follow-up, are likely to have also affected our results in conjunction with operative technique. It is not known whether a pedicled technique without injection might improve patency yet further. The outcome of the randomized trial of such “no-touch” veins against the radial artery will provide fascinating data complementary to our own. However, because the occurrence of accelerated atherosclerosis in vein grafts makes the duration of angiographic follow-up critical, unless this is similar between trials, comparison of data and hence comparison of techniques might be difficult.

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doi:10.1016/j.jtcvs.2010.06.012

DELAYED PARAPLEGIA IN TRANSITION COUNTRIES: ARE WE MISSING SOMETHING?

To the Editor:

The phenomenon of delayed neurologic deficit after thoracoabdominal aortic aneurysm repair was first documented fairly recently, and it has been considered a problem of modern thoracoabdominal surgery.¹ The incidence is somewhere between 2% and 30%, depending on the report.² Estrera and colleagues³ made an interesting observation that although the uses of adjuncts have decreased the overall paraplegia rate, there was a paradoxically higher incidence of delayed paraplegia in patients in whom they used adjuncts compared with those who had clamp-and-sew repairs.

Because the majority of these patients awaken with intact neurological function of the lower extremities, malperfusion combined with diminished cerebrospinal perfusion pressure could

take all the blame. We could assume that intraoperative spinal protection and values of distal aortic perfusion (DAP) pressure were optimal and that crucial hemodynamic changes occurred afterward during postoperative recovery. But is it so? By adjusting the pump flow rate, the DAP pressure can be maintained between 60 and 70 mm Hg, and by using a combination of partial exsanguination from a left atrium and retransfusion of blood, proximal pressures are maintained at 70 to 80 mm Hg, which is the main principle of spinal cord protection based on the early works of Lascinger and Glovitzki from the mid-1980s. They concluded that maintenance of a DAP pressure of greater than 60 to 70 mm Hg will uniformly preserve spinal cord blood flow in the absence of critical intercostal exclusion based on experiments performed on 6 dogs with somatosensory evoked potentials (SSEPs).⁴ Because SSEP data reflect the conductive capabilities of the white matter in the dorsal horn, which is less sensitive to hypoxia than the α motoneuron in the gray matter of the spinal cord, could the results of another measurement of DAP pressure be different? Furthermore, in that situation the concept of a collateral network becomes crucial, as popularized by Griep and Griep.⁵ The pressure in the collateral network is about 20 mm Hg, and therefore it is very dependent on pressure and central venous pressure changes. In the presence of uncorrected steal phenomena, DAP pressure can also decrease.

Therefore, it seems logical that even the slightest changes in pressures can have a deleterious effect on the vulnerable tissue of the spinal cord. For example, sometimes during the operation, after the proximal clamp is positioned, there is the slightest decrease in motor evoked potential (MEP) levels to between 80% and 90%, which can hold until a few intercostals are reattached or pressure manipulation is performed, when these values usually reach 100% again. According to Jacobs and

associates,⁶ after initial aortic double crossclamping, MEP levels remained adequate in 77% of patients, and in 23% significantly reduced MEP levels could be corrected with an increase in DAP pressure. This raises again the question of the appropriateness of DAP pressure. This is of special importance in transition countries, where use of MEPs and SSEPs is not common practice and where the whole strategy is based on DAP pressure.

Our paraplegia percentage is about 30% (delayed, 6%), and according to Jacobs and associates' data,⁶ our initial percentage would have been 23% at the start! Because we are all trying to keep mean arterial pressure (MAP) values very close to DAP pressure values in the operating room, does it not seem prudent to determine and keep DAP pressure at exactly estimated values during the operative procedure for each patient? Does distal aortic perfusion pressure have to be standardized for all patients? The majority of patients undergoing thoracoabdominal aortic reconstruction have hypertension as a traditional risk factor (80%). Vessels' autoregulatory mechanisms are at much higher levels and disturbed. After all, we cannot expect that the patient with hypertension of greater than 160 mm Hg or an MAP of greater than 110 mm Hg needs the same DAP pressure for spinal cord perfusion as the patient with normal tension. For example, a patient with an average MAP of 90 mm Hg has a spinal collateral network pressure of 20 mm Hg, but a patient with an MAP of 110 mm Hg has a spinal collateral pressure of 26.4 mm Hg. Therefore it is not the same thing. However, because we know the normal pressures in the collateral network of a healthy man (20 mm Hg), according to a formula (Bernard index and profundopopliteal collateral index), we can calculate the necessary DAP pressure for every patient undergoing thoracoabdominal aortic reconstruction.

