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Effects of anesthesia on cerebral oxygen saturation and prevention of brain injury during carotid endarterectomy

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Abstract

Background This study aimed to investigate the effects of general intravenous anesthesia and combined inhalation anesthesia on regional saturation of oxygen (rSO_2) and cerebral hemodynamics during carotid endarterectomy (CEA). Optimizing intraoperative brain protection strategies has become a key focus in CEA research.

Methods Fifty-four patients (43 males, 11 females, aged 44–80) undergoing unilateral CEA were randomly assigned to Group IVA (intravenous anesthesia) or Group CIA (combined inhalation anesthesia), with 27 patients each. Group IVA was maintained with propofol and remifentanil, while Group CIA used sevoflurane, propofol, and remifentanil, with sevoflurane stopped after carotid exposure. Hemodynamics were controlled at various stages: ±10% before clamping, + 20% during clamping (metaraminol), and 0 to -10% after exposure. HR, MAP, and rSO_2 were recorded at T0 (pre-induction), T1 (pre-clamping), T2 (post-clamping), T3 (5 min post-clamping), T4 (10 min post-clamping), T5 (15 min post-clamping), and T6 (15 min post-reperfusion). Blood samples were taken at T1, T6, and T7 (24 h post-surgery) for blood gas and S100-β analysis.

Results No significant differences in rSO_2 were observed at T0 and T6 ($P > 0.05$). However, Group CIA had significantly higher $rScO_2$ at T1, T2, T3, T4, and T5 ($P < 0.05$). From T2 to T5, rSO_2 increased in both groups ($P < 0.05$). MAP and HR showed no significant differences ($P > 0.05$). ΔrSO_2 increased more in Group CIA ($P < 0.05$). At T6, S100-β protein was higher in Group IVA ($P = 0.016$), and pH differed significantly at T1 ($P = 0.009$). No other significant differences were observed.

Conclusion Both intravenous and combined inhalation anesthesia may reduce rSO_2 decline during temporary clamping in CEA. Combined inhalation anesthesia showed a trend toward higher rSO_2 levels, potentially leading to better outcomes, but further studies are needed to confirm these findings.

Retrospectively registered clinical trial number ISRCTN17014575; Registration Date: 2024/6/10.

Keywords Carotid endarterectomy, Regional saturation of oxygen, Sevoflurane, Propofol

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Introduction

Carotid endarterectomy (CEA) is a proven intervention for reducing the risk of ipsilateral ischemic stroke, particularly in symptomatic patients and in select asymptomatic patients with high-risk carotid stenosis features [1]. However, during the procedure, temporary occlusion of the carotid artery may lead to inadequate cerebral perfusion, resulting in a 3% risk of perioperative cerebral infarction [2]. This risk is largely caused by inadequate cerebral perfusion during the temporary carotid artery occlusion [3, 4]. Therefore, optimizing intraoperative brain protection strategies has become a key focus in CEA research in recent years [5]. In CEA surgery, sevoflurane and propofol are commonly used general anesthetics. Propofol, as an intravenous anesthetic, is widely recommended for neurosurgical procedures due to its neuroprotective effects [6]. Studies have shown that propofol may help improve patient outcomes by reducing oxidative stress-induced damage, although these effects have primarily been observed in cases of mild ischemic injury [7, 8]. Meanwhile, sevoflurane, an inhalation anesthetic, at 1.0-1.5 minimum alveolar concentration (MAC), can protect endothelial cells from ischemia/reperfusion injury, maintain cerebral oxygen supply-demand balance, and reduce cerebral metabolic rate (CMRO2), potentially offering brain protection [9, 10]. The complementary mechanisms suggest a potential synergistic effect when the two anesthetics are combined, particularly during the critical periods of carotid artery clamping.

Additionally, near-infrared spectroscopy (NIRS) has gained attention as a superior method for monitoring regional cerebral oxygen saturation (rSO_2) and assessing the likelihood of cerebral ischemia [3, 4]. NIRS offers advantages such as convenience, rapidity, non-invasiveness, and continuous monitoring, enabling timely assessment of cerebral blood supply, vascular injury, and dynamic changes in cerebral tissue oxygen metabolism. Therefore, NIRS can serve as an "early warning" for cerebral hypoxia, assisting anesthesiologists in making timely and effective decisions [11]. Regional saturation of oxygen (rSO_2) reflects the balance of oxygen supply and demand in brain tissue, and is related to factors such as arterial oxygen saturation, hemoglobin, cerebral blood flow (CBF), and cerebral metabolic rate (CMRO2). Therefore, monitoring rSO_2 is a non-invasive and effective method for observing changes in cerebral blood flow during the induction of general anesthesia [12] and predicting the occurrence of cerebral ischemia.

