Traumatic Injuries to the Spinal Cord and Peripheral Nervous System

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KEYWORDS

• Peripheral nerve injury • Spinal cord injury • Trauma • Neurogenic shock
• Secondary injury

KEY POINTS

- Injuries to the peripheral nervous system and spinal cord regularly occur during blunt and penetrating trauma.
- Other life-threatening injuries should be prioritized in the setting of polytrauma before pursing definitive management of injuries to peripheral nerves or the spinal cord.
- Sharply transected peripheral nerve injuries should prompt consultation for immediate repair.
- Providers should have a low threshold for intubation during the acute management of spinal cord injuries because lower cervical and even thoracic injuries can result in insufficient airway protection or breathing.
- First-line management of neurogenic shock should be intravenous fluids followed by, if necessary, norepinephrine to maintain a mean arterial pressure of at least 85 mm Hg.

INTRODUCTION

All trauma, whether blunt or penetrating, has the potential to cause injury to the nervous system. This includes the brain and spinal cord of the central nervous system and the somatic and autonomic components of the peripheral nervous system (PNS). Traumatic injuries to the PNS are a significant source of morbidity. Peripheral nerve injury (PNI) can result in permanent disability and entail significant health care and patient costs. Acute costs associated with these injuries average nearly $6000 in the emergency department (ED) and $20,000 to $60,000 in inpatient expenses.[1–3] These costs do not consider the burden of decreased quality of life and long-term health care costs.

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Spinal cord injuries (SCIs) similarly can cause significant permanent disability and even death. The financial cost to patients and society from SCIs is significant, accumulating average expenses in the United States between $375,000 and $1,150,000 in the first year alone, depending on severity of injury. Subsequent annual expenses average between $45,000 to $200,000.[4]

Emergency medicine providers play an essential role in the recognition and subsequent management of these patients. Presentations can be subtle and sometimes are missed, risking morbidity and even mortality for patients, and subjecting providers to high medicolegal liability. Prior malpractice suits involving missed cervical injuries in blunt trauma patients, for example, have resulted in multimillion-dollar awards.[5]

This article covers the pathophysiology, clinical assessment, and management of traumatic PNIs and SCIs. The primary population of this review is adult patients. Please see the article "Neurologic Emergencies at the Extremes of Age," by Khoujah and Cobb for further discussion of pediatric and geriatric populations.

PERIPHERAL NERVE INJURY

Epidemiology

Traumatic PNI represents a significant burden of disease. PNIs are thought to have an incidence of more than 350,000 per year in the United States.[6] Previous estimates using the National Inpatient Sample and National Emergency Department Sample show widely varying estimates of injuries based on diagnosis codes and likely underestimate the true burden of injury. The overall trend in injuries has been relatively stable, with upper extremity PNI more common than lower extremity PNI.[1,2] Among trauma patients evaluated at a level 1 trauma center, 2.8% were identified as having a PNI.[7] European trauma registry data showed PNIs associated with 3.3% of severe upper extremity trauma and 1.8% of severe lower extremity trauma.[8,9] PNIs may remain occult in severely injured patients given priorities of resuscitation and concomitant injuries that limit the examination. A previous case series of traumatic brain injury patients identified a 34% incidence of PNIs not recognized on initial evaluation.[10]

Males account for 80% of traumatic PNIs with a mean age of approximately 40 years in registry data.[8,9] Blunt mechanisms, including motor vehicle accidents and falls, account for the majority of injuries but disproportionately higher rates of PNIs are observed in penetrating trauma then compared to the overall trauma population.[9] In upper extremity trauma, the ulnar nerve is injured most commonly, followed by the radial and median nerves.[2] The peroneal nerve is the lower extremity nerve most commonly injured followed by the sciatic and tibial nerves.[1] Sports-related acute nerve injury represents less than 1% of all injuries but has been increasing.[11]

Key Anatomy and Pathophysiology

The terms, *peripheral nerve* and *PNS*, encompass nervous tissue and supporting structures between the central nervous system and target tissues or sensory areas. These include cranial nerves (except for the optic nerves) and the autonomic nervous system, in addition to motor and sensory branches originating in the spinal cord. Peripheral nerves have several specialized connective tissues: the endoneurium, the perineurium, and the epineurium (**Fig. 1**). These define the structure of the nerve and are critical for regeneration.[12]

Most PNIs can be attributed to a combination of mechanisms including traction or stretch, contusion, transection, and compression. Other mechanisms of traumatic injury include ischemia, burns, and electrical injuries. Nerves are vulnerable to different mechanisms along their length due to the changing composition of the nerves and

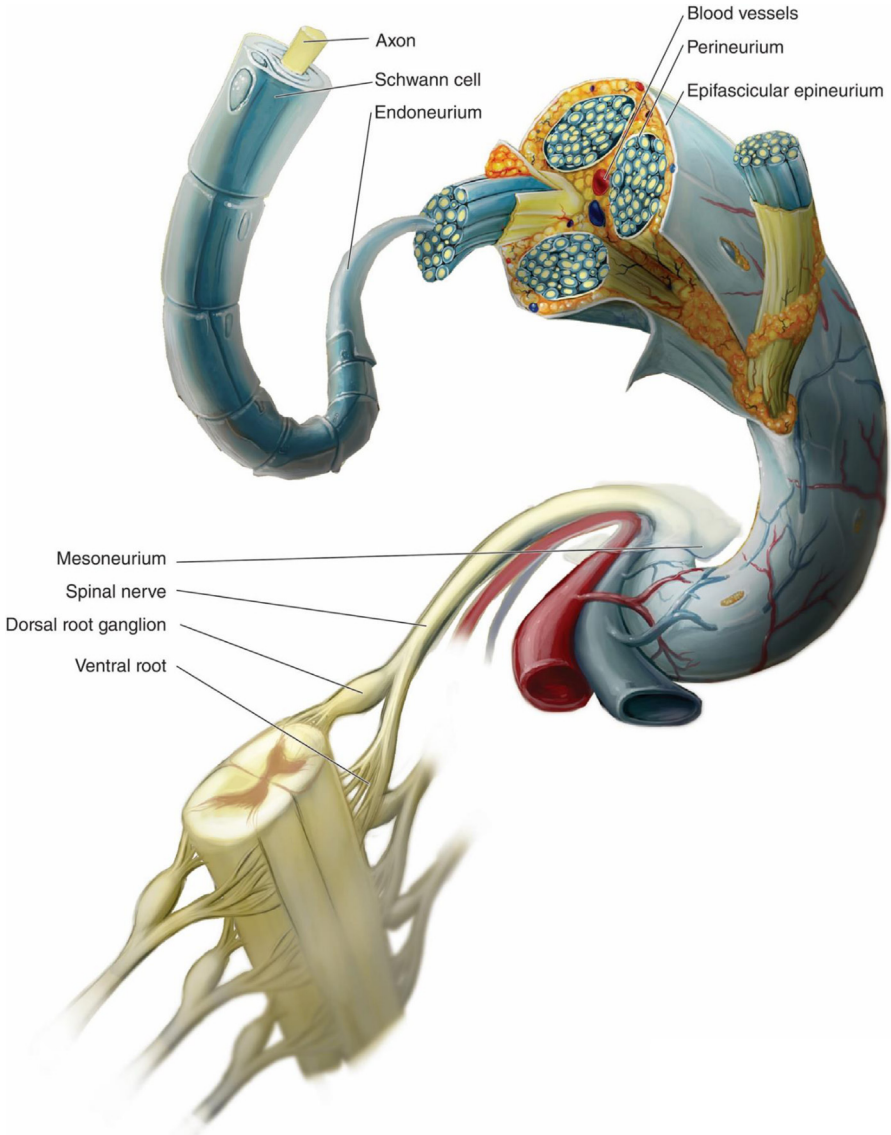

Fig. 1. Major anatomic components of a peripheral nerve. (*From* Smith BW, Sakamuri S, Spain DA, Joseph JR, Yang LJ, Wilson TJ. An update on the management of adult traumatic nerve injuries-replacing old paradigms: A review. J Trauma Acute Care Surg. 2019;86(2):299-306; with permission.)

regional anatomy. Nerve roots, for instance, lack both epineurium and perineurium and are relatively tethered to the spinal cord, making them vulnerable to traction and compression.[6] Proximity to bone makes nerves vulnerable to injury from fractures, whereas superficial nerves may be more easily contused or lacerated.

