



Special Issue: Current evidence and perspectives for hypertension management in Asia

Mid-term systolic BP variability beyond 24 h after successful recanalization predicts poor outcomes following ischemic stroke

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Received: 18 October 2025 / Revised: 13 February 2026 / Accepted: 28 February 2026 / Published online: 13 March 2026
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Abstract

Although intensive blood pressure (BP) lowering after mechanical thrombectomy (MT) may adversely affect outcomes, the prognostic significance of post-recanalization BP variability remains unclear. This study aimed to evaluate the association between systolic blood pressure (SBP) variability after successful recanalization and 90-day functional outcomes following MT. Among 342 consecutive patients who underwent MT between May 2014 and June 2025, 280 patients who achieved successful recanalization were included in this retrospective analysis. SBP was recorded from immediately after recanalization up to 72 h thereafter. BP variability indices, including variability independent of the mean (VIM), time rate, and coefficient of variation, were calculated. The primary outcome was defined as a modified Rankin Scale (mRS) score of 4–6 at 90 days. Associations between SBP variability and outcomes were assessed using multivariable logistic regression models. Of the 280 patients, 104 (37.1%) experienced poor functional outcomes. Higher SBP variability was significantly associated with unfavorable outcomes. In time-segmented analyses, only SBP variability during the 24–72-h period remained significantly associated with poor outcomes (aOR per 10-unit increase in VIM [VIM/10], 1.89; 95% CI, 1.20–3.06, $p = 0.005$). Sensitivity analyses excluding patients who received antihypertensive therapy during 24–72 h and those with symptomatic intracranial hemorrhage confirmed the robustness of the association between 24–72 h VIM and poor outcomes (aOR VIM/10, 1.94; 95% CI, 1.26–3.11, $p = 0.003$). In conclusion, these findings highlight the clinical importance of stabilizing BP beyond the first 24 h after recanalization.

Keywords Blood pressure variability · Endovascular thrombectomy · Functional outcome · Implemental hypertension

Introduction

The optimal blood pressure (BP) management strategy following mechanical thrombectomy (MT) for patients with acute ischemic stroke remains unclear, especially in those who achieve successful recanalization. Previous

observational studies have reported that lower systolic blood pressure (SBP) is independently associated with favorable functional outcomes [1, 2]. However, recent randomized controlled trials (RCTs [3–6]) have suggested that intensive SBP-lowering strategies within the first 24 h may have a neutral or even detrimental effect. The reasons for this discrepancy remain uncertain.

Previous studies mainly focused on BP levels or blood pressure variability (BPV) within the first 24 h after MT, and showed that higher SBP or greater SBP variability is associated with unfavorable outcomes [7]. However, cerebral autoregulation remains impaired for several days after acute ischemic stroke [8], and microvascular dysfunction, the “no-reflow” phenomenon [9, 10], reperfusion injury, and brain edema may continue to evolve during the early subacute phase after recanalization [11]. In patients treated with reperfusion therapy, a lower mean BP during the 24–72-h period was reportedly associated with better functional outcomes and

Supplementary information The online version contains supplementary material available at <https://doi.org/10.1038/s41440-026-02608-6>.

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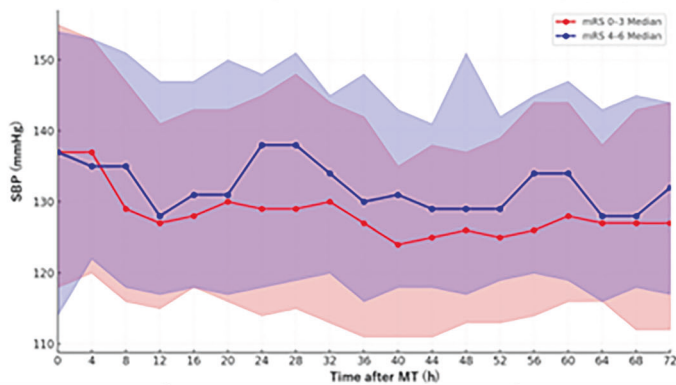
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Graphical Abstract

Mid-term Systolic BP Variability Beyond 24 Hours After Successful Recanalization Predicts Poor Outcomes

Median systolic blood pressure (SBP) over 72-h after successful recanalization following mechanical thrombectomy (MT)



$$VIM = k \times \frac{SD}{(\text{Mean SBP})^\beta}$$

- ✓ Participants: 280 MT patients
- ✓ The primary outcome: modified Rankin Scale (mRS) score of 4–6 at 90 days.
- ✓ Multivariable logistic regression analysis was performed using poor outcome as the dependent variable.

	Model 1 [[aOR,(95% CI)]]	P value	Model 2 [[aOR,(95% CI)]]	P value	Model 3 [[aOR,(95% CI)]]	P value
24-72-h VIM/10	1.69 (1.20-2.45)	0.002	1.83 (1.20-2.90)	0.008	1.94 (1.26-3.11)	0.003

Each IQR increase in VIM was associated with an average worsening of 0.44 points in the mRS score.

Point of view

- Clinical relevance
Mid-phase systolic blood pressure variability (24–72 h after successful recanalization) is independently associated with poor functional outcomes, highlighting the importance of blood pressure stabilization beyond the first 24 h after mechanical thrombectomy.
- Future direction
Prospective interventional studies are warranted to determine whether therapeutic strategies targeting blood pressure variability during the 24–72 h post-recanalization period can improve neurological outcomes.
- Consideration for the Asian population
Because blood pressure profiles, vascular stiffness, and stroke characteristics differ across ethnicities, validation of post-thrombectomy blood pressure variability management in Asian populations is clinically important.

a lower risk of hemorrhagic complications, highlighting the clinical importance of BP control beyond the first 24 h [12]. More recently, MT cohorts with frequent BP monitoring up to 72 h have shown that both BP levels and BPV over this period are related to functional outcomes [13].

Based on these clinical and pathophysiological considerations, we hypothesized that BPV in the mid-phase (defined as 24–72 h) after successful recanalization would be independently associated with poor functional outcomes. We focused on the first 72 h, because this period corresponds to the acute and early subacute phases, during which reperfusion-related injury is most active, and BP is routinely monitored with sufficient frequency to allow accurate assessment of BPV indices. The aim of this study was to evaluate whether BPV, particularly variability independent of the mean (VIM), during 24–72 h after successful recanalization (defined as modified treatment in cerebral infarction [mTICI] grade ≥2b), is associated with 90-day functional outcomes in acute ischemic stroke.

