

# Emergency Neurological Life Support: Traumatic Spine Injury

Deborah M. Stein<sup>1</sup> · William A. Knight IV<sup>2</sup>

Published online: 14 September 2017  
© Neurocritical Care Society 2017

**Abstract** Traumatic spine injuries (TSIs) carry significantly high risks of morbidity, mortality, and exorbitant health care costs from associated medical needs following injury. For these reasons, TSI was chosen as an ENLS protocol. This article offers a comprehensive review on the management of spinal column injuries using the best available evidence. Though the review focuses primarily on cervical spinal column injuries, thoracolumbar injuries are briefly discussed as well. The initial emergency department (ED) clinical evaluation of possible spinal fractures and cord injuries, along with the definitive early management of confirmed injuries, are also covered.

**Keywords** Spinal cord injury · Traumatic spine injury · Neurogenic shock · NEXUS · Canadian c-spine rules

## Introduction

It is estimated that the annual incidence of traumatic spinal injury (TSI) in the United States is approximately 40 per million of population, which equates to 12,000 new cases per year [1]. Mechanisms of spinal cord injuries are, in order of frequency:

- Motor vehicle collisions (42%)
- Falls (27%)
- Violence-related acts (15%)
- Sports injuries (8%)
- Other causes (9%) [1]

In over 50% of patients, injuries to the spine are isolated [2], while nearly 25% have concomitant brain, chest, and/or major extremity injuries [3]. Though classically thought to be a disease of young males, recent epidemiological studies on patients with TSI depict a bimodal distribution [4]. The first peak occurs in adolescents and young adults, as expected. However, the second peak occurs in the elderly population (age > 65 years) [4].

The life expectancy for a patient who sustains an TSI is significantly lower than that for the general population [1]. Average lifetime costs for a patient with TSI range from almost \$1,000,000 for a 50 year-old with an incomplete injury at any level to \$4,400,000 for a patient 25 years old with high tetraplegia [5].

Injuries to the spine tend to occur at areas of maximal mobility. Cervical TSIs account for over 50% of traumatic TSIs and are associated with much higher short- and long-term morbidity than injuries affecting the cord at the thoracic or lumbar level [5–7]. The most frequent injuries are incomplete tetraplegia (31%) followed by complete paraplegia (25%), complete tetraplegia (20%), and incomplete paraplegia (19%) [8].

## Diagnosis

Traditional teaching in evaluating a blunt trauma victim is that medical personnel must assume the patient has a spinal column injury until proven otherwise. Recently, a number

---

✉ Deborah M. Stein  
dstein@umm.edu

William A. Knight IV  
knightwa@ucmail.uc.edu; William.Knight@uc.edu

<sup>1</sup> University of Maryland School of Medicine, R Adams Cowley Shock Trauma Center, University of Maryland Medical Center, Baltimore, MD, USA

<sup>2</sup> Departments of Emergency Medicine and Neurosurgery, University of Cincinnati, Cincinnati, OH, USA

of organizations have suggested a change in the term “spinal immobilization” to “spinal motion restriction” and have suggested that spinal motion restriction, including the use of cervical collars and backboards should not be used in patients at low risk of spinal column injury [9–11]. The change in terminology was proposed with realization that it is very challenging to accomplish true spinal immobilization, without any movement of the spine during the nursing and medical evaluation of a patient that sustained trauma. Spinal motion restriction reflects the true approach to patients with suspected TSI. Spinal motion restriction should be considered for patients with plausible blunt mechanism of injury and any of the following:

- Altered level of consciousness or clinical intoxication
- Focal mid-line spine/bony pain and/or tenderness
- Focal neurologic signs and/or symptoms (e.g., numbness and/or motor weakness)
- Anatomic deformity or step-off of the spine
- Distracting non-spine traumatic injury (i.e. concomitant orthopedic or intra-abdominal injuries, etc.).

In a patient with any of the above risk factors, the spinal column should be restricted until an unstable injury can be excluded. In the prehospital setting, patients with risk factors for TSI are typically fitted with a cervical collar to provide cervical spinal column movement restriction, and patients are subsequently transferred to the hospital using spinal protection techniques. Spinal protection techniques may include the use of a cervical collar and/or backboard to assist with transfer or transportation. Patients should be maintained in a supine position, using log-roll technique to assist with movement when necessary. If necessary, the head of bed may be elevated utilizing reverse Trendelenberg positioning via lowering the feet of the patient, or placing material under a backboard—but keeping the patient in a strict supine position. If the patient is intoxicated and uncooperative with medical evaluation, chemical sedation or physical restraints may be indicated to assure proper protection of the spinal column and, more importantly, the spinal cord.

Once in the emergency department (ED), the immediate evaluation of a patient with a suspected cervical spinal injury is no different from any other trauma patient. The ABCs—airway, breathing, and circulation—take utmost priority. Generally, the diagnosis and treatment of the majority of spine injuries can be deferred to address other life-threatening injuries, such as hemorrhage or traumatic brain injury, as long as spinal protection is maintained. Clinicians should perform their primary survey; assessing the patient’s ABCs and disability. Lastly the physician should fully expose the patient looking for signs of injury.

During the disability portion of the primary survey, clinicians should quickly perform a basic neurologic

assessment. In trauma patients, this can be abbreviated to the patient’s Glasgow Coma Scale (GCS), pupil size and reactivity, and ability to move all four extremities. If the patient is intubated before these three items can be assessed, it becomes more difficult to assess prognosis.

After the primary survey is conducted to assess for potential life-threatening injuries, the secondary survey should be completed. The secondary survey entails a complete head-to-toe evaluation, including a more thorough history of present illness (if possible to obtain). In the suspected spinal injury patient, the entire spinal column and paravertebral musculature should be examined for deformity and palpated in a search for areas of focal tenderness. Vertebral fractures or subluxations may cause step-offs appreciated via palpation of the spinal column or areas of focal tenderness along the midline of the back/neck. The presence of priapism in male patients should always prompt further investigation of TSI. As during the primary survey, spinal precautions should be maintained while evaluating the patient. When assessing the cervical spine, it may be safer for the a portion of the rigid cervical collar to remain on the patient, keeping the head and neck stabilized while a clinician slips his or her hand behind the neck to assess the spinal column.

