JAMA Neurology | Original Investigation

Personalized Blood Pressure Targeting After Endovascular Therapy for Acute Ischemic Stroke
A Randomized Clinical Trial

Pol Camps-Renom, PhD; Marina Guasch-Jiménez, MD; Juan Álvarez-Cienfuegos, MD; Nicolás López-Hernández, MD; Ana Rodríguez-Campello, PhD; Herbert Tejada-Meza, MD; Luis López-Mesonero, MD; Laura Albert-Lacal, MD; Mari Mar Freijo-Guerrero, MD; Diana Tarruella-Hernández, MD; Alan Flores, MD; Juan Antonio Cabezas-Rodríguez, MD; Joan Miquel Fernández-Vidal, MD; Alejandro Martínez-Domeño, MD; Natalia Pérez de la Ossa, PhD; Anna Ramos-Pachón, MD; Ana Aguilera-Simón, BSc; Rebeca Marín, RN; Garbiñe Ezcurra-Díaz, MD; Álvaro Lambea-Gil, MD; Yolanda Silva, PhD; Diego José Corona-García, MD; Eva Giralt-Steinhauer, PhD; Javier Marta-Moreno, MD; Jesús Alberto Vizcaya-Gaona, MD; Ainara Sanz-Monllor, MD; Alain Luna, MD; Mikahela López Morales, PhD; Xavier Ustrell, MD; Francisco Moniche, PhD; Judit Solà-Roca, MSc; Xia Wang, PhD; Craig S. Anderson, MBBS, PhD; Luis Prats-Sánchez, PhD; Joan Martí-Fàbregas, PhD; for the HOPE Study Group

IMPORTANCE Optimal blood pressure (BP) management after successful endovascular therapy for acute ischemic stroke remains uncertain, as intensive lowering has shown no benefit or potential harm in prior trials.

OBJECTIVE To determine whether a reperfusion-guided systolic BP control strategy improves functional outcomes compared with guideline-recommended management after successful endovascular therapy for acute ischemic stroke.

DESIGN, SETTING, AND PARTICIPANTS This investigator-initiated, multicenter, prospective, randomized, open-label clinical trial with blinded end point assessment was conducted among adults with acute ischemic stroke due to anterior circulation large-vessel occlusion who achieved successful reperfusion (modified Thrombolysis in Cerebral Infarction [mTICI] score ≥2b) after endovascular therapy at 11 comprehensive stroke centers in Spain between June 14, 2021, and October 1, 2025, with 90-day follow-up. Data analysis was conducted from February 1 to March 12, 2026.

INTERVENTIONS Participants were randomly assigned (1:1) to a reperfusion-guided systolic BP strategy (140-160 mm Hg for mTICI score of 2b; 100-140 mm Hg for mTICI score of 2c/3) or guideline-recommended management (systolic BP <180 mm Hg) for 72 hours using antihypertensive agents or vasopressors as needed.

MAIN OUTCOMES AND MEASURES The primary outcome was a favorable functional outcome, defined as a modified Rankin Scale score of 0 to 2 at 90 days, assessed in the intention-to-treat population. Of 446 enrolled patients, 440 were included in the intention-to-treat analysis (mean age, 75 years; 53% women); 6 were excluded due to withdrawal or consent withdrawal.

RESULTS Among 440 patients (mean [SD] age, 75 [12] years; 233 [53.0%] women), 215 were assigned to the intervention group and 225 to the control group. At 90 days, 129 patients (60.0%) in the intervention group and 106 (47.1%) in the control group achieved a favorable functional outcome (absolute risk difference, 13.3%; 95% CI, 4.1%-22.6%; $P = .005$). Hemorrhagic transformation occurred in 48 patients (22.3%) in the intervention group and 71 (31.6%) in the control group (odds ratio, 0.62; 95% CI, 0.41-0.95). The rates of symptomatic intracranial hemorrhage (3.5% vs 3.9%) and 90-day mortality (15.4% vs 15.6%) did not differ between groups. Serious adverse events occurred in 34 patients (15.8%) in the intervention group and 27 (12.0%) in the control group.

CONCLUSIONS AND RELEVANCE In this randomized clinical trial, a reperfusion-guided BP strategy improved functional outcomes and reduced hemorrhagic transformation without increasing major safety events, supporting a tailored approach to postthrombectomy BP management.

TRIAL REGISTRATION ClinicalTrials.gov Identifier: NCT04892511

JAMA Neurol. doi:10.1001/jamaneurol.2026.1706
Published online June 8, 2026.

Visual Abstract

Supplemental content

Author Affiliations: Author affiliations are listed at the end of this article.

Group Information: A list of the HOPE Study Group members appears in Supplement 4.

Corresponding Authors: Pol Camps-Renom, PhD (pcamps@santpau.cat), and Luis Prats-Sánchez, PhD (lpratss@santpau.cat), Stroke Unit, Department of Neurology, Hospital de la Santa Creu i Sant Pau, Institut de Recerca Sant Pau, Carrer de Sant Antoni Maria Claret 167, Barcelona 08025, Spain.

Endovascular therapy has transformed the management of patients with acute ischemic stroke due to large-vessel occlusion.[1] Yet, despite high rates of successful recanalization being achieved, up to one-half of patients with angiographically successful reperfusion do not achieve a favorable functional outcome as defined by a modified Rankin Scale (mRS) score of 0 to 2.[2,3] This phenomenon, often referred to as clinically ineffective reperfusion,[4] has been attributed to a variety of mechanisms that include reperfusion injury, impaired microcirculatory flow, hemorrhagic transformation, and infarct progression.[5-8] Adjunctive strategies to endovascular therapy are needed to improve clinical outcomes.

Elevated systolic blood pressure (BP) that follows endovascular therapy has consistently been shown to be associated with hemorrhagic transformation, poor functional outcome, and mortality across individual and pooled studies.[9-11] These observations have led to the hypothesis that intensive BP lowering can mitigate reperfusion injury and improve outcomes. However, over the last several years, 6 randomized clinical trials have shown that such treatment produces either neutral or harmful effects,[12-17] findings that have been further reinforced in meta-analyses.[18,19] These results have led to changes in practice and guidelines, such as from the American Heart Association/American Stroke Association, which recommend against the use of intensive BP lowering following endovascular therapy for acute ischemic stroke.[20]

Several factors may account for the adverse effects of intensive BP-lowering therapy. Cerebral autoregulation may have been impaired due to chronic hypertension and intracranial atherosclerotic disease, which are both highly prevalent in the Asian participants of many of the trials.[13,14] Another potential cause of cerebral hypoperfusion is the uniform approach to achieving BP targets, without accounting for the degree of reperfusion achieved or allowing the use of vasopressors to maintain BP within a target range. Compared with patients with near-complete or complete reperfusion, generally defined by modified Treatment in Cerebral Infarction (mTICI) scores of 2c or 3, patients with incomplete angiographic reperfusion (mTICI score of 2b) may require higher perfusion pressures because they depend more heavily on collateral circulation.[21] Applying uniform BP targets to all patients after endovascular therapy may be inappropriate.

We conducted the Hemodynamic Optimization of Cerebral Perfusion After Endovascular Therapy (HOPE) randomized clinical trial to test the hypothesis that a protocol in which systolic BP target ranges are tailored according to final reperfusion status would improve functional outcome by reducing reperfusion injury while preserving cerebral perfusion after endovascular therapy for acute ischemic stroke.

Key Points

Question Does a reperfusion-guided systolic blood pressure control strategy improve functional outcomes after successful endovascular therapy for acute ischemic stroke compared with guideline-recommended management?

