DISSERTATION ON

PROSPECTIVE RANDOMISED CONTROLLED STUDY EVALUATING DEXMEDETOMIDINE AS AN ADJUVANT ADDED TO A LOCAL ANAESTHETIC MIXTURE OF BUPIVACAINE, LIGNOCAINE WITH ADRENALINE IN AXILLARY PLEXUS BLOCK AS COMPARED TO AXILLARY PLEXUS BLOCK WITH LOCAL ANAESTHETIC MIXTURE ALONE

Dissertation submitted in partial fulfillment of the regulations for the award of the degree of

M.D. DEGREE, BRANCH – X

ANESTHESIOLOGY

Of

TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

ESIC- MEDICAL COLLEGE & POSTGRADUATE INSTITUTE OF MEDICAL SCIENCE AND RESEARCH, KK NAGAR, CHENNAI- 78.

APRIL 2016

ENDORSEMENT BY THE DEAN/

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DEAN
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DECLARATION

I solemnly declare that this dissertation entitled "**PROSPECTIVE RANDOMISED CONTROLLED STUDY EVALUATING DEXMEDETOMIDINE AS AN ADJUVANT ADDED TO A LOCAL ANAESTHETIC MIXTURE OF BUPIVACAINE, LIGNOCAINE WITH ADRENALINE IN AXILLARY PLEXUS BLOCK AS COMPARED TO AXILLARY PLEXUS BLOCK WITH LOCAL ANAESTHETIC MIXTURE ALONE"** has been conducted by me at ESIC Medical College & PGIMSR, Chennai, under the guidance and supervision of **Dr. RADHIKA, M.D. and Dr. ILANGO GANESAN, M.D.,** Department of Anaesthesiology, ESIC Medical College & PGIMSR, Chennai. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.D. Branch X (Anaesthesiology).**

Date :

Place : Chennai (Dr. Joanny Benjamin)

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I am thankful to the Institutional Ethical Committee for their guidance and approval for the study.

I also thank the theatre staff for their help. I would like to thank all the patients for their participation and co-operation.

I am thankful to my parents and family members for their unconditional love and support.

Dr. Joanny Benjamin

CERTIFICATE OF APPROVAL

To

Dr. Joanny Benjamin,
PG in Department of Anesthesia,
ESIC Medical College & PGIMSR,
KK Nagar, Chennai-78.

Dear Dr. Joanny Benjamin,

The Institutional Ethical Committee of ESIC Medical College & PGIMSR reviewed and discussed your application for approval of the proposal entitled **"Prospective Randomized controlled study evaluating dexmedetomidine as an adjuvant added to a local anesthetic mixture of Bupivacaine, Lignocaine with adrenaline in axillary plexus block as compared to axillary plexus block with local anesthetic mixture alone"** at ESIC Medical College & PGIMSR, K K Nagar, Chennai 600 078 , No. **12/27/10/2014**.

The following members of the Ethical Committee were present in the meeting held on 27.10.2014 conducted at ESIC Medical College & PGIMSR, KK Nagar, Chennai-78.

S.No.	ETHICAL COMMITTEE MEMBERS
1.	Prof. A.V. Srinivasan, Chairperson, EC Member EMERITUS Professor, The Tamilnadu Dr. MGR Medical University Former Prof. & HOD., of Institute of Neurology, Madras Medical College
2.	Prof. V. Rajalakshmi, Vice Principal, ESIC Medical College & PGIMSR, EC Member
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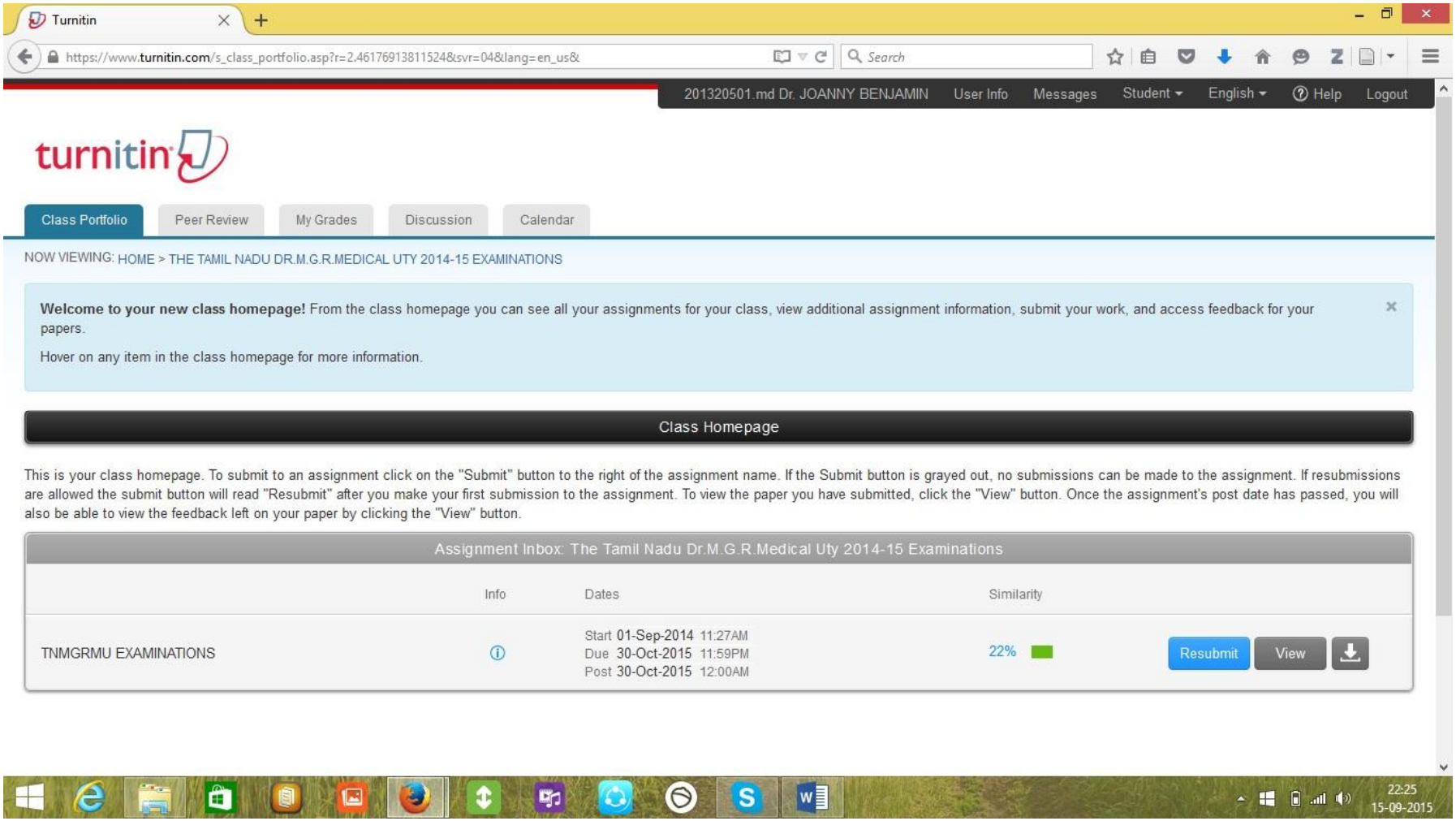
14.	Prof. C. Rajendiran, Department of General Medicine, EC Member
15	Dr. C.V. Aravindan, Scientist, EC Member
16.	Shri. K M Venugopal, Advocate, EC Member

The proposal is approved to be conducted in its presented form.

The Institutional Ethical Committee expects to be informed about the progress of the study and significant adverse effects occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

[DR. A.V. SRINIVASAN]
CHAIRPERSON
ETHICAL COMMITTEE

Date : 27.10.2014
Place : Chennai 78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DISSERTATION ON

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CONTENTS

ABSTRACT

TITLE :

PROSPECTIVE RANDOMISED CONTROLLED STUDY EVALUATING DEXMEDETOMIDINE AS AN ADJUVANT ADDED TO A LOCAL ANAESTHETIC MIXTURE OF BUPIVACAINE, LIGNOCAINE WITH ADRENALINE IN AXILLARY PLEXUS BLOCK AS COMPARED TO AXILLARY PLEXUS BLOCK WITH LOCAL ANAESTHETIC MIXTURE ALONE

SUBMITTED BY:

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BACKGROUND: Regional anaesthesia in the form of Brachial plexus block is an attractive option when compared to General anaesthesia, since it has the advantages of post-operative pain relief as well as decreased nausea and vomiting. This makes regional anaesthesia the preferred technique in day care surgery. Axillary plexus block is one of the most commonly performed regional nerve block for upper limb surgeries. The proximity of the nerves of brachial plexus to the axillary artery makes the identification of structures easy with both nerve stimulator and ultrasound guided block.

AIM: To compare the analgesic efficacy of dexmedetomidine added to bupivacaine, lignocaine with adrenaline mixture versus bupivacaine, lignocaine with adrenaline mixture alone in axillary plexus block for upper limb surgeries.

METHODS: This study is a prospective randomized controlled study. The study involved 60 patients. They were randomly divided into two groups, Group RD received axillary plexus block with 50 microgram of dexmedetomidine added to local anaesthetic mixture and Group RL received axillary plexus block with local anaesthetic mixture alone. Both the groups were followed up for the onset of sensory and motor blockade and the duration of analgesia using the Visual Analog Scale score.

RESULTS: Both the study and control groups were comparable in terms of age, height, weight and BMI. The mean arterial pressure of the patients in the dexmedetomidine group was 80.27 ± 6.39 mmHg whereas the mean arterial pressure in the control group was 97.47 ± 8.16 mmHg at the 45^{th} minute. The mean pulse rate of the patients in the dexmedetomidine group was around 70 bpm and around 85 bpm in the control group but none of the patients required atropine. The onset time of sensory block in the dexmedetomidine group was found to be 9.37 ± 1.7 minutes whereas in the control group it was found to be 15.83 ± 1.18 minutes. The onset time of motor block in the dexmedetomidine group was found to be 12.23 ± 1.85 minutes whereas in the control group it was found to be 18.77 ± 1.22 minutes. The mean duration of analgesia in the dexmedetomidine group was found to be 16.53 ± 1.41 hours whereas the mean duration of analgesia in the control group was found to be 9.70 ± 1.44 hours.

CONCLUSION: Dexmedetomidine in the dose of 50 microgram when added as an adjuvant to local anaesthetic mixture in axillary plexus block had a faster onset of sensory, motor block and significantly prolonged the duration of analgesia. They caused a minimal decrease in the mean arterial pressure and heart rate which did not warrant treatment.

KEY WORDS: Dexmedetomidine, axillary block, Visual Analog Scale (VAS)

INTRODUCTION

Regional anaesthesia in the form of Brachial plexus blocks is an attractive option when compared to General anaesthesia, since it has the advantages of post-operative pain relief as well as decreased nausea and vomiting. This makes regional anaesthesia the preferred technique in day care surgery[(1)].

Axillary plexus block is one of the most commonly performed regional nerve block for upper limb surgeries[(2)]. The proximity of the nerves of brachial plexus to the axillary artery makes the identification of structures easy with both nerve stimulator and ultrasound guided block.

Brachial plexus can be approached by any of the four techniques – inter-scalene approach, supra-clavicular approach, infra-clavicular approach and axillary approach[(3)]. Brachial plexus block is performed by placing the tip of the needle close to the nerves of the plexus and injecting the local anaesthetic solution.

The axillary plexus block is the safest of the four approaches to the brachial plexus[(4)]. It does not cause pneumothorax or phrenic nerve palsy caused by the other approaches of brachial plexus block. It has

many advantages over general anaesthesia like reduced risk of aspiration, increased post-operative pain relief and decreased post-operative opioid requirements.

Axillary plexus block was described by Halstead in 1884[(5)]. It can be used for surgeries involving the elbow, forearm and hand surgeries providing excellent analgesia and anaesthesia[(6)].

Local anaesthetic combinations of lignocaine with adrenaline and bupivacaine are injected into the axillary sheath to provide axillary plexus blockade. Different agents have been tried as adjuvants, added to the local anaesthetics to fasten the onset and prolong the duration of sensory and motor blockade. Some of the agents which have been studied are Dexamethasone, Fentanyl, Midazolam, Buprenorphine, Tramadol and Clonidine.

Alpha – 2 agonists like clonidine and dexmedetomidine have multiple benefits like analgesia, anaesthesia, sedation and sympatholysis. Dexmedetomidine is a centrally acting, selective alpha – 2 adrenergic agonist and is more potent than clonidine[(7)].

Dexmedetomidine produces sedation, anxiolysis[8], decreases anaesthetic requirement and maintains good hemodynamic stability intraoperatively. When a low dose of dexmedetomidine is added to the local anaesthetic mixture, it provides a faster onset of sensory and motor blockade and prolongs the duration of sensory and motor blockade without much hemodynamic instability.

Dexmedetomidine has different mechanisms of action[9] like:

1. Direct action on peripheral nerve
2. Central action and
3. Attenuation of inflammatory response.

In this study we investigated the addition of dexmedetomidine as an adjuvant to the local anaesthetic mixture in axillary plexus block, in comparison with local anaesthetic mixture alone in order to evaluate the beneficial effects of dexmedetomidine.

Aim of the Study

AIM OF THE STUDY

The aim of the study was to compare the analgesic efficacy of dexmedetomidine added to bupivacaine, lignocaine with adrenaline mixture versus bupivacaine, lignocaine with adrenaline mixture alone in axillary plexus block for upper limb surgeries.

Objectives

OBJECTIVES

The Objective of the study was to compare the

1. Onset of sensory and motor blockade and
2. Duration of analgesia between the two groups of patients who underwent upper limb surgeries under axillary block, with or without Dexmedetomidine.

Review of Literature

REVIEW OF LITERATURE

C A Mackay and D F Bowden[10] conducted a study in 1997 titled "Axillary brachial plexus block--an underused technique in the accident and emergency department". The main objective of the study was to compare the axillary plexus block with Bier's block for providing upper limb analgesia. Patients posted for elective upper limb procedures were given either axillary plexus block or Bier's block. Axillary block was performed with either perivascular or trans-arterial technique and the patients received 40ml of 1% lignocaine with adrenaline. Bier's block was performed with a single cuff tourniquet and received 3mg/kg of 0.5% prilocaine. Seventy five patients were included in the study. 39 patients received axillary plexus block and 36 patients Bier's block. The onset of anaesthesia was slower with axillary block group. But the duration of analgesia and patient satisfaction was more with axillary block group.

O'Donnell BD et al[11] conducted a study in 2009 named, "Ultrasound-guided axillary brachial plexus block with 20 millilitres local anesthetic mixture versus general anaesthesia for upper limb trauma surgery: an observer-blinded, prospective, randomized, controlled trial". The study was a randomised double blind controlled

study. The patients were randomised into two groups either ultrasound guided axillary block or general anaesthesia. Ultrasound guided axillary plexus block was performed using needle out of plane approach and 5ml of local anaesthetic mixture containing 2% lignocaine with adrenaline, 0.5% bupivacaine and 75microgram/ml of clonidine was injected in the respective nerve distributions of medial, ulnar, radial and musculocutaneous nerves. General anaesthesia was induced with fentanyl and propofol, maintained with O2/N2O/Sevoflurane mixture. Visual Analog pain scores were lower in patients who were given ultra-sound guided block. The patients had satisfactory anaesthesia and earlier discharge in the ultra-sound guided nerve block group.

Fatma Nur Kaya et al[(12)] conducted a study in 2009 regarding the effect of intra-venous dexmedetomidine in prolonging bupivacaine spinal anaesthesia. It was a double blinded randomised placebo controlled study. After obtaining written informed consent, seventy five patients classified under the ASA I and II category and undergoing TURP surgery under spinal anaesthesia were included in the study. After insertion of an 18gauge intra-venous catheter, the patients were divided into three groups – group one received 0.5microgram/kg of dexmedetomidine intravenously, group two received 0.05mg/kg of

midazolam intravenously and group three received physiological saline. Bupivacaine 0.5% 3ml was injected intrathecally. Sensory block was higher with dexmedetomidine group. Time for sensory regression of two dermatomes was longer with dexmedetomidine. Dexmedetomidine increased the time to first request of analgesic and the maximum Ramsay sedation score attained was greater with dexmedetomidine than with midazolam or saline.

Imasogie N[(13)] conducted a study in 2010 titled "A prospective, randomized, double-blind comparison of ultrasound-guided axillary brachial plexus blocks using 2 versus 4 injections". The study was a randomised double blind controlled study. One hundred and twenty patients posted for elective upper limb surgeries were divided into two groups. Group I received axillary plexus block with a 2 point skin puncture, 30ml of 0.5% ropivacaine was injected posterior to the axillary artery and the remaining 10ml of local anaesthetic for the musculocutaneous nerve. Group II received 40ml of 0.5% local anaesthetic using separate 10ml injections to the radial, ulnar, median and musculocutaneous nerves. The 2 – point injection technique was faster to administer. But the percentage of patients with complete block at 30minutes and the block success rate were similar in both the groups.

Hala S. Abdel-ghaffar[14] performed a study in 2011 regarding the effect of "Efficacy and safety of intraoperative dexmedetomidine in pediatric posttonsillectomy pain: Peritonsillar versus intravenous administration". The study design was a prospective randomised controlled double blind study. Eighty four children in the age group 5-12 years of age posted for elective tonsillectomy procedures were included in the study. After obtaining approval from the Institutional Ethical committee and written informed consent from the legal guardians, the children were divided into three groups using computer generated random numbers.

- Group I (DEX IV) – received 1microgram/kg of dexmedetomidine intravenously diluted in 50ml of 0.9% normal saline infused over 10minutes after induction of anaesthesia.
- Group II (DEXPT) – received 1microgram/kg of dexmedetomidine diluted in 4ml of 0.9% normal saline given by peritonsillar infiltration after intubation of trachea.
- Group III (Placebo) – received 50ml of 0.9% normal saline intravenous infusion and 4ml of 0.9% normal saline peritonsillar infiltration.

