measures
- Behavioral measures

Physiological measures

Changes in pulse, blood pressure and respiration reflect autonomic arousal. Autonomic responses to pain and their measurement form an important aspect of certain pain scales. Metabolic changes cause release of catecholamine, growth hormone, glucagon, cortisol, aldosterone and beta endorphins which have been documented in infants and children following noxious stimulation. Only plasma cortisol have been shown to correlate with behavioral responses to noxious stimuli.

Self reporting measures

1. **VISUAL ANALOGUE SCALE:** Visual analogue scale is the accepted and popular method of measurement of pain in adults and provides reproducible results in children down to an age of five years. VAS using 10 cm length scale marked “no pain” at one end to “excruciating pain” at the other end with 1 mm or 1 cm segments. The child is asked to identify a point on the scale which corresponds to his pain. A score of less than 4 is no pain, less than 6 implies tolerable pain and more than 6 means he needs medication. VAS can also be a 50 cm long, linear scale with no pain at one end and excruciating pain at the other end with no intermediate division but with descriptive red white colorings.
2. **OUCHER’S SCALE:** This scale displays six photographs of a child’s face showing increasing levels of discomfort. This scale is based on the mimic, vocalization and irritability.

Features characteristic of increasing pain are;

- a) Distortion of face such as lowering of the brow, broadening of the nasal root, angular and squarish mouth, tightly closed eyes and tightening of the jaw.
- b) Vocalization, changing from sobbing to pain cry. The children are asked to show the face which mimicked their expression

3. THE POKER CHIP SCALE: It quantitates the child's pain by the number of chips (0–4), he/she selects "pieces of hurt".

4. Analogue Chromatic Continuous Scale (ACCS): This system is potentially useful for children as young as 3 years old. Children tend to associate red and black colors with increased pain sensation. (The back is ruled for easy scoring).

Behavioural Observation Methods

Behavioural observation methods are the primary approach to accessing pain information from preverbal and nonverbal children. They score the behaviours which represent the reaction to pain and the scores are allotted according to the degree of alteration of a particular behaviour.

The behaviours scored include vocal behaviours such as cry, scream, verbally expressed pain and anxiety and non verbal behaviours such as muscle rigidity, torso movements, leg movements and facial expression.

The PBRS, CHEOPS, CHIPPS and TPPPS: Pain Behavior Rating Scale,

Children's Hospital Eastern Ontario Pain Scale, Children and Infants Postoperative Pain Scale and Toddler- Preschooler Postoperative Pain Scale are such scales. The observation in these scales has an observer bias.

The OBJECTIVE PAIN SCALE measures pain as a physiological variable-blood pressure along with behavioral changes. This has been shown to be a sensitive and reliable tool in evaluating postoperative pain in children who are not able to verbally comment upon their pain. This takes into account the systolic blood pressure, cry and its response, movement, agitation and verbal evaluation as described by Hannallah RS.

MATERIALS AND METHODS

Study Design: Prospective, randomized, double blinded Clinical study

Sampling technique: Simple Random sampling

Sample Size: Sample size calculation indicated that a total sample of 30 patients in each group would be required to have a large effect with 80% power using t-test with α error = 0.05 and β = 0.2.

Minimum sample size was calculated to be 40. Based on this I have included 60 patients in my study and 30 patients were selected in each group.

Institutional Ethical Committee clearance for the study was obtained. The study was conducted in Tirunelveli medical college at the Department of Anaesthesiology between April 2018 and June 2019. Written informed consent from the parents was also obtained.

INCLUSION CRITERIA:

- Children of both sexes aged 1 - 8 years
- ASA PS I/II
- Posted for Elective Infraumbilical surgery like Inguinal hernia repair, Urethroplasty, Hydrocele, etc..

EXCLUSION CRITERIA:

- History / evidence of infection at back
- Any known allergy to drugs

- Bleeding / Coagulation disorders
- H/O Developmental delay
- Sepsis
- Pre-existing neurological diseases

ALLOCATION: 2 Groups

GROUP RD: 30 children - receiving 0.25% Ropivacaine 1 ml/kg with Dexmedetomidine 2 µg/kg

GROUP RT: 30 children - receiving 0.25% Ropivacaine 1 ml/kg with Tramadol 2 mg/kg

On receiving the patient in the operation theatre, electrocardiogram (ECG), pulse oximeter (SpO₂) and non-invasive blood pressure (NIBP) were monitored and baseline parameters recorded.

Pre-medication was done with intravenous (IV) midazolam 0.05 mg/kg through already secured venous access.

Induction of anaesthesia was achieved with 50% nitrous oxide (N₂O) and 8% sevoflurane in oxygen in spontaneous ventilation.

After appropriate-sized laryngeal mask airway was inserted, sevoflurane concentration was reduced to 3% with 50% oxygen and 50% N₂O.

Thereafter, patients were placed in a lateral position and the skin of the back over the sacrum was disinfected using povidone-iodine solution, and

under aseptic precautions, single-dose caudal epidural injection was performed using a 25-gauge needle.

Needle position was confirmed by the pop sensed during penetration of the sacrococcygeal ligament, which was followed by the whoosh test using 0.5 ml of air. After negative aspiration of blood or cerebrospinal fluid, caudal medication was given as per the group assigned.

The time of caudal block was recorded, and the surgery was allowed to start 10 min after caudal injection. The inhaled concentration of sevoflurane was adjusted to achieve haemodynamic changes within 20% of the baseline values. No other analgesics, sedatives or narcotics were used intraoperatively.

Time taken for the administration of block was noted. Anaesthesia was maintained with sevoflurane and oxygen 50% and N₂O 50%. All the patients were monitored by a standard protocol in a uniform pattern during anaesthesia and surgery.

Continuous monitoring of vital parameters -heartrate (HR), ECG, respiratory rate, NIBP, SpO₂ -was done, and values were recorded before and after pre-medication, induction, caudal block, after incision and thereafter every 5 min until the surgery was over.

At the end of surgery, all anaesthetic drugs were discontinued. Total time of surgery was recorded. Any side effects such as breath holding/apnoea, hypotension, involuntary movements, nausea and vomiting were noted.

The occurrence of intraoperative Hypotension (fall in blood pressure > 20% from baseline) requiring a fluid bolus and bradycardia (fall in heart rate > 20% from baseline) requiring atropine was recorded.

Using the paediatric observational FLACC pain score with its 0–10 score range, each patient's pain intensity was assessed every 2 hour till 20 h, every 4 h till 24 h and until the first dose of rescue analgesia was given. Rescue analgesia was with paracetamol suppository 15 mg/kg, given when the FLACC score was ≥ 4 .

Motor block was assessed in the PACU on awakening by using a modified Bromage scale that consisted of 4 points:

0 = full motor strength (flexion of knees and feet),

1 = flexion of knees

2 = little movement of feet only,

3 = no movement of knees or feet.

However, younger children who could not move their legs on command were stimulated by tapping on the legs and feet.

Level of sedation was assessed by Ramsay sedation scale at every 1 hr after extubation and thereafter hourly until the Ramsay sedation score became 1 in all patients. Duration of post-operative sedation was deemed from the time of extubation until Ramsay sedation score was 2 or less.

The times recorded were

- Anaesthesia time (time from induction of anaesthesia to the end of surgery, when sevoflurane was discontinued)
- Time from caudal block to skin incision
- Time from caudal block to end of surgery
- Emergence time (time from the end of surgery to opening the eyes on calling).