The objective of this study is to compare the effects of intravenous anesthesia and combined sevoflurane anesthesia on rSO_2 levels during CEA to evaluate differences in brain protection between these two anesthetic approaches, thereby providing a basis for clinical anesthesia selection. This study hypothesizes that combined sevoflurane and propofol anesthesia will offer superior protection against ischemic brain injury compared to intravenous anesthesia alone, based on the complementary mechanisms of these anesthetics in maintaining cerebral oxygenation and reducing oxidative stress.

Methods

Study design

This prospective randomized controlled trial aims to compare the effects of intravenous anesthesia (Group IVA) and combined sevoflurane anesthesia (Group CIA) on cerebral oxygenation during CEA. The study will focus on comparing the impact on rSO_2, particularly during carotid artery clamping. The hypothesis is that combined sevoflurane anesthesia will provide better brain protection by improving cerebral oxygenation. The primary comparison will assess the trends of rSO_2 changes at different time points between the two groups.

General Information

Fifty-four patients (43 males, 11 females; aged 44 to 80 years) undergoing unilateral CEA surgery in the First Affiliated Hospital of Xinjiang Medical University were enrolled as participants. They were randomly assigned, using a random number table method, to two groups, A and B, with 27 patients in each group. The study was approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University. The final version of the experimental protocol, informed consent form, researcher manual, and Clinical Trial Observation Form (CRF) were developed and revised in accordance with the guidelines of the Ethics Committee.

Inclusion Criteria: Patients classified as grade II or III according to the criteria of the American Society of Anesthesiologists (ASA); no restrictions on age or sex; unilateral carotid artery stenosis indicated by transcranial angiography (≥ 70% stenosis in asymptomatic patients or ≥ 50% stenosis in symptomatic patients); capable of autonomous behavior and voluntarily signing informed consent.

Exclusion Criteria: Acute phase of cerebrovascular disease; carotid artery occlusion; non-visualization of distal carotid artery stenosis; persistent neurological deficits; long-term use of sedatives or antidepressants; systemic consumptive diseases; severe arrhythmias; myocardial infarction, heart failure, or poorly controlled severe hypertension; severe diseases of the respiratory system.

Anesthesia methods

Induction of anesthesia

Anesthesia induction was performed by intravenous injection of midazolam at 0.05–0.1 mg/kg, etomidate at 0.1–0.3 mg/kg, rocuronium bromide at 0.6 mg/kg,

and sufentanil citrate at 1 μg/kg. After successful tracheal intubation, adjustments were made to the oxygen flow rate, respiratory ratio, tidal volume, and ventilation frequency.

Maintenance of anesthesia

Group IVA: Propofol at 4–6 $mg/kg^{-1}h^{-1}$ and remifentanil at 0.1–0.3 $\mu g/kg^{-1}min^{-1}$ were pumped continuously until the end of the surgery. Group CIA: Continuous inhalation of sevoflurane at 1 MAC and continuous pumping of propofol at 2–4 $mg/kg^{-1}h^{-1}$ and remifentanil 0.1–0.2 $\mu g/kg^{-1}min^{-1}$ were used. Sevoflurane inhalation was stopped after carotid artery exposure and was replaced by continuous infusion of propofol at 4–6 $mg/kg^{-1}h^{-1}$ and remifentanil at 0.1–0.3 $\mu g/kg^{-1}min^{-1}$, until the end of surgery. Both groups received intermittent intravenous injections of rocuronium bromide at 0.15 mg/kg. The anesthetic drugs and respiratory parameters were adjusted intraoperatively to maintain the end-tidal carbon dioxide at 35–40 mmHg, BIS value at 40–60, and nasopharyngeal temperature at 36–37 °C using automatic warming blankets. Intraoperative fluid replacement followed the 4-2-1 rule to maintain blood volume, with efforts made to maintain Hct around 30%.

