“STUDY OF EFFECT OF NEBULISED DEXMEDETOMIDINE ON THE ONSET AND DURATION OF SPINAL ANAESTHESIA, A ONE YEAR, HOSPITAL-BASED, RANDOMISED CLINICAL TRIAL.”

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ABSTRACT

Title: Study of effect of nebulised dexmedetomidine on the onset and duration of spinal anaesthesia, a one year, hospital-based, randomised clinical trial

Background

Spinal anaesthesia is widely used for procedures below the umbilicus. Adjuvants like dexmedetomidine enhance its duration and quality by providing better pain relief and reducing local anaesthetic doses, though they have side effects. Dexmedetomidine, a potent alpha-2 adrenoceptor agonist, is gaining popularity due to its sedative, analgesic, and anxiolytic properties. This study examines the effect of nebulized dexmedetomidine on the onset and duration of spinal anaesthesia.

Aims and Objectives

To study the effect of nebulized dexmedetomidine on onset and duration of sensory and motor block in spinal anaesthesia. Also, to note the time to first rescue analgesia, and total analgesic requirement in the first twenty four hours postoperatively. To study the level of sedation after administration of the drug and to study side effects of the drug, if any.

Methodology

A randomised clinical trial was conducted on 80 patients undergoing infra-umbilical surgery. Patients were divided into two groups: Group I (dexmedetomidine nebulization) and Group II (saline

nebulization).Under strict asepsis, the patient received Spinal anaesthesia in L3-L4 space using 3.2 ml of 0.5% Bupivacaine(Hyperbaric) having the patient in sitting position.

Patient was then made supine.
Group I were then administered dexmedetomidine nebulisation at the dose of 1 mcg/kg diluted with Normal Saline up to 5 ml.
Group II patients were nebulised with 5 ml of saline.
Measurements included onset and duration of sensory and motor blocks, time to first rescue analgesia, and total analgesic requirements.

Results

Dexmedetomidine significantly prolonged the duration of both sensory and motor blocks and delayed the need for postoperative analgesia. No significant difference was noted in the onset of sensory block, but a faster onset of motor block was observed in the control group. Side effects were minimal, and dexmedetomidine was found to be a safe additive to spinal anaesthesia.

Conclusion

Nebulized dexmedetomidine effectively enhances spinal anaesthesia, offering extended pain relief and reduced postoperative analgesic requirements. Despite its promising results, further research with larger sample sizes and multi-center trials is needed for broader validation .

Keywords: Dexmedetomidine, spinal anaesthesia, nebulisation

LIST OF ABBREVIATIONS

ASA	-	American Society of Anaesthesiologists
HR	-	Heart Rate (bpm)
SBP	-	Systolic Blood Pressure (mm Hg)
DBP	-	Diastolic Blood Pressure (mm Hg)
SpO_2	-	Saturation of peripheral oxygen (%)
L_n	-	Lumbar sensory dermatome level
mcg.	-	micrograms
min.	-	minutes
kgs.	-	kilograms
cms.	-	centimetres
Mm Hg	-	Millimetres of Mercury
MW-U	-	Mann-Whitney U test
PCT	-	Paracetamol
CSF	-	Cerebrospinal fluid
RSS	-	Ramsay Sedation Score
Inj.	-	Injection

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INTRODUCTION

Spinal anaesthesia has been extensively used to provide anaesthesia for surgical procedures below the umbilicus.

However, the disadvantage of spinal anaesthesia is that its action is limited and may not be suitable for very long procedures. In some cases, the surgery may be prolonged, requiring conversion to general anaesthesia, which has its own set of disadvantages. Hence, to enhance spinal anaesthetic action, a number of drugs have been used as adjuvants to local anaesthetics while administering spinal anaesthesia.

These adjuvants provide improved spinal anaesthetic block, better pain relief, and reduce the required local anaesthetic dosage, thus minimising potential side-effects of the local anaesthetics. Common Adjuvants for Spinal anaesthesia include: Opioids like Fentanyl, Morphine, Sufentanil, Clonidine, Epinephrine, Ketamine,Magnesium Sulphate,Midazolam and Dexamethasone[1][2].

However, these adjuvants are associated with numerous side effects, which include pruritus (itching), Nausea and vomiting,Urinary retention, Psychomimetic effects (hallucinations, nightmares), Increased salivation, Hypotension, Bradycardia, Excessive Sedation, tachycardia, hypertension etc. Respiratory depression in case of opioids, and midazolam has also been reported. Hence, close monitoring is recommended particularly in such cases.

Dexmedetomidine is a new, potent, adrenergic drug that shows selective alpha 2 agonism (with an affinity of 1600 times to alpha 1). Dexmedetomidine binds to centrally-acting α_2-adrenergic receptors, particularly in the spinal cord. This action prevents norepinephrine release, resulting in analgesia and sedation. It also modulates pain pathways, providing a synergistic effect with local anaesthetics.

Dexmedetomidine, when administered Intravenously, has been found to prolong spinal anaesthesia. It also greatly extends the sensory and motor block duration, resulting in prolonged analgesia both

during and following surgery. Also, local anaesthetic dose reduction is made possible with the use of dexmedetomidine and hence, minimises potential side effects and toxicity. It also provides mild to moderate sedation without causing respiratory depression, making it ideal for patients who may benefit from some level of sedation during the procedure[3]. Patients receiving dexmedetomidine as an adjuvant often report lower pain scores and decreased need for postoperative analgesics.

However, Dexmedetomidine when given by Intravenous route is known to produce bradycardia and hypotension, which limits its use as spinal anaesthetic adjuvant. Nebulised dexmedetomidine has the advantage of not causing bradycardia unlike the intravenous route[4].

Therefore, this study was undertaken to determine the effect of nebulised dexmedetomidine, which is administered during the intraoperative phase, on spinal anaesthesia, particularly the onset and duration of spinal anaesthesia.

AIMS AND OBJECTIVES

Aim

To Study the effect of nebulised Dexmedetomidine on onset and duration of Spinal anaesthesia-a one year hospital based Randomised Clinical Trial

Objectives

Primary objective:-

- to investigate how nebulized dexmedetomidine affects the onset and duration of motor and sensory block during spinal anaesthesia.

Secondary objectives:

- To determine:
    1. Time to first postoperative rescue analgesia.
    2. Cumulative requirement of analgesia in the initial twenty four hours postoperatively.
    3. To study the level of sedation after administration of the drug.
    4. To study about the adverse effects of the drug, if present.

BASIC SCIENCES

ANATOMY

Spinal Cord:

The spinal cord is a long, cord-like structure that is a part of the Central Nervous System and is responsible for providing nerve supply from the trunk to all four peripheral limbs[26].

VERTEBRAL COLUMN

The main function of the vertebral column is to protect the spinal cord. It comprises of 33 vertebral bodies which comprises of:

- Cervical - 07
- Thoracic - 12
- Lumbar - 05
- Sacral - 05 (fused to 1)
- Coccyx - 04 (fused to 1)

CURVES OF THE SPINE

The vertebral column has four curves in adults.

- Cervical curve - Convex anterior
- Thoracic curve - Concave anterior
- Lumbar curve - Convex anterior
- Sacral curve - Concave anterior

The curves of the spine are important when the patient is in supine position[26]. The highest point of cervical and lumbar curves in supine position are at cervical-C5 and lumbar-L5, lowest points of thoracic and sacral at T5 and S2 respectively. Spread of local anaesthetic in the subarachnoid space is

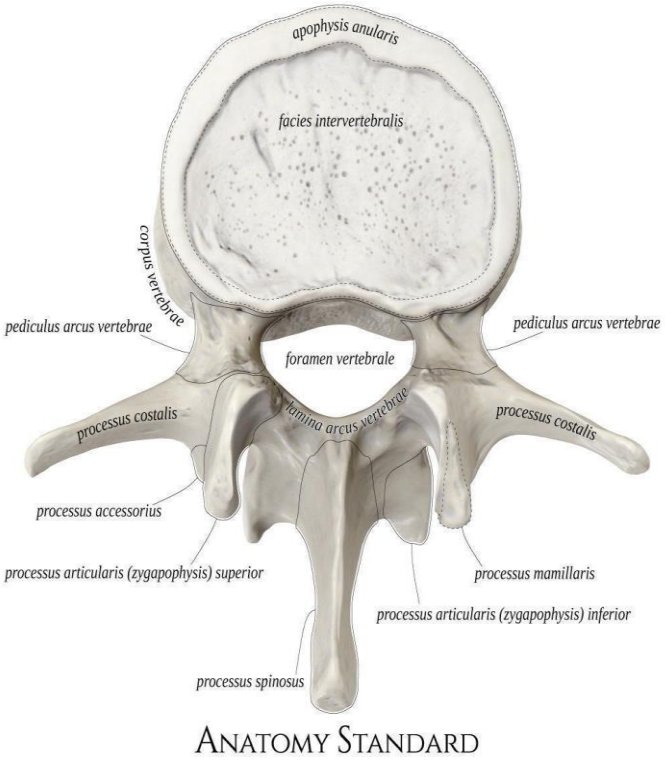
determined by the vertebral column curves, baricity of the local anaesthetic solution and positioning of the patient.

General Structure of the Lumbar Spine

The lumbar spine consists of five vertebral bodies in the lower back, labelled L1 through L5. These are the largest and most robust vertebrae, as they carry the majority of the body's weight and are subject to significant mechanical stress[26].

Components of Lumbar Vertebrae

Each lumbar vertebra shares a common structural anatomy, which includes several key components[26]:



1. Vertebral Body: The largest part of the vertebra, located at the front of the spine. It is cylindrical in shape and provides structural support and bears weight. The vertebral bodies of the lumbar spine are larger than those in other regions to support more weight.

2. Pedicles and Laminae: These structures form the vertebral arch, which is posterior to the vertebral body. The pedicles are short, stout processes that extend from the back of the vertebral body, connecting to the laminae, which join to complete the arch.

3. Spinous and Transverse Processes: The spinous process protrudes posteriorly from the vertebral arch and serves as a point of attachment for muscles and ligaments. The transverse processes extend laterally from each side of the vertebral arch, also serving as muscle and ligament attachment sites.

4. Articular Processes: These include superior and inferior projections that articulate with adjacent vertebrae to form facet joints, which guide and restrict the motion of the spine. The orientation of lumbar facet joints allows for significant flexion and extension while limiting rotation.

5. Intervertebral Foramina: These are openings formed between the pedicles of adjacent vertebrae through which spinal nerves exit the spinal canal.

Intervertebral Discs

Situated between the vertebral bodies are intervertebral discs, which act as cushions and shock absorbers[26]. Each disc has two components:

Nucleus Pulposus: A gel-like core that redistributes pressure across the disc.
Annulus Fibrosus: A tough, fibrous ring that encloses the nucleus pulposus and binds the vertebrae together.

Spinal Canal

The spinal canal runs through the centre of the vertebral arch and houses the spinal cord and its coverings. In the lumbar region, the spinal cord typically ends at the level of the first or second lumbar vertebra, continuing as the cauda equina, a bundle of nerve roots.

Ligaments

Several ligaments stabilise the lumbar spine, including:
Anterior and Posterior longitudinal ligaments: These run along the front and back of the vertebral bodies, providing support and stability.
Ligamentum Flavum: connects the laminae of adjacent vertebrae and preserves the integrity of the spinal canal.
Supraspinous Ligament: It is a strong fibrous ligament which connects the apices of the spinous processes from the C7 vertebra to the Sacrum. It is also known as LIGAMENTUM NUCHAE in the area above the C7 vertebra.

Interspinous Ligament: It is a thin membranous ligament which connects the spinous processes and it blends anteriorly with ligamentum flavum and posteriorly with supraspinous ligament.

Ligamentum Flavum: It comprises yellow elastic fibres and connects the adjacent lamina. It begins laterally at the root of the articular processes and extends posteriorly and medially to the point where laminae join to form the spinous process.

Functions of the Lumbar Spine

The lumbar spine plays several critical roles:

Support and Load Bearing: It supports the upper body's weight and distributes forces during movement.

Flexibility and Movement: It allows for flexion, extension, and slight lateral bending.

Protection: It encases and protects the lower spinal cord and cauda equina.

CEREBROSPINAL FLUID

It is a clear, colourless fluid found in the cranial and spinal subarachnoid spaces and in the ventricles. It is mainly formed by either secretion or ultrafiltration from the choroidal plexus of the lateral ventricles. It flows from the lateral ventricles into the third ventricle through the Foramen of Monroe and then into the fourth ventricle through the Aqueduct of Sylvius[26]. CSF then flows into the Cisterna Magna (cerebro-medullary cisterna) through the Foramen of Magendie and Foramen of Luschka. From the Cisterna Magna, CSF enters the Subarachnoid Space which bathes the brain and spinal cord before it is absorbed into the arachnoid granulations which are present over the cerebral hemispheres.

COMPOSITION OF CSF:

- Specific Gravity : 1.003 to 1.009 at 37°C
- Volume : 120 ml to 150 ml (25 ml to 35 ml in the spinal space)
- CSF pressure : 60-80 mmHg in the Lumbar space
- pH : 7.27 to 7.37
- PCO_2 : 48 mmHg

- HCO_3 : 23 mEq/L
- Sodium : 135-145 mEq/L
- Calcium : 2-3 mEq/L
- Phosphorus : 1.6 mg/dl
- Magnesium : 2-2.5 mEq/L
- Chloride : 15-20 mEq/L
- Proteins : 23-38 mg/dl

Spinal Anaesthesia

Spinal anaesthesia, also known as spinal block, subarachnoid block, or intradural block, is a form of regional anaesthesia involving the injection of a local anaesthetic into the subarachnoid space around the spinal cord[]. This space is filled with cerebrospinal fluid (CSF), which surrounds and cushions the spinal cord and brain. The procedure is typically performed in the lower back, in the lumbar region. The primary purpose of spinal anaesthesia is to block the transmission of nerve impulses, particularly pain signals, from the lower parts of the body during surgical procedures. This type of anaesthesia is commonly used for surgeries in the lower abdomen, pelvis, and lower extremities. It can be a preferable option for patients undergoing caesarean sections, orthopaedic surgeries on lower limbs, and various urological and gynaecological procedures.

Spinal anaesthesia offers several advantages, such as a significant reduction in pain and the avoidance of the risks and side effects associated with general anaesthesia, such as respiratory complications and postoperative nausea and vomiting. However, it also has potential drawbacks and risks, such as headaches caused by CSF leaks (post-dural puncture headache), hypotension (low blood pressure), and, in rare cases, nerve damage or infections.

PHYSIOLOGY OF SUBARACHNOID BLOCK

The various factors that control the different effects of a spinal anaesthetic technique are as follows[]:

- The amount and the type of drug
- Volume of the drug solution
- The site of injection
- The rate of injection
- Specific gravity of the solution- baricity and density
- Barbottage - repeatedly withdrawing and re-injecting local anaesthetic

The factors that affect the spread of local anaesthetics are:

1.Patient factors:

- Age
- Height
- Position
- Cerebrospinal fluid volume
- Spinal column configuration

2. Technical Factors:

- Site of injection
- Direction of the needle
- Dose of the local anaesthetic
- Baricity of the local anaesthetic
- Volume of the local anaesthetic

The nerve roots as well as spinal cord take up the local anaesthetics after injection into the subarachnoid space. More the surface area of exposure of the nerve root, more is the uptake of the drug. There are two mechanisms by which the local anaesthetic gets taken up:

- The first mechanism is by diffusion from the CSF into the pia mater and into the spinal cord. This is a slow process and only the most superficial portion of the spinal cord is affected.
- The second mechanism of local anaesthetic uptake is by extension into the spaces of Virchow-Robin which is the area of pia mater that surround the blood vessels which penetrate the Central Nervous System. These spaces connect with perineural clefts that surround the nerve cell bodies in the spinal cord.

The site of action is on both the anterior and posterior nerve roots, affecting the smaller nerve fibres first and the thick large motor nerve fibres last.

The sympathetic fibres are affected first and are last to recover. The sympathetic blockade is more diffuse and extends two to four segments above the motor block. Sympathetic blockade is the major determinant of physiologic response to spinal anaesthesia.

The motor blockade is usually the last to be affected and first to recover.

SEQUENCE OF SPINAL ANAESTHESIA BLOCKADE

- Vasomotor Block: Dilatation of skin vessels and increase cutaneous blood flow.
- Temperature Fibres: Cold sensation lost first followed by warm sensation.
- Loss of temperature discrimination
- Pain- Pin prick sensation lost first
- Loss of tactile sensation
- Motor paralysis
- Loss of pressure sensation
- Loss of proprioception and vibratory sensation

SYMPATHETIC BLOCKADE

Sympathetic fibres are usually blocked two to three segments above the sensory blockade and sensory blockade is two segments higher than motor blockade. Since the level of sympathetic denervation determines the magnitude of cardiovascular response to subarachnoid block, it could be anticipated

that higher the level of neural blockade, greater would be the changes in the cardiovascular and hemodynamic parameters.

PRE-PROCEDURAL CONSIDERATIONS:

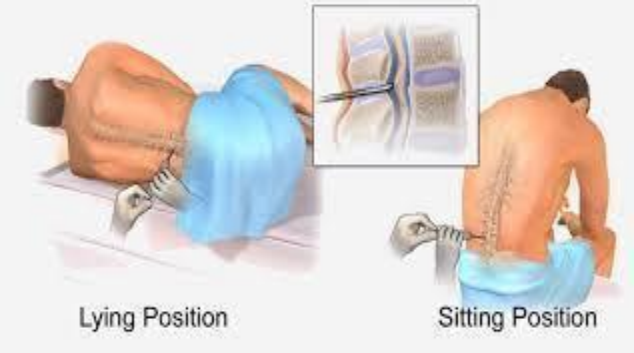
Before the administration of spinal anaesthesia, a thorough pre-anaesthetic assessment is essential. This includes evaluating the patient's medical history, physical examination, and any potential contraindications such as infection at the injection site, patient refusal, or specific neurological disorders. Patients are typically advised to fast for several hours prior to the procedure to reduce the risk of aspiration during surgery.

Positioning the Patient

Proper patient positioning is crucial to the success of spinal anaesthesia[26]. The two common positions used are:

1. Sitting Position: The patient sits on the edge of the bed or on a chair, with their feet supported by a stool. They are asked to arch their back, pushing the spine outwards to widen the spaces between the vertebrae, which facilitates easier needle insertion.

2. Lateral Decubitus Position: The patient lies on their side, with their legs curled towards the chest and the back arched outward. This position is often preferred for patients who cannot sit up due to medical conditions.



Technique and Local anaesthetic

A strict sterile technique is important to avoid infections such as meningitis. The area of the lower back where the needle will be inserted is cleaned with an antiseptic solution, and sterile drapes are used to cover the surrounding areas.

