A dissertaiton on

A COMPARATIVE STUDY OF 150µg OF BUPRENORPHINE WITH 0.3% BUPIVACAINE AND 0.3% BUPIVACAINE ALONE IN BRACHIAL PLEXUS BLOCK BY LOW INTERSCALENE APPROACH IN UPPER LIMB SURGERIES

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CERTIFICATE

This is to certify that the dissertation titled **"A COMPARATIVE STUDY OF 150µg OF BUPRENORPHINE WITH 0.3% BUPIVACAINE AND 0.3% BUPIVACAINE ALONE IN BRACHIAL PLEXUS BLOCK BY LOW INTERSCALENE APPROACH IN UPPER LIMB SURGERIES"** presented herein DR.R.RAJASEKAR is an original work done in the Department of Anaesthsiology, Govt.Stanley Medical College and Hospital, Chennai for the award of the degree of M.D. (Branch X) Anaesthsiology under my guidance and supervision during the academic period of 2004-2006.

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DECLARATION

I, Dr. R. RAJASEKAR solemnly declare that the dissertation titled "A COMPARATIVE STUDY OF 150µg OF BUPRENORPHINE WITH 0.3% BUPIVACAINE AND 0.3% BUPIVACAINE ALONE IN BRACHIAL PLEXUS BLOCK BY LOW INTERSCALENE APPROACH IN UPPER LIMB SURGERIES" is a bonafide work done by me in the department of Anaesthsiology, Stantly Medical College and Hospital, Chennai under the able guidance of Prof.R.Meenakshmi, M.D., D.A., Professor and HOD, Department of Anaesthsiology, Govt. Stanley Medical College and Hospital, Chennai-600 001.

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CONTENTS

INTRODUCTION

Regional anaesthesia is the blocking of peripheral nerve conduction in a reversible way by using local anesthetic agents, thereby one region of the body is made insensitive to pain and is devoid of reflex response to surgical stimuli. In this the CNS is spared, so that the patient is conscious, fully aware during the surgical procedure without recognizing pain.

Regional anaesthesia has many advantages over general anaesthesia for surgeries on upper extremities particularly in emergency surgeries.

They are:

(i) Proven to be the safest anaesthesia for high risk patients who are in greater risk due to stress imposed by general anaesthesia

(ii) Only method of anaesthesia which prevents all afferent impulses from the site of surgery reaching the CNS. Hence the need of poly pharmacy and its side effects are eliminated.

(iii) Along with complete pain relief and total muscle relaxation it produces vasodilatation , which improve blood circulation, and prevents tissue hypoxia.

(iv) Causes least disturbance to the normal physiology than any other type of anaesthesia.

(v) Postoperative pain relief is ensured for a longer duration by using long acting anaesthetic drug and for several days if continuous block using catheter technique is employed.

(vi) Many intra operative, postoperative complication of general anaesthesia are avoided.

(vii) It is cost effective and safe.

(viii) Avoids theatre pollution .

(ix) Safest technique for patients with full stomach.

The use of pneumatic tourniquet provides a bloodless field during upper extremity surgeries. The tourniquet pain is a concern because of the technical difficulty to block individual nerves. Brachial plexus block by supraclavicular approach is the solution in such a situation. There are different approaches for blocking brachial plexus, the common approaches are

(a) Supraclavicular approach

(b) Interscalene approach

(c) Axillary approach

Axillary approach has the lowest incidence of serious complication and can be performed with ease. Still there is limitation associated with axillary approach like,

- It is inadequate for operation on the arm and shoulder

- It is difficult to block the musculocutaneous nerve predictably with resultant sparing of the radial aspect of forearm and dorsum of hand.

- Tourniquet pain is not well tolerated

- Also abducting the arm by 90° for giving the block may be painful and even dangerous in traumatic lesions of the upper extremities.

Hence the brachial plexus block by low scalene approach is the method of choice for upper limb surgeries. In 1970 Alon Winne first demonstered interscalene approach of brachial block. William Steward Halsted first performed brachial plexus block in 1885. In 1911 Kulenkampff and Hirschel described the first percutanceous brachial plexus block by supraclavicular and axillary routes respectively.

Since then several techniques have been used to prolong the brachial plexus block.

- ❖ Continues infusion of local anaesthetic through catheters
- ❖ Addition of epinephrine and α_2 agonist like clonidine.
- ❖ Addition of opioids are being used as adjuvant to local anaesthetic solution e.g. Morphine, Buprenorphine and butorphanol.

Buprenorphine, a thebaine derivative of semi synthetic opioid compound acts both on mu-opioids receptor and Kappa receptors. It is used for pain relief and this is given by intra

muscular, intra venous, epidural and spinal routes and the dose in 0.3 to 0.6mg. It is many times more potent than morphine and it has side effects of nausea, vomiting, respiratory depression etc.

This study compares the effects of addition of Buprenorphine to local anaethetic solution with plain local anaesthetic solution for brachial plexus block,with regard to onset, time for total blockade, duration and quality of blockade.

AIM OF STUDY

The opioid receptors are found in the central nervous system. The presence of these receptors in the somatic and sympathetic peripheral nerves has also been documented. In this study an alternate is made to compare the effects of addition of buprenorphine to 0.3% bupivacaine versus 0.3% bupivacaine, and also to evaluate the action of buprenorphine on the peripheral nerve receptors in brachial plexus block.

