COMPARATIVE STUDY OF THE EFFECT OF ADDITION OF DEXMEDETOMIDINE AS AN ADJUVANT TO LOCAL ANAESTHETICS IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

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ABSTRACT

BACKGROUND
Brachial plexus block is a popular and widely employed regional nerve block of the upper extremity. A variety of adjuvants have been used to enhance the effect of local anaesthetics in peripheral nerve block. In this study, we evaluated the effect of Dexmedetomidine as an adjuvant to 0.5% Bupivacaine plus 2% Lignocaine hydrochloride with adrenaline (1 in 200000) in Brachial plexus block in terms of onset and duration of sensory and motor block, duration of analgesia and hemodynamic effects.

MATERIALS AND METHODS
Sixty patients of age 18-60 yrs. belonging to ASA grade I and ASA II who came for upper limb surgery below the shoulder joint under brachial plexus block through supraclavicular route were divided into two equal groups. Group BS was given 20 millilitres (ml) of 0.5% Bupivacaine + 20 mL of 2% lignocaine with adrenaline (1 in 20000) + one ml of Normal saline and Group BD was given 20 mL of 0.5% bupivacaine + 20 mL of 2% lignocaine with adrenaline (1 in 20000) + 50 mcg of Dexmedetomodine (1 mL). The following brachial plexus nerve block parameters were assessed: onset and duration of sensory and motor blockade, duration of analgesia, Ramsay sedation score, hemodynamic parameters, and any side effects.

RESULTS
There were no significant differences in the patient and surgery characteristics between the two groups. The onset of sensory blockade in group BD was 7.07±4.502 minutes and in group BS was 7.33±3.294 minutes (p value = 0.794) which is comparable. The onset of motor blockade was 14.87±5.38 minutes in group BD and 16.30±3.109 minutes (p value =0.211) in group BS which is statistically insignificant. The duration of sensory blockade (BD = 534.83±149.09 minutes vs BS = 302.17 ±65.465 minutes) and motor blockade (BD = 425.50±140.19 minutes vs BS=256.83±58.758 minutes) was longer in group BD compared to group BS with a p value <0.05 which is statistically significant. The duration of analgesia was also longer in group BD (582.83±198.44 minutes) when compared to group BS (326.33±60.78 minutes) with a p value = 0.002 which is statistically significant. Patients in group BD showed slightly higher Ramsay sedation scores when compared to group BS.

CONCLUSION
Dexmedetomidine is a good adjuvant to local anaesthetic agents, as its addition to bupivacaine and lignocaine was associated with prolonged sensory and motor blockade with relative hemodynamic stability and greater postoperative analgesia and no significant side effects.

KEYWORDS
0.5% Bupivacaine, 2% Lignocaine with Adrenaline, Brachial Plexus Block, Dexmedetomidine, Supraclavicular Route.

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BACKGROUND
Brachial plexus block is the most common regional anaesthetic technique employed for surgeries of the upper limb. Upper limb surgeries are increasingly being performed under brachial plexus block as it not only provides intraoperative anaesthesia but also extends analgesia even in the postoperative period, while avoiding the complications of general anaesthesia and hence improves the patient outcome.1 Brachial plexus block can be given through different approaches but supraclavicular approach is preferred over other routes for surgeries on the arm, forearm and hand. A variety of drugs have been studied as adjuvants for brachial plexus block including opioid and non-opioid agents.2,3,4 Alpha-2 adrenoreceptor agonists like clonidine, dexmedetomidine have been the focus of interest, nowadays, for their sedative, analgesic, perioperative sympatholytic, and cardiovasculaer stabilizing effects.2,4 Dexmedetomidine has eight times higher affinity for α2 receptors than that of Clonidine and this makes it a much...
more effective sedative and analgesic agent compared to clonidine.

