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Original article

Low-Dose Dexmedetomidine Infusion Without a Loading Bolus in Elective Spine Surgery Under General Anaesthesia: A Randomised Double-Blind Controlled Study of Haemodynamic Profile, Intraoperative Blood Loss, and Analgesic Outcomes

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ABSTRACT

Background: Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, has gained widespread application as an anaesthetic adjuvant owing to its sympatholytic, sedative, and analgesic properties. Its use in spine surgery, where controlled haemodynamics and minimisation of intraoperative blood loss are paramount, has been extensively studied at standard dosing regimens. However, the specific efficacy and safety profile of low-dose infusion at 0.5 µg/kg/hr without a preceding loading bolus in elective spine surgery under general anaesthesia remains insufficiently characterised in the Indian tertiary care setting.[1-4]

Objectives: The present study compared the effects of intravenous dexmedetomidine infusion (0.5 µg/kg/hr) versus normal saline on intraoperative haemodynamic parameters, surgical blood loss, emergence characteristics, and postoperative analgesic requirements in patients undergoing elective spine surgery.

Methods: A prospective, randomised, double-blind, controlled study enrolled 60 adults (ASA physical status I–II, aged 18–65 years, BMI <30 kg/m²) undergoing elective spine surgery under general anaesthesia at a tertiary care institution. Patients were allocated equally to Group A (dexmedetomidine 0.5 µg/kg/hr, n=30) or Group B (normal saline, n=30). The study infusion commenced on placement in the prone position and continued until the onset of skin closure. Haemodynamic variables (heart rate, mean arterial pressure, SpO2, respiratory rate, EtCO2), emergence time, tracheal extubation time, recovery time, subjective intraoperative blood loss grading, adverse events, and time to first postoperative rescue analgesia were recorded and analysed using independent samples t-test, chi-square test, and Fisher exact test (SPSS v22; significance threshold $p<0.05$).

Results: Both groups were demographically comparable at baseline ($p>0.05$). The dexmedetomidine group demonstrated a statistically significant reduction in intraoperative heart rate at 30 to 90 minutes ($p=0.023–0.050$) and in mean arterial pressure at 5 to 30 minutes ($p=0.030–0.050$) compared with the saline group. Emergence time (7.40±0.50 vs. 9.07±0.83 min; $p=0.001$), tracheal extubation time (9.33±0.55 vs. 11.07±0.87 min; $p=0.003$), and recovery time (11.47±0.51 vs. 13.13±0.94 min; $p=0.008$) were all significantly shorter in the dexmedetomidine group. Intraoperative blood loss was significantly lower in Group A ($p=0.017$).

Conclusion: Low-dose dexmedetomidine infusion at 0.5 µg/kg/hr without a loading bolus provides clinically meaningful haemodynamic attenuation, reduces intraoperative blood loss, facilitates earlier emergence and recovery from general anaesthesia, and extends the duration of postoperative analgesia in elective spine surgery patients. The predictable incidence of bradycardia warrants vigilance but was manageable with conservative measures. These findings support the utility of this dosing strategy as an adjunct in routine elective spine anaesthesia.

Keywords: *Dexmedetomidine; α2-adrenoceptor agonist; spine surgery; haemodynamic stability; intraoperative blood loss; postoperative analgesia*

1. INTRODUCTION

Spine surgery represents one of the most haemodynamically challenging surgical procedures encountered in anaesthetic practice. It is frequently associated with significant intraoperative blood loss, pronounced stress responses to positioning and surgical stimulation, and substantial postoperative pain, particularly given that most lumbar and thoracic procedures are performed with the patient in the prone position.[1] The prone position itself predisposes patients to haemodynamic lability by impeding venous return, compressing the vena cava and abdominal viscera, and altering cardiac filling pressures.[2,3] Dexmedetomidine is the pharmacologically active dextro-enantiomer of medetomidine. It exerts its clinical effects through highly selective agonism at α2-adrenergic receptors, with an α2:α1 receptor selectivity ratio of approximately 1620:1, substantially exceeding that of clonidine.[4-7]

