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Intrathecal pressure monitoring and cerebrospinal fluid drainage in acute spinal cord injury: a prospective randomized trial

Clinical article

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Object. Ischemia is an important factor in the pathophysiology of secondary damage after traumatic spinal cord injury (SCI) and, in the setting of thoracoabdominal aortic aneurysm repair, can be the primary cause of paralysis. Lowering the intrathecal pressure (ITP) by draining CSF is routinely done in thoracoabdominal aortic aneurysm surgery but has not been evaluated in the setting of acute traumatic SCI. Additionally, while much attention is directed toward maintaining an adequate mean arterial blood pressure (MABP) in the acute postinjury phase, little is known about what is happening to the ITP during this period when spinal cord perfusion pressure (MABP – ITP) is important. The objectives of this study were to: 1) evaluate the safety and feasibility of draining CSF to lower ITP after acute traumatic SCI; 2) evaluate changes in ITP before and after surgical decompression; and 3) measure neurological recovery in relation to the drainage of CSF.

Methods. Twenty-two patients seen within 48 hours of injury were prospectively randomized to a drainage or no-drainage treatment group. In all cases a lumbar intrathecal catheter was inserted for 72 hours. Acute complications of headache/nausea/vomiting, meningitis, or neurological deterioration were carefully monitored. Acute Spinal Cord Injury motor scores were documented at baseline and at 6 months postinjury.

Results. On insertion of the catheter, mean ITP was 13.8 ± 1.3 mm Hg (± SD), and it increased to a mean peak of 21.7 ± 1.5 mm Hg intraoperatively. The difference between the starting ITP on catheter insertion and the observed peak intrathecal pressure after decompression was, on average, an increase of 7.9 ± 1.6 mm Hg ($p < 0.0001$, paired t-test). During the postoperative period, the peak recorded ITP in the patients randomized to the no-drainage group was 30.6 ± 2.3 mm Hg, which was significantly higher than the peak intraoperative ITP ($p = 0.0098$). During the same period, the peak recorded ITP in patients randomized to receive drainage was 28.1 ± 2.8 mm Hg, which was not statistically higher than the peak intraoperative ITP ($p = 0.15$).

Conclusions. The insertion of lumbar intrathecal catheters and the drainage of CSF were not associated with significant adverse events, although the cohort was small and only a limited amount of CSF was drained. Intraoperative decompression of the spinal cord results in an increase in the ITP measured caudal to the injury site. Increases in intrathecal pressure are additionally observed in the postoperative period. These increases in intrathecal pressure result in reduced spinal cord perfusion that will otherwise go undetected when measuring only the MABP. Characteristic changes in the observed intrathecal pressure waveform occur after surgical decompression, reflecting the restoration of CSF flow across the SCI site. As such, the waveform pattern may be used intraoperatively to determine if adequate decompression of the thecal sac has been accomplished. *(DOI: 10.3171/2008.10.SPINE08217)*

Key Words • **cerebrospinal fluid** • **intrathecal pressure** • **spinal cord injury** • **spinal cord perfusion pressure**

Intensive scientific and clinical research efforts over the past 30 years have regrettably failed to produce convincingly efficacious therapies to reverse the dev-

Abbreviations used in this paper: ASIA = American Spinal Injury Association; ITP = intrathecal pressure; MABP = mean arterial blood pressure; SCI = spinal cord injury; SCPP = spinal cord perfusion pressure; SEM = standard error of the mean.

This article contains some figures that are displayed in color online but in black and white in the print edition.

astating paralysis of traumatic SCI, although much progress has been made in the overall medical, surgical, and rehabilitative care of these patients.[3] The maintenance of adequate vascular perfusion[8] and the decompression of the spinal cord[6] are 2 important aspects of the clinical management of the acutely spinal cord–injured patient that have received considerable attention in the past 2 decades, largely due to the belief that they have an influence on neurologic recovery.

With respect to maintenance of adequate vascular perfusion, it is accepted that systemic hypotension should be avoided in the acute injury phase, as this contributes to cord ischemia and secondary parenchymal damage.[18] Investigators have reported worsened neurological function in association with hypotension during the acute resuscitation of patients with SCI.[9] It is therefore common practice for clinicians to maintain the MABP in a patient with an acute SCI at a certain level (for example, 85–90 mm Hg) with the expectation that this will provide an acceptable level of vascular perfusion to the spinal cord.[13,19] However, it should be recognized that the actual perfusion to the spinal cord is not solely determined by the MABP. Rather, spinal cord perfusion is influenced both by the MABP and by the CSF pressure in the intrathecal space (the so-called ITP), such that the SCPP is calculated as the difference between the two. A decrease in SCPP (resulting in cord ischemia) can therefore occur with either a decrease in MABP or an increase in the ITP, or both.

In patients undergoing thoracoabdominal aortic aneurysm surgery, the reduction in SCPP can by itself lead to ischemic damage to the spinal cord and lower-extremity paralysis, one of the most feared complications of this surgery. This has led to the practice of intraoperatively draining CSF to lower the ITP—a practice that has not only been reported in prospective randomized clinical trials as a prophylactic measure to reduce the incidence of ischemic paraplegia,[2,14] but also has been reported in case series to reverse late-onset paraplegia when instituted after the ischemic paralysis has occurred.[1,15] The efficacy of CSF drainage in attenuating clinically meaningful ischemia to the spinal cord in patients with thoracoabdominal aortic aneurysm and the importance of ischemia in the pathophysiological milieu of secondary damage after acute traumatic SCI compelled us to study this intervention in patients with acute traumatic SCI. Such a practice of draining CSF to reduce ITP in this clinical setting has, to our knowledge, not been previously described.

The objectives of this study were threefold: 1) to evaluate the approach of draining CSF to lower ITP after acute traumatic SCI; 2) to evaluate changes in ITP before and after surgical decompression; and 3) to evaluate changes in CSF pressure waveform as they relate to surgical decompression.

Methods

Enrollment

Patients were recruited to participate in this prospective randomized clinical trial based on the following inclusion criteria: 1) ASIA grade A (complete) or B and C (incomplete) SCI upon presentation; 2) SCI between C-3 and T-11 inclusive; 3) presentation within 48 hours of injury; and 4) ability to undergo a valid, reliable neurological examination. Patients were excluded if they had concomitant head injuries, concomitant major trauma to the chest, pelvis, or extremities that required invasive intervention (for example, chest tube insertion, internal or external fixation), or were too sedated or intoxicated to undergo a valid neurological examination. The patients themselves had to provide informed consent (that is, third-party assent was not allowed), which was obtained by 1 of 3 research study nurses.

Following confirmation of eligibility and informed consent, the patients were block randomized using a pregenerated randomization table according to the severity of their paralysis (complete injury [ASIA Grade A] vs incomplete injury [ASIA Grade B or C]) to 1 of 2 groups: CSF drainage to lower ITP to 10 mm Hg or no CSF drainage. The clinical trial protocol was granted approval from both the university human ethics committee and the hospital clinical trials administrative body, and it was registered with the US National Institutes of Health (ClinicalTrials.gov identifier NCT00135278).

