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Original Research Article

A comparative study of intravenous versus perineural administration of dexmedetomidine in supraclavicular brachial plexus block using 0.75% ropivacaine by ultrasound guided technique in upper limb surgeries

Fahad Khan*, V. P. Singh

Department of Anaesthesia and Critical Care, Subharti Medical College, Meerut, Uttar Pradesh, India

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***Correspondence:**
Dr. Fahad Khan,
E-mail: Fahad.k.007@gmail.com

ABSTRACT

Background: Comparative study of intravenous versus perineural administration of dexmedetomidine in supraclavicular brachial plexus block using 0.75% ropivacaine by ultrasound guided technique in upper limb surgeries.
Methods: Patients in the age group 18-58 years both male and female, having ASA 1 and ASA 2, scheduled for elective surgery of unilateral upper limb surgeries were included and randomly divided into three groups' i.e. group RD, group RDI and group R and patients with chronic pain or taking any analgesics, ASA grade III and IV, bleeding disorders, history of brachial plexus injury, known allergy to the study drug, previous shoulder surgery, any psychiatric disorders, peripheral neuropathy, failed block, significant respiratory disease, hearing impairment, pregnant women, study were excluded.
Results: Time to sensory onset in group RD was as compared to group RDI and group R was found statistically significant ($p<0.001$). Duration of sensory block (analgesia) in group RD, group RDI and Group R was also statistically significant ($p<0.001$). The level of sedation of Group RDI and Group RD had highly significant value till 30 mins ($p<0.001$).
Conclusions: The central effects of dexmedetomidine also play some role in prolongation of sensory and motor block duration, as explained previously.

Keywords: Anesthesia, ASA Grade I/ II, Hemodynamic parameters

INTRODUCTION

Supraclavicular brachial plexus block is one of the most reliable and commonly performed techniques for regional anaesthesia of the upper extremity. Many additives to local anesthetics have been investigated including opioid and non opioid agentsin an attempt to increase the duration of the block in order to improve postoperative pain.[1-4]

Dexmedetomidine possesses analgesic properties, does not cause respiratory depression and has many other advantageous influences that may make it a useful and safe adjunct in many diverse clinical applications.[1-2] Both hypnotic and supraspinal analgesic effects of dexmedetomidine are mediated by noradrenergic neurons.[1] In addition, suppression of activity along the descending noradrenergic pathway (responsible for modulation of nociceptive transmission) terminates propagation of pain signals, resulting in analgesia or decreased awareness of noxious stimuli.

Dexmedetomidine has some advantageous pharmacologic characteristics compared with similar sedative

medications like greater hemodynamic stability compared to clonidine (because it is more selective for α2-AR) and preservation of both baroceptor reflex and heart rate response to a pressor response. Despite the robust preclinical data available, the clinical studies of dexmedetomidine conducted to date have varied in the surgical condition, block techniques, and local anesthetics with which dexmedetomidine was combined. The addition of dexmedetomidine to ropivacaine has been limited to volunteer studies and supraclavicular blocks have not been studied much in this context. Therefore, in this study we intend to study and obtain reliable clinical data on ultrasound guided supraclavicular brachial plexus block using 0.75% ropivacaine alone and 0.75% ropivacaine with dexmedetomidine perineurally and intravenously in terms of onset and duration of sensory (duration of analgesia) and motor block, sedation levels, hemodynamic parameters and complications, if any.

There are number of studies done which conclude that dexmedetomidine can be given through various routes of administration i.e. perineurally or intravenously in supraclavicular brachial plexus block but comparative study of perineural and intravenous administration of dexmedetomidine with ropivacaine 0.75% in supraclavicular brachial plexus block does not have very large database in literature. Thus, the present study was designed to compare the effect of dexmedetomidine in supraclavicular brachial plexus block in perineural and intravenous routes.

METHODS

It is a prospective, randomised, controlled, double blind clinical trial was done for a period of two years (24 months) at Medical college associated hospital in central India in Department of anaesthesiology and critical care, aimed to compare the effect of intravenous versus perineural administration of Dexmedetomidine in Supraclavicular brachial plexus block using 0.75% Ropivacaine by ultrasound guided technique in upper limb surgeries.

