DOI: 10.7860/JCDR/2026/81754.22431

Original Article

Anaesthesia Section

Efficacy of Dexmedetomidine Infusion on Propofol Requirement in Maintaining Depth of Anaesthesia in Elective Neurosurgical Procedures: A Randomised Controlled Study

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ABSTRACT

Introduction: Dexmedetomidine, a selective alpha-2 adrenergic receptor agonist, is gaining momentum in neurosurgical anaesthesia for its sedative, analgesic, and sympatholytic effects. Propofol, although widely used, has its risks, like hypotension and respiratory depression at high doses. When these two drugs are given together during neurosurgical procedures, evaluating those changes may provide their potential as an anaesthetic adjunct while reducing risks associated with excessive propofol use when used alone.

Aim: To evaluate the efficacy of Dexmedetomidine infusion in reducing intraoperative Propofol requirement and maintaining depth of anaesthesia during elective neurosurgical procedures.

Materials and Methods: This was a randomised controlled study conducted at the Department of Anaesthesiology, Krishna Institute of Medical Sciences, Krishna Vishwa Vidyapeeth (Deemed to be University), Karad, Maharashtra, India, from February 2023 to October 2024. It comprised 50 the American Society of Anesthesiologists Physical Status (ASA-PS) I–II patients aged 18-60 years undergoing elective neurosurgery. Patients were randomised into two groups: Group P (standard anaesthesia with Propofol infusion) and Group PD (same regimen plus Dexmedetomidine infusion: 1 µg/kg bolus over 10 minutes followed by 0.5 µg/kg/hr). Propofol was titrated to maintain the Bispectral Index (BIS) between 40-60. Haemodynamic parameters, total Propofol consumption (mg/kg/hr), and intraoperative complications were recorded and analysed using Repeated-measures One way-Analysis of Variance (ANOVA) or Student's t-test.

Results: The two study groups were comparable in age (group P: 42.3±10.5 years; group PD: 41.8±9.8 years; p-value=0.82), Body Mass Index (BMI) (group P: 24.9±2.5 kg/m2; group PD: 25.1±2.4 kg/m2; p-value=0.85). Group PD showed significantly reduced intraoperative Propofol consumption compared to group P (3.00±0.8 mg/kg/hr vs. 4.50±1.0 mg/kg/hr, p-value <0.001). Haemodynamic parameters {Heart rate (HR), Mean Arterial Pressure (MAP)} remained more stable in the Dexmedetomidine group across all intraoperative time points. In the group PD, the incidence of hypotension was slightly lower than in group P {3(12%) vs. 6 (24%)}. Target BIS range (40-60) was maintained in both the study groups. However, group PD consistently demonstrated lower BIS values (e.g., 52.0±3.8 vs. 55.2±4.5 at 5 min, p-value=0.02), with reduced variability (5.8±0.9% vs. 7.2±1.1%, p-value <0.001).

Conclusion: Dexmedetomidine infusion significantly reduces the intraoperative Propofol requirement while maintaining adequate depth of anaesthesia and providing superior haemodynamic stability in elective neurosurgical procedures. Its use as an adjuvant can enhance anaesthetic efficiency and patient safety when carefully monitored.

Keywords: Adjuvant, Efficiency, Haemodynamic stability, Neurosurgery, Safety

INTRODUCTION

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist (α2: α1 selectivity of 1620:1) with sedative, analgesic, and anxiolytic properties. Its role in anaesthesia, particularly neurosurgery, is growing given its potential to enhance anaesthetic quality, provide haemodynamic stability, and reduce the requirement for high doses of other agents, thereby minimising associated risks [1]. Propofol, commonly used for neurosurgical anaesthesia, is valued for its rapid onset and recovery. Higher dosages, however, have been associated with respiratory depression and hypotension. Application of Dexmedetomidine as an adjunct may reduce the total required Propofol dose while maintaining adequate anaesthetic depth and patient stability [2].