If transported on a backboard, the patient should be removed as soon as possible, ideally at the conclusion of the primary or secondary survey. Leaving a patient on the backboard can quickly lead to complications and additional pain [12]. Pressure ulcers or deep tissue injuries can develop when the pressure applied to the skin is greater than the diastolic blood pressure. Studies have shown that skin breakdown can occur in as quickly as within the first hour [12]. Tissue injury is more likely in elderly patients, obese patients, those who are on harder surfaces, and those who have suffered hypotension. Pressure ulcers and deep tissues injuries have been associated with higher mortality rates, the need for costly medical treatments, and longer hospital stays. In addition to injuries related to the backboard, increased pain complaints from lying on a hard board can result in unnecessary imaging, along with elevated cost and additional radiation exposure risk.

### Who to Image

To avoid unnecessary radiation exposure, patients with low or moderate pre-test probability of cervical spinal injury should undergo evaluation with a clinical decision rule before imaging. Both the NEXUS criteria [13] and the Canadian C-Spine Rules (CCR) [14, 15] are widely used within clinical practice in the evaluation of patients with suspected cervical spine injuries.

## **NEXUS (National Emergency X-Radiography Utilization Study) Low-Risk Criteria (NLC)**

In the NEXUS study, a clinical clearance protocol consisting of five criteria was validated with 100% sensitivity for the exclusion of cervical spinal injury [13]. The first criterion requires the practitioner to identify signs of intoxication in the patient. In the original study, this included even the detection of the smell of alcohol on a patient. The second criterion requires the practitioner to assess for the presence of focal neurologic deficits. The third criterion is the identification of painful distracting injuries. A distracting injury has no specific definition in the NEXUS study, but examples in the study that prevented clinical clearance were:

- Long bone fractures
- Large lacerations
- De-gloving or crush injuries
- Large burn(s)
- Visceral injuries needing surgical consultation
- Any injuries producing acute functional impairment [15].

With the fourth criterion, the practitioner should assess whether the patient has a normal level of alertness. Specifically, there should be no delay or inappropriate response to external stimuli by the patient. Lastly, to assess the fifth criterion—presence of posterior midline tenderness to palpation—the physician should unhook the velcro strap of the cervical collar and, with the anterior collar still in place, push on each vertebra, monitoring the patient for a response to pain. Using the NEXUS criteria, if no painful response is elicited, and the patient has met all prior criteria, the C-collar can be removed and C-spine imaging is not required.

## **Canadian C-Spine Rules (CCR)**

The CCR does not preclude clinical clearance solely due to posterior neck tenderness [16]. It includes both high-risk and low-risk criteria that allow clearance in patients 18–65 years old (see <http://www.mdcalc.com/canadian-c-spine-rule/>). Although it is more complicated, the greater specificity of the CCR may allow additional patients to be cleared when compared to the NEXUS criteria [16]. The presence of posterior neck tenderness may be one of the deciding points for which rule to choose. If the patient has posterior tenderness, NEXUS will not be usable, but the patient may still avoid imaging with the CCR.

In the CCR, the final stage of clearance is to have the patient rotate his or her head 45° to the left and right. The inability of the patient to perform this maneuver is an indication for further imaging. Though this stage was not a

reported part of the NEXUS criteria, it is still recommended as an appropriate final step in clearance. During this portion of the evaluation, the clinician should remember that minimal pain during active range of motion may be experienced by the patient. However, if the action proves too painful to complete, ligamentous injury is a possibility; therefore, the C-collar should be left in place and advanced imaging pursued.

## **Imaging**

Historically, a 3-view cervical spine radiograph series was the standard initial evaluation for cervical spine injury. However, if imaging is deemed appropriate by the clinician, The Eastern Association for the Surgery of Trauma (EAST) and the American College of Radiology have recommended that computed tomography (CT) with multiplanar reconstruction should be the initial imaging modality [17–19]. If plain radiographs are still used in suspected cervical spine injuries, they are only appropriate in patients who are risk-stratified to low pre-test probability.

If imaging is negative (radiograph or CT scan), the clinician should re-attempt to clinically clear the patient from the collar. If the patient still has persistent midline tenderness at the time of collar clearance, the collar should be left in place. If there is not any significant midline tenderness, the patient should be asked to range left and right 45° as mentioned above. If the patient is unable to range, the collar should be replaced. At this point, institutional protocol should dictate further imaging, consultation, or discharge in a cervical collar combined with appropriate region-specific follow-up (primary care physician, trauma surgeon, spine surgeon, etc.).

Clinical judgment must be used for the clearance of possible thoracolumbar (TL) spinal column injuries, as there are currently no validated guidelines. Focal tenderness over the thoracolumbar spine, neurologic deficit, and high-energy mechanism are risk factors that have been identified to be associated with TL spinal column injuries [19]. If a patient has focal bony spine tenderness after trauma, it is generally recommended to further pursue dedicated imaging.

Additionally, in patients with one vertebral column fracture, the presence of a second non-adjointing fracture has been estimated to have an incidence of up to 15% [20]. As a result, when one vertebral fracture has been identified, it is recommended that the entire spinal column undergo imaging to assess for concomitant fracture.

## Motor and Sensory Exams

If any neurological abnormalities are discovered during initial screening, a detailed neurologic examination of motor and sensory function at all spinal levels should be performed and the patient should be maintained with spinal motion restriction.

The neurological examination in any patient with suspected TSI should focus on the motor and sensory exams, as well as rectal tone and perineal sensation findings. If the patient has abnormality in any of these areas, the lesion should be localized to the highest spinal level where dysfunction is noted. As a general guide, some of the commonly referred to motor and sensory levels are:

### Motor

- C4—deltoid
- C5—biceps
- C6—wrist extensors
- C7—triceps
- T1—finger abduction
- L2—hip flexors
- L3—knee flexion
- L4—ankle dorsiflexion
- S1—plantar flexion

### Sensory

- C4—deltoid
- T4—nipple
- T10—umbilicus

The levels above refer to the respective myotomes and dermatomes for these regions of dysfunction. A rectal exam is of utmost importance in any patient with a suspected TSI, as decreased rectal tone may be the only sign of an TSI and helps differentiate complete from incomplete lesions, which is of vital importance in prognostication for recovery of function.

