

Comparative Study of Ropivacaine with Dexmedetomidine Versus Ropivacaine Alone In Interscalene Brachial Plexus Block for Upper Limb Surgeries – A Prospective Randomized Controlled Study

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Abstract:

INTRODUCTION:

Peripheral nerve blockade is a well-accepted concept of comprehensive anaesthetic care. Interscalene brachial plexus block is the preferred regional anaesthesia for upper limb surgeries. Several studies have demonstrated the effects of "dexmedetomidine", an alpha 2 agonist, in local, spinal, epidural anaesthesia when combined with local anaesthetic Ropivacaine. Dexmedetomidine provides analgesic benefits without major side effects.

AIM AND OBJECTIVES: To evaluate additional anaesthetic and analgesic effects derived from administration of dexmedetomidine to ropivacaine to brachial plexus block through Interscalene approach, with respect to onset and duration of sensory blockade, Onset and duration of motor blockade, Duration and rescue analgesia time, haemodynamics and complications.

MATERIALS AND METHODS: 60 patients aged between 18 and 60 years belonging to ASA 1 and ASA 2 divided into two groups. Each group consists of 30 patients.

GROUP A: Received 29ml 0.5% ropivacaine with 1ml normal saline.

GROUP B: Received 29ml 0.5% ropivacaine with 1µg per kg dexmedetomidine diluted to 1ml with normal saline.

After identifying, each patient is given brachial plexus block by interscalene approach using peripheral nerve stimulator. All patients are managed similarly and the effect of onset, duration and field of sensory blockade tested by pinprick test and motor blockade, haemodynamics, complications and post-operative pain evaluated. Rescue analgesia provided with Inj Tramadol 100mg IM. The results obtained were tabulated and analysed.

RESULTS: The onset and duration of sensory blockade were faster in group B than group A which was statistically significant. The onset and duration of motor blockade is longer in group B than in group A which was statistically significant. Time of rescue analgesia is significantly prolonged in group B than in group A. This difference was both clinically and statistically significant (<0.001).

Conclusion: The addition of dexmedetomidine to ropivacaine solution for brachial plexus block can modify the action of local anaesthetic solution by its local action. The dosage 1µg per kg used in the study significantly increases the duration of analgesia. There were no clinically significant side effects noted.

Key words: analgesia, dexmedetomidine, ropivacaine, side effects.

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I. Introduction:

HISTORY:

Pain is the mechanism for informing an organism of a dangerous situation ISAP¹ defined pain as an unpleasant sensory and emotional experience with actual or potential tissue damage or described concerning such damage. The conquest of pain has always been a human quest. In ancient days, drugs used to reduce the pain included alcohol, opium, hashish, and Mandragora. Incas practiced trephination, and their tradition holds the

Shaman performing the procedure chewed cocoa leaves and spat into the wound, producing the local anaesthetic effect.

ANATOMY OF BRACHIAL PLEXUS^{11, 12, 13, 14}:

Performing upper limb regional anaesthesia requires a thorough knowledge of brachial plexus anatomy. Brachial plexus is defined as the network of nerves that begin as spinal nerve roots and continue to the terminal branches that supply the upper extremity. Brachial plexus starts as the ventral primary rami of cervical nerves 5 to 8 (C5–C8), including a greater part of the first thoracic nerve (T1). It originates in the neck, passes laterally and inferiorly over 1st rib then enters the axilla. The parts of the brachial plexus from medial to lateral are roots, trunks, divisions, and cords.

DISTRIBUTION OF BRACHIAL PLEXUS:

These are divided into those that arise above the clavicle- the supraclavicular branches and those that arise below it, the infraclavicular branches.

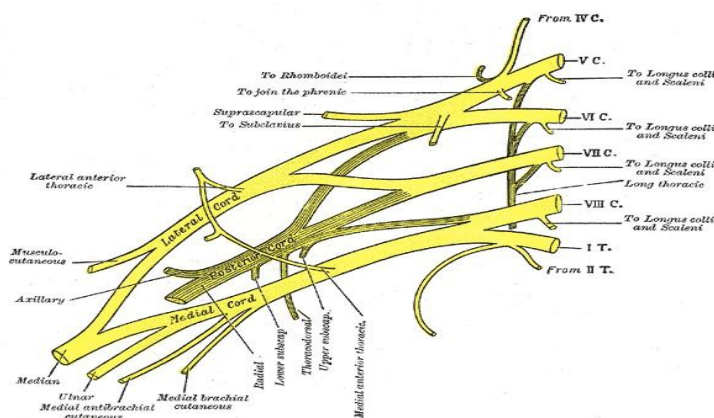


FIGURE NO 1: ANATOMY OF BRACHIAL PLEXUS

APPROACHES TO THE BRACHIAL PLEXUS:

There are four common approaches to block the brachial plexus namely 1. Interscalene, 2. Supraclavicular, 3. Infraclavicular and 4. Axillary.

INTERSCALENE BLOCK

The interscalene approach blocks the brachial plexus at the nerve root or trunk level.

Local anaesthetic is directed toward C5-C6 nerve roots or the superior trunk. Depending on the volume of local anaesthetic used, C7 and even C8 nerve roots may be blocked. The block is especially useful for procedures involving the shoulder, including the lateral 2/3rds of the clavicle, proximal humerus, and shoulder joint.

The block can be performed either as a single injection or continuous nerve block using a catheter. The block can be done using the paraesthesia technique, nerve stimulation technique, ultrasound guidance, or with a combination of nerve stimulation and ultrasonography.

