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Original Article

# Comparative Efficacy of Intrathecal Dexmedetomidine versus Fentanyl as Adjuvants to Hyperbaric Bupivacaine in Lower Limb Orthopaedic Surgery: A Prospective Randomised Double-Blind Study

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## ABSTRACT

**Background:** Intrathecal adjuvants are routinely added to bupivacaine to improve neuraxial blockade for lower limb orthopaedic surgery. Dexmedetomidine and fentanyl are the two agents most frequently studied in this role; dexmedetomidine acts through alpha-2 adrenergic agonism and fentanyl through mu-opioid receptor activation. Direct comparative evidence between the two remains limited and inconsistent.

**Objectives:** To compare sensory and motor block characteristics, haemodynamic profiles, postoperative analgesia, sedation and adverse effects of intrathecal dexmedetomidine versus fentanyl as adjuvants to hyperbaric bupivacaine in lower limb orthopaedic surgery.

**Methods:** This prospective, randomised, double-blind study enrolled 153 ASA I–II patients aged 20–65 years at SMS&R, Sharda University, Greater Noida, between June 2024 and December 2025. Patients were allocated to three groups (n = 51 each): Group B received 3 mL of 0.5% hyperbaric bupivacaine with 0.5 mL normal saline; Group BD received 3 mL of 0.5% bupivacaine with dexmedetomidine 5 µg; Group BF received 3 mL of 0.5% bupivacaine with fentanyl 25 µg. Block characteristics, haemodynamic parameters at 21 predefined time points, VAS pain scores, sedation scores and adverse effects were recorded over 24 hours. Analysis used one-way ANOVA with planned pairwise comparisons and chi-square or Fisher's exact tests.

**Results:** Group BD achieved the fastest sensory onset (8.08 ± 1.51 vs 9.14 ± 1.39 vs 9.75 ± 1.83 min; $p < 0.0001$), longest two-segment regression (151.39 ± 21.00 vs 107.31 ± 20.45 vs 91.35 ± 19.14 min; $p < 0.0001$) and longest time to rescue analgesia (367.02 ± 43.87 vs 274.84 ± 37.52 vs 197.35 ± 36.39 min; $p < 0.0001$) for BD, BF and B groups respectively. VAS scores were lower in Group BD at all postoperative time points ($p < 0.0001$). Bradycardia was numerically more frequent in Group BD (13.7%; $p = 0.395$). Shivering was numerically lower in both adjuvant groups (BD 5.9% vs BF 3.9% vs B 15.7%; $p = 0.092$). Cardiovascular parameters remained clinically acceptable across groups.

**Conclusion:** Intrathecal dexmedetomidine 5 µg with hyperbaric bupivacaine produced superior block characteristics, prolonged postoperative analgesia and an acceptable cardiovascular profile compared with fentanyl 25 µg and plain bupivacaine in lower limb orthopaedic surgery. Bradycardia was numerically more frequent and warrants monitoring.

***Keywords***: *Dexmedetomidine; Fentanyl; Intrathecal; Bupivacaine; Spinal*

*anaesthesia; Lower limb surgery; Postoperative analgesia.*

## INTRODUCTION

Lower limb orthopaedic procedures generate substantial perioperative pain. Tissue handling and bone manipulation contribute to postoperative discomfort that can delay mobilisation, prolong rehabilitation and extend hospital stay.[1,2] Spinal anaesthesia is the preferred technique for these surgeries as it delivers dense sensory and motor blockade with a favourable safety profile and avoids the airway risks of general anaesthesia.[3,4]

Bupivacaine is the most widely used local anaesthetic in spinal practice. It blocks sodium channels in nerve fibres and produces reliable surgical anaesthesia. Its duration of action is finite. Increasing the dose to cover the postoperative period raises the risk of haemodynamic instability and prolonged motor block.[5] This limitation prompted the use of intrathecal adjuvants that improve block quality and extend analgesia without raising the local anaesthetic dose.[6] Two classes of agents have attracted the most attention: opioids and alpha-2 adrenergic agonists.[7]