## American Spinal Injury Association (ASIA) Scale

The full examination recommended by the American Spinal Injury Association (ASIA) (<http://www.asia-spinalinjury.org>) includes a detailed motor and sensory examination. It is the preferred evaluation tool as recommended by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons [21].

ASIA also defines a five-element scale, the ASIA Impairment Scale (AIS), that is prognostic of neurologic recovery:

A. Complete—No motor or sensory function in the lowest sacral segment.

B. Incomplete—Sensory but not motor function is preserved in the lowest sacral segment.

C. Incomplete—Less than 1/2 of the key muscles below the neurological spinal level have grade 3 or better strength.

D. Incomplete—At least 1/2 of the key muscles below the neurological level have grade 3 or better strength.

E. Normal—Sensory and motor functions are normal.

Complete injuries, defined by absence of sensory or motor function below a spinal level, have a worse prognosis for functional recovery. One caveat is that in the setting of significant spinal shock, absence of sensation or function may be a manifestation of the spinal shock itself as opposed to the primary injury. Once the spinal shock resolves, incomplete injuries may become unmasked [22]. Incomplete injuries have a much better prognosis for functional recovery.

## Syndromes

A number of discrete neurologic syndromes have also been described. If present, these syndromes help indicate the extent and nature of the injury:

- *Anterior Cord Syndrome* Described as a loss of pain/temperature and motor function with preservation of light touch. It is caused by injury to the anterior spinal cord, commonly from contusion or occlusion of the anterior spinal artery. Anterior Cord Syndrome is associated with axial compression causing burst fractures of the spinal column with fragment retropulsion.
- *Central Cord Syndrome* Described as a loss of cervical motor function with relative sparing of lower extremity strength. This is most often due to hyperextension injury, commonly seen in elderly patients with cervical stenosis [23, 24]. It is usually not associated with a fracture, but rather with a buckling of the ligamentum flavum that contuses the cord, causing hemorrhage within the center of the cord. The amount of damage to the laterally located corticospinal tracts is variable and determines the amount of lower extremity weakness.
- *Brown-Séquard Syndrome* Described as a hemiplegia with loss of ipsilateral light touch and contralateral pain/temperature sensation. This is due to traumatic hemisection of the cord. It is most frequently seen with penetrating cord injury, often from missiles or knife wounds, or a lateral mass fracture of the spine.

## Management

### Initial Management in Confirmed or Suspected TSI

Once a fracture has been diagnosed, the patient should be maintained with spinal motion restriction during all treatments. As opposed to patients with spinal column injuries without deficit or patients with TL injuries, patients with cervical TSIs often have life threatening issues that are a direct consequence of their spine injury. These issues require emergent attention and take priority in the acute management of these patients.

### Airway

Patients with cervical TSI can be at exceptionally high risk of airway compromise due to a number of factors. Airway and soft-tissue edema or hematomas from direct neck trauma and local bleeding can contribute to airway compromise. In patients with high cervical TSI (C3–C5), loss of diaphragmatic innervation, as well as loss of chest and abdominal wall strength, contribute significantly to a patient's inability to maintain adequate oxygenation and ventilation. Patients with high (above C3) complete TSI will almost invariably suffer a respiratory arrest within minutes of initial injury and, if not intubated by prehospital providers, typically present in cardiac arrest.

As a general recommendation, all patients with a complete cervical TSI above C5 should be intubated as soon as possible [25, 21]. Patients with incomplete or lower injuries will have a high degree of variability in their ability to maintain adequate oxygenation and ventilation. General parameters for urgent intubation include:

- Obvious respiratory distress
- Dyspnea
- Complaint of inability to “catch my breath”
- Inability to hold breath for 12 s [21]
  - (Have patient count as high as they can. Less than 20 is concerning for respiratory compromise)
- Vital capacity < 10 mL/kg or decreasing vital capacity
- Appearance of “belly breathing” or “quad breathing”
  - abdomen protrudes out sharply with inspiration
- pCO<sub>2</sub> > 20 mmHg above baseline.

When in doubt, it is better to electively intubate a patient with a cervical TSI than to wait until it must be performed emergently. Patients will typically develop worsening of their primary injury shortly after admission due to cord edema and progressive loss of muscle strength; therefore, vigilance in monitoring these patients for worsening of

respiratory status is essential [25]. Providers should consider monitoring stable appearing patients with end-tidal CO<sub>2</sub> for an objective measurement of their ventilatory adequacy. Table 1 provides some absolute and relative indications for urgent intubation in patients with an acute cervical TSI.

Generally, patients with cervical TSI who require non-urgent intubation should be intubated by an experienced provider using an awake fiberoptic approach. This will minimize movement of the cervical spine and the risk of exacerbation of spinal cord injury in the setting of ligamentous or fracture instability. It will also allow for a neurological examination following intubation to document any changes. Patients who require urgent or emergent intubation should be intubated using rapid sequence intubation (RSI) [26]. Providers should strongly consider video laryngoscopy and/or airway adjuncts that help minimize cervical spine mobility, while optimizing visualization of the vocal cords. The cervical collar should be removed with in-line stabilization carefully maintained, and extreme care must be taken to avoid hyper-extending the neck to minimize the risk of worsening the injury (Tables 2, 3).

No particular RSI medication regimen is recommended, but it should be considered that many of these patients may already be vasodilated from loss of sympathetic tone. Therefore, medications that further diminish the catecholamine surge may result in exacerbation of hypotension and bradycardia [27–29]. Tracheal or laryngeal manipulation can also stimulate a bradycardic response in these patients, as can any degree of hypoxia [30, 31]. Atropine should always be immediately available when manipulating the airway of a patient with an acute cervical TSI. Though traditionally avoided in patients with TSI due to the risk of hyperkalemia from depolarization [31], succinylcholine is safe to use in the first 48 h after injury, prior to up-regulation of acetylcholine receptors [32].

