Comparison of Low Dose Dexmedetomidine and Clonidine as Adjuvants to Bupivacaine in Ultrasound Guided Supraclavicular Brachial Plexus Block

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Abstract

Background: Postoperative analgesia is a sine qua non in current clinical practice. So we are in need for an adjuvant that can prolong the action of local anaesthetics after single injection blocks. Dexmedetomidine and clonidine are two commonly used adjuvants. This study was undertaken to assess which among them proved to be a superior analgesic adjuvant in lower doses.

Materials and methods: After ethical committee approval study was conducted on 60 patients, aged 18-55 years, posted for upper limb surgery under USG guided supraclavicular brachial plexus block at St.John’s Hospital, Bangalore. The study was conducted from January 2015-January 2016- Prospective randomised double blind study. Preoperative baseline values of heart rate, blood pressure and oxygen saturation was recorded. Brachial plexus block by supraclavicular approach was carried out under USG guidance using strict aseptic precautions. Patients were assigned randomly to one of the two groups using computer generated tables:-

Group C - Clonidine 0.5 μg /kg added to 25ml of 0.375% bupivacaine.

Group D - Dexmedetomidine 0.5 μg /kg and 25ml of 0.375% bupivacaine.

The onset of sensory block and motor block, the duration of analgesia, duration of motor block and sedation scores were assessed. Complications were also noted.

Results: In our study, the mean onset of sensory block was 11.6 +/-3.4 minutes in group C, 14.4 +/-4.5 minutes in group D. The mean onset of motor block was 17.6 +/-4.9 in group C, 20.6 +/-5.9 in group D. The duration of sensory block in Group C was 9.7 +/-1.6 hours, 13.3 +/-1.9 hours in Group D. Duration of motor blockade (hrs) was 9.1 +/-1.7 in Group C, 12.1 +/-2.0 in Group D. Duration of sensory and motor blockade was longer in Dexmedetomidine group than Clonidine group. First rescue was required at 10.5 ± 1.7 hrs in Clonidine group and at 15 ± 2.2 hrs in Dexmedetomidine group. None of the subjects in Clonidine group had side effects; whereas 10% of subjects in Dexmedetomidine group had side effects.

Conclusions: Dexmedetomidine proves to be better adjuvant compared to clonidine as it notably prolongs analgesia and is also lesser complications at lower doses.

ABBREVIATIONS

α: Alpha; ANOVA: Analysis of Variance; ASA: American Society of Anaesthesiologist; β: Beta; bpm: Beats per minute; cc: Cubic centimetre; cm: Centimetre; CNS: Central Nervous System; DBP: Diastolic Blood Pressure; ECG: Electrocardiogram; FDA: Food and Drugs Administration; g: Grams; HR: Heart Rate; hrs: Hours; IU: International Units; IV: Intravenous; kg: kilograms; L: Litre; MAP: Mean Arterial Pressure; μg/mcg: microgram; mg: milligram; min: minutes; ml: millilitre; mm: millimetre; mm Hg: millimetre of mercury; pKa: Acid Dissociation Constant; SBP: Systolic Blood Pressure; SPSS: Statistical Package for the Social Science; SpO2: - Oxygen Saturation by Pulse Oximetry; USG: Ultrasonography; Vmax: Maximum Velocity

INTRODUCTION

Brachial plexus blocks provide alternative for general anaesthesia for upper limb surgeries and provide ideal operative conditions. Various drugs have been used as adjuvants to modify the block in terms of onset, quality, and duration and post operative analgesia. Bupivacaine is the most frequently used local anaesthetic [1-10] due to its long duration of action (4-8 hours). Adjuvants of recent interest include alpha 2 agonists- like clonidine, dexmedetomidine.

Clonidine, and α2-adrenergic agonist, has been used as an adjuvant to local anaesthetics in regional anaesthesia [11-20]. It is demonstrated that adding clonidine to intermediate and long-acting local anaesthetics during a single-shot peripheral nerve or
nerve plexus block provides a longer duration of analgesia and motor blockade by approximately 2 hours [21-30].

