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# EFFECT OF INTRAVENOUS DEXMEDETOMIDINE ON DURATION OF SPINAL ANESTHESIA WITH HYPERBARIC BUPIVACAINE

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**ABSTRACT:**

**Background:** One of the often employed anaesthetic techniques for lower limb procedures is the sub arachnoid block. Several adjuvants can extend the time and quality of the block. **Aim:** This study assessed the onset and duration of sensory and motor block, hemodynamic effect, postoperative analgesia, and side effects of dexmedetomidine administered intravenously together with hyperbaric intrathecal 0.5% bupivacaine. **Materials and Methods:** This study was carried out on 60 patients with ASA physical status 1 and 2, of either sex, 18–60 years of age, who were scheduled for elective lower limb procedures under spinal anaesthesia. Group 1 received 15 mg of hyperbaric bupivacaine, and Group 2 received 15 mg of hyperbaric bupivacaine + intravenous dexmedetomidine 0.5 microgram/kg in 10ml normal saline as a bolus dose. **Results:** The length of the sensory block, the motor block, and the duration of the analgesia were all significantly longer in the dexmedetomidine group than they were in the patients who just had a 15 mg Bupivacaine subarachnoid block. Dexmedetomidine group showed a significant reduction in heart rate, systolic blood pressure, and mean arterial pressure. **Conclusion:** Prior to subarachnoid block, a single dose of intravenous dexmedetomidine at 0.5 g/kg over 10 minutes increases the duration of sensory and motor blockage with arousable sedation but not respiratory depression.

**Keywords**: Dexmedetomidine, Analgesia, motor blockade

## INTRODUCTION:

It is possible for regional anesthesia and analgesia to offer top-notch operating conditions and protracted postoperative pain relief. In addition, it is well known to increase postoperative blood flow, tissue functionality, and healing, which contributes to its widespread use by lowering postoperative morbidity and mortality. The most often used localised treatment for lower back pain is the sub arachnoid block. Lower limb and abdominal procedures benefit greatly from its low cost and simplicity of administration. The sub arachnoid block offers a number of benefits, including simplicity, quick action, low failure rate, cost-effectiveness, superior level of blockade, and efficient muscular relaxation.[1] As a local anaesthetic, hyperbaric bupivacaine is frequently used to give sub arachnoid blocks. Early analgesic management is required in the postoperative period since subarachnoid block with local anaesthesia alone has a relatively short duration of action. To raise the quality Opioids, ketamine, midazolam, clonidine, dexmedetomidine, and other adjuvants have been used intravenously and intrathecally to blockade.[2] However, these adjuvants, particularly opioids, are linked to adverse reactions include pruritus, respiratory depression, urine retention, postoperative nausea, and vomiting. In order to increase the effects of local anaesthesia without causing respiratory depression, alpha 2 agonists have lately been employed as adjuvants. Dexmedetomidine, a selective 2 adreno receptor agonist with analgesic and sedative effects when administered as an adjuvant in regional anaesthesia, was used in our investigation. As there haven't been many trials utilising the same dosage of dexmedetomidine, we evaluated the quality, length of the block, and amount of time needed for supplemental analgesia in this study. Easy to give, the intravenous method is the only a lower dose is linked to fewer adverse effects. The purpose of this study was to assess the onset and duration of sensory and motor block, hemodynamic effect, postoperative analgesia, and side effects of dexmedetomidine administered intravenously together with hyperbaric intrathecal 0.5% bupivacaine.

## MATERIALS AND METHODS

The prospective comparative study was carried out on 60 patients with ASA physical status 1 and 2, of either sex, 18–60 years of age, who were scheduled for elective lower limb procedures under spinal anaesthesia after receiving written informed permission and clearance from the hospital ethics committee. **Inclusion criteria:** 18–60 years of age of both genders with ASA physical status 1 and 2, scheduled for elective lower limb procedures under spinal anaesthesia.

**Exclusion criteria:** Patients with a local infection at the block site, severe hypovolemia, elevated intracranial pressure, spinal deformities, bleeding and clotting disorders, allergies to local anaesthetics, patients with a history of chronic headache, pregnant women, patients with pre-existing hepatic and renal diseases, and patients with deformities .