In neurosurgery, precise control of anaesthetic depth is crucial for patient immobility, reflex suppression, and neurophysiological monitoring, especially when muscle relaxants aren't re-administered after intubation. Inadequate depth can compromise surgical outcomes, while excessive doses may affect monitoring accuracy and increase adverse events. Dexmedetomidine's pharmacologic profile may help balance these challenges by supporting stable sedation and haemodynamics throughout prolonged procedures [3]. Although Dexmedetomidine and Propofol have been studied individually, and in other surgical contexts [4,5], data on their combined use in neurosurgery remain limited [6]. Precise haemodynamic control is critical in these cases to avoid complications that include increased intracranial pressure and hypotension. While Dexmedetomidine may cause bradycardia, its potential to attenuate propofol-induced hypotension merits investigation [7,8].

Current researchers are also evaluating the intraoperative effects of Dexmedetomidine infusion on total Propofol requirement and associated haemodynamic changes during elective neurosurgical procedures [9,10]. There is a necessity for further research to expand upon the existing evidence and to elucidate the role of

Dexmedetomidine, particularly by examining its influence on propofol requirements and the concomitant haemodynamic alterations during elective cranial neurosurgical procedures. Although there are a limited number of studies that have compared these effects in the context of spinal surgeries, a more comprehensive understanding is warranted [11-13].

Hence, the current study aimed to evaluate the efficacy of Dexmedetomidine infusion in reducing intraoperative Propofol requirement and in maintaining the depth of anaesthesia during elective cranial procedures. The primary objective of the study was to measure the Propofol consumption (mg/kg/hr), and the secondary objectives were to evaluate intraoperative haemodynamics and complications such as hypotension and bradycardia.

MATERIALS AND METHODS

This was a randomised controlled study conducted at the Department of Anaesthesiology, Krishna Institute of Medical Sciences, Krishna Vishwa Vidyapeeth (Deemed to be University), Karad, Maharashtra, India. The study was performed over a period of 20 months from February 2023 to October 2024 after obtaining Ethics Committee Approval (Protocol No: 343/2022-2023; IEC No: KIMSDU/IEC/03/2023, Dated 05/04/2023) and duly signed patient's informed consent forms.

Sample size calculation: The sample size for the study was determined based on previous literature [14] that indicated a significant difference in the Propofol requirement when Dexmedetomidine was used as an adjuvant. Using the following formula for comparing two means:

$$N = \frac{(SD_P^2 + SD_{PD}^2)(Z_{1-}\alpha_{/2} + Z_{1-}\beta)^2}{(\overline{X}_P - \overline{X}_{PD})^2}$$

Where, SD_P and SD_{PD} represent the standard deviations of the propofol requirement in the two groups, $Z_{1-}\alpha_{/2}$ corresponds to the level of significance (α), and $Z_{1-}\beta$ corresponds to the power (1-β) of the study.

Substituting the values SD_P=1.0, SD_{PD}=1.2, $\overline{X}_P$=4.5, $\overline{X}_{PD}$=2.7, $Z_{1-}\alpha_{/2}$=2.58 (for α=0.01), and $Z_{1-}\beta$=2.33 (for 99% power), we obtained:

