

## Effect of Addition of Dexmedetomidine to a Mixture of Local Anaesthetics in Supraclavicular Brachial Plexus Block

Raghavendra Babu T.<sup>1</sup>, Divya Puskur Rao<sup>2</sup>, Lingaraju Y.K.<sup>3</sup>, Gurudatt C.L.<sup>4</sup>

<sup>1</sup>Assistant Professor <sup>2</sup>Junior Resident <sup>3</sup>Ex -Associate Professor <sup>4</sup>HOD, Department of Anaesthesiology, JSS University, Mysuru, Karnataka 570004, India.

### Abstract

**Background:** Supraclavicular brachial plexus block is a popular technique of anaesthesia for surgeries on upper limb. It is safe and effective & offers analgesia postoperatively. Certain group of drugs may be used as adjuvants to local anaesthetics to decrease the local anaesthetics doses and enhances the efficacy of analgesia while reducing the incidence of adverse reactions. This study was conducted to assess the effect of addition of Dexmedetomidine to a mixture of local anaesthetics for brachial plexus blockade in adult patients posted for elective upper limb orthopedic surgeries. **Objective:** To study the effect of addition of 50µg of Inj. Dexmedetomidine as an adjuvant to a mixture of 2% Lidocaine with adrenaline (1:200000) 15 ml + 0.5% Bupivacaine 15ml on time of onset, time for maximum effect, duration of sensory and motor blockade, duration of postoperative analgesia and to note perioperative haemodynamics and sedation scores. **Methods:** 60 patients of either gender in the age group of 20-60 years posted for elective upper limb surgeries were selected for the study. Patients were randomly divided into two groups (n = 30). Control Group C (n=30) received Inj. Lidocaine with adrenaline (1:2, 00,000) 2% = 15ml + Inj. Bupivacaine (0.5%) = 15ml + Inj. Sterile water = 5ml. Total volume = 35ml. The study Group- D (n=30) received Inj. Lidocaine with adrenaline (1:2, 00,000) 15ml+ Inj. Bupivacaine (0.5%) = 15ml + Inj. Dexmedetomidine 50µg (0.5 ml diluted to 5 ml sterile water= 5ml). Total volume = 35ml. They were compared for onset and duration of motor and sensory block and the duration of postoperative analgesia. **Results:** Onset of sensory block: Group D: 1.73 ± 0.9min vs Group C: 6.0 ± 1.8min (p<0.001). Onset of motor block: Group D: 2.8 ± 1.0min vs Group C: 8.5 ± 2.4 min (p<0.001). Duration of sensory block: Group D: 622 ± 166.4min vs Group C: 240.6 ± 63.02min (p<0.001). Duration of motor effect: Group D: 450 ± 136.2min vs group C (191.3 ± 48.9) min (p<0.001). Duration of analgesia: Group D: 1163.3 ± 334.6 min vs Group C: 351.3 ± 83.9min (p<0.001). **Conclusion:** As an adjuvant Inj. Dexmedetomidine, when added to local anaesthetics a) Shortens the time of onset of block, b) Prolongs the duration of sensory and motor block, c) Prolongs the duration of postoperative analgesia with better hemodynamic stability both intra operatively and post operatively.

**Keywords:** Adjuvant Dexmedetomidine; Local Anaesthetics; Supraclavicular Brachial Plexus Block; Upper Limb Orthopedics Surgery.

### Introduction

Once described as the “spinal of the arm,” a supraclavicular block offers dense anesthesia of the brachial plexus for surgical procedures on upper extremity. The block is performed at the level of the distal trunks and origin of the divisions, where the brachial plexus is confined to its smallest surface area [1]. It avoids the undesirable effects of polypharmacy

and the stress of laryngoscopy and tracheal intubation of general anaesthesia. Also, the postoperative period is usually uneventful [2].