Computer generated random tables were used to divide the patients into two groups at random. Ten minutes before the Sub Arachnoid block,

Group 1 received 15 mg of hyperbaric bupivacaine, and

Group 2 received 15 mg of hyperbaric bupivacaine + intravenous dexmedetomidine 0.5 microgram/kg in 10ml normal saline as a bolus dose.
Following pre-anesthesia during the evaluation, pertinent tests were performed, including an electrocardiogram (ECG), a chest x-ray, a urine routine, and a urine microscopy. These tests included haemoglobin estimation, total count, differential count, platelet count, liver function tests, renal function tests, serum electrolytes, coagulation profile, random blood sugar, and electrolytes. At 10 PM the night before surgery, all patients received oral premedication of Tab. Pantoprazole 40 mg and Tab. Alprazolam 0.25 mg. Patients, surgeons, anesthesiologists, nurses, and the researcher gathering the data were all unaware of the medication being used. Intraoperative \smonitoring included ECG, non-invasive blood pressures(NIBP) and pulse oximetry. The skilled anaesthetist administered sub arachnoid block to subjects who were ASA1 and ASA2 listed for lower limb procedures after obtaining ethical committee approval.
Additionally, he or she administered intravenous dexmedetomidine 0.5 mg/kg body weight in 10 ml of normal saline over a 10-minute period. 60 adult patients in this study, who were undergoing spinal anaesthesia for elective lower limb procedures, ranged in age from 18 to 50. Patients who received intravenous dexmedetomidine alone or in conjunction with sub arachnoid occlusion were examined until a total of 30 instances were obtained in each group. Using a computer-generated randomization table, the patients were divided into the control group and the dexmed group at random.An extensive pre-operative evaluation that comprised a full history, general physical examination, systemic examination, and laboratory investigations was performed at the time of admission. After thoroughly explaining the procedure, benefits, and risks to the patient in their native language, a written informed consent was obtained. Alprazolam 0.5 mg was administered to the patient the day before and the morning of the procedure. Basic physiological measurements, including heart rate, blood pressure, and SpO2, were taken. Before intrathecal placement of bupivacaine in Group 2, a bolus dose of 0.5 g/kg body weight

Dexmedetomidine in 10 ml Normal Saline was administered intravenously over a 10-minute period. After ensuring that the CSF was flowing freely, 3 ml of 0.5% heavy bupivacaine (15 mg) was injected intrathecally under aseptic conditions with the patient seated and at the L3-L4 or L4-L5 interspace. Immediately following the delivery of intrathecal drugs, patients were lying on their backs. The beginning of the sensory and motor blockade, the peak degree of the sensory blockade, and the duration of the blockade were all noted intraoperatively. Five-point sedation scale evidence was found. Every 2 minutes for the first 20 minutes, every 5 minutes until the end of the procedure, and then every 30 minutes after that, the following measurements were taken: HR, NIBP, ECG, and SpO2. Additionally, the duration of analgesia, sensory and motor blockage, as well as any negative side effects including nausea, vomiting, shivering, headaches following a spinal puncture, bradycardia, hypotension, etc., were observed dealt appropriately.

It was performed by one anaesthesiologist who was blinded to the group assignment. MAP, HR, RR, and peripheral oxygen saturation (SpO2) were recorded at each time point as follows; T1 = preoperative baseline, T2 = anaesthesia start, T3 and T4 = 5 and 10 min after anaesthesia, T5 = operation start, T6, T7, and T8 = 5, 10, and 15 min after operation, T9=postoperative value. Moreover, the incidence of adverse events including hypertension, hypotension, bradycardia (HR< 50 beats/min), respiratory depression (RR<10 breaths/min), and oxygen desaturation (SpO2<93%) were evaluated.