A local anaesthetic may be administered to the skin and superficial tissues to minimise discomfort during the needle insertion. This step is performed using a small needle before the introduction of the spinal needle.

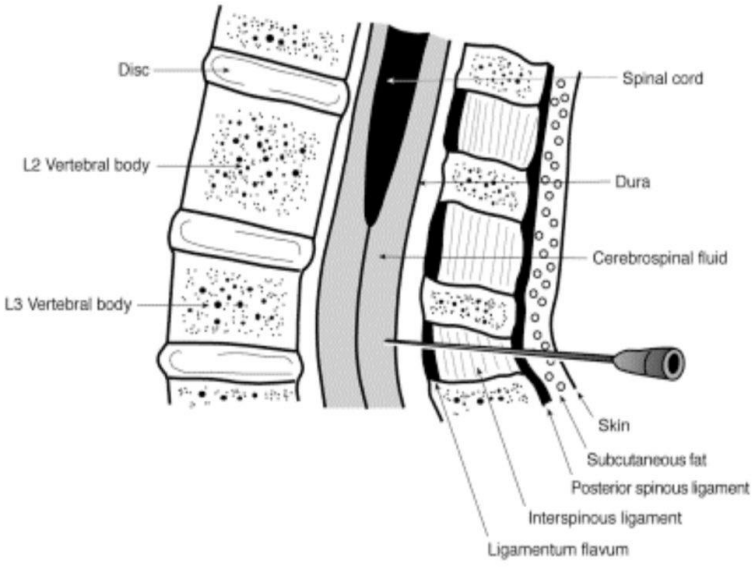
Needle Insertion and Identification of the Subarachnoid Space

Topographical Line of Tuffier: It is an imaginary line that passes between the highest points of the iliac crests which crosses the spine of the fourth lumbar vertebra in the upright position. When the patient is in lateral decubitus position it may pass through the L4-L5 interspace. The lumbar spaces are identified by using the superior iliac crest.

A long, thin, hollow needle is inserted through the skin between two lumbar vertebrae, typically between L3 and L4 or L4 and L5. This is guided by anatomical landmarks:
Intervertebral Spaces: Identified by palpating the back.

The needle passes through several layers:

1. Skin and Subcutaneous Tissue
2. Supraspinous Ligament
3. Interspinous Ligament
4. Ligamentum flavum
5. Dura Mater
6. Arachnoid Mater



When the needle penetrates the dura mater, it enters the subarachnoid space. The free flow of CSF through the needle confirms its correct placement in the subarachnoid space.

Injection of anaesthetic Agent

Once the subarachnoid space is correctly identified by the flow of CSF, a local anaesthetic is injected. The choice of anaesthetic agent and the volume depend on the type and duration of the surgery, as well as the desired level of sensory and motor block. Common agents include bupivacaine, lidocaine, and ropivacaine.

Monitoring and Managing Side Effects

After the anaesthetic is administered, the patient is carefully monitored. Blood pressure and heart rate are watched closely as hypotension is a common side effect due to sympathetic nerve blockage. Other potential complications include post-dural puncture headache, transient neurological symptoms, and, rarely, serious neurological injury.

The patient is typically laid flat or in a slight head-down position after the injection to allow the anaesthetic to distribute adequately by gravity within the CSF.

Post-procedural Care

Following the surgery, patients are monitored in a recovery area until the effects of anaesthesia wear off. They are assessed for return of motor and sensory function, and for any signs of complications such as headache or neurological deficits.

Indications of Spinal anaesthesia

Spinal anaesthesia is indicated for surgical procedures that require numbness in the lower half of the body[27]. Common indications include:

1. Surgical Procedures: Particularly effective for surgeries on the lower limbs (such as knee and hip surgeries), lower abdomen, pelvic regions, and for certain types of childbirth, including caesarean sections.

2. Pain Management: It can be used to manage pain during and after surgical procedures. It is often preferred because it allows patients to remain awake and avoid the side effects of general anaesthesia, such as nausea and respiratory complications.

3. Patient Preference: Some patients may prefer spinal anaesthesia to avoid the side effects and complications associated with general anaesthesia, especially those with respiratory issues.

4. Quick Recovery: It allows for a quicker recovery of cognitive functions compared to general anaesthesia, making it suitable for patients who need to be alert soon after the procedure.

Contraindications of Spinal anaesthesia

While spinal anaesthesia is beneficial, there are certain conditions where it should be avoided:

1. Patient Refusal: A patient's informed refusal is a primary contraindication.

2. Infection at the Site: The presence of an infection at the intended site of needle insertion increases the risk of spreading the infection into the central nervous system.

3. Bleeding Disorders: Conditions that impair coagulation (such as haemophilia) or treatment with anticoagulants are contraindications due to the risk of developing a spinal hematoma, which can compress the spinal cord.

4. Increased Intracranial Pressure: Patients with conditions that increase intracranial pressure (such as brain tumours) may be at risk of brain herniation following the reduction in cerebrospinal fluid pressure from the spinal tap.

5. Severe Hypovolemia: Spinal anaesthesia can exacerbate hypotension in patients with low blood volume, which can be dangerous.

6. Allergy to anaesthetics: Known allergies to the drugs used in spinal anaesthesia are a clear contraindication.

7. Certain Neurological Disorders: Conditions like multiple sclerosis can be worsened by spinal anaesthesia, although the data is not definitive.

8. Severe Aortic Stenosis: Due to the risk of significant hypotension, which can critically reduce cardiac output in patients with this condition.

9. Uncooperative Patient: Patients who are unable to remain still during the procedure may face increased risk of complications from needle misplacement.

PHARMACOLOGY

Local anaesthetic drugs are the drugs that cause reversible blockade of conduction of nerve impulses.

The properties of an ideal local anaesthetic agent are:

- Short latency
- High potency
- High diffusion
- Low toxicity
- Complete reversibility of its actions
- Prolonged duration of action
- No tachyphylaxis
- Stability and ability withstand heat sterilisation

CLASSIFICATION

They are divided into two groups depending on the link between the aromatic portion and the intermediate chain- the amino-ester group and the amino-amide group[27][29].

The amino-ester group has an ester link and they are Procaine, Chlorprocaine and Amethocaine.

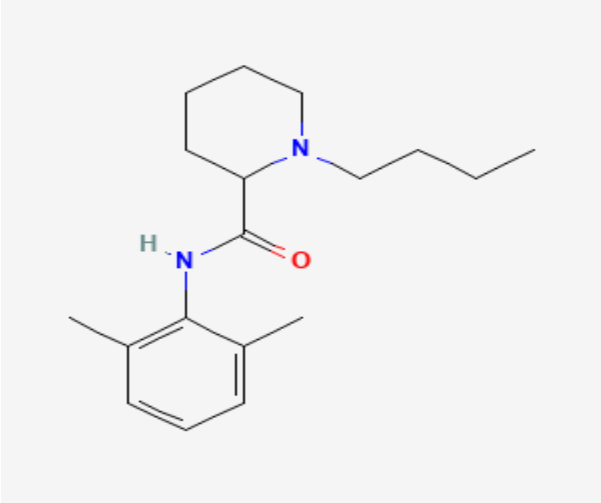
The amino-amide group has an amide link between the aromatic head and the intermediate chain and they are Lignocaine, Bupivacaine, Levobupivacaine, mepivacaine, Prilocaine, Ropivacaine and Etidocaine.

PHARMACOLOGY OF LOCAL ANAESTHETICS

These drugs produce reversible blockade of nerve conduction along the central and peripheral nerve pathways. When their concentration is increased gradually, the transmission of autonomic, somatic sensory and somatic motor impulses are interrupted in the same sequence. This leads to autonomic blockade, sensory anaesthesia and muscle paralysis in the area supplied. These drugs get gradually removed by absorption into the systemic circulation which leads to the reversal of the blockade.

MOLECULAR STRUCTURE

Local anaesthetics have a lipophilic group (usually an aromatic benzene ring) separated from a hydrophilic group (usually a tertiary amine) by an intermediate chain that includes an ester or amide linkage, which forms the basis of its classification as either esters or amides.



BUPIVACAINE

Bupivacaine is a long acting, amide local anaesthetic[29]. It was first prepared by A.F. Ekenstam in 1957 and introduced by Telivuo in 1963. It is chemically related to lignocaine and its structure is similar to that of Mepivacaine except for the amide containing group is a butyl piperidine. Its potency is approximately four times that of Lignocaine.

It is a popular drug for its long duration of action along with its tendency to provide both sensory and motor block and prolonged analgesia during the postoperative period.

Bupivacaine can be used to provide effective analgesia for several days through the use of indwelling catheters and continuous infusions.

PHYSICOCHEMICAL PROPERTIES

- Chemical Name: 1-N-butyl-(2,6-Dimethylphenyl)piperidine-2-carboxamide
- Molecular weight: 324.9
- Solubility: 28
- pKa: 8.1
- Half-life: 1.5 to 5.5 hours in adults and 8.1 hours in neonates
- Specific gravity: 1.026 at 37°C
- Volume of distribution: 73 litres

PHARMACOLOGY

The addition of a butyl group to the piperidine Nitrogen of Mepivacaine makes Bupivacaine 35 times more lipid soluble.

POTENCY

It is approximately 3-4 times more potent than Mepivacaine or Lignocaine.

ONSET AND DURATION OF ACTION

The onset of action of Bupivacaine is between 5-7 minutes and the maximum anaesthesia is achieved in 15-20 minutes.

The duration of action depends on the type of block- average duration of epidural block is 2.5-4 hours and average duration of spinal block is 2-3 hours.

MECHANISM OF ACTION

The mechanism of action of Bupivacaine is similar to that of Lignocaine.

Local anaesthetics bind to a specific site in the voltage gated sodium channels and block it thereby preventing sodium ion influx through the individual channels and preventing impulse propagation. Local anaesthetic binding to sodium channels does not alter the resting membrane potential.

As a consequence of more channels binding a local anaesthetic, the threshold for excitation and impulse conduction in the nerves increase and the rate of rise and magnitude of the action potential decrease and impulse conduction velocity slows. When a large amount of local anaesthetic concentrations occur, action potentials can no longer be generated and impulse propagation is abolished.

The mechanism of sodium conductance blockade:

- The cationic form of local anaesthetic drug acts on the receptors within the Na^+ channels on the cell membrane and blocks it. The local anaesthetic can reach the Na+ channel either via the lipophilic pathway directly across the lipid membrane or via the axoplasmic opening.
- The second mechanism is a non-specific action, i.e. by membrane expansion.

AVAILABLE CONCENTRATIONS OF BUPIVACAINE

0.25% and 0.5%

DOSAGE OF BUPIVACAINE

Maximum dosage: 3 mg/Kg body weight.

Adrenaline prolongs its action only marginally, if at all.

Tachyphylaxis is much less likely than with Lignocaine.

METABOLISM AND ELIMINATION

Bupivacaine gets metabolised by the following mechanisms:

- Aromatic hydroxylation
- N-dealkylation
- Amide hydrolysis
- Glucuronide- conjugation

The chief mechanism is N-dealkylation and the metabolite is N-desbutyl Bupivacaine.

The mean total of urinary excretion of Bupivacaine and its dealkylation and hydroxylation metabolites account for >40% of the total anaesthetic dose.

ACTIONS

ON CENTRAL NERVOUS SYSTEM

Higher concentrations of Bupivacaine produces dizziness and light-headedness followed by visual and auditory disturbances such as difficulty to focus and tinnitus.

Shivering and muscular tremors and tremors of facial muscles can occur.

The plasma concentration of Bupivacaine that is associated with seizures is 4.5 to 5.5 mcg/ml.

ON CARDIOVASCULAR SYSTEM

Usually, the cardiovascular system is more resilient to the toxic effects of high plasma concentrations of local anaesthetics.

Lignocaine concentrations <5 mcg/ml are devoid of adverse effects but causes decrease in automaticity. Lignocaine concentrations of 5-10 mcg/ml can produce profound hypotension due to arteriolar vascular smooth muscle relaxation direct myocardial depression.

Blockade of cardiac Na^+ channels by local anaesthetics contributes to anti-dysrhythmic properties.

With increase in concentration, more Na^+ channels get blocked and conduction and automaticity become affected adversely. This is evident by prolonged PR intervals and QRS complexes.

Accidental injection of Bupivacaine may result in precipitous hypotension, cardiac dysrhythmias and AV heart block.

Most common dysrhythmias include widening of QRS complexes, premature ventricular contractions and Ventricular tachycardia.

Cardiotoxicity with Bupivacaine is seen when its plasma concentration is 8-10 mcg/ml.

Pregnancy may increase sensitivity to the cardiotoxic effects of Bupivacaine.

Cardiotoxic threshold of Bupivacaine may be decreased in patients being treated with drugs like Digitalis, Calcium Channel Blockers and Beta Blockers.

Epinephrine and Phenylephrine can increase cardiotoxicity of Bupivacaine-induced inhibition of catecholamine-induced production of cyclic AMP.

Bupivacaine blocks cardiac Na+ channels during systole but due to its high lipid solubility it gets dissociated during diastole. This explains its persistent depressant effect on Vmax and hence greater cardiotoxicity. The R-enantiomer of Bupivacaine is more cardiotoxic.

Tachycardia can enhance frequency dependent blockade of cardiac Na+ channels by Bupivacaine.

Treatment of Bupivacaine toxicity:

Bretyllium 20 mg/Kg IV reverses Bupivacaine-induced cardiac depression and increases the threshold for ventricular tachycardia.

Lipid Emulsion is also used for the treatment of cardiotoxicity with its usage recommended at the earliest sign of toxicity. Initial bolus of 1.5 ml/Kg of 20% Lipid Emulsion followed by 0.25 ml/Kg/min infusion should be continued for at least 10 minutes after the circulatory stability is established.

RESPIRATORY SYSTEM

Local anaesthetics in very high plasma levels depresses medullary respiratory centre which can precipitate decreased oxygenation.

TOXICITY OF BUPIVACAINE

The toxic plasma concentration of Bupivacaine is >3 mcg/ml. The cardiotoxic effects of Bupivacaine become evident when the plasma concentration becomes 8-10 mcg/ml.

PHARMACOKINETICS

Bupivacaine levels are detectable in the blood 5 minutes after infiltration. Peak blood concentrations depend on the total dosage given and ranges between 0.14 to 0.18 mcg/ml. These levels are from 5 minutes to 2 hours of infiltration and reduces to 0.1 to 0.34 mcg/ml in approximately 4 hours.

Liver is the primary site of metabolism of Bupivacaine.

Bupivacaine crosses the placental barrier in very little concentrations with feto-maternal circulation ratios ranging from 0.2 to 0.4. It is also secreted in breast milk.

HYPERBARIC BUPIVACAINE

Isobaric Bupivacaine (Plain) has density equal to that of CSF. Hyperbaric Bupivacaine is prepared by the addition of Glucose (80 mg/ml) to the Isobaric Bupivacaine, with a density heavier than CSF. The difference in densities between the two is believed to affect their diffusion patterns and thus determine the spread, effectiveness and side-effect of the drugs.

Baricity is defined as the ratio of density (mass/volume) of the local anaesthetic's solution to that of the CSF at 37°C. It influences the distribution of local anaesthetic solution in the CSF, the spread and height of the block since gravity causes hyperbaric solutions to settle downward in the CSF and hypobaric solutions tend to rise. In contrast, gravity has no effect on the distribution of a truly isobaric solution.

DEXMEDETOMIDINE

Dexmedetomidine[3] is a highly selective alpha-2 adrenergic receptor agonist used primarily for sedation in intensive care settings, as well as for procedural sedation and as an adjunct in anaesthesia. Its development and usage offer insights into the evolution of sedative agents in medical practice.

Origin and Development

Dexmedetomidine is a synthetic derivative of medetomidine, a veterinary sedative. The key to dexmedetomidine's clinical effect is its high specificity for alpha-2 adrenoceptors compared to alpha-1 adrenoceptors. This selectivity is crucial for its action in reducing sympathetic nervous system activity while providing sedation and pain relief without significant respiratory depression.

Clinical Trials and FDA Approval

The drug underwent clinical trials in the 1990s. It was first approved by the U.S. Food and Drug Administration (FDA) in 1999 under the brand name Precedex. Initially, its use was limited to short-term sedation (less than 24 hours) in mechanically ventilated patients. However, over time, its clinical indications have expanded. By 2008, the FDA approved dexmedetomidine for sedation of non-intubated patients prior to and/or during surgical and other procedures, and later its use was extended to longer sedation durations.

Mechanism of Action

Dexmedetomidine works by binding to and activating alpha-2 adrenoceptors in the brain and spinal cord, which inhibits the release of norepinephrine, leading to sedation, hypnosis, and analgesia. Unlike other sedatives such as benzodiazepines or propofol, dexmedetomidine does not act on GABA receptors and therefore does not have the same level of respiratory depressant effects. This makes it an attractive option for sedating patients while allowing them to remain responsive and cooperative, a state sometimes referred to as "co-operative sedation."

Therapeutic Uses

Dexmedetomidine is used in various clinical settings:

ICU Sedation: It is commonly used for sedation in the intensive care unit (ICU), especially for patients who require a light level of sedation, where they can be easily awakened and cooperate with neurological examinations or ventilator weaning.

Procedural Sedation: It is used for sedation during non-invasive procedures such as mechanical ventilation, diagnostic imaging, and minor surgical procedures.

anaesthetic Adjunct: It is also used as a part of anaesthetic regimens in major surgeries to enhance sedation and provide analgesia, which can reduce the requirements for other anaesthetic drugs.

Recent Developments

Recent research has explored wider applications of dexmedetomidine, including its potential benefits in managing withdrawal symptoms, its use in paediatric medicine, and its possible neuroprotective effects.

In summary, dexmedetomidine represents a significant advancement in the pharmacological management of sedation and pain due to its efficacy and safety profile, particularly its minimal impact on respiratory function. Its development reflects ongoing efforts to improve patient outcomes and comfort in various medical settings.

is a potent alpha-2 adrenergic receptor agonist that has become an essential part of modern medical practice due to its unique pharmacologic profile. Here’s a detailed look at its mechanism of action, uses, and potential adverse effects:

Mechanism of Action

Dexmedetomidine exerts its effects primarily through stimulation of alpha-2 adrenoceptors, which are G-protein coupled receptors predominantly found in the brainstem. Activation of these receptors inhibits the release of norepinephrine, a neurotransmitter involved in the arousal and alertness pathways. This inhibition results in the sedative, anxiolytic, and analgesic effects of dexmedetomidine. The drug also has minimal effects on the respiratory centres in the brain, which distinguishes it from many other sedatives by preserving respiratory function.