Ulnar sparing is often seen with this block which limits its usefulness for distal surgical procedures.

PHARMACOLOGY OF ROPIVACAINE^{15, 16}:

Ropivacaine is a long acting regional anaesthetic that is structurally related to Bupivacaine. It is a pure s (-) enantiomer, developed for the purpose of reducing potential toxicity and improving relative sensory and motor block profile.

Ropivacaine is an optically pure s (-) enantiomeric form of the parent chiral molecule propivacaine. It belongs to the group of local anaesthetics, the pipercoloxylidides and has a propyl group on the piperidine nitrogen atom compared to bupivacaine, which has a butyl group.

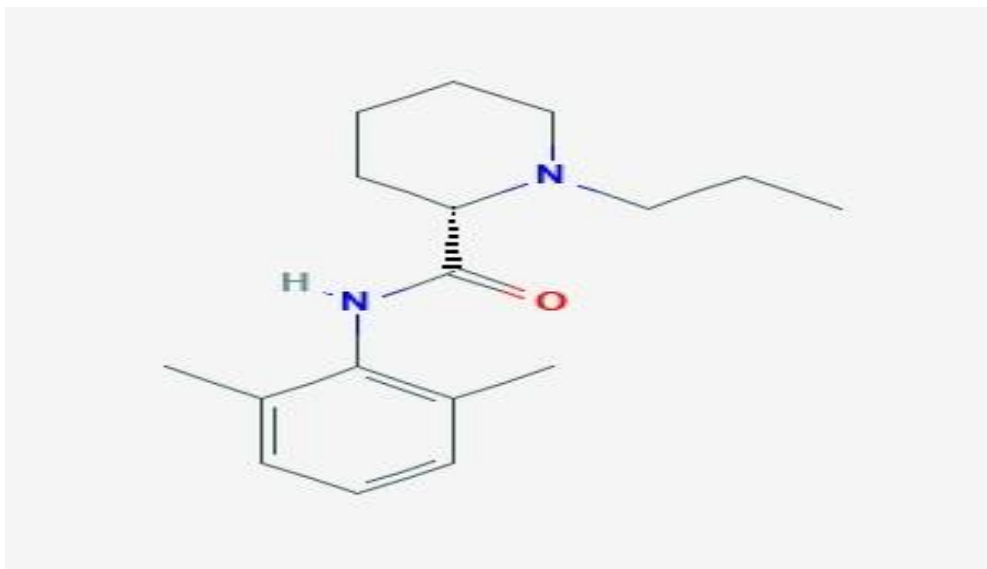


FIGURE NO 2: STRUCTURE OF ROPIVACAINE

Ropivacaine causes reversible inhibition of sodium ion influx, and thereby blocks impulse conduction in nerve fibres. This action is potentiated by dose-dependent inhibition of potassium channels. Ropivacaine is less lipophilic than Bupivacaine and is less likely to penetrate large myelinated motor fibres, therefore it has a selective action on the pain transmitting A beta and C nerves rather than A alpha fibres, which are involved in motor function.

Safe Dose: maximum single dose up to 3to3.5mg/kg.

DEXMEDETOMIDINE:

Dexmedetomidine is a highly selective α_2 -adrenergic agonist. The effects of dexmedetomidine can be reversed with α_2 -antagonist drugs.

Physicochemical Characteristics:

Dexmedetomidine is the active S-enantiomer of medetomidine, a highly selective α_2 adrenergic agonist and imidazole derivative that is used in veterinary medicine. Dexmedetomidine is water soluble and available as a parenteral formulation.

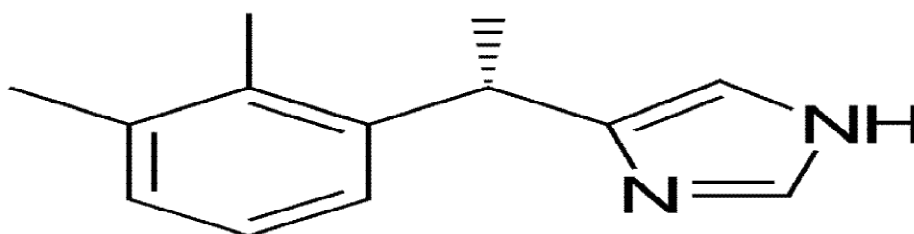


FIGURE NO 3: STRUCTURE OF DEXMEDETOMIDINE

Pharmacodynamics:

Dexmedetomidine produces its effects through activation of CNS α_2 -receptors.

Dexmedetomidine seems to decrease perioperative opioid consumption and improve pain scores, but analgesic benefit has not been shown in all settings.

Specifically, it may be beneficial for prevention of emergence delirium after paediatric anaesthesia.

At the other extreme of age, dexmedetomidine may be superior to propofol for reducing delirium in elderly patients requiring sedation after cardiac or non-cardiac surgery.

II. Material And Methods:

This study was carried out on patients of Department of Anaesthesiology, Siddhartha Medical College/General Hospital Vijayawada, Andhra Pradesh. A total 60 patients of either sex between 18 and 60 years of age under physical status ASA 1 and ASA 2 scheduled for elective upper limb surgeries were included after obtaining ethical clearance from the institution and informed written consent from the patients.