Dexmedetomidine is a highly selective alpha-2 agonist. It acts on presynaptic C-fibres and postsynaptic dorsal horn neurons, suppresses substance P release and hyperpolarises interneurons.[8] It does not cause respiratory depression. Fentanyl is a lipophilic synthetic opioid that binds mu-opioid receptors in the substantia gelatinosa; its onset is rapid and its analgesic effect well established.[9,10] Both agents are used in routine clinical practice. Published comparisons have produced inconsistent findings: some studies favour dexmedetomidine for prolonged analgesia, others point to fentanyl for better intraoperative comfort.[11-13] Sample sizes have been small and dosing strategies heterogeneous. A systematic review by Sun et al. (2017) pooled nine trials and found longer block duration with dexmedetomidine, but noted the limited quality of available evidence.[14]

The present study was designed to compare intrathecal dexmedetomidine (5 µg) and fentanyl (25 µg) as adjuvants to 0.5% hyperbaric bupivacaine in a three-arm randomised trial. The focus was on sensory and motor block characteristics, duration of postoperative analgesia, haemodynamic stability, sedation and adverse effects.

## MATERIALS AND METHODS

### Study Design, Setting and Duration

This prospective, randomised, double-blind comparative study was conducted at the Department of Anaesthesiology, School of Medical Sciences & Research (SMS&R), Sharda University, Greater Noida, India from June 2024 to December 2025.

### Ethical Considerations

The Institutional Ethics Committee of SMS&R, Sharda University approved the study protocol before data collection commenced (Approval Ref No. SU/SMS&R/76-A/2024/173 dated 20/04/2024). The study was prospectively registered with CTRI (CTRI/2024/08/072638 [Registered on: 19/08/2024]). All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each participant in his or her own language.

### Study Population and Eligibility Criteria

Patients aged 20–65 years with ASA physical status I–II, scheduled for elective lower limb orthopaedic procedures under spinal anaesthesia, were eligible. Exclusion criteria included ASA status greater than II, poorly controlled diabetes mellitus, significant neuromuscular or hepatic or adrenal or renal pathology, known hypersensitivity to amide local anaesthetics or dexmedetomidine or fentanyl, chronic therapy with corticosteroids or alpha-2 adrenergic antagonists or calcium channel blockers and any contraindication to spinal anaesthesia.

### Sample Size Estimation

Sample size was calculated from published data (mean ± SD: 6.28 ± 1.75 and 7.17 ± 1.45) using the formula $N = [(SD_1^2 + SD_2^2) \times (Z\alpha + Z\beta)^2] / (\text{mean difference})^2$. With $\alpha = 0.05$ and power of 80%, the minimum required sample was 51 per group. Three groups of 51 patients each were enrolled (total N = 153).

### Randomisation, Blinding and Intervention

Patients were randomised using the sealed-envelope technique. A senior anaesthesiologist who was not involved in outcome assessment prepared the study solution and was the only person aware of allocation. Group B received 3 mL of 0.5% hyperbaric bupivacaine with 0.5 mL of normal saline. Group BD received 3 mL of 0.5% hyperbaric bupivacaine with dexmedetomidine 5 µg diluted in 0.5 mL of normal saline. Group BF received 3 mL of 0.5% hyperbaric bupivacaine with fentanyl 25 µg (0.5 mL). Total intrathecal volume was 3.5 mL in all groups.

A wide-bore intravenous cannula was inserted in the operating theatre and patients received Ringer's solution at 10 mL/kg. Continuous monitoring was established: electrocardiography, pulse oximetry and non-invasive blood pressure. Spinal anaesthesia was performed under aseptic precautions with the patient seated. The L3–L4 or L4–L5 interspace was

identified and a 25-gauge Quincke needle was introduced via the midline approach. Free cerebrospinal fluid flow was confirmed; the study solution was then injected. The patient was placed supine immediately and received supplemental oxygen at 3 L/min via face mask.