$$N = \frac{(1.0^2 + 1.2^2)(2.58 + 2.33)^2}{(4.5\text{-}2.7)^2}$$

$$= \frac{(2.44)(24.1)}{3.24}$$

$$= 18.1$$

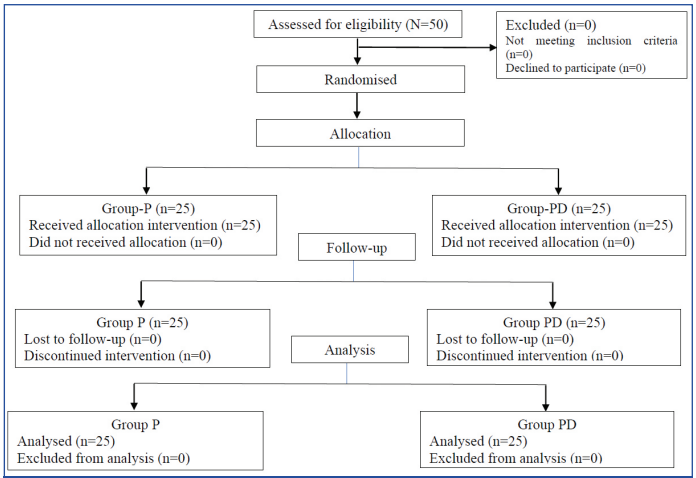
Hence, a minimum of 18 patients per group was required to achieve 99% power with an alpha level of 0.01. Considering an anticipated dropout rate of approximately 20%, the final sample size was increased to 25 patients per group to maintain adequate statistical power even if some participants were lost to follow-up.

Inclusion criteria: Patients whose age ranged between 18 and 60 years, ASA physical status I or II, and scheduled for elective neurosurgical procedures under general anaesthesia were included.

Exclusion criteria:

- Patients with known hypersensitivity or allergy to any of the study drugs (including Propofol, Fentanyl, Dexmedetomidine, Midazolam, or Cisatracurium). Patients presenting with trauma or those requiring emergency surgical interventions.
- Patients who were currently receiving antidepressant or antipsychotic medications were excluded.

Patients were randomised using a computer-generated sequence into two groups, viz., Group P (Control group) and Group PD (Intervention group) represented in the Consolidated Standards of Reporting Trials (CONSORT) flowchart [Table/Fig-1].



[Table/Fig-1]: Study CONSORT flow diagram.

Randomisation sequence was drawn using a computer-generated random number table. Allocation concealment (blinding) was done using Sequentially Numbered Opaque Envelopes (SNOPES). The names of patients fulfilling inclusion/exclusion criteria and consenting for participation in the study were sequentially entered on the cover of the opaque envelope and after that, the envelope was opened to reveal the study arm for the patient.

Study Procedure

Every patient underwent a routine pre-anaesthetic examination. Monitoring included Electrocardiograph (ECG), Non Invasive Blood Pressure (NIBP), Peripheral Oxygen Saturation (SpO_2), End-tidal Carbon Dioxide ($EtCO_2$), invasive arterial pressure, BIS, and neurophysiological monitoring. Premedication included intravenous (i.v.) Pantoprazole (40 mg), Metoclopramide (10 mg), and Paracetamol (1 g). Fentanyl (1 µg/kg) and Midazolam (0.02mg/kg) have been employed to induce sedation and analgesia. Propofol (1–2 mg/kg) and Cisatracurium (0.1 mg/kg) have been employed for promoting muscle relaxation. Tracheal intubation was followed, and patients were ventilated with Intermittent Positive Pressure Ventilation (IPPV) using oxygen and nitrous oxide in a 1:1 ratio and Sevoflurane {Minimum Alveolar Concentration (MAC) 1%}.

Group P (Control Group): Patients assigned to this group received the standard anaesthetic protocol. This included an induction with i.v. Propofol (administered at a dose of 1 to 2 mg/kg), Fentanyl (1 µg/kg) for analgesia, Midazolam (0.02 mg/kg) for sedation, and Cisatracurium (0.1 mg/kg) to facilitate neuromuscular blockade. Anaesthesia was maintained with a continuous Propofol infusion (titrated between 50 to 150 mcg/kg/min to keep the BIS within 40-60), alongside Sevoflurane at a MAC of 1% in an oxygen-nitrous oxide mixture (1:1). No Dexmedetomidine was administered in this group [4].

Group PD (Intervention group): Patients assigned to this group received an additional Dexmedetomidine loading dose (1 µg/kg over 10min) followed by continuous infusion (0.5 µg/kg/hr) as a maintenance dose [4].

