COMPARATIVE STUDY OF THE EFFICACY OF POTASSIUM CHLORIDE AND SODIUM BICARBONATE AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK

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CHENNAI

TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation titled **"COMPARATIVE STUDY OF THE EFFICACY OF POTASSIUM CHLORIDE AND SODIUM BICARBONATE AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK"** is a bonafide record work done by **Dr.SOBHANA DEVI.P.**under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X - Anaesthesiology.

PROF. Dr.S.C.GANESH PRABU, M.D, D.A,
Director,
Institute Of Anaesthesiology,
Madurai Medical College
Madurai.

DECLARATION

I **Dr.SOBHANA DEVI.P** solemnly declare that this dissertation titled **"COMPARATIVE STUDY OF THE EFFICACY OF POTASSIUM CHLORIDE AND SODIUM BICARBONATE AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK"** has been done by me. I also declarethat this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Doctor of Medicine degree Branch –X (Anaesthesiology) to be held in April 2013.

Place: Madurai Dr.SOBHANA DEVI.P

Date:

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CONTENTS

ANNEXURES

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

ANTI- PLAGIARISM CERTIFICATE

DIGITAL RECEIPT

INTRODUCTION

Pain is an unpleasant sensory and emotional experience that occurs in response to tissue damage.Every patient has the right to get rid of pain and it is the duty of every anaesthetist to provide adequate pain relief.

Pain relief is provided by various methods like oral drugs , nerve blocks but adequate pain relief is provided by interrupting the transmission of pain. Peripheral nerve blocks provide longer and more localized pain relief than systemic opioids and non-steroidal anti-inflammatory drugs.

In 1884,Koller demonstrated cocaine in ocular surface anaesthesia which was a new era in regional anaesthesia for the prevention of pain. And later Brachial plexus block has evolved in procedures for upper limb surgeries when William Halstead performed it for the first time in 1884.

Brachial plexus block is performed as a supplementation to general anaesthesia to reduce the analgesic requirements, better tourniquet tolerance as well as to provide post operative analgesia.Brachial plexus is used as a sole anaesthetic technique in situation where general anaesthesia is relatively contraindicated (eg) emergency situation where the starvation time is inadequate, and if proper precautions are not taken, may lead to aspiration of stomach contents.

Successful brachial plexus block for upper limb surgeries requires wide knowledge about the anatomy of its origin and its branches, complications that would occur in various approaches as well as the clinical application of local anaesthetics and various adjuvants added to it inorder to provide better quality of blockade as well as prolong the duration of post operative analgesia. At the same time the motor blockade should not be prolonged for a long time that prolongs the hospital stay.

Brachial plexus can be easily blocked because they lie in a sheath and by eliciting paraesthesia of one of the roots/trunk/cords can give a success rate by injecting large volume of local anaesthetic solution.

Most of the local anaesthetics developed between 1900-1940 were amino ester compounds like cocaine, procaine, chloroprocaine but they were associated with allergic reactions and short duration of action.Lofgren and associates synthesized lignocaine and Ekenstam synthesized Bupivacaine which improved the quality of Regional anaesthesia. Though lignocaine is short acting it is widely used till date. Bupivacaine, an amide local anaesthetic witha longer duration of action is also used in nerve blocks.

In most of the peripheral nerve blocks lignocaine and bupivacaine are mixed together and used. Lignocaine provides early onset of blockade whereas bupivacaine prolongs the duration of blockade. Ifan inadvertant

accidental injection of these two drugs occurs they produce serious complications involving cardiovascular and central nervous system. Hence in this study only bupivacaine is used so that side effect of atleast one drug is avoided if a situation arises to face the detrimental effects of local anaesthetic toxicity if accidentally injected intravascularly.

In order to provide better anaesthesia in the intraoperative period as well as to provide better analgesia in the post operative period,various adjuvants are added to local anaesthetic solution like Sodiumbicarbonate, Potassiumchloride, Adrenaline,Dexmedetomidine,Clonidine, Midazolam, Fentanyl,Tramadol, Dexamethasone, etc.

Adjuvants are added to improve the quality of anaesthesia and also to improve the duration of post operative analgesia. Classically Adrenaline is added which prevents the systemic absorption of the local anaesthetics and thus prolongs the duration of analgesia. But it has got detrimental effect of increasing the heart rate, blood pressure, and thereby it can cause coronary vasoconstriction. The other adjuvants can cause side effects like vomiting, pruritis, altered hemodynamic stability, sedation etc.

In this study 30ml of 0.375% of Bupivacaine with 0.2mmol(0.1ml) of potassium chloride of 15% solution which contains 20mmol/10ml is compared with 30ml of 0.375% of Bupivacaine with 0.179 mmol(0.2ml) of 7.5% of sodiumbicarbonate which contains 0.893mmol/litre regarding the

quality and quantity of blockade in brachial plexus block for upperlimb surgeries.

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AIM OF THE STUDY

TO COMPARE THE EFFICACY OF POTASSIUM CHLORIDE AND SODIUM BICARBONATE AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK.THE FOLLOWING PARAMETERS WERE COMPARED BETWEEN THE TWO GROUPS.

1.Onset of sensory block

2.Onset of motor block

3.Tourniquet tolerance

4.Duration of sensory block

5.Duration of motor block

6.Quality of block

7. Hemodynamic stability

8.Complications.

HISTORY

During sixteenth and seventeenth century a French surgeon Ambrose produced local anaesthesia by mechanical compression of nerve trunks which was later followed by many European surgeons. In 1860 NIEMANN extracted cocaine from coca leaves.

Carl Koller,an young Ophthalmologist from Vietnam performed experiments on himself and proved that cocaine had local anaesthetic effects. He used cocaine as topical anaesthetic and applied it on his own cornea .

In 1884, WILLIAM HALSTEAD(1852-1922)(1) first performed Brachial plexus at the level of their roots with cocaine and thereby he “freed the cords and nerves of the brachial plexus”.

WILLIAM HALSTED (1852-1922)(1) injected cocaine in to the nerve trunks and later his technique was followed by ALFRED HALL. Halsted and Hall worked together and they reported their success which was published on December 6,1884 in NewYork medical journal.

In 1887,GEORGE CRILE(1) introduced the technique of infiltrating the nerves under general anaesthesia to reduce stress and shock of surgery by using 0.5% cocaine.He first blocked the Brachial plexus and disarticulated the shoulder joint.

ALFRED HALL(1) reported that injecting 4%Cocaine (15mg) in to the forearm blocked the cutaneous nerve transmission and provided analgesia below the point of injection.Then 2ml(80mg) was injected in to the ulnar nerve at the level of elbow which produced blockade of the ulnar nerve distribution.

In 1902, HARVEY CUSHING(1) coined the term "Regional Anaesthesia". Harvey Cushing blocked the brachial plexus and sciatic plexus by direct vision under general anaesthesia inorder to reduce the intra operative anaesthetic requirements as well as to provide post operative analgesia.

In 1908, AUGUST BIER(1) (1861-1949) described Intravenous Regional anaesthesia. He injected procaine in to the vein of upperlimb by applying two tourniquets.

In 1911,G.HIRSCHEL(1) described the axillary block by blind injection technique. D.KULENKAMPFF(1) described the SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK by elicitngParaesthesia technique . In his study Kulenkampff placed the needle superficial to the first rib and the pleura. He injected the local anaesthetic solution in to his own plexus with 10ml of procaine at the mid-clavicular position lateral to subclavian artery. Because of the risk ofcomplications like pneumothorax and mediastinal emphysema, MULLEY (1)modified Kulenkampff's technique and described the lateral paravertebral approach which is now known as the 'Winnie block'.

In CARL SCHLEICH (1859-1922)(2) introduced the technique of infiltrating local anaesthetic solution in a diluted form (0.01%-0.2%) as an alternative to direct injection of nerve trunks.

LEONARD CORNING (1862-1934)(2) placed tourniquet that prevented blood loss. HENRICH F.BRAUN (1862-1934) (2) prolonged the effect of cocaine by adding Epinephrine which was described as 'CHEMICAL TOURNIQUET'. In 1905, Braun introduced Procaine which is less toxic than cocaine.Heinrich Braun wrote a book on local anaesthesia which was first translated in English and published in the year 1914.

In 1911,G.HIRSCHEL(2) was the first person to introduce the technique of percutaneous brachial plexus block through axillary approach. Later it was modified by GEORGE PITKIN(2) and R.H.DE JONG(2) which remains popular even today.

In 1917,BAZY(2) and V.PAUCHET(2) described infraclavicular approach of brachial plexus block which was later popularized by P.RAJ(2) in the year 1973. In 1912,M.KAPPIS (2)described the posterior paravertebral approach but there were high incidence of failure rate hence J.ETIENNE, V.PAUCHET and G.PITKIN(2) described anterior approach to the brachial plexus block.

In 1970,ALON P.WINNIE(2) introduced the technique of interscalene approach of Brachial plexus block. Winnie emphasized that scalene muscles

are more accurate landmark to locate the brachial plexus block than mid-clavicular position and subclavian artery.

In 1905, ALFRED EINHORN(2) synthesized procaine and used it till 1932. Later in 1930's long acting drug the Tetracaine and Dibucaine were synthesized. In 1943, LOFGREN(2) and LUNDQUVISIT (2)synthesized Lignocaine and it was introduced by TORSTEN GORDH(2) in 1948. In 1952 chloroprocaine was synthesized. In 1957 mepivacaine was synthesized. In 1963, EKENSTAM (2)synthesized Bupivacaine and TELIVUO(2) introduced bupivacaine in to clinical practice. In 1996 Ropivacaine was synthesized.

ANATOMICAL CONSIDERATIONS

The Brachial Plexus is situated superficially and can be easily blocked for upper limb surgeries as well as for post operative analgesia.

FORMATION OF BRACHIAL PLEXUS:(3)(2)(1)(4)(5)

The brachial plexus is formed by the union of anterior primary rami of fifth,sixth,seventh,eighth cervical vertebrae and first thoracic vertebrae. Sometimes the fourth cervical vertebra contributes to the plexus which is referred as the pre-fixed type and a contribution from second thoracic vertebra to the plexus is referred as the post-fixed type.

The roots emerges from the intervertebral foramina and passes behind the Foramen Transversarium and lies between the anterior and posterior tubercles of the transverse process. The roots lie inthe fibro-fatty space between the two sheaths in the groove formed between the scalenus anterior and scalenus medius. The anterior part of the sheath arises from the anterior tubercles and covers the posterior aspect of scalenus anterior whereas the posterior part of the sheath arises from the posterior tubercles and covers the anterior aspect of scalenus medius.

The sheath extends laterally along with the brachial plexus and enters the axilla.As they pass between the scalenus anterior and scalenus medius they are blocked by local anaesthetics .This technique is called the Interscalenae approach of brachial plexus block.

These nerve roots emergesfrom the intervertebral foramina and converges to form trunks. These trunks are situated vertically but not always and are named accordingly as superior,middle and inferior trunk.The superior trunk is formed from the contributions of fifth and sixth cervical nerve roots, the middle trunk is contributed from the seventh cervical nerve root and the inferior trunk is formed from the contribution of eigth cervical and first thoracic nerve roots.

The roots gives off few branches as well as receives few branches from the cervical sympathetic ganglion. They are,the roots of fifth cervical (C5) and sixth cervical(C6) , each receives a grey ramus communicans from the middle cervical sympathetic ganglion. C7 and C8 each receives a gray ramus communicans from the inferior cervical sympathetic ganglion. The first thoracic root gives a white ramus to; and receives a grey ramus from the first thoracic sympathetic ganglion. From the roots of fifth, sixth, seventh, eigth cervical roots the nerves to longus colli and Scaleni is given off. The fifth cervical root gives nerve to rhomboids(dorsal scapular nerve). The fifth cervical roots gives a branch to phrenic nerve;which also receives contributions from third and fourth cervical roots. The fifth, sixth and seventh cervical root gives a branch to

serratus anterior (long thoracic nerve of Bell).

The three trunks runs downwards and lateral to the lateral border of first rib just below the clavicle and divides in to anterior and posterior divisions. Once they divide they emerge below the clavicle and form thecords.The trunks gives off branches before they divide. They are, the nerve to subclavius from the front of the upper trunk (C5,C6); the supraclavicular nerve from the outer part of the upper trunk (C5,C6). The point of origin of the branches from the upper trunk is called Erb's point.

The three trunks as they run downwards and laterally across the posterior tuberclesof the vertebra and then across the first rib, they are blocked by supraclavicular ,subclavian perivascular approach and intersternocleidomastoid approach. The trunk gives off two branches before giving divisions.They are the suprascapular nerve C5,C6 and Nerve to subclavius C5,C6

The six divisions enters in to axilla and join as lateral,medial and posterior cord according to their relation with the axillary artery.Between the first rib and axilla the Brachial plexus are blocked by Infraclavicularapproach.

The lateral cord is formed by the union of anterior division of superior trunk and middle trunk. The medial cord is formed by the contribution of anterior division of inferior trunk. The posterior trunk is formed from the posterior divisionof all the three trunks. The cord once formed runs lateral to the lateral border of pectoralis minor muscle and gives off major terminal

branches.

The lateral cord before continuing as musculocutaneous nerve C5,C6,C7, it gives off the following two branches. They are lateral division of median nerve C6,C7 and the lateral pectoral nerve C5,C6,C7.

The medial cord before continuing as ulnar nerve it gives off the following four branches.They are medial division of median nerve C8,T1, medial cutaneous nerve of arm C8,T1,medial cutaneous nerve of forearm C8,T1,medial pectoral nerve C8,T1.

The posterior cord before terminates in to two major nerves the radial nerve C5,C6,C7,C8,T1 and axillary nerve C5 ,C6 it gives off the following three branches. They are the upper scapular nerve C5,C6,the nerve to lattismus dorsi C6,C7,C8, and the lower subscapular nerve C5,C6.

Relationship of Brachial plexus :

The roots lie between scalenus anterior and scalenus medius and lie above the second part of subclavian artery.The trunks are formed in the floor of the posterior triangle and are superficially placed.They are crossed by external jugular vein,trasverse cervical artery,inferior belly of omohyoid and supraclavicular nerve.

The upper and middle trunks lie above the subclavian artery and the lower trunk lies behind the subclavian artery. The trunks bifurcates in to divisions at the lateral border of first rib behind the clavicle.

The cords are formed at the apex of axilla and around the axillary artery.Initially the medial cord lies behind the axillary artery, the posterior and lateral cord lies lateral to the axillary artery.But later behind the pectoralis minor the cords lie according to their names as medial,lateral and posterior to axillary artery.

SIGNIFICANCE OF FIRST RIB:

The first rib lies in the horizontal plane slightly inclined downwards and forwards. It has head which articulates with the body of first thoracic vertebra. It has an upper surface and two transverse grooves. The subclavian vein lies in the anterior groove. The subclavian artery and lower trunk of brachial plexus lies in the posterior groove. Between the two grooves is the scalene tubercles where the scalenus medius is inserted. The outer border gives origin to serratus anterior. Importance of this first rib is it is kept as a guide to locate the plexus without puncturing the pleura.

SUBCLAVAIAN ARTERY:

It extends from its origin to the outer border of the first rib. The right subclavian artery is a branch from innominate artery which in turn is a branch from the arch of aorta and the left subclavian artery is a branch from the arch of aorta. The brachial plexus lies parallel and lateral to the third part of the subclavian artery. The third part of subclavian artery runs laterally and continues as the axillary artery. The terminal portion of the subclavian artery

lies behind the clavicle at its mid-point. Here the inferior trunk of brachial plexus is situated posterior to the third part of subclavian artery. The upper trunk is superior to the subclavian artery and the middle trunk is lateral to the subclavian artery.

SUBCLAVIAN VEIN:

The subclavain vein is protected by the clavicle and it is very unlikely to be punctured. The anterior scalene muscle separates thesubclavian vein from the brachial plexus**.**

TECHNIQUES OF BRACHIAL PLEXUS:(3)

1. Interscalene approach

i.Anterior approach:

a.Winnie

b.Meier

c.Modified lateral approach of Borgeat

ii.Posterior approach:

a.Kappis/pippa

b.Boezaart

2.Supraclavicular approach

a. Classic supraclavicular approach of Kulenkampff

b. Subclavian perivascular approach of Winnie and Collins.

c.Modified lateral paravascular approach of Moorthy

d.Plumb – bob technique

3.Infraclavicular approach :

a.Raj approach

b.Coracoid approach

c.Vertical infraclavicular approach

4.Axillary approach :

i.Transarterial injection

ii.Paraesthesia Technique

a.single injection technique

b.Multiple injection technique

1.**INTERSCALENE BRACHIAL PLEXUS BLOCK:**

Anterior approach(Winnie in 1970) : (2)(4)

The roots of brachial plexusemerges out from the intervertebral foramina and lies between the anterior and middle scalene muscles.They lie superficial and posterior to the second and third part of the subsclavian atery. The dome of pleura lies anterolateral to the inferior trunk**.**

Clinical Applications:

Interscalene approach is suitable for surgeries involving the shoulder. The upper trunk and middle trunk is blocked.

The Patient is explained about the procedure and made to lie insupine position with the neck extended and turned to the contralateral side and the arm held in any position.The interscalenae groove is palpated behind the sternocleidomastoid muscleby asking the patient to lift thehead slightly.The needle tip is inserted at the transverse process of sixth cervical vertebra which corresponds to the cricoid cartilage where the external jugular vein often crosses the sternocleidomastoid muscle.The 22 gauge, needle is inserted perpendicular to the skin and directed in amedial,dorsal and caudad at a 45º angulation.A click can be felt as the needle pierces the pre-vertebral fascia. The plexus is reached at a depth of 0.75cm-1.5cm. At a depth of 2cm the transverse process may be encountered, in such a situation the needle is walked across this structure to locate the plexus.Once Paraesthesia is elicited in the upper arm or shoulder then the local anaesthetic solution of around 40ml is injected after confirming negative aspiration. The inferior trunk is often spared which is overcome to some extent by applying digital pressure superior to the injection site and downward massage along with 45 º head up position.

Complications:

i. Phrenic nerve palsy(100% incidence) results in unilateral diaphragmatic paresis and reduces pulmonary function by 25%.

ii.injection in to vertebral artery results in convulsions.

iii. injection in to Epidural space

iv. injection in to Subarachnoid space

v.Recurrent laryngealnerve palsy results in hoarseness of voice

vi.sympathetic chain involvement results in Horner's syndrome

v.Pnemothorax is rare

vi.Nerve damage or Peripheral neuritis.

Drawback:

The inferior trunk is not blocked hence not suitable for forearm and hand surgeries.

MEIER APPROACH:

It is the modification of winnie approach in which the needle is directed at the posterior border of sternocleidomastoid cranially 2-3cm at the level of superior thyroid cartilage**.**

Modified lateral approach of Borgeat:

The needle is directed along the interscalenae space at the level of cricoid cartilage towards the posterior part of the upper and the middle trunk.

Interscalene(cervical approach):

The posterior approach was described by Kappis in 1912 and later by Pippa. In this technique the head is maximally flexed and the spinous process of C6 and C7 is palpated. Draw a line 3cm laterally between the spinous processes of C6 and C7.Then insert the needle perpendicular to the skin and at 5-6cm

distance the transverse process of C7 vertebra comes in contact with the needle.Then redirect the needle cranially where thebrachial plexus is situated and once contractions of the shoulder or abduction of arm occurs then the local anaesthetic is injected.

Drawback:

The lower trunk is spared so the intercostobrachial nerve has to be blocked in the medial aspect of arm for tourniquet tolerance.

Boezaart approach:

The needle is inserted at 45º angulation in a V shaped groove between the anterolateral border of trapezius muscle and the posteromedial border of levator scapulae and 30º caudad towards the sternal notch.

Side-Effects:

i.Phrenic nerve palsy 85%-100%

ii.Horner's syndrome 75%

iii.Recurrent laryngeal nerve palsy 20%

iv.Vessel puncture

v.Pneumothorax(rare)

Contraindications:

Contralateral phrenic nerve palsy or recurrent laryngeal nerve palsy

1. SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

Clinical Applications:

Indicated for surgeries on the elbow, forearm and hand.The distal trunk and the proximal division is blocked.