Dexmedetomidine is a dextro-enantiomer and active component of medetomidine [31-40] approved as intravenous sedative and coanalgesic drug. Its alpha2/alpha1 selectivity ratio is 8 times than that of clonidine [41].

Studies comparing clonidine and dexmedetomidine as an adjuvant to bupivacaine are reported in literature for blind technique of supravacular brachial plexus block. High dose of alpha-2 agonists is associated with side effects like hypotension and bradycardia. Very few studies have so far compared low dose of clonidine and dexmedetomidine.

Among the different techniques of supravacular brachial plexus block, the classical approach using paraesthesia being a blind technique is associated with 2 higher failure rates, injury to nerves and vascular structures. The requirement of higher volume and concentration of local anaesthetics as well as adjuvants decreases the safety margin in paraesthesia technique. Blocks using peripheral nerve stimulator also can cause injury to nerves and vascular structures [42]. Recently, the use of USG guidance for exact localization of nerve plexus has revolutionized the technique of regional anaesthesia [43]. It has improved the success rate as well as safety along with marked reduction of the dose of local anaesthetics [44]. Hence we decided to compare the efficacy of low dose adjuvants like clonidine and dexmedetomidine in USG guided supravacular block.

**MATERIALS AND METHODS**

**Source of data**

After ethical committee approval and written informed consent this study was conducted on 60 patients, aged 18-55 years, posted for upper limb surgery under USG guided supravacular brachial plexus block at St. John’s Hospital, Bangalore.

The study was conducted from January 2015-January 2016.

**Type of study:** Prospective Randomized double blind study.

**Inclusion criteria**

1. Adults aged 18-55 years, undergoing elective & emergency upper limb surgical procedures under supravacular brachial plexus block
2. Body mass index -17-35kg/m²
3. Adults belonging to either sex of ASA physical status Grade I & II

**Exclusion criteria**

1. Patient refusal
2. Patients with pre existing neurological disorders (peripheral neuropathy or motor weakness).
3. Known history of hypersensitivity to drugs used.
4. History of significant cardiac, respiratory, renal, hepatic or central nervous system diseases.

5. Inadequate block
6. History of coagulopathy or anticoagulant medication intake

**Method of collection of data**

- All the patients were kept nil per oral for 8 hours, prior to surgery and premedicated with tablet alprazolam 0.25 mg, tablet ranitidine 150 mg on previous night.
- Investigations like hemoglobin, Total Count, Differential Count, blood sugar, urine routine were noted also chest X ray, ECG if above 40 years were noted.
- Patients were assigned randomly according to a computer generated tables to one of the two groups:-
  - **Group C:** Clonidine 0.5 μg/kg added to 25ml of 0.375% bupivacaine.
  - **Group D:** Dexmedetomidine 0.5 μg/kg and 25ml of 0.375% bupivacaine.

The Anaesthesiologist performing the block as well as the one assessing the patient intra- and post-operatively were blinded to the treatment groups

- After shifting the patients to O.T, intravenous access was secured on opposite limb and crystalloid infusion was started.
- Preoperative baseline values of heart rate, blood pressure and oxygen saturation was recorded. Brachial plexus block by supravacular approach was carried out under USG guidance using strict aseptic precautions.
- The block was achieved with 25ml of 0.375% Bupivacaine combined with clonidine or dexmedetomidine.

**Assessment of sensory block** was done by pin prick method every minute in the dermatomes corresponding to median nerve, radial nerve, ulnar nerve and musculocutaneous nerve. Dull sensation to pin prick marked the onset of sensory block

- Sensory block was graded as-
  - Grade 0: Sharp pin felt
  - Grade 1: Analgesia, dull sensation felt
  - Grade 2: Anaesthesia, no sensation felt.