Its principal sites of action include the locus coeruleus of the brainstem, where activation of α2A-adrenoceptors suppresses noradrenergic firing to produce sedation and anxiolysis, and the dorsal horn of the spinal cord, where α2 receptor activation inhibits the transmission of nociceptive signals and reduces release of substance P. These mechanisms confer a unique pharmacological profile: sedation without respiratory depression, opioid-sparing analgesia, and sympatholytic attenuation of the cardiovascular stress response.[6,8] The US Food and Drug Administration (FDA) approved dexmedetomidine in 1999 for short-term sedation in mechanically ventilated intensive care patients. Its subsequent application as a perioperative adjuvant has been extensively validated, demonstrating reductions in volatile anaesthetic requirements, intraoperative opioid consumption, and postoperative pain scores across a wide range of surgical contexts.[8-10] Conventional dosing protocols for dexmedetomidine—a loading bolus of 1 µg/kg administered over 10 minutes followed by a maintenance infusion of 0.2–0.7 µg/kg/hr—are associated with a biphasic cardiovascular response characterised by a transient hypertensive surge from peripheral α2B-receptor stimulation, succeeded by bradycardia and hypotension as central sympatholysis predominates.[6,11] These haemodynamic perturbations carry particular clinical significance in spine surgery, where hypotension compromises spinal cord perfusion pressure and any augmentation of blood pressure may increase surgical field bleeding. Researchers have accordingly explored lower infusion rates without a preceding bolus as a means of preserving the beneficial properties of the drug whilst attenuating haemodynamic instability.[12,13] Despite a substantial body of literature on dexmedetomidine as an anaesthetic adjuvant, controlled data specifically evaluating a low-dose infusion of 0.5 µg/kg/hr without a loading bolus in elective spine surgery under general anaesthesia—particularly in prone position—remain limited. Earlier studies by Ozkose et al. and Keles et al. demonstrated haemodynamic advantages and anaesthetic-sparing effects with dexmedetomidine in prone-position surgery, yet employed differing dosing strategies and patient populations.[14,15] The present randomised double-blind controlled study was designed to characterise the intraoperative haemodynamic profile, blood loss, recovery characteristics, and postoperative analgesic requirements associated with low-dose dexmedetomidine infusion (0.5 µg/kg/hr, no bolus) compared with normal saline in adults undergoing elective spine surgery under general anaesthesia at a tertiary care institution in South India.

2. MATERIALS AND METHODS

2.1 Study Design and Ethics

This was a prospective, randomised, double-blind, placebo-controlled parallel-group trial conducted in the Department of Anaesthesiology at Sri Muthukumaran Medical College Hospital and Research Institute, Tamil Nadu, India, between November 2021 and August 2022. The study protocol received prior approval from the Institutional Ethics Committee (IEC: SMMCHRI/2021-22/123) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment. Patients were assured of the confidentiality of their data and their right to withdraw at any time without consequence.

2.2 Participants

2.3 Randomisation and Blinding

Eligible patients were randomly allocated by a lottery method to one of two treatment arms in a 1:1 ratio. A study pharmacist not involved in patient care prepared the identical 50-mL syringes containing either dexmedetomidine 0.5 µg/kg/hr (Group A; n=30) or an equivalent volume of isotonic normal saline (Group B; n=30). Both the attending anaesthesiologist and the outcome assessors were blinded to the allocation. Group allocation was revealed only upon completion of the study or in the event of a patient safety concern[16].

2.4 Anaesthetic Protocol

Patients were kept nil per oral for a minimum of six hours prior to surgery. Premedication consisted of oral pantoprazole 40 mg and alprazolam 0.5 mg administered the evening before surgery. On transfer to the operating theatre, two large-bore (18G) peripheral intravenous cannulae were sited, and standard monitoring was established, comprising non-

invasive blood pressure, pulse oximetry (SpO2), electrocardiography, and capnography. Baseline physiological parameters were recorded.

General anaesthesia was induced with intravenous glycopyrrolate 0.2 mg, fentanyl 1 µg/kg, and atracurium 0.5 mg/kg. Tracheal intubation was performed using an appropriately sized, cuffed, flexometallic (armoured) endotracheal tube. Anaesthesia was maintained with a mixture of oxygen and nitrous oxide (40:60) supplemented by sevoflurane (1–3%) in a closed circuit at a fresh gas flow of 2 L/min. The lungs were ventilated in volume-controlled mode with a tidal volume of 6–8 mL/kg and a respiratory rate of 10–12 breaths/min, targeting an end-tidal CO2 (EtCO2) of 30–40 mmHg.

Following induction, patients were positioned prone. This time point was designated T=0 minutes, from which all intraoperative measurements were referenced. The study infusion (dexmedetomidine 0.5 µg/kg/hr or normal saline) was commenced at T=0. Haemodynamic and respiratory parameters—heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), respiratory rate (RR), SpO2, and EtCO2—were recorded at 5-minute intervals from T=0 to T=90 minutes, and thereafter at 60-minute intervals until the end of surgery. The end-tidal sevoflurane concentration required to maintain a bispectral index of 40–60 was also documented.