Intraoperative hemodynamic management

Adjustments were made in correspondence with different stages of CEA surgery. (1) From the start of surgery to clamping of the carotid artery (common, internal, and external carotid arteries), the hemodynamic parameters of the patients were maintained within a ± 10% fluctuation range of the baseline values. (2) During temporary clamping of the carotid artery (common, external, and internal carotid arteries) to block blood flow, metaraminol (Aramine, Akorn Pharmaceuticals) was pumped intravenously to maintain the hemodynamic parameters within a fluctuation range of + 20% of the baseline values. (3) After carotid artery exposure, the hemodynamic parameters were maintained within a fluctuation range of 0 to -10% of the baseline values.

Cerebral oxygen saturation and hemodynamics record

To ensure the accuracy of cerebral oxygen saturation monitoring using the INVOS 5100 C cerebral oximeter (Somanetics, Troy, MI, USA), the sensor needs to be securely fixed on the patient's forehead. Since the surgical area is located on one side of the neck, signal instability or interference may occur during the procedure. Therefore, it is essential to optimize the placement of the device and properly secure the sensor to prevent detachment or displacement, ensuring stable and accurate monitoring data.

On this basis, heart rate (HR), mean arterial pressure (MAP), and regional cerebral oxygen saturation (rSO_2) were recorded at various time points to compare trends over time, including 5 min before anesthesia induction (T0), 5 min before carotid artery clamping (T1), immediately after clamping (T2), 5 min after clamping (T3), 10 min after clamping (T4), 15 min after clamping (T5), and 15 min after restoration of carotid artery blood flow (T6). All data were obtained through continuous monitoring, and 1-minute averages were extracted as research data. Additionally, arterial and venous blood samples were collected at T1, T6, and 24 h after surgery (T7) for arterial blood gas analysis and S100-β protein monitoring.

Statistical methods

SPSS 21.0 statistical software was used for analysis. Measurement data are expressed as mean ± standard deviation. Intra-group comparisons were performed using paired t-tests, and inter-group comparisons were made using repeated measures ANOVA. Count data were compared using chi-square tests. For the repeated measures ANOVA, the assumptions of sphericity and normality were tested, and the variances of the differences between the groups of interest were equal, satisfying the assumption of sphericity, and the variable was normally distributed at all levels. A p-value < 0.05 was considered statistically significant.

Results

No statistically significant differences were observed between the two patient groups in terms of age, sex, comorbidities, arrhythmias, and intake and output volumes (all $P > 0.05$, Table 1). The arrhythmias refer to newly observed or transient events during the study period.

No statistically significant differences in rSO_2 on the operated side were found between the two groups at T0 and T6 ($P > 0.05$). However, at T1, T2, T3, T4, and T5, the rSO_2 of Group CIA patients was significantly higher than that of Group IVA patients ($P < 0.05$). Compared with the T2 values, the rSO_2 values at the T3, T4, and T5 time points were markedly increased ($P < 0.05$). Blood pressure (MAP) and heart rate (HR) changes from T0 to T6 were consistent between the two groups, with no significant differences ($P > 0.05$). Although MAP was maintained within ± 20% of baseline, transient increases were observed during carotid artery clamping, likely due to strategies for maintaining cerebral perfusion. These fluctuations showed no significant differences between groups, indicating effective and consistent blood pressure control in both groups. The trends of changes in rSO_2, HR, and MAP are shown in Figs. 1, 2, 3 and 4; Table 2.

Comparison of the differences in rSO_2 (ΔrSO_2) at T1, T2, and T5 with the baseline (T0) values showed a

Table 1 General characteristics and Intraoperative Variables of Patients

	Group IVA	Group CIA	*P* value
Sex (male/female) (%)	81/19	78/22	0.735
Age (years)	61(58,69)	66(59,72)	0.447
Comorbidities			
Cerebral infarction (yes/no) (%)	48/52	41/59	0.584
Hypertension (Yes/No) (%)	74/26	56/44	0.154
Diabetes (Yes/No) (%)	41/59	37/63	0.780
Coronary heart disease (yes/no) (%)	30/70	7/20	0.761
Coronary stent (yes/no) (%)	7/93	19/81	0.224
Arrhythmias (yes/no) (%)	4/96	7/93	0.552
Arterial occlusion time (min, x ± s)	31 ± 7	27 ± 7	0.013

