

Induced Hypotensive Anaesthesia by Remifentanil and Dexmedetomidine during Supratentorial Craniotomy

Tushar Das¹, Mustafa Kamal², Saiful Mahmud Tusher^{3*}, Suravi Roy⁴, Nahidul Akbor⁵, Risul Islam⁶, Khadiga Akter⁷, Mohammad Ezazul Hoque⁸

ARTICLE INFO

Received: 9 June 2026
Accepted: 15 June 2026
Published Online: 19 June 2026

DOI: 10.5281/zenodo.20760802

Volume: 9, Number: 4, Page: 172-177

e-ISSN: 2789-5912
ISSN: 2617-0817

*Corresponding author



ABSTRACT

Background: Controlled hypotensive anaesthesia is routinely applied in supratentorial craniotomy to optimize the surgical field and reduce blood loss. Dexmedetomidine and remifentanil are widely used adjuvants with distinct pharmacological profiles. **Objective:** To compare the effects of dexmedetomidine and remifentanil as adjuvants to propofol–isoflurane anaesthesia on intraoperative haemodynamics, anaesthetic consumption and perioperative outcomes. **Methods & Materials:** This quasi-experimental study included 40 ASA I–II patients undergoing elective supratentorial craniotomy, allocated into two equal groups: dexmedetomidine (Group D) and remifentanil (Group R). Haemodynamic variables, drug requirements, blood loss, surgical duration, and recovery profile were recorded and analysed using SPSS v27. A p-value <0.05 was considered significant. **Results:** Significant reductions in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were observed in Group R compared to Group D during most intraoperative time points (p<0.05). MAP was consistently lower in Group R from 30 minutes to 3 hours post drug administration (p=0.001). Group R also demonstrated lower SBP and DBP during sustained intraoperative periods. Total propofol consumption was comparable between groups, although maintenance adjuvant requirements differed significantly (p=0.001). Blood loss was lower in Group R. Duration of surgery and anaesthesia were similar between groups. Recovery profiles were slightly better in Group R. **Conclusion:** Remifentanil-based controlled hypotensive anaesthesia provides more stable intraoperative haemodynamics and improved surgical conditions compared to dexmedetomidine in supratentorial craniotomy.

Keywords: Controlled hypotension, dexmedetomidine, remifentanil, supratentorial craniotomy, neuroanaesthesia

1. Senior Specialist, Neuro ICU, BRB Hospital, Dhaka, Bangladesh (ORCID: 0009-0008-5638-592X)
2. Professor and Chairman, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangladesh Medical University, Dhaka, Bangladesh (ORCID: 0000-0002-4665-1904)
3. Assistant Professor, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangladesh Medical University, Dhaka, Bangladesh (ORCID: 0009-0006-4297-3355)
4. NICU Medical Officer, Islami Bank Central Hospital, Kakrail, Dhaka, Bangladesh (ORCID: 0009-0004-9489-6940)
5. Register, National Institute of Neuro Sciences & Hospital, Dhaka, Bangladesh (ORCID: 0009-0002-5735-0784)
6. Specialist, Asgar Ali Medical College & Hospital, Dhaka, Bangladesh (ORCID: 0009-0008-2070-7536)
7. Junior Consultant, Department of Anaesthesiology, Uttara Adhunik Medical College Hospital, Dhaka, Bangladesh (ORCID: 0009-0002-9726-8195)
8. Resident, Bangladesh Medical University, Dhaka, Bangladesh (ORCID: 0009-0003-2998-8876)

INTRODUCTION

Induced hypotension, also termed controlled hypotension, refers to the deliberate reduction of arterial blood pressure during surgery to minimize blood loss, improve the surgical field visibility and reduce the need for blood transfusion. This technique holds particular importance in neurosurgical procedures such as supratentorial craniotomy, where minimizing intracranial pressure (ICP) and optimizing the operative field are prerequisites for successful outcomes. Controlled hypotensive anaesthesia (CHA) is widely employed in this setting, targeting a reduction in mean arterial pressure (MAP) by 20–30% from baseline values to enhance surgical conditions without compromising cerebral perfusion pressure (CPP) [1,2]. Supratentorial craniotomy is among the most technically demanding neurosurgical procedures, requiring a delicate balance

between adequate anaesthetic depth and hemodynamic stability. The surgical approach involves access to brain structures above the tentorium cerebelli, where fluctuations in blood pressure can have serious consequences including cerebral ischemia, haemorrhage, or increased ICP. Effective anaesthetic management must therefore ensure hemodynamic stability throughout the procedure while facilitating optimal operating conditions for the neurosurgeon [3,4].

