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Original Research Article

Dexmedetomidine versus Clonidine as an Adjuvant to Ropivacaine in Ultrasound-Guided Supraclavicular Brachial Plexus Block for Postoperative Analgesia in Upper Limb Surgery: A Randomised, Double-Blind, Comparative Study

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Abstract

Background: Ropivacaine offers a favourable cardiovascular and neurological safety profile for brachial plexus blockade but provides a duration of analgesia that is often insufficient for upper limb surgery. Alpha-2 adrenergic agonists are established perineural adjuvants, and comparative data between the two agents in common use, dexmedetomidine and clonidine, remain limited in Indian practice. This study compared dexmedetomidine and clonidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block.

Methods: Sixty adults of American Society of Anesthesiologists physical status I or II scheduled for elective upper limb surgery below the mid-humerus were randomised to receive ultrasound-guided supraclavicular brachial plexus block with 30 mL of 0.5 percent ropivacaine combined with either dexmedetomidine 1 microgram per kilogram (Group D) or clonidine 1 microgram per kilogram (Group C). Onset and duration of sensory and motor blockade, duration of analgesia, visual analogue scores, rescue analgesic consumption, sedation and adverse effects were recorded by a blinded observer.

Results: Sensory block onset was faster with dexmedetomidine (8.4 versus 11.2 minutes, $p < 0.001$), as was motor block onset (11.6 versus 14.8 minutes, $p < 0.001$). Duration of sensory block (486.3 versus 372.8 minutes), motor block (418.7 versus 324.5 minutes) and analgesia (612.4 versus 468.2 minutes) were all significantly longer with dexmedetomidine ($p < 0.001$ for each), and 24-hour rescue analgesic consumption was lower (96.7 versus 141.3 mg, $p < 0.001$). Sedation scores were higher with dexmedetomidine ($p = 0.018$), while haemodynamic adverse effects did not differ significantly.

Conclusion: Dexmedetomidine 1 microgram per kilogram was superior to clonidine at an equivalent dose as an adjuvant to ropivacaine in ultrasound-guided supraclavicular brachial plexus block, hastening block onset, prolonging sensory and motor blockade and extending postoperative analgesia by approximately two and a half hours, at the cost of greater but clinically acceptable sedation.

Keywords: Dexmedetomidine; Clonidine; Ropivacaine; Brachial Plexus Block; Ultrasonography, Interventional; Analgesia, Postoperative.

Introduction

Supraclavicular brachial plexus block provides dense and reliable anaesthesia for surgery of the upper limb below the shoulder, blocking the plexus at the level of the trunks and divisions where the neural elements are most compactly arranged. The introduction of ultrasound guidance has transformed the safety profile of the technique, permitting direct visualisation of the plexus, the subclavian artery, the first rib and the pleura, real-time observation of needle advancement and of local anaesthetic spread, and a consequent reduction in the incidence of pneumothorax that had historically limited enthusiasm for the supraclavicular approach. The block avoids the physiological consequences of general anaesthesia, provides excellent operating conditions, and extends analgesia into the early postoperative period, which is of particular value in day-care and short-stay orthopaedic practice. Ropivacaine has become the local anaesthetic of choice for peripheral nerve blockade in many units. As the pure S-enantiomer of a propyl homologue of

bupivacaine, it possesses reduced lipophilicity and correspondingly lower cardiotoxicity and central neurotoxicity than racemic bupivacaine, together with a degree of differential blockade that spares motor function relative to sensory blockade at lower concentrations. These properties are advantageous when large volumes are required for plexus anaesthesia. The principal limitation is duration. Single-injection ropivacaine blockade of the brachial plexus typically provides analgesia for six to eight hours, which frequently falls short of the period of most intense postoperative pain and necessitates early recourse to systemic analgesia at a time when the patient may already have been discharged from the recovery area.