**Assessment of motor block** was carried every minute till complete motor blockade.

- Motor block was determined according to a modified Bromage scale for upper extremities on a 3-point scale.
  - **Grade 0:** Normal motor function with full flexion and extension of elbow, wrist and fingers
  - **Grade 1:** Decreased motor strength with ability to move the fingers only
  - **Grade 2:** Complete motor block with inability to move the fingers

The block was considered incomplete when any of the segments supplied by median, radial, ulnar and musculocutaneous nerve did not have analgesia even after 30 min of drug injection.
Patients with incomplete block were supplemented with general anaesthesia and they were excluded from the study.

Haemodynamic variables such as heart rate, BP and oxygen saturation recorded every 5 min intraoperatively and every 60 min post-operatively.

**Duration** of sensory block (till appearance of pain requiring analgesia) and duration of motor block (till complete return of the muscle power) was recorded.

**Assessment of Sedation** was done by the Ramsay Sedation Score-

- **IF AWAKE:**
  - RAMSAY1: Anxious, agitated, restless
  - RAMSAY2: co-operative, oriented, tranquil
  - RAMSAY3: responsive to commands only

- **IF ASLEEP:**
  - RAMSAY 4: Brisk response to light glabellar tap or loud auditory stimulus
  - RAMSAY 5: Sluggish response to light glabellar tap or loud auditory stimulus
  - RAMSAY6: no response to light glabellar tap or loud auditory stimulus

**Assessment of Quality** of operative conditions was done according to the following numeric scale:

- Grade 4: (Excellent) No complaint from patient
- Grade 3: (Good) Minor discomfort with no need for the supplemental analgesics
- Grade 2: (Moderate): Pain that required supplemental analgesia
- Grade 1: (Unsuccessful) Patient given general anaesthesia

- **Duration of surgery** was noted.
- **The intra- and post-operative assessment** was done.
- **The rescue analgesia** was given with injection dicyclofenac sodium (1.5 mg/kg intramuscularly)
  - Side-effects like bradycardia, nausea, vomiting, dryness of mouth, hypotension and complications like pneumothorax, haematoma, local anaesthetic toxicity and post-block neuropathy in the intra- and post-operative periods was noted.

**Data collection tools:** Data was collected using the attached proforma

**RESULTS**

Mean age of subjects in Clonidine group was 31.1 ± 11.3 years and in Dexmedetomidine group was 31.5 ± 10.2 years. There was no significant difference in age distribution between two groups.

In the study 83.3% were male and 16.7% were female in both the groups. There was no significant difference in gender distribution of subjects between two groups.

Mean weight of subjects in Clonidine group was 62.5 ± 6.9 kgs and in Dexmedetomidine group was 64.9 ± 6.7 kgs. There was no significant difference in mean weight between two groups.

Both the groups were comparable in terms of age, sex and weight.

In Clonidine group 76.7% of subjects had ASA I, 16.7% had ASA IE and 6.7% had ASA II. In Dexmedetomidine group 96.7% had ASA I and 3.3% had ASA II.

Onset of sensory (11.6 ± 3.4), motor blockade (17.6 ± 4.9) was significantly faster in Clonidine group than Dexmedetomidine group.

Duration of sensory (13.3 ± 1.9) and motor blockade (12.1 ± 2.0) was longer in Dexmedetomidine group than Clonidine group. These observations were statistically significant.

First rescue was required at 10.5 ± 1.7 hrs in Clonidine group and at 15 ± 2.2 hrs in Dexmedetomidine group. This observation was statistically significant.

Significant difference in Sedation score was observed at 0 min and at 60 min. higher sedation score was observed in Clonidine group than Dexmedetomidine group. At other intervals there was no significant difference between two groups.

