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Critical Care Neurology (K Sheth, Section Editor)

Management of Acute Traumatic Spinal Cord Injury

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Opinion statement

Spinal cord injury (SCI) causes significant morbidity and mortality. Clinical management in the acute setting needs to occur in the intensive care unit in order to identify, prevent, and treat secondary insults from local ischemia, hypotension, hypoxia, and inflammation. Maintenance of adequate perfusion and oxygenation is quintessential and a mean arterial pressure >85–90 mm Hg should be kept for at least 1 week. A cervical collar and full spinal precautions (log-roll, flat, holding C-spine) should be maintained until the spinal column has been fully evaluated by a spine surgeon. In patients with SCI, there is a high incidence of other bodily injuries, and there should be a low threshold to assess for visceral, pelvic, and long bone injuries. Computed tomography of the spine is superior to plain films, as the former rarely misses fractures, though caution needs to be exerted as occipitocervical dislocation can still be missed. To reliably assess the spinal neural elements, soft tissues, and ligamentous structures, magnetic resonance imaging is indicated and should be obtained within 48–72 h from the time of injury. All patients should be graded daily using the American Spinal Injury Association classification, with the first prognostic score at 72 h postinjury. Patients with high cervical cord (C4 or higher) injury should be intubated immediately, and those with lower cord injuries should be evaluated on a case-by-case basis. However, in the acute setting, respiratory mechanics will be disrupted with any spinal cord lesion above T11. Steroids have become extremely controversial, and the professional societies for neurosurgery in the United States have given a level 1 statement against their use in all patients. We, therefore, do not advocate for them at this time. With every SCI, a spine surgeon must be consulted to discuss operative vs nonoperative management strategies. Indications for surgery include a partial or progressive neurologic deficit, instability of the spine not allowing for mobilization, correction of a deformity, and prevention of potential neurologic compromise. Measures to prevent pulmonary emboli from deep venous thromboembolisms are necessary: IVC filters are

recommended in bedbound patients and low-molecular weight heparins are superior to unfractionated heparin. Robust prevention of pressure ulcers as well as nutritional support should be a mainstay of treatment. Lastly, it is important to note that neurologic recovery is a several-year process. The most recovery occurs in the first year following injury, and therefore aggressive rehabilitation is crucial.

Introduction

Acute traumatic spinal cord injuries (SCI) impose a significant emotional and financial burden on patients, families, and society. It is estimated that the annual incidence of SCI is approximately 10–80 cases per million worldwide [1–3], with a male predominance (up to 80 %) and an average age of 38 years.[4] In the United States, there are 11,000 new cases per year, with more than two-thirds of patients under 30 years old. More injuries result in tetraplegia (51.7 %) than paraplegia (45.9 %). Motor vehicle accidents account for almost one-half of the injuries in the developed world, followed by falls, violence-related injuries (especially gunshot wounds), and sports accidents [4]. Alcohol plays a role in 25 % of SCI cases [2]. In patients older then 55 years, falls account for 60 % of SCI [3], as underlying spine disease such as cervical spondylosis, atlantoaxial instability, osteoporosis, and spinal arthropathies can predispose such patients to SCI [2, 5] The functional outcome is dependent on the severity of the initial injury, and thus the financial burden of SCI can vary widely between individual patients, with direct costs of 500,000 to 2 million US dollars accrued over a patient's lifetime [2].

SCI can be separated into primary and secondary injury, with the former resulting from pathologic flexion, rotation, extension, compression, contusion, shearing, as well as fracture-dislocation, ligamentous tears or disruption, and/or herniation of intervertebral disks. These primary events that occur at the time of trauma initiate a cascade of secondary molecular mechanisms similar to those found in traumatic brain injury or acute ischemic stroke: (1) ischemia from vascular injury that results in vasospasm, thrombosis, infarct, hemorrhage, and a loss of autoregulation; (2) hypoxia; (3) edema; (4) excitotoxicity from neurotransmitter accumulation, free radical buildup, and lipid peroxidation; (5) inflammation; and (6) apoptosis and delayed cell death [6].

Therefore, hospital treatment paradigms are designed to manage and prevent secondary SCI and associated medical comorbidities, as well as secondary insults that increase damage in previously injured spinal cord tissue. These insults include hypotension and hypoxia that decrease oxygen and glucose delivery to the injured cord, fever that increases metabolic demand, and hyperglycemia that exacerbates inflammation and injury. In this review, we will briefly discuss the evaluation of a patient with SCI, prognostic factors, standard imaging modalities, medical management in the intensive care unit (ICU), the steroid controversy, surgical management, and emerging therapies.

Evaluation of patient

Physical and neurologic examination

An initial assessment must include ABCs and strict adherence to ATLS guidelines. ABCs cannot be overemphasized as inadequate perfusion and oxygenation to an already injured spinal cord will trigger secondary cascades that will, in turn, increase morbidity and mortality and prevent neurologic recovery [7••]. The forces leading to SCI are typically significant enough to inflict additional traumatic injuries, such as long bone fractures, pelvic fractures, visceral injuries, and head injuries. Therefore, the examiner should have a high level of suspicion for additional injuries, especially given that in SCI sensation is likely to be compromised. Following ATLS, a neurologic examination should be

conducted, and the degree of injury classified using the American Spinal Injury Association (ASIA) international standards [Class II] [8••]. The initial clinical presentation is crucial for triaging, defining therapy, and predicting prognosis (Table 1) [8••] ASIA upper and lower extremity motor subscores are also valuable for following the time course of injury. It should be noted that the first reliable ASIA score for determining prognosis is at 72 h, and thus it is important that every patient be rescored after the initial assessment [9••]. With this knowledge, some institutions grade ASIA daily during a patient's entire hospitalization.