Numerous adjuvants have been investigated to extend block duration, among which the alpha-2 adrenergic agonists have the longest record of clinical use. Clonidine, a partial alpha-2 agonist with an alpha-2 to alpha-1 selectivity ratio of approximately 200 to 1, has been used perineurally for several decades. The meta-analysis of Popping and colleagues, pooling data from randomised trials of peripheral nerve and plexus blockade, established that clonidine prolongs the duration of both sensory and motor blockade and of postoperative analgesia when added to intermediate and long-acting local anaesthetics, at the cost of a dose-related increase in hypotension, bradycardia and sedation.[1] Dexmedetomidine is a more recently introduced and considerably more selective agent, with an alpha-2 to alpha-1 ratio of approximately 1620 to 1, and a pharmacodynamic profile that includes analgesia, sedation without significant respiratory depression, and anxiolysis. Its mechanism as a perineural adjuvant is not confined to alpha-2 receptor agonism at the level of the spinal cord and locus coeruleus. Experimental work by Brummett and colleagues demonstrated that perineural dexmedetomidine added to ropivacaine produced a dose-dependent prolongation of thermal antinociception in the rat sciatic nerve model, an effect attributable to blockade of the hyperpolarisation-activated cation current and consequent inhibition of the return of the axon to its resting potential after an action potential.[2]Clinical evaluation has broadly supported the experimental promise. Esmaoglu and colleagues demonstrated that dexmedetomidine added to levobupivacaine prolonged axillary brachial plexus block.[3] The systematic reviews of Abdallah and Brull and of Vorobeichik and colleagues concluded that perineural dexmedetomidine meaningfully enhances the quality and duration of brachial plexus blockade, while drawing attention to a dose-related increase in transient bradycardia, hypotension and sedation, and to the persisting uncertainty as to how much of the effect is genuinely perineural rather than systemic.[4,5] The direct comparison between the two alpha-2 agonists has attracted less attention. The randomised study of Swami and colleagues, comparing dexmedetomidine and clonidine as adjuvants in supraclavicular block, reported faster onset and longer duration with dexmedetomidine.[6] The systematic review and meta-analysis of El-Boghdadly and colleagues, which pooled randomised trials comparing the two agents specifically in supraclavicular blockade, found dexmedetomidine to prolong sensory block, motor block and analgesia by approximately 20 percent relative to clonidine, while conferring a substantially increased odds of transient bradycardia and of postoperative sedation.[7]

Two considerations justify further study. First, most of the available comparative evidence derives from blocks performed with bupivacaine or levobupivacaine, and much of it from landmark-based or nerve stimulator-guided techniques rather than ultrasound guidance. Ropivacaine has different physicochemical properties and a different pattern of differential blockade, and ultrasound guidance alters both the reliability and the volume requirement of the block, so that findings from the older literature cannot be assumed to transfer directly. Second, the balance of benefit and risk between the two agents is dose-dependent and therefore depends on the doses selected for comparison, yet many published comparisons have used doses that are not obviously equipotent. The present randomised, double-blind study was designed to compare dexmedetomidine and clonidine at an identical weight-based dose of 1 microgram per kilogram, added to a standardised volume and concentration of ropivacaine, in ultrasound-guided supraclavicular brachial plexus block, with the duration of postoperative analgesia as the principal outcome of interest.

Aims and Objectives

The study aimed to establish whether dexmedetomidine or clonidine, administered at an identical weight-based dose as a perineural adjuvant to ropivacaine, offered the more favourable profile in ultrasound-guided supraclavicular brachial plexus block. The primary objective was to compare the duration of postoperative analgesia, defined as the interval from completion of local anaesthetic injection to the first request for rescue analgesia, between patients receiving dexmedetomidine 1 microgram per kilogram and those receiving clonidine 1 microgram per kilogram. The secondary objectives were to compare the time to onset of sensory blockade and of motor blockade, the duration of sensory blockade and of motor blockade, visual analogue pain scores at fixed postoperative intervals, the total dose of rescue analgesic consumed in the first 24 postoperative hours, and the proportion of patients requiring more than one

dose of rescue analgesic. A further objective was to compare the safety and tolerability of the two agents by recording serial heart rate and mean arterial pressure, the incidence of bradycardia, hypotension, nausea, vomiting and dry mouth, and the depth of sedation as measured by the Ramsay sedation score, and to compare block success rates and overall patient satisfaction between the two groups.

Materials and Methods

Study Design and Setting: This prospective, randomised, double-blind, comparative study was conducted in the Department of Anaesthesia of the General Hospital, Chikkanayakanahalli, Tumkur District, Karnataka, over an eighteen-month period from January 2024 to June 2025. Approval of the Institutional Ethics Committee was obtained before the commencement of enrolment and the trial was registered prospectively with the Clinical Trials Registry of India. The study was conducted in accordance with the principles of the Declaration of Helsinki and with the National Ethical Guidelines for Biomedical and Health Research Involving Human Participants issued by the Indian Council of Medical Research. Written informed consent was obtained from every participant during the pre-anaesthetic evaluation on the day preceding surgery, at which time the visual analogue scale was explained and the participant was familiarised with its use.

Participants: Patients of either sex aged 18 to 60 years, of American Society of Anesthesiologists physical status I or II, scheduled for elective orthopaedic or plastic surgery of the upper limb at or below the level of the mid-humerus, with an anticipated duration of surgery of less than three hours, and willing to provide written informed consent were eligible for inclusion.

Patients were excluded if consent was refused, if physical status was III or above, if body mass index exceeded 35 kg per square metre, or if there was a coagulation disorder or ongoing anticoagulant therapy, local infection at the site of needle insertion, pre-existing peripheral neuropathy or brachial plexus injury, or known hypersensitivity to ropivacaine, dexmedetomidine or clonidine. Further exclusions comprised pregnancy or lactation, significant hepatic or renal impairment, uncontrolled hypertension, second or third degree atrioventricular block, resting bradycardia below 50 beats per minute, ejection fraction below 40 percent, current treatment with alpha-2 agonists or beta-adrenergic antagonists, chronic opioid or analgesic use, a history of psychiatric illness precluding reliable pain scoring, and inability to comprehend the visual analogue scale. Patients in whom the block failed and general anaesthesia became necessary were excluded from the analysis of block characteristics and were replaced to preserve the intended group sizes.

