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Wherefore Wingspan?

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COMMENTARY

Wherefore Wingspan?

Without industry-sponsored development of endovascular devices, the field of interventional neuroradiology, if it existed at all, would be relegated to a tiny niche specialty. We are dependent upon industry for essentially all aspects of our field, from access tools to microcoils. Many new devices represent obvious improvements over existing technology—for example, microcoils in place of detachable balloons for endosaccular aneurysm occlusion.

It is against this background that we are struggling with the proper application of the Wingspan stent (Boston Scientific, Natick, Mass). Wingspan was approved as a humanitarian device exemption (HDE), which is reserved for devices for which "no *comparable* [our italics] device. . . is available to treat. . . the condition."[1] The current treatment options for intracranial vascular disease include medical therapy, surgical bypass, and endovascular treatment, typically balloon angioplasty. When we use an HDE-approved device, we are acknowledging that other therapies are not "comparable" with Wingspan. From our vantage point, however, there is little or no information to make that assessment.

Intracranial angioplasty has been performed for many years, with most recent case series suggesting acceptable morbidity and durability of treatment by using that approach.[2-4] All of us have a mortal fear of intracranial angioplasty ending badly, such as dissection leading to acute occlusion or substantial arterial rebound with compromise of antegrade flow. Recent angioplasty series suggest that approximately 1 in 5 patients will "need" stent placement after angioplasty for dissection, rebound, or late restenosis.[3,4] We have limped along by using coronary stents to treat these feared outcomes, but often as not, the relatively stiff coronary devices are difficult or impossible to place where they are needed. Furthermore, we have watched the cardiologists struggle with high rates of restenosis with balloon angioplasty alone, which leads to near-universal application of stents in coronary interventions.

With the background of poor bailout options as well as an overall perception that stents are good, or at least better than a balloon alone, it stands to reason that we should yearn to embrace the Wingspan stent. One might conclude that Wingspan at the very least should be deployed for any postangioplasty result considered at risk for abrupt closure because there likely is no "comparable" device to treat these complications. However, should one apply Wingspan beyond a bailout option? Is there really no "comparable" device? Stated differently, is Wingspan better than a good angioplasty result?

The current peer-reviewed literature on Wingspan includes a single case series of 15 patients followed for 4 weeks[5] and a recent series comprising 78 patients with periprocedural follow-up.[6] These series suggested that the device probably is safe, or at least not dangerous. However, there are no data presented that would convince the reader that routine use of a Wingspan stent is better than a good angioplasty result without a Wingspan stent. (Indeed, those series had no cases of dissection or rebound after angioplasty, so we cannot even determine whether Wingspan can treat those eventualities). It is not yet clear that Wingspan can even improve substantially on the immediate angiographic result because its radial force likely is not that of a coronary stent. The data from the 1 published series noted improvement from 54% stenosis to 38% stenosis before and after Wingspan deployment.[5] The other series noted improvement from 44% stenosis to 27% stenosis before and after Wingspan deployment.[6] However, no statistical analysis was offered to determine whether these differences were statistically significant. Furthermore, no details were given as to whether the readers of the angiograms were blinded to the presence or absent of a stent, how many readers were included, and how much variability there was in the readings. In our experience, substantial variation can be present when one is quantifying stenoses in small arteries.

Notwithstanding our natural affinity for stents of all types, available data supporting intracranial stent placement rather than simple angioplasty remain sparse. The Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA) trial, which focused on stents, showed a nearly 13% annual stroke rate,[7] compared with an annual stroke rate of approximately 3% in the vascular territory of the treated stenoses in a recent angioplasty-focused series.[4] The SSYLVIA trial also demonstrated that 37% of patients who underwent 6-month follow-up angiography had lesion restenosis of greater than 50%, which is unacceptable.[7]

Specific to the Wingspan system, data are available from the HDE safety study,[8] as well as from the recently reported results of the Wingspan postmarket registry presented at the International Stroke Conference in February 2007.[9] Again, the data are not compelling in favor of primary stent placement. The Wingspan HDE study had a 10.3% rate of stroke in the territory of the stenosis or death, not dissimilar to the rate of medical therapy alone. The outcomes seen in the postmarket registry are similar to those of the HDE study, with a 3-month rate of stroke and death of 13.8%. Regarding lesion restenosis, its frequency of >50% was 24.5% in the postmarket registry, though this rate was based on follow-up of only 49 (38%) of 131 patients with a short-term follow-up average of 4.7 months.[9] This rate may increase with more and longer term follow-up. The purpose of this registry was to shed light on the safety and performance of the Wingspan stent-placement system. Unfortunately, this stent did not perform well in either criterion when compared with data on medical therapy alone or with primary angioplasty.

We are aware that single centers have applied Wingspan in larger series of patients, but long-term results of (or rationale for) routine use of Wingspan stents have not yet undergone peer review. Because long-term data are so limited, it is conceivable that Wingspan may lead to either no improvement or even worsening of late restenosis. Use of the Wingspan may lead to higher rates of subacute thrombosis. Anything could happen; witness the recent disclosure that drug-coated coronary stents are at risk for sudden thrombosis, even with patients on stable antiplatelet therapy.[10] The fact remains that we just do not know.

We suspect that journal reviewers will soon be evaluating a multitude of additional Wingspan stent case series, and likely these case series will find their way into the literature quickly. It is possible that outcomes from these case series will be so compelling as to allow valid comparison against historic con-

trols of angioplasty series. However, in all likelihood, the Wingspan stent data will be "pretty good," just as angioplasty alone seemed "pretty good" for a long time.

Consideration of these issues is not simply an intellectual exercise. There is at least a perception in our neuroradiology community and among our neurology colleagues that the Wingspan is good, and perhaps even revolutionary. Perhaps it is all in our imagination, but we feel some pressure to place a Wingspan stent irrespective of an angioplasty result. We wish we knew the truth.

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Erratum

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