A Dissertation submitted on

A COMPARATIVE STUDY OF DEXMEDETOMIDINE AND MAGNESIUM SULFATE AS ADJUVANTS WITH ROPIVACAINE FOR SPINAL ANAESTHESIA IN INFRAUMBILICAL SURGERIES AND POSTOPERATIVE ANALGESIA

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CERTIFICATE-I

This is to certify that this dissertation entitled **"A Comparative Study of Dexmedetomidine and Magnesium Sulfate as Adjuvants with Ropivacaine for Spinal Anaesthesia in Infraumbilical Surgeries and Postoperative Analgesia"** is a bonafide original work of **Dr.NIRMAL KUMAR.M** in partial fulfilment of the requirements for **Doctor of Medicine in Anaesthesiology-Branch X** examination of The Tamil Nadu DR. M.G.R. Medical University to be held in MAY- 2021.The period of study was from 2019 to 2020.

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This is to certify that this dissertation work **"A Comparative Study of Dexmedetomidine and Magnesium Sulfate as Adjuvants with Ropivacaine for Spinal Anaesthesia in Infraumbilical Surgeries and Postoperative Analgesia"** of the candidate **Dr.NIRMAL KUMAR M** with registration Number **201820205** for the award of **M.D.,** in the branch of **ANAESTHESIOLOGY X** personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows 13 percentage of plagiarism in the dissertation.

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DECLARATION

I, Dr. **NIRMAL KUMAR.M,** declare that this dissertation entitled **"A Comparative Study of Dexmedetomidine and Magnesium Sulfate as Adjuvants with Ropivacaine for Spinal Anaesthesia in Infraumbilical Surgeries and Postoperative Analgesia"** is a bonafide and genuine research work carried out by me in the Department of Anaesthesiology, Thanjavur Medical College Hospital, Thanjavur, during March 2019 to January 2020 under the guidance and supervision of Prof.Dr.C.Kumaran MD, Department of Anaesthesiology.

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CONTENTS

INTRODUCTION

Neuraxial anaesthesia is the most preferred technique for lower abdominal and lower limb surgeries. Spinal anaesthesia is considered superior to general anaesthesia. It minimizes or avoids the problem associated with general anaesthesia such as airway management, inhibits stress hormone release, decreases intraoperative blood loss, provides postoperative analgesia, and lowers the incidence of thromboembolic events.

Use of intrathecal adjuvants prolongs the duration of block, leads to a better success rate and patient satisfaction, and provides adequate pain management. A number of adjuvants have been studied to prolong the effect of spinal anaesthesia such as opioids (morphine, fentanyl, nalbuphine, buprenorphine), sodium bicarbonate, vasoconstrictors (epinephrine), N-methyl-d-aspartate antagonists (ketamine, magnesium sulfate), centrally acting α-2 adrenoceptor agonists (clonidine and dexmedetomidine), and γ-aminobutyric acid receptor agonists (midazolam). Thus, intrathecal additive is a reliable method to prolong the duration of spinal anaesthesia and prolong postoperative analgesia.

Dexmedetomidine is an agonist on the α2receptor found in the peripheral and central nervous system. Stimulation of the alpha receptors in the brain and spinal cord inhibits neuronal firing, causing hypotension, bradycardia, sedation, and analgesia. The analgesic action of the intrathecal α2-adrenoceptor agonist is depressing the release of C fiber transmitters and by hyperpolarization of

postsynaptic dorsal horn neurons. This antinociceptive effect may explain the prolongation of sensory block, but the prolongation of the motor block may result from the binding of α2 adrenoceptor agonists to motor neurons in the dorsal horn.

Magnesium sulfate blocks calcium influx and noncompetitively antagonizes N-methyl-d-aspartate receptor channels and prevents central sensitization from peripheral nociceptive stimulation, leading to analgesia. The analgesic action of intrathecal Mg+2 is primarily based on the regulation of calcium influx into the cell, that is natural physiological calcium antagonism.

With this background, this study was designed to compare the efficacy of intrathecal dexmedetomidine and magnesium sulfate ($MgSO_4$) for onset & duration of sensory & motor block, duration of analgesia, post operative pain & to evaluate the side effects, if any.

AIM

To evaluate and compare the efficacy of intrathecally administered dexmedetomidine and magnesium sulphate along with ropivacaine in patients undergoing infraumbilical surgeries.

OBJECTIVES

Comparative study of dexmedetomidine and magnesium sulphate as adjuvants with intrathecal ropivacaine in infraumbilical surgeries with respect to :

Time to onset of analgesia at T10.

Maximum sensory level achieved.

Time to achieve the maximum sensory level.

Mean time to regression to L1 dermatome.

Time to onset of motor block.

Maximum Bromage scale achieved.

Total duration of motor block.

Hemodynamic parameters.

SPINAL ANAESTHESIA

Spinal (subarachnoid/intrathecal) anaesthesia is a type of neuraxial anaesthesia in which local anaesthetic is injected into the cerebrospinal fluid to anesthetize nerves that exit the spinal cord. It is the most frequently employed method of administering regional anaesthesia.

ANATOMY

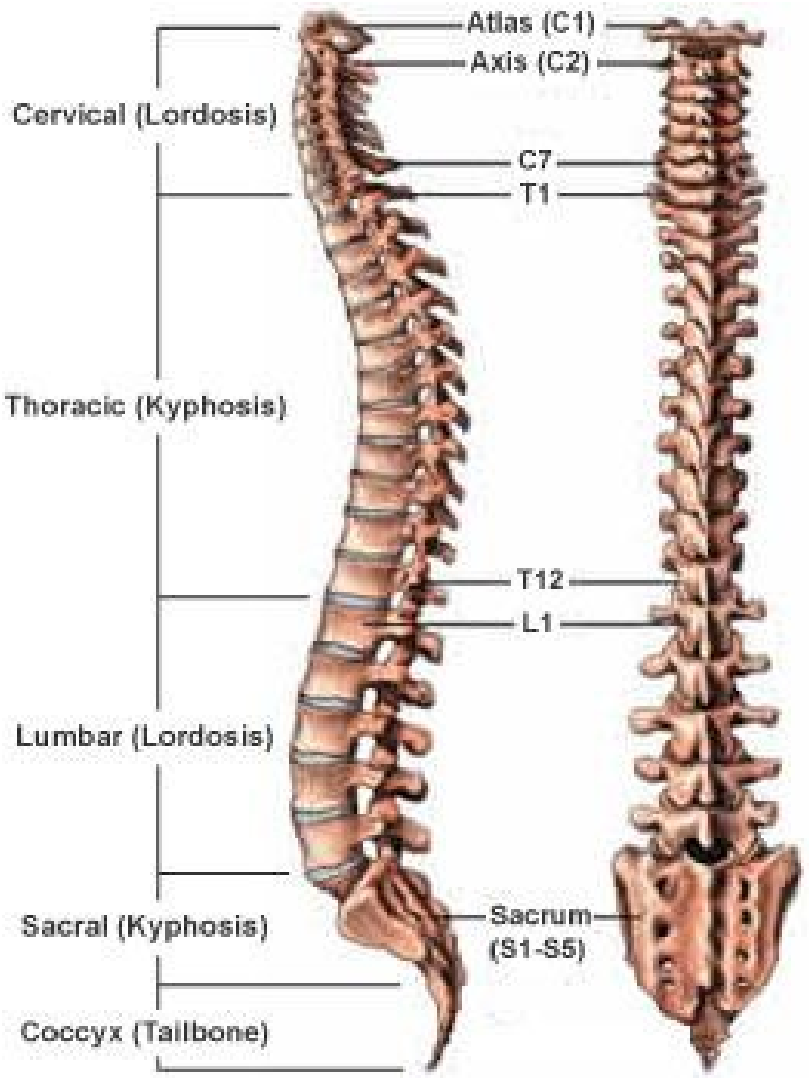
SPINE

It consists of 33 vertebrae:

- 7 cervical
- 12 thoracic
- 5 lumbar
- 5 fused to form sacrum
- 4 fused to form coccyx.

It has 4 curves – Thoracic and sacral curves have concave forward curvature, which are primary curvatures, formed at birth. Cervical and lumbar curves have forward convex curvatures, and are secondary curvatures formed after birth. It has two parts: Anterior solid segment or body, and Posterior segment or arch. Arch is attached to pedicles and lamina at the back. Spinous process extends backwards from the junction of two laminae. Transverse process is formed by junction of pedicles and laminae which extends outwards from each side of arch. Intervertebral foramen is formed from pedicles of each vertebral arch. Spinal nerves enter and exit through this foramen.

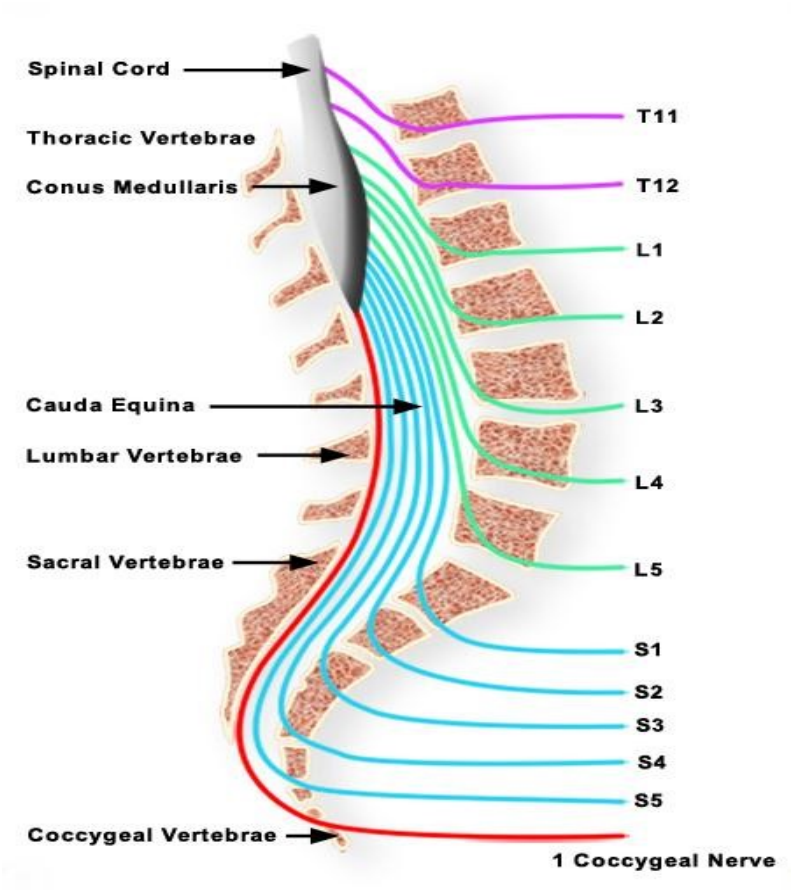
ANATOMY OF SPINE



SPINAL CORD

The spinal cord originates in the brain stem and continues through the foramen magnum in occipital bone, and ends in conus medullaris. It terminates at L3 in infants and lower border of L1 in adults or upper border of L2, due to differences in growth between bony spinal canal and central nervous system. It ends at conus medullaris from where lumbar, sacral and coccygeal nerve roots emerge to form cauda equina.

CAUDA EQUINA



LIGAMENTS

Supraspinous ligament- connects tips of spinous process.

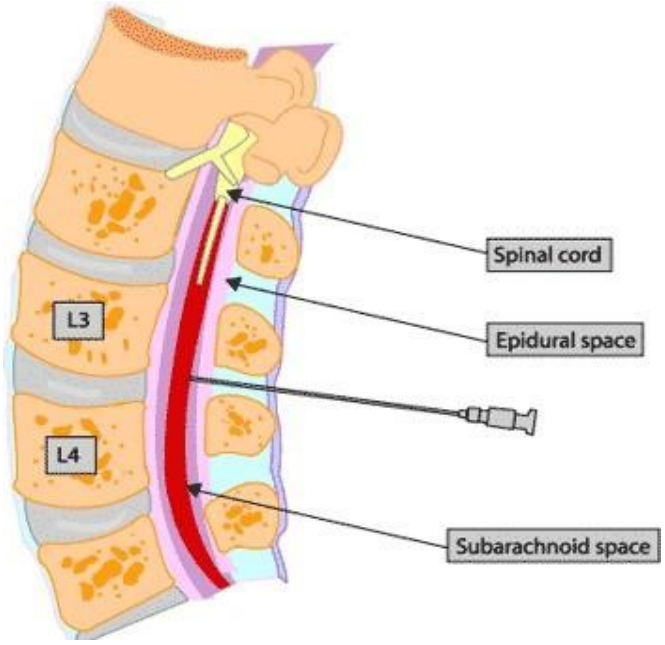
Interspinous ligament – connects spinous processes with one another.

Ligamentum flavum – connects lamina of adjacent vertebrae and consists of elastic fibres. It becomes progressively thicker from front to back and it is easily recognized by increased resistance to passage of needle.

Posterior longitudinal ligament.

Anterior longitudinal ligament.

SUBARACHNOID SPACE



MENINGES AND SPACES

Pia mater is the innermost layer closely attached to spinal cord and brain. It ends at filum terminale, and is highly vascularized. Arachnoid is an avascular membrane tightly attached to the outermost layer, which is the dura mater. It acts as a major barrier to the flow of drugs from the cerebrospinal fluid. Duramater is the third and outermost membrane of spinal canal. It is the continuation of cranial dura mater extending from foramen magnum to S2 level. Subarachnoid space lies between pia mater and arachnoid. CSF, spinal nerves, as well as network of trabeculae and blood vessels supplying spinal cord lie in this space. It extends from cerebral ventricles to the level of S2. Subdural space is a virtual space between dura and arachnoid and it contains serous fluid. Since spinal cord ends at L1 in adults lumbar puncture is usually done below the level of L2 vertebrae to avoid damage to spinal cord.

PHYSIOLOGY OF SUBARACHNOID BLOCK

The cerebrospinal fluid (CSF) is an ultrafiltrate of blood plasma. It is a clear, colourless body fluid, found in the brain and the spinal cord.The average volume in the adult ranges from 120-150 ml . It is secreted by choroid plexus at a rate of 0.3-0.4 ml/minute.

PHYSICAL PROPERTIES OF THE CEREBROSPINAL FLUID

Colour	:	colourless, clear.
Osmolarity	:	281 mOsm/L
pH	:	7.28 – 7.32
Glucose	:	45 – 80 mg /dl.
Proteins	:	20 – 40 mg / dl.
Specific gravity	:	1.006-1.008
Opening pressure	:	90-180 mm H_2O

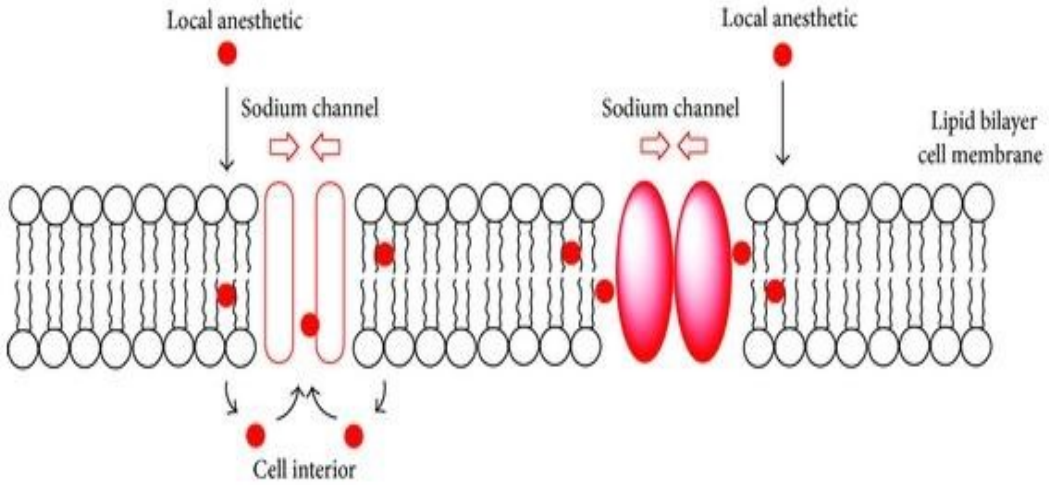
MECHANISM OF SPINAL ANAESTHESIA

The nerve roots leaving the subarachnoid space, are not covered by nerve sheath, and injection of local anaesthetic, blocks these nerve roots and produces reversible loss of sensation and motor function. The local anaesthetics block the conduction of nerve impulses in the posterior nerve root fibres, interrupting the somatic and visceral sensations. By blocking the anterior nerve root fibres, it interrupts efferent motor and autonomic outflow. When a membrane is

depolarized, the signal is transmitted as a wave of depolarization, i.e., the impulse along the nerve membrane. This impulse results in a conformational change of the channel for a brief period. This alteration generates the action potential, which is carried along the nerve. Subsequent return to resting membrane potential, is brought about by the sodium-potassium pump.

The local anaesthetics bind to the sodium channels at a specific site and prevent channel activation, thus inhibiting the sodium influx associated with membrane depolarization. Consequently, impulse conduction slows down, and the magnitude of the action potential decreases. The threshold for excitation increases progressively. Local anaesthetics may also bind to, and inhibit calcium (Ca^{2+}), potassium (K^{+}), transient receptor potential vanilloid 1 (TRPV1), and many other channels and receptors.

MECHANISM



ZONE OF DIFFERENTIAL BLOCKADE

Smaller and slower A∂ fibres are more sensitive to local anaesthetics than larger and faster conducting Aβ fibres, and myelinated fibres are more sensitive than unmyelinated fibres. Sympathetic blockade (sympathetic preganglionic B fibres-myelinated and small 1-3μm) extends two segments higher than the sensory block. Among the sensory fibres C fibres carrying cold temperature sensation are blocked earlier than A∂ fibres carrying pinprick sensation. These sensory fibres are blocked two segments higher than the motor block (Aα fibres –larger 12- 20μm, myelinated).

SPREAD OF LOCAL ANAESTHETICS IN SUBARACHNOID SPACE

After injecting the local anaesthetic into the subarachnoid space it mixes with CSF therefore the concentration of the drug decreases. Spread of local anaesthetics is also determined by the baricity of the solution. Baricity is a ratio comparing the density of a local anaesthetic solution at a specified temperature to the density of CSF at the same temperature. A hypobaric solution has a baricity less than that of CSF. Therefore hypobaric solution floats up to the nerves innervating the surgical site. A hyperbaric solution has a baricity greater than that of CSF. Hyperbaric solutions, settle to the most dependent aspect of the subarachnoid space depending upon the position of the patient. Hypobaric solution are prepared from isobaric solution by adding sterile distilled water.

Hyperbaric solutions are prepared from isobaric solutions by the adding dextrose. Isobaric solutions do not move under the influence of gravity in the CSF. Finally it undergoes vascular absorption into systemic circulation.

INDICATIONS FOR SUBARACHNOID BLOCK:

The surgeries which can be done under spinal anaesthesia, include:

Lower limb surgeries

Lower abdominal surgeries

Obstetric procedures

Urological procedures

Perineal and rectal surgeries

Gynaecological surgeries

CONTRAINDICATIONS FOR SUBARACHNOID BLOCK

ABSOLUTE	RELATIVE	OTHERS
Patient refusal. Bleeding diathesis Severe hypovolemia Infection at injection site Raised intracranial pressure	Sepsis Uncooperative patient Demyelinating disorder Left ventricular outflow tract obstruction. Hypertrophic Obstructive	Prior surgery at injection site Major blood loss surgeries

FACTORS INFLUENCING HEIGHT OF BLOCK:

IMPORTANT FACTORS :	DRUG FACTORS :
Dose	Concentration
Baricity	Viscosity
Position of patient	Volume
Pregnancy	
Advanced age	
Height.	

PATIENT FACTORS :	PROCEDURAL FACTORS
Anatomy of spine	Level of injection
Intra abdominal pressure	Type of needle
	Direction of needle tip

EFFECTS OF SPINAL ANAESTHESIA

Spinal anaesthesia blocks the sympathetic nervous system leading to an unopposed activity of the parasympathetic system. The effects of spinal anaesthesia on the various systems are :

CARDIOVASCULAR SYSTEM

As spinal anaesthesia produces blockade of peripheral sympathetic (T1-L2) and cardiac sympathetic(T1-T4) fibres if the level of blockade is higher, there is a decrease in stroke volume and heart rate. Because of vasodilation caused by the sympathetic blockade, there is decrease in both preload and afterload. The decrease in preload leads to decreased cardiac output. Decrease in venous return due to peripheral pooling of blood and blockade of the cardio accelerator fibres (T1-T4) leads to a decrease in the heart rate. This hypotension after neuraxial block is thought to be minimised by preloading or co-loading with crystalloids intravenously. When mean arterial pressure decreases less than 60mm Hg, or a 30% reduction from the baseline value, a mixed adrenergic agonist such as ephedrine may be used for the treatment. Ephedrine has direct and indirect β-adrenergic effects. It increases the heart rate and contractility and by indirect effects also produces vasoconstriction. Small doses of adrenaline (2-5mcg boluses) are also useful in treating spinal induced hypotension.If the hypotension persists, or is profound, vasopressor infusions need to be given. Excessive or symptomatic bradycardia needs to be treated with atropine.

RESPIRATORY SYSTEM

Due to paralysis of abdominal muscles there is a minimal decrease in vital capacity as a result of decrease in expiratory reserve volume. This is usually compensated by scalenes and sternomastoids.Caution may be needed in patients with severe lung disease who depend on accessory muscles of respiration.

However the risks of general anaesthesia requiring airway instrumentation must be weighed against the respiratory problems associated with a high spinal anaesthesia.

For surgeries above the umbilicus, spinal anaesthesia may not be a good option especially for patients with severe lung disease. Respiratory arrest due to spinal anaesthesia is rare, and is due to decreased perfusion of the brainstem respiratory centres and apnea resolves as blood pressure and cardiac output are restored.

GASTROINTESTINAL SYSTEM

Blockade of splanchnic sympathetic fibres from T6 to L1 level results in a contracted gut and hyperperistalsis. This leads to nausea and vomiting in 20% of patients. Atropine is effective in treating the nausea and vomiting that occurs due to a high spinal. Reduction in mean arterial pressure, correlates with a decrease in hepatic blood flow.

RENAL SYSTEM

As renal blood flow is maintained through autoregulation, spinal anaesthesia does not cause significant decrease in renal blood flow. Spinal anaesthesia blocks sympathetic and para sympathetic control of the bladder function. Though it is believed that regional anaesthesia is a frequent cause of urinary retention and necessitating bladder catheterisation, it is still questionable.

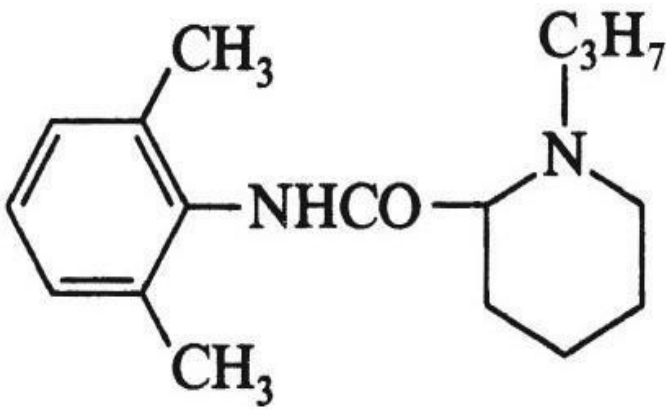
COMPLICATIONS OF SUBARACHNOID BLOCK :

Immediate :	**Delayed**
Hypotension	Post dural puncture headache
Bradycardia	Retention of urine
Toxicity due to intravascular injection	Backache
Allergic reaction to local anaesthetic	Meningitis
Hypoventilation -	Transient lesions of cauda equina
(due to brain stem hypoxia)	Sixth nerve palsy
	Anterior spinal artery syndrome
	Horner's syndrome.

