clinical trial that assessed the performance of NeoVas (Lepu Medical, Beijing, China), a novel poly-L-lactic acid bioresorbable scaffold, compared with the well-known XIENCE everolimus-eluting stent (Abbott Vascular, Santa Clara, California). I want to congratulate the investigators for pursuing research on bioresorbable scaffolds after the premature "disappearance" of the Absorb platform. Many physicians and several companies are at the window to see if the increased thrombotic risk observed during the first 3 years of the Absorb follow-up is related to a young and imperfect technology or if current-generation drug-eluting stents are unapproachable, in terms of safety and efficacy, by any bioresorbable technology.

The results of the study by Han et al. (1) show a comparable effect in terms of angiographic outcomes, with a similar primary endpoint of late lumen loss. In contrast, the superiority of this bioresorbable scaffold in terms of strut coverage (an absolute difference of 2.5%) and malapposed struts (an absolute difference of 0.6%) on optical coherence tomography seems of limited importance, given that the hypothesized mechanisms of scaffold thrombosis with Absorb were related to the high strut thickness, to an inadequate technique of implantation, or to premature scaffold dismantling (2–4).

I would also like to make the following observations: 1) Despite dilation with the same size or larger balloons, at similar pressure and with the same starting reference vessel diameter, patients treated with the NeoVas scaffold had smaller post-procedural in-device minimal luminal diameter (2.58 ± 0.40 mm vs. 2.78 ± 0.42 mm; $p < 0.001$), indicating more acute recoil than in patients treated with everolimus-eluting stents. 2) At 1 year, patients treated with the NeoVas scaffold also had greater in-device late loss (0.22 ± 0.33 mm vs. 0.16 ± 0.28 mm; $p = 0.05$), indicating either more chronic recoil or restenotic tissue formation than in patients treated with everolimus-eluting stents, with at least a contribution of the latter witnessed in the optical coherence tomographic substudy by greater device volume obstruction in the NeoVas cohort (24.5 ± 5.9 vs. 10.4 ± 7.0; $p < .001$).

Taken together, these findings could be of concern when the NeoVas scaffold is studied in larger numbers of patients with more complex anatomy.

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TO THE EDITOR

Don't Leave the Back Door Open

I read with great interest the article "Neurocognition and Cerebral Lesion Burden in High-Risk Patients Before Undergoing Transcatheter Aortic Valve Replacement: Insights From the Sentinel Trial" (1). I commend the goal of the investigators to try to minimize or eliminate zones of brain infarction (as seen by diffusion-weighted imaging lesions on magnetic resonance imaging) during transcatheter aortic valve replacement. It is also very useful that they attempted to determine the relationship of these new lesions with cognitive decline. However, they did not find a relationship with worsening cognitive deficit in patients with increased volume of these diffusion-weighted imaging lesions, because of what the investigators postulate may be a "floor" effect of the patient population (i.e., these patients were already compromised, such that any further decrease would be difficult to assess). Although this is almost certainly one of the factors, it is also important to view the data acquired.

The discrepancy in the device arm between protected territories of infarction and total volume of infarction, including unprotected territories (2) speaks volumes, literally and figuratively. If I subtract the median volume of infarction in the protected territories of infarction (102.8 mm^3) from the median total volume of infarcted tissue in all territories (294 mm^3), the difference is 191.2 mm^3. Therefore, 65% of lesions were outside the

protected territories. The device covers the innominate artery and left common carotid artery and thereby the right internal carotid artery, right vertebral artery, and left internal carotid artery territories, respectively. However, it does not cover the left subclavian artery, thereby leaving the left vertebral artery territory unprotected. Because most patients are either left vertebral dominant or codominant, this leaves not only the left vertebral artery territory at risk but the basilar artery and bilateral posterior cerebral artery territories at risk in most patients.

Using an analogy, to keep our homes protected from intruders, we not only lock the front and side doors but the back door as well. By leaving the back door (left subclavian and left vertebral arteries) open, we are allowing intruders (emboli) unfettered access to our home (brain).

Although it is possible that we may protect the whole brain and get similar results in terms of cognitive changes, we cannot blame everything on the "floor" until we lock all the doors.

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REPLY: Don't Leave the Back Door Open

Thank you for the opportunity to respond to Dr. Abrams's letter. Although we disagree on some points, we greatly appreciate his interest in our work and believe the discrepancies may be related to simple misunderstandings.

First, Dr. Abrams notes that we did not find a relationship between worsening cognitive deficits and new diffusion-weighted imaging lesions in the SENTINEL study (1). We need to point out, however, that we in fact found such a relationship in that study (reported in the paper and as Online Figure 1B [1]). In the paper, we did observe that prior transcatheter aortic valve replacement studies did not observe such a relationship, which may be the source of the confusion.

Although somewhat outside the scope of the paper, we agree with Dr. Abrams's main point that the question of protected territories is important in a broader context. Let us examine his claims. He performs a calculation and concludes that 65% of lesions were outside protected territories. Unfortunately, this seemingly straightforward calculation is invalid for 2 reasons. First, and most important, the estimate must be performed on the control group, not the treatment group. To see why, one can imagine an ideal device that could prevent every single lesion in its protected zone; then, by definition, 100% of lesions would be in the "non-protected" region, no matter how small that region was. Updated with this important correction, the result of the calculation is 42%. Second, subtracting and dividing medians as proposed is not permitted statistically. The more appropriate approach is to calculate the ratio across subjects and then take its median. When both of these corrections are taken into account, the result is considerably lower: 35%.

Finally, we appreciate Dr. Abrams's door analogy. However, the problem with this assertion is that it implies that emboli will actively seek out the back door after being shut out of the front, rather than just continuing down the street.

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