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Early Blood Pressure Variability and Cerebral Vasospasm Following Aneurysmal Subarachnoid Hemorrhage

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Abstract

Purpose

This study aims to determine whether acute blood pressure variability (BPV) predicts cerebral vasospasm after aneurysmal subarachnoid hemorrhage (aSAH).

Methods

Among 114 consecutive patients with aSAH discharged between May 2024 and May 2025, 85 patients were ultimately included in the analysis. Of these, 26 developed cerebral vasospasm and 59 did not. Systolic and diastolic BPV parameters, including the mean, maximum, minimum, range, standard deviation (SD), coefficient of variation (CV), and successive variation (SV), were calculated from blood pressure measurements recorded every 2 h during the first 24 h of admission. We used logistic regression and receiver operating characteristic curve (ROC) analyses to evaluate the association and predictive value of BPV.

Results

All BPV metrics were significantly associated with cerebral vasospasm ($P < 0.05$). CV and SV for systolic and diastolic blood pressure demonstrated the strongest predictive performance.

Conclusion

Acute-phase BPV was associated with cerebral vasospasm after aSAH occurrence. BPV indices, particularly CV and SV, demonstrated potential utility for identifying patients at increased risk of cerebral vasospasm. These findings require validation in larger prospective studies.

1. Introduction

Blood pressure management is an important component of treatment after aneurysmal subarachnoid hemorrhage (aSAH) [1, 2]. The key to improving the prognosis for patients with SAH lies in faster aneurysm clipping, blood pressure control, and reducing complications [3]. Blood pressure management in SAH is complex, depending on the disease progression and patient factors. This aims to prevent large blood pressure fluctuations and reduce side effects [4]. For instance, blood pressure variability (BPV) during the first 24 h of hyperacute intracerebral hemorrhage is independently linked to neurological deterioration and unfavorable outcomes at 3 months [5]. BPV measurements can evaluate overall variability or extreme blood pressure values. In patients with aSAH, maintaining systolic blood pressure (SBP) at a sufficiently high level to preserve cerebral perfusion may be beneficial [6]. Conversely, some studies have suggested that maintaining a lower maximum SBP (below 118 mmHg) in patients with

unsecured aneurysms is safe and could lead to better functional outcomes at 3 months [7]. To date, optimal blood pressure management in patients with SAH remains controversial.

Previous studies report that angiographic vasospasm affects up to 70% of SAH patients [8]. The condition has received considerable attention because of its association with poor clinical prognosis [9]. Vasospasm development significantly worsens patient prognosis and negatively impacts the use of medical resources. Factors such as hyponatremia, diabetes, and increased mean arterial pressure are associated with cerebral vasospasm [10–14]. However, the potential link between BPV and cerebral vasospasm remains uncertain, specifically during the first 24 h after admission.

Cerebral vasospasm may be identified using clinical findings, such as a decrease in Glasgow Coma Scale (GCS) score of more than 2 points or the development of new focal neurological signs [10]. The diagnostic criteria for vasospasm include a ≥ 33% reduction in the diameter of the involved cerebral artery, as measured by computed tomography angiography (CTA) or digital subtraction angiography (DSA)[15–17]. Recently, with the increasing adoption of point-of-care ultrasound, transcranial Doppler (TCD) has increasingly been used as a bedside monitoring tool in neurocritical care because of its noninvasive nature and accessibility [18]. Although there are currently many non-invasive and convenient methods available, we still rely on head CT scans to confirm whether a patient is experiencing vasospasm. Delayed cerebral ischemia (DCI), which occurs in approximately 20%–30% of patients with aSAH, remains an important contributor to poor prognosis. Early detection and risk prediction of DCI are therefore clinically important for DCI management. The long-term goal in the acute management of aSAH includes the administration of targeted cerebroprotective agents. However, other than nimodipine, none have been identified [19]. Conversely, in many cases, even with standardized blood pressure management, it remains challenging to prevent complications related to SAH. This study aims to clarify the association between early BPV and cerebral vasospasm in patients with aSAH and provide new intervention targets for clinical prevention and treatment.

2. Materials and Methods

2.1 Ethics Approval

The retrospective study was conducted at the First People's Hospital of Lianyungang between May 2024 and May 2025. Ethical approval was obtained from the institutional ethics committee (approval number KY-20250919001-01. The requirement for informed consent was waived because existing medical records were analyzed.

2.2 Patients

The study cohort comprised 114 consecutive patients with aSAH who were discharged from the First People's Hospital of Lianyungang between May 2024 and May 2025. Symptom onset was defined as the time of sudden severe headache or neurological deterioration, as reported by the patient or witnesses. Only patients admitted within 8 h of symptom onset were included. Twenty-nine patients were excluded

because of incomplete blood pressure data, uncertainty regarding symptom onset, or failure to undergo surgical treatment. The remaining 85 patients all underwent aneurysm clipping or embolization within 24 h of admission. A total of 44 patients underwent clipping, and 41 patients received embolization. Finally, 26 patients who developed cerebral vasospasm were included in the vasospasm group, and 59 patients without vasospasm were assigned to the non-vasospasm group. The inclusion criteria were as follows: (1) SAH confirmed by head CT; (2) anterior circulation single aneurysm rupture verified by brain CTA or DSA; (3) time from onset to hospital admission ≤ 8 h; (4) age between 18 and 75 years; Exclusion criteria were as follows: (1) more than 24 h elapsed between the onset of symptoms and the CT scan confirming SAH; (2) secondary SAH caused by vascular malformation, cerebral hemorrhage, or trauma; (3) patients with comorbidities such as heart failure, renal failure, coagulation disorders, and other severe conditions. The aneurysm treatment plan (surgical clipping or endovascular coil embolization) was determined based on the clinical judgment of the treatment team. All patients were managed according to the recommendations of the American Heart Association/American Stroke Association (AHA/ASA) guidelines [18, 20–22]. A flowchart is illustrated in Fig. 1.

