Considerable effort recently has been invested in developing effective therapeutic agents that can arrest, delay, or reverse cerebral ischemia and neural injury. Current research has focused mainly on 4 pharmacologic treatments: (1) heparin, (2) calcium channel blockers, (3) thrombolytic agents, and (4) neuroprotective medications. Current available research has neither supported nor discredited the efficacy of heparin for acute stroke management, and clear guidelines for its use are lacking. The prevailing evidence supports the use of antiplatelet agents for the treatment of thrombotic stroke. Warfarin seem warranted if the risk of bleeding is otherwise low. Calcium channel blocking agents, such as nimodipine, are effective in the prevention of death from vasospastic complications of subarachnoid hemorrhage (SAH) and are recommended for routine use during the first 21 days after hemorrhage. Several trials testing nimodipine for acute ischemic stroke do not shown any efficacy. The use of thrombolytic agents in acute stroke has been successful in achieving recanalization of occluded cranial arteries in studies that enrolled subjects within the first 2-6 hours after onset of infarction. Although the use of agents such as tissue plasminogen activator (tPA) seems promising, acute hemorrhagic complications have limited their use.

Management of spasticity requires careful evaluation, goal setting, and selection of appropriate therapies. Dantrolene has been used for many years for pharmacologic treatment of hemiplegic spasticity caused by stroke, but early use of Dantrolene did not improve function in a recent double-blind study. Spasms usually can be controlled adequately by small doses of diazepam before bedtime. For localized spasticity intramuscular neurolysis with phenol or intramuscular botulinum toxin injections can be very effective. Another class of medications for the treatment of acute stroke is the neuroprotective agents. In particular, the N-methyl-Daspartate (NMDA) receptor antagonists have shown the potential to delay neuronal injury. During ischemic injury, excitatory neurotransmitters, such as glutamate and aspartate, are released extracellularly. In high concentrations, these amino acids act on NMDA membrane receptors, causing an influx of cations that result in rapid neuronal death. Controlled studies testing the ability of NMDA antagonists to prevent cytotoxic injury during acute stroke are underway. Some other agents have been used to treat stroke, such as antioxidants, barbiturates, beta-adrenergic blockers, calcium channel blockers, corticosteroids, dextran, hyperventilation drugs, naloxone, and vasodilators. Various opinions exist concerning the usefulness of these agents, but their effectiveness has not been demonstrated.

Secondary prevention

The results from the North American symptomatic carotid endarterectomy trial and the European carotid surgery trial showed the beneficial effects of carotid

endarterectomy in individuals with high-grade carotid stenosis (70% to 99%).
Other studies has been unable to demonstrate the benefit of surgery in patients with less than 70% stenosis.
At present, there is no general consensus on appropriate treatment for an individual who presents with asymptomatic carotid artery stenosis of 60%. Perioperative risks greater than 3% can eliminate any benefit from surgery.
Carotid angioplasty is a possible alternative to carotid endarterectomy for the treatment of carotid stenosis. Some clinicians attributed the poor outcome in the stent group to inexperienced endovascular interventionalists. Further investigation into the efficacy of carotid angioplasty and stenting in the treatment of carotid stenosis is ongoing.
For patients with a noncardioembolic (atherothrombotic, lacunar, or cryptogenic) stroke or transient ischemic attack with no contraindication to antiplatelet therapy, the recommendation was to administer an antiplatelet agent regularly to reduce the risk of recurrent stroke and other vascular events. Initial therapy is considered aspirin 50 mg to 325 mg daily. For cardioembolic stroke, the recommendation was to use long-term oral anticoagulation to prevent stroke in patients with atrial fibrillation who have had a recent stroke or transient ischemic attack.