MIDAZOLAM AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR APPROACH OF BRACHIAL PLEXUS BLOCK

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

APRIL - 2011

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

ACKNOWLEDGEMENT

I am greatly indebted to **Dr. SP. Meenakshisundaram, M.D., D.A** Professor and Director, Institute of Anaesthesiology, Madurai Medical College, Madurai for his guidance and encouragement in preparing this dissertation.

My heartful thanks to **Dr. I. Chandrasekaran,, M.D., D.A,** former Professor and Director, Institute of Anaesthesiology, Madurai Medical College, Madurai for his guidance in doing this work.

My sincere thanks to **Dr.S.C.Ganeshprabhu, M.D.,D.A, Dr.T.Thirunavukarasu, M.D.,D.A, and Dr. R. Shanmugam, M.D., DCH.,** Additional Professors of Anaesthesiology, Madurai Medical College, Madurai for their constant support and their able assistance in completing this study.

I also thank my Assistant Professor **Dr.D.S.Sudhakar,M.D.,** for his guidance and postgraduate colleagues, Department of Anaesthesiology, for their kind cooperation for helping me in doing this study.

My profound thanks to **Dr.A.Edvin joe, M.D., Dean,** Madurai Medical College and **Dr.S.M.Sivakumar,M.S., Medical superintendent**, Government Rajaji Hospital, Madurai for permitting to utilize the clinical materials of this hospital in the completion of my dissertation.

I gratefully acknowledge my patients who gave their consent and co-operation for this study.

CONTENTS

INTRODUCTION

“For all the happiness that mankind can gain

it is not in pleasure but in relief from pain”

- JOHN DYRDEN

“Pain, like pleasure is passion of the soul,

that is an emotion and not one of the senses”

- PLATO and ARISTOLLE (ca 375 B.C)

Pain is a fundamental biological phenomenon. The International Association for the Study of pain has defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Pain is always under estimated and under treated. The relief of pain during surgery is the main part of anaesthesia.

In 1784 James Moore used mechanistic concepts to promote neural compression as a useful technique for the provision of surgical anaesthesia.

In 1855, neurologic pain can be treated by circum-neural injection of pain relieving drug. At the same year Gadecke (German) isolated of alkaloid from leaves of coca plant. In 1860 Albert Nieemann was successful in isolating and naming the alkaloid from the leaves of erythroxylon coca.

In 1884 idea of injecting cocaine into nerve introduced by William Halsted and Alfred Hall. After that Heinrich F Braun found that adding

epinephrine to cocaine prolong the effect of local anaesthetics. But later 1911 G.Hirschel performed first percutaneous axillary brachial plexus block.

In 1911 Kullenkampff introduced the classic supraclavicular approach of brachial plexus block. Winnie and Collins introduced the modified lateral perivascular approach. With introduction of barbiturate and cyclopropane, the enthusiasm for block anaesthesia waned in early 1940's. In current recent years however, the technique has had resurgence, due in large part to increased understanding of neural plasticity and the possibility of minimizing hospital stay length by effective use of regional block anaesthesia.

Blocking of the brachial plexus where it is most compactly arranged with less requirement of the anaesthetic solution and rapid onset of action provides ideal conditions for surgery maintains stable intraoperatively hemodynamics, decreases vasospasm, edema and postoperative pain. Because of bupivacaine has long duration of action, it is used most frequently among local anaesthetics for brachial plexus block.

Several adjuvants like opioids, clonidine, neostigmine, hyaluronidase, bicarbonate and dexamethasone found to prolong the analgesic effect without any unwanted systemic effects and reduce the total requirement of local anaesthetic used. Midazolam known to produce antinociception and potentiate the effect of local anaesthetic when given in neuraxial block. It produces this

effect by its action on Gamma Aminobutyric Acid-A (GABA-A) receptors and peripheral nerves contain these receptors.

This study involves the addition of an GABA receptor agonist, midazolam to local analgesic solution in supraclavicular approach of brachial plexus block and the effects are evaluated and compared with brachial plexus block using local analgesic solution alone

AIM OF THE STUDY

To compare the effectiveness of addition of Midazolam as an adjuvant to bupivacaine in supraclavicular approach of Brachial plexus block for prolonging the duration of post operative analgesia and prolonging the sensory blockade.

HISTORY

1858- Theory of pain was a separate and distinct sense was definitely formulated by Mortiz S.Schiff

1884-William Halsted and Alfred Hall – idea of injecting cocaine into nerve trunk

1911- G. Hirschel – first percutaneous axillary brachial plexus block

1911- D.Kulenkampff performed supraclavicular brachial plexus block

1943- Lidocaine was synthesized by Lofgreen and Lundquvisit

1956- Bupivacaine synthesized by Ekenstam

1963- Bupivacaine introduced clinically by Telivuo

1965- Melzock and Walts propounded the Gate Control Theory of pain.

ANATOMICAL CONSIDERATIONS

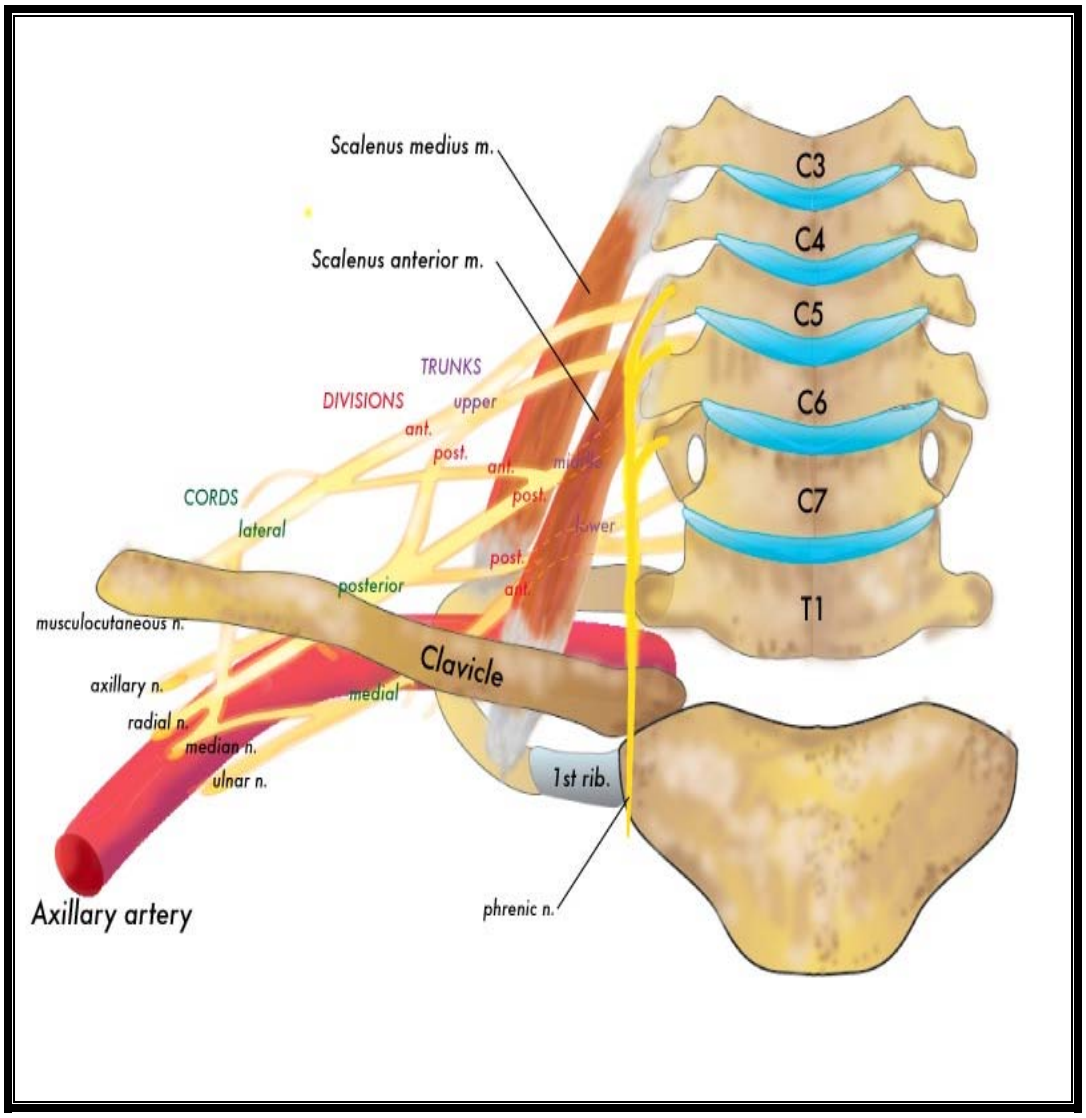
BRACHIAL PLEXUS BLOCK

Knowledge of the formation of the brachial plexus and of its distribution is essential to the intelligent and effective use of the brachial plexus block for the surgeries in the upper limb. Close familiarity with the vascular, muscular and fascia relationship of the plexus throughout the formation and distribution is equally essential to the mastery of various techniques of Brachial plexus Blockade.

Derivation of plexus :

The brachial plexus is derived from the anterior primary rami of the fifth , sixth, seventh , eighth cervical nerves and 1^{st} thoracic nerve, with variable contributions from the Fourth cervical(pre fixed) and second thoracic nerves(post fixed). (figure 1)

ANATOMY OF BRACHIAL PLEXUS BLOCK



COURSE:

After leaving their intervertebral foramina, the roots course anterolaterally and inferiorly to lie between the anterior and middle scalene muscles, which arise from anterior and posterior tubercles of cervical vertebrae respectively. Here unite to form the trunks.

C5, C6 → Superior trunk

C7 → middle trunk

C8, T1 → inferior trunk

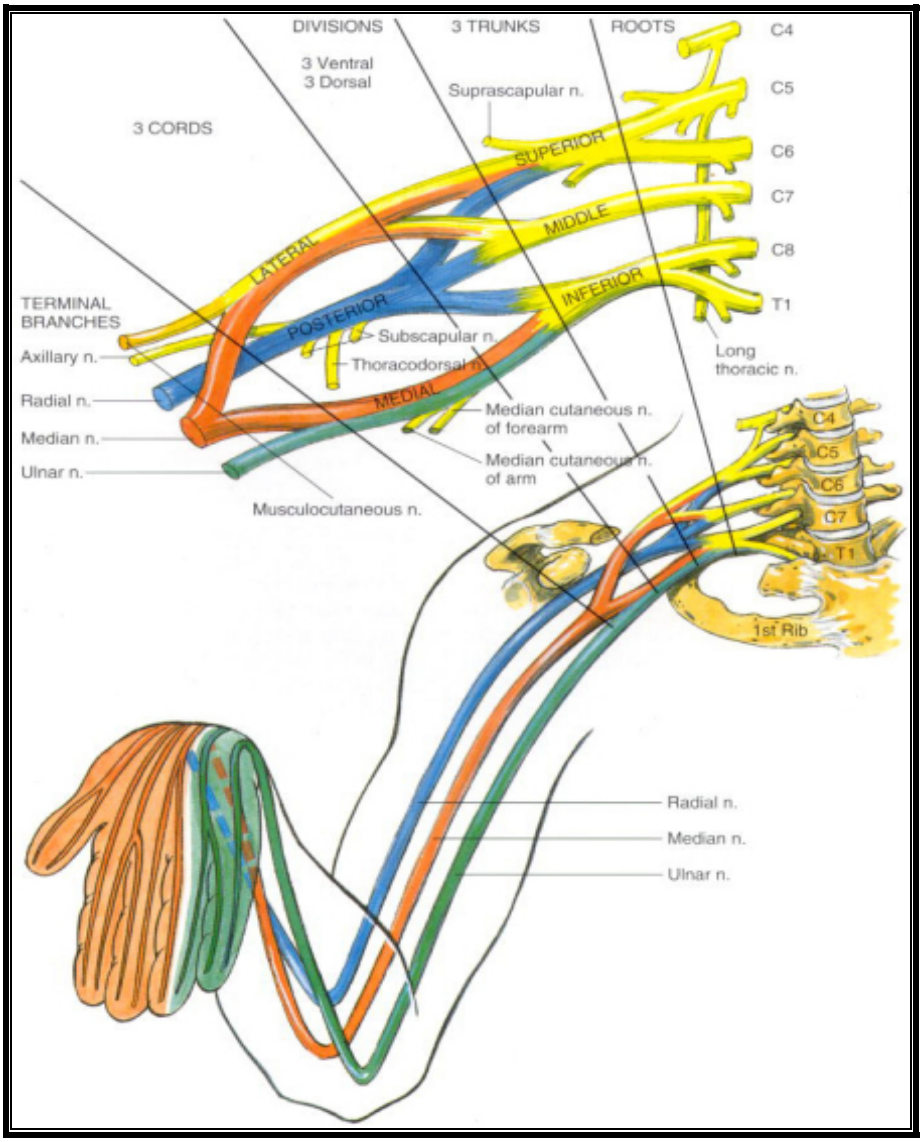
The prevertebral fascia invests both the anterior and middle scalene muscles, fusing laterally to enclose the brachial plexus in a facial sheath. Trunks emerge from the lower border of the muscle running inferiorly and anterolaterally covering towards the upper border of the 1st rib, where they lie cephaloposterior to the subclavian artery.

At the lateral edge of the 1st rib each trunk divides into anterior and posterior divisions passing inferior to mid portion of clavicle. They reunite within the axilla to form the lateral, medial and posterior cords and related to the second part of the axillary artery. The anterior divisions from upper and middle trunk unite to form the lateral cord. The posterior divisions from all three trunks

unite to form the posterior cord. The anterior divisions from the lower trunk continues as the medial cord. **(**figure 2**)**

At the lateral border of the pectoralis minor, the three cords divide into the peripheral nerves of the upper extremity.

BRACHIAL PLEXUS BLOCK COURSE



Lateral cord:

Lateral root of median nerve

Lateral pectoral nerve

Musculocutaneous nerve

Medial cord :

Medial root of median nerve

Medial cutaneous nerve of arm

Medial cutaneous nerve of forearm

Medial pectoral nerve

Ulnar nerve

Posterior cord:

Radial nerve

Axillary nerve

Upper and lower subscapular nerve

Nerve to latissimus dorsi

Branches from roots

Dorsal scapular nerve to Rhomboid muscles (C5)

Nerve to serratus anterior (C5, C6, C7)

Branches from trunk:

Nerve to subclavius (C5-C6)

Supra scapular nerve (C5-C6)

Relations:

Brachial plexus has its roots between the scalene muscles, trunks in the posterior triangle of the neck, divisions behind the clavicle and cords at the level of the Axilla and nerves beyond the axilla. In the course it lies superior and posterior to the subclavian artery. Dome of pleura is anteromedial to the lower trunk and postermedial to the subclavian artery .The trunks emerge between the fascia covering the anterior and middle scalene muscles.

