DEXMEDETOMIDINE AS AN ADJUVANT TO LIGNOCAINE IN INTRAVENOUS REGIONAL ANAESTHESIA

A STUDY OF 60 CASES

DISSERTATION SUBMITTED FOR DOCTOR OF MEDICINE

BRANCH X (ANAESTHESIOLOGY)

APRIL 2012

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI,

TAMILNADU
BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled “DEXMEDETOMIDINE AS AN ADJUVANT TO LIGNOCAINE IN INTRAVENOUS REGIONAL ANAESTHESIA” submitted by Dr.T.ARUN PRAKASH to the faculty of ANAESTHESIOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement in the award of degree of M.D., Degree Branch -X (ANAESTHESIOLOGY), for the April 2012 examination is a bonafide research work carried out by him under my direct supervision and guidance.

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DECLARATION

I, Dr.T.ARUN PRAKASH declare that the dissertation titled “DEXMEDETOMIDINE AS AN ADJUVANT TO LIGNOCAINE IN INTRAVENOUS REGIONAL ANAESTHESIA” has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.D., Degree, Branch X (ANAESTHESIOLOGY) degree Examination to be held in April 2012.

I also declare that this dissertation, in part or full was not submitted by me or any other to any other University or Board, either in India or abroad for any award, degree or diploma.

Place: Madurai

Date: 

Dr. T. ARUN PRAKASH
ACKNOWLEDGEMENT

I have great pleasure in expressing my deep sense of gratitude to Dr. T. THIRUNAVUKKARASU M.D., D.A., Professor and Director i/c of the Institute Of Anaesthesiology, Government Rajaji Hospital and Madurai Medical College, Madurai for his kind encouragement and valuable guidance during the period of this study, with which this dissertation would not have materialized.

I would like to place on record my indebtedness to my Professors Dr. S.C. GANESH PRABU, M.D., D.A., Dr. R. SHANMUGAM, M.D., and Dr. A. PARAMASIVAN, M.D., D.A., of the Institute of Anaesthesiology, Madurai Medical College, Madurai for their whole hearted help and support in doing this study.

I express my sincere thanks to Dr. EDWIN JOE, M.D., THE DEAN, Madurai Medical College and Government Rajaji Hospital for permitting me to utilize the clinical materials of this hospital.

I express my profound thanks to assistant professor Dr. S. SENTHIL KUMAR, M.D., D.A., for his valuable suggestions and technical guidance in doing this study.

Lastly, I am conscious of my indebtedness to all my patients for their kind co-operation during the course of study.
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PROFORMA

MASTER CHART
DEXMEDETOMIDINE AS AN ADJUVANT TO LIGNOCAINE IN INTRAVENOUS REGIONAL ANESTHESIA

ABSTRACT

**Background:** In this randomized, double-blind, prospective study, the effect of adding dexmedetomidine as an adjuvant to lignocaine in Intravenous regional anesthesia is evaluated in terms of sensory and motor block onset time, intraoperative sedation, incidence of tourniquet pain and postoperative analgesia.

**Methods:** After informed consent and ethical committee approval, sixty ASA I and II patients scheduled for the hand and forearm surgeries were included in this study. Patients with Raynaud disease, sickle cell anaemia or a history of allergy to local anesthetics and dexmedetomidine were excluded from the study. After the patients had been taken to operating room, blood pressure, peripheral oxygen saturation and heart rate were monitored. Patients divided into two groups, in Group A 3mg/kg 0.5% lignocaine with 0.5micrograms/kg dexmedetomidine and in Group B 3mg/kg 0.5% lignocaine were injected. Before the block, two cannula were placed, one in operative hand and other in the opposite hand. Operating hand is exsanguinated and using double tourniquet technique the drugs were injected in respective groups.
Intraoperatively mentioned vital parameters monitored regularly at 5, 10, 15, 20, 30, 40, 50, 60, 80, 90 and 120 minutes. Sensory and motor block onset time, intraoperative sedation, tourniquet pain time, hemodynamics, postoperative analgesia and side effects were noted if present. Sedation was assessed using Ramsay sedation scale and postoperative analgesia assessed by visual analogue scale. Time since tourniquet release to VAS more than 3 is considered total postoperative analgesia and rescue analgesia inj. diclofenac 75mg intramuscularly given and the study is completed.

**Results:** Both the Groups were comparable in respect of age, sex, weight and duration of surgery. In Group A onset of sensory block is 1.8±0.76 minutes and motor block is 13.63±1.54 minutes, in Group B it was 5.27±0.58 minutes and 18.07±1.26 minutes respectively. In Group A sensory and motor recovery time were 18.87±3.27 minutes and 25.6±3.82 minutes and in Group B they were 4.8±0.7 minutes and 2.53±0.51 minutes respectively. Sedation in Group A was 1.77±0.43 and in Group B was 1. In Group A postoperative analgesia is 416.2±45.73 minutes and in Group B was 11.33±0.96 minutes. No side effects were noted.
**Conclusion:** It is concluded that when Dexmedetomidine 0.5 micrograms/kg is added to lignocaine for Intravenous regional anesthesia, it provided quicker onset of sensory and motor blockade, lesser incidence of tourniquet pain, increased duration of post operative analgesia and better haemodynamic stability without any side effects.

**Keywords:** Dexmedetomidine, tourniquet, postoperative analgesia
INTRODUCTION

Intravenous Regional Anaesthesia (IVRA) since its birth in the hands of August Bier in 1908 has become a valuable instrument in the repertoire of anaesthesiologists. This method enjoyed wide popularity for a time. It is not long before simple and reliable techniques for blocking the brachial plexus developed, and the intravenous method declined in popularity.

It was revived in 1963 by Holmes, who used lignocaine because it appeared to give more reliable anaesthesia than procaine. Today intravenous regional anesthesia with slight technical modifications is an ideal method of providing anaesthesia for minor surgical procedures to the extremities performed on an ambulatory basis. It has the advantages of speed of onset, rapid recovery, reliability of blockade & cost effectiveness.

Adjuvants to local anaesthetics have greatly expanded the potential applications of regional anaesthesia by providing faster onset time, inhibition of tourniquet pain, prolonged post-operative analgesia and improved peri-operative analgesia apart from decreasing the risk of local anaesthetic toxicity.
In this regard, Dexmedetomidine—a parenterally administered α2 agonist—have analgesic effects by depressing the nerve action potentials especially in C fibres and also acting at α2-adrenergic receptors located at nerve endings. When given in combination with Local anaesthetics it decreases the peri-operative pain.

Additive effects of these agents result in greater patient satisfaction, rapid hospital discharge, cost effectiveness and minimal risks.
AIM OF THE STUDY

To assess

1. The effectiveness of Dexmedetomidine as an adjuvant in Intravenous regional anesthesia

2. The effectiveness of Dexmedetomidine in preventing tourniquet pain when added as an adjuvant in Intravenous regional anesthesia

3. The duration of postoperative analgesia, when Dexmedetomidine is added as an adjuvant in Intravenous regional anesthesia.
INTRAVENOUS REGIONAL ANAESTHESIA

Intravenous regional anaesthesia (IVRA) was first described by August Bier in 1908. He observed that when local anaesthetic was injected intravenously between two tourniquets on a limb, a rapid onset of anaesthesia occurred in the area between the tourniquets. The technique did not become popular until the 1960s when it was reintroduced by Holmes. Today, the technique is slightly modified, using either a single or a double tourniquet at one site and injecting local anaesthetic as distal as possible to the cuff. The double tourniquet is used to increase safety and to reduce tourniquet pain in the awake patient, but there is a possibility of accidental deflation of the wrong cuff, which may lead to toxic systemic levels of local anaesthetic.

