Blood Pressure in Acute Stroke
Still No Answer for Management

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What Is the Mechanism of Elevated BP in Patients With Acute Stroke?

BP is increased in ≥3 of 4 patients with acute stroke and mostly decreases spontaneously over the next few days. However, the mechanism of the transient rise in BP is unknown and the BP increase after major stroke is often claimed to be a stroke-specific response (ie, post-stroke hypertension). We have shown in a population-based study that acute post-stroke BP in patients with ICH was markedly raised when compared with premorbid levels and fell rapidly thereafter. However, in patients with major ischemic strokes, acute post-event SBP was much closer to premorbid values to which these patients were presumably accustomed, with no rise in SBP before the event and only a small post-event increment. These findings suggest that the acute raise in BP mainly reflects premorbid hypertension rather than a stroke-specific response. Whether the additional small post-event increment in SBP is a direct consequence of the stroke that serves to enhance perfusion of the penumbra over collaterals is a matter of debate. Additional factors such as headache, urine retention, infection, or the psychological stress of admission to hospital could also play a role.

Should Elevated BP in Acute Stroke Be Lowered?

Several trials have assessed the question, whether BP should be lowered in patients with acute stroke, given the worse outcome in patients with high BP levels. Although there is good evidence that early BP lowering is safe and might be beneficial in patients with ICH, BP-lowering trials in patients with ischemic stroke were either neutral or negative.7–12 There are several explanations why BP lowering in patients with ischemic stroke has not been beneficial: the lack of benefit and potential harm may be related to reducing BP to a level to which the patient is unaccustomed. In accustomed hypertension, the cerebral perfusion curve is shifted to the right, such that rapid BP lowering could compromise blood flow at a time when perfusion is already compromised. Furthermore, in ischemic stroke with persistent vessel occlusion lowering of BP is likely to decrease collateral flow. Because BP autoregulation is disturbed, BP lowering would reduce penumbral perfusion, which could accelerate loss of penumbral tissue and increase the core of the infarct rapidly. When the penumbra disappears, either because it is salvaged after reperfusion or because it is gradually lost into the growing infarct core while occlusion persists, elevated BP is no longer needed. This hypothesis is supported by the observation of declining BP after stroke. The pace of BP decline is faster in patients after successful recanalization of large-vessel occlusions when compared with patients with persistently blocked arteries.14
Should BP Be Lowered Before IVT?
In patients with stroke undergoing IVT, it is common practice to avoid SBP >185 mm Hg and diastolic BP >110 mm Hg. BP thresholds for IVT candidates were established during the NINDS pilot study (National Institute of Neurological Disorders and Stroke) and are based on thrombolysis databases showing baseline hypertension as a risk factor for parenchymal hemorrhage. Furthermore, there is an association of elevated BP levels with the reduced likelihood of recanalization in patients with stroke treated with IVT. However, data on BP lowering before IVT are conflicting. Two studies suggest that hypertensive patients receiving IVT and antihypertensives have a worse outcome than patients without antihypertensives. On the contrary, observational data suggest that BP lowering before IVT, even using aggressive measures, may not be associated with a poor outcome. Furthermore, in the International Stroke Trial 3, the association between BP and outcome or occurrence of ICH was not affected by IVT. Overall, the evidence in current guidelines that advocate effective BP control before and during recombinant tissue-type plasminogen activator infusion is weak. Further trials are required to address this question more reliably.

What Is the Best BP Management Before, During, and After EVT?
Even though Mulder et al have shown that the effectiveness of EVT is similar in the entire range of baseline BP, this study does not answer the question whether elevated BP before, during, or after EVT should be lowered. Goyal et al have shown that higher admission SBP is an independent predictor of increased final infarct volume and a lower likelihood of favorable functional outcome in patients with large-vessel occlusion treated with EVT. However, given the overall poor evidence of BP management in patients with ischemic stroke and given the pathophysiological considerations mentioned above, active BP lowering in acute stroke patients with proximal vessel occlusion before recanalization might be harmful. We are therefore rather conservative and treat in the acute stroke phase only extremely high and extremely low BP. The average patient deserves careful BP observation and treatment only of persisting hypertension after the acute phase of stroke.

Although there is increasing evidence that IVT and EVT are beneficial in patients with stroke irrespective of admission BP, further studies should address the optimal BP management before, during, and after thrombolysis and thrombectomy. Such trials should focus on selected patients with low or extremely high BP and by no means lump together all patients with acute stroke.

Disclosures
None.

References


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