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EDITORIAL

Is hypothermia a reliable adjunct for spinal cord protection in descending and thoracoabdominal aortic repair with regional or systemic cooling?

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Since DeBakey first carried out thoracoabdominal aortic repair with an aortic homograft using a temporary arterial shunt in 1956,[1] multidisciplinary approaches have been directed toward minimizing spinal cord ischemic injury during surgery of the descending thoracic and thoracoabdominal aorta. The principal modality has been distal aortic perfusion during the aortic cross-clamping, which was passive in the past with a temporary shunt tube or bypass and that currently has been active with a left heart bypass or a cardiopulmonary bypass (CPB). Other approaches include the following: intraoperative monitoring of spinal cord ischemia with somatosensory evoked potentials and transcranial motor evoked potentials; reattaching the responsible intercostal artery; increasing collateral flow by controlling the back-bleeding of the patent intercostal arteries with high cardiac output and arterial pressures with cerebrospinal fluid (CSF) drainage; increasing ischemic tolerance with pharmacological adjuncts including naloxane, steroid, barbiturates, and with hypothermia; reducing excitotocixity from neuronal ischemia with hypothermia, naloxane, and steroid; and attenuating reperfusion injury with steroid, hypothermia, and free radical scavengers. Particularly, in Japan, with recent great advances in diagnostic imaging modalities, preoperative demonstration of the arteria radicularis magna (Adamkiewicz artery) by magnetic resonance imaging (MRI) or computed tomography (CT) scans has been highlighted as a reliable guide for reattachment or preservation of the responsible intercostal arteries including the collaterals.

Since the 1950s hypothermia has been proven effective for protecting organs, including the central nervous system, in the cardiovascular surgical field.[2–5] When focusing on milestones of aortic surgery, systemic profound hypothermia at 12°–15°C was applied to aortic arch surgery for brain protection in 1975.[6] Thereafter, during the 1980s, based on systemic hypothermia, more sophisticated antegrade or retrograde cerebral perfusion has been established for more definitive cerebral safety.[7,8] For aortic surgery through a left thoracotomy, Kouchoukos et al. employed systemic deep hypothermia to protect the spinal cord as well as the brain during descending thoracic and thoracoabdominal aortic repair in 1995.[9] In most, an open aortic anastomotic technique was used with circulatory arrest, avoiding aortic cross-clamping. On the other hand, mild hypothermia around 32°C with distal perfusion of a partial CPB has been our routine, as in most of Japan.[10] During the surgery, transcranial motor evoked potentials are used to monitor spinal cord ischemia continuously. A couple of the intercostal arteries responsible for the spinal cord ischemia are aggressively reattached according to the preoperative demonstration of the Adamkiewicz artery by MRI and CT, while controlling back-bleeding from the other intercostal arteries without delay. In selected "high-risk" patients having an extent I and II thoracoabdominal aortic aneurysm, however, our tactics have lately been shifted to deep hypothermic surgery (around 20°C) with total CPB for more rigorous spinal safety. Even in this setting, motor evoked potentials can be recorded above

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25°C with adjusted anesthetics. Early rewarming after the proximal anastomosis has been another refinement for minimizing some of the adverse effects of systemic hypothermia by reducing the time of ventricular fibrillation and CPB.

As described above, systemic hypothermia has some drawbacks, such as coagulopathy, pulmonary dysfunction, cardiac arrhythmia leading to cardiac arrest or ventricular fibrillation, systemic edema due to fluid shift, in part due to prolonged CPB. To eliminate these shortcomings, the usefulness of regional cooling of the spinal cord was addressed in surgery for the descending thoracic and thoracoabdominal aorta. In 1975, Hansebout et al. first highlighted the effect of local hypothermia and steroid on recovery from experimental spinal cord compression injury.[11] Between 1992 and 1994, several impressive reports were published on regional cooling of the spinal cord in animal models.[12–14] In our country, Tabayashi and colleagues also addressed the impact of epidural cooling on the spinal cord protection in an animal experiment.[15] The rationale for this technique is to increase the ischemic tolerance of the spinal cord during the critical periods of aortic cross-clamping associated with the reduction of the spinal cord perfusion pressure. Around the same time, Davison, Cambria, and colleagues (Massachusetts General Hospital, or MGH, group) first adopted this technique clinically in eight patients undergoing descending thoracic and thoracoabdominal aortic resection.[16] With their method, at least 30 min (average 50 min) before the aortic cross-clamping, 4°C saline solution was infused into the epidural catheter until the CSF temperature decreased to 25°C. Another subarachnoidal catheter was also used to measure CSF pressure and, if necessary, to drain the CSF. They have continued ongoing efforts for the development of this technique. In the latest report of 240 patients requiring extent I to III thoracoabdominal aneurysmal repairs, the incidence of spinal cord injury of any severity was 12.1%, including the results of emergency cases.[17]

