Recent developments in stroke prevention have once more followed the cardiac paradigm, where removal of a symptomatic atherosclerotic source can improve clinical outcomes. Coronary artery stenting is now the mainstay of treatment of severe, symptomatic coronary artery disease. In the early 1990s, proof for the benefits of carotid endarterectomy for symptomatic extracranial atherosclerosis was provided by large randomized clinical trials. There is now accumulating experience and evidence concerning the role of carotid stenting for extracranial disease, with some encouraging results in high-risk patients, although considerable uncertainty of risks and benefits still exists.1,2,3

Analogous to extracranial disease, it seems biologically plausible that removal of an intracranial atherosclerotic source of embolism or distal hemodynamic disturbance might reduce the risk of stroke. As pointed out by our 2 protagonists, it is still early days, with limited evidence and lack of prospective, controlled trials of intracranial stenting. There is no doubt that patients with high-grade middle cerebral artery stenosis are at high risk of stroke, suggesting that aggressive interventions might be warranted for patients who fail optimal medical therapy. The hope that warfarin would fulfill this role has now been discounted, with lack of benefit of anticoagulation over aspirin and an unacceptably high risk of hemorrhage.4

Clearly, the periprocedural risks of intracranial stenting are considerable, but with technological advances and greater clinical experience, they are likely to diminish with time. The longer-term clinical benefits remain uncertain. Such developments would parallel the evolution of extracranial stenting in the era of the distal protection devices.5 Both protagonists have endorsed the importance of randomized controlled trials to determine the risks and benefits of intracranial stenting. We would support this approach, but question when this should occur, given the inevitable improvement in technology. For these trials, a key issue is how failed medical therapy should be defined. There are ongoing developments in secondary stroke prevention. Recently, statins have been shown to lead to regression of extracranial atherosclerosis, and 1 trial showed a reduction in the risk of stroke.6,7 Hence, the criteria for failed medical therapy need to be constantly reviewed. Is there a role for intracranial stenting for medically refractory high-risk patients in experienced centers? We would favor managing such patients in the context of phase II trials, so that additional safety and outcome data can be obtained while awaiting technological improvements and the inevitable phase III trials.

We should not underestimate the importance of intracranial atherosclerosis as a management issue in many parts of the world, particularly in Asia. At least half of the incident strokes occur in this part of the world, where the intracranial circulation is the dominant location of atherosclerosis. Given the importance of the problem, research in this area must have a high priority.

Disclosures
None.

References

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