Drain Insertion

Patients were log-rolled into the lateral position under the supervision of the spine surgeon, who maintained the cervical and thoracolumbar spine in neutral alignment during the placement of the catheter. Using strict aseptic technique, a lumbar puncture was performed at L2–3 or L3–4 and an intrathecal catheter (PERIFIX Custom Epidural Anesthesia Kit, Braun Medical, Inc.) was inserted and advanced 15–20 cm from the entry point on the skin surface. While this is an epidural catheter, it is routinely used at our institution for intrathecal drainage, particularly in patients with thoracoabdominal aortic aneurysms in whom CSF drainage is performed to lower ITP. As it emerged through the skin, the catheter was secured to the skin with a sterile adhesive dressing, and then the catheter was brought out over the shoulder and secured along its length to the skin with Mepore tape. After confirmation of CSF flow through the catheter, it was attached to a Becker External Drainage and Monitoring System (Medtronic, Inc.) for the drainage of CSF and the measurement of ITP caudal to the SCI. All patients were given prophylactic antibiotics (cefazolin or vancomycin) postoperatively for 24 hours, as is standard practice for cases involving such surgical wounds. Further antibiotic coverage specifically for the 72 hours that the catheter was indwelling was not instituted.

Intrathecal Pressure Monitoring and CSF Drainage

To zero the pressure transducer of the catheter system, a laser-sighting device mounted onto the external drainage and monitoring system was used to identify the midaxillary line as an estimate of the level of the right atrium. Initial zeroing was done with the patient lying completely horizontal, and the transducer was "re-zeroed"

with any changes in the bed height or inclination (particularly during the surgical procedures). The transducer was connected to a standard multichannel patient monitoring system (SpaceLabs Healthcare) to allow real-time recording of the MABP, ITP, and electrocardiogram.

Specific parameters measured included the starting ITP, the change in pressure following surgical decompression, and the ITP and MABP postoperatively until the drain was removed 72 hours later. In particular, the "peak" elevations in ITP were documented during the intra- and postoperative period, as such peak elevations in ITP represented episodes during which the SCPP would potentially be at its lowest (assuming a constant MABP). Tracings of the CSF pressure waveform were also taken and analyzed for changes in pattern before and after surgical decompression.

For patients randomized to CSF drainage, the external drainage and monitoring system was set to allow free drainage down to a level of 10 mm Hg, as is the typical target pressure for patients undergoing thoracoabdominal aortic aneurysm surgery.[11] Because ITP measurements and waveform recordings were not obtainable when the drain was opened to free drainage, each hour, the drain was momentarily closed off to record the ITP and the CSF pressure waveform. Once these were documented, the 3-way stopcock was turned back to open the system to drainage. In accordance with our protocol that specifically focused on patient safety during the intervention, during periods when the patient could not be examined neurologically, the drainage system was closed and only ITP monitoring was performed. These periods included the intraoperative and early postoperative periods, when the patient was under or recovering from general anesthesia, and postoperative periods, when the extent of pharmacological sedation made it impossible to adequately monitor the patient's neurological status. Additionally, we imposed a limit of 10 ml of CSF as the maximum volume of CSF that could be drained per hour. Samples of CSF were drawn daily for routine cell count and bacteriology studies (per our institutional protocol for indwelling intrathecal catheters). Additional CSF samples of 2–3 ml were drawn at 8-hour intervals for biochemical analysis (the subject of a separate initiative).

Recordings of CSF Pressure Waveforms

Normally, the CSF pressure waveform, as measured by extraventricular drains (in traumatic brain injury) or by lumbar intrathecal catheters inserted for nontraumatic conditions, is pulsatile, reflecting arterial pulsations within the ventricles. We noted the pulsatile or nonpulsatile nature of the CSF pressure waveform before and after surgical decompression to determine if the pulsatility of the waveform could indicate a reestablishment of the subarachnoid space across the injury site. Cerebrospinal fluid pressure waveforms and arterial pressure waveforms were recorded using General Electric Medical Systems Solar patient monitors. Each waveform was sampled at 120 Hz at a resolution of 0.2 mm Hg. Digital data from patient monitors were sent to a computer in real time via custom software and saved to hard disk for later analysis.

Clinical Evaluation

Upon their presentation to hospital, all patients underwent formal neurological testing according to ASIA standards, and then upper- and lower-extremity motor scores were recorded daily while the intrathecal drains were in place (in addition to the standard neurological monitoring done by the nursing staff twice per day). All baseline neurological examinations and subsequent neurological monitoring with the intrathecal catheter in place were conducted by clinical staff with substantial experience with the ASIA examination techniques. While the drain was in place, patients were monitored daily for changes in neurological function, symptoms of nausea/vomiting/headache suggestive of a CSF leak, and signs and symptoms of meningitis. The ASIA motor scores were then recorded at 6 months postinjury by an individual blinded to the patient's randomization group.

Statistical Analysis

Changes in ITP after spinal decompression in the intra- and postoperative periods were analyzed using paired t-tests. The generalized estimate equation was used to model the relationship between SCPP and MABP, ITP, and time for those patients randomized to CSF drainage and those randomized to no drainage. Pearson correlation coefficients were calculated to analyze the relationship between MABP and ITP. Mean values are presented ± SEM.

Results

Enrollment began in March of 2006, and 24 patients were subsequently recruited. In 2 patients with complete ASIA A injuries (1 cervical and 1 thoracic) in the no-drainage group, the catheter was deemed to have pulled out of the intrathecal space almost immediately after placement, and hence, these 2 patients were excluded from the analysis. This left 22 patients for the analysis, 11 randomized to undergo drainage and 11 to undergo no drainage (Tables 1 and 2). Fifteen male and 7 female patients were included, with an average age was 41.3 ± 2.7 years. Seventeen patients had cervical and 5 had thoracic injuries, and on admission, the severity of paralysis was determined to be ASIA Grade A in 12, Grade B in 6, and Grade C in 4 patients. One patient, a 66-year-old man randomized to the no-drainage group, died 15 months postinjury due to metastatic gastric adenocarcinoma. The ASIA motor scores were recorded 6 months postinjury. In 1 patient, a 55-year-old woman randomized to drainage, the initial and intraoperative ITPs were not recorded due to technical problems with the pressure transducer system that could not be rectified intraoperatively.

Adverse Events

Complications specifically related to either the intrathecal catheter or to the act of draining CSF were carefully documented on a daily basis for the 72 hours that the drain was in place. None of the 11 patients randomized to the CSF drainage group complained of a headache, nausea, or vomiting during this 72-hour period, suggest-

TABLE 1: Patients randomized to the no-drainage group: demographic, intraoperative, and postoperative data*

