"COMPARISION BETWEEN LATERAL DECUBITUS POSITION AND CONVENTIONAL SUPINE POSITION TO DETERMINE THE DISTANCE BETWEEN PLEURA AND BRACHIAL PLEXUS IN PATIENTS UNDERGOING ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES:A RANDOMISED CONTROL TRIAL"

By

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Dissertation

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KLE Academy of Higher Education & Research
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BELAGAVI, KARNATAKA

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LIST OF ABBREVIATIONS

ASA- American Society of Anaesthesiologists

USG- Ultrasound Guidance

US - Ultrasound

GA- General Anaesthesia

BP-Brachial plexus

BPB- Brachial plexus block

SCB-Supraclavicular block

SCBPB-Supraclavicular Brachial plexus block

IP-In-plane

LA-Local Anaesthetic

LAST-Local Anaesthetic systemic toxicity

VAS- visual analogue score

mins- Minutes

Min-Minimum

Max-Maximum

kg-Kilogram

ml- milliliters

NBM-Nil by mouth

NIBP- Non invasive blood pressure

PR-Pulse Rate

SpO2-Saturation percentage of oxygen

Iv-Intravenous

HS-Highly significant

VS-Very significant

S- Significant

NS-Not Significant

mg-Milligram

MHz-Megahertz

RS-Respiratory system

CVS-Cardiovascular system

CNS-Central nervous system

GIT-Gastrointestinal Tract.

S.D.-Standard deviation

ABSTRACT

Title-"Comparison between Lateral Decubitus position and Conventional Supine position to determine the distance between Pleura and Brachial Plexus in patients undergoing Ultrasound Guided Supraclavicular Brachial Plexus Block for Upper limb surgeries: A Randomized Control Trial"

Background & Aims

In recent times, regional anaesthesia has gained prominence as an effective and safer alternative to general anaesthesia ,whenever feasible, aiming to avoid the necessity of latter. The supraclavicular brachial plexus block(SCBPB) has emerged as an unparallel method of providing anaesthesia and pain relief to patients receiving surgical procedure on their upper limbs. Even with precise ultrasound guidance, because of the brachial plexus's close proximity to the pleura during supraclavicular approach can lead to pneumothorax, one of the major adverse outcome. Hence, the study aims to compare the distance between pleura and brachial plexus in lateral decubitus and conventional supine positions.

Materials & Methods.

60 patients undergoing elective surgical procedures on the upper extremities, utilizing ultrasound -guided brachial plexus block via supraclavicular approach were included and randomly assigned to two groups using computer- based randomization: Supine position (Group S) and Lateral Decubitus position(Group L). Comparison between these

two groups were done in terms of the distance from apex of pleura to the brachial plexus, the duration needed to perform the blockade, the rate of successful blockade and complications intra operatively and postoperatively.

Results

In the present study findings , a significant increase in the distance from the pleural apex to the lower trunk of the brachial plexus was noted in "Group L" in contrast to "Group S". In "Group L", the time taken to perform the procedure was shorter compared to "Group S". Both the groups had equal success rate of the block. No intra operative complications occurred in either of the groups.

Conclusion

The distance between pleura and brachial plexus was more and block performance time was less in Lateral decubitus position when compared to the conventional supine position. Therefore , recommended the lateral decubitus positional approach for administering supraclavicular brachial plexus block over conventional supine positional approach, as it has the potential to enhance safety while mirroring the success rate of the conventional supine position.

Keywords

Lateral Decubitus position, Supraclavicular approach of brachial plexus block, Ultrasound.

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INTRODUCTION

Brachial plexus block ,one of the common regional anesthesic procedure , utilized either as a substitute for or in conjunction with general anesthesia for surgeries on the upper limb. It is considered as a safe option to provide anaesthesia and analgesia. The benefit of this approach is that the patient remains conscious, aware, and retains normal airway reflexes throughout the procedure.[1]The most commonly used approaches for upper limb surgical procedures are supra-clavicular , Infra-clavicular and Axillary brachial plexus blocks.

Although there are numerous approaches of administering brachial block, supraclavicular approach stands out as the most effective in delivering dependable anaesthesia for upper limb procedures. It provides reliable anaesthesia to the entire arm. It is suitable for surgeries on the upper limb, which includes surgical procedures involving elbow, fore arm, and hand. This blockade takes place at the level where distal trunk meets the proximal division of the brachial plexus.

One important advantage of the supraclavicular block is the dense clustering of nerves in this area, which leads to a rapid and profound block. This characteristic has led to its comparison as “The Spinal anaesthesia of the Arm”.

The supraclavicular method for accessing the brachial plexus typically results in association with the quicker onset of anaesthesia and a high rate of success . The Supra-clavicular method is easier as it is more superficial block.

Despite the effectiveness of ultrasound- guided SCBPB in improving safety,the occurrence of adverse outcomes such as pneumothorax has been documented at a rate

ranging from 0.5 to 5% when administering supraclavicular brachial plexus block in the convenctional supine position.[2]

As the lung's apex is positioned behind the 1st rib , medially and posteriorly to the brachial plexus the likelihood of getting pneumothorax is maximum compared to other approaches. Other potential adverse outcomes includes blockade of phrenic nerve , Horner's syndrome and neuropathy.

The force exerted on the brachial plexus by traction is primarly influenced by the patient's posture. Elements such as spine retroflexion, and humerus rotation can alter this tension.[3]Therefore, modifying body positions can increase the margin of safety and reduce complications, like pneumothorax ,that may occur during US guided SCBPB.[3]

Literature search did not yield any studies comparing between lateral decubitus position and conventional supine position to evaluate the safety of US guided SCBPB in surgeries concerning the upper extremities.

Hence, here is an attempt to compare the lateral decubitus and conventional supine positions to assess "the distance between the pleura and the inferior trunk of brachial plexus" as a surrogate for the safety during supraclavicular brachial plexus block.

OBJECTIVES

PRIMARY OBJECTIVE:

To compare the distance between pleura and brachial plexus in lateral decubitus and conventional supine positions.

SECONDARY OBJECTIVE:

To compare block performance time, success rate of the block.

To study any other intra operative and postoperative complications, in patients scheduled for upper limb procedures, under supraclavicular brachial plexus block (SCBPB).

REVIEW OF LITERATURE

The brachial plexus, an intricate nerve network arising from the anterior-rami of spinal nerves C5 to T1, serves to innervate the upper limb, governing both motor and sensory functions. Its condensed structure makes it, an ideal target for achieving comprehensive anesthesia during upper limb procedures,which has become a regular practice.[4]The prevalence of problems, like pneumothorax and unintentional vascular injury, has led to an increase in the use of ultrasound guided BPBs as an anaesthetic approach for upper limb surgery.

Among the different techniques available, the supraclavicular approach stands out for its accessibility, enabling the insertion of a needle to administer local anesthetic near the plexus as it courses over the clavicle.

Recent studies have explored the impact of patient positioning on the safety and effectiveness of the block. Notably, research comparing the lateral decubitus position to the conventional supine position has shown promising results. The lateral position has been associated with a greater distance between the pleura and brachial plexus, thereby reducing the risk of pneumothorax. Additionally, this position has been linked to shorter block performance times and potentially fewer complications.

This literature review encompasses various prospective studies and randomized controlled trials that highlight the comparative benefits of the lateral decubitus position

over the conventional supine position. The review synthesizes these findings to provide a comprehensive understanding of the current best practices in brachial plexus blocks, emphasizing the importance of patient positioning in optimizing both safety and effectiveness. By doing so, it aims to guide clinical practice and inform future research directions in regional anesthesia.

In this prospective study conducted by Carl PC Chen et al. in 2020 they compared distance from plexus and pleura in three different body positions i.e., supine, sideways and lateral and found that the the distance in lateral position was almost twice that compared to supine position. There was no significant difference in comparision with sideways position i.e., 45^{o}. They also examined the correlation between body-mass index & the measured distances in those postures mentioned above.[3]

In this randomized prospective study, conducted by Anjana sahu et al., in 2019 concluded based on the analysis of 64 patients, it was determined that the lateral approach yielded significantly better outcomes in terms of blockade adequacy, tolerance to tourniquet, and lower risk of complications. Therefore, it can be inferred that the lateral approach appears to be the preferred method for supraclavicular block compared to the conventional classical approach. Moreover, in alternative circumstances such as patients with burns, contractures, or infections at the injection site of the classical approach, the lateral approach, featuring a distinct injection site, could serve as a practical alternative.[1]

In this prospective randomized comparitive study, conducted by Prasad PK et al., in 2014 among 50 patients , were inferred that supra clavicular approach of BPB in lateral

position with peripheral nerve stimulator presents as superior choice over the conventional supine positin because higher success rate and reduced complications.[5]

In this prospective nonrandomized, open level study carried out by Anjana Sahu et al. in 2010 among 82 patients was determined that the "supra clavicular approach of brachial plexus block" done in lateral position is linked to fewer adverse effects compared to other supraclavicular approaches and demonstrates greater effectiveness with a higher success rate as well.[6]

In this prospective,randomised and blinded study,conducted by Kumar A et al., in 2012 among 100 patients,inferred that the "lateral approach for SCBPB" represents a novel method that warrants routine clinical application due to its superior performance "in terms of onset, duration, extent, & success rate of block". Additionally, this approach is less invasive with fewer adverse events such as injury of vasculature and pleural cavity compared to the Macintosh technique.[7]

In this prospective observational study conducted by Dr.Dilip kothari in 2003 among 250 patients showed that the lateral approach for SCBPB is both safe and effective, exhibiting a higher success rate.[8]

In this case report given by Bacha Q.U et al. in 2015 has been concluded that in instances where pleuritic pain emerges during the administration of a brachial plexus block, one should maintain a high level of suspicion for pneumothorax, even immediately following the procedure. Furthermore, opting for ultrasound-guided techniques over the

conventional "Classical" approach to brachial plexus blocks is strongly advised due to its significantly lower complication rate.[9]

In another prospective double blind study conducted by Chun Woo Yang et al., among 100 patients in 2010 year their conclusion was that there was no notable distinction in clinical efficacy between the supraclavicular and infraclavicular approaches. However, it was noted that the supraclavicular block resulted in pneumothorax.[10] Due to the incidence of complications like pneumothorax and inadvertent vascular complications , Ultrasound guided BPBs are increasingly being used as an anaesthestic technique for the upper limb surgery.[10]

This case report given by Kumari A et al. in 2011 concluded that in cases of pleural puncture or pleuritic pain, relying solely on supine chest X-rays is not sufficient. Instead, utilizing on-table portable ultrasound as a complementary tool could aid in diagnosing even minor pneumothorax. Additionally, they recommend conducting serial chest X-rays over three days to monitor for the possibility of latent pneumothorax following a brachial plexus block.[11]

In this cross sectional conducted by Yadav N et al. in 2016 among 103 volunteers,their conclusion emphasizes the importance of prescanning the supraclavicular region to know the corner pocket depth of plexus before selecting an corresponding needle size. Additionally, they recommend limiting the advancement of the needle to no more than the anticipated depth of the corner pocket.[12]

In this prospective observational study carried out by Gauss, A et al. in 2014 concluded that the. reduction of the incidence of complications of periclavicular blocks is by due to performing under ultrasound guidance.[13]

In this prospective randomized study conducted by Honnannavar KA, Mudakanagoudar MS in 2017 among 60 patients drawn a conclusion that ultrasound guidance represents a safe and effective approach for performing the SCBPB. Complication rates are reduced with the use of ultrasound provides the visualization of underlying structures and the real time distribution of the spread of anesthetic drugs,enhancing precision during administration.[14]

The case report conducted by Abell DJ et al. in 2014 estimated the incidence of pneumothorax to be 0.4 per 1000 following ultrasound-guided supraclavicular block and proposed targeted training methods for needle visualization to mitigate this risk.[15]

In 2017 a systemic review of RCTs was conducted by Park et al.[16] where they compared supraclavicular and infraclavicular block after assessing ten RCT's involving 676 patients. They observed that there was no variance in the occurrence of successful blocks,time taken for performing block,theonset and total duration of block. They further analyzed that procedure related paresthesia i.e. Horner's syndrome,diaphragmatic paresis were greater in Supraclavicular block when compared to Infraclavicular block.

A prospective double blinded study, done by Chun Woo Yang et al.,[17] in 2010 compared "the efficacy of supraclavicular BPB and Infraclavicular BPB using

neurostimulation”among 100 patients and They noticed that there was no notable distinction in the duration taken to execute the block, onset,depth,success and duration of blockade. Horner’s syndrome was more frequent in supraclavicular block compared to Infraclavicular block. There was incidence of pneumothorax in supraclavicular approach.

In 2021, in a Randomised control trial conducted by ParvatiSreelal et al.[18] in 60 patients undergoing procedures for forearm under Supra-clavicular or Infra-clavicular brachial plexus block, compared the time to perform anaesthetic blockade, duration of block and associated problems such as Horner’s syndrome, injury to vascular structures and pneumothorax. There was greater incidence of horner’s syndrome in SCB group than ICB group and puncture of subclavian artery was observed in one case in supraclavicular block group which was statistically insignificant.

In study , conducted by Mustafa Tayfun Aldemir et al.[19] in 2018 on “Comparison of Infraclavicular and Supraclavicular brachial plexus block in upper extremity surgeries”among 50 patients.They utilized a nerve stimulator to perform blocks and following parameters i.e., time to perform anaesthetic blockade , depth of needle, the duration for the block to take effect, duration of sensory block , motor block, time for initial complain of pain. This study concluded that while there were no notable statistical differences in the "duration of motor and sensory block or the onset of postoperative pain", the supraclavicular block exhibited significantly shorter times for performing the block, needle depth, and block onset, so they inferred that supraclavicular block is superior to infraclavicular block.16

In 2002, Sandhu et al.[20] had performed Infra clavicular BPB in 126 patients, they noticed that 114 blocks were successful without any further anaesthetic requirement or the need to switch to general anaesthesia. Local or perineural administration was necessary in nine cases and three patients required GA. They concluded in their study that ultrasound had facilitated accurate deposition of drug and there is increase in successful performance of block and decrease in complications.