EXCLUSION CRITERIA:

1. Patient refusal.
2. Infection at the site of block.
3. H/O any previous reaction to the local anaesthetic.
4. Patients with injury to any of nerves of the upper limb.
5. Patients with ASA grade 3 and 4.
6. Patients with haemorrhagic disorder.
7. Pregnancy and lactation.
8. Patients with alcohol abuse.
9. H/O underlying psychiatric disease, renal, hepatic disease were excluded.

Routine investigations were done. On the day prior to surgery pre-anaesthetic evaluation was done and detailed history of cardiovascular system, respiratory system, central nervous system, drug therapy and drug allergy were taken general physical examination, systemic examination and airway assessment. All patients were explained about the anaesthetic technique and written informed consent taken. All patients received oral Alprazolam 0.5mg night before surgery and kept NPO for 8 hours prior to surgery. Patients are divided into group A and group B 30 each.

Group A received 29 ml of 0.5% ROPIVACAINE with 1ml NORMAL SALINE.

Group B received 29 ml of 0.5% Ropivacaine with 1 µg/kg wt of DEXMEDETOMIDINE diluted to 1ml with Normal saline.

After identifying, each patient is given brachial plexus block by Interscalene approach using a peripheral nerve stimulator.

All patients are managed similarly and the effect of onset, duration and field of sensory blockade (will be tested by pin prick test with a 3 point scale:0-no block, 1analgesia, 2-loss of touch) and motor blockade on a scale 0 to 3:

0=no motor block,

1=inability to abduct the shoulder or flex the elbow against resistance,

2=inability to abduct the shoulder or flex the elbow against the gravity,

3=inability to abduct the shoulder or flex the elbow and wrist against the gravity), haemodynamics, complications and post operative pain are evaluated.

Rescue analgesia provided with inj. Tramadol 100mg I.M.

The results obtained were tabulated and analysed.

PROCEDURE:

On the morning of the surgery, everything kept ready for emergency intubation and emergency drugs. After shifting the patient to operation theatre non-invasive minimum mandatory monitors were attached and the baseline pulse rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, ECG (lead II) and oxygen saturation were noted down. A wide bore I.V. cannula was secured. The patient was made to lie in the supine position; head turned away from the side to be blocked and shoulder depressed. The arm of the side to be blocked was kept adducted and maintaining strict aseptic precautions.



FIGURE NO 4: POSITION AND LANDMARKS FOR SUPRACLAVICULAR BRACHIAL BLOCK



FIGURE NO 5: TECHNIQUE OF INTERSCALENE BRACHIAL PLEXUS BLOCK WITH A PERIPHERAL NERVE STIMULATOR

A 2-inch 24G short bevel needle connected to a syringe and nerve locator was inserted through the skin wheal and advanced perpendicular to the skin. The following setting was used in nerve locator.

Frequency was set at 1 Hz as 2 Hz may cause unpleasant and vigorous muscle twitches. The positive electrode connected to ECG lead. The negative electrode to a port in the needle. Begin at 1.5 mA current strength and observe for twitch of pectorals, deltoid, biceps, forearm, and hand muscles. As soon as we observed the twitch, the current strength was decreased to 0.5mA with continued observation of twitch. Even at 0.5 mA current when we got a satisfactory twitch, the simulator was turned off, and the drug injected with repeated aspiration for blood. If the twitch disappeared on decreasing the current strength, the needle was repositioned to elicit the twitch response and again the procedure repeated.



FIGURE NO 6: PERIPHERAL NERVE STIMULATOR.

After proper point location, negative aspiration test for blood was done and drug mixture was then injected slowly. Care was taken that the needle did not get displaced. Immediately after drug injection, gently massage was done over the point of drug injection for even distribution of the drug.

Onset time of sensory blockade, Onset time of motor blockade, Duration of sensory blockade, Duration of analgesia, Duration of motor blockade, time for rescue analgesia and side effects and complications like hypotension, bradycardia, respiratory depression, nausea, vomiting allergic reactions pneumothorax in the intra and post-operative period were observed.

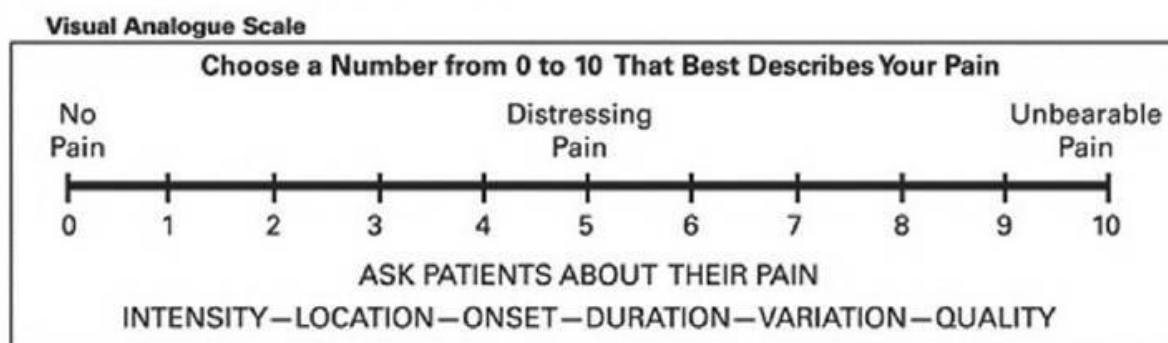


FIGURE NO 7: VISUAL ANALOGUE SCALE.

RAMSAY SEDATION SCALE:

1. Anxious, agitated or restless, or both.
2. Cooperative, oriented, and tranquil.
3. Responds to commands only.
4. Brisk response to a glabellar tap or auditory stimulus.
5. Sluggish response to a light glabellar (forehead) tap or loud sound stimulus.
6. No response.