Using a visual analogue pain score (VAS) between 0 and 10 (0 = no pain, 10 = most severe pain), the length of analgesia was measured. When the patients started to feel uncomfortable pain (VAS 4), rescue analgesic (IM Tramadol 2mg/kg) was administered. Continual checks were made for hemodynamic alterations as well. For the study's purposes, a heart rate of fewer than 40 beats per minute was deemed bradycardic, and any instances of it were treated with an IV injection of 0.6 mg atropine. Systolic blood pressure that was less than 90 mmHg or fell by more than 30% from the baseline value was regarded as hypotension and was treated as needed with IV fluids and ephedrine 6 mg at incremental doses. Until a VAS 4 or higher was reached, postoperative sensory block, motor block, and VAS ratings were recorded in the post-anesthesia care unit every 10 minutes.

All information was gathered, coded, entered into a Microsoft Excel sheet, and examined. In terms of mean deviation +/- standard deviation, the onset and duration of sensory and motor blockage were determined. The independent t-test was used to compare the groups. 0.05 was the threshold for significance. The analysis was carried out utilising SPSS 15.0.

**RESULTS:**

60 patients were selected in this study.

**Table 1: Distribution based on age of patients.**

| **Age in years** | **Groups** | | **Total** |
|---|---|---|---|
| | **Control** | **Dexmedetomidine** | |
| ≤21 | 3 | 2 | 5 |
| 22-31 | 9 | 7 | 16 |
| 32-41 | 4 | 8 | 12 |
| 42-51 | 7 | 6 | 13 |
| 52-61 | 7 | 7 | 14 |
| Total | 30 | 30 | 60 |
| **Gender** | | | |
| Female | 5 | 12 | 17 |
| Male | 25 | 18 | 43 |
| **ASA** | | | |
| 1 | 16 | 18 | 34 |
| 2 | 14 | 12 | 26 |

Table 1 shows that the highest number of patients (16) were present in 22-31 years followed by 52-61 years (14). Both groups were statistically significant. Age in Mean±SD in control group was 39.65±13.54 and in dexmedetomidine group was 39.11±12.45.
Females were 5 in control group and 12 in dexmedetomidine group and males were 25 in control group and 18 in dexmedetomidine group. 16 were present in ASA 1 in control group and 18 were present in ASA 1 in dexmedetomidine group and 14 were present in ASA 2 in control group and 12 were present in ASA 2 in dexmedetomidine group.

**Table-2: Distribution based on weight, height and BMI.**

| | **Groups** | | **P value** |
|---|---|---|---|
| | **Control (Mean±SD)** | **Dexmedetomidine (Mean±SD)** | |
| Height (cms) | 172.34±6.89 | 166.43±9.75 | 0.135 |
| Weight (Kg) | 70.87±1.32 | 69.65±8.69 | 0.232 |
| BMI ($Kg/m^2$) | 24.81±3.26 | 25.75±3.11 | 0.156 |

Table-2 shows that height in cms in control group was 172.34±6.89 and in dexmedetomidine group, it was 166.43±9.75, P value was 0.135, weight in kgs in control group was 70.87±1.32 and in dexmedetomidine group, it was 69.65±8.69, P value was 0.232. BMI in $Kg/m^2$ was 24.81±3.26 in control group and in dexmedetomidine group, it was 25.75±3.11, P value was 0.156.

**Table 5: Duration of sensory blockade, motor blockade and analgesia comparison between two groups.**

| | **Groups** | | **P value** |
|---|---|---|---|
| | **Control (Mean±SD)** | **Dexmedetomidine (Mean±SD)** | |
| Sensory block duration | 168.99±6.54 | 186.13±8.21 | 0.0001 |
| Motor block duration | 165.79±5002 | 193. 01± 6.27 | 0.0001 |
| Analgesia duration | 212.32±8.14 | 240.89±5.87 | 0.0001 |

Table 5 shows that the mean duration of analgesia in control group was 212.32±8.14 minutes and in dexmedetomidine group, it was 240.89±5.87 minutes. With a P value of 0.001, the sensory and motor blockage and analgesia were significantly prolonged in the dexmed group compared to the control group.

