Pediatric Spinal Cord Injury: Recognition of Injury and Initial Resuscitation, in Hospital Management, and Coordination of Care

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Abstract

Spinal cord injury is uncommon in the pediatric population with a lifelong impact for the patient and family. Knowledge of spine embryology, mechanisms of injury that lead to specific injuries, appropriate utilization of radiographic imaging based on suspected injury, prehospital and hospital management of various spinal cord injuries is essential for providers attending to traumatically injured patients. In addition to patients who present with soft tissue and bony injuries diagnosed with clinical examination and confirmed with computed tomography or magnetic resonance imaging, it is important to note that the pediatric population is at a higher risk for spinal cord injury without radiographic abnormality than the adult population. Patients who survive the acute phase of injury face long-term rehabilitation and have an increased risk of depression and mortality. Understanding the long-term sequelae of spinal cord injuries is also an essential management component of traumatically injured children. A program that provides long-term rehabilitation, psychosocial and spiritual support, and adaptive environmental supports gives patients and their families the best opportunity for long-term recovery. A review of the current literature on the diagnosis, management, and follow-up of pediatric spinal cord injury is presented.

Keywords

- ▶ spinal cord injury
- ▶ spinal cord injury without radiographic abnormality
- ▶ spinal shock
- ▶ neurogenic shock

Background

Spinal cord injury (SCI) is uncommon in children. It occurs 1.99 times per 100,000 children,¹ and accounts for 1.5% of all pediatric trauma admissions.² The incidence of SCI without radiographic abnormality (SCIWORA) is less clear, but occurs more commonly in children less than 8 due to anatomical differences in the developing pediatric spine. Age, sex, and socioeconomic status independently influence the risk of SCI.³ Males are two times as likely to suffer a SCI and, in the United States, African Americans are the most likely ethnic group to suffer a SCI.¹ Cervical injuries make up 80% of all SCIs in children.¹ Of these, 18% have atlanto-occipital dislocation.⁴ Furthermore, concomitant head injury may be

present in up to three quarters of patients with SCI, a third of these patients will have severe traumatic brain injury (TBI).⁵

Adults suffer from SCI more frequently. Incidence varies greatly by geography. In the United States the incidence is between 23.7 and 77 SCIs per 1,000,000.⁵ Age at onset varies with the cause of injury. The National SCI Statistical Center reports 50% of SCIs occur between 16 and 30 year with 80.7% of SCIs occurring in males.⁶

The financial burden of SCIs is significant and varies greatly with the level of injury.⁶ High SCIs can cost up to over \$1,000,000 for the 1st year and just under \$200,000 for each subsequent year.⁶ If the patient is injured at 25 years of age, the estimated lifetime cost is just over \$4.5 million.⁶ If the resultant injury is incomplete motor function at any level,

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the lifetime burden of a patient injured at 25 years of age is \$1.5 million.⁶

Many review articles on SCIs in adults and children have focused on diagnosis and surgical options. The aim of this article is to review recognition of injury, initial resuscitation, in-hospital management, coordination of care within the pediatric hospital, and ambulatory follow-up.

Embryology

Understanding how the spine develops sheds light on the relationship between age and the type of injury experienced by the child. The notochord and somites give rise to the structure that will become the spine and spinal cord in vertebrates. Vertebral bodies ossify from fusion of the dorsal and ventral ossification centers, beginning at 7 week gestation and continues through the fetal period all the way into young adulthood.⁷ The atlas, providing specialized articulation and movement to the skull, ossifies the anterior arch during the 1st year of life and completes ossification of the posterior arch by 3 years,⁸ fusing by 7 years.⁹ The second cervical vertebra, the axis, develops two separate ossification centers by the 28th gestational week.² The axis will fuse with its odontoid process before 6 years of age.² A superior ossification center within the odontoid process will not finish fusing with the main body of the dens until 12 years of age.¹⁰ If this fusion does not occur, the adolescent and adult patient is at risk for atlantoaxial instability.¹¹ Complete ossification of C2 does not occur until the third decade.² In contrast, the subaxial spine has mature vertebral bodies by 6 years. The anterior transverse processes fuse to the vertebrae by 6 years as well.² The spinous processes and epiphyseal rings are fused to the vertebrae by 25 years of age.⁹

The spine in children less than 8 years is hypermobile. Because the head is relatively larger than in adults, the fulcrum of injury moves cephalad, putting the occipitoatlantoaxial complex at risk.^{2,12,13} The lack of complete ossification and lax ligaments increases the risk of ligamentous injuries. The nature of spine injury in children older than 8 years is similar to that in adults.^{2,14}

Mechanisms of Injury

Acceleration /Deceleration

Occipitoatlantal and atlantoaxial dislocation occur in young children during high-speed collisions, auto versus pedestrian, and/or related to airbag injuries.¹⁵ There may be partial or absent neurologic deficits. Traction and cervical collar placement can worsen this injury by distraction.¹⁶ Because subluxation in children may be radiographically occult, sagittal computed tomography (CT) images and magnetic resonance imaging (MRI) can be quite helpful.² The tectorial membrane, a strong fibrous ligament that fixes the axis with the occiput, is essential to protect the occipitoatlantal and atlantoaxial articulations against vertical distraction.¹⁷ Surgical fusion is required when this ligament is ruptured.

