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Original Research Article

Comparative effects of propofol and dexmedetomidine on cerebral hemodynamics in intracranial surgery

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ABSTRACT

Background: Maintaining stable cerebral hemodynamics is essential during intracranial surgery to ensure adequate perfusion and minimize secondary injury. This study compared the effects of propofol and dexmedetomidine on cerebral and systemic hemodynamics in patients undergoing elective intracranial surgery.
Methods: In this prospective observational study, 95 adult patients were allocated into two maintenance anesthesia groups: propofol (n=48) and dexmedetomidine (n=47). Intraoperative parameters including mean arterial pressure (MAP), heart rate (HR), intracranial pressure (ICP), cerebral perfusion pressure (CPP), and regional cerebral oxygen saturation (rSO_2) were recorded at defined intervals. Brain relaxation was assessed at dural opening using a four-point scale. Postoperative recovery and complications were evaluated over 30 days.
Results: Dexmedetomidine produced higher mean CPP (75.6±5.8 versus 72.3±6.5 mmHg, p=0.010), lower ICP (15.9±3.8 versus 18.1±4.7 mmHg, p=0.015), and higher rSO_2 (77.5±6.3 versus 73.2±7.1%, p=0.003) compared to Propofol. Bradycardia occurred more often with Dexmedetomidine (29.8% versus 12.5%, p=0.035), while hypotension was more frequent with Propofol (41.7% versus 25.5%, p=0.096). Dexmedetomidine was an independent predictor of good brain relaxation (OR 3.46, p=0.015), along with adequate CPP (OR 5.37, p=0.003). ROC analysis identified CPP as the best predictor of optimal brain relaxation (AUC=0.86).
Conclusions: Dexmedetomidine offers superior cerebral perfusion and hemodynamic stability over propofol during intracranial surgery without increasing adverse events.

Keywords: Dexmedetomidine, Propofol, Cerebral hemodynamics, Intracranial pressure, Cerebral perfusion pressure

INTRODUCTION

Maintaining stable cerebral hemodynamics during intracranial surgery is essential for optimizing neurological outcomes and preventing secondary ischemic injury. Cerebral perfusion pressure (CPP), intracranial pressure (ICP), and autoregulatory capacity together determine the adequacy of cerebral blood flow (CBF), and deviations from optimal ranges can rapidly compromise tissue oxygenation and metabolism.[1] Evidence indicates that even short periods of CPP below 60 mmHg or ICP above 20 mmHg correlate with impaired autoregulation and higher risk of postoperative neurological deficits.[2,3] Intraoperative fluctuations in these parameters have been associated with cerebral edema, delayed recovery, and extended intensive care stay.[4] The stability of cerebral circulation, therefore, depends not only on maintaining systemic mean arterial pressure (MAP) but also on preserving the autoregulatory link between CBF and metabolic demand.[5,6] Within this framework, anesthetic agents play a pivotal role since their pharmacologic effects directly influence both cerebral metabolism and vascular tone.

Propofol and dexmedetomidine are two sedative-hypnotic agents frequently employed in neuroanesthesia due to their favorable neurophysiological profiles. Propofol acts as a γ-aminobutyric acid type A (GABA-A) receptor agonist, producing a dose-dependent reduction in cerebral metabolic rate of oxygen ($CMRO_2$) and parallel decreases in CBF, thereby preserving flow–metabolism coupling.[7] This property allows efficient control of ICP while maintaining autoregulatory responsiveness. However, Propofol's vasodilatory action and suppression of sympathetic tone often cause systemic hypotension, which can jeopardize CPP if not carefully titrated.[8] In contrast, dexmedetomidine, a selective α_2-adrenergic agonist, exerts central sympatholytic and peripheral vasoconstrictive effects, providing sedation and analgesia without respiratory depression. Its hemodynamic behavior is characteristically biphasic—an initial transient increase in MAP due to peripheral α_2B-receptor activation followed by sustained bradycardia and modest blood pressure reduction secondary to central α_2A-mediated inhibition of norepinephrine release.[9] Although dexmedetomidine tends to maintain stable MAP and CPP, several studies have shown mild increases in cerebrovascular resistance and a smaller degree of CBF reduction compared with Propofol.[10,11] These contrasting profiles highlight the need to balance cerebral protection with cardiovascular stability during neurosurgical anesthesia.

