Letters to the Editor

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- Include no more than 500 words of text, three authors, and five references
- Type with double-spacing
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Successful reversal of delayed paraplegia after endovascular stent grafting

To the Editor:

We read with great interest the recent report by Fleck and colleagues¹ wherein they describe a successful reversal of delayed paraplegia after endovascular stent grafting of an acute type B aortic dissection.

Despite the use of various strategies for the prevention of spinal cord ischemia, paraplegia and paraparesis may occur in 5% to 16% of operations involving conventional thoracic aortic aneurysm repair. It is generally agreed that neurologic deficits are attributable to the duration of spinal cord ischemia sustained during aortic crossclamping. The use of cerebrospinal fluid (CSF) drainage perioperatively has dramatically reduced the risk of paraplegia.^{2,3}

Stent-graft implantation for the treatment of acute type B aortic dissection, as well as descending thoracic aortic aneurysms, appears to be an effective and safe therapy.^{4,5} Although this transluminal approach avoids spinal cord ischemia resulting from aortic crossclamping, there still might be a risk for spinal cord injury due to occlusion of critical intercostal arteries covered by the stent graft. Application of short endografts and avoidance of placing stent grafts between Th8 and L2 is therefore recommended to prevent spinal cord ischemia.

Delayed spinal cord dysfunction is a complex phenomenon related to postoperative hypotension, increased CSF pressure, and ischemia-reperfusion injury. It was first described by Crawford and associates⁶ in 1988 after surgical repair of thoracoabdominal aortic aneurysm.

To our knowledge we reported for the first time full reversal of delayed paraplegia after endovascular thoracic aortic repair by the use of CSF drainage.⁷ Twelve hours after implantation of 3 Dacron-covered, self-expanding nitinol stent grafts (Talent, World Manufacturing Corporation, Sun-

rise, Fla) with a total length of 31 cm, a 74-year-old man with a true aneurysm of the descending aorta exhibited left leg paralysis and right-sided paresis. Immediate intrathecal catheter placement and drainage of CSF for 48 hours, maintaining a CSF pressure of 10 mm Hg, fully reversed the neurologic deficit. The rationale for the use of CSF drainage postoperatively is to decrease spinal cord edema. After thoracoabdominal aortic aneurysm surgery or endoluminal treatment, the postischemic spinal cord is edematous, causing spinal compression with decreased spinal cord perfusion. CSF drainage resolves the edema and restores normal spinal cord perfusion.

The authors have addressed a very pertinent issue in that CSF drainage is the most potentially therapeutic factor in reversal of delayed paraplegia after thoracic aortic aneurysm repair. In conclusion, we also would recommend peri-interventional routine CSF drainage for patients undergoing endoluminal stent-graft placement of lengthy descending thoracic aortic aneurysms and/or acute type B aortic dissection.

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12/8/127787

doi:10.1067/mtc.2002.127787

Reply to the Editor:

We appreciate the comments regarding our article by Oberwalder and colleagues and thank them for the valuable adjuncts regarding cerebrospinal fluid (CSF) drainage in stent-graft implantation.

Because of the restricted number of references in brief communications, we considered only publications in the high-impact journals. Nevertheless, we are aware of the report of Tiesenhausen and associates,¹ who described for the first time the impact of CSF drainage in a case of paraplegia after stent-graft implantation.

To close the entry tear in the descending aorta it is sometimes necessary to deploy a stent graft in the region (Th 8- L2) most prone to ischemia, but we agree that this should be avoided whenever possible. However, the risk of paraplegia has to be balanced against the risk of rupture of the aneurysm in these patients.

Routine use of CSF drainage in stent-graft implantations could help to reduce the risk of paraplegia in such cases.

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Reference

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12/8/127793

Alpha-stat strategy: Cause of ischemia in brains with old cerebral infarction despite selective cerebral hypothermic antegrade perfusion

To the Editor:

Washiyama and associates¹ are to be congratulated for experimentally verifying the anticipated susceptibility of brains with old infarction to become ischemic even during hypothermic selective continuous antegrade perfusion (SCP).

We do not doubt that SCP is better than no perfusion or retrograde perfusion, regardless of the pH management. However, despite SCP, ischemia developed in brains with old infarcts. Had the presented data been fully analyzed and interpreted without prejudice, the mechanisms of that ischemia would have been clear.

In our opinion, the alkalosis induced by alpha-stat hypothermic perfusion² explains every fact that the authors could not satisfactorily explain:

1. Although the authors failed to recognize and statistically analyze the increasing brain lactate efflux even in the control group from sample 1 to sample 4 during SCP, such increase seems to us significant. In our opinion, alpha-stat-induced alkalosis inhibits the creatine kinase catalyzed phosphorylation reactions.³ This causes failure to aerobically synthesize high-energy~P bonds and switch to anaerobic metabolism with

consequent adenosine triphosphate consumption and lactate production even in the control group without prior infarct, despite the continuous perfusion.

2. Further lactate efflux increase in the infarct group after rewarming. Failure to use glucose aerobically becomes overt after rewarming following alpha-stat hypothermic uninterrupted perfusion at 20°C for 60 minutes.⁴

As pointed out by the authors, the penumbra area of an infarct is dependent on collateral flow. The vasoconstriction and decreased brain flow caused by alpha-stat strategies will certainly reduce such collateral flow, maximizing the metabolic effects of pH management. Sakamoto and associates⁵ demonstrated brain anoxia development during early alpha-stat cooling before arrest. These effects are not limited to the period of cold perfusion but continue also during alpha-stat rewarming; thus, the lactate efflux at 32°C is maximal, even if circulatory arrest was not induced.

Obtaining the glutamate samples from the maxillary vein as often as the lactate samples would have answered whether the switch of aerobic to anaerobic metabolism and lactate efflux of the control group was enough to cause hypoxic excitotoxicity.⁶

pH-stat strategies increase brain blood flow and clearly should have been advantageous for perfusion of brains with old infarcts⁷; pH-stat hypothermic selective antegrade perfusion might prevent or minimize the metabolic effects of alpha-stat strategies even in brains with collateral flow-dependent penumbra areas.

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