"COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK"

By

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In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

Under the guidance of

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I hereby declare that this dissertation entitled **"COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK"** is a bonafide and genuine research work carried out by me under the guidance of **DR.VIJAY V. KATTI** Associate Professor Department of Anaesthesiology Shri.B.M.Patil Medical College , Hospital and Research Center, Vijayapur.

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DR. ANITA SANNAKKI

LIST OF ABBREVIATIONS

ASA	American Society of Anaesthesiologists
BP	Blood pressure
BT	Bleeding time
CVS	Cardiovascular system
CMS	Centimeters
CNS	Central nervous system
CT	Clotting time
ECG	Electrocardiogram
GA	General Anaesthesia
Hb	Haemoglobin
HR	Heart rate
I.P No	Inpatient number
ICU	Intensive care unit
IM	Intramuscular
IV	Intravenous
INJ	Injection
kg	Kilogram
L.A.	Local Anaesthetic
MAP	Mean arterial pressure
MIN	Minutes
mg	Milligram
mg/dL	Milli gram per deci liter
mm Hg	Milli meter of mercury
MHz	Megahertz

Mcg	Microgram
NIBP	Non invasive Blood pressure
NRS	Numerical rating scale
PR	Pulse rate
P/A	Per abdomen
RS	Respiratory system
RR	Respiratory rate
RBS	Random blood sugar
S.D	Standard deviation
SpO_2	Oxygen saturation
TC	Total count
yrs	Years

ABSTRACT

Background and objectives : Adjuncts to local anaesthetics for brachial plexus block may enhance the quality and duration of analgesia. Clonidine and Dexmedetomidine are both Alpha-2 adrenergic agonists, they are known to produce antinociception and enhance the effect of local anaesthetics when given epidurally, intrathecally or in various peripheral nerve blocks. The purpose of this study was to compare Dexmedetomidine and Clonidine with respect to their onset of sensory and motor block and duration of analgesia in upper limb surgeries..

Methods : A prospective, randomized study was conducted on 100 ASA I or II adult patients undergoing upper limb surgeries under supraclavicular brachial plexus block. Patients were randomly divided into two groups. Patients in group C (n = 50) were administered 35ml of 0.25% Bupivacaine with Clonidine 1mcg/kg and group D were administered 35ml of 0.25 % Bupivacaine with Dexmedetomidine 1mcg/kg.The onset time and duration of sensory and motor blockade were recorded. Haemodynamic variables (i.e., heart rate, noninvasive blood pressure, oxygen saturation), sedation scores and rescue analgesic requirements were recorded for 24 hrs postoperatively.

Results : The onset of sensory and motor block was significantly faster in group D compared to group C ($P < 0.05$). Duration of analgesia was significantly prolonged in group D compared to group C. Haemodynamics and sedation scores did not differ between groups in the post-operative period.

Conclusion : The faster onset of sensory and motor block is seen in Dexmedetomidine compared to Clonidine. Duration of analgesia is significantly prolonged in Dexmedetomidine compare to Clonidine.

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INTRODUCTION.

"For all the happiness mankind can gain is not in pleasure but in rest from pain"- John Dryden. Pain is an inevitable consequence of surgery. Surgical intervention to reduce human suffering is associated with pain and distress to patients.

Severe postoperative pain may have physiological consequences increasing the stress response to surgery, seen as a cascade of endocrine-metabolic and inflammatory events that ultimately may contribute to organ dysfunction, morbidity, increased hospital stay and mortality. Besides, restlessness caused by severe pain may contribute to postoperative hypoxemia[1].

Effective pain controlis essential for optimal care of surgical patients, especially in the postoperative period. Pain relief after surgery can be achieved by various regional anaesthetic techniques. Fundamental to modern neural blockade is the concept that pain is a sensory warning conveyed by specific nerve fibre, amenable in principle, to modulation or interruption anywhere in the nerve's pathway[2].

Upper limb surgeries are mostly performed under peripheral blocks such as the brachial plexus block. Peripheral nerve blocks not only provide intraoperative anaesthesia but also extend analgesia in the post-operative period without any systemic side-effects[3].

The brachial plexus block is one among the most popular regional nerve blocks performed for upper limb surgeries. Brachial plexus block provides a useful alternative to general anesthesia for upper limb surgeries. They achieve near ideal operating condition by producing complete muscular relaxation, maintaining stable intraoperative hemodynamics and the associated sympathetic block. The sympathetic block decreases

post-operative pain, vasospasm and edema. Supraclavicular approach for brachial plexus block is most commonly suitable for upper limb surgeries. The acceptance of regional anesthesia techniques has been limited by two major factors inherent in the local anaesthetic agents available for the blockade namely slow onset time, short duration of action and limited duration of postoperative analgesia. Sometimes the duration of blockade falls short of the total duration of the surgery incase of long surgical procedures resulting in the need to convert the procedure to general anaesthesia..Different adjuncts have been tried to fill the lacunae created by the local anaesthetics. There has always been a search for adjuvants to the regional nerve block with drugs that prolong the duration of analgesia but with lesser adverse effects.Alpha-2 adrenergic receptor agonists have been the focus of interest for their sedative, analgesic, peri operative sympatholytic and cardiovascular stabilizing effects with reduced anaesthetic requirements. They are mixed with local anaesthetic agents to extend the duration of spinal, extradural and peripheral nerve blocks. [4][5][6] The concurrent injection of alpha 2 adrenergic agonist drugs has been suggested to improve the nerve block characteristic of local anesthetic solutions. The search for the ideal additive continues, and led us to try the α2 adrenergic agent, Dexmedetomidine and Clonidine.

Our study is designed to compare Clonidine and Dexmedetomidine, used as an adjunct to Bupivacaine in supraclavicular brachial plexus block for upper limb surgeries, in terms of efficacy in onset, duration, potency of sensory, motor block, sedation score and analgesia.

AIMS & OBJECTIVES OF THE STUDY

AIMS:

To compare efficacy of Clonidine and Dexmedetomidine as an adjuvant to local anaesthetic agent in supraclavicular brachial plexus block with respect to onset and duration of sensory and motor block, potency and duration of analgesia.

OBJECTIVES:

The objectives of this study are to compare the efficacy of Clonidine and Dexmedetomidine when added to a local anaesthetic solution for supraclavicular brachial plexus block for upper limb surgeries with respect to

- Onset of sensory and motor blockade
- Duration of sensory and motor blockade
- Duration of postoperative analgesia
- Efficacy and potency of analgesia
- Sedation score intra and postoperatively
- Hemodynamic variables (HR, BP, SPO_2)
- Untoward side effects, if any.

EVOLUTION OF SUPRA CLAVICULAR BLOCK

Cocaine was the first local anaesthetic extracted from Erythroxylon coca by Niemann.[7] Carl Koller later brought into light the anaesthetic properties of Cocaine by desensitizing the frog's and rabbit's cornea with Cocaine.

Halsted and Hall(1895)[8,9]

Performed the first regional anaesthetic procedure and indeed performed the first operation under brachial plexus block when he freed the cords and nerves of the brachial plexus after blocking cervical roots in neck with cocaine solution.

Crile (1897) [10]

Blocked the plexus by supraclavicular approach using combination of local subcutaneous anaesthesia with injection at the plexus after exposure by dissection.

Hirschel (1911) [11]

Injected the brachial plexus blindly. He directed 2.5 inch needle from axilla passing it in front of plexus and behind the clavicle

Kulenkampff (1912)

Mastered blind supraclavicular approach of brachial plexus block.

Pitkins (1927)[11]

Modified Hirschel's approach using 6-8inch needle to reach transverse process of 6^{th} and 7^{th} cervical vertebrae from the axilla.

Bonica (1953)[12]

Listed Pneumothorax as the complication of importance with average incidence of 0.6% in Supraclavicular brachial plexus block.

J.M.Ritchie, Brend Ritchie and Paul Greengard (1965) [13]

Described the active structure of local anaesthetics.

REVIEW OF LITERATURE

Swami S S*et al*., compared Clonidine and Dexmedetomidine as an adjuvant to local anaesthetic agent in supraclavicular brachial plexus block with respect to onset and duration of sensory and motor block and duration of analgesia. Sixty ASA I and II patients scheduled for elective upper limb surgeries under supraclavicular brachial plexus block were divided into two equal groups in a randomized, double-blinded fashion. Group c received Clonidine 1 mcg/kg and Group d received Dexmedetomidine 1 mcg/kg added to Bupivacaine 0.25% (35 cc). Onset and recovery time of sensory and motor block, duration of analgesia and quality of block were studied in both the groups. They concluded that Dexmedetomidine when added to local anaesthetic in supraclavicular brachial plexus block enhanced the duration of sensory and motor block and also the duration of analgesia. The time for rescue analgesia was prolonged in patients receiving Dexmedetomidine. It also enhanced the quality of block as compared with Clonidine[6.]

Singelyn FJ *et al*., 1 conducted a study to evaluate the effects of Clonidine added to Mepivacaine on the duration of anaesthesia and analgesia after axillary brachial plexus block. They concluded that adding 150 mcg of Clonidine to Mepivacaine for brachial plexus block prolongs the duration of anaesthesia and analgesia and suggested that this effect of Clonidine is local rather than systemic[14].

Kapral S, Krafft P, Eibenberger K, Fitzgerld R, Gosch M, Weinstabl C. Conducted study on ultrasound guided supraclavicular approch for regional anesthesia of the brachial plexus block. Fourty patients undergoing surgery of the forearm and hand were selected to investigate use of ultrasonic cannula guidance for supraclavicular

brachial plexus block. Patients were randomized into groups (supraclavicular para vascular approach; n=20) and group a axillary approach, n=20 plexus block was performed using 30 ml Bupivacaine 0.5% Onset of sensory and motor block of the radial ,ulnar and median nerves was recorded in 10 min intervals for 1h) they concluded that complete sensory block of the radial, median and ulnar nerves was attained after an average of 40 min without a significant difference between the two groups. They concluded that ultrasound guided puncture for supraclavicuar block combines the safety of axillary block with the larger extent of block provided by the supraclavicular approach.[15.]

Vincent W. S. Chan, AnahiPerlas, Regan Rawson, RN, Olusegun Odukoya conducted study on ultrasound-guided supraclavicular brachial plexus block. Forty patients were undertaken for study. Ultrasound imaging was used to identify the brachial plexus before the block, guide the block needle to reach a target nerves and visualize the pattern of local anesthetic spread. For the first 29 patients, a Toshiba Core Vision Pro unit (Toshiba Corp., Tokyo, Japan) equipped with a linear 8-MHz probe was used. For the remaining 11 patients, a Philips ATL HDI 5000 Sono CT unit (Philips Medical Systems, Bothell, WA) equipped with a linear 5- to 12-MHz probe, colour Doppler, and compound imaging capability was used. They concluded that ultrasound guidance for supraclavicular brachial plexus block is clinically useful for accurate nerve localization and to minimize the number of needle attempts.[16]

Memis D, Turan A, Karamanlioglu B, PamukcuZ ,Kurt I conducted study on intravenous regional anaesthesia(IVRA) by adding Dexmedetomidine to Lidocaine. Thirty patients undergoing hand surgery were randomly assigned to 2 group to receive IVRA. They received 40 ml of 0.5% Lidocaine and either 1 ml of isotonic saline (group L, n=15) or 0.5 ug/kg Dexmedetomidine (group LD, n=15). Sensory and motor block onset and recovery times and anaesthesia quality were noted. Before and after the tourniquet application at 5, 10, 20, and 40 min, hemodynamic variables, tourniquet pain and sedation and analgesic use were recorded. They concluded that shortened sensory and motor block onset times, prolonged sensory and motor block recovery times, prolonged tolerance for the tourniquet, and improved quality of anaesthesia were found in group Dexmedetomidine[17].

Esmaoglu A, Yegenoglu F MD, Akin A, Yildirim C conducted study onthe quality of Dexmedetomidine added to Levobupivacaine to prolong axillary brachial plexus block. The primary endpoints were the onset and duration of analgesia. Sixty patient scheduled for elective forearm and hand surgery into two groups in a randomised double blind study. They concluded that by adding Dexmedetomidine to Levobupivacaine shortens the time and prolongs the duration of postoperative analgesia[18].

Singh S *et al*.,compared the effects of Clonidine added to Bupivacaine with Bupivacaine alone in supraclavicular brachial plexus block and observed the side-effects of both the groups. In this prospective, randomized, double-blinded, controlled trial, two groups of 25 patients each were investigated using (i) 40 ml of Bupivacaine 0.25% plus 150 mcg of Clonidine and (ii) 40 ml of Bupivacaine 0.25% plus 1 ml of normalsaline

0.9% , respectively. The onset of motor and sensory block and duration of sensory block were recorded along with monitoring of heart rate, non-invasive blood pressure, oxygen saturation and sedation. It was observed that addition of Clonidine to Bupivacaine resulted in faster onset of sensory block, longer duration of analgesia (as assessed by visual analogue score), prolongation of the motor block (as assessed by modified Lovett Rating Scale), prolongation of the duration of recovery of sensation and no association with any hemodynamic changes (heart rate and blood pressure), sedation or any other adverse effects. These findings suggest that Clonidine added to Bupivacaine is an attractive option for improving the quality and duration of supraclavicular brachial plexus block in upper limb surgeries[19].

Trifa *et al*., studied the addition of Clonidine to Ropivacaine in children by axillary brachial plexus block. Children aged 1-6 were randomly allocated to receive block with Ropivacaine 0.2% 0.4ml/kg plus saline in 1 ml or Ropivacaine 0.2% 0.4 ml/kg plus Clonidine 1 mcg per kg in 1 ml. They concluded that Ropivacaine alone provides sufficient analgesia and the addition of Clonidine does not improve overall postoperative analgesia but may increase the time for first analgesia required[20].

Agarwal S, Agarwal R, Gupta P have conducted study on by adding Dexmedetomidine prolongs the effect of Bupivacaine in supraclavicular brachial plexus block .Fifty patients posted for upper limb surgeries were enrolled for a prospective, randomised, double blind placebo-controlled trail. Patients were divided into two groups, the control group s and the study group sd. In group s(n=25) 30 ml of 0.325% Bupivacaine+1ml normal saline; and in group sd(n=25), 30ml 0.325% Bupivacaine+ 1ml (100mcg) Dexmedetomidine were given for supraclavicular brachial plexus block using

peripheral nerve stimulator. Onset and duration of sensory and motor blocks were assessed along with the duration of analgesia sedation and adverse effects. Hemodynamic parameters HR, systolic BP and diastolic BP were also mentioned Dexmedetomidine added as an adjuvant to Bupivacaine for supraclavicular brachial plexus block significantly shortens the time and prolong the duration of sensory motor block and duration of analgesia[21].

KirubaharR, Sundari B, Kanna V, Murugadoss K had conducted study on comparision of Clonidine and Dexmeditomidine as an adjuvant to Bupivacaine in supraclavicular brachial plexus block for upper limb for orthopedic procedure. Sixty patients were undertaken for the study, divided into two groups thirty each. group c received 35ml of 0.375% Bupivacaine and Clonidine 2 mcg/kg while group d received 35ml of 0.375% Bupivacaine and Dexmeditomidine 2 mcg/kg. The onset for sensory block and motor block in group D was lower when compared to group c. The mean time for total duration of sensory block and motor block was more in group d when compared to group c. Total duration of analgesia was higher in group d than in group c.[22]

Lee M J, Koo D J, Choi Y S, Lee K C, Kim H Y conducted a study on Dexamethasone or Dexmedetomidine as local anasethetic adjuvants for ultrasound-guided axillary brachial plexus blocks with nerve stimulation.Fifty one patients were undertaken for the study, divided into 3 groups 17 each. Group c received 20ml of 0.5% of Ropivacaine and 2ml of isotonic saline, while group d received 20ml of 0.5% Ropivacaine with 2ml(8mg) of Dexamethasone and group dm received 20ml of 0.5% Ropivacaine with 2ml(100mcg) of Dexmeditomidine a nerve stimulation technique with ultrasound was used in all the patients. The duration of sensory block was extended in

group d and group dm compared with group c($p<0.05$) but, there was no significant difference between group d and dm. Howevr there was no signifiacnt difference in onset time in all the 3 groups.[23]

WaindeskarV, Bhatia K, Garg S, Kumar J ,Songir S, Singla V conducted a study on Dexmedetomidine as an adjuvant to Levobupivacaine in ultrasound guided supraclavicular brachial plexus block. Sixty patients were included in the study divide into two groups thirty each. In group b 30ml of 0.325% Levobupivacaine and normal saline, In group bd 30ml of 0.325% of Levobupivacaine and 1mcg/kg Dexmedetomidine were given for ultrasound guided supraclavicular brachial plexus block. Dexmedetomidine added as an adjuvaint to Levobupivacaine for supraclavicular brachial plexus block significantly shortens the onset time and prolong the duration of sensory and motor block and duration of analgesia.[24]

THE ANATOMY OF BRACHIAL PLEXUS

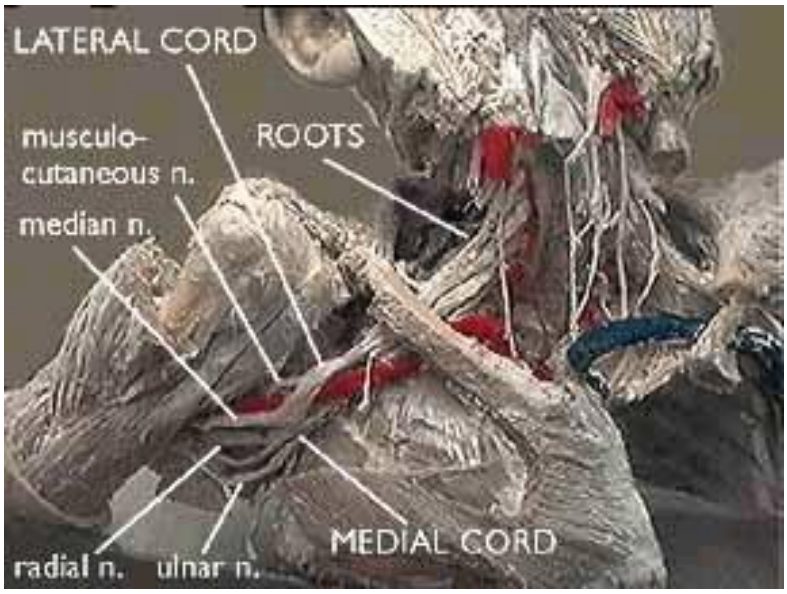


Fig.1. THE ANATOMY OF BRACHIAL PLEXUS

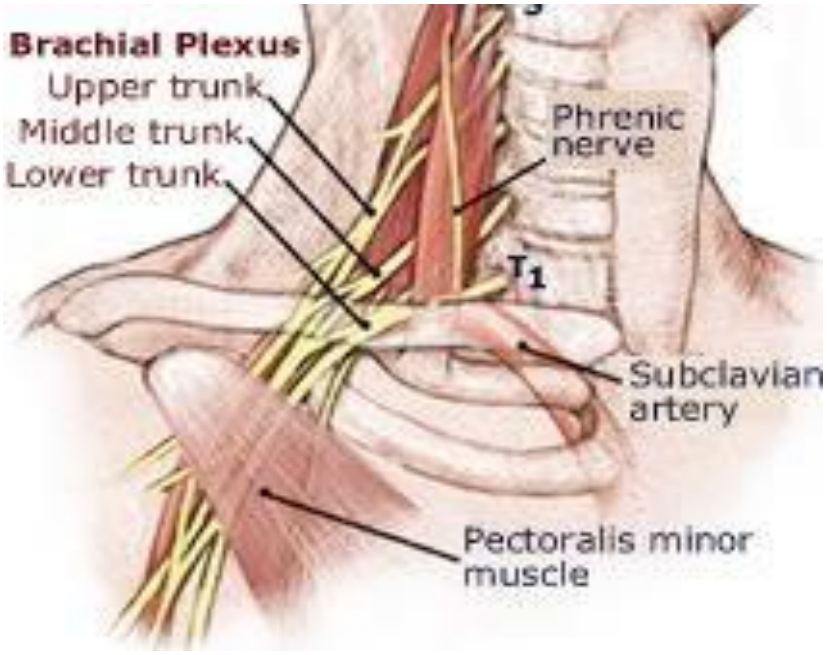


Fig.2. OUTLINE OF BRACHIAL PLEXUS AND ITS RELATIONSHIP

BRACHIAL PLEXUS –THE ANATOMY [25]

Brachial plexus provides the motor innervation and nearly all sensory supply of the upper limb. The plexus is formed by the anterior primary rami of fifth, sixth, seventh, eighth cervical and first thoracic nerves. Sometimes the plexus is derived mainly from fourth to eighth cervical nerve (prefixed plexus) or from sixth cervical nerve to second thoracic nerves (post fixed plexus). The components are designated according to their location as roots, trunks, divisions, cords and branches. Roots after emerging from intervertabral foramina unite to form trunks between scalene muscles. Each trunk divides into anterior and posterior divisions. The divisions in combination form cords which surrounds the axillary artery.

