A COMPARATIVE STUDY OF ANAESTHETIC EFFICACY OF 0.2% ROPIVACAINE ALONE AND 0.2% ROPIVACAINE WITH FENTANYL 50 MICS IN INTRAVENOUS REGIONAL ANAESTHESIA

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CERTIFICATE

This is to certify that the dissertation entitled **"**A COMPARATIVE STUDY OF ANAESTHETIC EFFICACY OF 0.2% ROPIVACAINE ALONE AND 0.2% ROPIVACAINE WITH FENTANYL 50 MICS IN INTRAVENOUS REGIONAL ANAESTHESIA" is the bonafide original work of **Dr.T.SUBASH KUMAR** in partial fulfillment of the requirements for M.D. Branch-X (anaesthesiology) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in April 2012.

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I **Dr. T. SUBASH KUMAR**, solemnly declare that dissertation titled, **"**A COMPARATIVE STUDY OF ANAESTHETIC EFFICACY OF 0.2% ROPIVACAINE ALONE AND 0.2% ROPIVACAINE WITH FENTANYL 50 MICS IN INTRAVENOUS REGIONAL ANAESTHESIA**"** is a bonafide work done by me at K.A.P.V. Government Medical College, during 2009-2012 under the guidance and supervision of my Professor & Head of the department of Anaesthesiology **Prof. Dr. N. JOTHI, M.D.,D.A** .

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – X) in Anaesthesiology.

Place: Trichy

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CONTENTS

INTRODUCTION

Intravenous regional anaesthesia is a method of producing analgesia and muscle relaxation of the distal part of a limb by intravenous local anaesthetic injection while circulation to the limb is occluded.

Intravenous regional anaesthesia was discovered by August Bier, professor of surgery in berlin in 1908. To administer intravenous anaesthesia he occluded the circulation in a segment of the arm with two tourniquets and then injected a solution of 0.5% procaine into a vein in the isolated segment. This technique was revived by Holmes in 1963, who used lignocaine because it appeared to give more reliable anaesthesia than procaine. Currently, intravenous regional anaesthesia sometimes referred to as Bier's block is regarded as one of the several alternative techniques for arm anaesthesia.

Various drugs such as procaine, prilocaine, lignocaine, bupivacaine has been used in intravenous regional anaesthesia. Among these, lignocaine is the drug commonly used. As lignocaine does not have post tourniquet deflation analgesia other local anaesthetic dugs have been tried to improve the duration and post operative analgesia.

A longer acting agent bupivacaine initially gained substantial popularity but was laden with potentially serious side effects. Bupivacaine has been identified as a fast-in, slow-out type of local anaesthetic that maintains high affinity and binds tightly to myocardial sodium receptors. Therefore, if high

plasma concentrations of bupivacaine are achieved irreversible cardiac arrest may occur.

The amide local anaesthetic ropivacaine is a pure -s- enantiomer and is structurally related to bupivacaine. The duration of effect of ropivacaine is almost similar to that of bupivacaine . Ropivacaine has been shown to result in less depression of the cardiac conduction system when compared with bupivacaine.

Intravenous ropivacaine, compared with bupivacaine and lignocaine in several studies has less cardiac and central nervous system side effects but has achieved better surgical anaesthetic conditions.

In an attempt to reduce the amount of local anaesthetic required or to improve the quality of the block or both, various additives such as opiods, muscle relaxants, alpha2 agonists have been tried in intravenous regional anaesthesia with various results. Among these fentanyl a synthetic opiod is used in this study along with ropivacaine.

AIM OF THE STUDY

The aim of the study is to evaluate,

1) the anaesthetic effects of 0.2% ropivacaine
2) to compare it with the addition of fentanyl 50 mics in intravenous regional anaesthesia.

The anaesthetic efficacy regarding,

1) onset of sensory block
2) onset of motor block
3) occurrence of tourniquet pain
4) duration of post op analgesia
5) side effects, if any

are noted and compared.

ANATOMY OF SUPERFICIAL VENOUS SYSTEM OF UPPER LIMB

The venous supply of the upper limb is arranged in two planes, which are separated by superficial aponeurosis. The superficial venous system is used for intravenous regional anaesthesia. The local anaesthetic is injected into this venous system and it is transported through a venous network into veins inside nerve trunks.

The dorsal digital veins converge to form three dorsal metacarpal veins that are interlinked to each other.The palmar digital veins drain into a dense vascular network on the palmar face of the hand and also into the dorsal veins by way of intercapillary veins. Most of the superficial veins join to form two large veins, the cephalic vein and the basilic vein. An accessory cephalic vein is often present. Hence by this venous network, the local anaesthetic is transported into veins inside nerve trunks.

The cephalic vein is the preaxial vein of the upper limb. It begins from the lateral end of the dorsal venous arch. The cephalic vein runs upward the root of anatomic snuff box, and winds around the lateral border of the dorsal forearm. In the proximal part of the forearm the cephalic vein runs medially to run along the anterior aspect of the forearm.

At the level of elbow, it runs between the tendons of brachioradialis and the

biceps brachii,crosses the lateral cutaneous nerve of forearm and then runs along the lateral border of biceps.It then pierces the deep fascia at the lower border of pectoralis major and then runs in the deltopectoral groove upto the infraclavicular fossa where it pierces the clavipectoral fascia and joins the axillary vein.

The accessory cephalic vein, when present arises from a venous network on the back of the forearm or from the lower part of cephalic vein. It joins the cephalic vein below or in front of the elbow. When the main cephalic vein drains into the basilic vein, the accessory cephalic may follow the course of the former above the elbow.

The basilic vein is the postaxial vein of the upper limb and arises from the medial end of the dorsal venous arch a little below the limb. It runs upward along the back of the medial border of the forearm, winds around this border to reach the ventral aspect of cubital fossa and then runs along the medial aspect of biceps brachii upto the middle of the arm, pierces the deep fascia and runs along the medial side of the brachial artery,upto the lower border of the teres major where it continues as axillary vein. About 2.5cm above the medial condyle of the humerus, the basilic vein is joined by the medial cubital vein. It is accompanied by the posterior branch of medial cutaneous nerve of the forearm and the terminal part of the dorsal branch of ulnar nerve. The superficial palmar venous network is drained by the median vein of forearm, which runs along the anterior aspect of the forearm, in front of the elbow, it

drains directly into the basilic vein.

Hence the nerve endings in the skin are easily reached by the local anaesthetic solution through these valveless venules. The superficial veins are essentially that collapse when theirlumen are not filled with blood. The thick outermost layer of a vein is made of connective tissue called tunica adventitia or tunica externa. Deeper are bands of smooth muscle called tunica media, which are in general is thin, as it does not function in a contractile manner. The interior is lined with endothelial cells called tunica intima.

The arterial system is the higher pressure portion of the circulatory system. systemic arteries can be divided into two types – muscular and elastic according to the relative composition of elastic and muscle tissue in their tunica media as well as their size and the make up of the internal and external elastic lamina. It is composed of three layers tunica adventitia, tunica media. Hence arteries are not easily collapsed when tourniquet is applied.

ANATOMY OF THE NERVE

Neurons in human central nervous system are in many different size and shapes. These cells have five to seven processes called dendrites that extend outward from the cell body and arborize extensively. A typical neuron also has a long fibrous axon that originates from a thickened area of cell body called axon hillock.The first portion of axon is called initial segment.The axon divides into terminal segments ending in a number of synaptic knobs.

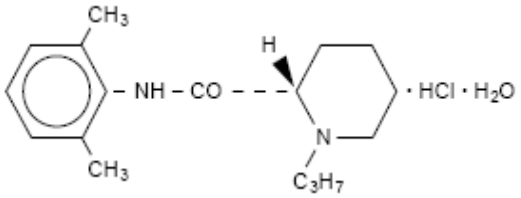
The axons are wrapped by a sheath of myelin ,the protein rich complex produced by Schwann cells. The myelin sheath envelops the axon except at the ending of node of Ranvier , a periodic 1 –micro metre constriction that are about 1 mm apart.

In intravenous regional anaesthesia, the initial effect being the blockade of peripheral small nerves and nerve endings. Block of large nerves at a proximal site (eg.cubital region in intravenous regional nerve block of the arm), as well as via intraneural venous distribution of the local anaesthetic solution also occurs. Then during compression of the tourniquet, ischaemia occurs leading to blockade of nerve conduction .

ROPIVACAINE

Ropivacaine hydrochloride, which is a member of the amino amide class of local anesthetics. It is a sterile, isotonic solution that contains the enantiomerically pure drug substance, sodium chloride for isotonicity and water for injection. Sodium hydroxide or hydrochloric acid may be used for pH adjustment.

Ropivacaine HCl is chemically described as S-(-)-1-propyl-2',6'-pipecoloxylidide hydrochloride monohydrate[8]. The drug substance is a white crystalline powder, with a molecular formula of $C_{17}H_{26}N_2O$•HCl•H_2O, molecular weight of 328.89 and the following structural formula:



At 25°C ropivacaine HCl has a solubility of 53.8 mg/mL in water, a distribution ratio of 14:1between n-octanol and phosphate buffer at pH 7.4 and a pKa of 8.07 in 0.1 M KCl solution. The pKa of ropivacaine is approximately the same as bupivacaine (8.1) and is similar to that of mepivacaine (7.7). However, ropivacaine has an intermediate degree of lipid solubility compared to bupivacaine and mepivacaine.

Ropivacaine Injection is preservative-free and is available in single dose containers in 2mg/ml (0.2%), 5mg/ml (0.5%), 7.5mg/ml (0.75%) and 10 mg/mL

(1%) concentrations. The specific gravity range from 1.002 to 1.005 at 25°C. When prolonged blocks are used, either through continuous infusion or through repeated bolus administration, the risks of reaching a toxic plasma concentration or inducing local neural injury must be considered.

Reactions to ropivacaine are characteristic of those associated with other amide-type local anesthetics. A major cause of adverse reactions to this group of drugs may be associated with excessive plasma levels, which may be due to overdosage, unintentional intravascular injection or slow metabolic degradation.