BASIC SCIENCES

Brachial plexus is a somatic plexus formed by the anterior rami of C5 to C8, and most of the anterior ramus of T1. The plexus originates in the neck, passes laterally and inferiorly over rib I, and enters the axilla. All the major nerves that innervate the upper limb originate from the brachial plexus, mostly from the cords. [21]

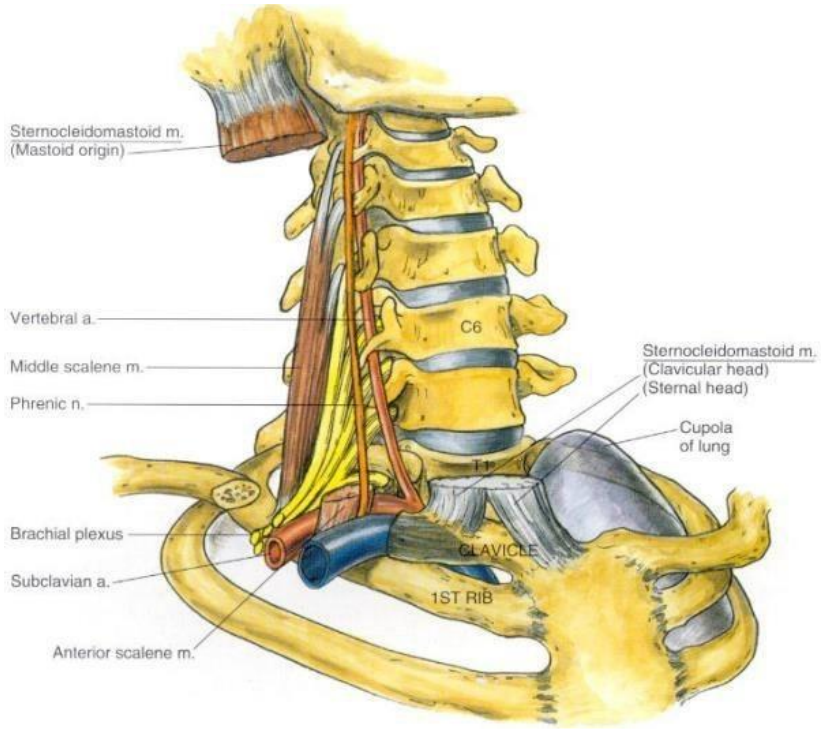


Figure 1-Anatomy of Brachial Plexus

Parts of brachial plexus (21-22)

- Roots
- Trunks
- Divisions
- Cords
- Nerves

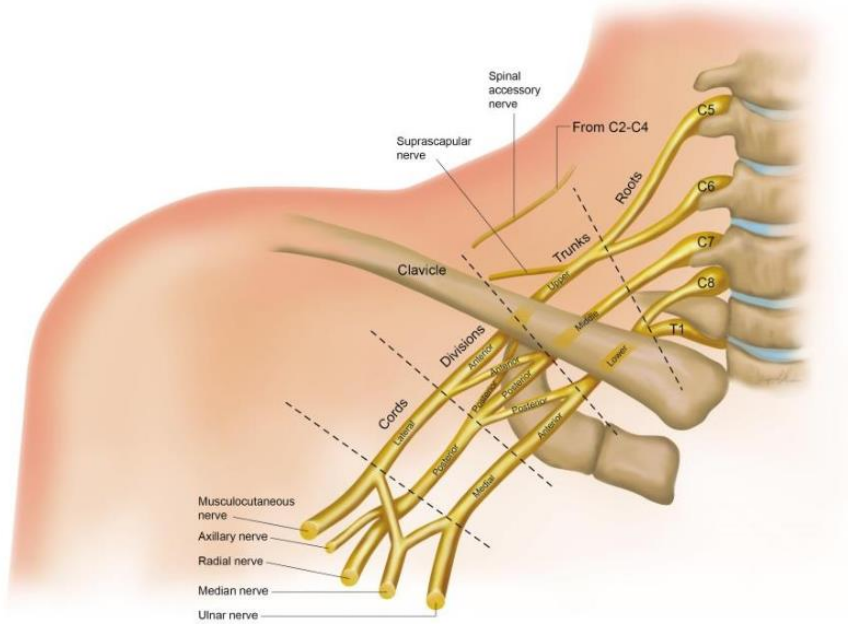


Figure 2-Parts of Brachial Plexus

ROOTS

Brachial plexus roots originate from the anterior rami of C5-C8 spinal nerves and most of T1.They pass between the anterior scalene and middle scalene muscles and enter posterior triangle of the neck. They lie superior and posterior to subclavian artery.

The origin of the plexus may shift by one segment either upward or downward and it is known as prefixed or postfixed plexus.

In prefixed segment contribution by C4 is large and from T2 is absent and in postfixed segment contribution from T1 is large and T2 is always present. C4 is absent and contribution from C5 is reduced.

TRUNKS

Roots join to from the three trunks of brachial plexus. They pass laterally over the first rib and enter the axilla.

Superior trunk- Formed by the union of C5 and C6 roots.

Middle trunk- It is the continuation of C7 root.

Inferior trunk- Formed by the union of C8 and T1 roots.

DIVISIONS

Each trunk divides into anterior and posterior division. The anterior divisions ultimately supply the anterior compartment of the arm and forearm through the peripheral nerves which arise from them. Nerves which are associated with the posterior compartment of arm and forearm arise from the posterior divisions.

CORDS

The three cords originate from the divisions. They are associated with the second part of axillary artery.

Lateral Cord- It is formed by the union of anterior divisions of upper and middle trunks (C5-C7); Medial cord-It is formed by the union of anterior division of lower trunk (C8-T1) Posterior cord-It is formed by the union of posterior divisions of all three trunks (C5-T1).

There are three parts of the axillary artery named for their positions above (medial to), behind, and below (lateral to) the pectoralis minor muscle. Typically, with a ultrasonogram probe placed to view the transverse axis of the cords, the medial cord lies inferior, the lateral cord superior, and the posterior cord posterior to the first part of the axillary artery. Immediately beyond the pectoralis minor muscle, the three cords diverge into the terminal branches, including the median, ulnar, radial axillary, and musculocutaneous nerves.

The phrenic nerve normally descends anterior the scalenus anterior muscle and crosses the muscle from lateral to medial as it descends and passes under the clavicle and through the superior thoracic aperture into the superior mediastinum.

Terminal branches of Brachial Plexus:

In distal axilla, the cord gives rise to terminal branches namely the ulnar, medial and radial nerves.

Branches of the roots:

Nerve to serratus anterior C5, C6,C7

Nerve to rhomboids C5(minor & major)

Branches of the trunk:(arise from the upper trunk)

Supra scapular nerve(C5,C6)

Nerve to subclavius(C5,C6)

Lateral cord:

Lateral pectoral nerve(C5, C6,C7)

Median nerve (lateralroot)(C5,C6,C7)

Musculocutaneous nerve(C5,C6,C7)

Medial cord:

Medial pectoral nerve(C8,T1)

Medial cutaneous division of arm (C8,T1)

Medial cutaneous division of fore arm(C8,T1)

Ulnar nerve (C8,T1)

Median nerve(medial root C8,T1)

Posterior cord:

Upper and lower subscapularnerve(C5,C6)

Thoraco dorsalnerve (C6, C7, C8) O

Axillary nerve (C5,C6)

Radial nerve (C5,C6,C7,C8)

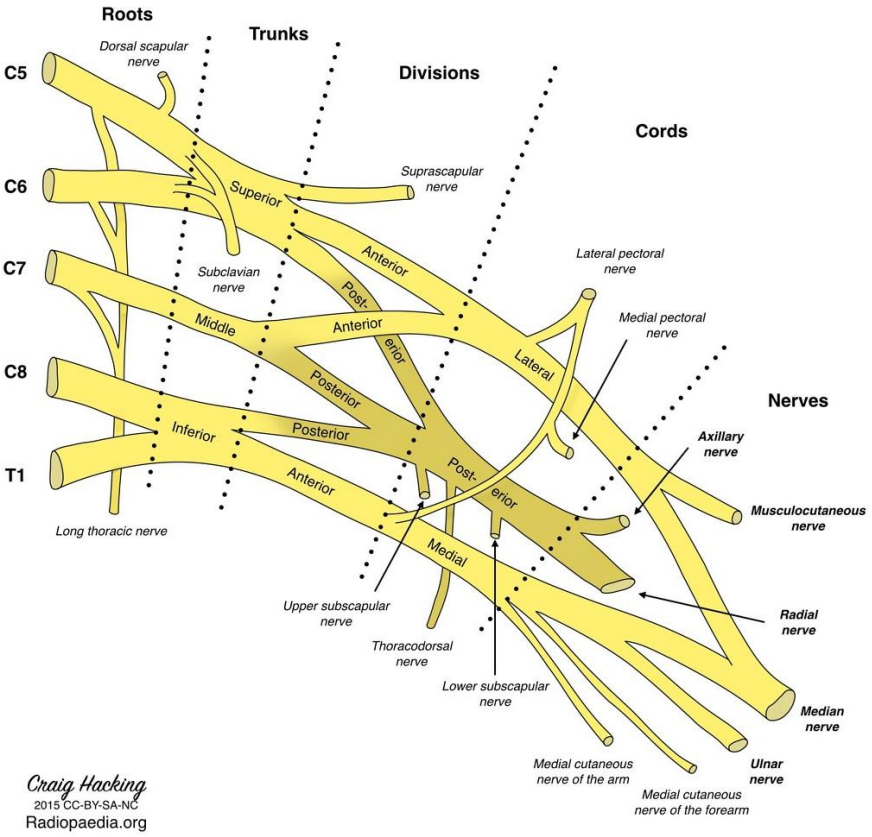


Figure 3-Schematic Diagram of Brachial Plexus

Landmark Technique[23]

Roots

The roots lie between the inter scalenus (anterior & medial) muscles.It is situated cephalo posterior to the second part of subclavian artery. It is the ideal landmark for classical interscalene block.

Trunks

In the posterior triangle, upper and middle trunks emerge above the subclavian artery as they traverse the first rib, but the lower trunk passes behind the artery. The trunks are

enclosed by the skin, platysma and deep fascia superficially. Trunks are overlied by external jugular vein, inferior belly of omohyoid and supraclavicular nerves. The trunks are easily identified by palpation. This landmark is often used for perivascular approach of brachial plexus block.

The divisions emerge from the trunks at the lateral border of first rib and exists behind the clavicle, and then descends into axilla. The rib hitching technique of brachial plexus is performed in this area.

Cords

The cords lie around the axillary artery at the apex of axilla. The Medial cord lies behind the artery,but the posterior and lateral cords are situated lateral to the artery. The infraclavicular approach causes the blockade at the junction of divisions &cords.

Different approaches of brachial plexus block [23]

The various approaches for brachial plexus include:

- Interscelene approach
- Supraclavicular approach
- Infraclavicular approach
- Axillary approach

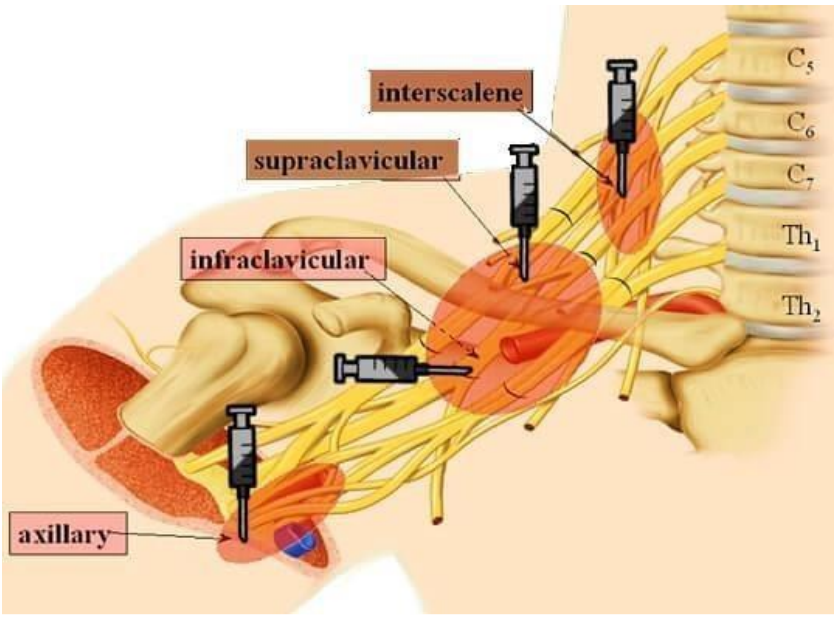


Figure 4-Different Approaches Of Brachial Plexus Block

Interscalene Brachial plexus Block [23]

It is indicated for surgical anaesthesia of the shoulder surgeries such as rotator cuff repair, acromioplasty, hemiarthroplasty and total shoulder replacement, humerus fractures upper arm, forearm but often insufficient for surgeries involving the medial aspect of the forearm or hand. It effectively blocks the proximal nerve roots, distal cervical plexus, and important nerves such as the suprascapular, which exit proximally from the plexus. Frequently it spares the lowest branches of the plexus (C8,T1) and is not suitable for distal upper limb procedures.

Supraclavicular Brachial Plexus Block [23]

It is indicated in surgeries involving arm, elbow, forearm and hand surgery. It targets the trunks or divisions of the brachial plexus depending on the location of the injection site. Here the brachial plexus is confined to its smallest surface area so

success rate of block is high. The three trunks carry the entire sensory, motor, and sympathetic innervation of the upper extremity, with the exception of the uppermost part of the medial side of the arm.

Infraclavicular Brachial Plexus Block [23]

It is indicated in surgeries involving arm, elbow, forearm and hand surgeries. It targets the cords of the brachial plexus and the nerves can be blocked next to the second part of the axillary artery at the level of the coracoid process. It offers excellent analgesia of entire arm and allows introduction of continuous catheters to provide prolonged post-operative pain relief. However the plexus at this level is situated deeper and it is more challenging to give the block.

Axillary Brachial Plexus Block[23]

It is useful in elbow, forearm and hand surgeries. It blocks four nerves

Musculocutaneous, Radial, ulnar and median nerves. The nerves targeted for axillary block course distally with the axillary artery and vein along the humerus from the apex of the axilla. This block is useful for surgery of the elbow, forearm, and hand. The ulnar,median, and radial nerves are the primary targets. The musculocutaneous nerve often leaves the plexus (via the lateral cord) proximal to this point and may be blocked separately during the axillary block (in the coracobrachialis muscle) or at mid-humeral locations (along its diagonal course through or beyond the coracobrachialis muscle).

Landmark Technique for Brachial plexus block.[24]

Interscelene Brachial plexus block-

It targets the roots of brachial plexus.The interscalene groove is palpated by rolling the fingers posteriorly off the lateral border of the sternocleidomastoid muscle.It lies between the inter scelenes muscles(Anterior and medial).After the patient relaxes, the prominent transverse process of C6 can often be felt directly in the groove and should be marked.

Supraclavicular Brachial plexus block.

It targets the trunks or divisions of the brachial plexus. The clavicle is palpated and midpoint of the clavicle is marked. A point is placed posterior to clavicle in sternocleidomastoid groove.Needle will be inserted at an angle of 45 degree in parasagittal plane at the superior border of the clavicle along the lateral edge of the sternocleidomastoid.

Infraclavicular Brachial plexus block-

There are different approaches of giving Infraclavicular brachial plexus block using landmark technique. With the patient's arm adducted and their hand resting on their abdomen, the medial aspect of the coracoid process is palpated. Needle is placed where clavicle meets the medial border of corocoid process at an angle of 0-15 degree. Usually posterior or medial cord of brachial plexus comes in contact.

Axillary Brachial plexus block

The axillary artery is marked as high in its course through the axilla. It is usually felt in the intramuscular groove between the coracobrachialis and the triceps muscles. It also passes between the insertions of the pectoralis major and the latissimus dorsi muscles on the humerus.The artery should be palpated and needle should be placed in cephalad direction and injected inferiorly and superiorly around the artery.