A.**Classic Supraclavicular Block:**

Position: Supine with neck extended and turned to contralateral side .

At the mid clavicular line at 1cm posterior to the clavicle and lateral to thesubclavian artery the needle is directed posteriorly caudally and medially. If the first rib is encountered the needle is systematically walked anteriorly and posterioly to locate the brachial plexus.

Complications:

Pneumothorax 0.5%-6%

Phrenic nerve palsy 40% to 60%

Horner's syndrome

Peripheral neuropathy

Cervical sympathetic nerve blockade.

B) Subclavian Perivascular Technique of Winnie and Collins:

This is the technique followed in this study.

Clinical Applications:

The Brachial plexus are arranged in a compact space as they cross the first rib. The blockade of three trunks are blocked reliably.

Position:

The patient lies in aSupineposture with neck extended and turned to the contralateral side.The arm to be anaesthetized is held in adducted position. The posterior border of the sternocleidomastoid muscle is palpated easily when the patient raises the head slightly. The palpated fingers can then roll over the belly of the anteriorscalene muscle in to the interscalene groove,where a mark is made 1.5 to 2.0cm posterior to the midpoint of the clavicle. Palpation of the subclavian artery at this site confirms the landmark. The 22 gauge needle is inserted posterior,medial, caudad to the subclavian artery; by eliciting paraesthesia at the elbow 30ml of 0.375% of Inj. Bupivacaine with0.2mmols(0.1ml) of Inj.potassium chlorideis injected in Group BK or 30ml of 0.375% of Inj.Bupivacaine with0.17mmol(0.2ml) of sodium bicarbonate is injected in Group BS.

Complications:

Pneumothorax 0.5% sometimes manifests after 12-24 hours

Horner's syndrome

Phrenic nerve palsy

Hemothorax

Hematoma formation

Drawback:

The area supplied by Ulnarnerve is spared.

C) Modified lateral paravascular approach of Moorthy:

The needle is directed laterally ,posteriorly and caudally along the course of the subclavian artery towards the axilla medial and inferior to the subclavian artery.

D.Plumb Bob Technique:

The plumb bob technique involves insertion of the needle at the point where the lateral border of the sternocleidomastoid muscle inserts in to the clavicle. After raising the skin wheal a 22 gaugeneedle is inserted which mimicks a plumb bob suspended over the needle entry site. Once paraesthesia is elicited 40ml of local anaesthetic solution is injected.

3.INFRACLAVICULAR BRACHIAL PLEXUS BLOCK:

i. Raj approach

ii.Coracoid approach

iii.Vertical infraclavicular approach

Raj Technique:

The needle is inserted 2.5cm below the midpoint of the clavicle. The axillary artery is aimed at the'' brachial line'' in the axilla. The brachial line runs from the transverse process of sixth cervical vertebra to the axillary artery in the axilla.

Drawback:

Technical difficulty

Lower success rate

Coracoid approach:

The needle is inserted 2cm medial and 2cm caudal to the coracoid process,Posterior and perpendicular to the skin and at about 4cm distance the cords of brachial plexus is located.

Vertical infraclavicular approach:

Between the ventral apophysis of acromion and the jugular notch the needle is directed lateral to axillary artery and axillaryvein at 4cm depth and thebrachial plexus is blocked.

Advantages:

Simple to perform

High success rate

Tourniquet toleranceis good

Patient position is comfortable

Catheter insertion is simple

Complications:

Pneumothorax

Hemothorax

Chylothorax (left side is more common)

4.AXILLARY BLOCK:

i.Transarterial injection

ii.Paraesthesia Technique

a.single injection technique

b.Multiple injection technique

Axillary block was first described by **Halstead** in 1884.

Position:

Supine with the arm in abduction of 90º.Palpate for the axillary artery pulsationat the mid- axillary fossaand go transarterially and inject the local anaesthetic. Also single needle or multiple needles are used. High success rate with multiple needle technique.

Advantages:

No possibility of pneumothorax

No Stellate ganglion block

No Recurrent laryngeal nerve palsy

No Phrenic nerve palsy

Complications:

Intra arterial injection

Post operative neuropathy

Hematoma

Infection

PHYSIOLOGICAL CONSIDERATION:(2)(4)(1)

INTRODUCTION:

Most local anaesthetics block voltage-gated sodium channels from inside the cell. Not all the nerve fibres are equally sensitive to local anaesthetics .The sensitivity is determined by the degree of myelination,axonal diameter,and various other anatomic or physiologic factors. Local anaesthetics produce transient sensory, motor and autonomic loss.

CLASSIFICATION OF PERIPHERAL NERVES ACCORDING TO ANATOMY,PHYSIOLOGY, AND FUNCTION:

The A fiber has four subtypes; they are alpha, beta, gamma and delta. The A alpha fibres are heavilymyelinated . The diameter of the nerve fibre is 6-22µm. They conduct at a velocity of 30-120msec, and carry the motor sensation. These fibres are sensitive to local anaesthetics.

The A beta fibre are heavily myelinated. The diameter of the nerve fibre is 6-22µm and conducts at a velocity of 30-120msecs. They carry tactile and proprioception. They are susceptible to local anaesthetics.

The A gamma fibre are myelinated . The diameter is 3-6µm and conducts at a velocity of 15-35 msecs.They carry motor sensation. They are more susceptible to local anaesthetics.

The A delta fibres are myelinated . The diameter is 1-4µm and conducts at a speed of 5-25msecs. They carry pain,cold temperature and touch sensation. They are susceptible to local anaesthetics.

The B fibres are myelinated . The diameter is less than 3µm and conducts at a velocity of 3-15msecs. They carry preganglionic autonomic sympathetic functions. They are susceptible to local anaesthetics.

The C fibres are subdivided into sympathetic sC fibres and dorsal dC fibres. The sympathetic C fibre is not myelinated . It has a diameter of 0.3-1.3µm and conducts at a velocity of 0.7-1.3. They carry various autonomic functions. The dorsal dC fibre is not myelinated. It has a diameter of 0.4-1.2µm and conducts at a velocity of 0.1-2.0msec. They carry various autonomic functions, pain ,warm temperature,touch. They are less susceptible to local anaesthetics.

THEORIES OF LOCAL ANAESTHETIC ACTION:

The cell membrane of neuron permits potassium ion more readily than sodium ion. Hence the intracellular anions accumulates and a negative charge is maintained(-70mV).

The neurons maintains a voltage difference of the membrane potential of -60 to -90mV. The sodium and potassium pump transports three sodium ions for every two potassium ions. This creates a concentration gradient between

intarcellular and extracellular ions thereby the diffusion of potassium ions extracellularly and diffusion of sodium ions intracellularly occurs. According to Nernst equation(2) the nerve at rest behaves like a'' potassium electrode''.

According to the **''CORE MANTLE EFFECT''** the arrangement of nerves with in a plexus is not random, those supplying the distal structures in its territory lie at the centre or core whereas those supplyingthe more proximal areas are in the outer layer or mantle. In most mixed peripheral nerves the proportion of motor and sensory fibres is same in both mantle and core but not the case with brachial plexus. Large proportion of core fibres are those providing rich sensory innervation to hand and mantle predominantly motor to shoulder and elbow. The first sign of successful brachial plexus is weakness of either shoulder or elbow joint.

NEUROTRANSMITTERS:

The inflammatory mediators involved in the transmission of pain are substance P, Bradykinins, potassium, cytokines, histamine, serotonin, prostaglandins. These neurotransmitters activate or sensitise the peripheral nociceptors to pain.

ACTION POTENTIAL:

The neurons have voltage-gated sodium and potassium channels that responds to a mechanical ,electrical,chemical stimuli when depolarisation occurs at -55mV.The sodium channel are activated leading to influx of sodium

ions and generates action potential and conducts the impulse via nerve axon. If the depolarization exceeds a threshold level of -55mV, the voltage gated sodium channels are activated. So a sudden spontaneous influx of sodium ions occurs and hence an action potential is generated. The increase in sodium ions intracellularly causes a reversal of membrane potential of +35mV . Subsequently a rapid decrease in sodium permeability occurs along with an increase in potassium conductance through voltage gated potassium channels so that more potassium exits from the cell rendering the cell to return to a resting state.

The Sodium channels has one alpha subunit and one or two beta subunits. The alpha subunit is larger and beta subunit is smaller.The voltage gated sodium channels exists in three states namely in resting, activated and inactivated state.

The local anaesthetics bind to alpha subunit and thereby they block the voltage gated channel of sodium from inside the cell and with increase in the concentration of local anesthetic the impulse conduction is slow and the action potential magnitude decreases and the impulse propogation is abolished.

The local anaesthetics have affinity to activated and inactivated state of sodium channel and hence they are voltage and time dependent. They also block the potassium,calcium and NMDA receptors.Drugs like Amitriptyline,Meperidine,Ketamine and Volatile anaesthetics blocks the

sodium channels. Tetrodotoxin bind to sodium channel.

The sensitivity to blockade depends on the diameter of axon,degree of myelination.Small diameter and lack of myelination sheath enhances the sensitivity.

Local anaesthetics consist of lipophilic group – a benzene ring and a hydrophilic group- a tertiary amine.and separated by an ester or amide linkage.

They are weak base and carries a positive charge to the tertiary amine group. Based on the intermediate chain, the local anaesthetics are classified as amides or esters.

The physicochemical properties of the local anaesthetics depends on the substitutions in the aromatic ring and intermediate chain linkage and alkyl group attached to amine nitrogen.

The potency correlates with lipid solubility.If lipid soluble the drugs easily penetrates the cell membrane.The lipid solubility and thereby the potency increases with the increase in total munber of carbon atom in the molecule.The potency of local anaesthetic is affected by the myelination, size of fiber,pH,frequency of nerve stimuli and the electrolyte concentration like hypercalcemia,hypokalemia which antagonize the block.

The onset of local anaesthetic depends on lipid solubility and the concentration of non-ionized form and the ionized water soluble which

depends on the pKa. The onset of action of local anaesthetic directly correlates with the pKa.The pKa of lignocaine is 7.8 and that of bupivacaine is 8.1. The pKa of a compound is defined as the pH at which its ionized form is equal to the un-ionized form.The local anaesthetics are prepared as water soluble hydrochloride salt pH(6-7).The pH of local anaesthetic solution with adrenaline ranges from 4 to 5 as adrenaline is unstable at a pH of 6-7. The carbonated solution of local anaesthetic than hydrochloride salt has shortened onset of action.Differential sensory blockade may be desirable in selection of local anaesthetics and only Ropivacaine and Bupivacaine has some selectivity for sensory nerve.

Alkalinization of local anaesthetic liberates the free base, the carbondioxide rapidly diffuses in to the axon interior and the pH falls, which forces the dissociation of local anaesthetic to the active cation form. This effect results in ‘’ion trapping’’. Further favouring the rapid movement of local anaesthetic in to the axon.

The three major factors that determine the conduction-blocking profile of the local anaesthetics are the following:

1.**Lipid solubility**- Approximately 90% of cell membrane is composed of lipids. Local anesthetics are highly lipid soluble, and are able to penetrate the neuronal membrane more readily than less lipid soluble agents.This is reflected biologically in their increased potency.

2.**Protein binding**- Local anaesthetic agents which bind readily to proteins (eg.Sodium channels) hence prolong the activity

3**.pKa**-The speed of onset of local anaesthetic is directly related to the rate of penetration through the neuronal membrane,which in turn related to the amount of drug available in its non-protonated base form.when injected into tissue at physiologic pH(7.4),the percentage of drug present in base form is inversely related to its pKa. Local anaesthetics with pKa value close to physiological pH will have more rapid onset than those with higher pKa values.

PHARMACOLOGY

MECHANISM OF LOCAL ANAESTHETICS:(2)(4)(6)

Local anaethetics when deposited near the nerve;there occurs hydrolysis of aminoesters resulting in the penetration of the free drug in to the nerve sheath. The local anaesthetics permeate the axon membrane and resides in the axoplasm.The pH depends on the pKa of the individual drug.

The local anaesthetics binds to the voltage –gated sodium channelsthereby conformational change occurs and inhibits it. During the onset and recovery the partial and incompletely blocked nerve fibres are further inhibited that causes additional use dependent (phasic action) binding to sodium channels. The rate of onset and recovery depends on sole diffusion of local anaesthetics.Prolonged blockade occurs by the dissociation from sodium channels in few seconds.

PHARMACOKINETICS OF LOCAL ANAESTHETICS:

Local anaesthetics are weak bases that has a pKa value above the physiological pH. Hence <50% of local anaesthetics exists as non-ionized state.Local anaesthetics with pKa nearer to physiologic pH have more rapid onset of action**.**

Absorption:

The local anaesthetics have rapid onset but shorter duration of action when injected in to the sub-arachnoid space and sub-cutaneously. The onset of

of intrathecal bupivacaine is 5 minutes whereas in brachial plexus block it is 20 to 30 minutes. This occurs because the local anaesthetic is deposited at some distance from the nerve and it takes time to diffuse through various barriers. The systemic absorption which depends on:

i.Site of injection:Higher the vascularity more is the absorption.

ii.Presence of vasoconstrictors: The addition of adrenaline can

decrease the absorption and increases the uptake , enhance the quality and prolongs the duration of action . The addition of adrenaline to lignocaine prolongs the duration but when adrenaline is added to bupivacaine it has little or no significanteffect.

iii. Local anaesthetic agent:

Highly tissue bound are more slowly absorbed. They have intrinsic vasodilator property except for the cocaine which is a vasoconstrictor.

Distribution:

It depends on tissue perfusion:Highly perfused organs has rapid uptake.

Tissue/blood partition coefficient:

Lipid solubility is responsible for the rapid uptake whereas protein binding is responsible for longer duration of action.

Tissue mass:Muscle is the greatest reservoir for local anaesthetics.

Metabolism and Elimination:

Esters:

Esters are metabolised by Pseudocholinesterase.Procaine and benzocaine are metabolized to p-aminobenzoicacid which are allergic.

Amides:

Amides are metabolized inliver by microsomal P-450 (N-dealkylationandhydroxylation) .In cirrhosis of liver,congestive cardiac failure, the dosage of bupivacaine and other amides should be reduced.

Effects on organ system:

A.Central nervous system:

It is vulnerable for toxicity. Early symptoms include circumoral numbness,Paresthesia of tongue,dizziness,tinnitus and blurred vision, restlessness,agitation,paranoia,nervousness,slurred speech,drowsiness, seizures,unconsciousness.

For seizures :Inj.Midazolam 0.3mg/kg ; Inj. Thiopentone 1-2mg/kg to be given.

B. Respiratory system:

Relaxation of bronchial smooth muscles Apnea occurs due to depression of medullary centre of respiration.

C.Cardiovascular system:

Depression of myocardial automaticity, contractility and conduction.The dose required to produce cardiovascular toxicity is three times than that of

central nervous system. The effects are tachycardia,hypertension or hypotension atrio-ventricular block,arrhythmia,ventricular tachycardia and ventricular fibrillation.

D.Immunological:

Due to the the preservative Methylparaben. They inhibit the inflammatory response that activates the neutrophils by lysophosphatidic acid.

E.Musculoskeletal system:

`Local anaesthetics are myotoxic so necrosis can occur on intramuscular injection.

F.Hematological:

Lignocaine decreases coagulation and enhances fibrinolysis.

Drug interaction:

The local anaestheic action is potentiated by succinyl choline, opioids,alpha adrenergic agonist, cimetidine, propranolol, sodiumbicarbontae and potassium. Their effect is inhibited by Dibucaine.

BUPIVACAINE:

Introduction:

Bupivacaine is an amide local anaesthetic. The drug was synthesized by EKENSTAM in 1957 but was clinically used in the year 1963 by TELIVUO. The structure of Bupivacaine is same as mepivacaine with a butyl group in piperidine ring which is responsible for high lipid solubility and protein binding.

Structure of bupivacaine:

Bupivacaine hydrochloride is chemically 2-piperidine carboxamide,1-butyl-N-2,6 dimethyl phenyl monochloride, monohydrate. Bupivacaine molecule is a tertiary amine, separated from aromatic ring system by an intermediate chain. This chain contains the amide linkage (-NHCO), hence it is classified as an aminoamide compound. This amide linkage contributes to anaesthetic potency. The aromatic ring system gives lipophilic character to its portion of molecule

Mechanism of action:

Bupivacaine blocks the impulse by reducing the current through voltage activated sodium channels and prevents opening of the channel by inhibitory conformational changes.Clinically the order of loss of nerve function is as follows:

1.pain

2.Temperature

3.Touch

4.Proprioception

5.Skeletal muscle tone

Metabolism:

Bupivacaine is metabolized by aromatic hydroxylation in liver . The piperidine side chain is metabolised to form pipecolylxylidine(desbutyl bupivacaine). It is one-eighth as toxic as bupivacaine.Both desbutyl bupivacaine and unchanged bupivacaine are slowly excreted in equal proportions in the urine, only 5% bupivacaine is excreted unchanged in urine.Most important plasma protein binding site is alpha -1 - acid glycoprotein.

Safety dose:

Adrenaline does not have any effect on absorption

Maximum single dose -2-3mg/kg

Concentration used:

Peripheral nerve block - 0.25% -0.5%

Spinal anaesthesia - 0.5%

Infiltration - 0.125%-0.25%

Epidural -0.125%-0.75%

Characteristics of Bupivacaine:

pKa	8.1
Volume of distribution	0.4-0.9 litres/kg
Clearance	0.47 litres/kg
Protein binding	95%
Peak time of action	0.15-0.5hrs
Half-life	1.2-2.4hrs
Peak plasma concentration	0.8µg/ml
Toxic plasma concentration	>3µg/ml

Side-Effects:

1.ALLERGIC REACTIONS :

Allergic reactions are very rare to Bupivacaine as it does not get metabolized to para amino benzoic acid like ester group of local anaesthetics.

2.CROSS-SENSITIVITY:

Cross sensitivity occurs due to the metabolite para amino benzoic acid of ester group of local anaesthetics. So a patient with a known allergy to ester group of local anesthetic can receive amide group of local anaesthetic.

3.SYSTEMIC TOXICITY:

Systemic toxicity of local anaesthetic occurs when the plasma concentration of the drug exceeds the prescribed level. Toxicity depends upon the accidental direct injection of local anaesthetic in to the intravascular compartment or by systemic absorption. The magnitude of systemic absorption depends upon:

i. Dose administered

ii. Vascularity of the injection site

iii. Presence of epinephrine in the solution

iv. Physicochemical properties of the drug.

The systemic absorption is greater in the intercostal nerve block, intermediate for epidural and least for brachial plexus block.

4.CENTRAL NERVOUS SYSTEM:

Low plasma concentration produces numbness of tongue and circumoral tissues. As the plasma concentration is increased it crosses blood brain barrier and produces the following effects: Initial symptoms are restlessness, tinnitus,vertigo, difficulty in focusing.Later slurred speech, skeletal muscle twitching over the face, extremities, drowsiness, tonic-clonic seizures, hypotension, apnea. The plasma concentration of Bupivacaine at which seizures occurs is 4.5-5.5µg/ml. The cause of seizures is depression of inhibitory cortical neurons by local anaesthetics in the central nervous system.

Another explanation is inhibition of gamma amino butyric acid in the temporal lobe or amygdala by local anaesthetics.

Treatment:

Oxygen, Benzodiazepines, Muscle relaxants and Intermittent positive pressure ventilation,Hyperventilation(as hypercapnia and aciodosis decreases the protein binding of the local anaesthetic).