**DISCUSSION**

Brachial plexus blocks are effective in providing both adequate intraoperative conditions as well as postoperative analgesia in upper limb surgeries. Plain bupivacaine can hardly provide analgesia beyond 3-8 hours following a peripheral nerve block [45,46] and hence prolongation of its action may be attempted through placement of indwelling catheters to provide continuous infusion of local anaesthetic or by addition of analgesic adjuvants. Indwelling catheter techniques are very effective but their use is limited because of difficulties in placement, inherent secondary failure rate, difficulties with catheter removal or sometimes infection [47-50]. Various adjuvants including midazolam, neostigmine, and dexamethasone in conjunction to local anaesthetics have been tried to prolong analgesia with varying degree of success.

Alpha 2 adrenergic agonists have been utilized for more than 100 yrs. Various routes of administration such as epidural, intrathecal and peripheral injections have been tried with local anaesthetics to prolong and improve the quality of anaesthesia. Clonidine and Dexmedetomidine are partial and selective alpha 2 agonist respectively.

Recent introduction of Ultrasound guidance has established its effectiveness and safety and revolutionised the practice of peripheral nerve blocks. It has improved the safety along with marked reduction in the dose of local anaesthetics and adjuvants [51-60]. USG helped us in visualizing the nerve roots and depositing the drug at the plexus.

The commonly missed area, above the first rib and inferomedial to the plexus and posterolateral to the subclavian artery called the ‘-corner pocket’, could be visualized with Ultrasound and spread of the drug to this area and around the plexus could be confirmed.
In the previous studies, higher dose of adjuvants to local anaesthetics were used. We chose 0.5 μg/kg of Dexmedetomidine and Clonidine in order to assess the efficacy of lower dose adjuvants with ultrasound guidance and also to reduce the side effects if any.

There are very few studies comparing the efficacies of low dose of α2 agonists as an adjuvant to 0.375% Bupivacaine using USG in Supraclavicular brachial plexus block. Hence the present study was undertaken among sixty patients aged 18-55 years to find the superior adjuvant among 0.5 μg/kg of clonidine and 0.5 μg/kg of Dexmedetomidine that can be combined with 25 ml of 0.375% bupivacaine under USG guidance. These two groups were compared in terms of onset, duration of sensory block, duration of motor block and adverse effects.

Patients fulfilling the inclusion criteria were grouped into either Group D or Group C according to the computer generated randomized tables. We chose 0.375% bupivacaine as our pilot study performed with 0.25% bupivacaine failed to provide complete motor block which was essential for the long surgical procedures. Sedative premedication was not administered in order to assess sedation scores. Both groups were comparable with respect to demographic data- age, gender and weight distribution and ASA physical status grade distribution. In our study we found that the mean onset of sensory block was 11.6 +/-3.4 minutes in group C and 14.4 +/-4.5 minutes in group D. The mean onset of motor block was 17.6 +/-4.9 in group C and 20.6 +/-5.9 in group D. This difference was statistically significant. The duration of sensory block was 9.7 +/-1.6 hours in Group C and 13.3 +/-1.9 in Group D. The duration of motor block 9.1 +/-1.7 hours in Group C and 12.1 +/-2 in Group D. The first rescue analgesic was required at 10.5 +/-1.7 hours in Group C and 14.4 +/-4.5 minutes in Group D. All these observations were statistically significant. 

Comparison of onset of sensory and motor blockade

Swami et al. [61-65], in 2012 conducted a randomized double blinded study in 60 ASA I and II patients who received supraclavicular brachial plexus block to compare clonidine 1mcg/kg and Dexmedetomidine 1mcg/kg added to 0.25% Bupivacaine using nerve stimulator. There was no statistically significant difference in onset of sensory and motor block between the two groups in their study. However in our study we found that the mean onset of sensory block was 11.6 +/-3.4 minutes in group C, 14.4 +/-4.5 minutes in group D. The mean onset of motor block was 17.6 +/-4.9 in group C and 20.6 +/-5.9 in group D. There was faster onset for motor and sensory block in Clonidine group. This difference was statistically significant.

