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Influence of Blood Pressure Change on Cerebral Perfusion During Interventional Embolization of Intracranial Aneurysm

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Abstract

Background : Rupture and hemorrhage of intracranial aneurysms constitute the leading cause of spontaneous subarachnoid hemorrhage. Endovascular embolization is the primary treatment for intracranial aneurysms, where intraoperative blood pressure management is critical to prevent aneurysm rupture and cerebral hypoperfusion. However, unified optimal intraoperative and postoperative blood pressure control standards remain undefined due to insufficient clinical evidence.

Methods: This study aimed to explore risk factors for cerebral hypoperfusion during intracranial aneurysm embolization and provide evidence for precise anesthetic management.

Results

Of the 89 patients included, 55 (61.8%) developed postoperative PI > 1.1. The hypoperfusion group had a higher proportion of hypertension (60.0% vs. 32.4%, *P* = 0.021) and greater intraoperative SBP variance (median 652.82 vs. 373.19 mmHg², *P* = 0.009). Multivariable analysis identified hypertension (a*OR* 2.72, 95% *CI*1.09–6.81, *P* = 0.033) and SBP variance (a*OR* 1.02, 95% *CI*1.00–1.04, P = 0.009) as independent predictors of PI > 1.1. A significant interaction was found between hypertension and SBP variance (*P* = 0.022); the effect of hypertension was strongest in patients with above-median SBP variance (aOR 20.68, 95% CI 2.25–190.49, *P* = 0.007). The nomogram integrating these two predictors showed moderate discrimination (AUC 0.706, 95% *CI* 0.599–0.813).

Conclusions: In conclusion, intraoperative systolic blood pressure variability and a history of hypertension are independently linked to postoperative cerebral hypoperfusion following endovascular embolization of intracranial aneurysms. Hypertensive patients are particularly sensitive to blood pressure changes. A baseline diastolic blood pressure of 96 mmHg appears to have a threshold effect. A straightforward nomogram based on these factors could aid in personalized blood pressure control and risk assessment during and after endovascular aneurysm interventions. Further validation in larger cohorts is necessary.

Introduction

Intracranial aneurysms typically arise from congenital defects or abnormal increases in vessel wall pressure, leading to localized, pathological bulging of the intracranial arteries[1, 2]. The prevalence of intracranial aneurysms is high, affecting more than 3% of the adult population [3,4].Once an aneurysm ruptures, the rates of disability and mortality increase rapidly. Aneurysm rupture is also the primary cause of subarachnoid hemorrhage[5, 6, 7].Early diagnosis of intracranial aneurysms, followed by effective intervention and treatment, is crucial for improving patient survival rates and quality of life. This issue remains a primary focus of clinical research and requires urgent attention[8, 9, 10].

The main treatment options for intracranial aneurysms are surgical clipping and endovascular embolization[11]. Surgical clipping is the most traditional approach. However, with rapid advancements

in endovascular techniques and the introduction of specialized materials such as detachable coils and flow-diverting stents, endovascular treatment has become the preferred option for most intracranial aneurysms[12]. This is especially true for patients who cannot tolerate open surgery or who have multiple high-risk factors[13]. Interventional procedures are characterized by minimal trauma, reduced stimulation, and rapid recovery. However, some patients still experience adverse outcomes, such as acute or chronic neurological dysfunction. These outcomes are associated with factors including the timing of intervention, the presence of hypertension, and the Fisher and Hunt-Hess grading scales[14, 15].

In the interventional treatment of intracranial aneurysms, general anesthesia is the preferred choice[16, 17]. The goals of anesthesia management are to maintain sufficient perfusion pressure to prevent cerebral ischemia while also controlling hypertension to reduce the risk of aneurysm rupture or exacerbation of intracranial hemorrhage. Therefore, it is crucial to maintain blood pressure within a specific range throughout the various stages of aneurysm surgery.

Under normal physiological conditions in healthy adults, cerebral blood flow remains constant due to the automatic dilation and constriction of blood vessels, a process known as the cerebral vascular autoregulation mechanism[18, 19]. However, various factors can weaken or abolish this autoregulatory mechanism. When this occurs, cerebral blood flow in the affected region becomes dependent on mean arterial pressure. In patients with intracranial aneurysms, cerebral autoregulation is impaired[20, 21]. As a result, both decreases and increases in blood pressure can alter cerebral perfusion pressure, potentially leading to cerebral ischemia or aneurysm rupture.Previous studies have shown that the risk of brain tissue hypoxia in severely head injured patients could be high when CPP is below the recommended threshold of 60 mmHg[22]. Before clamping or embolizing an aneurysm, elevated blood pressure raises the transmural pressure, thereby increasing the likelihood of rebleeding. Systolic blood pressure exceeding 160 mmHg is a risk factor for rebleeding in ruptured aneurysms [23]. However, due to variations in baseline blood pressure and differences in the degree of impairment of cerebral autoregulation among patients, there is currently no clear blood pressure threshold that both ensures adequate perfusion and minimizes rupture risk during aneurysm intervention. Randomized controlled trial data supporting such a threshold are lacking.

During surgery, blood pressure is affected by the patient’s condition, surgical stimulation, and anesthetic drugs. Monitoring baseline pressure is essential, and limiting intraoperative blood pressure fluctuations has become a critical component of clinical care. Blood pressure shows marked short-term variability between heartbeats, over minutes, and across 24 hours, as well as longer-term variation over days to months. These patterns arise from the combined action of multiple regulatory mechanisms and external stimuli rather than from random noise. Although many adverse outcomes depend primarily on absolute blood pressure, analyses of observational studies and clinical trials indicate that increased blood pressure variability also contributes to harm. Blood pressure variability (BPV) denotes the magnitude of pressure change over a defined interval. For assessment of short-term fluctuations, clinicians typically

use the standard deviation or variance of blood pressure; these traditional metrics remain the most commonly applied measures of BPV[24, 25].

This study investigates how intraoperative blood pressure fluctuations during endovascular aneurysm embolization affect cerebral perfusion and analyzes risk factors that influence cerebral blood flow, aiming to provide a rigorous theoretical foundation for anesthetic management in endovascular aneurysm embolization.

Methods

This study was approved by the Ethics Committee of the First Affiliated Hospital of Harbin Medical University.All methods were performed in accordance with the relevant guidelines and regulations, including the Declaration of Helsinki. Written informed consent was obtained from all patients or their legal representatives prior to enrollment.

2.1 Study Subjects

The study population comprised 96 patients who presented to the Department of Neurosurgery at the First Affiliated Hospital of Harbin Medical University (Qunli Campus) between July 7 and December 1, 2022, and were scheduled for elective or emergency intracranial aneurysm endovascular embolization under general anesthesia.

Patients who meet the following criteria will be enrolled in the study: Diagnosed with an intracranial aneurysm by digital subtraction angiography (DSA);Meeting the indications for endovascular intervention for intracranial aneurysms; American Society of Anesthesiologists Physical Status Classification (ASA) II–IV; The patient and their family have understood the purpose, procedures, methods, and underlying principles of this study and have signed the informed consent form.those meeting any of the exclusion criteria listed below will be excluded: Concurrent malignancies in other organ systems; Patients classified as Hunt-Hess grade V; In the presence of severe hepatic and renal dysfunction; Accompanied by infection; Accompanied by moyamoya disease or arteriovenous malformation.In the following cases, the patient will be knocked out.Intraoperative aneurysm rupture occurred; postoperative acute complications appeared; Individuals with incomplete or missing clinical data and information; Patients in whom bilateral upper limb radial artery puncture and catheterization attempt failed; Difficult endotracheal intubation or endotracheal intubation duration exceeding 30 seconds; Failure to acquire TCD data.