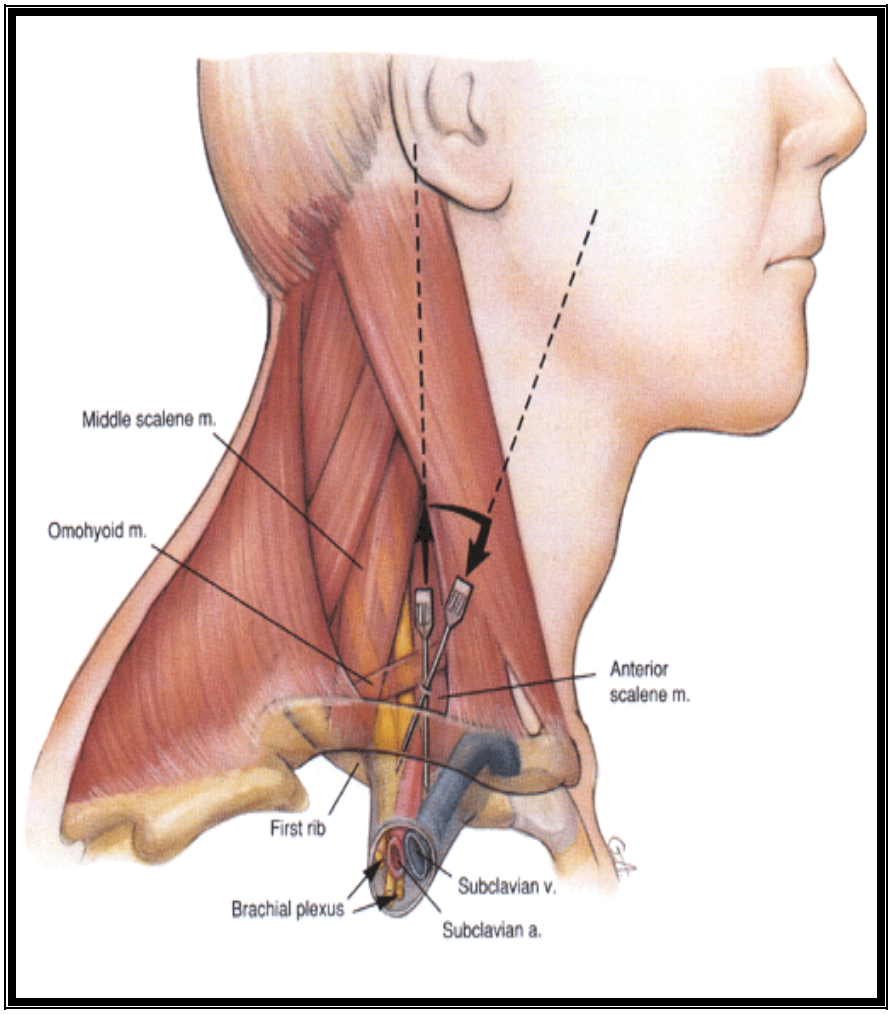
TECHNIQUE OF BLOCKADE

SUPRACLAVICULAR APPROACH OFBRACHIAL PLEXUS BLOCKADE

Classical Approach :

Anatomical landmarks: The three trunks are clustered vertically over the first rib cephaloposterior to the subclavian artery. Neurovascular bundle lies interior to the clavicle at about its midpoint.(figure 3)

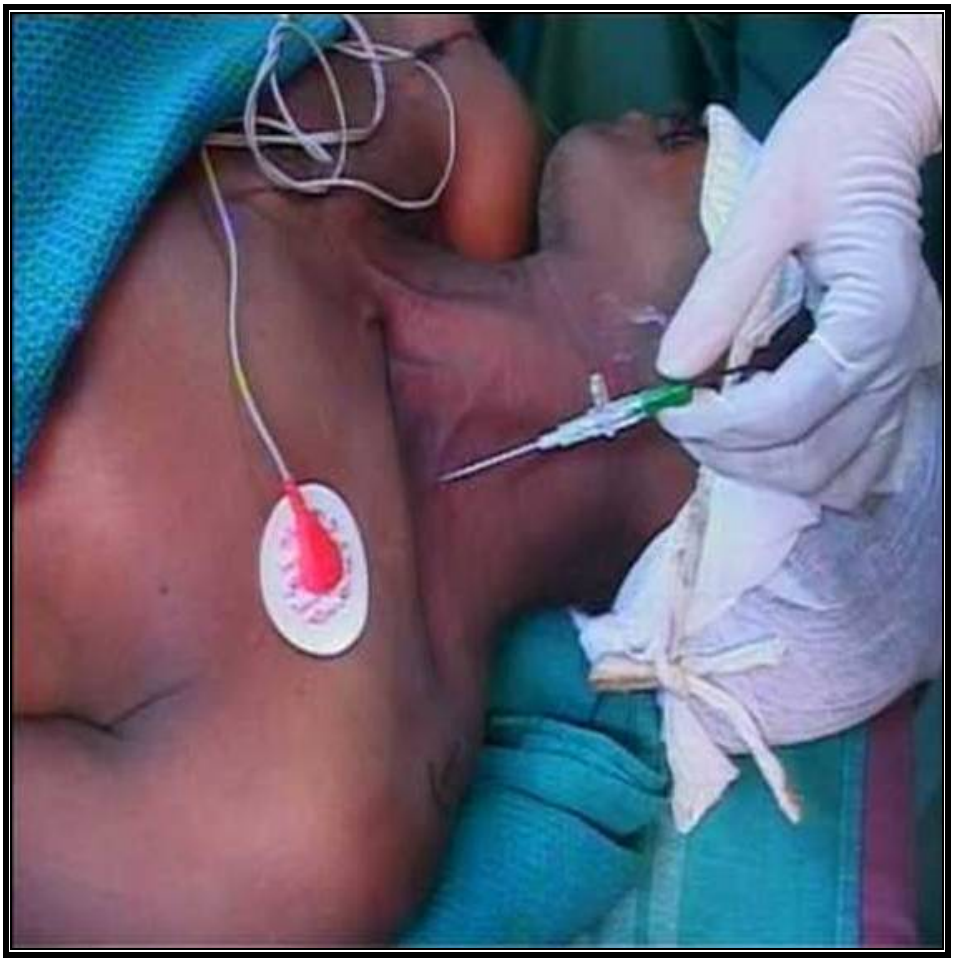
SUPRACLAVICULAR BLOCK



Position of the patient:

Patient is placed in a supine position with the head turned to opposite side from the side to be blocked. The arm is pushed down to depress the clavicle.

TECHNIQUE OF SUPRACLAVICULAR BLOCKADE



Technique (figure 4)

The head is turned slightly to the centralized side and surface landmarks is marked on the skin. The first line is drawn from mastoid process to the sternal insertion of the sternocleidomastoid muscle. The second line is drawn from midpoint of the first line to the midpoint of the clavicle. The point between the clavicular one third and cervical two third of this line is chosen as the needle

insertion site and the area is prepared and draped. A skin wheal performed with 1ml of lignocaine(1%) using a hypodermic needle. A 21 gauge short-beveled and 50mm long Teflon-coated needle is connected to the peripheral nerve stimulator which is set at 1 mA with 2 Hz frequency.

The needle is inserted perpendicular to both sides and directed to the neck at 60^0-70^0 angles to the skin at the sagittal plane until the muscle twitching of forearm is observed. The stimulation is gradually reduced to lower than 0.5mA while still the finger muscle twitching is observed.

The volume of local anaesthetic(0.375% bupivacaine) that is used is 30mL . When large volume are used the sheath may be felt to distend during injection and is easily distinguished from the subcutaneus swelling of an extra facial injection. To encourage the spread proximally distal pressure distal to the needle port may be used and distal pressure proximal to needle insertion point may help to encourage distal spread.

Complications:

Supraclavicular approach has the highest risk of pneumothorax 0.5-6% when compared to other techniques. Majority of pneumothorax takes 24hrs to develop and rarely develop in short duration. Tall thin patients with high apical pleura are prone to develop pneumothorax in shorter duration. Incidence of pneumothorax reduced by avoiding multiple probing and by using small needles.

Unilateral phrenic nerve block can occur but has no significance.

Horner syndrome – occurs when large volume is used, resolves spontaneously.

Unintentional intravascular injection.

Stellate ganglion block and recurrent laryngeal nerve palsies(very rare)

OTHER APPROCHES OF BRACHICAL PLEXUS BLOCK

Interscalene brachial plexus block:

The patient is positioned supine with head rotated 30° or less to the contralateral side. The trunks of the brachial plexus lie in the groove between the anterior and middle scalene muscles. At the level of cricoids cartilage where the external jugular vein crosses, lies the interscalene groove. After a skin wheal, the needle is inserted perpendicular to the skin and advanced in slightly medial and caudal direction until paresthesia or evoked contraction in the arm, wrist or hand is elicited. 30-40 ml of local anaesthetic is injected.

Complications:

Phrenic nerve is commonly blocked, which may lead to respiratory failure. Patients may display a Horner's syndrome (miosis, ptosis, anhidrosis) from stellate ganglion block. Recurrent laryngeal nerve may get blocked leading to hoarseness of voice. Inadvertent epidural, subarachnoid, subdural, subarachnoid or intravascular injection can occur because of the close proximity of these structures.

Infraclavicular brachial plexus block:

The infraclavicular approach blocks the brachial plexus at the level of the cords.

Classical approach: patient lies supine with head turned towards opposite side. The operative limb is abducted to 90° and the axillary pulse identified. 2cm caudal to the mid point of the clavicle, needle inserted at a 45° and directed towards axillary artery pulsation. Motor acivity of the hand is sought with the nerve stimulator and 30-40 ml of local anaesthetic is injected.

Coracoids approach: the patient's arm can be in any position 2cm medial and 2cm caudal to from the coracoids process, the needle is inserted perpendicular to the floor. After eliciting motor response 30-40 ml of local anaesthetic is injected.

Complications:

Pneumothorax, hemothorax and chylothorax are possible and occur at a high rate than supraclavicular approach.

Axillary brachial plexus block:

The patient is positioned supine with the arm abducted, the elbow flexed at 90°, externally rotated at the shoulder leaving the arm lying across the patient's head. Axillary block is performed by one of the following techniques.

Transarterial technique: The pulse of the axillary artery is identified as high as possible in the axilla. The needle is inserted until bright red blood ia aspirated. The needle is slightly advanced or withdrawn until blood aspiration ceases.40 ml of local anaesthetic is injected either anteriorly or posteriorly or 20ml anteriorly and 20ml posteriorly.

Paresthesia technique: the needle is directed toward the axillary artery to elicit a single, specific or multiple paresthesia and the drug is injected.

Axillary artery can also be blocked with the help of nerve stimulator or ultrasound.

Complications:

Complications are very low with axillary approach provided intravascular injection is avoided. Hematoma and infection are very rare.

PHARMACOLOGY OF BUPIVACAINE

Bupivacaine is an amide linked local anaesthetic. It is a hydrochloride salt of d(1)-1-butyl 2'6' pipecoloxylidide and is presented as a racemic mixture.(figure 5)

It was synthesized by Ekenstam in 1957.

First report of its use was published in 1963 by Telivuo.

It is derived from Mepivacaine and is very stable compound and may be autoclaved repeatedly.

Pka	-	8.1
Molecular weight	-	288
Protein binding	-	95%
Lipid solubility	-	28
Elimination half life	-	210mts
Toxic plasma concentration	-	>1.5μg/ml
Approximate duration of action	-	175mts

Availability:

Ampoules - 0.5% Bupivacaine hydrochloride 4cc

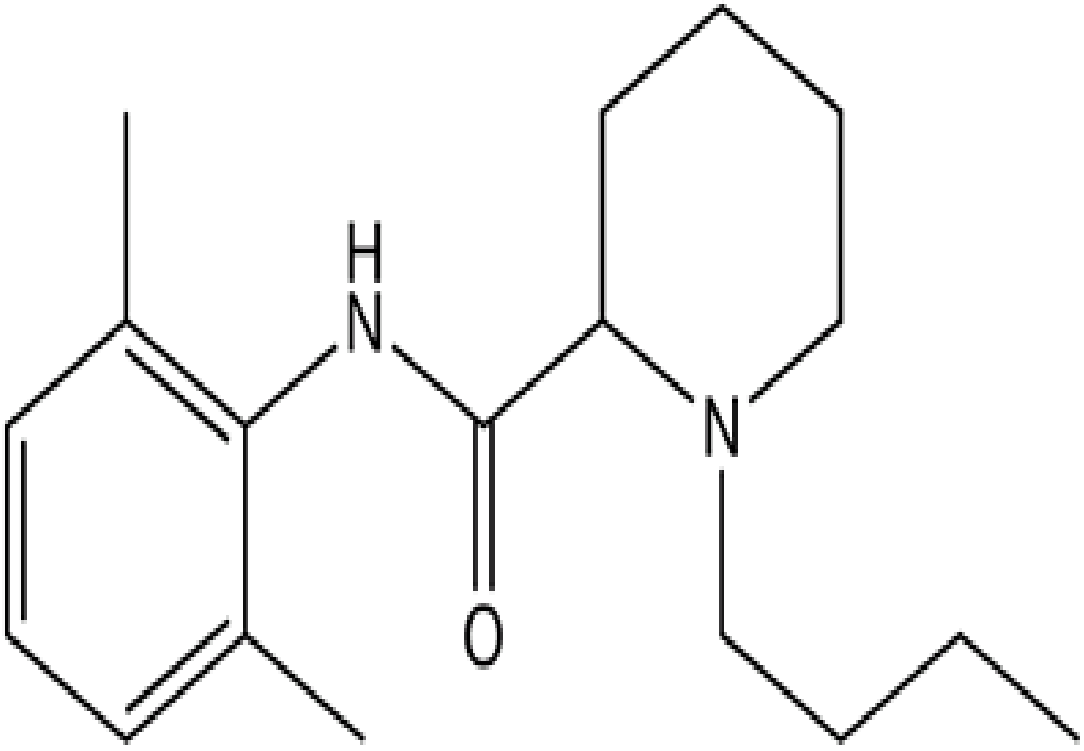
0.5% Bupivacaine hydrochloride with dextrose

(heavy) 4cc

Vials - 0.25% and 0.5% Bupivacaine hydrochloride 20cc

Dosage - Maximum dosage 3mg/kg body weight.

STRUCTURE OF BUPIVACAINE



Uses:

Spinal anaesthesia

Epidural anaesthesia

Caudal anaesthesia

Continuous epidural anaesthesia

Peripheral nerve block

Onset time and duration of action

Site of action	Onset (minutes)	Duration (minutes)
Intrathecal	5	180-240
Epidural	15-20	165-225
Brachial plexus	10-20	600

Pharmacokinetics:

Once injected intrathecally, it gets absorbed by the nerve rootlets and results in the desired effect. It is rapidly absorbed from the site of injection, but the rate of absorption depends on the vascularity at the site and presence of vasoconstrictors.

High lipid solubility of bupivacaine makes it easy for nerve and vascular tissue penetration. 80-95% of the absorbed bupivacaine binds to the plasma.

Distribution:

Rapid distribution phase: (α)

In this phase the drug is distributed to highly vascular region t½ of α - being 2.7 minutes.

Slow disappearance phase: (β)

In this phase the drug is distributed to slowly equilibrating tissues $t1/_2$ of β – being 28minutes.

Biotransformation and excretion phase : (δ)

$T1/_2$ of δ is 3.5hours. Clearance is 0.47 litre/minute.

Biotransformation:

Possible pathways of metabolism of bupivacaine include aromatic hydroxylation and conjugation. Only the N-dealkylated metabolite,

N-desbutyl bupivacaine has been measured in blood (or) urine after epidural (or) spinal anaesthesia. Alpha-1 acid glycoprotein is the most important plasma protein binding site of bupivacaine and its concentration is increased by many clinical situation including post operative trauma.

Excretion:

It is through the kidney, 4-10% of the drug is excreted unchanged.

Mode of Action:

a) Site of action:

Peripheral nerve rootlet fine nerve filaments

The spinal nerve rootlet fine nerve filaments having a large surface area are exposed to the local anaesthetics

Posterior and lateral aspects of the spinal cord itself.

b) Sodium Channel blockade:

They impede sodium ion access to the axon interior by occluding the transmembrane sodium channels thus delaying the process of depolarization and axon remains polarized. It is a non-depolarization blockade.

Pharmacodynamics:

It has got a longer duration of action but a slower onset.

