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Original Article

Effect of ultrasound-guided stellate ganglion block on cerebral oxygen metabolism and S100B protein during carotid endarterectomy

Fen Yang[1*], Yulong Du[2,3*], Chen Yuan[1], Li Han[1], Yue Zhao[1], Yang Xie[1], Wenyong Peng[3]

[1]Department of Anesthesiology, The Affiliated Suzhou Hospital of Nanjing Medical University, Suzhou Municipal Hospital, Suzhou 215002, Jiangsu, China; [2]School of Medicine, Shaoxing University, Shaoxing 312000, Zhejiang, China; [3]Department of Anesthesiology, Jinhua Municipal Central Hospital, Jinhua Hospital, Zhejiang University School of Medicine, Jinhua 321000, Zhejiang, China. [*]Equal contributors.

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Abstract: Objective: To investigate the effect of ultrasound-guided stellate ganglion block (SGB) on cerebral oxygen metabolism and serum S100B during carotid endarterectomy (CEA). Methods: Patients who were prospectively enrolled to receive CEA under elective general anesthesia were randomized into an SGB group and a control group (ChiCTR2000033385). Before anesthesia, the SGB group underwent ipsilateral SGB under ultrasound guidance, while the control group did not. Ultrasound-guided right subclavian internal jugular vein catheterization was performed under general anesthesia. Mean arterial pressure (MAP) and heart rate (HR) were monitored at various time points (T0-T4). Arterial and internal jugular venous bulb blood were collected for blood gas analysis, determining jugular venous oxygen saturation ($SjvO_2$), arteriovenous oxygen difference ($AVDO_2$), cerebral oxygen extraction ratio (COER), lactate production rate (LPR), and lactate-oxygen index (LOI). The serum concentration of S100B in the internal jugular venous bulb at each time point was measured. Results: The results revealed significantly lower HR during anesthesia induction and surgery in the SGB group, with more stable MAP and HR during endotracheal intubation and surgery compared to the control group ($P<0.05$). The control group exhibited decreases at T3 and a slight increase at T4. $SjvO_2$ was significantly higher in the SGB group, while $AVDO_2$ and COER gradually decreased over time, but they were significantly higher in the control group ($P<0.05$). LPR and LOI in both groups peaked at T3 and were significantly different between T4 and T2 ($P<0.05$). Serum S100B levels in both groups rose and then decreased at each time point, but they were consistently lower in the SGB group ($P<0.05$). Conclusion: SGB before CEA effectively suppresses the stress response, maintains intraoperative hemodynamic stability, improves brain tissue oxygen supply, and demonstrates a neuroprotective effect.

Keywords: Carotid endarterectomy, stellate ganglion, autonomic nerve block, ultrasound, interventional, oxygen, lactate, S100 calcium binding protein β subunit

Introduction

Stroke has emerged as the leading cause of death and disability in both urban and rural regions of China. Carotid artery stenosis plays a significant role in the onset of ischemic strokes. Carotid endarterectomy (CEA) is a proven method for treating both symptomatic moderate to severe and asymptomatic severe carotid artery stenosis. However, it carries risks of cardiovascular and respiratory complications. Therefore, maintaining systemic circulation and avoiding ischemia and low oxygen damage to critical organs like the heart, brain, and kidneys during the perioperative period is crucial [1, 2]. The risks of death, perioperative stroke, and myocardial injury significantly diminish the postoperative survival rate of patients undergoing CEA. Nonetheless, the methods to reduce the incidence of postoperative stroke are limited, and the absence of definitive prevention and treatment strategies remains a concern. The stellate ganglion, which is a fusion of the first thoracic ganglion and part of the sympathetic nervous system, has been studied for its potential benefits. Previous research indicates that stellate ganglion block (SGB) can enhance the balance of oxygen supply and demand, as well

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as cerebral oxygen metabolism in the brain, subsequently improving early postoperative cognition [3]. Furthermore, SGB has the ability to regulate stress hormones through its effects on the hypothalamic-pituitary-adrenal axis, thereby mitigating the adverse stress responses and reducing the impact of surgical stress on the body [4]. Additionally, SGB has demonstrated potential in reducing early ischemic brain injury and offering brain protection.

There are limited clinical studies specifically exploring its use in CEA for brain protection. Consequently, there is an insufficient body of research to conclusively affirm the neuroprotective effect of SGB in the context of CEA. S100B, a protein secreted by nerve cells, serves as an indicator of neuronal damage and is typically found at low concentrations in the blood of healthy individuals. In cases of severe brain damage, neurons may degenerate and undergo necrosis, leading to significant impairment of the blood-brain barrier and the release of S100B into the peripheral bloodstream. Thus, elevated levels of S100B in the peripheral blood suggest more extensive brain damage and neuronal injury [5]. In this study, we investigated the impact of ultrasound-guided SGB on cerebral oxygen metabolism and serum S100B levels during CEA, aiming to provide a theoretical foundation for clinical practice.

