Prevention, identification, and treatment of perioperative spinal cord injury

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Object. In this report, the authors suggest evidence-based approaches to minimize the chance of perioperative spinal cord injury (POSCI) and optimize outcome in the event of a POSCI.

Methods. A systematic review of the basic science and clinical literature is presented.

Results. Authors of clinical studies have assessed intraoperative monitoring to minimize the chance of POSCI. Furthermore, preoperative factors and intraoperative issues that place patients at increased risk of POSCI have been identified, including developmental stenosis, ankylosing spondylitis, preexisting myelopathy, and severe deformity with spinal cord compromise. However, no studies have assessed methods to optimize outcomes specifically after POSCIs. There are a number of studies focussed on the pathophysiology of SCI and the minimization of secondary damage. These basic science and clinical studies are reviewed, and treatment options outlined in this article.

Conclusions. There are a number of treatment options, including maintenance of mean arterial blood pressure > 80 mm Hg, starting methylprednisolone treatment preoperatively, and multimodality monitoring to help prevent POSCI occurrence, minimize secondary damage, and potentially improve the clinical outcome of after a POSCI. Further prospective cohort studies are needed to delineate incidence rate, current practice patterns for preventing injury and minimizing the clinical consequences of POSCI, factors that may increase the risk of POSCI, and determinants of clinical outcome in the event of a POSCI. (DOI: 10.3171/FOC.2008.25.11.E15)

KEY WORDS • complication • review • spinal cord injury

PERIOPERATIVE SCI is one of the most feared complications of spine surgery, potentially resulting in a devastating and debilitating outcome for the patient and tremendous stress for the surgeon. Fortunately, the incidence of POSCI is relatively uncommon with an estimated incidence varying from 0 to 3%, depending on the pathological entity treated, the spinal level, and surgical approach.^{25,28} Perioperative SCI involves a direct or indirect physiological insult to the spinal cord during immediate preparation for surgery, intraoperatively, or immediately postoperatively. This physiological insult leads to neuronal/axonal dysfunction or disruption, and consequently to motor, sensory, and/or autonomic impairment. Injury to the spinal cord may be complete or incomplete, and can result in temporary or permanent impairment. Numerous trials of pharmacological agents for traumatic

SCI have been performed, with some trials showing a potential benefit,^{7–11,30,31,65} however, no reviews exist in the literature concerning the causes and management of POSCIs. The lack of a formal protocol for management of POSCI is the impetus for this paper, which will review the pathophysiology and causes of POSCI, and then propose evidence-based recommendations for the prevention of and treatment of POSCI based on a systematic review of basic science and clinical studies.

Methods

Searches were performed on Medline (1966 to June 2008), Embase, and the Cochrane Collaboration. Hand searches were also performed in the *European Spine Journal*, *Spine*, and *Journal of Neurosurgery: Spine*, along with bibliography searches of pertinent studies. Search strategy for Medline were: “exp Spinal Cord Injuries/” and “Intraoperative Complications/ or Postoperative Complications/,” limited to the English language. Each of these searches produced 13185 and 94174 articles, respectively. Combining the searches resulted in 493 articles, and review of the articles and abstracts lead to 84 relevant articles.

Abbreviations used in this paper: CSF = cerebrospinal fluid; MABP = mean arterial blood pressure; MEP = motor evoked potential; MPSS = methylprednisolone sodium succinate; NASCIS = National Acute Spinal Cord Injury Study; OPLL = ossification of the posterior longitudinal ligament; POSCI = perioperative spinal cord injury; SCI = spinal cord injury; SSEP = somatosensory evoked potentials.

TABLE 1. Outline of treatment options minimizing the incidence of a POSCI

Options
1. Maintain MABP > 80 mm Hg including during induction
2. Start MPSS in the holding area using NASCIS II dosing and scheduling
3. Maintain spinal control/precautions
4. Use adequate lighting, magnification, and assistance
5. Multimodality intraoperative neuromonitoring with SSEP/MEP/EMG
6. Careful application of distractive forces and recognition if applied, that this can cause a stretch injury/ischemic injury to the spinal cord
7. Careful decompression with appropriate instrumentation to avoid trauma to neural structures

Articles were included if they discussed methods to prevent the occurrence of or minimize the effects of a POSCI, the pathophysiology of SCIs, or methods to minimize the effects of secondary damage after SCI. Perioperative SCI encompasses SCI or clinical worsening of SCI just prior to, during, and after surgery. Articles included both basic science and clinical studies.