Immobilization

In all cases of trauma, patients with neck pain, and/or neurologic compromise on examination should be assumed to have a cervical injury until proven otherwise. Therefore, the initial management of all cervical injuries should focus on immobilization with a hard, rigid cervical collar in order to limit further injury. A spine surgeon may choose to transition to a halo orthosis depending on the underlying pathology. If occipitocervical dislocation is diagnosed, the patient's cervical spine should be immobilized with sandbags in a neutral position or with a halo orthosis instead of a cervical collar, as the collar is a distracting device that can worsen occipitocervical dislocation [10•]. Similarly, traction in this instance is contraindicated [10•]. For other injuries, a spine surgeon may choose closed traction to reduce the injury.

For thoracic and lumbar trauma, the patients need to be kept on "spine precautions" in which they are kept flat or in reverse Trendelenburg in order to prevent movement. The patient needs to be logrolled to keep the body in one axis until the spine has been deemed stable. If the spine is unstable, the patient has to remain on spine precautions until surgically stabilized.

Prognostication

"What are the chances of neurologic recovery?" is a question concerning both patients and clinicians alike. The majority of neurologic improvement occurs in the first year, though patients can continue to make small improvements for 3–4 years afterwards [7••]. The ASIA examination is the most sensitive measure for assessing recovery and as mentioned above, 72 h is the first time point when the examination is reliable for prognostication [9••]. Therefore, we recommend caution when predicting recovery based on the initial ASIA evaluation or an examination obtained in just the first few days, as these will be less prognostic.

Based on epidemiologic data, about 49 % of SCI patients are ASIA grade A (complete injury), 13 % grade B, 16 % grade C, and 22 % grade D [11••]. Distinguishing between grade A and B, in which there is some preservation of sacral sensation in the latter, is especially important because a significantly greater number of individuals who present as grade B will eventually go on to ambulate. After all, determining whether a patient is likely to regain ambulation has a significant impact on their expected quality of life. Even with a poor initial presentation, 8.3 % of grade A injuries will improve to grade D or E at 1-year follow-up [11••]. For grade B injuries, 39.4 % will go on to grades D or E [11••, 12]. Thus, a significant number of people who come in with only sacral sensory sparing will go on to walk either with assistive devices or even independently. The anatomic basis for this recovery is thought to be the proximity of the

Table 1. ASIA Scale

Category	Description
A	Complete: No motor or sensory function is preserved below the neurologic level through sacral segments S4–S5.
B	Incomplete: Sensory but not motor function is preserved below the neurologic level and includes S4–S5.
C	Incomplete: Motor function is preserved below the neurologic level, and more than one-half of key muscles below the neurologic level have a muscle grade <3.
D	Incomplete: Motor function is preserved below the neurologic level, and at least one-half of key muscles below the neurologic level have a muscle grade ≥3.
E	Motor and sensory functions are normal.

spinothalamic tracts to the corticospinal tracts [13]. For grade C injuries, 61.8 % improve to D or E and for grade D, 97.3 % remain or go on to E [11••, 12].

Imaging and anatomy

General guidelines

In general, a computed tomography (CT) of the spine is superior to plain films [14••]. Magnetic resonance imaging (MRI) is required to fully assess the neural elements, soft tissues, and ligamentous structures, and should be obtained within 48–72 h of injury to reliably do so [14••, 15]. It is thought that edema decreases after the 48-to 72-h window and that after this time period an MRI will less reliably detect injury [14••, 15]. The MRI should include STIR sequences, which assess ligamentous injury with the most fidelity.

Cervical spine

The cervical spine is often considered as two regions: the upper cervical spine (occiput to C2) and the lower subaxial cervical spine (C3–C7). The upper cervical spine is greatly involved in flexion and extension, as well as rotation, with the ligaments accounting for the majority of the stability in this region [7••]. Given the wide canal size in the upper cervical spine, spinal cord injury in this region is rare [7••]. In the cervical spine, the degree of motion is secondary to the unique bony and ligamentous anatomy that often predisposes this region to injury. Given that 17 % of all cervical spine fractures occur at the cervicothoracic junction, it is crucial that this region be adequately imaged [16]. However, occipitocervical dislocations can result in significant SCI. These rare ligamentous injuries result from hyperflexion distraction, are unstable, and are usually fatal because of distortion of the brainstem and vertebral artery injury [17]. The two main professional societies for US neurosurgeons—the American Association of Neurological Surgeons (AANS) and the Congress of Neurological Surgeons (CNS) —issued consensus guidelines for assessing the spine in 2013 [14••]: for patients with neck pain and/or neurologic compromise, guidelines recommend obtaining a high-quality CT of the cervical spine over plain films because the latter can miss a significant amount of pathology [14••]. If high-quality CT imaging is unavailable, a three-view cervical spine series (anterioposterior, lateral, and odontoid views) is recommended14, making sure to include the cervicothoracic junction (ie, the scan must include T1).