Adjuvants are being used along with local anaesthetics in brachial plexus block to achieve fast, dense and prolonged blockade and to increase the duration of post-operative analgesia. The addition of alpha<sub>2</sub>-adrenergic agonists has been suggested to enhance the nerve block characteristics of local

**Corresponding Author:** Raghavendra Babu T., Assistant Professor, Department of Anaesthesiology, JSS University, Mysuru, Karnataka 570004, India.  
E-mail: [dr.babu77@gmail.com](mailto:dr.babu77@gmail.com)

Received on 08.07.2017, Accepted on 31.07.2017

anaesthetic solutions caused by local vasoconstriction, facilitation of C fiber blockade and a spinal action caused by slow retrograde axonal transport or simple diffusion along the nerve [3]. Unlike opioids and steroids which have various unwanted effects, the use of  $\alpha$ -2 agonists may improve block characteristics without producing much side effects. They may also help by decreasing the blood pressure thereby decreasing the blood loss and may also decrease PONV [4]. Dexmedetomidine  $\alpha$ 2 agonist can influence the characteristics of brachial plexus blocks. This study was undertaken to know how effective it is in the dose of 50 $\mu$ g along with lidocaine and bupivacaine in influencing the characteristics of block.

### Materials and Methods

After obtaining approval from Institutional Ethical Committee and written informed consent from the patients (60 patients) of ASA I and II physical status, aged between 20 and 60 years, belonging to either gender and posted for elective upper limb surgeries, were randomly allocated to two groups of 30 patients in each group. Patients were randomly divided into two groups (n = 30). Control Group C (n=30) received Inj. Lidocaine with adrenaline (1:2, 00,000) 2% = 15ml + Inj. Bupivacaine (0.5%) = 15ml + Inj. Sterile water = 5ml. Total volume = 35ml. The study Group- D (n=30) received Inj. Lidocaine with adrenaline (1:2, 00,000) 15ml+ Inj. Bupivacaine (0.5%) = 15ml + Inj. Dexmedetomidine 50 $\mu$ g (0.5 ml diluted to 5 ml sterile water= 5ml). Total volume = 35ml. Patients with known hypersensitivity to local anaesthetics, known case of any major bleeding disorder, local infection, any major illness, and patient on anticoagulants, patients with existing neurological disorder / nerve palsy, and pregnant patients were excluded from the study.

All patients were evaluated a day prior to surgery to rule out any of the above-mentioned exclusion criteria. Patients were instructed to be nil by mouth for 6 hours for solids and for 2 hours for clear fluids before surgery. Tablet Ranitidine 150mg and Tab Alprazolam 0.5mg were given orally on the night before the day of surgery. The haemodynamic variables such as heart rate, systolic blood pressure and diastolic blood pressure and mean arterial pressure were recorded preoperatively and the Supraclavicular brachial plexus block procedure to be performed was explained to the patient. Inj. Ondansetron 4 mg IV was administered and the patient was positioned in the position as, supine on

a straight table with the head turned away from the side to be blocked, neck extended with shoulder depressed and arm of the side to be blocked was adducted.

The study drugs were prepared by the anaesthesiologist who was involved with the randomization of patients and handed over to the observer anaesthesiologist who gave the block. The patient and observer were thus blinded to the study drugs. The area was painted and draped. The inter-scalene groove was identified and traced towards the clavicle and subclavian artery was palpated. The needle entry point was just above the subclavian artery in the inter-scalene groove. The needle was directed caudally as described by Winnie's subclavian perivascular approach. A peripheral nerve locator was used for accurate localization of the plexus with initial current of 1.5 mA. Twitches of the fingers at a current of 0.3mA were taken as the target before the drugs were injected.

Onset of sensory and motor block was assessed one minute after the end of injection of drug and every minute thereafter till peak effect occurred. A 3 point scale was used for the assessment of sensory block and motor block. Sensory block assessed by score- 0: Normal sensation; 1: Loss of pin pricks sensation (analgesia) 2: Loss of touch sensation (anaesthesia). Motor block was assessed by score- 0: Normal motor function; 1: Reduced strength but can move fingers (weak grip) 2: Complete motor block with inability to move fingers.

Sensory block was assessed by gentle pin prick method using tip of 23G needle at the thenar eminence (Median nerve), lateral side of dorsum of hand (Radial nerve), little finger (Ulnar nerve), lateral border of forearm (Musculocutaneous nerve). Time of onset of sensory block was defined as time interval from end of injection of drug to loss of sharp pin prick sensation in any of the areas supplied by the four nerves. Time for maximum sensory block was time interval from end of injection of drug to loss of pin prick sensation in the areas supplied by all the four nerves. Duration of sensory block was defined as time interval between end of local anaesthetic administration and the first pin prick sensation in the areas supplied by all the four nerves.

