

Trauma: Spinal Cord Injuries

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Purpose

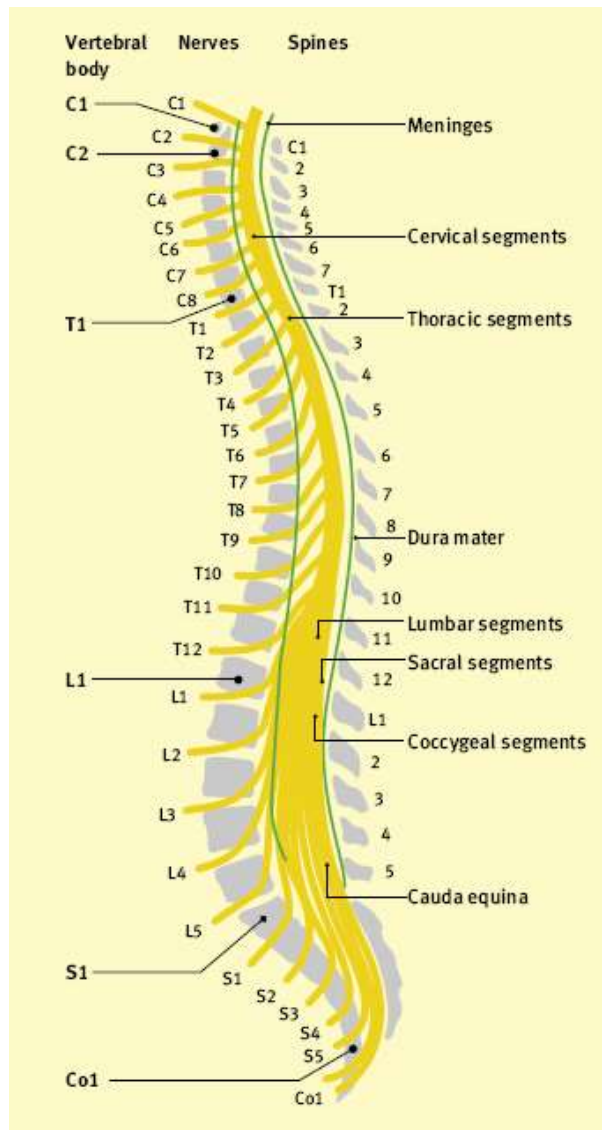
The purpose of this course is to explain different types of traumatic spinal cord injuries, including primary and secondary injuries, assessment, and management.

Goals

Upon completion of this course, the healthcare provider should be able to:

- Describe the anatomy of the spinal cord.
- Describe the 4 sections of the vertebral column.
- List the differences between upper motor neuron and lower motor neuron damage.
- Describe the functions related to different levels of spinal nerves.
- Describe the ABCDEs of initial assessment.
- Discuss secondary assessment.
- Describe the Glasgow Comas Scale,
- Describe 3 criteria for classification of spinal cord injuries.
- Describe sensory, manual muscle, and reflex testing.
- Describe the ASIA Impairment Scale (AIS).
- Describe the Canadian C-spine rule.
- Describe the pathophysiology of spinal cord injury.
- Differentiate among 3 types of shock.
- Discuss respiratory complications.
- Discuss the use of steroids and traction.
- Discuss management of DVT, PE, urinary retention, thermoregulation, pressure sores, GI problems, metabolic abnormalities, and autonomic dysreflexia.
- Differentiate between quadriplegia and paraplegia.
- Describe 6 cord syndromes.

Introduction



Spinal cord injuries have resulted in paralysis of over 1.25 million people in the United States with about 10,000 new injuries each year. The reasons vary, but work injuries (28%), motor vehicle accidents (24%) and sporting accidents (16%), primarily diving, cause the most injuries. In many cases, people suffer from multiple traumas and may, for example, also have brain injury.

Young males are the most at risk for spinal cord injuries, and gunshot wounds are an increasing cause of injury.

