Chapter 15
Spinal Cord Injury

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15.1 Introduction

Spinal cord injury (SCI) is one of the most devastating neurological injuries in both developed and developing nations. Worldwide, the incidence of SCI ranges from 3.6 to 195 per million [1]. In the United States, the incidence and prevalence of traumatic SCI are higher than in other developed countries [2]. The estimated annual incidence of SCI in the United States is 40 cases per million, totalling 12,000 new cases per year [3]. There are between 238,000 and 332,000 people with spinal cord injury currently living in the United States [1]. The incidence of SCI in men is four times higher than in women, and the average age of the SCI is increasing as the population continues to age [2]. Mean age of SCI occurrence has shifted from 29 in the 1970s to 40 in the 2010 [3]. While the occurrence of SCI is increasing, its severity is decreasing [2]. The most common syndromic presentations include incomplete tetraplegia

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J.L. White, K.N. Sheth (eds.), *Neurocritical Care for the Advanced Practice Clinician*, DOI 10.1007/978-3-319-48669-7_15

(41%), incomplete paraplegia (19%), complete paraplegia (18%) and complete tetraplegia (12%).

15.1.1 Morbidity and Mortality

An estimated 4,800 victims of SCI die before ever reaching the hospital [4]. For those alive when reaching the hospital, the overall mortality is highest in the first year after the injury, as well as in patients with advanced age and severer clinical pictures. Long term causes of death include infections (septicemia from urinary tract infections, respiratory infections, and decubiti) and pulmonary thromboembolism secondary to deep vein thrombosis. There has been no change in the mortality rate for septicaemia in the past 40 years and only a slight decrease in mortality due to respiratory diseases [5]. SCI is an extremely costly disease. In 2014, the annual expenses during the first year ranged from $347,000 to $1 million, depending on the severity of injury [5]. Each subsequent year ranged from $42,000 to $184,000 [5].

15.1.2 Mechanism of Injury

Motor vehicle collisions remain the leading cause of traumatic SCI in the United States, accounting for 38% of new SCI [5]. Falls, at 30%, are the leading cause of SCI in the geriatric population. Violent injuries account for 14% and sports injuries account for 9%. These mechanisms of injury are more often seen in the younger population [2, 3, 5]. Causes of non-traumatic SCI include disc herniation, metastatic disease of the spine, and spinal stenosis [7].

The majority of traumatic SCI cases compromise the cervical spine, resulting in increased short- and long-term morbidity when compared to thoracic and lumbar injuries [6]. Predictors of mortality include age at injury, neurologic level and clinical presentation (incomplete vs. complete injury) [7].

Spinal cord injuries typically occur when a powerful impact is applied to the spine resulting in fractures, ligamentous disruptions, and cord damage. The classification of SCI is based on location (craniocervical, subaxial cervical or thoracolumbar) or mechanism (flexion, extension, or axial load). There are four mechanisms of primary injury to the spinal cord [8].

- Impact with persistent compression: Seen in burst fractures, whereby bone fragments or an acute disc disruption impinges the spinal cord.
- Impact with transient compression and rapid realignment of the vertebral bodies: Seen in extension injuries causing varying levels of damage to the spinal cord. Transient compression of the cord can be caused by a bulging disc or osteophyte and thickened ligamentum flavum.
- Spinal Cord Injury Without Radiographic Abnormality (SCIWORA): Seen in patients with cervical spondylosis. This is characterized by an absence of radiological evidence of trauma on routine imaging, but a spinal cord contusion and/or edema may be noted on MRI.
- Laceration of the spinal cord: Caused by penetrating trauma, stab or gunshot, or by a bony fragment dislocation.

In thoracolumbar injuries, the "three column" concept states that at least two of the three columns of the spine need to be disrupted for an injury to be unstable, posing risk of further injury to the spinal cord [9]. Medical conditions such as ankylosing spondylitis and spinal spondylosis are the exceptions to this theory, as the underlying abnormalities of the spinal column can result in significant injury to the spinal cord with minimal bony injury.

15.1.3 Primary and Secondary Injury

There are two distinct pathophysiological phases in SCI. The primary phase occurs at the time of the event and is the result of the mechanical insult; this stage constitutes the perfect target for

preventive strategies. The secondary phase occurs minutes to days after the injury and is the focus of currently used medical interventions. Mechanisms for secondary injury include inflammation and edema, as well as vascular, electrolyte and biochemical changes. The severity of the secondary insult is determined by severity of the primary insult and the development of other systemic factors, such as hypotension and hypoxia. The goal of management following SCI is to minimize secondary injury by maintaining hemodynamic stability and optimizing medical status.