So, this is the study to

- To compare the effects and actions of 0.3% bupivacaine and addition of 150μg buprenorphine to 0.3% bupivaccine in brachial plexus block by low interscalene approach.

- To compare the onset, time taken for total blockade, duration and intensity of motor and sensory blockade.

- To evaluate the action of opioid , buprenorphine on the peripheral nerves.

ANATOMY OF BRACHIAL PLEXUS

Successful regional anaesthesia of the upper extremity requires knowledge of brachial plexus anatomy from its origin, where the nerves emerge from the inter vertebral foramina, to its termination as peripheral nerves. A through understanding of the plexus throughout its formation and distribution is equally essential in order to master the various techniques of brachial plexus analgesia.

In the course from the intervertebral foramina to the arm, the fibres that constitute the plexus are grouped as roots, trunks, divisions, cords and terminal branches which are formed through a complex process of combining, dividing, recombining and finally redividing.

The brachial plexus is formed by the union of the anterior primary rami of the 5^{th} to 8^{th} cervical nerves and the 1^{st} thoracic nerve with occasional contributions from the 4^{th} cervical (prefixed) above and 2^{nd} thoracic nerve (Post fixed) below. These roots unite to form trunks, which lie in the neck above the clavicle. They pass through the fascial space formed between the scalenes anterior and the scalene medius muscles accompanied by the subclavian artery to form a neurovascular bundle. This fascia continuous as the axillary sheath upto axilla and upper third of arm. Each trunk divides behind the clavicle, into anterior and posterior divisions, which unite in the axilla to form the cords. In the neck the plexus is broad above and converges to the first rib.

RELATION OF BRACHIAL PLEXUS

ANTERIOR RELATIONS

The skin, superficial fascia, platysma and supraclavicular branches of the cervical plexus, the deep fascia and external jugular vein. The clavicle and lower part of the scalenus anterior muscle are also anterior to the roots.

POSTERIOR RELATIONS

Scalenus medias and the long thoracic nerve.

INFERIOR RELATIONS

Related to the first rib.

SUPERIOR RELATIONS

Lies first above and then lateral to the subclavian artery.

SYMPATHETIC CONTRIBUTIONS TO THE PLEXUS

Close to their emergence, the 5^{th} and the 6^{th} cervical nerves each receive a grey ramus from the middle cervical sympathetic ganglion. The 7^{th} and the 8^{th} cervical nerve each receive a grey ramus from the inferior cervical ganglion.

The following is the summary of the formation of the brachial plexus at different levels and its major terminal branches.

TRUNKS

Upper trunk – formed by the anterior rami of C_5 and C_6

Middle trunk – formed by the anterior ramus of C_7

Lower trunk – formed by the anterior ramus of C_8 and T_1

DIVISIONS

Above the I^{st} rib each trunk divides into anterior and posterior divisions.

CORDS

(i) The Lateral cord by anterior divisions of upper and middle trunks ($C_5 – C_7$)

(ii) Medial cord – Anterior division by lower trunk ($C_8 – T_1$)

(iii) Posterior cord – Posterior divisions of all the three trunks ($C_5 – T_1$)

BRANCHES

Branches are given off from

- Roots
- Trunks
- Cords

FROM ROOTS

- Nerve to Serratus anterior (C_5, C_6, C_7)
- Muscular branches to Longus Cervices ($C_5 – C_8$)
- Nerve to three scalene ($C_5 – C_8$)
- Nerve to Rhomboids (C_5)
- A twig to phrenic nerve (C_5)

FROM THE TRUNKS

- Supra Scapular nerve (C_5 and C_6)
- Nerve to Subclavius (C_5 and C_6)

FROM THE CORDS

- **From Lateral Cord**

  (i) Lateral pectoral (C_5 - C_7)
  (ii) Lateral root of the Median (C_5 - C_7)
  (iii) Musculo Cutaneous (C_5 - C_7)

- **From Medial Cord**

  (i) Medial root of median nerve (C_8 - T_1)
  (ii) Medial Pectoral (C_8 - T_1)
  (iii) Medial Cutaneous N.of forearm (C_8 - T_1)
  (iv) Medial Cutaneous N.of arm (C_8 - T_1)
  (v) Ulnar (C_8 - T_1)

- **Posterior Cord**

  (i) Upper subscapular nerve ($C_5 – C_6$)

  (ii) Lower subscapular nerve ($C_5 – C_6$)

  (iii) Thoracodorsal ($C_6 – C_8$)

  (iv) Radial (C_5 - T_1)

  (v) Axillary (C_5 _ C_8)

The anaesthesiologist must have familiarity with the perineural structures that surround and accompany the brachial plexus as it leaves the vertebral column in its course to the upper arm and it is as important as the knowledge of the formation and distribution of the neural plexus itself. Palpable muscular and vascular landmarks allow accurate location of the plexus percutaneously. An appreciation of the fascial relations is absolutely essential since this is the basis for all the perivascular techniques.

After leaving the intervertebral foramina, the anterior primary rami of the nerves destined to become the brachial plexus travel in the gutter formed by the anterior and posterior tubercles of the corresponding transverse processes of the cervical vertebrae. After leaving the transverse process, the roots of the plexus descend in front of the middle scalene muscle, which arise from the posterior tubercles of the transverse process of the lower six cervical vertebrae. The insertion of this muscle on the firs rib is separated from that of the anterior scalene muscle by the inferior trunk of the brachial plexus. The anterior scalene muscle arises from the anterior tubercles of the transverse process of the third, forth, fifth and sixth

cervical vertebrae and inserts on the scalene tubercle of the first rib thus separating the subclavian artery from the subclavian vein.

