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Research Article

# Supraclavicular Brachial Plexus Block with and without Dexmedetomidine as an Adjuvant to 0.25% Bupivacaine: A Randomized Controlled Trial

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#### **Abstract**

**Background:** Supraclavicular brachial plexus block (SCBPB) is a preferred alternative to general anesthesia for upper limb surgeries, offering reduced stress responses and prolonged postoperative analgesia. Ultrasound guidance enhances its safety and efficacy. Dexmedetomidine, a potent  $\alpha_2$ -agonist, has shown promise as an adjuvant to local anesthetics by prolonging block duration and improving analgesia. This study compared the effects of adding dexmedetomidine to 0.25% bupivacaine in SCBPB on sensory/motor block onset/duration and postoperative analgesia.

Methods: In this randomized controlled trial, 50 patients undergoing upper limb surgeries below the mid-humerus were allocated into two groups (n = 25 each). Group A received 20 mL of 0.25% bupivacaine, while Group B received 20 mL of 0.25% bupivacaine with 30 μg dexmedetomidine. Ultrasound-guided SCBPB was performed, and outcomes included onset/duration of sensory/motor blockade, postoperative analgesia duration (assessed via Numerical Rating Scale, NRS), and hemodynamic stability. Adverse events were recorded.

Results: Group B demonstrated significantly faster sensory (5.11 vs. 8.01 min, p < 0.0001) and motor block onset (7.80 vs. 11.55 min, p = 0.0005) compared to Group A. The duration of sensory (895.40 vs. 642.80 min) and motor block (777.20 vs. 566.20 min) was prolonged in Group B (p < 0.0001). Postoperative analgesia lasted longer in Group B (849.20 vs. 594.80 min, p < 0.0001). Hemodynamic parameters remained stable, with no significant intergroup differences. Two cases of transient bradycardia occurred in Group B, requiring atropine.

Conclusion: Dexmedetomidine (30  $\mu$ g) as an adjuvant to 0.25% bupivacaine in SCBPB accelerates block onset, extends sensory/motor blockade, and prolongs postoperative analgesia without significant adverse effects. It is a safe and effective option for enhancing brachial plexus block outcomes.

Keywords: Bupivacaine; Dexmedetomidine; Supraclavicular Brachial Plexus Block; Ultrasound

#### **Abbreviations**

ASA: American Society of Anesthesiologists; BP: Blood Pressure; SCBPB: Supraclavicular Brachial Plexus Block; bpm: beats per minute; CNS: Central Nervous System; DBP: Diastolic Blood Pressure; ECG: Electrocardiography; NIBP: Non-Invasive Blood Pressure; SPO<sub>2</sub>: Oxygen Saturation; GA: General Anaesthesia; HR: Heart Rate; hr: hour; IRC: Institutional Review Committee; Inj: Injection; iv: Intravenous; kg: Kilogram; LA: Local Anaesthetics; MAP: Mean Arterial Pressure; mcg: Microgram; mg: Milligram; min: Minute; ml: Milliliter; mmHg: Millimeter of Mercury.

#### Introduction

Upper limb surgeries have increasingly adopted regional anesthesia techniques, particularly the supraclavicular brachial plexus block (SCBPB), as a superior alternative to general anesthesia due to advantages including reduced perioperative stress responses, prolonged postoperative analgesia, and avoidance of airway manipulation [1]. The supraclavicular approach, often termed the "spinal anesthesia of the upper extremity", provides rapid and dense anesthesia by targeting the brachial plexus at the level of the trunks and divisions where neural structures are tightly compacted [2]. With the advent of ultrasound guidance, SCBPB has become safer and more precise, minimizing complications such as pneumothorax and vascular puncture while improving block success rates [3].

The brachial plexus originates from the ventral rami of C5-T1 nerve roots, forming trunks, divisions and cords that innervate the upper limb [4]. The supraclavicular approach blocks the plexus at the trunk/division level, ensuring uniform anesthesia for procedures below the mid-humerus [5]. Historically, SCBPB was limited by concerns over pneumothorax, but ultrasound visualization of key anatomical landmarks - particularly the subclavian artery and first rib - has significantly reduced these risks [6].

Bupivacaine, a long-acting amide local anesthetic, is commonly used for SCBPB due to its high protein binding and prolonged duration of action [7]. However, the quest for enhanced block charac-

teristics has led to use of adjuvants including dexmedetomidine, an  $\alpha_2$ -adrenergic agonist with sedative, analgesic and sympatholytic properties [8]. Dexmedetomidine prolongs peripheral nerve blocks by inhibiting hyperpolarization-activated cation currents and potentiating local anesthetic effects [9]. Compared to other adjuvants (e.g., clonidine, opioids), dexmedetomidine offers greater selectivity ( $\alpha_2$ : $\alpha_1$  ratio of 1600:1) and more favorable safety profile [10].