In this volume, Tabayashi and colleagues (Tohoku University group) reported their 10-year experience of epidural perfusion cooling with outstanding outcomes—spinal cord injury in 3.9% and hospital deaths in 5.9%—for 102 patients undergoing descending thoracic and thoracoabdominal aortic repair. This regional cooling has been applied for the spinal cord only by them in Japan,[18,19] as the MGH group has advocated over the last decades.[20,21] Between them, the techniques of regional epidural cooling are similar in conjunction with the CSF drainage and measurement of CSF temperature. The outcome of the Tohoku University group was more favorable than that of the MGH group. One reason might be employment of distal perfusion with CPB or left heart bypass with mild systemic hypothermia at 31°–32°C by the Tohoku University group, which differed from the fundamental "clamp-and-saw" technique with normothermia without any assisted circulation by the MGH group. Meticulous surgical techniques of antegrade or retrograde segmental sequential repair for reduced spinal cord ischemia by the Tohoku group might be another factor for more favorable spinal safety, in conjunction with aggressive reattachment of the responsible intercostal arteries based on preoperative CT or MRI findings. Although the statistical analysis did not reveal any relevant factors for spinal cord injury, they noted that the main cause of spinal cord injury was likely due to unstable hemodynamics caused by massive bleeding. However, the causes of spinal cord injury are supposed to be multifactorial. Even with efficient epidural cooling, the spinal tolerance should be limited owing to relatively lower perfusion pressure of the spinal cord. Rushed reattachment of the responsible intercostal artery has technical difficulties. Given real-time monitoring of spinal cord ischemia, such as with transcranial motor evoked potentials, it would have been feasible to clarify the cause of the paraplegia.[10]

Some drawbacks of this novel epidural cooling have been recognized. One is a sharp rise in CSF pressure coincident with epidural cooling in some instances, which might be a risk factor for spinal cord ischemia.[22,23] The MHG group also warned about this increase in CSF pressure. The two groups tolerated this "modest and transient" increase because it can be controlled well with further CSF drainage or epidural cooling fluid drainage and because the risk of spinal cord ischemia is thought to be outweighed by the potential neuroprotective effect of epidural cooling.[20,21] From this point of view, it is important to maintain an arbitrary 30–40 mmHg gradient between the mean arterial pressure and the CSF pressure before aortic cross-clamping, with some caution. Otherwise, given a newly designed cooling catheter system, for example, with a closed countercurrent lumen, it is theoretically feasible to avoid this CSF pressure elevation.[23] On a related issue, one patient suffered an unexplained cerebrovascular accident associated with the highest CSF pressure. Interestingly, the MGH group experienced a similar case and recognized the difficulty of excluding the possibility of the increased CSF pressure contributing to the highly unusual but devastating lower cervical or upper thoracic spinal cord injury. Another technical question is about the temperature measurement of the spinal cord. The both groups measured the temperature of CSF—not that of the spinal cord—based on the correlation of the temperature between the spinal cord and the CSF. The practical con-

sideration of the degree of hypothermia required for spinal safety has been answered though a variety of animal experiments, which suggested 25°–27°C for uniform protection.[12–14] However, homogeneous cooling of the spinal cord might be uncertain. In this context, the local cooling times are all different and prolonged to an average of 50 min as reported by the MGH group, which is a defect of this method.[17]

Hypothermia is still the principle for organ protection, particularly for cerebral and spinal safety. Regional perfusion cooling into the epidural space is a unique and reliable method for spinal protection, minimizing the adverse effects of systemic hypothermia. Tabayashi and colleagues are to be congratulated for their outstanding outcomes and ongoing efforts in the development of epidural cooling. However, the unsolved complexity of this novel technique for spinal safety, including some drawbacks, seems to keep it from being in routine use.[23]

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