

# Effect of Dexmedetomidine as Adjuvant to Ropivacaine in Ultrasound Guided Supraclavicular Brachial Plexus Block for Upper Limb Surgeries

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## Abstract

**Background and Aims:** Different additives have been used to prolong Brachial plexus block. We evaluated the effect of adding Dexmedetomidine to Ropivacaine for Ultrasound guided Supraclavicular Brachial plexus blockade. The primary endpoints were the onset and duration of sensory and motor block and duration of analgesia. **Materials and Methods:** A total of 60 patients (18-60 years) satisfying inclusion and exclusion criteria posted for elective upper limb surgeries under supraclavicular Brachial plexus block were divided into two equal groups (Group R and RD) in a randomized, double-blind fashion. In group RD (n= 30) 30 ml 0.5% Ropivacaine plus 2 ml (50 micrograms diluted to 2ml ) of Dexmedetomidine and group R (n = 30) 30 ml 0.5% Ropivacaine plus 2 ml normal saline were administered in Ultrasound guided supraclavicular block. Sensory and motor block onset times and block durations, duration of analgesia, and side effects if any were recorded for each patient. **Results:** Demographic parameters were comparable in both groups. Onset time of sensory and motor block were shorter in Group RD (6.1±3.4 min and 8.9±3.08 min respectively) than in Group R (8.3±4.4 min and 13.0 ± 5.6 min respectively) (p = 0.001). Duration of sensory and motor blockade were longer in Group RD (630.50±208.2 min and 545.9±224.0 min respectively) than in Group R (400.8± 86.6 min and 346.9±76.9 min) (p = 0.001). Duration of analgesia was longer in Group RD (805.7±205.9 min) than in Group R (411.0±91.2 min) (p = 0.001). Intra-operative hemodynamics were significantly lower in group RD (P < 0.05) without any adverse side-effects. **Conclusions:** Dexmedetomidine when added to Ropivacaine for Brachial plexus block shortens the onset time and prolongs the duration of the block and the duration of post-operative analgesia.

**Keywords:** Dexmedetomidine; Ropivacaine; Supraclavicular Brachial Plexus Block; Ultrasound.

## Introduction

Brachial plexus block has evolved as an important tool in the anaesthesiologist's armamentarium as a safe alternative to general anaesthesia for upper limb surgery and for relief of perioperative pain. Its increased popularity is because of advancements in regional anaesthesia techniques in terms of local anaesthetic drugs, newer adjuvant drugs and use of Ultrasound for safe and successful conduct of block. It helps in reduced hospital stay, less financial burden and also leads to avoidance of undesirable side-effects of general anaesthesia.

Since the introduction of first Brachial plexus block using cocaine by Halstead (1884) the technique of Brachial plexus block has evolved from classical blind technique to use of nerve stimulators and Ultrasound guidance for supraclavicular Brachial plexus block [1]. Many additives to local anaesthetics' such as opioids, Clonidine, Neostigmine and Tramadol etc. have been used to increase the duration of the block, to improve postoperative pain management [2] and to avoid the need for placing catheter for continuous local anaesthetic drug infusion. Dexmedetomidine a newer  $\alpha_2$  - Adrenoreceptor agonist is currently in focus for its sedative, anxiolytic and analgesic properties.

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In human beings, Dexmedetomidine has also shown to prolong the duration of block and post-operative analgesia when added to local anaesthetic in various regional blocks [3,4]. Most human studies of Dexmedetomidine as an adjuvant to local anaesthetics involved combinations with bupivacaine or levobupivacaine [5,6].

Due to unique pharmacologic properties and fewer side effects, though Ropivacaine is being preferred for peripheral nerve blocks, when compared with Bupivacaine its shorter duration of motor blockade and duration of analgesia are its disadvantages and these can be overcome by adding adjuvants. However, there are very few published studies on Dexmedetomidine in combination with Ropivacaine. The current study is designed with aim to evaluate the effect of adding Dexmedetomidine to Ropivacaine 0.5% in supraclavicular Brachial plexus block in terms of onset and duration of sensory and motor block and duration of postoperative analgesia.

