

**ORIGINAL RESEARCH**

# Assessment of the effect of intravenous dexmedetomidine on post-operative analgesia in patients undergoing orthopedic lower limb surgeries under subarachnoid block

<sup>1</sup>Dr. Saurabh Singhal, <sup>2</sup>Dr. Shailendra Kumar<sup>1</sup>Associate Professor, <sup>2</sup>Assistant Professor, Department of Anaesthesia, FH Medical College and Hospital, Etmadpur, Agra, UP, India**Corresponding Author**

Dr. Shailendra Kumar

Assistant Professor, Department of Anaesthesia, FH Medical College and Hospital, Etmadpur, Agra, UP, India

Received: 16 January, 2024

Accepted: 21 February, 2024

**ABSTRACT**

**Background:** The majority of procedures involving the abdomen and lower limbs are best performed under regional anesthetic. This study evaluated the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine. **Materials & Methods:** 90 patients scheduled for orthopedic lower limb surgeries under sub arachnoid blocks were divided into 2 groups of 45 patients each. Drugs for both groups prepared in two 50 ml syringes- one for loading dose (labelled L) and the other for maintenance dose (labelled M). **Results:** Group I had 25 males and 20 females and group II had 23 males and 22 females. The mean duration of onset of sensory blockade was 7.2 minutes in group I and 6.1 minutes in group II. The duration of onset of motor blockade was 3.1 minutes in group I and 3.3 minutes in group II. The duration of recovery from sensory blockade was 215.0 minutes in group I and 161.5 minutes in group II. The duration of recovery from motor blockade was 238.2 minutes in group I and 195.4 minutes in group II. The sedation score was 2.7 in group I and 3.5 in group II. The difference was significant ( $P < 0.05$ ). The pain score (VAS) in group I and II was 2.3, 3.2 respectively. At 2 hours, it was 1.6 and 4.6, at 6 hours was 2.4 and 5.1, at 12 hours was 2.4 and 5.1 and at 24 hours was 2.1 and 6.2 respectively. The difference was significant  $P < 0.05$ . **Conclusion:** Arousable sedation produced by subarachnoid anesthesia is prolonged by intrathecal dexmedetomidine.

**Keywords:** dexmedetomidine, subarachnoid anesthesia

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

**INTRODUCTION**

The majority of procedures involving the abdomen and lower limbs are best performed under regional anesthetic. Numerous adjuvants, including epinephrine, phenylephrine, magnesium sulfate, neostigmine, opioids, and clonidine, have been administered intrathecal to extend the duration of the effect of bupivacaine.<sup>1</sup>  $\alpha_2$ -agonists work by activating G1-protein-gated potassium channels, which results in membrane hyperpolarization and lowers the firing rate of excitable cells in the central nervous system. This is the molecular mechanism underlying the analgesic activity of  $\alpha_2$ -agonists. Furthermore,  $\alpha_2$ -agonists prevent the release of neurotransmitters by decreasing the amount of calcium that enters the cell. These two methods indicate two distinct ways in which analgesia

can be influenced: first, by blocking neuron firing, and second, by obstructing the signal's ability to reach its neighbor.<sup>2,3</sup>

Dexmedetomidine operates at both the supraspinal and spinal levels, specifically the laminae VII and VIII of the ventral horns of the spinal cord. It has anxiolytic, sedative, analgesic, and sympatholytic qualities and can be delivered systemically or intrathecally. Because of its calming and analgesic properties, this highly selective  $\alpha_2$ -adrenergic agonist is frequently used as a premedication before general anesthesia.<sup>4</sup> A synergistic interaction between intrathecal dexmedetomidine and local anesthetics has been documented in earlier research, despite the paucity of clinical evidence addressing the effects of intravenous dexmedetomidine on the duration of

sensory and motor block of spinal anesthesia.<sup>5</sup>This study evaluated the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine.

**MATERIALS & METHODS**

The present study consisted of 90 patients scheduled for orthopedic lower limb surgeries under subarachnoid block. All patients were enrolled after obtaining their written consent.