Systemic Reactions

The most commonly encountered acute adverse experiences that demand immediate countermeasures are related to the central nervous system and the cardiovascular system. These adverse experiences are generally dose-related and due to high plasma levels that may result from overdosage, rapid absorption from the injection site, diminished tolerance or from unintentional intravascular injection of the local anesthetic solution. Factors influencing plasma protein binding such as acidosis, systemic diseases that alter protein production or competition with other drugs for protein binding sites, may diminish individual tolerance.

Neurologic Reactions

These are characterized by excitation and/or depression. Restlessness, anxiety, dizziness, tinnitus, blurred vision or tremors may occur, possibly proceeding to convulsions. However, excitement may be transient or absent, with depression being the first manifestation of an adverse reaction. This may quickly be followed by drowsiness merging into unconsciousness and respiratory arrest. Other central nervous system effects may be nausea, vomiting, chills, and constriction of the pupils.

The incidence of convulsions associated with the use of local anesthetics varies with the route of administration and the total dose administered. In a survey of studies of epidural anaesthesia, overt toxicity progressing to convulsions occurred in approximately 0.1% of localanaesthetic administrations.

The incidence of adverse neurological reactions associated with the use of local anaesthetics may be related to the total dose and concentration of local anaesthetic administered and are also dependent upon the particular drug used, the route of administration, and the physical status of the patient. Many of these observations may be related to local anaesthetic techniques, with or without a contribution from the drug.

Cardiovascular System Reactions

High doses or unintentional intravascular injection may lead to high plasma levels and related depression of the myocardium, decreased cardiac output, heart block, hypotension, bradycardia, ventricular arrhythmias, and possibly cardiac arrest .

Allergic Reactions

Allergic type reactions are rare and may occur as a result of sensitivity to the local anaesthetic.These reactions are characterized by signs such as urticaria, pruritus, erythema, angioneurotic edema (including laryngeal edema), tachycardia, sneezing, nausea, vomiting, dizziness, syncope, excessive sweating, elevated temperature, and possibly anaphylactoid symptomatology (including severe hypotension). Cross-sensitivity among members of the amide-type local anesthetic group has been reported. The usefulness of screening for sensitivity has not been definitively established.

In human volunteers given intravenous ropivacaine the mean (min-max) maximum tolerated total and free arterial plasma concentrations were 4.3 (3.4 to 5.3) and 0.6 (0.3 to 0.9) mcg/mL Clinical data from patients experiencing local anaesthetic-induced convulsions demonstrated rapid development of hypoxia, hypercarbia and acidosis within a minute of the onset of convulsions. These observations suggest that oxygen consumption and carbon dioxide production are greatly increased during local anaesthetic induced convulsions and

emphasize the importance of immediate and effective ventilation with oxygen which may avoid cardiac arrest.

Pharmacokinetics: ***Absorption***

The systemic concentration of ropivacaine is dependent on the total dose and concentration of drug administered, the route of administration, the patient's hemodynamic/circulatory condition, and the vascularity of the administration site . From the epidural space, ropivacaine shows complete and biphasic absorption. The half-lives of the 2 phases, (mean ± SD) are 14 ± 7 minutes and 4.2 ± 0.9 h, respectively. The slow absorption is the rate limiting factor in the elimination of ropivacaine that explains why the terminal half-life is longer after epidural than after intravenous administration. Ropivacaine shows dose-proportionality up to the highest intravenous dose studied, 80 mg,corresponding to a mean ± SD peak plasma concentration of 1.9 ± 0.3 mcg/ml.

In some patients after a 300 mg dose for brachial plexus block, free plasma concentrations of ropivacaine may approach the threshold for CNS toxicity. At a dose of greater than 300 mg, for local infiltration, the terminal half-life may be longer (> 30 hours).

Metabolism

Ropivacaine is extensively metabolized in the liver, predominantly by aromatic hydroxylation mediated by cytochrome P4501A to 3-hydroxy ropivacaine. After a single intravenous dose, approximately 37% of the total

dose is excreted in the urine as both free and conjugated3-hydroxy ropivacaine. The N-de-alkylated metabolite of ropivacaine (PPX) and 3-OH-ropivacaine are the major metabolites excreted in the urine during epidural infusion.

Elimination:

The kidney is the main excretory organ for most local anaesthetic metabolites. In total, 86% of the ropivacaine dose is excreted in the urine after intravenous administration of which only 1% relates to unchanged drug. After intravenous administration, ropivacaine has a mean ± SD total plasma clearance of 387 ± 107 mL/min, an unbound plasma clearance of 7.2 ± 1.6 L/min, and a renal clearance of 1 mL/min. The mean ± SD terminal half-life is 1.8 ± 0.7 h after intravascular administration .

Pharmacodynamics:

Systemic absorption of local anaesthetics can produce effects on the central nervous and cardiovascular systems. At blood concentrations achieved with therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility, and peripheral vascular resistance have been reported. Toxic blood concentrations depress cardiac conduction and excitability, which may lead to atrioventricular block, ventricular arrhythmias and to cardiac arrest, sometimes resulting in fatalities. In addition, myocardial contractility is depressed and peripheral vasodilation occurs, leading to decreased cardiac output and arterial blood pressure.

Following systemic absorption, local anaesthetics can produce central nervous system stimulation, depression or both. Apparent central stimulation is usually manifested as restlessness, tremors and shivering, progressing to convulsions, followed by depression and coma, progressing ultimately to respiratory arrest. However, the local anaesthetics have a primary depressant effect on the medulla and on higher centers. The depressed stage may occur without a prior excited stage.

FENTANYL

Fentanyl is a synthetic phenylpiperidine opiod of the 4-anilopiperidine series which is structurally related to pethidine. Commercially, fentanyl is formulated as a citrate available as an aqueous solution without preservatives. Each ml contains a base of 50 mcg of fentanyl.

PHYSICOCHEMICAL PROFILE

Molecular weight	528.29
Pka	8.4
% unionized at pH 7.4	8.5%
% bound to plasma proteins	84%
Potency	80 times more potent than morphine

PHARMACODYNAMICS

Analgesia

Analgesia results from action of fentanyl on opiod mu receptors both supraspinally in the brain and in the spinal cord. Intravenous fentanyl produces analgesia at plasma concentrations between 0.6 – 3 ng/ml.

Cardiovascular system

Arterial blood pressure, cardiac output and pulmonary vascular resistance remains unchanged even after large doses of intravenous fentanyl. Fentanyl like other opiod agonists causes bradycardia that responds to intravenous atropine. Peripheral vasodilatation is much lesser than morphine due to absence of histamine release.

Respiratory system

Fentanyl causes direct dose related respiratory depression due to its effect on medullary respiratory centre manifested as decreased sensitivity to carbon dioxide and reduced respiratory rate. This is typically seen when plasma concentrations exceed > 2 ng/ml. The degree of respiratory depression is affected by various factors such as type of surgery, age and individual pharmacodynamic response.

Central nervous system

Fentanyl causes less sedation than equianalgesic doses of morphine. In doses of 10 mcg/kg it causes dose related reduction in cerebral blood flow and CMRO2. Muscle rigidity probably reflects a manifestation of a catatonic state, a basic pharmacological property of opiods, related to enhancement of dopamine biosynthesis in the caudate nucleus.

Gastrointestinal tract

Fentanyl decreases gastrointestinal motility, increases intrabiliary pressure and causes a varying degree of nausea and vomiting. The vomiting is mediated via stimulation of the chemoreceptor zone in the area postrema.

Genitourinary system

Like other opiods, fentanyl causes relaxation of detrusor muscle and increase in the urethral sphincter tone leading to urinary retention. This is probably not dose related and is more common with central neuraxial administration.

PHARMACOKINETICS

Fentanyl is a potent opiod, highly lipophilic, producing a rapid onset of action of relatively short duration. After intravenous administration fentanyl is rapidly distributed to brain, heart and other highly perfused tissues. It also crosses the placental barrier easily. Peak effect occurs in 5 minutes. Within a short time the drug redistributes to the inactive tissue sites such as skeletal muscle and fat, associated with decrease in plasma concentration of drug, thus terminating its effect. About 75% of initial dose undergoes firstpass pulmonary uptake. When low doses are administered , redistribution terminates the effect and the drug appears short acting. With administration of large intravenous doses or continuous infusion, progressive saturation of inactive tissue sites occurs.

Pharmacokinetic profile

Volume of distribution at steady state	335 litres
Clearance	1530 ml/min
Effect-site equilibrium time	6.8 min
Hepatic extraction ratio	0.8-0.1
Context sensitive half time (4hrs infusion)	260 mins
Elimination half time	3.1 to 6.6 hrs

Metabolism

Fentanyl is biotransformed in the liver to inactive metabolites, primarily norfentanyl and several hydroxylation products. Only 4-7% of drug is excreted unchanged in the urine. Elimination half time of fentanyl is longer than morphine due to its high lipid solubility. Elimination half time is prolonged in elderly patients. A high hepatic extraction ratio means that the clearance of fentanyl is limited by hepatic blood flow.

INTRAVENOUS REGIONAL ANAESTHESIA

When general anaesthesia is contra indicated, regional would be a better approach. One such regional anaesthetic technique used in upper limb surgeries is intravenous regional anaesthesia. This technique was chosen for this study considering the following merits .

Merits:

1. Simple technique
2. Reliable and effective when properly used.
3. Rapid onset of action.
4. Rapid and prompt recovery from tourniquet release.
5. Good analgesia and adequate muscle relaxation.
6. Provides bloodless operative field.
7. Widely applicable to patients of different ages and physical status.

Contraindications:

1. Patient refusal
2. Absence of resuscitative equipments and drugs.
3. Allergy to local anaesthetics
4. Infection and cellulitis in the limb to be blocked
5. Conditions precluding use of tourniquet like
    a. scleroderma

b. hemolytic diseases such as sickle cell anaemia, thalassemia

c. Raynaud's disease

d. malignancy

6. Lengthy procedure

7. Patients with seizure disorders or with cardiac disorders.

PHARMACOKINETIC ASPECTS OF INTRAVENOUS REGIONAL ANAESTHESIA

The exact mechanism of analgesia and muscle paralysis produced by this technique is not clear. It has been suggested that large venous channels that surround the nerve provide access for the drug to the vascular channels in the core of nerves.