Anaesthetic emergence was considered as delayed if the time elapsed from the end of surgery to exiting the operating theatre was greater than 20 min.

The criteria for transferring the patient from operating room to PACU were being awake, moving all limbs, patent airway and normal respiratory pattern, normal oxygen saturation with no need for mandible support, stable hemodynamics, normothermia, and pain free.

Failure of the caudal block was defined as any increase in HR or mean arterial pressure (MAP) more than 20% of the pre-incision values. Failure of the caudal block was not reported in any patient.

STATISTICAL TESTS USED:

Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (trial version 22.0; SPSS Inc, Chicago). Descriptive statistics such as mean and standard deviation (SD) for continuous variables, frequencies and percentages were calculated for categorical variables. Line, Bar and Pie charts were used for visual representation of data. To check for association between scores and group, chi square test was used. In case of less expected cell values Fishers exact test was used. To compare the distribution of median ramsay scores in both groups Mann Whitney U test was used. Level of significance was set at 0.05.

RESULTS

Table 1: Age group distribution among both the study groups:

Age	Group RD N = 30	Group RT N = 30	Total N = 60	P value
1 – 3 years	17 (56.7%)	19 (63.3%)	36 (60%)	0.634
> 3 years	13 (43.3%)	11 (36.7%)	24 (40%)	
Total	30 (100%)	30 (100%)	60 (100%)	
Mean	3.2	3.0	3.1	
Standard deviation	1.2	1.4	1.3	
Range	1 - 5	1- 6	1 - 6	

Majority of the study population in both groups belonged to < 3years of age. In group RD 1 – 3years of age was 56.7% and > 3 years of age was 43.3%. In RT group 1-3 years of age consist of 63.3% and >3 years of age was 36.7%. This difference in age group distribution was not statistically significant with p=0.634

Chart 1: Age group distribution among both the study groups:

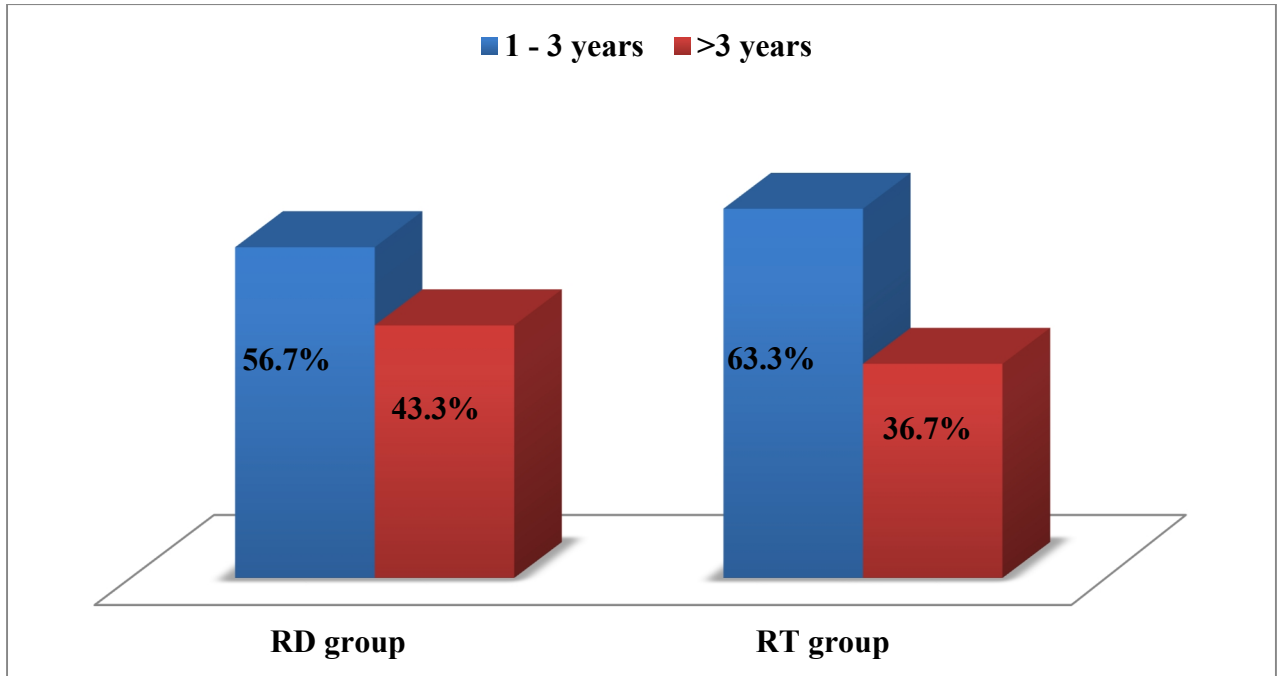


Table 2: Sex distribution among both the study groups:

Sex	Group RD	Group RT	Total	P value
Male	21 (70%)	22 (73.3%)	43 (71.6%)	0.774
Female	9 (30%)	8 (26.7%)	17 (28.4%)	
Total	30 (100%)	30 (100%)	60 (100%)	

Majority of the study population in both groups belonged to male. In group RD males were 70% and females were 30%. In RT group Males consist of 73.3% and females were 26.7%. This difference in sex distribution was not statistically significant with $p=0.774$.

Chart 2: Sex distribution among both the study groups:

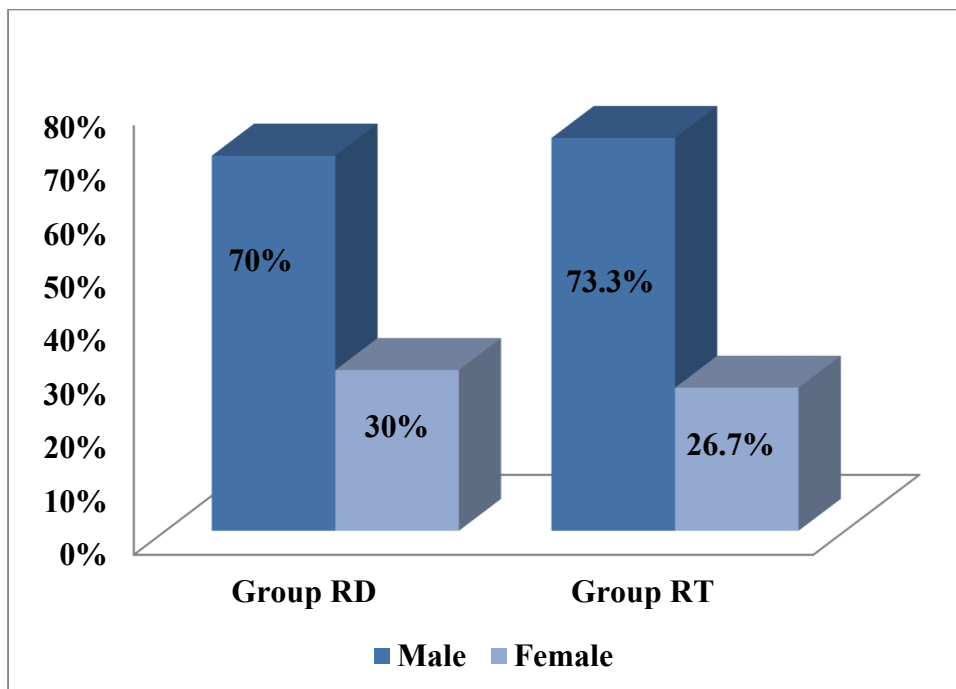


Table 3: Weight distribution among both the study group:

Weight	Group RD	Group RT	Total	P value
Mean±SD	13.4±3.4	13.7±7	13.6±3.7	
Range	8 - 20	8 - 22	8 - 22	0.759

The mean weight in group RD is 13.4kgs, mean weight in group RT is 13.7kgs. The mean weight was slightly more in group RT and this difference was not statistically significant with P=0.759.

