"A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES"

By

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Under the guidance of

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i

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ACKNOWLEDGEMENT

ABBREVATION

ASA   - American Society of Anaesthesiologist

ABPB   - Axillary brachial plexus block

BP   - Blood Pressure

ECG   - Electrocardiogram

gm   - gram

Hrs   - Hours

Kg   - Kilogram

LA   - Local Anaesthetic

µg   - Microgram

McN   - Musculocutaneous nerve

MN   - Median nerve

Min   - Minutes

ml   - Millilitre

NS   - Not significant

PN   - Perineural

PV   - Perivascular

RN   - Radial nerve

SCB   - Supraclavicular block

S.D.   - Standard Deviation

S.E.   - Standard E

UN   - Ulnar nerve

USG   - Ultrasonography

ABSTRACT

INTRODUCTION: Axillary brachial plexus block (ABPB) is safest among other methods of brachial plexus block because of its ease & reliability. The two approaches of USG guided ABPB are perivascular (PV) and perineural (PN).

Aim: This study was conducted to compare technique of USG guided perivascular and perineural ABPB for upper limb surgeries.

Objectives: Comparison of perivascular and perineural USG guided ABPB with aid of primary outcomes like performance & onset time, block success rate. Secondary out-comes are duration of sensory and motor block, number of needle passes, and adverse events.

SUBJECTS: This prospective randomized study was conducted on 106 patients ASA I & II posted for forearm, wrist and hand surgeries, who were allotted into Group I (PV) & Group II (PN) 53 each.

METHODS: In both methods, volume of drug used was 20 ml. The drugs used were 0.5%bupivacaine 8ml, 2% lignocaine with adrenaline 10ml and dexamethasone 2ml. In both methods, musculocutaneous nerve was blocked with 5ml. In perivascular technique, remaining 15ml volume of the drug was deposited anterior and posterior to axillary artery and in perineural technique 5ml of drug was injected around radial, ulnar, median nerve. The primary and secondary outcomes were noted. Mann-Whitney & Chi-Square test tests were used for statistical analysis.

RESULTS: Significant difference was observed between the two groups in performance time (PV-8.647±0.54min & PN-14.53±0.20min; p>0.0001), onset time (PV-19.48±2.83min & PN-13.86±1.81min; p>0.0001) and number of needle passes (PV-2.30±0.50 & PN-4.91±0.66; P>0.0001). Other parameters were comparable in both the groups.

CONCLUSION: USG guided perivascular ABPB is a simple technique compared to USG guided perineural ABPB as we have to identify only one structure in the

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perivascular ABPB. USG guided Perivascular ABPB is better than USG guided perineural ABPB in imaging time, needling time and performance time, but onset time was shorter in perineural block. The PV technique provides a safe alternative for PN USG guided-ABPB in patients with anatomical variation.

KEY WORDS**:** Ultrasonography, Brachial Plexus, Nerve Block

INDEX

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

In the history of anaesthesia, Halstead and Hall developed the technique of peripheral nerve blockade by describing the injection of cocaine into the ulnar, supratrochlear, musculocutaneous nerves in the 1880's. James Leonard Corning used Esmarch bandage in 1885 to arrest the local circulation, prolong the duration of block and decrease the uptake of local anaesthetic from tissues.[1]

Brachial plexus blocks provide a useful alternative to general anaesthesia for upper limb surgeries. They achieve near ideal operating condition by producing complete muscular relaxation and maintaining stable intra-operative hemodynamics. The sympathetic block produced reduces postoperative pain, vasospasm and oedema. These blocks have gained popularity because of several advantages over general anaesthesia like reduced incidence of nausea and vomiting, early mobility, adequate pain relief, early discharge.[2][3]

Also the use of ultrasound in the field of anaesthesia has added newer dimensions to our whole anaesthetic aspect and patient care. Ultrasound has various uses in our field like putting guided central lines, bed side echo in critical care set up and giving ultrasound guided regional blocks. USG guided nerve blocks increase the success rate as structures are directly visualized as compared to peripheral nerve locator guided and blind techniques.[4]

The axillary approach to brachial plexus is most common because of its feasibility, ease and acceptability. It is commonly used for forearm, wrist and hand surgeries.[1]The use of ultrasound in giving axillary block has improved success rate and reduced complications . The right placement of the local anaesthetics (LA) near

the desired nerve defines the success and quality of the nerve block. Currently there exists two techniques for ultrasound (USG) guided axillary brachial plexus block (ABPB), namely Perineural (PN) and Perivascular (PV).

The perineural technique of axillary brachial plexus block involves identification of nerves with the aid of USG and injecting local anaesthetics in the perineurium of Radial Nerve (RN), Ulnar Nerve (UN), Median Nerve (MN) and Musculocutaneous nerve. On the other hand perivascular (PV) technique of axillary brachial plexus block involves localization of axillary artery with aid of USG followed by injection of LA around the axillary artery, for coverage of MN, UN and RN. [5,6,7] A separate injection for Musculocutaneous nerve is required since it is not in close proximity to the artery. The widely used method of ABPB is perineural technique but it has certain limitations like risk of direct nerve injury in USG guided block due to anatomical variations and operator skills. Since it requires needle repositioning, it may even increase chances of vessel damage and other complications.

Several studies have been conducted on PV technique, which conclude that one or two injections around axillary artery are highly successful method. It is faster, with fewer injections and less discomfort and pain associated with the procedure. The concerns regarding these two techniques that still remains questioned are their efficacy and the safety.

Hence the present study was conducted to know which technique was better in terms of performance time of the block, success rate of the block, complications during the block. The above parameters were compared between perivascular and perineural technique of brachial plexus block in upper limb surgeries.

AIMS AND OBJECTIVES OF THE STUDY

Comparison of technique of perivascular and perineural ultrasound guided axillary brachial plexus block with aid of following outcomes:

- PRIMARY OUTCOME:

  - Performance time of the block (imaging time + needling time)

  - Onset of block (time from removal of the needle to obtaining surgical anaesthesia)

  - Block success rate (defined as block adequate to perform surgery without the need for supplementary blocks or anaesthesia)

- SECONDARY OUTCOME:

  - Duration of sensory block

  - Duration of motor block

  - Number of needle passes

  - Incidence of adverse events

REVIEW OF LITERATURE

DOWN THE HISTORY LANE:

In **1855, Freidrich Gaedcke** [1828-1890] isolated the most potent alkaloid of coca plant "cocaine". The compound was named "erythroxyline" by him.. Cocaine remains the first drug to be used for regional anaesthesia. [8][9]

In **1884, Karl Koller** [1857-1944] an Austrian ophthalmologist instilled a 2% solution of cocaine into his own eye to test its effectiveness as a local anaesthetic by pricking the eye with needle.

Later, **William Halstead** [1852-1922] performed the first brachial plexus block. Halsted exposed the roots surgically under local infiltration and injected each of them with a small amount of dilute Cocaine (0.1%) interneurally under direct vision. Only about 0.5 ml of local anaesthetic was required to produce complete anaesthesia. [10]

HISTORY OF BRACHIAL PLEXUS BLOCK:

1911-1912, Kulenkampff described percutaneous supraclavicular approach for the first time. He pointed out that above the clavicle the plexus lie under the skin as it passes over the first rib and is accessible to a percutaneous technique. The midpoint of the clavicle and subclavian artery provide a constant landmark, most frequently at the point where external jugular vein intersects the clavicle. He performed this on himself first and used 5 ml of Novocain. Later he increased it to 10 ml and was able to obtain complete anaesthesia. Direction of the needle was backwards, inwards and downwards. He emphasized that the purpose of the technique was not to hit the rib but to find the trunks by eliciting paraesthesia. He said that the rib just prevented pleural penetration. He used 4 cm needle. [11,12]

In **1926, Livingston,** carried out Kulenkampff's technique without producing paraesthesia as soon as the deep cervical fascia had been penetrated, 30 ml of 2% procaine was injected. He wrote that the plexus and the artery are separated from the surrounding structures by a fascial investment. [11]

In **1940, Patrick** chose to lay down a "wall of anesthetic" through which the plexus pass in its course over the first rib, where 60-70 ml of solution was injected during 5-6 insertions. The technique became the "standard technique" of supraclavicular block, subsequently referred to by many as the "classical supraclavicular technique".[11]

In **1942 Knight** modified Patrick's technique. He made the three injections through three separate needle insertion, parallel to one another. For the first time he utilized a directly caudal direction of needle insertion.

In **1944, Murphey** used a single injection technique and used lateral border of anterior scalene muscle as the landmark and direction of needle insertion was caudal as with Knight's technique, not medial or dorsal, as with most other techniques.

In **1949, Bonica and Moore** utilized Kulenkampff's and Patrick's technique and developed a technique which began with utilizing the classical landmarks for direction of needle insertion and demanded a definite paraesthesia prior to first injection. Then continued as Patrick's technique and laid down a wall of anaesthetic solution by "walking the rib" and made multiple injections during each withdrawal of the needle.

By late 1940s, clinical experience with brachial plexus block during peacetime and wartime surgery was extensive, and new approaches of brachial plexus block began to be described.

In **1946**, **F. Paul Ansbro** described a continuous brachial plexus block technique for the first time. He described that by securing a needle in the supraclavicular fossa that was attached to tubing connected to a syringe, he could inject incremental doses of local anesthetic.[13]

In **1964, Winnie and Collins** were the first to describe subclavian perivascular block.[14] This approach became popular because compared to the traditional Kulenkampff approach it had lower risk of pneumothorax. This concept is based on axillary compartment formed by two muscles, containing nerves and vessels. When this compartment can be identified and entered by needle,the plexus is blocked by single needle. In **1977**, **Selander** described a technique of continuous brachial plexus block by using an intravenous catheter secured in the axilla. [15]

In **2006, Ali Movafegh *et al*.** conducted a study y to evaluate the effect of lidocaine with dexamethasone on the onset and duration of axillary brachial plexus block. 60 patients scheduled for elective forearm and hand surgery were randomly allocated to receive axillary brachial plexus block with either 34 mL lidocaine 1.5% with 2 mL of isotonic saline chloride (control group, n 30) or 34 mL lidocaine 1.5% with 2 mL of dexamethasone (8 mg) (dexamethasone group, n30).. After block performance, sensory and motor blockade of radial, median, musculocutaneous, and ulnar nerves were recorded at 5, 15, and 30 min. They concluded that the addition of

dexamethasone to lidocaine 1.5% solution in axillary brachial plexus block prolongs the duration of sensory and motor blockade.[16]

In **2007, Vincent W.S Chan *et al*.** conducted a study with the purpose to ascertain if real time ultrasound guided axillary brachial plexus block improves the success rate in patients undergoing elective hand surgery. They concluded that ultrasound guidance, with/without concomitant nerve stimulation, improves the success rate of axillary brachial plexus block. [17]

In **2007, Casati Andrea *et al*.** conducted a study to test the hypothesis that ultrasound guidance can shorten the onset time of axillary brachial plexus block when compared with nerve stimulation guidance with multiple injection technique.60 patients with ASA I to III receiving axillary brachial plexus block with 20 cc ropivacaine were randomly allotted to receive either nerve stimulation (NS group, n=30) or ultrasound guidance (US group, n=30) for nerve location. The onset of motor and sensory block, need for general anaesthesia, or insufficient block, procedure related pain, success rate and patient satisfaction were recorded. They found that the onset of sensory block was shorter in group US than in group NS whereas no difference was observed in onset of motor block. No failed block was reported in both groups. Thus, multiple injection axillary blocks with ultrasound guidance provided comparable success rates and incidence of complication as compared with nerve stimulator. [18]

In **2008, Pfeiffer *et al*.** conducted a study on perivascular axillary block with sonographic guidance. To improve the failure rates of blind block, it was combined with sonographic guidance. The success rate and time factor were determined in 86 people sample size. The rate of complete blocks without the use of sonography was

approximately 72%, whereas using transpectoral ultrasound it was 96.5%. No patient with transpectoral sonography required general anaesthesia. The onset time using sonography was approximately 6 min. The perivascular axillary plexus block with transpectoral sonography, is an effective and efficient procedure. [19]

In **2009, De Quang Hieu Tran** *et al*. conducted a prospective, randomized, observer-blinded study to compared ultrasound-guided supraclavicular (SCB), infraclavicular (ICB), and axillary (AXB) brachial plexus blocks for surgery of the elbow, forearm, wrist, and hand. They related times and block-related pain scores for the SCB, ICB, and AXB. [20]

In **2010, Imasogie** *et al***.** achieved successful block of the median, ulnar, and radial nerves by circumferential deposition of local anesthetic surrounding the axillary artery, instead of selectively blocking individual nerve. [21]

In **2012, De Q.H. Tran** *et al***.** conducted a study to compare double-, triple-, and quadruple-injection ultrasound (US) guided axillary brachial plexus block (AXB) for upper-extremity surgery. They concluded that double-, triple-, and quadruple-injection US-guided perivascular AXB result in comparable success rates and total anaesthesia related times. As it requires fewer needle passes, the double-injection provides a simple alternative for US-guided. [22]

In **2012, Francisca Bernucci** *et al*. did a prospective, randomized, observer-blinded study to compare perivascular (PV) and perineural (PN) USG guided axillary brachial plexus block (AXB) for upper extremity surgery. The conclusion was PV and PN ultrasound-guided AXBs result in comparable success rates and total anaesthesia-

related times. In view of fewer needle passes and a shorter performance time, the PV technique provides a simple alternative for ultrasound-guided AXB. [5]

In **2012, Karin P. W. Schoenmakers *et al.*** did a prospective randomized, observer-blinded trial to study effect of local anaesthetic volume (15 vs. 40 mL) on the duration of US-Guided single shot axillary plexus block. 30 patients were randomly allocated to receive ultrasound guided AXB with either 15 (group 15 mL, n = 15) or 40 mL (group40 mL, n = 15) mepivacaine 1.5%. Onset, efficacy, and duration of sensory and motor block were compared. The overall median duration of sensory and motor block was significantly shorter in group 15 mL. Duration of sensory and motor block of individual nerves was significantly shorter in group 15 mL. Time to first request of postoperative analgesia was also reduced in group 15 ml. There were no differences in the other block characteristics. Conclusions: In AXB with mepivacaine 1.5%, reducing the dose from 40mL to 15mL (62.5%) shortens the overall duration of sensory and motor block by approximately 17% to 19%, reduces sensory and motor block duration of individual nerves by 18% to 40%, and decreases the time to first request of postoperative analgesia by approximately 30%. [23]

In **2013, Andrea P. Gonza´lez *et al.*** did a study to ascertain the minimum effective volume of lidocaine 1.5% with epinephrine 5 ug/mL in 90% of patients (MEV90) for double-injection ultrasound-guided axillary block (AXB). 50 patients were included in the study. They concluded that for double-injection ultrasound-guided ABPB, the MEV90 of lidocaine 1.5% with epinephrine 5 ug/mL is 5.5 and 23.5 mL for musculocutaneous nerve and perivascular injection respectively. More studies are needed to determine other concentrations of lidocaine, other local anaesthetic agents and other techniques for ultrasound guided. [24]

In **2014 Cho S *et al*.** conducted prospective, randomized, observer-blinded trial to compare 2 double-injection perivascular (PV) USG-guided techniques of axillary brachial plexus block (ABPB). ASA grade I-II, 50 patients undergoing surgery of the forearm, wrist or hand were randomly allocated to two groups. For PV12 group, 24 ml of 2% lidocaine was injected at the 12 o'clock position to axillary artery. Patients of PV6 group 24 ml of 2% lidocaine was injected at 6 o'clock position of axillary artery. For both groups, the musculocutaneous nerve was identified, and 5 ml of 2% lidocaine was deposited around nerve. The performance and the onset time were noted. The induction time (sum of performance and onset time), the block success rate, the need for rescue block, and incidence of adverse events were compared. The success rate was same (84%) in two groups. There was no difference between two groups in terms of performance time, onset time, induction time, vessel puncture, paresthesia, and numbness. They concluded that Double-injection perivascular ultrasound-guided axillary brachial plexus block can be performed at 12 o'clock or 6 o'clock position of axillary artery, and needle targeting position can be choosen by the performer by taking into consideration the site of surgery. Thus, perivascular double-injection technique may be an alter native technique for axillary brachial plexus block useful in case of difficult block. [25]

In **2014, S. Choi *et al*.** did a systematic review and meta-analysis to assess the contemporary literature and quantify the effects of dexamethasone on Brachial plexus block. They searched for randomized, placebo-controlled trials which compared brachial plexus block performed with Local anaesthetic alone with that performed with Local anaesthetic and dexamethasone. Nine trials (801 patients) were in the meta-analysis with 393 patients receiving dexamethasone (4−10 mg). The analgesic duration for long-acting Local anaesthetic was

prolonged by Dexamethasone from 730 to 1306 min [mean difference 576 min, 95% confidence interval (CI) 522–631] and for intermediate from 168 to 343 min (mean 175, 95% CI 73–277). Motor block was prolonged from 664 to 1102 min (mean 438, 95% CI 89–787). The most recent study showed identical prolongation with perineural or systemic administration of dexamethasone compared to a placebo. With this study they arrived at a conclusion that Perineural administration of dexamethasone with LA prolongs Brachial plexus block effects with no evidence of adverse events. [26]

In 2016 Uday *et al.* did a prospective, randomized clinical study to compare perivascular and perineural ultrasound guided axillary brachial plexus block using levobupivacaine. They concluded that the PV technique provides a simple alternative for PN USG guided ABPB. [6]

ANATOMY OF BRACHIAL PLEXUS

The knowledge of the neural components to be blocked with their relationship to surrounding vascular, muscular structures and their ultimate motor and sensory innervations is necessary. This knowledge provides guidance to choose the most suitable technique for a particular surgery. The knowledge aids in identifying bony, vascular, muscular and fascial relationships which serve as landmarks to guide the needle or the USG probe to suitable site, thus improving the success of the block and reducing the adverse events.