15.2 Case Presentation

A 40-year-old man presented to a trauma center following a motor vehicle collision. Upon presentation, his primary survey was significant for a blood pressure of 80/44 mm Hg with a pulse rate of 49. He is breathing comfortably but complaining of shortness of breath. On the secondary survey, he is awake, alert and oriented with 5/5 muscle strength in his biceps bilaterally, 4/5 strength in his wrist extensors on the right, 4/5 strength in his wrist extensors on the left, and 3/5 triceps muscle strength bilaterally. His only sensation below the nipple line was rectal sensation (American Spinal Injury Association (ASIA) 24 B). He is fluid resuscitated until target blood pressure is achieved proceeding with imaging studies. CT scan showed bilateral interfacet dislocation at C6–C7 and anterolithesis of cervical spine 6 over 7, interspinous process widening, and severe canal narrowing. Before MRI he developed worsening respiratory function with the development of “quad breathing” and was intubated. A central line was placed and a norepinephrine infusion was started for a blood pressure of 95/44 mm Hg. The MRI showed injury at C6–C7 with cord signal abnormality extending

from the C5 through T1 levels and C6–C7 anterior and middle column diskoligamentous injuries with a partial tear of the ligamentum flavum. After the MRI, the patient was placed in cervical traction to reduce the fracture, but one facet of the bilateral dislocation could not be reduced under fluoroscopic guidance. He was taken to the operating room for open reduction and posterior spinal fusion. Postoperatively, an MRI showed an increase in the signal abnormality starting at C3 and expending to T2. The patient was transferred to the intensive care unit (ICU) and a mean arterial pressure greater than 85 mm Hg was achieved using norepinephrine. He underwent an anterior cervical diskectomy and fusion on his second day of hospitalization. Reevaluation by the physical therapy team revealed an ASIA of 18 B. Post operatively, his course was complicated by hypotension and bradycardia, treated with enteral albuterol and midodrine and infusions of vasoactive medications. Five days post injury he developed acute ventilatory failure. He underwent a tracheostomy on the seventh day of hospitalization. With aggressive secretion clearance, he was weaned to tracheostomy collar on the 18th day of hospitalization and transferred to an acute rehabilitation facility on the 22nd day of hospitalization.

15.3 Initial Evaluation

The goal of management in patients with SCI is prevention and minimization of secondary injuries. The initial management for a patient with a traumatic SCI is the same as for any patient who sustains a traumatic injury as suggested by the American College of Surgeons' Advanced Trauma Life Support (ATLS®) course [10].

The primary survey identifies life threatening injuries requiring emergent intervention and rapid and systemic evaluation

Table 15.1 Indications for intubation of the patient with traumatic cervical spinal cord injury

Absolute indications
Complete spinal cord injury above C5 level
Respiratory distress
Hypoxemia despite attempts at oxygenation
Severe respiratory acidosis
Relative indications
Complaint of shortness of breath
Development of quad breathing[a]
Vital capacity of <10 mL/kg or decreasing vital capacity
Consideration should be given
Need to travel remote from emergency department (e.g., MRI, transfer to another facility)

[a]Quad breathing refers to the stereotypical breathing pattern in patients with cervical and upper thoracic spinal cord injury in which the chest wall retracts and the abdominal wall protrudes with inspiration (Adapted from Stein et al. [11])

which should be completed in the first minutes of the patient's arrival. See Table 15.1. Following the "ABCDE" approach (airway, breathing, circulation, disability, and exposure), risk of airway loss, inadequate oxygenation and ventilation, and hypotension require immediate attention.

Patients with cervical and high thoracic injuries experiencing impending respiratory failure often describe heaviness in the chest or inability to catch their breath, or may appear breathless while speaking. Loss of chest wall and abdominal innervation produce a pattern where the chest goes in and the abdomen goes out with diaphragmatic contraction, so call "quad breathing". Treatment of respiratory insufficiency in a patient with SCI should include urgent endotracheal intubation [12, 13]. Caution should be employed during intubation as tetraplegic patients can develop bradycardia and hypotension due to autonomic instability.

Once hemodynamic stability it achieved, the secondary survey should begin with a detailed exam to quantify neurologic disability.