Table 4: Duration of surgery among both the study group:

Duration of surgery	Group RD	Group RT	Total	P value
Mean±SD	28.3±12.2	21±12.5	24.6±12.8	
Range	10 - 50	10 - 50	10 - 50	0.031

The mean duration of surgery in group RD is 28.3 minutes, mean duration of surgery in group RT is 21 minutes. The mean duration of surgery was more in group RD and this difference was statistically significant with P<0.001.

Table 5: Baseline pulse rate, systolic blood pressure, diastolic blood pressure, spo2 among both the study group:

Baseline parameters	Group RD	Group RT	Total	P value
Pulse rate				
Mean±SD	111.3±7.1	112.3±8.2	111.8±7.6	0.641
Range	98 - 126	98 - 130	98 - 130	
SBP				
Mean±SD	114.4±7.6	117.3±7	115.8±7.4	0.123
Range	98 - 126	98 - 130	98 - 130	
DBP				
Mean±SD	70.7±4.4	72±4.3	71.4±4.3	0.262
Range	58 - 78	58 - 80	58 - 80	
SPO2				
Mean±SD	99.7±0.43	99.6±0.48	99.7±0.45	0.399
Range	99 - 100	99 - 100	99 - 100	

The mean pulse rate in group RD is 111/min, in group RT is 112/min minutes. This difference was not statistically significant with P=0.641.

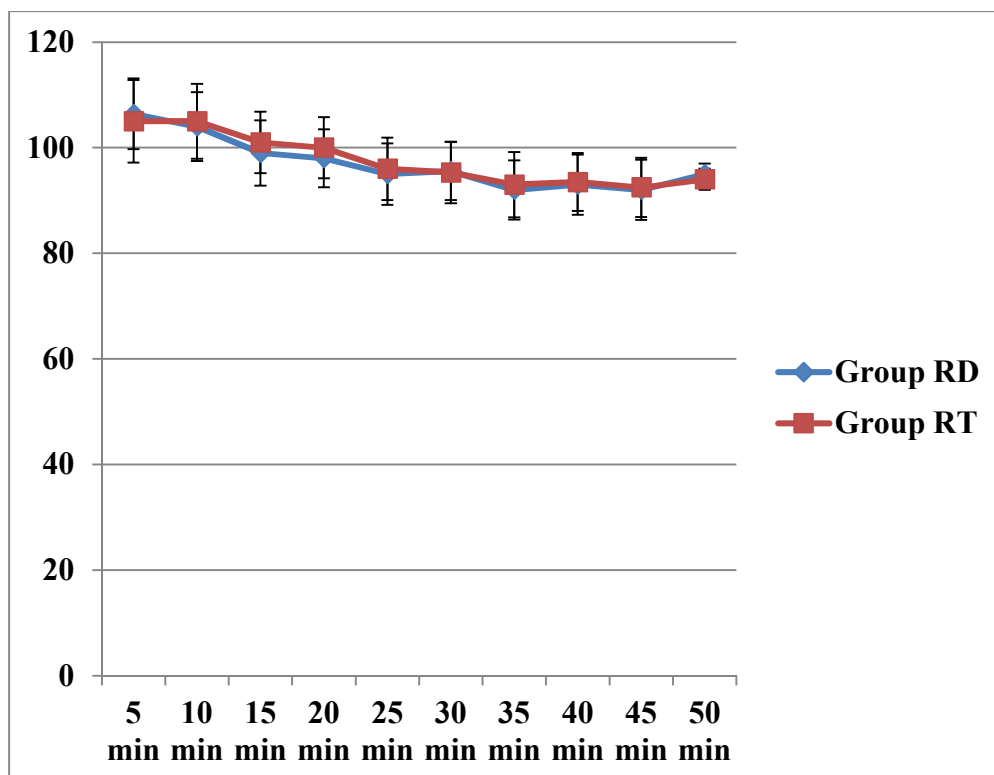
The mean SBP in group RD is 114mmhg and in group RT is 117/mmhg. This difference was not statistically significant with P=0.123.

The mean DBP in group RD is 70.7mmhg and in group RT is 72mmhg. This difference was not statistically significant with P=0.262.

The mean SPO2 in group RD is 99.7% and in group RT is 99.6%. This difference was not statistically significant with P=0.399.

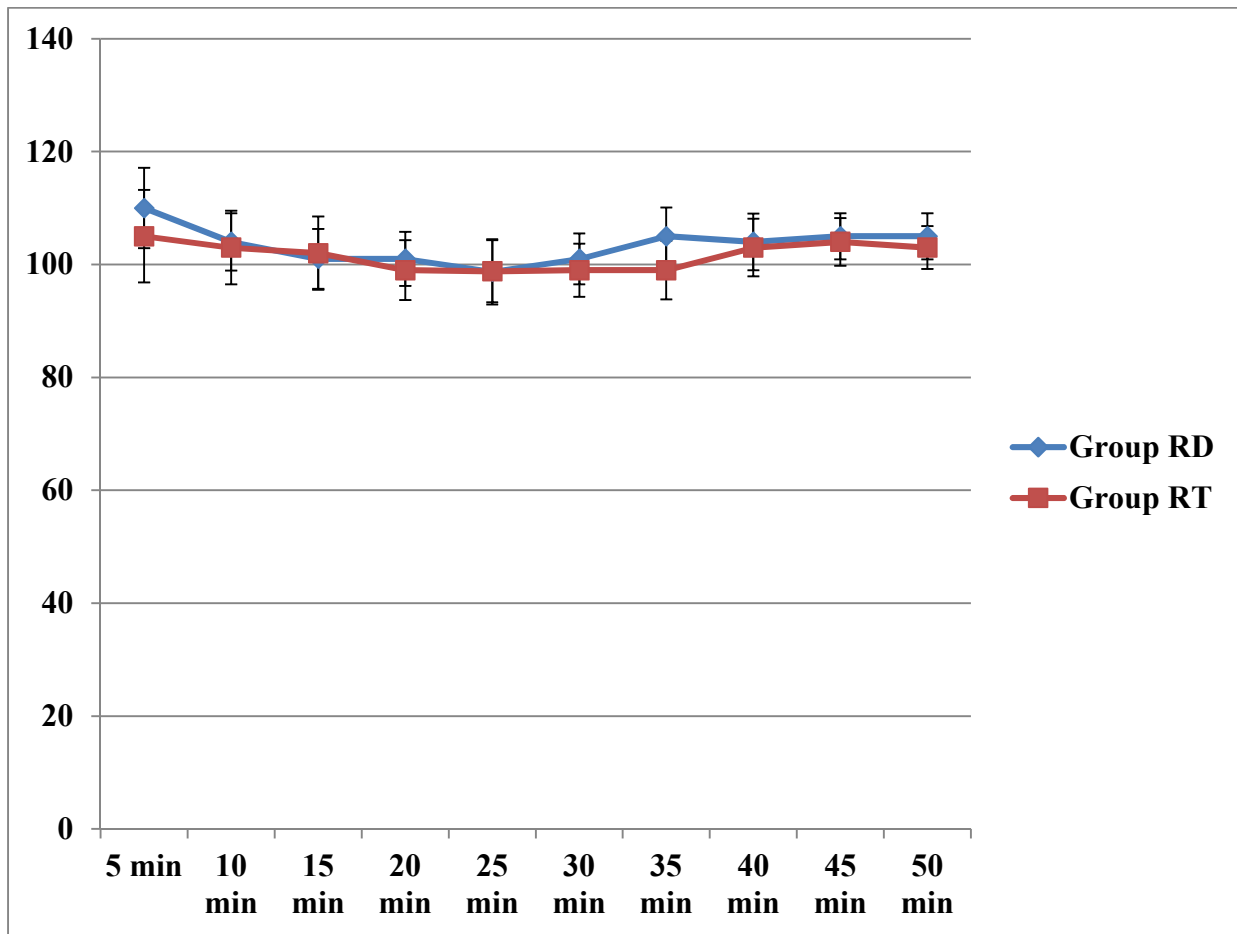
The baseline haemodynamic parameters were similar in both groups. There was no statistically significant difference.