2.3 Data Collection

Demographic characteristics, vascular risk factors, admission neurological status, and laboratory findings were collected from medical records. Neurological severity was assessed using GCS, Hunt–Hess Scale, and Modified Fisher Scale.

BPV: Blood pressure management followed international recommendations: SBP was maintained at < 160 mmHg with continuous nicardipine or urapidil. Unless refractory hypotension was present, all patients received nimodipine (oral or IV) as routine prophylaxis against vasospasm. Dexmedetomidine was used for analgesia and sedation during the treatment process. All blood pressure values within the first 24 h after admission were collected and integrated, including measurements recorded in the intensive care unit, during surgical transport, and throughout the intraoperative period. BP was recorded with an automated monitor from the non-paralyzed arm while the patient lay supine. BP values were recorded every 2 h during the first 24 h of admission. The mean, maximum, and minimum values of the systolic and diastolic blood pressure readings mentioned above were calculated. In addition, the following variability indicators were computed: Difference between maximum and minimum blood pressure (ΔBP: BPmax − BPmin); Standard deviation (SD: $\sqrt{1/(n-1)\sum_{(i=1)}^{(n-1)}(BP_i - BP_{mean})^2}$); Coefficient of variation (CV [%]: SD/BPmean × 100%); Successive variation (SV), calculated using the formula SV = $\sqrt{1/(n-1)\sum_{(i=1)}^{(n-1)}(BP_{i+1} - BP_i)^2}$[5,23].

Vasospasm Detection Method: The diagnosis of cerebral vasospasm was established based on non-contrast computed tomography (CT) of the head. Specifically, new-onset hypodensity in a location distinct from the aneurysm-bearing vascular territory, indicative of cerebral infarction, was used as the defining criterion.

2.4 Statistical Analysis

Statistical Package for the Social Sciences (version 26.0) and R (version 4.4.0) software were used. For categorical data, chi-square or Fisher's exact test was used, with results as n (%). For continuous data: a *t*-test (mean ± SD) was used for normal distribution and homogeneous variance; otherwise, the Mann–Whitney U test (median [IQR]) was used. Univariate and multivariate logistic regression were performed. ROC curves and AUC were calculated. A $P < 0.05$ was considered statistically significant.

3. Results

3.1 Comparison of Baseline Characteristics and Indicators in the Derivation Cohort

Several established risk factors for vasospasm, including smoking, hypertension, hemorrhage severity, albumin level, and creatinine level ($P < 0.05$), were associated with vasospasm in our cohort. Baseline and progression characteristics grouped by vasospasm are presented in Table 1.

Table 1
Baseline and progress characteristics between vasospasm and non-vasospasm groups.

Variables	Overall (N = 85)	Non-vasospasm group (N = 59)	Vasospasm group(N = 26)	χ2/t/z	P
Gender				0.032	1.000
Male	71 (83.53%)	49 (83.05%)	22 (84.62%)		
Famale	14 (16.47%)	10 (16.95%)	4 (15.38%)		
Smoking				22.562	<0.001
No	62 (72.94%)	52 (88.14%)	10 (38.46%)		
Yes	23 (27.06%)	7 (11.86%)	16 (61.54%)		
Alcoholconsumption				0.065	0.767
No	70 (82.35%)	49 (83.05%)	21 (80.77%)		
Yes	15 (17.65%)	10 (16.95%)	5 (19.23%)		
Hypertension				35.242	<0.001
No	58 (68.24%)	52 (88.14%)	6 (23.08%)		
Yes	27 (31.76%)	7 (11.86%)	20 (76.92%)		
Diabetes				2.975	0.114
No	71 (83.53%)	52 (88.14%)	19 (73.08%)		
Yes	14 (16.47%)	7 (11.86%)	7 (26.92%)		
Coronaryheartdisease				0.916	0.491
No	74 (87.06%)	50 (84.75%)	24 (92.31%)		
Yes	11 (12.94%)	9 (15.25%)	2 (7.69%)		
Hunt-Hess grade				0.517	0.472
1–3	57 (67.06%)	41 (69.49%)	16 (61.54%)		
4–5	28 (32.94%)	18 (30.51%)	10 (38.46%)		
Modified Fisher grade				9.263	0.002
1–2	64 (75.29%)	50 (84.75%)	14 (53.85%)		
3–4	21 (24.71%)	9 (15.25%)	12 (46.15%)		
GCS score				1.064	0.554
3–8	7 (8.24%)	6 (10.17%)	1 (3.85%)		