SITE OF ACTION :

The site and mode of action of the drug is still controversial. The three most probable sites of action are

a.The sensory nerve endings

b.The neuromuscular junction

c.The nerve trunks.

According to ADAMS ET AL (1964) the anaesthetic solution a far more effective when placed into the isolated vascular tree rather than in the tissues at

random. By this method, the capillaries perform an important function in the production of anaesthesia by transporting lignocaine very effectively to both large and small nerves.This would explain the rapid decrease in the large nerve conduction speed as well as peripheral anaesthesia which first appears close to the point of injection.Since lignocaine is effectively shut off from the general circulation, it does not reach the liver and is not destroyed thereby providing anaesthesia as long as it is trapped in the arm. On release of tourniquet the remaining solution seems to be very rapidly flushed out of the extremity.The pharmacoaspects of intravenous regional anaesthesia have been described by TUCKER &BOAS (1971). They described that after cuff release, the peak plasma concentration of lignocaine were 20-80 % less than when a same dose of lignocaine is injected by direct intravenous route. The peak level achieved was inversely proportional to total time of the tourniquet applied.

C.J.EVANS ET AL (1974) studied the duration of residual anaesthesia following release of tourniquet after intravenous anaesthesia. They found that the duration of block was related to duration action of the drugs when used clinically.With intravenous regional anaesthesia, the ability of the drug to remain in sufficient quantities to cause blockade of the neural transmission is important with regard to residual anaesthesia. As the drug reaches the nerve tissue by vascular channels and not by penetrating the nerve sheath , the duration of residual anaesthesia was found to be longest with ropivacaine and shortest with prilocaine. Both the safety and efficacy of the procedure depend on the interruption of the blood flow to the involved limb. Prolongation of the

tourniquet inflation time allows more local anaesthetic to be bound to tissues and thus, peak plasma concentration decrease proportionally with duration of the tourniquet time.After a tourniquet time of 10 minutes about 30% of the lignocaine dose is released immediately in the first flush of blood after the deflation of the cuff .After 30 minutes about 45% of the dose still remains in the arm.

MATERIALS AND METHODS

This study was conducted in 50 patients undergoing hand surgeries in KAPV Govt medical college hospital. After getting institutional ethical committee approval and after explaining the procedure in detail , informed consent obtained from every patient. The patients were assigned into two groups each containing 25 patients.

GROUP 1: Patients in this group received 40 ml of 0.2 % ropivacaine

GROUP 2: Patients in this group received 40 ml of 0.2 % ropivacaine with fentanyl 50 mics

SELECTION OF PATIENTS

The patients selected for this study were of ASA I and II, undergoing elective hand surgeries where the expected duration is less than an hour.

EXCLUSION CRITERIA :

Patients with history of any cardiovascular , respiratory or central nervous system disorders were excluded from the study. Patients with haematological disorders like sickle cell anaemia and thalassemia, patients with known hypersensitivity to ropivacaine ,patients with difficult airway , were also excluded from the study.

PREANESTHETIC ASSESSMENT

Physical status of all the patients were pre-operatively assessed. A thorough airway assessment was done. The following investigations were done on all the patients.

1. Hemoglobin
2. Urine analysis
3. Blood sugar
4. Blood urea and serum creatinine
5. Chest x - ray
6. Electrocardigram

RESUSCITATIVE MEASURES

The following equipments and drugs were kept ready to meet any emergency.

1. Boyle's apparatus
2. Laryngoscope with appropriate size blades
3. Endotracheal tubes of various sizes and connectors
4. Suction apparatus
5. Drugs like anticonvulsants, antihistamines, vasopressors, steroids and bronchodilators.

PROCEDURE

The patients were shifted into the operation theatre. The pulseoximeter, non-invasive blood pressure monitor and electrocardiographic monitor were connected to the patient. The vital parameters were recorded. A separate intravenous line was started in the non- operated limb. A vein in the dorsum of the hand of the operated limb was cannulated with 22G intravenous cannula. If the dorsum of the hand was involved in the surgery, a vein higher up in the forearm was chosen . It was firmly fixed , flushed with normal saline and stopper applied.

Exsanguination was accomplished by elevation of the limb for 5 minutes followed by use of esmarch bandage from fingertip to arm. In subjects where application of esmarch bandage was not feasible, emptying of veins was facilitated with compression of axillary artery with the limb elevated[3]. At the proximal end of the esmarch bandage, the first tourniquet was applied around the upper part of the arm over cotton wool padding. Then the tourniquet was inflated to 250 mmHg. Circulatory isolation of the arm was verified by inspection, absence of radial pulse and loss of pulseoximeter tracing of the ipsilateral index finger. Then 40ml of local anaesthesia solution was injected through the cannula at a rate of 1ml/second.

After ensuring complete analgesia below the first tourniquet, the second tourniquet was applied distal to the first tourniquet and inflated to 250mmHg.

The first tourniquet was then removed. The patients were observed for any toxic manifestations of local anaesthetics after release of the first tourniquet. The following parameters were recorded.

Time of onset of sensory block:

It is the time elapsed from injection of study drug to sensory block achieved in all dermatomes of the operated limb. This was checked by pinprick every minute till the onset of sensory block

Time of onset of motor block:

It is the time elapsed from injection of the study drug to the inability of voluntary movements in the operated limb. This was checked by asking the patient to flex the elbow and hand every minute till the onset.

Incidence of tourniquet pain:

Intraoperative tourniquet pain, if perceived was noted and documented.

Time of sensory block recovery (post op analgesia):

It is the time elapsed from tourniquet deflation to recovery of pain (VAS > 5) in all dermatomes of the operated limb.

Side effects:

Side effects after tourniquet release, if any was noted and documented.

ASSESSMENT OF PAIN

Pain is a personal, subjective experience . There are three major psychological dimensions of pain sensory –discriminative, motivational-affective and cognitive-evaluative. These categories of activity interact with one another to provide perceptual information on the location, magnitude and spatiotemporal properties of the noxious stimuli:motivational tendency towards escape or attack ; and cognitive information based on past experience. All three forms of activity could then influence motor mechanisms responsible for the complex pattern of overt responses that characterize pain.

Pain assessment should be ongoing, individualized and documented. The measurement of pain is important : to determine pain intensity, perception qualities and duration, to aid in diagnosis, to help decide on the therapy and to evaluate the relative effectiveness of different therapies.

Pain is subjective; changes in pain over time are difficult to interpret and have probably different meaning to each individual and there is no way to objectively quantify it. So the patient's self report provides the most valid measure of the experience. The ideal pain measure should be sensitive, accurate ,reliable, valid and useful for both clinical and experimental conditions and should be able to separate the sensory aspects of pain from the emotional aspects.

Visual Analogue Scale(VAS):

VAS is most commonly a straight 100-mm line, that has the the words "no pain" at the left most end and "worst pain imaginable" at the right most end.Patients are instructed to place a mark on the line indicating the amount of pain that they feel at the time of evaluation.The distance of this mark from the left end is then measured, and this number is used as a numeric representation of the patient's pain.

no pain **worst imaginable pain**

Advantages:

1. Sensitive to changes in a pain experience.
2. Its ease and brevity of administration and scoring.
3. Relatively easy to understand for most patients.
4. Its minimal intrusiveness.
5. Its greater sensitivity to detect intervention-based changes in pain.

Disadvantages:

1. Its attempt in assigning a single value to a complex, multidimensional experience.
2. Assesses only one dimension(intensity) of a complex experience.
3. Has a ceiling at the upper most end .

OBSERVATION AND RESULTS

Table 1

AGE DISTRIBUTION

Age distribution in years	Group 1	Group 2
20 -30	6	5
30-40	12	14
40-50	6	4
>50	1	2
Total	25	25

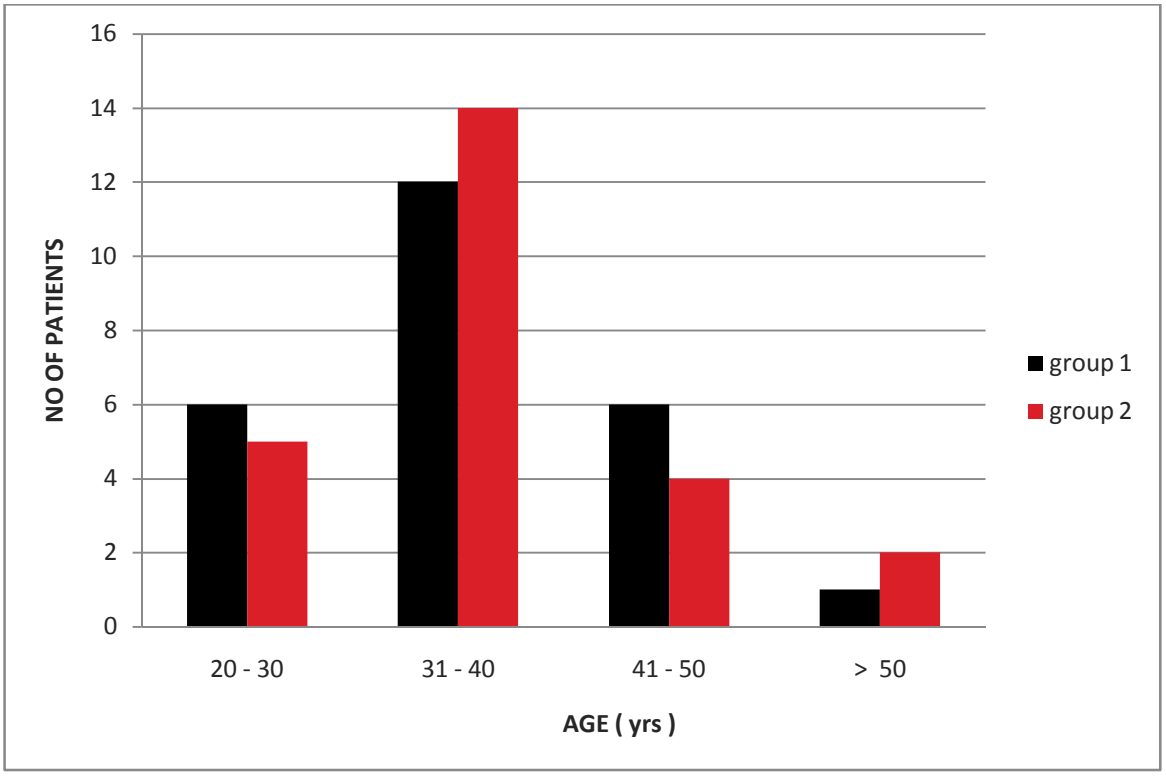
Mean age in years in group 1 is 35.20 ± 8.431