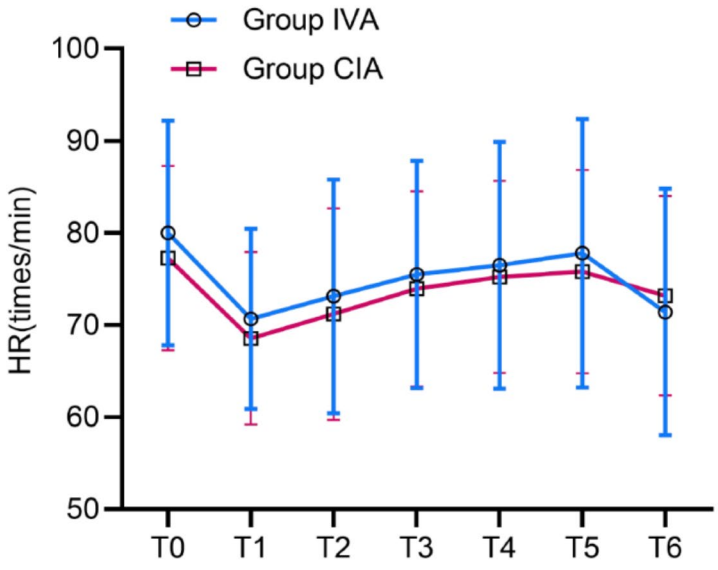

Fig. 1 Comparison of changes in HR between two groups at different time points

significantly greater increase in rSO_2 in Group CIA ($P < 0.05$, Table 3).

There was a difference in S100-β protein levels between the two groups at T6 (t = 2.491, $p = 0.016$). At T1, a highly significant ($P < 0.01$) difference in the pH was observed (t = 7.274, $P = 0.009$). No significant differences in the related indicators were found at the remaining time periods (Table 4; Fig. 5).

Follow-up after surgery: One patient in Group IVA developed contralateral large-area cerebral infarction after surgery, with no improvement after treatment. The possibility of contralateral carotid artery thrombus shedding to cause cerebral infarction was considered. Another patient in Group IVA experienced mild cerebral infarction on the operated side after surgery, manifested as grade 3 muscle strength on the contralateral limb, with no permanent neurological damage after treatment. No abnormalities were observed in Group CIA, and the prognosis of all patients was good. Although we were unable to fully collect follow-up data at 3, 6, 12, and 24 months post-surgery due to the impact of the COVID-19 pandemic, the available follow-up data showed no specific adverse reactions or recurrence in either group at these time points.