Limitations of Landmark Technique:

There are many variations in the anatomy of the brachial plexus, and in the course of the terminal nerves and vascular elements. Some of these variations may contribute to difficulty in performing peripheral nerve block since there may be unexpected nerve stimulator responses (e.g., if two nerves are conjoined) or poor localization by nerves stimulator or by ultrasonogram imaging (e.g., if the nerve follows a substantially different path),like prefixed (C4-C8), postfixed, (C5-T2), Then continuous, tubular sheath has been shown unlikely, especially in the axillary region

SONOANATOMY OF BRACHIALPLEXUS[25,26]

Ultrasound is a recently emerging technique for regional anesthesia. Ultrasound guided peripheral nerve blockade was first performed in supraclavicular region by La Grange and colleagues in 1978. Later developed by Kapral and coworkers in 1994. Advantages of Ultrasound:

- Enabling real time visualization of brachial plexus, rib, pleura and pulsating subclavian artery.

- Increase in safety because we can appreciate the needle placement and local anesthetic spread during the injection and enables further needle repositioning if needed.
- Rapid onset of nerve block occurs due to drug deposition near the plexus.
- Lesser volume of drug is needed than conventional techniques

ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK.

Supraclavicular Brachial Plexus block [23]

The supraclavicular block targets the trunks and divisions of the brachial plexus .The patient is positioned with the head turned approximately 45 degrees to the contralateral side. The linear probe is placed in a coronal oblique plane at the lateral end and just above the upper border of the clavicle. It is then moved medially until the subclavian artery is seen. With the subclavian artery in the middle the plexus is located superolateral to the artery, and the neurovascular structure structures lie above the first rib. Trunks/divisions of the brachial plexus appear as a cluster of hypoechoic grape like structures. A 50 mm needle is carefully advanced in-plane (lateral to medial) maintaining needle visualization throughout, and local anaesthetic deposited either close to the angle formed at the junction of first rib and subclavian artery, or injected carefully between divisions of the plexus.

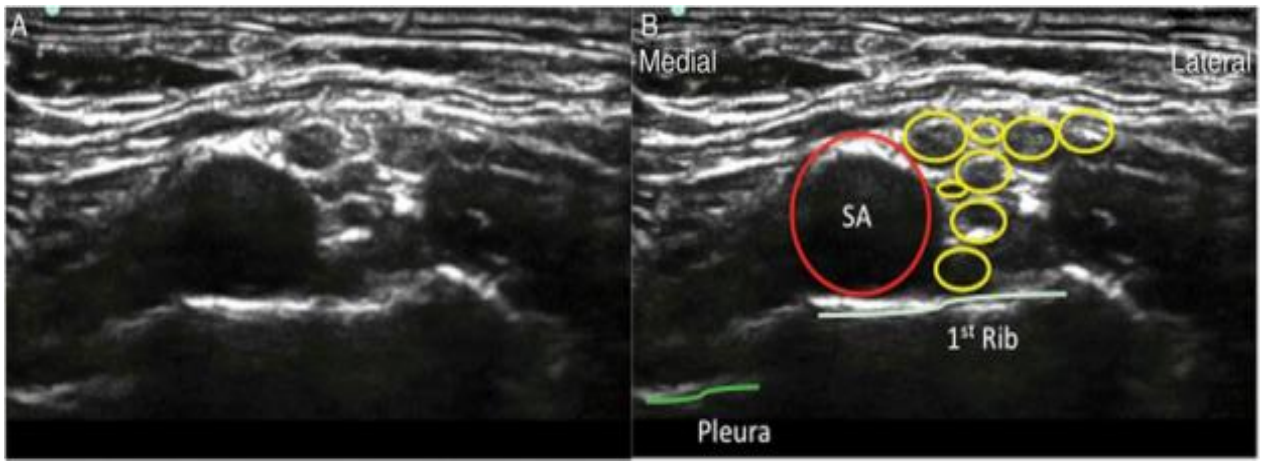


Figure 5-Supraclvicular Brachial plexus block

ULTRASONOGRAPHY[22,27,28]

Ultrasound waves are sound waves with a frequency greater than 20,000Hz.These frequencies are above the audible upper limit of human hearing. Medical ultrasound is the application of this ultrasound waves to visualize the internal organs of our human body. The frequencies used for this purpose, ranges from 3 to 20 MHz. In recent years, ultrasound is widely used in anaesthesia for obtaining vascular access and performing peripheral nerve blocks. Ultrasound guided techniques helps in increasing success rate and reduce its complications.

Ultrasound Pulse Generation

The ultrasound transducer contains multiple piezoelectric crystals which are interconnected electronically. When mechanical energy is applied to these crystals and some ceramics, they generate electrical energy. This phenomenon known as the "Piezoelectric Effect" was first described by the Curie brothers in 1880. They also

described the “Reverse Piezoelectric effect”, wherein application of electricity to these crystals produced vibrations which generate ultrasound waves.

Ultrasound Wavelength and Frequency

The wavelength and frequency are inversely related. High frequency ultrasound waves (10 to 20 MHz) give images with a high axial resolution but are more attenuated as we go deeper. Therefore, these transducers are optimal to image the superficial structures. Low frequency ultrasound waves (2 to 8 MHz) penetrate deeper but provide low axial resolution and are used to image deeper structures.

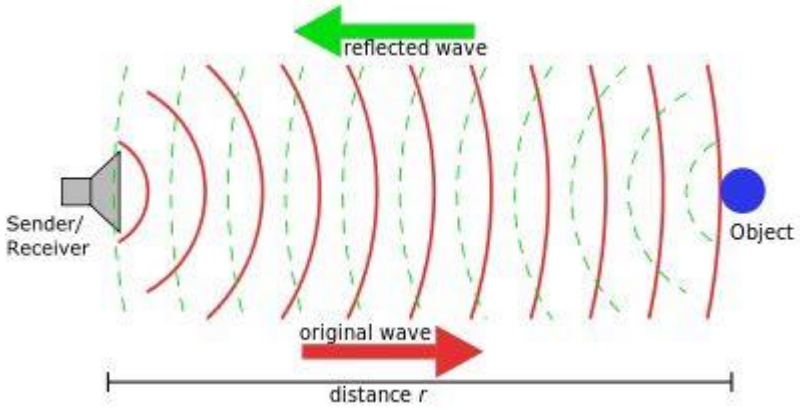


Figure 6-Principles of Ultrasonography

Ultrasound Tissue Interaction:

As the ultrasound waves travel through tissues, they are partly transmitted to deeper structures, partly reflected back to the transducer as echoes, partly scattered, and partly transformed to heat.

Reflection

For image generation, the echoes returned after hitting a tissue interface is of interest to us. The amount of echo returned after hitting a tissue interface is determined by a tissue

property called acoustic impedance. The intensity of a reflected echo is proportional to the mismatch in acoustic impedances between two mediums.

Refraction

The change in the direction of the ultrasound waves after hitting an interface between two media with different velocities of sound transmission is refraction. This causes artefacts as the returning echoes are incorrectly located.

Scattering

Ultrasound waves which incident on the tissues at right angles are reflected back to the transducer. If the waves are not at right angle, then the returning echoes are scattered in all directions in a non-uniform manner.

Absorption

Some of the ultrasound waves are absorbed by the tissue and are converted to heat.

Attenuation

As the ultrasound waves travel through tissue, the returning echoes will become weaker due to absorption, scattering and refraction.

Diffraction

The spreading out of the ultrasound waves as its moves further away from the source is diffraction.

Construction

The ultrasound probe has an array of individual transducers which acts as both a transmitter and a receiver. Each transducer emits a short burst of ultrasound and is quiescent until it detects the echoes returning. This is called “Pulsed Ultrasound”. The

speed of ultrasound in our body tissues is fairly constant at a speed of 1540m/s.The time taken for an echo to return is used determine the distancebetween the tissue and the probe.

Across the plane of an image, the ultrasound image is swept to form two dimensional images one line at a time. These lines are then summated to produce a frame. The frames are repeated to produce a real-time image. The brightness of the image depends upon the amplitude of the returning echo from the anatomical interfaces.

Scanning Modes

A-mode (amplitude mode): This displays a single echo signal against time to measure depth.

B-mode (brightness mode): It is a two dimensional image produced using an array of transducers and a series of reflected echoes.

M-mode (motion mode): is a specialized type of B-mode imaging where one particular line is ensonified repeatedly to examine a moving structure plotting out how the structure moves with time.

Ultrasound controls

Gain alters the brightness of the image by amplifying the received signal.

Time-Gain Compensation (TGC) differentially amplifies signals from different depths, allowing equal amplitudes from all depths to be displayed.

Focus adjusts the beam to be at its narrowest at the required depth to image the region of interest. It thereby improves lateral resolution

Depth can be adjusted to have the structure that is being examined to be in the centre of the screen.

Approaches and techniques

There are two basic approaches to ultrasound guidance. With the out-of- plane technique, the needle tip crosses the plane of imaging as an echogenic dot. With the in-plane approach, the entire tip and shaft of the advancing needle are visible.

Out-of-plane:

This technique involves insertion of needle at the midpoint of probe such that the needle cuts across the ultrasonic beam. The image obtained is a cross section of the needle shaft or tip. Path to target is shorter as compared to in-plane technique, but visibility of needle is not optimum, indirect markers like tissue movement or hydrodissection is needed to confirm placement.

Advantages:

1) Most similar to other approaches to regional block (nerve stimulation orpalpation)

2) Shorter needle path than with in-plane approaches

3) Along the nerve path (catheters)

Disadvantages:

Unimaged needle path, crossing the plane of imaging without recognition.

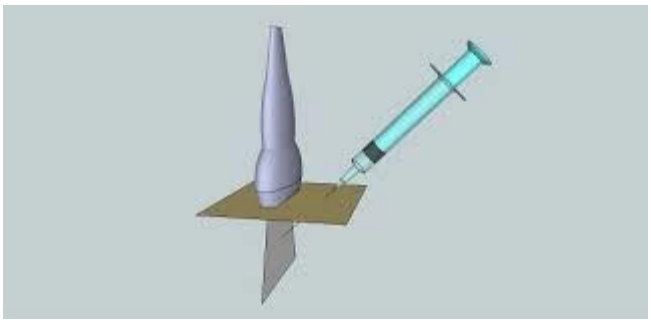


Figure 7-Out of plane approach

In-plane (IP):

In this technique needle is inserted along the length of ultrasound probe. It aligns the entire length of the beam with the shaft of needle. The image displayed will depict the entire needle shaft and its tip thereby improving the precision of nerve blocks. But the needle visibility depends on angle of insertion and the needle traverses a longer path to reach the target area.

Advantages:

Most direct visualization.

Disadvantages:

1) Partial line-ups (creating a false sense of security when the needle tip is not correctly identified.

2) Some unimaged needle path occurs with IP approach, but typically less than with OOP approach.

3) Longer paths and therefore more structures to cross with the block needle.

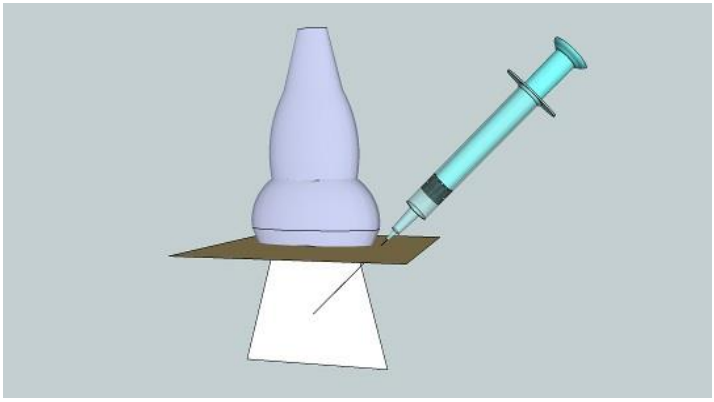


Figure 8-Inplane approach of ultrasonography

Ultrasound probes

Commonly used are three types

- Linear high frequency (6 to 12 MHz) probes which has high resolution and lesser penetration and is ideal for visualizing superficial structures.

- Curvilinear low frequency probes (2-5MHz) which has low resolution, higher penetration and is ideal for deeper structures like intraabdominal organs.

- Phased Array Probe also has low frequency (2MHz – 7.5MHz) gives a large depth with a small acoustic window, ideal for chest ultrasound

Figure 9-Ultrasound probes

Imaging

Ultrasound image is produced by echoes received as the Ultrasonic beam interacts with the tissues it travels through. Acoustic impedance of a structure is the function of the elasticity and density of the particular tissue. Materials with higher acoustic impedance transmit sound faster, and do not allow for continued compression by the impending wave. The sound beam is attenuated while traversingvarious tissues within the body. The beam will be scattered somewhat when it encounters varying tissues on the way with different acoustic impedances or it may be reflected back from structures and returns back to the transducer. Refraction and absorption by tissues may also attenuate the waves. Those tissues that reflect the wave are termed echoic and those which do not reflect the wave are termed anechoic. Always use plenty of sterile ultrasound gel to remove the air interface between the skin and probe. Air does not allow the passage of the ultrasound beam even though it has low Acoustic impedance. Bone has high acoustic index so it appears to be white on the ultrasound image as it is hyper reflective to the beam. Blood and other fluids appear to be black on the image since they are anechoicin nature. Soft tissue appears as grey on the sonographic image as they have medium echogenicity.

The nerves appear round or oval in transverse view and are hypo-echoic or they appear as honeycomb structures with septations inside them. Nerves are bordered by a hyper-echoic layer of connective tissue. Blood vessels will appear as circular hypoechoic to anechoiec

structures with a well-defined hyper- echoic border which is the vessel wall. Veins are compressible with thinner walls whereas arteries have thicker walls and appear pulsatile in nature. Muscles have fibrous-lamellartexture and appear as heterogeneous or homogeneous hypoechoic structures with hyper-echoic septa in between.

Basic principles of ultrasound guided nerve blocks.

- First involves the identification of anatomical structures like muscles, fascia, blood vessels and bones.

- Visualization of the nerve plexus or the fascial plane where drug should be deposited.

- Should be able to differentiate between normal and altered anatomy of the region scanned.

- Identify the correct plane for needle insertion to avoid trauma to vessels

- Strict aseptic technique

- Real time visualization of needle when it is inserted inside.

-Once the target is reached, inject a small volume of drug or saline and see the spread and confirm location, else reposition the needle.

- Do frequent aspiration during injection of drug to rule out intravascular injection.

- Complete visualization of the spread of total volume of local anaesthetic drug injected.

- Always keep ready all resuscitation equipment, drugs and standard monitoring.

LOCAL ANAESTHESIA (OR) REGIONAL ANAESTHESIA:[29]

Local anesthesia can be defined as loss of sensation, in a discrete region of the body caused by disruption of impulse generation or propagation. Nerve fibers can be classified according to fiber diameter, presence (type A and B) or absence (type C) of myelin, and function. The nerve fiber diameter influences conduction velocity. Larger diameter correlates with more rapid nerve conduction. The presence of myelin also increases conduction velocity. This effect results from insulation of the axolemma from the surrounding media, forcing current to flow through periodic interruptions in the myelin sheath (i.e., nodes of Ranvier) Local anesthetics act on a wide range of molecular targets, but they exert their predominant desired clinical effects by blocking sodium ion flux through voltage-gated sodium channels. Voltage-gated sodium channels are complex transmembrane proteins comprising large alpha subunits and much smaller beta subunits.

