Trauma: Spinal Cord Injury

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KEYWORDS

• Spine • Spine trauma • Spinal cord injury • Spinal cord syndromes • Spinal shock • Spine immobilization

KEY POINTS

- Hypotension following trauma should be considered secondary to hemorrhage until proven otherwise, even in patients with early suspicion of spinal injury. Neurogenic shock and spinal shock are separate, important entities that must be understood.
- Hypoxia and hypotension should be aggressively corrected because they lead to secondary spinal cord injury, analogous to traumatic brain injury. Critical care support of multiple organ systems is frequently required early after injury.
- Early spinal decompression may lead to improved neurologic outcomes in select spinal cord injuries, and prompt consultation with spine surgeons is recommended.
- Computed tomography (CT) is the gold-standard screening study for evaluation of the spine after trauma and has significantly greater sensitivity and specificity compared with plain radiographs.
- High-quality CT imaging without evidence of cervical spine injury may be adequate for removal of the cervical immobilization collar in obtunded patients.

INTRODUCTION

Traumatic spine and spinal cord injury (SCI) occurred in roughly 17,000 US citizens in 2016, with an estimated prevalence of approximately 280,000 injured persons.[1] Although the injury has historically been a disease of younger adult men, a progressive increase in SCI incidence among the elderly has been reported over the last few decades.[2] Upwards of 70% of SCI patients suffer multiple injuries concomitant with spinal cord trauma, contributing to the high rates of associated complications during the acute and long-term phases of care.[3] SCI is associated with significant reductions in life expectancy across the spectrum of injury and age at time of insult.[1]

Patients who survive the initial injury face significant risks of medical complications throughout the rest of their lives. More than half of all SCI patients will develop complications during the initial hospital stay, with higher rates corresponding to increased

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injury severity, the presence of associated traumatic brain injury, and cerebrovascular damage occurring with cervical spine injury.[4,5] SCI may result in numerous multisystem complications particularly during the acute phase of care with long-term complications often related to infectious morbidity (Table 1). For the trauma/acute care surgeon managing SCI patients during the acute phase of injury, respiratory compromise and shock are of primary concern, as discussed later.

During the initial presentation and evaluation of SCI patients, almost all other injuries should take precedent in both evaluation and management, unless the SCI is impeding the airway (cervical spine) or the hemodynamics (neurogenic shock). No emergent imaging of the spine is required before a laparotomy or other life-saving surgical intervention. Spinal immobilization is adequate for initial prevention of further injury while addressing sources of hemorrhage. Prevention of hypotension and hypoxia is also critical to mitigating further neurologic injury. Even if one of the rare spinal cord emergencies is encountered, such as a progressively worsening examination with cord compression that requires surgical decompression, your job will be to stabilize the patient and address any other life-threatening injury before intervention by a spine surgeon. Hypotension in the setting of a suspected acute traumatic SCI should always be first assumed to be due to hypovolemia/hemorrhage, until ruled out.

ASSESSMENT OF SPINAL CORD INJURIES

The critical step in early evaluation of patients with possible SCI is recognition of patients at risk and a focused, yet thorough neurologic examination. Too often the

Table 1
Organ system complications following spinal cord injury

Organ System	Complications
Cardiovascular	Bradycardia/dysrhythmia Cardiac arrest Cardiogenic pulmonary edema
Pulmonary	Hypoventilation/respiratory failure Poor secretion control Acute respiratory distress syndrome Aspiration Pneumonia
Gastrointestinal	Gastric dysmotility Adynamic ileus Gastritis and ulceration Pancreatitis
Hematologic	Venothromboembolism
Neurologic	Neurogenic shock Depression Posttraumatic stress disorder Anxiety Autonomic dysreflexia
Genitourinary	Bladder dysfunction Urinary tract infection Priapism
Integument	Pressure ulceration

