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# ORIGINAL RESEARCH

## To assess the efficacy of bupivacaine against levobupivacaine in supraclavicular brachial plexus block

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**ABSTRACT**
**Aim:** The purpose of this research is to assess the efficacy of bupivacaine against levobupivacaine in supraclavicular brachial plexus block.
**Methods:** The research comprised 100 patients between the ages of 20 and 55 who had upper limb orthopaedic and soft tissue lesions and weighed more than 61 kilos and had ASA grades I and II. The first group received 1 ml (100g) dexmedetomidine mixed with 39 ml of 0.5 percentLevobupivacaine. As an anaesthetic agent, category II received 1 ml of 0.9 percent normal saline and 39 ml of 0.5 percentLevobupivacaine. The heart rate, respiration rate, SBP, DBP, pulse rate, and oxygen saturation were all monitored and recorded as soon as the patient entered the OT. In the previously secured intravenous line, ringer lactate was begun. The supraclavicular technique was used to provide brachial plexus block.
**Results:** The mean age in categories I and II was 34.25 and 35.87, respectively. Both outcomes were statistically significant. All research participants in Category I did not need post-operative analgesia, but all patients in Category II did. In Category I, 28 percent were mildly sleepy, compared to 100 percent in Category I. Both outcomes are statistically significant. In Category I, the average duration of sensory and motor block was about 4.1 minutes less than in Category II. (5.87 vs. 9.98 minutes) The onset of motor blockage in Category I was likewise 4.1 minutes sooner than in Category II (8.88 vs 12.96 min). The mean length of sensory block (577 vs 989 min) and motor block (596 vs 988 min) was 392 minutes shorter in each category. The duration of post-operative analgesia was 404 minutes shorter in both categories (579 vs 979 min). The findings are all statistically significant.
**Conclusion:** We believe that in peripheral nerve blocks requiring high amounts of local anaesthetic, levobupivacaine may be a feasible alternative due to its lower hazardous potential.
**Keywords:** Supraclavicular Brachial Plexus Block, Levobupivacaine, Bupivacaine,

### Introduction

Brachial plexus block is a localised method used for upper limb surgery. Regional blocks for upper limb surgeries have several advantages over general anaesthesia, including pre-emptive analgesia, stable intra-operative hemodynamics, a lower incidence of postoperative nausea and vomiting1, superior post-operative analgesia, less time in the post-anesthesia care unit (PACU), and a shorter hospital stay. Vasospasm and edoema are reduced by the accompanying sympathetic inhibition. Upper limb procedures may be conducted using a variety of regional blocks, including supraclavicular, infraclavicular, interscalene, axillary, and others. Ultrasound, peripheral nerve stimulator, and elicitation of paresthesia are some of the methods used to locate nerves.[1-3]
Levobupivacaine has a three to eight-hour duration of action and is hence the most often utilised local anaesthetic medication. For many years, this anaesthetic medicine has established itself as less expensive and safer. However, spotty or inadequate analgesia and delayed onset are only a few of the practical limitations. In trials, a few medicines are added to levobupivacaine to reduce these limits, improve the quality, and extend the duration of action and analgesia. Vasoconstrictors such as -adrenergic agonists, hyaluronidase, neostigmine, and opioids may be used to extend the duration and enhance the quality of brachial plexus block. However, these vasoconstrictors have been linked to a number of negative side effects. Many researchers experimented with new alpha 2 adrenergic agonists in order to locate the perfect additive. Alpha 2 adrenergic agonists not only reduce the need for intraoperative anaesthetic drugs, but they also have cardiovascular stabilising, sympatholytic

analgesic, and sedative characteristics. These may be administered in peripheral nerve blocks, intrathecal, epidural, or combined with local anaesthetic drugs to minimise the time of onset of nerve block, lengthen the duration of block, and increase the quality of blockade.[4]Dexmedetomidine, a 2 receptor agonist, is eight times more responsive than clonidine.[5,6]Researchers discovered that dexmedetomidine enhanced the onset and duration of motor / sensory blockage in numerous animals and people. It has extended the duration of analgesia when administered as an adjuvant to local anaesthetic drugs in peripheral nerve blocks.[7-14] The purpose of this research was to see how levobupivacaine 0.5 percent alone and with dexmedetomidine 100g as an adjuvant to levobupivacaine 0.5 percent affected the onset and duration of sensory and motor block, the length of perioperative analgesia, complications, and sedation score.