2.2 Management of Anesthesia

Before anesthesia, the anesthesiologist conducts a thorough assessment of the patient’s medical condition, including general status, cardiopulmonary function, hepatic and renal function, and endocrine system function. In addition to accurately assigning an ASA classification and understanding the functions of vital organs and systems, the anesthesiologist also pays close attention to specialized

neurological examinations, such as CT and DSA results, the presence or absence of cerebral herniation or cerebral edema, the patient's level of consciousness, and limb motor function. For patients with intracranial aneurysms, preoperative medical management typically involves restricting fluid intake or administering diuretics. The anesthesiologist carefully monitors the patient's fluid and electrolyte status, adjusts the preoperative medication regimen based on the patient's clinical condition, and makes appropriate modifications to the treatment plan.

All anesthesia-related procedures and skills involved in this study were performed by experienced anesthesiologists.

For patients meeting the inclusion criteria, the standard fasting period is 8 hours for solid foods and 4 hours for liquids. After the patient enters the operating room, an intravenous access is established, and non-invasive blood pressure monitoring of the upper limb, a 5-lead electrocardiogram, and pulse oximetry are initiated. If the Allen test on one of the patient's upper limbs is negative, radial arterial cannulation will be performed on that same side. Once successful radial arterial cannulation is achieved, the catheter is securely fixed in place. After successful radial arterial cannulation, the zero point is fixed at the fourth intercostal space along the midaxillary line and calibrated accordingly. All patients received combined intravenous and inhalational anesthesia. Airway management options included endotracheal intubation and laryngeal mask airway placement. After the anesthesiologist provides mask ventilation (with an oxygen flow rate exceeding 6 L/min) for 1 minute, intravenous rapid induction is carried out according to the following protocol: propofol 1.5–2.5 mg/kg; rocuronium 0.3–0.6 mg/kg; and sufentanil 0.4–0.5 μg/kg. Once the neuromuscular blocking agent has reached its peak effect (manifested by decreased resistance to mask ventilation and relaxation of the mandible), the anesthesiologist will proceed with either endotracheal intubation or laryngeal mask airway placement.After successful intubation or laryngeal mask airway placement, the anesthesiologist connects the patient to a ventilator and uses volume-controlled ventilation mode to regulate breathing. Ventilator parameters are adjusted as follows: tidal volume set at 6–8 ml/kg, respiratory rate at 10–12 breaths per minute, inspiratory-to-expiratory ratio of 1:2, fresh gas flow at 2 L/min, and oxygen concentration at 60%. During surgery, respiratory parameters are adjusted based on end-tidal CO_2 ($PetCO_2$) levels to maintain $PetCO_2$ within the range of 35–45 mmHg. Sevoflurane is used for anesthetic maintenance throughout the procedure, keeping the concentration above 0.6 MAC. Remifentanil is administered via continuous infusion at a rate of 0.05–0.1 μg/kg/min. During surgery, bispectral index (BIS) monitoring is performed continuously, maintaining BIS values between 40 and 60. Intermittent bolus doses of rocuronium bromide at 0.15 mg/kg are administered as needed. The volume deficit is determined based on the patient's fasting duration and body weight, with crystalloid solutions preferred. Twenty minutes before the end of surgery, dezocine 5 mg and ondansetron 10 mg are administered intravenously. Upon completion of the procedure, sevoflurane inhalation is discontinued, and remifentanil infusion is stopped. Once spontaneous breathing has been restored, neostigmine 0.02–0.04 mg/kg is administered intravenously, followed by half or one-third of the neostigmine dose of atropine to counteract residual neuromuscular blockade. The endotracheal tube or laryngeal mask is gently removed after slowly suctioning secretions from the oropharynx using a suction device. The patient is then provided with oxygen via a face mask,

and the oxygen flow rate is adjusted according to the patient's SpO_2 to maintain it above 95%. When the patient has fully regained consciousness (able to open eyes upon verbal stimulation and follow verbal commands), exhibits a firm and strong handshake, and is accompanied by the anesthesiologist, the patient is transferred back to the neurosurgical intensive care unit.

2.3 Management of Anesthetic Complications and Abnormal Situations

In the event of abnormal hemodynamic conditions during surgery—such as hypertension or hypotension—experienced anesthesiologists shall manage these situations according to the expert consensus and guidelines for this type of surgery. Vasoactive drugs shall be prepared in a standardized manner, and the usage of these drugs shall be meticulously documented.For abnormal hypertension (an increase in mean arterial pressure exceeding 20% above baseline), administer nicardipine intravenously at a dose of 2–10 μg/kg.For abnormal hypotension (a decrease in mean arterial pressure exceeding 20% below baseline), administer metaraminol intravenously at a dose of 0.2–0.5 mg per dose or ephedrine at a dose of 3–6 mg per dose.For a heart rate below 50 beats per minute, administer atropine intravenously at a dose of 0.3 mg per dose.For a heart rate exceeding 120 beats per minute, administer esmolol intravenously at a dose of 10–15 mg per dose. The above-mentioned drugs may be repeated as necessary.

2.4 Cerebral Hemodynamic Monitoring

Before the surgery begins, transcranial Doppler ultrasound (TCD) via the temporal window is used to measure the spectral and hemodynamic parameters of the middle cerebral artery (MCA) on the patient's healthy side. The sampling depth is set at 40–65 mm, with blood flow directed toward the probe, resulting in a positive-flow spectrum. The probe position is marked.

After the surgery is completed, the same method described above is used to record the hemodynamic parameters of the same vessel, at the same location, on the same side as before the surgery.

2.5 Blood Pressure-related Definations

Baseline Blood Pressure: After successful radial artery cannulation and calibration, record the direct arterial pressure at that time. Blood Pressure Variability: Every 5 minutes, record the systolic blood pressure, diastolic blood pressure, and mean arterial pressure from the time of successful radial artery cannulation and calibration until the radial artery catheter is removed. Calculate the variance of these three parameters, defining this value as blood pressure variability(1).

$$Blood\ Pressure\ \text{Variability} = \sum_{i=0}^{n} (\chi_i - \chi)^2 / (n-1) \quad (1)$$

Here, χ_i represents the blood pressure value, χ represents the average blood pressure, and n is the number of measurements.

Maximum Systolic Blood Pressure: The highest systolic blood pressure recorded from the moment the radial artery catheterization is successfully performed and calibrated until the radial artery catheter is removed. Minimum Systolic Blood Pressure: The lowest systolic blood pressure recorded from the moment the radial artery catheterization is successfully performed and calibrated until the radial artery catheter is removed.Blood Pressure Fluctuation Range is about increase range and decrease range which relies on the differences between maximum values, minimum values, and baseline values.

2.6 Statistical analysis

All data were entered into a computer and subjected to statistical analysis using SPSS 25.0. Categorical data are presented as number of cases (%), and between-group comparisons were performed using the Pearson chi-square test or Fisher's exact probability test. Continuous data that followed a normal distribution are described as "mean ± standard deviation" (x ± S); between-group comparisons were conducted using the t-test. For data that did not follow a normal distribution, results are expressed as median and interquartile range (M [P25, P75]); between-group comparisons were performed using the Mann-Whitney U test. Ordinal data are presented as number of cases (%), and between-group comparisons were conducted using the Mann-Whitney U test. Factors with $P < 0.05$ in the univariate analysis were included in the multivariate analysis. The multivariate analysis employed stepwise logistic regression to identify independent risk factors, and odds ratios (*ORs*) along with their *95% CI* were calculated. All statistical tests were two-tailed, and $P < 0.05$ was considered to indicate statistically significant differences.

Results

A total of 96 consecutive patients were initially enrolled in this study. Of these, seven patients were excluded for the following reasons: hemiplegia or aphasia at 3 h postoperatively (n = 2), radial artery cannulation failure (n = 3), and failure of Transcranial Doppler (TCD) examination due to a poor acoustic window (n = 2). Ultimately, 89 patients with complete data were included in the final analysis. According to the postoperative PI (rounded to two decimal places), 34 patients (38.2%) were classified into the non-hypoperfusion group (PI ≤ 1.1) and 55 patients (61.8%) into the hypoperfusion group (PI > 1.1) (Fig. 1).

