Regional cerebral oxygen saturation and postoperative delirium in endovascular surgery: a prospective cohort study

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Abstract

Background: Delirium is an acute mental disorder and common postoperative complication. Monitoring regional cerebral oxygen saturation (rSO2) in endovascular therapeutic surgery may allow real-time monitoring of cerebral desaturation, avoiding profound cerebral dysfunction, and reducing the incidence of delirium. We sought to examine the incidence of delirium in patients undergoing endovascular surgery.

Methods: This was a clinical cohort trial (registered with http://www.clinicaltrials.gov [NCT02356133]). We monitored the rSO2 of 43 patients undergoing general anesthesia and cerebral endovascular surgery. The occurrence of delirium after surgery was recorded with the Confusion Assessment Method (CAM). Multivariate logistic regression was performed to identify the main predictor of delirium.

Results: rSO2 was significantly different between the delirium and no-delirium groups. The occurrence of delirium was 35% in our cohort, and higher rSO2 desaturation scores were significantly associated with profound delirium (higher CAM score; odds ratio = 1.002; \(P=0.021\)). The maximum declines of systolic blood pressure were 24.86 (21.78–27.93) and 32.98 (28.78–37.19) in the no-delirium and delirium groups, respectively, which were significantly different (\(P=0.002\)) but not closely associated with delirium in multivariate analysis (\(P=0.512\)). Anesthesia, mechanical ventilation duration, and having two vascular risk factors differed significantly between groups but were poorly associated with delirium outcome.

Conclusions: Elevated rSO2 desaturation score was predictive of the occurrence of postoperative delirium following endovascular surgery. Monitoring rSO2 is invaluable for managing controlled hypotension during endovascular surgery and reducing postoperative delirium.

Trial registration: ClinicalTrials.gov, NCT02356133. Registered 1 February 2015. All statistical analysis results submitted August 4, 2018.

Keywords: Delirium, Regional cerebral oxygen saturation, rSO2

Background

Delirium is a complex neuropsychiatric syndrome that has a high prevalence in acute hospitals and is encountered across all healthcare settings. It is associated with adverse outcomes, including comorbidities and mortality. The incidence of postoperative delirium varies from 10% to 55%, and it is more common in patients with vascular dysfunction [1–3]. Previous studies indicated that several factors, such as age, education, ethanol consumption, preoperative cognitive status, and head trauma, are preoperative risk factors for delirium [4–9]. Intraoperative cerebral ischemia and cerebral oxygen desaturation have been proposed as possible mechanisms of postoperative cognitive dysfunction [10–12]. However, other important factors that have not been assessed, such as regional cerebral oxygen saturation (rSO2), hypotension, and hemoglobin, significantly affect cerebral perfusion and oxygen metabolism [13] and may further increase the risk of delirium.
Delirium after neurosurgery is mainly caused by cerebral hypoperfusion and cerebral anoxia [14–17]. A study using rSO2 during carotid revascularization validated rSO2 as a potential reliable marker of hypoperfusion with a sensitivity of 100% and specificity of 90.6% [18]. Another study noted that near-infrared spectroscopy decline during the coating of ruptured aneurysms was strongly associated with vasospasm during the procedure [19]. Monitoring rSO2 can quickly reflect changes in the balance between cerebral oxygen supply and demand. Early detection and management of predisposing risk factors for delirium would lead to improved outcomes for patients.

Endovascular therapy or interventions, also known as intra-arterial therapy, often require strict blood pressure control [20]. Cerebral aneurysm is a common cerebral vascular disease that requires endovascular surgery; long-term controlled hypotension is necessary during the perioperative period to ensure safe completion of the operation, especially in ruptured aneurysms [20]. Controlled hypotension during the surgical procedure reduces bleeding and improves visualization, but it may be related to postoperative cognitive dysfunction when cerebral perfusion is not maintained [21]. Intraoperative hypotension is an infrequent direct cause of cerebral oxygen desaturation and can contribute to injury caused by embolism or surgery, especially during the perioperative period. However, the appropriate blood pressure control regimen that leads to the most favorable clinical cerebral perfusion level in these patients remains inconclusive. Additionally, anesthesiologists have a significant challenge in maintaining the proper balance of rSO2. This requires real-time noninvasive monitoring that can directly reflect the relationship between cerebral oxygen supply and demand and cerebral perfusion.