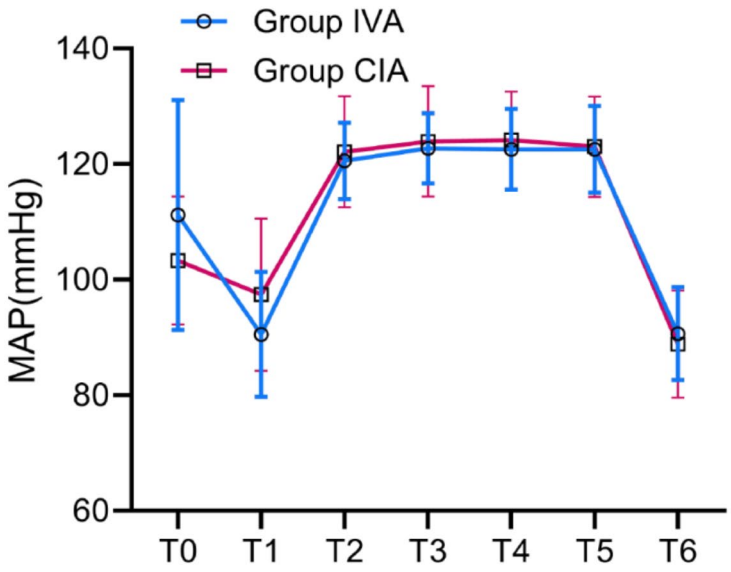

Fig. 2 Comparison of changes in MAP between two groups at different time points

Discussion

The characteristic feature of cerebral blood supply is that even if one or two branches of the nutrient vessels are impaired and the cerebral perfusion pressure varies within a specific range, cerebral autoregulation (CA) can still maintain a constant supply of blood to the brain, preventing hypoperfusion or hyperperfusion of the cerebral tissue [3]. The measurement of cerebral blood flow (CBF) during CEA is crucial [13]. While the velocity of CBF can be measured using intraoperative transcranial Doppler (TCD), it is challenging to do so in neurosurgical operating rooms. Therefore, NIRS monitoring of regional cerebral oxygen saturation is an attractive option. It is a non-invasive method for the evaluation of the balance between cerebral oxygen supply and demand. The near-infrared irradiation shows a high degree of penetration through the scalp and skull to reach deep brain tissue, allowing continuous measurement of the oxygen saturation in arterial blood, venous blood, the anterior cerebral artery, frontal lobe tissue, and other tissue layers. When determining factors (arterial oxygen saturation, hemoglobin) remain constant, fluctuations in rSO_2 are attributed to changes in CBF [14]. Therefore, by continuously monitoring the changes in rSO_2 (ΔrSO_2), it is possible to detect and prevent cerebral ischemia [15]. Studies have reported that rSO_2 values showing a > 20% decrease from baseline are associated with a sensitivity of 80% and specificity of 82% for the occurrence of neurological disease [16]. NIRS can identify the presence of cerebral ischemia in CEA patients during carotid artery clamping, with a sensitivity of 80% and specificity of 94%, 6.5 min faster than somatosensory-evoked potentials [17]. In this study, rSO_2 was found to increase rapidly with the onset of anesthesia until temporary occlusion of the carotid artery. The rSO_2 remained higher than baseline levels at the end of surgery. This is due to an increase in FiO_2 after tracheal intubation, leading to increased oxygen supply, changes in MAP during surgery, and decreased CBF caused by the general anesthetics. A previous study of healthy volunteers showed a similar pattern of rSO_2 changes to those observed here [18].

The effects of sevoflurane and propofol on CBF are different. Sevoflurane reduces the CBF at concentrations < 1.5 MAC but increases CBF at concentrations > 1.5 MAC, believed to be due to the vasodilatory effect of sevoflurane and the CA effect. Studies have shown that the middle cerebral artery blood velocity (MCABV) during propofol anesthesia is lower than that during sevoflurane anesthesia, consistent with the known effects of these drugs on CBF. This indicates that sevoflurane has inherent cerebral vasodilation properties, while propofol anesthesia reduces MCABV by 26% compared to the

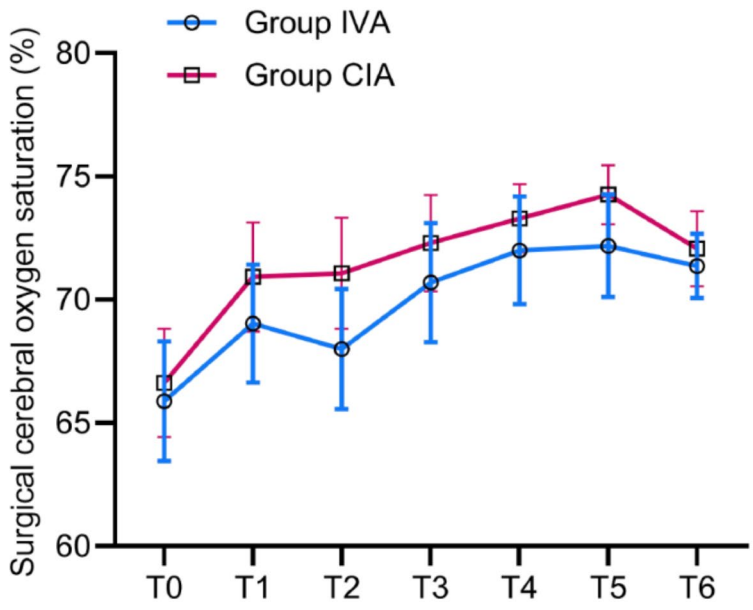

Fig. 3 Comparison of changes in operated side rSO_2 between two groups at different time points

values in conscious patients, with the decrease in CBF matching the decrease in cerebral metabolism caused by propofol [17]. Furthermore, it has been confirmed by positron emission tomography that the reductions in cerebral metabolism induced by propofol and sevoflurane in humans are similar, although propofol was associated with lower CBF and cerebral blood volume than sevoflurane [19]. In the present study, the concentration of sevoflurane in Group CIA was approximately 1 MAC throughout the entire surgery, with an average sevoflurane concentration of 1.36 vol%. The rSO_2 continued to rise relative to the baseline, possibly due to cerebral vasodilation, increased oxygen supply, and decreased cerebral oxygen metabolic rate.