Odontoid injuries can occur in children less than 7 years after falls or high-speed collisions.^{18,19} Children secured in a forward-facing car seat are at particular risk.²⁰ This injury results from

avulsion of the dens off the body of the axis. This type of injury commonly has a fatal outcome. Lateral radiographs and reconstructed CT images will typically identify this injury.²¹ Prompt immobilization with or without a halo is commonly required.²¹

Subaxial ligamentous injuries are more common in patients older than 8 years of age²² usually induced by motor-vehicle collisions. Symptom severity correlates with the angulation and/or subluxation between the adjacent vertebrae. MRI and CT are the typical diagnostic modalities.²³ Conservative management with immobilization is frequently sufficient to treat compression fractures while burst or tear-drop fractures require surgical stabilization.²²

Rotational Injury

Atlantoaxial rotator fixation can result from a fall or collision resulting in occipitoatlanto dislocation in relation to the axis.² This leads to functional fixation and occurs after a fall or collision. There are four types that describe a spectrum of severity.²⁴ Type I injury causes rotational fixation without transverse ligament disruption resulting in no anterior displacement. Type II injury results in minimal anterior displacement because of a disrupted transverse ligament. Type III rotational fixation occurs with disruption of the alar and transverse ligament causing rotational displacement of greater than 0.5 cm. In type IV injury, the odontoid process is deficient allowing for posterior dislocation. CT and MRIs are the preferred diagnostic studies. Type I improves with a soft collar with or without traction while types II to IV require surgical stabilization.²⁵

Flexion/Extension

Cervical cord neurapraxia results from lateral flexion of the neck away from the shoulder that was hit. This injury is more common in contact sports and represents a mild SCI without radiographic abnormality. Temporary sensory and motor symptoms involving one or all extremities can occur²⁶ lasting as little as 15 minutes, or as long as 48 hour. Immobilization for 2 week is usually sufficient treatment.²⁷

Imaging

Plain radiographs are usually adequate for screening an alert patient with a normal neurological examination.^{28,29} The lateral view is the most revealing, detecting around 80% of injuries.³⁰ Some authors question the utility of the open mouth view due to the pediatric variants of pseudosubluxation and physiological wedging. Flexion-extension radiographs evaluate ligament stability, but are not practical in the acute setting.³¹ CT scans are the modality of choice for severely injured children. This helps identify bony derangements, but can miss 4% of clinically relevant ligamentous injuries.³¹⁻³³ MRI continues to be the most sensitive modality for SCI including bony, ligamentous, and cord injuries.³²⁻³⁴

Spinal Cord Injury without Radiographic Abnormality

The incidence of SCIWORA ranges from 5 to 67%.³⁵ The etiology varies based on age. The anatomy of the developing

spine with increased hypermobility of the younger spine places pediatric patients at greater risk for developing SCI-WORA.² Nonaccidental trauma can lead to devastating SCI-WORA in infants.² Children develop SCIWORA from falls and pedestrian versus motor vehicle accidents. Adolescents develop SCIWORA from sports injuries and motor-vehicle collisions. The presence and severity of hemorrhage predicts the severity of impairment³⁵; a major hemorrhage on MRI is associated with Frankel grades B and C (► **Table 1**): patients will likely remain with the presenting impairment level long term. A minor hemorrhage is also associated with Frankel B and C grade deficits, but almost half of these patients improve to grade D at 6 months. Patients presenting with cord edema only will have a mixture of grades B and C deficits (44%), and grade D deficits (56%). By 6 months, nearly three quarters of the patients with edema only on presentation will improve to grade D deficits and as many as a quarter will completely recover.³⁵

SCIWORA is associated with occult instability.³⁵ In patients that have SCIWORA, minor trauma can lead to recurrent symptoms. Dynamic radiographic follow-up is recommended for all patients with SCIWORA. If MRI identifies extraneural findings only at presentation, immobilization for 2 week is recommended. If neural findings on MRI are identified at presentation, 12 week of immobilization is recommended.²

Prehospital Management

The primary goals of prehospital management are to stabilize the patient and obtain neutral positioning without further injuring the spine. An adequate airway and hemodynamic support must be assured. SCI can present with apnea, cardiac arrest, or severe hemodynamic instability that may or may not be responsive to inotropic support. Since it is difficult to assess the severity of TBI at the scene, it is critical to prevent hypotension by providing aggressive hemodynamic support. For similar reasoning, adequate ventilation must be assured.³⁷

Respiratory complications are of significant concern. Vital capacity can be reduced by 30% in patients with cervical spine injury.³⁸ Significant ventilation/perfusion mismatch has been noted³⁹ and respiratory insufficiency will occur in up to 70% of patients with complete cervical SCI.⁴⁰ Chin lift maneuvers to open the airway can lead to opening the disk space by 5 mm in a patient with C5–C6 instability.⁴¹ Higher SCI places

the patients at higher risk for further injury.⁴² Manual in-line immobilization is needed to assure adequate visualization, but can still lead to significant movement of the cervical spine.⁴³ A camera-assisted laryngoscope can be helpful, but does not assure cervical stability and may lead to longer time to placement of an endotracheal tube.⁴² Consequently, the safest method for placing an endotracheal tube is in the hands of a skilled provider, attempting to minimize movement.⁴³

Obtaining neutral position is difficult in younger patients. Spine boards are created for adults and do not account for pediatric anatomic differences. Children less than 8 years have a relatively large head: the occiput extends beyond the shoulder when the neck is neutral in the supine. A routine spine board will place the neck in flexion for young patients,⁴⁴ even with a well-fitting collar. Nypaver and Treloar⁴⁵ found that all 40 children less than 8 years evaluated for neutral position on a spine board required elevation of the torso by 2.5 cm to achieve neutral position. Herzenberg et al⁴⁶ recommends using a board with an occipital recess or elevate the torso to align the auditory meatus with his/her shoulders to achieve neutral positioning. Curran found the majority of patients had at least 5 degrees of kyphosis based on the Cobb angle measurement.⁴⁷ In their study, only 12% of patients were presented in the neutral position, despite varying techniques to try to achieve neutrality.⁴⁷ Huerta et al⁴⁸ describes the need to secure younger patients with a rigid collar, modified half-spine board, and tape to prevent the patients from moving. However, restricting the torso leads to a mean forced vital capacity decrease by 80%.⁴⁹ This can impair ventilation, a detriment to the early treatment of concurrent TBI.

