

PROSPECTIVE RANDOMIZED DOUBLE BLINDED PLACEBO CONTROLLED STUDY TO EVALUATE THE EFFECTS OF INTRAVENOUS DEXMEDETOMIDINE ON SPINAL BUPIVACAINE ANAESTHESIA

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ABSTRACT

BACKGROUND AND AIMS

There is a dearth of studies on the effect of intravenously administered Dexmedetomidine in Sub Arachnoid Block hence, this study was conducted to compare the effects of intravenously administered Dexmedetomidine prior to the administration of subarachnoid block with bupivacaine 0.5% heavy, on hemodynamic variables and the level and onset and duration of sensory and motor blockade.

MATERIAL AND METHODS

After obtaining ethical Committee approval, a double-blind, randomized prospective clinical study was conducted on 90 American Society of Anesthesiologist Grade I and II patients in the age group of 18-55 years, divided randomly into two groups: Group D received 50 ml solution containing Inj. Dexmedetomidine infusion at 0.5 mcg/kg for 10 minutes, and Group P received 50 ml of solution 0.9% Normal Saline as infusion at 10 minutes time. Subsequently Spinal Anaesthesia is carried out with Bupivacaine heavy 0.5%, and carried out recordings as per protocol. Besides Hemodynamic parameters other parameters observed were effectiveness, Sedation score; highest level of sensory block achieved; Motor and Sensory block; Time for first rescue analgesic requirement were recorded.

RESULTS

Group D (n=45) (Mean±SD) Sedation Score 3.42±0.621 and Group P (n=45) (Mean±SD) Sedation Score 1.80±0.405. Better sedation was seen in Group – D with a p value of less than 0.0001. The highest level of sensory blockade achieved was significantly higher in Group D when compared to the control group, and mean duration (in minutes) to achieve the highest sensory blockade in both the groups: Group D (n=45) (Mean±SD) 7.91±2.42 and Group P (n=45) (Mean±SD) 9.82±3.973 p Value < 0.0001. The highest level of sensory blockade achieved was significantly higher in Group D when compared to the control group, and mean duration (in minutes) to achieve the highest sensory blockade in both the groups. Group D (n=45) (Mean±SD) 7.91±2.42 and Group P (n=45) (Mean±SD) 9.82±3.973 p Value < 0.0001. Sensory Block duration in Group D- (n=45) (Mean±SD) 76.18 and in Group P - (n=45) (Mean±SD) 53.44, with p-value = <0.00001. Motor Block duration in Group D- (n=45) (Mean ±SD) 126.42 and in Group P - (n=45) (Mean±SD) 103.64, with p-value= <0.00001.

CONCLUSION

Dexmedetomidine with stable hemodynamic variables, there is significantly prolonged both the sensory and motor block duration with no associated significant changes in the. The level of sensory block attained was significantly higher in the study group and was attained more quickly than the control group. All patients achieved better sedation levels over the entire intraoperative course. It provided good post-operative analgesia with the time to the requirement of the first rescue analgesic being significantly longer.

KEYWORDS

Dexmedetomidine, Bupivacaine, Subarachnoid block.

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INTRODUCTION: Subarachnoid block has progressed greatly since 1885 and is used successfully in a number of different clinical situations. Subarachnoid Block is a well-

known technique used in various surgical procedures involving the supra/ infraumbilical regions and the lower limb. Subarachnoid Block, however, may promote some type of discomfort caused by the procedure itself or by a prolonged perioperative period paresis or paralysis, requiring the simultaneous administration of hypnotic, sedative and anxiolytic drugs. Benzodiazepines, Propofol and opioids have these properties and provide some comfort to patients. However, they affect the ventilator regulatory mechanisms and may lead to respiratory depression, with consequent

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hypercarbia and hypoxemia. Different agents, like Epinephrine, Phenylephrine, Adenosine, Magnesium Sulphate and Clonidine have been used as adjuncts to local anaesthetic drugs for prolonging the duration of subarachnoid block via the intrathecal route. 1. Alpha. 2. Adrenoceptor agonists are being increasingly used in critical care and anaesthesia. Besides sedation and analgesia, they also decrease sympathetic tone and attenuate the stress responses to anaesthesia and surgery. Hence, they are used as adjuvant drugs during regional and general anaesthesia to attenuate the pressor response to Endotracheal Intubation or to prolong the duration of regional anaesthesia.