We present an overview of the pathophysiology of SCI, presurgical causes of SCI, intraoperative causes of SCI in the cervical and thoracic spine, postoperative causes of SCI, and long-term management of POSCIs. Following the overview are 2 case examples in which recommendations to prevent a POSCI were used (Table 1); in the second case recommendations for the treatment of perioperative spinal cord complications once they have occurred are discussed (Table 2). Evidentiary tables are provided that summarize both the basic science and clinical literature (Tables 3–5).

Results

Pathophysiology of SCI

Spinal cord injury is the result of a 2-step process that has both primary and secondary mechanisms.^{69,79} The primary mechanism involves the initial injury, which may include compression, impaction, laceration, shearing, distraction, or ischemia. These mechanisms may occur at any point from the time of surgical planning, to the time of anesthesia, to intraoperatively, and in the

TABLE 2. Outline of treatment options minimizing the effects of a POSCI once it has occurred

Options
1. Prompt recognition of SCI
2. If possible, prompt reversal of offending cause of SCI
3. Maintain MABP > 80 mm Hg and correct hematocrit
4. Start MPSS in the holding area using NASCIS II dosing and scheduling
5. Stabilize the spinal region if it is unstable
6. HIGHLY CONTROVERSIAL: systemic cooling and CSF drainage

TABLE 3: Summary of basic science and clinical papers reviewed for the pathophysiology of acute SCI and presurgical causes and preoperative management of acute SCIs

Authors & Year	Data Class	Study Type
Kobrine et al., 1976		basic science
Rydevik & Lundborg, 1977		basic science
Kobrine et al., 1979		basic science
Senter & Venes, 1979		basic science
Tew & Mayfield, 1980	IV	nonsystematic review
Young et al., 1980		basic science
Demopoulos et al., 1982		basic science
Dahlin et al., 1984		basic science
Epstein et al., 1984	IV	case series
Bracken et al., 1985	I	randomized control trial (RCT)
Fehlings et al., 1989		basic science
Bracken et al., 1990	I	RCT
Tator & Fehlings, 1991		basic science
Geisler et al., 1991	I	RCT
Laroche et al., 1992	III	case control
Bracken et al., 1992	I	RCT
Hall, 1993		basic science
Naso et al., 1995		basic science
Vale et al., 1997	II	prospective study
Gerndt et al., 1997	III	case-control
Petitjean et al., 1998	I	RCT
Gisselsson et al., 1999		basic science
Sauerland et al., 2000	I	systematic review of RCTs
Sekhon & Fehlings, 2001	III	review of epidemiology and basic science
Bracken, 2001	I	systematic review of Level I evidence
Nakao et al., 2001		basic science
Geisler et al., 2001	I	RCT
Molano et al., 2002	III	case-control
Lee et al., 2007	III	case-control
Suberviola et al., 2008	III	case-control

immediate postoperative period. The secondary cascade includes a complex series of biochemical and cellular processes, initiated by the primary processes, which potentially cause further cell damage and death. This cascade of events includes: 1) vascular changes with loss of autoregulation, decreased blood flow, and hemorrhage; 2) ionic derangements, including elevated intracellular calcium, sodium, and potassium; 3) neurotransmitter accumulation, including glutamate and catecholamines; 4) free radical production and lipid peroxidation; 5) edema; 6) inflammation; and 7) apoptosis. Once complete disruption of the neural elements has occurred, the possibility of recovery is minimal, regardless of treatment. As a result, the best possible method for minimizing the effects of POSCI is through prevention and understanding the various potential causes from the time of surgical planning,

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TABLE 4: Summary of basic science and clinical papers reviewed for the surgical causes and intraoperative management of acute SCIs

Authors & Year	Data Class	Study Type
Dommissie, 1974		basic science
MacEwan, 1975	III	survey
Flynn, 1982	IV	case series
Emery et al., 1988	IV	case series
Fessler et al., 1988	IV	case series
Bertalanffy & Eggert, 1989	IV	case series
Graham et al., 1989	IV	case series
Tator & Fehlings, 1991	IV	case series
Fielding, 1992	IV	case series
Davison et al., 1994	IV	case series
Faciszewski et al., 1995	IV	case series
Graham et al., 1996	II	prospective cohort
Winter, 1997	V	nonsystematic review
Hoppenfeld et al., 1997	I	prospective, diagnostic testing
Dickman & Sonntag, 1998	II	prospective cohort
Albert et al., 1998		case series
Koscielniak-Nielsen et al., 1998	I	RCT
Maliszewski et al., 1999		basic science
Abumi et al., 2000	IV	case series
Haid et al., 2001	IV	case series
Harms & Melcher, 2001	IV	case series
Suk et al., 2001	IV	case series
Tabayashi et al., 2002	IV	case series
Deen et al., 2003	II	prospective cohort
Husted et al., 2003		basic science
Krassioukov et al., 2004	II	prospective, diagnostic criteria development
Gunnarsson et al., 2004	II	retrospective cohort
Tabayashi & Tabayashi, 2005		review article
Orchowski et al., 2005	IV	case series
Inoue et al., 2005	II	prospective cohort
Kwon et al., 2008	II	review article