1. Sedation: It induces sedation by reducing sympathetic nervous system activity and lowering the baseline level of arousal.
2. Analgesia: Provides pain relief through spinal cord actions and possibly by the modulation of pain pathways in the brain.
3. Anxiolysis: Reduces anxiety, which is helpful for patients undergoing stressful procedures.
4. Sympatholysis: Lowers blood pressure and heart rate by decreasing central sympathetic outflow.

Uses of Dexmedetomidine

Dexmedetomidine is versatile, with several clinical applications:

1. ICU Sedation: It is commonly used for sedation in intensive care units, especially for patients who need to be intubated and those who need to be responsive, such as during neurological evaluations or when adjustments in mechanical ventilation are necessary.
2. Procedural Sedation: It is effective for sedation during various procedures outside the operating room, including dental procedures, MRI scans, and other diagnostic or therapeutic interventions where deep anaesthesia is not required but sedation is beneficial.
3. anaesthetic Adjunct: In operative settings, dexmedetomidine is used to enhance the quality of anaesthesia, reduce the requirement for volatile anaesthetics and opioids, and facilitate smoother induction and recovery from anaesthesia.
4. Treatment of Withdrawal Symptoms: Its properties can mitigate symptoms associated with opioid and alcohol withdrawal.
5. Paediatric Use: Increasingly used in paediatric anaesthesia to provide sedation without severe respiratory depression.

Adverse Effects

While dexmedetomidine is generally well-tolerated, it can cause several side effects, particularly related to its cardiovascular effects:

1. Hypotension: As a sympatholytic, it can significantly reduce blood pressure, especially in volume-depleted patients or those concurrently receiving other vasodilators.
2. Bradycardia: The drug can slow the heart rate, which may be severe in some cases, particularly if patients are predisposed to bradycardia or are taking other medications that reduce heart rate.
3. Dry Mouth: Common with alpha-2 agonists due to reduced salivary secretion.
4. Transient Hypertension: Initial administration can lead to transient hypertension before the onset of central sympatholysis.
5. Nausea and Vomiting: Can occur, although less frequently than with other sedatives and anaesthetics.

In medical practice, dexmedetomidine's benefits are maximised when used judiciously, with careful monitoring of vital signs to manage its blood pressure and heart rate effects. Its ability to provide sedation and pain relief while allowing patients to remain responsive and breathe independently makes it a valuable tool in various clinical settings.

NEBULISATION

Nebulization is a method of administering medication directly into the lungs as a fine mist. It's commonly used for the treatment of respiratory diseases such as asthma, chronic obstructive pulmonary disease (COPD), and other respiratory infections. The process involves converting liquid medication into small aerosol droplets that can be inhaled through a mouthpiece or mask. Here's a detailed overview of the setup and process of nebulisation:

Equipment[5]

The primary equipment required for nebulisation includes:

1. **Nebulizer:** A device that converts liquid medication into a fine mist. There are several types of nebulizers:
   Jet Nebulizers: Use compressed air to generate aerosols. They are the most common type.

Ultrasonic Nebulizers: Use high-frequency sound vibrations to produce aerosols. They are quieter and faster, but they are not suitable for all types of medications.

Mesh Nebulizers: Use a fine mesh to create aerosols. These are portable, efficient, and fast.

2. **Compressor:** A small machine that provides the air flow needed for jet nebulizers to turn medication into an aerosol.

3. **Mouthpiece or Face Mask:** The mouthpiece is used by patients who can comfortably use it, as it usually leads to less medication waste. A face mask might be used for children or adults who find it difficult to use a mouthpiece.

4. **Medication:** Specific to the patient's condition, usually in liquid form.

5. **Tubing:** Connects the nebulizer to the air compressor.

Setup Process

1. Connect the Air Compressor: Place the compressor on a sturdy surface. Ensure it's plugged in but turned off before you start assembling the nebulizer.

2. Prepare the Medication: If the medication is stored in a refrigerator, allow it to reach room temperature. Measure the correct dose of medication, and if necessary, dilute it with saline as prescribed by the healthcare provider.

3. Assemble the Nebulizer: Open the medication cup, add the medication, and then close the cup. Attach one end of the tubing to the compressor and the other end to the nebulizer. Attach the mouthpiece or face mask to the nebulizer.

Nebulization Process

Once the nebulizer is set up, the patient can begin the treatment

1. Positioning: The patient should be sitting up straight in a comfortable, relaxed position. This posture helps maximise lung capacity and facilitate breathing.

2. Starting the Treatment: Turn on the compressor. The liquid medication will start to mist. The patient should breathe in and out normally through the mouthpiece, or breathe normally if using a mask. Encourage slow, deep breaths occasionally to ensure the medication is reaching deep into the lungs.

3. Duration: Treatment continues until all the medication is used, typically taking about 10-15 minutes. It’s important the patient continues to breathe the medication in until completion to ensure they receive the full dose.

4. Post-Treatment: After the treatment, it's important to clean the nebulizer components to prevent infections. Disassemble the nebulizer and wash the parts with warm, soapy water, then rinse and allow them to air dry.

<u>Additional Tips</u>

Ensure the nebulizer is cleaned and dried after each use.
Check the nebulizer and compressor for any signs of wear or damage regularly.
Always use the prescribed medication and follow the doctor's instructions regarding dosage and frequency.

Nebulization is an effective way to deliver medications directly to the lungs, making it a key treatment for various respiratory conditions. Proper setup, use, and maintenance of the nebulizer ensure effective treatment and minimise potential complications.

REVIEW OF LITERATURE

The 2013 systematic study by Faraj W. Abdallah et al.,[6] investigated the duration of sensory and motor blocks in patients receiving intravenous dexmedetomidine during spinal anaesthesia. Analysing data from 364 patients across seven randomised controlled trials, the study found that the sensory block duration is significantly augmented upon administration of intravenous dexmedetomidine by approximately 38%, motor block by about 21%, and delays the first analgesic requisition time by 60%. These findings suggest that dexmedetomidine enhances the efficacy of spinal anaesthesia, which could be beneficial in clinical settings. Dexmedetomidine was also linked to an (3.7-fold) increase in bradycardia, but no appreciable variations in BP or postoperative sedation was detected, and there were no reports of respiratory depression, indicating a favourable safety profile.

In a 2021 study by Ezhil Sekar et al,[7] the effect of administration of dexmedetomidine intravenously along with intrathecal hyperbaric bupivacaine for abdomen and lower limb procedures was observed. The randomised, double-blind trial included 212 adult patients divided into two groups. The test group were given infusions(loading dose of 1μg/kg and 0.5μg/kg maintenance), meanwhile for controls, saline administration was carried out. Results showed that dexmedetomidine markedly augmented the block times of both motor and sensory components without affecting the onset times in the group receiving dexmedetomidine, rescue analgesia requirement was delayed compared to control group, indicating prolonged analgesic effects. Hemodynamic stability was maintained, without significant side effects being observed, suggesting that intravenous dexmedetomidine could be beneficial for extending the effects of spinal anaesthesia with minimal adverse outcomes.

The study by Ola M. Zanaty et al.[8], conducted in 2015 investigated the effectiveness of nebulized ketamine, dexmedetomidine, and their combination as premedication in dental surgeries for paediatric outpatients. Sixty children aged between 3 to 6, were divided into three groups, receiving nebulized ketamine, dexmedetomidine, or a combination of both. The aim was to assess the sedation levels, ease of mask induction, hemodynamic stability, analgesia, and recovery behaviours among these groups. Results indicated that the combination group (DK) experienced significantly higher sedation levels and shorter recovery times compared to the groups receiving only one drug. Additionally, the

combination therapy led to smoother inductions of general anaesthesia and showed better postoperative analgesic effects without significant side effects. The study concluded that a nebulized combination of low-dose ketamine and dexmedetomidine provides a more effective and rapid premedication method for paediatric dental surgeries compared to using either drug alone, enhancing patient comfort and procedural efficiency.

In a randomised control trial by Satyajeet Misra and colleagues[9], the efficacy of preoperative dexmedetomidine nebulisation was examined to manage the hemodynamic stressor associated with laryngoscopy and intubation in adult patients. The study involved 120 patients who received either nebulized dexmedetomidine or saline before undergoing elective surgeries. It was found that dexmedetomidine significantly moderated the heart rate increase triggered by laryngoscopy compared to saline. Between two groups' systolic blood pressure fluctuations did not differ significantly from one another. Additionally, dexmedetomidine reduced the requirements for intraoperative anaesthetics and analgesics like propofol and fentanyl. In short-duration procedures, nebulized dexmedetomidine may be a good substitute for intravenous administration since there were no appreciable increase in post surgical vomiting or sore throat within the two groups.

In 2021, an RCT conducted by Ezhil Sekar et al[7], with the goal of comparing the effectiveness of the two methods as premedication for paediatric patients undergoing elective surgeries. In this randomised controlled experiment, 100 children between the ages of 2 and 6 were randomised to receive intravenous or nebulized dexmedetomidine. The outcomes showed that, in comparison to the intravenous method, nebulized dexmedetomidine produced more stable hemodynamics and sedative levels with less adverse effects such bradycardia and hypotension. Nebulized form is a better option for paediatric premedication in elective procedures since it also has greater patient and parent satisfaction with relation to overall experience and convenience of administration.

The study by N R R Kumar et al[11], conducted in 2020, examined the efficacy of dexmedetomidine nebulisation in reducing the intubation related hemodynamic stressor response. It inspected the effects of dexmedetomidine alongside a control group receiving saline in 100 patients who were divided randomly into two equal groups—one for control, the other for testing. Results indicated that

nebulized dexmedetomidine effectively reduced SBP,DBP and MAP immediately following laryngoscopy and intubation, without adverse effects like bradycardia. Additionally, dexmedetomidine showed a “dose-sparing effect” on propofol injected during induction, while reducing the need for other sedatives. This suggests nebulized dexmedetomidine as a viable premedication option to mitigate stress responses during intubation, improving patient outcomes in surgical settings.

The 2022 study by Vishwadeep Singh et al[12], evaluates the efficacy of intravenous versus nebulized dexmedetomidine in reducing the laryngoscopy and intubation induced stress response in 120 adult patients listed for elective surgeries. It specifically measures the hemodynamic response, the requirement for analgesics intra-operatively, and the incidence of postoperative sore throat. Findings reveal that nebulized dexmedetomidine is associated with better hemodynamic stability and fewer postoperative discomforts, such as sedation and sore throat, without increasing adverse effects. This suggests that nebulized dexmedetomidine could be a more suitable option for patients at risk of hypotension, bradycardia, or sedation, providing a comprehensive and safer approach.

In 2024, a randomised, double-blind research comparing intravenous and nebulized dexmedetomidine for blunting hemodynamic responses during laryngoscopy and intubation was carried out by Akshita Singla et al[13]. With 60 subjects divided into two groups, findings indicated that nebulized dexmedetomidine more effectively maintained intraoperative hemodynamics with fewer adverse effects compared to the intravenous route. The study suggests nebulized dexmedetomidine as a superior option for attenuating stress responses associated with laryngoscopy and tracheal intubation, offering a promising alternative for enhancing patient safety and comfort during surgical procedures.

Although there are several studies evaluating the effect of nebulised dexmedetomidine on stress response to laryngoscopy, intubation, its effect on characteristics of spinal anaesthesia has not been extensively studied, especially in Indian population. Studies have shown that IV Dexmedetomidine hastens the onset and prolongs the duration of spinal anaesthesia[14]. Nebulised dexmedetomidine is likely to show similar effects with fewer side effects. Hence, this study was undertaken.

MATERIALS AND METHODOLOGY

Study Design: Double Blind Randomised Clinical Trial

Source of Data: Data was acquired from patients who had undergone elective surgeries infraumbilically under Spinal Neuraxial Anaesthesia in Dr. Prabhakar Kore's Hospital and Medical Research Centre and KLE's Dr. Prabhakar Kore Charitable Hospital, Belagavi over a period spanning 1 year

Period of study: January 2023 to December 2023

Sample Size: Total sample size is 80

Sampling technique: The mean and standard deviation-based minimum sample size formula is

$$n = \frac{(z_\alpha + z_\beta)^2 (s_1^2 + s_2^2)}{(x_1 - x_2)}$$

where zβ correlates to the test's power and zα to its degree of significance. At a significance level of 5%, zα = 1.96 and zβ = 0.84 correspond to 80% test power.

Reference: "The effect of supplementation of low dose intravenous dexmedetomidine on characteristics of spinal anaesthesia with hyperbaric Bupivacaine- S.S. Harsoor, D Devika Rani et al." [14]

The motor block duration calculation takes into account the following parameter: The first group mean (259.11) is represented by x1, and the second group mean (231.16) is represented by x2.

The standard deviation of the first group is represented by s1 (53.1), whereas the standard deviation of the second group is represented by s2 (32.2).

After compilation of the values the obtained sample size turned out to be 39 in each group.

To round off the total sample size was increased to 40 in each group.

There were two groups, with 40 cases per group.

The total sample size was 80.

Inclusion Criteria:

- Either gender patients, and aged within 18 to 60 years undergoing Spinal Anaesthesia for elective infraumbilical surgery in supine position.
- Patients of Grade I to II of ASA.

Exclusion Criteria:

- ASA of Grade- III or more than III
- Patients with contraindication to Spinal anaesthesia
- Known allergy to Dexmedetomidine
- Pregnant females.
- Patients on pre-operative drugs that could possibly be potential confounders such as clonidine, gabapentin, pregabalin,steroids etc.

STUDY PROTOCOL:

After obtaining Ethical Committee approval, and applying inclusion and exclusion criteria, subjects undergoing infra-umbilical surgery in supine position under Spinal Neuraxial anaesthesia were included in the study.

Data collection procedure:

Randomization

Patients underwent randomisation and got allocated into one of the two study groups **Group I**(Patients that received Dexmedetomidine via nebulisation) and **Group II**(Patients that received Normal Saline via nebulisation) by a computer generated randomised number table.

After obtaining Institutional ethics committee clearance and CTRI registration, 80 patients were enrolled based on inclusion and exclusion criteria.

A day before the procedure, a pre-anaesthetic assessment was completed.

INVESTIGATIONS:

CBC

FBS

PT-INR

The above investigations were done in all patients.

ECG was done in patients aged over 40 years.

After taking informed consent, NBM status was confirmed on the day of surgery.

Pulse Oximeter, NIBP, ECG leads were attached and baseline reading was taken in the Preoperative room.

Patient was shifted into the OT. Another reading of pulse,NIBP and SpO_2 was then recorded.

Under strict asepsis, the patient then received Spinal anaesthesia in L3-L4 space using 3.2 ml of 0.5% Bupivacaine(Hyperbaric) having the patient in sitting position.

Patient was then made supine.

Group I patients were then administered dexmedetomidine nebulisation at the dose of 1 mcg/kg diluted with Normal Saline up to 5 ml.

Group II patients were nebulised with 5 ml of saline.

Intraoperative Pulse, SpO2, and NIBP was monitored at 5 minute intervals up to 30 minutes, then at 10 minute gaps upto 60 minutes and at 15 minute gaps until the surgery concluded.

Both the Motor and Sensory block onset was noted.

Using a cold swab touch, the sensory blockade's onset was observed.

Assessment of motor block was carried out with Bromage Scale[15] with the following score:-

0- No paralysis

1- Inability to flex hip, but ability to flex from knees to ankle

2- Inability to flex hips and knees, but has ability to flex ankles

3- Inability to flex ankles, only able to move toes.

4- Total paralysis

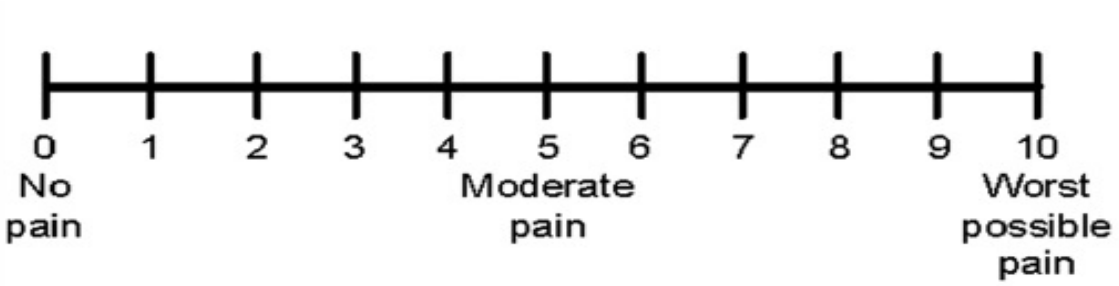
The duration of the sensory blockade was measured as the time it took for the block to recede by two segments[14]. A cold cotton swab was used to measure sensory blockade.

The total time of motor blockade recorded was the time required for bromage score 0 to return

Any intraoperative fall of HR over 15% of baseline was then promptly managed with Inj. Atropine 0.06mg IV while patient with fall in BP>20% from preoperative baseline was promptly given 3 mg of Mephentermine IV if required.

Intraoperatively, sedation was examined by employing the Ramsay Sedation Score(RSS) at 30 minute intervals till the duration of surgery or for 2 hours whichever was longer.

Post Operative area:



Visual analog scale (cms)

Pain was evaluated using the VAS Scale.

When VAS>3, the patient received Inj. Paracetamol 15mg/Kg BW. The time at which the patient asked for analgesics was noted and the cumulative analgesia in the initial 24 hours post surgery were also noted.

Ramsay Sedation Score

Ramsay Sedation Score (RSS) is a widely used scale for assessing patient's depth of sedation, particularly in critical care and perioperative settings. It provides a simple and effective way to measure the sedation depth. It was measured every half hour until the end of surgery.

RSS consists of six levels, each describing a specific state of patient sedation and responsiveness:

1. Score 1: Anxious or restless

2. Score 2: In a state of tranquil

3. Score 3: Only Responds to commands

4. Score 4: Loud vocal stimulus or light glabellar tap elicits brisk response

5. Score 5: loud vocal stimulus or light glabellar tap elicits sluggish response

6. Score 6: Not responsive to both tapping and vocal stimulus

RESULTS

The study focuses on the comparison of two groups. A randomised computer-generated table was utilised for the division of patients equally into two groups at random.

Data analysis was done by employing statistical software R version 4.4.0. and Microsoft Excel.Frequency and percentage were utilised to express categorical variables.The mathematical representation for all continuous variables was Mean ± SD. The QQ plot and “Shapiro-Wilk” test were used for analysing the normality of given values. The chi square test was employed to examine the degree of dependency among the categorical variables. Also, “Mann Whitney U” test was utilised to compare the distribution of various variables between groups. The variable distribution over time points was compared using the Friedman test. A P-value of 0.05 or less denotes statistical significance.