The fascia covering both the scalene muscles is derived from the prevertebral fascia, which splits to invest these muscles, and then fuses again at their lateral margins to form an enclosed interscalene space. Therefore, as the roots leave the transverses processes they emerge between two wall of the fascia covering the anterior and middle scalene muscles. In their descent toward the first rib to form the trunks of the plexus the roots may be considered to be sandwiched between the anterior and medial scalene muscles, the fascia of which serves as a sheath of the plexus. As the trunks approach the first rib they are arranged (as their designations – superior, middle and inferior-imply) one above the other vertically.

As the trunks of the plexus cross the first rib they are joined by the subclavian artery which lies in a plane anterior to the trunks, so that the inferior trunk lies behind the artery in the subclavian groove with the middle and superior trunks above the level of the vessel. At this level the artery and trunks are moving laterally across the ribs and invaginate the scalene fascia to form the subclavian perivascular space which is continuous medially and superiorly with the interscalene space and inferiorly and laterally with the axillary perivascular space.

The important concept is that there is a continuous, fascial enclosed perineural and perivascular space extending from the cervical transverse processes to several centimeters beyond the axilla. This space has been divided into an axillary perivascualr space and an interscalene space. The existence of such a continuous perineural space renders brachial

plexus block simple. The space described may be entered at any level and the volume of the anesthetic injected at that level would determine the extent of anesthesia. Thus the technique to be used in any case should be determined on the basis of the surgical site, the required level of anesthesia, and the physical status and habitus of the patient.

The upper medial aspect of the arm is not anaesthetized by the brachial plexus block technique since this area in innervated by the intercostobrachial nerve (T2). This nerve can be blocked by subcutaneous infiltration across the axillary artery using 3-5ml of local anaesthetic, which provides anesthesia for surgery or tourniquet.

The brachial plexus can be blocked at the level of the roots, trunks, cords, or peripheral branches. The block at each level has a distinct distribution of anesthesia, advantages, disadvantages and complications.

CLINICAL PHARMACOLOGY

A brief review of the clinical pharmacology of drugs used in this study is dealt with here.

LOCAL ANAESTHETICS

Local Anaesthetics are chemical compounds, which are capable of reversibly inhibiting the propagation of impulses in nerve cells.

The local anaethetics have a similar basic structure, consisting of an aromatic head, an intermediate chain and an amine tail. They are classified into

1. Amino esters : they have an ester link which connects aromatic portion and the amine.
2. Amino amides : they have an amide link which connects the aromatic portion and the amine.

BUPIVACAINE

Synthesized by Bo Af Evenstam in 1957 of AB Bofor in Sweden.

First came into clinical use in 1963.

Bupivacaine is an anilide compound. Chemical name is 1 n butyl DL

Piperidine carboxylic acid 2.6 dimethly anilide chloride.

Presentation

As a clear solution containing 0.25/0.5/0.75% Bupivacaine hydrochloride-the 0.25/0.5%

solutions are combined with 1:200000 adrenaline. The heavy solution contains 80mg/ml of glucose (with a specific gravity of 1.026) called bupivacine (heavy)

Physicochemical Properties

pKa = 8.1

Protein binding : 96%

Lipid solubility : 28

Pharmacokinetics

Absorption: The absorption of local anesthetics is related to

1. The site of injection (intercostals > epidural > brachial plexus > subcutaneous)
2. The dose – a linear relationship exists between the total doses and the peak blood concentrations achieved
3. The presence of vasoconstrictors which delay absorption

The addition of adrenaline to bupivacaine does not influence the rate of systemic absorption as,

a) The drug is highly lipid soluble and therefore uptake into fat is rapid

b) The drug has a direct vasoconstrictory effect

Routes of administration / doses

Bupivacaine may be administered topically in infiltration, intrathecally or epidurally. The therapeutic dose of bupivacaine is 2mg/kg (with or without adrenaline).

The drug acts within 10 to 20 minutes and has a duration of action of 5 to 16 hours.

Pharmacodynamics:

The possible pathways for metabolism of bupivacaine include aromatic hydroxylation, N-dealkylation, amide hydrolysis and conjugation. Only the N-dealkylated metabolite N-desbutyl bupivacaine has been measured in the blood or urine.

Alpha 1 acid glycoprotein is the most important protein-binding site of bypivacaine. 5% of the does is excreted in the urine as pipcolyloxylidine 16% is excreted unchanged. Clearance is 0.47 L/min and the elimination half-life is about 210 minutes.

Side Effects:

Systemic toxicity:

Cardiovascular system

Bupivacaine is markedly cardiotoxic. It binds specifically to myocardial proteins. In toxic concentrations the drug decreases the peripheral vascular resistance and myocardial contractility producing hypotension and possible cardiovascular collapse. Cardio toxic plasma concentrations of Bupivacaine are 8 to 10 μg/ml.

Central Nervous System

The principal effect of bupivacaine is reversible neural blockade. This leads to a characteristically biphasic effect in the central nervous system. During accidental overdosage or direct vascular injections the clinical signs are numbness of tongue, lightheadedness, visual and auditory disturbances, muscular twitching and tremors. The signs may progress to generalized convulsions of the tonic-clonic nature. When plasma levels continue to rise, CNS excitation is rapidly superseded by depression. (Drowsiness, disorientation and coma)

The typical plasma concentration of bupivacaine associated with seizures is 4.5 to 5.5

microgram/ml.