STATISTICAL ANALYSIS:

Descriptive data presented as mean with SD entered in MS-EXCEL and analysed in SPSS V22. Chi-square test, independent t-test were applied to find significance p value <0.05 was considered Statistical significance. p<0.001 was considered as highly significant.

III. Observations And Results:

DEMOGRAPHICS:

TABLE NO 1: Age Distribution.

Age	Group A		Group B	
	Count	%	Count	%
18-20	2	6.7%	4	13.3%
21-30	1	3.3%	6	20.0%
31-40	13	43.3%	6	20.0%
41-50	11	36.7%	6	20.0%
51-60	3	10.0%	8	26.7%
Total	30	100.00%	30	100.00%
P=0.67				

TABLE NO 2: Age Distribution- Mean and SD.

Variable	Group	N	Mean (in Years)	SD	P value
Age	Group A	30	41.33	10.53	0.67
	Group B	30	39.97	13.73	

There are 30 patients in each group. As shown in tables and graphs above most of the patients are below 50 years in both the groups. The mean age in Group A was 41.33±10.53 years, and that of group B is 39.97±13.73 years. The P value is 0.67. Hence there is no statistically significant difference regarding age, and both the groups are comparable regarding age distribution.

TABLE NO 3: Sex Distribution.

Sex	Group A		Group B		P-value
	Count	%	Count	%	
Female	3	10.0%	9	30.0%	0.10
Male	27	90.0%	21	70.0%	
Total	30	100.0%	30	100.0%	

As seen in the above table and graph males predominant in both the groups. There are 10% of females in group A compared to 30% in group B. There are 90% males in group A when compared to 70% in group B. The P value is 0.1. Hence there is no statistically significant difference in sex distribution, and both groups are comparable regarding sex distribution.

TABLE NO 4: Onset Time for Sensory Block (SOT).

Variable	Group	N	Mean (in Min.)	SD	P-value
SOT	Group A	30	22.97	2.30	<0.001
	Group B	30	10.53	1.81	

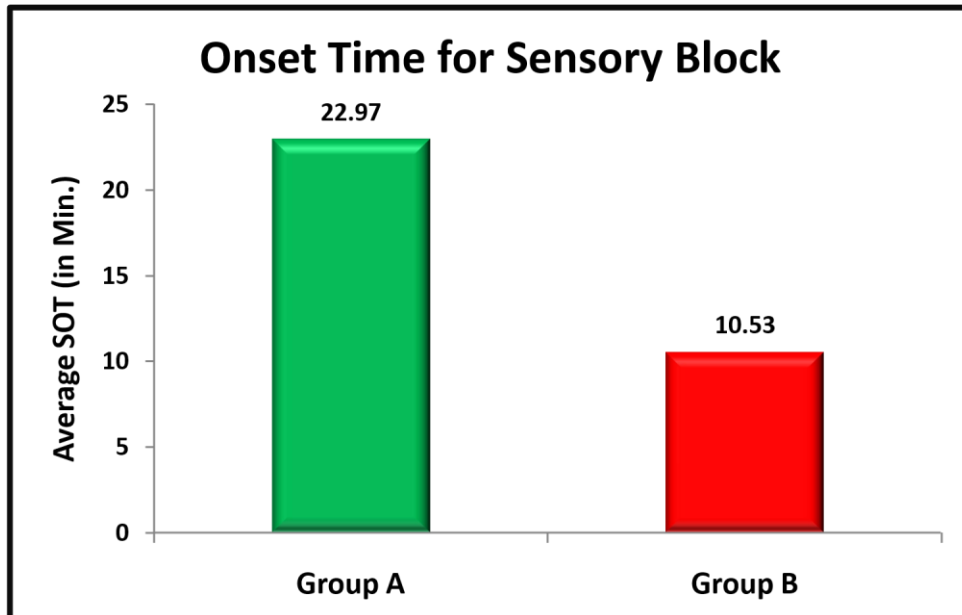


FIGURE NO 8: Bar Diagram Representing Onset Time for Sensory Block (SOT).

The mean time for onset of sensory block in group ropivacaine with dexmedetomidine was 10.53 ± 1.81 min when compared to 22.97 ± 2.3 min in ropivacaine group. The p-value is < 0.001 . Hence the onset of the sensory blockade is earlier in the dexmedetomidine group when compared with ropivacaine group, and this difference is statistically highly significant.

TABLE NO 5: Onset Time for Motor Blockade (MOT).

Variable	Group	N	Mean (in Min.)	SD	P-value
MOT	Group A	30	28.13	2.36	< 0.001
	Group B	30	15.10	1.83	