Intravenous infusions of lipid emulsions have become a standard treatment of LAST . The mechanism by which lipid is effective is not clear but is likely related to its ability to extract bupivacaine (or other lipophilic drugs like Ropivacaine) from aqueous plasma or tissue targets, thus reducing their effective free concentration (“lipid sink”). Accordingly, solutions of lipid emulsion should be stocked and readily accessible in any area where major conduction blockade is performed, as well as locations where overdoses from any lipophilic drug might be treated

BUPIVACAINE [29-31]

Bupivacaine is an amino amide class of local anaesthetic drug. It was first synthesized by Ekenstam in 1957 and its clinical use was started by LJ Telivuo in 1963. Since then, it has become one of the widely used local anaesthetic agents clinically.

Bupivacaine consists of a tertiary amine attached to a substituted aromatic ring by an amide linkage. The butyl group attached to the piperidine nitrogen makes bupivacaine more lipid soluble and potent. The molecular weight is 288. It is a chiral drug that exists as two enantiomeric forms – dextrorotary (R-) and levorotary (S-) forms. The pure levorotary form Levobupivacaine produce less cardiotoxicity compared to that of the racemic mixture.

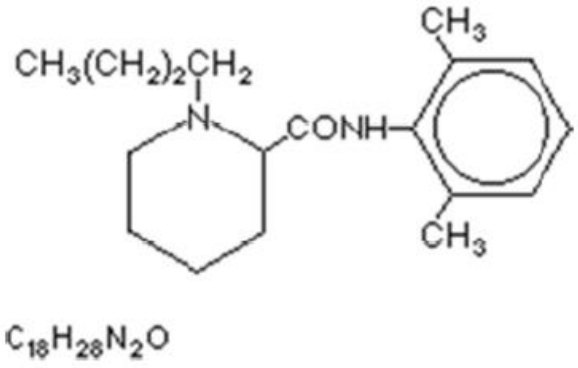


Figure 10- Structure of Bupivacaine

PHARMACODYNAMICS :

Bupivacaine permeates the nerve's axon membranes and accumulate within the axoplasm. Binding to sites on voltage-gated Na+ channels prevent opening of the channels by inhibiting theconformational changes that underlie channel activation.

On comparison with lignocaine, it is four times more potent but the onset of action is slower. The duration of action is considerably longer. The sensory blockade caused by bupivacaine is much more than the motor blockade.

PHARMACOKINETICS

It is a weak base with a pKa of 8.1. Bupivacaine is highly protein bound (95%) and most important plasma protein binding site is alpha1 acid glycoprotein. At physiological pH of 7.4, 17% is non-ionised.

The onset and duration of action depend on the dose, concentration, route of administration and vascularity of the site of administration. The volume of distribution is 54 L. The elimination half-life is 210 minutes. The Clearance is 0.32 L/min. Bupivacaine undergoes biotransformation in liver by aromatic hydroxylation, N-dealkylation, amide hydrolysis, and conjugation. The metabolites are excreted via the kidney. Less than 5% of the drug is excreted unchanged.

Dosage and preparations

Maximum dose of bupivacaine 2-3 mg/kg. Preparations available include 0.25%, 0.5% solutions in 10 ml and 20 ml vials, preservative free 0.5% bupivacaine and 0.75% bupivacaine for intrathecal injections.

Uses

- Peripheral nerve block (0.25-0.5%)
- Epidural Anaesthesia (0.25-0.5%)

- Spinal Anaesthesia (0.5%, 0.75%)
- Caudal Anaesthesia (0.25-0.5%)
- Infiltration Anaesthesia (0.25-0.5%)

Contraindications

- Known hypersensitivity to local anaesthetics
- Intravenous regional anaesthesia (IVRA)

Adverse effects

Local Anaesthesia Systemic Toxicity– Plasma concentration greater than 5mcg/ml due to overdosage, unintentional intravascular injection and slow metabolic degradation causes systemic toxicity.

Central Nervous System Toxicity

Non-specific signs of toxicity are metallic taste, circumoral numbness, diplopia, tinnitus, dizziness. Excitation is characterized by restlessness, anxiety,dizziness, tinnitus, blurred vision or tremors. Then, there is a depression of central nervous system causing drowsiness, unconsciousness and cardiac arrest.

Cardiovascular system effects

Part of the cardiac toxicity that occurs with high plasma concentrations of bupivacaine occurs because of the blockade of cardiac sodium channels. Accidental intravenous injection of bupivacaine causes cardiac dysrhythmias, atrioventricular block, ventricular tachycardia and ventricular fibrillation, bradycardia and asystole.

Pregnancy increases the sensitivity of cardiotoxic effects of bupivacaine.

LIGNOCAINE:[29,31]

Lidocaine, formerly also referred to as lignocaine, is an amide local anaesthetic agent. First synthesized between 1943 and 1946 by Nils Löfgren and Bengt Lundquist, it is a tertiary amine derived from xylidine, and its use rapidly became widespread given its superior safety profile compared to older local anaesthetic agents. The drug is commonly used for local anaesthesia, often combined with epinephrine (which acts as a vasopressor and extends its duration of action at a site by opposing the local vasodilatory effects of lidocaine). Given intravenously, it can be used during advanced airway management as an adjuvant to tracheal intubation, obtunding the hypertensive response to laryngoscopy and potentially reducing the incidence of myalgia and hyperkalemia when succinylcholine is given. Lidocaine is a class Ib antiarrhythmic agent on the Vaughan-Williams classification, and its use is indicated in the management of acute ventricular tachydysrhythmias. It also has roles as an adjuvant analgesic in managing acute and chronic pain.

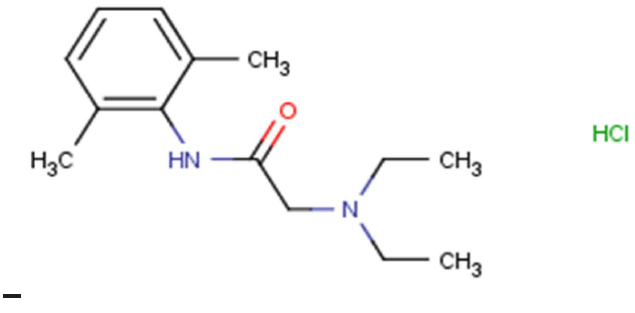


Figure-11: Chemical structure of Lignocaine

Mechanism Of Action:

The site of action of lidocaine is at sodium ion channels on the internal surface of nerve cell membranes. The uncharged form diffuses through neural sheaths into the axoplasm before ionizing by combining with hydrogen ions. The resulting cation binds reversibly to sodium channels from the inside, locking them in the open state and preventing nerve depolarization. As lidocaine is a weak base with a dissociation constant (pKa) of 7.7, approximately 25% of molecules will be un-ionized at a physiological pH of 7.4 and will be available to translocate inside the nerve cells, meaning that lidocaine has a more rapid onset of action than other local anaesthetics with higher pKa values. Efficacy decreases in the presence of inflammation; this can be due to acidosis decreasing the proportion of un-ionized lidocaine molecules, a faster decrease in lidocaine concentration due to increased blood flow, and potentially also through increased production of inflammatory mediators like peroxynitrite, which act directly on sodium channels. In cardiac myocytes, lidocaine slows the rise of the cardiac action potential during phase 0, thereby increasing the effective threshold potential.

Lidocaine is 65% protein-bound to albumin and alpha1-acid glycoprotein in the plasma, giving it a medium duration of action compared to other local anesthetic agents. It is less lipid-soluble than other agents, limiting its overall potency. Its volume of distribution is 0.7 to 1.5 L/kg, and it is metabolized by hepatic enzymes to active and inactive metabolites.

HALF-LIFE

The elimination half-life of lidocaine hydrochloride following an intravenous bolus injection is typically 1.5 to 2.0 hours. Because of the rapid rate at which lidocaine hydrochloride is metabolized, any condition that affects liver function may alter lidocaine HCl kinetics. The half-life may be prolonged two-fold or more in patients with liver dysfunction.

PHARMACOKINETICS:

Absorption:

In general, lidocaine is readily absorbed across mucous membranes and damaged skin but poorly through intact skin. The agent is quickly absorbed from the upper airway, tracheobronchial tree, and alveoli into the bloodstream. And although lidocaine is also well absorbed across the gastrointestinal tract the oral bioavailability is only about 35% as a result of a high degree of first-pass metabolism. After injection into tissues, lidocaine is also rapidly absorbed and the absorption rate is affected by both vascularity and the presence of tissue and fat capable of binding lidocaine in the particular tissues

The concentration of lidocaine in the blood is subsequently affected by a variety of aspects, including its rate of absorption from the site of injection, the rate of tissue distribution, and the rate of metabolism and excretion Subsequently, the systemic absorption of lidocaine is determined by the site of injection, the dosage given, and its pharmacological profile The maximum blood concentration occurs following intercostal

nerve blockade followed in order of decreasing concentration, the lumbar epidural space, brachial plexus site, and subcutaneous tissue. The total dose injected regardless of the site is the primary determinant of the absorption rate and blood levels achieved There is a linear relationship between the amount of lidocaine injected and the resultant peak anaesthetic blood levels.

Nevertheless, it has been observed that lidocaine hydrochloride is completely absorbed following parenteral administration, its rate of absorption depending also on lipid solubility and the presence or absence of a vasoconstrictor agent. Except for intravascular administration, the highest blood levels are obtained following

METABOLISM

Lidocaine is metabolized predominantly and rapidly by the liver, and metabolites and unchanged drug are excreted by the kidneys. Biotransformation includes oxidative N-dealkylation, ring hydroxylation, cleavage of the amide linkage, and conjugation.

N-dealkylation, a major pathway of biotransformation, yields the metabolites monoethylglycinexylidide and glycinexylidide. The pharmacological/toxicological actions of these metabolites are similar to, but less potent than, those of lidocaine HCl. Approximately 90% of lidocaine HCl administered is excreted in the form of various metabolites, and less than 10% is excreted unchanged. The primary metabolite in urine is a conjugate of 4-hydroxy-2,6-dimethylaniline

ROUTE OF ELIMINATION

The excretion of unchanged lidocaine and its metabolites occurs predominantly via the kidney with less than 5% in the unchanged form appearing in the urine. The renal clearance is inversely related to its protein binding affinity and the pH of the urine. This suggests by the latter that excretion of lidocaine occurs by non-ionic diffusion.

PHARMACODYNAMICS

Excessive blood levels of lidocaine can cause changes in cardiac output, total peripheral resistance, and mean arterial pressure. With central neural blockade these changes may be attributable to the block of autonomic fibres, a direct depressant effect of the local anaesthetic agent on various components of the cardiovascular system, and/or the beta-adrenergic receptor stimulating action of epinephrine when present. The net effect is normally a modest hypotension when the recommended dosages are not exceeded.

In particular, such cardiac effects are likely associated with the principal effect that lidocaine elicits when it binds and blocks sodium channels, inhibiting the ionic fluxes required for the initiation and conduction of electrical action potential impulses necessary to facilitate muscle contraction. Subsequently, in cardiac myocytes, lidocaine can potentially block or otherwise slow the rise of cardiac action potentials and their associated cardiac myocyte contractions, resulting in possible effects like hypotension, bradycardia, myocardial depression, cardiac arrhythmias, and perhaps cardiac arrest or circulatory collapse.

Moreover, lidocaine possesses a dissociation constant (pKa) of 7.7 and is considered a weak base. As a result, about 25% of lidocaine molecules will be un-ionized and available at the physiological pH of 7.4 to translocate inside nerve cells, which means lidocaine elicits an onset of action more rapidly than other local anaesthetics that

have higher pKa values. This rapid onset of action is demonstrated in about one minute following intravenous injection and fifteen minutes following intramuscular injection.The administered lidocaine subsequently spreads rapidly through the surrounding tissues and the anaesthetic effect lasts approximately ten to twenty minutes when given intravenously and about sixty to ninety minutes after intramuscular injection.

Nevertheless, it appears that the efficacy of lidocaine may be minimized in the presence of inflammation. This effect could be due to acidosis decreasing the amount of un-ionized lidocaine molecules, a more rapid reduction in lidocaine concentration as a result of increased blood flow, or potentially also because of increased production of inflammatory mediators like peroxynitrite that elicit direct actions on sodium channels.

TOXICITY

Symptoms of overdose and/or acute systemic toxicity involves central nervous system toxicity that presents with symptoms of increasing severity. Patients may present initially with circumoral paraesthesia, numbness of the tongue, light-headedness, hyperacusis, and tinnitus .Visual disturbance and muscular tremors or muscle twitching are more serious and precede the onset of generalized convulsions. These signs must not be mistaken for

neurotic behavior. Unconsciousness and grand mal convulsions may follow, which may last from a few seconds to several minutes. Hypoxia and hypercapnia occur rapidly following convulsions due to increased muscular activity, together with the interference with normal respiration and loss of the airway. In severe cases, apnoea may occur. Acidosis increases the toxic effects oflocal anaesthetics. Effects on the cardiovascular system may be seen in severe cases. Hypotension, bradycardia, arrhythmia and cardiac arrest may occur as a result of high systemic concentrations, with potentially fatal outcome.

Pregnancy Category B has been established for the use of lidocaine in pregnancy, although there are no formal, adequate, and well-controlled studies in pregnant women. General consideration should be given to this fact before administering lidocaine to women of childbearing potential, especially during early pregnancy when maximum organogenesis takes place. Ultimately, although animal studies have revealed no evidence of harm to the fetus, lidocaine should not be administered during early pregnancy unless the benefits are considered to outweigh the risks. Lidocaine readily crosses the placental barrier after epidural or intravenous administration to the mother. The ratio of umbilical to maternal venous concentration is 0.5 to 0.6. The fetus appears to be capable of metabolizing lidocaine at term. The elimination half-life in the newborn of the drug received in utero is about three hours, compared with 100 minutes in the adult [7]. Elevated lidocaine levels may persist in the newborn for at least 48 hours after delivery. Foetal

bradycardia or tachycardia, neonatal bradycardia, hypotonia or respiratory depression may occur.

Local anesthetics rapidly cross the placenta and when used for epidural, paracervical, pudendal or caudal block anaesthesia, can cause varying degrees of maternal, foetal and neonatal toxicity. The potential for toxicity depends upon the procedure performed, the type and amount of drug used, and the technique of drug administration. Adverse reactions in the parturient, fetus and neonate involve alterations of the central nervous system, peripheral vascular tone, and cardiac function.

Maternal hypotension has resulted from regional anaesthesia. Local anesthetics produce vasodilation by blocking sympathetic nerves. Elevating the patient's legs and positioning her on her left side will help prevent decreases in blood pressure. The foetal heart rate also should be monitored continuously, and electronic foetal monitoring is highly advisable.

Epidural, spinal, paracervical, or pudendal anaesthesia may alter the forces of parturition through changes in uterine contractility or maternal expulsive efforts. In one study, paracervical block anaesthesia was associated with a decrease in the mean duration of first stage labor and facilitation of cervical dilation. However, spinal and epidural anaesthesia have also been reported to prolong the second stage of labor by removing the parturient's reflex urge to bear down or by interfering with motor function. The use of obstetrical anaesthesia may increase the need for forceps assistance.