to the implementation of anesthesia, to the operation and immediately afterwards. Moreover, because the secondary injury events after neurotrauma evolve over minutes to hours, early recognition of POSCI is critical so that appropriate treatment can be instituted with the hope of minimizing the secondary injury cascade.

Presurgical Causes of POSCI

Although not necessarily the cause of SCI, a delay in recognizing or an inappropriate delay in surgical treatment for acute compression of the cervical or thoracic spinal cord can lead to progression of SCI while awaiting

TABLE 4: Summary of basic science and clinical papers reviewed for postsurgical causes and postoperative management of acute SCIs

Authors & Year	Data Class	Study Type
Miyamoto et al., 1960	IV	case series
Griffiths et al., 1978		basic science
Teplick & Haskin, 1983	IV	case series
Woolsey, 1985	IV	case series
Bower et al., 1989		basic science
Djukic et al., 1990	V	review, nonsystematic
Kotilainen et al., 1994	II	prospective cohort
Vandermeulen et al., 1994	V	nonsystematic review
Lawton et al., 1995	IV	case series
Baker et al., 1996	IV	case series
Weaver et al., 2001	IV	case series
Kou et al., 2002	III	case-control
Coselli et al., 2002	I	RCT
Acikbas et al., 2002	IV	case series
Uribe et al., 2003	IV	case series
Sorensen, 2003	III	case-control
Suri et al., 2003	II	retrospective cohort
Settepani et al., 2003	IV	case series
Huang et al., 2004		basic science
Curry et al., 2005	IV	case series
Schade et al., 2005	II	prospective cohort

treatment, and minimize cord recovery after surgery. In vitro, cord recovery has been shown to be related to the duration and the force of compression.^{15,66} Timely recognition of the pathological entity, its extent, and expedited treatment of patients with incomplete SCIs provide an optimal chance of recovery.

Another potential presurgical cause of POSCI is hyperextension of the neck during intubation and positioning. The method of intubation is critical especially in patients with severely tight cervical canals such as those with congenital stenosis, diffuse idiopathic skeletal hyperostosis, OPLL, and also those with severe spondylosis of the lumbar spine, as there is a correlation with spondylosis of the cervical spine.^{24,54} Other patients at risk for SCI from intubation, or iatrogenic worsening of an already existing SCI, are those with potentially unstable cervical spines who have rheumatoid arthritis, Down syndrome, traumatic cervical spine injuries, and hypermobile transition zones between fused regions, such as in Klippel-Feil syndrome. In either group of patients, a careful fiberoptic endotracheal intubation without hyperextension of the neck is an important consideration to prevent SCI.⁸¹ Following intubation, positioning of the patient should be performed with careful control of the spine. Transfer of the patient after intubation should be performed safely and with enough assistance to ensure gentle atraumatic positioning of the spine.

Hypotension is another potential presurgical cause of POSCI, and can occur during or after the induction

of anesthesia. Hypotension can lead to decreased cord perfusion and worsen an existing SCI, because the “cord at risk” cannot self-autoregulate.^{26,47,48,70,88} Maintaining hypotensive anesthesia to minimize blood loss is contraindicated in patients with preexisting SCIs or who are at high risk for intraoperative SCI. Vasopressors such as dopamine and levophed should be used at the start of induction to maintain the MABP > 80–85 mm Hg in cases where the cord is already injured or at risk for further cord injury.⁸³

Surgical Causes of POSCI in the Cervical Spine

In the anterior cervical spine, the risk of cord injury during anterior cervical discectomy is low, 0–0.3%.^{29,34} In cases of stenotic cervical spines with preexisting myelopathy, rates of SCI or worsening of preexisting myelopathies are higher, but remain low, 1–3% when an anterior approach is used.^{5,23,27} Adequate lighting and visualization with magnification of the anterior spinal cord is important during decompression. In general, no pressure or manipulation of the cord should happen. During instrumentation, great care must be maintained to ensure that all drill bits, retraction pins, and screws have lengths shorter than the sagittal diameter of the vertebral body.