Dexmedetomidine is the most recent agent in this group approved by FDA in 1999 for use in humans for analgesia and sedation in the intubated patients at the intensive care settings.¹ Dexmedetomidine^{2,3} is a novel clonidine-like compound known to have sedative, analgesic and cardiovascular stabilizing qualities. Kanazi et al. added dexmedetomidine 3 µg or clonidine 30µg to intrathecal bupivacaine, and were able to prolong duration of motor and sensory block with preserved hemodynamic stability and lack of sedation⁴. Sudo et al. were able to conclude that Dexmedetomidine prolongs spinal anaesthesia induced by Levobupivacaine 0.5% in guinea-pigs⁵. Dexmedetomidine was used intravenously as adjuvant agent to support spinal Prilocaine and significantly prolonged the sensory and motor block^{6,7,8}. However there is a dearth of studies that actually evaluated the effect of intravenously administered dexmedetomidine on the duration of sensory and motor block after intrathecal administration of Bupivacaine 0.5% and its subsequent effects on the haemodynamics, sedation and postoperative analgesic requirements. Hence, the objectives of this study were to compare the effects of intravenously administered dexmedetomidine prior to the administration of subarachnoid block with bupivacaine 0.5% heavy and the effects of intrathecal Bupivacaine 0.5% alone, on the level and onset of sensory and motor blockade and the duration of the sensory and motor blockade and its associated effects on sedation and the hemodynamic parameters of the patient. The objective of the present study is to compare the anaesthetic and hemodynamic effects of intravenously administered dexmedetomidine prior to the administration of subarachnoid block with Bupivacaine 0.5% heavy and the effects of intrathecal Bupivacaine 0.5% alone, on the level and onset of sensory and motor blockade and the duration of the sensory and motor blockade.

MATERIALS & METHODS: The study was approved by the hospital ethics committee of Hospital and informed consent was obtained from the patients. The study design is a prospective, placebo controlled, parallel group, randomized, and double blinded type.

The study population is ninety American Society of Anaesthesiologist (ASA) I & II adult patients.

1. Inclusion Criteria:

- a. ASA physical status I and II patients.
- b. Elective lower limb orthopaedic surgeries (open reduction with internal fixation surgeries of the tibia, ankle and foot, closed reduction and internal fixation surgeries of the foot, ankle and tibia, external fixator removal, Ilizarov's ring fixation and realignment, wound debridement and split skin graft surgeries of the lower limbs, implant exit surgeries of femur, tibia and ankle).
- c. Age above 18 years.

2. Exclusion Criteria:

- a. Emergency surgeries.
- b. American society of Anaesthesiologist (ASA) physical status III and IV patients.
- c. Patients using alpha 2 receptor antagonists, calcium channel blockers and angiotensin converting enzyme inhibitors.
- d. Patients with ventricular/ supraventricular arrhythmias, heart blocks.
- e. Use of any opioid or sedative in the week prior to surgery.
- f. History of alcohol or drug abuse.
- g. Allergy to the local anaesthetic or the study drug being used.
- h. Failed spinal subarachnoid block.
- i. Prolonged duration of surgery requiring supplementation with other anaesthetic / analgesic medications or conversion to general anaesthesia.

3. Randomization and Blinding Patients are allocated randomly to the two groups, Group D (Dexmedetomidine) and Group P (Placebo) using a computer generated random numbers table when they are received in the preoperative area. An anaesthesiologist and physiologist not involved in the study prepares syringes containing either dexmedetomidine or 0.9% saline. Both the syringes look alike, so study and placebo cannot be differentiated. After randomization, for the study group two hundred microgram (200 mcg) of dexmedetomidine in 2 ml is diluted with 48ml of 0.9% saline and prepared to 50ml in an infusion syringe. The concentration of dexmedetomidine is 4 mcg in 1ml of 0.9% saline. For the control group a 50ml 0.9% saline is prepared in an infusion syringe.

4. Study Groups:

- Group D: Study group receiving Inj. Dexmedetomidine infusion at 0.5 mcg/kg for 10 minutes.
- Group P: Control group receiving 0.9% Normal Saline as infusion at the stipulated rate.

