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Update of the Korean Clinical Practice Guidelines for Stroke: Blood Pressure Management

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Hypertension is a major risk factor for stroke, and appropriate management of blood pressure (BP) is crucial for both prevention and treatment. Since the 2009 publication of the Clinical Practice Guideline (CPG) by the Korean Stroke Society (KSS), significant advances have been made in BP management for stroke patients, particularly in cases involving intracerebral hemorrhage and in the context of endovascular reperfusion therapy for ischemic stroke. In light of recent evidence, the CPG Committee of the KSS initiated an update of the guidelines for BP management in acute stroke. The updated guidelines outline comprehensive strategies for BP management for both primary and secondary stroke prevention, which includes a total of 19 recommendations. These guidelines aim to provide healthcare professionals with up-to-date, evidence-based recommendations to support the improvement of quality of care for stroke patients.

Keywords acute ischemic stroke; blood pressure; guidelines.

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INTRODUCTION

Since the initial publication of the Clinical Practice Guideline (CPG) by the Korean Stroke Society (KSS) in 2009, clinical research on blood pressure (BP) management patients with acute stroke has significantly advanced.[1] Over the years, new evidence has led to updates in hypertension treatment guidelines from the American Heart Association (AHA), the European Society of Cardiology, and the Korean Society of Hypertension.[2-6]

Meanwhile, endovascular reperfusion therapy (ERT)—considered the most significant advancement in stroke treatment—has become the gold standard for patients with large vessel occlusion.[7-9] However, BP control after ERT remains controversial. Recently, several

randomized controlled trials (RCTs) and meta-analyses have addressed this issue.[10-13] These developments emphasize the necessity to revise hypertension-related guidelines for stroke management in South Korea.

The focus of this update on hypertension management stems from its role as the most important modifiable risk factor for stroke, as well as the potential impact of acute-phase BP control on patient outcomes. Unlike other risk factors, BP can be managed promptly through lifestyle changes and pharmacologic interventions, allowing clinicians to improve both immediate and long-term stroke care.

To reflect the evolving body of evidence, the CPG Committee of the KSS undertook a revision of the Korean guideline for BP management in acute stroke patients. In addition to addressing BP management during the acute phase, the revised guideline also provides comprehensive recommendations for the primary and secondary prevention of stroke, based on current evidence and international best practices. The aim of this updated guideline is to equip healthcare providers with evidence-based, up-to-date recommendations to support clinical decision-making and ultimately enhance the quality of care for stroke patients.

METHODOLOGY

Level of Evidence and Grade of Recommendation

We assigned the Level of Evidence (LOE) and Grade of Recommendation (GOR) for each recommendation based on the criteria from the US Agency for Healthcare Policy and Research (now the Agency for Healthcare Research and Quality) (Table 1).[14] This grading system was used to align with recommendations in other sections of the Korean CPG for stroke.

Consensus achievement

The CPG Committee of the KSS plans to announce the revised guidelines by incorporating input from 33 domestic stroke management experts using the modified Delphi method (Supplementary Table 1 in the online-only Data Supplement). This method, often employed in medical research, involves a structured group interaction where panelists typically engage through questionnaires and receive feedback from facilitators to reach consensus.

The panel of experts was asked to rate each recommendation on a 9-point scale, where 9 indicated strong agreement and 1 indicated strong disagreement, using the Research and Development Corporation method.[15] Ratings of 7–9 were classified as agreement, 4–6 as uncertainty, and 1–3 as disagreement. A consensus was considered as achieved if 75% or more of the experts agreed on a recommendation. If the agreement rate was below 75%, additional Delphi rounds were conducted with revised recommendations based on the panel's feedback.

In the first round of the Delphi process, 13 questions were evaluated, of which 11 reached consensus. By the conclusion of the third round, all questions had achieved the predefined threshold of 75% agreement. Further details are provided in Supplementary Table 2 (in the online-only Data Supplement).

THE PREVIOUS GUIDELINES

The previous 2009 Korean CPG included recommendations for BP control in patients with acute stroke, as well as hyper-

Table 1. Level of evidence and grade of recommendation

Level of evidence	
Ia	Evidence obtained from meta-analysis of randomized controlled trials
Ib	Evidence obtained from at least one randomized controlled trial
IIa	Evidence obtained from at least one well-designed controlled study without randomization
IIb	Evidence obtained from at least one other type of well-designed quasi-experimental study
III	Evidence obtained from well-designed non-experimental descriptive studies, such as comparative studies, correlation studies and case studies
IV	Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities
Grade of recommendation	
A (LOE Ia, Ib)	Required - at least one randomized controlled trial as part of the body of literature of overall good quality and consistency addressing specific recommendation
B (LOE IIa, IIb, III)	Required - availability of well conducted clinical studies but no randomized clinical trials on the topic of recommendation
C (LOE IV)	Required - evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities. This grade indicates absence of directly applicable clinical studies of good quality
GPP	Recommended best practice based on the clinical experience of the guideline development group

GOR, grade of recommendation; GPP, Good Practice Points; LOE, level of evidence.

tension management for the primary and secondary prevention of stroke.[1] The guideline for BP management after intracerebral hemorrhage (ICH) was updated in 2014.[16]

Primary prevention of stroke

Well-documented and modifiable risk factors

Hypertension

1) Regular BP monitoring is recommended in adults, particularly in the elderly or those with other cerebrocardiovascular risk factors (GOR: Good Practice Points [GPP]).

