Repair of descending thoracic and thoracoabdominal aortic aneurysms (TAAAs) is associated with a substantial risk of perioperative spinal cord ischemia that may or may not lead to permanent postoperative paralysis. Several techniques that aim to increase the ischemia tolerance time of the spinal cord during the period of aortic cross-clamping have been described in the literature. Hypothermia, left heart bypass, various shunts, myriad drugs, and cerebrospinal fluid (CSF) drainage have all been used alone or in combination to potentially improve neurologic outcome after surgery. CSF drainage and distal aortic perfusion have been shown to lower the incidence of neurologic complications after repair of type I and type II TAAAs. Distal aortic perfusion increases distal aortic pressure, and CSF drainage decreases CSF pressure. These techniques potentially lead to an augmentation of spinal cord perfusion pressure during the period of aortic cross-clamping. However, CSF drainage can be associated with potentially serious complications, including fracture of the catheter during removal, catheter-associated meningitis, and/or temporary abducens nerve palsy, among others. Also, epidural hematoma at the catheter insertion site can complicate the clinical assessment of postoperative spinal ischemic injury. Intracranial subdural hematomas, with excessive CSF drainage, have also been reported and are associated with significant morbidity and mortality. Subsequently, some authors have challenged the efficacy of CSF drainage and question the acceptable risk: benefit ratio of the technique. A patient who developed intracerebellar bleeding associated with CSF drainage after TAAA repair is presented. Additionally, the authors reviewed a series of 91 consecutive cases of descending thoracic and TAAA repairs in an effort to identify any potential association between neurologic complications and CSF drainage.

CASE REPORT*

A 42-year-old man with Marfan’s syndrome developed type B aortic dissection at age 37. The dissection extended from the left subclavian artery to the level of the aortic bifurcation and was treated initially with beta-adrenergic blockers and other antihypertensive medications. Three years later (at age 40), he underwent surgical repair of a focal aneurysmal dilatation of the proximal half of the descending thoracic aorta using a Dacron Hemashield graft (Vascutek USA, Inc, Ann Arbor, MI). Left-atrial and femoral-artery bypass, permissive hypothermia (core temperature of 32°C), and CSF drainage were used as adjuncts during this initial surgery to successfully prevent paraplegia. When admitted to the hospital, he complained of severe back pain and was found to have substantial aneurysmal dilatation (greatest diameter 6 cm) of the remaining thoracoabdominal aorta. He also had a history of substantial hypertension (treated with beta-adrenergic blockers, central alpha-agonists, and vasodilators). Renal and hepatic functions were normal. Both lung fields were clear via chest radiography. Transesophageal echocardiography revealed normal ventricular function and absence of significant valvular abnormalities. Coronary angiography revealed normal coronary arteries.