Comparative evidence between these agents remains inconsistent. Small clinical trials and observational studies in neurocritical care and elective neurosurgery report divergent outcomes for ICP, CPP, and hemodynamic stability. Propofol consistently lowers ICP but is frequently associated with hypotension requiring vasopressor support.[12,13] Dexmedetomidine provides smoother hemodynamic control yet may raise cerebrovascular resistance, potentially limiting reductions in ICP.[14] A multicenter retrospective analysis in neurocritical patients found lower rates of severe hypotension with dexmedetomidine compared to Propofol, though no difference in ICP trends or clinical outcomes.[15] Similarly, meta-analytic data from awake craniotomy cases revealed no superiority of either agent in ICP management but a lower incidence of intraoperative hypertension with dexmedetomidine.[16] Many of these studies are limited by small sample sizes, heterogeneous anesthetic protocols, and lack of advanced monitoring of cerebral blood flow or oxygenation such as transcranial Doppler (TCD) or near-infrared spectroscopy (NIRS). Furthermore, most research originates from high-income countries where invasive ICP monitoring is routinely available, reducing the generalizability of findings to resource-limited settings.

Local validation of cerebral hemodynamic responses under Propofol and dexmedetomidine is particularly important for low- and middle-income countries (LMICs) like Bangladesh, where patient physiology, anesthetic practices, and surgical environments differ significantly from those in high-income regions. Regional studies have documented that invasive ICP monitors are accessible in less than one-third of neurosurgical centers across South Asia due to financial, logistical, and training barriers.[17,18] Consequently, clinicians rely on noninvasive surrogates such as TCD-derived pulsatility index and NIRS-based regional cerebral oxygen saturation to infer intracranial dynamics.[19] These modalities, although indirect, provide valuable real-time indicators of cerebral perfusion adequacy and autoregulatory status and are increasingly recognized as practical alternatives for intraoperative monitoring in lower-middle-class-income countries (LMICs). Moreover, differences in vascular compliance, baseline blood pressure patterns, and anesthetic drug pharmacodynamics among South Asian populations may influence how Propofol or dexmedetomidine affect cerebral circulation. Without region-specific data, clinical practice continues to rely on extrapolation from studies in dissimilar populations and health-care infrastructures.

Given these gaps, there is a pressing need to evaluate how Propofol and dexmedetomidine influence cerebral hemodynamics under conditions reflective of neurosurgical practice in Bangladesh. A study employing noninvasive TCD and NIRS monitoring can provide a pragmatic yet physiologically robust comparison of their effects on CBF velocity, autoregulation, and cerebral oxygenation. Such evidence would not only clarify the balance between ICP control and hemodynamic stability but also inform anesthesia protocols tailored to the realities of LMIC neurosurgical care. Establishing locally validated data could strengthen perioperative safety and optimize anesthetic choices for intracranial surgery where access to advanced invasive monitoring remains limited.

METHODS

This prospective, comparative observational study was conducted at National Institute of Neurosciences and Hospital (NINS), Dhaka, Bangladesh from July, 2024 to June, 2025. A total of 95 patients were undergoing elective intracranial surgery under general anaesthesia. Participants were divided into two groups according to the primary anaesthetic agent administered for maintenance: group P (Propofol, n=48) and group D (Dexmedetomidine, n=47). All patients were aged between 18 and 70 years, belonged to ASA physical status I–III, and had no significant cardiovascular, hepatic, or renal impairment.[20]