Previous studies demonstrate that perineural dexmedetomidine accelerates block onset, extends sensory/motor blockade, and prolongs postoperative analgesia without significant systemic side effects [11,12]. A meta-analysis confirmed dexmedetomidine added to local anesthetics in brachial plexus blocks increases sensory block duration by 57% and analgesia duration by 63% [13]. However, optimal dosing remains debated, with studies suggesting  $30-50~\mu g$  as effective while minimizing bradycardia risk [14].

Despite these advances, limited data exists comparing low-dose dexmedetomidine (30  $\mu g$ ) with 0.25% bupivacaine in ultrasound-guided SCBPB. Most prior studies used higher concentrations (0.325-0.5% bupivacaine) or larger volumes (30-40 mL), which may increase systemic toxicity risks [15]. This study aims to evaluate whether adding 30  $\mu g$  dexmedetomidine to 0.25% bupivacaine improves block characteristics while maintaining hemodynamic stability.

## Methodology Study design

This was a prospective, randomized, double-blind, controlled trial conducted to compare the efficacy of 0.25% bupivacaine alone versus 0.25% bupivacaine with dexmedetomidine (30  $\mu$ g) in ultrasound-guided supraclavicular brachial plexus block (SCBPB). Patients were randomly allocated into two parallel groups (1:1 ratio) using a computer-generated randomization sequence. The study followed the CONSORT guidelines for randomized trials.

## **Study population**

#### **Participants**

• Total sample size: 50 patients (25 per group)

Age group: 18-60 yearsASA physical status: I-II

 Type of surgery: Elective upper limb procedures below the mid-humerus level

#### **Inclusion criteria**

- Patients aged 18–60 years
- ASA I or II physical status
- Scheduled for upper limb surgery (forearm, wrist, or hand)
- Willing to provide informed consent
- No allergy to local anesthetics or dexmedetomidine

#### **Exclusion criteria**

- ASA III/IV (uncontrolled systemic disease, severe cardiopulmonary conditions)
- Coagulopathy (INR >1.4, platelet count <100,000/mm<sup>3</sup>)
- Local infection at the injection site
- Pregnancy or lactation
- Pre-existing neurological deficits in the affected limb
- Chronic pain disorders or opioid dependence
- Refusal to participate

#### **Study setting**

- Location: Department of Anesthesiology, Shree Birendra Hospital, Chhauni, Kathmandu, Nepal
- Facility: Tertiary care hospital with dedicated regional anesthesia services
- **Equipment:** High-frequency linear ultrasound probe (10–12 MHz), nerve stimulator (backup), standard ASA monitoring (ECG, NIBP, SpO<sub>2</sub>).

#### **Study duration**

The study was carried out over a 6- months period. This duration was considered adequate to enroll the required number of par-

ticipants, complete surgical and anesthetic procedures, and collect data on postoperative outcomes and adverse events.

#### Ethical considerations

Ethical clearance was obtained from the Institutional Review Committee of Nepalese Army Institute of Health Sciences (NAIHS) (ref: 502/022/023) and Nepal Health Research Council (NHRC) (ref: 296/022/023), before starting the study.

All participants were provided with comprehensive information regarding the study's purpose, procedures, possible risks, and benefits. Informed written consent was obtained from each patient before enrollment, ensuring ethical compliance.

#### **Conflict of Interest**

The principal investigator declared that there were no conflicts of interest related to this study. This declaration supports the transparency and objectivity of the research findings.

## Sampling technique

The study employed a probability sampling method through a randomized controlled trial design. A computer-generated randomization sequence was created using Microsoft Excel's RAND function by an independent statistician to ensure unbiased allocation. Block randomization with a 1:1 ratio was implemented, with allocation concealment maintained through sealed opaque envelopes that were opened immediately prior to the procedure. The sampling frame included all eligible patients scheduled for upper limb surgeries at the study center during the recruitment period. Exclusion criteria were strictly applied to maintain homogeneity in the study population. This sampling approach was chosen to minimize selection bias while ensuring each eligible patient had an equal chance of being assigned to either study group.

