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Blood Pressure Management in Acute Ischemic Stroke

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ABSTRACT

Acute Ischemic Stroke (AIS) is a neurological emergency caused by a sudden reduction in cerebral blood flow due to arterial obstruction, commonly triggered by thrombosis or embolism. Optimal blood pressure management plays a critical role in preventing secondary brain injury, improving perfusion, and reducing the risk of complications such as recurrent ischemia or hemorrhagic transformation. This literature review aims to analyze current evidence and clinical guidelines regarding blood pressure control in AIS patients, both those undergoing reperfusion therapy and those receiving standard medical treatment. The review highlights the 2019 AHA/ASA recommendations emphasizing early diagnosis, reperfusion decision-making, and individualized blood pressure targets based on clinical conditions. In AIS patients without reperfusion therapy, a gradual reduction in blood pressure is considered safe only when systolic levels exceed 220/120 mmHg, whereas tighter control is required for patients receiving IV-tPA or mechanical thrombectomy to minimize bleeding risk. Various antihypertensive agents—including labetalol, nicardipine, hydralazine, enalaprilat, and sodium nitroprusside—are discussed based on onset, safety, and clinical suitability. Overall, the review underscores the importance of tailored blood pressure management as a key component in AIS treatment to optimize neurological outcomes and reduce mortality.

Keywords: Acute ischemic stroke, blood pressure management, reperfusion therapy, antihypertensive agents, AHA/ASA guidelines, cerebral perfusion

Introduction

Acute ischemic stroke (AIS), or non-hemorrhagic stroke, is a disruption of the brain's blood supply due to a blockage in an artery or vein (Yuliastuti, Faridah, Jauhar, & Kanan, 2025). This blockage can be caused by thrombosis (rupture of an atherosclerotic plaque, vascular dissection) or embolism (air, clots, or fat). Non-hemorrhagic strokes are classified into subtypes: cardio embolism, small vessel occlusion, large artery atherosclerosis, and stroke of unknown etiology (Greco, Raffo, Cutore, & Capodanno, 2025; Kolios, Barkas, Ntaios, & Milionis, 2025). Stroke symptoms can be characterized by focal neurological deficits based on the area of the brain and bone marrow affected by the blockage (Taksande, Taksande, & Malik, 2025). Stroke is the second leading cause of death worldwide, with an estimated incidence of seven million (Feigin et al., 2025; Venketasubramanian, 2025). The World Stroke Organization (WSO) states that one in four adults aged 25 and over will experience a stroke during their lifetime (Parry-Jones et al., 2025). More than 100 million people experience stroke symptoms, with 60 percent of stroke patients

under 70 and 16 percent under 50.

This type of stroke occurs chronically and slowly due to several risk factors, including high blood pressure, diabetes mellitus, dyslipidemia, atrial fibrillation, smoking, heart disease (heart failure, coronary heart disease), obesity, age over 55, and chronic kidney disease. High blood pressure is said to increase the risk of stroke by 2-4 times, through the mechanism of atherosclerotic plaque formation, thickening and changes in blood vessel structure, and causing acute symptoms if thrombus formation occurs due to endothelial damage . In patients with acute *AIS*, blood pressure management requires a complex therapeutic approach (Liao et al., 2025). Cerebral autoregulation will cause an increase in blood pressure after *AIS* to maximize perfusion to the ischemic area of the brain (Troudi Habibi, Micaux, Mauconduit, & Noulhiane, 2025).

There are several conditions that need to be considered, such as the onset of the stroke event, the reperfusion therapy plan, the blood pressure target, and the modalities used in post-*AIS* blood pressure management (Nartea, Savulescu, Potcovaru, & Poenaru, 2025; Rong et al., 2025). Appropriate blood pressure management will prevent further blood vessel damage, prevent recurrent cerebral ischemia (recurrent stroke), and prevent other cerebrovascular complications (such as hemorrhagic stroke, hypertensive encephalopathy, and cardiorenal complications) (Cozza & Boccardi, 2025). Below, the author will conduct a literature review related to blood pressure management in patients with acute-onset non-hemorrhagic stroke (Han et al., 2025).