Materials and methods

Study subjects

This study was designed as a prospective, single-center, single-blind, randomized controlled trial. It has received approval from the Ethics Committee of Suzhou Hospital, Nanjing Medical University. Informed consent was obtained from all patients and their family members prior to participation. Participants were assigned to either the observation or control group based on a computer-generated random number table or randomly selected numbers, ensuring the random allocation of participants. The group assignment was not disclosed to participants at any time point during the trial. Both surgical and anesthetic procedures were uniformly managed by the same team of neurosurgeons and anesthesiologists, adhering to a standardized protocol. This approach was intended to guarantee both a consistent surgical process and anesthesia management throughout the study.

Patients undergoing CEA under elective general anesthesia from June 2021 to April 2023 were recruited and allocated into two groups: the SGB (observation) group and the control group, using the random number table. The inclusion criteria were as follows: (1) Aged between 40 and 75 years; (2) Presenting with either symptomatic unilateral carotid stenosis of at least 50% or asymptomatic unilateral carotid stenosis of at least 70%; (3) Classified as American Society of Anesthesiologists (ASA) grade II to III; (4) Undergoing CEA. The exclusion criteria included: (1) A history of acute myocardial infarction within the past three months, angina pectoris, or stroke within the past one to six months; (2) A previous history of carotid stent placement or CEA on one side; (3) Coagulopathy; (4) Psychological illness, central nervous system disease, or infectious disease; (5) Drug allergy; (6) Severe organ insufficiency, including heart, liver, or kidney failure; (7) Inability to achieve satisfactory blood pressure control within 5 minutes; (8) Surgical bleeding exceeding 800 mL; (9) Issues related to internal carotid artery stump pressure or reflux pressure, or the need for transfer due to other conditions.

Sample size estimation

Using the sample size calculation method of comparing two groups, the formula for calculating the sample size is as follows:

$$n_1 = n_2 = 2pq(Z_\alpha+Z_\beta)^2/(P_1-P_2)$$

(1) n represents the sample size of each group. (2) Generally, α is set at 0.05, meaning that the probability of making a type 1 error is 0.05. When the Z-value is two-sided, $Z_\alpha = 1.96$. (3) 1-β represents the detection efficiency, and 0.9 is generally selected, which means that when the two overall groups have a difference, α can find the difference according to the prescribed test level. When 1-β = 0.9, $Z_\beta = 1.28$. (4) P_1 represents the prevalence of the observation group, and P_2 represents the prevalence of the control group. P represents the mean of P_1 and P_2, and q represents the mean of 1-P_1 and 1-P_2.

For this study, α = 0.05, $Z_\alpha = 1.96$, 1-β = 0.9, and $Z_\beta = 1.28$, as well as $P_1 = 0.20$ and $P_2 = 0.40$ expected for the overall complication rate in the SGB group and the control group, so $n_1 = n_2 = 41$, i.e., a sample size of 82 patients.

Methods of SGB and subclavian vein catheterization

The patient was positioned supine with the side undergoing the SGB corresponding to the side of the CEA. The head was tilted 45 degrees to the contralateral side and slightly backward. A linear array ultrasound probe, utilizing an in-plane technique with an adjusted frequency of 15 MHz, was employed. The probe was positioned at the level of the sixth cervical vertebra's transverse process, with its long axis parallel to the plane of the cricoid cartilage. The probe was then slid laterally along the neck and adjusted to clearly display the transverse process of the sixth cervical vertebra, the carotid artery, the jugular vein, and the longus colli muscle. An In-plane injection was administered from a lateral approach traversing laterally from the carotid sheath to the fascia of the longus colli muscle, with delivering 4 mL of the local anesthetic mixture containing 0.25% ropivacaine and 1% lidocaine. The onset of Horner's syndrome (characterized by miosis, ptosis, enophthalmos, conjunctival congestion, facial flushing, and an increase in facial skin temperature - indicated by more than three positive signs) within 15 minutes of the injection served as an indication of a successful SGB. Complications related to the puncture were monitored. The control group did not receive the SGB procedure.

The SGB was initiated 30 minutes after the start of anesthesia induction. Once the anesthesia was adequately established, the right subclavian vein was cannulated under ultrasound guidance following aseptic preparation and draping. The catheter was advanced until resistance was felt, indicating entry into the internal jugular vein. It was then securely fixed in place, and blood samples were collected through the catheter during the procedure. A volume of 5 mL of blood was drawn for monitoring, taking care to minimize the impact of heparin or saline solution on the experimental results. The catheter connected to the transducer was used to monitor the internal jugular vein bulb pressure (JBP).

