Several observations have demonstrated spontaneous elevation of blood pressure in the first 24-48 hrs after stroke onset with a significant spontaneous decline after few days. However, it may be important to maintain the hypertensive state due to the damaged autoregulation in the ischemic brain and risk of cerebral hypoperfusion exacerbated by the lowered systemic blood pressure.

For many years the use of blood pressure augmentation (“induced hypertension”) has been studied in animal models and humans as a means of maintaining or improving perfusion to ischemic brain tissue. This approach is now widely used in neurological care unites to treat delayed neurological deficits after subarachnoid hemorrhage, but its use in ischemic stroke patients remains anecdotal. Several studies have addressed this question and administrated vasopressors, including phenyleprine and norepineprine, to patients with acute stroke. Despite of documents improvement in CBF, the concept was abandoned because of the increased risk of hemorrhage and brain edema.

Despite of the somewhat confusing and unclear data current ESO guidelines recommended that blood pressure up to 200 mm Hg systolic or 110 diastolic may be tolerated in the acute phase without intervention unless there are cardiac complications. According the American guidelines it is generally agreed that patients with markedly elevated blood pressure may have their blood pressure lowered in not more than 15% during the first 24 hours after the onset of stroke. There is an indication to treat blood pressure only if it is above 220 mm Hg systolic or if the mean blood is higher than 120 mm Hg. No data is available to guide selection of medication for lowering of blood pressured in the setting of acute ischemic stroke. The recommended medication and doses are based on general consensuses.