e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(1); 685-690

Original Research Article

Clinical Evaluation of Dexmedetomidine for Sedation during Regional Anesthesia

Rashmi Shankarrao Jawanjal[1], Sachin Ramesh Gondane[2], Nitin Ramesh Gondane[3]

[1]Assistant Professor, Department of Anaesthesia, Dr PDMMC Hospital Amravati, Maharashtra India
[2]Assistant Professor, Department of Anaesthesia, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India
[3]Senior Resident, Department of Anaesthesia, Rajmata Jijau Maternity and Child Hospital, Airoli, Navi Mumbai, Maharashtra India

Received: 26-11-2025 / Revised: 25-12-2025 / Accepted: 26-01-2026
Corresponding Author: Dr. Sachin Ramesh Gondane
Conflict of interest: Nil

Abstract:

Background: Adequate sedation during regional anesthesia improves patient comfort and operating conditions while preserving spontaneous respiration and hemodynamic stability. Dexmedetomidine, a selective α_2-adrenergic agonist, has emerged as a useful sedative agent due to its anxiolytic and analgesic properties with minimal respiratory depression.

Material and Methods: This prospective, randomized, double-blind, controlled study included 60 adult patients (ASA physical status I–II) undergoing elective surgery under regional anesthesia. Patients were randomly allocated into two groups of 30 each. Group D received intravenous dexmedetomidine with a loading dose of 0.5 µg/kg followed by a maintenance infusion of 0.2–0.5 µg/kg/h, while Group C received an equivalent volume of normal saline. Sedation was assessed using the Ramsay Sedation Scale. Hemodynamic parameters, adverse events, need for rescue sedation, and patient and surgeon satisfaction were recorded and analyzed statistically.

Results: Demographic variables and baseline characteristics were comparable between the groups. Sedation scores were significantly higher in Group D at all measured time points (10, 30, and 60 minutes; $p < 0.01$). The lowest recorded heart rate and mean arterial pressure were significantly lower in the dexmedetomidine group compared to the control group ($p < 0.01$), though these changes were clinically manageable. The requirement for rescue sedation was significantly greater in Group C (20%) compared to Group D (0%; $p = 0.01$). The incidence of bradycardia and hypotension was higher in Group D but did not reach statistical significance. Patient and surgeon satisfaction scores were significantly higher in the dexmedetomidine group ($p < 0.01$).

Conclusion: Dexmedetomidine provides effective and reliable sedation during regional anesthesia, with improved sedation quality and satisfaction and a reduced need for rescue sedation, while maintaining acceptable hemodynamic safety.

Keywords: Dexmedetomidine; Regional Anesthesia; Procedural Sedation; Hemodynamic Stability; Patient Satisfaction.

DOI: 10.25258/ijcpr.18.1.115

Introduction

Regional anesthesia offers high-quality intraoperative analgesia while preserving spontaneous ventilation and airway reflexes; however, many patients still experience anxiety, discomfort from positioning or tourniquet inflation, and intolerance of ambient noise or surgical manipulation, creating a frequent need for titratable procedural sedation [1]. Contemporary procedural sedation guidance emphasizes structured monitoring, readiness to manage airway/ventilation events, and selection of sedatives that balance patient comfort with cardiorespiratory safety [1].

Dexmedetomidine is a highly selective α2-adrenoceptor agonist that produces a distinctive sedative state mediated via central α2 activity (notably in the locus coeruleus), with patients often remaining easily arousable and cooperative; importantly, clinically relevant respiratory depression is typically limited compared with many GABAergic sedatives.[2] These properties have made dexmedetomidine attractive for sedation when maintaining spontaneous breathing is desirable, including during regional anesthesia. At the same time, its sympatholytic and peripheral vascular effects can lead to clinically meaningful hemodynamic changes such as bradycardia and

hypotension, and dose–response variability is well recognized [2].

