TRAUMA QUALITY PROGRAMS

AMERICAN COLLEGE OF SURGEONS

BEST PRACTICES GUIDELINES

SPINE INJURY

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BEST PRACTICES GUIDELINES
SPINE INJURY

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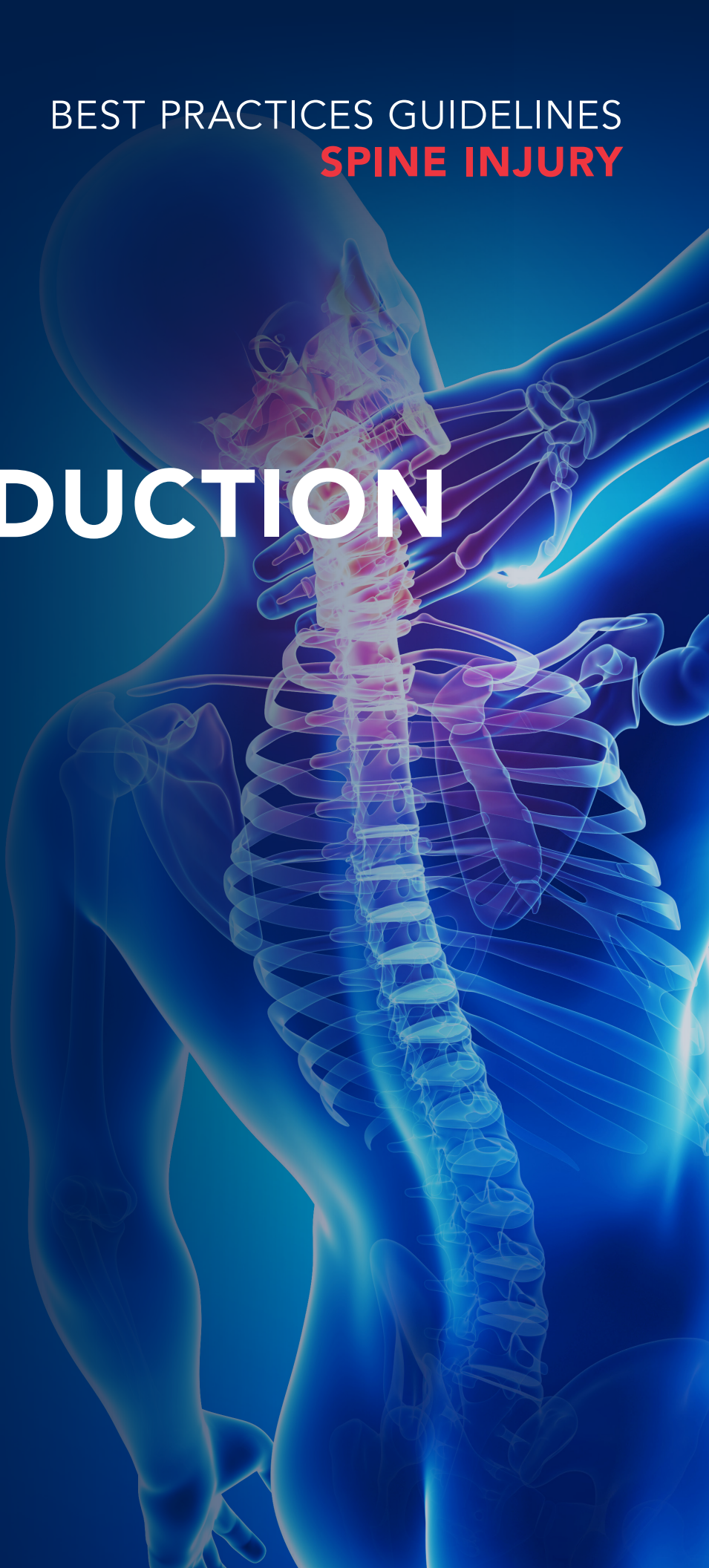
INTRODUCTION



TRAUMA QUALITY PROGRAMS
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SPINE INJURY

INTRODUCTION

Fractures of the spinal column represent a small proportion of all fractures from traumatic injury with an incidence ranging from 4 to 23 percent.[1,2] However, their impact on the individual and the health care system is significant because of their potential for long-term disability, the associated health care consequences, and costs. Despite improvements in the understanding of basic spinal fracture patterns, more reliable classification, and injury severity assessment systems, controversy remains in the management of these injuries.[1] The threat of irreversible neural tissue injury and the presence of multiple traumatic injuries that may include life-threatening abdominal and thoracic injuries leads to complex decision making. Timing of surgical care for spinal injuries depends not only upon early decompression to improve or prevent further neurologic injury, but also on the need to first stabilize the patient's hemodynamics or treat other life-threatening injuries. Operative versus nonoperative spinal column management is not always a clear decision, such as when progressive deformity or secondary neurologic deterioration is unlikely or it is unclear that surgical stabilization will contribute to an improved quality of life. Globally, the aging population is increasing and with it is the incidence of traumatic spinal injuries. The aging spine becomes increasingly vulnerable to injury as it stiffens and becomes osteoporotic. Comorbidities and frailty present management and outcome challenges.

This publication is intended to provide an evidence-based, practical guide for the evaluation and management of an adult patient with a spinal injury, including both spinal column fracture (SCF) and spinal cord injury (SCI). When evidence is poor or absent, best practices are then based upon expert opinion which has been drawn from leaders in the fields of neurosurgery and orthopaedic surgery.

This Best Practices Guideline (BPG) begins with the epidemiology of spinal injury, then reviews pre-hospital spinal motion restriction, cervical collar clearance, and appropriate imaging. It goes on to include details about completing a physical exam for evaluation of spinal cord injury and the classification systems for both spinal column injury and spinal cord injury. The indications for nonoperative and operative management are discussed for both blunt and penetrating spinal injuries. The use of mean arterial pressure (MAP)-directed therapy is considered along with the limitations of its supporting data. Specific areas of management that are targeted include venous thromboembolism (VTE) prophylaxis, neurogenic and spinal shock, SCI-induced bradycardia, ventilator management in a patient with high SCI, and analgesia. Because patients with SCI often have a prolonged hospital course, the guideline also includes information on prevention and management of acute autonomic dysreflexia, spasticity, pressure ulcers, and neurogenic bowel and bladder. The importance of early mobilization and rehabilitation is reviewed. The last two sections cover implementation of the Spine BPG into trauma center protocols and integration of this information into the trauma center's performance improvement processes.

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IMPORTANT NOTE

The intent of the American College of Surgeons (ACS) Trauma Quality Programs (TQP) Best Practices Guidelines is to provide health care professionals with evidence-based recommendations regarding care of the trauma patient. The Best Practices Guidelines do not include all potential options for prevention, diagnosis, and treatment, and are not intended as a substitute for the provider's clinical judgment and experience. The responsible provider must make all treatment decisions based upon their independent judgment and the patient's individual clinical presentation. The ACS and any entities endorsing the Guidelines shall not be liable for any direct, indirect, special, incidental, or consequential damages related to the use of the information contained herein. The ACS may modify the TQP Best Practices Guidelines at any time without notice.

DISCLAIMER

The American Congress of Rehabilitation Medicine has reviewed this spinal cord injury best practice document and recommends it as an educational report. The rehabilitation key points in this document provide a practice-related, educational benefit to our members.

BEST PRACTICES GUIDELINES
SPINE INJURY

KEY FACTORS OF THE INITIAL SPINE EVALUATION

EPIDEMIOLOGY

KEY POINTS

- Vehicular trauma and unintentional falls are the leading mechanisms of spinal cord injury.
- Older adults are more susceptible to spinal injuries related to their increased risk for low velocity falls and the presence of underlying arthritic and osteoporotic conditions.

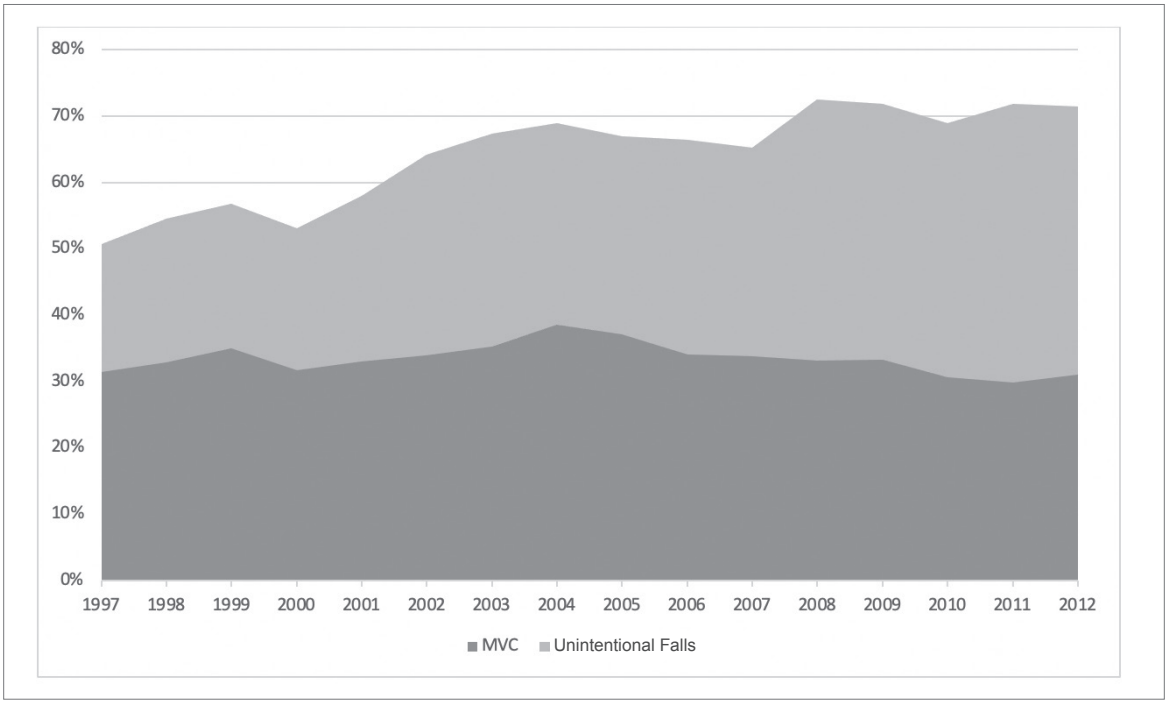
Spinal cord injury (SCI) incidence varies worldwide,[1] and in the United States an estimated 55 new cases per million population occur annually, not counting the cases who die before transport.[2,3] Little population-based data exists estimating the incidence of spinal column fractures (SCF) not associated with SCI; however, extrapolated data indicate the SCF incidence is approximately 8 times higher than SCI.[4-6] Of all blunt trauma patients included in the National Trauma Databank in 2010, approximately 13 percent had at least one SCF.[7]

The SCI incidence rate remains relatively stable, but the total number of SCI cases has increased because of population growth.[3,8-10] The epidemiology of these injuries has shifted, potentially reflecting changes in human behaviors or a higher rate of survival to hospital admission. The most important factor is the increased population of older adults[11] who are particularly susceptible to spinal injuries.[3,10,12,13]

Vehicular trauma consistently accounts for approximately 30 to 40 percent of SCI; however, unintentional falls, especially among older adults, are an increasingly important mechanism of injury, accounting for up to 66 percent of SCI in some countries (Figure 1).[3,9,13] Firearm injuries account for a small, but significant proportion of SCI (approximately 5 percent).[3] Sports-related SCI varies between countries, and in some countries account for 10 percent of all SCI. Diving and skiing are the sports associated with the highest risk for SCI.[14]

Older adults are more susceptible to spinal injuries related to their increased risk for low velocity falls and the presence of underlying conditions (e.g., osteoporosis and osteopenia, ankylosing spondylitis, and diffuse idiopathic skeletal hyperostosis) which may render the spine highly unstable even after a minor injury.[15] Osteoporotic spinal fractures in older adults from low energy falls accounted for more than 1.4 million hospitalizations worldwide in 2000,[16] and this number has increased significantly.[3,17,18] The upper cervical spine is the most common injury location in older adults.[19] SCI in older adults tends to be incomplete, manifesting as central cord syndrome, cervical extension/distraction injuries, and odontoid fractures.[19,20] Acute traumatic central cord syndrome due to cervical spondylosis is the most common incomplete SCI in this population.

Figure 1. Distribution of spinal cord injuries (SCI) based on motor vehicle collisions (MVC) and unintentional falls in the United States from 1997 to 2012



Modified from: Jain NB, Ayers GD, Peterson EN, et al. Traumatic spinal cord injury in the United States, 1993-2012. *JAMA*. 2015;313(22):2236. Used with permission.

The early mortality associated with SCF is estimated at approximately 4 percent, however, SCI has an associated mortality that is almost twice as high (7 percent)[5] with tetraplegic patients having the highest risk.[21] After the acute phase, this mortality increases and almost doubles at one year.[21] Compared with other trauma patients, those admitted with SCI injuries have a 2.5 fold increased length of stay, at approximately 12 days.[22] Compared with SCF from other causes, such as osteoporosis, trauma accounts for the highest associated cost of hospitalization, although the treatment modality appears to be the main driver of cost.[23] The total national charge attributable to SCI-related hospitalizations was approximately $1.7 billion in 2009.[22] Factors, such as need for re-hospitalization, post-injury rehabilitation, long-term complications, long-term care, and lost years of employment are not accounted for in these charges.[24] Additionally, for SCF patients, charges do not account for the long-term recovery, chronic pain, and the inability to return to baseline activity level.[25]

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PRE-HOSPITAL SPINAL MOTION RESTRICTION

KEY POINTS

- Spinal motion restriction (SMR) can be achieved with a backboard, scoop stretcher, vacuum splint, ambulance cot, or other similar devices.
- When SMR is indicated, apply it to the entire spine due to the risk of noncontiguous injuries.
- Assure that a sufficient number of properly trained individuals are available to assist with patient transfers to minimize the risk for displacement of a potentially unstable spinal injury.

The American College of Surgeons Committee on Trauma (ACS-COT), the American College of Emergency Physicians (ACEP) and the National Association of Emergency Medical Services Physicians (NAEMSP) recently published a detailed, joint consensus statement on spinal motion restriction for the trauma patient in the pre-hospital setting and during transport.[1]

The term "spinal motion restriction" (SMR) is recommended instead of immobilization, as current techniques limit or reduce undesired motion of the spine, but they do not provide true spinal immobilization. A backboard, scoop stretcher, vacuum splint, ambulance cot, or other similar devices may be used to achieve SMR. Refer to Table 1 for adult indications for SMR following blunt trauma. No role for SMR exists in penetrating trauma patients.

Table 1. Spinal motion restriction indications after blunt trauma[2]

Indications for Adults
▪ Acutely altered level of consciousness (e.g., GCS <15, evidence of intoxication)
▪ Midline neck or back pain and/or tenderness
▪ Focal neurologic signs and/or symptoms (e.g., numbness or motor weakness)
▪ Anatomic deformity of the spine
▪ Distracting circumstances or injury (e.g., long bone fracture, degloving, or crush injuries, large burns, emotional distress, communication barrier, etc.) or any similar injury that impairs the patient's ability to contribute to a reliable examination

From: National Association of EMS Physicians. EMS spinal precautions and the use of the long backboard – A joint position statement of the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma. 2018. https://naemsp.org/home/news/spinal-motion-restriction-in-the-trauma-patient--/ Accessed July 5, 2020. Used with permission.

When SMR is indicated in adults, apply it to the entire spine due to the risk of noncontiguous injuries. A critical component of SMR is the application of an appropriate size cervical collar. Keep the head, neck, and torso in alignment by placing the patient on a long backboard, a scoop stretcher, a vacuum mattress, or an ambulance cot. SMR cannot be properly performed with a patient in a sitting position.

All patient transfers pose a risk for a displacement of an unstable spine injury. Minimize flexion, extension, and rotation with the use of a long spine board, a scoop stretcher, or a vacuum mattress.

Consider removal of extrication devices during transport only if an adequate number of trained personnel are present to minimize unnecessary movement during the removal process. The risks of patient manipulation must be weighed against the benefits of device removal. If transport time is expected to be short, it may be better to transport a patient on the device and remove it on arrival at the hospital.

Once at the hospital, expeditiously evaluate patients for removal from the long backboard, scoop stretcher, or vacuum mattress. Facilities need a procedure to assure that a sufficient number of properly trained individuals are available to assist with patient transfers so the risk of inadvertent displacement of a potentially unstable spinal injury is minimized. Reducing the number of patient transfers is also important.

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CERVICAL COLLAR CLEARANCE

KEY POINTS

- The cervical collar can be discontinued without additional radiographic imaging in an awake, asymptomatic, adult trauma patient presenting with all of the following: a normal neurological exam, no high-risk injury mechanism, free range of cervical motion, and no neck tenderness.
- Removal of a cervical collar is recommended for adult blunt trauma patients who are neurological asymptomatic and have a negative helical cervical computed tomography (CT) imaging.
- A negative helical cervical CT scan is recommended as sufficient to remove a C-collar in an adult blunt trauma patient who is obtunded/unevaluable.

In the setting of acute trauma, improper cervical spine (C-spine) motion restriction and delayed diagnosis can lead to catastrophic neurological damage.[1-3] Blunt trauma is associated with a 2 to 6.6 percent risk of C-spine injury (boney and/or ligamentous).[4] The recommended standard of care for patients transported by emergency medical services (EMS) is maintenance of C-spine motion restriction until further assessment by an appropriate provider. Annually, approximately 5 million patients are placed in cervical collars.[5]

C-spine motion restriction is a critical component of initial trauma management, as is timely clearance and removal of the cervical collar, as well as backboards and other devices, used for spinal motion restriction. Risks associated with prolonged cervical collar use include delayed tracheostomy, deep venous thrombosis, aspiration, pneumonia, increased intracranial pressure, pressure ulcers, and difficult airway management.[4,6,7] Guidance from recent studies update the *2013 Guidelines for the Management of Acute Cervical and Spinal Cord Injuries*[8] for the management of cervical collars in adult patients, and is summarized below.

Awake, Asymptomatic Patient

The asymptomatic, awake blunt trauma patient is the most common presentation for evaluation of C-spine injury and removal of cervical collars placed in the pre-hospital setting.[8-14] Two established criteria guiding C-spine evaluation are the National Emergency X-Radiography Utilization Study Group (NEXUS) rules and the Canadian C-Spine rules (CCR).[15,16]

The NEXUS criteria were derived from the study of 4,309 asymptomatic patients in a cohort of 34,069 blunt trauma patients conducted before the routine, systematic use of CT scans. All patients had standard 3-view cervical x-rays and cervical CT scans as deemed necessary. Five essential criteria for low risk of cervical injury were identified: no posterior midline-cervical tenderness, no neurological deficit, normal alertness, no evidence of intoxication, and no painful distracting injury. The resulting analysis revealed a negative predictive value of 99.8 percent and sensitivity of 99.0 percent (Level I evidence) for traumatic cervical injury.[15]

The CCR study examined three criteria in 8,924 adults to warrant further imaging and continued motion restriction of the C-spine.[16] Patients were screened first for high risk injury factors (e.g., older than 65 years, dangerous mechanism, or abnormal neurological exam). Patients then needed to meet low risk-factor criteria [simple rear-end motor vehicle collision, sitting in the emergency department (ED), ambulatory at the time of injury, delayed (not immediate) onset of neck pain, or absence of midline C-spine tenderness] before performance of a cervical range of motion assessment. During the cervical range of motion test, patients were asked to move their head 45 degrees to the right and left. Failure (pain or inability to perform free range of motion) warrants further imaging and continued C-spine motion restriction. The CCR sensitivity for C-spine injury was reported at 100 percent. In 2003, a comparative prospective analysis of the NEXUS and CCR criteria found that the CCR criteria were significantly more sensitive and reduced the need for additional radiographic assessment when compared with NEXUS.[17]

In 2010, a meta-analysis of 14 Class 1 medical studies used rigid inclusion criteria for the clearance of cervical collars.[18] It was determined that a patient meeting the following criteria did not require further radiographic assessment to clear the cervical collar (negative predictive value of 99.8 percent and a sensitivity of 98.1 percent):

- Alert/unaltered
- Asymptomatic with no neurological deficit
- Complete a free range of cervical motion exam
- No sign of a distracting injury

An awake, asymptomatic adult trauma patient with a normal neurological exam, without a high-risk injury mechanism, free range of cervical motion, who presents without neck tenderness can have the cervical collar discontinued without additional radiographic assessment.

Awake Symptomatic Patient

Awake patients who fail the initial assessment for cervical collar clearance require proper radiographic evaluation for C-spine injury. Past recommendations suggested the use of plain radiograph to assess for clinically relevant fractures.[19-22] However, multiple randomized control trials with Class I evidence now consider plain radiography to be insufficient to identify clinically relevant fractures.[23-27] Plain films have a sensitivity ranging from 45 to 64 percent while modern helical cervical CT scans have sensitivity as high as 100 percent. The Western Trauma Association studied more than 10,000 patients and found CT had sensitivity of 98.5 percent for cervical injury (Level II evidence).[28] Duane et al. prospectively studied more than 9,000 patients and determined a CT sensitivity of 100 percent for cervical injury (Level III evidence).[29]

The utility of magnetic resonance imaging (MRI) after a negative CT was questioned for awake patients with continued neurological symptoms. In 2016, a prospective study of 10,756 patients by Inaba, et al. found only three false negative CT scans that missed clinically relevant symptoms consistent with central cord syndrome.[28] Study authors recommended that patients with negative CT scans but persistent neurological symptoms have an MRI. In 2017, Maung et al. found that patients with a negative CT but persistent neurological symptoms had an abnormal MRI rate of 27.6 percent; however, a definitive statement on the clinical significance of these findings could not be made because only 1.4 percent of patients had surgery after MRI (Level IV evidence).[30] A study by Malhotra et al. with 712 patients having a negative CT found 20.6 percent had an abnormal MRI, and 0.42 percent of patients had a change in management due to MRI findings (Level III evidence).[30] A meta-analysis of 23 studies demonstrated an exceptionally low rate (16 missed injuries in 5,286 patients) of unstable fractures missed by CT and found on MRI (Level 1 evidence).[31]

Removal of a cervical collar is recommended for adult blunt trauma patients who are neurological asymptomatic with a negative helical cervical CT. MRI is not required for removal of a cervical collar. However, at the treating physician's discretion, it can be considered in patients with persistent neurological symptoms, concern for ligamentous damage, high risk degenerative/pathological changes, despite a negative CT scan.