Variables	Overall (N = 85)	Non-vasospasm group (N = 59)	Vasospasm group(N = 26)	χ2/t/z	P
9–12	31 (36.47%)	20 (33.90%)	11 (42.31%)		
13–15	47 (55.29%)	33 (55.93%)	14 (53.85%)		
Age	53.24 ± 5.61	52.98 ± 5.86	53.81 ± 5.04	-0.622	0.536
Height	165.19 ± 8.00	165.71 ± 7.00	164.00 ± 9.96	0.909	0.366
Weight	69.41 ± 9.70	69.65 ± 8.96	68.85 ± 11.38	0.351	0.726
BMI	25.42 ± 3.12	25.36 ± 2.96	25.56 ± 3.52	-0.274	0.785
Hemoglobin	87.67 ± 15.93	86.40 ± 16.47	90.54 ± 14.55	-1.106	0.272
Cholesterol	3.91 [3.24;4.75]	3.96 [3.25;4.86]	3.76 [3.17;4.70]	-0.448	0.654
Creatinine	62.80 [54.30;77.10]	58.40 [51.90;67.30]	77.50 [64.95;85.85]	-3.877	< 0.001
Albumin	43.36 ± 6.18	44.99 ± 5.84	39.66 ± 5.37	3.969	< 0.001

The BPV metrics in patients with aSAH of vasospasm revealed a lower SBPmean and SBPmin, a higher SBPmax, SBPmax-min, SBP SD, SBP CV, and SBP SV. The same applies to diastolic blood pressure (DBP) parameters (Table 2).

Table 2
BPV metrics comparison between vasospasm and non-vasospasm groups.

Variables	Overall (N = 85)	Non-vasospasm group (N = 59)	Vasospasm group(N = 26)	z	*P*
SBPmean	133.46 [130.08;134.38]	133.85 [132.92;134.58]	129.50 [128.69;131.46]	-4.131	< 0.001
SBPmax	145.00 [138.00;151.00]	132.00 [128.00;133.00]	150.00 [145.25;152.00]	-5.317	< 0.001
SBPmin	128.00 [122.00;130.00]	129.00 [127.50;130.00]	121.50 [120.00;123.75]	-4.817	< 0.001
SBPmax-min	12.00 [9.00;18.00]	10.00 [9.00;12.00]	19.00 [17.00;22.00]	-4.794	< 0.001
SBP SD	3.37 [2.78;5.30]	3.10 [2.65;3.68]	5.74 [4.94;6.59]	-4.902	< 0.001
SBP CV	0.03 [0.02;0.04]	0.02 [0.02;0.03]	0.04 [0.04;0.05]	-4.907	< 0.001
SBP SV	1.00 [0.82;1.34]	0.92 [0.79;1.05]	1.51 [1.23;1.66]	-5.179	< 0.001
DBPmean	75.08 [73.15;75.85]	75.46 [74.42;75.96]	72.73 [71.25;74.10]	-4.131	< 0.001
DBPmax	78.00 [78.00;79.00]	78.00 [78.00;78.00]	80.00 [78.00;80.00]	-4.494	< 0.001
DBPmin	71.00 [64.00;72.00]	72.00 [70.00;72.00]	63.00 [60.00;66.00]	-4.878	< 0.001
DBPmax-min	7.00 [6.00;16.00]	6.00 [6.00;8.00]	17.00 [12.50;18.75]	-5.028	< 0.001
DBP SD	2.25 [1.80;4.42]	1.98 [1.74;2.64]	4.98 [4.21;5.94]	-5.069	< 0.001
DBP CV	0.03 [0.02;0.06]	0.03 [0.02;0.04]	0.07 [0.06;0.08]	-5.036	< 0.001
DBP SV	0.68 [0.57;1.04]	0.63 [0.55;0.72]	1.22 [1.04;1.30]	-5.093	< 0.001

The Mann–Whitney U test revealed that all SBP and DBP variability indices listed in Table 2 were significantly different between groups *(P* < 0.05).

Univariate analysis demonstrated that all these indices were significantly associated with vasospasm (all *P* < 0.05). After adjustment for smoking, hypertension, Modified Fisher Scale grade, creatinine, and albumin, all except DBPmax remained significant (all *P* < 0.05) (Table 3).

Table 3
Logistic regression analysis on the predictive factors for treatment outcomes.