Subjects and methods

Study population and study protocol

Acute ischemic stroke patients were selected from the Stroke Database Registry, a prospective registry of acute stroke patients admitted to our single center in Japan. We conducted a retrospective analysis of 342 consecutive patients with large vessel occlusion (LVO) who were treated with MT and who were hospitalized between May 2014 and June 2025. Inclusion criteria in this study were: (1) age ≥ 18 years; (2) successful reperfusion of the occluded artery, defined as an mTICI grade of $\geq 2b$; and (3) availability of SBP measurements for the 72-h period following successful reperfusion. Exclusion criteria were: (1) contraindications to antihypertensive therapy; (2) patients who underwent more than one MT procedure during hospitalization; (3) pre-stroke functional disability, defined as a modified Rankin Scale (mRS) score of 4–5; and (4) patients for whom post-stroke 3-month mRS scores could not be obtained. This study was approved by the Ethics Committee of Kagoshima Medical Center (approval no. 2024-03). The committee waived the requirement for patient consent and instead provided those patients from whom data were collected with the opportunity to opt out of the study. The study protocol adhered to the ethical guidelines of the Declaration of Helsinki, as revised in 1975.

Data collection and blood pressure measurement

Clinical data were obtained from our institutional stroke database and electronic medical records. Collected variables included: sex, age, pre-stroke mRS score, body mass index (BMI), systolic and diastolic BP on admission, initial National Institutes of Health Stroke Scale (NIHSS) score, current smoking status (defined as active smoking within the past year), heavy alcohol consumption (defined as ≥ 3 servings of sake per day, equivalent to ≥ 60 g of pure alcohol [14]), previous history of any type of stroke (ischemic stroke, intracerebral hemorrhage, or transient ischemic attack), use of anticoagulants prior to onset (including type), use of antiplatelet agents prior to onset, presence of hypertension (defined as the use of anti-hypertensive agents before admission, a history of diagnosis of hypertension but not on any medication, or use of anti-hypertensive agents at discharge), presence of diabetes mellitus (defined as the use of oral hypoglycemic agents or insulin, or performing dietary therapy before admission, a history of diagnosis of diabetes but not on any medication, or glycosylated hemoglobin level (National Glycohemoglobin Standardization Program) $\geq 6.5\%$ on admission), presence of dyslipidemia (defined as the use of cholesterol-lowering drugs or performing dietary therapy before

admission, a history of diagnosis of hypercholesterolemia but not on any medication, or serum LDL-cholesterol ≥ 140 mg/dL on admission), presence of atrial fibrillation (AF) (either known prior to admission or newly diagnosed during hospitalization), and laboratory values. Neuroimaging findings were also obtained, including the time of magnetic resonance imaging (MRI), time from hospital arrival to MRI, Alberta Stroke Program Early CT Score (DWI-ASPECTS) [15], infarct location, presence and site of major cerebral artery occlusion, and ischemic stroke subtype according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification [16]. Data regarding thrombolytic therapy included whether or not it was administered, and the time from arrival to treatment initiation. For MT, we recorded: timeline, procedure performed [combined technique only, a direct aspiration first pass technique (ADAPT) only, Stent retriever only], number of catheter passes, and final mTICI grade [17]. Evaluated items also included the presence of any intracerebral hemorrhage (ICH), parenchymal hematoma (PH) type 1 or 2 based on the European Cooperative Acute Stroke Study (ECASS) classification of hemorrhagic transformation [18], symptomatic ICH (sICH) (defined according to ECASS II criteria as any ICH with a ≥ 4 -point increase from baseline NIHSS score) [18], subarachnoid hemorrhage (SAH), vasospasm (defined as any degree of vasospasm within the treated vessel), and embolization in the same or a new/distal territory after thrombectomy [19]. Additional assessments included NIHSS score at 24 h after MT, early neurological deterioration (END, defined as a worsening of ≥ 2 points in the NIHSS score within 72 h after MT) [20, 21], MRI findings at 24–48 h, presence of a midline cranial shift (defined as a ≥ 5 mm deviation on FLAIR imaging at 24–48 h) [22], and Alberta stroke program early CT score (ASPECTS). The clinical outcomes assessed included the mRS score at 90 days and 90-day mortality. MT procedures were performed using a Siemens ARTIS zee biplane angiography system (Siemens, Erlangen, Germany) under conscious sedation. Cerebral digital subtraction angiography and MT were performed via femoral or brachial artery puncture. For anterior circulation lesions, an 8-Fr balloon-guide catheter was used, while for the posterior circulation, a 5-Fr or 6-Fr Fubuki guide sheath (Asahi Intecc Co., Ltd., Aichi, Japan) was employed. Thrombectomy was principally performed using a stent retriever, an aspiration catheter, or a combination of both. The choice of retrieval technique was at the discretion of the neurointerventionalist. Data on pre-hospital use of antithrombotic medications, smoking status, alcohol consumption, and stroke history were obtained through patient interviews or from caregivers when necessary.

BP was measured non-invasively using the Nihon Kohden CSM-1701 Lite Scope G7 or other automatic BP

monitors certified by the Association for the Advancement of Medical Instrumentation (AAMI) and the European Society of Hypertension (e.g., Omron 3 Series Upper Arm [BP7150], Omron 5 Series [BP7200]), all of which are supported by grade A clinical validation. In general, patients with acute stroke were admitted to the stroke unit after MT, where BP was recorded every 15 min during the first 2 h, every 2–4 h thereafter up to 24 h, and every 4 h after the initial 24-h period. During neurological monitoring, BP was measured using automated bedside monitors under routine clinical conditions, predominantly in the supine or semi-recumbent position. BP management was at the discretion of the attending neurologist. Data on target SBP and antihypertensive use within the first 72 h post-recanalization were extracted from the patients' clinical records. To avoid the influence of measurement error, only SBP values between 30 and 300 mmHg were included in the analysis. Maximum and minimum SBP values were calculated by averaging the two highest and two lowest readings from bilateral upper arm measurements, to minimize bias from outliers. No strict lower SBP threshold was predefined. BP management focused on avoiding hypotension while preventing excessive hypertension, with individualized adjustments based on clinical and neurological status.

Blood pressure management after mechanical thrombectomy followed a standardized institutional protocol. During the first 24 h after successful recanalization, the target SBP was generally set at ≤ 160 mmHg, based on previously published literature [23, 24]. During the first 24 h after recanalization, intravenous nicardipine, a calcium channel blocker, was used as the first-line antihypertensive agent when needed. Nicardipine was exclusively administered as a continuous intravenous infusion; bolus dosing was not used in any patient. The infusion was initiated when SBP persistently exceeded 160 mmHg, and was tapered or discontinued when SBP persistently fell to ≤ 120 mmHg, at the discretion of the treating physician. Among the 280 patients, continuous intravenous nicardipine infusion within the first 24 h after recanalization was administered in 43 patients (15.3%). Between 24 and 72 h after recanalization, antihypertensive management included intravenous nicardipine, oral calcium channel blockers (amlodipine), and oral selective β 1-blockers (bisoprolol), depending on the patient's clinical condition. During this period, a continuous intravenous nicardipine infusion was used in 35 patients (12.5%) under the same regimen as described above. Oral antihypertensive agents were administered in 34 patients (12.1%), including amlodipine in all 34 patients and bisoprolol in five patients, i.e., five patients received both amlodipine and bisoprolol concomitantly. In patients with sICH, SBP was strictly controlled with a target of ≤ 140 mmHg, in accordance with guideline-based management. The same SBP target (≤ 140 mmHg) was also applied

in patients with minor SAH. In contrast, BP management was individualized in patients with asymptomatic ICH classified as hemorrhagic infarction type 1 or 2 (HI1 or HI2 according to the ECASS classification). In these cases, SBP was generally maintained below 160 mmHg in the absence of neurological deterioration or radiological evidence of hematoma expansion. If neurological worsening or hematoma enlargement was suspected, stricter BP control was implemented at the discretion of the treating physician.