Volatile anaesthetics such as isoflurane, intravenous agents including propofol and opioids such as remifentanil are commonly employed in neuroanaesthesia. These agents are frequently supplemented with adjuvants capable of modulating sympathetic tone and providing hemodynamic stability. Among these, dexmedetomidine has attracted considerable interest for its alpha-2

adrenergic agonist properties, which reduce sympathetic outflow and confer sedative, analgesic and anaesthetic-sparing effects [5,6].

Remifentanil is an ultra-short-acting synthetic opioid metabolized by plasma esterases, providing a half-life of approximately 10–20 minutes and allowing rapid titration and precise hemodynamic control. Its rapid onset and offset make it particularly advantageous in procedures requiring tight control of arterial pressure and swift neurological assessment upon emergence. Studies have demonstrated its efficacy in maintaining intraoperative hemodynamic stability during neurosurgical procedures [7,8].

Dexmedetomidine, as a selective alpha-2 adrenergic receptor agonist, provides sedation closely resembling natural sleep without significant respiratory depression, making it valuable in neuroanaesthesia. By acting on locus coeruleus alpha-2

receptors, it reduces norepinephrine release and sympathetic tone, resulting in dose-dependent decreases in heart rate and blood pressure. Its cerebroprotective properties, including preservation of cerebral autoregulation and reduction of ICP, further support its use in cranial procedures [9].

Despite the recognized roles of both agents in CHA, comparative data between dexmedetomidine and remifentanyl specifically concerning hemodynamic profiles, drug consumption patterns, blood loss and recovery outcomes in patients undergoing supratentorial craniotomy remain limited. Both agents reduce anaesthetic requirements and provide hemodynamic stability, but their pharmacokinetic and pharmacodynamic differences may translate into clinically meaningful distinctions in the neurosurgical context [10].

The present study was designed to fill this gap by directly comparing the intraoperative hemodynamic effects of dexmedetomidine and remifentanyl as adjuvants to isoflurane-propofol anaesthesia during supratentorial craniotomy. The study further evaluates propofol and adjuvant consumption, surgical duration, intraoperative blood loss, recovery scores and surgeon satisfaction. By systematically assessing these endpoints, the investigation aims to identify the more favourable pharmacological strategy for achieving controlled hypotension in patients undergoing this complex neurosurgical procedure.

OBJECTIVES

The objective of this study was to compare the effects of dexmedetomidine and remifentanyl as adjuvants to isoflurane-propofol-based anaesthesia on intraoperative haemodynamics, anaesthetic drug consumption, intraoperative blood loss, recovery profile and surgeon satisfaction in patients undergoing supratentorial craniotomy.

METHODS & MATERIALS

This was a single-blinded quasi-experimental study conducted at the Department of Anaesthesia, Analgesia and Intensive Care Medicine in collaboration with the Department of Neurosurgery, Bangladesh Medical University (BMU), Dhaka, Bangladesh, over a period of 12 months (March 2024 to February 2025). Forty patients aged 18 to 65 years with American Society of Anaesthesiologists (ASA) physical status I or II, scheduled for elective supratentorial craniotomy under general anaesthesia, were enrolled.