3.1Basic characteristics

A total of 89 patients were enrolled and classified into two groups according to the postoperative pulsatility index (PI; rounded to two decimal places): 34 patients in the PI ≤ 1.1 group and 55 patients in the PI > 1.1 group. As shown in Table 1, the two groups were generally comparable with respect to demographic and clinical characteristics, including age (58.35 ± 10.04 vs. 60.85 ± 8.81 years, $P = 0.221$), sex (female, 67.6% vs. 58.2%, $P = 0.504$), and body mass index (23.30 ± 2.97 vs. 23.70 ± 3.15 kg/m², $P = 0.545$). No significant between-group differences were observed in ASA grade ($P = 0.517$), Hunt-Hess grade ($P = 0.142$), the presence of SAH ($P = 0.120$), multiple aneurysms ($P = 0.203$), aneurysm location ($P = 0.759$), aneurysm size ($P = 0.519$), airway management ($P = 0.082$), or blood loss ($P = 0.716$). However, the proportion of patients with a history of hypertension was significantly higher in the PI > 1.1 group

than in the PI ≤ 1.1 group (60.0% vs. 32.4%, *P* = 0.021; ASD = 0.276), indicating a baseline imbalance that was adjusted for in subsequent multivariable analyses.(Table 1)

Perioperative vasoactive drug use and intraoperative hemodynamic management are summarized in Table 2. The proportions of patients receiving metaraminol (26.5% vs. 25.5%), ephedrine (44.1% vs. 36.4%), nicardipine (29.4% vs. 34.5%), and esmolol (8.8% vs. 18.2%) were comparable between the PI ≤ 1.1 and PI > 1.1 groups (all *P* > 0.05), as were the corresponding drug doses (all *P* > 0.05). No significant between-group differences were observed in admission blood pressure (SBP 124.15 ± 25.57 vs. 132.25 ± 20.63 mmHg, *P* = 0.104; DBP 76.38 ± 11.69 vs. 80.29 ± 11.21 mmHg, *P* = 0.119), OR-entry blood pressure (SBP 145.76 ± 23.12 vs. 152.60 ± 24.75 mmHg, *P* = 0.198; DBP 86.82 ± 11.59 vs. 86.64 ± 12.21 mmHg, *P* = 0.943;), or baseline blood pressure (SBP 134.96 ± 19.58 vs. 142.43 ± 17.57 mmHg, *P* = 0.066; DBP 81.60 ± 8.99 vs. 83.46 ± 9.21 mmHg, *P* = 0.353; MAP 99.39 ± 11.60 vs. 103.12 ± 10.65 mmHg, *P* = 0.124). Although the PI > 1.1 group showed a trend toward higher admission MAP (*P* = 0.078) and baseline SBP (*P* = 0.066), no comparison reached statistical significance.(Table 2)

Table 1
Baseline characteristics of patients stratified by postoperative pulsatility index (PI), presented as mean ± SD or nunber(%)

Variable	**Overall (n = 98)**	**PI ≤ 1.1 (n = 34)**	**PI>1.1 (n = 55)**	***P* value**	**ASD**
Age(years)	59.90 ± 9.32	58.35 ± 10.04	60.85 ± 8.81	0.221	0.265
Sex				0.504	0.095
Female	55(61.8%)	23(67.6%)	32(58.2%)		
Male	34(38.2%)	11(32.4%)	23(41.8%)		
BMI(kg/m^2)	23.55 ± 3.07	23.30 ± 2.97	23.70 ± 3.15	0.545	0.133
ASA				0.517	0.068
2	3(3.4%)	1(2.9%)	2(3.6%)		
3	80(89.9%)	32(94.1%)	48(%)		
4	6(6.7%)	1(2.9%)	5(9.1%)		
Airway management				0.082	0.134
Laryngeal mask airway	79(88.8%)	33(97.1%)	46(83.6%)		
Tracheal intubation	10(11.2%)	1(2.9%)	9(16.4%)		
Comorbidity					
Hypertension	44(49.4%)	11(32.4%)	33(60.0%)	0.021	0.276
Diabetes	11(12.4%)	3(8.8%)	8(14.5%)	0.521	0.057
Hunt-Hess Grade				0.142	0.109
0	73(82.0%)	30(88.2%)	43(78.2%)		
1	6(6.7%)	0(0.0%)	6(10.9%)		
2	7(7.9%)	3(8.8%)	4(7.3%)		
3	1(1.1%)	1(2.9%)	0(0.0%)		
4	2(2.2%)	0(0.0%)	2(3.6%)		
Aneurysm Location				0.759	0.046
Anterior circulation	76(85.4%)	30(88.2%)	46(83.6%)		
Posterior circulation	13(14.6%)	4(11.8%)	9(16.4%)		
Aneurysm size				0.519	0.041

Variable	Overall (n = 98)	PI ≤ 1.1 (n = 34)	PI>1.1 (n = 55)	*P* value	ASD
Small	58(65.2%)	22(64.7%)	36(65.5%)		
Medium	26(29.2%)	10(29.4%)	16(29.1%)		
Large	3(3.4%)	2(5.9%)	1(1.8%)		
Giant	2(2.2%)	0(0.0%)	2(3.6%)		
Subarachnoid hemorrhage	13(14.6%)	2(5.9%)	11(20.0%)	0.120	0.141
Muiltple aneurysms	21(23.6%)	11(32.4%)	10(18.2%)	0.203	0.142

ASD, Absolute standardized difference; values < 0.1 indicate negligible imbalance.

BMI, Body Mass Index

ASA, American Society of Anesthesiologists

Table 2
Hemodynamic variables of patients, presented as mean ± SD or number(%).

Variables	**Overall (n = 98)**	**PI ≤ 1.1 (n = 34)**	**PI>1.1 (n = 55)**	***P*value**	**ASD**
Use of vasoactive drugs					
Dose of metaraminol use(mg)	0.39 ± 1.12	0.24 ± 0.58	0.47 ± 1.35	0.334	0.224
Proportion of metaraminol use	23(25.8%)	9(26.5%)	14(25.5%)	>1.000	0.010
Dose of ephedrine use(mg)	1.64 ± 2.22	1.79 ± 2.24	1.55 ± 2.22	0.610	0.112
Proportion of ephedrine use	35(39.3%)	14(44.1%)	20(36.4%)	0.614	0.078
Dose of nicardipine use(mg)	0.31 ± 0.54	0.23 ± 0.46	0.37 ± 0.58	0.232	0.269
Proportion of nicardipine use	29(32.6%)	10(29.4%)	19(34.5%)	0.788	0.051
Dose of esmolol use(mg)	1.63 ± 4.11	0.88 ± 0.28	2.09 ± 4.68	0.179	0.311
Proportion of esmolol use	13(14.6%)	3(8.8%)	10(18.2%)	0.355	0.094
Blood pressure of admission to the hospital					
Admission systolic blood pressure(mmHg)	129.16 ± 22.85	124.15 ± 25.57	132.25 ± 20.63	0.104	0.349
Admission diastolic blood pressure(mmHg)	78.80 ± 11.49	76.38 ± 11.69	80.29 ± 11.21	0.119	0.341
Blood pressure of entering OR					
OR-entry systolic blood pressure(mmHg)	149.99 ± 24.23	145.76 ± 23.12	152.60 ± 24.75	0.198	0.285
OR-entry diastolic blood pressure(mmHg)	86.71 ± 11.91	86.82 ± 11.59	86.64 ± 12.21	0.943	0.016
Baseline blood pressure					
Baseline of systolic blood pressure(mmHg)	139.57 ± 18.62	134.96 ± 19.58	142.43 ± 17.57	0.066	0.402

Blood pressure of admission to the hospital:The first cuff blood pressure measured in the ward when the patient was admitted to the hospital;Blood pressure of entering OR:On the day of the patient's surgery, the first cuff pressure measured upon entering the operating room;Baseline blood pressure:After the patient underwent radial artery puncture and catheterization, the direct arterial pressure was stabilized for three cardiac cycles after zero point calibration