All the patients in the age group of 18-58 years both male and female, having ASA 1 and ASA 2, scheduled for elective surgery of unilateral upper limb surgeries, were included in the study. The patients with significant chronic pain or taking any analgesics, ASA grade III and IV, bleeding disorders, history of brachial plexus injury, known allergy to the study drug, previous shoulder surgery, any psychiatric disorders, peripheral neuropathy, failed block, significant respiratory disease, hearing impairment, pregnant women, study were excluded. The study received ethical approval from the Ethics Committee of the institution and was conducted in accordance with the principles laid out in the Declaration of Helsinki and government regulations.

The sampling of the cases was done by simple randomization according to a computer generated random number table, by anesthesiologist who was blinded about the study. The patients were randomly divided three groups'.

Group RD: received 19.5 ml of 0.75% ropivacaine + 0.75µg/kg dexmedetomidine (total vol-0.5 ml) using ultrasound guidance and 100ml NS i/v over 15mins slowly.

Group RDI: received 19.5ml of ropivacaine + 0.5ml NS in the block and 0.75 microgm/kg dexmedetomidine in 100 ml NS i/v.

Group R: received 19.5 ml of 0.75% ropivacaine + placebo (0.5ml NS), using ultrasound guidance and 100ml NS i/v slowly over 15 mins.

After written informed consent, patients were subjected to a routine pre-anesthetic evaluation (PA checkup) prior to surgery. Detailed medical history clinical history, physical examination and relevant hematological and biochemical screen was done. Patients were advised for preoperative fasting as per latest American Society of Anesthesia (ASA) practice guidelines. A night before the surgery, all patients received Tab. Alprazolam (0.25mg). In the Operation theatre, intravenous line with 18 G cannula was secured and Ringer's lactate solution was started as choice of fluid commencing at the normal infusion rate of 3-5mL/kg.

All the blocks were performed by one person using a transportable ultrasound system (Sonosite Micromaxx; Sonosite Inc., Bothell, WA, USA) with a 38 mm 8-13 Mhz linear high frequency ultrasound transducer (HFL-38).

After sterile preparation of the skin and the ultrasound probe, first the brachial plexus was visualized and then the block was performed using local anesthetic mixture according to group R, RDI or group RD with a 22 gauge needle. The predetermined volume of the drug was administered around the brachial plexus after careful aspiration to avoid accidental intravascular needle procedure. Sensory block, Motor blockade, Hemodynamic parameters was assessed by method used by Chinnappa J et al.[1] Level of sedation was assessed using modified ramsay sedation scale.[1]

Statistical plan

This trial design was non inferiority, to establish that the duration of analgesia of Group RDI in not inferior to Group RD. and the sample size was calculated on presumption based on previous studies.[1,2] The significant difference in the mean duration of analgesia between Group RDI and Group RD was considered to be 50 min with standard deviation of 200 min. The sample size was calculated. Assuming α-error (significance) of 0.05 and power (1-β) of 90%, the effective sample size on the basis of duration of analgesia was 60 in each group for

the comparison. Formula for calculating the sample size is as follows-

$n \propto power * var/\alpha 1 * \Delta$

Var = variability in outcome measure
$\alpha 1$ = value of one side of α
Δ = minimal clinically important difference.

Baseline characteristics were compared using standard descriptive statistics. Visual analogue scale was presented as mean or median (interquartile range (IQR)) as required. The duration of analgesia was analysed by one-way ANOVA and. Categorical data were presented as percent of total. The results were considered significant if *P* value is <0.05 and highly significant if P value is <0.001. All the statistical analysis was done with SPSS 21 version.

RESULTS

Demographic Profile-The demographic details of the study patients are depicted. Patients in the three study groups were comparable (Table 1).

Table 1: Demographic profile.