Anaesthesia was induced with propofol 2-3mg/kg and atracurium 0.5mg/kg given to facilitate endotracheal intubation. No NSAIDs, opioid, paracetamol or additional propofol were given during the procedure. Neuromuscular reversal done with neostigmine 0.05mg/kg and atropine 0.02mg/kg and the children were extubated awake after return of protective airway reflexes. They were then placed in the post-tonsillectomy recovery position. The time to extubation was significantly prolonged in group DEX IV. The time to first analgesic requirement was also significantly prolonged in group DEX IV and DEXPT. The mean usage of paracetamol rescue analgesia was significantly lower in group DEX IV and DEXPT. The mean sedation scores were significantly higher in group DEX IV. The request for more than one analgesic dose was higher in the Placebo group.

Hala E A Eid MD[15] conducted a study regarding the effect of dose related prolongation of spinal anaesthesia with the addition of dexmedetomidine to hyperbaric bupivacaine. Forty eight adult patients under the ASA I and II physical status scheduled for elective anterior cruciate ligament repair were included in the study. They were randomised into three equal groups – group D1 received 3ml of 0.5% hyperbaric bupivacaine and 10microgram of dexmedetomidine, group

D2 received 3ml of 0.5% hyperbaric bupivacaine and 15microgram of dexmedetomidine, group B received 3ml of 0.5% bupivacaine and normal saline. The time to 2-segment regression and sensory regression was significantly prolonged in both the dexmedetomidine groups. The post-operative pain scores were significantly reduced in groups D1 and D2. The post-operative Ramsay sedation scores were also significantly increased in the dexmedetomidine groups.

D. Marhofer et al[16] performed a study in 2012 regarding the effect of dexmedetomidine when added to ropivacaine as an adjuvant to prolong peripheral nerve blockade. After obtaining approval from the Ethical committee and getting informed consent, thirty six adult patients in the age group 18 – 45 years planned for volunteer controlled study with an ultra-sound guided ulnar nerve block were included in the study. Patients with anatomical abnormalities of the forearm, obesity (BMI > 30Kg/m2), coagulopathy and known allergy to local anaesthetics were excluded from the study. After starting an intra-venous cannula, ultrasound visualisation of the ulnar nerve was done and ulnar nerve block was performed (UNB).

- Ropivacaine (R group) – received 3ml of 0.75% ropivacaine plus 0.2ml of saline and iv 5ml of saline.

- Ropivacaine plus peri-neural dexmedetomidine (RpD) – received 3ml of 0.75% ropivacaine plus 20microgram of dexmedetomidine and iv 5ml of saline.

- Ropivacaine with systemic dexmedetomidine (RsD) – received 3ml of 0.75% ropivacaine plus 0.2ml of saline and 20microgram of dexmedetomidine mixed with 4.8ml of saline iv.

The onset of sensory block was not statistically significant between the three groups. The onset time of motor block was significantly faster in the group RpD (perineural dexmedetomidine). The duration of sensory and motor blockade was significantly increased when 20microgram of dexmedetomidine is added peri-neurally or systemically.

H. Kang et al[17] conducted a study in 2012 regarding the effect of dexmedetomidine when added to pre-emptive ropivacaine infiltration for post-operative pain after inguinal herniorrhaphy. Fifty two adult male patients posted for elective hernia repair were randomly allocated into two groups. Group RO patients received 10ml infiltration of 0.2% ropivacaine to skin 2minutes prior to skin incision and group RD patients received 10ml infiltration of 0.2% ropivacaine with

1microgram/kg of dexmedetomidine via the same technique. All the patients were given general anaesthesia. Induction was done with 5mg/kg of thiopental iv and 0.6mg/kg of rocuronium iv. The trachea was intubated and maintained with O2 : N2O – 1.5 : 1.5 and sevoflurane 2 – 3%. All the surgeries were carried out by the same surgeon. The primary outcomes were the VAS pain scores, amount of fentanyl consumption and the frequency to push button (FPB) in the PCA system. The total amount of fentanyl consumption and the frequency to push button was significantly decreased in the group RD as compared group RO.

Kenan Kaygusuz, MD et al[(18)] conducted a study on 2012. They studied “The effect of adding dexmedetomidine to levobupivacaine in axillary brachial plexus block”. Sixty four patients with ASA I & II physical status posted for forearm and hand surgeries with axillary plexus block were included. They were randomly divided into two groups (n = 32) – group L and group D. Group L patients received 39ml of 0.5% levobupivacaine with 1ml of isotonic saline and Group D patients received 39ml of 0.5% levobupivacaine with 1ml of dexmedetomidine 1microgram/kg. Hemodynamic parameters like mean arterial blood pressure, heart rate and peripheral oxygen saturation was

noted throughout the procedure. The onset times of sensory and motor block was significantly decreased in the group D compared to group L. The duration of sensory and motor block and the time to first analgesic use was significantly increased in group D compared to group L. The total need for analgesics was also significantly decreased in the group D.

Ahmed Sobhy Basuni[19] performed a study in 2013 regarding the effect of addition of dexmedetomidine to low dose levobupivacaine spinal anaesthesia for knee arthroscopy surgeries. The study design was a prospective randomised controlled double blind study. Sixty adult patients of both the sexes under the ASA physical status I and II posted for knee arthroscopy surgeries were included in the study. Patients were randomised into two groups using computer generated random numbers.

- Group D – received 5mg of 0.5% levobupivacaine and 3microgram of dexmedetomidine intrathecally.
- Group F – received 5mg of levobupivacaine and 10microgram of fentanyl intrathecally.

Prior to subarachnoid block, monitors like electrocardiography, pulse oximetry and non-invasive blood pressure were connected and baseline values recorded. The patients were pre-loaded with 250ml of

Ringer Lactate solution. Subarachnoid block was performed with 25gauge Quincke's needle at the L3-4 space in the sitting position with the bevel facing up. The onset of sensory block, the time to highest sensory block and the time to highest Bromage score were faster in group D. The duration of sensory block and the intensity of motor block were superior in group D as compared to group F.

Ji Eun Kim and Na Young Kim[20] published a study in 2013 regarding the effects of addition of dexmedetomidine to low dose bupivacaine spinal anaesthesia in elderly patients undergoing transurethral prostatectomy. Fifty four elderly patients posted for transurethral prostatectomy was included in the study. Using computer generated random numbers they were divided into two groups.

- Group S – received 6mg of 0.5% bupivacaine with 0.3ml of dexmedetomidine (3 microgram)
- Group D – received 6mg of 0.5% bupivacaine with 0.3ml of preservative free normal saline.

Prior to spinal anaesthesia, the patients were hydrated with 300 ml of 0.9% normal saline. During the surgical procedure, the fluid was minimally infused to avoid overloading. Spinal block was performed at

L3 – L4 space with a 25gauge Quincke's needle with the patient in the lateral decubitus position. The end points of the study were time to 2 segment regression of the sensory dermatomes from the peak sensory level. The values of Mean arterial pressure and Heart rate were not significantly different between the two groups despite the patients being elderly. The time to reach the peak sympathetic and sensory block was significantly shorter in group D. The time to 2 segment regression of sensory dermatomes was significantly longer in group D and the requirement of post-operative analgesics were significantly shorter in group D as compared to group S.

B. Maharani and M. Sathya Prakash[21] conducted a study in 2013 comparing the addition of dexmedetomidine or buprenorphine as adjuvants to spinal anaesthesia. Sixty adult patients posted for infra-umbilical and lower limb surgeries under the ASA physical status I and II were included in the study. They were randomly divided into two groups

- Group A – received 15mg of 0.5% bupivacaine plus 10microgram of dexmedetomidine intrathecally.

- Group B – received 15mg of 0.5% bupivacaine plus 60microgram of buprenorphine intrathecally.

Addition of dexmedetomidine to bupivacaine significantly decreased the onset time of sensory blockade, prolonged the duration of sensory and motor blockade and the time for first analgesic requirement.

Waleed A. Almarakbi and Abdullah M. Kaki[22] conducted a study in 2014 regarding the effect of addition of dexmedetomidine to bupivacaine in transverse abdominis plexus block in prolonging post-operative pain relief. After approval from Institutional ethical committee and obtaining informed consent, 50 patients over the age of 18years under the ASA physical status I and II scheduled for abdominal hysterectomy were included in the study. During the pre-operative assessment, the Visual Analog Scale for pain assessment with 0 meaning no pain and 10 meaning worst pain was explained to the patient. Inj. Midazolam 0.03mg/kg was given 15 minutes prior to induction of anaesthesia. General anaesthesia was standardised in both the patient groups. Fentanyl 2microgram/kg, propofol 2mg/kg and cis-atracurium 0.1mg/kg was given for tracheal intubation. Anaesthesia was maintained with O2/Air/Sevoflurane mixture. Randomisation was done

using computer generated software into two equal groups. Transverse abdominis block was performed in both the groups.

- Group B – received 20ml of 0.25% bupivacaine plus 2ml of normal saline

- Group BD – received 20ml of 0.25% bupivacaine plus 2ml of normal saline with 0.5 microgram/kg of dexmedetomidine

The post-operative requirement of morphine and time to first analgesic requirement was noted. The total dose of morphine required was significantly lower in group BD. The time to first analgesic requirement was also significantly longer in group BD as compared to group B.

Saumya Biswas and Ratan Kumar Das[23] conducted a study in 2014 regarding the effect of dexmedetomidine as an adjuvant added to levobupivacaine in supraclavicular brachial plexus block. After Ethical committee approval and obtaining informed consent, sixty patients under the ASA physical status I and II scheduled for elective forearm and hand surgeries were included in the study. They were divided into two groups of thirty patients each using computer generated random numbers.

- Group L – received 35ml of levobupivacaine 0.5% and 1ml of normal saline

- Group LD – received 35ml of levobupivacaine 0.5% and 1ml of dexmedetomidine (100microgram)

On arrival to the operating room, baseline monitors of BP, SpO2 and HR were recorded. An intra-venous line was started with an 18gauge cannula and infusion of Ringer lactate started. The systolic and diastolic pressures were significantly lower in group LD than in group L. Sensory and motor block duration were significantly longer in group LD as compared to group L.

Yu Zhang et al[24] performed a study in 2014 regarding "perineural administration of dexmedetomidine in combination with ropivacaine prolongs axillary brachial plexus block". The study was a prospective, randomized controlled double blind trial. Forty five adult patients in the age group 25 – 60 years of age posted for elective forearm and hand surgeries were included in the study after obtaining Ethical committee approval and informed consent. Patients were divided into three groups –

- Group R – received 40ml of 0.33% ropivacaine along with 1ml of normal saline.
- Group DR1 – received 40ml of 0.33% ropivacaine along with 1ml of dexmedetomidine (50microgram)
- Group DR2 – received 40ml of 0.33% ropivacaine along with 1ml of dexmedetomidine (100microgram)

Standard monitoring was attached and a 20guage peripheral cannula was inserted in the contra-lateral arm. Using nerve stimulator technique, three point axillary plexus block was performed – radial or ulnar, median and musculocutaneous nerve. Thirteen millilitres of local anaesthetic was injected in the site of radial or ulnar and median nerve respectively. Nine millilitres of solution was injected in the musculo-cutaneous nerve distribution and the remaining six millilitre was injected in the skin subcutaneously to block the intercosto brachial nerve. The onset time of sensory and motor blockade was not significantly different in the three groups. The duration of sensory blockade was significantly increased in the group DR2 than the other groups. The incidence of side effects was also higher in the DR2 group (hypotension, bradycardia). Bradycardia was treated with atropine injection.

Samy E. Hanoura et al published their study in 2015. They conducted the study “dexmedetomidine improves the outcome of a bupivacaine brachial plexus axillary block: a prospective comparative study”. After obtaining informed consent, patients in the age group 23 – 56 years under the ASA physical status I & II posted for elective orthopaedic upper limb surgeries were included in the study. The patients were divided into equal groups –

- Group D – received axillary block with 40ml of 0.25% bupivacaine and 1ml of dexmedetomidine (100microgram)
- Group B – received axillary block with 40ml of 0.25% bupivacaine and 1ml of normal saline.

The axillary block was performed using nerve stimulator technique. The parameters like block performance time, latency time, duration of sensory and motor block and duration of analgesia were noted. The severity of pain assessed using the Visual Analog Scale (VAS) over a 10 point scale. The latency time was statistically shorter in the group D compared to group B. The duration of motor block was not statistically different and the duration of sensory block was significantly longer in the group D compared to group B.

Vinod Hosalli and Anilkumar Ganeshnavar conducted a study in 2015. They compared the addition of dexmedetomidine or clonidine to levobupivacaine in ultrasound guided axillary plexus block. After obtaining informed consent, sixty ASA physical status I & II patients undergoing bony orthopaedic procedures in the upper limb were selected for the study. The patients were randomly divided into 2 groups by slips in the box technique.

- Group C – received 36ml of 0.5% levobupivacaine and 1microgram/kg of clonidine.
- Group D – received 36ml of 0.5% levobupiacaine and 1microgram/kg of dexmedetomidine.

The patients were taken to the operating room and the baseline values of heart rate, blood pressure and Electrocardiography were recorded. The axillary block was performed with the ultrasound system. 6ml of local anaesthetic was injected to anaesthetise the musculocutaneous nerve. The radial, ulnar and median nerve were anaesthetised with 10ml of local anaesthetic each. There was no significant difference in the onset times of sensory and motor block in the two groups. The duration of sensory and motor block was

significantly longer in the group D compared to group C. Pulse rate, Systolic and diastolic pressure was significantly lower in group D compared to group C.

Don Sebastian and Ravi M[25] conducted a study in 2015 comparing the addition of dexmedetomidine or clonidine to ropivacaine in supraclavicular plexus block. The study was a randomised double blind controlled study. After obtaining Ethical committee approval and informed consent, sixty adult patients in the age group 18 – 55 years under the ASA physical status I and II were included in the study. They were randomly divided into two groups using computer generated numbers.

- Group C – received 29ml of 0.5% ropivacaine with 1ml of clonidine (50microgram)
- Group D – received 29ml of 0.5% ropivacaine with 1ml of dexmedetomidine (50microgram)

On arrival to the operating room, intravenous line started with an 18gauge intra-venous cannula and Ringer lactate infusion was started. Basic monitors of heart rate, blood pressure and haemoglobin oxygen saturation were connected. The supra-clavicular block performed with

the classical nerve stimulator technique. The current stimulation was started with a stimulation intensity of 2.0mA and pulse width of 100ms. Once the desired response was attained, current was gradually reduced till 0.4mA and the drug solution injected if the desired response persisted. There were no significant difference in the general characteristics of the patient like age, weight or height. The onset of sensory block and motor block was significantly shorter in group D compared to group C. The duration of sensory block and motor block was significantly longer in group D.

Hem Anand Nayagam, N Ratan Singh and H Shanti Singh[26] conducted a study in 2015 regarding the effect of addition of fentanyl and dexmedetomidine to low dose bupivacaine heavy in spinal anaesthesia. The study was a prospective randomised double blind controlled study. 150 patients were selected after approval from the Institutional Ethical committee and obtaining informed consent. They were randomly divided into two groups by computer generated random numbers.

- Group F – received 0.8ml of 0.5% bupivacaine heavy with 0.5ml of 25microgram fentanyl and 0.3ml of normal saline intrathecally.

- Group D – received 0.8ml of 0.5% bupivacaine heavy with 0.05ml of 5microgram dexmedetomidine and 0.75ml of normal saline intrathecally.

The time to reach the T10 sensory segment was not significantly different between the two groups. The time to reach peak sensory level was significantly faster in group D. The total analgesic requirements was also significantly lesser in group D.

Ayşe Ülgey et al conducted a study regarding the “The Analgesic Effects of Incisional Levobupivacaine with Dexmedetomidine after Total Abdominal Hysterectomy”. Fifty adult patients posted for elective hysterectomy under the ASA physical status I and II were included in the study. The patients after obtaining informed consent were divided into two equal groups – group L received 40ml of 0.25% levobupivacaine infiltration of the surgical area and group LD received 40ml of 0.25% levobupivacaine and 2microgram/kg dexmedetomedine 5minutes prior to surgical incision. The rescue analgesic requirement was significantly lower in group LD. The Visual Analog Scale at 0, 2 and 4 hours were significantly less in group LD as compared to group L.

SS Harsoor et al[27] conducted a study in 2015 regarding the "Effect of supplementation of low dose intravenous dexmedetomidine on characteristics of spinal anaesthesia with hyperbaric bupivacaine". After obtaining informed consent, fifty adult patients posted for elective lower abdominal and lower limb surgeries were included in the study. They were randomly divided into two groups – group D received 0.5microgram/kg of dexmedetomidine intravenous bolus given 10minute prior to subarachnoid block and then 0.5microgram/kg/hour till the duration of surgery and group C received similar volumes of normal saline. They concluded that administration of dexmedetomidine intravenously fastens the onset of sensory blockade and prolongs the duration of sensory and motor blockade.

Alpha 2 Adrenergic Receptors

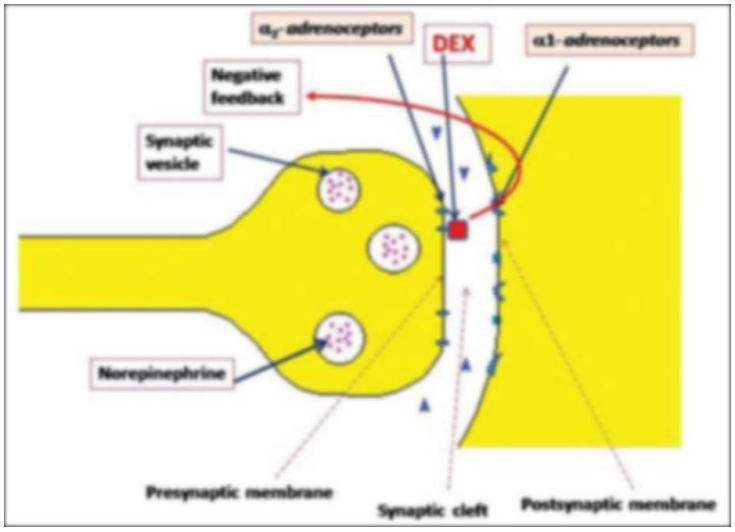
ALPHA – 2 ADRENORECEPTOR

The primary sympathetic neurotransmitters nor-adrenaline and adrenaline exert their central and peripheral actions through specialised receptors called adrenergic receptors.