Because occipitocervical dislocation can be still missed on a high-quality CT, guidelines recommend that a spine surgeon review the CT in conjunction with a radiologist to assess for potential dislocation [14••]. In the subaxial spine, bony elements and ligaments play a more equal role in maintaining stability. Given that the spinal canal is smaller in this region, SCI is more common [7••]. The three most common types of injury are compressive flexion, compressive extension, and distractive flexion [18]. To assess the neural elements and ligamentous tissues, an MRI should be obtained within 48–72 h, with STIR sequences being the most sensitive for detecting ligamentous injury [14••, 15]. If there are fractures of the transversarium foramina or a concern for vertebral artery injury based on the modified Denver Screening Criteria, the level-1 AANS/CNS recommendation is to obtain a CT angiogram [19]. If there is a vertebral artery injury, neurology and neurosurgery should be consulted to discuss anticoagulation vs antiplatelet therapy versus endovascular/surgical intervention [19].

Thoracic and lumbar spine

The thoracic spine is more stable than the cervical spine because it articulates with the rib cage and sternum, and has coronally oriented facet joints [7••]. Significant trauma is required to destabilize the thoracic spine, though a smaller spinal canal size results in an overall higher incidence of SCI [7••]. The thoracolumbar junction (T10–L2) marks the transition from a rigid thoracic spine to a more mobile lumbar spine, along with a change in the orientation of the facets, and in axial loading. The unique anatomy of this transition zone makes this region responsible for 40 % of all SCI [7••]. When imaging the thoracic spine, besides assessing the alignment, the amount of kyphosis must be determined to be either normal or traumatic from ligamentous injury. Burst fractures are the most common type of fracture, followed by Chance fractures [7••]. In the lower lumbar spine (L3–L5), spinal cord injury is much rarer because the spinal cord typically ends at L1–L2. Given the large vertebral bodies, extensive musculature, and orientation of the facets, only 4 % of spinal fractures occur in this region [7••]. For both the thoracic and lumbar spine, CT is preferred over plain films to assess for bony fractures and misalignment, whereas MRI is utilized to assess neural elements, soft tissues, and ligaments [14••, 15].

Medical treatment and management

Hemodynamic instability

Hemodynamic and ventilatory failures are common after SCI, especially following injuries of the cervicothoracic spine. In these cases, ischemia is the most common culprit for continued secondary neurologic insult [20]. Therefore, in patients with acute SCI, cardiac, hemodynamic, and respiratory monitoring in an ICU setting are crucial to detect cardiopulmonary insufficiency [21••]. Within the spinal cord, direct injury to the microcirculation can lead to alterations in blood flow [7••]. At the systemic level, SCI is often accompanied by hemodynamic instability from the loss of sympathetic tone.

In the setting of hypotension, providers should first differentiate between hypovolemic systemic shock and neurogenic shock. Sympathetics supplying the

heart exit the spinal cord in the ventral roots of T1–T5. Injury at or above these levels will disrupt sympathetic outflow leading to unopposed parasympathetic input. The end result of neurogenic shock is, therefore, peripheral vasodilation leading to warm extremities and hypotension with bradycardia [7••]. Hypotension should be treated with blood transfusions, volume replacement, and vasopressors as needed. Spinal shock, on the other hand, is a physiological dysfunction that results in decreased tone and hyporeflexia in the setting of an upper motor neuron lesion. The resolution of spinal shock is marked by the return of the bulbocavernous and deep tendon reflexes [7••]. In the end, hypotension with concomitant loss of local spinal cord autoregulation of the microcirculation will compromise spinal cord perfusion and worsen ischemic injury.

Needless to say, hypotension (SBP <90 mm Hg) must be corrected as soon as possible [21••] in order to prevent ischemic insult to the spinal cord and to potentially enhance neurologic outcomes [22]. There is a level III recommendation to maintain mean arterial blood pressure of at least 85–90 mm Hg for the first 7 days following acute SCI [21••], a guideline, which we follow at our institution. First line treatment for hypotension is volume resuscitation with at least 1–2 L of crystalloids, followed by blood transfusions, depending on the presence of additional injuries and the hematocrit. However, if the increased blood volume and venous return is not matched by sufficient cardiac output, volume restoration alone will be inadequate to increase blood pressure, and vasopressors should be initiated. Given the loss of sympathetic tone, the agent of choice should have both α- and β-adrenergic properties, such as norepinephrine or dopamine [7••]. Phenylephrine can also be used, but caution should be used when doing so because its purely α-adrenergic effects will increase cardiac afterload and may worsen bradycardia [23].