Before the induction of anesthesia, a 14-G Tuohy needle was inserted in the intervertebral space between L4 and L5, and a silastic catheter (Medtronic, Inc, Minneapolis, MN) was advanced 10 cm within the intrathecal space over a wire. After confirmation of clear CSF drainage, anesthesia was induced with intravenous opioids, benzodiazepines, and a nondepolarizing muscle relaxant. A left-sided double-lumen endotracheal tube was inserted to facilitate surgical exposure. Anesthetic maintenance was achieved with additional intravenous opioid, benzodiazepine, and muscle relaxant, as well as an inhalation anesthetic agent. Bilateral radial arterial catheters were used, and CSF pressure was continuously monitored during the operation and in the postoperative period. CSF was freely drained to maintain a CSF pressure of 10 mmHg. The patient was placed in the lateral position, and the surgical incision was made at the sixth intercostal space. The diaphragm was divided circumferentially, and the left retroperitoneum was entered. The aorta was exposed from the previous graft to the aortic bifurcation and was treated initially with Medtronic EDM lumbar drainage kit and collection system; Medtronic, Inc, Minneapolis, MN) was advanced 10 cm within the intrathecal space over a wire. After confirmation of clear CSF drainage, anesthesia was induced with intravenous opioids, benzodiazepines, and a nondepolarizing muscle relaxant. A left-sided double-lumen endotracheal tube was inserted to facilitate surgical exposure. Anesthetic maintenance was achieved with additional intravenous opioid, benzodiazepine, and muscle relaxant, as well as an inhalation anesthetic agent. Bilateral radial arterial catheters were used, and CSF pressure was continuously monitored during the operation and in the postoperative period. CSF was freely drained to maintain a CSF pressure of 10 mmHg. The patient was placed in the lateral position, and the surgical incision was made at the sixth intercostal space. The diaphragm was divided circumferentially, and the left retroperitoneum was entered. The aorta was exposed from the previous graft to the aortic bifurcation. The patient was then heparinized (1 mg/kg). The left inferior pulmonary vein and left common femoral artery were cannulated with hep-
arin-coated cannulae and connected to a Biomedicus centrifugal pump (Medtronic; BioMedicus, Eden, MN). Next, the aorta was cross-clamped, and left atrial-to-femoral artery bypass was initiated. Distal flows were maintained between 2.0 to 3.0 L/min. At the same time, the proximal systolic pressure was maintained between 110 to 130 mmHg. A Dacron Hemashield graft was anastomosed to the previous graft proximally. The T7 to L1 intercostal arteries were reimplanted into the graft using the island technique. The celiac, superior mesenteric, and both renal arteries were also reattached to the graft using the same technique. The distal anastomosis was performed at the level of the aortic bifurcation. During the operation, the body temperature was allowed to drift passively to 32°C. A total of 250 mL of CSF were drained over the 10 intraoperative hours.

The operation proceeded uneventfully, and the patient was transferred to the intensive care unit (ICU). During the ICU stay, the CSF pressure was monitored continuously and CSF were drained as needed to maintain a CSF pressure of 10 mmHg. The patient emerged from general anesthesia within 12 hours of ICU arrival without evidence of any neurologic deficit. The patient was hemodynamically stable at this time, with adequate cardiac output and urine output. During the first 12 postoperative hours, only 80 mL of CSF were drained. Within the next 7 hours, 250 mL of CSF were drained, with CSF pressures ranging from 11 to 22 mmHg. Shortly thereafter (approximately 20 hours after ICU arrival), the CSF became blood tinged and the patient became unresponsive. It was also noted at this time that the pupils were dilated. An emergent computed tomography scan of the head without contrast was obtained. This revealed moderate hydrocephalus, subarachnoid hemorrhage, and a large midline intracerebellar hemorrhage with extension into both cerebellar hemispheres and the fourth ventricle. It was thought at this time that the intracerebellar hemorrhage had resulted from an acute herniation secondary to abrupt intracerebral hypotension secondary to CSF drainage. Hyperventilation was initiated, and a ventriculostomy tube was inserted by a neurosurgeon. The lumbar CSF catheter was removed, and an epidural blood patch was performed to hopefully prevent further CSF leakage. Over the course of several days, the patient’s neurologic status gradually returned to baseline. The ventriculostomy tube was removed, and the patient was discharged home.

**RETROSPECTIVE SERIES REVIEW**

Subsequently, as a part of ongoing quality assurance efforts, and after approval from the Hospital Human Investigation Committee, the outcomes of all patients undergoing TAAA repair from May 1998 to December 2004 in the authors’ hospital were retrospectively reviewed. All procedures reviewed were performed by the same clinical anesthesia and surgery teams (1 surgeon performed the vast majority of these procedures). Neurologic outcomes were retrospectively reviewed in an attempt to discover any potential relationship between postoperative stroke, cerebral hemorrhage, and paraplegia/paraparesis to the use of lumbar CSF drainage.

A total of 91 patients were reviewed. CSF drainage was used in 54 patients. Various bypass techniques were also used in an effort to reduce the incidence of spinal cord ischemia, including left-atrial femoral-arterial (LAFA) bypass in 51 patients, atrial-distal bypass in 2 patients, and femoral-femoral bypass in 13 patients (Fig 1). Permissive hypothermia was used in all patients. Patients in whom proximal aortic clamping was not feasible were managed with deep hypothermic circulatory arrest (DHCA). Most of the patients with type IV TAAA did not require distal bypass or CSF drainage.