Nerve stretching can be part of normal function with changes in length as nerves cross over joints and at extremes of physiologic movement. Extremes of stretching overwhelm the ability of the connective tissue to compensate and result in injuries

with associated hematomas and scarring.[13] Avulsion is an extreme stretch or traction injury causing mechanical failure and disruption of the nerve, often occurring at nerve roots and is associated with significant morbidity.

Compression can cause ischemic injury from direct or indirect pressure (eg, associated compartment syndrome). A classic example is compression of the radial nerve against the humerus as it travels in the radial groove, producing a Saturday night palsy. Hydrostatic forces from penetrating injury also can cause nerve injury or disruption. Crush injuries can occur directly or via entrapment from dislocation-relocation or associated fractures. Laceration or transection mechanisms can be divided into sharp and blunt.

Classification of Peripheral Nerve Injuries

In 1942, Seddon[14] proposed a classification scheme that is still in primary use today for grading nerve injuries based on severity of disruption to the nerve and supporting structures. Seddon divided injuries as neurapraxia, axonotmesis, and neurotmesis (**Fig. 2**). Sunderland[15] later expanded this to 5 degrees of injury (**Table 1**).

Presentation and Examination of Peripheral Nerve Injuries

Traumatic PNI initially is a clinical diagnosis and, because PNIs by themselves typically are not life threatening, other more dangerous and time-sensitive causes and associated injuries must be considered. In the trauma patient, prompt global assessment and resuscitation should be undertaken prior to detailed investigation for nerve injury. All sensory or motor abnormalities should be evaluated for alternative causes, especially central causes, such as intracranial hemorrhage and SCIs.[16] An evolving deficit should prompt evaluation for a dynamic process like progressive edema, hematoma formation, pseudoaneurysm formation, or shifting of fractures.

Evidence of nerve injury should prompt consideration of associated fractures, hematomas, compartment syndrome, and arterial injuries. Because nerves typically travel along the neurovascular bundles, and blood vessels are vulnerable to the same forces, approximately 13% of upper extremity PNIs from civilian trauma have an associated vascular injury.[9,17] Injuries associated with warfare and penetrating injury have an even higher association between PNIs and vascular injuries, with arterial injuries present in 48 of 119 patients in a case series of PNIs from the Balkan conflict.[18] Traumatic injuries typically present with maximal deficits.

It is critical to determine open versus closed injuries because this significantly alters management.[19–23] Exploration of an open wound and assessment of the wound mechanism can help identify an associated clinical nerve injury. A clean, sharp transection versus a blunt, ragged transection can affect the urgency of repair.[20,21,23] Providers should use motor grading and sensory testing to determine the severity and likely anatomic location(s) of injury (**Fig. 3** for sensory distribution of major peripheral nerves). Two-point discrimination is the preferred mode of testing for sensory injury with a recent case series of hand injuries, demonstrating 98.6% sensitivity for detecting nerve injury with a 2-point discrimination tool compared to 82.5% for dry gauze.[16,24] Tinel sign also may be present acutely at the area of injury with advancing location and increased pain present in regenerating injuries and developing neuromas, respectively.[25]

Providers should be prepared to recognize several classic PNIs of the upper (**Table 2**) and lower (**Table 3**) extremities.

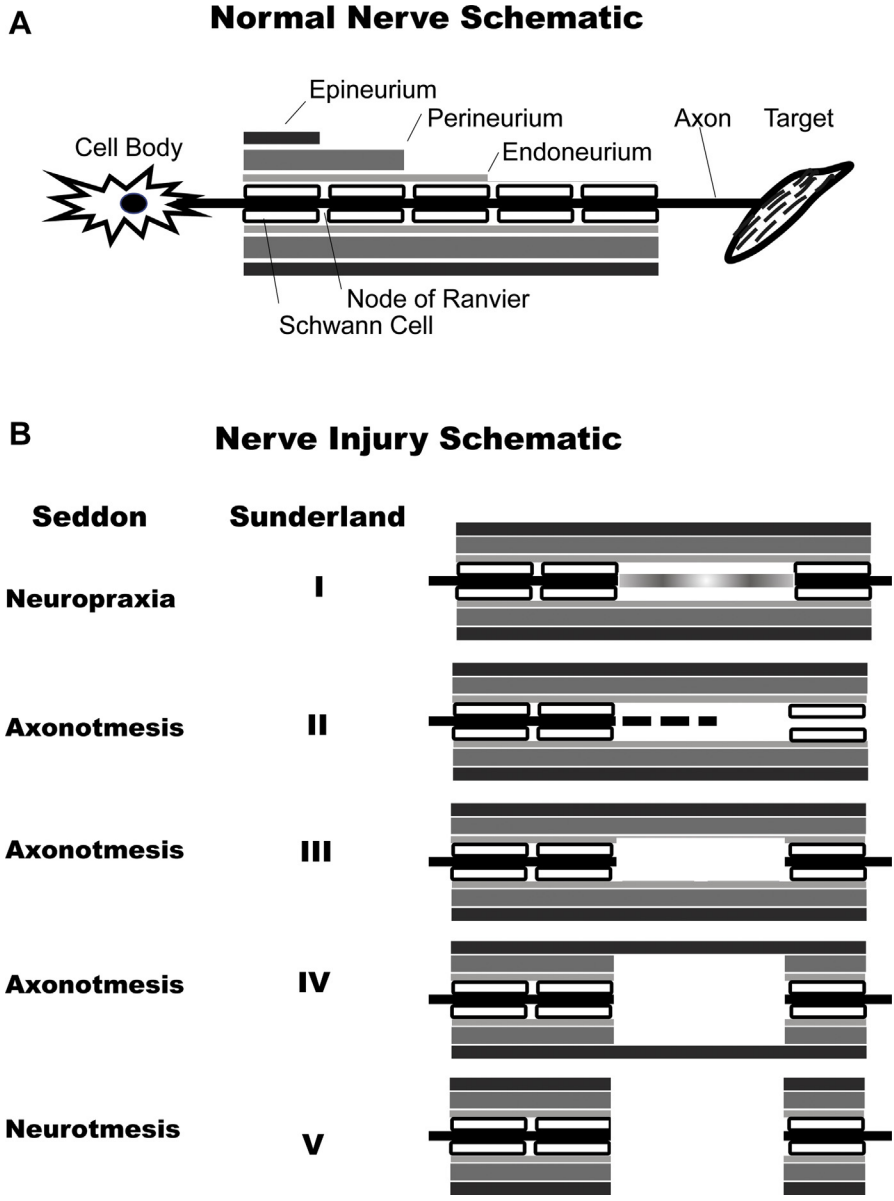

Fig. 2. (*A*) Intact peripheral nerve anatomy. (*B*) Anatomic schematic of PNI grades.

Diagnostic Evaluation of Peripheral Nerve Injuries

Clinical examination combined with potential surgical exploration, electromyography, and nerve conduction studies is important for overall assessment of PNIs. Additional diagnostics during acute presentations in the ED are largely supplements to the clinical examination and evaluate primarily for associated injuries and alternative causes.

Table 1
Seddon and Sunderland classification schemes of peripheral nerve injuries

Seddon	Sunderland	Clinical Correlate	Pathologic Correlate	Prognosis for Spontaneous Recovery	Surgical Intervention
Neurapraxia	1	Compression, ischemia	Demyelination	Good	Unnecessary
Axonotmesis	2	Ischemia, crush, percussion	Axon degeneration	Good to fair	Usually unnecessary
	3		Endoneural injury	Intermediate	May be required
	4		Perineural injury	Poor	May be required
Neurotmesis	5	Avulsion, transection	Epineural Injury	Poor	Required

From National Spinal Cord Injury Statistical Center. 2019 Annual Statistical Report for the Spinal Cord Injury. Model Systems. University of Alabama at Birmingham: Birmingham, Alabama; With permission.