Patients with uncontrolled hypertension, severe cardiac disease, or those receiving beta-blockers were excluded. Standard pre-anaesthetic evaluation and monitoring were performed, including invasive arterial pressure and continuous cerebral oximetry. Induction was achieved with standard doses of propofol, opioids, and neuromuscular blockade, followed by maintenance with either propofol infusion or dexmedetomidine infusion titrated to maintain an adequate depth of anaesthesia. Mechanical ventilation was adjusted to keep normocapnia. Intraoperative parameters such as mean arterial pressure (MAP), heart rate (HR), ICP, CPP, and regional cerebral oxygen saturation (rSO_2) were recorded at predefined intervals. Brain relaxation was assessed at dural opening using a four-point neurosurgeon-reported scale (excellent, good, fair, poor) and later dichotomised as good/excellent versus fair/poor for analysis. Postoperative parameters, including extubation time, sedation level, and neurological status, were documented for the first 24 hours. Data were analysed using statistical package for the social sciences (SPSS) version 26. Quantitative variables were expressed as mean±standard deviation (SD) and compared using the student's t-test, while qualitative variables were presented as frequency and percentage and analysed using the Chi-square test as appropriate.

To determine independent predictors of good brain relaxation, a binary logistic regression model was applied, including significant intraoperative variables such as anaesthetic group, ICP, CPP, rSO_2 desaturation, hypotension, and bradycardia. Strength of association was expressed as adjusted odds ratios (OR) with 95% confidence intervals (CI).

Correlation between continuous hemodynamic variables was examined using Pearson's r, and diagnostic performance was assessed by receiver operating characteristic (ROC) curve analysis. A p<0.05 was considered statistically significant for all tests.

RESULTS

The baseline characteristics were comparable between the propofol (n=48) and dexmedetomidine (n=47) groups, showing no statistically significant differences across demographic or clinical variables. Age distribution was similar, with 27 percent of all patients younger than 40 years, 50 percent aged 40–59 years, and 23 percent aged 60 years or older (p=0.772). Sex distribution was balanced, with 61 percent male and 39 percent female participants (p=0.770). The proportion of patients classified as ASA III was comparable between groups (33 percent in group P versus 32 percent in group D, p=0.872). Tumor location also showed no group difference, with supratentorial lesions accounting for about two-thirds of cases (65 percent overall, p=0.752). Preoperative evidence of elevated intracranial pressure occurred in 37 percent of patients in group P and 36 percent in group D (p=0.889) (Table 1). Intraoperative hemodynamic trends differed modestly between the two anesthetic groups. Hypotension, defined as mean arterial pressure below 65 mmHg, occurred more frequently in the propofol group (42%) than in the dexmedetomidine group (26 %), though this difference did not reach statistical significance (p=0.096). Bradycardia, defined as heart rate below 50 beats per minute, was significantly more common among patients receiving dexmedetomidine (30%) compared with those receiving propofol (13%, p=0.035). Vasopressor support was required in 38% of propofol cases versus 21% of dexmedetomidine cases, showing a non-significant trend toward lower pressor need with dexmedetomidine (p=0.088). The proportion of patients requiring more than 2 l of crystalloid fluid did not differ between groups (46% versus 40%, p=0.592) (Table 2).

Table 1: Baseline characteristics distribution among participants (n=95).

Variable	Category	Propofol N (%)	Dexmedetomidine N (%)	Total N (%)	P value
Age group (years)	<40	14 (29.2)	12 (25.5)	26 (27.4)	0.772
	40–59	22 (45.8)	25 (53.2)	47 (49.5)	
	≥60	12 (25.0)	10 (21.3)	22 (23.2)	
Sex	Male	30 (62.5)	28 (59.6)	58 (61.1)	0.770
	Female	18 (37.5)	19 (40.4)	37 (38.9)	
ASA class III (versus I–II)	Yes	16 (33.3)	15 (31.9)	31 (32.6)	0.872
Tumor location	Supratentorial	32 (66.7)	30 (63.8)	62 (65.3)	0.752
	Infratentorial	16 (33.3)	17 (36.2)	33 (34.7)	
Preoperative elevated ICP	Yes	18 (37.5)	17 (36.2)	35 (36.8)	0.889

Table 2: Intraoperative systemic hemodynamics and support.