Anaesthesia in both groups has been maintained with Propofol infusion titrated (50-150 µg/kg/min) to maintain BIS 40-60 and Sevoflurane (MAC 1%).

The BIS values, Propofol doses, and haemodynamic parameters {HR, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and MAP} were recorded and assessed at pre-induction and at 5, 15, 30, 60, 90, and 120 minutes, intraoperatively. Incidents of hypotension (SBP <90 mmHg or >20% fall) were treated with Phenylephrine (25 µg i.v.), and bradycardia (HR <50 bpm) was managed with Atropine (0.5 mg i.v.). At the end of surgery, anaesthetic agents were tapered, and neuromuscular blockade was reversed with Neostigmine (50 µg/kg) and Glycopyrrolate (20 µg/kg). Patients were extubated once standard criteria were met and

monitored in recovery. The data were collected by an independent observer who was unaware of the group allocation.

STATISTICAL ANALYSIS

The data was entered in Microsoft Excel 2021, and statistical analysis was carried out using Statistical Package for the Social Sciences (SPSS), IBM® version 21.0. Categorical data were represented as frequencies and percentages, and quantitative data were represented as means with Standard Deviations (SD). Repeated-measures One way-ANOVA or Student's t-test has been employed for comparing parametric variables based on data distribution. A p-value ≤0.05 was considered statistically significant.

RESULTS

The demographic and patient characteristics are summarised in [Table/Fig-2]. The two study groups were comparable concerning age (group P: 42.3±10.5 years; group PD: 41.8±9.8 years; p-value=0.82), BMI (group P: 24.9±2.5 kg/m^2; group PD: 25.1±2.4 kg/m^2; p-value=0.85), and ASA-PS (p-value=0.77). No significant differences were observed in preoperative characteristics.

Characteristics	Group P	Group PD	p-value
Age (in years)	42.3±10.5	41.8±9.8	0.82
Gender (M/F), n	14/11	15/10	0.78
ASA-PS (I/II), n	16/9	15/10	0.77
Weight (kg)	68.2±9.1	69.0±8.8	0.75
Height (cm)	165.5±8.2	166.0±7.9	0.8
BMI (kg/m^2)	24.9±2.5	25.1±2.4	0.85

[Table/Fig-2]: Demographic and patient characteristics of study participants.
Values were expressed as mean±Standard Deviation (SD) unless otherwise stated; p-value based on unpaired t-test;

Group PD showed a significantly lower propofol requirement (3.00±0.8 mg/kg/hr) compared to group P (4.50±1.0 mg/kg/hr, p-value <0.001), indicating a ~33% reduction. There was no significant difference in duration of surgery (p-value=0.68) and recovery times (p-value=0.35) between group P and group PD [Table/Fig-3]. Moreover, the total Propofol dose has been further reduced to 360±70 mg in group PD as compared to group P (540±80 mg) with a statistically significant difference (p-value <0.001).

Parameters	Group P	Group PD	p-value
Propofol consumption (mg/kg/hr)	4.50±1.0	3.00±0.8	<0.001
Duration of surgery (min)	180±25	182±28	0.68
Recovery time (min)	12.5±2.5	13.0±2.8	0.35

[Table/Fig-3]: Comparison of intraoperative Propofol consumption, duration of surgery and recovery time.
Values were expressed as mean±standard deviation (SD); p-value based on unpaired t-test

Target BIS range (40-60) was maintained in both the study groups. However, group PD consistently demonstrated lower BIS values (e.g., 52.0±3.8 vs. 55.2±4.5 at 5 min, p-value=0.02), with reduced variability (5.8±0.9% vs. 7.2±1.1%, p-value <0.001), suggesting more stable anaesthetic depth [Table/Fig-4].