In this study, we assessed the effect of Dexmedetomidine as an adjuvant to Bupivacaine and Lignocaine in supraclavicular brachial plexus block in terms of onset and duration of sensory and motor blockade, duration of analgesia, sedation, haemodynamic effects and Complications /side effects if any. Both Bupivacaine and Lignocaine with adrenaline have been used in this study to get an adequate volume of local anaesthetic solution for effective blockade without increasing the toxicity.

**MATERIALS AND METHODS**

This prospective, randomized study was performed on 60 adult patients of age 18-60 years, undergoing elective upper limb surgeries under supraclavicular brachial plexus block in Santhiram Medical College and Hospital from June 2018 to January 2019. Approval from Hospital ethical committee was taken and written informed consent was obtained from patients. All the patients were assessed for ASA physical status. After preanaesthetic evaluation and basic laboratory investigations, patients were taken up for surgery.

**Inclusion Criteria**

Patients posted for orthopedic surgeries on upper limb belonging to ASA grade 1 and 2 in the age group of 18-60 years of either sex.

**Exclusion Criteria**

ASA class III, iv, v, and vi, Patient refusal, patients with bleeding disorders, peripheral neuropathies, Patients with known allergy to local anaesthetics or dexmedetomidine, Local infection at the site of injection, Patients on any sedatives or antipsychotic drugs, Hepatic failure, renal failure, uncontrolled Diabetes mellitus, Patients on adrenoreceptor agonist and antagonist therapy.

The patients were explained about the anaesthetic procedure during pre-anaesthetic evaluation and also before surgery in the operation theatre. They were also educated about the visual analogue scale.

**Procedure**

The patients were randomly divided into two equal groups: Group BS (n=30) received a total of forty-one ml solution containing 20 ml of 0.5% Bupivacaine+ 20 ml of 2% Lignocaine with adrenaline (1 in 200000) +1 ml of normal saline.

Group BD (n=30) received a total of forty-one ml solution containing 20 ml of 0.5% Bupivacaine + 20 ml of 2% Lignocaine with adrenaline (1 in 200000) +50 micrograms of Dexmedetomidine (1 ml).

On arrival of patient in the operation theatre, baseline heart rate, blood pressure and oxygen saturation were recorded. Intravenous cannulation with 18 G cannula was secured in the non-operating upper limb.

Neural localization was achieved by using peripheral nerve stimulator (Stimuplex, Braun, Germany) connected to a 22G, 50 mm long needle. Plexus block was considered successful when at least three out of the four nerve territories (ulnar, radial, median, and musculocutaneous) were effectively blocked for both sensory and motor block.

Sensory block was assessed by pinprick test using 3-point scale- 0) Normal sensation, 1) Loss of sensation to pinprick, 2) Loss of sensation to touch.

Motor blockade was assessed using Bromage 3 point score- 1) Normal sensation, 2) Decreased motor strength with ability to move fingers only, 3) Complete motor block with inability to move fingers.

Onset of sensory block was defined as the time elapsed between the end of local anaesthetic administration and complete sensory block. Onset of motor block was defined as the time elapsed from the injection of drug to complete motor block.

Sedation of the patient was assessed using Ramsay Sedation Score. Sensory and motor blocks were evaluated every 1 minute during the first 5 minutes, then every 3 minutes until 30 minutes.

Heart rate, NIBP, SpO2, Respiratory rate was noted every 5 minutes(mins) during the first 15 mins, then every 15 mins throughout the surgery and every 30 minutes in the first hour of postoperative period.

I.m Injection of Tramadol 100 mg was given as rescue analgesic when patients complained of pain which was assessed with visual analogue scale (VAS). Patients with VAS >4 (mild, annoying pain) received Inj. Tramadol 100 mg, I.V for post op pain, when required. Duration of analgesia was taken as the time interval between drug injection and the time of first analgesic request after surgery. Adverse events like hypotension (20% decrease in relation to baseline) and bradycardia (heart rate <50 beats per minute) were corrected with inj. Mephentermine in 6 mg incremental doses and 0.6 mg atropine respectively.