Variables	RD (n=30) (Mean±SD)	RDI (n=30) (Mean±SD)	R (n=30) (Mean±SD)	p value
Age	36.61±8.201	36.26±7.228	35.28±7.561	0.991
Weight	60.27±9.107	60.27±9.107	59.52±10.419	0.451
Gender (M/F)	16/14	17/13	19/11	0.213
ASA (I/II)	19/11	16/14	18/12	0.882

Data are presented in Mean ± Standard Deviation; p value <0.05= statistically significant and p value < 0.001= highly significant statistically; p value >0.05= statistically non-significant

Block characteristics

The group (group RD) which received perineural dexmedetomidine and ropivacaine using ultrasound guidance showed a significantly better block characteristics as comparison to group which received ropivacaine using ultrasound guidance intravenous without intravenous dexmedetomidine (R) but the difference in the duration of analgesia between (group RD) and group (RDI and R) is less than 50 minutes, which is in agreement with our non-inferiority hypothesis. Time to sensory onset was 8.47±2.501 mins in group RD and in group RDI it was 9.50±3.048 mins as compared to control group R (11.93±1.639 mins) (p<0.001) (Table 2). Duration of sensory block (analgesia) was 1051.20±91.785 mins in group RD and in group RDI it was 1020.80±121.910 mins, and 568.13±90.086 in R (p< 0.001) (Table 2).

Mean time for sensory and motor onset in the perineural (RD), intravenous dexmedetomidine (RDI) and control group(R) is 8.47±2.501min and 9.50±3.048min and 11.93±1.639 and 12.43±3.559mins and13.80±3.890 and 17.67±2.510 mins respectively (Table 2). Though the perineural dexmedetomidine had faster onset and longer duration of the block than the intravenous dexmedetomidine group, still the readings were statistically non-significant.

Table 2: Time for motor and sensory onset and durations among groups.

	Group RD	Group RDI	Group R	ANOVA	
	Mean ±SD	Mean ±SD	Mean ±SD	f	p value
Sensory Onset (min)	8.47±2.501	9.50±3.048	11.93±1.639	15.634	<0.001
Motor Onset (min)	12.43±3.559	13.80±3.890	17.67±2.510	19.447	<0.001
Duration of Sensory Block (min)	1051.20±91.785	1020.80±121.910	568.13±90.086	209.788	< 0.001
Duration of Motor Block (min)	999.73±125.506	906.93±158.779	447.43±45.751	144.848	<0.001

Data are presented in Mean ± Standard Deviation; p value <0.05= statistically significant and p value < 0.001= highly significant statistically; p value >0.05= statistically non-significant

Sedation score

At baseline all the two groups were comparable. Mean level of sedation in group RDI when compared to group RD at different time intervals showed that in intravenous dexmedetomidine group, sedation started at 10 min where as in perineural dexmedetomidine group it started at 20 mins. The level of sedation had highly significant value till 30 mins (<0.001). At onset of sedation the value of mean level of sedation was 3.55±0.78 and 1.93±0.25 in group RDI and RD respectively (Table 3) which shows that patients in intravenous dexmedetomidine group were moderately sedated whereas that in perineural dexmedetomidine group were only slightly sedated.

Hemodynamic parameters

At baseline all the two groups were comparable for mean heart rate, systolic, diastolic and mean blood pressure. Mean heart rate, mean SBP, mean DBP and mean MAP in Group RDI was lower than Group RD at different time intervals post block.

Table 3: Mean level of sedation among groups.

Sedation score	RD (Mean±SD)	RDI (Mean±SD)	R (Mean±SD)	ANOVA p-value
Base line	1	1	1	1
10 min	1	3.55±0.78	1	<0.001
20 min	1.97±0.13	2.59±0.68	1.89±0.31	<0.001
30 min	1.93±0.25	2.34±0.55	1.82±0.48	<0.001
40 min	1.90±0.31	2.10±0.31	1.79±0.57	<0.001
50 min	1.87±0.43	1.93±0.26	1.75±0.65	0.554
60 min	1.80±0.61	1.86±0.44	1.71±0.71	0.996
70 min	1.82±0.56	1.92±0.54	1.70±0.68	0.882
80 min	1.80±0.53	1.87±0.59	1.71±0.56	0.665
90 min	1.78±0.54	1.78±0.56	1.68±0.62	0.702

Data are presented in Mean ± Standard Deviation; p value <0.05= statistically significant and p value < 0.001= highly significant statistically; p value >0.05= statistically non-significant

Table 4: Intra operative changes in mean heart rate among groups.