In a 5-year review, Graham³⁷ revealed that the rate of SCI from a posterior approach was higher than when an anterior approach was used for decompression; the overall rate of neurological injury was 2.18 and 0.64%, respectively. Posterior cervical instrumentation has also been shown to be safe and low risk. Lateral mass screw insertion has been shown to be reliable with minimal risk to the spinal cord because screws are directed away from the cord.¹⁸ Neurological injury with lateral mass screws are usually nerve root injuries, occurring at a rate of 1.3–1.8%.^{18,36} Pedicle screws placed at C-7 are also safe; Vaccaro et al.^{1,3} showed no occurrence of SCI when screws were placed in conjunction with a laminoforaminotomy. The use of transarticular screws at C1–2, although a technically demanding procedure, has been shown to be a safe method of fusion, with several authors demonstrating no occurrence of cord injury.^{20,40} The C-1 lateral mass/C-2 pars screw fixation technique is also a safe alternative in patients who cannot undergo transarticular screw fixation due to anatomical limitations such as a high riding vertebral artery or fixed subluxation of C-1 on C-2. Harms and Melcher⁴² showed that the technique can be performed without injury to the cord. For posterior instrumentation, sagittally reconstructed CT scans should be obtained preoperatively to assess the bone anatomy and the trajectories for cervical pedicle screws, to minimize the chance of cord or vertebral artery injury.

Surgical Causes of POSCI in the Thoracic Spine

The gray matter of the midthoracic spine from T-4 to T-9 is considered to be a vascular watershed zone.²² Hypotension or hypovolemia should be avoided and the MABP should be maintained higher than 80–85 mm Hg. This may require the use of vasopressors such as dopamine starting prior to or at the time of anesthesia induction.

Intraoperative thoracic SCIs occur by a variety of different mechanisms during anterior approaches to the thoracic spine. Disruption of the radiculomedullary feeding arteries of the thoracic cord, including the artery of Adamkiewicz can lead to spinal cord infarction.⁵⁸ Sacrifice of segmental arteries in the thoracic spine should be done at the midvertebral body. The risk of left transthoracic unilateral sacrifice of segmental vessels is 0.75%.⁶⁴ However, bilateral disruption of segmental arteries is not as safe, as shown by the vascular surgery experience with aortic aneurysms.³⁵

A clear understanding of the cord location is required prior to thoracic cord decompression. The pedicle ipsilateral to the anterior thoracic approach can be removed to enter the thoracic canal and location of the cord. Anterior instrumentation with screws should be directed away from the spinal cord. The patient should also be in the straight lateral position to ensure the screws are not misdirected into the canal.

Posterior thoracic decompression should be performed similar to cervical spine decompression, with minimal pressure or mobilization of the spinal cord. The Kerrison rongeur should be maintained perpendicular to the bone, and the footplate parallel to the dura mater. Instruments should be held in both hands at all times.

Instrumentation of the posterior thoracic spine with pedicle screws is a relatively safe procedure. In a series of 4604 thoracic pedicle screws, inserted for deformity correction, 0.8% of patients suffered neurological complications.⁷⁵ Medial pedicle breaches place the spinal cord at risk, especially during deformity correction, in which the cord is typically draped on the concavity of the spine. Preoperative axial CT scan images should be reviewed to assess the diameter of the pedicle isthmus and ensure that the pedicle can be cannulated, and to assess the pedicle trajectory. If the pedicle is too narrow for screw placement, alternative fixation techniques such as an extrapedicular approach to screw placement in the thoracic spine or the use of pedicle hooks should be considered.⁴⁵

Underlying cord diseases such as syringomyelia, tethered cord, or an associated Chiari malformation must be recognized prior to deformity correction. On clinical examination of apparent idiopathic scoliosis patients with neurological signs, patients younger than 11 years of age, with a left thoracic scoliosis, or significant neck or back pain should be further investigated with MR imaging.⁴⁶ Syringomyelia is a risk factor for neurological complications during correction of scoliosis and should be assessed prior to deformity correction.

During coronal or sagittal plane deformity correction, the spinal cord may undergo distraction as the curvature is straightened, leading to disruption of the blood supply to the cord.⁵⁷ Winter⁸⁶ reported on 2031 procedures for scoliosis or kyphosis with no complete SCIs and 0.3% partial cord injuries. Intraoperative neuromonitoring may detect changes, allowing the surgeon to reverse or decrease the amount of deformity correction. Clear and reliable communication between the surgeon and the neuromonitoring team must exist. An alternative or complement to intraoperative neuromonitoring, is the Stagnara Wake-up test to assess the anterior motor por-

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tion of the spinal cord.^{39,49,52} However, this requires preoperative planning with the anesthesiologist and a patient who can understand and comply with directions. The ankle-clonus test is another intraoperative test of motor function as described by Hoppenfeld et al.⁴³ that can be used to test anterior cord function. However, the use of multimodality evoked potential monitoring (MEP and SSEP) combined with continuous and evoked electromyography has largely supplanted the wake-up and ankle clonus tests.

