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An evaluation of the addition of Dexmedetomidine to Levobupivacaine for supraclavicular brachial plexus block in upper limb orthopaedic surgeries

Atul Dixit[1], Shakti Singhal[2], Charu Neema[3], Sadhna Sanwatsarkar[4], Manohar Bhatia[5]

[1]*Professor, Department of Anesthesiology, Sri Aurobindo Medical College and Post Graduate Institute, Indore (M.P), India*

[2]*PG Scholar 3rd year, Department of Anesthesiology, Sri Aurobindo Medical College and Post Graduate Institute, Indore (M.P), India*

[3]*Assistant Professor, Department of Anesthesiology, Sri Aurobindo Medical College and Post Graduate Institute, Indore (M.P), India*

[4]*Professor and Head, Department of Anesthesiology Sri Aurobindo Medical College and Post Graduate Institute, Indore (M.P), India*

[5]*MD Community Medicine/PSM, G. R. Medical College, Gwalior, M.P., India*

ABSTRACT

Background and objectives: Adjuncts to local anaesthetics for brachial plexus block may enhance the quality and duration of analgesia. Dexmedetomidine, a selective α2-adrenoceptor agonist, has been used as an adjuvant during regional and local anesthesia. The purpose of this study was to assess the effect of dexmedetomidine added to Levobupivacaine in supraclavicular brachial plexus block. **Methods:** A prospective, randomized, single blinded pilot study was conducted on 40 ASA I or II adult patients undergoing upper limb orthopaedic surgeries under supraclavicular brachial plexus block. Patients were randomly divided into two groups. Patients in Group L(n = 20) were administered 29mL of 0.5% Levobupivacaine plus 1 ml NS and group LD (n=20) were given 29 ml of 0.5% levobupivacaine with dexmedetomidine 1μg/kg. The onset time and duration of sensory and motor blockade were recorded. **Results:** The onset of sensory and motor block was significantly faster in Group LD compared to Group L ($P < 0.05$). Rescue analgesic requirements were significantly less in Group LD compared to Group L ($P < 0.05$). **Conclusion:** Dexmedetomidine(1μg/kg) in combination with 29mL of levobupivacaine (0.5%) hastened onset of sensory and motor block . and improved postoperative analgesia when used in brachial plexus block, without producing any adverse events.

Keywords: Supraclavicular brachial plexus block; Dexmedetomidine; Levobupivacaine.

Introduction

Pain is as old as life. The most humane application of chemistry for the solace of mankind has been in the discovery of chemical compounds that prevent pain. Regional nerve blocks are blocks based on the concept that pain is conveyed by nerve fibres which are amenable to interruption anywhere along their pathway.[1]Supraclavicular brachial plexus block is a very popular mode of anaesthesia for various upper limb surgeries due to its effectiveness in terms of cost and performance, margin of safety and good postoperative analgesia.

**Correspondence*

Dr. Atul Dixit

Professor, Department of Anesthesiology, Sri Aurobindo Medical College and Post Graduate Institute, Indore, M.P., India

A variety of local anaesthetics and adjuvants have been studied for brachial plexus blockade. Levobupivacaine is a local anaesthetic drug belonging to amino amide group. It is the s.enantiomer of bupivacaine. Previous studies have shown levobupivacaine to have a greater margin of clinical safety with respect to both CVS AND CNS effect compared with racemicbupivacaine .[2] Dexmedetomidine is approximately eight times more selective towards alpha-2 adrenergic receptors.[3]. It has shown to prolong the duration of block and post operative analgesia when added to local anaesthetics in various regional blocks.[4,5,6]Our study evaluates the effects of addition of dexmedetomidine to levobupivacaine for supraclavicular brachial plexus block in upper limb orthopaedic surgery. The effects were studied in terms of: 1) Onset of sensory and motor blockade.2) Duration of sensory and motor blockade.3)Sedation score intra

and post-operatively.4) Haemodynamic variables (HR, BP, O2 saturation 5) Number of rescue analgesics in post-operative 24 hours. The above effects were compared with that of plain Levobupivacaine (0.5%).