Fig 1. Distribution of aortic pathology, CSF drainage, and other means of spinal cord protection. AD, atrial-distal bypass; FF, femoral-femoral bypass.

Of the 3 patients diagnosed with embolic stroke, 1 patient had a descending thoracic aneurysm with significant atherosclerosis of the aortic arch, which was believed to be the cause of the embolism. This patient recovered completely after hos-
hospital discharge. The second patient had a large aortic arch and descending thoracic aneurysm with significant amount of clot and underwent repair using FF bypass and DHCA. Postoperatively, this patient was found to have multiple embolic strokes, presumably from clot embolization. The third patient who experienced embolic stroke had a significant history of peripheral vascular disease and prior stroke, underwent uneventful repair of a type IV TAAA, and developed an embolic stroke 10 days after the operation. Two patients in the non-CSF drainage group had embolic strokes after repair.

In addition to the patient presented in this case report, 2 other patients were identified during retrospective series review who developed postoperative intracerebral hemorrhage. One of these patients had TAAA repair with LAFA bypass and CSF drainage. The day after surgery, it was noted that the CSF became blood tinged after 135 mL were drained over 2 hours. The patient remained comatose after the operation, and a computed tomography scan of the head revealed hydrocephalus and intracerebellar hemorrhage. This patient was treated with ventriculostomy insertion but did not survive. The other patient had a descending thoracic aneurysm repair with CSF drainage. On ICU admission, the pupils were fixed/dilated. A computed tomography scan of the head revealed hydrocephalus and a large right cerebellar hemorrhage. No information was available concerning the amount of CSF drained. Brain death was confirmed in this patient a few days later. Paraplegia/paraparesis occurred in 3 patients undergoing TAAA type II and type III aneurysm repair in the CSF drainage group. The incidence of neurologic complications in those patients treated with CSF drainage and without CSF drainage is presented in Table 1.

### TABLE 1. Complications in Groups With and Without Cerebrospinal Fluid Drainage

<table>
<thead>
<tr>
<th>CSF Drainage</th>
<th>Stroke</th>
<th>Cerebral hemorrhage</th>
<th>Paraplegia</th>
<th>Seizures</th>
<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>With CSF drainage (n = 54)</td>
<td>5.5%</td>
<td>5.5%</td>
<td>5.5%</td>
<td>1.8%</td>
<td>11.0%</td>
</tr>
<tr>
<td>Without CSF drainage (n = 37)</td>
<td>5.4%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
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</table>

Abbreviation: CSF, cerebrospinal fluid drainage.
It has been shown that during the period of cross-clamping for repair of descending and TAAA aneurysms, the distal aortic pressure decreases. Concurrently, the central venous pressure may increase, leading to an increase in production of CSF. Consequently, CSF pressure increases as well. CSF drainage reduces CSF pressure and may therefore augment spinal cord perfusion. Coexistent use of distal aortic perfusion should also augment spinal cord perfusion.

Numerous reports indicate that CSF drainage may be beneficial and likely safe. Safi and Miller revealed that the use of distal aortic perfusion through LAFA bypass and CSF drainage may decrease the incidence of neurologic complications, particularly in the high-risk group of patients with type I and II aneurysms. Distal aortic perfusion through LAFA bypass increases distal aortic pressure, whereas CSF drainage decreases pressure during aortic cross-clamping, both leading to augmentation of spinal cord perfusion.

