Intracranial Stenting With Wingspan
Still Awaiting a Safe Landing

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The rate of 5% (5 strokes with no fatalities). Over a mean follow-up of 21.4±8.2 months, with no patients lost to follow-up, there were an additional 4 ipsilateral strokes giving a cumulative primary event rate of 9% or approximately 5.04%/year. The secondary event rate (nonipsilateral stroke, intracerebral hemorrhage, nonstroke mortality, TIA, or TLR) was 15%; 9% <30 days (7 TIA, 2 emergency TLR) and 6%>30 days (TIA). Angiographic follow-up was available in 44% at a mean of 8.6 months: 26.7% had an in-stent stenosis with approximately 43% symptomatic.

What these 2 studies show is that the current generation of the Wingspan stent is highly deliverable and may be associated with a low risk of perioperative complications. However, the device is associated with a high risk of restenosis of approximately 30% in the first year, which is often symptomatic requiring repeat intervention. The restenosis occurs mostly within the first 6 months and the risk of recurrent events and restenosis is probably reduced beyond 6 months. Clinical events were associated with cessation of dual antiplatelet therapy, particularly within the first 3 months. The longer-term outcomes are not as consistent. The Fiorella et al study appears to show a higher risk of recurrent events and if TLR is included as an outcome, then the annual event rate is approximately 30%/year, which is greater than the 22.5%/year rate noted in the >70% subgroup in Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) and far greater than the approximately 12%/year rate seen overall. The results by Jiang et al are more favorable toward stenting, particularly because they treated the highest-risk (>70% stenosis) patients.

There were many notable differences between the 2 studies. To begin with, the outcomes in the Fiorella et al study were not determined by independent neurologists but by “investigators at the individual sites,” whereas Jiang et al used independent stroke neurologists. On the other hand, some (percentage unknown) of the follow-up in the latter study was by “telephone.” Jiang et al published detailed exclusion criteria including: age ≥75 years, lesions >15 mm long, ≤2 mm diameter, and cardiac sources of embolism. The study by Fiorella et al does not detail any patient or lesional characteristics as either inclusion or exclusion criteria. There were also major differences in the interventional protocols; although both groups gave their patients aspirin and clopidogrel, Jiang et al gave every patient intravenous nimodipine.

The opinions in this editorial are not necessarily those of the editors or of the American Heart Association.

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Intracranial atherosclerotic disease (ICAD) is a major cause of stroke and yet there are no proven effective treatments for it. Medical therapy has been associated with a high rate of recurrence, particularly in those with the most severe stenoses who have a 22.5% risk of stroke or death in the first year after a stroke. Clearly better alternatives are needed. Surgical bypass has been shown in at least 1 trial to be worse than medical therapy in the era before thienopyridines, statins, and angiotensin-converting enzyme inhibitors. In this issue of Stroke, Fiorella and colleagues describe their longer-term experience with intracranial stenting with the Wingspan stent system and last year in this Journal, Jiang and colleagues published their long-term experience with 100 consecutive patients also treated with the Wingspan stent system. The question is do these publications broaden our knowledge enough to alter management of patients with ICAD? Do they prove that endovascular therapy is superior to medical treatment and that it is safe enough to be performed routinely?

The article by Fiorella et al represents the largest (N=158) published series of Wingspan in the United States to treat ≥50% symptomatic stenoses. The immediate perioperative stroke and death rates were 5.7% (N=9) and 2.5% (N=4), respectively. Over a mean follow-up period of 14.2 months (only 110 patients [69.6%] had 12 months of follow-up), the primary end point (any perioperative stroke or death and any ipsilateral stroke thereafter) was noted in 15.7% of patients or approximately 13.2%/year. Seventy-six percent of the ipsilateral strokes occurred within the first 6 months with no events beyond 12 months. These event rates did not include transient ischemic attacks (TIAs) and did not include target lesion revascularization (TLR) for in-stent stenosis. The exact number of TLR events was not presented, but the authors had previously published an in-stent stenosis rate of 27.9% with 80.6% of these undergoing TLR.

Jiang and colleagues reported on 100 consecutive patients from a single center with a 99% stent placement success rate. The authors reported a 30-day perioperative stroke and death rate of 5% (5 strokes with no fatalities). Over a mean follow-up of 21.4±8.2 months, with no patients lost to follow-up, there were an additional 4 ipsilateral strokes giving a cumulative primary event rate of 9% or approximately 5.04%/year. The secondary event rate (nonipsilateral stroke, intracerebral hemorrhage, nonstroke mortality, TIA, or TLR) was 15%; 9% <30 days (7 TIA, 2 emergency TLR) and 6%>30 days (TIA). Angiographic follow-up was available in 44% at a mean of 8.6 months: 26.7% had an in-stent stenosis with approximately 43% symptomatic.
stenting is to restore nonturbulent flow to the distal territory and stenting will cause plaque shifting into the ostia of the perforators completing the infarct. This was not an exclusion criterion in the Fiorella or Jiang studies or SAMMPRIS.