The brachial plexus (Fig 1) is a network of nerve fibres, running from the spine It is formed by the ventral rami of the lower four cervical and first thoracic nerve roots (C5-C8, T1). The plexus proceeds through the neck, the axilla (armpit region), and then into the arm. It is a network of nerves passing through the cervico-axillary canal to reach axilla and innervates brachium (upper arm), antebrachium (forearm) and hand (Fig 2). The plexus may sometimes include C4 nerve root also, then it is called as pre-fixed and sometimes it may also include T2 nerve root, then it's called as post-fixed. [27,28]

The brachial plexus is divided into Roots, Trunks, Divisions, Cords, and Branches.

THE ROOTS

A pair of spinal nerves leave spinal cord (one left, one right) at level of each vertebra. This nerve then splits into posterior and anterior nerve fibres. The brachial plexus starts as the anterior fibres of the spinal nerves C5, C6, C7, C8 and T1. The roots of the brachial plexus are formed by these 5 nerves . The roots may be pre-fixed or post-fixed as described above.

THE TRUNKS

The trunks part of the brachial plexus is formed by three nerve fibres. The C5 and C6 roots converge together, and the C8 and T1 roots join.

These three trunks are named so for their anatomical position:

1. Superior trunk: By merging of C5 and C6 roots.

2. Middle trunk: A continuation of C7.

3. Inferior trunk: By merging of C8 and T1 roots.

THE DIVISIONS

Each trunk splits into two divisions. One division travels toward the front called the anterior division. The other travels towards the back called the posterior division. These divisions merge again in the next part of the brachial plexus.

THE CORDS

These six divisions regroup to form three cords. The cords are named with respect to their position with the axillary artery. The posterior cord is formed from the three posterior divisions of the trunks (C5-C8,T1) The lateral cord formed from the anterior divisions from the upper and middle trunks (C5-C7). The medial cord is simply a continuation of the anterior division of the lower trunk (C8,T1)

BRANCHES:

Posterior cord:

1. 1.Upper subscapular nerve

2. Lower subscapular nerve

3. Thoracodorsal nerve

4. Axillary nerve

5. Radial nerve

Lateral cord:

1. Lateral pectoral nerve

2. Musculocutaneous nerve

3. Lateral root of median nerve

Medial cord:

1. Medial pectoral nerve

2. Medial cutaneous nerve of fore arm

3. Medial cutaneous nerve of arm

4. Medial root of median nerve

5. Ulnar nerve
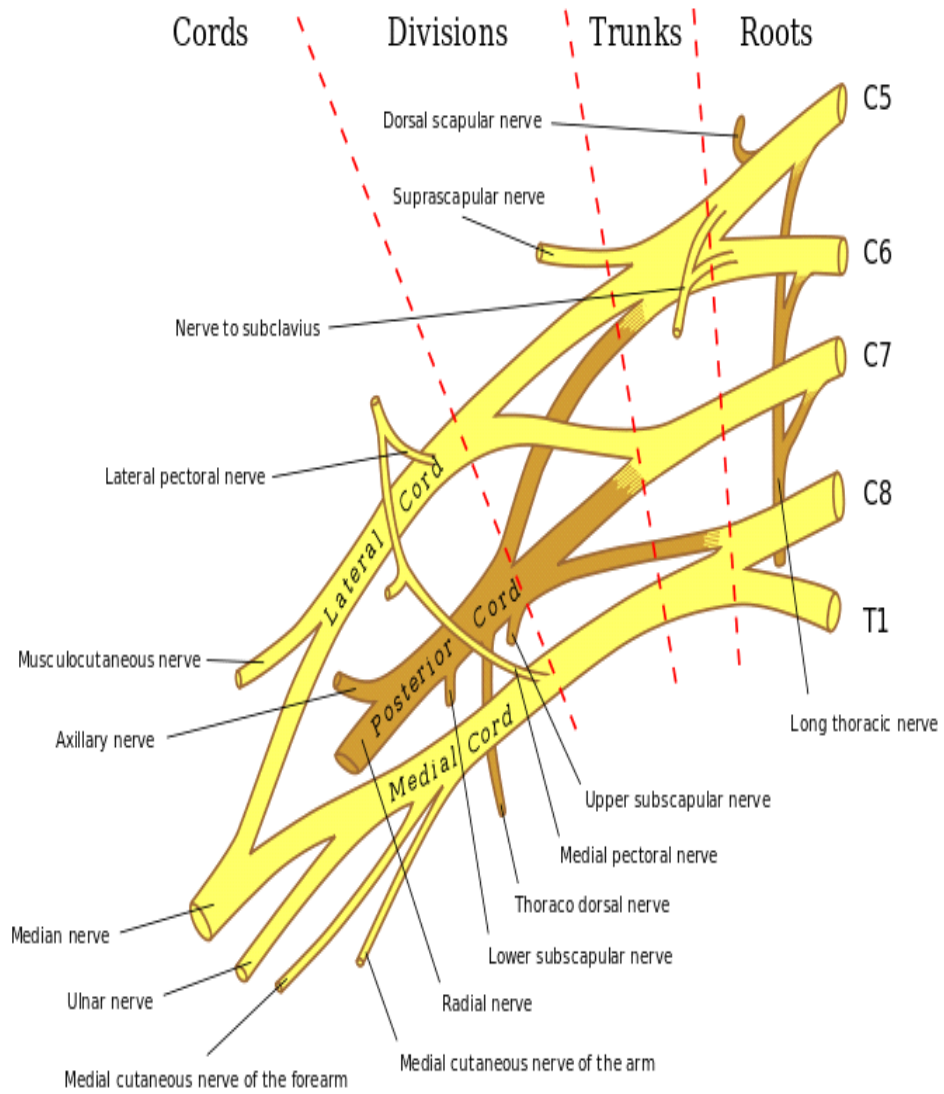
Fig. 1- Anatomy of Brachial Plexuses
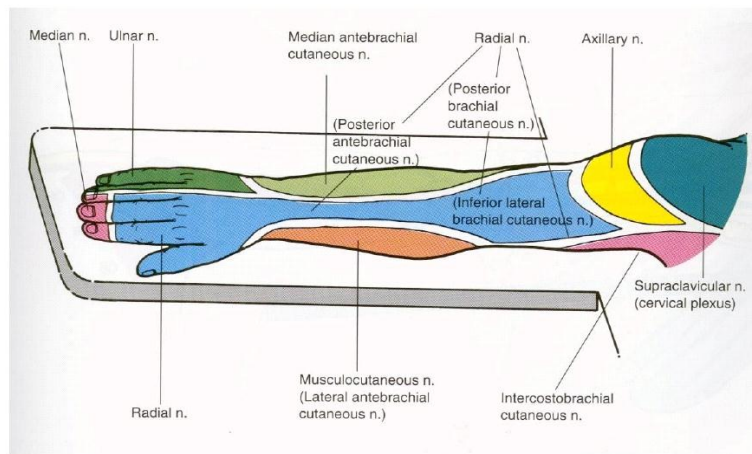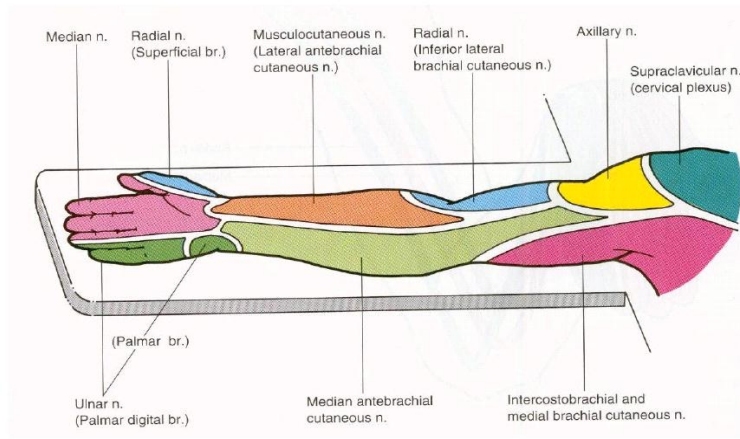
Fig. 2- Sensory Innervations of Palmar And Dorsal Surface

Relations

The brachial plexus initially lies between the anteriorscalene and the middle scalene muscles and travel across the posterior tri-angle of the neck. It is anteriorly covered by the skin, superficial fascia, platysma, deep fascia and the scalenus anterior. The supraclavicular nerves, nerve to subclavius, inferior belly of omohyoid, external jugular vein and transverse cervical artery cross over the brachial plexus. The posteriorly it is related to scalenus-medius and the long thoracic nerve. Inferiorly lie the first rib and the first digitation of serratus anterior with the subclavian artery anteriorly and the scalenus-medius behind. The dome of the pleura, covered by Sibson's fascia, lies inferomedial to the plexus just before it traverses the first rib. In the axilla, the posterior and lateral cords are lateral to the first part of the axillary artery, whilst the medial cord is behind it. The second part of the artery is surrounded by the cords. The cords here obtain their names from their relative positions to axillary artery: medial, posterior and lateral. Except the medial root of the median nerve, the branches of the cords maintain this relationship to the third part of the axillary artery, and these positions reflect their distribution in the limb. [27,28]

BRACHIAL PLEXUS SHEATH

The connective tissue that envelopes brachial plexus, subclavian artery and the axillary artery is derived from the pre-vertebral fascia, connective tissue of anterior and middle scalenes. This densely organized tissue leaves the deep cervical fascia proximally but becomes more loosely arranged distally. The sheath blends distally with the fascia of the biceps and brachialis muscle. The demonstration for the existence of connective tissue septae was done by anatomic dissection, histologic examination and contrast CT scans. The thin connective tissue septae frequently adhere to nerves and vessels leaving no free space between layers and compartmentalizing the components of the sheath. It is cylindrical to conical in shape, wide proximally and narrows distally. It has volume of 42ml and length 8-10cms (Fig 3).

Anaesthetic implications:

Anaesthesia might be rapid and complete in onset in some nerves, but delayed and incomplete or unsuccessful in others because of these connective tissue septae. The incidence of partial block is an exception rather than the rule, so septae apparently are of little clinical significance as the local anaesthetic can percolate through them.
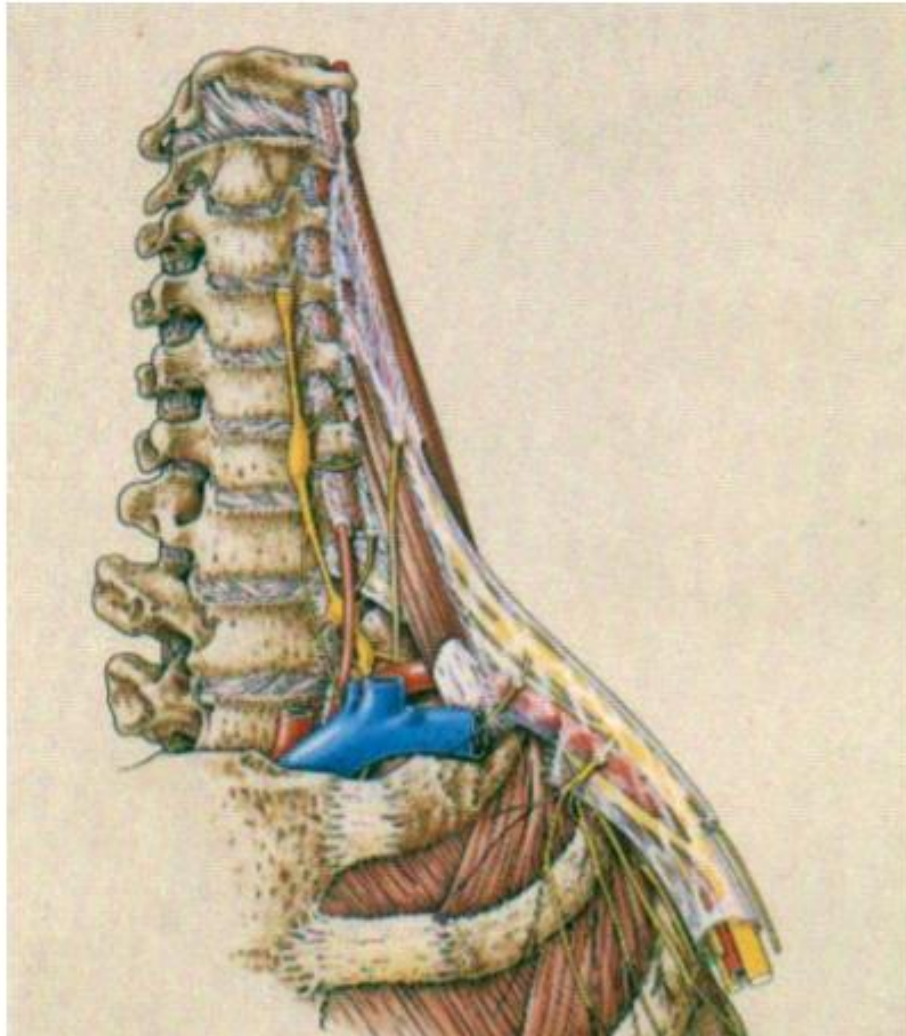
Fig No 3- Brachial Plexus Sheath

ULTRASONOGRAPHY

Ultrasound are sound waves sound waves >20,000 Hertz which are greater than the upper limit of human hearing. The audible sound frequencies are < 15000 to 20000 Hz.[4] Medical ultrasound imaging use frequency ranges of 2 -15 MHz. An ultrasound wave is a form of acoustic energy and is generated when multiple piezoelectric crystals inside a transducer (i.e., the probe) vibrate at high frequency in response to alternating current. The rapid vibration, which is transmitted to patient through a conductive gel, propagates longitudinally into the body as a short, brief series of compressions and rarefactions. With increasing frequencies the sound tends to behave more like electromagnetic beams and is reflected like light beams. They are reflected by much smaller objects (due to shorter wavelengths), and do not propagate easily through the gaseous media.

The wavelength is inversely related to the frequency f by the sound velocity c:c = λf. Meaning that the velocity equals the wavelength times the number of oscillations per second, and thus: λ =c/f.[4]

The speed of sound is different in different materials. It is dependent on the acoustical impedance of the material. But, the sonographic instrument assumes that the acoustic velocity is constant at 1540 m/s.[4] With this assumption, in a human body with non-uniform tissues, the beam is defocused and image resolution is decreased. Basically, all ultrasound imaging are performed by emitting a pulse. This pulse is partially reflected from a boundary between two tissue with different densities, and partly transmitted (Fig 4).

The difference in impedance of the two tissues determines the reflection. The ratio of the amplitude (energy) of the reflected pulse and the incident is called the

reflection coefficient. The ratio of the amplitude of the incident pulse and transmitted pulse is called the transmission coefficient. These two are dependent on the differences in acoustic impedance of the two materials. The acoustic impedance of a medium is the speed of sound in the material × the density.

The reflecting structures not only reflect directly back to the transmitter, but the ultrasound is scattered in several directions. The structures reflecting the waves are usually called as scatterers. The time taken for sound to travel the distance to the scatterer and back is the time lag between emitting and receiving a pulse i.e. twice the range, r, to scatterer at the speed of sound, c, in the tissue. Thus, r=ct/2

The pulse is thus emitted, and the system awaits to get the reflected signals, and calculates the depth of the scatterer on the basis of the time from emission to reception of the signal. The total time for getting the reflected ultrasound is determined by the preset depth desired in the image.

