Hypertension is frequent and transient after acute stroke. Observational studies have shown a U-shaped relationship between blood pressure (BP) immediately after stroke and clinical outcome. In one study the best prognosis was associated with a systolic BP around 150 mm Hg, whereas in another, the worst was associated with a rapid decrease of pressure and with a baseline pressure above and below 180 mm Hg.

Whether or not such BP increase should be corrected early after stroke is debated. It is established that a smooth BP reduction reduces the risk of cerebral edema, hemorrhagic transformation, stroke recurrence, and cardiovascular complications. In contrast, during the first 24 to 48 h, a BP decrease may hamper cerebral perfusion, extend the ischemic area, induce irreversible damage, and worsen the disabling consequences of the initial stroke because cerebral blood flow autoregulation is transiently lost in the tissues surrounding the ischemic core. Therefore, a reasonably high BP is desirable to reduce the brain damage, until the autoregulation is restored and any further neurologic improvement unlikely. The BP target during this acute phase is around 180 mm Hg systolic and 105 mm Hg diastolic in previously hypertensives and 160 to 180/90 to 100 mm Hg in previously normotensives.

Angiotensin-converting enzyme (ACE) inhibitors could cause an abrupt BP decrease in patients with activation of the renin-angiotensin system. Therefore, Eveson and co-workers examined the safety and efficacy of lisinopril, at the starting dose of 5 mg, orally, for 1 week, in mild hypertensives, starting at 20 h after ischemic stroke. They carefully excluded patients at risk of excessive hypotension with ACE inhibitors (renovascular hypertensives, previous diuretic administration, evidence of dehydration, congestive heart failure, and others). The BP reduction with lisinopril was not associated with neurologic and clinical outcome different from placebo, which, to the investigators’ interpretation, is an indication of its efficacy and safety after acute stroke, both of which are doubtful for the following reasons. The BP decrease after the first lisinopril dose was larger than recommended in some stroke patient despite a careful selection (up to 55 mm Hg systolic and 25 mm Hg diastolic 4 h after dosing). The similar outcome of treated patients despite the lower BP suggests that either the antihypertensive drug administration after less than 24 h from stroke onset is ineffective on clinical outcome or that any benefit is overridden by a too large short-term BP reduction. It is worth noting also that the article is a preliminary study and may not have sufficient statistical power to address the impact of marked BP reduction in the relatively small number of subjects in which it occurred. Yet it might become the basis for general acceptance of lisinopril in the acute post-stroke period and fall into the melting pot of meta-analyses on efficacy and safety of antihypertensive drugs after stroke.

Until we have more convincing data, BP control within the first hours after stroke with ACE inhibitors rests only on sound clinical reasoning, starting at very low doses, with careful up-titration. Whether this is useful for the patient’s clinical outcome is still unproven.

References


See related article on page 270.