Case No.	Age (yrs), Sex	Mechanism of Injury	Spinal Injury	Site/ ASIA Grade	% Canal Compromise	Surgery	Timing of Op (hrs postinjury)	Pre- and Intraop Period (mm Hg)			Postop Period (mm Hg)				ASIA Motor Score		
								Initial ITP	Peak ITP Post-decompression	Mean ITP	Mean ITP	Peak ITP	Mean MABP	Mean SCPP	Baseline	6 Mos Postop	Segmental Recovery (cervical only)
1	29, M	blow to head (work-related)	C-5 burst fracture	C-6, C	46.1	ant C-5 vertebrectomy, C4–6 fusion	9.5	10	33	22.4	18.0	33	100.3	82.6	57	98	41
2	34, M	fall from ladder	C5–6 fracture dislocation	C-6, B	38.9	ant C5–6 & C6–7 discectomy, C5–7 fusion; pst C5–7 laminectomy & fusion	24.5	2	4	3.7	11.9	19	63.3	52.0	20	29	9
4	42, M	MVA	C6–7 fracture dislocation	C-6, B	41.5	pst C5–7 laminectomy, C4–T1 fusion	20.5	23	18	20.4	23.0	40	75.9	52.3	20	24	4
7	46, F	fall from ladder	C6–7 fracture dislocation	C-6, A	45.1	pst C6–7 laminectomy, C4–T6 fusion	25.75	7	10	8.7	17.2	26	74.6	57.0	10	14	4
12	50, F	MVA (rollover)	C5–6 fracture dislocation	C-5, A	43.8	ant C-5 vertebrectomy, C4–6 fusion; pst C4–6 laminectomy & fusion	25.5	6	29	19.8	19.2	29	74.3	54.5	9	13	4
14	23, M	MVA (head-on collision)	C-6 teardrop fracture	C-6, A	51.2	ant C-6 vertebrectomy, C5–7 fusion	19	14	16	13.8	15.1	24	82.6	67.2	23	31	8
15	23, M	blow to head (work-related)	C6–7 bilat facet dislocation	C-6, A	53.3	pst C4–T1 fusion	10.5	10	13	14.0	22.7	38	74.8	51.9	25	28	3
19	30, M	MVA (rollover)	C5–6 fracture dislocation	C-4, A	79.2	ant C-6 vertebrectomy, C5–7 fusion; pst C4–T1 laminectomy & fusion	24	25	27	28.6	24.0	30	73.4	49.6	4	8	4
21	46, M	bicycle accident	C-5 burst fracture	C-5, C	40.9	ant C-5 vertebrectomy, C4–6 fusion	32.5	21	25	23.0	12.1	25	82.8	70.8	16	70	54
23	31, M	MVA (rollover)	C6–7 fracture dislocation	C-6, A	53.9	posterior C6–7 laminectomy, C4–T2 fusion	40	23	25	21.5	24.6	44	71.8	47.2	17	40	23

(continued)

TABLE 1: Patients randomized to the no-drainage group: demographic, intraoperative, and postoperative data* *(continued)*

Case No.	Age (yrs), Sex	Mechanism of Injury	Spinal Injury	Site/ ASIA Grade	% Canal Compromise	Surgery	Timing of Op (hrs postinjury)	Pre- and Intraop Period (mm Hg)			Postop Period (mm Hg)				ASIA Motor Score		
								Initial ITP	Peak ITP Post-decompression	Mean ITP	Mean ITP	Peak ITP	Mean MABP	Mean SCPP	Baseline	6 Mos Postop	Segmental Recovery (cervical only)
6	66, M	fall from standing height	C4–5 hyperextension w/ laminar fracture	C-4, C	29.6	pst C3–6 laminectomy, C3–6 fusion	19.5	13	24	18.5	17.7	29	78.9	60.7	20	44	24
overall†	38.2 ± 2.8			11 cervical: 6 A, 2 B, 3 C	47.6 ± 2.7		22.8 ± 2.6	14.0 ± 2.4	20.4 ± 2.7	17.7 ± 2.1	18.7 ± 1.4	30.6 ± 2.3	77.5 ± 2.8	58.7 ± 3.3	20.1 ± 4.2	36.3 ± 8.1	16.2 ± 5.3

* ant = anterior; MVA = motor vehicle accident; NA = not applicable; pst = posterior.

† Except for Site/ASIA Grade category, all values are presented as the mean ± SEM.

ing that they were suffering lumbar CSF leakage. One patient randomized to the no-drainage group complained of a transient headache while the intrathecal drain was in place. No patient in either arm exhibited the signs or symptoms of meningitis during the 72-hour period. One patient randomized to the no-drainage group suffered a gram-negative posterior cervical wound infection and subsequently developed meningitis, but these infectious complications occurred over 1 week after the catheter had been removed from his lumbar spine. He was successfully treated with antibiotic agents. No patient in either group suffered a neurological deterioration during drain insertion, routine CSF sampling, or CSF drainage.

Changes in ITP With Surgical Decompression

For all 22 patients, the mean time between injury and catheter insertion was 21.6 ± 1.8 hours. The 22 patients randomized to both the drainage and the no-drainage groups were analyzed as a single cohort during this time because drainage was not initiated during the operation due to the inability to clinically assess neurological function during this period. In all patients, however, the catheters were connected to a pressure transducer for measurement of ITP during the surgical procedure.

On insertion of the catheter, ITP in all 22 patients was 13.8 ± 1.3 mm Hg. After the spinal cord was directly decompressed (anterior discectomy or vertebrectomy, or posterior laminectomy), the ITP in all but 1 patient increased. The maximal extent to which the ITP increased was of particular interest, given that this peak would represent the point at which SCPP would potentially be at its lowest. The mean peak ITP documented intraoperatively after completion of the surgical decompression was 21.7 ± 1.5 mm Hg. This represented an increase of 7.9 ± 1.6 mm Hg over the initial ITP reading ($p < 0.0001$, paired t-test). The mean ITP over the course of the procedure following surgical decompression was 17.9 ± 1.2 mm Hg. This represented an increase of 3.9 ± 0.9 mm Hg over the initial ITP reading ($p < 0.0001$, paired t-test) (Fig. 1). For 1 patient (Case 4) whose ITP decreased after decompression, the initial ITP was already high when the catheter was inserted (23 mm Hg) and then decreased by 5 mm Hg. Interestingly, when the patient arrived in the recovery room, the ITP had increased to 27 mm Hg and then subsequently increased to as high as 40 mm Hg in the postoperative period.

The details of the surgical procedures and time to surgery are included in Tables 1 and 2. As is indicated in the tables, almost every patient underwent a direct surgical decompression of the cord, in addition to the indirect decompression achieved with realignment of the spinal column. The changes in ITP intraoperatively typically began after the surgeon identified that the cord decompression was complete and pulsations of the cord beneath the dura mater could be observed.

Changes in ITP Postoperatively

Intrathecal pressure recordings resumed when the patient arrived in the recovery room or the intensive care unit. Beginning from this time, the catheters were left in

TABLE 2: Patients randomized to the CSF drainage group: demographic, intraoperative, and postoperative data*