Evidence across adult procedural sedation settings indicates a consistent safety trade-off: compared with controls, dexmedetomidine is associated with fewer desaturation events but a higher incidence of bradycardia and hypotension, with modest prolongation of recovery time in some settings [3]. In neuraxial anesthesia specifically, randomized comparative data have shown that intravenous dexmedetomidine can provide effective intraoperative sedation and may influence spinal block characteristics and postoperative analgesic profiles when compared with conventional sedatives (e.g., midazolam) [4]. More recently, randomized-trial meta-analyses focusing on sedation during regional anesthesia continue to position dexmedetomidine as a common comparator, with outcomes centered on sedation adequacy alongside respiratory and hemodynamic event profiles [5].

Despite expanding literature, clinically meaningful uncertainty persists regarding the optimal dosing strategy that achieves satisfactory sedation without excess bradycardia/hypotension, particularly across varied surgical populations and local practice environments [2–5]. Therefore, the present study was designed to clinically evaluate intravenous dexmedetomidine for sedation during regional anesthesia, with emphasis on sedation quality and perioperative hemodynamic and respiratory safety outcomes [2–5].

Material and Methods

Study Design and Setting: This prospective, randomized, double-blind, controlled study was conducted in a tertiary care teaching hospital. All procedures were performed in accordance with the ethical standards laid down in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Study Population: Adult patients aged 18–65 years scheduled for elective surgical procedures under regional anesthesia were assessed for eligibility.

Inclusion Criteria

- Patients of either sex
- American Society of Anesthesiologists (ASA) physical status I or II
- Planned surgery under spinal or peripheral nerve block
- Ability to understand and comply with study procedures

Exclusion Criteria

- Known allergy or hypersensitivity to dexmedetomidine
- Significant cardiovascular disease (e.g., heart block, uncontrolled hypertension, severe bradycardia)
- Hepatic or renal dysfunction
- Chronic use of sedatives, opioids, or alpha-2 agonists
- Pregnant or lactating women
- Body mass index >35 kg/m²
- Refusal to participate

Sample Size Calculation: Sample size was calculated based on the primary outcome variable of sedation adequacy, measured using the Ramsay Sedation Scale (RSS). Assuming a minimum clinically significant difference of 0.8 units between groups, a standard deviation of 1.2, a power of 80%, and a two-sided alpha error of 0.05, a minimum of 27 patients per group was required. To account for potential dropouts, 30 patients were enrolled in each group, resulting in a total sample size of 60 patients.

Randomization and Blinding: Patients were randomly allocated into two groups using a computer-generated random number table. Group allocation was concealed in sealed, opaque envelopes opened just before drug preparation. The anesthesiologist responsible for intraoperative monitoring and data collection, as well as the patient, were blinded to group assignment. Study drugs were prepared by an independent anesthesiologist not involved in data collection.

Study Groups and Drug Administration

- **Group D (Dexmedetomidine group):** Patients received intravenous dexmedetomidine at a loading dose of 0.5 µg/kg diluted in 20 mL of normal saline administered over 10 minutes, followed by a maintenance infusion of 0.2–0.5 µg/kg/h adjusted to achieve the target sedation level.
- **Group C (Control group):** Patients received an equivalent volume of normal saline administered in an identical manner.

The infusion was initiated after confirmation of adequate sensory block and continued until completion of surgery.

Regional Anesthesia Technique: All patients received standardized regional anesthesia appropriate for the planned surgical procedure (spinal anesthesia or peripheral nerve block). Spinal anesthesia was performed using 0.5% hyperbaric bupivacaine under aseptic precautions. Adequate block was confirmed before initiation of sedation.

Monitoring and Data Collection: Baseline parameters including heart rate, systolic and diastolic blood pressure, mean arterial pressure, respiratory rate, and peripheral oxygen saturation (SpO_2) were recorded prior to drug administration. These parameters were subsequently recorded at 5-

minute intervals for the first 30 minutes and then every 15 minutes until the end of surgery.