Findings In this multicenter randomized clinical trial including 440 patients, a higher proportion of patients in the intervention group achieved a favorable functional outcome at 90 days compared with the control group, and this difference was statistically significant. Hemorrhagic transformation occurred less frequently in the intervention group, while mortality and symptomatic intracranial hemorrhage did not differ significantly between groups.

Meaning These findings suggest that reperfusion-guided blood pressure management may improve functional recovery without increasing safety risks after endovascular therapy.

Methods

Study Design

HOPE was an investigator-initiated and investigator-conducted, multicenter, prospective, randomized, open-label, blinded-end point (PROBE design), phase 3 clinical trial, conducted at 11 comprehensive stroke centers in Spain. The trial protocol and statistical analysis plan have been published[22,23] and are available in Supplement 1 and Supplement 2, respectively. This report adheres to the Consolidated Standards of Reporting Trials (CONSORT) reporting guidelines.[24] The study protocol was reviewed and approved by the lead ethics committee of the Hospital de la Santa Creu i Sant Pau and by the ethics committees of each participating center. To avoid treatment delay, verbal consent was obtained at the time of initial assessment and randomization; written informed consent was subsequently obtained after the endovascular procedure once participants were clinically stable, or from an authorized next of kin for participants who were unable to provide consent. Data analysis was conducted from February 1 to March 12, 2026.

Participants

Participants were eligible for inclusion if they were aged 18 years or older, were previously functionally independent (score of 0-2 on the mRS), and had achieved successful reperfusion after endovascular therapy (defined as an mTICI score ≥2b) for an acute ischemic stroke. The site of occlusion was confined to the anterior circulation, any of the terminal internal carotid artery, proximal M1 or M2 segments of the middle cerebral artery, proximal A1 segment of the anterior cerebral artery, or a tandem occlusion, within 24 hours of the onset of symptoms. Participants were not required to be hypertensive (systolic BP >140 mm Hg) to be eligible. Key exclusion criteria included having an Alberta Stroke Program Early Computed Tomography Score of less than 6, acute ischemic stroke of the posterior circulation, congestive heart failure, arterial dissection, and the presence of nonrevascularized intracranial or extracranial stenosis of 50% or more at the end of the endovascular procedure. Patients were also ineligible if they were participating in another trial that might interfere with the outcome assessments. Full details of the inclusion and exclusion criteria are provided in the eAppendix in Supplement 3.

Randomization and Allocation Concealment

Conditional stratified randomization was performed using an online tool (Clinapsis; Clinical Epidemiology and Healthcare

Services) according to reperfusion status (mTICI scores of 2b vs mTICI scores of 2c or 3) and center. Participants were to be allocated (1:1) to the hemodynamic optimization protocol or standard care within 1 hour after the last angiographic image series. Investigators had 1 hour additionally to achieve the assigned systolic BP target range (eFigure 1 in Supplement 3).

Interventions

Participants allocated to the hemodynamic optimization (intervention) group were assigned to either of 2 systolic BP target ranges according to the degree of angiographic reperfusion after endovascular therapy: 100 to 140 mm Hg for mTICI scores of 2c or 3, and 140 to 160 mm Hg for an mTICI score of 2b. These targets were established based on contemporaneous observational evidence to balance the risks of hypoperfusion and reperfusion injury. Participants assigned to the control group received standard care in accordance with guideline recommendations at the time (systolic BP of ≤180 mm Hg).[25]

For participants to achieve the assigned systolic BP target ranges in the intervention group for 72 hours after endovascular therapy, the protocol specified that intravenous labetalol or urapidil were to be used as first-line antihypertensive agents. If the assigned systolic BP range could not be attained with these agents, alternative intravenous antihypertensive agents were permitted (eFigure 2 in Supplement 3). The choice of first-line antihypertensive agent was made at the discretion of the treating physician. For patients requiring BP augmentation, isotonic saline was recommended as first-line therapy, followed by phenylephrine, ephedrine, or norepinephrine if necessary (eFigure 3 in Supplement 3). No pharmacological intervention was administered if patients spontaneously achieved the assigned systolic BP target range.

Noninvasive BP monitoring was performed as follows: in the first 24 hours, BP was measured at least every 30 minutes in the intervention group and at least every hour in the control group; from 24 to 72 hours, BP was measured at least every hour in the intervention group and at least every 6 hours in the control group. BP lowering was to be promptly interrupted in the event of predefined safety conditions, including hemorrhagic transformation of the cerebral infarction within the first 72 hours (defined as parenchymal hemorrhage type 2 [PH2] or any symptomatic intracerebral hemorrhage [sICH] according to the Heidelberg classification),[26] early recurrent stroke, or hemodynamic instability. In cases of PH2 or sICH, stricter BP control (target systolic BP <140 mm Hg) was recommended at the discretion of the treating physician.

Procedures and Outcome Evaluation

Screening logs were maintained at each participating center for patients admitted with acute ischemic stroke from large-vessel occlusion during the study period. Demographic characteristics, baseline imaging, and clinical and treatment data were collected on all participants at the times of presentation and randomization. Sex was defined as that assigned at birth. Follow-up assessments were conducted at 24, 48, and 72 hours after randomization, at hospital discharge, and at 90 days (±15 days). The 90-day outcomes were assessed by certified personnel at each center who were blinded to treatment allocation, whereas participants and treating clinicians were not blinded due to the nature of the intervention.

The protocol required a follow-up noncontrast computed tomographic scan at 24 hours (±12 hours) and recommended another at 72 hours (±12 hours). All these images were uploaded to a centralized imaging platform (Collective Minds) for blinded assessment by trained clinicians unaware of the treatment allocation.

All data were monitored by external qualified personnel of the Spanish Clinical Research Network (Instituto de Salud Carlos III). Key variables (age, prestroke mRS score, baseline National Institutes of Health Stroke Scale [NIHSS] score, time from symptom onset to randomization, and final mTICI score), which were used in the primary outcome analysis, were verified against source medical records for 100% of enrolled participants. In addition, a comprehensive source data verification, including all remaining study variables, was performed in 50% of participants. All serious adverse events were reported according to standardized criteria and reviewed by a designated study monitor.

An independent data and safety monitoring board (DSMB) reviewed the trial for safety and conduct. They performed an early evaluation following publication of the ENCHANTED2-MT (Intensive Blood Pressure Control After Endovascular Thrombectomy for Acute Ischaemic Stroke) trial after 130 patients had been enrolled.[13] Another review occurred at a preplanned interim analysis after 250 participants had been enrolled. Interim analyses were conducted according to a prespecified statistical analysis plan (Supplement 2), using O'Brien-Fleming boundaries to assess efficacy and harm and using conditional power analyses to evaluate futility.[23] The DSMB had access to unblinded data, while investigators remained blinded, and recommended continuation of the trial without modification at each review.