RESULTS:

The data contains measurements of 80 subjects, further grouped equally into **GROUP I** (test) and **GROUP II** (control) of 40 subjects each. The following table represents the variation in age across the groups .

Table 1: age over groups comparison.

Variable	Sub Category	Group I	Group II	p-value
Age (years)	Mean ± SD	41.8 ± 13.62	47.83 ± 13.47	0.0569

Group II's mean age is greater than Group I's, but the age distributions of the two groups do not differ significantly.

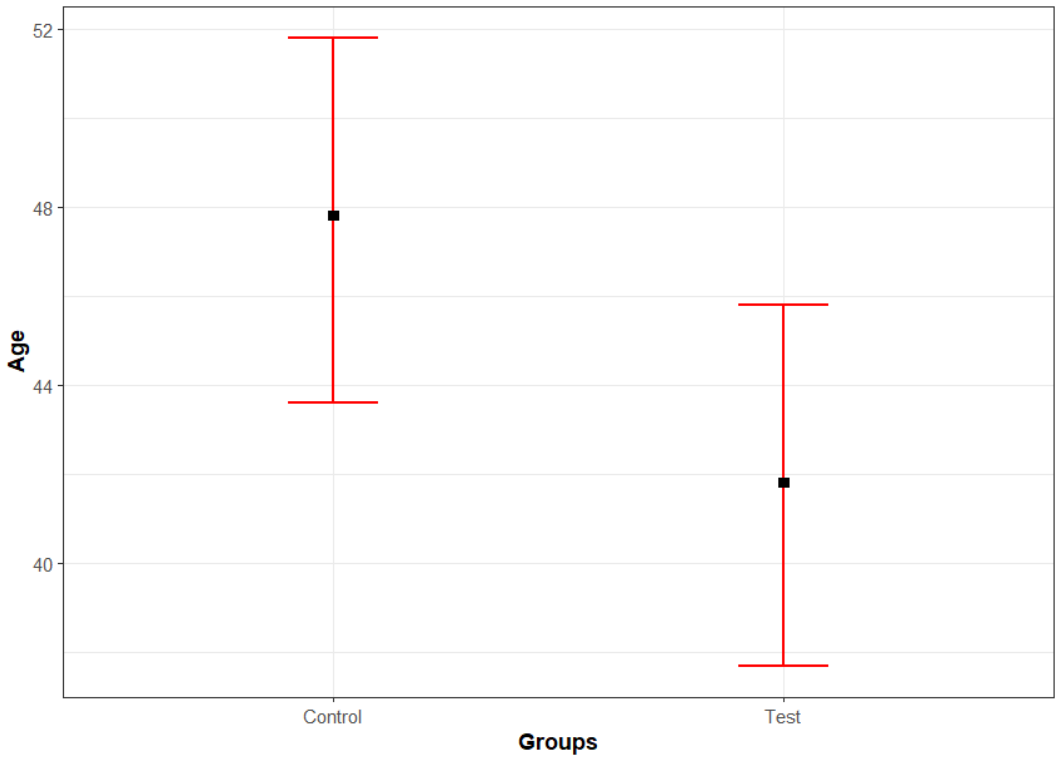


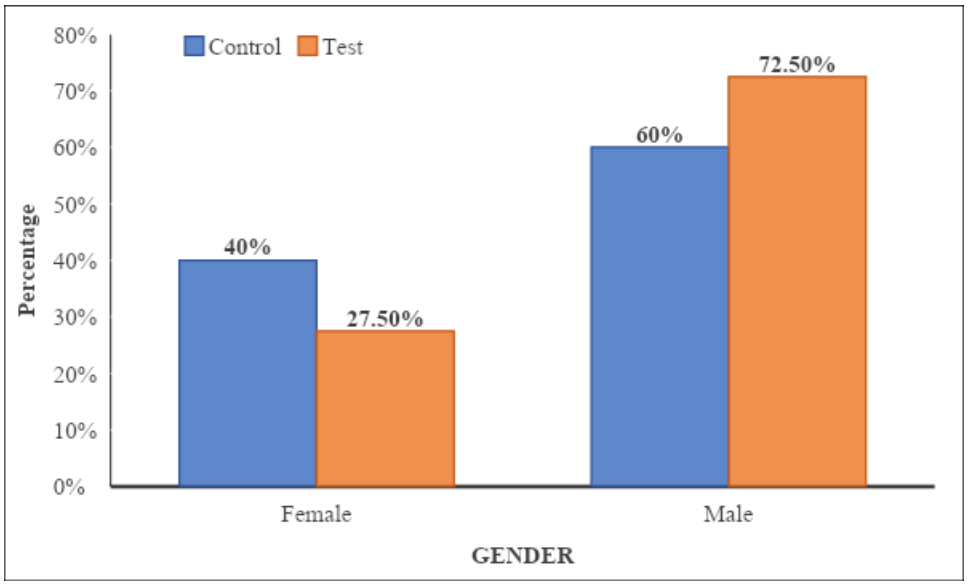
Figure 7: age over groups- Mean plot.

The table underneath gives a comparison of gender among groups.

Table 2: Comparison of gender over groups.

Gender	Group I	Group II	p-value
Female	11(27.5%)	16 (40%)	0.2371
Male	29 (72.5%)	24 (60%)	

Despite a slightly higher proportion of males among Group I compared to Group II, the Chi-square test indicates that it is of negligible significance in the gender distribution between both groups.



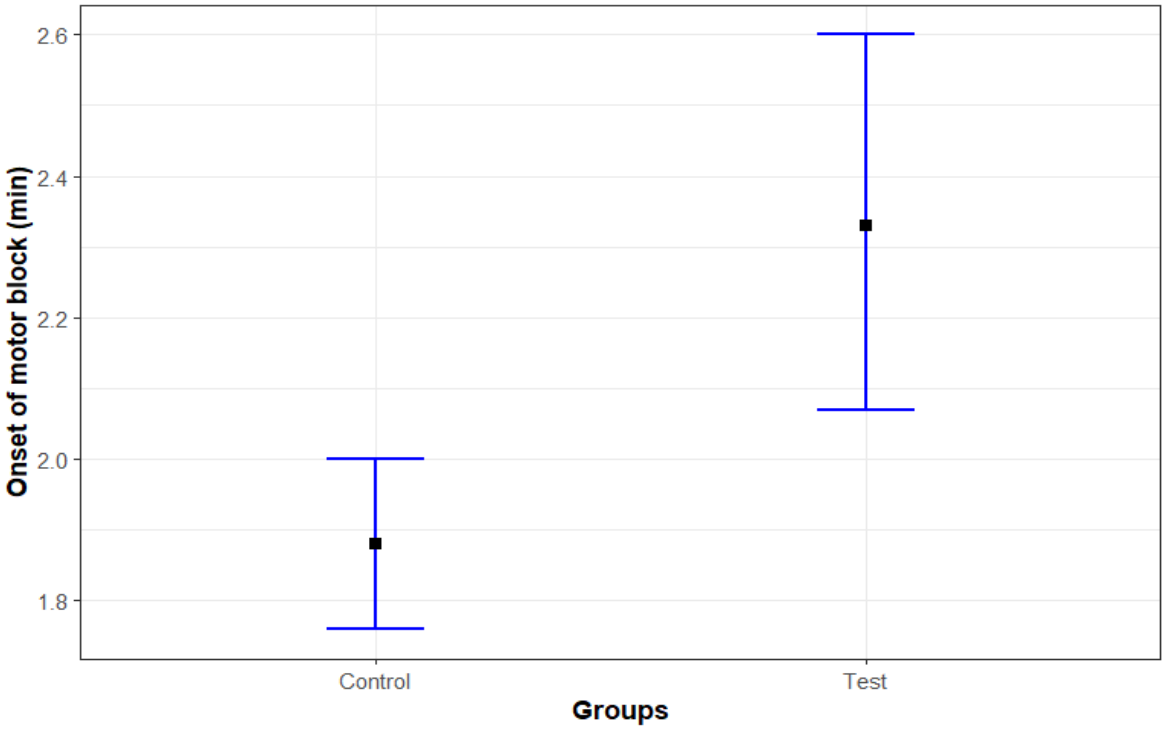
Graph 2: Distribution of gender over groups.

The following table gives an analysis of sensory and motor parameters over groups.

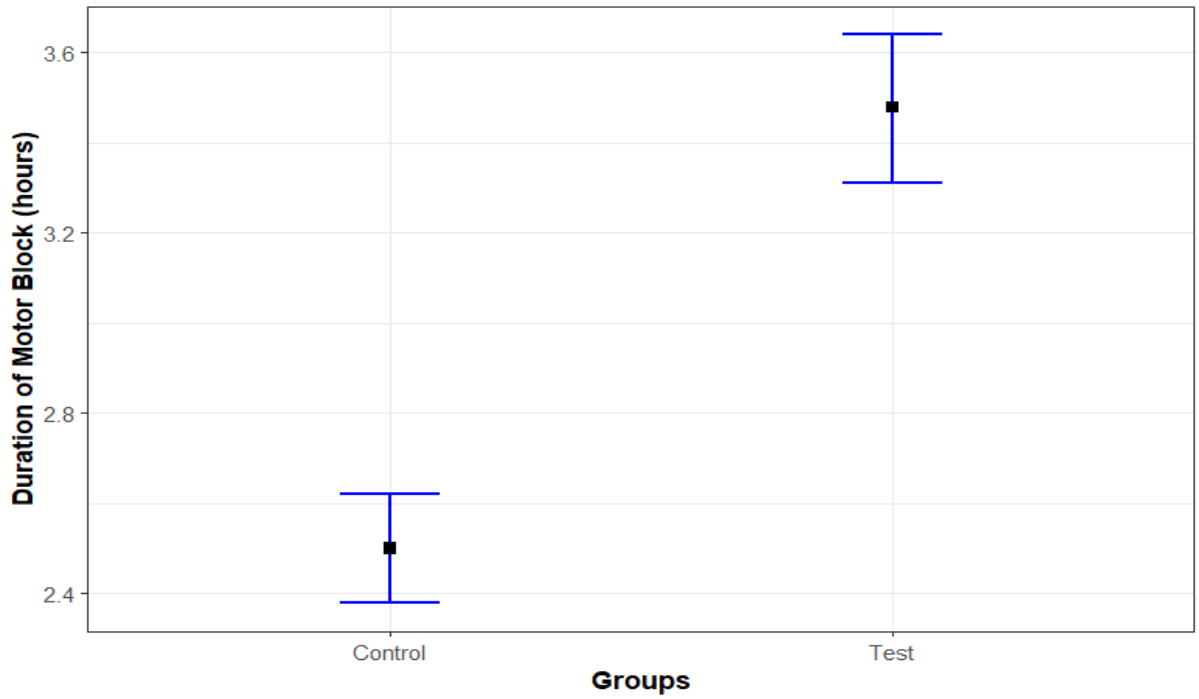
Table 3: Comparison of sensory and motor parameters over groups.

Variables	Category	Group I	Group II	p-value
Motor block Onset(min)	Mean ± SD	2.33 ± 0.89	1.88 ± 0.39	**0.0191**
Sensory block Onset(min)	Mean ± SD	2.03 ± 0.71	2.24 ± 1.32	0.4331
Duration of motor block (hours)	Mean ± SD	3.48 ± 0.53	2.5 ± 0.4	< **0.001**
Duration of sensory block (hours)	Mean ± SD	3.18 ± 0.5	2.79 ± 0.38	**0.0019**

The MW-U test reveals significant distinctions regarding the motor block onset and duration, and sensory block duration between the two groups. Specifically, Group I exhibits a more delayed motor block onset and higher durations of both motor and sensory blocks in comparison to the control group. Notably, there was negligible significance observed in the sensory block onset between both groups.

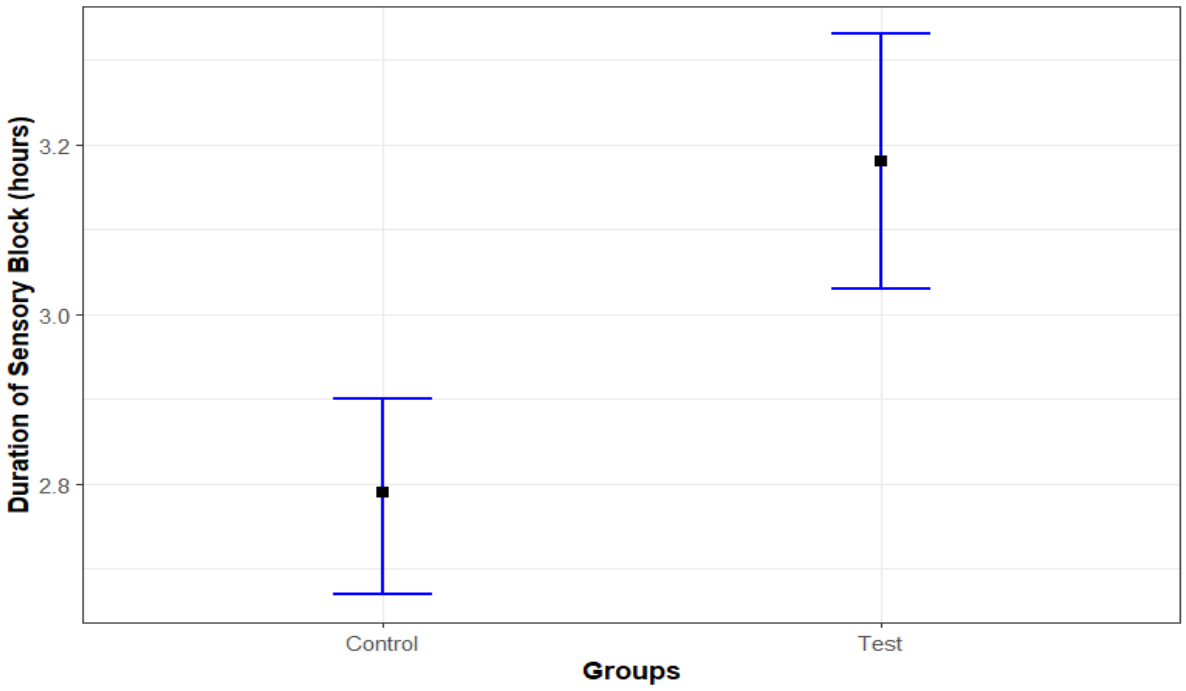


Graph 3: Mean plot comparing motor block onset over groups.



Graph -

4:Mean plot comparing motor block durations over groups.



Graph 5: Mean plot showing sensory block duration over groups.

The following table gives the comparison of Time for 1st post-operative rescue analgesia over groups.

Table 4: Comparison of 1st Rescue Analgesic Time over groups.

Variables	Sub Category	Group I	Group II	p-value
1st Rescue Analgesic time (hrs)	Mean ± SD	6.68 ± 2.2	6.18 ± 3.67	< **0.001**

Group II's first rescue analgesia time was less than Group I's. It gets established that this difference is statistically significant.

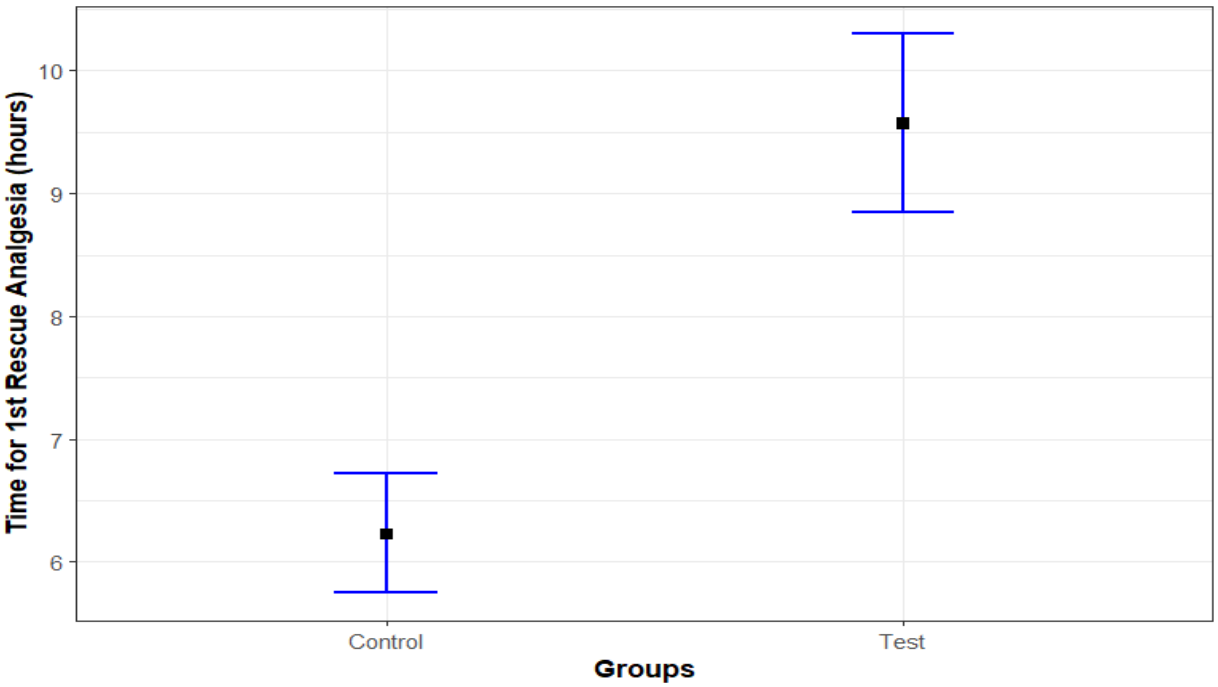


Figure 11: Mean plot of time for 1st post-operative rescue analgesia over groups.