Sedation was assessed using the Ramsay Sedation Scale. The target sedation level was defined as RSS 3–4. Pain was assessed using a visual analog scale when applicable. Surgeon and patient satisfaction were recorded postoperatively using a 5-point Likert scale.

Outcome Measures

Primary Outcome

- Quality and adequacy of sedation as assessed by the Ramsay Sedation Scale

Secondary Outcomes

- Hemodynamic stability (heart rate and blood pressure changes)
- Incidence of adverse events such as hypotension, bradycardia, hypoxia, nausea, or vomiting
- Need for rescue sedation
- Patient and surgeon satisfaction scores

Management of Adverse Events: Bradycardia (heart rate <50 bpm) was treated with intravenous atropine 0.6 mg. Hypotension (mean arterial pressure decrease >20% from baseline) was managed with intravenous fluids and, if required, vasopressors. Hypoxia (SpO_2 <94%) was managed with supplemental oxygen and airway support as necessary.

Statistical Analysis: Data were entered into a spreadsheet and analyzed using standard statistical software. Continuous variables were expressed as mean ± standard deviation and compared using the independent Student's t-test. Categorical variables were expressed as numbers and percentages and analyzed using the chi-square test. A p-value <0.05 was considered statistically significant.

Results

The two groups were comparable with respect to age, sex distribution, body weight, ASA physical status, and duration of surgery. No statistically significant differences were observed between Group D and Group C for any baseline demographic or perioperative variables ($p > 0.05$ for all comparisons) (Table 1).

Sedation levels, assessed using the Ramsay Sedation Scale, were consistently higher in Group D compared to Group C at all recorded time points. At 10 minutes, the mean sedation score in Group D was significantly greater than that in Group C (3.2 ± 0.6 vs. 2.1 ± 0.5; $p < 0.001$). This difference persisted at 30 minutes (3.6 ± 0.5 vs. 2.3 ± 0.6; $p < 0.001$) and at 60 minutes (3.5 ± 0.4 vs. 2.4 ± 0.6; $p < 0.001$) (Table 2).

Baseline heart rate and mean arterial pressure were comparable between the two groups. The lowest recorded heart rate during the intraoperative period was significantly lower in Group D compared to Group C (62.1 ± 6.8 bpm vs. 71.4 ± 7.9 bpm; $p < 0.001$). Similarly, the lowest mean arterial pressure observed intraoperatively was lower in Group D than in Group C (78.4 ± 6.2 mmHg vs. 86.9 ± 7.1 mmHg; $p < 0.001$) (Table 3).

The incidence of bradycardia was higher in Group D compared to Group C, although this difference did not reach statistical significance (16.7% vs. 3.3%; $p = 0.08$). Hypotension occurred in 13.3% of patients in Group D and 6.7% of patients in Group C ($p = 0.39$). No episodes of hypoxia were recorded in either group. The requirement for rescue sedation was significantly higher in Group C compared to Group D (20% vs. 0%; $p = 0.01$) (Table 4).

Patient satisfaction scores were significantly higher in Group D compared to Group C (4.6 ± 0.5 vs. 3.7 ± 0.6; $p < 0.001$). Surgeon satisfaction scores also favored Group D (4.7 ± 0.4 vs. 3.9 ± 0.5; $p < 0.001$) (Table 5).

Table 1: Demographic Characteristics and Baseline Variables

Parameter	Group D (Dexmedetomidine) n = 30	Group C (Control) n = 30	p value
Age (years)	42.6 ± 11.3	44.1 ± 10.8	0.58
Gender (M/F)	17 / 13	18 / 12	0.79
Weight (kg)	64.8 ± 8.6	66.2 ± 9.1	0.51
ASA I / II	19 / 11	18 / 12	0.79
Duration of surgery (min)	78.4 ± 18.9	75.7 ± 17.6	0.56

Table 2: Intraoperative Sedation Scores (Ramsay Sedation Scale)