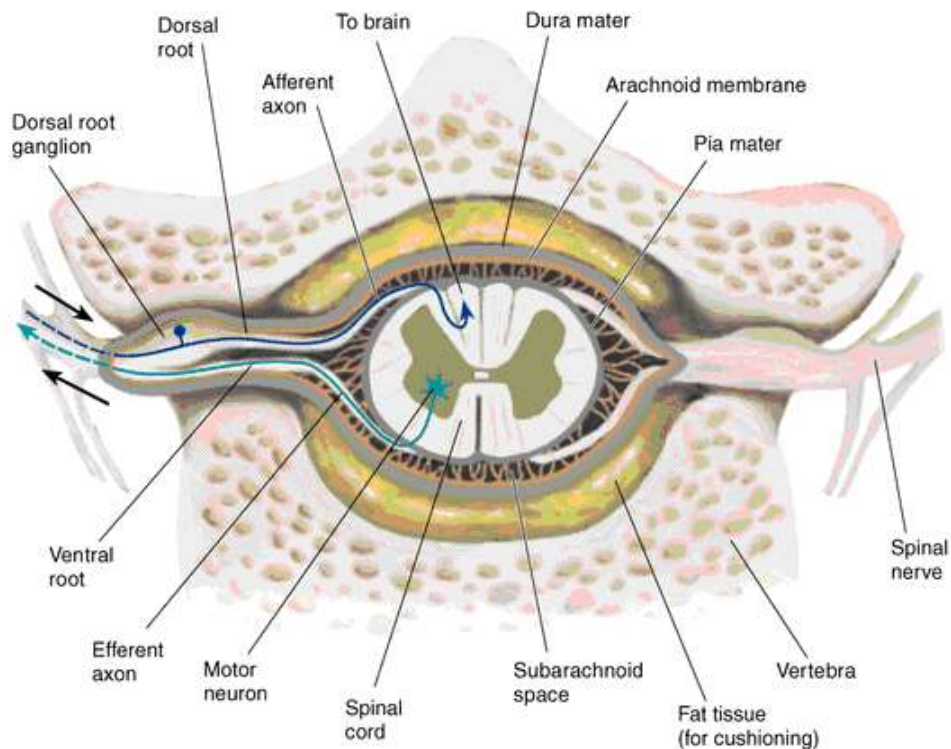
Approximately half of all spinal cord injuries involve the cervical spine (primarily C4 to C7), and half of spinal cord injuries result in complete quadriplegia.

The spinal cord extends as a continuous structure from the medulla at the base of the skull to the first lumbar vertebra (L1), where it tapers into a fibrous band called the *conus medullaris*.

At L2 the nerve roots (*cauda equina*) extend beyond the conus. The spinal cord is approximately 18 inches (45 cm) long in an adult and about finger width.

The vertebral column comprises 7 cervical, 12 thoracic, 5 lumbar, and 5 fused sacral vertebrae that protect the spinal cord. Intervertebral discs and facet joints cushion and allow movement. Nerve roots exit from the vertebral column through the intervertebral foramina (openings).

► **Cross Section of the Spinal Cord, Showing Route Taken by Afferent and Efferent Axons**



In the spinal cord, gray matter is at the center in an H-shape and is surrounded by white matter that contains both afferent (ascending) and efferent (descending) nerve fibers. Like the brain, the spinal cord is surrounded by the meninges.

The spinal cord contains 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each nerve has a dorsal root and a ventral root. The dorsal roots transmit sensory information, such as temperature, proprioception, touch, and pain, from specific areas of the body, known as dermatomes. The ventral roots transmit motor impulses.

There are 6 ascending tracts: 2 conduct sensation (touch, vibration, position, pressure), 2 conduct sensory impulses necessary for coordinated muscle contraction, and 2 conduct sensation of pain, temperature, fine touch, and vibratory sense from the upper body.