Chart 3: Intraoperative trends of pulse rate in both groups:



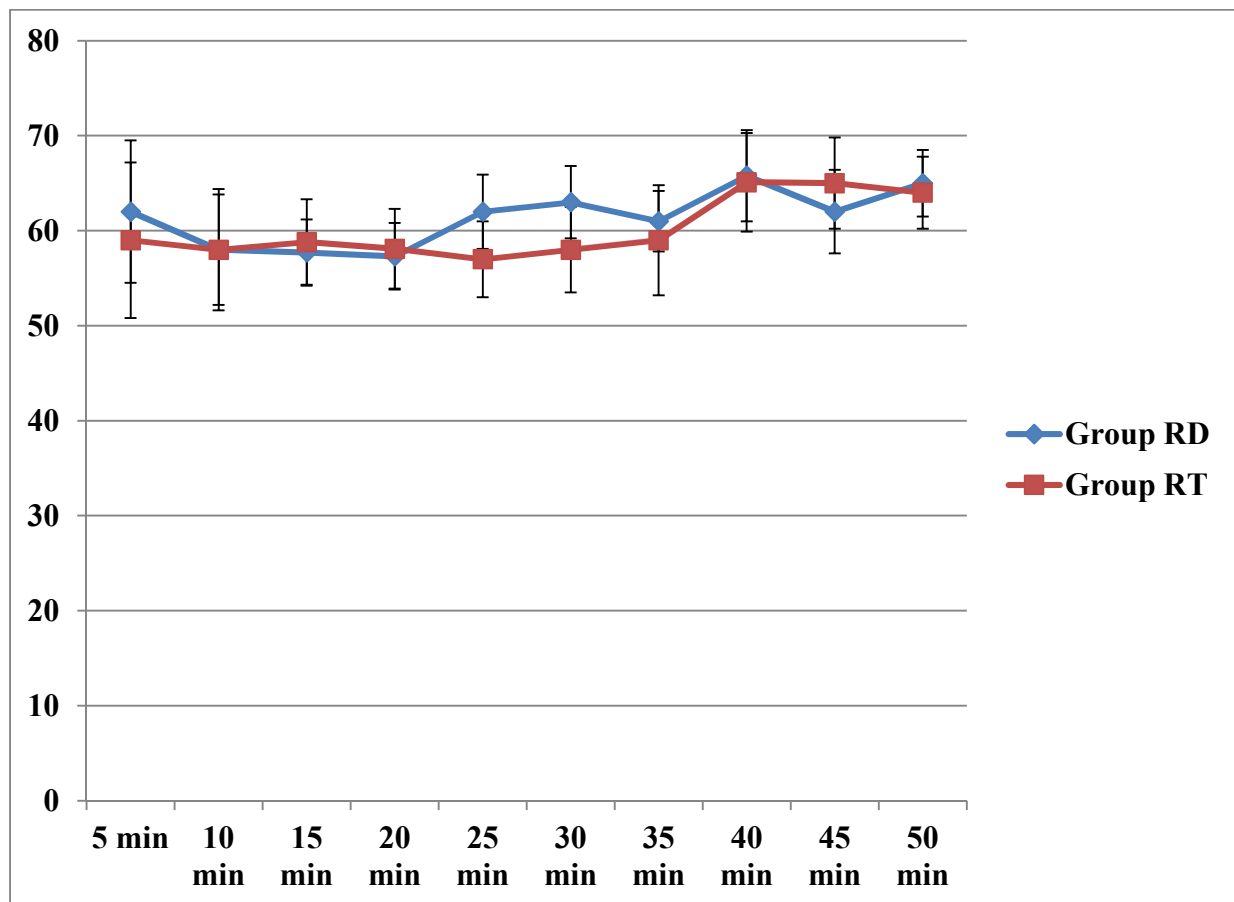
The intraoperative pulse rate was measured at regular time interval during the course of anaesthesia. The pulse rate remained stable in both groups. There was no statistically significant difference in pulse rate across both groups intraoperatively.

Chart 4: Intraoperative trends of SBP in both groups:



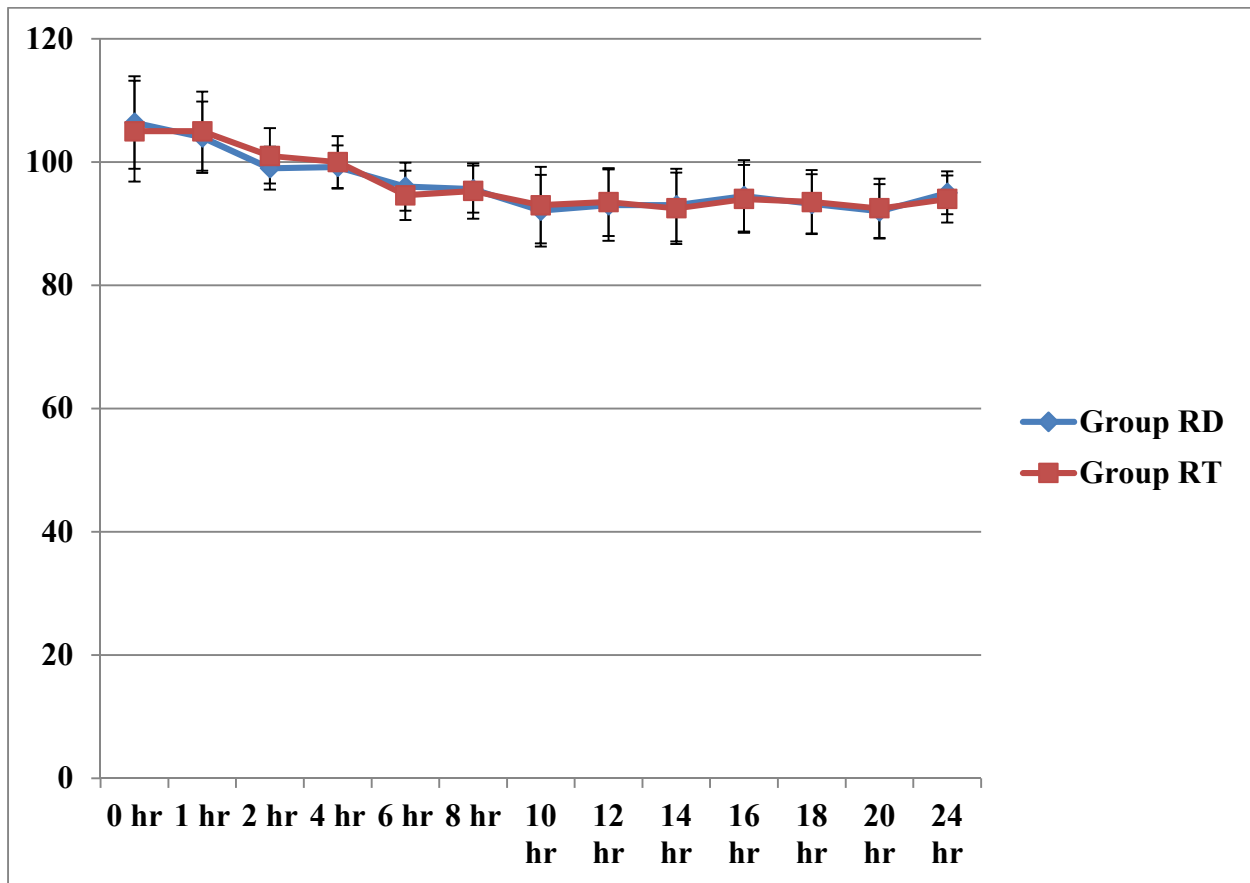
The intraoperative Systolic blood pressure was measured at regular time interval during the course of anaesthesia. The systolic blood pressure remained stable in both groups. There was no statistically significant difference in pulse rate across both groups intraoperatively.

