

Review Article

Early treatment of hypertension in acute ischemic and intracerebral hemorrhagic stroke: Progress achieved, challenges, and perspectives

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Manuscript received August 5, 2013 and accepted September 27, 2013

Abstract

Hypertension is the leading risk factor for ischemic and intracerebral hemorrhagic subtypes of stroke. Additionally, high blood pressure (BP) in the acute cerebrovascular event is associated with poor outcome, and a high percentage of stroke survivors have inadequate control of hypertension. The present is a systematic review of prospective, randomized, and controlled trials carried out on safety and efficacy of antihypertensive treatment of both subtypes of acute stroke. Six trials involving 7512 patients were included, which revealed controversies on the speed and the goals of treatment. These controversies could be due at least in part, from the fact that some studies analyzed the results of antihypertensive treatment in ischemic and intracerebral hemorrhagic subtypes of acute stroke together, and from a different prevalence of past-stroke in the randomized groups. Further research is necessary to establish whether standard antihypertensive treatment provides greater benefit than simple observation in patients with ischemic acute stroke and Stage 2 hypertension of JNC 7, albeit they were not candidates for acute reperfusion. In that case, the target reduction in BP could be 10% to 15% within 24 hours. The recently published INTERACT 2 has provided evidence that patients with hemorrhagic stroke may receive intensive antihypertensive treatment safely with the goal of reducing systolic BP to levels no lower than 130 mm Hg. It is important to take into account that marked BP lowering in acute stroke increases the risk of poor outcome by worsening cerebral ischemia from deterioration of cerebral blood flow autoregulation. *J Am Soc Hypertens* 2014;8(3):192–202. © 2014 American Society of Hypertension. All rights reserved.

Keywords: Acute stroke; blood pressure elevation.

Introduction

Stroke is the third-ranked cause of death in the world and a major cause of permanent disability. The annual incidence of stroke around the world is 15 million, with a mortality of 5 million, and permanent sequelae, disability, and dependency on others of 5 million patients.¹ In the US, stroke is the fourth cause of death after heart disease, cancer, and chronic lower respiratory disease.^{2,3} Hypertension is the leading risk factor for stroke,⁴ increasing its incidence seven-fold.⁵ The American Heart Association/the American Stroke Association projections reports that by 2030, the

prevalence of hypertension will increase 7.2% from 2013 estimates.⁶ It is predicted that the stroke rate will rise significantly in the coming decades.⁷ Most studies have found that high blood pressure (BP) in the acute phase of stroke is associated with poor outcome.^{8–12} The first International Stroke Trial found a “U-shaped” relationship between systolic blood pressure (SBP; measured, on average, 24 hours after stroke) and outcome in patients with ischemic stroke where both high and low SBP were independently associated with poor outcome.^{11,12} In this trial, early death increased by 17.9% for every 10 mm Hg below 150 mm Hg of SBP ($P < .0001$), and by 3.8% for every 10 mm Hg above 150 mm Hg ($P = .016$). Also, the recurrence of stroke within 2 weeks increased by 4.2% for every 10 mm Hg increase in SBP ($P = .023$). Early recurrence of stroke was associated with unfavorable outcome, thus linking BP at admission with increased rate of death or dependency at 6 months. Mortality possibly caused by brain edema was independently associated with high SBP ($P = .004$). On the other

Conflict of interest: none.

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hand, low SBP was associated with severe stroke (total anterior circulation syndrome). A meta-analysis of 37 studies, that included a total of 9008 patients with ischemic or intracerebral hemorrhagic acute stroke, reported a U- and J-shaped relationships between on-treatment SBP difference and early death (within 1 month), mortality at the end of 90-day follow-up, and combined death or dependency at the end of follow-up.¹³ Nevertheless, some observational studies found a more linear relationship between increased in-hospital BP during acute ischemic stroke with worse clinical outcomes.^{14,15} The Virtual Stroke International Stroke Trial Archive (VISTA) Collaboration informed that large variability and high levels of SBP in the hyperacute stages of ischemic stroke were associated with high incidence of severe neurological events and major neurological disability outcome.¹⁶