Methodology

After obtaining approval of the ethics committee and written informed consent, 40 American Society of Anaesthesiologists grade-1 and grade- 2 patients of either sex aged 20 to 50 years scheduled for elective upper limb orthopedic surgeries e.g., open reduction and internal fixation for fracture radius, fracture ulna, fracture shaft humerus, under supraclavicular brachial plexus block with no prior premedication were included in this prospective, randomized, single blinded study. Unwilling patients, patients with history of allergy to local anaesthetics, infection at local local site of block, history of convulsions, bleeding disorders, cardiac, respiratory, renal or liver ailment, sensory neuropathy and motor deficit in the arm on which surgery is to be performed and patients with weighing more than 90kg were excluded from the study. The patients were assigned to 2 groups, each containing 20 patients. Control group - Group-L which received 29 ml Levobupivacaine (0.5%) and 1ml normal saline (NS) and the study group - Group LD: Received 29 ml of mixture of Levobupivacaine (0.5%) and Dexmedetomidine (1µ/kg).All patients were monitored for anaesthesia and analgesia upto 24 hours post-operatively, the sensory block was evaluated by a Hollmen scale Score [1] = Normal sensation of pinprick; [2] = Weaker sensation of pin prick felt as compared with other upper limb; [3] = Pin prick recognized as touch with blunt object; [4] = No perception of pin prick .The onset and duration of sensory loss and motor blockade was studied. The loss of pinprick sensation was checked every 3 minutes till the onset of loss of sensation and then every ½ hourly till the sensations were regained.The motor blockade was assessed every 3 minutes till the loss of movements and then every ½ hourly till the movements are regained. It was evaluated by Modified Bromage scale:

4 Full strength in relevant muscle groups
3 Strength reduction, but able to move against resistance
2 Ability to move against gravity, but not against resistance
1 Discrete movements (trembling) of muscle groups
0 Absence of movements

Onset of sensory block was defined as the time elapsed between injection of drug and complete loss of pin prick, while onset of motor blockade was defined as the time elapsed from injection of drug to complete motor block. Sedation score described by University of Michigan Sedation Scale (UMSS) was used to assess sedation. 1 – Awaked & Alert, 2 – Minimally Sedated: tired/sleepy responding to verbal stimulus, 3 – Moderately Sedated: somnolent/sleeping, responding to mild physical stimulus, 4 – Deeply Sedated: deep sleep, responding to moderate to severe physical stimulus. 5 – Unarousable.

Heart rate, non-invasive blood pressure and O2 saturation were monitored. Duration of sensory block (the time elapsed between injection of drug and appearance of pain requiring analgesia) and duration of motor block (the time elapsed between injection of drug and complete return of muscle power) were recorded. Intramuscular injection of Diclofenac sodium was given as rescue analgesic when patients complained of pain. Number of rescue analgesics in 24 hours of post-operative period was recorded.

Statistical analysis

All parameters were analyzed using SPSS version 21 and STAT 9.0 software. The data among groups were compared using unpaired t test.

Results

The minimum age of the patient was 20 years and the maximum age was 50 years. The mean age of the patients in group L was 32.50 ± 10.440 and in group L+D was 35.10 ±8.854. Age incidences between two groups were comparable. The mean time for onset of sensory block in group L was 13.50 ± 0.607 min, and L+D 6.85±0.745 min. The statistical analysis by Unpaired t test showed that, the time for onset of sensory block in group L+D was significantly faster when compared to groupL (P< 0.001). **(Table 1(a))**.The mean time for onset of motor block in group L+D was 13.25 ± 0.550 min and in group L was 16.55±0.605. The statistical analysis by Unpaired t test showed that, the time for onset of motor block was significantly faster in group L+D when compared to group L (P< 0.000). **(Table 1(b))**.Patients of all two groups were observed for 24 hours. Time was noted when the patient asked for rescue analgesics. The mean duration of sensory block in group L+D was 15.55 ± 0.605 hours and in group L was 11.10±1.373. The statistical analysis by Unpaired t test showed that the duration of sensory block in group L+D was significantly longer when compared to group L (P < 0.000). **(Table 2(a))**.The mean duration of motor block in group L+D was 13.85 ± 0.366 hours, and in group L was 9.10±1.119 hours. The statistical analysis by