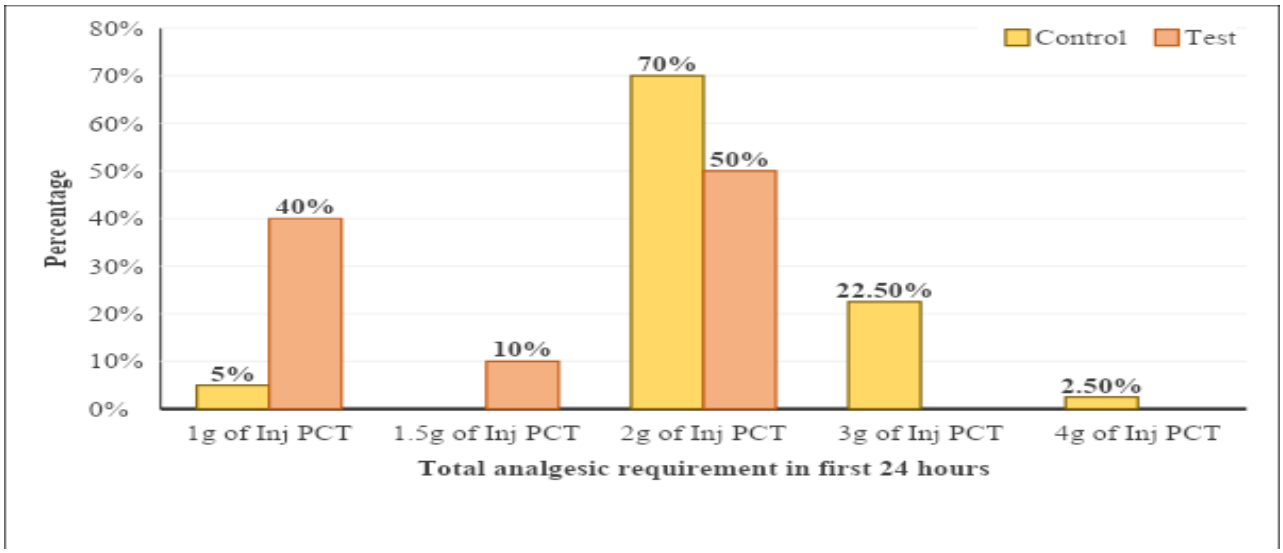
The following table compares Total analgesia required in the initial 24 hours post operatively over groups.

Table 5: Comparison of total analgesic requirements in initial 24 hours postoperatively over groups.

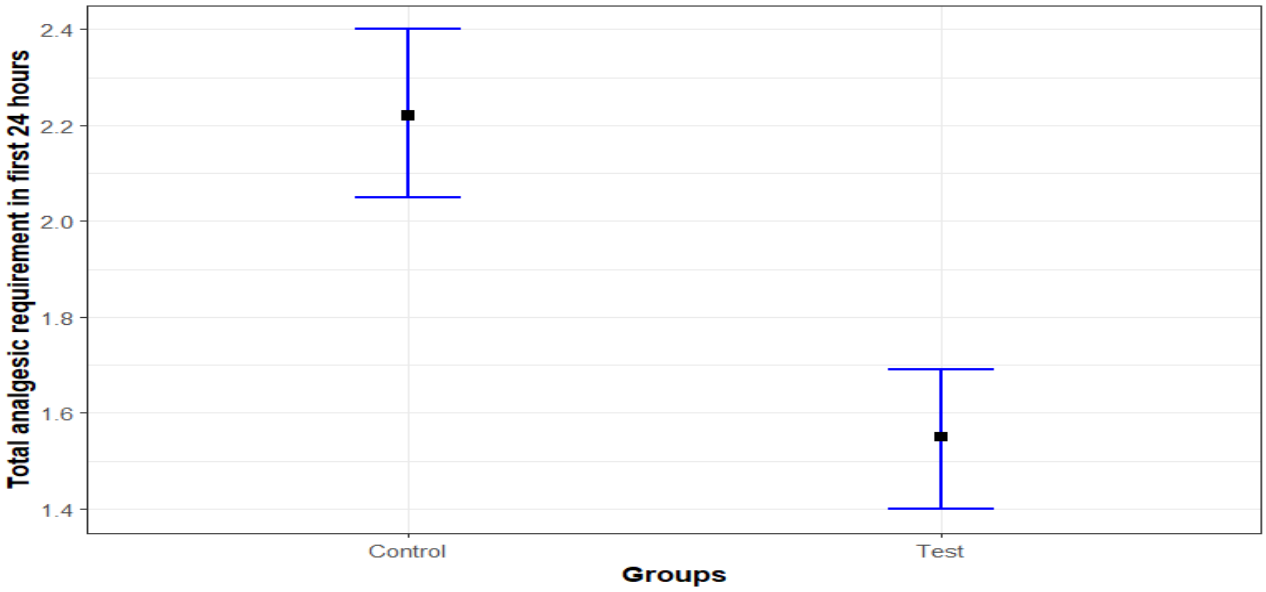
Variables	Sub Category	GROUP I	GROUP II	p-value
Analgesia required in first 24 hours	1g of Inj. PCT	16 (40%)	2 (5%)	< **0.001**
	1.5g of Inj. PCT	4 (10%)	0	
	2g of Inj. PCT	20 (50%)	28 (70%)	
	3g of Inj. PCT	0	9 (22.5%)	
	4g of Inj. PCT	0	1 (2.5%)	
	Mean ± SD	1.55 ± 0.48	2.22 ± 0.58	< **0.001**

In terms of total post-operative analgesic requirement within the first 24 hours, a notable difference was examined between both groups. In Group I, 40% of subjects required 1g of Inj. PCT, 10% required 1.5g, and the remaining 50% required 2g. On the other hand, in group II, only 5% required 1g of Inj. PCT, 70% required 2g, 22.5% required 3g, and 2.5% required 4g. This implies that Group I required less analgesics overall over the first 24 hours than Group II. Both the Chi-square test and

MW-U test confirmed significant differences in the distribution of total analgesic requirement over the groups.



Graph 6: Distribution of total analgesia for initial 24 hours postoperatively over groups.



Graph 7: Mean plot of total post-operative analgesia for the first 24 hours over groups.

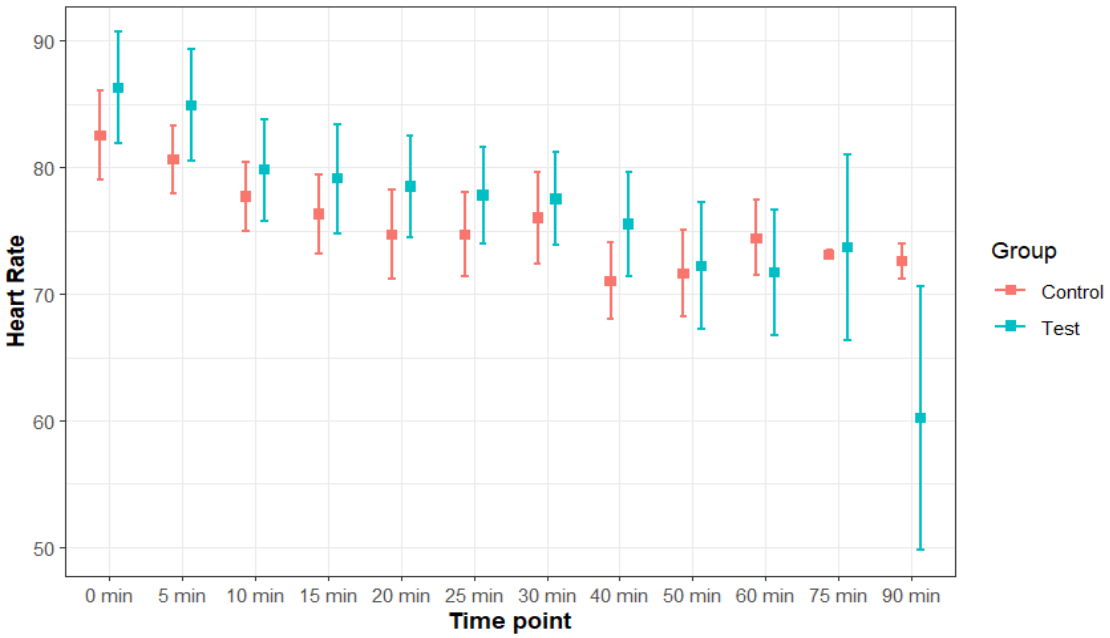
The following table compares Heart rate variation among time and both groups.

Table 6: Comparison of Heart rate among time and groups.

Time	Group I	Group II	p-value
0 min	86.28 ± 13.69	82.53 ± 11.05	0.2608
5 min	84.88 ± 13.72	80.62 ± 8.37	0.1252
10 min	79.8 ± 12.54	77.67 ± 8.47	0.3188
15 min	79.08 ± 13.4	76.33 ± 9.64	0.2127
20 min	78.47 ± 12.46	74.7 ± 10.92	0.1013
25 min	77.83 ± 11.94	74.7 ± 10.21	0.2611
30 min	77.54 ± 11.21	76.03 ± 11.31	0.7382
40 min	75.49 ± 11.9	71.04 ± 7.94	0.2300
50 min	72.21 ± 13.23	71.64 ± 8.92	0.6865
60 min	71.68 ± 12.91	74.45 ± 6.24	0.1625
75 min	73.67 ± 14.8	73.14 ± 0.38	0.6165
90 min	60.22 ± 13.52	72.57 ± 1.51	0.0503
120 min	85 ± 0	80	1
p-value	**< 0.001**	**< 0.001**	
End of Surgery	73.58 ± 15	75.25 ± 10.27	0.6634

As per the MW-U test,a difference of negligible significance was observed in the distribution of heart rate between the control and test groups at any specific time point.

However, the Friedman test indicates a difference of significance in the distribution of heart rate across various time points within both the control and test groups. This suggests that heart rate varies significantly over time within each group,irrespective of the group assignment.



Graph 8: Mean plot of heart rate over time and groups.

The underlying table compares spo2 over time and among the 2 groups.

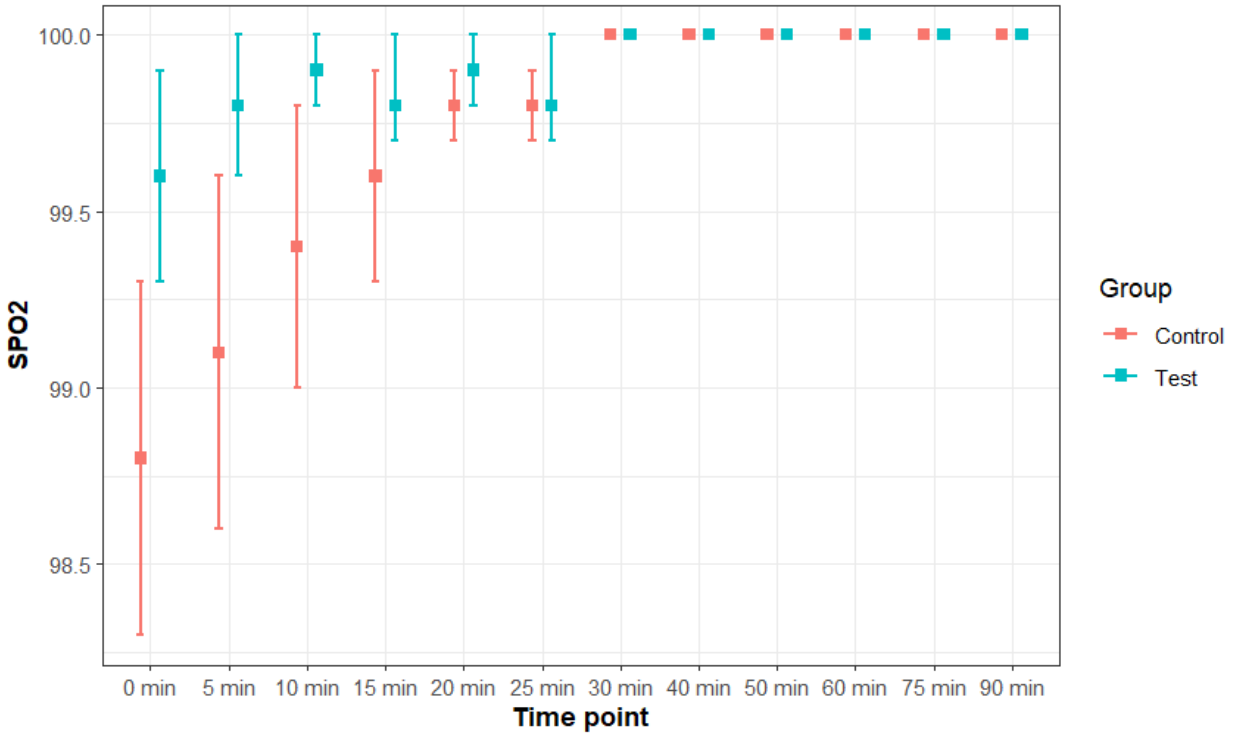
Table 7: Comparison of SPO2over time and groups.

Time	GROUP I	GROUP II	p-value
0 min	99.58 ± 0.96	98.8 ± 1.59	0.135
5 min	99.78 ± 0.58	99.12 ± 1.6	0.1074
10 min	99.92 ± 0.27	99.4 ± 1.22	0.0714
15 min	99.85 ± 0.53	99.6 ± 0.81	0.1067
20 min	99.92 ± 0.27	99.8 ± 0.41	0.1067
25 min	99.85 ± 0.53	99.8 ± 0.41	0.1566
30 min	100 ± 0	100 ± 0	1
40 min	100 ± 0	100 ± 0	1
50 min	100 ± 0	100 ± 0	1
60 min	100 ± 0	100 ± 0	1
75 min	100 ± 0	100 ± 0	1
90 min	100 ± 0	100 ± 0	1
120 min	100 ± 0	100	1

p-value	> 0.9999	> 0.9999	
End of Surgery	100 ± 0	100 ± 0	1

As per MW-U test, a difference of negligible significance was observed in the distribution of SPO2 among the 2 groups at any other time points.

Furthermore, according to the Friedman test, negligible significant changes were noted among the distribution of SPO2 over time points within both the control and test groups.



Graph 9: Mean plot of SPO2 over time and groups.

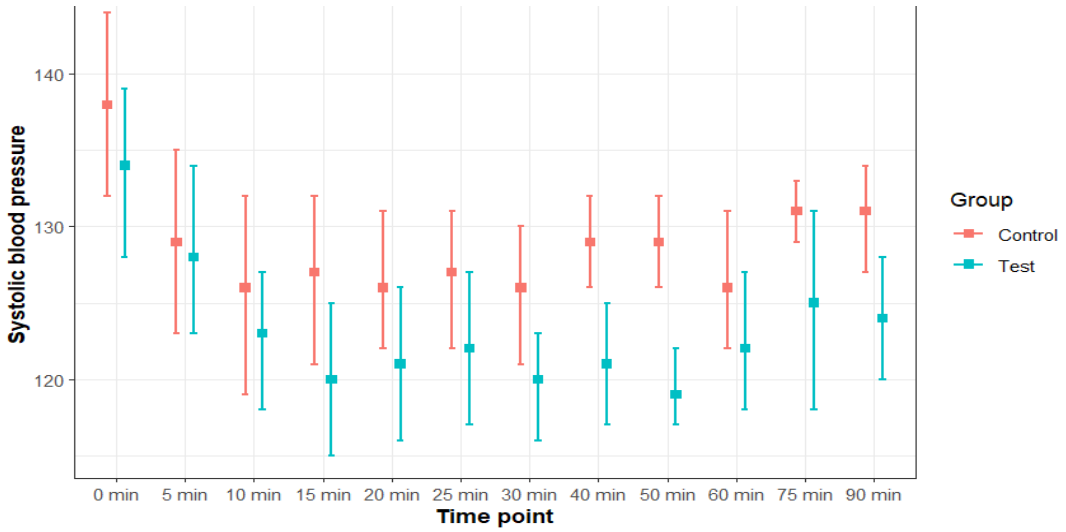
The following table gives the comparison of SBP over time and groups.

Time	GROUP I	GROUP II	p-value
0 min	133.88 ± 17.59	137.62 ± 18.48	0.6641
5 min	128.45 ± 16.88	129.15 ± 19.36	0.6139
10 min	122.58 ± 15.06	125.62 ± 19.38	0.0816
15 min	120.3 ± 16.14	126.7 ± 16.26	0.0592
20 min	121.17 ± 15.55	126.42 ± 13.5	0.0825
25 min	122 ± 15.6	126.55 ± 12.97	0.1136
30 min	119.69 ± 11.35	125.5 ± 12.84	0.0539
40 min	120.74 ± 11.4	129.39 ± 7.72	0.0501
50 min	119.45 ± 7.65	129.07 ± 6.68	**< 0.001***
60 min	122.25 ± 11.38	126.1 ± 9.78	0.1079
75 min	124.67 ± 12.58	131.14 ± 2.27	**0.0363**
90 min	124.22 ± 5.33	130.57 ± 3.78	**0.0062**
120 min	125 ± 0	125	1
p-value	**< 0.001**	**< 0.001**	
End of Surgery	123.28 ± 14.37	126.35 ± 13.06	0.1467

Table-8:Comparison of SBP over time and groups.

According to the MW-U test, significant changes to the distribution of SBP between Group I and Group II were observed at **50 minutes (p < 0.001)**, **75 minutes (p = 0.0363)**, and **90 minutes (p = 0.0062)**. Other significant changes in SBP distribution among the 2 groups were not found at other time points.

Additionally, the Friedman test revealed significant differences in the distribution of SBP over time points within both the control and test groups.



Graph 10: Mean plot of SBP over time and groups.

The following table gives the comparison of DBP over time and groups.

Table 9: A comparison of DBP over time and groups.

Time	GROUP II	GROUP I	p-value
0 min	87.9 ± 9.64	84 ± 10.52	0.0856
5 min	81.67 ± 9.78	81.58 ± 11	0.8657
10 min	79.5 ± 10.28	77.4 ± 9.48	0.1609
15 min	79.2 ± 6.91	77.5 ± 10.05	0.8505
20 min	79.28 ± 5.99	77.1 ± 8.89	0.4801
25 min	79.83 ± 5.7	77.55 ± 8.09	0.2620
30 min	78.17 ± 5.49	77.67 ± 8.74	0.8205
40 min	80.46 ± 5.47	78.69 ± 8.58	0.2126
50 min	81.82 ± 4.6	74.48 ± 7.66	**0.0002**
60 min	80.35 ± 4.83	77.36 ± 7.39	0.3350
75 min	81 ± 0	81.56 ± 5.12	**0.0366**
90 min	81.14 ± 0.38	81.78 ± 5.78	**0.0144**
120 min	84	87 ± 0	0.324
p-value	**< 0.001**	**< 0.001**	-
End of Surgery	80.25 ± 5.86	77.38 ± 8.29	0.1741

According to the MW-U test, significant differences with regards to the distribution of DBP between Group I and II were observed at **50 minutes (p = 0.0002)**, **75 minutes (p = 0.0366)** and **90 minutes (p = 0.0144)**. Significant differences in DBP distribution among the 2 groups were not found at other time points.

Additionally, the Friedman test revealed significant differences in the distribution of DBP over time points within both the control and test groups.