Obtunded and Unevaluable Patient

The proper radiographic assessment for obtunded or unevaluable patients is extremely important given the difficulty in accurate clinical assessment and concern for an unidentified injury causing catastrophic neurological damage. The 2013 guidelines highlighted the inadequacy of dynamic imaging (flexion-extension films), and its use was not recommended for initial clearance.[19,25,32-37] An 11 study meta-analysis (1550 total patients) determined that the diagnostic utility of MRI in CT-negative obtunded patients was clinically significant, altering management in 6 percent of patients (Level II evidence).[38] A second meta-analysis by Muchow et al., determined that MRI identified 20.9 percent of injuries not diagnosed on initial radiograph or CT imaging, and recommended that MRI become the gold standard.[39] These studies were criticized because of serious design flaws, including false endpoints, poorly matched populations, inclusion of a pediatric study, and dissimilar imaging protocols.[19] The 2013 guidelines recommended the possible use of MRI after a normal CT for patients who are obtunded/unevaluable.[19]

A more recent 17-study meta-analysis with 14,327 obtunded/intubated patients by Panczykowski et al. reported a negative predictive value of 100 percent, and both a sensitivity and specificity greater than 99.9 percent with a modern helical CT.[40] The analysis confirms that a modern helical CT scan alone is sufficient to clear the cervical collar in the obtunded/unevaluable patient. A 10-study meta-analysis with 1,859 obtunded patients by Raza et al. reported a normal CT sensitivity of 93.7 percent and negative predictive value of 99.7 percent.[41] The authors also reported their own retrospective cohort of 53 obtunded trauma patients with a CT and MRI and reported a CT sensitivity of 100 percent.[41] Badhiwala et al. conducted a detailed subset analysis of studies with high quality CT (1-3 mm slices) and found that after a negative CT that MRI did not change the management (collar or surgery) for any patient.[42]

In 2015, the Eastern Association for the Surgery of Trauma (EAST) updated its recommendation for C-spine evaluation to a Level III recommendation: a negative helical CT is sufficient for clearing the C-spine and removing cervical collars.[43] Several subsequent studies re-affirmed this recommendation. Inaba et al. prospectively studied 10,576 patients and documented that high quality CT scan had a sensitivity and negative predictive value of 98.5 percent and 99.97 percent, respectively.[28] Judicious use of MRI was recommended for suspected neurological compromise. Duane et al. examined a more than 9,000 patients and noted no missed cervical injuries in obtunded/unevaluable patients with negative CT scans.[29]

A negative helical cervical CT scan is recommended as sufficient to remove a cervical collar in an obtunded/unevaluable adult blunt trauma patient. When available, obtain spine surgeon consultation about the need for cervical MRI for patients when concerned about neurological symptoms and/or ligamentous injury referable to the spinal column.

Special Considerations: Older Adult Patients

The NEXUS criteria do not include age-related contingencies, while the CCR considers age greater than 65 years as a risk a factor. The 2013 SCI guidelines, along with the EAST guidelines, do not mention any special consideration for older adult patients. Healey et al. reported that 20 percent of patients older than 55 years with spine fractures were asymptomatic, with no midline tenderness on presentation for blunt trauma.[44] The reported rate of cervical surgery for "asymptomatic" patients older than 55 years was similar to the "symptomatic" population (Level III evidence). The authors concluded that pain was not equally assessed in the older population. A study applying the NEXUS criteria to an older adult trauma population (older than 65 years) found a sensitivity of 100 percent (Level III evidence).[45] The *ACS TQIP Best Practices Guidelines in Imaging* recommend more liberal imaging for older adults.[46]

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IMAGING

KEY POINTS

- Plain radiographs of the cervical and thoracolumbar spine are *not* recommended in the initial screening of spinal trauma because of their low sensitivity.
- Non-contrast, multidetector computerized tomography (MDCT) is the initial imaging modality of choice to evaluate the cervical and thoracolumbar spine.
- MRI is the only modality for evaluating the internal structure of the spinal cord.
- Consider universal screening for blunt cerebrovascular injury for all patients with major trauma using a whole-body CT scan.

After using the clinical decision rules and assessing risk for SCF or SCI during the primary and secondary survey, patients at risk for spinal trauma are further evaluated with dedicated imaging studies. The aim of imaging is to avoid preventable neurological deterioration and to guide short- and long-term management of spinal injury.[1] Plain radiographs of the cervical and thoracolumbar spine are *not* recommended in the initial screening of spinal trauma because of their low sensitivity.

Computerized Tomography (CT)

Non-contrast, MDCT is the initial imaging modality of choice to evaluate both the cervical and thoracolumbar spine. The sensitivity of this modality exceeds 98 percent for the cervical spine[2] and approaches 100 percent for the thoracolumbar spine.[3] The use of MDCT offers a significant increase in imaging acquisition speed and in high spatial resolution, with increased coverage of the patient. Three-dimensional images are easily obtained and offer greater detail.[4] Overall, this technology offers a high degree capability and utility for examining the spine.

The cervical spine is imaged from the skull base or craniocervical junction, through the cervicothoracic junction. Thinner slices increase sensitivity but may add to the radiation burden. Slices of no greater than 3 mm are recommended to obtain reformatted images in axial, coronal, and sagittal planes in the cervical spine.[5] When combined with a CT of the brain, the cervical spine CT can be completed rapidly.[6] The current recommendation from the American College of Radiology Practice Parameter for the performance of CT of the spine is for slices no greater than 3 mm when imaging the cervical spine, which differs from the *ACS TQIP Best Practices Guidelines in Imaging* published in 2018.

The thoracolumbar spine is imaged in the same fashion with slice thickness not to exceed 5 mm.[5] Thoracolumbar spine imaging may be reconstructed from images concurrently obtained for evaluation of the chest, abdomen and pelvis in a multi-trauma patient. Routine reformatting of these images is not required. A more selective imaging approach is appropriate for patients with a high suspicion of spine injury or when an injury is identified on the non-reformatted images.

Despite the high sensitivity of the MDCT in identifying bony abnormalities, interpretation may be difficult in patients with severe degenerative changes or osteopenia. In addition, ligamentous or cord injuries will not be depicted, although they may be suggested by certain patterns of injury, indicating a requirement for MRI.

Magnetic Resonance Imaging (MRI)

Although MDCT of the spine is considered the first line imaging modality in the acute setting, MRI often provides complimentary data, especially in patients with discordant CT and clinical findings. MRI allows for direct visualization of the spinal cord, nerve roots, and discs, and it provides better soft-tissue contrast. It is the only modality for evaluating the internal structure of the spinal cord.[7]

Obtain an MRI after consultation with a spine specialist, if able. MRI findings, especially in the acute setting, may mislead clinicians. Clinically insignificant soft tissue abnormalities may be detected that may trigger additional, unnecessary workup or interventions.[1,8]

MRI has a role as a complementary imaging study for patients with SCF identified on MDCT, when a concern exists about spinal stability and/or SCI, and when a surgical intervention is planned.

MRI may be selectively performed in patients with clinical findings that do not correlate with the MDCT findings. Examples include patients with neurological deficits and a normal spine MDCT, and patients with out-of-proportion pain and a normal MDCT, or a MDCT demonstrating severe degenerative changes or osteopenia.

It is not necessary to perform routine MRI for unevaluable patients with a normal MDCT and no apparent neurological deficit to complete the screening for spinal injuries.[9,10]

In comparison to MDCT, MRI requires significantly more time for completion. The patient must be cooperative and avoid movement for the duration of the study. Conducting an MRI may pose challenges when evaluating the following patients: on a ventilator, hemodynamic instability, are claustrophobic, or unable to remain still for a prolonged period of time. Often, the help of anesthesia is requested and occasionally, some patients require endotracheal intubation and sedation to complete the MRI. The presence of metallic implants may interfere with the images, and they may preclude patients from having the MRI performed.

Blunt Cerebrovascular Injury

Blunt force trauma resulting in blunt cerebrovascular injury (BCVI) has a reported incidence of 2.7 percent to 7.6 percent.[11,12] Undetected carotid and vertebral artery injury can lead to delayed therapy and increase the patient's risk of stroke. Untreated carotid and vertebral artery injuries have a mortality as high as 38 percent and 18 percent, respectively.[13] Initial clinical symptoms needing further investigation include the following:[14]

- Carotid bruit,
- Expanding neck hematoma,
- Neurological deficit,
- Evidence of ischemic stroke on head imaging, and
- Arterial bleeding.

High-risk patients can present with both carotid and vertebral injury.

Spine fractures are the single-most predictive factor of BCVI, with greatest risk to the vertebral artery.[15,16] Angiography studies reveal that up to 24 percent of patients with a C-spine injury have a concomitant vertebral artery injury. The highest-risk C-spine injury is subluxation, followed by fractures involving the transverse foramen and the high cervical spine at C1 to C3.[13,16] Patients with minor cervical injuries including vertebral body and laminar factures are the lowest risk for BCVI, but a reported association is still present.[17] Elderly patients with low-energy injury mechanisms, including ground level falls are at risk for BCVI.[18] Radiographic screening for BCVI with CT angiography is a sufficient and cost effective modality, and it is the recommended means of excluding this injury.[14,19,20]

Traditional screening criteria can miss 20 to 30 percent of BCVI.[11,12,21] Despite an increase in the number of screening indications, BCVI are still missed.[12,22] Several studies now recommend universal screening for all patients with major trauma using a whole-body CT scan (WBCT).[11,12,21] The WBCT includes a noncontrast CT head followed by a multi-slice CT scan (40 or 64 slice), incorporating a single IV contrast-enhanced pass from the circle of Willis through the pelvis. This imaging allows screening for BCVI while evaluating the cervical spine, chest, abdomen and pelvis.[11,12,22] Although some authors question the benefit of a universal WBCT scan for trauma patients, the practice is supported by the American College of Radiology's Appropriate Use Criteria of CT scans for major blunt trauma.[23,24] When a BCVI is found, initiate treatment with anti-coagulation or anti-platelet agents, if indicated once deemed clinically safe.

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PHYSICAL EXAMINATION

KEY POINTS

- Using the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) clinical documentation tool is a best practice.
- The neurologic level of injury is determined from the assessment of sensory and motor levels of injury.
- Make no assumptions about the extent of the neurologic injury in the presence of a depressed level of consciousness, extremity or pelvic fractures, burns or other injuries, such as to the brachial plexus, that may affect the results of sensory or motor deficit evaluation.

Initially evaluate all trauma patients based on the principles of Advanced Trauma Life Support®, independent of whether an SCF or SCI is suspected or confirmed.[1]

Primary Survey

Conduct the primary survey focusing on hemorrhage control, airway, breathing, circulation, disability, and exposure. Maintain cervical and thoracolumbar SMR throughout this phase, until the spine is further evaluated during the secondary survey. The primary goal during the "disability" evaluation, after assessing the Glasgow Coma Scale (GCS) score and pupillary response, is to identify any lateralizing signs by conducting a rapid assessment of motor function and reflexes in the extremities. In alert and cooperative patients, simply ask them to raise upper and lower extremities sequentially and observe for any differences.

Signs observed during the primary survey suggestive of an SCI include the following:

- Absence of equal movement in the upper and/or lower extremities that may be combined with a gross sensory deficit.
- Complete or partial loss of muscle tone and loss of bladder or bowel function.
- Priapism may be observed in male patients.[2]

Subtle signs of a SCI may include tachypnea and diaphragmatic breathing in high level SCI. Raise your index of suspicion when other explanations for these signs are adequately ruled out. Consider an SCI associated with neurogenic shock when circulatory shock is present and unexplained by hypovolemia and/or bleeding, especially when associated with bradycardia.

Be extremely cautious about attributing observed signs to a SCI when other life-threatening causes have not been adequately excluded, such as circulatory shock from bleeding. The primary survey is not intended to fully evaluate the spine during the initial evaluation for life-threatening injuries. However, if any clinical signs suggestive of SCI are identified, rapidly determine the level of injury because this information will aid in the differential diagnosis of observed signs and symptoms and will facilitate targeted treatment.

Spinal Motion Restriction during the Primary Survey. The same principles of pre-hospital SMR are applied during the primary survey. Refer to the section on Pre-Hospital Spinal Motion Restriction on page 9. Attempting to align the spine to aid with SMR is acceptable. *Do not, under any circumstances*, use force to move the patient's neck or thoracolumbar spine into a position that elicits pain. Remove the backboard expeditiously to minimize the risk of pressure ulcers and unnecessary discomfort. Maintaining a patient in a supine position on the firm gurney has the same SMR result as use of a backboard. Perform all additional examinations by log rolling the patient when necessary.

Airway Management. Securing the airway may be required during the primary survey. Secondary injuries to the spinal cord may result from inadequate SMR during airway maneuvers. However, most secondary injuries likely result from ongoing cord ischemia and/or edema, assuming that a reasonable effort was made to maintain SMR.[3] Complete immobilization of the cervical spine during endotracheal intubation is virtually impossible, and some degree of movement of the neck is to be expected.[3,4] It is important to make the effort to maintain SMR. Suggested strategies include:

- Maintain the cervical collar during airway manipulations.
- Apply manual inline stabilization of the head and neck with the removal of the anterior portion of the collar to allow for a wider mouth opening and easier visualization of the oropharynx and cords.[3]
- Secure the airway with an endotracheal tube using direct laryngoscopy or awake intubation with a bronchoscope.

Surgeons are encouraged to familiarize themselves with techniques to rapidly and safely secure the airway, because no specific technique has proven superior to another.[3]

Secondary Survey

The secondary survey aims to obtain a full and detailed history and physical examination after completion of the primary survey, and the patient is deemed to be stable and have no life-threatening injuries. The clinical decision tools to identify patients at risk for a spinal injury require further imaging investigative studies.

Documentation. For patients with suspected SCI, clearly detail the complete neurologic evaluation. Serial documentation of abnormal physical findings during subsequent evaluations allows for early identification of secondary injuries which may have implications in management decisions. Ensure this documentation includes the date and time performed and name of the examiner.

Mechanism of Injury. Mechanism of injury (MOI) often aids in the assessment of the risk for SCF and SCI. Any high-energy mechanism may result in spinal trauma. Examples include: high speed motor vehicle collisions (especially when associated with ejection or roll over); motorcycle, bicycle, and recreational vehicle collisions; crush injuries; falls from height; injuries leading to an axial load to the head (e.g., diving and auto versus pedestrian).[3,5–8]

Low energy mechanisms, such as ground level falls, place older adults at a high risk for SCF and SCI.[9–11] Age by itself, is considered a high-risk factor for spinal trauma, independent of mechanism.[8] Factors contributing to this high risk include osteoporosis, osteopenia, and a generalized decrease in the spinal column's physiologic protective capabilities due to degenerative and other age-related changes.[10]

History. If the patient is evaluable and cooperative, ask about neurologic signs and symptoms. Ask about the presence of new onset neck or back pain, any associated neurological deficit in the upper and/or lower extremities, such as abnormal sensation (numbness, tingling) or motor deficit. However, absence of such symptoms with a distracting injury may be misleading.

Review the patient's past medical history to assess the risk for an injury. Osteoporosis is one of the most common pathologies placing older adults at risk for spinal injury.[12] Other pre-existing conditions may also play a role, such as spinal lytic lesions from metastatic cancer, rheumatoid arthritis, and ankylosing spondylitis.[10,13] Seek information about pre-existing neurologic deficits, because they have implications in the assessment of acute SCI. Inquire about any history of prior injuries to the spine and the presence of spinal implants from prior surgeries or interventions.

Associated Injuries. Consider all trauma patients who present with severe injuries at high risk for associated spinal trauma. Similarly, consider the presence of specific injuries identified during the primary and secondary survey when assessing the risk for a spinal injury. For example, traumatic brain injuries (TBI), manifested by a decreased GCS score, place trauma patients at a high risk for an associated C-spine injury.[14–16] Complex maxillofacial trauma, especially when a result of high energy mechanism, is another example.[14,17–19] A high proportion of patients with a pelvic fracture have associated spinal trauma to the cervical, thoracolumbar and sacral region.[20] Thoracic trauma, including multiple rib fractures, sternal, scapular and clavicular fractures increase the risk for associated cervical and thoracic spinal injuries.[19,21] Calcaneal fractures resulting from a fall from height, indicate a spinal injury risk, usually at the level of the lumbar region.[22] Patients who present with a seatbelt-shaped abdominal contusion are also at risk for thoracolumbar spine trauma from a flexion-distraction injury.[23,24] Also consider patients to be at high risk who require emergent surgical exploration for a gunshot wound or blunt traumatic injury, when an associated spinal injury may be found on postoperative imaging.[25–27]

Structured Spine Physical Examination

A structured physical examination of the entire spine is performed during the secondary survey with inline SMR. A comprehensive neurological evaluation is performed.

Cervical Spine. Ask the patient to not move the neck as the cervical collar is opened, flipping the anterior portion to one side. SMR is applied as the examiner inspects the neck for abrasions, contusions, hematomas, or open

wounds. The entire cervical spine is palpated along the midline (spinous processes) while evaluating for pain and/or tenderness, step offs, and any other deformities. The paraspinal regions are also evaluated bilaterally for the presence of tenderness, pain and/or muscle contraction. The cervical collar is then secured in place. To complete the examination of the cervical spine, the same steps are repeated when the patient is logrolled to evaluate the thoracolumbar spine.

Thoracolumbar and Sacral Spine. Perform this examination when the backboard is removed, if present. The complete evaluation of the thoracolumbar spine requires the patient to be in a standing, sitting, supine, and prone position. However, this is not feasible when SMR must be maintained during the acute phase of trauma care, until an injury has been ruled out. With the patient logrolled using inline SMR, inspect the back for obvious spinal deformities, as well as contusions, abrasions, hematomas, and/or open wounds. The cervical spine region is also inspected to complete that examination. Systematically palpate the entire thoracolumbar and sacral midline to evaluate for pain, tenderness, step offs, gaps or any other deformities.

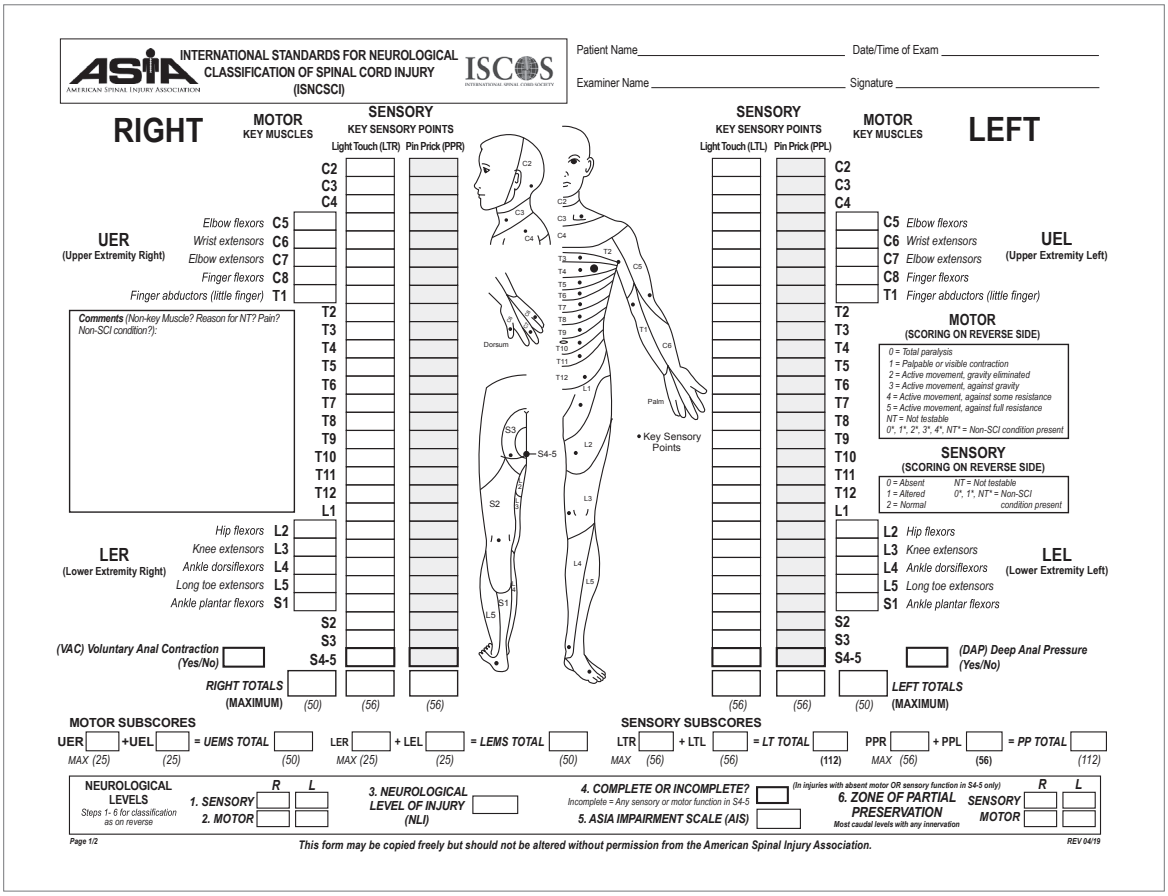
When an SCI is suspected, perform a digital rectal exam before rolling the patient back to the supine position after removal of the backboard.