Coselli and associates reported a prospective randomized trial, revealed that peroperative CSF drainage reduces the rate of paraplegia after repair of type I and II TAAA. CSF was drained freely to achieve a CSF pressure less than 10 mmHg. Overall, CSF drainage resulted in an 80% reduction in the relative risk of perioperative deficits. Additionally, sequential clamping of the aorta, permissive hypothermia (core temperature of 34°C), and reimplantation of critical intercostal arteries (patent T7 to L1 intercostal arteries) can help to prevent cord ischemia during surgical repair. Plestis and associates also used CSF drainage and LAFA bypass in the repair of descending thoracic and TAAA type I to III and observed no episodes of paraplegia. Heller and Chaney reported a case of paraplegia that developed immediately after the removal of a CSF drainage catheter. Lastly, Cheung and associates retrospectively reviewed the use of CSF catheters in 135 TAAA patients and reported no serious neurologic sequelae associated with CSF drainage to a pressure of 10 to 12 mmHg.

Nonetheless, there have been reports of complications associated with CSF drainage. Fracture of the CSF drainage catheter during removal has been reported. Meningitis, particularly in cases of prolonged use of the catheter, is a potentially serious life-threatening complication. Early diagnosis and appropriate antibiotic treatment are effective in the majority of these cases. Weaver and associates report a case of intradural hematoma compressing the cauda equina and creating paraplegia in a patient who had successful repair of a type III TAAA. Dardik and associates report an association between the total amount of CSF drained over a period of days and the development of subdural hematoma and suggest that intracranial subdural hematoma might develop from excessive lumbar CSF drainage (because of stretching and tearing of dural veins). In this series of 230 patients, the incidence of subdural hematoma was 3.5%, with an associated mortality of 50%. Seven of the 8 patients with subdural hematoma had CSF drained through a “pop-off” valve to a pressure of 5 cmH₂O and the other to a pressure of 10 cmH₂O. The average amount of drained CSF was 690 mL per patient with subdural hematoma, which was significantly higher than the average of 359 mL of CSF drained from patients without subdural hematoma. It has been postulated that the success of CSF drainage is dependent on the unmitigated flow of CSF, as advocated by Hollier and Moore. The amount of CSF drained is dependent on the CSF pressure, which should be kept below 10 to 12 mmHg. When CSF drainage is limited to a maximum of 50 mL, such practice may limit the spinal cord protective effects of the technique.

In all 3 patients with intracerebral bleeding presented in this series, there was a significant amount of CSF drainage over a short period of time. CSF pressure was monitored and drainage was set to maintain a CSF pressure of 10 mmHg perioperatively. In none of these patients was there a technical problem on insertion of the catheter. The mechanism of bleeding was most likely dural vein tearing, associated with herniation, secondary to intracranial hypotension stemming from excessive CSF drainage. In all cases, the appearance of blood-tinged CSF heralded severe neurologic injury. There were no significant problems in those patients who did not experience bloody CSF either intraoperatively or in the immediate postoperative period. The appearance of blood-tinged CSF appears to be an indication to obtain a computed tomography scan to rule out any potentially correctable intracranial pathology. CSF drainage, as guided by CSF pressure, may potentially lead to serious intracranial bleeding. It is possible that this problem may go underrecognized in the immediate postoperative period when attention is usually focused on hemodynamic optimization and patients remain sedated.

Currently, in the authors’ institution, CSF drainage is guided by CSF pressure. CSF is drained to achieve a CSF pressure of 10 mmHg. However, unrestricted drainage of CSF has been eliminated. No more than 10 mL of CSF are allowed to be drained in any 1 hour time period. Clearly, a prospective study must be undertaken to determine the safe amount of CSF that can be drained in any given time period. After drainage of 10 mL of CSF, if CSF pressure remains elevated, additional CSF drainage is drained in the next hour. As such, no more than 240 mL of CSF are allowed to be drained in any 24-hour period. Since instituting this protocol for all CSF drainage patients (38 patients), no cases of intracerebral hemorrhage or brain herniation have been observed. Likewise, no cases of paraplegia have been observed.