**Table-3: Vitals distribution at baseline before administering study drugs.**

| **Vitals** | **Dexmedetomidine** | **Midazolam** | **P value** |
|---|---|---|---|
| Mean SBP | 119± 14 mm of Hg | 120± 17 mm of Hg | 0.93 |
| Mean DBP | 75± 9 mm of Hg | 76± 10 mm of Hg | 0.74 |
| Mean Heart rate | 97± 15 bpm | 96± 16 bpm | 0.9 |
| Mean Respiratory rate | 23± 4 bpm | 22± 6 bpm | 0.4 |
| Mean SpO2 | 93± 5 | 92± 4 | 0.6 |

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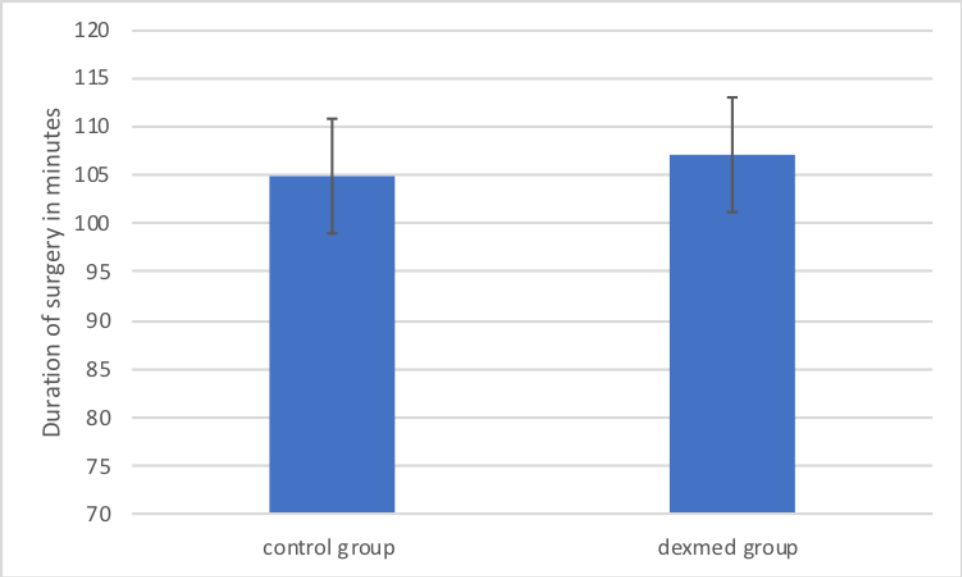
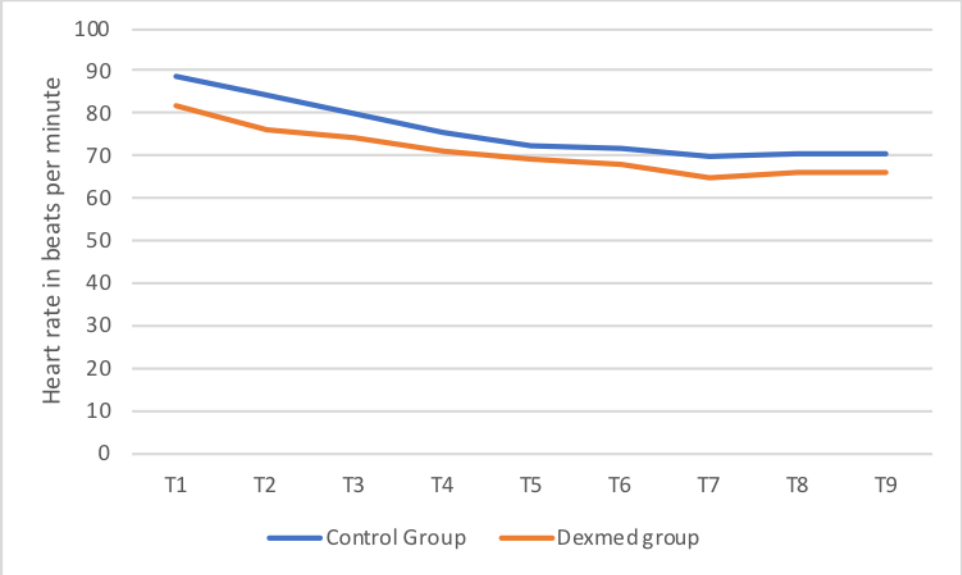
As we can see from the p values above, there was no significant difference in the baseline vitals between the two study groups.