SBP was recorded at predefined intervals from 0–72 h after recanalization. BPV was assessed using standard deviation (SD), coefficient of variation (CV), VIM, and time rate (TR). VIM reflects BPV independent of mean SBP, while TR captures the magnitude and speed of successive SBP fluctuations over time. The detailed definitions and calculation methods for all BPV indices are provided in Supplementary Text 1.

Imaging protocols

At our hospital, patients with suspected stroke basically undergo MRI at admission. Thereafter, patients with LVO undergo computed tomography (CT) immediately after MT. Patients who undergo MT routinely undergo a second MRI 24 ± 12 h after admission. The CT and MRI platform parameters used in this study are described in Supplementary Text 2.

Statistical analysis

Statistical analyses were performed using JMP version 15 software (SAS Institute, Cary, NC, USA). The primary outcome of this study was a poor functional outcome at 90 days (mRS score of 4–6). As secondary analyses, we also examined the association between VIM and sICH, Δ DWI-ASPECTS [(DWI-ASPECTS 24 h after MT) – (initial DWI-ASPECTS)], and midline shift. SBP variability parameters (SD, CV, VIM, and TR) were analyzed as continuous variables and assessed using univariate and multivariable logistic regression models.

First, we conducted univariate analyses comparing patients with poor outcomes to those with favorable outcomes in terms of baseline characteristics, treatment parameters, SBP levels, and SBP variability indices. Categorical variables were compared using the chi-squared test or Fisher's exact test, while continuous variables were analyzed using the Mann–Whitney U test. Next, to examine associations between SBP indices and clinical outcomes, we performed multivariable logistic regression analysis using poor outcome as the dependent variable. Based on a previous study [25], we constructed three models: Model 1: adjusted for age and sex; Model 2: adjusted for age, sex, NIHSS score at admission, and DWI-ASPECTS 24 h after

MT; and Model 3: adjusted for age, sex, smoking status, diabetes, hypertension, hyperlipidemia, baseline NIHSS score, DWI-ASPECTS at 24 h, presence of sICH, mTICI grade, and mean SBP from 0–72 h. Δ DWI-ASPECTS, being a continuous variable, was assessed using multiple linear regression to evaluate its association with outcomes. We calculated variance inflation factors (VIFs) to avoid multicollinearity among explanatory variables. Variables with high collinearity (VIFs ≥ 10) were excluded from the models. To identify the time windows during which BPV was most strongly associated with outcomes, we divided the 72-h period into four time segments (0–8 h, 8–16 h, 16–24 h, and 24–72 h) and evaluated the association between the SBP indices of each segment and poor outcomes using the same three logistic regression models. To account for potential confounding by antihypertensive treatment and complications, we conducted sensitivity analyses excluding patients who received antihypertensive therapy between 24 and 72 h after recanalization, and patients who developed sICH. These analyses also used multivariable logistic regression models with adjustment for the same covariates as above. Furthermore, we examined the association between 24–72 h VIM and an mRS score of 4–6 at 90 days using a quartile-based approach. Patients were categorized into four groups according to the distribution of 24–72 h VIM: Quartile 1 (0 to <25th percentile), Quartile 2 (25th percentile to <median), Quartile 3 (median to <75th percentile), and Quartile 4 (≥ 75 th percentile). To address potential bias related to the frequency of BP measurement, we performed additional analyses accounting for the number of SBP measurements obtained during the 24–72-h period after recanalization. The number of BP measurements during this period was included as an additional covariate in multivariable logistic regression models. Furthermore, stratified analyses were conducted according to the median number of BP measurements during 24–72 h

post-MT to evaluate the robustness of the association between BPV and clinical outcomes across different monitoring intensities. Finally, to explore the relationship between overall SBP variability and directional SBP changes, we calculated the Spearman correlation coefficient between VIM and TR. A negative correlation suggested the presence of intermittent sharp fluctuations (spikes) despite an overall declining trend in SBP. A p -value of <0.05 was considered statistically significant in all analyses.

Results

Supplementary Figure 1 shows the study flowchart. Of the 342 patients screened, 297 met the inclusion criteria. After excluding three patients who underwent MT more than once, 13 patients with pre-stroke mRS scores of 4–5, and one patient with missing mRS data at 3 months, a total of 280 patients were included in the final analysis. Their baseline characteristics, procedural results, and outcomes stratified by 90-day outcome status (good vs poor) are presented in Supplementary Table 1. Thrombectomy techniques were not significantly associated with 90-day functional outcomes (Supplementary Table 1). The proportion of patients with poor versus good outcomes did not differ significantly across techniques (combined technique: 68.9% vs. 60.3%, $p = 0.216$; ADAPT only: 26.7% vs. 33.3%, $p = 0.317$; stent retriever only: 4.4% vs. 6.4%, $p = 0.583$). SBP and BPV indices are summarized in Supplementary Table 2, and the time-course of SBP medians is shown in Fig. 1.

At 3 months, poor functional outcomes (mRS score 4–6) were observed in 104 patients (37.1%), and good outcomes (mRS score 0–3) in 176 patients (62.9%). Among BPV indices, VIM and SD were significantly higher in the poor outcome group, whereas CV and TR did not differ

Fig. 1 Median systolic blood pressure (SBP) over 72 h after successful recanalization in patients with good outcomes (mRS score 0–3, red line) and poor outcomes (mRS score 4–6, blue line). Shaded areas represent interquartile ranges (IQR)