Previous research on blood pressure management in *AIS* has yielded several major guidelines and systematic reviews (Zhang et al., 2025). The American Heart Association/American Stroke Association (AHA/ASA) 2019 guidelines provide comprehensive recommendations for early management of *AIS* patients, emphasizing individualized blood pressure targets based on reperfusion therapy status . The European Stroke Organisation (ESO) 2021 guidelines similarly address blood pressure control strategies, though with some variations in threshold recommendations (Berge et al., 2021; Rubiera et al., 2022; Steiner et al., 2025). A systematic review by Bath et al. (2022) in Stroke journal analyzed blood pressure management within the first 24 hours, highlighting the delicate balance between preventing hemorrhagic transformation and maintaining cerebral perfusion. Additionally, the Taiwan Stroke Society 2020 guidelines offer region-specific insights into optimal blood pressure targets during the acute phase.

Despite these comprehensive guidelines, several knowledge gaps remain, particularly regarding optimal blood pressure targets in specific patient subgroups, the comparative efficacy of different antihypertensive agents in the acute setting, and the management of blood pressure variability. Furthermore, there is ongoing debate about permissive hypertension versus early aggressive control in patients not receiving reperfusion therapy, with conflicting evidence from trials such as CATIS and SCAST (Sultan et al., 2024; Van Dorst et al., 2021). These inconsistencies, combined with limited data from resource-limited settings where advanced monitoring may be unavailable, necessitate a comprehensive synthesis of current evidence (Alsabri et al., 2025; Dangi, Sharma, & Vageriya, 2025).

The urgency of this review stems from several critical factors. First, blood pressure management guidelines for *AIS* are continuously evolving as new clinical trial data emerge, requiring clinicians to stay updated with the latest evidence-based recommendations. Second, inappropriate blood pressure management in the acute phase can lead to devastating consequences, including hemorrhagic transformation, cerebral edema, or extension of ischemic

injury, making this a time-sensitive clinical priority. Third, in resource-limited settings where access to advanced stroke care and continuous monitoring may be restricted, clinicians need clear, practical guidance on blood pressure management that can be applied with available resources. Finally, with the increasing availability of reperfusion therapies globally, there is a pressing need for standardized approaches to blood pressure control that optimize outcomes while minimizing complications.

The novelty of this review lies in its comprehensive synthesis of recent evidence (2019-2025) following major guideline updates, with a specific focus on practical clinical application. Unlike previous reviews that primarily summarize individual guidelines, this review provides direct comparisons of pharmacological agents used in *AIS* blood pressure management, evaluating their onset of action, safety profiles, and suitability for different clinical scenarios. Furthermore, this review integrates recommendations from multiple international guidelines (AHA/ASA, ESO, Taiwan Stroke Society) to identify consensus areas and highlight remaining controversies. The review also addresses the practical considerations for clinicians in various healthcare settings, including the selection of appropriate antihypertensive agents based on availability and monitoring capabilities, making it particularly relevant for real-world clinical practice.

The primary objective of this literature review is to provide a comprehensive, evidence-based analysis of blood pressure management strategies in acute ischemic stroke patients. Specifically, this review aims to: (1) synthesize current international guidelines and recent clinical evidence regarding optimal blood pressure targets in *AIS* patients with and without reperfusion therapy; (2) compare the pharmacological properties, efficacy, and safety profiles of commonly used antihypertensive agents in the acute stroke setting; and (3) identify practical considerations for blood pressure management in diverse clinical environments. The benefits of this review include providing clinicians with a clear, updated reference for blood pressure management decisions in *AIS*, potentially improving treatment standardization across different healthcare settings, and ultimately contributing to better neurological outcomes and reduced mortality in stroke patients. The implications extend to clinical practice guidelines, medical education, and future research directions in acute stroke care.

Research Method

This literature review was conducted using a systematic approach to identify, evaluate, and synthesize relevant evidence on blood pressure management in acute ischemic stroke. A comprehensive literature search was performed using multiple electronic databases including PubMed, Google Scholar, and Scopus, covering publications from 2019 to 2025 to capture the most recent evidence following major guideline updates. The search strategy employed a combination of Medical Subject Headings (MeSH) terms and keywords, including "acute ischemic stroke," "blood pressure management," "hypertension," "reperfusion therapy," "thrombolysis," "mechanical thrombectomy," and "antihypertensive agents."

The inclusion criteria for this review encompassed: (1) clinical practice guidelines from major international stroke organizations (AHA/ASA, ESO, Taiwan Stroke Society); (2) systematic reviews and meta-analyses examining blood pressure management in AIS; (3) randomized controlled trials and observational studies investigating blood pressure targets and antihypertensive interventions in acute stroke patients; and (4) publications in English with full-

text availability. Exclusion criteria included case reports, conference abstracts without full manuscripts, and studies focusing exclusively on hemorrhagic stroke or chronic stroke management.

