Critical Care Management for Patients with Spinal Cord Injury

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Introduction

Spinal cord injury (SCI) is a complex condition defined by frequently irreversible damage to the spinal cord parenchyma, which may or may not be accompanied by damage to the osseoligamentous elements of the spine. Injury is classically secondary to a traumatic event, such as a motor vehicle collision, a fall, a sports-related injury, or a different high-impact event. Though SCI can occur at any level between the craniocervical junction and the conus medullaris, it is most often associated with damage to the cervical spine, and injuries to this region produce the most profound deficits. The mobile cervical spine is particularly vulnerable to injury, as it lacks the protection and rigid support that the rib cage affords the thoracic spine.

Management of traumatic SCI requires multidisciplinary care, beginning with rapid response teams who must quickly assess the patient's condition in the field and prepare him or her for safe transfer to a higher level of care. If the diagnosis of SCI is suspected in the field, the most expeditious method of transport is recommended to get the patient to a facility with expertise in treating these patients [1]. Care at inpatient centers includes the management of post-injury hypotension and posttraumatic compression of the spinal cord as well as stabilizing the patient for surgery. Secondary considerations include prophylaxis for venous thromboembolic events, decubitus ulcer formation, and infection. Care continues after discharge from the acute care hospital with physical rehabilitation and, in the most severe cases, permanent inpatient care, which is usually reserved for ventilator-dependent patients or those with concomitant severe traumatic brain injury. In this chapter, we briefly touch on the immediate management of SCI and then emphasize the complex critical care needs and controversies surrounding the management of these patients. We conclude with a brief discussion relating to prognosis and outcomes of patients with SCI.

Epidemiology and Background of Spinal Cord Injury

Frequency

The most recent estimates published by the National Spinal Cord Injury Center at the University of Alabama at Birmingham estimate that approximately 288,000 persons in the United States are afflicted by some form of SCI, with 17,700 new cases occurring each year (Table 13.1) [2]. Classically, affected persons have been young men in their 20s [3]; however, the average age at the time of injury has increased steadily since the 1970s and is currently 43 years old [2–4]. Similarly, the proportion of victims who are female has also increased [5]. Behind both of these statistics is an overall increase in the incidence of SCI as opposed to a simple shift in demographics [3, 6]. This change is driven by a rise in the prevalence of motor vehicle collisions precipitating SCI [3, 7–9].

Etiology

Multiple injury etiologies can result in SCI, but the most common are vehicular collisions, falls, sports-related injuries, violence, and self-harm (Table 13.1) [2, 3, 10–12]. The relative contribution of each etiology varies by patient age and by country. Falls are most common among those over the age of 45, increasing steadily with age, and motor vehicle collisions are most common among those younger than 45,

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Table 13.1 Select studies of spinal cord injury epidemiology and etiology in North America

Study	Incidence/prevalence	Etiology	
Chen et al. (2016) [5]	N/R	Vehicular	42.4%
		Falls	22.0%
		Violence	17.3%
		Sports	10.2%
		Medical/ surgical	2.7%
		Other	5.3%
Cripps et al. (2011) [10][a]	*P* = 39 per million Mortality in first-year status post-SCI = 7% Mortality in years 1–10 = 13%	Vehicular	47%
		Falls	20%
		Violence/ self-harm	15%
		Work-related	14%
		Sports/ recreation	11%
DeVivo et al. (1980) [124]	*P* = 906 per million	N/R	N/R
DeVivo, (2012) [3]	*I* = 40 per million per year *P* ≈ 844 per million	Vehicular	48.3%
		Fall	21.8%
		Violence	12.0%
		Sports	10.0%
		Other	7.9%
Ditunno et al. (1994) [125]	*I* = 38 per million per year *P* = 760 per million	Vehicular	45%
		Fall	22%
		Violence	16%
		Sports	13%
Ergas et al. (1985) [126]	*I* = 47 per million per year *P* = 1,009 per million	N/R	N/R
Griffin et al. (1985) [42]	*I* = 54.8 per million per year *P* = 473 per million	N/R	N/R
Harvey et al. (1990) [127]	*P* = 721 per million	N/R	N/R
Jackson et al. (2004) [12]	*I* = 40 per million per year *P* = 854 per million 54% with tetraplegia 45.3% with paraplegia	Vehicular	45.6%
		Fall	19.6%
		Violence	17.8%
		Sports	10.7%
		Other	6.3%
Kumar et al. (2018) [11]	*I* = 51.0 per million per year 50.08% cervical; 24.06% thoracic; 24.21% lumbosacral	Vehicular	41.6%
		Sports	8.6%
Lasfargues et al. (1995) [128]	*I* = 35.6 per million per year *P* = 787 per million	N/R	N/R
Lee et al. (2014) [15][a]	*I* = 38.4 per million per year 47% with paraplegia 53% with tetraplegia	Vehicular	47%
		Falls	22%
		Violence/ self-harm	16%
		Sports/ recreation	10%
National SCI Statistical Center (2018) [2]	*I* = 54 per million per year *P* = 884 per million	Vehicular	38.3%
		Fall	31.6%
		Violence	13.8%
		Sports/ recreation	8.2%
		Medical/ surgical	4.6%
		Other	3.5%
Noonan et al. (2012) [129]	*I* = 68 per million per year *P* = 2,525 per million	N/R	N/R
Savic et al. (2017) [4]	N/R	Vehicular	46.1%
		Fall	31.3%
		Sports	12.3%
		Hit by object	5.1%
		Violence	3.7%
		Other	1.5%

Abbreviations: *N/R* not reported, *P* prevalence, *I* incidence
[a]Derived from World Health Organization statistics

accounting for a progressively smaller proportion of cases with increasing patient age [13]. Injuries among the younger population are higher overall though, making motor vehicle collisions the most common cause of SCI among Americans.