	Univariate Logistic regression		Multivariate Logistic regression	
	OR(95%CI)	***P***	**OR(95%CI)**	***P***
SBPmean	0.616(0.495, 0.768)	< 0.001	0.528(0.327, 0.747)	0.002
SBPmax	1.321(1.071, 1.630)	0.009	2.453(1.456, 5.106)	0.004
SBPmin	0.699(0.604, 0.810)	< 0.001	0.486(0.251, 0.703)	0.004
SBPmax-min	1.345(1.189, 1.523)	< 0.001	2.968(1.541, 14.613)	0.047
SBP SD	1.112(1.066, 1.161)	< 0.001	1.392(1.155, 2.214)	0.027
SBP CV	1.142(1.083, 1.205)	< 0.001	1.610(1.214, 3.454)	0.048
SBP SV	1.607(1.317, 1.960)	< 0.001	3.081(1.743, 9.359)	0.005
DBPmean	0.604(0.467, 0.781)	< 0.001	0.689(0.466, 0.970)	0.041
DBPmax	1.797(1.244, 2.595)	0.002	1.488(1.010, 2.377)	0.055
DBPmin	0.743(0.658, 0.839)	< 0.001	0.750(0.607, 0.884)	0.002
DBPmax-min	1.318(1.179, 1.473)	< 0.001	1.300(1.127, 1.559)	0.001
DBP SD	2.681(1.800, 3.994)	< 0.001	2.505(1.521, 4.732)	0.001
DBP CV	1.969(1.496, 2.591)	< 0.001	1.858(1.320, 2.860)	0.001
DBP SV	1.826(1.441, 2.314)	< 0.001	1.903(1.376, 3.006)	0.001

3.2 Time-dependent Analysis of Blood Pressure Variability and Vasospasm

In the time-dependent analysis of mean SBP, patients who developed cerebral vasospasm exhibited significantly lower average SBP between 2 and 24 h post-ictus compared with those without cerebral vasospasm (Fig. 2). Similar patterns were observed for mean DBP between 4 and 24 h (Fig. 3). These findings represent associations and do not establish a causal relationship between blood pressure levels and the subsequent development of cerebral vasospasm.

3.3 Nomogram Construction and Validation

All BPV indices demonstrated significant predictive value ($P < 0.001$), with AUCs ranging from 0.654 to 0.854. SBP SV (AUC = 0.854) exhibited the highest predictive ability among SBP indices, followed by SBP SD and SBP CV (both 0.835). For DBP, SD, and SV (all 0.847) performed best. BPV parameters (SD, CV, SV) generally exhibited higher AUCs than mean blood pressure levels (Table 4, Figs. 4–5).

Table 4
ROC distribution between vasospasm and non-vasospasm groups.

SBPmean	AUC	Cutoff	ACC	SEN	SPE	AUC.low	AUC.up	*P*
	0.782	**131.54**	**0.835**	**0.864**	**0.769**	**0.663**	**0.902**	**< 0.001**
SBPmax	0.654	141.50	0.729	0.385	0.881	0.528	0.780	0.010
SBPmin	0.823	125.00	0.835	0.864	0.769	0.718	0.929	< 0.001
SBPmax-min	0.826	16.50	0.871	0.808	0.898	0.711	0.941	< 0.001
SBP SD	0.835	4.30	0.859	0.808	0.881	0.720	0.950	< 0.001
SBP CV	0.835	0.03	0.859	0.808	0.881	0.721	0.950	< 0.001
SBP SV	0.854	1.15	0.835	0.846	0.831	0.756	0.952	< 0.001
DBPmean	0.782	74.27	0.8	0.78	0.846	0.659	0.906	< 0.001
DBPmax	0.767	78.50	0.8	0.654	0.864	0.656	0.877	< 0.001
DBPmin	0.828	69.00	0.847	0.864	0.808	0.715	0.940	< 0.001
DBPmax-min	0.839	11.50	0.859	0.769	0.898	0.726	0.951	< 0.001
DBP SD	0.847	4.07	0.882	0.769	0.932	0.738	0.957	< 0.001
DBP CV	0.842	0.06	0.882	0.769	0.932	0.735	0.948	< 0.001
DBP SV	0.847	0.89	0.882	0.846	0.898	0.729	0.966	< 0.001

4. Discussion

4.1 Early BPV and Cerebral Vasospasm

Despite advances in treatment, cerebral vasospasm remains a major factor contributing to disability and mortality in aSAH. [24–26]. Our study suggests that patients who later developed vasospasm exhibited greater BPV during the acute phase following admission. In our study, smoking, hypertension, Modified Fisher Scale grade, albumin, and creatinine are the main influencing factors for vasospasm. This aligns with reports that smoking triggers vasospasm [27, 28] and with Khan et al. revealing that a history of hypertension and bleeding severity (Modified Fisher Score) are associated with post-SAH vasospasm [29]. Naoya Matsuda et al. suggested that SAH increases ox-LDL and LOX-1, which may play a role in vasospasm pathogenesis [30]. Furthermore, hypercholesterolemia worsens SAH prognosis [31–33], and multiple studies have confirmed that statins improve SAH outcomes [34–36]. Our results are consistent with these findings. High serum creatinine levels on admission independently predict unfavorable outcomes after SAH. Elevated creatinine levels may reflect impaired renal function and systemic endothelial dysfunction, promoting vascular hyperactivity and vasospasm [37]. As a readily available

marker, creatinine can help identify high-risk patients [38]. Our study found that even a slight increase in creatinine, not reaching renal impairment levels, was associated with vasospasm.