Key steps of CEA

During the surgery, the carotid artery was carefully and gently dissected to prevent activating the carotid sinus baroreflex, which could result in reflexive bradycardia or cardiac arrest during manipulation. Following the blockade of the carotid artery, decisions regarding blood pressure improvement and shunting were made based on the variations in cerebral blood flow velocity and local cerebral oxygen saturation as indicated by transcranial Doppler ultrasound monitoring. This approach aimed to avert intraoperative cerebral ischemia. Additionally, vasoactive drugs were administered to lower blood pressure, thereby preventing postoperative hyperperfusion syndrome. During operation, the carotid artery was slowly and gently separated to avoid triggering the carotid sinus baroreflex during manipulation, resulting in reflex bradycardia and cardiac arrest. The carotid artery was slowly and gently separated during surgery, and invasive anesthesia with 2% lidocaine 5 ml around the carotid sinus if necessary to avoid triggering the carotid sinus baroreflex with reflex bradycardia and cardiac arrest, but did not cause SGB. The carotid artery was separated slowly and gently during the operation, and 2% lidocaine, 5 ml around the carotid sinus as necessary to avoid triggering the carotid sinus pressure reflex and cardiac arrest during manipulation. After carotid artery blockade, whether to improve blood pressure and diversion was determined according to the changes of cerebral blood flow velocity and local cerebral oxygen saturation monitoring index shown by transcranial Doppler ultrasound to prevent intraoperative cerebral ischemia. Vasoactive drugs were used to reduce blood pressure in order to prevent postoperative hyperperfusion syndrome.

Data collection and monitoring indicators

General patient demographics and clinical data were gathered, encompassing gender, age, height, body mass index, ASA classification, preoperative comorbidities, and details of intraoperative management including dosage of vasoactive medications, duration of blockade, anesthesia duration, surgery duration, and fluid administration volume. Additionally, the incidence of postoperative complications such as hoarseness, hematoma formation, dizziness, phrenic nerve blockade, nausea, and vomiting was meticulously documented.

Hemodynamic values were closely monitored at various stages throughout the procedure. These included heart rate (HR) and mean arte-