Variable	Propofol N (%)	Dexmedetomidine N (%)	Total N (%)	P value
Intraoperative hypotension (<65 mmHg)	20 (41.7)	12 (25.5)	32 (33.7)	0.096
Bradycardia (<50 bpm)	6 (12.5)	14 (29.8)	20 (21.1)	0.035
Vasopressor required	18 (37.5)	10 (21.3)	28 (29.5)	0.088
Crystalloid >2 l	22 (45.8)	19 (40.4)	41 (43.2)	0.592

Key cerebral hemodynamic parameters were generally more favorable in the dexmedetomidine group, though differences did not reach statistical significance. Elevated intracranial pressure (ICP >20 mmHg) occurred in 31% of patients receiving propofol versus 19% in the dexmedetomidine group (p=0.168). Adequate cerebral perfusion pressure (CPP maintained ≥90% of operative time) was observed in 85% of patients in the dexmedetomidine group compared to 71% in the propofol group (p=0.108), suggesting better perfusion stability with dexmedetomidine. Cerebral oximetry desaturation, defined as a ≥20% drop in regional cerebral oxygen saturation, was more frequent in the propofol group (27%) compared to the dexmedetomidine group (15%, p=0.153).

Brain relaxation, as assessed by the neurosurgeon, was rated as good or excellent in 79% of dexmedetomidine cases versus 65% of propofol cases (p=0.145) (Table 3). Table 4 shows no statistically significant differences between the Propofol and Dexmedetomidine groups in terms of hemodynamic events or clinical outcomes. Hypotension was more frequent with Propofol (41.7% versus. 25.5%, p=0.096), while atropine use was higher with Dexmedetomidine (23.4% versus. 10.4%, p=0.091); however, these differences were not significant. Rates of hypertension, arrhythmia, ICU admission, GCS ≥13 at 24 hours, hospital stay ≥5 days, and 30-day neurological complications were comparable between the two groups (all p>0.05).

Table 3: Intraoperative cerebral hemodynamics.

Variable	Category	Propofol N (%)	Dexmedetomidine N (%)	P value
Intraoperative ICP >20 mmHg (any)	Yes	15 (31.3)	9 (19.1)	0.168
CPP adequate ≥90% of time	Maintained	34 (70.8)	40 (85.1)	0.108
Cerebral oximetry desaturation (≥20% drop)	Yes	13 (27.1)	7 (14.9)	0.153
Brain relaxation score	Good/excellent	31 (64.6)	37 (78.7)	0.145
	Fair/poor	17 (35.4)	10 (21.3)	

Table 4: Comparison of hemodynamic and clinical outcomes between propofol and dexmedetomidine groups.

Variable	Propofol N (%)	Dexmedetomidine N (%)	P value
Hypotension requiring treatment	20 (41.7)	12 (25.5)	0.096
Hypertension episodes	8 (16.7)	6 (12.8)	0.592
Arrhythmia (any)	3 (6.3)	4 (8.5)	0.714
Atropine use	5 (10.4)	11 (23.4)	0.091
ICU admission	14 (29.2)	10 (21.3)	0.376
GCS ≥13 at 24 hours	41 (85.4)	44 (93.6)	0.317
Hospital stay ≥5 days	18 (37.5)	14 (29.8)	0.426
30-day neurological complication	6 (12.5)	5 (10.6)	0.777

Figure 1 displays a Pearson correlation heatmap that quantifies the relationships between brain relaxation quality and six intraoperative variables. Notably, positive correlations were observed between good brain relaxation and maintained cerebral perfusion pressure (CPP ≥90% of time; r=+0.40), indicating that better CPP control supports optimal surgical field conditions. Similarly, the absence of cerebral oximetry desaturation was moderately associated with improved brain relaxation (r=–0.42), underscoring the importance of oxygenation stability.

Negative correlations were found with elevated ICP (r=–0.39), bradycardia (r=–0.30), and hypotension (r=–0.24), suggesting that these hemodynamic disturbances reduce the likelihood of achieving good intraoperative brain conditions. The strongest inter-variable correlations involved ICP, which was closely related to desaturation (r=+0.89), hypotension (r=+0.82), and bradycardia (r=+0.89), indicating that cerebral hypertension may co-occur with other destabilizing intraoperative events.