Sample Size Calculation: The sample size was calculated for the primary outcome, the duration of postoperative analgesia. On the basis of previously published comparative data, the standard deviation of the duration of analgesia was taken as 90 minutes and the minimum clinically important difference between the two agents as 60 minutes. The formula for comparison of two independent means was applied:

$$n \text{ per group} = 2 \times (Z_{1-\alpha/2} + Z_{1-\beta})^2 \times \sigma^2 / d^2$$

Substituting $Z_{1-\alpha/2} = 1.96$ for a two-sided alpha of 0.05, $Z_{1-\beta} = 1.28$ for 90 percent power, $\sigma = 90$ minutes and $d = 60$ minutes:

$$n = 2 \times (3.24)^2 \times (90)^2 / (60)^2$$

$$n = 2 \times 10.50 \times 8100 / 3600$$

$$n = 2 \times 10.50 \times 2.25 = 47.25 / 2 = 23.6 \text{ per group}$$

Twenty-four participants per group were therefore required. Allowing for block failure, protocol violation and loss of postoperative data, the group size was increased to 30, giving a total sample of 60 participants and providing a margin of power in excess of that specified.

Randomisation and Blinding: Participants were allocated in a 1:1 ratio to one of two groups using a computer-generated random number sequence. Allocation was concealed in sequentially numbered, sealed, opaque envelopes opened only after the participant had entered the operating room. Group D received 30 mL of 0.5 percent ropivacaine with dexmedetomidine 1 microgram per kilogram, and Group C received 30 mL of 0.5 percent ropivacaine with clonidine 1 microgram per kilogram. The adjuvant was diluted to a volume of 1 mL and added to the ropivacaine by an anaesthesiologist who took no further part in the conduct of the case, so that the final volume and appearance of the study solution were identical in the two groups. The patient, the anaesthesiologist performing the block, the observer recording all block characteristics and postoperative data, and the surgical team remained blinded to allocation throughout. The randomisation code was broken only after completion of data analysis.

Block Technique: All participants were kept fasting according to institutional protocol and received oral alprazolam 0.25 mg and ranitidine 150 mg on the night before surgery. On arrival in the operating room, standard monitoring comprising electrocardiography, non-invasive arterial pressure measurement and pulse oximetry was established, and baseline heart rate, systolic, diastolic and mean arterial pressures and peripheral oxygen saturation were recorded. An intravenous

cannula was secured in the contralateral limb and Ringer lactate was commenced. No sedative premedication was administered in the operating room, in order to avoid confounding the assessment of sedation.

With the participant supine, the head turned 45 degrees to the contralateral side and a small roll placed between the scapulae, the supraclavicular fossa was prepared with chlorhexidine in alcohol and draped. Ultrasonographic imaging was performed using a portable ultrasound system with a high-frequency linear array transducer operating at 6 to 13 MHz, enclosed in a sterile sheath. The transducer was placed in the coronal oblique plane in the supraclavicular fossa to obtain the characteristic short-axis view of the subclavian artery, the hypoechoic neural cluster lateral and superficial to it, the hyperechoic first rib and the underlying pleura. A 22-gauge, 50 mm insulated short-bevel needle was advanced in plane from lateral to medial. The initial deposition was made at the intersection of the first rib and the subclavian artery, the so-called corner pocket, and the remainder of the study solution was deposited by repositioning the needle within the neural cluster under continuous vision, with intermittent aspiration before each incremental injection and continuous observation of the pattern of spread. The total volume of 30 mL was administered over approximately three minutes. Needle insertion to end of injection time was recorded.

Assessment of Block and Outcome Definitions: Sensory blockade was assessed every two minutes for the first 30 minutes in the distributions of the median, ulnar, radial and musculocutaneous nerves using a three-point scale, where 0 denoted normal sensation to pinprick, 1 loss of sensation to pinprick with preserved sensation to touch, and 2 loss of sensation to both pinprick and touch. Onset of sensory blockade was defined as the interval from the end of injection to the attainment of a score of 1 or above in all four nerve distributions, and duration of sensory blockade as the interval from onset to the return of a score of 0 in any distribution.

Motor blockade was assessed on the modified Bromage scale for the upper limb, where 0 denoted full muscle power, 1 reduced power with the ability to move fingers only, and 2 complete motor blockade. Onset of motor blockade was defined as the interval from the end of injection to the attainment of a score of 1 or above, and duration as the interval from onset to complete recovery of motor power. The block was considered a failure and general anaesthesia was administered if adequate surgical anaesthesia had not been established within 30 minutes of injection.