Cardio vascular system:

It reduces cardiac output by reducing the sympathetic tone, by slowing the heart rate and by reducing the venous return, it produces a fall in arterial blood pressure but it is relatively slow and is seldom very profound.

It produces a fall in central venous pressure. It causes an increase in lower limb blood flow. It causes a reduction in incidence of deep vein thrombosis.

Respiratory System:

Spinal blockade seldom, if ever causes respiratory problem.

Gastro intestinal tract:

There is an increase in gastro intestinal motility and emptying of the gastric contents are better.

Toxicity:

Toxicity is related to plasma level of unbound drug and more likely due to an inadverdant intravenous injection. Systemic toxicity reactions primarily involve central nervous system and cardio vascular system. The blood level required to produce central nervous system toxicity is less than that required to produce circulatory collapse.

Central Nervous System Toxicity:

Initial symptom includes feeling of light headedness and dizziness, followed by visual and auditory disturbances. Objective signs are excitatory and includes shivering, muscle twitching and tremor. Ultimately generalized tonic, clonic seizure occurs.

Cardiovascular System Toxicity:

The rate of depolarization in fast conducting tissue of purkinje fibres and ventricular muscle is decreased. The rate of recovery of bupivacaine induced block is slower than that of lignocaine. Extremely high concentration of the drug causes sinus bradycardia and cardiac arrest.

PHARMACOLOGY OF MIDAZOLAM

History:

It is the first clinically used water soluble benzodiazepine

Fryer and Walser synthesized it on 1976

It was the first benzodiazepine that was produced primarily for use in anaesthesia

Structure:

Midazolam is a water soluble solution containing 1(or) 5mg/ml of midazolam with benzylalcohol as preservative. Preservative free midazolam is also available.

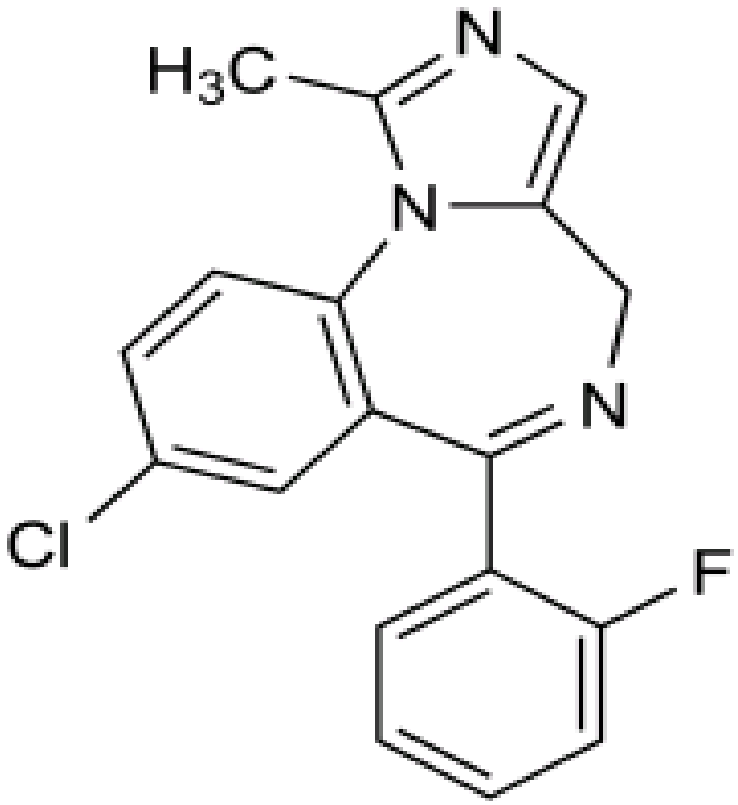
Imidazole ring of its structure, accounts for its stability in solution rapid metabolism and water solubility.(figure 6)

pH of the parenteral form of midazolam is 3.5.

It has pH dependent solubility i.e if pH is more than 4 it is lipid soluble and if pH less than 4 it is water soluble.

Molecular weight of midazolam is 362. pKa : 6.2

STRUCTURE OF MIDAZOLAM



Metabolism:

In liver midazolam is biotransformed to 1 and 4 hydroxymidazolam by oxidative pathway by cytochrome p450 (cyp 3A4 enzyme) 1 hydroxy midazolam is the principal metabolite and has Y2 the activity of parent compound. These metabolites are active ones. But these are rapidly conjugated to glucouronic acid and excreted in urine more rapidly than midazolam. So there will be no prolonged sedation on single dose.

Pharmacokinetics:

Midazolam is extensively bound to plasma protein, about 95% bound to albumin.

Its volume of distribution is 1.1-1.7 L/kg.

Elimination half life is 1.7-2.6 hour.

Clearance is 6.4-11 ml/kg/min.

Plasma level required for hypnosis and amnesia dring surgery are 100-200ng/ml.

Awakening usually occurs at a level lower than 40ng/ml.

Increasing age and obesity prolong the elimination half life.

Pharmacokinetic of oral midazolam:

It is rapidly absorbed from gastro intenstional tract and 50% reached the circulation reflecting substantial hepatic first pass effect.

Mechanism of action:

Midazolam, interaction with $(GABA)_A$ BZD receptor ð< subunit, causes chloride channel opening which increases chloride ion conductance which causes hyperpolarization and therefore resistance to neuronal transmission.

Various effects of benzodiazepines is related to amount of receptor occupancy which corresponds to plasma concentration.

If receptor occupancy is 20%, it causes anxiolysis.

If receptor occupancy is 30-50%, it causes sedation.

If receptor occupancy is >60%, it causes unconsciousness.

Pharmacological action:

Onset	:	Intravenous	:30-60 sec
	:	Oral	:15-30sec
Duration	:	Intravenous	:30-60min
	:	Oral	: 45-90min

Effect on central nervous system:

Midazolam produces sedation, anxiolysis, anticonvulsant effect, muscle relaxation and unconsciousness. These effects are dose dependent according to percentage of receptor occupancy. It produces both antero grade amnesia, dose related reduction in cerebral blood flow and $CMRO_2$. Its cerebral protective effect is superior to diazepam but inferior to barbiturate. Central vasomotor response to CO_2 is presumed. It does not prevent increased in ICP following trachea incubation. It's a potent anticonvulsant.

Effect on respiratory system:

Midazolam produces dose related ventilator depression which is greater than with other benzodiazepines. It is more pronounced by intravenous route following fast administration along with opioids but insignificant when given through other routes (oral).

Onset of respiratory depression is rapid within 3 ninutes and the action lasts longer for even 60-120 minutes on intravenous administration.

Slope of ventilator response curve to carbondioxide are flatter than normal.

Incidence of apnea induction is similar as with thiopentone which is greater in old age, debilitated state COPD patients and in presence of other respiratory depressant drugs.

Effect on cardiovascular system

It decreases arterial pressure by decreasing systematic vascular resistance which is dose dependent and greater than with other benzodiazepines but similar to thiopentone.

It produces variability in heart rate changes because it impairs baroreceptor and also decreases the vagal tone.

Effect on foetus:

Midaolam has less placental transfer than other benzodiazepines but produces greater neonatal depression than thiopentone and propofol.

Drug interaction:

Erythromycin inhibits the metabolism of midazolam and causes two to three fold prolongation and intensification of its effects.

Antifungal agents like itraconazole and ketaconazole increase the serum concentration of midazolam.

Calcium channel blockers inhibit cytochrome P450 enzymes leading to central neurons system depression.

Cimetidine inhibits metabolism of midazolam, but it id greater with diazepam than midazolam.

Ethanol, barbiturates and other central nervous system depressant drugs potentiate the sedative effects of midazolam.

It reduces the minimum alveolar concentration of volatile agents as much as 30%.

Hepatic clearance inhibited by fentanyl

Hepatic clearance is 5 times greater than lorazepam and 10 times greater than diazepam.

Routes of administration:

Commonly used route is intravenous. Other available routes are intramuscular route sedation, oral route for premedication, intra nasal and rectal routes premedication in children.

Dosage:

Oral	:	0.5-0.7mg/kg
Rectum	:	0.25-0.5mg/kg
Intra nasal	:	0.2-0.5mg/kg
Intramuscular	:	0.05-0.15mg/kg
Intravenous	:	0.05-0.15mg/kg
Sublingual	:	0.1mg/kg

CLINICAL USE:

1) **Premedication:**

Midazolam is an useful premedication because of various available routes of administration and its sedative, anxiolytic, amnestic action.

Midazolam is an useful premedicant in children, especially in the oral formulation which was approved by the US food and drug administration in 1998.

Oral route of administration in children is very popular because of its easy administration, but the problem is its bitter taste, which can be minimized by sugar solution.

2) **Intravenous sedation:**

Midazolam is an effective intravenous sedative for therapeutic procedures and regional anaesthesia.

It is also useful in painless producers like cardio version and electroconvulsive therapy.

Its advantages over other benzodiazepines are : it is water soluble, has less or no venous irritation, rapid onset, short duration and less postoperative sedation.

3) **Induction and maintenance of anaesthesia:**

It is the benzodiazepine of choice as an induction agent.

Intravenous administration of midazolam in doses of 0.2-0.3mg/kg over 30-60 sec will produce induction of anaesthesia which is 50-100% slower than with thiopentone

It is used to implement opioids (or) inhaled anaesthetics during maintainance of anaesthesia. It reduces the anaesthetic requirement of halothane by 30%

4) Paradoxical vocalcord motion:

Non organic upper airway obstruction (shorter dose:0.5-1mg/v) midazolam is an effective treatment

5)Treatment of grandmal seizures:

Which occurs with systemic toxicity due to local anaesthetics.

6) Epidermal block midazolam as an sole agent or in combination of local anaesthetic to provide excellent analgesia in labour analgesia epidermal midazolam is also unresponsive to maximum use oral or epidermal as do patients reflex sympathetic dystrophy neuropathic pain.

7) Spinal anaesthesia midazolam combined to local anaesthetic improves the quality and duration of the block, minimize the tourniquet pain during lower limb surgery

8) Caudal anaesthetic:

Midazolam combined to local anaesthetics increases the duration of anaesthesia and analysis by 2-3 lines without hemodynamic side effect.

9) Peripheral nerve blocks:

Midazolam prolongs the duration of anaesthetics and analysis of local anaesthetics.

10) Used in acutely agitated psychiatric patient.

NERVE STIMULATORS

History :

The first description of electrical stimulation to locate the brachial plexus was recorded by Perthes in 1912. However, the acceptance of peripheral nerve blocks was not realized until the 1960s when electronic advances and the consequent introduction of more convenient solid-state units were made. Greenblatt and Denson have demonstrated that motor nerves can be stimulated without eliciting pain and that the current required to stimulate the nerve depends on the distance between the needle and the target nerve.

The nerve stimulator is an excellent solution to reduce the dependency of the anaesthesiologist on anatomical knowledge and variations, and also increases the success rate compared to the paraesthesia technique. It is helpful in defining anatomical land marks for the entry point, especially in growing children ,obese patients and in case of distorted anatomy.

Components:

The nerve stimulator case with an on/off switch and a dial selecting the amplitude of the current. Two leads to complete the circuit. One is connected to

an ECG skin electrode and the other to the locating needle. The polarity of the leads should be clearly indicated and colour-coded with the negative lead being attached to the needle.

Mechanism of action:

A small current (0.25-0.5mA) is used to stimulate the nerve fibres causing the motor fibre to contract.

The frequency is set at 1-2Hz Tetanic stimuli are not used because of the discomfort caused.

The duration of the stimulus should be short (1-2ms) to generate painless motor contraction.

The nerve stimulator is battery operated to improve patient safety.

Nerve location can be very accurately defined especially when low currents are used. The success rate of technically difficult nerve blocks can be increased by using a nerve stimulator. A sciatic nerve block with a success rate of over 90% can be achieved in experienced hands, compared to about 50% without using a nerve.

Nerve blocks can be performed while the patient is anaesthetized or heavily sedated as the response is visibly monitored with no need to elicit paraesthesia. However, the use of neuromuscular blocking agents will abolish any muscular contractions

Problems in practice and safety features:

Higher currents will stimulate nerve fibres even if the tip of the needle is not adjacent to the nerve. The muscle fibres themselves can also be directly stimulated when a high current is used. In both situations the outcome will be an unsuccessful block once the local anaesthetic solution has been injected.

It is not recommended to use nerve stimulator designed to monitor the extent of neuromuscular blockade for regional nerve blocks. These are high output devices which can damage the nervous tissue.

It should be remembered that using the nerve stimulator is no excuse for not having a sound knowledge of surface and neuroanatomy required to perform regional anaesthesia.

Nerve stimulator for nerve blocks:

It has two leads, one to the skin and the other to the needle.

A small current of 0.5mA or less is used with a frequency of 1-2Hz.

The stimulus is of short duration (1-2ms)

REVIEW OF LITERATURE

1. Mohammad Need Khan, Noreen Laiq, Mohammad Arif and Shahid Khan ; **Journal of the college of physicians and surgeons Pakistan 2008**,vol 18(11):674-678 Midazolam with bupivacaine for improving analgesia quality in brachial plexus block for upper limb surgeries. In this study 50 patients were randomly allocated in two groups. So that 25 patients (A group) received 30mL of 0.5% bupivacaine. Other 25 patient (B group) received 30mL of 0.5% bupivacaine plus 0.5% of preservative free midazalom 50µ/kg. Onset of sensory and motor block appeared earlier in group B than group A($p<0.001$).In B group the onset of sensory block occurred in 14±3.1 minutes compared to 22±3.5 minutes in group A. Onset time of moor block in group B was 10.5±2.40 minutes compared to 18.5±3.50 minutes in group A. The mean duration of complete analgesia was significantly prolonged($p=0.002$) in the bupivacaine midazolam group. Sedation scores were higher in group B compared to group A($p<0.001$) They concluded that Combination of midazolam 50µ/kg + 30mL of 0.5% bupivacaine for supraclavicular quickened the onset of sensory and motor block and also improved quality of analysis as manifested by lower pain scores , a prolonged effect and reduced requirements for rescue analgesia.

2. Koj J, Yalindra KB, Nidhi B.P **canadian journal of anaesthesia 2005/52:8/PP822-826** brachial plexus block of midazolam bupivacaine improves analgesia. In this study 40 patients ASA I & II group B 30mL of 0.5% bupivacaine and group BM 30mL of bupivacaine with midazalom 50μ/kg. In group BM, onset of sensory block occurred in 12 ± 2.9 minutes compared to 20±3.8 minutes in group B. onset time of motor block in group BM was 9.2 ± 2.38 minutes compared to 17.1 ± 3.83 minutes in group B. in both the groups motor block occurred earlier than sensory block but the duration of sensory block was not different between the two groups. Lower pain scores were observed in group BM compared to group B. all patients in group B required rescue analgesia, while only 3 patients of group BM required rescue analgesics. The number of rescue analgesic doses required was significantly higher in group B compared to group BM. Sedation scores differed between the groups as patients in group B were all awake throughout the intraoperative period, while in group BM, 4 patients at 10 minutes and 7 patients at 20 minutes were sedated and responded to verbal stimulation. No patients in group BM required assistance for airway maintenance due to sedation

3. M. H. Kim and Y.M.Lee **British journal of anaesthesia 2001. 86 (1): 77-79.** Intrathecal midazolam with bupivacaine increases the analgesic effects of spinal anesthesia for hemorrhoidectomy : 45 pts were studied as 3 groups.

control group 1ml of bupivacaine+0.2 ml of NS, study group BM1 1 ml of bupi+0.2mlof midazolam(1mg),BM2 group 1ml of bupivacaine +0.4ml of midazolam (2mg)). In this study they concluded that total rescue analgesic requirements were less in study groups than control groups.

