IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 25, Issue 3 Ser. 4 (March. 2026), PP 42-46
www.iosrjournals.org

The Effect Of Dexmedetomidine As An Adjuvant To Bupivacaine In Supraclavicular Brachial Plexus Block For Upper Limb Surgeries Using Ultrasound Guidance, A Randomized Controlled Trial Study

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Abstract:
Background: *The advantages of using regional anaesthetic methods are widely known. These advantages can be extended by using additives with local anaesthetics. This investigation sought to determine the impact of mixing dexmedetomidine with bupivacaine for supraclavicular block.*
Methods: *Supraclavicular block was given using ultrasound guidance to 60 patients of ASA I & ASA II of either sex undergoing elective upper limb surgeries in this randomized, double-blind study. Group C (n = 30) received 30 mL 0.25% bupivacaine + 1 mL normal saline while Group D (n = 30) received 30 mL 0.25% bupivacaine +1 µg/kg dexmedetomidine (1 mL). Patients were watched for any adverse effects, hemodynamic changes, the onset, duration of sensory and motor block, and total analgesic duration.*
Results: *Compared to group C, group D experienced a faster onset (P<0.001), longer sensory and motor block durations, and longer analgesia (P < 0.0001). In group D, there was a substantial decrease in heart rate (HR) at 30, 60, 90, and 120 minutes compared to the baseline (P < 0.05). Still, not a single patient's heart rate dropped below 50 beats per minute. The Mean Arterial Pressure (MAP) did not change. Group D patients experienced more effective sedation than group C patients (P < 0.05). In both groups, there were no recorded adverse events.*
Conclusion: *Faster onset, prolonged duration of sensory & motor block, prolonged analgesia with hemodynamic stability, and sufficient sedation were the outcomes of using dexmedetomidine as an adjuvant to bupivacaine in supraclavicular block.*
Keywords: *Bupivacaine, Dexmedetomidine, supraclavicular block.*
Date of Submission: 14-03-2026 Date of Acceptance: 24-03-2026

I. Introduction

Upper limb surgeries are typically performed under brachial plexus block. Many substances have been tested as adjuvants to prolong the time that local anaesthetics (LA) offer and post-operative analgesia. The dexmedetomidine is an active D- isomer of medetomidine and is similarly related to clonidine. Dexmedetomidine is a specific Alpha2 agonist with an α2: α1 ratio of 1620:1 and metabolized in two ways via liver glucuronidation and cytochrome P450 [14]. Since dexmedetomidine is an α2 adrenergic drug with sedative, analgesic, sympatholytic, and cardiovascular stabilizing properties, we investigated using it as an adjuvant to bupivacaine. This study compared the safety and effectiveness of dexmedetomidine as a postoperative analgesic for brachial plexus blocking in conjunction with bupivacaine.

II. Methodology

A prospective clinical trial that was double-blind, randomized, was started after obtaining clearance from the hospital ethical committee. 30 patients belonging to ASA Grade I and II, weighing between 40kg and 75kg and belonging to either sex, were chosen to undergo upper limb procedures. Based on the adjuncts that were provided to them, the patients were split into two groups of 30 patients each. Using a sealed envelope technique, a random number table was used to achieve the randomization. An anesthesiologist who was not involved in this study produced the medication solutions. The two sets of study subjects were:
Group C [control group] who received Injection Bupivacaine (0.25%) 30mL + 1mL normal saline.

Group D [study group]: Received injection Bupivacaine (0.25%) 30mL + Dexmedetomidine 1 μg/kg diluted to 1mL

The patients fulfilling the inclusion criteria will be enrolled for the study after obtaining informed consent.

All the patients who are undergoing elective upper limb surgeries will be given with 0.25mg of alprazolam and 40mg of pantaprazole orally at night before the day of surgery & also be advised to be nil per orally from 12am onward on the night before surgery. Blood pressure, oxygen saturation, and baseline heart rate were noted. Ringer's lactate was started in the unaffected arm following the insertion of an 18 G intravenous cannula.

Under the supervision of an experienced anesthesiologist using ultrasound (US) guidance, all patients underwent brachial plexus block via the supraclavicular route. The brachial plexus was located, and the needle was inserted under US supervision. Before administering the medication, 0.5 mL boluses of saline were injected to verify the needle's correct location. 31 mL of the medication solution was administered following a negative aspiration.

An additional anaesthesiologist evaluated the patients for onset and duration of sensory and motor block, the duration of analgesia, and any adverse effects. The treatment groups were concealed from both anaesthesiologists.