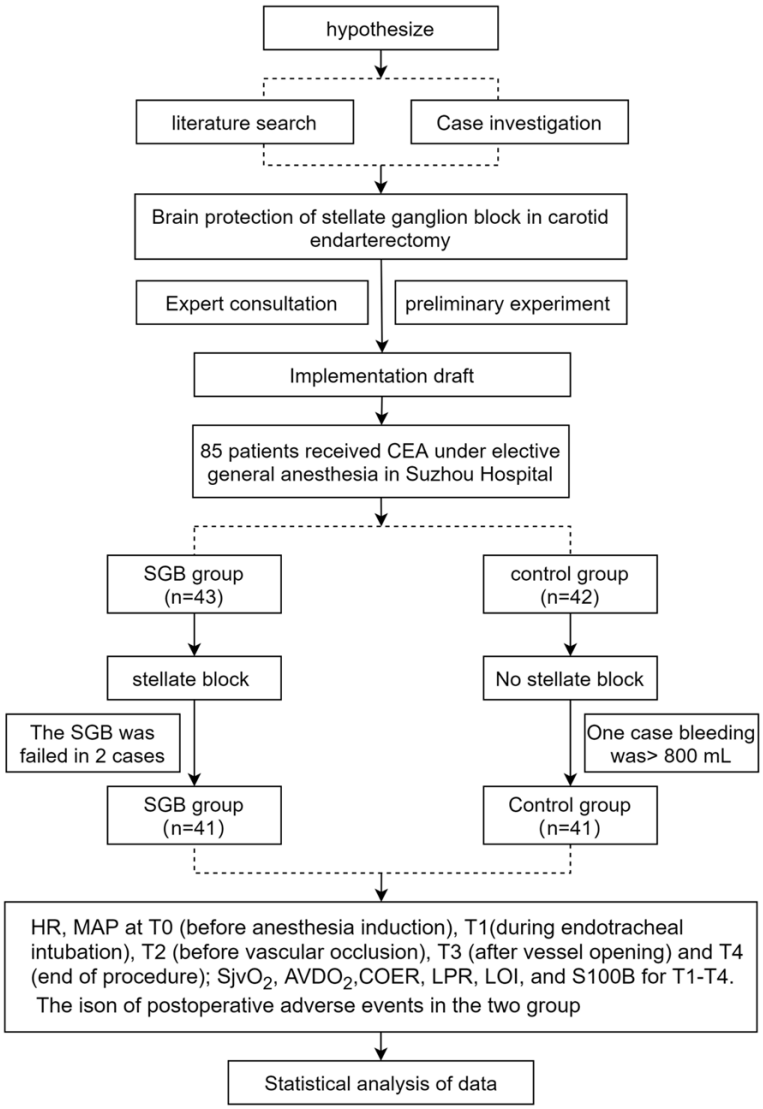

Figure 1. Flow chart. Note: HR: heart rate; JBP: internal jugular vein bulb pressure; MAP: mean arterial pressure; $AVDO_2$: arteriovenous oxygen difference; COER: cerebral oxygen extraction ratio; LPR: lactate production rate; LOI: lactate-oxygen index; $SjvO_2$: jugular venous oxygen saturation; SGB: stellate ganglion block; T1: before endotracheal intubation; T2: before vascular blockade; T3: after vessel opening; T4: at the end of procedure.

rial pressure (MAP) measured at five key time points: T0 (before anesthesia induction), T1 (before endotracheal intubation), T2 (before vascular blockade), T3 (after vessel opening), and T4 (at the end of procedure). Additionally, the JBP was assessed at T1, T2, T3, and T4 to provide comprehensive insights into the patient's hemodynamic status during these critical phases of the operation.

Cerebral oxygen metabolism indices: Blood samples from the radial artery and the internal jugular vein bulb were analyzed for blood gas parameters at time points T1 through T4. The measurements included arterial oxygen pressure, arterial oxygen saturation, arterial lactate levels, arterial hemoglobin concentration, as well as jugular venous oxygen pressure, jugular venous oxygen saturation ($SjvO_2$), lactate levels in the internal jugular vein, and hemoglobin concentration in venous blood. Utilizing the Fick principle, calculations were performed to determine oxygen content of the blood, arteriovenous oxygen difference ($AVDO_2$), cerebral oxygen extraction ratio (COER), lactate production rate (LPR), and lactate-oxygen index (LOI).

Method of serum S100B test

Serum levels of S100B were determined using an enzyme-linked immunosorbent assay kit provided by Shanghai Biyuntian Biotechnology Co., Ltd., China (catalogue number: DTA00C). The normal range for S100B is established between 0.02 to 0.15 μg/L. For this measurement, 5 mL of venous blood was drawn from the cubital vein on the non-infusion side and allowed to clot naturally at room temperature for 20 minutes before being centrifuged at 3000 rpm for another 20 minutes. The supernatant serum was then carefully collected and stored at -80°C until analysis. The assay was conducted in strict accordance with the manufacturer's instructions (see **Figure 1**).

Statistical analysis

Data analysis was conducted using SPSS statistical software, version 26.0. Measured data that followed a normal distribution were pre-

Table 1. Comparison of preoperative general data for patients undergoing carotid endarterectomy ($\bar{x}$ ± sd)

Index	Control group (n = 41)	SGB group (n = 41)	T/χ^2 value	*P* value
Age (year)	61.3±7.8	62.1±8.1	0.456	0.649
Man (n, %)	19 (46.34)	20 (48.78)	0.049	0.825
Height (cm)	167.24±10.31	169.45±11.06	0.936	0.352
BMI (kg/m^2)	24.51±3.11	24.72±3.45	0.289	0.773
ASA classification (n, %)			0.201	0.654
Grade II	16 (39.02)	18 (43.90)		
Grade III	25 (60.98)	23 (56.10)		
Complication (n, %)				
Hypertension	20 (48.78)	18 (43.90)	0.196	0.658
Coronary disease	9 (21.95)	11 (26.83)	0.265	0.607
Diabetes	18 (43.90)	22 (53.66)	0.781	0.377

Note: ASA: American Society of Anesthesiologists; BMI: body mass index; SGB: stellate ganglion block.

Table 2. Comparison of intraoperative data in patients undergoing carotid endarterectomy ($\bar{x}$ ± sd)

Index	Control group (n = 41)	SGB group (n = 41)	T value	*P* value
Ephedrine dosage (mg)	21.63±7.31	20.38±6.98	0.792	0.431
Atropine dosage (mg)	13.69±6.32	12.53±7.01	0.787	0.434
Essellol dosage (mg)	13.36±6.15	15.11±5.97	1.307	0.195
Urapidil dosage (mg)	18.72±4.43	20.38±5.34	1.532	0.129
Glyceryl trinitrate dosage (mg)	15.59±6.92	17.23±7.36	1.039	0.302
Noradrenalin dosage (mg)	221.63±57.28	211.54±54.33	0.818	0.416
Blockade time (min)	32.64±5.81	34.23±6.13	1.205	0.232
Operation time (min)	112.28±12.53	116.83±13.01	1.613	0.111
Anesthesia time (min)	143.67±15.37	145.59±16.11	0.552	0.582
Transfusion volume (mL)	1831.26±361.52	1843.63±355.94	0.156	0.876
Bleeding volume (mL)	713.47±23.67	726.17±29.78	1.229	0.223
Urinary volume (mL)	687.22±223.71	677.54±240.32	0.185	0.854

Note: SGB: stellate ganglion block.

sented as mean ± standard deviation ($\bar{x}$ ± sd). The t-test and repeated measures analysis of variance, with subsequent post hoc Bonferroni correction, were employed for statistical comparisons. Counted data were expressed as frequencies and analyzed using the chi-square (χ^2) test or Fisher's exact test, as appropriate. A *p*-value of less than 0.05 was considered significant.

