Editorial

Is cerebrospinal fluid drainage safe and of potential therapeutic benefit after acute traumatic spinal cord injury?

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Following acute neurotrauma, including traumatic brain injury (TBI) and spinal cord injury (SCI), the primary mechanical trauma is followed by a series of pathological events including ischemia, oxidative cell injury, glutamatergic excitotoxicity, apoptosis, inflammation, and edema. In acute severe TBI, standard clinical management involves placing an intraventricular catheter that can be used to measure intracranial pressure and drain CSF. Of note, this approach has not been used in the setting of acute SCI, for a variety of reasons including concerns that release of CSF below a swollen, compressed cord could result in neurological deterioration. However, there are some theoretical advantages to placing a lumbar subarachnoid catheter following acute SCI: 1) there is evidence that drainage of CSF may enhance neurological recovery following ischemic cord injury in the setting of aortic surgery; 2) measurement of spinal CSF pressures could allow targeted hypertensive therapy directed at maintaining spinal cord perfusion pressures (SCPPs) above a critical threshold; and 3) in the future, measurement of critical biomarkers in the CSF may provide prognostic and therapeutic information.

To resolve issues related to the safety of lumbar spinal subarachnoid catheter placement and drainage of CSF following acute SCI, Kwon and colleagues undertook a prospective randomized clinical trial in 24 patients with ASIA Grade A–C cervical and thoracic SCI. Two patients were excluded from the analysis for technical reasons. Lumbar subarachnoid catheters were inserted 21 hours after injury and maintained for 72 hours. Patients were randomly assigned to undergo drainage or no drainage. The CSF waveforms were dampened at initial placement of the lumbar subarachnoid catheter and release of CSF (which can occur around a catheter also).

Interesting data were obtained with regard to ITP and CSF pressure waveforms. The ITP rose in all but one case after surgical decompression, which suggests that there was a subarachnoid block at the injury site that dampened transmission of CSF pressures. Unfortunately there was not a standardized protocol for mean arterial blood pressure (MABP) management. This was reflected in differences in the MABPs between the drainage and no-drainage groups, which rendered the small changes in apparent SCPP difficult to interpret. Peak ITP in the postoperative period was significantly higher than the peak intraoperative value in the no-drainage group but not in the drainage group. The CSF waveforms were dampened at initial placement of the lumbar subarachnoid catheters but assumed a more robust appearance after surgical decompression. In some clinical cases, spinal cord swelling or an epidural hematoma also contributed to a delayed attenuation of the waveform. Cerebrospinal fluid drainage was not associated with improved neurological recovery at 6 months, although the amount of CSF drained was fairly minimal (in 7 of 11 patients < 51 ml was drained). This reflected a conservatism by the authors against draining excessive amounts of CSF for fear of causing harm, technical issues related to the catheter, and the presence of relative subarachnoid block due to cord swelling.

In summary, the study by Kwon and colleagues suggests that lumbar subarachnoid catheters can be placed safely in patients with acute SCI, particularly if spinal cord decompression is undertaken. This provides the opportunity to assess CSF for biomarkers that could provide prognostic and therapeutic insights. However, it is unclear whether measurement of lumbar ITP will provide meaningful acute physiological data given the high rate of subarachnoid fluid block that occurs after traumatic SCI (and which may persist in the initial acute period, even with extradural cord decompression—as shown in some of the MR imaging studies in the article). Although this study failed to show any therapeutic benefits to CSF drainage after acute SCI, the amounts of CSF drained were minimal.

I look forward to further prospective studies of CSF drainage from this group to address some of the key outstanding issues raised by this small pilot study. In particular, elucidation of the potential role of CSF biomarkers will be of great interest to the field.


See the corresponding article in this issue, pp 181–193.
References


Response

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We thank Dr. Fehlings for his insightful review of our article. We sought to examine the safety and feasibility of active CSF drainage to reduce intrathecal pressure and improve SCPP—in essence, to examine the potential applicability of an intervention that appears to be neuroprotective in thoracoabdominal aortic aneurysm surgery. We were aware of the potential danger associated with such drainage, although the reports of neurological deterioration appeared to be related to a more chronic subarachnoid space occlusion (for example, from tumors and not from acute trauma). Nonetheless, we undertook a very conservative drainage protocol, with respect to the amount of CSF drained, the speed at which the ITP could be lowered, and the times that we could actually drain CSF (only when the patients were capable of being examined). As Dr. Fehlings points out, this certainly reduced the amount of CSF actually drained and limits the interpretation of how applicable active CSF drainage would be in this patient population.

The changes in ITP after decompression were quite striking. As Dr. Fehlings correctly points out, there was not a standardized protocol for maintaining the MABP. Nonetheless, the Pearson correlation coefficients show that increases in ITP were not met by concomitant changes in MABP, in either the patients randomized to receive drainage or no drainage. Hence, the absence of a standardized protocol does not change the fact that when ITP rises, such rises are not naturally offset by changes in the MABP.

Ultimately, this raises a much larger question as to whether such monitoring of ITP is worthwhile in the routine clinical care of acute SCI patients. Considering that aggressive hemodynamic support to minimize cord hypoperfusion is one of the few things that we have to offer these patients, it would seem rational to optimize our clinical delivery of that support. If the ITP changes are indeed associated with a reduction in SCPP, then such knowledge might benefit clinicians in how they manage these patients. Of course, we would accept that it is technically a bit more challenging to know exactly what the SCPP is at the site of injury where there may or may not be ongoing extradural compression. Nonetheless, even in patients with a pulsatile waveform (indicative of a patent subarachnoid space) we did witness large increases in ITP, which presumably are being applied to the injured cord. We contend that this may be quite meaningful from a pathophysiological standpoint, given the spinal cord’s considerable vulnerability to ischemic injury. Currently, we are prospectively seeking to determine how other aspects of routine clinical care, such as changing vasopressor agents, may influence ITP and SCPP.

Finally, this initiative has provided the very valuable opportunity to biochemically examine the CSF and has revealed a number of potential biomarkers of injury severity. Such biomarkers may help to classify the severity of injury in patients whose neurological status is obscured by sedation/intoxication or head injuries and predict neurological recovery. We have identified a series of promising CSF biomarkers in this regard and are currently preparing this work for publication. (DOI: 10.3171/2008.11.SPINE08682)