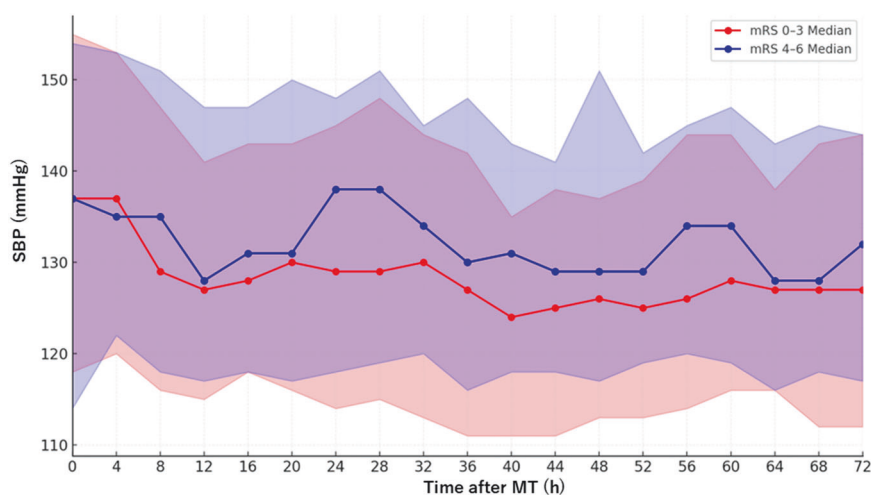


Table 1 Correlation between VIM and mRS scores of 4–6 at 90 days

	Model 1 [(aOR, (95% CI)]	P value	Model 2 [(aOR, (95%CI)]	P value	Model 3 [(aOR, (95% CI)]	P value
0–8 h VIM/10	2.56 (0.91–7.29)	0.073	2.01 (0.60–6.73)	0.255	2.62 (0.70–10.0)	0.152
8–16 h VIM/10	1.12 (0.59–2.12)	0.719	1.01 (0.49–2.06)	0.973	1.06 (0.48–2.27)	0.891
16–24 h VIM/10	0.66 (0.36–1.18)	0.165	0.56 (0.27–1.10)	0.096	0.63 (0.29–1.29)	0.207
24–72 h VIM/10	1.72 (1.23–2.45)	0.001	1.97 (1.30–3.08)	0.001	1.89 (1.20–3.06)	0.005

Model 1: age, sex. Model 2: age, sex, NIHSS score, DWI-ASPECT 24 h after MT. Model 3: age, sex, smoking, diabetes, hypertension, hyperlipidemia, initial NIHSS score, DWI-ASPECT 24 h after MT, sICH, mTICI, 0–72 h mean SBP

aOR adjusted odds ratio, ASPECTS Alberta Stroke Program Early Computed Tomographic Score, CI confidence interval, DWI diffusion-weighted imaging, MT mechanical thrombectomy, mRS modified Rankin Scale, mTICI modified Thrombolysis in Cerebral Infarction, NIHSS National Institutes of Health Stroke Scale, SBP systolic blood pressure, sICH symptomatic intracranial hemorrhage, VIM variability independent of the mean, VIM/10 VIM per 10-unit increase

Table 2 Correlation between VIM and mRS scores of 4–6 at 90 days, excluding patients with antihypertensive therapy within 24–72 h after recanalization and sICH patients

	Model 1 [(aOR, (95% CI)]	P value	Model 2 [(aOR, (95% CI)]	P value	Model 3 [(aOR, (95% CI)]	P value
24 to 72-h VIM/10	1.69 (1.20–2.45)	0.002	1.83 (1.20–2.90)	0.008	1.94 (1.26–3.11)	0.003

Model 1: age, sex. Model 2: age, sex, NIHSS score, DWI-ASPECTS 24 h after MT. Model 3: age, sex, smoking, diabetes, hypertension, hyperlipidemia, initial NIHSS score, DWI-ASPECTS 24 h after MT, mTICI, and 24–72 h mean SBP

aOR adjusted odds ratio, ASPECTS Alberta Stroke Program Early Computed Tomographic Score, CI confidence interval, DWI diffusion-weighted imaging, MT mechanical thrombectomy, mRS modified Rankin Scale, mTICI modified Thrombolysis in Cerebral Infarction, NIHSS National Institutes of Health Stroke Scale, SBP systolic blood pressure, sICH symptomatic intracranial hemorrhage, VIM variability independent of the mean, VIM/10 VIM per 10-unit increase

significantly. In group-wise comparisons, SBP remained significantly higher in the poor outcome group during the mid-phase after recanalization (16–52 h) (Fig. 1, Supplementary Table 2). In multivariable logistic regression analyses (Supplementary Table 3), 0–72 h VIM per 10-unit increase (VIM/10) was independently associated with a poor outcome (Model 3: adjusted OR [aOR] 2.07, 95% CI 1.08–4.11, $p = 0.029$). Additional analyses also showed that 0–72 h VIM /10 was significantly associated with sICH (Model 3: aOR 0.26, 95% CI 0.10–0.56, $p = 0.001$) and midline shift, but not with Δ DWI-ASPECTS.

When VIM was stratified into time epochs (0–8 h, 8–16 h, 16–24 h, and 24–72 h), only 24–72 h VIM/10 was significantly associated with poor outcomes (Model 3: aOR 1.89, 95% CI 1.20–3.06, $p = 0.005$) (Table 1). Similarly, 24–72 h VIM/10 was also significantly associated with sICH in a separate model (Model 3: aOR 0.31, 95% CI 0.15–0.55, $p = 0.001$) (Supplementary Table 4). Sensitivity analysis excluding 35 patients who received antihypertensive therapy during the 24–72-h period and 22 patients with sICH still demonstrated a significant association between 24–72 h VIM/10 and poor outcomes (Model 3: aOR 1.94, 95% CI 1.26–3.11, $p = 0.003$) (Table 2), confirming the robustness of the findings. The results were consistent after additional adjustment for pre-stroke mRS (Supplementary Table 5). In the quartile-based approach

(Supplementary Table 6), in multivariable logistic regression analyses, patients in the highest VIM quartile (Quartile 4) had a significantly higher risk of poor functional outcomes (aOR 5.05, 95% CI 1.92–18.8, $p = 0.001$). Receiver operating characteristic (ROC) analysis, performed to explore the potential clinical threshold of 24–72 h VIM for predicting poor functional outcomes, indicated an area under the ROC curve (AUC) of 0.803, suggesting good discriminative performance. The optimal cut-off value of 0.55, identified using the Youden index, yielded a sensitivity of 70.4% and specificity of 84.6%. At this threshold, the positive predictive value for poor outcomes was 91.5%, and the negative predictive value was 55.0% (Supplementary Fig. 2).

Stratified analyses performed according to baseline DWI-ASPECTS categories, to evaluate the impact of baseline infarct burden, showed a weak association between 24–72 h VIM and poor functional outcomes in patients with DWI-ASPECTS < 6. In contrast, among patients with DWI-ASPECTS ≥ 6 —particularly those with DWI-ASPECTS ≥ 9 or 10—higher 24–72 h VIM/10 was significantly associated with poor functional outcomes (aOR 2.66, 95% CI 1.07–7.83, $p = 0.034$) (Supplementary Tables 7 and Supplementary Fig. 3). No significant interaction between VIM and baseline DWI-ASPECTS was detected (Supplementary Table 8).