Fig.3. FORMATION OF BRACHIAL PLEXUS

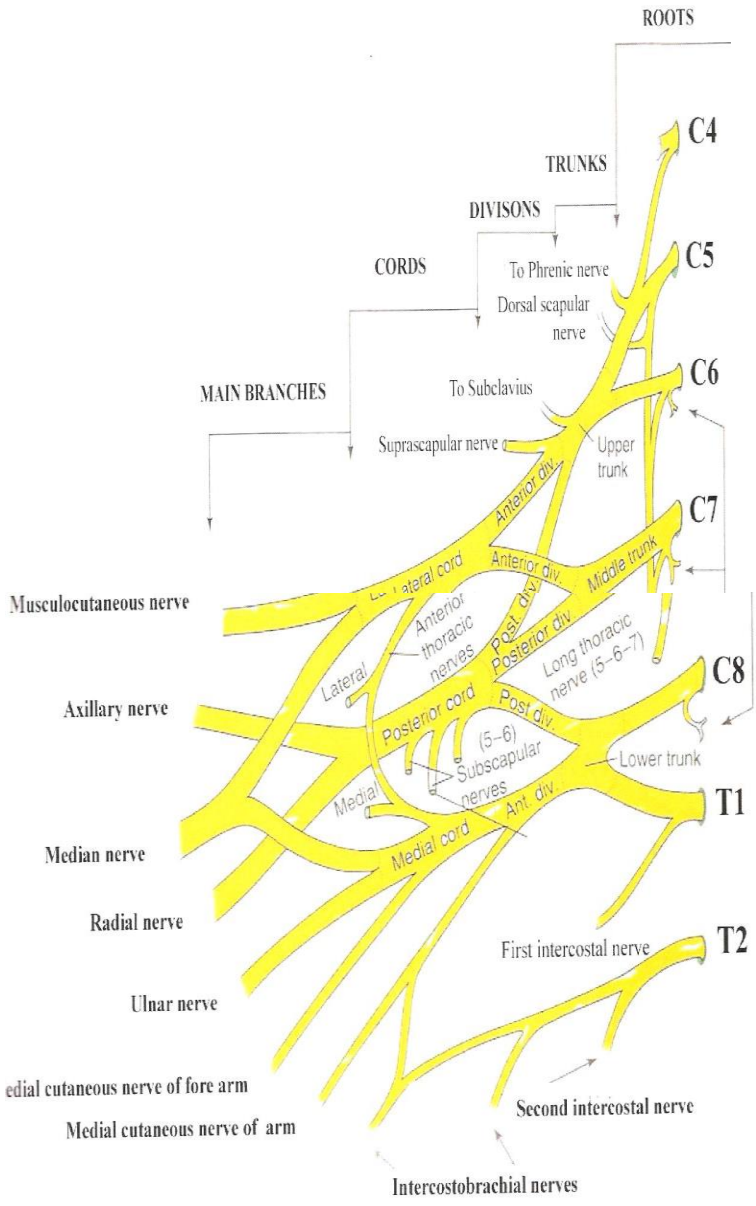


TABLE NO. 1

COMPONENTS OF THE BRACHIAL PLEXUS

Roots	Five	From anterior primary rami of spinal Nerves C5 to T1
Trunks	Three	Upper from C5 and C6 roots Middle from C7 root Lower from C8 and T1 roots
Divisions	Six	Each trunk divides into anterior and Posterior divisions.
Cords	Three	Formed by fusion of divisions Lateral- by anterior divisions of upper and middle trunk Medial- by anterior division of Lower trunk only. Posterior- by posterior divisions Of all the three trunks

TABLE NO.2 BRANCHES OF BRACHIAL PLEXUS

SUPRACLAVICULAR BRANCHES. These arise from roots or trunks

From Roots	1.Nerves To Scalene Muscles	C5,6,8
	2. Dorsal Scapular Nerve	C5
	3. Long Thoracic Nerve	C5
From Trunks	1.Nerve To Subclavius	C5, 6
	2. SuprascapularNerve	C5, 6

INFRACLAVICULAR BRANCHES .They arises from the cords,

Lateral Cord.	1.Lateral Pectoral Nerve	C5, 6, 7
	2 MusculocutaneousNerve	C5, 6, 7
	3.LateralRoot Of Median Nerve.	C5, 6, 7
Medial Cord	1.Medial Pectoral Nerve	C8, T1
	2.Medial Cutaneous Nerve Of Arm	C8, T1
	3.Medial Cutaneous Nerve Of Forearm	C8, T1
	4.Ulnar Nerve	C7,8,T1
	5.Medial Root Of Median	C8,T1
Posterior Cord	1.Upper Subscapular Nerve	C5,6
	2.Lower Subscapular Nerve	C5,6
	3.ThoracodorsalNerve	C6,7,8
	4.Axillary Nerve	C5,6
	5.Radial Nerve	C5,6,7,8,T1

SUPRA CLAVICULAR BRACHIAL PLEXUS BLOCK

INDICATIONS

Supraclavicular brachial plexus block produces rapid, reliable anaesthesia for surgical procedures of upper extremity.

Areas blocked are – Arm, forearm and hand except area over tip of shoulder (C3,C4) and inner aspect of upper arm (T2, intercostobrachial nerve).

CONTRAINDICATIONS

- General – patient refusal, hypersensitivity to local anesthetics, disorder of hemostasis, preexistent neurologic deficit, respiratory failure, infection at site.
- Specific-particular stature(short neck, stiffneck)
- Associated disease – goiter.

Radiotherapy sequel, past history of cervical node resection, contralateral recurrent laryngeal nerve palsy.

USG Technique

SONOANATOMY OF BRACHIAL PLEXUS[26]

The brachial plexus can be scanned in supraclavicular area using a high frequency 5-14 MHz linear ultrasound probe held in an oblique plane either coronal or sagittal which scans both in longitudinal and horizontal direction

The prominent landmark is subclavian artery which is identified immediately

superior to first rib as a pulsatile hypoechoic tennis ball like image on ultrasound. The first rib can be viewed as a bright hyperechoic rim with a drop out bony acoustic shadow. The brachial plexus is normally seen superior, supero-lateral or superomedial to subclavian artery as multiple hypoechoic areas often described as a honeycomb pattern or "bunch of grapes". The brachial plexus may acquire a triangular, linear or vertical (or oblique) arrangement of trunks/division/cords around subclavian artery in supraclavicular region on ultrasound

The pleura appears as a hyperechoic line at same level as more "shiny" than the rib. Movement of the pleura can be seen with respiration. The hyperechoic pleural shadow does not have a drop out acoustic shadow, which differentiates it from the rib shadow. The scalenus anterior and medius muscles is seen as hypoechoic structures on ultrasound scan which can be followed commencing from their origin to the point of insertion on first rib. The phrenic nerve lies on anterior surface of scalenus anterior muscle from C4-C7 level in the neck. The dorsal scapular and long thoracic nerves pass through scalenus medius muscle and appear as hypoechoic structures.Sometimes part of brachial plexus passes through scalenus anterior or medius muscles and can be seen as small, round or oval hyperechoic or hypoechoic structures.

The thyrocervical trunk and transverse cervical artery appears similar to the nerve trunks on ultrasound view. The pulsations of smaller arteries are masked by the strong pulsations of subclavian artery. These vessels may fall in the field of nerve block needle trajectory or course along or through the brachial plexus. This poses a risk of vascular injury, hematoma formation or inadvertent intra-vascular injection. Colour Doppler helps in differentiation of brachial plexus from arteries by demonstrating colour enhancement.

Thus ideally the proposed nerve block needle trajectory should be scanned with color flow doppler. In addition, veins are collapsible and can be identified by applying and releasing pressure with help of ultrasound probe while scanning.

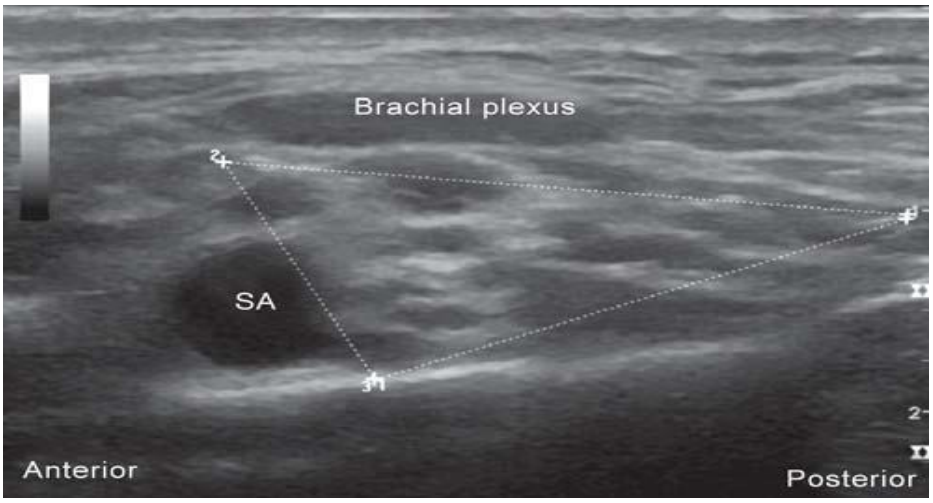


Fig. 4a: Supraclavicular brachial plexus arranged in triangular pattern.Plexus is seen as rounded to oblong hypoechoic structures surrounded by hyperechoic rim. Subclavian artery (SA) is seen as large rounded hypoechoic structure which is pulsatile in real time.

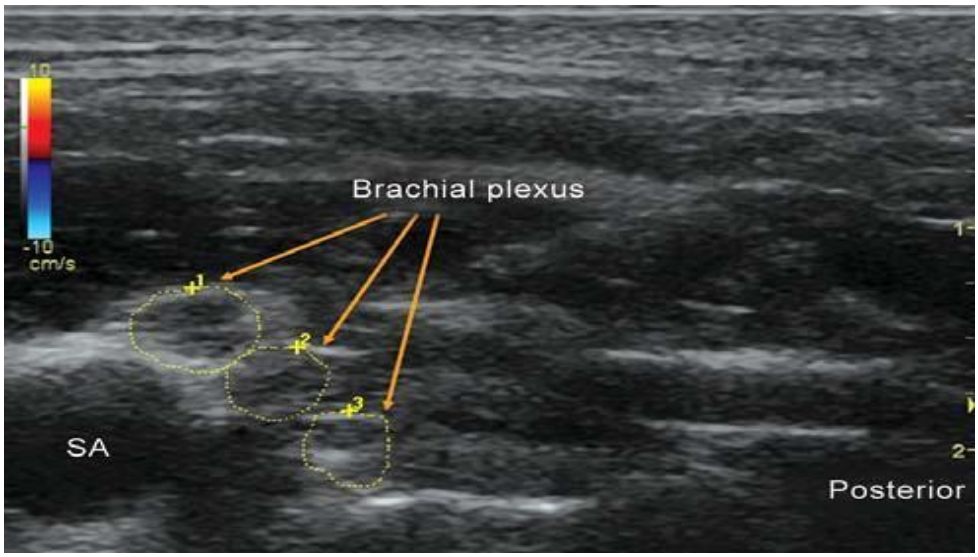


Fig. 4b: Supraclavicular brachial plexus arranged as vertical/obliquelyarranged circles. Plexus is seen as hypoechoic round structure

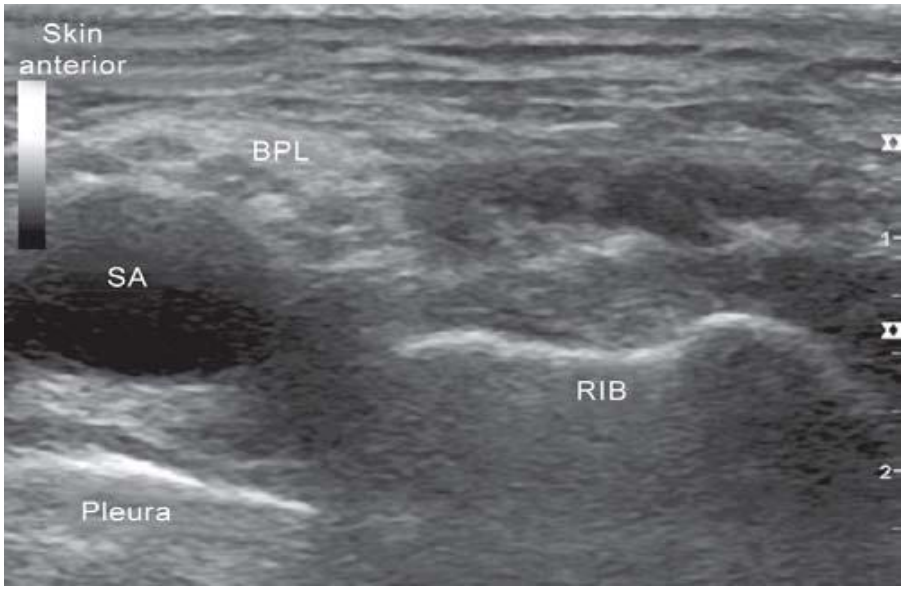


Fig. 4c: Showing pleura and rib. Rib is seen as linear hyperechoic area with acoustic shadowing, pleura is visualised as hyperechoic structure without acoustic shadow. SA-subclavian artery; BPL- brachial plexus.

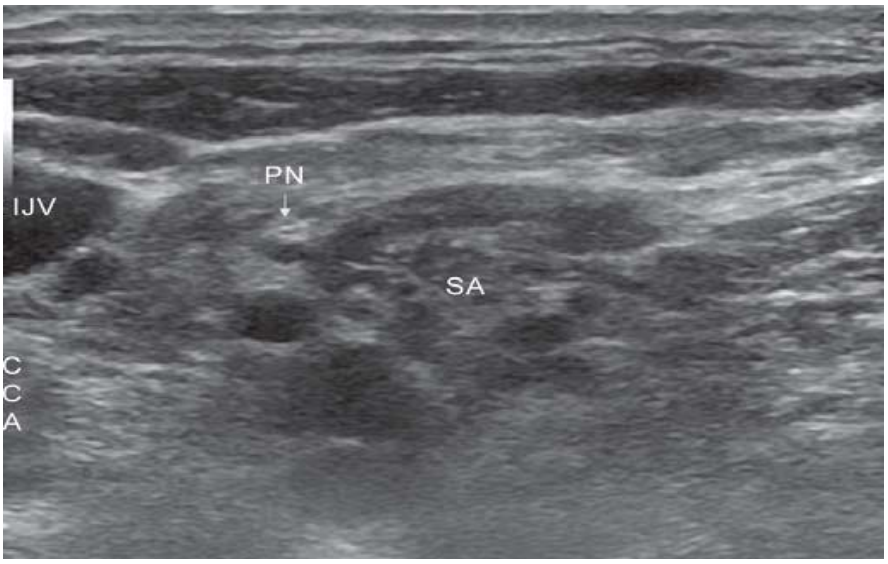


Fig. 4d: Showing phrenic nerve -PN as hyperechoic structure on anterior surface of scalenus anterior -SA muscle. IJV- internal jugular vein; CCA- common carotid artery

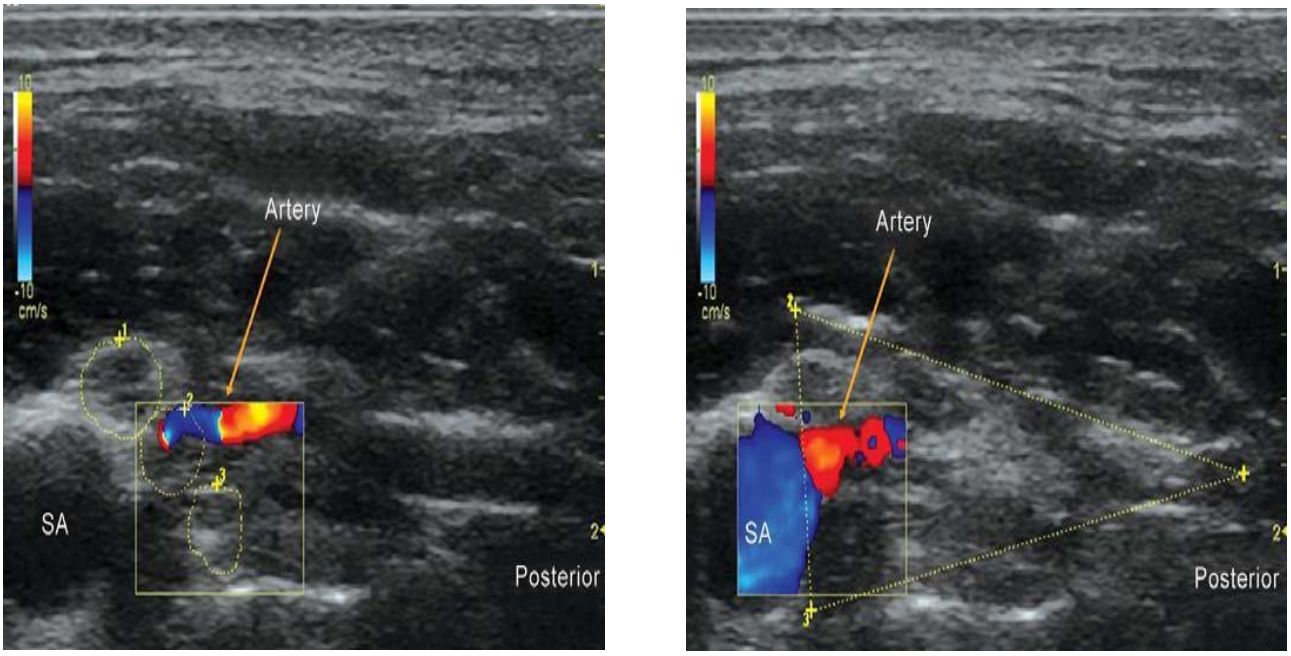


Fig 4e: Showing a branch of subclavian artery coursing through brachial plexus on colour doppler. SA-subclavian artery.

COMPLICATIONS [27]

Vascular Puncture

Internal jugular vein may be punctured at skin wheal infiltration. Simple digital compression is required before continuing, the likelihood of arterial puncture implies not to pinpoint behind and too medial from mid clavicle. Best is to withdraw and redirect the needle when perceiving artery pulsation at the needle tip.

Pleural Puncture

The most significant complication of supraclavicular approach for blocking brachial plexus is development of pneumothorax. The incidence of pneumothorax is one percent with this technique and much higher in inexperienced hands. A pneumothorax must be suspected when there is dyspnea, cough or pleuritic chest pain but the diagnosis can be confirmed only by chest x-ray.

Phrenic Nerve Block

Phrenic nerve block occurs in 40-60% of patient because of spread of local anaesthetic to the anterior surface of anterior scalene muscle. The effect is avoided if anaesthetic is deposited deep on the middle trunk on division or cord. This is rarely symptomatic. Radiographic confirmation may be obtained.

Recurrent Laryngeal Nerve Block

It causes transient dysphonia, occurs in 1% of case and only on the right side because recurrent laryngeal nerve loops around the subclavian artery on the right side and arch of aorta on the left.

Nerve Damage Or Neuritis

It results from the needle trauma or faulty positioning of anaesthetised arm peri operatively. Other remote causes include excessive tourniquet time, concentrated solution with vasoconstrictor and susceptible host tissue.[27]

Horner's Syndrome

It consists of ptosis, miosis, anhydrosis and enophthalmos. It usually follows stellate ganglion block. It is found in 10% of cases, after interscalene block.

Toxic Reaction To Drug

It is likely to occur if there is over dosage of drug or inadvertent intravascular injection is made, but can be avoided with proper negative aspiration test before drug injection.

MECHANISM OF LOCAL ANAESTHESTIC ACTION [28]

Local anaesthetics block impulses by interfering with the function of sodium channels. The diffusion of deposited local anaesthetic drug molecule near the nerve is a function of tissue binding, removal by the circulation, and local hydrolysis of the amino ester anaesthetics. The net result is the penetration of nerve sheath by the remaining drug molecules. Only about 5% of injected dose actually penetrates into the nerve.

Local anaesthetic molecules then permeate the nerves axon membrane and equilibrate there in the axoplasm. The speed and extent of these processes depend on particular drugs pKa and on the lipophilicity of its base and cation species

Binding of local anaesthetics to the sites on voltage-gated sodium channels prevents opening of channels by inhibiting conformational changes that underlie channel

activation. During onset of and recovery from local anaesthesia impulse blockade is incomplete, and partially blocked fibers are further inhibited by repetitive stimulation, which produces an additional use dependent or frequency dependent binding to Na+ channels. In simple, local anaesthetics shift sodium channels towards an "inactivated" state which cannot be directly opened by stimulation

Local anaesthetics inhibit the stimulated channels (Phasic block) more than resting channels (tonic block). Modulated receptor hypothesis has been proposed to account this. The hypothesis rests on the notion that sodium channels normally respond to membrane depolarization by passing through defined conformational states, beginning at rest , activating through the closed intermediate form to reach an open (o) form, and then closing to an inactivated (in) form/state. According to the hypothesis, local anaesthetics have higher affinity for open and especially, inactivated Na+ channels than for resting channels

Each membrane has peak sodium current that is five to six times greater than that necessary to initiate an action potential; this is the safety factor of conductance. Local anaesthetics reduce this safety factor by progressively interrupting sodium channel excitability and when it reaches zero, conduction fails. Impulse extinction is the probability that an arriving impulse will stop at a given area of a nerve. It is related to local anaesthetic dose and concentration, length of nerve fiber exposed to local anaesthetic and by the presence or absence of myelination.

Variability in the onset of conduction block in different fibers within the same nerve is referred to as temporal onset. It is related to fiber size, spatial relationship within

the nerve fiber and concentration of injected local anaesthetic. The larger the fiber, the slower is the onset of block and higher the concentration of the anaesthetic is required to achieve the same frequency of impulse extinction.