Adrenergic receptors are present in nearly all the peripheral tissues and in the central nervous system neurons. Three types of adrenergic receptors are present – alpha 1, alpha 2 and alpha 3[28]. They belong to the cell surface G-protein coupled type of receptors.

Alpha 2 receptors are subdivided into three subtypes alpha 2A, alpha 2B and alpha 2C[29].

1. Alpha 2A – gene on chromosome 10 and involved in sedation and analgesia
2. Alpha 2B – gene on chromosome 4 and involved in hemodynamic effects
3. Alpha 2C – gene on chromosome 2



ALPHA – 2 AGONISTS

Since 1970s, alpha 2 adrenergic agonists have been used for the treatment of hypertension and for drug withdrawal. Alpha 2 agonists produce diverse responses like analgesia, anxiolysis, sedation and sympatholysis.

Recently the Food and Drug Administration approved the usage of two novel alpha 2 agonists – clonidine and dexmedetomidine for the usage in Intensive Care Unit sedation. Its use has now been investigated as an adjuvant to prolong the effects of epidural, spinal and peripheral nerve blocks.

MECHANISM OF ACTION:

1. Inhibits the adenylate cyclase enzyme[30] responsible for the production of 3,6 – cyclic adenosine monophosphate resulting in decreased availability of cyclic AMP. Cyclic AMP mediates phosphorylation of many of the intra-cellular target proteins. This results in hyper-polarisations of the neuronal cell membrane which results in decreased firing rate of excitable cells.

2. N-type voltage gated calcium channels are inhibited resulting in decreased entry of calcium ions which results in decreased catecholamine secretion.

3. Activates the alpha 2 adrenoreceptor in the pre-synaptic region resulting in decreased release of sympathetic neurotransmitters.

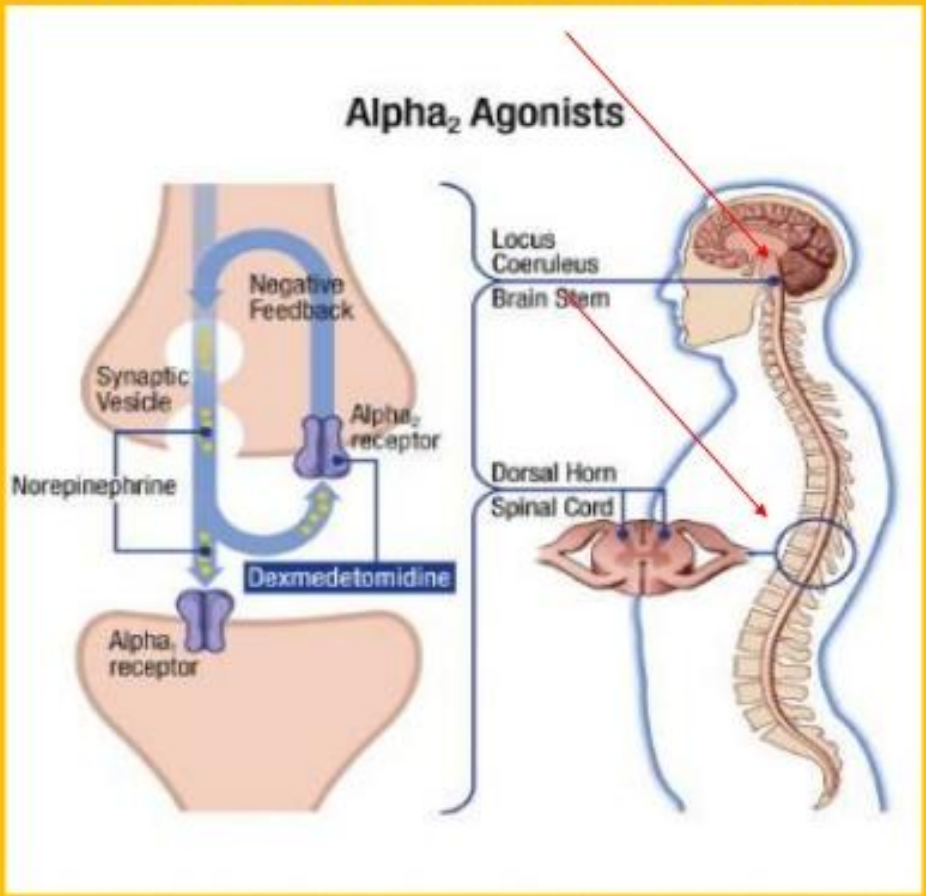
Dexmedetomidine produces all these effects by their multiple mechanisms of action and thereby avoiding multiple pharmacology and their combined side effects.

There is no clear mechanism by which the alpha 2 agonists produce analgesia. Possible mechanisms may be supra-spinal effect in the locus ceruleus and spinal effects in the substantia gelatinosa resulting in the production of analgesia.

On addition of dexmedetomidine, the excitable nerve cells can neither fire nor propagate the signal to the neighbouring cells resulting in analgesia by central, spinal and peripheral actions.

The ratio of alpha 2 : alpha 1 activity for dexmedetomidine is 1620 : 1 and 220 : 1 for clonidine. Hence dexmedetomidine is a more selective alpha 2 adrenergic agonist than clonidine.

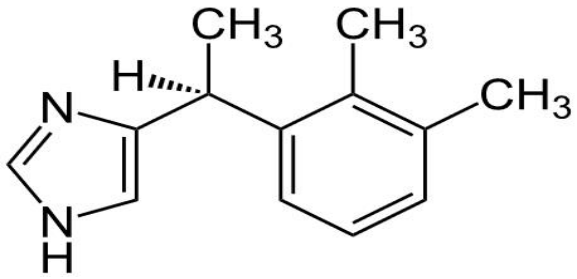
Alpha 2 Agonists Pathways



Pharmacology of Dexmedetomidine

PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine is the S-enantiomer of medetomidine[31], used widely in the veterinary practice. Chemically Dexmedetomidine is (S)-4-[1-(2,3-dimethylphenyl)ethyl]-3H-imidazole.



PHARMACOKINETICS

ABSORPTION:

Dexmedetomidine is inactive orally and the conventional route of administration is the intra-venous route. Dexmedetomidine has good bio-availability with nasal, intra-muscular, buccal, sublingual, neuraxial and intra-articular routes.

DISTRIBUTION:

Elimination half life	-	2-3 hours
Volume of distribution	-	118 litres

PROTEIN BINDING:

Dexmedetomidine is 95% protein bound to albumin. Protein bound fraction decreases with hepatic impairment. Dexmedetomidine does not displace phenytoin, propranolol, warfarin, digoxin and theophylline from plasma proteins.

METABOLISM:

Biotransformation occurs in the liver to inactive metabolites. Metabolism occurs by N-methyl glucuronidation in the liver and the glucuronide metabolites are excreted in the urine. Hence the dosage of dexmedetomidine must be decreased in patients with hepatic failure.

ADDITIONAL BENEFITS:

Dexmedetomidine has antisialagogue, antishivering and decongestant properties. Dexmedetomidine has minimal respiratory depression compared to other drugs with a better hemodynamic stability.

ADVERSE EFFECTS:

The most notable side effects of dexmedetomidine are bradycardia, hypotension, dry mouth, nausea and vomiting. Transient hypertension can be seen in large doses and sudden discontinuation can lead to a withdrawal syndrome with agitation, irritability and hypertensive crisis. It is classified as a Category C drug in pregnancy (Animal studies have observed risk but well controlled human studies not available).

PHARMACODYNAMICS

EFFECTS ON THE CARDIOVASCULAR SYSTEM:

Dexmedetomidine causes a dose dependent inhibition of the vasomotor centre leading to bradycardia and hypotension. Dexmedetomidine is not a myocardial depressant. On administration, a biphasic response in BP occurs – an initial hypertensive phase due to peripheral alpha 2B receptor activation and a subsequent hypotensive phase due to presynaptic alpha 2A receptor activation.

EFFECTS ON THE RESPIRATORY SYSTEM:

Dexmedetomidine has no significant effects on the respiratory system and respiratory depression does not occur.

EFFECTS ON THE CENTRAL NERVOUS SYSTEM:

Dexmedetomidine decreases cerebral excitation, decreases cerebral blood flow and cerebral metabolic oxygen demand thus maintaining the balance between cerebral oxygen demand and supply.

EFFECT ON GENITOURINARY SYSTEM:

Dexmedetomidine decreases renin release, increases glomerular filtration rate and increases sodium and water excretion by their action on the peripheral alpha 2 receptors.

CLINICAL USES

ATTENUATION OF STRESS RESPONSE:

Dexmedetomidine decreases the stress responses to tracheal intubation at a dose of 1 microgram / kg.

AS AN ANAESTHETIC ADJUVANT TO GENERAL ANAESTHESIA:

Dexmedetomidine decreases the anaesthetic requirements of all the volatile anaesthetics and opioid analgesics resulting in their decreased usage.

AS AN ADJUVANT TO NEURAXIAL ANAESTHESIA:

Dexmedetomidine at a dose of 5 microgram when added intrathecally to hyperbaric 0.5% bupivacaine can prolong the post-operative analgesia

AS AN ADJUVANT TO PERIPHERAL NERVE BLOCK:

Dexmedetomidine when added to bupivacaine at a dose of 0.5 microgram / kg can prolong the duration of analgesia

IN MONITORED ANAESTHESIA CARE:

Dexmedetomidine can be used for procedures like fibre-optic bronchoscopy, dental procedures and ophthalmic procedures with decreased opioid requirements and better post-operative outcome.

SEDATION IN INTENSIVE CARE UNIT:

The sedation produced by dexmedetomidine mimics normal sleep with minimal respiratory depression, decreased agitation and good hemodynamic stability.

Pharmacology of Bupivacaine

PHARMACOLOGY OF BUPIVACAINE

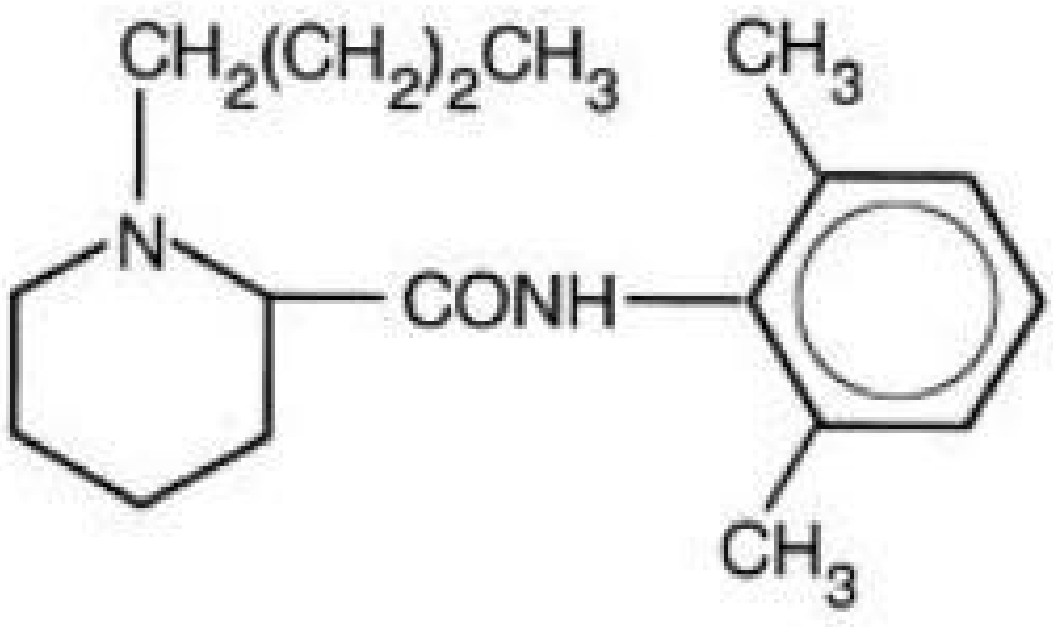
Bupivacaine is an amide group of long acting local anaesthetic. It was synthesized in 1957 by A. F. Ekenstam.

It is produced as a racemic mixture containing equal quantities of S and R enantiomers. It is supplied as a hydrochloride salt for clinical usage.

CHEMICAL NAME:

(2S)-1-Butyl-N-(2,6-dimethylphenyl)-piperidinecarboxamide

CHEMICAL STRUCTURE:



PHYSIO-CHEMICAL PROPERTIES:

Molecular weight	-	290 gm/mol
pKa	-	8.1
Solubility in water	-	1 in 25
Solubility in alcohol	-	1 in 8
Octanol/water partition coefficient	-	high
Lipid solubility	-	28
Plasma protein binding	-	95%

MECHANISM OF ACTION:

Local anaesthetics produce blockade of sodium channels resulting in decreased sodium entry into the cells thereby preventing the depolarisation of the cells. Thus the nerve signals and the action potential cannot be propagated.

PHARMACOKINETICS:

Bupivacaine is rapidly absorbed from the injection site. The rate of rise of plasma concentration and the peak plasma concentration attained depends on the route of administration. Inter-individual variability occurs in the time taken to attain the peak plasma concentration and can vary from 5 – 30 minutes after the drug administration.

Steady state volume of distribution	-	70 litres
Clearance	-	0.48 L/min
Alpha t ½	-	2.8 min
Beta t ½	-	28 min
Gamma t ½	-	3.5 hours

METABOLISM:

Bupivacaine can undergo any one of the following metabolic pathways – aromatic hydroxylation, N-methyl dealkylation, amide hydrolysis and conjugation. The metabolites of bupivacaine are excreted in the liver and hence renal disease does not affect the metabolism of bupivacaine. The fraction of drug excreted unchanged in urine is less than 10 %.

ONSET OF ACTION IN NERVE BLOCKS	-	15 – 20minutes
DURATION OF ACTION IN NERVE BLOCKS	-	8 – 10hours

The pKa or the dissociation constant is defined as the pH at which 50% of the drug is ionised and 50% of the drug is in the unionised form. A drug having a pKa close to the physiological pH will have more drug in the unionised form. The unionised fraction is the easily diffusible

form of the drug. Hence a drug having more unionised form of drug at physiological pH will be faster acting.

Bupivacaine has a clinical profile different from that of lignocaine. It has a pKa of 8.1 and has slower onset of action compared to lignocaine. But it is 4 times more potent than lignocaine and the sensory block produced by bupivacaine is more marked.

CLINICAL USES:

- Central neuraxial anaesthesia
- Peripheral nerve blocks
- Infiltration anaesthesia

PREPARATIONS:

- 0.25%, 0.5% Solutions available in 10 and 20ml vials.
- 5mg/ml of 0.5% bupivacaine and 80mg of dextrose in 4ml ampules for intrathecal injection.

CONTRAINDICATIONS:

- History of hypersensitivity to amide local anaesthetics
- Intravenous regional anaesthesia
- Paracervical block

Pharmacology of Lignocaine

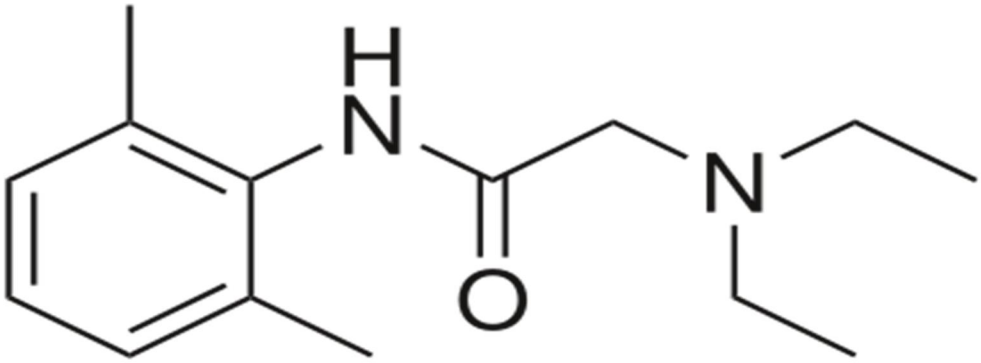
PHARMACOLOGY OF LIGNOCAINE

Lidocaine is an amide group of local anaesthetics. Lidocaine was discovered in 1943 by Nils Lofgren and then on is in routine usage.

CHEMICAL FORMULA:

2-(diethylamino)-N-(2, 6 dimethylphenyl)acetamide

CHEMICAL STRUCTURE:



PHYSIO-CHEMICAL PROPERTIES:

Molecular weight	-	234gm/mol
pKa	-	7.6
Lipid solubility	-	less than bupivacaine
Plasma protein binding	-	60-80%

MECHANISM OF ACTION:

Lidocaine acts in the similar way as all the local anaesthetics act by blocking the fast voltage gated sodium channels located in the neuronal cell membranes resulting in decreased action potential propagation.

PHARMACOKINETICS:

On entering the circulation, lidocaine is 60 – 80% plasma protein bound.

Volume of distribution	-	1-2L/min
Elimination half life	-	90-120mins

METABOLISM:

Lidocaine is principally metabolised by N-dealkylation in the liver via the CYP3A4 metabolic pathway. It is converted into mono-ethylglycinexylidide which is pharmacologically active and then to glycinexylidide which is inactive. Most of the metabolites are excreted in the urine.

ONSET OF ACTION	-	45-90seconds
DURATION OF ACTION	-	10-20minutes

The pKa of lidocaine is 7.6 and is close to the physiological pH and has more number of unionised drug at the site of action and hence has a faster onset of action compared to bupivacaine.

PREPARATIONS:

Intravenous – 2% and 1% solutions with or without adrenaline

Topical gel/patches – 5% lidocaine

Topical sprays – 10% lidocaine

CLINICAL USES:

- Central neuraxial anaesthesia
- Peripheral nerve blocks
- Infiltration anaesthesia
- Anaesthesia for airway procedures like Fibre Optic Bronchoscopy

Adverse effects of Local Anaesthetics

ADVERSE EFFECTS OF LOCAL ANAESTHETICS:

Adverse effects are due to excess plasma concentrations of the drug which can be due to over-dosage of the drug, unintentional intra-vascular injection of the drug or slower rate of degradation.