Intravenous Regional Anesthesia is technically simple and does not require specific anatomical knowledge. Success rate is 96–100% with a low incidence of side-effects. It is a reliable, simple and safe method of providing anaesthesia for minor surgical procedures to the extremities if it is administered by experienced clinicians.
Advantages and Disadvantages of intravenous regional anaesthesia

Advantages:

It has faster onset and rapid recovery. It is a simple and reliable technique providing good muscle relaxation in the absence of local infection and with adequate equipment.

Disadvantages and Complications

Postoperative analgesia is poor and provides limited time of surgical anaesthesia for less than 90 minutes. There is potential for systemic local anaesthetic toxicity. Nerve damage can occur secondary to direct compression by the tourniquet and very rarely compartment syndrome and loss of limb can occur as complications.

Mechanism of action:

Local anaesthetic diffuses into the small veins surrounding the nerves and then into the vasa nervorum and capillary plexus of the nerves, leading to a core to mantle (centrifugal) conduction block in the nerves involved. Local anaesthetic then diffuses into the small nerves in the skin, blocking their conduction. The tourniquet produces ischaemia, which contributes to the analgesic action of the local anaesthetic by blocking nerve conduction and motor end plate function. Tourniquet application for 20 minutes alone produce analgesia to pinprick without the injection of
any local anaesthetic. However, the speed of onset and the density of anaesthesia are greater with injection of local anaesthetic.

**Indications:**

Intravenous Regional Anesthesia is used for surgical interventions on the hand, forearm or elbow that will not exceed 1 hour. These include reduction of forearm fractures, excision of wrist ganglia and palmar fasciotomy. Intravenous Regional Anesthesia is particularly useful for tendon grafting because it enables the surgeon to observe movement and tension of the grafted tendon after deflating the tourniquet before closing the wound, anaesthesia continued with a wrist block. Intravenous Regional Anesthesia can also be used for surgery on the foot, ankle or lower leg, for example for removing plates, screws or foreign bodies. Surgery on the elbow or knee is poorly tolerated using Intravenous Regional Anesthesia.

**Contraindications**

Absolute contraindications include sickle cell disease, Raynaud’s disease or scleroderma, allergy to local anaesthetics and patient refusal. Relative contraindications include severe hypertensive or peripheral vascular disease, local infection, and skeletal muscle disorders or Paget’s
disease where the local anaesthetic may spread to the systemic circulation through the venous channels in bone.

**Procedure**

Before the procedure the patient should be starved for 6 hours and monitored closely using standard monitoring. Patient should be placed on a tipping trolley and adequately informed about the procedure and have consented to it.

The equipment required for Intravenous regional anesthesia includes pneumatic tourniquet which is checked for leaks before the procedure, a pressure gauge, Esmarch bandage or Rhys-Davis exsanguinator, local anaesthetic solution, resuscitation equipment and drugs.

**INTRAVENOUS REGIONAL ANESTHESIA OF THE ARM:**

A 22 G cannula is placed intravenously as distal as possible in the arm to be anaesthetized. Venous access is established in the opposite arm to allow administration of fluids or drugs if necessary. In the double tourniquet technique, two tourniquets each 6 cm wide is applied on the arm with generous layers of padding, ensuring that no wrinkles are formed and the tourniquet edges do not touch the skin. The arm is exsanguinated either by using the Esmarch bandage or a Rhys-Davis exsanguinator. If this is impossible, exsanguination can be achieved by elevating the arm for
2–3 minutes while compressing the axillary artery. The distal tourniquet is inflated to at least 100 mmHg higher than the patient’s systolic blood pressure or the tourniquet is inflated until the radial pulse was not felt. The proximal tourniquet is inflated to the same pressure. After ensuring inflation, the distal cuff is deflated.

Before injecting local anaesthetic it must be confirmed that no radial pulse is palpable. The local anaesthetic is then injected slowly. A standard volume for injection into the upper limb is 40 ml, which can be increased to 50 ml in a fit, large adult. If the injection is too rapid, the venous pressure may exceed the tourniquet pressure and the local anaesthetic solution may escape into the systemic circulation. Surgical anaesthesia is usually achieved within 15 minutes. The distal tourniquet, which overlies part of the anaesthetized arm, can then be inflated and the proximal one deflated to relieve tourniquet pain.

The cuff should not be deflated until 20 minutes after local anaesthetic injection because systemic toxic doses of local anaesthetic may occur. After 20 minutes, 30% of the injected drug is fixed within the tissues and is unavailable for immediate release into the systemic circulation. Cuff deflation should be performed in cycles with deflation/inflation times of less than 10 seconds until the patient no longer
exhibits signs of systemic toxicity for example, tingling of the lips, tinnitus and drowsiness. Severe signs of systemic toxicity include bradycardia, hypotension, electrocardiogram abnormalities, seizures and loss of consciousness. Maximum blood levels of local anaesthesia occur within 10 minutes of cuff deflation. Therefore, the patient should be monitored closely for 30 minutes following tourniquet release. With lignocaine, 2.5–3 mg/kg, and cuff deflation after 10 minutes, blood levels have been reported to be less than 2 micrograms/ml.

If severe central nervous system intoxication occurs, appropriate resuscitation guidelines should be followed. Emergency drugs must be readily available and 100% oxygen should be administered.

**INTRAVENOUS REGIONAL ANESTHESIA OF THE LEG:**

The basic technique is the same as for the arm but the dose and volume of local anaesthetic has to be doubled for intravenous regional anesthesia of the leg, which is associated with an increased potential for local anaesthetic toxicity. The tourniquet pressure must be higher in the leg approximately 350–400 mmHg, to occlude blood flow in the femoral artery. This may increase the occurrence of tourniquet pain. Tourniquets may be applied to the thigh, two tourniquets about 9 cm wide or one at the
calf below the head of the fibula and one at the thigh. The latter is for safety in case of distal cuff failure and is not usually inflated.

**Choice of drugs**

Many local anaesthetic drugs, with or without additives, have been used for intravenous regional anesthesia, but 0.5% prilocaine, 3–6 mg/kg, is the drug of choice because it has less systemic toxicity and is partially taken up in the lungs before reaching the systemic circulation. The usual dose is 40 ml (200 mg) without epinephrine. However, the manufacturers have ceased production of 0.5% prilocaine. 1% prilocaine remains available and is licensed for intravenous regional anesthesia, though its stability is not guaranteed if diluted. If prilocaine is unavailable then 0.5% lignocaine, 3 mg/kg, is used. If intravenous regional anesthesia is applied to the leg a larger volume must be injected (up to 100 ml). Prilocaine can be used undiluted for which maximum recommended dose is 400 mg in adults but lignocaine is commonly diluted to lower concentrations (e.g. 0.2–0.25%). For a 60kg patient 9ml of 2% lignocaine diluted to 36ml is 0.5% is used for Intravenous regional anesthesia.