The physical examination of the thoracolumbar spine has very low sensitivity. The level of pain and/or tenderness often does not correlate with the level of injury on imaging. Most importantly, however, a normal exam has a low sensitivity in ruling out these injuries.[8,18,28]

The International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI)

When a SCI is suspected, attempt to rapidly identify the level of injury. The ISNCSCI is a clinical documentation tool published by the American Spinal Injury Association (ASIA) and maintained by the International Standards Committee of ASIA and the International Spinal Cord Society (ISCoS). A best practice is use of this tool to document the level and severity of a SCI (refer to Figure 2).[29] See guidelines for assessment and documentation that follow.

Figure 2. The International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI)



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Be aware that this tool does not include all the elements of a comprehensive neurological examination, such as deep tendon reflexes or evaluation of position sense. It includes elements only required to determine the ASIA impairment scale (AIS), i.e., the sensory and motor level of injury. Nonetheless, its performance with the patient in a supine position with the use of common clinical measures, minimal equipment, and in virtually any clinical setting or phase of care, makes it an attractive and useful tool when evaluating patients with SCI.

Sensory Level of Injury. The sensory level refers to the most caudal segment of the spinal cord with normal sensory function. This level is evaluated by examining the corresponding dermatomes. Figure 2 outlines the distribution of dermatomes and Table 3 lists these distributions.

Perform a systematic assessment of sensory level with light touch and a pinprick of each dermatome using the face as control. Grade each sensory level as:

0 = Absent

1 = Altered (either decreased/impaired sensation or hypersensitivity)

2 = Normal

NT = Not testable

An asterisk "*" tag is added to grades 0, 1 or NT in the ASIA worksheet (the only abnormal indicators) when an impairment is due to a non-SCI condition.

Additional Guidance:

- Perform the light touch sensory scoring with strokes (not to exceed 1 cm) across the skin using a cotton tip applicator.
- Test the dermatomes in the anterior torso (chest and abdomen) at the midclavicular line.
- Perform the pinprick (sharp-dull discrimination) sensory scoring with a clean safety pin. Apply a single sharp and dull stimulus using consistent pressure with the safety pin.
- The most caudal aspect of the spinal cord (S4/S5) is examined in the same fashion as all dermatomes.
- A digital rectal examination is performed to determine sensory awareness. It is recorded as present or absent *deep anal pressure*.

After determining the sensory level on the right and left sides for light touch and pinprick, the most caudate level where sensation is normal bilaterally (grade of 2 for both, right and left) is reported as the normal sensory level.

Table 3. Areas of innervation of spinal nerves[30]

Spinal Nerve	Area of Innervation
C2	At least 1 cm lateral to the occipital protuberance (alternatively 3 cm behind the ear)
C3	Supraclavicular fossa (posterior to the clavicle) and at the midclavicular line
C4	Over the acromioclavicular joint
C5	Lateral (radial) side of the antecubital fossa (just proximal to elbow crease)
C6	Thumb, dorsal surface, proximal phalanx
C7	Middle finger, dorsal surface, proximal phalanx
C8	Little finger, dorsal surface, proximal phalanx
T1	Medial (ulnar) side of the antecubital fossa, just proximal to the medial epicondyle of the humerus
T2	Apex of the axilla
T3	Midclavicular line and the third intercostal space (IS) found by palpating the anterior chest to locate the third rib and corresponding IS below it
T4	Fourth IS (nipple line) at the midclavicular line
T5	Midclavicular line and the fifth IS (midway between T4 and T6)
T6	Midclavicular line and the sixth IS (level of xiphisternum)
T7	Midclavicular line and the seventh IS (midway between T6 and T8)
T8	Midclavicular line and the eighth IS (midway between T6 and T10)
T9	Midclavicular line and the ninth IS (midway between T8 and T10)
T10	Midclavicular line and the tenth IS (umbilicus)
T11	Midclavicular line and the eleventh IS (midway between T10 and T12)
T12	Midclavicular line and the mid-point of the inguinal ligament
L1	Midway distance between the key sensory points for T12 and L2
L2	On the anterior-medial thigh at the midpoint drawn connecting the midpoint of inguinal ligament (T12) and the medial femoral condyle
L3	Medial femoral condyle above the knee
L4	Medial malleolus
L5	Dorsum of the foot at the third metatarsal phalangeal joint
S1	Lateral heel (calcaneus)
S2	Mid-point of the popliteal fossa
S3	Ischial tuberosity or infragluteal fold
S4-S5	Perianal area less than one cm. lateral to the mucocutaneous junction (taken as one level)

Motor Level of Injury. The strength of five key muscle groups in the upper extremities and five in the lower extremities is tested bilaterally to determine the motor level of injury (Table 4.) Provide resistance to the movement of muscle groups in both directions and assess for any indication of diminished strength.[29,31] Muscle strength graded using a 6-point score (0 to 5):

0/5 = Total paralysis

1/5 = Palpable or visible contraction

2/5 = Active movement, full range of motion (ROM) with gravity eliminated

3/5 = Active movement, full ROM against gravity

4/5 = Active movement, full ROM against gravity and moderate resistance in a muscle specific position

5/5 = Active movement, full ROM against gravity and full resistance in a functional muscle position, normal for an otherwise unimpaired person

NT = Not testable (e.g., immobilization, severe pain prevents grading, limb amputation, or contracture of greater than 50 percent of the normal ROM)

An asterisk "*" tag is added to 0, 1, 2, 3, 4 or NT (e.g., the only abnormal indicators) when an impairment is due to a non-SCI condition in the ASIA worksheet.

Additional Guidance:

- Assign a score for each key muscle group of the right and left upper and lower extremities.
- Complete the motor assessment by evaluating the contraction of the external anal sphincter around the examiner's finger. Scoring is reported as present or absent *voluntary anal contraction*.
- The normal motor level on each side is determined by a minimum score of 3, with all the proximal key muscle groups being 5 (normal.)
- The overall motor level score is the last normal for both (a minimum of 3 bilaterally.) If for any reason the motor level cannot be tested or determined, the motor level score is considered to be the same as the sensory level score.

Table 4. Key muscle groups for the upper and lower extremities with corresponding neurologic level and muscle movement(s)

Extremity	Root Level	Muscle Group	Muscle Movement(s)
UPPER	C5	Elbow flexors	**Shoulder:** Flexion, extension, abduction, adduction, internal and external rotation **Elbow:** Supination
	C6	Wrist extensors	**Elbow:** Pronation **Wrist:** Flexion
	C7	Elbow extensors	**Finger:** Flexion at proximal joint, extension **Thumb:** Flexion, extension and abduction in plane of thumb
	C8	Long finger flexors	**Finger:** Flexion at metacarpophalangeal (MCP) joint **Thumb:** Opposition, adduction and abduction perpendicular to palm
	T1	Small finger abductor	**Finger:** Abduction of the index finger
LOWER	L2	Hip flexors	**Hip:** Adduction
	L3	Knee extensors	**Hip:** External rotation
	L4	Ankle dorsiflexors	**Hip:** Extension, abduction, internal rotation **Knee:** Flexion **Ankle:** Inversion and eversion **Toe:** Metatarsophalangeal (MP) and interphalangeal (IP) joint extension
	L5	Long toe extensors	**Hallux and Toe:** Distal and proximal interphalangeal joints (DIP and PIP) flexion and abduction
	S1	Ankle plantar flexors	**Hallux:** Adduction

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Neurological Level of Injury (NLI). The neurologic level of injury is defined as the levels where motor function and sensation are both intact bilaterally:

- The most distal or caudal level at which the motor (minimum strength of 3 bilaterally with all levels proximally being 5) plus
- The most distal level where sensation is intact on light touch and pinprick with all proximal levels being intact.

Description of the NLI is reported as complete or incomplete. The injury is considered incomplete when sacral sparing exists. Sacral sparing is present when light touch and pinprick sensation are intact at the S4/S5 level, and deep anal pressure and voluntary anal contraction are present. The *zone of partial preservation* is reported only for complete injuries, and it refers to those dermatomes and myotomes caudal to the sensory and motor levels that remain partially innervated.

At this point, an ASIA Impairment Scale (AIS) classification is assigned, based on whether the injury is complete (ASIA-A) or incomplete (ASIA-B, C, or D), which is based on the preservation of sensory and/or motor function and the number of key muscle groups with partial preservation (Table 5).

Table 5. ASIA Impairment Scale (AIS) and description of associated deficits[29]

ASIA Impairment Scale	Description of Deficits
Asia-A: Complete	No sensory or motor function is preserved in the sacral segments S4-5.
Asia-B: Sensory Incomplete	Sensory, but not motor function, is preserved below the neurological level and includes the sacral segments S4-S5 (light touch or pin prick at S4-S5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.
Asia-C: Motor Incomplete	Motor function is preserved at the most caudal sacral segments for voluntary anal contraction OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments S4-S5 by light touch, pinprick or deep anal pressure), 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.
Asia 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.
Asia 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.
ND	For documentation of the sensory, motor and NLI levels, the ASIA-AIS, and/or the zone of partial preservation when they cannot be determined from examination results.

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Additional Elements of a Complete Neurological Examination

Cranial Nerves. Complete evaluation of the 12 cranial nerves is part of a complete neurological evaluation. It has implications for spinal trauma, because any deficits may indicate TBI and a higher associated risk for spinal injury. Certain maneuvers to test these, e.g. the oculocephalic reflex ("doll's eyes"), may not be feasible initially when the requirement for SMR exists.[31]

Reflexes. The deep tendon reflexes are tested by stimulating a muscle stretch (myotatic) with the use of a percussion hammer. Reflexes represent the sensory and motor functionality of motor fibers of a respective spinal level. Test reflexes bilaterally and note asymmetries. Some reflexes are graded simply as present or absent, such as the plantar stimulation and the bulbocavernosus reflex. Other reflexes are rated based on a scale from 0 to 5, as follows:

0 = Absent reflex

1+ = Trace response

2+ = Normal response

3+ = Brisk response

4+ = Non-sustained clonus (repetitive vibratory movements)

5+ = Sustained clonus

Commonly examined reflexes include the biceps (C5/C6,) triceps (C7,) patellar (L2/L3/L4,) ankle (S1,) and plantar flexion (S1). In the acute phase of a complete SCI, all reflexes below the level of injury are absent,[31] however, bulbocavernosus reflex (S3-S4) may remain intact. Assess the bulbocavernosus reflex by briskly squeezing the glans penis or clitoris, or by pulling gently on the bladder indwelling catheter, while feeling for increased rectal tone. Absence of this reflex indicates the presence of spinal shock which usually resolves within a few hours to days. Return of this reflex with continued absence of all other reflexes below the level of injury indicates a complete SCI.[32] With spinal shock, the bulbocavernosus reflex returns first and other reflexes follow gradually. Complete resolution of spinal shock is preceded by a phase of hyperreflexia.[33]

Proprioception. Proprioception is an individual's ability to determine body segment positions and movements in space. Orient the patient first. Ask them to close their eyes and report movement when a joint is flexed and extended, and then to discriminate between flexion and extension. The exam starts distally, at the interphalangeal joint of the big toes bilaterally, and continues proximally to the ankle, knee, and hip. Examination of the upper extremity joints is performed starting at the metacarpophalangeal joint, and continues proximally to the wrist, elbow and shoulder.

Tertiary Survey. A tertiary survey is conducted after admission when the patient is stabilized and all injuries were addressed. Also perform another complete evaluation of the spine, especially if no imaging was previously performed. Obtain imaging at admission and during the hospital course as needed. Review imaging to rule out a missed spinal injury.

The Patient Who Cannot Be Evaluated. A depressed level of consciousness is a risk factor for an associated spinal injury. When completing the physical examination described above is not feasible, use imaging studies to evaluate these patients while maintaining SMR. The presence of extremity or pelvic fractures, burns, or other injuries, such as to the brachial plexus, may affect the results of sensory or motor deficit evaluation. Clearly document these limitations or factors and make no assumptions about the absence or presence of a neurological deficit. Perform a comprehensive examination of the spine as soon as the patient becomes evaluable.

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BEST PRACTICES GUIDELINES

SPINE INJURY

CLASSIFICATION AND MANAGEMENT OF INJURY

SPINE INJURY CLASSIFICATION SYSTEMS

KEY POINTS

- Spine trauma classification systems include specific injury characteristics, as well as the patient's medical or neurologic status.
- Patient scores for the classification system are used to guide decision making regarding surgery or nonsurgical management.

The use of a spine trauma classification system helps evaluate the urgency and severity of spinal injury with other concomitant injuries in the polytrauma patient. It also facilitates communication among the multidisciplinary team including emergency physicians, intensivists, and surgeons.

Many classification systems previously proposed for spinal trauma ranged from anatomic criteria to mechanistic descriptions. Fractures and dislocations of the spine were originally described based on their stability, with risk for increased deformity and subsequent neurological injury.[1-4] While these systems incorporated morphological characteristics and inferred spinal stability from radiological assessment, important factors such as the patient's medical or neurological status were not integrated.

Novel comprehensive classification systems were developed using literature reviews and expert consensus to address these limitations and to facilitate communication and guide treatment. Examples include the Cervical Subaxial Injury Classification System (SLIC) and the Thoracolumbar Injury Classification System (TLICS).[5,6] Both systems include the patient's neurologic status in a point-based scoring system. The AO Spine Injury Classification System was developed as a more comprehensive evaluation system that also includes the upper cervical spine, sacral injuries, and patient characteristics such as underlying medical condition, and other spinal disorders.

Cervical Subaxial Injury Classification System (SLIC)

The SLIC describes injuries to the subaxial cervical spine (C3-C7) and is a point-based system. It consists of 3 categories: fracture morphology, integrity of the discoligamentous complex (DLC), and neurological status (refer to Table 6).[6]

Table 6. Cervical Subaxial Injury Classification (SLIC)

Injury Morphology (Max 4)	Points
▪ No fracture/abnormality	0
▪ Compression	1
▪ Burst	+1 = 2
▪ Distraction (e.g., hyperextension, facet perch)	3
▪ Rotation/translation (e.g., facet dislocation, unstable tear-drop, advanced flexion compression injury)	4
Disco-ligamentous Complex (DLC) Integrity (Max 2)	
▪ Intact	0
▪ Indeterminate (isolated interspinous space widening, signal change on MRI only).	1
▪ Disrupted (e.g., widening of disc space, facet perch or facet dislocation)	2
Neurological Injury (Max 4)	
▪ Intact	0
▪ Root Injury	1
▪ Complete spinal cord injury	2
▪ Incomplete spinal cord injury	3
▪ Ongoing cord compression in setting of neurological deficit	+1

Adapted from: Vaccaro AR, Hulbert RJ, Patel AA, et al. The Subaxial Cervical Spine Injury Classification System. *Spine (Phila Pa 1976)*. 2007; 32(21): 2365-2374. doi:10.1097/BRS.0b013e3181557b92

Fracture morphology grading is based on fracture patterns discerned on CT scans.

- Compression injuries are noted as visible loss of height through part or an entire vertebral body (including traditional "burst" fractures) and are assigned either 1 or 2 points.
- Distraction injuries (more severe injuries evidenced by anatomic dissociation in the sagittal plane) are given 3 points. These include flexion-distraction injuries with facet perch that disrupt the strong capsular constraints and bony articulation of the facet joints posteriorly, as well as extension-distraction injuries disrupting anterior constraints (anterior longitudinal ligament, intervertebral disc and body).
- Rotation/translation injuries represent horizontal displacement of one cervical segment with respect to the other and are given 4 points. Translation injuries are noted to be present when any visible translation is not related to degenerative causes (e.g., unilateral or bilateral facet-fracture dislocations, "floating" lateral mass, pedicle fractures). Angulation ≥ 11° in the axial plane is suggested as a threshold for presence of rotational injury.[7]

Integrity of the DLC represents a critical factor in treatment determination. It is best assessed on MRI, but it may be inferred on CT or plain radiographs. The DLC is comprised of the anterior and posterior longitudinal ligaments, the intervertebral disc, ligamentum flavum, interspinous and supraspinous ligaments, and facet capsules. Indeterminate injuries to the DLC are assigned 1 point. These include injuries with isolated T2 signal change on MRI through posterior ligaments, or injuries with isolated interspinous splaying or widening. DLC disruption is assigned 2 points and is present with clear widening of the disc space or abnormal facet alignment.

The neurological status of the patient is considered with the third category. Patients with a root level injury are assigned 1 point, whereas those with complete spinal cord and incomplete spinal cord injuries are assigned 2 and 3 points, respectively. An additional point is added if significant compression is present in the presence of a neurologic deficit (e.g., congenital or spondylotic stenosis without overt fracture or ligamentous injury).

Based on the recommendations of this system, injuries with a total SLIC score of 1-3 are treated nonoperatively, those with a total score ≥ 5 are best treated operatively, whereas those with a score of 4 can be treated operatively or nonoperatively, based on the treating physician's best judgment.

Thoracolumbar Injury Classification System (TLICS)

The TLICS classification, describing injuries to the thoracolumbar spine, is also comprised of three categories: injury morphology, integrity of the posterior ligamentous complex (PLC), and neurologic status (Table 7).[5]

Table 7. Thoracolumbar Injury Classification System (TLICS)

Injury Morphology	Points
No fracture/abnormality	0
Compression	1
Burst	+1 = 2
Translation/rotation	3
Distraction	4
Posterior Ligamentous Complex (PLC) Integrity	
Intact	0
Suspected/indeterminate	2
Disrupted	3
Neurological Injury	
Intact	0
Root Injury	1
Complete spinal cord injury	2
Incomplete spinal cord injury	3
Cauda equina syndrome	3

Adapted from: Vaccaro AR, Lehman RA, Hurlbert RJ, et al. A new classification of thoracolumbar injuries: The importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. *Spine (Phila Pa 1976)*. 2005; 30(20): 2325–2333.

Injury morphology grading includes the following:

- Compression fractures (injuries of the anterior vertebral body with resultant kyphosis) are assigned 1 point. If the vertebral body fracture extends to the posterior vertebral body (traditional "burst" fracture), an additional point is given.
- Rotational injuries are more severe and are best assessed on axial CT scans. Any horizontal separation of the spinous process or malalignment of pedicles above or below the injury is suggestive of rotational injury. Translation is readily assessed on sagittal or coronal imaging. Rotation/translation injuries imply torsional or shear forces on the spine and are assigned 3 points.
- Distraction injuries are present when the cranial vertebral segment is circumferentially disrupted from the caudal segment, indicating significant spinal instability. They are assigned 4 points.

The PLC in the TLICS consists of the supraspinous ligament, interspinous ligament, ligamentum flavum, and facet joint capsules. Disruption of the PLC is assigned 3 points and can be inferred with widening of the interspinous space, or facet joint diastasis, perching, or subluxation. Suspected/indeterminate PLC injuries are given 2 points.

Evaluation of the neurologic status includes 1 point assigned for a root level injury, 2 points for a complete (motor and sensory) cord-level injury, and 3 points for an incomplete cord-level injury. In the lumbar spine, an evolving cauda equina injury is assigned 3 points.

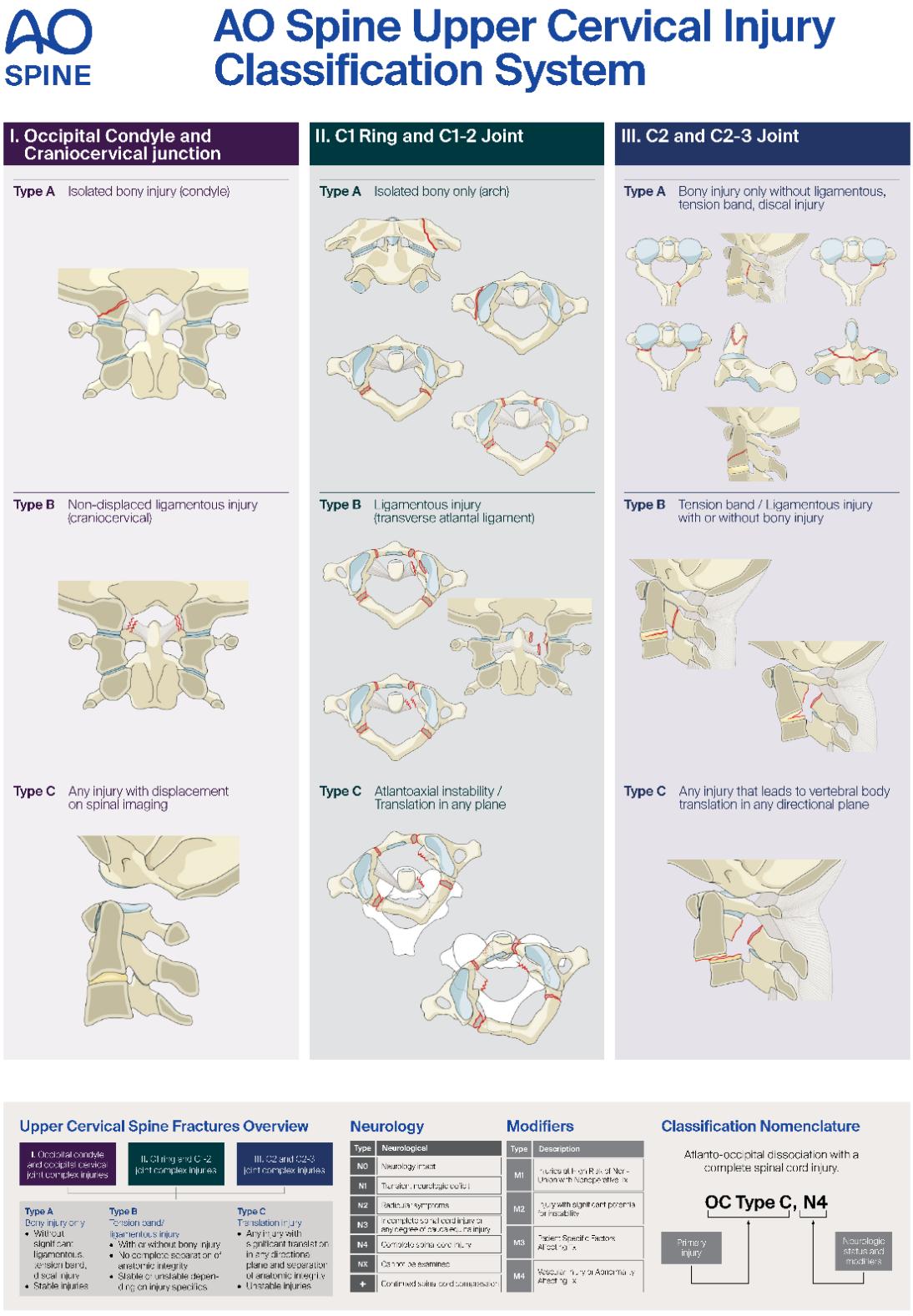
Patients with a combined score of 3 or less on the TLICS can be treated nonoperatively, but those with a total score ≥ 5 can be treated operatively. Those with a score of 4 can be treated conservatively or operatively, based on the treating physician's best judgment.