Following recovery from neurogenic shock, patients with cervical and high thoracic SCI can still have resting hypotension because of sympathetic disruption. However, the majority of these individuals (50 %–90 %) will also have autonomic dysreflexia resulting in episodes of significant hypertension up to 300 mm Hg [7••]. These episodes of hypertension are trigged by nonpainful or painful sensory stimuli, such as a full bowel or bladder, below the level of the SCI. Autonomic dysreflexia can occur as early as 4 days after the injury and can result in a life-threatening crisis if not recognized [7••]. Most episodes can be managed by eliminating the stimulus (emptying the bladder, evacuating the bowel, or changing patient position) [24], but sometimes pharmacologic intervention is required. Conversely, orthostatic hypotension is quite common in the acute SCI phase, especially during changes in patient position (attempting to stand up, transferring to the wheelchair); however, 41 % of individuals with SCI who have orthostatic hypotension are asymptomatic [25]. In those who have symptomatic orthostatic hypotension, maintaining adequate circulating volumes, positioning more slowly, and pharmacologic agents such as fludrocortisone, midodrine, pyridostigmine, caffeine, and epoetin can be helpful [7••].

Respiratory compromise and failure

Respiratory failure and compromise are common in SCI, especially with injury to the cervical cord [26]. The decision to intubate is usually related to respiratory

compromise from one or more of the following: loss of innervation of the diaphragm (C3–C5), fatigue of innervated respiratory muscles, hypoventilation ($PaCO_2 > 50$ mm Hg, pH<7.30), V/Q mismatch, secretion retention, as well as other associated injuries. Intubation may complicate the opportunity for a closed spine reduction, which is safer in an awake and communicative patient. However, it should be noted that, in the acute setting, cough function is abnormal with any injury above T11 [7••]. More specifically, with an injury at C1–C3 cough is absent, C4–T1 it is nonfunctional, T2–T4, it is weak, and T5–T10 it is poor [7••]. Only injuries at T11 and below will allow for normal cough function in the acute setting. Vital capacity in the acute phase will also be abnormal if the injury is above T11. More specifically, at C1–C3 it is normal only 0 %–5 % of the time, C4 it is normal 10 %–15 % of the time, C5–T1 it is 30 %–40 %, T2-T4 it is 40 %–50 %, T5–T10 it is 75 %–100 %, and at injuries below T11 it is typically normal [7••]. It is safe to assume that patients injured at C4 and above will fail to maintain a respiratory effort in the acute phase and have a significant chance of requiring a tracheostomy within the first week [7••]. Injuries at C5 may or may not require intubation in the acute setting and, therefore, need to be evaluated on a case-by-case basis as well as watched carefully for respiratory failure. As expected, patients with thoracic SCIs, especially in the lower thoracic region, have a lower rate of intubation compared with those with cervical injuries [27]. However, a significant proportion (up to 50 %) still gets intubated because of additional injuries such as flail chest, pulmonary contusions, pneumothoraces, and hemothoraces [27].

Nevertheless, suboptimal respiratory function leads to relative hypoxemia and worsening of spinal cord ischemia. We recommend that all patients with cervical spine injuries, in addition to those with lesions at C4 and above, be considered at a low threshold for intubation, as approximately one-third will eventually require ventilator support [28]. In those patients who are not intubated, additional monitoring for signs of respiratory decompensation and subsequent respiratory failure, such as a vital capacity <1 L and a rising PCO_2, is crucial. We recommend proceeding with intubation if clinicians are at all considering it, as proactive intubation under controlled settings is always preferable to emergency situations with potentially prolonged periods of hypoxemia. It goes without saying that in patients with known or presumed cervical spine injuries, intubation must be performed with manual in-line traction and/or with a cervical collar to prevent further neurologic injury. In this setting, an experienced team should be the ones to perform orotrachael intubation and a fiber-optic setup may additionally be required [29].

Over time, respiratory muscles become spastic and more rigid, actually allowing for improvement in respiratory function [30]. Ventilatory weaning is related to the level of injury: on average 65 days for C1–C4, 22 days for C5–C8, and 12 days for thoracic injuries [31]. To help prevent pneumonia and maintain secretions, patients are usually kept upright. However, tetraplegic patients maintain better ventilation in the supine position, and this must be weighed against the concern for secretions and potential aspiration [32]. In these patients, flaccid abdominal musculature leads to overdistention of the diaphragm and decreased minute ventilation, which can be ameliorated with abdominal binders [33].

Other organ system general recommendations

Upon the patient's arrival, a Foley catheter should be placed to avoid bladder distention from a neurogenic bladder, with a subsequent transition to intermittent catheterization to decrease the chance of infection. Neurogenic bowel is also common and an aggressive bowel regimen should be the standard of care. SCI patients are at high risk for stress ulcers, particularly if steroids have been given, and patients should, therefore, be maintained on gastrointestinal prophylaxis. Deep venous thrombus prophylaxis with either subcutaneous heparin or lovenox is paramount, though it should be noted that low-molecular weight heparin is associated with a statistically significant lower amount of deep vein thrombosis compared with unfractionated heparin [34]. For patients who are bedbound or with limited mobility, an inferior vena cava (IVC) filter should be considered. IVC filters are known to be effective in preventing pulmonary emboli and have a low complication rate [35]. SCI patients are also at high risk for pressure ulcers, and optimal medical management of underlying conditions such as diabetes and peripheral vascular disease must be maintained with a multidisciplinary approach to nursing, nutrition, and physical/occupational therapy. In any bedbound or limited mobility patient, pressure ulcers should be assumed to occur and, thus, must be monitored for daily.