Prilocaine can cause methaemoglobinaemia but unless doses in excess of 600 mg are used it is clinically insignificant in most patients. Although one has to be aware that in patients with anaemia or cardiac
conditions even small amounts of methaemoglobin can significantly impair the oxygen-carrying capacity of their red blood cells. Intravenous regional anaesthesia with prilocaine in these patients should be considered carefully for its benefits.

Other local anaesthetic agents have been used but do not provide superior analgesia or more rapid onset of block. Severe toxic reactions and death have been observed with bupivacaine and its use is contraindicated. In one study, 0.2% ropivacaine was intraoperatively as effective as 0.5% prilocaine but postoperative analgesia was prolonged;

Additives to local anaesthetics have not been consistently shown to have an effect during intravenous regional anesthesia but may increase the length of postoperative analgesia, probably because of a systemic effect following tourniquet release. The reported enhancement of intravenous regional anesthesia with pethidine, 1 mg/kg, may reflect intrinsic local anaesthetic activity of the drug.

It was shown that addition of muscle relaxants produced marked muscle relaxation but did not augment analgesia.

Ketamine alone appears to provide good sensory analgesia but some patients lost consciousness and exhibited the typical features of ketamine anaesthesia after tourniquet release.
Many other drugs have been studied, but only the addition of $\alpha_2$ agonists clonidine 150 micrograms or Dexmedetomidine 0.5 micrograms/kg or the non-steroidal anti-inflammatory drugs ketorolac, 20 mg, or tenoxicam, 20 mg, to the local anaesthetic solution appeared to be effective in prolonging postoperative analgesia and relieving tourniquet pain. Guanethidine and calcium-channel blockers have been evaluated in the context of chronic pain management only.
TOURNIQUET

Intravenous regional anaesthesia is a method of producing analgesia of the distal part of a limb by intravenous injection, while circulation to the limb is occluded.

Occlusion of the limb was done previously by winding an esmarch bandage proximally up the arm. At present occlusion of the limb is achieved by pneumatic tourniquet. Unfortunately, the tourniquet is not physiologic and is associated with number of disadvantages.

SITE OF APPLICATION:

The upper arm and thigh have sufficient muscle bulk to distribute the cuff pressure evenly and they are the recommended sites.

CUFF WIDTH:

The American Heart Association concluded that if a sphygmomanometer cuff has a width of 20% greater than the diameter of the upper arm or 40% of the circumference of the thigh to a maximum of 20cm, then the pressure in the underlying central artery will be equal to that in the cuff. Modern silicone cuffs tend to be smaller than this, measuring 90mm cuff width of bladder 70mm size for the arm and 105mm cuff width of bladder 75mm size for the leg.
The tissues immediately underlying the cuff should be protected with cotton wool. This is not necessary with correctly applied modern silicone cuff

**PRESSURE:**

It is based on the unsedated patient’s blood pressure measured on the ward preoperatively. The recommended cuff pressure for the upper limb is systolic blood pressure plus 100mmHg and for lower limb twice systolic blood pressure. This higher pressure is needed because there is often not enough room above the operating site for full sized cuff.

**TOURNIQUET TIME:**

The recommended time for upper limb is 90minutes. Two hours should be regarded as a maximum but this will not be safe for all patients. The surgeon should be notified about tourniquet time every half an hour. The safest time is the shortest tourniquet time.

**PHYSIOLOGIC CHANGES CAUSED BY LIMB TOURNIQUETS:**

Neurologic effects are abolition of somatosensory evoked potentials and nerve conduction which occurs with in 30minutes, application for more than 60minutes causes tourniquet pain and hypertension, application for more than 2hours may result in
postoperative neuropraxia and evidence of nerve injury may occur at a skin level underlying the edge of the tourniquet

**MUSCLE CHANGES:**

It produces cellular hypoxia within 8 minutes, decrease in the cellular creatine level, progressive cellular acidosis, endothelial capillary leak after 2 hours and progressive coldness of limb.

**SYSTEMIC EFFECTS OF TOURNIQUET INFLATION:**

Arterial and pulmonary artery pressures become elevated, although this effect is usually slight to moderate if only one limb is occluded.

**SYSTEMIC EFFECTS OF TOURNIQUET RELEASE:**

They are transient fall in core temperature, transient metabolic acidosis, transient fall in central venous oxygen tension, transient increase in central venous carbon dioxide tension, rapid release of acid metabolites into central circulation, transient fall in pulmonary and systemic arterial pressures, transient increase in end-tidal carbon dioxide and increased oxygen consumption.

**TOURNIQUET PAIN:**

Patients receiving spinal anaesthesia may develop a poorly defined aching or burning sensation in the distal extremity about one hour after tourniquet inflation. Although the mechanism and neural pathways for this
severe aching and burning sensation defy precise explanation, unmyelinated, slow –conduction C fibres, which are relatively resistant to local anaesthetic blockade, probably play a critical role. Even during general anaesthesia, tourniquet pain can be revealed by a gradually increasing mean arterial blood pressure. The tourniquet pain and its accompanying hypertension influenced by many factors including anaesthetic techniques, intensity and level of block, choice of local anaesthetic and supplementation of the block with opioids. Cuff deflation invariably and immediately relieves the sensation of tourniquet pain and its hypertension. Systemic opioids namely inj.fentanyl 1micrograms/kg can be used in relieving tourniquet pain.
PHARMACOLOGY OF LIGNOCAINE

Lignocaine is a synthetic amide-linked anaesthetic of intermediate potency and duration. In 1943 Lofgren synthesized Lignocaine in Sweden. First used by Gordh in 1948.

Lignocaine is the standard to which all other local anaesthetics are compared. It is currently the most widely used local anaesthetic. In addition, it is a popular antiarrythmic. It can be given by all routes.

**Mechanism of action:**

Lignocaine prevent transmission of nerve impulses by inhibiting passage of sodium ions through ion-selective sodium channels in the nerve membranes. This slows the rate of depolarization such that the threshold potential is not reached and thus action potential is not propagated. But resting membrane potential is not altered. Lignocaine binds to the inner portion receptor (i.e Sodium channel) after entering the cell membrane.

**Physiochemical properties:**

- Molecular weight: 234
- Weak base with a pKa: 7.6 – 7.8
- Very stable, not decomposed by boiling, acids or alkalies
- It is less lipid soluble than that of Bupivacaine
**Pharmacokinetics:**

**Absorption:**

It is absorbed from the site of application or injection into the bloodstream. Rate of absorption depends on the blood flow to the area and use of epinephrine.

**Metabolism and Excretion:**

Metabolised in liver by oxidative dealkylation to monoethylglycine xylidide followed by hydrolysis of this metabolite to xylidide. Metabolism is dependant on hepatic blood flow. Monoethylglycine xylidide has 80% activity of the parent drug and xylidide has 10% activity of the parent drug. 75% of xylidide is excreted in the urine as 4 – hydroxyl – 2,6 – dimethylaniline.

**Onset of action:**

It has rapid onset occurring for topical anaesthesia in 5-10 minutes, for conduction anaesthesia in small nerves it is 5-10 minutes and for large nerves it is 10-15 minutes and for Intravenous administration it is 1-2 minutes.