## Materials and Methods

After ethical committee approval and written informed consent, 60 American Society of Anaesthesiologist (ASA) grade I or II patients, scheduled for elective upper limb surgery below mid-humerus level under supraclavicular Brachial plexus block were enrolled in this prospective, randomized, double-blind controlled trial.

The patients were randomized into two groups based on block randomization.

*Group R:* 30 patients received 30 ml of 0.5% Ropivacaine +2 ml saline.

*Group RD:* 30 patients received 30 ml of 0.5% Ropivacaine +2 ml Dexmedetomidine (1/2 ml of Dexmedetomidine diluted to 2ml).

### *Inclusion Criteria*

ASA I and II, 18-60 years, both sexes, mid humerus, elbow, forearm and hand surgeries were included.

### *Exclusion Criteria*

Patient refusal, coagulopathy, ASA III and above, H/O severe cardiovascular, pulmonary, kidney, liver disease, neurological, psychiatric, neuromuscular disorder, infection/sepsis/allergy, pneumothorax, and peripheral neuropathy were excluded.

Pre-anesthetic assessment of all the patients was done the day before scheduled surgery. Patients were premedicated with tablet alprazolam 0.25 mg and tablet ranitidine 150 mg on night before surgery.

Coded study drug solutions were prepared by an Anaesthesiologist not involved in further study and handed over to concerned Anaesthesiologist for administration. After shifting the patient to operating table, standard anaesthesia monitoring in the form of the baseline measurement of heart rate, non-invasive arterial blood pressure, and peripheral oxygen saturation (SpO<sub>2</sub>) was started. Intravenous access was achieved using 20G cannula in the non operative arm and ringers lactate was started. With all aseptic precautions, Supraclavicular brachial plexus block, was given with the patient lying supine with the head turned to opposite side. The high frequency linear probe of Ultrasound machine (sonosite micromax machine) is placed over the supraclavicular region and Brachial plexus is identified and is approached in plane using a 23G, 55mm needle. The local anaesthetic solution was injected after careful aspiration, and spread was seen encircling the trunks.

Assessment of sensory blockade was done in the dermatomal areas corresponding to median nerve, radial nerve, ulnar nerve and musculocutaneous nerve. Sensory blockade was tested using pin prick method along the distribution of the four nerves. Sensory block is graded [5] as-Grade 0=sharp pin felt, Grade 1= analgesia, dull sensation felt, Grade 2= anaesthesia, no sensation felt. Sensory onset is considered when there is no sensation to pin prick (Grade 2) along the distribution of any of the above -mentioned nerves. The duration of sensory blockade is defines as time interval between onset of sensory blockade and complete resolution of anesthesia of all the nerves.

Motor blockade assessment was done using the modified Bromage [6] scale for upper extremities on a three point scale. Grade 0 = normal motor function with full extension of elbow, wrist and fingers. Grade 1=decrease motor strength with ability to move fingers and/or wrist only, Grade 2= complete motor blockade with inability to move fingers. Onset of motor blockade is considered when there is Grade 2 motor blockade. The duration of motor block is defined as the time interval between the onset of motor block and recovery of complete motor function of the hand and forearm.

Sensory and motor blockade and vital parameters were assessed every minute after the completion of drug injection until the onset of block and then every 10 min intraoperatively and half hourly after the end

of surgery until first 12 hrs, thereafter hourly until the block had completely worn off.

Injection Diclofenac sodium 75 mg intramuscular was administered when VAS [7] score was  $\geq 4$ . The time between the end of local anaesthetic administration and first rescue analgesic administration was recorded as the duration of analgesia.

#### Statistical Analysis

Data were expressed as mean  $\pm$  standard deviation for quantitative variables, number, and percentage for categorical variables Chi-square ( $\chi^2$ ) test was used to compare in between groups.  $p < 0.05$  was considered statistically significant.