The ability of a motor vehicle collision to cause SCI is likely due to the large forces routinely exerted on the spine during vehicular collisions, which are significantly greater than those seen in the other common etiologies. At greatest risk of injury in these collisions is the cervical spine as it (1) is relatively unrestrained compared to the thoracic and lumbar spines and (2) lacks the support of the rib cage and robust paraspinal musculature of the thoracolumbar spine. Consequently, cervical spine injury is associated with over 50% of patients with SCI after a motor vehicle collision [14], helping to explain the relatively high frequency of tetraplegia among SCI patients [12, 15]. These cervical cord injuries are commonly accompanied by bony injury and resultant cord compression. Accordingly, nearly half of patients (48.8%) will require acute surgical interventions as part of their treatment plan to decompress the spinal cord, realign the vertebral column, or both [14].

Acute Management of Spinal Cord Injury

The acute management of SCI can be separated into field management, transport of the patient to a higher level of care, and inpatient stabilization with medical and surgical interventions. Optimal management should consist of respiratory and hemodynamic monitoring in an intensive care unit [9] along with appropriate medical and/or surgical interventions. Most of the guidelines described in this chapter are derived from the 2013 recommendations made by a joint committee of the American Association of Neurological Surgeons and Congress of Neurological Surgeons [1, 16–35]. Evidence and recommendations were made based upon a modified version of the North American Spine Society Rating Schema for evaluating evidence level [36]. A review of the 2013 guidelines demonstrates that high-quality medical research on the topic of SCI management is too limited to support recommendations with level I or II data. Because of this, most recommendations are actually based on level III data. Table 13.2 lists the

Table 13.2 Level I or II recommendations for the treatment of traumatic spinal cord injury [1, 16–35]

Topic	Recommendations	
	Level I	*Level II*
Prehospital		
Prehospital immobilization [16]	None	All patients with documented or suspected cervical spine injury should have neck immobilized On-scene triage should be conducted by experienced EMS responders Immobilization is not required for a neurologically intact patient who is awake, alert, and oriented to name, location, and date (AAO×3) without neck pain or tenderness
Transportation [1]	None	None
Initial in-house management		
Clinical assessment [19]	Spinal cord Independence measure III should be used for assessment, care, and follow-up of SCI patients. International spinal cord injury basic pain data set should be used to assess pain in SCI patients	ASIA impairment scale and motor testing should be used for neurological examination of acute SCI patients
Radiographic assessment [21]	**AAO×3, asymptomatic**	
	No radiographic examination necessary Discontinue cervical immobilization	None
	AAO×3, symptomatic	
	Obtain high-quality CT of cervical spine. If CT unavailable, obtain 3-view C-spine radiographs (AP, lateral, odontoid views).	None
	Obtunded/unevaluable	
	Obtain high-quality CT of cervical spine. If CT unavailable, obtain 3-view cervical-spine radiographs (AP, lateral, odontoid views).	If normal CT, but high clinical suspicion of SCI, refer to clinician with greater experience in acute SCI treatment.
Closed reduction of fracture-subluxation [22]	None	None
Cardiopulmonary management [23]	None	None
Pharmacologic therapy [24]	Methylprednisolone administration is *not* recommended for patients with acute SCI GM-1 ganglioside administration is *not* recommended	None
DVT/VTE prophylaxis [18]	Prophylaxis against DVT/VTE in SCI patients. Use LMWH, rotating bed, or multimodal intervention. Alternatively, use heparin + compression stockings or electrical stimulator.	Do not use low-dose heparin as monotherapy. Do not use oral anticoagulants. Start VTE prophylaxis within 72 hours. Prophylaxis against DVT/VTE for ≥6 months.
Nutrition [20]	None	Use indirect calorimetry to determine caloric needs of SCI patient.
Management by injury		
Occipital condyle fracture [25]	None	Obtain CT to visualize injury.
Atlanto-occipital dislocation [26]	Obtain CT to determine condyle-C1 interval in pediatric patients with suspected AO dislocation.	None
Atlas fractures [28]	No	None
Axis fractures [27]	None	Consider surgical stabilization if type II odontoid fracture in patient ≥50 years old
Combination atlas-axis fractures [29]	None	None
Os odontoideum [30]	None	None
Subaxial fracture [31, 32]	None	None
Central cord syndrome [33]	None	None
SCIWORA [35]	None	None
Vertebral artery injury [17]	Obtain CT angiogram in patients with C-spine trauma that meets Denver screening criteria for vertebral artery injury	None

Abbreviations: *EMS* emergency medical services, *AAO×3* awake, alert, and oriented, *SCI* spinal cord injury, *ASIA* American Spinal Injury Association, *CT* computed tomography, *AP* anteroposterior, *DVT* deep vein thrombosis, *VTE* venous thromboembolism, *LMWH* low-molecular-weight heparin, *AO* atlanto-occipital, *SCIWORA* spinal cord injury without radiographic abnormality

relevant level I and II recommendations for the management of a patient with acute SCI. If there is insufficient evidence to make recommendations regarding the timing of surgery, the adage "Time is spine" is widely used [9, 37].

Field Management

The first step in the treatment of a patient with acute SCI is safely expediting their transfer to a higher level of care. This involves determining whether the patient is stable enough to move from the site of injury and, if not, implementing interventions necessary to stabilize him or her for transfer. Failure to do the latter is thought to precipitate up to 25% of all SCI, underlining the importance of high-quality field management in the care of patients with SCI [38].