Data such as name, age, gender, etc. was recorded. Patients were divided into two groups of 45 patients each. Drugs for both groups were prepared in two 50 ml syringes- one for loading dose (labelled L) and the other for maintenance dose (labelled M). Dexmedetomidine in a dose of 0.7 µg/kg was given as

a loading dose in the recovery room and patients of group II received normal saline. The study drug was premixed to a total volume of 50 ml and administered through an infusion pump @ 1ml/min as maintenance, 20 min after loading dose subarachnoid block with 0.5% hyperbaric bupivacaine given. Parameters such as sensory block, motor blockade, level of sedation, postoperative analgesia, total duration of analgesia, systemic arterial blood pressure, heart rate, pulse oximetry, and electrocardiography were recorded at base line, after subarachnoid block at 3 minutes intervals until 20 min and then at 5 min interval until the end of surgery were recorded. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

**RESULTS**

**Table I Distribution of patients**

Gender	Group I	Group II
Male	25	23
Female	20	22

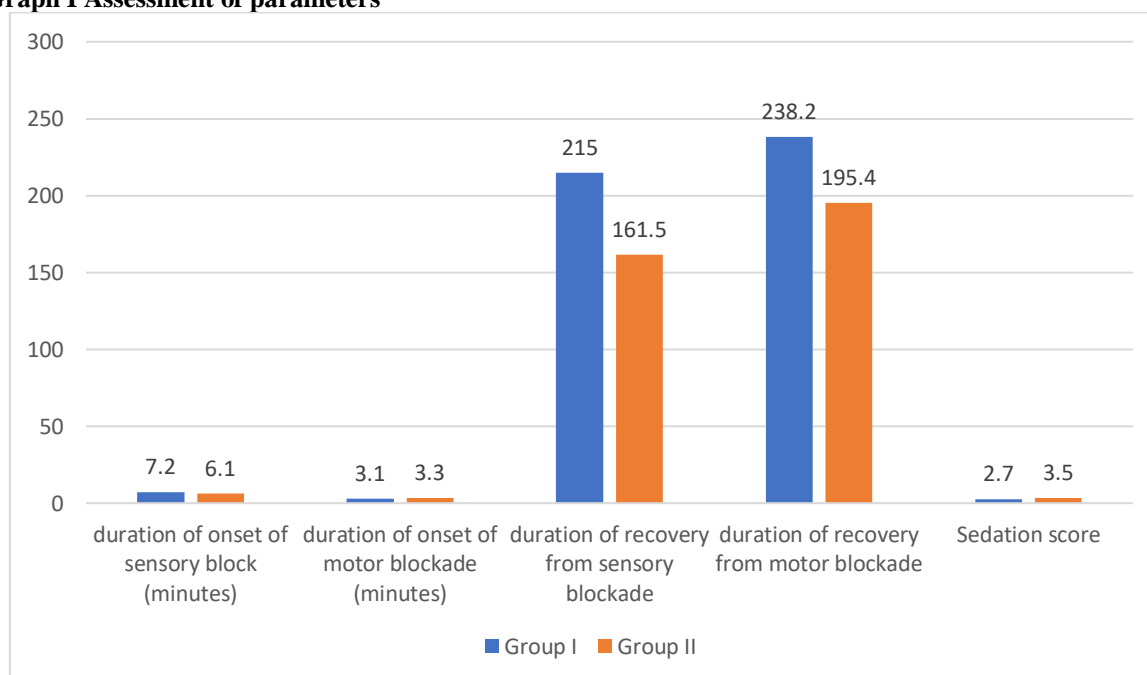
Table I shows that group I had 25 males and 20 females and group II had 23 males and 22 females.

**Table II Assessment of parameters**

Parameters	Group I	Group II	P value
duration of onset of sensory block (minutes)	7.2	6.1	0.71
duration of onset of motor blockade (minutes)	3.1	3.3	0.42
duration of recovery from sensory blockade	215.0	161.5	0.01
duration of recovery from motor blockade	238.2	195.4	0.02
Sedation score	2.7	3.5	0.56

Table II, graph I show that the mean duration of onset of sensory blockade was 7.2 minutes in group I and 6.1 minutes in group II. The duration of onset of motor blockade was 3.1 minutes in group I and 3.3 minutes in group II. The duration of recovery from sensory blockade was 215.0 minutes in group I and 161.5 minutes in group II. The duration of recovery from motor blockade was 238.2 minutes in group I and 195.4 minutes in group II. The sedation score was 2.7 in group I and 3.5 in group II. The difference was significant (P< 0.05).

**Graph I Assessment of parameters**



**Table III Assessment of post-operative pain**

Time intervals	Group I	Group II	P value
2 hours	2.3	3.2	0.84
6 hours	1.6	4.6	0.01
12 hours	2.4	5.1	0.02
24 hours	2.1	6.2	0.01

Table III shows that the pain score (VAS) in group I and II was 2.3, 3.2 respectively. At 2 hours, was 1.6 and 4.6, at 6 hours was 2.4 and 5.1, at 12 hours was 2.4 and 5.1 and at 24 hours was 2.1 and 6.2 respectively. The difference was significant  $P < 0.05$ ).