Mean age in years in group 2 is 37.70 ± 7.929

Table 2

SEX DISTRIBUTION

Sex	Group 1	Group 2
Male	18	16
Female	7	9
Total	25	25


16
14
12
10
8
6
4
2
0
NO OF PATIENTS
20 - 30
31 - 40
41 - 50
> 50
AGE (yrs)
group 1
group 2

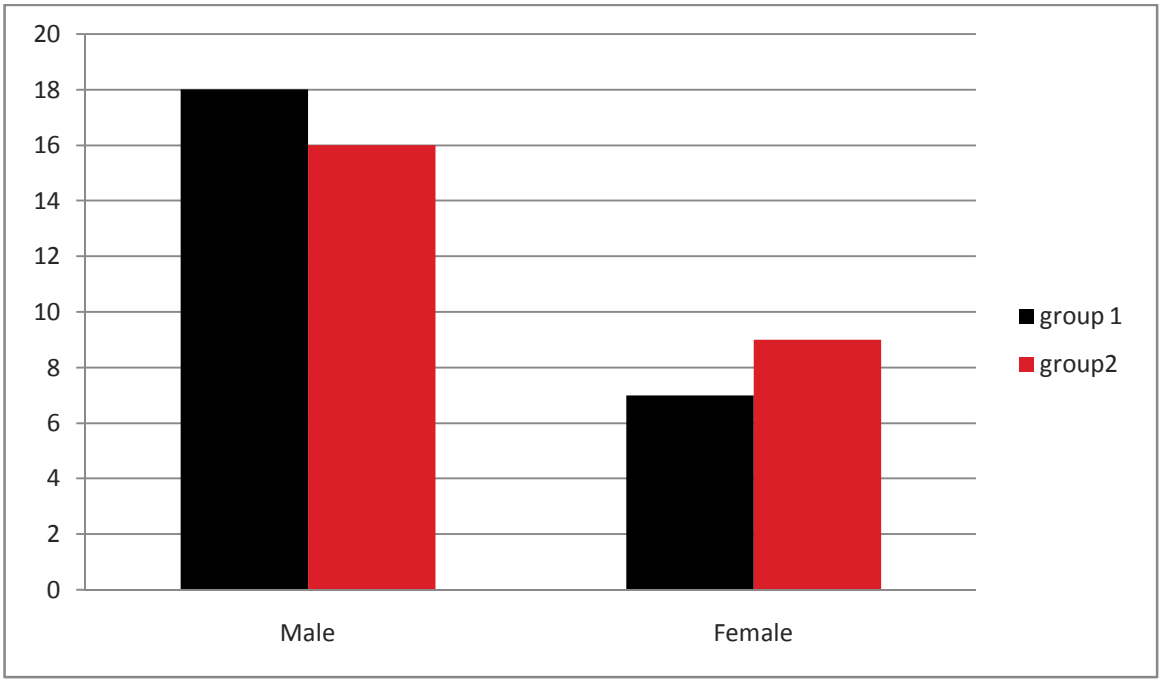

20
18
16
14
12
10
8
6
4
2
0
Male
Female
group 1
group2

Table 3

WEIGHT DISTRIBUTION

Weight in kgs	Group 1	Group 2
<50	6	5
50-60	14	10
>60	5	10
Total	25	25

Mean weight(kgs) in group 1 is 55.00 ± 6.843

Mean weight(kgs) in group 2 is 58.12 ± 6.629

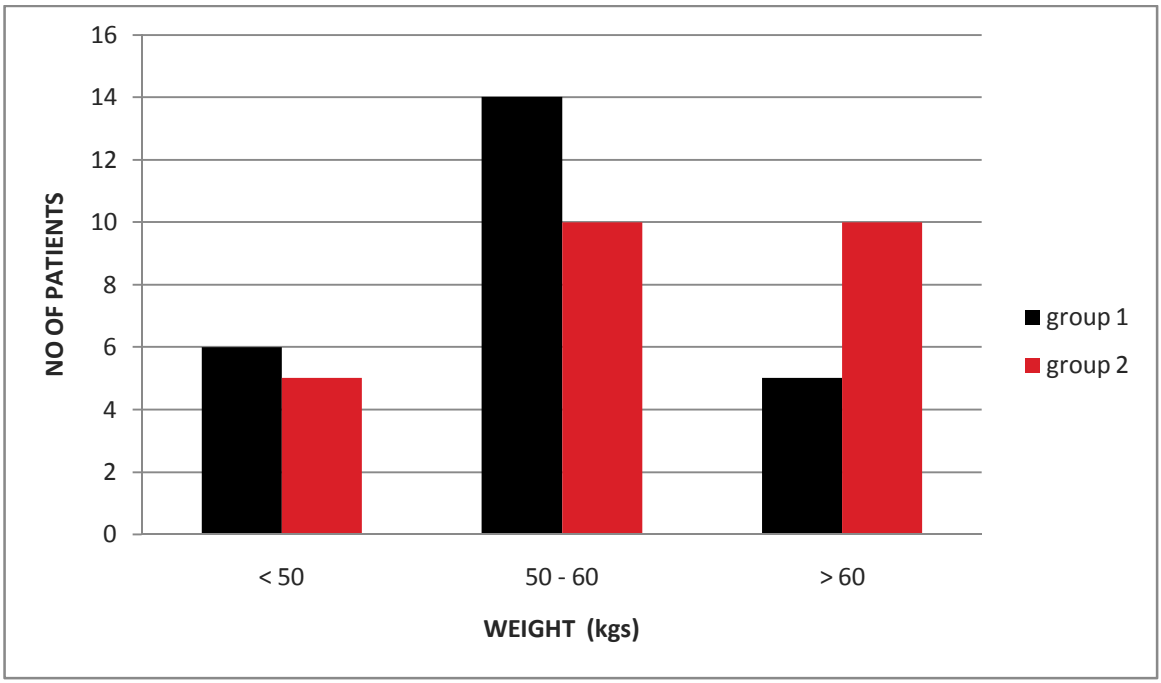

16
14
12
10
8
6
4
2
0
NO OF PATIENTS
< 50
50 - 60
> 60
WEIGHT (kgs)
group 1
group 2

Table 4

TIME OF ONSET OF SENSORY BLOCKADE

Time of onset of sensory block in minutes	Group 1	Group 2
< 5	0	3
5 -7	8	17
>7	17	5

Meantime of sensory block onset in group 1 is 7.88 ± 1.363

Meantime of sensory block onset in group 2 is 6.24 ± 1.714

The meantime required for onset of sensory block in group 1 was more than group 2.

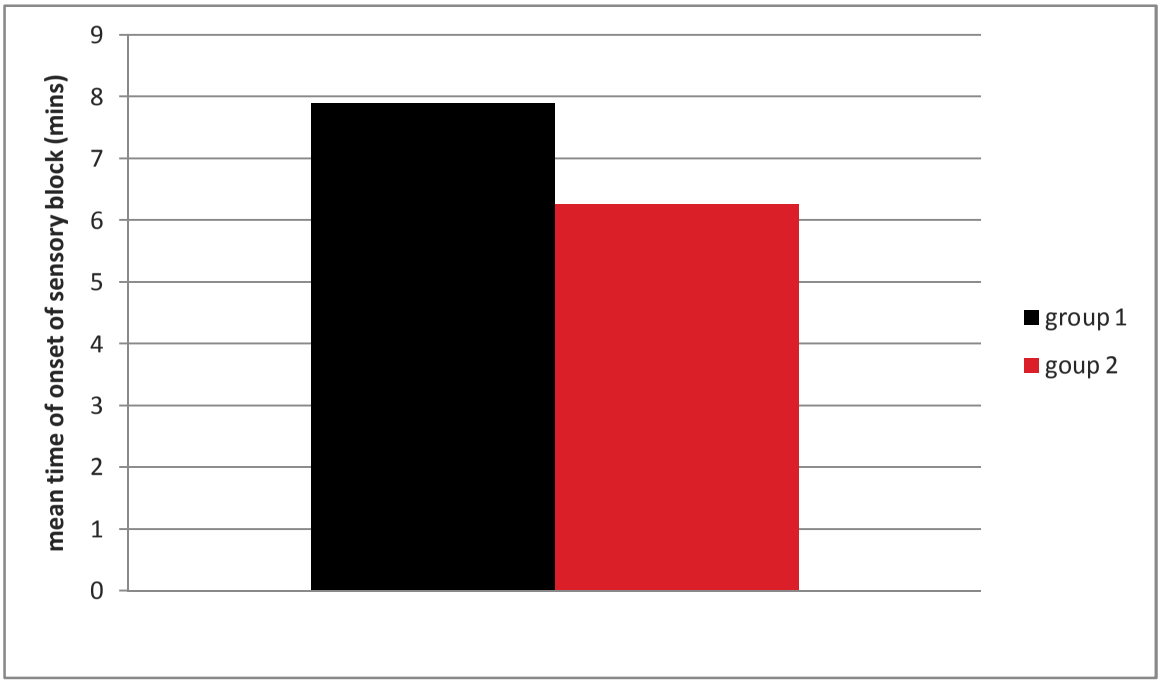

mean time of onset of sensory block (mins)
9
8
7
6
5
4
3
2
1
0
group 1
goup 2

Table 5

TIME OF ONSET OF MOTOR BLOCK

Time of onset of motor block in minutes	Group 1	Group 2
<5	0	0
5-10	6	16
>10	19	9

Meantime of onset of motor block in group 1 is 11.8 ± 1.825

Meantime of onset of motor block in group 2 is 9.88 ± 1.691

The mean time required for onset of motor block was more in group 1 than group 2.