Motor block was assessed by evaluating the thumb abduction (Radial nerve), thumb opposition (Median Nerve), thumb adduction (Ulnar nerve) and flexion at elbow (Musculocutaneous nerve). The time of onset of motor block was defined as time interval from end of injection of drug to onset of (Grade- I) motor block, time for maximum effect was defined as time interval

from end of injection of drug to complete (Grade-2) motor blockade and duration of motor block was defined as the time interval between the end of local anaesthetic administration and complete motor recovery of the muscles supplied by all the 4 nerves.

Pain was assessed using a 10 point Visual Analogue Score (VAS) [5]. Duration of analgesia was calculated as the time interval from the end of injection of local anaesthetic till the patient developed VAS of  $\geq 4$  and requires rescue analgesia. Rescue analgesia was administered in the form of Inj. Diclofenac Sodium 1mg/kg intramuscularly. Time when rescue analgesia was requested and the total number of times analgesics were administered over a period of 24 hours was also noted.

Sedation was assessed using the Ramsay Sedation Score [6]: Score- 1: Patient anxious/ agitated or both; Score-2: Patient co-operative, oriented and tranquil; Score- 3: Patient responds to commands only; Score- 4: Brisk response to light glabellar tap; Score- 5: Sluggish response to glabellar tap; Score-6: No response

Haemodynamic parameters e.g. Heart rate, blood pressure, respiratory rate and oxygen saturation (SpO<sub>2</sub>) were recorded before giving the block, monitored immediately after giving the block and every 10 min after the block for 1 hour and every 20 min thereafter till the end of surgery. Postoperatively same parameters were observed immediately after the

end of surgery, every 10 min there-after for the 1st hour, every 30 min for the next 40 hours and thereafter every time we visited the patient for observing the effect of block and VAS score. Effect of sensory block, motor block and VAS were assessed every 30 min for first 2 hours, every 60 min for next 4 hours and then at 9, 12, 18 and 24 hours.

Patients were observed for complications e.g. hematoma, hypotension, pneumothorax, bradycardia, inadvertent arterial puncture, respiratory depression, post block neuropathy, dry mouth, infection, hypersensitivity, nausea, vomiting and local anaesthetic toxicity in the perioperative period. Episodes of hypotension were defined as fall in systolic blood pressure of more than 20% of preoperative value. Bradycardia was defined as heart rate less than 60 beats/ min and respiratory depression was defined as fall in SpO<sub>2</sub> less than 90% were noted.

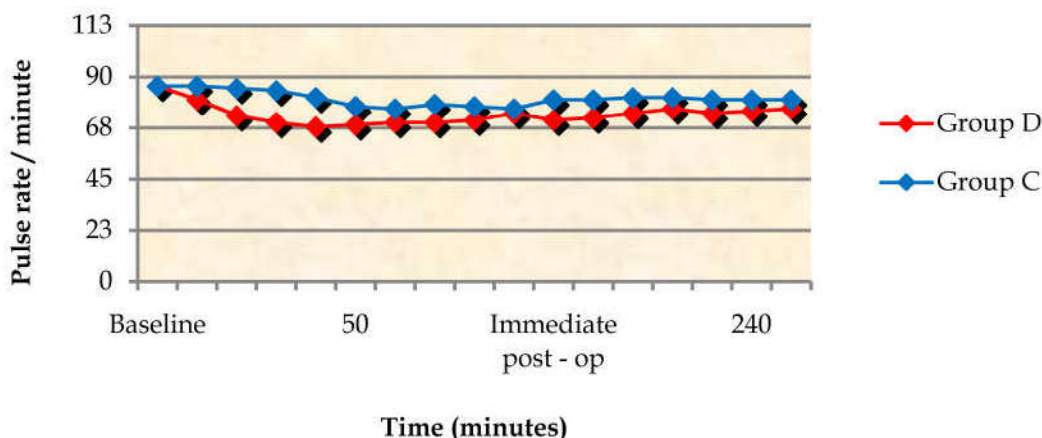
### Results

Demographic data and duration of surgery in both the groups were comparable without significant difference ( $p > 0.05$ : not significant). Number and type of different surgeries were comparable and there was no significant difference statistically.