Chart 5: Intraoperative trends of DBP in both the groups:



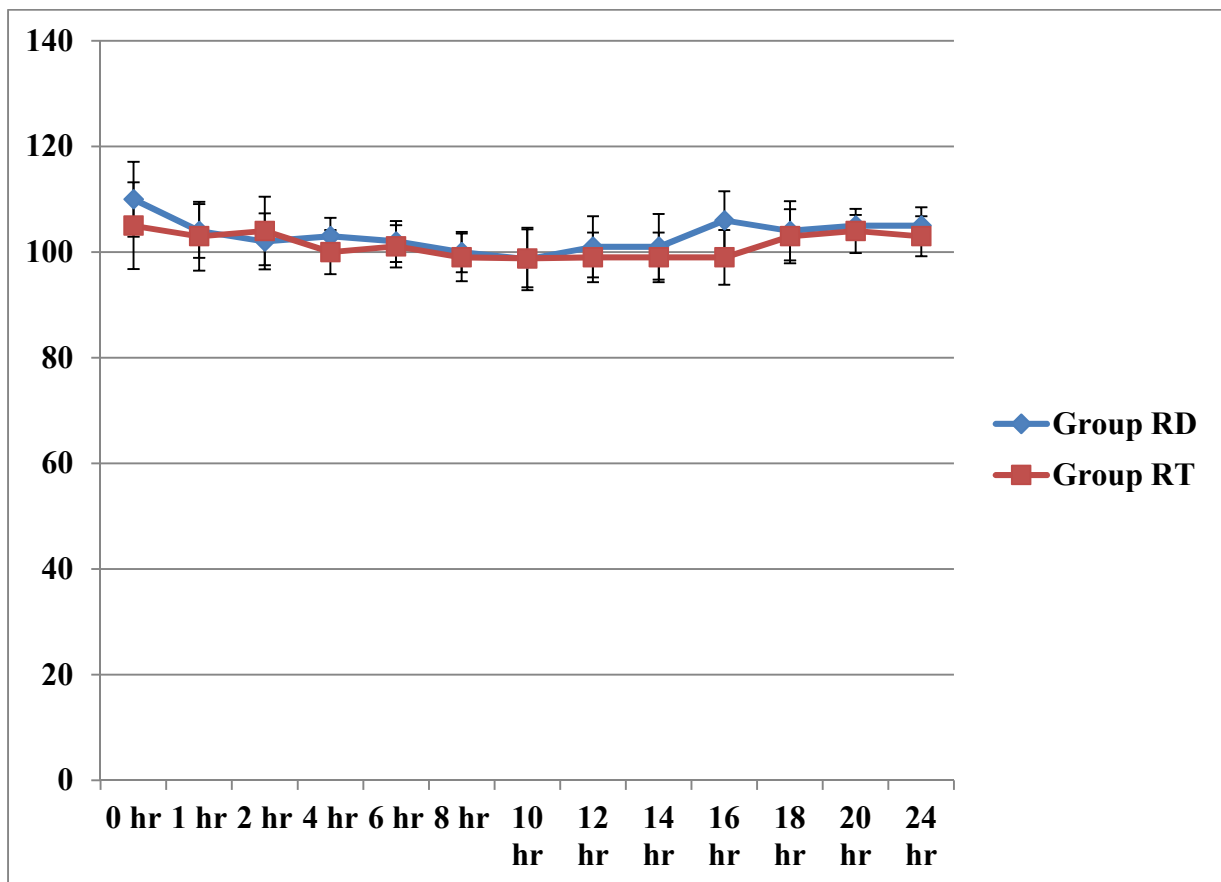
The intraoperative diastolic blood pressure was measured at regular time interval during the course of anaesthesia. The diastolic blood pressure remained stable in both groups. There was no statistically significant difference in pulse rate across both groups intraoperatively

Chart 6: Post-operative trends of pulse rate in both groups:



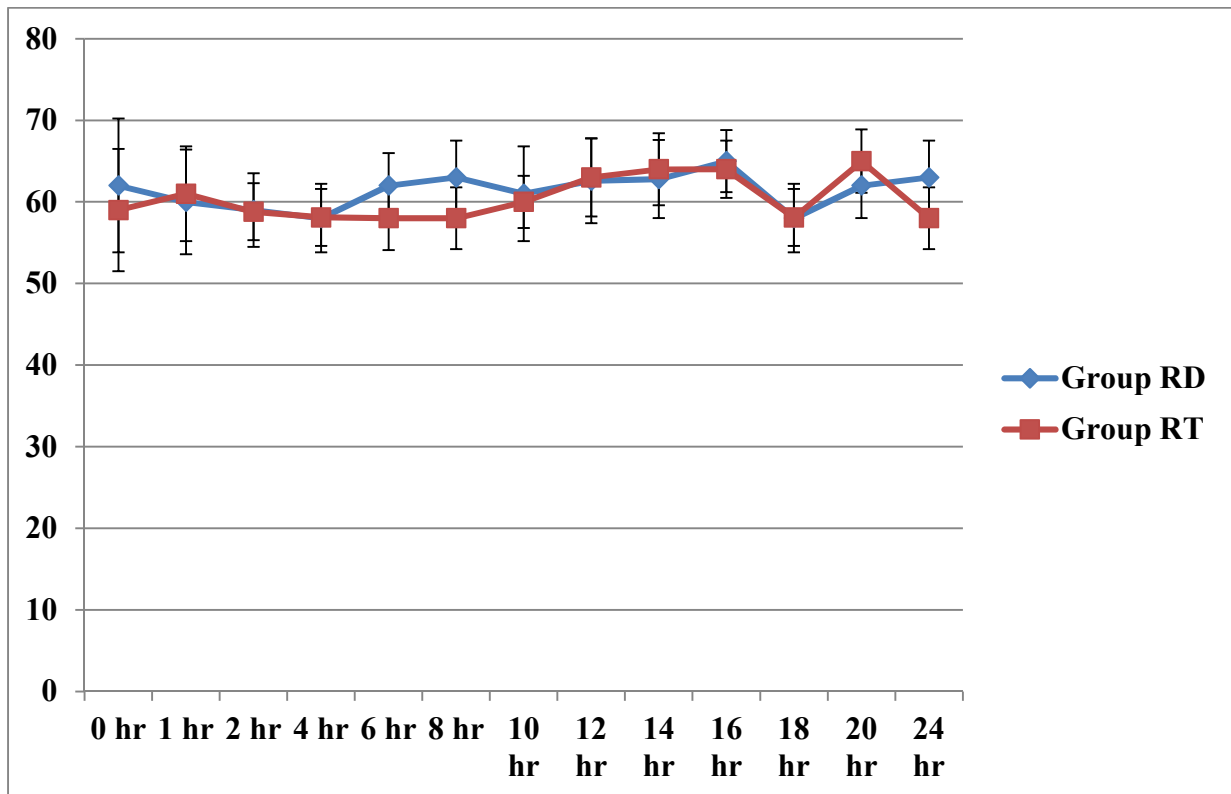
The post-operative pulse rate was measured at regular time interval following surgery. The pulse rate remained stable in both groups. There was no statistically significant difference in pulse rate across both groups post-operatively.