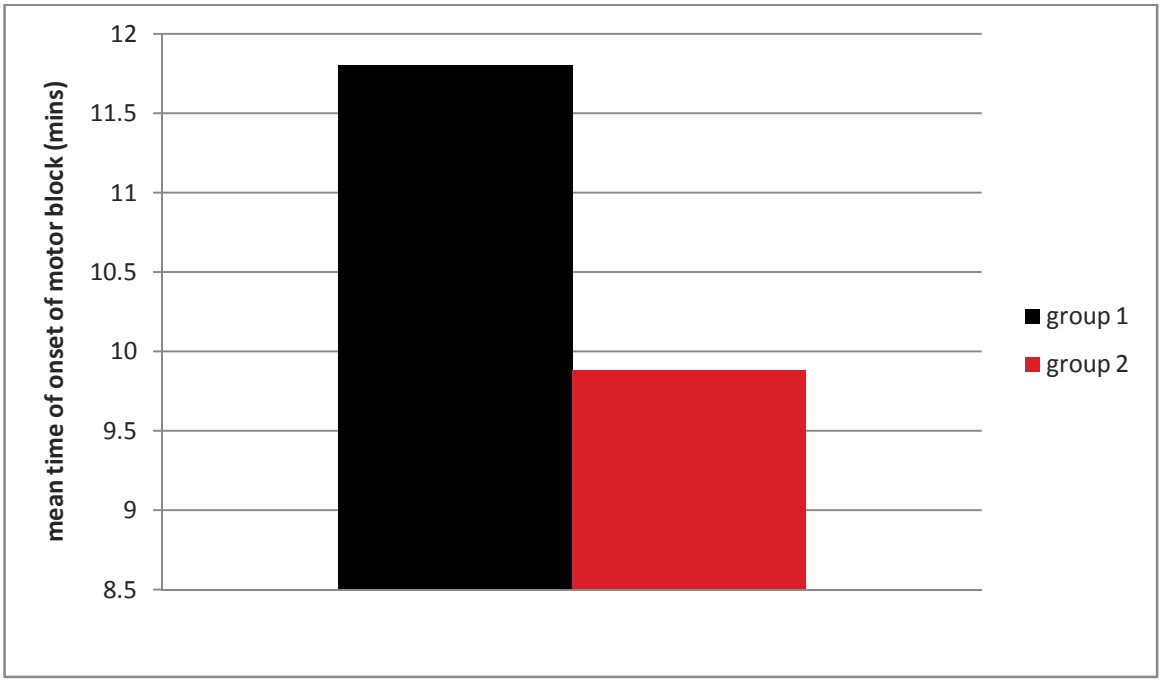

mean time of onset of motor block (mins)
12
11.5
11
10.5
10
9.5
9
8.5
group 1
group 2

Table 6

INCIDENCE OF TOURNIQUET PAIN DURING

I NTRAOPERATIVE PERIOD

Tourniquet time in minutes	Group 1	Group2
5	0	0
10	0	0
15	1	0
20	2	0
30	1	0
40	0	0

No of patients perceived tourniquet pain in group 1 is 4. In group 2 there was no incidence of tourniquet pain.

Table 7

TOTAL DURATION OF SURGERY

Total duration of surgery in minutes	Group 1	Group 2
<30	10	11
30-40	12	7
40-50	3	6
>50	0	1

Meantime of surgery(min) in group 1 is 30.6 ± 10.033

Meantime of surgery(min) in group 2 is 31.36 ± 11.365

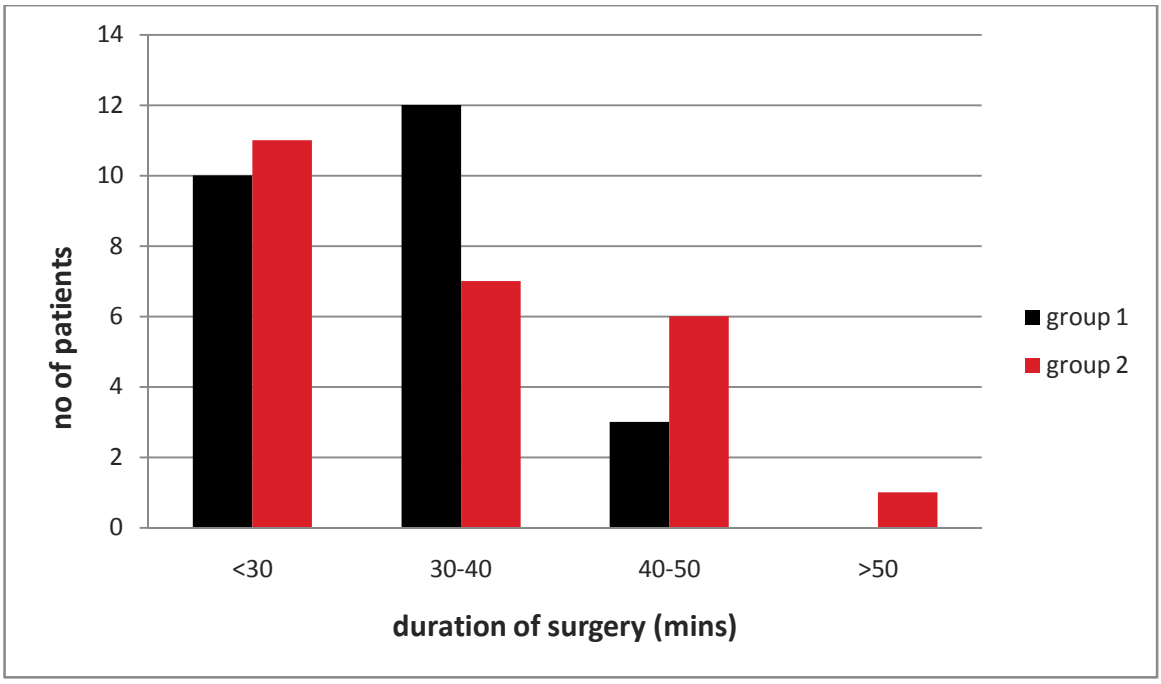
Table 8

TOTAL DURATION OF TOURNIQUET

Total duration of tourniquet in minutes	Group1	Group 2
30-40	19	18
40-50	5	4
>50	1	3

Meantime of duration of tourniquet (min) in group 1 is 37.4 ± 7.921

Meantime of duration of tourniquet (min) in group 2 is 38.00 ± 9.574


14
12
10
8
6
4
2
0
no of patients
<30
30-40
40-50
>50
duration of surgery (mins)
group 1
group 2

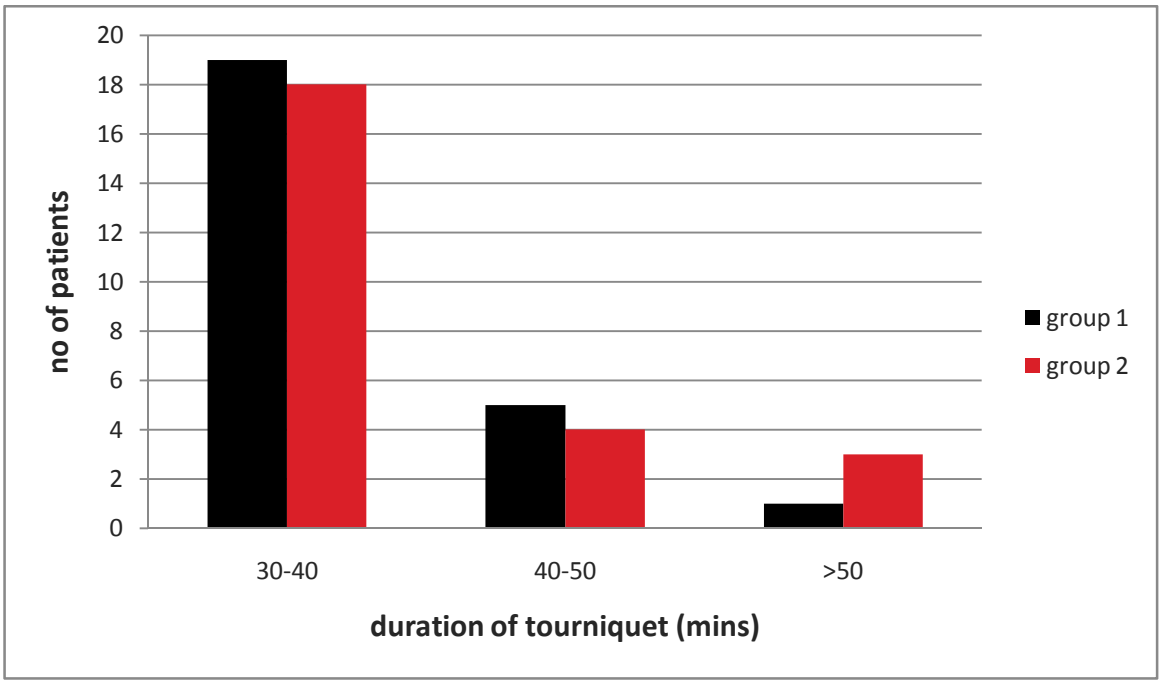

20
18
16
14
12
10
8
6
4
2
0
no of patients
30-40
40-50
>50
duration of tourniquet (mins)
group 1
group 2

Table 9

VARIATIONS IN BLOOD PRESSURE AND HEART RATE AFTER TOURNIQUET RELEASE

% of variation in blood pressure	Group 1	Group 2
0 – 10%	16	21
10 – 20%	3	2
>20%	4	1