Results

Comparison of preoperative general data and intraoperative data

During the study period, a total of 85 patients were enrolled. However, two patients were excluded due to an ineffective SGB, and one patient was excluded due to intraoperative bleeding exceeding 800 mL, resulting in 82 patients completing the trial. No patients were transferred during surgery, and there were no cases of dropout during the postoperative follow-up. The SGB and control groups each comprised 41 patients, with no statistically significant differences observed in the preoperative and intraoperative baseline data (as shown in **Tables 1** and **2**).

Comparison of HR, MAP, and JBP at the corresponding time points in the two groups

There were no significant differences in HR and MAP at T0 (before anesthesia induction) between patients in the SGB and control groups. HR and MAP decreased at T1 (before endotracheal intubation), T2 (before vascular blockade), T3 (after vessel opening), and T4 (at

Table 3. Comparison of HR, MAP and JBP between the SGB and control groups

Index	T0	T1	T2	T3	T4	F value	*P* value
HR							
Control	71.48±12.35	81.32±10.79[b]	67.48±9.05[b]	65.67±8.46[b]	64.65±8.87[b]	18.823	0.000
SGB	72.32±11.84	70.42±7.51[a]	57.36±8.19[a,b]	56.55±7.46[a,b]	54.78±7.21[a,b]	38.714	0.000
MAP							
Control	88.88±9.23	82.01±12.27[b]	92.64±11.79[b]	93.66±12.56[b]	94.72±10.54[b]	8.521	0.000
SGB	89.69±10.12	86.57±11.58	84.71±11.45[a]	82.87±10.39[a,b]	81.61±12.55[a,b]	3.281	0.012
JBP							
Control	-	8.61±3.02	8.93±3.43	8.67±2.59	9.03±2.55	0.196	0.898
SGB	-	8.89±2.56	9.07±2.34	8.75±2.21	9.14±2.63	0.214	0.886

Note: Compared with the control group, [a]P<0.05; compared with T0, [b]P<0.05. HR: heart rate; JBP: internal jugular vein bulb pressure; MAP: mean arterial pressure; SGB: stellate ganglion block; T0: before anesthesia induction; T1: before endotracheal intubation; T2: before vascular blockade; T3: after vessel opening; T4: at the end of procedure.

the end of procedure), with significant differences observed between T2, T3, T4, and T0 in the SGB group (P<0.05). In the control group, HR was significantly higher at T1 but decreased at T2, T3, and T4 compared to T0 (P<0.05); MAP was significantly lower at T1 and then increased at T2, T3, and T4 compared to T0 (P<0.05). HR and MAP at T2, T3, and T4 were significantly lower in the SGB group than in the control group (P<0.05). There were no significant changes in internal JBP from T1 to T4, and no statistical differences were found between the two groups (as shown in **Table 3**).

Comparison of $SjvO_2$, $AVDO_2$, COER, LPR, and LOI at the corresponding time points in the two groups

In the SGB group, $SjvO_2$, $AVDO_2$, and COER exhibited minimal changes at T2 and T3; $SjvO_2$ increased at T4, while $AVDO_2$ and COER decreased. In the control group, $SjvO_2$, $AVDO_2$, and COER all decreased at T3 and slightly increased at T4. $SjvO_2$ was significantly higher in the SGB group compared to the control group, whereas $AVDO_2$ and COER were significantly lower (P<0.05). The LPR and LOI peaked at T3 and decreased at T4, with a significant difference observed between T4 and T1 (P<0.05). Both LPR and LOI were significantly higher in the control group compared to the SGB group (P<0.05, as shown in **Table 4**).

Comparison of serum S100B at the corresponding time points in the two groups

Serum S100B levels exhibited a pattern of initially increasing and then decreasing, peaking at T3. Within-group comparisons revealed significant differences between T2, T3, T4, and T1 (P<0.05). Between-group comparisons showed no significant difference at T1; however, S100B levels in the SGB group from T2 to T4 were significantly lower than those in the control group (P<0.05, as indicated in **Table 5**).

Comparison of the incidence of postoperative adverse events in the two groups

The incidence of postoperative adverse events was significantly lower in the SGB group compared to the control group, showing a significant difference between the two groups (P<0.05, **Table 6**).