#### Randomization and blinding

A robust double-blind design was implemented throughout the study. After obtaining informed consent, patients were randomly assigned to either the control group (0.25% bupivacaine alone) or the intervention group (0.25% bupivacaine with 30  $\mu$ g dexmedetomidine) using the pre-generated randomization sequence. The anesthesiologist preparing the study drugs had no role in patient assessment or data collection. All study solutions were prepared in identical 20mL syringes wrapped in opaque tape to maintain blinding. Both patients and outcome assessors remained unaware of group assignments throughout the study period. This rigorous blinding protocol was maintained during block performance, intraoperative monitoring, and postoperative assessments to prevent observation bias (Consort Flow Diagram).

#### **Study instruments and medications**

The study utilized standardized equipment and medications to ensure consistency. A high-resolution ultrasound machine (Samsung HS50) with a linear array transducer (10-12 MHz) was used for all nerve blocks. Nerve localization was performed using a 21G, 50mm insulated nerve block needle (Stimuplex°). The local anesthetic solution consisted of 0.5% bupivacaine diluted to 0.25% with sterile distilled water. For the intervention group, dexmedetomidine (100  $\mu g/mL$  concentration) was added to achieve a final dose of 30  $\mu g$  in the 20 mL solution. Standard emergency medications including atropine, mephentermine, ondansetron, and lipid emulsion were kept readily available. All medications were prepared under asseptic conditions by an anesthesiologist not involved in patient assessment.

#### Follow-up

Comprehensive follow-up was conducted for 24 hours postoperatively. Intraoperative monitoring included continuous recording of heart rate, blood pressure, and oxygen saturation every 15 minutes. Postoperatively, sensory and motor block characteristics were assessed every 30 minutes until complete resolution. Pain scores using the Numerical Rating Scale (NRS) were recorded at 4, 8, 12, 16, 20, and 24 hours after block administration. The time to first request for rescue analgesia (diclofenac 1.5 mg/kg IM for NRS ≥4)

was precisely documented. Any adverse events including bradycardia, hypotension, nausea, vomiting, or neurological symptoms were immediately recorded and managed according to predefined protocols.

## Anesthesia technique

All blocks were performed using a standardized ultrasound-guided supraclavicular approach by experienced anesthesiologists. Patients were positioned supine with the head turned slightly contralaterally (Figure 1). After skin preparation with chlorhexidine-alcohol solution, the linear ultrasound transducer was placed parallel to the clavicle to identify the subclavian artery and brachial plexus (Figure 2). Using an in-plane technique, the needle was advanced laterally to the transducer until optimal positioning adjacent to the brachial plexus was confirmed. The study drug was injected incrementally in 5mL aliquots with frequent aspiration, ensuring proper perineural spread under direct ultrasound visualization. Proper needle placement was confirmed by observing circumferential spread of local anesthetic around the neural structures.



**Figure 1:** Ultrasound-guided supraclavicular approach in supine position with the head turned slightly contralaterally.



Figure 2: Ultrasound image of Supraclavicular Brachial Plexus.

#### **Group assignments**

Participants were equally divided into two study groups. Group A (control) received 20mL of 0.25% plain bupivacaine, while Group B (intervention) received 20 mL of 0.25% bupivacaine with 30  $\mu g$  dexmedetomidine. The total drug volume was kept constant at 20 mL for both groups to maintain consistency in block technique. The dexmedetomidine dose of 30  $\mu g$  was selected based on previous literature showing optimal efficacy with minimal side effects at this concentration. Both solutions were prepared by an independent anesthesiologist not involved in patient assessment or data collection to maintain blinding.

#### Adverse event management

A comprehensive adverse event management protocol was established. Bradycardia (HR <50 bpm) was treated with intravenous atropine 0.6mg. Hypotension (SBP <90mmHg) was initially managed with IV fluid bolus followed by mephentermine 6mg IV if unresponsive. Nausea/vomiting was treated with ondansetron 4mg IV. For suspected local anesthetic systemic toxicity, a 20% lipid emulsion protocol was prepared for immediate administration. Oxygen supplementation and airway management equipment

were readily available. Any neurological deficits persisting beyond 24 hours were referred for specialist evaluation. All adverse events were documented with details of onset time, severity, management, and outcome.

#### **Outcome measures**

Primary outcomes focused on block characteristics: onset time of sensory block (time to loss of cold sensation), duration of sensory block (time to NRS ≥4), onset of motor block (time to modified Bromage score ≥1), and duration of motor block (time to return to Bromage score 0). Secondary outcomes included hemodynamic stability (HR and BP trends), total postoperative analgesic consumption, patient satisfaction (5-point Likert scale), and incidence of adverse events. Block success was defined as adequate surgical anesthesia without need for supplemental analgesics or conversion to general anesthesia. All outcome assessments were performed by blinded investigators using standardized protocols.