Sensory block was assessed by pinprick and motor block by the modified Bromage scale. Primary endpoints were time from injection to highest sensory level, time to two-segment regression, time to sensory regression to S1, onset to Bromage 3 and regression to Bromage 0. Secondary endpoints included time to first rescue analgesia (tramadol 1 mg/kg IV when VAS exceeded 3), haemodynamic parameters (heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure) at 21 predefined intraoperative time points, postoperative VAS pain scores and Ramsay Sedation Scores at 0, 1, 2, 4, 6, 12, 18 and 24 hours and adverse effects including hypotension, bradycardia, shivering and postoperative nausea and vomiting.

Analysis was conducted on an intention-to-treat basis. Continuous variables were summarised as mean ± standard deviation and compared using one-way ANOVA with planned pairwise comparisons. Categorical variables were expressed as counts and proportions and analysed with chi-square or Fisher's exact tests. Repeated haemodynamic, VAS and sedation measurements were analysed at predefined time points using the same inferential approach, as applicable. The significance threshold was $p < 0.05$.

## RESULTS

### Sample Accounting and Baseline Characteristics

All 153 patients completed the study. Each group contained 51 participants with no loss to follow-up or protocol deviation. The participant flow is shown in Figure 1.

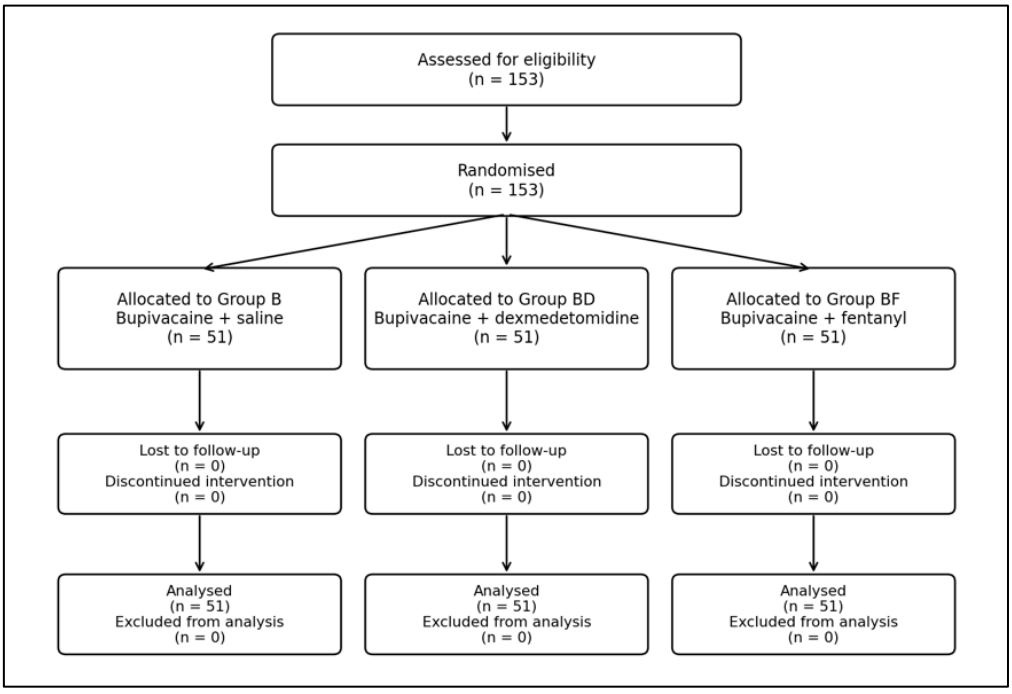


**Figure 1 – CONSORT flow diagram showing participant enrolment, allocation, follow-up and analysis**

Baseline characteristics were comparable across groups (Table 1). Mean age ranged from 36.08 to 38.27 years ($p = 0.5263$). Sex distribution did not differ ($p = 0.1718$). Height, weight and BMI were similar (all $p > 0.85$). ASA class and surgical case-mix were balanced.