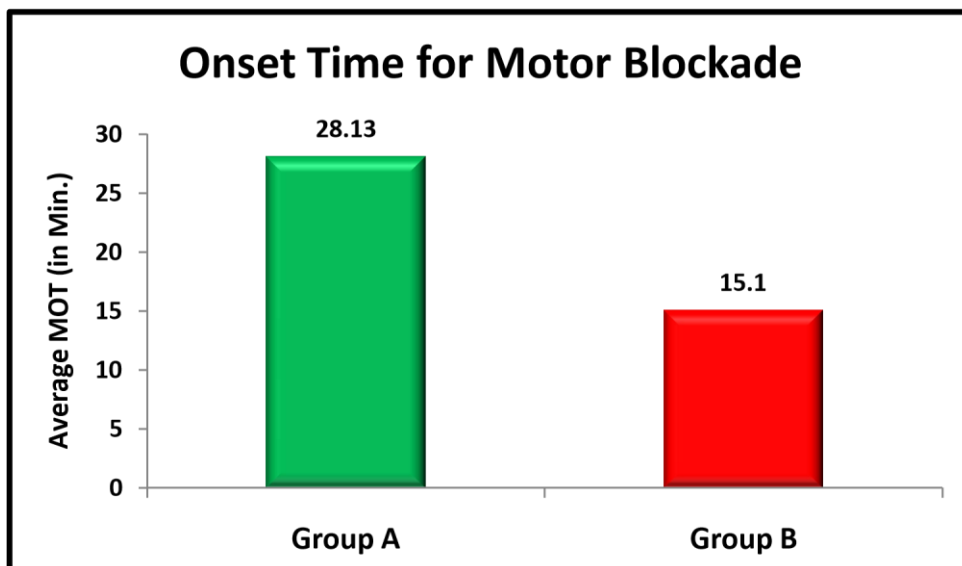


FIGURE NO 9: Bar Diagram Representing Onset Time for Motor Blockade (MOT).

The onset of the motor blockade in group A is 28.13 ± 2.36 min, and that of group B is 15.1 ± 1.83 min. The P value is < 0.001 . Hence the onset of the motor blockade is earlier in the Dexmedetomidine group, and the difference is statistically significant.

TABLE NO 6: Duration of Sensory Blockade (DOSB).

Variable	Group	N	Mean (in Hr.)	SD	P-value
DOSB	Group A	30	7.83	0.54	<0.001
	Group B	30	12.80	0.84	

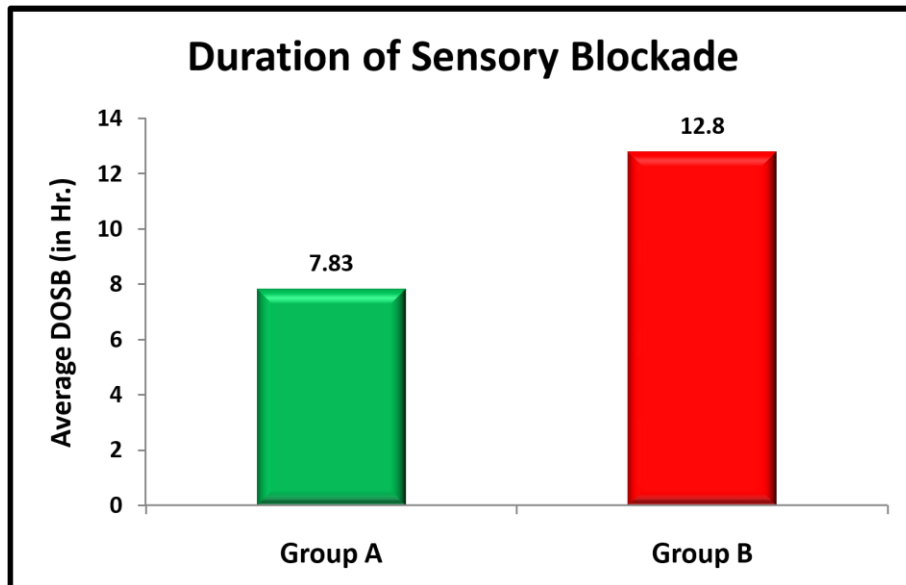


FIGURE NO 10: Bar Diagram Representing Duration of Sensory Blockade (DOSB).

The duration of sensory blockade, in group A is 7.83 hrs, and that of group B is 12.8 hrs. The P value is < 0.001. Hence the duration of the sensory blockade is more in the dexmedetomidine group when compared to ropivacaine group, and the difference is statistically highly significant.

TABLE NO 7: Duration of Motor Blockade (DOMB).

Variable	Group	N	Mean (in Hr.)	SD	P-value
DOMB	Group A	30	7.07	0.51	<0.001
	Group B	30	11.42	0.99	

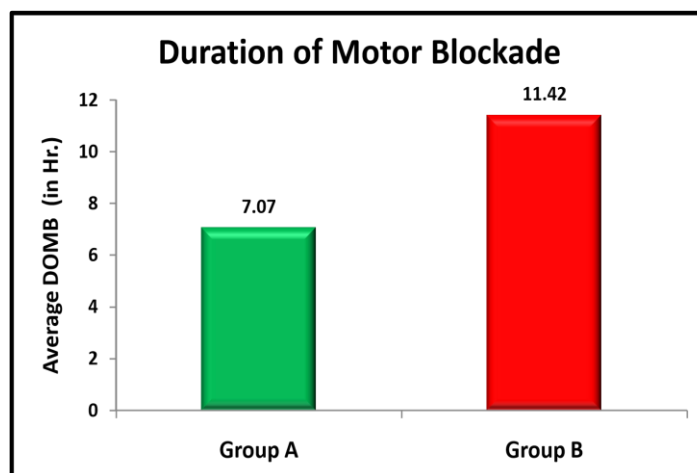


FIGURE NO 11: Bar Diagram Representing Duration of Motor Blockade (DOMB).

The duration of motor blockade, in group A is 7.07± 0.51hrs. The duration of motor block in group B is 11.42±0.99hrs. Hence the motor blockade lasts longer in the dexmedetomidine group when compared to ropivacaine group. The difference is statistically significant.

TABLE NO 8: Duration of Analgesia (DOA).