ALLERGIC REACTIONS:

The presence of rash, utricaria and laryngeal edema is highly suggestive of bupivacaine induced allergic reaction. It can be due to the preservative methylparaben used with the local anaesthetics. Cross sensitivity among the different groups of local anaesthetics has been reported.

CENTRAL NERVOUS SYSTEM REACTIONS:

Low plasma concentrations characteristically produces numbness of the tongue and the circumoral regions. As the plasma concentration increases the patient exhibits restlessness, slurred speech, skeletal muscle twitching and progresses to tonic-clonic seizures. Seizures are followed by CNS depression accompanied with hypotension.

CARDIO-VASCULAR SYSTEM REACTIONS:

Local anaesthetics can produce cardiac sodium channel blockade resulting in cardiac dysarrhythmias, atrio-ventricular block, ventricular tachycardia and ventricular fibrillation. After an accidental intravascular injection, the protein binding sites are fully saturated resulting in large amount of the unbound fraction of the drug available for binding to the conducting tissues of the heart. Pregnancy may increase the sensitivity of the cardiotoxic effects of local anaesthetics.

TREATMENT OF SYSTEMIC TOXICITY OF LOCAL ANAESTHETICS:

- The treatment is primarily supportive. Stop administration of the local anaesthetic injection immediately.
- Airway, breathing and circulation must be maintained.
- Avoid hypoxia, hypercapnia and acidosis.
- Benzodiazepines can be given for the prevention and treatment of seizure activity.

- Sympathomimetic agents like ephedrine and epinephrine, defibrillators and amiodarone must be available for cardiovascular complications.

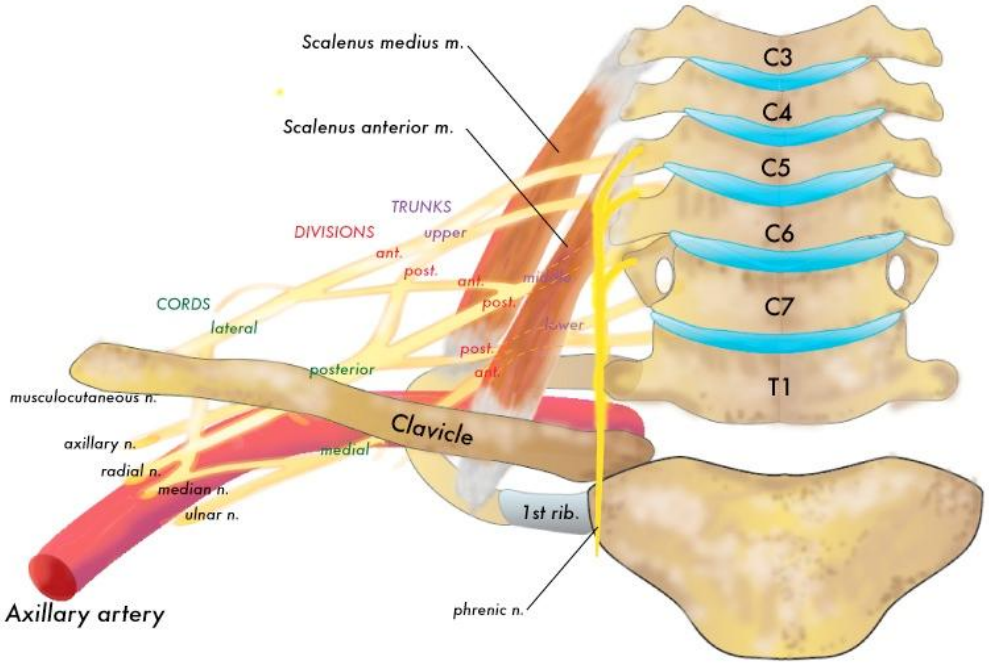
- Lipid emulsions by acting as a plasma sink. The bolus dose of lipid emulsion is 1.5ml/kg of 20% solution followed by an infusion of 0.25ml/kg/minute for 10minutes.

Anatomy of Brachial Plexus

ANATOMY OF BRACHIAL PLEXUS

FORMATION:

The brachial plexus is formed by the anterior rami of the nerve roots C5 – 8 and T1. There may be contributions above from C4 (pre-fixed) and below from T2 (post-fixed). These variations are associated with either the presence of a cervical rib or an anomalous 1st rib.



The roots of the plexus C5-C8 & T1 emerge from the inter-vertebral foramina. The nerve roots of C5, C6 and C7 pass behind the foramen transversarium of their respective cervical vertebra and then lies between the anterior and posterior tubercles of the respective transverse process. Then the nerve roots lies between the scalenus anterior and medius.

The roots of the C5 and C6 unite to form the upper trunk, C7 nerve root continues as the middle trunk and C8 and T1 unite to form the lower trunk. The brachial plexus lies between two sheaths of fibrous tissue formed form the posterior and anterior tubercles such that the local anaesthetic is injected in this sheath to produce a brachial plexus block.

The trunks then pass downward and laterally at the base of the posterior triangle and across the base of the first rib. Trunks divide into an anterior division and posterior division at the lateral border of the first rib.

The divisions then divide to form the three cords – lateral, medial and posterior. They are named in relation to the axillary artery. The lateral cord is formed by the anterior division of the upper and lower trunks. The medial cord is formed by the continuation of the anterior

division of the lower trunk. The posterior cord is formed by the posterior divisions of all the three trunks.

The branches of the lateral cord are:

- Lateral pectoral nerve (C5 – C7)
- Lateral root of median nerve(C5 – C7)
- Musculocutaneous nerve (C6, 7)

The branches of the medial cord are:

- Medial pectoral nerve (C8, T1)
- Medial cutaneous nerve of arm (C8, T1)
- Medial cutaneous nerve of forearm (C8, T1)
- Medial head of median nerve (C8, T1)
- Ulnar nerve (C7 – 8, T1)

The branches of the posterior cord are:

- Upper subscapular nerve (C5, C6)
- Nerve to lattismus dorsi
- Lower subscapular nerve (C5, C6)
- Axillary nerve (C5, C6)
- Radial nerve (C5 – 8, T1)

Brachial Plexus Blocks

BRACHIAL PLEXUS BLOCKS

The choice of the brachial plexus block depends on the desired site to be blocked and the risk of pneumothorax that accompanies the clavicular approaches.

INTERSCALENE BLOCK:

The brachial plexus is blocked at the level of upper trunk at the level of the sixth cervical vertebra. The space between the anterior and middle scalene muscles is the inter-scalene groove and the needle is inserted in the groove at right angles. Paraesthesia can be elicited or contractions in the deltoid or biceps brachialis muscle can be elicited by nerve stimulator technique.

Blockade of the cervical sympathetic chain or the phrenic nerve commonly occurs and are accompaniments of the block. Complications include accidental intra-vascular, epidural or subarachnoid injections. They are less effective for surgeries of the hand as the lower trunks are not blocked.

SUBCLAVIAN PERIVASCULAR APPROACH:

Subclavian perivascular approach targets the brachial plexus as it crosses over the 1st rib. Used to provide analgesia of the whole arm, can be occasionally deficient over the territory supplied by the ulnar nerve. The inter-scalene groove at the level of 6th cervical vertebra is identified and followed down to the neck and the needle entry is made just posterior to the subclavian artery pulsation.

Any periclavicular approach is associated with a risk of pneumothorax due to puncture of the pleura. In order to avoid the risk of pneumothorax, the 1st rib is used as a backstop.

AXILLARY BRACHIAL PLEXUS BLOCK:

Axillary plexus block targets the terminal branches of the brachial plexus mainly radial, ulnar and median nerves as they enter the axilla along with the axillary artery. The block is not useful for surgeries of the shoulder or upper arm as the nerves supplying them have already left the plexus at this point.

Axillary block is most appropriate for surgeries involving the elbow, forearm and hand. The axillary artery is palpated as high up in the axilla and fixed throughout the procedure. A single injection

technique or a multiple injection technique can be performed. In multiple injection technique, individual nerves are identified and 10ml of the local anaesthetic is injected around each nerve.

Materials and Methods

MATERIALS AND METHODS

"Prospective randomized controlled study evaluating dexmedetomidine as an adjuvant added to a local anaesthetic mixture of bupivacaine, lignocaine with adrenaline in axillary plexus block as compared to axillary plexus block with local anaesthetic mixture alone"

Was duly submitted before the Institutional Ethical Committee and after getting approval from the ethical committee the study was done on 60 patients.

STUDY DESIGN:

The study design was a Prospective Randomized Controlled study.

SAMPLE SIZE CALCULATION:

The study population consisted of 60 adult patients classified under the ASA 1 or 2 posted for upper limb surgical and orthopaedic procedures below the mid-humerus level.

INCLUSION CRITERIA:

- 18 – 60 years of age
- ASA physical status 1 or 2
- Patients undergoing elbow, forearm and hand surgeries
- Patients who gave valid informed consent

EXCLUSION CRITERIA:

- Lack of written informed consent
- Patients with diabetes and renal disease
- Pregnancy
- Patients with baseline heart rate less than 60bpm
- Patients on sedatives, opioids in the week prior to surgery

STUDY CENTRE:

- ESIC MEDICAL COLLEGE & PGIMSR, KK NAGAR over a period of ten months.

PRE-OPERATIVE ASSESSMENT:

All the patients were duly examined on the day before surgery and pre-operative assessment chart was checked. Any specific complaints of the patient like pain, anxiety must be sought out. The height and weight of the patient was measured. The airway assessment and the nutritional status of the patient was examined.

All the systems were examined in detail. All the pre-operative investigations like complete blood count, haematocrit, renal function tests, blood grouping and typing, chest radiography and

electrocardiography were evaluated and the risk was stratified based on American Society of Anaesthesiologist's grading.

INFORMED CONSENT:

All the patients satisfying the inclusion criteria were explained about the nature of this study and a written valid informed consent was obtained from all the patients.

PRE-MEDICATION:

All the patients were kept nil per oral for 8 hours and were pre-medicated with tablet alprazolam 0.5mg, tablet ranitidine 150mg and tablet metoclopramide 10mg on the night prior to surgery.

PREPARATION:

All the patients on arrival into the operating room, electrocardiography, pulse oximetry and non-invasive blood pressure monitors were connected and basal parameters recorded. An intra-venous access with 18 gauge cannula was started. Patients were randomly allocated into either of the two groups – group RD or group RL by slips in the box technique.

MATERIALS:

DRUGS

- IV Dexmedetomidne
- 0.5% bupivacaine
- 2% lignocaine with adrenaline
- Emergency resuscitation drugs

EQUIPMENTS

- Insulated stimulator needle
- Peripheral nerve stimulator
- ECG electrode
- Two 20ml(Luerlock) syringes
- One tuberculin syringe of 1ml
- Two stainless sterile bowls on each for iodine and spirit
- Sterile gauze pieces
- Equipment for administration of General Anaesthesia, if required.

MONITORS

- ELECTROCARDIOGRAPHY
- PULSE OXIMETRY
- NON – INVASIVE BLOOD PRESSURE

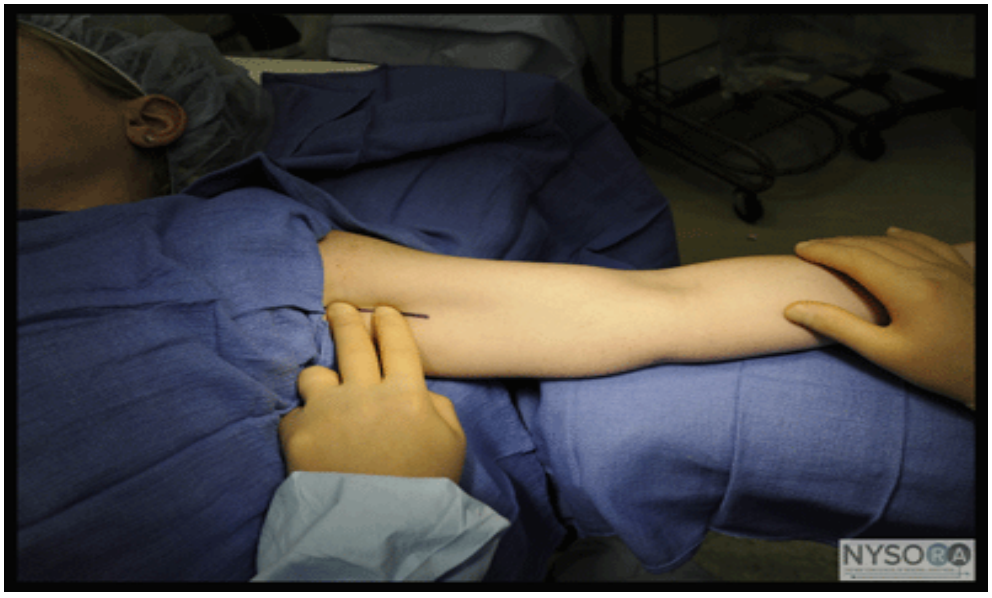
TECHNIQUE:

AXILLARY PLEXUS BLOCK (Nerve Stimulator technique)

The patient is placed in the supine position with the head facing opposite to the side being blocked. The arm is abducted to 90 degrees.

Axilla was aseptically cleaned and draped. The operator stood on the side to be blocked so that for a left side block the palpation was done with the left hand and the needle is manipulated with the right and the operator stands on the left side.

The axillary artery is palpated as high up in the axilla and is fixed firmly against the humerus between the index and middle finger of the palpating hand. The artery is fixed firmly throughout the procedure. Local infiltration of 1ml of 2% lignocaine given.



An insulated needle will be used to perform this technique. The needle is connected to nerve locator by the electrodes and is properly grounded with the help of ECG leads.

Group RD receives 20ml of 0.5% bupivacaine, 10ml of 2% lignocaine with adrenaline and 1ml of 50microgram dexmeditomidine.

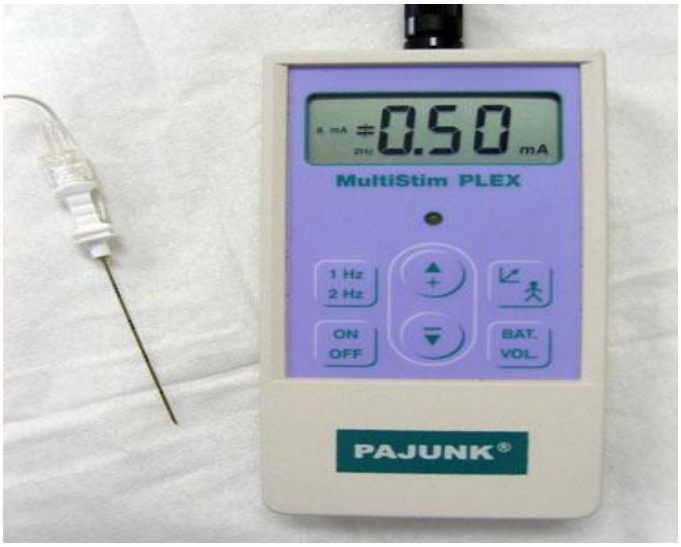
Group RL receives 20ml of 0.5% bupivacaine, 10ml of 2% lignocaine with adrenaline and 1ml of normal saline.

The stimulation is started with an intensity of 2.0 mA and a pulse width of 100 microseconds. Once the skin is entered, the needle is inserted to a depth of 1 -2 cm below the artery until a desired response – extension of wrist and fingers are obtained. 10 – 15ml of local anaesthetic is injected after negative aspiration of blood.

The needle is then withdrawn upto the skin and inserted above the artery until the response – flexion of the fingers and wrist is obtained. The needle is inserted till further until the median nerve twitch appears. 5 – 10 ml of local anaesthetic is injected after negative aspiration of blood

The needle is then inserted into the bulk of coracobrachialis till the biceps branchii twitch occurred and the remaining 5 – 10ml of local anaesthetic is injected.

As a goal we decided to elicit an isolated muscle twitch in the fingers either in flexion or extension for the desired response. Wrist flexion and extension of fingers is taken as the acceptable response and the current is gradually reduced till the twitch appears above 0.5 mA. 3 minute massage is done to facilitate an even drug distribution.



BLOCK EVALUATION:

Sensory block is assessed using pinprick method using the end of a 27guage needle at 0, 2, 5, 10, 15, 20 and 30 minutes in the median, ulnar, radial and musculocutaneous nerve distribution.

GRADES OF SENSORY BLOCKADE:

GRADE 0	-	Sharp pain felt
GRADE 1	-	Analgesia, dull sensation felt
GRADE 2	-	Anaesthesia, no sensation felt

Motor block is evaluated using Modified Bromage scale at 0, 10, 20 and 30 minutes.

GRADES OF MOTOR BLOCAKDE:

GRADE 0 - Normal motor function with full flexion and extension of elbow, wrist and fingers.

GRADE 1 – Decreased motor strength with ability to move the fingers only.

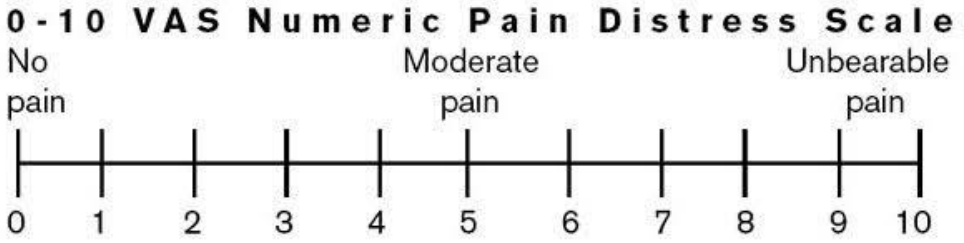
GRADE 2 – Complete motor block with inability to move the fingers.

ONSET TIME:

The onset time of sensory and motor blockade is defined as the time interval between the end of local anaesthetic injection and

- Loss of sensation to pin prick (sensory score – 1)
- Paresis (motor scale – 1) in the distribution of all peripheral nerves respectively.

Surgery is allowed to proceed when complete anaesthesia is achieved. Post-operative follow up is carried out in the recovery and post-operative ward for the Visual Analog score.