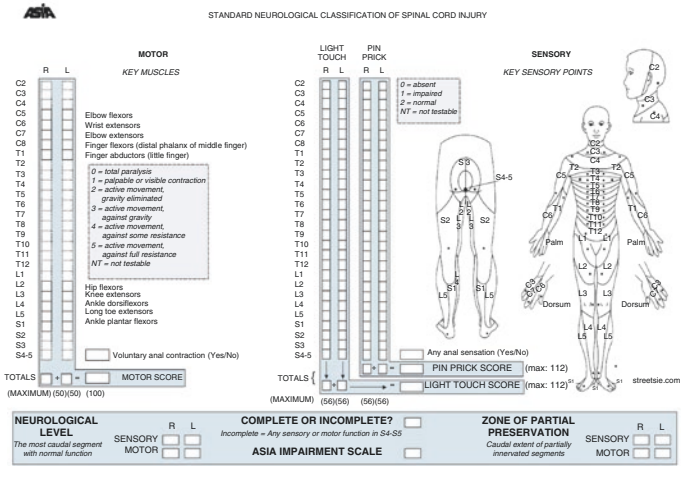
15.3.1 Imaging

CT scan is the modality of choice for initial evaluation. It detects bony injury and dislocation [14].

MRI is useful in detecting injury in the obtunded patient with a suspected SCI where physical exam is less reliable. Additionally, MRI is used for diagnosis, to plan operative interventions, and for prognostication, as it is the modality which determines the degree of edema and hemorrhage in the spinal cord [15].

15.3.2 Evaluation and Classification of SCI

The American Spinal Injury Association (ASIA) developed a standard classification of SCI which is widely used [16] (Fig. 15.1). The ASIA examination tests 2 components; sensory and motor. The sensory examination tests 28 dermatomes bilaterally for two aspects of sensation, light touch and pin prick. The motor examination tests ten paired myotomes bilaterally for strength. The American Spinal Injury Association has also described the ASIA Impairment scale which is the standard for determination of completeness of an SCI. Complete injuries are losses of all sensory and motor function below the level of injury ASIA A. Incomplete injuries retain some neurologic function below the level of injury ASIA B-D. ASIA should be assessed at least daily as a decreased score is usually a result of spinal cord edema ascension, new or worsening spinal cord hemorrhage, or ischemia.



MUSCLE GRADING

0 total paralysis

1 palpable or visible contraction

2 active movement, full range of motion, gravity eliminated

3 active movement, full range of motion, against gravity

4 active movement, full range of motion, against gravity and provides some resistance

5 active movement, full range of motion, against gravity and provides normal resistance

5* muscle able to exert, in examiner's judgement, sufficient resistance to be considered normal if identifiable inhibiting factors were not present

NT not testable. Patient unable to reliably exert effort or muscle unavailable for testing due to factors such as immobilization, pain on effort or contracture.

ASIA IMPAIRMENT SCALE

☐ **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.

☐ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

☐ **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

☐ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

☐ **E = Normal:** Motor and sensory function are normal.

CLINICAL SYNDROMES (OPTIONAL)

☐ Central cord
☐ Brown-Sequard
☐ Anterior cord
☐ Conus medullaris
☐ Cauda equina

STEPS IN CLASSIFICATION

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

1 Determine sensory levels for right and left sides.

2 Determine motor levels for right and left sides.
Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level.

3 Determine the single neurological level.
This is the lowest segment where motor and sensory function is normal on both sides, and is the most cephalad of the sensory and motor levels determined in steps 1 and 2.

4 Determine whether the injury is Complete or Incomplete (sacral sparing).
*If voluntary anal contraction = **No** AND all S4-5 sensory scores = **0** AND any anal sensation = **No**, then injury is COMPLETE*
Otherwise injury is incomplete.