Selected articles were critically appraised for methodological quality, relevance, and level of evidence. Data extraction focused on blood pressure target recommendations, timing of intervention, types of antihypertensive agents used, clinical outcomes (mortality, disability, hemorrhagic transformation), and safety considerations. The extracted information was synthesized narratively, organizing findings by clinical scenario (with versus without reperfusion therapy) and therapeutic approach. Particular attention was given to identifying areas of consensus across guidelines as well as highlighting ongoing controversies or knowledge gaps in the field.

Results and Discussion

Guideline for AIS Therapy

In the 2019 AHA/ASA guidelines, the management of AIS emphasizes the onset of events and prompt and appropriate management of patients with AIS.8 The in-hospital examination system emphasizes establishing a diagnosis using a non-contrast head CT scan. Other essential tests include a complete blood count, blood clotting factor analysis, and blood sugar levels. The outcome of optimal examination and diagnosis is to determine reperfusion therapy steps.9 Thrombolytic therapy, or IV tissue plasminogen activator (IV-tPA) or IV alteplase, is recommended within 3–4.5 hours of onset. The use of IV Tenecteplase as an alternative to alteplase is recommended for patients planning mechanical thrombectomy.10

Another recommended reperfusion therapy is mechanical thrombectomy, with the optimal time of action being 6–24 hours after onset. However, mechanical thrombectomy has certain clinical and diagnostic criteria for optimal performance. Reperfusion therapy is considered the primary treatment for brain tissue obstruction in AIS. Secondary prevention of AIS includes ant thrombolytics (aspirin, dual antiplatelet therapy (DAPT), and anticoagulants), seizure management, glucose control, cardiac function monitoring, and regular blood pressure management. Specialized monitoring in a stroke unit and ongoing rehabilitation are also essential therapies in the management of AIS patients.11

Blood Pressure Management In AIS Without Reperfusion Therapy

Based on the 2019 AHA/ASA guidelines, routine management of AIS is not recommended if blood pressure does not exceed 220/120 mmHg in the first 24-48 hours after onset, and there are no comorbidities or other worsening that require immediate antihypertensive therapy (coronary heart disease, acute decompensated heart failure, aortic dissection, post-fibrinolysis hemorrhagic stroke, pre-eclampsia or eclampsia, and hypertensive emergency).11 The rationale for permissive hypertension in this context relates to the preservation of cerebral perfusion pressure in the setting of impaired autoregulation. When cerebral autoregulation is disrupted following ischemic injury, cerebral blood flow becomes pressure-dependent, and elevated systemic blood pressure may help maintain adequate perfusion to the ischemic penumbra.

If blood pressure is <220/120 mmHg, a gradual decrease in blood pressure within 48-72 hours after onsen is said to be safe, but does not simultaneously reduce mortality or therapeutic outcomes of AIS patients. This recommendation is supported by clinical trial evidence from

studies such as CATIS (China Antihypertensive Trial in Acute Ischemic Stroke) and SCAST (Scandinavian Candesartan Acute Stroke Trial), which demonstrated that early aggressive blood pressure lowering did not improve functional outcomes and may potentially worsen neurological deficits in some patients.12,13

At blood pressure ≥ 220/120 mmHg, and there are no other comorbidities, a decrease in blood pressure is recommended by 15 percent within 48-72 hours after onset. If the patient's blood pressure is >140/90 mmHg, with good clinical condition and a history of hypertension, initial or repeated doses of antihypertensives are considered safe. In addition to general blood pressure, the 2019 AHA/ASA stated that hypovolemia and hypotension should be managed in patients with AIS to prevent impaired brain tissue perfusion.8 Maintaining adequate intravascular volume is crucial for cerebral perfusion, and aggressive volume resuscitation with isotonic saline is recommended when hypotension is present, with a target systolic blood pressure of at least 140 mmHg to ensure adequate cerebral perfusion pressure.

Blood Pressure Management In AIS With Reperfusion Therapy

AIS patients undergoing reperfusion therapy such as IV tissue plasminogen activator (IV-tPA) or endovascular therapy (EVT) require more stringent blood pressure control.12 The physiological basis for tighter blood pressure control in these patients relates to the increased risk of hemorrhagic transformation following reperfusion. Thrombolytic agents disrupt the fibrin matrix of the thrombus but can also affect the blood-brain barrier integrity, making elevated blood pressure a significant risk factor for symptomatic intracranial hemorrhage.