In this prospective cohort study, our aim was to examine the perioperative risk factors associated with the development of delirium in patients following endovascular surgery. We sought to observe the appropriate range for rSO2 using noninvasive cerebral oxygen saturation monitoring and to assess control of hypotension during endovascular intervention surgeries to reduce the incidence of postoperative delirium. Our hypothesis was that rSO2 would correlate with postoperative delirium after cerebral endovascular surgery.

Methods
Patient characteristics and power calculations
This prospective observational study was approved by our university’s institutional review board (IRB 0810-758). Written informed consent was obtained from all patients participating in the trial. The trial was registered with http://www.clinicalTrials.gov (NCT02356133; date of registration, February 1, 2015). Our methodology followed the international guidelines for observational studies. The trial was conducted and reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) 2010 statement (Fig. 1 and Additional file 1: Table S1). We used G*Power to conduct power analysis. To calculate the required study size, we considered the results of previous studies performed in a similar population. To detect time points in mean rSO2 values recorded during surgery, accepting a two-tailed α error of 5% and β error of 10%, 39 patients were required. Generally, the limiting values for the reference interval are the 0.025 and 0.975 fractiles of the results distribution in the population. In the present study, only high rSO2 desaturation score was likely to be of clinical interest. Therefore, the use of the 0.05 fractile as the high reference limit made the most sense [22, 23]. We recruited 43 prospective consecutive patients with intracranial aneurysms who were scheduled for endovascular surgery with general anesthesia between May 1, 2015, and January 1, 2017, at Xuanwu Hospital. Inclusion criteria were as follows: (1) elective intracranial aneurysm embolization, (2) 35–70 years old, (3) American Society of Anesthesiologists (ASA) physical status score of II–IV, and (4) body mass index (BMI) ranging from 22 to 45 kg·m⁻². Exclusion criteria were as follows: (1) preexisting neuropsychiatric disorders or inability to correctly perform neurocognitive tests on the patient, (2) emergency operation, (3) diagnosis of coma, (4) depression, or (5) cognitive impairment.

Anesthetic technique and protocol
Patients’ demographic characteristics were recorded. After admission to the operating room (OR), patients were administered 8 ml·kg⁻¹ Ringer solution with an intravenous maintenance dose of 4 ml·kg⁻¹·h⁻¹. Patients were monitored with a five-lead electrocardiogram (ECG), invasive arterial pressure measurements, oxygen saturation measured by pulse oximetry (SpO2), and rSO2. Baseline blood pressure, heart rate, and rSO2 were acquired 15 min after admission to the OR. Anesthetic induction was performed using intravenous etomidate (0.2 mg·kg⁻¹), sufentanil (0.3 μg·kg⁻¹), and rocuronium
(0.5 mg·kg\(^{-1}\)). Tracheal intubation was performed for mechanical ventilation when satisfactory muscle relaxation was achieved (approximately 3 min), followed by connection to an anesthesia machine (Datex-Ohmeda Avance S5; GE Healthcare, Chicago, IL, USA) for volume control ventilation with a tidal volume of 8 ml·kg\(^{-1}\), respiratory rate of 12 min\(^{-1}\), inspiration/expiration ratio of 1:2, and end-tidal carbon dioxide pressure (PETCO\(_2\)) of 35–40 mmHg. SpO\(_2\) was maintained at 99–100% during the general anesthesia period. An infusion of 0.06 mg·kg·min\(^{-2}\) rocuronium, 0.3 mg·kg·min\(^{-2}\) propofol, 0.3 \(\mu\)g·kg·min\(^{-2}\) remifentanil, and 0.4–0.6% sevoflurane was administered continuously during surgery. Nimodipine (1–3 ml·h\(^{-1}\)) was administered to control hypotension during the operation.