4.2 BPV as a Predictor and Potential Mechanisms

Early studies revealed that BPV is a trigger for vascular events and a noninvasive predictor of neurological prognosis in stroke [39]. We found that BPV predicts cerebral vasospasm after SAH, which is consistent with the findings of Corinne Fischer et al. that BPV influences DCI [40]. The mechanism for this may involve the first 24 h after aSAH, which serves as a critical phase for early brain injury (EBI) when brain homeostasis is severely impaired. Higher BPV during this window may exacerbate microcirculatory hypoperfusion or intermittent hyperemia. This intensifies cellular apoptosis and endothelial damage, setting the stage for cerebral vasospasm [41, 42]. Furthermore, our data indicate that both systolic and diastolic BVP, measured within 24 h of admission or EBI phase, are linked to later vasospasm. The early hours after admission, specifically around the 2 h mark when antihypertensive treatment often begins, represent a critical window.

4.3 Patient-Centered Communication and Individualized Targets

Effective clinician-patient communication is essential for successful treatment. Accurate assessment of treatment prognosis and outcomes directly targets the issues of greatest concern to patients and their families. Our findings offer valuable insights for clinicians. When a patient with aSAH exhibits high BPV, vasospasm may occur, which can worsen the neurological prognosis. Nonetheless, these values are intended as general references rather than fixed targets, as they do not apply to every patient. Ultimately, management should be tailored to each patient based on their overall clinical presentation and how it evolves over time.

4. 4 Limitations and Future Outlook

Our findings should be interpreted with caution due to the retrospective, single-center design. The relatively small sample raises the possibility of model overfitting, and external validation is lacking. The BPV analysis mainly focused on the first 24 h after admission; longer-term monitoring could further verify the impact of BPV on SAH-related vasospasm. The retrospective design limited our ability to account fully for several potential confounders. They include antihypertensive treatment intensity, nimodipine administration, sedation strategies, timing of aneurysm repair, and rebleeding events. These factors may have influenced BPV measurements and subsequent clinical outcomes.

5. Conclusions

Early BPV was associated with cerebral vasospasm in patients with aSAH. Although BPV may have value as an available hemodynamic marker, larger prospective studies are required before its clinical utility can be established.

Declarations

Institutional Review Board Statement: The study involving human participants was approved by Institutional Review Board of the First People's Hospital of Lianyungang (protocol code KY-20250919001-01). The study was conducted in accordance with the Declaration of Helsinki.

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Conflicts of Interest: The authors declare no conflicts of interest.

Author Contribution

Conceptualization: YYM, PPZ, XLZ Data curation: LZ, ZYZ, XHZ Formal analysis: YYMFunding acquisition: YYM, PPZ Investigation: LZ, XHZ Methodology: YYM, PPZ, XLZ Project administration: XLZ Resources: XLZ Software: LZ Supervision: XLZ Validation: XHZ, YQY Visualization: YYMWriting – original draft: YYM, PPZ Writing – review & editing: XLZAll authors reviewed the manuscript