Adapted from Stricsek G, Ghobrial G, Wilson J, et al. Complications in the management of patients with spine trauma. Neurosurg Clin N Am 2017;28:150–2; with permission.

steps of physical examination are deferred to the all-knowing computed tomographic (CT) scanner. This delay can slow recognition of SCI and establishment of baseline function and delay consultation of appropriate specialists and the initiation of preventive measures avoiding further secondary injury. The performance of such an examination is frequently overlooked in busy trauma bays. The main points are that in addition to global neurologic disability (Glasgow Coma Scale and pupil examination), the secondary survey of the patient should include sensory and motor testing in upper and lower extremity muscle groups as well as an anorectal examination for tone and sensation. **Table 2** lists the key muscle groups and their corresponding motor level that should be checked to determine the motor level of the injury. Both upper and lower extremities and the right and left sides should be tested, because certain syndromes may cause "skip" patterns or have unilateral asymmetric deficits (eg, central cord syndrome, Brown-Séquard). Both the motor and the sensory level should be clearly documented in the neurologic portion of the admission history and physical examination as well as communicated to any consulting spine provider.

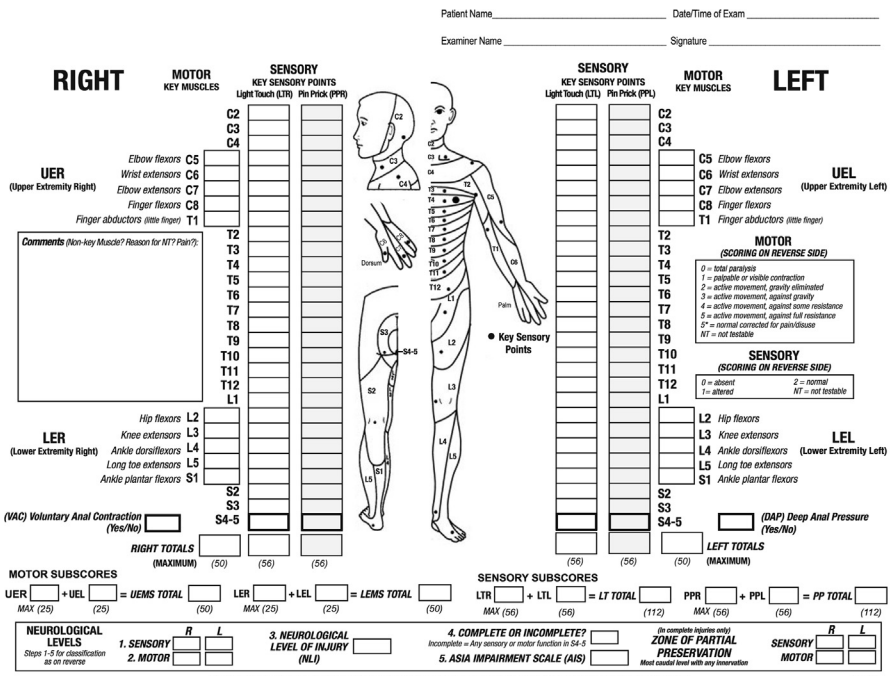
The distinction of complete and incomplete cord injury should be made because this may influence operative decision making such as decompressive laminectomy or removal of bone fragments that are compressing the spinal cord. A "complete" injury is an injury pattern in which there is absolutely no spine-mediated neurologic function below the level of the injury. An "incomplete" injury is one in which there is *any* function below the level of injury, typically in the form of intact sensation (such as perineal) or slight distal motor function. Sacral root sparing, which may allow some residual anal sphincter function or sensation or slight movement of a great toe, is an indication that the injury is incomplete and carries a better prognosis for recovery of some degree of neurologic function.