It has protein binding of 70% bounded to α1 acid glycoproteins with volume of distribution 91 litres. Lignocaine has a triphasic distribution
Rapid distribution phase (α): In this phase, the drug is distributed to highly vascular regions with $t^{\frac{1}{2}}\alpha$ of 1 minute. Next is slow disappearance phase (β): here the drug is distributed to slowly equilibrating tissues with $t^{\frac{1}{2}}\beta$ of 9.6 min. Last is slow transformation and excretion phase (δ): Where the $t^{\frac{1}{2}}\delta$ is 1.6 hrs with clearance of 0.95 litres per minute.

**Availability:**

a) 5% heavy 2 ml ampoules which contain 50 mg of lignocaine / ml with 75 mg – 100 mg of dextrose

b) 2% lignocaine (xylocard) without preservative – 50 ml vial for intravenous use

c) 2% lignocaine – plain – 30 ml vial – contains methyl and propyl paraben as preservative

d) 2% lignocaine with 1 in 200000 Adrenaline

e) 4% lignocaine with 1 in 200000 Adrenaline – 30 ml vial.

f) 4% lignocaine viscus

g) 4% lignocaine aqueous solution

h) 10% lignocaine spray

i) 2% lignocaine Jelly

j) 2% lignocaine ointment

k) 5% lignocaine ointment
Pharmacodynamics:

Local actions:

Causes nerve blockade with loss of pain and temperature sensation, touch, motor power and vasomotor tone in the region supplied by the nerves blocked.

Systemic actions:

The results of systemic absorption from the site of administration or intravenous administration

1. Cardiovascular system:

It has a stabilizing effect on the cell membranes of cardiac tissue. Lignocaine depresses myocardial automaticity by antagonizing the spontaneous phase IV depolarization and reduces the duration of effective refractory period. Myocardial contractility and conduction velocity are depressed at higher concentrations. These effects result from direct cardiac muscle membrane changes occurring due to cardiac sodium channel blockade. It stabilizes the membrane of damaged and excitable cells, tending to suppress ectopic foci.

2. Vascular smooth muscle:

Produces vasodilatation
3. **Respiratory system:**

   Lignocaine depresses hypoxic drive (the ventilatory response to low $P_aO_2$). Apnea can result from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to the local anaesthetic agents. Relaxes bronchial smooth muscle. Intravenous lignocaine may be effective in blocking the reflex bronchoconstriction associated with intubation.

4. **Central nervous system:**

   Produces a sequence of stimulation followed by depression. Produces sedation on intravenous administration. Intravenous administration decreases cerebral blood flow and attenuates the rise in intracranial pressure that accompanies intubation. Infusion of lignocaine is capable of reducing the Minimal Alveolar Concentration of volatile anaesthetics by 40%.

5. **Musculoskeletal:**

   Lignocaine is myotoxic leading to lytic degeneration, edema and necrosis.

6. **Haematological:**

   It decreases coagulation and enhances fibrinolysis
**Indications:**

1. For infiltration block, peripheral nerve blocks, epidural, spinal and topical anaesthesia & intravenous regional anaesthesia.

2. Antiarrythmic:

   Lignocaine is a class IB antiarrythmic. It is used to treat
   
   a) Ventricular tachyarrythmias
   
   b) Arrhythmias following acute Myocardial Infarction during cardiac surgery
   
   c) In digitalis toxicity – because it does not worsen AtrioVentricular block

3. Prevention or treatment of increases in intracranial pressure during intubation - antitussive effect may be the reason.

4. Reflex induced bronchospasm is also attenuated by intravenous administration of lignocaine

5. Suppresses noxious reflexes such as coughing & sympathetic stimulations associated with endotracheal suctioning and intubation.

6. Used intravenously as an analgesic for certain chronic pain states

7. Used as a supplement to general anaesthesia.
**Contraindications:**

1. Hypersensitivity
2. Should not be used with vasoconstrictor in digits of hand, feet and penis
3. Stokes Adams syndrome, severe degree of heart block

**Doses:**

Maximum recommended dose:

a) Plain - 3 mg / kg
b) with adrenaline - 7 mg / kg
c) for reflex suppression - 1.5 mg / kg iv.

**Drug interactions:**

β Blockers:

Coadministration of beta blockers, increases serum levels of lignocaine and its toxicity by decreasing lignocaine’s metabolism.

Anticonvulsant agents:

Increases lignocaine’s metabolism

Non depolarizing muscle relaxant

Blockade is potentiated by lignocaine

Opioids and α2 adrenergic agonists:

Potentiate lignocaine’s pain relief
Antiarrythmic agents

   Potentiate the cardiac effects of lignocaine

**Toxicity:**

   Mostly due to systemic absorption of locally administered lignocaine
or due to accidental intravenous administration of large doses of
lignocaine. The central nervous system is mostly vulnerable.

**Blood levels and symptoms:**

   4 micrograms / ml      :   Light headedness, tinnitus, circumoral and
tongue numbness
                        (anticonvulsant and antiarrythmic activity)

   6 micrograms / ml      :   visual disturbances

   8 micrograms / ml      :   muscular twitching

   10 micrograms / ml     :   convulsions

   12 micrograms / ml     :   Unconsciousness

   15 micrograms / ml     :   Coma

   20 micrograms / ml     :   respiratory arrest

   26 micrograms / ml     :   cardiovascular collapse
Treatment of toxicity:

Continuous monitoring of Cardiovascular and Respiratory status helps to identify the toxicity earlier.

- If convulsions occur barbiturates or benzodiazepines can be given.
- Succinylcholine 1mg/kg to paralyse the patient and aids in controlling the seizures.
- Cardiac toxicity like fibrillation can be treated by defibrillation
- Ventilatory support – 100 % oxygenation, intubation and ventilation
- Maintain B.P. by rapid infusion of I.V. fluids, use of vasopressors and put the patient in Trendelenberg’s position.
- Maintain fluid and electrolyte balance.

Adverse effects:

1. Allergic and hypersensitivity reactions
   Due to the preservative used – methyparaben

2. Cardiovascular system:
   Bradycardia, hypotension
DEXMЕDЕTOMIDINE HYDROCHLORIDE
**PHARMACOLOGY OF DEXMEDETOMIDINE**

Dexmedetomidine hydrochloride is a 4-((s)-alpha,2,3-trimethylbenzyl)imidazole monohydrochloride or 4-[(1r)-1-(2,3-dimethylphenyl) ethyl]-3h-imidazole hydrochloride.

Molecular weight 236.74 1043

Dexmedetomidine is a new alpha2-agonist used as a short-term (less than 24 h) sedative analgesic in the intensive care unit. Clonidine is the prototype of alpha2-agonist. Dexmedetomidine when compared to clonidine is a much more selective alpha 2-adrenoceptor agonist, the alpha2/alpha1 selectivity of dexmedetomidine is 1620 and hence is 8 times more powerful alpha2-adrenoceptor than clonidine. In addition, dexmedetomidine is shorter-acting drug than clonidine and has a reversal drug for its sedative effect, atipamezole. These properties render dexmedetomidine suitable for sedation and analgesia during the whole perioperative period: as premedication, as an anesthetic adjunct for general and regional anesthesia, and as postoperative sedative and analgesic.

Alpha2-receptors are found in many sites throughout the body. Alpha2-adrenoceptors are found in peripheral and central nervous systems, in effector organs such as the liver, kidney, pancreas, eye, vascular smooth muscles and platelets. Alpha2-adrenoceptors are divided into three
subtypes; the *subtype a*, the predominant subtype in central nervous system, is responsible for the sedative, analgesic and sympatholytic effect; the *subtype b*, found mainly in the peripheral vasculature, is responsible for the short-term hypertensive response, and the *subtype c*, found in the central nervous system, is responsible for the anxiolytic effect.