Rescue medication protocols were pre-specified: intraoperative hypertension (sustained MAP >100 mmHg for >1 min) was managed with an initial bolus of fentanyl 1 µg/kg, followed, if required after 15 minutes, by a further fentanyl 0.5 µg/kg bolus, and then labetalol 5 mg IV. Hypotension (MAP <60 mmHg) was managed by reducing sevoflurane concentration, a fluid bolus, and ephedrine IV as indicated; sustained bradycardia (HR <50 bpm) was treated with atropine 0.6 mg IV. Infusion was discontinued if refractory haemodynamic instability persisted despite two courses of corrective therapy.

Paracetamol 1 g IV and ondansetron 8 mg IV were administered to all patients before wound closure. The study infusion was discontinued upon commencement of skin closure. Sevoflurane was stopped at wound dressing. On placement in the supine position, 100% oxygen was administered at 4 L/min, and neuromuscular blockade was reversed with neostigmine 2.5 mg and glycopyrrolate 0.4 mg. Tracheal extubation was performed upon restoration of airway reflexes and spontaneous ventilation.

2.5 Outcome Measures

The primary outcomes were intraoperative haemodynamic parameters (HR, MAP) and emergence time. Secondary outcomes included tracheal extubation time (time from discontinuation of sevoflurane to extubation), recovery time (time to achieving an Aldrete score ≥9), subjective intraoperative blood loss (graded by the operating neurosurgeon on a 4-point scale: 0=no impairment, 1=slight impairment, 2=impaired, 3=heavily impaired surgical field), adverse events (hypotension, bradycardia, hypertension, tachycardia, nausea/vomiting), and postoperative rescue analgesia requirements (need for rescue analgesic within 12 hours and time to first rescue analgesic).

2.6 Statistical Analysis

Data were entered into a Microsoft Excel spreadsheet and analysed using SPSS Statistics version 22 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as mean ± standard deviation (SD). Categorical variables are presented as absolute counts and percentages. The independent samples t-test was used to compare means between the two groups. Chi-square test or Fisher exact test (applied when any expected cell count was <20%) was used to compare proportions. Repeated haemodynamic measurements were compared at each predefined time point. A two-tailed p-value of <0.05 was considered statistically significant. No imputation was performed for missing data.

3. RESULTS

A total of 60 patients were enrolled, with 30 in each group. No patient was withdrawn or lost to follow-up. The principal findings are summarised in Tables 1–7 and illustrated in Figures 1–8.

3.1 Baseline Demographic and Clinical Characteristics

As presented in Table 1, the two groups were well matched for all baseline variables. The mean age was 41.77±9.14 years in Group A and 42.10±9.71 years in Group B (p=0.892). Gender distribution was comparable (Group A: 43.3% female; Group B: 50% female; p=0.683). Mean body weight was 70.13±8.04 kg versus 70.59±8.71 kg (p=0.854). The proportion of patients classified as ASA grade I did not differ significantly between groups (60.0% vs. 50.0%; p=0.302). Mean operative duration was 168.40±5.47 minutes in Group A and 164.83±5.05 minutes in Group B (p=0.751). No statistically significant between-group differences were observed for any baseline variable.

Table 1. Baseline demographic and clinical characteristics of study participants

Variable	Group A (Dexmedetomidine) n = 30	Group B (Normal Saline) n = 30	p-value
Age (years), mean ± SD	41.77 ± 9.14	42.10 ± 9.71	0.892
Age range (years)	26 – 56	22 – 58	—
Sex: Female, n (%)	13 (43.3%)	15 (50.0%)	0.683
Sex: Male, n (%)	17 (56.7%)	15 (50.0%)	—
Body weight (kg), mean ± SD	70.13 ± 8.04	70.59 ± 8.71	0.854
ASA grade I, n (%)	18 (60.0%)	15 (50.0%)	0.302
ASA grade II, n (%)	12 (40.0%)	15 (50.0%)	—
Duration of surgery (min), mean ± SD	168.40 ± 5.47	164.83 ± 5.05	0.751

ASA = American Society of Anesthesiologists. Values are expressed as mean ± SD or n (%). p-values derived from independent samples t-test (continuous variables) or chi-square test (categorical variables).