Time	Group RD	Group RDI	Group R	ANOVA	
	Mean ± SD	Mean ± SD	Mean ± SD	f	p value
Baseline	87.23 ± 9.61	87.23 ± 9.61	89.23 ± 14.29	0.308	0.735
5 min	76.65±4.345	72.43±3.871	85.32±7.554	0.431	<0.001**
10 min	75.40 ± 3.34	68.93 ± 4.92	81.53 ± 9.52	28.384	<0.001**
15 min	72.83 ± 2.52	68.47 ± 2.22	82.80 ±7.369	91.61	<0.001**
20 min	66.97±1.866	66.57±2.725	77.80±10.390	30.754	<0.001**
25 min	56.30±2.261	52.13±2.403	77.10±13.296	57.004	<0.001**
30 min	58.21±2.112	55.21±2.386	80.67±9.012	60.201	<0.001**
40 min	58.40±2.283	56.87±3.267	82.87±8.059	218.014	<0.001**
50 min	58.40±2.313	58.17±2.335	80.37±9.946	133.333	<0.001**
60 min	58.40±2.608	59.43±3.104	80.13±10.190	112.477	<0.001**
70 min	58.20±2.219	58.30±2.231	83.20±8.352	234.462	<0.001**
80 min	58.77±2.359	59.37±2.414	78.67±11.372	81.954	<0.001**
90 min	59.63±2.451	59.21±2.322	79.86±11.487	78.211	<0.001**

Data are presented in Mean ± Standard Deviation; p value <0.05= statistically significant and p value < 0.001= highly significant statistically; p value >0.05= statistically non-significant

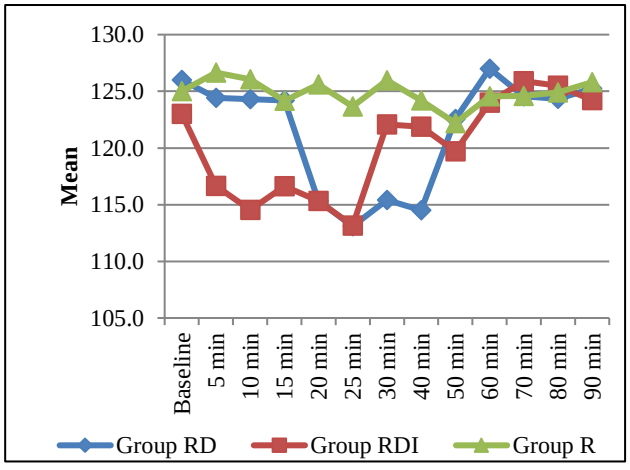

Figure 1: Changes in mean intra operative systolic blood pressure among groups.

The RDI group took longer time for heart rate to get stabilized (40 mins (56.87±3.267 bpm) as compared to RD group 30 mins (58.21±2.112 bpm) (Table 4).

Statistically significant difference was noted mean SBP at 30 and 40 minutes between RD and RDI groups (RD group: 115.40±6.667 mmHg and 114.50±7.09 mmHg (RDI group: 122.07±5.54 mmHg and 121.87±5.355 mmHg) (Figure 1). In RD group maximum fall in DBP was seen from 15 mins till 30 mins with readings being 72.03±9.114, 72.5±7.899 and 72.43±9.254 mmHg respectively (Figure 2).

On the other hand, significant decrease in MAP in group RD was seen at 25 and 30 mins with the values being 83.47±5.431mmHg and 84.70±6.058mmHg when compared with 93.70±5.572mmHg and 94.70±5.069mmHg in group RDI (Figure 3).