Piezoelectric effect

Ultrasound is generated by vibrations of piezoelectric crystals that when compressed and decompressed by an alternating current applied across the crystal, the same crystals can act as receivers of reflected ultrasound, the vibrations induced by the ultrasound pulse.[4]

Piezoelectric effect is produced by voltage between surfaces of a solid dielectric (nonconducting) substance when a mechanical stress is applied to it. A small current is also produced. The effect was discovered in 1883 by Pierre Curie in 1883.She received the noble prize for same. This effect is exhibited by certain crystals like quartz ,ceramic materials, Rochelle salt. When a voltage is applied across certain surfaces of a solid that exhibits the piezoelectric effect, the solid undergoes some

mechanical distortion. Piezoelectric materials are used in transducers e.g. phonograph cartridges, microphones, and strain gauges, which produce electrical output from a mechanical input, and in earphones and ultrasonic radiators, which produce mechanical output from electric inputs.

Transducer

Typically a sound wave is produced by a piezoelectric transducer encased in a probe. The sound is focused by the shape of the transducer, a lens in front of the transducer or by a complicated set of control pulses from the ultrasound scanning machine. This produces an arc-shaped sound wave from the transducer's face. The wave travels into the body and comes into focus at a desired depth. Whenever there is encounter of sound waves with a material having different density (acoustical impedance), it is partially reflected back to the probe and is detected as an echo. The time taken for the echo to travel back to the probe is measured. This measured time is used to calculate the depth of the tissue interface causing the echo.

Transducers use phased array techniques to allow the sonographic machine to change the depth and direction of focus. All piezoelectric transducers are made of ceramic. Materials on the face of the transducer allow the sound to be transmitted efficiently into the body (usually a rubbery coating, a form of impedance matching). Also, a water-based gel is placed between the patient's skin and the probe. The sound wave is partly reflected from the layers between different tissues with different densities. Sound is reflected wherever there are density changes in the body e.g. blood cells in blood plasma, small structures in organs, etc. Some of the reflections return to the transducer.

Fig. 4- Sonosite Machine with Linear Transducer

To generate a 2D-image, the ultrasonic beam is swept. Transducer may be swept mechanically by either rotating or swinging. Or a 1D phased array transducer may be use to sweep the beam electronically. The received data is processed and used to construct the image.

Doppler ultrasonography is used to study blood flow, heart contractility, see inferior vena cava fullness, muscle motion, to do a DVT scan in ICU, regional blocks. The different detected speeds are represented in color for ease of interpretation, like leaky heart valves: the leak shows up as a flash of unique color.

Display Modes

4 different modes of ultrasound are used in medical imaging. These are:

1] **A-mode** (amplitude modulation): A-mode is the simplest mode. The received energy at certain time i.e. from a certain depth, can be displayed as energy amplitude. The greater the reflection at interface, the larger the signal amplitude will appear on the A-mode screen.

2] **B-mode** (Brightness): The amplitude can also be displayed as the brightness of the certain point representing the scatterer. In this mode a linear array of transducers simultaneously scans a plane through the body that can be viewed as a two-dimensional image on screen. This mode is most commonly used.

3] **M-mode** (motion mode): If the scatterers are moving, the motion curve can be traced in m-mode. A rapid sequence of B-mode scans whose images follow each other in sequence on screen enables to see and measure range of motion.

4] **D-mode** (Doppler mode): This mode makes use of the Doppler effect. The Doppler information is displayed graphically using spectral Doppler, or as an image using color Doppler or power Doppler. This Doppler shift falls in the audible range and is presented audibly using stereo speakers: it produces a very distinctive, synthetic, pulsing sound. It can be used to identify artery or vein by specific sounds produced by each on Doppler.

TRANSDUCER MANIPULATION[4]

One of essential skills to acquire for regional block with ultrasound is transducer manipulation. Standardized nomenclature has been established:

- Sliding (moving contact). Sliding the transducer along the known course of the nerve using a short axis view often with nerve identification.

- Tilting (side to side). The echo brightness of peripheral nerves will vary with degree of tilt. Optimization of this angle is critical to promote nerve visibility.

- Compression. It is often used to confirm venous structures.

- Rocking (in-plane, toward/away from indicator). Rocking is often necessary to improve needle and anatomic structure visibility when working room is limited.

- Rotation. Some rotation of probe will produce true short axis views

- Anisotropy is the change in echogenicity with inclination of the transducer. In general, when objects are image obliquely, they appear less echogenic. This relationship is most pronounced for tendons, but muscles and nerves also show this relationship.

NERVE IMAGING WITH ULTRASOUND

Fascicles of peripheral nerves can be detected with high resolution ultrasound imaging. This fascicular echo-texture is most distinguishing feature of nerves, also called as '**honeycomb architecture**. Nerves can be round, oval or triangular. Nerve shape can change along the path, but cross section remains same. [4]

Although direct nerve imaging has led to phenomenal increase in USG guided regional anaesthesia, the identification of nearby structures is also critical. These structures permit favourable distribution of local anaesthetic so that nerve contact

with block needle is not necessary. Successful drug injections should clarify the borders of the nerve.

SCANNING APPROACH[4]

There are two scanning approaches to track needle movement in real time and visualize needle advancement. The **in-plane** approach is performed by passing the block needle beneath the long axis of the beam, hence, allowing full visualization of the needle tip and shaft. The **out-of-plane** approach is performed by passing the block needle beneath the short axis of the beam; thus, the needle appears as a bright "dot" in short axis.

The in-plane approach is more difficult to perform because it requires precise alignment of the ultrasound beam with the needle and nerve. For the out-of-plane approach, accurate needle tip localization can be difficult in the absence of a special echogenic design. In such case, the needle tip position is often inferred by observing local tissue movement and a dorsal ultrasound shadow at the time of needle advancement or tissue expansion at the time of fluid injection.

ULTRASOUND ARTIFACTS IN REGIONAL BLOCKS

An artifact is appearance of structure on an ultrasound which is actually not present or disappearance of an existent structure from the image, the knowledge of artifacts is essential to avoid errors while giving USG guided blocks.[29]

Following types of artifacts are seen:

1. Enhancement-When ultrasound passes through any sonolucent structure like fluid filled cavity, the image of the area behind that particular structure is enhanced.

2. Reverberation-When echoes are reflected from strong reflecting surface the exact replica of reflecting surface is seen at double the distance from transducer.

3. Shadowing-When ultrasound cannot penetrate a given tissue like bone, it causes a hyperechoic shadow beneath the bone. This is called as shadowing effect.

4. Bayonet-Sometimes when needle is inserted in plane and almost perpendicular to ultrasound beam, it appears broken or bent. This is called bayonet. It is because ultrasound travels with different speeds in different medium.eg-it travels faster in muscle and slower in adipose tissue.

DIFFERENT TECHNIQUES OF BRACHIAL PLEXUS BLOCK USING USG

A] Blocks above the clavicle

Level of the roots - Interscalene brachial plexus block

Trunks - Subclavian brachial plexus block

B] Blocks below the clavicle

Division/Cords - Infraclavicular brachial plexus block

Cords/Terminal nerves - Axillary brachial plexus block

Ultrasound allows direct visualization of peripheral nerves, the block needle, and local anaesthetic distribution. This imaging modality has proven highly useful to guide targeted drug injections and needle placement.

ULTRASOUND GUIDED AXILLARY BRACHIAL PLEXUS BLOCK

The axillary approach to brachial plexus was first demonstrated in 1884 by William Halsted when he injected cocaine under direct vision [30]. In 1911, G. Hirschel performed the first percutaneous axillary block [31]. It was only after Burnham's publication in 1959[32] that this block gained popularity among anesthetists.

Reding in 1921 is thought to be the first to highlight the importance of the neurovascular sheath in the axillary plexus block. His description of the anatomy of the brachial plexus within the axilla included discussion of nerve bundle surrounded by a fascial sheath. Reding was also aware that blocking of the musculocutaneous nerve required injection of local anesthetic within the coracobrachialis muscle since nerve was not contained within the sheath. [33]

In 1958 Preston Burnham, an orthopedic surgeon revived the neurovascular sheath approach for blocking the brachial plexus. While repairing an axillary laceration in a child, Burnham noted that the nerves entering the axilla were proximal to the axillary artery. Additionally, a fascial sheath surrounded both nerves and vasculature. If the sheath were entered with one pass of a needle, multiple nerves could be bathed with local anaesthetic.

In 1981, Abramowitz and Cohen described the use of Doppler ultrasound to identify the axillary artery for the first time, thereby aiding the performance of axillary plexus block for upper limb surgery. [34] But the use of B-mode ultrasound in 1989 for axillary block performance heralded the era of ultrasound-guided peripheral nerve block. [35]

Axillary brachial plexus block offers several advantages over other approaches. The technique is relatively simple, and complications are very less as

compared to interscalene (spinal cord, vertebral artery puncture) or supraclavicular (pneumothorax). The block is easy to perform because of its superficial location.

Indication:

- Surgical anaesthesia for elbow, forearm, and hand procedures,
- Cutaneous anesthesia for superficial procedures of the inner arm, for example, brachiobasilic fistula formation,
- Chronic pain treatment.

Anatomic consideration of axillary block include the following: (Fig 5) [1]

- The neurovascular bundle is multi-compartmental.
- The important landmark is the axillary artery.
- There is a large degree of anatomical variability in nerve positions around the axillary artery, extended scanning up and down the arm is recommended to locate the nerves accurately. (Fig 6)
- At this level, the musculocutaneous nerve has already left the sheath and lies with the coracobrachialis muscle.
- Adequate anaesthesia for the tourniquet requires intercoastobrachial nerve block (IcBN). IcBN is the lateral branch of the anterior ramus of T2 and provides cutaneous innervation to the upper medial and posterior part of the arm. It can be blocked by subcutaneous infiltration along the medial aspect of the arm from the anterior axillary line to the border of triceps. Using a landmark technique 5 – 10 ml of local anaesthetic is required.

Patient positioning (Fig 9)- All of the axillary block techniques require the patient to be positioned supine, with the arm abducted 90° and the head turned toward the contralateral side. The axillary artery pulse should be palpated and its location marked as a reference point.

Techniques of Axillary Block [1]- Several methods of identifying the axillary sheath have been described, all with reportedly good results

- Paresthesia technique- paresthesia can be sought with a 25-gauge, 2-cm needle, beginning with radial nerve or with the nerves supplying the surgical site. Smaller needles and a short needle bevel may be associated with a less frequent risk of nerve damage. Each paresthesia is injected with 10 mL of local anaesthetic.

- A nerve stimulator can also be used with an insulated needle to locate the nerves. Stimulation with a low current threshold (0.5 mA). This decreases onset time, but increases block performance time compared with higher-threshold stimulation (1.0 mA).

- A short-bevel needle can be advanced until the axillary sheath is entered, as evidenced by a fascial click, whereupon 40 to 50 mL of solution is injected after negative aspiration.

- A transarterial technique can be used, whereby the needle pierces the artery and 40 to 50 mL of solution is injected posterior to the artery. Alternatively, half of the solution is injected posterior and half is injected anterior to the artery. Great care must be taken to avoid intravascular injection with this technique, particularly because the pressure of injection within the compartments of the axillary sheath may move anatomic structures in relation to the immobile needle.

Ultrasound anatomy (Fig 8) - The transducer position is short axis to the arm, just distal to pectoralis major insertion (Fig 7). The structures of interest are superficial 1-3cm. Also, the pulsating axillary artery can be identified usually within cm of skin surface on the anteromedial aspect of proximal arm. One or more axillary veins can be seen just medial to the artery. Undue pressure on transducer may obliterate the veins making them invisible and prone to puncture if care not taken. Surrounding the axillary artery are three or four principal branches of brachial plexus: the median (superficial and lateral to artery), the ulnar (medial to the artery), and the radial (posterior and lateral or medial to the artery) nerves. The nerves are seen as round hyperchoic structures. Many variations can be seen in position of the nerves although the ones mentioned above are more commonly seen. Three muscles surround the neurovascular bundle, the bicep brachii(medial), corachobrachialis(lateral) and triceps (medial and posterior). The forth principal nerve, the musculocutaneous nerve is found between the fascial layers of bicep brachi and coracobrachialis muscle as fish mouth appearance.

Patient positioning and technique with arm Abducted to 90 degrees with head turned towards opposite side. The pectoralis major muscle is palpated as it inserts onto the humerus and transducer is placed on skin immediately distal to that point. Sliding transducer across axilla will bring axillary artery in view and other nerves can also be visualized.

With proper positioning, skin is cleaned with disinfectant. Transducer positioned in short axis to identify axillary artery. Once artery identified, other nerves surrounding it are identified. Also scan for position of musculocutaneous nerve with transducer moving slight proximally. The needle is inserted in plane from the cephalad aspect and drug is deposited anterior and posterior aspect of axillary artery.

This method is also called as perivascular method of axillary brachial plexus block. In other method we target each nerve individually and give drug at each nerve and visualize drug spread. This method is called perineural method of axillary brachial plexus block. The musculocutaneous nerve is separately blocked in both methods. Two or three redirections and injections are usually necessary for reliable blockade.

Complications-

- Vascular puncture and intravascular injection may lead to systemic LA toxicity.

- Hematoma is rare (0.2% even using a transarterial technique) but may cause vascular insufficiency and compressive nerve injury. The compressible nature of the axillary vessels means that the axillary block is the approach to the brachial plexus most suitable for use in patients with mild coagulation abnormalities.

- Neurological injury
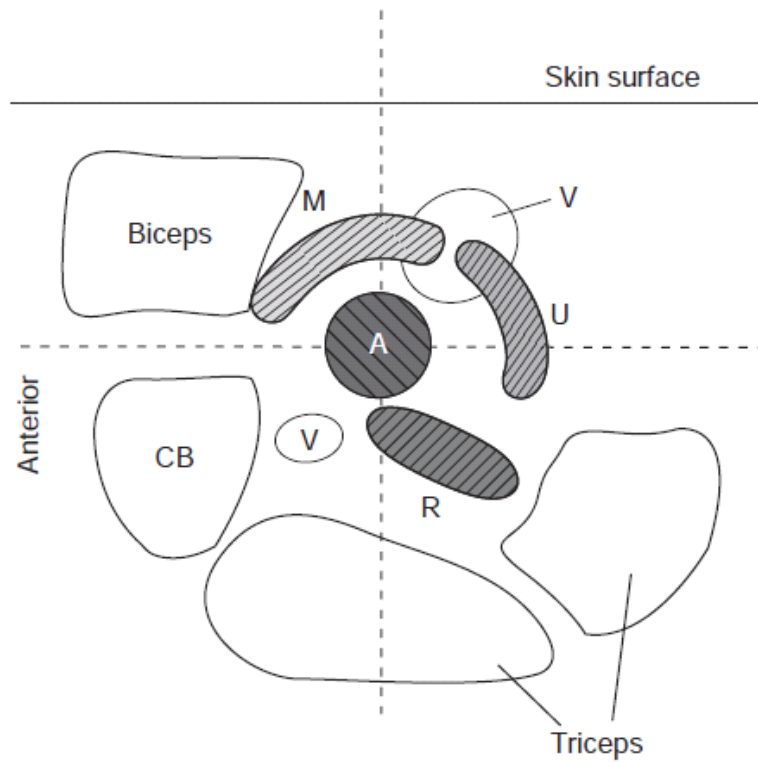
Fig 5 - Cross-Sectional Anatomy At The Level Of Axilla With Approximate Locations Of Nerves In Relation To Axillary Artery.