The first step in verifying a patient's clinical stability is to evaluate the "ABCs". That is, check his/her *a*irway for obstruction, look for signs of spontaneous respirations (*b*reathing), and ascertain whether he/she has a palpable pulse (*c*irculation) [9]. This brief assessment is used to evaluate all trauma patients for potentially correctable life-threatening issues. Once these have been addressed, consideration is given to rapidly transporting the patient to a trauma center. Patients should also be evaluated briefly for alertness and orientation to name, location, and date (awake, alert, and oriented times three; also known as AAO×3). The responses to these questions are important when assessing a patient for a traumatic brain injury, which occurs frequently in traumatic injury and often accompanies SCI. Those patients who are coherent and oriented can be questioned about axial neck pain and the circumstances surrounding their injury. Patients who are neurologically intact and who deny axial neck pain, distracting injuries, or circumstances consistent with an injury to the spine can be transferred to the nearest trauma facility without cervical immobilization [16]. Such patients, especially those involved in high-velocity collisions, should still be evaluated at a trauma hospital where the possibility of associated injuries can be assessed. Those who report axial neck or back pain or those with a suspected SCI should be immobilized with an appropriate rigid cervical orthosis and placed on a backboard before being transferred to the nearest trauma center for neurological and radiological evaluation [1, 39]. Exact protocols vary by emergency medical services region, but total spinal immobilization is universally recommended, though prior systematic reviews have failed to document high-quality evidence to suggest that this intervention prevents or reduces long-term neurological injury [40]. The type of cervical collar used for immobilization appears not to alter outcomes significantly, so field providers are encouraged to use the collar with which they are most familiar [38].

Transit

Only low-quality evidence exists describing transport of patients with SCI to higher levels of care [1]. Despite this, recommendations are quite strong in terms of timing; patients with SCI *should be moved to the next level of care as soon as they are stable enough for transport*. Types of transportation include ambulance, plane, and helicopter; the chosen mode should be whichever allows for the most expeditious movement of the patient to a center with experience treating acute SCI. That said, transfer to a higher level of care should not be substantially delayed for medically unstable patients if they are waiting to be transported to a specialized spinal cord treatment center.

Injury Assessment

Once the patient is stabilized, care can turn to the assessment of the SCI. SCI can be classified by either the level of vertebral body injury or the level of SCI. The degree of concordance between the two is high in the upper cervical spine and steadily decreases as one moves caudally due to differences in the postnatal growth rates of the spinal cord and bony spine. The level of the injured vertebra(e) is most important when considering surgical intervention, as it identifies that region of the spine that might require decompression, stabilization, or realignment. The neurological level is more important than the vertebral level when articulating the severity of the injury.

Neurological Assessment

In general, injuries that are more cephalad beget greater functional disability and are associated with poorer long-term survival and greater permanent disability [3, 4, 41–49]. However, clinicians treating SCI must use a standardized description of the neurological injury, which provides normative information regarding injury prognosis. Currently, the gold standard for injury assessment is the American Spinal Injury Association (ASIA) Impairment Scale, with which all trauma providers should be familiar (Table 13.3) [3, 4, 41–49]. This scale grades the extent of SCI based upon motor output in the C5–T1 and the L2–S1 myotomes (each graded on the familiar 0–5 manual motor testing scale), the presence or absence of a sensory level (based on pinprick and light touch sensation in the C2–S4/5 dermatomes), and rectal tone. Those with full strength and no sensory or rectal findings are graded as *ASIA E*; those with 0/5 motors, with a documented sensory level, and with no rectal tone are *ASIA A* [50]. Injuries intermediate to these extremes are classified as *ASIA B* (0/5 motors, sensory level, rectal tone intact),

Table 13.3 American Spinal Injury Association (ASIA) impairment scale and motor scoring system [9]

ASIA impairment scale					
Grade	Description				
E	*Normal*. Sensation and motor function preserved in all segments				
D	*Motor incomplete*. Strength of key muscle functions is ≥3/5 on manual motor testing in ≥50% of groups below the injury level				
C	*Motor incomplete*. Preservation of motor function for voluntary anal contraction *or* patient meets criteria for ASIA B with sparing of motor function such that ≥50% of groups below injury level are <3/5 on manual motor testing				
B	*Sensory incomplete*. Sensory but not motor function preserved below the level of neurological injury *and* no motor function is present ≥3 levels below the level of neurological injury				
A	*Complete injury*. No sensory/motor function preserved in S4–S5 segments				
ASIA motor scoring system: muscle groups					
Upper extremity			*Lower extremity*		
Root	**Muscle group**	**Movement**	**Root**	**Muscle group**	**Movement**
C5	Biceps brachii	Elbow flexion	L2	Iliopsoas	Hip flexion
C6	Wrist extensors	Wrist extension	L3	Quadriceps	Knee extension
C7	Triceps	Elbow extension	L4	Tibialis anterior	Foot dorsiflexion
C8	Finger flexors	Flex middle finger	L5	Extensor hal. Long.	1st digit dorsiflexion
T1	Hand intrinsics	Abduct fifth digit	S1	Gastrocnemius	Foot plantar flexion
Manual motor testing scale					
Grade	*Interpretation*		*Grade*	*Interpretation*	
5	Full strength		2	Active movement with gravity removed	
4	Active movement against resistance		1	Flicker or trace contraction	
3	Active movement against gravity		0	No contraction/total paralysis	

ASIA C (<3/5 strength in most groups below injury, sensory intact, rectal tone present), or *ASIA D* (≥3/5 strength in most groups above the level of injury, sensory intact, rectal tone present) [51]. The ASIA International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) worksheet can be found here: https://asia-spinalinjury.org/international-standards-neurological-classification-sci-isncsci-worksheet/.