Perioperative monitoring and data acquisition

The following variables were recorded for all patients: general patient data, including age, sex, weight, height, BMI, preoperative hemoglobin, and ASA physical status; and medical history of hypertension, diabetes mellitus, coronary artery disease, and stroke. Patients who had two systemic vascular comorbidities (including history of hypertension, diabetes mellitus, coronary disease, and/or stroke) were classed as having two risk factors; those with three systemic vascular comorbidities (including history of hypertension, diabetes mellitus, coronary disease, and/or stroke) were classed as having three risk factors. During the operation, we monitored standard noninvasive vital signs, including systolic blood pressure (SBP), diastolic blood pressure (DBP), ECG data, SpO\(_2\), and PETCO\(_2\). In addition, we recorded anesthesia, mechanical ventilation, and operation duration. Finally, rSO\(_2\) indices were continuously recorded in the brain during the operation using a cerebral oximeter (C2030C; CAS Medical Systems Inc., Branford, CT, USA). Briefly, bilateral rSO\(_2\) probes were placed on the patient’s forehead and stabilized. Cerebral oxygen data were recorded bilaterally every minute. The maximum SBP (%) and DBP (%) declines were calculated as follows: \[
\left(\frac{\text{lowest SBP recorded during the operation}}{\text{SBP baseline score}}\right) \times 100. \left(\frac{\text{lowest DBP recorded during the operation}}{\text{DBP baseline score}}\right) \times 100. \]
Baseline SBP and DBP both indicate preoperative levels 15 min after entry into the OR.

rSO\(_2\) desaturation score

To relate intraoperative rSO\(_2\) to clinical outcomes and hemodynamic variables, we used the rSO\(_2\) desaturation score as a measure of the degree of desaturation, where the score was calculated using the baseline and recorded rSO\(_2\) over time [24–26]. The rSO\(_2\) desaturation score is expressed as a product with the units of percentage per minute, as described below. M = measured rSO\(_2\); B = baseline rSO\(_2\). Z is calculated as the measured rSO\(_2\) – threshold rSO\(_2\) according to the following definition: if M < B, then \(Z = M\); if M ≥ B, then \(Z = 0\). The rSO\(_2\) desaturation score for each patient was calculated using the following formula: rSO\(_2\) desaturation score = \((\Sigma Z) \times t\), where \(t\) = total number of minutes from anesthesia induction until exit from the OR [24–26], which accounts for both depth and duration of desaturation below the baseline of each patient. Complete rSO\(_2\) datasets were acquired for 39 patients. For patients with bilateral cerebral rSO\(_2\) sensors, mean rSO\(_2\) was used for analysis.

Postoperative delirium assessment

We used the Confusion Assessment Method (CAM) delirium score to evaluate the degree of delirium [27]. Diagnosis of delirium was assessed using an algorithm based on the CAM [28]. CAM diagnostic criteria included attention dysfunction, confusion, level of consciousness, disorientation, memory loss, perception dysfunction, psychomotor excitement and retardation, volatility, and sleep-wake cycle changes [29]. Rapid CAM diagnosis of delirium requires the following four key delirium characteristics: (1) acute onset and disease fluctuations, (2) inattention, (3) disordered thinking, and (4) changes in the level of consciousness (any state of consciousness other than fully conscious). The diagnosis of delirium requires the presence of (1) and (2), accompanied by (3) or (4) or both [24]. The diagnosis of delirium was performed by three nurses who had undergone training for 1 week to ensure consistency.

Statistical analysis

IBM SPSS Statistics software (version 22.0; IBM, Armonk, NY, USA) was used to analyze the data. The patients were divided into two groups according to the incidence of delirium. The presence or absence of delirium was evaluated as a dichotomous variable. Continuous variables were expressed as mean and 95% confidence interval (95% CI) and were compared using \(t\) tests. Categorical data were expressed as frequency and percentage and were compared using chi-square tests. The distribution of data was evaluated using the Kolmogorov-Smirnov test. Significant variables from the \(t\) and chi-square tests were included in Spearman’s rank correlation analysis. Logistic regression analysis was performed to analyze the association between delirium and predictors correlated with delirium.