The duration of postoperative analgesia, the primary outcome, was defined as the interval from the end of local anaesthetic injection to the first request for rescue analgesia or to the first recording of a visual analogue score of 4 or above, whichever occurred earlier. Pain was assessed on a 10 cm visual analogue scale at 2, 4, 6, 8, 12, 18 and 24 hours postoperatively. Rescue analgesia consisted of intravenous diclofenac 75 mg, repeated at a minimum interval of 12 hours, with intravenous tramadol 50 mg administered if pain persisted. Sedation was graded on the Ramsay sedation scale at 15-minute intervals intraoperatively and at hourly intervals for six hours thereafter. Heart rate and mean arterial pressure were recorded at baseline and at 5, 10, 15, 30, 60, 90 and 120 minutes. Bradycardia was defined as a heart rate below 50 beats per minute and treated with atropine 0.6 mg, and hypotension as a fall in mean arterial pressure exceeding 20 percent of baseline and treated with a fluid bolus and mephentermine 6 mg as required. Nausea, vomiting, dry mouth and any neurological complication were recorded. Patient satisfaction was assessed at 24 hours on a four-point scale as excellent, good, fair or poor.

Statistical Analysis: Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics version 26.0. The normality of continuous variables was assessed by the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean with standard deviation and compared between the two groups by the independent samples Student t test. Non-normally distributed variables were expressed as median with interquartile range and compared by the Mann-Whitney U test. Categorical variables were expressed as frequencies with percentages and compared by the Pearson chi-square test, with the Fisher exact test used where any expected cell frequency was below five. Serial haemodynamic variables were analysed by repeated-measures analysis of variance with the Greenhouse-Geisser correction applied where appropriate. A two-tailed p value below 0.05 was considered statistically significant.

Results

Table 1: Baseline demographic and surgical characteristics of the two groups (N = 60)

Variable	Group D (n = 30)	Group C (n = 30)	Test statistic	p value
Age, years (mean ± SD)	36.8 ± 11.4	38.2 ± 12.1	t = 0.46	0.647
Weight, kg (mean ± SD)	64.2 ± 9.6	65.8 ± 10.2	t = 0.62	0.535
Height, cm (mean ± SD)	164.6 ± 7.8	165.2 ± 8.1	t = 0.29	0.771
Body mass index, kg/m² (mean ± SD)	23.7 ± 3.2	24.1 ± 3.4	t = 0.47	0.641
Male sex, n (%)	19 (63.3)	21 (70.0)	$\chi^2 = 0.27$	0.602
ASA physical status II, n (%)	11 (36.7)	13 (43.3)	$\chi^2 = 0.30$	0.583
Type of surgery, n (%)			$\chi^2 = 0.68$	0.878
Forearm fracture fixation	12 (40.0)	11 (36.7)		
Wrist or hand surgery	9 (30.0)	11 (36.7)		
Humerus or elbow procedure	6 (20.0)	5 (16.7)		
Soft tissue or tendon repair	3 (10.0)	3 (10.0)		
Duration of surgery, min (mean ± SD)	84.6 ± 21.3	87.2 ± 23.8	t = 0.45	0.657
Needle insertion to end of injection, min (mean ± SD)	5.8 ± 1.2	6.1 ± 1.4	t = 0.89	0.377
Baseline heart rate, beats/min (mean ± SD)	78.4 ± 9.2	79.6 ± 9.8	t = 0.49	0.626
Baseline mean arterial pressure, mmHg (mean ± SD)	92.6 ± 8.4	93.4 ± 8.9	t = 0.36	0.722

Group D, dexmedetomidine 1 microgram/kg with ropivacaine; Group C, clonidine 1 microgram/kg with ropivacaine; SD, standard deviation; ASA, American Society of Anesthesiologists.

Table 2: Onset and duration of sensory and motor blockade (N = 60)

Block characteristic	Group D (n = 30)	Group C (n = 30)	Test statistic	p value
Onset of sensory block, min (mean ± SD)	8.4 ± 1.9	11.2 ± 2.3	t = 5.14	<0.001
Onset of motor block, min (mean ± SD)	11.6 ± 2.2	14.8 ± 2.7	t = 5.03	<0.001
Time to complete sensory block, min (mean ± SD)	14.2 ± 2.6	18.4 ± 3.1	t = 5.69	<0.001
Time to complete motor block, min (mean ± SD)	17.8 ± 3.1	22.6 ± 3.6	t = 5.53	<0.001
Duration of sensory block, min (mean ± SD)	486.3 ± 62.4	372.8 ± 55.1	t = 7.46	<0.001
Duration of motor block, min (mean ± SD)	418.7 ± 58.9	324.5 ± 51.2	t = 6.61	<0.001
Sensory block prolongation, ratio of means	1.30	1.00 (reference)	—	—
Motor block prolongation, ratio of means	1.29	1.00 (reference)	—	—

SD, standard deviation. Sensory blockade assessed on a three-point pinprick scale in the median, ulnar, radial and musculocutaneous distributions; motor blockade assessed on the modified Bromage scale for the upper limb.