Time point	Group P	Group PD	p-value
Pre-induction	97.5±1.2	97.4±1.1	0.89
5 min	55.2±4.5	52.0±3.8	0.02
15 min	54.8±4.1	52.3±3.6	0.03
30 min	55.0±4.3	52.8±3.9	0.04
60 min	54.6±4.0	53.0±3.8	0.12
90 min	54.5±3.9	53.2±3.7	0.20
120 min	54.7±4.1	53.5±3.9	0.25

[Table/Fig-4]: Comparison of BIS.
Values were expressed as mean±Standard Deviation (SD); p-value as compared to pre-induction based on repeated-measures One-way ANOVA followed by Dunnett's multiple comparison post-hoc test

Post-intubation SBP (135.0±10.5 mmHg vs. 140.0±12.0 mmHg; p-value=0.03), DBP (81.0±6.5 mmHg vs. 85.0±7.5 mmHg; p-value=0.01), and MAP (98.0±8.0 mmHg vs. 102.0±9.0 mmHg; p-value=0.01) were significantly lower in group PD compared to group P [Table/Fig-5].

The HR (bpm) was also significantly lower in group PD at five minutes when compared with group P (80.0±6.5 bpm vs. 85.5±7.2 bpm, p-value=0.005), reflecting better sympathetic control [Table/Fig-6].

The incidences of hypotension occurred in 6 (24%) and 3 (12%) of group P and group PD patients, respectively. The incidences of bradycardia occurred in 2 (8%) and 4 (16%) of patients in group P and group PD, respectively. Standard interventions have been effectively employed for managing all episodes [Table/Fig-7].

DISCUSSION

In the current study, baseline demographic characteristics such as age, gender, ASA-PS, and BMI did not differ significantly between the two study groups. This similarity ensured appropriate comparability between groups, consistent with the findings of Sen S et al., and Roy A et al., who also reported no significant demographic differences between Dexmedetomidine and control/placebo groups [11,15]. With comparable demographics, the differences observed in subsequent intraoperative parameters can be attributed to the intervention rather than baseline variability.

A major finding of the current study was the significant reduction in Propofol consumption among patients receiving an additional loading dose of Dexmedetomidine (3.00 vs. 4.50 mg/kg/hr; p-value <0.001). This ~33% reduction aligns with existing evidence demonstrating the anaesthetic-sparing effect of Dexmedetomidine. Studies by Dutta A et al., and Walia C et al., reported similar reductions in Propofol requirement when Dexmedetomidine was used as an adjunct [14,16]. Ngwenyama NE et al., also established the role of Dexmedetomidine in reducing Propofol requirements during both induction and maintenance of anaesthesia [17]. These consistent findings reinforce the clinical utility of Dexmedetomidine in minimising hypnotic drug use during neurosurgery.

Regarding depth of anaesthesia, both groups maintained BIS values within the target range of 40-60; however, the Dexmedetomidine group showed significantly lower BIS values with reduced variability (52.0% vs. 55.2% at 5 min, p-value=0.02; variability 5.8% vs. 7.2%, p-value <0.001). These results are in agreement with Chattopadhyay U et al., who reported lower BIS values with Dexmedetomidine compared to Propofol [5]. Additional studies have documented that Dexmedetomidine used as an infusion decreases BIS during intraoperative monitoring [18], and Kasuya Y et al., demonstrated that equivalent doses of Dexmedetomidine produce lower BIS scores than Propofol [19]. Studies have even suggested Dexmedetomidine as a potential maintenance agent offering superior BIS control [5]. Thus, the current study findings support existing literature indicating enhanced cortical suppression and stable anaesthetic depth with Dexmedetomidine.