- Jinjil et al. [66-70], 2015 evaluated Dexmedetomidine 1μg/kg and Clonidine 1μg/kg as adjuvant to 0.25% Ropivacaine in USG guided supraclavicular block. They found that onset of sensory block was in Dexmedetomidine group was 9.7 +/-1.5 minutes, in Clonidine group it was 12.9 +/-1.4 minutes. The onset of motor block in Dexmedetomidine group was 15.7 +/-1.5 and in Clonidine group it was 20.4 +/-1.8 minutes. There was a statistically significant faster onset for sensory and motor blockade in Dexmedetomidine group.

- In 2014, Rao et al. [71-74], conducted a randomised double blind prospective study to compare clonidine and Dexmedetomidine as an adjuvant in supraclavicular brachial plexus block where they noted that dexmedetomidine group had faster onset.

- In most of the studies Dexmedetomidine showed faster onset which was not comparable to our study. According to Haramritpal et al. [73], Khade Amit [75] (2013) the onset of sensory and motor blockade were found to be faster in dexmedetomidine group than plain local anaesthetic group.

In contrary to the above mentioned studies, Rachana Gandhi et al. [67], in 2012, the Control group-C received injection bupivacaine (0.25%) 38 ml plus 2 ml normal saline, dexmedetomidine group-D received injection bupivacaine (0.25%) 38 ml plus Dexmedetomidine 30 μg (2 ml) observed that in control group onset of motor and sensory blockade was faster. This was not comparable to other studies which showed slower onset of motor and sensory block with Dexmedetomidine.

- Some studies showed that addition of Clonidine shortens the onset of block and improved the quality of analgesia like Singh et al. [59], in 2010 compared the effects of clonidine added to bupivacaine alone in supraclavicular brachial plexus block in a prospective, randomized, double blinded controlled trial. Two groups of 25 patients each were investigated using 40ml of 0.25% bupivacaine plus 0.150 mg of clonidine and 40ml of 0.25% bupivacaine plus 1 ml NaCl 0.9%. It was observed that addition of clonidine to bupivacaine resulted in faster onset of sensory block.

Kohli et al. [62], randomly allocated 60 adult patients undergoing upper limb surgeries under supraclavicular block into 2 groups. Thirty patients received 1μg/kg clonidine and the rest received 2 μg/kg clonidine added to 30ml of 0.5% bupivacaine. The onset of sensorimotor block was faster in the higher dose group.

Some studies failed to find any advantage of addition of Clonidine like- Singelyn et al. [52], Murphy et al. [54], Hutschala et al. [55], BirbalBaj et al. [64], Kumkum Gupta et al. [65], who observed no hastening of onset of block with clonidine added to local anaesthetic, irrespective of the dosage of clonidine used.

Studies may show different results for the onset of the block comparing Dexmedetomidine and clonidine which may be due to varying outcome measures used. It also can be due to a variation of the pharmacokinetic effects resulting from using different concentration and volume of drugs or may be due to different block approaches like USG guided blocks, blind approach or nerve stimulator guided [76-84].

Duration of analgesia and motor block

In our study, duration of sensory block was recorded till appearance of pain requiring analgesia and duration of motor block was recorded till complete return of the muscle power. This was in comparison with studies performed by Swami et al. [61], Esmaoglu et al. [85], Gandhi R et al. [67], and Biswas et al. [70].

In Swami et al. [61], the duration of sensory block and motor block in clonidine group was 227 +/- 48.36 and 292.67 +/- 59.13 minutes respectively while it was 413.97 +/- 87.13 and 472.24 +/- 90.06 minutes respectively in dexmedetomidine group. They
Table 1: Age distribution of subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Clonidine</th>
<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Age</td>
<td>31.1</td>
<td>11.3</td>
<td>31.5</td>
</tr>
</tbody>
</table>

Table 2: Gender distribution of subjects.

