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Atherosclerotic disease affects the great vessels of the neck and head and can be the cause of profound neurologic insult. Surgical repair of lesions at the origin of these vessels is not as simple as that for cervical carotid bifurcation stenosis, and therefore, indications for endovascular therapy must be more flexible. Specifically, surgical repair of the aortic branches is difficult, but their repair is straightforward from an endovascular approach. The stenoses are typically neither as friable as carotid bifurcation lesions nor as often a source of emboli, but these data are not determined accurately. These stenoses can be dilated with or without stent placement with a high rate of success and durability and with low morbidity (1–9). Because of the difficult surgical approach and the relative safety and efficacy of endovascular reconstruction, angioplasty and stenting have largely replaced the direct surgical approach for repair of these lesions.

Indicator	Threshold of Performance (%)
Ability to successfully access vessels	>95
Ability to deploy the stent	>95
Successful completion of procedure without neurologic sequelae	>95
Mortality	<2

Angioplasty and Stenting for Intracranial Atherosclerotic Disease

Intracranial atherosclerotic disease accounts for a significant portion of all ischemic strokes. The authors who presented the Warfarin-Aspirin Symptomatic Intracranial Disease Study (10) stated that 5% to 10% of all ischemic strokes are directly attributable to intracranial atherosclerotic lesions, thus accounting for approximately 50,000 to 70,000 strokes per year. African-American and Asian persons may have more intracranial vascular stenoses than extracranial carotid stenoses (11–14).

Previous studies have indicated a stroke risk of 8% per year to >20% per year with aspirin therapy for various intracranial stenoses of >50% severity, with or without previous symptoms. Warfarin therapy may lower this risk to <10% per year (10), but a recent report indicates that even warfarin therapy may be associated with an extremely high failure rate (15). Furthermore, another trial found that

the medium term risk of stroke from an asymptomatic lesion (36% stroke, 45% death) was similar to the risk of stroke from a symptomatic lesion (45% stroke, 42% death), possibly because the insult from these lesions may not be embolic in nature but rather due to hemodynamic insufficiency (16). No trial of patients with extracranial stenoses has systematically evaluated the risk of stroke when treated with warfarin or found such a high risk of stroke as that found in the studies of intracranial stenoses. In addition, no trial has evaluated the true risk of a severe (>70%) intracranial stenosis, which would be expected to have a higher risk than the 50% diameter stenosis lesions previously studied.

For comparison, intracranial atherosclerotic disease is associated with a far higher risk of stroke and/or death than an unruptured aneurysm or an unruptured AVM. Intracranial atherosclerotic stenosis presents a higher risk for stroke than does a dural AVF, spinal AVM, or vertebral AVF. Available evidence suggests that even asymptomatic intracranial atherosclerotic stenosis is associated with a far higher risk for stroke than is asymptomatic extracranial carotid stenosis and that symptomatic intracranial stenosis probably has a higher cumulative risk than does symptomatic extracranial stenosis. Therefore, severe intracranial stenosis is one of the most intrinsically high risk vascular conditions encountered by neurointerventional surgeons. Although medical therapy probably decreases the risk of subsequent stroke from intracranial stenosis, it has been shown to have a disappointingly high failure rate (15–17). An additional trial is now underway to further evaluate the risk of stroke when patients are treated by medical means only.

Therapy for intracranial stenosis has been attempted by surgical as well as medical means. The Extracranial-Intracranial Bypass Study showed that vascular bypass procedures did not provide clinical benefit, with the worst results occurring in the group expected to do the best (those with stenosis of the MCA) (17).

Because of the high intrinsic risk of intracranial atherosclerotic stenosis, efforts to find and/or perfect a treatment for this condition should continue. Although new pharmaceutical agents are being developed, there currently is no available medication that effectively lowers the risk of stroke to an acceptable level (10, 15). Intracranial angioplasty and/or stenting offers the chance to maintain or re-

store normal hemodynamics and decrease the risk of stroke (18–41). New catheters, balloons, and stents allow the procedure to be performed with greater safety and success than was previously possible. Recent reported results indicate that this procedure is now relatively safe (35, 38, 40, 41).

The ASITN, therefore, maintains that intracranial angioplasty is appropriate for certain selected patients and can be performed with acceptable success rates and with beneficial results. Note, however, that no disease should be treated if the therapy presents a higher risk than the condition. For this reason, intracranial angioplasty for atherosclerotic stenosis should be performed only by a qualified neurointerventional surgeon on appropriately selected patients.

Success Rates for Intracranial Angioplasty

The ASITN encourages efforts to further study this disease process and its effective therapy. The ASITN maintains that it is premature to establish definitive success rates and threshold values for this procedure, particularly because the term intracranial stenosis encompasses lesions in different locations and with varying anatomy and that risk: benefit ratios are dependent on severity of stenosis, location, symptoms, and other factors. As additional data become available, the Society will endeavor to establish guidelines for technical success rates, complication rates, and threshold criteria. Success rates for emergent intracranial angioplasty associated with neurovascular rescue are particularly dependent on the preexisting cerebrovascular and parenchymal statuses and cannot be specified.

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