Mean % of variation in rise in blood pressure in group 1 is 9.24 ± 8.664

Mean % of variation in rise in blood pressure in group 2 is 6.87 ± 4.559

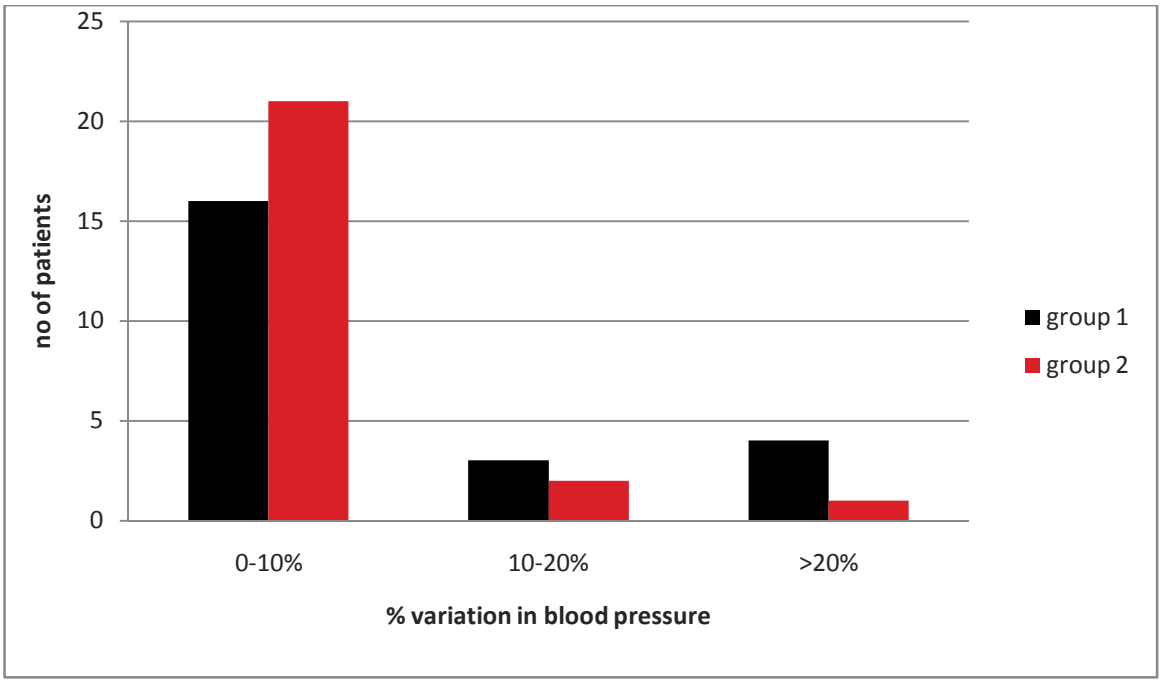
Two patients in group 1 and one patient in group 2 had fall in blood pressure after tourniquet release.

Table 10

% increase in heart rate	Group 1	Group 2
0 – 10%	16	15
10 – 20%	5	7
>20%	4	3

Mean % of increase in heart rate in group 1 is 9.34 ± 9.472

Mean % of increase in heart rate in group 2 is 10.39 ± 8.118


25
20
15
10
5
0
no of patients
0-10%
10-20%
>20%
% variation in blood pressure
group 1
group 2

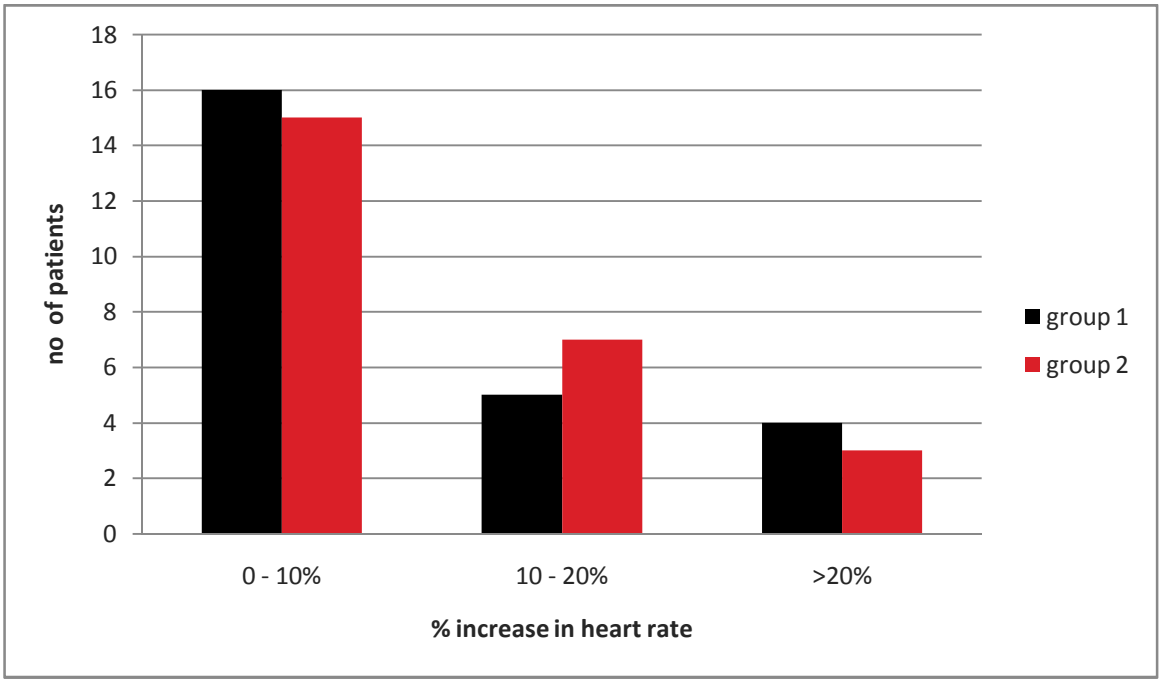

18
16
14
12
10
8
6
4
2
0
no of patients
0 - 10%
10 - 20%
>20%
% increase in heart rate
group 1
group 2

Table 11

DURATION OF POST OPERATIVE ANALGESIA (VAS>5)

Group	Meantime and standard deviation of post op analgesia (mins)
Group 1	120.76 ± 20.755
Group 2	137.52 ± 24.036

Meantime of post operative analgesia in group 2 is more than group 1.

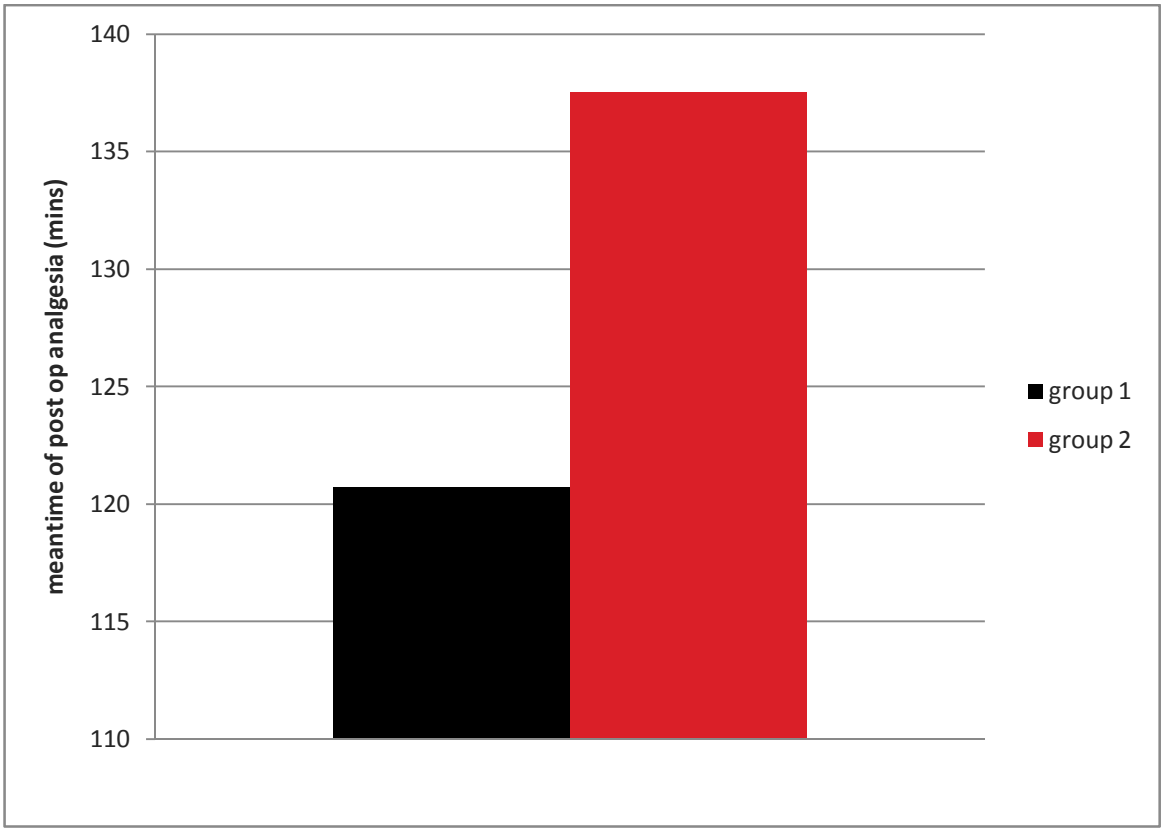

140
135
130
125
120
115
110
meantime of post op analgesia (mins)
group 1
group 2

Table 12

SIDE EFFECTS

Side effects	Group 1	Group 2
Tinnitus	0	0
Light headedness	2	1
Perioral numbness	0	0
Vomiting	0	1
Nausea	0	2
dizziness	1	0
Vertigo	0	0
Skin rashes	0	0
Arrhythmias	0	0
convulsions	0	0

Incidence of side effects in both groups are nearly same. Incidence of nausea and vomiting was slightly higher in group 2.

DISCUSSION

Demographic profile

	Group 1	Group 2	p value
Age	35.20 ± 8.431	37.70 ± 7.929	< 0.05
Weight	55.00 ± 6.843	58.12 ± 6.629	
Sex (m:f)	18:7	16:9	

The demographic profile of both groups is comparable. The difference between both the groups is statistically not significant.