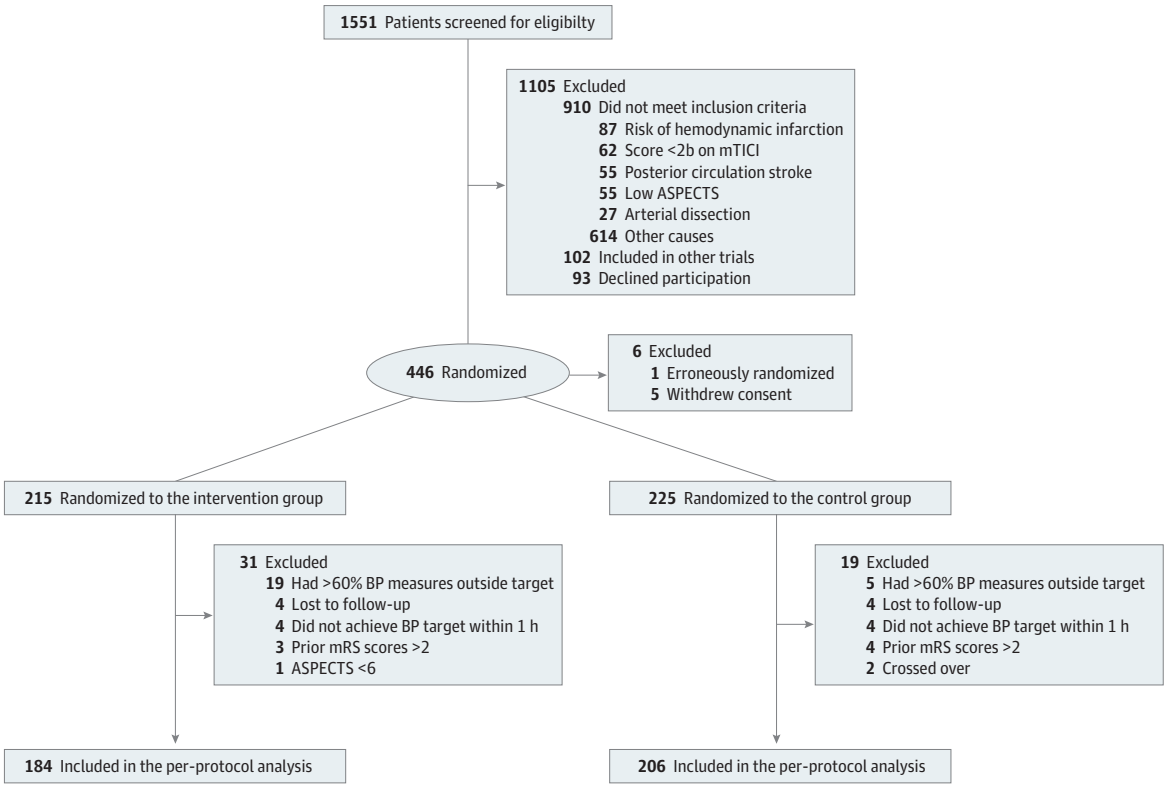
Outcomes

The primary outcome was a favorable functional outcome, defined by scores of 0 to 2 on the mRS, assessed at 90 days. The secondary efficacy outcome was a shift in the distribution of scores on the mRS at 90 days. The safety outcomes were early neurological deterioration, defined as an increase in scores of 4 or more on the NIHSS at 24 hours and 72 hours; any hemorrhagic transformation, sICH, and PH2 at 24 hours according to the Heidelberg bleeding classification system[26]; and 90-day all-cause mortality. The extended brain-imaging outcomes, as detailed in the statistical analysis plan (Supplement 2), will be reported separately.[24]

Statistical Analysis

The trial was designed with 80% power (2-sided $\alpha = .05$) to detect a 10% absolute difference in favorable functional outcome between the randomized groups according to observational data,[9] as no randomized clinical trial data were available at the time. Following publication of a meta-analysis of the first 4 clinical trials on BP management after endovascular therapy,[18] the sample size was subsequently recalculated as 393 participants in each group (eAppendix in Supplement 3).

Figure 1. Flow Diagram of Allocation, Follow-Up, and Analysis of Trial Participants



ASPECTS indicates Alberta Stroke Program Early Computed Tomography Score; BP, blood pressure; mRS, modified Rankin Scale; mTICI, modified Treatment in Cerebral Infarction.

The primary outcome was analyzed in the intention-to-treat population using an unadjusted logistic regression model. An adjusted analysis including prespecified prognostic variables (age, prestroke mRS score, baseline NIHSS score, and time from symptom onset to randomization) was performed as a sensitivity analysis. The secondary outcome of a shift in scores on the mRS at 90 days was assessed in a similar way using ordinal logistic regression. These analyses were also undertaken in the per-protocol population, as prespecified in the statistical analysis plan (Supplement 2).[23] Missing data on the outcome due to loss to follow-up were imputed using the last-observation-carried-forward method, typically from the time of discharge from the hospital, provided that the proportion of imputed cases did not exceed 5% of the overall sample. A sensitivity analysis using multiple imputation was also performed; further details are provided in the eAppendix in Supplement 3.

Safety outcomes were assessed with logistic regression, with the same statistical approach as the effect analysis but without inclusion of prior mRS scores. Various measures of systolic BP control, including central tendency (mean and SD), variability (coefficient of variation and average real variability), time-in-target ratio, and episodes of systolic BP lower than 100 mm Hg, were analyzed by treatment group and mTICI score. Differences in achieved systolic BP between treatment groups were assessed using a linear mixed-effects model, including treatment group (control vs intervention), reperfusion status (mTICI score of 2b vs 2c or 3), time, and their interactions as fixed effects. Participant-level random effects were used to account for within-patient correlation of repeated measurements. Pairwise comparisons of predicted marginal means across treatment group-mTICI subgroups were used to estimate mean differences in systolic BP.

The treatment effect was assessed across 8 prespecified subgroups with tests of interaction. The subgroup of collateral status (good vs poor) will be reported separately. All analyses were performed using Stata version 19 statistical software (StataCorp LLC).

Results

The trial was stopped early due to the lack of ongoing funding. Between June 14, 2021, and October 1, 2025, of 1551 screened patients, 446 were randomized to treatment (eTable 1 in Supplement 3). The main reason for exclusion was ineligibility due to extracranial or intracranial stenosis. One patient was withdrawn by the investigator prior to data collection for not meeting inclusion criteria, and 5 withdrew consent immediately after randomization. Therefore, 440 patients were included in the final analysis (215 assigned to the intervention group and 225 to the control group), of whom 8 (1.8%) had the primary outcome imputed (**Figure 1**).

Table 1. Baseline Characteristics of Participating Patients

Characteristic	Intervention (n = 215)	Control (n = 225)
Age, mean (SD), y	75 (12)	75 (12)
Sex, No. (%)		
Female	119 (55.3)	114 (50.7)
Male	96 (44.7)	111 (49.3)
Prestroke mRS score, median (IQR)	0 (0-1)	0 (0-1)
Medical history, No. (%)[a]		
Hypertension	153 (71.2)	177 (78.7)
Diabetes	62 (31.3)	63 (31.3)
Hypercholesterolemia	119 (60.1)	132 (65.7)
Prior stroke	19 (9.6)	25 (12.4)
Coronary artery disease	24 (12.1)	27 (13.4)
Atrial fibrillation	62 (31.3)	78 (38.3)
Current smoking	29 (14.7)	24 (11.9)
Obesity[b]	25 (12.6)	34 (16.9)
Medication, No. (%)[c]		
Antihypertensive	139 (84.2)	160 (88.4)
Aspirin or other antiplatelet	42 (25.5)	33 (18.2)
Statin or other lipid-lowering drug	84 (50.9)	99 (54.7)
Anticoagulation	58 (35.2)	75 (41.4)
NIHSS score, median (IQR)	15 (9-19)	17 (11-21)
ASPECTS, median (IQR)	9 (8-10)	9 (8-10)
Site of intracranial occlusion, No. (%)		
M1 segment	140 (65.1)	138 (61.3)
M2 segment	59 (27.4)	55 (24.4)
Terminal internal carotid artery	15 (7.0)	30 (13.3)
A1 segment	1 (0.5)	2 (0.9)
Tandem occlusion, No. (%)[d]	17 (7.9)	12 (5.3)
CT perfusion performed, No. (%)	156 (72.6)	160 (71.1)
Use of intravenous thrombolysis, No. (%)	68 (31.6)	80 (35.6)
Passes during endovascular therapy, median (IQR), No.[e]	1 (1-2)	1 (1-2)
Use of carotid stenting, No. (%)	9 (4.2)	14 (6.2)
Use of intracranial stenting, No. (%)	6 (2.8)	2 (0.9)
Use of general anesthesia, No. (%)	113 (52.6)	114 (50.7)
Time from onset or last time seen well to reperfusion, median (IQR), h	5 (3-8)	5 (3-8)
mTICI score at end of procedure, No. (%)[f]		
2b	20 (9.3)	23 (10.2)
2c	69 (32.1)	68 (30.2)
3	126 (58.6)	134 (59.6)
Time from onset or last time seen well to randomization, median (IQR), h	6 (4-9)	5 (4-9)
Stroke etiology, No. (%)[g]		
Atherosclerosis	19 (9.4)	25 (11.9)
Cardioembolic	121 (59.6)	130 (61.6)
Other determined cause	4 (2.0)	3 (1.4)
Undetermined	59 (29.1)	53 (25.1)