Postoperative Causes of SCI

Postoperative causes of SCI include epidural hematomas and infection. New postoperative neurological deficits warrant neuroimaging consisting of plain radiographs and MR imaging. Most surgical procedures will cause the development of small and clinically inconsequential epidural hematomas;^{50,80} however, those large enough to cause spinal cord compression and warrant surgical intervention are rare, with an incidence of 0.1%.⁵⁵ Epidural hematomas are typically diagnosed within 24 hours after surgery, although there have been reported cases occurring more than 3 days after surgery.⁸² Risk factors for developing epidural hematomas with spinal cord compression include multilevel decompressions and patients with coagulopathy.⁵¹ Emergency decompression should be performed within 8 hours as most patients will make good or partial neurological recovery.⁸⁴

Epidural abscesses are also a potential cause of spinal cord compression postoperatively and are very rare. Overall, 1 in 10,000 hospital admissions per year are for epidural abscesses, of which 16% were due to postoperative infections.⁴ Progression of symptoms include back pain, then radicular pain and weakness, followed by paralysis. *Staphylococcus aureus* is the most common causative pathogen. Patients may be normothermic and have normal white blood cell counts.¹³ Magnetic resonance imaging with and without Gd is the imaging modality of choice. Typically, the lesion is isointense with the spinal cord on T1-weighted images and hyperintense on T2-weighted images;²¹ Gd enhances the epidural abscess. Urgent surgical decompression along with antibiotic therapy is warranted if the patient has neurological deficits, if the microorganism is not known, or if the infection is not responding to antibiotic therapy. Epidural abscesses caused by a known organism in patients without deficits can potentially be treated medically.⁷³

Long-Term Care of Patients with POSCIs

The long-term prognosis for patients with POSCIs is variable and can depend on the patient's age, extent of injury, and whether the injury is complete or incomplete. However, based on the cervical myelopathy literature, patients with signal changes on both T1- and T2-weighted images have a worse chance of recovery compared to patients who only have signal changes on T2-weighted images.^{61,76} Serial MR imaging examinations can be performed to assess potential improvement in the cord signal changes.

The surgeon should facilitate appropriate multidisciplinary care for the patient to facilitate optimal long-term

recovery and outcome and should also make a commitment to conduct long-term follow-up with the patient. The goals of SCI rehabilitation are to help the patient achieve as much independence as possible through the treatment of all affected systems. The pulmonary system requires careful attention, with frequent mobilization and deep inspiriometry to minimize atelectasis and pneumonia. If the cord injury is above C-4, a ventilator or a diaphragmatic stimulator may be potentially required. The integuments should be carefully assessed, especially the sacral and heel regions. Patients who cannot independently turn in bed need frequent turning every 2 hours and the use of air mattresses to minimize pressure on these regions. Genitourinary care involves teaching the patient to do frequent, intermittent self-catheterizations. Care should be taken to observe for autonomic dysreflexia. Should a hypertensive crisis occur, catheter drainage should be checked in addition to the bowels; 10-mg of nifedipine can be administered during a hypertensive crisis. Bowel training should also be undertaken with stool softeners and suppositories. Counseling and support is required to help the patient adapt psychologically. All of these goals are best reached at specialized SCI rehabilitation centers and typically involve a long-term stay of 3–5 months. Depending on the level of the injury, the degree of patient independence can vary.⁸⁷ Patients with an injury at C-5 or higher cannot achieve functional independence. Patients with an injury at C-6 require occasional assistance with transfers but can use a modified wheelchair. Patients with an injury at C-7 or lower can become completely independent.

Illustrative Case 1: POSCI Prevention Protocol

This 75-year-old man of Asian descent presented with a progressively worsening gait imbalance and clumsiness in both hands, which had started 1 year prior to his presentation. There was no history of trauma, and he had no bowel or bladder dysfunction. Previously, he was able to ambulate without walking aids, but now required a walker. He had significant worsening of his handwriting ability, difficulty in buttoning his shirts, and repeated episodes of dropping light objects such as coffee cups. In terms of medical history, he had mild, diet-controlled Type 2 diabetes mellitus.