Table 3: Postoperative analgesia, visual analogue scores and rescue analgesic requirement (N = 60)

Variable	Group D (n = 30)	Group C (n = 30)	Test statistic	p value
Duration of analgesia, min (mean ± SD)	612.4 ± 78.6	468.2 ± 69.3	t = 7.53	<0.001
Visual analogue score (mean ± SD)				
2 hours	0.3 ± 0.5	0.4 ± 0.6	t = 0.70	0.487
4 hours	0.6 ± 0.6	0.8 ± 0.7	t = 1.19	0.240
6 hours	0.9 ± 0.7	1.6 ± 0.9	t = 3.36	0.001
8 hours	1.4 ± 0.7	2.9 ± 1.0	t = 6.72	<0.001
12 hours	2.1 ± 0.8	3.4 ± 1.1	t = 5.24	<0.001
18 hours	2.6 ± 0.9	3.3 ± 1.0	t = 2.85	0.006
24 hours	2.8 ± 0.9	3.2 ± 1.0	t = 1.63	0.109
24-hour diclofenac equivalent, mg (mean ± SD)	96.7 ± 32.4	141.3 ± 38.6	t = 4.84	<0.001
More than one rescue dose, n (%)	8 (26.7)	19 (63.3)	$\chi^2 = 8.15$	0.004
Supplementary tramadol required, n (%)	2 (6.7)	7 (23.3)	Fisher exact	0.145

SD, standard deviation. Duration of analgesia defined as the interval from the end of local anaesthetic injection to the first request for rescue analgesia or the first visual analogue score of 4 or above. 95% confidence interval for the between-group difference in duration of analgesia 105.9 to 182.5 minutes.

Table 4: Serial heart rate and mean arterial pressure following block (N = 60)

Time point	HR Group D (beats/min)	HR Group C (beats/min)	MAP Group D (mmHg)	MAP Group C (mmHg)
Baseline	78.4 ± 9.2	79.6 ± 9.8	92.6 ± 8.4	93.4 ± 8.9
5 min	76.2 ± 8.9	78.1 ± 9.4	91.4 ± 8.1	92.6 ± 8.6
10 min	73.6 ± 8.6	76.4 ± 9.1	89.8 ± 7.9	91.2 ± 8.4
15 min	70.8 ± 8.4	74.2 ± 8.8	88.2 ± 7.6	90.1 ± 8.2
30 min	66.4 ± 8.2*	71.8 ± 8.9	86.4 ± 7.4	88.6 ± 8.1
60 min	64.8 ± 7.9*	70.6 ± 8.4	85.9 ± 7.2	87.9 ± 7.8
90 min	65.2 ± 8.1*	71.2 ± 8.6	86.8 ± 7.1	88.4 ± 7.6
120 min	67.6 ± 8.4	72.4 ± 8.8	88.1 ± 7.3	89.6 ± 7.9

Values are mean ± standard deviation. HR, heart rate; MAP, mean arterial pressure. *$p < 0.05$ for between-group comparison at that time point by unpaired t test. Repeated-measures analysis of variance: group by time interaction for HR $F = 4.36$, $p = 0.004$; for MAP $F = 1.84$, $p = 0.126$.

Table 5: Sedation and adverse effects (N = 60)

Variable	Group D (n = 30)	Group C (n = 30)	Test statistic	p value
Ramsay sedation score ≥3, n (%)	17 (56.7)	8 (26.7)	$\chi^2 = 5.55$	0.018
Peak Ramsay score, median (IQR)	3 (2–3)	2 (2–3)	U = 285.5	0.011
Ramsay score ≥5, n (%)	0 (0.0)	0 (0.0)	—	—
Bradycardia requiring atropine, n (%)	5 (16.7)	1 (3.3)	Fisher exact	0.195
Hypotension requiring intervention, n (%)	4 (13.3)	2 (6.7)	Fisher exact	0.671
Dry mouth, n (%)	6 (20.0)	4 (13.3)	$\chi^2 = 0.48$	0.488
Nausea, n (%)	2 (6.7)	3 (10.0)	Fisher exact	1.000
Vomiting, n (%)	1 (3.3)	2 (6.7)	Fisher exact	1.000
Desaturation below 94%, n (%)	0 (0.0)	0 (0.0)	—	—
Local anaesthetic systemic toxicity, n (%)	0 (0.0)	0 (0.0)	—	—
Pneumothorax, n (%)	0 (0.0)	0 (0.0)	—	—
Persistent neurological deficit at day 7, n (%)	0 (0.0)	0 (0.0)	—	—

IQR, interquartile range. Bradycardia defined as heart rate below 50 beats per minute; hypotension defined as a fall in mean arterial pressure exceeding 20% of baseline.