3.2 Emergence and Recovery Characteristics

Table 2 presents the emergence and recovery profile. All three time metrics were significantly shorter in Group A (dexmedetomidine) compared with Group B. Mean emergence time was 7.40±0.50 min versus 9.07±0.83 min (p=0.001). Mean tracheal extubation time was 9.33±0.55 min versus 11.07±0.87 min (p=0.003). Mean recovery time was 11.47±0.51 min versus 13.13±0.94 min (p=0.008). These results indicate that dexmedetomidine infusion was associated with a significantly accelerated emergence from anaesthesia and a faster return to full consciousness (Figure 1). The estimated reduction in emergence time was approximately 1.7 minutes, a clinically meaningful difference given the cumulative effect on operating theatre efficiency in high-volume surgical centres.

Table 2. Comparison of emergence and recovery characteristics between groups

Parameter	Group A (Dexmedetomidine) n = 30, mean ± SD	Group B (Normal Saline) n = 30, mean ± SD	p-value
Emergence time (min)	7.40 ± 0.50	9.07 ± 0.83	**0.001***
Tracheal extubation time (min)	9.33 ± 0.55	11.07 ± 0.87	**0.003***
Recovery time (min)	11.47 ± 0.51	13.13 ± 0.94	**0.008***

** Statistically significant (p < 0.05). Values expressed as mean ± SD. Emergence time = time from cessation of sevoflurane to eye opening and response to verbal commands; Tracheal extubation time = time from cessation of sevoflurane to safe tracheal extubation; Recovery time = time to achieve Aldrete score ≥9.*

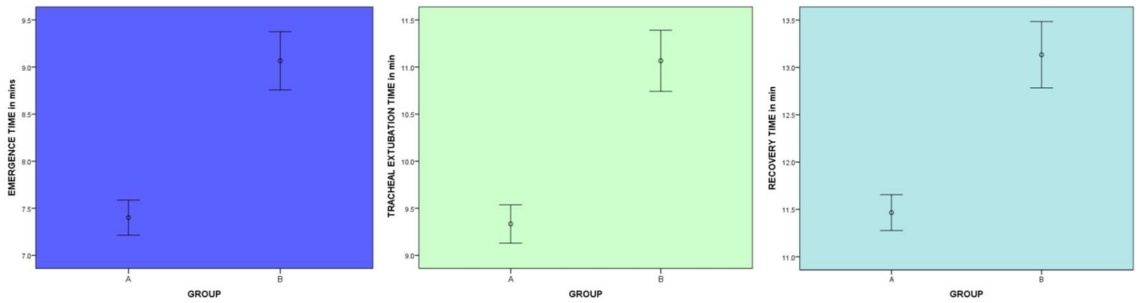


Figure 1. Bar chart comparison of emergence, tracheal extubation, and recovery times (mean ± SD) between Group A (dexmedetomidine) and Group B (normal saline). Asterisks (*) denote statistically significant differences (p<0.05). Group A demonstrates significantly reduced times across all three recovery parameters.

3.3 Intraoperative Haemodynamic Parameters

3.3.1 Heart Rate

Table 3 presents the serial intraoperative heart rate data. Both groups had comparable baseline heart rates at T=0 (Group A: 71.33±5.41 bpm; Group B: 71.50±4.86 bpm; p=0.583). Statistically significant between-group differences emerged

from T=30 minutes onwards, persisting through T=90 minutes (Figure 2). At T=30 min, mean HR was 67.10±4.83 bpm in Group A versus 71.70±4.83 bpm in Group B (p=0.050). At T=60 min, the respective values were 67.67±4.31 versus 71.10±4.22 bpm (p=0.034). The heart rate in Group A returned to Group B levels by the post-operative hour, confirming that the haemodynamic effect was confined to the active infusion period.

Table 3. Serial intraoperative heart rate comparison (beats per minute, mean ± SD)

Time Point	Group A (Dexmedetomidine) Mean ± SD	Group B (Normal Saline) Mean ± SD	p-value
T = 0 min	71.33 ± 5.41	71.50 ± 4.86	0.583
T = 5 min	71.70 ± 5.00	71.43 ± 4.68	0.525
T = 10 min	71.23 ± 4.20	71.70 ± 4.27	0.856
T = 15 min	68.77 ± 4.33	71.37 ± 4.36	0.083
T = 20 min	67.60 ± 5.06	71.40 ± 5.73	0.071
T = 30 min	67.10 ± 4.83	71.70 ± 4.83	**0.050***
T = 40 min	67.60 ± 4.32	71.47 ± 4.19	**0.043***
T = 50 min	67.93 ± 3.90	71.97 ± 3.89	**0.040***
T = 60 min	67.67 ± 4.31	71.10 ± 4.22	**0.034***
T = 70 min	67.33 ± 4.25	71.73 ± 4.18	**0.030***
T = 80 min	67.38 ± 4.43	71.20 ± 3.80	**0.050***
T = 90 min	67.23 ± 4.24	72.20 ± 4.13	**0.023***
T = 1 hour	69.50 ± 3.99	71.33 ± 3.18	0.148
T = 2 hours	70.17 ± 4.07	71.70 ± 4.03	0.429
T = 3 hours	71.43 ± 4.15	71.50 ± 3.59	0.791
T = 4 hours	71.03 ± 5.30	71.13 ± 5.75	0.309