5. Theatre Preparation:

- IV Fluids Ringer lactate 500ml solution is prepared and intravenous drip set desired.
- Emergency Drug Tray.
- Inj. Atropine Sulphate 0.6 mg/ ml in a 2 ml syringe.
- Inj. Ephedrine 6mg/ml in a 5 ml syringe.
- Inj. Adrenaline (1:10000) – 10 ml syringe.
- Anaesthesia machine checking which includes both open circuit and closed circuit.
- Intubation Trolley: - Appropriate sized Mask, Airway, Endotracheal tube and working Laryngoscope and Ryle's tube.
- Suction Apparatus.
- Trolley with the necessary items for the subarachnoid block. (25g Quincke needle, 0.5% hyperbaric bupivacaine solution. (4ml), 2% lignocaine vial, 5ml syringe, 2 ml syringe, adequate size gloves and a spinal tray).

6. Monitoring: Once the patient shifted inside operation theatre.

- Pulse oximeter probe was attached.
- O₂ by mask 6L/min is started.
- ECG connected – Leads II was monitored.
- Non-invasive blood pressure monitor cuff was tied to the arm.
- 18 G peripheral venous line was secured preferably on the dorsum of the left hand.
- The base-line heart rate, systolic, mean and diastolic blood pressures, with oxygen saturation were noted.

Study drug infusion was commenced in a double-blinded fashion. The patient received either Dexmedetomidine (0.5mcg/kg) or 0.9% saline (0.5mcg/kg) as a 10 minutes as infusion. The infusion rate would be calculated presuming that both the syringes contain the study drug. Vital signs were recorded and represented as HR, MAP, SpO₂ and RR. Vital signs along with the oxygen saturation (SpO₂) were recorded at 5 minutes and 10 minutes after the infusion has been started. The infusion was then stopped.

We had defined the following parameters for the study.

1. Hypotension was defined as systolic blood pressure <30% of baseline value or less than 90mmHg, whichever was lower.
2. Tachycardia was defined as heart rate >25% of baseline value.
3. Bradycardia was defined as heart rate < 60 beats/min with associated hemodynamic instability.
4. An arrhythmia was defined as any rhythm other than normal sinus rhythm.

Administering the Subarachnoid Block: Subarachnoid block was performed with aseptic technique by a trained anaesthesiologist in the sitting position through the L3-L4 or L4-L5 space using a 25-G Quincke needle, 3 ml of hyperbaric Bupivacaine 0.5% is to be injected with a rate of 0.2

ml/ second in all patients. After administration of subarachnoid block patients were laid back to the supine position and received oxygen 3 L/min via a face mask throughout the procedure. After performing the subarachnoid block, the vital signs along with oxygen saturation (SpO₂) were recorded every 5 minutes in the operation room and every 15 minutes in the Post Anaesthesia Care Unit (PACU) until the patient was discharged to the ward, after having achieved complete reversal of sensory and motor block.

Assessment of the Sensory and Motor Block: Sensory blockade was assessed using pinprick and cold (iced cube) in the mid-axillary line. Recovery time for sensory blockade is defined as two-dermatome regression of anaesthesia from the maximum level. Motor block is assessed immediately after sensory block assessment using a Modified Bromage Scale.

MODIFIED BROMAGE SCALE 60:

- 0 = no paralysis;
- 1 = unable to raise extended leg.
- 2 = unable to flex knee.
- 3 = unable to flex ankle.

Motor block duration is the time for return to Modified Bromage Scale score of 1. Sensory and motor block were assessed every 2 min for the first 10 min and thereafter every 10 min during surgery and postoperatively. The highest sensory block level and recovery time of both sensory and motor block were recorded.

The Ramsay sedation score is used for assessment of level of sedation.

RAMSAY SEDATION SCORE 61:

- 1 = anxious and agitated.
- 2 = cooperative and tranquil.
- 3 = drowsy but responsive to command.
- 4 = asleep but responsive to a glabellar tap.
- 5 = asleep with a sluggish response to tactile stimulation.
- 6 = asleep and no response.

The score was evaluated every 10 min.

Excessive sedation was defined as a score greater than 4/6.

Hypotension was defined as a systolic blood pressure of less than 30% of the baseline and if occurred, was treated with a bolus administration of 300 ml of Ringer's solution over 5 min and 6 mg of intravenous ephedrine.