Table 6: Block success and patient satisfaction at 24 hours (N = 60)

Variable	Group D (n = 30)	Group C (n = 30)	Test statistic	p value
Successful block without supplementation, n (%)	29 (96.7)	28 (93.3)	Fisher exact	1.000
Local infiltration supplementation, n (%)	1 (3.3)	1 (3.3)	Fisher exact	1.000
Conversion to general anaesthesia, n (%)	0 (0.0)	1 (3.3)	Fisher exact	1.000
Patient satisfaction grade, n (%)				
Excellent	18 (60.0)	9 (30.0)		
Good	9 (30.0)	11 (36.7)		
Fair	3 (10.0)	10 (33.3)		
Poor	0 (0.0)	0 (0.0)		
Excellent or good, n (%)	27 (90.0)	20 (66.7)	$\chi^2 = 4.81$	0.028
Willing to receive same technique again, n (%)	28 (93.3)	24 (80.0)	Fisher exact	0.254

Group D, dexmedetomidine 1 microgram/kg with ropivacaine; Group C, clonidine 1 microgram/kg with ropivacaine.

Participant flow and baseline characteristics: Sixty-eight patients were assessed for eligibility, of whom eight were excluded, five because they did not meet the inclusion criteria and three because consent was declined. Sixty participants were randomised, 30 to Group D and 30 to Group C. One participant in Group C required conversion to general anaesthesia for an inadequate block and

one participant in Group D required supplementation of the ulnar territory with local infiltration by the surgeon; both were retained in the analysis on an intention-to-treat basis, with block duration data censored at the time of supplementation. The two groups were comparable in age (36.8 ± 11.4 years in Group D versus 38.2 ± 12.1 years in Group C, t = 0.46, p = 0.647), weight (64.2 ± 9.6 kg versus 65.8 ± 10.2 kg, t = 0.62, p = 0.535), sex distribution (χ^2 = 0.27, p = 0.602) and physical status (χ^2 = 0.30, p = 0.583). The distribution of surgical procedures, the mean duration of surgery and the needle insertion to end of injection time did not differ significantly between the groups. Baseline characteristics are presented in Table 1.

Block Characteristics: Dexmedetomidine produced a significantly faster onset of both sensory and motor blockade. The mean time to onset of sensory blockade was 8.4 ± 1.9 minutes in Group D compared with 11.2 ± 2.3 minutes in Group C (t = 5.14, p < 0.001), and the mean time to onset of motor blockade was 11.6 ± 2.2 minutes and 14.8 ± 2.7 minutes respectively (t = 5.03, p < 0.001). The time to attainment of complete sensory blockade, defined as a score of 2 in all four nerve distributions, was 14.2 ± 2.6 minutes in Group D and 18.4 ± 3.1 minutes in Group C (t = 5.69, p < 0.001).

The duration of blockade was likewise significantly prolonged with dexmedetomidine. Sensory blockade persisted for 486.3 ± 62.4 minutes in Group D compared with 372.8 ± 55.1 minutes in Group C, a mean difference of 113.5 minutes (t = 7.46, p < 0.001). Motor blockade persisted for 418.7 ± 58.9 minutes and 324.5 ± 51.2 minutes respectively, a mean difference of 94.2 minutes (t = 6.61, p < 0.001). Expressed as a ratio, dexmedetomidine prolonged sensory blockade by 30.4 percent and motor blockade by 29.0 percent relative to clonidine. Block characteristics are set out in Table 2.

Postoperative Analgesia: The duration of postoperative analgesia, the primary outcome, was 612.4 ± 78.6 minutes in Group D and 468.2 ± 69.3 minutes in Group C, a mean difference of 144.2 minutes or approximately two and a half hours (t = 7.53, 95 percent confidence interval for the difference 105.9 to 182.5 minutes, p < 0.001). Visual analogue scores were significantly lower in Group D from the eighth postoperative hour onwards. At 8 hours the mean score was 1.4 ± 0.7 in Group D and 2.9 ± 1.0 in Group C (t = 6.72, p < 0.001), at 12 hours 2.1 ± 0.8 and 3.4 ± 1.1 respectively (t = 5.24, p < 0.001), and at 18 hours 2.6 ± 0.9 and 3.3 ± 1.0 (t = 2.85, p = 0.006). By 24 hours the difference was no longer significant (2.8 ± 0.9 versus 3.2 ± 1.0, t = 1.63, p = 0.109). Scores at 2, 4 and 6 hours were uniformly low in both groups and did not differ.

Total rescue analgesic consumption over the first 24 postoperative hours, expressed as diclofenac equivalent, was 96.7 ± 32.4 mg in Group D and 141.3 ± 38.6 mg in Group C (t = 4.84, p < 0.001). The proportion of participants requiring more than one dose of rescue analgesic was 8 of 30 (26.7 percent) in Group D and 19 of 30 (63.3 percent) in Group C (χ^2 = 8.15, p = 0.004). Supplementary tramadol was required by 2 participants (6.7 percent) in Group D and 7 participants (23.3 percent) in Group C (Fisher exact test, p = 0.145). Analgesic outcomes appear in Table 3.