5.CARDIOVASCULAR SYSTEM:

A.Direct cardiac effects:

Bupivacaine is more cardio toxic than central nervous system.It is more resistant to toxic effects of high plasma concentration of local anaesthetics than central nervous system. Bupivacaine dissociates more slowly than lignocaine and blocks the sodium channels during diastole. Toxicity is enhanced by acidosis, hypoxemia, hypercarbia. Accidental intravascular injection causes hypotension, cardiac dysrhythmias, atrioventricular block, pregnancy increases the sensitivity of bupivacaine to cardiac toxicity due to increased free drug that occurs as a result of low alpha 1- acid glycoprotein. Tachycardia enhances frequency dependent blockade of sodium channels.The threshold for cardiac toxicity for bupivacaine may be decreased in patients on beta blockers, calcium

channel blockers,and on digitalis. Epinephrine and phenylephrine increases the cardio toxixity of Bupivacaine by enhancing the inhibition of cyclic Adenosine mono phosphate. The electrocardiograph changes would be

prolonged P-R interval, QRS interval, re-entry ventricular dysrhythmias.The R-enantiomer of bupivacaine is more cardiotoxic than S – enantiomer.

Treatment:

Lipid emulsion:Bolus 1.5 ml/kg over 1min.Continuous infusion 0.25ml/kg. Repeat bolus once or twice persistent cardiovascular collapse double the infusion rate to 0.5ml/kg/min.Recommended upper limit is approximately 10ml/kg.

Inj.Bretylium 5 to 10mg/kg i.v or i.m every 10 to 30 mins to a maximum dose of 30mg/kg.

B.Direct peripheral vascular effects:

Local anaesthtetics exerts biphasic effect on the peripheral vascular smooth muscles. Low concentration of Bupivacaine produces vasoconstriction whereas at high concentration produces vasodilatation . Exception to this is the Cocaine which produces vasoconstriction at all concentrations.

6.LOCAL TISSUE TOXICITY:

Bupivcaine when injected intramuscularly produces skeletal muscle damage which is more potent and more localized than lignocaine and prilocaine. But this effect is reversible and muscle regeneration occurs more rapidly with in two weeks.

POTASSIUM CHLORIDE:

Potassium ion is the second most common cation in the body. The concentration of potassium in the extracellular compartment is 4 meq/Litre and in the intracellular compartment it is 150meq/Litre .the total potassium present in the body is 3500mequivalents approximately. Potassium ion is a catalyst of numerous enzymatic reactions. It is also involved in the function of excitable cell membranes of nerves, skeletal muscle and cardiac muscles and is directly involved in the function of kidney.

A nerve impulse can be effectively blocked by an accumulation of potassium ions outside the neuron.Thus blockade produced by bupivacaine is re-inforced and prolonged by increased potassium ion concentration out of the nerve membrane as a result of the administration of exogenous potassium chloride.

Each 10ml ampoule contains 20mmoles of potassium ions, 1ml contains 2mmoles. The concentration in which it is available is 15%. In this study potassium chloride used is 15% of 0.2mmol of potassium chloride in 30ml of 0.375% of bupivacaine.

SODIUM BICARBONATE:

Alkalinization (carbonation) of Bupivacaine increases the pH>6 and thus it is less dependent on the buffering capacity of the tissues pH=7.4). On injection of this alkalinised solution the free base is liberated, carbondioxide rapidly diffuses into the axon interior and here the pH falls, which forces dissociation of local anaesthetic to the active cationic form. This effect results in 'ion-trapping' further favouring the rapid movement of the local anaesthetic into the axon. In this study it is used as 7.5% of 0.17mmol/litre (0.2ml) in 30ml of 0.375% of bupivacaine.

The sodium bicarbonate is available in 7.5% and 8.4% concentrations in 10ml ampoule. In this study the 7.5% concentration is used. It consists of 893mmoles of sodium bicarbonate in one litre in 7.5% concentration. In this study 0.2ml(0.17mmoles) of 7.5% of sodium bicarbonate is added to 30ml of 0.375% of Bupivacaine solution.

MATERIALS AND METHODS

This is a prospective randomizedsingle blinded study conducted at Government Rajaji Hospital, attached to Madurai Medical College, Madurai. Sixty patients of ASA grade I&II of either sex undergoing upper limb surgeries were randomly allocated into two groups; the Group BK and Group BS. Each group comprises of 30 patients. The brachial plexus block is performed with the patient lying supine with the head turned to contralateral side. Surgery was done under Subclavian perivascular approach of Brachial plexus Block by eliciting paraesthesia technique. The drug received by the two group are:

GROUP BK: 0.375% of Inj.Bupivacaine 30ml + Inj.Potassiumchloride 0.2mmol

(4 units in a Insulin syringe=0.1ml)

GROUP BS: 0.375% of Inj.Bupivacaine 30ml +Inj.Sodiumbicarbonate 0.17mmol

(8 units in a insulin syringe =0.2ml)

The following parameters were noted.

1.The onset of sensory loss

2.The onset of motor blockade

3.The duration of surgery

4.Intraoperative vital parameters(Pulse rate, Blood pressure, SPO_2, Respiratory rate, Electrocardiography)

5.Tourniquet tolerance

6.Duration of motor blockade

7.Duration of sensory blockade

8.Complications.

INCLUSION CRITERIA:

1.Age>18yrs

2.Both sexes

3.ASA I & II

4.Weight>40kg

EXCLUSION CRITERIA

1.Patient's refusal

2.Infection at the puncture site

3.Patients with documented neuromuscular disorders

4.Patients with hyperkalemia

5.Patients with respiratory compromise

6.Patients with known allergy to local anaesthetic drug

7ASA grade III and IV patients

8.Psychiatric illness

9.BMI> 35

10. Pregnancy

11.Bleeding diathesis

12. On Anti-coagulants

13.Peripheral Neuropathy

PreOperative period:

The following investigations were done: Hemoglobin, Random blood sugar, Serum Electrolytes, Urine- albumin and sugar, Chest x-ray, Electrocardiogram.

Procedure:

After ethical committee approval, an informed consent was obtained from the patients.The patients are explained about the procedure in their own mother tongue regarding the paraesthesia that will be elicited during the technique which is perceived as a ‘’electric shock’’ like or ‘’tingling’’ sensation in the elbow or in the fore-arm. Once the patient experiences the sensation he or she has to communicate with the performer.Then anintravenous access is secured and all the necessary monitors like SPO_2,Non- invasive blood pressure and

Electrocardiogram were connected. Anaesthesia machine checked and all the resuscitative equipments like endotracheal tube,laryngoscope, and drugs like calcium gluconate, midazolam, thiopentone, atropine, adrenalinewere kept ready and proceeded with the block.

All Patients were premedicated with a Inj.Midazolam 0.1mg/kg intramuscular to reduce anxiety one hour before surgery.

Technique:

The patient is positioned in supine, with the head turned 30º to the contralateral side and the arms are held in adduction position close to his or her body .A shoulder roll is kept in such a way to produce an head down of 30º.The area of neck is aseptically painted and draped. The posterior border of the sternocleidomastoid muscle is traced from the mastoid process to the insertion at the sternal and clavicular joint.The posterior edge of the sternocleidomastoid muscle is easily palpated by asking the patient to lift the head which makes it prominent. The subclavian artery is palpated from the lateral edge of the clavicular head of sternoclavicular joint at the level of sixth cervical vertebra.

An intradermal wheal is raised approximately 1cm above the midclavicular point. A 22 gauge intramuscular needle which is connected to the extension

tube with a 10ml syringe and a second anaesthetist is ready with loadedlocal anaesthetic solution to inject the drug . The needleis inserted posterior, downwards and in a medial direction and slowly walked in for eliciting paraesthesia. Once paraesthesia is elicited,30ml of 0.375% of inj. Bupivacaine with Inj. potassium chloride 0.2mmol in Group BK or 0.375% of inj. Bupivacaine with inj. sodium bicarbonate 0.17mmol in Group BS is injected by the second anaesthetist after confirming negative aspiration.

The time of onset of sensoryblock , is recorded using pinprick in fourth cervical tofirst thoracic dermatome once in every one minute till the blockade occurs.There after every one hour till patient regained normal sensations.The onset of sensory block is the time of injection of drug to time of loss of pain on pinprick. The sensory blockade is assessed and scored as follows.

SENSORY BLOCKADE IS SCORED AS:

0-No pain

1-Mild grimace

2-Moderate pain-Withdrawal

3-Severe pain-Screams

The time of onset of motor blockade is recorded by modified Bromage score. It is assessed every one minute till the motor blockade occurs. Thereafter every one hour it is assessed till recovery of motor power (flexion of elbow) .

MODIFIED BROMAGE SCORE:

0 :Able to raise extended arm to 90 º for 2secs

1 : Able to flex elbow and move fingers but unable to raise the extended arm

2 : Unable to flex the elbow but able to move the fingers

3 : Unable to move arm, elbow, fingers.

The duration of surgery , the duration of sensory block, the duration of motor block, the vital parameters and complications were recorded. The duration of sensory blockade was the time of onset of sensory block to the recurrence of pain to pinprick. The duration of motor blockade was the time of onset of loss of movements to the recurrence of movements.

The heart rate ,blood pressure , arterial saturation were recorded every 5 minutes intraoperatively. The patients were monitored for bradycardia, hypotension, convulsions,restlessness, disorientation, drowsiness or any other complications.

The quality of sensory and motor blockade was assessed based on the sensory and motor blockade as, complete sensory and motor blockade, some sparing of sensory and motor blockade and required supplemental drugs to continue surgery or total failure of sensory or motor blockade .

Grade 1: No supplemental drugs like opioids or sedatives arerequired in the intra operative period to continue the surgery

Grade 2: Analgesics and sedatives are given as supplementation due to inadequate blockade.

Grade 3: Due to complete failure of the blockade and hence converted to general anaesthesia and these patients are excluded from the study.

The change in pH observed in this study:

0.5%20ml of Bupivacaine	pH 5
0.375% of 30ml Bupivacaine	pH 5
0.375% of Bupivacaine with 0.1ml of potassium chloride	pH 5
0.375% of Bupivacaine with 0.2ml of sodiumbicarbonate	pH 7

DATA ANALYSIS

Statistical Tools (To be included at the end of Materials and Methods)

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

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 chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATION AND RESULTS

Group BK :Patients receiving 0.375% bupivacaine (20cc of 0.5% bupivacaine + 10cc of normal saline) with 0.2 mmol (4units in a insulin syringe=0.1ml) of 15% of inj.potassiumchlorideGroup BS : Patients receiving 0.375% bupivacaine (20cc of 0.5% Bupivacaine + 10cc of normal saline) with0.2ml of 7.5% of inj.Sodiumbicarbonate 0.17mmol(8units in a insulin syringe=0.2ml)

Table 1 : Age distribution

Age group	Age in years	
	Group BK	Group BS
Range	18 – 60 yrs	18 – 60 yrs
Mean	40.7 yrs	37.9 yrs
SD	14.3 yrs	14.5 yrs
'p'	0.4717 Not significant	

Age distribution in Group BK varies from 18-60years with a mean value of 40.7 and Standard deviation of 14.3 years. In Group BS it varies from 18-60years with a mean value of 37.9 years and standard deviation of 14.5 years On comparing both groups p value is 0.4717 which is not significant.

Table 2 : Sex distribution

Sex	Group BK		Group BS	
	No	**%**	**No**	**%**
Male	23	76.7	28	93.3
Female	7	23.3	2	6.7
Total	30	100	30	100
'p'	0.5 Not significant			

Sex distribution in Group BK Males were 23 and females were 7 whereas

In Group BS males were 28 and females were 2 whose p value is 0.5 which is statistically not significant.

Table 3 : ASA Grade

ASA Grade	Group BK		Group BS	
	No	**%**	**No**	**%**
I	24	80	22	73.3
II	6	20	8	26.7
Total	30	100	30	100
'p'	0.7602 Not significant			

In Group BK 24 patients come under ASA I and 6 in ASA II whereas in Group BS 22 patients come under ASA I and 8 in ASA II which are Statistically not significant

4.BMI

Group	BMI		
	Range	**Mean**	**S.D.**
Group BK	21.2–24.0	23.2	1.8
Group BS	21.6– 24.2	23.8	4.6
‘p’	0. 2675 Not Significant		

The BMI in Group BK ranges between 21.2-24.0with a mean of 23.2 and standard deviation 1.8 and in Group BS the BMI ranges from 21.6-24.2 with a mean of 23.8 and standard deviation of 4.6 whose p value is not significant.

Table 5 : Time of onset of sensory block

Parameter	Time of onset of sensory block (in minutes)	
	Group BK	**Group BS**
Range	6-8	8 – 11
Mean	7.63	9.83
SD	0.56	0.64
'p'	**0. 0001** **Significant**	

In Group BK the mean onset time for sensory block is 6-8mins, Standard deviation is 0.56 , whereas in Group BS the range is 8-11mins with a mean of 9.83mins and Standard deviation of 0.64 whose p value is 0.0001 which is statistically significant

Table 6 : Time of motor onset

Parameter	Time of motor onset (in minutes)	
	Group BK	Group BS
Range	3 – 4	5 – 6
Mean	3.9	5.2
SD	0.31	0.41
'p'	0. 0001 Significant	

In Group BK the onset of motor block ranges from3-4mins with a mean of 3.9mins And Standard deviation of 0.31whereas in Group BS the range varies from 5-6mins with a mean of 5.2,With a Standard deviation of 0.41 whose p value is significant.

Table 7: Duration of Surgery

Parameter	Duration of Surgery (in minutes)	
	Group BK	Group BS
Range	60 – 90	60-90
Mean	88	83
SD	14.1	18.1
‘p’	0. 0147 Significant	

In Group BK the duration of surgery varies from 60-90mins with a mean of 88mins and standard deviation of 14.1 and in Group BS it ranges from 60-90mins with a mean of 83 and standard deviation of 18.1 whose p value is 0.0147 which is not significant.

Table 8: Torniquet Tolerance

Torniquet Tolerance	Group BK		Group BS	
	No.	%	No.	%
Good	28	93.3	29	90
Fair	2	6.7	1	10
'p'	0.5 Not significant			

Tourniquet tolerance is Good in 28 patients and fair in 2 in Group BK Whereas it is Good in 29 patients and fair in 1 patient in Group BS whose p value is0.5 Which is not statistically significant.The fair category of patient did not require supplemental analgesics or sedatives.

Table 9: Quality of Anaesthesia

Quality of anaesthesia	Group BK		Group BS	
	No.	%	No.	%
Grade 1	28	93.3	29	90
Grade 2	2	6.7	1	10
'p'	0.5 Not significant			

The quality of anaesthesia is graded as grade 1 in 28 patients and grade 2 in 2 patients in Group BK Whereas it is grade 1 in 29 patients and grade 2 in 1 patient in Group BS whose p value is 0.5 which is not statistically significant.The grade 1 did not require any supplemental drugs like opioids or sedatives; the grade 2 patients received opioids and sedatives to proceed with surgery.

Table 9: Systolic Blood Pressure

Group	Systolic Blood Pressure (mm/Hg)		
	Range	Mean	S.D.
Group BK	100 - 140	117	13.2
Group BS	100 – 140	119	13.7
'p'	0. 555 Not Significant		

In Group BK the systolic blood pressure varies from 100-140mmHg with a mean of 117mmHg and standard deviation of 13.2 and in Group BS it ranges from 100-140mmHg with a mean of 119 and standard deviation of 13.17 , whose p value is 0.555 which is not significant

Table 10 :Diastolic Blood Pressure

Group	Diastolic Blood Pressure(mm/Hg)		
	Range	Mean	S.D.
Group BK	60 – 90	70.7	8.3
Group BS	60 – 90	70.3	8.1
‘p’	0. 9425 Not Significant		

In Group BK the Diastolic blood pressure varies from 60-90mmHg with a mean of 70.7mmHg and standard deviation of 8.3 and in Group BS it ranges from 60-90mmHg with a mean of 70.3 and standard deviation of 8.1,whose p value is0.9425 which is not significant

Table 11:Pulse Rate

Group	Pulse Rate		
	Range	Mean	S.D.
Group BK	66 – 92	75.9	7.3
Group BS	60 – 94	76	9.3
'p'	0. 8236 Not Significant		

In Group BK the pulse Rate varies from 60-92/min with a mean of

75.9/min and standard deviation of 75.9 and in Group BS it ranges f60-94/min with a mean of 76 and standard deviation of 9.3 which is not significant.

Table 12 :SPO_2

Group	SPO_2		
	Range	Mean	S.D.
Group BK	97 – 99	98.5	0.7
Group BS	97 – 99	98.5	0.7
'p'	1.0 Not Significant		

In Group BK the Spo_2 varies from 97%-99% with a mean of 98.5% and standard deviation of 0.7 and in Group BS it ranges from 97%-99% with a mean of 98.5% and standard deviation of 0.7 whose p value is 1.0 which is not significant

Table 13: ECG changes

Group	Complications			
	Present		Absent	
	No	%	No	%
Group BK	-	-	30	100
Group BS	-	-	30	100

In Group BK and in Group BS there were no ECG changes

Table 14 :Sensory Block Duration

Group	Sensory Block Duration (in minutes)		
	Range	Mean	S.D.
Group BK	420 – 480	476.6	13.2
Group BS	600 – 720	608	26.1
‘p’	0. 0001 Significant		

In Group BK the duration of sensory blockade the varies 420-480mins with a mean of 476.6mins and Standard deviation of 13.2. In Group BS 600-720 mins with a mean 608 mins and Standard deviation of 26.1 and the P value between the two groups is significant.

Table 15 :Duration of Motor Block

Group	Duration of Motor Block (in minutes)		
	Range	Mean	S.D.
Group BK	240 – 270	245	11.4
Group BS	360 – 420	417	12.1
‘p’	0. 0001 Significant		

In Group BK the duration of motor block ranges from 240-270mins with a mean of 245mins and Standard deviation of 11.4 and in Group BS the doration ranges from 360-420mins with a mean of 417mins and Standard deviation 12.1 whose p value is 0.0001 which is statistically significant

Table 16 : Complications

Group	Complications			
	Present		Absent	
	No	%	No	%
Group BK	-	-	30	100
Group BS	-	-	30	100

In Group BK and Group BS complications like nausea ,vomiting. Hypotension, hypertensionTachycardia. Bradycardia.

REVIEW OF LITERATURE

1.**British Journal of Anaesthesia [1986 Mar, 58(3):297-300]**

A double blinded comparison of prilocaine and prilocaine with potassium chloride and of plain bupivacaine with bupivacaine plus potassium chloride, in brachial plexus blockade by axillary approach was obtained in two groups of 20 patients**.** They observed that addition of potassium chloride 0.2mmol to a solution of 0.25% Bupivacaine 40ml for brachial plexus blockade has advantages over the use of the plain solution. The sensory blockade suitable was achieved by 25mins (it takes a minimum of 30mins for axillary block with a plain solution to be suitable for surgery). The addition of potassium chloride 0.2mmol resulted in a more rapid onset of sensory loss.

2.British Journal of Anesthesia 1966;38:857-864

Bromage,P.R et al., and Burfoot,M.F et al.(1966): He conducted a comparative study between lignocaine with potassium chloride and lignocaine with hyaluronidase in epidural anaesthesia which revealed that hyaluronidase impairs the quality of sensory blockade rather than enhancingit,whereas potassium chloride gave rise to a shortened latency of spread and a more intense quality of sensory blockade.

3.M.D.Bedder et al and associates :Anesthesia Analgesia 1988;67:68:

A comparative study between Bupivacaine and alkalinized bupivacaine in brachial plexus block was conducted between two groups of 30 patients in each group. Group I received 0.5%bupivacaine (pH,5.5) 3mg/kg, while Group II received alkalinized bupivacaine 0.5% (pH,7.05-7.15) 3 mg/kg. The onset of sensory block, motor block was assessed. The time to onset of sensory blockade(Group I,17.7±1.8min and in Group II ,16.3±0.9min) did not differ significantly between the groups. Similarly, no difference in time to onset of motor blockade (Group I,6.9±1.7min and in Group II 6.3±1.5min) or time to peak motor effect(GroupI 18.1±1.9min; Group II, 15.1±1.9mins) was observed. Regression of post operative sensory and motor blockade was similar in both groups.