There are 8 descending tracts (upper motor neurons): 2 conduct muscle impulses and control voluntary muscle activity, 3 involve autonomic functions (perspiration, circulation, pupil dilation) and involuntary muscle control, 1 conducts impulses for voluntary head

and facial muscle movement, and the last 2 involve voluntary muscle movement.

Some of the motor nerve pathways, contained in the spinal cord, represent the pathways of the extrapyramidal system (making connections from the anterior horn cells to the automatic control centers in the brain) and others are components of reflex arcs. Spinal cord injury can result in lesions of upper motor neurons and/or lower motor neurons.

Upper motor neuron damage	Lower motor neuron damage
<ul style="list-style-type: none"> • Loss of voluntary control (paralysis). • Increased muscle tone. • Muscle spasticity. • No muscle atrophy. • Hyperreflexia. 	<ul style="list-style-type: none"> • Loss of voluntary control (paralysis). • Decreased muscle tone. • Muscle flaccidity. • Muscle atrophy. • Hyporeflexia.

The nerves at different levels of the spinal column control various functions, so injuries result in predictable outcomes.

Level	Functions
C1-C6	Neck flexors.
C1-T1	Neck extensors.
C3, C4, C5	Innervate diaphragm.
C5, C6	Shoulder movement. Raises arm (deltoid). Flexes elbow (biceps).
C6	Supinates arm.
C6, C7	Extends elbow (triceps), wrist (extensors). Pronates wrist.
C7, T1	Flexes wrist. Innervates small muscles of hand.
T1-T6	Innervates intercostals and upper trunk (above waist).
T7-L1	Innervates abdominal muscles.
L1-L4	Thigh flexion
L2-S1	Thigh abduction
L5-S2	Leg extension at hip (gluteus maximus). Plantar flexion of foot. Toe flexion.

L2-L4	Leg extension at the knee (quadriceps femoris).
L4-S2	Leg flexion at the knee (hamstrings).
L4-S1	Toe extension.
S2-S4	Bladder and bowel function/control.

Initial assessment/Intervention

With suspected spinal cord or vertebral injury, the patient should be immediately immobilized as an estimated 3 to 25% of injuries to the spinal cord occur during transport or resuscitation. All patients with pain along the spine or paresis/paralysis should be assumed to have spinal cord injuries until appropriate evaluation can be completed.

Primary assessment

The standard ABCDE evaluation should be completed as well as cranial nerve assessment, followed by a more extensive neurological examination once the patient has stabilized. Because spinal cord injuries are often associated with other types of injuries, such as traumatic brain and/or abdominal injuries and fractures, the evaluation must focus on identifying all possible injuries.

Airway	<ul style="list-style-type: none"> Examine the airway for obstructions, such as loose teeth, foreign bodies. Lacerations and bone instability may be obstructive. Examine the trachea for deviation and observe for signs of circumoral cyanosis (sign of hypoxia). Auscultate the airway and listen for turbulence. With spinal cord injury, prevertebral swelling and hematoma may occur, and this can compromise the airway.
Breathing	<ul style="list-style-type: none"> Immediate intubation may be indicated for high cervical injuries, but care must be used to avoid flexion of the neck. Manual inline immobilization or fiberoptic intubation is recommended. Neurologic status must be assessed along with pulmonary and respiratory function. High diaphragmatic/abdominal breathing is an indication of high cervical injury.
Circulation	<ul style="list-style-type: none"> Monitor blood pressure, pulse, temperature, color, and indications of cyanosis (circumoral, peripheral), including oxygen saturation continuously. Use venous access to restore intravascular volume, BP, and perfusion.