**Pharmacodynamics of dexmedetomidine**

The highest densities of alpha2-receptors are found in the locus ceruleus, the predominant noradrenergic nuclei of the brainstem and an important modulator of vigilance. Presynaptic activation of the alpha2-adrenoceptor in the locus ceruleus inhibits the release of norepinephrine and results in the sedative and hypnotic effects. In addition, the locus ceruleus is the site of origin for the descending medullospinal noradrenergic pathway, known to be an important modulator of nociceptive neurotransmission.

Stimulation of the alpha2-adrenoceptors in this area terminates the propagation of pain signals leading to analgesia. Postsynaptic activation of alpha2-adrenoceptors in the CNS results in decrease in sympathetic activity leading to hypotension and bradycardia. At the spinal cord, stimulation of alpha2-receptors at the substantia gelatinosa of the dorsal horn leads to inhibition of the firing of nociceptive
neurons and inhibition of the release of substance P10. Also, the alpha2-
adrenoceptors located at the nerve endings have a possible role in the
analgesic mechanisms of alpha2-agonists by preventing noradrenaline
release.

**Pharmacokinetics of dexmedetomidine**

Dexmedetomidine, an imidazole compound, is the active d-isomer of
medetomidine. Following intravenous administration, dexmedetomidine
exhibits the following pharmacokinetic parameters: a rapid distribution
phase with distribution half-life (t ½ α) of 6 minutes; elimination half-
life (t ½ β) of 2 hours; volume of distribution (vss) 118 litres. Dexmedetomidine exhibits linear kinetics when infused in the dose range
of 0.2-0.7 micrograms/kg/h for not more than 24 hours. Dexmedetomidine
undergoes almost complete biotransformation through direct
glucuronidation and cytochrome p450 metabolism. Metabolites of
biotransformation are excreted in the urine (95%) and feces. The average
protein binding of dexmedetomidine is 94%. Dexmedetomidine is a white
powder that is freely soluble in water and pKa of 7.1

**Dosage**

It is supplied as 100 micrograms/ml 2 ml vial which must be diluted
with 48 ml of 0.9% sodium chloride solution prior to administration. For
adult patient, dexmedetomidine is administered by a loading infusion of 0.5-1 micrograms/kg over 10 minutes, followed by a maintenance infusion of 0.2 to 0.7 micrograms/kg/hr. The effect appears in 5-10 minutes, and is reduced in 30-60 minutes. The maintenance infusion is adjusted to achieve the desired level of sedation.

**Perioperative uses of dexmedetomidine**

**i – Premedication**

Dexmedetomidine possesses anxiolytic, sedative, analgesic, antisialagogue and sympatholytic properties. Intramuscular dexmedetomidine at a dose of 1 microgram/kg is used for premedication.

**ii – Intraoperative uses of dexmedetomidine**

Intraoperative uses of dexmedetomidine include its use as adjunct to general anesthesia, as adjunct to regional anesthesia, in monitored anesthesia care (MAC), or as a sole agent for total intravenous anesthesia (TIVA).

**iii – Use of dexmedetomidine in the postoperative period**

Dexmedetomidine’s special properties favor its use in the recovery room. In addition to its sympatholytic effects, analgesic effects, decreased rate of shivering and the preservation of respiratory function allows the continuation of the dexmedetomidine infusion in the extubated,
spontaneously breathing patient. The possibility of ongoing sedation and sympathetic block could be beneficial in reducing high rates of early postoperative ischemic events in high-risk patients undergoing non-cardiac surgery.

**Contraindication**

i) Pre-existent severe bradycardia and conduction problems.

ii) In patients with reduced ventricular functions (ejection fraction<30%)

iii) In patients who are hypovolemic or hypotensive.

**Adverse effects**

The common adverse effects of dexmedetomidine include hypotension, hypertension, nausea, bradycardia, atrial fibrillation, hypoxia and various atrioventricular blocks.
REVIEW OF LITERATURE

1. Addition of dexmedetomidine to lidocaine for intravenous regional anaesthesia. Esmaoglu, A.; Mizrak, A.; Akin, A.; Turk, Y.; Boyaci, A. Erciyes University Medical Faculty, Department of Anaesthesiology and Reanimation, Kayseri, Turkey.

   Esmaoglu et al tested the use of adjuncts for intravenous regional anaesthesia (IVRA) for surgical procedures in terms of their intraoperative effects and postoperative analgesia in forty patients undergoing hand surgeries. They concluded that addition of 1 micrograms kg$^{-1}$ dexmedetomidine to lidocaine in intravenous regional anaesthesia improved the quality of anaesthesia and decreased analgesic requirements, but had no effect on the sensory and motor blocks onset and regression times.


   A recent study by Memis et al found that the addition of dexmedetomidine 0.5 micrograms kg$^{-1}$ to lidocaine for Intravenous regional anaesthesia leads to significant decreases in sensory and motor...
blocks onset time, improves quality of anesthesia, postoperative analgesia without any side effects.

3. Dexmedetomidine: Clinical Application as an Adjunct for Intravenous Regional Anesthesia. Usha Ramadhyani, MD, Jason L. Park, MD, Dominic S. Carollo, MS, MD, Ruth S. Waterman, MD, Bobby D. Nossaman, MD

Intravenous regional anesthesia is limited by the development of tourniquet pain and its inability to provide postoperative analgesia. In this study it was found that adding dexmedetomidine as an adjuvant in intravenous regional anaesthesia improved block quality, prolong post deflation analgesia and decreased tourniquet pain.

4. Adding clonidine to lidocaine for intravenous regional anesthesia prevents tourniquet pain. Gentili M, Bernard JM, Bonnet F.

Gentili et al were the first to report the efficacy of clonidine in intravenous regional anaesthesia in decreasing tourniquet pain. Reuben and Sklar evaluated the safety and efficacy of administering intravenous regional anaesthesia with 1 micrograms/kg clonidine in the management of complex regional pain syndrome of the knee and found that clonidine was a useful treatment modality for its management without significant side effects.
5. Clonidine versus ketamine to prevent tourniquet pain during intravenous regional anesthesia with lidocaine. Gorgias NK, Maidatsi PG, Kyriakidis AM,

They compared the efficacy of a 1 micrograms/kg clonidine added to intravenous regional anesthesia with lidocaine to prevent tourniquet pain and found that the addition of 1 micrograms/kg clonidine to lidocaine for intravenous regional anesthesia delayed the onset of unbearable tourniquet pain and decreased analgesic consumption for tourniquet pain relief.

6. Lurie et al. evaluated the efficacy of 1 micrograms/kg clonidine added to intravenous regional anesthesia -lidocaine in decreasing the onset of severe tourniquet pain and found that it delayed the onset time.

7. Fentanyl and Dexmedetomidine premedication before intravenous regional anesthesia in minor outpatient hand surgery.

Jaakola et al. demonstrated the analgesic efficacy of dexmedetomidine in human tourniquet pain. In their study, a single intravenous dose of fentanyl and dexmedetomidine (0.25, 0.5, and 1 micrograms/kg) was administered. They found that dexmedetomidine clearly demonstrated an analgesic effect in the tourniquet pain.
8. Jaakola assessed the efficacy and safety of intravenous dexmedetomidine as a premedication before intravenous regional anaesthesia. She found that 1 micrograms/kg dexmedetomidine was an effective premedication before intravenous regional anaesthesia because it reduced patient anxiety and sympathoadrenal responses.

