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Commentary: Prime and prevent paraplegia

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Paraplegia in patients is tragic. Thoracoabdominal aortic aneurysm surgery can be a cause. Reported rates of spinal cord ischemia and resultant paraplegia vary with the extent of aortic resection. The rate after open repair of Crawford type II aneurysm has been reported to be as high as more than 10%.¹ Although the risk appears to be mitigated with endovascular stent-graft repair, it still happens in approximately 6% of patients.²

The spinal cord receives blood supply from multiple sources, including the branches off of named arteries, such as vertebral arteries, subclavian arteries, segmental arteries, and hypogastric arteries, as well as the paraspinous collateral network. Open thoracoabdominal aortic aneurysm procedure impairs spinal cord perfusion by reducing the systemic blood pressure (especially distal to the aortic clamp), increasing the drainage pressure, and temporarily or permanently terminating some of the inflow sources (eg, segmental arteries). Patient factors, such as extension of the preexisting aortic and iliac disease and previous history of aortic surgery, may further compromise the perfusion.

Preventive measures have been extensively studied and implemented. The systemic blood pressure may be augmented with induced proximal hypertension and distal perfusion, and the drainage pressure can be managed with cerebrospinal fluid drainage. Management of the segmental/intercostal arteries is as important. Honkanen and colleagues³ present spinal cord priming in a piglet model. In the priming group (n = 6), sequential ligation of the 8 superior most segmental spinal arteries was performed in a stepwise fashion at 5-minute intervals, followed by aortic occlusion for 25 minutes. In the

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This promising animal model shows a profound influence of timing in segmental artery ligation on the development of the postoperative paraplegia.

control group (n = 6), the aorta was first crossclamped and the 8 superior segmental spinal arteries were ligated within 5 minutes of aortic occlusion. At 5-day follow-up, 50% of the control animals developed paraplegia compared with 0% in the priming group. The perioperative motor-evoked potential assessment and histological study provided further supportive evidence of spinal protection with the priming. Here, acute sequential ligation of segmental arteries just before aortic crossclamping appears to have a beneficial effect.

Flow into the collateral network, which is an increasingly recognized important mechanism of spinal cord perfusion, is augmented in response to occlusion of feeder vessels. Selective ligation of these vessels has been posited to have a protective effect on the spinal cord, by increasing flow from other arterial feeders and reducing the risk of steal through the segmental vessels in the setting of a clamped and open aorta.^{4,5} A similar concept of ischemic preconditioning has been utilized with some success in preventing ischemic injury, but this effect has generally been assumed to be mediated by angiogenesis that takes days to weeks to manifest before surgery.^{6,7} The authors have developed a beautiful model that might be 1 further step toward a clinical benefit in these contexts; however, we know no aorta can be replaced using their model. The aorta was clamped on at 1 site, and was not opened at all.

The abyss of the valley between these experimental models and the actual clinical practice remains within the dense fog. We must keep trying.

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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