Table 1: Demographic data

Variables	Group- D (n=30)	Group- C (n=30)
Age $\pm$ SD (mean years)	35 $\pm$ 7	38 $\pm$ 8.5
Gender (Male: Female)	22 : 8 (80% : 20%)	25 : 5 (70% : 30%)
Weight $\pm$ SD (mean kilogram)	51.8 $\pm$ 2.3	51.6 $\pm$ 2.3
Duration of surgery (mean min $\pm$ SD)	74.7 $\pm$ 24.1	71.3 $\pm$ 23.4

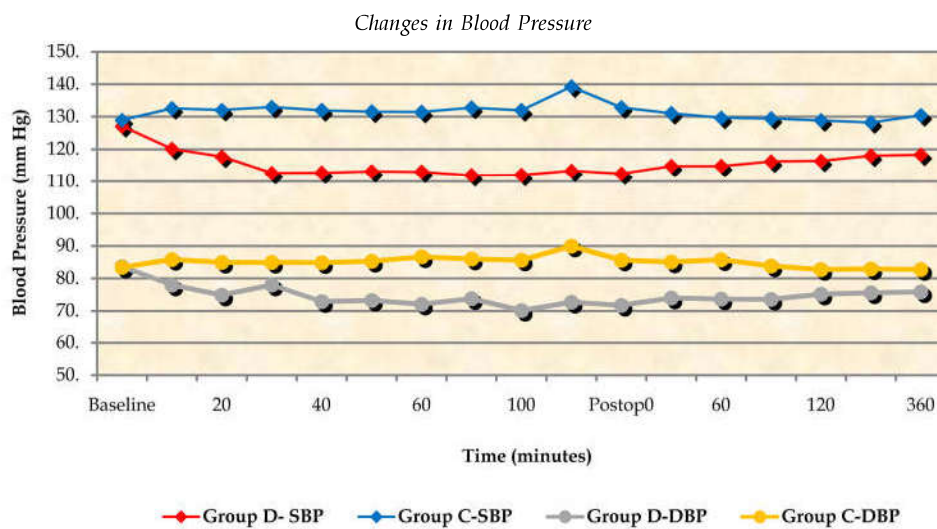
Changes in Heart Rate (Per Minute)



Graph 13: Perioperative pulse rate

**Table 2:** Parameters

Parameters (min= minutes)	Group- D (N=30)	Group- C (N=30)	P value
Onset of sensory block	6.0 ± 1.8	1.73 ± 0.9	< 0.001
Time for Maximum sensory block	12.6 ± 3.9	4.4 ± 1.6	< 0.001
Duration of sensory block	240 ± 63.02	624 ± 166.4	< 0.001
Onset of motor block	8.5 ± 2.4	2.8 ± 1.0	0.0001
Time for Maximum motor block	16.1 ± 4.4	6.1 ± 2.1	0.0001
Duration of Motor Block	191 ± 48.9	450 ± 136.2	< 0.001
Duration of perioperative analgesia	1163.3 ± 334.6	351.3 ± 83.9	< 0.001

**Graph 14:** Perioperative systolic blood pressure

When compared to Group- C patients, the patients in Group- D had significantly earlier onset of sensory and motor blockade as (p value- 0.001: highly significant). Group- D patients achieved maximum effect of sensory and motor blockade early as compared to Group- C patients which was highly significant (p value- 0.001). Total duration of sensory block and motor block was significantly prolonged in Group- D as compared to Group- C (p value- 0.001: highly significant). The total duration of perioperative analgesia was longer in Group- D patients as compared to patients in Group- C and the difference was statistically highly significant (p value- 0.001). By the end of 7th hour, all 30 patients (100%) in Group- C required rescue analgesia while only 4 patients (13.33%) required rescue analgesia in Group- D. The requirement of rescue analgesia was much faster in Group- C as compared to Group- D (p value- 0.001). By the end of 24 hours, only 18 patients (60%) required rescue analgesia in Group- D. Group- D patients had significantly longer duration of postoperative analgesia and the requirement of additional analgesics was significantly reduced (p value- 0.001). In Group- D, 40% of patients had a Ramsay sedation score of 1 point higher than patients in Group- C with score of 2.

