

Dexmedetomidine's Pain Relief Safety and Effectiveness in Brachial Plexus Block in Supraclavicular Area: A Prospective Study

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Abstract

Background: The objective of this research was to see how Dexmedetomidine affected the onset and duration of brachial plexus block and postoperative analgesia in individuals having upper limb procedures. **Methods:** This quasi-experimental study was conducted in the Department of Anaesthesia, Rajshahi Medical College Hospital, and Tertiary Hospital in Rajshahi, Bangladesh. From June 2018 to December 2020. There were two groups of (n=60) adult patients randomly assigned to upper-limb and hand procedures each. When it came to the control group, they were given the same amount of Dexmedetomidine (dexmed) as the dexmedetomidine (dexmed) group. Still, they were also given the same amount of 0.75% bupivacaine and 2% lidocaine (with adrenaline) as the dexmedetomidine (dexmed) 1 µg/kg. Ultrasound-guided supraclavicular brachial plexus block administered a total of 0.5 mL/kg in both groups. In addition to hemodynamic stability, patients were monitored for postoperative pain, analgesia duration, and side effects. **Results:** The dexmed group had a faster onset of motor blockade and a longer duration of sensory and motor blockade ($P < 0.0001$). For the dexmed group, the postoperative analgesic period was also prolonged compared to that of controls 12 [10.5–13.5] hours and 17 [10.5–19.5] hours in the control and dexmed group, respectively [95% confidence interval, -5 {-5, -4}, $P < 0.0001$). Within the first 24 hours following surgery, the dexmed group used rescue analgesics less frequently. ($P < 0.0001$). Except for 8 and 10 hours after surgery, postoperative pain scores were equivalent between groups when pain levels were lower in the dexmed group. More sedated individuals in the dexmedetomidine group remained sedated for 2 hours longer. ($P < 0.0001$). We did not have any occurrences of bradycardia, hypotension, respiratory depression, or disorientation. **Conclusions:** According to our findings, adding Dexmedetomidine to Bupivacaine-lidocaine increased the duration of the supraclavicular brachial plexus block and reduced postoperative analgesia in patients with upper limb surgery.

Keyword: Supraclavicular Block, Dexmedetomidine, Brachial Plexus Block, Rajshahi Medical College.

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INTRODUCTIONS

The supraclavicular brachial plexus block may be done alone or in combination with general anaesthesia to manage postoperative pain in patients following upper extremity surgery[1]. With the potential for deleterious effects, [2] a variety of adjuvants have been added to local anaesthetics in an attempt to prolong block [3] duration and postoperative analgesia while also increasing patient comfort. [4, 5].

The efficacy of α_2 -adrenoceptor in several regional anaesthetic procedures and agonists has been developed [6]. After a brachial plexus block, clonidine increased the duration of anaesthesia and analgesia, although long-acting local anaesthetics have had mixed effects [7, 8].

There's a specific receptor activator in dexmedetomidine α_2 -adrenoceptor agonist with an eight

times higher potency than clonidine [9]. Dexmedetomidine was found to be superior to clonidine in terms of anaesthesia quality, tourniquet tolerance, and postoperative analgesia in a study comparing the effects of Dexmedetomidine and clonazepam added to bupivacaine all through Bier's block [10]. When used in peripheral nerve blocks, Dexmedetomidine is safe and effective when combined with long-acting local anaesthetics [11]. Dexmedetomidine was administered intrathecally or perineurally without causing any abnormalities on the histopathology report [12]. It's been proven in several clinical investigations that administering Dexmedetomidine together with neuraxial and peripheral nerve blocks will extend the sensory and motor blockade. Dexmedetomidine [13, 14].

Only a few studies have looked at Dexmedetomidine in brachial plexus block, however. For the purpose of this research, the researchers wanted to see how Dexmedetomidine, combined with bupivacaine and lidocaine, would affect the start and duration of the supraclavicular brachial plexus block, as well as the analgesia experienced by patients after that.

METHODS

This quasi-experimental study was conducted in the Department of Anaesthesia, Rajshahi Medical College Hospital, and Tertiary Hospital in Rajshahi, Bangladesh. From March 2018 to October 2020. A total (n=60) of the patient (20–60 years old) with ASA physical status I and II, scheduled for upper limb and hand procedures, were enrolled after acquiring written informed permission from the patients. This research did not include patients using an adrenal receptor agonist or antagonist or had any upper-limb neurologic condition, history of cardiac disease, renal or liver failure, allergy to local anaesthetic. Patients were instructed not to eat or drink anything solid; liquids could be consumed two hours before the operation.