A = axillary artery; CB = coracobrachialis muscle; M = approximate location of median nerve; R = approximate location of radial nerve; U = approximate location of ulnar nerve; V = veins
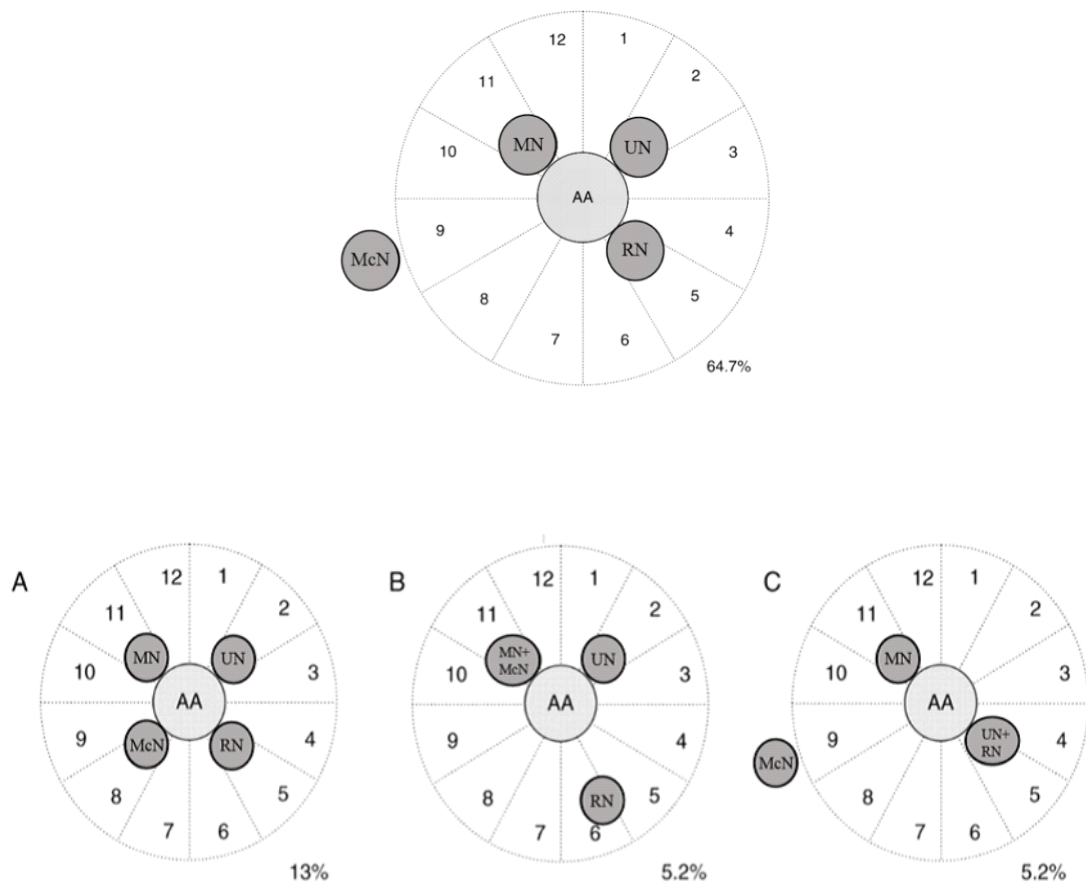
Fig 6- Illustration Of Anatomical Variability Of Main Nerves At The Level Of The Axilla. (Left Side Lateral, Right Side Media)l

MN = median nerve UN = ulnar nerve RN = radial nerve McN = musculocutaneous nerve

Fig 7- Probe And Needle Placement For USG Guided Axillary Block
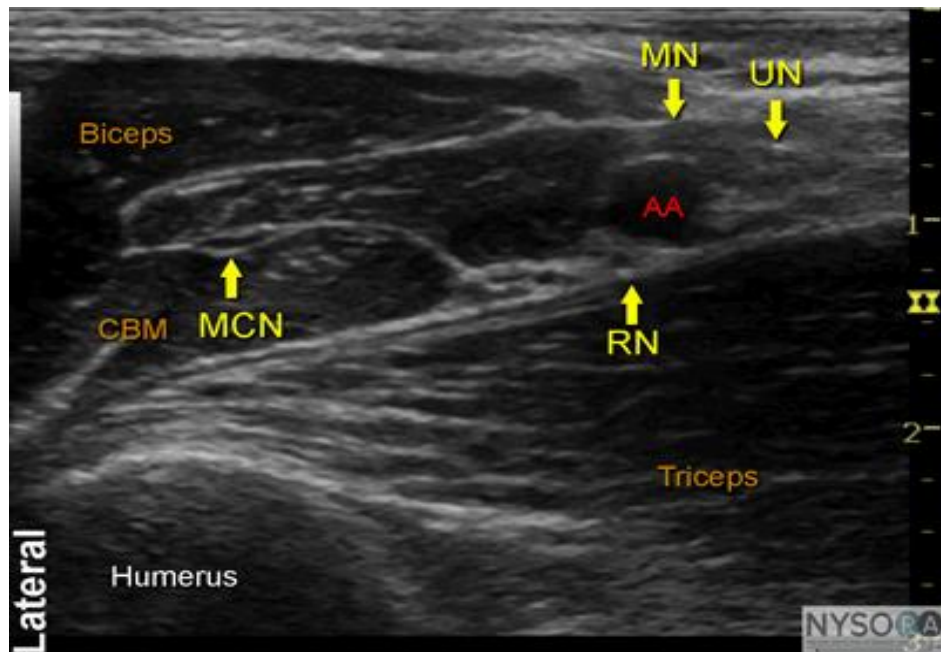
Fig 8- USG Image For axillary Block

AA-axillary artery, CBM-coracobrachialis, McN-musculocutaneous nerve,

UN-ulnar nerve, RN-radial nerve, MN-median nerve

Fig 9- Position Of The Patient With Ultrasound Probe Placed On Axilla And Needle Insertion.

PHARMACOLOGY OF LOCAL ANESTHETICS

For selecting an appropriate local anaesthetic drug for a specific clinical situation, one should know the clinical pharmacology of the local anaesthetic drugs and the adjuvant. The effect of local anaesthetics is exerted either by inhibiting the excitatory process in the nerve endings or in the nerve fibres. The following sequence of events is accepted as the mechanism of action of local anaesthetic agents: [36]

- Binding of the local anesthetic to the receptor sites in the nerve membrane.

- Reduction in sodium permeability

- Decrease in the rate of depolarization

- Failure to achieve threshold potential

- Lack of propagation of action potential

- Conduction blockade

The pharmacological activity of local anaesthetic agents is influenced by their chemical structure, lipid solubility, protein binding and pKa.

- **Chemical structure** [36]

  Based on chemical structure local anaesthetics can be classified

  {A} Aminoesters- Procaine, cocaine, tetracaine, choroprocaine. They have an ester linkage between the benzene ring and the intermediate chain. These are hydrolyzed in plasma by pseudocholinesterase. Primary metabolite of ester compounds is paraminobenzoic acid (PABA), which has allergic potential.

{B} Aminoamides- Lidocaine, mepivacaine, bupivacaine, ropivacaine.

They have an amide link between the benzene ring and intermediate chain. These are degraded in the liver by microsomal enzymes. The amide drugs are not metabolized to paraaminobenzoic acid and do not produce allergic reactions. Multi-dose vials of amide local anaesthetic may contain methylparaben which is a paraaminobenzoic acid derivative with allergic potential.

- **Lipid solubility**

Lipid solubility is the primary determinant of intrinsic anaesthetic potency of local anaesthetic. Potency increases as a function of lipid solubility until a blood/lipid partition coefficient of 4 is reached. Further increase in lipid solubility does not cause a further increase in the local anaesthetic potency. Depending on the lipid solubility and potency, local anaesthetic drugs can be divided into 3 groups:

a. **Low lipid solubility/potency:** Lipid partition coefficient < 1. These drugs must be administered in high concentrations (2 to 3 %) to achieve effective neural blockade. e.g procaine and chloroprocaine.

b. **Intermediate lipid solubility/potency:** Lipid partition coefficient =1-3. These drugs may be given in concentrations of 1 to 2%.e.g lidocaine, mepivacaine, and prilocaine.

c. **High lipid solubility/potency:** Lipid partition coefficient >4. These drugs are clinically effective at concentrations <1%.e.g tetracaine, bupivacaine, and ropivacaine.

- **Protein binding**

Addition of larger chemical radicals to the amine or aromatic end of a local anaesthetic compound increases its binding to protein, which determines local anaesthetic duration. Protein binding of some local anaesthetics is as follows:

Bupivacaine---95%

Tetracaine------95%

Ropivacaine---94%

Mepivacaine--74%

Lidocaine---65%

Procaine---6%

- **PKa**

Pka is the pH at which ionized and unionized fractions of a substance are present in an equal amount. It is the unionized fraction that primarily diffuses across the nerve membrane. The onset of local anaesthetic effect will be determined by total amount of unionized fraction of the local anaesthetic agent. The percentage of local anaesthetic, which is present in the unionized form (cation or base) when injected into the tissue at (pH 7.4) is inversely proportional to the pKa of the agent. As the pH of the local anaesthetic solution goes down, the unionized fraction will decrease and when the pH increases, the unionized fraction increases. There is a correlation between the onset of block and pKa of local anaesthetic drug. Drugs with pKa of 7.6-7.8(lidocaine, mepivacaine, prilocaine) have more rapid onset of action than do bupivacaine and tetracaine which have a pKa of 8.1 and 8.6 respectively.,

ADJUVANT DRUGS [37]

- Epinephrine- Epinephrine is a commonly used additive to local anaesthetics when performing peripheral nerve blocks. Epinephrine has been shown to increase block intensity as well as duration of anaesthesia and analgesia with intermediate-acting local anaesthetics. As a vasoconstrictor with strong alpha-1 effects, epinephrine decreases systemic absorption of the local anaesthetic limiting peak plasma levels and prolonging block time. The drug also provides a marker for intravascular injection in dilute concentrations due to its beta-1 effects. Adjuvant use of epinephrine will have systemic effects, including tachycardia and increased cardiac inotropy, and therefore its use in patients with a significant cardiac history should be carefully considered. The drug should probably be avoided when performing a block to an area receiving diminished or absent anastomotic blood flow. Due to concerns about ischemic neurotoxicity, doses administered in concentrations of 1:400,000 (2.5mcg/ml) or less may be prudent. Epinephrine administered perineurally decreases extrinsic blood supply when administered in higher concentrations, though there is no evidence this effect is detrimental to humans.

- Clonidine-It prolongs duration of local anaesthetics by synergistic alpha-2effects. It has lesser or no prolongation with Bupivacaine and Ropivacaine but prolongs the duration with Mepivacaine-Lidocaine by 40-400% with the addition of 100 micrograms of clonidine. Larger doses are not additive and cause more side effects.

- Sodium bicarbonate, hyaluronidase: onset time was reduced, and the duration was variable.

- Opioids: Onset time was reduced, and the duration was prolonged, but reports were controversial.

- Dexamethasone- The drug clinically appears to lengthen the sensory, motor, and analgesic time of peripheral nerve blocks when added to both intermediate and longer-acting local anaesthetics. The mechanism by which this effect occurs has yet to be determined. At the time of writing, a number of studies have been published showing a beneficial effect of dexamethasone as an adjunct to local anaesthetics in regional anaesthesia and pain medicine procedures. Dexamethasone use in epidural steroid injections is increasingly popular among pain practitioners because of the medication's pharmacologic profile in comparison with other corticosteroids: dexamethasone is non-particulate and void of neurotoxic preservatives. Concern over ischemic neurotoxicity has been raised due to the drug's effect, like epinephrine, of decreasing normal nerve tissue blood flow as demonstrated by topical application of 0.4% dexamethasone to the exposed sciatic nerve in rats. As when using epinephrine, it would seem prudent to properly select candidates for adjunctive use of dexamethasone excluding patients at greatest risk for ischemic nerve injury (e.g., poorly controlled diabetes, pre-existing nerve injury, or demyelinating disorder).

BUPIVACAINE[36,37]

Source: Bupivacaine, was first prepared by A.F. Ekenstam in 1957.

Chemistry: The molecular weight of the chloride salt is 325 and that of the base form is 288. It has a melting point of 258°C.

Chemical name: l-n-butyl-DL-piperidine-2 carboxylic acid-2,6 dimethylamilidehydrochloride.
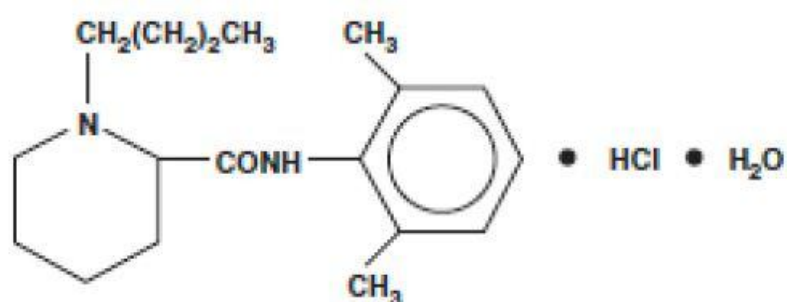
Fig 10- CHEMICAL STRUCTURE OF BUPIVACAINE

Chemical Properties:

1) **Solubility:** The base is sparingly soluble, but the hydrochloride is readily soluble in water.

2) **Stability and sterilization:** highly stable, can withstand repeated autoclaving.

3) **pH of saturated solution:** 5.2

4) **Melting point:** 247-258°C

5) **Specific gravity:** 1.021 at 37°C

Potency:

Bupivacaine is 3 to 4 times more potent than Lidocaine. The duration of action for local anaesthesia is also two to three times longer than Lidocaine. Bupivacaine's anaesthetic index is 3.0 to 4.0.

Mechanism of action:

The primary action is on the cell membrane of the axon, on which it produces electrical stabilization. The large transient increase in permeability to sodium ions necessary for propagation of the impulse is prevented. Thus, the resting membrane potential is maintained and depolarization in response to stimulation is inhibited. It blocks the generation and the conduction of nerve impulses, by increasing the threshold for electrical excitation in the nerve, by slowing the propagation of nerve impulse, and by reducing the rate of rise of the action potential. Generally, the progression of anaesthesia is related to the diameter, myelination and conduction velocity of affected nerve fibres. Clinically, the order of loss of nerve function is as given below: (1) pain, (2) temperature, (3) touch, (4) proprioception and (5) skeletal muscle tone.

Concentration available:

• 0.25%, 0.5%.

• 0.25% and 0.5% soluble in isotonic saline

• 0.5% solution in 8% dextrose - Hyperbaric

These doses may be repeated in 3-4 hours, but the maximum dose in 24 hours is 400mg. The addition of vasoconstrictor produces a very slight increase in the duration of action. The dosage depends on no. of factors like area to be blocked, technique used, no. of segments to be blocked, vascularity of tissue etc.

Pharmacodynamics:

The onset of action of Bupivacaine is between 4 and 6 minutes and maximum anaesthesia is obtained between 15 and 20 minutes. The duration of anaesthesia varies according to the type of block. The average duration for epidural block is about 3.5-5 hours, spinal block is 2-3 hrs and 5 to 6 hour for nerve blocks.

Pharmacokinetics:

Bupivacaine can be detected in the blood within 5 minutes of infiltration or following epidural. Plasma levels are related to the total dose administered. Peak levels of 0.14-1.18 µg/ml are found within 5 minutes to 2 hours after the administration of anaesthesia and they gradually declined to 0.1 to 0.34 µg/ml by 4 hours. In plasma, drug binds avidly with protein (l-acid glycoprotein) to the extent of 70-95%. The order of protein binding for this drug is- Bupivacaine, Mepivacaine and Lidocaine. Conversely, the unbound active fraction is one seventh that of Lidocaine and one fifth that of Mepivacaine.

Metabolism and elimination:

The liver is the primary site of metabolism. The drug is metabolized partly by N-dealkylation primarily to pipecolyloxylidine, 4-hydroxy-bupivacaine and N-disbutyl-bupivacaine. It crosses the placental by passive diffusion (umbilical vein/maternal ratio is 0.31 to 0.44). The high protein binding capacity of the agent is probably the reason why less diffusion occurs across the placenta. No foetal effects have been noted. About 10% of drug is excreted unchanged in urine within 24 hours

Actions:

Central nervous system: Bupivacaine overdose leads to light headedness, dizziness followed by visual and auditory disturbances such as difficulty to focus and tinnitus. Shivering, muscular tremors and tremors of muscles of face and distal part of extremities can occur. Ultimately generalized tonic clonic convulsions can occur. It can cause respiratory arrest also. Since Bupivacaine is a potent drug, smaller doses can cause rapid onset of toxic symptoms when compared to other drugs.

Autonomic nervous system:

Myelinated preganglionic beta fibres have a faster conduction time and are more sensitive to the action of local anaesthetics including bupivacaine. Involvement of preganglionic sympathetic fibres is the cause of widespread vasodilatation and consequent hypotension that occurs in epidural and paravertebral blocks. When used for conduction blockade, all local anaesthetics particularly Bupivacaine produce higher incidence of sensory than motor fibres blockade.

Cardiovascular system:

The primary cardiac effect is a decrease in the maximum rate of depolarization in the purkinje fibres and ventricular muscle. This is due to a decrease in the availability of sodium channels. Action potential duration and the effective refractory period are reduced. The depression of rapid phase of depolarization (V-max) in purkinje fibres and ventricular muscle by Bupivacaine is far greater compared to Lignocaine. Also the rate of recovery of block is slower with Bupivacaine. Therefore, Bupivacaine is highly arrhythmogenic. The cardiac contractility is reduced, which is by blocking the calcium transport. Low concentration of Bupivacaine produces vasoconstriction while a higher dose causes vasodilatation.

Respiratory system:

Respiratory depression may be caused if excessive plasma level is attained. This may be due to depression of respiratory medullary center.

Adverse reactions:

Adverse reactions occur with excessive plasma levels which can be due to overdose, inadvertent intra venous injections or slow metabolic degradation. These manifest as both effects on CNS and CVS. The CNS effects are characterized by excitation or depression. The first manifestation may be nervousness, dizziness, blurring of vision or tremors following drowsiness, convulsions, unconsciousness and probably respiratory arrest. Other side effects include nausea, vomiting, chills, constriction of pupils and tinnitus. The CVS manifestation includes hypotension and cardiac arrest, in obstetrics foetal bradycardia may occur. Allergic reactions include urticaria, bronchospasm and hypotension.

Treatment of adverse reaction:

- Treatment is mainly symptomatic.

- Maintain circulation and support ventilation with oxygen or controlled ventilation.