Variables	Overall (n = 98)	PI ≤ 1.1 (n = 34)	PI>1.1 (n = 55)	*P*value	ASD
Baseline of diastolic blood pressure(mmHg)	82.75 ± 9.12	81.60 ± 8.99	84.46 ± 9.21	0.353	0.204
Baseline of mean arterial blood pressure(mmHg)	101.69 ± 11.10	99.39 ± 11.60	103.12 ± 10.65	0.124	0.335
Blood pressure of admission to the hospital:The first cuff blood pressure measured in the ward when the patient was admitted to the hospital;Blood pressure of entering OR:On the day of the patient's surgery, the first cuff pressure measured upon entering the operating room;Baseline blood pressure:After the patient underwent radial artery puncture and catheterization, the direct arterial pressure was stabilized for three cardiac cycles after zero point calibration					

3.2Fluctuations of Intraoperative blood pressure

Intraoperative blood pressure variability is summarized in Table 3. The PI > 1.1 group exhibited significantly higher SBP SD [median 25.55 (IQR 18.32, 30.63; range 3.28–46.67) vs. 19.45 (16.07, 23.12; 8.93–38.03) mmHg, *P* = 0.011] and SBP variance [652.82 (335.80, 938.52; 10.76–2177.80) vs. 373.19 (258.21, 512.16; 79.82–1446.59) mmHg², *P* = 0.009] than the PI ≤ 1.1 group, whereas DBP SD and DBP variance did not differ significantly between groups (both *P* = 0.301). MAP SD and MAP variance showed borderline trends (*P* = 0.068 and *P* = 0.064, respectively).

Regarding direct arterial pressure extremes, the PI > 1.1 group had higher direct arterial baseline SBP, higher intraoperative SBP maximum [172.00 (151.50, 188.50; 125.00–236.00) vs. 154.50 (140.25, 169.50; 107.00–202.00) mmHg, *P* = 0.018], and higher SBP minimum [92.00 (85.00, 103.50; 71.00–127.00) vs. 88.00 (84.25, 94.25; 73.00–118.00) mmHg, *P* = 0.043], with no significant difference in baseline DBP (*P* = 0.748) or MAP (*P* = 0.089).

The systolic blood pressure fall amplitude (baseline SBP − minimum SBP) was significantly greater in the PI > 1.1 group compared to the PI ≤ 1.1 group (67.61 vs 58.47 mmHg, *P* = 0.037, ASD = 0.475). In contrast, the systolic blood pressure rise amplitude (maximum SBP − baseline SBP) showed no significant difference between groups (4.64 vs 5.97 mmHg, *P* = 0.342, ASD = 0.214). These findings suggest that higher post-operative pulsatility index is associated with greater intra-operative systolic blood pressure declines but not with the magnitude of pressure increases.

Table 3
Intraoperative blood pressure variability stratified by postoperative PI presented as median (IQR [range]) or mean ± SD

Variable	Overall (n = 89)	PI ≤ 1.1 (n = 34)	PI > 1.1 (n = 55)	*P* value	ASD
Blood pressure variability					
SBP SD(mmHg)	22.37 (17.08, 29.37 [3.28, 46.67])	19.45 (16.07, 23.12 [8.93, 38.03])	25.55 (18.32, 30.63 [3.28, 46.67])	0.011	0.550
SBP variance($mmHg^2$)	497.27 (291.86, 862.84 [10.76, 2177.80])	373.19 (258.21, 512.16 [79.82, 1446.59])	652.82 (335.80, 938.52 [10.76, 2177.80])	0.009	0.634
DBP SD(mmHg)	10.79 (8.17, 13.51 [3.08, 30.13])	10.68 (8.28, 12.18 [5.06, 26.82])	10.99 (8.13, 15.21 [3.08, 30.13])	0.301	0.310
DBP variance($mmHg^2$)	116.52 (66.67, 182.42 [9.47, 907.95])	114.17 (68.58, 148.27 [25.58, 719.50])	120.75 (66.02, 231.33 [9.47, 907.95])	0.301	0.341
MAP SD(mmHg)	14.44 (11.43, 18.52 [3.50, 31.35])	13.06 (10.89, 15.56 [6.64, 30.53])	15.49 (11.46, 19.94 [3.50, 31.35])	0.068	0.440
MAP variance($mmHg^2$)	210.34 (130.69, 342.98 [12.22, 982.59])	170.66 (119.72, 242.22 [40.09, 931.84])	239.80 (131.28, 397.62 [12.22, 982.59])	0.064	0.477
Direct arterial blood pressure extremes and range of changes(mmHg)					
SBP maximum(mmHg)	165.30 ± 25.51	156.43 ± 24.65	169.81 ± 24.94	0.018	0.539
SBP minimum(mmHg)	93.06 ± 12.78	92.00 ± 10.62	93.59 ± 13.81	0.581	0.124
SBP ascent range	4.64 ± 9.34	5.97 ± 11.60	3.97 ± 7.98	0.342	0.214
SBP descent range	67.61 ± 29.59	58.47 ± 30.52	72.25 ± 28.24	0.037	0.475

SBP:Systolic blood pressure;DBP:Diastolic blood pressure;MAP:Mean arterial pressure;SD:The standard deviation of intraoperative sampling and direct arterial pressure;SBP maximum and minimum:The maximum and minimum values of blood pressure during the collection process;SBP ascent range: The maximum minus the baseline of SBP;SBP descent range: The baseline minus the minimum of baseline.

3.3Multivariate logistic regression analysis

A multivariate logistic regression analysis was performed to identify independent risk factors associated with postoperative pulsatility index (PI) > 1.1 using a cutoff value of 1.1. Among the variables evaluated,

a history of hypertension and systolic blood pressure (SBP) variance emerged as significant independent predictors.

Patients with a history of hypertension had 2.72 times higher odds of developing a postoperative PI > 1.1 compared to those without such history (adjusted odds ratio [a*OR*] = 2.72, 95% confidence interval [*CI*]: 1.09–6.81, *P* = 0.033). Additionally, for every one unit increase in SBP variance, the odds of having a PI > 1.1 increased by 2% (a*OR* = 1.02, 95% *CI*: 1.00–1.04, *P* = 0.009).(Table 4)

Table 4
Independent risk factors affecting cerebral blood flow perfusion

Independent risk factors	***OR* values**	**95%*CI***	***P* value**
Hypertension history	2.72	1.09,6.81	0.033
SBP variance	1.02	1.00,1.04	0.009
After adjustment for covariates, no statistically significant associations were observed for age, sex, ASA physical status, or type of surgical approach (all P > 0.05).			

3.4 Subgroup analysis and effect modification analyses

In subgroup analyses stratified by age (< 65 vs. ≥65 years), ASA physical status (II–III and IV), and intraoperative SBP variance categories, the association between hypertension and postoperative PI > 1.1 was concentrated in patients with higher SBP variance. When the cohort was dichotomized at the median SBP variance (497.27), hypertension was strongly associated with PI > 1.1 in the above-median group (a*OR* 20.68, 95% *CI* 2.25–190.49, *P* = 0.007) but not in the below-median group (a*OR* 1.26, 95% *CI* 0.35–4.50, *P* = 0.724). Consistent with this, in tertile-based analyses hypertension was significantly associated with PI > 1.1 in the second tertile (a*OR* 9.59, 95% *CI* 1.24–74.27, *P* = 0.030) and the third tertile (a*OR* 11.31, 95% *CI* 1.02–125.88, *P* = 0.048) of SBP variance, whereas no significant association was observed in the lowest tertile (a*OR* 0.75, 95% *CI* 0.15–3.69, *P* = 0.725). SBP variance itself remained a significant predictor in the second tertile (a*OR* 1.011, 95% *CI* 1.000–1.021, *P* = 0.040) but not in the highest tertile. In the age-stratified analysis, SBP variance was significantly associated with PI > 1.1 among patients younger than 65 years (a*OR* 1.0014, 95% *CI* 1.000–1.003, *P* = 0.038) but not among those aged 65 years or older. Within ASA II–III patients, SBP variance was a significant predictor (a*OR* 1.002, 95% *CI* 1.000–1.003, *P* = 0.011), whereas hypertension was not (a*OR* 2.20, 95% *CI* 0.83–5.81, *P* = 0.111). Formal interaction tests revealed no significant effect modification by age for either hypertension (interaction *P* = 0.495) or SBP variance (interaction *P* = 0.688). Overall, these findings suggest that the effect of hypertension on postoperative PI > 1.1 is more pronounced under conditions of greater intraoperative blood pressure variability.(Fig. 2)