PHARMACOLOGY OF BUPIVACAINE[29,30,31,32]

Bupivacaine is an amide type of local anaesthetic which was synthesized in Sweden in 1957 by Ekenstam and his colleagues and used clinically by L.J Telivuo in 1963. It is represented as (1-butyl 2-piperidyl). Its chemical structure is given below:

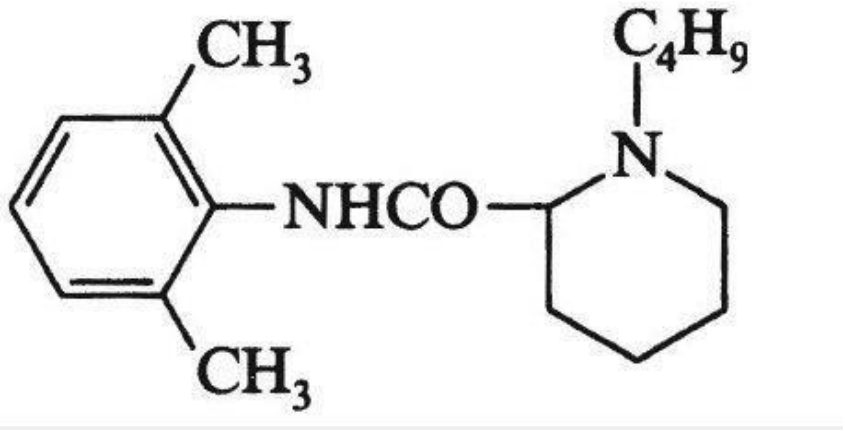


Fig 5 Structure Of Bupivacaine

Chemical formula: I- n- butyl- DL- piperidine- 2- carboxy acid-2-b-dimethyl anilide hydrochloride. (l- butyl- 2 piperidyl) Bupivacaine is a tertiary amine (a base) attached in an aromatic ring by amide linkage. The aromatic ring system gives lipophilic character to its molecule, whereas the tertiary amine end is relatively hydrophilic

PHYSICOCHEMICAL PROPERTIES:

1) **Molecular Weight (Chloride):** 324

2) **Molecular Weight (Base):** 288

3) **Protein binding capacity** is 85 to 95% in adults, neonates – 50 to 70%

4**) pKa** -value at 25 degree is 8.1

5) **Half Life-** Adults – 8hrs, neonates – 9hrs

6) **Melting Point**– 2470c

7) **Specific Gravity** – 1.035 – 1.040

8**) Partition Coefficient**– 27.5

9) **PH** – 4.6-6

Bupivacaine is a base and is a highly lipid soluble but its hydrochloride salt is soluble in water. Lipid solubility is important in redistribution of the drug and primary determinant in its intrinsic local anaesthetic potency. It has pKa of 8.1 and is largely unionized at higher pH and freely diffuses across cell membranes. Chemically it is highly stable and can be boiled for several hours in both, strongly alkaline or acidic media.

MECHANISM OF ACTION:

The primary action is on the cell membrane where it produces electrical stabilization. The large transient increases in the permeability of impulse are prevented, thus the resting potential is maintained and depolarization in response to stimulation is inhibited. The principal action of Bupivacaine is to prevent the conduction of nerve impulse by inhibiting the passage of Na+ ions through ion selective Na+ channels in nerve membranes. This in turn impedes the depolarization of the nerve membrane such that the threshold potential for propagation of action potential is not reached. Local anaesthetic solutions when deposited near a nerve which is their principal site of action; permeate the nerves axonal membrane and reside in the axoplasm. The speed and extent

of this process depends on particular drug‟s pKa and lipophilicity of its base and cation species. Only the unionized form can penetrate the cell membrane. However, cations are the active form of the local anaesthetic molecule and it acts on the interior of the cell membrane. Hence carbonated local anaesthetics have an enhanced effect and less evidence of tachyphylaxis. Binding of local anaesthetic to sites on voltage gated sodium channels inhibits the conformational changes that underlie channel activation. Local anaesthetics bind in the channel's pore and also occlude the path of sodium ions.

In order to act, local anaesthetic molecules must first penetrate the surrounding tissues and the nerve sheath. Therefore Sensitivity of different fibres to be blocked by local anaesthetics is different. And within any one fibre, there is a tendency for smaller, slower conducting fibers to be more readily blocked than the large and fast conducting fibres. The order of sensitivity to blockade is pre-ganglionic, pain, temperature, touch, and proprioception and motor fibres.

Distribution:

α -half-life - 2.7 minutes (uptake by rapidly equilibrating tissues)

β -half-life - 28 minutes (distribution by slowly perfused tissue)

γ -half-life - (biotransformation and excretion)

PHARMACOKINETICS:

Absorption: A dose of local anaesthetic eventually is absorbed into the circulation. Lipid solubility of the agent determines the proportion remaining in the aqueous phase available for rapid removal by the blood and the proportion taken up by the tissues which can be altered by local anaesthetics itself and the addition of adrenaline. Absorption of Bupivacaine is dependent on the site of injection, dosage, and the use of Epinephrine. It is first distributed to tissues with high blood flow. Uptake by less perfused tissues is not significant.

Distribution: In the plasma, it is bound to plasma proteins principally to acid glycoprotein to an extent partly related to its potency and lipid solubility. Since protein binding of Bupivacaine is very high i.e. 95%, it crosses placenta to a limited extent. The uptake of Bupivacaine into lung tissue is high. This pulmonary extraction limits the concentration of drug that reaches the systemic circulation for distribution to the coronary and cerebral circulations.

The volume of distribution is (0.9±0.4) L/kg;

Half-life is (2.4±1.2) hours.

As Bupivacaine is very lipid soluble and has high protein binding considerable portion of the drug deposited in the CSF is rapidly distributed into neuroaxis. Hence, the CSF concentrations do not reflect actual neural uptake. Epinephrine unlike the Lignocaine does not affect the duration of block with Bupivacaine but decreases the plasma uptake and may help to identify intravascular injection..

Biotransformation and excretion: The metabolism and excretion phase of Bupivacaine is the longest which accounts for its longer duration of action. Slower metabolism renders sustained increases in plasma concentrations and thus systemic toxicity more likely. Cumulative drug effects are also more likely. Bupivacaine undergoes enzymatic degradation in the liver and possible mechanisms include aromatic hydroxylation, N-dealkylation, amide hydrolysis and conjugation. Biodegradation of bupivacaine depends on the blood flow and enzymatic activity of the liver. Elimination is prolonged by a reduction in splanchnic blood flow. The main products are pipecolyloxlidine and N-dealkylated metabolites (N-desbutylbupivacaine) which are excreted mainly through the kidney. The mean total urinary excretion of Bupivacaine and its dealkylation and hydroxylation metabolites accounts for more than 40% of the total anaesthetic dose. Less than 5% is excreted unchanged in urine. The clearance is about 0.58 l/ min.

PHARMACOLOGICAL EFFECTS

The effects produced may be:

a) **Local:** Nerve blockade and a direct effect on smooth muscles.

b) **Regional:** Loss of pain, temperature, touch, motor power, and vasomotor tone in the region supplied by the blocked nerves.

c) **Systemic:** Effects due to systemic absorption of drug

CARDIOVASCULAR SYSTEM:

Bupivacaine penetrates the myocardium rapidly but leaves it very slowly – "fast in, slow out" drug. Bupivacaine depresses the rapid phase of depolarization in Purkinje fibers and ventricular muscle to a greater extent than Lignocaine. In addition rate of recovery from a dose dependent block is slower in Bupivacaine affected papillary muscle than in Lignocaine since Bupivacaine leaves the heart muscle slowly. Bupivacaine prolongs conduction time through various parts of heart. Extremely high concentration of Bupivacaine depresses spontaneous pacemaker activity in the sinus node, resulting in sinus bradycardia and sinus arrest. Bupivacaine also depresses cardiac contractility. So, it is more cardio toxic than lignocaine. Bupivacaine is more arrhythmogenic than Lignocaine. The local anaesthetic and cardiodepressant potency ratio is 4:1 (Bupivacaine: Lignocaine).

The cardiac elecrophysiological toxicity ratio is 16:1.

Bupivacaine exerts a biphasic effect on peripheral vascular smooth muscle. Low concentration of Bupivacaine produces vasoconstriction while high concentration produces vasodilatation

CENTRAL NERVOUS SYSTEM:

Bupivacaine can cause nervous system toxicity ranging from feeling of lightheadedness, dizziness to generalized convulsions followed by a state of generalized CNS depression. Convulsive dose is 3.6±0.1 mg/kg IV. Toxicity occurs at plasma level at 1.6-2μg/kg/ml and convulsions occur at plasma level 2.3-5μg/kg/ml.

Bupivacaine when used within therapeutic dosages and with careful and correct techniques is safe without any significant side effects

DOSAGE:

The usual preparation available to us is 0.5% solution of heavy Bupivacaine, which is made hyperbaric by the addition of 80 mg/cc of dextrose.

Dose: The highest recommended dose of Bupivacaine in adults is 2 mg /kg body weight, with maximum of 150 mg over 4 hours and 400 mg during 24 hours Intrathecally, maximum dose is 20 mg in adults. For subarachnoid block - Onset time: 5-7 minutes Duration of action: 3-4 hour.

TOXICITY:

The higher the levels of local anaesthetic in the blood, the higher are the chances of adverse systemic reactions. The chances of toxicity are even more in the presence of hypoxia, hypercarbia and pregnancy.

Cardiovascular System:

Bradycardia, primary cardiac failure due to direct myocardial depression, circulatory collapse, hypotension and cardiac arrest can occur.

Central Nervous System:

Cortical stimulation causing anxiety, excitement, sweating, disorientation, tremors, twitching, and convulsions may be seen.

Local tissue toxicity:

Bupivacaine employed locally rarely produces localized nerve damage.

Allergy:

Bupivacaine may rarely cause allergic reaction.

In respiratory system –

Respiratory depression and apnea due to medullary center depression are caused by the drug. Most of the respiratory cases Bupivacaine toxicity involved obstetric patients. The reason is still not clear. It may be due to more frequent use of Bupivacaine for obstetric purpose.

Cause of adverse reaction:

- Injection in excessive dose

- Inadvertent intravascular injection.

- Slow metabolic degradation due to high protein binding and lipid solubility.

- Sensitivity to local anaesthetics.

MANAGEMENT:

a) Prevention

- Not to exceed maximum allowable dose as per mg/kg.
- Slow injection of the drug with repeated aspiration to prevent intravascular injection.
- Confirmation of the drug and its expiry date

b) Airway to be secured in cases of convulsions

c) IV fluids and vasopressors to combat hypotension.

d) Correction of acidosis, hypoxia and hypercarbia.

e) Intralipids: Twenty percent of intralipid 1ml/kg over 1minute repeated every 3 to 5 minutes to a maximum of 3 ml/kg, followed by a continuous infusion of 0.25 ml/kg/minute until haemodynamic stability is restored. Exact mechanism how intralipid reverses LA toxicity is unclear, but postulated

mechanisms include an action as a lipid sink or improving fatty acid delivery to the heart for energy metabolism.

Uses:

1) Subarachnoid Block:

Bupivacaine heavy 0.5% is used for this purpose. 3-4cc volume is used. Prolonged analgesia and good muscle relaxation is achieved.

2) Epidural Block:

Bupivacaine is used extensively for lumbar epidural blockade when given by intermittent injections in concentrations of 0.0625% to 0.5%. There is considerable variability in the quality of motor blockade achieved, with complete motor block at a higher concentration and almost no motor block below 0.1%.

3) Peripheral Nerve Blocks:

a) Bupivacaine used in concentration of 0.125 to 0.5% onset of action is 10-15 minutes and gives good analgesia postoperatively for 5-7 hours.

PHARAMACOLOGY OF CLONIDINE HYDROCHLORIDE [33,34]

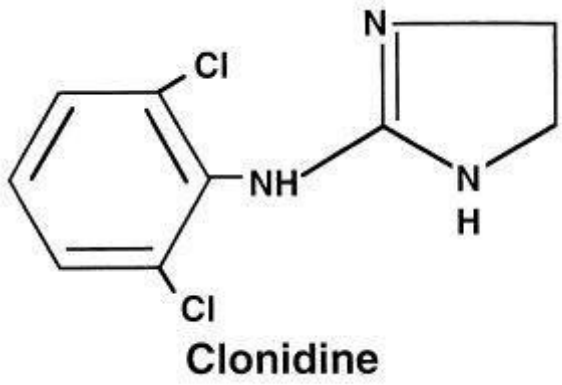


Fig 6 Structure of clonidine

N – (2,6 – dichlorophenyl) -4, 5 –dihydro – 1H – imidazol – z – amine

Clonidine is a direct acting ά2 agonist prescribed historically as an antihypertensive agent. In addition to its antihypertensive effect, in recent studies, Clonidine has been demonstrated to be an effective sedative and analgesic which reduces the amount of anaesthetic agents required when used as part of anaesthetic technique. Therefore, a reconsideration of possible new indications for Clonidine in clinical anaesthesiology is justified

PHYSIOCHEMICAL CHARACTERISTICS

Clonidine is a white or almost white crystalline powder. It is soluble in water and in dehydrated alcohol. A 5% solution in water has pH of 4.0 to 5.0 stored in airtight containers at a temperature of 25^0c, excursion permitted between 15^0c and 30^0c.Molecular weight of Clonidine hydrochloride (C9H9CL2N3.HCl) is 266.6.

MECHANISM OF ACTION

ANALGESIA:

Alpha-2 adrenoreceptors are located on primary afferent terminals(both at peripheral and spinal endings), on neurons in the superficial laminae of the spinal cord, within several brainstem nuclei implicated in analgesia, supporting the possibility of analgesic action at peripheral, spinal and brainstem sites. In contrast to blood, there is a strong correlation between Clonidine concentration in cerebrospinal fluid (CSF) and analgesia after Clonidine administration. Clonidine is rapidly absorbed into the spinal CSF compartment after epidural administration with concentrations peaking 30-60min after injection. Cerebrospinal fluid is clearly not the site of action of Clonidine for analgesia and the drug can reach sites producing analgesia in the spinal cord or elsewhere. As with other lipophilic drugs, it is possible to achieve analgesia from systemic, epidural or intrathecal administration of Clonidine. However, Clonidine is more potent after neuraxial than systemic administration, indicating a spinal site of action favouring neuraxial administration. This action of Clonidine on alpha2-adrenoreceptors has been shown by partial reversal of epidural Clonidin's analgesia and sedation by administering the alpha2- adrenergic antagonist, yohimbine although the effects on blood pressure and heart rate were not reversed. There are also suggestions that (in animal studies) Clonidine causes analgesia, in part, by spinal cholinergic activation due to increase in acetylcholine concentrations in the dorsal more than ventral horn in the spinal cord. . Inhibition of substance – p release is also believed to be involved in the analgesic effect. Clonidine also enhances both the sensory and motor blockade from epidural or peripheral nerve block injection of local anaesthetics. Different possible mechanisms

have been suggested. First the ability of Clonidine to modify the function of potassium channels in isolated neurons in vitro (cell membranes become hyperpolarized) may be the mechanism for profound decrease in anaesthetic requirements produced by Clonidine. Second Clonidine may cause high local vasoconstriction, in clinical setting thereby reducing vascular removal of the local anaesthetic however there is little evidence for this mechanism with the clinically used concentrations.

Sedation:

Sedation usually accompanies the use of Clonidine through its actions on the locus coeruleus. Sedation after epidural Clonidine likely reflects systemic absorption and vascular redistribution to higher centers. The quality of sedation produced by alpha 2 agonists differs from sedation produced by drugs (Midazolam, Propofol) that act on gama-aminobutyric acid receptors (GABA). Clonidine acting on alpha2- adrenergic receptors inhibits the sleep regulatory physiological processes in the locus coeruleus via a G-protein mediated mechanism and produces sedation. The result is a calm patient who can be easily aroused to full consciousness. Drugs that activate GABA receptors produce a clouding of consciousness and can cause paradoxical agitation as well as tolerance and dependence.

Clonidine produces dose-dependent sedation over the dose range 50-900mcg of rapid onset (<200min) regardless of route of administration. After a large epidural bolus dose (700mcg) sedation is intense for 4-6h. In many cases sedation is a desirable property and several studies have demonstrated the reduced need for other sedatives and anxiolytic medications when Clonidine is administered intra operatively.

Peripheral action of clonidine

Clonidine was initially used for its antihypertensive properties. The central actions are mediated through $\alpha_2$adrenoceptors, which are situated at locus coeruleus and dorsal horn of spinal cord. But, specific peripheral effects of Clonidine appear to be less obvious because α_2 adrenoceptors are not present on the axon of the normal peripheral nerve. [73] There have been four proposed mechanisms for the action of Clonidine in peripheral nerve blocks. These mechanisms are centrally mediated analgesia, α_2 β adrenoceptor-mediated vasoconstrictive effects, attenuation of inflammatory response and direct action on peripheral nerve. The direct action of Clonidine on the nerve can be explained on the basis of a study conducted by Dalle *et al.,* They proposed that Clonidine, by enhancing activity-dependent hyperpolarisation generated by the Na/K pump during repetitive stimulation, increases the threshold for initiating the action potential causing slowing or blockage of conduction. Kosugi *etal.,* [35] examined the effects of various adrenoceptor agonists including Dexmedetomidine, Tetracaine, Oxymetazoline and Clonidine, and also an $\alpha_2$adrenoceptor antagonist (Atipamezole) on compound action potential (CAP) recorded from frog sciatic nerve, and found that CAPs were inhibited by $\alpha_2$adrenoceptor agents so that they are able to block nerve conduction.

Haemodynamics

Clonidine affects blood pressure in a complex fashion after neuraxial or systemic administration because of opposing actions at multiple sites. In the nucleus tractus solitarius and locus coeruleus of the brainstem, activation of postsynaptic ά2 – adrenergic receptors reduces sympathetic drive. It also activates non-adrenergic imidazoline –

preferring binding sites in the lateral reticular nucleus, thereby producing hypotension and an antiarrthymic action. In the periphery, its action on presynaptic ά2 – adrenoceptors at sympathetic terminals reduce the release of norepinephrine causing vasorelaxation and reduced chronotropic drive. These brainstem and peripheral effects of ά2 - adrenoceptor stimulation are counterbalanced by direct peripheral vasoconstriction through its action on ά1- adrenoceptors from circulating concentrations of Clonidine. As a result, the dose – response for Clonidine by neuraxial or systemic administration is U-shaped, with peripheral vasoconstriction from circulating drug concentrations at high doses opposing central sympatholysis. In addition to brainstem and peripheral sites of actions, neuraxial administration of Clonidine directly inhibits sympathetic preganglionic neurons in the spinal cord. The rather complex action of intrathecally injected α2-adrenergic receptor agonists on hemodynamic variables further depends on the segmental site of injection, the patient's position, the rate of injection, and the temperature of the injected solution. Furthermore, combining α2-adrenergic receptor agonists with local anaesthetics can potentially increase the degree of sympatholysis and resulting hypotension. However, clinical studies in surgical patients on this matter have only infrequently reported increased reductions in arterial BP or heart rate in patients who received both intrathecal Clonidine and local anaesthetics.

Clonidine reduces heart rate partly by a presynaptically mediated inhibition of norepinephrine release at the neuroreceptor junction and partly by vagomimetic effect.

Hemodynamic effects of Clonidine after neuraxial or systemic administration begin within 30 min and reach maximum within 1-2h and last approximately 6-8 h after a

single injection. Delayed onset of hypotension has not been observed with the use of Clonidine for analgesia alone or in combination.

Respiratory depression:

Alpha sub2-adrenergic agonists do not induce profound respiratory depression even after massive overdose nor do they potentiate respiratory depression from opioids.

Renal:

Salt and water retention can occur and is due to reduced sympathetic tone. Conversely a diuretic effect during general anaesthesia has been described after administration of oral Clonidine, 2.5 to 5.0 mcg/kg, as preanaesthetic medication.

GIT:

Constipation is also relatively common side effect of Clonidine and incidence is about 10% is due to antisecretory effect on the intestine.

Dermatological: -

Rash, erythema, allergic contact dermatitis, angioneuroticedema, urticaria, alopecia and pruritus may occur. Skin reactions have been reported in up to 50% of patients using Clonidine transdermal patches.

Other: -

Clonidine prevents opioid induced skeletal muscle rigidity and produces skeletal muscle flaccidity; alpha 2 agonists have no effect on the responses evoked by

neuromuscular blocking drugs. Clonidine hydrochloride has been associated with acute attack of porphyria and is considered unsafe in porphyria patients.

PHARMACOKINETICS

Clonidine is highly lipid soluble and hence rapidly absorbed after oral, intravenous and epidural administration. After epidural administration, Clonidine is rapidly and extensively absorbed into the spinal CSF compartment, with concentration peaking 30 to 60 minutes after injection. There is a strong correlation between Clonidine concentration in the CSF and analgesia after epidural Clonidine administration. Epidurally administered Clonidine readily partitions into plasma via the epidural veins and attains systemic concentrations (0.5 – 2 ng / ml) that are associated with a hypotensive effect mediated by the central nervous system. After intravenous administration it is readily distributed into extravascular sites including the central nervous system.

Molecular mass 230.093 gm / ml

Bioavailability 75 – 95%

Protein binding 20 – 40%

Volume of distribution 2.1 + 0.4 L / Kg

Elimination T ½ 9 + 2 hours

Onset time 26 + 11 minutes

METABOLISM

In the liver Clonidine undergoes hydroxylation to form major metabolite p-hydroxyclonidine. Only 50% of the drug is metabolized in the liver and the remaining is excreted as unchanged drug in the urine. Plasma albumin is the most important protein binding site for Clonidine and varies between 20 – 40% in vitro.