2) Lifestyle modification is recommended for prevention and treatment of hypertension (weight loss if overweight, low-fat/low-salt diet, exercise, moderate drinking, and no smoking). If necessary, drug therapy should be initiated to lower BP (LOE: Ia, GOR: A).

3) The target BP is <140/90 mm Hg for primary prevention of stroke (LOE: Ia, GOR: A).

4) In patients with diabetes and/or renal disease, the target BP is <130/80 mm Hg (LOE: Ia, GOR: A).

5) Systolic hypertension in the elderly should be treated with the same principles and methods as other hypertensions (LOE: Ia, GOR: A).

6) For primary stroke prevention, an adequate BP control is the most important rather than choosing a specific class of antihypertensive agent. However, given no compelling indications, calcium channel blockers (CCBs) or renin-angiotensin system inhibitors are recommended over beta-blockers (LOE: Ia, GOR: A).

Acute stroke management

Acute treatment

General supportive care

Blood pressure

1) For systolic blood pressure (SBP) ≤220 mm Hg or diastolic blood pressure (DBP) ≤120 mm Hg in acute ischemic stroke, a deferral of aggressive BP lowering is recommended (LOE: IV, GOR: C).

2) If the thrombolytic therapy is under way, BP lowering agents can be used to lower SBP <185 mm Hg and DBP <110 mm Hg (LOE: IV, GOR: C).

3) Though a uniform BP lowering is not recommended in acute ischemic stroke, an adequate BP reduction is needed in the following conditions that increase the risk for hypertensive complications: hypertensive encephalopathy, aortic aneurysm with renal artery invasion (LOE: Ia, GOR: A), heart dysfunction, aortic dissection, acute myocardial infarction, acute renal failure, and intravenous heparin use (LOE: IV, GOR: C).

4) For hypotension occurring in acute stroke patients, causal analysis is recommended. Hypovolemia, if present, may be corrected with saline supplementation. Correction is also recommended for arrhythmia that reduces cardiac output (LOE: IV, GOR: C).

Treatment of ICH (update in 2014)

Medical treatment of ICH

BP management after ICH

1) The suggested BP targets in patients with acute ICH are described below (LOE: III, GOR: B). Drugs that can be used for BP control in spontaneous ICH are shown in Table 2.[16]

(1) if SBP >200 mm Hg or mean arterial pressure (MAP) >150 mm Hg, then consider aggressive BP reduction with continuous intravenous infusion of drugs, with frequent BP measuring every 5 minutes.

(2) if SBP >180 mm Hg or MAP >130 mm Hg and there is any possibility of an intracranial pressure (ICP) elevation, then consider ICP monitoring and reducing BP using intermittent or continuous intravenous infusion while maintaining cerebral perfusion pressure (MAP-ICP) of 50–70 mm Hg.

(3) if SBP >180 mm Hg or MAP >130 mm Hg and there is no evidence of an ICP elevation, then consider a modest reduction of BP using intermittent or continuous intravenous infusion (MAP of 110 mm Hg or BP of 160/90 mm Hg) and clinically reexamine the patient every 15 minutes.

2) In patients with acute ICH, when SBP is measured between 150 and 220 mm Hg, BP may be safely lowered to 140 mm Hg within 1 hour (LOE: Ib, GOR: A).

3) Although selection of the antihypertensives after stroke or transient ischemic attack (TIA) is still controversial because of insufficient evidence, combination therapy of angiotensin-converting enzyme (ACE) inhibitors with diuretics

Table 2. Drugs that can be used for blood pressure control in spontaneous intracerebral hemorrhage

Drug name	Bolus dose	Infusion rate
Labetalol	5–20 mg, every 15 minutes	2 mg/min (up to 300 mg/day)
Nicardipine	No indication	5–15 mg/h
Esmolol	250 ug/kg, loading dose	25–300 ug/kg per minute
Hydralazine	5–20 mg, every 30 minutes	1.5–5 ug/kg per minute
Nitroprusside	No indication	0.1–10 ug/kg per minute
Nitroglycerine	No indication	20–400 ug/min

may be considered (LOE: Ib, GOR: A).

Secondary prevention

Risk factor control

Hypertension

1) In patients with stroke or TIA, antihypertensive treatment beyond the hyperacute phase reduces recurrent stroke and other cardiovascular events (LOE: Ia, GOR: A). The benefit of antihypertensive treatment is independent of prior history of hypertension. Adequate BP control is therefore recommended in all patients with stroke (LOE: Ib, GOR: A).

2) Determination of the antihypertensives and target BP should be individualized after taking into account of characteristics of patient such as steno-occlusion in intra- and extracranial vessels, diabetes, and renal disease (LOE: IV, GOR: C).

3) Although selection of the antihypertensives after stroke or TIA is still controversial because of insufficient evidence, combination therapy of ACE inhibitors with diuretics may be considered (LOE: Ib, GOR: A).

4) For an adequate BP control, drug therapy should be accompanied by lifestyle modifications (LOE: IV, GOR: C).

THE UPDATED GUIDELINES

Acute ischemic stroke

General recommendation for patients who do not receive recanalization therapy

[Recommendations]

• In patients with acute ischemic stroke with BP <220/120 mm Hg who are not candidates for intravenous tissue plasminogen activator (IV-TPA) or ERT and have no comorbidities requiring BP lowering, reducing BP within 48−72 hours of stroke onset is not recommended due to uncertain benefit (LOE: Ib, GOR: A) (revised from the previous recommendation).