The use of some local anaesthetic drug products during labour and delivery may be followed by diminished muscle strength and tone for the first day or two of life. The long-term significance of these observations is unknown. Foetal bradycardia may

occur in 20 to 30 percent of patients receiving paracervical nerve block anaesthesia with the amide-type local anaesthetics and may be associated with foetal acidosis. Foetal heart rate should always be monitored during paracervical anaesthesia. The physician should weigh the possible advantages against risks when considering a paracervical block in prematurity, toxaemia of pregnancy, and foetal distress. Careful adherence to the recommended dosage is of the utmost importance in obstetrical paracervical block. Failure to achieve adequate analgesia with recommended doses should arouse suspicion of intravascular or foetal intracranial injection. Cases compatible with unintended foetal intracranial injection of local anaesthetic solution have been reported following intended paracervical or pudendal block or both. Babies so affected present with unexplained neonatal depression at birth, which correlates with high local anaesthetic serum levels, and often manifest seizures within six hours. Prompt use of supportive measures combined with forced urinary excretion of the local anesthetic has been used successfully to manage this complication.

It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when lidocaine is administered to a nursing woman.

Dosages in children should be reduced, commensurate with age, body weight and physical condition.

EPINEPHRINE (ADRENALINE)[32]

PHARMACOLOGY

Epinephrine (β-[3,4-dihydroxyphenyl]-α-methylaminoethanol) is an endogenous catecholamine that is produced by the adrenal medulla. It is synthesized in the body from the amino acid tyrosine by a series of enzymatic steps. Both the endogenous form and the synthetic form are the levorotatory (L) isomer of the catecholamine, which is 15 times more active than the dextrorotatory (R) isomer.

CHEMICAL STRUCTURE

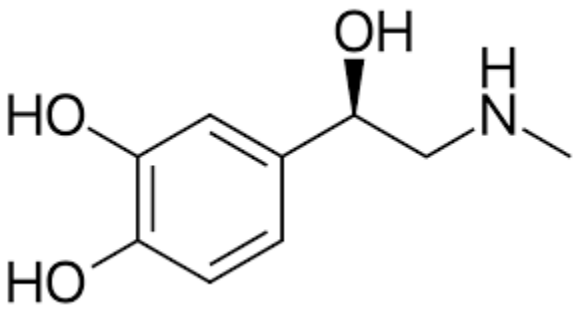


Figure-12: Chemical structure of adrenaline

Although epinephrine is both an α- and a β-adrenergic agonist, it is the α-adrenergic properties that cause vasoconstriction. Epinephrine is only stable in acidic environments, which slows the onset of the local anesthetic and makes its injection painful.As

mentioned above, these limitations are mitigated by the addition of sodium bicarbonate, which neutralizes the pH. The maximum vasoconstrictive effect of epinephrine takes place within 7 to 15 minutes, and the duration of action is short (approximately 60 minutes). Epinephrine is rapidly inactivated in tissue primarily by enzymatic transformation to metanephrine or normetanephrine. These substances are then conjugated in the liver. Epinephrine also undergoes direct degradation in the liver by monoamine oxidase and catechol O-methyltransferase. Once metanephrine and normetanephrine are conjugated, they are excreted in the urine in the form of sulfates and glucuronides. Either sequence results in the production of vanillylmandelic acid (VMA), which can be detected in the urine

MECHANISM OF ACTION

Epinephrine has complex target organ effects. It is a potent agonist at both alpha- and beta- receptors throughout the body except for the sweat glands and facial arteries. Epinephrine is a nonselective adrenergic agonist; it stimulates $alpha_1$-, $alpha_2$-, $beta_1$-, and $beta_2$-adrenergic receptors, although the degree of stimulation at these receptors may vary depending on the dose administered (i.e., the circulating concentration of epinephrine at the receptor). Stimulation of $alpha_1$-receptors by epinephrine leads to arteriolar vasoconstriction. Stimulation of presynaptic $alpha_2$-receptors inhibits norepinephrine release via negative feedback while stimulation of post-synaptic $alpha_2$-receptors also leads to arteriolar vasoconstriction. Stimulation of $beta_1$-receptors induces a positive

chronotropic and inotropic response. Stimulation of $beta_2$-receptors by epinephrine leads to arteriolar vasodilation, bronchial smooth muscle relaxation, and increased glycogenolysis. Subsequent to binding at the adrenergic receptor, the intracellular actions of epinephrine are mediated by cyclic adenosine monophosphate (cAMP). The production of cAMP is augmented by beta-stimulation and attenuated by alpha-stimulation.

The major therapeutic effects of systemic epinephrine include: bronchial smooth muscle relaxation, cardiac stimulation, vasodilation in skeletal muscle, and stimulation of glycogenolysis in the liver and other calorigenic mechanisms. The effects of epinephrine on smooth muscle are varied and determined by relative receptor density and hormonal effects. When used topically in the eye in patients with open-angle glaucoma, epinephrine lowers intraocular pressure, produces a brief mydriasis, and may improve the coefficient of aqueous outflow. When used topically on the skin or mucosal surfaces, epinephrine constricts arterioles, thus producing local vasoconstriction and hemostasis in small blood vessels.

Epinephrine primarily exerts its relaxant effect on bronchial smooth muscle via stimulation of $beta_2$-receptors. $Beta_2$-stimulation also prevents mast cell secretion of histamine and other autocoids, thus antagonizing its effect on end organs and reversing bronchoconstriction and edema. Furthermore, alpha-stimulation may decrease secretions from the bronchial mucosa, attenuating the development of edema. There is some

evidence that epinephrine's alpha properties make it more effective than pure beta-agonists for the treatment of some pulmonary conditions such as bronchiolitis in children.

The potent cardiac effects of epinephrine are primarily mediated via stimulation of $beta_1$-receptors on the myocardium and conducting system of the heart. The stimulation of these receptors results in both increased inotropic and chronotropic effects. Systolic blood pressure is usually elevated as a result of increased inotropy, although diastolic blood pressure is decreased secondary to epinephrine-induced vasodilation. As a result, pulse pressure is increased.

Epinephrine indirectly causes coronary artery vasodilation, particularly during cardiac arrest. Epinephrine can simultaneously increase myocardial oxygen supply (secondary to coronary vasodilation) and increase oxygen demand (secondary to a positive inotropic and chronotropic effect on the heart). Increased myocardial excitability and automaticity markedly increase the potential for developing dysrhythmias. Nonspecific beta-stimulation by epinephrine, combined with moderate alpha agonism, results in inotropic effects equal to those of dopamine and dobutamine but greater chronotropic effects than either agent.

Blood flow to skeletal muscles is augmented by epinephrine via $beta_2$-stimulation, resulting in vasodilation. Stimulation of $alpha_1$-receptors by epinephrine leads to arteriolar vasoconstriction while stimulation of $beta_2$-receptors by epinephrine leads to arteriolar vasodilation. At normal therapeutic doses, this effect is only mildly countered

by the vasoconstriction caused by alpha-stimulation. At higher doses, however, vasoconstriction and elevation of both peripheral vascular resistance and blood pressure can occur.

The metabolic effects of epinephrine relate primarily to the regulatory processes that control glucose concentration in the plasma. $Beta_2$-stimulation of the skeletal muscle and liver increases glycogenolysis. Alpha-stimulation of the liver increases gluconeogenesis and inhibits insulin release by the pancreatic islet cells. Furthermore, in adipose cells, beta-stimulation will induce the catabolism of triglycerides, therefore increasing plasma free fatty acids. Serum potassium concentrations fluctuate after administration of epinephrine. Initially, hyperkalemia occurs secondary to release of potassium ions from hepatocytes. Hypokalemia quickly follows as potassium ions are taken up by the skeletal muscle.

Epinephrine is most often used as a vasoconstrictor injected with a local anesthetic.

It has four main purposes:

1. To prolong the effect of the local anesthetic

2. To reduce systemic absorption of the local anesthetic

3. To aid in hemostasis

4. To decrease the volume of the local anesthetic injected.

CONTRAINDICATIONS

ABSOLUTE	RELATIVE
• Sensitivity to sodium metabisulphite • Pheochromocytoma	• Cardiac disease: unstable angina, recent myocardial infarction, recent coronary artery bypass graft (CABG) surgery, refractory arrhythmias, uncontrolled hypertension, uncontrolled congestive heart failure • Pregnancy • Poorly controlled hyperthyroidism • Poorly controlled diabetes mellitus • Severe peripheral vascular disease • Angle-closure glaucoma • Cocaine abuse

VISUAL ANALOGUE SCALE[33,34]

The experience of pain is unquestionably subjective. Factors such as personality, memories of past painful events, emotional state, age, cultural background, context, and other variables all contribute to how individuals perceive and communicate their pain.

In any clinical context or scenario, successful pain management relies on a meticulous and accurate assessment of pain-an individual approach.

Multiple studies and research findings concur that pain intensity (PI) is the most crucial aspect of pain assessment in clinical practice, irrespective of the specific disease or condition.

Extensive evidence supports the widespread use of the 100 mm visual analog scale (VAS) as the predominant assessment tool.

A Visual Analog Scale (VAS) is a simple but valuable instrument that tries to measure a characteristic or attitude that is believed to range across a continuum of values and cannot easily be directly measured.

It usually is a horizontal line, 100 mm in length, anchored by word descriptors at each end, for example, that represent the severity of symptoms from 0 “no symptoms” to 10 “very severe symptoms.” The patient marks on the line the point that the patient believes represents his or her perception of his or her current state.

One of the advantages of VAS is its ease of use and provision of a quick assessment. However, a disadvantage of using such a scale is the lack of extreme values of the VAS because patients tend to rate their symptom severity by avoiding the extremes.[2]

Figure 13 - VISUAL ANALOGUE SCALE

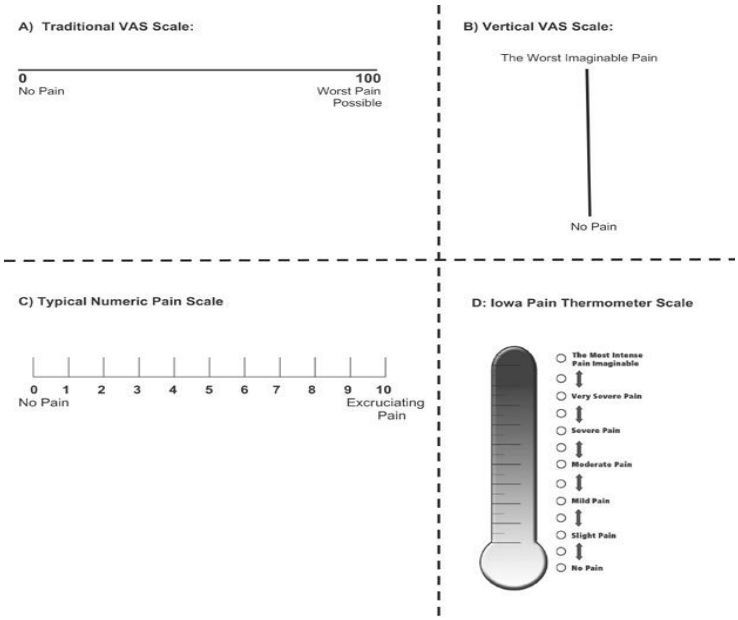


Figure14:

VARIOUS OTHER PAIN SCALES

A) Traditional VAS scale

B) Vertical VAS scale

C) Typical Numeric pain scale

D) Lowa pain thermometer scale

METHODOLOGY

Title : “Comparision between Lateral Decubitus position and Conventional Supine position to determine the distance between Pleura and Brachial Plexus in patients undergoing Ultrasound Guided Supraclavicular Brachial Plexus Block for Upper limb surgeries: A Randomised Control Trial” which was done in “Department of Anaesthesiology, KLE’S Dr.Prabhakar Kore Hospital, Belagavi during the period of March 2023 to February 2024”.

Type of study: A Randomised Control Trial.

Source of Data:60 ASA I,II, & III patients, aged between 18-70 years,of both gender, undergoing elective surgical procedures related to upper limb, at K.L.E’s Dr. PKC Hospital & Medical Research Centre, Nehru Nagar, Belagaum -10

Designof the study : Hospital based clinical study

Total duration of the study : one year

Study population : Adult patients posted for surgery of upper limb under regional anesthesia between March 2023 to February 2024 at “KLE’S Dr.Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi-10.”were recruited as per inclusion and exclusion criteria.

Sample Size- 60 patients in total.

Random sampling procedure- A one year randomized control study. Randomization was achieved by computer generated randomization chart.

Sample SizeCalculation:

Sample size formula:

Using a two-proportion analysis, the minimum required sample size formula is determined by

$$n = \frac{(z_\alpha + z_\beta)^2 \bar{p}(1-\bar{p})}{d^2}$$

Herep$_1$and p$_2$ denote the proportions within the two groups.

$$p = \frac{p_1 + p_2}{2} \text{ and } d = p_1 - p_2$$

"The value of zα is associated with the chosen level of significance, while the value of zβ is linked with the power of the test. For a significance level of 5%, zα equals 1.96, and for a test power of 80%, zβ equals 0.84".

The parameters considered in the calculation were the incidence of complications such as pneumothorax, vessel puncture, block failure.

By taking proportion p_1 = 59.4% and p_2 = 18.75% the acquired sample size was 23 in each group.

To ensure more definitive results the sample size was raised to 30

There would be two groups with size of 30 each.

Inclusion Criteria:	Exclusion Criteria:
• Age : 18yrs to 70yrs • ASA - I , II and III • Any elective procedures related to upper limb • Consent given	• ASA IV • BMI >25 Kg/m^2 • Any Coagulopathies • Any infection at the site of the injection • Any allergies related local anaesthetic.

Ethical Clearance: Before commencing the study, approval was obtained from the Institutional Ethical and Research Committee, J N M C, Belagavi.

Informed Consent:

Type of research and the intervention being done was explained to all of the patients who met the selection criteria.

Prior to enrollment, handwritten informed consent was obtained from every patient.

Study protocol:

Following approval from the ethics committee , obtaining written informed consent from patients, the study enrolled 60 patients scheduled for upper limb surgery under brachial

plexus block. Patients meeting the specified inclusion , exclusion criteria and consenting to participate were included .

By computerized randomization, patients are divided in to 2 groups

GROUP S: In this group block was performed with patients in **supine position.**

GROUP L: In this group block was performed with patients in **lateral decubitus** position.

Before the day of surgery, a comprehensive pre-anaesthetic evaluation and standard investigations were conducted. After confirming patient's nil-by-mouth status, intravenous access was secured using 18G or 20 G IV cannula. The patient was then shifted inside the operating theater, where standard equipment to monitor vital signs, non- invasive blood pressure., heart rate, ECG & pulsoximeter, were applied.

The patients were randomised in to one of the two groups to receive supraclavicular brachial plexus block .

GROUP S: In this group, patients are **positioned supine** with a recline of 30 degrees. A small pillow was positioned beneath the head, and the head was turned to the opposite side of the procedural site.

GROUP L : In this group, patients were**placed in a lateral decubitus position**, with the upper limb on the same side comfortably positioned infront of the chest wall. The

shoulder joint and the humerus were rotated towards the chest through adduction and internal rotation .

With strict adherence to aseptic protocols, a high-frequency linear probe was employed, positioned parallel to the clavicle at its midpoint. Identification of the brachial plexus, major artery like subclavian , pleural line, and 1st rib were conducted. The plexus was identified as a cluster of hypoechoic structures situated laterally, posteriorly, and superficially to the subclavian artery. "Distance from the apex of pleura and the inferior trunk of brachial plexus" was measured in 2 different positions in 2 different groups.

