IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 16, Issue 9 Ver. V (Sep. 2017), PP 71-75
www.iosrjournals.org

Efficacy of Dexmedetomidine as an adjutant to Levobupivacaine for Supraclavicular Brachial Plexus Block

Dr. Mallika Mohindra[1,*] Dr. Anupama Gupta[2]
[1] *Senior Resident, Department of Anaesthesiology, SMS Medical College, Jaipur*
[2]*Assistant Professor, Department of Anaesthesiology, SMS Medical College, Jaipur*
Corresponding Author: [*]Dr. Anupama Gupta

Abstract
Background and Aims: *This prospective, randomised double blind study was conducted to establish the effects of addition of dexmedetomidine to levobupivacaine for supraclavicular brachial plexus block.*
Methods and Material: *Sixty patients of ASA I&II, scheduled for upper arm surgery under supraclavicular block were enrolled to receive either 30 ml levobupivacaine 0.5% and 1 ml sodium chloride (Group I) OR 30 ml levobupivacaine 0.5% and 1 ml dexmedetomidine 100 microgram (Group II). Block was performed using nerve locator. The onset of sensory and motor block, duration of block and analgesia, VAS score and time of first rescue analgesia, haemodynamic variables and any side effects were monitored.*
Results: *Demographic data was comparable between the groups. Sensory block onset time was 10.54±2.333 min in group I and 3.24±0.951 min in group II; motor block onset time was 12.21±2.529 min in group I and 3.83±1.197 min in group II; sensory block duration was 7.79±2.007 hours and 16.31±2.606 hours in group I and II respectively; motor block duration was 9.18±1.701 hour in group I and 17.52±2.098 hour in group II; time of first analgesic requirement was longer in group II than in group I ($p<0.05$). Intraoperative HR and BP were significantly lower in group II as compared to group I ($p<0.05$). Bradycardia was observed in one patient in group II with no other adverse effects in either of the groups.*
Conclusions: *Hence, addition of dexmedetomidine to levobupivacaine for supraclavicular block significantly prolonged the duration of postoperative analgesia with added advantage of conscious sedation, haemodynamic stability with minimal side effects.*

Keywords*: Adjuvant, dexmedetomidine, levobupivacaine, supraclavicular brachial plexus block*

Date of Submission: 07 -09-2017 Date of acceptance: 20-09-2017

I. Introduction

Supraclavicular brachial plexus blocks are being used widely for various upper limb surgeries to provide optimum surgical anaesthesia along with postoperative pain relief. Since the introduction of long acting local anaesthetic agents with better safety profile and technical advancements, use of peripheral nerve blocks has increased tremendously. Levobupivacaine, the pure L-isomer of bupivacaine having lesser cardiac and CNS toxicity, has emerged as a safer alternative for regional anaesthesia than racemic bupivacaine. Many drugs have been used as an adjuvant to local anaesthetic agents to provide superior quality of block along with prolonged duration of postoperative analgesia.Alpha-2 adrenergic receptor agonists are being used widely as adjuvants nowadays. They prolong the duration of analgesia by blocking the so-called hyperpolarisation-activated cation current (Ih current). Dexmedetomidine, being highly selective α2-AR agonists has more pronounced effect on inhibition of nerve fibre action potentials as compared to clonidine.[1] Dexmedetomidine provides effective analgesia and sedation without affecting haemodynamic and respiratory stability. In this study, we investigated whether adding dexmedetomidine to levobupivacaine for supraclavicular brachial plexus block would affect the characteristics of sensory and motor blocks and duration of analgesia.

II. Material And Methods

After the approval of the Hospital Ethical Committee and written informed consent from the patients, sixty patients of 18-60 years, either sex, ASA physical status I and II posted for upper limb surgery under supraclavicular brachial plexus block were enrolled in a prospective, randomized, double-blind, placebo-controlled trial. Patients with previous nerve deformity or brachial plexus injury, severe liver or kidney disease, patients having opposite side pneumothorax or collapsed lung, with known allergy to LAs, local infection and coagulopathies were excluded from the study. Patients were randomly allocated into two group by drawing sequential numbered, opaque sealed envelopes, containing a code based on computer generated random number list.