X-ray and computed tomography evaluation

Peripheral nerves are not imaged by plain radiographs and are poorly imaged with computed tomography (CT). These images can evaluate for associated injuries. Individual nerve injuries may prompt specific radiographs to identify commonly associated fractures or dislocations (eg, hook of hamate fracture in distal ulnar nerve injury or evaluation for a Bankart or Hill-Sachs lesion suggestive of previous dislocation in axillary nerve dysfunction). Although CT is inadequate for direct evaluation of

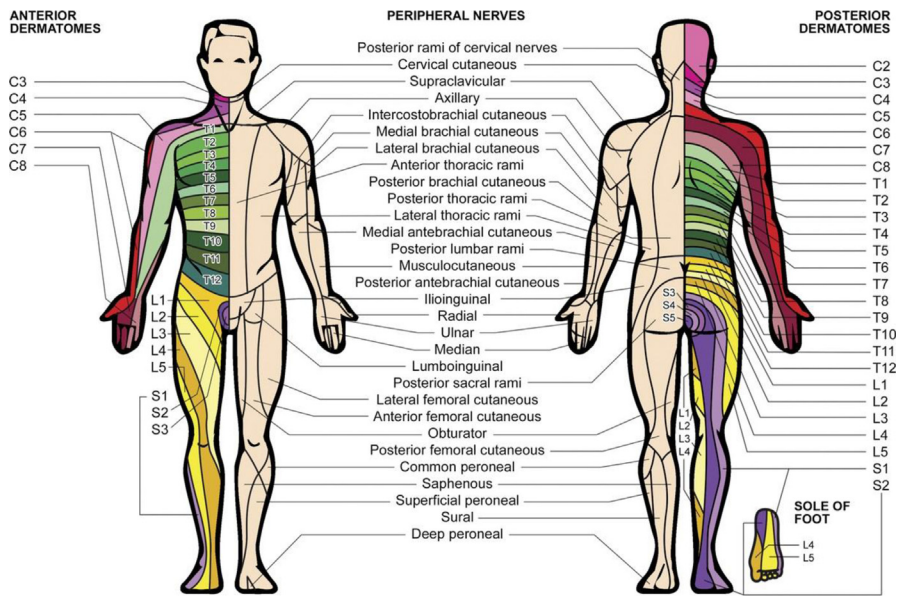

Fig. 3. Comparison of peripheral nerve fields and dermatomes. (*From* Smith BW, Sakamuri S, Spain DA, Joseph JR, Yang LJ, Wilson TJ. An update on the management of adult traumatic nerve injuries-replacing old paradigms: A review. *J Trauma Acute Care Surg*. 2019;86(2):299–306; with permission.)

Table 2
Classic peripheral nerve injuries of the upper extremities

Nerves	Example Mechanism(s)	Major Deficits	Pathologic Correlate
Brachial plexus	Stinger/burner Seatbelt injury	Variable; typically, C5-C6 or C8-T1, depending on direction of forces Autonomic deficits (eg, Horner syndrome) in C8-T1 lesions	Neuropraxia, nerve avulsion in severe trauma
Axillary	Shoulder dislocation Surgical neck fracture of humerus	Sensory: deltoid area Motor: shoulder flexion and abduction	Neuropraxia
Radial	Midshaft humerus fracture Saturday night palsy	Sensory: dorsal medial hand Motor: wrist extension, finger extension	Neuropraxia
Ulnar (proximal)	Elbow dislocation Medial epicondyle fracture cubital compression	Sensory: ulnar hand Motor: grip strength, fourth and fifth digits flexion (proximal)	Axonotmesis
Median	Supracondylar fracture of humerus Laceration (typically distal)	Sensory: palmar radial hand Motor: thumb opposition, second and third digits flexion	Variable
Long thoracic nerve	Penetrating chest, axilla, or supraclavicular trauma	Motor: scapular protraction (may significantly impair upper extremity function)	Neurotmesis

Table 3
Classic peripheral nerve injuries of the lower extremities

Nerves	Example Mechanism(s)	Major Deficits	Pathologic Correlate
Sciatic	Posterior hip dislocation Penetrating trauma	Sensory: posterior and lateral leg, dorsal and plantar foot Motor: knee flexion, ankle dorsiflexion and plantarflexion	Variable
Peroneal	Knee dislocation Fibular fracture Contusion	Sensory: dorsal foot Motor: ankle dorsiflexion and eversion	Variable
Inferior gluteal	Posterior hip dislocation	Sensory: none Motor: hip extension and extension of the flexed thigh	Variable
Tibial	Tibial fracture Knee dislocation	Sensory: plantar foot Motor: ankle plantarflexion	Variable

nerve injury, it has added utility for evaluating soft tissue lesions and vascular structures[25] CT myelography is sensitive and specific for brachial plexus injury and nerve root avulsion in later phases of injury.[26]

Magnetic resonance imaging

Magnetic resonance imaging (MRI), also called magnetic resonance neurography, is superior to CT for PNIs due to significantly improved contrast resolution, ability to assess nerve edema, and evolving use of sequences to assess nerve integrity.[25,27,28] The utility of MRI in the immediate or early evaluation of suspected injury is unclear, because there are no established guidelines and because of typically conservative overall management strategy of closed nerve injuries. Given the limitations of electrodiagnostic testing in the acute phase, there may be select cases of MRI that allow for earlier intervention.[29]

Ultrasonography

Ultrasonography, along with MRI, is the other preferred imaging technique for nerve injury. Ultrasound offers high spatial resolution, the ability to perform dynamic maneuvers, and comparatively low cost but with limited contrast resolution, limited ability to image deeper structures, and significant operator dependence.[28,30–32] Nerves are imaged best with a high-frequency linear array and have a characteristic echotexture due to bundles of nerve fibers or fascicles.

Superficial nerves are well visualized and can be traced along their course to evaluate for swelling, size, or echotexture changes (eg, loss of internal architecture) that may indicate neuropraxia, axonotmesis/neurotmesis, and disruption of nerve continuity.[30–32] These can be accentuated by dynamic maneuvers. Ultrasound is able to characterize much of the course of the most commonly injured nerves and can identify areas of nerve entrapment.[27,33,34]

The role of ultrasound in the ED is evolving but has been shown in some cases to be superior to MRI in evaluation of nerve lesions.[35] Ultrasound evaluation changes management in as many as 58% of cases, including decisions on immediate versus delayed surgery, identification of complete nerve disruption, detection of foreign bodies, and detection of multiple areas of injury.[36,37]

Electrodiagnostic testing

Electrodiagnostic testing is the interrogation of nerve function using electrical impulses and is widely used for evaluation of nerve function, including in traumatic injuries. This technique does not have a role in the acute setting because electromyography and nerve conduction studies cannot differentiate between neuropraxia, axonotmesis, and neurotmesis immediately after injury.[38,39] Neuropraxia and higher-grade injuries can be differentiated by 1 week postinjury. PNI features are variable between injury areas and type, and serial evaluations over time are used to help gauge recovery and plan interventions.