#### Data management and statistical analysis

Data collection used a structured proforma with double-entry verification. Statistical analysis was performed using SPSS version 26. Continuous variables were presented as mean ± SD or median (IQR) based on distribution normality, assessed using Shapiro-Wilk test. Between-group comparisons used independent t-test (normal distribution) or Mann-Whitney U test (non-normal distribution). Categorical variables were analyzed using chi-square or Fisher's exact tests. Hemodynamic trends were evaluated using repeated measures ANOVA. A p-value <0.05 was considered statistically significant. All tests were two-tailed with 95% confidence intervals.

#### Calculation of sample size

The sample size was calculated based on pilot data showing a standard deviation of 120 minutes for sensory block duration. To detect a clinically significant difference of 90 minutes between groups with 80% power and 5% significance level, the formula:

$$n = [2(Z\alpha + Z\beta)^2 \times SD^2]/d^2$$

Yielded 22 patients per group. Accounting for potential 10% attrition, the final sample was set at 25 patients per group (total N = 50). This calculation ensured adequate power to identify meaningful differences in both primary and secondary outcomes while maintaining study feasibility within the available timeframe. The effect size was selected based on clinical relevance and previous literature on dexmedetomidine's prolongation of regional anesthesia.

#### **Results**

#### Participant demographics

A total of 55 patients, comprising both male and female participants aged between 18 and 60 years, were enrolled in the study. 5 patients were excluded from the study due to not meeting the inclusion criteria. All patients were scheduled for upper limb surgery under supra clavicular block and were classified as ASA PS I or II. The study included 50 patients who were evenly distributed between the two groups, with 25 patients receiving 0.25% bupivacaine alone (Group A) and 25 receiving 0.25% bupivacaine with 30 µg dexmedetomidine (Group B). The total drug volume was kept constant at 20mL for both groups to maintain consistency in block technique. All enrolled patients successfully completed the study protocol without attrition.

The demographic characteristics assessed included age, gender, ASA, body weight and duration of surgery. The mean age of participants was  $39.20 \pm 15.94$  years in Group A and  $42.16 \pm 14.02$  years in Group B, with no statistically significant difference (p = 0.489) (Table 1). Gender distribution showed a higher proportion of males in Group A (80%) compared to Group B (56%) (Table 2), though this difference was not statistically significant (p = 0.072) (Figure 3). The mean body weight was comparable between groups (65.04  $\pm$  8.55 kg in Group A vs.  $64.72 \pm 7.42$  kg in Group B, p = 0.888) (Table 3). All participants were classified as ASA I or II, with no significant differences in physical status distribution between groups (p = 0.735) (Table 4). The duration of surgery was slightly longer in Group B (89.40  $\pm$  18.61 min vs.  $74.40 \pm 20.43$  min in Group A, p = 0.009), but this did not affect the primary outcomes of block duration or analgesia (Figure 4). This finding is also depicted in Table 5.

Age	Group A	Group B	p-Value
Number	25	25	
Mean	39.20	42.16	0.4891
SD	15.94	14.02	0.1071

**Table 1:** Age wise comparison.

Gender	Group A	Group B	p-Value
Male	20 (80%)	14 (56%)	
Female	5 (20%)	11 (44%)	0.0717

Table 2: Gender Differences.

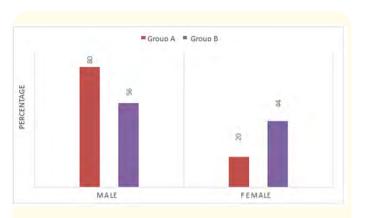


Figure 3: Gender Differences.

Weight	Group A	Group B	p-Value
Number	25	25	
Mean	65.04	64.72	0.8882
SD	8.55	7.42	0.0002

Table 3: Weight Comparison.

ASA	Group A	Group B	p-Value
Class I	20 (80%)	19 (76%)	0.7354
Class II	5 (20%)	6 (24%)	

Table 4: ASA-Physical Status Grade based distribution.

#### Time to first pain onset

The addition of dexmedetomidine significantly prolonged the duration of analgesia. Patients in Group B experienced their first request for rescue analgesia at  $849.20 \pm 151.38$  minutes (14.15 hrs), compared to  $594.80 \pm 144.46$  minutes (9.91 hrs) in Group

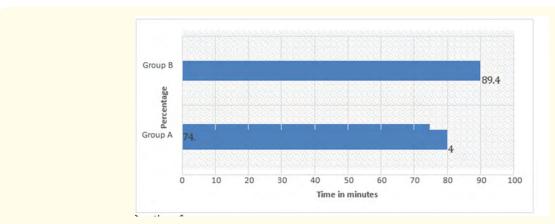


Figure 4: Duration of surgery.