	Group 1	Group 2	p value
Duration of surgery (mins)	30.6 ± 10.033	31.36 ± 11.365	0.803
Duration of tourniquet (mins)	37.4 ± 7.921	38.00 ± 9.574	0.810

In our study, there was no significant difference between the two groups for blood pressure, pulse rate during preoperative and intraoperative time. Meantime of duration of surgery and tourniquet duration of both groups showed no significant difference.

	Group 1	Group 2	p value
Onset of sensory block (mins)	7.88 ± 1.363	6.24 ± 1.714	< 0.05
Onset of motor block (mins)	11.8 ± 1.825	9.88 ± 1.691	<0.05

In our study, the sensory block onset time was quicker in ropivacaine plus fentanyl group(6.24+/-1.714min) when compared to ropivacaine group (7.88 +/- 1.363 min)and this was statistically significant. (p value 0.0005- independent sample test). These values were consistent with the findings of **T.T.Nieme** et al (2006).

The onset time of motor block also more in ropivacaine group (11.8 ± 1.825 min) when compared to ropivacaine with fentanyl group (9.88 ± 1.691 min). This was statistically significant. (p value = 0.0003 - paired t test). This finding correlated with the results of **Noelle hua lim et al, 1999.** They found that the addition of fentanyl and mivacurium to the prilocaine enhances the onset of motor blockade.

Iurie Acalovschi et al studied the effect of meperidine alone in intravenous regional anaesthesia. They concluded that the use of meperidine in IVRA developed sensory and motor block, demonstrating the local anesthetic action of the drug. The motor-blocking activities were more marked than the sensoryblocking activities.

Pang et al. in their study demonstrated the effect of tramadol in intravenous regional anaesthesia. They were able to induce a sensory block to pinprick, touch and cold at the intradermic injection site of 5% tramadol similar to that of 1% lidocaine. The suggested site of action of tramadol was the nerve endings and a possible associated central effect of tramadol was excluded because of the small doses used (25 mg). In our study, fentanyl a synthetic opiod also lowered the onset time of sensory and motor block when it was added to ropivacaine when compared to ropivacaine alone.

	Group 1	Group 2	p value
% variation in rise in blood pressure	9.24 ± 8.664	6.87 ± 4.557	0.675
% of increase in heart rate	9.34 ± 9.472	10.39 ± 8.118	0.245

The hemodynamic variations after tourniquet release were minimal in both the groups and the difference between the both groups is insignificant.

(p value >0.05).

In our study the incidence of tourniquet pain is higher in ropivacaine group. 4 patients in ropivacaine group perceived intra operative tourniquet pain while this was nil in patients who received fentanyl along with ropivacaine. This

finding correlated with the findings made by **Ibrahim Asif et al** who compared ropivacaine 0.2% and lignocaine 0.5%.

	Group 1	Group 2	p value
Duration of post op analgesia (mins)	120.76 ± 20.755	137.52 ± 24.036	0.011

The duration of post op analgesia was higher in ropivacaine plus fentanyl group (137.52 ± 24.036 min) when compared to ropivacaine group (120.76 ± 20.755 min) and this difference was statistically significant. (p value is 0.011 - student paired t test). **Lim and Ong, 2003** studied the effect of fentanyl with lignocaine and found post operative visual analog scores are significantly reduced in patients receiving fentanyl 1 mic/kg. This finding also correlated with the results of **Ashok kumar B Puttappa et al ,2010**.

The incidence of side effects was similar in both the groups. Patients who received fentanyl along with ropivacaine showed higher incidence of nausea and vomiting when compared to ropivacaine group. This result is similar to that of **M.T. Pikaknen, P.H. Rosenberg, 2007** who studied the effect of fentanyl along with prilocaine in intravenous regional anaesthesia and found that the incidence of nausea and vomiting is higher in fentanyl group.

SUMMARY

We conducted a randomised control study in 50 ASA I and II patients aged 19 to 60 for elective hand surgeries by intravenous regional anaesthetic technique at KAPV Govt medical college hospital, Trichy .

This study was to evaluate the,

1) anaesthetic efficacy,
2) post tourniquet release analgesia
3) side effects of ropivacaine 0.2% alone and to compare it with adding fentanyl 50 mics.

The patients were divided into two groups namely group 1 and group 2.

1) Sensory block onset,
2) motor block onset,
3) incidence of tourniquet pain,
4) post tourniquet release analgesia
5) side effects ,if any

were evaluated.

We found that fentanyl 50 mics when added with ropivacaine 0.2% in intravenous regional anaesthesia improved the anaesthetic efficacy, lengthened post tourniquet analgesia and reduced the incidence of tourniquet pain as compared to Ropivacaine alone, which has been proved statistically significant using quantitative and qualitative analysis.

CONCLUSION

We conclude that ropivacaine 0.2% with fentanyl 50 mics in intravenous regional anaesthesia has good anaesthetic efficacy, lengthened post operative analgesia and less incidence of intra operativetourniquet pain with minimal alterations in hemodyanamics after tourniquet release when compared to ropivacaine 0.2% alone.

REVIEW OF LITERATURES

1) Peng PW, Coleman MM, McCartney CJ, Krone S, Chan VW, Kaszas Z, Vucem 2002 Nov-Dec; 0.375% ropivacaine provides effective anesthesia and superior postoperative analgesia compared with 0.5% lidocaine when forearm IVRA is used.

2) Ropivacaine is effective for IVRA and improves postoperative analgesia. Muscle relaxants enhance the motor block and when combined with fentanyl allow for an equivalent quality of IVRA with 50% reduction in LA dose. Authors: Flamer D, Peng PW Published Date November 2011 Volume 2011:

3) Comparison of ropivacaine 2 mg ml^{-1} and prilocaine 5 mg ml^{-1} for i.v. regional anaesthesia in outpatient surgery

   T. T. Niemi1,*, P. J. Neuvonen2 and P. H. Rosenberg1, February 18, 2006 slightly slower recovery after ropivacaine in the innervation area of the median nerve ropivacaine plasma concentrations were markedly higher than those of prilocaine

4) Comparison of Ropivacaine 0.2% and Lidocaine 0.5% for Intravenous Regional Anesthesia in Volunteers ,Maximilian W. B. Hartmannsgruber, MD*, ,David G. Silverman, MD*, Thomas M. Halaszynski, DMD, MD*, Vonda Bobart, MD September 1999 vol. 89 no. 3 727 , postdeflation hypoalgesia and motor blockade were prolonged with ropivacaine, and

postdeflation lightheadedness, tinnitus, and drowsiness were more prominent with lidocaine.

5) IAshok Kumar B Puttappa MD FCARCSI Registrar in Anaesthesia, Mater U with Lignocaine, Fentanyl And Pancuronium, group L, 40 cc of 3mg/kg of 0.5% lignocaine diluted in normal saline was used for administering IVRA,and in patients of group F 40 cc of 1.5mg/kg of 0.25% lignocaine combined with fentanyl 1mg/kg , and in patients of group P 1.5mg/kg of 0.25% lignocaine combined with fentanyl 1mg/kg and pancuronium 0.5mg was used. The time for onset of sensory blockade in group L was 11.76.± 3.08 minutes , group F was 12.96±3.06 minutes , and group P was 10.20±3.52 minutes which was clinically comparable in all the groups. However the time for onset of motor blockade was significantly delayed in group F (21.1±2.7 minutes) as compared to other two groups: group L (12.5±2.6minutes and group P (11.36±3.5 minutes).
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10) Armstrong P, Power I, Wildsmith JA. Addition of fentanyl to prilocaine for intravenous regional anaesthesia. *Anaesthesia*. 1991;46(4):

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2) Asik I, Kocum AI, Goktug A, Turhan KS, Alkis N. Comparison of ropivacaine 0.2% and 0.25% with lidocaine 0.5% for intravenous regional anesthesia. *J Clin Anesth*. 2009;21(6):401–407.
3) Atanassoff PG, Ocampo CA, Bande MC, Hartmannsgruber MW,Halaszynski TM. Ropivacaine 0.2% and lidocaine 0.5% for intravenous regional anesthesia in outpatient surgery. *Anesthesiology*. 2001;95(3):
4) Chan VW, Weisbrod MJ, Kaszas Z, Dragomir C. Comparison of ropivacaine and lidocaine for intravenous regional anesthesia in Volunteers; a preliminary study on anesthetic efficacy and blood level. *Anesthesiology*. 1999;90(6)
5) Ismail O, El-Bahnasawe N, Badran A, Ali HG. Clinical efficacy of Ropivacaine 0.2% for intravenous regional anaethesia. *Egypt Anaesth*.2004;20(2):
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7) Heath ML. Deaths after intravenous regional anaesthesia. *Br Med J (Clin Res Ed)*. 1982;285(6346):913–914

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9) Brown BL, Fink BR. The history of neural blockade and pain management. In: Cousins MJ, Bridenbaugh PO, editors. *Neural Blockade in Clinical Anesthesia and Management Of Pain*. 3rd ed. Philadelphia, PA: Lippincott-Raven; 1998:3–34.

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12) Alayurt S, Memis D, Pamukcu Z. The addition of sufentanil, tramadol, or clonidine to lignocaine for intravenous regional anaesthesia. *Anaesth Intensive Care*. 2004;32(1):22–27.