With IV-tPA, the risk of post-treatment bleeding increases, and blood pressure should be lowered to ≤185/110 mmHg. However, aggressive blood pressure reduction can lead to cerebral hypoperfusion and further ischemic brain tissue expansion. AHA/ASA guidelines, based on the National Institute of Neurological Disorder and Stroke rt-PA Stroke Study (NINDS tPA trial)8, recommend that patients undergoing IV-tPA with blood pressure >185/110 mmHg and no contraindications for intravenous alteplase therapy receive blood pressure reduction before the procedure. The recommended blood pressure target during and 24 hours after the procedure is <180/105 mmHg. Clinical evidence from the ENCHANTED trial (Enhanced Control of Hypertension and Thrombolysis Stroke Study) suggested that intensive blood pressure lowering (target systolic BP 130-140 mmHg) within 6 hours of thrombolysis did not improve functional outcomes compared to guideline-recommended targets, but did reduce the risk of intracerebral hemorrhage, supporting the current guideline recommendations.13 High blood pressure after IV-tPA therapy increases the risk of brain hemorrhage and worsens outcomes.8,13

In endovascular therapy, careful blood pressure management is essential due to the risks of both reperfusion injury and cerebral hemorrhagic transformation, as well as the potential for hypoperfusion if blood pressure is lowered excessively before vessel recanalization. Blood pressure monitoring is recommended before, during, and after EVT therapy. The 2019 AHA/ASA guidelines recommend optimal blood pressure before EVT (within 24 hours of onset) is <185/105 mmHg.8 If mechanical thrombectomy is performed before IV-tPA, then the recommended blood pressure is ≤185/110 mmHg before the procedure and ≤180/105 mmHg. The post-thrombectomy blood pressure management strategy should be individualized based on recanalization status. If reperfusion fails, the recommended systolic blood pressure is ≥150 mmHg to maintain collateral flow and optimize perfusion to the persistently ischemic tissue, and if reperfusion is successful,

the recommended systolic blood pressure is <140 mmHg to minimize the risk of hemorrhagic transformation in the reperfused territory. If hemorrhagic stroke occurs after EVT therapy, then the recommended blood pressure is <140 mmHg.14

Blood Pressure Medication

The antihypertensive modality commonly used in the management of hypertension in patients with AIS is the use of short-acting antihypertensives, to prevent long-term effects of the drug. Short-acting intravenous agents are preferred because they allow for rapid titration and quick reversal if blood pressure decreases excessively, providing greater control in the dynamic acute stroke setting where neurological status can change rapidly. In hypertensive emergencies, antihypertensive treatment is given in the form of beta-blockers or calcium channel blockers (CCBs).15

The following section provides a detailed comparison of commonly used antihypertensive agents in acute ischemic stroke, including their mechanisms of action, pharmacokinetics, and clinical considerations:

The dose of esmolol in hypertensive emergencies is 500-1000 µg/kg i.v. bolus in 1 minute or 50-250 µg/kg/minute continuous i.v. drip. Esmolol is an ultra-short-acting selective β1-adrenergic blocker with rapid onset (1-2 minutes) and short half-life (9 minutes), making it highly titratable, though it is less commonly used in stroke due to concerns about potential cerebral vasoconstriction.

The dose of labetalol is 0.25-0.5 mg/kg i.v. bolus in 2-4 minutes, or an infusion of 5-20 mg/hour. Labetalol, a combined α- and β-adrenergic blocker, is one of the most frequently used agents in acute stroke due to its predictable blood pressure reduction without significantly affecting cerebral blood flow. It has an onset of action of 5-10 minutes and duration of 3-6 hours, providing good controllability. In cases of AIS, the labetalol dose differs from that in hypertensive emergencies, namely 10-20 mg i.v. bolus or infusion in 1-2 minutes.

The dose of nicardipine is 5 mg/hour continuous i.v. drip, the dose can be increased by 2.5 mg/hour every 15 minutes to the maximum dose.16 Nicardipine, a dihydropyridine calcium channel blocker, is another first-line agent for acute stroke with onset of action within 5-15 minutes. It causes cerebral vasodilation and may improve cerebral blood flow, though this theoretical benefit has not been conclusively demonstrated in clinical trials. Its titratable infusion allows for precise blood pressure control. The nicardipine dose in AIS is similar to that in hypertensive emergencies, 5 mg/hour i.v. continuous drip, the dose can be increased by 2.5 mg/hour every 5-15 minutes to a maximum dose (15 mg/hour).