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Figures

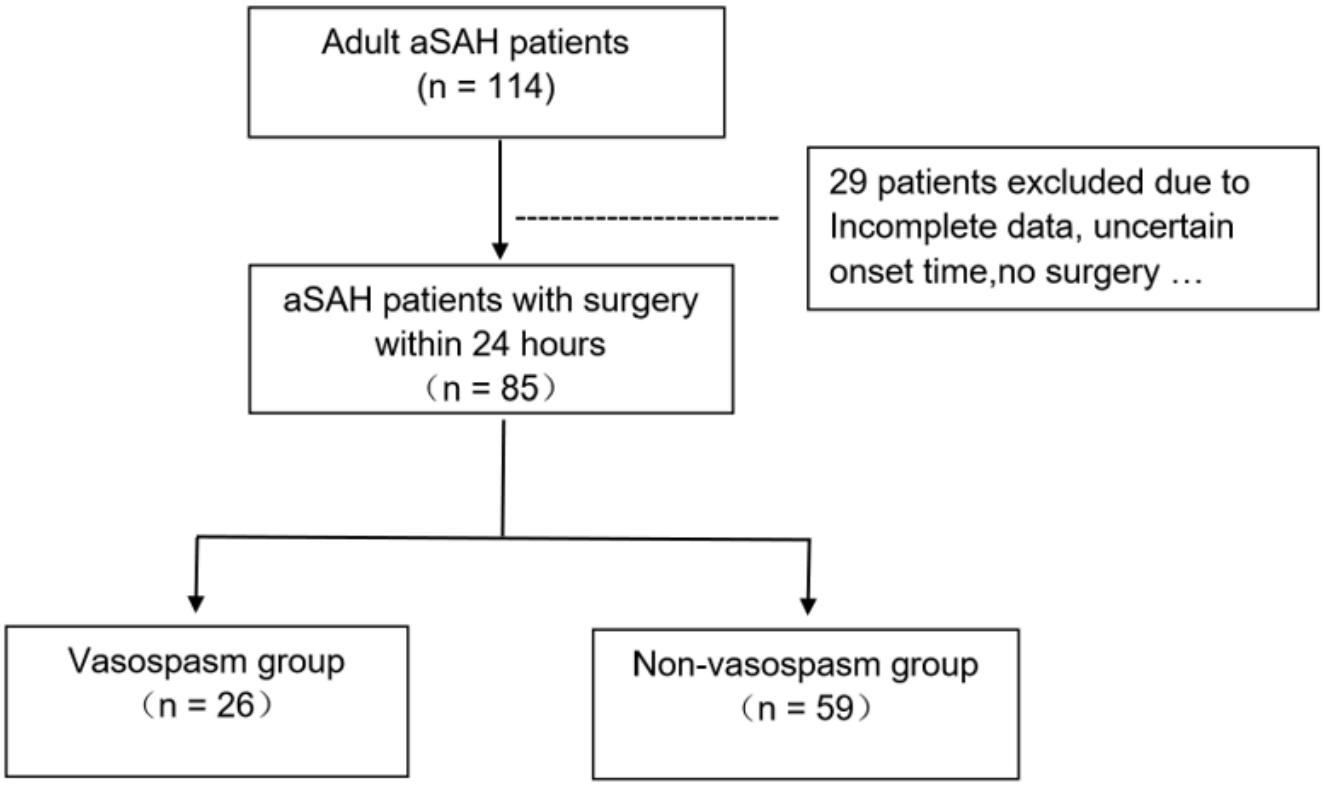


Figure 1

Flowchart between vasospasm and non-vasospasm groups.

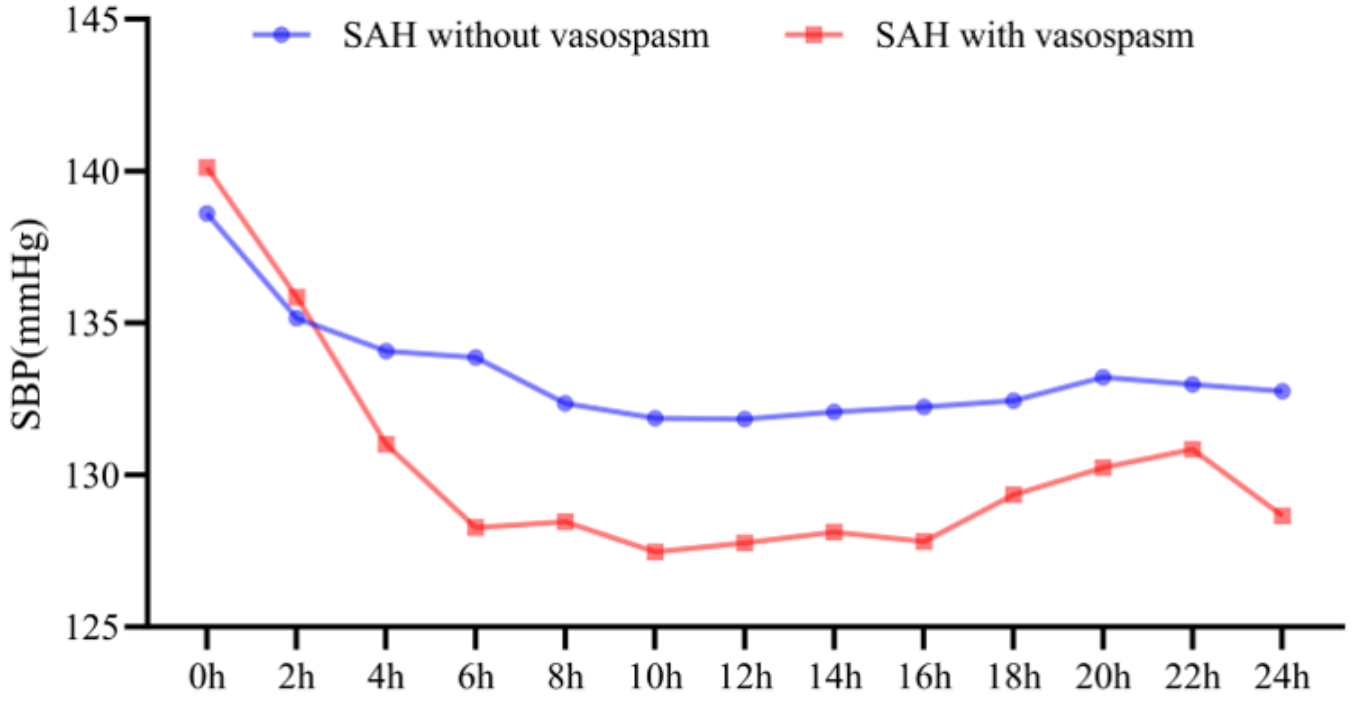


Figure 2

Mean SBP profiles during the first 24 h after admission (blue: without vasospasm; red: with vasospasm).