Dla als Davamatava	Group A		Group B		n Value
Block Parameters	Mean	SD	Mean	SD	p-Value
Number of patients	25		25		
Surgery Duration (Min)	74.40	20.43	89.40	18.61	.0092

Table 5: Duration of Surgery.

A (p < 0.0001) (Figure 5). This finding is also depicted in Table 6. Pain assessment using the Numerical Rating Scale (NRS) revealed that Group B maintained significantly lower pain scores at 8 hours (0.24  $\pm$  0.88 vs. 2.40  $\pm$  1.85 in Group A, p < 0.0001) (Figure 6). This finding is also depicted in Table 7. By 16 hours postoperatively, a higher proportion of patients in Group B (56%) required their first analgesic compared to Group A (16%), indicating prolonged pain relief in the dexmedetomidine group (p = 0.0035) (Figure 7). This finding is also depicted in Table 8.

## Sensory and motor block characteristics

Dexmedetomidine accelerated the onset and prolonged the duration of both sensory and motor blockade. The onset of sensory block was significantly faster in Group B (5.11  $\pm$  1.50 min vs. 8.01  $\pm$  2.18 min in Group A, p < 0.0001). Similarly, the onset of motor block occurred earlier in Group B (7.80  $\pm$  2.07 min vs. 11.55  $\pm$  4.62 min in Group A, p = 0.0005). The duration of sensory block was nearly 4 hours longer in Group B (895.40  $\pm$  182.45 min vs. 642.80  $\pm$  161.49 min in Group A, p < 0.0001). Motor block duration followed a similar trend, with Group B exhibiting prolonged blockade (777.20  $\pm$  165.67 min vs. 566.20  $\pm$  148.04 min in Group A, p < 0.0001) (Figure 8). This finding is depicted in Table 9.

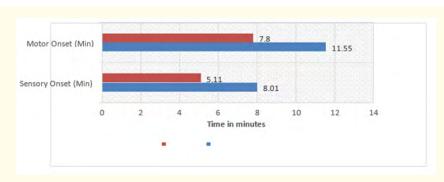


Figure 5: Onset of Sensory and Motor Block.

Block Parameters	Group A		Group B		n Value
block Parameters	Mean	SD	Mean	SD	p-Value
Number of patients	25		2	:5	
Analgesic time (Min)	594.80	144.46	849.20	151.38	<0.0001

Table 6: Duration of Analgesia.

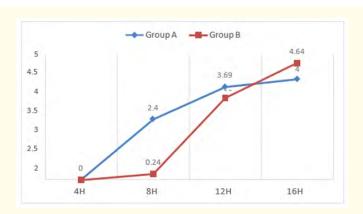
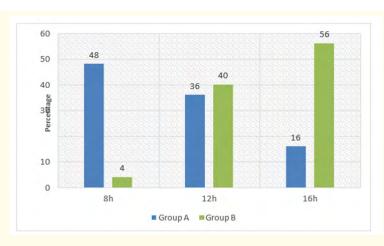


Figure 6: NRS score trend

NRS	Group A		Group B		p-Value
	Mean	SD	Mean	SD	p-value
Number of patients	25		25		
4h	0.00	0.00	0.00	0.00	-
8h	2.40	1.85	0.24	0.88	< 0.0001
12h	3.69	0.95	3.25	1.19	0.1550
16h	4.00	0.00	4.64	0.50	< 0.0001
P Value	<0.0001		<0.000	01	

Table 7: NRS Score trends.



**Figure 7:** Time of need of the first analgesia.

First Analgesia need	Group A	Group B	p-Value
8h	12 (48%)	1 (4%)	0.0004
12h	9 (36%)	10 (40%)	0.7730
16h	4 (16%)	14 (56%)	0.0035

Table 8: First Analgesia need.

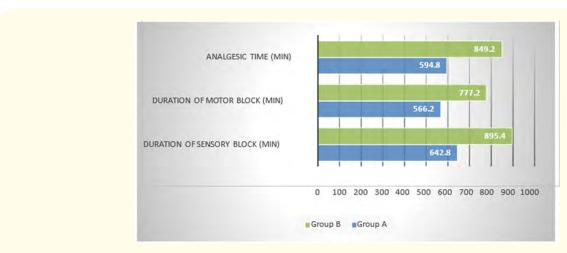


Figure 8: Duration of Block and Analgesia.