This report is limited by its retrospective nature and the lack of power to determine true statistical significance. Further studies are necessary to reveal the potential relationships between the pattern of CSF drainage, postoperative intracranial hemorrhage, and spinal cord paraplegia. Guidelines for drainage of CSF need to be prospectively tested. Although reduction of CSF pressure to 10 mmHg may prove most beneficial for the prevention of spinal cord injury, it is clear that drainage to this endpoint in certain patients is not without deleterious consequences. The exact amount of CSF that can be safely drained in...
any particular patient in any particular time period is uncertain. However, what appears certain is that excessive drainage, as guided by CSF pressure measurements, may be problematic and potentially injurious.

COMMENTARY 1

Thoracoabdominal aortic aneurysm surgery is a serious undertaking and paraplegia after TAAA repair is one of its most serious complications. Despite modern anesthetic and surgical techniques, mortality after TAAA repair averages 22.3% based on data from a random sampling of hospitals in the United States.14 The paraplegia rate after TAAA repair reported in the medical literature has ranged from 2% to 27%.15 In the typical elderly patient with atherosclerotic peripheral vascular disease undergoing TAAA, paraplegia is a lethal complication. Paraplegia is believed to be caused by spinal cord infarction from vascular insufficiency. Reconstruction of the descending aorta with an interposition graft requires the sacrifice or reimplantation of segmental arteries that supply the anterior spinal artery. With the loss of segmental arteries, the anterior spinal artery relies on its arterial supply from the vertebral arteries and pelvic collaterals, making the spinal cord prone to malperfusion. Spinal cord infarction after TAAA repair typically involves the lumbar cord and extends into the mid- or high-thoracic region. Infarction in the territory of the anterior spinal artery predominantly impairs motor function but can cause motor and sensory loss or asymmetric defects. Paralysis of abdominal muscles impairs respiration and contributes to postoperative respiratory failure.

A number of clinical strategies have been developed and tried in an attempt to decrease the mortality and paraplegia rate associated with TAAA repair. Strategies include passive shunting or partial left-heart bypass to provide distal aortic perfusion, intraoperative somatosensory- or motor-evoked potential monitoring to detect spinal cord ischemia, reimplantation of segmental arteries, selective cooling of the spinal cord with epidural infusion of cold saline, mild or moderate systemic hypothermia, deep hypothermic circulatory arrest, lumbar CSF drainage, arterial pressure augmentation, and pharmacologic neuroprotection with drugs such as glucocorticoids or naloxone. The heterogeneity of the patient population, regional variability in surgical techniques, and the combined use of multiple techniques in individual patients have made it difficult to isolate the therapeutic efficacy of any single intervention. The continued use of many of these spinal cord protection strategies and the absence of consensus on a single best approach to this problem suggests that a definitive solution has yet to be devised.

Of the many strategies described to protect the spinal cord from ischemia and infarction, lumbar CSF drainage has been one of the most extensively studied techniques.7 Lumbar CSF drainage is supported by the physiologic rationale that decreasing the lumbar CSF pressure would improve spinal cord perfusion pressure if arterial pressure is maintained. Although still controversial, the efficacy of lumbar CSF drainage for the prevention and treatment of paraplegia after TAAA repair has been supported by case reports, randomized trials, and case series.7,16-18 Complications attributed to lumbar CSF drainage that have been described including intraspinal hematoma, subdural hematoma, intracranial hypotension, meningitis, and catheter fracture.7 Based on existing reports in the literature, the risk of complications related to lumbar CSF drainage has been rare compared with the risk of paraplegia.7 Only 2 cases of hemorrhagic complications at the lumbar CSF catheter site have been described in the literature.7 Remote subdural hematoma as a consequence of intracranial hypotension was observed in 3.5% (8/230) of patients undergoing lumbar CSF drainage in 1 series, but patients in that clinical series had lumbar CSF drained to a pressure of 5 cmH₂O or the equivalent of 3.7 mmHg.7