The patients were explained about Visual Analog Scale in the pre-operative assessment and the VAS scores were subsequently assessed in the post-operative period. VAS score of 4 indicated moderate pain and the rescue analgesic is administered at a VAS score of 4 or more.

VAS SCORE 0 – 2 - No pain

VAS SCORE 2 – 4 - Mild pain

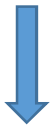
VAS SCORE 4 – 6 - Moderate pain

VAS SCORE 6 – 8 - Severe pain

VAS SCORE 8 – 10 - Unbearable pain

PATIENT FLOW CHART

Patients pre-operatively assessed and
posted for elective upper limb surgeries



Informed written consent obtained

Patient taken to the operating room

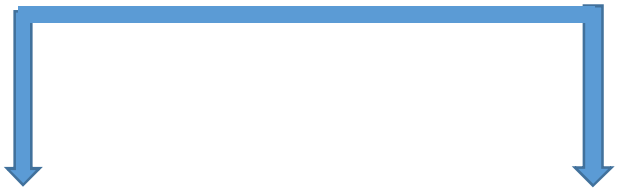
WHO checklist carried out

Groups randomized by Slips in the box technique

Pulse Oximetry, Electrocardiography,

Non-invasive blood pressure monitors connected

Axillary block performed using the nerve stimulator technique



Group RD :	**Group RL :**
• Bupivacaine 0.5% (20ml) • Lidocaine with adrenaline 2%(10ml) • Dexmedetomidine 50mcg (1ml)	• Bupivacaine 0.5% (20ml) • Lidocaine with adrenaline 2%(10ml) • Normal saline (1ml)

Block assessment :

Sensory block - Pin prick method

Motor block - Modified Bromage scale

Post – operative follow up - Duration of analgesia

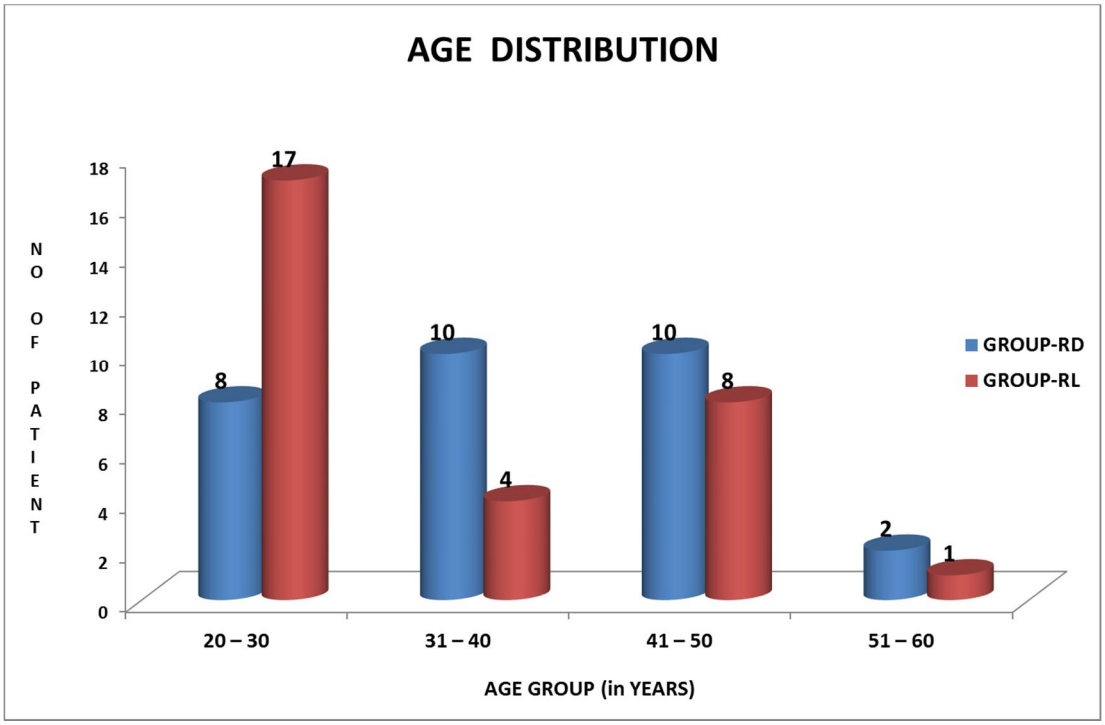
Results and Statistics

RESULTS AND STATISTICS

Sixty patients were included in the study and they were randomised by slips in the box technique into two groups – 30 patients belonged to the study group and 30 patients belonged to the control group. The demographical characteristics of the two groups were studied and found no significant difference between the two groups in terms of age,@ body mass index.

TABLE-1 : AGE DISTRIBUTION

Age Group	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
20 – 30	8	26.67	17	56.67
31 – 40	10	33.33	4	13.33
41 – 50	10	33.33	8	26.67
51 – 60	2	6.67	1	3.33
TOTAL	30	100	30	100
Chi square value	6.37			
p-value	0.10			
Significant	Not Significant			



GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

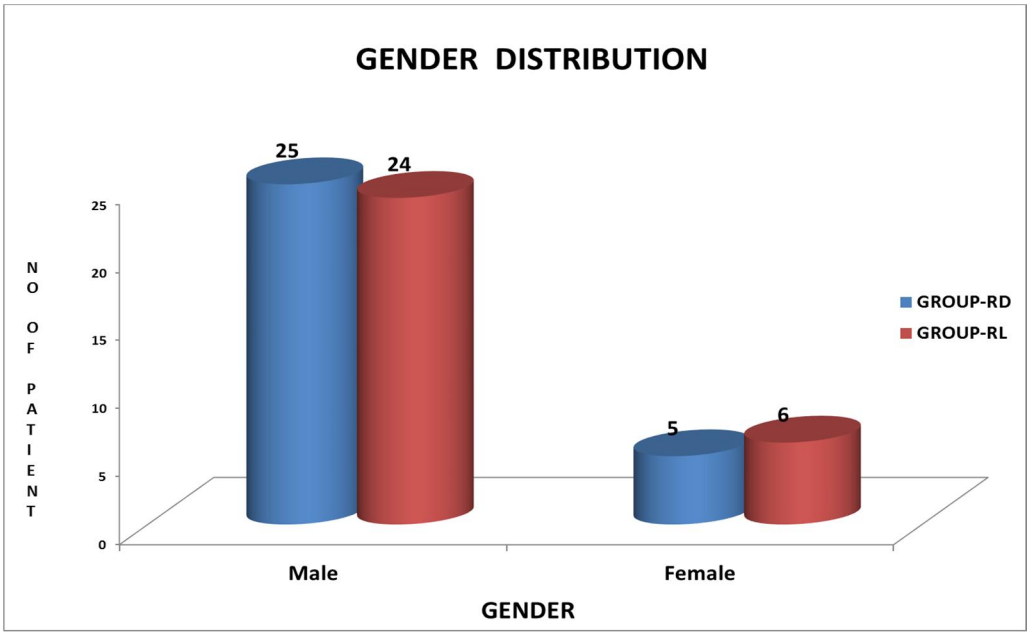
Both the groups are similar in distribution in term of age.

Mean Age (in Years)

Group	**Mean**	**Standard Deviation**
GROUP-RD	37.60	10.07
GROUP-RL	33.13	9.99
t-value	1.73	
p-value	0.09	
Significant	**Not Significant**	

TABLE-2 : SEX DISTRIBUTION

Sex	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
Male	25	83.33	24	80.00
Female	5	16.67	6	2.00
TOTAL	30	100	30	100
Chi square value	0.11			
p-value	0.74			
Significant	Not Significant			



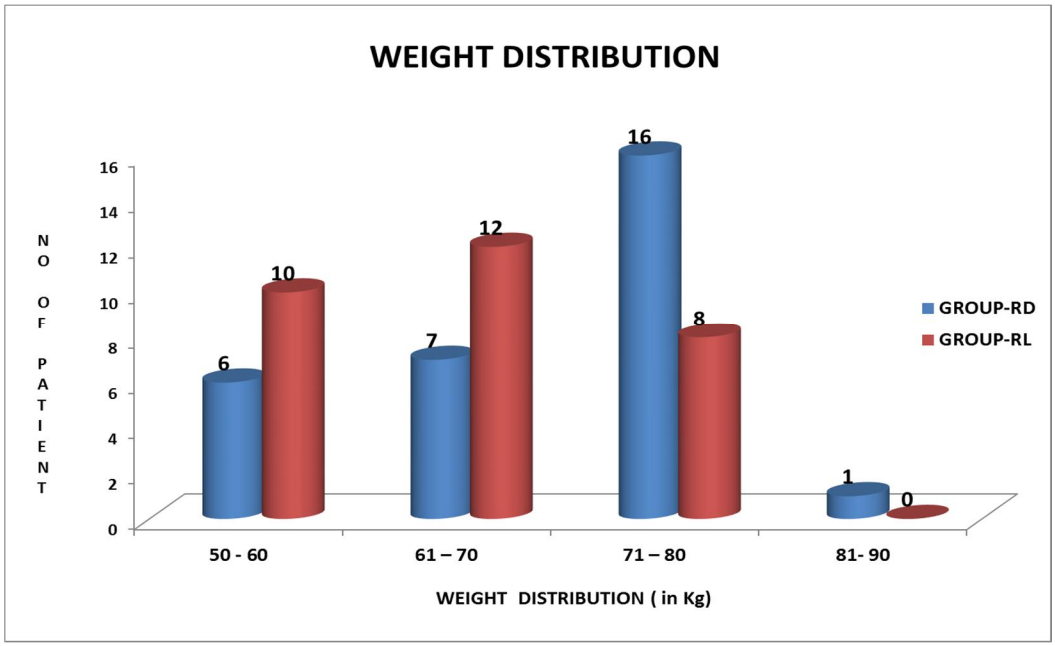
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

No statistically significant difference in sex distribution between the groups.

TABLE-3 : Weight Distribution

Weight in kgs	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
50 – 60	6	20.00	10	33.33
61 – 70	7	23.33	12	40.00
71 – 80	16	53.33	8	26.67
81- 90	1	3.33	0	0.00
TOTAL	30	100	30	100
Chi square value	5.98			
p-value	0.11			
Significant	Not Significant			



GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

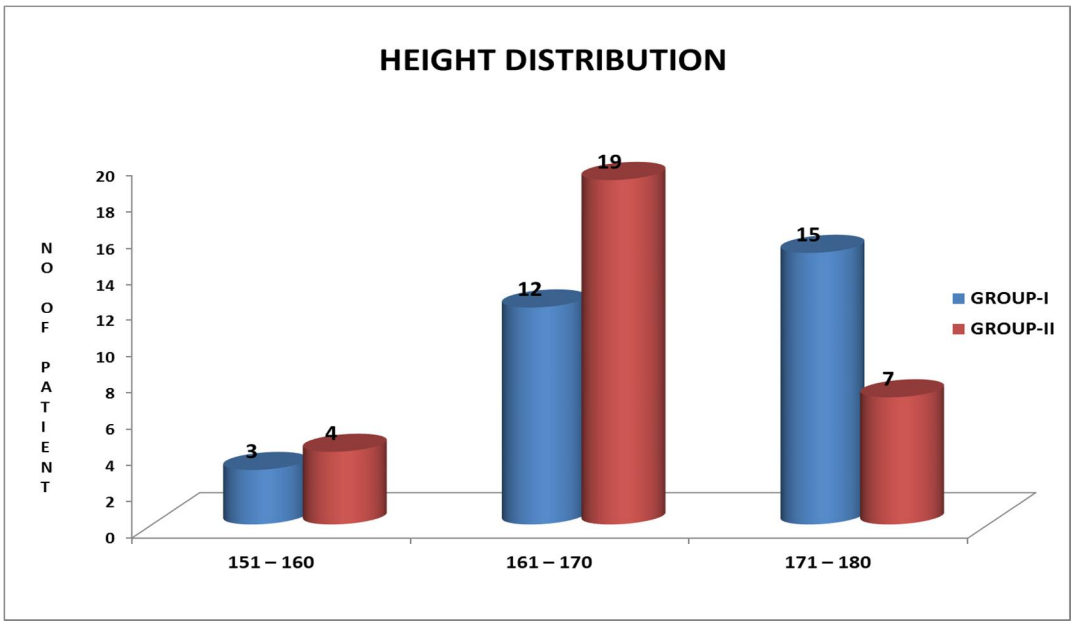
Mean Weight (Kg)

Group	Mean	Standard Deviation
GROUP-RD	70.60	9.18
GROUP-RL	67.47	8.22
t-value	1.39	
p-value	0.77	
Significant	**Not Significant**	

The mean weight distribution of the two groups are similar.

TABLE-4 : Height Distribution

Height in cms	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
151 – 160	3	10.00	4	13.33
161 – 170	12	40.00	19	63.34
171 – 180	15	50.00	7	23.33
TOTAL	30	100	30	100
Chi square value	4.63			
p-value	0.10			
Significant	Not Significant			



GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

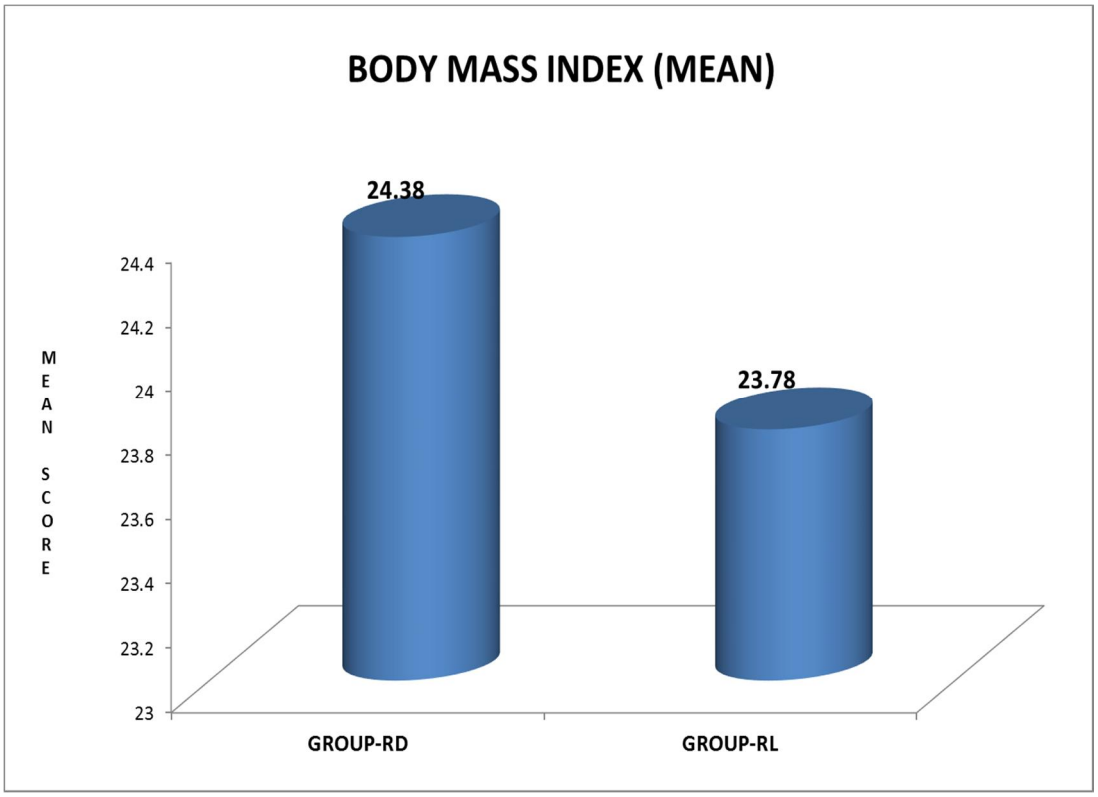
Mean Height (cm)

Group	Mean	Standard Deviation
GROUP-RD	169.97	5.86
GROUP-RL	168.20	5.02
t-value	1.25	
p-value	0.22	
Significant	**Not Significant**	

The mean height distribution of the two groups are similar.

TABLE-5 : Body Mass Index (Mean)

Group	Mean	Standard Deviation
GROUP-RD	24.38	2.60
GROUP-RL	23.78	2.09
t-value	0.98	
p-value	0.33	
Significant	**Not Significant**	



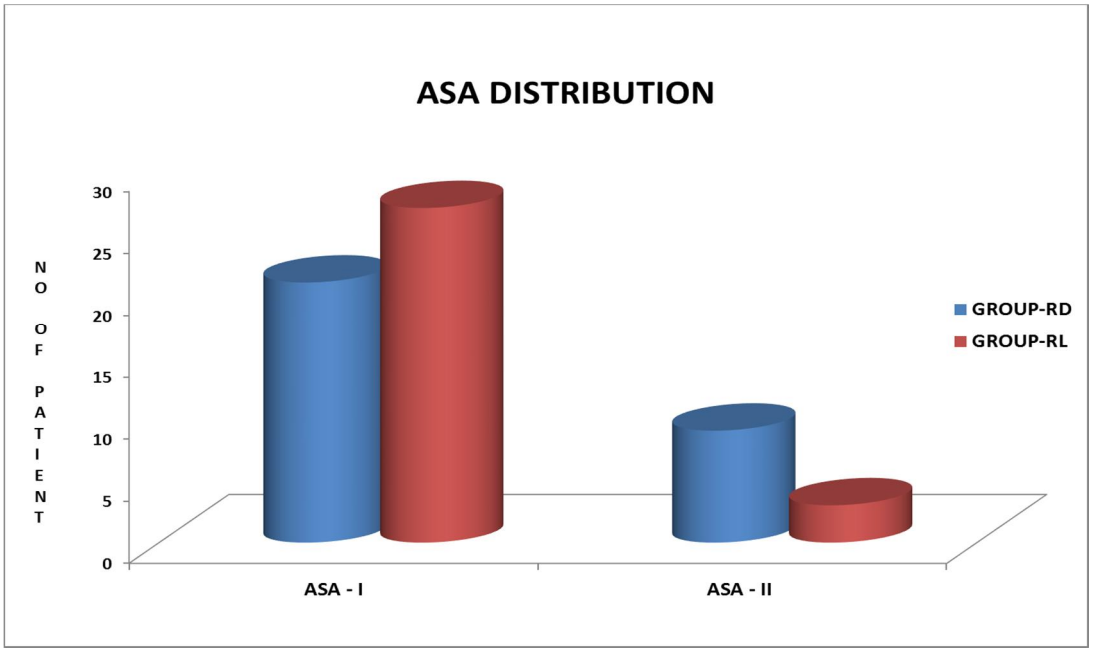
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The BMI of both the groups were similar in distribution.