4. Min Soo Kim, M.D., Bum Sang Hwang, M.D., Byeong Mun Hwang, M.D., Seong Sik Kang, M.D. **Korean journal of Anesthesiology 2008 feb:54(2)-164-172** This study was conducted to evaluate the effects of fentanyl and midazolam when used as adjuvant in a supraclavicular brachial plexus block. 100 adult patients with an ASA status of I-II performed under a supraclavicular brachial plexus block were prospectively evaluated in this study. The patients were randomly divided into 4 study groups: Group 1, which received 40 ml of 1.5% lidocaine, Group 2, which received 3 mg of midazolam with 40 ml of 1.5% lidocaine, Group 3, which received 100 μg of fentanyl with 40 ml of 1.5% lidocaine, and Group 4, which received 3 mg of midazolam and 100 μg of fentanyl with 40 ml of 1.5% lidocaine. In this study they concluded that although the addition of 3 mg of midazolam and 100 μg of fentanyl to lidocaine in a supraclavicular brachial plexus block does not affect the onset of sensory or motor block, it does prolong the duration of analgesia and motor block.

5. Mahajan et al **British Journal of Anaesthesia November 2010 volume 91.** In this study they observed clinically and statistically prolonged postoperative

analgesic effects in bupivacaine midazolam group compared to plain bupivacaine caudal block.

6. Prakash and Joshi **Reginonal Anaesthesia and Pain Medicine volume 31 issue 3.** They used two different concentrations of midazolam and demonstrated prolonged analgesia without any unwanted effects with higher concentrations. No evidence of neurologic or urologic symptoms have been observed by administrations of 2 mg intrathecal midazolam. In this study midazolam was used at a dose of 50μg-kg^{-1} without any significant adverse effects.
7. Gulec et al **European journal anaesthesia 1998:15 161.5.** Bupivacaine with midazolam to provide prolonged postoperative analgesia compared to bupivacaine-morphine combination when given caudally.
8. Batra and Tomoki **Anaesthesia and Analgesia 2003 may 96(5) 1386-91.** They obtained effective analgesia with intrathecal midazolam without observing any adverse effects. The mechanism by which midazolam causes prolonged analgesia could be due to its action on GABA-A receptors present in the brachial plexus and producing antinociception. The action of midazolam on GABA receptors is well-established. Various researchers have demonstrated the presence of GABA receptors in peripheral nerves and presence of GABA receptors within the temporomandibular joint

9. Henry Iskander et al **Anaesthesia and Analgesia 2003 April volume 96 No.:4-982-986.** Suprascapular nerve block improves analgesia postoperatively for arthroscopic shoulder surgery performed under GA.50 patients ASA I&II patients were selected and were given suprascapular block with GA in one group and only GA in other group. The suprascapular nerve can performed and selected with 0.03μ/kg of midazolam . In those who under were suprascapular nerve block along with GA post operative analgesia prolonged.

10. Feld and coworkers, **Anesthesiology 1990,73: 831-834.** Efficacy of oral midazolam premedication in children. Efficacy of oral midazolam premedication in children was compared with different doses of midazolam, 0.25mg,0.5kg,o.75 and placebo and observed that Midazolam o.75mg/kg produced significant sedation at 30 min after premedication and decreased anxiety on separation from mother. After surgical procedure lasting 1 to 3 hrs recovery was not prolonged by oral midazolam. In this study concluded that oral midazolam 0.5 to 0.75mg/kg is an effective premedication for children undergoing outpatient surgery. Midazolam when administered orally should be mixed with a sweetener such as oral acetaminophen because it is bitter.

11. Warner DL, Carbret J, Velling D 1995 **Paediatric Anaesthesia, France** . Ketamine plus midazolam a most effective pediatric oral premedicant Healthy children between 1.5 and 7 years old divided groups of 20 each. Group 1 received midazolam 0.5mg/kg, Group II ketamine mg/kg and Group

III a mixture of midazolam 0.4mg/kg, ketamine 4mg/kg with atropine 0.02mg/kg given orally 20-30min prior to surgery. The children at the time of parental separation and again when mask-induction was begun and observed that ketamine along with midazolam has 100% success rate on parental separation and mask induction. In this study concluded that indicate that mixture of ketamine and midazalom is the mask effective predictive oral premedicant.

12. Bano F, Haider S, Sultan ST **Department of anesthesiology and surgical I.C.U, Dow medical college and civil hospital, Karachi**. To compare the duration and side effects of postoperative analgesia of caudal bupivacaine and bupivacaine-midazolam mixture. In this study sixty children, aged 1-8 years ASA physical status I and II undergoing inguinal and urogenital surgery were randomly allocated to receive either 0.25% bupivacaine 0.75ml/kg(group A)or 0.25% bupivacaine 0.75ml/kg along with 0.1% midazolam 50mg/kg(group B) by caudal route immediately after induction of general anesthesia. Anesthesia was maintained till the end of surgery and observed that the duration of analgesia was 21.41+/-2.7 hours in bupivacaine midazolam group and 9.97+/-2.25 hours in bupivacaine group which showed a significant difference ($p<0.001$). There was no significant difference in heart rate, respiratory rate, blood pressure and the incidence of side effects in both groups ($p=0.716$). The sedation score were significantly

higher in bupivacaine-midazolam group during first hour postoperatively(*p=0.003).In this study concluded that addition of midazolam to caudal bupivacaine provides longer duration of postoperative analgesia without having significant side effects but with higher sedation score for 1hour postoperatively.

13. **European journal of anaesthesiology (2003), 20:11:904-910.** Improvement in postoperative pain relief by the addition of midazolam to an intrathecal injection of buprenorphine and bupivacaine. In this study was prospective, randomized, and observer blinded. It involved 60 patients (30 per group),ASA I and II age 20-40 yr, undergoing minor and intermediate lower abdominal surgery under spinal anaesthesia. Patients were randomized in to two groups: the control group received a spinal injection of hyperbaric bupivacaine (15 mg) plus buprenorphine (0.15mg) and the experimental group injection of the same two drugs and doses but supplemented with intrathecal midazolam(2 mg) and observed that the duration of postoperative analgesia in the control group was 9.24 ±2.57 hr (mean±SEM) and 21.33±12.69 hr in the midazolam treated group ($p<0.001$). Patients treated with intrathecal midazolam had better pain relief judged by visual analogue score on coughing ($p=0.0013$) and a nursing mobility score ($p<0.00001$). Adverse effects were minor and their incidence was similar in both groups. In this study concluded that we

conclude that intrathecal midazolam 2mg improves the quality and duration of postoperative pain afforded by intrathecal buprenorphine and bupivacaine.

14. Reeves JG, Fragen RJ, Vinik HR, Greenblatt DJ, **Anesthesiology 1985;62: 310-24** Midazolam: It is an imidazobenzodiazepine with unique properties, it is a water soluble in its acid formation but is highly lipid soluble in vivo. Drug produces hypnosis, amnesia, antianxiety. Uses include premedication, induction of anaesthesia and maintenance sedation for diagnostic and therapeutic procedures.

MATERIALS AND METHODS

This study was conducted at Government Rajaji Hospital attached to Madurai medical college. 60 patients of ASA grade I or II of either sex and age more than 20 years undergoing upper limb surgery (mostly orthopedic and plastic surgeries) included, patients allergic to local anaesthetics and any contraindication to midazolam were excluded from this study. Patients were randomly allocated into two groups BM or B. Each group comprises of 30 patients surgery was done under supraclavicular approach of Brachial plexus Block.

Procedure

After ethical committee approval, informed consent was obtained from the patients. No premedication was given to the patients. Intravenous access was obtained, anaesthesia machine checked, resuscitative equipments and drugs were kept ready. Supraclavicular block was performed by subclavian perivascular by using the peripheral nerve locator.

In GROUP BM: Patients received supraclavicular block with 30ml of 0.375% Bupivacaine + 50 microgram/kg of preservative-free midazolam.

In GROUP B : Patients received supraclavicular block with 30ml of 0.375%Bupivacaine alone

Care was taken so that the toxic dose of the local anaesthetics were not exceeded according to the weight of the patients.

PARAMETERS OBSERVED

1.Onset of sensory blockade

Onset of sensory blockade was taken as abolishment of pins prick pain over the distribution of ulnar and median and was assessed every minute after the performance of the block.

2. Onset of motor blockade:

Onset of motor blockade was assessed every 2 minute after the block using four point scale

	score
Normal power	0
Weakness but able to move arm	1
Not able to move arm but the fingers	2
Complete motor Blockade	3

Attaining a score of 2 was considered as the onset of motor Block.

3.Duration of surgery :

4. Duration of motor blockade:

When (3) in the four point scale changes to (2) the motor blockade is said to reverse. The duration of motor block is noted from the time from scale (3) to scale (0).

5. Duration of sensory blockade:

6. Duration of analgesia :

The pain was assessed using Visual Analogue scale having 10cm length numbered from 0 to 10. Patient was explained about the visual Analogue scale as 0 - No pain and 10 the worst possible pain and was asked the score in visual analogue scale.

The patient was observed every 30 minutes after the surgery is over till the motor block reverses and thereafter hourly for 6 hrs; 2 hourly for next 6 hrs and than of 24 hours.

a) Time of which VAS score is greater than 5 is noted and patient was given intramuscular NSAID (Injection – Diclofenec)
b) Duration of post operative analgesia; the period of time after the surgery till the patient needs analgesic (VAS score more than 5)

6. Vital parameters;

Pulse rate,

Blood pressure,

Respiratory rate are monitored periodically.

7. Sedation score :

Brain & Ready score was employed

0 - Fully awake

1 Drowsy

2 Drowsy but arousable on touch or call

3 Drowsy but arousable on deep stimuli

4 somnolent

8. Side effects noted are

sedation,

hypotension,

bradycardia.

Patients in whom the block was unsuccessful due to total failure of missed dermatomes which needed intravenous supplementation or general anaesthesia were excluded from the study.

OBSERVATIONS AND RESULTS

This study comprised of two groups. The patients in group BM received 0.375% Bupivacaine 30ml + 50µ/kg preservative free midazolam. In group B received 0.375% Bupivacaine 30ml alone.

Age & Weight :

Age distribution in the group BM varied from 15 years to 60 years with mean age of 33.6 years and standard deviation of (12.4).

In bupivacaine group (group B) Age varied from 16 years to 75 years with mean value of 39.5 years and standard deviation of (14) as shown in table 1 and figure 8. There is no significant difference in both groups ('p' value 0.1131, >0.05)

Weight of the patients in the group BM had a mean value of 52.3 kg with standard deviation of 4.4. In bupivacaine group mean value of 54.6 and standard deviation of 5.3 as shown in Table 2 and figure 9. so there is no significant difference in both groups ('p'= 0.0721, >0.05)

Table - 1

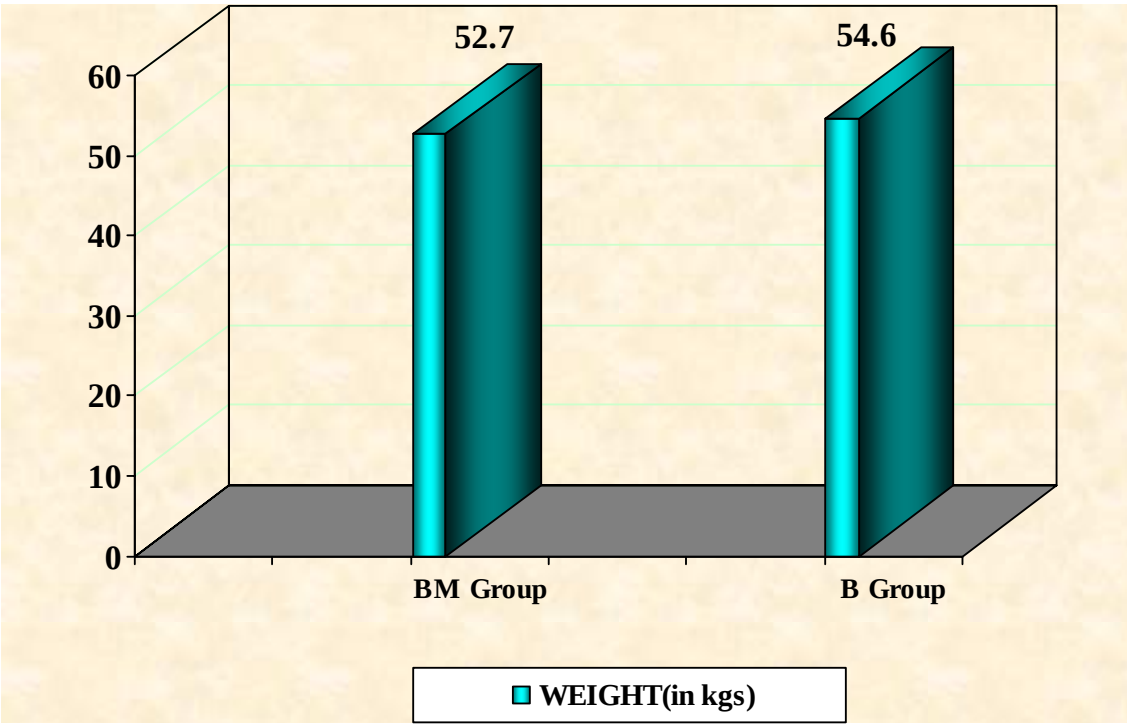
Age Distribution

Age in hours	BM Group		B Group	
	NO	Percentage	NO	Percentage
Up to 20 years	4	13.3	1	3.3
21-30 years	11	36.7	6	20
31-40 years	7	23.3	12	40
41-50 years	6	20.0	5	16.7
Above 50 years	2	6.7	6	20
Total	30	100	30	100
Range	15-60 Years		16-75 Years	
Mean	33.6 Years		39.5 Years	
SD	12.4 Years		14 Years	
'p'		**0.1131**		

Table 2

Weight Distribution

Weigh(in Kgs)	Group BM (No of patients)	Group B (No of patients)
Range	45-60	42-60
Mean	52.7	54.6
S.D	4.4	5.3
'p'	**0.0721**	



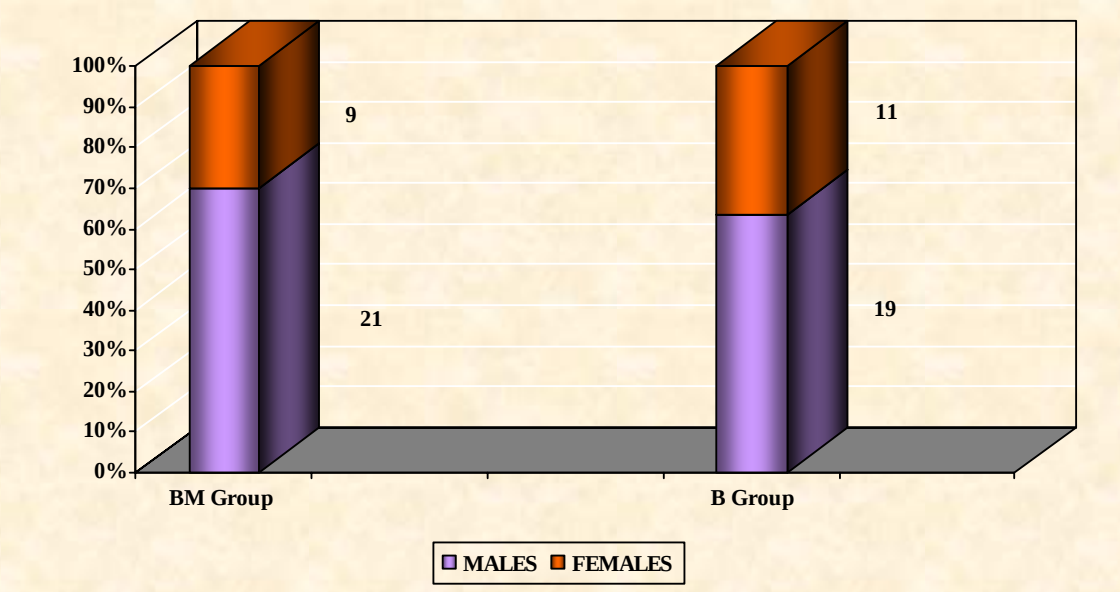
Sex :