In addition to sensorimotor impairment, damage to the cervical and high thoracic spinal cord may compromise autonomic nervous system output, causing diaphragm paralysis (injuries at or above the level of the phrenic nerve cell bodies, C3–C5) and complete loss of sympathetic tone (via loss of descending input to cell bodies situated in the T1–L2 segments) [9]. Loss of sympathetic tone is especially germane to the acute medical management of SCI patients as it produces spinal or neurogenic shock, which is characterized by hypotension, bradycardia, a widened pulse pressure, and a distributive-type circulatory shock. In this state, occasional outflow from the unregulated sympathetic nerves can trigger reflex spinal sympathetic stimulation with subsequent vasoconstriction and intermittent hypertension. In turn, parasympathetic output is increased at levels above the injury, worsening vasodilation and hypotension, a clinical phenomenon known as *autonomic dysreflexia*—most common in injuries above the T7 level [52, 53]. This condition is discussed in greater detail below.

Radiographic Assessment

After assessing the neurological status of the patient, the next step in management is acquisition of cervical spine imaging. In the setting of suspected or confirmed head trauma, patients should undergo rapid head computed tomography (CT) to rule out intracranial pathology [54, 55]. This may be accompanied by concomitant imaging of the cervical spine. In the absence of suspected head trauma, the imaging algorithm is dictated by the patient's neurological status. Based upon level I evidence provided by the National Emergency X-Radiography Utilization Study Group, patients who are awake, not intoxicated, neurologically asymptomatic, and have no distracting injuries do not require cervical spine imaging or continued immobilization [56]. Patients who are awake but who demonstrate signs of neurological injury should undergo high-quality CT to characterize the bony injury. If high-quality CT is unavailable, 3-view (i.e., anteroposterior, lateral, odontoid views) radiographs of the cervical spine should be acquired, and the patient should be transferred to a facility capable of evaluating intracranial injury. Some authors [9] also recommend high-quality CT of the thoracic and lumbar spine, given the possibility of concurrent thoracic and/or lumbar injuries that may be masked by neurological dysfunction secondary to cervical spine injury [8]. Acquisition of dynamic radiographs with flexion and extension views may be considered in persistently symp-

tomatic patients, as they may be useful in diagnosing underlying instability.

CT and radiography are limited in their ability to assess soft tissue damage, such as that resulting from trauma to the intervertebral discs, vertebral ligaments, and neural structures. For this reason, urgent magnetic resonance (MR) imaging of the spine is also recommended in patients with persistent neurological injury [9, 57]. Short T1 inversion recovery (STIR) sequences provide the best assessment of the soft tissue injuries, especially to the spinal cord, since injury-related edema and tissue disruption both appear as T2 hyperintensities [14]. Many of these changes frequently resolve within the first 48 hours of injury, underlining the importance of expediting MR acquisition in patients with persistent neurological injury [38]. Additionally, MR allows for the identification of intracanalicular disk fragments that might injure the cord upon application of traction, so some providers recommend acquiring MR before traction is used [9].

For patients who are comatose or whose examination results are inconsistent, the default management strategy should be to assume cervical spine injury and take steps to rule out this injury. Consequently, patients in this last category should undergo high-quality CT of the entire neuraxis to rule out both intracranial pathology and multilevel injury [38].

Classification Systems

In an effort to standardize radiographic classification of cervical spine injury and to streamline management of these patients, several classification systems have been devised for cervical spine injury. Anatomically, injuries can be classified as occurring at the atlanto-occipital junction, atlantoaxial junction, or subaxial spine.

Atlanto-occipital injuries involve trauma to the skull base, atlas (C1), or ligamentous structures of the occipitocervical junction. Skull base trauma can be categorized into three classes: class I injuries comprise comminuted fractures of the occipital condyle, class II injuries include basilar skull fractures, and class III injuries describe avulsion fracture of the alar ligaments [38]. Atlas trauma is similarly divided into three classes: type I injures result from fracture of the dorsal arch, type II injuries from lateral mass fracture on one side, and type III injuries—so-called Jefferson fractures—from three or more fracture sites around the atlantal ring. Lastly, compromise of the occipitocervical ligamentous complex may be associated with atlanto-occipital dislocation, an often-fatal complication seen in roughly 1% of patients presenting with cervical spine trauma [58].

Fractures of the axis (C2) are especially common among elderly patients [59]. Among this population, the greatest burden of disease is caused by fractures of the odontoid process, which account for nearly 90% of cases of axis trauma [59]. Axis fractures are divided into three classes based upon the level of the fracture: type I fractures consist of avulsion fractures at the tip, type II fractures occur through the waist of the dens, and type III fractures involve the body of the axis [60]. Among young and middle-aged adults, Hangman's fractures—fractures through the bilateral *pars interarticularis*—are also relatively common [59]. These injuries are significantly more destabilizing than odontoid fractures and are graded based upon the degree to which the dissociated segments remain apposed to one another [61]. Type I fractures involve less than 3 mm of displacement, type II injuries involve >3 mm displacement with >11° of angulation in the odontoid, and type III fractures are associated with bilateral C2/3 facet dislocation. Thankfully, the vast majority of axis fractures are not associated with a concomitant neurological injury.