Group I (n = 30) - received 30 ml of 0.5% levobupivacaine and 1 ml of isotonic sodium chloride solution.
Group II (n = 30) - received 30 ml of 0.5% levobupivacaine and 1 ml (100 mcg) of dexmedetomidine.

To maintain blinding, drug preparation and procedure was performed by two separate anaesthetists blinded to the drug study, while the observation was done by attending anaesthetist. After confirming fasting status, standard anaesthetic monitoring was established using electrocardiogram, pulse oximeter and noninvasive blood pressure monitor. A 20G i.v. cannula was inserted in non-operative arm and lactated Ringer's solution started at 15ml/kg/h. For supraclavicular approach, the patient was placed in supine position with adduction of the arm to be anaesthetised and head extended and turned away from the side to be blocked. The medial and lateral borders of the clavicle were identified as the first rib generally lies beneath the midpoint of clavicle. The landmark was confirmed by sliding down the fingers in the interscalene groove till the arterial pulsation of the subclavian artery felt. A skin wheal was then raised 0.5 to 1 cm posterior to the midpoint of clavicle and a 22-gauge, short bevelled nerve stimulating needle inserted in a caudal, slightly medial and posterior direction. The needle was connected to the negative lead of the nerve locator, preset in the motor testing mode with a current setting of 2-3 mA and the patient's arm observed. When the patient got a distal contraction of the upper limb, the current was reduced to 0.6 mA. After observing the contractions at this reading also, the drug solution was injected.

Sensory block was assessed by loss of sensation to pin prick in the midline using a 22 gauge blunt hypodermic needle every minute using Hollmen scale (1- normal sensation of pin prick, 2- pin prick felt as sharp pointed but weaker compared with the same area in the other limb, 3- pin prick recognised as touch with blunt object and 4- no perception of pin prick). A sensory block of scale 3 was considered as endpoint for surgery. Onset of sensory block was taken as time from injection of drug to Hollmen sensory scale of 2. Duration of sensory block was taken as time elapsed between performing the block to regression of sensory block to scale of ≤ 2.Motor block was assessed using Hollmen scale (1-normal muscle action, 2-slightly weak muscle action, 3-very weak muscular action and 4-complete loss of muscle action). The test was performed every minute till scale 2. A motor block of scale 3 was considered as endpoint for surgery. Onset of motor block was taken as time from injection of drug to Hollmen motor scale of 2.Duration of motor block was taken as time elapsed between performing block to regression of motor scale to lower degree.

Postoperative pain was assessed by Visual analogue scale (VAS) score (0-no pain, 1-3-mild pain, 4-7-moderate pain, 8-10-severe pain) at 2hrs, 4hrs, 6hrs, 8hrs, 10hrs, 12hrs, 18hrs and 24hrs after surgery. Whenever VAS score reached ≥ 4, rescue analgesia was given in the form of intravenous injection diclofenac 75mg. Time of first request of analgesia was noted. Monitoring of haemodynamic variables at 0, 3,6,9,15,20,25,30, 45, 60,75, 90, and 120 min. Sedation score was assessed according to the Ramsay Sedation Scale (RSS) from 1-6 (1 = anxious, agitated, restless; 2 =cooperative, oriented, tranquil; 3 = responds to commands only; 4 = brisk response to light glabellar tap or loud noise;5 = sluggish response to light glabellar tap or loud noise; 6 =no response).

Adverse effects such as hypotension (i.e. 20% decrease relative to baseline), bradycardia (i.e. 20% decrease relative to baseline), nausea, vomiting, and hypoxemia (SpO2 <90%) were also documented. The decoding of the groups was done at the end of the study followed by statistical analysis of the results. **Statistical analysis** was done using SPSS version 17.0. Chi Square test was for non-parametric data and Student Unpaired t-test for parametric data for inter group comparison. P value of less than 0.05 was considered significant and less than 0.001 as highly significant. The power of our study was >94% taking into considerations the parameters such as onset and duration of sensory and motor block and duration of analgesia. Because of 2 failure cases in group I and 1 failure case in group II statistical analysis was applied on 28 patients in group I and 29 patients in group II.