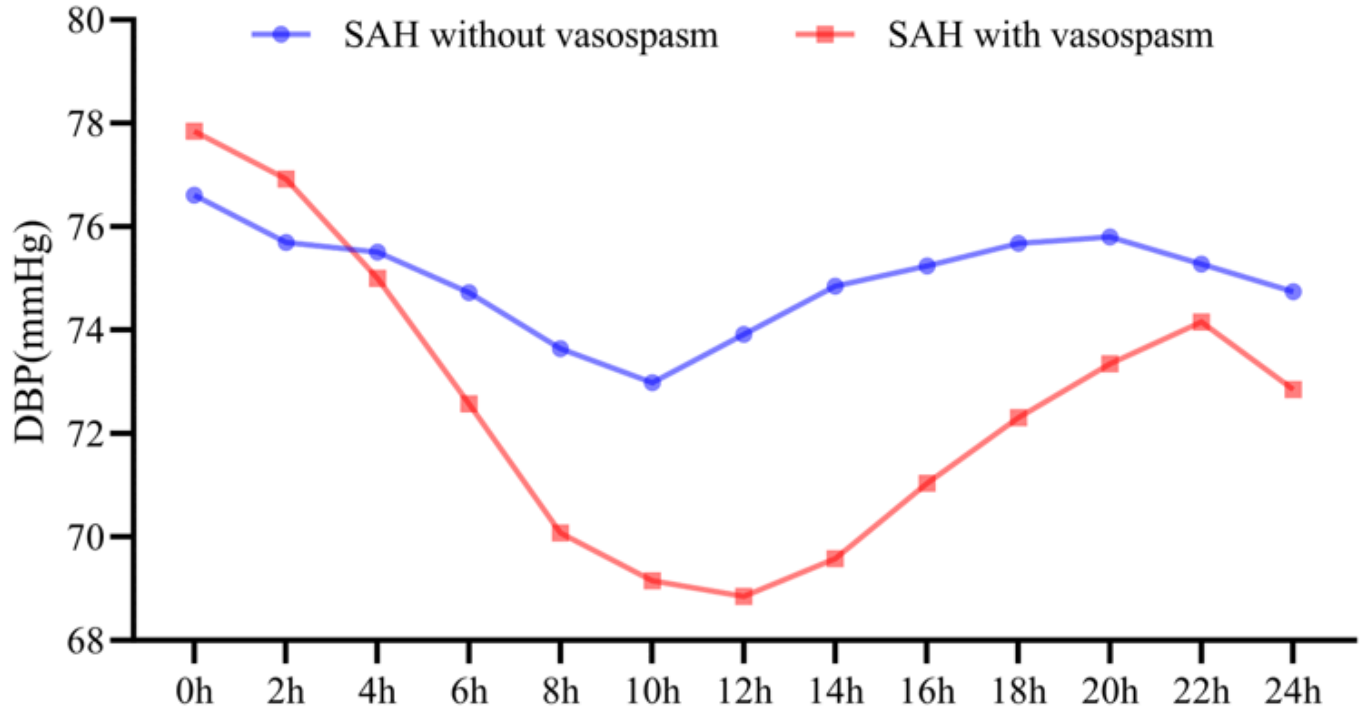


Figure 3

Mean DBP profiles during the first 24 h after admission (blue: without vasospasm; red: with vasospasm).