** Statistically significant ($p \leq 0.05$). Values expressed as mean ± SD (beats per minute). Independent samples t-test applied at each time point.*

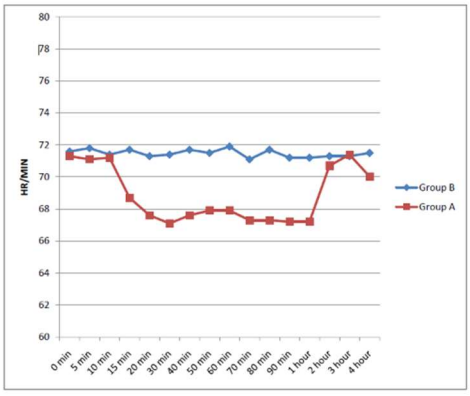


Figure 2. Line graph showing serial mean heart rate (bpm) across the intraoperative and early postoperative period for Group A (dexmedetomidine, solid line) and Group B (normal saline, dashed line). Statistically significant between-group differences are observed from T=30 min through T=90 min. Error bars represent ±1 SD.

3.3.2 Mean Arterial Pressure

Table 4 details the serial MAP data. Baseline MAP was comparable between groups (Group A: 88.30±5.27 mmHg; Group B: 87.07±4.85 mmHg; p=0.878). Statistically significant reductions in MAP were observed in Group A relative to Group B at T=5 through T=30 minutes (p=0.030–0.050). At T=5 min, MAP was 85.43±4.80 mmHg in Group A compared with 87.00±4.83 mmHg in Group B (p=0.030). By T=40 minutes, MAP values had converged and remained comparable through the remainder of the intraoperative period, suggesting that the initial central sympatholytic effect of dexmedetomidine attenuated as steady-state infusion conditions were reached (Figure 3).

Table 4. Serial intraoperative mean arterial pressure comparison (mmHg, mean ± SD)

Time Point	Group A (Dexmedetomidine) Mean ± SD	Group B (Normal Saline) Mean ± SD	p-value
T = 0 min	88.30 ± 5.27	87.07 ± 4.85	0.878
T = 5 min	85.43 ± 4.80	87.00 ± 4.83	**0.030***
T = 10 min	85.27 ± 4.81	86.87 ± 4.94	**0.045***
T = 15 min	85.10 ± 4.57	86.30 ± 4.55	**0.046***
T = 20 min	84.40 ± 4.34	87.67 ± 4.96	**0.050***
T = 30 min	85.87 ± 4.63	86.07 ± 4.85	**0.050***
T = 40 min	85.43 ± 4.80	86.00 ± 4.83	0.333
T = 50 min	86.27 ± 4.81	86.87 ± 4.94	**0.050***
T = 60 min	86.10 ± 4.57	87.30 ± 4.55	0.460
T = 70 min	87.40 ± 4.34	86.93 ± 4.84	0.350
T = 80 min	86.73 ± 4.66	86.33 ± 4.86	0.235
T = 90 min	84.20 ± 4.71	88.27 ± 4.58	0.060
T = 1 hour	87.03 ± 4.50	88.93 ± 4.92	0.070
T = 2 hours	88.43 ± 6.83	89.00 ± 4.53	0.120
T = 3 hours	89.13 ± 4.59	89.50 ± 4.58	0.320
T = 4 hours	89.10 ± 4.55	89.31 ± 4.55	0.110

** Statistically significant ($p \leq 0.05$). Values expressed as mean ± SD (mmHg). Independent samples t-test applied at each time point.*

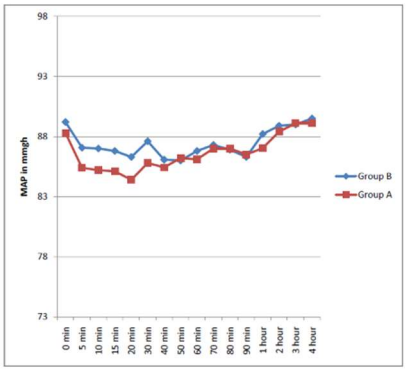


Figure 3. Line graph comparing serial mean arterial pressure (mmHg) between Group A (dexmedetomidine) and Group B (normal saline). Statistically significant reductions in Group A are observed from T=5 min through T=30 min, converging thereafter.