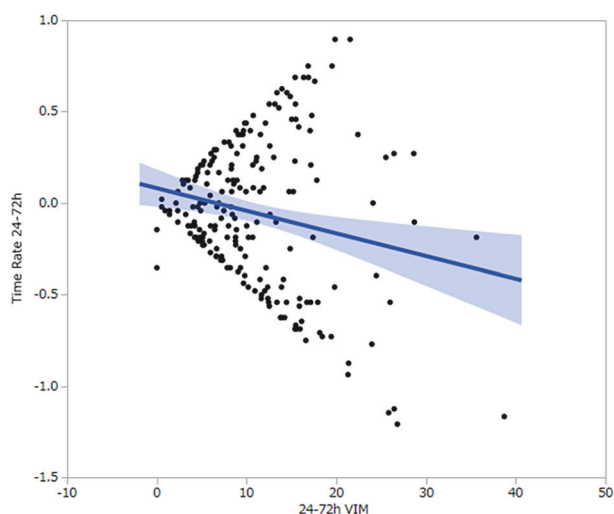


Fig. 2 Relationship between variability independent of the mean (VIM) and time rate (TR) of systolic blood pressure (SBP) between 24 and 72 h after recanalization. A significant negative correlation was observed between VIM and TR (Spearman's $\rho = -0.162$, $p = 0.013$), indicating a tendency to greater SBP variability in patients with an overall declining SBP trajectory

In continuous outcome analyses using the ordinal mRS score at 90 days, higher BPV remained independently associated with worse outcomes. When VIM was scaled per IQR increase, each IQR increase in VIM was associated with an average worsening of 0.44 points in the mRS score (partial regression coefficient [β] 0.44, 95% CI 0.17–0.71, $p = 0.002$) (Supplementary Table 9).

We also conducted an additional analysis to evaluate the potential influence of BP measurement frequency on the results. In this cohort, the median number of BP measurements was 33 (IQR 30–38) during 0–72 h and 17 (IQR 15–20) during 24–72 h post-MT. After additional adjustment for the number of BP measurements during 24–72 h, higher 24–72 h VIM/10 remained independently associated with poor functional outcomes at 90 days (adjusted OR 1.84; 95% CI 1.23–2.88; $p = 0.002$) (Supplementary Table 10). Moreover, in stratified analyses based on the median number of BP measurements (≥ 17 vs. <17), the association between higher 24–72 h VIM/10 and poor outcome was consistently observed in both strata, with no evidence of effect modification by monitoring frequency (Supplementary Table 11).

Correlation analyses demonstrated a significant inverse relationship between 24–72 h VIM and TR during the same period (Spearman's $\rho = -0.162$, $p = 0.013$) (Fig. 2). This association was further confirmed in multivariable regression analysis (Model 3: standardized partial regression coefficient -0.15 ; $\beta -0.25$; 95% CI -0.24 to -0.07 ; $p < 0.001$) (Supplementary Table 12). Collectively, these findings suggest that despite an overall declining trend in

SBP, intermittent sharp fluctuations (spikes) in SBP were present during the 24–72-h period after recanalization.

Regarding END, even in analyses that included END as a covariate for poor functional outcomes at 90 days (mRS score 4–6) as the dependent variable, 24–72 h VIM/10 remained independently associated with unfavorable outcomes, although the effect size was modestly attenuated (Supplementary Table 13). In multivariable logistic regression analyses with END as the outcome, higher VIM/10 during the 24–72-h period was independently associated with the occurrence of END (aOR 1.79; 95% CI 1.13–3.07; $p = 0.013$) (Supplementary Fig. 4). In contrast, VIM during earlier time windows (0–8 h, 8–16 h, and 16–24 h) showed weak or non-significant associations with END. In additional outcome analyses, higher VIM during the 24–72-h period was significantly associated with worse neurological severity at discharge and on day 7, as well as with mortality (Supplementary Tables 14–16).

Detailed etiological and anatomical characteristics of patients with large artery atherosclerosis are shown in Supplementary Table 17. In additional multivariable analyses adjusted for post-treatment stenosis rate and procedural factors reflecting residual vascular pathology, including percutaneous transluminal angioplasty, emergency carotid artery stenting, and emergency intracranial stenting, higher 24–72 h VIM/10 remained independently associated with poor functional outcomes (aOR 1.72, 95% CI 1.13–2.73; $p = 0.009$), whereas post-treatment stenosis rate itself was not significantly associated with outcomes (Supplementary Table 18).

When poor functional outcome was alternatively defined as an mRS score of 3–6 at 90 days, higher VIM/10 during the 24–72-h period showed a borderline significant association with unfavorable outcomes in the fully adjusted model (Supplementary Table 19). Furthermore, in sensitivity analyses excluding patients who received antihypertensive therapy during the 24–72-h period and those with sICH, the direction of the association remained consistent, although statistical significance was not reached (Supplementary Table 20).

Discussion

This study demonstrated that BPV—specifically VIM during the 24–72-h period after successful recanalization—is independently associated with poor functional outcomes following MT for ischemic stroke. This association remained significant in sensitivity analyses excluding patients who received antihypertensive therapy during the first 24–72 h post-MT and those with sICH, supporting the robustness of our findings. Importantly, the prognostic impact of 24–72 h VIM was not materially modified by

baseline infarct burden in DWI-ASPECTS-stratified and interaction analyses, and it was also evident in continuous outcome analyses: each IQR increase in VIM was associated with an average 0.44-point worsening in the ordinal 90-day mRS score. Collectively, these results highlight the clinical importance of BP profiles beyond the initial 24 h post-recanalization.

A major strength of this study is the identification of BPV during a relatively delayed phase (24–72 h post-recanalization), rather than the early post-treatment period, as an independent predictor of unfavorable outcomes. By using VIM, the study captured true fluctuations in SBP unaffected by average levels, providing a more robust indicator of hemodynamic instability. This finding suggests that not merely absolute BP values—such as mean or maximum SBP—but “BP stability” itself may influence prognosis. Previous studies have also demonstrated that BPV indices, such as TR and CV, contribute to infarct progression and poor outcomes [12, 13, 25, 26]. On the other hand, recent randomized controlled trials (e.g., BP-TARGET [3], OPTIMAL-BP [6]) have shown that intensive BP reduction within the first 24 h post-recanalization does not significantly affect outcomes. Moreover, Samuels et al. reported that higher peak SBP within 6 h post-MT was associated with an increased risk of sICH and poor outcomes, while excessively low SBP followed a non-linear risk pattern [27]. Additionally, Sakamoto et al. reported that low SBP and high CV within the first 24 h were associated with infarct growth [26]. However, the importance of BP control beyond this window remains underexplored.

Another noteworthy finding of this study is that the significant association between VIM and poor outcomes persisted even in the analysis excluding patients who underwent antihypertensive therapy and those who developed sICH. This differs from previous reports, such as the individual patient data meta-analysis by Katanos et al., which suggested that both high and low SBP were associated with an increased risk of poor outcomes [1], as well as the multicenter study by Anadani et al., which showed that higher mean SBP was associated with worse outcomes [2]. In contrast, our findings suggest that the magnitude of BPV may have a more significant impact on clinical outcomes as compared to absolute blood pressure value. Furthermore, given the weak association between VIM and Δ DWI-ASPECTS, together with the consistent prognostic impact of VIM across different baseline DWI-ASPECTS strata, VIM is unlikely to be merely a surrogate marker of infarct expansion. While Woods et al. reported that acute-phase BPV was significantly associated with infarct expansion [28], our study did not find a clear correlation with infarct growth indicators. This suggests that VIM may reflect broader neurological deterioration, including cerebral edema and metabolic disturbances. These findings are

consistent with those of Tudor et al., who indicated that microcirculatory disturbances and blood-brain barrier disruption may progress over several dozen hours after stroke onset and contribute to poor outcomes [9]. Moreover, as shown by Horie et al. and Mujanovic et al., the no-reflow phenomenon and microvascular dysfunction can persist even after successful recanalization [10, 29]. Abrupt fluctuations in blood pressure in such vulnerable cerebral perfusion states may pose a risk of further secondary injury.

