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How Do We Spin Wingspan?

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COMMENTARY

How Do We Spin Wingspan?

We applaud Levy et al[1] for the prompt publication not only of the overall restenosis rates in their initial large series of Wingspan stent (Boston Scientific, Natick, Mass) cases but also with their follow-up article in this issue of the *American Journal of Neuroradiology* (*AJNR*)[2] of rates of restenosis based on age and location. In their initial series detailing the periprocedural results, they concluded, "Our initial experience indicates that this procedure represents a viable treatment option for this patient population." Notwithstanding these apparently promising periprocedural results from the initial series, the rationale for using the Wingspan without understanding the long-term outcomes was questioned in a recent *AJNR* commentary in which one of us was a coauthor.[3] In that commentary, it was noted that the viability of the Wingspan depended on further follow-up data, specifically the rate of late restenosis. Such further follow-up data are now available.

In their new article, Levy et al[2] begin their introduction with "Endovascular treatment of symptomatic intracranial stenoses has recently progressed with the availability of Wingspan. . . . " On the basis of our reading of the first follow-up article and the current article, with disturbingly high rates of restenosis, we would probably choose a different word from "progressed." From our perspective, it would be reasonable to have written, "Endovascular treatment of intracranial stenosis has recently taken off like wildfire with the availability of Wingspan despite a lack of any convincing evidence that it represents an improvement in patient therapy."

The Humanitarian Device Exemption (HDE) approval that was granted for Wingspan by the US Food and Drug Administration (FDA) is supposed to be available "when no comparable device is available to treat or diagnose the condition."[4] However, comparable coronary devices have been successfully used to treat intracranial stenosis before and after the introduction of Wingspan, and these coronary devices might lead to lower rates of restenosis than those seen with Wingspan. The overall restenosis rate in the study by Levy et al[1] was 31%, even though they excluded 4 cases of complete occlusion. Including those cases of complete occlusion would have increased the reported rate of restenosis by approximately 4%. Those authors also used a new restenosis definition that biases toward a lower rate of restenosis than previous definitions. Specifically, in addition to a binary decision regarding greater than or less than 50% stenosis, the lesion also had to have progressed at least 20% to be considered "restenosis." This additional criterion of at least 20% restenosis would diminish reported rates of restenosis as compared with prior articles that used the binary criterion, greater than or less than 50%, alone (eg, a 36% stenosis that progressed to 55% would not be counted as a restenosis). In the SSYLVIA trial of balloon-expandable stents to treat intracranial stenosis,[5] the binary restenosis rate was 32.4% for intracranial arteries 6 months following treatment. The Neurolink stent used in SSYLVIA was never FDA-approved or marketed, but these results could be reasonably expected to apply to coronary balloon-expandable stents used in the intracranial circulation. Wojak et al[6] reported a 27.4% restenosis rate in a series of patients treated primarily with angioplasty, by using balloon-expandable stents very selectively in only a few of these patients. Wojak et al defined "restenosis" as "any worsening of stenosis after angioplasty" (Joan Wojak, personal communication, October 4, 2007), which biases toward a higher restenosis rate than the binary restenosis definition. With different restenosis definitions among various articles, we are forced to compare "apples and oranges," but the restenosis rate for Wingspan appears worse than published data on other devices, despite applying a restenosis definition that biases toward a lower rate of restenosis.

The data discussed previously certainly do not statistically prove that recurrence rates are worse with Wingspan as compared with balloon angioplasty alone or balloon-expandable stents, but we believe that it would be foolish to ignore this distinct possibility. The widespread use of Wingspan seems to be driven by the HDE approval process rather than evidence of efficacy and safety, and ironically, this HDE process that is supposed to make devices available to patients who have no other options may have incited the widespread used of Wingspan at the expense of physicians largely giving up the off-label use of potentially more effective coronary devices. The low radial force self-expanding design of the Wingspan stent may very well not be the best device to treat intracranial atherosclerosis. Maybe angioplasty alone or a balloon-expanded stent placement is a better option. Moreover, as Levy et al[2] noted, drug-eluting stents may be important in the avoidance of restenosis of intracranial atherosclerosis in the future.

Although subgroup analysis, as presented in the new article, may help uncover important biologic differences based on age, sex, or lesion location, we worry that our community may lose sight of the forest for the trees. The authors have concluded that ". . . avoiding these (high risk) lesions, the rates of in-stent restenosis . . . after Wingspan can be substantially reduced."[2] Faced with the same data and comparing it to published restenosis rates for non-Wingspan procedures, we might have written something like "by avoiding treatment of any lesions with Wingspan, the rates of restenosis might be substantially reduced." We, the community of physicians, really have to continue to ponder what the real value of Wingspan is, and we must demand more data about safety and efficacy relative to other treatment options.

It is puzzling that the FDA requires physicians to use HDE devices under institutional review board approval and supervision but then provides no requirements or guidelines about the systematic collection of data—that is, there is a lack of postmarket surveillance for devices approved under an HDE, to our knowledge. Many of us are involved in FDA-mandated postmarket surveillance registries for carotid stents, devices that seem pretty safe and have already been the object of hundreds of studies comprising thousands of patients. However, for devices approved under an HDE after registries of a few dozen patients, we are not aware of a similar mandate for postmarket studies. To us, this seems like a regulatory inconsistency.

Our comments may seem harsh or mean-spirited. However, we are merely trying to point out that HDE approval is given by the FDA following the submission of minimal data

and we need to keep an open mind about the safety and efficacy, or lack thereof, of such devices. Moreover, we should not ignore our past experience with off-label use of coronary devices to treat effectively intracranial atherosclerosis. We do not fault industry for gaining approval that they deem appropriate and then marketing the device in an FDA-approved manner, but, if we, the treating physicians, do not demand more and better data, it is likely that the Wingspan saga will not be unique.

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