								Pre- & Intraop Period (mm Hg)			Postop Period (mm Hg)					ASIA Motor Score		
Case No.	Age (yrs), Sex	Mechanism of Injury	Spinal Injury	Site/ASIA Grade	% Canal Compromise	Surgery	Timing of Op (hrs postinjury)	Initial ITP	Peak ITP Postdecompression	Mean ITP	Mean ITP	Peak ITP	Mean MABP	Mean SCPP	Vol of CSF Drained (ml)	Baseline	6 Mos Postop	Segmental Recovery (cervical only)
3	47, M	MVA	T-3 burst fracture	T-3, B	66.1	pst T-3 laminectomy, T1–5 fusion	37	10	22	18.0	15.0	22	78.3	62.5	25	50	50	
5	64, F	mountain biking	C5–6 hyperflexion w/ spondylosis	C-5, C	47.2	pst C4–6 laminectomy, C4–7 fusion	26	10	28	19.0	16.1	28	82.8	66.6	14.5	37	96	59
8	37, M	MVA (struck by train)	T8–9 fracture dislocation	T-8, B	33.6	pst T5–11 fusion	22	14	27	13.5	20.6	33	89.4	69.2	0	50	50	
10	37, M	diving	C4–5 fracture dislocation	C-5, A	38.5	pst C4–6 laminectomy, C4–6 fusion	16	20	24	22.0	14.0	24	79.6	64.2	327	8	15	7
13	40, M	mountain biking	T3–4 fracture dislocation	T-3, A	74.2	pst T-3 laminectomy, T1–6 fusion	23	12	16	11.9	18.7	30	82.6	64.0	354	50	48	
16	31, F	MVA (rollover)	T-9 burst fracture	T-9, A	76.9	pst T8–9 laminectomy, T7–11 fusion	42	13	19	16.7	8.4	12	79.9	71.5	10	50	50	
17	45, F	fall down stairs	C5–6 fracture dislocation	C-6, A	26.6	ant C5–6 discectomy, C5–6 fusion	22.5	14	23	18.6	11.5	14	88.5	76.6	129	23	26	3
18	46, F	fall off balcony	C-5 burst fracture	C-4, A	56.6	ant C5–6 vertebrectomy, C4–7 fusion	11.5	18	31	25.5	23.0	33	83.6	60.6	29	4	8	4
20	60, M	fall from ladder	C5–6 facet subluxation	C-5, B	7.5	pst C3–6 laminectomy, C3–7 fusion	19	14	19	16.4	19.1	35	89.2	68.7	42	7	58	51
24	27, M	mountain biking	C5–6 fracture dislocation	C-4, A	35.9	ant C-5 vertebrectomy, C4–6 fusion	13.5	11	23	19.3	17.7	36	82.1	64.4	314	6	11	5
11	55, F	fall from moving car	T9–10 fracture dislocation	T-9, B	39.8	pst T9–10 laminectomy, T6–12 fusion	24				26.8	42	79.5	52.7	51	50	50	
	44.5 ± 2.4			6 cervical, 5 thoracic: 6 A, 4 B, 1 C	45.7 ± 6.4		23.3 ± 2.8	13.6 ± 1.0	23.2 ± 1.4	18.1 ± 1.2	17.4 ± 1.6	28.1 ± 2.8	83.2 ± 1.2	65.6 ± 1.9	117.8 ± 42.8	30.5 ± 6.3	42.0 ± 7.7	21.5 ± 7.9

* Except for Site/ASIA Grade category, all values are presented as the mean ± SEM.

place for an average of 56.3 ± 4.5 hours. In patients randomized to CSF drainage, the drainage was initiated once their neurological status could be monitored. The patients randomized to the drainage or no-drainage group were analyzed as distinct groups during this postoperative period, given that this was the time during which drainage was initiated.

During this postoperative period, the ITP in patients from both arms of the study generally decreased, but in both arms there were hours where the ITP transiently increased even beyond the peak ITP that was observed intraoperatively. In the patients randomized to the no-drainage group, the mean peak recorded ITP during the postoperative period was 30.6 ± 2.3 mm Hg. In each patient this represented, on average, an increase of 10.3 ± 2.8 mm Hg over the peak ITP recorded intraoperatively ($p = 0.0047$, paired t-test). In the patients randomized to undergo CSF drainage, the peak recorded ITP during the postoperative period was 28.1 ± 2.8 mm Hg, which was not significantly higher than the peak ITP recorded intraoperatively ($p = 0.15$, t-test). Excluding the 1 patient in this drainage group whose intraoperative CSF pressures were lost, the peak recorded ITP postoperatively was 26.7 ± 2.7 mm Hg, which was again not significantly higher than the peak intraoperative postdecompression ITP ($p = 0.23$, paired t-test) (Fig. 1).

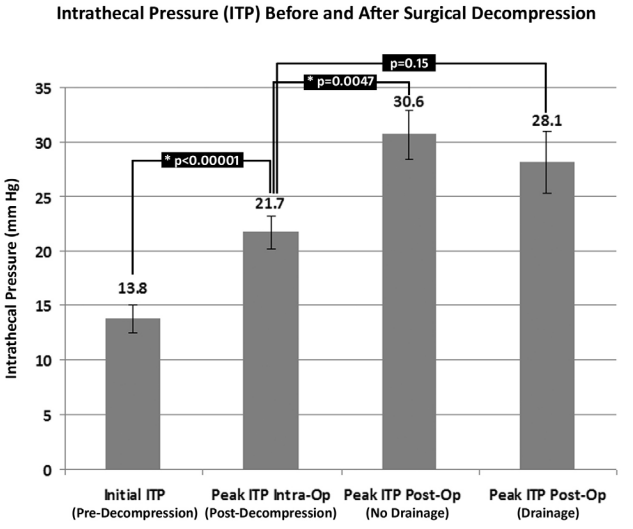

Fig. 1. Bar graph showing changes in ITP. In all 22 patients, the mean ITP was 13.8 ± 1.3 mm Hg. Intraoperatively, after the surgical decompression was completed, the ITP increased significantly. The mean "peak" ITP recorded intraoperatively after decompression was 21.7 ± 1.5 mm Hg, a statistically significant increase over the initial ITP ($p < 0.00001$). In the postoperative period, CSF drainage was initiated. In patients randomized to the no-drainage group, the mean peak ITP recorded was 30.6 ± 2.3 mm Hg, which was significantly higher than the intraoperative peak ITP ($p = 0.0047$). In patients randomized to receive drainage, the peak ITP recorded was 28.1 ± 2.8 mm Hg, which was not statistically higher than the peak intraoperative ITP ($p = 0.15$). *Asterisk* indicates a significant difference.

The Volume of CSF Drained

The volume of CSF drained during the postoperative period in patients randomized to the CSF drainage group was 117.8 ± 42.8 ml. In many patients, this drainage was limited by the predefined rules of the study protocol, which did not permit CSF drainage during times when the patients were not clinically examinable. Such periods of time were frequent during the postoperative period due to pharmacological sedation. Additionally, the protocol we established did not permit > 10 ml of CSF to be drained per hour.

Spinal Cord Perfusion Pressure During Postoperative Monitoring

Over the course of the postoperative period, the mean SCPP was 65.5 ± 1.9 mm Hg in the CSF drainage group and 58.7 ± 3.3 mm Hg in the no-drainage group. This increase in SCPP in the drainage group trended toward statistical significance ($p = 0.084$) with a 2-tailed t-test and was statistically significant with a 1-tailed t-test ($p = 0.042$) (the latter being reasonable given the expectation that draining CSF would only increase, and not decrease the SCPP). During this time, the mean MABP was 83.2 ± 1.2 mm Hg in the drainage group and 77.5 ± 2.8 mm Hg in the no-drainage group ($p = 0.076$, t-test). The mean ITP was 17.4 ± 1.6 and 18.7 ± 1.4 mm Hg in the drainage and the no-drainage groups, respectively.

Working under the assumption that ITP was influenced by CSF drainage and MABP was influenced by other clinical factors such as the amount of inotropic drugs or intravenous fluids given over the course of the postoperative period, we statistically modeled the relationship between the SCPP and the treatment (drainage vs no drainage), time, and MABP. This revealed a significant relationship between MABP and SCPP, with an estimated 1.02 ± 0.01 mm Hg increase in SCPP for every 1.0–mm Hg increase in MABP (95% CI 0.9916–1.0478, $p < 0.0001$). The relationship between treatment (drainage vs no drainage) and SCPP was not statistically significant, with an estimated 2.5 ± 2.0–mm Hg increase in SCPP in the drainage group compared with the no-drainage group (95% CI –1.41 to 6.49, $p = 0.2077$). In general, the SCPP increased in all groups over time, with an estimated 0.062 ± 0.28–mm Hg increase per hour (95% CI 0.008–0.116, $p = 0.0245$) (Fig. 2).