PHARMACOLOGY OF ROPIVACAINE :

GENERIC NAME Ropivacaine Hydrochloride injection

CHEMICAL STRUCTURE



It is a member of the amino amide class of local anaesthetics.

It is chemically described as S-(-)-1-propyl-2`,6`pipecoloxylidide

hydrochloride monohydrate. Ropivacaine belongs to pipecoloxylidide group of local anaesthetics with a propyl group attached to the piperidine nitrogen. However, it differs from other drugs in the group in that they are racemic preparations, while ropivacaine is the first drug to be available as a pure S-(-) enantiomer.

PHYSICOCHEMICAL PROPERTIES

The drug substance is a white crystalline powder, with a chemical formula of $C_{17}H_2N_2OHClH_2O$. The pKa of ropivacaine is approximately the same as bupivacaine and is similar to that of mepivacaine . However, ropivacaine has an intermediate degree of lipid solubility compared to bupivacaine and mepivacaine determined by the N heptane/buffer partition coefficient.

Molecular weight (base)	274
PKa	8.1
Potency	4
Protein binding in %	94
Fraction % non ionized at pH7.4	17
Partition Coefficient	2.9

MECHANISM OF ACTION

Ropivacaine is a member of the amino amide class of local anesthetics and is supplied as the pure S-(-) enantiomer. Local anesthetics block the conduction of nerve impulses by blocking the sodium ion channels, thereby decreasing sodium ion conductance and preventing depolarization of the cell membrane.

Parameters	Value
Elimination (t 1/2 in min)	108
Clearance (L/min)	0.44
Vdss (L)	59
Protein Binding (%)	94

PHARMACOKINETICS

ABSORPTION

The systemic absorption of ropivacaine after intrathecal injection is slow with peak plasma concentration being achieved much later than bupivacaine. This may be due to the intrinsic vasoconstrictor property of ropivacaine at low concentrations.

BIODEGRADATION AND METABOLISM

Ropivacaine is metabolized in liver into 2,6-pipecoloxylidide and 3-hydroxyropivacaine by cytochrome P-450 enzymes. Both metabolites have significantly less local anaesthetic potency than ropivacaine. About 1% is excreted unchanged in the urine. Its clearance is higher than bupivacaine and

elimination half-time shorter. The higher clearance may offer an advantage over bupivacaine in terms of systemic toxicity. It has a lipid solubility intermediate between lignocaine and bupivacaine and is highly bound to alpha1 acid glycoprotein.

SYSTEMIC TOXICITY

Central Nervous System Toxicity

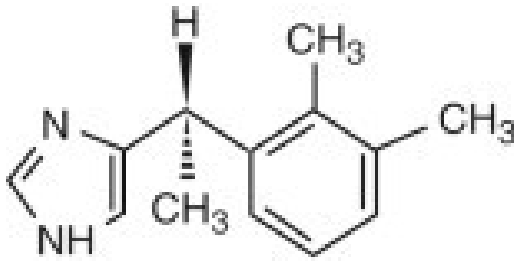
Ropivacaine produces similar spectrum of symptoms involving the central nervous system like bupivacaine but the duration of symptoms is shorter with the former. Moreover, studies have shown that higher doses and free plasma concentrations of ropivacaine were tolerated before symptoms were elicited.

Cardiovascular System Toxicity:

Cardiovascular effects are less pronounced with ropivacaine. The very slow reversal of Na+channel blockade after a cardiac action potential, which is a hallmark of bupivacaine, is considerably faster with ropivacaine. In addition, the negative inotropic potency of ropivacaine on isolated cardiac tissue appears to be considerably less than that of bupivacaine. Studies in animals show that aggressive cardiac resuscitation after an intentional intravenous bolus in dogs leads to effective reversal of the toxic effects far more frequently with ropivacaine than with bupivacaine indicating that ropivacaine is less cardiotoxic.

The greater safety of ropivacaine than bupivacaine may be related both to the reduced toxicity of the single (S) - isomer and the difference between the propyl and butyl –N- piperidine substituent.

PHARMACOLOGY OF DEXMEDETOMIDINE



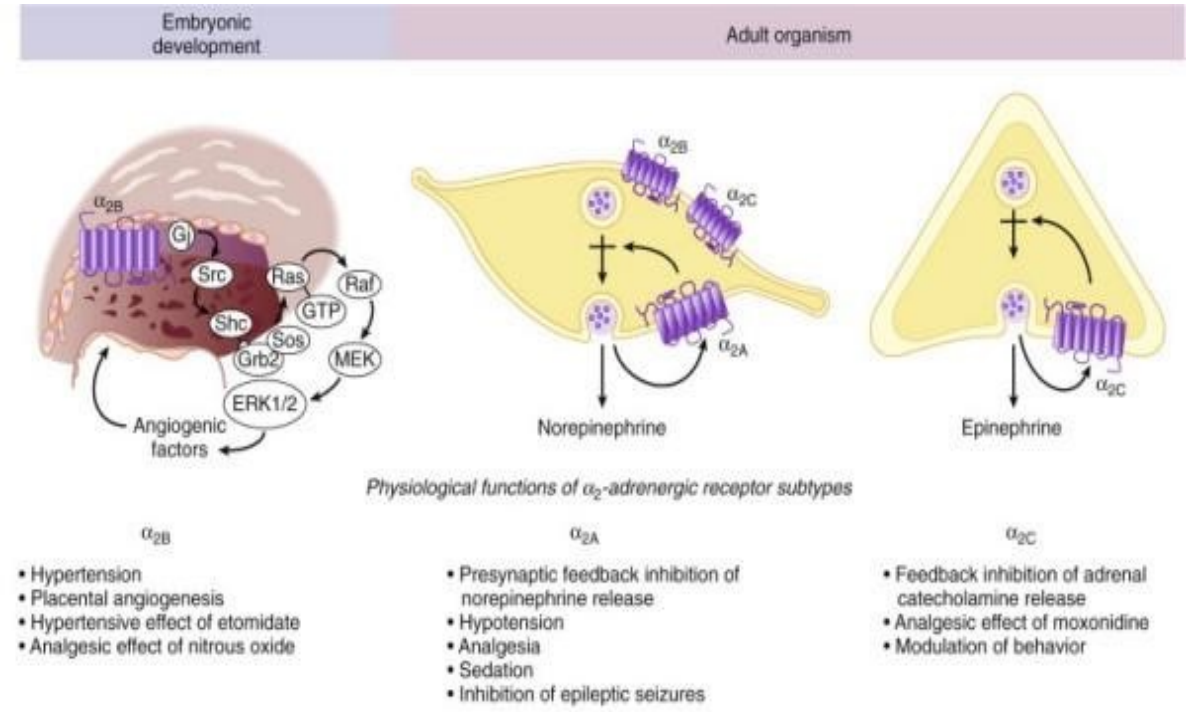
Dexmedetomidine is an α2-agonist that received FDA approval in 1999 for use as a short-term (less than 24 h) sedative analgesic in the intensive care unit. Clonidine, the prototype of α2-agonist, is widely used as an adjunct to anesthesia and pain medicine; however, it has been little used as a sedative. With dexmedetomidine, there are a number of reasons for the growing and renewed interest in the use of α2-adrenoceptors agonists as sedatives.

Dexmedetomidine compared to clonidine is a much more selective α2- adrenoceptor agonist, which might permit its application in relatively high doses for sedation and analgesia without the unwanted vascular effects from activation of α1- receptors. In addition, dexmedetomidine is a short acting drug than clonidine and has a reversal drug for its sedative effect, atipamezole.

These properties render dexmedetomidine suitable for sedation and analgesia during the whole perioperative period: as premedication, as an anesthetic adjunct for general and regional anesthesia and as postoperative sedative and analgesic.

Physiology of α2-adrenoceptors

α2 - receptors are found in many sites throughout the body. α2 - adrenoceptors are found in peripheral and central nervous systems, in effector organs such as the liver, kidney, pancreas, eye vascular smooth muscles and platelets. Physiologic responses mediated by α2 – adrenoceptors vary with location and can account for the diversity of their effects.



The different physiologic functions of α2 adrenoreceptors. The top panel depicts the three α2 receptor subtypes acting as presynaptic inhibitory feedback receptors to control the release of norepinephrine and epinephrine from

peripheral or central adult neurons. Also, a negative feedback loop has been seen in the adrenal gland. Alpha2B receptors have been involved in the development of the placental vascular system during prenatal development. The lower panel lists a series of physiologic effects with its associated α2 adrenoreceptors.

The classification of α2 - receptors based on anatomical location is complicated since these receptors are found in presynaptic, postsynaptic and extrasynaptic locations. α2 adrenoceptors are divided into three subtypes; each subtype is responsible uniquely for some of the actions of α2 - receptors.

α2A - predominant subtype in CNS, is responsible for the sedative, analgesic and sympatholytic effect.

α2B - found mainly in the peripheral vasculature, is responsible for the short-term hypertensive response.

α2C - found in the CNS, is responsible for the anxiolytic effect.

All the subtypes produce cellular action by signaling through a Gprotein which couples to effector mechanisms. This coupling appears to differ depending on the receptor subtype and location. The α2A-adrenoceptor subtype seems to couple in an inhibitory fashion to the calcium channel in the locus ceruleus of the brainstem, whereas, in the vasculature, the α2Badrenoceptor sub type couple in an excitatory manner to the same effector mechanism.

Mechanism of action of Dexmedetomidine

The mechanism of action of dexmedetomidine is unique and differs from the currently used sedative drugs. α2 - adrenoceptors are found in many

sites through the CNS, however, the highest densities of α2-receptors are found in the locus ceruleus, the predominant noradrenergic nuclei of the brainstem and an important modulator of vigilance. Presynaptic activation of the α2A adrenoceptor in the locus ceruleus inhibits the release of norepinephrine (NE) and results in the sedative and hypnotic effects. In addition, the Locus Ceruleus is the site of origin for the descending medullospinal noradrenergic pathway, known to be an important modulator of nociceptive neurotransmission. Stimulation of the α2-adrenoceptors in this area terminates the propagation of pain signals leading to analgesia. Postsynaptic activation of α2-adrenoceptors in the CNS results in a decrease in the sympathetic activity leading to hypotension and bradycardia. Also, activation of the α2-adrenoceptors in the CNS results in an augmentation of cardiac vagal activity. Combined, these effects can produce analgesia, sedation and anxiolysis.

At the spinal cord, stimulation of α2-receptors at the substantia gelatinosa of the dorsal horn leads to inhibition of the firing of nociceptive neurons and inhibition of the release of substance P. Also, the α2- adrenoceptors located at the nerve endings have a possible role in the analgesic mechanisms of α2-agonists by preventing NE release. The spinal mechanism is the principal mechanism for the analgesic action of Dexmedetomidine, even though there is a clear evidence for both a supraspinal and peripheral sites of action.

α2 - receptors are located on the blood vessels where they mediate vasoconstriction and on sympathetic terminals, where they inhibit NE release. The responses of activation of α2-adrenoceptors in other areas include

contraction of vascular and other smooth muscles; decreased salivation, decreased secretion, and decreased bowel motility in the gastrointestinal tract, inhibition of renin release, increased glomerular filtration, and increased secretion of sodium and water in the kidney; decreased insulin release from the pancreas, decreased intraocular pressure, decreased platelet aggregation and decreased shivering threshold by 2°C.

Pharmacodynamics of Dexmedetomidine

α - adrenoceptors agonists have different α2/α1 selectivity. Clonidine, the first developed and the most known α2-agonist is considered as a partial α2-agonist since its α2/α1 selectivity is 200:1 while the α2/α1 selectivity of dexmedetomidine is 1620:1 and hence it is 8 times more powerful α2-adrenoceptor agonist than clonidine and is considered as a full α2 adrenoceptor agonist. The α2-adrenoceptor selectivity of dexmedetomidine is dose-dependent; at low to medium doses or at slow rates of infusion, high levels of α2 - adrenoceptor selectivity are observed, while high doses or rapid infusions of low doses are associated with both α1 and α2 activities.

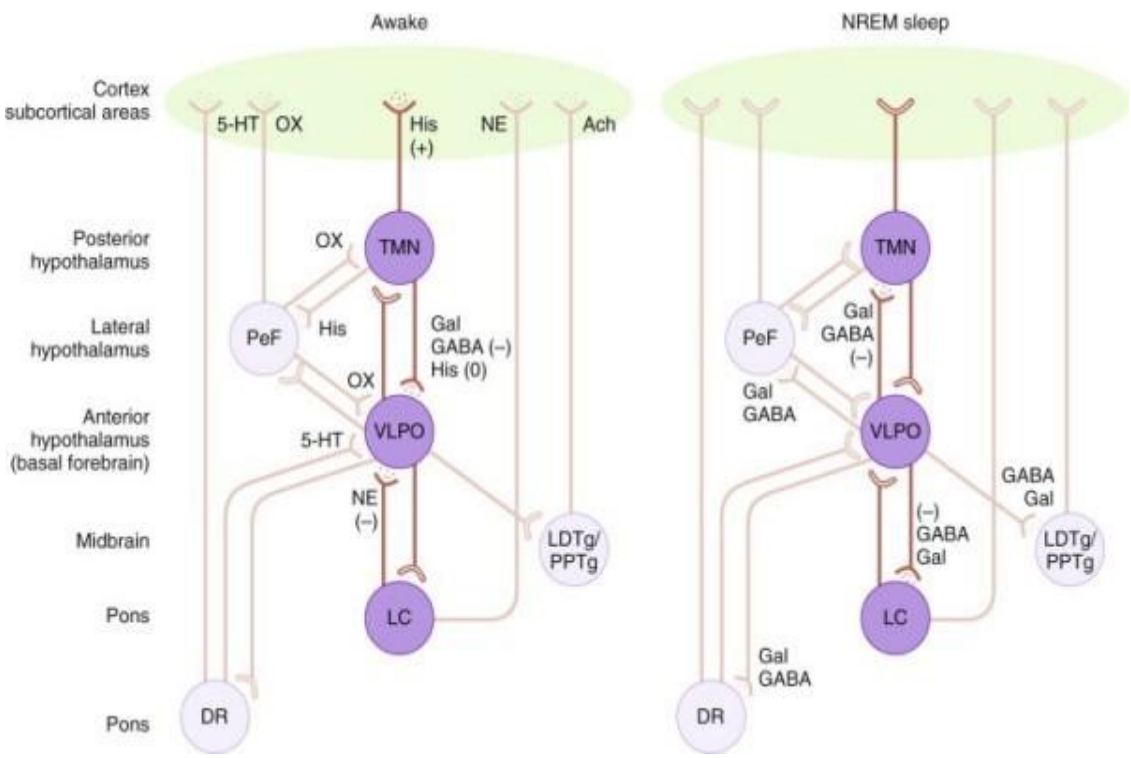
CNS effects

Dexmedetomidine induced sedation qualitatively resembles normal sleep. The participation of non rapid eye movement sleep pathways seems to explain why patients who appear to be “deeply asleep” from dexmedetomidine are relatively easily aroused in much the same way as occurs with natural sleep.

This type of sedation is branded “cooperative” or “arousable”, to distinguish it from the sedation induced by drugs acting on the GABA system such as midazolam or propofol, which produce a clouding of consciousness. Sedation induced by dexmedetomidine is dose-dependent; however, even low doses might be sufficient to produce sedation.

However, clinical studies showed that systemic administration of the α2 -adrenoceptor agonists, dexmedetomidine and clonidine produce sedative and opioid-sparing effects in the perioperative setting, providing indirect evidence for some analgesic efficacy, although it is difficult in this special setting to distinguish between sedation and analgesia as a cause for this opioid-sparing effect. While the analgesic effect of systemic dexmedetomidine is still debatable, administration of an α2-agonist (clonidine) via the intrathecal or epidural route provides analgesic effects in postoperative pain and in neuropathic pain state without severe sedation.

This effect is due to sparing of the supraspinal CNS sites from excessive drug exposure resulting in robust analgesia without heavy sedation.



The stimulation of the locus caeruleus (LC) by dexmedetomidine (right diagram) releases the inhibition the LC has over the ventrolateral preoptic nucleus (VLPO). The VLPO subsequently releases γ-aminobutyric acid (GABA) onto the tuberomammillary nucleus (TMN). This inhibits the release of the arousal-promoting histamine on the cortex and forebrain, inducing the loss of consciousness.

Respiratory effects

α2 - adrenoceptors do not have an active role in the respiratory center. Therefore, dexmedetomidine throughout a broad range of plasma concentration has minimal effects on the respiratory system. Coadministration of dexmedetomidine with other sedatives, hypnotics or opioids is likely to cause additive effects.

Cardiovascular effects

Dexmedetomidine does not appear to have direct effects on the heart. In the coronary circulation, dexmedetomidine causes a dose dependent increase in coronary vascular resistance and oxygen extraction, but the supply/demand ratio is unaltered. A biphasic cardiovascular response has been described after the administration of dexmedetomidine. A bolus of 1 μg/kg results in a transient increase in blood pressure (BP) and a reflex decrease in heart rate (HR), especially in the young healthy patients. This initial response is attributed to the direct effects of α2B-adrenoceptor stimulation of vascular smooth muscle. This response can be attenuated by a slow infusion over 10 min, but even at slower infusion rates, the transient increase in mean BP and the decrease in HR over the first 10 min is shown.

This initial response lasts for 5 to 10 min and is followed by a decrease in BP of 10-20% below baseline and by stabilization of the HR below baseline values. Both these effects are presumably caused by an inhibition of central sympathetic outflow that overrides the direct effects of dexmedetomidine on the vasculature. Hypotension and bradycardia induced by dexmedetomidine are reversed by ephedrine and atropine respectively, but large doses are required. Dexmedetomidine decreases the heart rate in dose-dependent manner in children. This effect is attributed to a centrally mediated sympathetic withdrawal, which results in unregulated cholinergic activity.

Pharmacokinetics of Dexmedetomidine

Dexmedetomidine, an imidazole compound, is the active d-isomer of medetomidine. Following intravenous administration, dexmedetomidine exhibits the following pharmacokinetic parameters: a rapid distribution phase with a distribution half-life (t½ α) of 6 min, a terminal elimination half-life (t ½β) of 2 hours and a steady-state volume of distribution (Vss) of 118 liters and a clearance about 39L. Dexmedetomidine exhibits linear kinetics when infused in the dose range of 0.2-0.7 μg/kg/h for no more than 24 hours. Dexmedetomidine undergoes almost complete biotransformation through direct glucuronidation and cytochrome P450 metabolism. Metabolites of biotransformation are excreted in the urine (95%) and feces. It is unknown if they had intrinsic activity.

The average protein binding of dexmedetomidine is 94%, with negligible protein binding displacement by fentanyl, digoxin, theophilline,lidocaine and ketorolac. There have been no sex or age-based differences in the pharmacokinetics of dexmedetomidine. The dose of dexmedetomidine should be decreased in patients with hepatic or renal impairment. Dexmedetomidine does cross the placenta and should be only used during pregnancy if the potential benefits justify the potential risk to fetus. Dexmedetomidine is a white powder that is freely soluble in water and has a pka of 7.1. It is supplied as 100 μg/ml 2 ml vial which must be diluted with 48 ml of 0.9% sodium chloride prior to administration.

For adult patient, dexmedetomidine is administered by a loading infusion of 0.5-1 μg/kg over 10 minutes, followed by a maintenance infusion of 0.2 to 0.7 μg/kg/h.

The effect appears in 5-10 min, and is reduced in 30-60 min. The maintenance infusion is adjusted to achieve the desired level of sedation. The most frequently observed adverse events in patients receiving dexmedetomidine for ICU sedation include hypotension, hypertension, nausea, bradycardia and atrial fibrillation. Most of these events occur during or after the loading dose, therefore, reducing or omitting the loading dose could result in decreasing the incidence and severity of these adverse events.

Appropriate patient selection for dexmedetomidine administration is crucial; because it decreases sympathetic nervous activity, its effects may be most pronounced in patients with decreased autonomic nervous system control such as the elderly, diabetic patients, patients with chronic hypertension or severe cardiac disease such as valve stenosis or regurgitation, advanced heart block, severe coronary artery disease or in patients who are already hypotensive and/or hypovolemic.

Dexmedetomidine does not affect the synthesis, storage or metabolism of neurotransmitters and does not block the receptors, thus providing the possibility of reversing the hemodynamic effects with vasoactive drugs or the specific alpha2- antagonist, Atipamezole which acts by increasing the central turnover of norepinephrine. Its duration of action is 2 hours .

Perioperative uses of dexmedetomidine

I – Premedication

Dexmedetomidine possesses anxiolytic, sedative, analgesic, antisialogogue and sympatholytic properties, which render it suitable as a premedication agent. Dexmedetomidine potentiates the anesthetic effects of all intraoperative anesthetics (intravenous, volatile or regional block). Bohrer showed that preoperative administration of intravenous or intramuscular dexmedetomidine resulted in a decrease in the induction dose of thiopentone by up to 30%. The administration of intramuscular dexmedetomidine at a dose of 1 μg/kg for premedication in outpatient cataract surgery resulted in sedation, and decrease in intraocular pressure without significant hypotension or bradycardia. Also the administration of dexmedetomidine for premedication decreases oxygen consumption intraoperatively by 8% and postoperatively by 17%. Indications for the use of dexmedetomidine as premedication include patients susceptible to preoperative and perioperative stress, drug addicts and alcoholics, chronic opioid users and hypertensive patients.

II – Intraoperative uses of dexmedetomidine

Intraoperative uses of dexmedetomidine include its use as an adjunct to general anesthesia, as an adjunct to regional anesthesia, in monitored anesthesia care (MAC) or as a sole agent for total intravenous anesthesia (TIVA).

1– Use of dexmedetomidine as adjunct to general anesthesia

The use intraoperative dexmedetomidine may increase hemodynamic stability because of attenuation of the stress-induced sympathoadrenal responses to intubation, during surgery and during emergence from anesthesia. Talke evaluated the effects of varying plasma concentrations of dexmedetomidine on HR, BP and catecholamines concentrations during emergence from anesthesia in the setting of vascular surgery. This study demonstrated that dexmedetomidine attenuates the increases in heart rate and plasma norepinephrine levels observed during the emergence from anesthesia.

Administration of intravenous dexmedetomidine produces an anesthetic-sparing effect. Aho showed 25% reduction of maintenance concentrations of isoflurane in patients undergoing hysterectomy. Khan found 35%-50% reduction in isoflurane concentrations with either low or high doses of dexmedetomidine. Fragen noted 17% reduction in sevoflurane requirements for maintenance of anesthesia in elderly patients. In addition, the use of dexmedetomidine produces intraoperative and postoperative opioid-sparing effect. Aho administered dexmedetomidine at dose of 0.4 μg/kg in patients undergoing laparoscopic tubal ligation and found a 33% decrease in morphine use postoperatively.

Talke investigated the muscle relaxant effects of dexmedetomidine on the neuromuscular junction and found no clinically relevant effects. Dexmedetomidine reduces the vasoconstriction threshold and the shivering threshold and is associated with a lower incidence of shivering.

2 – Use of dexmedetomidine for regional anesthesia

The use of dexmedetomidine as adjuvant in regional anesthesia is still not validated. Maarouf explored the effect of epidural dexmedetomidine on the incidence of postoperative shivering in patients undergoing orthopedic surgery. He found that patients who received dexmedetomidine at a dose of 100 μg added to 20 ml 0.5% bupivacaine showed lower incidence in postoperative shivering when compared to patients who received epidural bupivacaine alone (10% vs.36%). Memis noted that the addition of 0.5 μg/kg dexmedetomidine to lidocaine for intravenous regional anesthesia improves the quality of anesthesia and perioperative analgesia without causing side effects. Kanazi et al investigated the effect of adding a small dose of 3 μg of intrathecal dexmedetomidine to 12 mg bupivacaine. They found a significant prolongation of sensory and motor block as compared to bupivacaine alone. In this study, the effect of 3 μg intrathecal dexmedetomidine was similar to that produced by the addition of 30 μg of intrathecal clonidine.