Results

Patient demographics and perioperative variability between groups

On the basis of the inclusion and exclusion criteria, 39 patients were analyzed in our study. Four patients were excluded for the following reasons: an underlying dementia diagnosis (\(n = 1\)), coma (\(n = 1\)), and canceled operation (\(n = 1\)). Figure 1 shows the selection procedure for the patients. A total of 38 patients were safely discharged from the hospital after surgery; one patient experienced severe delirium and died on the fifth day after surgery.
Postoperative delirium occurred in 14 of 39 patients (35%). There were no different of age, height, weight, BMI, Hemoglobin, and ASA physical status in delirium group and no delirium group (Table 1). Preoperatively, there were no significant differences between the groups in terms of patient sex or history of hypertension, diabetes mellitus, coronary disease, and stroke. The number of patients with two risk factors was significantly lower in the no-delirium group ($P = 0.010$) (Table 2). There was no significant difference in operation duration ($P = 0.147$) or maximum DBP decline ($P = 0.300$) between groups (Table 3). Mechanical ventilation duration and anesthesia duration were significantly higher in the delirium group (mechanical ventilation duration, 143.85; 100.51–187.18; anesthesia duration, 168.46; 127.06–209.86) than in the no-delirium group (mechanical ventilation duration, 118.65; 105.18–132.13; $P = 0.044$; anesthesia duration, 133.46; 120.13–146.79; $P = 0.023$) (Table 3). The maximum SBP decline was significantly higher in the delirium group (32.98; 28.78–37.19) than in the no-delirium group (24.86; 21.78–27.93; $P = 0.002$) (Table 3). Furthermore, rSO2 desaturation score was significantly higher in the delirium group (1267.44; 866.82–1668.07) than in the no-delirium group (231.95; 55.70–408.21; $P = 0.001$) (Table 3).

**Correlations between delirium and different variables**

The difference in rSO2 desaturation score between the groups was closely associated with delirium ($r = 0.597$; $P = 0.001$) (Table 4). Furthermore, maximum SBP decline was significantly correlated with delirium ($r = 0.465$; $P = 0.003$) (Table 4). The incidence of two systemic vascular risk factors was also correlated with delirium ($r = 0.486$; $P = 0.002$) (Table 4). However, anesthesia duration (in minutes) and mechanical ventilation duration (in minutes) were not correlated with delirium (anesthesia duration, $r = 0.183$; $P = 0.264$; mechanical ventilation duration, $r = 0.114$; $P = 0.489$) (Table 4).

**Logistic regression analysis of the preoperative predictors of delirium**

Logistic regression analysis revealed that rSO2 desaturation score was a significant positive predictor of postoperative delirium (OR = 1.002; $P = 0.021$) (Table 5), suggesting that an increase in rSO2 desaturation was a surrogate for increased cerebral ischemia and insufficient oxygen supply, leading to an increased risk of postoperative delirium. Table 5 summarizes the results of logistic regression analysis between the potential predictors for delirium. There was no significant association between maximum SBP decline and delirium based on multivariate logistic regression (OR = 1.059; $P = 0.512$). There was a very weak association between the incidence of two systemic vascular risk factors and postoperative delirium; however, this did not reach statistical significance (OR = 3.593; $P = 0.361$) (Table 5). To determine the appropriate reference intervals for rSO2 scores for the best monitoring of controlled hypoperfusion, we calculated the 95% prediction interval based on the one-sided high reference limit among healthy individuals. We found that the threshold rSO2 desaturation score was 493.96, indicating that a person with a value above this reference limit had a higher risk of delirium.