For example, $Km = MAP - DAP / MAP$ (with Km as the spinal collateral

network pressure), where MAP is 110 mm Hg, Km is 26.4 mm Hg (0.264 ratio), and DAP pressure is 83.6 mm Hg. We can see from the previous calculation that the necessary DAP pressure is much higher than that we planned to use. In a situation with suboptimal DAP pressure, subtle and discrete spinal cord ischemia (ischemic penumbra) with all metabolic disturbances might emerge. The spinal cord might be disturbed but not impaired. Now the crucial moment is intercostal artery revascularization associated with discrete or silent spinal ischemia, preparing the field for spinal cord edema. Safi and associates² suggested that this complication is a result of a spinal cord compartmental syndrome that might arise from unstable blood pressure, secondary increased cerebrospinal fluid pressure, or both. Therefore, paradoxically, reattached intercostals arteries might cause postrevascularization compartment. Those observations and assumptions can be investigated by using spinal damage core predictors, such as S-100 protein and C-lactate levels, in correlation with suboptimal DAP pressure and any possible changes in MEP potentials. Until that time, we are forced to fight the battle against neurological deficit with the only choice we have.

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doi:10.1016/j.jtcvs.2010.04.035

Reply to the Editor:

We appreciate the interest of Ilić and colleagues in our previous work on delayed neurological injury after thoracoabdominal aneurysm (TAAA) repair.¹ They acknowledge that paraplegia remains significant, complicating TAAA repair in as many as 30% of their cases, with 6% occurring in a delayed fashion. They suggest that without neurological monitoring, such as somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs), distal aortic perfusion (DAP) remains their primary adjunct in preventing paraplegia. As such, they suggest that by calculating the spinal collateral network pressure for each patient, the necessary DAP pressure can be determined. By maintaining this calculated distal pressure goal, optimal "spinal perfusion dynamics" can provide protection against paraplegia.

We concur that DAP is integral for protection against paraplegia during TAAA repair. However, it is not the only adjunct involved in spinal cord protection. Ultimately, spinal cord protection requires a multifaceted approach that also involves cerebrospinal fluid drainage, intercostal artery reimplantation, and moderate hypothermia. Although no single compo-

nent predominates in importance, some aspects might become more significant in certain patients depending on the conditions encountered. Thus in our opinion all of these measures (DAP, cerebrospinal fluid drainage, intercostal artery reimplantation, and moderate hypothermia) are necessary for spinal cord protection, all of which are provided by most aortic surgeons.

As suggested by Ilić and colleagues, optimizing DAP pressure has become very important. We have generally maintained a DAP pressure of between 60 and 80 mm Hg, as was the case in our previously mentioned study. Maintaining DAP pressure, however, must be balanced with maintaining adequate proximal arterial pressure. It is important not to sacrifice proximal arterial pressure for maintaining DAP pressure. Providing distal aortic pressure likely contributes to protecting against spinal cord ischemia. However, considering the concept of the collateral network for the spinal cord,² the proximal arterial pressure remains an equal, if not more important, contribution. Caution must also be considered in cases of severely atheromatous disease because retrograde DAP can initiate devastating embolization. In such cases DAP is not begun until the ostia of the primary vessels are secured. The main consideration regarding DAP that must be recognized is that this DAP is not pulsatile. Pulsatile antegrade perfusion to the spinal cord and the collateral network is important; what degree of pulsatility is unknown.

In our recent work on neuromonitor-guided TAAA repair, we used SSEPs and MEPs to guide the conduct of our repair and to aid in determining whether reimplantation of intercostal arteries was required (Estrera et al, unpublished data, 2010). We agree with Ilić and colleagues that MEPs are more sensitive than SSEPs, but both were relatively insensitive. Interesting was the fact that the maneuver that was most frequently associated with recovery of MEP and SSEP signals was intercostal artery reattachment.