SIDE EFFECTS

The most common side effects produced by Clonidine are drowsiness, dry mouth, bradycardia and hypotension. It also causes inhibition of orgasm in women. Rebound hypertension can occur after abrupt discontinuation of Clonidine therapy (1.2 mg / day) as early as 8 hours and as late as 36 hours after the last dose. Rebound hypertension can usually be controlled by reinstituting Clonidine therapy or by administering a vasodilating drug such as Hydralazine or Sodium Nitroprusside.

CLINICAL USE

usual daily adult dose is 0.2 to 0.3 mg orally. Transdermal clonidine patch designed for weekly administration is useful for surgical patients who are unable to take oral medications.

Hypertension

Treatment of patients with severe hypertension or renin dependent disease . The usual daily adult dose is 0.2 to 0.3 mg orally. Transdermal clonidine patch designed for weekly administration is useful for surgical patients who are unable to take oral medications.

Anaesthetic use of Clonidine:

1. **Premedication:**

The sedative effect can be useful when Clonidine is used as a premedicant. In addition it also has an anaesthesia- sparing effect. Alpha-2 adrenergic agonists reduce the dose of intravenous hypnotics and also reduce the MAC of the volatile anaesthetic agents. Clonidine has been recommended in doses of 4 mcg/kg orally or intranasally and in doses of 5µg/kg rectally provides adequate sedation.

Its use as a premedicant is particularly useful in certain subgroup of patients like

- Drug addicts and alcoholics who give problems like withdrawal symptoms.
- Chronic pain and palliative care patients
- Hypertensive patients

2. **Control of haemodynamic response**:

The haemodynamic effects of alpha-2 adrenergic agonists are both central and peripheral. In addition alpha-2 adrenergic agonists depress presynaptic sympathetic neurons in the lateral horn of the thoracic spinal cord. It should be noted here that this effect is reversed by the local administration of cholinesterase inhibitor neostigmine. Clonidine prevents hypertension and tachycardia during laryngoscopy and intubation as well as during surgical stimulation

1. Postoperative analgesia and Regional anesthesia

Clonidine increases the analgesic effect of opiates and interacts with cholinergic neurons to do so. They augment local anaesthetic blockade and prolong duration.

2. Central Neuraxial Block

a. **Epidural:** If used as a sole agent to produce epidural analgesia large doses (up to 2 - 3000 mcg/ day) are needed to produce long term analgesia. At these doses significant sedation, bradycardia, hypotension are common. Thus its use as a sole is not popular at all. It is used more commonly as a combination with opioids and or local anesthetics to provide good to excellent analgesia with minimal side effects. The dose in combination with other agents is limited to 10 - 15 mcg / hour

b. **Spinal**: Clonidine produces analgesia of shorter duration but without the associated risk of respiratory depression or urinary retention. The maximum dose of intrathecal Clonidine is 1-2 mcg/kg. Giving Clonidine with local anesthetics improves the quality and duration of the block, minimizes the

tourniquet pain during lower limb surgery, and prevents shivering.

c. **Caudal:** Caudal Clonidine combined with local increases the duration of anaesthesia and analgesia by a factor of 2 or 3 without haemodynamic side effects. The dosage recommended in the caudal route is 1-2 mcg / kg.

Epidural Clonidine has also been suggested for use in labour analgesia Clonidine has been given alone or in combination with sufentanil and bupivacaine. Clonidine does cross the placental barrier but no adverse events have been documented in the newborns. To avoid hypotension and bradycardia in the foetus as well, the recommended dose of Clonidine has been suggested as100 mcg during labour

3**. Peripheral Nerve Blocks:**

Clonidine is commonly used as an adjuvant to local anesthetics in peripheral nerve blocks where it prolongs the duration of anaesthesia as well analgesia. This effect is obtained at relatively small doses (1-2 mcg/kg) which obviously reduce the risks of side effects.

Other uses are:

1. Prevention of emergence agitation:
2. Decreasing Minimum Alveolar Concentration (MAC) of sevoflurane:
3. Postoperative nausea and vomiting (PONV):
4. Controlled hypotension:
5. In cardiovascular surgery:
6. Post-operative shivering:

A dose of 1.5mcg/kg is required to stop shivering in 5 minutes after drug administration.

7. Daycare Surgery:
8. Attenuation of pressor response to tracheal intubation and extubation:

   Children premedicated with rectal clonidine 2.5 mcg/kg did not have a rise in neuropeptide Y, a marker of major adrenergic activation during tracheal intubation. Oral clonidine 4 mcg/kg given 10 minutes before induction attenuated hemodynamic changes associated with tracheal extubation

9. Anaesthetic sparing effect:

   Oral clonidine premedication at a dose of 2-4 mcg/kg decreases the dose of intravenous barbiturate required for induction of anaesthesia.

10. Treatment of spasticity:

    Clonidine is used in children diagnosed with cerebral palsy or traumatic brain injury.

Precautions

Use cautiously in:

- Renal insufficiency, serious cardiac or cerebrovascular disease
- Elderly patients
- Pregnant or breastfeeding patients.

Interactions

Drug-drug.

- Amphetamines, beta-adrenergic blockers, MAO inhibitors, prazosin, tricyclic antidepressants ------ decreased antihypertensive effect
- Beta-adrenergic blockers ------- increased withdrawal phenomenon
- CNS depressants (including antihistamines, opioids, sedative-hypnotics) ------ additive sedation
- Epidurally administered local anesthetics--- prolonged clonidine effects
- Levodopa: decreased levodopa efficacy
- Myocardial depressants (including beta-adrenergic blockers): additive bradycardia
- Other antihypertensives, nitrates: additive hypotension Verapamil: increased risk of adverse cardiovascular reactions

Drug-herbs. Capsicum: reduced antihypertensive effect

Drug-behaviors. Alcohol use: increased sedation

DOSAGE GUIDELINES: CLONIDINE DOSE

Oral - 3-5µg/Kg

Intrathecal - 15µg to 30µg

Epidural - 1µg/kg (or) 50µg . 30 µg /hr (for infusion)

Intravenous - 50 – 75 µg (or) 1µg/kg 15 minutes prior to induction for intubation response attenuation;150 – 300 µg (or) 3 µg / kg for hypertensive crisis; 30 µg given slowly for shivering management.

PREPARATION:

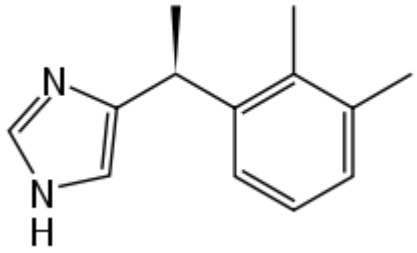
Clonidine is available in 100 microgram and 150 microgram tablets. Also it is available in Injectable form as clear preservative free preparation in1 ml ampoules of 150 microgram.

PHARMACOLOGY OF DEXMEDETOMIDINE [6,36,37,38,]

Dexmedetomidine is a potent, highly selective alpha-2 adrenoreceptor agonist. The α2:α1 binding selectivity ratio of Dexmedetomidine is 1620:1 compared to 220:1 for Clonidine.

Dexmedetomidine is available as 100 µg/ml strength in 0.5 ml, 1 ml and 2 ml ampoules. It is a preservative free solution and contains no additives or stabilizers.

Physicochemical Characteristics



Dexmedetomidine, the pharmacologically active d-isomer of medetomidine

Fig 7 structure of Dexmedetomidine

(4,[5]-[1-(2,3-dimethylphenyl)-ethyl] imidazole is a highly specific and selective alpha-2 adrenoreceptor agonist.

MpKa: 7.1

Molecular mass: 236.7

pH 4.5-7.0

Solubility:Water soluble

Absorption and distribution

Dexmedetomidine exhibits linear pharmacokinetics. After IV administration, Dexmedetomidine has an onset of action of approximately 15 minutes. Peak concentrations are usually achieved within 1 hour after continuous IV perfusion.Dexmedetomidine is also absorbed through the transdermal, oral or intramuscular routes, with a mean bioavailability of 82 and 104%, from the latter two routes respectively. The distribution phase is rapid, with a half-life of distribution of approximately 6 minutes and elimination half life of 2 hours.

Protein binding to serum albumin and α1-glycoprotein is 94% and is constant across the different plasma concentrations. There is negligible protein binding displacement by Fentanyl, Ketorolac, Theophylline, Digoxin, Lignocaine and all drugs commonly used during anaesthesia and in the intensive care unit.

Its steady state volume of distribution is 118 litres and its distribution half life is 6 minutes in adults over dose ranges of 0.2-0.7 µg/kg per hour, an elimination half life of between 2.0 and 2.5 hours and a clearance of 39 litres/hour.

Metabolism and excretion

Dexmedetomidine is extensively metabolized in the liver through glucuronide conjugation and biotransformation by the cytochrome P 450 enzyme system. There are

no known active or toxic metabolites. However, hepatic clearance may be decreased by as much as 50% of normal with severe liver disease. No differences have been seen between healthy patients and those with renal impairment.

Pharmacodynamics

Cardiovascular effects

The cardiovascular effects of Dexmedetomidine are mediated via adrenoreceptors in both the central and peripheral nervous systems resulting in sympatholysis. It has a biphasic blood pressure response, a short hypertensive and subsequent hypotensive response. The initial response lasts for 5 to 10 minutes and is followed by a decrease in blood pressure of approximately 10% to 20% below baseline and a stabilisation of the heart rate, also below the baseline values: both of these effects are caused by the inhibition of the central sympathetic outflow overriding the direct stimulating effects. Bradycardia and sinus arrest can occur which will respond to anticholinergics.

Central nervous system

Dexmedetomidine reduces cerebral blood flow and cerebral metabolic rate. Activation of these receptors in the brain and spinal cord inhibits neuronal firing causing hyperpolarisation of excitable cells, producing analgesia, sedation and anxiolysis. Sedation mediated through the locus coeruleus closely mimics endogenous sleep. It produces good degree of sedation, still patients are easily arousable. It does not affect intracranial or lumbar cerebrospinal fluid pressure or cerebral perfusion pressure.

When administered via the neuraxial route, it confers some analgesic and antinociceptive actions. Being highly lipophilic, Dexmedetomidine is rapidly absorbed into cerebrospinal fluid and binds to alpha-2A adrenoreceptors of dorsal horn of spinal cord. It prolongs the duration of both sensory and motor blockade caused by local anaesthetics.

It is also said to provide neuroprotection via modulation of the proapoptotic and antiapoptotic proteins.

Respiratory system

Dexmedetomidine does not cause respiratory depression. Upper airway patency is maintained despite good sedation.

Thermoregulation

Dexmedetomidine interferes with thermoregulation by diminishing shivering, vasoconstriction and non-shivering thermogenesis. It attenuates shivering mediated via a dose dependent decrease in thermoregulatory vasoconstriction and shivering thresholds.

Endocrine and renal effects

Dexmedetomidine activates peripheral presynaptic alpha-2 adrenoreceptors which reduces the release of catecholamines, and hence attenuate sympathetic response to surgery.

Alpha-2 agonists exert a diuretic effect by inhibiting action of vasopressin at the collecting duct. They also enhance osmolal clearance through vasopressin independent pathways, possibly mediated by the alpha-2b receptor.

Miscellaneous

Activation of the alph-2 receptors in other areas causes decreased salivation, decreased secretion and bowel motility in the gastrointestinal tract; contraction of vascular and other smooth muscle; inhibition of renin release, increased glomerular filtration, and increased secretion of sodium and water in the kidney; decreased intraocular pressure and decreased insulin release from the pancreas.

Adverse Effects

Bradycardia and hypotension are the most common side-effects and can be described as an adverse exaggeration of their clinical adverse effects. Severe bradycardia leading to cardiac arrest has been reported with the use of Dexmedetomidine. Other reported side effects are hypertension, nausea, vomiting, dry mouth, atrial fibrillation, etc. Rapid administration of Dexmedetomidine infusion (loading dose of 1 μg/ kg if given in less than 10 minutes) may cause transient hypertension.

Clinical Applications of Dexmedetomidine

Alpha-2 agonists produce number of effects such as analgesia, anxiolysis, sedation, and sympatholysis which are desirable perioperatively.

Premedication

Dexmedetomidine possesses anxiolytic, sedative, analgesic, antisialogogue and sympatholytic properties. At IV doses of 0.33 to 1 μg/kg given 15 minutes before surgery it can minimize the cardiovascular side effects. Dexmedetomidine 1 μg/kg has been used effectively via nasal route as premedication.

Intraoperative use

- **As adjunct to general anaesthesia**

Dexmedetomidine attenuates the stress-induced sympathoadrenal responses to intubation, surgery and emergence from anaesthesia. It potentiates the action of all intraoperative anaesthetics and decreases perioperative oxygen consumption. Administration of intravenous Dexmedetomidine produces an anaesthetic sparing effect. It also has perioperative opioid-sparing effect. Dexmedetomidine use is associated with a lower incidence of shivering. It maintains upper airway patency despite good degree of sedation. This property makes it suitable for the management of difficult airway with a Dexmedetomidine infusion of 0.5 to 1.5 μg/kg/hr.

- **In regional anaesthesia**

Epidural Dexmedetomidine 1 μg/kg is associated with sedation, analgesia, sympatholysis and prolongation of local anaestheticaction.Addition of 0.5 μg/kg Dexmedetomidine to Lignocaine for intravenous regional anaesthesia improves the quality of anaesthesia and perioperative analgesia without causing side effects. 3 μg of intrathecal Dexmedetomidine with 12 mg Bupivacaine caused a significant prolongation of sensory and motor block. Dexmedetomidine 1-2 μg/kg when combined

with 0.25% Bupivacaine decreases the anaesthetic requirements, incidence of emergence agitation and the need for analgesics postoperatively.

- In monitored anaesthesia care

Dexmedetomidine has been found to be useful as baseline sedative for patients undergoing procedures under monitored anaesthesia care as it provided greater patient satisfaction, reduced opioid requirements, and avoided respiratory depression. Dexmedetomidine effects are similar to that of Midazolam and Fentanyl, but without respiratory depression for patients undergoing diagnostic transesophageal echocardiography. Dexmedetomidine with fentanyl has been satisfactorily used for sedation and analgesia during extracorporeal shockwave lithotripsy.It has also been shown to be effective drug for monitored anaesthesia care for patients undergoing cataract surgeries on outpatient basis.Continuous infusion of Dexmedetomidine without a loading dose has shown benefits in terms of cardiovascular stability and early discharge from health care unit.

- Sedation in intensive care unit(ICU)

Dexmedetomidine has become popular sedative agent in ICU because of its ability to produce cooperative sedation in the form of patients remaining awake, calm, and retaining their ability to communicate their needs. The maintenance of natural sleep during sedation might speed recovery time in the ICU. It is recommended for use only for 24 hours.

- Procedural sedation

Dexmedetomidine is an attractive agent for short term procedural sedation and has been safely used in colonoscopy, awake carotid endarterectomy, shockwave lithotripsy, vitreoretinal surgery, elective awake fiberoptic intubation, paediatric MRI. It produces a unique form of sedation in which patients become responsive as well as calm and cooperative when aroused, and then back to sleep when not stimulated.

Contraindications Patient with heart block

- Infusion over 24 hours.
- In obstetric procedures, cesarean section deliveries, as the safety has not been studied.
- Patients with pre-existent severe bradycardia and related bradydysrhythmias
- Patients with impaired ventricular functions (ejection fraction <30%).
- Patients who are hypovolemic or hypotensive.
- Patient with raised intracranial tension.
- Used with Caution when other vasodilators or negative chronotropic agents are administered, and with renal or hepatic impairment, dose reductions needed as may accumulate with long-term infusions.

MATERIALS AND METHODS.

SOURCE OF DATA:

This study was carried out in the Department of Anaesthesiology, _____ _____. Study was conducted from December 2015 to August 2017.

METHOD OF COLLECTION OF DATA:

Study Period: One and half year.

Sample Size: Total 100 patients of either sex, 50 patients in each group scheduled for upper limb surgeries. In a study, it was found that the quality of block received by group Dexmedetomidine 80% and by group C Clonidine 40%. With these proportion at 95% confidence level and considering 90% of power in the study the sample size worked out is 50.

$$n = \frac{(Z_\alpha + Z_\beta)^2 x\ p\ x\ q\ x\ 2}{d^2}$$

$Z_{\alpha\beta}$=Z value at α level=95%

Z_β=Z value at β level =90

P= common proportion

q=100-p

d=differences between parameters.

STATISTICAL DATA:

1. Data was represented in the form of frequencies and percentages and diagrams
2. Association between qualitative variables were assessed by Chi-Squaretest fisher's exact test.
3. Analysis of Quantitative data between the two groups was done using unpaired t-test if data passes 'Normality test' and by Mann-Whitney Test if data fails 'Normality test'
4. P value was considered significant if <0.05 and highly significant if <0.001

Randomization:

Total of 100 patients were studied

Patients of either sex was randomly allocated into Group C and Group D.

Group C – received Bupivacaine 0.25% (35 ml) + inj Clonidine 1 mcg / kg.

Group D-- received Bupivacaine 0.25% (35 ml) + inj Dexmedetomidine 1 mcg/kg

INCLUSION CRITERIA:

Patients with

- ASA I and II physical status.
- Patient with age group of 18 to 60yrs

Patient's undergoing elective upper limb surgeries

EXCLUSION CRITERIA:

- ASA III and ASA IV
- Patient with known Hypersensitive to study drugs
- Patient's refusal
- Patient's with heart block
- Local cutaneous infections at the site of injection
- Patients with coagulopathy or on anticoagulants
- Patients with central and peripheral neuropathy
- Pregnant and lactating patients
- Patients with heart blocks

a) For the procedure:

A portable tray covered with sterile towels containing :

- Sterile syringes - one 20ml and one 10ml.
- Hypodermic needles of 5 cm length, 22 G.
- Bowl containing povidone iodine and spirit.
- Sponge holding forceps.
- Towels and towel clips.
- Sterile gauze pieces.

b) For emergency resuscitation:

- The anesthesia machine, emergency oxygen source (E type cylinders), pipeline O2 supply, working laryngoscopes, appropriate size endotracheal tubes and connectors.
- Working suction apparatus with suction catheter.
- Oropharyngeal airways.
- Intravenous fluids.
- Drugs : Thiopentone, Succinylcholine, Hydrocortisone, Atropine, Adrenaline, Aminophylline, Mephenteramine, Calcium gluconate and Sodium bicarbonate.

c) Monitors:

- Pulse oximeter.
- ECG
- NIBP monitor

METHODOLOGY

- After ethical committee approval and written informed consent, a randomized prospective clinical study was carried out on 100 American society of anaesthesiology (ASA) grade I and II patients of either sex aged 18-60 years , undergoing various bony orthopedic surgeries on the upper limb under supraclavicular brachial plexus block. The study was conducted in two groups of

50 patients each. The patients were randomly assigned using “randomized computer generated slip” to one of the following groups:

- Group C : received Bupivacaine 0.25% (35 ml) + Clonidine 1 mcg / kg.
- Group D : received Bupivacaine 0.25%(35ml) + Dexmedetomidine1 mcg / kg.
- On the arrival in the operation room, baseline heart rate, blood pressure and oxygen saturation were recorded. An IV line was be secured with 18 G cannula in the unaffected limb and Ringer’s lactate was started.
- All the patients received supraclavicular brachial plexus block using Sonosite M Turbo ultrasound machine.
- Following negative aspiration, 35mL of a solution containing local anaesthetic combined with Clonidine or Dexmedetomidine as mentioned were injected. A 3 min massage was performed to facilitate an even drug distribution.
- Sensory block was assessed by the pin prick method. Assessment of sensory block was done at each minute after completion of drug injection in the dermatomal areas corresponding to median nerve, radial nerve, ulnar nerve and musculo-cutaneous nerve till complete sensory blockade. Sensory onset was considered when there was dull sensation to pin prick along the distribution of any of the above mentioned nerves. Complete sensory block was considered when there was complete loss of sensation to pin prick.
- Sensory block was graded as –

Grade 0- sharp pin felt

Grade 1- analgesia , dull sensation felt

Grade 2- anaesthesia , no sensation felt

- Assessment of motor block was carried out each minute till complete motor blockade after the drug injection. Onset of motor blockade was considered when there was Grade 1 motor blockade. Peak motor block was considered when there was Grade 2 motor blockade.

- Motor block was determined according to modified Bromage scale for upper extremities on 3 point scale.

  Grade 0- Normal motor function with full flexion extension of elbow wrist fingers.

  Grade 1- Decrease motor strength with ability to move the fingers only.

  Grade 2- Complete motor block with inability to move the fingers.

- This block was incomplete when any of the segments supplied by median, radial, ulnar and musculo cutaneous nerve did not have analgesia even after 30 minutes of drug injection. These patients were supplemented with IV Fentanyl (1-2µg/kg) and Midazolam (0.02 mg/kg). When more than one nerve remained unaffected it was considered a failed block in this case general anaesthesia was given. Patients were monitored for hemodynamic variables heart rate, blood pressure and oxygen saturation every 30min after the block intra operatively and every 60 mins post operatively. The sedation on patient was assessed by Ramsay sedation score. At

the end of the procedure, quality of blockade was assessed according to numerical score.