	<ul style="list-style-type: none"> • Evaluate possible causes for hypotension, a common finding with SCI, often indicating bleeding from other injuries. Hypotension found with bradycardia often indicates spinal cord injury. • Note skin temperature. Warm skin may indicate adequate perfusion or neurogenic shock.
Dysfunction/ disability	<ul style="list-style-type: none"> • Assess responsiveness Glasgow Coma Scale (GCS). • Assess neurological status. • Assess motor ability by observation, pressure to nail bed, or sternal rub: <ul style="list-style-type: none"> ○ Decreased spontaneous movement and/or flaccidity may be associated with local injury or spinal cord injury. • Assess reflexes to determine level of injury and integrity of the spinal cord. • Immobilize patient with rigid backboard and cervical spine collar until spinal cord injury is ruled out.
External examination	<ul style="list-style-type: none"> • Note lacerations, fractures, edema, and bruises.

Secondary assessment

Once life-threatening injuries are addressed, a secondary examination should be completed, including a head to toe examination and complete neurological examination and manual of the spine. A manual examination can be done by sliding the hand along the spine as the patient lies in supine position or by positioning the patient with a carefully supported partial log roll for direct observation.

The abdomen must be carefully assessed for traumatic injuries as paralysis with lack of sensation and diaphragmatic breathing may mask typical symptoms, such as abdominal swelling and pain.

About 10% of those with unstable spinal cord injuries have a second injury as well. Priapism (persisting for at least 4 hours) is an indication of a high spinal cord lesion.

Note: Patients should not be left on a rigid spinal board for more than 30 minutes but should be placed on a pressure-relieving mattress.

Glasgow Coma Scale

Because half of spinal cord injuries involve the cervical spine (C4-C7) and other injuries are common, all patients with possible

spinal cord injuries should be assessed for level of consciousness. A number of different grading systems are used, but the Glasgow Comas Scale is the most common.

Glasgow coma scale	
Eye opening	4: Spontaneous. 3: To verbal stimuli. 2: To pain [not of face]. 1: No response.
Verbal	5: Oriented. 4: Conversation confused, but can answer questions. 3: Uses inappropriate words. 2: Speech incomprehensible. 1: No response.
Motor	6: Moves on command. 5: Moves purposefully to respond to pain. 4: Withdraws in response to pain. 3: Decorticate posturing [flexion] in response to pain. 2: Decerebrate posturing [extension] in response to pain. 1: No response.

The total possible scores range from 3 to 15, with lower scores indicating increasing morbidity. Injuries and/or conditions are classified according to the total score:

- **Mild** (80%): GCS score 13 to 15 with brief period of loss of consciousness (LOC). Prognosis is good and mortality rates are <1%.
- **Moderate** (10%): GCS score 9 to 12. Patient is usually confused but able to follow simple commands. Patient may have focal neurological deficits. Prognosis is good with mortality rate <5%.
- **Severe** (10%): GCS of =/<8 (coma). Patient is unable to follow commands and requires airway control. ICP is often elevated and the cause of death or disability. Mortality rates are about 33%. Patients who survive usually have significant disabilities.

Note: When documenting the GCS score for patients who are intubated ("tubed"), this should be indicated when reporting the score:

- Intubated: 9 [T].
- Intubated and pharmacologically paralyzed 9[TP].

Neurological assessment and classification of injury

Spinal cord injuries are classified according different criteria:

- **Mechanism of injury:** Flexion, hyperextension, flexion-rotation, extension-rotation, and compression. The unstable is the flexion-rotation injury because damage to the ligaments that provide stability to the spine may occur.
- **Level of injury:** Skeletal (vertebral) and neurological (lowest segment with normal sensory and motor functions bilaterally).
- **Degree of injury:** Complete or incomplete.

Sensory testing

Sensory levels are evaluated bilaterally at 28 key sensory points (note dots on following diagrams) in the dermatomes that correspond to different sensory nerves. Bilateral assessment is done with both light touch and pinprick for levels C2 through S4-5 (perineal area). The following assessment scale is utilized for each key point (both for touch and pinprick).

Score	Sensation
0	No sensation.
1	Altered, impaired sensation.
2	Normal sensation.