**Discussion**

Our results provide support for rSO2 desaturation score as an independent predictor of postoperative delirium following high-risk cerebral endovascular interventions of

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**Table 1** Demographic and preoperative characteristics of the delirium and no delirium groups

| Variable         | Delirium (no) ($n = 25$) | Delirium (yes) ($n = 14$) | $P$ value |
|------------------|--------------------------|---------------------------|-----------|
| Age (years)      | 52.62 (47.72–57.51)      | 54.08 (46.16–62.00)       | 0.731     |
| Height (cm)      | 168.19 (165.42–170.96)   | 164.54 (160.78–168.29)   | 0.114     |
| Weight (kg)      | 73.62 (68.21–79.02)      | 70.31 (66.89–73.73)      | 0.401     |
| BMI (kg m$^{-2}$)| 25.85 (24.48–27.22)      | 25.99 (24.63–27.34)      | 0.892     |
| Hemoglobin (g/L)| 142.04 (136.13–147.95)   | 140.23 (133.75–146.71)   | 0.300     |
| ASA physical status | 2.983 (2.121–3.165)   | 2.891 (2.753–3.521)     | 0.580     |

**Table 2** Patient characteristics and preoperative clinical prediction of delirium

| Variable                      | Delirium (no) ($n = 25$) | Delirium (yes) ($n = 14$) | $P$ value |
|-------------------------------|--------------------------|---------------------------|-----------|
| Male sex, n (%)               | 18 (46.15%)              | 7 (17.95%)                | 0.104     |
| History of hypertension       | 8 (20.51%)               | 6 (15.38%)                | 0.816     |
| History of diabetes mellitus  | 1 (2.56%)                | 4 (10.26%)                | 0.261     |
| History of coronary artery disease | 0 (0)                  | 3 (7.69%)                | 0.255     |
| History of stroke             | 1 (2.56%)                | 3 (7.69%)                | 0.065     |
| Two risk factors              | 1 (2.56%)                | 7 (17.95%)                | 0.010*    |
| Three risk factors            | 1 (2.56%)                | 2 (5.13%)                | 0.254     |

Data are expressed as number (%), analyzed using chi-square test

**Table 3** Postoperative delirium patient characteristics and preoperative clinical prediction of delirium

| Variable                      | Delirium (no) ($n = 25$) | Delirium (yes) ($n = 14$) | $P$ value |
|-------------------------------|--------------------------|---------------------------|-----------|
| Male sex, n (%)               | 18 (46.15%)              | 7 (17.95%)                | 0.104     |
| History of hypertension       | 8 (20.51%)               | 6 (15.38%)                | 0.816     |
| History of diabetes mellitus  | 1 (2.56%)                | 4 (10.26%)                | 0.261     |
| History of coronary artery disease | 0 (0)                  | 3 (7.69%)                | 0.255     |
| History of stroke             | 1 (2.56%)                | 3 (7.69%)                | 0.065     |
| Two risk factors              | 1 (2.56%)                | 7 (17.95%)                | 0.010*    |
| Three risk factors            | 1 (2.56%)                | 2 (5.13%)                | 0.254     |

Data are expressed as mean (95% confidence interval), based on $t$ test and chi-square test for analysis

BM1 Body mass index, ASA American Society of Anesthesiologists

*P < 0.05
Mortality is increased by 11% for every additional 48 h of active delirium, highlighting the requirement for timely detection and treatment. In our study, the incidence of early postoperative delirium was 35%, which is consistent with the findings of previous studies [41, 42] and lower than the incidence of delirium among patients with cancer (49.8%) or trauma ICU patients (38.7%) [15, 43]. We described the incidence of delirium using an algorithm based on the CAM. This tool was preferred because it has been validated previously and is the gold standard for the diagnosis of delirium [44, 45].

In addition, we observed that the degree of maximum SBP decline was higher in the delirium group than in the nondelirium group, which indirectly suggested that a hypotensive and hypoperfusion state may affect the postoperative incidence of delirium. Hypotension is the main physiological mechanism for reducing cerebral perfusion and oxygenation. Controlled hypotension is widely used during endovascular surgical procedures to reduce bleeding and improve the procedure of micronavigation and embolization [46, 47]. There are three reasons underscoring the importance of controlling hypotension for aneurysm endovascular surgery. First, high blood pressure increases the incidence of bleeding, surgery failure rates, and risk of aneurysm rupture [48]. Second, cerebral hyperperfusion syndrome may be caused by rapidly increased blood flow into chronically hypoperfused parenchyma with resultant impaired autoregulation, which has been noted following the clipping of intracranial aneurysms and carotid stenting. Therefore, hypotension is an intraprocedural technique that can decrease the risk of cerebral hyperperfusion syndrome [49]. Third, clinically controlled hypotension prevents wire-induced vessel injury during endovascular therapy because blood pressure fluctuates significantly in patients with fragile vascular status [50].