When examining the ITP, MABP, and SCPP over time (Fig. 2), we observed that during the first 36–40 hours postoperatively, the ITPs were similar between the drainage and no-drainage groups, but after 40 hours, the ITP was lower in the patients undergoing CSF drainage. Conversely, during the first 40 hours postoperatively, the MABP in the drainage group appeared to be higher than that in the no-drainage group. Hence, the increased SCPP in the drainage group seemed to be most attributable to the increased MABP during the first 40 hours and then more attributable to the decreased ITP after that. We therefore modeled the effect of treatment arm and MABP on the SCPP by using the generalized estimate equation before and after the 40th postoperative hour. Indeed, in the first 40 hours, the effect of changes in MABP on the SCPP was significant, such that for every 1–mm Hg in-

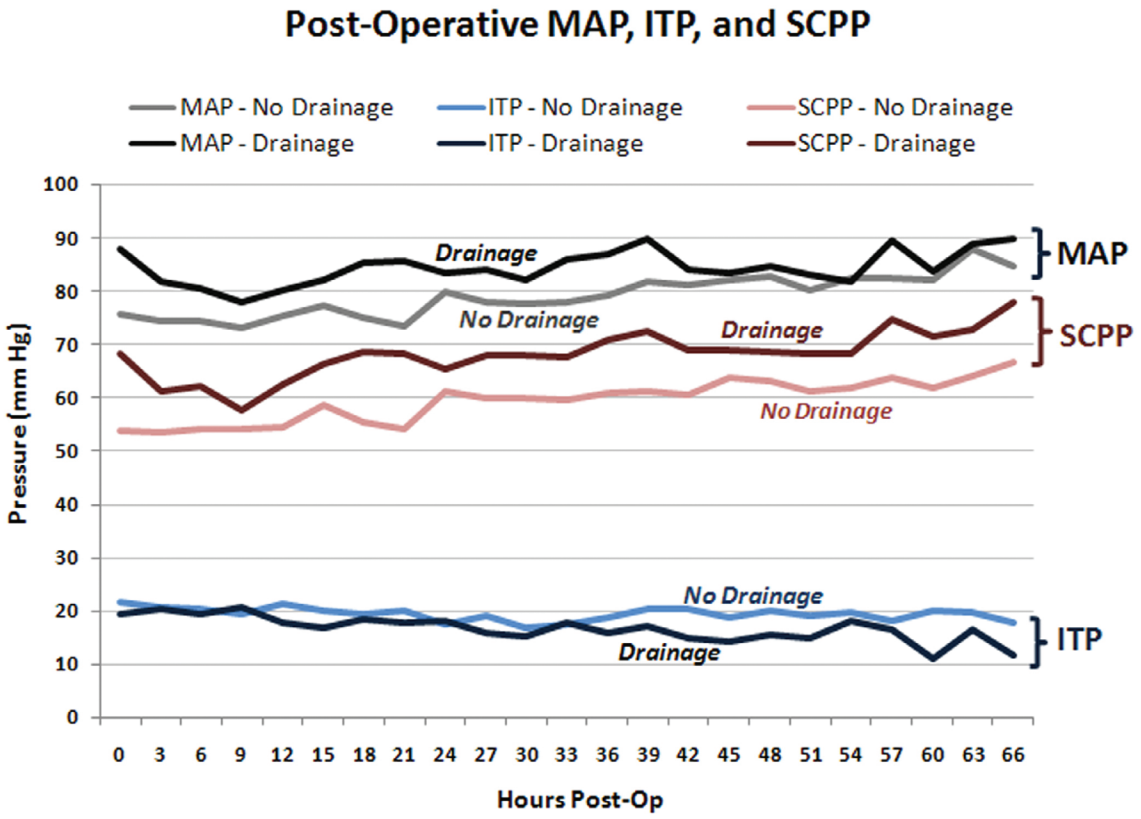

FIG. 2. Line graph demonstrating changes in MABP (MAP), ITP, and SCPP during the postoperative course. Here, the MABP, SCPP, and ITP of the drainage and the no-drainage groups are plotted over time, beginning with when the patients arrived in the recovery room or intensive care unit postoperatively. The SCPP was consistently higher in the drainage group, likely attributable to the higher MABP early on, and lower ITP in the drainage group in the latter stages.

crease in MABP there was a 0.96 ± 0.04–mm Hg increase in SCPP ($p < 0.0001$). However, the drainage group had only a 2.00 ± 2.00–mm Hg increase in SCPP ($p = 0.334$). After 40 hours, the effect of drainage doubled, such that the drainage group had a 4.51 ± 2.5–mm Hg increase in SCPP ($p = 0.077$). The effect of MABP on SCPP was still prominent, with a 1.00 ± 0.3–mm Hg increase for each mm Hg increase in MABP ($p < 0.0001$).

It is unclear why the patients in the CSF drainage group had a higher MABP early in the postoperative course. Because the CSF drainage group had 4 thoracic cord injuries whereas the no-drainage group was composed entirely of cervical cord injuries, we considered whether less severe neurogenic shock in the patients with thoracic injuries might bring the mean MABP up in the CSF drainage group. However, the mean MABPs over the postoperative period of the thoracic and cervical patients of the drainage group were very similar (84.1 vs 82.7 mm Hg, respectively).

We also considered the possibility that a relationship existed between MABP and ITP that might influence how one clinically managed the MABP. Specifically, we were interested in knowing if the transient peaks in ITP (mean 30.6 ± 2.3 mm Hg in the no-drainage group and 28.1 ± 2.8 mm Hg in the drainage group) were also associated with increases in MABP that would attenuate the effect of increased ITP on the SCPP. The Pearson correlation coefficient between MABP and ITP was estimated at each hour and was found to be, in most cases, negative. This indicates that increases in ITP were not associated with increases in MABP, and, if anything, the negative coefficients indicate that the ITP increases were more commonly associated with decreases in MABP. Hence, the peaks in ITP that we observed in the postoperative course are potentially associated with periods of relative ischemia for the spinal cord, as reflected by a decrease in the SCPP (Fig. 3).

Cerebrospinal Fluid Pressure Waveform

The other striking qualitative observation from this cohort of patients was the CSF waveform pattern recorded by the ITP transducer system. Normally, the CSF waveform is pulsatile, reflecting arterial pulsations within the ventricles. When the catheters were inserted (prior to surgical decompression), we noted that in all patients the CSF waveform pattern was flat, with no pulsations. We noted a uniformly flat waveform in all patients upon insertion of the CSF catheter. After direct surgical decompression, the CSF waveform typically changed dramatically (in addition to the increase in pressure). Postdecompression,