- If required, supportive treatment with intra venous fluids and vasopressors should be started to restore the cardiovascular stability.

- Diazepam (0.1- 0.2 mg/kg) or Thiopentone (2-3 mg/kg) can be used to control convulsions

- Muscle relaxant and controlled ventilation with oxygen can also be used.

- Allergic reactions are to be treated with corticosteroids.

- For Ventricular fibrillation and ventricular tachycardia Amiodarone (5mg/kg iv) or defibrillation (2-6 joule/kg) to be used

Treatment of overdose:

There is animal evidence that intralipid, a commonly available intravenous lipid emulsion can be effective in treating severe cardiotoxicity secondary to local anesthetic overdose and human case reports of successful use in this way. Dose of intralipid is 1.5ml/kg i.v over 1 min followed by 0.25-0.5ml/kg/min iv for next 10 minutes. [38,39]

The cardiovascular collapse CC/ CNS ratio:

The CC/CNS dose ratio for Bupivacaine is 3.7±0.5. Studies show that 3 times drug was required to induce irreversible cardiovascular collapse as was needed to produce convulsions.

Developments:

Levobupivacaine is the (S)-enantiomer of bupivacaine with a longer duration of action and also produces less vasodilation.

LIGNOCAINE [37]

Classification-It is an amide local anaesthetic.

First synthesized- In 1943, in Sweden, by Lofgren.

Lignocaine has comparatively rapid onset of action and intermediate potency and duration of action.

Molecular weight- 234.
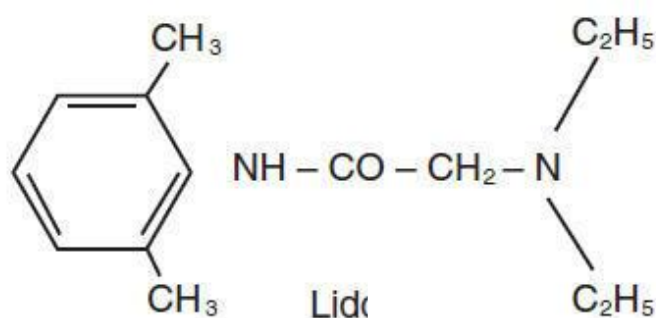
Fig 11- Chemical structure of lignocaine

pKa- 7.61 at 36°C.

Metabolism About 60-75% of lignocaine is protein bound. It is metabolized principally in the liver by oxidative dealkylation to monoethylglycinexylidide, followed by hydrolysis of the metabolite to xylidide. Xylidide has only 10% of cardiac dysrrhythmic action. About 75% xylidide is excreted in the urine as 4 - hydroxy 2, 6- dimethylalanine (Govino and Vassallo 1976). The partition coefficient is 2.9. Clearance of local anaesthetic from the plasma parallels hepatic blood flow. Hepatic disease or reductions in hepatic blood flow, as does occur during anaesthesia, can reduce the rate of metabolism of lignocaine.

The maximum safe dose- 4mg/kg for plain lignocaine and7mg/kg body weight for lignocaine with adrenaline

Adverse effects-Lignocaine produces numbness of tongue and circumoral tissues at low concentration. As the plasma concentration continues to rise, it readily crosses the blood-brain barrier and produces CNS changes. Restlessness, vertigo, tinnitus and difficulty in focusing occur initially. Later on slurred speech, skeletal muscle twitching occur. Plasma concentration above 5-10 mcg/ml are known to produce CVS toxicity. Acute elevation in plasma concentration (above 10 mcg/ml) may produce hypotension due to smooth muscle relaxation. At 25-30 mcg/ml cardiac output decreases by 40% and contractility by 50%. Hypercarbia, acidosis, hypoxia all potentiate the toxic effects of lignocaine; and allergic reactions are rare.

MATERIALS AND METHODS

SOURCE OF DATA:

  This study was carried out in the Department of Anesthesiology, B.L.D.E.(Deemed to be University) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

METHOD OF COLLECTION OF DATA

Study Design: A comparative clinical study.

Study Period: One and half year from December 2016 to August 2018

Sample Size: With the Mean of two groups as 8.2 for PV (Perivascular) group, 15.7 for PN (Perineural) group and Standard Deviation as 2.3 for PV, 3.2 for PN group, the minimum sample size per study group - 53.

Formula used:-

$$N = 2\left[\frac{(Z_{1-\alpha/2}+z_\beta)*S}{d}\right]^2$$

$Z_{1-\alpha/2}$ Level of significance=95%

$Z_{1-\beta}$ Power of the study=80%

S- Standard deviation (From previous study) (5)

d- Clinically significant difference in mean

METHOD OF STATISTICAL AMNALYSIS

Data obtained was entered into a Microsoft excel spreadsheet. The categorical data was expressed in terms of rates and percentage; and continuous data was expressed in terms of mean± standard deviation. Data analysis was carried out using SPSS Version 17. Software. Mann Whitney 'U' test was used to compare quantitative variables of two groups. The categorical data was compared using Chi-square test. The probability value (p-value) less than 0.05 (p<0.05) was considered to be statistically significant.

RANDOMIZATION

The study population of 106 patients matched age and sex undergoing forearm, wrist or hand surgery were randomly selected by and divided by computer into two groups with 53 patients in each group.

Group I –Perivascular (PV) USG-guided ABPB will be performed.

Group II –Perineural (PN) USG-guided ABPB will be performed.

INCLUSION CRITERIA:

- Age between 18 and 60 years

- American Society of Anaesthesiologists (ASA) status I and II

- Patients presenting for forearm, wrist or hand surgery

EXCLUSION CRITERIA:

- Inability to consent

- Allergy to local anaesthetic agents

- Local infection

- Coagulopathy

- Pre-existing neuropathy

- Prior surgery in the axilla

Methodology

Pre-anaesthetic evaluation:

A thorough pre-anaesthetic evaluation was done with history of underlying medical illness, previous history of surgery, anaesthetic exposure and hospitalization taken.

Examination included; General condition of the patient with vital signs- heart rate, blood pressure, respiratory rate, height and weight. Systemic examination of cardiovascular system, respiratory system, central nervous system and the vertebral system was done. Airway assessment by mallampati grading was done. Examination of axilla done to see for any signs of local infection, lesions or scars. Baseline investigation of complete blood count, urine routine, radiograph of the chest, and ECG was done. Axilla part preparation was advised.

Procedure explained to the patient and informed written consent was taken patients were kept nil by mouth at least for six hours prior to surgery.

Procedure

On the day of surgery, nil by mouth status of patient confirmed. Patient was taken to Operation theatre. Standard monitoring devices including ECG leads, sphygmomanometer cuff, and pulse oximeter connected, and baseline values recorded. IV line secured with 20G cannula and patient premedicated with Inj.Ondansetron 0.15mg/kg IV, Inj.glycopyrolate 0.01mg/kg and Inj.Midazolam 0.1mg/kg IV. Patient was positioned supine with arm abducted to 90 degree & elbow flexed to 90 degree, with dorsum of hand resting on the bed or pillow.

The block site painted with povidine iodine solution and spirit and draped with a sterile towel. Sterile gel applied to ultrasound probe and probe covered by sterile cover. The block performed from below (facing the patient) ensuring in-line alignment of patient, operator and ultrasound machine. The USG probe (SonoSite M-Turbo machine) placed across the axilla, approximately at the junction of biceps brachii and pectoralis major muscle. The pulsating axillary artery visualized, and the probe moved to locate the individual nerves around the artery.

In both groups a mixture of 8ml of 0.5% Bupivacaine Hydrochloride, 10ml 2% Lignocaine Hydrochloride with Adrenaline and 2ml of Dexamethasone (4mg/ml), amounting to 20 ml of local anaesthetic was given. In the Group I (PV), the imaging time was defined as the time required visualizing the musculocutaneous nerve and the axillary artery. In the Group II (PN), the imaging time was the time needed to localize all 4 nerves. After obtaining a satisfactory image, using an in-plane technique, the 22-23gauge, insulated needle was advanced towards the musculocutaneous nerve. 5ml of local anaesthetic drug mixture was deposited around the musculocutaneous nerve in both groups. In Group I (PV), the needle was

advanced and remaining 15ml of local anaesthetic drug mixture was injected anterior and posterior to the axillary artery incrementally.

In Group II (PN), the radial nerve was anaesthetized after the musculocutaneous nerve and then needle withdrawn towards the skin and redirected towards median and ulnar nerves. These nerves were individually anaesthetized with 5 ml of local anaesthetic drug mixture.

The needling time defined as time interval between introduction of needle and the end of local anaesthetic injection through needle was recorded. Performance time defined as sum of imaging time and needling time was recorded. After LA injection through the needle, measurement of sensory and motor blockade was carried out every 5 min till 30 min. Toxicity of local anaesthetic like peri-oral numbness, dizziness or convulsions were looked for. Sensory blockade of the musculocutaneous nerve, median nerve, radial nerve and ulnar nerve was graded according to a 3point scale using pin prick test: 0 = Sharp pin sensation felt, 1 = analgesia (dull sensation felt), or 2 = anaesthesia (no sensation felt).

Sensory blockade of the musculocutaneous nerve, median nerve, radial nerve and ulnar nerve was assessed in the corresponding dermatomal areas. After the completion of the block procedure, sensory onset was considered when there was dull sensation to pin prick (Grade 1) along the distribution of any of the above mentioned nerves. The duration of sensory block was defined as the time interval between the end of LA administration and the complete resolution of anaesthesia on all nerves.

Motor blockade assessment was based on the modified Bromage scale for upper extremities on a 3 point scale. Grade 0 = normal motor function with full extension of elbow, wrist and fingers, Grade 1 = decrease motor strength with ability to move fingers and/or wrist only and Grade 2 = complete motor blockade with

inability to move fingers. Onset of motor blockade was considered when there was Grade 1 motor blockade after completion of block procedure. Peak motor block was considered when there was Grade 2 motor blockade. The duration of motor block was defined as the time interval between the end of LA administration and the recovery of complete motor function of the hand and forearm.

Postoperatively, motor and sensory blockade and vitals of the patient was noted half hourly till the block completely wears off. The block was considered as failure when analgesia to pin prick was not elicited at the site of surgical incision even after 30 min of drug administration. The onset and duration of sensory block, the onset and duration of motor block, number of failed blocks and complications in terms of block related pain, paraesthesia and vascular puncture was noted.

In case of pain during surgery, the block was considered a failure, and the patients were allowed to receive intravenous narcotics, general anaesthesia, rescue blocks, or local infiltration by surgeon. The patient's anthropometric data and the level of procedural pain immediately after block placement, using 10-cm visual analogue scale was recorded. (Fig 12)
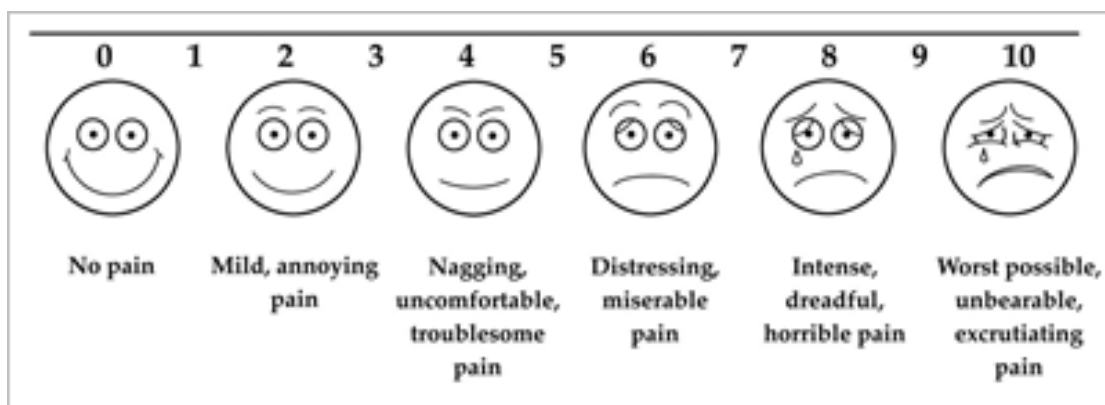
Fig 12 : Visual Analogue Pain Scale

Parameters studied

- Imaging time, s

- Needling time, s

- Performance time, min

- Onset of block, min

- Block success rate (%)

- No. Of needle passes

- Duration of the sensory block, min

- Duration of the motor block, min

- Block related pain (0-10)

- Vascular puncture, n (%)

- Local anaesthetic toxicity

RESULTS

A comparative study between USG guided perivascular ABPB and perineural ABPB done on 106 patients divided into 2 groups of 53 each in the age group of 18-60yrs. The following observations were made.

Table No 1: Distribution of patients according to Age (Years)

Age(Years)	Group I (PV)	Percentage	Group II (PN)	Percentage
<= 20	4	7.5	2	3.8
21 - 30	15	28.3	22	41.5
31 - 40	10	18.9	12	22.6
41 - 50	12	22.6	9	17.0
51+	12	22.6	8	15.1
Total	53	100.0	53	100.0

Graph No 1: Distribution of patients according to Age(Years)
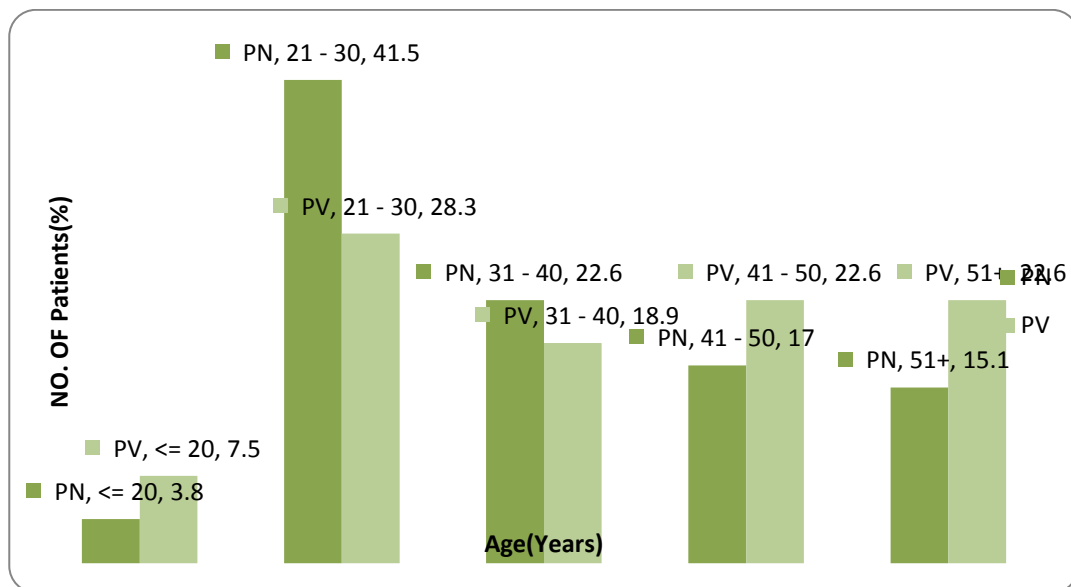
Table N0 2: Percent distribution of patients according to Age (Years)

Age (Years)	Group I (PV)	Percentage	Group II (PN)	Percentage	Chi square test
<= 20	4	7.5	2	3.8	
21 – 30	15	28.3	22	41.5	
31 – 40	10	18.9	12	22.6	P=0.4930 NS
41 – 50	12	22.6	9	17.0	
51+	12	22.6	8	15.1	
Total	53	100.0	53	100.0	

The percentages of patients belonging to age ≤20 were 7.5% in Group I (PV) and 3.8% in Group II (PN). The percentages of patients belonging to age 21-30 were 28.3% in Group I (PV) and 41.5% in Group II (PN). The percentages of patients belonging to age 31-40 were 18.9% in Group I (PV) and 22.6% in Group II (PN). The percentages of patients belonging to age 41-50 were 22.6% in Group I (PV) and 17.0% in Group II (PN). The percentages of patients belonging to age 51+ were 22.6% in Group I (PV) and 15.1% in Group II (PN). The age distribution between the two groups was not statistically significant and age was comparable in both groups (p value> 0.05).