Onset of sensory block was evaluated every 3 minutes following drug injection, using the pin prick method and three-point rating system Grade-0 for no block, Grade-1 for sensory blockade with persistence of touch, and Grade-2 for total sensory blockage. Time from injecting of drug to Grade-2 sensory block was known as the "onset of sensory block." Time from the moment study drug was injected to until the sensory block was fully resolved (Grade-0) was the duration of the sensory block. For the assessment of motor blockade, modified Bromage scale[2] was used. On this scale,

Grade 0 denoted the capacity to move the elbow, wrist, and fingers freely.
Grade 1 denoted the inability to extend the arm for two seconds.
Grade 2 denoted the inability to extend the arm or flex the forearm.
Grade 3 denoted the inability to do any of these three actions.

The period of time between a drug injection and a grade 3 motor block was noted as onset of motor block. And the period of time between the injection of study drug and the full restoration of hand and forearm motor function (Grade-0) was taken as the total motor block duration.

Block was considered incomplete when, even after 30 minutes of drug injection, any of the segments supplied by the median, radial, ulnar, and musculocutaneous nerve lacked complete sensory or motor block. Following their exclusion from the research, these individuals would've been given general anesthesia. Only in those situations where it was feasible to mobilize forearm elbow motor blockade was examined; in all other circumstances, finger movement was the only thing evaluated.

Using the Numeric Rating Scale (1-10), postoperative pain was evaluated hourly in the surgical ward and recovery area [3]. A pain score of

0	No pain
1- 3	mild pain
4 - 6	moderate pain
7 - 10	severe pain

Rescue analgesic drug selected was intramuscular Inj. PCT 1g, which was administered for a pain score of 4 and above. The interval between the drug injection and the initial request for analgesia was considered as the duration of analgesia. Every 8^{th} hour after that, Inj. PCT 1g was repeated if required. During the intraoperative and postoperative phases, all patients were monitored for any adverse reactions, including nausea, vomiting, dry mouth, hematoma, and post-block neuropathy. The sample size was determined using the timing of the initial analgesic request as the main variable. Pilot research was carried out, with five patients in each group. Assuming validity of the difference between the time of initial analgesic request and effect size achieved, we computed the number of patients needed in each group to achieve power 0.8 and significance level of 0.05 for the study. SPSS (standard statistical software SPSS) software Inc., version 16.0 for Windows, was used to code, enter, and analyze the data. For quantitative variables, the mean, standard deviation, minimum, and maximum were used to summarize the data; for categorical variables, the relative frequencies (percentages) were used. T-tests were used to analyze normally distributed variables between the two groups. The Mann Whitney test was used to evaluate variables that were not regularly distributed. To evaluate categorical variables, Pearson's Chi-square ($\chi 2$) test was employed. When the anticipated frequency is less than 5, the Fisher exact test was utilized instead. A statistically significant result was defined as $P < 0.05$, and a very significant result as $P < 0.001$.

III. Results:

In terms of demographic characteristics, the patients in the two groups were similar. In both groups, the baseline hemodynamic values were similar. In contrast to group C , group D's heart rate significantly decreased from baseline at 30, 60, 90, 120 minutes (P<0.001), even though none of the patients in group D developed bradycardia. When mean arterial pressures (MAP) were compared between the two groups, there was no statistically significant difference (P > 0.05).

Block Characteristics

Onset of sensory block

According to Table 1, the onset of sensory block occurred in 11.76 ± 3.24 minutes for group C and 9.24 ± 2.8 minutes for group D. There was a statistically significant difference between the two groups regarding the onset of sensory block. P-value is less than 0.01.

Table 1: Onset of sensory block

Parameter	Group C (Mean± S.D)	Group D (Mean± S.D)	P = value
Onset of sensory block (min)	11.76 ± 3.24	9.24 ± 2.8	0.01

Onset of motor block: Table 2 indicates that the start of motor block occurred in 18.53 ± 2.43 minutes for group C and 13.8 ± 1.9 minutes for group D. There was a statistically significant difference between the two groups regarding the onset of motor block. The significance level is less than 0.01.

Table 2: Onset of motor block

Parameter	Group C (Mean± S.D)	Group D (Mean± S.D)	P = value
Onset of motor block (min)	18.53 ± 2.43	13.8 ± 1.9	0.01

Duration of Sensory block: Table 3 demonstrates that the duration of sensory block was 700 ± 43.9 minutes for group D and 210 ± 33.85 minutes for group C. In terms of the duration of the sensory block, there was a statistically significant difference between the two groups. P-value is less than 0.01.