III. Results

Both the groups were comparable with respect to age, sex ratio, ASA physical status [Table I].The sensory and motor block onset was significantly faster in group II than in group I. The mean sensory block onset time was 3.24 ± 0.9 min in group II as compared to 10.54 ± 2.3 min in group I(P =0.000). The mean motor block onset time was 3.83 ± 1.1 min in group II as compared to 12.2 ± 2.5 min in group I (P =0.000) [Table 2] The duration of sensory block was prolonged in group II (16.3 ± 2.6 hrs) as compared to group I (7.79 ± 2.0hrs) (P=0.000) [Table 2]. The duration of motor block was also prolonged in group II (17.5 ± 2.0 hrs) as compared to group I (9.18 ± 1.7 hrs) (P=0.000) [Table 2]. The mean duration of analgesia was significantly longer in group II (1273.7 ± 83.1 min) in comparison to group I (678.6 ± 20.4 min) (P =0.000) [Table 2]. HR, SBP and DBP in group II were significantly lower than in group I (P < 0.001) intra-operatively [Figures 1, 2, and 3]. Bradycardia was observed in one patient in the group II which responded to injection atropine. The mean Ramsay sedation score was 3.9 ±0.6 in group II and 2.00 in group I. Other side effects like hypotension, nausea, vomiting, hypoxemia, pruritis or urinary retention were not observed in either group [Figure 4].

IV. Tables & Figures

Table 1: Demographic profile, type and duration of surgery

Parameters	Group I	Group II	P value
ASA status(I/II)	24/4	23/6	
Sex ratio(m/f)	21/7	22/7	
Age(yrs)	34.93 ± 14.66	35.62 ± 15.03	0.861

Table 2: Characteristics of sensory and motor block, Sedation score

Parameters	Group I (Mean ± S.D)	Group II (Mean ± S.D)	P value
Onset of sensory block (min)	10.54 ±2.33	3.24±0.95	0.000
Onset of motor block (min)	12.21 ± 2.52	3.83 ± 1.19	0.000
Duration of sensory block (hrs)	7.79 ± 2.00	16.31 ± 2.60	0.000
Duration of motor block (hrs)	9.18 ± 1.70	17.52± 2.09	0.000
Rescue analgesia (min)	678.68 ± 20.49	1273.79 ± 83.13	0.000
Sedation score	2.00 ± 0.00	3.90 ±.61	0.000

