Changes in Cerebrospinal Fluid Pressure and Lactate Concentrations during Thoracoabdominal Aortic Aneurysm Surgery

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Background: Although ischemic injury to the spinal cord is a well-known complication of aortic surgery, no metabolic markers have been identified as predictors of an adverse outcome. This study evaluated the effect of cerebrospinal fluid (CSF) drainage, with and without distal femoral perfusion or moderate hypothermia on blood and CSF lactate concentrations and CSF pressure during thoracoabdominal aortic aneurysm surgery.

Methods: Three nonconcurrent groups of patients were studied prospectively: patients with normal body temperature (35°C) but without distal femoral bypass (n = 6), patients with normal body temperature with bypass (n = 7), and patients with hypothermia (30°C) and bypass (n = 8). In all patients, CSF pressure was recorded before, during, and after aortic cross-clamping. During the surgical repair, CSF drainage was performed using a 4-Fr intrathecal silicone catheter. Blood and CSF lactate concentrations were measured throughout the operation.

Results: Significant increases in blood (49%) and CSF (17.5%) lactate concentrations were observed during and after thoracic aortic occlusion in patients with normothermia and no bypass (P < 0.02 and 0.05, respectively). Distal perfusion attenuated the increase in both blood and CSF lactate (P < 0.01), and a further reduction was achieved with hypothermia of 30°C (P < 0.001). Patients who became paraplegic showed a greater increase in CSF lactate concentrations after aortic clamp release compared with those who suffered no neurologic damage (275% vs. 123% of baseline; P < 0.05). Increased CSF pressure of 42–60% (P < 0.005) was noted soon after thoracic aortic occlusion, both with and without distal femoral bypass.

Conclusions: Incremental reductions in CSF lactate concentrations were achieved using distal femoral bypass and hypothermia. The reduction in CSF lactate correlated with the methods used to protect the spinal cord during thoracoabdominal aortic aneurysm surgery and was associated with better outcome. Decompression by distal bypass of the hemodynamic overload caused by aortic occlusion was insufficient to eliminate the acute increase in CSF pressure. Cerebrospinal fluid lactate measurements during high aortic surgery may accurately represent the spinal cord metabolic balance. (Key words: Bypass, centrifugal pump: left atrial-femoral artery; left ventricle-femoral artery. Cerebrospinal fluid pressure. Complications: paralysis; spinal cord injury. Lactate: blood; cerebrospinal fluid. Surgery: thoracoabdominal aortic aneurysm. Techniques: spinal; cerebrospinal fluid drainage. Temperature: hypothermia.)

SEVERE ischemic injury to the spinal cord during surgical repair of thoracoabdominal aortic aneurysm is a frequent complication after thoracic aorta cross-clamping (AXC). Important factors that exacerbate spinal cord ischemia are the unique anatomy of the anterior spinal artery and the sudden increase in cerebrospinal fluid (CSF) pressure after AXC. The lower thoracic segment of the anterior spinal artery is by nature narrow and has little collateral blood supply. Thus the acute decrease in blood flow after AXC and increased resistance to flow due to increased CSF pressure are important factors that contribute to reduced spinal perfusion pressure and the development of spinal cord ischemia during high aortic occlusion.

Recent accumulated experience emphasizes that using one method of spinal cord protection does not protect against spinal cord injury and that it may be helpful to combine various methods. Common methods of spinal cord protection are distal aortic perfusion or bypass, hypothermia, and CSF drainage. In an animal model and in humans, cooling of the spinal cord helps pro-
tect against the motor and sensory dysfunction evoked by ischemia. Distal aortic perfusion does not reliably eliminate ischemic injury, despite definite improvement in spinal cord blood flow, probably because of the preferential flow away from the high-resistance lower thoracic segment of the spinal artery.\textsuperscript{9} Reduction of CSF pressure at the time of AXC by draining CSF improved the spinal perfusion pressure during the vulnerable period in animal studies.\textsuperscript{7,9} However, the efficacy of CSF drainage when used as the sole measure of spinal cord protection is still questionable.\textsuperscript{9,10} Increased CSF lactate concentrations are an established marker for severe brain trauma in animals.\textsuperscript{11} A concomitant increase in blood lactate, which was attributed to the systemic response to trauma, was also observed.\textsuperscript{12} Despite these findings in animals, there are no clinical data regarding generation of lactate from the spinal cord during aortic surgery, or using this parameter to assess the effectiveness of various methods of spinal cord protection during aortic surgery. In addition, the change in CSF pressure, at the spinal cord level in patients during aortic clamping and distal aortic perfusion, has not been determined.