Inclusion criteria:

1. Patients aged 18-65 years
2. ASA physical status I or II
3. Mallampati class I or II
4. Scheduled for elective supratentorial craniotomy
5. Glasgow Coma Scale score of 15
6. Provided written informed consent

Exclusion criteria:

1. Known hypersensitivity to dexmedetomidine, remifentanyl, or propofol
2. Severe hepatic or renal impairment
3. History of significant cardiovascular disease or cardiac conduction defects
4. Uncontrolled hypertension or severe respiratory disease
5. Neurological conditions such as cerebral aneurysms or impending intracranial hypertension
6. History of psychiatric illness
7. Pregnant or lactating women
8. Emergency surgery
9. Refusal to provide informed consent

Data Collection Procedure

All patients were kept nil per oral for eight hours before surgery. Baseline preoperative investigations and anthropometric measurements were recorded. Inside the operating room, central venous access was established and patients received pantoprazole 40 mg intravenously as premedication. Baseline heart rate, non-invasive blood pressure, MAP and oxygen saturation (SpO₂) were recorded using a multiparameter monitor before induction. The forty patients were equally allocated into two groups using computer-generated random numbers: Group D (dexmedetomidine) and Group R (remifentanyl), each comprising 20 patients. Both groups received propofol 2 mg/kg intravenously for induction and a maintenance infusion of 50-150 mcg/kg/min via syringe pump. Suxamethonium chloride 1.5 mg/kg was administered for intubation, followed by vecuronium bromide 0.1 mg/kg for neuromuscular relaxation with intermittent top-up doses of 0.01 mg/kg at 20-30-minute intervals. Isoflurane was maintained at 0.4 MAC with oxygen and nitrous oxide in a 50:50 ratio. Intravenous paracetamol 15 mg/kg was given for analgesia.

Group D received dexmedetomidine 1 mcg/kg as an intravenous bolus over 10 minutes (5 minutes before induction), followed by a continuous intraoperative infusion at 0.8 mcg/kg/hr. Group R

received remifentanyl 1 mcg/kg as an intravenous bolus 5 minutes before induction, followed by a continuous infusion at 0.4 mcg/kg/min throughout surgery. Haemodynamic parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and MAP were monitored and recorded at baseline, before drug administration, 5 minutes, 30 minutes and at 30-minute intervals thereafter until leaving the operating theatre. The Modified Aldrete Score was assessed 10 minutes after surgery and at the time of transfer to the recovery room.

Ethical Consideration

The study protocol received formal approval from the Institutional Review Board (IRB) and Ethical Review Committee of BMU (former BSMMU) before commencement. Written informed consent was obtained from all patients or their legal guardians in an easily comprehensible local language before enrolment. Patients were clearly informed of the study purpose, procedures, potential benefits and risks and were assured of their right to withdraw at any time without consequences to their medical care. Participant confidentiality was maintained by coding all research data, restricting data access to the principal investigator and assigning unique identification numbers to each participant. No financial incentives were provided for participation.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics, Version 27 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as mean plus or minus standard deviation. Comparison of numerical variables between the two groups was performed using the independent samples Student t-test. Categorical variables were compared using the chi-square test. A p-value of less than 0.05 was considered statistically significant.

RESULT

Table 1 demonstrates that both groups were well-matched in demographic characteristics. The mean age was 40.40±9.13 years in Group D and 40.48±9.28 years in Group R (p=0.975). Body mass index was similarly comparable (23.5±1.4 kg/m² vs 23.2±1.3 kg/m², p=0.473). The distributions of ASA class, gender and Mallampati class showed no statistically significant differences between the groups (p>0.05 for all), confirming demographic homogeneity and enabling valid intergroup comparisons.

Table I

Demographic characteristics of the patients in both Groups (n=40).

Characteristics	Group D (n=20)		Group R (n=20)	
Age (years)	40.40±9.13		40.48±9.28 (p=0.975)	
BMI (kg/m ²)	23.5±1.4		23.2±1.3 (p=0.473)	
ASA Class	Class I	13 (65%)	14 (70%)	0.239
	Class II	7 (35%)	6 (30%)	0.237
Gender	Male	8 (40%)	7 (35%)	0.773
	Female	12 (60%)	13 (65%)	0.772
Mallampati Class	Class I	14 (70%)	8 (40%)	0.152
	Class II	6 (30%)	12 (60%)	0.15

Table II reveals that baseline heart rate was comparable between groups (Group D 83.8±6.1 bpm; Group R 84.2±7.3 bpm; p=0.852). From 5 minutes after drug administration through 3.5 hours, Group R consistently demonstrated significantly lower heart rates compared to Group D

(p=0.001 at each of these time points). The most pronounced difference was observed at 3 hours (Group R 70.8±4.1 vs Group D 84.1±6.5 bpm). Heart rate values in both groups returned to comparable ranges at 4 hours, 10 minutes following anaesthesia discontinuation and at the time of leaving

the operating theatre, indicating that the more pronounced bradycardic effect of remifentanyl was sustained during the operative period but resolved toward the end.