Pediatric trauma patients require aggressive prehospital resuscitation. However, this need is complicated by difficulties obtaining neutral position. There is currently not an adequate device available to allow for adequate stabilization in a neutral position without potentially restricting the patient's ventilation.

Hospital Management

Early Management

Initial management should continue to focus on stabilization. The patient should remain on spine immobilization until imaging can be accomplished and definitive management delineated. All patients should be taken off all spinal boards

Table 1 Frankel grading of neurologic deficits associated with spinal cord injury

Grade	Neurologic deficits
A (complete)	Complete motor and sensor loss below the level of injury
B (sensory only)	Complete motor loss below the lesion. Sacral sparing can be present
C (motor useless)	No functional motor power below the lesion although some motor power may be apparent
D (motor useful)	Useful motor power below the lesion is present
E (recovery)	Free of neurologic abnormality

Source: Adapted from Frankel et al.³⁶

immediately upon finishing initial evaluation in the emergency department (ED). Remaining on spine boards for as little as 2 hours can lead to decubitus ulcers. Acute SCI patients are at risk for neurogenic, hemorrhagic, and mixed shock. Initial stabilization should occur in the ED and continue in the intensive care unit (ICU).

Neurogenic shock (NS) is distributive in nature, resulting in hypotension without a compensatory rise in heart rate. Patients with NS may respond to intravenous fluid loading, but will likely also need vasoactive and/or inotropic support. The risk of NS increases with higher SCI.⁵⁰ The incidence of NS depends on the severity of injury: complete cervical SCI results in a 90% likelihood of requiring some type of hemodynamic support⁵¹ and up to 82% of SCI patients experienced volume-resistant shock within 7 days.⁵² The data to support definitive pharmacologic management of NS is limited. However, the distributive nature of NS lends itself to treatment with α agonists while bradycardia lends itself to treatment with β agonists. Utilizing agents with activity on both receptors can prevent reflexive bradycardia.⁵³ Consequently, norepinephrine, dopamine, and epinephrine are reasonable infusions.

There may be coincident hemorrhage depending on the mechanism of injury, posing significant complications for the treatment of shock. Aggressive fluid resuscitation including crystalloid and colloid is required with concurrent hemorrhagic shock while awaiting emergent surgical intervention for primary control of the source. Avoidance of hypotension is necessary to ensure adequate end organ perfusion, particularly in the brain.

Although the term "spinal shock" is often used in the clinical setting interchangeably with NS,⁵⁴ it specifies a marked reduction or loss of neurologic reflexes and motor function below the injury level.⁵⁵ The mean duration of spinal shock ranges from 4 to 6 weeks after injury.⁵⁴

As in TBI, secondary injury can occur during the early resuscitation period. Cellular mechanisms lead to intracellular increase in sodium and calcium, glutamate toxicity, free radical damage, and lipid peroxidation with activation of membrane lipases.⁵⁶ Arachidonic acid accumulates and activates the inflammatory cascade; secondary cord edema and ischemia then leads to autodestruction of the spinal cord at the level of injury.⁵⁷ Steroids have been investigated as a treatment to suppress early secondary injury. According to a subgroup analyses in a 2002 Cochrane review (updated in 2012),^{58,59} adult patients treated with methylprednisolone (MP) within 8 hours of injury showed improved motor recovery. However, many of the studies included in the analysis are small with less than 60 patients, and have varied patient populations with variable outcomes. The benefit of steroids is even less certain in the pediatric population. A small pediatric study published in 2011 showed complete recovery in 13 out of 15 children between 8 and 16 years within 24 hours after administration of methylprednisolone.⁶⁰ However, no injury was more severe than spinal cord contusion and no patient needed surgery for stabilization. Steroids remain controversial without clear benefit and are not currently recommended in the treatment of pediatric SCI.⁶¹

Intensive Care Management

ICU management does not differ from prehospital and early in-hospital management in the focus of care. However, additional considerations include adrenal insufficiency, autonomic dysfunction, ongoing shock, temperature dysregulation, shock bowel, and neurogenic bowel and/or bladder. Early coordination of care with surgical and rehabilitation services has the potential to contribute to an improved outcome.

As discussed above, a large percentage of SCI patients experience some degree of shock. Management of shock is crucial in the initial stages of the ICU stay. Recognizing and differentiating NS and hemorrhagic shock is necessary. Beginning neuroprotective measures until TBI is excluded can prevent secondary injury. Patients with SCI and subsequent NS are at an increased risk for relative adrenal insufficiency. In a study of nearly 200 patients with SCI, Pastrana et al⁶² identified adrenal insufficiency in 22% of the patients that had NS. These patients may benefit from supplementation with hydrocortisone. It is imperative that these patients are closely monitored and their shock aggressively treated.