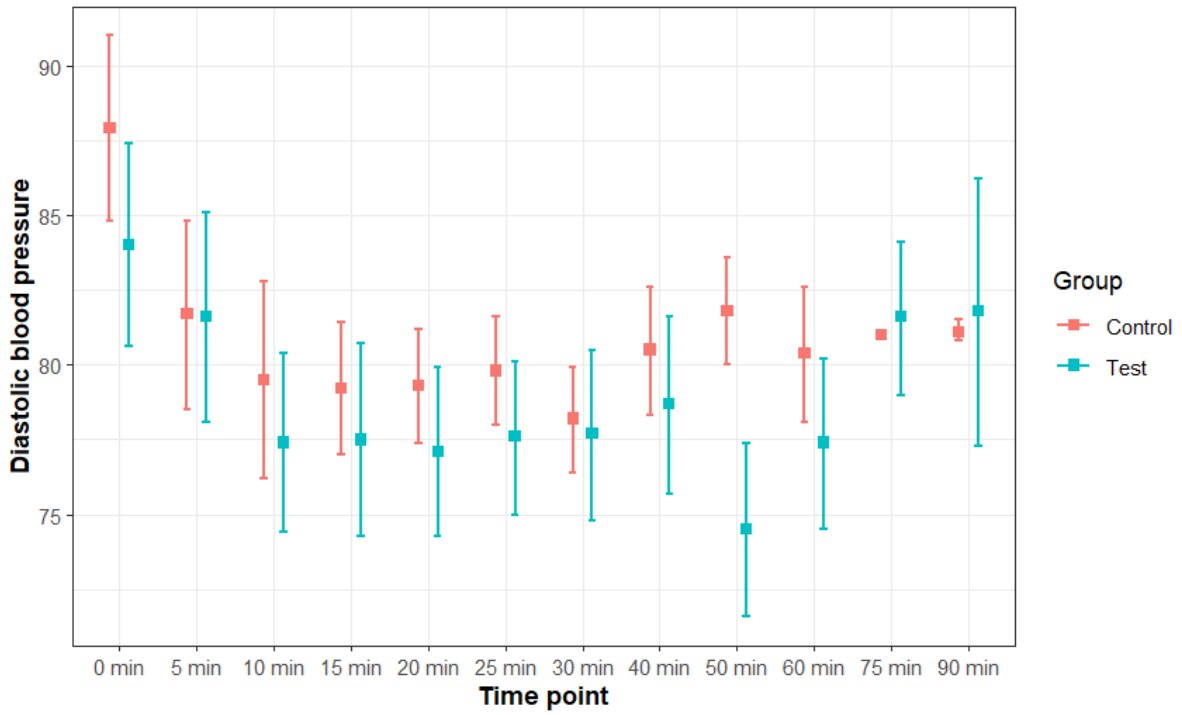


Figure 16: DBP over time and group by Mean plot.

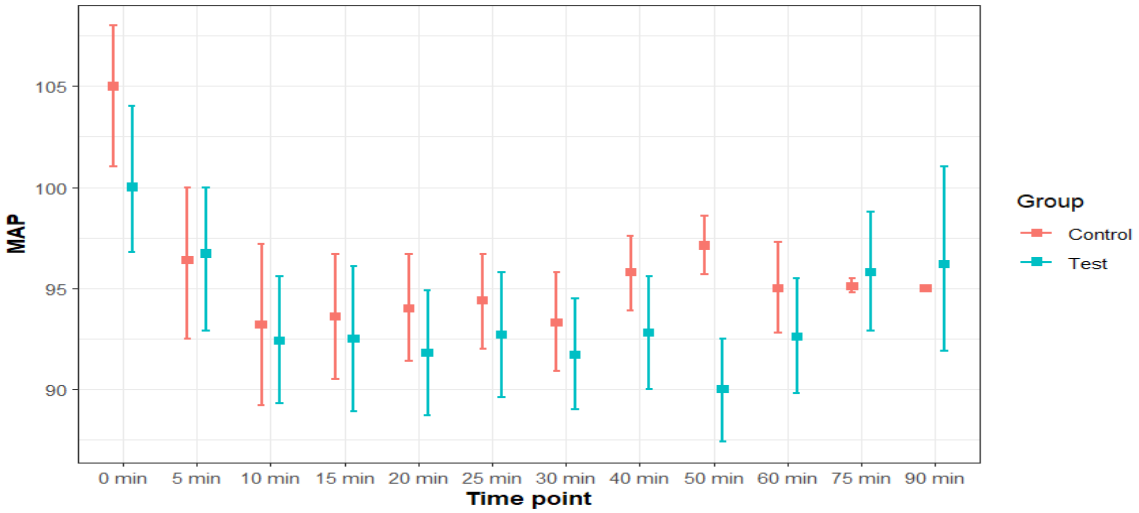
The underlying table compares MAP over time and groups.

Table 10: Comparing MAP variation across time and groups.

Time	GROUP II	GROUP I	p-value
0 min	104.55 ± 12.13	100.47 ± 11.44	0.1343
5 min	96.35 ± 12.17	96.7 ± 11.8	0.9576
10 min	93.17 ± 12.48	92.45 ± 9.81	0.5615
15 min	93.6 ± 9.85	92.53 ± 11.3	0.6125
20 min	94.05 ± 8.43	91.8 ± 9.78	0.4960
25 min	94.38 ± 7.31	92.67 ± 9.64	0.1811
30 min	93.33 ± 7.72	91.72 ± 8.51	0.2378
40 min	95.14 ± 0.38	95.83 ± 5.96	> 0.9999
50 min	97.14 ± 3.72	89.97 ± 6.64	**< 0.001**
60 min	95.05 ± 4.74	92.64 ± 7.31	0.8739
75 min	95.75 ± 4.68	92.77 ± 8.16	**0.0463**
90 min	95 ± 0	96.22 ± 5.61	**0.0053**
120 min	98	100 ± 0	0.863
p-value	**< 0.001**	**< 0.001**	
End of Surgery	95 ± 8.3	92.7 ± 8.87	0.6918

As per the MW-U test, stark differences among the distribution of MAP between Group I and Group II were observed at **50 minutes (p < 0.001)**, **75 minutes (p = 0.0463)** and **90 minutes (p = 0.0053)**.No differences of significance in MAP distribution between the groups were found at other time points.

Additionally, the Friedman test revealed significant differences in the distribution of MAP over time points within both the control and test groups.



Graph 12: Mean plot of MAP over time and groups.

The following table gives the comparison of RSS over time and groups.

Table 11: Comparison of RSS over time and among the 2 groups.

Time	GROUP II	GROUP I	p-value
0 min	1 ± 0	1 ± 0	> 0.9999
30 min	1.9 ± 0.3	2.08 ± 0.57	0.0945
60 min	2 ± 0	2.61 ± 0.5	**< 0.001**
90 min	2 ± 0	2.7 ± 0.48	**0.0082**
120 min	2	2.33 ± 0.58	**0.042**
p-value	0.062	**< 0.001**	-
End of Surgery	1.93 ± 0.27	2.42 ± 0.5	**< 0.001**

According to the MW-U test, stark contrasts in the distribution of RSS between the Group I and Group II was observed at **60 minutes (p < 0.001)** and subsequent intervals. Specifically, RSS was increased in GROUP I in comparison to GROUP II at this time point. Also, there were significant changes among the distribution of RSS between the groups observed at subsequent time points. Additionally, the RSS at the end of surgery was markedly higher in the GROUP I compared to the Group II (**p < 0.001**).

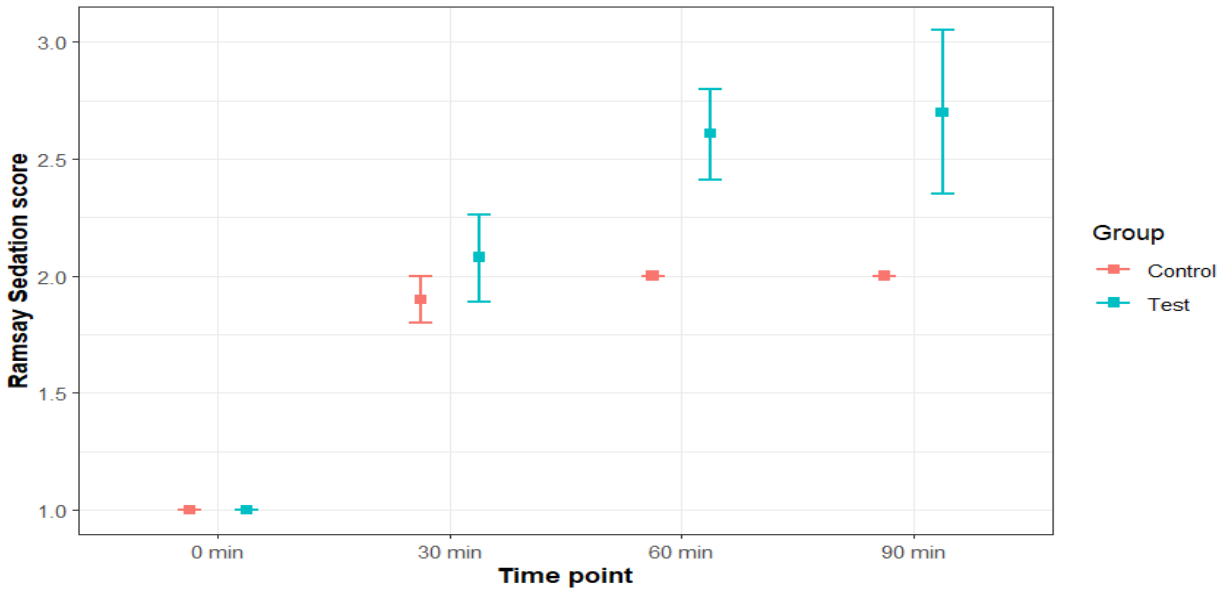


Figure 14: Mean plot of Ramsay sedation score over time and groups.

Some of the less common side effects of Dexmedetomidine include Dry Mouth, Dizziness,Respiratory Depression.

None of these side effects of dexmedetomidine was noted in the sample studied.

DISCUSSION

The study was done to compare nebulized dexmedetomidine's effects upon the action of spinal anaesthesia, specifically the onset and duration between the test and control groups, each comprising 40 subjects.

According to our study, when nebulized dexmedetomidine is given to patients undergoing infraumbilical procedures, it enhanced motor and sensory block time during spinal anesthesia. Demographically, age and gender distribution was comparable between both groups. The average age in Group I was 41.8±13.62 and group II being 47.83±13.47 respectively, having a p-value of 0.0569. The gender distribution of females amongst Group I and Group II was 11 and 16 respectively. Similarly, amongst males, 29 males were enrolled in Group I and 24 had enrolled in Group II with a p-value of 0.2371. This ensured that bias pertaining to demographics do not occur during the sample size collection.

There were significant changes noted mainly with regard to block duration, of both motor and sensory component. Specifically, the nebulised dexmedetomidine group exhibits a longer blockade of both motor and sensory blocks in comparison to the control group. nebulised dexmedetomidine group and Saline group had an average of 3.48±0.53 hrs, and 2.5±0.4 hrs in case of motor block duration respectively, with a **p-value** of **<0.001** and 3.18±0.5 hrs and 2.79±0.38 hrs respectively, for sensory block duration with a **p-value** of **0.0019**.

Regarding onset of sensory and motor block, onset of sensory block is slightly faster in Dexmedetomidine group compared to control group, value being 2.03±0.71 min in Group I and 2.24±1.32 min in Group II when compared however, this difference was found to be statistically insignificant. Onset of motor block was faster in the control group when compared to the Dexmedetomidine group, values being 1.88±0.39 min and 2.33±0.089 in Group II and Group I respectively.

When compared, this difference was found to be statistically significant with **p-value = 0.0191**.

This maybe probably due to the fact that the patient received dexmedetomidine nebulisation after spinal anaesthesia was administered.

The time to first rescue analgesic in the post operative period was also studied. The time for first rescue analgesia was delayed in Group I (6.68± 2.2 hrs) as against Group II where the patients requested analgesia much earlier(6.18± 3.67 hrs). When compared, this difference was found to be statistically significant **p- value <0.001**.

The total amount of rescue analgesia required in the first 24 hrs post operatively was also compared. Patients in Group I required 1.55±0.48 g of Inj. PCT while patients in Group II required 2.22± 0.58 g of Inj. PCT. When compared, this difference was found to be statistically significant with a **p value of <0.001** indicating reduced requirement of analgesia in Group I., indicating better pain management in the nebulised dexmedetomidine group as opposed to normal saline because of its analgesic property possibly by the modulation of pain pathways in the brain[].

In Group I, 16 patients requested for 1g of Inj. PCT, 4 subjects requested 1.5g of Inj. PCT and 20 subjects required 2g of Inj PCT. On the other hand, in Group II, 2 patients asked for 1g of Inj. PCT, 28 subjects requesting 2g of Inj. PCT, 9 patients wanting 3g of Inj. PCT and 1 subject requiring 4g of Inj. PCT., with a **p-value of <0.001**. This indicates that Dexmedetomidine provides ample analgesia and reduces the need for excessive analgesics post-operatively.

Regarding physiological parameters like heart rate and oxygen saturation, no significant differences were observed at all given time points(p-value 0.6634 and 1 respectively) or over time within each group.

Blood pressure measurements revealed significant differences at specific time points. Group I had lower SBP and lower DBP after 50 minutes (**p-value <0.001** and **0.002** respectively), 75 minutes(**p-value of 0.0363** and **0.0366** respectively) and 90 minutes(**p-value of 0.0062** and **0.0144** respectively) after nebulisation. Both SBP and DBP showed significant variations over time within each group (**p-value <0.001**). MAP values were markedly reduced in Group I at 50 minutes (**p-value <0.001**), 75 minutes (**p-value =0.0463**) and 90 minutes (**p-value=0.0053**) with significant variations over time in both groups(**p-value=<0.001**). Although the observed drop in BP was significant statistically, it was not clinically significant enough to require any intervention.

We also studied the effect of dexmedetomidine nebulisation on intra operative patient sedation. Patients receiving dexmedetomidine nebulisation showed higher RSS compared to patients receiving Saline nebulisation. When compared, this difference was found to be statistically significant at 60 min, 90 min, 120 min, and at the end of surgery, **p value** being **<0.001**, **0.0082**, and **0.042**, **<0.001** respectively. Within the group, patients in the dexmedetomidine group showed higher scores towards the end of surgery probably due to the sedative effects of dexmedetomidine.

Overall, the results show that action of spinal anaesthesia can be effectively prolonged with nebulized dexmedetomidine., with adequate sedation without leading to apnoea and improves postoperative pain management, with a manageable impact on heart rate and blood pressure. This suggests that dexmedetomidine can be beneficial in clinical settings for enhancing the efficacy and comfort of spinal anaesthesia procedures especially when administered via nebulisation.

In 2018, a research conducted by A.Jayaprakash et al[16], the study found that intravenous dexmedetomidine enhanced spinal anaesthesia by increasing the sensory block level, extending the regression of two-segment sensory anaesthesia, and prolonged motor block duration. Sedation scores were higher, and first post-operative analgesic time was significantly delayed. Side effects included bradycardia and hypotension. These findings were similar to our study except that bradycardia was of negligible significance in our study.

In another trial by Nikhila R et al[17]., it was observed that intravenous dexmedetomidine significantly enhanced spinal anaesthesia by increasing the sensory block level, prolonging the duration, and extends motor block duration. Sedation scores were higher, and the first analgesic requirement time was significantly prolonged, indicating improved post-operative analgesia. However, limitations of the study included hypotension and bradycardia, which highlights the need for careful hemodynamic monitoring and the advantage of using nebulisation over intravenous method. Findings of this study had siimilarities to ours.

A trial conducted by Agrawal et al[15]., "compared the effects of dexmedetomidine and clonidine as adjuvants to hyperbaric bupivacaine in spinal anaesthesia for lower limb surgery". They came to the conclusion that dexmedetomidine was more effective than clonidine for prolongation of the sensory and motor blockade. But it was observed that both did not significantly alter the times of sensory and motor block onset, suggesting that their primary effect is on the duration of the blockade rather than the onset, similar to our study findings.

Limitations

Smaller sample size reduces the power of study conducted.

It was conducted upon ASA I and II patients. The results may vary among ASA III and above patients.

Since the patients were given nebulisation after administration of sub-arachnoid block, there was no hastening of the onsets of sensory or motor block.

It was a single centre study, thus limiting its generalisability.

Future scope:

Further study maybe done by nebulisation before administration of spinal anaesthesia to see its effect mainly on motor and sensory block onset.

Also, smaller dose of 0.5mcg/kg BW can be evaluated for its effects on onset and duration of Sub-arachnoid anaesthesia.

CONCLUSION

This study, which was done to analyse the effects of nebulised dexmedetomidine on the onset and duration of spinal anaesthesia, shows that nebulised dexmedetomidine is a safe and reliable adjuvant to supplement sub-arachnoid block.

Nebulised dexmedetomidine prolongs the duration of both sensory and motor block. The onset of sensory and motor block was, however, not hastened.
Nebulised dexmedetomidine also prolongs the time taken to 1st rescue analgesic dose as well as reduces the requirement of analgesics in the first 24 hours post operatively.
It however, produces significant sedation. No other side effects were observed.

SUMMARY

The study aims to investigate the effect of nebulized dexmedetomidine on the onset and duration of spinal anaesthesia in patients undergoing infra-umbilical surgery. It was Conducted as a randomised clinical trial with 80 patients. The study divided participants randomly into two equal groups; Group I and Group II. Under strict asepsis, the patient then received Spinal anaesthesia in L3-L4 space using 3.2 ml of 0.5% Bupivacaine(Hyperbaric) having the patient in sitting position.

The patients were then made supine. Group I were then administered dexmedetomidine nebulisation at the dose of 1 mcg/kg diluted with Normal Saline up to 5 ml. Group II patients were nebulised with 5 ml of saline. Results indicated that dexmedetomidine significantly prolonged both sensory and motor block durations, delaying the need for postoperative analgesia without notable side effects. These findings suggest that nebulized dexmedetomidine enhances the efficacy of spinal anaesthesia, providing extended pain relief and reducing the need for additional analgesics post-surgery. Further research is recommended to confirm these benefits across broader clinical settings.

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ANNEXURE-I

KAHERs JNMC
BELAGAVI
INFORMED CONSENT FORM

1. **Study of effect of nebulized Dexmedetomidine on the onset and duration of Spinal Anaesthesia-a one year hospital based Randomized Clinical Trial**

Objective: To study the effect of dexmedetomidine nebulisation on onset and duration of sensory and motor block in spinal anaesthesia

2. ***Introduction:*** *Mr. /Mrs.* _____ we are requesting you to enrol yourself in the study titled **"Study of effect of nebulized Dexmedetomidine on the onset and duration of Spinal Anaesthesia-a one year, hospital based, Randomised Clinical Trial** conducted by Dr.Rishabh G. Mankale Post Graduate in M.D. Anaesthesiology under the guidance of Dr.Vandana A. Gogate, Professor, Department of Anaesthesiology, J.N. Medical College, Belagavi under KAHER, Belagavi.

Explanation of procedure: If you agree to enrol in my study, I will ask you about the present and past medical history. Then you will be clinically examined in detail. On the day of surgery, you will receive nebulization in the pre-operative room

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study. **Authorization for publication of aggregated data:** Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups.