4.Indian Journal of Anaesthsia 2003;47(4):283-286.

Dr.Ruby Mehtal ,Dr.D.D.Verma,Dr.Veena Gupta ,Dr.A.K.Gurwara et al conducted a study and observed that alkalinization of 2% lignocaine hydrochloride with adrenaline leads to alteration in its onset of action and duration of action. 60 patients between the age group 20-60years were subjected to the study.Group I received 20ml of lignocaine hydrochloride with adrenaline

(pH=3.21). Group II received 20ml of freshly prepared solution by addition of 1mlof 7.5% sodium bicarbonate to standard solution (pH=6.21). Group III received 20ml of alkalinized standard solution prepared by addition of 2ml of 7.5% sodiumbicarbonate (pH=6.67).Supraclavicular brachial plexus block was performed . The onset of sensory block,onset of motor block,quality of block and duration of block was assessed.It was observed that raising the pH of solution from 3.21 to 6.21 produced a reduction in latency of sensory block. (18.35 minutes to 10.35minutes),a reduction in latency of motor block (20.65 minutes to 12.2minutes) and increased in the duration of block.Further increase of pH from 6.21 to 6.67 did not confer any added advantage. Frequency of complete block in solution with pH6.21 was seen to increase as compared to pH 3.21 solution(35% to 80%).

5.Masui Journal of Anaesthesia 2006; 62 : 116-118

A comparative study between two groups of patients (200 each), one group receiving hyaluronidase mixed anaesthetic and the other sodiumbicarbonate buffered anaesthetic. Peribulbar block was performed.The groups were compared

for effectiveness of the anaesthesia, its onset, duration of blockade. They concluded that sodium bicarbonate was shown to reduce the time of onset of sensory block and to increase the quality of blockade without anyadverse effects.

6. Indian journal of Anaesthesia 1990;38:119-122.

Khosa DS, Thind SS, Gupta HK, Jain et al conducted a study where potassium chloride was added to lignocaine and bupivacaine solutions in brachial plexus block. The time of onset of sensory block , motor block and duration of analgesia was noted. They concluded that addition of potassium chloride to bupivacaine in brachial plexus block significantly enhanced the onset of sensory blockade and prolonged the total duration of analgesia when compared to lignocaine.

7. British journal of Anaesthesia 1983;

Kircha SS, Barsa J,Fink BR et al. conducted a study and observed that Potentiation of nerve block in vivo by physiological adjuvants in the solution. Addition of potassium chloride doubled the duration of blockade produced by a solution of lignocaine in plain isotonic sodium chloride whereas conduction in isolated nerve can be accelerated by raising the extraneural potassium

concentration.

8.Hardy et al performed a study to assess the minimum concentration of bupivacaine on stellate ganglion block, and the effect of potassium chloride added to bupivacaine. He concluded that potassium chloride has a major effect on the minimum concentration of bupivacaine for sympathetic blockade.

9.Parris and Chamber et al conducted a study and concluded that potassium chloride made no differences to the characteristics of the block with prilocaine,but resulted in a more rapid onset of sensory loss when added to bupivacaine.

10.Fink and Calkins et al conducted a study and concluded that conduction in isolated nerve can be accelerated by raising the extraneural potassium chloride concentrations.

11. Ritchie et al confirmed that the cation is the anaesthetically active form of local anaesthetic and uncharged molecule is essential for penetration in to the intracellular receptor site. The alkaline solution are more effective in sheathed preparations. Bupivacaine 0.5% has a pKa of 8.1. When sodiumbicarbonate added to Bupivacaineit results in rapid onset of sensory block and increased

the quality of blockade.

12.Galindo et al concluded that pH adjusted solutions of local anaesthetics pH(7-7.4). produced a more rapid onset of blockade and duration of analgesia.pH adjusted bupivacaine was studied using a prospective randomized double blinded design in epidural analgesia for parturients.0.25% Bupivcaine with the pH increased from 5.65 to 7.26 was used. The time of onset of sensory blockade was reduced while the time to reach peak effect was unaffected with alkalinization. A statistically significant increased duration of analgesia was observed (79.4 Vs 96.5 mins) with alkalinized bupivacaine 0.25%

13. Hilgier et al compared 0.5% bupivacaine with epinephrine 1:200,000 (pH 3.9) with an alkalinized solution of bupivacaine with 1:200,000 (pH 6.4) when used for brachial plexus block. He reported that alkalinization of the bupivacaine solution increases onset and prolongs the duration of sensory blockade in subclavian perivascular brachial plexus blockade.

DISCUSSION

Brachial plexus block is widely used in our day to day practice for elective as well as emergency upperlimb surgeries. It provides better intra-operative as well as post operative analgesia. Inorder to provide better quality of anaesthesia intraoperatively as well as to prolong the duration of post operative analgesia various adjuvants are added to local anaesthetic solution. Among the adjuvants sodiumbicarbonate and potassium chloride have stood the test of time, apart from the opioids.

The addition of sodiumbicarbonate to Bupivacaine alkalinizes the pH of the local anaesthetic solution thereby free base is liberated, ion trapping occurs and hence the onset of sensory block is enhanced rapidly. The addition of potassium chloride to Bupivacaine increases the extracellular concentration and depolarizes the nerve membrane and thus blocks the conduction of nerve impulses. The addition of potassium chloride to bupivacaine shortens the onset of sensory and motor blockade whereas the addition of sodiumbicarbonate to bupivacaine prolongs the duration of sensory and motor blockade in brachial plexus block.

The addition of potassium chloride as an adjuvant to Bupivacaine solution will cause depolarization of the cell membrane and thereby it

provides better quality of analgesia, better tourniquet tolerance as well as prolongs the duration of analgesia . It also enhances the onset of sensory blockade which would be prolonged with plain bupivacine alone without any detrimental side effect even when injected intravascularly because 0.2mmoles of potassium chloride used in this study is too low to cause the cardiovascular complications.

Sodium bicarbonate when added as an adjuvant to Bupivacaine will change the pH to alkaline state and thereby it enhances the onset of sensory blockade as well as provides better quality of analgesia, better tourniquet tolerance and prolongs the duration of analgesia.

The addition of potassium chloride and alkalinization of the local anaesthetic solution is used which is cost effective as well as do not have any adverse effect on the hemodynamic status of the individual.The addition of carbonates and alkalinization have stood the test of time.

This study was carried out in sixty patients of ASA I and II with demographic data in terms of age, weight, sex, body mass index were similar in both the groups.

The mean time of onset of sensory blockade in Group BK7.63mins, whereas in Group BS the mean time of onset of sensory block is 9.8mins. The

mean time of onset of motor blockade in Group BK is 3.9mins ,whereas in Group BS the mean time of onset of motor blockade is 5.2 mins. The result of this study supports the findings of Khosa et al who showed that addition of potassium chloride to bupivacaine significantly enhanced the onset of both sensory and motor blockade.In contrast to this study the delayed onset of blockade proposed by Parris and Chamber et al may be due to the lower concentration of Bupivacaine (0.25%) when compared to this study (0.375%).

The quality of sensory and motor block was same in both the groups . The patients did not require supplemental analgesics or sedatives in both the group. Bromage and Burfoot also found the intense quality of blockade when potassium was added to lignocaine in epidural blockade. This is also correlates with the results of Parris and Chamber et al .

The duration of sensory and motor blockade was significantly increased in both the groups more so in BS group.This observation correlates with Khosa et al who found prolonged duration of analgesia.

SUMMARY

Sixty patients of ASA grade I&II of either sex undergoing upper limb surgeries were randomly allocated into two Groups BK and BS. Each group comprised of 30 patients.

GROUP BK: 0.375% of Inj.Bupivacaine + Inj.Potassiumchloride 0.2mmol (4 units in a Insulin syringe=0.1ml)

GROUP BS: 0.375% of Inj.Bupivacaine +Inj.Sodiumbicarbonate 0.17mmol (8 units in a insulin syringe =0.2ml)

Surgery was done under Subclavian perivascular approach of Brachial plexus Block. Supraclavicular brachial plexus block is given with the patient lying supine with the head turned to opposite side after eliciting paraesthesia.

The onset and duration of sensory loss and motor blockade are studied. The loss of pinprick sensation is checked in C4 toT2 skin dermatome every 1 minute till the onset of loss of sensation and every 1 hourly till the regain of sensation. The motor blockade is assessed every 1 minute by modified Bromage score and every 1 hourly till the regain of movements. The following parameters were observed:

1.Onset of sensory block

2.Onset of motor block

3.Duration of sensory block

4.Duration of motor block

5.Tourniquet tolerance

6.Quality of block.

7.Hemodynamic stability

8.Complications

Potassium chloride when added as an adjuvant to Bupivacaine has sensory onset of 6-8mins(7.63±0.56) and motor onset of 3-4mins(3.9±0.31).Sodiumbicarbonate when added as an adjuvant to Bupivacaine has sensory onset of 8-11 mins(9.8±0.64) and motor onset of 5-6mins(5.2±0.41).The duration of motor block is 240-270mins(245±11.4) in Group BK and 360-420 mins(417±12.1) in Group BS.The duration of sensory block is 420-480mins(476.6±13.2)in Potassium chloride BK group whereas the duration of sensory block is 600-720 mins(608±26.1) in Sodiumbicarbonate BS group.

CONCLUSION

Potassium chloride when added as an adjuvant to Bupivacaine has sensory onset (Range(mean±standard deviation)) of 6-8mins(7.63±0.56) and motor onset of 3-4 mins(3.9±0.31)

Sodiumbicarbonate when added as an adjuvant to Bupivacaine has sensory onset of 8-11 mins(9.8±0.64) and motor onset of 5-6mins(5.2±0.41)

The duration of motor block is 240-270mins(245±11.4) in Group BK and 360-420 mins(417±12.1) in Group BS.

The duration of sensory blockade is 420-480mins(476.6±13.2) in Potassium Chloride group whereas 600-720 mins(608±26.1) in Sodiumbicarbonate group.

Hence it is concluded that , in brachial plexus blockade,

The addition of potassium chloride as an adjuvant to bupivacaine shortens the onset time of sensory and motor blockade whereas the addition of sodiumbicarbonate prolongs the duration of sensory and motor blockade.The quality of anaesthesia is good in both the groups.

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34.Pharmacology and physiology in Anaesthesiology practice by

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4)Clinical anaesthesiology by G.Edward Morgan Jr. Fourth edition

5)Lee ‘s Synopsis of Anaesthesia; Thirteenth edition

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Robert K. Stoelting ; Fourth edition

STUDY PROFORMA

1.NAME :

I.P.NO: ASA:

2.AGE& SEX :

3.WEIGHT :

4.DATE& TIME OF ADMISSION :

5.DIAGNOSIS :

6.PROCEDURE :

7.HISTORY : ALLERGY TO DRUGS, BLEEDING DISORDERS, SYSTEMIC ILLNESS

8.CLINICAL EXAMINATION : PR,BP, SPO2, ,BMI, CVS,RS,CNS

9.BASIC INVESTIGATIONS:

HAEMOGLOBIN-

RENAL PARAMETERS-

BLOOD SUGAR

SERUM ELECTROLYTES-

CHEST X RAY PA VIEW-

ECG-

10.ANAESTHETIC TECHNIQUE: SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK BY SUBCLAVIAN PERIVASCULAR APPROACH

11.DOSAGE OF DRUG :

GROUPBK :INJ. BUPIVACAINE 0.375% (30ml)+ INJ. POTASSIUM CHLORIDE 0.1ML(0.2mmol/l)

GROUP BS :INJ. BUPIVACAINE 0.375% (30ml)+ INJ. SODABICARBONATE 7.5%(0.2ml)

12.SENSORY BLOCKADE : (Loss of Pin Prick at C4 to T2 Dermatome every 2 mins till blockade.)

ONSET TIME:

13.MOTOR BLOCKADE : (Modified Bromage score): Every 2 minstill Blockade

14.TOURNIQUET TOLERANCE: Good or Fair

15.QUALITY OF ANAESTHESIA: Grade 1/Grade 2/Grade 3

SENSORY BLOCKADE IS SCORED AS:

0-No pain

1-Mild grimace

2-Moderate pain-Withdrawal

3-Severe pain-Screams

MODIFIED BROMAGE SCORE:

0 : Able to raise extended arm to 90º for 2secs

1 : Able to flex elbow and move fingers but unable to raisr the extended arm

2 : Unable to flex the elbow but able to move the fingers

3 : Unable to move arm, elbow, fingers.

THE QUALITY OF ANAESTHESIA:

Grade 1:No supplemental drugs like opioids or sedatives are required in the intra operative period to continue the surgery

Grade 2: Analgesics and sedatives are given as supplementation due to inadequate blockade.

Grade 3:Due to complete failure of the blockade and hence converted to general anaesthesia and these patients are excluded from the study.

DURATION OF SURGERY :

TOURNIQUET : TIME OF APPLICATION :

TIME OF DEFLATION :

MONITORING OF VITALS EVERY 15 MINS : (ANXIOLYTICS, SEDATIVES, ANTI EMETICS AND OTHERS TO BE MENTIONED)

VITALS	**5MINS**	**15MINS**	**30MINS**	**45MINS**	**1HR**	**1.15HRS**	**1.30 HRS**	2HRS	2.30 HRS
PULSE									
B.P									
RR PATTERN									
SPO2									
ECG									
SIDE EFFECTS									
REMARKS									

POST OP PERIOD : MONITORING OF VITALS :

PARAMETERS	30MINS	1HR	2HR	4HRS	6HRS	8HRS	10HRS	12HRS	24HRS
PULSE									
B.P									
RR									
SPO2									
VAS SCORE									
SIDE EFFECTS									
REMARKS									

RECOVERY OF SENSORY BLOCKADE :

(TIME FROM BRACHIAL PLEXUS BLOCK UNTIL THE TIME AT WHICH PT REQUIRED POST OP ANALGESIA)

(POST OP ANALGESIA GIVEN WHEN VAS(VISUAL ANALOGUE SCORE) SCORE GOES ABOVE 4)

RECOVERY OF MOTOR BLOCKADE :

(TIME AT WHICH PT COULD FLEX THE ELBOW).

GUIDE TO MASTER CHART

ABBREVIATIONS USED IN MASTER CHART:

F-Finger

F1-Thumb

F2-Ring finger

F3-Middle finger

F4-Ring finger

F5-Little finger

Rt-Right

Lt-Left

Amp-Amputation

#-Fracture

BB- Both Bone

FA- Fore- Arm

Dis Rad- Distal Radius

Inj- Injury

Tend- Tendon

Dislcn- Dislocation

cmc-Carpo meta carpal joint

jt- joint

SOR- Shaft of Radius

Seg- Segment

Gang- Ganglion

Ze-Zone

Gr-Grade

SSG- Split skin graft

ORIF- Open Reduction and internal fixation

k-wire- Kirschner’s wire

Ex-Fix- External fixation

Trmtic- Traumatic

BE- Below Elbow

Fix- Fixator

Rev Amp- Revision Amputation

GROUP - BK: INJ.0.375 % BUPIVACAINE WITH POTASSIUM CHLORIDE 0.2mmol															
S. No	NAME	AGE / SEX	IPNO	BMI	ASA	DIAGNOSIS	PROCUDURE	SENSORY ONSET MINS	MOTOR ONSET MINS	DURATION OF SURGERY (MINS)	TOURNIQUET TOLERENCE	QUALITY OF ANAESTHESIA	DURATION OF SENSORY BLOCK MINS	DURATION OF MOTOR BLOCK MINS
1	BANUMATHY	18/F	13966	21	I	Flexor injury F3 F4	REPAIR	8	3	60	GOOD	Grade 1	420	240
2	ARUMUGATHAMMAL	60/F	67370	22	II	Raw area Rt	SSG	8	3	60	GOOD	Grade 1	430	240
3	SAIBUDDIN	19/M	65613	22.6	I	Traumtic ampF2	EXT.FIX	8	3	60	GOOD	Grade 1	480	270
4	MUTHU	23/M	9278	22.8	I	Galeazzi #	ORIF	8	5	60	GOOD	Grade 1	480	270
5	AMIRTHJAVALLI	58/F	21455	24	II	#BB FA	ORIF	8	5	60	GOOD	Grade 1	480	240
6	REKHA	23F	59681	23.8	I	#BB FA	ORIF	8	5	60	GOOD	Grade 1	480	270
7	KALYANASUNDARAM	25/M	5732	21.6	I	#DISTAL	ORIF	8	5	60	GOOD	Grade 1	480	270
8	GOPALAKRISHNAN	26/M	62341	23.8	I	#ULNA STYLOID	K WIRE	8	5	60	GOOD	Grade 1	480	270
9	SUSEELA	19/F	60325	22.6	I	FleX Tendon injF3	REPAIR	8	5	90	GOOD	Grade 1	480	270
10	ARIVAZHAGAN	28/M	63201	22.4	I	Zone II F4Tendon	TENDON	8	5	60	GOOD	Grade 1	480	270
11	MAHESHWARAN	28/M	936	21.7	I	Dislocation Lt	K WIRE	8	5	60	GOOD	Grade 1	480	270
12	THAVAMANI	31/M	82649	23.9	I	Flexor injury Zone	REPAIR	8	5	90	GOOD	Grade 1	480	270
13	BALABURUGAN	32/M	5794	23.8	I	Lt hand Raw area	SSG	8	5	60	GOOD	Grade 1	480	270
14	LAKSHMANAN	35/M	1637	22.7	I	# Distal Radius	ORIF	8	5	90	GOOD	Grade 1	480	270
15	SURESHKUMAR	35/M	4340	21.4	I	Raw area Lt FA	SSG	8	5	90	GOOD	Grade 1	480	270
16	KARUPPUSAMY	35/M	64007	23.6	I	DistalF4PSNerve	REPAIR	8	5	60	GOOD	Grade 1	480	270
17	APPAVU	38/M	65676	23.2	II	#RADIUS LT	ORIF	8	5	60	GOOD	Grade 1	480	240
18	MAHENDRAN	38/M	70429	21.8	I	Raw area Lt FA	SSG	8	5	90	GOOD	Grade 1	480	270
19	KANNAN	38/M	813	22.9	I	Raw area BE Rt	SSG	8	5	60	GOOD	Grade 1	480	270
20	KALI	40/M	3662	23.5	II	Raw area BE Lt	SSG	8	5	60	GOOD	Grade 2	480	270
21	THANGARAJ	50/M	58327	23.6	II	#SOR Lt	ORIF	8	5	60	GOOD	Grade 1	480	240
22	PANCHAVARNAM	48/F	57621	22.4	I	#BB FA Lt	ORIF	8	5	60	FAIR	Grade 1	480	270
23	KALEESHWARI	54/F	62326	21.8	II	Raw area Rt FA	SSG	8	5	60	GOOD	Grade 1	480	270
24	ARUMUGAM	60/M	58234	22.6	I	#Elbow	ORIF	8	5	60	GOOD	Grade 1	480	240
25	KARUPPAIAH	58/M	63801	23.7	I	#SOR	ORIF	8	5	90	GOOD	Grade 1	480	270
26	SHARATHKUMAR	20/M	19900	23.9	I	#BB FA Lt	ORIF	8	5	60	GOOD	Grade 1	480	270
27	RAJKUMAR	60/M	19816	21.8	I	Raw area Rt FA	SSG	8	5	90	FAIR	Grade 1	480	270
28	ARUMUGAM	54/M	57224	22.6	I	#ELBOW RT	ORIF	8	5	60	GOOD	Grade 1	480	270
29	SIVAKUMAR	34/M	20225	21.5	I	Amputation BE Rt	SSG	8	5	60	GOOD	Grade 2	480	270
30	RAVIRAM	32/M	60322	23.7	I	#Elbow LT	ORIF	8	5	60	GOOD	Grade 1	480	270