Respiratory compromise may be significant in this population as well. As the spinal shock improves, the respiratory drive may improve. The strategy for mechanical ventilation will depend on the nature of the injury. Trauma places the patient at greater risk of primary (pulmonary contusion, rib fractures, pneumothorax) and secondary lung disease (acute respiratory distress syndrome, fluid shifts) and the need for more aggressive pulmonary management. As the lungs recover from the initial insult, it is imperative to consider tracheostomy early in mechanically ventilated patients. Nearly half of patients with significant cervical cord injury may require prolonged mechanical ventilator support.⁶³ Tracheostomy allows early mobilization and rehabilitation within the ICU.

Bradycardia may persist due to loss of sympathetic input on heart rate and vascular tone. Supraspinal sympathetic reflexes may be impaired.^{64,65} Standard procedures that result in increased vagal tone such as tracheal suctioning and intubation may prompt significant bradycardia that necessitates treatment with atropine.^{54,66} SCI patients are at higher risk for nonspecific ST changes as well and other dysrhythmias.^{67,68} Orthostatic hypotension may persist after initial hemodynamic instability resolves, contributing to sensations of light-headedness, dizziness, nausea, visual, and auditory disturbances. These patients are known to have reduced catecholamine levels.^{69,70} Spinal shock recovery can improve orthostatic hypotension. Otherwise, adaptation of the renin-aldosterone system over time aids in overcoming orthostatic hypotension.⁷¹

SCI patients also suffer from autonomic dysfunction due to unopposed afferent stimulation distal to the injury level of the cord lesion leading to hypertension, headache, diaphoresis, and chills above the level of the cord injury.^{71,72} Some patients also suffer from congestion, anxiety, and nausea. Autonomic dysfunction can occur in both the acute and chronic phase of SCI.⁵⁴ It is more profound in patients with cervical SCI. The response is triggered by noxious and non-noxious stimuli below the level of the SCI or it can occur

spontaneously. An increase in systemic norepinephrine is thought to cause the response.^{71,73,74} Norepinephrine leads to vasoconstriction distal to the SCI with a reflexive dilation of the vascular beds (where central control exists) and bradycardia.^{73,75,76} Autonomic dysfunction is thought to occur multiple times a day and can be asymptomatic. Factors include increased bladder pressure, catheterization, fractures, and any other procedure or pathologic condition that would normally provoke a pain response.^{71,77}

Thermoregulation occurs more commonly with higher SCI and can be present from the acute phase onward.⁵⁴ It may manifest in one of three ways: poikilothermia, "quad fever," and exercise-induced fever. Poikilothermia refers to body temperature that varies according to environmental temperature and includes either hyperthermia or hypothermia.⁵⁴ "Quad fever" denotes a common fever occurring for several weeks to months after injury⁷⁸ and is a diagnosis of exclusion. Exercise-induced fever is a newer diagnosis under consideration in the medical literature and is not presently well understood. It occurs more commonly with exercise in tetraplegic patients than in paraplegic patients with a longer-than-normal return to normothermia.⁷⁹

Shock bowel syndrome can result from SCI with NS. This often results from a prolonged period of bowel hypoperfusion, leading to dilated, thickened bowel loops, constriction of large caliber vessels, and significant contrast enhancement.⁸⁰ The renal parenchyma may show marked enhancement as well. Treatment demands aggressive volume resuscitation to restore perfusion to injured bowel.

Bowel and bladder motility problems are common to patients with SCI. Proper hygiene demands a regular regimen for both bowel and bladder evacuation to provide optimal comfort and minimize infection risk. Neurogenic bowel regimens utilized depend on aggressive use of stool softeners and/or laxatives. Occasionally, timed toileting with medications may be sufficient.⁸¹ Otherwise, digital stimulation may be required with timed toileting.⁸¹ Neurogenic bladder regimens often require clean intermittent catheterization. Medications to help control incontinence such as oxybutynin can be beneficial.⁸¹

Patients with significant SCI are at risk of venous thromboembolism (VTE). Factors include immobility, the presence of central venous lines, venous stasis, and coagulopathy early on. The incidence of VTE in pediatric trauma patients is thought to be between 0.2 and 0.33%.⁸² Adolescents with SCI have a higher risk for VTE than children or infants. Among patients with trauma, SCIs are an independent risk factor for VTE.⁸² There is no significant evidence to support a protocolized delivery of VTE prophylaxis. However, the clinician should provide either mechanical or pharmacologic prophylaxis depending on which is appropriate for each patient.⁸² Early surgical stabilization and mobility can reduce the risk of VTE.

Surgical approaches continue to improve and offer early stabilization opportunities. Bone grafting, transarticular screw fixation, occipitocervical fusion, and halos are among methods available to the neurologic and orthopedic surgeon.² The surgical approaches are beyond the scope of this article,

realizing that optimal timing for surgery depends on the circumstances of each patient, and multidisciplinary involvement.

Relative immobility leads to general loss of bone mineralization and osteopenia. Biering-Sørensen et al⁸³ describes a 25 to 50% decrease in lower extremity long bone density in SCI. Higher SCIs are associated with more long bone demineralization. More than 1 in 10 patients with SCI develops spontaneous, pathologic fractures secondary to osteopenia.⁸⁴ Vitamin D deficiency is common affecting up to 54% of adults with SCI.⁸⁵ Consequently, it is important to evaluate this cohort for vitamin D deficiency.

Long-Term Care

Early evaluation by a physiatrist can be invaluable. The rehabilitation service can assist in managing neurogenic bowel, bladder, VTE prophylaxis, autonomic dysfunction, and vitamin supplementation. As the child survives the acute phase of SCI, chronic debility can present particularly difficult challenges to the child and their family. Pediatric SCI length of stay is among the longest in rehabilitation centers.⁸⁶ The child with SCI has a 31% increase in the annual odds of dying.⁸⁷ The challenges in chronic SCI management include regaining function for activities of daily living, maintaining muscle mass, and reestablishing family and community involvement.

