Commentary: Surgical repair of thoracoabdominal aortic aneurysm—Still room for improvement

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Central Message

Open TAA repair is still associated with a relevant incidence of spinal cord damage. This article, which points out mechanisms for potential improvement, will be the benchmark for the coming years.

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Four decades ago, surgical repair of thoracoabdominal aortic aneurysm (TAA) was one of the biggest challenges in cardiovascular surgery.¹ Apart from in-hospital mortality, spinal cord damage (SCD) was the most dreaded complication.¹ In the meantime, much progress has been made, and the pathophysiology of SCD is now much better understood. Different approaches have been developed to protect against SCD, such as distal perfusion, staged clamping, hypothermia, and cerebrospinal fluid drainage.^{2,3} Reattachment of intercostal arteries is believed by some to be the most critical intervention,^{2,3} even though this has been discussed controversially.^{4,5} The impression has been created that—at least in very experienced hands—TAA repair has become a relatively safe procedure.

In view of this optimism, the series of Coselli and coauthors⁶ presented in this issue of the *Journal*, the largest on TAA type II repair published to date, presents a seemingly sobering view. With an operative mortality of 10% and an incidence of SCD that is close to 14%, the risk of serious or devastating complications is still substantial. Thus, conventional, open surgery can be seen in a very critical light. The cumulative incidence of complications can be interpreted as a strong message that we should emphasize and primarily use interventional treatment in treating this challenging patient cohort.

On the other hand, the conclusion is not that simple. Interestingly, 40% of the reported SCDs occurred as delayed events, and in many instances these events were related to hypotension or low cardiac output,⁶ which theoretically should be avoidable. Reimplantation of the intercostal arteries proved protective against SCD⁶; thus, better preoperative definition of the critical intercostal pair or pairs could possibly enhance this protective effect even further. Finally, the probability of SCD was increased in the presence of atherosclerosis and diabetes mellitus,⁶ that is, diseases that involve endothelial dysfunction. SCD occurred less frequently in younger patients with less atherosclerosis, consistent with previous observations of

the same group.⁷ It is thus conceivable that the risk of SCD after surgical TAA repair can be decreased further by specific measures, including patient selection.

By comparison, the current experience with endovascular treatment of TAA is very limited.^{8,9} Lower mortalities and lower SCD rates than with surgical repair have been published, albeit in very heterogeneous patient populations with few type II TAAs.^{8,9} The results of endovascular therapy will have to be compared against the current data presented here by Coselli and coauthors⁶ as the benchmark. More importantly, late results will have to be taken into consideration. Thoracic endovascular aortic repair has been associated with significantly increased late mortality,¹⁰ and the role of endoleaks in contributing to this association is as yet unclear. Interventional treatment of abdominal aneurysm has been associated with a variable but relevant incidence of late rupture or the need for aortic reintervention.¹⁰ On an anecdotal basis, treatment failure has also been observed more than casually after intervention of TAA.⁷ For endovascular treatments for TAA to show equivalent early results, they will have to prove successful in a cohort with similar patient profile.⁶

Thus, surgical repair of type II TAA currently remains the criterion standard against which new approaches have to be

compared. The current analysis of Coselli and colleagues⁶ points out that despite 4 decades of improvement in TAA surgery, there is still room for potential improvement in its results.

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