<table>
<thead>
<tr>
<th>Group</th>
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<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Number (n)</td>
<td>%</td>
<td>Number (n)</td>
</tr>
<tr>
<td>Male</td>
<td>25</td>
<td>83.3%</td>
<td>25</td>
</tr>
<tr>
<td>Female</td>
<td>5</td>
<td>16.7%</td>
<td>5</td>
</tr>
</tbody>
</table>

χ² = 0.00, df = 1, p = 1.000

Table 3: Weight comparison between two groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Clonidine</th>
<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Weight</td>
<td>62.5</td>
<td>6.9</td>
<td>64.9</td>
</tr>
</tbody>
</table>

Table 4: Demographic profile.

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>Group C</th>
<th>Group D</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (years)</td>
<td>Mean +/-11.3</td>
<td>31.5 +/-10.2</td>
<td>0.905 NS</td>
</tr>
<tr>
<td>SEX ratio (male/female)</td>
<td>25:5</td>
<td>25:5</td>
<td>1.00 NS</td>
</tr>
<tr>
<td>WEIGHT (Kg)</td>
<td>Mean +/-6.9</td>
<td>64.9 +/-6.7</td>
<td>0.168 NS</td>
</tr>
</tbody>
</table>

NS: Not significant; S: Significant

Table 5: ASA Grade between two groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Clonidine</th>
<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA</td>
<td>Number</td>
<td>%</td>
<td>Number</td>
</tr>
<tr>
<td>I</td>
<td>28</td>
<td>93.3%</td>
<td>29</td>
</tr>
<tr>
<td>II</td>
<td>2</td>
<td>6.7%</td>
<td>1</td>
</tr>
</tbody>
</table>

χ² = 0.3509, df = 1, p = 0.5536

Table 6: Onset, duration of anaesthesia and First rescue between two groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Clonidine</th>
<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of sensory (min)</td>
<td>11.6 ± 3.4</td>
<td>14.4 ± 4.5</td>
<td>0.008*</td>
</tr>
<tr>
<td>Onset of motor (min)</td>
<td>17.6 ± 4.9</td>
<td>20.6 ± 5.9</td>
<td>0.036*</td>
</tr>
<tr>
<td>Duration of sensory (hrs)</td>
<td>9.7 ± 1.6</td>
<td>13.3 ± 1.9</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Duration of motor (hrs)</td>
<td>9.1 ± 1.7</td>
<td>12.1 ± 2.0</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>First rescue (hrs)</td>
<td>10.5 ± 1.7</td>
<td>15.0 ± 2.2</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

concluded that dexmedetomidine enhanced the duration of sensory and motor block and also the duration of analgesia. The duration of sensory and motor block was almost twice compared to clonidine. It also enhanced the quality of block as compared with Clonidine. Jinjil et al. [66], 2015 comparing Dexmedetomidine and Clonidine 1μg/kg each as adjuvants to 0.25% Bupivacaine in USG guided Supraclavicular brachial plexus block showed total duration of sensory block was 690 minutes and 330 minutes in Dexmedetomidine group and Clonidine group respectively.

A RCT conducted by Esmagouli et al. [85], study assigned 60 patients to receive either 40 ml of 0.5% levobupivacaine with 1ml dexmedetomidine (100μg) or 40ml of 0.5% levobupivacaine with 1ml of saline and found that sensory and motor onset time were significantly faster in study group compared to control group. Duration of sensory block (minutes) in Group L: 673.00 +/- 73.77, in Group LD: 887 +/- 66.23. Duration of motor block (minutes) in Group L: 575.00 +/- 65.00, in Group LD: 773.00 +/- 67.62.