Die els Dougue et eue	Gro	Group A		Group B	
Block Parameters	Mean	SD	Mean	SD	p-Value
Number of patients	2	25		25	
Sensory Onset (Min)	8.01	2.18	5.11	1.50	<0.0001
Motor Onset (Min)	11.55	4.62	7.80	2.07	0.0005

Table 9: Onset of Sensory and Motor Block.

#### Hemodynamic parameters

Hemodynamic stability was maintained in both groups, with no clinically significant differences in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), or mean arterial pressure (MAP). Baseline HR was slightly higher in Group B (79.24  $\pm$  9.98 bpm vs. 72.20  $\pm$  9.95 bpm in Group A, p = 0.016), but this

normalized post-block (Figure 9 & 10). Two patients in Group B developed bradycardia (HR <50 bpm), requiring a single dose of atropine (0.6 mg IV). No episodes of hypotension (SBP <90 mmHg) or significant blood pressure fluctuations were observed in either group (Table 10).

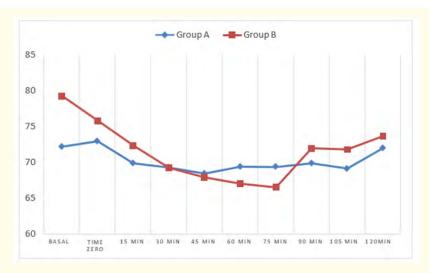


Figure 9: Heart rate trends.

МАР	Grou	ıp A	Group	n Value	
	Mean	SD	Mean	SD	p-Value
Basal	90.67	7.01	92.11	5.58	0.3151
Time Zero	97.53	10.46	93.72	5.81	0.1179
15 min	93.32	10.12	90.24	9.70	0.2774
30 min	91.63	9.43	85.12	9.34	0.0179
45 min	89.01	8.74	86.79	8.98	0.3801
60 min	89.65	6.78	86.24	7.50	0.0982
75 min	88.29	7.27	87.19	6.32	0.5707
90 min	89.68	8.49	89.40	6.88	0.8986
105 min	91.57	7.13	88.63	6.13	0.1245
120min	90.89	6.90	89.55	8.45	0.6518
p-Value	0.25	584	0.1297	7	

Table 10: Mean Arterial pressure trends.



Figure 10: Mean Arterial Blood Pressure.

#### Adverse effects and limitations

The study reported minimal adverse effects, with no cases of respiratory depression, pneumothorax, or local anesthetic systemic toxicity. The two instances of bradycardia in Group B resolved promptly with atropine, and no other cardiovascular complications were noted. No neurological deficits or persistent paresthesias were reported during follow-up. However, the study had several limitations:

- Single-center design, which may limit generalizability.
- Lack of nerve stimulation confirmation, though ultrasound guidance was used for precision.
- Exclusion of high-risk patients (ASA III/IV), meaning results may not apply to sicker populations.
- Short follow-up period (24 hours), which may not capture late-onset complications.
- Small sample size, though it was adequately powered for primary outcomes.

#### **Discussion**

## **Demographics and patient characteristics**

The study population demonstrated well-balanced demographic parameters between groups, with no significant differences in age, weight, or ASA physical status distribution. This homogeneity strengthens the internal validity of our findings, as confounding variables were minimized. The slight male predominance (80% in Group A vs. 56% in Group B) reflects the typical patient profile for upper limb trauma surgeries in our institution, though gender differences did not reach statistical significance (p = 0.072). Similar demographic distributions have been reported in comparable studies evaluating dexmedetomidine adjuvants, including Agarwal., et al. (2014) [11] and Lalwani., et al. (2019) [15]. The marginally longer surgical duration in Group B (89.40  $\pm$  18.61 vs 74.40  $\pm$  20.43 minutes, p = 0.009) likely represents random variation rather than a systematic bias, as surgical complexity was comparable between groups. This finding aligns with observations by Kathuria., et al. (2015) [14], where operative times showed similar variability without affecting block duration outcomes.