GROUP -BS: INJ.0.375% BUPIVACAINE WITH SODIUMBICARBONATE 0.17mmol														
S. No.	NAME	AGE / SEX	IPNO	BMI	ASA	DIAGNOSIS	PROCUDURE	SENSORY ONSET MINS	MOTOR ONSET MINS	DURATION OF SURGERY (MINS)	TOURNIQUET TOLERENCE	QUALITY OF ANAESTHESIA	DURATION OF SENSORY BLOCK MINS	DURATION OF MOTOR BLOCK MINS
1	KANNAN	18/M	4340	21.7	I	Flex inj F3	REPAIR	8	5	60	GOOD	Grade 1	600	360
2	PALPANDI	18/M	83641	22.8	I	Raw area Rt	SSG	9	5	90	GOOD	Grade 1	660	420
3	RAJALAKSHMI	19/F	73519	23.2	I	TrmticAmpF2	EXT.FIX	9	5	60	GOOD	Grade 1	660	420
4	SIVAKUMAR	25	564	24.1	I	Galazzei #	ORIF	9	5	90	GOOD	Grade 1	660	360
5	UNJAMMAL	56/F	1984	23.8	II	Amputation BE	SSG	9	5	60	GOOD	Grade 1	660	420
6	PRAKASH	34/M	80813	22.6	I	FlexinjF3 F4	REPAIR	10	5	90	GOOD	Grade 1	660	420
7	RAMAKRISHNAN	38/M	81873	23.9	I	Raw area Rt	SSG	10	5	90	GOOD	Grade 1	660	420
8	POORNAHARI	34/M	368	22.7	I	TramticF2-F5	EXT.FIX	10	5	60	GOOD	Grade 1	660	420
9	KANNUSAMY	36/M	1140	21.9	I	Galazzei # RT	ORIF	10	5	60	GOOD	Grade 1	660	420
10	THANGAPPAN	38/M	73999	22	II	#BB FA	ORIF	10	5	90	GOOD	Grade 1	660	360
11	BASKARAN	19/M	81489	22.8	I	Raw area BE	SSG	10	5	60	FAIR	Grade 1	660	420
12	RAMASAMY	54/M	83698	23.6	II	#F3F4 Rt Hand	K WIRE	10	5	60	GOOD	Grade 1	660	420
13	SHAHULHAMEED	24/M	4300	23.9	I	SOR Lt	ORIF	10	5	60	GOOD	Grade 2	660	420
14	SATHYARAJ	56/M	9111	24.1	II	Flexor tendon	REPAIR	10	5	60	GOOD	Grade 1	660	420
15	RAJESH	34/M	7979	22.7	I	Raw area FA	SSG	10	5	60	GOOD	Grade 1	660	360
16	ALAGARSAMY	38/M	9468	21.9	I	#Montaggia L	ORIF	10	5	60	GOOD	Grade 1	660	420
17	RAMU	60/M	9341	22.8	II	#Distal Rad L	ORIF	10	5	60	FAIR	Grade 1	660	420
18	GANDHI	58/M	2233	22.9	II	#olecranon	K WIRE	10	5	60	GOOD	Grade 1	660	360
19	KALI	34/M	80459	23.8	I	# Shaft Rad L	ORIF	10	5	60	GOOD	Grade 1	660	420
20	THAVAMANI	38/M	5980	24.2	I	Seg # BB FA	ORIF	10	5	60	GOOD	Grade 1	660	420
21	AJITH	19/M	2631	23.4	I	#BB Rt hand	ORIF	10	5	60	GOOD	Grade 1	600	420
22	PRABHAKARAN	32/M	1449	23.8	I	BE amp R	REV.AMPUTA	10	5	60	GOOD	Grade 1	660	420
23	KALIYAPERUMAL	38/M	1442	21.9	I	GANGLION RT	EXCISION	10	5	90	GOOD	Grade 1	660	420
24	VIJAY	24/M	3662	22.3	I	Lt Ulnar Bursitis	EXCISION	10	5	60	GOOD	Grade 1	660	420
25	VELLAIYAN	45/M	82649	22.9	I	# BB FA Rt	ORIF	10	6	60	GOOD	Grade 1	660	360
26	NAGAPPAN	48/M	35	23.6	I	# BB FA Lt	ORIF	10	6	90	GOOD	Grade 1	660	420
27	MUNIYANDI	54/M	77739	23.8	II	TraumatAmputatio	EXT.FIX	10	6	60	FAIR	Grade 1	600	420
28	GANESAN	39/M	76422	21.9	I	# Gr-I Distal radius	EXT.FIX	10	6	60	GOOD	Grade 1	660	420
29	PANDIYAN	47/M	89063	23.4	I	#BB Rt hand	ORIF	10	6	60	GOOD	Grade 1	660	420
30	ALAGUPILLAI	59/M	83093	23.9	II	Rt BE amputation	REVISION	10	6	60	GOOD	Grade 1	660	420

Ref. No. 5336 /E4/3/2012

Govt. Rajaji Hospital,
Madurai.20. Dated: .08.2012

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 28.06.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

---

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

21/8/12

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Dr. Sobanadevi. P	M.D Anaesth	Potassium chloride vs. sodium bicarbonate as adjuvantto bupivacaine in enhancing brachial plexus block.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.
She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8.She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

17.8.12

DEAN I/c

To
All the above members and Head of the Departments concerned.
All the Applicants.

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
MADURAI 625020.

COMPARATIVE STUDY OF THE EFFICACY OF POTASSIUM CHLORIDE AND SODIUM BICARBONATE AS AN ADJUVANT TO BUPIVACAINE IN SUPRACLAVICULAR SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