Table II

Comparison of Heart Rate (HR) between the two Groups (n=40) at different time points.

Time Point	Group D (n=20)	Group R (n=20)	p-value
Baseline (T0)	83.8±6.1	84.2±7.3	0.852
Before drug administration (T1)	83.2±7.6	79.0±7.9	0.063
5 min after drug administration (T2)	86.0±12.0	73.2±7.4	0.001*
30 min after drug administration (T3)	85.4±15.3	72.0±10.1	0.001*
1 hr after drug administration (T4)	83.4±11.1	71.7±8.7	0.001*
1.5 hrs after drug administration (T5)	84.4±13.5	72.7±8.2	0.001*
2 hrs after drug administration (T6)	84.6±6.8	73.0±8.8	0.001*
2.5 hrs after drug administration (T7)	86.1±6.7	72.8±8.7	0.001*
3 hrs after drug administration (T8)	84.1±6.5	70.8±4.1	0.001*
3.5 hrs after drug administration (T9)	87.2±10.6	72.3±6.3	0.001*
4 hrs after drug administration (T10)	86.7±8.6	80.1±6.3	0.404
10 min following end of anaesthesia (T11)	85.9±7.0	81.0±8.3	0.079
During leaving OT (T12)	86.1±7.0	82.5±6.0	0.054

Table III shows that baseline SBP did not differ significantly between the groups (128.3±7.3 mmHg in Group D vs 124.7±6.4 mmHg in Group R; p=0.075). Statistically significant differences in SBP

emerged from 30 minutes through 3 hours after drug administration, with Group R demonstrating consistently lower values (105.5±8.3 to 108.4±3.8 mmHg) compared to Group D (116.0±8.1 to 116.3±2.8

mmHg), all with p=0.001. These differences resolved at 3.5 hours and beyond, with comparable values recorded at the time of leaving the operating theatre (119.8±5.7 vs 120.3±5.8 mmHg, p=0.773).

Table III

Comparison of Systolic Blood Pressure (SBP) between the two Groups (n=40) at different time points.

Time Point	Group D (n=20)	Group R (n=20)	p-value
Baseline (T0)	128.3±7.3	124.7±6.4	0.075
Before drug administration (T1)	121.8±7.8	120.9±6.9	0.66
5 min after drug administration (T2)	113.1±13.7	109.0±12.1	0.288
30 min after drug administration (T3)	116.0±8.1	105.5±8.3	0.001*
1 hr after drug administration (T4)	117.0±5.6	102.7±6.8	0.001*
1.5 hrs after drug administration (T5)	113.0±7.1	103.1±7.3	0.001*
2 hrs after drug administration (T6)	113.6±6.2	103.5±7.6	0.001*
2.5 hrs after drug administration (T7)	115.0±4.5	103.7±5.7	0.001*
3 hrs after drug administration (T8)	116.3±2.8	108.4±3.8	0.001*
3.5 hrs after drug administration (T9)	119.2±6.5	110.0±4.1	0.06
4 hrs after drug administration (T10)	115.2±6.2	106.0±4.3	0.06
10 min following end of anaesthesia (T11)	117.2±6.5	110.0±4.1	0.083
During leaving OT (T12)	120.3±5.8	119.8±5.7	0.773

Table IV shows that DBP values at baseline and up to 30 minutes were comparable between the groups. Significant differences were observed from 1 hour through 3.5 hours after drug administration, with Group

R consistently exhibiting lower DBP values. The differences were most pronounced at 2.5 hours (72.6±3.2 mmHg in Group D vs 68.5±4.5 mmHg in Group R, p=0.002) and 1.5 hours (71.5±3.9 vs

67.8±5.1 mmHg, p=0.006). No significant differences were observed after 4 hours or at the time of leaving the operating theatre.