Multivariable logistic regression was performed with postoperative PI > 1.1 (PI rounded to two decimal places) as the dependent variable, and hypertension and intraoperative systolic blood pressure (SBP) variance as covariates, in the overall cohort (diamond, bold) and in prespecified subgroups (squares). (A) Hypertension: a*OR*s are plotted on a logarithmic scale; (B) intraoperative SBP variance: a*OR*s per unit

increase in SBP variance (± 1 unit), plotted on a linear scale. Points represent a*OR*s and horizontal lines represent 95% confidence intervals (*CI*s). The vertical dashed line indicates the null value (a*OR* = 1). Overall cohort (n = 89): hypertension a*OR* = 2.70 (95% *CI* 1.06–6.86, *P* = 0.037); SBP variance a*OR* = 1.0013 (95% *CI* 1.000–1.002, *P* = 0.026). Subgroups were stratified by age (< 65 vs. ≥ 65 years), ASA physical status II–III, and tertiles or median split of intraoperative SBP variance (median = 497.27). In the above-median SBP variance subgroup (n = 44), hypertension was significantly associated with PI > 1.1 (a*OR* = 20.68, 95% *CI* 2.25–190.49, *P* = 0.007); no significant association was observed in the below-median subgroup (n = 45; a*OR* = 1.26, 95% *CI* 0.35–4.50, *P* = 0.724). N (events), number of patients (number with postoperative PI > 1.1); a*OR*, adjusted odds ratio; *CI*, confidence interval.

In the overall cohort, both hypertension (aOR 2.70, 95% CI 1.06–6.86; P = 0.037) and intraoperative SBP variance (aOR 1.001 per unit increase, 95% CI 1.000–1.002; P = 0.026) were independently associated with postoperative PI > 1.1 in multivariable logistic regression.

A statistically significant multiplicative interaction was detected between hypertension and intraoperative SBP variance (interaction aOR 1.003 per unit increase in SBP variance, 95% CI 1.000–1.007; P for interaction = 0.049; likelihood-ratio test P = 0.022), indicating that the effect of hypertension on PI > 1.1 increased with greater intraoperative blood pressure variability.(Table 5)

Table 5
Effect modification analyses

Interaction term	**Interaction a*OR* (per unit)**	**95%*CI***	**Wald *P***	**LR *P***
HTN*SBP Variance	1.003	1.000-1.007	0.049	0.022

Stratifying patients at the median SBP variance (497.27) confirmed this effect modification: hypertension was strongly associated with PI > 1.1 among patients with above-median SBP variance (a*OR* 20.68, 95% *CI* 2.25–190.49; *P* = 0.007), whereas no significant association was observed in the below-median group (a*OR* 1.26, 95% *CI* 0.35–4.50; *P* = 0.724). Tertile-based analyses were consistent, with significant associations in the second (a*OR* 9.59, 95% *CI* 1.24–74.27; *P* = 0.030) and third (a*OR* 11.31, 95% *CI* 1.02–125.88; *P* = 0.048) tertiles of SBP variance but not in the lowest tertile (a*OR* 0.75, 95% *CI* 0.15–3.69; *P* = 0.725).

In contrast, no significant effect modification by age was observed for either hypertension (*P* = 0.495) or SBP variance (*P* = 0.688). Restricted cubic spline analysis indicated a near-linear association of SBP variance with PI > 1.1 without significant deviation from linearity (*P* for nonlinearity = 0.814). The combined hypertension–SBP variance model showed moderate discrimination for postoperative PI > 1.1 (AUC 0.706, 95% CI 0.599–0.813).

3.5 Restricted cubic spline and threshold analyses

To explore potential non-linear relationships and threshold effects, restricted cubic spline (RCS) regression and single-changepoint threshold analyses were performed for nine continuous blood pressure parameters.

After adjustment, four parameters showed significant monotonic linear associations with postoperative PI > 1.1: intraoperative SBP variance (*OR* 1.0015 per unit, 95% *CI* 1.0003–1.0026; *P* = 0.012), SBP maximum (*OR* 1.023 per mmHg, 95% *CI* 1.004–1.042; *P* = 0.015), baseline SBP (*OR* 1.021 per mmHg, 95% *CI* 1.003–1.038; *P* = 0.019), and MAP variance (*OR* 1.0025 per unit, 95% *CI* 1.0000–1.0049; *P* = 0.048). Restricted cubic spline analysis revealed no significant departure from linearity for any of these parameters (all *P* for non-linearity > 0.14), indicating that the risk increased monotonically across the observed range without a discernible threshold.

In contrast, baseline DBP demonstrated a statistically significant threshold effect: although the overall linear association was not significant (*P* = 0.569), the likelihood-ratio test identified an optimal changepoint at 96 mmHg *(P* = 0.006), above which the slope of the association with postoperative PI > 1.1 increased steeply. Threshold candidates for SBP variance (163) and SBP descent range (24) reached nominal significance but were based on small numbers of observations at the lower end of the distribution and should be interpreted cautiously.

The magnitude of intraoperative SBP ascent was excluded from curve fitting because hypertensive episodes were rare in this cohort (median ≈ 0 mmHg) and it showed no association with postoperative PI > 1.1 (OR 0.99, 95% CI 0.94–1.04; P = 0.627).(Figure 3)

OR. odds ratio; PI, pulsatility index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; LR, likelihood ratio. SBP ascent range was excluded from the curve-fitting analysis because it was essentially zero in most patients, (limited surgical hypertensive episodes) and showed no significant association, (OR 0.99, P = 0.627); it is therefore not tabulated but is addressed in the Discussion

Figure 3 Restricted cubic spline curves of the association between each continuous blood pressure parameter and postoperative PI > 1.1. Solid curves = adjusted odds ratio; shaded areas = 95% *CI* (univariable); red dotted lines = spline knots; red solid line = significant threshold (baseline DBP only, *P* < 0.05)

3.6 Predictive model

A nomogram was constructed from the multivariable logistic regression model to provide a clinically accessible tool for predicting postoperative cerebral hypoperfusion (postoperative PI < = 1.1). The nomogram included two predictors: hypertension and intraoperative SBP variance (Fig. 4B. Hypertension (yes vs no) contributed 100 points and SBP variance contributed 0 to 100 points (200 to 700 $mmHg^2$). The discrimination of the nomogram was moderate, with an area under the receiver operating characteristic curve (AUC) of 0.706 (95% *CI* 0.599–0.813). Internal validation using 1,000 bootstrap resamples yielded an optimism-corrected AUC of 0.689, indicating minimal overfitting (optimism =

0.017). The Brier score was 0.208 (apparent) and 0.224 (bias-corrected). The calibration curve showed good agreement between predicted and observed probabilities (Fig. 4A).

Discussion

In this single-center prospective cohort of 89 patients undergoing endovascular embolization of intracranial aneurysms under general anesthesia, we investigated whether intraoperative blood pressure variability (BPV) is associated with postoperative cerebral hypoperfusion, assessed using transcranial Doppler (TCD)-derived pulsatility index (PI). We found that a history of hypertension (adjusted odds ratio [a*OR*] 2.72, 95% *CI* 1.09–6.81; *P* = 0.033) and intraoperative systolic blood pressure (SBP) variance (a*OR* 1.02 per unit, 95% *CI* 1.00–1.04; *P* = 0.009) were independently associated with postoperative PI > 1.1, and that a simple two-variable nomogram integrating these predictors achieved moderate discrimination (AUC 0.706, 95% *CI* 0.599–0.813). These findings indicate that the magnitude of intraoperative blood pressure fluctuation, rather than the absolute pressure level alone, is an important modifiable determinant of early cerebral perfusion after aneurysm embolization.

