A Comparative Study of Peripheral Nerve Stimulated Guided Supraclavicular Brachial Plexus Block Using Bupivacaine -lignocaine With Adrenaline and Dexmedetomidine Added To Bupivacaine - Lignocaine With Adrenaline.

*Dr. Avinash B. Pawar, ** Dr. H. S. Rawat

* Resident, ** Professor and Head, Department of Anaesthesia, Dr. Vithalrao Vikhe Patil Foundation's Medical College Ahmednagar

Corresponding Author : Dr. H. S. Rawat

Mail id : avinash.pawar90@gmail.com

Mobile No.: 8793300036

Address : Department of Anaesthesia, Dr. Vithalrao Vikhe Patil Foundation's Medical College, Ahmednagar

Abstract :

Background : Alpha-2 agonists are mixed with local anaesthetic agents to extend the duration of spinal, extradural and peripheral nerve blocks. We add Dexmedetomidine as an adjuvants to local anaesthetic in supraclavicular brachial plexus block with respect to onset and duration of sensory and motor block and duration of analgesia. **Methods :** A randomized prospective observational clinical study was carried out on 60 American Society of Anaesthesiologist (ASA) Grade I and II patients of either sex, aged 18–50 years, undergoing various bony orthopaedic surgeries on the upper limb under supraclavicular brachial plexus block. The study was conducted in two groups of 30 patients each , group 1: Patients received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) + 2 ml of 7.5% (w/v) Sodium bicarbonate + normal saline (0.5ml) ,group 2: Patients received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) +2 ml of 7.5% (w/v) Sodium bicarbonate + Dexmedetomidine (0.5ml). **Results :** The onset of sensory block and duration of sensory block was 5.83 ± 1.5 and 690 ± 87 min, respectively, in group 2 and onset of motor block and duration of motor block was 8.43 ± 1.5 and 353.17 ± 42.41, respectively, in group 2 which was statistically highly significant as compared to group 1 which was 8.83 ± 1.01 and 386 ± 42.33 for onset of motor block and duration of sensory block respectively and 11.53 ± 1.75 and 270 ± 31.6 for the onset of motor block and duration of motor block in group 2, respectively. The need for the rescue analgesia was 456.21 ± 97.99 in group 2 and 289.67 ± 62.50 in group 1, which was also highly significant. **Conclusion :** Dexmedetomedine, when added to local anaesthetic in supraclavicular brachial plexus block prolongs the duration of sensory and motor block and enhances the quality of block. The time for rescue analgesia was prolonged in patients receiving Dexmedetomidine.

Introduction : The word pain is derived from the word "POENA" meaning punishment. Pain is a biological phenomenon that is easy to comprehend but difficult to define. It is a complex interaction that involves sensory, emotional and behavioural factors and therefore its definition include all of these aspect.Recently, anaesthesiologist has become increasingly involved in the provision of postoperative analgesia and the development of pain management services.[1]

The aim of postoperative pain relief is to provide subjective comfort in addition to inhibition trauma induced nociceptive impulses to blunt autonomic and somatic reflex response to pain and subsequently enhance restoration of function by allowing patient to breath , cough and to be ambulatory more easily , preventing postoperative pulmonary complications and inhibing sympathetic nervous system stimulation , resulting in the earlier discharge from the hospital and minimizing the total hospital expenditure.[2]

Peripheral nerve block is now well accepted component of comprehensive anaesthetic care. The supraclavicular brachial plexus block is one among the most popular regional nerve blocks performed. It is also known as" SPINAL OF THE ARM." 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 analgesia requirement, early ambulation,

overcoming the disadvantages of general anaesthesia without any systemic side-effects. [3]

The limitation of local anaesthetic is slower onset of action, shorter duration of action and reduced motor and sensory blockade.There has always been a search for adjuvants to the regional nerve block to fill the lacunae created by local anaesthetic. The search for the ideal additive continues, and led us to try the novel $\alpha 2$ adrenergic agent, Dexmedetomidine.

Alpha-2 adrenergic receptor agonists have been the focus of interest for their sedative, analgesic, perioperative sympatholytic and cardiovascular stabilizing effects with reduced anaesthetic requirements. Furthermore, various methods of administration, such as epidural, intrathecal and peripheral injections, have been tried either alone or in combination with another drug to prolong and intensify the anaesthesia.[4]

Aim and Objectives : To compare Dexmedetomidine as adjuvant to mixture of 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) + 2 ml of 7.5% (w/v) Sodium bicarbonate in supraclavicular brachial plexus block in respect to-

1. Onset time of sensory block and total duration of sensory block.
2. Onset time of motor block and total duration of motor block.
3. Rescue analgesia

Materials and methods :

Source of Data : Patients scheduled for upper limb surgery in, DVVPF's Medical College and Hospital, Ahmednagar, was selected for the study over a period of one year from December 2017 to April 2018.