9. Addition of dexmedetomidine or lornoxicam to prilocaine in intravenous regional anaesthesia for hand or forearm surgery. Kol IO, Ozturk H, Kaygusuz K, Gursoy S, Comert B, Mimaroglu C. Department of Anaesthesiology, Cumhuriyet University School of Medicine, Sivas, Turkey.

   Addition of dexmedetomidine or lornoxicam to prilocaine in intravenous regional anaesthesia decreased Visual Analogue pain scores, improved anaesthesia quality and decreased analgesic requirement.

10. A systematic review of adjuncts for intravenous regional anaesthesia for surgical procedures. Andrew Choyce MBCHB FRCA and Philip Peng, MBBS FRCPC. From the Department of Anaesthesia, King’s college Hospital, Denmark Hill, London, UK and the Toronto Western Hospital, Canada.
The authors tested the use of adjuncts for intravenous regional anaesthesia (IVRA) for surgical procedures in terms of their intraoperative effects and postoperative analgesia.

They concluded that, there is good evidence to recommend Non-steroidal anti-inflammatory drugs in general and ketorolac in particular for improving post operative analgesia after intravenous regional anaesthesia. Clonidine also appears to improve post operative analgesia and prolong tourniquet tolerance. Opioids are disappointing by this route. Only 30 mg meperidine has substantial postoperative benefit but at the expense of post deflation side effects. Muscle relaxants improve motor block and aid fracture reduction.

11. An evaluation of the analgesic efficacy of intravenous regional anaesthesia with lidocaine and ketorolac using a forearm versus upper arm tourniquet. Scott s. Reuben, MD., Robert B. Steinberg, MD, PhD, Holly Maciolek, RN and Poornachandran Manikantan MD, Department of Anaesthesiology, Tuff’s University School of Medicine, Massachusetts.

In this study, they assessed the analgesic efficacy of administering intravenous regional anaesthesia by administrating lidocaine and ketorolac with either a forearm or upper arm tourniquet
for outpatient hand surgery. They concluded that forearm tourniquet intravenous regional anaesthesia with 0.5% lidocaine and ketorolac provides both a longer duration of sensory block and prolonged postoperative analgesia compared with upper arm intravenous regional anaesthesia.

12. Local anaesthetic adjuvants for neuraxial and peripheral blockade – James R. Hebl, MD, Department of Anaesthesiology Mayo – clinic, USA.

In this study, they studied about various local anaesthetic additives such as opioids, alpha – 2 agonists (clonidine), acetylcholine esterase inhibitors (neostigmine) and N methyl – D aspartate receptor antagonists (Ketamine) and Non-steroidal anti-inflammatory drugs.

They demonstrated that more effective postoperative analgesia can be achieved when Ketorolac is used in conjunction with lidocaine for intravenous regional anaesthesia. They hypothesized that more effective analgesia was obtained during intravenous regional anaesthesia administration because a higher concentration of ketorolac existed at the site of surgical trauma, where inflammatory mediator synthesis occurred.

13. Comparison of wound infiltration with ketorolac Versus intravenous regional anaesthesia with ketorolac for postoperative analgesia
following ambulatory hand surgery. - Reuben SG, Duprat MM, Tufts University school of Medicine, spring field, Massachusetts, USA.

The purpose of this study was to assess the analgesic effectiveness of ketorolac administered with lidocaine via intravenous regional anaesthesia (IVRA) or via wound infiltration following ambulatory hand surgery. They concluded that ketorolac provides similar postoperative analgesia after ambulatory hand surgery when administered with lidocaine either by intravenous regional anaesthesia or by wound infiltration.


A. Turan, B. Karamanlyog M, D. Memis, G. Kaya and Z. Paukcy ; Department of Anaesthesiology and Reanimation, Trakya University, Turkey.

In this study, thirty patients undergoing hand surgery were randomly assigned to two groups to receive intravenous regional anaesthesia. They have used 0.5 mg of neostigmine as a additive. They found shortened sensory and motor block onset times, improved quality of anaesthesia and prolonged sensory and motor block recovery times, and prolonged time to first analgesic requirement in neostigmine group.
15. 0.5 % versus 1.0% 2 – chloroprocaine for Intravenous Regional Anaesthesia: A prospective randomized, Double – Blind Trial.

Stephan C. Marsch, MD, D Phil, Mathias Sluga, MD, Wolfgang Studer, MD, Jonas Barandun, MD, Domenic Scharplatz, MD and Wolfgang Ummentioter MD, From the Departments of Anaesthesia and surgery, Switzerland.

In this randomised prospective double – blind study they tested the hypothesis that compared with 40 ml chloroprocaine 0.5 %, 40 ml chloroprocaine 1 % results in an earlier onset of analgesia duration and improves distal tourniquet tolerance during intravenous regional anaesthesia. These beneficial effects must be weighed against a fourfold increase in signs of systemic local anaesthetic toxicity.
MATERIALS AND METHODS

This is a prospective randomized double blind control study conducted at Government Rajaji Hospital attached to Madurai Medical College.

After obtaining approval from the ethical committee 60 patients of ASA grade I & II age between 20-60 years who came for upper limb surgeries lasting for less than 90 minutes were included in this study.

Patients with history of allergy to local anaesthetics, sickle cell disease, raynaud’s disease, scleroderma, local infection, Pagets disease and patients with inadequate starvation less than 6 hours and patients who had contraindication to Dexmedetomididine were excluded from this study. Preanaesthetic evaluation was done.

All patients were premedicated with Inj. Midazolam 0.15mg/kg intramuscularly 45 minutes before surgery. Resuscitation equipment and drugs were kept ready. The initial pulse rate, blood pressure and arterial oxygen saturation were recorded and then continuous monitoring was done during the procedure.

A 22 G cannula was placed intravenously as distal as possible in the arm to be anaesthetized. Venous access was established in the opposite arm to allow administration of fluids or drugs if necessary. The double
The proximal tourniquet was inflated to at least 100 mm Hg higher than the patients systolic blood pressure. Before injecting local anaesthetic, radial pulse was palpated and confirmed that there was no pulse. The local anaesthetic is then injected slowly over 90 seconds. A standard volume of 40 ml of 0.5% lignocaine or 40 ml of 0.5% lignocaine with 0.5 micrograms/kg dexmedetomidine was injected. Patients were divided into two groups according to the drug which they received. Group A patients received 40 ml of 0.5% lignocaine with 0.5 micrograms/kg Dexmedetomidine and Group B patients received 40 ml of 0.5% lignocaine and after achieving surgical anaesthesia, the distal tourniquet which overlies part of the anaesthetized arm was inflated and the proximal one was deflated. After that the surgeons were allowed to proceed.

Intraoperatively, Pulse rate, Blood Pressure, Respiratory rate, SPO2, signs of drug toxicity were monitored regularly. If patient complained of tourniquet pain (VAS >3), they were supplemented with Inj. Fentanyl 1
microgram/kg IV and intercostobrachial nerve block with local infiltration around the cuff. The cuff was not deflated until 30 minutes after local anaesthetic injection even if surgery was completed before 30 minutes and not inflated more than 90 minutes. Cuff deflation was performed in cycles with deflation / inflation times of less than 10 seconds until the patient no longer exhibits signs of systemic toxicity. Patients were observed for 30 minutes after surgery.