Variable	Group	N	Mean (in Hr.)	SD	P-value
DOA	Group A	30	8.75	0.61	<0.001
	Group B	30	14.05	0.80	

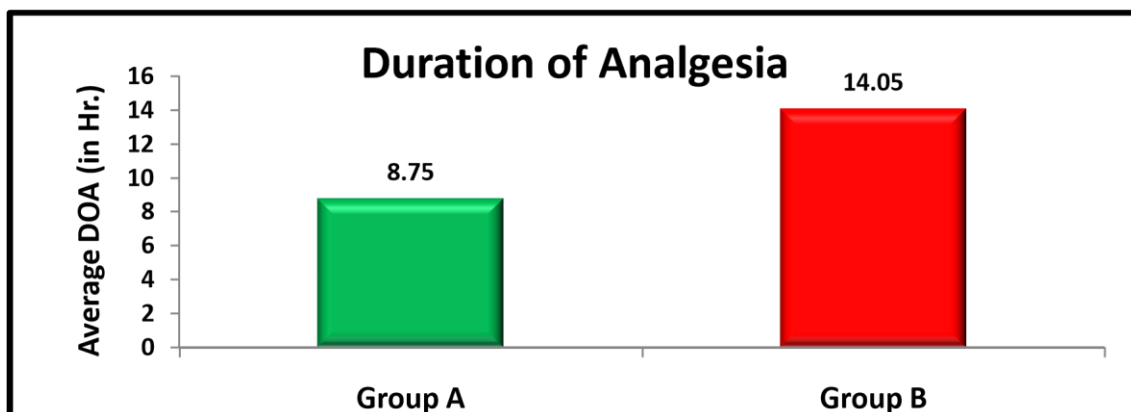


FIGURE NO 12: Bar Diagram Representing Duration of Analgesia (DOA)

Time till the patients complains pain i.e., VAS > 1 is taken as duration of analgesia. It is 8.75±0.61 hrs in group A, and it is 14.04±0.8 hrs in group B. Hence dexmedetomidine prolongs analgesia duration and is statistically highly significant.

TABLE NO 9: Time of Rescue Analgesia (TORA)

Variable	Group	N	Mean (in Hr.)	SD	P-value
TORA	Group A	30	9.32	0.75	<0.001
	Group B	30	14.69	1.45	

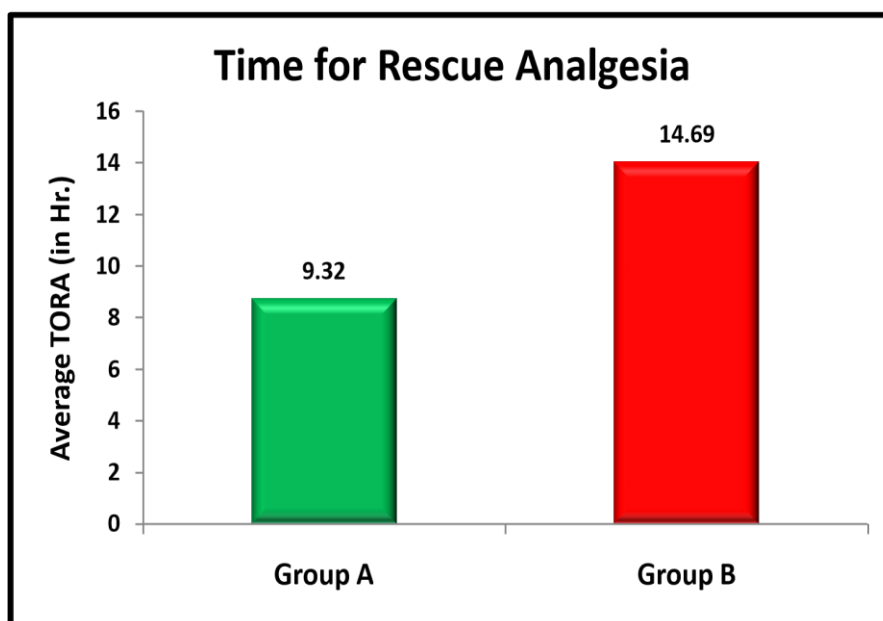


FIGURE NO 13: Bar Diagram Representing Time for Rescue Analgesia (TORA)

Time till patients complain pain and asks for analgesic i.e., VAS > 4 is taken as time of rescue analgesia. It is 9.32 ±0.75 hrs in group A, and it is 14.69 ±1.45 hrs in group B. The requirement of rescue analgesic was earlier in the ropivacaine group. Hence dexmedetomidine prolongs analgesia duration and is statistically highly significant.

significant. There was no statistically significant reduction in heart rate upon addition of dexmedetomidine to ropivacaine for interscalene brachial plexus block.

The above graph and table show the sedation which was calculated by a 6 point Ramsay sedation score. Though there was increased sedation in dexmedetomidine plus ropivacaine group, it was not clinically significant, and the patients were not deeply sedated but they were tranquil.

IV. Discussion:

Interscalene brachial plexus block is an effective, time-tested regional anaesthetic technique for surgeries of upper extremities.

It is not only an excellent alternative, but also offers several perioperative advantages over general anaesthesia like reduced stress response, less blood loss, provides superior surgical conditions, provides optimal postoperative analgesia and reduces the incidence of postoperative nausea and vomiting, providing early ambulation and reduced length of hospital stay, leading to satisfactory patient acceptance and improved clinical outcomes.

However, the use of Single-shot Interscalene Brachial plexus blocks has a major limitation of short-lived analgesia, generally lasting less than 24 hours.