Abbreviations: ASPECTS, Alberta Stroke Program Early Computed Tomography Score; CT, computerized tomography; mRS, modified Rankin Scale; mTICI, modified Thrombolysis in Cerebral Infarction; NIHSS, National Institutes of Health Stroke Scale.

[a] Data were available for 399 patients.

[b] Obesity was defined as a body mass index (calculated as weight in kilograms divided by height in meters squared) of 30 or higher.

[c] Data were available for 346 patients.

[d] Tandem occlusion was defined as an intracranial large-vessel occlusion associated with at least 70% concomitant stenosis or occlusion of the ipsilateral internal carotid artery.

[e] Data were available for 436 patients.

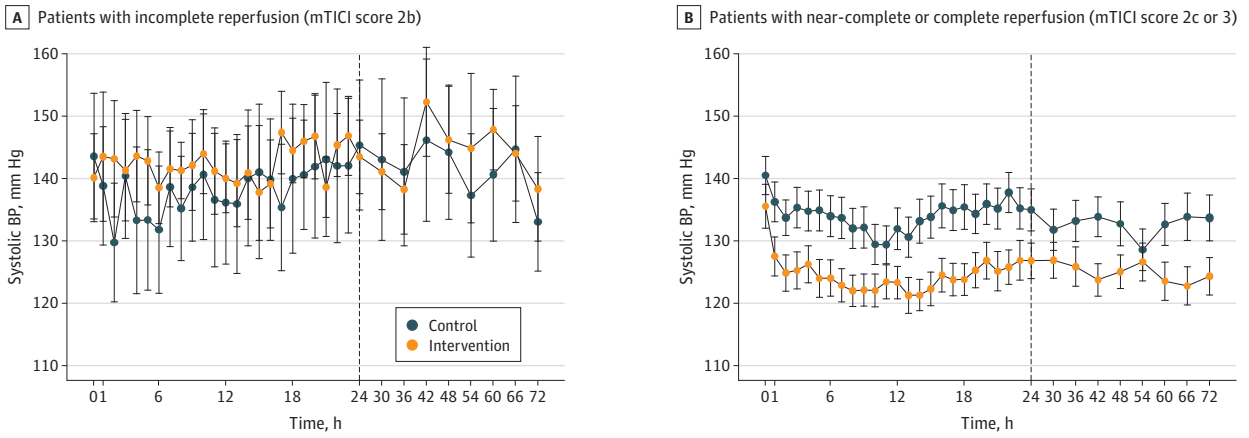
[f] The mTICI scale is an angiographic scale grading reperfusion after endovascular treatment (a score of 2b indicates 50%-89% reperfusion; a score of 2c, 90%-99% reperfusion; and a score of 3, 100% reperfusion).

[g] Data were available for 414 patients. The cause of stroke was assessed according to the diagnostic workup performed during hospitalization, including medical history, clinical features, imaging results, laboratory test results, and cardiac studies, including at least a transthoracic echocardiogram and 24-hour electrocardiographic recording.

Baseline demographic, clinical, imaging, and management characteristics were well balanced between the 2 groups, except for history of hypertension being more frequent in the control group (**Table 1**). The mean (SD) age was 75 (12) years, and 233 participants (53.0%) were female. The median severity of neurological impairment at baseline on the NIHSS was 16 (IQR, 10-20). After endovascular therapy, 43 participants (9.8%) were classified as having incomplete reperfusion (mTICI score of 2b) and 397 (90.2%) as having complete or near-complete reperfusion (mTICI scores of 2c or 3). Stroke was attributed to a cardioembolic source in 251 participants (60.6%), to atherosclerosis in 44 (10.6%; 39 extracranial and 5 intracranial), and to other determined causes in 7 (1.6%); 112 (25.5%) were considered embolic strokes of undetermined source.

The mean (SD) systolic BP was 150 (25) mm Hg at the time of admission and 138 (23) mm Hg before randomization. During the first 24 hours, the mean systolic BP was lower in the intervention group with near-complete or complete reperfusion (mTICI score of 2c or 3) compared with the control group (mean [SD], 125 [14] vs 134 [18] mm Hg, respectively; $P < .001$). Among patients with incomplete reperfusion (mTICI score of 2b), the mean (SD) systolic BP in the intervention group was 143 (6) mm Hg (**Figure 2**; eTable 3 in Supplement 3). The median (IQR) time-in-target ratio was 79% (63%-92%) in the intervention group (47% [39%-58%] in the mTICI 2b subgroup) and 100% (100%-100%) in the control group. Additional hemodynamic parameters are presented in eTable 3 in Supplement 3. A higher proportion of participants in the intervention group with available information received antihypertensive treatment to maintain the assigned systolic BP target compared with the control group (mTICI score of 2b: 11 of 13 [84.4%]; mTICI score of 2c or 3: 108 of 167 [64.8%]; control: 31 of 225 [25.2%]) (eTables 2 and 3 in Supplement 3). Similarly, vasopressor use among participants with available information was more frequent in the intervention group (mTICI score of 2b: 12 of 19 [63.2%]; mTICI score of 2c or 3: 16 of 142 [11.3%]) than in the control group (2 of 105 [1.9%]). No serious adverse events attributable to vasopressor use were identified. Over the 72-hour postrandomization study period, the mean systolic BP remained lower in the intervention group with an mTICI score of 2c or 3 than in either the control group or the intervention

Figure 2. Line Graphs Showing Patterns of Systolic Blood Pressure (BP)



Mean systolic BP is shown for patients with incomplete reperfusion (modified Treatment in Cerebral Infarction [mTICI] score of 2b) (A) and patients with near-complete or complete reperfusion (mTICI score of 2c or 3) (B). Error bars indicate SD.