**Figure-1: Comparision of duration of surgery in minutes**



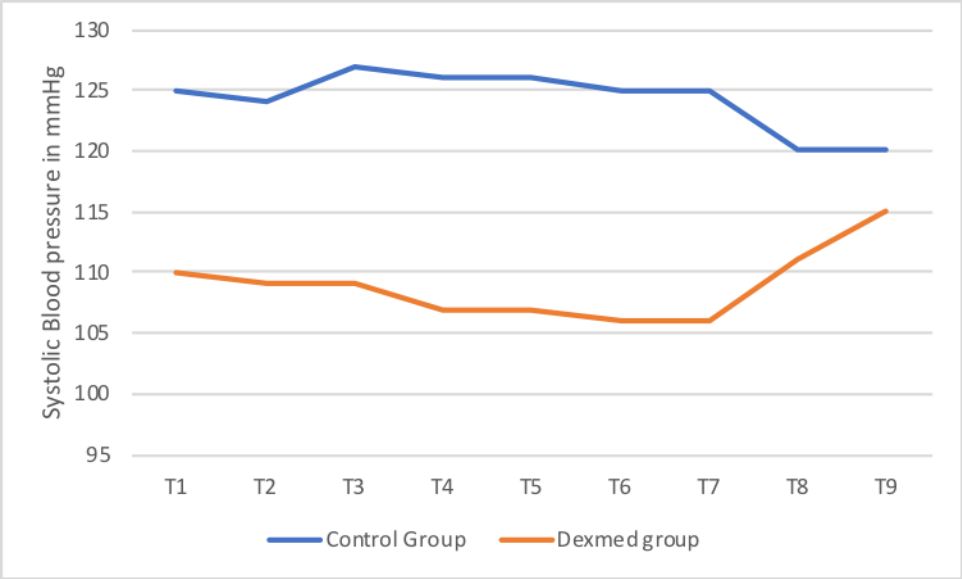

The duration of surgery in control group was 105 minutes and in dexmed group was 107 mins. There was no significant difference in the duration of surgery between the groups (P =0.650).

**Figure-2:comparison of mean heart rate between both the groups**



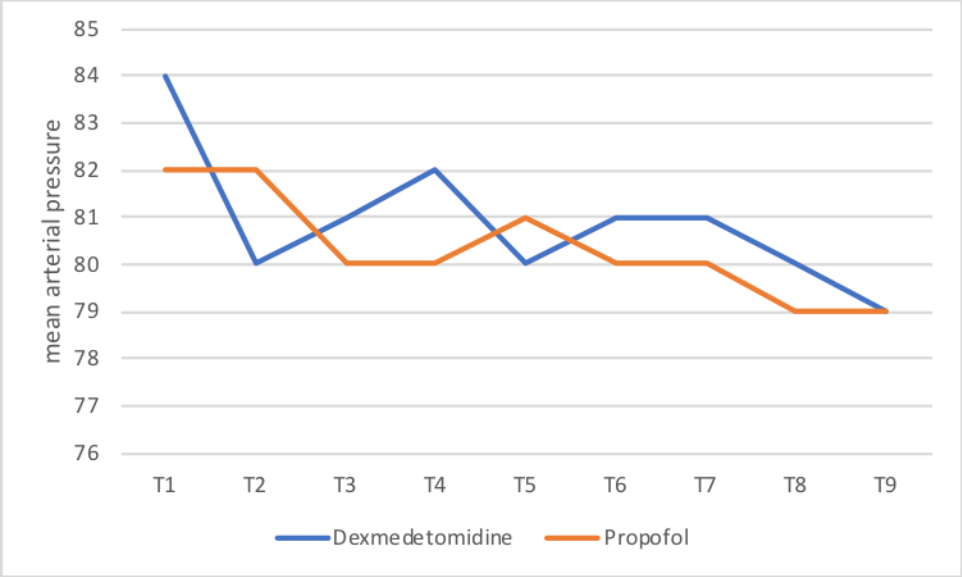

There was significant decrease in heart rate in dexmed group compared to control group (with p value of 0.0001).