Time Point	Group D	Group C	p value
10 min	3.2 ± 0.6	2.1 ± 0.5	<0.01
30 min	3.6 ± 0.5	2.3 ± 0.6	<0.01
60 min	3.5 ± 0.4	2.4 ± 0.6	<0.01

Table 3: Hemodynamic Parameters

Parameter	Group D	Group C	p value
Baseline HR (bpm)	78.3 ± 9.2	76.9 ± 8.7	0.54
Lowest HR (bpm)	62.1 ± 6.8	71.4 ± 7.9	<0.01
Baseline MAP (mmHg)	92.6 ± 7.5	91.8 ± 6.9	0.68
Lowest MAP (mmHg)	78.4 ± 6.2	86.9 ± 7.1	<0.01

Table 4: Adverse Events

Adverse Event	Group D (n = 30)	Group C (n = 30)	p value
Bradycardia	5 (16.7%)	1 (3.3%)	0.08
Hypotension	4 (13.3%)	2 (6.7%)	0.39
Hypoxia	0 (0%)	0 (0%)	—
Nausea / Vomiting	1 (3.3%)	3 (10%)	0.30
Rescue sedation required	0 (0%)	6 (20%)	0.01

Table 5: Patient and Surgeon Satisfaction Scores

Satisfaction Score	Group D	Group C	p value
Patient satisfaction	4.6 ± 0.5	3.7 ± 0.6	<0.01
Surgeon satisfaction	4.7 ± 0.4	3.9 ± 0.5	<0.01

Discussion

The present study demonstrates that intravenous dexmedetomidine, administered after establishment of regional anesthesia, produced a consistently deeper level of intraoperative sedation than placebo at all assessed time points, with concomitantly higher patient and surgeon satisfaction. This pattern aligns with controlled evidence showing that dexmedetomidine can reliably achieve target sedation during neuraxial techniques and reduce the requirement for additional "rescue" sedatives compared with placebo-based regimens [6]. Similarly, comparative work in spinal anesthesia settings has reported effective sedation with dexmedetomidine and favorable overall satisfaction profiles, supporting its practical utility when patient comfort and procedural conditions are key goals [7].

A clinically important finding in this study was the reduced requirement for rescue sedation in the control group relative to dexmedetomidine (20% vs. 0%). Reduced supplemental sedative needs have been reported in randomized, placebo-controlled dosing studies conducted during epidural or spinal anesthesia, where clinically relevant loading doses of dexmedetomidine increased the proportion of patients maintained at target sedation without propofol rescue [6]. In procedural settings outside the operating room, randomized comparative data also suggest that dexmedetomidine-containing sedation strategies can reduce additional sedative dosing and improve operator-rated conditions, albeit with greater cardiovascular side effects [8]. Taken together, these data support the interpretation that dexmedetomidine contributes to steadier maintenance of sedation targets, which plausibly translates into improved satisfaction scores observed in the present study.

Hemodynamic changes in our cohort—specifically lower intraoperative nadir heart rate and mean arterial pressure in the dexmedetomidine group—are consistent with the known sympatholytic profile of the drug and have been repeatedly observed across perioperative contexts. Notably, meta-analytic evidence in major spine surgery indicates higher odds of intraoperative hypotension and bradycardia with dexmedetomidine exposure, and highlights that the presence of a loading dose can increase these risks [9]. This is directionally concordant with our observation of a higher (though not statistically significant) incidence of bradycardia and hypotension in the dexmedetomidine group. Real-world and informatics-based safety evaluations further reinforce bradycardia as a recurring dexmedetomidine-associated adverse effect and underscore the need for vigilance regarding patient susceptibility and concomitant drug exposures [10]. Importantly, despite measurable hemodynamic depression in our study, events remained manageable and were not accompanied by serious respiratory compromise.