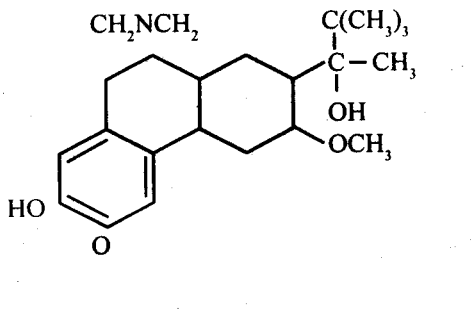
BUPRENORPHINE

Buprenorphine is a synthetic derivative of the alkaloid thebaine. Its main action is analgesia. It is used in the treatment of moderate to severe pain. It can be given through intramuscular in the dose of 0.3mg, intravenously 0.3mg, sublingually 0.2, 0.4mg and epidurally 0.3mg.

Mode of action:

The drug acts as partial agonist at mu-receipts but dissociates slowly from the latter leading to prolonged analgesia. Buprenorphine appears also to have a high affinity for kappa opioid receptors

Chemical structure



Special features

- This drug is many times potent than morphine

  Half life –166 minutes

Slow onset of action, peak flow occurs at 3 hrs and duration of action is more than 10hrs, elimination half life is 5 hours

Volume distribution (VD) is 2.8 L/kg

Clearance – 20ml/kg/min

- **"CEILING EFFECT"**

The increasing dose of buprenorphine above 0.15 to 1.2mg do not produce additional analgesic effect in adults.

- Reversal with naloxone is limited because of high affinity to mu receptors and slow dissociation from the receptor sites.

- Buprenorphine is not removed by hemodialysis

- **Effects**

CVS: Following administration of this drug the heart rate may decrease up to 25% and systolic blood pressure may fall by 10%

RS: The drug produces respiratory depression and an antitussive effect, similar to produced by the morphine.

CNS: It produces miosis

AS: It delays the gastric emptying time and has an emetic effect

GU: This drug has been shown to reduce the rate of urine output in animals

Metabolic effects: It decreases the luteinising hormone and increases the release of proteins

Toxicity and side effects: It is same as morphine. Drowsiness, dizziness, headache, dysphoria, nausea and vomiting may be produced. It appears to be less liable to produce dependence than pure mu-agonists

Kinetics

Absorption: The drug is absorbed orally; but undergoes a significant first pass metabolism and the sub lingual route is therefore preferred. The bio availability is 40-90% when administrated intramuscularly and 44 to 94% when administered sublingually.

Distribution: Only unchanged buprenorphine appears to reach the CNS. It is 96% protein bound

Metabolism: Occurs in the liver by dealkylation in the subsequent conjugation to glucoronide – the polar conjugates then appears to be excreted in the bile and hydrolyzed by bacteria in the GI tract

Excretion: Occurs predominantly via the faeces as unchanged buprinorphine the reminder is excreted in the urine as conjugated buprinorphine and dealkylated derivatives.

PHARMACOKINETICS OF LOCAL ANAESTHETICS IN BRACHIAL PLEXUS BLOCKADE

When a local anaesthetic is injected around a nerve trunk, it will soak the trunk in an advancing front. Transmission in fibres situated in the periphery of the trunk (mantle fibres) will be first blocked and those in the centre of the trunk(core fibres) last. Further, transmission in peripherally placed fibres will be blocked over a longer length of time compared to central fibres. Thus analgesia will appear first and last longest in the territory supplied by the peripheral fibres. If the pool of local anesthetics is small or if the injection was not accurate or too dilute, the fibres in the center of the trunk will escape blockade.

Theory of Winnie

The trunks are arranged so that the central fibres are the longest supplying the extremities of the limb while shorter fibres are arranged more peripherally as their area of supply is more proximal. Winnie groups the fibres into two the peripheral mantle fibres which contain motor fibres and core fibres which are mainly inner sensory peripheral motor fibres supply the muscles of the forearm and the central fibres carry sensation from the hand. Thus the onset of block in the limb is as follows.

Loss of motor power to the shoulder and upper arm

Loss of sensation on the upper arm

Loss of motor power of the forearm

Loss of sensation to the hand.

So the spread of block is from proximal to distal.

REVIEW OF LITERATURE

- Halsted (1885-1922) of New York in 1884, Matas of New orleans (1860-1957) and Crile (1864-1943) of Cleveland in 1897, injected the plexus under direct vision following exposure under local infiltration (Intra-neural block).

- In 1940 Patrick described the “Classical approach”

- In 1970 Dr.AlonWinne described interscalene approach

- In 1922, Labat used multiple needles technique to block the brachial plexus using the Chassaignac’s tubercle as the land mark.

- In 1911, Hirshel described the first percutaneous technique for blocking the brachial plexus by axillary approach and reported on its successful use in these patients.

- In 1926, Livingston described a technique that is fore-runner of subclavian perivascular technique.

- Kulenkampff, assistant to Heinrich Braun, after experimenting on himself used the supraclavicular technique in 1912.

- In 1949, Bonica and Moore advocated the “1st rib Walkover” technique.

- In 1989 Viel E.J. et al used Buprenorphine in brachial plexus block for post-operative pain relief.