Gandhi R et al. [67], in a study assigned 70 patients to either
These were in agreement with our study. Opioid requirements were also less in the dexmedetomidine group. It also showed that verbal rating scales for pain, postoperative sensory and motor block showed prolongation of duration of sensory and motor block by using dexmedetomidine in ultrasound guided infraclavicular brachial plexus block via supraclavicular route with the help of nerve stimulator. In group L (n=30) 35cc of levobupivacaine with 1ml of isotonic saline and in group LD (n=30) 35cc of levobupivacaine with 1ml of (100 microgram) of dexmedetomidine was given. They noted that durations of sensory and motor block were longer in group LD as compared to L (P<0.01).

Duration of motor block (minutes) Group L: 512± 60.13 and Group LD: 840±50.23. Duration of sensory block (minutes) Group L: 645± 70.11 and Group LD: 898±32.33.

Few recent studies use USG to perform blocks whose results were also comparable to our study- Agarwal S et al. [86], studied on Dexmedetomidine as an adjuvant to bupivacaine for brachial plexus block through supraclavicular approach showed that duration of analgesia was prolonged in dexmedetomidine group. The mean duration time for sensory and motor blocks for study group were 755.6 ± 126.8 and 702.0 ± 112.6 min, respectively; but for the control group, the mean duration were 234.8 ± 47.9 but 17% incidence of sedation with higher dose. Although sedation might be undesirable in certain situations like in high risk patients, mild sedation was desirable during that period as calm patient is ‘ideal’ for any regional technique.

As clonidine is a lipophilic drug, much of it gets absorbed systemically after perineural administration resulting in sedation [52]. α2 - adrenergic agonists produce sedation by central action by inhibition of substance P release in the nociceptive pathway at the level of dorsal root neuron and by activation of α2 – adrenergic receptors in locus ceruleus [70].

All these studies were comparable to our study which showed the duration of sensory block in Group C was 9.7+/-.16 hours, 13.3+/-.1.9 hours in Group D. Duration of motor blockade (hrs) was 9.1+/-.1.7 hours in Group C, 12.1+/-.2.0 hours in Group D. Duration of sensory and motor blockade was longer in Dexmedetomidine group than Clonidine group. First rescue was required at 10.5 ± 1.7 hrs in Clonidine group and at 15 ± 2.2 hrs in Dexmedetomidine group. All these observations were statistically significant.

38ml of 0.25% bupivacaine with 30 μg of dexmedetomidine or plain 38ml of 0.25% bupivacaine only. They showed that duration of sensory and motor blockade was longer by using 30 μg of dexmedetomidine (660.2 ± 60.4 and 732 ± 48.9) compared to only 0.25% bupivacaine control group (146.5 ± 36.4, 100.7 ± 48.3). We also noted similar results with prolonged duration of both sensory and motor blockade by using dexmedetomidine with Bupivacaine.

<table>
<thead>
<tr>
<th>Sedation Score</th>
<th>Clonidine</th>
<th>Dexmedetomidine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Median</td>
<td>SD</td>
</tr>
<tr>
<td>0 min</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
</tr>
<tr>
<td>5 min</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
</tr>
<tr>
<td>15 min</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
</tr>
<tr>
<td>30 min</td>
<td>2.4</td>
<td>2.0</td>
<td>0.7</td>
</tr>
<tr>
<td>60min</td>
<td>3.3</td>
<td>3.0</td>
<td>0.8</td>
</tr>
<tr>
<td>2 hours</td>
<td>2.7</td>
<td>2.0</td>
<td>1.1</td>
</tr>
<tr>
<td>6 hours</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
</tr>
<tr>
<td>12 hours</td>
<td>2.0</td>
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</tr>
<tr>
<td>24 hours</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Comparison of Sedation Scores between two groups.

AmmarS et al. [87], in their RCT on dexmedetomidine with bupivacaine in ultrasound guided infradavicular brachial plexus block showed prolongation of duration of sensory and motor blockade and analgesia which was comparable to our study. They also showed that verbal rating scales for pain, postoperative opioid requirements were also less in dexmedetomidine group. These were in agreement with our study.