QUALITY OF BLOCKADE:

Grade 4- Excellent , no complaints from patient

Grade 3- Good, minor complaints with no need for analgesics.

Grade 2- Moderate complaint that require supplemental analgesia

Grade 1- Unsuccessful, patient given general anaesthesia.

RAMSAY SEDATION SCORE

If awake

1. Anxious, agitated, restless
2. Cooperative, oriented, tranquil
3. Responsive to commands only. If asleep
4. Brisk response to light glabellar tap or loud auditory stimulus
5. Sluggish response to light glabellar tap or loud auditory stimulus
6. No response to light glabellar tap or loud auditory stimulus

Patients were assessed for duration of analgesia as per a numeric rating scale of 0 to 10.The numeric rating scale was recorded post operatively every 60 min till the score of 5.The rescue analgesia was given in the form of inj. Diclofenac sodium(1.5mg/kg)

intramuscularly at the Numerical Rating Scale of 5 and the time of administration was noted. All patients were observed for any side effects like nausea, vomiting, dryness of mouth and complications like pneumothorax, haemotoma, local anaesthetic toxicity and post block neuropathy in the intra and postoperative periods.

The duration of sensory block is defined as the time interval between the end of local anaesthetic administration and the complete resolution of anaesthesia on all nerves. The duration of motor block is defined as the time interval between the end of local of anaesthetic administration and the recovery of complete motor function of the hand and the forearm.

Preanaesthetic evaluation:

During preoperative visit patient's detailed history, general physical examination and systemic examination was carried out. History of any significant medical illness was elicited. Airway, respiratory system and cardiovascular system was assessed.

Only ASA grade I and II patients within the age group of 18 to 60 years of both sex undergoing upper limb surgeries was included in our study. A written informed consent was taken.

RESULTS

TABLE NO.3

Mean Age and Weight of cases between study groups

Variables	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
Age	34.40	13.53	36.18	12.76	0.5
Weight	58.5	4.9	57.2	3.7	0.149

Figure no.8: Mean Age and Weight of cases between study groups

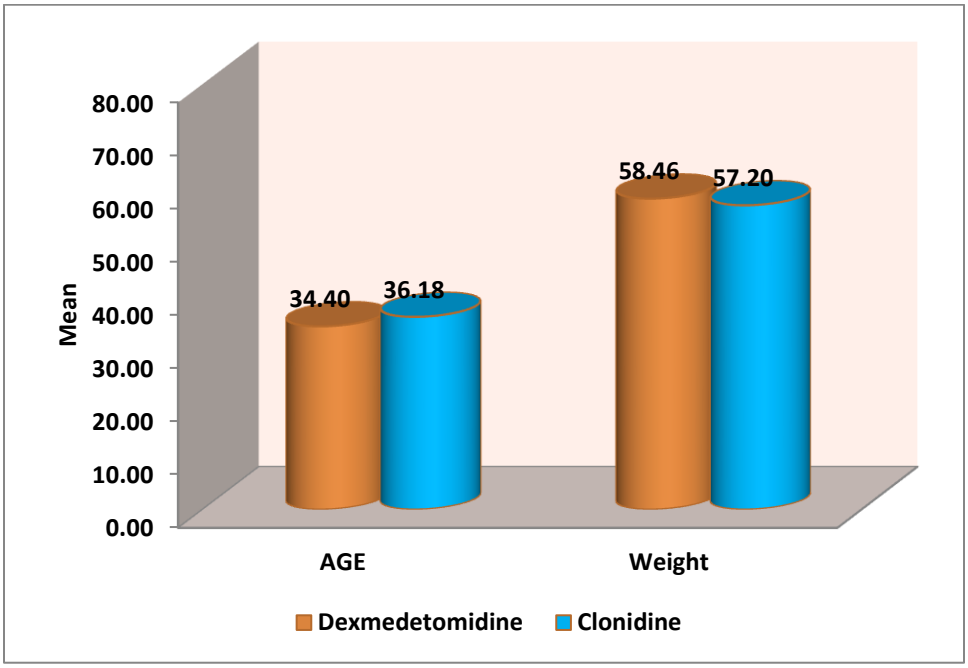
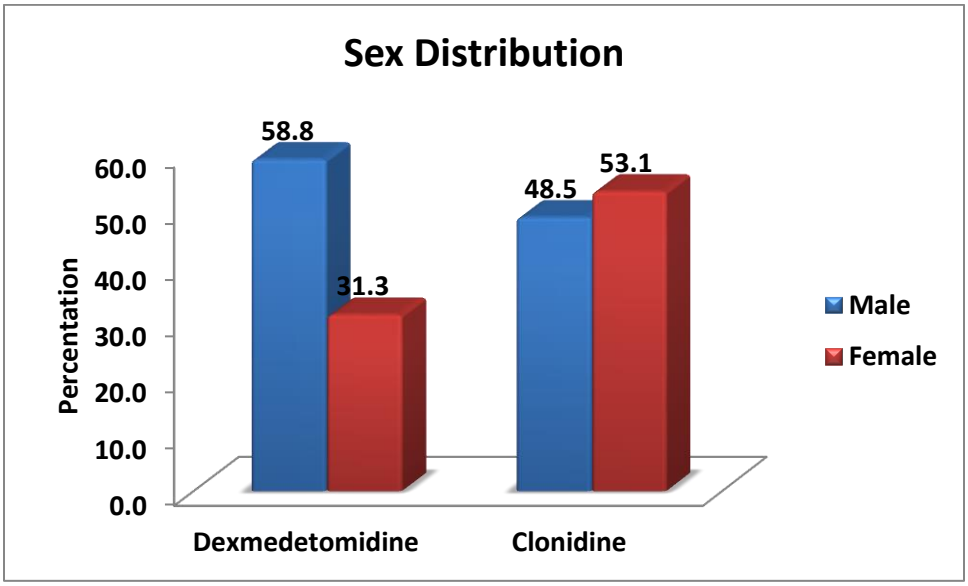


TABLE NO.4

Table: Distribution of sex between study groups

Sex	Dexmedetomidine		Clonidine		Total	p value
	N	%	N	%		
Male	40	58.8	33	48.5	68	0.115
Female	10	31.3	17	53.1	32	
Total	50	50.0	50	50.0	100	

Figure no.9: Distribution of sex between study groups



Interpretation: As shown in above tables and graphs demographic criteria like age, sex, weight are comparable in both groups. And there was no statistically significant difference between two groups. (P>0.05).

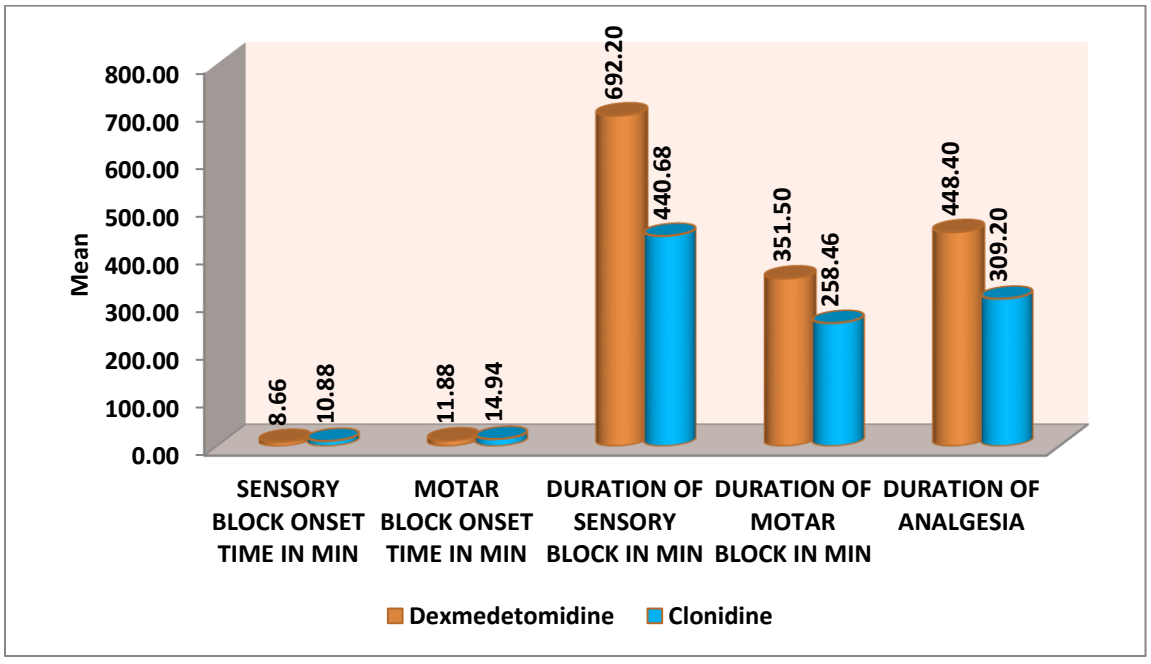
TABLE NO.5

Comparison of Mean Parameters of sensory and motor block and duration of analgesia between study groups

Variables	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
Sensory block onset time in min	8.66	1.24	10.88	1.98	<0.001*
Motor block onset time in min	11.88	2.77	14.94	1.97	<0.001*
Duration of sensory block in min	692.20	93.07	440.68	78.79	<0.001*
Duration of motor block in min	351.50	42.41	258.46	45.65	<0.001*
Duration of analgesia	448.40	91.97	309.20	37.30	<0.001*

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

Figure no.10 : Comparison of Mean Parameters of sensory and motor block and duration of analgesia between study groups



Interpretation: As shown above the graph onset of sensory block in group C in min 10.88 ± 1.98 and group D onset time is 8.66 ± 1.24 min. This difference was statistically significant. (P<0.05 is significant).

As shown above the graph onset of motor block in minutes in group C is 14.94 ± 1.97 min and group D onset time is 11.88 ± 2.77 min. This difference was statistically significant P<0.05 is significant.

The duration of sensory block in minutes in group C is 440.68 ± 78.79 min and group D is 692.20 ± 93.07min. This difference was statistically significant. P<0.05 is significant.

Duration of motor Block in minutes in group C is 258.46 ± 45.65min and duration of Motor Block in group D is 351.50 ± 42.41min. This difference was statistically significant. P<0.05 is significant.

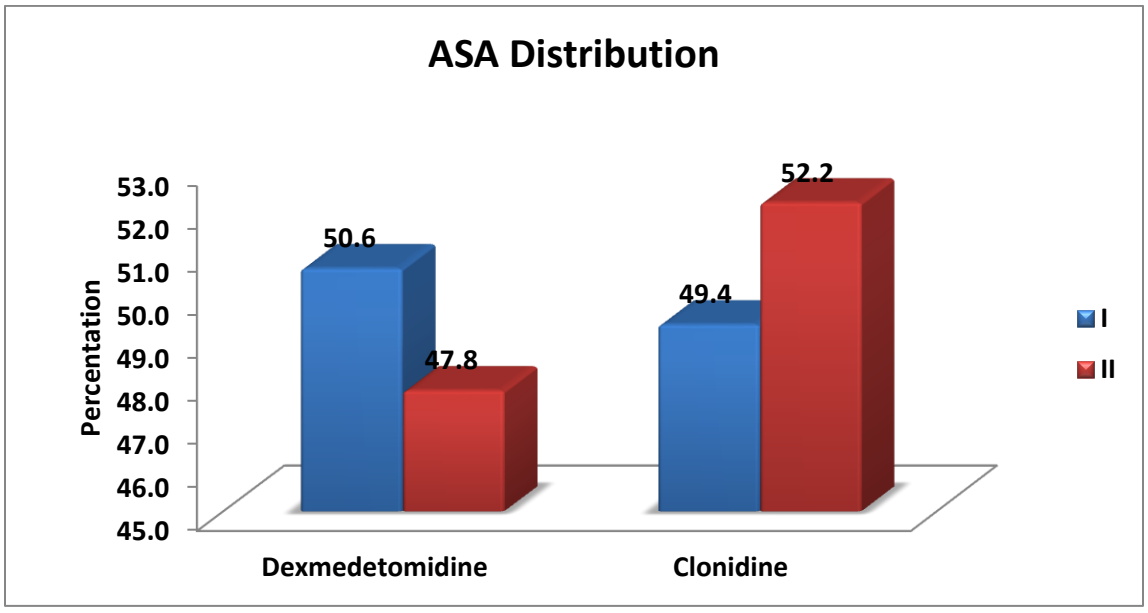
Duration of analgesia in minutes in group C 309. 20 ± 37.30 min and duration of analgesia in group D is 448.40 ± 91.97 min. This difference was statistically significant. P<0.05 is significant.

TABLE NO.6

Distribution of ASA grade between study groups

ASA	Dexmedetomidine		Clonidine		Total	p value
	N	%	N	%		
I	39	50.6	38	49.4	77	0.812
II	11	47.8	12	52.2	23	
Total	50	50.0	50	50.0	100	

Figure no.11: Distribution of ASA grade between study groups



ASA status of both the groups are comparable. There was no statistically significance difference between two groups. (P>0.05).

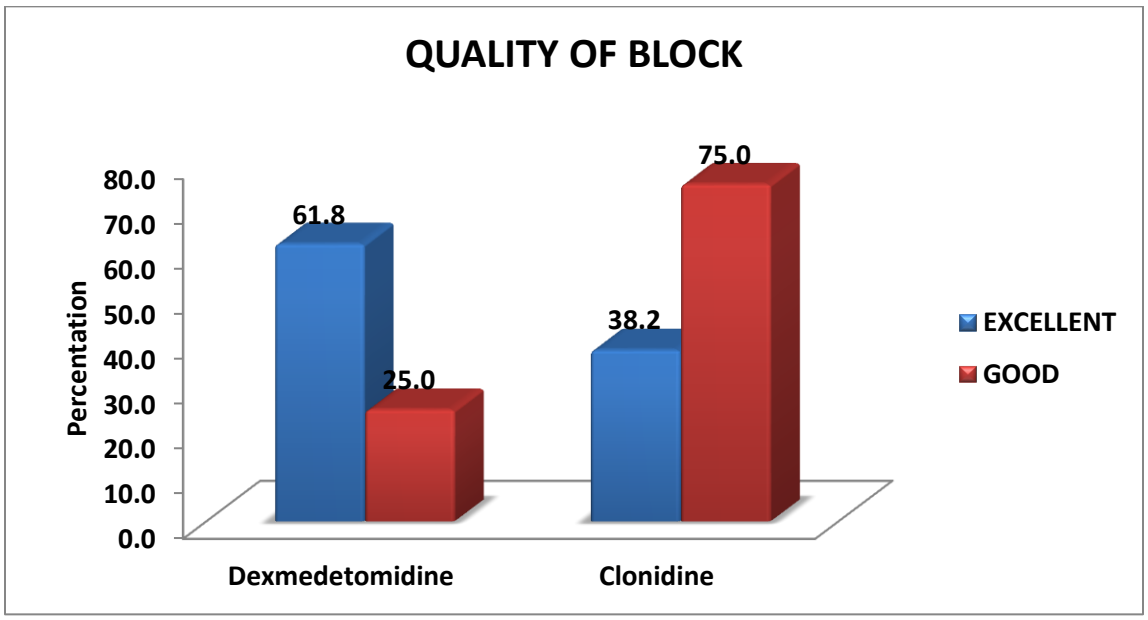
TABLE NO.7

Distribution of Quality of Block between study groups

QUALITY OF BLOCK	Dexmedetomidine		Clonidine		Total	p value
	N	%	N	%		
Excellent	42	61.8	26	38.2	68	<0.001*
Good	8	25.0	24	75.0	32	
Total	50	50.0	50	50.0	100	

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

Figure no.12: Distribution of Quality of Block between study groups



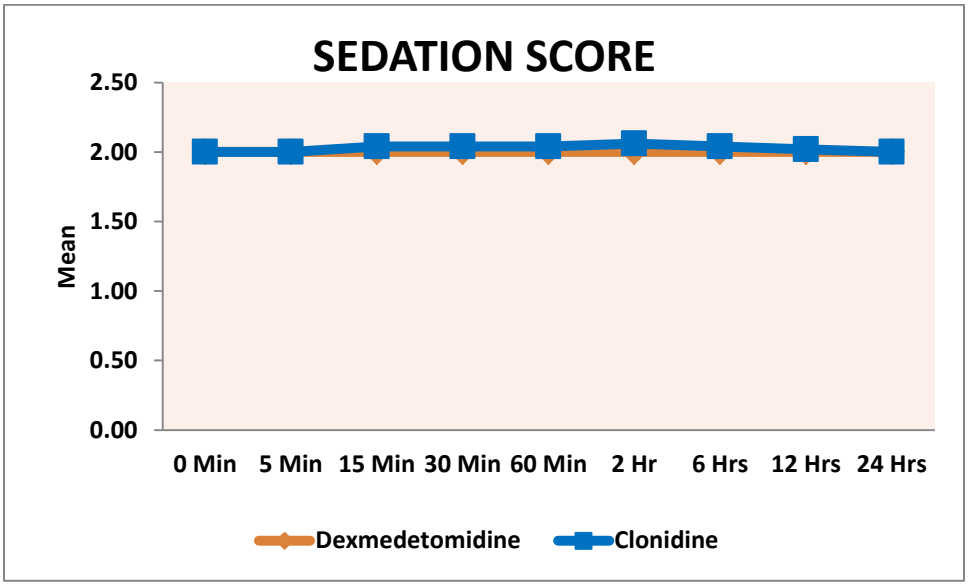
Out of 100 patients group C showed 31% excellent block and group D showed 61.8% excellent block and In group C showed 75% good block and group C showed 25% good block so there is significant P < 0.05 so group D showed most of them excellent block compared to group C.

TABLE NO.8

Comparison of Mean Sedation score between study groups

Sedation Score	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	2.00	0.00	2.00	0.00	-
5 Min	2.00	0.00	2.00	0.00	-
15 Min	2.00	0.00	2.04	0.20	0.156
30 Min	2.00	0.00	2.04	0.20	0.156
60 Min	2.00	0.00	2.04	0.20	0.156
2 Hr	2.00	0.00	2.06	0.24	0.08
6 Hrs	2.00	0.00	2.04	0.20	0.156
12 Hrs	2.00	0.00	2.02	0.14	0.32
24 Hrs	2.00	0.00	2.00	0.00	-

Figure 13: Comparison of Mean Sedation score between study groups



Sedation score of the both the Groups are comparable. There was no statistically significant difference between two groups. (P > 0.05).

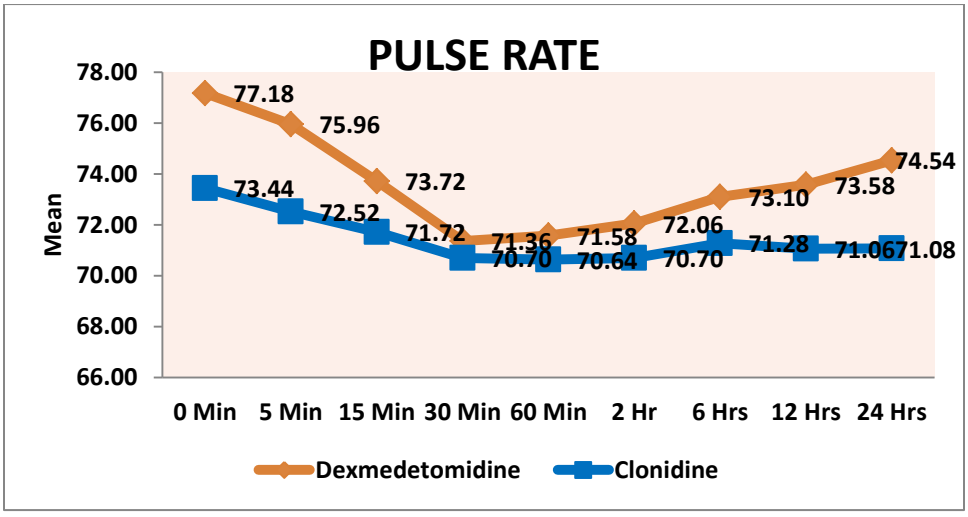
TABLE NO.9

Comparison of Mean Pulse Rate between study groups

pulse rate	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	77.18	7.20	76.44	6.31	0.167
5 Min	75.96	6.93	75.52	5.47	0.525
15 Min	73.72	7.78	71.72	6.08	0.155
30 Min	71.36	8.21	70.70	6.83	0.663
60 Min	71.58	8.66	70.64	7.01	0.552
2 Hr	72.06	8.89	70.70	7.02	0.398
6 Hrs	73.10	7.55	71.28	6.48	0.199
12 Hrs	73.58	6.50	72.06	5.73	0.092
24 Hrs	74.54	5.99	73.08	5.99	0.095

Note: *significant at 5% level of significant (p>0.05)

Figure no.14: Comparison of Mean Pulse Rate between study groups



The base line pulse rate in group C 76.44 ± 6.31 and group D 77.18 ±7.20. There was fall in Pulse rate compare to base line from 0 minute to 60 minutes which was continue up to 2 hours in group D and Lowest pulse rate was 71.36±8.21 and in group C the lowest pulse rate was 70.70.±6.23 However this fall in pulse rate was within physiological range. None of the patients developed bradycardia (pulse rate below 50). There was no ±statistical significant difference in pulse rate between two groups intra operatively and post operatively.