## Results

There was no statistically significant difference among the patients in the three groups with respect to age, height, sex ratio, duration of surgery, type of surgery and the ASA physical status (Table 1).

The sensory and motor block onset and duration was significantly quicker in group RD than in group R. (Table 2).

The mean sensory block onset time was  $6.1 \pm 3.4$  min in group RD as compared to  $8.3 \pm 4.4$  min in group R ( $p < 0.001$ ) (Figure 1).

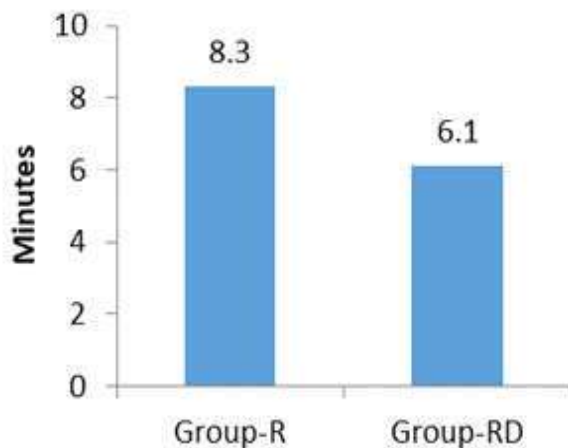
The mean motor block onset time was  $13.0 \pm 5.6$  min in group R when compared to  $8.9 \pm 3.08$  min in group RD. ( $p < 0.001$ ) (Figure 2).

**Table 1:** Comparison of demographic parameters

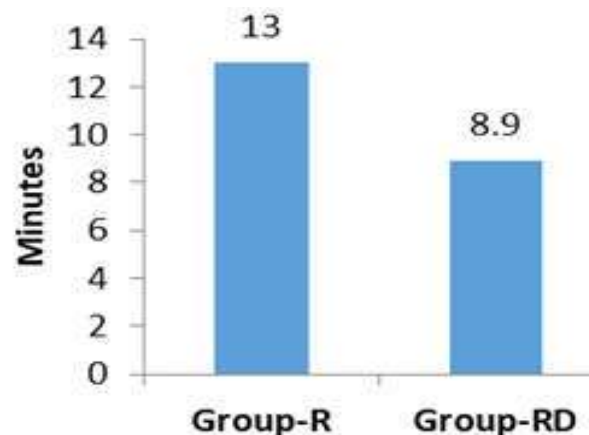
Demographic data	Group-R(n=30) (M $\pm$ SD)	Group-RD(n=30) (M $\pm$ SD)	P value
age(years)	36.86 $\pm$ 9.23	35.43 $\pm$ 8.84	0.54(students unpaired t-test)
Weight(Kg)	59.33 $\pm$ 7.86	57.96 $\pm$ 8.81	0.52(students unpaired t-test)
Sex ratio(male: Female)	23/7	25/5	0.37(Fischers Exact Test)
Duration of surgery(min)	115.33 $\pm$ 16.76	114.66 $\pm$ 16.33	0.87(Students unpaired t-test)

**Table 2:** Comparison of block characteristics

Block Characteristics	Group-R(n=30) (M $\pm$ SD)	Group-RD(n=30) (M $\pm$ SD)	P value
onset of sensory Sensory(min)	8.3 $\pm$ 4.4	6.1 $\pm$ 3.4	<0.001
onset of motor blockade(min)	13.0 $\pm$ 5.6	8.9 $\pm$ 3.08	<0.001
Duration of Sensory blockade(min)	400.8 $\pm$ 86.6	630.6 $\pm$ 208.2	<0.001
Duration of motor Blockade(min)	346.9 $\pm$ 76.9	545.9 $\pm$ 224.0	<0.001
Duration of analgesia(min)	411.0 $\pm$ 91.2	805.7 $\pm$ 205.9	<0.001