Table IV

Comparison of Diastolic Blood Pressure (DBP) between two Groups (n=40) at different time points.

Time Point	Group D (n=20)	Group R (n=20)	p-value
Baseline (T0)	80.7±5.8	79.4±4.3	0.383
Before drug administration (T1)	74.9±6.5	75.9±5.0	0.58
5 min after drug administration (T2)	68.0±10.1	70.5±8.5	0.444
30 min after drug administration (T3)	70.3±5.5	68.8±6.1	0.395
1 hr after drug administration (T4)	71.8±3.5	69.0±4.2	0.016
1.5 hrs after drug administration (T5)	71.5±3.9	67.8±5.1	0.006
2 hrs after drug administration (T6)	71.1±4.1	68.3±4.4	0.025
2.5 hrs after drug administration (T7)	72.6±3.2	68.5±4.5	0.002
3 hrs after drug administration (T8)	72.5±4.0	68.4±3.8	0.009
3.5 hrs after drug administration (T9)	76.7±3.5	69.5±3.3	0.031
4 hrs after drug administration (T10)	74.6±3.2	71.5±4.5	0.401
10 min following end of anaesthesia (T11)	76.3±4.3	73.7±4.2	0.24
During leaving OT (T12)	76.7±5.1	74.7±5.2	0.158

Table V indicates that MAP was comparable at baseline and before drug administration between the two groups. Significant reductions in MAP in Group R were observed from 30 minutes (81.2±6.4

vs 85.6±5.3 mmHg, p=0.011) through 3 hours (81.5±2.6 vs 87.1±2.5 mmHg, p=0.001) of drug administration. Beyond 3.5 hours, MAP values were not significantly different between groups and

both groups maintained haemodynamically acceptable MAP levels throughout the procedure.

Table V

Comparison of Mean Arterial Pressure (MAP) between two Groups (n=40) at different time points.

Time Point	Group D (n=20)	Group R (n=20)	p-value
Baseline (T0)	96.7±5.6	94.4±4.1	0.11
Before drug administration (T1)	90.7±5.6	89.1±4.0	0.78
5 min after drug administration (T2)	83.6±10.2	83.05±9.0	0.795
30 min after drug administration (T3)	85.6±5.3	81.2±6.4	0.011*
1 hr after drug administration (T4)	86.9±3.6	80.4±4.5	0.001*
1.5 hrs after drug administration (T5)	85.3±4.0	79.7±4.7	0.001*
2 hrs after drug administration (T6)	85.2±3.6	80.4±4.0	0.001*
2.5 hrs after drug administration (T7)	86.7±3.0	80.5±4.0	0.001*
3 hrs after drug administration (T8)	87.1±2.5	81.5±2.6	0.001*
3.5 hrs after drug administration (T9)	87.3±5.2	83.0±4.1	0.127
4 hrs after drug administration (T10)	87.3±3.6	83.0±3.5	0.234
10 min following end of anaesthesia (T11)	89.3±7.1	86.0±3.2	0.124
During leaving OT (T12)	91.3±4.5	89.0±4.4	0.473

Table VI demonstrates that the initial bolus dose and total propofol requirement were comparable between groups (Group D 1378.1±315.40 mg vs Group R 1457.7±186.6 mg; p=0.776). The induction

dose of adjuvant was similar in both groups (68.3±7.08 mcg in Group D vs 70.52±20.6 mcg in Group R; p=0.675). However, the maintenance dose of adjuvant was significantly higher in Group R

(5293.3±7.08 mcg) compared to Group D (178.3±40.08 mcg), reflecting the pharmacokinetic differences between remifentanyl and dexmedetomidine with a highly significant p-value of 0.001.

Table VI

Comparison of mean amount of Propofol and Adjuvants requirement during surgery between the Groups (n=40).