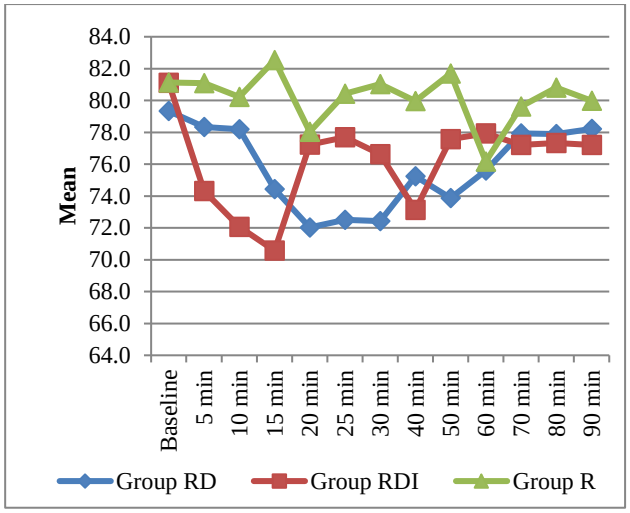

Figure 2: Changes in mean intra operative diastolic blood pressure among groups.

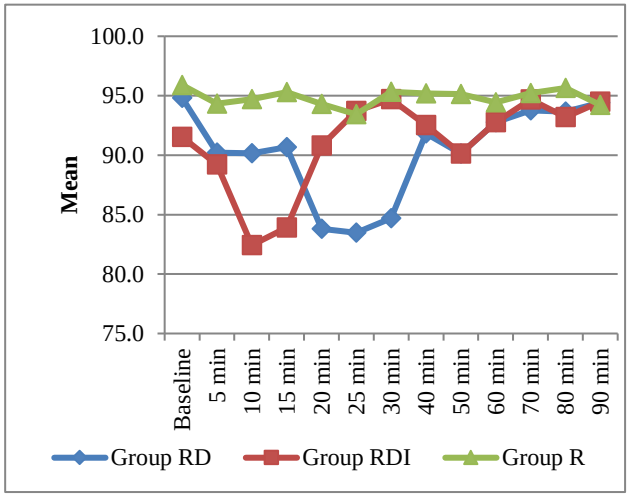

Figure 3: Changes in mean intra operative mean arterial pressure among groups.

DISCUSSION

The hypothesis of this prospective, randomized, double blind, controlled study was that adding 0.75µg/kg dexmedetomidine (0.5ml total volume) to 19.5ml ropivacaine 0.75% perineurally or by intravenous administration of 0.75µg/kg dexmedetomidine with supraclavicular brachial plexus block will shorten onset of the sensory and motor block, prolong the duration of sensory (duration of analgesia) and motor block, increase the level of sedation and decrease the total analgesic requirement with no side effects .

In the present study we compared the effect of ultrasound guided supraclavicular brachial plexus block using plain ropivacaine 0.75% (group R), 0.75% ropivacaine plus dexmedetomidine (group RD) and 0.75% ropivacaine plus dexmedetomidine I.V (group RDI) to study the onset of sensory and motor block, duration of sensory and motor block, level of sedation and hemodynamic variables. Study was conducted on 90 (30 in each group) ASA I or II patients of either sex, aged between 18 years to 58 years who were scheduled to undergo unilateral upper limb surgeries.

Demographic data

Both the groups were comparable in terms of age, sex distribution, weight and ASA grading in our study (Table 1).

Block characteristics

There was a statistical difference in the onset and longer duration of the block in the perineural dexmedetomidine group RD and the intravenous dexmedetomidine group RDI, in the present study. The group (group RD) which received perineural dexmedetomidine and ropivacaine using ultrasound guidance showed a significantly better block characteristics as comparison to group which received ropivacaine using ultrasound guidance intravenous without intravenous dexmedetomidine (R). Kathuria S et al, conducted a similar study using 50µg dexmedetomidine in perineural block (group D) given with 30ml Ropivacaine 0.5% and 50ml normal saline containing 50µg dexmedetomidine administered as IV infusion over 15min in intravenous dexmedetomidine group (D-IV).[12] There observations are similar to the present study and author concluded that dexmedetomidine as an adjuvant to 0.5% ropivacaine in ultrasound guided supraclavicular brachial plexus block shortens the sensory as well as motor block onset time prolongs sensory and motor block duration and also increases the duration of analgesia. This may be attributed to the central effects of intravenous dexmedetomidine playing some role in prolongation of sensory and motor block duration.

