A PROPOSAL FOR A PREDICTIVE MODEL OF PARAPLEGIA PREVENTION DURING THORACIC ENDOVASCULAR AORTIC REPAIR

To the Editor:

Thoracic endovascular aortic repair (TEVAR) has been promoted as a promising alternative to open surgical graft replacement, but despite initial promising results in spinal cord ischemia prevention, a paraplegia rate still varies between 0% and 10% depending on the surgical center. During the early 1990s, Wadouh and colleagues¹ published interesting experiments on animals regarding hemodynamic alterations and steal phenomena after aortic crossclamping (T4) as a consequence of blood originating from the area of lower blood resistance accompanied by a severe decrease in intrathecal O₂ pressure (Po₂).¹ By applying a second clamp (T13), they restored Po₂ to approximately half of the initial values and reduced steal phenomenon. By performing pressure measurements, they concluded that the increase in the Po₂ of the spinal cord occurs only when the arterial pressure between T-4 and T-13 reaches a minimum pressure of 40 mm Hg. Similar results were presented by Christiansson and colleagues,² who also performed histopathologic investigations and showed that reduction of Po₂ to 50% was accompanied with neuronal ischemic changes, which were

probably reversible. This model of double aortic crossclamping could be applied in TEVAR, in which the excluded aortic segment represents excluded aneurysm sac. According to their results, we can calculate a probable Po₂ during TEVAR by intraoperatively measuring pressure in the excluded aneurysm sac. First, preoperative calculated mean arterial pressure has to correlate with the pressure during the procedure. Then, before stent deployment, by temporary interruption of aortic blood flow (5 minutes) using balloon occlusive catheters in the proximal and distal sealing zones, we can measure intrasacal pressure. Because the mean arterial pressure, average Po₂ (35-40 mm Hg),¹ and intrasacal pressure measurements are known, we can calculate Po₂ for every patient undergoing TEVAR with a simple mathematic formula (proportion). All values less than 10 mm Hg^{1,3} would be associated with complete neuron destruction and probably with immediate paraplegia, whereas values of 15 to 25 mm Hg raise our concern of possible delayed paraplegia in terms of postoperative hemodynamic instabilities. In practice, this may be the way to differentiate possible candidates for neurologic deficit development. At this stage, we can only speculate on this issue because distinction between reversible and irreversible changes in regard to exact Po₂ values is not possible at this time. Furthermore, all studies were performed on animals and in terms of healthy aorta. The question remains whether these results would have been the same if the studies were applied in humans with degenerative aneurysms that are associated with a large amount of thrombus and intercostal origin occlusions. Performing these studies during TEVAR might reveal that key missing point.

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INTERCOSTAL ARTERY REIMPLANTATION: A DOUBLE- EDGED SWORD

To the Editor:

Delayed neurologic deficit, unlike immediate paraplegia, occurs after a period of normal neurologic evaluation after a thoracoabdominal aortic repair. The incidence of postoperative neurologic deficit, as high as 16% to 25% in the era of clamp-and-go, currently ranges between 3% and 16%, depending on the use of adjuncts and the extent of the aneurysm. Estrera et al¹ found an interesting observation that although the use of adjuncts have decreased the overall paraplegia rate, paradoxically there was a higher incidence of delayed paraplegia in patients in whom they used adjuncts compared with those who had clamp-and-sew repair, suggesting that various protective techniques such as intercostal artery re-implantation, distal aortic perfusion, and pharmacologic adjuncts might be the reason.¹ However, why was delayed paraplegia a rarity in the clamp-and-sew era? It seems that the answer can be found in the work

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