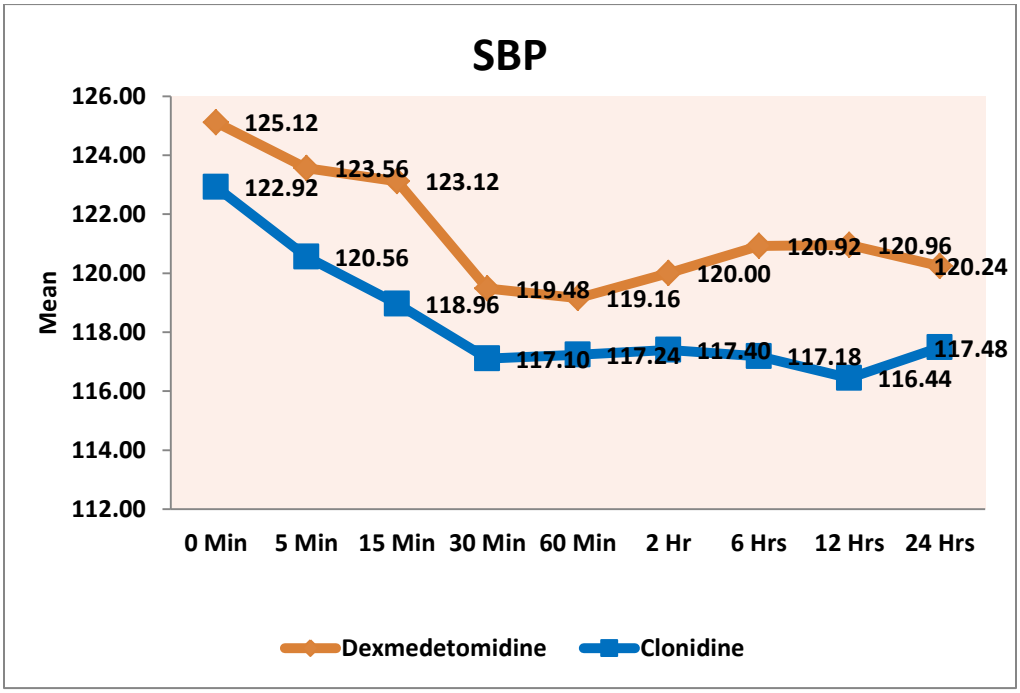
TABLE NO.10

Comparison of Mean SBP between study groups

SBP	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	125.12	10.18	122.92	9.05	0.256
5 Min	123.56	9.92	120.56	9.66	0.129
15 Min	123.12	10.37	118.96	9.38	0.038*
30 Min	119.48	12.19	117.10	9.60	0.281
60 Min	119.16	11.75	117.24	9.17	0.365
2 Hr	120.00	11.58	117.40	9.00	0.213
6 Hrs	120.92	10.37	117.18	9.88	0.068
12 Hrs	120.96	10.17	116.44	9.94	0.027*
24 Hrs	120.24	9.18	117.48	10.05	0.155

Note: *significant at 5% level of significant (p>0.05)

Figure no.15: Comparison of Mean Systolic Blood Pressure between study groups



There was statistically significant decrease in mean systolic blood pressure as compare to base line from 15 minutes to 2 hours after giving the block in group C. lowest systolic blood pressure was 117.10±9.60 at 15 minutes. group D lowest blood pressure 119.16±12.19 However this fall in systolic blood pressure was with in physiological range.

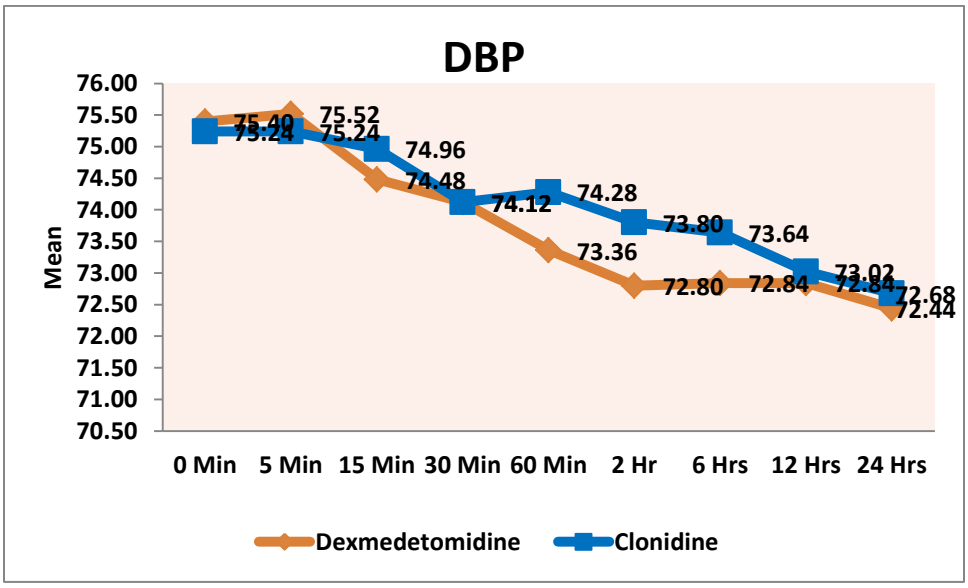
The base line systolic blood pressure in group D was 125.12 ± 10.18 and group C 122.92 ±9.05. There was no statistical significant difference in mean systolic blood pressure between the two groups. However this fall in systolic blood pressure was with in physiological range.

TABLE NO.11

Comparison of Mean DBP between study groups

DBP	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	75.40	6.62	75.24	7.35	0.909
5 Min	75.52	6.46	75.24	7.31	0.84
15 Min	74.48	6.53	74.96	5.90	0.701
30 Min	74.12	8.13	74.12	7.53	1
60 Min	73.36	6.07	74.28	6.21	0.455
2 Hr	72.80	5.84	73.80	6.34	0.414
6 Hrs	72.84	5.35	73.64	6.72	0.512
12 Hrs	72.84	5.70	73.02	5.55	0.109
24 Hrs	72.44	5.65	72.68	5.61	0.121

Figure no.16: Comparison of Mean DBP between study groups



The base line diastolic blood pressure in group C is 75.24 ± 7.35and group D 75.40 ± 6.62.There was no statistically significant difference in mean diastolic blood pressure as compare to base line.

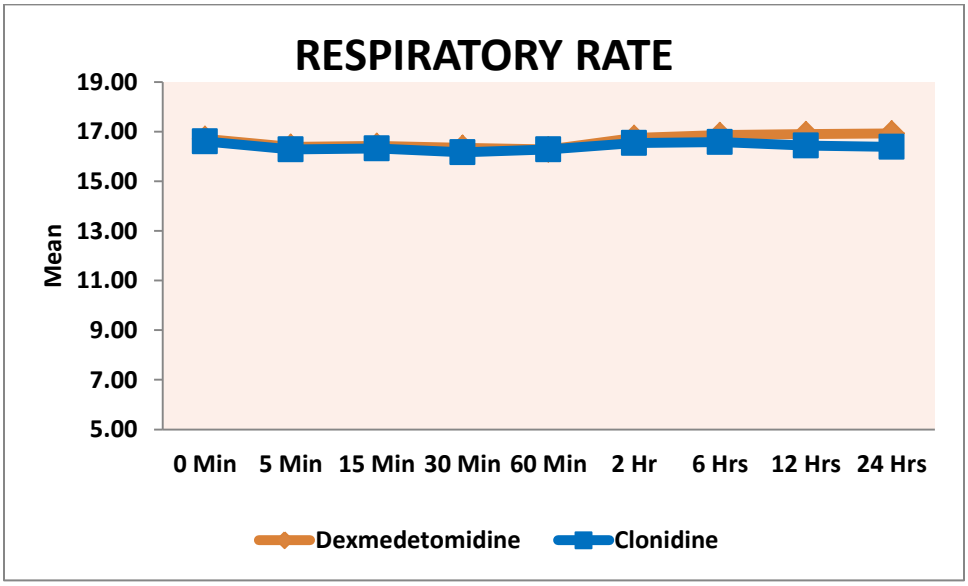
TABLE NO.12

Comparison of Mean Respiratory Rate between study groups

Respiratory rate	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	16.70	1.33	16.60	1.44	0.872
5 Min	16.38	1.54	16.28	1.33	0.841
15 Min	16.42	1.46	16.32	1.19	0.701
30 Min	16.34	1.72	16.16	1.27	0.409
60 Min	16.28	1.75	16.28	1.25	-
2 Hr	16.74	1.87	16.54	1.33	0.821
6 Hrs	16.86	1.85	16.58	1.40	0.903
12 Hrs	16.90	1.90	16.44	1.42	0.847
24 Hrs	16.92	1.98	16.38	1.19	0.701

Note: *significant at 5% level of significant (p>0.05)

Figure no.17: Comparison of Mean Respiratory Rate between study groups



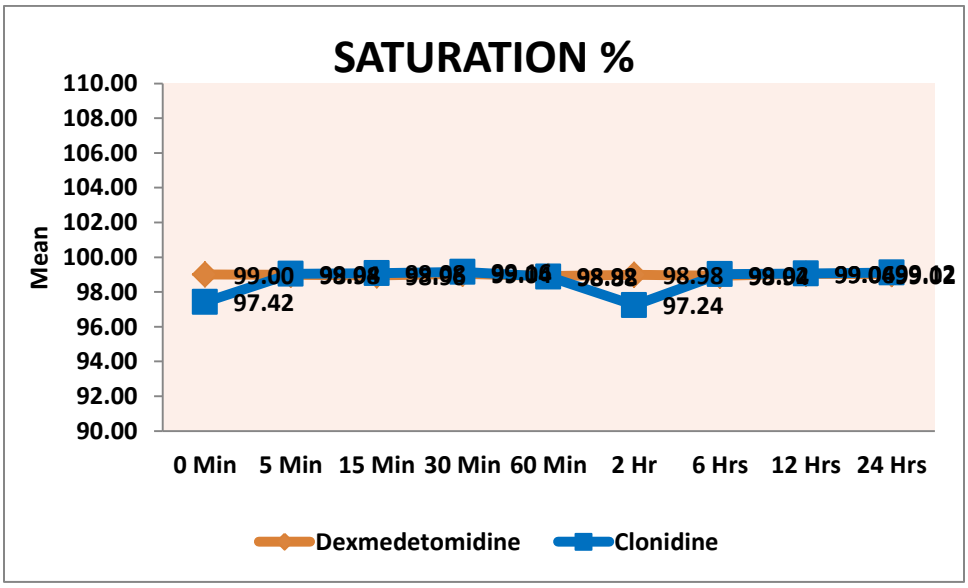
As shown above, the two Groups comparable with respect to base line respiratory rate 16.60 ± 1.44 in group C and 16.70 ± 1.33 in group D there was no statistically significant decrease in respiratory rate in both the groups comparable to base line. None of the patients in the both the groups showed respiratory depression (< 8 breaths/minutes).

TABLE NO.13

Comparison of Mean OxygenSaturation %between study groups

Saturation %	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	99.00	0.20	97.42	12.62	0.378
5 Min	98.98	0.32	99.04	0.57	0.517
15 Min	98.96	0.28	99.08	0.67	0.243
30 Min	99.04	0.28	99.16	0.51	0.149
60 Min	98.92	0.40	98.88	0.80	0.752
2 Hr	98.98	0.32	97.24	12.75	0.337
6 Hrs	98.94	0.31	99.02	0.74	0.484
12 Hrs	99.04	0.20	99.06	0.74	0.854
24 Hrs	99.02	0.25	99.12	0.59	0.274

Figure no.18: Comparison of Mean Oxygen Saturation %between study groups



As shown above, both groups comparable the baseline spo_2 in group C is 97.42±12.62 and group D is 99±00.20 .None of the patients in two groups desaturates throughout the observation period. Thus Dexmedetomidine and Clonidine in the doses given do not produce respiratory depression.

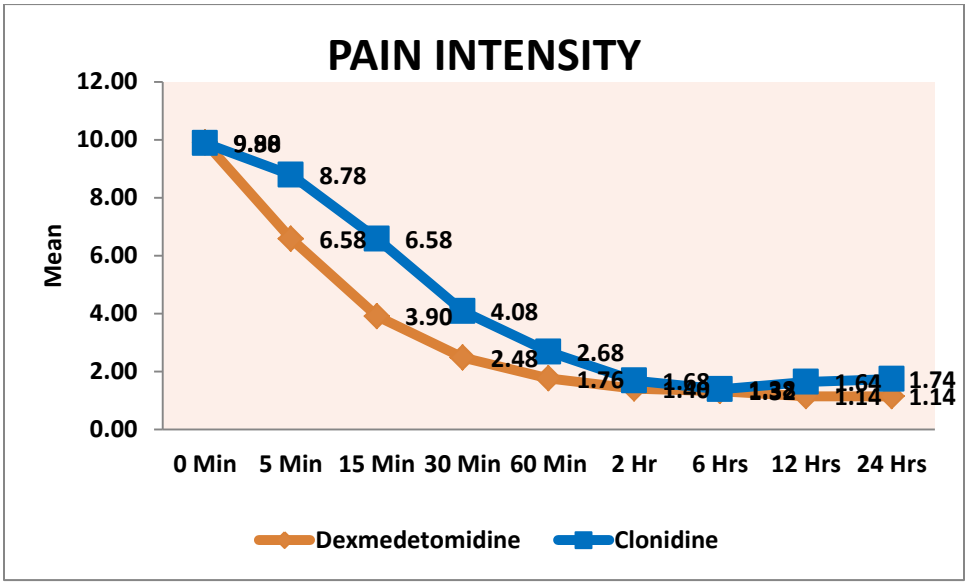
TABLE NO.14

Comparison of Mean Pain Intensity between study groups

Pain intensity	Dexmedetomidine		Clonidine		p value
	Mean	SD	Mean	SD	
0 Min	9.90	0.36	9.88	0.44	0.804
5 Min	6.58	1.81	8.78	0.58	<0.001*
15 Min	3.90	1.16	6.58	1.43	<0.001*
30 Min	2.48	1.18	4.08	1.55	<0.001*
60 Min	1.76	0.82	2.68	1.19	<0.001*
2 Hr	1.40	0.78	1.68	0.77	0.074
6 Hrs	1.32	0.68	1.38	0.49	0.615
12 Hrs	1.14	0.35	1.64	0.94	0.001*
24 Hrs	1.14	0.35	1.74	1.19	0.001*

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

Figure no.19: Comparison of Mean Pain Intensity between study groups



The base line Pain intensity showed in group C is 9.88 ± 0.44 and in group D 9.90 ± 036 and there was significant difference in two groups p < 0.05. Group D showed better in pain intensity compare to group C.

DISCUSSION

Brachial plexus block provides adequate anesthesia and post operative analgesia for all the upper limb procedures .

By giving peripheral nerve blocks, the patient remains conscious without any depression of the protective airway reflexes and it provides operative condition similar to or better than general anesthesia, with good muscle relaxation, good surgical field.

Various studies have shown that addition of several adjuvancts like Neostigmine[40,] Opioids[41,], Dexamethasone [42,] Hyaluronidas[43] Tramadol[44] and α_2 agonist like Clonidine[34] Dexmedetomidine[45] in local anaesthetic solution in peripheral nerve blocks prolonged the duration of analgesia, but the results have been inconclusive because of associated side effects or doubtful efficacy.

The study was conducted at our own institute with ethics and research committee approval. The study population included 100 ASA I and II patients undergoing upper limb surgeries. These patients were randomly divided into two groups, group D and group C of 50 patients each as per computer generated randomization

Group C received Bupivacaine 0.25% (35 cc) + inj Clonidine 1 mcg / kg.

Group D received Bupivacaine 0.25% (35 cc) + inj Dexmedetomidine 1 mcg / kg

In our study the onset of sensory block in group C was 10.88 ± 1.98 mins and that observed in group D was 8.66 ± 1.24 mins. This difference was statistically significant ($p<0.05$ is significant) which states group D has faster sensory onset time compared to group C. Kirubahar R *et al.*,[22] showed that the onset for sensory block in group D was 4.7±0.59 min and group C was 8.47+_1.04 min which was statistically

significant (p<0.001). Swami S S *et al.*,[6] showed that the onset of sensory block in group C was 2.33±1.21 mins and group D was 1.77±1.28 min which states onset of sensory block was faster in group D when compared to group C.

The mean time for onset of motor block in our study in group C was 14.94 ± 1.97 min and 11.88 ± 2.77 min in D group. The difference was found to be statistically significant. Kirubahar R *et al.*,[22] showed that onset of motor block in group C was 13.1±1.42 min and group D was 9.63±0.89 min which was statistically significant (P<0.001). Swami S S *et al.*,[6] showed onset of motor block in group D was 4.65±2.46 min and group C was 3.87±1.78 min showed group D onset time was faster compared to group C.

The mean duration of sensory block in group C was 440 ± 78.79 min and in group D was 692.20 ± 93.07 mins. This difference was statistically significant (p<0.05) duration of sensory block significantly prolonged in Dexmedetomidine group as compared to Clonidine group. Kirubahar R *et al.*,[22] showed that duration of sensory block in group C 319.1±32.74 min and group D 537.8±32.67min which is statistically significant (P<0.001). Swami S S *et al.*,[6] showed that duration of sensory of sensory block in group D was 413.97±87.31min and group C was 227.00±48.36 min which is stastically significant.

The mean duration of motor bock in C group was 258.46 ± 45.65 min and in group D was 351.50 ± 42.41 min. This difference was statistically significant. Kirubahur R et *al.*,[22] showed that duration of motor block in group C 319.1±32.74 min and group D 537.8±32.67 min which is statistically significant(P<0.001). Swami S. S *et al.*, [6] showed

that duration motor block in group D was 472.24±90.06 min and group C was 292.67±59 min which is statistically significant.

In our study showed that the duration of motor block significantly prolonged in Dexmedetomidine group as compared to Clonidine group.

Rachana G *et al*., studied about Dexmedetomidine with Bupivacine in brachial plexus block by supraclavicular approach they concluded that Dexmedetomidine provided longer duration of motor and sensory block and duration of analgesia.

The time to first rescue analgesia was 309.20 ± 37.30 min in C group and 448.40 ± 91.97 min in D group. This difference was statistically as well as clinically significant Kirubahur R *et al.,*[22] showed that duration of analgesia in group C was 375.23±32.6 min and group D 666.27±32.5 min which is statistically significant(P<0.001). Swami S S*et al.,*[6] showed that duration of analgesia in group D was 456.21+_97.99 min and group C was 289.67+_62.50 min which was statistically significant.

In our study both Dexmedetomidine and Clonidine have been found to have favourable effect on brachial plexus block characteristics though significant prolongation of postoperative analgesia is seen with Dexmedetomidine than Clonidine and Pain intensity in group D was better when compared to group C.

As our study showed group D shows 61.8% excellent block compared to group C which is 38.2% without any supplementry sedation .As shown in the above study conducted by Swami S S*et al.,*[6] group D showed 80% excellent block compared to group C 40% which was statistically significant.

Singh S *et al*.,[19] compared the effects of Clonidine (150 mcg) added to Bupivacaine with Bupivacaine alone on supraclavicular brachial plexus block. No side-

effects were observed in both the Clonidine and the control group throughout the study period.

Swami S S *et al.*,[6] compared Clonidine (1mcg/kg) group C and Dexmedetomidine (1mcg/kg) group D as an adjuvant to local anaesthetic agent in supraclavicular brachial plexus block . No side-effects (nausea, vomiting, dry mouth) were reported during the first 24 h in the post-operative period in both the groups.

In our study no patient developed any serious complications due to block procedure (pneumothorax, large hematoma , horners syndrome, prolonged nerve palsy) in both groups. Of those observed (sedation, nausea, vomiting, pruritis ,blurring of vision) their incidence was similar to that reported previously. As shown in the above graph heart rate, blood pressure and oxygen saturation are within physiological limits and patient is hemodynamically stable and not much of variability.

SUMMARY

We conducted this prospective, randomized, double blind, comparative study to assess and compare the safety and efficacy of α_2 agonists, Clonidine (1 mcg/kg) and Dexmedetomidine (1 mcg/kg) added to local anaesthetic (Bupivacaine 0.25%) as adjuvants in supraclavicular brachial plexus block for patients undergoing upper limb surgeries. The onset and duration of sensory and motor block along with post operative analgesia were compared. All 100 patients who were enrolled in the study, completed the study according to the protocol and were included in the analysis.

The two groups were as follows

Group C (n=50): Received Supraclavicular brachial plexus block with a combination of 0.25% Bupivacaine 35 ml with Clonidine. (1 mcg/kg) .

Group D (n=50): Received Supraclavicular brachial plexus block with a combination of 0.25% Bupivacaine 35ml with Dexmedetomidine. (1 mcg/kg)

The mean time for onset of sensory block in group D was 9.17±1.26 mins and that observed in group C was 11.07±2.14 mins. This difference was statistically significant

Mean duration of sensory block in group D was 690 ± 87.41 min and in group C was 470 ± 55 min. This difference was statistically significant ($p<0.05$)

Thus onset of sensory block is faster in Dexmedetomidine compared to Clonidine, both Dexmedetomidine and Clonidine prolong the duration of sensory block, more prolongation seen with Dexmedetomidine.

The mean time for onset of motor block in group D was 12.63 ± 2.18 min and 15.17±1.77 min in group C. The difference was found to be statistically significant

The mean duration of motor block in D group was 353.17 ± 41.24 min and in group C was 270.51 ± 51.61min. This difference was statistically significant .

Thus onset of motor block is faster in Dexmedetomidine compared to Clonidine, both Dexmedetomidine and Clonidine prolong the duration of motor block, more prolongation seen with Dexmedetomidine.