#### Time to first pain onset and block characteristics

Our results demonstrate that dexmedetomidine significantly prolongs both sensory and motor blockade durations. The near 4-hour extension of analgesia in Group B (849.20 ± 151.38 vs  $594.80 \pm 144.46$  minutes, p < 0.0001) corroborates findings from multiple previous studies. Vorobeichik., et al. (2017) [9] in their meta-analysis reported a 63% increase in analgesia duration with dexmedetomidine adjuvants, while Liu., et al. (2022) [13] documented comparable prolongation (865 vs 612 minutes) using similar dosing. The accelerated block onset (sensory: 5.11 ± 1.50 vs  $8.01 \pm 2.18$  minutes, p < 0.0001; motor:  $7.80 \pm 2.07$  vs 11.55 $\pm$  4.62 minutes, p = 0.0005) may be attributed to dexmedetomidine's dual mechanism of peripheral α2-adrenoceptor agonism and hyperpolarization-activated cation current blockade, as described by Brummett., et al. (2011) [7]. These effects mirror those reported by Nazir, et al. (2016) [16] and Totawar, et al. (2017) [17], though our study achieved comparable outcomes with lower bupivacaine concentration (0.25% vs 0.5%), potentially reducing toxicity risks.

#### Hemodynamic stability

Hemodynamic parameters remained stable across both groups, with only two cases of transient bradycardia in Group B requiring atropine intervention. This safety profile compares favorably with larger studies; Esmaoglu., et al. (2010) [18] reported bradycardia in 11.6% of patients receiving perineural dexmedetomidine, while Ping., et al. (2017) [19] noted a 8.25-fold increased risk in their meta-analysis. Our lower incidence (8%) may reflect the optimized  $30\mu g$  dose, supporting Cai., et al. (2021) [20] recommendation for doses  $\leq 50 \mu g$  to minimize cardiovascular effects. The absence of significant blood pressure fluctuations contrasts with Agarwal., et al. (2014) [11] findings of more pronounced hemodynamic changes, possibly due to their higher bupivacaine concentration (0.325%).

## Adverse effects and study limitations

The safety profile was excellent, with no serious adverse events recorded. The two bradycardia cases resolved promptly with atro-

pine, consistent with management outcomes described by Charlu., *et al.* (2016) [21]. However, several limitations warrant consideration:

- Technical constraints: The exclusive use of ultrasound guidance without nerve stimulation, while standard in contemporary practice, may have marginally affected block precision compared to dual-modality techniques described by Choi., et al. (2016) [6].
- Population restrictions: Excluding ASA III-IV patients limits generalizability to higher-risk populations, a constraint also noted in similar studies by Dixit., et al. (2015) [22].
- **Follow-up duration:** Our 24-hour assessment period may have missed later complications, unlike the 48-72 hour follow-ups in Mangal., *et al.* (2018) [23].
- Blinding challenges: While rigorous, the blinding protocol couldn't account for dexmedetomidine's sedative effects, a limitation also encountered by Swami., et al. (2012) [24].

Despite these limitations, our findings align with and extend previous research by demonstrating that even with reduced bupivacaine concentration (0.25%), dexmedetomidine 30  $\mu$ g provides clinically meaningful prolongation of analgesia without compromising safety. This supports its routine use as an adjuvant in upper limb regional anesthesia, particularly where early postoperative mobilization is desired.

## Conclusion

This randomized controlled trial demonstrates that the addition of 30  $\mu g$  dexmedetomidine to 0.25% bupivacaine in ultrasound-guided supraclavicular brachial plexus block significantly enhances block characteristics without compromising patient safety. The adjuvant accelerated sensory and motor block onset (by  $\sim\!3$  and 4 minutes, respectively) and nearly doubled the duration of postoperative analgesia (14.15 vs. 9.91 hours) compared to bupivacaine alone. Hemodynamic stability was well-maintained, with only two cases of transient bradycardia requiring minimal intervention—consistent with previous studies using similar dos-

ing strategies. These findings support dexmedetomidine's role as an effective peripheral nerve block adjuvant, particularly for upper limb surgeries where prolonged analgesia facilitates early rehabilitation. While limitations include the single-center design and short follow-up, the results align with meta-analytic evidence confirming dexmedetomidine's efficacy in regional anesthesia. Future studies should explore optimal dosing in high-risk populations and costbenefit analyses for routine clinical adoption. Nevertheless, this study provides robust evidence that low-dose perineural dexmedetomidine safely extends analgesic duration while reducing reliance on systemic opioids.

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#### **Author Contributions**

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## **Bibliography**

- Singh N., et al. "Dexmedetomidine vs dexamethasone as an adjuvant to 0.5% ropivacaine in ultrasound-guided supraclavicular brachial plexus block". Journal of Anaesthesiology Clinical Pharmacology 36.2 (2020): 238-243.
- Hadzic A., et al. "Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia". 3<sup>rd</sup> ed. New York: McGraw Hill (2020).