Table No 3: Distribution of patients according to Gender

Gender	Group I (PV)	Percentage	Group II (PN)	Percentage
Female	16	30.2	14	26.4
Male	37	69.8	39	73.6
Total	53	100.0	53	100.0

Graph No 2: Distribution of patients according to Gender
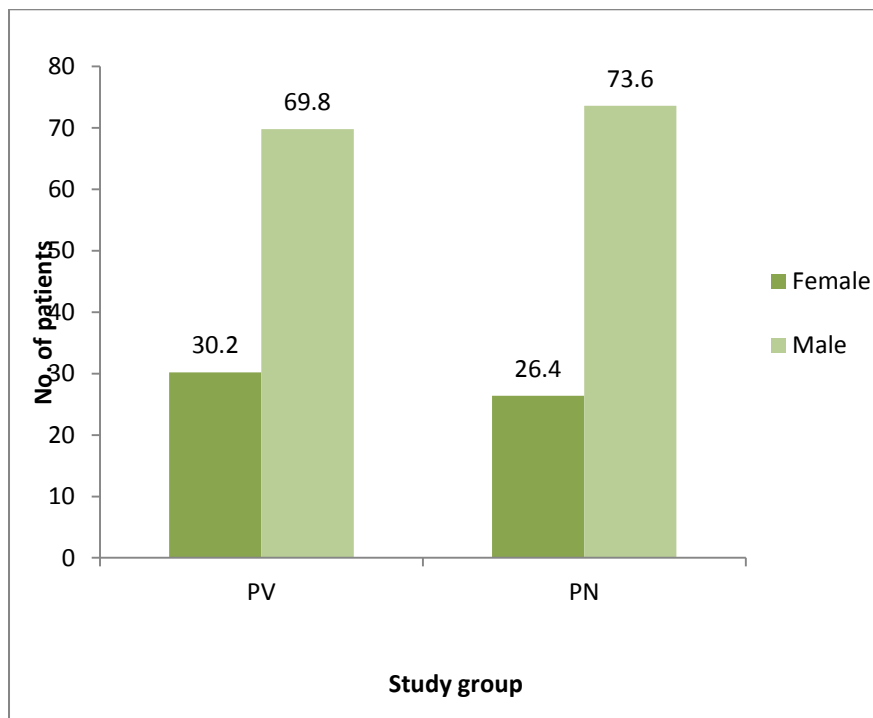
Table No 4: Comparison of Gender between Group I (PV) and Group II (PN)

Gender	Group I (PV)	Percentage	Group II (PN)	Percentage	Chi square test
Female	16	30.2	14	26.4	P=0.6663 NS
Male	37	69.8	39	73.6	
Total	53	100.0	53	100.0	

The numbers of female patients randomly selected in Group I (PV) were 16 and in Group II (PN) were 14. The percentage of randomly selected female patients was 30.2% in Group I (PV) and 26.4% in Group II (PN). The numbers of male patients randomly selected in Group I (PV) were 37 and in Group II (PN) were 39. The percentage of randomly selected male patients was 69.8% in Group I (PV) and 73.6% in Group II (PN). The gender distribution between two groups was not statistically significant. (p value> 0.05) and sex was comparable in two groups.

Table No 5: Distribution of patients according to ASA Grades

ASA Grades	Group I (PV)	Percentage	Group II (PN)	Percentage
I	42	79	40	75.5
II	11	21	13	24.5
Total	53	100.0	53	100.0

Graph No 3: Distribution of patients according to ASA Grades
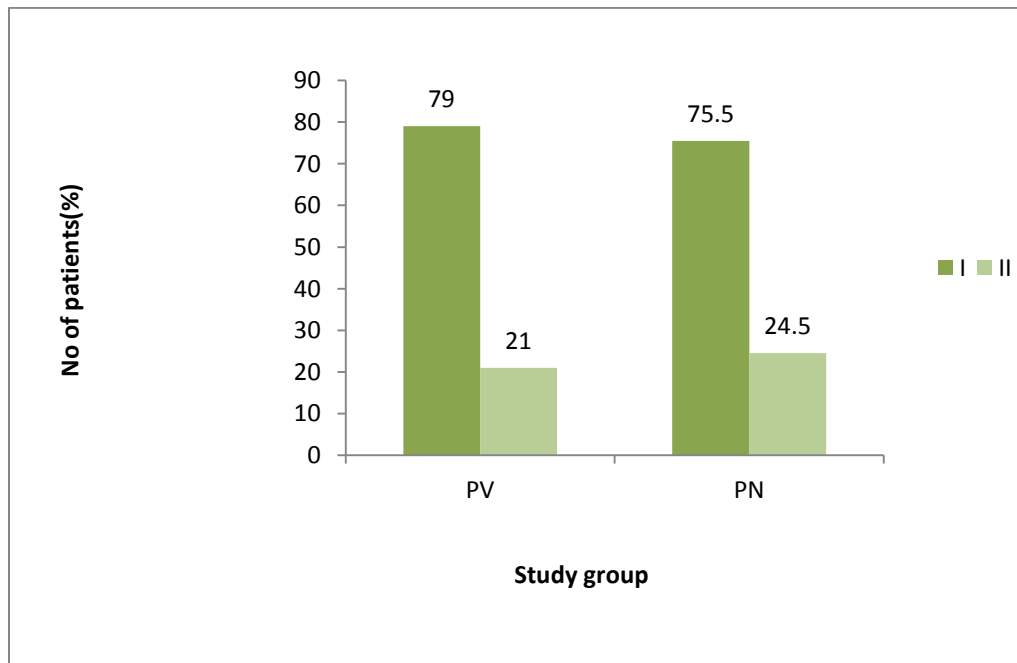
Table No 6: Comparison of ASA Grades between Group I (PV) and

Group II (PN)

ASA Grades	Group I (PV)	Percentage	Group II (PN)	Percentage	Chi square test
I	42	79	40	75.5	P=0.6425 NS
II	11	21	13	24.5	
Total	53	100.0	53	100.0	

The numbers of patients randomly selected with ASA Grade I in Group I (PV) were 42 and in Group II (PN) were 40. The percentage of randomly selected patients with ASA Grade I was 79% in Group I (PV) and 75.5% in Group II (PN). The numbers of patients randomly selected with ASA Grade II in Group I (PV) were 11 and in Group II (PN) were 13. The percentage of randomly selected patients with ASA Grade II was 21% in Group I (PV) and 24.5% in Group II (PN). Both the groups were comparable according to ASA distribution. (p value>0.05).

Table No 7: Imaging time comparison between Group I (PV) and Group II (PN)

Imaging Time [I] (sec)	N	Mean ± S.D.	Mann Whitney 'U' test
Group I (PV)	53	106.34±19.083	P<0.0001*
Group II (PN)	53	132.83 ±6.173	Difference is significant

Graph No 4: Imaging time comparison between Group I (PV) and Group II (PN)
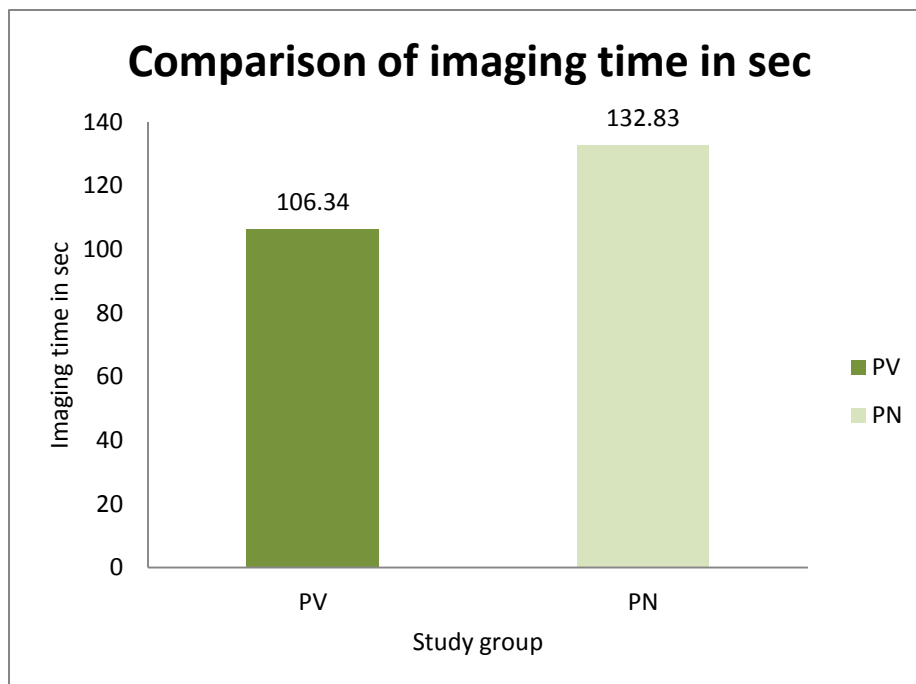
In Group I (PV), mean imaging time was 106.34±19.083 sec while in Group II (PN) it was 132.83±6.173sec, which was statistically significant (p<0.05).

Table No 8: Needling time comparison between Group I (PV) and Group II (PN)

Needling Time [N] (Sec)	N	Mean ± S.D.	Mann Whitney 'U' test
Group I (PV)	53	412.36 ± 28.192	P<0.0001*
Group II (PN)	53	739.09 ± 11.314	Difference is significant

Graph No 5: Needling time comparison between Group I (PV) and Group II (PN)
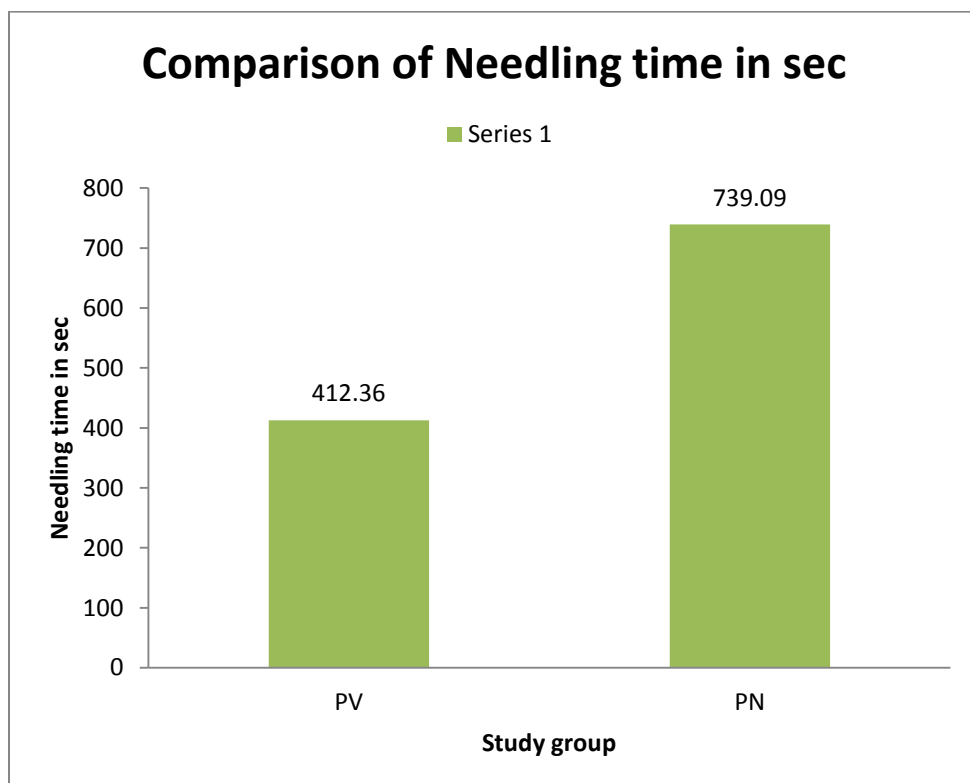
In Group I (PV), mean needling time was 412.36±28.192 sec whereas in Group II (PN), and it was 739.09±11.314sec which was statistically significant (p value<0.05)

Note: *Significance at 5% level of significance (p<0.05)

Table No 9: Performance time comparison between Group I (PV) and Group II (PN)

Performance Time[P[I+N}] (Min)	N	Mean ± S.D.	Mann Whitney 'U' test
Group I (PV)	53	8.647±0.5486	P<0.0001*
Group II (PN)	53	14.53±0.2092	Difference is significant

Graph No 6: Performance time comparison between Group I (PV) and Group II (PN)
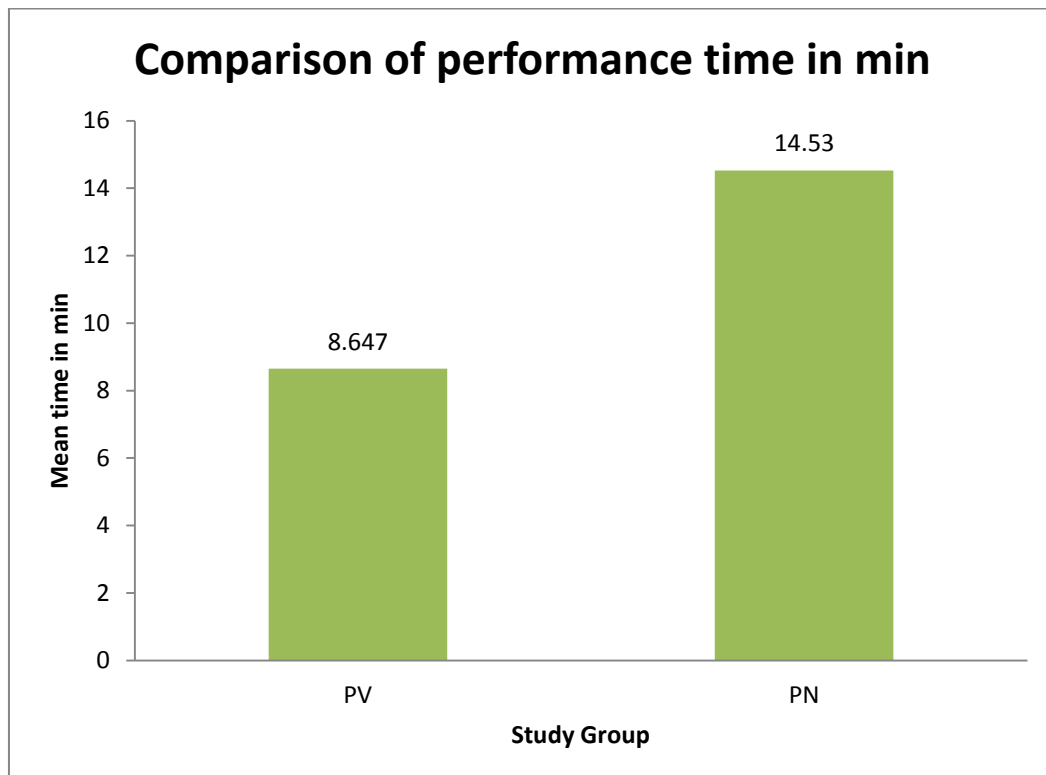
In Group I (PV), mean performance time was 8.6470±0.5486 min while in second Group II (PN), it was 14.53± 0.2092 min. Difference between the two was statistically significant (p<0.05).

Table No 10: Onset time comparison between Group I (PV) and Group II (PN)

Onset Time (Min)	N	Mean ± S.D.	Mann whitney 'U' test P Value
Group I (PV)	49	19.48 ± 2.82	P<0.0001*
Group II (PN)	50	13.86 ± 1.81	Difference is significant

Graph No 7: Onset time comparison between Group I (PV) and Group II (PN)
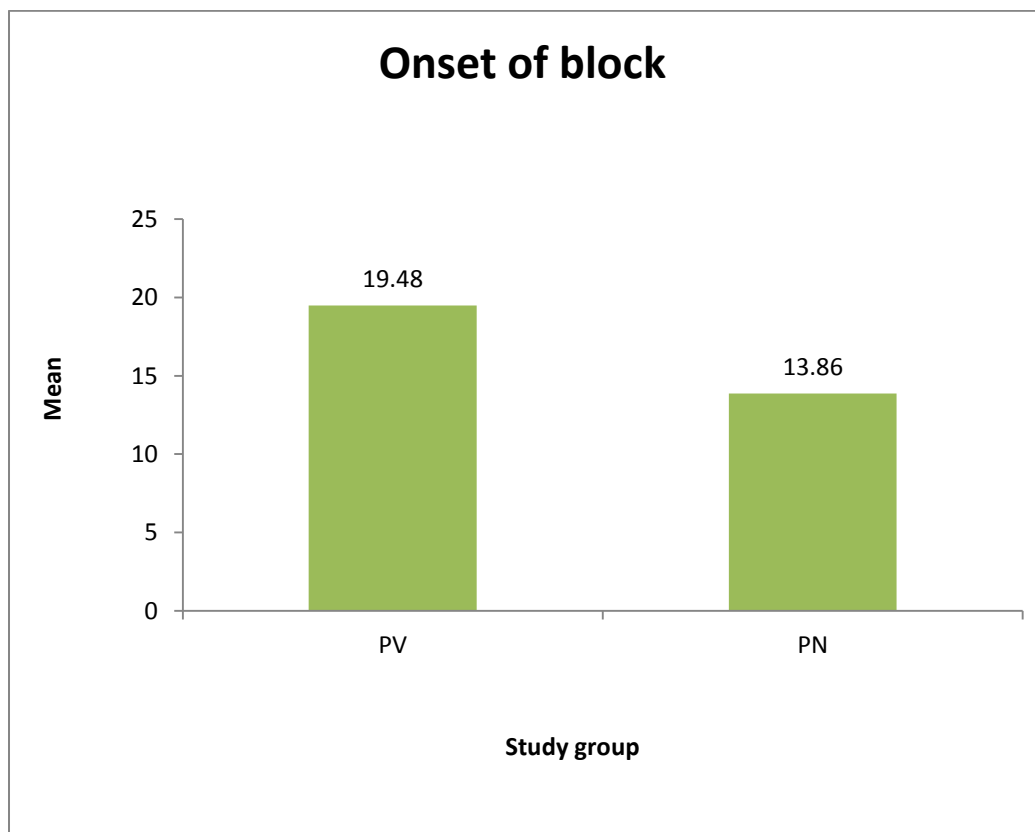
In Group I (PV) onset time was 19.48±2.82 min where as in Group II (PN) it was 13.86±1.81 min which is statistically significant (p value being <0.05).