Severe stenosis of the internal carotid artery can induce blood-brain barrier damage, impairing the autoregulatory functions of vasodilation or vasoconstriction of vessels on the surgical side. Park et al. [18] reported that after switching from sevoflurane to propofol in CEA patients, the pressure at the clamped end of the surgical carotid artery increased, which they attributed to the vasodilatory effect of sevoflurane and the vasoconstrictive effect of propofol. In the present study, the ΔrSO_2 and maximum increase in Group CIA were significantly higher than those in Group IVA, possibly because the decrease in CBF with sevoflurane at concentrations < 1.5 MAC was greater than that with propofol. Interestingly, there was no difference in ΔrSO_2 between the two groups on the contralateral side during the perioperative period, while a significant difference was observed on the surgical side. This is related to differences in the drug response between the surgical and contralateral sides. We believe that the combined inhalation anesthesia used in Group CIA improved the regulatory capacity of the surgical side region. Kuzkov et al. [20]. concluded that combined inhalation anesthesia was preferable for maintaining anesthesia during CEA, consistent with our results.

In addition, although the statistical significance ($P < 0.05$) of the 1–3% differences in rSO_2 between Group IVA and Group CIA at various time points indicates a consistent pattern favoring Group CIA, it is essential to assess whether these differences are clinically meaningful in the context of cerebral protection during CEA. rSO_2 values can vary among individuals due to baseline physiological differences, and even small increments in rSO_2 can be crucial in maintaining oxygen levels above ischemic thresholds, reducing the risk of perioperative neurological deficits. Studies have shown that maintaining rSO_2 above certain critical levels reduces the risk of perioperative neurological deficits [21, 22]. Therefore, even a 1–3% improvement in rSO_2 may help maintain safer oxygen levels during ischemia, and repeated small gains

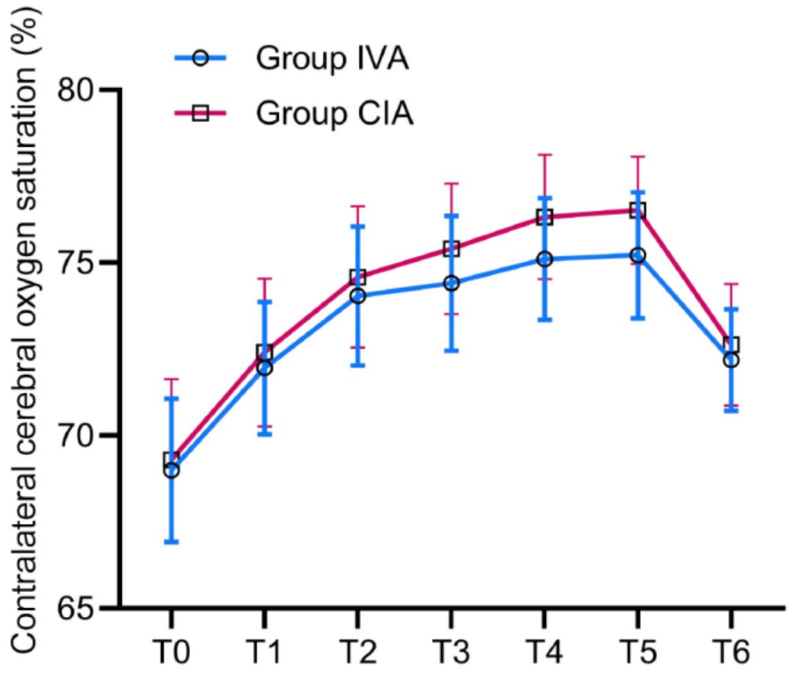

Fig. 4 Comparison of changes in non-operated side rSO_2 between two groups at different time points

Table 2 Comparison of rSO_2 at different time points

	Group	Number of cases	T0	T1	T2	T3	T4	T5	T6
Operated side	Group IVA	27	66 ± 2	69 ± 2	68 ± 2	71 ± 2	72 ± 2	72 ± 2	71 ± 1
	Group CIA	27	67 ± 2	71 ± 2	71 ± 2	72 ± 2	73 ± 1	74 ± 1	72 ± 2
Non-operated side	Group IVA	27	69 ± 2	72 ± 2	74 ± 2	74 ± 2	75 ± 2	75 ± 2	72 ± 1
	Group CIA	27	69 ± 2	72 ± 2	75 ± 2	75 ± 2	76 ± 2	77 ± 2	73 ± 2

Group IVA: Intravenous anesthesia group; Group CIA: Combined inhalation anesthesia group; T0: 5 min before anesthesia induction; T1: 5 min before carotid artery clamping; T2: Immediately after carotid artery clamping; T3: 5 min after carotid artery clamping; T4: 10 min after carotid artery clamping; T5: 15 min after carotid artery clamping; T6: 15 min after restoration of carotid artery blood flow