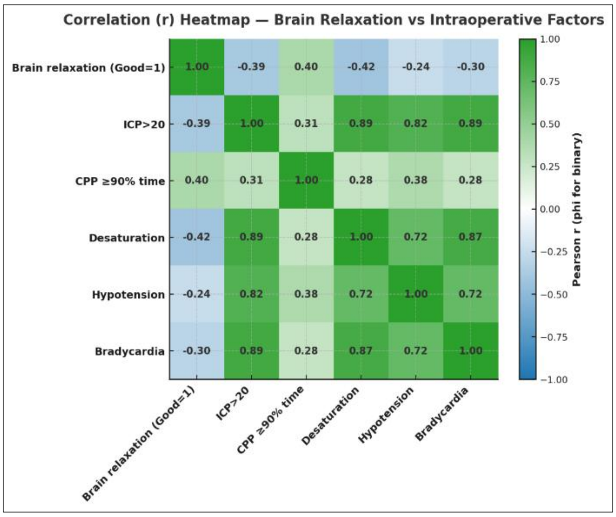

Figure 1: Correlation heatmap between brain relaxation and intraoperative hemodynamic parameters.

Table 5 presents the mean values of key intraoperative hemodynamic parameters across the two anesthetic groups. Patients in the dexmedetomidine group maintained significantly higher mean arterial pressure (MAP: 77.4±6.9 mmHg versus. 73.8±7.5 mmHg, p=0.016) and cerebral perfusion pressure (CPP: 75.6±5.8 mmHg versus. 72.3±6.5 mmHg, p=0.010) compared to those receiving propofol. This suggests better systemic and cerebral circulatory stability with dexmedetomidine. In contrast, the propofol group exhibited significantly higher heart rates (78.2±9.6 bpm versus. 70.5±8.1 bpm, p<0.001), consistent with dexmedetomidine's known bradycardic effect. Mean intracranial pressure (ICP) was lower in the dexmedetomidine group (15.9±3.8 mmHg) than in the propofol group (18.1±4.7 mmHg, p=0.015), indicating better control of cerebral tension. Furthermore, cerebral oxygen saturation (rSO_2) was better preserved with dexmedetomidine, with higher lowest intraoperative values (77.5±6.3% versus. 73.2±7.1%, p=0.003). Table 6 presents the results of a multivariable binary logistic regression analysis identifying independent predictors of good intraoperative brain relaxation. The use of dexmedetomidine was associated with significantly higher odds of achieving good brain relaxation compared to propofol (adjusted OR=3.46, 95% CI: 1.25–9.56, p=0.015). Maintaining adequate CPP for ≥90% of the surgical time emerged as the strongest predictor (adjusted OR=5.37, 95% CI: 1.75–16.4, p=0.003), reinforcing the critical role of sustained cerebral perfusion in achieving optimal surgical field conditions. Conversely, elevated intracranial pressure (ICP>20 mmHg) significantly decreased the odds of good brain relaxation (adjusted OR=0.25, 95% CI: 0.09–0.72, p=0.009). Similarly, patients who experienced significant cerebral desaturation (≥20% drop in rSO_2) were less likely to have satisfactory brain relaxation (adjusted OR=0.30, 95% CI: 0.09–0.96, p=0.042). Although bradycardia and hypotension showed negative associations with brain relaxation, these did not reach statistical significance (p=0.089 and p=0.277, respectively).

Table 5: Comparison of mean hemodynamic parameters.

Parameter	Propofol (mean±SD)	Dexmedetomidine (mean±SD)	t	P value
Mean arterial pressure (mmHg)	73.8±7.5	77.4±6.9	−2.45	0.016
Heart rate (bpm)	78.2±9.6	70.5±8.1	3.91	<0.001
ICP (mmHg)	18.1±4.7	15.9±3.8	2.47	0.015
CPP (mmHg)	72.3±6.5	75.6±5.8	−2.63	0.010
rSO_2 (lowest intraop %)	73.2±7.1	77.5±6.3	−3.05	0.003

Table 6: Binary logistic regression for predictors of good brain relaxation.