In group BM 21 patients were male and rest were female. In group B 19 patients were male and rest were female so there is no significant difference in both groups ('p' value 0.7842, >0.05)(Table 3, figure 10)

Table 3

Sex Distribution

Sex	BM Group		B Group	
	No of patients	%	No of patients	%
Male	21	70	19	63.3
Female	9	30	11	36.7
Total	30	100	30	100
'p'		**0.7842**		



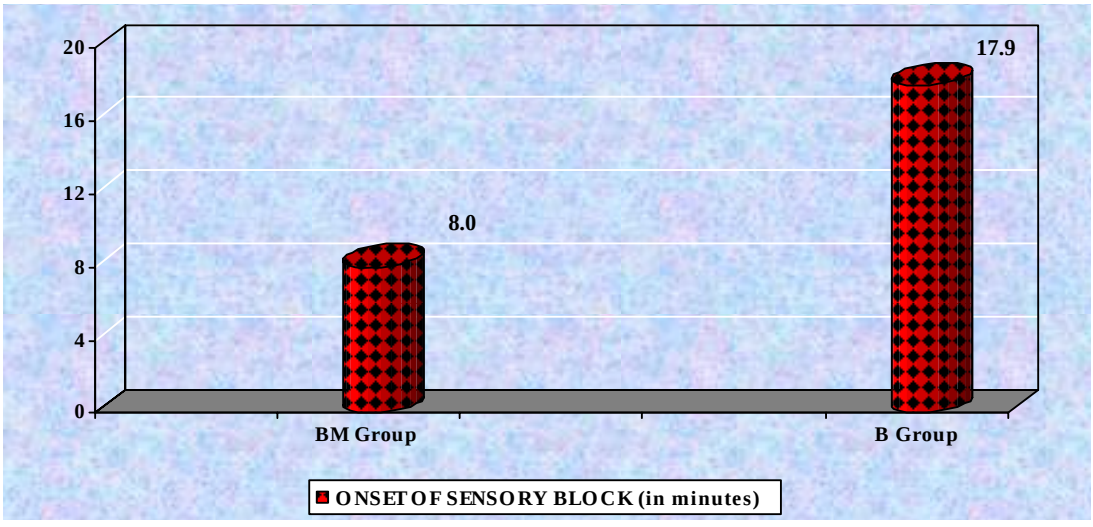
Onset of Sensory Block :

Time taken for the onset of sensory blockade in group BM varied from 8 minutes to a maximum of 10 minutes with mean values of 8 minutes with standard deviation of 0.8minutes. In group B it varied from 15 minutes to 19 minutes with mean value of 17.9 minutes with standard deviation of 0.8 as shown Table 4 ,figure 11

Table – 4 **Onset of sensory Block**

Onset of sensory block	Group BM	Group B
Range	8-10 minutes	15-19 minutes
Mean	8.0 minutes	17.9 minutes
S.D	0.8 minutes	0.8 minutes
'p'	**0.0001**	

The onset of sensory block was significantly faster in midazolam group ('p' value 0.0001, <0.05).



Onset of motor block :

Onset of motor block varied from 13 minutes to 20 minutes in the group BM with mean 16.4 minutes and standard deviation of 1.7.

In group B it varied from 20 minutes to 25 minutes with a mean of 21.8 minutes and standard deviation of 1.5 as shown in Table 5, figure 12.

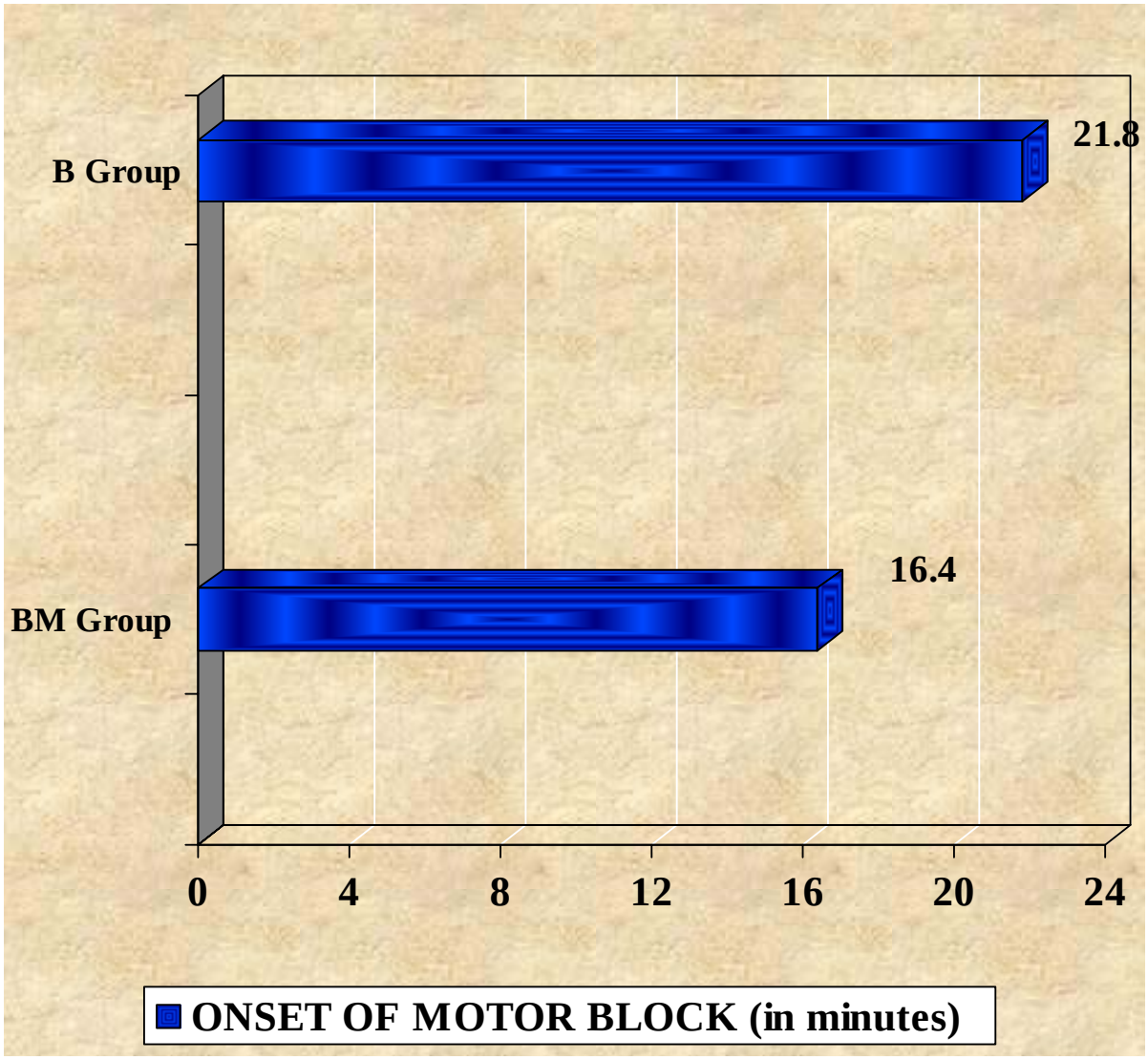
Table – 5

Onset of Motor Blockade

Onset of motor block	Group BM	Group B
Range	13-20 minutes	20-25 minutes
Mean	16.4 minutes	21.8 minutes
S.D	1.7 minutes	1.5 minutes
'p'	**0.0001**	

The onset of motor block was significantly faster in midazolam group ('p' value <0.0001 , <0.05).

Onset of Motor Blockade



Duration of Surgery:

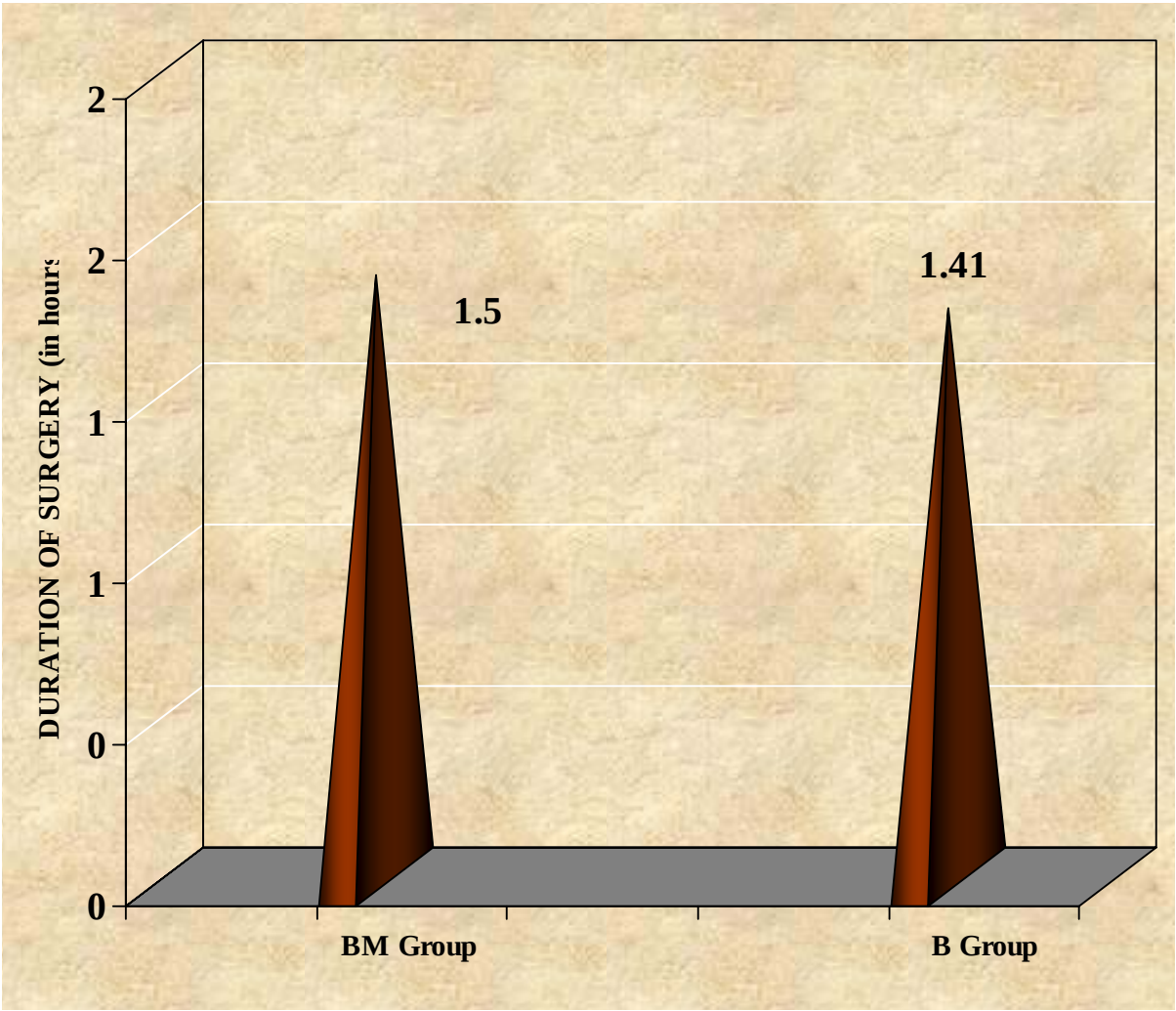
The duration of surgery in group BM varied from 0.5 to hours with mean of 1.49 hours standard deviation 0.64 hours. In group B, it varied from 0.5 to 3 hours with mean of 1.41 hours and standard deviation of 0.64 hours as shown in Table 6, figure 13.

Table – 6

Duration of Surgery

Duration of surgery (in hours)	Group BM	Group B
Range	0.5-3 hours	0.5-3 hours
Mean	1.49 hours	1.41 hours
S.D	0.64 hours	0.64 hours
'p'	**0.5511**	

Duration of surgery was comparable in both the groups ('p' value 0.5511>0.05)


DURATION OF SURGERY (in hours
2
2
1
1
0
0
1.5
1.41
BM Group
B Group

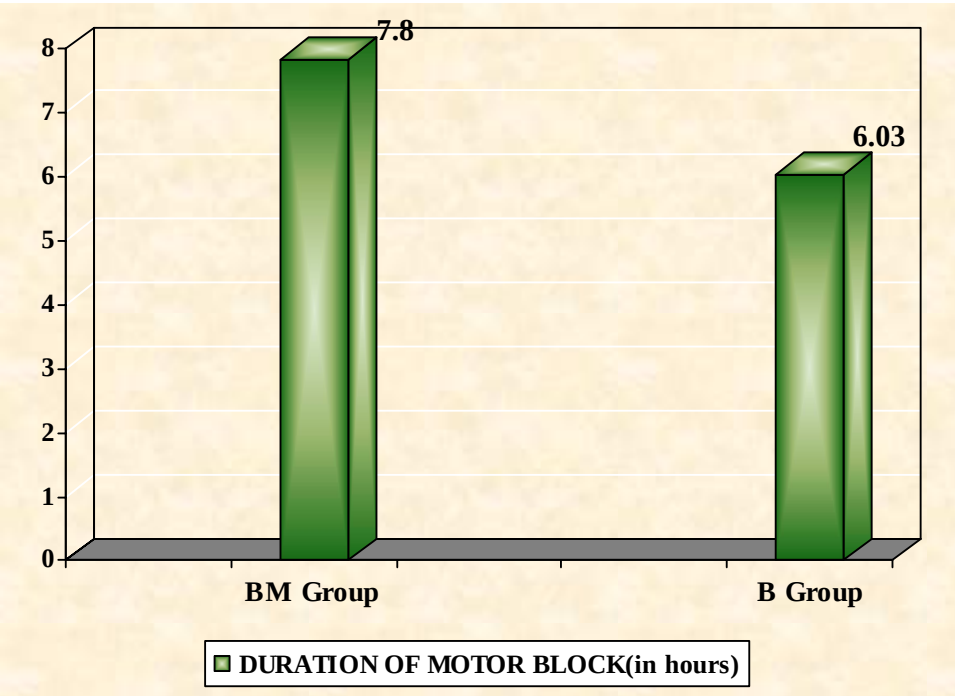
standard deviation of 0.32 hours as shown in Table 7, figure 14.

Table – 7

Duration of Motor Block

Duration of Motor Block	Group BM	Group B
Range	7-9 hours	5-7 hours
Mean	7.83 hours	6.03 hours
S.D	0.46 hours	0.32 hours
'p'	**0.0001**	

Duration of motor blockade was prolonged significantly in midazolam group ('p' value = 0.0001)



Duration of Sensory Block:

The duration of sensory block during in group BM varied from 11-15 hours with mean of 13.5 hours and a standard deviation 2.6 hours.

