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EDITORIAL COMMENTARY

Preventing spinal cord injury during thoracic aortic surgery: Simpler than we thought?

Hiroo Takayama, MD, PhD, and Michael A. Borger, MD, PhD

See related article on pages 360-6.

Although more than 2 decades has passed since the groundbreaking clinical work by Stanley Crawford and his colleagues, spinal cord injury and subsequent paraplegia remain the most dreaded complication after thoracoabdominal aortic aneurysm (TAAA) repair. In a recent review article studying 38,491 patients pooled from multiple

studies, the overall incidence of paraplegia or paraparesis was 7.1%, with a range of 0% to 32%.¹ The incidence decreased from 9.0% before 2000 to 7.0% between 2001 and 2007, and then to 5.9% between 2008 and 2013. Although the incidence of paraplegia appears to be decreasing, this complication remains a significant problem because of its grave impact on patient quality of life. Endovascular aneurysm repair is being performed more frequently in patients with aortic disease, but even these procedures are known to be associated with spinal cord injury. We must therefore continue to find ways to decrease the occurrence of this devastating complication in patients with TAAA.

The cause of spinal cord injury during TAAA repair is multifactorial. A multitude of preventative and therapeutic approaches have therefore been developed, including distal aortic perfusion, maintenance of mean systolic arterial pressure, cerebrospinal fluid drainage, hypothermia, epidural cooling, selective spinal cord perfusion, sequential aortic crossclamping, reimplantation of intercostal arteries,

From the Division of Cardiac, Thoracic and Vascular Surgery, Columbia University Medical Center, New York, NY.

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Address for reprints: Michael A. Borger, MD, PhD, Division of Cardiac, Thoracic, and Vascular Surgery, Columbia University Medical Center, 177 Fort Washington Ave, MHB 7GN-435, New York, NY 10032 (E-mail: mb3851@cumc.columbia.edu).

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identification and preservation of the artery of Adamkiewicz, and adjunctive pharmacologic therapies (steroids, mannitol, barbiturates, etc). Many of these practices are based on anecdotal clinical experience or animal models and have not been adequately assessed in large clinical studies. Furthermore, the relatively small number of patients who need TAAA repair makes prospective, randomized trials very unlikely in the foreseeable future. Sound large animal research represents the best current method of assessing such therapies. In this regard, Kise and colleagues have performed a relatively simple but perceptive study in a canine model simulating TAAA repair, as reported in this issue of the *Journal*.

Blood supply to the spinal cord is complex. The vertebral arteries join to form the anterior spinal artery that supplies the motor areas of the cord, and 2 posterior spinal arteries that originate from the subclavian arteries supply the proprioceptive and sensory areas of the cord. Notable work by Griep's group has contributed to our understanding of the spinal cord blood supply.² Etz and associates² demonstrated the detailed anatomy of the spinal cord collateral network circulation in a pig model. Thoracic and lumbar segmental arteries contribute to both the anterior spinal artery, and an extensive paraspinal network feeds the paraspinal muscles. The anterior spinal artery is connected in multiple locations to epidural arteries, and there are extensive connections between the intraspinal and paraspinal networks. The characteristics of this complex and extensive collateral spinal cord circulation from multiple sources offer a conceptual backbone for some of the clinical practices currently used in TAAA surgery, such as maintenance of mean systolic arterial pressure and distal aortic perfusion. Intuitively, such interventions would seem likely to lead to increased spinal blood flow through the collateral network; however, until now little experimental evidence has been provided to support this notion.

Delayed paraplegia after a hypotensive episode is a particularly frustrating situation that can occur early after TAAA repair. In addition, Okita's group has demonstrated that the duration of intraoperative hypotension after separation from cardiopulmonary bypass is an independent risk factor for paraplegia in patients with TAAA.³ These investigators also showed that augmentation of systemic blood pressure during spinal cord ischemia increases spinal cord blood flow and decreases postoperative neurologic complications in a rabbit model.⁴ Other animal studies have shown that when the thoracic aorta is occluded, hypotension results in loss of motor evoked potentials—reflecting cord ischemia—and that this change can be reversed by increasing systemic blood pressure.⁵

With regard to the distal aortic perfusion, there is surprisingly little supportive evidence that this strategy protects against spinal cord ischemia. Although it is commonly

used in TAAA surgery, the independent protective effect of distal aortic perfusion has not been demonstrated in either animal or human studies.

The study by Kise and colleagues in this issue of the *Journal* therefore provides us with simple and encouraging verification of our routine clinical practices of maintaining systolic arterial pressure and distal aortic perfusion. These investigators demonstrated a linear correlation between systemic blood pressure proximal to the aortic clamp and spinal cord perfusion pressure—as well as spinal cord blood flow—during clamping of multiple segmental arteries. Distal aortic perfusion also improved spinal cord blood flow, and the correlation between proximal blood pressure and spinal cord perfusion was maintained when distal aortic perfusion was eliminated. Importantly, Kise and colleagues observed that a one-third increase in systemic blood pressure was required to maintain preclamping spinal cord blood flow, and that a two-thirds increase was required in the absence of distal aortic perfusion. Although the study design did not allow either postoperative neurologic examination or postmortem histologic analysis, the take-home message is that higher systemic blood pressures during aortic clamping and distal aortic perfusion are simple and effective maneuvers to decrease the risk of spinal cord injury during TAAA repair.

One might ask whether there are any possible downsides to either of the previously mentioned maneuvers. Norepinephrine was used to increase blood pressure in the study of Kise and colleagues, even though α -constrictors have the potential risk of inducing spinal artery vasoconstriction or spasm. Kise and colleagues did not assess segmental artery constriction in their study; however, the observed increase in spinal cord blood flow argues against this phenomenon. Another possible risk of induced hypertension is dislodgement of the aortic clamp, but this is unlikely to occur if the systolic blood pressure is kept lower than 200 mm Hg. Intracerebral hypertension or hemorrhage is another possible risk, but this also unlikely to occur at systolic pressures below 200 mm Hg. We argue that there are few potential risks of induced hypertension or distal aortic perfusion during aortic clamping and suggest that this practice should be instituted for all patients undergoing TAAA repair.

The 2 interventions assessed in the current study of Kise and colleagues have already been implemented in most aortic surgery centers worldwide, although the degree of induced hypertension may be lower than the required minimum to maintain spinal perfusion that was observed by Kise and colleagues. Although further studies are required to assess other neuroprotective interventions or pharmacologic agents during TAAA repair, Kise and colleagues are to be congratulated for drawing our attention once more to 2 simple but very important, therapeutic options. Protecting the spinal cord may be

as simple as raising the blood pressure by one-third in the presence of distal aortic perfusion and by two-thirds when distal perfusion must be stopped. Preventing spinal cord injury during TAAA surgery may be simpler than we formerly thought.

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