3 – Use of dexmedetomidine in monitored anesthesia care

Dexmedetomidine confers arousable sedation with ease of orientation, anxiolysis, mild analgesia, lack of respiratory depression and hemodynamic stability at moderate doses. These properties allow dexmedetomidine to be an almost ideal agent for MAC despite its lack of amnesia and poor controllability because of its slow onset and offset. The efficacy, side effects, and recovery characteristics of dexmedetomidine were compared to propofol when used for MAC. This study showed that dexmedetomidine achieved similar levels of

sedation to propofol, albeit with a slower onset and offset of sedation. Neither dexmedetomidine nor propofol influenced respiratory rate, but propofol resulted in lower mean arterial pressure during the intraoperative period. In the recovery room, dexmedetomidine was associated with an analgesia sparing effect, slightly increased sedation, but no compromise of respiratory function or psychomotor responses. Dexmedetomidine in MAC was used successfully in many situations: when patient arousability needed to be preserved, as for awake craniotomy, for awake carotid endarterectomy and for vitreoretinal surgey. In addition, dexmedetomidine was used for sedation in difficult airway patients; during fiberoptic intubation, and for sedation of a patient with difficult airway undergoing lumbar laminectomy surgery in the prone chest position under spinal anesthesia.

4 – Use of dexmedetomidine as a sole anesthetic agent

Ramsay has used dexmedetomidine as a sole anesthetic agent. The report describes three patients who presented for surgery with potential airway management challenges. Dexmedetomidine was infused in increasing doses (up to 10 μg/kg/hr) until general anesthesia was attained. No respiratory depression was noted, only one patient required chin lift. Also no hypotension or severe bradycardia were noted. The rationale for this use of dexmedetomidine is based on its known properties to provide sedation, analgesia while avoiding respiratory depression at low doses. These effects were maintained at higher doses without hemodynamic instability.

III – Use of dexmedetomidine in the postoperative period

Dexmedetomidine's special properties favour its use in the recovery room. In addition to its sympatholytic effects, analgesic effects and decreased rate of shivering, the preservation of respiratory function allows the continuation of the dexmedetomidine infusion in the extubated, spontaneously breathing patient. The possibility of ongoing sedation and sympathetic block could be beneficial in reducing high rates of early postoperative ischemic events in high-risk patients undergoing non-cardiac surgery. During emergence from anesthesia, dexmedetomidine reduces NE levels significantly. However, patients who received intraoperative dexmedetomidine needed more fluids to avoid hypotension, a side effect that may be unfavorable in volume-sensitive patients with reduced left ventricular function. In addition, care should be taken in patients who depend on a high level of sympathetic tone or in patients with reduced myocardial function who cannot tolerate the decrease in sympathetic tone. Perioperative administration of dexmedetomidine could be beneficial in chronic opioid users and alcoholics, in high-risk patients as well as in cardiac patients with good to moderately decreased left ventricular function.

IV – Use of Dexmedetomidine in the pediatric-age group

Only few case reports about the use of dexmedetomidine in the pediatric age group are found in the literature . Tobias used dexmedetomidine for ICU sedation in a10-week old infant requiring mechanical ventilation and in a 14-y old patient after posterior spinal fusion for scoliosis. The use of dexmedetomidine at a dose of 0.25 μg/kg/hr for 24 h in these two cases resulted

in acceptable sedation without significant hemodynamic changes. Dexmedetomidine was also used for sedation and anesthesia in an 11-y old patient undergoing gastroscopy; however, it resulted in insufficient sedation. Another study conducted in pediatric-age group explored the use of intraoperative dexmedetomidine at different doses with the goal of reducing the post sevoflurane agitation in children aged 1-10 y.

The optimal dose of dexmedetomidine was 0.3 μg/kg and its use did not result in adverse effects.When compared with propofol for sedation during MRI, dexmedetomidine provides adequate sedation during the scan but has a slower recovery profile . One of the major advantages of dexmedetomidine over other sedatives is its respiratory effects, which are minimal in adults and children. it does not lead to extreme hypoxia or hypercapnia. Indeed, respiratory rate, CO_2 tension, and oxygen saturation are generally maintained during dexmedetomidine sedation in children.

PHARMACOLOGY OF MAGNESIUM SULFATE

It is a bivalent ion like calcium with an atomic weight of 24.312. Human body contains 1 mole (24g) of magnesium. It is the fourth common mineral salt in the body after phosphorus, calcium and potassium, second intracellular cation after potassium. In serum, magnesium is divided into three fractions-

1) Ionised,
2) Protein bound and
3) Contained in anion complexes.

These fractions account for 65%, 27%, and 8% in serum concentration respectively.

PROPERTIES OF MAGNESIUM SULFATE

CELLULAR PROPERTIES

Magnesium intervenes in the activation of membrane calcium ATPase and Na+ K+ ATPase involved in transmembrane ion exchange during depolarization and repolarization phases. It acts as a stabilizer of cell membrane and intracytoplasmic organelles.

ION CHANNELS

It acts as a regulator of different ion channels. It has a competitive antagonist action against calcium inflows thereby limiting the outflow of calcium from the sarcoplasmic reticulum. So it is a calcium channel blocker and calcium channel modulator.

CARDIOVASCULAR SYSTEM

It acts on calcium channels in the myocardial muscle and also acts indirectly on the cardiac muscle by inhibiting the calcium uptake on the troponin C of the myocytes, and thereby influencing myocardial contractility. Its vasodilatory action is due to its activation of cyclic AMP. This causes reduction in systolic blood pressure. Pulmonary vascular resistance is unaltered. coronary vascular resistance is reduced and it causes coronary vasodilation.

NEUROMUSCULAR TRANSMISSION

It has a preponderant presynaptic and postsynaptic effect. Magnesium acts competitively in blocking the entry of calcium into the presynaptic endings.

Presynaptic release of acetylcholine is reduced by magnesium, thereby decreasing the effect of acetylcholine on the postsynaptic receptors, which in turn increases the threshold of axonal excitation. It also produces progressive inhibition of catecholamine release from the adrenal medulla, adrenergic nerve endings and adrenergic postganglionic sympathetic fibres.

RESPIRATORY SYSTEM

It has bronchodilator action due to the inhibition of smooth muscle contraction, It also inhibits histamine release from the mast cells and acetylcholine release from the cholinergic nerve endings.

It is involved in hundreds of enzyme reactions in the body.

Acts as an antagonist of NMDA receptors and this explains its use in post-operative analgesia.

Magnesium sulphate increases production of prostaglandins causing vasodilatation of the small intracranial vessels, which is responsible for its anticonvulsant action.

CLINICAL USES

1) For Severe Preeclampsia and Eclampsia:

A loading dose of 4-6gm magnesium sulfate diluted in 100ml of normal saline given over 15min intravenously. Then 2 gm/hr in 100ml of IV infusion. (maintain serum levels between 4 and 7mEq/L). Intermittent injection: 4gm given slow IV followed by 10gm, dose divided into 5gm in each buttocks as deep intramuscular injection. Then every 4hrs 5gm intramuscularly upto 24hrs after delivery.

2) Magnesium sulfate has a tocolytic effect at serum levels of 8- 10mEq/L. Loading dose of 4-6gm over 20min intravenously, then after the contraction ceases, maintenance is done using 2-4gm per hour intravenously for 12-24 hours.
3) To reduce the stress response during intubation, magnesium sulphate is used in the dosage of 30-50mg/kg. intravenously.
4) In surgery for pheochromocytoma, it helps to maintain haemodynamic balance because it inhibits the catecholamine release from adrenal medulla and adrenergic nerve endings.
5) Seizures: In children with seizures, the 50% concentration should be diluted to a 20% solution for intramuscular injection. The dose for children is 20 to 40 mg (0.1 to 0.2 mL of a 20% solution)/kg of body weight, administered intramuscular as needed, to control seizures.
6) It is used postoperatively in patients who have undergone coronary artery bypass grafting to reduce the incidence of ventricular arrhythmias.
7) It is also used in the treatment of Torsades De Pointes, intravenously or intraosseously in the dosage of 25 to 50 mg/ kg (upto 2 gm).
8) Acute myocardial infarction: magnesium sulphate is used in the dose of 2gm intravenously over 5-15 min followed by 18 gm over 24hrs as infusion.
9) Total Parenteral Nutrition: In total parenteral nutrition, maintenance requirements for magnesium are not precisely known. The maintenance dose recommended for adults is 5 to 8 mEq magnesium/L of solution; typical

daily adult intake ranges from 10 to 24 mEq. For infants, the recommended intake ranges from 0.25 to 0.6 mEq/kg/day.

10) In barium poisoning: 1-2gm is used to counteract the intense muscle stimulating effects of barium.

11) In refractory bronchial asthma it is used for its bronchodilatory action.

12) Hypomagnesemia: in case of mild deficiency 1gm every 6 hours for 4 doses, in severe cases 1-5gms (2 – 10ml of 50% solution) in divided doses, repeated until the serum levels are normal.

13) Recent studies show its use in Tetanus patients, at a serum concentration of 2-4mEq/L, it gives good control of spasms and muscle rigidity.

14) Magnesium sulphate is used in the dose of 50 mg intrathecally for potentiation of opioid analgesia.

PRECAUTIONS

Since magnesium is eliminated from the body solely by the kidneys, the drug should be used with caution in patients with renal impairment. Urine output should be maintained at a level of 25 - 50 ml per hour. Monitoring serum magnesium levels, and the patient's clinical status is essential to avoid the consequences of over dosage in toxaemia of pregnancy. Safe dose of magnesium is assessed clinically by presence of the patellar reflex (knee jerk) and absence of respiratory depression (approximately 16 breaths or more/minute). Serum magnesium levels usually sufficient to control convulsions range from 3 to 6 mg/100 ml (2.5 to 5.0 mEq/L).The strength of the deep tendon reflexes begins to diminish when magnesium levels

exceed 4mEq/L. Reflexes may be absent at 10 mEq magnesium/L, where respiratory paralysis can occur .An injectable calcium salt should be immediately available to counteract the potential hazards of magnesium intoxication in eclampsia.

Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration. The drug should not be administered, unless solution is clear and container is undamaged. The unused portion of drug must be discarded. When administered intravenously the onset of action is immediate and duration of action is 30 min. On administration by intramuscular route, the onset of action takes 1hr and duration of action is 3-4 hrs.

Storage: 15-30degree centigrade. For IV use concentration of 20% or less should be used.

Rate of injection should be 1.5ml/hr.

DRUG INTERACTIONS

Central nervous system depressants: When barbiturates, opiates, general anaesthetics, or other CNS depressants are administered concomitantly with magnesium sulphate, dosage of these agents must be carefully adjusted because of the additive central depressant effects. Neuromuscular blocking agents: Excessive neuromuscular blockade has occurred in patients receiving parenteral magnesium sulphate and a neuromuscular blocking agent.

Cardiac glycosides: Magnesium salts should be administered with extreme caution in digitalized patients because serious changes in cardiac conduction,

which can result in heart block may occur if administration of calcium is required to treat magnesium toxicity.

ADVERSE REACTIONS

The adverse effects of parenterally administered magnesium usually are the result of magnesium intoxication. These include sweating, flushing, hypotension, depressed reflexes, flaccid paralysis, hypothermia, circulatory collapse, cardiac and CNS depression proceeding to respiratory paralysis. Hypocalcaemia with signs of tetany can occur secondary to magnesium sulphate therapy for eclampsia.

SYMPTOMS AND TREATMENT OF OVERDOSE

Magnesium toxicity is manifested by severe hypotension and respiratory paralysis. The first clinical sign to indicate magnesium toxicity is disappearance of patellar reflex. In the event of over dosage respiratory muscle paralysis occurs so artificial ventilation must be provided until a calcium salt can be injected intravenous to antagonize the effects of magnesium. In adults, intravenous administration of 5 to 10 mEq of 10% calcium gluconate will usually reverse respiratory depression or heart block due to magnesium toxicity. In extreme cases, peritoneal dialysis or haemodialysis may be required. Hypermagnesemia in the new born may require artificial ventilation via endotracheal intubation or intermittent positive pressure ventilation, as well as intravenous calcium.

REVIEW OF LITERATURE

1. ***Mahesh Kumar Mahala, Ramesh Chandra Sunar et al*** conducted a study "Comparison of analgesic effects of intrathecal dexmedetomidine versus magnesium sulphate as adjuvants to 0.75% isobaric ropivacaine in infraumblical surgeries". This study was designed to compare the efficacy of intrathecal dexmedetomidine and magnesium sulphate for onset and duration of sensory and motor block, duration of analgesia, post operative pain and to evaluate the side effects, if any.This hospital based interventional randomized double blind controlled study was conducted on 60 patients of ASA class I or II, 50-80 kgs of weight, 20-50 years of age, undergoing elective infraumbilical surgeries. The patients were randomly assigned into either group using a sealed envelope technique. Group A received 3 ml of 0.75% isobaric ropivacaine + 10mcg of dexmedetomidine in 0.5ml NS and Group B received 3 ml of 0.75% isobaric ropivacaine + 75mg of magnesium sulphate in 0.5ml NS.

The mean time to achieve T10 sensory level (onset of sensory block) of group A was 4.85 min, while that of group B was 6.52 min. This difference was statistically significant between two study groups ($p<0.001$).

The mean time to onset of motor block in group A was 9.93 min, while in group B was 12.11 min. This difference was statistically significant ($p<0.001$).

The mean time for two segment regression in group A was 252.0 min, while in group B was 175.80 min which was statistically significant ($p<0.001$).

The mean duration of motor block in group A was 226.03 min ,while in group B was 171.17 min which was statistically significant($p<0.001$).

The mean duration of analgesia in group A was 390.17 min, while in group B was 199.27 min which was statistically significant ($p <0.001$).

The mean VAS score was found to be lowest in Group A. This difference in VAS score between two groups was found to be statistically significant at all times from 90 minutes to 210 minutes. They concluded that both dexmedetomidine (10 mcg) and magnesium sulfate (75 mg) were effective and safe as adjuvants to isobaric ropivacaine when given intrathecally in patients undergoing infra umbilical surgeries .Dexmedetomidine appeared to be better in terms of earlier onset, prolonged sensory and motor block with prolongation of the duration of analgesia when compared to magnesium sulphate.

2. ***Dr Vani. K. Venu et al*** conducted a study "Comparison of intrathecal 0.75% isobaric ropivacaine and 0.75% isobaric ropivacaine with dexmedetomidine, for below umbilical surgeries in adults". In their study 34 patients were divided into 2 groups according to the drugs they received, Group R: received 2.5ml volume of 0.75% isobaric ropivacaine and 0.5ml normal saline(n=17). Group D: received 2.5ml volume of 0.75% isobaric ropivacaine and 5µg dexmedetomidine in 0.5ml normal saline (n=17). Onset of analgesia, highest sensory level obtained, time for maxiumum sensory block , onset and duration of motor blockade, duration of analgesia, hemodynamic stability and side effects were observed in both the groups. The mean Duration of motor block in Group R was 104 ± 12.1 min and in Group D was 182.9 ± 18.4 min,

addition of dexmedetomidine prolongs the duration of motor block about 80 min.Time to two segment regression: in Group R it was 68.2 ± 9.5 min and in Group D it was significantly prolonged to 112.4 ± 6.6 min. The heart rate, mean arterial pressure remained stable both during the intraoperative and postoperative period. They concluded that the addition of 5ug dexmedetomidine to 0.75% ropivacaine results in increased duration of analgesia and motor blockade compared to plain ropivacaine.

3. ***Vijayanand Kannabiran, Pushpa Rani Anand et al*** conducted a study "Comparison of the duration of analgesia, duration of sensory and motor blockade and incidence of side effects of intrathecal 0.75% isobaric ropivacaine with combination of 0.75% isobaric ropivacaine and dexmedetomidine". In their study, 100 patients were divided into two following groups randomly by lot method. Group R: received 3ml volume of 0.75% isobaric ropivacaine and 0.5ml normal saline. Group D: received 3ml volume of 0.75% isobaric ropivacaine and 5μg dexmedetomidine in 0.5ml normal saline. The following observations were made: Time to achieve maximum sensory block in minutes , time to two segment regression from highest sensory level in minutes, total duration of motor blockade in minutes, total duration of analgesia in minutes , highest VAS score and incidence of side effects. The mean duration of analgesia was 204.7± 20.61minutes in Group R and 430.9± 33.08 minutes in Group D. There was statistically significant difference among two groups in the mean duration of analgesia ($P<0.05$). The mean time to attain highest sensory block was 8.18± 1.79 minutes in Group R and 5.52±2.15 minutes in Group D. There was a

significant difference among two groups in the time to attain highest sensory block (P<0.05). The mean time for two segment regression was 96 ± 4.94minutes in Group R and 134 ± 6.06minutes in Group D. There was a significant difference among two groups in the duration two segment regression (P<0.05). The mean duration of motor blockade was 144.06 ± 18.75 minutes in Group R and 271.46 ± 33.40 minutes in Group D. The heart rate, mean arterial pressure remained stable both during the introperative and postoperative period in both the groups. They concluded that the addition of 5μg Dexmedetomidine to 0.75% Ropivacaine significantly prolonged the duration of analgesia, and the time to demand rescue analgesia.

4. ***Deepika Shukla, Anil Verma et al*** conducted a study "Comparative study of intrathecal dexmedetomidine with intrathecal magnesium sulphate used as adjuvants to bupivacaine".

A total of 90 patients classified as American Society of Anesthesiologists status I and II scheduled for lower abdominal and lower limb procedures were prospectively studied. Patients were randomly allocated to receive intrathecally either 15 mg hyperbaric bupivacaine plus 0.1 ml (10 μg) dexmedetomidine (group D, n =30) or 15 mg hyperbaric bupivacaine plus 0.1 ml (50 mg) magnesium sulphate (group M**,** n =30) or 15 mg hyperbaric bupivacaine plus 0.1 ml saline (group C, n =30) as control. The onset time of block, both sensory up to T10 dermatome and motor to bromage 3 scale, was rapid in group D (2.27 ± 1.09 and 3.96 ± 0.92) and delayed in the group M (6.46 ± 1.33 and 7.18 ± 1.38) in comparison with the control group C (4.14 ± 1.06 and 4.81 ± 1.03). The

regression time of block, both sensory up to T10 dermatome and motor to bromage 3 scale, was prolonged in the group D (352 ± 45 and 331 ± 35) and in the group M (265 ± 65 and 251 ± 51) when compared with the control group C (194 ± 55 and 140 ± 34). However,the duration was longest in the group D among the three groups. There was no significant difference in the mean values of heart rate and mean arterial pressures in the first hour after performing the spinal anesthesia and the first hour in the PACU between the three groups.They concluded that intrathecal dexmedetomidine supplementation of spinal block seems to be a good alternative to intrathecal magnesium as it produces earlier onset and prolonged duration of sensory and motor block without associated significant hemodynamic alterations. 10 micrograms of dexmedetomidine as adjuvant to spinal bupivacaine in surgical procedures of long duration and has minimal side-effects, and provides excellent quality of postoperative analgesia.

5. ***Abhishek Tyagi, Ravi Prakash et al*** conducted a study "Comparative evaluation of intrathecal dexmedetomidine and magnesium sulphate as adjuvants to bupivacaine for lower abdominal and lower limb surgeries". In their study 90 patients of either sex, in the age group of 18-60 years and ASA grade I and II posted for lower abdominal and lower limb surgeries were randomly allocated into three groups of 30 each. Group B: (n= 30) received intrathecal injection of a solution containing 15 mg of 0.5% bupivacaine (hyperbaric) and 0.1 ml of normal saline. Group D: (n= 30) received intrathecal injection of a solution containing 15 mg of 0.5% Bupivacaine (hyperbaric) and 5 mcg of Dexmedetomidine. Group M: (n= 30) received intrathecal injection of a solution

of 15 mg of 0.5% bupivacaine (hyperbaric) and 50 mg of magnesium sulphate. Each one of the solutions was made to a total volume of 3.1 ml. The time of onset to reach peak sensory and motor level, the regression for sensory and motor block, hemodynamic changes and side-effects were recorded. The mean time to onset of sensory block was significantly faster in group D (5.47 ± 1.81 min) than group B (6.73 ± 1.53 min) and group M (8.8 ± 1.54 min). The mean time to onset of motor block was rapid in Group D (5.92 ± 1.48 min) and delayed in Group M (8.8 ± 1.54 min) in comparison with the control Group B (6.33 ± 1.37 min). The time taken to reach maximum sensory level in Group D was 6.8 ± 2.27 min while in Group M 9.73 ± 1.8 min ,which was statistically significant with P value < 0.001.The mean duration to S2 segment regression was significantly higher in group D (323.27 ± 21.38 min) and group M (269.53 ± 12.18 min) than group B (203.1± 12.13 min). The mean time for total duration of motor block was prolonged in Group D (287.27 ± 19.22 min) and Group M (238.53 ± 11.84 min) when compared with the control Group B (168.77 ± 9.7 min). The patients were haemodynamically stable during the surgery with statistically insignificant adverse events. They concluded that dexmedetomidine has faster onset of sensory and motor blockade with prolonged duration of analgesia than magnesium sulphate. Both can be used as effective adjuvants to intrathecal hyperbaric bupivacaine with insignificant adverse events.

6. ***Rawdaa M. Elorabya, Amira A.Nasr Awad et al*** conducted a study "Effects of intrathecal dexmedetomidine vs intrathecal magnesium sulfate as adjuvants in spinal anesthesia". This study was designed to evaluate the onset,

duration, and regression of sensory and motor block of intrathecal dexmedetomidine vs magnesium sulfate as an adjuvant to 0.5% hyperbaric bupivacaine for spinal anesthesia. 60 patients aged 21–50 years, with American Society of Anesthesiologists status I, II scheduled for elective lower abdominal and lower limb surgeries, were divided into three equal groups in a randomized-controlled manner: the control group (S: n=20) received 15mg hyperbaric bupivacaine (3 ml) and 1 ml saline, the dexmedetomidine group (DXM: n=20) received 15mg hyperbaric bupivacaine (3 ml) and 10 μg of dexmedetomidine,and the magnesium sulfate group (Mg: n=20) received 15mg hyperbaric bupivacaine (3 ml) and 50mg of magnesium sulfate. Hemodynamic variables such as heart rate, systolic and diastolic blood pressure, onset of sensory and motor block, regression time, time to first analgesic request, and adverse effects were recorded for each patient. Onset time of sensory block in group S was 5.25±0.91min in group DXM was 4.25±1.45 min and in group Mg was 5.80±1.20 min. Onset time of motor block in group S was 5.50±0.61 min in group DXM was 3.95±1.47 min and in group Mg was 5.80±1.47 min. Time for sensory regression in group S was 160.25 ±12.40 min in group DXM was 381.25 ±49.63 min and in group Mg was 315.75 ±54.00 min. According to the time of first request of analgesia, the requirement for analgesia was significantly delayed in the DXM group in comparison with the Mg and S groups. They concluded that use of dexmedetomidine as an adjuvant to spinal bupivacaine in lower limb and lower abdominal surgeries is a good alternative to intrathecal magnesium sulphate as it produces earlier onset and prolonged duration of sensory and

motor block and provides excellent quality of postoperative analgesia.

7. ***Kavita Jain, Surendra K. Sethi et al*** conducted a study "Comparison of efficacy of intrathecal dexmedetomidine and magnesium sulfate as an adjuvant to 0.5% hyperbaric bupivacaine in patients undergoing infraumbilical surgeries under spinal anesthesia".