Intraoperatively the following parameters were noted:

1. Onset of action with sensory and motor blockade
2. Pulse rate, Blood Pressure, Respiratory rate, SPO2 were monitored regularly at 5,10,15,20,30,40,50,60,75,90 and 120 minutes
3. Duration of surgery
4. Sedation score
5. Need of Rescue analgesia
6. Side effects
7. Duration of blockade after cuff deflation both sensory & motor

Post operatively the time to first analgesic requirement was noted
RAMSAY SEDATION SCORE

1- Patient anxious and agitated or restless

2- Patient co-operative, oriented, and tranquil

3- Patient responds to commands only

4- Patient exhibits brisk response to light glabellar tap or loud auditory stimulus

5- Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus

6- Patient exhibits no response
DATA ANALYSIS

In this study totally 60 patients were included. Patients were divided into two groups according to the drug which they received. Group A patients received 40 ml of lignocaine with 0.5 microgram/kg Dexmedetomidine and Group B patients received 40 ml of 0.5% lignocaine. Data analysis was done with the help of computer using 

**Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate’s chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.
Age distribution

Group B
- 20-29 yrs: 9
- 30-39 yrs: 9
- 40-49 yrs: 7
- 50 & above: 5

Group A
- 20-29 yrs: 7
- 30-39 yrs: 9
- 40-49 yrs: 10
- 50 & above: 4

Legend:
- 20 - 29 yrs
- 30-39 yrs
- 40-49 yrs
- 50 & above
OBSERVATION AND RESULTS

Age distribution

<table>
<thead>
<tr>
<th>Age group</th>
<th>Group A</th>
<th></th>
<th>Group B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>20 – 29 years</td>
<td>7</td>
<td>23.3</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>30 – 39 years</td>
<td>9</td>
<td>30</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>40 – 49 years</td>
<td>10</td>
<td>33.3</td>
<td>7</td>
<td>23.3</td>
</tr>
<tr>
<td>50 &amp; Above</td>
<td>4</td>
<td>13.3</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100</td>
<td>30</td>
<td>100</td>
</tr>
<tr>
<td>Range</td>
<td>23 – 53 years</td>
<td></td>
<td>20-56 years</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>37.8 years</td>
<td></td>
<td>36.8 years</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>8.7 years</td>
<td></td>
<td>10.8 years</td>
<td></td>
</tr>
<tr>
<td>‘p’</td>
<td></td>
<td></td>
<td>0.6148</td>
<td>Not significant</td>
</tr>
</tbody>
</table>

Mean age of group A was 37.8 years and that of group B was 36.8 years. There was no significant difference (p = 0.6148).
Sex distribution

Group A
- MALE: 17
- FEMALE: 13

Group B
- MALE: 19
- FEMALE: 11
56.7 % of Group A and 63.3% of Group B were males. The sex distribution did not have any statistically significant difference (p > 0.05).
Weight

<table>
<thead>
<tr>
<th>GROUP</th>
<th>WEIGHT (in kgs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>52.5</td>
</tr>
<tr>
<td>B</td>
<td>52.9</td>
</tr>
</tbody>
</table>
Mean weights of the two groups of patients (52.5 kgs and 52.9 kgs) were not significantly different (p = 0.8471).
TYPE OF SURGERY

Group A

Group B

0% 20% 40% 60% 80% 100%

EXCISION  K.FX  REPAIR  SSG

12 11 4 3
## TYPE OF SURGERY

<table>
<thead>
<tr>
<th>Surgery done</th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Ganglion excision</td>
<td>12</td>
<td>40</td>
</tr>
<tr>
<td>K. Fix for # phalanx</td>
<td>11</td>
<td>36.7</td>
</tr>
<tr>
<td>Tendon Repair</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>SSG</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Ganglion excision and k wire fixation for # phalanx were the most common surgeries performed in both the groups.
SENSORY ONSET TIME

GROUP A

Sensory onset time (in minutes)

1.8

GROUP B

5.27
Sensory onset time

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sensory onset time (in minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Range</td>
<td>1-3</td>
</tr>
<tr>
<td>Mean</td>
<td>1.8</td>
</tr>
<tr>
<td>SD</td>
<td>0.76</td>
</tr>
</tbody>
</table>

| ‘p’        | 0.0001  |
|            | Significant |

The sensory onset time of Group A was $1.8 \pm 0.76$ minutes, This is significantly lower than the sensory onset time of Group B ($5.27 \pm 0.58$ minutes) with a ‘p’ value of 0.0001
Motor onset time (in minutes)

Group A: 13.63 minutes
Group B: 18.07 minutes
The motor onset time of Group A (13.63 ±1.54 minutes) was statistically significant ( p = 0.0001) from that of Group B ( 18.07 ±1.26 minutes).
DURATION OF SURGERY

![Graphic showing duration of surgery by groups A and B with durations of 46.83 and 47.43 minutes respectively.]
## DURATION OF SURGERY

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Duration of surgery (in minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Range</td>
<td>39-56</td>
</tr>
<tr>
<td>Mean</td>
<td>46.83</td>
</tr>
<tr>
<td>SD</td>
<td>5.07</td>
</tr>
</tbody>
</table>

‘p’

<table>
<thead>
<tr>
<th>0.6354</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not significant</td>
</tr>
</tbody>
</table>

Duration of surgery was similar in both the groups with no statistically significant difference.
RESCUE ANALGESIA

Group B

Group A

YES

NO

21

9

30
RESCUE ANALGESIA

<table>
<thead>
<tr>
<th>Rescue Analgesia</th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no</td>
<td>%</td>
</tr>
<tr>
<td>Yes</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>30</td>
<td>100</td>
</tr>
<tr>
<td>‘p’</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Significant

21 cases in Group B required rescue analgesia whereas not even a single patient in Group A required it. This was statistically significant (p = 0.0001).
SENSORY RECOVERY TIME

Sensory recovery time (in minutes)

GROUP A

GROUP B

18.87

4.8
SENSORY RECOVERY TIME

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sensory Recovery Time (in minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Range</td>
<td>10-22</td>
</tr>
<tr>
<td>Mean</td>
<td>18.87</td>
</tr>
<tr>
<td>SD</td>
<td>3.27</td>
</tr>
<tr>
<td>‘p’</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Sensory recovery time after the release of tourniquet was 18.87 ± 3.27 minutes in Group A and it was significantly lower at 4.8 ± 0.71 minutes in Group B.
Motor recovery time (in minutes)

GROUP A

GROUP B

25.6

2.53
MOTOR RECOVERY TIME

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Motor Recovery time (in minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td>Range</td>
<td>15 – 35</td>
</tr>
<tr>
<td>Mean</td>
<td>25.6</td>
</tr>
<tr>
<td>SD</td>
<td>3.83</td>
</tr>
<tr>
<td>‘p’</td>
<td></td>
</tr>
</tbody>
</table>

Motor recovery time was significantly longer (25.6 ± 3.83 minutes) for Group A than for Group B (2.53 ± 0.51 minutes) which is statistically significant with a ‘p’ value of 0.0001.
DURATION OF POSTOPERATIVE ANALGESIA

DURATION OF POSTOP ANALGESIA (in minutes)
DURATION OF POSTOPERATIVE ANALGESIA (VAS > 3)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Time when VAS &gt; 3 (in minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
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<tr>
<td>Range</td>
<td>280 – 490</td>
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<tr>
<td>Mean</td>
<td>416.2</td>
</tr>
<tr>
<td>SD</td>
<td>45.73</td>
</tr>
<tr>
<td>‘p’</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

VAS reached a score of 3 at $416.2 \pm 45.73$ minutes in Group A and at $11.33 \pm 0.96$ minutes in Group B. This difference was statistically significant with a ‘p’ value of 0.0001.
Mean arterial pressures for both the groups were similar at 1 minute and at 5 minutes (p > 0.05) which is not statistically significant.