Physical examination revealed that he had lost intrinsic muscle mass in both hands. Assessment of both legs and arms revealed spasticity, with Hoffman and Babinski signs present bilaterally. He had marked clonus in both ankles, and his reflexes were globally hyperreflexic. His gait was extremely unsteady and wide based, despite his use of a walker. His motor strength was grade 4 out of 5 on the Medical Research Council scale. Sensation in both arms and legs was intact to light touch, pin-prick, and proprioception.

Magnetic resonance images revealed severe cervical spinal stenosis extending from C2–3 down to C6–7 from severe spondylosis (Fig. 1). There was an area of signal change within the spinal cord behind C5–6 and C6–7 where the cord was most severely compressed. His cervical spine had maintained a lordotic sagittal alignment.

FIG. 1. Case 1. Cervical spine sagittal T2-weighted MR image showing compression of the spinal cord at multiple levels due to severe spondylosis, particularly at C5-6 and C6-7. There is signal change at these two levels.

The patient was admitted to the hospital and gave his consent to undergo posterior cervical decompression and segmental instrumented fusion for his cervical myelopathy from severe spondylosis. The posterior approach was chosen due to the multiple levels of compression. With his sagittal alignment, a posterior decompression would allow the cord to fall posteriorly away from the posterior vertebral body wall and disk bulges.

This surgical procedure had increased risks of SCI. To minimize the potential for injuring his spinal cord and worsening his myelopathy, we implemented the following protocol to reduce the chance of POSCI.

1. Maintain MABP Above 80 mm Hg

Dopamine treatment was initiated prior to anesthesia induction to ensure that his MABP was maintained above 80 mm Hg. In the setting of underlying spinal cord compression or another pathological entity, which renders the cord at risk for ischemia, we strongly advocate maintenance of spinal cord perfusion through the prevention of hypotension during induction, intraoperatively, and potentially postoperatively. This may require volume resuscitation using crystalloid, maintenance of a normal hematocrit (> 32%), and vasopressors such as dopamine, followed by levophed if needed. Epinephrine should be avoided as it exerts its effects only on the alpha-receptors, and can reduce spinal cord perfusion. No ideal MABP has been determined. However, previous clinical studies have used an empirical minimum of 80–85 mm Hg based on the management of patients with head injuries.⁸³

FIG. 2. Case 1. Midline postoperative sagittal T2-weighted MR image showing posterior cervical decompression from C-2 to C7-T1. Residual signal change is present in the spinal cord at C5-6 and C6-7.

2. Start Treatment With MPSS Preoperatively and Discontinue After 24 Hours

If the spinal cord is at high risk for intraoperative SCI or if work within the spinal cord is going to be done, MPSS can be started before surgery. Naso et al.⁶³ showed in their basic science rat model that high-dose MPSS administered prophylactically has a neuroprotective effect by minimizing secondary axonal injury in surgical trauma. The impact of SCI can be devastating, and MPSS offers potential benefits as exhibited in several randomized controlled trials; it also has a relatively safe medication profile.⁷ Based on the recommendations of Bracken et al.^{8,9} (NASCIS II), a 30 mg/kg bolus of MPSS is given over 15 minutes; this can be done before induction. After a 45-minute wait, an infusion of 5.4 mg/kg/hour is given until the patient is assessed postoperatively. If there is no evidence of SCI, and there is low risk of cord edema from the surgery, steroid treatment can be discontinued. If there is evidence of SCI and/or a moderate-to-high risk of edema in the cord (such as after an intramedullary tumor resection), MPSS can be continued for a total of 24 hours. Because MPSS can cause elevations of blood glucose, potentially predisposing patients to an increased risk of neuronal injury and counteracting the beneficial effects of MPSS, especially in patients with diabetes, blood glucose assessments should be made every 2 hours during infusion of MPSS.³³ Administration of insulin maybe required to maintain blood glucose within normal limits. In basic science studies, insulin has been shown to be neuroprotective, preventing neuronal cell death.⁶² Our patient, a Type 2 diabetic, received insulin from the anesthetist intraoperatively and an endocrinologist postoperatively to maintain a normal blood sugar profile during MPSS administration.

FIG. 3 Case 2. *Left:* Anteroposterior radiograph showing the broken left cervical rod. The patient has kyphoscoliosis from neurofibromatosis Type 1. *Right:* Lateral radiograph showing the fractured cervical rod at the cervicothoracic junction.