• In acute ischemic stroke patients with BP ≥220/120 mm Hg who are not candidates for IV-TPA or ERT and have no comorbidities requiring BP lowering, the benefit of BP lowering within 48 to 72 hours after stroke onset is uncertain. If BP lowering is required based on clinical judgment, BP lowering by approximately 15% during the first 24 hours can be considered (LOE: IV, GOR: C) (revised from the previous recommendation).

• In acute stroke patients with hypotension, the cause of hypotension should be identified and corrected (LOE: IV, GOR: C) (revised from the previous recommendation).

[Summary of evidence]

Several large RCTs—including the Continue or Stop post-Stroke Antihypertensives Collaborative Study (COSSACS), the Scandinavian Candesartan Acute Stroke Trial (SCAST), the China Antihypertensive Trial in Acute Ischemic Stroke (CATIS), and the Valsartan Efficacy on Modest BP Reduction in Acute Ischemic Stroke (VENTURE) trial—have investigated the effects of initiating or continuing antihypertensive therapy within 48−72 hours after acute ischemic stroke onset.[17-20] These studies, involving thousands of patients, showed no significant reduction in mortality, dependency, or major cardiovascular events with early BP lowering. For example, The COSSACS found no difference in 2-week mortality or dependency between continuation and discontinuation of antihypertensives.[17] The SCAST observed no cardiovascular benefit and a trend toward worse functional outcomes with candesartan.[18] The CATIS reported no mortality or dependency benefit from immediate treatment, and the VENTURE trial found no improvement in 90-day prognosis, with increased neurological deterioration in the treatment group.[19,20]

A meta-analysis of over 12,000 patients confirmed that while antihypertensive treatment effectively lowers BP during the acute phase of ischemic stroke, it does not improve short- or long-term outcomes.[21] Similarly, another meta-analysis of 22 studies (5,672 treatment and 5,416 control participants) found no significant differences in dependency or mortality rates between groups, despite effective BP reduction.[22] Based on these evidences, the recommendation for initiating BP lowering within 48−72 hours was upgraded to LOE Ib, GOR A.

Previous guidelines recommended BP lowering in conditions such as acute heart failure, acute coronary syndrome, aortic dissection, and hypertensive encephalopathy. However, these recommendations are based on general patient populations, with no systematic studies specifically evaluating the effects of BP lowering in acute ischemic stroke patients with these comorbidities. Furthermore, due to the lack of systematic evaluation of BP lowering in acute ischemic stroke patients with BP >220/120 mm Hg, all related evidence levels were downgraded to LOE IV and GOR C. The revised recommendation also highlights the uncertain benefit of immediate BP lowering in patients without comorbidities requiring BP reduction. Nonetheless, if antihypertensive treatment is initiated, a BP reduction of approximately 15% within the first 24 hours is recommended, based on the American College of Cardiology/AHA Task Force guidelines to improve clinical applicability.[2]

Regarding correction of hypotension in acute stroke patients, the LOE and GOR were maintained after the Delphi

consensus, with only minor wording adjustments made.

Patients who receive intravenous thrombolysis

[Recommendations]

• In patients who are treated with IV-TPA, in order to reduce the risk of ICH, it is reasonable to lower BP <185/110 mm Hg before treatment and to maintain BP <180/105 mm Hg during the first 24 hours (LOE: IV, GOR: C) (revised from the previous recommendation).

• In patients receiving IV-TPA, intensive BP lowering over 72 hours (target SBP 130−140 mm Hg) is not generally recommended, as it has not shown a clear benefit compared to less-intensive BP lowering (target SBP <180 mm Hg) (LOE: Ib, GOR: A). However, intensive BP lowering may reduce asymptomatic or symptomatic ICH, and therefore can be considered in selected patients (LOE: IV, GOR: C) (new recommendation).

[Summary of evidence]

Most guidelines recommend adjusting BP before and after IV-TPA treatment based on the National Institute of Neurological Disorders and Stroke (NINDS) trial and the European Cooperative Acute Stroke Study (ECASS) III trial protocol, which is recommended to lower BP to below 185/110 mm Hg before IV-TPA administration and maintain it below 180/105 mm Hg for the following 24 hours.[23,24]

Through the Safe Implementation of Thrombolysis in Stroke-International Stroke Thrombolysis Register (SITS-ISTR) analysis, it has been confirmed that higher SBP in patients undergoing intravenous thrombolysis treatment is strongly correlated with poor outcomes.[25] The best outcomes were observed in the group with SBP of 141−150 mm Hg, showing a U-shaped association. However, optimal BP may vary depending on the reperfusion status after IV-TPA, and recent observational studies have shown that lower BP in patients with reperfusion led to better outcomes and reduced the risk of symptomatic ICH.[26]

Meanwhile, the Enhanced Control of Hypertension and Thrombolysis Stroke Study (ENCHANTED) compared intensive BP lowering (target SBP 130−140 mm Hg) with guideline-based lowering (target SBP <180 mm Hg) for 72 hours after IV-TPA administration.[27] The primary outcome, measured by the modified Rankin Scale (mRS) score distribution, showed no significant difference between the groups (unadjusted odds ratio [OR] 1.01, 95% confidence interval [CI] 0.87−1.17). The intensive BP lowering group had a significantly lower rate of any intracranial hemorrhage (asymptomatic or symptomatic) (OR 0.75, 0.60−0.94), although rates of symptomatic intracranial hemorrhage were similar between groups. While neurological benefits of intensive BP lowering were unclear, the reduced risk of intracranial hemorrhage suggests that intensive BP lowering after IV-TPA may be considered for select patients at a low level of recommendation.