However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: Dr Harsha Hegde, Chairperson, Institutional Ethical Committee of JNMC, and Scientist D, ICMR

National Institute of Traditional Medicine Belagavi 94804225001

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **of the effect of Dexmedetomidine nebulisation on onset and duration of Spinal Anaesthesia-a one year hospital based Randomized Clinical Trial**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II

PROFORMA

Title- Study of effect of nebulized Dexmedetomidine on onset and duration of Spinal Anaesthesia-a one year hospital based Randomized Clinical Trial.

Patient's Name- I.P. NO.-

Age- Date of Examination-

Gender- Anaesthesiologist-

Address-

Pre-Anaesthetic Evaluation:-

Chief Complaints:

History of Presenting Illness:

Past History:

Family History:

General Physical Examination:

Height - Weight- BMI-

Pallor- Icterus- Cyanosis-

Clubbing- Edema-

PR- RR- Sp02-

Systemic Examination:

CVS: **RS:**

CNS: **GIT:**

Airway assessment:

Teeth-
Jaw movements-
Airway-
Spine-

Investigations:

Physical Status: ASA Grade-

Diagnosis:

Proposed Surgery:

Patient's Name- **I.P. NO.-**

Age- **Date of Examination-**

Gender- **Anaesthesiologist-**

Drug/Saline administered:

TIME DURATION (IN MINUTES)	HEART RATE (bpm)	Sp02 (%)	SBP (mm Hg)	DBP (mm Hg)	MAP (mm Hg)
0 Minutes					
5 Minutes					
10 Minutes					
15 Minutes					
20 Minutes					
25 Minutes					
30 Minutes					
40 Minutes					
50 Minutes					
60 Minutes					
90 minutes					
120 minutes					
End of surgery					

Onset of sensory block-

Onset of motor block-

Time duration of Sensory Block -

Time duration of Motor Block-

Time required for the 1st rescue analgesia-

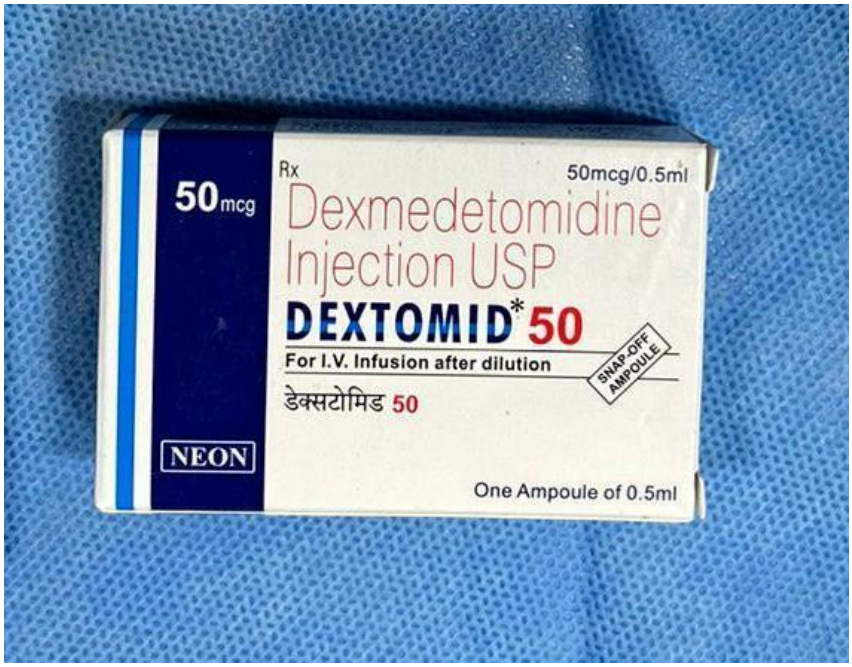
Total Analgesic requirement for the first 24 Hours:

Intra-operative assessment of sedation

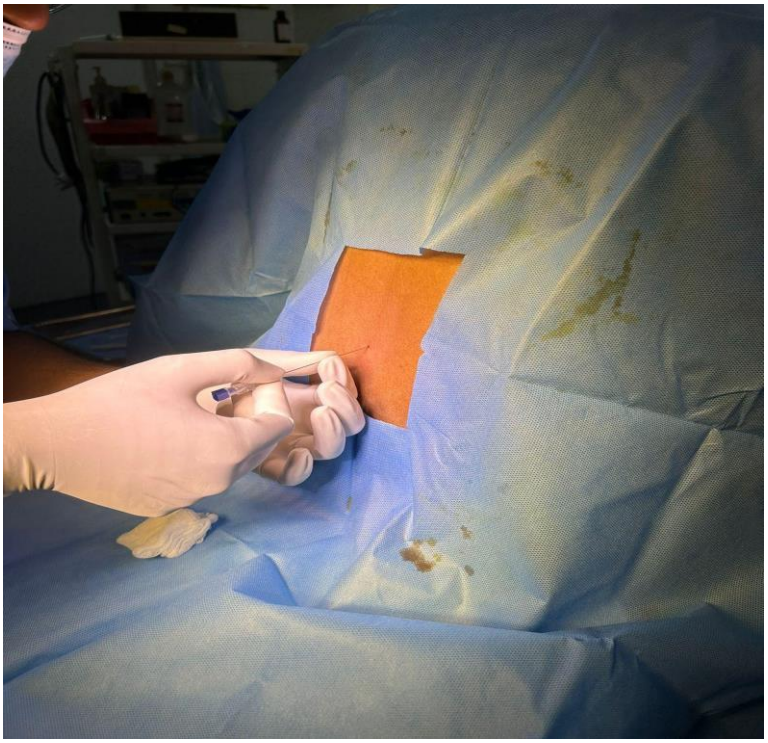
TIME (in minutes)	RAMSAY SEDATION SCORE
0 minutes	
30	
60	
90	
120	
150	
Till end of surgery	

ANNEXURE III: PHOTOGRAPHS

Photograph 1: Hyperbaric Bupivacaine 0.5%



Photograph 2: Dexmedetomidine drug



Photograph 3: Spinal anaesthesia

Photograph 4: Administering nebulisation

ANNEXURE IV: MASTER CHART

Sl. no.	Name	Age	Sex	HR(bpm)														
				0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx	
1	Ramesh Niralkur	30	M		76	75	73	71	68	65	62	61	60	61				61
2	Rajasab Diddi	32	M		93	89	89	93	86	83	83	85	86	86				86
3	Arjun Khira	18	M		102	100	98	96	95	98	95							95
4	Kajal V Kamble	29	F		84	82	81	78	76	78	81	83						83
5	Babu Pawar	60	M		106	110	94	95	94	93	93	93	94	94	99			99
6	Ravina	26	F		90	92	91	86	85	86	85							85
7	Balakrishna M P	52	M		76	78	70	73	69	62	68	64	61	63	64	53		53
8	Govinda	53	M		63	59	54	50	60	77	67	70	67	65				65
9	Bhimangoud B C	31	M		90	88	75	70	76	84	81	69	65	62	64			64
10	Satteppa Bhimaj	65	M		67	65	63	61	61	66								66
11	Mohan D Kitturk	45	M		103	97	94	90	97	92	84	82	78	78	75			75
12	Ramanna S. Pat	28	M		99	86	79	104	104	94	97	95	82					82
13	Yallappa	38	M		72	64	63	59	62	58	58	59	57	56	56	56		56
14	Shivanand	32	M		75	79	78	78	79	79	71	72						72
15	Sangappa M	41	M		111	103	101	96	94	87	87	86	85	81	74	84	85	85
16	Shivayogi Harog	34	M		76	75	73	71	68	65	62	61	60	61				61
17	Shakuntala K. K	45	F		76	75	73	71	68	65	62	61	60	61				61
18	Bahubali	52	M		93	89	89	93	86	83	83	85	86	86				86
19	Vishnu	45	M		106	110	94	95	94	93	93	93	94	94	99			99
20	Vivek M Kacheri	18	M		76	78	70	73	69	62	68	64	61	63	64	53		53
21	Mallappa S. Mirji	25	M		84	82	81	78	76	78	81	83						83
22	Toufiq	18	M		90	92	91	86	85	86	85							85
23	Gangaram P	60	M		76	78	70	73	69	62	68	64	61	63	64	53		53
24	Mahesh Kaderi	55	M		63	59	54	50	60	77	67	70	67	65				65
25	Gangaram H	60	M		90	88	75	70	76	84	81	69	65	62	64			64
26	Mangal Chandra	52	F		90	88	75	70	76	84	81	69	65	62	64			64
27	Murugappa Adiv	60	M		67	65	63	61	61	66	66							66
28	Suvarna	60	F		103	97	94	90	97	92	84	82	78	78	75			75
29	Rudrappa Vidya	18	M		111	103	101	96	94	87	87	86	85	81	74	84	85	85
30	Iqbal Ahmed Yad	55	M		76	75	73	71	68	65	62	61	60	61				61
31	Basappa Ramch	49	M		76	75	73	71	68	65	62	61	60	61				61
32	Manjula Shivapp	38	F		93	89	89	93	86	83	83	85	86	86				86
33	Shivanand Hiren	35	M		106	110	94	95	94	94	93	93	94	94	99			99
34	Padmavati	56	F		76	78	70	73	69	62	68	64	61	63	64	53		53
35	Kavita Ashok	48	F		84	82	81	78	76	78	81	83						83
36	Sagar Chandrak	32	M		90	92	91	86	85	86	85	85						85
37	Champavva	47	F		76	78	70	73	69	62	68	64	61	63	64	53		53
38	Shirin Bharumar	41	F		106	110	94	95	94	93	93	93	94	94	99			99
39	Annapurna Base	50	F		76	78	70	73	69	62	68	64	61	63	64	53		53
40	Mahesh Laxmari	39	M		84	82	81	78	76	78	81	83						83

	spo2 (%)															SBP(mm Hg)															DBP (mm Hg)													
	0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx		0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx		0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx
	97	98	99	100	99	98	100	100	100	100				100		123	116	111	107	108	109	114	121	123	130				130		72	71	70	70	69	70	71	72	72	72				72
	100	99	100	98	100	100	100	100	100	100				100		102	107	104	101	100	98	102	101	105	104				104		74	70	66	66	64	65	64	66	64	62				62
	100	100	100	100	100	100	100							100		120	116	112	114	118	114	110							110		70	70	64	66	67	66	65							65
	100	100	100	100	100	100	100	100						100		120	116	110	107	110	114	116	116						116		90	87	86	85	85	86	88	88						88
	98	100	100	100	100	100	100	100	100	100	100			100		149	134	132	135	140	136	129	139	128	142	146			146		81	75	74	83	78	79	77	76	75	80	81			81
	100	100	100	100	100	100	100							100		132	134	126	119	120	116	111							111		76	75	70	71	72	70	70							70
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	100	100	100	100	100	100	100	100	100	100				100		132	113	112	108	117	128	126	129	129	127				127		70	64	60	55	66	73	76	80	76	78				78
	100	100	100	100	100	100	100	100	100	100	100			100		146	135	126	129	133	146	133	132	122	121	121			121		102	98	88	87	89	90	93	95	86	87	85			85
	100	100	100	100	100	100								100		175	174	172	168	162	158								158		92	90	88	85	83	82								82
	100	100	100	100	100	100	100	100	100	100	100			100		122	122	122	122	120	119	115	113	112	111	112			112		87	85	85	85	85	85	83	82	80	80	80			80
	100	100	100	100	100	100	100	100	100					100		124	105	116	96	94	102	110	105	105					105		85	77	74	58	58	63	63	64	60					60
	100	100	100	100	100	100	100	100	100	100	100	100		100		113	112	111	111	112	111	111	110	109	108	107	110		110		72	72	72	72	72	71	70	70	68	67	67	68		68
	100	100	100	100	100	100	100	100						100		125	121	117	115	112	108	105	100						100		75	74	74	74	73	73	71	69						69
	100	100	100	100	100	100	100	100	100	100	100	100	100	100		139	138	138	137	138	139	136	133	123	119	126	126	125	125		98	95	96	94	91	86	86	86	62	85	91	88	87	87
	100	100	100	100	100	100	100	100	100	100				100		123	116	111	107	108	109	114	121	123	130				130		72	71	70	70	69	70	71	72	72	72				72
	97	98	99	100	99	98	100	100	100	100				100		123	116	111	107	108	109	114	121	123	130				130		72	71	70	70	69	70	71	72	72	72				72
	100	99	100	98	100	100	100	100	100	100				100		102	107	104	101	100	98	102	101	105	104				104		74	70	66	66	64	65	64	66	64	62				62
	98	100	100	100	100	100	100	100	100	100	100			100		149	134	132	135	140	136	129	139	128	142	146			146		81	75	74	83	78	79	77	76	75	80	81			81
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	100	100	100	100	100	100	100	100						100		120	116	110	107	110	114	116	116						116		90	87	86	85	85	86	88	88						88
	100	100	100	100	100	100	100							100		132	134	126	119	120	116	111							111		76	75	70	71	72	70	70							70
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	100	100	100	100	100	100	100	100	100	100				100		132	113	112	108	117	128	126	129	129	127				127		70	64	60	55	66	73	76	80	76	78				78
	100	100	100	100	100	100	100	100	100	100	100			100		146	135	126	129	133	146	133	132	122	121	121			121		102	98	88	87	89	90	93	95	86	87	85			85
	100	100	100	100	100	100	100	100	100	100	100			100		146	135	126	129	133	146	133	132	122	121	121			121		102	98	88	87	89	90	93	95	86	87	85			85
	100	100	100	100	100	100	100							100		175	174	172	168	162	158	158							158		92	90	88	85	83	82	82							82
	100	100	100	100	100	100	100	100	100	100	100			100		122	122	122	122	120	119	115	113	112	111	112			112		87	85	85	85	85	85	83	82	80	80	80			80
	100	100	100	100	100	100	100	100	100	100	100	100	100	100		139	138	138	137	138	139	136	133	123	119	126	126	125	125		98	95	96	94	91	86	86	86	62	85	91	88	87	87
	100	100	100	100	100	100	100	100	100	100				100		123	116	111	107	108	109	114	121	123	130				130		72	71	70	70	69	70	71	72	72	72				72
	97	98	99	100	99	98	100	100	100	100				100		123	116	111	107	108	109	114	121	123	130				130		72	71	70	70	69	70	71	72	72	72				72
	100	99	100	98	100	100	100	100	100	100				100		102	107	104	101	100	98	102	101	105	104				104		74	70	66	66	64	65	64	66	64	62				62
	98	100	100	100	100	100	100	100	100	100	100			100		149	134	132	135	140	136	129	139	128	142	146			146		81	75	74	83	78	79	77	76	75	80	81			81
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	100	100	100	100	100	100	100	100						100		120	116	110	107	110	114	116	116						116		90	87	86	85	85	86	88	88						88
	100	100	100	100	100	100	100	100	100	100	100	100		100		132	134	126	119	120	116	111	111						111		76	75	70	71	72	70	70	70						70
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	98	100	100	100	100	100	100	100	100	100	100			100		149	134	132	135	140	136	129	139	128	142	146			146		81	75	74	83	78	79	77	76	75	80	81			81
	100	100	100	100	100	100	100	100	100	100	100	100		100		151	147	128	126	123	122	122	121	119	118	119	126		126		94	95	82	83	82	82	82	81	81	81	80	82		82
	100	100	100	100	100	100	100	100						100		120	116	110	107	110	114	116	116						116		90	87	86	85	85	86	88	88						88