Bradycardia was defined as a heart rate < 60 bpm, and if occurred or haemodynamically unstable was treated with 0.6 mg of intravenous atropine.

The study hence analyses the following parameters:

1. Physiological variables including heart rate mean arterial pressure, oxygen saturation and respiratory rate.
2. Sedation score.

3. Highest level of sensory block achieved.
4. Time to attain the highest sensory block.
5. Motor and sensory block duration.
6. Time to the first rescue analgesic requirement.
7. Observed adverse outcomes.

STATISTICAL METHODS: Statistical analysis was done using SPSS (Statistical Package for the Social Sciences) 22 software. Data was expressed as either mean and standard deviation or numbers and percentages. The demographic data of patients were studied for both the groups. The means of the continuous variables (Age, BMI) were compared between the two groups using analysis of variance ANOVA, while the demographic data for the categorical variables (sex, ASA class) were compared using chi-square test. The P value of <0.05 was considered significant. Power analysis was done with a beta error of 0.8 and alpha error of 0.05 and the sample size was calculated to be 45 in each group.

RESULTS:

Study groups:

Group D – Dexmedetomidine GROUP (N = 45) - Study Group.

Group P – 0.9% Normal Saline GROUP (N = 45) - Control Group.

DEMOGRAPHICAL DATA: There were no significant differences between the two groups with respect to Age, Sex, and Body Mass Index (BMI).

1. **Age & Body Mass Index:** The mean age and body mass index was comparable in both the groups. The data was analysed using one way ANOVA test (Table1).

	Group – D (n=45) (Mean±SD)	Group – D (n=45) (Mean±SD)	p Value
Age (Yrs)	36.38±12.570	36.63±14.55	0.932
BMI (Kg/Sq.M)	28.1±4.6	26.6±3.7	0.23

Table 1: Mean age and body mass index in both groups

2. **Sex:** There was no significant difference in male and female percentage distribution between the two groups. The data was analysed using Pearson Chi Square test, with p value of 0.215 (not significant). (Table2)

	Group D(n=45)	Group P(n=45)	p Value
Male	37(82.2%)	41(91.1%)	0.215
Female	8(17.8%)	4(8.9%)	

Table 2: Percentage distribution of sex in both groups

Physiological Variables: There was no significant difference in the hemodynamic variables (Heart rate, Mean Arterial Pressure), Oxygen saturation and Respiratory rate assessed before the start of infusion, after the Dural puncture, perioperative and at the end of the surgery. Data were analysed and compared using one way ANOVA test with p values statistically insignificant. (Table 3, 4 5, 6)

Heart Rate (beats per minute)	Group – D n=45 (Mean±SD)	Group – D n=45 (Mean±SD)	p Value
Baseline	91.44±9.423	85.93±10.248	0.246
At the end of Infusion	83.5 ±14.099	80.5±11.128	0.682
After Dural puncture	82.41±9.076	78.7±10.692	0.372
End of Surgery	88.35±11.256	91.68±11.125	0.572

Table 3: Comparison of mean heart rate in between the groups at various intervals

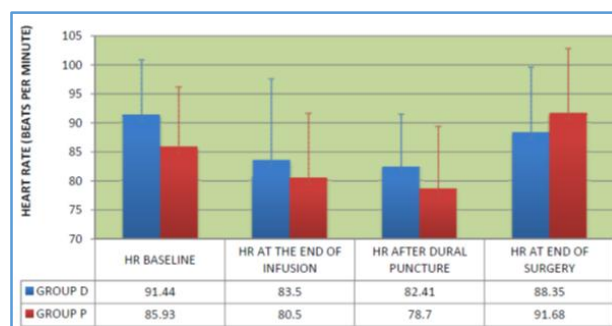


Figure 1: Bar Diagram

Mean arterial pressure (mm of Hg)	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Baseline	95.11±14.952	89.69±7.698	0.316
At the end of Infusion	82.85±11.579	92.05±13.121	0.076
After dural puncture	83.53±16.071	89.45±11.009	0.438
End of surgery	92.23±12.368	90.56±9.124	0.489

Table 4: Comparison of mean arterial pressure (MAP) in between the groups at various intervals