Patients who receive ERT

[Recommendations]

• In patients treated with ERT, to reduce the risk of ICH, it is reasonable to lower BP to <185/110 mm Hg before treatment (LOE: IV, GOR: C). For patients who achieved successful reperfusion (thrombolysis in cerebral infarction grade [TICI], 2b or 3) following the ERT, it is recommended to maintain SBP ≤180 mm Hg during the first 24 hours rather than aggressively lowering it below 140 mm Hg (LOE: Ia, GOR: A), as excessive BP reduction may be harmful (new recommendation).

[Summary of evidence]

Several pivotal clinical trials evaluating ERT for acute ischemic stroke have excluded patients with BP exceeding 185/110 mm Hg prior to the procedure to reduce the risk of ICH.[28-34] Consequently, no RCTs have specifically addressed optimal BP management before ERT, and current clinical guidelines are based primarily on the BP management protocols utilized in these trials, resulting in low LOE and GOR.

In contrast, several recent RCTs have investigated BP targets after ERT in patients with successful reperfusion. The Blood Pressure Targets in Acute Stroke to Reduce Hemorrhage After Endovascular Therapy (BP-TARGET) trial, compared an intensive BP-lowering strategy targeting SBP of 100−129 mm Hg within 24 hours after ERT versus a standard target of 130−185 mm Hg. The study found no significant difference between groups in the incidence of ICH or mortality, indicating no clinical advantage to aggressive BP lowering.[10] Similarly, the Enhanced Control of Hypertension and Thrombolysis Stroke Study-2/Mechanical Thrombectomy (ENCHANTED2/MT) trial randomized patients to very low SBP targets (<120 mm Hg for 72 hours) or standard targets (140−180 mm Hg) and observed that intensive BP lowering was associated with increased early neurological deterioration and worse functional outcomes at 90 days, without any reduction in ICH incidence. This suggests that excessive BP reduction may be harmful in this population.[11]

The Blood Pressure Endovascular Stroke Therapy II (BEST-II) trial demonstrated the lowering SBP to targets below 140 or 160 mm Hg after successful ERT was not associated with harm; however, it showed a low probability of clinical benefit compared to a target of ≤180 mm Hg, with estimated suc-

cess probabilities of 25% for <140 mm Hg and 14% for <160 mm Hg in future confirmatory trials.[12] The Optimal Blood Pressure Management After Mechanical Thrombectomy for Acute Ischemic Stroke (OPTIMAL-BP) trial compared patients with SBP <140 mm Hg to those with SBP between 140 and 180 mm Hg, finding that the lower BP target group had a reduced likelihood of achieving functional independence, suggesting a potential detrimental effect of overly aggressive BP reduction.[13]

These findings have been corroborated by meta-analyses of the aforementioned trials. A pooled analysis demonstrated that the rate of functional independence at 90 days was significantly lower in the intensive BP lowering group (45.1%) compared to the standard treatment group (55.9%), with no differences in mortality or ICH rates.[35] In another meta-analysis, the aggressive BP lowering group showed lower rates of functional independence at three months (OR 0.68, 95% CI 0.51−0.91) and walking without assistance (OR 0.65, 95% CI 0.53−0.81) compared to the standard treatment group. There were no differences in mortality or ICH between the groups.[36]

Taken together, these data strongly support maintaining SBP at or below 180 mm Hg for the first 24 hours following successful ERT, while advising against more aggressive BP lowering due to lack of added benefit and potential harm.
Currently, there is insufficient evidence to establish definitive guidelines for BP management in patients who have not achieved successful reperfusion (TICI: 0-2a). A retrospective analysis found that in patients with successful reperfusion, a SBP interval ≥160 mm Hg was associated with reduced functional independence at 3 months, whereas in those with unsuccessful reperfusion, no SBP interval was significantly associated with functional outcomes.[37] These findings suggest that BP management strategies should differ between patients with successful and unsuccessful ERT. Notably, elevated SBP levels and SBP ≥160 mm Hg was associated with an increased risk of symptomatic ICH, regardless of reperfusion status. In patients who fail to achieve reperfusion, excessive BP reduction may compromise cerebral perfusion and is not generally recommended. Instead, BP management should be individualized, considering the patient's overall clinical condition, the presence of cerebral edema, and the risk of hemorrhagic transformation.

Similarly, there is still insufficient evidence from RCTs to include BP management during the procedure in official guidelines. Several retrospective case-control studies have consistently shown that significant intraoperative drops in MAP during ERT for ischemic stroke are linked to worse functional outcomes. One study of 108 patients under general anesthesia found that a 40% decrease in MAP independently increased the risk of poor prognosis (mRS score >2).[38] Another study of 256 patients receiving conscious sedation reported that a MAP reduction greater than 10% was associated with poor outcomes, especially when MAP dropped below 100 mm Hg.[39] Similarly, a larger study involving 390 patients demonstrated that a greater decrease in MAP prior to reperfusion was associated with poorer functional outcomes at discharge and at 3 months.[40] However, a systematic review indicated that strict intraoperative control of SBP between 140 and 180 mm Hg could eliminate this negative association.[41]

General recommendation after emergent management

[Recommendation]

• In acute ischemic stroke, the continuation of antihypertensive medications during the acute phase is not recommended but may be considered safe. Initiation or resumption before discharge may be appropriate in neurologically stable patients (LOE: IIb, GOR: B) (new recommendation).