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14) Budd K, Langford R. Tramadol revisited. *Br J Anaesth*. 1999;82(4):

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A comparative study between 0.2% ropivacaine and 0.2% ropivacaine with fentanyl 50 mics in intravenous regional anaesthesia

Name: Age/Sex: Wt:

IP no: Address:

Diagnosis: Surgery:

Group 1: 0.2% ropivacaine 40 ml

Group 2: 0.2% ropivacaine 40 ml plus fentanyl 50 mics

Tourniquet time: Proximal-

Distal –

Time for onset of sensory block:

Time for onset of motor block:

Incidence of intra op tourniquet

pain:

Duration of sugery :

Duration of tourniquet:

Duration of post op analgesia:

TOURNIQUET TIME	B.P	PULSE	SPO2	SIDE EFFECTS

Side effects after tourniquet release:

A comparative study of anaesthetic efficacy of 0.2% ropivacaine alone and 0.2% ropivacaine with fentanyl 50 mics in intravenous regional anaesthesia

Abstract of the study:

A longer acting local anaesthetic ropivacaine has been successfully used in intravenous regional anaesthesia. Fentanyl, a synthetic opiod in a dose of 50 mics was used along with 0.2% Ropivacaine in this study and the anaesthetic efficacy was compared with 0.2% ropivacaine alone. AIM OF THE STUDY: The anaesthetic efficacy regarding onset of sensory and motor block, incidence of intraoperative tourniquet pain, duration of post op analgesia, variation in hemodyanamics after tourniquet release and side effects, if any were noted and compared. MATERIALS AND METHODS: 50 patients of ASA 1,2 status undergoing elective hand surgeries were included in this study. They were randomly allocated into 2 groups. Group 1 received 40 ml of 0.2% ropivacaine alone. Group 2 received 40 ml 0.2% ropivacaine along with fentanyl 50 mics. Onset time of sensory and motor block, incidence of tourniquet pain, variations in heart rate and blood pressure after tourniquet release, duration of post op analgesia and side effects were noted. RESULTS: Both the groups were comparable with respect to demographic characteristics. Onset of sensory block (mins) in group 1 was 7.88 ± 1.363 and in group 2 it was 6.24 ± 1.714 and this difference was statistically significant ($p < 0.05$). Onset of motor block in group 1 is 11.8 ± 1.825 mins and in group 2 9.88 ± 1.691. ($p < 0.05$). Meantime of duration of surgery

and tourniquet in both the groups showed no difference statistically. There was no significant difference in variations in hemodynamics after tourniquet release. Four patients in group 1 perceived intraoperative tourniquet pain and this was nil in patients who received fentanyl along with ropivacaine. The incidence of nausea and vomiting was present in patients who received fentanyl along with ropivacaine. Duration of post op analgesia (VAS > 5) in group 1 was 120.76 ± 20.755 mins and in group 2 it was 137.52 ± 24.036 mins and this difference is statistically significant ($p < 0.05$). CONCLUSION: We conclude that ropivacaine 0.2% with fentanyl 50 mics in intravenous regional anaesthesia has good anaesthetic efficacy, lengthened post operative analgesia and less incidence of intra operative tourniquet pain with minimal alterations in hemodyanamics after tourniquet release when compared to ropivacaine 0.2% alone.

									DURATION	DURATION		ON TOUR RELEASE							
SN	NAME	AGE	SEX	WT.	IP NO	DIAGNOSIS	procedure	SO	MO	SURGERY	TOURNIQUET	TOURNIQUET PAIN	SYS BP	PR	BP	PR	VAR IN BP	VAR IN PR	POST OP ANAL (min	COMPLICATION
1	rajendran	33	M	56	34523	inj L hand	tendon repair	9	11	35	40		130	82	140	83	7.69	1.21	120	
2	marimuthu	38	M	45	37654	cut injury R hand	tendon repair	8	12	40	45		128	88	132	90	3.12	2.27	148	
3	ramasamy	45	M	45	21763	inj R thumb	K wiring	6	13	20	30		118	79	128	82	8.47	3.79	140	dizziness
4	kokilavani	37	F	57	11123	ganglion l wrist	excision	8	14	25	30	at 15 mins	122	86	142	86	16.39	0	96	
5	arumugam	37	M	57	26713	imp dermoid	excision	8	12	20	30		136	91	138	94	1.47	3.29	122	
6	pattapan	47	M	58	20934	inj L hand	excision	9	14	35	40		130	78	152	80	16.92	2.56	105	
7	udhayadasan	38	M	46	13942	inj F4F5	WD &k wiring	8	10	40	45		122	69	130	71	6.55	2.9	130	
8	veerasamy	33	M	58	40651	cut injury R hand	tendon repair	9	12	45	50		130	72	136	75	4.61	4.16	158	
9	divakaran	28	M	64	51982	ganglion R hand	excision	6	12	30	35		116	74	122	78	5.17	5.4	120	
10	ranidevi	42	F	52	22981	ganglion L hand	excision	7	11	25	30		112	80	142	98	26.78	22.5	135	light headedness
11	rajapandian	31	M	39	52349	inj F4F5	WD &k wiring	9	14	45	50		132	92	110	98	-16.66	6.52	100	
12	selvi	33	F	49	51897	cut injury R hand	tendon repair	6	12	45	50	AT 20 mins	128	78	136	102	6.25	33.33	75	
13	vijayakumar	26	M	54	49812	ganglion R hand	excision	8	15	30	35		120	76	120	98	0	28.94	120	
14	rajarajan	23	M	58	52076	inj L hand	K wiring	9	14	15	30		136	68	138	76	1.47	11.76	145	
15	mariappan	52	M	52	51555	ganglion L hand	excision	6	10	20	30		140	81	124	82	-11.42	1.23	137	lightheadedness
16	arivoli	49	M	66	41423	inj L index	WD &k wiring	6	9	35	40		124	67	138	75	11.29	11.94	160	
17	umar sheik	34	M	48	29123	cut injury R hand	tendon repair	7	9	35	40	at 20 mins	122	76	156	87	27.86	14.47	112	
18	abdul razak	45	M	54	25412	inj L hand	excision	8	11	25	30		120	72	148	91	23.33	26.38	120	
19	lakshmi	21	F	64	33345	finger nodule	excision biopsy	8	11	20	30		128	79	132	85	3.12	7.6	116	
20	amutha	24	F	59	18723	foreign body	excision	11	13	30	35		132	82	136	96	1.51	17.07	98	
21	velyudham	36	M	62	44456	imp dermoid	excision	9	11	35	40		116	84	142	94	22.41	11.9	112	
22	praveenbabu	32	M	64	54123	cts	decompression	10	15	15	30		114	96	120	98	5.26	2.08	128	
23	silambayee	31	F	58	28754	ganglion R hand	excision	8	11	30	35		132	78	136	82	3.03	5.13	120	
24	adithyan	24	M	53	45198	raw area finger	flap cover	8	10	50	55	AT 30 mins	122	83	`124	84	1.64	1.2	90	
25	rasathi	41	F	57	25681	ganglion l wrist	excision	6	9	20	30		120	83	130	88	8.33	6.02	112	

SN	NAME	AGE	SEX	WT. k	IP NO	DIAGNOSIS	procedure	SO	MO	DURATION SURGERY	DURATION TOURNIQUE	TOURNIQUE SYS BP	PR	ON TOUR BP	RELEASE PR	VAR IN BP	VAR IN PR	POST OP ANAL (mir	COMPLICATION
1	selvakumari	31	F	56	10465	dermoid	excision	8	9	25	30	118	78	128	83	8.47	6.41	130	
2	aravanan	35	M	60	11234	ganglion R hand	excision	7	11	25	30	120	72	132	88	10	22.22	160	
3	subramani	54	M	49	23459	inj R thumb	K wiring	6	10	35	40	102	86	112	96	9.8	11.62	142	
4	chandra	36	F	55	45678	finger nodule	excision biopsy	8	12	30	35	114	77	120	86	5.26	11.68	128	
5	selvaraj	35	M	48	28934	imp dermoid	excision	6	9	20	30	132	86	138	94	4.54	9.3	150	nausea, vomiting
6	alagesan	42	M	70	34321	foreign body	excision	6	11	30	35	128	74	136	78	6.25	5.4	140	
7	veerapan	36	M	67	43214	lacerated wound thumb	suturing	5	9	20	30	124	72	130	74	4.83	2.77	98	
8	veerasamy	32	M	62	54378	cut injury R hand	tendon repair	7	10	50	55	138	90	126	92	-8.69	2.22	128	
9	mohanasundaram	37	M	48	743216	raw area finger	ssg	6	8	30	35	126	79	128	89	1.58	12.65	148	
10	alphonse	40	F	55	10621	ganglion L hand	excision	7	11	15	30	138	81	132	88	4.34	8.65	140	
11	sivakumar	36	M	64	12754	imp dermoid	excision	5	8	35	40	130	90	132	98	1.53	8.88	168	dizziness
12	chitra	39	F	51	20954	cut injury R hand	tendon repair	6	10	19	30	140	112	136	122	21.42	8.92	170	
13	arumugam	29	M	59	34521	ganglion R hand	excision	8	12	35	40	122	75	126	87	3.27	16	136	
14	ramkumar	28	M	61	41754	inj L hand	K wiring	4	9	30	35	124	68	130	77	4.83	13.23	180	
15	arokiasamy	58	M	49	38943	trigger finger	release	6	10	55	60	118	88	124	89	5.08	1.13	140	nausea, vomiting
16	dhandapani	46	M	67	32786	inj L index	WD &k wiring	4	9	50	55	114	76	120	84	5.26	10.52	120	
17	kannapiran	49	M	66	43768	cut injury R hand	tendon repair	3	9	40	45	126	79	138	89	9.52	12.65	160	
18	kumarasamy	43	M	49	43675	ganglion l wrist	excision	11	14	15	30	122	81	136	88	11.47	8.64	120	nausea, vomiting
19	neela	28	F	58	23908	cts	decompression	5	7	25	30	130	85	140	87	7.69	2.35	100	
20	mallika	27	F	55	32987	foreign body	excision	6	8	25	30	124	68	130	94	4.83	38.23	90	
21	ramasamy	36	M	65	42167	imp dermoid	excision	9	13	45	50	114	80	130	99	14.03	23.75	120	
22	selvam	36	F	64	23145	cts	decompression	5	8	40	45	130	92	130	94	0	2.17	130	
23	archana	39	F	57	20983	ganglion R hand	excision	5	10	20	30	120	81	132	87	10	7.4	110	
24	ravikumar	29	M	61	16792	cts	decompression	7	11	45	50	122	78	130	84	6.55	7.69	160	
25	neelaveni	42	F	57	13876	ganglion l wrist	excision	6	9	25	30	134	91	140	96	4.47	5.49	170	