Unpaired t test showed that the difference between duration of motor block in group LD was significantly longer when compared to group L (P < 0.000). **(Table 2(b))**

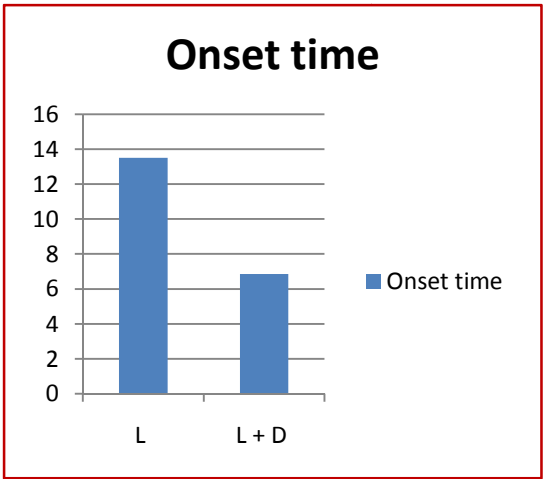
Table No. 1(a): Time for Onset of Sensory block (In Minutes)

Study Group	Onset time (Mean ± SD)	Mean difference	t value	p Value	Significance
L	13.50 ± .607	6.650	30.944	.000	HS
L + D	6.85 ± .745				

Table No. 1(b): Time for Onset of Motor Block (In Minutes)

Study Group	Onset time (Mean ± SD)	Mean difference	t value	p Value	Significance
L	16.55 ± .605	3.300	18.051	.000	HS
L + D	13.25 ± .550				

In group L, the mean pulse rate ranged from 70.50 ± 5.871 to 77.95 ± 7.141 beats / min. And group L+D,the mean pulse rate ranged from 69.50 ± 4.199 to 79.75 ± 4.723 beats/min.statistical analysis by Unpaired t test showed that there was no significant difference in pulse rate between the two groups (P > 0.05)



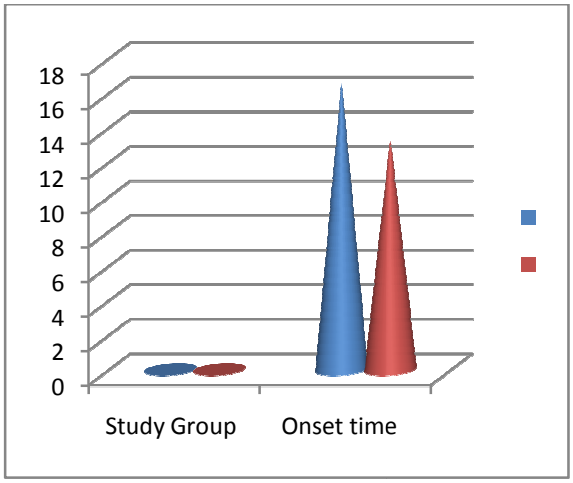


Fig. 1 (a): Time for Onset of Sensory block (Minutes) Fig. 1(b): Time for Onset of Motor Block (Minutes)

Table No. 2(a):Time for Duration of Sensory Block (In Hour)

Study Group	duration time (Mean ± SD)	Mean difference	t value	p Value	Significance
L	11.10 ± 1.373	-4.450	-13.267	.000	HS
L + D	15.55 ± .605				

Table No. 2(b): Time for Duration of Motor Block (In Hour)

Study Group	duration time (Mean ± SD)	Mean difference	t value	p Value	Significance
L	9.10 ± 1.119	-4.750	-18.038	.000	HS
L + D	13.85 ± .366				