Baseline heart rate and blood pressures were comparable in both the groups. (p > 0.05: not significant). In Group- D, at the end of the surgery there was a decrease of 15 beats/ min in the heart rate from the basal heart rate. In Group- C patients, there was only a slight decrease of 5 beats per minute from the basal heart rate. The patients in Group- D maintained stable blood pressures during the perioperative period. Both are statistically highly significant. Bradycardia was seen in 2 patients in Group- D which was treated with a single dose of Inj. Atropine 0.6mg IV. Bradycardia was responsive to Atropine. None of the patients in Group- C developed any complications

## Discussion

The effect of using only local anaesthetics for Brachial Plexus Block (BPB) will dissipate after a few hours and patient will experience severe pain thereafter. The efforts to prolong the duration of brachial plexus block by increasing the local anaesthetic doses are limited by their narrow therapeutic window [12]. Various adjuvants have

been used to prolong the duration of post-operative analgesia. Opioids, steroids, sodium bicarbonate and alpha-2 agonists are the more commonly used adjuvants. When opiates like morphine, buprenorphine, tramadol and fentanyl were used as adjuvants, many of the adverse effects of opioids like pruritus, PONV, respiratory depression, constipation and urinary retention were very common. Due to this reason, opioids are not very popular as adjuvants.

Since the early 1970s,  $\alpha_2$  adrenergic receptor agonists have been used successfully to treat patients with hypertension and patients withdrawing from long-term abuse of drugs or alcohol.  $\alpha_2$  Agonists produce diverse responses, including analgesia, anxiolysis, sedation, and sympatholytic, each of which has been reported in the treatment of surgical and chronic pain patients. Since adjuvants like opioids and steroids have various side effects, use of  $\alpha_2$  agonists may improve block characteristics without producing any side effects. They may further help by decreasing the blood pressure thereby decreasing the blood loss and may also decrease PONV [7]. Dexmedetomidine is approximately eight-times more selective than clonidine towards the  $\alpha_2$  adrenoceptor [7]. In previous clinical studies, intravenous Dexmedetomidine resulted in significant opioid sparing effects as well as a decrease in requirements of inhalational anaesthetics [8]. It has been reported in various animal studies that Dexmedetomidine enhances sensory and motor blockade and increases duration of analgesia [9,10,11,12]. In humans, it has been found that Dexmedetomidine prolongs the duration of block and postoperative analgesia when added to local anaesthetics in various regional blocks [2,13,14,15,16]. There number of studies using dexmedetomidine as adjuvant for supraclavicular brachial plexus block are limited. Hence, we chose to study dexmedetomidine as an adjuvant for BPB.

Various authors have used Dexmedetomidine in different doses and have found it to be effective. When we did the pilot study any dose above 50  $\mu\text{g}$  was producing profound sedation and bradycardia and doses below 50  $\mu\text{g}$  was not sufficient to produce significant effect on the duration of analgesia of local anaesthetics. Hence, we selected 50  $\mu\text{g}$  as the dose of Dexmedetomidine along with the traditionally used combination of 15 ml of lidocaine with adrenaline (1:200000) and 15 ml of 0.5% Bupivacaine. Dexmedetomidine acts on the reticular formation of the brain producing natural sleep like state, and hence an arousable sedation, will be highly beneficial for patients under regional anaesthesia. In our study, 12 patients had grade 3 Ramsay sedation score

compared to all the patients having Grade- 2 sedation in the control group. This is statistically significant. There was bradycardia in 2 patients in study group which was treated with Inj. Atropine 0.6 mg iv. The vital parameters like heart rate, systolic blood pressure, diastolic blood pressure and  $\text{SpO}_2$  were stable intraoperatively with statistically significant lower values in the Dexmedetomidine group. Similar findings were observed in the studies conducted by Esmoaglu et al.