However, blood pressure that is too low affects cerebral perfusion and oxygenation, which carries an increased risk in patients with cerebral vascular dysfunction. Previous studies have found that reduction in SBP to 140 mmHg can lead to delirium and poorer outcomes in endovascular

intracranial aneurysm. Measuring rSO₂ is a noninvasive technique that can be used for bedside monitoring of cerebral oxygen saturation. It offers real-time data acquisition, excellent sensitivity, rapidity, and persistence. It is currently the most widely used method for monitoring cerebral oxygen saturation [29]. In recent years, there has been a significant increase in the use of rSO₂ perioperatively, especially in cardiac, aortic, carotid, thoracic, neonatal, and geriatric surgeries, as well as in resuscitation [24, 30, 31]. The increasing use of rSO₂ in clinical practice has allowed the maintenance of a specific rSO₂ range shortening the length of tracheal extubation and duration of intensive care unit and hospital stay [32–34]. Appropriate maintenance of rSO₂ contributes to improved patient outcomes, reduces the occurrence of postoperative neurological dysfunction, and decreases mortality rates [35, 36]. Our findings suggest that cerebral oximetry with near-infrared spectroscopy allows the estimation of brain tissue oxygenation that can directly predict delirium. In our research, the rSO₂ desaturation score over time was used as a key index to directly evaluate cerebral oxygen supply status of patients and served as a predictor of postoperative delirium.

Previous research has reported that delirium results in prolonged hospital stay, increased medical costs, increased postoperative complications, increased mortality, and decreased functional status after discharge [37–40]. Mortality is increased by 11% for every additional 48 h

### Table 3 Intraoperative clinical factors of the delirium and no-delirium groups

| Variable                           | Delirium (no)   | Delirium (yes)   | P value |
|------------------------------------|-----------------|------------------|---------|
| Operation duration (min)           | 101.35 (87.37–115.32) | 123.08 (81.69–164.46) | 0.147   |
| Anesthesia duration (min)          | 133.46 (120.13–146.79) | 168.46 (127.06–209.86) | 0.023*  |
| Mechanical ventilation duration (min) | 118.65 (105.18–132.13) | 143.85 (100.51–187.18) | 0.044*  |
| Maximum decline in SBP (%)         | 24.86 (21.78–27.93) | 32.98 (28.78–37.19) | 0.002** |
| Maximum decline in DBP (%)         | 23.75 (20.01–27.49) | 25.71 (18.69–32.74) | 0.300   |
| rSO₂ desaturation score            | 231.95 (55.70–408.21) | 1267.44 (866.82–1668.07) | 0.001** |

rSO₂: Regional cerebral oxygen saturation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Data expressed as mean (95% confidence interval), based on t test

*P < 0.05

**P < 0.01

### Table 4 Spearman correlation analysis of the different predictors of delirium

| Variable                           | r     | Significance |
|------------------------------------|-------|--------------|
| rSO₂ desaturation score            | 0.597 | 0.001**      |
| Maximum decline in SBP (%)         | 0.465 | 0.003**      |
| Anesthesia duration (min)          | 0.183 | 0.264        |
| Mechanical ventilation duration (min) | 0.114 | 0.489        |
| Two risk factors                   | 0.486 | 0.002**      |