Chart 7: Post-operative trends of SBP in both groups:



The post-operative systolic blood pressure was measured at regular time interval following surgery. The systolic blood pressure remained stable in both groups. There was no statistically significant difference in systolic blood pressure across both groups post-operatively

Chart 8: Post-operative trends of DBP in both groups:



The post-operative diastolic blood pressure was measured at regular time interval following surgery. The diastolic blood pressure remained stable in both groups. There was no statistically significant difference in Diastolic blood pressure across both groups post-operatively

Table 6: Duration of analgesia distribution among both the study group:

Weight	Group RD	Group RT	Total	P value
Mean±SD	652±98	416±75	534±147	
Range	480 - 840	240 - 480	240 - 840	<0.001

The mean duration of analgesia in group RD is 652 minutes, mean duration of analgesia in group RT is 416 minutes. The mean duration of analgesia was more in group RD and this difference was statistically significant with $P<0.001$.

Table 7: Duration of motor block distribution among both the study group:

Weight	Group RD	Group RT	Total	P value
Mean±SD	204±40.4	188±20.7	196±32	
Range	180 - 300	180 - 240	180 - 300	0.059

The mean duration of complete motor block in group RD is 204 minutes, mean duration of complete motor block in group RT is 188 minutes. The mean duration of analgesia was more in group RD and this difference was not statistically significant with $P=0.059$

Table 8: Duration of sedation distribution among both the study group:

Weight	Group RD	Group RT	Total	P value
Mean±SD	212±40.8	142±29.4	177±50	
Range	120 - 300	120 - 180	120 - 300	<0.001

The mean duration of sedation in group RD is 212 minutes, mean duration of analgesia in group RT is 142 minutes. The mean duration of sedation was more in group RD and this difference was statistically significant with $P < 0.001$.

III. DISTRIBUTION OF FLACC SCORE

Table 9: At 4 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	30 (100%)	30 (100%)	60 (100%)	
>4	0 (0.0%)	0 (0.0%)	0 (0.0%)	0.492
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 4 hours all the patients in group RD and RT had a FLACC score of less than 4.

Table 10 At 6 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	30 (100%)	28 (93.3%)	58 (96.7%)	0.492
>4	0 (0.0%)	2 (6.7%)	2 (3.3%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 6 hours all the patients in group RD had a FLACC score of less than 4. 2 patients i.e 6.7% in group RT had FLACC score more than 4. This difference was not statistically significant with P value 0.492. (>0.05)

Table 11: At 8 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	30 (100%)	16 (53.3%)	46 (76.7%)	<0.001
>4	0 (0.0%)	14 (46.7%)	14 (23.3%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 8 hours all the patients in group RD had a FLACC score of less than 4. 14 patients i.e 23.3% in group RT had FLACC score more than 4. This difference was statistically significant with P value <0.001.

Table 12: At 10 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	26 (86.7%)	0 (0.0%)	26 (43.3%)	<0.001
>4	4 (13.3%)	30 (100%)	34 (56.7%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 10 hours 26 patients i.e 86.7% in group RD had a FLACC score of less than 4, 4 patients i.e. 13.3% had FLACC>4. In group RT 100% had FLACC score more than 4. This difference was statistically significant with P value <0.001.

Table 13: At 12 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	15 (50%)	0 (0.0%)	26 (43.3%)	<0.001
>4	15 (50%)	30 (100%)	34 (56.7%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 12 hours 15 patients i.e 50% in group RD had a FLACC score of less than 4, 15 patients i.e. 50% had FLACC>4. In group RT 100% had FLACC score more than 4. This difference was statistically significant with P value <0.001.

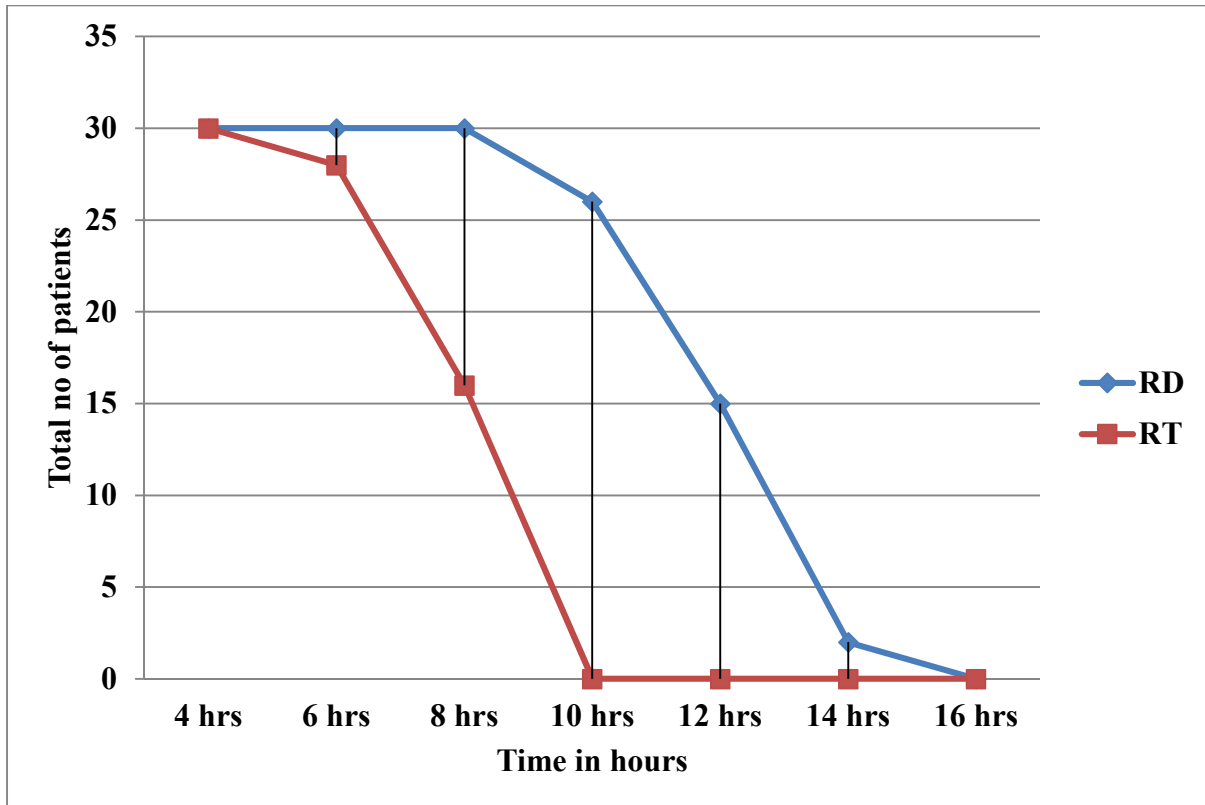
Table 14: At 14 hour proportion of adequate analgesia in both groups

FLACC SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<= 4	2 (6.7%)	0 (0.0%)	26 (43.3%)	<0.001
>4	28 (93.3%)	30 (100%)	34 (56.7%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 12 hours 2 patients i.e 6.7% in group RD had a FLACC score of less than 4, 28 patients i.e. 93.3% had FLACC>4. In group RT 100% had FLACC score more than 4. This difference was statistically significant with P value <0.001.