TABLE-6 : ASA Distribution

ASA	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
I	21	70.00	27	90.00
II	9	30.00	3	10.00
TOTAL	30	100	30	100
Chi square value	3.75			
p-value	0.05			
Significant	Significant			

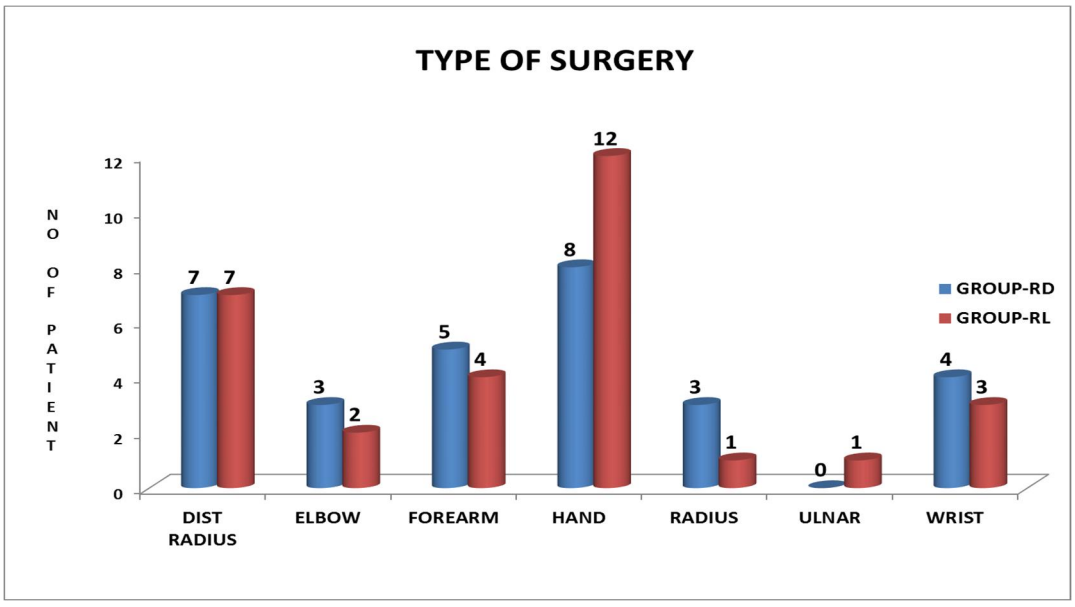


GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

TABLE-7 : Type of Surgery

Surgery	GROUP-RD		GROUP-RL	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
DIST RADIUS	7	23.33	7	23.33
ELBOW	3	10.00	2	6.68
FOREARM	5	16.67	4	13.33
HAND	8	26.67	12	40.00
RADIUS	3	10.00	1	3.33
ULNAR	0	0.00	1	3.33
WRIST	4	13.33	3	10.00
TOTAL	30	100	30	100
Chi square value	3.25			
p-value	0.78			
Significant	Not Significant			



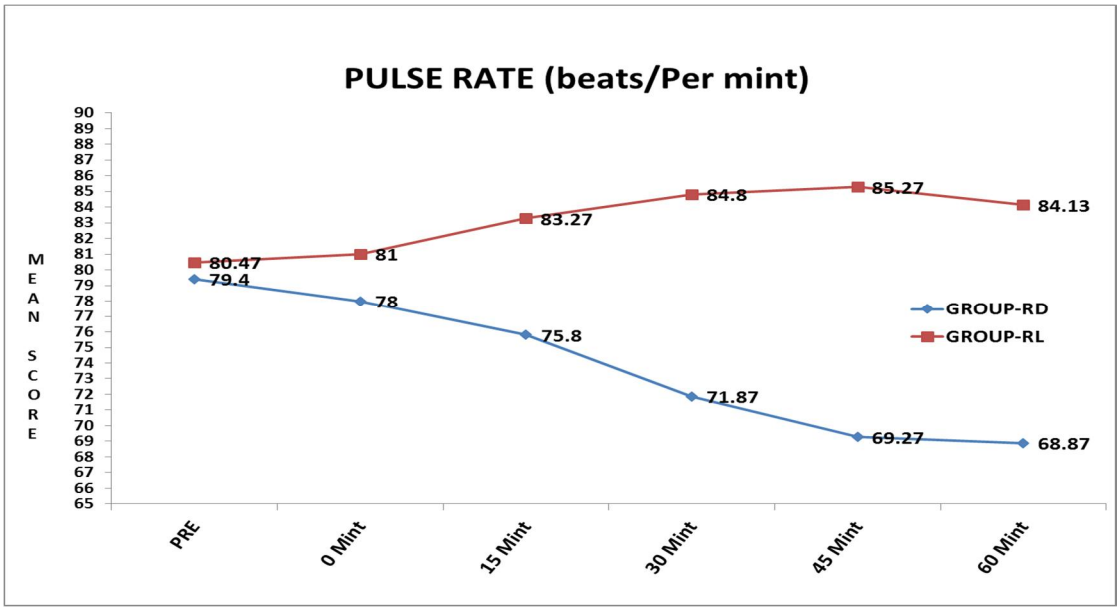
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

Higher percentages of surgeries were done in the hand and distal radius in both the groups.

TABLE-8 : PULSE RATE

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
PRE	79.40	7.81	80.47	8.80	0.50	0.62	NS
0 Mint	78.00	6.87	81.00	7.53	1.61	0.11	NS
15 Mint	75.80	7.90	83.27	7.02	3.87	0.000	Significant
30 Mint	71.87	8.71	84.80	6.72	6.44	0.000	Significant
45 Mint	69.27	7.82	85.27	7.23	8.23	0.000	Significant
60 Mint	68.87	7.98	84.13	6.45	8.15	0.000	Significant



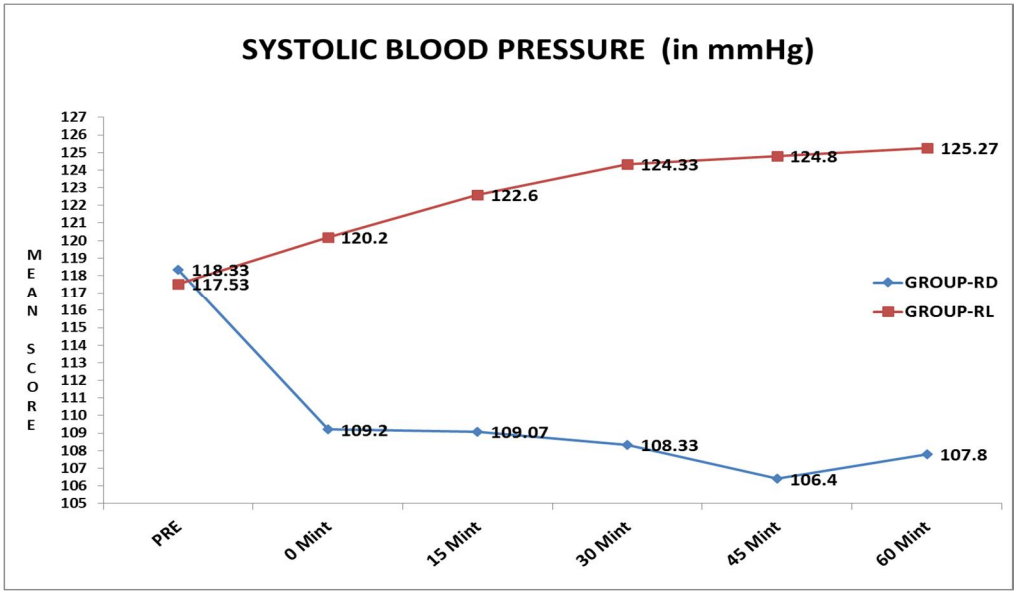
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The pulse rate showed statistically significant reduction in the dexmedetomidine group starting at 15th minute but without requiring any anti-cholinergics.

TABLE-9 : SYSTOLIC BLOOD PRESSURE

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
PRE	118.33	12.69	117.53	12.61	0.25	0.81	NS
0 Mint	109.20	20.56	120.20	11.29	2.57	0.01	Significant
15 Mint	109.07	7.08	122.60	11.68	5.43	0.000	Significant
30 Mint	108.33	8.52	124.33	10.82	6.37	0.000	Significant
45 Mint	106.40	6.90	124.80	9.58	8.54	0.000	Significant
60 Mint	107.80	7.58	125.27	8.97	8.15	0.00	Significant

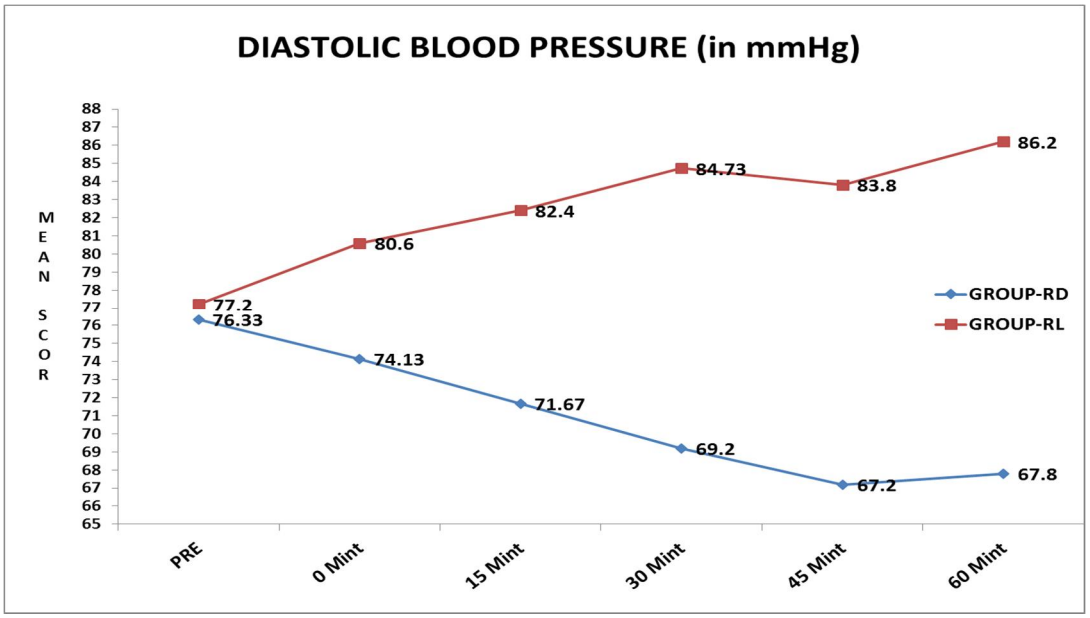


GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

TABLE-10 : DIASTOLIC BLOOD PRESSURE

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
PRE	76.33	10.98	77.20	12.01	0.29	0.77	NS
0 Mint	74.13	8.53	80.60	9.12	2.84	0.01	Significant
15 Mint	71.67	8.98	82.40	10.14	4.34	0.000	Significant
30 Mint	69.20	8.95	84.73	10.29	6.24	0.000	Significant
45 Mint	67.20	6.49	83.80	7.80	8.96	0.000	Significant
60 Mint	67.80	7.01	86.20	8.23	9.32	0.000	Significant

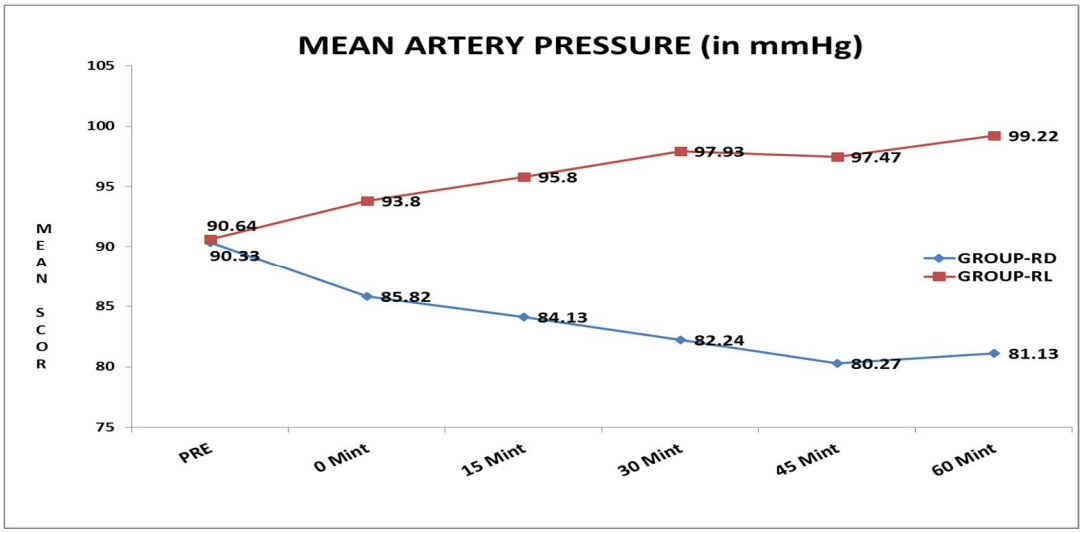


GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

TABLE-11 : MEAN ARTERY PRESSURE

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
PRE	90.33	11.39	90.64	12.06	0.10	0.92	NS
0 Mint	85.82	10.64	93.80	9.61	3.05	0.003	Significant
15 Mint	84.13	7.82	95.80	10.30	4.94	0.000	Significant
30 Mint	82.24	8.55	97.93	10.15	6.48	0.000	Significant
45 Mint	80.27	6.39	97.47	8.16	9.09	0.000	Significant
60 Mint	81.13	6.94	99.22	8.10	9.29	0.000	Significant



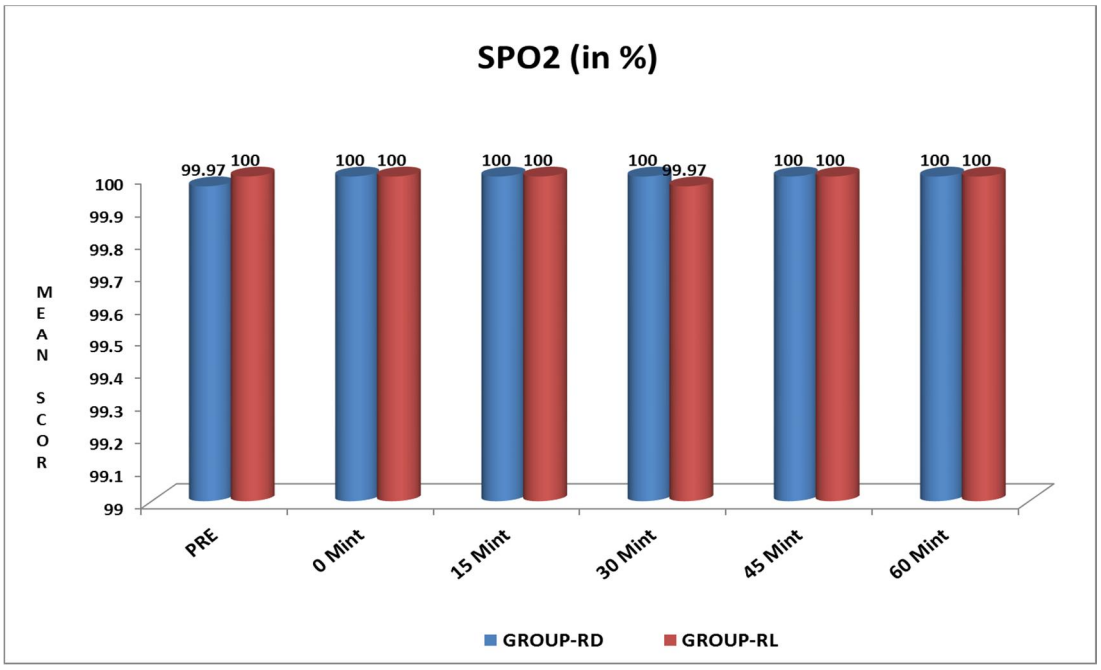
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The mean arterial pressure showed a significant reduction in the dexmedetomidine group starting from 15th minute in comparison with the control group without requiring treatment.

TABLE-12 : SPO2

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
PRE	99.97	0.18	100.00	0.00	1.00	0.32	NS
0 Mint	100.00	0.00	100.00	0.00			
15 Mint	100.00	0.00	100.00	0.00			
30 Mint	100.00	0.00	99.97	0.18	1.00	0.32	NS
45 Mint	100.00	0.00	100.00	0.00			
60 Mint	100.00	0.00	100.00	0.00			

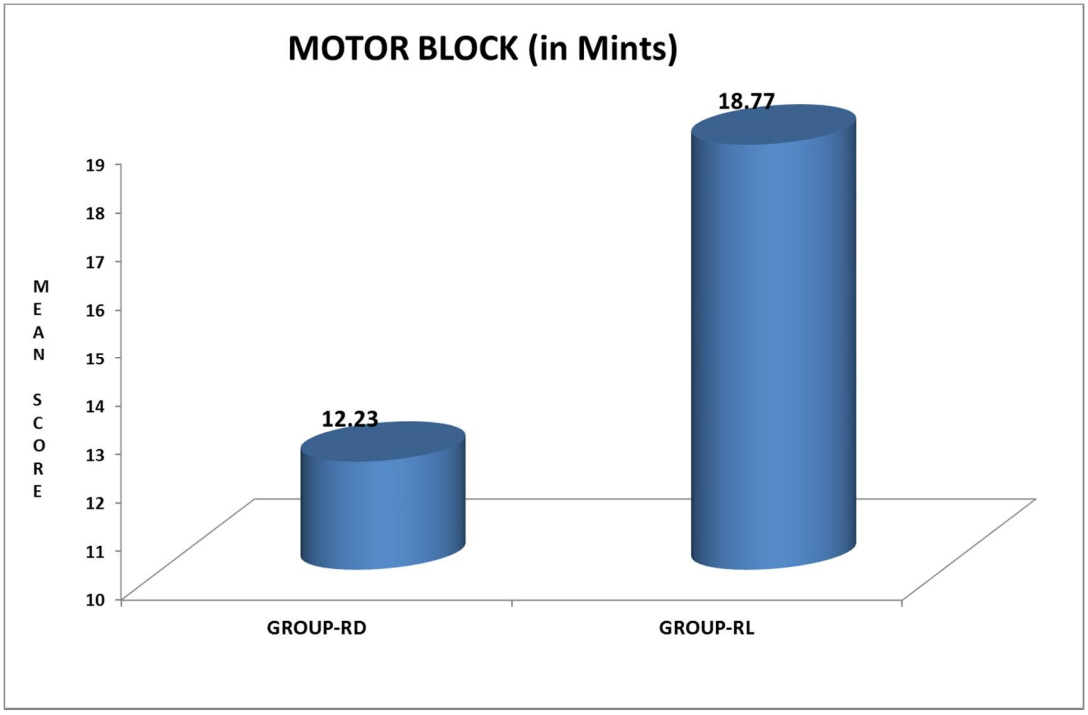


GROUP RD – Dexmedetomidine group
GROUP RL – Local anaesthetic group

No significant difference in SpO2 occurs between the two groups.