Requirements	Group D (n=20)	Group R (n=20)	p-value
Propofol Initial Bolus dose (mg)	136.6±14.40	134.4±13.6	0.932
Maintained dose of Propofol (mg)	1241.5±301	1323.3±173	0.719
Total required Propofol (mg)	1378.1±315.40	1457.7±186.6	0.776
Adjuvants required - Induction (Dexmedetomidine/Remifentanyl) (mcg)	68.3±7.08	70.52±20.6	0.675
Adjuvants required - Maintenance (Dexmedetomidine/Remifentanyl) (mcg)	178.3±40.08	5293.3±7.08	0.001*

Table VII shows no statistically significant difference in the duration of surgery (Group D 3.2±5.7 hours vs Group R 3.4±6.8 hours, p=0.641) or the duration of anaesthesia (Group D 4.6±6.0 hours vs Group R 3.9±7.0 hours, p=0.696) between

the two groups. This confirms procedural comparability and supports the validity of other outcome comparisons. Intraoperative blood loss was slightly lower in Group D (177±67 ml) compared to Group R (188±6.8 ml), though this difference did

not reach statistical significance (p=0.608). Both groups-maintained blood loss within clinically acceptable ranges without requiring intraoperative blood transfusion.

Table VIIComparison of duration of surgery, Blood loss and anaesthesia between two Groups ($n=40$).

Variable	Group D (n=20)	Group R (n=20)	p-value
Duration of Surgery (Hours)	3.2±5.7	3.4±6.8	0.641
Duration of Anaesthesia (Hours)	4.6±6.0	3.9±7.0	0.696
Blood loss during Surgery (ml)	177±67	188±6.8	0.608

Table VIII shows that a good recovery profile was observed in 60% of patients in Group R compared to 52% in Group D, though this difference was not statistically significant ($p=0.790$). Surgeon satisfaction

was similar across both groups, with 56% of surgeons satisfied in Group D and 48% in Group R ($p=0.760$). Despite the lack of statistical significance, the numerically higher recovery rate in Group R aligns with

remifentanyl's rapid offset and suggests a clinically relevant trend toward better early recovery.

Table VIIIComparison of recovery profile and surgeons' satisfaction between the two Groups ($n=40$).

Variable	Group D (n=20)	Group R (n=20)	p-value
Recovery profile - Bad	12 (48%)	10 (40%)	0.776
Recovery profile - Good	13 (52%)	15 (60%)	0.79
Surgeons' satisfaction - Satisfied	14 (56%)	13 (48%)	0.76
Surgeons' satisfaction - Unsatisfied	11 (44%)	12 (52%)	0.812

DISCUSSION

The present study compared dexmedetomidine and remifentanyl as adjuvants to isoflurane-propofol anaesthesia in patients undergoing supratentorial craniotomy, with a focus on intraoperative haemodynamics, drug consumption, blood loss and recovery outcomes. Baseline demographic characteristics including age, body mass index, ASA classification, gender distribution and Mallampati class were statistically comparable between the two groups, confirming baseline homogeneity and ensuring the reliability of subsequent comparisons.

The most striking haemodynamic finding was the superior blood pressure and heart rate control achieved with remifentanyl during the intraoperative period. Group R exhibited significantly lower heart rate from 5 minutes through 3.5 hours after drug administration and significantly lower SBP between 30 minutes and 3 hours ($p=0.001$), along with reductions in DBP and MAP during the same window. These findings are consistent with the recognized pharmacological profile of remifentanyl as an ultra-short-acting opioid capable of rapid and precise haemodynamic titration. Engelhardt et al. previously described remifentanyl's efficacy in maintaining intraoperative stability during neurosurgical procedures, attributing it to its predictable onset and rapid esterase-mediated metabolism [11]. Similarly, Kim et al. reported superior haemodynamic control with remifentanyl in patients undergoing craniotomy, noting its advantage of allowing swift adjustments to changes in surgical stimulation [12].

Dexmedetomidine, while producing meaningful reductions in sympathetic tone and blood pressure, showed comparatively

less intraoperative haemodynamic reduction during the operative period. This likely relates to its slower onset, longer context-sensitive half-life and the nature of its central sympatholytic mechanism, which, while effective, does not offer the same moment-to-moment titratability as remifentanyl. Zorrilla-Vaca et al. noted in their meta-analysis that dexmedetomidine provides stable haemodynamics but may be less responsive to acute intraoperative demands [10]. Bhana et al. similarly acknowledged that dexmedetomidine's cardiovascular effects, while predictable, are less rapidly titratable than opioid-based regimens [13].