At 16 hours all patients in group RD and RT had FLACC more than 4

Chart 9: Face, legs, activity, cry, consolability score: Number of patients with adequate caudal analgesia (<4) in both groups at different time intervals:



At 4 hours all patients achieved complete analgesia. The number of patients with complete analgesia steadily decreased in both groups. At 8 hours all the patients in group RD had complete analgesia but only 15 patients in group RT had complete analgesia. At 10 hours no patient in group RT had complete analgesia. This difference was statistically significant with $P < 0.05$. In group RT complete analgesia lasted for lesser time than group RD.

III. DISTRIBUTION OF MODIFIED BROMAGE SCALE FOR MOTOR BLOCK

Table 15: At 1 hour proportion of adequate complete motor block in both groups

MODIFIED BROMAGE SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
3	30 (100%)	30 (100%)	60 (100%)	0.145
Total	30 (100%)	30 (100%)	60 (100%)	

At 1 hour all the patients in group RD and RT had a Modified Bromage score of 3.

Table 16: At 2 hour proportion of complete motor block in both groups

MODIFIED BROMAGE SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<3	23 (76.7%)	28 (93.3%)	51 (85%)	0.145
3	7 (23.3%)	2 (6.7%)	9 (15%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At the end of 2 hours 23 patients i.e 76.7% in group RD had a modified bromage score of less than 3. In group RT 28 patients i.e. 93.% had modified bromage score of <3. In group RD 23.3% and in group RT 6.7% had Modifiedbromage score of 3. This difference was not statistically significant with P value 0.145.

Table 17: At 3 hour proportion of complete motor block in both groups

MODIFIED BROMAGE SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<3	26 (86.7%)	29 (96.7%)	55 (91.7%)	0.353
3	4 (13.3%)	1 (3.3%)	5 (8.3%)	
Total	30 (100%)	30 (100%)	60 (100%)	

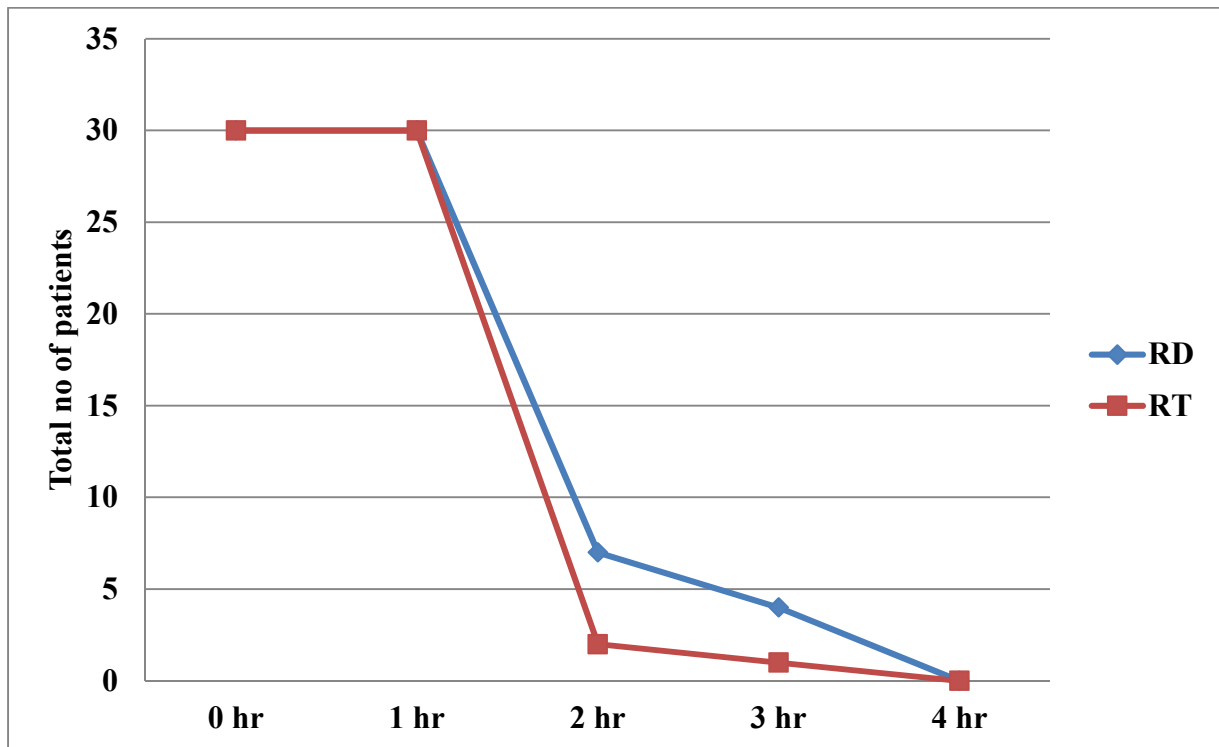
At the end of 3 hours 26 patients i.e 86.7% in group RD had a modified bromage score of less than 3. In group RT 29 patients i.e. 96.7% had modified bromage score of <3. In group RD 13.3% and in group RT 3.3% had Modifiedbromage score of 3. This difference was not statistically significant with P value 0.353

Table 18: At 4 hour proportion of complete motor block in both groups

MODIFIED BROMAGE SCORE	Group RD N = 30	Group RT N = 30	Total N = 60	P value
<3	30 (100%)	30 (100%)	60 (100%)	0.145
=3	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Total	30 (100%)	30 (100%)	60 (100%)	

At 4 hour all the patients in group RD and RT had a Modified Bromage score of less than 3.

Chart 10: Modified bromage score in both groups RD and RT:



The number of patients achieving complete motor nerve block was 30 in 1st hour in both the groups. It steadily decreased at 2 hours 7 had score 3 in RD and 2 in group RT. At 3 hours 4 had score 3 in RD and 1 had score 3 in RT. Both these difference was not significant. All the patients had recovered from complete motor block in both groups at 4 hours. The number of people achieving complete motor block was similar in both groups with $P > 0.05$.

III. DISTRIBUTION OF RAMSAY SEDATION SCORE FOR SEDATION

Table 19: Distribution of ramsay sedation score in both groups:

Time of assessment	Group RD N = 30	Group RT N = 30	P value
0 hour	3 (3 – 3)	3 (3 – 3)	----
1 hour	2 (2 -3)	2 (2 – 2)	----
2 hours	2 (1 – 2)	1 (1 – 2)	<0.001
3 hours	1 (1 – 2)	1 (0 - 1)	-----
4 hours	1 (0 – 2)	0 (0 – 1)	<0.001
5 hours	1 (0 – 2)	0 (0 – 0)	<0.001
6 hours	0 (0 - 0)	0 (0 – 0)	<0.001

The median sedation score was more in group RD. At 2, 4 and 5 hours the median score in group RD was 2, 1 and 1 respectively. At 2, 4 and 5 hours the median score in group RT was 1, 0 and 0 respectively. The score in RD group was significantly more than group RT at 2, 4 and 5 hours with $P < 0.001$. RD group achieved better sedation than RT group.