**Fig. 1:** Onset of sensory blockade (min)



**Fig. 2:** Onset of motor blockade (min)

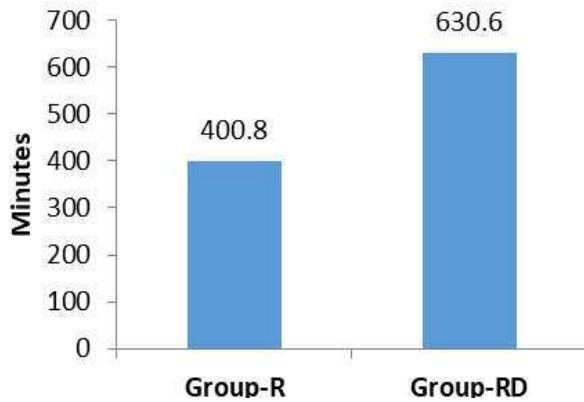


Fig. 3: Duration of sensory blockade (min)

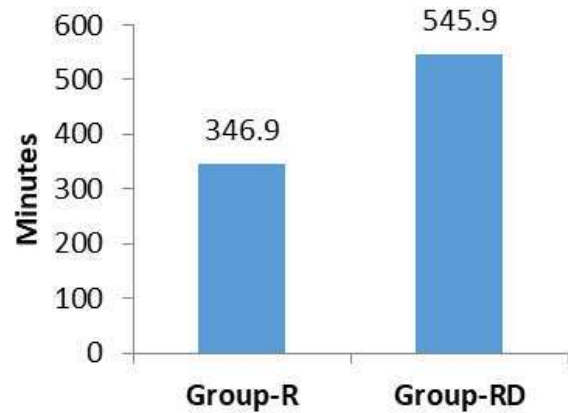


Fig. 4: Duration of motor blockade (min)

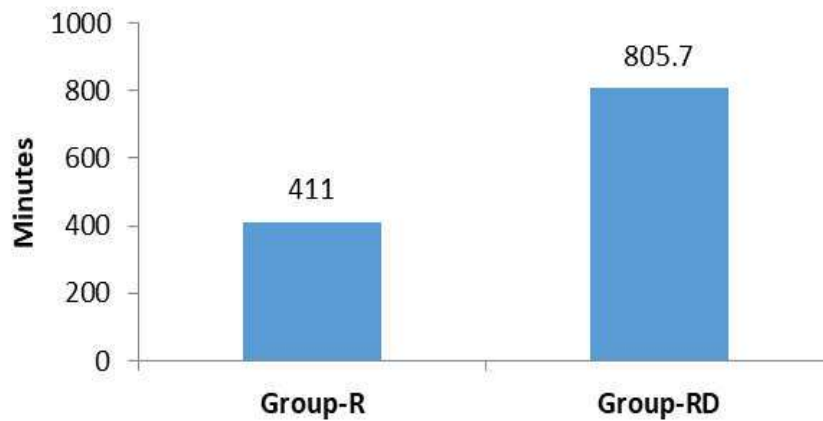


Fig. 5: Duration of analgesia (min)

Table 3: Ramsay sedation score

Score	Ramsay Sedation Score Response
1	Anxious or restless or both
2	Cooperative, oriented and tranquil
3	Responding to commands
4	Brisk response to stimulus
5	Sluggish response to stimulus
6	No response to stimulus

The duration of sensory as well as motor block was significantly prolonged in group RD as compared to group R. The duration of sensory block was more in group RD  $630.50 \pm 208.2$  min as compared to group R  $400.8 \pm 86.6$  min ( $P < 0.001$ ) (Figure 3). The duration of motor block was also more in group RD  $545.9 \pm 224.0$  min where as in group R it is  $346.9 \pm 76.9$  min ( $P < 0.001$ ) (Figure 4).