The current study also demonstrated superior haemodynamic stability in the Dexmedetomidine group, with significantly lower post-intubation SBP, DBP, MAP, and HR (all p-values <0.05). These findings reflect the sympatholytic properties of Dexmedetomidine, which reduces norepinephrine release and attenuates stress responses, as previously reported [18,20]. Literature consistently shows that Dexmedetomidine diminishes intubation related haemodynamic surges and provides better cardiovascular control, particularly beneficial in neurosurgical procedures. However, this enhanced sympatholysis was accompanied by a higher incidence of bradycardia in the Dexmedetomidine group (16% vs. 8%), consistent with prior observations by Chakrabarti D et al., [21]. Conversely, Roy A et al., reported no significant difference in bradycardia or hypotension between Dexmedetomidine and Propofol groups [15], highlighting variability in reported side-effect profiles. In the present

Time point	Group	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	p-value (SBP)	p-value (DBP)	p-value (MAP)
Baseline	Group P	128.4±10.1	78.6±7.2	95.2±8.5	0.75	0.82	0.93
	Group PD	127.6±9.8	79.0±6.8	95.1±8.3			
Post-intubation	Group P	140.0±12.0	85.0±7.5	102.0±9.0	0.03	0.01	0.01
	Group PD	135.0±10.5	81.0±6.5	98.0±8.0			
5 min	Group P	138.5±11.5	84.0±7.2	101.0±8.8	0.02	0.02	0.01
	Group PD	133.0±10.0	80.0±6.8	97.5±8.0			
15 min	Group P	137.0±11.0	83.5±7.0	100.5±8.5	0.04	0.03	0.04
	Group PD	132.5±10.0	80.5±6.5	98.0±8.0			
30 min	Group P	135.0±10.8	82.5±7.0	99.0±8.3	0.40	0.30	0.35
	Group PD	133.5±10.2	81.5±6.5	98.0±7.8			
60 min	Group P	133.0±10.0	81.0±6.5	98.0±8.0	0.55	0.40	0.60
	Group PD	132.0±9.8	80.0±6.0	97.5±7.5			
90 min	Group P	131.0±10.0	80.0±6.8	97.0±8.0	0.50	0.45	0.55
	Group PD	129.0±9.0	79.0±6.2	96.0±7.8			
120 min	Group P	130.0±9.0	79.0±6.5	96.0±7.5	0.45	0.50	0.60
	Group PD	128.0±8.0	78.0±6.0	95.5±7.0			

[Table/Fig-5]: Comparison of SBP, DBP, and MAP.
Values were expressed as mean±Standard Deviation (SD); p-value as compared to baseline based on repeated-measures one-way ANOVA followed by Dunnett's multiple comparison post-hoc test; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure

Time point	Group P	Group PD	p-value
Baseline	78.5±8.2	79.2±7.9	0.68
Post-intubation	88.0±7.5	82.0±6.8	0.01
5 min	85.5±7.2	80.0±6.5	0.005
15 min	83.2±6.9	80.5±6.3	0.04
30 min	81.5±6.5	80.0±6.0	0.30
60 min	80.0±6.0	79.5±5.8	0.65
90 min	79.0±5.8	78.5±5.5	0.70
120 min	78.8±5.7	78.0±5.4	0.85

[Table/Fig-6]: Comparison of HR.
Values were expressed as mean±Standard Deviation (SD); p-value as compared to baseline based on repeated-measures One-way ANOVA followed by Dunnett's multiple comparison post-hoc test; HR: Heart rate

Complications	Group P	Group PD
Hypotension	6 (24)	3 (12)
Bradycardia	2 (8)	4 (16)

[Table/Fig-7]: Incidences of intraoperative complications during surgery.
Values were expressed as n (%)

study, all episodes were transient and manageable, with no adverse impact on recovery or neurologic assessment.

Safety outcomes in the current study indicated a slightly higher occurrence of hypotension (24% vs. 12%) in the Dexmedetomidine group, although these events were clinically manageable. The dosing regimen used- loading dose of 1µg/kg over 10 minutes followed by infusion at 0.5 µg/kg/hr- was well-tolerated, with minimal adverse effects. Similar safety profiles have been noted in previous studies where Dexmedetomidine was used for intensive care unit sedation up to 24 hours [22], for short procedures, or as an adjunct to reduce intraoperative anaesthetic needs [23]. Reports of Dexmedetomidine as a sole anaesthetic agent remain rare [24,25], but the present findings support its effective integration as part of a balanced anaesthetic technique, particularly in neurosurgical practice requiring stable haemodynamics and predictable anaesthetic depth.