Discussion

Carotid endarterectomy (CEA) has emerged as the preferred treatment for significant carotid stenosis and for the prevention of ischemic strokes since its first successful performance in 1953 [6]. However, CEA can lead to perioperative neurologic complications, including ischemic brain injuries caused by minimal embolization. Furthermore, the temporary blockade and subsequent re-opening of the carotid artery may disrupt the mechanism of cerebral blood flow autoregulation, potentially leading to cerebral ischemia-reperfusion injury [7]. As a result, 27% to 31% of patients experience a slight neurological decline after undergoing CEA [8]. In response, researchers have conducted a series of studies aimed at reducing brain damage during surgery and enhancing postoperative neural function in patients undergoing CEA. These studies have examined

Table 4. Comparison of $SjvO_2$, $AVDO_2$, COER, LPR, and LOI between the SGB and control groups (n = 41, $\bar{x}$ ± sd)

Index	T1	T2	T3	T4	F value	*P* value
$SjvO_2$ (%)						
Control	51.28±3.65	52.33±3.71	50.15±3.42	54.66±4.61	10.091	0.000
SGB	61.45±7.32[a]	60.22±2.41[a]	59.30±3.06[a]	65.71±2.73[a]	39.140	0.000
$AVDO_2$ (ml/L)						
Control	56.78±4.28	54.21±11.30[b]	51.12±13.94[b]	53.31±11.2	1.746	0.159
SGB	46.59±6.83[a]	44.22±8.11[a]	42.35±9.62[a]	36.21±8.75[a,b]	11.49	0.000
COER (%)						
Control	40.72±4.82	38.08±4.62	37.12±4.34	39.31±5.22	4.349	0.005
SGB	33.46±4.45[a]	32.56±5.27[a]	30.74±4.81[a]	25.32±4.13[a,b]	24.853	0.000
LPR (%)						
Control	7.33±1.23	7.91±3.22	8.02±3.51	6.27±1.79[b]	3.841	0.011
SGB	3.37±1.46[a]	3.19±1.12[a]	4.24±1.57[a]	3.09±1.04[a]	6.511	0.000
LOI						
Control	0.024±0.003	0.025±0.005	0.026±0.004	0.020±0.004[b]	17.192	0.000
SGB	0.014±0.002[a]	0.016±0.003[a]	0.017±0.003[a]	0.015±0.003[a]	8.817	0.000

Note: Compared with the control group, [a]P<0.05; compared with T1, [b]P<0.05. $AVDO_2$: arteriovenous oxygen difference; COER: cerebral oxygen extraction ratio; LPR: lactate production rate; LOI: lactate-oxygen index; $SjvO_2$: jugular venous oxygen saturation; SGB: stellate ganglion block; T1: before endotracheal intubation; T2: before vascular blockade; T3: after vessel opening; T4: at the end of procedure.

Table 5. Comparison of serum S100B at each time point between the SGB and control groups (n = 41, μg/L, $\bar{x}$ ± sd)

Group	T1	T2	T3	T4	F value	*P* value
Control	0.45±0.14	0.85±0.14[b]	0.90±0.15[b]	0.87±0.17[b]	80.421	0.000
SGB	0.43±0.13	0.55±0.11[a,b]	0.62±0.12[a,b]	0.56±0.13[a,b]	17.220	0.000
T value	0.670	7.193	10.001	9.275		
P value	0.505	0.000	0.000	0.000		

Note: Compared with the control group, [a]P<0.05; compared with T1, [b]P<0.05. SGB: stellate ganglion block; T1: before endotracheal intubation; T2: before vascular blockade; T3: after vessel opening; T4: at the end of procedure.

Table 6. Comparison of the incidence of perioperative adverse events between the SGB and control groups (n, %)

Group	Hoarseness	Hematoncus	Dizzy	Diaphragmatic nerve block	Nausea and vomiting	Total
Control	3 (7.32)	5 (12.19)	2 (4.88)	0 (0)	4 (9.76)	14 (31.71)
SGB	1 (2.44)	2 (4.88)	1 (2.44)	1 (2.44)	1 (2.44)	6 (14.63)
χ^2 value	0.263	0.625	0.000	-	0.852	4.232
P value	0.608	0.429	1.000	-	0.359	0.039

Note: SGB: stellate ganglion block.

the impacts of various general anesthesia drugs and anesthetic techniques on brain injury during CEA and postoperative cerebral function [9, 10]. However, these interventions have shown limited effectiveness in improving neurological function post CEA, with a considerable number of patients still experiencing delayed neurocognitive recovery after the procedure. Additionally, some researchers have explored CEA under the guidance of different monitoring methods, such as anesthesia depth and cerebral electrical activity monitoring, and transcranial Doppler ultrasound for cerebral blood flow monitoring. These approaches hold some promise for the prevention of perioperative cerebral ischemia and reperfusion injury [11].