TABLE-13 : ONSET OF MOTOR BLOCK

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
Motor	12.23	1.85	18.77	1.22	16.13	0.000	Significant



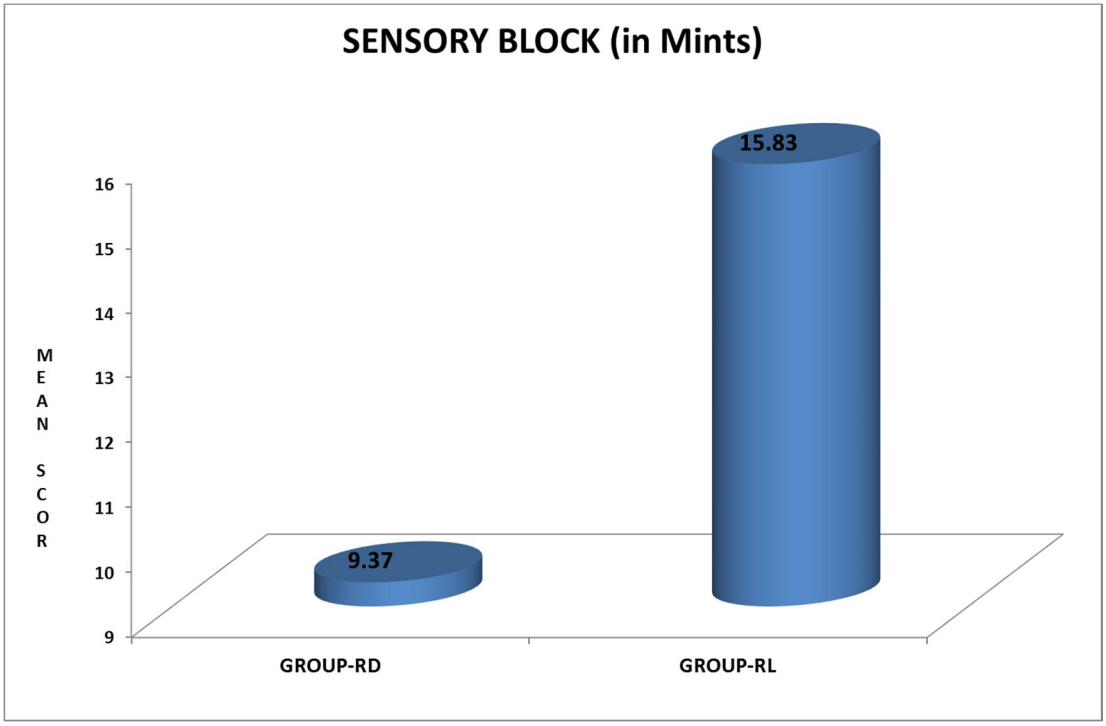
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The mean onset time of motor block in the dexmedetomidine group was 12.23 ± 1.85 minutes and was found to be significantly earlier than the local anaesthetic group.

TABLE-14 : ONSET OF SENSORY BLOCK

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
Sensory	9.37	1.10	15.83	1.18	22.01	0.000	Significant



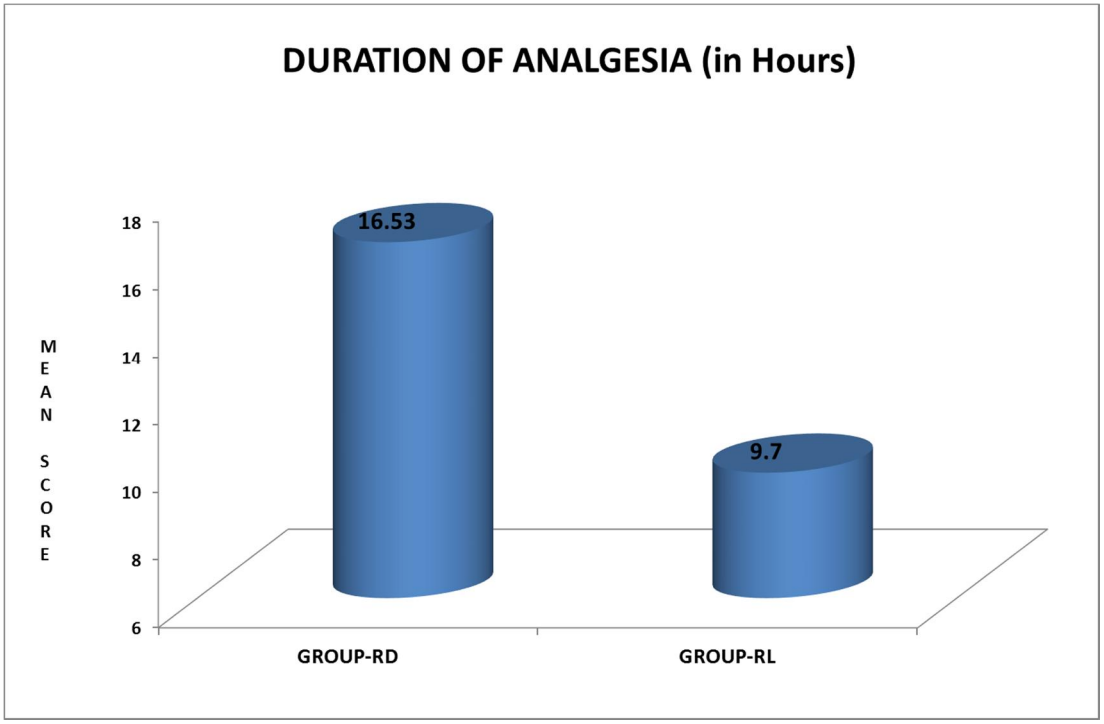
GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The mean onset time of sensory block in the dexmedetomidine group was 9.37 ± 1.10 minutes and was found to be significantly earlier than the local anaesthetic group.

TABLE-15 : DURATION OF ANALGESIA

	GROUP-RD		GROUP-RL		t-value	p-value	Significant
	Mean	Sd	Mean	Sd			
Analgesia	16.53	1.41	9.70	1.44	18.57	0.000	Significant



GROUP RD – Dexmedetomidine group

GROUP RL – Local anaesthetic group

The mean duration of analgesia in the dexmedetomidine group was 16.53 ± 1.41 hours and was found to be significantly longer than the local anaesthetic group.

TABLE-16 : Visual Analog Scale Score 0 – 4 HOURS

	GROUP-RD		GROUP-RL	
	Number	**Percentage**	**Number**	**Percentage**
V0	**30**	**100**	**28**	**93.33**
V2	**0**	**0**	**2**	**6.67**

All the patients in the dexmedetomidine group had a Visual Analog Scale score of 0(no pain) at the end of 4 hours whereas only 28 patients in the control group had a VAS score of 0 and two patients had a VAS score of 2 indicating mild pain requiring no treatment.

TABLE-17 : 4 – 8 HOURS

	GROUP-RD		GROUP-RL	
	Number	**Percentage**	**Number**	**Percentage**
V0	**30**	**100**	**5**	**16.67**
V2	**0**	**0**	**23**	**76.67**
V4	**0**	**0**	**2**	**6.67**

All the patients in the dexmedetomidine group had a Visual Analog Scale score of 0(no pain) at the end of 8 hours whereas only five patients in the control group had a VAS score of 0, twenty three patients had a VAS score of 2 and two patients had a VAS score of 4(moderate pain) that required treatment.

TABLE-18 : 8 – 12 HOURS

	GROUP-RD		GROUP-RL	
	Number	**Percentage**	**Number**	**Percentage**
V0	**23**	**76.67**	**0**	**0**
V2	**7**	**23.33**	**5**	**16.67**
V4	**0**	**0**	**25**	**83.33**

Twenty three patients had a VAS score of 0 and seven patients had a VAS score of 2(mild pain) in the dexmedetomidine group whereas in the control group five patients had a VAS score of 2 and twenty five patients had a VAS score of 4(moderate pain) requiring treatment at the end of 12 hours.

TABLE-19 : 12 – 16 HOURS

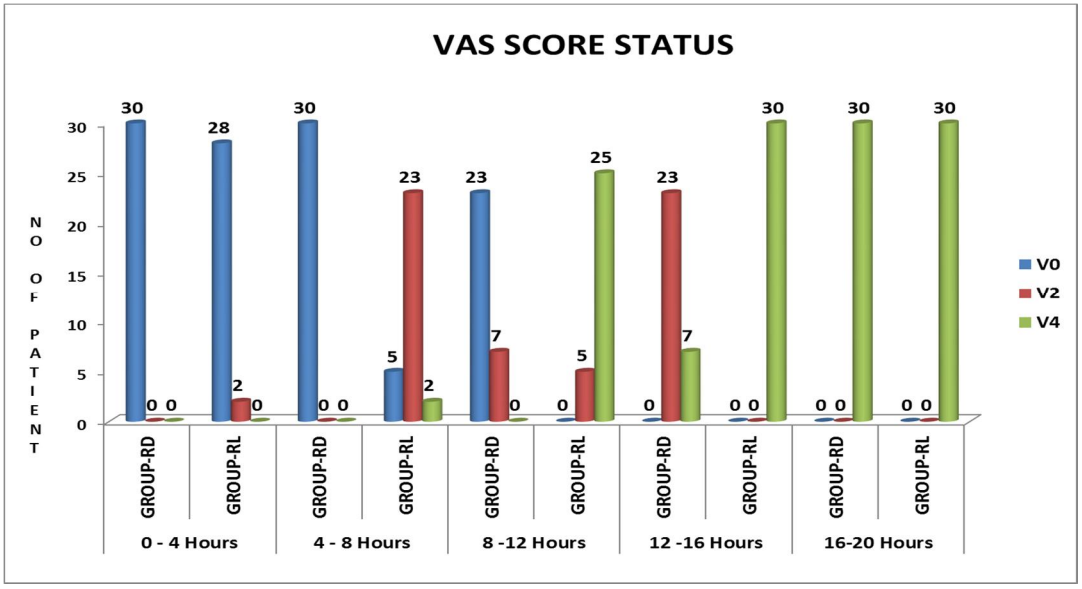
	GROUP-RD		GROUP-RL	
	Number	**Percentage**	**Number**	**Percentage**
V2	**23**	**76.67**	**0**	**0**
V4	**7**	**23.33**	**30**	**100**

Now at the end of 16 hours, in the dexmedetomidine group still twenty three patients had a VAS score of 2 and only seven patients had a VAS score 4 requiring treatment. In the control group, all the thirty patients had a VAS score of 4 requiring treatment.

TABLE-20 : 16 – 20 HOURS

	GROUP-RD		GROUP-RL	
	Number	**Percentage**	**Number**	**Percentage**
V4	**30**	**100**	**30**	**100**

At the end of 20 hours, all the patients in both the dexmedetomidine group and the control group had a VAS score of 4 requiring treatment.



Discussion

DISCUSSION

Axillary block is one of the most commonly performed regional nerve block for upper limb surgeries. Adjuvants have been added to local anaesthetic mixtures to fasten the onset of sensory and motor block and to prolong the duration of anaesthesia.

Alpha – 2 agonists are a group of recently discovered drugs. Two drugs have been identified – clonidine and dexmedetomidine. Dexmedetomidine has recently been approved for the usage of sedation in patients in the Intensive Care Unit.

Dexmedetomidine is a more selective alpha – 2 agonist than clonidine and has been found to prolong the effects of local anaesthetics in epidural, intrathecal and peripheral nerve blocks with the advantages of minimal respiratory depression and cardiovascular stability.

This prospective randomised controlled study conducted in 60 patients who underwent elective upper limb surgeries under axillary plexus block demonstrated that dexmedetomidine in the dose of 50microgram when added to local anaesthetic mixture prolonged the duration of analgesia significantly.

Both the study and control groups were comparable in terms of age, weight, height and BMI. The mean age of the patients in the dexmedetomidine group was 37.6 ± 10.07 years. The mean age of the patients in the control group was 33.13 ± 9.99 years. The mean body mass index of the patients in the dexmedetomidine was 24.38 ± 2.6. The mean body mass index of the patients in the control group was 23.78 ± 2.09. The variables were compared using independent sample test and Levene's test for the equality of variances and p value was found to be not significant.

The mean arterial pressure of the patients in the dexmedetomidine group was 80.27 ± 6.39 mmHg whereas the mean arterial pressure in the control group was 97.47 ± 8.16 mmHg at the 45^{th} minute. The mean pulse rate of the patients in the dexmedetomidine group was around 70 bpm in the dexmedetomidine group and around 85 bpm in the control group but none of the patients required atropine. Statistical analysis of the mean arterial pressure and mean pulse rate was done and the p value was found to be significant.

The grades of sensory and motor block were checked every 5minutes after performance of the axillary block using pin prick and Modified Bromage Scale respectively. The onset time of sensory and

motor blockade is defined as the time interval between the end of local anaesthetic injection and

- Loss of sensation to pin prick (sensory score – 1)
- Paresis (motor scale – 1) in the distribution of all peripheral nerves respectively.

The onset time of sensory block in the dexmedetomidine group was found to be 9.37 ± 1.7 minutes whereas in the control group it was found to be 15.83 ± 1.18 minutes. The onset time of motor block in the dexmedetomidine group was found to be 12.23 ± 1.85 minutes whereas in the control group it was found to be 18.77 ± 1.22 minutes. On statistical analysis by the independent sample test and the t test for equality of means has showed a faster onset times of sensory and motor block significantly with a p value of 0.0001***.

The patients were followed in the post-operative ward every 2 hours for the presence of pain by the Visual Analog Scale. The presence of VAS score 4 is taken as the endpoint of analgesia. The duration of analgesia is taken as the time from the performance of the block till the appearance of VAS score 4.

The mean duration of analgesia in the dexmedetomidine group was found to be 16.53 ± 1.41 hours whereas the mean duration of analgesia in the control group was found to be 9.70 ± 1.44 hours. At the 16^{th} hour the number of patients with a VAS score of 4 in the dexmedetomidine group was 7 whereas in the control group all the 30 patients had a VAS score of 4. At the 20^{th} hour all the 60 patients in both the groups had a VAS score of 4.

Yu Zhang et al in their study of 45 patients had demonstrated similar faster onset of sensory and motor block - 15.46 ± 3.67 minutes and 18.54 ± 5.24 minutes respectively on addition of 50microgram of dexmedetomidine to 0.33% ropivacaine. They also demonstrated prolongation of the duration of sensory and motor block - 804.00 ± 340.00 minutes and 737.73 ± 135.99 minutes respectively. The incidence of bradycardia and hypotension that required treatment was higher when 100microgram of dexmedetomidine was added to ropivacaine as compared to 50microgram of dexmedetomidine added to ropivacaine. Hence in our study we decided to add a low dose of dexmedetomidine to the local anaesthetic mixture to produce the desired response with minimal side effects. In our study we added dexmedetomidine to local anaesthetic mixture of bupivacaine and lignocaine with adrenaline and obtained comparable results.

Kenan Kaygusuz et al in their study of 60 patients demonstrated that the onset time of sensory and motor block in the dexmedetomidine group was found to be 7.75 minutes and 14.25 minutes respectively and was earlier than the control group. The duration of analgesia in this study was found to be 924.15 minutes and was comparable to our study. None of the patients who developed bradycardia, hypotension or hypertension required treatment, which was similar to our study.

Imasogie N in his study compared the ultrasound guided axillary plexus block using 2 point versus 4 point injections and concluded that the 2 point injection was faster to administer but the block success rate was similar in both the groups. In our study we performed the axillary plexus block using the 2 point injection technique.

Saumya Biswas evaluated the addition of dexmedetomidine to levobupivacaine in supraclavicular plexus block and concluded that the duration of sensory and motor block was significantly longer in the dexmedetomidine group. In our study we added dexmedetomidine to the local anaesthetic mixture of bupivacaine plus lignocaine with adrenaline.

All the patients in our study both study and control groups were monitored in the post-operative ward and were given supplemental oxygen at the rate of 2litres/min. Dexmedetomidine has the unique property in causing arousable sedation without any respiratory depression.

Conclusion

CONCLUSION

Dexmedetomidine in the dose of 50microgran when added as an adjuvant to local anaesthetic mixture in axillary plexus block had a faster onset of sensory and motor block(9.37 ± 1.1 and 12.23 ± 1.85 minutes) and significantly prolonged the duration of analgesia(16.53 ± 1.41 hours). They caused a minimal decrease in the mean arterial pressure and heart rate which did not warrant treatment.