The American Spinal Injury Association (ASIA) International Standards for Neurological Classification of SCI is the most frequently used and studied spinal injury severity assessment score.[6] ASIA injury scoring is dependent on the presence or absence of motor function and sacral nerve root sparing below the level of injury (**Fig. 1**). Knowledge of which muscle group is fired by each spinal cord level and a more thorough checking for neurologic function distal to the apparent spinal cord level (including

Table 2
Muscle function tested on physical examination and the corresponding motor level

Motor Level	Muscle Function
C5	Elbow flexion
C6	Wrist extension
C7	Elbow extension
C8	Finger flexion
T1	Finger abduction
L2	Hip flexion
L3	Knee extension
L4	Ankle dorsiflexion
L5	Great toe extension
S1	Ankle dorsiflexion

Adapted from Branco F, Cardenas DD, Svircev JN. Spinal cord injury: a comprehensive review. Phys Med Rehabil Clin N Am 2007;18(4):651–79, v; with permission.



Muscle Function Grading

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated

3 = active movement, full ROM against gravity

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position

5 = (normal) active movement, full ROM against gravity and full resistance in a functional muscle position expected from an otherwise unimpaired person

5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present

NT = not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of > 50% of the normal ROM)

Sensory Grading

0 = Absent

1 = Altered, either decreased/impaired sensation or hypersensitivity

2 = Normal

NT = Not testable

When to Test Non-Key Muscles:

In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).

Movement	Root level
Shoulder: Flexion, extension, abduction, adduction, internal and external rotation **Elbow:** Supination	**C5**
Elbow: Pronation **Wrist:** Flexion	**C6**
Finger: Flexion at proximal joint, extension. **Thumb:** Flexion, extension and abduction in plane of thumb	**C7**
Finger: Flexion at MCP joint **Thumb:** Opposition, adduction and abduction perpendicular to palm	**C8**
Finger: Abduction of the index finger	**T1**
Hip: Adduction	**L2**
Hip: External rotation	**L3**
Hip: Extension, abduction, internal rotation **Knee:** Flexion **Ankle:** Inversion and eversion **Toe:** MP and IP extension	**L4**
Hallux and Toe: DIP and PIP flexion and abduction	**L5**
Hallux: Adduction	**S1**

ASIA Impairment Scale (AIS)

A = Complete. No sensory or motor function is preserved in the sacral segments S4-5.

B = Sensory Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.

C = Motor Incomplete. Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments (S4-S5) by LT, PP or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body.
(This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.

D = Motor Incomplete. Motor incomplete status as defined above, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade ≥ 3.

E = Normal. If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

Using ND: To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.

Steps in Classification

The following order is recommended for determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.

2. Determine motor levels for right and left sides.
Defined by the lowest key muscle function that has a grade of at least 3 (on supine testing), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5).
Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.

3. Determine the neurological level of injury (NLI)
This refers to the most caudal segment of the cord with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively.
The NLI is the most cephalad of the sensory and motor levels determined in steps 1 and 2.

4. Determine whether the injury is Complete or Incomplete.
(i.e. absence or presence of sacral sparing)
*If voluntary anal contraction = **No** AND all S4-5 sensory scores = **0** AND deep anal pressure = **No,** then injury is **Complete.***
*Otherwise, injury is **Incomplete.***

5. Determine ASIA Impairment Scale (AIS) Grade:

Is injury Complete? **If YES, AIS=A** and can record ZPP (lowest dermatome or myotome on each side with some preservation)

NO ↓

Is injury Motor Complete? **If YES, AIS=B**

NO ↓ (No=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)

Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?

NO ↓ **AIS=C** **YES** ↓ **AIS=D**

If sensation and motor function is normal in all segments, AIS=E
Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Fig. 1. ASIA SCI evaluation and scoring sheet. (*From* the American Spinal Injury Association (ASIA). Available at: www.asia-spinalinjury.org/information/downloads. Accessed February 1, 2017; with permission.)

rectal examination) can help identify patients with incomplete injuries who may benefit from more urgent operative intervention. In addition, such an examination may help identify the possible injury location in order to better immobilize and prevent iatrogenic extension of the neurologic deficit. If spinal shock is present, it cannot be determined whether the injury is complete or incomplete until 24 to 48 hours after the shock period resolves.