**Table 1 – Baseline demographic, anthropometric and clinical characteristics by study group**

| Variable | Group B (n = 51) | Group BD (n = 51) | Group BF (n = 51) | Overall (N = 153) | p-value |
|---|---|---|---|---|---|
| Age (years), mean ± SD | 36.08 ± 9.63 | 37.98 ± 11.23 | 38.27 ± 10.88 | 37.44 ± 10.58 | 0.5263 |
| Sex (Male / Female) | 27 / 24 | 33 / 18 | 36 / 15 | 96 / 57 | 0.1718 |
| Height (cm), mean ± SD | 165.93 ± 7.88 | 165.57 ± 7.50 | 166.06 ± 7.69 | 165.85 ± 7.64 | 0.9452 |
| Weight (kg), mean ± SD | 67.47 ± 10.49 | 66.27 ± 12.13 | 66.47 ± 13.09 | 66.73 ± 11.89 | 0.8628 |
| BMI (kg/m²), mean ± SD | 24.71 ± 4.71 | 24.32 ± 5.01 | 24.33 ± 5.65 | 24.45 ± 5.11 | 0.9106 |

| | | | | | |
|---|---|---|---|---|---|
| ASA I / ASA II | 34 / 17 | 28 / 23 | 26 / 25 | 88 / 65 | 0.2488 |

*ANOVA for continuous variables; chi-square test for categorical variables. SD = standard deviation; BMI = body mass index; ASA = American Society of Anesthesiologists physical status classification.*

**Sensory and Motor Block Characteristics**

All block parameters differed across groups (ANOVA $p < 0.0001$ for each; Table 2). Sensory onset was fastest in Group BD (8.08 ± 1.51 min), intermediate in Group BF (9.14 min) and slowest in Group B (9.75 min). Two-segment regression was 151.39 ± 21.00 min in Group BD, 44 minutes longer than Group BF and 60 minutes longer than Group B. Sensory regression to S1 followed the same ranking: BD 263.10 min, BF 212.37 min, B 189.29 min. The BD-BF mean difference for two-segment regression was 44.08 minutes (95% CI 35.94 to 52.22), and the BD-B mean difference was 60.04 minutes (95% CI 52.15 to 67.93).

Motor block onset to Bromage 3 was fastest in Group BD (10.27 min) and slowest in Group B (12.45 min; $p < 0.0001$). The difference between Groups BD and BF did not reach significance ($p = 0.2796$). Regression to Bromage 0 was 297.73 ± 35.64 min in Group BD; this exceeded Group BF by 112 minutes and Group B by 145 minutes (all pairwise $p < 0.0001$). Time to first rescue analgesia was 367.02 min in Group BD, 274.84 min in Group BF and 197.35 min in Group B (all pairwise $p < 0.0001$). The BD–BF mean difference for time to first rescue analgesia was 92.18 minutes (95% CI 76.14 to 108.22), and the BD–B mean difference was 169.67 minutes (95% CI 153.83 to 185.51).

**Table 2 – Sensory and motor block characteristics and time to rescue analgesia by study group**

| Parameter (minutes) | Group B (mean ± SD) | Group BD (mean ± SD) | Group BF (mean ± SD) | ANOVA p-value | BD vs BF p-value |
|---|---|---|---|---|---|
| Time to highest sensory level | 9.75 ± 1.83 | 8.08 ± 1.51 | 9.14 ± 1.39 | **< 0.0001** | 0.0011 |
| Two-segment regression | 91.35 ± 19.14 | 151.39 ± 21.00 | 107.31 ± 20.45 | **< 0.0001** | **< 0.0001** |
| Regression to S1 | 189.29 ± 32.22 | 263.10 ± 35.88 | 212.37 ± 28.18 | **< 0.0001** | **< 0.0001** |
| Onset to Bromage 3 | 12.45 ± 1.93 | 10.27 ± 1.59 | 10.84 ± 1.79 | **< 0.0001** | 0.2796 |
| Regression to Bromage 0 | 153.20 ± 25.69 | 297.73 ± 35.64 | 185.41 ± 29.32 | **< 0.0001** | **< 0.0001** |
| Time to rescue analgesia | 197.35 ± 36.39 | 367.02 ± 43.87 | 274.84 ± 37.52 | **< 0.0001** | **< 0.0001** |

*All values expressed as mean ± SD in minutes. ANOVA = analysis of variance; S1 = first sacral dermatome.*

**Intra-operative Haemodynamic Profile**

Heart rate was lower in Group BD at most intraoperative time points. The clearest separation appeared between 3 and 30 minutes, when Group BD was 6–8 beats per minute below Group B (multiple pairwise $p < 0.001$). The difference narrowed later but Group BD maintained the lowest profile throughout. Blood pressures were broadly comparable. Systolic, diastolic and mean arterial pressures showed isolated significant differences at a small number of time points. The haemodynamic trajectories are presented in Figure 2.