In the case series described by Leyvi and associates, intracerebellar hemorrhage was observed in 5 out of 54 patients who had lumbar CSF drainage compared with 0 out of 37 patients who did not have lumbar CSF drainage for TAAA repair. Although the incidence of this complication was not statistically different between the 2 groups, the association is difficult to ignore. The authors speculate that excessive CSF drainage caused cerebellar herniation and intracerebellar hemorrhage. Although intracerebellar hemorrhage has not been previously reported as a complication of lumbar CSF drainage, remote cerebellar hemorrhage has been reported as a rare complication of spinal or supratentorial surgery.19,20 In every reported case of cerebellar hemorrhage after spinal surgery, dural opening with loss of CSF was a common feature.19 Similarly, remote cerebellar hemorrhage after supratentorial surgery was also associated with CSF volume loss from opening of CSF cisterns or the ventricular system.20 The pathophysiology of remote cerebellar hemorrhage is not completely understood but believed to be caused by intracranial hypotension from loss of CSF leading to caudal displacement of the cerebellum or cerebellar “sag,” occlusion of superior bridging veins within the posterior fossa, and subsequent hemorrhagic venous infarction within the cerebellum.

Besides the use of lumbar CSF drainage, the report by Leyvi and associates provides few clues to explain the high incidence of intracerebellar hemorrhage in their patient population. According to the clinical protocol described for the management of lumbar CSF drainage, lumbar CSF pressures were maintained in the range of 10 mmHg to 20 mmHg. This range of lumbar CSF pressure has not been previously associated with intracranial hypotension.2,3 Furthermore, the volume of CSF drained from the lumbar CSF catheter was not excessive and consistent with previous experience in published reports.7,7 Additional detail describing how lumbar CSF pressure was measured, the position of the lumbar CSF pressure transducer, the duration of lumbar CSF drainage, the position of patients during lumbar CSF drainage, or persistent CSF leak after catheter removal may provide additional insights to understand why patients undergoing lumbar CSF drainage in the series suffered intracerebellar hemorrhage. Preoperative aspirin use and perioperative hypertension have been implicated as possible risk factors for remote cerebellar hemorrhage after neurosurgical procedures and may have also contributed to the risk of this complication after TAAA repair.20 Anticoagulation for partial left heart bypass and postoperative coagulopathy or

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thrombocytopenia as a consequence of extracorporeal circulation may have also added to the risk of intracerebellar hemorrhage.

As the technique of lumbar CSF drainage for spinal cord protection for TAAA repair becomes more widely used, it is important to identify potential complications of the procedure, understand the underlying cause of complications, and formulate strategies to minimize risk. Based on the report by Leyvi and associates and related reports in the literature, it has become apparent that intracranial hypotension is a potentially serious complication of lumbar CSF drainage. The infrequency of this complication and an incomplete understanding of the risk factors contributing to this complication limit the ability to establish specific clinical guidelines for the management of lumbar CSF drainage. Based on the existing knowledge and postulated pathophysiology of remote intracerebellar hemorrhage, precise measurement of the lumbar CSF pressure, close monitoring of the volume and rate of CSF drainage, avoidance of antiplatelet or anticoagulant drugs, and early recognition and treatment of complications may improve the safety of the procedure. More experience is necessary to determine whether limiting the volume of CSF drainage, discontinuing lumbar CSF drainage for 24 hours before removing the lumbar CSF catheter, or keeping the patient in a supine position for at least 6 hours after removal of the lumbar CSF catheter are effective for decreasing the risk of this complication. More experience is also necessary to determine if epidural blood patch is an effective treatment for intracerebellar hemorrhage after lumbar CSF drainage.

The risks and benefits of lumbar CSF drainage for the prevention and treatment of spinal cord ischemia in patients undergoing TAAA repair need to be continuously reassessed. Until safer and more effective alternatives to lumbar CSF drainage for the prevention and treatment of postoperative paraplegia are developed, it is likely that lumbar CSF drainage will continue to be used. In the meantime, incremental progress can be made by systematically reporting complications to guide the implementation of clinical practices directed at decreasing the complication rate associated with lumbar CSF drainage.