Intraoperative BPV and impaired cerebral perfusion: direction of harm

Our findings back the involvement of BPV in postoperative cerebral hypoperfusion. In normal circumstances, cerebral blood flow is regulated by cerebral autoregulation across various arterial pressures[26][27]. Patients with intracranial aneurysms often have impaired autoregulation, leading to pressure-dependent cerebral perfusion[28][29].In this vulnerable population, notable fluctuations in blood pressure can result in episodes of inadequate cerebral perfusion. It is noteworthy that we observed a significantly greater reduction in systolic blood pressure within the hypoperfusion group compared to the non-hypoperfusion group (67.61 vs. 58.47 mmHg; P = 0.037), with no significant variance in systolic blood pressure elevation (P = 0.342). This unbalanced pattern suggests that during endovascular surgery, the primary mechanism through which blood pressure variability compromises cerebral perfusion is through hypotension rather than hypertensive surges. This finding aligns with the infrequency of clinically significant hypertensive episodes in our study cohort (median systolic blood pressure elevation equals 0 mmHg).

Hypertension amplifies the effect of BPV: effect modification

A key finding of this study is the significant multiplicative interaction between hypertension and intraoperative SBP variance (interaction a*OR* 1.003, 95% *CI* 1.000–1.007; *P* for interaction = 0.049; likelihood-ratio test *P* = 0.022).The clinical relevance of this statistical interaction was evident in subgroup analyses: hypertension was strongly associated with PI > 1.1 among patients with above-median SBP variance (a*OR* 20.68, 95% *CI* 2.25–190.49; *P* = 0.007), but showed no association below the median (a*OR* 1.26, 95% *CI* 0.35–4.50; *P* = 0.724). Tertile-based analyses yielded concordant results, with significant associations in the second and third tertiles but not in the lowest tertile.Mechanistically, chronic hypertension shifts the cerebral autoregulatory curve rightward and narrows the pressure window over which perfusion is maintained[30][31][32][33].Consequently, blood pressure fluctuations

may more readily traverse the impaired autoregulatory range in hypertensive patients. From a clinical perspective, this interaction underscores the importance of maintaining blood pressure stability in hypertensive patients undergoing aneurysm embolization, a group that can be identified preoperatively without the need for additional testing.

Comparison with previous literature

Our findings extend prior work in several ways. First, whereas previous studies of blood pressure management during aneurysm intervention have largely focused on absolute thresholds—such as SBP exceeding 160 mmHg as a risk factor for rebleeding[34],the present study emphasizes the variability of blood pressure as an independent predictor. Second, our observation that DBP variance and absolute baseline DBP did not differ between groups, while SBP- and MAP-related variability metrics trended toward significance, suggests that systolic fluctuation is the more sensitive indicator of perfusion risk in this setting. Third, the near-linear, threshold-free relationship between SBP variance and postoperative PI > 1.1 (*P* for nonlinearity = 0.814) indicates that risk increases monotonically with BPV across the entire observed range, arguing against a single safe cutoff and in favor of minimizing fluctuation regardless of absolute level. Discrepancies with studies reporting stronger associations of MAP-based metrics may reflect differences in blood pressure sampling frequency and outcome definitions, rather than true biological differences.

Subgroup findings: the role of age

SBP variance was a significant predictor of PI > 1.1 in patients younger than 65 years (aOR 1.0014, 95% CI 1.000–1.003; *P* = 0.038) but not in older patients, and formal interaction tests did not demonstrate effect modification by age for either hypertension (*P* = 0.495) or SBP variance (*P* = 0.688). This pattern should be interpreted cautiously: the absence of a significant association in the older subgroup may reflect limited statistical power (n = 29) rather than a true biological difference. In addition, within ASA II–III patients, SBP variance retained its association (aOR 1.002, *P* = 0.011) whereas hypertension did not, a divergence that is not fully explained by the overall interaction model. These subgroup results should therefore be considered hypothesis-generating, and we emphasize the primary finding of a significant HTN × SBP variance interaction in the overall cohort.

Threshold effects and clinical application of the nomogram

Curve-fitting analyses revealed a statistically significant threshold for baseline diastolic blood pressure (DBP), with a changepoint at 96 mmHg (likelihood-ratio *P* = 0.006). Above this threshold, the association with postoperative perfusion index (PI) > 1.1 steepened markedly, despite the absence of a significant overall linear effect. While this threshold necessitates external validation, it presents a concrete, clinically actionable target: maintaining baseline DBP below 96 mmHg—or at least preventing significant upward deviation—may confer protection in this population. To facilitate the application of the multivariable model in clinical practice, we developed a nomogram based on hypertension and intraoperative systolic blood pressure (SBP) variance. The nomogram demonstrated an apparent area under the curve (AUC) of

0.706 (95% *CI* 0.599–0.813), and internal validation using 1,000 bootstrap resamples yielded an optimism-corrected AUC of 0.689 (optimism = 0.017) and a bias-corrected Brier score of 0.224, indicating minimal overfitting. Although the discrimination is moderate and not diagnostic in nature, the tool's utility lies in providing an objective, easily calculated risk estimate for postoperative cerebral hypoperfusion at the conclusion of the procedure, which could inform the intensity of postoperative neurological monitoring.

Limitations

Several limitations merit attention. First, this study was conducted at a single center with a modest sample size (n = 89); the wide confidence intervals surrounding subgroup estimates, particularly the adjusted odds ratio (a*OR*) of 20.68, highlight the instability of these stratum-specific estimates. Second, blood pressure variability (BPV) was assessed using intermittent arterial pressure readings taken every 5 minutes, rather than through continuous waveform analysis, which may lead to an underestimation of true short-term variability. Third, postoperative perfusion index (PI) was measured only once, and we did not evaluate longer-term neurological outcomes; thus, the clinical significance of an isolated postoperative PI > 1.1 requires further validation. Fourth, while hypertension was accounted for in multivariable models, the baseline imbalance in hypertension between groups (60.0% vs. 32.4%; absolute standardized difference [ASD] = 0.276) and the potential for residual confounding by unmeasured factors (e.g., patterns of vasoactive drug administration, fluid management, and baseline cerebral hemodynamics) cannot be entirely ruled out. Finally, the nomogram was not externally validated, and its optimism-corrected performance was estimated solely through internal resampling. Future multicenter studies that incorporate continuous blood pressure monitoring, standardized vasoactive drug protocols, external validation of the nomogram, and correlation with functional neurological outcomes are necessary to corroborate these findings.

Conclusions

In conclusion, intraoperative systolic blood pressure (SBP) variance and a history of hypertension are independently associated with postoperative cerebral hypoperfusion (PI > 1.1) following endovascular embolization of intracranial aneurysms. A significant interaction suggests that hypertensive patients are particularly susceptible to fluctuations in blood pressure. Additionally, a threshold effect for baseline diastolic blood pressure (DBP) at 96 mmHg, along with a simple, well-calibrated nomogram derived from these two predictors, may enhance individualized blood pressure management and risk stratification during and after endovascular aneurysm procedures. Confirmation of these findings in larger, externally validated cohorts is necessary.

Declarations

Conflict of interest

The authors declare no conflicts of interest.

