Treating Hypertension in Acute Stroke
A Better Arrow for the Quiver

J. David Spence

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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here has been a longstanding controversy about whether high blood pressure should be treated in the setting of acute stroke.1,2 Normally, cerebral blood flow is maintained through a wide range of systemic mean arterial blood pressure, from ≈50 to 150 mm Hg.3,4 In the setting of cerebral ischemia (and probably also in the zone of injury around intracerebral hemorrhages), the ischemic zone partially loses autoregulation, so cerebral blood flow in that region becomes dependent on perfusion pressure.5 Many experts, therefore, recommended that blood pressure elevation, which is common in the setting of acute stroke, not be treated for fear of exacerbating stroke by reducing perfusion pressure and thereby reducing flow in the compromised but viable ischemic penumbra.

Because swelling in the region of ischemia raises tissue pressure, the cerebral perfusion pressure falls below systemic blood pressure, and it was thought that higher pressures might be beneficial. However, this is a double-edged sword, because pressures that are too high increase edema, leading to progressive infarction, causing tissue pressure to rise progressively, and reducing perfusion pressure farther and farther below systemic blood pressure. There is, therefore, a case for regulating blood pressure to an optimal level that maintains cerebral perfusion while minimizing exacerbation of edema. This may become possible through the recent development of methods to evaluate cerebral blood flow through widely available computerized tomography technology.6

Furthermore, as Del Maestro and I pointed out in 1985,7 there are some circumstances in which the blood pressure must be treated, despite the occurrence of a recent cerebral infarction: in hypertensive encephalopathy, in patients whose cerebral infarction is because of embolization from a recent myocardial infarction, with pulmonary edema resulting from or aggravated by high pressure, or in patients whose stroke was because of aortic dissection picking off a carotid origin, there is simply no choice but to treat the blood pressure. The question then becomes how and how low? A key issue in that regard is that drugs that cannot be controlled, such as sublingual nifedipine, are contraindicated in this situation.5,7 In principle, therefore, it is best to use short-acting drugs that are administered by intravenous infusion so that blood pressure can be carefully titrated. Transdermal administration of drugs would fit with that principle, because they can be stopped by removing the patch and cleansing the skin underneath.

With the recent widespread use of thrombolytic therapy for acute stroke, the need to control pressure to <185 mm Hg systolic and 110 mm Hg diastolic has become an imperative.8 The publication of the Acute Candesartan Cilexetil Therapy in Stroke Survivors (ACCESS) study,9 which showed improved outcomes with candesartan treatment between days 1 and 10 among patients with acute stroke and hypertension, has unfortunately not clarified this issue by much, because there was no blood pressure difference between patients treated with candesartan versus placebo. Furthermore, the administration of oral drugs, which cannot be retrieved if the pressure goes too low, is problematic.

In that regard, the report by Wilmot et al10 in this issue of Hypertension lends strong support to the use of transdermal nitrates to treat hypertension in acute stroke. They showed that blood pressure could be lowered substantially, without reduction of cerebral perfusion in the ischemic region. Important advantages of the nitrate patch are that it is easy to implement, and, more importantly, it can be removed if the pressure is dropping too low. The results of their main trial, the Efficacy of Nitric Oxide in Stroke Trial (ENOS), to determine whether blood pressure reduction improves outcomes in patients with high blood pressure and acute stroke, are, therefore, to be eagerly anticipated.

References
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