The duration of analgesia was significantly prolonged in group RD ( $805.7 \pm 205.9$  min) when compared with group R ( $411.0 \pm 91.2$  min) (Figure 5).

The total analgesic consumption in 24 h postoperatively was significantly higher in group R than group RD.

Patients in group RD were more sedated compared to group R. Most of the patients in our study had sedation score  $\leq 3$ . (Table 3).

No episode of hypoxemia or respiratory depression during 24 h period postoperatively was seen in any patient. Bradycardia was observed in 3 patients belonging to group RD intraoperatively that required no treatment. No episode of nausea, vomiting, or any other side-effect was observed.

## Discussion

Apart from sedative, analgesic, hemodynamic-stabilizing properties, and sympatholytic pharmacologic effects, the alpha ( $\alpha$ )-2-adrenergic receptor ( $\alpha_2$ -AR) agonists have been used to increase the duration of thermal anti-nociception and analgesia in some animal studies [8]. Animal studies have proven the combination of Dexmedetomidine with Ropivacaine to be safe and neuro-protective. The use of Dexmedetomidine decreases inflammation around peripheral nerves, thereby decreasing the potential for peripheral nerve injury. In human beings, the beneficial effects of adding Dexmedetomidine to local anaesthetics during regional anaesthesia and some peripheral nerve blockade procedures have proved to be efficacious for surgical patients [5]. We used 0.5% Ropivacaine for supraclavicular block. Ropivacaine is a long-acting regional anaesthetic that is structurally related to bupivacaine. It is a pure S (-) enantiomer, unlike bupivacaine. It developed for the purpose of reducing potential toxicity and improving relative sensory and motor block profiles. Ropivacaine has lower lipid solubility and has produced less central nervous system and cardiac toxicity than bupivacaine for which it is gaining popularity over bupivacaine for peripheral neural blockade when large volumes of local anaesthetic are required. Ropivacaine is as effective as bupivacaine and levobupivacaine when used in peripheral nerve blocks. Clinically adequate doses of Ropivacaine appear to be associated with a lower grade of motor block than bupivacaine [9].

However the brief duration of action of Ropivacaine may result in block resolution before the period of worst postoperative pain. Continuous catheter based nerve blocks can extend post operative analgesia but their placement require additional time, cost and skill. To overcome this various perineural adjuvants such as Opioids, Clonidine, Dexmedetomidine, Neostigmine, Midazolam [10], Dexamethasone [11], etc., were added to local anaesthetics in Brachial plexus block to achieve quick, dense and prolonged block.

The mechanism of the analgesic actions of  $\alpha_2$  agonists has not been fully elucidated and is probably multifactorial. A number of supraspinal and spinal sites modulate the transmission of nociceptive signals in the CNS. Peripheral  $\alpha_2$  Adrenoceptors may also mediate the antinociception [12].  $\alpha_2$  agonist by acting at any of these sites reduces nociceptive transmission, leading to analgesia. The

activation of inwardly rectifying  $G_i$ -protein-gated potassium channels resulting in membrane hyperpolarization and decreasing the firing rate of excitable cells in the CNS is considered to be a significant mechanism of the inhibitory neuronal action of  $\alpha_2$ -Adrenoceptors agonists. Reduction of calcium conductance into cells, thus inhibiting neurotransmitter release is other prominent physiologic action ascribed to  $\alpha_2$  adrenoceptors. This effect involves direct regulation of entry of calcium through N-type voltage-gated calcium channels and is independent of cAMP and protein phosphorylation and is mediated by  $G_o$  proteins [13]. These mechanisms represent two very different ways of effecting analgesia, that is, the nerve is prevented from firing, and it also prevents propagation of signals. Hence, it is mainly the direct peripheral action of Dexmedetomidine on nerves in block, which is responsible for these improvements rather than due to central action of Dexmedetomidine after absorption through block site into systemic circulation resulting in its systemic effects [14].