Strategies to prolong Brachial plexus block analgesia beyond the pharmacological duration of the Local Anaesthetic included placement of indwelling perineural catheters to allow prolonged infusion or co administration of adjuvants.

Ropivacaine is an important option for regional anaesthesia, postoperative pain management and labour analgesia due to the following reasons: $\frac{3}{4}$ Efficacy $\frac{3}{4}$ Lower propensity for the motor block. $\frac{3}{4}$ Reduced potential for central nervous system toxicity and cardiotoxicity.

A systemic review and meta-analysis by Abdallah² et al. suggested that a dose of 50-60 μ g maximises sensory block duration (by at least 52%, $P < 0.0001$) while minimizing the hemodynamic side-effects to none.

Mostly accepted theories for the mechanism of action are alpha two mediated vasoconstriction, centrally mediated analgesia through action on locus ceruleus, and spinal component through action on substantia gelatinosa.

The inhibition of I_h current caused by Dexmedetomidine is more profound on C fibres (pain) than on A alpha (motor) fibres making the effects of the drug more sensory-specific.

In a prospective, double-blinded and randomized controlled study conducted by Anjan Das³ et al. (2014) the mean age in RD group is 43 and R group is 44 years.

In a Random, controlled, and triple-blind study conducted by Suneet Kathuria⁴ et al. (2015), the mean age in groups C, D, D-4 is 32,30,34 years respectively.

Gender distribution between the two groups was also comparable with no statistically significant difference in the present study.

In the present study, the mean onset time for sensory blockade was 10.53 ± 1.81 min in Ropivacaine with Dexmedetomidine group and 22.97 ± 2.3 min in the Ropivacaine group.

A prospective, double-blinded and randomized controlled study by Anjan Das³, Saikat Majumdar, Susanta Halder, Surajit Chattopadhyay, Saswati Pal, Ratul Kundu, Subrata Kumar Mandal, Sandip Chattopadhyay showed the time taken to achieve sensory blockade in RD group was 14 ± 3 min and R group was 15 ± 5 minutes. These results are in concordance with our present study.

A study conducted by Suneet Kathuria⁴ et al (2015) showed sensory onset time in Ropivacaine with Dexmedetomidine group was 9 ± 4 minutes and in plain Ropivacaine group was 22 ± 8 minutes which is comparable to the present study.

A study conducted by Indira Gurajala⁵ et al (2015) showed sensory onset time in Ropivacaine with Dexmedetomidine group was 15-30 minutes and in plain Ropivacaine group was 20-45 minutes.

A prospective randomized double-blinded clinical trial by Ananda Bangera⁶, Mukka Manasa, and Prasad Krishna showed sensory onset time in Ropivacaine with Dexmedetomidine group was 16 ± 4 minutes and in plain Ropivacaine group was 20 ± 3 minutes which is comparable to the present study.

A study conducted by Vorobeichik L⁷, Brull R, Abdallah FW showed Dexmedetomidine expedited onset for both sensory (at least 40%, $P < 0.0001$) and motor (at least 39%, $P < 0.0001$) blocks.

In the present study onset of the motor blockade in group A was 28.13 ± 2.36 min, and that of group B was 15.1 ± 1.83 min. The P value is < 0.001 . Hence the onset of the motor blockade is earlier in the Dexmedetomidine group compared to plain Ropivacaine group.

A study conducted by Jithendra Chinnappa⁸ et al showed motor onset time in Ropivacaine with Dexmedetomidine group was 15 ± 6 minutes and in plain Ropivacaine group was 23 ± 5 minutes which is comparable to the present study.

A prospective, randomized double-blind study by HD Rashmi⁹, HK Komala et al showed motor onset time in Ropivacaine with Dexmedetomidine group was 15 ± 1 minutes and in plain Ropivacaine group was 20 ± 2 minutes.

These results are in concordance with the present study.

A prospective, randomized, double-blind study Vandana Mangal¹⁰ et al showed motor onset time in Ropivacaine with Dexmedetomidine group was 9 ± 2 minutes and in plain Ropivacaine group was 11 ± 2 minutes.

A prospective randomized double-blinded clinical trial by Ananda Bangera⁶ et al showed motor onset time in Ropivacaine with Dexmedetomidine group was 18 ± 3 minutes and in plain Ropivacaine group was 23 ± 3 minutes. The duration of sensory blockade, in group A that is plain Ropivacaine group was 7.83 hrs (469min), and that of group B that is Ropivacaine with Dexmedetomidine group was 12.8 hrs (768min.). The duration of the sensory blockade is more in the Dexmedetomidine group when compared to Ropivacaine group.

A prospective, double-blinded and randomized controlled study by Anjan Das³ et al found that duration of sensory blockade, in plain Ropivacaine group was 544 min, and that of Ropivacaine with Dexmedetomidine group was 846 minutes.

A study conducted by Suneet Kathuria⁴ et al (2015) found that duration of sensory blockade, in plain Ropivacaine group was 451 min, and that of Ropivacaine with Dexmedetomidine group was 789 minutes.

A study conducted by Indira Gurajala⁵ et al (2015) found that duration of sensory blockade, in plain Ropivacaine group was 420 min, and that of Ropivacaine with Dexmedetomidine group was 890 minutes.

A prospective randomized double-blinded clinical trial by Ananda Bangera⁶, Mukka Manasa, and Prasad Krishna showed that duration of sensory blockade, in plain Ropivacaine group was 494 min, and that of Ropivacaine with Dexmedetomidine group was 677 minutes.