Table 3: Duration of sensory block

Parameter	Group C (Mean± S.D)	Group D (Mean± S.D)	P = value
Duration of sensory block (min)	210 ± 33.85	700 ± 43.9	0.01

Duration of Motor Block: The motor block lasted 194 ± 28.67 minutes in group C and 685 ± 79.46 minutes in group D, according to Table 4, with a statistically significant difference between the two groups. P-value is less than 0.01.

Table 4: Duration of motor block

Parameter	Group C (Mean± S.D)	Group D (Mean± S.D)	P = value
Duration of motor block (min)	194 ± 28.67	685± 79.46	0.01

Duration of Analgesia: Table 5 demonstrates that analgesia in Group C lasted 220 ± 35.88 minutes, while in Group D it lasted 732 ± 88.45 minutes. The difference in time between the two groups was statistically significant, with a P value < 0.01.

Table 5: Duration of analgesia

Parameter	Group C (Mean± S.D)	Group D (Mean± S.D)	P = value
Duration of analgesia (min)	220 ± 35.88	732 ± 88.45	0.01

IV. Discussion

A very selective alpha-2 agonist, dexmedetomidine has sedative, analgesic, sympatholytic, and cardiovascular stabilising properties [4]. It results in a considerable sparing of opioids and a reduction in the need for inhalational drugs during anaesthesia.

[5] Research has indicated that dexmedetomidine prolongs the duration of regional blocks (spinal [6], epidural [7], caudal [8,9], axillary [10], supraclavicular [11] Bier's [12], and greater palatine nerve block [13]) and results in postoperative analgesia.

Because dexmedetomidine selectively acts on alpha-2 adrenoreceptors (α2:α1=1620:1), it causes sedation and analgesia without causing unintended vascular effects by activating alpha1-receptors. Furthermore, there is a medication called Atipamezole that can reverse the sedative effects of dexmedetomidine. Because of these characteristics, dexmedetomidine is a good analgesic and sedative during the perioperative phase. In our

investigation, a dose of 1 μg/kg of dexmedetomidine was used for supraclavicular block.

Prior research likewise employed a comparable dosage. [10,11] Using ultrasonography allowed us to monitor medication injections around the plexus in real time. Nevertheless, the amount of bupivacaine utilized was reduced to 30ml in our study because by using ultrasound guidance effective nerve blockage can be achieved [15] Patients in group D showed a significant decrease in HR from baseline. None of the patients, however, experienced hypotension or bradycardia. The alpha- 2 agonists' sympatholytic action caused the HR to decrease [10]. A few studies that utilised the same dose of dexmedetomidine as ours [10,16,17] reported cases of bradycardia that needed to be treated, whereas others [11] found no evidence of bradycardia.

Compared to the control group, the group receiving dexmedetomidine experienced a quicker onset of sensory and motor blockage. The local impact of dexmedetomidine on nerve compound action potential [18] and the amplification of LA's anaesthetic activity [19] may be the cause of the earlier onset. The outcomes were consistent with earlier research [10,11,17,20] that combined bupivacaine and dexmedetomidine in supraclavicular blocks.

Group D experienced longer post-operative analgesia and motor and sensory block durations. The central and peripheral effects of dexmedetomidine account for the outcome. It acts peripherally to provide analgesia by reducing norepinephrine production, which inhibits action potentials on nerves. Analgesia is produced centrally by inhibiting the release of substance P in the nociceptive pathway at the level of the dorsal root neuron [10].

Group D patients did not need sedatives because they were in a comfortable state. This resulted from dexmedetomidine's sedative activity on the CNS's subtype A and C alpha-2 adrenoreceptors (locus coerulus), which causes drowsiness, analgesia, and anxiolysis. [14]

V. Conclusion

Using 1 μg/kg of Dexmedetomidine as an adjuvant to 0.25% Bupivacaine in Supraclavicular brachial plexus block appears to improve outcomes by increasing the duration of analgesia, shortening the onset time of both sensory and motor blockade, and prolonging the duration of sensory blockade, all without notable side effects.

Dexmedetomidine, in summary, works well as an adjuvant to bupivacaine for supraclavicular block. It produces steady hemodynamics, a faster onset of block with a longer duration of analgesia, and efficient sedation.

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