Table 2. Primary, Secondary, and Safety Outcomes

Outcome	Participants, No. (%)		OR (95% CI)	*P* value
	Intervention (n = 215)	Control (n = 225)		
Primary outcome				
mRS score of 0-2 at 90 d[a]	129 (60.0)	105 (47.1)	1.71 (1.17-2.50)	.005
Adjusted[b]	NA	NA	1.71 (1.11-2.63)	.02
Secondary efficacy outcome, mRS score				
0, No symptoms at all	52 (24.2)	40 (17.8)	1.43 (1.03-2.00)	.03
1, No significant disability	36 (16.7)	35 (15.6)		
2, Slight disability	41 (19.1)	30 (13.3)		
3, Moderate disability	30 (14.0)	44 (19.6)		
4, Moderate to severe disability	20 (9.3)	33 (14.7)		
5, Severe disability	3 (1.4)	9 (4.0)		
6, Death	33 (15.3)	34 (15.1)		
Secondary safety outcomes				
Hemorrhagic transformation at 24 h	48 (22.3)	71 (31.6)	0.62 (0.41-0.95)	.03
sICH at 24 h[c]	7 (3.5)	8 (3.9)	0.89 (0.31-2.52)	.82
Any PH2 at 24 h[c]	7 (3.4)	9 (4.1)	0.81 (0.28-2.21)	.68
Neurological deterioration				
Within 24 h[d]	16 (7.6)	24 (10.9)	0.67 (0.35-1.30)	.24
Within 72 h[e]	14 (7.0)	13 (6.4)	1.10 (0.50-2.30)	.81
Serious adverse events				
Events reported, No.	46	37		
Patients with ≥1 serious adverse event	34 (15.8)	27 (12.0)	1.38 (0.80-2.37)	.25

Abbreviations: mRS, modified Rankin Scale; NA, not applicable; OR, odds ratio; PH2, parenchymal hemorrhage type 2; sICH, symptomatic intracerebral hemorrhage.

[a] Scores on the mRS of functional recovery range from 0 (no symptoms) to 6 (death); a score of 2 or less indicates functional independence.

[b] Adjusted for the following covariates: age, prior mRS score, baseline National Institutes of Health Stroke Scale, and time from onset to randomization.

[c] According to the Heidelberg bleeding classification.

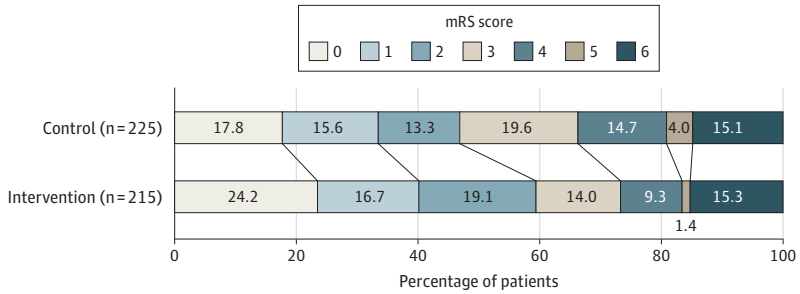
[d] Data were available for 431 patients.

[e] Data were available for 401 patients.

group with an mTICI score of 2b (eTables 3 and 4 in Supplement 3; Figure 2). In a linear mixed-effects model, there was a significant interaction between treatment group and mTICI score category. Pairwise comparisons showed that among patients with an mTICI score of 2c or 3, the intervention group achieved a mean systolic BP 9.0 mm Hg lower (95% CI, −12 to −6 mm Hg) than the control group ($P < .001$). In contrast, among patients with an mTICI score of 2b, the mean systolic BP was 3.0 mm Hg higher (95% CI, −6 to 12 mm Hg) in the intervention group compared with the control group ($P = .51$).

At 90 days, 129 of 215 participants (60.0%) in the intervention group and 106 of 225 participants (47.1%) in the control group achieved a favorable functional outcome, which corresponds to an absolute risk difference of 13.3% (95% CI, 4.1%-22.6%; $P = .005$) (**Table 2**). The corresponding odds ratio (OR) for a favorable outcome was 1.71 (95% CI, 1.17-2.50). In the adjusted sensitivity analysis, the effect size was similar (OR, 1.71; 95% CI, 1.11-2.63) (Table 2). In a sensitivity analysis using multiple imputation, the results were consistent (OR, 1.67; 95% CI, 1.14-2.45; $P = .008$). In the ordinal shift analysis, the

Figure 3. Bar Graph Showing Primary Functional Outcome Measured by Modified Rankin Scale (mRS) Score at 90 Days



common OR for a shift toward better mRS scores was 1.43 (95% CI, 1.03-2.00) (Table 2 and **Figure 3**). Efficacy outcomes in the per-protocol population were also consistent with the primary analysis and are presented in eTables 5 and 6 in Supplement 3.

Overall, no significant difference was identified between the intervention group and the control group with regard to serious adverse events (34 of 215 participants [15.8%] vs 27 of 225 participants [12.0%]) (Table 2). These events, categorized according to Medical Dictionary for Regulatory Activities (MedDRA) system organ classes and preferred terms, are reported in detail in eTable 9 in Supplement 3. The rate of hemorrhagic transformation at 24 hours was lower in the intervention group (48 of 215 participants [22.3%]) compared with the control group (71 of 225 participants [31.6%]) (OR, 0.62; 95% CI, 0.41-0.95). The rates of sICH (3.5% vs 3.9%) and 90-day mortality (15.4% vs 15.6%) did not differ between groups. The rates of early neurological deterioration were similar between the groups (Table 2).

In the prespecified subgroup analysis, there was no significant heterogeneity in the treatment effect (eTables 7 and 8 and eFigure 4 in Supplement 3).

Discussion

In this multicenter randomized clinical trial of patients with acute ischemic stroke of the anterior circulation who achieved successful reperfusion after endovascular therapy, a strategy of more intensive control of systolic BP tailored to reperfusion status led to an improved functional outcome and lower rate of hemorrhagic transformation than a more conservative management of systolic BP as recommended in guidelines. These findings should be interpreted with caution, given the reduced sample size due to early termination and the predominance of patients with excellent reperfusion (mTICI score of 2c or 3). Given that our findings contrast with those of previous randomized clinical trials, relevant methodological differences warrant careful consideration.

A key distinction of HOPE was the stratification of systolic BP target ranges according to final mTICI grade. Participants achieving near-complete or complete reperfusion (mTICI scores of 2c or 3) were assigned to a systolic BP range comparable to that used in previous trials (100-140 mm Hg), whereas those with incomplete reperfusion (mTICI score of 2b) had their systolic BP maintained at a higher range (140-160 mm Hg). This strategy was based on the pathophysiological premise that patients with incomplete reperfusion rely more heavily on their collateral circulation to sustain penumbral tissue.[21] Even so, the proportion of participants with an mTICI score of 2b was substantially lower in HOPE than in the BP-TARGET (Safety and Efficacy of Intensive Blood Pressure Lowering After Successful Endovascular Therapy in Acute Ischaemic Stroke)[12] and BEST-II (Blood Pressure Management After Endovascular Therapy for Acute Ischemic Stroke)[15] trials, which reported neutral results (10% vs 40%-45%). This proportion was, however, comparable to that observed in ENCHANTED2/MT,[13] in which intensive BP lowering was associated with harm. Moreover, subgroup analysis of HOPE showed no clear benefit of the intervention in participants with an mTICI score of 2b. In light of these findings, the intended reperfusion-stratified BP strategy may not have been fully achieved in practice, suggesting that the benefit observed in HOPE is unlikely to be solely attributable to the stratified approach to systolic BP control.

Other protocol distinctions were the implementation of rigorous BP monitoring over a longer period (72 hours), a defined lower systolic BP threshold for safety in both groups, and management strategies for hypotension including the use of vasopressors to maintain the target ranges as required. These aspects were driven by concerns that excessive reductions in systolic BP might compromise collateral perfusion and promote infarct expansion.[27] However, the impact of vasopressor support is likely to have been limited as it was only applied to a small proportion of participants.