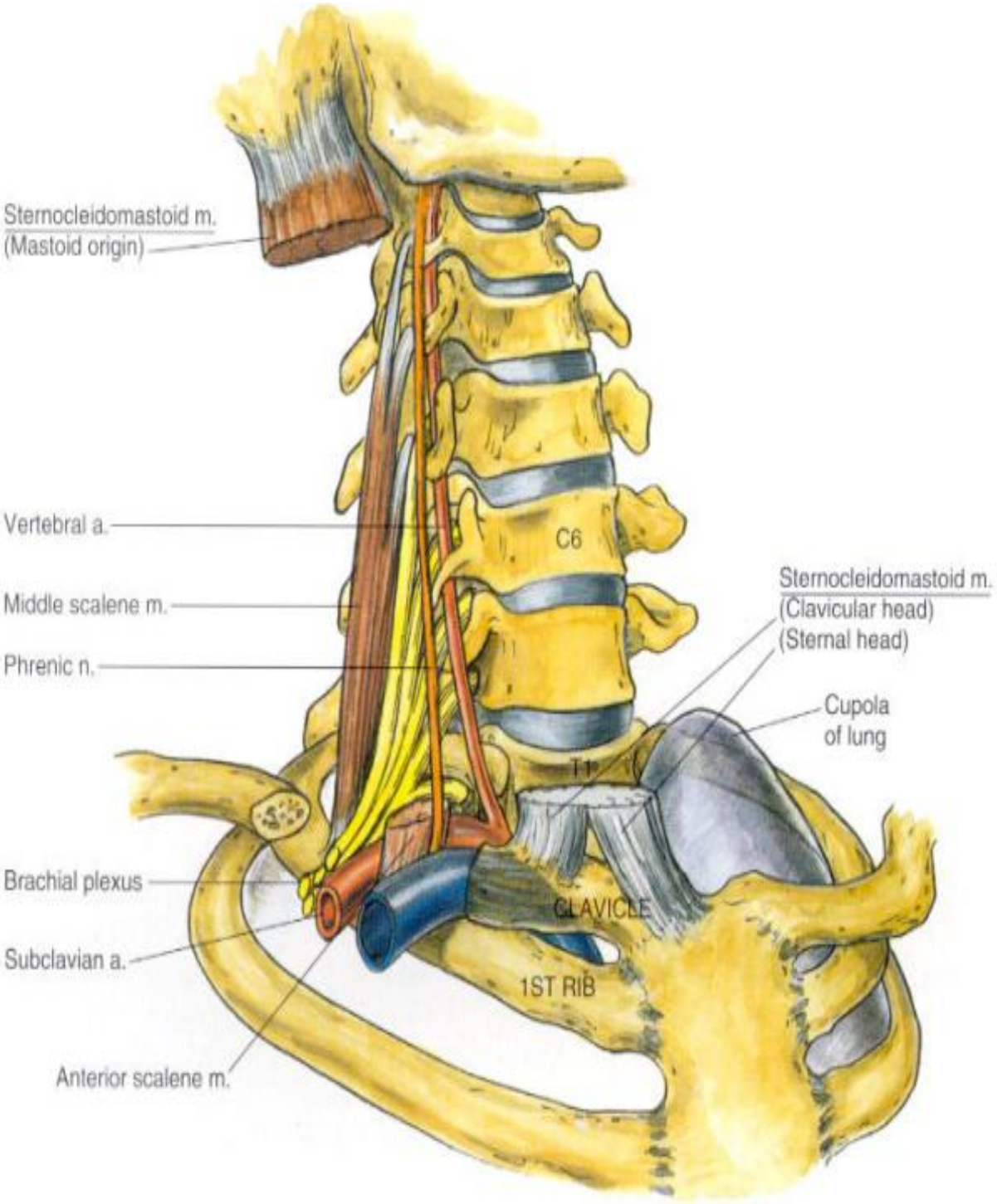
APRIL-2013

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

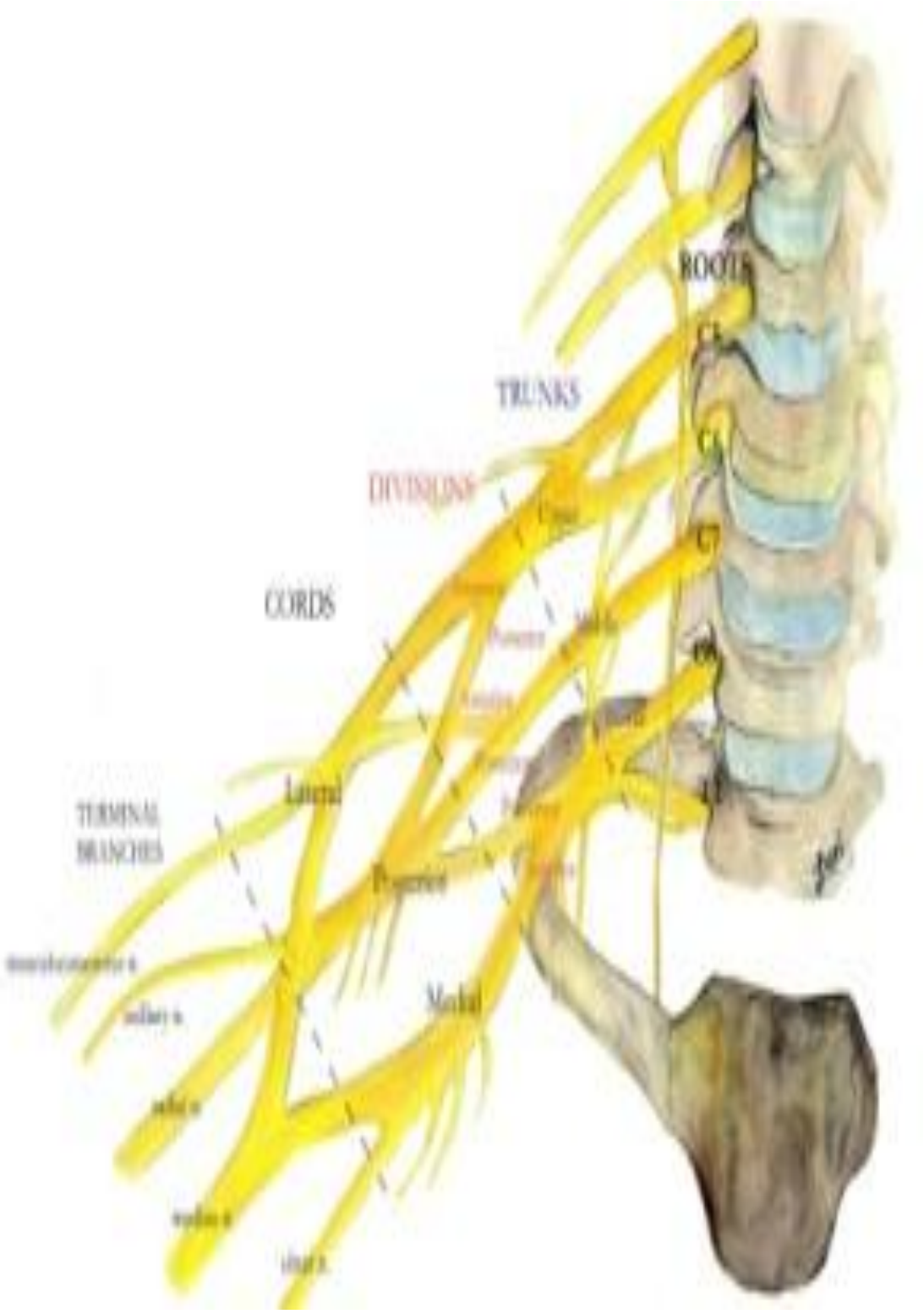
CHENNAI

PHOTOS & CHARTS

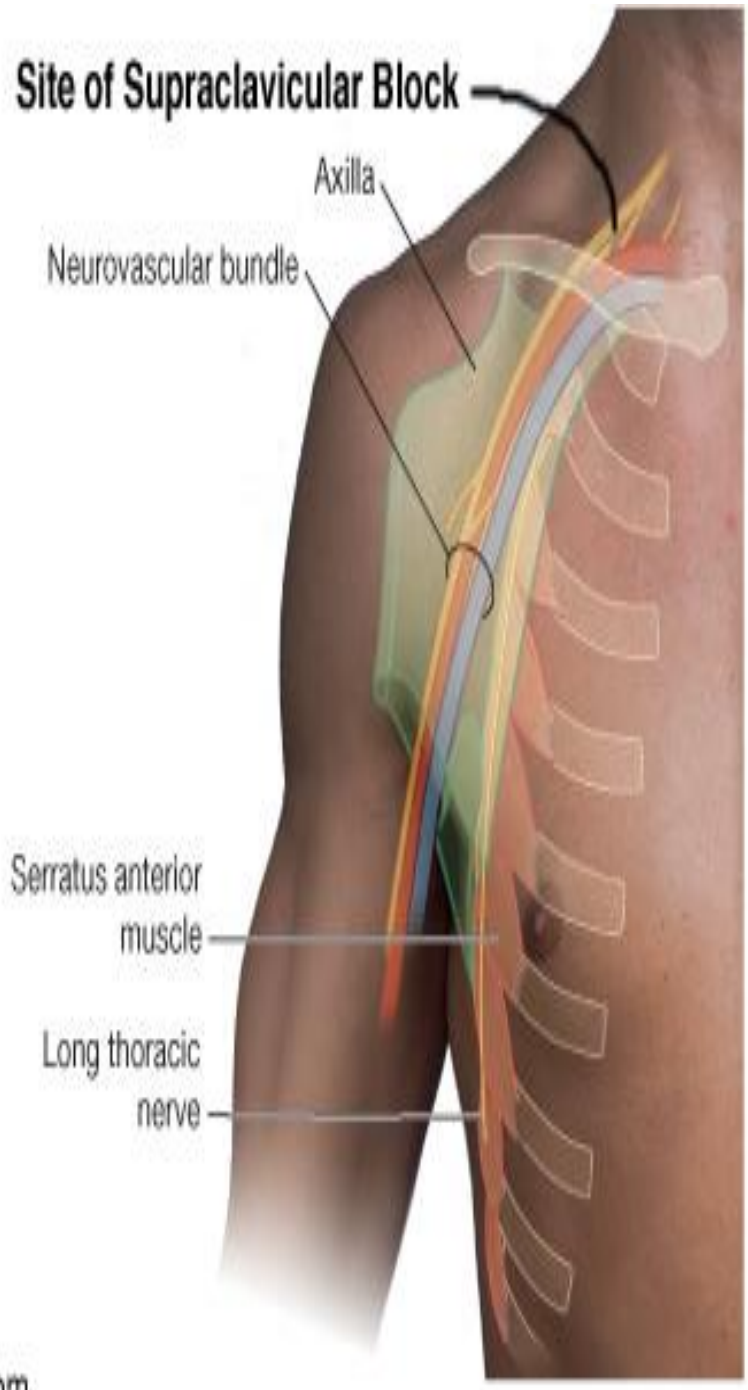
THE BRACHIAL PLEXUS AND ITS RELATION WITH SUBCLAVIAN VESSELS



THE BRACHIAL PLEXUS AND ITS RELATION WITH FIRST RIB

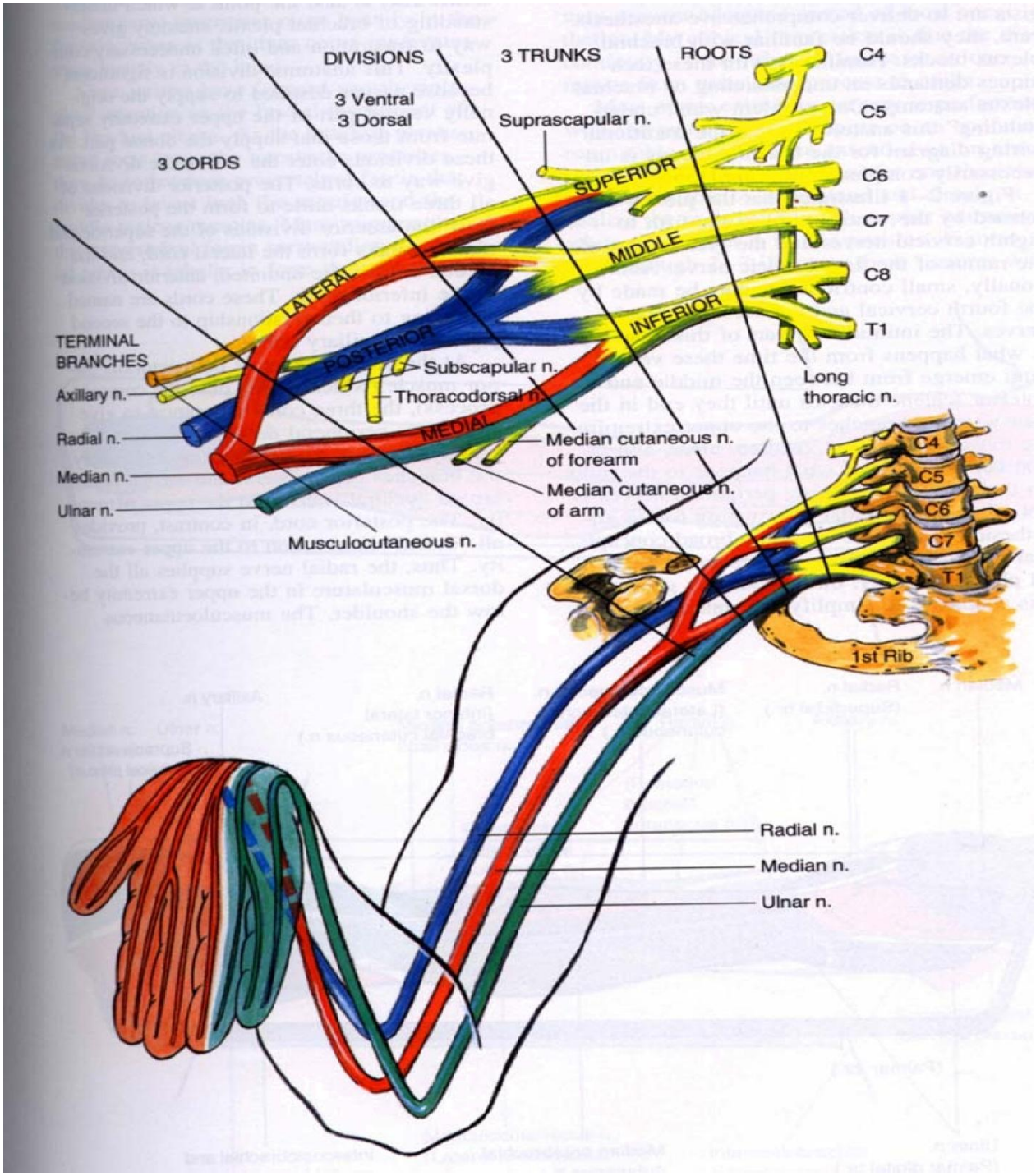


THE SITE OF SUPRACLAVICULAR BLOCK

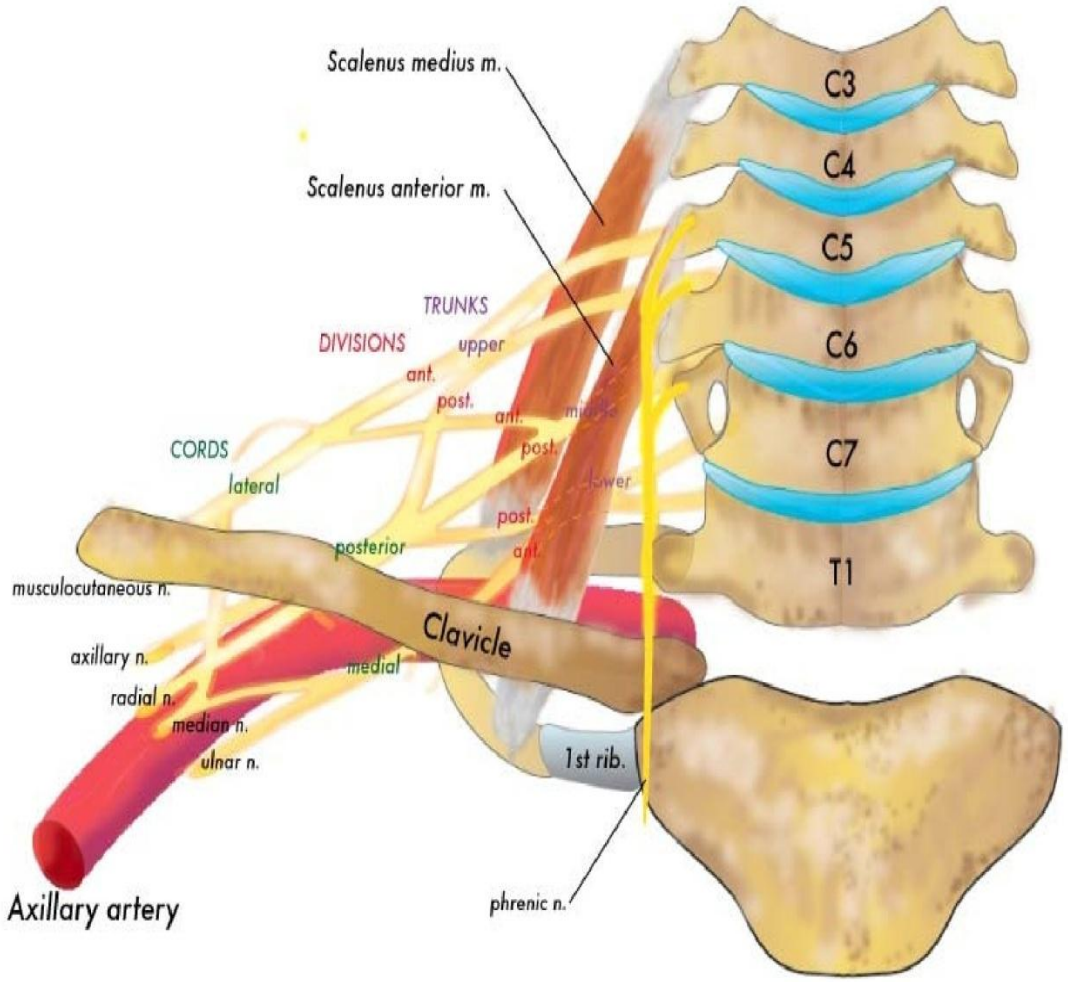


Modified from

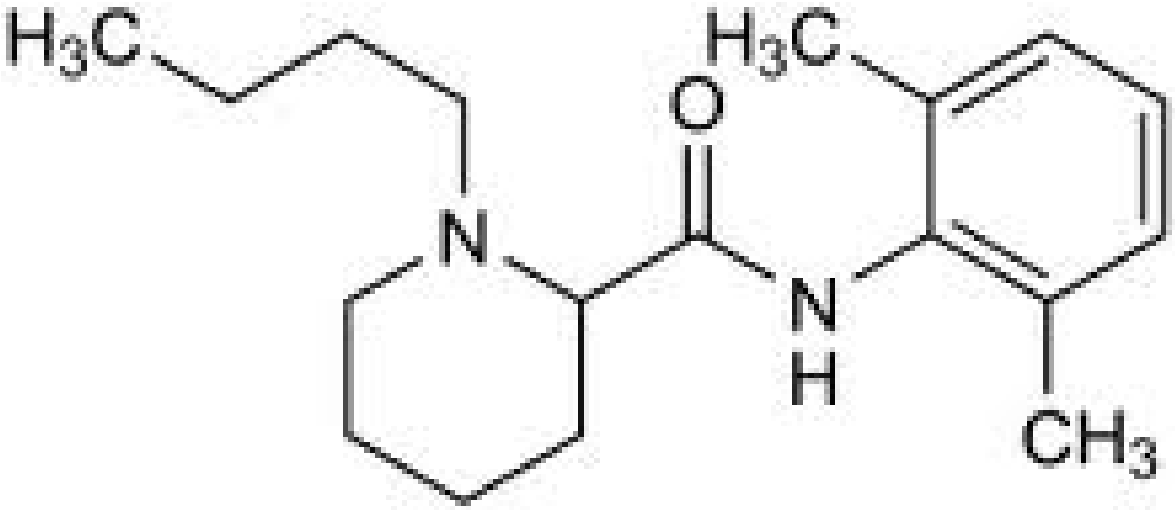
THE BRACHIAL PLEXUS AND ITS BRANCHES


DIVISIONS
3 TRUNKS
ROOTS
C4
3 Ventral
3 Dorsal
C5
Suprascapular n.
3 CORDS
SUPERIOR
C6
C7
MIDDLE
C8
LATERAL
INFERIOR
T1
TERMINAL
BRANCHES
POSTERIOR
Subscapular n.
Long
thoracic n.
Axillary n.
Thoracodorsal n.
MEDIAL
Radial n.
Median cutaneous n.
of forearm
Median n.
Median cutaneous n.
of arm
Ulnar n.
Musculocutaneous n.
C4
C5
C6
C7
T1
1st Rib
Radial n.
Median n.
Ulnar n.