[Summary of evidence]

In patients with acute ischemic stroke within 48 hours of onset, routine continuation or initiation of antihypertensive therapy has not demonstrated a clear benefit in reducing short-term mortality or functional dependency.

The COSSACS found no significant difference in 2-week mortality or dependency between patients who continued pre-stroke antihypertensive medications and those who temporarily discontinued them. Importantly, continued use was not associated with an increased risk of adverse events.[17]

Similarly, the CATIS trial showed that initiating antihypertensive treatment during hospitalization in patients with SBP between 140 mm Hg to 220 mm Hg did not improve 2-week mortality or functional outcome at 3 months, nor did it reduce cardiovascular events. However, a non-significant trend toward reduced lower rate of recurrent stroke (OR, 0.65 [95% CI, 0.40 to 1.04]; p=0.07) was observed in the treatment group.[19]

Based on current evidences, the continuation or initiation of antihypertensive therapy within 48 hours of acute ischemic stroke onset is supported by moderate-quality data from randomized trials with methodological limitations. Given the lack of demonstrated benefit in short-term mortality or functional outcomes, and a neutral safety profile, this approach receives a Class IIb recommendation. Accordingly, in patients with acute ischemic stroke, routine continuation of antihypertensive medications during the acute phase is not recommended but may be considered safe. Initiation or resumption before discharge may be appropriate

in neurologically stable patients.

Acute hemorrhagic stroke

[Recommendation]

• In patients with acute ICH who have an elevated SBP >220 mm Hg, it is reasonable to reduce BP with intravenous antihypertensive agent(s) infusion and close BP monitoring (LOE: III, GOR: B) (revised from the previous recommendation).

• In patients with acute ICH presenting within 6 hours of symptom onset and with SBP between 150 and 220 mm Hg, lowering of SBP to a target of around 140 mm Hg is reasonable to reduce the risk of hematoma expansion without increasing serious adverse events (LOE: IIa, GOR: B). However, aggressive BP lowering beyond this target (i.e., SBP <140 mm Hg) is not routinely recommended, as it may be associated with potential harm (revised from the previous recommendation).

[Summary of evidence]

Hypertension is a critical risk factor for ICH and is commonly observed during the acute phase of the event. Elevated BP may worsen hematoma expansion and is associated with poor outcomes, underscoring the need for careful management. The Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial (INTERACT) pilot RCT enrolled 404 participants with acute spontaneous ICH diagnosed by CT within 6 hours of symptom onset to compare early intensive BP lowering (target SBP 140 mm Hg) with standard guideline-based management (target SBP 180 mm Hg). The study showed that early intensive BP lowering is safe and can reduce the risk of hematoma expansion.[42] Additionally, the Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH) trial demonstrated that using intravenous nicardipine to lower SBP to 110−140 mm Hg is safe and feasible.[43]

The INTERACT2 trial enrolled patients with acute ICH diagnosed within 6 hours of symptom onset and compared intensive BP lowering (target SBP <140 mm Hg) with guideline-recommended management (target SBP <180 mm Hg). The trial showed no statistically significant reduction in death or major disability (mRS score of 3−6) at 90 days between the two groups. However, there was a modest but favorable shift in functional outcomes. Mortality rates and the contribution of cerebral hemorrhage to death were similar between groups. Intensive BP lowering did not increase neurological deterioration within 24 hours or the incidence of serious adverse events.[44] Additionally, two small studies published in the same year, elevated SBP and greater BP variability following ICH were associated with hematoma growth and early neurological deterioration or poorer neurological outcomes.[45,46]

The ATACH-2 trial, published in 2016, compared two SBP targets in patients with acute ICH treated within 4.5 hours of symptom onset: 110−139 mm Hg (intensive treatment) versus 140−179 mm Hg (standard treatment). The trial was terminated early after enrolling 1,000 patients due to futility. The primary outcome—death or major disability (mRS score of 4−6) at 3 months—did not significantly differ between groups (38.7% vs. 37.7%). However, the incidence of renal-related adverse effects within 7 days was significantly higher in the intensive group (9.0% vs. 4.0%). These findings suggest that targeting an SBP around 140 mm Hg may be optimal, and that more aggressive reduction below 140 mm Hg offers no additional benefit and may increase the risk of harm.[47]

Reflecting the results of several trials that it is reasonable to lower the SBP to 140 mm Hg in patients with acute ICH within 6 hours of onset with BP ranging from 150 to 220 mm Hg.[43-48] However, excessive BP lowering targeting a SBP <140 mm Hg is not recommended, as it offers no additional benefits and may potentially increase the risk of renal dysfunction; thus, it is categorized as "LOE IIa and GOR B." In addition, recent studies have shown a clear trend toward using SBP as the primary target, and to enhance practicality in clinical settings, the guideline eliminates the criteria based on MAP, unifying the recommendations around SBP.

Secondary prevention of stroke

[Recommendations]

• In patients with stroke or TIA who have previously or newly diagnosed hypertension of an established BP ≥140/90 mm Hg, antihypertensive treatment should be restarted or initiated several days after the stroke or TIA to reduce the risk of recurrent stroke and other cardiovascular events (LOE: Ia, GOR: A) (revised from the previous recommendation).