- 1964, Winnie described the subclavian perivascular technique for accurate localization

of the plexus.

- In 1980 field H.L. and Emson P.C, identified multiple opiate receptor sites on primary afferent fibres.

- In 1995, Wajima Z et as used Butorphanol in brachial plexus infusion for post-operative analgesia.

- In 1987, Majs K.S. et al produced local analgesia without anaesthesia using peripheral perineural morphine injection.

- Candido et al in their study noted the brachial plexus block with addition of buprenorphine to local anasthetic solution prolonged the duration of analgesia

MATERIAL AND METHODOLOGY

Forty adult patients of both sexes in the age group of 18 to 58 belonging to ASA I/II attending the Plastic&hand reconstructive surgery department at Stanley Medical College Hospital, Chennai-1 formed the material for the study. They were randomly divided into 2 groups I and II

Group I

20 patients received 30 ml of 0.3% bupivacaine plus 1ml of isotonic sodium chloride solution making the solution 0.3%.

Group II

20 patients received 18ml of 0.5% bupivacaine plus 1ml of 150 microgram of buprenorphine.

Patient selection

- Only ASA I & ASA II were included
- Age group between 18 to 58 years
- Weight between 40 to 70kg
- Both emergency and elective procedure involving the upper limb were included
- Both in patients and outpatients were included.

Exclusion criteria

- Patient refusal
- Clinically significant coagulopathy bacterial, fungal infection the injection site
- Pneumothorax,
- Known epileptic

Equipment

Sterile tray

Towel,

2 Nos. of Sterile cups

2 Nos. of 20ml glass syringes.

Sterile gloves

Swabs

Sponge holding forceps

Betadine solution 50 to 100ml

Appropriate needles

1 no. 23G 3.75 cm long needle

22G 4cm long short beveled blunt needle

Drugs used

0.5% sensorcaine 1 vial distilled water 2 vials

Bupergesic 1 amp (Buprenorphine 0.3mg)

Intra post operative-monitors

Pulse Oximeter - continuous

NIBP - 5 minutes interval

ECG - Lead II continuous

Initially the base line parameters were recorded before administering the block.

(i.e.) pulse

BP

S_PO_2 and

ECG

Then the block was administered. All these parameters were monitored intra and post operatively. They were monitored for 12hrs. Patients were carefully observed for the development of complication and side effects and treated if occurred.

Procedure

The block was given in the OT (or) the preparation room where all the facilities to resuscitate available. All patients were enquired for any drug reaction

- Intravenous line was started for all the patients with 18G (green) intravenous catheters and monitor were connected to the patient
- The patient has positioned on the table and proper illumination was done at the site of block
- For continuous neurological evaluation, no sedative drugs were administered pre-operatively.

LOW INTERSCALENE BLOCK

The interscalene approach to block brachial plexus was first described by Dr. Alon winne in 1970. In this study, the interscalene groove was identified at the lower level near the clavicle where the ant.scalene and medial scalene muscles converge.

POSITION

- The patient was placed in supine position with head turned to the contralateral side
- The arm of patient were adducted with the hands pointing towards the knee

- A rolled towel is placed lengthwise between the shoulders along the spine to give the best exposure of the area.

LANDMARKS

- The anesthesiologist stood at the head end of the patient

- The patient is asked to lift the head slightly to bring the clavicular head of the sternocleidomastoid muscle into prominence.

- The finger is placed lateral to the muscle and the patient is told to relax. Roll the index finger laterally across the belly of the sternocleido muscle until the interscalene groove in palpated. To appreciate the groove classically the patient was asked to inspire.

- The finger is then moved inferiorly down the groove until the pulse of the subclavian artery is palpated between the scalene muscles.

The following were the certain anatomical marks / relations to find out the interscalene groove.

- The interscalene groove is approximately 1.5 to 2.5cm above the midpoint of the clavicle.

- The external jugular vein is just above (or) near the groove.

- Subclavian artery is just medical to the groove.

- After finding out the interscalene groove the skin is cleaned with betadine and white spirit a skin wheel is raised at this point with 2ml of lignocaine with 23G needle about 2.3cm above the midpoint of the clavicle.

PROCEDURE

After sterile preparation of the region the 22G, 4cm needle was inserted through the skin wheal and above the palpating finger immediately lateral to the subclavian artery.

- It was directed 45˚ dorsolaterally parallel to the scalene muscles and towards the elbow of the patient. There was a click once the sheath is pierced and entered

- The patient felt paraesthesia of the hand and fingers once the tip of the needle crossed the perineural sheath.

In this technique paresthesia was obtained before the first rib was contacted. If paraesthesia was not elicited then the needle was withdrawn and redirected once again.

Eliciting paraesthesia especially “Distal paresthesia” was sought as a confirmatory index of being close to the nerves. Once achieved the percentage of successful blockade was found to be higher almost 99%.

Then the needle was carefully held at the same position and the drugs were injected. Potential pit falls were patient movement and failure to hold the needle at the point of

eliciting paresthesia.

The following precautions were taken during injection of anaesthetic drugs

(i) Before injection, aspiration was done to see any blood (inadvertent intravascular placement of needle) in the syringe,

(ii) If there was any shooting pain then the needle was slightly withdrawn (as the needle tip entered neural tissue)

(iii) Repeated aspirations were done after every 3-5ml of drugs injected. After injection the block was tested for both sensory (using pin prick) and motor using muscle contraction and compared with same stimulation or power in the contralateral arm.