The primary aim of rehabilitation is to gain independence with either restorative or compensatory interventions. An in-depth discussion of the available options is beyond the scope of this review. The reader is directed to the review by Bryden et al⁸⁸ for further details. SCI patients are at significant risk for the metabolic syndrome and associated disorders including insulin resistance.⁸⁹ Determining appropriate calorie requirements based on energy expenditure is crucial to maintaining healthy lifestyle. Conventional exercise may be difficult or impossible depending on the patients' limitations. However, advances in electrical stimulation, prostheses, and computer-based technology have greatly enhanced the patient's ability to return to independence and maintain an active lifestyle.⁸⁸

The patient's ability to be involved in the local community improves quality of life and emotional health. Kelly et al⁹⁰ describes an improved quality of life among children who participate in activities away from home. She also noted adolescents had an improved emotional quality of life with participation with a diverse group peers. Several studies have detailed the difficulties encountered by children with SCI, as they grow older: there is a higher incidence of tobacco use among unemployed young adults that sustained a SCI as a child⁹¹; lack of marriage, chronic pain, incontinence, and lack of community support are associated with depressive symptoms in the adult with SCI.⁹² By contrast, those that were married, had a college degree, and no depression were less likely to turn to substance abuse. Spiritual coping mechanisms are also important in achieving better quality of life.⁹³

SCI can have an important effect on the family of patients. In a study of siblings of patients with SCI, Akhtar et al⁹⁴ described feelings of neglect by parents as well as guilt for not being able to care for the sibling patient. However, in

family units that remained intact, and in which a positive outlook was maintained, these feelings were mitigated. Garma et al⁹⁵ suggests that an intact family unit is associated with a higher subjective rating of quality of life in the patient beyond what the parents themselves perceive.

In conclusion, pediatric SCIs remain uncommon. Prehospital management is focused on maintaining an adequate airway and circulation being careful not to exacerbate a potential spine and head injury. The transported child could benefit from specially designed immobilization boards that allow positional neutrality for the neck. Early care in the ED and ICU is focused on treating shock, respiratory insufficiency and stabilizing an unstable neck or spine all the while preventing secondary injury. Surgical approaches and rehabilitation services continue to offer more hope to the patient and family. Children with SCI are surviving more. Their length of stay is among the longest in pediatric centers. As we improve our ability to help children survive devastating injuries, we need to continue to provide robust help for patients to help them adjust and cope with long-term challenges as well as for families who bear great burdens to care for their children. Accomplishing these tasks will help these children make the transition from a potentially life ending injury to a life filled with new possibilities and hope for enduring relationships in the family and local community.