Another unique feature of HOPE was the enrollment criteria, where participants considered at high risk of hemodynamic infarction, primarily of intracranial or extracranial stenosis, were excluded. Consequently, the study population had a lower frequency of large-artery atherosclerosis compared with other trials, particularly those involving patients in Asia where the rate of this type of cerebrovascular disease is especially high (9%-12% in HOPE vs 49%-56% in ENCHANTED2/MT[13]). These patients are known to exhibit impaired cerebral autoregulation and reduced hemodynamic reserve.[28,29] HOPE therefore enrolled a substantially higher proportion of patients with cardioembolic strokes (60.6%) than in ENCHANTED2/MT (28%),

where observational data show that BP lowering was associated with improved outcomes.[30]

The profile of lower baseline and early postprocedural BP may have contributed to the treatment effect in HOPE as they have consistently been associated with better functional outcome.[31] Baseline systolic BP values were lower than those reported in OPTIMAL-BP (Intensive vs Conventional Blood Pressure Lowering After Endovascular Thrombectomy in Acute Ischemic Stroke)[14] and ENCHANTED2/MT,[13] likely due to an elevated systolic BP not being required for inclusion. This may have contributed to participants being selected with a greater preservation of cerebral autoregulation. In addition, the slightly lower prevalence of chronic hypertension in the intervention group compared with the control group may have contributed to a greater tolerance to BP lowering. Although these differences were modest, we cannot exclude the possibility that they may have influenced the observed treatment effects.

Finally, the intervention was associated with a lower incidence of hemorrhagic transformation, which is consistent with prior observational data suggesting this outcome is associated with even modest reductions in systolic BP after endovascular therapy.[10,32] Conversely, the rate of sICH was similar between the groups and comparable to that reported in previous trials. These results support the outcome of any hemorrhagic transformation being a suitable measure of safety in endovascular trials, as accumulating evidence indicates this negatively influences functional recovery regardless of immediate clinical impact.[33,34] From a physiological perspective, the reduction in hemorrhagic events observed in HOPE supports the hypothesis that controlled systolic BP management mitigates reperfusion. However, these findings should be interpreted with caution, as they differ from those of prior randomized clinical trials and may reflect a type I error due to limited sample size. Differences in outcome definitions and centralized adjudication may also have contributed to this discrepancy.

Strengths

Key strengths of this trial include the pragmatic, multicenter, randomized, controlled design with rigorous data-monitoring procedures for complete verification of critical variables and source data. The outcome assessment was masked and there was minimal loss to follow-up at 3 months, which supports the robustness of the primary end point. Despite having to stop prematurely, HOPE represents the second largest study to date, to our knowledge, evaluating BP control after endovascular therapy, and it incorporated several distinctive methodological features, including systolic BP targets tailored to reperfusion status, protocolized hemodynamic monitoring, and predefined strategies for the management of hypotension. These elements enhance internal validity and consistency of the findings.

Limitations

Several limitations should be acknowledged. First, the trial did not achieve the planned sample size due to funding constraints. Although the achieved sample still provided adequate power for detection of a larger-than-expected treatment effect, there is still the potential for the influence of chance and imprecision in the estimate, overall and across subgroups. Second, participants with incomplete reperfusion (mTICI score of 2b) were underrepresented, potentially introducing selection bias and limiting the interpretability of subgroup findings. In addition, there were numerical imbalances in baseline prognostic variables between groups, including a higher proportion of terminal internal carotid artery occlusions and slightly higher NIHSS scores in the control group, which may have contributed to outcome differences. Third, the exclusion of participants with intracranial or extracranial vasculopathy that was not successfully revascularized may also restrict the generalizability of the results to populations without significant stenosis. Fourth, although the protocol included predefined guidance for BP augmentation, only a small proportion of participants required vasopressor therapy. Accordingly, HOPE was not designed or powered to evaluate the clinical benefit of active BP augmentation strategies. Fifth, the use of different antihypertensive agents may have introduced additional variability in cerebrovascular physiology and represents a potential confounder. In addition, differences in the frequency of BP monitoring between groups may have influenced the intensity of BP management, although the high time-in-target ratio observed in the control group suggests this effect may have been limited. Finally, as the trial was conducted within a specific geographical region, extrapolation of these findings to populations with different baseline characteristics should be undertaken with caution. Taken together, these limitations suggest that the findings should be interpreted as hypothesis generating rather than practice changing, although they provide important insights into methodological features that should inform the design of future powered trials in this area. From a mechanistic perspective, the results do not alter current indications for adjunctive therapies after endovascular treatment, including intra-arterial thrombolysis, but rather appear to be complementary to emerging reperfusion strategies. They raise the hypothesis that optimized hemodynamic management may act synergistically with reperfusion strategies to further improve functional outcomes.

Conclusions

This multicenter randomized clinical trial found that in patients with acute ischemic stroke with large-vessel occlusion of the anterior circulation who achieved successful reperfusion after endovascular therapy, a tailored postprocedural systolic BP control strategy led to improved functional outcome and a lower rate of hemorrhagic transformation. The distinctive methodological features and population characteristics of HOPE, as compared with previous neutral or negative trials, suggest that the optimal BP management after endovascular therapy requires a nuanced and individualized approach.

ARTICLE INFORMATION

Accepted for Publication: April 25, 2026.

Published Online: June 8, 2026. doi:10.1001/jamaneurol.2026.1706

Author Affiliations: Stroke Unit, Department of Neurology, Hospital de la Santa Creu i Sant Pau, Institut de Recerca Sant Pau, Barcelona, Spain (Camps-Renom, Guasch-Jiménez, Fernández-Vidal, Martínez-Domeño, Pérez de la Ossa, Ramos-Pachón, Aguilera-Simón, Marín, Ezcurra-Díaz, Lambea-Gil, Prats-Sánchez, Martí-Fàbregas); Stroke Unit, Department of Neurology, Hospital Dr Josep Trueta, Girona, Spain (Álvarez-Cienfuegos, Silva); Neurology Department, Dr Balmis General University Hospital, Alicante Institute for Health and Biomedical Research, Alicante, Spain (López-Hernández, Corona-García); Stroke Unit, Department of Neurology, Hospital del Mar, Neurovascular Research Group, Hospital del Mar Research Institute, Barcelona, Spain (Rodríguez-Campello, Giralt-Steinhauer); Stroke Unit, Department of Neurology, Hospital Universitario Miguel Servet, Zaragoza, Spain (Tejada-Meza, Marta-Moreno); Grupo de Investigación en Neurociencias, Instituto de Investigación Sanitaria Aragón, Zaragoza, Spain (Tejada-Meza, Marta-Moreno); Interventional Neuroradiology Unit, Department of Radiology, Hospital Universitario Miguel Servet, Zaragoza, Spain (Tejada-Meza, Marta-Moreno); Stroke Unit, Department of Neurology, Complejo Asistencial Universitario de Salamanca, Salamanca, Spain (López-Mesonero, Vizcaya-Gaona); Stroke Unit, Department of Neurology, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biosanitaria Pascual Parrilla, Murcia, Spain (Albert-Lacal, Sanz-Monllor); Biobizkaia Health Research Institute, Grupo Neurovascular, Servicio Neurología, Hospital Universitario Cruces, Barakaldo, Spain (Freijo-Guerrero, Luna); Stroke Unit, Hospital Universitario La Fe, Valencia, Spain (Tarruella-Hernández, López Morales); Stroke Unit, Department of Neurology, Hospital Universitari de Tarragona Joan XXIII, Institut d'Investigació Sanitaria Pere Virgili, Tarragona, Spain (Flores, Ustrell); Department of Neurology, Stroke Unit, Instituto de Biomedicina de Sevilla, Hospital Universitario Virgen Del Rocío, Consejo Superior de Investigaciones Científicas, Universidad de Sevilla, Seville, Spain (Cabezas-Rodríguez, Moniche); Servei d'Epidemiologia Clínica i Salut Pública, Institut de Recerca Sant Pau, Universitat Autònoma de Barcelona, Barcelona, Spain (Solà-Roca); The George Institute for Global Health, University of New South Wales, Sydney, New South Wales, Australia (Wang, Anderson); Institute for Science and Technology for Brain-Inspired Intelligence, Fudan University, Shanghai, China (Anderson); Neurology Department, Royal Prince Alfred Hospital, Sydney, New South Wales, Australia (Anderson).