Although imaging of the spine is often thought of as being primarily about the bony structures, it is important to remember that complete assessment for spine injury and spinal stability requires assessment of the bone, the ligaments, and the spinal cord itself. Traditional plain radiograph imaging of the spine only evaluates the bony component and has essentially been replaced by the widespread availability and efficiency of modern multidetector CT imaging. Although the utility of plain radiograph screening of the cervical spine in select low-risk patients has been extensively studied, numerous studies have demonstrated the superiority of CT versus plain radiograph for screening acutely injured patients with suspected or at risk for spine injury.[7–9] Negative predictive values approaching 100% have been reported in several trials, particularly with relation to the presence of a clinically unstable injury or injury requiring intervention.[10–12] Although CT is ideal for screening identification of bony injury and suggestion of alignment abnormalities, the diagnostic accuracy of CT for spinal cord, ligamentous, and soft tissue injury is exceeded by MRI.[13,14] Patients with deficits on examination, CT findings suggestive of injury to the cord, disks, ligaments, or nerve roots, or unexplained neurologic examination may benefit from early MRI for diagnostic and therapeutic decisions.

SPINAL SHOCK VERSUS NEUROGENIC SHOCK

The terms “spinal shock” and “neurogenic shock” are often both used inappropriately or incorrectly, or are confused for one another in the clinical setting. Neurogenic shock is the hemodynamic consequence of the SCI, classically characterized by hypotension due to vasodilation and increased perfusion of the lower extremities (also known as “warm shock”). In cases of higher SCI (cervical spine), hypotension may often be accompanied by paradoxical bradycardia. This pattern is a relatively unique and specific hemodynamic pattern to SCI and should prompt immediate evaluation and interventions. Cervical spine and high thoracic spine injuries may result in loss of sympathetic cardiac stimulation (bradycardia) and vasomotor tone in the lower body (hypotension) that will benefit from early initiation of vasopressor medication along with standard trauma resuscitation to restore intravascular volume status.

Although neurogenic shock refers to a hemodynamic pattern, spinal shock refers to the neurologic examination findings that may be seen after an acute SCI. The diagnosis of spinal shock is made in the presence of complete loss of reflexes below the level of injury, including the monosynaptic pathways. If spinal shock is present, this means that it is not yet known what the ultimate amount of functional recovery will be. You will have to wait until the spinal shock period is over. If spinal shock is not present, or it has resolved, then whatever neurologic deficits you have at that time are likely to be fixed and permanent. Thus, for the patient presenting with paralysis in spinal shock, an unknown amount of functional recovery may still occur. Spinal shock is diagnosed through evaluation of the bulbocavernosus and/or cremasteric reflexes. If these reflexes are absent, then the patient is in spinal shock, and when they return, the shock period has ended.[15] Once the period of spinal shock has ended and the bulbocavernosus and/or cremasteric reflexes have returned, then the neurologic examination at that time likely reflects what the permanent level and degree of deficits will be.

MANAGEMENT OF ACUTE SPINAL CORD INJURY

The management of SCI begins with spine precautions (logrolling, cervical collar) and protection from further injury. Spinal immobilization precautions do not mean lying flat and motionless once the initial trauma evaluation has been completed. Reverse Trendelenburg position up to 30° will greatly benefit, and participatory pulmonary toilet should begin if they are not intubated. Ensure adequate pain control to maximize tidal volumes. Have a low threshold for nasogastric decompression because gastric ileus often accompanies SCI with paraplegia or quadriplegia. Similarly, bladder dysfunction is common, and a urinary catheter should be placed if not already present. Begin management of pressure points with padding and frequent patient repositioning immediately for paralyzed patients. Do not forget the psychological and emotional aspects of these injuries, particularly in young acutely injured patients. Early mental health professional and/or chaplain consultation to begin helping the patient deal with the almost uniform depression and grieving over the loss of bodily function that accompanies these injuries is essential.

