Chapter 2
Considerations for Spinal Cord Injury in the Athlete

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Introduction

In 1969, Frankel and colleagues first attempted to define spinal cord injuries [1]. In 1982, this was expanded by the American Spinal Injury Association (ASIA) with the addition of a 0–5 motor scale of 10 predefined motor groups, representing specific motor distributions. Today, the ASIA scale is the preferred method of choice utilized as a neurologic examination tool in the diagnosis of acute SCI [2] (Tables 2.1 and 2.2).

A catastrophic cervical spine injury occurs when there is a structural distortion of the cervical spinal column associated with actual or potential damage to the spinal cord [3]. In the cervical spine, sports-related injuries are grouped into three separate categories. This classification has the additional utility to aid decisions

Table 2.1 ASIA impairment scale

A – Complete	No sensory or motor function is preserved in the sacral segments S4–5
B – Sensory incomplete	Sensory but not motor function is preserved below the neurologic level and includes the sacral segments S4–5, with no motor function preserved more than three levels below the motor level on either side of the body
C – Motor incomplete	Motor function is preserved below the neurologic level, and more than half of key muscle functions below the neurologic level of injury have a muscle grade less than 3
D – Motor incomplete	Motor function is preserved below the neurologic level, and at least half of key muscle functions below that level have a strength grade greater or equal to 3
E – Normal	If sensation and motor testing are normal but the patient had deficits on prior evaluations

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Table 2.2 Key muscle group tested in ASIA evaluation

C5	Elbow flexors	L2	Hip flexors
C6	Wrist extensors	L3	Knee extensors
C7	Elbow extensors	L4	Ankle dorsiflexors
C8	Long finger flexor	L5	Great toe extension
T1	Small finger abduction	S1	Ankle plantar flexion

regarding the safe return to play for the athlete [4]. When a type 1 injury occurs, the athlete sustains a permanent SCI. A permanent SCI encompasses those with complete paralysis as well as incomplete SCI syndromes. In an athlete with normal radiographic studies, but deficits which completely resolve within minutes to hours, a type 2 injury is diagnosed. Finally, type 3 injuries include those with radiographic abnormalities without associated neurologic deficits.

Prehospital Immobilization and Transportation

It is critical that athletes with a SCI be assessed and managed in the immediate period of injury on the field as any standard trauma patient. This involves a systematic approach to rapidly assess the extent of injuries and begin life-preserving therapy established in the Advanced Trauma Life Support (ATLS) protocol, which emphasizes addressing airway, breathing, and circulation status. After initial stabilization by medical personal on the field, the athlete can be transported off the field while maintaining strict immobilization of the spine.

Neurologic deficits can develop after treatment has begun if proper immobilization is not utilized. In a 1983 publication, Podolsky and colleagues reported that up to 25% of spinal cord injuries had been caused by or worsened under medical care [5]. While this number may be an overestimate, it emphasizes the importance of safe transport and initial stabilization of the athlete with a possible SCI. On the field, the athlete is immobilized with a cervical collar and a spine backboard, and the head is secured. It is important to note that although a cervical collar can effectively stabilize most cervical injuries, with complete ligamentous disruption, the collar has minimal effect, emphasizing the importance of manual stabilization in these instances [6]. Patients with a SCI should be transferred immediately to a center that specializes in SCI, which has been linked to better neurologic outcomes, reduced length of stay, fewer complications, and reduced mortality [7, 8]. Upon arrival at the hospital, the helmet and shoulder pads should be removed, if they are still in place, before radiographic examination. Of note, logroll maneuvers should be avoided with employment of a lift-and-slide technique preferred, given that they create less motion of the injured segment [9]. After initial resuscitation and radiographic evaluation, decisions can be made regarding the management of the injury.

Unstable spine injuries should be initially reduced and temporarily stabilized with cervical traction (Gardner-Wells tongs or halo device). In cases where relevant,

early cervical traction for reduction of cervical fractures/dislocations is recommended to optimize alignment and minimize compression of the spinal cord [10, 11]. It is of critical importance to obtain a contrast-enhanced CT or MRI prior to reduction to ensure the absence of a herniated disc which can worsen a SCI upon attempted reduction in this setting.