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Author Contribution

Author ContributionsAll authors contributed to the conception and design of the study. Material preparation, data collection, and analysis were performed as follows.Y.Q. Liu: Formal analysis — performed all statistical analyses, including univariate and multivariable logistic regression, subgroup and interaction analyses, restricted cubic spline and threshold analyses, and nomogram construction and validation; Writing – original draft — drafted the initial manuscript and all tables and figures; Investigation — prepared the ethics application and obtained approval from the Ethics Committee of the First Affiliated Hospital of Harbin Medical University.P. Wu: Investigation — recruited and enrolled eligible patients and collected clinical and demographic data.X.T. Su: Investigation — performed transcranial Doppler ultrasound monitoring of the middle cerebral artery and acquired perioperative cerebral hemodynamic data; Data curation — organized, verified, and maintained the hemodynamic dataset.H.Y. Ma: Conceptualization — conceived the study idea and defined the research question and study design; Supervision — supervised the study conduct, data interpretation, and manuscript revision; Writing – review & editing — critically revised the manuscript for important intellectual content.All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data Availability

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

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Figures

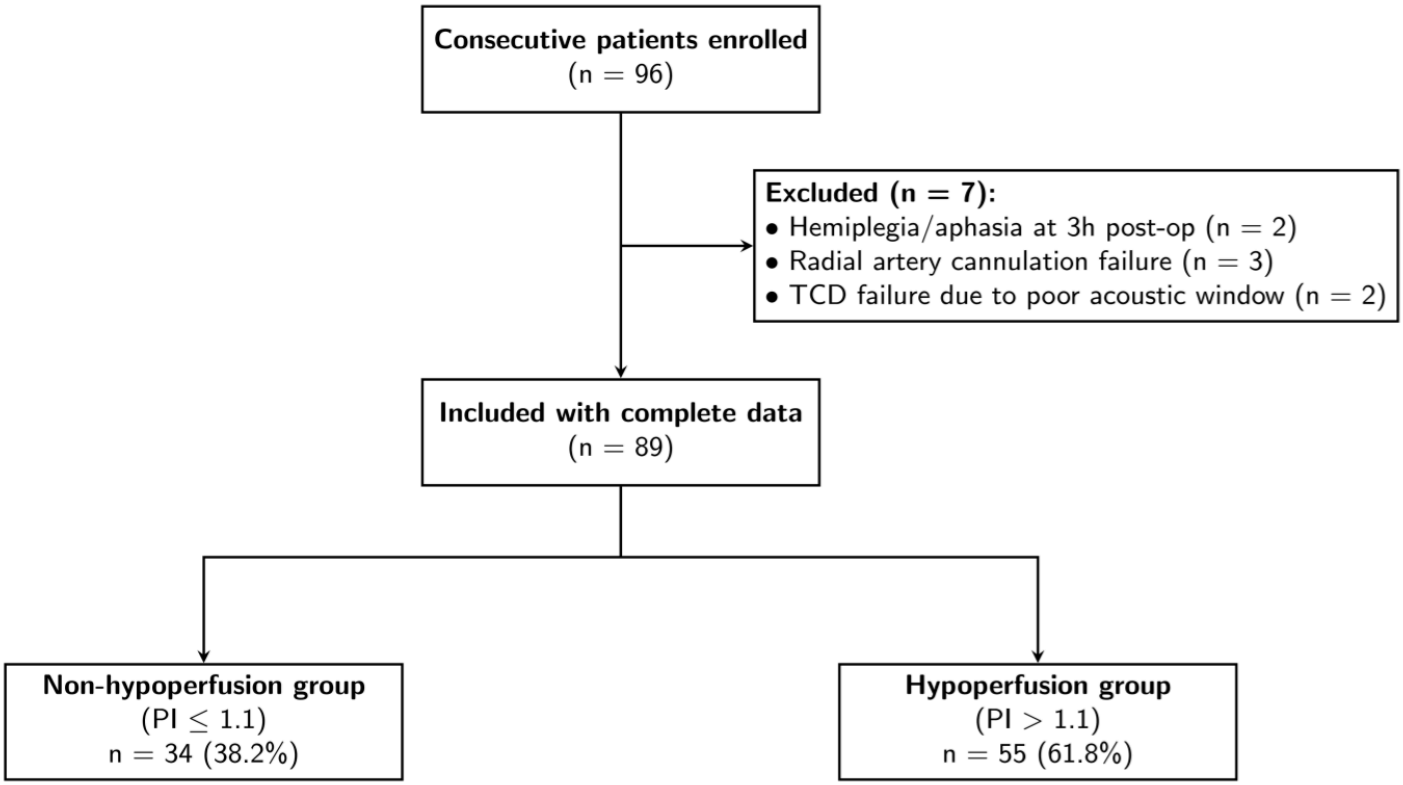


Figure 1

Flow diagram of participant recruitment and inclusion.

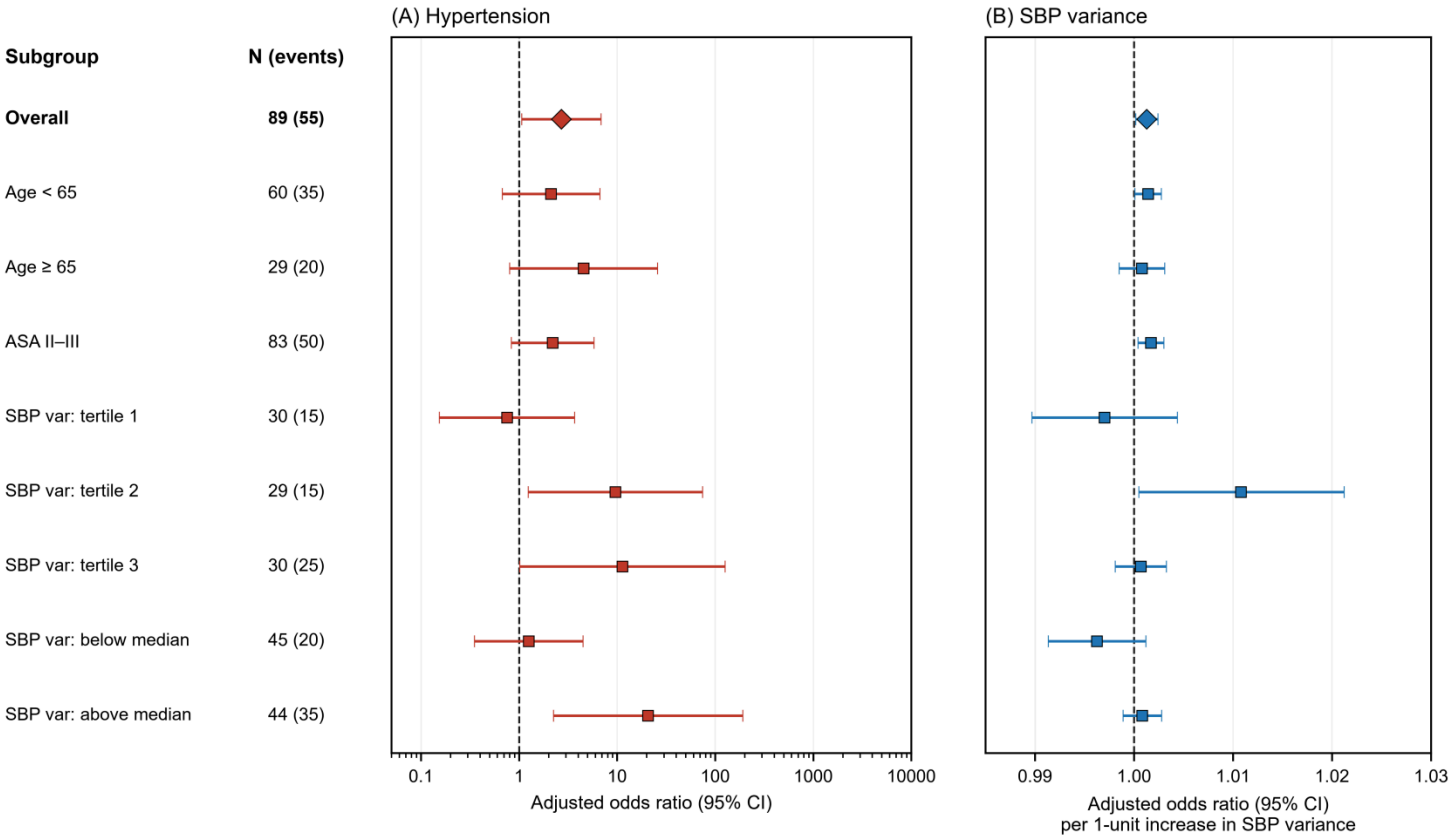


Figure 2

Forest plot of adjusted odds ratios (aORs) for postoperative pulsatility index (PI) > 1.1.