## Conclusion

Dexmedetomidine (50  $\mu\text{g}$ ) is an ideal adjuvant to a mixture of local anaesthetics in Supraclavicular Brachial Plexus Block in adult patients as it leads to an early onset and maximum effect of sensory and motor block, with a longer sensory block than motor block. It provides prolonged duration of postoperative analgesia with decreased requirement of rescue analgesics. It maintains stable perioperative hemodynamics with adequate intraoperative sedation and minimal side effects and complications.

## References

1. Urmey WF. Upper extremity blocks. In: Brown DL, editor. Regional Anaesthesia and Analgesia. Philadelphia: W.B.Saunders, 1996:254-278.
2. Swami SS, Keniya VM, Ladi SD, Rao R. Comparison of Dexmedetomidine and Clonidine ( $\alpha_2$  agonist drugs) as an adjuvant to local anaesthesia in supraclavicular brachial plexus block: A randomized double-blind prospective study. Indian J Anaesth 2012 May;56(3):243-9.
3. Gandhi R, Shah A, Patel I. Use of Dexmedetomidine along with Bupivacaine for brachial plexus block. Natl J of Med Res. 2012;2(1):67-69.
4. Fischer HBJ. Brachial plexus anaesthesia. In: principles and practice of regional anaesthesia; Wildsmith JAW, Armitage EN, Mc Clure JH, editors, third edition. London: Churchill Livingstone, 2003.p.193-204.
5. Eisenach JC, De Kock M, Klimscha W. Alpha2-Adrenergic agonists for regional anaesthesia. A clinical review of clonidine (1984-1995). Anesthesiology 1996;85:655-674.
6. Ribotsky BM, Berkowitz KD, Montague JR. Local anaesthetics. Is there an advantage to mixing solutions? J Am Podiatr Med Assoc. 1996 Oct;86(10):487-91.
7. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proceedings (Baylor University Medical Center). 2001;14(1):13-21.
8. Duka I, Gavras I, Johns C, Handy DE, Cavras H. Role of the postsynaptic alpha 2-adrenergic receptor subtypes in catecholamine induced vasoconstriction. Gen Pharmacol 2000;34:101-6.

9. Tanaka K, Oda Y, Funao T, Takahashi R, Hamaoka N, Asada A. Dexmedetomidine decreases the convulsive potency of Bupivacaine and Levobupivacaine in rats: involvement of  $\alpha_2$ -adrenoceptor for controlling convulsions. *Anesth Analg* 2005;100:687-96.
  10. Chen BS, Peng H, Wu SN. Dexmedetomidine, an  $\alpha_2$ -adrenergic agonist, inhibits neuronal delayed-rectifier potassium current and sodium current. *Br J Anaesth* 2009; 103:244-254.
  11. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colarco MD: The effects of increasing plasma concentrations of Dexmedetomidine in humans. *Anesthesiology*; 2000;93: 382-394.
  12. Brummett CM, Norat MA, Palmisano JM, Lydic R. Perineural administration of Dexmedetomidine in combination with Bupivacaine enhances sensory and motor blockade in sciatic nerve block without inducing neurotoxicity in rat. *Anesthesiology* 2008;109:502-11.
  13. Ozalp, G.; Tuncel, G.; Savli, S.; Celik, A.; Doger, C.; Kaya, M et al. The analgesic efficacy of Dexmedetomidine added to ropivacaine patient controlled interscalene analgesia via the posterior approach. *Eur J Anaesthesiol*; 2006 June; 23:220.
  14. Esmoğlu A, Yegenoglu F, Akin A, Turk CY. Dexmedetomidine Added to Levobupivacaine Prolongs Axillary Brachial Plexus Block. *Anesth Analg*; 2010;111:1549-51.
  15. Obayah GM, Refaie A, Aboushanab O, Ibraheem N, Abdelazees M. Addition of Dexmedetomidine to Bupivacaine for greater palatine nerve block prolongs postoperative analgesia after cleft palate repair. *Eur J Anaesthesiol*. 2010 Mar;27(3):280-4.
  16. NYSORA - The New York School of Regional Anesthesia - Supraclavicular Brachial Plexus Block [Internet]. Nysora.com. 2016 [cited 10 October 2016]. Available from: <http://www.nysora.com/techniques/nerve-stimulator-and-surface-based-ra-techniques/upper-extremitya/3258-supraclavicular-brachial-plexus-block.html>.
-