**Statistical Analysis**

The obtained data was analyzed using SPSS 22; descriptive data was expressed as Mean ± SD. Comparison of continuous variables was done by using independent samples ‘t’ test and categorical variables were compared by Chi-square test. The intraoperative hemodynamic variability was studied using the repeated measures linear model. “P” <0.05 was considered as significant.

**RESULTS**

The demographic data and surgical characteristics were comparable in both the groups as shown in Table 1. The onset of sensory and motor block was shorter in Group BD than in group BS (BD - 7.07 ± 4.5 min vs. 7.33±3.21 mins in BS and BD- 14.87± 5.38 vs. 16.30 ±3.10 mins in BS respectively) but the difference was statistically insignificant (Table 2).

The onset of Motor Blockade is 1.43 minutes less in Group (BD) when compared to Group (BS).
<table>
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<th>Parameter</th>
<th>Group BS</th>
<th>Group BD</th>
<th>p-Value</th>
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<tr>
<td>Age (Years)</td>
<td>38.4±16.09</td>
<td>41±13.14</td>
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<td>Male Gender (%)</td>
<td>16 (53.3%)</td>
<td>21 (70%)</td>
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<tr>
<td>Weight (Kgs.)</td>
<td>60.7±10.54</td>
<td>58.63±7.05</td>
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<td>ASA Grade</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 1 (%)</td>
<td>25 (83.3%)</td>
<td>23(76.67%)</td>
<td>NS</td>
</tr>
<tr>
<td>Grade 2 (%)</td>
<td>5 (16.7%)</td>
<td>7(23.33%)</td>
<td>NS</td>
</tr>
<tr>
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<td></td>
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<td>12</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>Radius /Ulna</td>
<td>14</td>
<td>16</td>
<td>NS</td>
</tr>
<tr>
<td>Hand</td>
<td>4</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Duration of Surgery (mins)</td>
<td>75±34.8</td>
<td>80.54±33</td>
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**Table 1. Demographic Data and Surgical Characteristics**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group</th>
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<th>Standard Deviation</th>
<th>p Value</th>
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<tr>
<td>Onset of Sensory Block (minutes)</td>
<td>BS</td>
<td>7.33</td>
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<td></td>
<td>BD</td>
<td>7.07</td>
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<tr>
<td>Onset of Motor Block (minutes)</td>
<td>BS</td>
<td>16.30</td>
<td>3.109</td>
<td>0.211</td>
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<tr>
<td></td>
<td>BD</td>
<td>14.87</td>
<td>5.380</td>
<td></td>
</tr>
<tr>
<td>Duration of Sensory Block (minutes)</td>
<td>BS</td>
<td>302.17</td>
<td>65.465</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>BD</td>
<td>534.83</td>
<td>149.069</td>
<td></td>
</tr>
<tr>
<td>Duration of Motor Block (Minutes)</td>
<td>BS</td>
<td>256.83</td>
<td>58.758</td>
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<tr>
<td></td>
<td>BD</td>
<td>425.50</td>
<td>140.199</td>
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<td>Duration of Analgesia (Minutes)</td>
<td>BS</td>
<td>326.33</td>
<td>60.784</td>
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<td></td>
<td>BD</td>
<td>582.83</td>
<td>198.441</td>
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</table>

**Table 2. Comparison of Block Characteristics in Both Groups**

The duration of sensory blockade was prolonged in Group BD (534.83±149.069 mins) when compared to Group BS (302.17±65.46 mins) and is statistically significant (p-0.001). The mean ± standard deviation of the duration of motor blockade (425.5±140.199 mins) was more in Group BD when compared to Group BS (256.83±38.75 mins) and is statistically significant (p-0.002). The duration of analgesia was also significantly prolonged in Group BD when compared to Group BS (582.83±198.44 vs. 326.33±60.78 mins, P 0.002).