Type of Study : Prospective randomised observational study.

Selection of Patient : Convenience type of non-probability sampling was used for selection of study subjects. A total of 60 patients posted for elective upper limb surgeries in Department of Anaesthesiology and Critical Care, DVVPF'S Medical College and Hospital, Ahmednagar, were included in the study after obtaining permission from the Hospital Ethics Committee.

Inclusion Criteria :

1. Age between18-50 years of either sex.
2. ASA physical status I and II.
3. Patients listed for upper limb surgeries involving arm and forearm.

Exclusion Criteria

1. Neurological lesions in the upper limb to be operated upon.
2. Diabetic neuropathy.
3. Psychiatric patients.
4. History of allergy to local anaesthetics.
5. Infection/swelling at proposed site of injection.
6. Bleeding disorders or patients on anticoagulant.
7. Any skeletal abnormality hindering surface markings.
8. Failure of block or conversion to GA.

Methodology : After written informed consent, a randomized prospective observational clinical study was carried out on 60 American Society of Anaesthesiologist (ASA) Grade I and II patients of either sex, aged 18–50 years, undergoing various bony orthopaedic surgeries on the upper limb under supraclavicular brachial plexus block. The study was conducted in two groups of 30 patients each. The patients were randomly assigned in 2 groups by the sealed envelope method:

Group 1: Patients received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) + 2 ml of 7.5% (w/v) Sodium bicarbonate + normal saline (0.5ml).

Group 2: Patients received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) +2 ml of 7.5% (w/v) Sodium bicarbonate + Dexmedetomidine 50 mcg (0.5ml).

Pre-anaesthetic evaluation was performed on the day before surgery. The procedure of block along with possible complications was explained to the patients, and written informed consent was obtained. All patients were given oral Alprazolam 0.5 mg and inj. Ranitidine 150 mg on the night before the surgery, and were fasting overnight.

On arrival in the operation room, baseline heart rate, blood pressure and oxygen saturation were recorded. An intravenous line was secured with 18G intrcath in the unaffected limb and Ringer's lactate was started.

All the patients received brachial plexus block through the supraclavicular approach by an experienced anaesthesiologist. Neural localization was achieved by using a nerve locator (Fisher and Paykel, New Zealand) connected to a 22 G, 50-mm-long stimulating needle (Stimuplex, Braun, Germany). The location end point was a distal motor response with an output lower than 0.5 mA in the median nerve region.

Following negative aspiration as mentioned above was 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 musculocutaneous nerve till complete sensory blockade. Sensory onset was considered when there was a 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 by the same observer at each minute till complete motor blockade after 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 a modified Bromage scale for upper extremities on a 3-point scale.[1]

Grade 0: Normal motor function with full flexion and extension of elbow, wrist and fingers

Grade 1: Decreased motor strength with ability to move the fingers only

Grade 2: Complete motor block with inability to move the fingers

The block was considered incomplete when any of the segments supplied by median, radial, ulnar and musculocutaneous nerve did not have analgesia even after 30 min of drug injection. When more than one nerve remained unaffected, it was considered a failed block. In this case, general anaesthesia was given intra-operatively. Patients were monitored for haemodynamic variables such as heart rate, blood pressure and oxygen saturation every 30 min after the block intra-operatively and every 60 min post-operatively. Sedation of patient was assessed by the Ramsay Sedation Score.(Ramsay,1974). Sedation was assessed using the Ramsay sedation score (RSS);

Level 1 Anxious and agitated, or restless or both

Level 2 Co-operative, oriented and tranquil

Level 3 Respond to commands only

Level 4 Brisk response to painful stimulus

Level 5 Sluggish response to painful stimulus

Level 6 No Response to painful stimulus

The duration of sensory block was 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 was defined as the time interval between the end of local anaesthetic administration and the recovery of complete motor function of the hand and forearm.

Assessment of blood loss was done and fluid was administered as per the loss. Duration of surgery was noted.