### Breathing

Patients with cervical TSI are at high risk of inadequate oxygenation and ventilation due to a combination of factors [25]. High cervical TSIs result in loss of diaphragmatic function and can cause apnea. The chest wall and abdominal musculature that are so vital for effective ventilation are often severely compromised, even in patients with incomplete injuries. This results in hypoventilation and a significant inability to generate an effective cough to clear secretions. Aspiration, retention of secretions, and the development of atelectasis contribute to further respiratory decompensation. Providers should consider using end-tidal CO<sub>2</sub> monitoring while determining the need for intubation.

Concomitant injuries such as pulmonary contusions and pneumothoraces are often found in the polytrauma patient.

**Table 1** Indications for intubation in patients with traumatic cervical spine injury

Absolute indications
Complete SCI above C5 level
Respiratory distress
Hypoxemia despite adequate attempts at oxygenation
Severe respiratory acidosis
Relative indications
Complaint of shortness of breath
Development of “quad breathing” Paradoxical abdominal work of breathing
Vital capacity (VC) of <10 ml/kg or decreasing VC
Consideration should be given
Need to “travel” remote from ED (MRI, transfer to another facility)

**Table 2** Checklist

Traumatic Spine Injury checklist for the first hour  
Checklist

- Spine immobilization with cervical collar and maintain spine precautions with “flat/bedrest” until seen by spine specialist
  - Keep SBP >90 mmHg with IV fluids and vasoactive medications as needed
  - Administer supplemental O<sub>2</sub> if SpO<sub>2</sub> <92%
  - Consider early intubation for failure of ventilation per Table 1
  - Rule out other causes of hypotension such as hemorrhage, pneumothorax, cardiac dysfunction
- Do not assume neurogenic shock

**Table 3** Communication

Traumatic Spine Injury communication regarding assessment and transfer/referral communication

- Age
- Mechanism of injury
- Vital signs
- Basic neurologic exam including any sensory deficit, motor deficit, “level” of deficit, and rectal tone and sensation
- Additional traumatic injuries
- Interventions and medications administered including IV fluids and blood products administered and any vasoactive infusions with dose
- CT scan including location of fractures, displacement of fragments, dislocation and/or MRI scan including spinal cord signal change and ligamentous injury noted

Up to 65% of patients with cervical TSI will have evidence of respiratory dysfunction on admission to the Intensive Care Unit (ICU) [33]. Supplemental oxygen should be supplied to all patients with cervical TSI to maintain an arterial saturation >92%, as hypoxemia is extremely detrimental to patients with neurological injury. Appropriate pre-oxygenation should be employed prior to intubation. Hypoxemia can cause severe bradycardia in patients with high cervical TSIs due to unopposed vagal stimulation [30, 31]. Non-invasive methods of ventilation should be used with caution in this patient population, as the inability to cough and clear secretions may lead to an increased risk of aspiration.

### Circulation

Patients with TSI above the T4 level are at high risk of the development of neurogenic shock [25]. The patient suffers an interruption of the sympathetic chain, resulting in unopposed vagal tone. This leads to a distributive shock with hypotension and bradycardia, though variable heart rates have also been described [34].

Patients with neurogenic shock are generally hypotensive with warm, dry skin, as opposed to patients with hypovolemic shock from hemorrhage. This is due to the loss of sympathetic tone, resulting in an inability to redirect blood flow from the periphery to the core circulation. However, in the patient with multiple injuries, other causes of hypotension, such as hemorrhagic shock or tension

pneumothorax, can be present. These causes must be identified and immediately addressed.

Bradycardia is a characteristic finding of neurogenic shock and may help to differentiate from other forms of shock. Care should be taken to avoid assuming that a patient has neurogenic shock because of a lack of tachycardia. Young healthy patients, elderly patients, and patients on pre-injury beta-blockers will often not manifest tachycardia in the setting of hemorrhage.

As a general rule, the higher and more complete the injury, the more severe and refractory the neurogenic shock [35]. These signs can be expected to last from 1 to 3 weeks. Patients may develop manifestations of neurogenic shock within hours to days following injury due to progressive edema and ischemia of the spinal cord, resulting in ascension of their injury [36, 37, 38]. Of note, the term “spinal shock” is not related to hemodynamics, but rather refers to the loss of spinal reflexes below the level of injury [38].

First line treatment of neurogenic shock is always fluid resuscitation to ensure euvoolemia [32]. The loss of sympathetic tone leads to vasodilation and the need for an increase in the circulating blood volume [39]. Once euvoolemia is established, second line therapy includes vasopressors and/or inotropes [40] (See also the ENLS Pharmacology manuscript). There is currently no established recommended single agent, though potential agents include:

- **Norepinephrine** Has both alpha and some beta activity, thereby improving both peripheral vasoconstriction and inotropy, contributing to both blood pressure and bradycardia, and is most likely the preferred agent.
- **Phenylephrine** A pure alpha-1 agonist that is very commonly used, and easily titrated. Phenylephrine lacks beta activity, does not treat bradycardia and may actually worsen the heart rate through reflexive mechanisms [32]. This is best used in patients with lesions below T 5 in whom bradycardia is less of a concern.
- **Dopamine** Also frequently used, but high doses (>10 mcg/kg/min) are needed to obtain the alpha vasoconstrictor effect. It does have significant beta effects at lower doses. If lower doses are used, it may lead to inadvertent diuresis, exacerbating relative hypovolemia. Dopamine is associated with increased arrhythmic events in all patients, and increased mortality in patients with cardiogenic shock [41]
- **Epinephrine** An alpha and beta-agonist that causes vasoconstriction and increased cardiac output. The high doses that may be required can lead to inadvertent mucosal ischemia. In most centers, epinephrine is rarely used or needed.
- **Dobutamine** Can be useful, as it is a pure beta agonist and inotrope that can affect bradycardia, and may be helpful for treatment of hypotension if the loss of

sympathetic tone causes cardiac dysfunction. Caution should be taken in patients who are not adequately volume loaded, as it may cause hypotension.