BD – Bupivacaine plus lignocaine with adrenaline plus dexmedetomidine group.

BS – Bupivacaine plus lignocaine with adrenaline plus normal saline group.
The baseline hemodynamic parameters were comparable in both the groups. There was no statistically significant difference in baseline heart rate between the two groups. However, there was statistically significant reduction in heart rate from 15 minutes onwards in dexmedetomidine group compared to saline group (P=0.000). (shown in graph 4). There was significant fall in systolic blood pressure from 30 minutes onwards and significant fall in diastolic blood pressure from 15 minutes onwards in dexmedetomidine group compared to saline group. (graphs 5 and 6).

The Ramsay sedation score was assessed at arrival and in postoperative period in the two patient groups. The score was the same in both groups on arrival as expected. After 15 minutes, sedation score was significantly higher in dexmedetomidine group (score 3 in 28 patients, score 2 in 2 patients) compared to the saline group (score 2 in all 30 patients and none recorded score 3) up to 480 minutes. (P=0.000).

The safety profile of using dexmedetomidine along with bupivacaine and lignocaine was carefully assessed. Only 2 patients in each group had bradycardia and remaining patients developed no side effects such as heart block, arrhythmias, hypotension, respiratory depression, nausea or vomiting. Bradycardia in those two patients was treated with 0.6 mg Atropine I.V. There was no statistically significant difference between the two groups.

DISCUSSION

Pain relief is of paramount importance in anaesthesia. Peripheral nerve blocks are routinely being employed, nowadays, as an alternative to general anaesthesia or spinal anaesthesia, as an adjunct to general anaesthesia and as well as for postoperative analgesia by anaesthesiologists for various surgeries. Brachial plexus block is being routinely employed now-a-days as an alternative to general anaesthesia for upper limb surgeries. Bupivacaine, a local anaesthetic has longer duration of action with high cardiotoxic potential. Lignocaine with adrenaline is a local anaesthetic with faster onset and shorter duration of action compared to bupivacaine, but its cardiotoxicity is less when compared to bupivacaine. In this study, we used a combination of 0.5% Bupivacaine and 2% Lignocaine with adrenaline to get an adequate volume of local anaesthetic for effective brachial plexus blockade without exceeding the toxic dose, so as to avoid local anaesthetic toxicity. We used peripheral nerve stimulator for guiding us in giving the block in our study as there is no availability of ultrasound in our operation theatre for ultrasound guided blocks. With ultrasound guided block, the volume of local anaesthetic used can be decreased. There has always been a search for adjuvants to prolong the duration of analgesia and improve the quality of block. Alpha 2 adrenergic agonists have been tried with local anaesthetics in various routes such as epidural, intrathecal, and peripheral nerve blocks in the recent past to prolong and improve the quality of anaesthesia.

In this study, we compared dexmedetomidine (an alpha-2 adrenergic receptor agonist) with bupivacaine 0.5% plus 2% lignocaine with adrenaline versus bupivacaine 0.5% plus 2% lignocaine with adrenaline in supraventricular brachial plexus block and found that the duration of sensory and motor blockade was significantly prolonged by adding dexmedetomidine. The duration of analgesia was also prolonged in the dexmedetomidine group (BD) with improved quality of block, sedation and hemodynamic stability, without any adverse effects.

In our study, the onset time of sensory blockade (BD 7.07 ± 4.5 mins vs BS 7.33±3.21 mins, P=0.79) and motor blockade (14.87±5.38 mins vs. BS 16.30±3.10 min, P=0.211) was comparable in both the groups. Ammar S et al, Esmaoglu etal and Gandhi et al, in their randomized controlled study found that onset time of sensory and motor block were significantly lower in dexmedetomidine group, whereas in our study there was no significant difference in onset of sensory and motor blockade between the two groups. This variation in above studies might be due to the difference in doses of dexmedetomidine and local anaesthetics used, and grading of motor and sensory blockade. In a study done by Bhaskar Babu B.D. et al, with dexmedetomidine as an adjuvant to bupivacaine and lignocaine, the onset time of sensory and motor blockade was comparable in both the groups, which is similar to our study.