References

- Parent S, Dimar J, Dekutoski M, Roy-Beaudry M. Unique features of pediatric spinal cord injury. *Spine* 2010;35(21, Suppl):S202–S208
- Mortazavi M, Gore PA, Chang S, Tubbs RS, Theodore N. Pediatric cervical spine injuries: a comprehensive review. *Childs Nerv Syst* 2011;27(5):705–717
- Chien LC, Wu JC, Chen YC, et al. Age, sex, and socio-economic status affect the incidence of pediatric spinal cord injury: an eleven-year national cohort study. *PLoS ONE* 2012;7(6):e39264
- Bulas DI, Fitz CR, Johnson DL. Traumatic atlanto-occipital dislocation in children. *Radiology* 1993;188(1):155–158
- Hagen EM, Rekand T, Gilhus NE, Grønning M. Traumatic spinal cord injuries—incidence, mechanisms and course. *Tidsskr Nor Lægeforen* 2012;132(7):831–837
- The University of Alabama at Birmingham. NSCISC National Spinal Cord Injury Statistical Center 2014. Available at: https://www.nscisc.uab.edu/PublicDocuments/fact_figures_docs/Facts%202014.pdf. Accessed September 9, 2014
- Keith LM, Persaud TVN, Mark GT. *Before We Are Born: Essentials of Embryology and Birth Defects*. 8th ed. Philadelphia, PA: Elsevier/Saunders; 2013:227–230
- Wang JC, Nuccion SL, Feighan JE, Cohen B, Dorey FJ, Scoles PV. Growth and development of the pediatric cervical spine documented radiographically. *J Bone Joint Surg Am* 2001;83-A(8):1212–1218
- Fesmire FM, Luten RC. The pediatric cervical spine: developmental anatomy and clinical aspects. *J Emerg Med* 1989;7(2):133–142
- Ogden JA. Radiology of postnatal skeletal development. XII. The second cervical vertebra. *Skeletal Radiol* 1984;12(3):169–177
- Liang CL, Lui CC, Lu K, Lee TC, Chen HJ. Atlantoaxial stability in ossiculum terminale. Case report. *J Neurosurg* 2001;95;(1, Suppl):119–121
- Brown RL, Brunn MA, Garcia VF. Cervical spine injuries in children: a review of 103 patients treated consecutively at a level 1 pediatric trauma center. *J Pediatr Surg* 2001;36(8):1107–1114
- Cirak B, Ziegfeld S, Knight VM, Chang D, Avellino AM, Paidas CN. Spinal injuries in children. *J Pediatr Surg* 2004;39(4):607–612
- Ware ML, Gupta N, Sun PP, Brockmeyer DL. Clinical biomechanics of the pediatric craniocervical junction and the subaxial spine. In: Brockmeyer DL, ed. *Advanced Pediatric Craniocervical Surgery*. New York, NY: Thieme; 2006:27–42
- Marshall KW, Koch BL, Egelhoff JC. Air bag-related deaths and serious injuries in children: injury patterns and imaging findings. *AJNR Am J Neuroradiol* 1998;19(9):1599–1607
- Dickman CA, Papadopoulos SM, Sonntag VK, Spetzler RF, Rekeate HL, Drabier J. Traumatic occipitoatlantal dislocations. *J Spinal Disord* 1993;6(4):300–313
- Sun PP, Poffenbarger GJ, Durham S, Zimmerman RA. Spectrum of occipitoatlantoaxial injury in young children. *J Neurosurg* 2000;93(1, Suppl):28–39
- Connolly B, Emery D, Armstrong D. The odontoid synchondrotic slip: an injury unique to young children. *Pediatr Radiol* 1995;25(Suppl 1):S129–S133
- Mandabach M, Ruge JR, Hahn YS, McLone DG. Pediatric axis fractures: early halo immobilization, management and outcome. *Pediatr Neurosurg* 1993;19(5):225–232
- Odent T, Langlais J, Glorion C, Kassis B, Bataille J, Pouliquen JC. Fractures of the odontoid process: a report of 15 cases in children younger than 6 years. *J Pediatr Orthop* 1999;19(1):51–54
- Sherk HH, Nicholson JT, Chung SM. Fractures of the odontoid process in young children. *J Bone Joint Surg Am* 1978;60(7):921–924
- Leonard JC. Cervical spine injury. *Pediatr Clin North Am* 2013;60(5):1123–1137
- Horn EM, Lekovic GP, Feiz-Erfan I, Sonntag VK, Theodore N. Cervical magnetic resonance imaging abnormalities not predictive of cervical spine instability in traumatically injured patients. Invited submission from the Joint Section Meeting on Disorders of the Spine and Peripheral Nerves, March 2004. *J Neurosurg Spine* 2004;1(1):39–42
- Fielding JW, Hawkins RJ. Atlanto-axial rotatory fixation. (Fixed rotatory subluxation of the atlanto-axial joint). *J Bone Joint Surg Am* 1977;59(1):37–44
- Pang D, Li V. Atlantoaxial rotatory fixation: part 3—a prospective study of the clinical manifestation, diagnosis, management, and outcome of children with atlantoaxial rotatory fixation. *Neurosurgery* 2005;57(5):954–972, discussion 954–972
- Torg JS, Corcoran TA, Thibault LE, et al. Cervical cord neurapraxia: classification, pathomechanics, morbidity, and management guidelines. *J Neurosurg* 1997;87(6):843–850
- Torg JS, Pavlov H, Genuario SE, et al. Neurapraxia of the cervical spinal cord with transient quadriplegia. *J Bone Joint Surg Am* 1986;68(9):1354–1370
- Nigrovic LE, Rogers AJ, Adelgais KM, et al; Pediatric Emergency Care Applied Research Network (PECARN) Cervical Spine Study Group. Utility of plain radiographs in detecting traumatic injuries of the cervical spine in children. *Pediatr Emerg Care* 2012;28(5):426–432
- Mower WR, Hoffman JR, Pollack CV Jr, Zucker MI, Browne BJ, Wolfson AB; NEXUS Group. Use of plain radiography to screen for cervical spine injuries. *Ann Emerg Med* 2001;38(1):1–7
- Baker C, Kadish H, Schunk JE. Evaluation of pediatric cervical spine injuries. *Am J Emerg Med* 1999;17(3):230–234
- Pollack CV Jr, Hendey GW, Martin DR, Hoffman JR, Mower WR; NEXUS Group. Use of flexion-extension radiographs of the cervical spine in blunt trauma. *Ann Emerg Med* 2001;38(1):8–11
- Schenarts PJ, Diaz J, Kaiser C, Carrillo Y, Eddy V, Morris JA Jr. Prospective comparison of admission computed tomographic scan and plain films of the upper cervical spine in trauma patients with altered mental status. *J Trauma* 2001;51(4):663–668, discussion 668–669