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Annexures

PROFORMA

Name of the patient: **Age:**

Sex: **Weight:**

Insurance No: **OT:**

Diagnosis: **Duration of Procedure:**

Surgeon: **Anaesthetist:**

PREOPERATIVE DETAILS

ASA GRADE:

VITALS:

BP	Pulse rate	Resp. rate	SpO2	Temp	ECG	Xray

Hb	RBS	RFT	LFT	Others

Onset of sensory block	Onset of motor block	Duration of analgesia

SIGNATURE OF INVESTIGATOR **SIGNATURE OF PATIENT**

WITNESS:

PATIENT CONSENT FORM

STUDY TITLE: ***Prospective randomized controlled study evaluating dexmedetomidine as an adjuvant added to a local anaesthetic mixture of bupivacaine, lignocaine with adrenaline in axillary plexus block as compared to axillary plexus block with local anaesthetic mixture alone.***

STUDY CENTRE: ESIC MEDICAL COLLEGE & PGIMSR,
KK NAGAR, CHENNAI – 78.

PARTICIPANT NAME: **AGE:** **SEX:**

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I understand that I will receive drugs to prolong the duration of analgesia using dexmedetomidine in axillary brachial plexus block. I have been explained that the anaesthetic technique is a standard and approved technique. This may help in future research in the field of anaesthesia. I consent to undergo this procedure.

INSURANCE NO:

DATE: **Signature/thumb impression of patient**

ஒப்புதல் படிவம்

1. எனக்கு _____
   அறுவை சிகிச்சை செய்யுமாறு இ.எஸ்.ஐ.ஸி.(ESIC) மருத்துவர் மற்றும் குழுவினரை வேண்டிக் கொள்கிறேன்.
2. நோயின் தன்மை :
   சிகிச்சை முறை :
3. மேலே குறிப்பிட்டுள்ள சிகிச்சைக்கு அக்குளில் மரத்துப் போகிற ஊசி போட்டு அறுவை சிகிச்சை செய்ய முழு சம்மதம் அளிக்கிறேன்.
4. அனைத்து மருத்துவ சிகிச்சை முறைகளின் நிறைகளும் குறைகளும் எனக்கு விளக்கப்பட்டன.
5. மேலே கொடுக்கபட்டுள்ள அனைத்தும் மருத்துவமனை நன்னெறி (Ethics) குழுவின் வரைமுரைக்கு உட்பட்டே நடக்கும் என மருத்துவர் விளக்கினார். மேலும் இந்த சிகிச்சை முறைகளுக்கு உடன்பட மறுக்கவும் எனக்கு உரிமை உண்டு என்பதை நான் அறிவேன்.
6. என் பெயர் உட்பட்ட அடையாளங்கள் மற்றும் நோய் / சிகிச்சை முறை பற்றிய தகவல்களை பிறருக்கு தெரிவிக்கபடாது என மருத்துவர் கூறினார்
7. என் சிகிச்சையின் போது கிடைக்கும் தகவல்களை மருத்துவ ஆராய்சிக்கு பயன்படுத்தவும் சம்மதம் அளிக்கிறேன்.

நான் இந்த ஒப்புதல் படிவத்தை படித்த / படித்து காண்பிக்க பட்ட பின்னரே இதன் சாராம்சத்தை முழுவதுமாக புரிந்து கொண்டு மேற்சொன்ன அறுவை / மயக்க சிகிச்சை முறைகளுக்கு முழுமனதுடன் சம்மதிக்கிறேன்.

சென்னை, ஒப்புதல் அளிப்பவர் :

தேதி : எண் :

சாட்சி :

KEY TO MASTER CHART

GROUPS:

GROUP RD	-	Dexmedetomidine group
GROUP RL	-	Local anaesthetic group

PARAMETERS:

BMI	-	Body mass index
PR	-	Pulse rate
SYS	-	Systolic blood pressure
DIA	-	Diastolic blood pressure
MAP	-	Mean arterial pressure
ON_SB	-	Onset of sensory blockade
ON_MB	-	Onset of motor blockade
DUR	-	Duration of analgesia
V0	-	VAS score of 0
V2	-	VAS score of 2
V4	-	VAS score of 4

MASTER CHART

NAME	GROUP	AGE	SEX	IP NO	WEIGHT	HEIGHT	BMI	ASA	LEVEL OF SUR	PR-0	SYS-0	DIA-0	MAP-0	ON_MB	ON_SB	PR-30	SYS-30	DIA-30	MAP-30	PR-60	SYS-60	DIA-60	MAP-60	DURATION O	0 - 2hr	2 - 4hr	4 - 6hr	6 - 8 hr	8 - 10hr	10-12hr	12-14hr	14-16hr	16-18hr	18-20hr
ANANDAN	D	37	M	51-22801803	76	166	27.5802003	I	DIST RADIUS	80	110	70	83.333333	17	11	74	108	66	80	80	112	74	86.666667	18	V0	V0	V0	V0	V0	V0	V2	V2	V4	V4
LATHA	L	34	F	51-24496545	56	160	21.875	I	WRIST	70	100	60	73.333333	20	15	66	100	60	73.333333	74	120	78	92	10	V0	V0	V0	V2	V4	V4	V4	V4	V4	V4
VIJAYA	D	58	F	51-21883979	50	162	19.0519738	II	ELBOW	80	130	90	103.33333	14	10	76	124	70	88	72	110	70	83.333333	17	V0	V0	V0	V0	V0	V0	V2	V2	V4	V4
DELHIBABU	L	25	M	51-15502683	68	170	23.5294118	I	HAND	88	108	60	76	20	18	88	128	92	104	86	126	88	100.66667	8	V0	V0	V2	V4	V4	V4	V4	V4	V4	V4
MURUGAN	D	32	M	51-23351679	70	166	25.4028161	I	HAND	70	110	70	83.333333	13	10	60	108	70	82.666667	62	108	70	82.666667	16	V0	V0	V0	V0	V0	V0	V2	V4	V4	V4
KUPPAN	L	45	M	51-16310334	70	170	24.2214533	II	FOREARM	86	130	90	103.33333	19	16	88	140	90	106.66667	84	130	90	103.33333	10	V0	V0	V0	V2	V4	V4	V4	V4	V4	V4
CHANDRA MEHRA	D	26	M	51-23137955	60	166	21.7738424	I	HAND	66	110	70	83.333333	14	11	60	100	60	73.333333	62	108	60	76	16	V0	V0	V0	V0	V0	V0	V2	V4	V4	V4
RAJESH	L	30	M	51-16672842	70	170	24.2214533	I	ELBOW	80	120	80	93.333333	20	18	78	130	90	103.33333	74	130	90	103.33333	7	V0	V0	V2	V4	V4	V4	V4	V4	V4	V4
GOPINATH	D	20	M	51-15585981	50	158	20.0288415	I	RADIUS	90	100	60	73.333333	13	10	78	100	60	73.333333	78	100	60	73.333333	15	V0	V0	V0	V0	V0	V0	V2	V4	V4	V4
MOORTHY	L	42	M	51-23023071	80	170	27.6816609	II	FOREARM	80	140	90	106.66667	18	16	90	130	90	103.33333	88	120	86	97.333333	10	V0	V0	V0	V2	V4	V4	V4	V4	V4	V4
LATHA	D	38	F	51-24496545	56	160	21.875	I	FOREARM	90	100	60	73.333333	14	10	80	96	60	72	80	100	60	73.333333	16	V0	V0	V0	V0	V0	V0	V2	V4		
SELVAGANAPATHY	L	21	M	51-24577656	60	160	23.4375	I	DIST RADIUS	70	110	70	83.333333	20	17	80	120	80	93.333333	86	120	80	93.333333	8	V0	V0	V2	V4						
PADMA	D	55	M	51-23596179	80	162	30.4831581	II	DIST RADIUS	80	140	90	106.66667	15	11	82	126	80	95.333333	76	110	70	83.333333	15	V0	V0	V0	V0	V0	V2	V4			
SURESH BABU	L	22	M	51-21229459	54	166	19.5964581	I	ULNAR	90	94	60	71.333333	18	14	92	100	62	74.666667	90	100	60	73.333333	8	V0	V0	V2	V4						
PARTHIBAN	D	32	M	51-16894422	74	172	25.0135208	I	RADIUS	76	128	90	102.66667	14	10	70	110	70	83.333333	66	108	60	76	16	V0	V0	V0	V0	V0	V0	V2	V4		
KAVITHA	L	24	F	51-23313701	54	158	21.6311489	I	FOREARM	70	100	60	73.333333	18	16	80	120	80	93.333333	82	116	80	92	10	V0	V0	V0	V2	V4					
PRABHU	D	22	M	51-24367321	60	166	21.7738424	I	HAND	88	110	70	83.333333	12	10	80	100	60	73.333333	72	100	60	73.333333	15	V0	V0	V0	V0	V0	V0	V2	V4		
DILLI	L	28	M	51-15502683	68	170	23.5294118	I	HAND	80	116	70	85.333333	20	18	84	120	90	100	86	124	90	101.33333	12	V0	V0	V0	V0	V2	V4				
MOHAN	D	32	M	51-11721471	80	172	27.0416441	I	FOREARM	90	126	70	88.666667	12	9	80	110	70	83.333333	78	120	80	93.333333	14	V0	V0	V0	V0	V0	V2	V4			
GANARAJ	L	23	M	51-23372772	66	168	23.3843537	I	HAND	66	100	60	73.333333	21	17	76	100	60	73.333333	72	120	80	93.333333	9	V0	V0	V0	V2	V4					
SURESH	D	48	M	51-15981532	70	172	23.6614386	I	ELBOW	90	130	90	103.33333	13	10	88	130	90	103.33333	80	120	76	90.666667	14	V0	V0	V0	V0	V0	V2	V4			
MURUGAN	L	34	M	51-22504048	78	170	26.9896194	I	RADIUS	80	110	70	83.333333	20	18	78	130	90	103.33333	88	120	84	96	9	V0	V0	V0	V2	V4					
INDIRAN	D	42	M	51-16359180	80	172	27.0416441	II	FOREARM	84	130	90	103.33333	10	8	70	120	90	100	70	108	70	82.666667	16	V0	V0	V0	V0	V0	V0	V2	V4		
MANIKANDAN	L	30	M	51-16864833	74	168	26.2188209	I	HAND	80	100	60	73.333333	18	16	82	120	80	93.333333	80	110	70	83.333333	10	V0	V0	V0	V2	V4					
VISHWANATHAN	D	50	M	51-27629971	66	174	21.7994451	I	HAND	80	100	60	73.333333	9	11	84	110	70	83.333333	70	116	70	85.333333	16	V0	V0	V0	V0	V0	V0	V2	V4		
VINOTH KUMAR	L	30	M	51-21822534	80	174	26.4235698	I	HAND	90	116	80	92	20	17	96	124	70	88	80	120	80	93.333333	9	V0	V0	V0	V2	V4					
VISHWA	D	40	M	51-15265343	74	172	25.0135208	II	ELBOW	80	110	70	83.333333	11	9	80	100	60	73.333333	68	100	70	80	15	V0	V0	V0	V0	V0	V2	V4			
SUBRAMANIAN	L	50	M	51-11559315	78	168	27.6360544	I	DIST RADIUS	76	130	90	103.33333	20	16	84	120	92	101.33333	88	126	90	102	7	V0	V0	V2	V4						
ANBU	D	24	M	51-18435165	66	179	20.598608	I	DIST RADIUS	68	110	70	83.333333	12	10	70	108	70	82.666667	76	110	70	83.333333	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
MUKESH	L	30	M	51-61627154	70	172	23.6614386	I	HAND	70	120	76	90.666667	18	15	76	126	90	102	70	130	90	103.33333	8	V0	V0	V2	V4						
KANNIAMMAL	D	46	F	51-20306787	54	160	21.09375	II	RADIUS	80	136	90	105.33333	12	10	76	120	86	97.333333	80	110	70	83.333333	17	V0	V0	V0	V0	V0	V0	V0	V2	V4	
SUMATHY	L	55	F	51-13896291	60	162	22.8623685	I	DIST RADIUS	86	128	90	102.66667	18	15	90	136	96	109.33333	82	120	80	93.333333	10	V0	V0	V0	V2	V4					
KRISHNA BAHADUR	D	32	M	51-27228586	76	170	26.2975779	I	DIST RADIUS	76	120	70	86.666667	12	9	66	110	76	87.333333	62	110	70	83.333333	15	V0	V0	V0	V0	V0	V0	V2	V4		
SIVARAMAN	L	42	M	51-15128116	66	168	23.3843537	I	ELBOW	90	130	90	103.33333	17	15	86	128	90	102.66667	90	140	96	110.66667	8	V0	V0	V2	V4						
SENTHIL ALAGAR	D	45	M	51-13483628	78	176	25.1807851	I	HAND	76	110	70	83.333333	13	10	66	100	60	73.333333	60	110	68	82	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
BIRENDER KUMAR	L	29	M	51-21134756	60	164	22.3081499	I	HAND	90	140	90	106.66667	16	14	88	128	90	102.66667	90	130	96	107.33333	12	V0	V0	V0	V0	V2	V4				
MANIKANDAN	D	25	M	51-23539951	78	170	26.9896194	I	HAND	70	110	70	83.333333	14	10	66	106	60	75.333333	68	100	60	73.333333	16	V0	V0	V0	V0	V0	V0	V2	V4		
PRASANTH	L	20	M	51-23448521	66	174	21.7994451	I	HAND	100	130	90	103.33333	20	16	98	130	90	103.33333	92	130	96	107.33333	12	V0	V0	V0	V0	V2	V4				
ABEL	D	38	M	51-36913690	70	172	23.6614386	II	HAND	90	110	70	83.333333	13	8	90	110	70	83.333333	82	120	80	93.333333	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
MARIA SUDHA	L	30	F	51-15024415	56	160	21.875	I	FOREARM	86	126	90	102	20	16	92	130	90	103.33333	90	130	96	107.33333	12	V0	V0	V0	V0	V2	V4				
ARUN KUMAR	D	23	M	51-21510291	74	168	26.2188209	I	FOREARM	72	110	70	83.333333	11	9	66	100	60	73.333333	58	96	60	72	17	V0	V0	V0	V0	V2	V4				
PALANI	L	30	M	51-24002885	80	178	25.2493372	I	DIST RADIUS	70	110	70	83.333333	17	15	84	108	70	82.666667	82	126	90	102	10	V0	V0	V0	V2	V4					
CHINNA	D	42	M	51-21303162	72	170	24.9134948	I	FOREARM	90	140	90	106.66667	10	8	70	110	72	84.666667	70	120	80	93.333333	17	V0	V0	V0	V0	V0	V0	V2	V4		
ARUNKUMAR	L	26	M	51-16247603	72	170	24.9134948	I	HAND	76	110	70	83.333333	18	14	80	120	86	97.333333	80	118	90	99.333333	10	V0	V0	V0	V2	V4					
NARENDRA BETHR	D	40	M	51-43645231	78	172	26.365603	II	HAND	90	140	90	106.66667	13	10	76	110	70	83.333333	70	120	80	93.333333	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
GANAPATHY	L	50	M	51-17255306	80	172	27.0416441	II	DIST RADIUS	80	130	90	103.33333	19	16	88	136	90	105.33333	88	142	96	111.33333	10	V0	V0	V0	V2	V4					
MARIMOON SAIBA	D	30	F	51-21387018	70	166	25.4028161	I	WRIST	76	110	70	83.333333	10	7	60	100	60	73.333333	60	100	60	73.333333	16	V0	V0	V0	V0	V0	V0	V2	V2		
SARATH KUMAR	L	20	M	51-24198094	58	166	21.0480476	I	DIST RADIUS	72	110	70	83.333333	18	15	90	130	90	103.33333	90	140	90	106.66667	9	V0	V0	V0	V2	V4					
UNNI	D	47	M	51-23161266	76	176	24.535124	I	WRIST	76	110	70	83.333333	10	8	60	100	66	77.333333	56	98	60	72.666667	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
PRADEEP KUMAR	L	43	M	51-16162239	70	174	23.1206236	I	HAND	80	120	90	100	20	16	92	130	96	107.33333	80	128	76	93.333333	10	V0	V0	V0	V2	V4					
PARTHIBAN	D	45	M	51-15151359	78	178	24.6181038	I	DIST RADIUS	76	130	90	103.33333	10	8	72	110	76	87.333333	60	96	60	72	20	V0	V0	V0	V0	V0	V0	V0	V0	V2	V4
AMIRTHAVALLI	L	41	F	51-22998000	60	166	21.7738424	I	WRIST	90	120	80	93.333333	18	15	82	136	92	106.66667	98	126	88	100.66667	10	V0	V0	V0	V2	V4					
MOHAN	D	48	M	51-11721474	72	180	22.2222222	II	DIST RADIUS	86	110	70	83.333333	13	9	60	100	58	72	66	110	66	80.666667	16	V0	V0	V0	V0	V0	V0	V2	V4		
BUELA	L	45	F	51-15236476	70	166	25.4028161	I	WRIST	76	108	70	82.666667	18	15	82	120	80	93.333333	78	120	86	97.333333	11	V0	V0	V0	V0	V2	V4				
NEELAMALAR	D	43	F	51-15358549	82	174	27.0841591	II	WRIST	70	110	70	83.333333	13	9	62	108	70	82.666667	58	100	70	80	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
VENKATACHALAM	L	40	M	51-13393854	70	178	22.0931701	I	HAND	96	130	90	103.33333	18	15	88	130	90	103.33333	90	140	90	106.66667	12	V0	V0	V0	V2	V4					
CHANDRAN	D	39	M	51-14169789	80	178	25.2493372	I	WRIST	70	130	90	103.33333	10	8	68	108	70	82.666667	60	108	70	82.666667	17	V0	V0	V0	V0	V0	V0	V0	V2	V4	
PRASANTH KUMAR	L	20	M	51-24732174	60	166	21.7738424	I	HAND	76	110	70	83.333333	18	15	86	120	86	97.333333	84	126	90	102	10	V0	V0	V0	V2	V4					
SURENDER	D	29	M	51-24383836	68	170	23.5294118	I	DIST RADIUS	72	130	90	103.33333	10	8	66	108	76	86.666667	66	106	60	75.333333	18	V0	V0	V0	V0	V0	V0	V0	V2	V4	
VASUDEVAN	L	35	M	51-23278959	70	168	24.8015873	I	DIST RADIUS	70	130	90	103.33333	18	16	80	140	90	106.66667	82	130	90	103.33333	10	V0	V0	V0	V2	V4					