All inotropes and vasopressors may be administered through a peripheral IV in an emergency until definitive central access is established.

The American Association of Neurological Surgeons (AANS) and the Congress of Neurological Surgeons' (CNS) Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries recommend maintenance of mean arterial blood pressure (MAP) at 85–90 mmHg for the first 7 days following acute TSI to improve spinal cord perfusion [42]. This is based on uncontrolled studies that demonstrated benefit in patients who were maintained with a MAP of 85 for 7 days following injury [43, 22]. Providers should maintain caution when inducing elevated blood pressure in patients with concomitant injuries, especially traumatic brain injuries. An overall risk/benefit analysis should be applied to each individual patient prior to reflexively starting or protocolizing an elevated MAP goal in a patient with a TSI.

#### *Immobilization of Confirmed Injuries*

Confirmed cervical spinal column fractures should be kept stabilized in a cervical collar with “log-roll” precautions off the backboard as discussed above until definitive management can be arranged. The initial goal of treatment should be to prevent further injury caused by spine motion with resultant worsening of neurologic outcome. An additional goal would be to minimize skin breakdown while maintaining spinal stabilization.

Studies have demonstrated that Philadelphia™ collars and Miami J™ collars are more effective than standard emergency medical services (EMS) collars in reducing cervical spinal column range of motion [44]. Miami J™ collars have also been shown to apply the least amount of pressure to the facial tissues of the patient compared to other cervical immobilizing collars [44]. Miami J™ collars are indicated in stable cervical spinal column injuries from C2 to C5. A thoracic extension can be added if immobilization is needed for a stable injury from C6 to T2. It should be noted that there is not a cervical collar that will prevent a determined or delirious patient from moving his or her head, potentially worsening injury. Agitated patients may require aggressive pain control and sedation to minimize mobility of the cervical spine.

Patients with spinal column injuries have historically been moved only with “log-roll” precautions once in the hospital, and this remains the standard of care in many centers. However, the method has been called into question by some practitioners given that significant movement of

the spinal column can still occur. The High Arm In Endangered Spine (HAINES) method has been recommended by some clinicians given that it may minimize movement of the spine compared to the traditional log-roll method [11, 13]. With the patient lying supine, the knees are bent, and one arm is abducted to 180° with the other arm across the patient's chest. With a clinician providing inline stabilization while on the side of the patient with the arm across the chest, the patient can be gently rolled to his or her side, and a transfer device can be placed underneath the patient.

### *Definitive Treatment*

The mainstay of treatment for TSIs is decompression of the spinal cord to minimize additional injury from cord compression; surgical stabilization of unstable ligamentous and bony injury; and minimizing the effect of secondary complications, such as venous thromboembolic disease, pressure ulcer prevention, respiratory failure, and infections. Early consideration should be given to placement of indwelling urinary catheters, both to monitor volume status and prevent urinary retention [25, 45]. Once an indwelling urinary catheter is appropriate for removal, the care team should initially perform frequent bladder urine volume assessments, and straight catheter for urine greater than 400 cc to prevent bladder distension and overflow incontinence. Additionally, stress ulcer prophylaxis should be initiated early following injury, due to an increased risk of gastrointestinal bleeding in patients with cervical TSI [46–48]. There are few therapeutic options for the injured spine itself. Though there has been extensive research in the field, no neuroprotective therapy has been definitively proven effective in improving outcome following traumatic spinal cord injury [32].

### *Steroids*

The use of steroids following TSI was based on experimental work in animal models that suggested methylprednisolone has neuroprotective effects through an anti-inflammatory mechanism [49, 50]. This led to the National Acute Spinal Cord Injury Studies (NASCIS) trials. NASCIS II concluded there was efficacy of high dose methylprednisolone in patients who had received the drug within 8 h after injury [51, 52]. This was based on patient's experiencing neurologic improvement in 1–2 sensory levels from their original injury.

As a result, this regimen quickly became the standard of care. However, there has been extensive debate and discussion about the validity of the results, as well as an inability to confirm the results in additional trials [53–59]. Moreover, extensive concerns have been raised about

increased complications, such as pneumonia and gastrointestinal bleeding in patients treated with steroids following acute cervical TSI [60, 61, 42].

Based on these circumstances, the most recent version of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons' Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries state: "Administration of methylprednisolone (MP) for the treatment of acute spinal cord injury (SCI) is not recommended. Clinicians considering MP therapy should bear in mind that the drug is not Food and Drug Administration (FDA) approved for this application. There is no Class I or Class II medical evidence supporting the clinical benefit of MP in the treatment of acute TSI. Scattered reports of Class III evidence claim inconsistent effects likely related to random chance or selection bias. However, Class I, II, and III evidence exists that high-dose steroids are associated with harmful side effects including death." [62] An additional 15 medical societies have also stated that steroids should not be considered the standard of care after spinal cord injury.