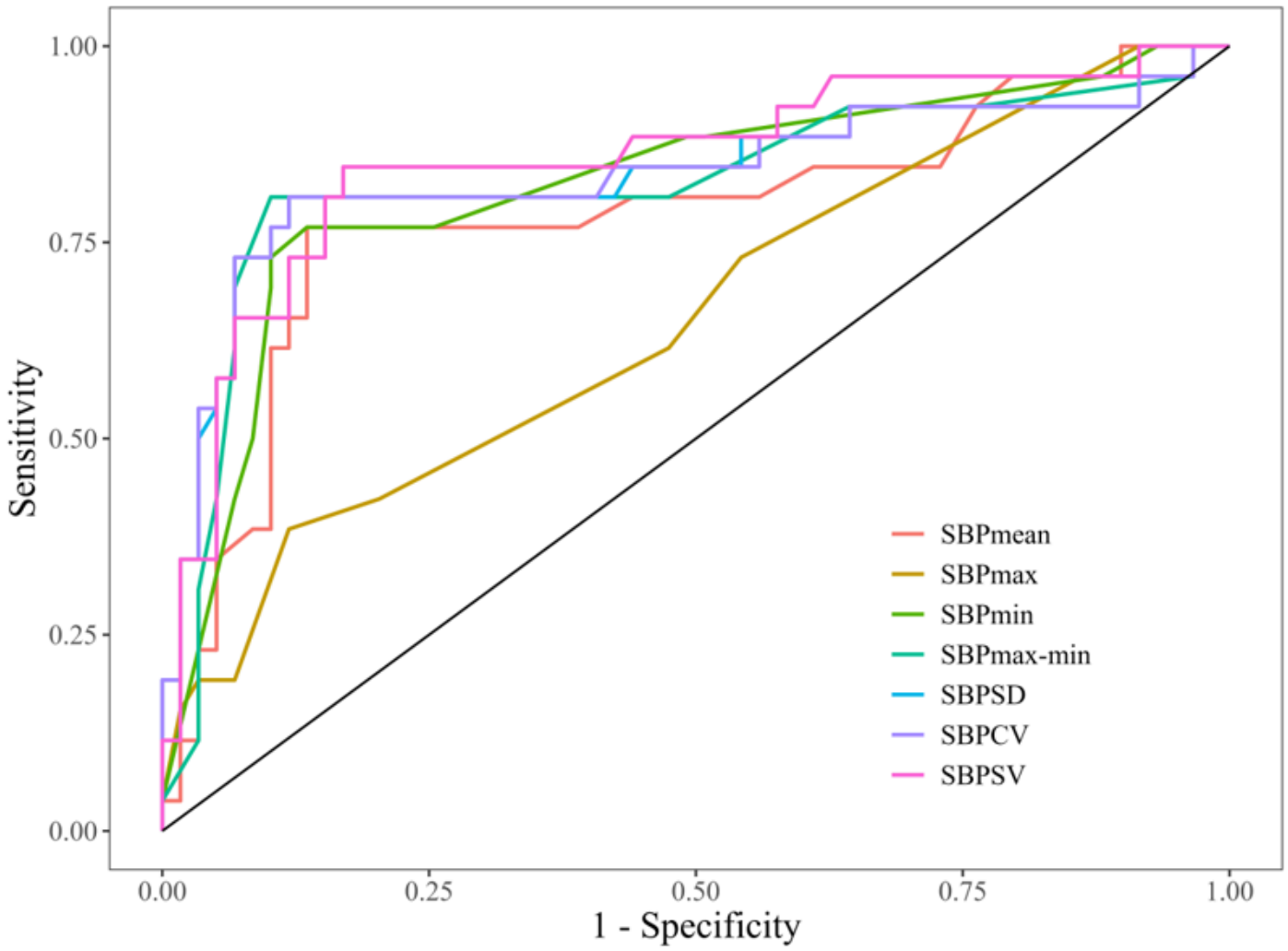


Figure 4

ROC of the 24 h SD of the SBP.

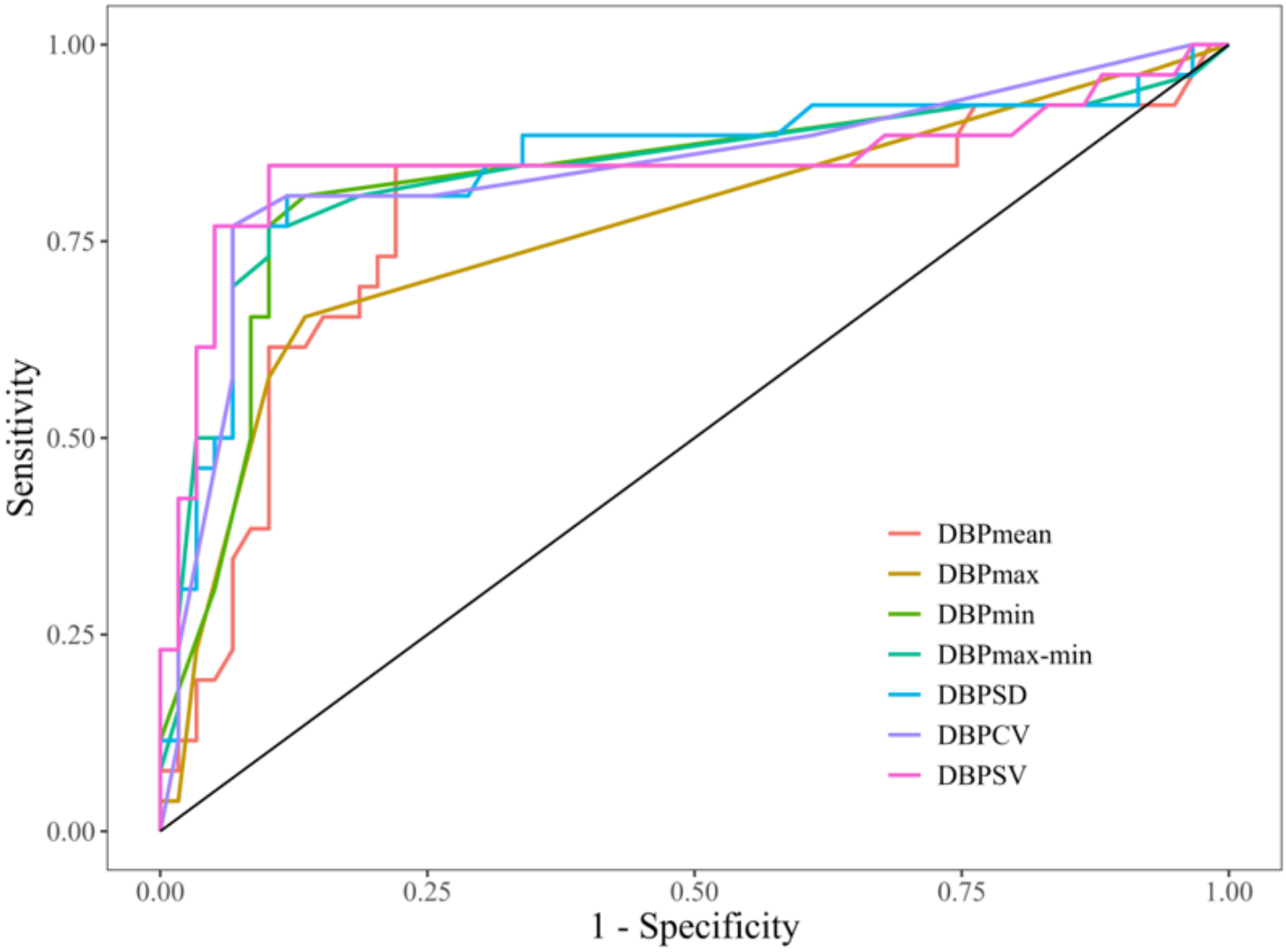


Figure 5

ROC of the 24 h SD of the DBP.