Adjunct Treatment/Pathophysiology of Spinal Cord Injury

To understand the currently investigated adjunct treatment options, a basic understanding of the pathophysiology of SCI is essential. An acute SCI can be thought of as an initial traumatic primary injury with a secondary injury that follows as a result of the progressive cascade of events that results in tissue destruction and systemic autonomic consequences.

The primary injury results from a mechanical insult to the spinal cord most commonly a result of failure of the integrity of the spinal column, leading to compressive and often sustained forces on the spinal cord. The result is disruption of neuronal axons, blood vessels, and cell membranes [12, 13]. This triggers a cascade of processes that define the secondary injury phase.

During the secondary injury phase, necrosis results from mechanical disruption of cellular membranes, with simultaneous upregulation of cytokines and release of glutamate, which may reach excitotoxic levels [14]. Ongoing hemorrhage with increasing edema continues with ischemia resulting from local effects (i.e., thrombosis, vasospasm, microvascular disruption) as well as from systemic autonomic effects on the cardiovascular system caused by the SCI itself. The resultant hypoxia leads to impaired neuronal homeostasis and further cell death [14]. The cellular inflammatory response, driven predominantly by macrophages, is thought of as the primary mediator of the progressive secondary injury. Through regulation of perfusion pressure and the potential addition of a neuroprotective agent/strategy, the early stages of the secondary injury are thought to be critical areas where medical intervention can benefit the patient.

Depending on the level of injury, SCI can be complicated by respiratory and cardiovascular dysfunction. Innervation to the muscles of inspiration and expiration may be compromised leading to decreased forced vital capacity and peak expiratory flow rate [15, 16]. This can lead to insufficient oxygen delivery to the spinal cord, which can be further worsened by systemic hypotension resulting from traumatic disruption of the descending vasomotor pathways of the spine. These carry supraspinal innervation to the preganglionic sympathetic neurons in the intermediolateral cell column between T1 and L2. Hypotension results from decreased sympathetic supply to the peripheral vascular system, and bradycardia may occur due to unopposed parasympathetic supply to the heart through the intact vagal nerve [17]. Lehmann and colleagues found that patients with severe cervical SCI are more likely to have bradycardia, hypotension, and cardiac dysrhythmias than patients with mild cervical SCI or thoracolumbar injury [18]. In addition to the aforemen-

tioned mechanisms of impaired ventilation, any pulmonary injury itself may be present and leads to poor gas exchange and decreased lung compliance. Furthermore, painful chest wall injuries may decrease ventilation.

It is recommended that hypotension be corrected as soon as possible with a goal mean arterial blood pressure maintained between 85 and 90 mmHg for the first 7 days following an acute SCI [19]. If a pressor is needed, norepinephrine is favored with dobutamine as second line when increased cardiac output is desired. Phenylephrine should be avoided in patients with a SCI level above T6 due to its proclivity to trigger reflex bradycardia as it is purely a peripheral vasoconstrictor.

Corticosteroid Administration

The 2013 American Association of Neurological Surgeons and the Congress of Neurological Surgeons (AANS/CNS) Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injury included the level 1 recommendation that the administration of methylprednisolone sodium succinate (MPSS) is not recommended [20]. MPSS has been the most extensively studied steroid in the medical management of acute SCI and is thought to work by its anti-inflammatory effects and halting peroxidation of neuronal membrane lipids [21–23]. The most frequently cited studies in the use of MPSS in acute SCI are the three National Acute Spinal Cord Injury Study (NASCIS) trials [24–26]. In all of the primary analyses, no significant difference was detected in motor, sensory, or functional recovery. However, post hoc analyses of NASCIS II data demonstrated that those receiving MPSS (30 mg/kg bolus at admission followed by 5.4 mg/kg/h for 23 h) within 8 h of injury improved significantly in both sensory and motor functions [26]. These differences remained significant 1 year post-injury. Additional post hoc analyses of NASCIS III data showed significantly greater motor recovery if a 48-h MPSS protocol (30 mg/kg bolus at admission followed by 5.4 mg/kg/h for 47 h) was used instead of a 24-h protocol, when treatment was started within 3–8 h [26]. The results from the third study also demonstrated no benefit to extending treatment past 24 h if MPSS was administered within the first 3 h after SCI. However, the 48-h MPSS protocol did show an increased incidence of severe pneumonia and severe sepsis ($p = 0.02$ and $p = 0.07$, respectively). High-dose MPSS has also been associated with increased prevalence of wound infections and death due to respiratory complications. Despite increased morbidity, there is no demonstration of increase in mortality with MPSS use [27].