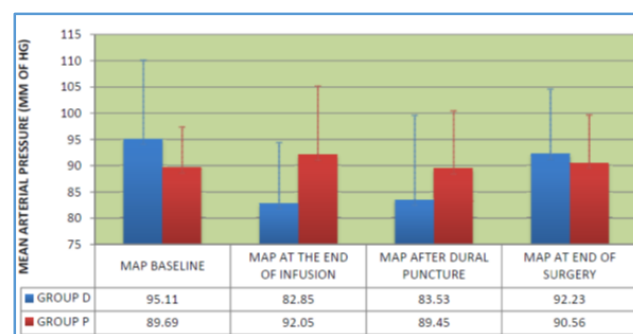


Figure 2: Bar Diagram

Oxygen Saturation (%)	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Baseline	99.11±0.11	98.51±1.121	0.155
At the end of infusion	99.9 98±0.089	99.95±0.05	0.256
After dural puncture	99.85±0.089	99.94±0.05	0.389
End of surgery	100	100	NS

Table 5: Comparison of percentage oxygen saturation in between the groups at various intervals

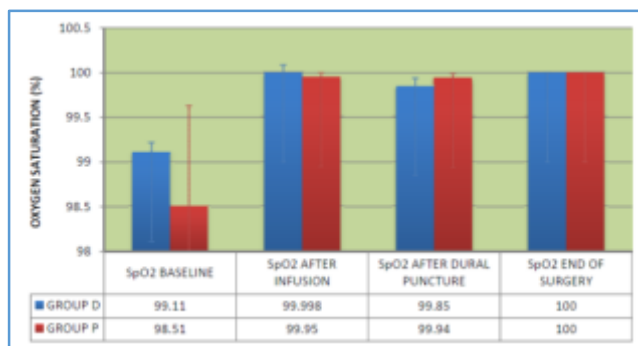


Figure 3: Bar Diagram

Respiratory Rate	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Baseline	16.16±1.397	16.87±1.914	0.266
At the end of infusion	16.89±1.548	17.12±2.156	0.596
After dural puncture	14.56±1.568	15.58±1.426	0.698
End of surgery	16.16±1.397	16.87±1.914	0.266

Table 6: Comparison of respiratory rate in between the groups at various intervals

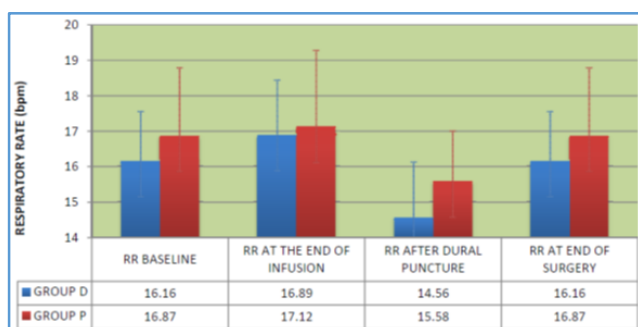


Figure 4: Bar Diagram

Sedation Score: There was significant difference in the sedation score between the two groups. Better sedation was seen in the patients who received intravenous dexmedetomidine to the control group. The data was analysed using the one way ANOVA test with a p value of less than 0.0001.

	Group – D (n=45) (Mean +SD)	Group – D (n=45) (Mean +SD)	p Value
Sedation Score	3.42+0.621	1.80+0.405	< 0.0001

Table 7

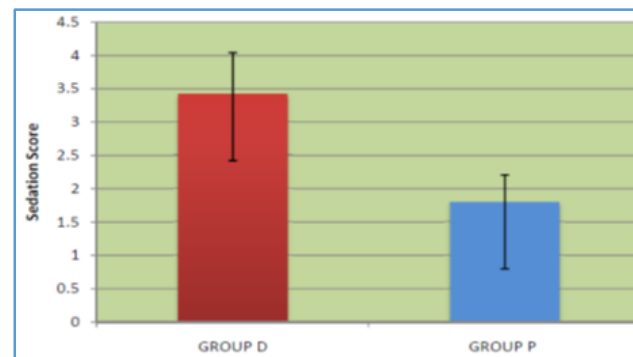


Figure 5: Bar Diagram

Effects on the Sensory and Motor Blockade: The highest level of sensory blockade achieved was significantly higher in Group D when compared to the control group with 1 patient attaining a level of T2, 12 patients attaining a level of T6 and 32 patients attaining a level of T4 in Group D in comparison to 1 patient attaining a level of T12, 19 patients attaining a level of T10, 4 patients attaining a level of T9, 18 patients attaining a level of T8 and 3 patients attaining a level of T6 in the control group.