This study evaluated the effect of distal femoral bypass and moderate hypothermia on CSF lactate concentrations during thoracoabdominal aortic aneurysm surgery. In addition, the CSF pressure response to application of AXC was examined in patients operated on with and without distal femoral bypass.

**Materials and Methods**

**Patients**

The study was approved by the institutional committee on clinical investigation, and written informed consent was obtained from each patient. Three groups of patients undergoing thoracic or thoracoabdominal aortic aneurysm repair were examined: those with normothermia without distal femoral bypass (n = 6), those with normothermia with distal femoral bypass (n = 7), and those with hypothermia with distal femoral bypass (n = 8). No active cooling was used in the first two groups, whereas in the third group, active cooling to 30°C was performed before AXC. The decision to use distal bypass or the cooling management was unrelated to the patient’s medical condition and was not random. Instead it reflected a change in surgical practice during the study period.

**Operative Procedure**

The left-sided bypass was performed with a centrifugal pump (Bio-Medicus, Eden Prairie, MN), and anticoagulation (70 U/kg heparin) was initiated to achieve an activated clotting time value of 200 s. The inflow to the pump was from a canulla inserted into either the aorta, left atrium, or the tip of the left ventricle, and blood was returned to the patient through the left femoral artery. For cooling and rewarming, the blood was passed through a heat exchanger (Avecor, Plymouth, MN). Cooling the patient to a nasopharyngeal temperature of 30°C was performed slowly, before applying the AXC, to avoid development of cardiac dysrhythmias. In addition, patients were rewarmed slowly at the end of the aortic repair.

Selective spinal angiography was performed before operation to identify the point of origin, patency, and degree of blood supply to the cord through a “critical” intercostal or lumbar artery. A finding of such patent artery, arising from the aneurysmal wall, signifies a markedly increased risk for postoperative paralysis,\textsuperscript{13} and every effort was made to reimplant the artery during the aortic repair. Usually two pairs of significant intercostal arteries, at the relevant thoracic segment, were sutured into an appropriately sized ellipse cut from the graft.\textsuperscript{3} Importantly the aneurysms were repaired segment by segment from proximal to distal ends, thereby maintaining perfusion to the spinal cord for all but the time required for direct anastomosis.

**Anesthetic Protocol and Monitoring**

Before they arrived in the operating room, all patients received standard intramuscular premedication, which consisted of 0.15 mg/kg morphine, 0.1 mg/kg diazepam, and 0.005 mg/kg scopolamine.

Continuous blood pressure was monitored using radial and femoral arterial catheters. Central venous and pulmonary artery pressures were also monitored. In all patients, a double-lumen endobronchial tube was inserted with fiberoptic guidance, and ventilation was monitored continuously with oxygen and carbon dioxide analyzers and a pulse oximeter. Fentanyl (25–30 µg/kg), midazolam, and pancuronium bromide were used to induce anesthesia. Isoflurane or enflurane were administered as required in concentrations of 0.5–0.75 minimum alveolar concentration to minimize their depressant effect on signal quality of the evoked potentials.\textsuperscript{14} Somatosensory and epidural-evoked potentials or motor-evoked potentials were used to monitor spinal cord activity. Previously we described the yield of the
CSF LACTATE AND PRESSURE IN TAAA SURGERY

Technique and method of insertion. All cardiovascular parameters were recorded continuously on a nine-channel recorder (model 7758A; Hewlett-Packard, Waltham, MA).

Mean distal femoral pressure was maintained at 80–90 mmHg while the AXC was on, by adjusting bypass flow rate (approximately 2 l/min), fluid administration, and vasoactive drugs if necessary. Hemodynamic parameters such as central venous pressure (CVP) and pulmonary capillary wedge pressure were measured continuously but usually did not serve as a guide for pump management during the AXC period. In patients in whom distal bypass was not used, control of systemic blood pressure and myocardial overload was achieved by administering anesthetics and nitrovasodilators for a limited interval. Metabolic acidosis was reversed by administering sodium bicarbonate at the discretion of the anesthesiology team.