Table 3 Comparison of operated side ΔrSO_2 between the two groups [%, M(Q1,Q3)]

	T1-ΔrSO_2	T2-ΔrSO_2	T3-ΔrSO_2	T4-ΔrSO_2	T5-ΔrSO_2	T6-ΔrSO_2
Group IVA	3 (2,4)	2 (1,3)	5 (3,6)	6 (5,8)	6 (5,8)	5 (4,7)
Group CIA	4 (3,5)	4 (3,6)	6 (4,7)	6 (5,8)	7 (6,9)	5 (5,7)
Z-value	-3.012	-4.159	-1.640	-1.155	-2.526	-0.053
P-value	0.003	< 0.001	0.101	0.248	0.012	0.958

Table 4 Comparison of related indicators at T1, T6, and T7 between the two groups (x ± s)

Group	S100-β protein (ng/ml)			pH		Blood lactic acid	
	T1	T6	T7	T1	T6	T1	T6
Group IVA	0.10 ± 0.03	0.16 ± 0.04	0.17 ± 0.39	7.41 ± 0.03	7.38 ± 0.04	1.03 ± 0.28	1.11 ± 0.33
Group CIA	0.12 ± 0.04	0.13 ± 0.04	0.10 ± 0.05	7.44 ± 0.03	7.40 ± 0.04	1.09 ± 0.32	1.06 ± 0.26
t	2.090	2.491	0.717	7.274	2.024	0.400	0.480
p	0.154	0.016*	0.401	0.009**	0.161	0.530	0.491

* $p < 0.05$ ** $p < 0.01$

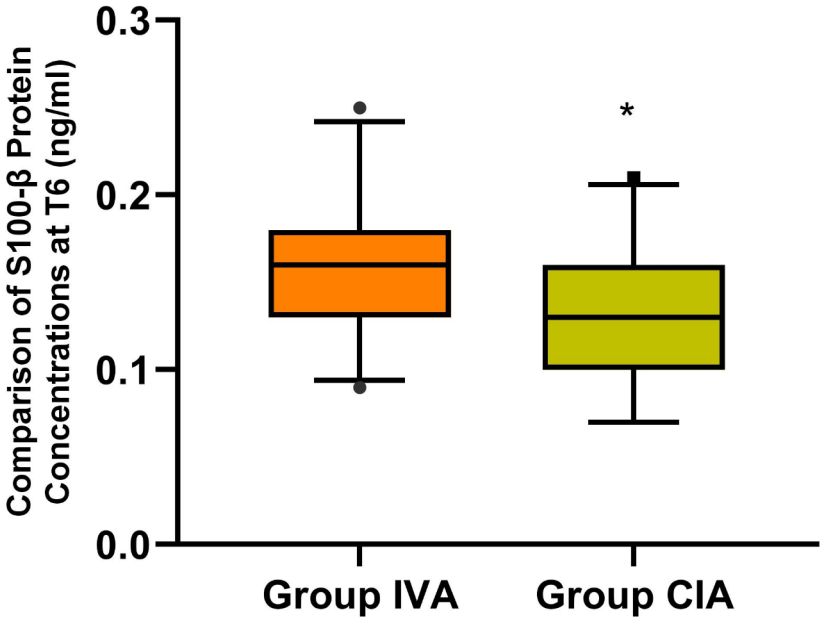

Fig. 5 Comparison of S100-β protein concentrations at T6 between groups A and B, * $p < 0.05$

could potentially cumulatively reduce the duration and severity of cerebral hypoxia, thereby lowering the risk of brain damage.

The formation of oxygen free radicals is believed to be related to neuronal damage caused by ischemia-reperfusion during CEA. Propofol is reported to inhibit the release of excitatory amino acids, reduce intracellular calcium influx, and scavenge oxygen free radicals, thereby reducing the neurotoxicity of excitatory amino acids, protecting cell membranes, and providing preventive and therapeutic effects on brain ischemia-reperfusion injury [23]. One study observed a decreased incidence of neurological dysfunction in animals subjected to cerebral ischemia under sevoflurane anesthesia, with the neuroprotective effect persisting for 8 weeks after ischemia [24]. In the present study [25], it was observed that S100-β protein levels increased to varying degrees during carotid artery clamping. A significant elevation in S100-β protein levels was closely associated with the occurrence of neurological symptoms, suggesting a strong link between its increase and ischemic injury to brain tissue. In this study, all patients maintained stable rSO_2 levels on the non-operated side, with no significant decreases observed. This may explain why the increase in S100-β protein observed was less pronounced compared to other studies, and it may also limit the applicability of our results in scenarios involving more severe decreases in rSO_2 levels. Additionally, although we took measures to reduce extracerebral contamination, it may still have impacted the monitoring results, particularly in interpreting S100-β protein levels, thus introducing potential confounding factors. Future studies should include a broader patient population to better understand the relationship between rSO_2 levels and increases in S100-β protein.