## DISCUSSION

For abdominal and lower limb procedures, hyperbaric bupivacaine subarachnoid block (SAB) is frequently used.<sup>6</sup> Opioids and  $\alpha_2$  agonists are the most frequently utilized intrathecal adjuvants to local anesthetics to boost their potency and extend the duration of SAB. Studies have also demonstrated that the duration of the sensory and motor blockage achieved with subarachnoid block can be extended by administering dexmedetomidine intravenously.<sup>7</sup> Additionally, the duration of spinal anesthesia was prolonged when dexmedetomidine was infused intravenously before spinal anesthesia or as a loading dose followed by continuous infusion during the operation.<sup>8,9</sup> This study evaluated the effects of intravenous dexmedetomidine on spinal anesthesia with 0.5% hyperbaric bupivacaine.

We found that group I had 25 males and 20 females and group II had 23 males and 22 females. 75 patients who had been hospitalized for lower limb and non-traumatic surgeries were recruited for a prospective, randomized, double-blind, placebo-controlled trial by Sharma et al.<sup>10</sup> The patients were split into three groups: Twenty mL of 0.9% NaCl was injected intravenously into Group B ( $n = 25$ ), which received intrathecal 2.4 mL of 0.5% bupivacaine + 0.2 mL sterile water after ten minutes; twenty mL of 0.9% NaCl was injected intravenously into Group B Dex IT ( $n = 25$ ), which received intrathecal 2.4 mL of 0.5% bupivacaine + 0.2 mL (5  $\mu\text{g}$ ) of dexmedetomidine; twenty-five mL of intravenous dexmedetomidine (1  $\mu\text{g}/\text{kg}$ ) in 20 mL 0.9% NaCl over ten minutes, and then intrathecal 2.4 mL of 0.5% bupivacaine + 0.2 mL of sterile water. Sedation score, as well as the onset and progression of motor and sensory blockage, were noted. While the three groups had identical sensory and motor block onsets, Group B Dex IT > Group B Dex IV > Group B ( $P < 0.001$ ) had longer motor recovery and two-segment sensory regression. While drugged, the patients in Group B Dex IV and Group B Dex IT were readily awakened.

We found that the mean duration of onset of sensory blockade was 7.2 minutes in group I and 6.1 minutes in group II. The duration of onset of motor blockade was 3.1 minutes in group I and 3.3 minutes in group II. The duration of recovery from sensory blockade was 215.0 minutes in group I and 161.5 minutes in group II. The duration of recovery from motor blockade was 238.2 minutes in group I and 195.4 minutes in group II. The sedation score was 2.7 in

group I and 3.5 in group II. Reddy et al<sup>11</sup> evaluated the efficacy of intravenous dexmedetomidine premedication with clonidine and placebo on spinal blockade duration, postoperative analgesia and sedation in patients undergoing surgery under bupivacaine intrathecal block.<sup>75</sup> patients of the American Society of Anesthesiologists status I or II, scheduled for orthopedic lower limb surgery under spinal anesthesia, were randomly allocated into three groups of 25 each. Group DE received dexmedetomidine 0.5  $\mu\text{g}/\text{kg}$ (-1), group CL received clonidine 1.0  $\mu\text{g}/\text{kg}$ (-1) and placebo group PL received 10 ml of normal saline intravenously before subarachnoid anesthesia with 15 mg of 0.5% hyperbaric bupivacaine. Onset time and regression times of sensory and motor blockade, the maximum upper level of sensory blockade were recorded. Duration of postoperative analgesia and sedation scores along with side effects were also recorded. The sensory block level was higher with dexmedetomidine ( $T_4 \pm 1$ ) than clonidine ( $T_6 \pm 1$ ) or placebo ( $T_6 \pm 2$ ). Dexmedetomidine also increased the time ( $243.35 \pm 56.82$  min) to first postoperative analgesic request compared with clonidine ( $190.93 \pm 42.38$  min,  $P < 0.0001$ ) and placebo ( $140.75 \pm 28.52$  min,  $P < 0.0001$ ). The maximum Ramsay sedation score was greater in the dexmedetomidine group than other two groups ( $P < 0.0001$ ).