In group L, the mean systolic blood pressure ranged from 113.35 ± 9.086 to 119.55 ± 7.149 mmhg. And group L+D, the mean systolic blood pressure ranged from 110.15± 4.158 to 119.85 ± 4.308 mmhg. statistical analysis by student's unpaired 't' test showed that there was no significant difference in systolic blood pressure between the two groups (P > 0.05).In group L, the mean diastolic blood pressure ranged from 72.10 ± 5.457 to 76.05 ± 4.904 mm hg. And group L+D, the mean diastolic blood pressure ranged from 70.35 ± 5.480 to 77.30 ± 3.672 mm hg.Statistical analysis by Unpaired t test showed that there was no significant difference in diastolic blood pressure between the two groups (P > 0.05).In group L, the mean O2 saturation ranged from 99.50 ± 0.761% to 99.95 ± 0.224%. In group L+D the mean O2 saturation ranged from 99.50 ± 0.761% to 99.95 ± 0.224%. The statistical analysis byUnpaired t test showed that there was no significant difference in O2 saturation between the two groups (P > 0.05).The total number of rescue analgesics used in the form of I/V Diclofenac 75 mg. In group L+D, 100% patients required only 1 rescue analgesic dosage in post-op 24 hours. In group L, 75% patients required 2 and 25% of patient required 3 rescue analgesic doses in post-op 24 hours. In group L, all patients were awake and alert and had sedation score of 1. In group L+D 9 patients had score of 1 in 15min,score 2 in 11 patients in 15 min, In 30min score of 1 in 11 patients, score 2 in 9 patients in 30min. and in 20 patients had score of 1 in 60min. None of the patients had sedation score of 3 and above during the study period. Statistical analysis of sedation score by Chi-square test showed that the difference in sedation score was significant (P < 0.05).

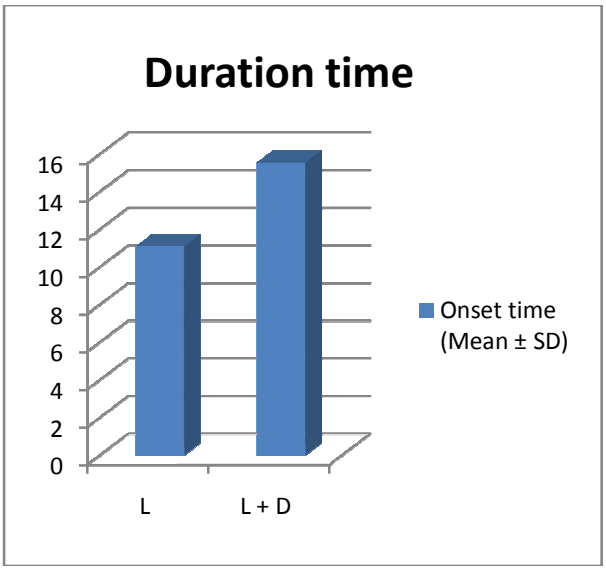


Fig. 2(a): Time for Duration of Sensory Block (Hour)

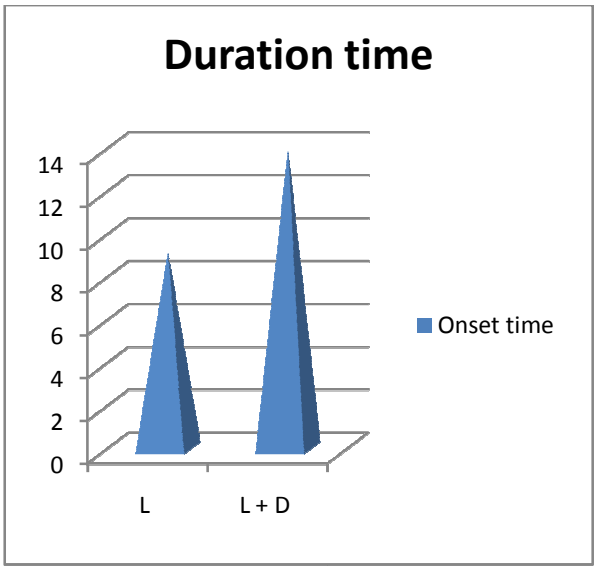


Fig. 2(b): Time for Duration of Motor Block (Hour)