A computer-generated random number table was used to assign patients at random to one of two groups. Seals on opaque envelopes contained the group allotment information, only revealed immediately before the block was administered. The control group received equal volumes of 0.75% ropivacaine and 2% lidocaine with adrenaline (1:2,00,000). In contrast, the dexmed group received 1 µg/kg of Dexmedetomidine and equal volumes of 0.75% ropivacaine and 2% lidocaine with adrenaline. Injections of 0.5 mL/kg up to 40 mL were given by an anesthesiologist who was not engaged in the data gathering process.

According to the previously mentioned procedure, a supraclavicular block was administered to the patient. 19 The patient was positioned supine with the head tilted 45 degrees to the contralateral side during the procedure. Ultrasound was used to see the brachial plexus after the skin was sterilized in the

supraclavicular fossa. With lidocaine anaesthetizing the skin and subcutaneous tissue, the outer end of a 22-gauge 50-mm needle (B. Braun, Germany) was attached to the probe and advanced along the long axis until it was lateral to the round pulsating, hypoechoic subclavian artery on the top of the hyperechoic, hyperechoic first rib.

The needle was removed. Neurostimulation was used to confirm the correct needle location, and ultrasonography was used to guide the injection of the study medication. Attempts were counted, and block performance time was logged (the time it took from probe placement to the conclusion of the local anaesthetic administration). In addition to phrenic nerve palsy, vascular puncture and Horner's syndrome were reported as complications. Anesthesiologists who have conducted at least 10 blocks using the research approach completed all of the blocks before starting the study. The anesthesiologist executed the block, and the patients had no idea which group they were in.

At baseline, when the block was administered, and then every 5 minutes until the procedure was completed, patients' heart rates (HRs), noninvasive blood pressures (NIBPs), and oxygen saturation levels were monitored. Any time there is a drop in blood pressure (20 % decrease in mean arterial pressure in relation to baseline values), bradycardia (HR < 50 beats/min), or hypoxemia (Spo2 <90%) was recorded. Sensory block was assessed by pinprick sensation using a 3-point scale (0–2, 0 = normal sensation, 1 = decreased pain sensation to pinprick, 2 = loss of pain sensation to pinprick) in the median, ulnar, radial, and musculocutaneous nerve locations every 5 minutes for 30 minutes. At regular intervals, finger abduction (with the radial nerve) was evaluated, as was thumb and fifth finger pinching with the median nerve, and thumb and second finger pinching with the ulnar nerve. The onset of sensory and motor block was timed (from the moment the block was administered until all four nerve areas lost pain feeling), and the results were compared. After the local anaesthetic injection, a successful block was defined as total sensory and motor blockade in all locations tested within 30 minutes. Local anaesthetic infiltration was used to prevent surgical site anaesthetic failure.

A local anaesthetic was administered to ease the patient's discomfort if any arose during the procedure. (2% lidocaine with adrenaline) infiltration or IV fentanyl 1 to 2 µg/kg. Patient comfort and the necessity for block supplementation were classified as surgical efficacy. Until the block was resolved, sensory and motor function was tested every 30 minutes following surgery. The duration of the sensory block was determined by counting the time elapsed from the time the block was administered and when the anaesthetic on all nerves was completely resolved. The duration of the motor block was determined by

measuring the time it took from when the block was administered until the hand and forearm had fully recovered their motor function.

After surgery, patients were observed by an anesthesiologist who was unaware of their group assignment for the next 24 hours in the post-anaesthesia care unit. Each half-hour for the first two hours, every hour thereafter, and again at 24 hours post-op monitored heart rate and blood pressure. Postoperative pain was also measured. (VAS, 0–10, 0 = no pain, 10 = maximum imaginable pain). Injection diclofenac 1 mg/kg IV was given if VAS was >4. Diclofenac was provided if the pain persisted after 30 minutes; 1 mg/kg IV tramadol was delivered. In the 24 hours following surgery, the duration of analgesia (time between delivery of block and the first rescue analgesia) and total analgesic demand were recorded.

In order to determine the level of postoperative sedation, researchers used a 4-point scale (1 = wide awake; 2 = mild sedation, responding to verbal command; 3 = moderate sedation, responding to glabellar tap; 4 = deep sedation, responding to a deep painful stimulus). Patients were closely observed following surgery in case they developed symptoms of postoperative nausea and vomiting (PONV). 4-point objective score (1 = no PONV; 2 = mild nausea, no vomiting; 3 = excessive nausea or vomiting; 4 = vomiting ≥ 2 times). Dexmedetomidine's adverse effects were noted, such as dry mouth, hypotension, bradycardia, and disorientation. Patients were monitored for any signs of neurologic dysfunction for a total of seven days.