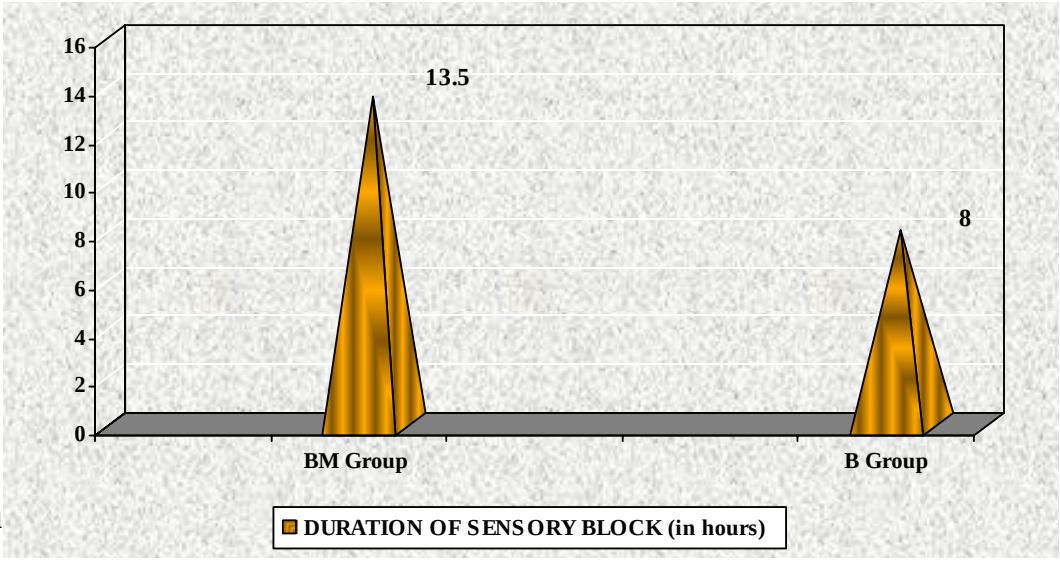
In group B it varied from 5-9 hours with mean of 8.0 hours and standard deviation 0.7 hours as shown as table 8 and figure 15.

Table -8

Duration of Sensory Block

Duration of Sensory Block	Group BM	Group B
Range	11-15 hours	5-9 hours
Mean	13.5 hours	8.0 hours
S.D	2.6 hours	0.7 hours
'p'	**0.0001**	

Duration of sensory blockade was prolonged significantly in midazolam group ('p' value 0.0001, <0.05)



Du

The duration of analgesia till the patient demands systemic analgesic (ie. VAS score > 5) varied from 15-18 hours in group BM with a mean of 16.8 hours and a standard deviation of (0.9 hrs).

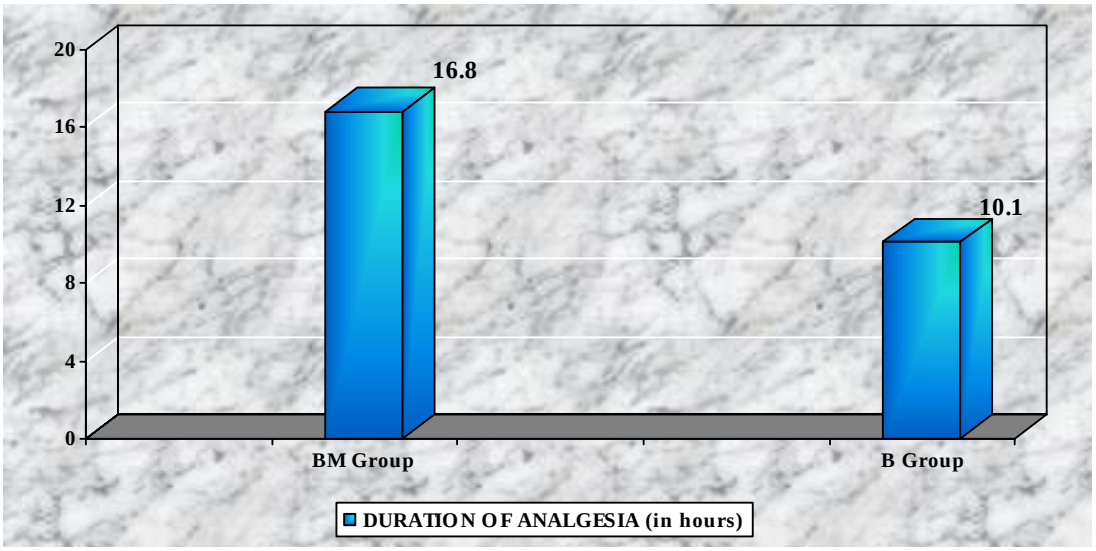
In the group B , it varied from 8-16 hours with mean of 10.1 hours and standard deviation of 1.8 hours as shown in Table 9, figure 16

Table – 9

Duration of Analgesia

Duration of Analgesia (in hours)	Group BM	Group B
Range	15-18 hours	8-16 hours
Mean	16.8 hours	10.1 hours
S.D	0.9 hours	1.8 hours
'p'	**0.0001**	

Duration of analgesia was significantly prolonged in midazolam group ('p' value 0.0001, <0.05)



Sedation Score :

In group BM, it was mean 1.6 $\pm$ 0.1, in group B it was mean 1.0 $\pm$ 0 as shown in the table 10 and figure 17a,17b.

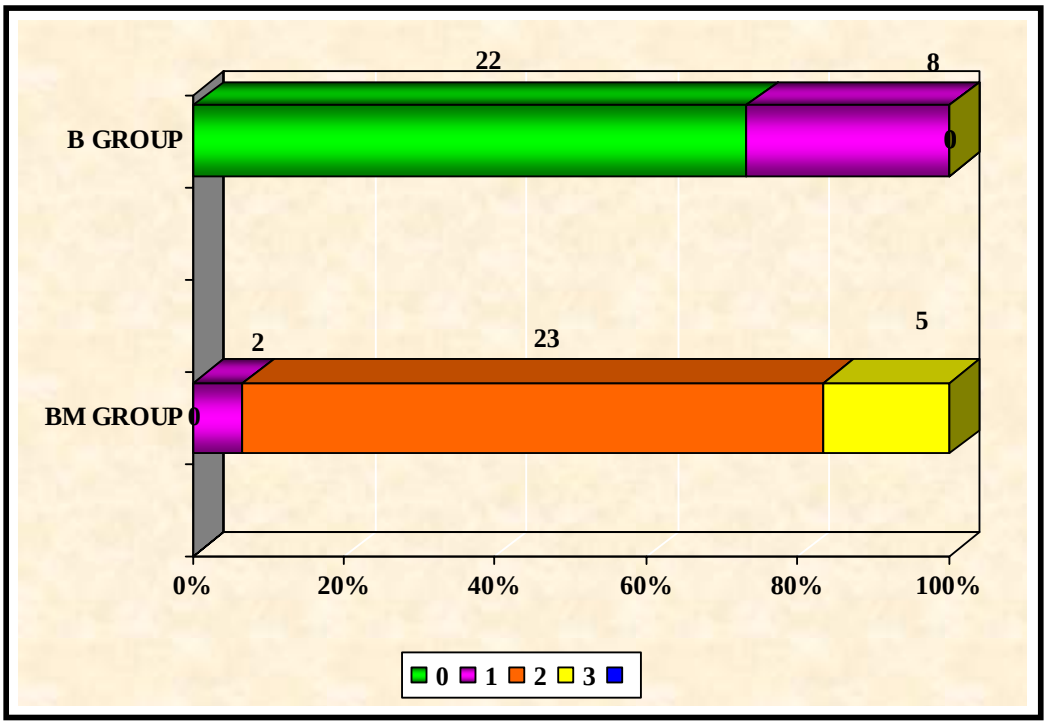
Table – 10Sedation Score

Sedation Score	Group BM	Group B
Range	1-3	0-1
Mean	2.1	0.33
S.D	0.48	0.28
'p'	**0.0001**	

The sedation score was varied from 1-3 BM group and 0-1 in B group. The mean value of sedation score was 2.1 in BM group with standard deviation of 0.48. the mean value of sedation score 0.33 in B group with standard deviation of 0.28.

Midazolam group has significant sedation score than bupivacaine group ('p' value 0.0001. < 0.05).

Sedation Score



Haemodynamic Monitors:

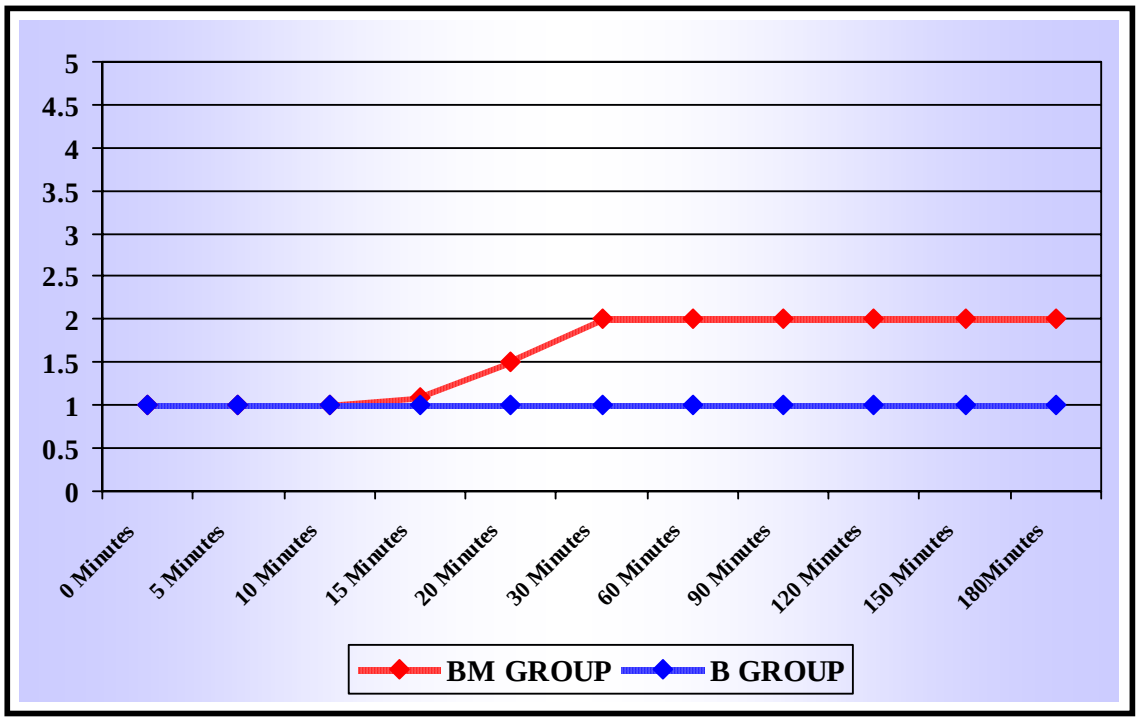
Table-11

Pulse Rate

Pulse Rate		
	BM group	B group
Mean	87.7	87.8
S.D	6.3	4.7
'p'	**0.8244**	

Pulse rate in both the group was comparable in various time interval. The statistical analysis showed 'p' value of 0.8244(>0.05)(Table 11,figure 18)

SEDATION SCORE AT VARIOUS TIME INTERVALS



PULSE RATE

AT VARIOUS TIME INTERVALS

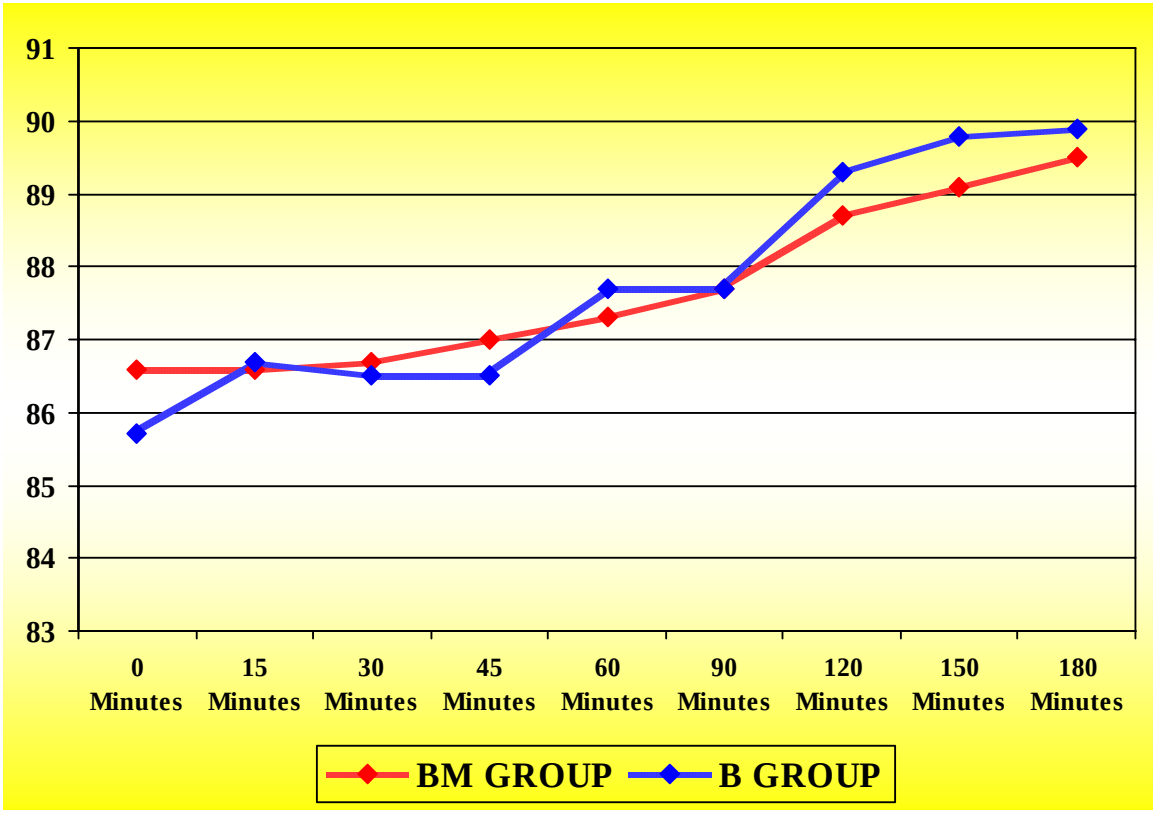


Table -12

Systolic Blood Pressure

Systolic B.P.		
	BM Group	**B Group**
Mean	121.0	122
S.D	5.9	4.1
'p'	**0.8591**	

Systolic BP in both the group was comparable in various time interval. The 'p' value 0.8591(>0.05) was not statistically significant.(Table 12,figure 19)

SYSTOLIC B.P.AT VARIOUS TIME INTERVALS

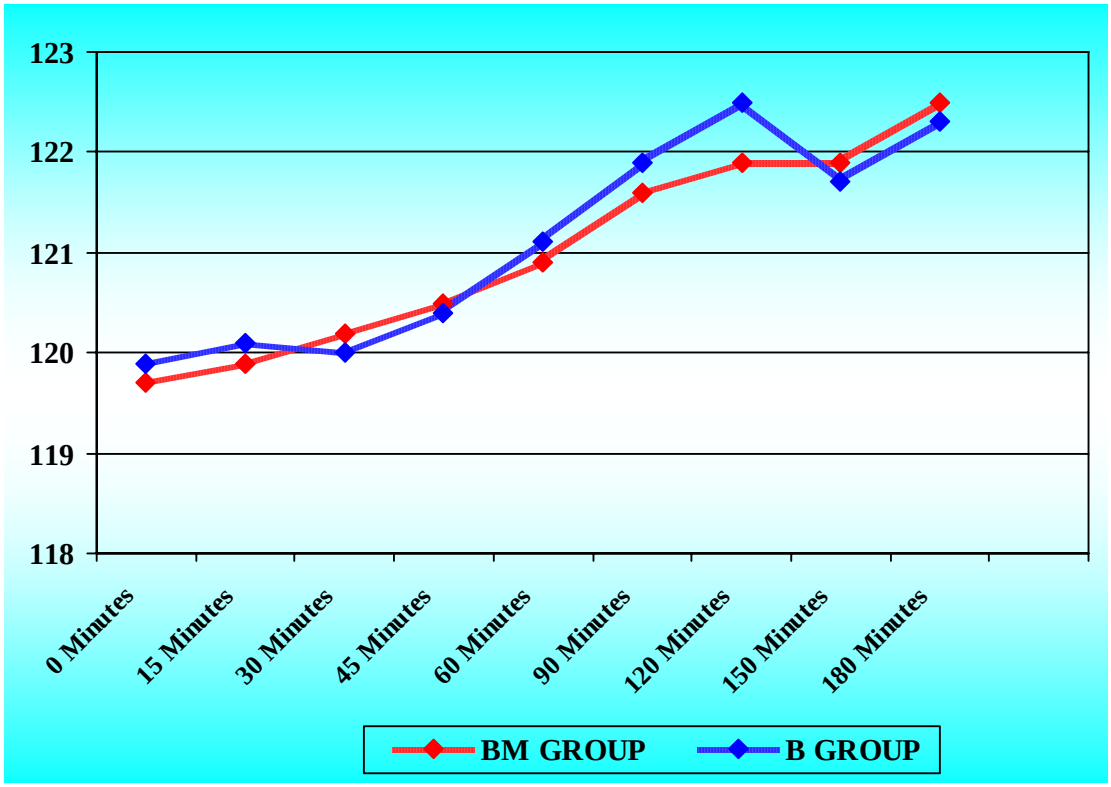


Table -13

Diastolic B.P.