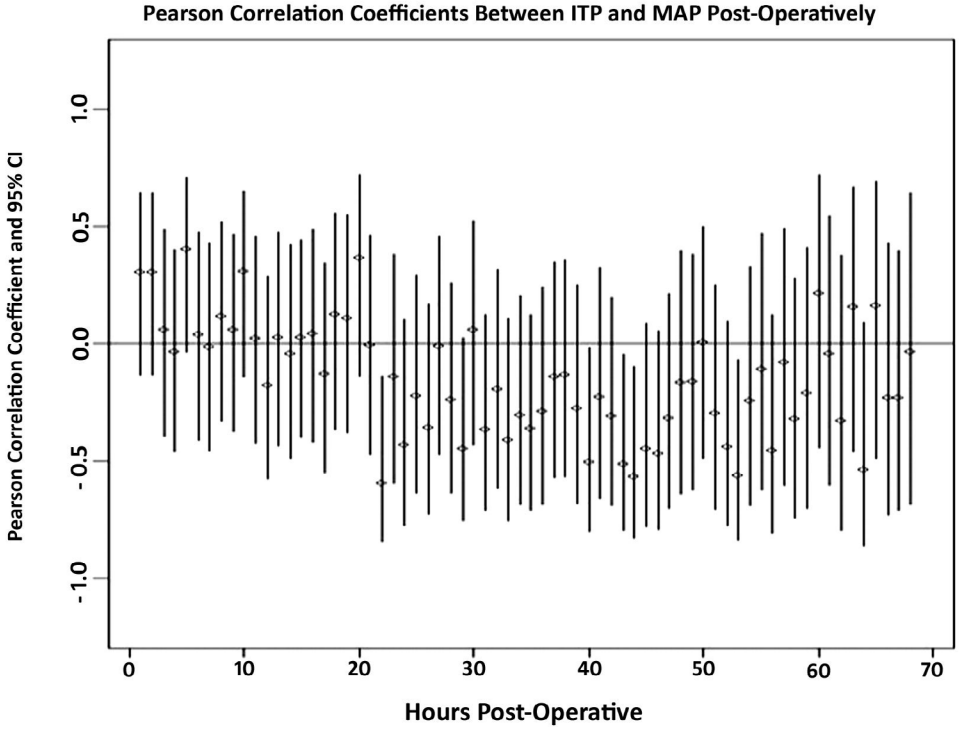

Fig. 3. Graph showing the Pearson correlation coefficients between MABP and ITP during the postoperative course. To establish whether changes in MABP and ITP were related and, more specifically, if transient peaks in ITP were associated with similar increases in MABP, the Pearson correlation coefficients and the 95% CIs between MABP and ITP were plotted at each hour postoperatively. In general, the correlation coefficients are low and, if anything, are more frequently negative than positive, suggesting that transient increases in ITP are not associated with increases in MABP that would attenuate the lowering of SCPP.

the intrathecal waveform became pulsatile again, temporally matching the pulsations of the electrocardiograph and arterial pressure recordings (Fig. 4). These findings strongly suggest that prior to surgical decompression, the flow of CSF across the injury site is restricted by the combination of spinal deformity, displaced bone, or disc in the spinal canal, as well as by swelling of the spinal cord. In keeping with this, the maximal extent of spinal canal occlusion was ~ 47%, as measured off the midsagittal image on the preoperative CT or MR image using the technique described by Fehlings and colleagues.[5,7] With this degree of canal occlusion, the intrathecal space clearly appears to be occluded on the MR imaging, indicating that CSF flow across the injury site is restricted.

Additionally, we did observe in a number of patients that, during the first 48 hours in the postoperative period, their pulsatile waveform reverted back to the flat pattern that was observed at the time of catheter insertion. At this stage MR imaging revealed ongoing compression of the thecal sac, typically in the presence of an epidural hematoma (Fig. 5). In the absence of neurological deterioration, however, these findings were managed without further surgical intervention. They did, however, suggest that the presence of a pulsatile CSF pressure waveform is reflective of a fully decompressed spinal cord and restoration of CSF flow around the cord and "continuity" of the intrathecal space across the injury site. Conversely, a flattened waveform without pulsations is reflective of CSF flow blockage across the injury site, which could be caused by ongoing extrinsic compression upon the thecal sac from bone, disc, or epidural blood, or by intrinsic swelling of the cord filling to occupy the intrathecal space (or a combination).

Motor Recovery

Although the primary objective of this study was not to assess the neurological efficacy of CSF drainage, an examiner blinded to the randomization did compare ASIA motor grades at the time of arrival and at 6 months postinjury. Unfortunately, we did not stratify the findings according to injury level (cervical or thoracic), and by chance all 11 patients in the no-drainage group had cervical SCI (1 patient had a thoracic injury but his drain fell out early and he was not included). In the no-drainage group, there were 5 patients with thoracic injuries, and 6 with cervical injuries. Comparing the segmental motor recovery of patients with cervical injuries only, there was no significant difference in the motor score change at 6 months between those randomized to receive drainage and no drainage (21.0 ± 10.8 vs 15.4 ± 5.5 motor points, respectively), although our study was most certainly not powered sufficiently to assess the effect of CSF drainage on neurological recovery.[4] The similar motor scores at least suggest that there was no detrimental or adverse effect of CSF drainage on neurological outcome.