None of the patients in any group required intraoperative supplementation with analgesia or GA during the surgical procedure.

The duration of analgesia was 448.40 ± 91.97 min in D group and 309.20±37.30 min in C group. This difference was statistically significant.

Thus both Dexmedetomidine and Clonidine have been found to have favourable effect on duration of postoperative analgesia . Significant prolongation of duration of analgesia is seen with Dexmedetomidine as compared to Clonidine

Both Dexmedetomidine and Clonidine when used in the mentioned doses above does not produce haemodynamic instability and respiratory depression.

None of the patients in study group developed bradycardia (pulse rate<50/min), hypotension (fall in SBP> 20% baseline) or respiratory depression (RR< 8/min and SpO_2 90% on room air.)

No serious side-effects (pneumothorax, large hematoma ,horners syndrome, prolonged nerve palsy) were reported in both groups .Sedation score was evaluated in both groups but all patients were arousable and none of the patient developed respiratory complication.

CONCLUSION

From our study, the use of α-2 agonists, Dexmedetomidine (1 mcg/kg) and Clonidine (1 mcg/kg) as adjuvants to local anaesthetic solution (0.25% Bupivacaine) in supraclavicular brachial plexus block for upper limb surgeries, we conclude that faster onset of sensory and motor block is seen with Dexmedetomidine as compared to Clonidine. Duration of sensory and motor block and postoperative analgesia is significantly prolonged with Dexmedetomidine as compared to Clonidine. Haemodynamic parameters, side effects and sedation scores are comparable between the two groups.

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ANNEXURE I

ETHICAL CLERANCE CERTIFICATE

ANNEXURES II

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT : "COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK".

PRINCIPAL INVESTIGATOR:

Department of Anaesthesiology

PG GUIDE :

Associate Professor,

Department of Anaesthesiology

PURPOSE OF RESEARCH:

I have been informed that this study is **:"COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL**

PLEXUS BLOCK.”

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

PROCEDURE:

I understand that I will be doing**: “COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK”.**

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience some pain while doing supraclavicular brachial plexus and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that my/my wards participation in this study will help in finding out **“COMPARITIVE STUDY TO KNOW THE EFFICACY OF DEXMEDETOMIDINE & CLONIDINE AS AN ADJUVANT TO LOCAL ANESTHESIA IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK”.**

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator’s research file and identified only

by a code number. The code key connecting name to numbers will be kept in a separate secure location.

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

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time.

is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

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

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

REFUSAL OR WITHDRAWL OF PARTICIPATION:

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

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

INJURY STATEMENT:

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

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

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

Date:

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

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

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

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

ANNEXURE – III

PROFORMA

Patient name - Date -

Address-

I.P. number -

Age - Sex - Male/Female Weight –

Height –

Diagnosis -

Proposed Surgery -

ASA - Consent -

Medical and surgical history -

Examination in brief -:

General Physical

Examination

Vitals -: Pulse-

Respiratory rate: B.P. - Airway assessment -

Systemic examination -:

R.S. - C.V.S. -

C.N.S. - P/A -

PREOPERATIVE INVESTIGATIONS -:

Hb -

TLC/DLC -

Platelet count - BT/CT -

RBS - mg/dl

Blood Urea :

Serum Creatinine :

Chest x ray if required :

ECG :

Other investigations -:

Bupivacaine sensitivity test-

MonitorsAttached :

Pulse

B.P.

SpO2

ECG

PARAMETERS OBSERVED INTRA-OP :

Onset time of sensory blockade : (Min)

Onset time of motor blockade :(Min)

Duration of sensory blockade: (Min)

Duration of motor blockade :(Min)

Duration of Analgesia : (Min)

Quality of blockade :

Sedation Score :

Side effects : Nausea/vomiting

Bradycardia/hypotension

Sedation

MONITORING

Time	PulseRate Permin	B.P (mmHg)	Res.Rate/ min	SpO_2 %	Pain intensity .NRS(0 to 10)	Sedation score
0min						
5min						
15min						
30min						
60min						
2Hrs						
6Hrs						
12Hrs						
24Hrs						

Rescue Analgesics in post operative 24 hours

Study ends when patient demands for analgesic in postoperative period.

DATE: STAFF SIGNATURE:

KEY WORDS TO MASTER CHART

ASA	:	American Society of Anaesthesiologist
I.P. No	:	Inpatient number
Kg	:	Kilogram
Hrs	:	Hours
Min	:	Minutes
mmHg	:	Milli meter of mercury
Wt	:	Weight

Sl No	NAME	IP NO	AGE	SEX	WT	DIAGNOSIS	OPERATION	ASA	SENSORY BLOCK ONSET TIME IN MIN	MOTOR BLOCK ONSET TIME IN MIN	DURATION OF SENSORY BLOCK IN MIN	DURATION OF MOTOR BLOCK IN MIN	DURATION OF ANALGESIA	QUALITY OF BLOCK	SEDATION SCORE								PULSE RATE								
															0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs
1	DANDIRAM	13905	38Y	MALE	60KG	RT DER #	CRIF with external Fixation	I	8 MIN	12 MIN	610 MIN	400	480	EXCELLENT	2	2	2	2	2	2	2	2	2	76	72	70	72	72	70	72	70	74
2	PARVATI	15248	40Y	FEMALE	62KG	Rt. 5th PIP Dislocation	CRIF with K Wire	II	9	13	520	300	360	EXCELLENT	2	2	2	2	2	2	2	2	2	66	64	66	64	66	64	62	64	66
3	SACHIN	15073	21Y	MALE	60KG	Lt. 1st Interphalangel Joint #	CRIF with K Wire	II	10	12	670	320	360	EXCELLENT	2	2	2	2	2	2	2	2	2	82	84	86	80	82	84	86	82	84
4	GURUSANGAYYA	14050	60Y	MALE	62KG	Rt. Olecranon Implant Insitu	Implant Removal	II	7	9	780	320	480	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	68	64	72	76	74	72	70
5	SHIVANAND	97278	30Y	MALE	68KG	Rt. Colles #	Close Reduction with K Wire	I	7	9	790	310	420	EXCELLENT	2	2	2	2	2	2	2	2	2	82	80	84	78	76	74	78	76	74
6	SHARANAPPA	14506	22Y	MALE	56KG	Rt. 3rd PIP Joint #	CRIF with K Wire	I	9	15	710	350	480	GOOD	2	2	2	2	2	2	2	2	2	68	66	64	62	64	62	60	64	68
7	SHRISHAIL	14307	30Y	MALE	56KG	Rt. DER #	ORIF with Plating	I	8	13	670	330	360	EXCELLENT	2	2	2	2	2	2	2	2	2	66	64	64	62	58	56	64	68	64
8	LAXMAN	42054	55Y	MALE	62KG	Lt. Distal Radius Shaft #	ORIF with Plating	I	9	14	670	330	360	GOOD	2	2	2	2	2	2	2	2	2	84	82	84	83	84	82	83	82	83
9	SIDDANAGOUDA	414412	38Y	MALE	60KG	Rt. Colles #	CRIF with K Wire	I	9	11	640	350	480	GOOD	2	2	2	2	2	2	2	2	2	84	78	80	82	76	70	68	70	72
10	SHARANAPPA	3407	33Y	MALE	58KG	Rt. Distal Ulna #	CRIF with K Wire	I	7	10	750	440	600	EXCELLENT	2	2	2	2	2	2	2	2	2	90	86	84	67	64	76	82	84	80
11	KALLU	3347	50Y	MALE	58KG	Rt. Distal end Radius #	CR with External Fixator	I	8	12	700	425	480	EXCELLENT	2	2	2	2	2	2	2	2	2	85	86	74	66	68	80	78	80	82
12	ASHOK	15781	50Y	MALE	68KG	Lt. Hand 4th Phalanx #	CRIF with K Wire	II	7	10	750	440	480	EXCELLENT	2	2	2	2	2	2	2	2	2	76	72	74	76	74	72	72	71	70
13	SHRIDEVI	108801	50Y	FEMALE	55KG	Rt. Colles #	CRIF with K Wire	I	8	10	850	410	600	GOOD	2	2	2	2	2	2	2	2	2	74	72	74	72	74	72	70	72	70
14	SANJEEV	15661	65Y	MALE	68KG	Rt.DER implant insitu	Implant Removal	II	8	10	860	430	480	GOOD	2	2	2	2	2	2	2	2	2	74	72	70	68	72	70	72	70	74
15	MALASIDDA	15599	55Y	MALE	68KG	Rt. Ulna with Implant Insitu	Implant Removal	II	9	11	600	300	380	EXCELLENT	2	2	2	2	2	2	2	2	2	72	70	68	74	72	70	72	68	70
16	VISHAL	15575	18Y	MALE	62KG	Rt. 4th Metacarpal #	CRIF with K Wire	I	8	10	700	400	480	EXCELLENT	2	2	2	2	2	2	2	2	2	92	86	84	86	92	94	89	84	85
17	AVINASH	150091	26Y	MALE	58KG	RT DER #	CRIF with K Wire	I	8	9	630	320	360	GOOD	2	2	2	2	2	2	2	2	2	62	64	63	63	64	58	62	64	70
18	SHRIDEVI	15349	30Y	FEMALE	60KG	Rt. DER #	Ligamentaxis	I	8	13	840	420	480	EXCELLENT	2	2	2	2	2	2	2	2	2	84	82	80	78	82	84	84	86	82
19	MUSTAK	14078	18Y	MALE	62KG	Lt. Cubital Valgus Deformity	Deformity Correction Osteotomy	I	8	10	630	350	460	EXCELLENT	2	2	2	2	2	2	2	2	2	90	88	92	94	90	88	86	84	86
20	RAGHAVENDRA	183380	40Y	MALE	60KG	L DER#	CRIF with k wire	I	8	13	670	340	420	GOOD	2	2	2	2	2	2	2	2	2	82	76	66	60	58	56	58	66	72
21	LAXMI	20591	40Y	FEMALE	56 KG	Old united L Radius implant insitu	implant Removal	I	8	13	690	360	480	EXCELLENT	2	2	2	2	2	2	2	2	2	80	76	66	60	58	56	66	76	82
22	RAMANAGOUDA	16117	30Y	MALE	50 KG	Rt. DER #	CRIF with External Fixation	I	9	13	520	300	480	GOOD	2	2	2	2	2	2	2	2	2	64	66	59	62	64	66	68	64	66
23	SIDDAPPA	140101	50Y	MALE	50 KG	R DER implant in situ	implant Removal	I	8	14	760	370	420	EXCELLENT	2	2	2	2	2	2	2	2	2	76	74	68	72	70	68	66	66	70
24	REVANSIDDHA	5405	21Y	MALE	58 KG	Rt. Both Bone #	ORIF with Plating	I	8	13	840	420	540	GOOD	2	2	2	2	2	2	2	2	2	79	76	72	70	68	68	74	76	72
25	SHARAN	10004	18Y	MALE	58 KG	Rt. DER #	CRIF with K Wire	I	8	14	760	370	540	EXCELLENT	2	2	2	2	2	2	2	2	2	86	90	80	88	86	87	80	82	78
26	SANGEETA	450809	35Y	FEMALE	60 KG	Rt. DER #	CRIF with K Wire	I	10	14	760	350	540	EXCELLENT	2	2	2	2	2	2	2	2	2	84	82	78	76	82	84	84	82	81
27	SIDDARTHA	37752	42Y	MALE	56 KG	Rt. DER #	CRIF with External Fixation	I	12	14	760	300	420	EXCELLENT	2	2	2	2	2	2	2	2	2	76	74	76	72	74	72	70	68	72
28	MALLIKARJUN	39643	30Y	MALE	58 KG	Rt. Distal ulna #	ORIF with Plating	I	9	15	710	350	480	GOOD	2	2	2	2	2	2	2	2	2	68	66	64	58	56	68	70	68	70
29	MALKAPPA	38403	22Y	MALE	52 KG	Lt. United Old Implant Insitu	Implant Removal	I	9	10	800	360	420	GOOD	2	2	2	2	2	2	2	2	2	78	76	74	72	78	74	76	74	72
30	MALLAPPA	17640	18Y	MALE	52 KG	1st 4th Metacorpal #	CRIF with K Wire	I	8	13	520	300	480	EXCELLENT	2	2	2	2	2	2	2	2	2	76	72	74	74	76	72	74	76	74
31	JAKIR	42116	40Y	MALE	58 KG	Rt.ulna #	ORIF with plating	I	9	15	710	350	600	EXCELLENT	2	2	2	2	2	2	2	2	2	80	84	88	84	80	78	76	78	80
32	SHANTAMMA	4891	30Y	FEMALE	60 KG	Rt. Elbow Dislocation	Open reduction with plating	I	8	14	760	320	540	GOOD	2	2	2	2	2	2	2	2	2	76	78	64	68	67	72	74	72	74
33	KALLA	3342	50Y	MALE	64 KG	L Both bone #	ORIF with plating	II	10	12	670	320	420	EXCELLENT	2	2	2	2	2	2	2	2	2	82	80	76	70	66	68	72	74	72
34	ARUN	17234	30Y	MALE	58 KG	Rt. DER #	CR with External Fixator	I	11	18	630	310	420	EXCELLENT	2	2	2	2	2	2	2	2	2	78	80	82	84	80	76	74	74	76
35	ABHISHEK	17172	18Y	MALE	58 KG	Rt. Old United Radius Implant Insitu	Implant Removal	I	9	11	650	340	480	GOOD	2	2	2	2	2	2	2	2	2	82	80	76	72	70	78	76	74	72
36	DAYANAND	17082	28Y	MALE	60/9	Rt. Old United Radius Implant Insitu	Implant Removal	I	7	9	790	310	540	EXCELLENT	2	2	2	2	2	2	2	2	2	72	70	68	66	68	66	68	62	66
37	MALLAPPA	16968	18Y	MALE	46/9	Lt. Distal 5th Phalanx #	CRIF with K Wire	I	8	11	790	380	460	GOOD	2	2	2	2	2	2	2	2	2	68	66	64	70	72	68	66	68	64
38	SIKANDAR	16471	30Y	MALE	56 KG	Lt. both Bone #	ORIF with Plating	I	9	15	710	350	420	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	68	64	66	64	67	68	69
39	NEELAMMA	39936	55Y	FEMALE	58 KG	Lt. Forearm Implant Insitu	Implant Removal	II	9	11	640	340	360	EXCELLENT	2	2	2	2	2	2	2	2	2	78	80	76	70	70	72	76	80	82
40	ASHOK	36296	35Y	MALE	56 KG	Lt. BB Forearm #	ORIF with Plating	I	9	13	520	300	360	EXCELLENT	2	2	2	2	2	2	2	2	2	78	76	74	72	78	80	82	78	82
41	CHANDRASHEKAR	2797	42Y	MALE	54 KG	Lt. DER #	ORIF with K Wire	II	8	11	790	380	420	GOOD	2	2	2	2	2	2	2	2	2	90	88	86	67	70	72	66	68	74
42	ANNAPPA	16187	48Y	MALE	64 KG	Lt. DER #	ORIF with Plating	II	7	9	790	310	360	EXCELLENT	2	2	2	2	2	2	2	2	2	80	78	76	74	82	80	84	82	83
43	NAVYA	18664	24Y	FEMALE	56 KG	Rt both bone#	ORIF with plating	I	9	13	570	360	420	EXCELLENT	2	2	2	2	2	2	2	2	2	70	72	68	64	60	58	64	68	72
44	SHIVANAND	97278	30Y	MALE	60 KG	Rt. Colles #	CR with Casting	I	13	13	570	360	480	EXCELLENT	2	2	2	2	2	2	2	2	2	86	84	86	84	82	84	86	84	86
45	GURUBAI	8884	50Y	FEMALE	55 KG	Lt. DER #	CRIF with External Fixation	I	10	12	670	320	540	EXCELLENT	2	2	2	2	2	2	2	2	2	72	78	76	68	66	72	74	78	76
46	ASIF	9744	25Y	MALE	65 KG	Rt. Ulna #	ORIF with Nailing	I	11	18	630	310	480	EXCELLENT	2	2	2	2	2	2	2	2	2	72	70	68	68	72	72	68	74	72
47	IRAGOUD	9258	60Y	MALE	62 KG	Rt. DER #	CRIF with K Wire	II	9	11	650	340	420	GOOD	2	2	2	2	2	2	2	2	2	74	72	68	62	58	56	64	68	72
48	RAVINDRA	8801	25Y	MALE	62 KG	Elbow Dislocation	ORIF with K Wire	I	9	10	510	350	480	EXCELLENT	2	2	2	2	2	2	2	2	2	72	74	68	62	60	72	70	72	70
49	MOHAN	55698	30Y	MALE	56 KG	Rt. Elbow Dislocation	CRIF with K Wire	I	8	11	750	440	480	EXCELLENT	2	2	2	2	2	2	2	2	2	79	80	76	68	66	64	72	76	78
50	CHANDRASHEKAR	17306	24Y	MALE	50 KG	Rt. 3rd Middle Phalanx #	CRIF with K Wire	I	8	10	650	320	420	EXCELLENT	2	2	2	2	2	2	2	2	2	66	68	72	76	74	78	76	72	74