For injuries of the subcervical spine, the most common scoring system is the subaxial cervical spine injury classification (SLIC) system developed by the AOSpine Classification Group (Table 13.4) [62]. Under this system, injuries are graded with respect to morphology and any associated neurological deficit. A similar system was developed for the thoracolumbar spine, which also incorporates damage to the posterior ligamentous complex (Table 13.5) [63]. Both systems have been validated by their authors [64] and by independent groups [65, 66]. Recently, updated versions of these classification systems have been published [64, 67–71],

Table 13.4 The subaxial cervical spine injury classification (SLIC) system

Characteristic	Points
Morphology	
No abnormality	0
Compression	1
Burst	+1 (2)
Distraction (e.g., facet perch, hyperextension)	3
Rotation/translation (e.g., facet dislocation, unstable teardrop, or advanced-stage flexion-compression injury)	4
Neurological status	
Intact	0
Root injury	1
Complete cord injury	2
Incomplete cord injury	3
Continuous cord compression in setting of neuro deficit	+1
Score	**Management**
< 4	Nonoperative treatment
4	Operative vs. nonoperative
≥ 5	Operative treatment

The data presented in this table are republished with permission from Vaccaro et al., 2007 [62]

Table 13.5 The thoracolumbar injury classification and severity score (TLICS)

Characteristic	Points
Morphology	
No abnormality	0
Compression	1
Burst	+1 (2)
Translational/rotational	3
Distraction	4
Integrity of the posterior ligamentous complex	
Intact	0
Suspected/indeterminate injury	2
Injured	3
Neurologic status	
Intact	0
Nerve root	2
Cord, conus medullaris	
Complete	2
Incomplete	3
Cauda equina	3
Score	**Management**
<4	Nonoperative treatment
4	Operative vs. nonoperative
≥5	Operative treatment

The data presented in this table are republished with permission from Vaccaro et al., 2005 [63]

though their utility is less well-established than the original systems.

All of the abovementioned systems are based on the bony injury observed in the traumatized region, which demonstrates the need for high-quality CT in the evaluation of patients presenting with traumatic SCI. However, as described earlier, many injuries may be limited to spinal cord and soft tissue trauma, which is only recognizable on MR imaging. Furthermore, some have suggested that the degree of permanent deficit is related to the extent of cord injury. One classification system that uses cord signal changes on MR imaging to classify cord injury is the Brain and Spinal Cord Injury Center (BASIC) score [72]. This internally validated metric uses T2-weighted imaging to grade spinal cord lesions based upon the extent of T2 signal hyperintensity and presence (or absence) of spinal cord hemorrhage. Though limited in its ability to guide the decision to stabilize, it does appear to predict a patient's neurological recovery to a reasonable degree of accuracy.

Hemodynamic Pathophysiology and Therapeutic Interventions After Acute Spinal Cord Injury

As discussed above, acute SCI is often accompanied by hemodynamic instability owing to decreased sympathetic tone and autonomic dysreflexia. Therefore, all patients with acute SCI should receive continuous hemodynamic monitoring in an intensive care unit. Key to management is maintaining sufficient perfusion of the cord, with a target systolic blood pressure of ≥100 mm Hg and a mean arterial pressure of 85–90 mmHg [23]. Target oxygen saturation is ≥90% to prevent cord ischemia, as parenchymal hypoxia has been associated with greater neuronal death and is consequently likely to decrease the odds of neurological recovery [73]. These targets should be kept for a period of 5–7 days, at which point insufficient evidence exists to affirm their efficacy. Maintaining mean arterial pressure above 85 mm Hg can be accomplished via a combination of volume repletion with crystalloids and colloids (in patients with signs of volume depletion) and/or vasopressors, including dopamine (1–20 μg/kg/min), dobutamine (5–15 μg/kg/min), epinephrine (1–10 μg/min), norepinephrine (1–20 μg/min), or phenylephrine (10–100 μg/min) [57]. Norepinephrine followed by dopamine is the preferred regimen for lesions above the T7 segment, due to their combined chronotropic and inotropic effects (i.e., required to address loss of sympathetic output through the thoracic cardiopulmonary nerves), whereas pure vasoconstrictors such as phenylephrine are preferred for lesions at T7 or below [57].

Autonomic Dysreflexia

Paradoxically, acute hypertension is also a concern in patients with injury above the level of T6, as such injuries can produce a condition known as *autonomic dysreflexia*. Concern for autonomic dysreflexia is raised in patients with traumatic SCI who demonstrate acute elevations in systolic blood pressure (>20–30 mmHg) with associated bradycardia [74]. It is most common in the chronic phase of those with complete tetraplegia secondary to high cervical injuries (which disrupt descending vasomotor pathways), but it has also been noted in the acute setting. Treatment consists of prophylaxis and control of hypertension (target systolic blood pressure ≤150 mmHg) [75].

The most commonly used means of prophylaxis is periodic urinary catheterization since urinary bladder distension and irritation is the most common trigger of autonomic instability. In cases where urinary catheterization fails, level I evidence supports interventions aimed at decreasing efferent stimulation, including botulinum toxin injection into the detrusor muscle [76–78], intravesical resiniferatoxin injection [79, 80], and/or an oral anticholinergic [81]. Sacral denervation procedures may also be indicated in refractory cases; however, this is supported by only low-quality evidence [82].

Control of blood pressure in patients with autonomic dysreflexia begins with nonpharmacologic management, including removal of constrictive clothing and elevating the patient

to a seated position. Blood pressure should be checked every 5 minutes until the event has resolved. In patients for whom this is ineffective at terminating the episode, administration of an antihypertensive agent should be considered. No specific agent is recommended, but regimens previously suggested to be effective include nifedipine (10 mg; bite and swallow; level II evidence), nitrates (level V evidence; no clinical evidence), captopril (25 mg sublingual; level IV evidence), terazosin (level IV evidence), and prazosin (0.5–1 mg bid/tid PO; level 1 evidence). Monitoring of blood pressure and heart rate should be continued for at least 2 hours after symptom resolution [57].