Of note, there also appears to be a relationship between surgical timing and the safety of MPSS in acute SCI. In a multivariate analysis performed by Fehlings and colleagues, the primary data from STASCIS demonstrated that the 24-h MPSS protocol in combination with early surgery predicted significantly improved neurologic recovery at 6 months [28]. Particular consideration should also be given to the athlete with a cervical SCI. In this population, improvement in motor function is likely to have the greatest impact [29].

Hypothermia

The early induction of hypothermia has also been anecdotally reported to be beneficial in acute SCI. The mechanism for the neuroprotective benefit of systemic therapeutic hypothermia has yet to be elucidated [30]. It is hypothesized to result from reductions in cellular apoptosis [31], inflammation [32], glutamate excitotoxicity [33], edema, and other additional factors. In a phase I trial in patients with acute SCI, 14 patients were treated with 48 h of 33 °C intravascular hypothermia [34]. At the 1-year follow-up, 6/14 (42.9%) converted from complete SCI to incomplete. This is favorable considering the commonly reported value of 20% reported in the literature [35]. This underpowered study gathered enough data to garner support for further studies.

Medications

Vast research has been conducted on potential pharmacologic agents that aid in neuroprotection; unfortunately few therapeutic benefits have been realized from these studies. The three agents with the most current literature are GM-1 ganglioside, riluzole, and minocycline [36]. GM-1 ganglioside is an endogenous substance found in the mammal central nervous system and has shown to be anti-cytotoxic and anti-apoptotic. Preclinical animal trials demonstrated improvement in motor score at 3–5 days post-injury. Phase II trials in humans have shown to improve ASIA motor score at 1-year post-injury. However, phase III randomized control trial (RCT) showed no difference in motor scores at 52-week follow-up. Riluzole has been another highly investigated pharmacologic agent; it is a sodium channel blocker currently used in amyotrophic lateral sclerosis (ALS). Preclinical and phase I/II studies have shown improvement in ASIA motor score with its administration. It is currently undergoing a multicenter phase III RCT. Minocycline is a tetracycline antibiotic with anti-inflammatory properties. Preclinical and phase I studies have demonstrated improved motor scores, and phase II/III studies are currently underway. In addition to the above agents, newer neuroprotective and neuroregenerative therapies continue to be studied. As SCI research expands, providers will need to remain up to date with developing evidence-based standards.

Surgical Timing

Evidence exists that persistent compression of the spinal cord is a reversible form of secondary injury [37]. The Surgical Timing Acute Spinal Cord Injury Scale (STASCIS) was an international, multicenter prospective cohort study designed to determine whether early decompression (within 24 h) versus late (after 24 h) was

more beneficial after traumatic cervical SCI. An improvement of two or more grades of the ASIA Impairment Scale (AID) was seen in 19.8% of early surgery patients compared to 8.8% in the late surgery patients [28]. This consensus for early decompression has been demonstrated beneficial in the thoracolumbar region [38] and cauda equina syndrome [39]. From these studies it can be concluded that when feasible, early decompression is desirable.