**Methods and materials**

This comparison research was conducted in the department of Anaesthesia after receiving ethical permission from the institution. The research comprised 100 patients between the ages of 20 and 55 who had upper limb orthopaedic and soft tissue lesions and weighed more than 61 kilos and had ASA grades I and II. The research excluded patients with CKD, pregnant women, hypertension, diabetes, cerebrovascular accident, COPD, coronary artery disease on anticoagulants, and those with a history of bleeding problems. All patients had a thorough medical history, physical examination, and fitness evaluation. All patients must have a complete blood count as well as additional tests such as random blood sugar, blood urea, bleeding time, serum creatinine, clotting time, and an ECG. All of the patients were divided into two equal groups. The first group received 1 ml (100g) dexmedetomidine mixed with 39 ml of 0.5 percentLevobupivacaine. As an anaesthetic agent, category II received 1 ml of 0.9 percent normal saline and 39 ml of 0.5 percentLevobupivacaine. The heart rate, respiration rate, SBP, DBP, pulse rate, and oxygen saturation were all monitored and recorded as soon as the patient entered the OT. In the previously secured intravenous line, ringer lactate was begun. The supraclavicular technique was used to provide brachial plexus block.

For neural localization, a nerve finder was employed, which was connected to a 22 G, 50-mm-long stimulating needle. The localization end point was a distal motor response in the median nerve area with an output less than 0.5 milliamperes. Following negative aspiration, a local anaesthetic solution in a marked coded syringe was administered. The sensory block was evaluated using the pin prick technique. Sensory onset was defined as a dull feeling to a pin prick along the distribution of any two of the following nerves: the musculocutaneous nerve, the radial nerve, the median nerve, and the ulnar nerve. Full sensory block occurs when there is complete lack of feeling to a pin prick. Bromage scale has been modified. Motor and sensory blockages were evaluated every 30 minutes for 30 minutes before injection, and then every 30 minutes until they resolved. [15] At 0, 5, 10, 15, 30, 60, 90, and 120 minutes, oxygen saturation (SpO2), heart rate, and diastolic blood pressure (SBP) were measured. Side effects such as HR less than 50% min (bradycardia) and BP less than 20% of resting conditions (hypotension) were addressed with suitable treatments. Then we recorded the duration of motor and sensory blockages after the procedure began. When the subject's visual analogue score was more than 5, a rescue analgesia such as injectable diclofenac sodium 75mg (1.5mg/kg) was given. The Ramsay Sedation Scale (RSS) was utilised to measure sedation before and after the block.

**Results**

Males made up 64% of the Category I population, while females made up 36%. It was 58 percent and 42 percent in Category II, respectively. The two categories had little statistical significance. (See Table 1) The mean age in Categories I and II was 34.25 and 35.87, respectively. Both outcomes were statistically significant. All research participants in Category I did not need post-operative analgesia, but all patients in Category II did. In Category I, 28 percent were mildly sleepy, compared to 100 percent in Category I. Both outcomes are statistically significant. Table.1 In Category I, the average duration of sensory and motor block was about 4.1 minutes less than in Category II. (5.87 vs. 9.98 minutes) The onset of motor blockage in Category I was likewise 4.1 minutes sooner than in Category II (8.88 vs 12.96 min). The mean length of sensory block (577 vs 989 min) and motor block (596 vs 988 min) was 392 minutes shorter in each category. The duration of post-operative analgesia was 404 minutes shorter in both categories (579 vs 979 min). The findings are all statistically significant. From the fifth minute after medication induction until two hours, diastolic blood pressure, systolic blood

pressure, and heart rate remained lower than baseline for Category I. (See Tables 2-4.) There was bradycardia in 26% of Category I patients but none in Category II.

**Table 1: Demographic profile of two categories**

| | | Category I=50 | | Category II=50 | | Significance |
|---|---|---|---|---|---|---|
| **Profile** | | No | % | No | % | |
| Sex | Male | 32 | 64 | 29 | 58 | 0.33 |
| | Female | 18 | 36 | 21 | 42 | |
| Analgesia (Post operative) | Not needed | 50 | 100 | 0 | 0 | <0.001 |
| | Needed | 0 | 0 | 50 | 100 | |