STATISTICAL ANALYSIS

SAS 9.4 was used to do the statistical analysis (SAS Institute Inc.). The classified or skewed data were compared using the Mann-Whitney U test, whilst the normally distributed data were compared with independent 2-sample t-tests. An ANOVA with repeated measurements was used to compare HR and blood pressure, and then an ANOVA with Bonferroni correction was used as a post hoc analysis. This study used the Mann-Whitney U test to compare block onset and duration, analgesia duration, pain score and sedation score. Sensory block, motor block, and

analgesia duration were all included in the survival study to see how long patients would survive. The Hodges-Lehmann estimate for a Wilcoxon rank-sum test, utilizing PROC NPAR1WAY of SAS 9.4, was used to construct the confidence intervals for differences between group medians. According to pilot research, the sample size was estimated using a 2-group t-test with a 0.05 2-sided significance threshold for postoperative analgesia duration, with a mean value of 800 minutes and an SD of 240 minutes. A clinically significant difference was defined as a 25% variation in the time required for postoperative analgesia. It was necessary to have 27 patients in each group to achieve a power of 0.9. the trial included 60 patients in total.

RESULTS

The study had 54 patients who all finished it. In the control group, two patients declined regional anaesthetic, while one patient received dexmed and was returned home on the same day as surgery, making them ineligible for the study. For this reason, there were 27 patients in each group.

In terms of demographics and operation time, the groups were comparable (Table 1). Reconstructive operations on the forearm and hand were the most common in both patient groups. The time it took to complete a block and the number of times it attempted were comparable between groups. Two individuals in the dexmed group required block augmentation with local anaesthetics after 30 minutes due to insufficient blocks in the dexmed group. Two of the patients in the control group were extremely nervous and required intraoperative midazolam augmentation. Fentanyl or block supplementation was not needed by any of the patients in either group.

The onset period of motor block was shortened when Dexmedetomidine was added to a ropivacaine-lidocaine solution (Table 2). The dexmed group's sensory and motor block durations were considerably greater than those of the control group. ($P < 0.0001$). As with postoperative analgesia, the dexmed group's time on dexmed was longer than the control group's, 12 [10.5–13.5] hours and 17 [10.5–19.5] the time spent in the dexmed and control groups, respectively, [95% confidence interval $-5 \{-5, -4\}$, $P < 0.0001$).

Table-1: Demographic Data

	Control group	Dexmed group
	(n = 27)	(n = 27)
Age (y), mean \pm SD	30.41 \pm 11.90	33.81 \pm 10.64
Height (cm), mean \pm SD	165.37 \pm 3.23	161.19 \pm 4.51
Weight (kg), mean \pm SD	59.93 \pm 8.29	62.52 \pm 9.22
Sex ratio (M:F), n	21:6	19:8
Duration of surgery (min), mean \pm SD	84.04 \pm 34.12	89.63 \pm 31.10
Block performance time (min), mean \pm SD	5.93 \pm 0.68	5.78 \pm 1.09

Dexmed = dexmedetomidine.

Table-2: Onset Time and Duration of Block and Postoperative Analgesia

		Control group (n = 27)	Dexmed group (n = 27)	95% CI of median difference ^a	P value
Onset of	sensory block, min, median (IQR)	10 (10–10)	10 (10–10)	—	0.129
Onset of	motor block, min, median (IQR)	15 (15–15)	10 (10–10)	5 (5, 5)	<0.0001
Duration	of sensory block, h, median (IQR)	11 (8.5–11.5)	15 (15–15)	-4 (-5, -4)	<0.0001
Duration	of motor block, h, median (IQR)	10 (7.5–10.5)	13 (10.5–13.5)	-3 (-3, -3)	<0.0001
Duration	of analgesia, h, median (IQR)	12 (10.5–13.5)	17 (10.5–19.5)	-5 (-5, -4)	<0.0001

Two patients in the dexmed group had an inadequate block at 30 min and required block supplementation with local anaesthetics. These 2 patients were censored for survival analysis (time to event). IQR = interquartile range; Dexmed = dexmedetomidine; CI = confidence interval.