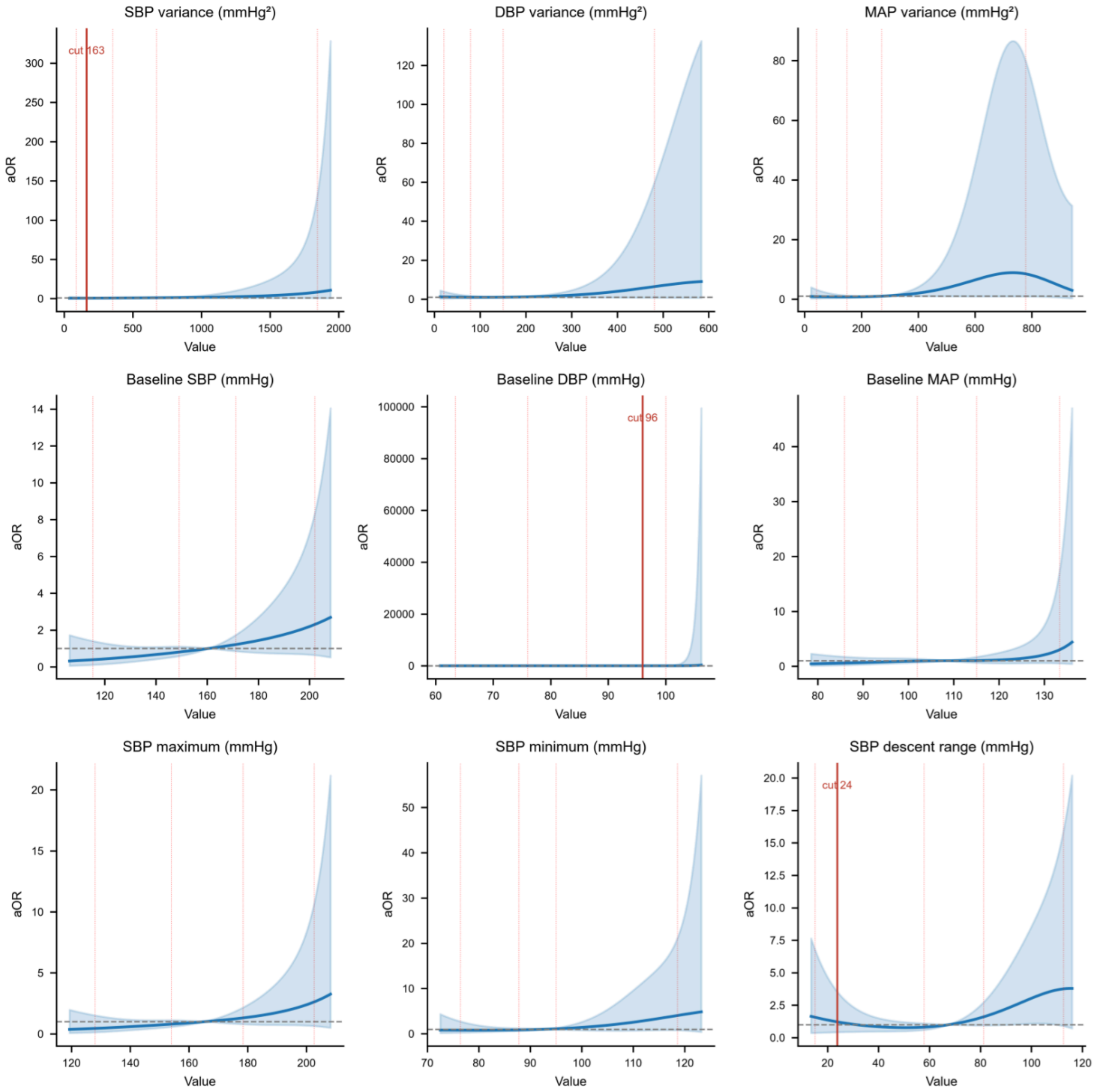


Figure 3

Restricted cubic spline curves of the association between each continuous blood pressure parameter and postoperative PI > 1.1. Solid curves = adjusted odds ratio; shaded areas = 95% *CI* (univariable); red dotted lines = spline knots; red solid line = significant threshold (baseline DBP only, $P < 0.05$)

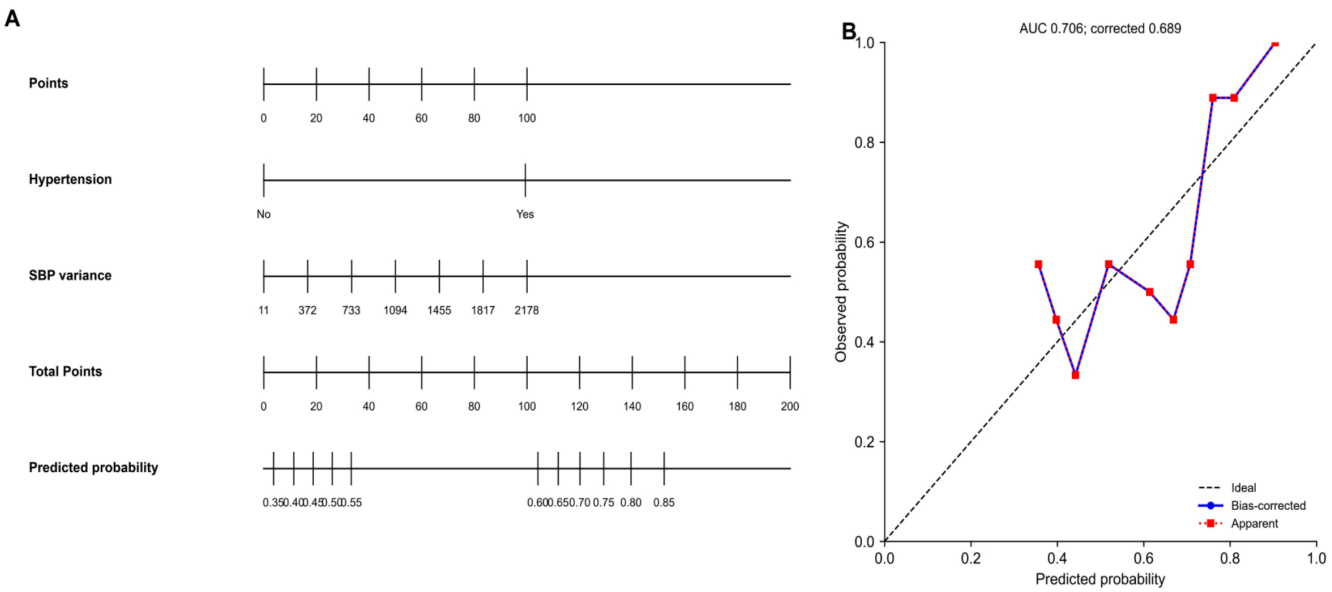


Figure 4

(A) The dashed line represents the ideal calibration where predicted probability equals observed probability. The solid blue line shows the bias-corrected calibration estimated by 1,000 bootstrap resamples; the dotted red line shows the apparent calibration. Predicted probabilities were grouped into deciles; observed probability is the actual proportion of the outcome within each group. Deviations from the ideal line indicate miscalibration.(B). Nomogram for predicting postoperative PI <= 1.1 (cerebral hypoperfusion) after neurosurgery.A multivariate logistic regression model incorporating hypertension (HTN) and intraoperative systolic blood pressure (SBP) variance was used to construct the nomogram. Points are assigned for each predictor according to its regression coefficient (HTN: absent = 0, present = 100; SBP variance: 200 = 0 to 700 = 100 points, linear). The total points are summed and projected onto the bottom axis to obtain the predicted probability of the outcome. The apparent AUC of the model was 0.706 (95% CI 0.599-0.813); after 1,000 bootstrap resamples, the optimism-corrected AUC was 0.689, indicating minimal overfitting. Brier score: 0.208 (apparent) and 0.224 (bias-corrected).

Supplementary Files

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