The findings of the current study carry meaningful implications for clinical practice, especially within neurosurgical anaesthesia. The marked reduction in propofol usage among patients receiving Dexmedetomidine indicates that its use as an adjunct can improve the efficiency of anaesthetic drug administration. This anaesthetic-sparing effect is particularly beneficial in neurosurgery, where minimising drug-related adverse effects is essential for preserving cerebral haemodynamics and reducing postoperative risks. Enhanced haemodynamic stability suggests that Dexmedetomidine may decrease episodes of cardiovascular fluctuation. Maintaining such stability is critical for ensuring consistent cerebral perfusion, limiting changes in intracranial pressure, and ultimately supporting improved surgical outcomes.

Additionally, the stable depth of anaesthesia associated with Dexmedetomidine, demonstrated by reduced BIS variability, may help lower the risk of intraoperative awareness and promote smoother postoperative recovery. These advantages may translate into shorter emergence times and quicker readiness for neurological evaluation, thereby improving overall perioperative management. Reduced propofol consumption and fewer intraoperative complications may also provide cost benefits for healthcare systems. Collectively, these results support the potential integration of Dexmedetomidine into routine anaesthetic protocols, encouraging more balanced regimens that capitalise on the synergistic actions of multiple agents. Incorporating Dexmedetomidine into neurosurgical practice may thus enhance patient safety, recovery quality, and satisfaction by reducing anaesthetic-related adverse effects.

Future research should include multicentre trials with broader and more varied patient populations to confirm the anaesthetic-sparing and haemodynamic advantages demonstrated in the study. Long-term follow-up examining postoperative recovery, neurocognitive outcomes, and overall quality of life would further clarify the sustained impact of Dexmedetomidine use. Investigations into different dosing strategies and infusion rates may also help refine its clinical application by optimising safety and effectiveness. Comparative studies evaluating Dexmedetomidine alongside other adjunctive agents could further identify the most effective approaches for achieving stable intraoperative conditions.

Limitation(s)

Current research has limitations despite its advantages. First, generalisation may be impacted by a single-centre design. Second, the findings may not apply to emergency neurosurgical cases or high-risk patient populations. Third, long-term outcomes such as neurocognitive recovery were not assessed. Finally, although intraoperative techniques were standardised, minor variations could still influence results. Future studies with larger sample sizes, multicentre cohorts and extended follow-up are warranted to confirm these findings and evaluate optimal Dexmedetomidine dosing.

CONCLUSION(S)

The current study findings demonstrated that Dexmedetomidine loading dose (1 µg/kg over 10 min) followed by continuous infusion (0.5 µg/kg/hr) as maintenance dose significantly lowered Propofol dosage with stable BIS values, better control of haemodynamic parameters, and reduced intraoperative complications like hypotension. Furthermore, recovery time and duration of surgery were unaffected following intervention of Dexmedetomidine loading dose (1 µg/kg over 10 min) followed by continuous infusion (0.5 µg/kg/hr) as maintenance dose. Hence, incorporating an additional loading dose of Dexmedetomidine (1 µg/kg over 10 min) as an adjuvant into standard anaesthetic protocols may enhance intraoperative safety and patient outcomes in patients undergoing elective neurosurgeries.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jul 08, 2025
- Manual Googling: Nov 24, 2025
- iThenticate Software: Nov 26, 2025 (9%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jul 02, 2025**
Date of Peer Review: **Sep 15, 2025**
Date of Acceptance: **Nov 28, 2025**
Date of Publishing: **Feb 01, 2026**