**Figure-3: Shows the comparison of Systolic blood pressure between both the groups**




There was also significant decrease in systolic (P = 0.0001)

**Figure-4: Shows the comparison of Diastolic blood pressure between both the groups**




There was no significantchange in the diastolic blood pressure. (P = 0.05).

**Table-4: Comparison of occurrence of adverse events among both the groups**

| Adverse events | Control group | Dexmed group | P Value |
|---|---|---|---|
| Hypotension | 5 | 6 | 0.043 |
| Hypertension | 1 | 3 | 0.3 |

| | | | |
|---|---|---|---|
| Nausea | 4 | 4 | |
| Intraoperative shivering | | | |

5 patients in the control group, 6 patients in the dexmed group experienced hypotension. As a result of bradycardia, 3 patients in the dexmed group received an injection of atropine as opposed to 1 patient in the control group. In both groups, 4 patients experienced nausea. No patients in either group experienced side effects such respiratory depression, vomiting, shivering, itching, or pruritus.

**Table-5: Time to attain sedation and numerical rating scale with satisfaction between both the groups**

| Time to attain sedation | Mean | SD | P value |
|---|---|---|---|
| Control group | 15 | 4 | <0.001 |
| Dexmedetomidine group | 11 | 5 | |
| **Procedure on numerical rating scale association** | | | |
| Control group | 3 | 1.1 | <0.001 |
| Dexmedetomidine group | 5 | 1.7 | |
| **Patients sedation satisfaction during procedure on verbal rating** | | | |
| Control group | 7 | 0.7 | <0.001 |
| Dexmedetomidine group | 4.8 | 0.8 | |

Patients in the dexmed group experienced quicker sedation, which was statistically significant (p=0.0001).

When used as an adjuvant to spinal anaesthesia, intravenous dexmedetomidine in a bolus dose of 0.5 microgram/kg prolongs sensory and motor blockage. Excellent postoperative pain relief is provided by it. It significantly lowers blood pressure and heart rate. When intravenous dexmedetomidine is administered as a bolus dosage of 0.5 microgram/kg over a period of 10 minutes, the frequency of problems such bradycardia, respiratory depression, and hypotension is reduced.

## DISCUSSION:

In order to extend the duration of spinal anaesthesia, various medications have been used as adjuvants. anaesthesia. Dexmedetomidine, a 2 agonist, works by attaching to 2 receptors in the brain to induce drowsiness and anxiolysis. Locus Ceruleus, which suppresses sympathetic activity and reduces norepinephrine release, lowers heart rate and blood pressure. By attaching to spinal cord adrenoreceptors, it alleviates pain. Dexmedetomidine has a significant impact on the onset and duration of spinal anaesthesia when used as an adjuvant, either intrathecally or intravenously. Dexmedetomidine's adverse effects, including bradycardia and hypotension, are