Similar to studies of traumatic brain injury, avoidance of hypotension (systolic blood pressure <90 mm Hg) and hypoxia is critical to avoiding further secondary SCI.[16,17] Neurogenic shock may manifest as hypotension, which is poorly responsive to fluid resuscitation but responds briskly to vasopressor agents. In pure neurogenic shock, there is no associated tachycardia; the extremities may be warm and dry rather than cold and clammy, and typically, the patient has a significant cervical SCI. Treatment involves judicious volume resuscitation and the use of vasopressors to support blood pressure. A purely peripheral vasoconstrictor such as Neo-Synephrine is often used, but in the multitrauma patient or the patient with associated bradycardia, a balanced vasopressor such as norepinephrine is a better choice. Maintaining the mean arterial pressure greater than 85 mm Hg for up to 7 days after injury has been associated with improved ASIA scores and is recommended in current guidelines.[18,19] The concept of spinal cord perfusion pressure monitoring through direct measure of intraspinal pressure (ISP) after placement of an intradural catheter has demonstrated significant promise. Small trials have demonstrated safety and efficacy of monitoring ISP at the site of maximal cord edema for up to 1 week after injury.[20–22]

Controversy persists over the utility of high-dose systemic steroids following blunt SCI. The series of National Acute Spinal Cord Injury Studies (NASCIS) I-III trials failed to demonstrate any difference in neurologic outcomes after high-dose methylprednisolone.[23–25] Post hoc subgroup analyses in the later NASCIS trials showed a 5-point (ASIA) motor scoring improvement across 15 muscle groups but did not correlate with any measure of functional improvement or effect upon disability. In contrast, patients receiving high-dose steroids consistently suffered increased rates of infectious complications, gastric ulceration, venothromboembolic events, wound complications, and trended toward increased mortality related to pulmonary complications. Numerous additional trials confirmed the NASCIS outcomes with similar complication profiles.[26,27] The use of high-dose steroids after penetrating SCI has consistently shown poor outcomes.[28–30] Previous guidelines had recommended intravenous steroid bolus and infusion for all patients with blunt SCI and no contraindication to treatment. However, this approach has now been widely abandoned because of the highly questionable clinical benefit identified in the NASCIS trials as well as more recent data indicating no benefit to steroid use. Recent evidence-based management guidelines recommend against the use of high-dose steroids for all acute SCI.[31] There may be a role for steroid use in highly select or atypical types of spinal cord trauma and injury, and this decision should be at the discretion of the managing spine surgeon.

Not infrequently, the examination of an injured patient will suggest neurologic deficit without any obvious supporting radiographic findings. These SCI without radiographic abnormalities (SCIWORA) represent a challenging group because limited evidence and support for treatment have been established. Unfortunately, the ultimate neurologic outcome in SCIWORA patients appears correlated with the initial deficits on examination similar to those patients with radiographic abnormalities and neurologic deficits.[32] MRI appears particularly useful in identifying subtle intraneural and extraneural abnormalities that may be associated with the neurologic deficits and correlate with outcomes.[33] Supportive care is similar to that described previously for those patients with neurologic injury and radiographic abnormalities. Despite increased reports on populations of SCIWORA patients, limited high-quality data are available, and no randomized controlled trials of management have been conducted.