MAP (mm Hg)														Onset of Motor	Onset of senso	Duration of mo	Duration of sen	TIme for 1st res	Total requireme	Ramsay Sedation Score					
x 0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx							0 min	30 min	60 min	90 min	120 min	End of Sx
89	87	86	85	85	86	88	90	90	93				93	1 min 15 sec	1 min 25 sec	3 hrs 30 min	3 hrs	8 hrs	1g of Inj. PCT	1	1	2			2
83	82	79	78	76	76	77	78	78	76				76	1 min 35 sec	1 min 25 sec	4 hrs 20 min	4hrs 10 min	12 hrs	1g of Inj. PCT	1	3	3			3
87	86	79	82	84	82	80							80	2 min 10 sec	1 min 50 sec	4 hrs	3 hrs 30 min	8 hrs	2g of Inj PCT	1	2				2
100	97	94	92	90	95	97	97						97	2 min 20 sec	2 min 10 sec	2 hrs 30 min	2 hrs 30 min	9 hrs	2 g of Inj PCT	1	2				2
101	90	93	106	99	100	93	97	94	102	102			102	3 min 30 sec	3 min	3hrs 30 min	3 hrs 10 min	9 hrs	1g of Inj PCT	1	2	3			3
95	94	86	85	86	83	82							82	1 min 30 sec	2 min	3 hrs 20 min	3 hrs	16 hrs	1g of Inj. PCT	1	2				2
113	112	97	97	96	95	95	94	94	93	93	97		97	2 min 20 sec	2 min	4 hrs	3 hrs 30 min	7 hrs	2g of Inj. PCT	1	3				3
91	80	77	73	83	91	93	96	94	94				94	1 min 50 sec	1 min 15 sec	3 hrs 45 min	4 hrs	5 hrs	2g of Inj PCT	1	2	3	3	3	3
117	110	101	101	104	109	106	107	98	98	97			97	4 min	3 min	3 hrs 15 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2	2			2
120	118	116	113	109	107								107	1 min 30 sec	1 min 5 sec	2 hrs 45 min	2 hrs 30 min	10 hrs	1g of Inj. PCT	1	2				2
99	97	97	97	97	96	94	92	91	90	91			91	2 min 20 sec	1 min 50 sec	4 hrs	3 hrs	8 hrs	2g of Inj. PCT	1	1	2			2
98	86	88	71	70	76	79	78	75					75	2 min 30 sec	2 min	3 hrs 30 min	2hrs 50 min	9 hrs	1g of Inj. PCT	1	2	2			2
86	85	85	85	85	84	84	83	82	81	80	82		82	2 min 5 sec	1 min 45 sec	3 hrs 30 min	3 hrs	8 hrs	1.5g of Inj PCT	1	2	3	2		2
92	90	88	88	86	85	82	79						79	1 min 30 sec	1 min 15 sec	3 hrs 45 min	3 hrs 30 min	11 hrs	1.5g of Inj PCT	1	2				2
112	109	110	109	107	104	103	102	82	96	103	101	100	100	3 min 30 sec	3 min	3 hrs 15 min	2 hrs 55 min	6 hrs	2g of Inj PCT	1	2	3	2	2	2
89	87	86	85	85	86	88	90	90	93				93	3 min 20 sec	3 min	3 hrs 10 min	2 hrs 40 min	8 hrs	2g of Inj PCT	1	2	2			2
89	87	86	85	85	86	88	90	90	93				93	2 min 15 sec	2 min	3 hrs	2 hrs 40 min	6 hrs	2g of Inj PCT	1	1	2	3		3
83	82	79	78	76	76	77	78	78	76				76	1 min 20 sec	1 min 15 sec	4 hrs 5 min	3 hrs 45 min	12 hrs	1.5 g of Inj PCT	1	3	3			3
101	90	93	106	99	100	93	97	94	102	102			102	1 min 15 sec	1 min 25 sec	3 hrs 30 min	3 hrs	10 hrs	2g of Inj PCT	1	2	3			3
113	112	97	97	96	95	95	94	94	93	93	97		97	1 min 35 sec	1 min 25 sec	4 hrs 20 min	4hrs 10 min	12 hrs	1g of Inj. PCT	1	3	3			3
100	97	94	92	90	95	97	97						97	2 min 20 sec	2 min 10 sec	2 hrs 30 min	2 hrs 30 min	12 hrs	1 g of Inj PCT	1	2				2
95	94	86	85	86	83	82							82	2 min 30 sec	2 min	3 hrs 20 min	3 hrs	8 hrs	2g of Inj. PCT	1	2				2
113	112	97	97	96	95	95	94	94	93	93	97		97	2 min 20 sec	2 min	4 hrs	3 hrs 30 min	12 hrs	1g of Inj. PCT	1	3	3	3		3
91	80	77	73	83	91	93	96	94	94				94	1 min 50 sec	1 min 15 sec	3 hrs 45 min	4 hrs	7 hrs	2g of Inj PCT	1	2	3			3
117	110	101	101	104	109	106	107	98	98	97			97	4 min	3 min	3 hrs 15 min	3 hrs 5 min	12 hrs	1g of Inj PCT	1	2	2			2
117	110	101	101	104	109	106	107	98	98	97			97	4 min	3 min	3 hrs 15 min	3 hrs 5 min	10 hrs	2g of Inj PCT	1	2	2			2
120	118	116	113	109	107	107							107	1 min 30 sec	1 min 5 sec	2 hrs 45 min	2 hrs 30 min	8 hrs	2g of Inj PCT	1	2				2
99	97	97	97	97	96	94	92	91	90	91			91	2 min 20 sec	1 min 50 sec	4 hrs	3 hrs	10 hrs	2g of Inj. PCT	1	1	2			2
112	109	110	109	107	104	103	102	82	96	103	101	100	100	4 min 30 sec	4 min 10 sec	3 hrs	2 hrs 40 min	12 hrs	1g of Inj. PCT	1	2	3	2	2	2
89	87	86	85	85	86	88	90	90	93				93	1 min 20 sec	1 min 15 sec	4 hrs 5 min	3 hrs 45 min	12 hrs	1.5 g of Inj PCT	1	2	2			2
89	87	86	85	85	86	88	90	90	93				93	1 min 15 sec	1 min 25 sec	3 hrs 30 min	3 hrs	8 hrs	1g of Inj PCT	1	1	2	3		3
83	82	79	78	76	76	77	78	78	76				76	1 min 35 sec	1 min 25 sec	4 hrs 20 min	4hrs 10 min	12 hrs	2g of Inj. PCT	1	3	3			3
101	90	93	106	99	100	93	97	94	102	102			102	3 min 30 sec	3 min	3hrs 30 min	3 hrs 10 min	12 hrs	1g of Inj PCT	1	2	3			3
113	112	97	97	96	95	95	94	94	93	93	97		97	2 min 20 sec	2 min	4 hrs	3 hrs 30 min	9 hrs	2g of Inj. PCT	1	3	3	3		3
100	97	94	92	90	95	97	97						97	2 min 20 sec	2 min 10 sec	2 hrs 30 min	2 hrs 30 min	12 hrs	1 g of Inj PCT	1	2				2
95	94	86	85	86	83	82	82						82	1 min 30 sec	2 min	3 hrs 20 min	3 hrs	8 hrs	2g of Inj. PCT	1	2				2
113	112	97	97	96	95	95	94	94	93	93	97		97	2 min 20 sec	2 min	4 hrs	3 hrs 30 min	12 hrs	1g of Inj. PCT	1	3	3	3		3
101	90	93	106	99	100	93	97	94	102	102			102	3 min 30 sec	3 min	3hrs 30 min	3 hrs 10 min	8 hrs	2g of Inj PCT	1	2	3			3
113	112	97	97	96	95	95	94	94	93	93	97		97	2 min 20 sec	2 min 10 sec	4 hrs	3 hrs 30 min	7 hrs	2g of Inj. PCT	1	2	3	3		3
100	97	94	92	90	95	97	97						97	2 min 20 sec	2 min 10 sec	2 hrs 30 min	2 hrs 30 min	12 hrs	1 g of Inj PCT	1	2				2

Sl. no.	Name	Age	Sex	HR(bpm)													
				0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx
1	Shanta	65	F	85	95	88	84	82	80	78	76	74	70	74	76	80	80
2	Priyanka Rohan	25	F	76	75	73	71	68	65	62	61	60	61				61
3	Divakar D. Salar	48	M	78	76	72	71	65	64	63	62	61					61
4	Chetana	30	F	84	83	81	78	76	75	76							76
5	Kamalavva	51	F	70	68	66	61	60	62	64	68	72	74				74
6	Padmavati	43	F	82	80	78	78	81	80	85							85
7	Lalita Hanchimai	60	F	82	80	78	78	81	80	85							85
8	Nagavva Hirema	25	F	71	79	73	73	72	70	71	72	72	71				71
9	Shankarappa	60	M	102	92	91	86	85	88	92	84	86	84				84
10	Vishwanath V. H	29	M	76	74	72	70	68	72	73	74	75	72	73	72		72
11	Mallayya	67	M	78	76	72	71	65	64	63	62	61					61
12	Shivaji R Bedaka	65	M	104	98	94	101	101	96	95							95
13	Shivanand Hiren	43	M	78	76	72	71	65	64	63	62	61					61
14	Shakuntala	57	F	84	83	81	78	76	75	76							76
15	Shivaputrappa v	59	M	70	68	66	61	60	62	64	68	72	74				74
16	Naveen Patil	18	M	82	80	78	78	81	80	85							85
17	Raju Khot	45	M	82	80	78	78	81	80	85							85
18	Manjunath	32	M	71	79	73	73	72	70	71	72	72	71				71
19	Shabi N. Gawde	66	M	102	92	91	86	85	88	92	84	86	84				84
20	Padmavati	43	F	76	74	72	70	68	72	73	74	75	72	73	72		72
21	Annapurna Basa	50	F	78	76	72	71	65	64	63	62	61					61
22	Suvarna V	60	F	104	98	94	101	101	96	95							95
23	Shivarudra	34	M	84	83	81	78	76	75	76							76
24	Agoji I Bharmapp	32	M	70	68	66	61	60	62	64	68	72	74				74
25	Lalita K	50	F	82	80	78	78	81	80	85							85
26	yamanappa	60	M	82	80	78	78	81	80	85							85
27	Sugandha Patil	58	F	71	79	73	73	72	70	71	72	72	71				71
28	Anand M. devan	44	M	102	92	91	86	85	88	92	84	86	84				84
29	Mohammad Sali	60	M	76	74	72	70	68	72	73	74	75	72	73	72		72
30	Channappa T. C	62	M	78	76	72	71	65	64	63	62	61					61
31	Mamataz Nadaf	51	F	102	92	91	86	85	88	92	84	86	84				84
32	Shashidharayya	60	M	76	74	72	70	68	72	73	74	75	72	73	72		72
33	tulsabai Ranu	57	F	78	76	72	71	65	64	63	62	61					61
34	Sidappa M. gudl	33	M	104	98	94	101	101	96	95							95
35	Sunil Kamble	27	M	78	76	72	71	65	64	63	62	61					61
36	Iranna Gangapp	47	M	71	79	73	73	72	70	71	72	72	71				71
37	Arjun Gawde	38	M	102	92	91	86	85	88	92	84	86	84				84
38	Sunita N. Desai	57	F	76	74	72	70	68	72	73	74	75	72	73	72		72
39	Lakmappa Karig	47	M	78	76	72	71	65	64	63	62	61					61
40	Mallappa K	55	M	76	74	72	70	68	72	73	74	75	72	73	72		72

spo2 (%)															SBP(mm Hg)														DBP (mm Hg)													
0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx	0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	75 min	90 min	120 min	End of Sx	0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 mi	50 mir	60 mi	75 mi	90 mir	120 mi	End of Sx
100	100	100	100	100	100	100	100	100	100	100	100	100	100	124	98	101	104	110	111	106	104	114	120	126	122	125	125	86	66	68	68	74	78	76	78	78	80	81	82	84	84
100	100	100	100	100	100	100	100	100	100				100	123	116	111	107	108	109	114	121	123	130				130	72	71	70	70	69	70	71	72	72	72				72
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
99	100	100	100	100	100	100							100	130	126	121	116	120	118	120							120	80	78	75	76	77	78	80							80
100	99	100	100	100	100	100	100	100	100				100	140	114	116	122	128	131	136	134	131	136				136	90	78	80	82	84	85	84	86	88	88				88
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
100	100	100	100	100	100	100	100	100	100				100	143	142	142	147	139	137	128	124	117	111				111	96	93	92	90	89	89	81	87	86	84				84
100	100	100	100	100	100	100	100	100	100				100	110	100	92	116	120	117	115	122	128	122				122	71	63	59	73	76	72	71	71	75	75				75
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
95	100	100	100	100	100	100							100	188	178	166	167	165	164	163							163	93	93	90	89	89	88	87							87
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
99	100	100	100	100	100	100							100	130	126	121	116	120	118	120							120	80	78	75	76	77	78	80							80
100	99	100	100	100	100	100	100	100	100				100	140	114	116	122	128	131	136	134	131	136				136	90	78	80	82	84	85	84	86	88	88				88
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
100	100	100	100	100	100	100	100	100	100				100	143	142	142	147	139	137	128	124	117	111				111	96	93	92	90	89	89	81	87	86	84				84
100	100	100	100	100	100	100	100	100	100				100	110	100	92	116	120	117	115	122	128	122				122	71	63	59	73	76	72	71	71	75	75				75
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
95	100	100	100	100	100	100							100	188	178	166	167	165	164	163							163	93	93	90	89	89	88	87							87
99	100	100	100	100	100	100							100	130	126	121	116	120	118	120							120	80	78	75	76	77	78	80							80
100	99	100	100	100	100	100	100	100	100				100	140	114	116	122	128	131	136	134	131	136				136	90	78	80	82	84	85	84	86	88	88				88
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
99	100	100	100	100	100	100							100	120	116	112	106	108	110	109							109	80	78	74	70	71	74	70							70
100	100	100	100	100	100	100	100	100	100				100	143	142	142	147	139	137	128	124	117	111				111	96	93	92	90	89	89	81	87	86	84				84
100	100	100	100	100	100	100	100	100	100				100	110	100	92	116	120	117	115	122	128	122				122	71	63	59	73	76	72	71	71	75	75				75
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
100	100	100	100	100	100	100	100	100	100				100	110	100	92	116	120	117	115	122	128	122				122	71	63	59	73	76	72	71	71	75	75				75
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
95	100	100	100	100	100	100							100	148	138	136	137	135	134	133							133	93	93	90	89	89	88	87							87
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
100	100	100	100	100	100	100	100	100	100				100	143	142	142	147	139	137	128	124	117	111				111	96	93	92	90	89	89	81	87	86	84				84
100	100	100	100	100	100	100	100	100	100				100	110	100	92	116	120	117	115	122	128	122				122	71	63	59	73	76	72	71	71	75	75				75
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81
97	96	97	98	99	99	100	100	100					100	150	141	136	130	127	131	130	135	134					134	96	86	84	80	78	80	81	82	84					84
100	100	100	100	100	100	100	100	100	100	100	100		100	147	141	141	138	135	132	132	135	134	135	132	132		132	97	90	88	84	82	82	80	81	81	80	81	81		81

													MAP (mm Hg)														Onset of Motor	Onset of senso	Duration of mo	Duration of sen	TIme for 1st res	Total requireme	Ramsay Sedation Score					
in	15 mir	20 min	25 mir	30 mir	40 mi	50 mir	60 mi	75 mi	90 mi	120 mi	End of Sx	0 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 mir	75 mir	90 mir	120 min	End of Sx							0 min	30 min	60 min	90 min	120 min	End of Sx
68	68	74	78	76	78	78	80	81	82	84	84	98	76	79	80	88	92	89	92	93	94	96	95	98	98	2 min 45 sec	2 min 10 sec	2 hrs	3 hrs 30 min	6hrs	3g of Inj PCT	1	2	2	2	2	2
70	70	69	70	71	72	72	72				72	89	87	86	85	85	86	88	90	90	93				93	1 min 50 sec	1 min 45 sec	3hrs	3 hrs 20 min	4 hrs	2g of Inj PCT	1	2	2			2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	8 hrs	2g of Inj. PCT	1	2	2			2
75	76	77	78	80							80	97	94	89	88	90	90	93							93	1 min 30 sec	1 min	2 hrs 15 min	2 hrs 35 min	6 hrs	3g of Inj PCT	1	1				1
80	82	84	85	84	86	88	88				88	107	90	92	95	98	100	101	103	104	105				105	2 min	1 min 50 sec	1hr 45 min	2 hrs 10 min	5 hrs	2g of Inj PCT	1	2	2			2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	1 min 15 sec	1 min 30 sec	2 hrs	2 hrs 30 min	4 hrs	3g of Inj. PCT	1	2				2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	2min 10 sec	2min	2hrs 45 min	3 hrs 15 min	5 hrs	3g of Inj. PCT	1	2				2
92	90	89	89	81	87	86	84				84	112	109	109	107	106	1-5	97	99	96	92				92	1 min 30 sec	1 min 20 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	3g of Inj PCT	1	2	2			2
59	73	76	72	71	71	75	75				75	84	75	70	87	911	87	86	88	93	91				91	2 min 10 sec	1 min 40 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2				2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	6 hrs	4g of Inj PCT	1	2	2			2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	4 hrs	2g of Inj. PCT	1	2				2
90	89	89	88	87							87	125	121	115	115	114	113	112							112	2 min 10 sec	1 min 40 sec	2 hrs 45 min	2 hrs 30 min	10 hrs	1g of Inj PCT	1	2				2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	6 hrs	2g of Inj. PCT	1	2	2			2
75	76	77	78	80							80	97	94	89	88	90	90	93							93	1 min 30 sec	1 min	2 hrs 15 min	2 hrs 35 min	6 hrs	3g of Inj. PCT	1	1				1
80	82	84	85	84	86	88	88				88	107	90	92	95	98	100	101	103	104	105				105	2 min	1 min 50 sec	1hr 45 min	2 hrs 10 min	5 hrs	2g of Inj PCT	1	1	2			2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	1 min 15 sec	1 min 30 sec	2 hrs	2 hrs 30 min	8 hrs	2g of Inj. PCT	1	2				2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	2min 10 sec	2min	2hrs 45 min	3 hrs 15 min	7 hrs	2g of Inj. PCT	1	2				2
92	90	89	89	81	87	86	84				84	112	109	109	107	106	1-5	97	99	96	92				92	1 min 30 sec	1 min 20 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2				2
59	73	76	72	71	71	75	75				75	84	75	70	87	911	87	86	88	93	91				91	2 min 10 sec	1 min 40 sec	2 hrs 50 min	3 hrs 5 min	5 hrs	2g of Inj PCT	1	2				2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	4 hrs	2g of Inj PCT	1	2	2	2		2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	6 hrs	2g of Inj. PCT	1	2				2
90	89	89	88	87							87	125	121	115	115	114	113	112							112	2 min 10 sec	1 min 40 sec	2 hrs 45 min	2 hrs 30 min	5 hrs	3g of Inj PCT	1	2				2
75	76	77	78	80							80	97	94	89	88	90	90	93							93	1 min 30 sec	1 min	2 hrs 15 min	2 hrs 35 min	6 hrs	3g of Inj. PCT	1	1				1
80	82	84	85	84	86	88	88				88	107	90	92	95	98	100	101	103	104	105				105	2 min	1 min 50 sec	1hr 45 min	2 hrs 10 min	5 hrs	2g of Inj PCT	1	2	2			2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	1 min 15 sec	1 min 30 sec	2 hrs	2 hrs 30 min	6 hrs	2g of Inj. PCT	1	2				2
74	70	71	74	70							70	93	90	85	79	81	85	81							81	2min 10 sec	2min	2hrs 45 min	3 hrs 15 min	10 hrs	2g of Inj. PCT	1	2				2
92	90	89	89	81	87	86	84				84	112	109	109	107	106	105	97	99	96	92				92	1 min 30 sec	1 min 20 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2	2			2
59	73	76	72	71	71	75	75				75	84	75	70	87	91	87	86	88	93	91				91	2 min 10 sec	1 min 40 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2	2			2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	6 hrs	2g of Inj PCT	1	2	2	2		2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	4 hrs	2g of Inj. PCT	1	2				2
59	73	76	72	71	71	75	75				75	84	75	70	87	91	87	86	88	93	91				91	1 min 15 sec	1 min 25 sec	3 hrs 30 min	3 hrs	8 hrs	1g of Inj PCT	1	2				2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	6 hrs	2g of Inj PCT	1	2	2	2		2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	8 hrs	2g of Inj. PCT	1	2				2
90	89	89	88	87							87	111	108	105	105	104	102	100							112	2 min 10 sec	1 min 40 sec	2 hrs 45 min	2 hrs 30 min	10 hrs	2g of Inj PCT	1	2				2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	8 hrs	2g of Inj. PCT	1	2	2			2
92	90	89	89	81	87	86	84				84	112	109	109	107	106	105	97	99	96	92				92	1 min 30 sec	1 min 20 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2	2			2
59	73	76	72	71	71	75	75				75	84	75	70	87	91	87	86	88	93	91				91	2 min 10 sec	1 min 40 sec	2 hrs 50 min	3 hrs 5 min	6 hrs	2g of Inj PCT	1	2	2			2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	6 hrs	2g of Inj PCT	1	2	2	2		2
84	80	78	80	81	82	84					84	114	101	97	97	94	97	97	98	100					100	1 min 40 sec	48 sec	2 hrs 40 min	3 hrs	8 hrs	2g of Inj. PCT	1	2				2
88	84	82	82	80	81	81	80	81	81		81	114	105	102	97	96	96	95	95	96	96	95	95		95	2 min 20 sec	1 min 40 sec	2 hrs 10 min	2 hrs 20 min	6 hrs	3g of Inj PCT	1	2	2	2		2