dose-dependent. The occurrence of these side effects is reduced when the loading dosage is administered as an infusion over ten minutes. In our study, we examined the effectiveness of spinal anaesthesia using bupivacaine alone against spinal anaesthesia employing intravenous dexmedetomidine as an adjuvant. In the current study, 60 patients were evaluated and were undergoing lower limb procedures under spinal anaesthesia who were between the ages of 18 and 50 and belonged to ASA classes I and II. Dexmedetomidine bolus dose of 0.5 g/kg/hr was administered intravenously over a 10-minute period to 30 patients who met the inclusion criteria prior to spinal anaesthesia, while the other 30 patients did not. The following outcomes were evaluated: onset of sensory and motor blockade, peak level of block, duration of sensory and motor blockade, length of analgesia, and hemodynamic modifications and difficulties. Both groups had comparable demographic information, surgery length, and ASA grading. The findings of our study demonstrate that, in comparison to spinal anaesthesia with bupivacaine alone, intravenous dexmedetomidine bolus administration of 0.5mcg/kg over 10 minutes followed by sub arachnoid block with hyperbaric bupivacaine 15mg considerably prolonged both sensory and motor block. In senior intensive care patients, Sule Akin et al.[3] investigated the effects of intravenous dexmedetomidine as an adjuvant to epidural analgesia. Patients in the treatment group received a 0.6 g/kg loading dose spread out over 30 minutes, followed by a 0.2 g/kg/hr continuous infusion. When compared to the control group with superior hemodynamic stability, they found that the visual analogue scale ratings in the dexmed group were significantly lower. A research by Mi Hyeon Lee et al.[4] compared two bolus doses of intravenous dexmedetomidine (0.5 g/kg and 1 g/kg) given as an adjuvant to spinal anaesthesia to a control group that only received spinal anaesthesia. They discovered that there were no statistically significant variations in the length of spinal anaesthesia between the two dexmedetomidine bolus groups, and that both bolus dosages of intravenous dexmedetomidine enhanced the duration of spinal anaesthesia. In a study by Chilkunda N. et al.[5], two groups—one getting a dexmedetomidine infusion of 1 g/kg bolus dose followed by 0.5 g/kg/hr infusion and the other receiving a normal saline infusion—were used to examine the effects of intravenous dexmedetomidine on hyperbaric bupivacaine spinal anaesthesia. They discovered that intravenous dexmedetomidine increases the duration of bupivacaine spinal anesthesia's sensory and motor blockage while lowering heart rate, mean/systolic/diastolic blood pressure, and providing good sedation and less post-operative shivering. As a result, we decided to utilise a lower dose of dexmedetomidine, a 0.5mcg/kg bolus dose, as an adjuvant to 0.5% hyperbaric bupivacaine. Age in Mean±SD in control group was 39.65±13.54 and in dexmedetomidine group was 39.11±12.45. The majority of patients in each group were between the ages of 52 and 61. Height in cms in control group was 172.34±6.89 and in dexmedetomidine group, it was 166.43±9.75, P value was 0.135, weight in kgs in control group was 70.87±1.32 and in dexmedetomidine group, it was 69.65±8.69, P value was 0.232. Regarding age, height, and weight, there was no statistically significant difference between the two groups. Sule Akin et al.'s[6] investigation of the effects of intravenous dexmedetomidine as a supplement to epidural

analgesia in senior intensive care patients, patients with a mean age of 75.66±3.86years—found promising results. On subjects with a similar age distribution and anthropometric measurements to those in our study, Chilkunda N et al,[5] Deepika Shukla et al[6], and SS Harsoor et al,[7] conducted studies on the effects of intravenous dexmedetomidine on spinal block with bupivacaine. Both groups experienced sensory and motor blocks at roughly the same time. The results of Faraj W. Abdullah et al.[8] and Agarwal S. et al.[9], who showed no difference in the onset time between patients receiving dexmedetomidine infusion as an adjuvant to spinal anaesthesia and spinal anaesthesia with only bupivacaine, were in agreement with these findings. The commencement of the sensory and motor block between the dexmedetomidine group and the control group did not differ significantly, according to Mi Hyeon Lee et al[4]. In the current investigation, there was no significant difference between the groups, and the majority of the patients in both groups experienced sensory block at the T6 level. This was in line with the conclusions reached by Myoung Hun Kim et al.[10], who found no appreciable variation in the degree of block achieved. However, a similar study conducted by Mi Hyeon Lee et al[4] and SS Harsoor et al[7] revealed that a lower level of block was achieved with dexmedetomidine, perhaps as a result of the intrathecal administration of a smaller dose of bupivacaine (0.5% hyperbaric bupivacaine) at doses of 12 mg and 12.5 mg, respectively. For the current investigation, we used a dose of 15 mg of 0.5% hyperbaric bupivacaine. Dexmedetomidine dosages of 0.5 g/kg and 1 g/kg were tested with bupivacaine doses, and Mi Hyeon Lee et al[4] discovered that the effect was dose dependent on the onset and regression of sensory and motor block. The prolonged duration of the sensory block caused by the intravenous 0.5 g/kg bolus dosage of dexmedetomidine employed in our investigation is consistent with the findings of Faraj W Abdullah et al[8] who compared the dexmedetomidine group and control group. Similar studies by Murat Tetkin et al.[11] and Velayudha Sidda et al.[12] demonstrated that an intravenous dexmedetomidine infusion prolonged sensory block. The sensory block is prolonged when intravenous dexmedetomidine is paired with spinal bupivacaine. However, at supra spinal level 2, the effect of intravenous dexmedetomidine in extending sensory blockade of spinal anaesthesia is still unknown. Although the exact mechanism by which intravenous dexmedetomidine prolongs spinal anesthesia's sensory blocking is yet unknown, the locus ceruleus in the brain stem contains a significant concentration of 2 receptors at the supraspinal level. This area gives rise to the medullo-spinal noradrenergic pathway, a crucial regulator of nociceptive neurotransmission. The locus ceruleus receptors that dexmedetomidine interacts with extend the sensory blockage. Additionally, our current investigation demonstrated that the motor block was significantly prolonged in the dexmedetomidine infusion group compared to the spinal anaesthetic group simply. This was equivalent to the findings of Murat Tekin et al.[11] and Faraj W. Abdullah et al.[8] Dexmedetomidine was shown to have a considerably longer duration of motor block compared to the control group in a study identical to this one carried out by Velayudha Sidda et al.[12]. By selectively blocking myelinated A fibres engaged in sensory conduction over unmyelinated C fibres involved in motor conduction, dexmedetomidine results in a greater degree of differential blockage. Faraj