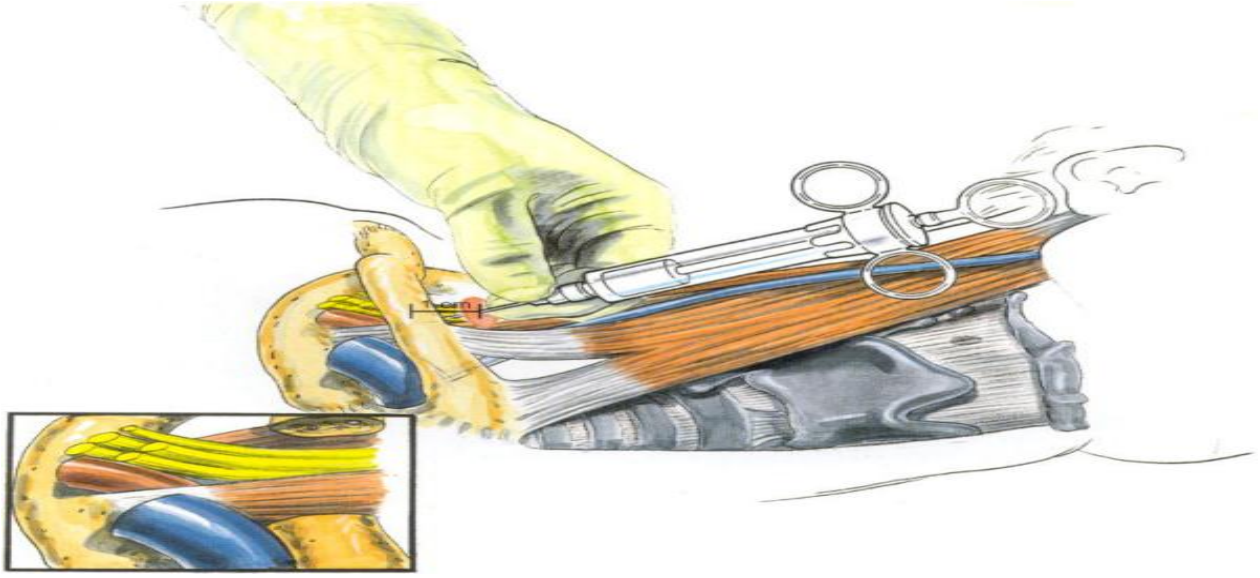
THE FORMATION OF BRACHIAL PLEXUS



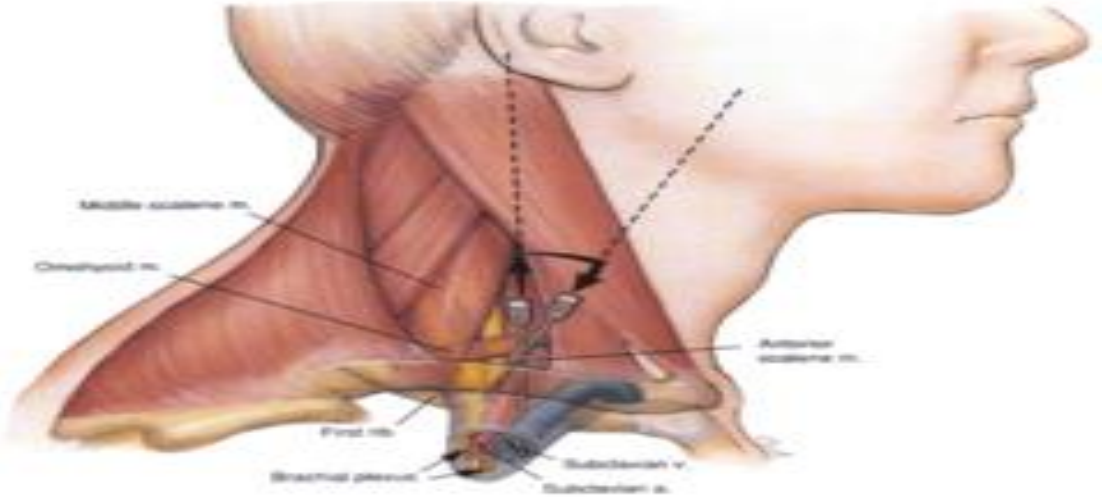
STRUCTURE OF BUPIVACAINE



WALKING THE NEEDLE TO ELICIT PARAESTHESIA

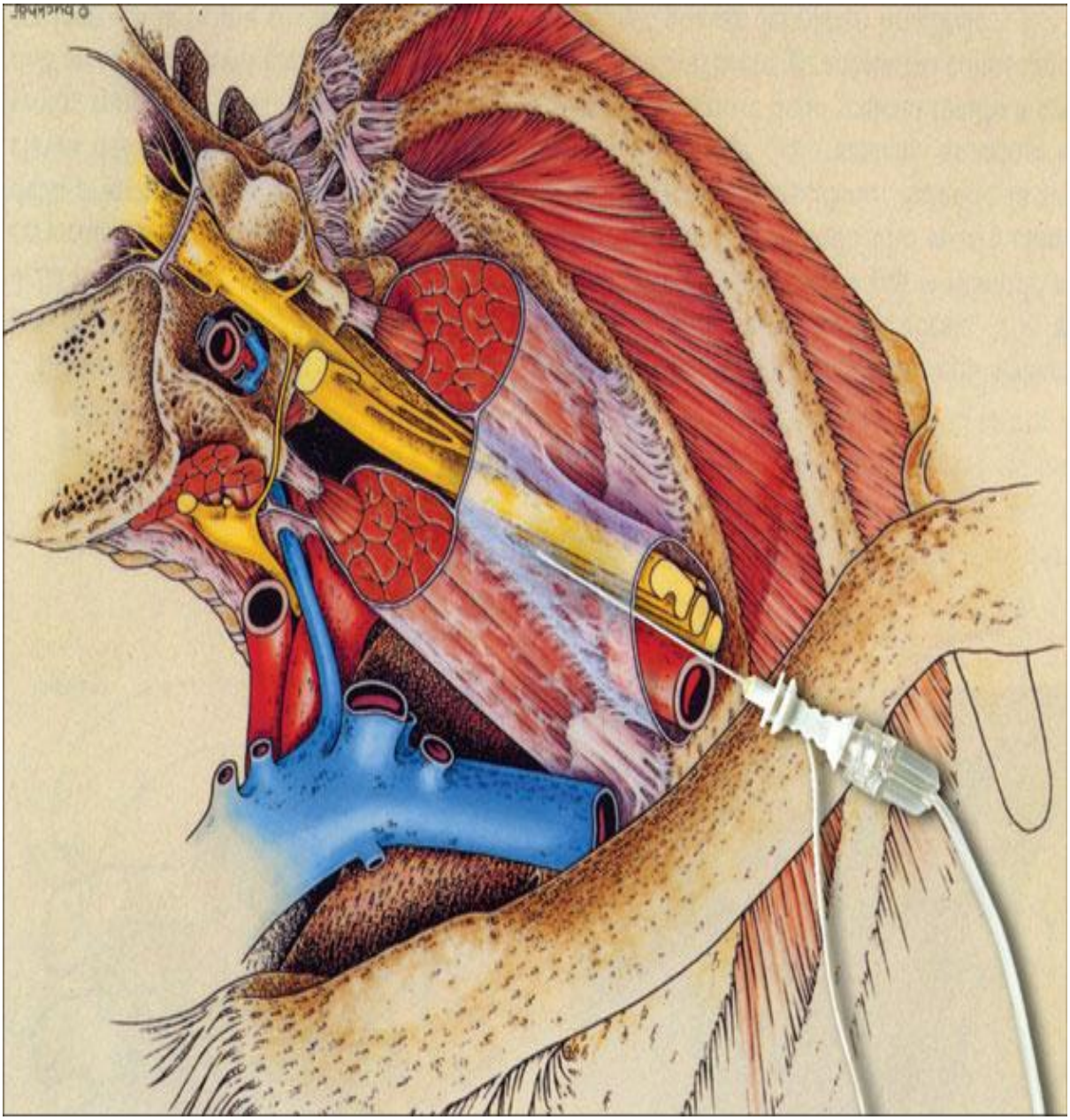


SUBCLAVIAN PERIVASCULAR APPROACH OF BRACHIAL PLEXUS BLOCK

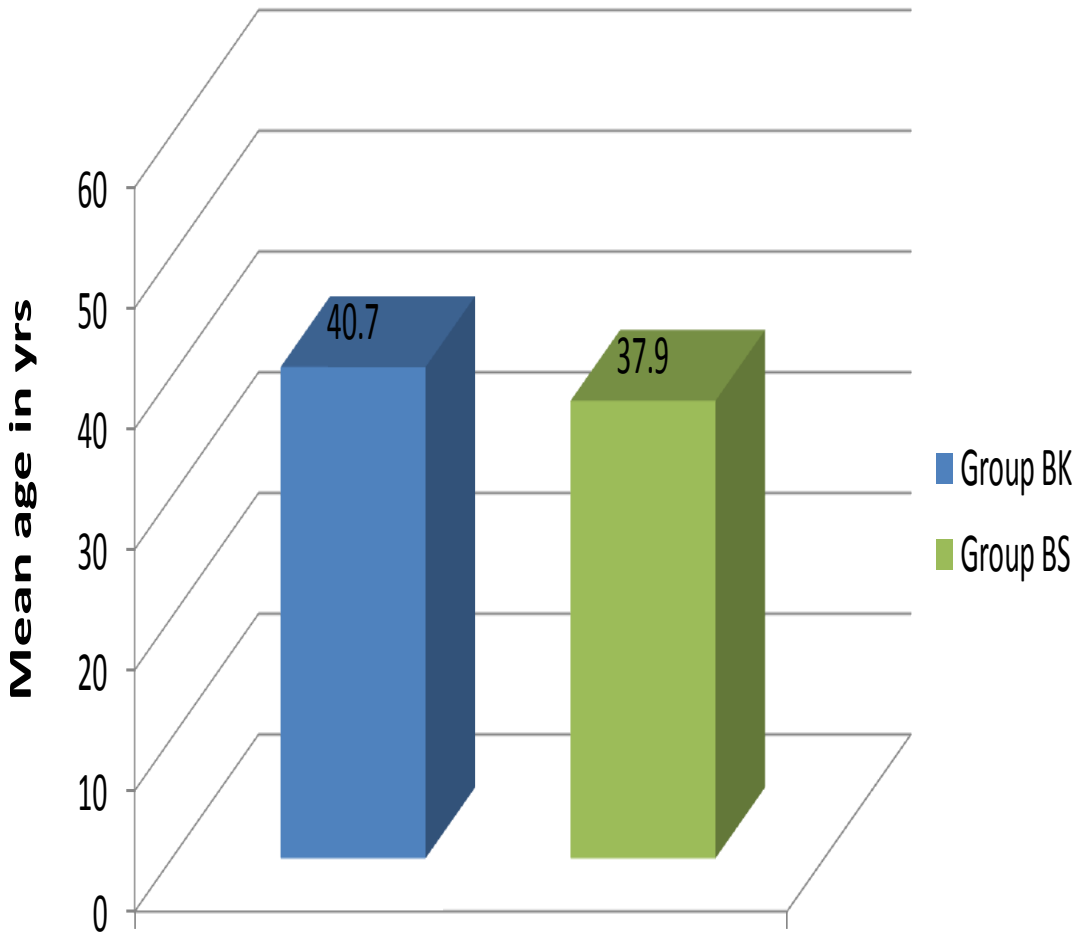


ALL THE THREE ROOTS OF BRACHIAL PLEXUS ARE

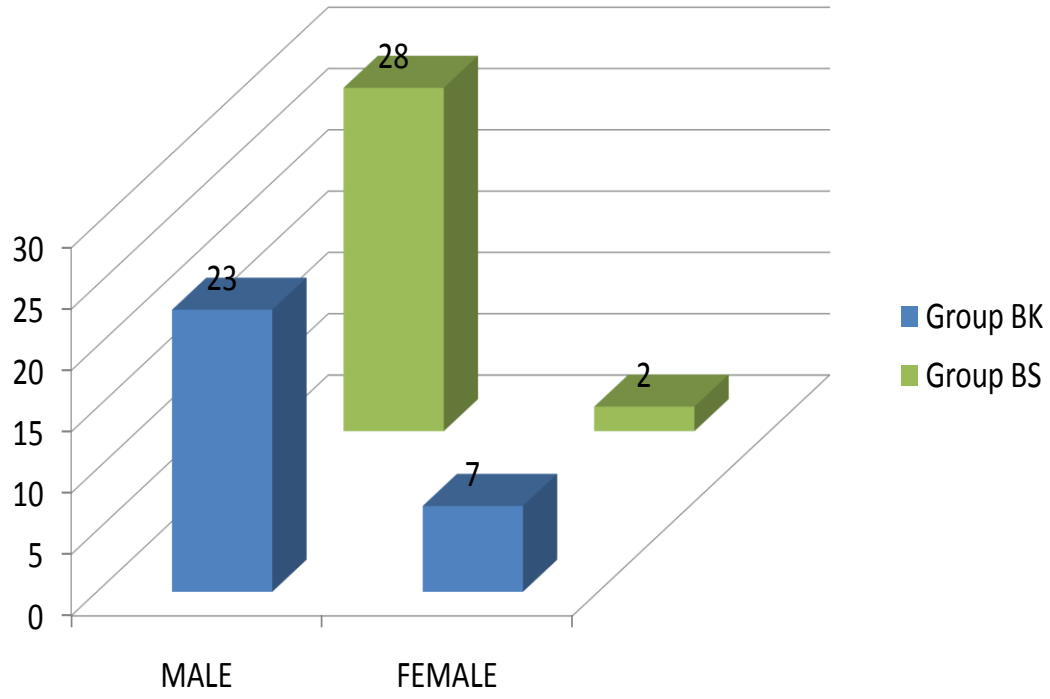
ENCLOSED IN A SINGLE SHEATH



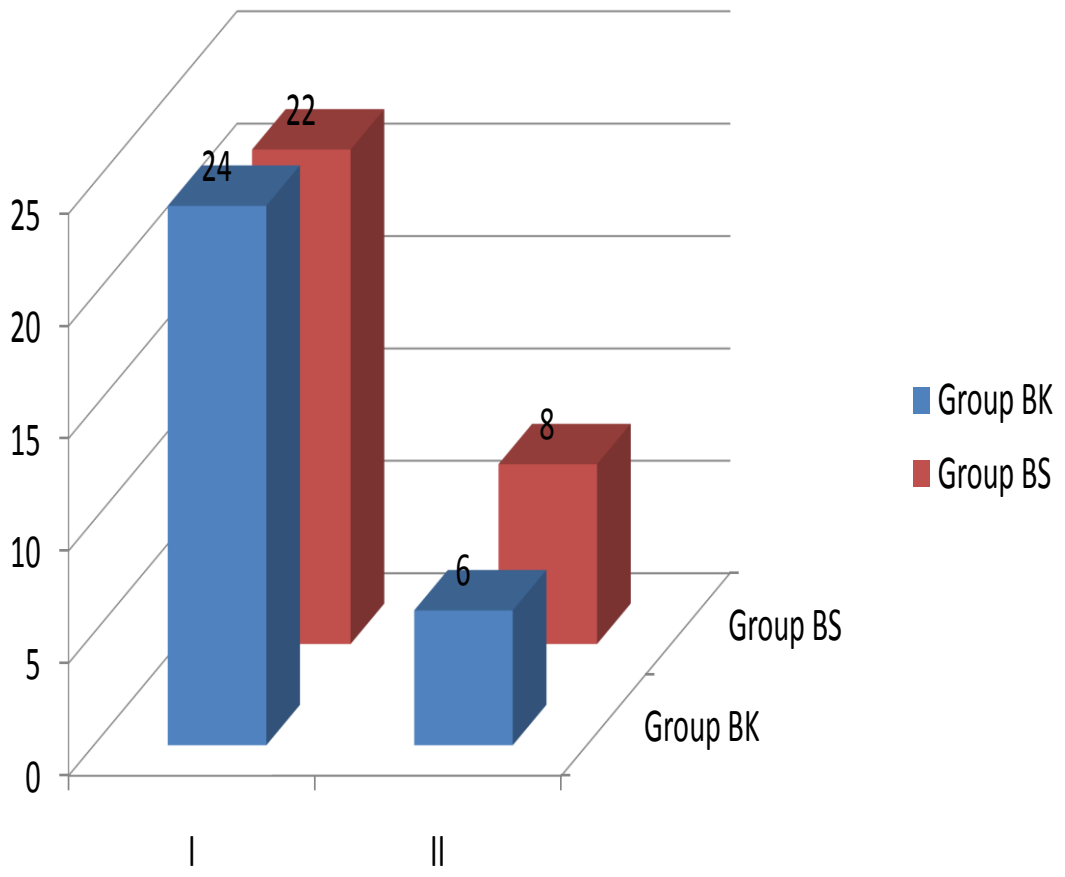
AGE



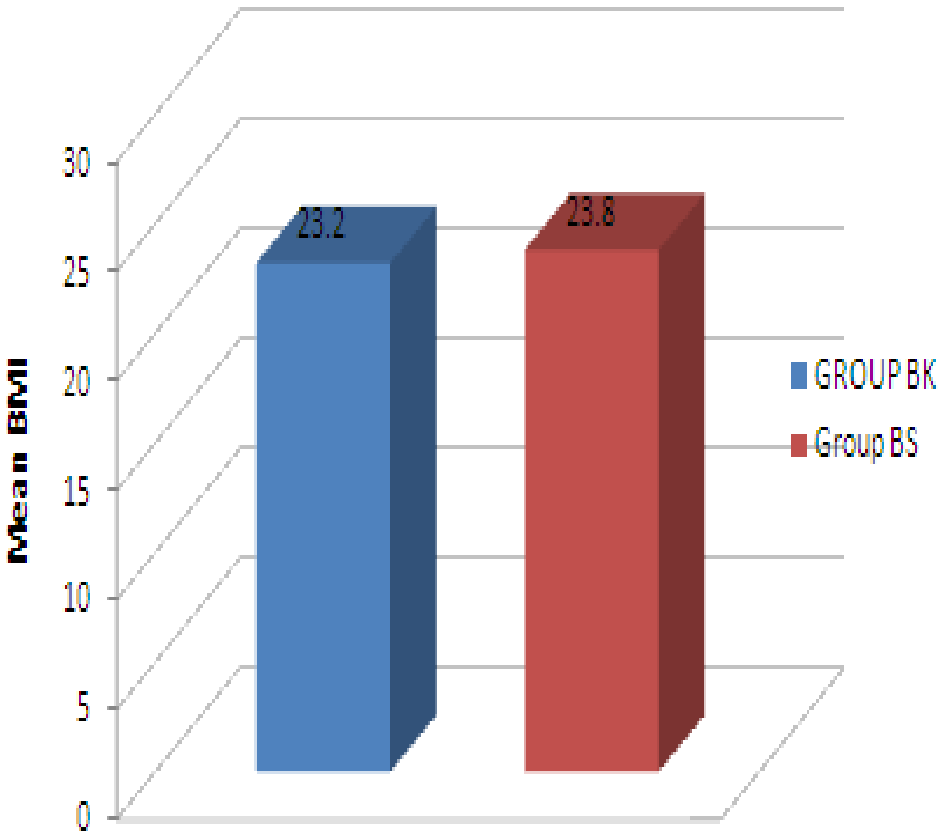
SEX



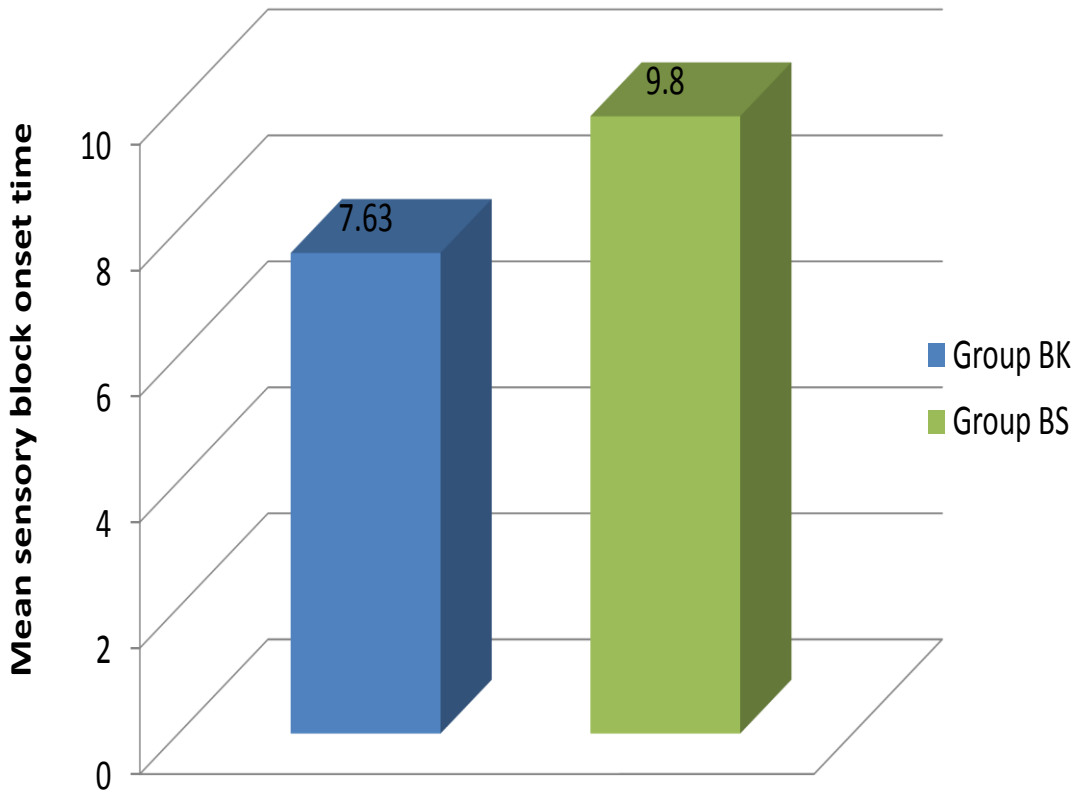
ASA



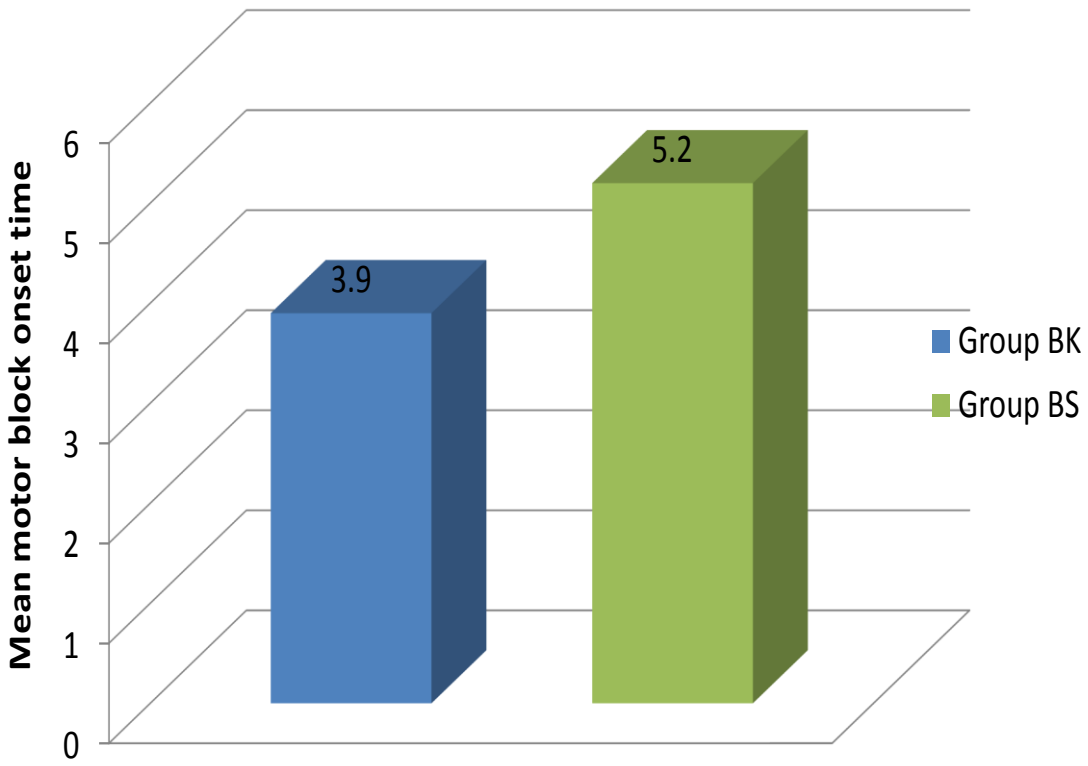
BODY MASS INDEX



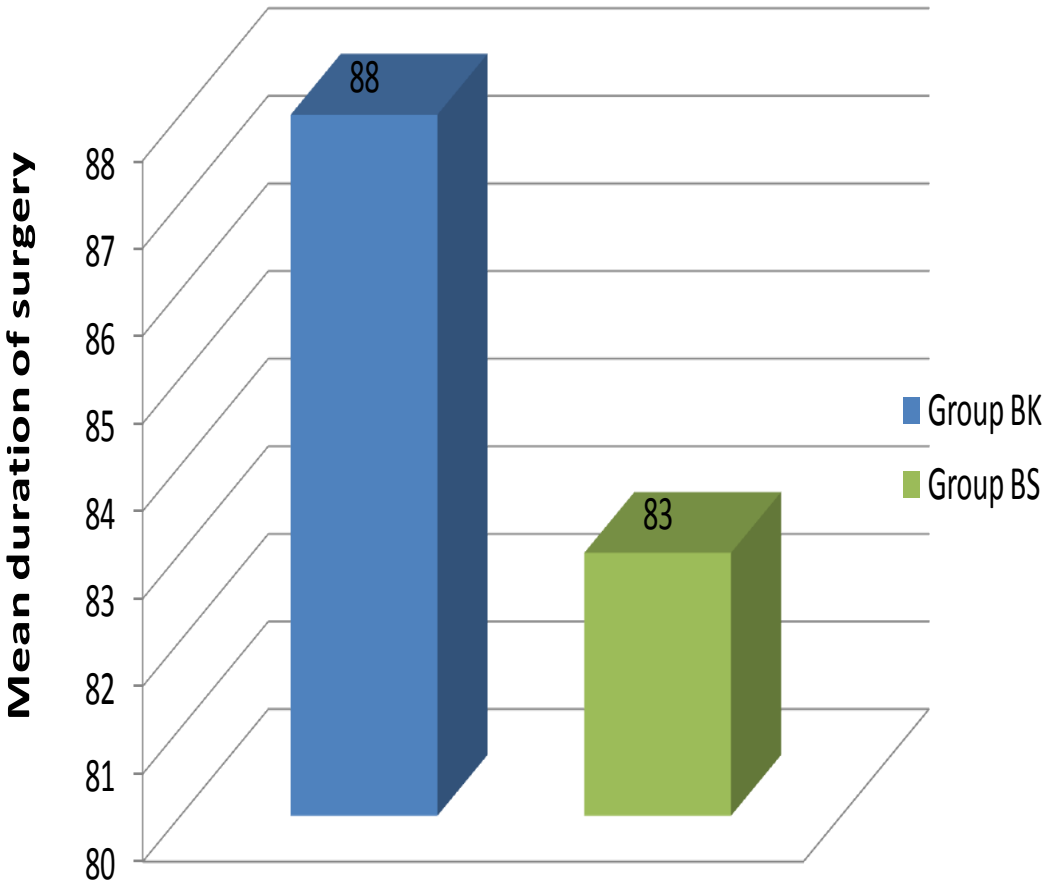
SENSORY BLOCK ONSET TIME



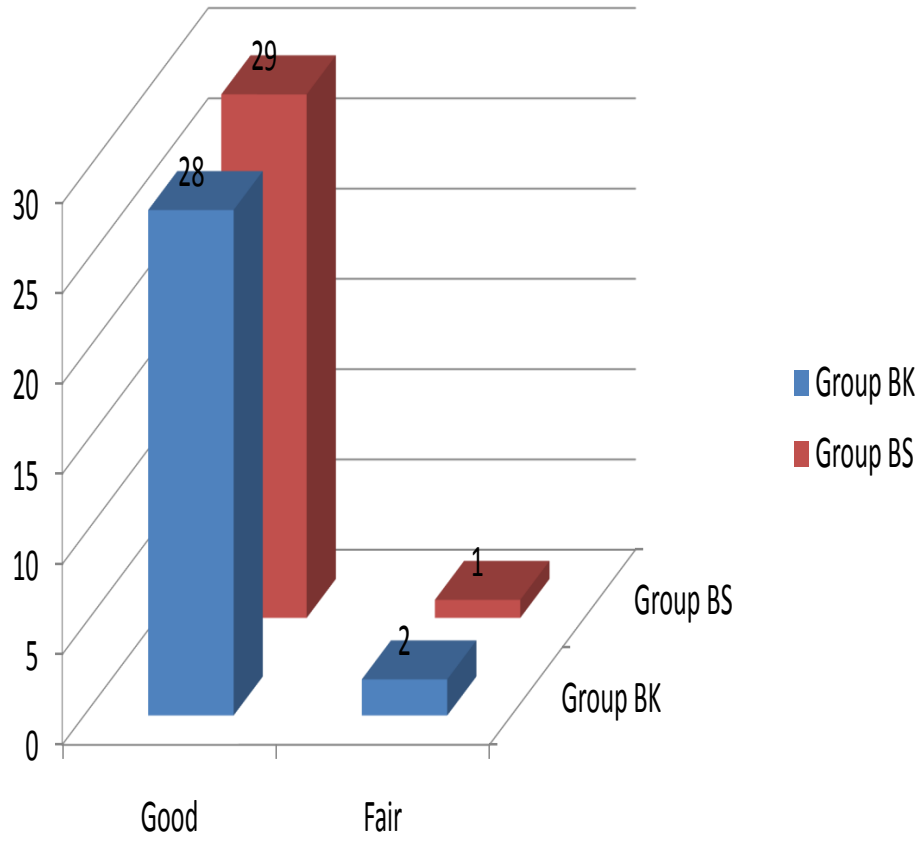