Pain was assessed by Visual Analogue Scale (VAS). VAS will be recorded and assessed at an every 5 min for the first 30 min; then half hourly for the first 8 hr then every 1 hourly till patient complained of pain equivalent to a VAS score of 4.

All patients were observed for any side-effects like nausea, vomiting, dryness of mouth and complications like pneumothorax, haematoma, local anaesthetic toxicity and post-block neuropathy in the intra- and post-operative periods.

Result :

Statistical analysis : The data was analysed by SPSS version (Statistical Package for Social Sciences) software. Unpaired t-test was applied for demographic data, haemodynamic parameters, onset and duration of sensory and motor blockade and duration of analgesia. Fisher exact test was applied for assessment of quality of block. P-value was considered significant if <0.05 and highly significant if <0.001.

TABLE 1:

A] Demographic data:

	Group A	Group B	Statistical significance
Age (yr)	37.76 ± 8.63	34.63 ± 7.95	NS
Weight (kgs)	58.63 ± 5.12	58 ± 6.67	NS
Sex (M/F)	14/16	12/18	NS
ASA (I/II)	13/17	12/18	NS
Duration of surgery (min)	130.9 ± 22.95	138.73 ± 22.77	NS

Data were expressed as means± standard deviation. Significant figures of P value

Significant ($P < 0.05$) ,Highly significant ($P<0.01$),Not significant($p>0.05$)

There was no statistically significant difference among the two groups as regards Age, sex ASA grade, weight, height, and duration of surgery.

Table 2 : Characteristics of Block

Parameters	Mean±SD		
	Group 1	Group 2	
Onset of Sensory Block(min)	8.83 ± 1.01	5.83 ± 1.5	HS
Onset of Motor Block(min)	11.53 ± 1.75	8.43 ± 1.5	HS
Duration of Sensory block(min)	386 ± 42.33	690 ± 87	HS
Duration of Motor block(min)	270 ± 31.6	353.17±42.41	HS
Rescue analgesia (min)	289.67±62.50	456.21±97.99	HS

Data were expressed as means± standard deviation. Significant figures of P value

Significant ($P < 0.05$), Highly significant ($P<0.01$),Not significant($p>0.05$)

Highly significant findings observed within the groups. Addition of Dexmedetomedine prolongs the duration of block and the results which were similar with previous studies but in our study we observed highly significant findings in both the groups. During our study we also observed side effects and found that there is no significant complication encountered during the procedure in both the groups.

Discussion : Brachial plexus block is a versatile and reliable regional technique with multiple applications. It is a block of roots, divisions and cords first performed by Halsted in 1884. There are many approaches to block brachial plexus. Supraclavicular approach was first described by Kulenkampff in 1911.[1]

Brachial plexus block results in anaesthesia that is limited to a restricted portion of body, not disturbing the metabolism of rest of the body, leading to early ambulation and early discharge of the patient. The only limitation in brachial plexus is the duration of the block. To overcome this limitation many strategies have been and are being developed, like use of adjuvants to local anaesthetic mixture. Supraclavicular brachial plexus block is a commonly performed regional anaesthetic technique for forearm and hand surgeries, and provides good surgical anaesthesia. Supraclavicular approach is easy to perform even if the arm is immobilized.

Presynaptic activation of alpha-2A adrenoceptor in the locus coeruleus inhibits the release of norepinephrine and results in the sedative and hypnotic effects. In addition, the locus coeruleus is the site of origin for the descending medullo spinal noradrenergic pathway known to be an important modulator of nociceptive neurotransmission. Stimulation of alpha-2 adrenoceptors in this area terminates the propagation of pain signals leading to analgesia. Postsynaptic activation of alpha-2 receptors in the CNS results in decrease in sympathetic activity leading to hypotension and bradycardia.[5] Alpha-2 adrenoceptors present on primary afferent terminal (peripheral and spinal endings) in the superficial laminae of the spinal cord and within several brainstem nuclei have been implicated in the analgesia, supports the possibility of analgesic action of alpha agonist at peripheral, spinal and brainstem site. The direct action of clonidine on the nerve can be explained 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.[6]

Dexmedetomidine, an imidazole compound is the pharmacologically active s-enantiomer of medetomidine. The specificity of Dexmedetomidine for the alpha-2 receptor is 8 times that of clonidine, with an α-2 / α-1 binding affinity ratio of 1620:1 and hence, considered as the full agonist at alpha-2 receptors. Alpha-2 agonists provide sedation, analgesia, muscle relaxation & anxiolysis.[7]