Cerebrospinal Fluid Drainage and Monitoring

Cerebrospinal fluid drainage and CSF pressure measurements were performed with a 4-Fr intrathecal silicone catheter (Codman & Shurtleff, Randolph, MA), which was introduced at L2–3 or L3–4 intervertebral space via a 14-gauge Tuohy needle. Blood and CSF samples were taken at predetermined times and analyzed for lactate concentrations using a glucose/lactate analyzer (YSI 2300 STAT; Yellow Springs Instruments, Yellow Springs, OH). These samples were taken at baseline (after anesthesia induction), before AXC, 10 min after AXC, 30 min after AXC or immediately before proximal AXC release (whichever came first), immediately after proximal aortic declamping, and when all cross clamps were removed from the aorta. Cerebrospinal fluid pressure was monitored continually, but during the period of AXC, CSF was aspirated to compensate for the increase in CSF pressure and then allowed to drain freely into an external drainage system that was placed 8 cm above the spinal cord level. During this period, CSF pressure was measured intermittently every 3 to 5 min and was always maintained lower than the mean femoral arterial pressure.

Analysis

Statistical analysis of time-related changes was done by analysis of variance for repeated measures. The mean lactate value from all measurements taken during aortic repair and after AXC release were compared with baseline and among groups. Statistical differences among groups at the same time interval were compared using the unpaired Student's t test, and a probability value less than 0.05 was considered significant. All data are given as mean ± SD.

Results

Table 1 shows demographic data. All patients were studied angiographically for localization of the great radicular artery arising from a patent intercostal artery. The "critical" artery was identified in 10 of the 21 patients (48%), arising between T8 and T12. The change in operative practice during the study by using distal aortic perfusion and by reducing patients' body temperatures before the aortic repair are efforts made by surgical and anesthesia teams to improve surgical outcome. Patients in all groups had similar demographic characteristics of age, sex, and concomitant diseases. The mean core body temperature as measured by the pulmonary artery catheter was 35.1 ± 0.7°C in the patients with normothermia without bypass, 35.2 ± 0.3°C in those with normothermia and bypass, and 30.2 ± 0.2°C in the patients with hypothermia and bypass. In the latter group, the nasopharyngeal temperature was very

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Distal Perfusion</th>
<th>Temperature (°C) at AXC</th>
<th>Spinal Artery</th>
<th>Outcome</th>
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<tr>
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</tr>
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</tr>
<tr>
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<td>None</td>
<td>34.3</td>
<td>T9</td>
<td>Died in OR</td>
</tr>
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<td>ND</td>
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<td>71</td>
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<td>T9</td>
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<td>ND</td>
<td>Uncomplicated</td>
</tr>
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AXC = aortic cross clamp; ND = not demonstrated; OR = operating room; FA = femoral artery; FV = femoral vein; LV = left ventricle; AO = aorta; LA = left atrium.
Fig. 1. Concentration (mean ± SD) of lactate in the blood (circle) and in cerebrospinal fluid (CSF; triangle) in three groups of patients: those with normal body temperature but without distal femoral bypass (solid line; n = 6); those with normal body temperature and distal femoral bypass (dotted line; n = 7); and those with hypothermia and distal femoral bypass (dashed line; n = 8). The increase in blood and CSF lactate with release of the proximal aortic cross clamp (AXC off) and, later, release of the distal clamp (all AXC off) was significant compared with baseline (BL) in all groups (P < 0.001 to 0.05). The difference in blood and CSF lactate among groups was also significant: *P < 0.01 between blood lactate concentrations and CSF lactate in the two normothermic groups; **P < 0.001 between the CSF lactate concentrations in the hypothermic group and CSF lactate in the two normothermic groups (probability value determined by analysis of variance).

similar to blood temperature. Atrial fibrillation developed in one patient when his core temperature reached 30.5°C, and rewarming to 31°C spontaneously caused return of the sinus rhythm. The mean AXC period in patients operated on without femoral bypass was 45 ± 16 min. When distal femoral bypass was used, management of AXC was based on the sequential clamping approach. The proximal AXC was released and moved to the graft after 20 ± 8 min. The distal AXC was repositioned further distally after 48 ± 28 min.

Mean concentration of lactate in the CSF at baseline (Fig. 1) in all patients was significantly higher than in the blood (1.4 ± 0.4 mM/L vs. 1.1 ± 0.4 mM/L, respectively; P < 0.002). The increase in blood and CSF lactate concentrations at 30 min of aortic repair and immediately after aortic declamping was significant in all three groups compared with baseline values (P < 0.001 to P < 0.05).

