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, Wadoudh 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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by Moore and Hollier.² After performing experiments they concluded that an intermediate level of spinal cord ischemia in the rabbit of 20 to 21 minutes usually would result in full return of neurologic function after restoration of blood flow to the spinal cord. They concluded that the rabbit would have been able to hop normally and would show no functional change; however, this often (71%) was followed by the onset of recurrent and progressive paraplegia within 14 to 48 hours. By using this logic, we can assume that neither surgeons of the clamp-and-sew era nor modern vascular surgeons would have been able to create a proximal anastomosis and re-implantation of the intercostal arteries in 20 minutes. The precise cause of delayed-onset paraplegia remains unresolved, but with the experiment by Moore and Hollier² in mind, it seems that delayed hyperperfusion with resultant cord edema in a closed space all may have a role in causing delayed-onset paraplegia. Since the introduction of various adjuncts in thoracoabdominal aortic surgery we have strived to maintain perfect hemodynamic conditions and blood flow through the spinal cord. However, in hypertensive patients with disturbed autoregulatory mechanisms it is very hard to keep blood pressures within optimal ranges.³ Therefore, even the slightest intraoperative changes in pressures can have a deleterious effect on the vulnerable tissue of the spinal cord. For example, sometimes during the surgery, after the proximal clamp is positioned, there is usually a slight decrease in motor-evoked potential levels to between 80% and 90%, which can hold until a few intercostals are reattached or pressure manipulation is performed, at which point these values usually reach 100% again. Greiner et al⁴ showed a higher percentage of patients with delayed paraplegia in whom motor-evoked potential changes during surgical intervention were detected. However, by having all those adjuncts prepared, a lot of surgeons would not be in a hurry to re-implant

the intercostal arteries because distal aortic pressures are optimal and motor-evoked potentials are not decreasing significantly. According to Coselli et al, intercostal re-implantation time is usually longer than 40 minutes.⁵ In a situation with suboptimal distal aortic pressure, subtle and discrete spinal cord ischemia (ischemic penumbra) with its metabolic disturbances might emerge. The spinal cord might be disturbed, but not impaired. The crucial point is the intercostal artery revascularization after a 20-minute period, which is associated with discrete or silent spinal ischemia, preparing the field for spinal cord edema. By re-implanting the intercostal arteries we are in fact setting the stage for possible delayed paraplegia. This could be compared with late revascularization of the ischemic leg. Therefore, paradoxically, reattached intercostal arteries might cause a postrevascularization compartment syndrome. The benefits of postoperative spinal fluid drainage are not in fact a consequence of a huge fluid production but instead the increase of fluid pressure inside the rigid spinal channel. Any other postoperative factors such as hypotension and other hemodynamic alterations could only worsen the situation, however, these are not by themselves reasons for impairment. On the other hand, use of adjuncts during surgery probably has no bearing on delayed paraplegia more than 48 hours after surgery because the spinal cord edema would have resolved by that time. Further attempts must be made to maintain more accurate intraoperative blood flow values, perhaps by calculating spinal collateral network pressure measurements during surgical procedures, which could help us avoid this dreadful complication.

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TWILIGHT OF THE GODS

To the Editor:

"God is dead." Nietzsche's maxim has particular reverence pertaining to the removal of the transcendental barrier between patient and doctor that British cardiac surgeons executed in the past decade. Their decision to disclose their individual mortality figures to the public has been met with enthusiasm by the British National Health Service, so much so that it plans to emulate this feat across the medical profession at large, proposing that at least 10 further specialties disclose their mortality figures by the end of 2013.¹ But this seemingly noble act may be a little premature.

Recent events in England, namely the needless closure and subsequent reopening of the congenital cardiac unit in Leeds 2 months ago, demonstrate