**Table 2: Heart rate of two categories**

| Time in min | Category II | Category I | P value |
|---|---|---|---|
| **Heart rate** | Mean±Sd | Mean±Sd | P-value |
| 0 | 86.74±3.69 | 85.78±6.98 | 0.55 |
| 5 | 84.44±5.85 | 80.85±7.36 | 0.036 |
| 10 | 82.87±4.69 | 77.65±6.35 | 0.007 |
| 15 | 80.52±6.36 | 73.87±4.63 | <0.001 |
| 30 | 77.59±5.88 | 70.99±7.58 | <0.001 |
| 45 | 76.98±4.69 | 68.58±4.69 | <0.001 |
| 60 | 76.32±4.87 | 68.79±7.58 | <0.001 |
| 75 | 79.41±6.39 | 72.23±6.36 | <0.001 |
| 90 | 80.69±5.47 | 72.87±4.63 | <0.001 |
| 120 | 83.12±6.96 | 74.55±5.52 | <0.001 |

**Table 3: Systolic BP of two categories**

| Time in min | Category I | Category II | P-value |
|---|---|---|---|
| **Systolic BP** | Mean±Sd | Mean±Sd | P-value |
| 0 | 132.22±7.36 | 135.02±7.74 | 0.18 |
| 5 | 127.69±5.22 | 125.36±5.23 | 0.11 |
| 10 | 122.41±4.36 | 116.03±4.63 | <0.001 |
| 15 | 122.33±3.33 | 115.11±3.55 | <0.001 |
| 30 | 121.02±5.55 | 114.36±4.19 | <0.001 |
| 45 | 122.03±6.21 | 113.47±5.82 | <0.001 |
| 60 | 122.15±6.87 | 115.66±6.12 | <0.001 |
| 75 | 126.24±7.98 | 115.63±5.11 | <0.001 |
| 90 | 127.54±5.56 | 116.33±6.74 | <0.001 |
| 120 | 129.11±3.32 | 118.02±9.52 | <0.001 |

**Table 4: Diastolic BP of two categories**

| **Diastolic BP** | Category I | Category II | P-value |
|---|---|---|---|
| | Mean±Sd | Mean±Sd | P-value |
| 0 | 82.23±7.77 | 80.42±6.99 | 0.22 |
| 5 | 80.44±6.97 | 77.77±5.85 | 0.15 |
| 10 | 76.63±5.89 | 72.69±3.69 | 0.011 |
| 15 | 74.58±7.52 | 70.75±7.52 | 0.012 |
| 30 | 74.45±6.99 | 67.23±5.55 | <0.001 |
| 45 | 74.25±3.33 | 68.22±4.66 | <0.001 |
| 60 | 74.44±4.65 | 68.24±5.85 | <0.001 |
| 75 | 76.74±5.87 | 68.31±6.39 | <0.001 |
| 90 | 78.46±6.33 | 72.26±8.25 | <0.001 |
| 120 | 80.84±7.15 | 73.45±7.15 | <0.001 |

## Discussion

Because it delivers adequate intraoperative anaesthesia and postoperative analgesia, brachial plexus block is close to the optimal anaesthetic approach for upper limb procedures. The most often used local anaesthetic medication for brachial plexus block is racemic bupivacaine. However, deaths due to cardiovascular (CVS) and central nervous system (CNS) toxic effects have been reported after unintentional intravascular injection of racemic bupivacaine, which has been linked to the dextro (R+) enantiomer. Following that, levobupivacaine, a pure s-enantiomer of bupivacaine, emerged as a safer option with a therapeutic profile comparable to racemic bupivacaine but a superior safety profile. When administered as an adjuvant to levobupivacaine 0.5 percent, dexmedetomidine 100 g delays the onset of sensory and motor blockade and prolongs the analgesic impact of motor and sensory block. The mechanism of action of 2 agonists that cause sedation and analgesia is unknown, however it seems to be complex. Centrally, 2 agonists suppress the release of substance P at dorsal root neurons in the pain pathway while activating 2 adrenoceptors in the locus coeruleus, resulting in analgesia and sedation. Peripherally, 2 agonists inhibit noradrenaline release, cause analgesia, and exert 2 receptor-independent restrictions on nerve action potentials. Dexmedetomidine's peripheral effect is caused by hyperpolarization of cation currents. The neuron will not revert from hyperpolarized to resting membrane state for future firing. [16-19]Several other authors' studies have also shown that adding dexmedetomidine lowers the start time of motor and sensory block and extends the duration of postoperative analgesia. [20-24]The addition of dexmedetomidine enhances the patients' hemodynamic stability. Several more researches done throughout the globe found similar results. [25]Bradycardia was seen in one-fifth of the individuals who received dexmedetomidine. This is consistent with the results of Talke et al. [26]

## Conclusion

We believe that in peripheral nerve blocks requiring high amounts of local anaesthetic, levobupivacaine may be a feasible alternative due to its lower hazardous potential.

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