We also observed a significant inverse correlation between VIM and TR during the 24–72-h period post MT, suggesting that even when SBP showed an overall gradual decline, many patients experienced intermittent spike-like fluctuations. This aligns with Chang et al.’s assertion that SBP variability itself may act as a key mediator of poor outcomes [25]. In other words, an SBP trajectory characterized by gradual decline with intermittent sharp fluctuations may lead to hemodynamic instability and worsen outcomes. From a clinical perspective, BP management during the 24–72 h post-recanalization should not only aim to achieve target BP levels, but also minimize fluctuations. Practical strategies may include avoiding rapid bolus antihypertensive drug administration, selecting antihypertensive agents with stable pharmacokinetics, and addressing potential triggers of BPV, such as positional changes, pain, or agitation. Furthermore, the negative association observed between VIM and sICH indicates that our findings likely do not merely reflect hemorrhagic complications. Instead, they may represent a non-hemorrhagic mechanism of outcome deterioration, such as perfusion instability, cerebral edema, or secondary neuronal injury. This interpretation is supported by findings from Kellert et al., who reported that BPV after MT was associated with poor functional outcomes independent of sICH [30], which is consistent with our results.

An important methodological consideration in the present study is the definition of poor functional outcomes. Unlike recent BP management trials after MT [3–6]), which were primarily designed to enroll patients with small to moderate infarct cores, our cohort included a substantial proportion of patients with large infarct cores. In fact, approximately 32% of patients had DWI-ASPECTS < 6, a subgroup in which achieving functional independence (mRS 0–2) is often unrealistic even after successful recanalization. In this clinical context, dichotomization of outcomes into mRS scores of 0–3 versus 4–6 has been commonly adopted in recent MT studies [31] focusing on large-core infarction, since even avoidance of severe disability or death represents a clinically meaningful therapeutic goal in these patients. Accordingly, in order to better reflect real-world clinical practice, we defined poor outcomes as mRS scores of 4–6, which represents a unique strength of this study compared with previous investigations

of BPV. Importantly, sensitivity analyses using the more conventional definition of poor outcomes of mRS scores of 3–6 demonstrated a consistent trend toward an association between 24–72 h BPV and unfavorable outcomes, although statistical significance was borderline.

Our additional analyses provided important insights into the potential relationship between mid-phase BPV and END. In the present cohort, higher BPV during the 24–72-h period after recanalization was independently associated with the occurrence of END, whereas BPV during earlier time windows showed weaker or no significant associations. Importantly, when END was incorporated as an additional covariate in the multivariable model for poor functional outcomes at 90 days post-MT, higher 24–72 h BPV remained independently associated with unfavorable outcomes, although the magnitude of the association was modestly attenuated. This finding suggests that END may partially mediate, but does not fully explain, the relationship between mid-phase BPV and long-term functional outcomes. In other words, BPV beyond 24 h after MT may contribute to a poor prognosis, both, through early neurological worsening and through other mechanisms affecting subacute and chronic recovery.

Taken together, these results suggest that for patients who achieve successful recanalization after MT, it is not sufficient to merely lower SBP; rather, suppressing SBP variability may be an important management strategy for improving outcomes. In particular, BP monitoring and stabilization beyond 24 h after recanalization should be emphasized. Further, in addition to intrinsic hemodynamic instability, several aspects of routine clinical management during the 24–72-h period may act as triggers of BPV. In our cohort, intravenous nicardipine was administered in 19.6% of patients, and its titration or discontinuation was at the discretion of the treating neurologist. Withdrawal of continuous intravenous antihypertensive therapy may induce transient “rebound-like” fluctuations in BP, while simultaneously allowing SBP values to reflect more natural, unmasked hemodynamic behavior. Furthermore, early mobilization and rehabilitation are typically initiated during this period in patients without residual LVO. Transitions from bed rest to sitting, standing, and ambulation are known to influence systemic BP and cerebral perfusion, particularly in the context of impaired autoregulation after stroke. Although the timing and intensity of rehabilitation could not be systematically quantified in this retrospective study, such management-related factors may partly contribute to increased SBP variability during the mid-phase after MT. Notably, randomized trials such as HeadPoST [32] and AVERT [33] have demonstrated that early head-position changes or very early mobilization can significantly affect cerebral perfusion dynamics and clinical outcomes, underscoring the importance of patient handling when

interpreting BP variability in the post-recanalization period. In clinical practice, these findings also highlight the need to select and adjust antihypertensive agents in a manner that avoids abrupt BP fluctuations during early recovery.

In this study, as shown in Supplementary Table 1, current smoking showed a seemingly paradoxical association with prognosis, with a lower prevalence of current smokers in the poor-outcome group despite the well-established adverse vascular effects of smoking. This counterintuitive pattern is likely attributable to residual confounding and selection bias rather than any protective effect of smoking. In our cohort, the absolute number of current smokers was small, and smokers tended to have a more favorable baseline risk profile, including younger age and less pre-stroke disability, which may have contributed to better crude outcomes. Importantly, smoking status was included as an adjustment covariate in all multivariable analyses, and the independent association between 24–72 h SBP VIM and poor functional outcome remained robust. Therefore, this univariate finding does not alter the interpretation of our main results.

This study has several limitations. First, it was a retrospective analysis conducted at a single center and included only East Asian patients. Second, SBP measurements obtained during the 24–72-h period represented a combination of “natural” hemodynamic values and values influenced by medical intervention. Antihypertensive agents, either intravenous or oral, were administered in 19.6% of patients during the observation window, indicating that approximately one-fifth of SBP measurements may have been affected by active BP-lowering therapy. Nevertheless, even this proportion of therapeutic modulation may introduce confounding in the interpretation of BPV. Additionally, most of the study period preceded the publication of recent RCTs on BP control after successful recanalization [3–6]. Therefore, the SBP management strategies employed in this study may differ from current real-world clinical practice. Third, there was some variability in the timing and frequency of BP measurements, which may have affected the calculation of VIM. The median frequency of SBP monitoring was approximately once every 2–4 h. Moreover, a clinically relevant cutoff value for absolute VIM remains to be established, and further investigation is needed before its practical implementation. Fourth, AF was present in nearly 70% of our cohort, and a substantial proportion of patients likely had systemic atherosclerosis based on their vascular risk profile. Both conditions may contribute to increased BPV. AF is characterized by beat-to-beat irregularity and impaired cardiac output regulation, which can translate into greater short-term BP fluctuations. Likewise, systemic atherosclerosis and arterial stiffness may impair baroreflex sensitivity and reduce the vascular system’s ability to buffer BP changes, thereby amplifying BPV.