The maximum normal score is 118 for light touch and 118 for pinprick (2 X 28 = 56 X 2 for R and L sides = 118). In addition, the patient is assessed for deep anal pressure.



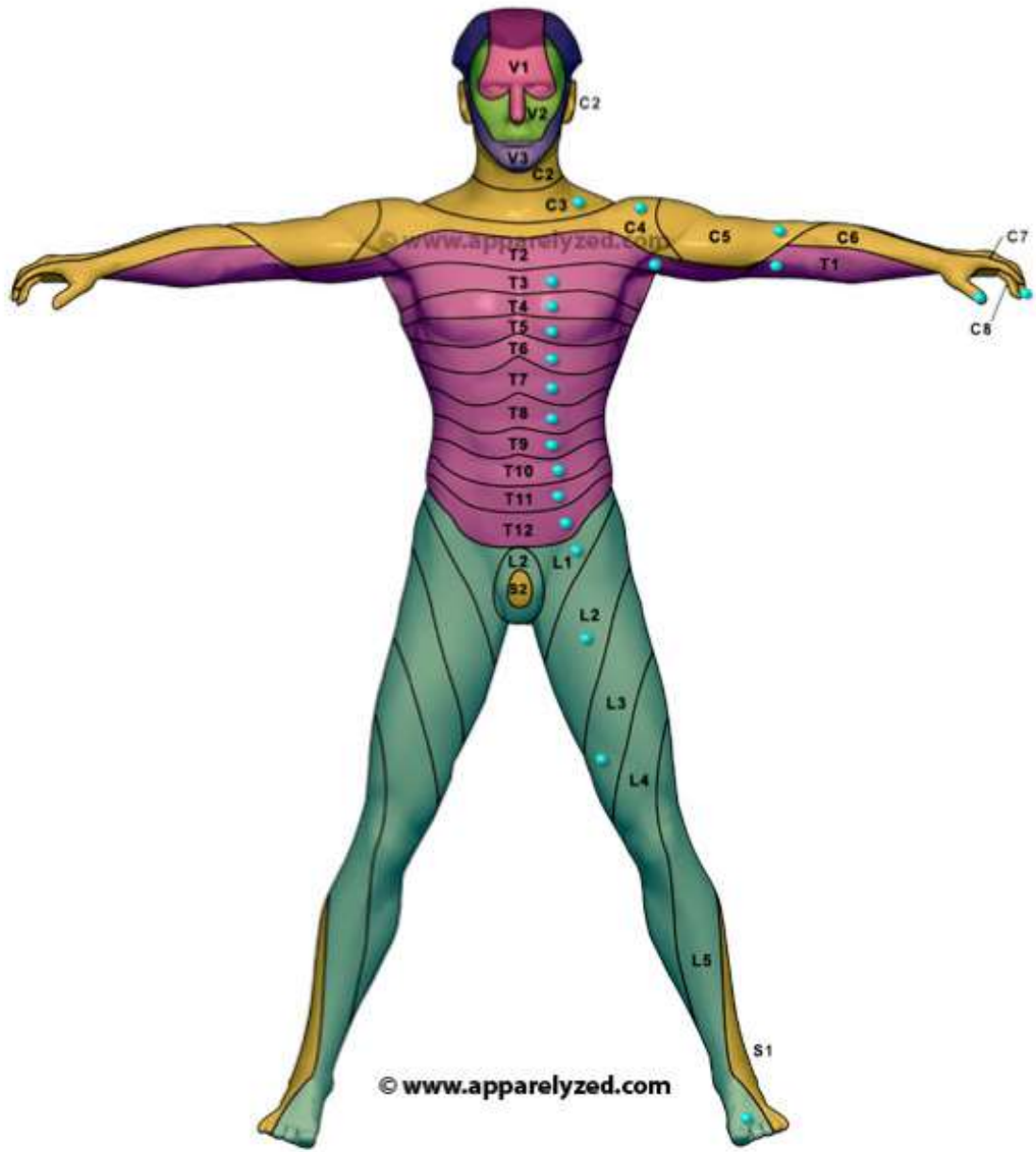
used for pinprick testing.