This study found a significant difference in pH at T1 between the two groups ($P = 0.009$), possibly due to differences in anesthesia regimens. The CIA group used sevoflurane in the early stage, which may have affected CO_2 clearance and acid-base balance through metabolic processes or interactions with alveolar ventilation. Differences in ventilatory and metabolic regulation between intravenous and inhalation anesthesia may also contribute to pH changes. Although $PaCO_2$ was not directly monitored, $ETCO_2$ was maintained at 35–40 mmHg through adjustments to ventilation parameters, a range generally considered sufficient to maintain normal $PaCO_2$. While pH changes may affect the oxygen dissociation curve, this typically requires larger fluctuations. The limited pH change observed in this study and the lack of significant rSO_2 differences at T1 (see Table 3) suggest that the pH change had minimal impact on cerebral oxygenation.

This study has several limitations. First, the relatively small sample size may limit the generalizability of the findings, and the lack of formal sample size calculation may affect the accuracy of statistical power, thereby limiting the interpretability of the results. Second, the study was conducted at a single center, and variations in practice patterns across different institutions could affect the

applicability of the results. Additionally, the follow-up period was only 24 h, which does not allow for the assessment of the long-term effects of the anesthesia methods. Another limitation is that some potential confounding factors, such as patients' baseline health status and differences in intraoperative management, were not fully controlled for in this study, which may impact the accuracy of the results. Although BIS values were maintained within 40–60, specific BIS data and fluctuation trends were not recorded or reported, which may limit the assessment of anesthesia depth consistency between groups. Future studies should record and analyze BIS changes to verify the consistency of depth control across different anesthesia regimens. Furthermore, we did not use tools such as Mini-Mental State Examination to assess postoperative delirium, agitation, and cognitive function. Additionally, integrating clinical outcome assessments with rSO_2 monitoring is crucial to further enrich the clinical significance of our study findings. Moreover, the monitoring PaO_2 and glucose levels play an important role in regulating oxidative stress and free radical production [26, 27]. Incorporating the analysis of PaO_2 and glucose values into CEA anesthesia management protocols is essential for optimizing cerebral oxygenation and reducing oxidative stress. We also recognize the importance of PCO2 in influencing cerebral rSO_2. Although PCO2 was not monitored in this study, we understand its potential impact on the results and will consider including this parameter in future study designs. These additions will help to better understand the mechanisms of anesthesia on brain protection and optimize clinical strategies.

Conclusion

In conclusion, this study suggests that both general intravenous anesthesia and combined inhalation anesthesia may help mitigate the decrease in rSO_2 caused by temporary clamping during CEA. Combined inhalation anesthesia showed a tendency toward improved rSO_2 levels compared to intravenous anesthesia, which might be associated with better outcomes. However, further studies are needed to confirm these findings.

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Author contributions
Study concept and design: A.N., Y.L. , S.L.,J.Y.; Analysis and interpretation of data: J.Y., Y.L.; Drafting of the manuscript: Aikebaier.Nuermaimaiti, Shan-shan Li; Critical revision of the manuscript for important intellectual content: J.Y., Y.L.; Statistical analysis: A.N., S.L.; Study supervision: all authors.All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

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Data availability
The data used to support the findings of this study are available from the corresponding author upon request.

Declarations

Ethics approval and consent to participate
The study was approved by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University, and informed consent was obtained from all subjects(Clinical Trial Number: ISRCTN17014575; Registration Date: 2024/6/10, https://www.isrctn.com/ISRCTN17014575?q=ISRCTN17014575&filters=&sort=&offset=1&totalResults=1&page=1&pageSize=10). The final version of the experimental protocol, informed consent form, researcher manual, and Clinical Trial Observation Form (CRF) were developed and revised in accordance with the guidelines of the Ethics Committee.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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