Management of Peripheral Nerve Injuries

Disposition and follow-up

Treatment can vary widely after initial evaluation of possible nerve injury because, depending on the type of injury, it may be supportive or surgical. Smith and colleagues outlined a proposed approach to management of nerve injury building on the approach of Grant and colleagues (**Fig. 4**).[20,23]

The rule of 3s can be helpful in considering the appropriate timing of follow-up and intervention in subspecialty care. Sharp nerve transections are best explored and repaired within 3 days. Open injuries that are ragged or contused may be best

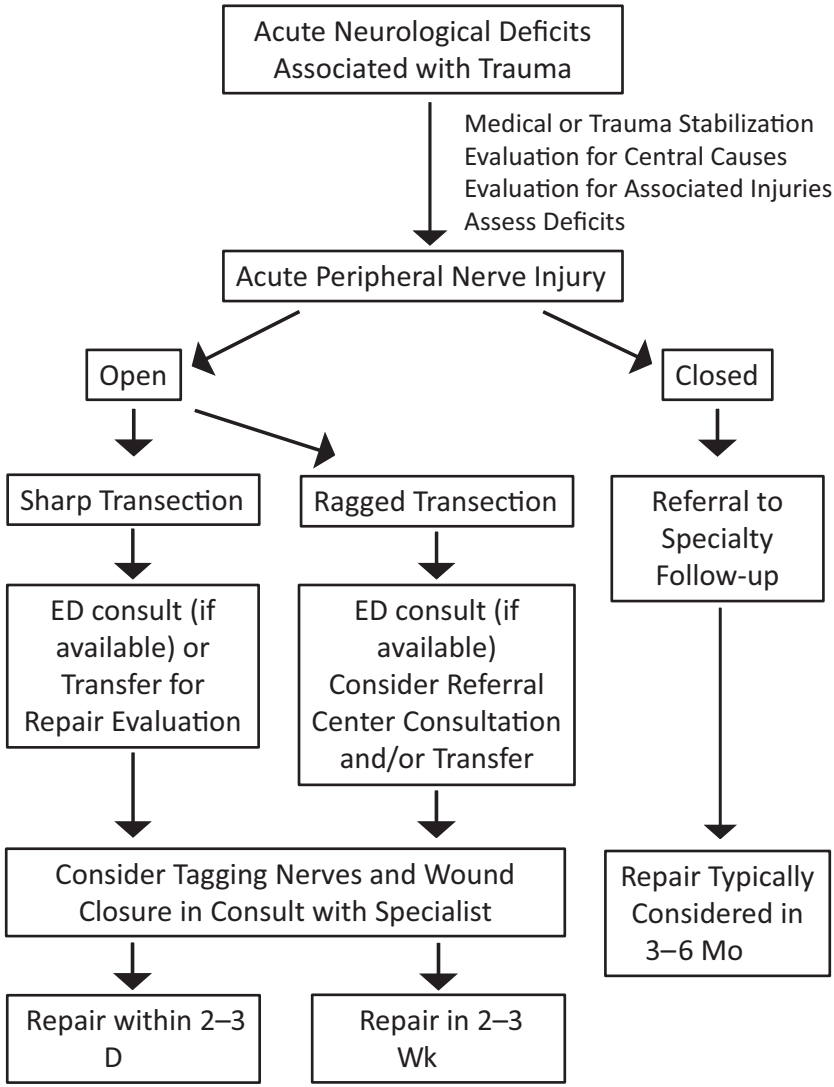

Fig. 4. Proposed treatment algorithm for PNIs. (*Adapted from* Smith BW, Sakamuri S, Spain DA, Joseph JR, Yang LJ, Wilson TJ. An update on the management of adult traumatic nerve injuries-replacing old paradigms: A review. *J Trauma Acute Care Surg*. 2019;86(2):299–306; with permission.)

explored for repair after 3 weeks to allow demarcation and healing of associated injuries as healthy nerve ends are needed for repair. Closed injuries typically are considered for surgery after 3 months postinjury.[16]

After stabilization and assessment, sharply transected PNIs should prompt consultation for immediate repair or transfer. Transections without cleanly incised ends for anastomosis should still prompt discussion with specialty care and should have urgent follow-up. Closed injuries also should have urgent referral to specialty care not only for

possible surgery but also because such patients benefit from comprehensive rehabilitation services.

Wound Management

Wound management in the ED in part is driven by need for specialty care or transfer. Wounds should be decontaminated, explored, and assessed for foreign bodies, tetanus status updated, and pain addressed. Closure should be done in consultation with a specialist if the patient is not a candidate for immediate evaluation or transfer and if within the scope of the emergency provider's practice. Nerve ends can be tagged with suture to local structures to maintain nerve length, which facilitates better identification of nerves and preserves nerve length on re-exploration.

SPINAL CORD INJURY

Epidemiology

SCI affects approximately 300,000 individuals in the United States, with approximately 17,810 new cases occurring per year.[4] Like most trauma patients, these individuals tend to be younger and male. Overall, there is an almost 4:1 male predominance among new SCIs in the United States. Paralleling the aging population of the United States, the mean age of patients with acute traumatic SCI has risen gradually from 29 years to 43 years.[4] The most common age at the time of injury for the past several years is 19 years, and more than a quarter (25.61%) of all cases occur between the ages of 16 years and 22 years.[40] Non-Hispanic blacks make up approximately 24% of new cases despite representing approximately only 13% of the US population.[4]

The most common cause of SCIs varies with age and other factors, such as gender and race. Notably, the top 3 causes for both genders are the same: auto accidents, followed by falls and gunshot wounds (**Table 4**). Over the age of 45 years, falls become the leading cause of SCIs in the United States. The proportion of SCIs from vehicular accidents, acts of violence, and sports-related injuries have been declining from their peaks, while the proportion of SCIs from falls and medical/surgical complications have been increasing.[40]

Globally, approximately 750,000 traumatic SCIs occur each year.[41] Etiologies and consequences of SCIs vary in other countries. Higher-income countries tend to

Table 4
Ten most common causes of spinal cord injury by gender (all ages)

Rank	Cause of Spinal Cord Injuries Among Men (% of Total Cases)	Cause of Spinal Cord Injuries Among Women (% of Total Cases)
1	Auto accident (28.6)	Auto accident (46.6)
2	Fall (22.8)	Fall (23.1)
3	Gunshot wound (16.6)	Gunshot wound (9.3)
4	Motorcycle accident (7.1)	Medical/surgical complication (5.4)
5	Diving (6.5)	Diving (2.4)
6	Hit by falling/flying object (3.2)	Motorcycle accident (2.2)
7	Medical/surgical complication (2.3)	Pedestrian (2.0)
8	Bicycle (1.9)	Horseback riding (1.2)
9	Pedestrian (1.4)	Person-to-person contact (1.1)
10	Person-to-person contact (1.0)	Bicycle (1.0)

Data from National Spinal Cord Injury Statistical Center. 2019 Annual Statistical Report for the Spinal Cord Injury. Model Systems. University of Alabama at Birmingham: Birmingham, Alabama.

have older populations and see a bimodal distribution of traumatic SCIs, with peaks between the ages of 18 years and 32 years and at ages greater than 65 years. These older populations also see higher rates of tetraplegia with falls. Work-related falls in younger patients are more common in low-income countries.[42]

Although acute SCIs may involve any part of the spine, certain regions are more common. The needed flexibility of the cervical spine for flexion, extension, and rotation makes this region highly vulnerable to injury. The cervical spine is the most common site of injury in motor vehicle accidents and falls. Complete and incomplete tetraplegia consequently has made up approximately 60% of acute traumatic SCI cases since 2015.[4]

Key Anatomy and Pathophysiology

The spinal cord exits the foramen magnum and travels the length of the spine to the conus medullaris. The anterior-posterior diameter remains relatively constant, with transverse enlargements occurring in cervical and lumbar spine, around C5 and L3, respectively.[43] The bony boundaries of the spinal canal are relatively wide in the upper cervical spine, which can help protect the spinal cord from potentially devasting injuries in this area. The relative area of the cervical canal compared to the cord gets progressively smaller, increasing the chance of SCI in the lower cervical spine.[44]

The spinal cord contains several important paired nerve tracts:

- Corticospinal tracts—located both anteriorly/medially and posteriorly/laterally. These are the major descending motor pathways.
- Spinothalamic tracts—located anteriorly/laterally. These ascending pathways communicate light touch, temperature, and pain to the brain.
- Dorsal columns—located posteriorly/medially. These ascending pathways communicate deep touch, proprioception, and vibration to the brain.