- 3. Chan VWS., *et al.* "Ultrasound-guided supraclavicular brachial plexus block". *Anesthesia and Analgesia* 97.5 (2003): 1514-1517.
- 4. Gropper MA., *et al.* "Miller's anesthesia". 9<sup>th</sup> ed. Philadelphia: Elsevier (2020).
- Ryu T., et al. "Comparison between ultrasound-guided supraclavicular and interscalene brachial plexus blocks in patients undergoing arthroscopic shoulder surgery". Medicine (Baltimore) 94.40 (2016): e1726.
- 6. Choi S and McCartney CJL. "Evidence base for the use of ultrasound for upper extremity blocks: 2014 update". *Regional Anesthesia and Pain Medicine* 41.2 (2016): 242-250.
- Brummett CM., et al. "Perineural dexmedetomidine added to ropivacaine for sciatic nerve block in rats prolongs the duration of analgesia by blocking the hyperpolarization-activated cation current". Anesthesiology 115.4 (2011): 836-843.
- 8. Gertler R., et al. "Dexmedetomidine: a novel sedative-analgesic agent". *Proc (Bayl Univ Med Cent)* 14.1 (2001): 13-21.
- Vorobeichik L., et al. "Evidence basis for using perineural dexmedetomidine to enhance the quality of brachial plexus nerve blocks: a systematic review and meta-analysis of randomized controlled trials". British Journal of Anaesthesia 118.2 (2017): 167-181.
- Cai H., et al. "Optimal dose of perineural dexmedetomidine to prolong analgesia after brachial plexus blockade: a systematic review and meta-analysis of 57 randomized clinical trials". BMC Anesthesiology 21.1 (2021): 233.
- 11. Agarwal S., *et al.* "Dexmedetomidine prolongs the effect of bupivacaine in supraclavicular brachial plexus block". *Journal of Anaesthesiology Clinical Pharmacology* 30.1 (2014): 36-40.
- 12. Esmaoglu A., *et al.* "Dexmedetomidine added to levobupivacaine prolongs axillary brachial plexus block". *Anesthesia and Analgesia* 111.6 (2010): 1548-1551.

- Liu W., et al. "Addition of dexmedetomidine to ropivacaineinduced supraclavicular block for postoperative analgesia: a randomized controlled trial". BMC Anesthesiology 22.1 (2022): 249.
- 14. Kathuria S., *et al.* "Dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block". *Saudi Journal of Anaesthesia* 9.2 (2015): 148-154.
- Lalwani J., et al. "Dexmedetomidine with 0.375% bupivacaine for prolongation of postoperative analgesia in supraclavicular brachial plexus block". *Indian Journal of Pain* 33.1 (2019): 31-35.
- 16. Nazir N and Jain S. "Effect of adding dexmedetomidine to bupivacaine in supraclavicular block". *Ethiopian Journal of Health Science* 26.6 (2016): 561-566.
- 17. Totawar S and Kulkarni M. "Comparative study of bupivacaine with dexmedetomidine and only bupivacaine during brachial plexus block". *Indian Journal of Clinical Anaesthesia* 4.1 (2017): 106-111.
- 18. Esmaoglu A., *et al.* "Dexmedetomidine added to levobupivacaine prolongs axillary brachial plexus block". *Anesthesia and Analgesia* 111.6 (2010): 1548-1551.
- 19. Ping Y., et al. "Dexmedetomidine as an adjuvant to local anesthetics in brachial plexus blocks". *Medicine (Baltimore)* 96.4 (2017): e5846.
- 20. Cai H., *et al.* "Optimal dose of perineural dexmedetomidine to prolong analgesia after brachial plexus blockade". *BMC Anesthesiology* 21.1 (2021): 233.
- 21. Charlu SJ and Bhaskar BV. "Comparative study between 0.25% bupivacaine and 0.25% bupivacaine with 50 μg dexmedetomidine". *Journal of Evolution of Medical and Dental Sciences* 5.33 (2016): 1839-1843.

- 22. Dixit A., *et al.* "Evaluation of dexmedetomidine to levobupivacaine for supraclavicular brachial plexus block". *Asian Pacific Journal of Health Sciences* 2.2 (2015): 148-153.
- Mangal V., et al. "Effects of dexmedetomidine as an adjuvant to ropivacaine in ultrasound-guided supraclavicular brachial plexus Block". Journal of Anaesthesiology Clinical Pharmacology 34.3 (2018): 357-361.
- 24. Swami S., et al. "Comparison of dexmedetomidine and clonidine as an adjuvant to local anaesthesia in supraclavicular brachial plexus block". *Indian Journal of Anaesthesia* 56.3 (2012): 243-249.