"Ultrasound - Guided Supra clavicular Brachial plexus blockade"

The SCBPB was carried out with patient position as per the group. The linear USG probe was positioned at coronal or oblique plane just above clavicle at around midpoint. Trunks, divisions of brachial plexus trunks and divisions are visualized superiorly to the 1st rib lateral to subclavian artery. The 23G needle was introduced under vision by in-plane technique, and in a lateral-to-medial direction under the visualization of ultrasound. 20cc of a mixture of equal volume (2%)lidocaine preserved with (1:200,000) epinephrine and bupivacaine(0.5%) is injected .The drug's dispersion within the brachial plexus and its subsequent centrifugal distribution into the trunks and divisions was observed.

The following "parameters" were measured in both the groups

The "**Distance between pleura and the Brachial plexus**" is measured from apex of pleura to the lower trunk of brachial plxus

The procedure time for the block was determined by recording the time taken from identifying the brachial plexus, inserting the needle and injecting the drug around the plexus.

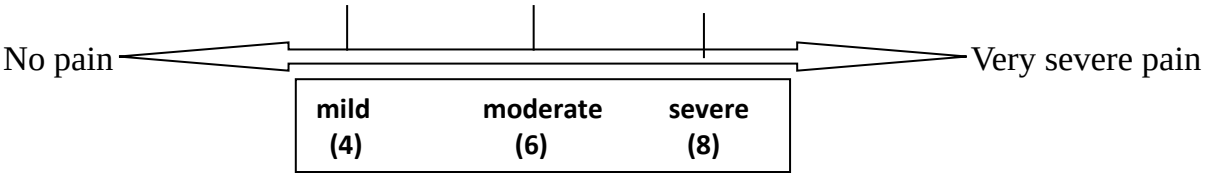
Success of the block was evaluated by achieving comprehensive surgical anesthesia, which encompasses both sensory and motor blockade.

Postoperative pain relief in patients was monitored in the post-anesthesia recovery unit, as well as upon the restoration of motor function by observing the movement of fingers.

The patients was monitored for vital parameters heart rate, NIBP, SPO2 and VISUAL

ANALOG SCORE.

In either group ,VISUAL ANALOG SCORE.



If , Visual-Analog Scale (VAS) score, was more than 4, intravenous paracetamol at a dose of 20 mg/kg was administered as a rescue analgesic.

If the Visual-Analog Scale (VAS) score, exceeds 4 within 6 hours of administering paracetamol, IV Diclofenac sodium, as a second rescue analgesic, 1.5 mg/kg, was given when required.

The patients was monitored for 24hrs post operatively for VAS scores.

Complications-Patient were monitored for problems both during surgery like vascular injury, development of hematoma and for 24 hours following surgery for any injury to nerve, pnemothorax, Horner's syndrome, diaphragmatic paralysis.

Statistical Analysis:

The study analysis to compare two groups was done as follows. Mean and standard deviation was computed for continuous quantitative variables. Inter-group continuous variables were compared using suitable statistical techniques such as the unpaired Student's t-test. Within-group comparisons of two quantitative variables were analyzed using "Student's paired t-test".

Presentation of categorical data was done by using rates, ratios, and percentages. "Chi-square test or Fisher's exact test" was utilized to evaluate the association among the outcome and clinical as well as demographics of patients

Median was utilized to represent discrete variables.

Nonparametric tests were utilized for comparing discrete variables.

Appropriate graphs were utilized to illustrate the comparison

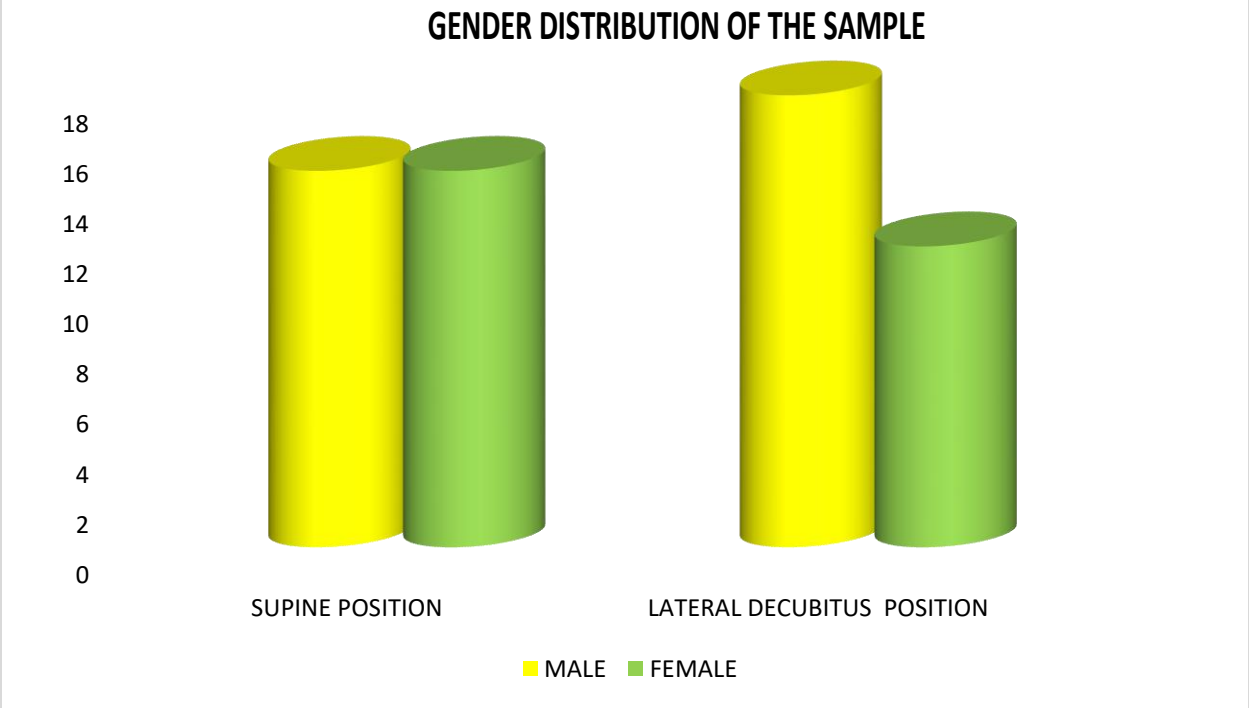
In all tests, a significance level of less than 5% (0.05) was deemed significant.

RESULTS

PARAMETER	SUPINE POSITION		LATERAL DECUBITUS POSITION	
GENDER	NUMBER	%	NUMBER	%
MALE	15	50	18	60
FEMALE	15	50	12	40
TOTAL	30	100	30	100

Table 1
" GENDER DISTRIBUTION" OF THE SAMPLE

GRAPH 1



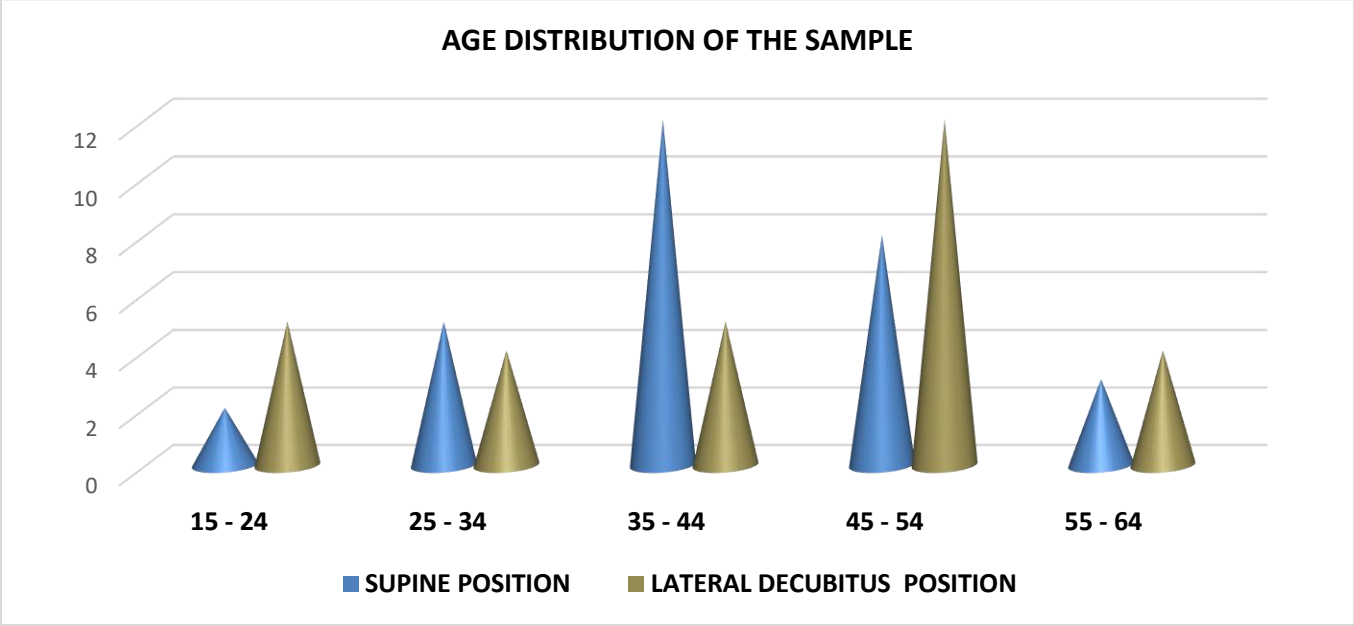
There was no extreme variation in gender distribution in Group S and Group L in our study.

	SUPINE POSITION		LATERAL DECUBITUS POSITION	
AGE	**NUMBER**	**%**	**NUMBER**	**%**
15 - 24	2	6.67	5	16.67
25 - 34	5	16.67	4	13.33
35 - 44	12	40.00	5	16.67
45 - 54	8	26.67	12	40.00
55 - 64	3	10.00	4	13.33
TOTAL	**30**	**100**	**30**	**100**

Table 2

"AGE DISTRIBUTION" OF SAMPLE

GRAPH 2



The mean age of Group S (48.63 years) was slightly higher but comparable to that of Group L (42.73 years). Group L had a minimum age of 18 years, slightly lower than Group S's minimum of 19 years, while the maximum age was 66 years for both groups S and I.

	SUPINE POSITION				LATERAL DECUBITUS POSITION					
	MEAN	S.D	MIN	MAX	MEAN	S.D	MIN	MAX	P VALUE	INFERENCE
AGE	48.63	13.47	19	66	42.73	13.50	18	66	0.0955	NS
BMI	22.6	1.74	19.6	24.8	21.77	1.76	18.8	25	0.0713	NS

Table 3

" DEMOGRAPHIC DATA" OF THE SAMPLE

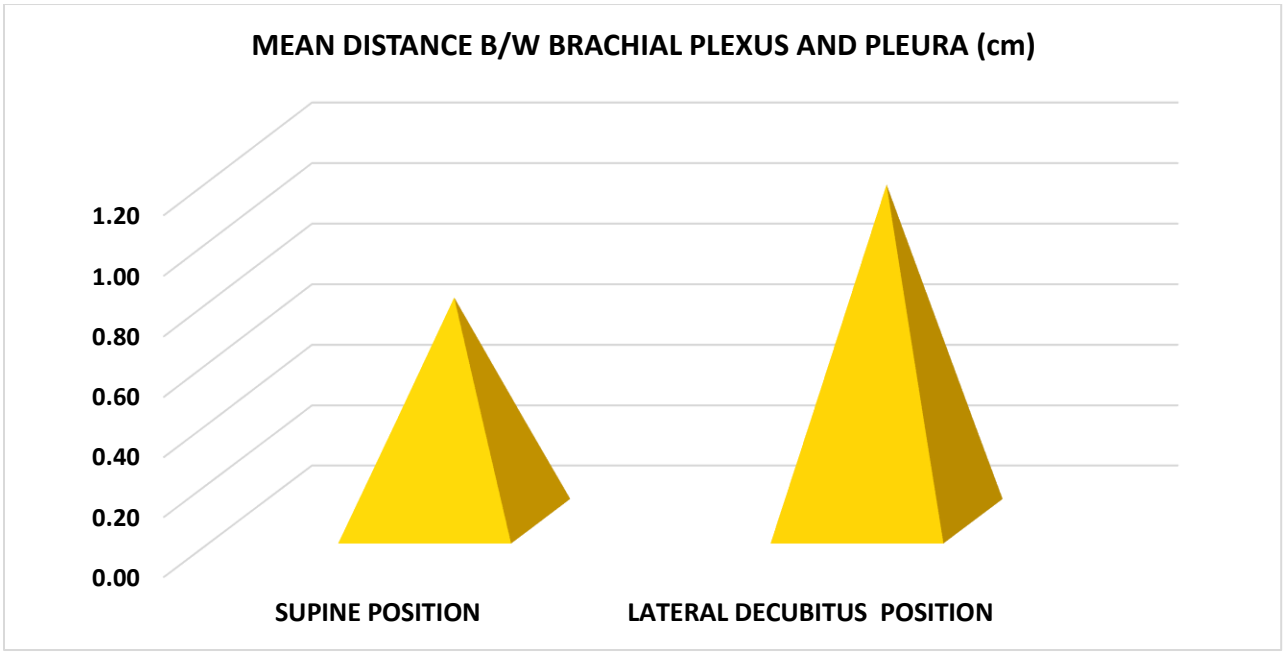
The average BMI of Group S (22.6 kg/m^3) closely matched that of Group L (21.77 kg/m^3). Group L had a minimum BMI of 18.8 kg/m^3, slightly lower than Group S's minimum of 19.6 kg/m^3. The maximum BMI observed was 24.8kg/m^3 for Group S and 25 kg/m^3 for Group I.

The age, gender, BMI, ASA grades, are comparable as the p value shows >0.005

	SUPINE POSITION				LATERAL DECUBITUS POSITION					
	MEAN	S.D	MIN	MAX	MEAN	S.D	MIN	MAX	p VALUE	INFERENCE
DISTANCE B/W BRACHIAL PLEXUS AND PLEURA (cm)	0.74	0.17	0.48	1.1	1.12	0.18	0.82	1.47	< 0.0001	HS

Table 4

MEAN DISTANCE B/W BRACHIAL PLEXUS AND PLEURA (cm)



GRAPH 3

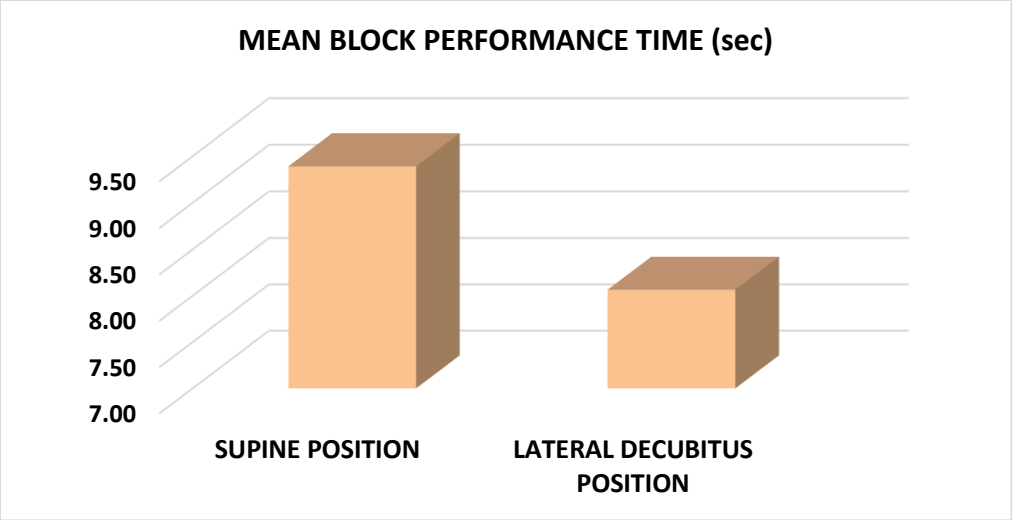
In the present study the mean distance b/w brachial plexus and pleura in group L is 1.12 cm with range varies from 0.82 cm to 1.47 cm and in the group S having the mean distance b/w brachial plexus and pleura is 0.74 cm with range varies from 0.48 cm to 1.1 cm showing statistically high significance with P valve < 0.0001.