AO Spine Trauma Classification

The AO Spine Trauma Classification was developed to be comprehensive, easy to use, and address shortcomings of the SLIC and TLICS systems by including patient-specific characteristics.[8–11] Four separate classification systems for spinal trauma are defined, including the upper cervical spine (Occiput-C2), subaxial cervical spine (C3-7), thoracolumbar spine (T1-L5), and sacrum (S1-5, including coccyx) (refer to Figures 3-6). Detailed descriptions for each classification are available online at the AO Spine website.[12] Each classification evaluates the morphology of the injury, neurologic status, and patient-specific clinical modifiers. A surgical decision-making algorithm using the injury severity score exists to guide operative treatment.[8,13]

- For the upper cervical spine, injury morphology is divided into three categories: injuries to the occipital condyle and craniocervical junction, C1 ring and C1-2 joint, C2 and C2-3 joint.
- The subaxial cervical spine and the thoracolumbar classification both consist of 3 main categories (type A compression injuries, type B tension band injuries, and type C translation injuries) with the cervical classification containing two additional modifiers to describe bilateral (type BL) or facet injuries (type F).
- Sacral injuries are morphologically categorized into three main categories: lower sacro-coccygeal fractures (type A), posterior pelvic injuries (type B), and spino-pelvic injuries (type C).
- Neurologic injuries are described hierarchically, as follows:
  - N0 denotes patients that are neurologically intact,
  - N1 indicates a transient neurologic deficit,
  - N2 denotes a nerve root injury or radiculopathy,
  - N3 is an incomplete spinal cord injury or cauda equina injury, and
  - N4 indicates a complete spinal cord injury.
  - Nx is used when the neurological status is unknown (patient unable to be examined) and the plus sign (+) modifier is used to denote ongoing spinal cord compression.
- Patient-specific clinical modifiers were designed to represent heterogeneity within spinal trauma and are denoted with M.[10] These include variables that may significantly impact or change patient treatment (e.g., the presence of significant soft tissue damage, presence of a significant disc herniation in a cervical bilateral facet dislocation, or posterior tension band injury). Examples of patient facts that may affect treatment include the presence of significant medical comorbidities or bone metabolic disease such as ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, or osteoporosis.

Figure 3. AO Spine Upper Cervical Classification System



"The AO Spine Injury Classification Systems were developed and funded by AO Spine through the AO Spine Knowledge Forum Trauma, a focused group of international spine trauma experts. AO Spine is a clinical division of the AO Foundation, which is an independent medically-guided not-for-profit organization. Study support was provided directly through the AO Spine Research Department and AO ITC, Clinical Evidence."

Figure 4. AO Spine Cervical Subaxial Classification System

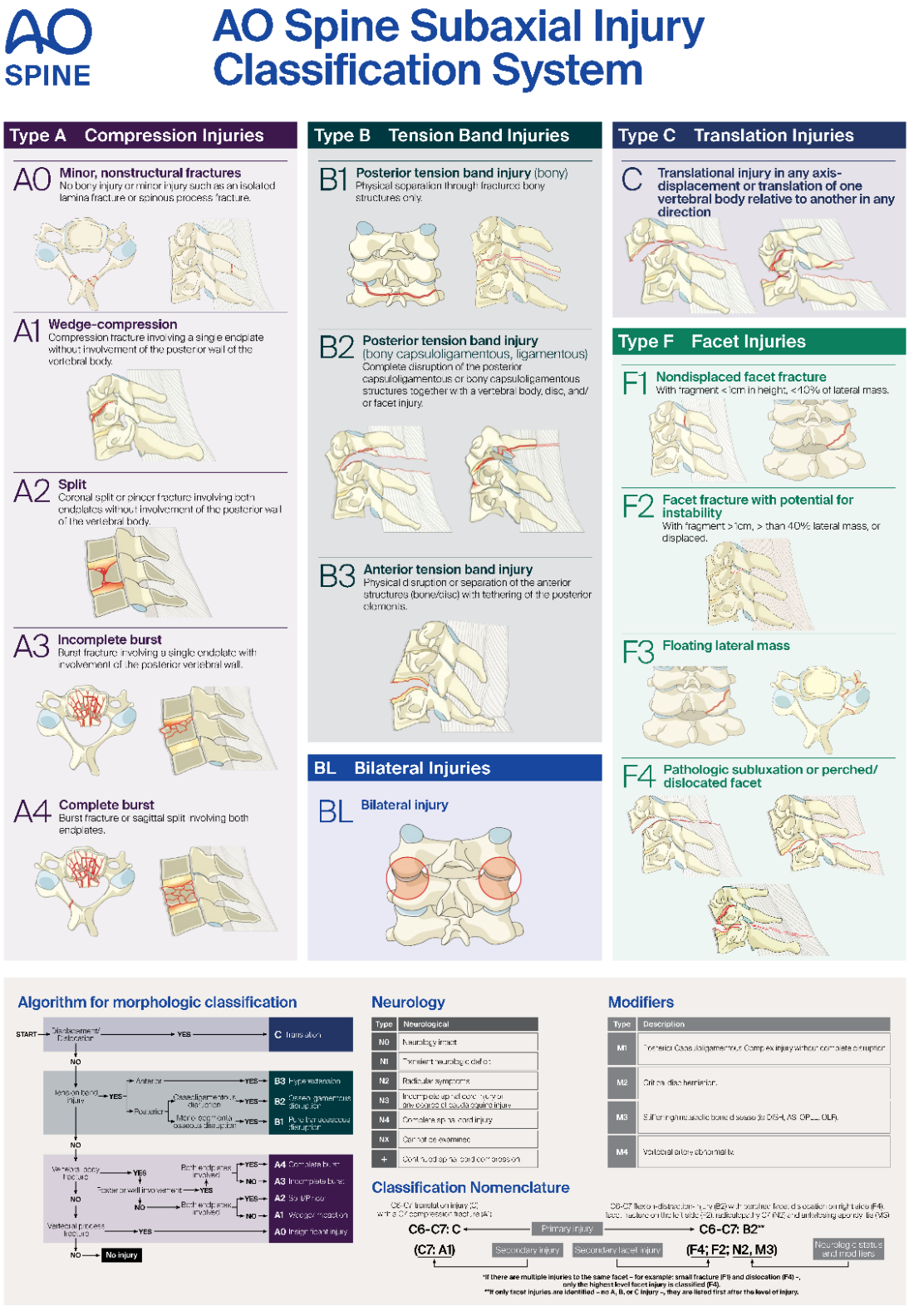


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Figure 5. AO Spine Thoracolumbar Classification System

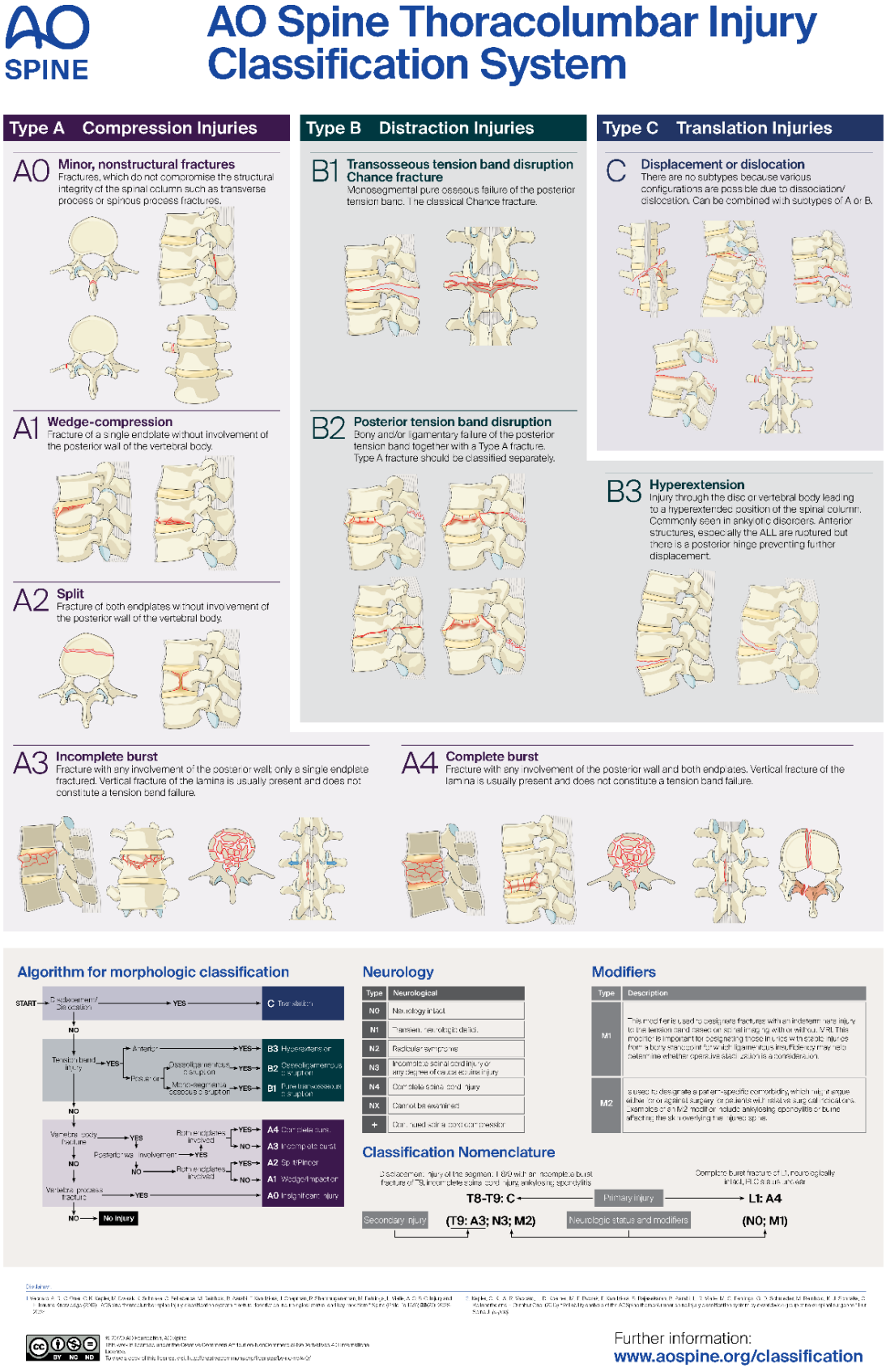

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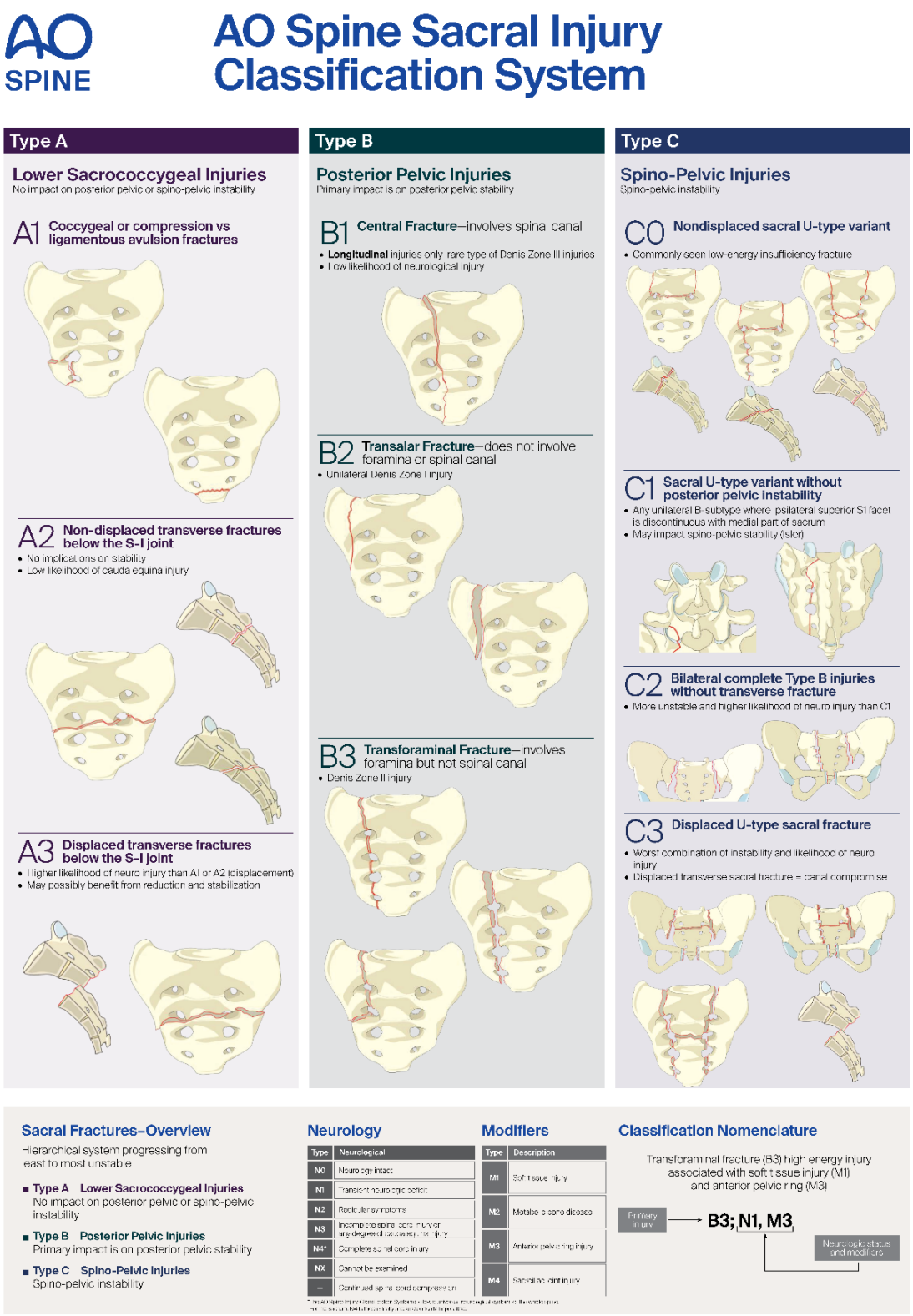
Figure 6. AO Spine Sacral Classification System



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SPINAL CORD INJURY CLASSIFICATION

KEY POINTS

- To accurately assign an ASIA impairment grade, complete the assessment after the period in which spinal shock may occur.
- After assessing sensation and motor function, the level of injury is defined as the lowest spinal segment with intact sensation and anti-gravity motor function preservation.

Anatomical Considerations

Acute SCI is classified descriptively, based on the level of injury and degree of function the patient exhibits on physical examination. Understanding the relevant functional anatomy and major subdivisions of the spinal cord is needed to correlate clinical findings with the location of the lesion.

The gray matter of the spinal cord is located centrally in a butterfly or H shape as seen on a cross section. The gray matter has paired dorsal horns posteriorly and paired ventral horns anteriorly. The dorsal horns primarily constitute a sensory zone, which receives afferent fibers from the dorsal roots of the spinal nerves. The ventral horns contain motor neurons and primarily constitute a motor zone. The white matter surrounding the gray matter is located in the periphery of the spinal cord. Its paired dorsal columns posteriorly are mostly a sensory zone. Its ventral columns anteriorly and lateral columns laterally include a mixture of sensory and motor zones. All columns include ascending and descending sensory and motor tracts or pathways.

Table 8 details the function of selected ascending (sensory or afferent) and descending (motor or efferent) white matter pathways.

Injury Classification

Injury to the cervical spinal cord results in *tetraplegia*, with impaired function in the upper and lower extremities. Injury to the thoracic or distal spinal cord results in *paraplegia*, in which function of the upper extremities is preserved.[1] Patients with an *incomplete* SCI have some residual function distal to the level of the injury, while those with *complete* SCI have permanent lack of function distal to the level of the injury.

Incomplete Spinal Cord Syndromes

Incomplete spinal cord syndromes refer to lesions involving certain structural and anatomic regions of the cord. They are usually associated with variable preservation of motor and sensory function below the level of injury, with no involvement of the head and neck. These lesions are often encountered following traumatic injuries, but they also result from other etiologies, e.g., tumors, infections, or systemic diseases. Incomplete SCI manifests with a constellation of physical impairments and can be categorized by several descriptive syndromes.

Central Cord Syndrome. Central cord syndrome is the most common incomplete spinal cord injury, typically occurring after a fall with hyperextension injury to the cervical spine.[2] Older adults with degenerative changes and spinal spondylosis are predisposed, and they are increasingly seen with this type of injury following a low

Table 8. Selected white matter ascending and descending tracts and their function

Direction	Tract	Column	Function
Ascending	Lateral Spinothalamic	Lateral	Pain, temperature
	Ventral Spinothalamic	Ventral	Pressure, crude touch
	Dorsal Column	Dorsal	Vibration, proprioception
Descending	Corticospinal (lateral and ventral)	Lateral, Ventral	Skilled motor activities
	Reticulospinal (lateral and ventral)	Lateral, Ventral	Regulation of voluntary movements and reflexes

velocity injury mechanism, such as a ground level fall.[3-5] In young patients, cord compression usually results from a fracture dislocation or herniation secondary to hyperflexion injury.[6] Clinically, this syndrome manifests with prominent weakness in the upper extremities and less weakness in the lower extremities. Tendon reflexes are lost at the level of the spinal cord lesion. Bladder symptoms are rare, and when present, usually include urinary retention. Pain and temperature sensations are preserved above and below the level of the lesion. However, with disruption of crossing spinothalamic fibers in the ventral commissure, pain and temperature sensations may be absent at several adjacent dermatomes, creating the "suspended sensory level".[7]

Anterior Cord Syndrome. This syndrome results from a vascular-type injury to the anterior spinal artery, the primary blood supply to the anterior spinal cord. The anterior two-thirds of the spinal cord is affected. This syndrome characteristically results in loss of motor function and pain and temperature sensation distal the level of the lesion; however, proprioception, vibratory sense, two-point discrimination, and fine touch are preserved.[8]

Posterior Cord Syndrome. This incomplete SCI, characterized by isolated loss of proprioception, is an extremely rare injury. Motor function, and all other sensory functions, are preserved. This pattern is due to the dorsal location of the proprioceptive tracts in the spinal cord.

Brown-Sequard Syndrome. This syndrome can be caused by sharp hemi-transection of the spinal cord from blunt traumatic injury and cervical epidural hematoma. It has the best prognosis of all incomplete SCIs. This syndrome manifests as ipsilateral loss of motor function at the level of injury, as well as loss of dorsal column function (proprioception, vibratory sensation). Contralateral loss of pain and temperature sensation also occurs, which is related to the decussation of the spinothalamic tracts, typically occurring one to two levels below the level of the injury.

Conus Medullaris Syndrome. Injury or compression to the distal aspect of the spinal cord, typically between T12 and L1 is the cause of this syndrome.[9] It manifests with bladder and bowel dysfunction, sexual dysfunction, loss of sensation around the perineum, and mild motor symptoms involving the lower extremities.[9]

Cauda Equina Syndrome. This syndrome's constellation of symptoms is related to an injury to the intrathecal nerve roots distal to the conus that comprise the cauda equina. Incomplete cauda equina syndrome presents with bilateral sciatica, variable sensory and motor deficits in the lower extremities, which may be asymmetric. Complete cauda equina syndrome has these symptoms plus urinary and/or bowel retention or incontinence.[9,10]

Acute Phase Conditions

Patients sustaining SCI often present with somatic dysfunction, including circulatory dysregulation from loss of sympathetic tone. *Neurogenic shock* is characterized by hypotension without compensatory tachycardia resulting from injury to the spinal cord's autonomic pathways that lead to collapse of resting sympathetic tone and systemic vascular resistance.[11] This constellation of findings can lead to end organ failure and death. Differentiate neurogenic shock from *spinal shock*, a loss of all spinal cord function and reflex activity.[12] Spinal shock is characterized by flaccid areflexic paralysis, including loss of the bulbocavernosus reflex. Typically, acute spinal shock resolves within 48 hours of injury. See section on Spinal Shock on page 52 for more information.