<table>
<thead>
<tr>
<th>MAP at</th>
<th>MAP Value (Mean±SD) for</th>
<th>‘p’</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
<td></td>
</tr>
<tr>
<td>1 minute</td>
<td>94.9 ± 4.9</td>
<td>95.0 ± 5.7</td>
<td>0.8581</td>
</tr>
<tr>
<td>5 minutes</td>
<td>95.5 ± 3.3</td>
<td>94.8 ± 3.6</td>
<td>0.623</td>
</tr>
</tbody>
</table>
PULSE RATE

<table>
<thead>
<tr>
<th>Pulse rate at</th>
<th>Pulse rate (Mean ± SD) for</th>
<th>‘p’</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A</td>
<td>Group B</td>
<td></td>
</tr>
<tr>
<td>1 minute</td>
<td>82.7± 5.8</td>
<td>81.6 ±6</td>
<td>0.3926</td>
</tr>
<tr>
<td>5 minutes</td>
<td>82.0 ±4.7</td>
<td>82.1 ±4.9</td>
<td>0.9465</td>
</tr>
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Both at 1 minute and at 5 minutes, there was no statistically significant differences in the pulse rates between the two groups.
SEDATION SCORE

Group A

Group B

0% 20% 40% 60% 80% 100%

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Legend:
- 1
- 2
### SEDATION SCORE

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- **Range**: 1-2
- **Mean**: 1.77
- **SD**: 0.43
- **p**: **0.0001**
  - Significant

In group A, 7 cases had a sedation score of 1 and 23 had a score of 2. In group B, 30 cases had sedation score of 1.
DISCUSSION

Intravenous regional anaesthesia uses local anaesthetics administered to one particular limb by occluding the arm proximally to provide conduction blockade. It must be safe, not threatening or unpleasant to the patient, allow adequate surgical access to the operative site, and cause as little disturbance as possible to the internal homeostatic mechanisms.

Intravenous regional anaesthesia has many advantages. It is simple, reliable with rapid onset and recovery. Despite these advantages intravenous regional anaesthesia has its own limitations like lack of postoperative analgesia and tourniquet pain which causes discomfort to the patient. In this study, we attempted to eliminate these disadvantages by adding Dexmedetomidine as an adjuvant.

Comparison of results:

In this study both Group A (lignocaine with Dexmedetomidine) and Group B (lignocaine only) patients were comparable in respect of age, sex, weight and duration of surgery.

In my study, the onset of sensory and motor blockade was rapid in Group A compared to Group B. Duration of blockade after cuff
deflation, both sensory and motor has similar recovery profile. This results correlate with studies conducted by Esmaoglu, A et al.

Incidence of tourniquet pain which was assessed by supplementation during surgery was significantly less in Group A (0%) than Group B (70%) which was statistically significant and the p value is 0.0001. Similar study conducted by Memis et al shows incidence of tourniquet pain was less with when Dexmedetomidine as an additive.

The duration of post operative analgesia which was assessed by time to first analgesic requirement, in Group A is 416.2 ± 45.73 minutes and in Group B is 19.4±/- 11 minutes. The p value is 0.0001, which is highly significant. The sedation score in Group A was 1.77±0.43 and in Group B was 1.00. The p value is 0.0001

The mechanism of tourniquet pain remains unclear despite the role of A fibers and unmyelinated C fibere. Dexmedetomidine depress nerve action potentials, especially in C fibres, by a mechanism independent of the stimulation of $\alpha$-2-adrenergic receptors. This mechanism accounts for strengthening of the local anesthetic block achieved by perineural administration of the drug and could be implicated in the effect seen in this study. Finally, $\alpha$-2-adrenergic receptors located at nerve endings may have a role in the analgesic
effect of the drug by preventing norepinephrine release. In this study, it is found that dexmedetomidine, in addition to its local anesthetic effect, delayed the onset of tourniquet pain and reduced intra- and postoperative analgesic requirement.

Considering all the above said factors Dexmedetomidine in the dose of 0.5 micrograms/kg can be used as a adjuvant for intravenous regional anaesthesia with improved duration of postoperative analgesia and decreased incidence of tourniquet pain.
SUMMARY

This study was carried out at Govt. Rajaji hospital, Madurai, in 60 patients of age 20-60 years, with ASA physical status I, who underwent forearm and hand surgeries. They were divided into two groups of 30 each.

Group A : Intravenous regional anesthesia with 40ml of 0.5% lignocaine with dexmedetomidine 0.5micrograms/kg added as an adjuvant

Group B : Intravenous regional anesthesia with 40ml of 0.5% lignocaine

This study showed that

1. Dexmedetomidine when added with lignocaine in Intravenous regional anesthesia provided longer duration of postoperative analgesia than with lignocaine alone.

2. Sensory and motor onset time were decreased in Group A than Group B

3. Patient comfortability in terms of sedation is better in Group A than Group B

4. Less incidence of tourniquet pain in Group A than Group B

5. Cardiovascular stability was similar in both the groups

6. No side effects were noted in both the groups.
CONCLUSION

From the data and the statistical analysis it is concluded that when Dexmedetomidine 0.5micrograms/kg is added to lignocaine for Intravenous regional anesthesia, it provided quicker onset of sensory and motor blockade, lesser incidence of tourniquet pain, increased duration of post operative analgesia and better haemodynamic stability without any side effects.
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22. Stephan C. Marsch, MD, D Phil, Mathias Sluga, MD, Wolfgang studer, MD, Jonas Barandun, MD, Domenic Scharplatz, MD and Wolfgang Ummentioter MD, From the Departments of Anaesthesia and surgery, Switzerland. 0.5 % versus 1.0% 2 – chloroprocaine for
Intravenous Regional Anaesthesia: A prospective randomized, Double-Blind Trial.
PROFORMA

DEXMЕDEТОMIDINE AS AN АDJUVANT TO LIGNОCAINE IN INTRAVENOUS REGIONAL ANESTHESIA

Name : Age / Sex :

IP No. : Weight :

Diagnosis : ASA Risk :

Surgery :

Premedication :

Group A: Intravenous regional anesthesia with 40ml of 0.5% lignocaine with dexmedetomidine 0.5micrograms/kg added as an adjuvant

Group B: Intravenous regional anesthesia with 40ml of 0.5% lignocaine

1. Onset of action Sensory :

Motor :

2. Side effects noted :

3. Duration of surgery :
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4. Supplementation:

5. Duration blockade after cuff deflation
   - Sensory:
   - Motor:

6. Post operative:
   - Time to first analgesic requirement
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