- 33 Flynn JM, Closkey RF, Mahboubi S, Dormans JP. Role of magnetic resonance imaging in the assessment of pediatric cervical spine injuries. *J Pediatr Orthop* 2002;22(5):573–577
- 34 Kaiser ML, Whealon MD, Barrios C, Kong AP, Lekawa ME, Dolich MO. The current role of magnetic resonance imaging for diagnosing cervical spine injury in blunt trauma patients with negative computed tomography scan. *Am Surg* 2012;78(10):1156–1160
- 35 Pang D. Spinal cord injury without radiographic abnormality in children, 2 decades later. *Neurosurgery* 2004;55(6):1325–1342, discussion 1342–1343
- 36 Frankel HL, Hancock DO, Hyslop G, et al. The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. I. Paraplegia 1969;7(3):179–192
- 37 Kochanek PM, Carney N, Adelson PD, et al; American Academy of Pediatrics–Section on Neurological Surgery; American Association of Neurological Surgeons/Congress of Neurological Surgeons; Child Neurology Society; European Society of Pediatric and Neonatal Intensive Care; Neurocritical Care Society; Pediatric Neurocritical Care Research Group; Society of Critical Care Medicine; Paediatric Intensive Care Society UK; Society for Neuroscience in Anesthesiology and Critical Care; World Federation of Pediatric Intensive and Critical Care Societies. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents—second edition. *Pediatr Crit Care Med* 2012;13(Suppl 1):S1–S82
- 38 Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis* 1981;124(1):41–44
- 39 Casha S, Christie S. A systematic review of intensive cardiopulmonary management after spinal cord injury. *J Neurotrauma* 2011;28(8):1479–1495
- 40 Hachen HJ. Idealized care of the acutely injured spinal cord in Switzerland. *J Trauma* 1977;17(12):931–936
- 41 Aprahamian C, Thompson BM, Finger WA, Darin JC. Experimental cervical spine injury model: evaluation of airway management and splinting techniques. *Ann Emerg Med* 1984;13(8):584–587
- 42 Hastings RH, Marks JD. Airway management for trauma patients with potential cervical spine injuries. *Anesth Analg* 1991;73(4):471–482
- 43 Lennarson PJ, Smith D, Todd MM, et al. Segmental cervical spine motion during orotracheal intubation of the intact and injured spine with and without external stabilization. *J Neurosurg* 2000;92(2, Suppl):201–206
- 44 Rozzelle CJ, Aarabi B, Dhall SS, et al. Management of pediatric cervical spine and spinal cord injuries. *Neurosurgery* 2013;72(Suppl 2):205–226
- 45 Nypaver M, Treloar D. Neutral cervical spine positioning in children. *Ann Emerg Med* 1994;23(2):208–211
- 46 Herzenberg JE, Hensinger RN, Dedrick DK, Phillips WA. Emergency transport and positioning of young children who have an injury of the cervical spine. The standard backboard may be hazardous. *J Bone Joint Surg Am* 1989;71(1):15–22
- 47 Curran C, Dietrich AM, Bowman MJ, Ginn-Pease ME, King DR, Kosnik E. Pediatric cervical-spine immobilization: achieving neutral position? *J Trauma* 1995;39(4):729–732
- 48 Huerta C, Griffith R, Joyce SM. Cervical spine stabilization in pediatric patients: evaluation of current techniques. *Ann Emerg Med* 1987;16(10):1121–1126
- 49 Schafermeyer RW, Ribbeck BM, Gaskins J, Thomason S, Harlan M, Attkisson A. Respiratory effects of spinal immobilization in children. *Ann Emerg Med* 1991;20(9):1017–1019
- 50 Mallek JT, Inaba K, Branco BC, et al. The incidence of neurogenic shock after spinal cord injury in patients admitted to a high-volume level I trauma center. *Am Surg* 2012;78(5):623–626
- 51 Vale FL, Burns J, Jackson AB, Hadley MN. Combined medical and surgical treatment after acute spinal cord injury: results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg* 1997;87(2):239–246
- 52 Levi L, Wolf A, Belzberg H. Hemodynamic parameters in patients with acute cervical cord trauma: description, intervention, and prediction of outcome. *Neurosurgery* 1993;33(6):1007–1016, discussion 1016–1017
- 53 Stratman RC, Wiesner AM, Smith KM, Cook AM. Hemodynamic management after spinal cord injury. *Orthopedics* 2008;31(3):252–255
- 54 Krassioukov AV, Karlsson AK, Wecht JM, Wuermser LA, Mathias CJ, Marino RJ; Joint Committee of American Spinal Injury Association and International Spinal Cord Society. Assessment of autonomic dysfunction following spinal cord injury: rationale for additions to International Standards for Neurological Assessment. *J Rehabil Res Dev* 2007;44(1):103–112
- 55 Dittunno JF, Little JW, Tessler A, Burns AS. Spinal shock revisited: a four-phase model. *Spinal Cord* 2004;42(7):383–395
- 56 Agrawal SK, Fehlings MG. Mechanisms of secondary injury to spinal cord axons in vitro: role of Na⁺, Na⁺-K⁺-ATPase, the Na⁺-H⁺ exchanger, and the Na⁺-Ca²⁺ exchanger. *J Neurosci* 1996;16(2):545–552
- 57 Pettiford JN, Bikhchandani J, Ostlie DJ, St Peter SD, Sharp RJ, Juang D. A review: the role of high dose methylprednisolone in spinal cord trauma in children. *Pediatr Surg Int* 2012;28(3):287–294
- 58 Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database Syst Rev* 2002;(3):CD001046
- 59 Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database Syst Rev* 2012;1:CD001046
- 60 Arora B, Suresh S. Spinal cord injuries in older children: is there a role for high-dose methylprednisolone? *Pediatr Emerg Care* 2011;27(12):1192–1194
- 61 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):1515–1524
- 62 Pastrana EA, Saavedra FM, Murray G, Estronza S, Rolston JD, Rodriguez-Vega G. Acute adrenal insufficiency in cervical spinal cord injury. *World Neurosurg* 2012;77(3–4):561–563
- 63 Lemons VR, Wagner FC Jr. Respiratory complications after cervical spinal cord injury. *Spine* 1994;19(20):2315–2320
- 64 Mathias CJ. Orthostatic hypotension and paroxysmal hypertension in humans with high spinal cord injury. *Prog Brain Res* 2006;152:231–243
- 65 Claydon VE, Steeves JD, Krassioukov A. Orthostatic hypotension following spinal cord injury: understanding clinical pathophysiology. *Spinal Cord* 2006;44(6):341–351
- 66 Mathias CJ. Bradycardia and cardiac arrest during tracheal suction—mechanisms in tetraplegic patients. *Eur J Intensive Care Med* 1976;2(4):147–156
- 67 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
- 68 Prakash M, Raxwal V, Froelicher VF, et al. Electrocardiographic findings in patients with chronic spinal cord injury. *Am J Phys Med Rehabil* 2002;81(8):601–608
- 69 Mathias CJ, Christensen NJ, Frankel HL, Peart WS. Renin release during head-up tilt occurs independently of sympathetic nervous activity in tetraplegic man. *Clin Sci (Lond)* 1980;59(4):251–256
- 70 Claydon VE, Krassioukov AV. Orthostatic hypotension and autonomic pathways after spinal cord injury. *J Neurotrauma* 2006;23(12):1713–1725
- 71 Teasell RW, Arnold JM, Krassioukov A, Delaney GA. Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury. *Arch Phys Med Rehabil* 2000;81(4):506–516
- 72 Mathias CJ, Frankel HL. Autonomic disturbances in spinal cord lesions. In: Mathias C, Bannister R, eds. *Autonomic Failure: A Textbook of Clinical Disorders of the Autonomic Nervous System*. New York, NY: Oxford University Press; 2002:494–513