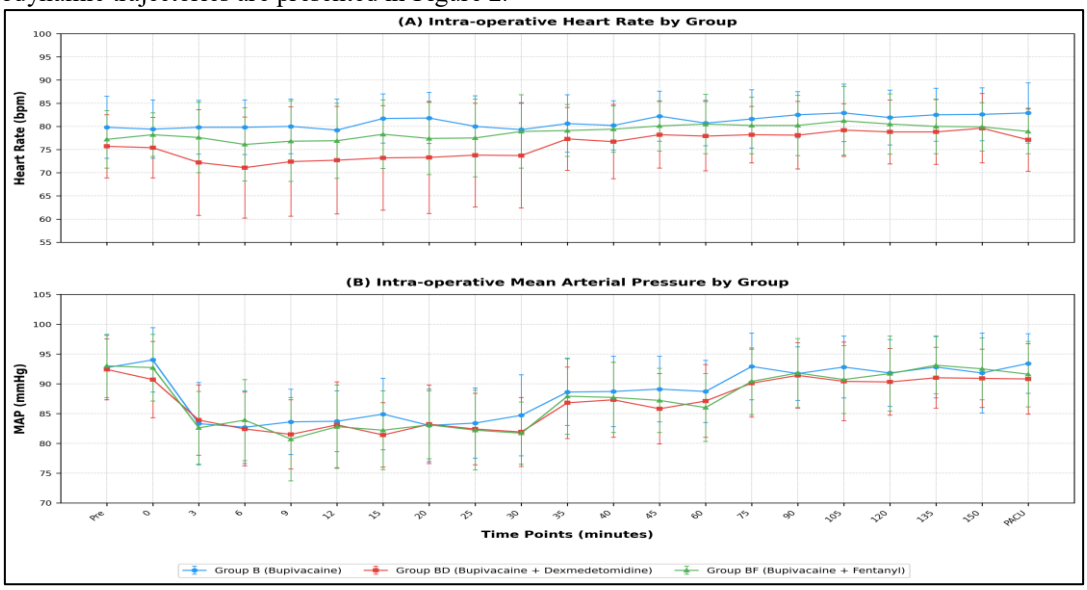


**Figure 2 – Intra-operative heart rate (Panel A) and mean arterial pressure (Panel B) across 21 time points by study group. Values are mean ± SD.**

**Postoperative Pain and Sedation**

VAS pain scores differed between groups at every time point from 0 to 24 hours (all $p < 0.0001$; Table 3 and Figure 3). Group BD had the lowest scores at each interval. Group B had the highest. Group BF was intermediate. The gap widened from 4 hours onward; at 24 hours, Group BD scored 3.3 ± 0.6 compared with 4.0 ± 0.8 in Group BF and 4.9 ± 0.6 in Group B. At 24 hours, the BD–BF mean difference was -0.70 VAS units (95% CI -0.98 to -0.42), and the BD–B mean difference was -1.60 VAS units (95% CI -1.84 to -1.36).

Sedation scores were higher in Group BD during the first 12 hours ($p < 0.0001$ at each interval). All patients remained arousable. Scores converged by 18 hours and were statistically indistinguishable by 24 hours ($p = 0.2694$; Table 3).

