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Commentary: Determining the role of priming for spinal cord protection during open aneurysm surgery: Have we used the right model?

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Spinal cord injury (SCI) is the Achilles' heel of thoracic and thoracoabdominal aneurysm (TAA) surgery performed by either an open or thoracic endovascular aneurysm repair (TEVAR). It results in increased morbidity and mortality and, more importantly, contributes to a marked reduction in quality of life. The introduction of neuroprotective adjunct strategies that increase spinal cord perfusion and reduce metabolism and oxygen demands have now become the standard of care in both open and TEVAR TAA procedures and have significantly reduced the incidence of perioperative SCI.¹⁻⁴

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CENTRAL MESSAGE

Priming before open TAA repair may limit spinal cord injury, but a more relevant model is needed to determine its role in clinical practice.

Nevertheless, the incidence of SCI has not been totally eliminated and has been reported to average 8.26% (3.6% paresis and 5.0% paraplegia) for open TAA repairs⁵ and 12% for TEVAR (4%-8% paraplegia).⁶ This has led to the development of other strategies to reduce the incidence of SCI following TAA procedures. O'Callaghan and colleagues⁷ demonstrated that a staged TEVAR approach reduced the incidence of SCI from 37% to 11% ($P = .03$). In this series, the interval between stages averaged 5 months. Whereas staged procedures may reduce the incidence of SCI, there is concern for the combined morbidity and mortality of 2 separate surgeries, as well as the possibility of death during the interval period between the 2 procedures. Furthermore, patients with multiple comorbidities and those who develop a complication following the first procedure may never complete the

second procedure. To avoid the morbidity of 2 separate procedures, but preserve the potential of promoting spinal cord collateral flow achieved with staging, Ertz and colleagues^{8,9} developed the technique of selective coil embolization before both open and TEVAR TAA repairs. In this technique, selected segmental vessels are occluded proximally at their ostial segments using coils placed using an endovascular approach to allow the development of a collateral spinal network to augment flow to the spinal cord. This technique was used in 2 patients who subsequently underwent TEVAR⁹ and in 2 patients who underwent an open procedure 4 and 8 weeks following coil embolization.⁸ Preconditioning of the spinal cord resulted in the absence of any SCI in these 4 patients. There are, however, several issues with this technique. Coil embolization of segmental arteries is not a simple procedure and may be especially difficult in patients with tortuous vessels and those who have thrombus in the aneurysm sac. To preserve spinal collateral flow, the vessels must be occluded only at the ostium. It is not known specifically how many and which vessels need to be occluded to achieve adequate spinal collateral flow and the necessary time interval between coil embolization and the subsequent procedure, TEVAR or open. Finally, there are potential risks with the procedure, including acute SCI, the potential for injury to the segmental artery being coiled that can result in significant hemorrhage, and the inability to completely occlude the vessel.

In this issue of the *Journal*, Honkanen and colleagues¹⁰ report the results of a technique to stimulate collateral spinal blood flow by sequentially occluding intercostal arteries immediately before aortic clamping to avoid the delay and complexity of coil embolization before TAA surgery. In their porcine model, 12 piglets underwent a left thoracotomy and 8 thoracic intercostal vessels (T4-T11) were identified. In 6 animals, the aorta was clamped just distal to the left subclavian artery for 25 minutes during which time the 8 vessels were permanently occluded with clips within the first 5 minutes of aortic clamping. In another 6 primed animals, the vessels were occluded in a craniocaudal direction at 5-minute intervals. Fifteen minutes after the last segmental artery was occluded, the aorta was crossclamped for 25 minutes. Animals were observed under anesthesia for 5 hours, were extubated and their neurologic recovery was assessed daily for 5 days using a modified Tarlov score, and after which they were euthanized and their spinal cord was examined by a neuropathologist for evidence of necrosis in both the white and gray matter. Tarlov scores showed better neurological recovery in the animals that were primed before crossclamping after the first postoperative day, but there was no significant difference after that time period. The spinal cords of the primed animals showed less histopathologic changes in the T10 to T13 regions. At

the end of the 5-day observation period, 3 out of 6 control animals were paraplegic versus 0 out of 6 in the primed group.

In view of these beneficial results, should priming be incorporated as a technique to prevent SCI in patients undergoing open TAA surgery? Unfortunately, the model used by the authors differs greatly from the current standards of care to prevent SCI employed in open surgery for TAA repairs. As noted by the authors, the anatomy of the spinal cord differs between adult humans and the piglets used in these experiments. The aorta was crossclamped for only 25 minutes and only 1 crossclamp was applied. Heparin was not administered during the crossclamp period to limit spinal cord thrombosis. Animals were kept at normothermia and hindleg blood pressure was consistently <50 mm Hg. Most importantly, established bundled protocols, which have been shown to significantly reduce the incidence of SCI, were not employed.¹⁻⁴ These protocols include cerebrospinal fluid drainage to maintain a pressure <10 mm Hg during the crossclamp and postoperative periods; permissive hypothermia (32°C-34°C) to decrease metabolic demands; institution of mechanical support (left heart bypass) to maintain mean arterial pressure >90 mm Hg and a distal femoral artery pressure >50 mm Hg; attenuation of the ischemic-reperfusion injury using sodium bicarbonate after unclamping, mannitol to act as a hydroxyl scavenger and raise osmolarity,¹¹ steroids to reduce inflammation, and naloxone to reduce the release of excitatory neurotransmitters from ischemic spinal cord neurons¹²; as well as improving spinal cord oxygen delivery by maintaining a cardiac index >2.0 L/min/m² and hemoglobin >10 g/dL. Instituting these spinal cord bundles has lowered the incidence of SCI to as low as 0.65% to 3.0% in recent studies involving TEVAR patients.¹⁻³

The development of SCI following open TAA repairs is multifactorial and its prevention and treatment will require multiple strategies to maximize spinal cord perfusion and increase spinal cord ischemic tolerance. Honkanen and colleagues¹⁰ should be encouraged to continue to study priming before open TAA repair in a more clinically relevant model to determine whether priming will eventually become a component of existing bundles to limit SCI following open surgery for TAA.

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