Diastolic B.P.		
	BM	**B**
Mean	70.7	71.6
S.D	5.9	4.3
'p'	**0.684**	

Diastolic BP in both the group was comparable in various time interval. The 'p' value 0.684 (> 0.05)was not statistically significant.(Table 13,figurte 20)

DIASTOLIC B.P. AT VARIOUS TIME INTERVALS

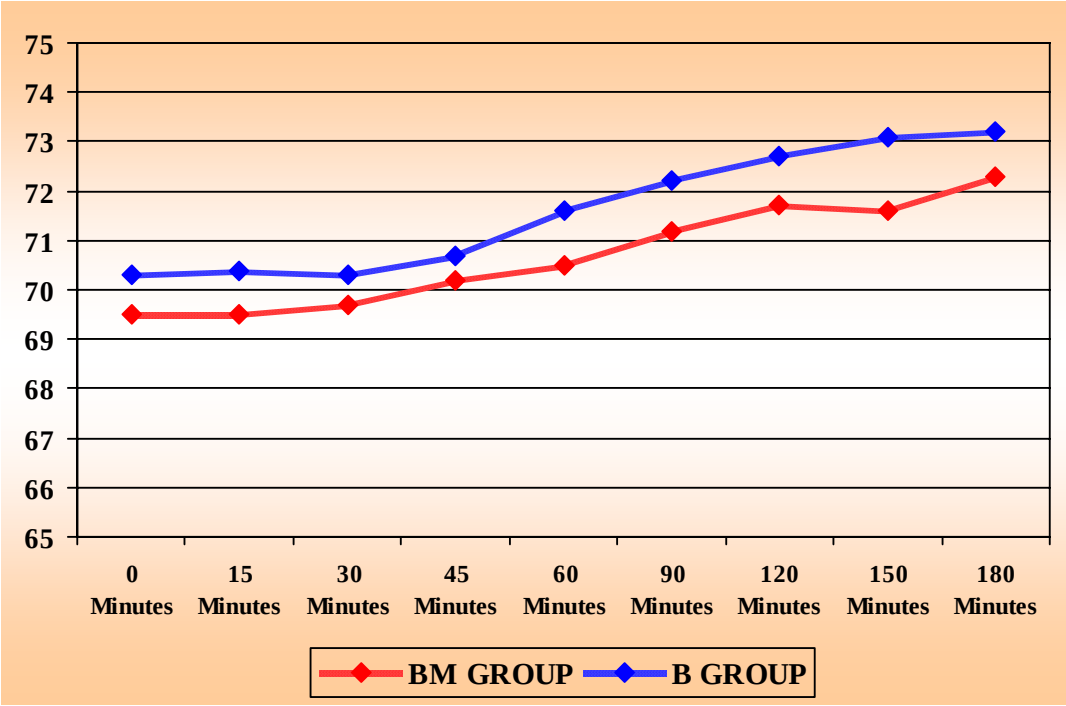


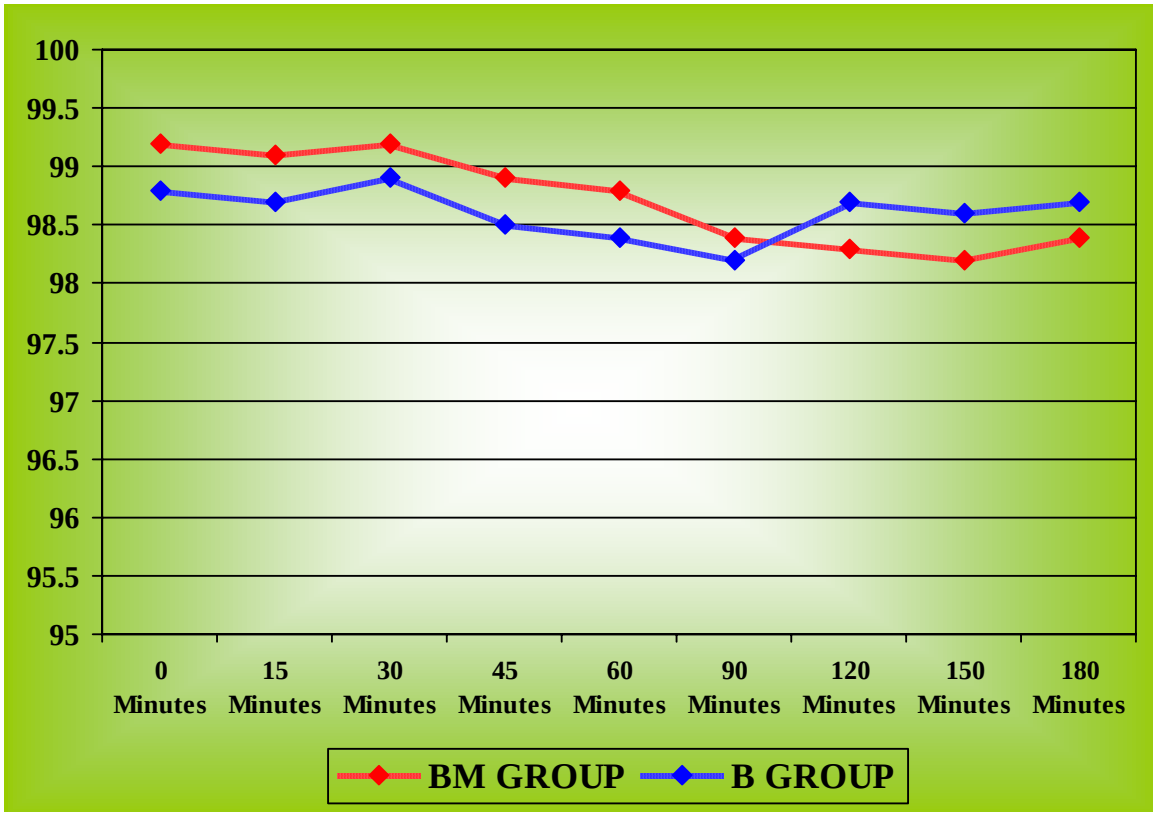
Table-14

SPO_2

SPO_2		
	BM group	**B group**
Mean	98.7	98.5
S.D	0.6	0.6
'p'	**0.1724**	

SPO2 at various time intervals was comparable in both groups. The 'p' value 0.1724 (> 0.05) was not statistically significant.(Table 14,figure 21)

SpO_2 - AT VARIOUS TIME INTERVALS



Statistical Tool:

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidermiological Information Package (EPI 2010).**

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant

DISCUSSION

GABA RECEPTOR:

The **$GABA_A$ receptor** (**$GABA_AR$**) is an ionotropic receptor and ligand-gated ion channel. Its endogenous ligand is γ-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the central nervous system. Upon activation, the $GABA_A$ receptor selectively conducts Cl^- through its pore, resulting in hyperpolarization of the neuron. This causes an inhibitory effect on neurotransmission by diminishing the chance of a successful action potential occurring ligands which contribute to receptor activation typically have anxiolytic, anticonvulsant, amnesic, sedative, hypnotic, euphoriant, and muscle relaxant properties. Some such as muscimol may also be hallucinogenic. Ligands which decrease receptor activation usually have opposite effects, including anxiogenesis and convulsion. Some of the subtype-selective negative allosteric modulators such as α5IA are being investigated for their ionotropic effects, as well as treatments for the unwanted side effects of other GABAergic drugs.

Midazolam was synthesized in 1976 by fryer and walser. First used in 1986, by Nilsson and colleagues. Generally midazolam act on GABA-A receptors and benzodiazepene receptors. Various studies showed that midazolam act on GABA-A receptors in the spinal cord. When it was used as a

adjuvant with local anaesthetics in intrathecal, epidural, caudal blocks, it prolonged the duration of analgesic effect of local aneasthetics.

Midazolam augmented both the duration of GABA-medicated synaptic current and the amplitude of GABA induced current by acting on the GABA-benzodiazepines receptors in substantia gelatinosa neurons; this would increase the inhibitory GABA nergic transmission. This may be possible mechanism for antinociception by midazolam.

By statistical analysis of two groups the age, sex weight and duration of surgery was comparable in both groups.

Onset of sensory blockade :

Mean onset of sensory block in group BM was 8.0 ± 0.8 minutes and in group B, it was 17.9 ± 0.8 minutes. The difference between the two groups was statistically insignificant with a p value of 0.0001 ($p<0.05$).

Onset of motor blockade :

Mean onset of motor blockade in group BM was 16.4 ± 1.7 minutes and in group B it was 21.8 ± 1.5 minutes. The difference between the two groups was statistically insignificant with a p value of 0.0001($p<0.05$).

On addition of midazolam to the local anaesthetic solution there is difference between the onset of sensory and motor blockade compared to the bupivacaine. Nasreen laiq et al study also reported similarly that the onset of

sensory blockade was 14±3.1minutes in midazolam + bupivacaine group compared to 18.5±3.5minutes in plain bupivacaine group. The onset of motor blockade was 10.5±2.4minutes in midazolam + bupivacaine group compared to 18.5±3.5minutes in plain bupivacaine group which is statistically significant.

Duration of Motor blockade:

Mean duration of motor block from score 3-3 in group BM (midazolam) was 7.83 ± 0.46 hours and in group B 6.03 ± 0.32hours. The difference between the two groups was statistically significant with a p value of 0.0001($p<0.05$).

Addition of midazolam to local anaesthetic solution have significant prolonged duration of motor blockade. This results correlates with studies conducted by Nasreen laiq et al in midazolam group it was 7.65±3.20 hours, compared to bupivacaine group it was 5.20±2.10hours.

Duration of Sensory block :

The mean duration of sensory block in Group BM was 13.5 $\pm$ 2.6 hours and in group B it was 8.0 $\pm$ 0.7hours.

The difference between the two groups was statistically significant with a p value of 0.0001 ($p<0.05$).

Addition of midazolam to local anaesthetic solution prolonged the sensory block significantly when compared to bupivacaine group. This is comparable with the study done by Koj jarbo, Yatindra kumar batra et al in which the duration of sensory blockade was 7±4.32 hours in Midazolam + bupivacaine group compared to 5.95±1.4 hours in plain Bupivacaine group.

Duration of analgesia :

The mean duration of analgesia is till the VAS score > 5 and in group BM(midazolam) it was 16.8 ± 0.9hours and in group B it was 10.1 +0.9hours.

The difference between the two groups was statistically significant with a p value of 0.0001 ($p<0.05$).

Addition of midazolam to local anaesthetic solution prolonged the post operative analgesia significantly when compared to adrenaline group. This results correlate favourably with studies conducted by Nasreen laiq et al .The mean duration of complete analgesia was significantly prolonged ($p=0.002$) in the bupivacaine - midazolam group.

Sedation score:

The sedation score in both groups is noted. The sedation score in group BM (midazolam) it was mean 2.1±0.48, in group B it was mean 0.33±0.28.In midazolam group since the sedation score was not more than 3, the respiratory function was not compromised. So intra operative sedation is well observed in midazolam group.

Haemodynamics:

In this study no significant difference was observed with respect to the pulse rate, systolic and diastolic blood pressure and SPO_2. This findings is consistent with the observation made by Nasreen laiq et al, who concluded that there was no significant haemodynamic changes after administration of midazolam and bupivacaine.

Side Effects :

Patients were observed for the side effects such as sedation, hypotension and bradycardia. In both groups there is no incidence of hypotension and bradycardia. No complication related to brachial plexus block were observed.

Gulec et al reported that bupivacaine– midazolam prolonged postoperative analgesia compared to bupivacaine – morphine combination when given caudally.

Tucker and associates reported the analgesic effects of intrathecal midazolam in combination with intrathecal fentanyl in labouring patients.

Goodchild and serrao the opioid antagonist naltrindole acts on ð-receptors and suppresses the antinociceptive effect of intrathecal midazolam, suggesting that intrathecal midazolam is responsible for the release of an endogenous opioid acting at spinal ð-receptors.

In this study, the addition of midazolam to the local anaesthetic solution produce difference between the onset of sensory and motor blockade when

compared to bupivacaine group. The duration of analgesia is significantly higher in midazolam group when compared to bupivacaine group. The duration of sensory blockade is also increased in midazolam group. These inferences provides midazolam produces a prolonged sensory and motor blockade.

SUMMARY

60 patients of ASA undergoing upper limb surgeries were randomly assigned into two groups, Group BM (midazolam) and Group B (Bupivacaine)

Surgery was done under supraclavicular approach of brachial plexus blockade.

The Patients in Group BM received 30ml of 0.375% bupivacaine 5μ/kg midazolam and . In group B received 30 ml of 0.375% of bupivacaine . Parameters observed were time of onset of sensory block and motor block, duration of motor blockade, duration of sensory block analgesic, duration of analgesia, sedation score, Haemodynamic and side effects.

Study shows that

On addition of midazolam to local anaesthetic solution there is difference between the onset of sensory block and onset of motor blockade to bupivacaine

1. On addition of midazolam to local anaesthetic solution significantly prolongs the duration of analgesia in 15-18 hours compared to bupivacaine group which is 8-16 hours

2. Addition of midazolam to local anaesthetic solution increases the duration of motor blockade by 7-9 hours compared to bupivacaine which is 5-7 hours.

3. On addition of midazolam to local anaesthetic solution increases the duration of sensory block 15 hours compared to bupivacaine which is 5-9 hours.

4. Haemodynamics did not differ between both group during surgery and post operating period.

5. In midazolam group intraoperative sedation is well observed without compromising respiratory function.

6. There are no side effects like hypotension and bradycardia in midazolam and bupivacaine group.

CONCLUSION

The addition of midazolam to local anaesthetic solution in supraclavicular approach to brachial plexus,

a) Produces significantly faster onset of sensory & motor blockade.
b) Prolongs the duration of sensory & motor blockade and duration of analgesia.