**Table 3 – Postoperative VAS pain scores and Ramsay Sedation Scores by group across time points**

| Time Point | VAS – Group B (mean ± SD) | VAS – Group BD (mean ± SD) | VAS – Group BF (mean ± SD) | VAS p-value | RSS – Group B (mean ± SD) | RSS – Group BD (mean ± SD) | RSS – Group BF (mean ± SD) | RSS p-value |
|---|---|---|---|---|---|---|---|---|
| 0 h | 2.1 ± 0.8 | 0.4 ± 0.6 | 1.0 ± 0.6 | **< 0.0001** | 2.0 ± 0.3 | 2.5 ± 0.5 | 2.4 ± 0.5 | **< 0.0001** |
| 1 h | 1.7 ± 0.7 | 0.2 ± 0.4 | 0.8 ± 0.6 | **< 0.0001** | 2.0 ± 0.4 | 2.6 ± 0.5 | 2.2 ± 0.5 | **< 0.0001** |
| 2 h | 2.4 ± 0.6 | 0.7 ± 0.5 | 1.2 ± 0.7 | **< 0.0001** | 1.8 ± 0.5 | 2.4 ± 0.5 | 2.1 ± 0.5 | **< 0.0001** |
| 4 h | 3.3 ± 0.7 | 1.4 ± 0.6 | 2.3 ± 0.7 | **< 0.0001** | 1.4 ± 0.5 | 2.1 ± 0.4 | 1.9 ± 0.4 | **< 0.0001** |
| 6 h | 3.7 ± 0.8 | 2.0 ± 0.8 | 2.6 ± 0.7 | **< 0.0001** | 1.4 ± 0.5 | 2.0 ± 0.4 | 1.8 ± 0.4 | **< 0.0001** |
| 12 h | 4.1 ± 0.6 | 2.6 ± 0.7 | 3.5 ± 0.7 | **< 0.0001** | 1.1 ± 0.3 | 1.7 ± 0.5 | 1.4 ± 0.5 | **< 0.0001** |
| 18 h | 4.8 ± 0.8 | 3.1 ± 0.7 | 3.6 ± 0.7 | **< 0.0001** | 1.0 ± 0.2 | 1.2 ± 0.4 | 1.0 ± 0.2 | 0.0157 |
| 24 h | 4.9 ± 0.6 | 3.3 ± 0.6 | 4.0 ± 0.8 | **< 0.0001** | 1.1 ± 0.2 | 1.2 ± 0.4 | 1.1 ± 0.3 | 0.2694 |

*VAS = Visual Analogue Scale (0–10); RSS = Ramsay Sedation Score. All values are mean ± SD. p-values from one-way ANOVA.*

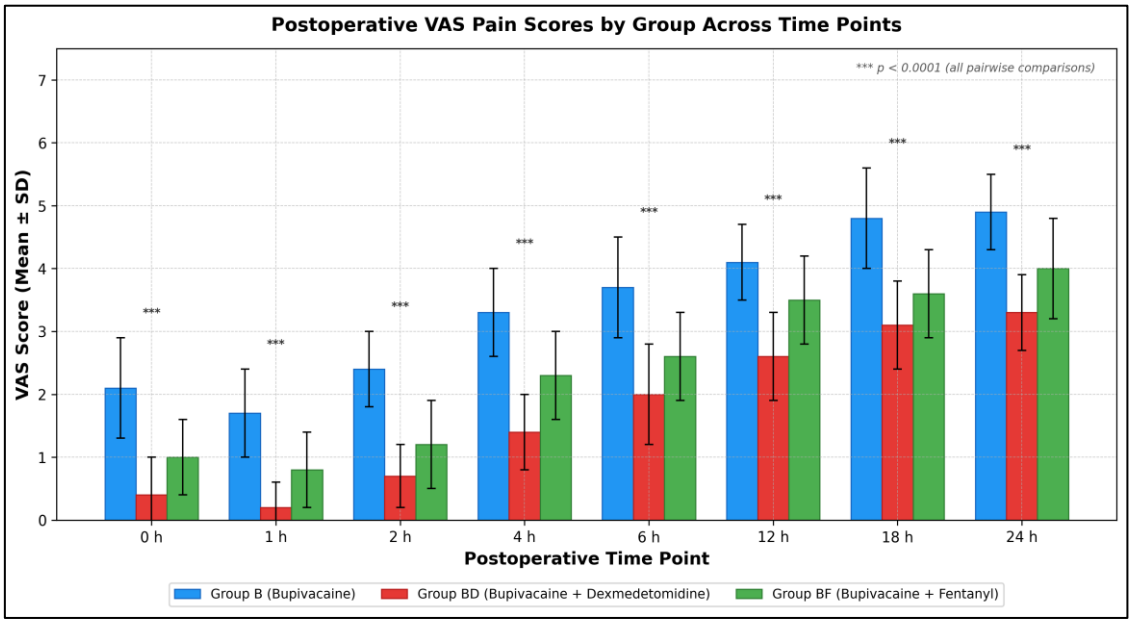


**Figure 3 – Postoperative VAS pain scores by group across time points (0–24 hours). Values are mean ± SD. *** $p < 0.0001$ for all pairwise comparisons at each time point.**