Although the present study was not designed to quantify the specific contributions of AF or atherosclerotic burden to BPV, these comorbidities may partly account for inter-individual differences in BP dynamics and should be considered in future mechanistic and interventional studies. Lastly, our imaging protocol was primarily MRI-first, and the lack of perfusion imaging at occlusion or collateral flow assessments might have led to underestimation of the initial ischemic burden. Future studies should include sensitivity analyses using standardized measurement frequencies, high-resolution data from continuous arterial pressure monitoring, and interventional trials targeting BPV suppression—specifically VIM during the 24–72-h period—as a primary outcome or therapeutic target, to clarify causal relationships and assess feasibility in clinical practice.

Perspective of Asia

Post-thrombectomy blood pressure management varies across regions, and recent evidence suggests that deviations from individualized autoregulatory blood pressure limits may worsen neurological outcomes [34]. Asian populations, in whom intracranial atherosclerosis and hypertension-related vascular changes are more prevalent, may have a different susceptibility to blood pressure fluctuations compared with Western cohorts. In addition, regional differences exist in post-thrombectomy practice, including preferred antihypertensive agents, thresholds for blood pressure lowering, and the intensity of early hemodynamic control. Variations in anesthetic strategy, such as the use of general anesthesia versus conscious sedation, may further influence peri-procedural blood pressure stability. These differences suggest that blood pressure targets derived from Western studies may not be universally applicable. Further multicenter studies including Asian populations are warranted to establish region-specific and physiology-guided blood pressure management strategies after thrombectomy.

Conclusion

In conclusion, this study demonstrated that greater VIM of SBP during 24–72 h after successful recanalization is associated with poor functional outcomes of acute ischemic stroke, independent of sICH and the use of antihypertensive therapy. These findings highlight the importance of incorporating BPV suppression—and not just the determination of BP targets—into post-MT management strategies. Future interventional studies targeting BPV beyond 24 h after recanalization are warranted to explore the potential for improving clinical outcomes following MT for ischemic stroke.

Acknowledgements The authors thank Forte Science Communications (<https://www.forte-science.co.jp>) for language editing of this manuscript.

Compliance with ethical standards

Conflict of interest Yuki Hamada: reports lecture fees from Daiichi Sankyo Co. Ltd., Otsuka Pharmaceutical Co. Ltd., Novartis Pharma Co. Ltd., Medtronic Co. Ltd., Medico's Hirata Co. Ltd., Johnson & Johnson Co. Ltd., Merit Medical Systems Inc., and Kaneka Co. Ltd. outside the submitted work. Hideki Matsuoka: reports lecture fees from Daiichi Sankyo Co. Ltd., Pfizer Co. Ltd., Otsuka Pharmaceutical Co. Ltd., Kowa Co. Ltd., UCB Japan Co. Ltd., Novartis Pharma Co. Ltd., and AstraZeneca Co. Ltd. outside the submitted work. Takeo Sato: reports lecture fees from Pfizer Japan Inc. and Kowa Co. Ltd. outside the submitted work. Kyosuke Hamada: reports lecture fees from Medtronic Co., Ltd., and Japan Lifeline Co., Ltd. outside the submitted work. Takumi Shimada: none Yutaro Kawabata: none Kana Iwamoto: none Mei Ikeda: none Go Takaguchi: none Yujiro Higuchi: none Hiroshi Takashima: reports lecture fees from Daiichi Sankyo Co. Ltd., Otsuka Pharmaceutical Co. Ltd., Biogen Japan Ltd., Eisai Co. Ltd., FP Pharmaceutical Corp., Alnylam Pharmaceuticals Inc., Kyowa Kirin Co. Ltd., Alexion Pharmaceuticals Inc., Argenx Japan K.K., Cyberdyne Inc., Takeda Pharmaceutical Co. Ltd., CSL Behring K.K., Asahi Kasei Medical Co. Ltd., Chugai Pharmaceutical Co. Ltd., Sumitomo Pharma Co. Ltd., Amgen Inc., Japan Blood Products Organization, Cosmic Corporation Co. Ltd., and Ono Pharmaceutical Co. Ltd. outside the submitted work. Hiroshi Takashima also reports scholarship donations from Shin Nippon Biomedical Laboratories Ltd. and Asahi Kasei Medical Co. Ltd. outside the submitted work.

References

1. Katsanos AH, Malhotra K, Ahmed N, Psychogios MN, Turc G, Khatri P, et al. Blood pressure after endovascular thrombectomy and outcomes in patients with acute ischemic stroke: an individual patient data meta-analysis. *Neurology*. 2022;98:e291–e301.
2. Anadani M, Arthur AS, Tsvigoulis G, Alawieh A, Orabi Y, Chalhoub R, et al. Blood pressure goals and clinical outcomes after successful endovascular therapy: a multicenter study. *Ann Neurol*. 2020;87:830–9. <https://doi.org/10.1002/ana.25724>.
3. Mazighi M, Richard S, Lapergue B, Sibon I, Gory B, Berge J, et al. Safety and efficacy of intensive blood pressure lowering after successful endovascular therapy in acute ischaemic stroke (BP-TARGET): a multicentre, open-label, randomised controlled trial. *Lancet Neurol*. 2021;20:265–74.
4. Yang P, Song L, Zhang Y, Zhang X, Chen X, Li Y, et al. Intensive blood pressure control after endovascular thrombectomy for acute ischaemic stroke (ENCHANTED2/MT): a multicentre, open-label, blinded-endpoint, randomised controlled trial. *Lancet*. 2022;400:1585–96.
5. Mistry EA, Hart KW, Davis LT, Jovin TG, Demchuk AM, Donnan GA, et al. Blood pressure management after endovascular therapy for acute ischemic stroke: the best-II randomized clinical trial. *JAMA*. 2023;330:821–31.
6. Nam HS, Kim YD, Heo J, Choi MA, Sung J, Bae H-J, et al. Intensive vs conventional blood pressure lowering after endovascular thrombectomy in acute ischemic stroke: the OPTIMAL-BP randomized clinical trial. *JAMA*. 2023;330:832–42.
7. Mistry EA, Mehta T, Mistry A, Arora N, Starosciak AK, De Los Rios La Rosa F, et al. Blood pressure variability and neurologic outcome after endovascular thrombectomy: a secondary analysis of the BEST study. *Stroke*. 2020;51:511–8.