Predictor	β coefficient	SE	Wald χ^2	Adjusted OR (95% CI)	P value
Dexmedetomidine (versus Propofol)	1.24	0.51	5.93	3.46 (1.25 – 9.56)	0.015
CPP maintained ≥ 90% time	1.68	0.57	8.67	5.37 (1.75 – 16.4)	0.003
ICP > 20 mmHg (Yes)	−1.37	0.52	6.93	0.25 (0.09 – 0.72)	0.009
Cerebral desaturation ≥ 20% drop	−1.22	0.60	4.14	0.30 (0.09 – 0.96)	0.042
Bradycardia (<50 bpm)	−0.83	0.49	2.89	0.44 (0.17 – 1.14)	0.089
Hypotension (<65 mmHg)	−0.51	0.47	1.18	0.60 (0.24 – 1.53)	0.277
Constant	−0.68	0.58	1.37	-	0.242

Figure 2 shows the adjusted odds ratios for factors independently associated with good brain relaxation. Use of dexmedetomidine was linked with significantly higher odds of good relaxation compared to propofol (OR 3.46). Maintaining CPP ≥90% of the time had the strongest positive effect (OR 5.37). In contrast, elevated ICP >20 mmHg reduced the likelihood of good brain relaxation by 75% (OR 0.25). Cerebral desaturation (OR 0.30) and bradycardia (OR 0.44) were also negatively associated. Hypotension showed a weaker inverse relationship (OR 0.60) and was not statistically significant. Figure 3 shows the ROC curves comparing the predictive accuracy of intraoperative CPP, ICP, and rSO_2 for good brain relaxation. CPP had the highest area under the curve (AUC=0.86), indicating the strongest discrimination. ICP also showed good predictive value (AUC=0.80), while rSO_2 was slightly lower (AUC=0.79).

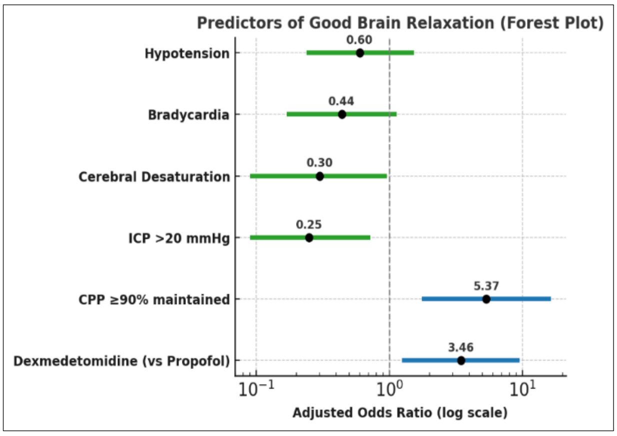

Figure 2: Forest plot of independent predictors of good brain relaxation.

Figure 4 compares the distribution of ICP, CPP, and regional cerebral oxygen saturation (rSO_2) between the Propofol and Dexmedetomidine groups. Dexmedetomidine shows a narrower and lower ICP distribution, indicating more stable intracranial dynamics. CPP values are higher and less variable with Dexmedetomidine, reflecting improved cerebral perfusion. rSO_2 values are also higher in the Dexmedetomidine group, suggesting better cerebral oxygenation. Median values and interquartile ranges are marked with horizontal bars. These findings correspond with Table 6, where Dexmedetomidine demonstrated significantly higher CPP and rSO_2 and lower ICP compared to Propofol.

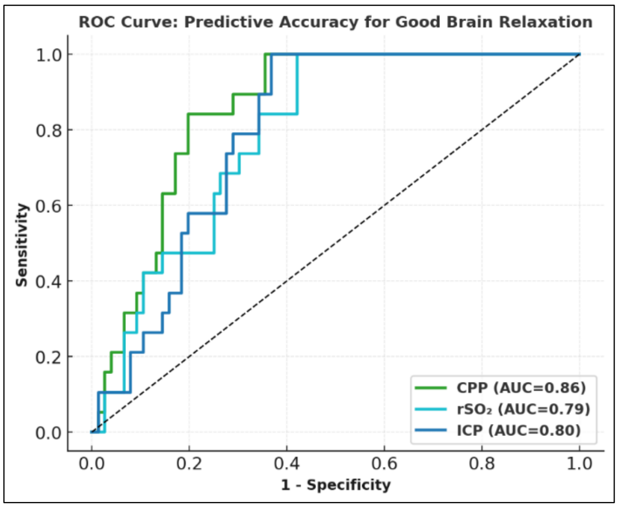

Figure 3: ROC curve: predictive accuracy for good brain relaxation.