5 Determine ASIA Impairment Scale (AIS) Grade:
Is injury Complete?
NO → **If YES,** AIS=A Record ZPP (For **ZPP** record lowest dermatome or myotome on each side with some (non-zero) preservation)
Is injury motor incomplete?
YES → **If NO,** AIS=B (Yes=voluntary anal contraction OR motor function more than three levels below the motor level on a given side.)
Are at least half of the key muscles below the (single) neurological level graded 3 or better?
NO → AIS=C
YES → AIS=D

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

Fig. 15.1 ASIA Standard neurological classification of spinal cord injury (From International Standards for Neurological Classification of Spinal Cord Injury [16])

15.3.3 Specific Clinical Syndromes [11]

Central cord syndrome

- Most common, seen in older adults
- Extensive weakness noted in the upper extremities compared to the lower extremities
- Existing degenerative ligament and/or osteophytic changes to spine prior to hyperextension injury of the neck causing the spinal cord to be squeezed or pinched
- Usually not associated with a bony injury or evidence of spinal instability

Brown-Sequard syndrome

- Typically from penetrating spinal cord injury or a lateral mass fracture of the spine
- Hemiplegia with ipsilateral loss of light touch and proprioception with contralateral loss of pain and temperature sensation

Anterior cord syndrome

- Motor and sensory pathways in the anterior part of the spinal cord are injured
- Cause is not usually traumatic
- Ischemic insult from disruption of flow in the anterior spinal artery
- Poor prognosis for recovery

Posterior cord syndrome

- Result of vascular compromise to the spinal cord
- Rarely occurs from trauma
- Posterior aspect of spinal cord affected
- Loss of proprioception with preservation of motor function, pain and temperature sensation and light touch

Cauda equina syndrome

- Traumatic cause is typically retropulsion of a fracture fragment in the lumbosacral region resulting in lower spinal nerve root compression
- Non traumatic cause most commonly results from a massive herniated disc in the lumbar region
- Other non-traumatic causes are spinal lesions/tumors, lumbar stenosis, spinal hemorrhages, spinal arteriovenous malformations, birth abnormalities, spinal anesthesia
- May include one or more symptoms:
  - Severe low back pain
  - Motor weakness/sensory loss or pain in one or both legs
  - Saddle numbness
  - Recent onset of bladder dysfunction (incontinence or retention)
  - Recent onset bowel dysfunction
  - Abnormal sensation in the bladder or rectum
  - Recent onset of sexual dysfunction
  - Loss of reflexes in the lower extremities

15.3.4 Spinal Shock

Spinal shock was first described by Whytt in 1750 as the loss of sensation with motor paralysis with gradual recovery of reflexes [17]. Following spinal cord injury, the reflexes above the injury level remain intact while the reflexes below the injury level become depressed or absent. In SCI, the term ‘shock’ does not refer to the circulatory system, and should not be confused with neurogenic shock. Ditunno et al. [18] described spinal shock in a four phase model.

- Phase 1: areflexia or hyporeflxia, 0–24 h post injury.

- Phase 2: reflexes return, 1–3 days post injury.
- Phase 3: early hyper-reflexia, day 4 to 1 month post injury.
- Phase 4: spascity/hyper-reflexia, 1–12 months post injury.

15.4 Management and Interventions

The majority of treatment for patients with spinal cord injury is supportive, with focus on minimizing secondary injury, and preventing and treating complications as they occur. Patients with injury in the cervical or high thoracic spine are at high risk of organ failure and require high-level intensive care support [19]. It should be expected that over hours to days following injury the neurologic deficits will worsen, which in turn will trigger further cardiovascular and respiratory dysfunction.

15.4.1 Neurogenic Shock

Neurogenic shock can occur in patients with SCI on or above T6. It is caused by the loss of supraspinal control of the sympathetic nervous system [20, 21]. This causes hypotension and stimulation of the vagus nerve, with unopposed parasympathetic activity leading to bradycardia and the block of the atrioventricular node [21]. Neurogenic shock is a form of distributive shock with excessive vasodilatation and the characteristic finding of bradycardia. Patients are usually hypotensive with warm and dry skin. Neurogenic shock may not be present on admission but can develop over days to hours and last for 1–3 weeks. The first line therapy is fluid resuscitation to maintain euvolemia. Second line treatment is the use of pressors, inotropes or a combination of both (Table 15.2). Treating hypotension and hypoperfusion is

Table 15.2 Vasoactive agents used to treat neurogenic shock

Agent	α Activity	β Activity	Considerations
Norepinephrine	+++	++	Probably the preferred agent
Phenylephrine	++	None	May worsen bradycardia
Dopamine			
Low dose (3–10 mcg/kg/min)	+	++	May lead to inadvertent diuresis at low dose
High dose (10–20 mcg/kg/min)	++	+++	
Epinephrine	+++	++	Rarely needed
Dobutamine	None	+++	May cause hypotension if not euvolemic

Adapted from Stein et al. [11]
+ small effect, ++ moderate effect, +++ large effect

paramount, as these are known mediators of secondary injury. Severe hemodynamic abnormalities will ultimately resolve after the first 2–6 weeks post injury, however patients with SCI can have life-long alterations in cardiovascular function.