The spinal cord branches into 31 pairs of spinal nerves, named for the anatomic location of their origin. This includes 8 cervical nerves (C1–C8) that exit the spinal column above their associated vertebra except for the C8 spinal nerve, which exits between the seventh cervical and first thoracic vertebra. The thoracic, lumbar, and sacral spinal nerves all exit below their associated vertebra.

Although SCI can occur in isolation, it frequently is associated with injuries of the vertebral column. Any underlying spinal disease can significantly increase the risk of injury to the bony spine and consequently the spinal cord. Many examples are associated with aging (like cervical spondylosis and osteoporosis). Spinal arthropathies like ankylosing spondylitis or rheumatoid arthritis may affect younger patients as well.[45,46] Additionally, congenital conditions like the atlantoaxial instability seen in Down syndrome and medication side effects like corticosteroid-induced osteoporosis may place patients at increased risk.[47]

The mechanism of any traumatic neurologic injury may be classified broadly as blunt versus penetrating. Blunt mechanisms are the leading cause of trauma in general and can cause SCI through excessive flexion/extension, rotational movements, shearing, or compressive forces. Penetrating injuries may be due to bullets, knives, or other missiles (like shrapnel) related to the traumatic event. This mechanism classically produces a transection injury of the spinal cord or vertebral fractures with associated SCIs. In rare cases, indirect damage to the spinal cord may occur. High-velocity missiles may cause contusion of the spinal cord as their kinetic energy dissipates despite never physically violating the spinal axis.[48] Case reports describe this phenomenon also occurring with low-velocity bullets.[49]

Classification of Spinal Cord Injuries

The source of SCIs may be primary or secondary. Primary injury encompasses all the initial mechanical insults (eg, compression, shearing, and laceration) affecting nerves at the time of injury. Secondary injury occurs over the following minutes to hours and causes further damage to the spinal cord through edema and additional cellular death. Secondary injury is a complex and poorly understood collection of processes like hypoxia, inflammation, and ischemia but represents an important therapeutic target for emergency physicians and spinal cord specialists.

The degree of injury is classified broadly as complete or incomplete. A complete injury causes total loss of sensation and motor function below the level of injury. Incomplete injuries are highly variable with symptoms that may range from relatively minor to near-complete paralysis. The most widely accepted scale for classifying SCI severity is the American Spinal Injury Association (ASIA) Scale. Grade A is assigned to patients with a complete cord injury, whereas grades B, C, and D identify progressively less severe degrees of incomplete injury. ED providers should be familiar with the ASIA International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) worksheet (**Fig. 5**). Its use allows for a rapid and accurate assessment of a patient's deficits, clear communication with specialists, and longitudinal assessment of the patient.

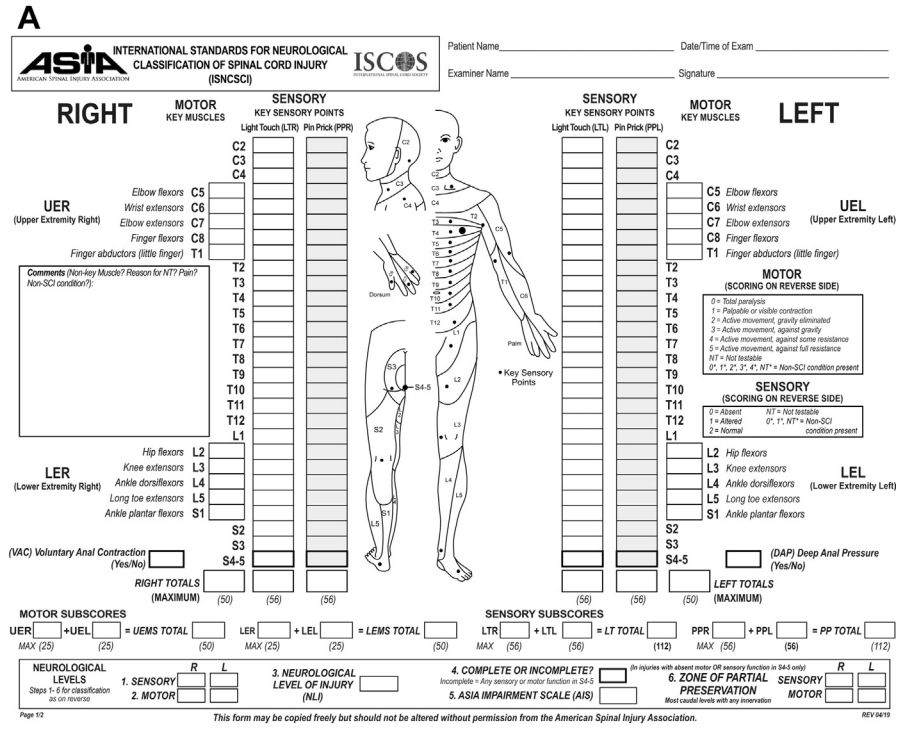

Fig. 5. (*A*) Page 1 of the ISNCSCI worksheet, including dermatomal map and key motor assessments. (*B*) Page 2 of the ISNCSCI worksheet, including motor and sensory grading scales and overall ASIA impairment scale. (*From* ©2020 American Spinal Injury Association; reprinted with permission.)

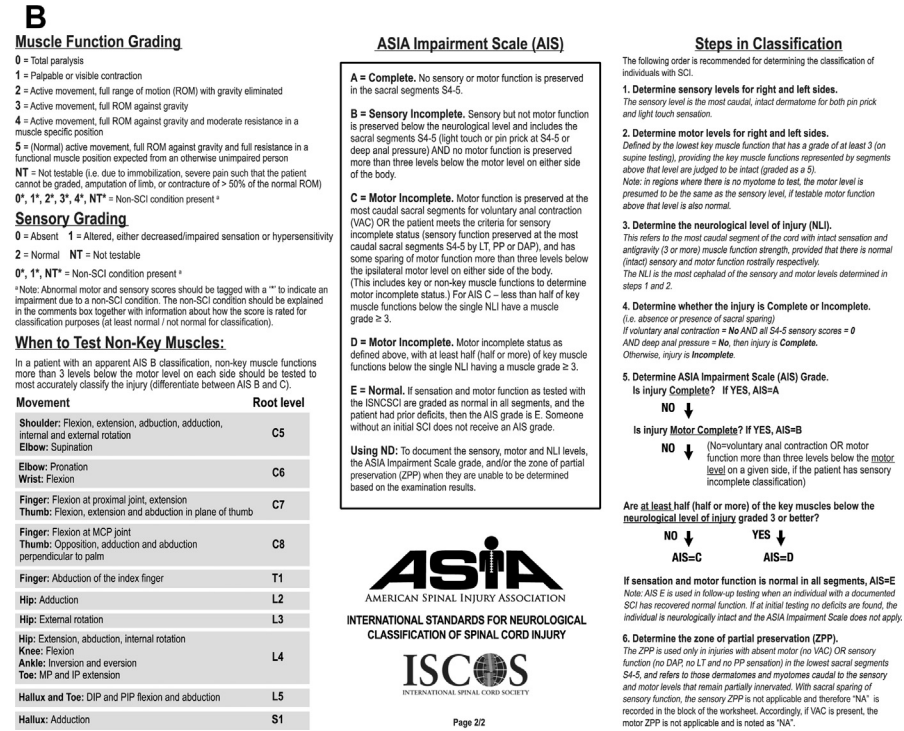

Fig. 5. (*continued*)

Presentation and Examination of Spinal Cord Injuries

Initial evaluation can be challenging, and providers must have a low suspicion for suspecting SCI in trauma patients. Mechanism and associated injuries can be important clues (**Table 5**). High-energy blunt trauma should raise concern, because most SCIs in

Table 5
Classic mechanisms of traumatic spinal cord injuries and commonly associated spinal and systemic injuries

Mechanism	Common Spinal Injuries	Common Associated Injuries
Car accident	Variable	Variable
Motorcycle accident	Thoracic injuries	Head injury (especially if unhelmeted)
Pedestrian struck	Variable	Lower limb fractures
Fall from a height	Thoracolumbar injuries if feet first	Pelvic and lower limb fractures
Diving	C1 burst fracture, C5–C6 fractures	Head injury
Winter sports	Thoracolumbar injuries	Variable
Football/rugby	Cervical injuries	
Gunshot wounds	Variable	Variable

Data from Aito S, D'Andrea M. Clinical Assessment in Spinal Cord Injury. In: Chhabra HS, ed. *ISCoS Textbook on Comprehensive Management of Spinal Cord Injuries*. Wolters Kluwer; 2015.

the United States occur during blunt trauma and as many as 80% of SCI patients have associated polytrauma.[4,50] The emergency provider must remember that SCIs also can result from a low-energy mechanism, especially within vulnerable populations like the elderly.