120 patients belonging to American Society of Anesthesiologists (ASA) physical status I or II aged 18 to 65 years of either sex were enrolled and randomly allocated into two groups. Group D (n = 60) received intrathecal bupivacaine (hyperbaric) 0.5% 12.5 mg (2.5 ml)+ dexmedetomidine 5 µg (in 0.5 ml NS) while Group M (n = 60) received bupivacaine (hyperbaric) 0.5% 12.5 mg (2.5 ml) + magnesium sulfate 75 mg (in 0.5ml NS) = 3 ml. Onset of sensory and motor block, time to reach peak level of sensory block, time to two segment regression, duration of sensory and motor block, duration of analgesia, sedation score, hemodynamic changes, and side effects were noted.

Onset of sensory and motor block were significantly faster in Group D (2.78 ± 0.34 min and 3.73 ± 0.43 min) compared to Group M (6.47 ± 0.43 min and 7.72 ± 0.48 min) ; ($P < 0.05$) . The maximum sensory level achieved in group dexmedetomidine was T6 and in group magnesium sulfate was also T6 which was statistically insignificant. Group D (131.70 ± 5.74 min) showed significantly prolonged time to two segment regression compared to Group M (102.78 ± 6.54 min); ($P < 0.001$). Duration of sensory and motor block were significantly prolonged in Group D (339.75 ± 23.57 min and 314.38 ± 14.93 min) when compared to Group M (248.18 ± 12.89 min and 228.81 ± 11.01 min);

(P < 0.05). Duration of analgesia was significantly prolonged in Group D (348.26 ± 22.35 min) than Group M (268.01 ± 11.31 min); (P < 0.001). The patients remained hemodynamically stable in both groups without undue sedation and minimal side effects; (P > 0.05). They concluded that dexmedetomidine (5 μg) leads to faster onset as well as prolonged duration of both sensory and motor block, prolonged duration of postoperative analgesia in comparison to magnesium sulfate (75 mg).

8. ***Sunil BV, Sahana KS et al*** conducted a study "Comparison of dexmedetomidine and magnesium sulfate as adjuvants with hyperbaric bupivacaine for spinal anesthesia : a double blind controlled study". The purpose of this study was to evaluate the onset and duration of sensory and motor block as well as adverse effects of adding dexmedetomidine or magnesium to hyperbaric bupivacaine for spinal anesthesia . 90 patients were enrolled in the study , group B, group M and group D consisted of 30 patients each. Patients allocated to group B received 3 ml hyperbaric 0.5% bupivacaine 15 mg + 0.5 ml of preservative free normal saline .Patients allocated to group D received 3 ml hyperbaric 0.5% bupivacaine 15 mg + 0.5 ml of normal saline containing 10 μg dexmedetomidine. Patients allocated to group M received hyperbaric 0.5% bupivacaine 15 mg + 0.5 ml preservative free normal saline containing 50 mg magnesium sulphate.

Time to reach T10, time to bromage 3, time for regression to L1 dermatome, hemodynamic parameters and side effects were noted. Time to reach sensory block at T10 in group B was 4.15±1.14 min, in group M was 6.46±1.32

min, in group D was 3.27±0.86 min ($p < 0.05$). Time to Bromage 3 in group B was 4.81±1.30 min ,in group M was 7.38±1.21 min, in group D was 3.54±1.03 min ($P < 0.05$).

Time for regression to L1 in group B was 160.5±21.9 min ,in group M was 236.6+34.5 min, in group D was 345.2±43.5 min ($P < 0.001$). Time to Bromage 0 in group B was 153.7±22.5 min in group M was 219.0+23.3 min in group D was 322.1±38.5 min ($P < 0.001$).The time to reach T10 sensory dermatome, Bromage 3 motor block and regression of the sensory block to L1 dermatome and motor block to Bromage scale 0 were statistically significant between group D, group M and group B. Onset of sensory block and time to Bromage 3 was rapid in group D whereas onset of both sensory and motor block was delayed in group M which is highly significant ($p < 0.001$) when compared to group D. Regression of sensory block to L1 dermatome and motor block to Bromage 0 was highly significant ($p<0.001$) between three study group, haemodynamic stability was maintained in all the three study groups **.**They concluded that, addition of dexmedetomidine prolonged the sensory and motor block significantly when used with hyperbaric bupivacaine intrathecally, without increasing the incidence of significant adverse effects. We support the addition of dexmedetomidine 10 µg with bupivacaine in spinal anesthesia when prolongation of spinal anesthesia is desired.

9.***Zameer Farooq , Neha Gupta et al*** conducted a study " Magnesium sulphate and dexmedetomidine used intrathecally as adjuvant to bupivacaine: a study**".** This study was conducted to evaluate the onset of sensory and motor

block, maximum duration of sensory and motor block, highest sensory level and bromage level achieved and side effects induced by dexmedetomidine and magnesium sulphate when given intrathecally with 0.5% hyperbaric bupivacaine for spinal anaesthesia .90 patients, of ASA grade I and grade II, age 18-65 years, of either gender, height 150 cm and above and weight 50 kg to 80 kg, scheduled for lower abdominal and lower limb surgery under spinal anaesthesia. They were randomly assigned according to table of randomization into three groups, 30 patients in each group. Patients in Group A were given 15 mg hyperbaric Bupivacaine and 10 μg (0.1 ml) Dexmedetomidine and those in Group B were given 15 mg hyperbaric Bupivacaine and 50 mg (0.1 ml) magnesium sulphate. Patients in Group C were given 15 mg hyperbaric Bupivacaine and normal saline (0.1 ml) as control. Onset of sensory block in group dexmed was rapid (2.27 ± 0.69 min), and level (dermatome) of block, at the time of onset of sensory block, was higher in group dexmed as compared to other two groups, and comparable in group magnesium and group control. Onset of motor block was rapid (3.60 ± 1.22 min), and bromage grade was higher in group dexmed at the time of onset of motor block. On the other hand, magnesium delayed the onset of both sensory (3.40 ± 1.30 min) and motor block (4.87 ± 1.36 min). Maximum sensory level achieved in group dexmed was T5 while in group magnesium sulfate was T6 which was statistically insignificant The highest sensory level achieved was comparable to group dexmed. They concluded that intrathecal dexmedetomidine as adjuvant to spinal block seems to be superior than intrathecal magnesium sulphate as it produces earlier onset and peak sensory block without associated

significant haemodynamic alterations.

10. ***Alka Shah, Ila Patel et al*** conducted a study "Haemodynamic effects of intrathecal dexmedetomidine added to ropivacaine intraoperatively and for postoperative analgesia".

This study was conducted on 50 ASA 1 and 2 planned for lower limb and lower abdomen surgery. 50 patients of ASA 1 and 2 scheduled for lower limb and lower abdominal surgery were selected. Each patient received 4 ml volume of 0.75% isobaric ropivacaine + 5 microgram dexmedetomidine. At the intervals of 1 minute, 2 minute, 5 minute, 10 minute, 20 minute, 30 minute and 1 hour, 2 hour and 3 hour reading of pulse rate and blood pressure were recorded. Postoperatively, pain scores were recorded by using Visual Analogue Scale between 0 and 10 (0= no pain, 10 = the most severe pain) initially every 1 hour for 2 hours, then every 2 hours for next eight hours and then after every 4 hours till 24 hours. Injection diclofenac 75 milligram intramuscular was given as rescue analgesia when Visual Analogue Score was 4 or more. Pulse rate at 1 minute (base line) 94.4 ± 2.15 ,2 minute 98.5 ± 4.16 ; 5 minute 88.44 ± 4.52 ;10 minute85.96 ± 1.71 ; 20 minute 83.12 ± 2.10 ; 30 minute 82.60 ± 1.97 ; 1 hour 80.64 ± 3.05 ; 2 hour ;82.20 ± 3.50 ; 3 hour 82.12 ± 5.15.There are no significant changes in systolic blood pressure after induction 1 minute (base line) 111 ± 5.74; 2 minute 110 ± 4.10; 5 minute 98.24 ± 4.74; 10 minute 94.08 ± 5.80; 20 minute 100.24 ± 4.37; 30 minute 103.2 ± 4.32; 1 hour 103.3 ± 2.32; 2 hour 105.33 ± 4.42 ; 3 hour 106.56 ± 3.80. There are no significant changes in diastolic blood pressure after induction 1 minute (base line) 74. 56 ± 6.12 ;2

minute 72.4 ± 3.1; 5 minute 68.8 ± 5.80;10 minute 65.36 ± 4.92; 20 minute 66.56 ± 3 ;30 minute 69.56 ± 3.89; 1 hour 70 ± 4.86;2 hour 71 .36 ± 3.72 ; 3 hour 72.8 ± 4.35.

Onset of sensory block (minute) 4.8 ± 1.2 , time to achieve maximum block (minute) 11.7 ± 1.7, time of two segment regression from highest sensory level (minute) 125.6 ± 16.5, time of regression to S2 (minute) 468.3 ± 36.8, time of rescue analgesia (minute) 478.4 ± 20.9, highest pain score on visual analogue score (0-10) 4.4 ± 1.4. Number of diclofenac injection in first 24 hour postoperatively 0.97 ± 0.19. The combination of ropivacaine + dexmedetomidine has negligible side effects. They concluded, 5 microgram dexmedetomidine seems to be an attractive alternative as an adjuvant to spinal ropivacaine in surgical procedures, especially those requiring long time. This combination (ropivacaine and dexmedetomidine) provides very good quality of haemodynamic stability. It has excellent quality of postoperative analgesia with minimal side effects.

11. ***R Srinivasan , R Selvarajan et al*** conducted a study "Clinical effects of intrathecal ropivacaine and ropivacaine with dexmedetomidine in inguinal hernia cases". 58 patients were divided into 2 groups. Group 1 administered with 0.75% isobaric ropivacaine 3 ml + 0.5 ml normal saline and Group 2 administered with 0.75% isobaric ropivacaine 3 ml + 5 mg dexmedetomidine in 0.5 ml normal saline. SBP, DBP, PR were observed ,time for onset of sensory block at T10 level, motor block (time to bromage 2), 2 segment regression time, regression to S2 dermatome ,totsl duration of motor block , total doses of rescue

analgesics needed were also noted.There was no significant difference in PR,SBP,DBP between the two groups throughout the procedure. The onset of sensory block at T10 level in group 1 was 8.0±1.8 min and in group 2 was 5.58±3.56 min (P<0.0001). Motor block (bromage 2) in group 1 was 10.14±5.2 min and in group 2 was 5.37±3.6 min (P<0.0001). 2 segment regression in group 1 was 89.0±18.2 min and in group 2 was 131.7±11.4 min (P <0.0001). Time for regression to S2 dermatome was 243.1±20.2min in group 1 and 297.9±25.3 min in group 2 (P <0.0001). Time taken for first rescue analgesia was 217.2±17.5 min in group 1 and 453.1±20.2 min group 2 (P<0.0001). They concluded that ropivacaine is an ideal, comfortable safe drug of choice for intrathecal use in inguinal hernia surgery cases, by adding dexmedetomidine, for prolongation of analgesia.

MATERIALS AND METHODS

The study was conducted at Thanjavur medical college between 2019-2020. After obtaining Ethical Committee approval ,50 ASA I- II patients undergoing infraumblical surgeries were randomly allotted into two groups.

Study design :

A prospective randomized double-blinded study.

Sample size :

50 patients were selected and randomly divided into two groups GROUP 'D' and GROUP 'M' .

Group D : 25 patients received 3 ml of 0.75% isobaric ropivacaine hydrochloride with 10µg of dexmedetomidine in 0.5 ml Normal Saline.

Group M : 25 patients received 3 ml of 0.75% isobaric ropivacaine hydrochloride with 75 mg of MgSO4 in 0.5 ml Normal Saline.

INCLUSION CRITERIA

1. Patients of age 20 to 65 years of either sex.
2. ASA grade I and II.
3. Patients undergoing infraumblical surgeries under spinal anaesthesia.

EXCLUSION CRITERIA

1. Patient's unwillingness.
2. Patients with clinically significant cardiovascular, respiratory, hepatic, renal, neurological, psychiatric and metabolic diseases.
3. Patients with coagulation disorders, any life-threatening disease, signs of sepsis, previous injury, deformity or previous surgery of spine, anticipated difficulty in regional anesthesia.
4. Allergy to study drugs , pregnancy, and lactation.

MATERIALS USED :

- 23 G spinal needle
- 2 ml,5 ml, sterile syringes
- Hypodermic needles – 18G & 26G
- 18G venflon and intravenous fluids
- Sterile Bowl, sponge holding forceps, gauze, povidone iodine solution.
- Sterile gown, gloves
- Local anaesthetic solution – 2% lignocaine
- 0.75% ropivacaine 4 ml vial, dexmedetomedine 100 µg 1ml ampoule,magnesium sulphate 2 g – 2 ml ampoule.

- Emergency drugs like Inj.Adrenaline, Inj.Atropine, Inj.Ephedrine, Inj.Dopamine, Inj.Thiopentone, Inj.Succinylcholine.
- Emergency resuscitation equipments - working laryngoscope, cuffed endotracheal tubes of appropriate size, airway, suction apparatus with suction catheter.
- Multipara monitor for monitoring of pulse rate, non-invasive blood pressure, electrocardiogram ,oxygen saturation.

METHODOLOGY

Preoperative preparation :

Thorough preanesthetic checkup of all patients including all routine investigations was done. The procedure was explained to the patient and written informed consent was taken. Pain visual analog scale (VAS) scores were explained to all patients.

Premedication was given as tablet alprazolam 0.25 mg a night before surgery, injection glycopyrrolate 0.2 mg, and injection midazolam 0.04 mg/kg body weight by intravenous route just before the procedure in the preop room.

Preoperatively, pulse rate and noninvasive systolic and diastolic blood pressure of the patients were recorded. In the operation theater, intravenous line was secured with 18-gauge intricate, and all the patients were preloaded with 10 ml/kg body weight of Ringer lactate solution over 15 to 20 min. Multipara monitors were connected, and baseline pulse rate, noninvasive systolic and

diastolic blood pressure, oxygen saturation (SpO_2), and electrocardiogram (ECG) were recorded. also. Oxygen was routinely administered through oxygen mask at the rate of 5 L/min.

Procedure :

Patients were put in the lateral decubitus position. After scrubbing, washing and wearing sterile gown and gloves, back of the patient was cleaned with povidone-iodine scrub and then painted with povidone-iodine solution. The area was draped with sterile sheet. L3 and L4 space was located . Skin wheal was raised with 2% lignocaine and then with midline approach a 23-gauge spinal needle was inserted in the space. After the free flow of cerebrospinal fluid, drug was injected in the space.

The following parameters were observed :

1.Hemodynamic parameters :

Continuous multipara monitoring (pulse rate, respiratory rate, noninvasive systolic and diastolic blood pressure, SpO_2, and ECG) was done for hemodynamic response. Readings were recorded preoperatively, intra-operatively every 3 min for the first 15 min, and thereafter every 15 min till the end of surgery in both the groups.

2.Time to onset of analgesia at T10

Time interval between the end of administration of drug and the onset of sensory block to T10 level which was evaluated by eliciting pin prick test every minute till complete sensory block to T10.

3. Maximum sensory level achieved :

Sensory block was assessed by the loss of sensation to pinprick in the midline using a 22-gauge blunt hypodermic needle every 3 min interval till no change in level occurred.

4.Time to achieve the maximum sensory level.

The time taken to achieve maximum level was noted.

5.Mean time to regression to L1 dermatome.

The time taken for the sensory level to recede by L1 dermatome from the maximum sensory level which was evaluated by eliciting pin prick test.

6.Time to onset of motor block :

The degree of motor block was assessed every 3 min for first 30 min by the modified Bromage scale.

0 = No motor blockade

1 = Inability to raise extended leg but can flex knee

2 = Inability to flex knee, can flex ankle

3 = No movement, unable to flex ankle joint.

7.Maximum Bromage scale achieved.

8.Total duration of motor block.

Duration of motor block was recorded from onset time to time when the patient was able to lift the extended leg.

9. Total doses of rescue analgesics in 24 hours :

After completion of surgery, patient were monitored postoperatively for sensory block, motor block and analgesia (according to VAS) every 30 min for 1 h and then hourly till first rescue analgesia was given in the form injection tramadol 50–100 mg intravenously when VAS >3.

Bradycardia (defined as heart rate <60 beats/min) was treated with intravenous atropine 0.5 mg. Hypotension (defined as systolic blood pressure <20% of baseline value) was treated with intravenous ephedrine as per required and additional Ringer's lactate solution.

In case of failed neuraxial block and total spinal, patient was given general anesthesia and the case was excluded from the study.

OBSERVATIONS AND RESULTS

Statistical analysis:

The statistical analyses were performed using SPSS version 21. Data were presented as mean with Standard deviation for normal distribution (Age, Heart rate, blood pressure and various time durations). Data were presented as frequency with proportion (%) for categorical data (Type of surgery, maximum sensory level etc.,). Unpaired 't' test was used to compare the means following between dexmedetomidine and $MgSO_4$ group. Chi Square test (Fisher's exact test)was used to compare the categorical variables between the groups. $p<0.05$ and $p<0.0001$ were considered statistically significant.

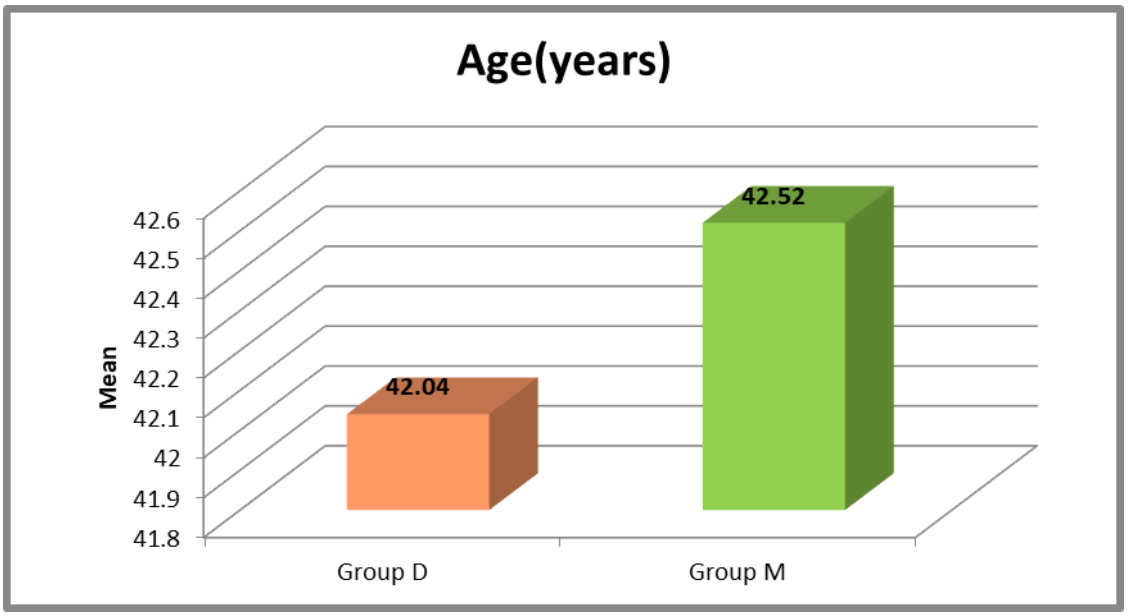
AGE DISTRUBUTION

Table 1: Comparison of age in years between the group D and group M in the study (Unpaired t test)

S. No	Parameter	Group D(N= 25) (Mean±SD)	Group M(N=25) (Mean±SD)	P value	Statistical test
1	Age (years)	42.04 ±10.16	42.52± 11.68	0.877	Unpaired 't' test

The mean age in average years was 42.04 ± 10.16 (years) in group D and 42.52 ± 11.68 (years) in group M . There was statistically no significant difference between two groups($p>0.05$)

Fig 1: Comparison of age in years between the group D and group M in the study (Unpaired t test)



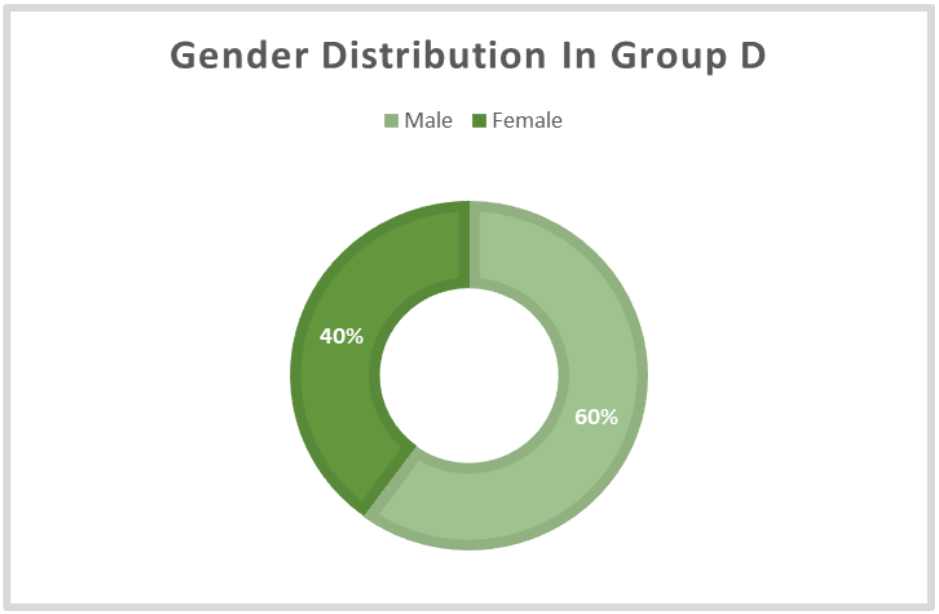
GENDER

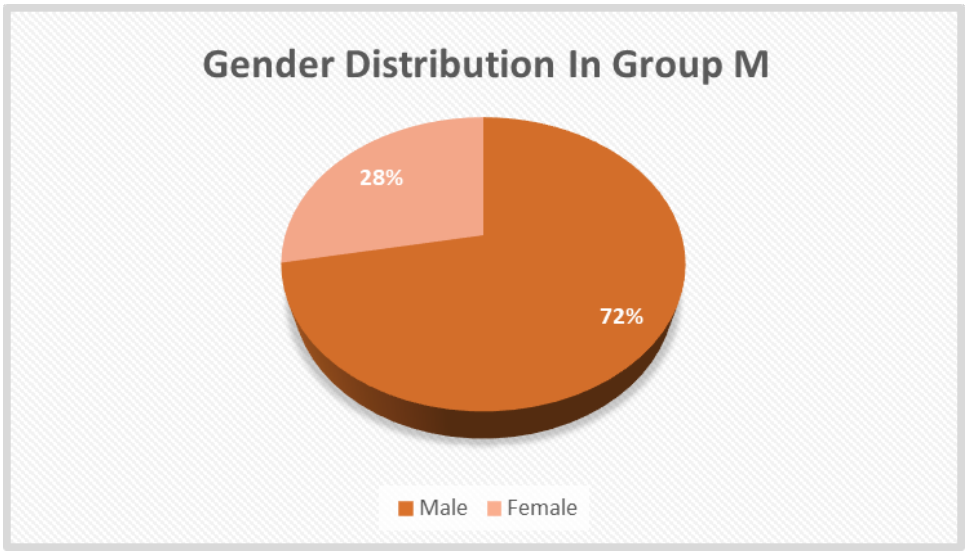
Table 2: Comparison of gender ratio between the Group D and group M in the study

S. No	Gender	Group D (N= 25) (%)	Group M (N=25) (%)	P value
1	Male	15 (60)	18(72)	0.551
2	Female	10 (40)	7 (28)	
Total		25	25	

There was statistically no significant association in sex distribution between two groups (p>0.05).

Fig 2: Showing gender distribution of the study participants in both the groups





SENSORY BLOCK CHARACTERISTICS:

Table 4: showing sensory block characteristics :

Parameters	Group D(N=25)	Group M (N=25)	P value
Mean Onset time of sensory block in minutes to T10 level	4.440±0.50	6.680±0.69	**<0.001**
Maximum sensory level achieved	T5	T6	0.675
Time to achieve maximum sensory level (min)	8.960±0.78	12.880±0.72	**<0.001**
Mean time to regression to L1 dermatome (min)	286.0±23.97	213.30±55.31	**<0.001**

There was a statistical significant association in the mean onset time sensory block at T10 level, time to achieve maximum sensory level and mean time to regression to L1 dermatome in group D with P value < 0.001.