American Spinal Injury Association (ASIA) Classification[13]

Use the ASIA classification of SCI to stratify the severity of the injury. To accurately assign an ASIA impairment grade, complete the assessment after the period in which spinal shock may occur. Refer to Table 5 for the ASIA classification descriptions. Refer to the Physical Examination section and Figure 2 on page 19 for documentation of the ASIA assessment.

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NONOPERATIVE MANAGEMENT

KEY POINTS

- Occipital condyle fractures without neural compression or cranio-cervical misalignment can be managed successfully with a rigid or semi-rigid cervical orthosis.
- Select treatment for cervical fractures on an individual basis, based on fracture type and patient factors, including age.
- A best practice for stable thoracolumbar fractures without neurologic deficits is adequate pain control and early ambulation without a brace.

Occipital Fractures

Occipital condyle fractures (OCFs) are relatively uncommon, and they were rarely diagnosed until the widespread adoption of CT for trauma patients. OCFs were first classified by mechanism and fracture morphology: Type I (axial loading, comminuted, usually nondisplaced); Type II (direct trauma, linear, usually with concomitant skull base fractures); and Type III (rotation or lateral flexion, avulsion fragment, highest potential for ligamentous damage). Type I and II OCFs were deemed biomechanically stable, while Type III were potentially unstable.[1]

OCF reclassification incorporated ligamentous injury to better assess stability: Type I (nondisplaced fractures, either linear or comminuted); Type IIA (displaced fractures, without evidence of occipito-atlantal instability); and Type IIB (displaced fractures, with radiographic of ligamentous instability). Management guidance included: Type I OCFs do not require immobilization, Type IIA can be managed with a rigid cervical collar, and Type IIB require either halo immobilization or surgical fixation.[2]

Maserati et al. simplified these classification schemas by focusing on the criteria most essential in clinical decision-making. The presence of neural compression or cranio-cervical misalignment is an indication for surgical intervention. Other OCFs can be managed successfully with a rigid or semi-rigid cervical orthosis.[3] While a halo device can be considered for rigid external immobilization with similar outcomes,[4] particularly in the setting of bilateral OCFs, a cervical collar is recommended for improved patient comfort and compliance.[5] However, the presence of cervical fractures or instability, as well as other sequelae of polytrauma, may influence the management of OCFs.[6]

Cervical Fractures

Review the Spine Injury Classification section for the classifications and surgical indications for atlanto-axial and sub-axial cervical fractures. Similar to OCFs, management may be influenced by presence of other injuries. Without definitive evidence recommending any specific form of conservative management, treatment must be tailored individually, based on fracture type and patient factors.

Atlas. Stable C1 fractures can be successfully managed nonoperatively.[7,8] The utility of the various systems developed to classify atlas fractures lies in their guidance to assess the integrity of the transverse atlantal ligament, which is critical for C1 stability. Stable C1 fracture patterns include:

- Fractures of the anterior arch alone (e.g., Gehweiler Type I);
- Fractures of the posterior arch alone (e.g., Gehweiler Type II);
- Minimally displaced burst fractures of both the anterior and posterior arches (unilateral or bilateral), without radiographic evidence of transverse ligament disruption (e.g., Gehweiler Type IIIa);
- Minimally displaced fractures of the C1 lateral mass (e.g., Gehweiler Type IV); and
- Isolated fractures of the C1 transverse process (e.g., Gehweiler Type V).

These fractures are all best managed initially in a cervical collar.[8] Surgery is often indicated for transverse ligament disruption, usually with displacement of the lateral masses greater than 7 mm, or other evidence of atlanto-occipital or atlanto-axial instability.

Halo cervical traction for 6 to 12 weeks can be considered for unstable atlas fractures consisting of bony avulsion of the transverse ligament (e.g., Gehweiler Type IIIa).[9] Surgery with modern operative techniques is recommended to stabilize these fractures. These fractures may eventually heal in a halo-vest, but the halo-vest is less commonly used because of its known risk profile (e.g., dysphagia, skin complications, and respiratory issues), especially for geriatric patients.[8-10]

For cases in which the C1 lateral mass is fractured and displaced (e.g., Gehweiler Type IV), closed reduction and halo-vest treatment for 6 to 12 weeks may be an acceptable alternative, especially in young patients. If the lateral mass can be realigned with ligamentotaxis, the risks of atlanto-axial fusion can be avoided. However, these patients need to be monitored closely with follow-up imaging (e.g., at 3, 6, and 12 weeks), to evaluate for joint incongruence that would require surgical fixation, and for posttraumatic arthritis that could indicate collapse of the joint.[8,11]

Axis. Fractures of C2 can occur in the odontoid process or in the vertebral body itself. Most of the kinetic energy to C2 is absorbed by the dens, and a number of classifications exist to organize C2 fractures on this basis. It is acceptable to manage all types of odontoid fracture initially with a cervical collar, followed by close observation and radiographic imaging to monitor for nonunion or malalignment.[12,13] C2 vertebral body fractures (not Hangman's fractures) that do not involve the dens are less common,[14] but generally they are stable and best managed in a cervical collar. Shear fractures of the tip of the dens (e.g., Anderson D'Alonzo Type I) are usually stable, if they are not associated with occipito-cervical dislocation. A best practice is to manage these fractures in a cervical collar.[12,13] Similarly, odontoid fractures extending into the C2 body (e.g., Anderson D'Alonzo Type III) can also heal with cervical immobilization. Although higher rates of fusion were observed with rigid immobilization in a halo device, use of a cervical collar is acceptable if risk factors for nonunion are not present (e.g., significant angulation, displacement of the dens more than 5 mm, comminution of the odontoid fragment, and advanced age).[12,13]

Treatment options remain controversial for C2 fractures at the base of the dens, between the transverse ligament and the body of the axis (e.g., Anderson D'Alonzo Type II).[15] If these fractures do not meet criteria for operative stabilization, recommendations are:

- Initial management with external immobilization using a halo device in younger patients (less than 50 years old)
- A cervical collar in patients older than 75 years.

The rationale for this bimodal approach is based on the high rate of nonunion in younger patients, and the morbidity with halo fixation in older adult patients.[15,16] Studies report conflicting outcomes for both operative and nonoperative management of type II odontoid fractures in the older adult.[17-21] Nevertheless, fibrous nonunion without radiographic instability may be an acceptable endpoint for these fractures among older adult patients, and this can be achieved with a cervical collar.[17]

Most cases of traumatic spondylolistheses of C2 (Hangman's fractures) are managed operatively. However, isolated hairline fractures of the ring of the axis and those with minimal displacement (e.g., Effendi Type I & II) may be managed with external cervical immobilization, in either a cervical collar or a halo device depending on the severity of the fracture.[22]

Subaxial. Subaxial cervical spinal injuries can occur as compression failures of the anterior elements, tension band failures of the posterior elements, translation injuries, or facet injuries. Prior to considering nonoperative management, exclude indications for surgical intervention such as compression of neural elements or biomechanical instability.

In selected patients consider deferring surgical management of stable non-displaced fractures of all types (e.g., isolated spinous and transverse process fractures, unilateral non-displaced facet fracture, minimally displaced facet fractures, and unilateral facet dislocations in the subaxial cervical spine without evidence of spinal cord injury), and manage them with immobilization in a rigid cervical orthosis.[23-26] Observe and reassess these patients with dynamic cervical radiographs at 6 weeks (or a similar time-point).

Thoracolumbar Fractures

Similar to subaxial cervical spinal fractures, stable thoracolumbar fractures in patients without neurologic deficits can be safely managed nonoperatively, with acceptable outcomes in pain, employability, and residual deformity.[27-29] Prolonged bedrest is not indicated for these patients and a best practice involves adequate pain control and early ambulation without a brace.[30]

Limited high-quality evidence demonstrates that early mobilization without orthosis can lead to similar pain relief, quality of life, and functional outcome for up to 5-10 years, when compared with the use of thoracolumbar orthosis.[31-34] While not necessary, a brace can be prescribed for patient comfort, if desired.[35]

Sacral Fractures

Nonoperative management of sacral fractures, most commonly insufficiency fractures, consists of prolonged bedrest, analgesia, and progressive mobilization with weight-bearing restrictions as tolerated.[36,37] This strategy is most appropriate for isolated fractures that are not associated with unstable injuries to the pelvic ring, and for patients with minimal neurologic deficit who can tolerate prolonged immobility.

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PENETRATING SPINAL INJURY

KEY POINTS

- The vast majority of penetrating spinal cord level injuries result in complete (ASIA A) injuries.
- Few gunshot injuries of the spinal cord require surgical stabilization.
- Steroids are not recommended for penetrating spinal injury.

No standardized pathway currently exists for the management of ballistic penetrating spinal cord injury (pSI), despite the rising prevalence of urban, civilian gun violence. Gunshot wounds (GSWs) are the third most common cause of spinal trauma after motor vehicle crashes and falls.[1,2] The majority of civilian gunshot wounds are from relatively low muzzle velocity handguns. The mechanism for pSI is complex and involves higher energy transfer than occurs with blunt spinal injury. Thus, the overall prognosis for pSI is substantially worse than for blunt SCI.[1-11]

Management of pSI in urban trauma centers is complex. These victims routinely have polytrauma, including multiple penetrating wounds, that takes precedence. Mechanisms of pSI generally result in less mechanical instability than blunt trauma. Overall, the strength of available data is relatively low, and most studies have relatively short follow up.[12,13]

Initial Assessment

Initial assessment requires strict adherence to ATLS priorities. Radiographic and neurologic evaluation is often delayed as many patients are taken emergently to the OR for other injuries. When possible, perform a detailed neurologic exam and document it using ISNSCI criteria, including a rectal examination. Refer to Figure 2 on page 19. A CT scan of the neural axis, including vascular imaging for cervical injuries, is the primary imaging modality.[14]

Immobilization

Some GSW victims arrive by police drop-off with no standard SMR protocols. Most victims that arrive by ambulance have standard SMR precautions, including backboards and cervical collars. However, collars can be removed to deal with airway and hemorrhage issues in cases of cervical penetrating injuries. Cervical collars can be continued for pain or concerns of instability. However, the majority of GSW injuries at any level of the spine do not require SMR.[15-18]

Surgical Indications

The vast majority of cord level injuries from penetrating spinal trauma result in complete (ASIA A) injuries. Surgery does not appear to improve neurologic recovery for cervical and thoracic cord level injuries. Incomplete cord level injuries (ASIA B or above) are not common. Patients with these injuries can be considered for surgery if ongoing compression occurs, taking into account the higher risk of operative management that includes worsening neurologic status. In patients with neurologic deterioration and ongoing neural element compression, surgery may be indicated. While no high-quality data exists, neurologic improvement for incomplete cauda (lumbar) level injuries with ongoing compression from bullet/bone fragments in the canal is reported. Weigh the potential neurologic improvement against the considerable morbidity associated with surgical intervention.

The surgical indications for neurologic issues are limited, and few GSW injuries require surgical stabilization; however, surgical stabilization occurs most often with cervical level injuries. No consensus exists regarding the classification of GSW bony injuries, and most often the posterior ligamentous complex (PLC) is intact. Treating more severe cases with immobilization and serial imaging is a reasonable option, reserving surgical intervention for documented instability. Research does not support surgery to remove a bullet from the spinal canal for the following reasons: to prevent pain, to reduce infection risk even with transgression of a hollow viscus or airway, to prevent migration, or to prevent development of syrinx. Surgery may be indicated for

a persistent cerebrospinal fluid (CSF) leak.[19-27] Lead poisoning caused by retained lead bullet fragments is extremely rare. If concerned, monitor the patient's lead level for a year following the injury. If the lead level is rising, consider treatment with chelation agents or removal of the fragment, if technically feasible.[28]

Medical Management

Steroids are not indicated for pSI. Insufficient data exist to recommend blood pressure augmentation for pSI. With regard to antibiotic coverage, especially for projectiles crossing contaminated spaces, insufficient evidence exists to give guidelines, but the recommended range is from 2 to 10 days of antibiotic coverage.[29,30]

Role of MRI

The role of MRI imaging in GSW injuries to the spine remains unresolved. While the vast majority of civilian ordinance is MRI compatible, partial steel-jacketed ammunition is potentially an issue in the MRI. Radiologists are generally reluctant to allow MRI if the ballistic composition is unknown, especially when it is in proximity to vital structures. The one clinical scenario in which MRI is very useful occurs when the patient has a neurologic deficit without compression in the canal or a trajectory across the canal. MRI in these situations often shows concussive/blast like changes in the cord, and rules out other issues, such as epidural hematoma. Myelogram would be another option for these cases.[31-33]

Stab and Puncture Injuries to the Spinal Cord

In the U.S., stab injuries occur less frequently than ballistic injuries. Surgical intervention may be considered for the following indications: removal of a retained foreign body, infection and sepsis, acute CSF fistula, hematoma formation, cord compression from a bony fragment or soft tissue, progressive neurological deterioration, and persistent chronic CSF leakage. Implement local wound debridement, tetanus prophylaxis, and a short course of prophylactic antibiotics to prevent meningitis or local infection.[34]

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CONCOMITANT INJURIES AFFECTING TIMING OF SPINAL INTERVENTION

KEY POINTS

- Clinical judgment plays an important role in determining the optimal timing of spinal surgery in polytrauma patients to achieve early spinal stabilization while assuring the patient's hemodynamic stability.
- Resuscitation and positioning the patient on the operating room (OR) table must be planned to manage potential complications associated with other injuries.

Early spinal cord decompression and stabilization for blunt SCI patients is recommended;[1] however some delay in surgical stabilization may be required until cardiopulmonary and hemodynamic stabilization is achieved. It is essential to balance the need to optimize neurologic and systemic outcomes associated with early mobilization, while minimizing the morbidity and mortality associated with surgery on an inadequately resuscitated patient. Benefits of early mobilization include avoidance of pulmonary complications (e.g., adult respiratory distress syndrome), decubitus ulcers, contractures, and other complications. Regional anatomical considerations such as thoracic cage, aortic, cardiac, and pulmonary injuries, must be considered when treating patients with thoracic spine trauma. During discussions with trauma care providers, the intensivist, and the anesthesiologist review resuscitation endpoints to determine hemodynamic stability and readiness for surgery, e.g., base deficit, lactic acid levels, blood pressure, and volume status.

While numerous retrospective and prospective studies have examined cohorts of SCI patients regarding the relationship between various cut-offs for surgical timing and outcome, very few studies delve into the details of polytrauma. It is highly likely that the severity of other injuries and co-morbidities play a role in patient selection for and timing of surgery when these factors are not controlled. No systematic large trial of randomized polytrauma patients examining different surgical timing by either spinal cord or spinal column injury, level of SCI, or patterns of polytrauma/co-morbidities exists. As a result, clinical judgment plays an important role in determining the optimal timing of spinal surgery to effect surgical stabilization.

One study using ACS Trauma Quality Improvement Program (TQIP) data, suggested that for patients *without* SCI, the optimal timing for spine stabilization surgery with the lowest rate of major complications occurred at 21 hours post-injury, reflecting the time necessary to stabilize patients.[2] This study also reported that the risk of major complications increased after 24 hours. These results can be extrapolated to some degree for patients with SCI. While early decompression of the spinal cord is important, SCI patients may experience worsening hemodynamic instability related to the spinal cord injury itself. A national Japanese study demonstrated no change in length of stay or in-hospital mortality in isolated cervical SCI patients treated less than 24 hours or between 24 hours and 7 days.[3] A retrospective study of polytrauma patients with cervical or upper thoracic spinal column trauma concluded that major complication risk increased after 36 hours, even with logistic regression analyses accounting for factors including: generally older patients, higher body mass index (BMI), and lower likelihood of a spinal cord injury.[4] One prospective cohort study in SCI patients demonstrated no differences between ultra-early (< 4 hours) and early (< 24 hours) surgery,[5] consistent with the concept that it is important to take the time to ensure stabilization and proper pre-operative planning.

A secure airway assuring adequate ventilation and respiration and cardiopulmonary stability is essential especially for patients who will be positioned in the prone or lateral decubitus positions for surgical therapy. For approaches involving the anterior cervical spine, airway security is a must, because the prepping and draping procedures may limit access to the airway. If the patient has a tracheostomy, take care to maintain sterility of the anterior neck when prepping and draping for an anterior cervical spine approach. Recognize that the presence of pulmonary contusions may result in progressive ventilation and respiration difficulties during surgery. This is an important consideration for patient positioning and OR table configuration selections. Use OR tables that allow for better thoracic excursion when lung injury is present and the prone position is required.

Stabilization of circulating blood volume and blood pressure in the polytrauma patient with blood loss, or patients in neurogenic shock is also key to sustaining life, minimizing end-organ ischemia, and maintaining perfusion of the spinal cord to optimize outcome. Placing patients in the prone position to achieve posterior stabilization of the cervical, thoracic, or lumbosacral spine can increase risk for sudden decompensation and cardiac arrest upon turning. Thus, it is critical to ensure that the patient is stabilized with respect to heart rate and blood pressure. Also confirm no ongoing internal hemorrhage from solid organ or viscus injury, long bone fractures, or other injuries. While evidence is variable, it is generally well accepted that maintenance of spinal cord perfusion pressure is important, particularly early in the course by keeping mean arterial blood pressure at 85 to 90 mmHg to avoid hypotension (see below).[6]

Correction of coagulopathy must be performed pre-operatively, especially for those patients undergoing large open incisions and dissections in which estimated blood loss (EBL) is anticipated to be significant. Consider the use of cell saver technology to auto-transfuse operative blood loss, but do not plan for this to be the only mechanism of protection. Vigilance for ongoing and worsening coagulopathy in large surgeries from operative blood loss is also critical. If minimal EBL is anticipated, coagulopathy may be corrected pre-operatively and intraoperatively.

To avoid contamination, it is recommended that patients with open abdominal or viscus injuries be stabilized from a posterior approach. For posterior thoracolumbar or cervical stabilization approaches, take care when positioning these patients in the prone position to avoid compression of the abdomen. Carefully consider OR table configurations.

Patients with severe TBI or blunt vascular injury are at risk for cerebral or spinal cord ischemia. Operative timing must be tailored to the general risk of progressive intracranial hypertension and tissue infarction.

Further work is necessary to more closely define particular risk factors that should dictate the timing of spinal stabilization surgery in the polytrauma patient.

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BEST PRACTICES GUIDELINES
SPINE INJURY

CARE OF THE SPINAL CORD INJURED PATIENT

NEUROGENIC SHOCK AND SYSTEMIC PRESSURE-DIRECTED THERAPY

KEY POINTS

- Injuries to the cervical and high thoracic spine cause vasoplegia and neurogenic shock due to a loss of sympathetic tone.
- Avoid hypotension in patients with SCI. Weigh the decision to use mean arterial pressure (MAP) goals of 85-90 mmHg for 7 days against the limitations of data, and the risk for utilizing vasopressors, prolonged immobilization, need for invasive monitoring, and the consumption of limited critical care resources.
- An agent with both alpha- and beta-adrenergic activity is recommended to treat both the hypotension and bradycardia associated with symptomatic denervation.

The spinal cord is an end organ susceptible to secondary injury from hypoperfusion in the setting of trauma. Injury to the cervical and high thoracic cord result in sympathetic dysfunction with subsequent bradycardia and hypotension—neurogenic shock—putting the cord at further risk for hypoperfusion injury.

In addition, the current recommendation is to maintain the MAP between 85 to 90 mmHg for a total of seven days after injury. This recommendation is based on two prospective cohort studies (Class III data) looking at a total of 127 patients with both cervical and thoracic injuries having the expected distribution of complete and incomplete injuries. These two studies and subsequent low-level studies demonstrate variable correlation. Additionally, only one time point (7 days) was used in both prospective studies.[1-5]

Reasonable agreement to avoid hypotension in the setting of SCI also comes from animal and clinical experience; however, the definition of hypotension is variable.[6] Some report a blood pressure of 90/60 mmHg as the threshold for relative hypotension, which corresponds to a calculated MAP of 70 mmHg. The reported upper limit of normal blood pressure for younger patients is 120/80 mmHg, which corresponds to a calculated MAP of 93 mmHg. Therefore, it can be argued that MAP targets of 85-90 mmHg represent normotension not augmentation. At least five clinical trials are ongoing or recently completed focused on MAP-directed therapy and/or spinal cord perfusion pressure (SCPP). Measurement of SCPP is based on lumbar drains or pressure monitoring catheters (SCPP = MAP minus intrathecal pressure). These study findings are not yet widely distributed.[7,8]

Avoid hypotension in SCI. The decision to use MAP goals of 85-90 mmHg for 7 days has to be weighed against the limitations of the data, and the risks for vasopressor use, prolonged immobilization, invasive monitoring, and prolonged consumption of limited critical care resourses.[9] The risks of using lumbar drains and/or intrathecal catheters to lower the intrathecal pressure and increase perfusion pressure across the neuraxis include CSF leak, infection, and catheter placement complications.

Treatment of hypotension due to neurogenic shock following SCI initially includes volume resuscitation, taking care to avoid volume overload. This is followed by vasopressors as needed. No consensus exists regarding the best vasoactive agent; however, an agent with both alpha- and beta-adrenergic activity is recommended to treat both hypotension and bradycardia associated with sympathetic denervation.[10-12]

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PHARMACOLOGIC MANAGEMENT OF SPINAL CORD INJURY

KEY POINTS

- The use of methylprednisolone within 8 hours following SCI cannot be definitively recommended.
- No other potential therapeutic agents have yet demonstrated efficacy for motor recovery and neuroprotection.