The hydralazine dose is 10-20 mg i.v., repeated every 4-6 hours, if necessary, until reaching the maximum dose (40 mg/day). Hydralazine is a direct arterial vasodilator with onset of 10-20 minutes, but its use is limited by unpredictable blood pressure response, reflex tachycardia, and prolonged duration of action (3-8 hours), making it less favorable in the acute stroke setting where precise control is needed.

The enalaprilat dose is 0.625-1.250 mg i.v. every 6 hours. Enalaprilat, the active form of enalapril, is an ACE inhibitor with onset of 15 minutes and peak effect at 1-4 hours. While generally safe, its relatively slow and variable response makes it less suitable for acute blood pressure management requiring rapid titration.

The sodium nitroprusside dose is 0.3-0.5 mcg/kg/minute i.v. infusion. Sodium nitroprusside

is a potent, rapidly acting (onset 30-60 seconds) arterial and venous vasodilator that allows for precise blood pressure control. However, its use in acute stroke is controversial and generally reserved for refractory hypertension, as it may cause cerebral vasodilation leading to increased intracranial pressure and potential "steal" phenomenon, where blood flow is diverted away from ischemic areas. Additionally, prolonged infusions carry the risk of cyanide toxicity.

The dose of glyceryl trinitrate is 5 mg/day transdermal. Glyceryl trinitrate (nitroglycerin) is primarily a venodilator with slower onset when given transdermally. While some studies have explored its use in acute stroke, it is not considered a first-line agent for acute blood pressure management due to its slow titratability and concerns about increasing intracranial pressure.

Table 1 summarizes the comparative characteristics of these antihypertensive agents

Agent	Onset of Action	Duration	Primary Mechanism	Advantages in AIS	Limitations in AIS
Labetalol	5-10 min	3-6 hours	α- and β-blockade	Predictable response, preserves CBF	Contraindicated in asthma, heart failure
Nicardipine	5-15 min	1-4 hours	Calcium channel blockade	Titratable, may improve CBF	Requires continuous infusion
Sodium nitroprusside	30-60 sec	1-2 min	Direct vasodilation	Rapid, precise control	Risk of ICP increase, cyanide toxicity
Hydralazine	10-20 min	3-8 hours	Direct arterial dilation	Bolus administration	Unpredictable response, prolonged effect
Enalaprilat	15 min	12-24 hours	ACE inhibition	Generally safe	Slow onset, variable response

Each of these six medications has advantages and disadvantages that must be considered in the context of individual patient characteristics, comorbidities, and available monitoring capabilities. Sodium nitroprusside has the fastest onset of action compared to other antihypertensives, followed by labetalol and nicardipine.10,17 In clinical practice, labetalol and nicardipine are most commonly preferred as first-line agents due to their favorable balance of efficacy, safety, and controllability in the acute stroke setting.

Discussion

Blood pressure management in acute ischemic stroke represents a critical therapeutic challenge that requires careful balancing of competing physiological priorities. The fundamental tension lies between maintaining adequate cerebral perfusion to salvage ischemic penumbra and preventing complications such as hemorrhagic transformation, cerebral edema, and systemic end-organ damage.

The pathophysiology underlying blood pressure elevation in AIS involves multiple mechanisms. Following ischemic injury, cerebral autoregulation—the brain's intrinsic ability to maintain constant blood flow despite changes in perfusion pressure—becomes impaired within the ischemic territory and penumbra. This impairment creates a state of pressure-passive perfusion, where cerebral blood flow becomes directly dependent on systemic blood pressure.

Simultaneously, the body initiates a physiological stress response involving activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis, further elevating blood pressure. This reactive hypertension can be viewed as a compensatory mechanism attempting to maintain perfusion to threatened brain tissue. Understanding these mechanisms helps explain the rationale for permissive hypertension in certain clinical contexts.

The differential approach to blood pressure management based on reperfusion therapy status reflects different risk-benefit profiles. In patients not receiving reperfusion therapy, the primary concern is maintaining adequate cerebral perfusion, particularly in the first 24-48 hours when the ischemic penumbra may still be salvageable through collateral circulation. The threshold of 220/120 mmHg for intervention balances the risk of hypertensive complications against the need for cerebral perfusion. Evidence from trials such as CATIS and SCAST demonstrated that early aggressive blood pressure lowering in this population did not improve outcomes and may have contributed to infarct expansion in some patients, validating the more conservative approach in current guidelines.