COMMENTARY 2

A case of intracerebellar hemorrhage after TAAA is described. The timing of the event, on the second postoperative day, while the patient was already awake and neurologically intact but CSF drainage was still in place triggered a possible causative etiology and the catheter was thus withdrawn. During the operation and in the postoperative period, considerable amount of CSF was drained while the CSF pressure was kept within the normal range (above 11 mmHg). When the patient’s condition deteriorated and the intracranial bleeding was discovered, an epidural blood patch procedure was performed in an attempt to prevent the ongoing leakage of CSF. The authors, on this occasion, retrospectively reviewed the neurologic outcome of their patients operated on for TAAA surgery, 91 patients over a 6-year period, and revealed 2 more cases of cerebellar bleeding (only in the group of patients in whom CSF drainage was used). These findings raise some issues to be considered, such as the incidence of cerebrovascular events after descending aortic surgery and their etiology and the potential correlation to CSF drainage.

Stroke after aortic surgery is a devastating complication and is usually thromboembolic in origin, which can result in ischemic or hemorrhagic presentation. Large case series in patients undergoing TAAA surgery define the incidence of these neurologic events; however, the exact nature and location of injury are rarely given. In a recent study examining a predictive model for adverse outcome in elective TAAA surgery in 1,108 patients, 15 (1.4%) suffered stroke.21 Another study involved all types of thoracic aortic surgery, including aortic arch replacement; the stroke rate was higher, 5.1% in this series of 1,157 patients,22 and an additional 6.6% of patients experienced transient neurologic dysfunction (again, the exact nature of the injury received less attention). Goldstein and associates,23 in reviewing their experience in 317 patients, found a higher stroke rate in aortic arch and descending aorta surgery, 6.9% and 8.1%, respectively; two thirds of their cases were embolic in etiology and the rest ischemic and hemorrhagic. In all patients undergoing descending thoracic aortic surgery, CSF drainage was used routinely. The authors report that by giving special attention to antiembolic measures, they dramatically reduced the rate of neurologic events. The location of infarct and hemorrhage was not limited to the middle cerebral artery territory but was evident in all brain zones, including the cerebellum. They also revealed that stroke has a devastating impact on survival (28% in-hospital mortality and only approximately 30% were still alive 2 years after the event). In another study reviewing 841 patients who were operated on for TAAA,24 the stroke rate was 4.5% and significant predictors for the neurologic deficit were the use of adjuncts such as CSF drainage and distal aortic perfusion.

Recently, sporadic reports on intracranial hemorrhage have occurred while spinal catheters were still in place, implicating CSF drainage as a possible explanation for the cerebral bleeding. McHardy and associates25 described a case of a patient in whom the spinal drainage catheter was withdrawn on the second postoperative day; but on the fourth day, while sitting in a chair, he complained of severe headache and then collapsed. Large acute subdural hematoma was diagnosed, and the patient died soon after. The authors suggest that the intentional drainage of CSF, which was followed by continual CSF leakage through the punctured dura, may have caused acute cerebral hypotension on the change in patient position, with traction on brain structures and tearing of the thin-walled dural veins and subsequent subdural hemorrhage. Darlid and associates described in a retrospective review that 8 patients out of 230 (3.5%) developed subdural hematomas. However, only 1 patient developed neurologic symptoms while the spinal drainage catheter was still in place. Acute presentation of subdural hematoma occurred in 6 of the patients between the second and the sixteenth postoperative day; only 2 of these patients survived. The authors did not give the exact location of the cerebral bleeding nor did they report other neurologic complications such as ischemic stroke or transient symptoms. Their multivariate analysis emphasized that the only significant factor

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that correlated to the occurrence of subdural hematoma was the volume of CSF drained.