Expert Opinion

- Acute SCI is an initial traumatic injury with a secondary injury due to a biochemical cascade. Since the initial injury has already occurred, most modalities of management focus on reducing the secondary injury cascade.
- Initial care should concentrate on removing mechanical compression of the cord and maintaining spinal cord perfusion.
- Mechanical compression on the cord can be removed via reduction techniques and/or surgery. There is evidence to support better outcomes with early surgical intervention.
- Avoidance of spinal cord hypoperfusion is of the utmost importance and should be emphasized as soon as the SCI is diagnosed. Current recommendations would suggest maintaining a mean arterial pressure (MAP) greater than 85–90 mmHg for 7 days post-injury.
- Although the NASCIS trials demonstrate complications with steroid administration, there is also evidence of neurologic improvement. Given that most athletic SCIs are likely to be isolated injuries, without the same comorbidities of the general trauma population, they may be a population ideally suited for high-dose steroid administration. Since the benefits outweigh the risks, we would recommend athletes with an isolated SCI receive IV steroids for acute SCI. This assumes administration of the steroids within 8 h of injury.
- Pharmacologic agents focused on neuroprotection and regeneration remain in their infancy. Further research is warranted before these promising modalities can be utilized in standard practice.

References

1. Frankel HL. Ascending cord lesion in the early stages following spinal injury. Paraplegia. 1969;7(2):111–8.
2. Committee AIS, Committee AE, Rupp R. Assessor accuracy of the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI)-recommendations for reporting items. Spinal Cord. 2018;56(8):819–20.
3. Banerjee R, Palumbo MA, Fadale PD. Catastrophic cervical spine injuries in the collision sport athlete, part 2: principles of emergency care. Am J Sports Med. 2004;32(7):1760–4.
4. Bailes JE, Hadley MN, Quigley MR, Sonntag VK, Cerullo LJ. Management of athletic injuries of the cervical spine and spinal cord. Neurosurgery. 1991;29(4):491–7.

5. Podolsky SM, Hoffman JR, Pietrafesa CA. Neurologic complications following immobilization of cervical spine fracture in a patient with ankylosing spondylitis. Ann Emerg Med. 1983;12(9):578–80.
6. Rechtine GR, Del Rossi G, Conrad BP, Horodyski M. Motion generated in the unstable spine during hospital bed transfers. J Trauma. 2004;57(3):609–11; discussion 11–2.
7. Theodore N, Hadley MN, Aarabi B, et al. Prehospital cervical spinal immobilization after trauma. Neurosurgery. 2013;72 Suppl 2:22–34.
8. Parent S, Mac-Thiong JM, Roy-Beaudry M, Sosa JF, Labelle H. Spinal cord injury in the pediatric population: a systematic review of the literature. J Neurotrauma. 2011;28(8):1515–24.
9. Del Rossi G, Horodyski MH, Conrad BP, Di Paola CP, Di Paola MJ, Rechtine GR. The 6-plus-person lift transfer technique compared with other methods of spine boarding. J Athl Train. 2008;43(1):6–13.
10. Gelb DE, Aarabi B, Dhall SS, et al. Treatment of subaxial cervical spinal injuries. Neurosurgery. 2013;72 Suppl 2:187–94.
11. Gelb DE, Hadley MN, Aarabi B, et al. Initial closed reduction of cervical spinal fracture-dislocation injuries. Neurosurgery. 2013;72 Suppl 2:73–83.
12. Kakulas BA. A review of the neuropathology of human spinal cord injury with emphasis on special features. J Spinal Cord Med. 1999;22(2):119–24.
13. Rowland JW, Hawryluk GW, Kwon B, Fehlings MG. Current status of acute spinal cord injury pathophysiology and emerging therapies: promise on the horizon. Neurosurg Focus. 2008;25(5):E2.
14. 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.
15. Berlly M, Shem K. Respiratory management during the first five days after spinal cord injury. J Spinal Cord Med. 2007;30(4):309–18.
16. Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. Am Rev Respir Dis. 1981;124(1):41–4.
17. Furlan JC, Fehlings MG. Cardiovascular complications after acute spinal cord injury: pathophysiology, diagnosis, and management. Neurosurg Focus. 2008;25(5):E13.
18. Lehmann KG, Lane JG, Piepmeier JM, Batsford WP. Cardiovascular abnormalities accompanying acute spinal cord injury in humans: incidence, time course and severity. J Am Coll Cardiol. 1987;10(1):46–52.
19. 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.
20. Hurlbert RJ, Hadley MN, Walters BC, et al. Pharmacological therapy for acute spinal cord injury. Neurosurgery. 2013;72 Suppl 2:93–105.
21. Hall ED, Braughler JM. Effects of intravenous methylprednisolone on spinal cord lipid peroxidation and (Na+ + K+)-ATPase activity. Dose-response analysis during 1st hour after contusion injury in the cat. J Neurosurg. 1982;57(2):247–53.
22. Hawryluk GW, Rowland J, Kwon BK, Fehlings MG. Protection and repair of the injured spinal cord: a review of completed, ongoing, and planned clinical trials for acute spinal cord injury. Neurosurg Focus. 2008;25(5):E14.
23. Saville LR, Pospisil CH, Mawhinney LA, et al. A monoclonal antibody to CD11d reduces the inflammatory infiltrate into the injured spinal cord: a potential neuroprotective treatment. J Neuroimmunol. 2004;156(1–2):42–57.
24. Bracken MB, Collins WF, Freeman DF, et al. Efficacy of methylprednisolone in acute spinal cord injury. JAMA. 1984;251(1):45–52.
25. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Engl J Med. 1990;322(20):1405–11.
26. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. JAMA. 1997;277(20):1597–604.