To date, no definitively proven successful pharmacologic therapy is available to mitigate SCI.[1] The trials for the National Spinal Cord Injury Studies (NASCIS) were conducted in the 1980's and 1990's.[2,3,4,5] These studies identified a subgroup of patients who demonstrated better motor scores after receiving methylprednisolone within 8 hours of injury compared with placebo. This was further supported by a Cochrane review.[6] Unfortunately, studies also demonstrated side effects that included infection, gastrointestinal bleeding, hyperglycemia and death.[7]

National and international associations have split on their recommendations for the use of methylprednisolone. The use of methylprednisolone within 8 hours following SCI cannot be definitively recommended. Its use needs to be balanced against the known potential complications on an individual basis.[8]

Studies of other potential therapeutic agents, including GM1 ganglioside, minocycline, thyrotropin-releasing hormone, nimodipin, gacylidine (GK-11), riluzole, and granulocyte colony stimulating factor, among others, have either failed to demonstrate efficacy for motor recovery and neuroprotection, or are being studied in ongoing clinical trials.[8,9,10,]

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VENOUS THROMBOEMBOLISM PROPHYLAXIS

KEY POINTS

- Initiate chemoprophylaxis as early as medically possible, typically within 72 hours of injury, to reduce the risk of venous thromboembolism (VTE).
- Determine the duration of chemoprophylaxis on an individual patient basis considering injury severity, mobility status, bleeding risk, and other co-morbidities.
- Surveillance duplex ultrasound for VTE in asymptomatic patients is not recommended, but it can be considered in high-risk patients who cannot have chemoprophylaxis during the acute period.

SCI patients have an elevated risk of developing VTE, with a typical incidence ranging between 40 percent to 70 percent.[1-9] The risk of both deep vein thrombosis (DVT) and pulmonary embolism (PE) in SCI patients increases with age, concomitant long-bone injuries, personal history of prior VTE, and higher degrees of spinal cord injury (e.g., ASIA A), among others.[1-11] The risk is highest during the acute period, with most VTE typically developing between 72 hours and 2 weeks of injury, and the risk tapers at 3 months post-injury. Patients with SCI also have a higher risk for chronic VTE for at least 1-year post-injury, and this is associated with a 3 to 9 percent mortality rate due to PE alone.[1, 6-16]

No standard guidelines currently exist for chemoprophylaxis initiation in SCI patients. Numerous studies report patients with SCI have a significantly higher rate of developing symptomatic VTE when not started on mechanical prophylaxis or chemoprophylaxis in the acute period of injury.[1-16] A best practice is to start chemoprophylaxis as early as medically possible, typically within 72 hours of injury, to reduce the risk of VTE. Initiate mechanical prophylaxis (e.g., sequential or pneumatic compression devices and compression stockings) immediately after the injury, if able, especially for patients with bleeding risk or other contraindications for chemoprophylaxis. Evaluate patients on a case-by-case basis regarding initiation of these therapies, taking into account the risks associated with them, such as bleeding or lower extremity fractures.

No one chemoprophylaxis agent is definitively superior for SCI patients; however, a recent meta-analysis found that low molecular weight heparin (LMWH) was better than unfractionated heparin for DVT and VTE prevention in adult trauma patients.[17] Low molecular weight heparin (LMWH) is the most extensively studied, and it safely reduces VTE incidence without significantly increasing bleeding complications.[18-22] For patients with traumatic SCI requiring surgical intervention, no significant increased post-operative complications occurred when LMWH was initiated within the acute period after surgery.[12-16,18,21-24] However, full anticoagulation is reported to increase the bleeding risk acutely after injury, and no benefit was reported over prophylactic dosing.[21-25] Recent studies suggested increased efficacy when using direct oral anticoagulants (DOACs), especially in orthopaedic populations or for prophylaxis in rehabilitation facilities; however, insufficient data in SCI populations exist at this time to warrant further recommendations.[26-27]

Insufficient evidence exists to support a standard duration of treatment. Typically, courses are continued during SCI rehabilitation placement, but often do not extend past the three-month interval, regardless of motor function. The risk of VTE trends toward the general population average risk the further a patient is from the initial injury.[11-16, 27-28] It is recommended that providers determine the duration of chemoprophylaxis on an individual patient basis considering injury severity, mobility status, bleeding risk, and other co-morbidities.

Surveillance duplex ultrasound for VTE in asymptomatic patients is currently not recommended. It is neither sensitive nor specific in this population, and no evidence exists that it decreases the rates, morbidity, or mortality of VTE in asymptomatic SCI patients.[27-30] Studies using surveillance ultrasound screening upon patient admission to rehabilitation facilities detected DVTs in 6 to 30 percent of SCI patients, however, most of these studies had either low rates of chemoprophylaxis adherence or excluded patients receiving chemoprophylaxis.[12, 21-29] For high risk patients who cannot have chemoprophylaxis initiated within 72 hours, consider using duplex imaging to exclude DVTs until adequate therapy can begin.[27-30]

Prophylactic placement of inferior vena cava (IVC) filters has no identified benefit. There is no evidence that IVC filters decrease the rate of PEs in SCI patients, and IVC filter placement has its own procedural risk.[30-33] Alternatively, consider temporary IVC filter placement in high-risk patients with known DVTs who cannot be started on anticoagulation, until definitive treatment can be safely initiated.

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SPINAL SHOCK

KEY POINTS

- Spinal shock is a total or near-total areflexia with the complete loss or suppression of motor function and sensation distal to the anatomical lesion.
- Spinal shock can persist from days to weeks, and it can be prolonged due to toxic or septic syndromes.
- The end of spinal shock for most patients is seen with the early return of the deep plantar reflex and with the bulbocavernosus, cremasteric, ankle jerk, Babinski sign, and knee jerk recovering in a progressive order.

Spinal shock refers to the sudden and transient depression of neural function below the level of an acute spinal cord lesion after injury.[1] The term is often a source of confusion for providers, frequently applied incorrectly to describe hypovolemic shock and/or neurogenic shock, leading to the risk of patient mismanagement. Spinal shock causes a temporary or permanent, complete or near-complete segmental interruption of neurotransmission. It is seen as a total or near-total areflexia, as well as the complete loss or suppression of motor function and sensation distal to the anatomical lesion.[2] Assumptions on the extent of injury based on physical exam during spinal shock are unreliable. Do not delay emergent, decompressive interventions.

Spinal shock following SCI is associated with a high energy fracture-dislocation, ligamentous injury, and rotational distraction. It is most common in men (80 percent) and the young (average age 29 years).[3] Major mechanisms of injury associated with spinal shock include motor vehicle crashes (45 percent), and SCI associated with domestic injuries or falls (34 percent).[3] Controversy exists regarding the association of spinal shock with patient prognosis; however, the magnitude of spinal shock is proportional to the severity and rapidity of anatomic injury.[4]

- The temporal evolution of SCI with spinal shock is well described from animal model studies.[4]
- Initially hemorrhage and protein extravasation are seen at the spinal level of direct injury.
- By four hours, central hemorrhagic necrosis is present in the entire central gray matter and the adjacent white matter.
- By 24 hours, the central gray matter and most of the white matter are necrotic.
- Edema of the spinal cord peaks by day three to six, and it may persist for up to two weeks.
- Two months following injury, only the outer rim of white matter is present.
- By one to two years, cavitary healing occurs with the resorption of necrotic debris and autolysis due to lysosomal accumulation.

For less severe injuries, the surrounding white matter, with associated long neuronal tracts, may be preserved, potentially leading to an incomplete injury pattern. Rapidly enacted interventions for preservation (e.g., decompression and stabilization) are needed to avoid secondary injury related to hypoxemia or hypotension.

The loss of reflex function is common at the spinal cord level associated with direct injury. However, the more distal the origin of the cord reflex is from the site of injury, the higher the likelihood for preservation. Some patients with high cervical cord injuries are reported to retain distal sacral reflexes, specifically the bulbocavernosus and the anal wink.[4] Do not confuse these spared reflexes with the sacral sparing associated with a partial SCI. Patients with spinal shock and a complete SCI lack sacral sensation. Reflexes immediately cranial to the injury may also become depressed because of the loss of ascending, distal cord influence.

Spinal shock can persist from days to weeks, and it can be prolonged due to toxic or septic syndromes. During the recovery period, neuronal healing, collateralization, and reorganization may occur. With the SCI-related lack of supraspinal inhibition, the re-innervation of posterior root axons can lead to spastic muscle spindle reflexes.[4] Reflexive muscle spasticity does not indicate the end of spinal shock, it indicates the reorganization of local reflex arcs.

A lack of consensus exists to define the end of spinal shock. Providers may interpret the end of spinal shock from one of the following:[5]

- The appearance of the bulbocavernosus reflex, occurring within several days of injury,
- The return of deep tendon reflexes which can take several weeks,
- The return of reflexive bladder function.

Common to most patients is the early return of the deep plantar reflex with the bulbocavernosus, cremasteric, ankle jerk, Babinski sign, and knee jerk recovering in a progressive order. Cutaneous reflexes often recover before deep tendon reflexes, rather than a caudal to cranial presentation favored by some authors.[5] During the 3 to 6 week period following injury, most patients see significant bladder and vasovagal response recovery. However, spinal cord areas with permanent damage may never have the return of their associated reflexes. As patients progress, optimizing the presence or absence of reflex arcs becomes one focus for rehabilitation services.

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SPINAL CORD INJURY-INDUCED BRADYCARDIA

KEY POINTS

- Sinus bradycardia is the most common dysrhythmia occurring during the acute phase following spinal cord injury.
- Cardiovascular instability is often precipitated by suctioning, turning, and hypoxia.
- Treatment of persistent bradycardia or intermittent episodes of severe bradycardia may include a beta-2 adrenergic agonist (albuterol), chronotropic agents (atropine, epinephrine, dopamine, norepinephrine), or phosphodiesterase inhibitors (aminophylline, theophylline).

Cardiovascular abnormalities commonly occur during the acute stage of SCI at level T-6 or higher. Hypotension (both supine and orthostatic), cardiac dysrhythmias (primarily bradycardia) and autonomic dysreflexia are due to the SCI-associated disequilibrium between the sympathetic and parasympathetic nervous systems. Cardiac sympathetic preganglionic neurons exit the spinal cord from T1 to T6 while parasympathetic neurons reach the heart via the recurrent laryngeal and vagus nerves.[1,2,3] The SCI-associated disruption in descendent sympathetic tracts results in unopposed parasympathetic tone.[4-8] The patient's inability to vasoconstrict the vascular beds in the viscera and extremities leads to blood pooling, impaired venous return, and low cardiac output resulting in hypotension. The degree of sympathetic cardiovascular dysfunction is directly related to the location and severity of SCI. A post-mortem examination of human spinal cord tissue revealed that individuals with more severe hypotension, bradycardia, and autonomic dysreflexia in the acute stage following injury had more extensive areas of degeneration within the spinal cord white matter.[9]

Loss of sympathetic control can lead to a low resting blood pressure, orthostatic hypotension, loss of diurnal fluctuation of blood pressure, and increased susceptibility to cardiac dysrhythmias. Sinus bradycardia is the most common dysrhythmia in the acute stage following SCI; however, other cardiac irregularities are attributed to SCI, including repolarization changes, atrioventricular blocks, supraventricular tachycardia, ventricular tachycardia, and primary cardiac arrest.[10-13]

Cardiovascular instability is often precipitated by tracheal stimulation (suctioning), turning, and hypoxia. Most cardiovascular changes are observed in the acute phase, ranging from immediately after injury to 6 weeks post injury.[9,12,13]

Initial management of SCI must ensure adequate blood pressure with fluid resuscitation and the use of vasopressors with both alpha- and beta-adrenergic actions (norepinephrine). Persistent bradycardia or intermittent episodes of severe bradycardia can be treated with a beta-2 adrenergic agonist (albuterol), chronotropic agents (atropine, epinephrine, dopamine, norepinephrine) or phosphodiesterase inhibitors (aminophylline, theophylline).[14-17] Rarely, patients with medication-resistant bradycardia may benefit from pacemaker insertion; however, this is associated with a 4 to 5 percent device complication rate.[18-20] In a retrospective study of patients with cervical SCI-induced bradycardia, enteral albuterol reduced the frequency of symptomatic bradycardia and resulted in less rescue therapy using chronotropic agents or need for pacemaker insertion.[14] Enteral albuterol has a rapid onset of action (30 minutes), and peak plasma concentrations are achieved within 2 hours. Duration is 4 to 6 hours with a half-life of 2.7 to 6 hours.[14] For treatment of orthostatic hypotension, the alpha-adrenergic receptor antagonist, midodrine, is recommended.[21,22]

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VENTILATOR MANAGEMENT IN HIGH SPINAL CORD INJURY

KEY POINTS

- Early tracheostomy is recommended to aid in mechanical ventilation during the acute and more chronic phases of care for patients with SCI.
- Consider stimulation of the diaphragm in high-SCI patients in order to plan long-term ventilator strategies and determine a patient's potential to wean from the ventilator.

Care of patients with high SCI (at or above C4) is complicated by the following physiologic changes: decreased or absent respiratory drive, low pulmonary volumes, weak or absent cough, weak and uncoordinated respiratory muscle function (including diaphragm), and chest wall rigidity. Additionally, global immobility leads to potential dysfunction related to pulmonary emboli. Patients with injuries at lower C-Spine levels may also have significant respiratory dysfunction due to loss of chest wall innervation and poor or absent cough. This constellation of problems makes weaning from the ventilator very difficult without innovative strategies. Complicating patient management further is autonomic dysfunction, resulting in increased secretions, bronchospasm, pulmonary edema, and disordered breathing during sleep.[1] The propensity for these issues to contribute to atelectasis and pneumonia can further complicate ventilator management. Early tracheostomy is recommended to aid in mechanical ventilation during the acute and more chronic phases of care (Refer to section on Placement of Tracheostomy Following Cervical Stabilization on page 58).

The main goals of patient management include restoration and maintenance of lung volumes that cannot be achieved spontaneously. These goals can be accomplished in a variety of ways, e.g., continuous positive airway pressure (CPAP), intermittent positive pressure breathing (IPPB) generally later in the course, or specific ventilator modes such as airway pressure release ventilation (APRV) in cases of severe atelectasis. Later in the course, the use of assisted coughing, insufflation, breath stacking, and glossopharyngeal breathing can be used, many of which can be achieved non-invasively.[1] Additional therapeutics potentially beneficial include mucolytics and beta agonists that help to attenuate the reflexive bronchoconstriction and increased mucous production in this patient population.

Approaches by Cervical Injury Level

Injuries at the C1 to C3 level cause diaphragmatic paralysis, generally resulting in permanent ventilator dependence. However, some patients are able to use self-ventilatory techniques for brief periods.

Patients with C3 to C4 injuries experience diaphragmatic dysfunction, but they may have partial ventilatory function. However, these patients have reduced tidal volumes and vital capacity. Longer periods off of mechanical ventilation may be possible, and some patients can achieve mechanical ventilation only at night. These patients also have the potential for non-invasive ventilatory techniques at home, especially while upright.

Regardless of injury level, techniques to help SCI patients become ventilator-free for at least some time include therapies such as optimization of pulmonary toilet and medications, high volume ventilation, and non-invasive ventilation.[2] Diaphragm pacing is beneficial for the patient in these ways:

- Helps improve respiratory mechanics (specifically spontaneous tidal volume),[3]
- Reduces weaning time and achieves independence from the ventilator,[3] and
- Potentially decreases hospital cost and length of stay.[4,5]

Consider stimulation of the diaphragm in the high-SCI patient to plan long-term ventilator strategies. An inability to stimulate the diaphragm supports a future inability to wean from ventilation.[6]

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PLACEMENT OF TRACHEOSTOMY FOLLOWING CERVICAL STABILIZATION

KEY POINTS

- Tracheostomy can be performed early after anterior cervical spinal stabilization without increasing the risk of infection or other wound complications.
- Open and percutaneous tracheostomy are both safe techniques.

Early tracheostomy after cervical SCI improves outcomes, reduces morbidity and mortality, and improves hospital length of stay,[1-7] especially with higher cord levels when prolonged ventilation is anticipated.[8] Published reports variably define early tracheostomy as less than seven days, less than four days, and 1 to 2 days post-injury. Admission ASIA motor score can be used to predict the need for subsequent tracheostomy.[9]

Anterior cervical stabilization is often required for unstable spinal column injuries and injuries that result in anterior spinal cord compression. Of concern is the proximity of the required incision for spinal stabilization to that of a midline tracheostomy incision. The potential for wound infection or dehiscence of the stabilization incision is particularly worrisome with secretions from tracheostomy site potentially contaminating the incision. The potential complication of esophageal injury during anterior spinal procedures has implications for deep infection impacting the tracheal tissues as well.

Evidence exists that early tracheostomy can be performed safely,[10,11] after anterior cervical spinal stabilization without significantly increasing the risk of infection or other wound complications.[10,12-17] Safe techniques include both open and percutaneous[18] tracheostomy. Consider performing tracheostomy early (within days) of SCI, even in the setting of an anterior cervical spinal stabilization procedure. Additionally, do not alter the standard of care for airway management when timing the cervical surgery.

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ANALGESIA IN SPINAL CORD INJURY

Refer to the *ACS TQIP Best Practices Guidelines for Acute Pain Management in Trauma Patients*

KEY POINTS

- Pain management is a priority in the care of the acutely injured SCI patient to relieve suffering and to prevent dysautonomia symptoms triggered by pain.
- Implement a multimodal approach for the acute pain management of patients with SCI.

Patients with SCI experience various types of pain including nocioceptive somatic, nocioceptive visceral, and neuropathic pain.[1,2] Patients experience pain above, at, and below the level of the lesion, and it is often experienced in areas where sensation is altered or absent. Both hyperesthesia and allodynia (pain experienced in response to non-painful stimuli) symptoms are described.[1] Nociceptive somatic pain may be caused by spinal column injury from destruction and damage to bone, muscle, tendon, and other soft tissues, expansile hematoma, and other local tissue trauma.

Following hemodynamic stabilization, the provision of comfort and pain management becomes a priority in the care of the acutely injured SCI patient. In addition to suffering from the pain, dysautonomia symptoms can occur in response to pain in the SCI patient.

Self-reported pain assessment of the SCI patient with critical injuries can be challenging because of paralysis that impedes gestures and the presence of intubation or tracheostomy that impedes speech. Pain assessment tools recommended for the non-verbal patient in the ICU include both the Critical Care Pain Observation Tool and the Behavioral Pain Score.[3,4] However, both of these tools rely on observation of movement or muscle tension which cannot be used when motor, muscle tone, and/or sensory function are altered. Health care providers need to rely on other assessment parameters such as facial grimacing, blinking in response to questions, or patient proxy (family) report of subtle communication methods. Vital signs remain unreliable indicators for pain assessment, especially in the setting of cardiovascular autonomic dysfunction.

When the patient is able to self-report, perform a comprehensive pain assessment using measures that address neuropathic pain.[5]

It is important to perform a home medication reconciliation at the earliest opportunity after admission, especially for patients with pre-existing psychiatric/psychological disorders. It is essential to assess for a history of substance abuse (drugs and alcohol), as well as pre-morbid chronic pain. Consider consulting with chaplaincy or mental health professionals early in the patient's hospitalization to help the patient cope with depression and loss.[6]

The multimodal approach to pain management is recommended in the acute pain management of SCI patients. Initial pain regimens may include a combination of opiates, acetaminophen, or non-steroidal anti-inflammatory agents.[1,7] Although the literature is mixed regarding the impact of NSAID use on union/nonunion, there is support for its use in the acute post-operative phase. Chronic long-term use may have detrimental effects on bone healing.[8] However, several pre-clinical studies in animal models report that NSAID use may be protective against inflammatory processes after SCI.[9]

For neuropathic pain, anticonvulsants (gabapentinoids, such as gabapentin and pregabalin) and antidepressants (tricyclic antidepressants, such as amitriptyline or novel antidepressants) are recommended over other modalities.[1,2] Knowledge of the side effects and prescribing nuances of these medications is critical. Longer-term management of neuropathic pain may include novel strategies such as spinal cord stimulation.[10]

It is essential to remember that the inability to perceive sensation and pain may result in occult problems (e.g., intestinal ischemia, cholecystitis, bladder spasm, etc.). Maintain vigilance in the examination of the abdomen, and aggressively work-up unexplained signs of infection

and potential causes of dysautonomia.

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AVOIDANCE OF ASSOCIATED SYMPTOMS OF SPINAL CORD INJURY

KEY POINTS

- Treat acute autonomic dysreflexia by sitting the patient upright, removing tight-fitting garments, correcting the inciting stimulus, and if needed, administering quick onset, short-acting antihypertensives to reduce blood pressure.
- Spasticity is managed with physical therapy, and in some cases, anti-spasticity medications.
- Use clinical judgment and a validated assessment tool to assess skin breakdown risks, and prevent decubitus ulcers by avoiding known modifiable risk factors such as pressure, shear force, and moisture to the skin.

Injury to the spinal cord can generate a number of medical complications, including dysautonomia, muscle spasm and spasticity, and decubitus ulcer or pressure injury. While these complications may first occur during acute hospitalization or rehabilitation, they often remain problematic for the patient throughout their life. Prevention, early recognition, and timely treatment of these complications are essential to optimize patient outcomes. Upon hospital or rehabilitation discharge educate patients and their caregivers on the cause, prevention, and appropriate interventions when one of these potential complications occur.