Intraspinal pressure, spinal perfusion pressure, and blood flow

The benefit of placing lumbar drains to decrease intrathecal pressure (ITP) and subsequently improve spinal cord perfusion is unproven. A randomized trial of 22 patients demonstrated that drainage of cerebrospinal fluid, albeit a minimal amount, did not lead to a significant decrease in ITP nor did it cause any adverse events [36]. Monitoring surprisingly showed an increase in ITP following decompression in both groups possibly related to an elimination of the pressure gradient across the level of injury [36]. The pressure waveform was a useful measure of restoration of cerebrospinal fluid flow throughout the cord and intrathecal catheters may be useful for monitoring purposes. Although we do not routinely employ them at our institution, they have been used by the cardiothoracic surgery community following aortic aneurysm repair, in order to help prevent spinal cord ischemia, when spinal radiculomedullary arteries have been potentially compromised [37].

Pharmacologic treatment

Methylprednisolone

The National Acute Spinal Cord Injury Study (NASCIS) trials recommended that methylprednisolone be given in cases of blunt SCI less than 3 h old as a 30 mg/kg bolus followed by a 5.4 mg/kg/h infusion over 24 h, as well as for injuries 3–8 h-old as a bolus followed by a 24- to 48-h infusion [38, 39••]. For SCIs older than 8 h, NASCIS did not recommend giving steroids [38, 39••]. Based on new level 1 recommendations in 2013 by the AANS/CNS, methylprednisolone should not be given in the treatment of acute SCI because of associated harmful side effects including infections, sepsis, gastrointestinal bleeding, poor wound healing, psychiatric side effects, and even death [39••, 40••, 41, 42]. Therefore, the use of steroids have since started to fall out of favor among neurosurgeons over concern for potential complications [41]. At our

institution, across hospital services, we no longer use steroids in SCI patients. However, there still remains controversy regarding the new AANS/CNS guidelines, with some authorities stating that there is limited new evidence to justify this new recommendation [43••]. Those who disagree with the AANS/CNS guidelines acknowledge the concerns over the NASCIS trials, but point out that the trials support a small, but significant improvement in long-term motor function when methylprednisolone was initiated within 8 h of injury [43••]. These authors go on to state that there were only weak trends toward higher risks of infections and gastrointestinal complications and thus they believe that the use of steroids should be left to the discretion of the physician, in order to balance risk and benefit [43••]. They go on to advocate strongly for the consideration of methylprednisolone in patients with cervical SCI undergoing decompression based on a Cochrane meta-analysis demonstrating that these patients experience some benefit [43••, 44••].

GM-1 ganglioside (Sygen)

GM-1 mimics endogenous neurotrophic factors by stimulating nerve growth and repair, and reducing glutamate-mediated excitotoxicity [7••]. A phase 3 randomized controlled trial failed to show significant benefit, and the updated AANS/CNS guidelines state that the administration of GM-1 ganglioside for the treatment of acute SCI is not recommended [40••].

Surgery

Surgery

The details of intraoperative decision-making and operative techniques are beyond the scope of this text (ie, anterior, lateral, posterior, and combined approaches), but the overall goals of surgery and key points of when to operate will be briefly discussed. Overall, the decision to operate is made on a case-by-case basis, with the main goals to (1) decompress the neural elements in order to preserve or improve neurologic function, (2) establish a stable spine in order to prevent neurologic decline and/or deformity, and (3) return the patient to a higher functional capacity by allowing for mobilization. As with any surgical intervention, the risks and benefits must be weighed against nonoperative treatments.

The classic indication for surgery has been neurologic compromise and deteriorating neurologic function in the setting of an operative lesion. For example, the "Surgical Timing In Acute Spinal Cord Injury Study" (STASCIS) demonstrated that decompression within 24 h of injury is associated with improved neurologic outcome, defined as at least a two-grade improvement on the ASIA scale at 6-month follow-up [45••]. Impingement of the neural elements, particularly the spinal cord, can be relieved with decompression of the compressive forces, which can range from bony fractures, spondylosis, spondylolisthesis, disc herniation, as well as a hypertrophied ligament. Decompression is thought to help prevent further primary injury, as well as secondary injury from ischemia, vasospasm, edema, inflammation, free radicals, and apoptosis [7••]. Following decompression, the amount of tissue removed from the spinal column both dictates immediate stability and predicts future stability. If the spine is unstable preoperatively, it will likely be made unstable with

decompression and/or decompression will lead to a greater likelihood of instability in the future. The increased risk of instability pushes the surgeon toward performing a fusion operation. Besides preventing further neurologic deterioration, repair of an unstable spine at an earlier time point will accelerate the rehabilitation process. In addition, surgery will help prevent many of the complications seen with prolonged immobilization such as pneumonia, deep venous thrombosis, pulmonary emboli, as well as decubitus ulcers [46]. Finally, earlier stabilization of the spine can help prevent deformity and later, progressive pain and neurologic deterioration [46].

In terms of the cervical spine, the unique anatomy of the occipitocervical junction makes anterior approaches difficult, and, thus, stabilization is usually from a posterior approach with fusion [7••]. Ligaments do not normally heal well and thus nonoperative management of ligamentous disruption is unlikely to be successful. This is in contrast to ligamentous injury, which can heal nonoperatively. Isolated C1 and C2 fractures rarely cause spinal cord injury given the wide spine column, but fractures in the subaxial spine have a higher rate of SCI and more often need operative intervention [7••].