Author Contributions: Dr Camps-Renom had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Drs Camps-Renom and Guasch-Jiménez contributed equally to this work.
Concept and design: Camps-Renom, Freijo-Guerrero, Marta-Moreno, Solà-Roca, Anderson, Prats-Sánchez, Martí-Fàbregas.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Camps-Renom, Guasch-Jiménez, Aguilera-Simón, Corona-García, Prats-Sánchez, Martí-Fàbregas.
Critical review of the manuscript for important intellectual content: Camps-Renom, Álvarez-Cienfuegos, López-Hernández, Rodríguez-Campello, Tejada-Meza, López-Mesonero, Albert-Lacal, Freijo-Guerrero, Tarruella-Hernández, Flores, Cabezas-Rodríguez, Fernández-Vidal, Martínez-Domeño, Pérez de la Ossa, Ramos-Pachón, Marín, Ezcurra-Díaz, Lambea-Gil, Silva, Giralt-Steinhauer, Marta-Moreno, Vizcaya Gaona, Sanz-Monllor, Luna, López Morales, Ustrell, Moniche, Solà-Roca, Wang, Anderson, Prats-Sánchez, Martí-Fàbregas.
Statistical analysis: Camps-Renom, Fernández-Vidal, Ramos-Pachón, Solà-Roca, Wang.
Obtained funding: Camps-Renom, López-Mesonero, Martí-Fàbregas.
Administrative, technical, or material support: Camps-Renom, Guasch-Jiménez, Tejada-Meza, Fernández-Vidal, Aguilera-Simón, Marín, Ezcurra-Díaz, Lambea-Gil, Silva, Giralt-Steinhauer, Marta-Moreno, López Morales, Moniche, Anderson, Prats-Sánchez.
Supervision: Camps-Renom, Guasch-Jiménez, Rodríguez-Campello, López-Mesonero, Freijo-Guerrero, Cabezas-Rodríguez, Pérez de la Ossa, Ramos-Pachón, Silva, Marta-Moreno, Vizcaya Gaona, Ustrell, Moniche, Anderson, Prats-Sánchez, Martí-Fàbregas.

Conflict of Interest Disclosures: Dr Camps-Renom reported grants from the Instituto de Salud Carlos III (ISC III), Ministerio de Ciencia e Innovación, and Fondo Europeo de Desarrollo Regional (FEDER) during the conduct of the study; and personal fees from Daiichi Sankyo and Amgen and being a member of the Spanish Society of Neurology, Catalan Society of Neurology, and European Stroke Organization outside the submitted work. Dr Guasch-Jiménez reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study; and grants from Beca Proyecto Ictus of the Spanish Society of Neurology, personal fees from Daiichi Sankyo, and Bial, and being a member of the Spanish Society of Neurology and European Stroke Organization outside the submitted work. Dr Albert-Lacal reported personal fees from Daiichi Sankyo, Bayer, Shionogi, Jazz Pharmaceuticals, and KRKA outside the submitted work. Dr Ramos-Pachón reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study. Ms Aguilera-Simón reported grants from ISC III, Ministerio de Ciencia e Innovación, FEDER, and Redes de Investigación Cooperativa Orientadas a Resultado en Salud (RICORS-ICTUS) during the conduct of the study. Dr Ezcurra-Díaz reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study; and being a member of the Spanish Society of Neurology, Catalan Society of Neurology, and European Stroke Organization outside the submitted work. Dr Lambea-Gil reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study; and personal fees from Bial, Daiichi Sankyo, UCB Pharma, and Spanish Society of Neurology and being a member of the Spanish Society of Neurology, European Academy of Neurology, and European Stroke Organisation outside the submitted work. Dr Sanz-Monllor reported personal fees from KRKA, Daiichi Sankyo, Bayer, Esteve, Alter, and Novartis and being a member of the Spanish Society of Neurology and the Murcian Society of Neurology outside the submitted work. Ms Solà-Roca reported personal fees from Fundació Institut de Recerca de l'Hospital de la Santa Creu i Sant Pau during the conduct of the study. Dr Anderson reported grants from the National Health and Medical Research Council of Australia and Medical Research Foundation of the UK and personal fees from Auzone BioTech Shanghai, Merck, AstraZeneca, and Bayer outside the submitted work. Dr Prats-Sánchez reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study. Dr Martí-Fàbregas reported grants from ISC III, Ministerio de Ciencia e Innovación, and FEDER during the conduct of the study. No other disclosures were reported.

Funding/Support: The HOPE trial has been funded by ISC III, Ministerio de Ciencia e Innovación (ICI20/00137), and FEDER. The HOPE team has been supported by RICORS-ICTUS, ISC III, and Ministerio de Ciencia e Innovación (RD21/0006/0006 and RD24/0009/0010). This study was funded by ISC III and the Ministerio de Ciencia e Innovación (ICI20/000137, RD21/0006/0006, and RD24/0009/0010).

Role of the Funder/Sponsor: The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Group Information: A list of the HOPE Study Group members appears in Supplement 4.

Data Sharing Statement: See Supplement 5.

Additional Contributions: We acknowledge Yaira Mathison, BSc (Institut de Recerca Sant Pau), for coordination of trial monitoring and review of serious adverse events; she received no compensation. We thank Juan Arenillas, PhD (Hospital Clínico Universitario de Valladolid), and Mònica Millán, PhD (Hospital Germans Trias I Pujol), for their role as members of the data and safety monitoring board and for conducting the interim analyses; they received no compensation. We thank all members of the HOPE Study Group for their contributions to patient recruitment, data collection, and study conduct. We are grateful to the nursing teams of the stroke units and critical care departments for their support in blood pressure measurements throughout the study. Finally, we sincerely thank all patients and their families for their participation in this clinical trial.

REFERENCES

1. Goyal M, Menon BK, van Zwam WH, et al; HERMES Collaborators. Endovascular thrombectomy after large-vessel ischaemic stroke:

a meta-analysis of individual patient data from five randomised trials. *Lancet*. 2016;387(10029):1723-1731. doi:10.1016/S0140-6736(16)00163-X

2. Ghozy S, Kacimi SEO, Azzam AY, et al. Successful mechanical thrombectomy in acute ischemic stroke: revascularization grade and functional independence. *J Neurointerv Surg*. 2022;14(8):779-782. doi:10.1136/neurintsurg-2021-018436

3. Zaidat OO, Fifi JT, Nanda A, et al; COMPLETE Registry Investigators. Endovascular treatment of acute ischemic stroke with the Penumbra system in routine practice: COMPLETE registry results. *Stroke*. 2022;53(3):769-778. doi:10.1161/STROKEAHA.121.034268

4. Nie X, Leng X, Miao Z, Fisher M, Liu L. Clinically ineffective reperfusion after endovascular therapy in acute ischemic stroke. *Stroke*. 2023;54(3):873-881. doi:10.1161/STROKEAHA.122.038466

5. Khatri R, McKinney AM, Swenson B, Janardhan V. Blood-brain barrier, reperfusion injury, and hemorrhagic transformation in acute ischemic stroke. *Neurology*. 2012;79(13)(suppl 1):S52-S57. doi:10.1212/WNL.0b013e3182697e70

6. Zhou Y, He Y, Yan S, et al. Reperfusion injury is associated with poor outcome in patients with recanalization after thrombectomy. *Stroke*. 2023;54(1):96-104. doi:10.1161/STROKEAHA.122.039337