Before assessing sensation, the examiner should conduct a test on unaffected areas, such as the face, to determine if the patient is able to distinguish a sharp sensation (as from a pinprick) from dull or light touch pressure alone and can differentiate. Light touch is assessed with a cotton swab or strand of cotton or in some cases a gloved hand. A disposable pin is

When assessing, the sensation is graded as normal if it is the same as in the unaffected area but as altered if it is perceived differently, such as when a pinprick is felt as less sharp. If the pinprick is perceived only as touch or pressure, it is graded as absent. If the patient has an area of hyperesthesia, all touch may be perceived as sharp, so if the patient cannot differentiate between dull and sharp (such as opposite ends of a pin), pinprick is graded as absent.

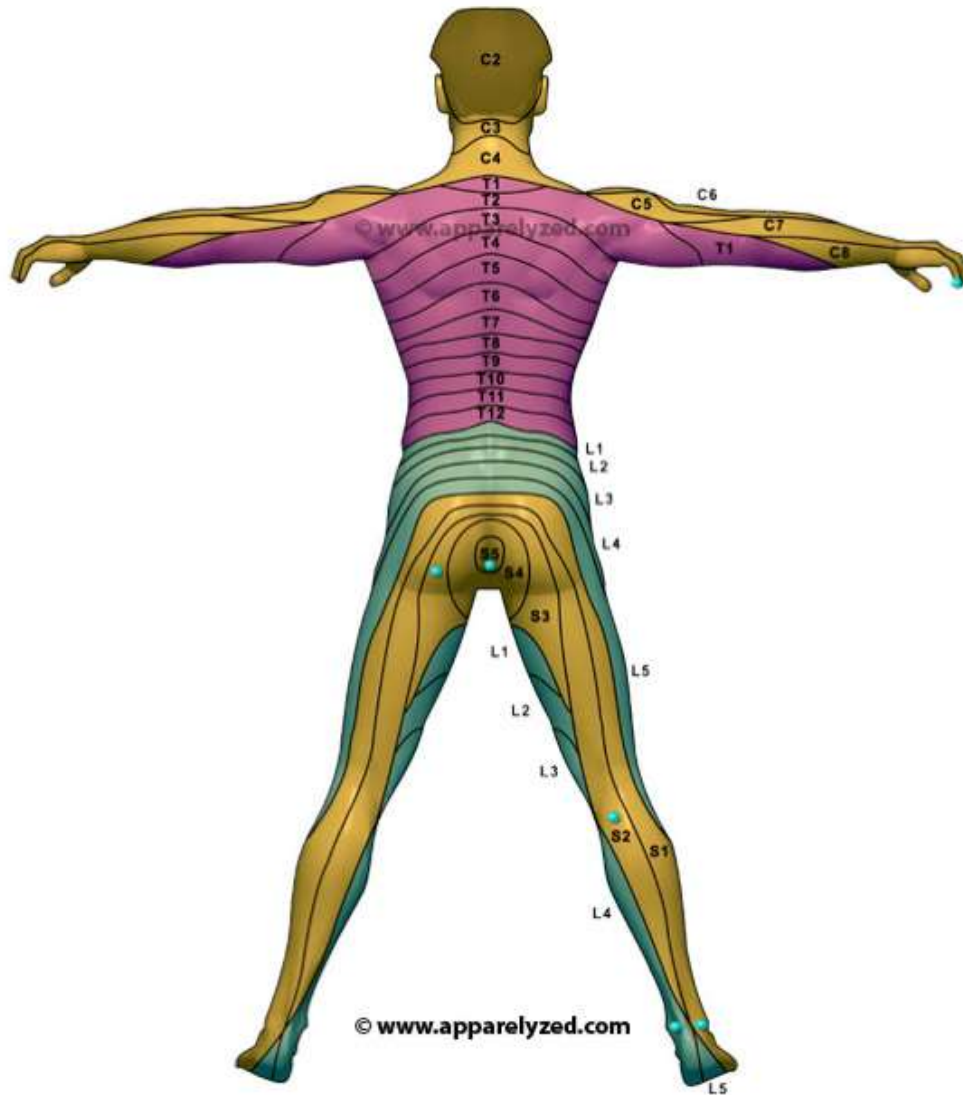
Deep anal pressure is tested by an external rectal examination to determine if the patient feels a sensation of pressure as the finger is moved against the anal sphincter. Any reliable perception of pressure indicates an incomplete injury.