Highest level of sensory block	Group D (n=45)	Group P (n=45)
T2	1(2.22%)	
T4	32(71.11%)	
T6	12(26.67%)	3(6.67%)
T8		18(40%)
T9		4(8.89%)
T10		19(42.2%)
T12		1(2.22%)

Table 8

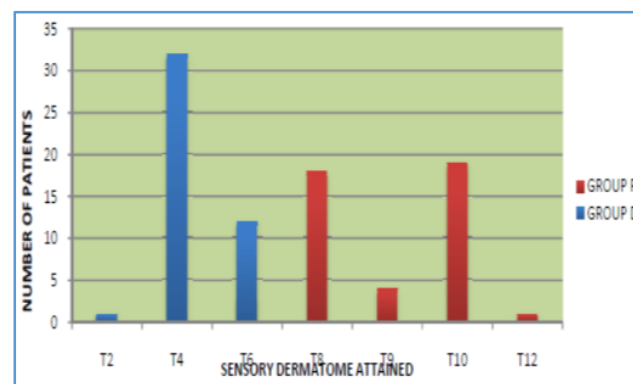


Figure 6: Bar Diagram

The highest level of sensory blockade was achieved significantly faster in Group D than in the control group. The data was analysed using the one way ANOVA test with a p value of less than 0.0001.

	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Duration (in Minutes)	7.91 ±2.42	9.82±3.973	< 0.0001

Table 9

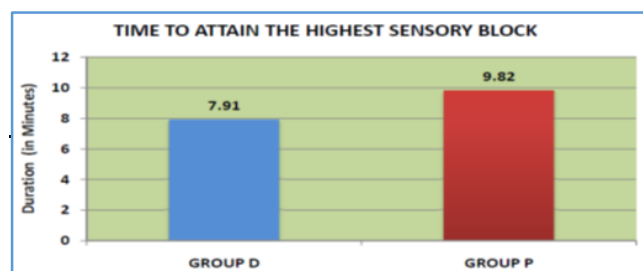


Figure 7: Bar Diagram

Both Sensory block duration (defined as regression by two dermatomes) and Motor Block duration (defined as regression to Modified Bromage score of 1) was significantly prolonged in Group D when compared to the control group. The data was analysed using the one-way ANOVA test with a p value of less than 0.0001.

	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Sensory block	76.18 ±	53.44 ±	<0.0001
Motor block duration	126.42 ±	103.64 ±	
	11.044	17.266	<0.0001

Table 10

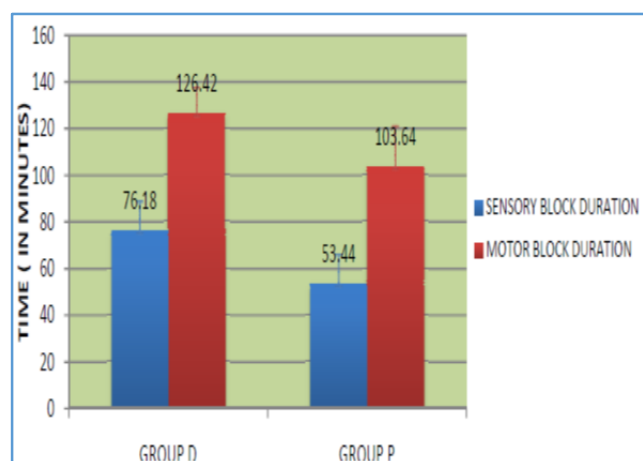


Figure 8: Bar Diagram

Post-Op Analgesic Requirements: The time to the requirement of the first rescue analgesic was significantly longer in Group D when compared to the control group. The data was analysed using the one-way ANOVA test with a p value of less than 0.0001.