Diaphragm Paralysis and Ventilatory Support

Patients with high cervical injuries (C1–C4) often present with respiratory insufficiency (36–83% of cases) secondary to loss of phrenic nerve input; concomitant head trauma may also contribute to respiratory insufficiency [83]. Consequently, all patients with suspected cervical spine injury should be evaluated for respiratory support and intubation [84]. Goals for respiratory management focus on maintaining adequate oxygenation of cord and peripheral tissues [85]. In patients breathing spontaneously, supplemental oxygen should be provided as needed, and patients should be encouraged to take deep breaths to avoid atelectasis (incentive spirometry is useful in this regard). Adequate analgesia should also be provided to facilitate deep respirations without depressing respiratory drive.

In patients requiring ventilatory support—that is, those with vital capacity below 15 mL/kg, increased $PaCO_2$, or inspiratory pressure ≥ -14.5 mmHg— ventilation and tracheostomy should be considered early, especially when prolonged intubation is anticipated. Ventilatory targets for these patients are similar to those established for head injury: $PaCO_2$ should be maintained in the range of 26–30 mm Hg [83, 85, 86]. Though some groups have recommended 0 mm Hg end-expiratory pressures to avoid air trapping, evidence supporting this is insufficient, and it is recommended that patients receive tidal volumes of 10–12 mL/kg and positive end-expiratory pressures of 5–7 cm H_2O. The use of positive end-expiratory pressure should be evaluated carefully in patients with concomitant traumatic brain injury because of the theoretic potential to increase intracranial pressure. Some evidence suggests that tidal volumes greater than 20 mL/kg may decrease the time to wean, but the strength of this evidence is moderate at best.

Weaning patients with SCI from the ventilator should be done via progressive ventilator-free breathing, in other words, gradual increases in respirator-free time, starting with FiO_2 of 10% above respirator baseline and 5 minutes of disconnection per hour [83]. If the patient reaches 48 hours without ventilator support and achieves an inspiratory pressure < -15 mm Hg, discontinuation of the ventilator may be considered. Alternatively, if the patient has been ventilator-dependent for more than 72 hours, tracheostomy should at least be considered to establish a definitive airway. Tracheostomy is also more comfortable for the patient and allows for improved pulmonary hygiene [57].

Respiratory insufficiency in patients with SCI is associated with an increased risk of respiratory infection [83], which is the most common cause of mortality among patients with SCI [47]. This is especially true for those with lesions above the mid-thoracic level: these patients have lost innervation of most expiratory musculature, which is responsible for effective coughing and clearing of airway secretions [83]. This is compounded by increased respiratory secretions in patients with autonomic dysfunction. In these patients, secretions should be regularly cleared via postural/gravity-assisted drainage and manually assisted coughing (i.e., chest compressions as the patient attempts to cough). Percussion or vibration of the thorax may also facilitate secretion clearance. In patients with cervical spine instability or thoracic trauma, manual-assisted coughing should be avoided until the patient is cleared for these maneuvers by the spinal surgeon, due to the risk of further injury secondary to application of mechanical forces to the chest wall.

Steroid Use

Acute SCI can itself be divided into primary and secondary injury phases [9]. The primary injury phase is the injury that occurs secondary to physical insults to the cord and nerve roots: namely, contusion, transection, and laceration with or without persistent compression [9]. This damage occurs at the time of injury and is at present irreversible. The second phase of injury is that which occurs in the hours to days following the injury and is mediated by a combination of oxidative and inflammatory damage. It is this second-phase damage that pharmacological treatment of traumatic SCI seeks to lessen or reverse.

The most widely evaluated medication used in the treatment of acute SCI is high-dose intravenous methylprednisolone. Methylprednisolone is purported to work by reducing cord swelling and inflammation, two of the primary mediators of secondary injury (via free-radical production) and glial scarring. Three large clinical trials called the NASCIS trials [87–91] evaluated the clinical efficacy of intravenous methylprednisolone. Except for a secondary analysis of the NASCIS II demonstrating a minor improvement in motor scores, no significant difference was observed in terms of neurological recovery among patients receiving methylprednisolone and those receiving placebo treatment [92].

Reanalysis of these results by an independent third party noted several inconsistencies in the data analysis, calling into question the motor improvement noted by the NASCIS II authors. These discrepancies included the authors' decision to report only right-sided motor scores, despite collecting bilateral motor scores, and to arbitrarily divide patients receiving methylprednisolone within 8 hours of injury from those treated more than 8 hours post-injury [90, 91]. Furthermore, the documented motor score differences were clinically meaningless as measured by the ASIA Functional Impairment Measure (FIM) [90], and methylprednisolone significantly increased the risk of adverse effects [93] (specifically gastrointestinal hemorrhage and wound infection).

For these reasons, the 2013 American Association of Neurological Surgeons/Congress of Neurological Surgeons Joint Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injury recommended against the routine use of methylprednisolone for patients with acute cervical SCI, as the likelihood of an adverse event far exceeds the likelihood of any therapeutic improvement. Contradictory guidelines developed by AOSpine suggested a 24-hour infusion of high-dose methylprednisolone (30 mg/kg bolus + 5.4 mg/kg/hours × 23 hours) to SCI patients presenting within 8 hours of injury [94]. The AOSpine group reached this recommendation through meta-analysis of three randomized controlled trials and one prospective trial [87, 92, 93, 95, 96], which found a moderately superior improvement among methylprednisolone-treated patients without a concomitant rise in complication rates. Notably, the group acknowledged this to be a weak recommendation and did not include the results of either NASCIS I or NASCIS III in their meta-analysis. Furthermore, the authors acknowledged that, although their included evidence supported a statistically significant improvement in motor function, it is unknown whether this translates to a clinically meaningful benefit.

Consequently, we believe that the preponderance of evidence suggests *there is little to no neurological benefit to be gained from administering methylprednisolone in patients with acute SCI*. However, if a patient presents within 8 hours of injury and has a low risk for complications (e.g., a younger patient without medical comorbidities), there might be a small clinical benefit as suggested by a recent meta-analysis [97]. Most groups agree that there is no benefit to methylprednisolone administration more than 8 hours after an acute injury.