Although the evidence from the NASCIS trials is controversial, the results show motor improvement when given within 8 hours of the SCI. MPSS administration for acute SCI is supported by numerous basic science studies that have demonstrated the ability of MPSS in deactivating free-radical oxidation products that demyelinate neurons during the secondary cascade following a SCI and reducing edema and inflammation.^{19,41} MPSS is a relatively safe drug as shown by Sauerland et al.⁶⁷ where a systematic review of 51 trials using high dose MPSS versus placebo or nothing showed no evidence of harm for spine surgery. There was no evidence of increased risk for gastrointestinal bleeding, wound complications, pulmonary complications, or death.

3. Maintain Careful Spine Control

Fiberoptic intubation was used in our patient to minimize neck extension. The patient was positioned supine on a Jackson table with a Mayfield attachment, and was then turned prone with the rotating frame.

4. Use Adequate Lighting, Magnification, and Assistance

5. Use Multimodal Intraoperative Neuromonitoring

We used SSEPs, MEPs, and electromyography recordings (for cervical and lumbar procedures). No intraoperative signal changes occurred.

6. Recognize That Distractive Forces Applied to the Spine May Cause a Combined Stretch/Ischemic Injury

Distraction, when necessary, should be applied carefully and under neurophysiological monitoring. In our patient, no distraction was required. This protocol is more applicable in deformity correction cases.

7. Start Decompression Away From the Tightest Stenosis

Instruments should be held with both hands at all times. A high-speed drill should be used to thin out the compressive osseous structures to an “egg-shell” thickness. Only fine instruments (curettes and thin foot-plate Kerrison punches) should be used for decompression in the cervical and thoracic spine. In our patient, 2 posterior troughs were created at the junction between the lamina and the lateral masses using a Midas bur, followed by removal of the lamina and ligamentum flavum.

Outcome of Protocol

Utilizing this protocol, we successfully decompressed the cervical spine in our patient with no adverse outcomes (Fig. 2). Six weeks postoperatively, the patient reported improvement in his gait. See Table 1 for an overview of the treatment options outlined here to help minimize the probability of a POSCI from occurring.

Illustrative Case 2: Treatment of POSCI Recognized Postoperatively

This 33-year-old woman with known cervicothoracic kyphoscoliosis due to neurofibromatosis Type 1, underwent an upper thoracic pedicle subtraction osteotomy with a plan to do a staged anterior cervicothoracic decompression. However, due to a dural tear during the first stage of surgery, the second stage procedure was delayed. While waiting for the second stage surgery, the patient fell and broke her posterior hardware (Fig. 3). She returned to the operating room for revision of the posterior instrumentation to replace the broken rod and gently correct the loss of her original deformity correction.

Awake fiberoptic intubation was performed for both surgeries, and her MABP was maintained over 80 mm Hg throughout the operation. Perioperative steroids were not used prior to the first or second operation; MEPs and SSEPs were done during both operations. The Wake-up

FIG. 4. Case 2. Sagittal T2-weighted MR imaging demonstrating compression of the spinal cord caused by the kyphotic deformity at the cervicothoracic junction.

test was not performed either time. At baseline in the second case, no MEPs could be obtained and the SSEPs were fine. No significant blood loss occurred and the surgical time was 2 hours. When the patient awoke, she had complete paraplegia in both legs, and C-7 paresis on the right, and C-6 paresis on the left due to anterior cord syndrome secondary to deformity correction in the setting of residual ventral compression.

The SCI was immediately recognized, along with its cause from the kyphotic cervicothoracic region causing ongoing residual ventral compression (Fig. 4). She was taken back to the operating room emergently for an anterior cervicothoracic decompression and fusion. Dopamine was administered preoperatively to maintain her MABP above 80 mm Hg. Methylprednisolone sodium succinate was administered preoperatively under the NASCIS II protocols. An awake fiberoptic intubation was performed. Both MEP and SSEP activity was present in the upper extremities and there were no lower limb MEPs and SSEPs. Following the procedure, some MEP and SSEP activity returned. Postoperative imaging demonstrated excellent decompression (Fig. 5), and she returned to her baseline level of functioning over the next 6 months.