7. Ng FC, Churilov L, Yassi N, et al. Prevalence and significance of impaired microvascular tissue reperfusion despite macrovascular angiographic reperfusion (no-reflow). *Neurology*. 2022;98(8):e790-e801. doi:10.1212/WNL.0000000000013210

8. Sperring CP, Savage WM, Argenziano MG, et al. No-reflow post-recanalization in acute ischemic stroke: mechanisms, measurements, and molecular markers. *Stroke*. 2023;54(9):2472-2480. doi:10.1161/STROKEAHA.123.044240

9. Goyal N, Tsivgoulis G, Pandhi A, et al. Blood pressure levels post mechanical thrombectomy and outcomes in large vessel occlusion strokes. *Neurology*. 2017;89(6):540-547. doi:10.1212/WNL.0000000000004184

10. Katsanos AH, Malhotra K, Ahmed N, et al. Blood pressure after endovascular thrombectomy and outcomes in patients with acute ischemic stroke: an individual patient data meta-analysis. *Neurology*. 2022;98(3):e291-e301. doi:10.1212/WNL.0000000000013049

11. Mistry EA, Mistry AM, Nakawah MO, et al. Systolic blood pressure within 24 hours after thrombectomy for acute ischemic stroke correlates with outcome. *J Am Heart Assoc*. 2017;6(5):e006167. doi:10.1161/JAHA.117.006167

12. Mazighi M, Richard S, Lapergue B, et al; BP-TARGET Investigators. 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(4):265-274. doi:10.1016/S1474-4422(20)30483-X

13. Yang P, Song L, Zhang Y, et al; ENCHANTED2/MT Investigators. 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(10363):1585-1596. doi:10.1016/S0140-6736(22)01882-7

14. Nam HS, Kim YD, Heo J, et al; OPTIMAL-BP Trial Investigators. Intensive vs conventional blood pressure lowering after endovascular thrombectomy in acute ischemic stroke: the OPTIMAL-BP randomized clinical trial. *JAMA*. 2023;330(9):832-842. doi:10.1001/jama.2023.14590

15. Mistry EA, Hart KW, Davis LT, et al. Blood pressure management after endovascular therapy for acute ischemic stroke: the BEST-II randomized clinical trial. *JAMA*. 2023;330(9):821-831. doi:10.1001/jama.2023.14330

16. Katsanos AH, Catanese L, Sahlas DJ, et al. Blood pressure management following endovascular stroke treatment: a feasibility trial and meta-analysis of outcomes. *Stroke Vasc Interv Neurol*. 2024;4(3):e001287. doi:10.1161/SVIN.123.001287

17. Zhang X, Cao L, Wu S, et al; IDENTIFY Trial Investigators. Early intensive blood pressure management after endovascular treatment in ischaemic stroke (IDENTIFY): a multicentre, open-label, blinded-endpoint, randomised controlled trial. *Lancet Reg Health West Pac*. 2025;59:101589. doi:10.1016/j.lanwpc.2025.101589

18. Gharaibeh K, Aladamat N, Mierzwa AT, et al. Blood pressure after successful endovascular therapy: a systematic review and meta-analysis of randomized control trials. *Ann Neurol*. 2024;95(5):858-865. doi:10.1002/ana.26907

19. Song L, Mao Y, Sandset EC, et al. Early blood pressure management in acute ischaemic stroke before and after reperfusion treatment. *Lancet Neurol*. 2026;25(2):206-212. doi:10.1016/S1474-4422(25)00378-3

20. Prabhakaran S, Gonzalez NR, Zachrison KS, et al; Peer Review Committee. 2026 Guideline for the early management of patients with acute ischemic stroke: a guideline from the American Heart Association/American Stroke Association. *Stroke*. Published online January 26, 2026. doi:10.1161/STR.0000000000000513

21. Jeong HG, Kim BJ, Kim H, et al. Blood pressure drop and penumbral tissue loss in nonrecanalized emergent large vessel occlusion. *Stroke*. 2019;50(10):2677-2684. doi:10.1161/STROKEAHA.119.025426

22. Camps-Renom P, Guasch-Jiménez M, Martínez-Domeño A, et al. A randomised trial on haemodynamic optimisation of cerebral perfusion after successful endovascular therapy in patients with acute ischemic stroke (HOPE). *Cerebrovasc Dis*. 2025;54(4):559-566. doi:10.1159/000540606

23. Camps-Renom P, Guasch-Jiménez M, Pérez de la Ossa N, Solà-Roca J, Martí-Fàbregas J. Statistical analysis plan for the HOPE trial: a randomised trial on haemodynamic optimisation of cerebral perfusion after endovascular therapy in patients with acute ischemic stroke. *medRxiv*. Preprint posted December 17, 2025. doi:10.64898/2025.12.16.25342410

24. Hopewell S, Chan AW, Collins GS, et al. CONSORT 2025 statement: updated guideline for reporting randomized trials. *JAMA*. 2025;333(22):1998-2005. doi:10.1001/jama.2025.4347

25. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2019;50(12):e344-e418. doi:10.1161/STR.0000000000000211

26. von Kummer R, Broderick JP, Campbell BCV, et al. The Heidelberg bleeding classification: classification of bleeding events after ischemic stroke and reperfusion therapy. *Stroke*. 2015;46(10):2981-2986. doi:10.1161/STROKEAHA.115.010049

27. Galecio-Castillo M, Quispe-Orozco D, Farooqui M, et al. Association between intraprocedural drops in blood pressure and infarct growth rate patterns after acute large-vessel occlusions. *J Neurointerv Surg*. 2024;16(12):1275-1281. doi:10.1136/jnis-2023-020899

28. Sheriff FG, Ahmad A, Inam ME, Khatri R, Maud A, Rodriguez GJ. A systematic review on the assessment of cerebral autoregulation in patients with large vessel occlusion. *Front Neurol*. 2023;14:1287873. doi:10.3389/fneur.2023.1287873

29. Liu Y, Li S, Tian X, et al. Cerebral haemodynamics in symptomatic intracranial atherosclerotic disease: a narrative review of the assessment methods and clinical implications. *Stroke Vasc Neurol*. 2023;8(6):521-530. doi:10.1136/svn-2023-002333

30. Anadani M, Arthur AS, Alawieh A, et al. Blood pressure reduction and outcome after endovascular therapy with successful reperfusion: a multicenter study. *J Neurointerv Surg*. 2020;12(10):932-936. doi:10.1136/neurintsurg-2019-015561

31. Petersen NH, Kodali S, Meng C, et al. Blood pressure trajectory groups and outcome after endovascular thrombectomy: a multicenter study. *Stroke*. 2022;53(4):1216-1225. doi:10.1161/STROKEAHA.121.034408

32. Anadani M, Arthur AS, Tsivgoulis G, et al. Blood pressure goals and clinical outcomes after successful endovascular therapy: a multicenter study. *Ann Neurol*. 2020;87(6):830-839. doi:10.1002/ana.25716

33. Guasch-Jiménez M, Ezcurra Díaz G, Lambea-Gil Á, et al; Catalan Stroke Code and Reperfusion Consortium (CICAT). Influence of asymptomatic hemorrhagic transformation after endovascular treatment on stroke outcome: a population-based study. *Neurology*. 2025;104(9):e213509. doi:10.1212/WNL.0000000000213509

34. Wang Y, Maeda T, You S, et al; ENCHANTED Investigators. Patterns and clinical implications of hemorrhagic transformation after thrombolysis in acute ischemic stroke: results from the ENCHANTED study. *Neurology*. 2024;103(11):e210020. doi:10.1212/WNL.0000000000210020