**Adverse Effects**

The adverse effect profile showed no statistically significant intergroup difference for the recorded complications (Table 4). Bradycardia was numerically more frequent in Group BD (7/51, 13.7%) than in Group BF (4/51, 7.8%) or Group B (3/51, 5.9%; $p = 0.395$). Shivering was numerically lower in Groups BD (3/51, 5.9%) and BF (2/51, 3.9%) than in Group

B (8/51, 15.7%; p = 0.092). Hypotension rates were comparable (B 9.8%, BD 13.7%, BF 17.6%; p = 0.5156). PONV did not differ across groups (p = 0.3353). No patient in any group developed respiratory depression.

**Table 4 – Adverse effects and complications by study group**

| Adverse Event | Group B n (%) | Group BD n (%) | Group BF n (%) | $\chi^2$ p-value | Significance |
|---|---|---|---|---|---|
| Hypotension | 5 (9.8) | 7 (13.7) | 9 (17.6) | 0.5156 | NS |
| Bradycardia | 3 (5.9) | 7 (13.7) | 4 (7.8) | 0.395 | NS |
| Shivering | 8 (15.7) | 3 (5.9) | 2 (3.9) | 0.092 | NS |
| PONV | 5 (9.8) | 2 (3.9) | 6 (11.8) | 0.3353 | NS |
| Respiratory depression | 0 (0) | 0 (0) | 0 (0) | – | – |

*PONV = postoperative nausea and vomiting; NS = not significant; Sig = statistically significant (p < 0.05). Chi-square or Fisher's exact test as appropriate.*

## DISCUSSION

Intrathecal dexmedetomidine produced faster block onset, longer sensory and motor block duration and extended the pain-free period compared with both fentanyl and plain bupivacaine. Group BD showed delayed time for rescue analgesia by approximately 92 minutes over Group BF and 170 minutes over Group B. VAS scores were lower in Group BD at every postoperative time point. Cardiovascular parameters remained acceptable across all three regimens. Bradycardia was numerically more frequent with dexmedetomidine, although this difference was not statistically significant, and shivering was numerically lower in both adjuvant groups.

Rahimzadeh et al. (2018) compared the same three regimens in 90 patients using identical drug doses.[11] Their dexmedetomidine group showed a two-segment regression of 149.00 ± 23.17 min and a rescue analgesia interval of 496.63 ± 70.19 min. The fentanyl group achieved 88.90 ± 12.85 min and 296.33 ± 44.83 min for the same parameters. Sensory onset was also faster with dexmedetomidine in their cohort (3.14 ± 0.74 vs 3.52 ± 0.91 min; p = 0.03). These findings align with ours, though their rescue analgesia interval with dexmedetomidine was considerably longer than the 367 minutes we observed. The difference likely reflects variation in surgical case-mix and pain assessment protocols between centres.

Agrawal et al. (2023) studied 90 patients with the same drug doses and confirmed prolonged sensory regression to S1 (120 ± 23.5 min) and delayed rescue analgesia (245 ± 3.6 min) with dexmedetomidine.[15] Vashishth and Varun (2020) reported similar results in a two-arm trial of 100 patients: sensory regression to S1 was 306.00 ± 13.32 min with dexmedetomidine versus 206.14 ± 16.69 min with fentanyl (p < 0.001); the duration of analgesia was 373.0 ± 16.26 versus 302.40 ± 16.01 min (p < 0.001).[16] Ghaly et al. (2023), using a higher dexmedetomidine dose of 10 µg in unilateral spinal anaesthesia, found an even longer rescue interval of 409.63 ± 74.60 min.[17] Sun et al. (2017), in a pooled analysis of nine RCTs, confirmed that intrathecal dexmedetomidine extends both sensory and motor blockade without increasing haemodynamic instability.[14]