Concerning drug consumption, total propofol requirements were comparable between groups, which is consistent with the documented anaesthetic-sparing properties of both dexmedetomidine and remifentanyl. Dexmedetomidine reduced anaesthetic requirements through its alpha-2 agonist-mediated sedative and analgesic effects, while remifentanyl contributed through its opioid receptor-mediated analgesia. Bekker et al. demonstrated that dexmedetomidine substantially reduces intraoperative opioid and anaesthetic needs in neurosurgical patients [14]. The markedly higher maintenance adjuvant dose required in Group R (5293.3 ± 7.08 mcg vs 178.3 ± 40.08 mcg, $p=0.001$) reflects the continuous pharmacokinetic demand of remifentanyl's rapid metabolism by plasma esterases, contrasted with dexmedetomidine's considerably lower dosing requirement over equivalent time intervals.

Intraoperative blood loss was marginally lower in Group D (177 ± 67 ml) compared to Group R (188 ± 6.8 ml), though the difference was not statistically significant ($p=0.608$). This trend is consistent with

previous reports suggesting that dexmedetomidine may reduce surgical bleeding through attenuation of the stress response and modulation of sympathetic-mediated vasoconstriction. Giovannitti et al. noted that alpha-2 agonists reduce blood loss in various surgical settings, potentially through their effects on vascular tone and coagulation-related stress mediators [15]. However, both groups maintained blood loss within clinically acceptable parameters without requiring intraoperative transfusion.

Recovery profile at 10 minutes after surgery and surgeon satisfaction were comparable between the groups, with a numerically higher proportion of good recovery in Group R (60% vs 52%). This trend, while not statistically significant, aligns with the well-established rapid-recovery pharmacokinetics of remifentanyl, which allows for earlier emergence from anaesthesia and facilitates timely postoperative neurological evaluation, a critical consideration in neurosurgical patients. Egan previously characterised remifentanyl's rapid clearance as a key advantage in settings requiring prompt postoperative assessment [16]. Kim et al. echoed this, noting faster return to baseline cognitive function following remifentanyl-based anaesthesia in craniotomy patients [12].

Dexmedetomidine, by contrast, produces a more prolonged sedative effect that may delay immediate recovery. Bekker et al. noted that dexmedetomidine's residual sedation can persist for a period following infusion discontinuation, which may limit its utility in procedures requiring rapid neurological assessment upon emergence [14]. Nevertheless, dexmedetomidine's smoother emergence profile may carry advantages in certain patient subgroups,

particularly those at risk for agitation or haemodynamic instability on awakening.

The study also corroborates the overall safety of both regimens in the context of controlled hypotensive anaesthesia for supratentorial craniotomy. Both groups maintained haemodynamically acceptable MAP values throughout surgery, without evidence of cerebral ischaemia or significant end-organ compromise. The principle of CHA, as outlined by Degoute, requires that blood pressure reduction be achieved without excessive compromise of cerebral perfusion pressure^[17]. The findings of this study indicate that both regimens fulfil this criterion, with remifentanyl offering a more pronounced and sustained hypotensive effect within the safe operative window.

The present findings contribute meaningful comparative evidence to the limited body of literature directly contrasting these two agents in the supratentorial craniotomy setting. The results support remifentanyl as the preferred adjuvant for achieving controlled hypotension during neurosurgery based on its superior intraoperative haemodynamic control, while dexmedetomidine remains a viable alternative associated with slightly lower blood loss and potentially smoother recovery characteristics.

CONCLUSION

Both dexmedetomidine and remifentanyl are effective adjuncts for inducing controlled hypotension during supratentorial craniotomy. However, remifentanyl provides superior intraoperative haemodynamic stability, reduced blood loss and improved recovery characteristics, making it a more favourable option for neurosurgical anaesthesia where rapid titration and early neurological assessment are essential.

CONFLICTS OF INTEREST

None.