The rationale for choosing this concentration of 0.5% Ropivacaine in our study is supported by the study done by Klein et al. [15], who found that for Interscalene Brachial plexus block, increasing the concentration of Ropivacaine from 0.5% to 0.75% failed to improve onset or duration of block, suggesting that the risk of increased total dose of local anaesthetic may be avoided. Hickey and co-workers [16] have shown that 0.25% Ropivacaine when used for Subclavian perivascular Brachial plexus block for upper limb surgery required frequent analgesia supplementation due to the low concentration of local anaesthetic used.

In our study we used Ultrasound guided supraclavicular block the advantage being, avoidance of intraneuronal / intravascular injection, visualization of spread of local anaesthetic, low volume of drug, faster onset of action, and decreased need for rescue analgesia. Abrahams et al. [17] concluded that Ultrasound method improves the quality of blockade when compared to peripheral nerve stimulator for nerve identification.

Chan et al. [18] conducted study in 188 patients and demonstrated that axillary Brachial plexus block significantly improved the success rate under the guidance of Ultrasound with or without nerve stimulation. Finally, unsuccessful block was not encountered in any of the groups in our study.

Esmaoglu et al. [6] have concluded that Dexmedetomidine (100 $\mu$ g) when used as an additive

to 40 ml of 0.5% levobupivacaine prolongs auxiliary Brachial plexus block duration. They have shown that Dexmedetomidine shortened the sensory block onset time ( $9.03 \pm 1.15$  min in Dexmedetomidine group vs.  $10.46 \pm 1.30$  min in control group), the motor block onset time ( $9.50 \pm 1.04$  min in Dexmedetomidine group vs.  $11.10 \pm 1.24$  min in control group) and prolonged the duration of the sensory block ( $887 \pm 66.23$  min in Dexmedetomidine group and  $673 \pm 73.77$  min in control group), duration of the motor block ( $773 \pm 67.62$  min in Dexmedetomidine group and  $575 \pm 65$  min in control group) and postoperative analgesia ( $1008.69 \pm 164.04$  min in Dexmedetomidine group and  $887.14 \pm 260.82$  min in control group).

Swami et al. [19] concluded that Dexmedetomidine ( $1 \mu\text{g}/\text{kg}$ ) when added to local anaesthetic (35cc, bupivacaine 0.25%) 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 ( $1 \mu\text{g}/\text{kg}$ ).

In the accordance with study by Swami et al. and Esmoğlu et al. in our study no significant serious side effects were reported in any group except for lower pulse rates and blood pressures observed in Dexmedetomidine groups that were managed conservatively.

Hajashareef HM, Murugan et al. [20] in their study of comparison of Ropivacaine with Dexmedetomidine concluded that the mean onset of sensory block was 9.2 min in Group R and 6.8 min in the Group RD, which is statistically significant ( $p < 0.0001$ ). The mean onset of motor block was 13.12 min in Group R and 9 min in the Group RD, which is statistical significant ( $p < 0.0001$ ). The mean duration of sensory block was 506.2 min in Group R and 709 min in the Group RD. The mean duration of motor block was 478.8 min in Group R and 669.2 min in the Group RD ( $P < 0.0001$ ).

In comparison with this study in our study, we have found that addition of Dexmedetomidine ( $50 \mu\text{g}$ ) to 30 ml Ropivacaine 0.5% in Ultrasound-guided supraclavicular Brachial plexus block resulted in a quick onset of sensory and motor block, prolonged duration of both sensory and motor block, delayed time to first request for analgesia supplementation, that is, prolonged duration of analgesia, and a good quality of analgesia when compared with Ropivacaine alone.

## Conclusion

We conclude that addition of 50mcg Dexmedetomidine to Ropivacaine 0.50% solution in Ultrasound guided Supraclavicular Brachial plexus block hastens the onset of sensory and motor blockade, prolongs the duration of sensory and motor blockade when compared to Ropivacaine alone. It also significantly delays the demand for analgesia supplementation, and is not associated with any major side-effect.

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