Rescue Analgesic	Group D (n=45) (Mean±SD)	Group P (n=45) (Mean±SD)	p Value
Time (in Minutes)	444.66±123.864	312.22±132.582	<0.0001

Table 11

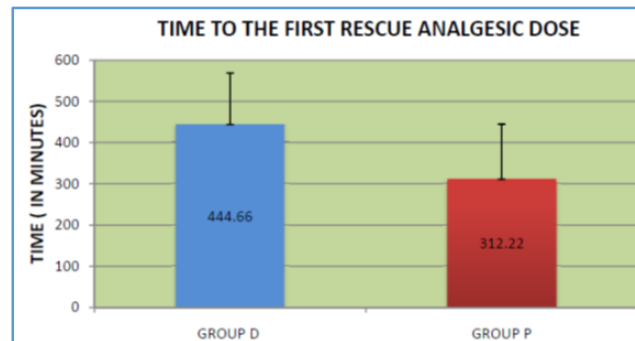


Figure 9: Bar Diagram

COMPLICATIONS:

Complications	Group D (n=45)	Group P (n=45)
Hypotension	1 (2.2%)	0
Bradycardia	13 (28.9%)	9(20%)
Excessive sedation	0	0
Arrhythmia	0	0

Table 12: Observed Side effects

Observed side effects included hypotension and bradycardia along with excessive sedation and arrhythmia. Only one case of hypotension was noted in the study group with the patient positioned in the right lateral for an implant exit of the femur, which was insignificant. Thirteen people (28.9%) of the study group developed bradycardia when compared to nine (20%) in the control group, which required no active treatment and was insignificant.

DISCUSSION: Different drugs have been used as adjuvant to local anaesthesia in order to prolong the duration of spinal analgesia. Clonidine, a α_2 agonist, has been used widely in the intrathecal,⁹ oral and intravenous routes to prolong the duration of spinal analgesia. It is known to have prolonging effect on sensory and motor blocks when used as an oral premedication within 2 h before bupivacaine spinal anesthesia.¹⁰ The intravenous administration of clonidine within 1 hour after the spinal block prolonged bupivacaine spinal analgesia for approximately 1 hour without adverse effect.⁴ Dexmedetomidine, also a α_2 agonist, is pharmacologically related to clonidine, has 8 times more affinity for α_2 receptors⁴ than does clonidine. It produces sedation and anxiolysis by binding to α_2 receptors in the locus ceruleus, which diminishes the release of norepinephrine and inhibits sympathetic activity, thus decreasing heart rate and blood pressure. It produces analgesia by binding to adrenoceptors in the spinal cord.⁵ It has been used as adjuvant to local anaesthesia in the

intrathecal route and has significant effect on onset and duration of spinal anesthesia.⁴ Dexmedetomidine has an onset of action of 30 min when the maintenance dose is used. Use of standard loading dose (1µg/Kg/hr infused over 10 minutes) 40, decreases the onset of action of dexmedetomidine. Side effects of dexmedetomidine, such as hypotension and bradycardia, are dose dependent. Infusion of loading dose over 10 min followed by the maintenance dose decreases the incidence of side effects. Jorm et al¹¹ found that dexmedetomidine has an inhibitory effect on the locus ceruleus located at the brain stem. This supraspinal action could explain the prolongation of spinal anaesthesia after intravenous administration of dexmedetomidine. The noradrenergic innervation of the spinal cord arises from the noradrenergic nuclei in the brain stem including the locus ceruleus, the A5, and the A7 noradrenergic nuclei. Neurons in the locus ceruleus are connected to the noradrenergic nuclei in the brain stem. Axon terminals of the noradrenergic nuclei reach lamina VII and VIII of the ventral horns of the spinal cord. The activity of the noradrenergic neurons is decreased by agonists acting at α₂-adrenergic receptors on the locus ceruleus cell bodies. Therefore, inhibition of the locus ceruleus results in disinhibition of the noradrenergic nuclei and exerted descending inhibitory effect on nociception in the spinal cord.¹² The mechanism of motor block is unclear, the analgesic effects of α₂-adrenergic agonists could be mediated through supraspinal, spinal, and peripheral actions. There is some evidence that clonidine results in direct inhibition of impulse conduction in the large, myelinated α fibers and the 50% effective concentration (EC₅₀) measured approximately 4-folds of that in small, unmyelinated C fibers. This could explain the less prolonged motor block compared with sensory block, as conduction of motor nerve fibers was less inhibited than sensory nerve fibers at the same concentration of clonidine. The same process might be applied to dexmedetomidine, and would explain the more sensory than motor block prolongation. Dexmedetomidine is known to have sedation effect;⁶ providing better conditions for the surgeon and the patient, provided that hemodynamic stability is preserved.