There are many unanswered questions regarding the endovascular techniques, particularly whether it was wise not to postdilate after deploying the Wingspan stents. The instructions for use prohibit postdilation presumably to avoid stent strut fracture, but to not do so may increase the risk of acute and subacute stent thrombosis and early restenosis. In my practice, I postdilate all Wingspan stents to ensure optimal stent wall opposition as well as to maximize the residual lumen area, which is the best predictor of restenosis, a fact learned from my interventional cardiology colleagues. Interventional cardiologists have amassed a wealth of information on angioplasty and stenting of vessels similar in size to the cerebral vessels and affected with the same disease. These data include information on optimal periprocedural and maintenance medical management, who should have stenting, who is at highest risk for perioperative events and restenosis, how to avoid periprocedural events and restenosis, which devices are better than others, how to monitor for restenosis, how to manage restenosis, etc. In short, the answers to many of the questions that remain outstanding in the management of ICAD may already be available in the coronary literature. For example, dual antiplatelet therapy has clearly been proven essential and its prolonged use is associated with better outcomes compared with aspirin monotherapy in patients with coronary stents. The cardiac literature is replete with studies of variable clopidogrel and aspirin response, which have been associated with a markedly increased rate of perioperative adverse events, including acute stent thromboses. An assessment of clopidogrel response was not part of the SAMMPRIS trial and has only been mentioned in a few reports of intracranial interventions. In addition, postoperative heparin has been associated with increased complications and no significant benefits after coronary artery stenting, yet many reports of stenting for ICAD, including the article by Jiang et al, describe prolonged heparin use, some with an associated increased rate of intracerebral hemorrhage. Al- most all coronary stents on the market today are balloon-expandable not self-expanding like Wingspan. Although self-expanding stents were touted to continue to expand after placement and to decrease the risk of restenosis, the reality was that they incited greater neointimal proliferation and the same degree of late luminal loss.

The most important information we can glean from the cardiac literature is how to select patients for stenting and how to reduce complications and restenosis. For example, diabetic patients and those with small vessels (ie, <3 mm diameter but especially <2.5 mm diameter) have among the highest rates of restenosis. Although Jiang et al did not stent patients with vessels <2 mm, Fiorella et al do not give us this information or the information on who developed restenosis; on a related note, restenosis and TLR are considered major adverse events in the coronary literature and they need to be reported as such in the neurointerventional literature. Ensuring complete stent expansion and wall opposition, especially in calcified lesions, and detection and treatment of residual
dissections can prevent acute thrombosis. The coronary literature also tells us that powerful tools for the reduction of restenosis are the drug-eluting stents. There are many other facts that can help us design better trials and devices for the treatment of ICAD. Briefly, however, what is needed now is: (1) a concerted effort to better define which patients are best treated with medical therapy and which by endovascular therapy; (2) standardized definitions of qualifying events and outcomes; (3) functional imaging in all patients to determine that distal territory tissue is at risk rather than tissue supplied by perforators; (4) definitions of which patients have atherosclerosis as the etiology as opposed to vasculopathies or vasculitides; (5) a standardized, well thought out endovascular approach that takes into account the vast knowledge base gained from the endovascular treatment of coronary artery disease; (6) validation studies of the potential benefits of awake interventions; (7) next-generation devices that minimize the neointimal response yet that are easily and safely delivered to the cerebral vessels; (8) mandatory cerebral angiography in all patients to provide the true denominator for rates of restenosis and stent thrombosis along with angiographic definitions of both that take into account the continuous rather than dichotomous nature of in-stent stenosis<sup>6</sup>; (9) standardized perioperative and postoperative antithrombotic therapy as well as universal risk factor modification; (10) patient and caregiver education must be standardized and given to all with emphasis on compliance with dual antiplatelet and disease-modifying therapies; and (11) long-term follow-up for 5 to 10 years must also be performed because of the risk of atherosclerotic lesion recurrence/progression that differs from the early cause of in-stent stenosis, neointimal hyperplasia.

Finally, we need to move away from an endovascular dogma that says “my mentor or chief did it this way so it must be the right way” to a more rigorous scientific approach that is focused on understanding the disease and one that is not afraid to acknowledge that physicians of other specialties may have answers to our most pressing questions. We do not need to reinvent the endovascular wheel for every vascular bed; our patients with ICAD deserve better.

**Disclosures**

The author is the site Principal Investigator in the Vitesse Intracranial Stent Study for Ischemic Therapy (VISSIT) trial of a balloon-expandable stent sponsored by Micrus Endovascular, Inc and is on the Speakers’ Bureau for the BMS/Sanofi partnership.

**References**


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