We found that the pain score (VAS) in group I and II was 2.3, 3.2 respectively. At 2 hours, was 1.6 and 4.6, at 6 hours was 2.4 and 5.1, at 12 hours was 2.4 and 5.1 and at 24 hours was 2.1 and 6.2 respectively. Dinesh et al<sup>12</sup> examined how 0.5% hyperbaric bupivacaine affects spinal anesthesia when administered intravenously together with dexmedetomidine. Group D experienced a considerably longer regression time ( $220.7 \pm 16.5$  min) to the modified Bromage scale 0 than group C ( $131 \pm 10.5$  min) ( $P < 0.001$ ). Group D had a higher level of sensory block ( $T 6.88 \pm 1.1$ ) compared to group C ( $T 7.66 \pm 0.8$ ) ( $P < 0.001$ ). Group D exhibited substantially longer durations for both the two-dermatomal regression of sensory blockade ( $137.4 \pm 10.9$  min vs.  $102.8 \pm 14.8$  min) and sensory block ( $269.8 \pm 20.7$  min vs.  $169.2 \pm 12.1$  min) when compared to group C ( $P < 0.001$ ). Group D had greater intraoperative Ramsay sedation scores ( $4.4 \pm 0.7$ ) than group C ( $2 \pm 0.1$ ) ( $P < 0.001$ ). Compared to group C, a greater percentage of patients in group D (33% vs. 4%;  $P < 0.001$ ) developed bradycardia. In

comparison to group C, group D had a lower mean 24-hour analgesic demand and a longer delay to initially request a postoperative painkiller ( $P < 0.001$ ).

## CONCLUSION

Authors found that arousable sedation produced by subarachnoid anesthesia is prolonged by intrathecal dexmedetomidine.

## REFERENCES

1. Shaikh SI, Dattatri R. Dexmedetomidine as an adjuvant to hyperbaric spinal bupivacaine for infra-umbilical procedures: A dose-related study. *Anaesth Pain Intensive Care*. 2014;18:180–5.
2. Kubre J, Sethi A, Mahobia M, Bindal D, Narang N, Saxena A. Single dose intravenous dexmedetomidine prolongs spinal anesthesia with hyperbaric bupivacaine. *Anesth Essays Res*. 2016;10:273–7.
3. Esmaoğlu A, Sümeýra TÜ, Bayram A, Aynur AK, Fatih UĞ, Ülgey A. The effects of dexmedetomidine added to spinal levobupivacaine for transurethral endoscopic surgery. *Balkan medical journal*. 2013 Jun 1;2013(2):186-90.
4. Annamalai A, Singh S, Singh A, Mahrous DE. Can intravenous dexmedetomidine prolong bupivacaine intrathecal spinal anesthesia? *J Anesth Clin Res*. 2013;4:1–5.
5. Songir S, Kumar J, Saraf S, Waindeskar V, Khan P, Gaikwad M. Study of the effect of intrathecal dexmedetomidine as an adjuvant in spinal anesthesia for gynecological surgery. *Int J Med Res Rev*. 2016;4:602–7.
6. Nethra SS, Sathesha M, Aanchal D, Dongare PA, Harsoor SS, Devikarani D. Intrathecal dexmedetomidine as adjuvant for spinal anesthesia for perianal ambulatory surgeries: A randomised double-blind controlled study. *Indian J Anaesth*. 2015;59:177–81.
7. Singh AK, Singh Y, Jain G, Verma RK. Comparison of two different doses of intrathecal dexmedetomidine as adjuvant with isobaric ropivacaine in lower abdominal surgery. *Anesth Essays Res*. 2015;9:343–7.
8. Kumari R, Kumar A, Kumar S, Singh R. Intravenous dexmedetomidine as an adjunct to subarachnoid block: A simple effective method of better perioperative efficacy. *Journal of anaesthesiology, clinical pharmacology*. 2017 Apr;33(2):203.
9. Harsoor SS, Rani DD, Yalamuru B, Sudheesh K, Nethra SS. Effect of supplementation of low dose intravenous dexmedetomidine on characteristics of spinal anaesthesia with hyperbaric bupivacaine. *Indian Journal of Anaesthesia*. 2013 May;57(3):265.
10. Sharma A, Varghese N, Venkateswaran R. Effect of intrathecal dexmedetomidine versus intravenous dexmedetomidine on subarachnoid anesthesia with hyperbaric bupivacaine. *Journal of Anaesthesiology, Clinical Pharmacology*. 2020 Jul;36(3):381.
11. Reddy VS, Shaik NA, Donthu B, Sannala VK, Jangam V. Intravenous dexmedetomidine versus clonidine for prolongation of bupivacaine spinal anesthesia and analgesia: A randomized double-blind study. *Journal of anaesthesiology, clinical pharmacology*. 2013 Jul;29(3):342.
12. Dinesh CN, Tej NS, Yatish B, Pujari VS, Kumar RM, Mohan CV. Effects of intravenous dexmedetomidine on hyperbaric bupivacaine spinal anesthesia: A randomized study. *Saudi Journal of Anaesthesia*. 2014 Apr;8(2):202.