Based on all the above evidences, the present study was conducted to evaluate the effects of intravenous dexmedetomidine on the sensory and motor block of spinal bupivacaine anaesthesia and to also note its effects on the sedation and hemodynamic variables of the patient. The study involved 90 ASA I and II patients of either sex divided into two groups of 45 each.

- Group D – received Inj. Dexmedetomidine at 0.5 mcg/kg over 10 minutes as an infusion after which subarachnoid block was instituted with 3 ml of 0.5% bupivacaine (hyperbaric) solution.
- Group P – received 0.9% normal saline infusion over 10 minutes and served as control/placebo. Very few studies had been conducted to analyse the effects of intravenous Dexmedetomidine on Spinal Bupivacaine anaesthesia.

Fatma Nur Kaya, Belgin Yavascaoglu, Gurkan Turker et al¹³ compared intravenous Midazolam to Dexme-

detomidine and stated that intravenous Dexmedetomidine and not Midazolam prolongs spinal Bupivacaine anaesthesia. They concluded that Intravenous dexmedetomidine, but not midazolam, prolonged spinal bupivacaine sensory blockade. It also provided sedation and additional analgesia.¹³

Victor Whizar-Lugo, Irma A. Gómez-Ramirez, Roberto Cisneros-Corral et al conducted a study comparing the effects of intravenous dexmedetomidine to intravenous clonidine on spinal Bupivacaine anaesthesia and concluded that intravenous dexmedetomidine as well as intravenous clonidine given after spinal bupivacaine anaesthesia were able to prolong spinal anaesthesia when they were compared to placebo, without any significant side effects.¹⁴

Mahmud Mal-Mustafa, Izdiad Z Badran, Hamdi M Abu-Ali et al hypothesized that Dexmedetomidine given intravenously would prolong spinal Bupivacaine anaesthesia and concluded that intravenous dexmedetomidine administration prolonged the sensory and motor blocks of bupivacaine spinal analgesia with good sedation effect and hemodynamic stability.¹⁵ In our study, we found the highest level of sensory blockade achieved was significantly higher in the study group when compared to the control group, with sensory levels of T4 to T6 being achieved in the study group. Moreover the level of sensory blockade was also achieved faster in the study group than the control group. Dexmedetomidine as an intravenous infusion prior to the administration of subarachnoid block significantly prolonged the sensory and motor block duration in our study. However, Time to sensory regression was defined in a varied manner when compared to the previous studies. Lugo et al¹⁴ defined sensory regression as the time for the block to regress to L5 – S2, Mustafa et al defined sensory regression as the time for the block to regress to S1 dermatome. Motor block duration, likewise, was defined as the time taken for the Modified Bromage score to reach 0 in the study conducted by Mustafa et al¹⁵ and Lugo et al. Lugo et al¹⁴ however found no significant effect of Dexmedetomidine on the motor block duration in his study.

Hemodynamic changes (bradycardia, hypotension) were without clinical impact in our study in congruence with the findings of the other studies as well. Thirteen patients in the study group developed bradycardia compared to nine from the control group, which however needed no active intervention. One patient from the study group had an episode of hypotension who underwent an implant exit of the femur under right lateral position which needed a bolus dose of 6mg of ephedrine after which the Mean arterial and systolic pressures were well maintained above the normal values. The sedation score was significantly higher in the study group when compared to the control group with sedation scores ranging from 2 to 4 in the study group. No excessive sedation was observed in the study population. The mean time to the requirement of the first rescue analgesic was significantly higher in the study group, which was again in congruence with the findings of the previous studies.

CONCLUSION: We can thus conclude from the present study that Intravenous Dexmedetomidine administered as an infusion at the rate of 0.5mcg/kg over ten minutes prior to the administration of Subarachnoid Block for lower limb surgeries resulted in the following:

- There is stable hemodynamic variables. It significantly prolonged both the sensory and motor block duration with no associated significant changes in the hemodynamic variables.
- The level of sensory block attained was significantly higher in the study group and was attained more quickly than the control group.
- All patients achieved better sedation levels over the entire intra operative course.
- It provided good post-operative analgesia with the time to the requirement of the first rescue analgesic being significantly longer.

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