Evidence for other pharmacological interventions is currently limited. The antibiotic minocycline [98] has shown some ability to improve neurological recovery and is undergoing a phase II clinical trial (NCT01828203). A phase III clinical trial of riluzole (NCT01597518) is also currently underway. To date, no definitive results are available from either trial.

Traction

In patients with acute cervical SCI, traction has been a well-studied, valuable adjunct in the treatment of pathology where there is misalignment secondary to a fracture, dislocation, or combination of the two. Barring any ligamentous injury at the craniocervical junction, traction can generally be used in awake patients and is most commonly applied via Gardner-Wells tongs or a halo ring. It should be noted that there is a theoretical risk of worsening deficit following traction; however, in the overwhelming majority of cases, it can provide immediate realignment of the spinal column with decompression of the spinal cord [22].

Prior to application of traction, CT of the cervical spine is evaluated to rule out injury at the level of the craniocervical junction. The patient is treated with a nonsedating pain medication (e.g., morphine, fentanyl) and muscle relaxant (diazepam) and is then placed supine [99]. Pin sites are scrubbed with 70% alcohol followed by povidone/iodine, and the pins are treated with bacitracin gel. Pins should be placed at the external auditory meatus, or in the case of jumped facets (with the intention of inducing flexion correction), 3 cm posterior to the external auditory meatus. In cases where a halo ring is being used for traction, the anterior pins should be placed 1 cm above the orbital rim and the posterior pins should be placed over the mastoid [99].

After application of initial traction, fluoroscopy and radiography are used intermittently to evaluate for correction of the injury and to prevent over-distraction at the craniocervical junction. Traction should be discontinued if the patient is unable to tolerate the procedure, demonstrates neurological deterioration, or displays evidence of over-distraction on radiography. If the patient remains neurologically intact and tolerates the procedure, traction may be progressively increased in 5- to 10-lb increments, with new radiographs/fluoroscopy images acquired after each weight increase. Traction should be increased progressively until the fracture is reduced (approximately 5–10lbs/cervical level), the patient is unable to tolerate the procedure, or the patient demonstrates neurological deterioration [100]. If the fracture is reduced, a halo vest or other cervical orthosis can be applied to maintain the correction. Traction can also be maintained until surgery, if indicated. Although there is a paucity of high-quality evidence to support the use of traction in the setting of acute cervical spine injury, a recent review reported that closed reduction with Gardner-Wells tongs or a halo ring is successful in reducing the spinal deformity in 80–90% of cases [57].

Surgical Intervention

Surgical intervention for acute traumatic SCI consists of neural element decompression, correction of deformity, and

stabilization across the injured region. Prolonged compression of the spinal cord after trauma contributes to worsening secondary injury by generating ongoing parenchymal ischemia, presumably through compression of the cord vasculature. This is supported both by extensive primary research [37, 101–103] and by several meta-analyses [104–107], which have demonstrated significantly better neurological outcomes for patients who underwent surgical decompression within 24 hours of injury. In fact, some of these studies have recommended that decompression takes place within 8 hours of injury (or as soon as possible) for optimal outcomes [107, 108]. In addition to achieving improved neurological outcomes, Bourassa-Moreau et al. [109] and Carreon and Dimar [110] both demonstrated that early decompression (i.e., within 24 hours of injury) was associated with lower rates of pneumonia and urinary tract infection. Pursuant to these results, most surgeons (≥80%) currently recommend rapid decompression (≤24 hours) for patients with complete or incomplete SCI, with most (72.9%) recommending decompression within 6 hours, if possible, for patients with incomplete SCI [111].

In addition to surgical decompression, some evidence suggests that duraplasty with or without drainage of cerebrospinal fluid may aid in neurological recovery. Evidence supporting its use is limited to animal studies [112]. In a porcine model, mean arterial pressure elevation with cerebrospinal fluid drainage was associated with significant improvements in spinal cord perfusion compared to mean arterial pressure management alone [112]. This evidence supports a Monro-Kellie-like model of spinal cord fluid dynamics, wherein residual elevation in intrathecal pressure decreases spinal cord perfusion pressure, increasing hypoxia and neuronal damage secondary to free radical production [113].

Concomitant spinal fixation with instrumentation is recommended for patients with mechanically unstable injuries or for patients in whom multilevel decompression may cause iatrogenic destabilization. The selection of approach and stabilization levels is ultimately up to the treating surgeon with multiple factors being taken into consideration. In general, all deformities should be corrected and fixated, instrumenting at least one (and sometimes more) level above and below the unstable segment. For occipitocervical instability, this means fixation from the skull into the subaxial spine, using a combination of an occipital plate and lateral mass fixation, or wiring with a malleable titanium rod in pediatric patients. Similarly, for patients with isolated subaxial spine trauma, lateral mass fixation is most commonly used. Here the main caveat is that fusion should extend past the cervicothoracic junction in patients demonstrating instability of the low subaxial spine to prevent progressive cervicothoracic deformity. For isolated atlantoaxial instability, posterior fusion with transarticular or a lateral mass screw technique can be quite effective [114, 115]. Lastly, anterior odontoid screw fixation can be effective for select younger patients with type II odontoid fractures and no evidence of concurrent injury to the posterior osseoligamentous complex.

Other Considerations

Decubitus Ulcers

Although more worrisome in the chronic setting, decubitus ulcers are a complication seen in a 10–30% of patients with SCI and are associated with healthcare costs of $1.2 to $1.3 billion annually [116]. Ulcers most commonly present on the buttocks (31%), lateral thighs (26%), and sacrum (18%) [9] and can be prevented with daily skin cleaning, inspection, and frequent rotation of the patient throughout the day. When sores develop, they should be cleaned thoroughly with aseptic technique and debridement of necrotic tissue where necessary.