### **Algorithm Section**

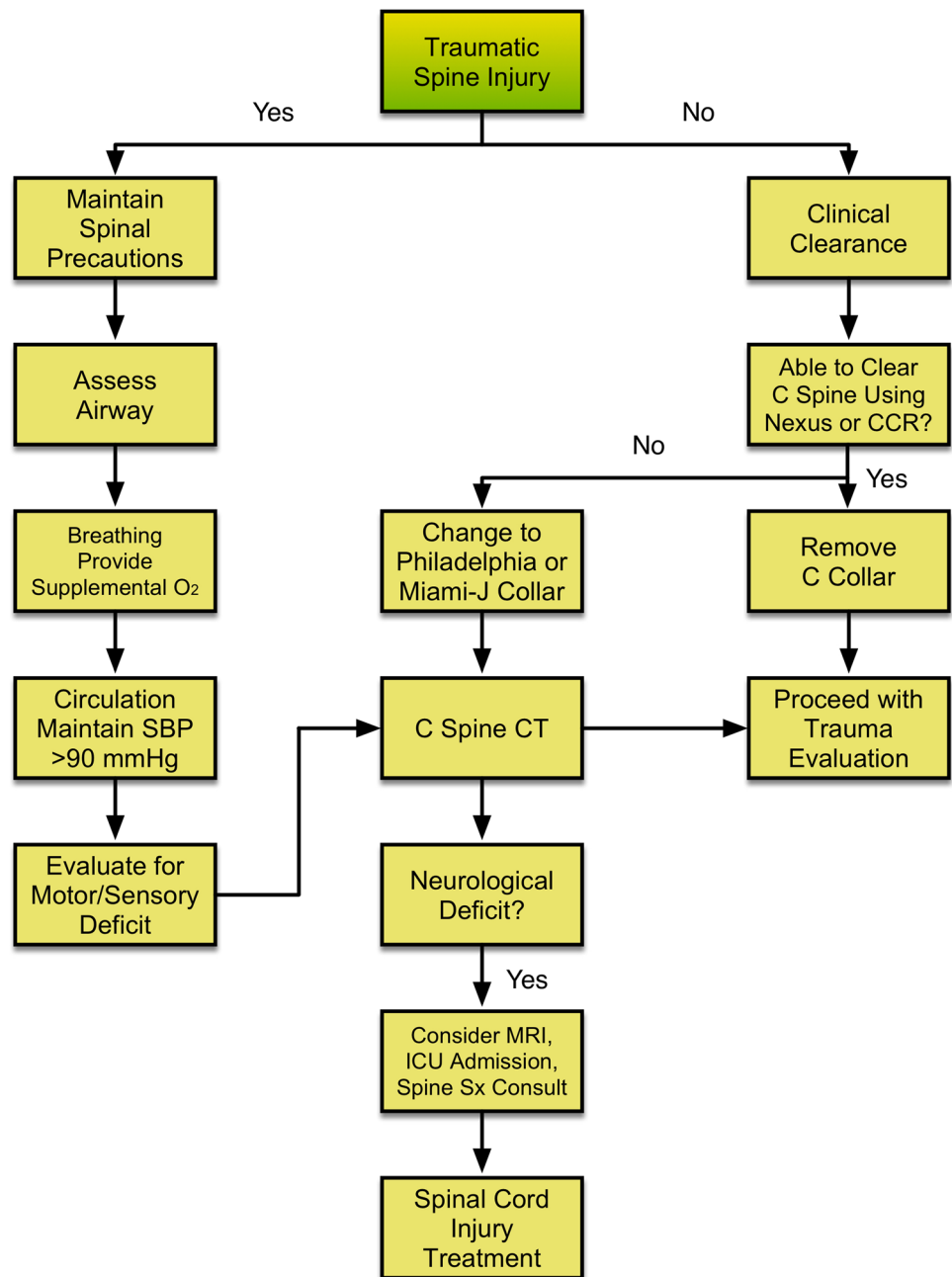
The updated ENLS traumatic spinal cord injury algorithm is below. Figure 1 Given the necessary attention to airway, breathing and circulation, as well as spinal motion restriction, the detailed steps of the algorithm have been best described in the management section of this manuscript. A patient with a suspected spinal cord injury should be maintained in strict spinal motion restriction throughout their evaluation. Immediate attention to adequate airway protection, ventilation/oxygenation adequacy and shock are paramount to the management of these patients. A thorough trauma secondary survey to evaluate for concomitant traumatic injuries is necessary. Finally, clearance from spinal motion restriction as soon as medically feasible is recommended to allow for early mobility, as well as removal of unnecessary lines, indwelling tubes and devices.

### **Unique Pediatric Considerations**

Although rare, TSI is a devastating condition in children. The vertebral column is more flexible in children 9 years old and younger, making the spinal cord more susceptible to injury, including an increased risk of atlanto-axial dislocation [63–67]. In infants, TSI may contribute to morbidity and mortality in victims of non-accidental trauma [67]. Young pediatric patients are also at risk of spinal cord injury without radiographic abnormality



**Fig. 1** ENLS traumatic spinal cord injury protocol



(SCIWORA), a condition that should always be considered in children with neurologic changes, concern for TSI, or with an unreliable exam, in the absence of abnormalities on plain films or CT scan imaging [68]. The risk of TSI is higher in children with Down syndrome in whom ligaments are more lax and atlantoaxial instability may be present in approximately 20% of patients. In children with TSI whose mechanism involves high-energy thoracic trauma, injury to the carotid or vertebral arteries should be considered. Angiography should be pursued in children with unexplained coma, ischemic changes on brain imaging, or

clinical signs of stroke. Skull base fractures or several facial trauma are additional risk factors.

As is the case in adult TSI, there are no established neuroprotective treatments for pediatric SCI. The initial approach includes surgical decompression in selective cases, and avoidance of secondary insults that may aggravate the initial injury (i.e. hypoxia and hypotension). While the optimal blood pressure range for children with SCI has not been established, systolic blood pressure above the 5th percentile for age should be maintained ( $SBP = 70 \text{ mmHg} + \text{age in years} \times 2$ ). Spinal motion restriction should be maintained in pediatric patients in

whom the clinicians have a high suspicion for TSI. Children can be more challenging to maintain in a restricted position, and patient selection is important. Special attention to positioning is important, as the large head size predisposes young children to flexion of the neck. Careful selection if an appropriate sized neck collar is also important to prevent skin lesions, inadvertent neck movement, or obstruction of the child's cerebral venous circulation. Clinicians should approach pediatric patients with the same algorithm as adult patients, with priority given to the airway, breathing and circulation. Shock in a child can be particularly confounding, given the higher physiologic reserves in pediatric patients.

The main systemic complications of TSI in children include respiratory failure, hemodynamic instability, autonomic dysreflexia, pain, venous thromboembolism, psychological distress, neurogenic bladder and bowel, hypercalcemia and skin pressure ulcers. Delayed stabilization even in cases of complete SCI may be beneficial to facilitate early mobilization and maintain spinal alignment.