8. Valls Carbó A, Palomar A, Laredo C, Werner M, Dorado L, Remollo S, et al. Relevance of persistent perfusion deficits on clinical outcomes after successful endovascular treatment: a prospective serial magnetic resonance study. *Front Neurol*. 2025;16:1478240 <https://doi.org/10.3389/fneur.2025.1478240>.
9. Tudor T, Spinazzi EF, Alexander JE, Goyal N, Seker F, Sharma VK, et al. Progressive microvascular failure in acute ischemic stroke: a systematic review, meta-analysis, and time-course analysis. *J Cereb Blood Flow Metab*. 2024;44:192–208.
10. Mujanovic A, Ng F, Meinel TR, Fiebach JB, Liebeskind DS, Nogueira RG, et al. No-reflow phenomenon in stroke patients: a systematic literature review and meta-analysis of clinical data. *Int J Stroke*. 2024;19:58–67.
11. Zhang Y, Jiang M, Gao Y, Zhao W, Wu C, Li C, et al. No-reflow phenomenon in acute ischemic stroke. *J Cereb Blood Flow Metab*. 2024;44:19–37.
12. Choi KH, Kim JM, Kim JH, Kim JT, Park MS, Choi SM, et al. Optimal blood pressure after reperfusion therapy in patients with acute ischemic stroke. *Sci Rep*. 2019;9:5681. <https://doi.org/10.1038/s41598-019-42240-8>.
13. Tao M, Li Y, Peng Y, Zhang X, Liu S, Tang T, et al. Blood pressure fluctuation during 72 h after endovascular therapy and prognosis in acute ischemic stroke patients. *J Endovasc Ther*. 2024;33:462–72. <https://doi.org/10.1177/15266028241266235>.
14. Reynolds K, Lewis B, Nolen JD, Kinney GL, Sathya B, He J. Alcohol consumption and risk of stroke: a meta-analysis. *JAMA*. 2003;289:579–88.
15. Pexman JH, Barber PA, Hill MD, Sevrick RJ, Demchuk AM, Hudon ME, et al. Use of the Alberta Stroke Program Early CT Score (ASPECTS) for assessing CT scans in patients with acute stroke. *AJNR Am J Neuroradiol*. 2001;22:1534–42.
16. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtypes of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in acute stroke treatment. *Stroke*. 1993;24:35–41.
17. Tomsick TA, Broderick JP, Carrozzella JA, Khatri P, Hill MD, Palesch YY, et al. Revascularization results in the interventional management of stroke II trial. *AJNR Am J Neuroradiol*. 2008;29:582–7.
18. Hacke W, Kaste M, Fieschi C, von Kummer R, Dávalos A, Meier D, et al. Randomised double-blind placebo-controlled trial of thrombolytic therapy with intravenous alteplase in acute ischaemic stroke (ECASS II). Second European-Australasian Acute Stroke study investigators. *Lancet*. 1998;352:1245–51.
19. Mokin M, Setlur Nagesh SV, Ionita CN, Levy EI, Siddiqui AH. Comparison of modern stroke thrombectomy approaches using an in vitro cerebrovascular occlusion model. *Am J Neuroradiol*. 2015;36:547–51.
20. Cuadrado-Godia E. Early neurological deterioration, easy methods to detect it. *Indian J Med Res*. 2015;141:266–8.
21. Seners P, Turc G, Oppenheim C, Baron JC. Incidence, causes and predictors of neurological deterioration occurring within 24h following acute ischaemic stroke: a systematic review with pathophysiological implications. *J Neurol Neurosurg Psychiatry*. 2015;86:87–94.
22. Wijdicks EFM, Sheth KN, Carter BS, Greer DM, Kasner SE, Kimberly WT, et al. American Heart Association Stroke Council. Recommendations for the management of cerebral and cerebellar infarction with swelling: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2014;45:1222–38.
23. Mistry EA, Mistry AM, Nakawah MO, Chitale RV, James RF, Volpi JJ, et al. Systolic blood pressure within 24 h after endovascular thrombectomy and outcomes in patients with large vessel occlusion strokes: post hoc analysis of the escape randomized clinical trial. *JAMA Neurol*. 2017;74:1019–25.
24. Molina CA, Chamorro Á, Rovira À, De Miquel MA, Serena J, Roman LS, et al. Blood pressure management after endovascular therapy: results from the REVASCAT trial. *Stroke*. 2018;49:416–22.
25. Chang JY, Park JY, Song JY, Yoo JS, Lee KB, Jung JW, et al. Mediation of time-related blood pressure variability on intensive blood pressure lowering and functional outcomes post endovascular therapy: a Post Hoc analysis of the OPTIMAL-BP trial. *J Am Heart Assoc*. 2025;14:e039723 <https://doi.org/10.1161/JAHA.124.039723>.
26. Sakamoto Y, Takeuchi Y, Nagano Y, Yamashita S, Ishizuka K, Minami S, et al. Associations between systolic blood pressure and infarct growth after thrombectomy for acute stroke. *Int J Stroke*. 2025;20:17474930251358139 <https://doi.org/10.1177/17474930251358139>.
27. Samuels N, van den Berg SA, de Ridder IR, van der Ende NAM, van der Schaaf IC, et al. Blood pressure in the first 6 h following endovascular treatment for ischemic stroke is associated with intracranial hemorrhage and functional outcome. *J Neurointerv Surg*. 2021;13:787–91. <https://doi.org/10.1136/neurintsurg-2020-016797>.
28. Woods AG, Lillicrap T, Hood R, Spratt NJ, Parsons MW, Levi CR, et al. Blood pressure variability is associated with infarct growth in acute ischaemic stroke. *Cerebrovasc Dis*. 2024;53:449–56. <https://doi.org/10.1159/000538261>.
29. Horie N, Inoue M, Morimoto T, Hayashi K, Hayashi T, Tateishi Y, et al. Recanalization does not always equate to reperfusion: no-reflow phenomenon after successful thrombectomy. *Stroke*. 2025;56:183–9. <https://doi.org/10.1161/STROKEAHA.124.043211>.
30. Kellert L, Hametner C, Rohde S, Bendszus M, Hacke W, Ringleb PA, et al. Impact of Blood pressure variability on outcome after mechanical thrombectomy: a post hoc analysis of the randomized controlled endovascular stroke trials (SYNTHESIS Expansion, MR CLEAN, ESCAPE, REVASCAT, and SWIFT PRIME). *Stroke*. 2017;48:2767–72.
31. Sarraj A, Yoshimura S, Thomalla G, Huo X, Arquizaan C, Yoo AJ, et al. Mechanical thrombectomy for large ischemic stroke: a critical appraisal of evidence from 6 randomized controlled trials. *Stroke*. 2025;56:1917–27.
32. Anderson CS, Arima H, Lavados P, Munoz Venturrelli P, Pontes-Neto O, Wang X, et al. Head position and cerebral perfusion in acute ischemic stroke: the HeadPoST trial. *N Engl J Med*. 2017;376:2437–47.
33. Bernhardt J, Langhorne P, Dewey H, Thrift A, Donnan G, Anderson C, et al. Very early mobilization after stroke: the AVERT randomized controlled trial. *Lancet*. 2015;386:46–55.
34. Petersen NH, Begunova L, Olexa M, Kumar A, Chen Y, Dhar R, et al. Autoregulation-guided blood pressure targets after stroke thrombectomy: impact on secondary brain injury and neurologic outcomes. *Neurology*. 2026;106:e214577 <https://doi.org/10.1212/WNL.0000000000214577>.

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