Motor block was evaluated by thumb abduction (Radial nerve), thumb adduction (ulnarnerve), thumb opposition (Median nerve) and flexion of the elbow in supination and pronation of the forearm (musculocutaneous nerve).

The Hollmen's scale was used to assess both sensory any motor blockade.

THE HOLLMEN 'S SCALE

SENSORY BLOCKADE

1. O Normal sensation of pin prick
2. + Pin prick felt as sharp pointed but weaker compared with the same are in the other upper extremity
3. ++ Pin prick recognized as touch with a blunt object

4. +++ No perception of pin prick

MOTOR BLOCKADE

1. O Normal muscle function
2. + Slight depression in muscle function as compared with the preanaesthetic power.
3. ++ Very weak muscular action persisting in muscle
4. +++ Complete block with absent muscular action

The nerves studied in the block were

Sensory

1. Lateral cutaneous nerve of arm
2. Medial cutaneous nerve of arm
3. Medial cutaneous nerve of forearm
4. Posterior cutaneous nerve of forearm
5. Lateral cutaneous nerve of forearm
6. Median nerve
7. Ulnar nerve
8. Radial nerve

Motor

Median (N)

Ulnar (N)

Radial (N)

Musculocutaneous nerve

Evaluation was carried out for every minute after completion of the injection and the time of onset was noted for both sensory and motor blockade.

Onset of blockade both sensory and motor was defined as a minimum of grade 2 in Hollmen's scale.

Blockade was considered as complete when sensory and motor scores were atleast grade 3 in Hollmen's scale.

Once the blockade was complete the patient was wheeled into the theatre and surgery was allowed to proceed.

Duration of the sensory blockade was considered as the time interval between the local anaesthetic administration and the to onset of pain in the anasthetic arm during the post operative period.

The duration of the motor blockade was considered as the time interval between the local anaesthetic administration and the recovery of the muscle power.

Separate never blocks or supplementation with IV sedation and analgesia were carried out for five cases of which the blockade was inadequate / not taken up.

MONITORING

During this regional anaesthetic procedure, the systemic toxicity due to delayed absorption of local anaesthetic drugs and the narcotic buprenorphine was monitored for about an hour after surgery. Ventilation, oxygenation and the consequences of surgery such as tourniquet pain were also monitored.

- The vital signs PR/S_PO_2 were monitored continuously and RR / BP intermittently.
- The every 5 to 10 minutes till the procedure was over
- Data were expressed as mean ± sd.
- 'P' value < 0.05 was considered significant.

OBSERVATIONS

This study was conducted in government Stanley Hospital in the department of plastic surgery and hand reconstrive surgery. Forty patients scheduled to undergo elective / emergency upper arm surgery were included in the study and were randomly divided into two groups of twenty each

Group I : Twenty patients received 30ml of 0.3% bupivacaine and 1ml of isotonic sodium chloride solution.

Group II : Twenty patients received 30ml of 0.3% bupivacaine and 1ml of 0.15mg of buprenorphine

PHYSICAL CHARACTERISTICS

Both the group of patients was studied statistically with respect to age, sex and weight. The groups were also compared with respect to onset of blockade, time taken for total blockade, total duration of blockade and the intensity of blockade.

DEMOGRAPHIC PROFILE

The distribution of age in the two groups are shown below

TABLE-1

AGE DISTRIBUTION

Age distribution	Group I	Group II
18-28	11	13
28-38	45	4
38-48	34	2
48-58	0	1

P value 0.5086 (not significant)

The distribution of weight in the two groups are shown below

TABLE-2

WEIGHT DISTRIBUTION

Weight in Kgs	Group I	Group II
30-40	1	0
40-50	7	6
50-60	6	10
60-70	6	4

P value 0.3019 (not significant)

The distribution of sex in the two groups are shown below

TABLE-3

SEX DISTRIBUTION

Sex	Group I	Group II
Male	16	17
Female	4	3

ONSET TIME FOR BLOCKADE (MINUTES)

S.No	Group I		Group II	
	Sensory	**Motor**	**Sensory**	**Motor**
1.	8	5	2	1
2.	10	6	4	3
3.	7	3	3	2
4.	7	5	5	4
5.	7	5	8	7
6.	6	4	5	3
7.	7	5	5	3
8.	5	3	4	2
9.	6	4	5	3
10.	5	3	4	2
11.	6	4	4	3
12.	7	5	6	3
13.	5	3	4	2
14.	6	4	3	2
15.	6	3	4	2
16.	7	4	4	2
17.	8	5	4	3
18.	7	3	4	3
19.	6	4	3	2
20.	7	3	4	3
Mean	6.65	4.05	4.25	2.75

P value:

Sensory : <0.0001 (Significant)

Motor : 0.0005 (Significant)

TIME TAKEN FOR TOTAL BLOCKADE (MINUTES)

S.No	Group I		Group II	
	Sensory	Motor	Sensory	Motor
1.	28	24	20	17
2.	20	17	22	18
3.	19	17	26	22
4.	19	15	25	23
5.	25	22	42	35
6.	21	19	27	23
7.	23	22	22	18
8.	19	15	25	22
9.	22	16	22	19
10.	20	15	17	16
11.	21	16	20	17
12.	20	18	19	16
13.	18	15	19	17
14.	19	17	17	16
15.	21	18	16	15
16.	21	17	16	14
17.	19	16	22	20
18.	18	15	28	20
19.	19	16	21	18
20.	18	15	22	18
Mean	20.5	17.25	22.4	19.2