In the thoracic spine, it is important to assess the degree of spinal canal compromise and neurologic injury, as these elements will determine if surgery is required. As discussed above, if there is no neurologic injury and no compression of the neural elements, fractures can usually be managed conservatively. However, some fractures that involve all three columns of the spine may not heal conservatively, and the potential need for surgical stabilization will need to be discussed with a spine surgeon [7••]. Potential accepted indications for surgical intervention in the thoracic spine include incomplete neurologic injury, greater than 50 % vertebral body height loss with posterior ligamentous disruption, fracture dislocations, and three-column injury [7••, 47]. It should be noted that in patients for whom deformity is not corrected and bony compression remains, cerebrospinal fluid obstruction can lead to a post-traumatic syrinx and cause delayed neurologic deterioration above the level of the injury [48]. Posterior decompression alone in the thoracic spine will promote kyphosis and could worsen neurologic injury, and, thus, fusion should be considered. In addition, if the anterior and middle columns (vertebral body) are disrupted, a posterior decompression alone will lead to a very unstable spine, and, thus, fusion is also indicated.

In the lower lumbar spine, given the stability of the pelvis and iliolumbar ligaments, fractures without neurologic injury can be potentially treated with bracing alone [49]. As in the thoracic spine, surgical intervention for incomplete neurologic injury has good evidence [7••] and is recommended even in patients with complete neurologic injury as decompression and stabilization allows for shorter ICU stays, decreases morbidity from recumbency, decreases pain, decreases deformity, and protects from potential future neurologic deterioration [7••, 50].

Emerging therapies

Hypothermia

Hypothermia by reducing metabolic demand has been shown to be protective in several randomized trials of traumatic brain injury, but no trials have been conducted specifically for SCI [7••]. Mild hypothermia has been shown to result in improved neurologic function and reduced histopathologic damage in several animal models [51–53]. A retrospective analysis comparing 14

complete cervical ASIA A patients treated with 48 h of modest (33 °C) intravascular hypothermia to 14 age-matched historical controls and six patients (42.8 %) who were incomplete at approximately 1-year follow-up [54], suggested some benefit. A series of 20 patients demonstrated encouraging results for neurologic improvement, but randomized clinical trials are needed to confirm this data [55]. At this time, hypothermia remains experimental for SCI, and we have not been employing it for our SCI patients. We do advocate for normothermia and strict avoidance of hyperthermia using Tylenol, cooling blankets, ice, and an Arctic Sun as needed.

Pharmacologic neuroprotective agents

Several agents have been tried to date without benefit: Gacyclidine, a noncompetitive NMDA receptor antagonist; nimodipine; naloxone; and GM-1 ganglioside [7••]. New trials include riluzole, a sodium channel-blocking anticonvulsant drug that is approved for the treatment of amyotrophic lateral sclerosis and that protects against sodium and glutamate-mediated secondary injuries. Given an already well-established safety profile, a phase 1 trial was completed in 2012 [56•]. Neurologic outcomes were also examined and were promising, thus, paving the way for larger randomized trials [56•]. Many other agents and molecular pathways are being looked at in animal models but are not ready for primetime, and, thus, we do not discuss them here. For those interested, the reference supplied is an outstanding resource for those interested in SCI [7••].

Compliance with Ethics Guidelines

Conflict of Interest

Ryan A. Grant, Jennifer L. Quon, and Khalid M. Abbed declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. National Spinal Cord Injury Statistical Center (University of Alabama at Birmingham): Spinal cord injury facts and figures. 2013; http://www.nscisc.uab.edu. Accessed Sept 2014.
2. Sekhon LH, Fehlings MG. Epidemiology, demographics, and pathophysiology of acute spinal cord injury. Spine. 2001;26(24 Suppl):S2–12.
3.• Thompson C, Mutch J, Parent S, Mac-Thiong JM. The changing demographics of traumatic spinal cord injury: an 11-year study of 831 patients. J Spinal Cord Med. 2014. doi: 10.1179/2045772314Y.0000000233. Discusses the current demographics of SCI, as well as the increase in elderly injuries.
4. Jackson AB, Dijkers M, Devivo MJ, Poczatek RB. A demographic profile of new traumatic spinal cord injuries: change and stability over 30 years. Arch Phys Med Rehabil. 2004;85(11):1740–8.
5. Myers ER, Wilson SE. Biomechanics of osteoporosis and vertebral fracture. Spine. 1997;22(24 Suppl):25s–31.
6. Tator CH, Fehlings MG. Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. J Neurosurg. 1991;75(1):15–26.

7.•• Fehlings MG, Vaccaro AR, Boakye M, Rossignol S, Ditunno JF, Burns AS. Essentials of spinal cord injury: basic research to clinical practice. Thieme 2013. Outstanding textbook reviewing the current basic science, clinical science, and evidence for the contemporary treatment of SCI, as well as emerging therapies. For those interested in SCI, we recommend referencing this text.

8.•• Hadley MN, Walters BC, Aarabi B, et al. Clinical assessment following acute cervical spinal cord injury. Neurosurgery. 2013;72 Suppl 2:40–53.

Part of the new AANS/CNS 2013 spine guidelines reviewing the most relevant literature regarding the neurologic examination and making recommendations for clinical management based on the level of evidence.