A prospective, randomized, double-blind study by Vandana Mangal¹⁰ et al found that duration of sensory blockade, in plain Ropivacaine group was 543 min, and that of Ropivacaine with Dexmedetomidine group was 613 minutes.

A prospective, randomized double-blind study by HD Rashmi⁹, HK Komala et al found that duration of sensory blockade, in plain Ropivacaine group was 524 min, and that of Ropivacaine with Dexmedetomidine group was 717 minutes.

In the present study the duration of motor blockade, in group A that is plain Ropivacaine was 7.07 ± 0.51 hrs (427 ± 30 min). The duration of motor block in group B that is Ropivacaine with Dexmedetomidine was 11.42 ± 0.99 hrs (685 ± 59 min). Hence the motor blockade lasts longer in the Dexmedetomidine group when compared to Ropivacaine group.

A study conducted by Jithendra Chinnappa⁸ et al showed duration of motor block in Ropivacaine with Dexmedetomidine group was 545 ± 227 minutes and in plain Ropivacaine group was 346 ± 76 minutes, which is comparable to the present study.

A prospective randomized double-blinded clinical trial by Ananda Bangera⁶ et al showed that duration of motor blockade, in plain Ropivacaine group was 526 ± 70 min, and that of Ropivacaine with Dexmedetomidine group was 712 ± 89 minutes which is comparable to the present study.

A study conducted by Suneet Kathuria⁴ et al (2015) found that duration of motor blockade, in plain Ropivacaine group was 387 ± 129 minutes, and that of Ropivacaine with Dexmedetomidine group was 754 ± 180 minutes.

A study conducted by Indira Gurajala⁵ et al (2015) showed that duration of motor blockade Ropivacaine with Dexmedetomidine group was 660-1110 minutes and in plain Ropivacaine group was 300-450 minutes.

In the present study, the duration of analgesia was 8.75 ± 0.61 hrs in group A i.e. plain Ropivacaine group, which was less than that of Ropivacaine with Dexmedetomidine group that is 14.04 ± 0.8 hrs.

This prolongation of analgesic effect by using Dexmedetomidine as an adjunct to brachial plexus block is also noted in several studies.

A prospective, randomized, double-blind study by HD Rashmi,⁹ HK Komala et al showed the duration of analgesia in Ropivacaine with Dexmedetomidine group was 872 ± 11.24 minutes and in plain Ropivacaine group was 590 ± 15.24 minutes.

These results are in concordance with the present study.

It is taken as the time from onset of block till the patient complains of unbearable pain and requests for analgesic or VAS score > 4 . Patients were given injection Tramadol 100 mg IM.

The time for rescue analgesia in our study was 559 ± 45 minutes in Ropivacaine group, and it was 881 ± 87 minutes in Ropivacaine with Dexmedetomidine group. This showed that addition of $1 \mu\text{g}$ per kg of Dexmedetomidine to Ropivacaine not only prolonged the duration of analgesia but also increased the time required for a rescue analgesic administration.

Studies conducted by Anjan Das³ et al., Suneet Kathuria⁴ et al., Vandana Mangal¹⁰ et al., Indira Gurajala⁵ et al. showed reduced number of rescue analgesia.

None of the patients had hypotension (defined as decrease in blood pressure by 20%) and maintained hemodynamic parameters well within the normal range.

A prospective, randomized, double-blind study by Vandana Mangal¹⁰ et al. observed none of the patients had hypotension and maintained the hemodynamic parameters within the normal range. Bradycardia was observed in 2 patients in the dexmedetomidine group, which responded to a single dose of injection atropine.

A study conducted by Jithendra Chinnappa⁸ et al. showed hemodynamic variables were comparable between their two groups during the study intervals except that intra operatively 4 patients in dexmedetomidine group developed hypotension and one patient had bradycardia.

A prospective, randomized, double-blind study by HD Rashmi⁹, HK Komala et al. showed a statistically significant difference ($P < 0.05$) in HR between two groups from 10 min after the block and also in the mean arterial pressures.

The spo2 values were comparable between the two groups in the present study, which was similar to the studies conducted by Anjan Das³ et al., Agarwal et al. and Suneet Kathuria⁴ et al.

Also, in the present study, there was no excessive sedation in either of groups, and the Ramsay Sedation score was 2 in Ropivacaine with Dexmedetomidine group and 1 in the Ropivacaine group. The sedative effect of perineural dexmedetomidine may be due to its partial vascular uptake and transport to the central nervous system where it acts and produces sedation.

A prospective, randomized, double-blind study by Vandana Mangal¹⁰ et al. showed sedation scores were higher in patients receiving dexmedetomidine compared to the control group. Intra operatively, more sedation was observed from 20 minutes to 120 minutes time point in the dexmedetomidine group. The modified Ramsay sedation score for the dexmedetomidine group was either 3/6 or 4/6 in a majority of the cases, while that for the control group, it was 2/6. No patient experienced airway compromise or required airway assistance because of sedation in their study.

V. Conclusion:

The addition of Dexmedetomidine to Ropivacaine solution for brachial plexus block can modify the action of the local anaesthetic solution by its local action. The dosage 1µgm/kg body weight used in the study significantly increased the duration of analgesia. There were no clinically significant side effects noticed. Hence Dexmedetomidine can form an useful adjuvant for Ropivacaine when used for brachial plexus block without any adverse effects with faster and prolonged block characters.

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