P value:

Sensory : 0.187 (Not Significant)

Motor : 0.1047 (Not significant)

TOTAL DURATION OF BLOCKADE (MINUTES)

S.No	Group I		Group II	
	Sensory	Motor	Sensory	Motor
1.	270	225	602	572
2.	370	325	825	735
3.	356	316	679	634
4.	310	275	720	685
5.	380	320	589	559
6.	345	300	520	475
7.	420	340	740	680
8.	319	304	680	620
9.	349	329	745	680
10.	310	290	564	504
11.	320	290	745	720
12.	340	320	695	675
13.	310	290	687	660
14.	320	294	673	627
15.	345	335	790	733
16.	310	290	653	560
17.	320	300	748	688
18.	330	310	800	770
19.	290	275	562	502
20.	310	290	595	565
Mean	331.2	300.9	680.6	632.2

P value:

Sensory : <0.0001 (Significant)

Motor : <0.0001 (Significant)

INTENSITY OF BLOCKADE

The intensity of blockade was graded using the Hollmen's Scale. The sensory and motor blockade of both the groups were compared.

	Grading	Group I	Group II
Sensory	1	0	0
	2	13	1
	3	7	5
	4	0	14
Motor	1	0	0
	2	7	4
	3	13	9
	4	0	7

P value:

Sensory : <0.0001 (Significant)

Motor : 0.0165 (Significant)

DISCUSSION

In this prospective, randomized study, 40 patients satisfying the selection criteria underwent brachial plexus block with or without addition of buprenorphine, onset, completion, duration and intensity of blockade were compared and statistically analyzed. Results show that addition of 150 micrograms of buprenorphine to local anaesthetic solution 0.3% bupivacine causes significant increase in duration and intensity of the sensory and motor blockade, shortens the onset of sensory and motor blockade and there was no difference in the time taken for total blockade.

In this discussion we attempt a comparison of findings drugs in our study to these already quoted in the literature.

Viel and colleagues postulated three possible mechanism of action of prolonged analgesia produced by peripheral administration of opioids.

1. The dorsal nerve root tissues contain μ (mu) receptor finding sites. The axonal flow (or) movement of opioid receptor and various macromolecule in the neurons had been demonstrated by young and colleagues and bi-directional axonal flow of these proteins was demonstrated by Laduron. Young and colleagues demonstrated that these moving receptors circulated endorphins and their ligands in addition to

exogenous opioids. The existence of such receptors is the base for the hypothesis that opioids act directly on the peripheral nervous system.

2. These opioids may diffuse from brachial plexus sheath to extramural and subarachnoid spaces and then bind with opioid receptors in the dorsal horn. Dauggard, dahl and Christensen measured morphine concentration in spinal fluid after perineural injection of morphine around femoral nerve and found that the concentration was inadequate to produce analgesia by acting directly on the spinal cord.

3. The evidence of axonal flow or various macromolecules suggests possible centripetal axonal transport of opioid into the substantial gelatinous after perinural injection.

Low doses of peripherally administrated opioids can produce anti-nociceptive effects mediated by peripheral opioid receptor in the inflamed tissue of rat. Stein and colleagues postulate that activation of these neuronal receptors causes attenuation of the excitability of the nociceptive input terminals or inhibition of release of excitatory transmitters or both.

ONSET OF BLOCK

Ortells polo et al., in their study of modified supraclasiscular perivasular technique

recorded an onset time of 4.9 ± 0.2 minutes and the time for motor paralysis at 15.2 ± 6.9 minutes.

Vonguises et al, in their study recorded onset time of 6.95 ± 3.07 minutes

Odoom et al recorded the onset time for motor weakness between 5 to 10 minutes.

All the above studies support our study, in which the onset time for both motor and sensory was around 3-5 minutes. There was statistical significant in the onset of sensory and motor blockade.

The mean time of onset of block

Group I : 4.05 ± 0.94451 minutes for Motor and 6.65 minutes for sensory

Group II : 2.75 ± 1.20852 minutes for Motor and 4.25 minutes for sensory

There was statistically significant difference in the time taken for onset of block, between the two groups.

TIME TAKEN FOR TOTAL BLOCKADE

The mean time for total blockade,

17.05 ± 2.63329 minutes for motor and 20.5 minutes for sensory in Group I

19.2 ± 4.53756 minutes for motor and 22.4 minutes for sensory in Group II

There was no statistically significant difference in the time taken for total blockade between the two groups.

DURATION OF BLOCK

Vong vises et al in their study of parascline approach to brachial plexus block recorded average duration of anaesthesia of five hours.

Jean Marc Bernard et al recorded mean duration of action of 260 ± 27 minutes

- Dalens et al recorded motor blockade lasting around 6 hours
- Carlo D. Franco et al in their study of subclasion perivascular practical plexus blocks recorded duration of action lasting upto 6 hours.
- Kapral et al in their study recorded sensory blockade of 299 ± 84 minutes and motor blockade of 259 ± 76 minutes.

Candido et al in their study recorded thirty hours of post operative analgesia in brachial plexus block by adding buprenorphine.

In our study the mean total duration of sensory blockade in 331.2 ± 33.5443 minutes in Group I and 680.6 ± 86.2722 minutes in Group II.