9.•• Furlan JC, Noonan V, Singh A, Fehlings MG. Assessment of impairment in patients with acute traumatic spinal cord injury: a systematic review of the literature. J Neurotrauma. 2011;28(8):1445–77.

ASIA classification is the most important for SCI prognostication, but is not reliable in the first 72 h.

10. Ramani PS. Surgical techniques in spinal surgery: cervical spine. Jaypee Brothers Medical Publishers. New Delhi, India; 2010.

11.•• van Middendorp JJ, Goss B, Urquhart S, Atresh S, Williams RP, Schuetz M. Diagnosis and prognosis of traumatic spinal cord injury. Global Spine J. 2011;1(1):1–8.

A study demonstrating that over one-third of SCI patients presenting only with sacral sensory sparing will go on to ambulate within 1 year.

12. Stover SL. Review of forty years of rehabilitation issues in spinal cord injury. J Spinal Cord Med. 1995;18 (3):175–82.

13. Kirshblum SC, O'Connor KC. Predicting neurologic recovery in traumatic cervical spinal cord injury. Arch Phys Med Rehabil. 1998;79(11):1456–66.

14.•• Ryken TC, Hadley MN, Walters BC, et al. Radiographic assessment. Neurosurgery. 2013;72 Suppl 2:54–72.

Part of the new AANS/CNS 2013 spine guidelines reviewing the most relevant literature regarding radiological workup of patients with SCI and making recommendations of modality based on the level of evidence.

15. Muchow RD, Resnick DK, Abdel MP, Munoz A, Anderson PA. Magnetic resonance imaging (MRI) in the clearance of the cervical spine in blunt trauma: a meta-analysis. J Trauma. 2008;64(1):179–89.

16. Kwon BK, Vaccaro AR, Grauer JN, Fisher CG, Dvorak MF. Subaxial cervical spine trauma. J Am Acad Orthop Surg. 2006;14(2):78–89.

17. Bucholz RW, Burkhead WZ. The pathological anatomy of fatal atlanto-occipital dislocations. J Bone Joint Surg (Am Vol). 1979;61(2):248–50.

18. Allen Jr BL, Ferguson RL, Lehmann TR, O'Brien RP. A mechanistic classification of closed, indirect fractures and dislocations of the lower cervical spine. Spine. 1982;7(1):1–27.

19.•• Harrigan MR, Hadley MN, Dhall SS, et al. Management of vertebral artery injuries following non-penetrating cervical trauma. Neurosurgery. 2013;72 Suppl 2:234–43.

Part of the new AANS/CNS 2013 spine guidelines reviewing the most relevant literature regarding verterbral artery injury and recommending a CTA as the imaging modality of choice, as well as consultation with neurosurgery and stroke neurology to discuss further medical management and potential surgical/interventional options.

20. Tator CH. Experimental and clinical studies of the pathophysiology and management of acute spinal cord injury. J Spinal Cord Med. 1996;19(4):206–14.

21.•• Ryken TC, Hurlbert RJ, Hadley MN, et al. The acute cardiopulmonary management of patients with cervical spinal cord injuries. Neurosurgery. 2013;72 Suppl 2:84–92.

Part of the new AANS/CNS 2013 spine guidelines reviewing the most relevant literature regarding cardiopulmonary managment of patients with SCI and making recommendations for clinical management based on the level of evidence.

22. Ball PA. Critical care of spinal cord injury. Spine. 2001;26(24 Suppl):S27–30.

23. Blood pressure management after acute spinal cord injury. Neurosurgery. 2002;50(3 Suppl):S58–62.

24. Krassioukov A, Warburton DE, Teasell R, Eng JJ. A systematic review of the management of autonomic dysreflexia after spinal cord injury. Arch Phys Med Rehabil. 2009;90(4):682–95.

25. Illman A, Stiller K, Williams M. The prevalence of orthostatic hypotension during physiotherapy treatment in patients with an acute spinal cord injury. Spinal Cord. 2000;38(12):741–7.

26. Hassid VJ, Schinco MA, Tepas JJ, et al. Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. J Trauma. 2008;65(6):1328–32.

27. Cotton BA, Pryor JP, Chinwalla I, Wiebe DJ, Reilly PM, Schwab CW. Respiratory complications and mortality risk associated with thoracic spine injury. J Trauma. 2005;59(6):1400–7.

discussion 1407–1409.

28. Gardner BP, Watt JW, Krishnan KR. The artificial ventilation of acute spinal cord damaged patients: a retrospective study of forty-four patients. Paraplegia. 1986;24(4):208–20.

29. Shatney CH, Brunner RD, Nguyen TQ. The safety of orotracheal intubation in patients with unstable cervical spine fracture or high spinal cord injury. Am J Surg. 1995;170(6):676–9.

discussion 679–80.

30. McMichan JC, Michel L, Westbrook PR. Pulmonary dysfunction following traumatic quadriplegia. Recognition, prevention, and treatment. JAMA. 1980;243 (6):528–31.

31. Jackson AB, Groomes TE. Incidence of respiratory complications following spinal cord injury. Arch Phys Med Rehabil. 1994;75(3):270–5.

32. Estenne M, De Troyer A. Mechanism of the postural dependence of vital capacity in tetraplegic subjects. Am Rev Respir Dis. 1987;135(2):367–71.