MOTOR BLOCK ONSET TIME



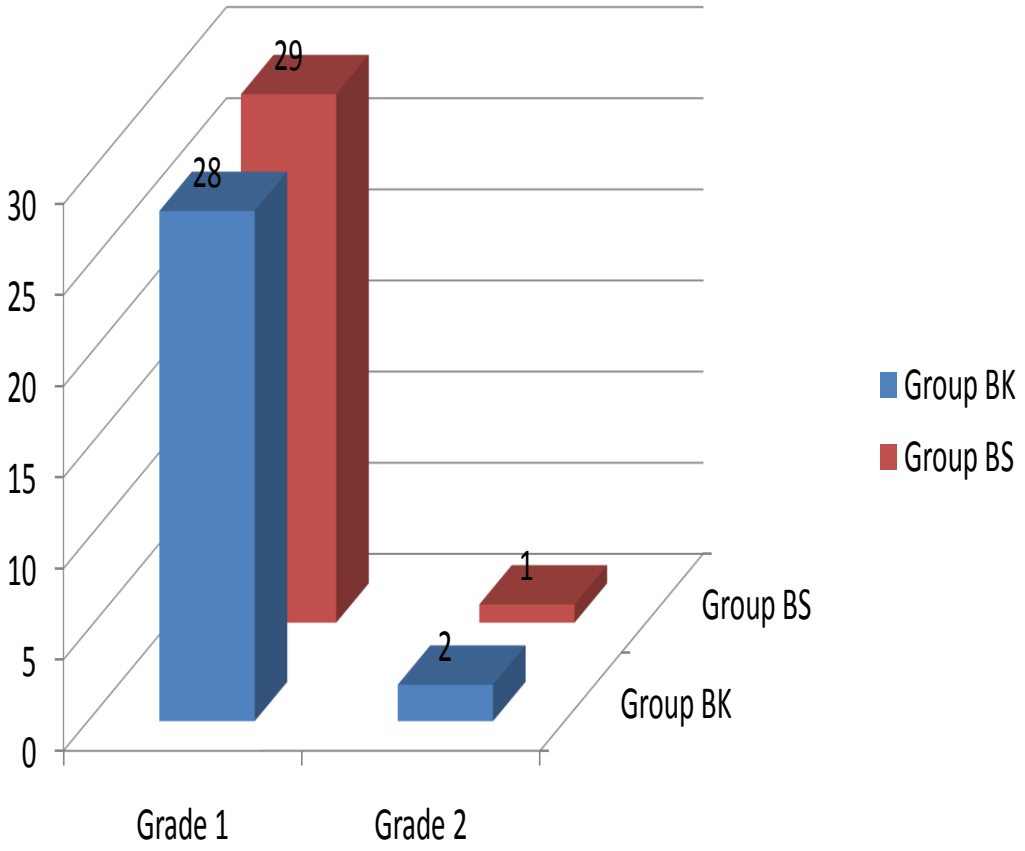
DURATION OF SURGERY



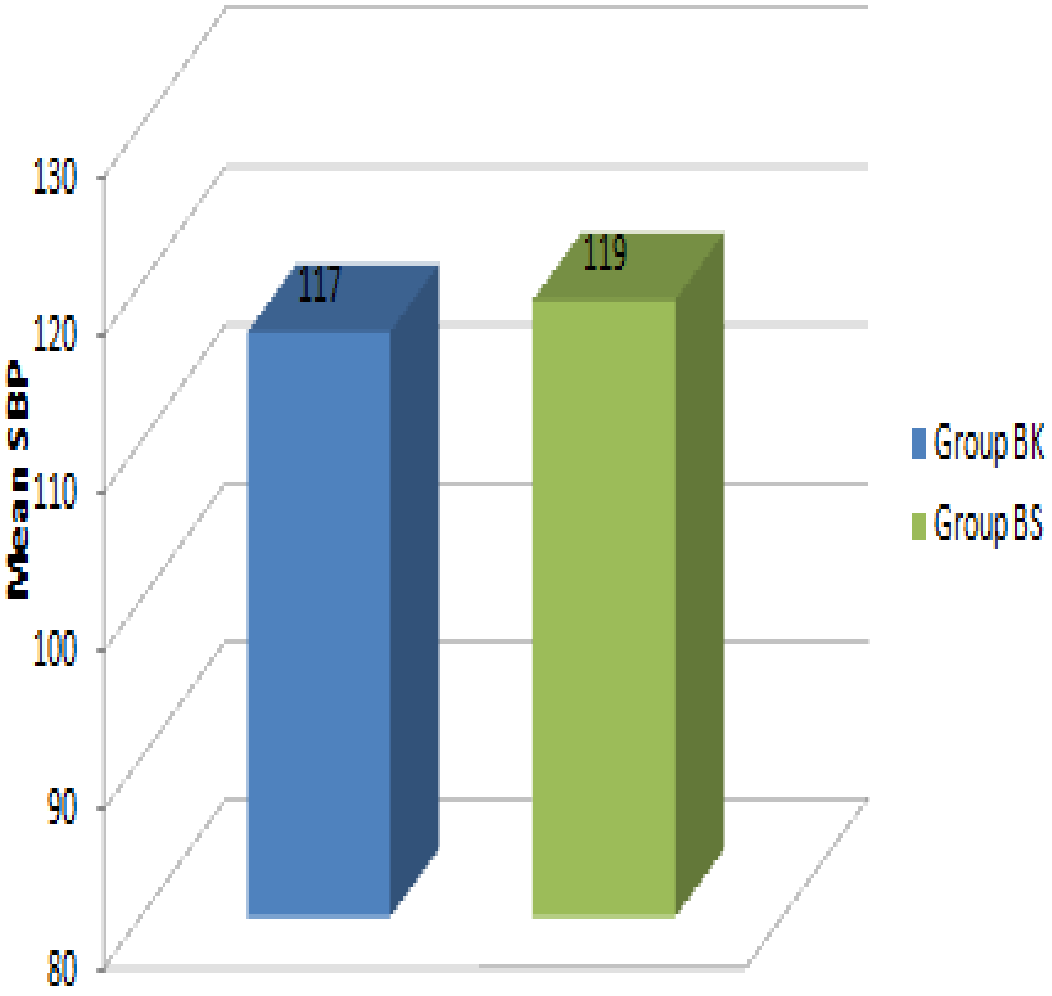
TOURNIQUET TOLERANCE



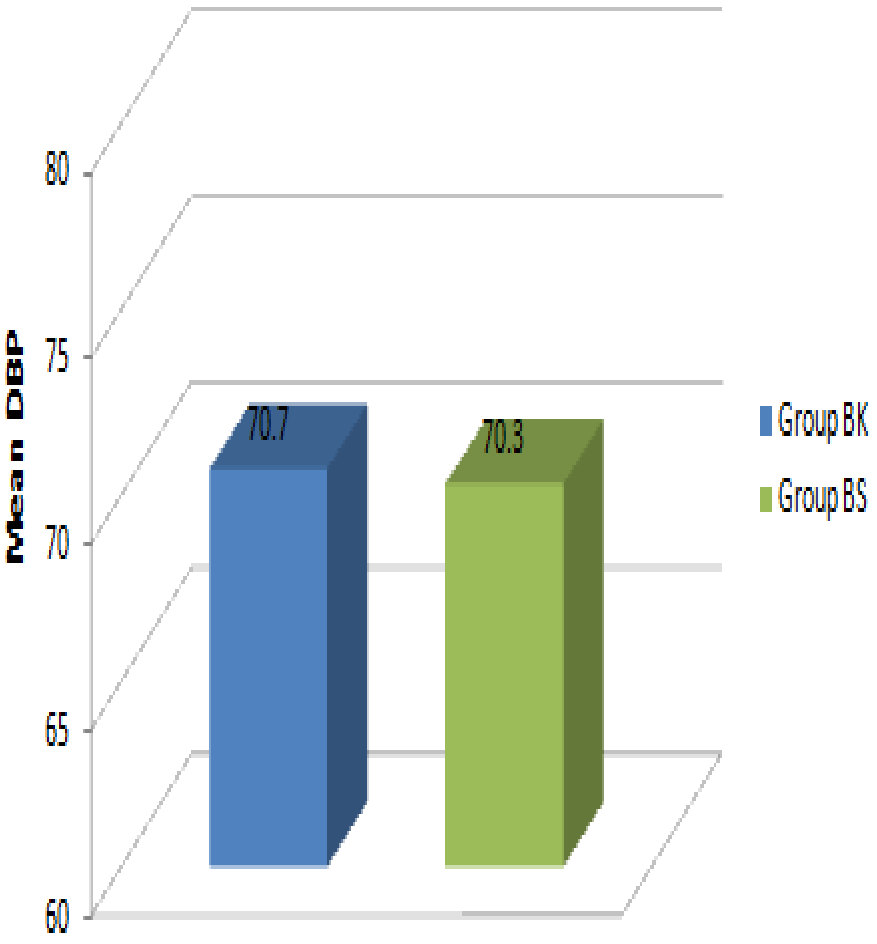
QUALITY OF ANAESTHESIA



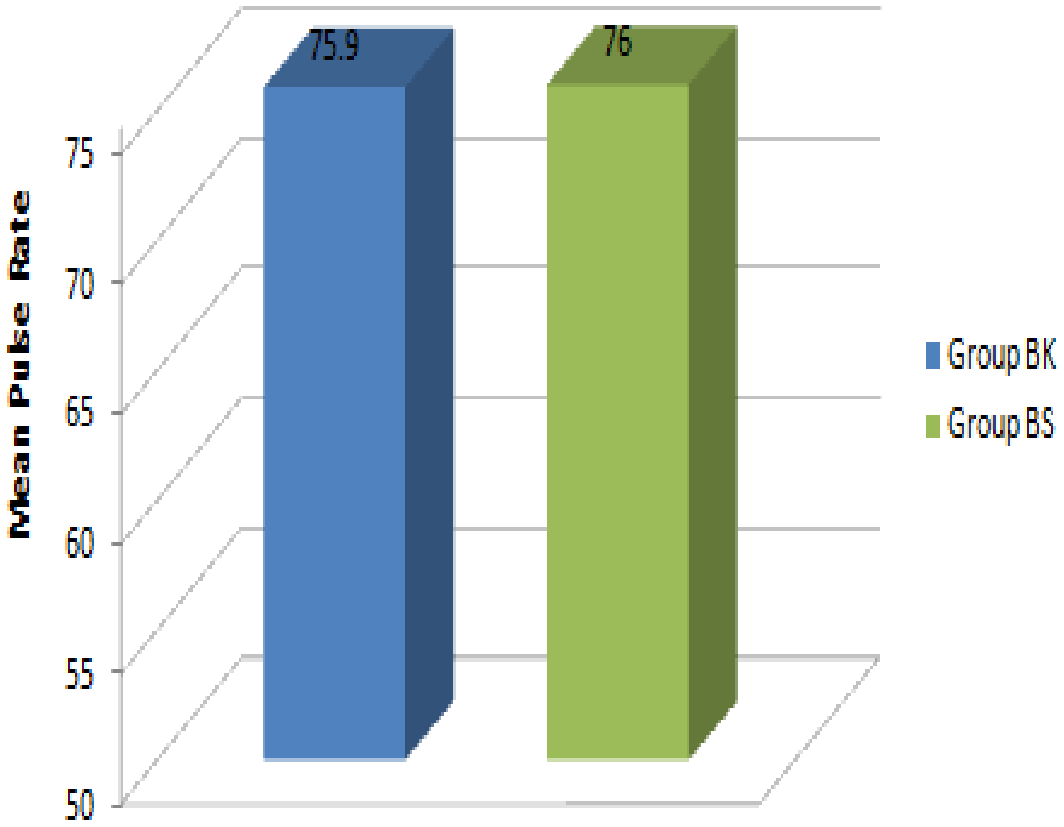
SYSTOLIC BLOOD PRESSURE



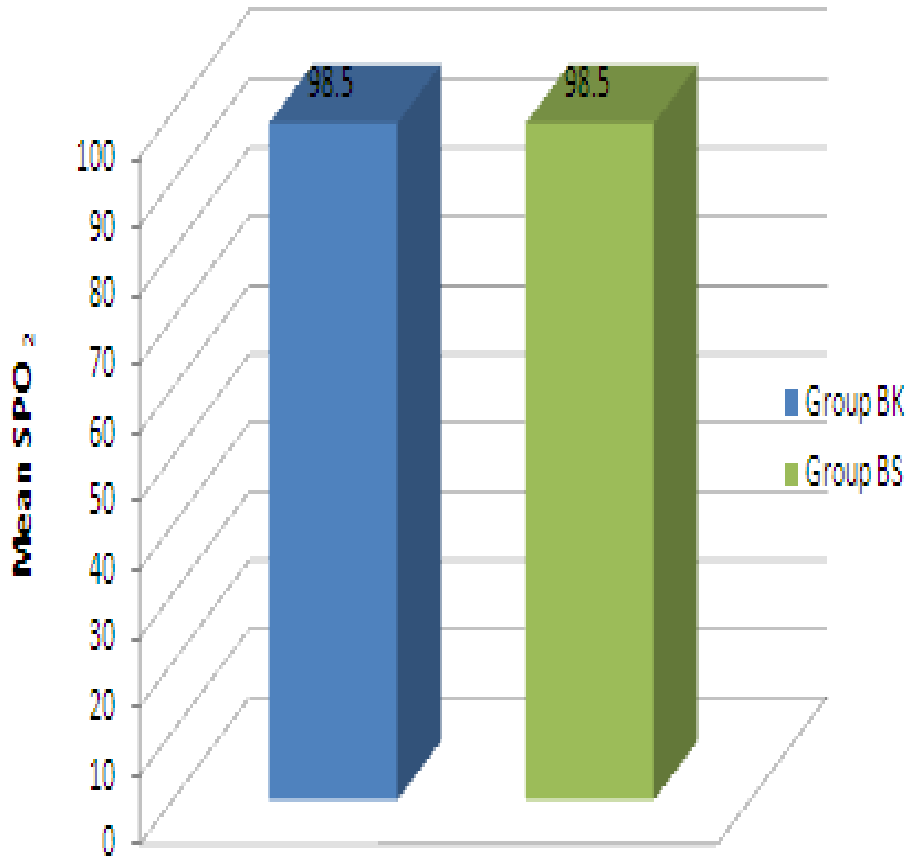
DIASTOLIC BLOOD PRESSURE



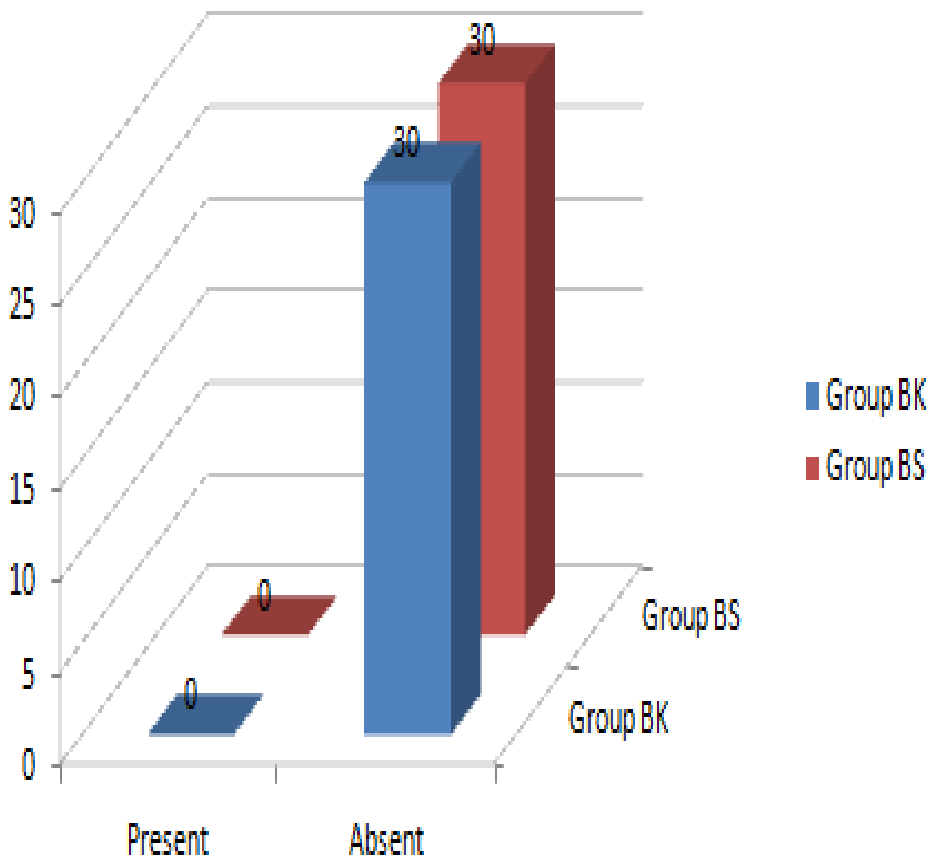
PULSE RATE



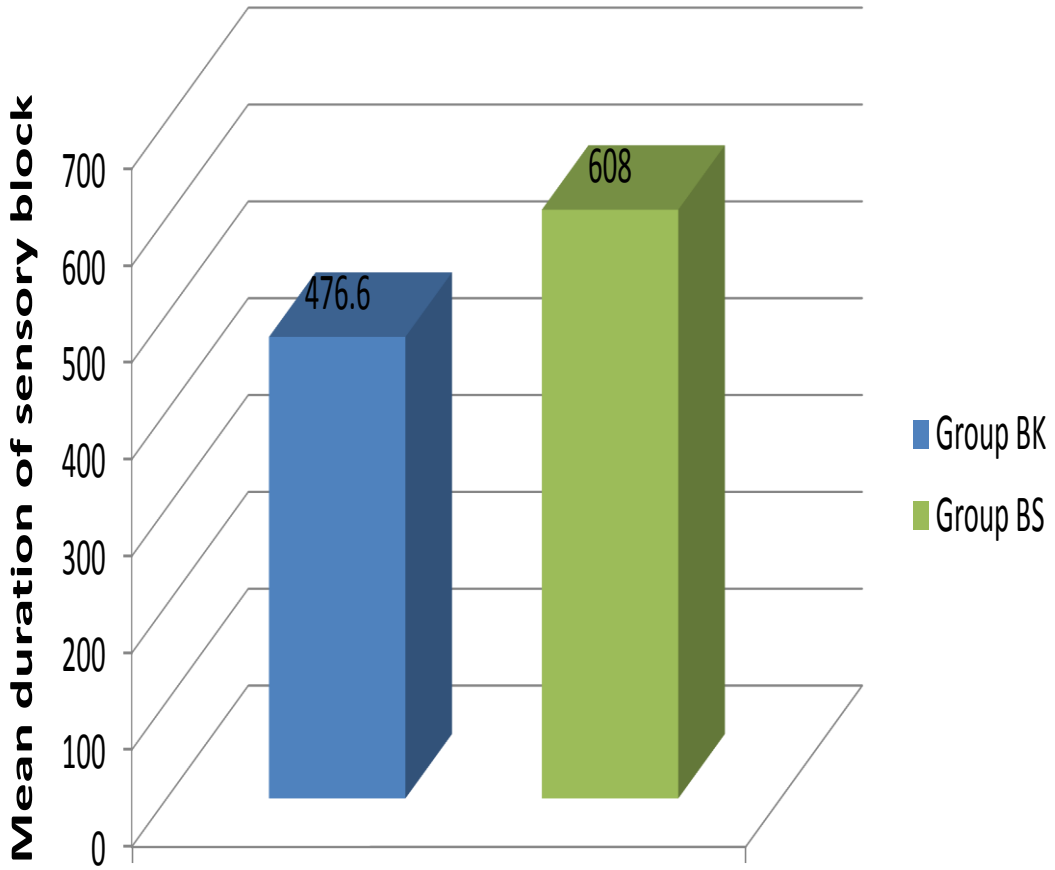
SPO_2


Mean SPO 2
100
90
80
70
60
50
40
30
20
10
0
98.5
98.5
Group BK
Group BS

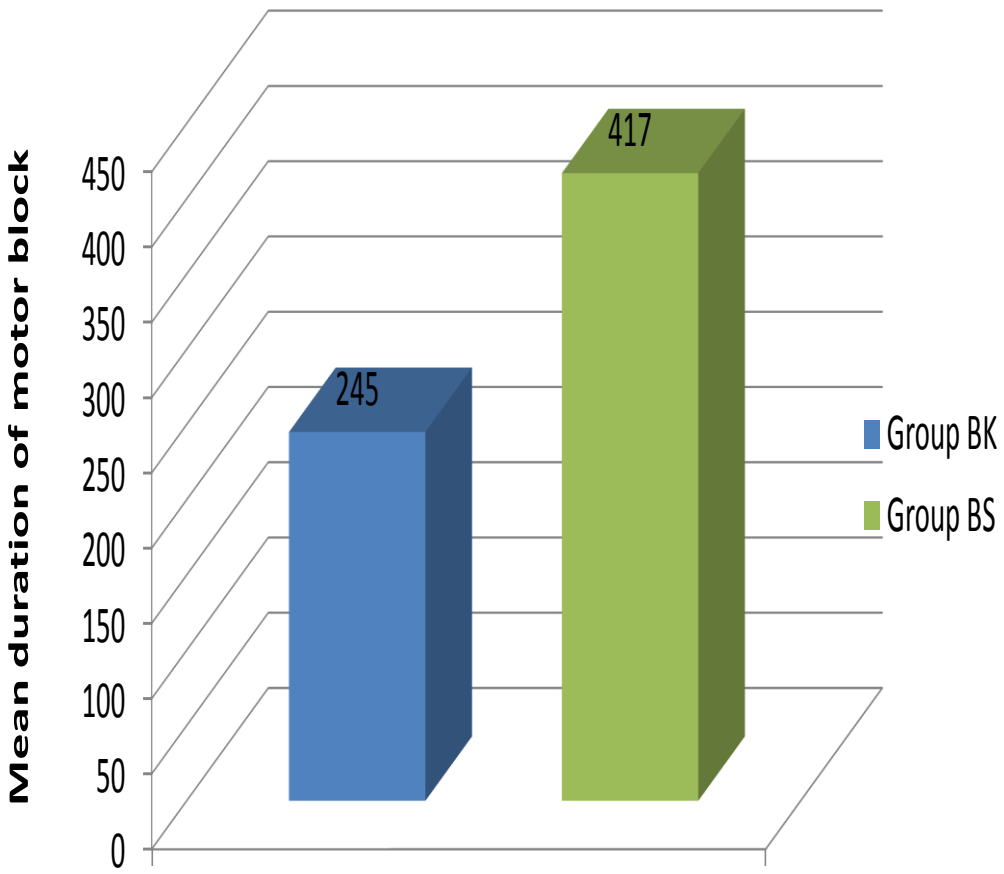
ECG CHANGES


30
30
0
0
30
25
20
15
10
5
0
Present
Absent
Group BS
Group BK
Group BK
Group BS

DURATION OF SENSORY BLOCK



DURATION OF MOTOR BLOCKADE



COMPLICATIONS