The mean total duration of motor blockade in 300.9 ± 26.0139639 minutes in Group I

and 632.2 ± 85.0669087 minutes in Group II

The duration of sensory and motor blockade was prolonged in Group II with a statistically significant P value <0.0001.

NTENSITY OF BLOCK

In our study the intensity of block significantly increased in sensory and motor blockade.

Intensity of sensory block

In group I : 7 patients had sensory blockade of grade 3 and 13 patients had grade 2 blockade

13 patients has motor blockade of grade 3 and 7 patients has blockade of grade 2.

In Group II : 14 patients has sensory blockade of grade 4, 5 patients has grading 3 and are patient of grade 2.

7 patients has motor blockade of grade 4, 9 patients had grading 3 and 4 patients of grade 2.

The P value was <0.0165 for motor and <0.0001 for sensory blockade which is statistically significant.

Complications and side effects

There was two incidences of arterial puncture without formation of hematoma. Needle was again repositioned and drug administrated – Blocks were successful.

- There was no other incidence of pnumothorax, neurological deficit, phrenie nerve palsy or horner's syndrome.
- There was incidences of nausea and vomiting in Group II patients.
- There was inadequate blockade in 4 patients in Group I and "ulnar sparing" in Group II. The Group I patients were converted to general anaesthsia and the Group II patient was given separate ulnar nerve block.

Heart rate, Bp, O_2 saturation and respiration were monitored and were stable. No differences were noted among the groups.

SUMMARY

1. Onset time of sensory and motor blockade is shorter in Group II than control Group I

2. There was no difference in the time for total sensory and motor blockade in both the groups

3. These was significant increase in the duration of blockade in the Group II than Group I

4. There was significant increase in the intensity of sensory and motor blockade in the Group II than Group I.

5. There was no complication due to addition of 150 micrograms of buprenorphine to 0.3% bupivacaine

CONCLUSION

We conclude that in brachial plexus block by low interscalne approach, the addition of 150μg of buprenorphine with 0.3% bupivacaine provides intense, prolonged sensory and motor blockade without complication when it is compared to the block produced by plain 0.3% of bupivacaine solution alone.

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PROFORMA

Name : Age/Sex: Wt: IP.No: Surgery:

ASA Status : Premed :

Group I/II Conc.& Vol of Solution : Technique :

Onset time Complete Blockade

Sensory :

Blockade :

Motor :

		0	5	10	15	20	30	60	120	180	240	300	360	420
Vitals :	PR BP RR SPO2													
Sensory & Motor Score														
Radial N	S													
	M													
Median N	S													
	M													
Ulnar N	S													
	M													
Musculocut N	S													
	M													
Sensory Score														
Lateral Cut N of arm														
Medial Cut N of arm														
Medial Cut N of Forearm														
Lateral Cut N of forearm														

Duration of Blockade : Sensory Tourniquet position

Motor Tourniquet pain

Grading of Block

Complication:

Paraesthesia / Neurological deficient	Nausea / Vomiting
LA Toxicity	Vascular Puncture
Pneumothorax	

MASTER CHART

CONTROL -0.3% BUPIVACAINE Alone

	NAME	IP NO	SEX	AGE	WEIGHT	ONSET M	ONSET S	COM M	COM S	DUR. M	DUR. S	INT.M	INT.S	COMMENTS
1	Madankumar	245039	M	18	35	5	8	24	28	225	270	2	2	
2	Devanathan	246673	M	60		6	10	17	20	325	370	3	3	
3	Suresh	247796	M	30	59	3	7	17	19	316	356	3	2	
4	Krishnan	247941	M	41	55	5	7	15	19	275	310	2	2	
5	Bappiz	246702	M	25	48	5	7	22	25	320	380	3	2	KET.30mg IV
6	Jeevanandam	248009	M	34	62	4	6	19	21	300	345	3	3	
7	Vairamani	227840	M	34	51	5	7	22	23	340	420	3	2	
8	Prakash	248523	M	24	46	3	5	15	19	304	319	3	3	
9	Jothi	248530	F	23	45	4	6	16	22	329	349	3	2	
10	Anbaesivan	248731	M	42	61	3	5	15	20	290	310	2	2	
11	Loganathan	249045	M	28	52	4	6	16	21	290	320	3	2	
12	Perumal	248082	M	34	64	5	7	18	20	320	340	3	3	
13	Ramesh	244057	M	18	45	3	5	15	18	290	310	2	2	
14	Subhashini	249152	F	18	48	4	6	17	19	294	320	2	2	CAMP2mg
														KET.30mg IV
15	Muniammal	249057	F	25	54	3	6	18	21	335	345	2	3	CAMP5mg
														KET.30mg IV
16	Ramu	249091	M	19	41	4	7	17	21	290	310	3	2	
17	Anilkumar	249095	M	48	64	5	8	16	19	300	320	3	3	
18	Jeyanthi	249019	F	25	48	3	7	15	18	310	330	2	2	CAMP5mg
														KET.30mg IV
19	Subramani	249150	M	40	64	4	6	16	19	275	290	3	3	
20	Manikandan	249168	M	24	53	3	7	15	18	290	310	3	2	
			MEAN	30.5	52.368	4.05	6.65	17.25	20.5	300.9	331.2	2.65	2.35	
			SD	11.20385	8.3879	0.94451	1.1821	2.6333	2.50263	26.013964	33.5443	0.489	0.489	