The timing of surgical spinal decompression in the setting of compressive phenomena, such as epidural hematoma, cord edema or hemorrhage, or impinging bony fragments and foreign bodies, has been extensively studied. Preclinical models have demonstrated that the extent and duration of cord compression correlate with ultimate neurologic deficit.[34,35] Human series have suggested that early decompression in the first 8 to 24 hours after injury in those patients with incomplete SCI is associated with improved neurologic outcomes. In the Surgical Timing in Acute Spinal Cord Injury Study, patients with cervical SCI that underwent decompression in the first 24 hours after injury had a significant improvement in ASIA grade.[36] Additional studies have confirmed the benefits of early decompression for these incomplete patients; however, not all trials of time to decompression have shown neurologic improvement with early intervention.[37] Early surgical decompression is also associated with decreased pulmonary morbidity and duration of mechanical ventilation as well as decreased intensive care unit and hospital length of stay.[38,39] Unfortunately, surgical decompression in patients presenting with ASIA A complete injury has not shown significant improvements in neurologic outcomes.[40,41]

As previously noted, one of the early measures in the assessment and stabilization of a suspected SCI patient is the prevention of future mechanical injury due to instability of the spinal column. Although logroll precautions, maintenance of axial alignment, pressure point management, and attentive nursing care are generally adequate for thoracolumbar spinal protection, the cervical spine is generally protected with specialized cervical immobilization devices, or “c-collars.” Controversy persists regarding the actual effectiveness and necessity of these devices to maintain proper alignment and prevent skeletal motion.[42–45] In addition, these hard devices may be associated with development of pressure ulceration in upwards of 30% of patients.[46] For the obtunded trauma patient with associated brain injury or critical illness in whom the cervical spine cannot be cleared by standard imaging and physical examination, a particular challenge arises. Traditionally, these patients were left in a hard cervical collar until examinable or until an MRI or other imaging adjunct demonstrated no evidence of injury. Recent studies from multiple centers have demonstrated the safety of cervical collar removal after a negative high-quality CT given negative predictive values of 100% for unstable injuries.[12] As a result of these studies, practice patterns are changing with expected decreased utilization of MRI resources and prevention of complications of hard cervical immobilization devices.

MANAGEMENT OF PENETRATING SPINE TRAUMA

Penetrating SCI is most commonly secondary to gunshot wounds and typically results in complete SCI due to direct trauma to the cord and associated blast effect as well as

secondary hemorrhage and ischemia.[47] In civilian trauma centers, the thoracolumbar spine is the region most frequently injured. The management options for open spine trauma are not much different than those for closed spine trauma, even in patients with open vertebral column fractures. The wound must be managed with irrigation and debridement of all nonviable tissues and early antibiotic coverage. The choice of antibiotics is generally the same as for patients with open extremity fractures, and there are no good data on recommended duration of therapy (ranges of 48 hours to 10 days reported). Patients with associated intestinal injuries, particularly if those injuries communicate with the spinal column injury, may require broader coverage. These devastating injuries will obviously require multidisciplinary care for optimal outcomes.

Incomplete SCI, cauda equina syndrome, or evidence of cord compression after penetrating injury due to hematoma or bony fragments may benefit from surgical decompression.[48] In addition, surgical stabilization of unstable vertebral column injuries is necessary in any patient likely to survive the acute phase of care. Exploration of the injuries may be required for debridement, neurologic deterioration due to cord compression, or persistent cerebrospinal fluid leak. Wound infection, spinal column instability, and cerebrospinal fluid leaks were the most common complications in a series of penetrating SCI.[49] These penetrating injuries are frequently managed conservatively as outcomes have been shown to be equivocal or even more favorable in nonoperative management.[50]