15.4.2 Cardiovascular Evaluation and Management

Aggressive management of hypotension is recommended and is associated with improvements in neurologic outcome [14, 22]. Treatment of hypotension and neurogenic shock following spinal cord injury initially involves volume resuscitation followed by pressor administration as outlined above. Current recommendations are to maintain mean arterial blood pressure between 85 and 90 mm Hg for the first 7 days following acute cervical spinal cord injury [22]. Oral α-receptor agonists, such as midodrine hydrochloride and pseudoephedrine, can be used

with IV vasoactive medications and may be helpful in the subacute stages following injury for management of persistent hypotension [23].

Treatment of bradycardia following spinal cord injury is typically reserved for symptomatic patients. For symptomatic cases, use of β-agonist therapy with enteral albuterol may be helpful, atropine can be used for episodic bradycardia, and pacemaker placement may be indicated if patients have persistent symptomatic bradycardia.

Some patients will have recurrent and sustained bradycardia that can progress to asystole if left untreated [20, 21].

15.4.3 Respiratory Evaluation and Management

Respiratory dysfunction occurs in over 65% of patients with cervical SCI. Of these, 40% meet criteria for respiratory failure by standardized organ dysfunction scales [19]. The primary cause of respiratory failure is intrinsic dysfunction from denervation of the muscles essential for adequate ventilation, resulting primarily in hypercarbia. Trauma patients may have concomitant injuries such as pulmonary contusions, hemothoraces and pneumothoraces also compromising respiratory function through hypoxemic failure. Patients with high cervical and thoracic injuries who do not require immediate intubation need close observation as the injury progression in the spinal cord from edema and ischemia may worsen respiratory function. Increased respiratory secretions, ineffective cough, and increased bronchial tone are additional contributory factors in respiratory dysfunction.

Tracheostomy placement is sometimes indicated for both prolonged ventilation and secretion management. In general, admission ASIA score of less than 10 will require tracheostomy [24].

If tracheostomy is indicated, early tracheostomy may reduce ICU length of stay and decreased time of mechanical ventilation.

15.4.4 Ventilator Management

Patients with spinal cord injury have a high incidence of pneumonia and benefit of a protocolized ventilator management [25]. Complete high cervical spine injury at the C1 to C3 with invariably need mechanical ventilation due to loss of diaphragm innervation (Table 15.3). Ventilator weaning can be challenging for many SCI patients and high and/or complete injuries may require long-term mechanical ventilation.

Initial ventilation of the tetraplegic patient should be aimed at a higher tidal volume; risk of barotrauma should be reduced if peak airway pressure is kept under 40 [22].

Table 15.3 The three major muscle groups of the respiratory system

Muscle group	Function	Innervation
Diaphragm	Major muscle of respiration	C3 to C5
	During inhalation, the diaphragm contracts and moves downward	
	During exhalation, the diaphragm relaxes, allowing for passive recoil	
Intercostal muscles	During inhalation, the external intercostal muscles contract and elevate the rib cage	T1 to T11
	During exhalation, the internal intercostal muscles contract and pull the ribs down	
Abdominal muscles	Essential for an effective cough	T6 to L1
	During exhalation, the abdominal muscles contract and compress the abdominal contents and push the diaphragm up	
Accessory muscles	Elevate the rib cage and assist in deep ventilation	C1 to C3
	Inadequate alone for effective ventilation	

Adapted from Stein and Sheth [26]

Early aggressive pulmonary therapy is associated with decreased time on the ventilator, fewer pulmonary complications and improved survival [27]. These include:

- Assisted coughing techniques including manual assistance and devices that assist by delivering a deep breath and during exhalation with a rapid reversal of flow which helps expectoration of secretions
- Chest physiotherapy and positioning
- Intrapulmonary percussive ventilation (IPV) which can be utilized for patients to mobilize mucous and improve delivery of nebulized medications

Abdominal binders are helpful during the acute phase as they keep the abdominal contents from protruding and have a traction effect on the diaphragm [27]. Or in prolonged cases, diaphragmatic pacer implantation for patients with high cervical SCI allows for the possibility for these patients to wean from full ventilator support [28].