Associated injuries and contributing factors may limit the ability to get an accurate history. SCIs may occur in the context of substance use and up to 35% of cases may be associated with moderate or severe traumatic brain injury.[51] Thus, providers should assume any confused or unconscious trauma patient to have a possible SCI until proved otherwise.

Patients who can provide a history may describe symptoms concerning for SCIs. Any report of spinal pain, sensory loss, weakness, or other potentially neurologic symptom (eg, urinary retention) should raise concern.

Initial physical examination of a patient with suspected SCI should not differ from that of any other trauma patient. It should follow a protocolized trauma algorithm, such as the primary and secondary surveys of advance trauma life support (ATLS), with additional emphasis on spinal precautions.

The primary survey in the ED may reveal several key complications in severe SCI patients (**Table 6**).

The secondary survey better characterizes the injury or even recognize subtle SCI not identified during the primary survey. Providers should assess major myotomes and dermatomes bilaterally (**Table 7**). Approximately 20% of patients present with a recognizable spinal cord syndrome (**Table 8**).[52]

A rectal examination is required in all cases of potential SCI because decreased tone may be the only presenting abnormality. This examination also is essential for distinguishing incomplete versus complete injuries, which greatly affects prognosis and potentially the timing of interventions. The anal mucocutaneous junction is the lowest dermatome (S4/S5) and should be assessed by light touch and/or deep anal palpation (DAP). DAP is performed by inserting the provider's index finger and applying gentle pressure against the anorectal wall or by squeezing the anus between the examiner's inserted index finger and external thumb. Although DAP is not required for the sensory evaluation, a digital rectal examination is required to assess voluntary contraction of

Table 6
Key complications of acute spinal cord injury to identify during primary survey

Primary Survey Component	Key Assessments
Airway	• Exclude associated face/neck injuries that may directly compromise the airway (eg, swelling, bleeding, deformity). • Identify if paralysis prevents patient from protecting the airway (eg, insufficient cough to clear secretions).
Breathing	• Use continuous pulse oximetry and capnography. • Insufficient oxygenation and/or ventilation may be due to SCI (eg, paradoxic abdominal breathing) or an alternative injury (eg, pneumothorax, hemothorax, or flail chest).
Circulation	• Assess for systemic hypotension. • Hypotension, with or without associated bradycardia, may be seen in neurogenic shock. • Exclude other potential sources of shock first (eg, hemorrhage).
Disability	• Estimate level and severity of injury as quickly as possible.

Table 7
Major myotomes and dermatomes

Spinal Nerve	Associated Myotome	Associated Dermatome
C5	Elbow flexion (biceps)	
C6	Wrist extension	
C7	Elbow extension (triceps)	
C8	Finger flexion	
T1	Finger abduction	
T4		Nipple
T10		Umbilicus
L2	Hip flexion	
L3	Knee extension	
L4	Ankle dorsiflexion	
L5	Great toe extension	
S1	Plantar flexion	
S2–S4	Voluntary anal contraction	
S4/S5		Anal mucocutaneous junction

the external anal sphincter. The presence of priapism in male patients suggests, but is not required for, diagnosis of complete cord injury.

Diagnostic Evaluation of Spinal Cord Injuries

Laboratory evaluation

There are no laboratory tests specific to the diagnosis of SCI within the ED. Patients with significant blunt or penetrating injuries should empirically receive typical trauma laboratory studies, including any required prior to operative intervention or reversal of coagulopathy. Some laboratory abnormalities may identify potential contributors to secondary injury. Providers should identify and potentially treat

- Anemia
- Significant electrolyte abnormalities
- Hypoxia or hyperoxia

Ongoing research outside the ED uses the presence of inflammatory cytokines within a patient's cerebrospinal fluid to predict the degree of injury and likelihood of neurologic recovery but does not currently have a role in ED diagnosis.[55]

Imaging

Initial imaging should be used to identify unstable bony injuries, especially within the cervical spine. Historically, it was felt plain radiographs were sufficient to detect most bony injuries.[56] These images involve multiple views and can require manipulation of the patient. A significant portion of patients, despite this optimization, does not have sufficient visualization of the entire cervical spine on plain radiography and subsequently require CT.[57] Head-to-head comparisons of plain radiographs and CT are limited, but CT has been promoted consistently in obtunded patients and has a higher sensitivity.[58] National guidelines, such as those from the Eastern Association for the Surgery of Trauma (EAST), and more recent studies support the regular use of cervical CT in appropriate patients.[59–61] Patients with SCI demonstrate neurologic deficits and,

Table 8
Presentation of classic of spinal cord injuries

	Name	Mechanism/Pathology	Presentation	Pearls
Complete	Complete cord injury	Variable mechanisms All tracts damaged	Areflexic flaccid paralysis distal to level of injury Complete less of sensation distal to level of injury	The immediately adjacent dermatome and myotome may have partial function. Males may have transiently high-flow priapism at the time of injury, which rarely requires intervention.[54]
Incomplete	Anterior cord syndrome	Flexion, retropulsion of fracture fragments, or occlusion of the anterior spinal artery Damages anterior 2/3 of the spinal cord (corticospinal and spinothalamic tracts)	Motor function and sensations of pain/temperature are lost (below level of injury). Deep touch, pressure, vibration, and proprioception are preserved.	Poor likelihood of recovery[52]
	Brown-Sequard syndrome	Classically from a penetrating injury Lateral hemisection of spinal cord	True hemisection causes loss of ipsilateral motor function, ipsilateral light touch and proprioception, and contralateral pain and temperature sensation.	Incomplete hemisections are common and cause symptoms related to which tracts are involved.[52] Best prognosis for ambulation[52]
	CCS	Classically from a hyperextension injury Buckling of the ligamentum flavum causes localized injury to the center of the spinal cord	Bilateral weakness, greatest in the upper extremities, and greatest in the distal muscle groups Variable sensory loss	One of the most common SCIs in adults (approximately 10% of cases) Has a favorable prognosis compared with other SCI syndromes[53]
	Conus medullaris syndrome	Traumatic injuries at T12 or L1 causing damage to the sacral cord	Urinary retention and stool incontinence with possible (usually mild) lower extremity involvement (mix of upper and lower motor neuron findings) Saddle anesthesia	Will have both upper and lower motor neuron signs
	Posterior cord syndrome	Mechanisms include hyperextension and occlusion of the posterior spinal artery.	Bilateral loss of vibration and proprioception	Traumatic causes are extremely rare.

Temporary	Spinal shock	Temporary physiologic injury to the spinal cord (nerve pathways remain anatomically intact)	Flaccid paralysis, hypotonia, areflexia Loss of sensory function Loss of autonomic function	Usually lasts hours to days, but weeks also are possible Hypotension is not a defining feature, but it may cause low blood pressure via neurogenic shock.
Non-SCI	Cauda equina syndrome	Injury to lumbosacral nerve roots and therefore not a true SCI	Bladder/bowel dysfunction and possible lower extremity involvement (lower motor signs only) Saddle anesthesia	Often has asymmetric lower extremity weakness[52] Better prognosis for recovery given regenerative properties of nerve roots[52]

therefore, likely already require a head CT in the setting of trauma. It is time efficient and appropriate to pair this head imaging with a cervical CT.