Venous Thromboembolism

Deep venous thrombosis and pulmonary embolism are two common complications seen in 40% or more of patients with SCI [39]. The current recommendation is to implement venous thromboembolism prophylaxis with low-molecular-weight heparin (40 mg SQ qday) along with conservative measures, including compression stockings and/or serial compression devices. Vena cava filters are not recommended as a routine prophylactic measure but can be considered for select patients in whom anticoagulation fails or who are not candidates for anticoagulation and/or mechanical devices [18]. In all patients with SCI, early mobilization and rehabilitation may also help to reduce the risk of venous thromboembolism.

Sphincteric Dysfunction

Most patients with acute SCI develop bladder and bowel dysfunction. Neurogenic bladder may be addressed with intermittent Valsalva maneuver in a select minority of patients but most require intermittent sterile catheterization to drain the bladder. Failure to treat neurogenic bladder results in accumulation of urine in the renal pelvis with resultant hydronephrosis and the potential for chronic renal failure, which historically has contributed significantly to mortality among SCI patients [57].

The posttraumatic bowel similarly requires intervention to promote passage of stool, which can be accomplished with dietary modification, rectal stimulation when necessary, and stimulant laxatives (e.g., senna, bisacodyl, sodium picosulfate). In patients with permanent bowel dysmotility, colostomy may be recommended. However, consideration of colostomy requires in-depth discussion with the patient and is almost never required [39]. One additional clinically important gastrointestinal sequela of SCI is the acute stress

ulcer, also known as a *Cushing ulcer*. Patients should receive antisecretory agents for prophylaxis against this clinical entity in the acute setting; proton pump inhibitors (e.g., omeprazole 20–40 mg PO qday) are first-line treatments [57]. If these lesions become symptomatic, patients should have a nasogastric tube placed to facilitate ulcer healing, along with resuscitation with blood or intravenous fluids to address the hypovolemic state (1:1:1 packed red cells:fresh frozen plasma:platelets is preferred).

Infection

Spinal cord injury denervates the spleen and other secondary lymphoid organs. This is thought to contribute to the post-injury immunodeficiency seen in SCI patients. Immunodeficiency increases the risk of a clinically significant infection in patients, especially those with compromised respiratory function [52, 53].

Prognosis and Outcomes of Patients with Spinal Cord Injury

Prognosis of patients with acute SCI revolves around two main concerns: survival and neurological recovery, which can be thought of as the "quantity and quality of life." The latter is strongly predicted by the severity of injury at diagnosis, with over 97% of patients who were *ASIA D* at diagnosis being ambulatory at 1 year postoperatively, compared to a mere 8% of patients who were *ASIA A* at diagnosis [117]. Other factors, including younger age at the time of injury (patients younger than 65 years vs. patients 65 years and older) and greater lower extremity motor strength immediately post-injury, have been demonstrated to positively predict ambulation in multivariable analyses [3, 46, 50]. These factors are also associated with an increased ability to complete post-injury rehabilitation, raising the question of whether older, more severely injured patients generally are less able to recover or whether worse baseline function prevents completion of rehabilitation and subsequently inhibits recovery. At present, the evidence is insufficient to disentangle these two possibilities. However, it is obvious that the poor condition of some SCI patients contributes to the substantial costs of traumatic SCI. Cao et al. reported that direct costs of treatment are directly related to injury severity. For example, among patients with high cervical spine injury, direct costs for those with *ASIA D* injuries were reportedly $359,783, compared to $1,102,403 for patients who were *ASIA A* [2, 118]. These differences persist following discharge, imposing a significant financial burden upon patients and family members [2, 119–122].

Patients with SCI also have lower life expectancies than persons without. As with functional disability and care cost, actuarial survival is highly dependent on the age at injury and the extent of neurological injury [2]. Young patients with mild injury (e.g., *ASIA D*, age 20) have almost normal life expectancies (interval life expectancy 52.9 vs. 59.6 yr), whereas elderly patients (age > 60) with ventilator-dependent injury have drastically abridged life expectancies (3.7 vs. 23.2 yr) [2]. Regardless of grade and age at the time of injury, the strongest predictor of long-term survival appears to be surviving at least 1 year after the injury [3, 42]. During the first post-injury year, death most commonly results from medical complications (e.g., pneumonia and other respiratory diseases) rather than from the neurological injury itself [47]. Of course, the severity of the neurological injury undeniably contributes to mortality risk and to the likelihood of a prolonged inpatient stay [3, 4, 42, 44, 45, 48, 49]. Consequently, there is an almost exponential increase in mortality with increasing injury severity. To this end, it is likely that the significant improvement in overall mortality following SCI seen over the past three decades [3, 48] is at least in part due to the significant improvements in intensive care unit mortality [123]. Current investigations seek to further improve both life expectancy and neurological recovery after SCI, as there is still much room for improvement.

Conclusions

Traumatic SCI is an increasingly common clinical entity that requires multimodal management. Implementation of care must be performed in an expedient fashion and should be customized to include surgical decompression and/or stabilization, medical management of cardiopulmonary function, and post-injury rehabilitation based upon the unique characteristics of the patient and the injury. At present several contested issues remain, most notably the use of high-dose methylprednisolone during the acute injury phase. Similarly, several interventions have been universally recommended (e.g., surgical decompression), but they are supported only by poor-quality evidence. Moving forward, it is incumbent upon practitioners in neurosurgery and critical care medicine to procure data to investigate current interventions and to establish truly evidence-based treatment protocols.

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