Autonomic Dysreflexia

Autonomic dysreflexia (AD) or autonomic hyperreflexia can occur in both complete and incomplete SCI, usually above level T6.[1] The condition consists of an uninhibited sympathetic response to a precipitating physiologic stimulus below the level of the spinal cord lesion, e.g., bladder distension, pressure sores, occult fractures, or bowel impaction. Sympathetic hyperactivity causes vasoconstriction below the spinal cord lesion, leading to a dramatic rise in blood pressure, followed by a compensatory parasympathetic response that causes vasodilation above the injury with or without bradycardia.[2,3] AD manifests most commonly as headache, diaphoresis, flushing, anxiety, nausea, and nasal congestion.[4] If not treated, hypertension can cause potentially life-threatening stroke, seizures, myocardial infarction, or pulmonary edema.[5] Treatment of acute AD consists of reducing the blood pressure by sitting the patient upright, followed by removal of tight-fitting garments, and correction of the inciting stimulus (most commonly fecal impaction or an obstructed indwelling urinary catheter). If hypertension persists despite nonpharmacologic interventions, administer a short-acting antihypertensive (e.g., nifedipine, captopril) with quick onset.[3] Prevention of AD is focused on avoiding potential precipitants.

Spasticity

Spasticity is an upper motor neuron syndrome that occurs after SCI, resulting in hyperexcitability of the tonic stretch reflex, which manifests as increased muscle tone in response to passive stretch.[6] Spasticity can worsen quality of life by impairing activities of daily living and causing pain, immobility, and muscle spasms.[7] It is important to note that symptoms of spasticity may also be beneficial by enhancing stability for sitting, standing, dressing, and transfers. Management must carefully strike a balance to optimize functional outcome.[6] With the combination of immobility and spasticity, muscles resting in a shortened position for prolonged periods can form contractures, due to reorganization of the collagen matrix.[8,9]

Physical therapy is a mainstay of treatment. Strengthening exercises, postural management, manual stretching, and orthoses are treatments used to maintain range of motion and prevent contractures.[10]

Although rehabilitation is the first-line management of spasticity, anti-spasticity medications are often prescribed to patients with SCI, despite limited efficacy in clinical trials. Consider pharmacologic agents as an adjunct in patients with debilitating spasticity, despite physical therapy interventions. Patients have an individualized response to each medication, so the ideal regimen is often determined empirically. Baclofen, an agonist in the GABAergic system, is the most commonly used agent. If effective, it is administered orally or injected intrathecally long-term using an implantable infusion pump. Baclofen reportedly helps most with flexor spasms.[6] Monitor patients with implanted pumps for signs of withdrawal due to pump failure, a clinical emergency.

Other agents include tizanidine, an alpha-adrenergic agonist; benzodiazepines (e.g., diazepam, clonazepam); and dantrolene, which inhibits calcium release in the muscle itself.[11] None have demonstrated significant efficacy in terms of improvement in functional measures or ability to perform activities of daily living (ADLs). Tizanidine can reduce hypertonia, diazepam can improve hyperactive reflexes and painful spasms, and dantrolene can augment range of motion.[6]

Refer to Table 9 for oral pharmacologic agents used for treatment of spasticity after SCI. Other studied pharmacologic options include cannabinoids, injection of botulinum toxin, and chemical neurolysis/denervation of peripheral nerves.[10]

Table 9. Oral pharmacologic agents for treatment of post-spinal cord injury spasticity

Drug	Mechanism of Action	Dosing	Considerations
Baclofen[12-17]	Not fully elucidated. Centrally acting gamma-aminobutyric acid (GABA) analog that inhibits monosynaptic and polysynaptic reflexes at the spinal level; action at supra-spinal sites may also occur	Dosage range: 5 to 20 mg per dose given 2 to 4 times daily (MAX 80 mg/day)	▪ Reduces flexor tone, and frequency and severity of flexor or extensor spasm ▪ Effects seen in cervical or thoracic injuries, including complete spinal transections ▪ Titrate slowly on initiation and discontinuation to avoid excess central nervous system (CNS) depression or withdrawal. ▪ Use reduced dose and/or frequency in patients with renal impairment as renal elimination predominates ▪ Moderate anticholinergic burden
Diazepam[16,18,19]	Benzodiazepine that exerts anxiolytic, anticonvulsant, sedative, muscle-relaxant, and amnestic effects through potentiation of GABA-mediated CNS inhibition	Dosage range: 2 to 10 mg, given 3 or 4 times daily (MAX dose not clearly established; based on effect tolerance)	▪ Reduces tone and spasms, but possibly less effective than baclofen for flexor spasms ▪ Titrate slowly on initiation and discontinuation to reduce risk of excess CNS depression or withdrawal ▪ CNS depressant effects ▪ Metabolized in the liver to active metabolites with long half-lives ▪ Half-life increases proportionally with age
Dantrolene[19,20]	Directly affects contractile response of skeletal muscle by interfering with release of calcium from the sarcoplasmic reticulum causing dissociation of excitation-contraction coupling	Initiate 25 mg once daily and increase weekly to 25 to 100 mg, 3 to 4 times daily. Slowly titrate to lowest effective dose. (MAX 400 mg/day)	▪ Reduces clonus, mass reflex movements, and abnormal resistance to passive stretch ▪ Reduces muscle tone and hyperreflexia ▪ Contraindicated in active hepatic disease ▪ Severe hepatotoxicity possible. More common in women over 35 years of age, with concomitant hepatotoxic agents, and in elderly; baseline and ongoing liver function monitoring required. ▪ Generalized muscle weakness occurs and may limit functional improvement ▪ Discontinue if no observed benefit after 45 days of therapy
Tizanidine[16,21,22]	Central alpha-2-adrenergic agonist that lowers sympathetic outflow resulting in decreased resting muscle tone	Initiate 2 mg three times daily as needed and tolerated. Increase by 2 to 4 mg per dose every 1 to 4 days. (MAX 36 mg/day)	▪ Reduces muscle tone and frequency of muscle spasms ▪ Short duration of effect; time doses when minimizing spasticity most desired ▪ Titrate slowly on initiation or discontinuation to reduce side effects or withdrawal ▪ Can cause syncope, hypotension and orthostasis ▪ High anticholinergic burden: xerostomia, asthenia, dizziness, and somnolence common ▪ Administer consistently with or without food due to significant changes in absorption and drug levels ▪ Contraindicated with strong inhibitors of CYP1A2 (e.g., ciprofloxacin) ▪ Metabolized hepatically and eliminated renally; use reduced dose and/or frequency with renal or hepatic impairment

Courtesy of Jennifer Frawley, PharmD, BCPS, BCCCP, Trauma Critical Care Pharmacy Clinical Specialist, Department of Pharmacy, University of Maryland Medical Center

Decubitus Ulcer

Decubitus ulcers are a common complication in patients with acute SCI. A reported incidence of pressure injuries during acute hospitalization ranges from 9 to 36 percent.[23-29] The prevalence noted upon admission to rehabilitation is as high as 33.3 percent.[24,30,31] Up to 25 percent of patients reported having a stage II or higher pressure injury in the year following SCI.[32] Diseases of the skin are the second leading cause for hospital readmission in the first year after SCI.[32] Acquisition of a pressure injury during SCI acute hospitalization is a predictor for readmission to acute care from rehabilitation.[33] Boney prominences are at high risk for pressure injuries and common anatomic locations for decubiti include the sacrum, coccyx, ischial tuberosities, trochanters, elbows, heels, ankles, knees and occiput.[24,31,34]

Risk Factors. Factors that put the patient with SCI at risk for development of pressure injuries include lack of sensation, loss of motor function, spasticity, edema, slowed capillary refill, fecal and urine incontinence, collagen changes and reduced muscle mass.[2,23,26] Additional risk factors include the possibility of hypoxia, hypotension, and acidosis. Hypotension was found to be the strongest predictor of pressure injury development.[26] Patients with complete SCIs, particularly in the cervical region, are at greater risk for pressure ulcers.[25,28,34,35] Older adults with SCI often have more friable skin, poor peripheral perfusion and decline in collagen, normal elastin, and muscle mass, making skin breakdown more likely.[36,37] The presence of co-morbidities, e.g., diabetes, pulmonary or vascular disease, can increase risk of decubiti.[38] Pulmonary complications and urinary tract infections (UTI) are also associated with increased risk for pressure injuries in patients with SCI.[28,35,39] Patients who are malnourished or have a history of pressure injuries also are at higher risk following SCI.[34] Psychological, social and cognitive issues, such as depression, anxiety, impaired cognition, lack of motivation, substance abuse, lack of knowledge, and non-compliance may also make development of decubiti more likely.[34,40,41]

Interventions used to treat SCI also place the patient at risk, including devices such as backboards, cervical collars, other braces and splints, nasogastric and endotracheal tubes, fecal management systems, and oxygen tubing.[24,26,34] Prolonged immobilization while awaiting spinal stabilization can contribute to risk of pressure injury development.[42,43] Prompt transfer to a specialty referral center accustomed to managing patients with acute traumatic SCI was found to reduce the incidence of pressure injuries.[24,44]

Decubitus ulcers can compromise patient outcomes, including delayed surgery or transfer to rehabilitation, prolonged bedrest, increased hospital length of stay and costs, need for additional surgical interventions, hospital readmission, triggered autonomic dysreflexia, decreased functional outcome, and reduced quality of life and self-esteem.[23,45-51] Deterioration of decubitus ulcers can lead to infection, sepsis, and death.[52,53]

Assessment and Prevention. Assess the patient's skin thoroughly and regularly for evidence of pressure injury. Clinical judgment and a validated risk assessment tool are recommended to assess risks for skin breakdown,[34,38] such as the Braden Scale,[54,55] Norton Scale,[56,57] or the Spinal Cord Injury Pressure Ulcer Scale (SCIPUS).[58-60] No consensus exists regarding the best tool or frequency of risk assessment. Identifying the patient's pressure injury risk factors guides the implementation of targeted preventative interventions.[34]

Prevent decubitus ulcers by avoiding known modifiable risk factors including pressure, shear force and moisture to the skin.

- Protect the patient from prolonged contact with unpadded surfaces and remove the patient from the backboard as soon as possible.[34,61]
- Switch the cervical collar to one that is well-padded if it must remain in place.
- Ensure linen and padding near the patient is dry. Keep tubes, folds in the bedding and other non-essential firm medical devices from beneath the patient.
- Use pressure redistribution surfaces (e.g., specialty mattress or bed, seat cushion, heel protectors, and padding beneath the elbows and between the knees when turned) to protect boney prominences and soft tissues from injury.[34] When selecting a mattress for a patient with an unstable spinal injury, consult the vendor to ensure it is acceptable for use with a patient who has an unstable vertebral column injury.
- Mobilize the patient as soon as possible.[38]
- Use a lift or lift sheet to avoid friction and shear on the patient's skin when moving or turning the patient.

- Reposition or turn the patient at least every 2 hours to relieve pressure. Avoid direct pressure on the trochanters when positioning patients on their sides.[34]
- Use an appropriate pressure redistribution seat cushion when mobilizing a patient to a chair. Select a chair that minimizes pressure, provides sufficient support and maintains patient stability with flexibility in the leg and back elevation (e.g., high back wheelchair).[34,38]
- Regularly perform pressure relief maneuvers (e.g., weight shifts, side or forward leans, push-ups) when the patient is out of bed to promote perfusion to skin and muscle. Consider individualized patient risk factors, routine, capabilities, and skin assessment findings when determining the ideal regimen for a pressure relief routine.[38,62,63]
- Consult with physical and occupational therapists to facilitate patient mobilization and proper positioning.
- Provide good skin hygiene.
- Make sure any brace or splint fits well. Provide good skin care beneath the device; checking for any redness or skin breakdown each time the brace or splint is removed.[2] Ensure the skin is dried well after cleaning, and any padding or liner is clean and dry before reapplying.

Care of systemic factors that put the patient at risk for decubitus ulcers is vital. Ensure sufficient blood pressure and systemic oxygenation. Initiate interventions to reduce orthostatic hypotension prior to sitting the patient up and getting the patient out of bed. Implement a bowel and bladder management plan that keeps stool and urine away from the skin to reduce risk for skin breakdown.[34] Promote good nutrition to foster maintenance of skin integrity,[38] and consider a dietitian consultation to help determine the appropriate nutritional intake.

Management. At first sign of skin breakdown relieve pressure at the site. Minimize time a patient is positioned on the area of skin injury, and use support surfaces and positioning practices to avoid pressure on the decubitus ulcer.[34] Consult with a wound ostomy continence nurse to obtain input on solutions for pressure, friction, and moisture reduction, and treatment of skin breakdown. Devise and implement an interdisciplinary plan for managing the patient's skin and any decubitus ulcer. This plan typically includes prescription of specific agents to cleanse and treat the wound including any dressing needed. Antibiotics may be prescribed if an infection is associated with the wound. Electrical stimulation may be used to enhance closure of stage III or IV pressure injuries.[34,64,65] If nonsurgical interventions prove ineffective, surgical wound debridement and coverage may be necessary.

Educate the patient with SCI and their family about the risk for decubitus ulcers and preventative interventions to use. Teach the importance of good skin care and regular skin self-assessments. Education and patient encouragement to take responsibility for skin care and pressure ulcer prevention needs to be reinforced throughout the patient's life.[34]

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NEUROGENIC BOWEL AND BLADDER ACUTE CARE MANAGEMENT

KEY POINTS

- Initiate a bowel management program for all patients with acute spinal cord injury.
- The goal of effective bladder management is to preserve upper urinary tract structures and minimize urinary tract infections. Customize bladder management after acute spinal cord injury to the individual, weighing potential benefits and risks, such as fluid status, comorbid injuries and conditions, and personal preferences.

Neurogenic bowel and bladder dysfunction are highly prevalent following traumatic SCI. Early and effective management of neurogenic bowel and bladder symptoms helps reduce the incidence of secondary complications including autonomic dysreflexia, skin breakdown, ileus, and UTI.

Neurogenic Bowel Management

In the first few days after acute SCI, patients are at risk for ileus because the gut wall is hypotonic and unresponsive to stimuli. Colonic transit time is severely prolonged during the initial weeks post injury, and more than 80 percent of individuals with SCI experience some degree of bowel dysfunction.[1-5] The most commonly reported symptoms of neurogenic bowel dysfunction include constipation (32 to 56 percent), fecal incontinence (27 to 86 percent), abdominal distension or discomfort (22 to 33 percent), and the need for digital stimulation or manual disimpaction (66 percent).[2,3,6-14] Symptoms of neurogenic bowel dysfunction are more severe in individuals with complete SCI compared with incomplete SCI. Constipation due to neurogenic bowel dysfunction can contribute to autonomic dysreflexia when SCI is at or above T6.[3,15-17]

Increased external anal sphincter tone and increased rectal tone and contractility can result from injuries above the S2 level and lead to reflex defecation.[18-21] Injuries at or below the S2 level can cause reduced external anal sphincter tone and reduced rectal tone and contractility, leading to fecal impaction and incontinence.[22-24] The physiological terms reflexic and areflexic bowel are commonly used to classify neurogenic bowel dysfunction. However, individuals with injuries above S2 may show no sign of reflexic bowel function, while others with injuries at the conus medullaris or cauda equina may demonstrate some residual reflexic bowel function.

A bowel management program is indicated for all individuals with acute SCI who do not require surgical intervention (e.g., ostomy), either because of neurogenic bowel dysfunction severity or secondary complications. Bowel management for individuals with reflexic and areflexic neurogenic bowel dysfunction includes diet and fluid management, oral medications (e.g., stool softeners, laxatives, prokinetic agents, and rectal medications), and assisted defecation by means of manual disimpaction and/or positioning. Mechanical rectal stimulation with digital stimulation and rectal stimulants such as suppository or mini-enemas may be useful for individuals with reflexic neurogenic bowel dysfunction. Goals of bowel management include:[25-27]

- Passing stool daily or every other day,
- Moderate to large amounts of soft, formed, bulky stool and complete emptying of the rectal vault,
- Bowel evacuation at a consistent time of day, and
- Completion of bowel care in less than 30-60 minutes.

Surgical diversion via colostomy or ileostomy may be required for severe neurogenic bowel dysfunction and/or severe skin breakdown in the perineal or perirectal regions.

Neurogenic Bladder Management

Most patients with traumatic SCI have impaired micturition and voiding due to neurogenic bladder dysfunction, which can lead to upper and lower urinary tract complications. The overall goal of effective bladder management is to preserve the upper urinary tract structures and minimize lower tract complications.[28]

During an initial period of spinal shock, detrusor areflexia occurs and the bladder has no contractions, resulting in overflow incontinence. Recovery of skeletal muscle reflexes, which may occur after 6 to 8 weeks, leads to gradual recovery of uninhibited bladder contractions for individuals with injuries above the S2 level. Detrusor-sphincter dyssynergia, intermittent or complete failure of external urinary sphincter relaxation

during a bladder contraction and voiding, is a frequent problem that prevents complete bladder emptying in suprasacral SCI.[29,30] High intravesical pressures due to prolonged bladder contractions may cause hydronephrosis and renal deterioration. Impaired detrusor contractility may continue for patients with injuries at S2 or below. These individuals may experience progressively decreased bladder compliance, resulting in increased intravesical pressure with filling that can contribute to upper tract deterioration over time.[31]

Indwelling catheterization is commonly used for initial bladder management to monitor urinary output when maintaining fluid balance is essential.[32] Urethral catheterization is most commonly used initially.

Suprapubic catheter placement is considered as the initial bladder management method for individuals with known or suspected urethral abnormalities, such as stricture, false passages, bladder neck obstruction or urethral fistula, perineal skin breakdown, or the presence of prostatitis, urethritis, or epididymo-orchitis. Suprapubic catheter placement can be performed under local anesthesia; however, consider general or spinal anesthesia to manage autonomic dysreflexia while distending the bladder for safe catheter placement.[33]

Intermittent catheterization is recommended as the optimal bladder management method once precise urinary output measurement is no longer necessary. Intermittent catheterization is performed 4 to 6 times every 24 hours, but it can vary depending on oral fluid intake. Avoid bladder distension and bladder volumes greater than 400 mL to reduce the risk for UTI.[34] Intermittent catheterization may not be feasible for individuals with abnormal urethral anatomy, cognitive impairment, inability to adhere to a catheterization schedule, persistently high fluid intake, or a tendency to develop autonomic dysreflexia with bladder filling despite catheterization.

UTI and urethral trauma are the most frequent complications related to intermittent catheterization. However, the overall incidence of UTI, bladder stones, poor bladder compliance, upper tract deterioration, and urethral erosions is higher in patients treated with indwelling catheters compared with intermittent catheterization for bladder management. When indwelling catheterization is indicated beyond the acute phase of traumatic SCI, suprapubic catheterization is recommended as a long-term option. It avoids the risk of irreversible urethral injury and lower urinary tract complications such as epididymitis and periurethral abscess. Refer patients for ongoing urologic follow-up including urodynamic assessment following their acute hospitalization.

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MOBILIZATION AND REHABILITATION FOR ACUTE TRAUMATIC SPINAL CORD INJURY

This section was prepared with participation from the American Congress of Rehabilitation Medicine's (ACRM) Spinal Cord Injury Interdisciplinary Special Interest Group.

KEY POINTS

- Begin physical and occupational therapy treatment for patients with acute SCI within the first week after injury once medical readiness is determined, even if patients remain on some level of sedation, vasoactive support, or mechanical ventilation.
- Provide holistic early education to patients and caregivers to help them monitor for adverse events, participate in their recovery, and plan for future care.
- Discharge patients with an acute SCI to a comprehensive acute inpatient rehabilitation facility with expertise in SCI when possible.

Early mobility and initiation of physical and occupational therapy within the intensive care unit (ICU) is demonstrated to be beneficial to patients with acute traumatic SCI.[1-3] Implementing an early mobility program is safe, well tolerated, and cost effective; and it has the benefit of decreasing delirium days, increasing ventilator-free days, improving functional outcomes, and increasing the rate of direct discharge to home.[1,4] Adverse events during early physical and occupational therapy are addressed in many studies. While a risk for dislodging essential lines and tubes exists (e.g., peripheral IVs, central lines, urinary catheters and endotracheal tubes), these events occurred in a small percentage of cases and can be avoided when proper clinical judgment and trained staff are used. [5,6] However, limited evidence exists regarding the ideal timing for initiating physical and occupational therapy for patients who experience an acute SCI.

Mobilization

Early mobility, driven by bedside nursing, are all passive interventions focused on passive range of motion, repositioning in bed, and lateral transfers to stretchers for transport or cardiac chairs for upright positioning.[4,7] Consultation with a physical medicine and rehabilitation (PMR) physician, physical, occupational, and speech therapy early in a patient's hospital course is essential to optimize participation in active interventions.