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PROFORMA

COMPARISON OF MIDAZOLAM AS ADJUVANTS TO BUPIVACAINE IN SUPRACLAVICULAR APPROACH OF BRACHIAL PLEXUS BLOCK

Name: Age: Sex: ASA Grade :

Address : Weight: IP No :

Diagnosis :

Surgery :

Investigations:

Hb% : Albumin Blood urea - mg%

Urine

Sugar - mg%

Sugar creatinine - mg%

ECG : X ray chest:

Group B : Supraclavicular block with Bupivacaine 0.375% 30 ml

Group BM : Supraclavicular block with bupivacaine 0.375% 30 ml

+ 50μ/kg preservative free midazolam.

OBSERVATION :

Onset of sensory blockade :

Onset of motor blockade :

Duration of surgery :

Duration of Motor blockade :

Duration of sensory block :

Duration of analgesia :

Sedation score :

Side Effects :

a. Hypotension : Yes / No

b. Bradycardiya : Yes / No

Motor Block :

0 - Normal power

1 - Paresis but able to move arm

2 - Not able to move arm but able to move fingers

3 - Complete motor blockade

Sedation score:

Brain & Ready score was employed

0- Fully awake

1- Drowsy

2- Drowsy but arousable on touch or call

3- Drowsy but arousable on deep stimuli

4- Somnolent

MASTER CHART BM GROUP

S.No.	GROUP	Name	Age	Sex	I.P. No.	Weight	ASA	Diagnosis	Surgery	Onset of sensory block mts	Onset of Motor blockl mts	Duration of Surgery Hrs/Mts	Duration of motor block (hrs)	Duration of Sensory block (hrs)	Duration of analgesia	S.S.Average	pluse mts	S.BP. mm.Hg.	D.BP.m.Hg	SPO2 %	Hypertension	Brady cardia
1	BM	Murugesan	35	M	44167	49	I	Rt. Supra condylar #	ORIF	7.0	15.0	2.5	8.0	14.0	16.0	1.5	90.2	114.2	63.8	99.3	Nil	Nil
2	BM	Mantharam	40	M	199783	52	I	Lt. MCP joint dislocation	ORIF ćfixation	7.0	18.0	2	8.0	15.0	17.0	1.6	95.3	126.0	74.2	98.2	Nil	Nil
3	BM	Sakthivelu	24	M	82292	48	I	Cut Injury Rt hand	Wound debriment K. wirefixation	8.0	20.0	3	7.0	14.0	18.0	1.5	89.6	116.4	72.7	97.7	Nil	Nil
4	BM	Samayam	24	M	42369	49	I	Crush Injury Rt hand	Amputation	8.0	20.0	1.5	8.0	13.3	18.0	1.5	86.9	128.2	78.2	98.7	Nil	Nil
5	BM	Sureshkumar	20	M	47455	50	I	Post electrical Burns RF_3 (Rt middle finger)	Contraction release	7.0	18.0	1.5	8.0	14.0	17.0	1.6	88.9	114.4	62.9	97.4	Nil	Nil
6	BM	Singaravelu	49	M	41427	48	I	Crush Injury Rt	Wound debridement sketetal stabistation	9.0	16.0	0.5	8.0	15.0	17.0	1.6	89.8	117.1	67.1	98.3	Nil	Nil
7	BM	Thangaiah	23	M	65501	50	I	Raw area Rt cubital fossa	SSG	10.0	16.0	1	8.0	13.0	16.0	1.6	78.2	125.6	74.9	98.4	Nil	Nil
8	BM	Mani	23	M	673441	52	I	Cut injury Lt little finger	Cleaning suturing	8.0	20.0	0.75	8.0	15.0	16.0	1.7	87.8	128.7	78.7	98.3	Nil	Nil
9	BM	Subulakshmi	39	F	27678	50	I	Raw area Rt FA	SSG	7.0	18.0	1.5	8.0	14.0	17.0	1.7	89.3	113.1	62.4	99.4	Nil	Nil
10	BM	Muthumari	30	F	71296	56	I	Olecranon # Rt	ORIF	8.0	16.0	0.75	8.0	13.0	15.0	1.5	87.3	118.0	67.3	99.4	Nil	Nil

11	BM	Samsutheen	50	M	6201	50	I	Median & ulnar Neve palsy	Repair	7.0	13.0	1.5	8.0	14.0	16.0	1.5	94.9	119.1	69.8	97.4	Nil	Nil
12	BM	Mariyam Beevi	60	F	7148	53	I	Lt Isolated # Radial & Proximal ⅓	ORIF plate ostosynthesis	8.0	15.0	1	8.0	13.0	16.0	1.6	95.6	124.2	72.2	98.4	Nil	Nil
13	BM	MuthuLakshmi	45	F	100046	49	I	# BB Forearm	ORIF Nailing Bone grafting	7.0	16.0	2	7.0	14.0	17.0	1.6	78.4	124.9	76.0	98.6	Nil	Nil
14	BM	Shanthi	15	F	99900	45	I	# Shaft of humerous	ORIF	7.0	17.0	1.5	8.0	14.0	18.0	1.6	88.7	128.0	78.9	99.3	Nil	Nil
15	BM	Velammal	60	F	90082	48	I	Raw area Rt hand	SSG	8.0	16.0	1	7.0	1.0	18.0	1.6	95.8	125.3	74.0	99.6	Nil	Nil
16	BM	Rajesh Kanna	22	M	104250	47	I	Rt Distal MP Joint of Index fingerinjury	ORIF plate-osteosynthesis	8.0	17.0	1	8.0	14.0	17.0	1.7	88.2	113.3	60.9	98.7	Nil	Nil
17	BM	Madhan	19	M	73456	48	I	Cut injury Rt wrist	Suturing k wire fixalion	8.0	17.0	1	8.0	14.0	16.0	1.5	92.7	127.6	76.4	98.6	Nil	Nil
18	BM	Sivakumar	38	m	96840	52	I	Crush injury , Brachial N injury	Brachial A Anastomosis	8.0	15.0	3	9.0	14.0	17.0	1.5	93.1	126.4	73.6	99.3	Nil	Nil
19	BM	Pandi	37	M	21666	50	I	Raw area Rt elbow	Flap cover	8.0	18.0	2	8.0	18.0	18.0	1.6	89.3	122.9	71.8	99.1	Nil	Nil
20	BM	Suriya	42	M	24248	58	I	Rt hand crush injury	Wound debridement	9.0	15.0	1.5	8.0	15.0	17.0	1.6	93.3	112.4	62.4	98.3	Nil	Nil
21	BM	Anitha	24	F	25363	56	I	Rt Hand post Infective sequelae	Contracture release flap cover	8.0	16.0	1.5	8.0	14.0	16.0	1.5	94.9	115.8	71.3	98.7	Nil	Nil
22	BM	Suresh Raja	42	M	28200	58	I	Lt thumb & contraction	Release & flap cover	9.0	17.0	2	7.0	13.0	17.0	1.5	77.8	114.0	62.4	97.6	Nil	Nil
23	BM	Sathiya	20	F	2311	55	I	Lt Little finger	Tendon repair	7.0	15.0	1.5	8.0	13.0	18.0	1.5	77.8	127.6	77.8	98.4	Nil	Nil

24	BM	Perumal	40	M	26762	58	I	Bone exposed Raw area PF3 &PIP foint	SSG	8.0	16.0	2	7.0	14.0	18.0	1.6	87.3	121.8	72.2	99.4	Nil	Nil
25	BM	Thangamathy	40	F	22450	57	I	Contracture of Rt Index finger	Release ć Flap cover	9.0	16.0	2	8.0	13.0	16.0	1.5	91.6	114.9	64.2	98.8	Nil	Nil
26	BM	Karuppasamy	24	M	29143	58	I	Rt radial Nervepalsy	Repair	8.0	15.0	1.5	8.0	14.0	17.0	1.6	92.2	122.7	72.2	98.1	Nil	Nil
27	BM	Velladurai	24	M	36363	57	I	Raw area Rt hand	SSG	9.0	16.0	0.75	7.0	13.0	16.0	1.6	77.8	111.3	61.8	98.9	Nil	Nil
28	BM	Pandi	28	M	59333	58	I	Implant Rt fore arm	Removal	8.0	15.0	0.75	8.0	14.0	18.0	1.5	75.3	120.0	68.9	99.0	Nil	Nil
29	BM	Sakthivelu	23	M	44805	59	I	Raw area Rt hand	SSG	8.0	16.0	0.75	8.0	13.0	16.0	1.5	78.7	128.4	78.9	99.2	Nil	Nil
30	BM	Buvaneshwari	48	M	65372	60	I	Raw area Rt hand	SSG	8.0	15.0	1.5	8.0	13.0	16.0	1.6	84.0	128.0	77.8	99.0	Nil	Nil

MASTER CHART B GROUP

S.No.	GROUP	Name	Age	Sex	I.P. No.	Weight	ASA	Diagnosis	Surgery	Onset of sensory block mts	Onset of Motor blockl mts	Duration of Surgery Hrs/Mts	Duration of motor block (hrs)	Duration of Sensory block (hrs)	Duration of analgesia	S.S.Average	pluse mts	S.BP. mm.Hg.	D.BP.m.Hg	SPO2 %	Hypertension	Brady cardia
31	B	Karthika Rani	16	F	16769	48	I	Mallet Finger	Correction	15.0	20.0	1.5	6.0	8.0	9.0	1.0	85.7	117.1	66.2	98.6	Nil	Nil
32	B	Alex	23	M	48449	49	I	Raw area Rt hand	SSG	17.0	22.0	1.5	6.0	8.0	10.0	1.0	83.7	113.6	62.9	98.7	Nil	Nil
33	B	Rajathi	48	F	10315	52	I	Lt soft tissue Injury Middle finger	SSG	18.0	20.0	0.75	6.0	8.0	11.0	1.0	88.6	122.2	74.2	98.6	Nil	Nil
34	B	Suresh Kumar	24	M	8806	48	I	Proximal Phalangeal Lt finger	ORIF	18.0	22.0	0.75	6.0	8.0	9.0	1.0	82.9	122.9	73.8	99.1	Nil	Nil
35	B	Chandra	50	F	4262	60	I	Lt carpal turnnel syndrome	Correction	17.0	22.0	1	6.0	8.0	10.0	1.0	79.1	117.3	69.1	98.4	Nil	Nil
36	B	Kalaiamml	50	F	70149	46	I	non union BB Rt fore arm	EOF	18.0	20.0	1.5	6.0	9.0	10.0	1.0	85.8	119.8	71.6	99.6	Nil	Nil
37	B	Karutha Kumar	45	M	71179	60	I	Dupuytren's contracture	Contracture release	19.0	20.0	0.75	6.0	5.0	9.0	1.0	88.0	123.3	74.4	98.6	Nil	Nil
38	B	Nagalakshmi	40	F	71759	59	I	Ulnar N. Palsy	Repair	19.0	23.0	1	6.0	8.0	10.0	1.0	92.2	118.7	70.7	99.0	Nil	Nil
39	B	Samiyappan	58	M	66012	60	I	Flexor contraction Rt hand	tendinolysis	18.0	24.0	1.5	5.0	7.0	9.0	1.0	94.0	120.7	70.7	98.6	Nil	Nil
40	B	Ramakrishan	35	M	74488	58	I	Distal Humerus Lt	ORIF Plate osteosynthesis	18.0	22.0	2	6.0	8.0	10.0	1.0	86.0	122.0	70.4	98.9	Nil	Nil
41	B	RadhaKumari	50	M	27363	57	I	compound supracondylar #	Wound debridement	18.0	24.0	1.5	6.0	8.0	10.0	1.0	90.4	118.4	68.2	97.4	Nil	Nil

42	B	MuthuKumar	32	M	33271	49	I	Raw area Rt hand dorsum	SSG	18.0	25.0	1.75	6.0	9.0	10.0	1.0	93.3	121.6	71.6	98.0	Nil	Nil
43	B	Arunachalam	40	M	42003	56	I	shaft of humerus Lt#	ORIF	19.0	25.0	2	6.0	8.0	9.0	1.0	93.6	121.8	69.8	98.3	Nil	Nil
44	B	Raju	65	M	48088	60	I	Galazi Dislocation Neck of humerus Lt#	ORIF plate osteosynthesis	18.0	20.0	2.5	6.0	8.0	11.0	1.0	84.9	121.8	72.2	98.3	Nil	Nil
45	B	SelvaKumar	32	M	55449	56	I	F2 –F3 cut Rt finger	Repair	19.0	23.0	0.5	6.0	9.0	10.0	1.0	87.8	128.2	77.1	98.4	Nil	Nil
46	B	Saravanan	32	M	54874	58	I	Supracondylar #	Repair	18.0	21.0	0.75	6.0	8.0	9.0	1.0	88.2	122.7	73.3	97.4	Nil	Nil
47	B	DhanaLakshmi	75	F	60658	54	I	Shaft of humerous Lt lower 1/3	ORIF	18.0	20.0	3	6.0	8.0	9.0	1.0	92.4	127.1	76.7	98.2	Nil	Nil
48	B	MuthuPillai	55	M	63893	42	I	Radial N palsy	ORIF plate osteosynthesis	18.0	23.0	1.5	6.0	8.0	10.0	1.0	87.3	121.8	74.0	99.1	Nil	Nil
49	B	Marimurthu	38	M	59183	60	I	Lunate dislocation lt	repair	17.0	23.0	2.5	7.0	8.0	10.0	1.0	78.2	120.9	74.2	97.7	Nil	Nil
50	B	Rani	35	F	63357	58	I	Rt bb forearm Implant in situ	Implant removal ORIF with P.O	18.0	20.0	2	6.0	9.0	10.0	1.0	90.2	118.4	68.7	99.1	Nil	Nil
51	B	Papathi	55	F	79314	49	I	Rt soft of humerus	ORIF Osteo synthesis	18.0	20.0	2	6.0	8.0	9.0	1.0	92.0	114.9	62.7	98.6	Nil	Nil
52	B	Karthikeyan	32	F	73004	50	I	BB forearm RF	K wire fixation	18.0	21.0	2	6.0	8.0	9.0	1.0	95.6	127.3	77.3	99.6	Nil	Nil
53	B	JeyaRaj	23	M	76606	52	I	Radial N palsy rt	Repair	18.0	22.0	1.5	7.0	7.0	8.0	1.0	90.4	125.8	76.2	98.4	Nil	Nil
54	B	KunthiDevi	52	F	76606	52	I	Flexor tendon dying	Repair	18.0	20.0	0.67	6.0	8.0	9.0	1.0	88.7	114.7	63.8	98.8	Nil	Nil
55	B	Shammugam	35	M	77835	60	I	FT4 contractions	Release	18.0	22.0	0.67	6.0	8.0	10.0	1.0	78.4	121.1	72.0	98.3	Nil	Nil
56	B	JeyaKumar	30	M	67747	59	I	Post Burns contracture Rt elbow	Release SSG	18.0	23.0	1.5	6.0	8.0	11.0	1.0	84.4	124.4	73.8	97.3	Nil	Nil

57	B	Pandi	37	M	276668	49	I	Rt Raw area elbow	flap cover SSG	17.0	22.0	1	6.0	8.0	16.0	1.0	80.0	118.7	72.4	98.1	Nil	Nil
58	B	Sathayamurthy	31	M	53236	60	I	RF_2 deformity	Correction K wire fixation	18.0	21.0	1.25	6.0	8.0	16.0	1.0	91.3	125.8	74.9	97.8	Nil	Nil
59	B	Chellapandi	25	M	39011	59	I	PP & MF # J RF	ORIF	17.0	22.0	0.75	6.0	8.0	9.0	1.0	90.4	127.1	76.9	98.7	Nil	Nil
60	B	Alagammal	21	F	5460	58	I	PBC raw area	SSG	18.0	21.0	0.67	6.0	8.0	10.0	1.0	89.6	112.7	63.3	98.2	Nil	Nil