Within 24 hours after surgery, the dexmed group used considerably less diclofenac (median [interquartile range [2, 1–3] doses vs the control group, $P < 0.0001$) than did the control group. Tramadol wasn't necessary for any of the patients in this study. Both

groups had similar postoperative pain levels, except 8 and 10 hours postoperatively, when the dexmed group's VAS values were lower than the control group's (Fig. 1).

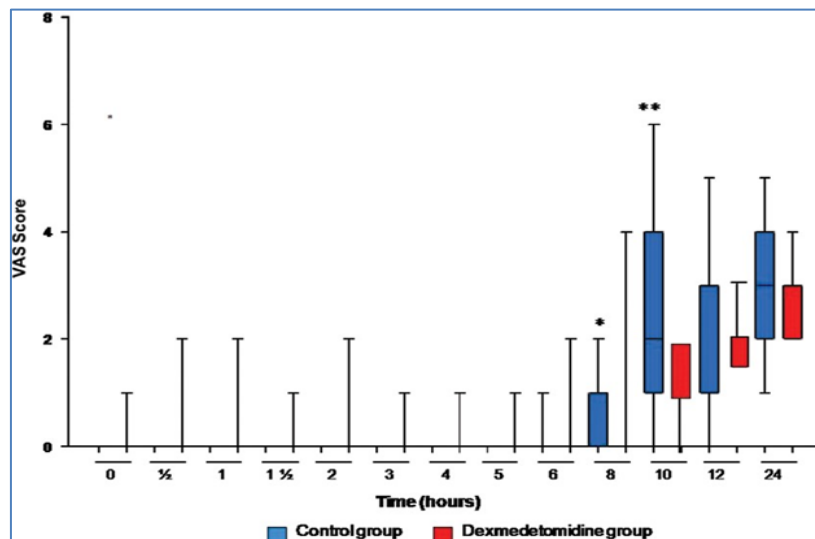


Fig-1: Both groups experienced similar levels of 24-hour postoperative visual analogue pain ratings. Median, 25th and 75th percentiles are shown in boxes (interquartile range).

The dexmed group's heart rate and systolic blood pressure were lower than those of the control group at every time point except for the baseline (Figs. 2).

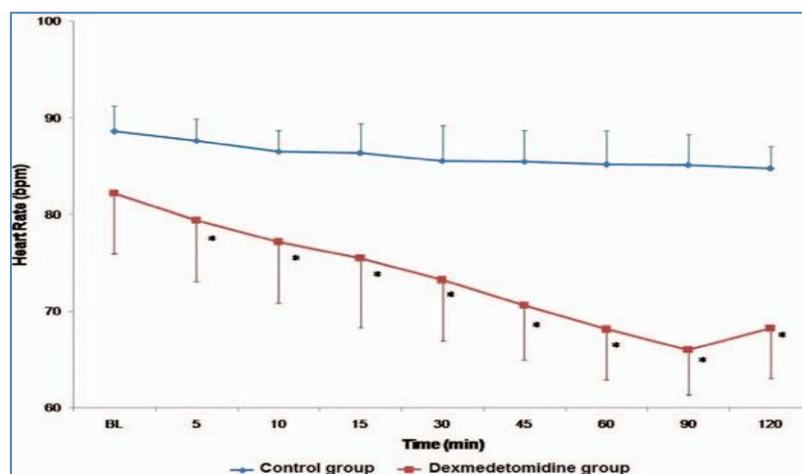


Fig-2: Heart rates during surgery (mean value) in the two groups at different periods in time. The bar indicates that the SD is present

In contrast, neither hypotension nor bradycardia was seen. It was easy to rouse the patients in the dexmed group (Fig. 3) despite being sedated for 30 minutes, 60 minutes, 90 minutes, and two hours.

There was no respiratory depression or hypoxia in any of the individuals. There was no sedation in either group after 2 hours. There was no dizziness or PONV in any of the subjects.