SYSTOLIC BLOODPRESSURE mm Hg.									DIASTOLIC BLOODPRESSURE (mmHg)									RESPIRATORY RATE									SATUARATION %									PAIN INTENSITY								
0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs
130	136	130	130	130	132	130	126	130	80	84	80	70	74	76	70	74	70	18	18	16	16	16	16	18	18	16	99	99	99	99	99	99	99	99	99	10	8	5	2	2	1	1	1	1
120	122	124	120	124	124	110	120	110	70	70	70	70	70	70	70	72	74	18	18	16	16	15	16	16	18	18	99	99	99	99	99	99	99	99	99	10	5	4	2	1	1	1	1	1
120	122	124	120	118	120	122	120	120	76	72	76	70	70	72	76	70	80	18	18	16	16	15	16	16	15	15	99	99	99	99	99	99	99	99	99	10	8	4	2	2	1	1	1	1
130	132	128	120	112	110	110	110	120	80	82	86	80	70	72	70	80	70	17	16	16	18	16	17	18	18	17	99	99	99	100	99	99	99	99	99	10	5	4	2	2	2	2	1	1
130	130	136	130	126	130	126	130	130	70	72	70	80	70	80	70	82	70	18	16	18	18	17	17	16	16	16	99	99	99	99	99	99	99	99	99	10	8	5	2	2	2	2	1	1
140	130	136	130	132	130	136	130	130	72	78	74	70	72	70	72	70	70	18	16	16	15	15	16	16	18	18	99	99	99	99	99	99	99	99	99	10	9	5	4	2	1	1	1	1
100	104	100	100	96	98	100	110	120	70	72	70	70	70	72	70	72	70	18	18	16	16	16	18	18	17	17	99	99	99	99	99	99	99	99	99	10	5	2	2	1	1	1	1	1
140	144	150	130	130	140	140	140	130	90	90	80	90	80	80	82	70	70	18	18	18	18	16	18	17	17	16	99	99	99	99	99	99	99	99	99	10	5	3	3	2	2	1	1	1
140	140	130	140	130	130	120	110	110	80	90	90	80	80	70	70	70	80	15	14	15	14	16	18	16	17	18	99	99	99	99	99	99	98	99	99	10	8	5	4	3	1	1	1	1
130	120	120	110	110	120	110	120	120	80	82	80	84	80	70	74	70	74	15	14	15	14	16	18	16	18	16	98	99	99	99	98	99	98	99	99	9	8	5	4	3	3	3	2	2
130	130	126	120	110	104	102	100	120	80	80	78	76	70	80	70	70	80	18	20	20	19	18	19	20	20	20	99	99	99	98	98	99	98	99	99	8	8	5	5	2	2	2	2	2
130	132	130	130	128	130	130	130	136	80	80	84	80	80	70	80	80	80	18	18	16	16	15	16	14	14	16	99	99	99	99	99	99	99	99	99	10	5	4	2	2	1	1	1	1
124	120	124	116	120	130	120	130	120	78	80	78	72	70	68	72	74	76	15	14	15	14	14	13	15	16	15	99	98	99	99	99	98	99	99	99	10	10	5	5	3	3	2	1	1
124	120	124	120	110	120	120	120	120	70	80	70	70	72	74	70	72	74	16	18	18	16	17	17	18	16	16	99	99	99	99	99	99	99	99	99	10	5	4	1	1	1	1	1	1
140	130	130	140	130	120	126	128	128	90	80	80	90	82	80	84	84	86	15	14	15	14	16	16	18	16	20	99	99	99	99	99	99	99	99	99	10	8	5	4	3	1	1	1	1
110	110	100	100	104	100	100	100	100	70	72	60	64	60	62	64	70	68	16	16	18	18	19	20	18	19	19	99	99	99	99	99	99	99	99	99	9	8	5	4	3	4	4	2	2
140	130	120	120	120	130	130	130	130	80	70	70	80	70	70	70	80	70	18	17	18	16	15	17	17	18	15	99	99	99	99	99	99	99	99	99	10	5	3	1	1	1	1	1	1
130	130	130	136	130	136	130	126	124	80	70	80	82	90	82	80	80	80	17	18	16	16	14	16	16	15	16	99	99	99	99	99	99	99	99	99	10	5	3	1	1	1	1	1	1
120	116	110	112	110	112	110	112	110	70	70	70	72	70	72	70	70	70	18	18	16	16	16	15	15	15	15	99	99	99	99	99	99	99	99	99	10	5	2	2	2	1	1	1	1
126	128	130	124	126	120	120	124	120	70	80	70	72	80	70	72	74	80	18	18	15	16	18	15	15	18	18	99	99	99	99	99	99	99	99	99	10	5	2	1	1	1	1	1	1
126	128	120	126	130	126	126	120	126	70	80	80	70	70	80	70	70	70	18	16	15	16	18	17	18	17	18	99	99	99	99	99	99	99	99	99	10	5	2	1	1	1	1	1	1
110	106	104	100	96	92	110	110	100	70	70	70	60	60	60	70	72	68	17	16	19	20	22	20	18	16	15	99	99	99	99	99	99	99	99	99	10	5	2	1	1	1	1	1	1
130	130	130	130	120	120	120	120	130	80	70	74	80	70	80	70	80	70	17	16	17	18	17	17	18	19	18	99	99	99	99	99	99	99	99	99	10	5	2	2	2	1	1	1	1
110	106	104	100	96	92	110	110	100	70	70	70	60	60	60	70	72	68	17	16	19	20	22	20	18	16	15	99	99	99	99	99	99	99	99	99	10	5	2	1	1	1	1	1	1
130	130	130	130	120	120	120	120	130	80	70	74	80	70	80	70	80	70	17	16	17	18	17	17	18	19	18	99	99	99	99	99	99	99	99	99	10	5	2	2	2	1	1	1	1
130	120	120	130	120	120	130	120	110	70	90	72	70	70	70	70	70	70	17	18	17	20	18	20	20	18	19	99	99	99	100	99	99	99	99	99	10	5	4	4	1	1	1	1	1
130	120	110	90	90	100	110	100	120	80	70	70	60	62	60	60	70	70	15	16	17	18	18	19	20	22	20	99	100	100	100	100	100	100	100	100	10	8	3	3	1	1	1	1	1
110	110	120	100	110	110	120	120	120	70	72	70	70	70	76	70	80	70	17	18	17	19	20	17	17	18	19	99	99	98	99	97	98	99	99	99	10	7	5	2	2	1	1	1	1
100	100	100	100	100	100	100	100	120	70	60	66	64	70	66	70	66	70	18	17	18	16	15	16	16	15	16	99	99	99	99	99	99	99	100	100	10	9	5	3	2	2	1	1	1
100	100	110	120	120	130	130	140	130	60	70	70	70	70	70	80	90	80	18	17	18	17	19	20	19	21	20	99	99	99	99	99	100	99	99	99	10	5	4	2	2	2	2	2	2
130	130	130	130	130	130	130	120	120	90	80	70	90	80	70	70	80	90	18	16	18	17	18	19	20	17	22	99	99	99	99	99	99	99	99	99	10	3	1	1	1	1	1	1	1
130	130	134	130	136	126	130	132	126	80	70	76	80	84	76	80	82	86	18	16	16	15	16	14	16	16	16	99	99	99	99	99	99	99	99	99	10	9	5	4	2	1	1	1	1
130	130	120	110	130	130	130	120	130	70	80	70	70	70	80	70	80	70	17	18	17	20	18	20	20	18	19	99	100	99	99	99	99	99	99	99	10	5	3	1	1	1	1	1	1
110	120	130	120	130	120	130	120	110	70	70	70	70	70	70	70	70	70	18	17	20	17	18	20	22	20	20	99	99	99	99	99	99	99	99	99	10	9	5	3	1	1	1	1	1
130	130	130	130	130	130	130	130	130	70	80	70	70	80	90	80	90	80	15	15	15	15	14	15	16	16	16	99	99	99	99	99	99	99	99	99	10	6	5	1	1	1	1	1	1
130	132	130	128	130	130	130	120	126	70	70	72	70	72	74	72	78	72	18	16	16	18	18	16	16	18	18	99	99	99	99	99	99	99	99	99	10	5	2	1	1	1	1	1	1
120	122	120	116	110	112	110	110	112	70	72	70	70	70	72	70	72	74	16	18	18	17	18	18	16	16	16	99	99	99	99	99	99	99	99	99	10	5	4	2	1	1	1	1	1
120	120	120	104	100	100	100	100	100	70	72	74	70	70	76	74	72	70	18	16	16	18	16	18	16	18	16	99	99	99	99	99	99	99	99	99	10	5	4	2	2	1	1	1	1
130	128	130	128	132	126	130	132	126	76	72	72	70	78	80	72	78	80	16	18	16	18	16	16	18	18	16	99	99	99	99	99	99	99	99	99	10	4	3	2	1	1	1	1	1
130	130	130	130	130	120	110	120	110	80	70	80	90	80	70	70	70	70	15	16	17	17	16	18	18	20	20	99	99	99	99	99	99	99	99	99	10	9	5	2	2	2	2	2	2
126	116	120	114	110	126	124	126	124	80	70	82	84	80	70	80	70	82	15	16	15	17	17	18	18	19	19	99	99	99	99	99	99	99	99	99	10	8	5	4	3	1	1	1	1
126	120	110	116	120	120	130	120	110	80	82	70	70	70	72	70	74	70	18	17	18	16	15	16	17	17	17	99	99	99	99	99	99	99	99	99	10	5	4	1	1	1	1	1	1
124	120	116	120	124	120	120	120	120	78	80	78	76	72	68	72	74	80	15	16	14	15	15	14	15	14	14	99	98	99	99	99	99	99	99	99	10	8	5	4	4	3	3	2	2
130	130	132	138	130	136	120	126	124	70	80	84	86	80	84	80	84	82	16	17	16	15	16	17	18	16	17	99	99	99	99	99	99	99	99	99	10	9	5	3	1	1	1	1	1
110	120	130	100	100	110	120	110	100	70	70	70	70	70	70	70	70	70	15	14	15	14	15	14	15	15	15	99	99	99	99	99	99	99	99	99	10	5	2	2	1	1	1	1	1
130	130	130	120	120	120	130	130	130	90	80	90	90	80	70	90	80	70	15	14	15	14	14	15	15	14	14	100	99	98	99	99	99	99	99	99	10	9	4	3	1	1	1	1	1
130	120	120	110	120	110	120	130	120	70	70	70	70	70	70	70	80	80	15	14	15	15	14	15	14	15	15	99	98	99	99	98	99	98	99	99	10	8	4	2	2	1	1	1	1
130	120	130	130	120	130	120	130	130	70	70	80	70	70	70	70	80	70	15	14	15	14	15	15	14	14	14	99	99	98	99	99	98	99	99	98	10	6	5	2	2	2	2	1	1
110	100	110	100	100	100	110	120	110	70	70	70	60	70	70	70	70	70	15	14	15	15	15	14	14	16	16	99	99	99	99	99	99	99	99	99	10	7	4	3	2	1	1	1	1
130	130	110	110	120	120	130	130	120	80	70	60	62	80	70	80	70	70	15	14	15	14	15	14	15	14	15	99	99	99	99	99	99	99	99	99	10	9	5	4	2	1	1	1	1
130	130	128	126	124	120	128	126	124	80	80	78	76	78	76	76	76	76	15	16	14	14	14	15	15	15	15	99	99	99	99	99	99	99	99	99	9	8	4	4	4	4	3	2	2
130	130	130	120	130	130	126	130	126	80	82	80	76	72	70	70	70	70	18	16	18	16	14	16	16	16	14	99	99	99	99	99	99	99	99	99	10	5	4	2	1	1	1	1	1

Sl No	NAME	IP NO	AGE	SEX	WT	ASA	DIAGNOSIS	OPERATION	SENSORY BLOCK ONSET TIME IN MIN	MOTOR BLOCK ONSET TIME IN MIN	DURATION OF SENSORY BLOCK IN MIN	DURATION OF MOTOR BLOCK IN MIN	DURATION OF ANALGESIA	QUALITY OF BLOCK	SEDATION SCORE							PULSE RATE									BP	
															0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min
1	GOVIND	28505	36y	MALE	58	II	R 2nd Metacrapal #	Tendon Repair with ex.Fixator	10	16	450	280	300	GOOD	2	2	2	2	2	2	2	2	2	78	76	80	82	76	78	76	76	76	130	120
2	KRISHNARAJ	5397	29Y	MALE	56	I	Proxi Implant Insitu in Ulna	Implant Removal	12	15	450	210	340	EXCELLENT	2	2	2	2	2	2	2	2	2	88	84	86	88	88	86	86	86	84	130	124
3	MAHESHWARI	52598	18Y	FEMALE	56	I	Right Thumb #	Tendon Repair	12	15	460	210	320	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	74	72	74	74	72	74	72	120	120
4	GURUNATH	54718	44Y	MALE	60	I	Right colles#	CRIF with K Wire	11	13	450	33	300	EXCELLENT	2	2	2	3	3	3	2	2	2	78	76	74	74	76	74	74	76	74	130	120
5	SHARAN	6057	35Y	MALE	58	I	Left DER #	CRIF with K Wire	9	14	480	230	380	GOOD	2	2	2	2	2	3	2	2	2	80	78	76	80	78	78	76	78	78	124	120
6	MAHADEVI	6477	31Y	FEMALE	56	I	Ulna Shaft #	CRIF with Nailing	9	12	420	280	310	EXCELLENT	2	2	2	2	2	2	2	2	2	78	76	74	76	74	74	76	76	76	118	110
7	MALLANNA	11687	46Y	MALE	58	I	Old united Implant Insitu ulna	Implant Removal	10	13	410	280	310	EXCELLENT	2	2	3	3	2	2	3	2	2	70	72	74	72	72	74	76	74	72	116	116
8	SIDDAYYA	26143	60Y	MALE	68	II	Left DER #	CR with Casting	11	13	380	260	280	GOOD	2	2	2	2	2	2	2	2	2	70	68	66	64	62	60	64	66	68	120	120
9	MAHADEVI	26272	52Y	FEMALE	56	I	Left DER #	CRIF with External Fixator	10	15	500	270	280	GOOD	2	2	2	2	2	2	2	2	2	64	66	67	64	62	64	66	64	66	100	100
10	LINGARAJ	215748	40Y	MALE	60	I	R DER#	ORIF WITH Plating	17	20	440	300	320	GOOD	2	2	2	2	2	2	2	2	2	74	76	72	74	76	72	76	72	76	140	142
11	YAMANAPPA	38391	51Y	MALE	56	II	R Distal Radius #	ORIF with Plating	9	14	500	260	280	EXCELLENT	2	2	3	3	2	2	3	3	2	78	72	76	74	75	76	76	77	77	120	122
12	RAJESHWARI	441270	35Y	FEMALE	60	I	R Colles #	CRIF with K Wire	13	17	410	240	300	GOOD	2	2	2	2	2	2	2	2	2	72	70	68	66	66	76	72	70	68	110	120
13	KAMALA	35090	50Y	FEMALE	45	II	Left Distal end Radius #	CRIF with K Wire	9	14	400	240	300	GOOD	2	2	3	2	2	2	2	2	2	76	74	78	74	76	78	77	76	74	124	126
14	PRATEEK	39794	19y	MALE	58	I	Right Distal end Radius #	CRIF with K Wire	13	17	410	240	280	GOOD	2	2	3	3	2	2	2	2	2	68	66	64	66	66	64	66	66	62	130	130
15	SIDDARAM	19025	18Y	MALE	56	I	L Forearm Both Bone #	ORIF with Nailing	10	16	480	230	280	EXCELLENT	2	2	2	2	2	2	2	2	2	78	74	76	72	70	74	72	70	72	130	126
16	VINAYAK	293738	55y	MALE	56	II	R Old United Ulna Implant Insitu	Implant Removal	9	16	500	230	360	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	70	74	72	74	76	74	76	124	122
17	GURUBASAPPA	27637	45Y	MALE	58	I	3rd Proximal Interphalangeal Joint #	ORIF with Jess Wire	9	14	500	260	320	EXCELLENT	2	2	2	2	2	2	2	2	2	72	70	76	72	74	74	76	74	72	120	124
18	NEELAMMA	19229	35Y	FEMALE	56	I	R 3rd Metacarpal #	CRIF with K Wire	10	13	34	240	280	EXCELLENT	2	2	2	2	2	2	2	2	2	92	88	86	88	84	86	88	84	88	130	128
19	SUBASH	18810	23Y	MALE	62	I	L Olecranon #	CRIF with TBW	10	13	340	240	260	GOOD	2	2	2	2	2	2	2	2	2	72	70	67	66	65	66	65	66	67	120	110
20	BASAVARAJ	19386	49y	MALE	62	II	R DER#	CRIF with external Fixator	13	17	410	240	280	GOOD	2	2	2	3	3	3	2	2	2	70	72	73	74	72	68	72	70	72	130	126
21	AKSHAY	18753	20Y	MALE	52	I	L Both Bone #	ORIF with Plating	9	15	470	240	200	GOOD	2	2	3	3	2	2	3	2	2	66	67	64	66	66	62	64	66	62	114	120
22	ANIL	35652	18y	MALE	46	I	R Radius # with Nail Insitu	Implant Removal	11	13	450	330	300	GOOD	2	2	2	2	2	2	2	2	2	74	70	68	56	54	52	64	68	64	140	130
23	YASH	122246	25Y	MALE	56	I	R Both Bone #	ORIF with Plating	10	14	340	240	280	GOOD	2	2	2	2	2	2	2	2	2	78	79	76	72	74	76	72	74	76	120	118
24	SHANTAMMA	17924	40Y	FEMALE	58	I	R Colles #	CRIF with K Wire	10	15	500	270	300	EXCELLENT	2	2	2	2	2	2	2	2	2	68	66	64	58	56	62	60	62	64	130	113
25	VISHNU	17782	35Y	MALE	57	I	R Thumb Tendon Repair	Tendon Repair	9	14	480	230	320	GOOD	2	3	2	2	2	3	2	2	2	64	66	68	66	67	66	64	67	68	120	130
26	KIRAN	26504	30Y	MALE	58	I	R DER #	CRIF with K Wire	12	16	510	300	360	EXCELLENT	2	2	3	3	2	2	3	2	2	67	68	64	66	64	66	66	62	66	120	130
27	SANTOSH	26168	48Y	MALE	56	I	L DER #	CRIF with External Fixator	9	12	470	300	330	EXCELLENT	2	2	2	2	2	2	2	2	2	67	66	68	67	66	65	64	66	64	110	104
28	VASANTH	13268	19Y	MALE	56	I	Crush Injury Over 3rd Finger	Debridement & Suturing	9	14	380	260	340	EXCELLENT	2	2	2	2	2	2	2	2	2	78	76	74	76	76	74	78	78	78	120	110
29	PUSHPA	14601	26Y	FEMALE	52	I	CLW Over Arm	Debridement & Suturing	14	16	410	280	300	EXCELLENT	2	2	2	2	2	2	2	2	2	66	68	64	66	68	68	68	70	68	122	120
30	SUMATI	144225	48Y	FEMALE	56	II	L DER #	CRIF with K Wire	12	14	350	210	280	GOOD	2	2	2	2	2	2	2	2	2	78	74	76	74	76	78	76	76	76	110	120
31	CHANDU	17703	20Y	MALE	66	I	3rd Proximal Phalanx #	CRIF with K Wire	9	15	500	280	300	EXCELLENT	2	2	2	2	2	2	2	2	2	62	66	64	66	62	68	67	68	66	110	110
32	SHEELA	18070	53Y	FEMALE	56	II	R Distal end Radius #	ORIF with Plating	9	14	500	300	320	GOOD	2	2	2	2	2	2	2	2	2	76	70	68	64	66	64	62	66	68	130	128
33	ASHOK	17750	20Y	MALE	60	I	R Distal end Radius #	CRIF with K Wire	10	16	500	270	300	GOOD	2	2	2	2	2	2	2	2	2	74	72	68	64	66	64	62	66	68	120	110
34	HUSENSAB	18018	38Y	MALE	62	II	R Metacarpal #	CRIF with K Wire	10	16	500	230	280	GOOD	2	2	2	2	2	2	2	2	2	70	76	68	66	64	70	76	74	68	120	114
35	GANGABAI	21502	56Y	FEMALE	60	I	Colles #	CRIF with k Wire	9	14	520	230	300	GOOD	2	2	2	2	2	2	2	2	2	66	67	68	66	67	66	67	68	67	120	110
36	AKASH	19234	19Y	MALE	51	I	L DER #	CRIF with K Wire	12	16	500	270	300	EXCELLENT	2	2	2	2	2	2	2	2	2	68	66	64	68	66	64	66	64	68	126	128
37	REKHA	216991	48Y	FEMALE	58	II	Both Bone #	ORIF with Plating	11	13	450	330	360	EXCELLENT	2	2	2	2	2	2	2	2	2	73	74	75	70	72	74	73	72	70	130	130
38	SHARADA	12379	40Y	FEMALE	59	I	L Metacarpal#	CRIF with K Wire	15	20	350	240	260	EXCELLENT	2	2	2	2	2	2	2	2	2	80	80	82	78	80	78	74	76	78	120	118
39	JAREENA	179256	40Y	FEMALE	55	I	R Metacarpal #	CRIF with K Wire	10	14	500	270	320	GOOD	2	2	2	2	2	2	2	2	2	67	68	67	66	67	66	68	67	68	120	117
40	HARINDRA	5559	31Y	MALE	55	I	L 1st Metacarpal #	CRIF with K Wire	11	13	380	260	280	EXCELLENT	2	2	2	2	2	2	2	2	2	70	68	66	64	62	60	64	66	68	110	110
41	ALTAF	18601	25Y	MALE	57	II	L Distal end Radius #	ORIF with Plating	9	14	500	260	300	GOOD	2	2	2	2	2	2	2	2	2	80	82	78	74	78	72	74	70	68	130	106
42	GUDDAPPA	23170	55Y	MALE	58	I	Both Bone #	ORIF with Plating	15	20	350	210	240	GOOD	2	2	2	2	2	2	2	2	2	86	84	82	88	86	84	88	84	86	112	114
43	NAGEVENI	12329	22Y	FEMALE	58	I	L Both Bone Implant Insitu	Implant Removal	12	16	510	300	360	EXCELLENT	2	2	3	3	2	2	3	2	2	70	68	64	66	64	66	66	62	66	120	130
44	BASAVARAJ	19081	50Y	MALE	56	I	Old United Olecranon Implant Insitu	Implant Removal	11	13	450	330	360	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	76	72	74	76	72	70	70	136	130
45	MANAPPA	24332	40Y	MALE	60	I	R DER Scaphoid #	ORIF with Plating	9	14	500	260	300	GOOD	2	2	2	2	2	2	2	2	2	80	82	78	74	78	72	74	70	68	126	106
46	SURESH	18409	32Y	MALE	58	I	L Olecranon #	Radial Neck Excision with Plating	12	14	400	300	350	EXCELLENT	2	2	2	2	2	2	2	2	2	76	74	78	74	72	76	72	74	74	130	130
47	GIRISH	18756	55Y	MALE	56	II	Humerus #	ORIF with Plating	13	15	420	300	360	EXCELLENT	2	2	2	2	2	2	2	2	2	74	72	71	70	73	72	73	74	73	130	132
48	REKHA	28037	45Y	FEMALE	58	II	L DER #	CRIF with K Wire	12	16	480	250	360	EXCELLENT	2	2	2	2	2	2	2	2	2	64	66	64	66	68	64	64	66	68	114	116
49	KAMALABAI	14980	56Y	FEMALE	56	I	L DER #	CRIF with k Wire	9	14	500	300	400	EXCELLENT	2	2	2	2	2	2	2	2	2	78	76	74	74	74	76	76	74	76	120	118
50	SHANTA	23245	60Y	FEMALE	60	I	L DER #	CRIF with K Wire	15	20	440	300	320	GOOD	2	2	2	2	2	2	2	2	2	72	71	68	66	68	64	68	64	60	150	150

SYSTOLIC BLOODPRESSURE (mmHg)							DIASTOLIC BLOODPRESSURE (mmHg)									RESPIRATORY RATE									SATUARATION %									PAIN INTENSITY								
15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs	0 Min	5 Min	15 Min	30 Min	60 Min	2 Hr	6 Hrs	12 Hrs	24 Hrs
118	118	122	118	118	120	120	90	80	80	80	80	82	80	82	80	14	14	16	14	14	14	14	14	14	100	100	100	100	100	100	100	100	100	10	9	8	5	4	2	1	1	1
124	124	126	126	126	126	126	90	84	80	80	80	80	80	80	80	14	14	16	14	14	14	14	14	14	100	100	100	100	100	100	100	100	100	10	9	8	5	2	1	1	1	1
124	120	120	124	124	126	126	80	80	70	70	80	80	80	80	80	14	13	12	13	12	13	13	13	14	100	99	98	98	97	98	98	98	98	10	9	8	5	1	1	1	4	4
124	120	120	124	124	124	124	90	78	78	78	78	80	80	80	80	14	14	16	14	16	16	14	14	14	100	100	100	100	100	100	100	100	100	10	8	9	5	4	2	2	2	2
118	116	116	118	118	118	116	80	76	78	74	74	78	80	80	78	14	14	14	16	14	14	14	14	14	100	100	100	99	99	100	100	100	100	10	9	5	2	1	1	1	1	1
110	110	112	112	116	112	112	74	68	70	72	70	70	74	70	70	14	16	14	14	14	16	14	14	14	100	100	100	100	100	100	100	100	100	10	8	5	4	2	1	1	1	1
118	120	122	126	120	122	122	80	70	72	72	70	70	70	70	72	14	15	14	15	14	15	15	14	14	99	99	98	99	98	99	98	99	98	10	9	8	7	5	1	1	4	6
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