## References

- Singh A, Tetreault L, Kalsi-Ryan S, Nouri A, Fehlings MG. Global prevalence and incidence of traumatic spinal cord injury. *Clin Epidemiol*. 2014;6:309–31.
- Lindsey R, Gugala Z, Pneumatics S. *Injury to the vertebrae and spinal cord*. 6th ed. New York: McGraw-Hill; 2011.
- Saboe LA, Reid DC, Davis LA, Warren SA, Grace MG. Spine trauma and associated injuries. *J Trauma*. 1991;31:43–8.
- Jabbour P, Fehlings M, Vaccaro AR, Harrop JS. Traumatic spine injuries in the geriatric population. *Neurosurg Focus*. 2008;25:E16.
- DeVivo MJ, Krause JS, Lammertse DP. Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil*. 1999;80:1411–9.
- DeVivo MJ, Ivie CS 3rd. Life expectancy of ventilator-dependent persons with spinal cord injuries. *Chest*. 1995;108:226–32.
- McKinley WO, Jackson AB, Cardenas DD, DeVivo MJ. Long-term medical complications after traumatic spinal cord injury: a regional model systems analysis. *Arch Phys Med Rehabil*. 1999;80:1402–10.
- Annual Report for Spinal Cord Injury Model Systems. 2010. [https://www.nscisc.uab.edu/public\\_content/annual\\_stat\\_report.aspx](https://www.nscisc.uab.edu/public_content/annual_stat_report.aspx). Accessed 2 Feb 2012.
- Sundström T, Asbjørnsen H, Habiba S, et al. Prehospital use of cervical collars in trauma patients: a critical review. *J Neurotrauma*. 2014;31(6):531–40.
- Kang DG, Lehman RA Jr. Spine immobilization: prehospitalization to final destination. *J Surg Orthop Adv*. 2011;20(1):2–7.
- Sporer K. Why we need to rethink C-spine immobilization. *EMS World*. 2012;41(11):74–6.
- Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. *Ostomy Wound Manag*. 2008;54(26–8):30–5.
- Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. *N Engl J Med*. 2000;343:94–9.
- Bandiera G, Stiell IG, Wells GA, et al. The Canadian C-spine rule performs better than unstructured physician judgment. *Ann Emerg Med*. 2003;42:395–402.
- Ullrich A, Hendey GW, Geiderman J, Shaw SG, Hoffman J, Mower WR. Distracting painful injuries associated with cervical spinal injuries in blunt trauma. *Acad Emerg Med*. 2001;8:25–9.
- Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. *N Engl J Med*. 2003;349:2510–8.
- Como JJ, Diaz JJ, Dunham CM, et al. Practice management guidelines for identification of cervical spine injuries following trauma: update from the eastern association for the surgery of trauma practice management guidelines committee. *J Trauma*. 2009;67:651–9.
- Suspected Spinal Trauma. 2009. [http://www.acr.org/SecondaryMainMenuCategories/quality\\_safety/app\\_criteria/pdf/ExpertPanelonMusculoskeletalImaging/SuspectedCervicalSpineTraumaDoc22.aspx](http://www.acr.org/SecondaryMainMenuCategories/quality_safety/app_criteria/pdf/ExpertPanelonMusculoskeletalImaging/SuspectedCervicalSpineTraumaDoc22.aspx). Accessed May 2012.
- Frankel HL, Rozycki GS, Ochsner MG, Harviel JD, Champion HR. Indications for obtaining surveillance thoracic and lumbar spine radiographs. *J Trauma*. 1994;37:673–6.
- Holmes JF, Miller PQ, Panacek EA, Lin S, Horne NS, Mower WR. Epidemiology of thoracolumbar spine injury in blunt trauma. *Acad Emerg Med*. 2001;8:866–72.
- Durga P, Sahu BP, Mantha S, Ramachandran G. Development and validation of predictors of respiratory insufficiency and mortality scores: simple bedside additive scores for prediction of ventilation and in-hospital mortality in acute cervical spine injury. *Anesth Analg*. 2010;110:134–40.
- Licina P, Nowitzke AM. Approach and considerations regarding the patient with spinal injury. *Injury*. 2005;36(Suppl 2):B2–12.
- Aarabi B, Alexander M, Mirvis SE, et al. Predictors of outcome in acute traumatic central cord syndrome due to spinal stenosis. *J Neurosurg Spine*. 2011;14:122–30.
- Albert TJ, Levine MJ, Balderston RA, Cotler JM. Gastrointestinal complications in spinal cord injury. *Spine*. 1991;16:S522–5.
- Velmahos GC, Toutouzias K, Chan L, et al. Intubation after cervical spinal cord injury: to be done selectively or routinely? *Am Surg*. 2003;69:891–4.
- Crosby ET. Airway management in adults after cervical spine trauma. *Anesthesiology*. 2006;104:1293–318.
- Yoo KY, Jeong CW, Kim SJ, et al. Altered cardiovascular responses to tracheal intubation in patients with complete spinal cord injury: relation to time course and affected level. *Br J Anaesth*. 2010;105:753–9.
- Pasternak JJ, Lanier WL. Neuroanesthesiology update 2010. *J Neurosurg Anesthesiol*. 2011;23:67–99.
- Yoo KY, Jeong SW, Kim SJ, Ha IH, Lee J. Cardiovascular responses to endotracheal intubation in patients with acute and chronic spinal cord injuries. *Anesth Analg*. 2003;97:1162–7.
- Raw DA, Beattie JK, Hunter JM. Anaesthesia for spinal surgery in adults. *Br J Anaesth*. 2003;91:886–904.
- Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. *Anesthesiology*. 1975;43:89–99.
- Early Acute Management in Adults with Spinal Cord Injury Clinical Practice Guidelines. 2008. [www.pva.org](http://www.pva.org). Accessed May 2012.
- Stein DM, Menaker J, McQuillan K, Handley C, Aarabi B, Scalea TM. Risk factors for organ dysfunction and failure in patients with acute traumatic cervical spinal cord injury. *Neurocrit Care*. 2010;13:29–39.
- Bilello JF, Davis JW, Cunningham MA, Groom TF, Lemaster D, Sue LP. Cervical spinal cord injury and the need for cardiovascular intervention. *Arch Surg*. 2003;138:1127–9.