• For specific antihypertensive classes, thiazide diuretics or the combination of thiazide diuretics plus ACE inhibitors are recommended (LOE: Ia, GOR: A). Angiotensin receptor blockers (ARBs) as an alternative to ACE inhibitors can be recommended (LOE: IIa, GOR: B).

• Selection of specific antihypertensive agents and the target BP level should be individualized after considering comorbidities, stroke subtypes, and steno-occlusion of intra- and extracranial cerebral arteries (LOE: IV, GOR: C) (revised from the previous recommendation).

• In patients with a lacunar infarction due to small-vessel occlusion, targeting SBP of less than 130 mm Hg may be considered to reduce the risk of ICH (LOE: IIb, GOR: B) (new recommendation).

• Lifestyle modifications should be combined with drug

therapy to achieve optimal BP control (LOE: Ia, GOR: A) (revised from the previous recommendation).

[Summary of evidence]

The first two recommendations are basically not changed from the previous recommendation, which was based on result of the Perindopril Protection Against Recurrent Stroke Study (PROGRESS) and the Post-stroke Antihypertensive Treatment Study (PATS). The PROGRESS, involving 6,105 patients with stroke or TIA, found that combination therapy with the ACE inhibitor perindopril and the diuretic indapamide reduced stroke recurrence by 43% and major vascular events by 40%, while monotherapy with perindopril alone showed no significant benefit.[48] The PATS with 5,665 stroke patients, demonstrated that indapamide significantly decreased the incidence of stroke and major cardiovascular events over an average of two years.[49]

In addition, a 2017 meta-analysis of 14 studies with over 42,000 patients showed that antihypertensive treatment significantly reduces recurrent stroke, disabling or fatal stroke, and cardiovascular death, with the lowest risks observed when SBP was maintained below 130 mm Hg, suggesting this as an optimal target for secondary stroke prevention.[50] As the result of meta-analysis are added since 2017, the LOE is upgraded to Ia from Ib.

Based on the large RCTs like PROGRESS and PATS trial, thiazide diuretics or the combination of thiazide diuretics plus ACE inhibitors are primarily recommended for BP control in patients with a history of stroke. However, ARBs may be considered as an alternative to ACE inhibitors based on supporting evidence.

In the PRoFESS (Prevention Regimen for Effectively Avoiding Second Strokes) trial, telmisartan showed a modest, though not statistically significant, reduction in recurrent stroke, with a post-hoc analysis suggesting greater benefit after 6 months.[51] Similarly, the MOSES (Morbidity and Mortality After Stroke, Eprosartan Compared with Nitrendipine for Secondary Prevention) study demonstrated that eprosartan was more effective than nitrendipine in reducing vascular events in patients with prior stroke.[52] A meta-analysis further supports the use of ARBs, showing a 7% relative risk reduction in recurrent stroke and a 9% reduction in overall vascular risk.[53] Recently, the FABULOUS (Fimasartan-Based Blood Pressure Control after Acute Cerebral Ischemia Study) demonstrated that in patients with recent ischemic stroke or TIA, fimasartan-based antihypertensive achieved target BP in about two-thirds of patients within 24 weeks and was generally well tolerated, with about 70% of patients reaching target BP within 24 weeks and very few adverse events reported.[54] Therefore, ARBs can be recommended as a reasonable alternative for BP management in stroke patients.

Several meta-analyses have shown that CCBs exhibit similar or even superior stroke prevention effects compared to other classes of antihypertensive medications in primary prevention study.[55,56] However, there is a lack of evidence supporting the effectiveness of CCBs in secondary stroke prevention.

BP management after ischemic stroke should be individ-

Table 3. The key recommendations from the 2018 Korean Society of Hypertension Guidelines for the management of hypertension

Recommendations	Class	Level
1. Lifestyle modification is recommended in the population with elevated BP and prehypertension* and all hypertensive patients.	I	A
2. In patients with grade 1 hypertension† and at moderate-to-high risk, prompt initiation of drug treatment is recommended along with lifestyle interventions.	I	A
3. Prompt initiation of BP-lowering drug treatment is recommended in patients with high risk‡ or grade 2 hypertension§, simultaneous with the initiation of lifestyle for achieving target goal BP.	I	A
4. For hypertensive patients at low to moderate risk, target BP of 140/90 mm Hg is recommended.	I	A
5. For elderly hypertensive patients, a target SBP <140 mm Hg can be considered.	IIa	B
6. It is recommended that SBP be lowered to below 140 mm Hg in hypertensive patients with diabetes.	I	A
7. It is recommended that DBP be lowered to below 85 mm Hg in hypertensive patients with diabetes.	IIa	C
8. For CKD patients with hypertension, target BP of 140/90 mm Hg is recommended.	I	A
9. In patients with BP higher than 160/100 mm Hg or more than 20/10 mm Hg above the target BP, two drugs can be prescribed in combination to maximize the antihypertensive effect and to achieve rapid BP control.	IIa	C
10. Thiazide or thiazide-like diuretics can be used as first-line drugs with a preference for chlorthalidone or indapamide.	IIa	B

*Prehypertension is defined as a SBP of 130–139 mm Hg or a DBP of 80–89 mm Hg; †Patients aged ≥50 years with CVD, PAD, aortic disease, heart failure, or LVH; ‡Grade 1 hypertension is defined as a SBP of 140–159 mm Hg or a DBP of 90–99 mm Hg; §Grade 2 hypertension is defined as a BP ≥160/100 mm Hg.