33. Goldman JM, Rose LS, Williams SJ, Silver JR, Denison DM. Effect of abdominal binders on breathing in tetraplegic patients. Thorax. 1986;41(12):940–5.
34. Ploumis A, Ponnappan RK, Maltenfort MG, et al. Thromboprophylaxis in patients with acute spinal injuries: an evidence-based analysis. J Bone Joint Surg (Am Vol). 2009;91(11):2568–76.
35. Johns JS, Nguyen C, Sing RF. Vena cava filters in spinal cord injuries: evolving technology. J Spinal Cord Med. 2006;29(3):183–90.
36. Kwon BK, Curt A, Belanger LM, et al. Intrathecal pressure monitoring and cerebrospinal fluid drainage in acute spinal cord injury: a prospective randomized trial. J Neurosurg Spine. 2009;10(3):181–93.
37. Fedorow CA, Moon MC, Mutch WA, Grocott HP. Lumbar cerebrospinal fluid drainage for thoracoabdominal aortic surgery: rationale and practical considerations for management. Anesth Analg. 2010;111(1):46–58.
38. Ducker TB, Zeidman SM. Spinal cord injury. Role of steroid therapy. Spine. 1994;19(20):2281–7.
39.•• Bydon M, Lin J, Macki M, Gokaslan ZL, Bydon A. The Current role of steroids in acute spinal cord injury. World Neurosurg. 2013;82(5):848-854. Nice review discussing the methyprednisolone controversary and current evidence of use in SCI.
40.•• Hurlbert RJ, Hadley MN, Walters BC, et al. Pharmacological therapy for acute spinal cord injury. Neurosurgery. 2013;7 Suppl 2:93–105.
Part of the new AANS/CNS 2013 spine guidelines reviewing the most relevant literature regarding steroid use in patients with SCI and making a level 1 recommendation against the use of steroids.
41. Schroeder GD, Kwon BK, Eck JC, Savage JW, Hsu WK, Patel AA. Survey of cervical spine research society members on the use of high-dose steroids for acute spinal cord injuries. Spine. 2014;39(12):971–7.
42. Khan MF, Burks SS, Al-Khayat H, Levi AD. The effect of steroids on the incidence of gastrointestinal hemorrhage after spinal cord injury: a case-controlled study. Spinal Cord. 2014;52(1):58–60.
43.•• Fehlings MG, Wilson JR, Cho N. Methylprednisolone for the treatment of acute spinal cord injury: counterpoint. Neurosurgery. 2014;61 Suppl 1:36–42.
High profile counterpoint advocating that AANS/CNS level-1 guideline against the use of steroids in SCI is unfounded and that steroids have their role in certain patient populations.
44.•• Bracken MB. Steroids for acute spinal cord injury. Cochrane Database Syst Rev. 2012;1:Cd001046. Another outstanding review of the current evidence for the use of steroids in SCI.
45.•• Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). PLoS One. 2012;7(2):e32037.
The STASCIS trial found that surgery prior to 24 h is associated with improved neurologic outcome, defined as at least a 2 grade improved in ASIA scoring at 6 month follow-up.
46. Duh MS, Shepard MJ, Wilberger JE, Bracken MB. The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. Neurosurgery. 1994;35(2):240–8.
discussion 248–9.
47. Cantor JB, Lebwohl NH, Garvey T, Eismont FJ. Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing. Spine. 1993;18(8):971–6.
48. Holly LT, Johnson JP, Masciopinto JE, Batzdorf U. Treatment of posttraumatic syringomyelia with extradural decompressive surgery. Neurosurg Focus. 2000;8(3):E8.
49. Seybold EA, Sweeney CA, Fredrickson BE, Warhold LG, Bernini PM. Functional outcome of low lumbar burst fractures. A multicenter review of operative and nonoperative treatment of L3–L5. Spine. 1999;24(20):2154–61.
50. Dai LD. Low lumbar spinal fractures: management options. Injury. 2002;33(7):579–82.
51. Marion D, Bullock MR. Current and future role of therapeutic hypothermia. J Neurotrauma. 2009;26(3):455–67.
52. Batchelor PE, Kerr NF, Gatt AM, et al. Hypothermia prior to decompression: buying time for treatment of acute spinal cord injury. J Neurotrauma. 2010;27(8):1357–68.
53. Strauch JT, Lauten A, Spielvogel D, et al. Mild hypothermia protects the spinal cord from ischemic injury in a chronic porcine model. Eur J Cardiothorac Surg. 2004;25(5):708–15.
54. Levi AD, Casella G, Green BA, et al. Clinical outcomes using modest intravascular hypothermia after acute cervical spinal cord injury. Neurosurgery. 2010;66(4):670–7.
55. Hansebout RR, Hansebout CR. Local cooling for traumatic spinal cord injury: outcomes in 20 patients and review of the literature. J Neurosurg Spine. 2014;20(5):550–61.
56.•• Wilson JR, Fehlings MG. Riluzole for acute traumatic spinal cord injury: a promising neuroprotective treatment strategy. World Neurosurg. 2014;81(5–6):825–9.
Riluzole is an exciting and potentially new medication to treat SCI, that already has a well known safetyprofile given its use in ALS.