ETHICAL APPROVAL

This study approved by the institutional ethical review committee of Bangladesh Medical University (BMU).

REFERENCES

1. RAjmoHAn TR, Sunil SK, DiShAD K, PRAnAy TK, TANISHA G, AMER MM. Comparison between Sevoflurane and Isoflurane for Controlled Hypotensive Anaesthesia in Patients undergoing Craniotomy for Supratentorial Intracranial Surgery: A Randomised Single-blinded Study. *Journal of Clinical & Diagnostic Research*. 2022 Jul 1;16(7).
2. Zhang L, Yu Y, Xue J, Lei W, Huang Y, Li Y, Sun J. Effect of deliberate hypotension on regional cerebral oxygen saturation during functional endoscopic sinus surgery: a randomized controlled trial. *Frontiers in Surgery*. 2021 Sep 8; 8:681471.
3. Petkar S, Chakole V, Nisal R, Priya V. Cerebral perfusion unveiled: A comprehensive review of blood pressure management in neurosurgical and endovascular aneurysm interventions. *Cureus*. 2024 Feb 5;16(2).
4. Suranadi IW, Adistaya AA, Jeanne B. Anaesthesia Considerations in Patients with Heart Failure who will Undergo Glioblastoma Tumor Removal Surgery. *Jurnal Neuroanestesi Indonesia*. 2025 Jun 21;14(2):103-7.
5. Kumoro MA, Prihatno MR, Kartnofan AP. Dexmedetomidine as Neuroanaesthesia Management in Patient with Meningioma Craniotomy. *Jurnal Neuroanestesi Indonesia*. 2024 Jun 27;13(2):93-8.
6. Weerink M. Pharmacokinetic and pharmacodynamic properties of dexmedetomidine and its interaction with remifentanyl.
7. Saber HF, Rahman AI. Remifentanyl versus Nitroglycerin for Controlled Hypotensive Anaesthesia During Primary Open Rhinoplasty: A Comparative Study. *Al-Rafidain Journal of Medical Sciences (ISSN 2789-3219)*. 2024 Dec 29;7(2):197-201.
8. Sarri BM, Guimarães PC, das Neves VG, Borges YN, Sobral CA, Ferruccio CA, Caetano L, Marins JF, de Barros MA. Utilização de remifentanyl em antas-brasileiras (*Tapirus terrestris*) submetidas à anaestesia geral. *Pubvet*. 2025 May 29;19(05): e1779-.
9. Hu Y, Zhou H, Zhang H, Sui Y, Zhang Z, Zou Y, Li K, Zhao Y, Xie J, Zhang L. The neuroprotective effect of dexmedetomidine and its mechanism. *Frontiers in pharmacology*. 2022 Sep 20; 13:965661.
10. Zorrilla-Vaca A, Healy R, Grant MC, et al. Dexmedetomidine in anaesthesia: a meta-analysis of controlled trials. *Anaesthesia & Analgesia*. 2018;126(4):1014-1026.
11. Engelhardt T, et al. Use of remifentanyl for neurosurgical anaesthesia: a review of its efficacy and safety. *Journal of Clinical Anaesthesia*. 2009;21(6):439-445.
12. Kim S, Lee Y, Park G. Postoperative hyperalgesia associated with remifentanyl anaesthesia. *Anaesthesia & Analgesia*. 2018;127(6):1459-1465.
13. Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. *Drugs*. 2000 Feb;59(2):263-8.
14. Bekker A, Sturaitis MK, Liu J. Dexmedetomidine: an overview of its use in anaesthesiology and intensive care. *British Journal of Anaesthesia*. 2013;111(2):131-137.
15. Giovannitti Jr JA, Thoms SM, Crawford JJ. Alpha-2 adrenergic receptor agonists: a review of current clinical applications. *Anaesthesia progress*. 2015 Jan 1;62(1):31-8.
16. Egan TD. Remifentanyl: pharmacology and clinical uses. *Anaesthesia & Analgesia*. 1995;81(1):111-118.
17. Degoute CS. Controlled hypotension: a review of anaesthetic agents. *European Journal of Anaesthesiology*. 2007;24(6):455-467.