Table No 11: Comparison of number of needle passes between Group I (PV) and Group II (PN)

Number Of Needle Passes	N	Mean ± S.D.	Mann whitney 'U' test P Value
Group I (PV)	53	2.31 ± 0.50	P<0.0001*
Group II (PN)	53	4.90 ± 0.66	Difference is significant

Graph No 8: Comparison of number of needle passes between Group I (PV) and Group II (PN)
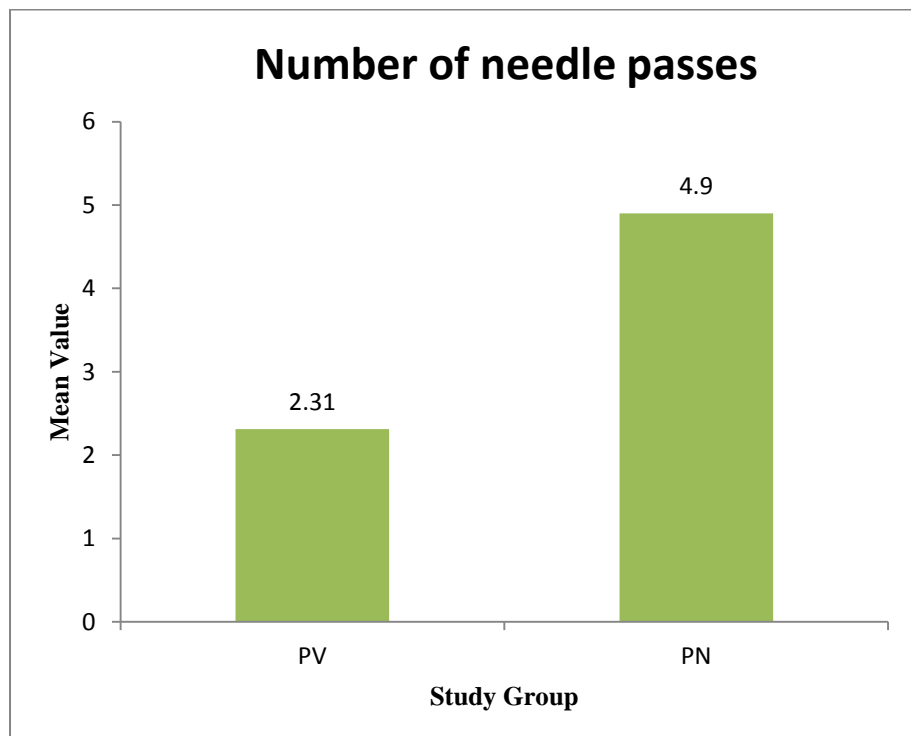
In Group I (PV) mean number of needle passes was 2.31±0.50 where as in Group II (PN) it was 4.90±0.66 which is statistically significant (p value being <0.05).

Table No 12: Distribution of patients according to Block success

Block success	Group I (PV)	Percentage	Group II (PN)	Percentage
Yes	49	92.5	50	94.3
No	4	7.5	3	5.7
	53	100.0	53	100.0

Graph No 9: Distribution of patients according to Block success
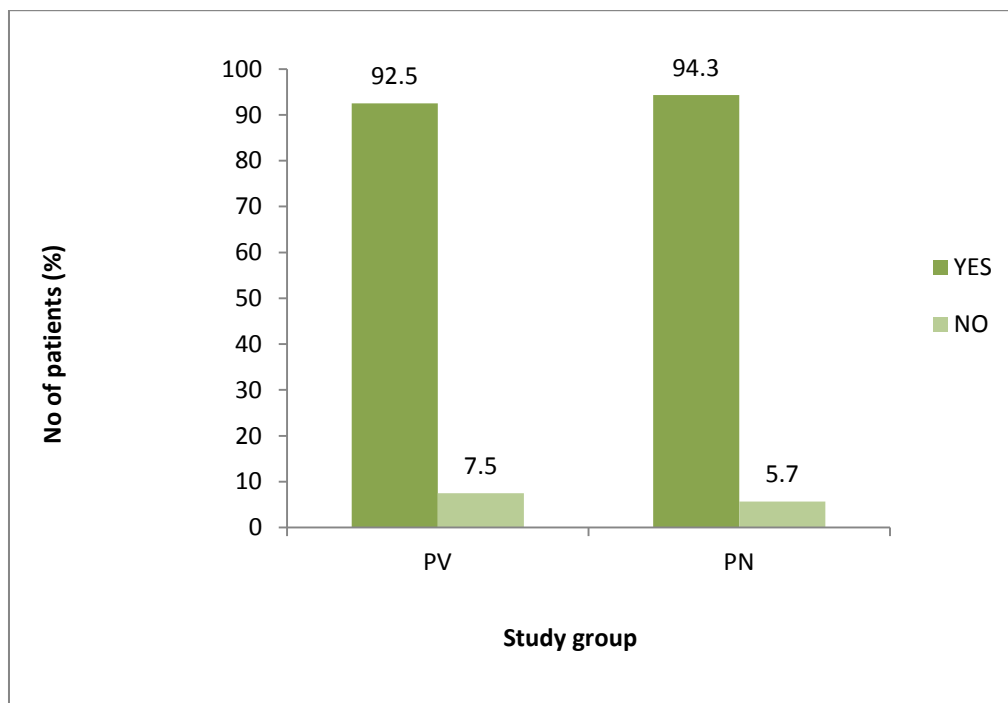
Table No 13: Comparison of Block success between Group I (PV) and Group II (PN)

Block success	Group I (PV)	Percentage	Group II (PN)	Percentage	Chi square test
No	4	7.5	3	5.7	P=0.6957 NS
Yes	50	92.5	49	94.3	
	53	100.0	53	100.0	

There were 4 cases of failure in Group I (PV) and 3 cases of failure in Group II (PN). The success rates of Group I (PV) was 92.5% and Group II (PN) was 94.3%. The success rates of Group I (PV) and Group II (PN) was comparable and the difference is not statistically significant (p value>0.05).

Table No 14: Comparison of duration of Sensory block between Group I (PV) and Group II (PN)

Duration of sensory block (Min)	N	Mean ± S.D.	Mann whitney 'U' test P Value
Group I (PV)	49	736.0 ± 12.45	0.1449
Group II (PN)	50	730.71 ± 17.73	Difference is not significant

Graph No 10: Comparison of duration of Sensory block between Group I (PV) and Group II (PN)
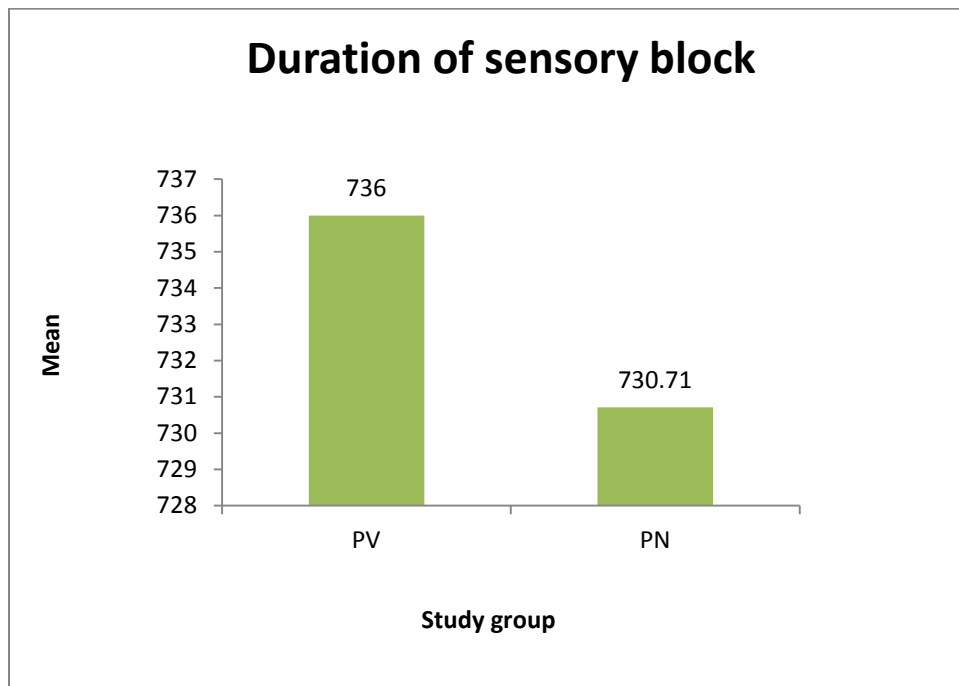
In Group I (PV) duration of Sensory block was 736.0 ±12.45min where as in Group II (PN) it was 730.73 ±17.73min which was not statistically significant (p value being >0.05).

Table No 15: Comparison of duration of motor block between Group I (PV) and

Group II (PN)

Duration of motor block (Min)	N	Mean ± S.D.	Mann whitney 'U' test P Value
Group I (PV)	50	642.55 ± 11.51	0.065
Group II (PN)	49	638.6 ± 11.56	Difference is not significant

Graph No 11: Comparison of duration of motor block between Group I (PV) and
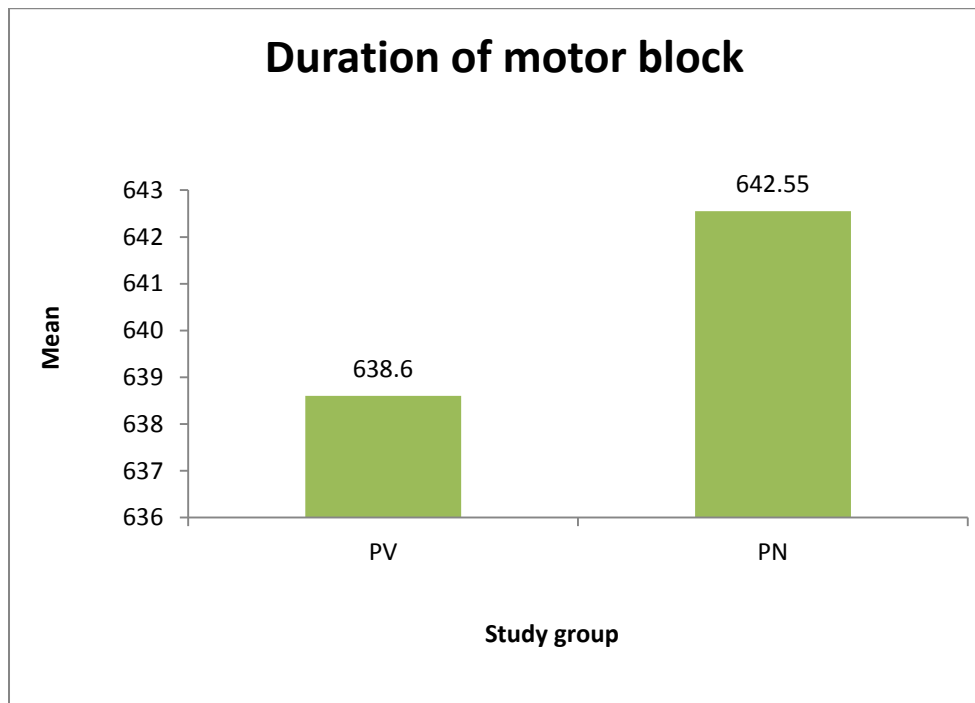
Group II (PN)

In Group I (PV) duration of motor block was 638.6 ±11.56min where as in Group II (PN) it was 642.55±11.51min which was not statistically significant (p value being >0.05)

Table No 16: Distribution of patients according to Local anaesthetic toxicity (LA)

Local anaesthetic toxicity (LA)	Group I (PV)	Percentage	Group II (PN)	Percentage
No	53	100.0	53	100.0
Yes	0	0	0	0
Total	53	100.0	53	100.0

Graph No 12: Distribution of patients according to Local anaesthetic toxicity (LA)
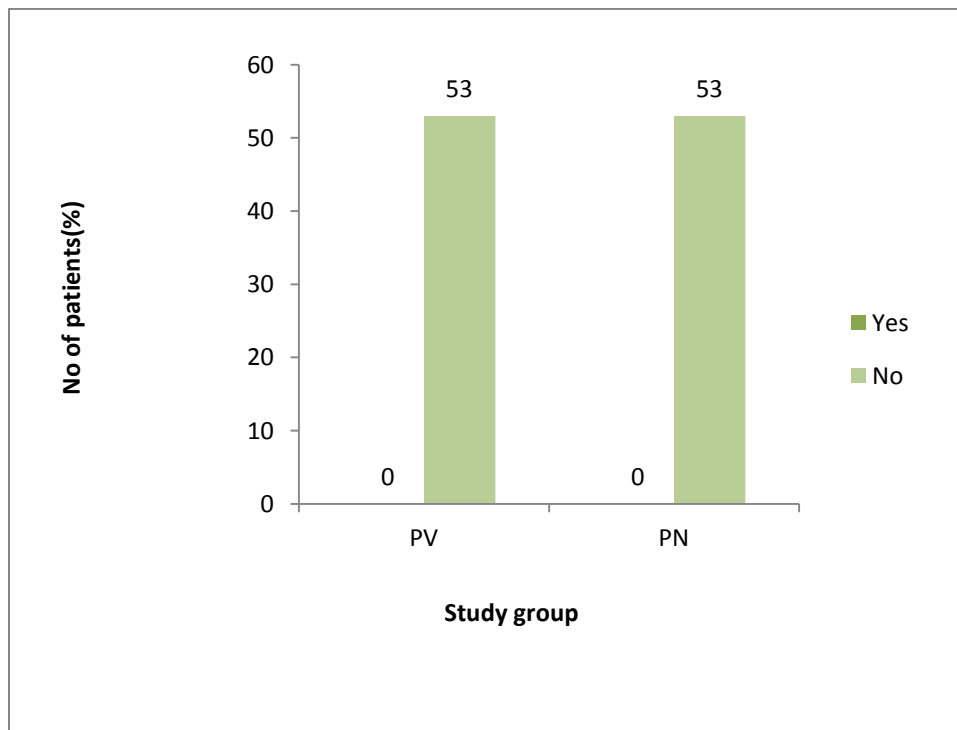
There were no incidences of local anaesthetic toxicity among both Group I (PV) and Group II (PN) group.

Table No 17: Distribution of patients according to Vascular puncture n(%)

Vascular puncture n (%)	Group I (PV)	Percentage	Group II (PN)	Percentage
0	43	81.1	53	100
1	8	15.1	0	0
2	2	3.8	0	0
Total	53	100.0	53	100.0

Graph No 13: Distribution of patients according to Vascular puncture n(%)
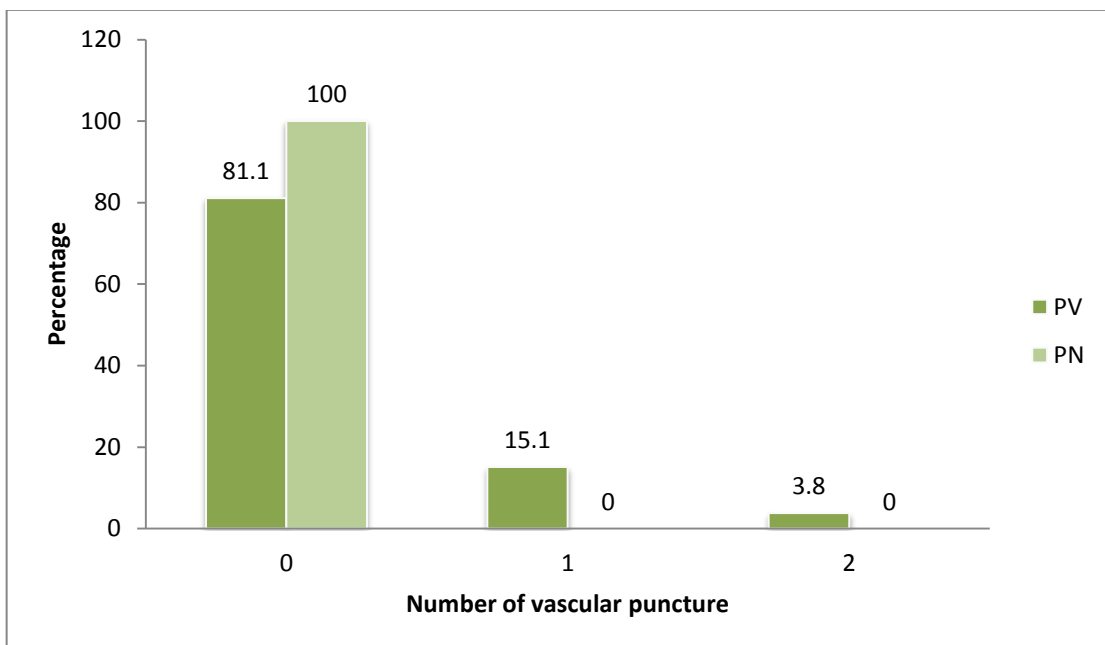
10 patients in Group I (PV) had vascular puncture out of which 8 patients had vascular puncture once and 2 patients had vascular puncture twice **.**18.9% of patients in Group I (PV) had vascular puncture; there were no vascular puncture in Group II (PN).