DISCUSSION

Caudal anaesthesia is a routinely done regional block for the purpose of intraoperative and postoperative analgesia. It helps to reduce the use of systemic analgesic intraoperatively.

In our study the mean duration of analgesia in group RD is 652 minutes, mean duration of analgesia in group RT is 416 minutes. The mean duration of analgesia was more in group RD and this difference was statistically significant with $P < 0.001$.

The mean duration of sedation in group RD is 212 minutes, mean duration of sedation in group RT is 142 minutes. The mean duration of sedation was more in group RD and this difference was statistically significant with $P < 0.001$.

Correlation of the study results with previous studies as follows.

Savita Gupta et al demonstrated that Caudal Dexmedetomidine added to Ropivacaine prolonged the duration of analgesia.

Saadawy et al compared caudal bupivacaine 0.25% with dexmedetomidine $1\mu\text{g}/\text{kg}$ and caudal bupivacaine alone and showed that the duration of analgesia was significantly longer with dexmedetomidine ($P < 0.001$); No statistically significant difference in hemodynamics between both

groups; Dexmedetomidine had better quality of sleep and a prolonged duration of sedation($P < 0.05$). This study showed that caudal dexmedetomidine $2\mu\text{g}/\text{kg}$ with 0.25% Ropivacaine also has similar results like Saadawy et al.

Neogi M et al compared Clonidine $1\mu\text{g}/\text{kg}$ and Dexmedetomidine $1\mu\text{g}/\text{kg}$ as an adjuncts to Ropivacaine 0.25% for caudal analgesia in paediatric patients and concluded that addition of both clonidine and dexmedetomidine with ropivacaine administered caudally, significantly increases the duration of analgesia. The patients stay haemodynamically stable and there are no undue side effects. The mean duration of analgesia was 6.32 ± 0.46 hours in group R, 13.17 ± 0.68 hours in group C and 15.26 ± 0.86 hours in group D. The prolongation of duration of analgesia was significant in both groups C and D in comparison to group R. The incidence of adverse effects was statistically insignificant between the three groups.

SUMMARY

Caudal Dexmedetomidine 2 μ g/ kg with 0.25%Ropivacaine 1ml/kg for paediatric lower abdominal surgeries achieved significant post operative pain relief up to 11 hours. It has stable hemodynamics in the intraoperative and post operative period. It provides acceptable sedation in the post operative period. No other analgesic supplementation was needed.

CONCLUSION

Caudal Ropivacaine 0.25% with Dexmedetomidine 2 µg/kg provided longer duration of analgesia and reduced requirement for rescue analgesic in the post-operative period compared to Caudal Ropivacaine 0.25% with Tramadol 2 mg/kg. Thus, dexmedetomidine with ropivacaine can be used as an alternative to tramadol with ropivacaine for paediatric infraumbilical surgeries through the caudal route as a safe and effective agent.

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**நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)**

ஆய்வு செய்யப்படும் தலைப்பு:

பங்கு பெறுவரின் பெயர்:

பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை குறிக்கவும் ✓
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	<input type="checkbox"/>
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	<input type="checkbox"/>
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	<input type="checkbox"/>
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	<input type="checkbox"/>
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.	<input type="checkbox"/>

பங்கேற்பவரின் கையொப்பம் / இடம்

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம் / இடம்

ஆய்வாளரின் பெயர்

மையம்

கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை

சாட்சியின் கையொப்பம் / இடம்

பெயர் மற்றும் விலாசம்

S.No	Group	Age(yrs)	Sex	Weight(kg)	Procedure	Duration(mins)	PRE OPERATIVE VITALS		
							HR(/min)	BP(mm Hg)	SPO2(%)
1	RD	2	M	10	IHR	30	115	124/70	99
2	RD	4	M	15	URT	45	105	130/70	100
3	RD	3	F	11	IHR	20	110	120/60	100
4	RD	1	M	8	PVSL	20	120	110/60	100
5	RD	2	M	10	IHR	25	118	110/60	100
6	RD	5	M	18	IHR	40	98	130/70	100
7	RD	4	F	15	IHR	20	102	116/80	99
8	RD	3	F	14	IHR	25	105	110/60	99
9	RD	2	M	11	IHR	40	122	125/70	100
10	RD	3	M	13	PVSL	15	115	110/60	100
11	RD	4	F	16	IHR	25	108	110/70	100
12	RD	5	M	20	URT	50	102	130/80	100
13	RD	1	M	9	PVSL	15	120	102/70	100
14	RD	3	M	14	IHR	20	110	110/60	100
15	RD	5	M	18	IHR	35	102	110/70	100
16	RD	4	M	16	URT	50	108	125/60	99
17	RD	2	F	11	IHR	15	112	110/60	100
18	RD	3	F	12	IHR	20	108	110/60	100
19	RD	4	M	16	URT	45	110	120/70	100
20	RD	5	M	20	IHR	35	102	120/76	100
21	RD	3	M	10	PVSL	15	112	105/68	100
22	RD	4	M	14	IHR	20	106	110/62	100
23	RD	1	M	8	PVSL	10	126	105/74	99
24	RD	2	F	11	IHR	30	120	110/58	100
25	RD	2	F	10	IHR	20	118	100/42	100
26	RD	4	M	15	URT	45	106	110/56	100
27	RD	3	M	13	IHR	20	115	105/48	100
28	RD	3	M	12	CIR	15	116	110/50	100
29	RD	5	F	18	IHR	40	110	130/70	99
30	RD	4	M	16	URT	45	120	126/68	99
31	RT	3	F	12	IHR	20	120	105/60	100
32	RT	4	M	15	IHR	35	105	110/60	99
33	RT	2	M	10	IHR	15	110	100/56	100
34	RT	1	F	9	IHR	15	125	100/60	100
35	RT	2	M	11	URT	45	110	110/60	100
36	RT	5	M	20	IHR	20	102	120/60	100
37	RT	6	M	22	IHR	15	105	120/50	99
38	RT	5	F	18	IHR	15	110	110/60	99
39	RT	4	M	16	PVSL	10	112	110/70	100
40	RT	3	M	14	URT	40	120	105/60	100
41	RT	1	M	9	IHR	20	125	100/70	100
42	RT	2	M	10	IHR	15	120	106/60	100
43	RT	5	M	21	IHR	15	98	110/70	100
44	RT	4	F	17	IHR	20	110	110/56	99
45	RT	5	M	19	IHR	10	102	112/60	99
46	RT	3	M	13	IHR	20	105	100/70	99
47	RT	2	F	11	IHR	15	112	100/56	100
48	RT	1	F	8	IHR	10	1206	102/60	100
49	RT	1	M	9	PVSL	10	130	100/70	100
50	RT	2	M	11	IHR	20	120	110/60	100
51	RT	4	M	17	URT	50	110	110/70	100
52	RT	3	F	14	IHR	20	115	100/60	99
53	RT	5	M	21	URT	45	105	120/60	99
54	RT	4	M	14	PVSL	10	112	115/60	99
55	RT	3	F	11	IHR	15	110	110/70	99
56	RT	1	M	9	IHR	10	130	100/60	100
57	RT	3	M	15	IHR	15	110	108/70	100
58	RT	2	M	12	PVSL	10	105	100/68	100
59	RT	2	M	11	URT	50	115	110/70	100
60	RT	3	M	14	IHR	20	106	100/80	100