This study aimed to investigate a feasible and effective method that can activate endogenous neuroprotective mechanisms to prevent perioperative cerebral ischemia-reperfusion injury in CEA. It has been hypothesized by some researchers that intracranial blood vessels are regulated by the autonomic nervous system, and it has been confirmed that SGB can enhance intracranial blood flow and improve cerebral circulation by modulating autonomic nerves [12, 13]. Research indicates that SGB can block the conduction of sympathetic ganglionic fibers, preventing intracranial artery spasms, and leading to the dilation of ipsilateral head and neck blood vessels. Furthermore, by inhibiting sympathetic nerve excitation, SGB can lower blood catecholamine levels and reduce neuropeptide Y release, thereby diminishing the stress response, maintaining hemodynamic stability, regulating cytokine release, and modulating immune cell activity to preserve homeostasis in the body environment [14-17]. With the straightforward procedure and low incidence of related complications, ultrasound-guided SGB has been extensively utilized in the realms of pain management and anesthesia. Its application in the treatment of cerebrovascular diseases is often favored due to its lack of increased surgical risk. Consequently, SGB has the theoretical potential to mitigate complications such as brain injury in neurosurgery, making its mechanism particularly relevant for CEA.

In neurosurgery, intense stimuli such as endotracheal intubation, the use of a headrest, and incisions trigger severe stress reactions, leading to sympathetic excitation and an increase in cortisol levels. This, in turn, causes a rise in blood pressure and HR [18]. This study, incorporating SGB before CEA, observed that HR peaked at T0, with no significant alteration at T1, a decline at T2, and stabilization at T3 and T4. Conversely, the control group saw an increase in HR at T1, a decrease from T2 to T4, yet it remained above the initial T0 levels. In comparison, HR in the SGB group exhibited minimal fluctuations at T1, indicating that SGB may mitigate the stress response, resulting in a more stable HR throughout the surgical process. Furthermore, MAP in the SGB group decreased before stabilizing, while in the control group, it initially decreased then increased. The comparative analysis revealed that MAP was steadier and exhibited less variability in the SGB group. Fewer SGB patients experienced hemodynamic fluctuations requiring medicinal intervention compared to the control group, suggesting that preoperative SGB enhances the stability of the circulatory system during surgery.

Possible mechanisms through which SGB contributes to hemodynamic stability include: (1) Suppressing sympathetic excitation, thereby reducing the stress response; (2) Decreasing the release of noradrenaline and neuropeptide Y, along with a reduction in sympathetic ganglia activity, which in turn diminishes adrenaline secretion [19]; (3) Modulating the function of the hypothalamic-pituitary-adrenal axis, leading to decreased levels of cortisol, aldosterone, angiotensin, serotonin, and substance P, and an increase in insulin secretion. This helps regulate the endocrine system and mitigate the intraoperative stress response [20]. The findings of this study further indicate that SGB helps prevent fluctuations in HR and MAP, thereby contributing to the maintenance of hemodynamic stability.

Hemodynamic stability is crucial for ensuring adequate cerebral blood flow and a consistent supply of cerebral oxygen; excessively low levels can lead to cerebral ischemia, while excessively high levels might result in intracranial hemorrhage. Beyond maintaining an appropriate depth of anesthesia, it is equally vital to preserve the stability of cerebral blood flow and the equilibrium of cerebral oxygen supply. Hence, during vascular blockade, particular emphasis should be placed on hemodynamic stability. Although the JBP does not directly reflect intracranial pressure, previous studies have indicated a correlation between the two [21]. This study found no significant changes in JBP throughout the surgical procedure, with no statistically significant difference between the two groups. This suggests that SGB has a minimal effect on intracranial pressure. The underlying mechanism might be that SGB facilitates venous return. While SGB may enhance cerebral perfusion, it does not lead to an increase in brain volume. Consequently, there is no significant change in intracranial pressure. Furthermore, the brain's autoregulation of blood pressure may also contribute to the absence of significant alteration in intracranial pressure.