Haemodynamic Profile: Repeated-measures analysis of variance demonstrated a significant group by time interaction for heart rate (F = 4.36, p = 0.004) but not for mean arterial pressure (F = 1.84, p = 0.126). Heart rate declined in both groups from baseline, with a greater and more sustained reduction in Group D. Mean heart rate was significantly lower in Group D at 30 minutes (66.4 ± 8.2 versus 71.8 ± 8.9 beats per minute, t = 2.44, p = 0.018), at 60 minutes (64.8 ± 7.9 versus 70.6 ± 8.4 beats per minute, t = 2.75, p = 0.008) and at 90 minutes (65.2 ± 8.1 versus 71.2 ± 8.6 beats per minute, t = 2.78, p = 0.007). Mean arterial pressure fell modestly in both groups without significant between-group difference at any time point. Serial haemodynamic data are presented in Table 4.

Sedation and Adverse Effects: A Ramsay sedation score of 3 or above at any point during the observation period was recorded in 17 of 30 participants (56.7 percent) in Group D and 8 of 30 participants (26.7 percent) in Group C (χ^2 = 5.55, p = 0.018).

The peak median Ramsay score was 3 (interquartile range 2 to 3) in Group D and 2 (interquartile range 2 to 3) in Group C (U = 285.5, p = 0.011). No participant in either group attained a score of 5 or 6, and no participant developed respiratory depression, desaturation below 94 percent, or required airway intervention.

Bradycardia requiring atropine occurred in 5 participants (16.7 percent) in Group D and 1 participant (3.3 percent) in Group C, a difference that did not reach statistical significance (Fisher exact test, p = 0.195). Hypotension requiring intervention occurred in 4 participants (13.3 percent) and 2 participants (6.7 percent) respectively (Fisher exact test, p = 0.671). Dry mouth was reported by 6 participants (20.0 percent) in Group D and 4 participants (13.3 percent) in Group C (χ^2 = 0.48, p = 0.488). Nausea and vomiting were infrequent and comparable between the groups. No participant in either group developed features of local anaesthetic systemic toxicity, pneumothorax, Horner syndrome requiring

intervention, or any persistent neurological deficit at follow-up on the seventh postoperative day. Sedation and adverse effect data appear in Table 5.

Block Success and Patient Satisfaction: The block was successful without supplementation in 29 of 30 participants (96.7 percent) in Group D and 28 of 30 participants (93.3 percent) in Group C (Fisher exact test, p = 1.000). Patient satisfaction at 24 hours was graded excellent or good by 27 of 30 participants (90.0 percent) in Group D and 20 of 30 participants (66.7 percent) in Group C ($\chi^2 = 4.81$, p = 0.028). No participant in either group graded satisfaction as poor. Success and satisfaction data are shown in Table 6.

Discussion

This randomised, double-blind comparison found that dexmedetomidine 1 microgram per kilogram was superior to clonidine at an identical weight-based dose when added to 0.5 percent ropivacaine for ultrasound-guided supraclavicular brachial plexus block. Dexmedetomidine hastened the onset of both sensory and motor blockade by approximately three minutes, prolonged sensory blockade by around 30 percent and motor blockade by around 29 percent, and extended the duration of postoperative analgesia by roughly two and a half hours, with a corresponding reduction of nearly one third in 24-hour rescue analgesic consumption. These benefits were accompanied by greater sedation and a numerically higher incidence of bradycardia, neither of which required more than simple intervention in any participant.

The direction and magnitude of these findings agree closely with the pooled evidence. El-Boghdadly and colleagues, in a systematic review and meta-analysis restricted to randomised trials comparing perineural dexmedetomidine with clonidine in supraclavicular blockade, reported ratios of means of approximately 1.2 for the duration of sensory blockade, motor blockade and analgesia in favour of dexmedetomidine, together with hastened onset of both sensory and motor blockade.[7] The prolongation observed here, at 1.30 for sensory and 1.29 for motor blockade, is at the upper end of that pooled estimate but well within the range of the constituent trials. The randomised study of Swami and colleagues, which compared the two agents in supraclavicular block, likewise found faster onset and significantly longer duration of both blockade and analgesia with dexmedetomidine.[6] Tripathi and colleagues, using bupivacaine as the base local anaesthetic, reached the same conclusion.[8]

The mechanistic basis for the superiority of dexmedetomidine is probably twofold. The greater alpha-2 selectivity of dexmedetomidine, roughly eightfold that of clonidine, means that a given weight-based dose delivers substantially greater alpha-2 agonism, so that the comparison performed here, and in most of the published literature, is not a comparison at equipotent doses. This is an important qualification, since part of the observed advantage may reflect a dose difference in pharmacological terms rather than an intrinsic superiority of the molecule. The second component is the mechanism identified experimentally by Brummett and colleagues, namely blockade of the hyperpolarisation-activated cation current, which prolongs the recovery of the axon to its resting potential and is not shared to the same degree by clonidine.[2] The observation that dexmedetomidine hastened onset as well as prolonging duration favours a genuine local action rather than a purely systemic alpha-2 effect, since a systemically mediated analgesic action would not be expected to accelerate the establishment of conduction blockade.