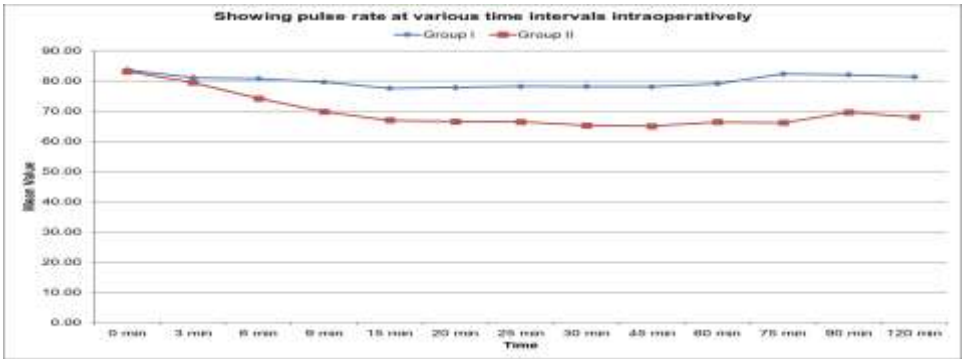

Figure 1: Intraoperative trends of heart rate

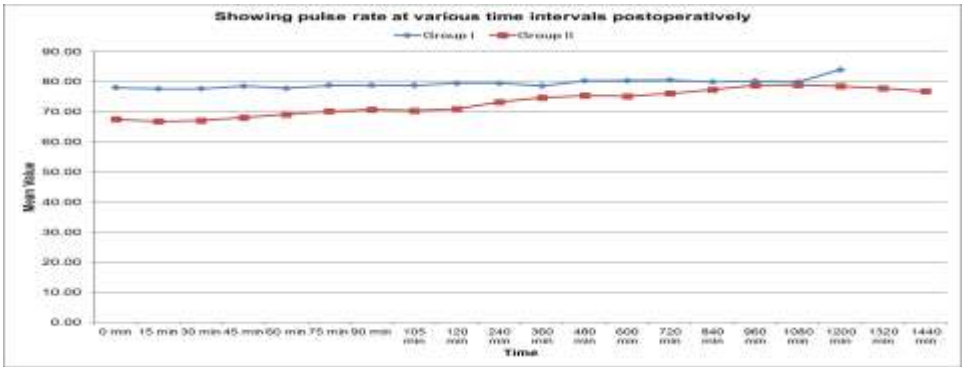

Figure 2: Post-operative trends of heart rate

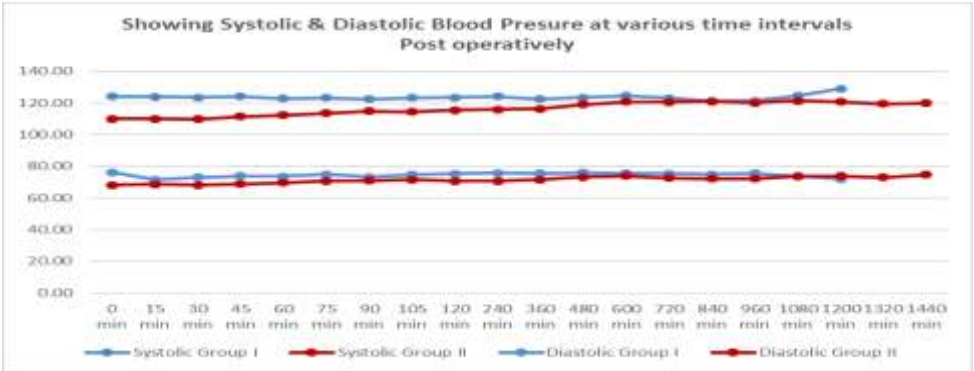

Figure 3: Intraoperative and postoperative trends of blood pressure

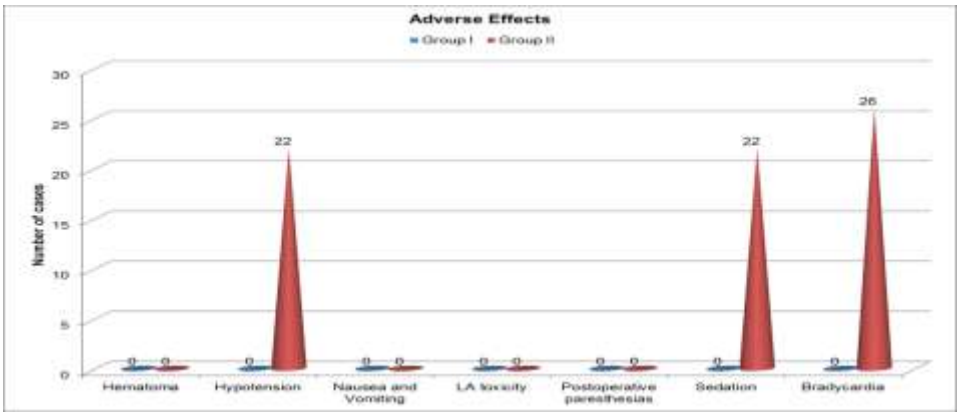

Figure 4: Incidence of adverse effects

IV. Discussion

Primary outcome of the study showed that addition of 100 mcg dexmedetomidine to 0.5% levobupivacaine resulted in longer duration of analgesia. It also hastened onset of sensory and motor block and prolonged duration of sensory and motor block. The mechanism of prolongation of motor and sensory block of local anaesthetic agents by α2-AR agonists is not fully understood yet. It is not due to altered systemic absorption, as the plasma level of bupivacaine was not altered after the addition of intrathecal clonidine to bupivacaine spinal injection.[2] In clinical practice it has been observed that the use of α2 agonists reduce the anaesthetic and the analgesic requirement to a large extent because of their analgesic action by depressing the release of C-fibre transmitters and by hyperpolarization of postsynaptic dorsal horn neurons.

Studies by Brummett et al., showed that dexmedetomidine enhances duration of bupivacaine anesthesia and analgesia of sciatic nerve block in rats without any evidence of histopathological damage to the nerve.[8,9] In another study, dexmedetomidine added to ropivacaine increased the duration of sciatic nerve blockade in rats, most likely due to the blockade of hyperpolarization-activated cation current (i.e., a direct effect on the peripheral nerve activity).[10] Kousugi et al in their study found high concentrations of dexmedetomidine inhibit compound action potentials in frog sciatic nerves without α2 adrenoreceptors activation in a concentration dependent manner and reversibly.[11] Yoshitomi et al., demonstrated that dexmedetomidine as well as clonidine enhanced the local anaesthetic action of lignocaine via peripheral α-2A adrenoceptors.[12]In our study, the onset time of both sensory and motor components of brachial plexus blocks were shortened, duration of sensorimotor block and analgesia prolonged in the study group**.** Similar findings were observed by Swami et al, Ammar et al. in their study on addition of dexmedetomidine to bupivacaine 0.25% in brachial plexus block.[13]