rSO₂: Regional cerebral oxygen saturation, SBP: Systolic blood pressure

**P < 0.01
As a 20% reduction in rSO2 from baseline). Hypotension occurred in 99% of patients undergoing surgery, but only 10% of patients experienced cerebral desaturation (defined as a 20% reduction in rSO2 from baseline). Hypotension was observed almost 100 times as often as cerebral desaturation [11]. This indicates that hypotension is not fully coincidental with cerebral desaturation, of which the direct outcome is delirium. Our results revealed a drop in maximum SBP, with a difference of approximately 10 mmHg between groups. Nevertheless, no contribution of delirium was observed in multivariate logistic regression analysis of the predictors of delirium. Therefore, it is important to consider the optimal range for controlled hypotension to ensure appropriate brain perfusion. The current lack of certain safety limits for controlling hypotension suggests that direct monitoring to measure end organ perfusion or a validated surrogate measure may be more useful in endovascular surgery. Our data indicated that early and continuous rSO2 monitoring is a particularly important strategy to detect and further protect patients from delirium.

The etiology of delirium can be multifactorial. We observed that anesthesia and mechanical ventilation durations were significantly different between groups; therefore, anesthesia accumulation may be a significant risk for delirium, as reported in other studies [52]. These factors may indirectly affect delirium outcome. The potential mechanisms underlying their impact on delirium may include hemodynamic changes, neurotoxicity, delays in treatment, or prolonged intubation. Medical history of hypertension, diabetes mellitus, coronary artery disease, and stroke indicates a fragile vascular status. Our research demonstrated that a combination of two of these factors increased the risk of postoperative delirium. Patients with systemic vascular disease have a reduced tolerance for a decline in cerebral blood flow during anesthesia. Furthermore, there is an increased risk of cerebral hypoperfusion in systemic vascular disease, which may lead to cerebral metabolic dysfunction.

We determined rSO2 using near-infrared spectroscopy, which provided an objective measure of the supplementation and consumption of oxygen in the brain. In summary, we observed that changes in the rSO2 desaturation score were closely associated with the occurrence of delirium following cerebral endovascular intervention. We argue that treatments to prevent cerebral oxygen saturation falling below the rSO2 desaturation score threshold of 493.96 should be considered to prevent postoperative delirium in patients undergoing endovascular surgery. rSO2 is an important parameter for minimizing delirium and subsequent negative adverse events in high-risk cerebral vascular interventions. Our findings support intraoperative monitoring of cerebral oxygen saturation as a guide for controlled hypotension, which will reduce the risk of postoperative delirium.

There are two limitations of this study. All patients were recruited from a single center. Second, we observed that anesthesia and mechanical ventilation durations were significantly different between groups. We are planning further studies to address this and plan to record total anesthesia load to enable a more detailed assessment of the effects of anesthesia.

**Conclusions**

In conclusion, this study demonstrated that rSO2 was a useful monitoring tool for anesthesiologists to maintain and achieve ideal hypotension and ensure cerebral perfusion and oxygenation in patients undergoing cerebral endovascular intervention.

**Additional file**

Additional file 1: STROBE statement: checklist of items that should be included in reports of cohort studies. (DOC 84 kb)

**Abbreviations**

ASA: American Society of Anesthesiologists; BMI: Body mass index; CAM: Confusion Assessment Method; CI: Confidence interval; DBP: Diastolic blood pressure; ECG: Electrocardiogram; OR: Odds ratio; OR: Operating room; PETCO2: End-tidal carbon dioxide pressure; rSO2: Regional cerebral oxygen saturation; SBP: Systolic blood pressure; SpO2: Oxygen saturation measured by pulse oximetry

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Authors’ contributions
XW performed the research and drafted the manuscript. HL contributed to data acquisition. YL repeated part of the experimental research. MY analyzed and interpreted the data. KF contributed to the statistical analysis and the elaboration of the manuscript. GZ and TW contributed to the design of the research and agreed to be accountable for all aspects of this work. All authors read and approved the final manuscript.

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Availability of data and materials
The raw data of this study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate
This prospective observational study was approved by the local ethics committee (Capital Medical University Institutional Review Board, China, January 23, 2015; IRB #0810-758). Written informed consent was obtained from all patients participating in the trial. The trial was registered with http://www.ClinicalTrials.gov (NCT02356133; date of registration, February 1, 2015). Our methodology followed the international guidelines for observational studies.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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