In our study, the duration of sensory and motor blockade was prolonged in BD (bupivacaine + lignocaine + dexmedetomidine) group when compared to BS (bupivacaine + lignocaine + normal saline) group (p<0.05) (Table 2). Duration of sensory blockade was prolonged by 232.66 minutes and motor blockade by 168.7 minutes in the...
dexmedetomidine group. Esmaoglu et al., Ammar et al., Gandhi R et al, also found in their studies that administering dexmedetomidine as an adjuvant to bupivacaine in brachial plexus block resulted in a prolongation of sensory and motor block duration which is consistent with our results. The mechanism by which alpha-2 adrenergic receptor agonists produce analgesia and sedation is not fully understood but is likely to be multifactorial.

In our study, Duration of analgesia was prolonged in group (BD) when compared to group BS. Esmaoglu et al., Gandhi et al. and Ammar et al., in their studies also found that there was increase in time to first analgesic request, when Dexmedetomidine was used as an adjunct to local anesthetics in various blocks when compared with local anesthetics alone.

In our study, we found that heart rate was decreased from base line in the BD (bupivacaine + dexmedetomidine) group after 15 minutes (p<0.05) when compared to (BS) group (graph 4). Systolic blood pressure was decreased from baseline in BD i.e. dexmedetomidine group from 30 minutes onwards in comparison to BS group (graph 5). There was significant reduction in Diastolic blood pressure also from 15 minutes (p<0.05) onwards compared to baseline in group BD i.e. dexmedetomidine group when compared to BS group (graph 6). Gandhi R et al, showed no intraoperative hemodynamic variability between the two groups. However, in Esmaoglu et al. study, there was significant reduction in systolic blood pressure from 15 mins, diastolic pressure after 60 mins. and heart rate after 10 mins in dexmedetomidine group, which is similar to our study. The difference in hemodynamics was noted probably due to the difference in the dose of dexmedetomidine used. (30 mcg in study by Gandhi R et al, 100 mcg in study by Esmaoglu et al, and 50 mcg in our study).

Bradycardia was observed in 7 out of 30 patients in Esmaoglu et al study, in only 2 out of 35 patients in Gandhi R et al study and in 2 out of 30 patients in our study. This was probably due to the higher dosage of dexmedetomidine (100 mcg) used in Esmaoglu etal study. In both Gandhi Retal and Esmaoglu et al, there were no incidences of hypotension requiring vasopressors or any other adverse side effects, which was comparable to our study. Ozalp et al12 also noted similar results in their study. No significant side effects were reported in both the groups in our study and also in the study by Swami etal,13 esmaoglu et al.

Sedation was assessed using Ramsay sedation score in our study. There was no statistical significance in both groups until first 15 minutes after which patients in (BD) group showed a score of 3 most of the times, with statistical significance of P<0.05. Ozalp et al. and Esmaoglu et al. also observed that patients who received dexmedetomidine as an adjuvant in brachial plexus block were sedated throughout the surgery.

Centrally-acting alpha-2 agonists produce analgesia and sedation by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neuron and by activation of alpha-2 adreceptors in the locus coeruleus.2

Limitations of Our Study
1. We did not use ultrasound guided block.
2. The dose of dexmedetomidine for peripheral nerve blocks needs to be optimized.

CONCLUSION
Dexmedetomidine is a good adjuvant to local anaesthetic agents, as its addition to bupivacaine and lignocaine was associated with prolonged sensory and motor blockade, mild sedation and prolonged analgesia with better hemodynamic stability. Increased quality of block with hemodynamic stability, along with mild sedation and lack of significant side effects such as respiratory depression makes dexmedetomidine an attractive choice as an adjuvant for supraclavicular brachial plexus block.

REFERENCES