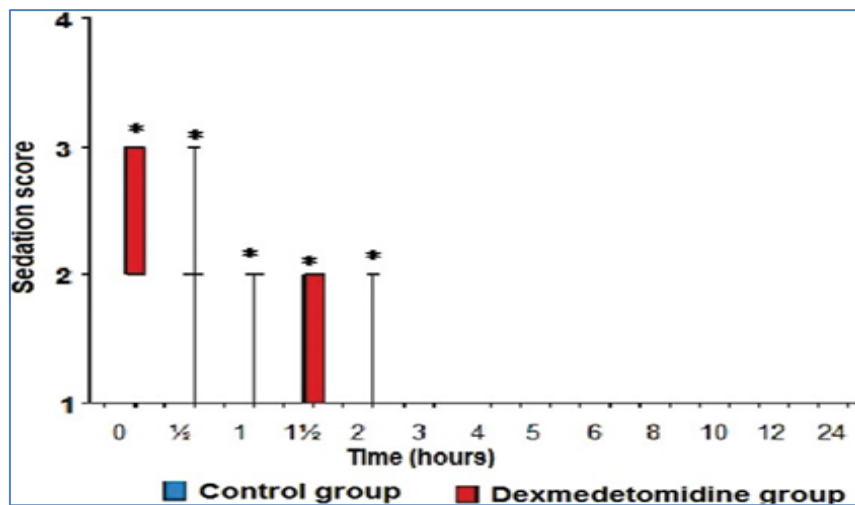


Fig-3: Both groups had similar 24-hour postoperative sedation levels. Squares indicate median, whereas whisker caps show the 10 per cent and 90 percentiles (interquartile range). The difference between the two groups was statistically significant. at 0, 1/2, 1, and 2 h, $P < 0.0001$.

DISCUSSION

In the present study, adding Dexmedetomidine to ropivacaine-lidocaine in a supraclavicular brachial plexus block increased block duration and postoperative analgesia in patients having upper limb surgery. It reduced the need for a rescue analgesic, we discovered. Additionally, dexmedetomidine-treated individuals experienced a faster onset of motor block.

Abdallah and Brull [15] found that administering Dexmedetomidine as part of a brachial plexus block significantly prolonged the duration of the motor block and increased the time until the first analgesic request, as opposed to local anaesthetic alone, in a recent meta-analysis (total 4 trials, 1 on supraclavicular block) [16–18]. When administered intrathecally, Dexmedetomidine accelerated the onset of sensory block, but not when administered perineurally. According to the results of a recent study, patients who received Dexmedetomidine had longer interscalene block duration and lower pain levels than those in the control group [19].

α_2 -adrenergic receptor agonists' mechanism of analgesia is believed to be complex. Peripherally, α_2 -agonists reduce norepinephrine release and suppress nerve fibre action potentials without affecting the α_2 -receptor, which results in analgesia. These drugs work by inhibiting substance release from dorsal root neurons and activating α_2 -adrenoceptors in the locus coeruleus to provide analgesia centrally. Dexmedetomidine's analgesic action is due to an increase in a hyperpolarization-activated cation channel, which inhibits the neuron from recovering from a

hyperpolarized state to a resting membrane potential state for further firing, according to Brummett *et al.* [11].

α_2 -agonists are known to cause hypotension and bradycardia as well as other harmful effects. Adding 100 μg of Dexmedetomidine to 0.5 % bupivacaine produced bradycardia in 7 out of 30 patients, according to Esmaoglu *et al.* [14]. The current investigation showed that no one in the dexmed group had bradycardia or hypotension despite the decreased HRs (50–60 beats/min). A reduced dosage of Dexmedetomidine might be to blame. According to earlier trials employing a similar dosage, patients receiving Dexmedetomidine had no episodes of hypotension or bradycardia [20]. No other side effects were noted, such as dizziness, pruritus, respiratory depression, or hypoxemia.

Experimental investigations have shown that administering dexmedetomidine perineurally is completely risk-free. In research, dexmedetomidine at doses up to 40 $\mu\text{g/kg}$ was found to have no impact on nerve axons or myelin sheaths. It may even help reduce the acute perineural inflammation caused by bupivacaine without inflicting any permanent harm to the nerve. Dexmedetomidine has been used successfully in neuraxial blocks at doses up to 2 $\mu\text{g/kg}$ in people [21]. Dexmedetomidine was administered at a reduced dosage out of an abundance of caution in this trial. Patients who received Dexmedetomidine showed no signs of brain damage.

Two individuals in the dexmed group had an insufficient block. One of the patients was fat, while the other had leprosy-related nerve deformities. The two groups did not have a further vascular puncture,

Horner's syndrome, or pneumothorax. One drawback of our research was using ropivacaine and lidocaine as a single agent. a combination of local anaesthetics was proposed.