"BLOCK PERFORMANCE TIME"

	SUPINE POSITION				LATERAL DECUBITUS POSITION					
	MEAN	**S.D**	**MIN**	**MAX**	**MEAN**	**S.D**	**MIN**	**MAX**	**p VALUE**	**INFERENCE**
BLOCK PERFORMANCE TIME (min)	9.39	2.46	5.6	14.6	8.07	1.55	5.56	13	< 0.0001	HS

Table 5

COMPARISON OF "TIME TAKEN TO PERFORM BLOCK"



GRAPH 4

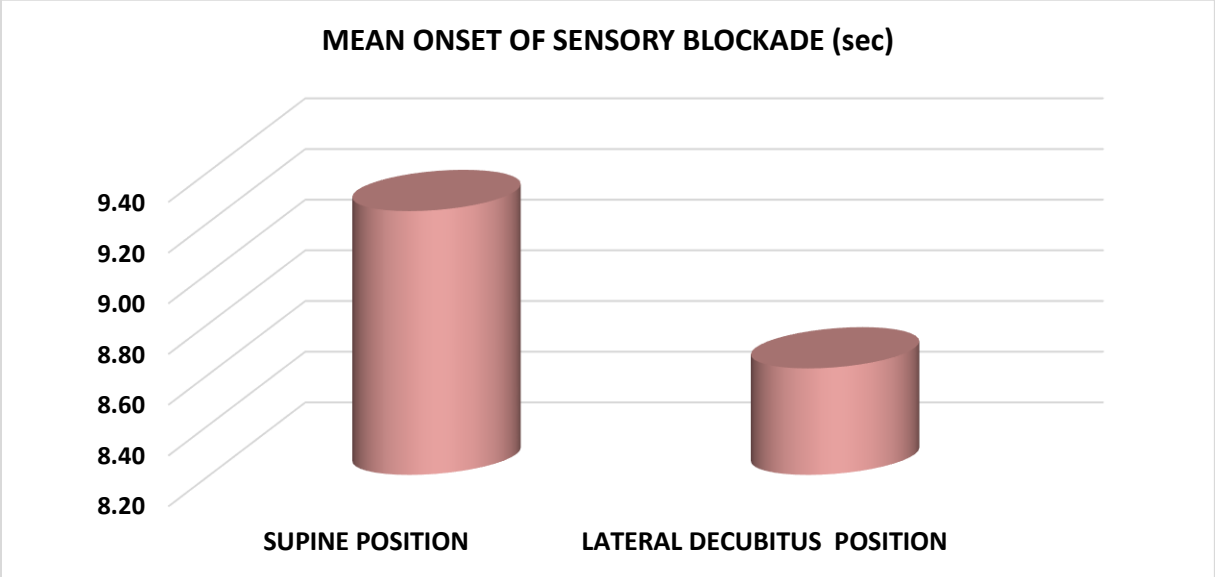
In present study mean time taken to perform block in Group L was 8.07mins and in Group S was 9.39mins and it was significant statistically. In Group L, the shortest time recorded was 5.56 minutes, while in Group S, it was slightly longer at 5.6 minutes. The maximum time taken was 14.6 mins in "Group S" and 13 mins in "Group L".

"ONSET OF SENSORY BLOCKADE"

	SUPINE POSITION				LATERAL DECUBITUS POSITION					
	MEAN	S.D.	MIN	MAX	MEAN	S.D.	MIN	MAX	p VALUE	INFERENCE
ONSET OF SENSORY BLOCKADE (min)	9.24	0.94	7.98	11.12	8.62	0.68	7.49	9.91	0.0049	VS

Table 6

GRAPH 5



COMPARISON OF "ONSET OF SENSORY BLOCKADE TIME"

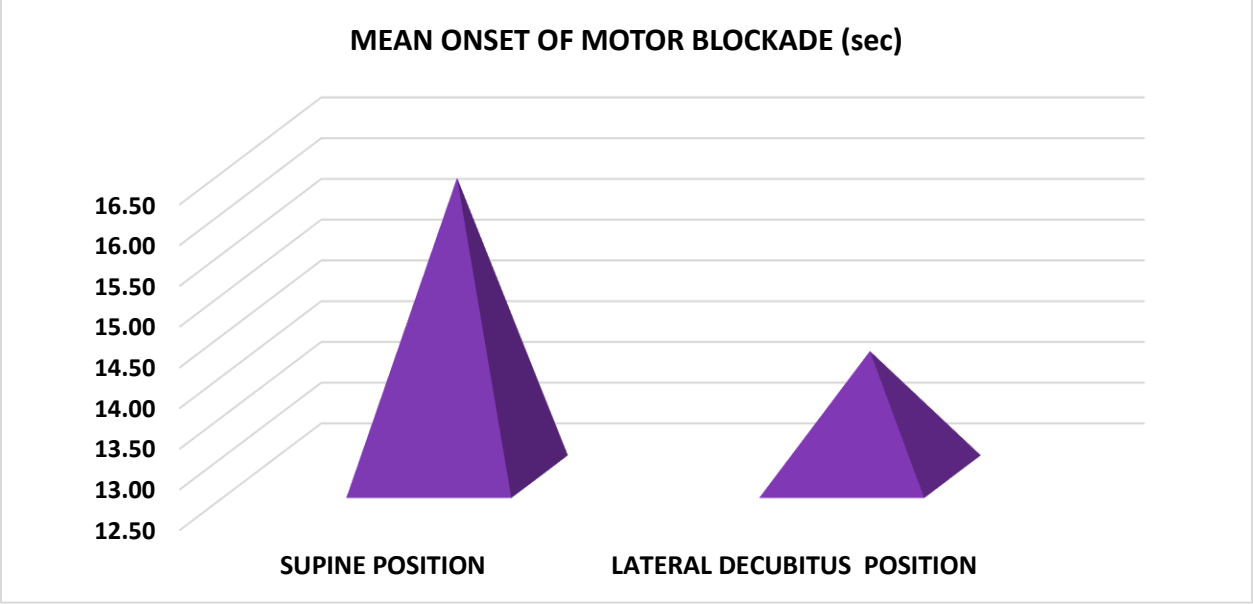
Mean Sensory onset time was early in Group L with mean of 8.62mins and in Group S 9.24mins and was significant statistically. This can be explained in view of increased distance between pleural apex & lower trunk of brachial plexus, facilitates easier needle access to the intended site and rapid injection of anaesthetic drug. Minimum time for onset in "Group L"& "Group S" was 7.49 mins and 7.98min respectively and maximum time for onset in Grp L was 9.91 mins & in Grp S was 11.12mins.

"ONSET OF MOTOR BLOCKADE"

	SUPINE POSITION				LATERAL DECUBITUS POSITION					
	MEAN	S.D	MIN	MAX	MEAN	S.D	MIN	MAX	p VALUE	INFERENCE
ONSET OF MOTOR BLOCKADE (min)	16.17	2.55	11.45	20.4	14.04	1.82	11	16.88	0.0005	HS

Table 7

GRAPH 6



COMPARISON OF "ONSET OF MOTOR BLOCKADE"

Mean Motor onset duration was early in Group L with mean of 14.04 mins than Group S which was 16.17 mins and was statistically significant. The minimum onset time in Group L was 11mins and Group S was 11.45 mins and maximum onset time in Group L and Group S was 16.88mins and 20.4 mins

Table 8

	INTRA OPERATIVE COMPLICATIONS			
	SUPINE POSITION		LATERAL DECUBITUS POSITION	
	NUMBER	%	NUMBER	%
VESSEL PUNCTURE	3	10.00	1	3.33
NIL	27	90.00	29	96.67
TOTAL	30	100.00	30	100.00

FOR THE ABOVE TABLE THE VALUE OF p USING CHI-SQUARE TEST , APPLYING YATE'S CORRECTION, IS 0.6048(NS).

THERE IS NO SIGNIFICANT DIFFERENCE IN THE NUMBER OF INTRA OPERATIVE COMPLICATIONS.THERE ARE NO POST OPERATIVE COMPLICATIONS IN EITHER OF THE GROUP

DISCUSSION

The utilization of ultrasound guidance has revolutionized the practice of anesthesia, rendering various nerve blocks notably safer and more precise in contemporary times. Among these, the brachial plexus block through supra clavicular approach has emerged as a most common alternative to general anesthesia for surgeries on the upper limb , providing equivalent levels of surgical anesthesia while minimizing the need for multiple medications and mitigating the diverse complications associated with general anesthesia.

This block can also be performed with ease in patients under any ASA grade with very little exceptions like coagulopathies , drug allergies. It can be considered as one of the best choice of anaesthesia in conditions like, high cardiac risk patients.

Compared to the traditional anesthesia practices like landmark techniques and nerve stimulator techniques ,performing USG guided blocks increased safety, accuracy and decreased various complications like vascular ,nerve injury and drug related toxicity by decreasing the requirement.

Supraclavicular brachial blocks are performed at the distal trunks and proximal divisions. At this level, the plexus is more superficial, making the block easier to execute. Plexus at this level are more compact, hence a small amount of local anesthesia is sufficient to achieve reliable anaesthesia.

Pneumothorax, phrenic nerve paralysis, vascular tissue puncture, and even Horner syndrome are among the most common consequences. Although ultrasound guidance has been demonstrated to enhance the safety of the brachial plexus block thriugh supra clavicular approach, traditional supine position approach has been found to have a 0.5 to 5% incidence of problems such as pneumothorax.. This incidence can be explained in relation to the distance between the pleura and the inferior trunk of the plexus.

In this present study we compared ultrasound-guided blockade of brachial plexus through supraclavicular approach in two distinct positions involving 60 patients randomly assigned to 2 groups : "Group S" (supine position) and "Group L" (lateral decubitus position).

In present study there was no extreme variation in gender distribution in Group S and Group L in our study.

In this present study the average age of Group S (48.63 years) was slightly higher but comparable to that of Group L (42.73 years). Group L had a minimum age of 18 years, slightly lower than Group S's minimum of 19 years, while the maximum age was 66 years for both groups S and I.

In this present study the average BMI of Group S (22.6 kg/m^3) was almost similar to that of Group L (21.77 kg/m^3). In Group L, the lowest BMI observed was 18.8 kg/m^3, while in Group S, it was slightly higher at 19.6 kg/m^3. The highest BMI recorded was

24.8 kg/m^3 for Group S and 25 kg/m^3 for Group I. The demographic profile were comparable in view of age , gender, BMI , ASA grades.

In the present study the mean distance b/w brachial plexus and pleura in group L is 1.12 $\pm$ 0.31cm with range varies from 0.82 cm to 1.47 cm and in the group S the mean distance b/w brachial plexus and pleura is 0.74$\pm$ 0.35 cm with range varies from 0.48 cm to 1.1 cm showing statistically high significance with P valve < 0.0001. This suggests that the distance was more in group L than group S.These finding was similar in the perspective of increase in the distance b/w brachial plexus and pleura in the study conducted by Carl PC Chen et al . A potential explanation could be that in the "lateral decubitus" position , the humeral head undergoes internal rotation, the shoulder gets adducted, with the upper limb on the side where the anaesthetic blockade was planned was comfortably positioned against the chest wall. The brachial plexus may get shifted upward due to decreased tension on it as well as the effect of gravity. According to Mihara et al., the brachial plexus's traction and strain forces are affected by specific spine and shoulder placement angles.

In present study mean time taken to perform block in Group L was 8.07mins and in Group S was 9.39mins and it was significant statistically. The shortest duration recorded was 5.56 mins in "Group L" and 5.6 mins in "Group S". The maximum time was 14.6mins group S.and 13min in group L. This indicates that the time period for executing the block was relatively shorter in the L grp when compared to the S grp. No study Compared these 2 positions under US guided SCBPB , in relation to block performance

time . Results from other studies carried out by Amit Vadhel et al. & by Prasad PK et al. utilizing the landmark technique with nerve stimulator contradict our findings.

In this study, the average onset time for anesthetic sensory blockade was faster in "Group L", with an average of 8.62 minutes, compared to 9.24 mins in "Group S", and this "difference in time was statistically significant". Minimum time for onset in "Group L" and "Group S" was 7.4 mins and 7.98min respectively and maximum time for onset in Grp L was 9.91 mins & in Grp S was 11.12mins. In this present study mean Motor onset time was early in Group L with mean of 14.04 mins than Group S which was 16.17 mins and was statistically significant. The minimum onset time in Group L was 11mins and Group S was 11.45 mins and maximum onset time in Group L and Group S was 16.88mins and 20.4 mins. This can be explained in view of increased distance between pleural apex & lower trunk of brachial plexus, facilitates easier needle access to the intended site and rapid injection of anaesthetic drug. The study conducted by Anjana Sahu et al. utilizing a nerve stimulator exhibited outcomes that are consistent with the results observed in our current study. In the studies, carried out by Amit Vadhel et al. and by Prasad PK et al., utilizing the landmark technique with a nerve stimulator, no disparity in sensory , motor onset time were observed.

In this present study there were no significant intraoperative or postoperative complications majorly pneumothorax in any of the designated positions which was occured in Anjana Sahu et al study which performing block in convenctional supine approach but which was conducted using nerve stimulator.

Various other complications like vessel puncture, horners syndrome,phrenic nerve nerve was very negligable compared to other studies but those studies are majorly conducted without using ultrasound.

There are few limitations in our study;

1) It is a single centered study with small sample size needed multicentric studies with large sample size as there was not much reasearch done in USG guided supraclavicular bachal plexus block in lateral decubitus postion.
2) Performer bias – different performers for different patients although all the performers are skilled and experienced anaesthesiologists
3) Patients with high Body mass index was not taken in our study .

CONCLUSION

To conclude in the distance between pleura and brachial plexus is more and block performance time is less in Lateral decubitus position when compared to the conventional supine position,and hence can be the better viable option for supraclavicular brachial plexus block.

SUMMARY

60 patients belonging to "American Society of Anaesthesiologists" grade I , II & III who were electively indicated for surgeries involving upper-limb were divided randomly into "Group S"(conventional supine position) and "Group L"(lateral decubitus position), by computerised randomisation method. patients in both groups underwent a brachial plexus block using USG with 20cc of a mixture of equal volume 2% lidocaine and 1:200,000 epinephrine bupivacaine 0.5% .

Following " parameters were measured" in present study

- The distance between pleura nad brachial plexus
- Duration required to perform the procedure
- The loss of Sensory component due to block
- The loss of motor component due to block
- Complications.