W. Abdullah et al.[8] and Mi Hyeon Lee et al.[4] had demonstrated that dexmedetomidine infusion considerably extended the duration of analgesia. The findings of the current study support those of the aforementioned authors because our prolonged duration of analgesia was statistically significant. Dexmedetomidine infusion as an adjuvant to spinal anaesthesia increased the duration of analgesia in comparison to solitary spinal anaesthesia, as shown by SS Harsoor et al[7]., Velayudha Sidda et al.[12], and others. The dorsal horn's 2C and 2A receptors are stimulated, which inhibits the release of substance P and glutamate, two pro-nociceptive transmitters. The analgesic effect is additionally sustained by hyperpolarization of the unmyelinated C fibres (sensory). In our study, we discovered that intraoperative systolic blood pressure and mean arterial pressure were statistically significantly lower in the dexmedetomidine group than in the control group. The results of Mi Hyeon Lee et al.[4] and Chilkunda et al.[5], which showed a substantial difference in blood pressure variation between the two groups, were in agreement with this finding. However, research by Harsoor et al[7]. and Murat Tekin et al[11]. found that the difference in blood pressure was not statistically significant. This finding was in contrast to other studies. The heart rate in the dexmed group was significantly lower than in the control group, according to the current study. Most investigations have identified bradycardia as a significant adverse effect, with incidences ranging from 30% to 40% and occasionally requiring atropine treatment. However, because a smaller bolus dose was employed in our trial, the incidence of bradycardia was minimal, which is consistent with the findings of Harsoor et al[7]. and Faraj W Abdullah et al[8]. In contrast to the control group, our study found that the dexmedetomidine group experienced less intraoperative shivering and higher sedation. These results agreed with those of Murat Tekin and colleagues[11]. There was no discernible difference in intraoperative shivering between the dexmed and control groups, according to studies by Chilkunda et al[5]. and Myoung et al[10]. Dexmedetomidine used intravenously prolongs the effects of local anaesthesia while also sedating the patient.

## CONCLUSION

When used as an adjuvant to spinal anaesthesia, intravenous dexmedetomidine in a bolus dose of 0.5 microgram/kg prolongs sensory and motor blockage. Excellent postoperative pain relief is provided by it. It significantly lowers blood pressure and heart rate. When intravenous dexmedetomidine is administered as a bolus dosage of 0.5 microgram/kg over a period of 10 minutes, the frequency of problems such bradycardia, respiratory depression, and hypotension is reduced.

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