Supraclavicular Brachial Plexus Block

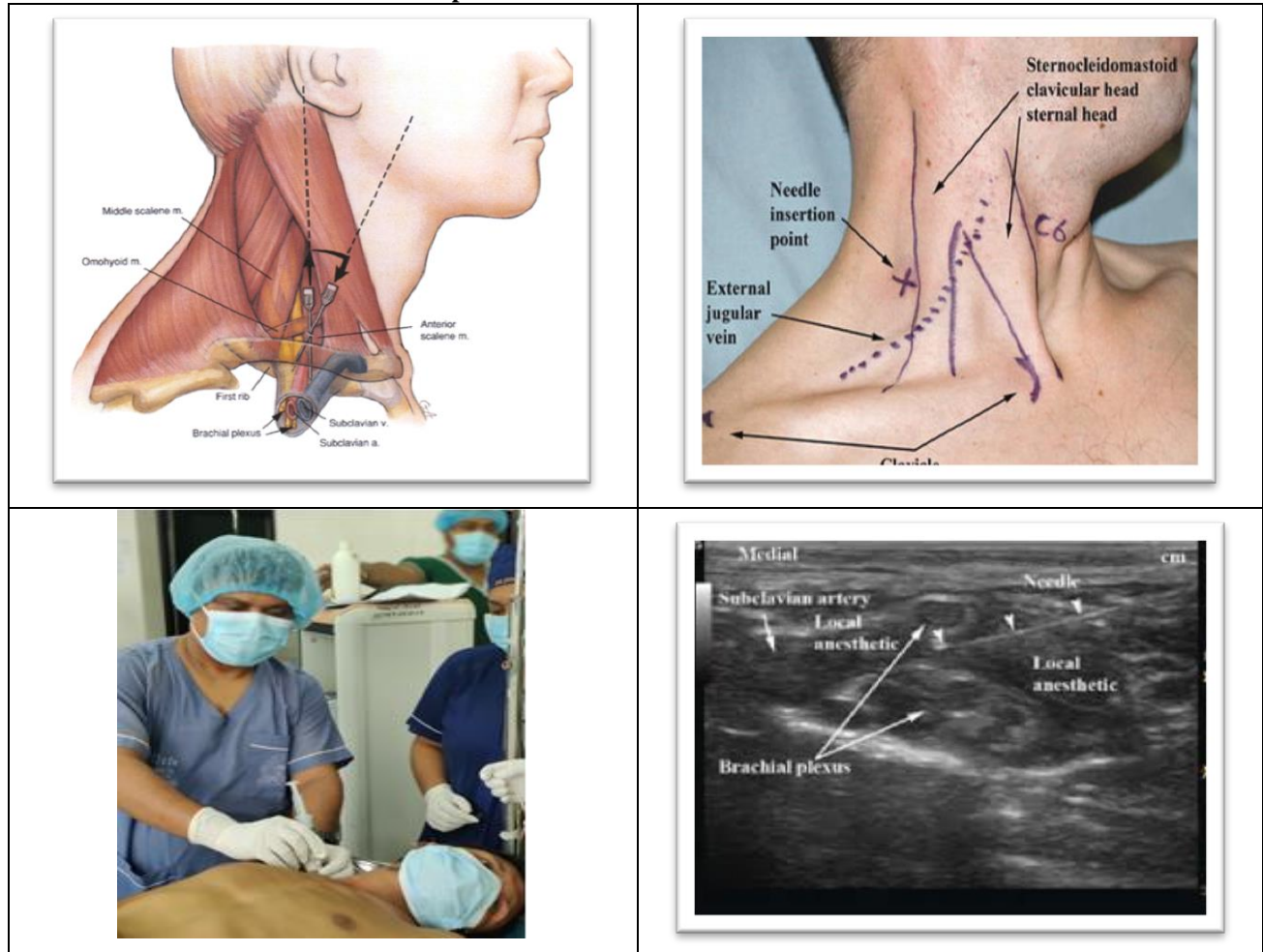


Fig-4: Supraclavicular Brachial Plexus Block Procedure

The fact that we failed to check plasma dexmedetomidine levels is another drawback of our investigation. Dexmedetomidine at the dose of (1 µg/kg) was administered intravenously for postoperative analgesia and was shown to be safe. We also avoided using systemic analgesics since experimental investigations have demonstrated that perineural Dexmedetomidine has analgesic effects only in the peripheral nervous system, not the central nervous system [22]. Similar findings were found in human volunteer research that was conducted very recently.

CONCLUSIONS

According to our findings, incorporating dexmedetomidine (1 µg/kg) to local anesthetics in supraclavicular block prolongs block duration and postoperative analgesia. Even though the dexmed group

had lower HR and blood pressure and more sedation, there were no severe side effects. Other peripheral nerve blocks with Dexmedetomidine require more research to confirm their effectiveness and safety.

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