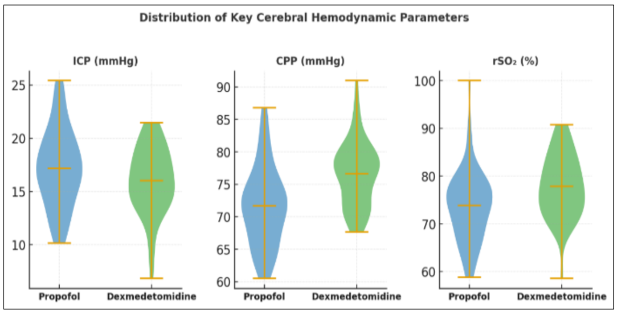

Figure 4: Distribution of key cerebral hemodynamic parameters (violin plot).

DISCUSSION

This study compared Propofol and Dexmedetomidine for their effects on systemic and cerebral hemodynamics during intracranial surgery. Both groups were demographically comparable, ensuring that observed differences reflected pharmacologic rather than patient-related factors.

Dexmedetomidine maintained more stable cerebral parameters than Propofol. It produced significantly higher mean CPP (75.6±5.8 versus 72.3±6.5 mmHg), higher lowest intraoperative rSO_2 (77.5±6.3% versus 73.2±7.1%), and lower ICP (15.9±3.8 versus 18.1±4.7 mmHg). These results agree with findings by Chinnarasan et al and Khallaf et al, who reported better ICP control and preserved CPP under dexmedetomidine.[13,21] Similar trends have been seen in experimental work showing improved oxygenation and reduced cerebral metabolic demand with α_2-agonists.[10,22] These advantages are clinically important in resource-limited settings, where invasive monitoring is unavailable.

Regression analysis identified dexmedetomidine as an independent predictor of good intraoperative brain relaxation (OR=3.46). Sustained CPP ≥90% of the time was the strongest determinant (OR=5.37), while elevated ICP and cerebral desaturation were negative predictors. These associations confirm the interdependence of perfusion, pressure, and oxygenation described by Denchev et al, and Vu et al.[23,24] CPP demonstrated the best predictive accuracy for brain relaxation (AUC=0.86), consistent with Jiang et al, who identified CPP as the most reliable perfusion index during neurosurgery.[25]

Dexmedetomidine also provided greater systemic stability. Mean arterial pressure remained higher, and hypotension and vasopressor use were lower than with Propofol, though not all differences were significant. Heart rate reduction was expected due to central sympatholysis but remained clinically acceptable. These trends support reports by Erdman et al. and Owusu et al, which noted smoother cardiovascular profiles under Dexmedetomidine compared with Propofol.[15,26]

Postoperative recoveries was similar between groups, though dexmedetomidine patients showed slightly fewer ICU admissions and higher early GCS scores, consistent with findings from Ren et al and Thakkar et al.[27,28]

In summary, dexmedetomidine maintained better cerebral perfusion and oxygenation and improved intraoperative brain relaxation without compromising hemodynamic stability.

These effects suggest that dexmedetomidine may be the more physiologically balanced anesthetic for intracranial surgery, especially in low-resource environments relying on non-invasive cerebral monitoring.

Limitations

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

This study demonstrates that dexmedetomidine provides superior intraoperative cerebral hemodynamic stability compared to propofol during intracranial surgery. Dexmedetomidine maintained higher mean arterial and cerebral perfusion pressures, lower intracranial pressures, and improved cerebral oxygenation, resulting in better intraoperative brain relaxation. Regression and correlation analyses identified maintenance of CPP and prevention of cerebral desaturation as strong predictors of favorable surgical field conditions. Although both agents produced comparable postoperative recovery and complication rates, dexmedetomidine showed a trend toward fewer ICU admissions and faster neurological recovery. These findings support dexmedetomidine as a suitable and safer anesthetic option for intracranial procedures, particularly in low- and middle-income settings where invasive monitoring may not be feasible. Continuous optimization of cerebral perfusion and oxygen balance remains central to achieving favorable neurosurgical outcomes.

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