The reports described earlier present confusing data regarding the incidence and nature of brain injury after descending aortic surgery. Large series of patients produce rates of 5% to 8% in this type of surgery, most ischemic in nature, whereas others describe only cases of intracerebral bleeding. Regardless, the presentation of such high incidence of neurologic injury should be taken seriously, and if part of it is a result of an iatrogenic maneuver taken by the medical team, calculating actual risk-benefit status of the maneuver should be seriously considered. A comprehensive cautionary approach should be applied, and preventive measures should be considered when such techniques applied. Despite conflicting data, the role of CSF drainage as a causative etiology to subarachnoid, intracerebral, or subtentorial hemorrhage should be addressed. It may be attributed to cerebral hypotension-related traction effect, which subsequently may trigger a tear of bridging veins and intracranial hemorrhage. The occurrence of cerebral bleeding after routine procedures involving CSF shift, whether in the format of diagnostic lumbar puncture or ventriculoperitoneal shunt, has been previously described and has been attributed to the sudden pressure shifts. However, the incidence of such devastating complications in those commonly performed procedures is extremely rare. The rarity of this phenomenon undermines the hypothesis that a dural puncture with subsequent CSF leak is the sole mechanism behind these intracranial hemorrhages. On the other hand, intraparenchymal hemorrhage is relatively common in patients with cerebrovascular disease or widespread vasculopathy, to which the present patient population usually belongs. Thus, any intracranial hemorrhagic complication should probably be first attributed to the native cerebrovascular disease in these patients. This risk is compounded by the previously described high incidence of embolic events in these patients, of which the hemorrhage is a secondary complication of the ischemic stroke. Goldstein and associates have proposed a systematic approach to help reduce embolic events during aortic surgery, which includes intraoperative transesophageal echocardiography visualization of aortic atheromas, careful debridement of unstable atheromas, prevention of retrograde arterial flow by early application of the proximal aortic cross-clamp, and carbon dioxide flooding of the surgical field to replace air in possible gas embolization. From the stroke rate published in the literature, it seems that the probability for intracranial bleeding and for ischemic stroke after high aortic surgery are similar. Nevertheless, coexistent intracerebral bleeding while intensive CSF drainage is applied, or while a spinal drainage system is in place, should not be ignored and alert clinicians to take careful measures to help prevent any neurologic complications.

Interventions involving CSF sampling, spinal anesthesia administration, ventricular pressure relief, or drainage may lead to the development of ischemic symptoms such as cranial nerve palsy, and such symptoms may last for an extended period because of the continuous leakage of CSF through the dural puncture hole. A substantial loss of CSF may retract the brain from the meninges or, in the case of the cerebellum, from the tentorium. This may cause a temporary occlusion of stretched veins, increase intraparenchymal venous pressure, and create the appropriate environment for venous hemorrhage. The leakage of CSF into the epidural space can be significant, more than 200 mL per day, and thus, if symptoms persist, possible curative measures (increased oral fluid intake, intravenous hydration, or epidural blood patch) should be performed.

The caution advocated in the use of CSF drainage mandates a careful approach to the volume of fluid allowed to drain. The goal of CSF drainage is to improve spinal perfusion pressure; thus, during operation and during the cross-clamp period, a positive value of 10 mmHg of CSF pressure is reasonable to maintain. In the postoperative period, the spinal cord is edematous for an extended period of time, and therefore a tight control of blood pressure and prevention of hypotensive episodes are crucial. New-onset paraplegia is frequently reported in patients who suffer prolonged episodes of systemic hypotension. Aggressive management of blood pressure and active CSF drainage may reverse these neurologic symptoms. During the postoperative period, modulation of CSF pressure should be used only as a secondary method in maintaining spinal perfusion pressure, and CSF may be drained whenever CSF pressure exceeds the normal value of 15 mmHg. The practice of free drainage of CSF in the postoperative period should be reexamined in light of the current reports of possible neurologic hemorrhagic complications. The use of a large-bore needle for the introduction of the 4F spinal drainage catheter creates a large hole that remains in the dura after catheter removal. The practice of prophylactic performance of epidural blood patch after catheter withdrawal may be considered to potentially seal the dural hole and to stop the chronic leak of CSF. Careful monitoring of the patient’s neurologic status in the postoperative period is required to detect possible ischemic changes in the spinal cord and for the possible appearance of central neurologic complications. Prompt identification and proper treatment may help prevent the adverse outcome of such events.

REFERENCES