Table 5: Comparison of mean onset time of sensory block in minutes to T10 level

Parameter	Group D(N=25)	Group M (N=25)	P value
Mean Onset time of sensory block in minutes T10 level	4.440±0.50	6.680±0.69	**<0.001**

There was a statistical significant association in the mean onset time sensory block at T10 level in group D with P value < 0.001.

Fig 3: Comparison of mean onset time of sensory block in minutes to T10 level

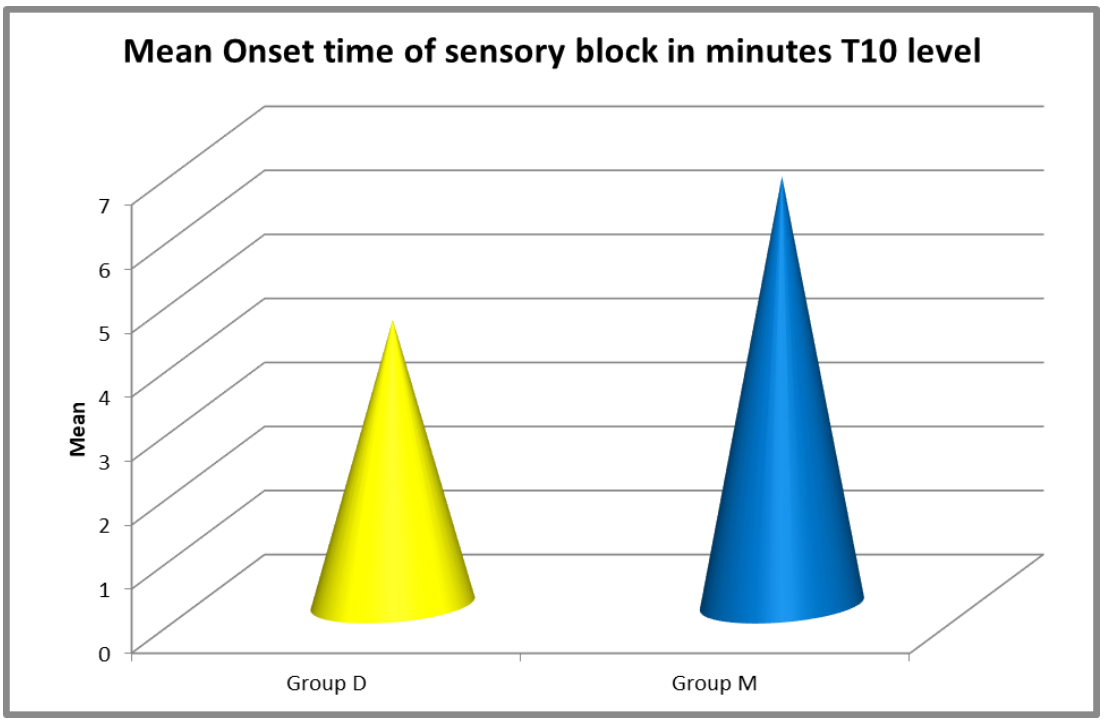


Table 6: Comparison of time taken (mins) to achieve maximum sensory level

Parameter	Group D(N=25)	Group M (N=25)	P value
Time to achieve maximum sensory level (min)	8.960±0.78	12.880±0.72	**<0.001**

There was a statistical significant association in time to achieve maximum sensory level in group D with P value < 0.001.

Fig 4: Comparison of time taken (mins) to achieve maximum sensory level

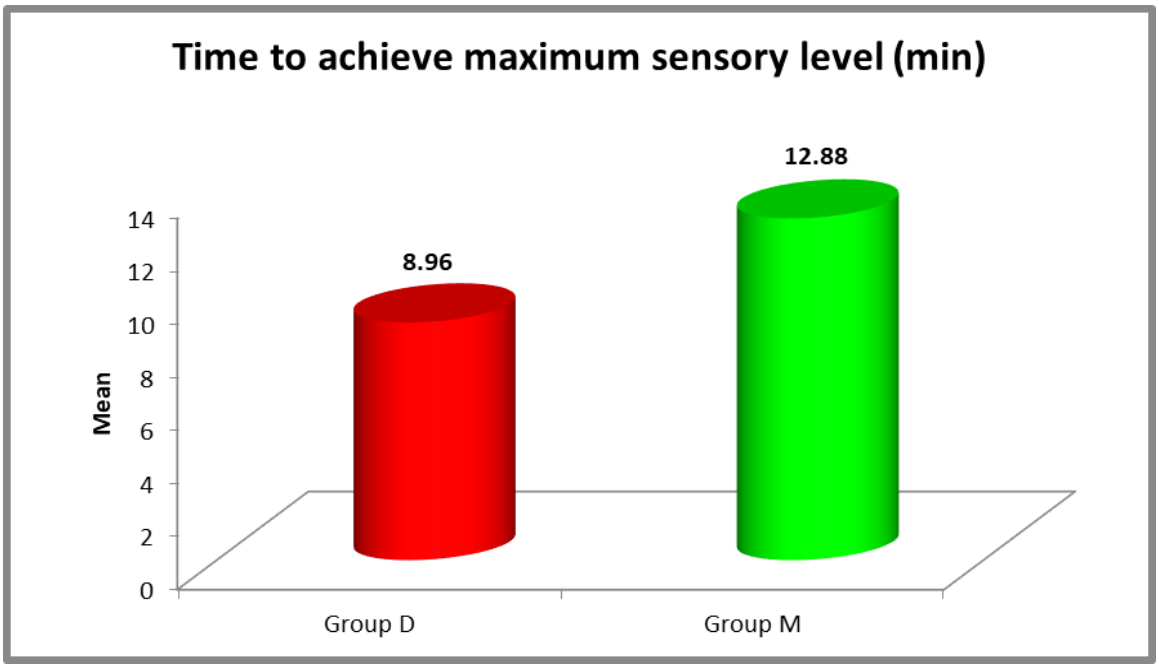
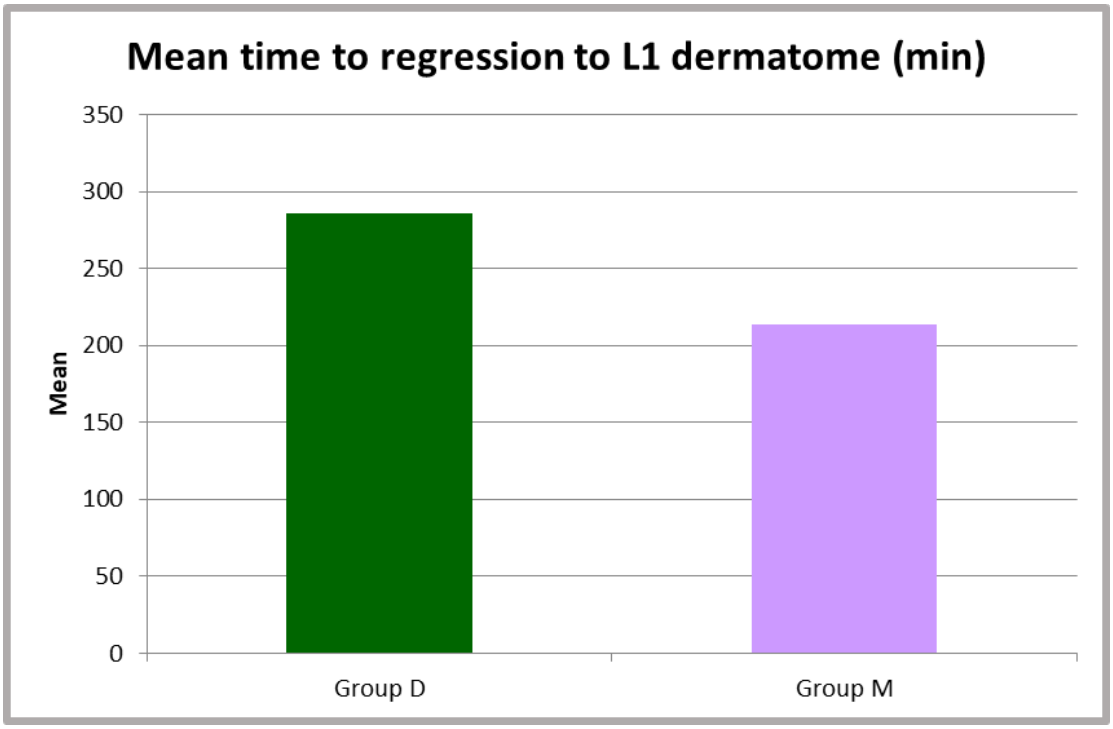


Table 7: Comparison of mean time (mins) to regression to L1 dermatome

Parameter	Group D(N=25)	Group M (N=25)	P value
Mean time to regression to L1 dermatome (min)	286.0±23.97	213.30±55.31	**0.001**

There was a statistical significant association mean time to regression to L1 dermatome in group D with P value < 0.001.

Fig 5: Comparison of mean time (mins) to regression to L1 dermatome



MOTOR BLOCK CHARACTERISTICS

Table 8: showing motor block characteristics :

S.No	Parameter	Group D (N= 25) (Mean±SD)	Group M(N=25) (Mean ±SD)	P value	Statistical test
1	Mean Onset time of motor block	8.40 ± 0.645	12.92 ± 1.350	0.001	Unpaired 't' test
2	Maximum bromage scale achieved	3	3	0.786	
3	Total Duration of motor block (mins)	223.60±17.29	168.20±18.30	**<0.001**	

Mean Onset time of motor block :

Table 9 : Comparison of Mean onset time of motor block between the groups in the study population. (Unpaired 't' test) :

S. No	Parameter	Group D (N= 25) (Mean±SD)	Group M(N=25) (Mean ±SD)	P value	Statistical test
1	Mean Onset time of motor block	8.40 ± 0.645	12.92 ± 1.350	0.001	Unpaired 't' test

The mean time for motor block was 8.40 ± 0.645 (minutes) in group D and 12.92 ± 1.35 (minutes) in group M. There was a statistically significant association among two groups in time for complete motor block with p<0.00.

Fig 6: Mean Onset Time Of Motor Block :

Comparison of time to achieve complete motor blockade in minutes between the groups in the study.

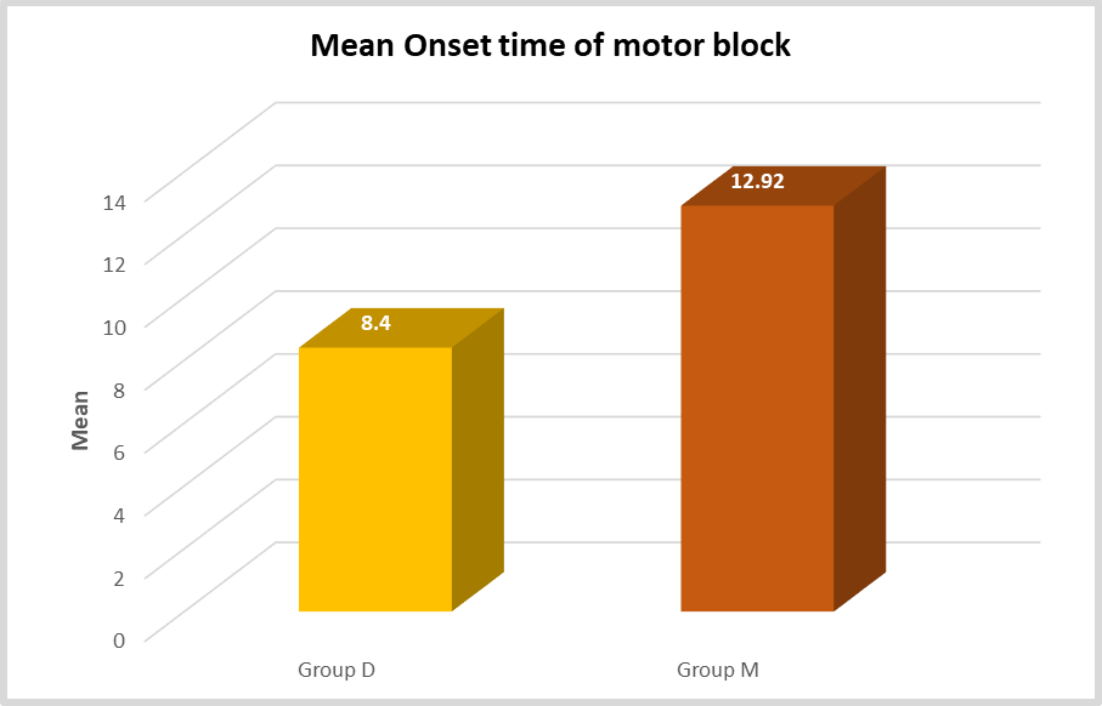


Table 10:Comparison of Total duration of motor block (mins)

Parameter	Group D(N=25)	Group M (N=25)	P value
Total Duration of motor block (mins)	223.60±17.29	168.20±18.30	**<0.001**

There was a statistical significant association in the total duration of motor block in group D with P value < 0.001.

Fig 7: Comparison of Total duration of motor block (mins)

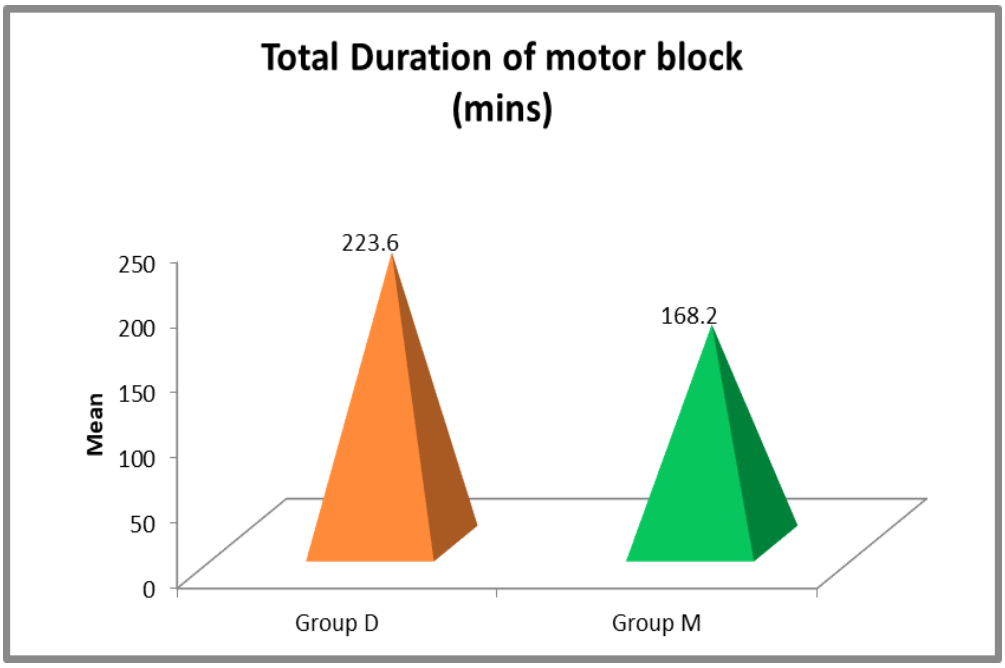


Table 11: Post operative parameters :

Parameters	Group D(N=25)	Group M (N=25)	P value
Total duration of Analgesia(mins)	381.60±30.09	223.00±18.20	**<0.001**
Total doses of tramadol in 24 hours	1.240±0.435	2.640±0.489	**<0.001**

There was a statistical significant association in the total duration of analgesia and total doses of tramadol in 24 hours in group D with P value < 0.001.

Table 12 :Comparison of total duration of analgesia (mins)

Parameter	Group D(N=25)	Group M (N=25)	P value
Total duration of Analgesia(mins)	381.60±30.09	223.00±18.20	**<0.001**

There was a statistical significant association in the total duration of analgesia in group D with P value < 0.001

Fig 8:Comparison of total duration of analgesia (mins)

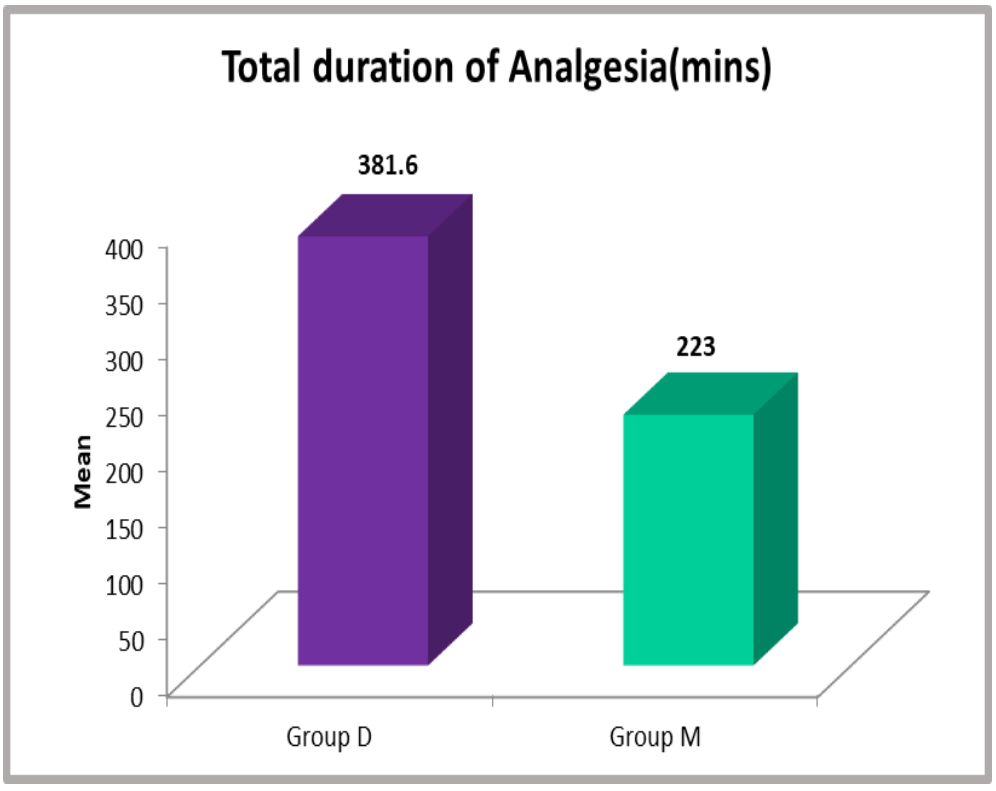
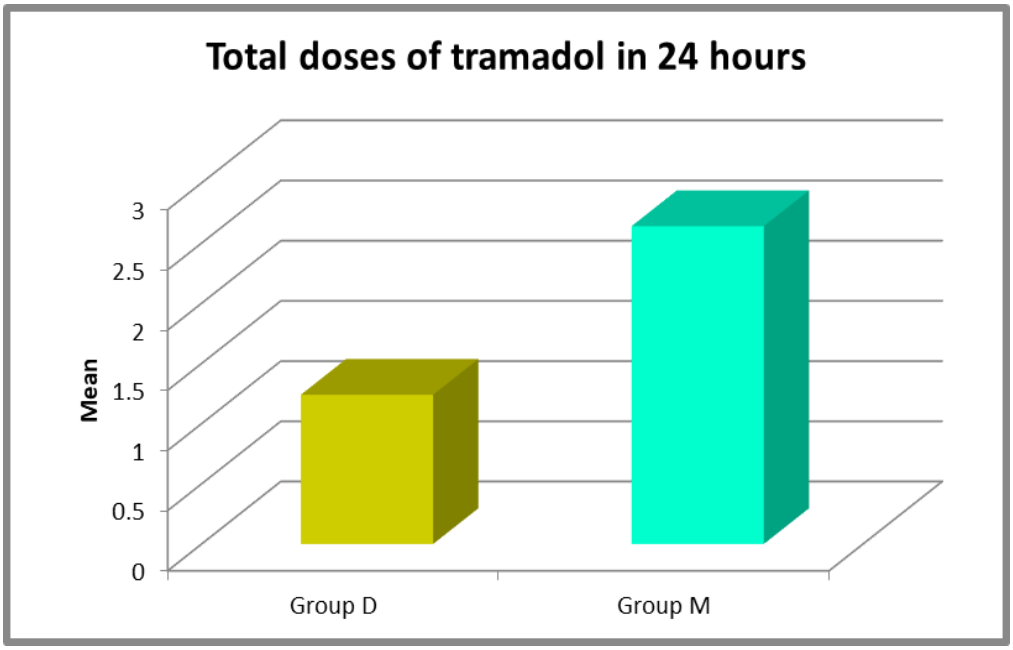


Table 13 : Comparison of total doses of tramadol in 24 hours

Parameter	Group D(N=25)	Group M (N=25)	P value
Total doses of tramadol in 24 hours	1.240±0.435	2.640±0.489	**<0.001**

There was a statistical significant association in total doses of tramadol in 24 hours in group D with P value < 0.001

Fig 9: Comparison of total doses of tramadol in 24 hours



HEART RATE

Table 14 : Comparison of heart rate (bpm) between two groups in the study population. (Unpaired 't' test)

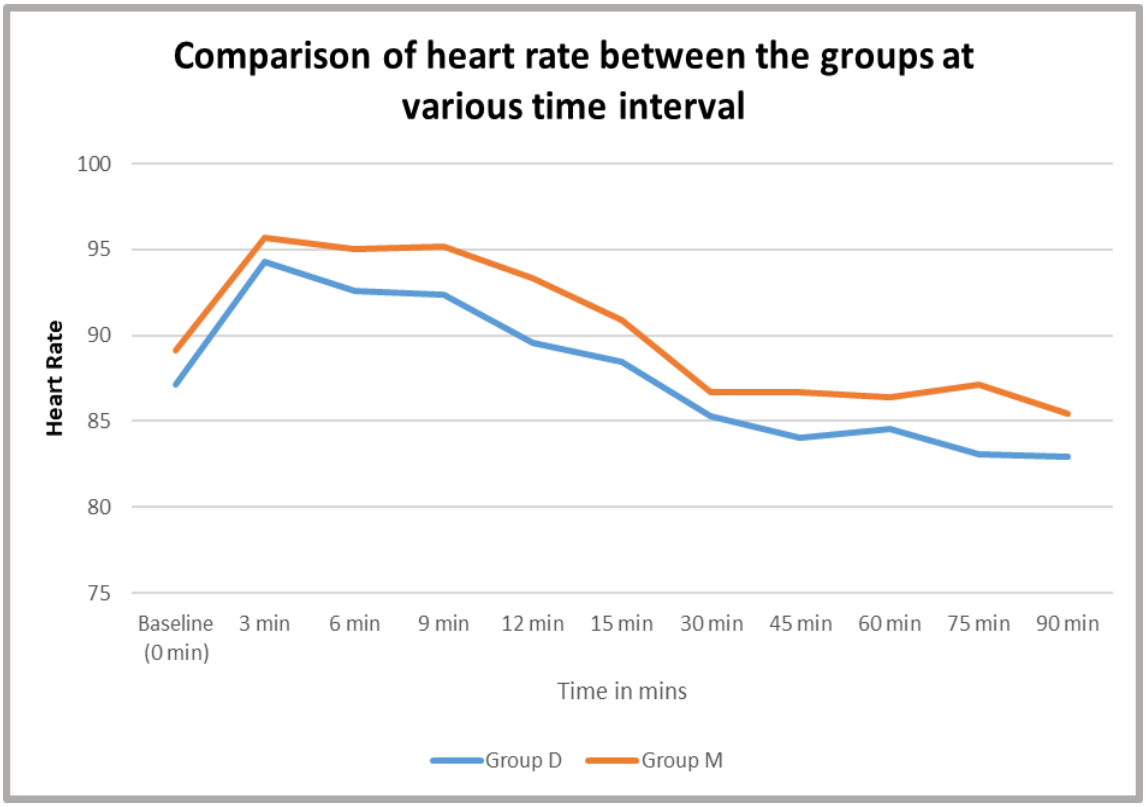
The following table shows the heart rate variations which showed statistical difference in both groups

S. No	Time (in mins)	Group D (N= 25) (Mean ±SD)	Group M (N=25) (Mean ±SD)	P value
1	Baseline (0 min)	87.16 ± 9.57	89.12 ± 8.59	0.450
2	3 min	94.32± 9.14	95.68 ± 7.65	0.571
3	6 min	92.56 ± 8.02	95.04 ± 7.16	0.255
4	9 min	92.36 ± 6.20	95.20 ± 6.42	0.118
5	12 min	89.56 ± 9.05	93.32 ± 7.20	0.111
6	15 min	88.48 ± 7.78	90.89 ± 6.39	0.255
7	30 min	85.28± 4.67	86.72 ± 5.07	0.302
8	45 min	84.04± 5.38	86.68 ± 5.21	**0.085**
9	60 min	84.58 ± 5.97	86.38 ± 6.02	0.321
10	75 min	83.05 ± 6.55	87.11 ± 4.77	**0.034**
11	90 min	82.93 ± 4.52	85.43 ± 3.75	0.121

The heart variation in between two groups was significant at 45min and 75 min P value<(0.05).