Therapists must identify and follow medical parameters for management prior to implementing bedside physical, occupational, and speech therapy. Medical complexities (e.g., varying hemodynamics, lines and tubes, utilization of braces, and medical devices) can be perceived as barriers to implementation of bedside therapy, but communication and coordination within the multidisciplinary team can overcome these barriers and ensure success.[2,4,7-9] When physical and occupational therapy do not have an established role within the ICU, studies report and support the value for implementing these quality improvement programs in the ICU.[2,4,8,9]

It is recommended that physical and occupational therapy begin treatment for patients with acute SCI within the first week after injury once medical readiness is determined, even if patients remain on some level of sedation, vasoactive support, or mechanical ventilation.[10] No studies directly examine the timing and effectiveness of initiating physical, occupational, and speech therapy in acute SCI, but delays in initiating rehabilitation can prolong return to independence in ADLs and negatively impact quality of life outcomes.[11-13] The goal of implementing rehabilitation early within the hospital stay is to reduce secondary complications, improve functional outcomes, improve patient independence in mobility and ADLs, and help minimize psychosocial distress resulting from injury.[14,15]

Physical, occupational, and speech therapy evaluations consist of a thorough review of the presenting injury, past medical history, and hospital events. During the initial evaluation, therapists perform a systems-review examination to identify physical impairments. Goals and realistic outcomes are then developed based on the patient's needs. The realistic outcomes are then used to provide patients with a reasonable understanding of their prognosis based on injury level, ASIA classification, medical comorbidities, motivation, and family support.[15] Once impairments are determined, therapists develop a plan of care that includes the frequency of treatment and interventions to meet the identified goals. Limited evidence exists about interventions and therapy dosage that produce the best outcomes.[11] Rehabilitation taxonomies help guide therapists to determine ideal interventions and their frequency.[14-18]

Categories of intervention for physical and occupational therapy include: range of motion (ROM)/stretching, strengthening, bed mobility, balance, upright tolerance, endurance, transfer training, wheelchair mobility, seating prescription, skin management, wound care/edema management, equipment prescription, airway clearance, splinting, and patient/family education.[10,14-16] Categories of speech therapy intervention include: functional and augmentative communication, swallowing management, and cognitive/language deficits.[10] Use these categories of interventions, as well as expected long-term functional outcomes to determine which interventions to initiate and prioritize within the acute care environment. Refer to Table 10 for long-term functional outcomes for specific injury levels.

Medical complexities often complicate the progress of therapy in the acute care environment. Prepare and plan for intervention modifications.[15,16]

- Provide the following to all patients: pressure relief schedules with assistance as needed, ROM, strengthening, and consideration for splinting.
- Be diligent in monitoring the skin for pressure injury because the rate is high in the acute phase. Educate patients and families to report signs of pressure injury if utilizing upright sitting schedules or bracing/splinting.
- Anticipate that patients will experience an abnormal hemodynamic response to position change during initial therapy sessions. Progressive head-of-bed elevation and using lower extremity and abdominal compression may help prevent this.

A best practice is to provide holistic early education to patients and caregivers enabling them to monitor for adverse events, participate in their recovery, and plan for future changes including discharge planning from acute care.[10]

Table 10. Expected long-term outcomes based on injury level

Injury Level	Activities of Daily Living	Mobility	Interventions
C1-4	Feeding and communication possible with adaptive and augmentative equipment	Power chair with tongue, chin, head, or breath control. Requires mechanical assistance for pressure relief	Recommendations for adaptive equipment such as call bells. Family engagement in interventions
C5	Feeding and hygiene with set-up assistance and adaptive equipment	Power chair with hand control, may propel over short distances on level surfaces	Strengthen partially intact muscles Momentum strategy for bed mobility and transfers
C6	Feeding and dressing with set-up assistance	Propel manual wheelchair on level surfaces	Active pressure relief Static sitting balance Wheelchair positioning (high back) Tenodesis development
C7-8	Independent with feeding, dressing, bathing, toileting. May require adaptive equipment.	Propel manual wheelchair on most or all surfaces, including outdoors	Dynamic sitting Slideboard transfers Fine motor training
T1-L1	Independent in all self-care areas	Stand with bracing/frame for exercise	Wheelchair propulsion, level ground Lower extremity self-management
L2	Independent in all self-care areas	Potential household ambulation with bracing and assistive devices	Pop-over transfers Low back wheelchair training
L3	Independent in all self-care areas	Potential for community ambulation with bracing and assistive devices	Assisted standing weight physiologic weight-bearing
L4-S1	Independent in all self-care areas	Potential for community ambulation without assistive devices	Standing balance training Assisted transfers and gait training

Adapted from: Saif D, Saif A, Sarhan F. An overview of traumatic spinal cord injury: Part 3. Rehabilitation. *British Journal of Neuroscience Nursing*. 2013 Aug; 9(4): 187–194; Campagnolo DI, Kirshblum S, Nash MS, et al. *Spinal cord medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2011; Braddom RL. *Physical medicine and rehabilitation*. St. Louis, MO: Elsevier Health Sciences; 2010.

Rehabilitation

Discharge from acute care to an inpatient rehabilitation facility, rather than a skilled nursing facility improves long-term outcomes, functional independence, quality of life, and reduces long-term morbidity.[16,19] Ideally, patients with acute SCI are discharged to a comprehensive acute inpatient rehabilitation facility with expertise in SCI. Medical factors that may preclude discharge to an inpatient rehabilitation facility include: limited oral intake, reliance on total parenteral nutrition (TPN), extensive wounds, persistent requirement for mechanical ventilation, or multi-limb involved polytrauma. Long-term acute care hospitals (LTACH) may be most appropriate for initial rehabilitation in this situation, with eventual transfer to an inpatient rehabilitation facility. A best practice is to connect patients/caregivers with a practitioner or liaison within an inpatient rehab facility, to facilitate transfer or to guide provision of an appropriate level of care when the patient is medically ready. Even though inpatient rehabilitation facilities may be scarce, advocate for patients with acute SCI who would benefit the most from these services.[16,19]

Peer-to-peer support initiatives have been implemented in the acute hospital, inpatient rehabilitation and community settings for decades. While little empirical evidence exists about the benefit of these programs on physical health, it is believed they have psychological and social benefit to patients, their family members and peers themselves.[20-22] Peer support for trauma patients during acute hospitalization reduces depression and rehospitalizations.[23-25] Peer mentors can also be a valuable source of information and education for clinicians working in settings where SCI is not common.[26] The Trauma Survivors Network program of the American Trauma Society provides resources for developing a peer support initiative as part of integrated care within trauma centers.[25,27]

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BEST PRACTICES GUIDELINES
SPINE INJURY

IMPLEMENTATION AND INTEGRATION OF THE *ACS TQIP SPINE INJURY BEST PRACTICES GUIDELINES*

IMPLEMENTING THE *ACS TQIP SPINE INJURY BEST PRACTICES GUIDELINES*

KEY POINTS

- The trauma medical director, trauma program manager, trauma liaisons, registrars, and staff have a leadership role in implementing the *ACS TQIP Spine Injury Best Practices Guidelines*, supporting care of the patient with SCI, and monitoring guideline compliance.
- A stakeholder workgroup, receiving its directives from the trauma medical director and the trauma operations committee, implements the spine injury management BPG.
- The workgroup reviews the *ACS TQIP Spine Injury Best Practices Guidelines* and completes a gap analysis related to the current spine care in the trauma center.

Implementing trauma center best practices guidelines begins with the trauma medical director (TMD), trauma program manager (TPM), the trauma liaisons, and registrars as leaders and change agents. These individuals are responsible for the oversight, management, and continuous commitment to improving care within the trauma center and the trauma system, regardless of level of trauma center designation. These leaders define the leadership structure, culture, and implementation processes for the BPG that fosters stakeholder engagement. This process includes the following:

- The spine practice guideline interdisciplinary workgroup, with a defined leader and reporting structure, is charged with reviewing the spine guideline and determining the need to complete a gap analysis that compares current practice to the recommended BPG.
- An educational plan is developed for the implementation of the spine management guidelines and sustaining of the new practices.
- Documentation is integrated into the electronic medical record (EMR) to facilitate reporting consistency and to track outcomes.

The spine injury management guideline workgroup is charged with comparing current practice to those recommended in the BPG to identify gaps.[1] This gap analysis identifies opportunities to align the trauma center's spine management practices with the *ACS TQIP Spine Injury Best Practices Guidelines.* Trauma centers that serve as a referral center for spine injuries may choose to not complete the full gap analysis, but to review the guideline recommendations for potential opportunities. The workgroup, in conjunction with the trauma center's operations committee, establishes the priorities for changes. Progress reports regarding the completion of these identified tasks are provided to the trauma operations committee. Refer to Table 11 for examples of gap assessment tools.

Table 11. *ACS TQIP Spine Injury Best Practices Guidelines* gap analysis

Management Guidelines	Met	Partially Met	Not Met	Priority	Comments
Trauma activation criteria and the inclusion of potential spinal injuries					
Spinal Injury Classification Systems is utilized in the documentation standards					
Pre-hospital care integration and spinal motion restriction indications					
Resuscitation guidelines and specific emphasis on hypovolemic, neurogenic, and spinal shock					
Transfer priorities related to spine injury(ies)					
C-spine clearance process, removal of the cervical collar, and documentation					
Concomitant injuries and their priority of coordination with spinal injuries					
Measures to prevent hypoperfusion and hypothermia					
Measures to maintain a MAP between 85 and 90 mmHg					
Coordination of patient monitoring during diagnostic imaging (CT and MRI)					
Specific imaging recommendations for suspected spinal injuries (SCI and vertebral fractures) and concomitant BCVI					
Management recommendations for injuries to specific regions: cervical, thoracolumbar, and sacral					
Operative indications for spinal injury management					
Critical care management of spinal injuries					
Recommendations for early tracheostomy					
Bradycardia and potential causes, treatment modalities					
Pharmacologic management of spinal injuries					
Respiratory therapist role in spine injury management					
Rehabilitation's team role in the ICU and management of spine injuries					
Coordination of early mobilization					
Analgesia management					
Management of co-morbidities and prevention of hospital events associated with spinal injuries					
Discussion of the goals of care with the patient and family					
Care coordination and handoff during transitions of care					
Discharge planning coordination and patient's/ family's understanding of follow-up care and follow-up appointments					
Coordination of discharge from acute care to inpatient rehabilitation facility					
Expected long-term outcomes based on spine injury level					
Psychological support for acute stress for the patient and family					
Peer-to-peer support opportunities					

Once the gap analysis is completed the next step is to revise or develop the trauma center's spine injury management guidelines for the phases of care provided by the trauma center. The spine injury management guideline is reviewed and approved by the trauma operations committee and the TMD. The operations committee is responsible for the dissemination of and communication about the revised spine injury management guidelines to individuals who participate in trauma care.

The next priority is the development of an interdisciplinary educational plan for the guideline that defines the spine management guideline needs for each unit and phase of care. This educational plan outlines the expectations for the various health professional roles involved in spinal injury assessment and management, as well as the specific tasks associated with assessment, documentation, interventions, and re-assessment. Refer to Table 12 for *ACS TQIP Spine Injury Best Practices Guidelines* Educational Plan Elements.

Table 12. Educational plan elements for the *ACS TQIP Spine Injury Best Practices Guidelines*

Spine Injury Management Guideline Education	Priority for Education	Targeted Staff
Epidemiology and injury patterns for spine injury		
Trauma activation guidelines and response		
Resuscitation management with consideration of suspected spine injury		
Resuscitation management with consideration of spine injury concomitant injuries		
Imaging recommendations and coordination of monitoring during procedures		
Measures to prevent hypoperfusion and hypothermia		
Importance of maintaining a MAP between 85 and 90 mmHg		
Management recommendations of operative and nonoperative management for specific spine injury patterns		
ICU care and coordination with early rehabilitation and mobilization		
Bradycardia management and interventions		
Management for specific regional injuries: cervical, thoracolumbar, and sacral with and without spinal cord injury		
Pharmacologic management		
Analgesia management		
Respiratory therapist role in spine injury management		
Role of the rehabilitation physician, as well as the physical, occupational, and speech therapists in spine injury management		
Measures to prevent co-morbidities and complications		
Coordination and hand-off during transitions of care		
Discharge planning and coordination		
Expected long-term care outcomes		
Psychological care and support for the patient and family		
Peer-to-peer support and opportunities		
Trauma Survivors' Network Information		
Importance of consistent documentation to include the Spine Injury Classification System and "ASIA" Impairment Scale		

The trauma operations committee may define a separate stakeholder group to define which elements of the spine management guidelines can be integrated into the EMR to facilitate consistent documentation. These consistent documentation parameters assist with tracking the phases of care, progress, and outcomes.

The date for BPG implementation is established by the trauma operations committee. The performance improvement and outcome measures to monitor compliance of the spine injury management guidelines are defined prior to implementation.

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INTEGRATING THE *ACS TQIP SPINE INJURY BEST PRACTICES GUIDELINES* INTO TRAUMA CENTER PERFORMANCE IMPROVEMENT

KEY POINTS

- The interdisciplinary workgroup defines elements of the *ACS TQIP Spine Injury Best Practices Guidelines* to monitor through the trauma performance improvement processes.
- After approval by the trauma operations committee, the approved elements are integrated into the existing Trauma Performance Improvement and Patient Safety (PIPS) Plan to monitor compliance.
- The spine injury management BPG performance improvement elements are integrated into the current structure and processes of the PIPS plan.

The interdisciplinary workgroup defines and recommends key elements of the *ACS TQIP Spine Injury Best Practices Guidelines* for integration into the trauma performance improvement processes. After approval of these key elements from the trauma operations committee, they are integrated, reviewed, addressed, and reported through the structure and processes of the Trauma PIPS Plan. These trauma performance improvement recommendations are applicable to the facility's trauma activations and trauma admissions with spine injury. This includes any direct admits for the trauma or spine service. Please refer to Table 13 for Spine Injury Management Guideline Performance Improvement Recommendations.

Regional System Integration

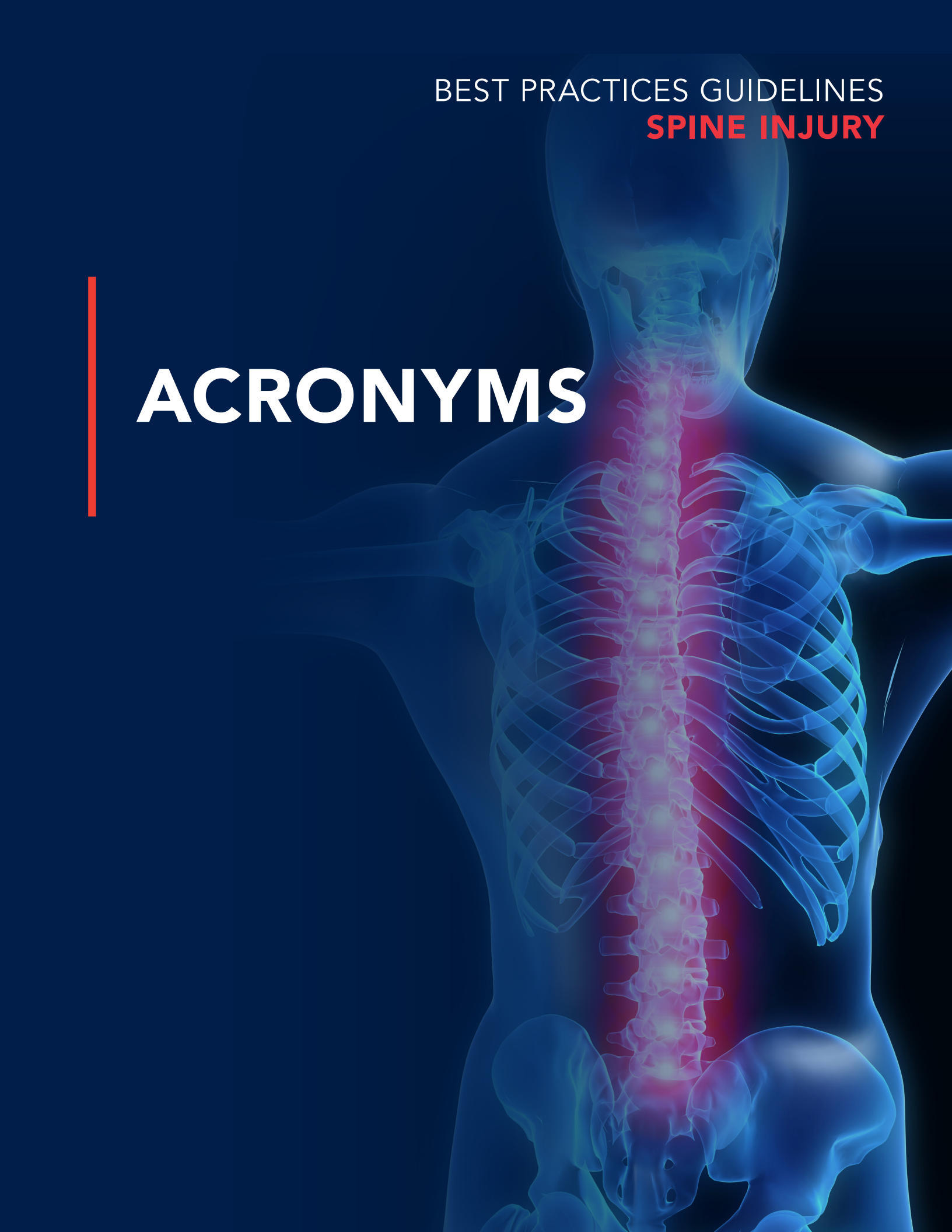
A regional system may choose to develop a regional collaborative to review and coordinate spine injury care across the region. This collaborative initiative is interdisciplinary and needs to include rehabilitation services, and psychosocial services. The spinal injury collaborative defines its priorities and focus, which may require regional data related to spine injury and outcomes. Potential priorities for development of regional spine injury guidelines include a focus on the following:

- Pre-hospital care, field triage, and destination, requiring trauma center data sharing related to spine injury outcomes
- Early access to rehabilitation, requiring the region to identify the various levels of rehabilitation services available and the services they provide.
- Identifying community psychological and peer-to-peer support for the patients with spine injury.

Table 13. *ACS Spine Injury Best Practices Guidelines* performance improvement recommendations
Each trauma center will define the PIPS elements they will review.

Performance Improvement Recommendations	Reviewed for Each Patient and Reported
Pre-hospital care and coordination of hand-off are appropriate.	
Pre-hospital cervical spine motion restrictions are appropriate.	
Cervical spine clearance, removal of the cervical collar, and documentation are appropriate.	
Neurological deficit is documented and recorded.	
Ongoing neurological assessment and documentation are appropriate.	
Timely spine service consultation is initiated.	
Standardized Spine Injury Classification System and the ASIA Impairment Scale are utilized to standardize assessment and documentation.	
Considerations for concomitant injuries are addressed.	
Timeliness and coordination for monitoring during imaging are appropriate.	
Measures to prevent hypoperfusion and hypothermia are initiated.	
If transfer is needed, the timeliness and coordination of the transfer to the receiving hospital are appropriate.	
If need for transfer is identified, the imaging considerations are addressed.	
The team managing the patient defines the goal time for stabilization of the injury interventions, and the goal time is met.	
Tracheostomy for cervical spine injury(ies) occurs within 7 days or the defined goal time established for the specific patient.	
MAP is maintained between 85 and 90 mmHg.	
ICU care incorporates early rehabilitation and mobilization measures.	
Bowel and bladder guidelines are initiated.	
DVT prophylaxis is initiated.	
Measures to prevent skin breakdown and common complications associated with spine injuries are initiated.	
Discharge planning and coordination align with the patient's rehabilitation needs.	
Psychological support and resources are provided to the patient and family, starting in the ICU setting.	
Feedback to the EMS providers and transferring facility are completed within thirty days.	

ACRONYMS



A

AAST – American Association for the Surgery for Trauma
ACRM – American College of Rehabilitation Medicine
ACS COT – American College of Surgeons Committee on Trauma
AD – autonomic dysreflexia
ADLs – activities of daily living
AIS – ASIA impairment scale
AP – anterioposterior
APRV – airway pressure release ventilation
ASIA – American Spinal Injury Association
ATLS – Advanced Trauma Life Support

B

BCVI – blunt cerebrovascular injury
BPG – best practices guidelines

C

CCR – Canadian C-Spine rules
CNS – central nervous system
CPAP – continuous positive airway pressure
CSF – cerebrospinal fluid
C-spine – cervical spine
CT – computed tomography

D

DLC – discoligamentous complex
DOAC – direct oral anticoagulants
DVT – deep vein thrombosis

E

EAST – Eastern Association for the Surgery of Trauma
EBL – expected blood loss
ED – emergency department
EMR – electronic medical record
EMS – emergency medical services

G

GCS – Glasgow Coma Scale
GSW – gunshot wound

I

ICU – intensive care unit
IPPB – intermittent positive pressure breathing
IS – intercostal space
ISCoS – International Spinal Cord Society
ISNCSCI – International Standards for Neurological Classification of Spinal Cord Injury

L

LMWH – low molecular weight heparin

M

MDCT – multidetector computed tomography
MAP – mean arterial pressure
MOI – mechanism of injury
MVC – motor vehicular collision

N

NAEMSP – National Association of Emergency Medical Services Physicians
NASCIS – National Spinal Cord Injury Studies
NEXUS – National Emergency X-Radiography Utilization Study Group
NLI – neurologic level of injury

O

OCF – occipital condyle fractures
OR – operating room

P

PE – pulmonary embolism
PIPS – performance improvement and patient safety
PLC – posterior ligamentous complex
PMR – physical medicine and rehabilitation
pSI – penetrating spinal injury

R

ROM – range of motion

S

SCPP – spinal cord perfusion pressure
SCF – spinal column fracture
SCI – spinal cord injury
SCIPUS – Spinal Cord Injury Pressure Ulcer Scale
SLIC – cervical subaxial injury classification system
SMR – spinal motion restriction

T

TBI – traumatic brain injury
TLICS – thoracolumbar injury classification system
TMD – trauma medical director
TPM – trauma program manager
TPN – total parenteral nutrition
TQIP – Trauma Quality Improvement Program
TQP – Trauma Quality Programs

U

UTI – urinary tract infection

V

VTE – venous thromboembolism

W

WBCT – whole-body CT

BEST PRACTICES GUIDELINES

SPINE INJURY

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