3.3.3 Oxygen Saturation (SpO2), Respiratory Rate, and End-Tidal CO2

As summarised in Table 5, no statistically significant between-group differences were detected in SpO2, respiratory rate, or EtCO2 at any time point throughout the intraoperative and early postoperative periods (all p>0.05). Mean SpO2 values in both groups remained above 99% throughout the study. Respiratory rate and EtCO2 values were well within the physiologically normal range in both groups at all time points. These data confirm that dexmedetomidine infusion at the studied dose did not induce respiratory depression or compromise ventilatory efficiency under controlled mechanical ventilation conditions (Figures 4 and 5).

Figure 04: comparison of spO_2

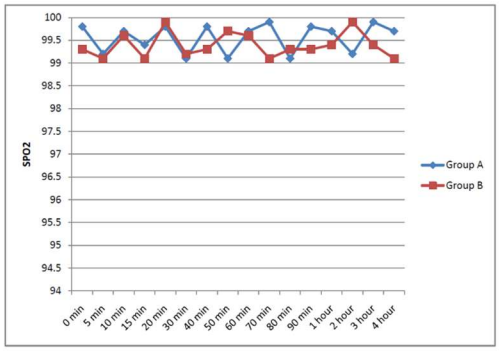


Figure 5: comparison of RR

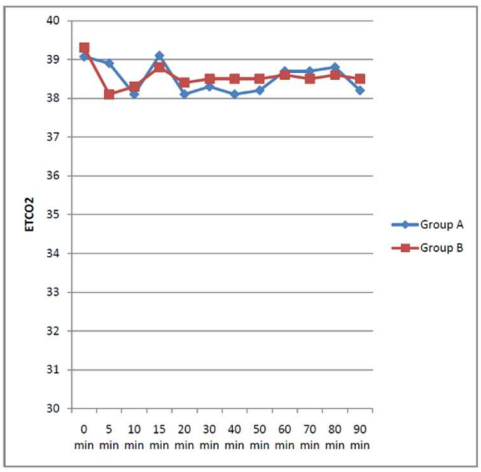


Table 5. Summary of SpO2, respiratory rate (RR), and end-tidal CO2 (EtCO2) at selected time points			
Parameter / Time Point	**Group A Mean ± SD**	**Group B Mean ± SD**	**p-value**
SpO2 (%) at T=0	99.83 ± 0.79	99.31 ± 0.81	0.751
SpO2 (%) at T=30	99.13 ± 0.73	99.27 ± 0.69	0.870
SpO2 (%) at T=60	99.77 ± 0.86	99.63 ± 0.67	0.237
SpO2 (%) at T=90	99.80 ± 0.66	99.27 ± 0.69	0.211
RR (breaths/min) at T=0	14.10 ± 1.27	14.47 ± 1.11	0.238
RR (breaths/min) at T=30	13.60 ± 1.16	13.17 ± 0.83	0.103
RR (breaths/min) at T=60	13.23 ± 1.31	13.10 ± 0.89	0.140
RR (breaths/min) at T=90	13.93 ± 1.29	13.60 ± 1.13	0.291
EtCO2 (mmHg) at T=0	39.07 ± 1.26	39.30 ± 1.06	0.430
EtCO2 (mmHg) at T=30	38.30 ± 1.26	38.46 ± 1.36	0.260
EtCO2 (mmHg) at T=60	38.77 ± 1.17	38.92 ± 1.26	0.341
EtCO2 (mmHg) at T=90	38.23 ± 2.14	38.50 ± 1.56	0.392
RR = respiratory rate; EtCO2 = end-tidal carbon dioxide. All p-values >0.05 (non-significant). Values expressed as mean ± SD.			

3.4 Intraoperative Blood Loss

Subjective intraoperative blood loss, graded by the operating neurosurgeon on the four-point scale, was significantly lower in Group A compared with Group B (p=0.017; Figure 6). The majority of Group A patients (56.7%) were rated as grade 0 (no impairment of surgical field), compared with 20.0% in Group B. Grades 2 and 3 (impaired to heavily impaired) were more frequently assigned in Group B. This finding is consistent with the modest but statistically

significant reduction in mean arterial pressure observed in Group A during the early intraoperative period, which likely reduced hydrostatic pressure at the surgical site. No patient in either group required intraoperative transfusion.