Discussion

Scientific confirmation of the cardiac toxicity of bupivacaine in 1980s[] stimulated experimental studies with its enantiomers, which indicated lower cardiopressor activity of S(-) bupivacaine (levobupivacaine).[7,8,9,10] Considering the greater toxicity potential and the cardiovascular effects of the racemic mixture ,levobupivacaine seems a good indication for brachial block. To date, there has been an increasing use of some adjuncts (eg, opioids, α2-adrenoreceptor agonists) to local anesthetics to improve the block quality in peripheral nerve blocks. It was suggested in some studies that the addition of α2 agonists to local anesthetics in peripheral nerve blocks improved the block quality and extended the block duration.[11,12,13,14,15].The mechanism of action of α2-adrenoceptor agonists in peripheral nerve blocks is not understood fully.. The most probable mechanisms include vasoconstriction, central analgesia, and anti-inflammatory effects.[12-15] Clonidine, the prototype of alpha 2 agonists has been synthesized in early 1970.Dexmeditomidine is a new alpha 2 agonist that received USFDA approval in 1999 for use as a short term (less than 24 hrs.) Sedative analgesic in the ICU. alpha adrenoceptors agonists

have different alpha 2/alpha 1selectivity.Dexmedetomidine is a more selective α2 agonist than clonidine. Many studies evaluated the effects of dexmedetomidine on neuroaxial and peripheral nerve blocks[4,5,13]and dexmedetomidine was reported to be safe and effective in these studies. In a study that compared the effects of adding either clonidine or dexmedetomidine to lidocaine during a Bier block, it was found that adding dexmedetomidine improved the quality of anesthesia and analgesia more than the addition of clonidine. In 2 other studies, a dexmedetomidine–lidocaine mixture was used to provide a Bier block and was found to improve the quality of anesthesia and reduce postoperative analgesic requirement.[5,13] Yoshitomi *et al*., demonstrated that dexmedetomidine as well as clonidine enhanced the local anesthetic action of lignocaine via peripheral α-2A adrenoceptors. [16]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. [17,18] 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 direcct on the peripheral nerve activity). [19]Bajwa et al had compared dexmedetomidine and clonidine in epidural anesthesia and concluded that dexmedetomidine is a better neuraxial adjuvant compared with clonidine for providing an early onset of sensory analgesia and prolonged postoperative analgesia [20]. Esmaoglu et al evaluateds the effects of dexmedetomidine in axillary brachial plexus blocks. Esmaoglu et al found that adding dexmedetomidine to levobupivacaine for an axillary brachial plexus block shortens both the sensory and motor block onset time, extends the block duration, and the analgesia period.They also indicated that dexmedetomidine may lead to bradycardia. Bradycardia did not occur in our study, which is another point on which our study differs. We thought that the different results of the study by Esmaoglu set al, such as the shortened motor block onset time and the occurrence of bradycardia, in contrast to those of our study, could be related to their use of the higher dexmedetomidine dose of 100 μg in all patients.[13]In our study, we compared the effects of addition of dexmedetomidine 1μg/kg (Group L+ D) to levobupivacaine 0.5%, with the control (group L) in supraclavicular brachial plexus block. The result of our study shows that all patients in the two groups were comparable with respect to demographic profile, duration of surgery and type of surgery. With these doses, we had stable haemodynamics in patients in all the two groups .There was no significant difference in pulse rate, mean systolic and mean diastolic blood pressure and oxygen saturation in the two groups ($p > 0.05$).We also found that onset of sensory block and motor block was faster with Group L+ D as compared to Group L, was statistically significant. The duration of sensory and motor block in Group L+ D was longer than Group L, and it was also statistically significant. The requirement of rescue analgesic in group L+D was lesser than Group L. .None of the patients had sedation score of 3 and above during the study period. Statistical analysis of sedation score by Chi-square test showed that the difference in sedation score was significant ($P < 0.05$).

Conclusion

In our study we found that dexmedetomidine when added to levobupivacaine for supraclavicular brachial plexus block shortens the onset of sensory and motor blockade and prolongs their duration . The prolonged duration of analgesia significantly decreases the need for additional analgesics in dexmeditomidine group . The added advantage of conscious sedation, hemodynamic stability, and minimal side effects makes dexmedetomidine a potential adjuvant for nerve blocks. Since ours is a pilot study, further studies with large sample sizes are warranted to validate these finding.

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