In the patients with normothermia without bypass (Fig. 1), the mean concentration of blood lactate increased by 490%, from a baseline value of 1.1 ± 0.3 mM/L to 6.5 ± 3.3 mM/L (P < 0.02) during the surgical procedure. Mean CSF lactate in this group increased from 1.5 ± 0.2 mM/L to 4.1 ± 2.2 mM/L (173%; P < 0.05) during the same time intervals. Femoral bypass improved distal perfusion and attenuated the lactate increase in the patients operated on at normal body temperature, both in the blood and CSF, by 245% and 135%, respectively (P < 0.01 vs. baseline). We observed the smallest lactate increase with systemic hypothermia of 30°C. In this group, blood lactate increased from 1.1 ± 0.4 mM/L to 2.2 ± 0.6 mM/L (100%), whereas CSF lactate only increased from 1.4 ± 0.3 mM/L to 1.9 ± 0.3 mM/L (36%).

In comparisons among the groups, use of distal bypass significantly attenuated the increase in CSF lactate in the patients with normothermia (P < 0.01), and hypothermia reduced even further lactate accumulation in the CSF (P < 0.001).

Of the patients with normothermia, four patients had complicated courses. One died during operation of uncontrolled bleeding. Three patients were diagnosed as having dense paraplegia after operation; two of them died within the first 36 h. In the patients having hypothermic bypass, all patients had uncomplicated recovery. In the three patients with normothermia who became paraplegic (Fig. 2), both blood and CSF lactate increased to more than 10 mM/L.
increased dramatically (by 841 ± 350% and 275 ± 31%, respectively); however, the increase in CSF lactate became evident only with spinal cord reperfusion after aortic declamping \((P < 0.05\) compared with patients with normal outcome and compared with baseline lactate values).

We observed an acute increase in CSF pressure in all patients immediately after applying AXC (fig. 3), which was similar in patients with and without distal bypass. Cerebrospinal fluid pressure increased in those operated on without bypass from 15 ± 4 mmHg to 22 ± 6 mmHg (42%; \(P < 0.005\)), and in those with bypass from 13 ± 4 mmHg to 21 ± 3 mmHg (60%; \(P < 0.001\)). Central venous pressure values did not increase after we applied AXC, but we noted a change from 12.6 ± 6.2 mmHg before AXC to 13.5 ± 6.1 mmHg after AXC in those operated on without distal bypass; similarly, we found no change in CVP values when bypass was used.

We observed a second peak in CSF pressure soon after aortic declamping and it was associated with hypercarbia and metabolic acidosis, which developed on reperfusion.

**Discussion**

A continuous attempt is being made to define criteria useful for predicting spinal cord ischemia during thoracoabdominal aortic aneurysm surgery. Intraoperative identification of neural tissue at risk will promote aggressive surgical and anesthetic interventions to prevent the development of nonreversible damage to the spinal cord. Because of the effort of the team to reduce the rate of severe neurological sequelae, use of distal bypass and later of hypothermia in our study was a natural development of the surgical technique, which prevented us from performing a randomized study. Increased lactate production is a well-established biochemical characteristic of compromised brain tissue. Nevertheless, we are unaware of clinical studies that report an increase of CSF lactate in spinal cord trauma or secondary to ischemic injury after high thoracic aortic surgery. In this study, we observed a definite increase in CSF lactate in patients having surgery at normal body temperature. The increase in lactate concentrations occurred during occlusion of the thoracic aorta and persisted throughout the operation. The magnitude of this increase depended on the methods used to protect the spinal cord. Thus both distal femoral bypass and hypothermia have additive effects on attenuation of lactate concentrations in the CSF. In the three paraplegic patients, the increase in CSF lactate occurred only after release of AXC. It is possible that such late appearance of lactate in the CSF is related to postischemic reperfusion of a spinal cord that is particularly affected by an insufficient collateral blood supply.

Several studies\(^{12,16,17}\) showed that in experimental brain or spinal cord injury, the increase in CSF lactate persisted for several hours after the insult. Sustained high CSF lactate concentrations may indicate the continuing production of lactate from injured neural tissue and ongoing tissue acidosis.\(^{17}\) Although the integrity of the blood–brain barrier usually prevents penetration of blood lactate into the CSF,\(^{18-20}\) we cannot rule out the possibility that lactate was absorbed from the systemic circulation into the CSF; that is, the integrity of the blood–brain barrier was impaired by the ischemic injury.

Our consistent observations that the initial CSF lactate concentrations, before application of AXC, were always greater than those in the blood might be attributed to the release of lactate into a relatively closed fluid reservoir of CSF, and, possibly, to its reduced clearance through the intact blood–brain barrier. The substantial production of lactate in patients operated on while their body temperatures were normal and without distal bypass suggests that using CSF drainage alone is not suffi-

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**Fig. 3.** Cerebrospinal fluid (CSF) pressure (mean ± SD) in patients operated on without the assistance of distal bypass (circle; \(n = 6\)) and in those in whom distal femoral bypass was applied (triangle; \(n = 15\)). The increase in CSF pressure observed 2 min after application of aortic cross-clamping (AXC) was significantly greater compared with pre-AXC measurements in both groups (\(* P < 0.001; \# P < 0.005\)). An increase in CSF pressure was also observed soon after release of the AXC.