No hypoxic episodes occurred in either group in our dataset, supporting the feasibility of maintaining adequate oxygenation during regional anesthesia with protocolized monitoring. This finding is compatible with broader clinical pharmacology and perioperative literature describing minimal respiratory depression with dexmedetomidine compared with many alternative sedatives, particularly when titrated to effect [11]. Inagaki and colleagues similarly evaluated dexmedetomidine during epidural/spinal anesthesia and identified protocol-defined respiratory events across groups, but without clear excess severe respiratory compromise attributable to dexmedetomidine versus placebo under their study conditions [6]. Collectively, these findings support the role of

dexmedetomidine as a sedation option when preservation of spontaneous ventilation is prioritized, while still emphasizing the importance of dose titration and monitoring.

While our study did not evaluate neurocognitive outcomes, emerging peri-spinal anesthesia evidence in older adults suggests potential postoperative delirium advantages for dexmedetomidine when compared with propofol. A large randomized controlled trial reported a lower incidence of postoperative delirium with dexmedetomidine sedation versus propofol in healthy older adults undergoing lower-extremity orthopedic surgery with spinal anesthesia [12]. A separate propensity score-matched retrospective analysis in elderly orthopedic patients undergoing spinal anesthesia also reported a lower delirium incidence associated with dexmedetomidine sedation compared with propofol [13]. These findings are not directly inferable from our dataset, but they provide context that dexmedetomidine's clinical profile may extend beyond intraoperative sedation quality in selected high-risk populations, warranting future studies that incorporate standardized delirium screening alongside hemodynamic endpoints.

From a comparative-sedation perspective, recent trials evaluating newer agents during spinal anesthesia have consistently used dexmedetomidine as a benchmark, frequently noting lower heart rates and greater bradycardia/hypotension concerns with dexmedetomidine relative to alternatives such as remimazolam [14]. Although our control arm was placebo rather than an active sedative comparator, our hemodynamic findings align with the broader comparative literature: dexmedetomidine tends to deliver effective sedation at the cost of greater cardiovascular slowing, underscoring the importance of individualized dosing and predefined management pathways for bradycardia and hypotension.

The present study has limitations. First, the sample size was modest and may not have been powered to detect differences in relatively infrequent adverse events. Second, the analysis was restricted to ASA I–II adults, limiting generalizability to frailer patients or those with significant conduction disease or autonomic dysfunction. Third, sedation was assessed using a clinical scale; incorporation of objective sedation monitoring (e.g., processed EEG indices) could improve comparability across studies. Nonetheless, the findings provide clinically coherent evidence that dexmedetomidine can enhance sedation quality and satisfaction during regional anesthesia while producing predictable, monitor-responsive hemodynamic effects.

Conclusion

Dexmedetomidine provided effective and consistent sedation during regional anesthesia, achieving higher sedation scores and greater patient and surgeon satisfaction compared to control, while maintaining overall hemodynamic stability. Although reductions in heart rate and mean arterial pressure were observed, these changes were clinically manageable and not associated with serious adverse events. The reduced need for rescue sedation further supports the reliability of dexmedetomidine as a sedative adjunct in patients undergoing procedures under regional anesthesia.