Table No 18: Comparison between Group I (PV) and Group II (PN)

Variables	Group I (PV) Mean ± S.D.	Group II (PN) Mean ± S.D.	Mann Whitney 'U' Test
Age	38.302 ± 12.8146	36.21 ± 12.253	P=0.4965 NS
Weight	58.4340 ± 6.92406	59.06 ± 6.49383	P=0.6440 NS
Imaging Time (Sec)	106.34 ± 19.083	132.83 ± 6.173	P<0.0001*
Needling Time(Sec)	412.36 ± 28.192	739.09 ± 11.314	P<0.0001*
Performance Time(Min)	8.647 ± 0.5486	14.53 ± 0.2092	P<0.0001*
Onset Of Block (Min)	19.48 ± 2.83	13.86 ± 1.81	P<0.0001*
No. Of Needle Passes	2.31 ± 0.503	4.90 ± 0.66	P<0.0001*
Duration Of Sensory Block, Min	736.0 ± 12.45	730.71 ± 17.73	P=0.1449 NS
Duration Of Motor Block, Min	638.6 ± 11.56	642.55 ± 11.51	P=0.065 NS

DISCUSSION

The brachial plexus block is routinely performed for surgeries of upper limb. Brachial plexus blocks provide a useful alternative to general anaesthesia for upper limb surgeries for their several advantages over general anaesthesia. Axillary brachial plexus block is relatively simple and safe among the four approaches to brachial plexus. With the advent of ultrasound technology, there is a marked improvement in the success rate, shorter onset time and reduction in the volume required for successful block.[40]

The ability to correctly identify nerves and put an adequate amount of local anaesthetic around them so that there is complete impregnation of nerves, forms the basis of brachial plexus block. The established methods of nerve location were based on either paraesthesia elicitation or identification of the proper motor response on nerve stimulation. Each of these two techniques has been reported to have a low sensitivity for detection of needle-to-nerve contact.[41] The introduction of Ultrasound guidance into clinical practice as a possible option to identify peripheral nerves, offers the potential advantage of optimizing the spread of the local anaesthetic solution around the nerves under sonographic vision.[42]

Just as nerve stimulator guided technique, an ultrasound-guided brachial plexus block can be performed in various places like axilla, supraclavicular, infraclavicular, and interscalene. There are many reports comparing various methods for nerve stimulator guided technique. Many researchers have compared the ultrasound-guided technique to the nerve stimulator guided technique, but there are not many comparative studies between the various methods for ultrasound-guided nerve blocks.

While comparing the methods for the brachial plexus block, the success rate has been reported as the most important indicator. But, with ultrasound's introduction the success rate has reached 95-100%.[30, 31] Therefore, a different indicator is required in comparative studies today, the block performance time and the onset time are considered as an important indicator after the success rate.[43]

As the anaesthetic time is delayed, the turnover ratio of the operation theatre decreases and the inconvenience the patients are subjected to increases. Anaesthetic time is the sum of the performance time and onset time. Just as the onset time, the performance time also causes inconvenience to the patients. In our study performance time was taken as sum of imaging and needling times. Therefore, primary aim of this study was to determine the performance time, onset time and block success rate. Secondary aims were to compare duration of motor and sensory block and incidence of adverse events during the performance of USG guided ABPB for upper limb surgeries.

We conducted a prospective randomized study on 106 patients ASA I and ASA II to compare USG guided perivascular axillary brachial plexus block and perineural axillary brachial plexus block for upper limb surgeries. 106 patients undergoing forearm, wrist and hand surgeries were randomly allotted in two groups. Group I (PV) (n=53) received perivascular axillary block and Group II (PN) (n=53) received perineural axillary block. In both methods, volume of drug used was 20 ml. The drugs used were 0.5%bupivacaine 8ml, 2% lignocaine with adrenaline 10ml and dexamethasone 8mg (2ml). In both methods, musculocutaneous nerve was blocked with 5ml out of 20ml volume of the drug. In perivascular technique, remaining 15ml

volume of the drug was deposited anterior and posterior to the artery where as in perineural technique 5ml of drug was targeted at each of radial, ulnar, median nerve after sonographic visualization.

All the patients in two groups were comparable with respect to the demographic parameters: age, sex and weight.

In the Group I (PV) the mean age was 38.30±12.81 years and in Group II (PN) was 36.21±12.25years. Group I (PV) consisted of 16 females and 37 males whereas the Group II (PN) consisted of both 14 females and 39 males. The mean weight of patients in Group I (PV) was 58.43±6.92 kg and in Group II (PN) was 59.06±6.49 kg.

In our study, the mean imaging and needling time for Group I (PV) were 106.34±19.08sec and 412.36±28.19sec respectively. The mean imaging time and needling time for Group II (PN) were 132.83±sec and 739.09±11.31sec respectively. The Group II (PN) required more time because we had to identify all the four nerves and then deposit local anaesthetic at each nerve. In comparison the Group I (PV) required only the identification and imaging of musculocutaneous nerve and axillary artery. Hence, both imaging time and needling time were greater in Group II (PN). The total performance time which is sum of imaging time and needling time was more in Group II (PN) than Group I (PV) group. The mean performance time in Group I (PV) was 8.647±0.55mins and in Group II (PN) was 14.53±0.21mins.The difference was statistically highly significant (p<0.001).

In a similar study done by Francisca Bernucci *et al* in 2012[5], the mean imaging and needling times for perivascular group were 0.75min (45sec) and 7.5mins (450sec) and for perineural group were 2.45min (147sec) and 13.2 min (792sec). The

mean performance time in this study was 8.2 mins for Group I (PV) and 15.7 mins for Group II (PN). These results are consistent with our study except that we took more time for imaging in perivascular technique. In 2009, DQ Tran *et al* [20] did a prospective randomized comparison between USG guided supraclavicular, Infraclavicular and axillary blocks. Axillary block was given by perivascular technique. The mean imaging and needling time for axillary block were 1min and 7.35mins. The mean performance time was 8.5mins. In another study done by DQ Tran *et al* in 2012[21] to compare double, triple, quadruple USG guided axillary block, the needling time for double injection technique was 1.3mins and imaging time for same was 9.5mins. The mean performance time for double injection technique was 11mins. Thus, we took a comparatively more time for imaging and needling. In above studies the blocks were mostly performed by trained persons. In our study, our being a tertiary learning institute blocks were performed by junior residents under the guidance of teachers. Hence, the time delay is attributed to the learning curve.

The mean number of needle passes in Group I (PV) was 2.30±0.50 and in Group II (PN) group was 4.90±0.66. The difference was significant (p<0.001). In the study by Francisca Bernucci *et al* [5] in 2012, the PV technique required fewer needle passes 3.5 [SD, 1.0] vs 8.2 [SD, 2.2]; P = 0.000).

The mean onset time in Group I (PV) was 19.48±2.83mins whereas for Group II (PN) the mean onset time was 13.86±1.81mins. In the study by Francisca Bernucci *et al* [5] in 2012, the mean onset time for Group I (PV) was 18.9mins and for Group II (PN) group was 13.8mins. In other study by DQ Tran *et al* [20] in 2009, the mean onset time for perivascular axillary block was 17.8mins. Another study done by DQ Tran *et al* in 2012 to compare double, triple, quadruple USG guided axillary block, the mean onset time for axillary block was 18.6mins. The difference in this time can

be related to the drug used and its storage. The various theories favouring the accelerated onset and improved block consistency for the Group II (PN) group are that the local anaesthetics may have been delivered more intimately to nerves by targetingq each nerve. The ratio of neural to non-neural tissue is different along the brachial plexus and may influence individual nerve permeability to local anaesthetic. Another possible mechanism is involvement of threshold number of nodes of Ranvier for conduction block. More likely, however, was the greater surface area of neural tissue available to local anaesthetic in the combined group compared with the Group I (PV). Thus Group II (PN) had shorter onset time as compared to Group I (PV) in our study. The difference was highly significant (p<0.001).

The success of block which we have defined as the surgery getting completed without any other form of anaesthesia being required was comparable in both groups. There were 4 cases of failure in Group I (PV) out of 53 cases whereas in Group II (PN) 3 cases of failure. The success rate was 92.5 in Group I (PV) and 94.3 in Group II (PN). In those 7 cases 4 received IV Inj Fentanyl 1mcg/kg, 1patient received rescue block and 2patient received general anaesthesia. The patients in whom block failed were excluded from calculation of onset time, duration of motor & sensory block in both groups.

This difference may be due to the fact that in PN technique we target each nerve in comparison to PV technique where we inject drug around the axillary artery. Hurried approach done in operation theatre due to unavailability of block room must have also contributed to failure.

In our study the only complication seen was vascular puncture in Group I (PV) in 10 cases out of 53 patients. The needle was redirected, and block given successfully. No complications were seen in Group II (PN) patients. Sites *et al* [44] had reported that the most common error occurring while giving blocks with USG guidance is failure to visualize entire needle length before advancement. This vascular puncture can be reflected by this study. During performance of the block any incidence of hematoma formed due to vascular puncture was treated by application of pressure and performance of the block continued.

In our study the duration of motor block in Group I (PV) was 638.60± 11.56min and in Group II (PN) was 642.55±11.51min. The duration of sensory block in Group I (PV) was 736.0±12.45min and in Group II (PN) was 730.71±17.73min.

In our study there were no incidence of convulsions and paraesthesia in both the groups. The vital parameters like heart rate, blood pressure and saturation values were similar in both groups.

In our study, we found that perivascular axillary brachial plexus block has shorter performance time and fewer needle passes as compared to perineural axillary block in upper limb surgeries, but onset time was shorter in perineural block. Therefore USG guided ABPB offers many clinical advantages that contribute to improved patient outcome as well as lower healthcare costs.

CONCLUSION

USG guided perivascular ABPB is a simple technique compared to USG guided perineural ABPB as we have to identify only one structure in the perivascular ABPB. USG guided Perivascular ABPB is better than USG guided perineural ABPB in mean imaging time, mean needling time and mean performance time, but onset time was shorter in perineural block.

Though USG guided ABPB technique provides direct visualisation of block performance but does not completely eliminate risk of intravascular and intraneural injection. With undue precautions while performing the procedure USG guided Perivascular ABPB can be a safe and effective regional technique suitable for upper limb surgeries compared to USG guided perineural ABPB in elective and emergency care.

SUMMARY

We conducted a randomized prospective study on 106 healthy patients ASA I and ASA II to compare "**Ultrasound guided perivascular axillary block and perineural axillary block** "Valid consent was obtained. Pre-operatively patients were explained   about the study. 106 patients after written consent were randomly   allotted in two group: Group I PV (n=53)- to receive USG guided perivascular axillary block and Group II PN(n=53) to receive USG guided perineural axillary block. The imaging time, needling time, performance time, onset time, total anaesthesia related time, success of block and complications were all recorded.

In our study, age, sex, weight are not confounding factors and hence study is comparable on the basis of demographic data.

We found that,

- The mean imaging time in Group I (PV) was 106.34±19.083sec and in Group II (PN) group was 132.83±6.17sec (p<0.0001).

- The mean needling time in Group I (PV) was 412.36±28.19sec and in Group II (PN) was 739.09±11.31sec (p<0.0001).

- The mean performance time in Group I (PV) was 8.647±0.54min and in Group II (PN) group was 14.53±0.20min (p<0.0001).

- The mean number of needle passes in Group I (PV) was 2.31±0.50 and in Group II (PN) group was 4.90±0.66 (p<0.0001).

- The mean onset time in Group I (PV) was 19.48±2.83min and in Group II (PN) was 13.86±1.81min (p<0.0001).

For all above, p was <0.05 and the difference were statistically significant.

- The mean duration of motor block in Group I (PV) was 638.6 ± 11.51min and in Group II (PN) was 642.55±11.51 min. Both group were comparable in this respect (p >0.05).

- The mean duration of sensory block in Group I (PV) was 736.0 ±12.45min and in Group II (PN) was 730.71± 17.73min. Both group were comparable in this respect (p >0.05)

- The success of block was comparable in both groups.

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ANNEXURES

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE
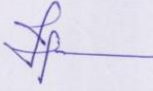
INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04/10/2016 at 3-00pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "A comparative Clinical study of periv ascecular and peripheral axillary Brachial plexus block for upper limb surgeries,,

Name of P.G. student ___Vinuta Vastrad___
___Dept of Anaesthesiology___

Name of Guide/Co-investigator Dr ___Sridevi Malimane___
___Associate professor in Anaesthesiology___

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

<u>Following documents were placed before E.C. for Scrutinization</u>
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT : **"A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES"**

PRINCIPAL INVESTIGATOR: **Dr. VINUTA VASTRAD**

Department of Anesthesiology

BLDE (Deemed to be University)

Shri B.M. Patil Medical College Hospital & Research Centre, Sholapur Road Vijayapur-03

PG GUIDE **: Dr. SRIDEVI MULIMANI**

Associate Professor, Dept. of Anesthesiology

BLDE (Deemed to be University)

Shri B.M. Patil Medical College Hospital & Research Centre, Sholapur Road Vijayapur-03

PURPOSE OF RESEARCH:

I have been informed that this study is: **"A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK   FOR UPPER LIMB SURGERIES"**

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I understand that I will be participating in the study: **"A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK   FOR UPPER LIMB SURGERIES"**

RISKS AND DISCOMFORTS:

I understand that my ward may experience some pain during the procedure and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that my wards participation in this study will help in finding out: **"A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK   FOR UPPER LIMB SURGERIES".**

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time.**Dr. VINUTA VASTRAD** is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that **Dr. VINUTA VASTRAD** will terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly due to my participation in this study, such injury will be reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: **Dr. VINUTA VASTRAD**

                                                                  (Investigator)

Patient's signature   Witness

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. VINUTA VASTRAD** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give my consent to participate as a subject in this research project.

_____   _____

(Participant)   Date

_____   _____

(Witness to above signature)   Date

SCHEME OF CASE TAKING:

PROFORMA

STUDY: "A COMPARATIVE CLINICAL STUDY OF USG GUIDED PERIVASCULAR AND PERINEURAL AXILLARY BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES"

PATIENT DETAIL: DATE:

- Name: Age/ Sex: IP No: Wt:

  Ward: Group allotted by randomization: Group I / Group II

- 1. Type of the surgery: Duration   of   surgery (min):

  2. Indication:

- Significant History:

- General Physical Examination:

  Pallor   Icterus Cyanosis   Clubbing   Koilonychia

  Lymphadenopathy Oedema

  Teeth   Dentures

- Vital Parameters

  Pulse   Blood Pressure Respiratory Rate   Temperature

- Systemic Examination

  - Cardiovascular system

  - Respiratory system

  - Central nervous system

  - Per abdomen

- Airway Assessment:

  Mallampatti Grade: Cervical Spine:

  Mouth Opening: Neck Movement:

- I n v e s t i g a t i o n

  Hemoglobin: TLC:

  S.Urea: S.Creatinine:

  LFT's: Platelet count:

  Urine routine:

  Chest X-ray (PA view):   ECG:

  - ASA grade:

  - Procedure

- Premedication

- Thorough intra-operative monitoring will be carried out and complications will be

  looked for post-operatively for 24hrs.

X. Parameters

Block Performance Data		
Parameters	Group I (PV)	Group II (PN)
Imaging time, s		
Needling time, s		
Performance time, min		
Onset of block, min		
Block success rate (%)		
No. Of needle passes		
Duration of the sensory block, min		
Duration of the motor block, min		
COMPLICATIONS		
Parameters	Group I (PV)	Group II (PN)
Vascular puncture, n (%)		
Local anaesthetic toxicity(LA)		

Date   Signature

KEY TO THE MASTER CHART

ASA grade   - American society of anaesthesiologists' grade

IP NO   - Inpatient number

LA   - Local anaesthetic toxicity

Min   - Minutes

PN   - Perineural

PV   - Perivascular

S   - Seconds

SL NO   - Serial number