27. Chikuda H, Yasunaga H, Takeshita K, et al. Mortality and morbidity after high-dose methylprednisolone treatment in patients with acute cervical spinal cord injury: a propensity-matched analysis using a nationwide administrative database. Emerg Med J. 2014;31(3):201–6.
28. 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.
29. Fehlings MG, Wilson JR, Cho N. Methylprednisolone for the treatment of acute spinal cord injury: counterpoint. Neurosurgery. 2014;61 Suppl 1:36–42.
30. Batchelor PE, Skeers P, Antonic A, et al. Systematic review and meta-analysis of therapeutic hypothermia in animal models of spinal cord injury. PLoS One. 2013;8(8):e71317.
31. Shibuya S, Miyamoto O, Janjua NA, Itano T, Mori S, Norimatsu H. Post-traumatic moderate systemic hypothermia reduces TUNEL positive cells following spinal cord injury in rat. Spinal Cord. 2004;42(1):29–34.
32. Chatzipanteli K, Yanagawa Y, Marcillo AE, Kraydieh S, Yezierski RP, Dietrich WD. Posttraumatic hypothermia reduces polymorphonuclear leukocyte accumulation following spinal cord injury in rats. J Neurotrauma. 2000;17(4):321–32.
33. Farooque M, Hillered L, Holtz A, Olsson Y. Effects of moderate hypothermia on extracellular lactic acid and amino acids after severe compression injury of rat spinal cord. J Neurotrauma. 1997;14(1):63–9.
34. 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.
35. van Middendorp JJ, Hosman AJ, Pouw MH, Group E-SS, Van de Meent H. ASIA impairment scale conversion in traumatic SCI: is it related with the ability to walk? A descriptive comparison with functional ambulation outcome measures in 273 patients. Spinal Cord. 2009;47(7):555–60.
36. Haller J, Bice M, Lawrence B. Mediating the secondary effects of spinal cord injury through optimization of key physiologic parameters. J Am Acad Orthop Surg. 2016;24(3):160–71.
37. Tator CH. Recognition and management of spinal cord injuries in sports and recreation. Neurol Clin. 2008;26(1):79–88; viii.
38. Cengiz SL, Kalkan E, Bayir A, Ilik K, Basefer A. Timing of thoracolumbar spine stabilization in trauma patients; impact on neurological outcome and clinical course. A real prospective (rct) randomized controlled study. Arch Orthop Trauma Surg. 2008;128(9):959–66.
39. Chau AM, Xu LL, Pelzer NR, Gragnaniello C. Timing of surgical intervention in cauda equina syndrome: a systematic critical review. World Neurosurg. 2014;81(3–4):640–50.