In present study the observations are as follows-

- The distance between the pleura and brachial plexus was greater in "group L" compared to "group S", and this difference has statistically importance .
- The duration required to perform the BPB through supraclavicular approach in "Group S "was longer compared to "Group L", and this difference has statistically significance.
- Loss of sensory, motor components were observed to be faster in the supraclavicular brachial plexus block in Group L in comparison to Group S.

- The effectiveness of both the blockades was similar with not much difference and complications .

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ANNEXURE I -

INFORMED CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr. /Mrs. /Miss. _____, we are requesting you to enroll yourself in the study titled **"COMPARISION BETWEEN LATERAL DECUBITUS POSITION AND CONVENTIONAL SUPINE POSITION TO DETERMINE THE DISTANCE BETWEEN PLEURA AND BRACHIAL PLEXUS IN PATIENTS UNDERGOING ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES -A RANDOMISED CONTROL TRIAL."**

- Respected Sir/Madam, we request you to participate in our study as you are eligible for it. During the study you will be asked some questions regarding your medical history and you are supposed to answer to the best of your knowledge.
- Your participation in this research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N.Medical College. If you decide to participate you are free to withdraw at any time.
- **Objective:** The study is to compare the distance between pleura and brachial plexus in lateral decubitus and conventional supine positions to assessthe safety of ultrasound guided supraclavicular brachial plexus block.
- **Introduction:**The study involvesultrasound guided supraclavicular brachial plexus in 2 different positions in 2 different groups to assess the safety of the blockby preventing the serious complication such as pneumothorax following the block.

- **Explanation of procedure:**If you agree to enrol in my study, I will ask you present, past and family history. Then you will be clinically examined in detail. you will then be positionedin one of the two different positions(lateral decubitus/supine)under ultrasound guidance the drug will be injected in the same position to perform the supraclavicular block and observed for the results.
- **Withdrawal from participation in the study:** Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.
- **Possible benefits from participating in the study:** You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.
- **Possible risks from participating in the study:** There are no risks involved in participating in this study.
- **Privacy and confidentiality:** The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.
- **Financial incentives:** You will not receive any payment for participating in this study.

- **Authorization for publication of aggregated data:** Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.
- **Questions:** In case if you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.
- **Legal rights:** By signing this consent form, we are not waving any of your legal rights.

INFORMED CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

I am making a voluntary decision to participate in the study **"COMPARISION BETWEEN LATERAL DECUBITUS POSITION AND CONVENTIONAL SUPINE POSITION TO DETERMINE THE DISTANCE BETWEEN PLEURA AND BRACHIAL PLEXUS IN PATIENTS UNDERGOING ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES -A RANDOMISED CONTROL TRIAL"**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE - II - PROFORMA

"**COMPARISION BETWEEN LATERAL DECUBITUS APPROACH AND CONVENTIONAL SUPINE APPROACH TO ASSESS THE SAFETY IN ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR DISTAL UPPER LIMB SURGERIES: A RANDOMISED CONTROL TRAIL**".

Group allotted :**L/S**

Name : Age :

Gender : Weight :

Height : Date of Examination :

Address : Occupation :

Pre examination evaluation

Past History :HTN DM IHD Arrhythmia Valvular heart diseases .

General physical examination

Weight (Kg) : Height (in cm): BMI:

Temperature (^{0}F): Pallor: Cyanosis:

Icterus: Clubbing:

PR : BP: RR: spo2 :

Systemic examination:

RS : CNS :

CVS : GIT :

Preoperative physical status **ASA STATUS I II III IV**

Proposed Surgery:

Pre-Operative baseline values:

HR: BP: SpO2:

Monitors Attached:

Pulse Oxymeter: NIBP: ECG:

Position of the patient :

Distance between pleura and brachial plexus :

Block Performance Time:

Sensory Blockade:

	Time(In Minutes)
a)Onset of Sensory blockade	
b)Time taken for complete blockade	
c)Total duration of blockade	

Motor Blockade:

	Time(In minutes)
a)Onset of Motor Blockade	
b)Time taken for complete blockade	
c)Total duration of Blockade	

Duration of Analgesia :

Intraoperative Complications- YES NO

Postoperative Complications- YES NO

Signature of the Anaesthesiologist-

Signature of the Witness-

Signature of Chief Investigator-

ANNEXURE III- PHOTOGRAPHS

PHOTOGRAPH 1: USG machine

PHOTOGRAPH 2: Linear ultrasound probe

PHOTOGRAPH 3: Patient and USG machine Position

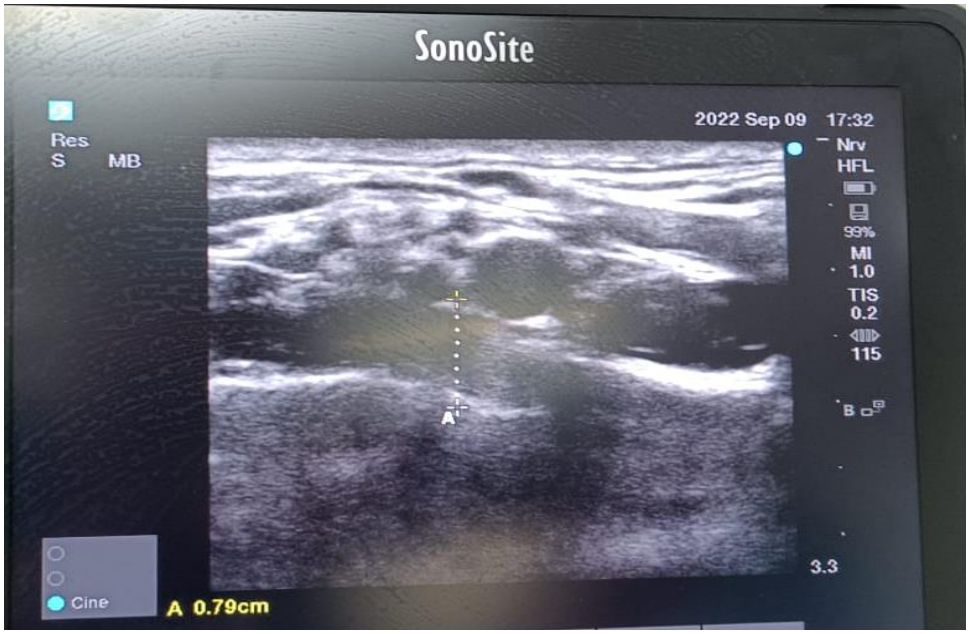
(CONVENTIONAL SUPINE POSITION)

PHOTOGRAPH 4: Distance from the pleura to the brachial plexus

(CONVENTIONAL SUPINE POSITION)

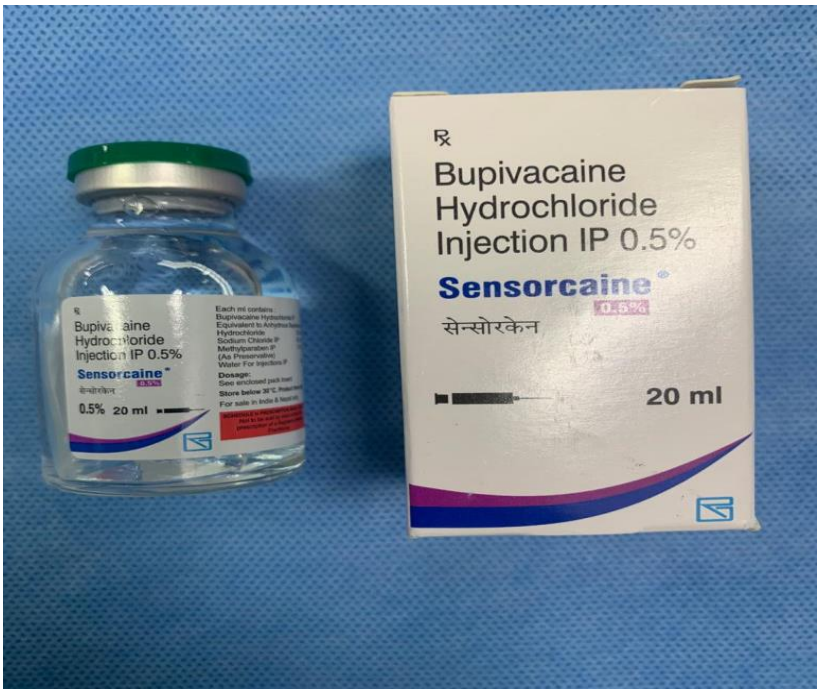
PHOTOGRAPH 5: Patient and USG machine Position

(LATERAL DECUBITUS POSITION)



PHOTOGRAPH 6: Distance from the pleura to the brachial plexus

(LATERAL DECUBITUS POSITION)



PHOTOGRAPH 7 : 0.5% Bupivacaine

PHOTOGRAPH 8 : 2% Lignocaine and Adrenaline (1:200000)

ANNEXURE IV- MASTER CHART

SUPINE POSITION											
S. NO		**AGE (yr)**	**SEX**	**BMI (kg/ m^2)**	**ASA**	**DISTANCE B/W BRACHIAL PLEXUS AND PLEURA(cm)**	**BLOCK PERFORMANCE TIME(min)**	**ONSET OF SENSORY BLOCKADE(min)**	**ONSET OF MOTOR BLOCKADE(min)**	**INTRA OPERATIVE COMPLICATIONS**	**POST OPERATIVE COMPLICATIONS**
1		65	M	21.9	II	0.53	12.2	8.23	16.55	nil	nil
2		28	M	20.6	I	0.58	11.4	8.91	17.43	NIL	NIL
3		30	f	24.4	I	0.77	9.2	11.12	20.24	nil	nil
4		21	M	19.6	I	0.92	7.5	9.35	18.54	nil	nil
5		60	M	24.2	III	0.63	11.82	9.78	18.23	nil	nil
6		51	M	24.8	II	0.55	13.89	9.11	17.56	nil	nil
7		19	M	19.8	I	0.69	10.8	10.22	14.57	VESSEL	nil
8		34	m	22.4	II	0.88	7.6	7.98	11.93	nil	nil
9		62	m	24.8	III	0.48	14.6	8.11	12.41	nil	nil
10		46	f	23.9	I	1.1	5.89	9.39	16.45	nil	nil
11		66	f	24.1	III	0.53	10.9	9.85	18.33	nil	nil
12		46	m	22.6	II	0.74	10.1	9.5	17.43	nil	nil
13		44	m	23.1	I	0.91	8.14	9.35	11.93	nil	nil
14		56	f	24	I	0.69	8.85	7.98	18.3	nil	nil
15		59	m	20.2	II	0.71	9.1	9.78	14.17	nil	nil
16		64	f	23.2	III	0.99	6.56	9.31	20.4	VESSEL	nil
17		56	m	22.6	II	0.75	8.12	8.33	17.43	nil	nil
18		58	f	24.4	II	0.85	8.56	11.12	15.71	nil	nil
19		53	f	22.8	II	0.52	11.2	7.98	16.15	nil	nil
20		52	f	23.6	III	0.63	10.9	9.11	14.7	nil	nil
21		42	f	20.6	I	0.5	13.5	9.05	18.23	nil	nil
22		50	f	20.6	I	0.56	10.6	10.52	17.43	nil	nil
23		47	m	20.1	III	0.96	5.9	9.71	11.93	nil	nil
24		30	m	21.1	II	0.73	8.4	8.2	16.24	VESSEL	nil
25		52	f	24.3	I	0.88	7.3	9.15	14.37	nil	nil
26		52	f	22.3	III	0.77	7.4	7.98	16.65	nil	nil
27		34	m	19.8	I	0.68	11.3	9.11	17.63	nil	nil
28		54	f	23.9	II	0.74	8.1	11	19.24	nil	nil
29		66	f	24.6	III	0.99	6.3	9.66	11.45	nil	nil
30		62	f	23.7	II	1	5.6	8.32	13.45	nil	nil
	TOTAL	48.633		22.6		0.742	9.391	9.240333333	16.16933333		

LATERAL DECUBITUS POSITION											
S.NO		**AGE**	**SEX**	**ASA**	**BMI (kg/m²)**	**DISTANCE B/W BRACHIAL PLEXUS AND PLEURA(cm)**	**BLOCK PERFORMANCE TIME(min)**	**ONSET OF SENSORY BLOCKADE(min)**	**ONSET OF MOTOR BLOCKADE(min)**	**INTRA OPERATIVE COMPLICATIONS**	**POST OPERATIVE COMPLICATIONS**
1		24	m	I	21.4	0.87	6.32	8.31	15.32	nil	nil
2		66	m	III	23.6	0.82	5.56	8.56	16.88	nil	nil
3		47	f	I	25	0.96	6.56	9.71	14.27	nil	nil
4		31	m	I	20.2	1.21	8.12	7.79	11.43	nil	nil
5		24	m	II	19.3	0.88	8.56	9.11	12.45	nil	nil
6		44	f	II	22.9	1.47	9.66	8.31	15.2	nil	nil
7		52	m	I	22.1	1.3	8.9	8.66	11.3	nil	nil
8		50	f	III	20.2	1.1	13	8.59	16.8	nil	nil
9		40	f	II	23.2	1.13	8.92	7.79	14.47	nil	nil
10		50	m	I	21.4	1.33	8.05	8.31	15.12	nil	nil
11		27	m	III	19.8	0.99	8.89	9.31	12.5	nil	nil
12		36	m	II	21.3	0.97	8.14	7.91	11.63	nil	nil
13		43	f	II	22.2	1.22	9.12	8.11	16.48	nil	nil
14		60	f	III	23.3	1.15	7.69	8.49	12.44	nil	nil
15		52	f	III	24.8	0.94	7.3	8.46	15.39	VESSEL	nil
16		49	m	I	19.6	1.3	8.3	9.73	14.7	nil	nil
17		20	m	I	18.8	1.11	8.2	9.1	13	nil	nil
18		27	m	II	20.1	1.4	6.23	7.7	16.5	nil	nil
19		18	m	II	19	0.98	6.68	8.3	11	nil	nil
20		30	m	III	22.3	1	7.12	8.54	15.45	nil	nil
21		55	m	II	21.3	1.36	7.11	9.78	13.61	nil	nil
22		54	m	I	24.2	1.2	5.77	9.16	12.1	nil	nil
23		23	m	I	21.1	0.98	7.33	8.61	14.5	nil	nil
24		49	f	II	20.7	0.89	6.14	7.49	13.11	nil	nil
25		48	m	I	22.1	1.35	7.78	8.66	15.2	nil	nil
26		64	f	III	24.2	1.1	10.56	9.91	11.73	nil	nil
27		50	m	II	20.8	1.38	9.22	8.36	16.55	nil	nil
28		44	f	I	21.4	0.96	8.6	9.6	12.43	nil	nil
29		52	f	II	24.2	1.13	8.67	7.7	15.2	nil	nil
30		53	f	II	22.6	0.97	9.45	8.52	14.56	nil	nil
	TOTAL	43			21.77	1.115	8.065	8.619333333	14.044		

ANNEXURE – V - KEY TO MASTER CHART

ASA	American society of Anaesthesiologists (Grades I -III)
cm	Centimeter
kg/m^3	Kilogram/meter cube
min	Minutes
yr	year
BMI	Body mass index