One of the strengths in our study was that we assessed sedation scores based on Ramsay scoring. No sedative premedication on the day of surgery were given to avoid interference in scoring sedation. Significant difference in Sedation score was observed at 0 min and at 60 min. Highest sedation score was observed in Clonidine group at 60 minutes. None of the patient experienced airway compromise or required airway assistance. At other intervals there was no significant difference between two groups. Although sedation might be undesirable in certain situations like in high risk patients, mild sedation was desirable during that period as calm patient is ‘ideal’ for any regional technique.

Popping et al. [57], in 2009 did a meta analysis of 20 studies (1,054 patients, 573 received clonidine) and concluded that clonidine hastened the duration of analgesia and motor block by about 2 hours when added to intermediate or long acting local anaesthetics.

**Sedation scores**

One of the strengths in our study was that we assessed sedation scores based on Ramsay scoring. No sedative premedication on the day of surgery were given to avoid interference in scoring sedation. Significant difference in Sedation score was observed at 0 min and at 60 min. Highest sedation score was observed in Clonidine group at 60 minutes. None of the patient experienced airway compromise or required airway assistance. At other intervals there was no significant difference between two groups. Although sedation might be undesirable in certain situations like in high risk patients, mild sedation was desirable during that period as calm patient is ‘ideal’ for any regional technique.

As clonidine is a lipophilic drug, much of it gets absorbed systemically after perineural administration resulting in sedation [52]. α2 - adrenergic agonists produce sedation by central action by inhibition of substance P release in the nociceptive pathway at the level of dorsal root neuron and by activation of α2 – adrenergic receptors in locus ceruleus [70].

Kohli et al. [62], compared two different doses of clonidine -1 μg/kg and 2 μg/kg with local anaesthetic and concluded that there was no hemodynamic alteration with lower dose clonidine but 17% incidence of sedation with higher dose.

The quality of sedation produced by α2 - agonists differs from the sedation produced by drugs like midazolam, propofol that act on gamma aminobutyric acid (GABA) receptors. Sedation produced by α2 - agonists reflects decreased sympathetic activity.
nervous system activity, resulting in a calm patient who can be easily aroused to full consciousness.

We have done this study in ASA physical status 1 and 2 patients, we need to be extremely vigilant when we supplement opioids or benzodiazepines along with alpha 2 agonists for sedation in patients who are prone for respiratory compromise.

**Side effects**

Most of the studies showed reversible bradycardia <10% which is comparable to our study [57].

Side effects like bradycardia observed in 7 out of 30 patients in Esmaoglu et al. [85], study; 2 out of 35 patients in Gandhi R et al. [67], study and where as in our study none of the subjects in Clonidine group had bradycardia, were as 3(10%) of subjects in Dexmedetomidine group had Bradycardia, this probably due to the dosage of dexmedetomidine used which is 100 μg in Esmaoglu et al. [85], study and 30 μg in study by Gandhi R et al, [67].

In 1998, Singelyn et al. [52], observed that a minimum of 0.5 mcg/kg clonidine needed to be given perineurally to prolong analgesia after brachial plexus block without production of any adverse effects. McCartney et al. [56], took up 27 studies (1,385 patients) and concluded that clonidine proved to be a beneficial adjuvant when added to intermediate-acting local anaesthetics and side effects were limited to doses up to 150 mcg [86-88].
There were no complications due to supravcavicular block like pneumothorax, injury to vascular structures, horner's syndrome as the technique was finer with ultrasound guidance. The commonly missed areas are well visualized with Ultrasound and spread of the drug to this area and around the plexus was confirmed. Thus the use of Ultrasound helped us to minimize the dose requirement of local anaesthetics in combination with adjuvants.

**CONCLUSION**

Dexmedetomidine proves to be better adjuvant compared to clonidine as it notably prolongs analgesia and also has fewer complications at lower doses. But the prolonged motor block is still a matter of concern and the search for an adjuvant that selectively prolongs analgesia without impairing motor function continues.

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