More than three-quarters of patients with acute stroke have SBP higher than 140 mm Hg on admission to the hospital.^{17–21} In two-thirds of patients BP declines spontaneously over the first week after stroke onset and returns to pre-stroke levels. Table 1 shows the major guidelines of the American Heart Association/the American Stroke Association, the European Stroke Organization (ESO), and the European Stroke Initiative (EUSI) for the early management of BP in both subtypes of stroke.^{22–27} These guidelines recommend a target goal of BP < 140/90 mm Hg after

the acute period of stroke, or <130/80 mm Hg if there are comorbidities, such as diabetes mellitus or chronic kidney disease. Table 2 shows the dose and bioavailability of agents that may be used intravenously in acute stroke.

Disruption of Cerebral Autoregulation in Acute Stroke

The autoregulation of cerebral blood flow (CBF) is the mainstay adaptive mechanism to ensure that the arrival rate of blood flow and nutrients remains within the range (50–160 mm Hg) of fluctuations in arterial BP.²⁸ Cerebral arterioles adjust their resistance according to systemic BP levels, and autonomic nerve activity protects the homeostasis of the neurovascular unit. Vasoactive peptides and nitric oxide are essential mediators in CBF changes in particular regions of the brain. Chronic hypertension induces adaptive changes, including remodeling of cerebral arteries that lead to a reduction in their lumen.²⁹ Patients with chronic hypertension have an increase in the minimum values of BP of the cerebral autoregulation curve compared with matched normotensive subjects. Dynamic cerebral autoregulation is the physiologic process that maintains CBF relatively constant in the face of beat-to-beat BP changes. Static cerebral autoregulation refers to CBF adjustments in response to more prolonged BP changes and is a measure of the overall

Table 1

Management of hypertension in ischemic and intracerebral hemorrhagic acute stroke subtypes (adapted from^{22–24,25–27})

Acute Ischemic Stroke	Acute Intracerebral Hemorrhagic Stroke
In patients eligible for acute reperfusion, if their BP was not >185/110 mm Hg, administer: Labetalol IV bolus or nicardipine IV infusion, or other agents (hydralazine, enalaprilat, esmolol, nitroglycerin). If SBP >180–230 mm Hg or DBP >105–120 mm Hg not controlled with labetalol or nicardipine, or DBP >140 mm Hg, consider the administration of sodium nitroprusside. When target BP is reached, adjust the dose to keep desired levels of BP. During and after reperfusion therapy, BP must be maintained at ≤180/105 mm Hg. From the beginning of rtPA, monitor BP every 15 minutes for 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours. In patients who are not candidates for acute reperfusion therapy, when SBP <185 mm Hg or DBP <120 mm Hg, temporarily withdraw or reduce previous antihypertensive medications during the first 24 hours of acute ischemic stroke. Keep the patient under observation unless there is aortic dissection, acute myocardial infarction, pulmonary edema, or hypertensive encephalopathy. If SBP >220 or DBP in the range of 121–140 mm Hg, administer labetalol IV or nicardipine as IV infusion, aiming for a 10% to 15% reduction of BP. If DBP >140 mm Hg, give sodium nitroprusside as IV infusion, titrating the dose for a 10% to 15% lowering of BP (ie, searching for a lower limit of 185/120 mm Hg).	When SBP is 150–220 mm Hg, acute lowering to 140 mm Hg with antihypertensive agents administered IV is probably safe. If SBP is >200 mm Hg, or MAP >150 mm Hg, administer antihypertensive agents via IV with frequent BP monitoring. If SBP is >180 mm Hg or MAP >130 mm Hg in the absence of elevated intracranial pressure, BP target is 160/90 mm Hg, carrying out frequent patient reexaminations. Nitroglycerin and sodium nitroprusside must be used with caution in intracerebral hemorrhage because they increase intracranial pressure.

BP, blood pressure; DBP, diastolic blood pressure; IV, intravenous; MAP, mean arterial pressure; rtPA, recombinant tissue type plasminogen activator; SBP, systolic blood pressure.

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