Spinal cord ischemia (SCI) remains the most destructive and impressive complication following aortic aneurysm repair. During the last three decades several strategies have been developed in the battle against paraplegia, especially in open surgery, leading to significant improvements. Adjunctive measures in open repair include re-implantation of intercostal and lumbar arteries, active cooling of the patient by means of extracorporeal circulation, cerebrospinal cord fluid (CSF) drainage, neuromonitoring, and blood pressure management. In endovascular repair of thoracoabdominal aortic aneurysms (eTAAA), the options to improve or regain spinal cord circulation are more limited and mainly focus on CSF drainage and maintenance of adequate mean arterial pressure.

Since eTAAA does not require aortic cross-clamping and is not associated with the massive trauma of open repair, it has been assumed that the incidence of SCI would be significantly lower than traditional surgery. Dias et al. present their experience with 72 patients undergoing eTAAA during a 6 year period, assessing short-term neurological outcome and the effect of a standardized protocol aiming for early diagnosis and treatment of SCI. They encountered a very satisfying overall 30 day mortality of 6.9%, including 29% mortality in patients with ruptured TAAA. They used anatomical criteria to grade the extent of the aneurysms according the Crawford classification, indicating that the extent of aortic coverage by the endografts was even larger than in open repair.

Obviously the most striking outcome is the overall SCI rate of 31%, with a 50% incidence of paraplegia in type II aneurysms. When comparing their initial SCI experience (39.4%) and the results after implementing the standardized protocol (23.7%), it is clear that experience, early diagnosis of SCI, and subsequent intervention can reduce this complication, however, the incidence remains very high.

What can we learn from this article? First that it describes the experience in daily practice, especially because the data come from a very well established and competent center. The authors should be acknowledged for this transparent documentation. Second, the results confirm earlier experience from the highest volume center where SCI occurred in more than 20% of patients after eTAAA, similar to open repair. As in the experience of Dias, the most important risk factor was the extent of the aortic coverage. It should be emphasized, however, that Dias et al. used anatomical criteria for the Crawford classification, whereas Greenberg et al. categorized on the basis of the extent of the aortic repair. This indicates that in the experience of Dias even more aortic segments were covered, explaining the higher SCI rate.

Based on this knowledge the question of which patients with TAAA should be treated with eTAAA or by means of open surgery arises. For both options it is evident that these complex procedures, requiring dedicated multidisciplinary teams, adequate infrastructure, specifically trained intensive care units and extensive experience should be performed in high volume centers. SCI rates after open surgery in high volume centers, mainly reported as single center experiences, range between 3% and 15% in type II TAAA. On the basis of the reported high SCI events after eTAAA, although not scientifically supported, young and “fit-for-surgery” patients should be offered open surgical repair, especially if they suffer from connective tissue diseases, whereas older patients can be selected for eTAAA. Ideally, these decisions are taken in centers where both strategies are routine and where multidisciplinary teams consider the best option for the patient.

More insight in the black box of spinal cord circulation is needed to understand the multiple mechanisms leading to spinal cord ischemia. Besides the anatomical issues such as extent of coverage there are many pathophysiological events during open and endovascular repair which contribute to spinal cord infarction, including embolization, insufficient collateral connections, hypotension, peri-arterial edema, inflammatory responses, and pre-existing comorbidities. It is imperative to understand all these mechanisms and to adjust our strategies in order to win the battle against paraplegia.
REFERENCES


A 59 year old male presented with a 35 mm thrombosed left popliteal artery aneurysm (PAA). It was treated by thrombolysis, and venous bypass via a medial approach, with double ligation proximal and distal to the aneurysm. Eight years later, he returned with another large popliteal mass. Magnetic resonance imaging revealed a 12 cm PAA, but no visible flow. The patent bypass is marked with a red arrow. During the operation, two patent popliteal artery branches were identified. This finding, similar to endotension after endovascular aneurysm repair, might have been avoided had the original operation been performed via the posterior approach.