AIRWAY MANAGEMENT

The need for appropriate airway management is of particular importance for patients with cervical SCIs. Most patients with high cervical SCIs will present with quadriplegia and respiratory distress or arrest and clearly require intubation. The difficult patient population is the lower cervical spine injury (C5-C7) and upper thoracic spine (T1-T6), who frequently present with no obvious respiratory distress due to the ability to continue shallow breathing. Be wary of these patients: previous reports have demonstrated that up to 50% will slowly decompensate and require a delayed emergent airway intervention.[51,52] This can result in secondary SCI due to hypoxia and trauma from manipulation during emergent intubation attempts. Over a period of several hours to days, the shallow breathing will result in progressive atelectasis, pulmonary consolidation or pneumonia, and finally, acute hypoxic decompensation. The insidious airway collapse in this setting can be severely harmful or even fatal and should be anticipated. A low threshold for intubating these patients semi-electively for the initial hospital period or before transfer may be prudent, because up to 30% will require intubation in the first 24 hours after cervical SCI.[53] Factors suggestive of early intubation include higher level of injury (above C5), complete paralysis, the presence of associated injuries (particularly chest wall or intrathoracic), and low lung volumes on chest radiograph. If you have the capability to measure and follow vital capacity, then this may be a useful adjunct to identify the patient progressing to respiratory failure.

Numerous publications discuss the methods for intubation in the patient with a cervical spine injury and the potential impact on spinal mobility. Direct laryngoscopy with manual in-line stabilization of the cervical spine during the procedure has been shown to be safe and effective in 2 large studies.[54,55] If available, fiberoptic intubation is safe and avoids significant spinal motion but requires manual stabilization during the procedure as well if the collar is released. Finally, a surgical airway is always an option and may be necessary for patients who will require long-term mechanical ventilation and/or pulmonary toilet. Consideration should be given to potential incision location for anterior cervical spine surgical stabilization if indicated. Although the definitions of

what constitutes "early" tracheostomy vary in the literature, those patients who require or are projected to require mechanical ventilation for more than 2 weeks after injury certainly may benefit from tracheostomy. Improved secretion control, enhanced facilitation of mechanical ventilation weaning, patient comfort, ability to participate in rehabilitation, and possibly reduced risks of pneumonia and resource utilization have been associated with tracheostomy in SCI patients.[56–58]

SPINAL CORD SYNDROMES

Although simple complete traumatic SCI is relatively straightforward, with a dense and complete neurologic deficit below the level of injury, there are several spinal cord syndromes involving injuries to an isolated segment that have a much more varied and subtle presentation. These syndromes can be easily missed or misdiagnosed if a thorough neurologic examination and appropriate differential diagnosis are not performed. **Table 3** reviews the cause, diagnosis, and management for the common spinal syndromes. Although relatively uncommon, these syndromes have very characteristic presentations and common etiologic/mechanistic factors that

Table 3
Cause, diagnosis, and management for the common spinal syndromes

Syndrome	Cause	Examination Findings	Management
Central cord	Hyperflexion or extension, usually elderly with existing spinal stenosis; most common syndrome	Motor weakness of arms > legs with sacral sensory sparing	No proven benefit of prolonged immobilization Course of steroids may benefit Physical therapy and rehabilitation Spinal decompression
Brown-Séquard (cord hemisection)	Spinal hemisection, often gunshot or knife wound	Ipsilateral loss of motor and proprioception; contralateral loss of pain and temperature sensation	Spinal stabilization if unstable Course of steroids Physical therapy
Anterior cord	Damage to anterior 2/3 of cord, usually direct injury or ischemia from anterior spinal artery injury	Loss of motor function and pain/temperature with preserved proprioception and light touch sensation	Worst prognosis with low chance of muscle recovery Physical and occupational therapy
Conus medullaris	Injury to sacral cord and lumbar nerve roots, upper lumbar (L1) fractures, disk herniation, tumors	Bowel, bladder, and sexual dysfunction with areflexia, normal leg motor function, bulbocavernosus present with high lesion	Emergent surgical decompression Course of steroids GM1 ganglioside (100 mg) intravenous (IV) Bowel/bladder training
Cauda equina	Injury to lumbar/sacral nerve roots, lumbar (L2 or lower), or sacral fractures, also pelvic fractures, herniated disk, tumors	Weakness or flaccid leg paralysis, high lesions spare bowel/bladder, bulbocavernosus absent	Emergent surgical decompression Course of steroids GM1 ganglioside (100 mg) IV Bowel/bladder training