15.4.5 Other Systems and Considerations

Complications are the leading cause of morbidity and mortality in SCI patients. The leading cause of death is infection. Screening and treatment of infection is extremely important. These patients are also susceptible to deep vein thrombosis (DVT), skin breakdown, gastrointestinal hemorrhage and pulmonary embolism.

15.4.6 Urinary Tract

Timely Foley catheter removal and urinary tract infection (UTI) prevention are of paramount importance. Consider removal of indwelling urinary catheter once the patient is hemodynamically stable.

Once Foley catheters are removed, patients often require intervention to ensure adequate bladder emptying. Intermittent catheterization of the bladder can be done every 4–6 h, with the goal of keeping bladder volume below 500 ml to prevent over-distention. Suprapubic catheters should be considered with urethral abnormalities, recurrent urethral obstructions, and difficulty with urethral catheter insertion. If patient is voiding without catheterization, scan bladder or catheterize at least once after voiding to ensure the bladder is emptying and there is not a high post-void residual.

15.4.7 Gastrointestinal Tract

Maintenance of adequate nutrition is extremely important and enteral nutrition should be started as soon as feasible. A formal swallow evaluation is recommended prior to oral food and liquids as dysphagia can be present in up 41% of patients with tetraplegia [14, 27]. Acute SCI patients are at high risk for gastrointestinal bleeding and should be prescribed stress ulcer prophylaxis.

Initiate a bowel training program early as patients with SCI can have decreased bowel motility. Bowel distention, ileus, and inadequate evacuation can occur and lead to nausea, vomiting, and high gastric residuals which could lead to aspiration and other complications. Aim for one bowel movement per day with the combined use of oral and rectal medications and digital stimulation as needed.

15.4.8 Venous Thromboembolism (VTE) Prevention

At a minimum, pneumatic compression stockings and mechanical compression should be used in all patients from the time of admission. Prevention of VTE with chemical prophylaxis such

as low molecular weight heparin is typically acceptable to start within 72 h of injury. Discuss timing with surgeon if the patient underwent operative fixation.

The placement of a prophylactic inferior vena cava filter (IVC) recommended only when use of chemical VTE prophylaxis is contraindicated. Recommended duration of treatment is 3 months.

15.4.9 Skin

Skin breakdown is one of the more common complications following SCI and prevention must start in the acute setting. Skin assessment and pressure reduction using specialty mattress and padding with frequent turning and repositioning is required. Temperature should be monitored and regulated. The disruption of the autonomic nervous system in patients with cervical and high thoracic SCI can result in poikilothermia where surrounding temperature will be assumed by the body [14].

15.4.10 Pain

Pain can be muscular or neuropathic or both. Consider the use of muscle relaxants to treat spasm as well medications to treat neuropathic pain in addition to opioids. Patients with an incomplete SCI may have allodynia – a hypersensivity to touch. In these patients attempt to minimize pain by decreasing contact with the area as much as possible.

15.4.11 Autonomic Dysreflexia [29]

Autonomic dysreflexia can occur following SCI and may affect patients in the acute or chronic phase. It is more typically seen in patients with high thoracic or cervical spinal cord injuries.

Any noxious stimulus (bladder distension or fecal impaction) which occurs below the level of injury can lead to a dangerous rise in systolic blood pressure due to hyperactive thoracic sympathetic reflex activity, a loss of supraspinal sympathetic control, and inadequate parasympathetic response. Autonomic dysreflexia is defined as a greater than 20% increase in systolic blood pressure with a change in heart rate and at least one sign (e.g., sweating, piloerection, facial flushing) or symptom (e.g., headache, blurred vision, stuffy nose).

Although prevention is ideal, once autonomic dysreflexia occurs, first-line therapy is to remove the stimuli. If hypertension persists, pharmacologic intervention should be instituted with calcium channel blockade or nitrates. If left untreated, malignant hypertension can result in intracranial hemorrhage, retinal detachment, seizures, coma, myocardial infarction, pulmonary edema, and death.

15.4.12 Prognosis

Patients and families will ask about potential for neurologic recovery. The ASIA impairment scale offers some information when discussing prognosis as complete injuries are less likely to recover neurologically than incomplete injuries. Overall life expectancy is shorter for patients with SCI as compared to the general population. Mortality rates are the highest in the first year following injury regardless of age or severity of injury.

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