Conversely, patients receiving reperfusion therapy face a markedly increased risk of hemorrhagic transformation due to disruption of the blood-brain barrier and the effects of thrombolytic agents. The NINDS tPA trial established the blood pressure threshold of 185/110 mmHg pre-treatment and 180/105 mmHg during and after treatment based on the hemorrhage rates observed in that landmark study. Subsequent trials, including ENCHANTED, have explored whether even more intensive blood pressure control might be beneficial, but have not demonstrated clear superiority of intensive lowering while confirming the importance of avoiding excessive hypertension. The challenge in the endovascular therapy population is further complicated by the need to maintain perfusion during the procedure itself, particularly in cases where recanalization is delayed or incomplete.

The selection of antihypertensive agents in acute stroke requires consideration of several factors beyond mere blood pressure reduction. The pharmacokinetic profile—particularly onset of action, duration of effect, and reversibility—is crucial in the dynamic acute stroke setting where clinical status can change rapidly. Labetalol and nicardipine have emerged as preferred first-line agents due to their relatively rapid onset, titratable dosing, and evidence suggesting preservation of cerebral blood flow. The theoretical concern about sodium nitroprusside causing intracranial pressure elevation and "cerebral steal" has limited its use to refractory cases, though definitive clinical evidence of harm in stroke patients is limited. The relative paucity of head-to-head trials comparing different antihypertensive agents in the acute stroke setting represents an ongoing knowledge gap.

Blood pressure variability in the acute phase of stroke has emerged as an independent predictor of poor outcomes, possibly more important than mean blood pressure levels. High variability may reflect impaired baroreflex function and may cause fluctuations in cerebral perfusion, potentially contributing to infarct expansion or hemorrhagic transformation. This finding suggests that the manner in which blood pressure is controlled—favoring smooth, gradual reduction over abrupt changes—may be as important as the target itself. Current guidelines do not provide specific recommendations for managing blood pressure variability, representing an area for future research and guideline development.

The implementation of blood pressure guidelines in resource-limited settings presents unique challenges. Continuous blood pressure monitoring, availability of intravenous

antihypertensive agents, and intensive care unit capabilities vary significantly across different healthcare systems. In settings where nicardipine or labetalol may not be readily available or affordable, clinicians must rely on alternative agents while being cognizant of their limitations. The development of context-specific protocols that maintain fidelity to evidence-based principles while accommodating local resources is an important area for quality improvement initiatives.

Future research directions should address several persistent questions. First, whether blood pressure management strategies should be further individualized based on imaging characteristics, such as collateral circulation status, infarct core size, or penumbral volume. Second, the optimal blood pressure targets in specific patient subgroups, including those with chronic hypertension, small vessel disease, or posterior circulation strokes. Third, the role of newer monitoring technologies, such as transcranial Doppler or near-infrared spectroscopy, in guiding blood pressure management. Finally, implementation science research examining how to optimize adherence to blood pressure guidelines in diverse clinical settings.

Despite the substantial progress in understanding and managing blood pressure in AIS, significant uncertainties remain. The evidence base is strongest for patients receiving IV thrombolysis, moderate for those undergoing mechanical thrombectomy, and weakest for those receiving no reperfusion therapy. Most clinical trials have been conducted in well-resourced academic centers with access to specialized stroke care, potentially limiting generalizability. Nonetheless, current guidelines provide a reasonable evidence-based framework for blood pressure management that, when implemented appropriately, can optimize outcomes for most acute ischemic stroke patients.

Conclusion

Blood pressure management is crucial in treatment of acute-onset acute ischemic stroke (AIS), with differentiated targets based on reperfusion therapy status: more permissive targets (up to 220/120 mmHg) for patients not receiving reperfusion, and stricter control (≤185/110 mmHg) for those undergoing thrombolysis or mechanical thrombectomy. The preferred antihypertensive agents are short-acting, titratable intravenous medications like labetalol and nicardipine, which balance maintaining cerebral perfusion and reducing hemorrhagic risk. Clinical decisions should be individualized, considering stroke severity, comorbidities, and resource availability, while adhering to evidence-based guidelines to optimize neurological outcomes and reduce mortality. Future research should focus on refining blood pressure targets for specific patient subgroups, comparing the efficacy and safety of different antihypertensive agents in the acute setting, and developing practical protocols for blood pressure management in resource-limited environments to further improve stroke care worldwide.

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