The bradycardic trend in Group BD was consistent and expected. Alpha-2 agonists reduce central sympathetic outflow and increase vagal tone; the effect is amplified when the drug acts directly on brainstem cardiovascular centres through intrathecal delivery.8 Rahimzadeh et al. (2018) observed a different pattern, with fentanyl producing greater haemodynamic reductions. The difference in hemodynamic findings could be due to the response of each individual to the drug, demographic profile, volume of IT injectate and volume of diluent used. 11 Agrawal et al. (2023) and Vashishth and Varun (2020) found no significant intergroup differences.15,16 The larger sample in our study (51 per group versus 30–50 in comparator trials) may have provided the power to detect the small heart rate differences. The numerically higher bradycardia rate in Group BD (13.7% vs 5.9% in Group B; p = 0.395) is pharmacologically plausible and is consistent with findings from Elkenany et al. (2024), who reported bradycardia in approximately one-third of their dexmedetomidine recipients.18 This observation supports continuous heart rate monitoring when dexmedetomidine is administered intrathecally. Arterial pressures remained stable across all groups; all bradycardic episodes were transient and responded to conservative management with Inj Atropine 0.6 mg.

Shivering was numerically lower in both adjuvant groups. Only 3 of 51 patients (5.9%) in Group BD and 2 of 51 (3.9%) in Group BF developed shivering, compared with 8 of 51 (15.7%) in Group B (p = 0.092). Dexmedetomidine lowers the shivering threshold through alpha-2 receptor-mediated thermoregulatory modulation; fentanyl achieves a similar effect through opioid receptor activation in the hypothalamus.8

The sedation assessment in this study was more granular than in most comparator trials. Ramsay Sedation Scores were recorded at eight predefined intervals from 0 to 24 hours. Group BD maintained higher scores during the first 12 hours, indicating deeper but clinically appropriate sedation. All patients remained arousable. The scores converged by 18 hours and were indistinguishable by 24 hours. Vashishth and Varun (2020) described qualitatively greater sedation with

dexmedetomidine but did not quantify it systematically.[16] Agrawal et al. (2023) mentioned sedation only in passing.[15] The structured temporal data from our study fills a gap in the existing evidence base.

The pharmacological basis for the observed difference lies in the complementary spinal and supraspinal actions of dexmedetomidine. At the spinal level, presynaptic alpha-2 receptor activation inhibits the release of glutamate and substance P; postsynaptic activation hyperpolarises dorsal horn interneurons.[8,19] Supraspinal receptor activation enhances descending inhibitory pathways. These mechanisms act synergistically with bupivacaine and prolong both sensory and motor blockade more effectively than mu-opioid receptor agonism by fentanyl.[9,10]

**Strengths:** This study enrolled 153 patients across three arms, making it one of the larger comparisons in this field. The design included a plain bupivacaine control group, rigorous double-blinding, 21-time-point haemodynamic monitoring, structured hourly sedation assessment over 24 hours and zero attrition. The surgical case-mix was broad and well documented.

**Limitations:** Single dose of each adjuvant was studied so dose response data were not generated. Secondly analgesic consumption was not assessed beyond time to first rescue analgesic.

## CONCLUSION

Intrathecal dexmedetomidine 5 µg added to 0.5% hyperbaric bupivacaine produced faster block onset, longer sensory and motor block duration and superior postoperative analgesia compared with fentanyl 25 µg and plain bupivacaine in lower limb orthopaedic surgery. Pain scores were lower at every postoperative interval over 24 hours. Cardiovascular parameters remained acceptable in all three groups. Bradycardia occurred numerically more often with dexmedetomidine and requires monitoring; shivering was numerically lower with both adjuvants. Dexmedetomidine is a suitable intrathecal adjuvant when prolonged blockade, extended analgesia and moderate sedation are desired. Dose-response studies and multicentre validation are needed.

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