- 73 Karlsson AK, Friberg P, Lönnroth P, Sullivan L, Elam M. Regional sympathetic function in high spinal cord injury during mental stress and autonomic dysreflexia. *Brain* 1998;121(Pt 9):1711–1719
- 74 Karlsson AK, Elam M, Friberg P, Biering-Sørensen F, Sullivan L, Lönnroth P. Regulation of lipolysis by the sympathetic nervous system: a microdialysis study in normal and spinal cord-injured subjects. *Metabolism* 1997;46(4):388–394
- 75 Gao SA, Ambring A, Lambert G, Karlsson AK. Autonomic control of the heart and renal vascular bed during autonomic dysreflexia in high spinal cord injury. *Clin Auton Res* 2002;12(6):457–464
- 76 Corbett JL, DeBarge O, Frankel HL, Mathias C. Cardiovascular responses in tetraplegic man to muscle spasm, bladder percussion and head-up tilt. *Clin Exp Pharmacol Physiol* 1975 (Suppl 2):189–193
- 77 Karlsson AK. Autonomic dysreflexia. *Spinal Cord* 1999;37(6):383–391
- 78 Sugarman B, Brown D, Musher D. Fever and infection in spinal cord injury patients. *JAMA* 1982;248(1):66–70
- 79 Price MJ, Campbell IG. Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc* 2003;35(7):1100–1107
- 80 Kwon OY, Lee JS, Choi HS, Hong HP, Ko YG. Shock bowel caused by neurogenic shock: computed tomography findings. *J Emerg Med* 2012;43(4):e251–e253
- 81 Vogel LC, Hickey KJ, Klaas SJ, Anderson CJ. Unique issues in pediatric spinal cord injury. *Orthop Nurs* 2004;23(5):300–308, quiz 309–310
- 82 Thompson AJ, McSwain SD, Webb SA, Stroud MA, Streck CJ. Venous thromboembolism prophylaxis in the pediatric trauma population. *J Pediatr Surg* 2013;48(6):1413–1421
- 83 Biering-Sørensen F, Bohr H, Schaadt O. Bone mineral content of the lumbar spine and lower extremities years after spinal cord lesion. *Paraplegia* 1988;26(5):293–301
- 84 Betz RR. Unique management needs of pediatric spinal cord injury patients: orthopedic problems in the child with spinal cord injury. *J Spinal Cord Med* 1997;20(1):14–16
- 85 Oleson CV, Patel PH, Wuermsler LA. Influence of season, ethnicity, and chronicity on vitamin D deficiency in traumatic spinal cord injury. *J Spinal Cord Med* 2010;33(3):202–213
- 86 Kim CT, Greenberg J, Kim H. Pediatric rehabilitation: trends in length of stay. *J Pediatr Rehabil Med* 2013;6(1):11–17
- 87 Shavelle RM, Devivo MJ, Paculdo DR, Vogel LC, Strauss DJ. Long-term survival after childhood spinal cord injury. *J Spinal Cord Med* 2007;30(Suppl 1):S48–S54
- 88 Bryden AM, Ancans J, Mazurkiewicz J, McKnight A, Scholtens M. Technology for spinal cord injury rehabilitation and its application to youth. *J Pediatr Rehabil Med* 2012;5(4):287–299
- 89 Johnston TE, McDonald CM. Health and fitness in pediatric spinal cord injury: medical issues and the role of exercise. *J Pediatr Rehabil Med* 2013;6(1):35–44
- 90 Kelly EH, Klaas SJ, Garma S, Russell HF, Vogel LC. Participation and quality of life among youth with spinal cord injury. *J Pediatr Rehabil Med* 2012;5(4):315–325
- 91 Hwang M, Chlan KM, Vogel LC, Zebracki K. Substance use in young adults with pediatric-onset spinal cord injury. *Spinal Cord* 2012; 50(7):497–501
- 92 January AM, Zebracki K, Chlan KM, Vogel LC. Symptoms of depression over time in adults with pediatric-onset spinal cord injury. *Arch Phys Med Rehabil* 2014;95(3):447–454
- 93 Chlan KM, Zebracki K, Vogel LC. Spirituality and life satisfaction in adults with pediatric-onset spinal cord injury. *Spinal Cord* 2011; 49(3):371–375
- 94 Akhtar M, Kennedy P, Webster G, Graham A. What about us? Children's experiences of living with a sibling with spinal cord injury. *J Pediatr Rehabil Med* 2012;5(4):301–313
- 95 Garma SI, Kelly EH, Daharsh EZ, Vogel LC. Health-related quality of life after pediatric spinal cord injury. *J Pediatr Psychol* 2011;36(2): 226–236