$SjvO_2$, $AVDO_2$, and COER are indicative of the balance of oxygen supply in the brain. A decrease in $SjvO_2$ alongside increases in $AVDO_2$ and COER suggests an inadequate oxygen supply, with $SjvO_2$ levels below 50% signaling insufficient cerebral perfusion [22]. In this study, an increase in $SjvO_2$ was observed at T3 in both groups, potentially due to vasospasm or reduced cerebral perfusion following arterial blockade. Fluctuations in $SjvO_2$ were more marked in the control group compared to the SGB group, with $SjvO_2$ consistently higher at each time point in the SGB group. $AVDO_2$ and COER reached their peak at T2 and were at their lowest at T4, with a general decreasing trend observed throughout the procedure. The reduction at T4 might be attributed to the enhancement of brain blood and oxygen supply post vascular reconstruction. $AVDO_2$ and COER were significantly lower in the SGB group than in the control group, possibly because SGB mitigates intracranial vasospasm, dilates intracranial vessels, increases cerebral blood flow, and thereby improves oxygen delivery to brain tissue. In this study, the $SjvO_2$ was higher in the SGB group compared to the control group, whereas the $AVDO_2$ and COER were lower. These findings suggest that the SGB group experienced an enhanced cerebral oxygen supply, which is advantageous for the surgical process and prognosis of CEA. Lactate serves as an anaerobic metabolite of cells, with its production increasing under conditions of hypoxia due to enhanced anaerobic metabolism in brain cells. Consequently, a higher lactate level is indicative of more severe brain tissue ischemia. By analyzing blood gases from the internal carotid vein bulb and radial artery, the LPR and LOI can be calculated, providing insight into the extent of brain tissue ischemia from an energy metabolism perspective. The results indicated that both LPR and LOI were lower in the SGB group compared to the control group, signifying that SGB indeed enhances cerebral perfusion and oxygen supply to brain tissue, thereby reducing lactate production.

S100B belongs to a category of low-molecular-weight calcium-binding proteins, found predominantly in astrocytes, oligodendrocytes, and Schwann cells. In conditions of ischemia and hypoxia within brain tissue, S100B production and secretion are elevated through an acute glial response. With further disruption of the blood-brain barrier, S100B is able to cross into the bloodstream [23]. Consequently, serum S100B levels can indicate the presence and extent of neural damage, serving as a specific biomarker for evaluating the neuroprotective effects of SGB on brain tissue [24-27]. Sago et al. reported that the release of proinflammatory cytokines interleukin (IL)-8 and IL-6 is heightened during cellular hypoxia [28]. Research conducted by Xiong et al. demonstrated that SGB mitigates the release of IL-8 and IL-6, thereby reducing cellular damage triggered by inflammatory mediators [29]. Davis et al.'s study revealed that SGB can modulate the apoptotic process through the regulation of cytokine secretion [30]. Quanshoubo's research indicated that SGB, in the context of brain hypoxia, can diminish ischemia and reperfusion injury in brain tissue by balancing the levels of endothelin and calcitonin gene-related peptide [31]. This study observed that serum S100B levels were at their lowest at T2, peaked at T3, and declined at T4, with these fluctuations corresponding to the progression of the surgical procedure. The elevation at T3 suggests brain tissue damage due to surgical intervention, whereas the reduction at T4 indicates improved blood supply to the brain tissue. Furthermore, serum S100B levels were consistently lower in the SGB group compared to the control group at all measured time points, indicating that brain tissue damage was comparatively reduced in patients receiving SGB.

This study had several limitations: (1) The sample size was small. Future studies should aim to increase the sample size and incorporate multicenter research to enhance the robustness of the findings. (2) There was no long-term follow-up, such as assessments of cognitive function and vascular events at 3 or 6 months post procedure. It is essential to conduct future studies with long-term follow-up to investigate the enduring effects of SGB. Additionally, the possible adverse effects of SGB warrant careful consideration. First, the injection of local anesthetics into blood vessels can cause sensitization in some patients. Furthermore, the inclusion of hormones or other drugs in local anesthetics during multiple injections may impair the stellate ganglia [32]. Secondly, improper technique may lead to needle-induced damage to neck vessels, resulting in local hematoma, or pneumothorax and hemothorax

due to incorrect needle penetration angles [33, 34]. Hence, greater vigilance is required regarding the complications associated with SGB in clinical settings.

In conclusion, SGB prior to CEA can significantly mitigate the intraoperative stress response, contribute to hemodynamic stability, enhance intraoperative cerebral oxygen metabolism, and lower serum S100B levels. These effects collectively offer a protective impact on brain tissue.

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Disclosure of conflict of interest

None.

Address correspondence to: Yang Xie, Department of Anesthesiology, The Affiliated Suzhou Hospital of Nanjing Medical University, Suzhou Municipal Hospital, No. 26 Daoqian Street, Gusu District, Suzhou 215002, Jiangsu, China. Tel: +86-136-06212680; E-mail: xieydoctor@126.com; Wenyong Peng, Department of Anesthesiology, Jinhua Municipal Central Hospital, Jinhua Hospital, Zhejiang University School of Medicine, No. 365 Renmin East Road, Jinhua 321000, Zhejiang, China. Tel: +86-13566996989; E-mail: jhyypwy@163.com

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