Esmaoglu et al.[14] concluded that addition of dexmedetomidine to levobupivacaine for axillary brachial plexus blockade shortened the block onset time, prolonged the duration of motor and sensory effects, and extended postoperative analgesia. Kaygusuz et al. [15] evaluated the addition of dexmedetomidine 1μg/kg to 0.5% levobupivacaine in axillary brachial plexus block and observed similar results. In a recent study by Marhofer et al.[16] The effect of dexmedetomidine on 0.75% ropivacaine for ulnar nerve block was studied, they also concluded that onset of motor block was faster, and the duration was significantly prolonged by the perineural administration of dexmedetomidine. In a study by Agarwal et al Ramsey sedation score was either 2 or 3 in dexmedetomidine group and 1 in control group. In our study too we had a similar observation. Sedation is due to action on locus coeruleus, which inhibit the release of norepinephrine.[3, 4] Sedation after epidural α2 agonists is due to its systemic absorption and vascular redistribution to higher centers.[5, 6, 7] It is an add-on advantage for peripheral nerve blocks to bring down the stress associated with the surgery.

Our study findings are comparable to previous studies conducted by Esmaoglu et al in 2010[14] and Agarwal et al[17].Esmaoglu et al observed bradycardia in seven out of 30 patients in study group and Agarwal et al in 1 out of 25 patients while we observed it in only one out of 30 patients .There was significant fall in HR and BP in dexmedetomidine group but fall was not more than 20% from baseline in any patient. Our results are in accordance with that of Swami SS et al.[18]

V. Conclusion

To conclude, in this study we found that dexmedetomidine when added to levobupivacaine for supraclavicular brachial plexus block shortens the onset times for sensory and motor blocks and prolongs its duration. The significantly prolonged duration of analgesia obviates the need for any additional analgesics. The added advantage of conscious sedation, hemodynamic stability, and minimal side effects makes it a potential adjuvant for nerve blocks.

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*Dr.Anupama Gupta. "Efficacy of Dexmedetomidine as an adjutant to Levobupivacaine for Supraclavicular Brachial Plexus Block." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.9 (2017): 71-75