References

1. Hinkelbein J, Lamperti M, Akeson J, Santos J, Costa J, De Robertis E, et al. European Society of Anaesthesiology and European Board of Anaesthesiology guidelines for procedural sedation and analgesia in adults. Eur J Anaesthesiol. 2018 Jan;35(1):6-24. doi: 10.1097/EJA.0000000000000683.
2. Weerink MAS, Struys MMRF, Hannivoort LN, Barends CRM, Absalom AR, Colin P. Clinical Pharmacokinetics and Pharmacodynamics of Dexmedetomidine. Clin Pharmacokinet. 2017 Aug;56(8):893-913. doi: 10.1007/s40262-017-0507-7.
3. Fonseca FJ, Ferreira L, Rouxinol-Dias AL, Mourão J. Effects of dexmedetomidine in non-operating room anesthesia in adults: a systematic review with meta-analysis. Braz J Anesthesiol. 2023 Sep-Oct;73(5):641-664. doi: 10.1016/j.bjane.2021.12.002.
4. Kumar S, Hussain M, Arun N, Kumar A, Kumar M. The Effect of Midazolam and Dexmedetomidine Sedation on Block Characteristic Following Spinal Bupivacaine: A Randomized Comparative Study. Anesth Essays Res. 2020 Jul-Sep;14(3):497-503. doi: 10.4103/aer.AER_85_20.
5. Na HS, Park SH, Koo BW, Bang S, Shin HJ. A Comparison of the Safety and Efficacy of Remimazolam and Dexmedetomidine for Sedation in Surgical Patients Under Regional Anesthesia: A Meta-Analysis of Randomized Controlled Trials. Medicina (Kaunas). 2025 Apr 14;61(4):726. doi: 10.3390/medicina61040726.
6. Inagaki Y, Yamakage M, Sakamoto A, Okayama A, Oya N, Hiraoka T, et al. The Efficacy and Safety of Dexmedetomidine for Sedation During Surgery Under Epidural or Spinal Anesthesia: A Randomized, Double-Blind, Placebo-Controlled Study. Yonago Acta Med. 2022 Jan 4;65(1):14-25. doi: 10.33160/yam.2022.02.002.
7. Shah PJ, Dubey KP, Sahare KK, Agrawal A. Intravenous dexmedetomidine versus propofol for intraoperative moderate sedation during

spinal anesthesia: A comparative study. J Anaesthesiol Clin Pharmacol. 2016 Apr-Jun;32(2):245-9. doi: 10.4103/0970-9185.168172.
8. Liu Y, Shen Y, Zhong H. Dexmedetomidine-Propofol Versus Propofol Alone for Sedation in Upper Gastrointestinal Endoscopic Ultrasound: A Randomized Comparative Study. Clin Ther. 2025 Aug;47(8):554-558. doi: 10.1016/j.clinthera.2025.05.013.
9. Wang M, Che JX, Chen L, Song TT, Qu JT. Effect of Dexmedetomidine on Intraoperative Hemodynamics and Blood Loss in Patients Undergoing Spine Surgery: A Systematic Review and Meta-Analysis. Chin Med Sci J. 2024 Mar;39(1):54-68. doi: 10.24920/004294.
10. Zhu X, Kuppa SA, Morris R, Bui L, Liu X, Hill A, et al. Evaluation of Dexmedetomidine-Associated Bradycardia and Related Drug–Drug Interactions Using Electronic Health Record (EHR) and miRNA Target Analysis. Curr Issues Mol Biol. 2025 Dec 10;47(12):1028. doi: 10.3390/cimb47121028.
11. Lee S. Dexmedetomidine: present and future directions. Korean J Anesthesiol. 2019 Aug;72(4):323-330. doi: 10.4097/kja.19259.
12. Shin HJ, Woo Nam S, Kim H, Yim S, Han SH, Hwang JW, et al. Postoperative Delirium after Dexmedetomidine versus Propofol Sedation in Healthy Older Adults Undergoing Orthopedic Lower Limb Surgery with Spinal Anesthesia: A Randomized Controlled Trial. Anesthesiology. 2023 Feb 1;138(2):164-171. doi: 10.1097/ALN.0000000000004438.
13. Park JW, Kim EK, Lee HT, Park S, Do SH. The Effects of Propofol or Dexmedetomidine Sedation on Postoperative Recovery in Elderly Patients Receiving Lower Limb Surgery under Spinal Anesthesia: A Retrospective Propensity Score-Matched Analysis. J Clin Med. 2021 Jan 3;10(1):135. doi: 10.3390/jcm10010135.
14. Hong SW, Park JY, Rhee KY, Kim SH. Comparison emergence of sedation, using dexmedetomidine and remimazolam, in spinal anaesthesia - double blinded randomized controlled trial. Int J Med Sci. 2024 Jun 3;21(8):1552-1558. doi: 10.7150/ijms.95736.