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In Reply: Is There a Contribution of Anesthesia to the Outcome of the SAMMPRIS Trial?

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We appreciate the interest in our paper on the mechanisms of stroke after intracranial angioplasty and stenting for symptomatic atherosclerotic stenosis. While it is possible that general anesthesia played a role in some of the perioperative events, it is unlikely to be a major contributor to the risk of intracranial angioplasty and stenting for several reasons. The primary hypothesis advanced by Dr. Jantzen is that general anesthesia may have played a synergistic role with embolic or mechanical arterial obstruction, owing to hemodynamic factors (such as hypotension or pCO₂-mediated vasodilation that might impair autoregulation).

First, the majority of the 30 perioperative events were either hemorrhagic or not clinically evident on awakening.¹ Second, as noted by Dr. Jantzen, the majority (11) of the 14 strokes that were clinically evident on awakening were local perforator strokes. The remaining three were embolic (n=2) or mixed embolic and perforator (n=1). It is very difficult to invoke a hemodynamic mechanism for any of these events, particularly the local perforator strokes. The mechanism of displacement of plaque into perforator origins is supported by good evidence.² In addition, strokes in these perforator territories have not been associated with hemodynamic impairment, as compared to the typical “watershed distribution” strokes in the centrum semiovale and corona radiata.³

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Disclosures:

Colin P. Derdeyn, MD has relationships with companies that manufacture medical devices for the treatment of cerebrovascular disease in general, although none directly involved in this study. These are: W.L. Gore and Associates (Scientific Advisory Board and Consultant), Microvention, Inc (Angiographic Core Lab for clinical trial); Penumbra, Inc (DSMB member for clinical trial); Pulse Therapeutics (Chair, Scientific Advisory Board).

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Third, the argument advanced by the authors that the Wingspan stent may have narrowed the parent artery is unfounded. Stent placement occurred after angioplasty, and in no patient was the luminal diameter worse after treatment compared to baseline. While the parent artery and regional perforators were certainly occluded during the inflation of the angioplasty balloon, balloon occlusion of a perforator-rich vessel segment for a duration of less than two minutes is very unlikely to result in perforator stroke. We know this empirically from procedures that involve balloon occlusion of these vessels under anesthesia, such as balloon-assisted aneurysm coiling or liquid embolic delivery, that local perforator stroke is rarely, if ever, described.⁴

Fourth, we have some data from SAMMPRIS regarding procedural hypotension. We required documentation of hypotension (systolic blood pressure less than 100 mm Hg) during the procedure and out to 24 hours afterwards. None of the patients with procedural strokes had evidence of peri-procedural hypotension.

Finally, the extension of associative data from acute stroke patients to these subacute patients without active ischemia is also problematic. These studies are largely retrospective, non-randomized series of patients undergoing emergent endovascular treatment for acute stroke, not in stable patients undergoing elective intracranial stenting.^{5, 6} There is no good evidence that general anesthesia increases the risk of procedural stroke in elective carotid or intracranial revascularization procedures. In fact, the only randomized trial addressing this showed no significant difference in outcome after carotid endarterectomy in patients undergoing general vs. local anesthesia.⁷

In summary, we agree that control of CO₂ and blood pressure have important theoretical implications for perioperative management of patients with intracranial stenosis undergoing angioplasty and stenting and that it is important to monitor and maintain these within reasonable ranges. However, there is no indication from the SAMMPRIS data that these factors played a significant role in the peri-procedural stroke observed in the trial.

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