HEART RATE

Fig 10: Comparison of heart rate between the groups at various time interval between the study depicted by line-trend diagram



SYSTOLIC BLOOD PRESSURE

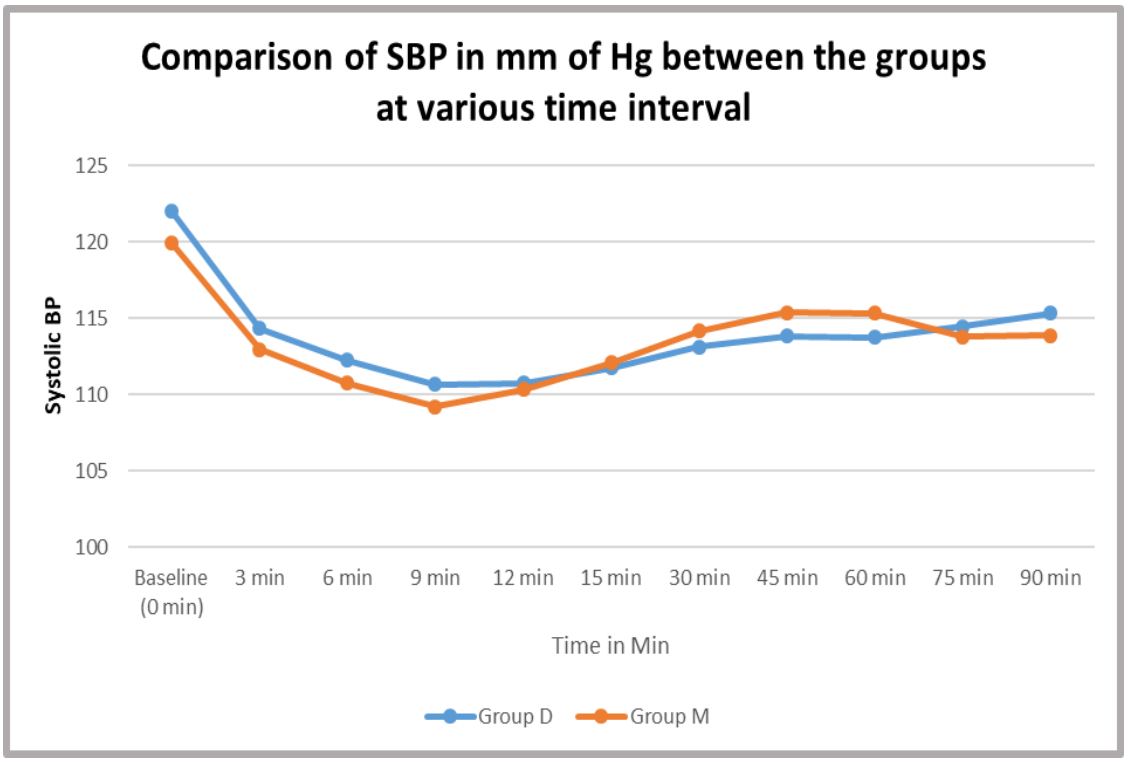
Table 15 : Comparison of systolic blood pressure (mm of Hg) between two groups in the study population.(Unpaired 't' test)

S. No	Time (in mins)	Group D (N= 25) (Mean ±SD)	Group M (N=25) (Mean ±SD)	P value
1	Baseline (0 min)	122±8.46	119.92±7.33	0.358
2	3 min	114.32±7.82	112.96±7.19	0.525
3	6 min	112.24±6.61	110.72±6.67	0.426
4	9 min	110.64±6.39	109.20±6.27	0.425
5	12 min	110.72±4.57	110.32±5.58	0.783
6	15 min	111.76±3.57	112.08±5.27	0.803
7	30 min	113.12±3.32	114.16±4.27	0.342
8	45 min	113.84±4.72	115.36±5.25	0.287
9	60 min	113.75±3.92	115.33±5.87	0.288
10	75 min	114.45±4.97	113.78±6.13	0.702
11	90 min	115.33±4.11	113.86±3.88	0.330

The systolic BP variation between two groups was not statistically significant.P value >0.05

COMPARISON OF SYSTOLIC BLOOD PRESSURE

Fig 11: Comparison of SBP in mm of Hg between the groups at various time interval between the study depicted by line-trend diagram



DIASTOLIC BLOOD PRESSURE

Table 16 : Comparison of diastolic blood pressure (mm of Hg) between two groups in the study population.(Unpaired 't' test)

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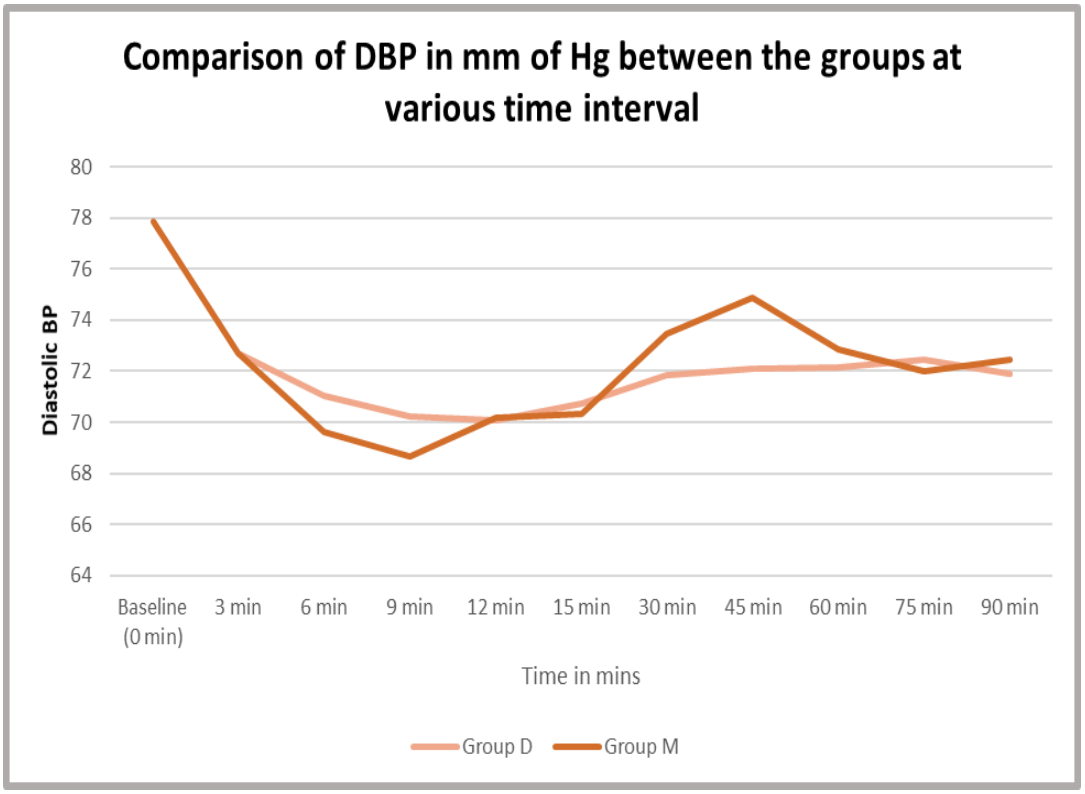
S. No	Time (in mins)	Group D (N= 25) (Mean ±SD)	Group M (N=25) (Mean ±SD)	P value
1	Baseline (0 min)	77.84±5.47	77.84±5.25	1
2	3 min	72.72±4.92	72.72±4.99	1
3	6 min	71.04±4.28	69.62±4.74	0.385
4	9 min	70.24±4.21	68.64±3.59	0.155
5	12 min	70.08±2.97	70.16±3.64	0.933
6	15 min	70.72±2.70	70.32±2.35	0.580
7	30 min	71.84±3.55	73.44±4.84	0.190
8	45 min	72.08±3.18	74.88±6.03	**0.046**
9	60 min	72.17±3.17	72.86±4.02	0.524
10	75 min	72.45±2.46	72.00±3.66	0.625
11	90 min	71.87±1.92	72.43±3.69	0.608

The diastolic BP variation between two groups was statistically significant at 45 min with (P value<0.05).

COMPARISON OF DIASTOLIC BLOOD PRESSURE

Fig 12:Comparison of DBP in mm of Hg between the groups at various time interval between the study depicted by line-trend diagram.



SIDE EFFECTS:

Table 17 : Comparison of side effects between group D and M

SIDE EFFECTS	Group D (N=25) (%)	Group M (N=25) (%)	P value
Hypotension	3(12)	3(12)	>0.05
Bradycardia	0	0	
Vomitting	0	0	

Using Chi square association between the side effects between the groups was calculated, 12 % of the patients in group D and 12% of the patients in group M had hypotension, which was statistically insignificant ($p>0.05$).

DISCUSSION

Spinal anaesthesia is simple to perform, uses small dose of drugs, offers rapid onset of action, reliable surgical anaesthesia with good muscle relaxation. These advantages are sometimes offset by a relatively short duration of action and complaints of postoperative pain when effect wears off. Spinal anaesthesia with ropivacaine hydrochloride is increasingly being used these days owing to its lack of cardiotoxicity and neurotoxicity. The efficacy of local anaesthetics can be enhanced using adjuvants such as opioids, α2 agonists, magnesium, neostigmine, ketamine. Prolongation of the duration of spinal block is desirable both for long procedures and for postoperative pain relief.

In our study, we compared the effect of intrathecally administered magnesium and dexmedetomidine with ropivacaine in terms of onset, duration and regression of sensory and motor block, hemodynamic profile as well as the side effects .

In our study Group D patients received 3 ml of 0.75% isobaric ropivacaine hydrochloride with 10 μg of dexmedetomidine in 0.5ml NS and Group M patients received 3 ml of 0.75% isobaric ropivacaine hydrochloride with 75 mg of $MgSO_4$ in 0.5 ml NS.

DEMOGRAPHIC DATA :

The patients in both groups did not show any statistically significant difference with respect to age, gender, ASA classification and type of surgery.

ONSET OF SENSORY BLOCK TO T10 LEVEL

In our study, we found out that onset of sensory block was earlier in Group D in comparison to Group M which was statistically significant. The mean onset time of sensory block at T10 level in Goup D is 4.440±0.50 min and in Group M is 4.440±0.50 min which is statistically significant with P<0.001.

This result correlated with following studies ,in the study conducted by ***Mahesh Kumar Mahala et al*** [15] **,** the addition of dexmedetomidine with ropivacaine provided early onset of sensory block at T10 level . The mean time to achieve T10 sensory level (onset of sensory block) of group dexmed was 4.85 min, while that of group magnesium sulfate was 6.52 min. This difference was statistically significant.***Deepika Shukla et al***[18] concluded that, the mean time of onset of analgesia at T10 in group dexmedetomidine was 2.27 ± 1.09 min which was faster than group magnesium sulfate 6.46 ± 1.33. ***Sunil et al'***[22] concluded that the time to reach sensory block at T10 in group plain bupivacaine was 4.15±1.14 min, in group magnesium sulfate was 6.46±1.32 min, in group dexmedetomidine was 3.27±0.86 min ($p < 0.05$) which was statistically significant.***Srinivasan et al***[25] conducted a study using dexmedetomidine as adjuvant with intrathecal ropivacaine in which, the onset of sensory block at T10 level was rapid in group dexmedetomidine 5.58±3.56 min when compared with plain ropivacaine group which was 8.0±1.8 min (P<0.0001).

TIME TO ACHIEVE MAXIMUM SENSORY LEVEL :

In our study time to achieve maximum sensory level in Group D was 8.960±0.78 min and in Group M was 12.880±0.72 min which was statistically significant with

P value < 0.001. Similar results were observed in, in a study conducted by ***Ravi Prakash et al***[19] while comparing dexmedetomidine and magnesium sulfate as adjuvants with bupivacaine, the time to taken to reach maximum sensory level in Group D (Dexmedetomidine) was 6.8 ± 2.27 min while in Group M (magnesium sulfate) 9.73 ± 1.8 ,which was statistically significant with P value< 0.001. ***Kavitha jain et al***[21] also concluded that the highest level of sensory block achieved was significantly earlier in Group D (Dexmedetomidine) 9.98 ± 0.54 min as compared to Group M (Magnesium sulfate) 17.35 ± 0.52 min with P value < 0.001.

MAXIMUM SENSORY LEVEL ACHIEVED :

In our study maximum sensory level achieved in Group D was T5 and Group M was T6 (p value 0.675) which was statistically insignificant between the two groups. Similar results were observed in the following studies ,***Farooq et al***[23] conducted a study in which maximum sensory level achieved in group dexmedetomidine was T5 while in group magnesium sulfate was T6. ***Kavitha jain et al***[21] concluded that the maximum sensory level achieved in group dexmedetomidine was T6 and in group magnesium sulfate was also T6 which were statistically insignificant.

TIME TO REGRESSION TO L1 DERMATOME :

In our study the mean time to regression to L1 dermatome (min) in Group D was 286.0±23.97 min while in group M was 235.30±55.31 min with P value < 0.001.

These results correlated with ***Sunil et al's study*** [22] in which time for regression to L1 in group B (plain bupivacaine) was 160.5±21.9 min ,in group M (magnesium sulfate) was 236.6+34.5 min, in group D (dexmedetomidine) was 345.2±43.5 min with P value <0.001.

And also ***Raviprakash et al***[19] in their study observed that the mean duration to S2 segment regression was significantly higher in group Dexmedetomidine 323.27 ± 21.38 min and group Magnesium sulfate 269.53 ± 12.18 min than group Bupivacaine 203.1± 12.13 min which was statistically significant. ***Srinivasan et al***[25] also observed that the time for regression to S2 dermatome was 243.1±20.2 min while adding dexmedetomidine with ropivacaine and 297.9±25.3 min while plain ropivacaine was used (P <0.0001) in inguinal hernia cases.

ONSET OF MOTOR BLOCK :

In our study the mean onset time of motor block in Group D was 8.40 ± 0.645 min while in Group M was 12.92 ± 1.350 min with P <0.001.Similar results were observed in in a study conducted by ***Mahesh Kumar et al***[15] in which the mean time to onset of motor block in group A (isobaric ropivacaine with dexmed) was 9.93 min, while in group B (isobaric ropivacaine with

magnesium sulfate) was 12.11 min. This difference was statistically significant ($p<0.001$).***Rawada et al***[20] conducted a study in which they concluded that the onset time of motor block in group S (plain Bupivacaine) was 5.50±0.61 min, in group DXM (Bupivacaine with Dexmedetomidine) was 3.95±1.47 min and in group Mg (Bupivacaine with Magnesium sulfate) was 5.80±1.47 min. ***Raviprakash et al***[19] also concluded that, the mean time to onset of motor block was rapid in Group D (5.92 ± 1.48 min) and delayed in Group M (8.8 ± 1.54 min) in comparison with the control Group B (6.33 ± 1.37 min).

Similar result was observed in the study conducted by ***Kavitha jain et al***[21] where the mean time for onset of motor block in Group Dexmedetomidine was 3.73 ± 0.43 min and in Group Magnesium sulphate was 7.72 ± 0.48 min where there was faster onset of motor block by adding dexmedetomidine with hyperbaric bupivacaine.

MAXIMUM BROMAGE SCALE ACHIEVED :

In our study the maximum bromage scale achieved was 3 in both the groups , which was statistically insignificant.Similar results were observed in studies conducted by ***Mahesh Kumar et al*** [15], ***Vani et al***[16] , ***Deepika et al***[18] ***and Sunil et al***[22] **.**

TOTAL DURATION OF MOTOR BLOCK :

In our study the total duration of motor block was 223.60±17.29 min in Group D and 168.20±18.30 min in Group M with P<0.001.

Similar results were observed in, the study conducted by ***Mahesh Kumar et al***[15] , the mean duration of motor block in group A was 226.03 min ,while in group B was 171.17 min which was statistically significant(p<0.001).***Vani et al***[16] also concluded that adding dexmedetomidine to isobaric ropivacaine prolonged the total duration of motor block , the total duration of motor block in Group R was 104 ± 12.1 min and in Group D was 182.9 ± 18.4 min, addition of dexmedetomidine prolongs the duration of motor block about 80 min.

Similar results were observed in the study conducted by ***Deepika Shukla et al*** [18] , that the regression time of motor block, was prolonged in the group Dexmed (331 ± 35 min). ***Kavitha jain et al***[21] observed that the duration motor block were significantly prolonged in Group Dexmedetomidine (314.38 ± 14.93 min) when compared to Group Magnesium sulfate (228.81 ± 11.01 min) (P < 0.05).Similar results were observed in studies conducted by ***sunil et al*** where addition of dexmedetomidine with hyperbaric bupivacaine prolonged the duration of motor blockade.

TOTAL DURATION OF ANALGESIA :

In our study the total duration of analgesia in Group D was 381.60±30.09 min and in Group M was 223.00±18.20 min with P value < 0.001.

Similar results were observed in the study conducted by ***Mahesh Kumar et al*** [15] The mean duration of analgesia in group A(ropivacaine with dexmedetomidine) was 390.17 min, while in group B(ropivacaine with magnesium sulfate) was 199.27 min which was statistically significant (p <0.001), the addition of dexmedetomidine with isobaric ropivacaine prolonged the duration of analgesia.***Vani et al*** [16] observed that the mean duration of analgesia was 204.7± 20.61minutes in Group R(plain ropivacaine) and 430.9±33.08 minutes in Group D(ropivacaine with dexmedetomidine). There was statistically significant difference among two groups in the mean duration of analgesia (P<0.05).***Kavitha jain et al***[21] also concluded that adding dexmedetomidine with hyperbaric bupivacaine prolonged the total duration of analgesia.The total duration of analgesia was significantly prolonged in Group D (348.26 ± 22.35 min) than Group M (268.01 ± 11.31 min) (P < 0.001).

TOTAL DOSES OF RESCUE ANALGESICS (INJ.TRAMADOL) IN 24 HOURS :

In our study the total number of doses of tramadol required in Group D was 1.240±0.435 and in Group M was 2.640±0.489 with P < 0.001.

The total number of doses of rescue analgesics required was less in Group D.

Similar results were observed in the studies conducted by ***Rawadaa et al***[20] and ***Srinivasan et al***[25] where there was lesser requirements of rescue analgesics in 24 hrs ,while using dexmedetomidine as an adjuvant .

HEMODYNAMIC STABILITY :

There was no much difference between the two groups in terms of heart rate , systolic BP and Diastolic BP. The heart rate variation between two groups were significant at 45 min and 75 min but were comparable. The systolic BP variation between two groups was not statistically significant, P value >0.05.The diastolic BP variation between two groups was statistically significant at 45 min and is comparable. Significant hypotension and bradycardia were not observed and hemodynamic stability was maintained in both the groups and, which correlated with the studies conducted by ***Alka shah et al***[24] and ***Deepika Shukla et al***[19]**.**

SIDE EFFECTS :

In our study even though hypotension was observed in 12 % of the patients in both the groups, it was found to be clinically and statistically insignificant (p>0.05). There were no reports of bradycardia and vomiting in both the groups.Similar results were observed in studies conducted by ***Raviprakash et al***[19] and ***Rawadaa et al***[20] ,as they concluded that addition of dexmedetomidine did not produce any significant side effects.

SUMMARY

Double blinded prospective randomized study was done to evaluate the efficacy of intrathecally administered dexmedetomidine and magnesium sulfate to isobaric ropivacaine in 50 patients belonging to ASA I and II ,aged between 20 to 65 years undergoing infraumbilical surgeries.

Written informed consent was taken and pre anaesthetic evaluation was done .

All cases received 3 ml of 0.75% isobaric ropivacaine with either 10 microgram of dexmedetomidine (Group D) or 75 mg of magnesium sulfate (Group M) in 0.5ml normal saline

In the intra operative period following parameters were observed :

Time of onset of sensory block.

Time of onset of motor block.

Time to achieve the maximum sensory level.

Duration of analgesia.

Mean time to regression to L1 dermatome.

Maximum Bromage scale achieved.

Total duration of motor block.

Hemodynamic parameters (HR, SBP ,DBP).

Side effects.

The following observations were made :

1. Addition of dexmedetomidine as adjuvant to intrathecal ropivacaine produced early onset of sensory and motor block when compared to magnesium sulfate.
2. Time taken to reach maximum sensory level was shorter in dexmedetomidine group (Group D) compared to magnesium sulfate group (Group M).
3. The maximum Bromage scale achieved was 3 in both the groups.
4. The maximum sensory level achieved in Group D was T5 while in Group M was T6.
5. Clinically significant bradycardia and hypotension were not observed in both the groups.
6. The mean time for regression to L1 dermatome was prolonged in Group D compared to Group M.
7. No side effects were noted in both the groups.
8. The total duration of analgesia and motor block was significantly prolonged in Group D compared to Group M.
9. The total doses of rescue analgesics needed in 24 hrs was less in Group D compared to Group M.

LIMITATIONS OF THE STUDY

1) Only ASA 1 and 2 patients were included for the study.

2) Since blood loss varies with different types of surgeries, comparison of hemodynamic changes were less reliable, as hemodynamic parameters can vary with blood loss.

CONCLUSION

To conclude, dexmedetomidine seems to be a better adjuvant to intrathecally administered ropivacaine in infraumbilical surgeries when compared with magnesium sulfate with regard to early onset of sensory and motor block, maximum level of sensory block achieved, faster onset of highest level of sensory block with better hemodymanic stability and also prolonging the total duration of analgesia with minimal side effects.

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PROFORMA

Name : Group : D / M

Age :

Sex :

IP No :

Weight :

Height :

BMI :

ASA Grading :

Plan :

Duration Of Surgery :

Baseline (Pre Op) :

PR : /min

BP : mm/Hg

Spo2 : %

	Heart rate	Systolic BP	Diastolic BP	MAP	SPO2
Baseline (Pre op)					
1 min					
3 min					
6 min					
9 min					
12 min					
15 min					
30 min					
45 min					
60 min					
75 min					
90 min					
120 min					

Sensory Block Characteristics

Onset time of sensory block at T10 level =

Maximum sensory level achieved =

Time to achieve maximum sensory level =

Time for regression to L1 dermatome =

Motor block characteristics

Onset time of motor block =

Maximum Bromage scale achieved =

Total duration of motor block =

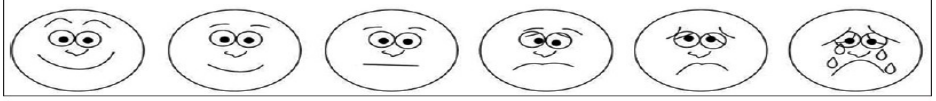
Postoperative parameters

Total duration of analgesia =

Total doses of rescue analgesics in 24hrs =

Visual analogue scale (post op) :

Where 0 - no pain and 10 - maximum imaginable pain.