BP, blood pressure; CKD, chronic kidney disease; CVD, cardiovascular disease; DBP, diastolic blood pressure; LVH, left ventricular hypertrophy; PAD, peripheral artery disease; SBP, systolic blood pressure.

Table 4. Summary of current recommendations

Recommendations	Comment
Primary prevention of stroke	
Well-documented and modifiable risk factor	
Hypertension	
1. Hypertension is the most important and well-documented modifiable risk factor for stroke; therefore, treatment of hypertension is strongly recommended for the prevention of both ischemic and hemorrhagic stroke (LOE: Ia, GOR: A).	Revised from the previous recommendation
2. In adults with hypertension, other modifiable cardiovascular risk factors should be screened and managed (GOR: GPP).	Revised from the previous recommendation
3. In all patients with hypertension, lifestyle modifications should be recommended (LOE: Ia, GOR: A).	Revised from the previous recommendation
4. The optimal target BP level for primary stroke prevention remains unclear. To reduce the risk of stroke and overall cardiovascular diseases, we recommend following the 2018 Korean Society of Hypertension Guidelines regarding the initiation of antihypertensive agents and the target BP level (GOR: GPP).	New recommendation
5. Selection of antihypertensive agents should be individualized according to the patient's comorbidities and characteristics. For adults without compelling indications, CCBs, ACE inhibitors/ARBs, or diuretics or their combinations are preferred over beta-blockers for the primary stroke prevention (LOE: Ia, GOR: A).	Revised from the previous recommendation
Acute stroke management	
Acute treatment	
General supportive care	
Blood pressure	
1. In patients with acute ischemic stroke with BP <220/120 mm Hg who are not candidates for IV-TPA or ERT and have no comorbidities requiring BP lowering, reducing BP within 48–72 hours of stroke onset is not recommended due to uncertain benefit (LOE: Ib, GOR: A).	Revised from the previous recommendation
2. In acute ischemic stroke patients with BP ≥220/120 mm Hg who are not candidates for IV-TPA or ERT and have no comorbidities requiring BP lowering, the benefit of BP lowering within 48 to 72 hours after stroke onset is uncertain. If BP lowering is required based on clinical judgment, BP lowering by approximately 15% during the first 24 hours can be considered (LOE: IV, GOR: C).	Revised from the previous recommendation
3. In patients who are treated with IV-TPA, in order to reduce the risk of ICH, it is reasonable to lower BP <185/110 mm Hg before treatment and to maintain BP <180/105 mm Hg during the first 24 hours (LOE: IV, GOR: C).	Revised from the previous recommendation
4. In patients receiving IV-TPA, intensive BP lowering over 72 hours (target SBP 130–140 mm Hg) is not generally recommended, as it has not shown a clear benefit compared to less-intensive BP lowering (target SBP <180 mm Hg) (LOE: Ib, GOR: A). However, intensive BP lowering may reduce asymptomatic or symptomatic ICH, and therefore can be considered in selected patients (LOE: IV, GOR: C).	New recommendation

Table 4. Summary of current recommendations (continued)

Recommendations	Comment
5. In patients who are treated with ERT, in order to reduce the risk of ICH, it is reasonable to lower BP <185/110 mm Hg before treatment (LOE: IV, GOR: C). For patients who achieved successful reperfusion (thrombolysis in cerebral infarction grade 2b or 3) following the ERT, it is recommended to maintain systolic BP ≤180 mm Hg during the first 24 hours rather than aggressively lowering it below 140 mm Hg (LOE: Ia, GOR: A), as excessive BP reduction may be harmful.	New recommendation
6. In acute ischemic stroke, the continuation of antihypertensive medications during the acute phase is not recommended but may be considered safe. Initiation or resumption before discharge may be appropriate in neurologically stable patients (LOE: IIb, GOR: B).	New recommendation
7. In acute stroke patients with hypotension, the cause of hypotension should be identified and corrected (LOE: IV, GOR: C).	Revised from the previous recommendation
Acute stroke management	
Treatment of intracerebral hemorrhage (update in 2014)	
Medical treatment of intracerebral hemorrhage	
Blood pressure management after intracerebral hemorrhage	
1. In patients with acute ICH who have an elevated SBP >220 mm Hg, it is reasonable to reduce BP with intravenous anti-hypertensive agent(s) infusion and close BP monitoring (LOE: III, GOR: B).	Revised from the previous recommendation
2. In patients with acute ICH presenting within 6 hours of the onset who have an SBP level between 150 mm Hg and 220 mm Hg, lowering of SBP to a target of around 140 mm Hg is reasonable to reduce the risk of hematoma expansion without increasing serious adverse events (LOE: IIa, GOR: B). However, aggressive BP lowering beyond this target (i.e., SBP <140 mm Hg) is not routinely recommended, as it may be associated with potential harm.	Revised from the previous recommendation
Secondary prevention	
Risk factor control	
Hypertension	
1. In patients with stroke or TIA who have previously or newly diagnosed hypertension of an established BP ≥140/90 mm Hg, antihypertensive treatment should be restarted or initiated several days after the stroke or TIA to reduce the risk of recurrent stroke and other cardiovascular events (LOE: Ia, GOR: A).	Revised from the previous recommendation
2. For specific antihypertensive classes, thiazide diuretics or the combination of thiazide diuretics plus ACE inhibitors are recommended (LOE: Ia, GOR: A). ARBs as an alternative to ACE inhibitors can be recommended (LOE: IIa, GOR: B).	Revised from the previous recommendation
3. Selection of specific antihypertensive agents and the target BP level should be individualized after considering comorbidities, stroke subtypes, and steno-occlusion of intra- and extracranial cerebral arteries (LOE: IV, GOR: C).	Revised from the previous recommendation
4. In patients with a lacunar infarction due to small-vessel occlusion, targeting SBP of less than 130 mm Hg may be considered to reduce the risk of ICH (LOE: IIb, GOR: B).	New recommendation
5. Lifestyle modifications should be combined with drug therapy to achieve optimal BP control (LOE: Ia, GOR: A).	Revised from the previous recommendation

ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker; ERT, endovascular reperfusion therapy; GOR, grade of recommendation; GPP, Good Practice Points; IV-TPA, intravenous tissue plasminogen activator; LOE, level of evidence; SBP, systolic blood pressure; TIA, transient ischemic attack.

ualized based on stroke subtype, comorbidities, and the presence of intra- or extracranial arterial stenosis.[57] In large artery atherosclerosis, elevated BP (especially SBP ≥160 mm Hg and DBP ≥90 mm Hg) is associated with a higher risk of recurrent stroke, as shown in the WASID trial.[58] While overly aggressive BP lowering is not always necessary, maintaining excessively high BP may worsen outcomes. In patients with carotid artery occlusion (as in the COSS trial), achieving BP ≤130/85 mm Hg was associated with reduced risk of ipsilateral stroke.[59] Conversely, in the Vertebrobasilar Flow Evaluation and Risk of Transient Ischemic Attack and Stroke (VERiTAS) trial, intensive BP lowering (<140/90 mm Hg) increased stroke risk, highlighting the need to balance perfusion and prevention of recurrence based on vascular territory and collateral flow.[60]

In contrast, for small vessel occlusion (lacunar infarctions), where hypertension-related lipohyalinosis is the primary pathology, stricter BP control appears more favorable. The Secondary Prevention of Small Subcortical Strokes (SPS3) study trial found that targeting SBP <130 mm Hg, while not significantly reducing stroke recurrence compared to a higher target (130−149 mm Hg), did significantly lower the risk of ICH. These findings suggest that a tailored BP target—rather than a uniform threshold—should be adopted according to stroke mechanism and vascular status to optimize outcomes and minimize complications.[61]

Primary prevention of stroke

[Recommendations]

- Hypertension is the most important and well-documented modifiable risk factor for stroke; therefore, treatment of hypertension is strongly recommended for the prevention of both ischemic and hemorrhagic stroke (LOE: Ia, GOR: A) (revised from the previous recommendation).
- In adults with hypertension, other modifiable cardiovascular risk factors should be screened and managed (GOR: GPP) (new recommendation).
- In all patients with hypertension, lifestyle modifications should be recommended (LOE: Ia, GOR: A) (revised from the previous recommendation).
- The optimal target BP level for primary stroke prevention remains unclear. To reduce the risk of stroke and overall cardiovascular diseases, we recommend following the 2018 Korean Society of Hypertension Guidelines regarding the initiation of antihypertensive agents and the target BP level (GOR: GPP) (revised from the previous recommendation).
- Selection of antihypertensive agents should be individualized according to the patient's comorbidities and characteristics. For adults without compelling indications, CCBs, ACE inhibitors/ARBs, or diuretics or their combinations are preferred over beta-blockers for the primary stroke prevention (LOE: Ia, GOR: A) (revised from the previous recommendation).

[Summary of evidence]

Hypertension is the most prevalent modifiable risk factor for stroke and has a high population attributable risk.[62] Lowering BP alone can significantly reduce major cardiovascular disease events, coronary heart disease, stroke, heart failure, and all-cause mortality.[56,63,64] Additionally, BP management goals should not be determined solely by BP levels but should also consider accompanying risk factors and vascular diseases.[65-68] In terms of primary stroke prevention, it is considered more appropriate to provide recommendations aimed at reducing overall cardiovascular disease risk rather than specialized guidelines solely for stroke prevention. This context may also explain why the American Stroke Association has not made any revisions to its primary stroke prevention guidelines since 2014.[69]

Therefore, this guideline recommends following the 2018 Korean Society of Hypertension Guidelines for initiating anti-hypertensive therapy and determining target BP levels.[5] This approach aims to reduce the risk of both stroke and overall cardiovascular disease. To aid clinical decision-making, Table 3 summarizes the key recommendations from the 2018 guidelines.

IMPLICATIONS

There is an increasing emphasis on effective BP management as a critical factor in stroke treatment and prevention. Hypertension is a significant risk factor for stroke, and its control is essential to reduce both the incidence and mortality associated with cardiovascular events, including stroke. The revised guidelines provide robust evidence supporting BP management for acute stroke, secondary prevention, and primary prevention. All the updated recommendations are summarized in Table 4, while changes between the previous and revised guidelines are detailed in Supplementary Table 3 (in the online-only Data Supplement). Adhering to these updated guidelines serves as a valuable resource for clinicians, ensuring that patients receive evidence-based care tailored to their specific needs.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.3988/jcn.2025.0177.

Availability of Data and Material

All data generated or analyzed during the study are included in this published article (and its supplementary information files).

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Conflicts of Interest

Sang-Bae Ko and Keun-Hwa Jung, contributing editors of the *Journal of Clinical Neurology*, were not involved in the editorial evaluation or decision to publish this article. All remaining authors have declared no conflicts of interest.

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