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cient to maintain an adequate metabolic balance of the spinal cord.

We propose that CSF lactate concentrations have general diagnostic value in evaluating various methods to protect the spinal, particularly when compared with blood lactate concentrations drawn simultaneously. Thus the stepwise decrease in blood and CSF lactate concentrations with the different methods used in the present study suggests that the multimodal approach to protect the spinal cord during high aortic surgery might be better than using a single method. The utility, on the other hand, of intraoperative CSF lactate concentrations as an indirect predictor of imminent spinal ischemia is still questionable.

After clamping the thoracic aorta, we observed a substantial increase in CSF pressure that was not accompanied by a parallel increase in CVP. The unchanged CVP values are related to intentional control of the hemodynamic status by medications or by the centrifugal pump flow. The finding of CSF pressure increase is consistent with several previous reports that also showed that increasing CSF pressure substantially reduces spinal cord blood flow. However, we have also observed an equal increase in CSF pressure in those patients in whom femoral bypass was initiated before aortic clamping. This finding has not been described before and shows that despite preload and afterload reduction of the heart and systemic circulation in the upper part of the body, the mechanisms governing the increase in CSF pressure after AXC are not eliminated.

Several theories in the literature have tried to explain the increase in CSF pressure and indicate that multiple factors act simultaneously. Important determinants in the regulation of CSF pressure after AXC are the increase in cardiac preload (an increase in CVP), in arterial blood pressure, and in carbon dioxide levels. Stokland and colleagues have shown in animals that the preload is increased secondary to translocation of blood from the lower body after applying the AXC. If the inferior vena cava is simultaneously clamped, there is no increase in CSF pressure. Distal aortic perfusion is supposed to prevent venous overload after aortic clamping; nevertheless, we have observed a significant increase in CSF pressure. One possible explanation for the increase in CSF pressure is that distal aortic perfusion is performed with a centrifugal pump, which does not use an oxygenator to drain a large amount of blood from the circulation and thus is less effective in preventing an increase in intraspinal venous engorgement compared with arteriovenous bypass.

Because other factors such as aortic blood pressure and carbon dioxide levels remained constant, it seems that application of AXC activated an additional mechanism to those previously mentioned. Spinal venous outflow through the radiculospinal veins has a vascular waterfall characteristic that creates a physiologic valve that acts as an antireflux mechanism. Thus a possible explanation for the increase in CSF pressure is that AXC induces a sympathetically mediated vasoconstriction in the systemic and spinal cord vasculature. This increased tone in the thin-walled spinal veins may decrease the critical closing pressure needed to provoke collapse of the radiculospinal veins as they pass through the dura, with consequent venous engorgement and an increase in CSF pressure.

Control of venous return to the heart, either by mechanical or pharmacologic means, would eliminate the direct effect of increased CVP on spinal outflow pressure. Nevertheless, the acute increase in CSF pressure, which was still evident, probably justifies CSF drainage. Decompression of the spinal canal is particularly important, because blood flow from the distal aortic perfusion is directed more to the spinal cord below the entry of the arteries radiculares magna than above it. Thus improving spinal perfusion pressure will increase the tolerated ischemic time for the cord during aortic surgery. Reduction in the spinal cord metabolic rate by cooling the patient to 30°C will reduce oxygen consumption by nearly 50% and therefore will help preserve a favorable energy state. Hypothermia may also provide a protective effect on the cord by inhibiting neuronal release of excitatory amino acids such as glutamate. The limited duration of controlled moderate hypothermia, followed by active rewarming of the patient back to normal temperature with the bypass machine, usually minimizes the incidence of hypothermia-related complications.

In conclusion, we showed that CSF lactate concentrations are increased in patients in whom spinal cord injuries develop during thoracoabdominal aortic aneurysm repair. Moderate systemic hypothermia induced by distal aortic perfusion provides greater reduction in lactate accumulation than does normothermic bypass alone.

Increased CSF pressure after AXC, followed by a continuous increase in CSF lactate concentrations despite
aggressive CSF drainage, suggests the need for a prospective randomized clinical study assessing the effects of multiple modalities on spinal cord protection during high-risk aortic surgery.

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