35. Maiorov DN, Fehlings MG, Krassioukov AV. Relationship between severity of spinal cord injury and abnormalities in neurogenic cardiovascular control in conscious rats. *J Neurotrauma*. 1998;15:365–74.
36. Gondim FA, Lopes AC Jr, Oliveira GR, et al. Cardiovascular control after spinal cord injury. *Curr Vasc Pharmacol*. 2004;2:71–9.
37. Krassioukov A, Claydon VE. The clinical problems in cardiovascular control following spinal cord injury: an overview. *Prog Brain Res*. 2006;152:223–9.
38. Nacimiento W, Noth J. What, if anything, is spinal shock? *Arch Neurol*. 1999;56:1033–5.
39. Levi L, Wolf A, Belzberg H. Hemodynamic parameters in patients with acute cervical cord trauma: description, intervention, and prediction of outcome. *Neurosurgery*. 1993;33:1007–16 **discussion 16–17**.
40. Stevens RD, Bhardwaj A, Kirsch JR, Mirski MA. Critical care and perioperative management in traumatic spinal cord injury. *J Neurosurg Anesthesiol*. 2003;15:215–29.
41. De Backer D, Biston P, Devriendt J, Madl C, Chochrad D, Aldecoa C, Brasseur A, Defrance P, Gottignies P, Vincent JL. SOAP II investigators. Comparison of dopamine and norepinephrine in the treatment of shock. *N Engl J Med*. 2010;362(9):779–89.
42. Ryken T, Hurlbert RJ, Hadley MN, Aarabi B, Dhall SS, Gelb DE, Rozzelle CJ, Theodore N, Walters BC. The acute cardiopulmonary management of patients with cervical spinal cord injuries. *Neurosurgery*. 2013;72(3):84–92 **Supplement**.
43. Vale FL, Burns J, Jackson AB, Hadley MN. Combined medical and surgical treatment after acute spinal cord injury: results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg*. 1997;87:239–46.
44. Tescher AN, Rindfleisch AB, Youdas JW, et al. Range-of-motion restriction and craniofacial tissue-interface pressure from four cervical collars. *J Trauma*. 2007;63:1120–6.
45. Aresco C, Stein D. Cervical spine injuries in the geriatric patient. *Clin Geriatr*. 2010;18(2):30–35.
46. Kiwerski J. Bleeding from the alimentary canal during the management of spinal cord injury patients. *Paraplegia*. 1986;24:92–6.
47. Walters K, Silver JR. Gastrointestinal bleeding in patients with acute spinal injuries. *Int Rehabil Med*. 1986;8:44–7.
48. Braughler JM, Hall ED. Lactate and pyruvate metabolism in injured cat spinal cord before and after a single large intravenous dose of methylprednisolone. *J Neurosurg*. 1983;59:256–61.
49. Hall ED. The neuroprotective pharmacology of methylprednisolone. *J Neurosurg*. 1992;76:13–22.
50. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med*. 1990;322:1405–11.
51. Bracken MB, Shepard MJ, Collins WF Jr, et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data. Results of the second National Acute Spinal Cord Injury Study. *J Neurosurg*. 1992;76:23–31.
52. Hugenholtz H, Cass DE, Dvorak MF, et al. High-dose methylprednisolone for acute closed spinal cord injury—only a treatment option. *Can J Neurol Sci*. 2002;29:227–35.
53. Hugenholtz H. Methylprednisolone for acute spinal cord injury: not a standard of care. *CMAJ*. 2003;168:1145–6.
54. Nesathurai S. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. *J Trauma*. 1998;45:1088–93.
55. Short DJ, El Masry WS, Jones PW. High dose methylprednisolone in the management of acute spinal cord injury—a systematic review from a clinical perspective. *Spinal Cord*. 2000;38:273–86.
56. Coleman WP, Benzel D, Cahill DW, et al. A critical appraisal of the reporting of the National Acute Spinal Cord Injury Studies (II and III) of methylprednisolone in acute spinal cord injury. *J Spinal Disord*. 2000;13:185–99.
57. Ducker TB, Zeidman SM. Spinal cord injury. Role of steroid therapy. *Spine*. 1994;19:2281–7.
58. Hurlbert RJ. Methylprednisolone for acute spinal cord injury: an inappropriate standard of care. *J Neurosurg*. 2000;93:1–7.
59. Matsumoto T, Tamaki T, Kawakami M, Yoshida M, Ando M, Yamada H. Early complications of high-dose methylprednisolone sodium succinate treatment in the follow-up of acute cervical spinal cord injury. *Spine*. 2001;26:426–30.
60. Galandiuk S, Raque G, Appel S, Polk HC Jr. The two-edged sword of large-dose steroids for spinal cord trauma. *Ann Surg*. 1993;218:419–25 **discussion 25–7**.
61. Gerndt SJ, Rodriguez JL, Pawlik JW, et al. Consequences of high-dose steroid therapy for acute spinal cord injury. *J Trauma*. 1997;42:279–84.
62. Hurlbert RJ, Hadley MN, Walters BC, Aarabi B, Dhall SS, Gelb DE, Rozzelle CJ, Ryken TC, Theodore N. Pharmacological therapy for acute spinal cord injury. *Neurosurgery*. 2013;72(3):93–105 (**supplement**).
63. Bailey DK. The normal cervical spine in infants and children. *Radiology*. 1952;59:712–9.
64. Fesmire FM, Luten RC. The pediatric cervical spine: developmental anatomy and clinical aspects. *J Emerg Med*. 1989;7:133–42.
65. Sullivan CR, Bruwer AJ, Harris LE. Hypermobility of the cervical spine in children; a pitfall in the diagnosis of cervical dislocation. *Am J Surg*. 1958;95:636–40.
66. Bohlman HH. Acute fractures and dislocations of the cervical spine. An analysis of three hundred hospitalized patients and review of the literature. *J Bone Joint Surg Am*. 1979;61:1119–42.
67. Hadley MN, Sonntag VK, Rekatte HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery*. 1989;24:536–40.
68. Pang D, Pollack IF. Spinal cord injury without radiographic abnormality in children—the SCIWORA syndrome. *J Trauma*. 1989;29:654–64.