Not all trauma patients require cervical imaging and several well-known clinical decision rules may be used to limit imaging in low-risk patients. The National Emergency X-Radiography Utilization Study (NEXUS) criteria were developed in the late 1990s and validated shortly afterward and demonstrate high sensitivity but relatively poor specificity.[62] The Canadian C-Spine Rule (CCR) was developed shortly afterward and appears to have better sensitivity, better specificity, and an overall lower rate of imaging utilization than the NEXUS criteria.[63] The CCR notably excludes patients 65 years of age or older, which is reasonable given the possibility of significant injury despite a relatively minor mechanism in this demographic.[64] Both of these clinical decision rules exclude patients with neurologic findings, such those expected in SCIs.

Unfortunately, no validated clinical decision rules exist for imaging other areas of the spine and providers should defer to clinical judgment. The American Association for the Surgery of Trauma TL-Spine Multicenter Study Group proposed the following criteria for imaging of the thoracolumbar spine in the setting of trauma[65]:

- Physical examination findings (pain, tenderness, and deformity)
- High-risk mechanism (eg, crush injury, motor vehicle collision with roll-over or ejection, or pedestrian struck)
- Neurologic deficit
- Glasgow coma scale less than 15
- Distracting injury
- Intoxication
- Age greater than 60 years

The American College of Radiology Appropriateness Criteria for suspected spine trauma rate noncontrast CT of the thoracic and lumbar spine as “usually appropriate” in the setting of blunt trauma meeting these criteria.[66] It is important to recognize there is a significant occurrence of noncontiguous vertebral fractures.[67,68] Consequently, the entire spine should be imaged if 1 vertebral column fracture is identified.

CT imaging can suggest SCI but does not routinely allow direct visualization of the spinal cord. MRI is an important adjunct in SCI assessment and captures the spinal cord and associated soft tissue in exquisite detail. SCIs can occur in the absence of vertebral fracture from processes better seen on MRI, such as hemorrhage or edema. Consequently, MRI still should be considered in patients even after a negative CT if concerning features still exist (eg, neurologic deficit).[69] The 2015 EAST guidelines, acknowledging very low-quality evidence, provided a conditional recommendation that cervical collars can be removed from obtunded adult blunt trauma patients after high-quality cervical spine CT.[70]

Decisions about MRI typically should be made in conjunction with a surgical consultant. There are numerous potential indications for MRI in the setting of spinal trauma, such as[71]

- Distinguishing hemorrhagic versus nonhemorrhagic SCIs (important for prognosis)
- Distinguishing acute versus chronic vertebral fractures
- Identifying ligamentous injuries (may be missed on CT)
- Identifying disc herniations (important before some closed reduction attempts)
- Identifying hematomas causing cord compression (important for operative planning and before some closed reduction attempts)

- Identifying vascular injuries that can cause spinal cord infarctions

The term, *SCI without radiographic abnormality* (*SCIWORA*), was developed prior to the widespread availability of MRI. It usually is described in pediatric populations. The term, *adult SCIWORA*, is more controversial. Central cord syndrome (CCS), for example, is relatively common and frequently occurs without any vertebral fracture. MRI is essential to the diagnosis and prognosis of such cases.

Management of Spinal Cord Injuries

The initial management of a patient with suspected SCI should not differ from that of any other trauma patient, other than emphasizing stabilization of the spine. Although SCIs can be lethal, such deaths often occur in the prehospital environment secondary to respiratory arrest. The identification and treatment of more common life-threatening injuries (hemorrhage, pneumothorax, splenic laceration, and so forth) take precedent in patients with SCIs who survive to the ED. Providers should follow a standardized trauma algorithm, such as ATLS, to identify and address such injuries.

That said, patients with SCIs are a vulnerable population at risk of unique complications. Providers must promptly take several key steps (**Table 9**) and consider the following therapeutic actions and medical complications:

- Spinal motion restriction
- Respiratory compromise
- Hemodynamic compromise
- Minimizing secondary injury
- Surgical intervention

Table 9
Key management items for patients with acute traumatic spinal cord injury in the emergency department

Key Step	Key Details
Spinal motion restriction	• Apply a cervical collar • Miami J or Philadelphia collar preferred
Medical resuscitation	• Exclude other life-threatening injuries • Obtain definitive airway if indicated • Maintain oxygen saturation >92% • Maintain MAP >85–90 mm Hg
Neurologic examination	• Ideally per the ISNCSCI worksheet • At minimum assess for major motor/sensory deficits, rectal tone/sensation, and an estimated spinal level of deficit
Radiographic evaluation	• CT is appropriate initial imaging • Whole-spine imaging if any vertebral fracture is identified • Obtain MRI if indicated
Early consultation with a surgical specialist	• Assess need for closed reduction • Assess need for early surgical intervention
Avoid steroid administration	• Unless specifically requested after discussion with surgeon or mandated by institutional policy
Transfer (if needed)	• Ideally to a definitive SCI care facility • Consider need for intubation prior to transport

Spinal motion restriction

Most patients with potential SCI arrive with a cervical collar and often on an EMS backboard. Patients should be removed from the backboard as soon as is safely allowed. Cervical collars should remain only for suspected or confirmed cervical fractures and ideally be exchanged from EMS collars to a Miami J collar or equivalent.[72] As emphasized within the 2018 joint policy statement from the American College of Emergency Physicians and the American College of Surgeons Committee on Trauma, the term, *spinal motion restriction*, is preferred to the term, *spinal immobilization*.[73]

Efforts should be made throughout the physical examination and subsequent management to minimize movements of the spine. These includes keeping the patient flat, using log-roll techniques, and maintaining cervical spine support during movement or procedures like intubation. Head of bed elevation still can be accomplished if there is concern of concomitant head injury with reverse Trendelenburg positioning.

Respiratory compromise

A significant proportion of patients with SCI require intubation, especially those with cervical injury.[74] Patients with complete cord injuries above C5 should be intubated prophylactically.[72] Intubation should be performed with manual in-line stabilization in which a second individual maintains the patient's cervical spine in a neutral position after careful removal of the anterior component of the cervical collar.[75] Video-assisted laryngoscopy can be used to maintain a neutral position. This is distinctly different from the sniffing or bed-up–head-elevated positions commonly used during ED intubations. The second provider may approach from either the head or side of the bed. Rapid sequence intubation is appropriate, although fiberoptic intubation also may be considered if time permits.

High cervical spine injuries above C3 typically result in respiratory and subsequently cardiac arrest unless rapidly intubated. Traumatic arrest in the field without signs of alternative causes may provide a clue to a high cervical injury.

Patients with incomplete and/or lower SCIs also may require intubation. Although the phrenic nerve to the diaphragm originates from the C3–C5 spinal nerves, the innervations of additional respiratory and accessory muscles originate lower in the spine. The intercostal muscles, for example, are controlled by the thoracic spinal nerves T1–T11 via intercostal nerves. Loss of the internal intercostals for inhalation can reduce vital capacity and lead to atelectasis and hypoxia. The intercostals are critical to stabilizing the chest wall. Paralysis of these muscles consequently allows the chest wall to contract with activation of the diaphragm, leading to paradoxical abdominal breathing (ie, quad breathing) and a corresponding severe drop in ventilatory ability.

The external intercostals and abdominal wall muscles assist with active exhalation and coughing. Abdominal wall muscles are innervated by a combination of branches from the lower intercostal nerves (T6–T12) and the ilioinguinal/iliohypogastric nerves (L1).[76] Compromise of these muscles can prevent a patient from coughing and clearing secretions adequately. This issue may be exacerbated in acute tetraplegia because these patients may develop increased secretions and bronchial constriction (theorized to be secondary to unopposed vagal activity).[77]

Given the many respiratory complications of acute SCIs, providers should use continuous pulse oximetry and continuous capnography to monitor the oxygenation and ventilatory effort. There are numerous potential indications for intubation in SCIs, and emergency providers should have a low threshold to preemptively establish a definitive airway under controlled conditions (**Table 10**).