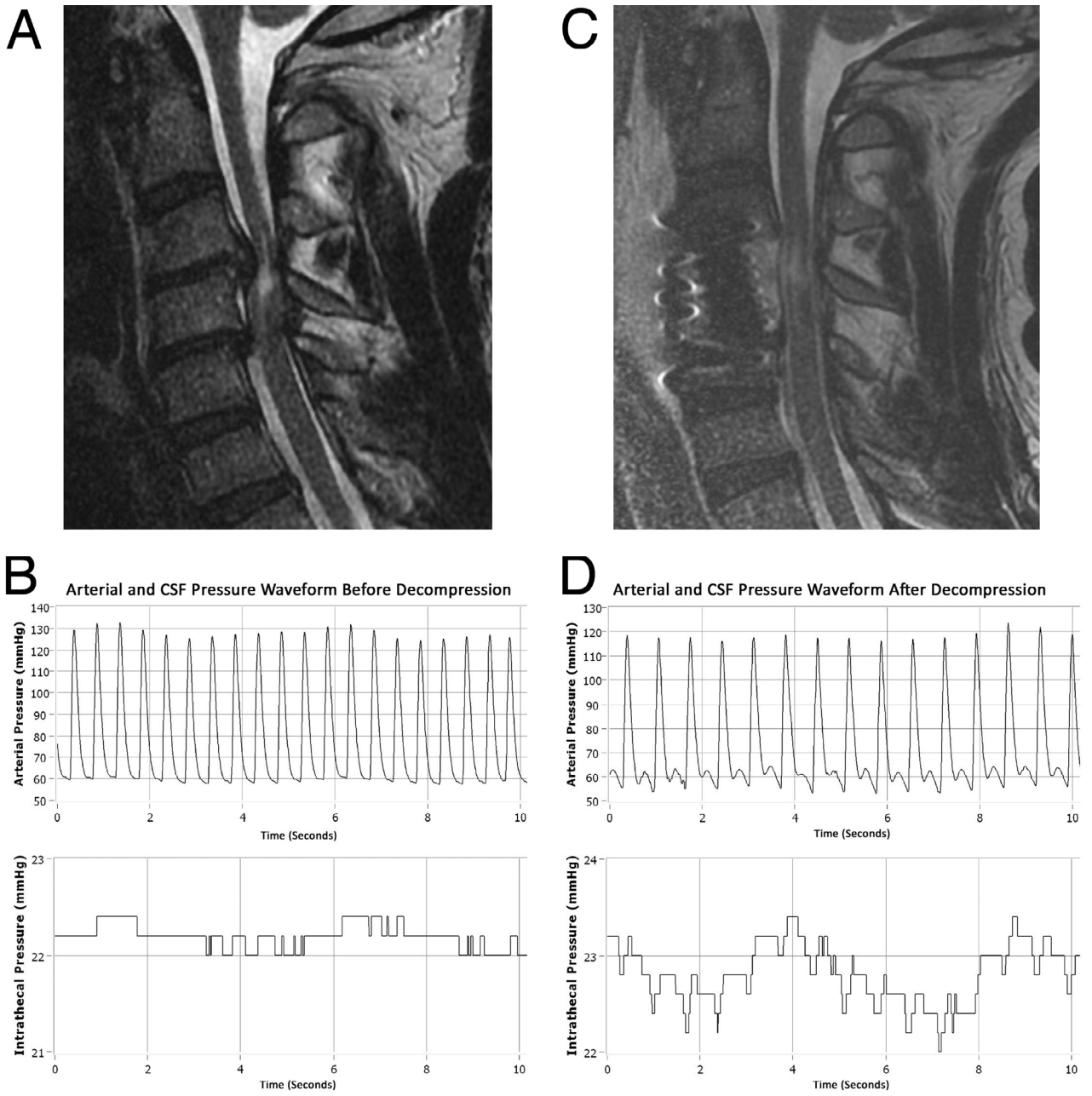

FIG. 4. Sagittal MR images and related ITP waveform before and after surgical decompression. A: Prior to surgical decompression, the thecal sac is compressed and CSF flow is blocked at the site of injury. Ventricular pulsations are therefore not transmitted across the injury site to the intrathecal catheter, which is positioned in the lumbar spine. B: The CSF waveform is essentially flat and does not change with respiration. Following decompression, continuity of the subarachnoid space has been restored above and below the injury site (C) and the CSF waveform (D) becomes pulsatile and undulates according to respiration. Note that the CSF pulsations match the arterial pulse waveform.

Discussion

While the drainage of CSF to lower ITP and improve SCPP is a common neuroprotective practice in thoracoabdominal aortic aneurysm repair, its application in acute traumatic SCI, to the best of our knowledge, has not been previously described. The primary goal of this prospective randomized study was to evaluate the practice of CSF drainage in patients with acute traumatic SCIs. Specifically, we were interested in demonstrating whether this was an effective method for reducing ITP and, hence, potentially improving perfusion to the spinal cord. The randomized design was chosen to help in interpreting the contribution of CSF drainage to the changes in intrathecal (and hence spinal cord perfusion) pressure, and to the adverse events. We observed no major adverse events specifically linked to the catheter insertion or drainage. In particular, no patients suffered symptoms of a CSF

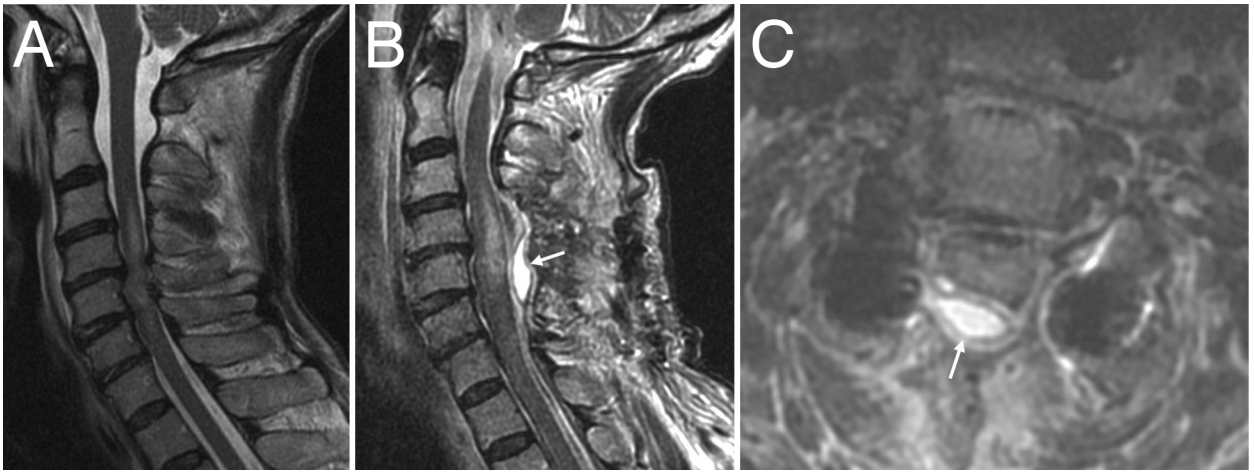

FIG. 5. Magnetic resonance images obtained in a 64-year-old woman who suffered an incomplete cervical SCI after falling from a bicycle and hyperextending her neck. A: Preoperative MR image revealing diffuse stenosis and signal change. Note that the thecal sac appears occluded from C4–5 to C5–6. Her waveform was flat and CSF pressure was 10 mm Hg upon insertion of the drain, and so despite being randomized to the CSF drainage group, the patient did not undergo drainage. Rather, she underwent a posterior laminectomy and fusion, during which her CSF pressure increased from 10 to 28 mm Hg. Her CSF waveform became pulsatile in keeping with the surgeon's direct visualization of the dural pulsations after the posterior laminectomy was completed. On postoperative Day 2 the pulsatile nature of her waveform diminished. B and C: Postoperative MR imaging revealed increased swelling and edema of the spinal cord, as well as a small posterior epidural hematoma compressing the thecal sac *(arrow).*

leak, neurological deterioration, or meningitis while the catheter was in place, and hence, there was no difference in these rates of adverse events between patients randomized to the drainage or no-drainage group.

As for the approach of CSF drainage in this population of patients, with our current protocol we did not achieve a significant decrease in the ITP in the patients randomized to CSF drainage. The study protocol mandated that drainage be ceased during periods in which clinical examination was not possible, for fear of neurological deterioration going unnoticed during the decreasing of ITP. Neurological deterioration after lumbar puncture in the presence of subarachnoid space occlusion due to malignancies is a well-recognized complication,[10] although we were unaware of any reports of such a complication in the setting of acute trauma. Hence, we were extremely conservative with the drainage protocol, which ultimately undoubtedly contributed to the low volume of CSF that we drained and the minimal effect on ITP. It was evident in the acute postoperative period that such periods during which formal neurological examination was impossible were quite frequent, particularly in the patients with cervical quadriplegia who often required ongoing mechanical ventilation (and sedation) postoperatively. Hence, in some patients the volume of CSF drained was very small (in 1 patient, no CSF was drained). Additionally, we treated a number of patients in whom, when the drain was opened for drainage, the 10 ml of CSF poured out within a few minutes, forcing us to close off the drain due to the drainage limit of 10 ml/hour that we had imposed. This likely contributed to the ineffective lowering of ITP in patients randomized to CSF drainage, particularly in the early stages postoperatively during which many of the patients remain sedated from their surgery and/or ongoing mechanical ventilation. Given our experience with this technique and the lack of observed adverse events, one might consider a clinical protocol by which CSF drainage was allowed to occur either during periods when the patients were somewhat sedated or with a higher hourly limit to more purposefully reduce the ITP.

With respect to evaluating changes in ITP after surgery and in the postoperative period, we were somewhat surprised by our findings. We initially hypothesized that the ITPs would be elevated upon insertion of the catheter and speculated that surgical decompression would decrease ITP in addition to relieving direct mechanical impingement on the cord. On both accounts, we found exactly the opposite. The ITPs were, on average, below 14 mm Hg on drain insertion, and they then increased significantly after surgical decompression. Such increases in ITP lower SCPP and therefore may add to the contribution that parenchymal ischemia makes in the pathophysiology of secondary damage after acute SCI.