the managing physician should be aware of. Central cord syndrome is almost always a flexion/extension injury in an elderly patient with preexisting spinal stenosis. Although spinal immobilization is often maintained in these patients because of the presence of neurologic deficits, it is typically not an unstable spine injury, and there is no proven benefit of immobilization with a cervical collar. Brown-Séquard (or "cord hemisection") is extremely uncommon and typically only seen after direct penetrating injury to the spinal cord that results in different unilateral and contralateral deficits. Anterior cord syndrome is typically a vascular cause related to injury or interruption of flow through the anterior spinal artery. For all of the spinal cord syndromes presenting with fixed and established defects, management is usually expectant and aimed at treating symptoms and pain.[59,60] However, for any patient with a progressively worsening neurologic deficit, emergent consultation with a spine surgeon for possible decompression should be a priority.

VENOUS THROMBOEMBOLISM AFTER SPINAL CORD INJURY

Venous thromboembolism (VTE) events and appropriate prophylaxis are major concerns in the acute and long-term management of SCI patients. A recent large population study of roughly 48,000 SCI patients found an approximately 2.5-fold increased risk of deep venous thrombosis (DVT) and 1.6-fold increased risk of pulmonary embolism compared with controls. The risks were greatest within the first 3 months following injury and with increasing age.[61] Additional studies confirm the heightened risk in the early period after injury and with increased patient age, pointing to the significance of falls in older patients.[62,63] Overall incidence of VTE after SCI in recent publications suggests an incidence of approximately 3% to 5%, with associated early mortality of 7.5%, but up to 20% in patients greater than 70 years of age.[2,63] For unclear reasons, the location of SCI along the spine may also be associated with VTE risk. Maung and colleagues[64] found upper thoracic SCI to be associated with a higher rate of VTE compared with other spinal level injury. Variation in reported incidence of VTE is likely due to the timing and modality of screening methods. Duplex and Doppler ultrasonography are the most frequently and conveniently used screening modalities today.[65] There is no clear consensus on the timing or schedule for screening after acute SCI; however, the systematic review by Furlan and Fehlings[66] suggests that weekly screening for asymptomatic DVT in the early high-risk period following SCI may be appropriate.

In recognizing the increased risk of VTE following SCI, optimal preventive measures are an important facet of care for these patients. Until recently, controversy persisted regarding the optimal treatment with mechanical and/or chemoprophylaxis. A series of studies has confirmed the superior efficacy of low-molecular-weight heparin (LMWH) versus unfractionated subcutaneous heparin for the prevention of VTE and a lower associated bleeding risk.[67,68] Optimal dosing of LMWH remains controversial. Studies suggest that standard daily or twice daily enoxaparin dosing may not achieve therapeutic anti-Xa levels in trauma patients; however, no consensus for dose adjustment currently exists.[69,70] Combined preventive therapy with gradient elastic stockings or sequential pneumatic compression devices of the lower extremities and LMWH may offer an even greater reduction in VTE risk after SCI.[71] Early initiation of chemoprophylaxis (<72 hours from injury) has been associated with a significant reduction in the incidence of VTE after SCI (2% vs 26%) and recommended in at least one consensus guideline.[72,73] For patients with contraindications to chemoprophylaxis or VTE despite treatment, retrievable vena cava filter placement may be appropriate. However, outside

these unique situations, studies suggest empiric filter placement offers no benefit over routine mechanical/chemoprophylaxis for acute SCI.[74–76]

SUMMARY

The impact of a SCI in any trauma patient can range from a minor nuisance to devastating paralysis, and unfortunately, the full spectrum of these injuries is frequently seen after trauma. Although much of the damage is done at the time of presentation and irreversible immediately, adherence to comprehensive supportive care aimed at treating the injury and preventing secondary injury may make a significant difference in the patient's ultimate functional outcome. Every physician should be able to perform a quick but thorough neurologic examination and understand the implications of significant examination findings such as spinal shock.

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