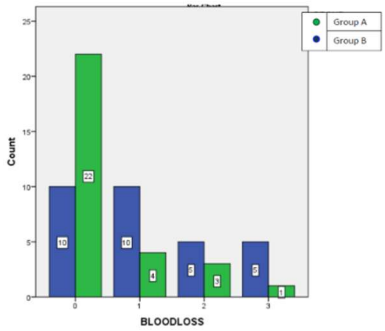


Figure 6. Stacked bar chart illustrating the distribution of intraoperative blood loss grades (0 = no impairment; 1 = slight; 2 = impaired; 3 = heavily impaired) in Group A (dexmedetomidine) and Group B (normal saline). p=0.017 (Fisher exact test).

3.5 Adverse Effects

Table 6 summarises the adverse event profile. Bradycardia was the only adverse event that differed significantly between groups, occurring in 6 patients (20%) in Group A compared with none in Group B (p=0.023). All episodes were transient and responded to a single IV dose of atropine 0.6 mg; no patient required discontinuation of the infusion for this indication. The incidence of intraoperative hypotension (Group A: 4/30, 13.3%; Group B: 1/30, 3.3%; p=0.324) did not reach statistical significance. No patient in either group experienced hypertension, tachycardia, or postoperative nausea and vomiting.

Table 6. Incidence of intraoperative adverse events

Adverse Event	Group A (Dexmedetomidine) n = 30, n (%)	Group B (Normal Saline) n = 30, n (%)	p-value
Bradycardia (HR <50 bpm)	6 (20.0%)	0 (0%)	**0.023***
Hypotension (MAP <60 mmHg)	4 (13.3%)	1 (3.3%)	0.324
Hypertension	0 (0%)	0 (0%)	—
Tachycardia	0 (0%)	0 (0%)	—
Nausea and vomiting	0 (0%)	0 (0%)	—

** Statistically significant (p < 0.05). Fisher exact test applied. HR = heart rate; MAP = mean arterial pressure.*

3.6 Postoperative Rescue Analgesia

Table 7 details postoperative rescue analgesic requirements. Within the first 12 postoperative hours, rescue analgesia was required by 6 patients (20.0%) in Group A and 2 patients (6.7%) in Group B (p=0.024). Critically, among those who required rescue analgesia, the mean time to first analgesic request was significantly longer in Group A (14.33±1.03 hours) compared with Group B (11.00±1.00 hours; p=0.012. This pattern indicates that dexmedetomidine conferred a meaningfully extended duration of postoperative analgesia—postponing the onset of clinically significant pain by more than three hours relative to the saline group—consistent with the drug's well-characterised spinal and supraspinal antinociceptive mechanisms.[16,17]

Table 7. Postoperative rescue analgesia requirements within 12 hours

Parameter	Group A (Dexmedetomidine) n = 30	Group B (Normal Saline) n = 30	p-value
Required rescue analgesia within 12 h, n (%)	6 (20.0%)	2 (6.7%)	**0.024***
Did not require rescue analgesia, n (%)	24 (80.0%)	28 (93.3%)	—
Time to first rescue analgesia (hours), mean ± SD *	14.33 ± 1.03	11.00 ± 1.00	**0.012***

** Statistically significant (p < 0.05). Fisher exact test for proportions; independent samples t-test for time to rescue analgesia. † Time to rescue analgesia calculated only among patients who required rescue analgesia.*

4. DISCUSSION

The present study demonstrates that a low-dose dexmedetomidine infusion of 0.5 µg/kg/hr, initiated without a loading bolus upon placement in the prone position, provides several clinically meaningful perioperative benefits in adult patients undergoing elective spine surgery under general anaesthesia. These include intraoperative heart rate and mean arterial pressure attenuation, reduced surgical field blood loss, accelerated emergence from anaesthesia, and prolonged postoperative analgesia—without compromising respiratory function or the quality of ventilatory control.

