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Short Communication
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# Spinal Cord Injury during Thoracic Endovascular Aneurysm Repair

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## Short Communication

Spinal cord injury (SCI) is a devastating complication of thoracic aortic aneurysm repair. Thoracic endovascular aneurysm repair (TEVAR) is a less invasive treatment for thoracic aortic aneurysm repair. This short communication describes the recent knowledge of SCI during TEVAR.

Two theories have been proposed to explain spinal cord circulation: one based on anatomy (intercostal or lumbar arteries) and one based on a dynamic demand-dependent (collateral) blood supply. Understanding the anatomy of spinal cord blood supply is essential for developing strategies to prevent SCI. In 1881, Albert W. Adamkiewicz suggested that the most important input to the ASA is a single dominant branch of a segmental artery in the thoracic or upper lumbar region, now referred to as the artery of Adamkiewicz [1]. Adamkiewicz's concept became the predominant theory and rationale for their implantation of intercostal and lumbar arteries, each arising from the regional segmental arteries and, supplying the cervical, thoracic and lumbosacral regions of the spinal cord. However, Adamkiewicz's idea has proven to be controversial: opponents argue that reimplantation of the segmental arteries during thoracic aortic aneurysm repair is the best strategy for preserving spinal cord blood flow to the spinal cord. Despite attempts to avoid SCI with this approach, there continues to be a definite, irreducible incidence of SCI after extensive thoracoabdominal aortic aneurysm repair Reattachment of the intercostal and lumbar segmental arteries is not possible during TEVAR, but the incidence of SCI after TEVAR is lower than that after open surgery. It has been hypothesized that an axial network of small arteries feeds the spinal canal, perivertebral tissues and paraspinal muscles and receives input from the subclavian, internal thoracic, lumbar and hypogastric arteries [2]. These small arteries are interconnected and are also connected with the anterior and posterior spinal arteries and provide blood flow to the spinal cord. This arterial network allows for increased blood flow from one source when another source is impaired. Spinal cord blood flow is reduced if an alternative, lower resistance pathway becomes patent elsewhere in the circulation.

Independent risk factors for the development of delayed onset SCI include a perioperative mean arterial pressure of less than 70 mmHg, complications of CFS drainage, previous abdominal aortic aneurysm repair (if the hypogastric arteries have been compromised), significant preoperative renal failure, left subclavian artery coverage without revascularization, and the use of multiple stent-grafts (coverage of a long segment). However, the simultaneous closure of two independent arterial spinal cord vessels, particularly when combined with intraoperative hypotension, has been shown to be the most important risk factor for symptomatic SCI, irrespective of the covered length or previous aortic repair, reinforcing the importance of the collateral arterial network.

The risk of SCI resulting from TEVAR may be decreased by with cerebrospinal fluid (CSF) drainage [3]. In clinical studies, CSF pressure reduction has been shown to increase spinal cord perfusion pressure. However, there is still no consensus on the role of CSFD in TEVAR. Coverage of the left subclavian artery compromises the proximal collateral circulation to the vertebral and internal thoracic arteries. Coverage of long segments of the thoracic aorta using more than two stent grafts also limits spinal cord perfusion by compromising important intercostal and lumbar segmental arteries supplying the anterior spinal cord artery. Prior AAA surgery diminishes spinal cord perfusion by compromising pelvic and hypogastric collaterals. Other independent risk factors include age, number of patent lumbar arteries, emergency surgery, prolonged duration of the procedure and iliac artery injury. Postoperative hypotension and increased cerebrospinal fluid pressure are also associated with the increased risk of neurologic deficits after TEVAR. These results are predictable based on the physiological principle that spinal cord perfusion pressure is equal to the difference between the mean atrial pressure and the cerebrospinal fluid pressure. Blood pressure

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augmentation and waking the patient early from anesthesia can help us detect signs of SCI. When SCI is detected, CSF drainage and arterial blood pressure augmentation can prevent delayed SCI. Coverage of the thoracic aorta by TEVAR and the exclusion of relevant segmental arteries are associated with relatively low rates of SCI, suggesting that an exclusively anatomical explanation of SCI is inadequate. Collateral arterial networks, anesthesia stability, and the duration of ischemia are all important contributing factors. Despite advances in our understanding of spinal cord perfusion, postoperative SCI remains a serious and challenging complication of thoracic aortic aneurysm repair.

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