One could alternatively argue that if a large pressure gradient exists across the occluded thecal sac at the injury site (with a very high pressure system rostral and a low-pressure system caudal to the injury), then the surgical decompression serves to relieve the high intrathecal and low SCPP that is endured by the spinal cord parenchyma proximal to the occluded thecal sac. Unfortunately, safely obtaining direct ITP recordings proximal to the SCI would be difficult, unless there was a concomitant head injury requiring extraventricular drainage. Such patients were clearly excluded from our experimental protocol, which required the patients to be able to undergo a neurological examination.

The changes in ITP, as measured with our lumbar intrathecal catheter, suggest to us that the compression upon the spinal cord and thecal sac caused by the malalignment of the spinal canal or displaced bone and disc fragments

occludes the intrathecal space and obstructs CSF flow, thereby establishing a fairly significant pressure gradient across the injury site. The mechanical occlusion of the intrathecal space is suggested by the high degree of canal occlusion on preoperative imaging studies (almost 50%) and the consistently flat CSF pressure waveform upon insertion of the drain. Decompression may relieve this occlusion, and the high-pressure system within the thecal sac proximal to the injury site is resolved into the distal thecal sac where the catheter is placed, causing a rise in the ITP very quickly after the decompression is achieved. It is also possible that the restoration of CSF flow after surgical decompression of the thecal sac may be attenuated or blocked altogether by swelling of the cord itself if the cord expands to fill the intrathecal space.

Both the rise in ITP that occurred after surgical decompression and the fact that further increases were noted in many patients during the 72 hours when the drain was active have important implications for the acute management of the spinal cord injured patient. For many years it has generally been accepted that clinicians treating acute SCI should endeavor to maintain a high MABP and, at the very least, avoid systemic hypotension.[8] Recommendations have therefore been made to keep the MABP above 85–90 mm Hg,[13,19] although this in some cases mandates the use of substantial doses of inotropic agents (the effects of which are not benign either). At our institution, consensus is lacking among the surgical, anesthesiology, and intensive care staff about what the target level at which MABP should absolutely be maintained. Hence, in patients who are being treated with vasopressors and intravenous fluids and in whom urine output is good but MABP falls short of 85 to 90 mm Hg, the decision is sometimes made to not give additional vasopressors. This aspect of the clinical management was not controlled, as it was assumed that randomization would make irrelevant the differences among patients. Unfortunately, for reasons that were not clear, the patients randomized to the CSF drainage group had higher MABPs than those randomized to the no-drainage group. Given the small sample size, this difference (which trended toward significance) was probably spurious.

Whatever one chooses for a "target MABP", there is a tacit assumption that this will provide "adequate" perfusion to the spinal cord, without any consideration for the ITP. Considering that the SCPP is the calculated difference between the MABP and the ITP, it follows that a more meaningful interpretation of the perfusion to the cord should take into consideration the ITP. To this date, we have not previously seen any documentation of the ITPs in patients with acute spinal cord injuries, and how these pressures change in the acute perioperative period.

Our observation of this ITP increase indicates that the perfusion to the spinal cord may actually decrease after the spinal decompression is performed (assuming a constant MABP), and may transiently decrease even further in the acute postoperative period. While the exact clinical significance of this is unknown, it seems only logical that this fact be considered in the management of systemic blood pressure, one of the few controllable clinical parameters that is thought to influence neurological function after SCI. In the setting of traumatic brain injury, intracranial pressure monitoring and strict attention to hemodynamic support are widely considered to be important in influencing clinical outcome.[17] It is recognized that the SCPP is merely a calculated entity based on the measured MABP and ITP. Hence, it is only a gross representation of the true spinal cord perfusion that, at a microvascular level, will almost certainly differ in different regions of the injured spinal cord. Nonetheless, from a clinical perspective, it is only the SCPP that can be calculated based on readily measurable parameters, and hence, it is relevant to consider how it might be monitored in the acute postinjury phase for patients with traumatic SCIs.

The other somewhat unexpected finding in this study is the waveform changes. In the first 2 patients we enrolled, the flat waveform prompted us to search for technical problems with the catheter or pressure transducer, but it soon became clear that the flat waveform pattern frequently became pulsatile after the surgical decompression. These pulsations, which are otherwise identical to those seen when monitoring intracranial pressure, suggest that, when they are present, the patency of the intrathecal space from the cranium to the lower lumbar spinal canal has been restored. In cases in which the pulsatile waveform reverted to a flat waveform, MR imaging revealed that the intrathecal space was again occluded, further strengthening the interpretation of these observations.

The relevance of a patent intrathecal space is speculative to some extent. We do not contend that obstruction to CSF flow due to thecal sac compression necessarily represents ongoing spinal cord compression, although occlusion of the thecal sac would be expected to occur in most cases if encroachment on the spinal canal were sufficient to compress the spinal cord parenchyma. Alterations and obstructions in CSF flow due to pia-arachnoid scarring have been postulated to be etiologically related to syrinx formation,[12,16] and so one could hypothesize that failing to restore the subarachnoid space around the injured spinal cord might predispose to this late complication. Swelling of the spinal cord within the dural sac of an otherwise realigned and decompressed spinal canal may also result in such obstruction of CSF flow, and it is possible that once the swelling subsides, CSF flow would be restored. We did not perform MR imaging to demonstrate whether the subarachnoid space and CSF flow were restored in the subsequent weeks to months postinjury.

In summary, we found that the installation of an intrathecal catheter and drainage of CSF were safe in patients with acute traumatic SCIs, although our population was small and only a limited amount of CSF was drained. While we did not observe a significant lowering of the ITP compared with that in patients who did not undergo CSF drainage, we believe that the parameters of our clinical trial protocol, which limited the amount and the times of CSF drainage, likely contributed to this. Our observations of the rise in ITP postdecompression suggest that a large pressure gradient exists across the injured spinal cord prior to decompression. The postdecompression rise in ITP indicates a drop in SCPP in the setting of a constant MABP, an observation that should be considered in the perioperative management of blood pressure in these

patients. Such monitoring of ITP is routinely performed in patients with traumatic brain injury and is used to guide clinical decision making with respect to hemodynamic resuscitation. Given the perceived importance of ischemia in the pathophysiology of secondary damage after acute traumatic SCI and the lack of available clinical measures outside of surgical decompression and blood pressure management to influence neurological outcome, it would seem reasonable to consider such ITP monitoring in the clinical care of these patients. Our study was not powered to establish the neurological efficacy of CSF drainage, and given the limited CSF that we were actually able to drain (and minimal overall effect on the ITP), we would need to consider modifying the drainage parameters of the protocol prior to considering a larger scale trial.

Conclusions

A prospective randomized clinical trial was conducted in patients with acute SCI to evaluate the role of CSF drainage and to measure ITPs in the acute injury phase. With the CSF drainage protocol that was instituted (which did not allow drainage during times when the patients' neurological function could not be measured), a significant reduction in ITP was not achieved. However, significant increases in ITP were observed intra- and postoperatively after spinal decompression, with a decrease in SCPP. Also, the CSF pressure waveform pattern was found to be useful for documenting decompression of the spinal cord and restoration of CSF flow around the injury site. These findings, and in particular the substantial increases in ITP postsurgery, indicate that a more sophisticated approach to hemodynamically managing acute SCI could entail the use of CSF pressure monitoring, as is done in patients with severe traumatic brain injury.

Disclosure

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