Not all published evidence is uniformly favourable, and the safety findings deserve particular attention. Vorobeichik and colleagues, reviewing the evidence basis for perineural dexmedetomidine in brachial plexus blockade, concluded that the agent enhances block quality but drew attention to a clinically relevant increase in transient bradycardia, hypotension and sedation, and noted the difficulty of separating perineural from systemic effects in trials that lack a systemic control arm.[5] Abdallah and Brull reached similar conclusions and emphasised the dose dependence of the adverse effect profile.[4] Schnabel and colleagues, in a meta-analysis with trial sequential analysis, confirmed the efficacy signal while cautioning that the evidence for safety endpoints remained underpowered.[9] The present study, with 30 participants per group, is likewise underpowered for adverse events, and the numerically higher incidence of bradycardia in the dexmedetomidine group, 16.7 percent against 3.3 percent, did not reach statistical significance but is directionally consistent with the pooled estimates and should not be dismissed. The higher sedation scores with dexmedetomidine are unambiguous and were statistically significant. In an inpatient orthopaedic setting this is arguably a benefit rather than a harm, but it is a consideration in day-care practice where early discharge is intended, and it complicates the interpretation of patient-reported pain scores in an unblinded patient.

The choice of dexamethasone as an alternative adjuvant merits comment, since the comparison between alpha-2 agonists is not the only one available to the clinician. Choi and colleagues established that perineural dexamethasone prolongs brachial plexus blockade, and the indirect meta-analysis of Albrecht and colleagues concluded that dexamethasone is superior to dexmedetomidine as a perineural adjunct for supraclavicular

block.[10,11] The network meta-analysis of Sehmbi and colleagues, which examined perineural and intravenous administration of both agents, reached a broadly concordant conclusion while noting that the intravenous route achieves much of the benefit of the perineural route for dexamethasone.[12] These findings do not invalidate the present comparison, which addressed the choice between the two alpha-2 agonists that remain in widest use in Indian practice, but they indicate that the alpha-2 agonists may not represent the optimal adjuvant class and that a three-way comparison would be informative.

The absence of a significant difference in mean arterial pressure, despite the significant difference in heart rate, is consistent with the biphasic haemodynamic profile of alpha-2 agonists, in which peripheral alpha-2B mediated vasoconstriction partially offsets the centrally mediated sympatholysis.

Koraki and colleagues reported a comparable pattern when dexmedetomidine was added to ropivacaine in supraclavicular block, with a modest reduction in heart rate but preserved arterial pressure.[13] The block success rate exceeded 93 percent in both groups, which is consistent with contemporary ultrasound-guided series and reflects the reliability conferred by direct visualisation, and no participant developed pneumothorax or local anaesthetic systemic toxicity, in keeping with the improved safety profile attributed to ultrasound guidance for the supraclavicular approach.

Limitations

Several limitations should be acknowledged. The study was conducted at a single centre with a modest sample size calculated for the primary analgesic endpoint and is consequently underpowered to detect differences in the incidence of bradycardia, hypotension and other adverse events. The two agents were compared at an identical weight-based dose rather than at equipotent doses, and the observed superiority of dexmedetomidine may therefore be partly attributable to greater effective alpha-2 agonism rather than to any intrinsic property of the molecule. No systemic control group was included, so the study cannot determine what proportion of the observed benefit was mediated perineurally as opposed to by systemic absorption, a limitation shared with the great majority of the published literature in this field. Only a single dose of each agent was studied, precluding any inference about dose-response. Follow-up for neurological complications extended to the seventh postoperative day, which is adequate for the detection of clinically evident deficit but insufficient to exclude rare late sequelae. Finally, the greater sedation produced by dexmedetomidine may have influenced patient-reported visual analogue scores and satisfaction ratings, an unavoidable consequence of comparing agents that differ in sedative potency. Adequately powered multicentre trials using equipotent dosing and incorporating a systemic control arm, and preferably a dexamethasone comparator, are needed to define the optimal perineural adjuvant for ropivacaine-based brachial plexus blockade.

Conclusion

Dexmedetomidine 1 microgram per kilogram added to 0.5 percent ropivacaine in ultrasound-guided supraclavicular brachial plexus block produced a faster onset of sensory and motor blockade, a longer duration of sensory and motor blockade, and a significantly longer duration of postoperative analgesia than clonidine at the same weight-based dose. The duration of analgesia was extended by approximately 144 minutes and 24-hour rescue analgesic consumption was reduced by nearly one third, with a higher proportion of participants requiring only a single dose of rescue analgesia and higher overall patient satisfaction.

These advantages were obtained at the cost of significantly greater sedation and a numerically higher incidence of transient bradycardia, neither of which required more than simple intervention in any participant, and neither of which was associated with respiratory depression or any lasting sequel. Dexmedetomidine can therefore be recommended in preference to clonidine as an alpha-2 agonist adjuvant to ropivacaine for supraclavicular brachial plexus block in the inpatient setting, with the qualification that heart rate should be monitored during the first two hours and that the additional sedation requires consideration where early discharge is planned. Because the two agents were compared at an identical weight-based rather than an equipotent dose, and because dexamethasone may offer a superior alternative, comparative studies employing equipotent alpha-2 dosing and a dexamethasone comparator remain warranted.

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