Various studies have shown that Dexmedetomidine prolongs the duration of sensory and motor block and provide a very good analgesia when used as an adjuvant to local anaesthetics for nerve blocks. [8,9,10] Our study was conducted to evaluate the efficacy of Dexmedetomidine as adjuvant to when added to 0.5% Bupivacaine and 2% Lignocaine with Adrenaline 2 ml of 7.5% (w/v) Sodium bicarbonate. In our study we observed that the onset of sensory block in Group 1 (8.83 ± 1.01) and group 2 is (5.83 ± 1.5) which is less as compared to other studies but there is highly significant difference between the studied group. Also, onset of motor block in Group 1(11.53 ± 1.75) and group 2 is (8.43 ± 1.5) which is less as compared to other studies but there is highly significant difference between the studied group. In this study we compared duration of sensory block, duration of motor block and rescue analgesia time and found that there was difference observed in two groups which is highly significant. In this study we 2 ml of 7.5% (w/v) Sodium bicarbonate in each group which also helps in early onset of block which was not done in previous studies.[11]

Conclusion : To conclude, we would like to state that patients who received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) +2 ml of 7.5% (w/v) Sodium bicarbonate + Dexmedetomidine (0.5ml) prolongs the duration of sensory and motor block and enhances the quality of block as compared with the patients who received 0.5% Bupivacaine (15ml) + 2% Lignocaine with Adrenaline (15ml) + 2 ml of 7.5% (w/v) Sodium bicarbonate + normal saline (0.5ml).

References :

1. Swami SS, Keniya VM, Ladi SD, Rao R. Comparison of dexmedetomidine and clonidine (α2 agonist drugs) as an adjuvant to local anaesthesia in supraclavicular brachial plexus block: A randomised double-blind prospective study. Indian Journal of Anaesthesia. 2012;56(3):243-249. doi:10.4103/0019-5049.98767.
2. Jones ME, Maze M. Can we characterize the central nervous system actions of alpha-2 adrenergic agonists? Br J Anaesth 2001;86(1):1-3.
3. Derbyshire DR, Chmielewski A, Fell D, et al. Plasma catecholamine response to tracheal intubation. Br J Anaesth 1983;55(9):855-859.
4. Brummett CM, Norat MA, Palmisano JM, et al. Perineural administration of dexmedetomidine in combination with bupivacaine enhances sensory and motor blockade in sciatic nerve block without inducing neurotoxicity in rat. Anesthesiology 2008;109(3):502-511.
5. Eisenach JC, De Kock M, Klimscha W. Alpha(2)-adrenergic agonists for regional anesthesia. A clinical review of clonidine (1984-1995). Anesthesiology 1996;85(3):655-674.
6. Halaszynski, T.M. 2012. Dexmedetomidine: A look at a promising new avenue of use. Saudi J Anaesth, 6:104-6.
7. Esmaoglu, A, Yegenoglu, F, Akin, A, Turk, C.Y. 2010. Dexmedetomidine added to Levobupivacaineprolongs axillary brachial plexus block. AnaesthAnalg., 111:1548-51.
8. Brummett, C.M., Norat, M.A., Palmisano, J.M., Lydic, R. 2008. Perineural administration of dexmedetomidinein combination with bupivacaine enhances sensoryand motor blockade in sciatic nerve block withoutinducing neurotoxicity in rat. Anesthesiology, 109:502-11.
9. Dr. Ruby Mehta, Dr. D. D. Verma, Dr. Veena Gupta, Dr. A. K. Gurwara. TO STUDY THE EFFECT OF ALKALINIZATION OF LIGNOCAINE HYDROCHLORIDE ON BRACHIAL PLEXUS BLOCK, Indian J. Anaesth. 2003; 47 (4) : 283-286: AL
10. Bisui B, Samanta S, Ghoshmaulik S, Banerjee A, Ghosh TR, Sarkar S. Effect of Locally Administered Dexmedetomidine as Adjuvant to Levobupivacaine in Supraclavicular Brachial Plexus Block: Double-blind Controlled Study.Anesthesia, Essays and Researches. 2017;11(4):981-986. doi:10.4103/aer.AER_55_17.
11. Thakur A, Singh J, Kumar S, Rana S, Sood P, Verma V. Efficacy of Dexmedetomidine in two Different Doses as an Adjuvant to Lignocaine in Patients Scheduled for Surgeries under Axillary Block. Journal of Clinical and Diagnostic Research 2006;40(2)116-118.