0	2	4	6	8	10
Very happy, no hurt	Hurts just a little bit	Hurts a little more	Hurts even more	Hurts a whole lot	Hurts as much as you can imagine

30 min		13 hrs	
1 hr		14 hrs	
2 hrs		15 hrs	
3 hrs		16 hrs	
4 hrs		17 hrs	
5 hrs		18 hrs	
6 hrs		19 hrs	
7 hrs		20 hrs	
8 hrs		21 hrs	
9 hrs		22 hrs	
10 hrs		23 hrs	
11 hrs		24 hrs	
12 hrs			

SIDE EFFECTS :

1.Hypotension **Y / N**

2.Bradycardia **Y / N**

3.Vomiting **Y / N**

INFORMATION SHEET

1) We are conducting a study on “Comparison of Dexmedetomidine and Magnesium Sulfate as Adjuvants with Ropivacaine for Spinal Anesthesia in Infraumbilical Surgeries and Postoperative Analgesia”.

2) The purpose of the study is to find whether dexmedetomidine will be a better alternative to magnesium sulfate as an adjuvant to intrathecal ropivacaine .

3) The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research , no personally identifiable information will be shared.

4) Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.

5) The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator Signature of the patient /guardian

Date :

GROUP D

S.NO	NAME	GROUP	AGE	SEX	WEIGHT(kg)	HEIGHT(cm)	ASA	onset time of sensory block at T10 level (mins)	Maximum sensory level achieved	Time to achieve maximum sensory level (mins)	Time to regression to L1 dermatome (mins)	Onset time of motor block (mins)	Maximum Bromage scale achieved	Total duration of motor block (mins)	Total duration of analgesia (mins)	Total doses of rescue analgesics in 24 hrs	Basecline (pre OP)			1 min			3 min			6 min			9 min			12 min			15 min			18 min		
																	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP
1	MARY	D	42	F	64	140	I	4	T5	10	300	8	3	225	405	1	98	120	78	102	122	78	106	116	78	100	114	74	97	116	74	99	112	76	96	110	70	98	112	72
2	RAVI	D	37	M	70	172	I	5	T5	10	330	8	3	230	400	1	93	130	86	102	130	84	106	126	80	94	122	76	90	124	78	88	116	72	90	114	72	87	114	74
3	SUGUNA	D	52	F	62	140	II	4	T5	10	285	8	3	240	435	1	78	110	70	82	108	70	86	104	66	88	106	66	96	96	64	102	98	66	94	106	70	94	112	72
4	GUNASEKARAN	D	30	M	63	166	I	4	T5	9	270	8	3	225	370	2	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72
5	TAMILVANAN	D	36	M	70	172	I	5	T5	10	270	8	3	250	420	1	70	130	80	74	136	84	72	122	76	78	124	76	80	116	70	71	114	68	73	118	74	74	110	70
6	NAGARAJAN	D	45	M	72	166	I	5	T5	8	310	7	3	220	410	1	85	130	78	88	126	76	89	126	78	93	122	72	87	118	70	84	116	72	85	112	72	80	110	70
7	SENTHIL	D	27	M	78	180	I	4	T5	9	280	8	3	230	380	1	86	120	72	88	116	70	100	98	66	106	100	66	104	102	68	96	104	64	92	108	68	94	112	72
8	REVATHI	D	38	F	60	152	I	4	T5	8	270	9	3	210	360	2	98	110	70	102	110	66	106	106	66	108	108	68	99	104	66	97	110	68	101	110	70	103	112	70
9	RAJAGOPALAN	D	48	M	73	170	II	5	T5	9	300	9	3	210	390	1	68	130	70	72	126	72	76	120	70	73	116	68	80	118	72	74	116	72	76	116	68	74	118	70
10	BHUVANA	D	32	F	60	140	I	4	T5	8	315	8	3	240	390	1	78	128	86	88	126	86	90	126	84	92	122	82	90	116	78	96	116	76	86	110	72	82	112	68
11	HEMALATHA	D	25	F	48	138	I	4	T5	9	300	8	3	210	360	2	102	110	70	106	112	74	103	108	68	106	110	68	98	104	66	100	112	70	96	110	70	92	106	68
12	KARTHICK	D	21	M	62	168	I	5	T5	10	285	9	3	220	330	2	104	126	82	106	124	78	102	118	76	98	112	76	90	112	72	86	110	70	90	112	72	94	114	72
13	TAMILARASI	D	30	F	52	140	I	4	T5	9	300	8	3	240	420	1	98	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
14	SUBBULAKSHMI	D	51	F	70	138	II	5	T5	9	240	8	3	255	375	2	80	130	80	86	124	78	88	118	76	98	108	70	100	106	68	104	104	66	98	106	64	96	106	68
15	SWAMINATHAN	D	54	M	65	140	I	4	T5	9	270	8	3	210	360	1	90	120	80	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	76	114	70
16	MANIVASAGAM	D	47	M	69	160	I	5	T5	9	300	8	3	210	390	1	82	124	84	84	120	80	88	112	72	92	110	68	96	104	64	90	112	70	90	116	74	86	122	78
17	ARULDOSS	D	40	M	78	172	I	4	T5	9	240	9	3	210	360	1	88	134	86	90	130	82	96	118	76	86	120	78	94	116	76	92	112	72	93	116	72	86	118	72
18	ANJAMMAL	D	49	F	60	140	I	4	T5	10	270	10	3	240	390	1	86	118	74	90	116	78	94	114	76	92	110	70	96	114	76	88	110	70	90	116	76	86	118	70
19	MUTHURAJ	D	55	M	70	170	I	4	T5	8	285	9	3	210	360	1	92	120	78	98	112	68	94	108	70	88	110	68	90	112	70	82	110	70	84	114	72	78	110	70
20	ULAGANATHAN	D	48	M	74	168	I	5	T5	8	300	9	3	240	420	1	78	110	70	86	112	70	88	106	68	86	108	68	88	108	70	80	110	70	78	112	72	76	108	72
21	RADHAKRISHNAN	D	58	M	66	145	I	5	T5	8	300	9	3	240	390	1	92	136	84	96	134	80	98	126	76	94	122	76	88	118	76	90	118	74	86	116	72	90	112	74
22	SUSILA	D	50	F	55	136	II	4	T5	9	310	9	3	220	375	1	98	132	78	100	126	78	104	124	78	96	122	76	98	110	70	102	104	66	106	104	64	104	108	68
23	RAMAYI	D	44	F	58	140	II	5	T5	10	270	8	3	225	360	1	90	124	84	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	76	114	70
24	NAVSHADALI	D	41	M	70	168	II	5	T5	8	240	9	3	180	300	2	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68	86	110	70	80	114	72
25	PANEERSELVAM	D	51	M	73	166	I	4	T5	8	310	8	3	200	390	1	82	118	78	88	110	72	90	110	68	96	110	70	90	112	72	84	114	68	80	114	70	82	112	68

S.NO	NAME	21 min			24 min			27 min			30 min			45 min			60 min			75 min			90 min			105 min			120 min			Side Effects		
		HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	Hypotension	Bradycardia	Vomiting
1	MARY	90	118	76	93	116	76	97	116	74	94	118	78	91	114	72	97	114	72	98	116	72										**0**	**0**	**0**
2	RAVI	83	112	70	90	116	74	88	118	76	80	118	76	82	116	74																0	0	0
3	SUGUNA	86	118	74	87	110	70	82	108	68	88	112	68	93	104	66	91	106	66	89	106	68										0	0	0
4	GUNASEKARAN	86	114	74	82	112	70	84	110	68	81	112	72	83	116	76	84	108	70	88	110	70										0	0	0
5	TAMILVANAN	79	112	68	84	116	72	82	118	74	90	114	70	88	116	70	83	118	74	71	122	70										0	0	0
6	NAGARAJAN	84	114	74	86	110	72	91	118	74	93	116	72	88	110	72	90	116	72													0	0	0
7	SENTHIL	88	114	70	84	116	72	82	122	76	80	118	70	86	120	68	84	116	70	80	120	72										1	0	0
8	REVATHI	94	108	70	97	108	68	90	112	68	88	110	72	87	116	74	82	114	72													0	0	0
9	RAJAGOPALAN	78	116	72	70	112	70	74	116	70	78	114	72	72	118	74	76	120	72	70	122	74	74	124	76							0	0	0
10	BHUVANA	83	114	74	79	110	70	85	112	72	88	114	72	78	112	74	87	110	70	83	108	70	86	112	72	80	110	70				0	0	0
11	HEMALATHA	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	88	104	70	86	108	70							0	0	0
12	KARTHICK	98	110	70	88	112	74	90	114	70	84	116	70	88	114	68	90	116	70	92	116	72	88	114	74	94	118	76	88	116	74	0	0	0
13	TAMILARASI	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70	84	114	72	88	110	70	0	0	0
14	SUBBULAKSHMI	96	112	70	90	110	70	89	112	70	84	112	72	76	110	70	78	112	72	82	114	72										1	0	0
15	SWAMINATHAN	80	110	70	82	112	72	78	110	72	88	108	68	82	110	72	78	112	74	80	114	74	82	112	70							0	0	0
16	MANIVASAGAM	88	120	78	84	124	82	82	130	80	80	120	84	80	128	82	82	122	80	76	116	76	78	120	70							0	0	0
17	ARULDOSS	88	110	76	90	112	72	92	114	74	84	116	76	86	118	74	90	116	70	82	122	76	80	118	74							0	0	0
18	ANJAMMAL	80	112	68	82	110	70	86	112	68	90	110	70	85	110	72	88	112	70	82	114	74	78	116	74	80	116	74				0	0	0
19	MUTHURAJ	80	112	72	76	114	70	82	110	72	78	114	68	82	110	70	84	114	74	86	116	74	90	112	72							0	0	0
20	ULAGANATHAN	80	110	70	82	108	68	78	108	70	80	110	70	82	112	70	76	110	70	80	112	72	82	114	70							0	0	0
21	RADHAKRISHNAN	86	110	72	90	112	76	92	116	74	84	116	72	80	118	74	82	116	76	86	112	72	82	114	72							0	0	0
22	SUSILA	100	110	70	92	116	72	88	114	72	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	90	116	74				1	0	0
23	RAMAYI	80	110	70	82	112	72	78	110	72	88	108	68	82	110	72	78	112	74	80	114	74										0	0	0
24	NAVSHADALI	82	114	74	80	110	70	82	112	72	80	110	70	78	114	72	76	116	78	80	116	78	82	120	70							0	0	0
25	PANEERSELVAM	80	114	70	82	112	68	84	114	70	86	110	70	82	114	68	86	116	72	78	120	74	82	118	72							0	0	0

GROUP M

S.NO	NAME	GROUP	AGE	SEX	WEIGHT(kg)	HEIGHT(cm)	ASA	onset time of sensory block at T10 level (mins)	Maximum sensory level achieved	time to achieve maximum sensory level (mins)	Time to regression to L1 dermatome (mins)	Onset time of motor block (mins)	Maximum Bromage scale achieved	Total duration of motor block (mins)	Total duration of analgesia (mins)	Total doses of rescue analgesics in 24 hrs	Baseline (pre OP)			1 min			3 min			6 min			9 min			12 min			15 min			18 min		
																	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP
1	JAYARAMAN	M	47	M	82	170	I	6	T6	13	145	13	3	190	225	3	86	120	72	88	116	70	94	110	68	98	108	70	98	112	68	92	104	64	92	118	70	84	120	72
2	POUNAMMAL	M	38	M	50	148	I	7	T6	13	170	13	3	150	210	3	80	120	72	84	116	68	88	112	70	88	104	64	92	108	70	90	112	72	90	116	70	86	112	72
3	LAKSHMI	M	60	F	60	150	II	7	T6	13	195	13	3	150	230	2	90	130	74	92	124	70	96	120	68	98	118	68	102	110	64	98	108	72	106	106	64	104	106	66
4	KALIYAMOORTHY	M	53	M	70	168	I	6	T6	12	180	13	3	180	240	3	82	120	82	88	116	80	96	114	74	96	110	70	94	106	68	94	106	68	90	118	70	92	124	76
5	RANJITH KUMAR	M	21	M	56	158	I	7	T6	12	200	14	3	180	220	3	88	124	84	84	120	80	88	112	72	92	110	68	96	104	64	90	112	70	90	116	74	86	122	78
6	ULAGANATHAN	M	40	M	76	170	I	7	T6	13	195	13	3	150	225	3	86	120	84	88	116	80	90	112	72	94	110	70	96	108	70	96	102	68	92	104	70	94	112	74
7	VIJAYAKUMARI	M	51	F	64	156	I	6	T6	13	170	12	3	180	200	3	92	116	74	96	108	70	94	104	68	98	104	64	102	100	62	102	112	68	94	114	70	92	114	76
8	KUMARESAN	M	45	M	70	170	I	6	T6	12	190	9	3	150	225	2	78	128	86	88	126	86	90	126	84	92	122	82	90	116	78	96	116	76	86	110	72	82	112	68
9	ILAYARAJA	M	39	M	80	170	I	7	T6	12	210	12	3	165	230	2	92	120	84	98	116	82	98	104	78	96	102	68	102	102	66	102	98	64	98	104	68	106	104	68
10	DEVI	M	39	F	65	140	II	6	T6	12	190	10	3	195	220	3	98	110	70	102	110	66	106	106	66	108	108	68	99	104	66	97	110	68	101	110	70	103	112	70
11	NATHIYA	M	19	F	48	140	I	7	T6	14	180	13	3	150	210	3	102	110	70	106	112	74	103	108	68	106	110	68	98	104	66	100	112	70	96	110	70	92	106	68
12	RAJKUMAR	M	23	M	65	165	I	6	T6	13	180	12	3	180	225	2	100	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
13	PRABHAKARAN	M	22	M	60	168	I	6	T6	14	170	14	3	160	260	2	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72
14	DHANALAKSHMI	M	46	F	70	145	II	7	T6	13	210	12	3	190	250	3	98	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
15	SANJEEVI RAMAN	M	44	M	74	170	I	6	T6	13	180	13	3	180	240	2	96	122	78	98	118	76	102	116	74	96	114	72	100	112	70	96	110	70	88	112	72	92	110	70
16	HASEENA BEGUM	M	38	F	60	140	I	6	T6	14	150	13	3	150	210	3	92	120	84	98	116	82	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76
17	RENGARAJ	M	53	M	67	170	II	7	T6	12	170	14	3	165	200	3	98	132	78	100	126	78	104	124	78	96	122	76	98	124	70	96	120	76	90	118	76	88	118	76
18	THENMOZHI	M	55	F	74	146	I	7	T6	13	200	15	3	150	195	3	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68	86	110	70	80	114	72
19	MAHALINGAM	M	55	M	60	157	I	8	T6	12	195	12	3	180	255	2	85	130	78	88	126	76	89	126	78	93	122	72	87	118	70	84	116	72	85	112	72	80	110	70
20	BALAKANNU	M	57	M	72	160	I	6	T6	13	210	14	3	210	240	2	98	120	78	102	122	78	106	116	78	108	98	66	106	98	64	104	100	66	102	102	66	98	108	70
21	MUTHUSAMY	M	47	M	55	164	I	7	T6	14	165	13	3	175	205	3	78	128	86	88	126	86	90	126	84	92	122	82	88	118	76	90	114	68	92	116	72	88	114	72
22	CHELLAPPA	M	39	M	60	170	I	6	T6	13	180	15	3	150	200	3	90	124	84	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	86	110	70
23	RAMARAJAN	M	55	M	65	168	II	7	T6	14	190	13	3	145	210	3	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76	92	122	70	88	120	82
24	RANJITKUMAR	M	40	M	70	172	II	8	T6	12	210	14	3	180	240	2	90	116	78	94	114	76	92	110	70	96	114	76	88	110	70	90	116	76	86	118	70	80	112	68
25	MARUTHAMUTHU	M	37	M	68	165	I	8	T6	13	180	14	3	150	210	3	68	130	70	72	126	72	76	120	70	73	116	68	80	118	72	74	116	72	76	116	68	74	118	70

S.NO	NAME	21 min			24 min			27 min			30 min			45 min			60 min			75 min			90 min			105 min			120 min			Side Effects		
		HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	Hypotension	Bradycardia	Vomiting
1	JAYARAMAN	82	124	68	84	116	74	82	122	76	80	118	70	86	120	68	84	116	70	90	124	76											**0**	**0**	**0**
2	POUNAMMAL	84	122	74	82	118	72	80	120	72	84	120	72	86	118	70	80	120	76														0	0	0
3	LAKSHMI	92	108	70	94	110	70	90	116	72	92	120	70	94	122	70	92	124	76	90	130	70	84	128	72								1	0	0
4	KALIYAMOORTHY	84	122	80	88	128	74	86	130	72	80	120	80	82	122	84	84	130	80	80	126	72											0	0	0
5	RANJITH KUMAR	88	120	78	84	124	82	82	130	80	80	120	84	80	128	82	82	122	80														0	0	0
6	ULAGANATHAN	92	110	76	84	116	80	88	120	82	84	122	84	80	120	88																	0	0	0
7	VIJAYAKUMARI	92	114	76	88	110	74	92	124	72	94	120	70	90	122	82	90	124	80														0	0	0
8	KUMARESAN	83	114	74	79	110	70	85	112	72	88	114	72	78	112	74	87	110	70	83	108	70	86	112	72								0	0	0
9	ILAYARAJA	92	106	68	88	110	70	94	116	78	92	118	82	90	120	86																	1	0	0
10	DEVI	94	108	70	97	108	68	90	112	68	88	110	72	87	116	74																	0	0	0
11	NATHIYA	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	88	104	70	86	108	70								0	0	0
12	RAJKUMAR	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70	84	114	72					0	0	0
13	PRABHAKARAN	86	114	74	82	112	70	84	110	68	81	112	72	83	116	76	84	108	70	88	110	70											0	0	0
14	DHANALAKSHMI	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70								0	0	0
15	SANJEEVI RAMAN	86	108	68	90	110	68	88	114	70	82	112	70	82	114	74	78	116	72	80	116	78	82	116	80	80	116	82					0	0	0
16	HASEENA BEGUM	92	122	70	88	120	82	94	122	84	92	118	82	90	120	86																	0	0	0
17	RENGARAJ	90	114	68	92	116	72	88	114	72	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	90	116	74					0	0	0
18	THENMOZHI	82	114	74	80	110	70	82	112	72	80	110	70	78	114	72	76	116	78	80	116	78	82	120	70								0	0	0
19	MAHALINGAM	84	114	74	86	110	72	91	118	74	93	116	72	88	110	72	90	116	72	88	118	74	82	116	76								0	0	0
20	BALAKANNU	90	112	74	93	116	76	97	116	74	94	118	78	91	114	72	97	114	72	98	116	72											1	0	0
21	MUTHUSAMY	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	80	118	70	82	116	78	78	116	80								0	0	0
22	CHELLAPPA	90	112	72	94	114	72	98	110	70	88	112	74	90	114	70	84	116	70	88	114	68	90	116	70	82	118	72					0	0	0
23	RAMARAJAN	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72	82	116	78	84	118	72	0	0	0
24	RANJITKUMAR	82	110	70	86	112	68	90	110	70	85	110	72	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	0	0	0
25	MARUTHAMUTHU	78	116	72	70	112	70	74	116	70	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68					0	0	0

KEY TO MASTERCHART

S.NO	Serial Number
0	No
1	Yes
F	Female
M	Male
ASA	American Society of Anaesthesiologists
Pre –op	Preoperative
min	Minutes
HR	Heart Rate (in beats/ minute)
SBP	Systolic Blood Pressure (in mmHg)
DBP	Diastolic Blood Pressure (in mmHg)

LIST OF ABBREVIATIONS

ASA - American Society of Anaesthesiologist

BP - Blood Pressure

PR - pulse rate

CNS - Central nervous system

CVS - Cardiovascular system

I.V - Intravenous

Kg - Kilogram

μg - Microgram

mg - Milligram

ml - Millilitre

MAP - Mean Arterial Pressure

SPO_2 - Oxygen saturation

SD - standard deviation

G - Gauge

GROUP D

S.NO	NAME	GROUP	AGE	SEX	WEIGHT(kg)	HEIGHT(cm)	ASA	onset time of sensory block at T10 level (mins)	Maximum sensory level achieved	Time to achieve maximum sensory level (mins)	Time to regression to L1 dermatome (mins)	Onset time of motor block (mins)	Maximum Bromage scale achieved	Total duration of motor block (mins)	Total duration of analgesia (mins)	Total doses of rescue analgesics in 24 hrs	Baseline (pre OP)			1 min			3 min			6 min			9 min			12 min			15 min			18 min		
																	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP
1	MARY	D	42	F	64	140	I	4	T5	10	300	8	3	225	405	1	98	120	78	102	122	78	106	116	78	100	114	74	97	116	74	99	112	76	96	110	70	98	112	72
2	RAVI	D	37	M	70	172	I	5	T5	10	330	8	3	230	400	1	93	130	86	102	130	84	106	126	80	94	122	76	90	124	78	88	116	72	90	114	72	87	114	74
3	SUGUNA	D	52	F	62	140	II	4	T5	10	285	8	3	240	435	1	78	110	70	82	108	70	86	104	66	88	106	66	96	96	64	102	98	66	94	106	70	94	112	72
4	GUNASEKARAN	D	30	M	63	166	I	4	T5	9	270	8	3	225	370	2	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72
5	TAMILVANAN	D	36	M	70	172	I	5	T5	10	270	8	3	250	420	1	70	130	80	74	136	84	72	122	76	78	124	76	80	116	70	71	114	68	73	118	74	74	110	70
6	NAGARAJAN	D	45	M	72	166	I	5	T5	8	310	7	3	220	410	1	85	130	78	88	126	76	89	126	78	93	122	72	87	118	70	84	116	72	85	112	72	80	110	70
7	SENTHIL	D	27	M	78	180	I	4	T5	9	280	8	3	230	380	1	86	120	72	88	116	70	100	98	66	106	100	66	104	102	68	96	104	64	92	108	68	94	112	72
8	REVATHI	D	38	F	60	152	I	4	T5	8	270	9	3	210	360	2	98	110	70	102	110	66	106	106	66	108	108	68	99	104	66	97	110	68	101	110	70	103	112	70
9	RAJAGOPALAN	D	48	M	73	170	II	5	T5	9	300	9	3	210	390	1	68	130	70	72	126	72	76	120	70	73	116	68	80	118	72	74	116	72	76	116	68	74	118	70
10	BHUVANA	D	32	F	60	140	I	4	T5	8	315	8	3	240	390	1	78	128	86	88	126	86	90	126	84	92	122	82	90	116	78	96	116	76	86	110	72	82	112	68
11	HEMALATHA	D	25	F	48	138	I	4	T5	9	300	8	3	210	360	2	102	110	70	106	112	74	103	108	68	106	110	68	98	104	66	100	112	70	96	110	70	92	106	68
12	KARTHICK	D	21	M	62	168	I	5	T5	10	285	9	3	220	330	2	104	126	82	106	124	78	102	118	76	98	112	76	90	112	72	86	110	70	90	112	72	94	114	72
13	TAMILARASI	D	30	F	52	140	I	4	T5	9	300	8	3	240	420	1	98	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
14	SUBBULAKSHMI	D	51	F	70	138	II	5	T5	9	240	8	3	255	375	2	80	130	80	86	124	78	88	118	76	98	108	70	100	106	68	104	104	66	98	106	64	96	106	68
15	SWAMINATHAN	D	54	M	65	140	I	4	T5	9	270	8	3	210	360	1	90	120	80	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	76	114	70
16	MANIVASAGAM	D	47	M	69	160	I	5	T5	9	300	8	3	210	390	1	82	124	84	84	120	80	88	112	72	92	110	68	96	104	64	90	112	70	90	116	74	86	122	78
17	ARULDOSS	D	40	M	78	172	I	4	T5	9	240	9	3	210	360	1	88	134	86	90	130	82	96	118	76	86	120	78	94	116	76	92	112	72	93	116	72	86	118	72
18	ANJAMMAL	D	49	F	60	140	I	4	T5	10	270	10	3	240	390	1	86	118	74	90	116	78	94	114	76	92	110	70	96	114	76	88	110	70	90	116	76	86	118	70
19	MUTHURAJ	D	55	M	70	170	I	4	T5	8	285	9	3	210	360	1	92	120	78	98	112	68	94	108	70	88	110	68	90	112	70	82	110	70	84	114	72	78	110	70
20	ULAGANATHAN	D	48	M	74	168	I	5	T5	8	300	9	3	240	420	1	78	110	70	86	112	70	88	106	68	86	108	68	88	108	70	80	110	70	78	112	72	76	108	72
21	RADHAKRISHNAN	D	58	M	66	145	I	5	T5	8	300	9	3	240	390	1	92	136	84	96	134	80	98	126	76	94	122	76	88	118	76	90	118	74	86	116	72	90	112	74
22	SUSILA	D	50	F	55	136	II	4	T5	9	310	9	3	220	375	1	98	132	78	100	126	78	104	124	78	96	122	76	98	110	70	102	104	66	106	104	64	104	108	68
23	RAMAYI	D	44	F	58	140	II	5	T5	10	270	8	3	225	360	1	90	124	84	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	76	114	70
24	NAVSHADALI	D	41	M	70	168	II	5	T5	8	240	9	3	180	300	2	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68	86	110	70	80	114	72
25	PANEERSELVAM	D	51	M	73	166	I	4	T5	8	310	8	3	200	390	1	82	118	78	88	110	72	90	110	68	96	110	70	90	112	72	84	114	68	80	114	70	82	112	68