Marhofer D et al, added dexmedetomidine as an adjuvant to ropivacaine in patients undergoing ultrasound guided ulnar nerve block.[13] They used 20µg dexmedetomidine through I/V and perineural route along with 3ml of 0.75% ropivacaine. The results obtained in this study are consistent with the results of our study except that surprisingly the time of sensory block was not shortened.

Faraj W et al, demonstrated that dexmedetomidine, whether applied perineurally or intravenously, is an effective local anesthetic adjunct capable of selectively prolonging the duration of inter scalene block analgesia and reducing the cumulative analgesic consumption at 24h without prolonging the duration of motor blockade.[14]

Agarwal A et al, added dexmedetomidine to bupivacaine in patients undergoing supraclavicular brachial plexus block. They showed that addition of dexmedetomidine significantly shortened the onset of block time and prolonged the duration of sensory and motor blocks and duration of analgesia.[15] These findings are consistent with the findings of our study.

The mechanism of the analgesic actions of α2 agonists has not been fully elucidated and is probably multifactorial. Peripheral α2 adrenoceptors may also mediate the antinociception. α2 blockers by acting at any of these sites reduce nociceptive transmission, leading to analgesia. The activation of inwardly rectifying G1-protein-gated potassium channels resulting in membrane hyperpolarization and decreasing the firing rate of excitable cells in the CNS is considered to be a significant mechanism of the inhibitory neuronal action of α2-adrenoceptor agonists. This effect involves direct regulation of entry of calcium through N-type voltage-gated calcium channels and is independent of cAMP and protein phosphorylation and is mediated by G0 proteins. These mechanisms represent 2 very different ways of effecting analgesia, that is, the nerve is prevented from firing, and it also prevents propagation of signals to the neighbors.

The extensive search on the mechanism of action causing prolongation of duration of motor block in intravenous dexmedetomidine did not result in fruitful outcome. However, we can assume that mechanism may be multifactorial which may be difficult to explain and also the fact that our sample size was relatively small though good enough to be statistically viable. The mechanism of analgesic effect can be extrapolated to motor effect to some extent as changes occurring at molecular and ionic level. However, this may best be described as hypothesis at this stage.

Sedation

In our study it was observed that intravenous dexmedetomidine group shows higher level of sedation i.e. 3.55±0.78 at 10 mins as compared to group RD (1.97±0.13) (Table 3). In a study conducted by Kathuria S et al, patients in perineural dexmedetomidine group and intravenous dexmedetomidine were more sedated compared to control group.[11] Most of the patients in their study had sedation grade ≤3. These findings are consistant with the findings of our study.

Hemodynamics

In our study the baseline heart rate, SBP, DBP and MAP were comparable between the three groups. The RDI group took longer time for heart rate to get stabilized (40 mins (56.87±3.267 bpm) as compared to RD group 30 mins (58.21±2.112 bpm) (Table 4). The RDI group reported lower SBP, DBP and MBP as compared to RD group, at different time points but non of the group reported a fall which required any intervention in any group.

The dexmedetomidine results in decreased systemic vascular resistance and indirectly decreased myocardial contractility, cardiac output, and systemic blood pressure. The decrease in HR caused by α-2 agonist can also be explained on the basis of their central action whereby they decrease sympathetic outflow and nor-epinephrine release.[16]

Studies done by Esmaoglu et al, and Agarwal S et al, findings which are also consistent with findings of our study.[17,14]

Hence, we infer that the of ultrasound guided supraclavicular brachial plexus block using plain ropivacaine 0.75% (group R), 0.75% ropivacaine plus dexmedetomidine (group RD) is not inferior to 0.75% ropivacaine plus dexmedetomidine IV (group RDI) in the onset of sensory and motor block, duration of sensory and motor block, whereas level of sedation and hemodynamic variables were almost comparable. It is mainly the direct peripheral action of dexmedetomidine on nerves, which is responsible for improvements in the onset of sensory and motor block, duration of sensory and motor block rather than due to central action of dexmedetomidine. However, the central effects of dexmedetomidine also play some role in prolongation of sensory and motor block duration, as explained previously.