Treatment Options After an Intraoperative SCI

Similar to the second case example, which highlighted the management of a postoperatively recognized SCI, several steps can be taken to help minimize damage from the secondary cascade following recognition of an intraoperative SCI. First, the occurrence of a SCI must be recognized and acknowledged in order for actions to be taken to reduce the impact of the injury as soon as possible. This involves multimodal neuromonitoring during surgery, and regular and frequent spinal cord neurologic

FIG. 5. Case 2. Sagittal CT reconstruction showing decompression of the anterior cervicothoracic junction. A fibular allograft from C-4 to T-2 was used along with an anterior cervical plate.

testing after surgery. If possible, the cause of the intraoperative SCI should be removed such as the instrument or screw or reversal of the deformity correction. Secondly, the MABP should be maintained above 80–85 mm Hg and the hematocrit should be corrected. Maintenance of the MABP may require the use of vasopressors such as dopamine and/or levophed. Treatment with MPSS can also be initiated as soon as possible after identification of the SCI. Although meta-analysis data suggest that there are no increases in rates of side effects of MPSS,⁶⁷ a number of other authors have documented potential increases in rates of infections, blood sugar levels, and gastrointestinal bleeding.^{32,56,60,74} Furthermore, the NASCIS trials applied only to blunt, closed SCI and not open spinal cord trauma.

Relative systemic cooling or local cooling can also be performed to minimize oxygen demand from the spinal cord, reducing the potential ischemic damage following a cord injury. However, this is experimental and is not routinely used or offered at most spine surgical centers. The vascular surgery literature for thoracoabdominal aortic aneurysm repairs have shown that cooling the epidural space is potentially neuroprotective and safe.^{53,77,78} The optimal temperature is not known. However, the use of normal saline chilled to 4°C to cool the epidural space to 25°C has been shown to be effective in minimizing postoperative neurological complications.¹⁷

If the area operated on is mechanically unstable, the region should be stabilized with screw fixation to create an optimal area for neurological recovery. If cross-links are to be placed for the construct, they should be placed away from the region of injury to minimize scatter and artifact during follow-up CT and MR imaging scans.

Following the completion of surgery, an epidural

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drain can be placed. Cerebrospinal fluid drainage is often used as an adjunct for spinal cord protection following thoracoabdominal aortic aneurysm repair. Coselli et al.¹² showed in a randomized control trial with and without CSF drainage following thoracoabdominal aortic aneurysm that 2.6% of patients developed neurologic deficits/paraplegia with CSF drainage versus 13% of patients treated without CSF drainage.

The rationale behind CSF drainage is based on animal studies where it was shown that decreasing CSF pressure to < 10 mm Hg enhances perfusion of the cord and decreases the risk of ischemic damage.^{6,38,59} Duration of treatment and the actual volume of CSF drainage is not known. However, the risks associated with CSF drainage include meningitis and overdrainage which can result in intraspinal hematomas, subdural/intracerebral hematomas, and acquired foramen magnum stenosis secondary to cerebellar tonsillar herniation.^{2,14,16,68,71,85} It is thought that tonsillar herniation occurs due to the pressure difference between the supratentorial and lumbar cistern compartments.⁷² Studies have shown CSF production to be ~ 20 ml/hour.⁴⁴ Because of these risks, it is recommended that CSF drainage be ~ 15–20 ml/hour and discontinued after 72 hours to minimize infection risk.

Discussion

Based on our review of the literature, no study has looked at prevention or minimization of the effects of POSCIs. As a result, for this article, treatment options have been developed utilizing evidence from a variety of basic science and clinical studies obtained from a systematic review, both, for prevention and for treatment of recognized POSCIs. As a result, no firm guidelines can be provided and only treatment options are listed. Furthermore, basic science studies may not appropriately translate into clinical practice. Further research would be beneficial in providing better clinical data.

Randomized control trials, considered the gold standard level of evidence, would not be appropriate for the study of POSCIs. It would be challenging to randomize a patient with a POSCI and patient enrollment would be poor. In addition, the number of cases per year would be small, making it impractical to enroll enough patients.

A dedicated study on POSCI can be accomplished through a prospective multicenter cohort study to identify the incidence of POSCI and risk factors that increase the odds of POSCI from occurring and determinants of outcome after POSCI occurs. In addition, the study would provide an overview of what methods are being used to prevent POSCI and what is being done in the event of a POSCI.

Several of the potential treatment options, in the event of a POSCI occurring such as systemic cooling and CSF drainage are controversial and are predominantly found within the literature on thoracoabdominal aneurysm surgery rather than in the spinal surgical literature. These treatment options warrant further investigation prior to implementation.

Conclusions

Perioperative SCIs are a risk of surgery around the spinal cord. Multimodal neuromonitoring has been assessed as a method to minimize the chance of POSCI. However, no studies exist that detail how to reduce the impact of a POSCI should it occur. In the present study, we outlined treatment options based on our systematic review of the basic science and clinical literature. Further prospective studies are needed to accurately assess what is being performed in clinical practice to minimize POSCI, identify the incidence of POSCI, and identify factors that determine outcome after POSCI.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Manuscript submitted July 15, 2008.

Accepted October 1, 2008.

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