S.NO	NAME	21 min			24 min			27 min			30 min			45 min			60 min			75 min			90 min			105 min			120 min			Side Effects		
		HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	Hypotension	Bradycardia	Vomiting
1	MARY	90	118	76	93	116	76	97	116	74	94	118	78	91	114	72	97	114	72	98	116	72										**0**	**0**	**0**
2	RAVI	83	112	70	90	116	74	88	118	76	80	118	76	82	116	74																0	0	0
3	SUGUNA	86	118	74	87	110	70	82	108	68	88	112	68	93	104	66	91	106	66	89	106	68										0	0	0
4	GUNASEKARAN	86	114	74	82	112	70	84	110	68	81	112	72	83	116	76	84	108	70	88	110	70										0	0	0
5	TAMILVANAN	79	112	68	84	116	72	82	118	74	90	114	70	88	116	70	83	118	74	71	122	70										0	0	0
6	NAGARAJAN	84	114	74	86	110	72	91	118	74	93	116	72	88	110	72	90	116	72													0	0	0
7	SENTHIL	88	114	70	84	116	72	82	122	76	80	118	70	86	120	68	84	116	70	80	120	72										1	0	0
8	REVATHI	94	108	70	97	108	68	90	112	68	88	110	72	87	116	74	82	114	72													0	0	0
9	RAJAGOPALAN	78	116	72	70	112	70	74	116	70	78	114	72	72	118	74	76	120	72	70	122	74	74	124	76							0	0	0
10	BHUVANA	83	114	74	79	110	70	85	112	72	88	114	72	78	112	74	87	110	70	83	108	70	86	112	72	80	110	70				0	0	0
11	HEMALATHA	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	88	104	70	86	108	70							0	0	0
12	KARTHICK	98	110	70	88	112	74	90	114	70	84	116	70	88	114	68	90	116	70	92	116	72	88	114	74	94	118	76	88	116	74	0	0	0
13	TAMILARASI	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70	84	114	72	88	110	70	0	0	0
14	SUBBULAKSHMI	96	112	70	90	110	70	89	112	70	84	112	72	76	110	70	78	112	72	82	114	72										1	0	0
15	SWAMINATHAN	80	110	70	82	112	72	78	110	72	88	108	68	82	110	72	78	112	74	80	114	74	82	112	70							0	0	0
16	MANIVASAGAM	88	120	78	84	124	82	82	130	80	80	120	84	80	128	82	82	122	80	76	116	76	78	120	70							0	0	0
17	ARULDOSS	88	110	76	90	112	72	92	114	74	84	116	76	86	118	74	90	116	70	82	122	76	80	118	74							0	0	0
18	ANJAMMAL	80	112	68	82	110	70	86	112	68	90	110	70	85	110	72	88	112	70	82	114	74	78	116	74	80	116	74				0	0	0
19	MUTHURAJ	80	112	72	76	114	70	82	110	72	78	114	68	82	110	70	84	114	74	86	116	74	90	112	72							0	0	0
20	ULAGANATHAN	80	110	70	82	108	68	78	108	70	80	110	70	82	112	70	76	110	70	80	112	72	82	114	70							0	0	0
21	RADHAKRISHNAN	86	110	72	90	112	76	92	116	74	84	116	72	80	118	74	82	116	76	86	112	72	82	114	72							0	0	0
22	SUSILA	100	110	70	92	116	72	88	114	72	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	90	116	74				1	0	0
23	RAMAYI	80	110	70	82	112	72	78	110	72	88	108	68	82	110	72	78	112	74	80	114	74										0	0	0
24	NAVSHADALI	82	114	74	80	110	70	82	112	72	80	110	70	78	114	72	76	116	78	80	116	78	82	120	70							0	0	0
25	PANEERSELVAM	80	114	70	82	112	68	84	114	70	86	110	70	82	114	68	86	116	72	78	120	74	82	118	72							0	0	0

GROUP M

S.NO	NAME	GROUP	AGE	SEX	WEIGHT(kg)	HEIGHT(cm)	ASA	onset time of sensory block at T10 level (mins)	Maximum sensory level achieved	Time to acmeve maximum sensory level (mins)	Time to regression to L1 dermatome (mins)	Onset time of motor block (mins)	Maximum Bromage scale achieved	Total duration of motor block (mins)	Total duration of analgesia (mins)	Total doses of rescue analgesics in 24 hrs	Baseline (pre OP)			1 min			3 min			6 min			9 min			12 min			15 min			18 min		
																	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP
1	JAYARAMAN	M	47	M	82	170	I	6	T6	13	145	13	3	190	225	3	86	120	72	88	116	70	94	110	68	98	108	70	98	112	68	92	104	64	92	118	70	84	120	72
2	POUNAMMAL	M	38	M	50	148	I	7	T6	13	170	13	3	150	210	3	80	120	72	84	116	68	88	112	70	88	104	64	92	108	70	90	112	72	90	116	70	86	112	72
3	LAKSHMI	M	60	F	60	150	II	7	T6	13	195	13	3	150	230	2	90	130	74	92	124	70	96	120	68	98	118	68	102	110	64	98	108	72	106	106	64	104	106	66
4	KALIYAMOORTHY	M	53	M	70	168	I	6	T6	12	180	13	3	180	240	3	82	120	82	88	116	80	96	114	74	96	110	70	94	106	68	94	106	68	90	118	70	92	124	76
5	RANJITH KUMAR	M	21	M	56	158	I	7	T6	12	200	14	3	180	220	3	88	124	84	84	120	80	88	112	72	92	110	68	96	104	64	90	112	70	90	116	74	86	122	78
6	ULAGANATHAN	M	40	M	76	170	I	7	T6	13	195	13	3	150	225	3	86	120	84	88	116	80	90	112	72	94	110	70	96	108	70	96	102	68	92	104	70	94	112	74
7	VIJAYAKUMARI	M	51	F	64	156	I	6	T6	13	170	12	3	180	200	3	92	116	74	96	108	70	94	104	68	98	104	64	102	100	62	102	112	68	94	114	70	92	114	76
8	KUMARESAN	M	45	M	70	170	I	6	T6	12	190	9	3	150	225	2	78	128	86	88	126	86	90	126	84	92	122	82	90	116	78	96	116	76	86	110	72	82	112	68
9	ILAYARAJA	M	39	M	80	170	I	7	T6	12	210	12	3	165	230	2	92	120	84	98	116	82	98	104	78	96	102	68	102	102	66	102	98	64	98	104	68	106	104	68
10	DEVI	M	39	F	65	140	II	6	T6	12	190	10	3	195	220	3	98	110	70	102	110	66	106	106	66	108	108	68	99	104	66	97	110	68	101	110	70	103	112	70
11	NATHIYA	M	19	F	48	140	I	7	T6	14	180	13	3	150	210	3	102	110	70	106	112	74	103	108	68	106	110	68	98	104	66	100	112	70	96	110	70	92	106	68
12	RAJKUMAR	M	23	M	65	165	I	6	T6	13	180	12	3	180	225	2	100	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
13	PRABHAKARAN	M	22	M	60	168	I	6	T6	14	170	14	3	160	260	2	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72
14	DHANALAKSHMI	M	46	F	70	145	II	7	T6	13	210	12	3	190	250	3	98	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
15	SANJEEVI RAMAN	M	44	M	74	170	I	6	T6	13	180	13	3	180	240	2	96	122	78	98	118	76	102	116	74	96	114	72	100	112	70	96	110	70	88	112	72	92	110	70
16	HASEENA BEGUM	M	38	F	60	140	I	6	T6	14	150	13	3	150	210	3	92	120	84	98	116	82	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76
17	RENGARAJ	M	53	M	67	170	II	7	T6	12	170	14	3	165	200	3	98	132	78	100	126	78	104	124	78	96	122	76	98	124	70	96	120	76	90	118	76	88	118	76
18	THENMOZHI	M	55	F	74	146	I	7	T6	13	200	15	3	150	195	3	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68	86	110	70	80	114	72
19	MAHALINGAM	M	55	M	60	157	I	8	T6	12	195	12	3	180	255	2	85	130	78	88	126	76	89	126	78	93	122	72	87	118	70	84	116	72	85	112	72	80	110	70
20	BALAKANNU	M	57	M	72	160	I	6	T6	13	210	14	3	210	240	2	98	120	78	102	122	78	106	116	78	108	98	66	106	98	64	104	100	66	102	102	66	98	108	70
21	MUTHUSAMY	M	47	M	55	164	I	7	T6	14	165	13	3	175	205	3	78	128	86	88	126	86	90	126	84	92	122	82	88	118	76	90	114	68	92	116	72	88	114	72
22	CHELLAPPA	M	39	M	60	170	I	6	T6	13	180	15	3	150	200	3	90	124	84	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	86	110	70
23	RAMARAJAN	M	55	M	65	168	II	7	T6	14	190	13	3	145	210	3	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76	92	122	70	88	120	82
24	RANJITKUMAR	M	40	M	70	172	II	8	T6	12	210	14	3	180	240	2	90	116	78	94	114	76	92	110	70	96	114	76	88	110	70	90	116	76	86	118	70	80	112	68
25	MARUTHAMUTHU	M	37	M	68	165	I	8	T6	13	180	14	3	150	210	3	68	130	70	72	126	72	76	120	70	73	116	68	80	118	72	74	116	72	76	116	68	74	118	70

S.NO	NAME	21 min			24 min			27 min			30 min			45 min			60 min			75 min			90 min			105 min			120 min			Side Effects		
		HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	Hypotension	Bradycardia	Vomiting
1	JAYARAMAN	82	124	68	84	116	74	82	122	76	80	118	70	86	120	68	84	116	70	90	124	76										**0**	**0**	**0**
2	POUNAMMAL	84	122	74	82	118	72	80	120	72	84	120	72	86	118	70	80	120	76													0	0	0
3	LAKSHMI	92	108	70	94	110	70	90	116	72	92	120	70	94	122	70	92	124	76	90	130	70	84	128	72							1	0	0
4	KALIYAMOORTHY	84	122	80	88	128	74	86	130	72	80	120	80	82	122	84	84	130	80	80	126	72										0	0	0
5	RANJITH KUMAR	88	120	78	84	124	82	82	130	80	80	120	84	80	128	82	82	122	80													0	0	0
6	ULAGANATHAN	92	110	76	84	116	80	88	120	82	84	122	84	80	120	88																0	0	0
7	VIJAYAKUMARI	92	114	76	88	110	74	92	124	72	94	120	70	90	122	82	90	124	80													0	0	0
8	KUMARESAN	83	114	74	79	110	70	85	112	72	88	114	72	78	112	74	87	110	70	83	108	70	86	112	72							0	0	0
9	ILAYARAJA	92	106	68	88	110	70	94	116	78	92	118	82	90	120	86																1	0	0
10	DEVI	94	108	70	97	108	68	90	112	68	88	110	72	87	116	74																0	0	0
11	NATHIYA	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	88	104	70	86	108	70							0	0	0
12	RAJKUMAR	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70	84	114	72				0	0	0
13	PRABHAKARAN	86	114	74	82	112	70	84	110	68	81	112	72	83	116	76	84	108	70	88	110	70										0	0	0
14	DHANALAKSHMI	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70							0	0	0
15	SANJEEVI RAMAN	86	108	68	90	110	68	88	114	70	82	112	70	82	114	74	78	116	72	80	116	78	82	116	80	80	116	82				0	0	0
16	HASEENA BEGUM	92	122	70	88	120	82	94	122	84	92	118	82	90	120	86																0	0	0
17	RENGARAJ	90	114	68	92	116	72	88	114	72	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	90	116	74				0	0	0
18	THENMOZHI	82	114	74	80	110	70	82	112	72	80	110	70	78	114	72	76	116	78	80	116	78	82	120	70							0	0	0
19	MAHALINGAM	84	114	74	86	110	72	91	118	74	93	116	72	88	110	72	90	116	72	88	118	74	82	116	76							0	0	0
20	BALAKANNU	90	112	74	93	116	76	97	116	74	94	118	78	91	114	72	97	114	72	98	116	72										1	0	0
21	MUTHUSAMY	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	80	118	70	82	116	78	78	116	80							0	0	0
22	CHELLAPPA	90	112	72	94	114	72	98	110	70	88	112	74	90	114	70	84	116	70	88	114	68	90	116	70	82	118	72				0	0	0
23	RAMARAJAN	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72	82	116	78	84	118	72	0	0	0
24	RANJITKUMAR	82	110	70	86	112	68	90	110	70	85	110	72	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	0	0	0
25	MARUTHAMUTHU	78	116	72	70	112	70	74	116	70	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68				0	0	0

GROUP M

S.NO	NAME	GROUP	AGE	SEX	WEIGHT(kg)	HEIGHT(cm)	ASA	onset time of sensory block at T10 level (mins)	Maximum sensory level achieved	Time to acmeve maximum sensory level (mins)	Time to regression to L1 dermatome (mins)	Onset time of motor block (mins)	Maximum Bromage scale achieved	Total duration of motor block (mins)	Total duration of analgesia (mins)	Total doses of rescue analgesics in 24 hrs	Baseline (pre OP)			1 min			3 min			6 min			9 min			12 min			15 min			18 min		
																	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP
1	JAYARAMAN	M	47	M	82	170	I	6	T6	13	145	13	3	190	225	3	86	120	72	88	116	70	94	110	68	98	108	70	98	112	68	92	104	64	92	118	70	84	120	72
2	POUNAMMAL	M	38	M	50	148	I	7	T6	13	170	13	3	150	210	3	80	120	72	84	116	68	88	112	70	88	104	64	92	108	70	90	112	72	90	116	70	86	112	72
3	LAKSHMI	M	60	F	60	150	II	7	T6	13	195	13	3	150	230	2	90	130	74	92	124	70	96	120	68	98	118	68	102	110	64	98	108	72	106	106	64	104	106	66
4	KALIYAMOORTHY	M	53	M	70	168	I	6	T6	12	180	13	3	180	240	3	82	120	82	88	116	80	96	114	74	96	110	70	94	106	68	94	106	68	90	118	70	92	124	76
5	RANJITH KUMAR	M	21	M	56	158	I	7	T6	12	200	14	3	180	220	3	88	124	84	84	120	80	88	112	72	92	110	68	96	104	64	90	112	70	90	116	74	86	122	78
6	ULAGANATHAN	M	40	M	76	170	I	7	T6	13	195	13	3	150	225	3	86	120	84	88	116	80	90	112	72	94	110	70	96	108	70	96	102	68	92	104	70	94	112	74
7	VIJAYAKUMARI	M	51	F	64	156	I	6	T6	13	170	12	3	180	200	3	92	116	74	96	108	70	94	104	68	98	104	64	102	100	62	102	112	68	94	114	70	92	114	76
8	KUMARESAN	M	45	M	70	170	I	6	T6	12	190	9	3	150	225	2	78	128	86	88	126	86	90	126	84	92	122	82	90	116	78	96	116	76	86	110	72	82	112	68
9	ILAYARAJA	M	39	M	80	170	I	7	T6	12	210	12	3	165	230	2	92	120	84	98	116	82	98	104	78	96	102	68	102	102	66	102	98	64	98	104	68	106	104	68
10	DEVI	M	39	F	65	140	II	6	T6	12	190	10	3	195	220	3	98	110	70	102	110	66	106	106	66	108	108	68	99	104	66	97	110	68	101	110	70	103	112	70
11	NATHIYA	M	19	F	48	140	I	7	T6	14	180	13	3	150	210	3	102	110	70	106	112	74	103	108	68	106	110	68	98	104	66	100	112	70	96	110	70	92	106	68
12	RAJKUMAR	M	23	M	65	165	I	6	T6	13	180	12	3	180	225	2	100	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
13	PRABHAKARAN	M	22	M	60	168	I	6	T6	14	170	14	3	160	260	2	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72
14	DHANALAKSHMI	M	46	F	70	145	II	7	T6	13	210	12	3	190	250	3	98	116	76	102	114	76	106	110	70	96	112	68	100	110	68	92	112	72	88	110	70	86	110	68
15	SANJEEVI RAMAN	M	44	M	74	170	I	6	T6	13	180	13	3	180	240	2	96	122	78	98	118	76	102	116	74	96	114	72	100	112	70	96	110	70	88	112	72	92	110	70
16	HASEENA BEGUM	M	38	F	60	140	I	6	T6	14	150	13	3	150	210	3	92	120	84	98	116	82	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76
17	RENGARAJ	M	53	M	67	170	II	7	T6	12	170	14	3	165	200	3	98	132	78	100	126	78	104	124	78	96	122	76	98	124	70	96	120	76	90	118	76	88	118	76
18	THENMOZHI	M	55	F	74	146	I	7	T6	13	200	15	3	150	195	3	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68	86	110	70	80	114	72
19	MAHALINGAM	M	55	M	60	157	I	8	T6	12	195	12	3	180	255	2	85	130	78	88	126	76	89	126	78	93	122	72	87	118	70	84	116	72	85	112	72	80	110	70
20	BALAKANNU	M	57	M	72	160	I	6	T6	13	210	14	3	210	240	2	98	120	78	102	122	78	106	116	78	108	98	66	106	98	64	104	100	66	102	102	66	98	108	70
21	MUTHUSAMY	M	47	M	55	164	I	7	T6	14	165	13	3	175	205	3	78	128	86	88	126	86	90	126	84	92	122	82	88	118	76	90	114	68	92	116	72	88	114	72
22	CHELLAPPA	M	39	M	60	170	I	6	T6	13	180	15	3	150	200	3	90	124	84	94	114	76	98	110	70	88	108	72	86	110	68	80	110	70	82	112	72	86	110	70
23	RAMARAJAN	M	55	M	65	168	II	7	T6	14	190	13	3	145	210	3	98	104	78	96	102	68	102	102	70	102	104	64	90	112	72	94	118	76	92	122	70	88	120	82
24	RANJITKUMAR	M	40	M	70	172	II	8	T6	12	210	14	3	180	240	2	90	116	78	94	114	76	92	110	70	96	114	76	88	110	70	90	116	76	86	118	70	80	112	68
25	MARUTHAMUTHU	M	37	M	68	165	I	8	T6	13	180	14	3	150	210	3	68	130	70	72	126	72	76	120	70	73	116	68	80	118	72	74	116	72	76	116	68	74	118	70

S.NO	NAME	21 min			24 min			27 min			30 min			45 min			60 min			75 min			90 min			105 min			120 min			Side Effects		
		HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	HR	SBP	DBP	Hypotension	Bradycardia	Vomiting
1	JAYARAMAN	82	124	68	84	116	74	82	122	76	80	118	70	86	120	68	84	116	70	90	124	76										**0**	**0**	**0**
2	POUNAMMAL	84	122	74	82	118	72	80	120	72	84	120	72	86	118	70	80	120	76													0	0	0
3	LAKSHMI	92	108	70	94	110	70	90	116	72	92	120	70	94	122	70	92	124	76	90	130	70	84	128	72							1	0	0
4	KALIYAMOORTHY	84	122	80	88	128	74	86	130	72	80	120	80	82	122	84	84	130	80	80	126	72										0	0	0
5	RANJITH KUMAR	88	120	78	84	124	82	82	130	80	80	120	84	80	128	82	82	122	80													0	0	0
6	ULAGANATHAN	92	110	76	84	116	80	88	120	82	84	122	84	80	120	88																0	0	0
7	VIJAYAKUMARI	92	114	76	88	110	74	92	124	72	94	120	70	90	122	82	90	124	80													0	0	0
8	KUMARESAN	83	114	74	79	110	70	85	112	72	88	114	72	78	112	74	87	110	70	83	108	70	86	112	72							0	0	0
9	ILAYARAJA	92	106	68	88	110	70	94	116	78	92	118	82	90	120	86																1	0	0
10	DEVI	94	108	70	97	108	68	90	112	68	88	110	72	87	116	74																0	0	0
11	NATHIYA	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	88	104	70	86	108	70							0	0	0
12	RAJKUMAR	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70	84	114	72				0	0	0
13	PRABHAKARAN	86	114	74	82	112	70	84	110	68	81	112	72	83	116	76	84	108	70	88	110	70										0	0	0
14	DHANALAKSHMI	88	112	70	96	106	68	98	106	70	88	110	72	92	112	74	84	114	70	86	110	70	90	112	70							0	0	0
15	SANJEEVI RAMAN	86	108	68	90	110	68	88	114	70	82	112	70	82	114	74	78	116	72	80	116	78	82	116	80	80	116	82				0	0	0
16	HASEENA BEGUM	92	122	70	88	120	82	94	122	84	92	118	82	90	120	86																0	0	0
17	RENGARAJ	90	114	68	92	116	72	88	114	72	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	90	116	74				0	0	0
18	THENMOZHI	82	114	74	80	110	70	82	112	72	80	110	70	78	114	72	76	116	78	80	116	78	82	120	70							0	0	0
19	MAHALINGAM	84	114	74	86	110	72	91	118	74	93	116	72	88	110	72	90	116	72	88	118	74	82	116	76							0	0	0
20	BALAKANNU	90	112	74	93	116	76	97	116	74	94	118	78	91	114	72	97	114	72	98	116	72										1	0	0
21	MUTHUSAMY	90	116	72	84	118	72	88	114	68	90	114	70	84	116	72	80	118	70	82	116	78	78	116	80							0	0	0
22	CHELLAPPA	90	112	72	94	114	72	98	110	70	88	112	74	90	114	70	84	116	70	88	114	68	90	116	70	82	118	72				0	0	0
23	RAMARAJAN	86	112	72	92	114	70	94	112	70	90	108	68	96	106	68	98	108	72	90	110	70	84	112	72	82	116	78	84	118	72	0	0	0
24	RANJITKUMAR	82	110	70	86	112	68	90	110	70	85	110	72	88	106	70	90	108	68	93	106	72	88	110	72	94	108	72	96	106	76	0	0	0
25	MARUTHAMUTHU	78	116	72	70	112	70	74	116	70	77	110	74	82	112	72	86	110	70	88	108	68	90	108	68	84	110	68				0	0	0