The present study does have some limitations in regard to assessing the duration of a nerve block that is expected to last more than 12h. This aspect was not considered as study duration would have been very long, generating additional data unmanageable in one study.

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REFERENCES

1. Karakaya D, Büyükgöz F, Bariş S, Güldoğuş F, Tür A. Addition of fentanyl to bupivacaine prolongs anesthesia and analgesia in axillary brachial plexus block. Reg Anesth Pain Med. 2001;26(5):434-8.
2. Movafegh A, Nouralishahi B, Sadeghi M, Nabavian O. An ultra-low dose of naloxone added to lidocaine or lidocaine-fentanyl mixture prolongs axillary brachial plexus blockade. Anesth Analg. 2009;109(5):1679-83.
3. Jarbo K, Batra YK, Panda NB. Brachial plexus block with midazolam and bupivacaine improves analgesia. Can J Anaesth. 2005;52(8):822-6.
4. Movafegh A, Razazian M, Hajimaohamadi F, Meysamie A. Dexamethasone added to lidocaine prolongs axillary brachial plexus blockade. Anesth Analg. 2006;102(1):263-7.
5. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: A review of clinical applications. Curr Opin Anaesthesiol. 2008;21:457-61.
6. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg. 2000;90:699-705.

7. Nelson LE, You T, Maze M, Franks NP. Evidence that the mechanism of hypnotic action in dexmedetomidine and muscimol-induced anesthesia converges on the endogenous sleep pathway. Anesthesiol. 2001;95:A136-8.
8. Chinnappa J, Shivanna S, Pujari VS, Anandaswamy TC. Efficacy of dexmedetomidine with ropivacaine in supraclavicular brachial plexus block for upper limb surgeries. J Anaesthesiol Clin Pharmacol. 2017;33(1):81-85.
9. Borgeat A, Ekatodramis G, Dumont C. An evaluation of the infraclavicular block via a modified approach of the Raj technique. Anesth Analg. 2001;93:436-41.
10. Chinnappa J, Shivanna S, Pujari VS, Anandaswamy TC. Efficacy of dexmedetomidine with ropivacaine in supraclavicular brachial plexus block for upper limb surgeries. J Anaesthesiol Clin Pharmacol. 2017;33(1):81-85.
11. Das B, Lakshmegowda M, Sharma M, Mitra S, Chauhan. Supraclavicular brachial plexus block using ropivacaine alone or combined with dexmedetomidine for upper limb surgery: A prospective, randomized, double-blinded, comparative study. Rev Esp Anestesiol Reanim. 2016 Mar;63(3):135-40.
12. Kathuria S, Gupta S, Dhawan I. Dexmedetomidine as an adjuvant to ropivacaine in supraclavicular brachial plexus block. Saudi J Anaesth. 2015;9:148-54.
13. Marhofer D, Kettner SC, Marhofer P, Pils S, Weber M, Zeitlinger M. Dexmedetomidine as an adjuvant to ropivacaine prolongs peripheral nerve block:a volunteer study. Br J Anaesth. 2013;110(3):438-42.
14. Faraj W. Tim D. Vincent W. I.V and perineural dexmedetomidine similarly prolong the duration of analgesia after ISB. Anesth. 2016;124:683-95.
15. Agarwal S, Aggarwal R, Gupta P. Dexmedetomidine prolongs the effect of bupivacaine in supraclavicular brachial plexus block. J Anaesthesiol Clin Pharmacol. 2014;30:36-40.
16. Jang H, Hee Y, Tong J, Jong-Kwon J, Young D, Doo I, et al. Comparison of dexmedetomidine and epinephrine as an adjuvant to 1% mepivacaine in brachial plexus block. Korean J Anesthesiol. 2014;66(4):283-9.
17. Esmaoglu A, Yegenoglu F, Akin A, Turk CY. Dexmedetomidine added to levobupivacaine prolongs axillary brachial plexus block. Anesth Analg. 2010;111(6):1548-51.

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