The haemodynamic findings are consistent with the established pharmacodynamic profile of dexmedetomidine. Infusion without a bolus avoids the transient peripheral α2B receptor-mediated hypertensive surge and instead allows a gradual, predictable decline in central sympathetic outflow, resulting in moderate reductions in HR and MAP.[6,11]

In our study, significant heart rate reductions emerged from T=30 minutes and persisted through T=90 minutes. The initial absence of significant heart rate differences (T=0 to T=20 min) is expected with bolus-free infusion; the gradual onset is precisely the clinical advantage. Batra et al. and Tanskanen et al. similarly reported stable haemodynamics without bolus dexmedetomidine in intracranial surgery patients.[17-18]

The reduction in MAP was confined to the 5–30-minute window, after which values equilibrated between groups. This transient nature likely reflects compensatory cardiovascular adjustments, including restored baroreceptor reflex activity—a hallmark of dexmedetomidine that distinguishes it from volatile anaesthetics and opioids, which impair baroreceptor function.[6,19]

Intraoperative blood loss, assessed by the surgeon using a standardised four-point scale, was significantly lower in the dexmedetomidine group. Mean arterial pressure is recognised as the primary determinant of haemostatic field quality in spine surgery; even modest reductions in MAP in the early prone phase translate to appreciably less surgical bleeding.[20] This is corroborated by our MAP data—a reduction of approximately 2–4 mmHg in Group A over the initial 30 minutes was accompanied by a meaningful improvement in surgical field conditions. Hazrati et al. similarly reported a statistically significant reduction in intraoperative blood loss with 0.5 µg/kg/hr dexmedetomidine infusion during hip fracture surgery (476±178 vs. 620±190 mL; p=0.04).[19] The mechanism is not exclusively haemodynamic. Clonidine—which shares the same α2 receptor class as dexmedetomidine—has been shown to produce vasoconstriction via peripheral α2 adrenoceptors in vascular smooth muscle, independent of systemic blood pressure reduction. Dexmedetomidine likely mediates a similar local vasoconstrictive effect in the surgical wound vasculature.[20]

A notable finding in the present study was the significantly shorter emergence, extubation, and recovery times observed in the dexmedetomidine group. This is counterintuitive at first glance, given dexmedetomidine's sedative properties. However, anaesthetic-sparing effects are well established—dexmedetomidine reduces the MAC of volatile agents by 25–50%, allowing a lower sevoflurane concentration to be maintained throughout surgery. Termination of a lower concentration of sevoflurane is expected to produce faster washout and earlier resumption of consciousness.[19,20] Ozkose et al. similarly documented accelerated recovery and extubation in patients receiving dexmedetomidine in the prone position.[15] The extended postoperative analgesic effect is clinically important. The mean time to first rescue analgesia was 14.33 hours in Group A versus 11.00 hours in Group B—a statistically and clinically meaningful 3.3-hour extension. This is consistent with the dual supraspinal and spinal mechanisms of α2 agonist analgesia: inhibition of locus coeruleus noradrenergic firing reduces descending pain facilitation, while presynaptic α2 receptor stimulation in the dorsal horn inhibits release of glutamate and substance P, directly dampening nociceptive transmission.[7,16]

The 20% incidence of bradycardia in Group A, compared with 0% in Group B, is the principal safety signal emerging from this study. This rate aligns with published data: bradycardia is a class effect of α2 agonists and has been reported in up to 40% of patients receiving full bolus-plus-infusion dexmedetomidine regimens. Importantly, all episodes in the current study were transient, clinically manageable with a single atropine dose, and did not necessitate infusion discontinuation or adversely affect outcomes. The absence of bradycardia in Group B and the higher absolute rate in Group A (relative to some low-dose infusion studies without a bolus) merit consideration in patient selection: individuals with pre-existing conduction abnormalities, those on beta-blockers, or those at risk for sinus node dysfunction should be monitored closely.[6,11]

Neither respiratory depression nor nausea and vomiting was observed in any patient in either group, reinforcing the favourable safety profile of dexmedetomidine with respect to respiratory function—a key advantage over opioid-based adjuvant strategies.[7,20]

5. CONCLUSION

Low-dose dexmedetomidine infusion at 0.5 µg/kg/hr without a loading bolus is an effective and safe adjuvant for general anaesthesia in adults undergoing elective spine surgery in the prone position. The regimen attenuates intraoperative heart rate and mean arterial pressure during the active infusion period, reduces intraoperative surgical field blood loss, and accelerates emergence, extubation, and recovery from anaesthesia. It also extends the duration of postoperative analgesia. The increased incidence of transient, atropine-responsive bradycardia in the treatment group is a predictable and manageable class effect that does not offset the clinical benefits at this dosing level. These findings support routine incorporation of this strategy into balanced anaesthetic protocols for elective spine surgery, with appropriate patient selection and haemodynamic monitoring.

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