Table 10
Potential indications for intubation in spinal cord injury

Indication	Details
Severe injury	• Complete SCI of C4 or above
Airway compromise	• Physical compromise • Inability to clear secretions
Work of breathing	• Persistent or increasing tachypnea • Persistent or progressive hypoxemia • Elevated or progressive end-tidal carbon dioxide • Consider in cases of subjective shortness of breath and/or development of paradoxic abdominal breathing
Travel	• Consider preemptively for transfers to other facilities (especially cervical) • Consider ahead of prolonged studies (such as MRI)

Hemodynamic compromise

Cord injuries above T6 can cause hemodynamic compromise. Loss of sympathetic outflow to the peripheral vasculature and heart causes a distributive shock picture with decreased vascular resistance and, sometimes, bradycardia. This process is known as neurogenic shock. The exact incidence is unknown, but 1 review estimated it to occur in 20% of cervical injuries and 7% of thoracic injuries.[78] Bradycardia is not required for diagnosis but tends to occur with more rostral injuries. There are no standardized cutoffs, but several studies have used systolic blood pressure of less than 100 mm Hg and heart rate less than 50 beats per minute to identify neurogenic shock. Hypothermia also may be seen.

Neurogenic shock should be a diagnosis of exclusion. Emergency providers should thoroughly evaluate for other causes of hypotension or bradycardia (eg, hemorrhagic shock, cardiac tamponade, tension pneumothorax, medications, and age). Hypovolemic/hemorrhagic shock (compared to neurogenic) classically demonstrates tachycardia instead of bradycardia, cool skin instead of warm, and reduced urine output instead of normal.

If a diagnosis of neurogenic shock still is suspected, then patients should initially receive intravenous fluids (or transfusion, if indicated) to mitigate the underlying vasodilatory effects.[79] Intractable hypotension should be treated further with vasopressors. Historically, patients with neurogenic shock received phenylephrine or dopamine. More recent guidelines recommend norepinephrine as the first-line agent because of its more favorable side-effect profile.[72] Providers should target a mean arterial pressure (MAP) of 85 to 90 mm Hg. Atropine may be used for bradycardia.

Minimizing secondary injury

Damage to the spinal cord continues after the initial injury. Emergency providers should take steps to minimize this ongoing harm. Key principles include avoiding any physiologic extremes, such as hypoxia/hyperoxia or hyperthermia. Hypotension should be corrected. The role of therapeutic hypothermia in SCIs is unclear and without evidence to recommend routine use at this time, but it remains an active area of research.[80,81] Regardless of this research, fever should be avoided.

Current guidelines recommend a MAP goal greater than 85 mm Hg for all patients with acute traumatic SCIs.[82] Several early uncontrolled case series arbitrarily used this value for 7 days.[83,84] Subsequent study has continued to suggest neurologic

improvement in patients who avoid MAPs less than 85 but still consists of uncontrolled case series and retrospective cohorts with an overall low quality of evidence.[85,86] Given current recommendations, ED patients with SCIs who are below this target should receive intravenous fluids and, if necessary, vasopressors to maintain this blood pressure goal. As discussed previously, norepinephrine is the preferred agent if vasopressor support is required. There are at least 2 ongoing randomized controlled trials comparing the traditional MAP goals of 85 mm Hg to 90 mm Hg to lower targets of 65 mm Hg to 70 mm Hg.[81]

Corticosteroids historically were assumed to have a regular role in minimizing secondary injury after SCIs with near ubiquitous use despite never being studied outside of animal models. Subsequent studies on this topic, typically using methylprednisolone, have generated controversial results. The National Acute Spinal Cord Injury Study (NASCIS) I was one of the first randomized controlled trials to look at minimizing secondary injury but only included alternative doses of methylprednisolone without a placebo because of the untested but prevailing assumption of benefit.[87] NASCIS II included a placebo arm but did not show benefit in the primary analysis.[88] Subsequent subgroup analysis suggested some motor benefit to those receiving methylprednisolone within the first 8 hours of injury. This finding prompted NASCIS III, which again excluded a true placebo group and instead further assessed timing (within 3 hours vs within 8 hours of injury, respectively) and duration (24 hours vs 48 hours, respectively).[89]

Subsequent meta-analyses has questioned the net benefit of such protocols given the relatively mild reported motor benefit and competing concerns of side effects.[90,91] The previous trials have shown or suggested higher rates of wound infections, severe pneumonia, sepsis, and gastrointestinal complications.[55] In recent years, the use of corticosteroids in SCIs has decreased significantly and most related surgical or trauma societies either have recommended against use or that insufficient evidence exists to recommend this therapy.[82,92,93] The AO Spine 2017 guidelines remain the lone major exception and still propose methylprednisolone be considered if initiated within 8 hours of a cervical injury.[94]

Surgical interventions

Emergency providers should discuss cases early with a surgical specialist once an injury has been identified. Approximately 80% of patients in the National Spinal Cord Injury Statistical Center's registry required at least 1 major surgical intervention (internal fixation, laminectomy, neural canal restoration, or open reduction) during their index hospitalization.[40] Unfortunately, the exact indications and timing for surgery and/or closed reduction are not well defined and remain an active area of research. The need for timely decompression and/or stabilization of an SCI in the setting of polytrauma should be balanced against any additional potentially life-threatening injuries.

The goal is to minimize additional secondary injury through decompression and prevent subsequent acute injuries through stabilization. In some cases, this may be achieved by closed reduction. This technique is appropriate in awake individuals with a reliable neurologic examination and cervical injuries amenable to traction reduction, including certain fractures, subluxations, and/or dislocations. It can be performed with a halo ring (for lighter traction weights) or Gardner-Wells skull tongs (capable of handling higher weights).[95] This technique is not appropriate for those with calvarial injury or abnormality (eg, fracture, prior bony cranial intervention, or congenital abnormality) or those with degenerative diseases of the spine or injuries that may worsen with longitudinal traction (eg, hangman fracture). Closed reduction still may be attempted in some obtunded patients after a prereduction MRI.[96]

The need for immediate surgical intervention should be determined through consultation with an emergency provider's local neurosurgical or spine specialist. Practice patterns can vary, but common indications include ongoing compression of the spinal cord, progressive neurologic deficits, and/or unstable spinal injuries. Historically there was concern that early intervention was associated with increased complications and poorer outcomes. More recent research has suggested that early intervention, which unfortunately has variable definitions between 8 hours and 72 hours, may benefit outcomes without any significant increase in complications.[81] Some surgeons still may prefer a delayed approach in complete SCI and CCS, although this practice also has been challenged recently with AO Spine weakly recommending in 2017 that early surgery be considered for CCS.[97,98]

SUMMARY

Traumatic injuries to the nervous system remain a worldwide challenge. It is imperative emergency providers maintain a low index of suspicion for both PNIs and SCIs, especially in the context of polytrauma, because these diagnoses carry significant implications for morbidity and mortality in these patients. Although some patients may be managed conservatively, it is crucial emergency providers identify cases requiring emergent intervention and be familiar with the most significant consequences.

CLINICS CARE POINTS

- The ulnar and peroneal nerves are the most commonly injured peripheral nerves in the upper and lower extremities, respectively.
- Peripheral nerve injury should prompt consideration of associated fractures and arterial injuries.
- Sharp nerve transections should be repaired within 3 days, open/ragged injuries within 3 weeks, and closed injuries within 3 months.
- Use tools like continuous capnography to monitor for respiratory insufficiency after acute spinal cord injury. Maintain a low threshold for intubation.
- Fluid resuscitation and, if needed, norepinephrine should be used to treat neurogenic shock and/or achieve higher mean arterial pressure goals.
- Acute traumatic spinal cord injuries should be discussed promptly with a specialist as early intervention may be beneficial.

DISCLOSURE

The authors have nothing to disclose.

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