FIGURE 1. Representative transverse sections of radial artery, no-touch, and conventionally harvested saphenous veins. Examples of complete sections are shown at the top left of each panel. The main panels have been used for the identification of endothelial cells with CD31 immunohistochemistry (dark brown staining). The lining endothelium of the lumen can be seen in all sections. In the radial artery (A) endothelial cells of the vasa vasorum are relatively sparse and restricted to the adventitia/media border. In the no-touch saphenous vein (B) there is a higher density of vasa vasorum that penetrate more deeply into the media. In conventionally prepared vein grafts (C) the number of vasa vasorum are reduced because of removal of and damage to the surrounding tissue at harvesting. Scale bar = 0.5 mm.

vasa vasorum are reduced in number or damaged (Figure 1).⁴ Endothelial integrity is also compromised with endoscopic harvesting, and although this method has gained popularity in recent years, it is difficult to imagine how vein removal can be achieved without considerable damage to the vein's endothelium, including that of the vasa vasorum. The vasa vasorum of veins is more pronounced than in arteries because oxygen and nutrients are supplied mainly from the lumen, and this difference is visually striking where the density in the SV is considerably higher than the RA (Figure 1 and Dreifaldt, unpublished data).

Although there are conflicting data regarding the SV or RA being the second graft of choice, we believe that preservation of normal vein structure with the Örebro no-touch technique² is essential for improved vein graft performance. The clinical and angiographic outcomes of a randomized clinical trial due to end this year comparing no-touch harvested SV with RA grafts are eagerly awaited.

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Reply to the Editor:

We thank Dreifaldt and colleagues for their interest and their comments. We are aware of their previous pub-

lished experience with atraumatic harvesting of the saphenous vein and its potential effect on graft patency.

We agree that minimization of endothelial disruption by means of high-pressure distension is important to early (and possibly later) graft function. Although their technique describes removal of the saphenous vein without inflation, harvesting in the Radial Artery Patency and Clinical Outcomes trial permitted injection of the saphenous vein with a blood/Ringers/papaverine/milrinone solution mixture while avoiding distension against a closed outflow. Furthermore, although manipulation or palpation of the vein was minimized and the vessel wall never held, the vein was removed with its adventitia intact but devoid of any surrounding fatty or fibrous tissue, unlike the broader pedicled technique referenced.

We hope that careful handling of the conduit preserves endothelial integrity of the lumen and the vasa vasorum, and this might contribute to the superiority of the patency we have reported compared with historical controls. However, other factors, such as selection of the largest non-left anterior descending coronary artery target and careful attention to secondary prevention during rigorous clinical

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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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