T3 assessment can be problematical because of differences in the distribution of the dermatome because the C4 dermatome extends to varying distances down anterior chest wall. If T3 is tested too high on the chest wall, it may indicate innervation by C4 rather than T3. Therefore, if sensation seems to be present in T3 but is absent in T1, T2, and T4, then T3 is graded as absent.



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It's important to note that deep pressure sensation may sometimes be present even in the absence of sensation from light touch or pinprick but is not considered as part of the evaluation and does not indicate incomplete injury unless it is evident in the perineal area.

Manual motor testing

Motor function is assessed by manual motor testing (MMT) for both the upper and lower extremities bilaterally at key muscles to determine the level of spinal cord injury.

Arm		Leg	
C5	Elbow flexors (Biceps)	L2	Hip flexors
C6	Wrist extensors	L3	Knee extensors

C7	Elbow extensors (Triceps)	L4	Ankle dorsiflexors
C8	Finger flexors (distal phalanx of middle finger)	L5	Long toe extensors
T1	Finger abductors (5 th finger)	S1	Ankle plantar flexors



The muscle function grading system is used to evaluate each muscle with scores ranging from 0 (total paralysis) to 5 (normal movement). A perfect score with no motor impairment is 50 (25 for arm muscles and 25 for leg muscles).

Additionally, the anal sphincter should be evaluated to determine if the patient has voluntary contraction. The examiner inserts a finger into the rectum and asks the patient to tighten the sphincter as though holding in a bowel movement. This is graded as absent or present for contraction. Voluntary contraction

indicates an incomplete injury.

Score	Muscle function
0	Complete paralysis.
1	Palpable or visible muscle contraction.
2	Active movement with full ROM with gravity eliminated.
3	Active movement with full ROM against gravity.
4	Active movement with full ROM against gravity and moderate resistance in a muscle specific position.
5	Normal active movement with full ROM against gravity and full resistance in a muscle specific position in an otherwise unimpaired individual.
5*	Same as above except for identified inhibiting factors, such as pain or disuse). Movement, ROM against gravity, and resistance are sufficient to be considered normal.
NT	Not testable (immobilization, severe pain, coma, limb amputation, contracture of >50 of ROM).

Note that other muscles, such as the diaphragm, deltoid, and abdominals, should also be observed and graded to evaluate progression of symptoms even though not part of the official grading system for muscles.

Proper positioning is especially important for accurate grading. For example, the forearm should be placed horizontally across the chest when testing for the triceps. The examiner should determine the passive range of motion of the joint for the muscle being evaluated and should position the body part and stabilize proximal to the part being tested. Resistance should be applied perpendicularly.

Reflex testing

Some reflexes (such as anal wink and Babinski) are graded as simply present or absent, but extremity reflexes are graded according to a 0-4 point scale:

- 0 = Absent reflex activity.
- 1 = Decreased reflex activity.
- 2 = Normal reflex activity.
- 3 = Increased reflex activity.
- 4 = Markedly exaggerated reflex activity.
-



Weak or absent responses often indicate damage to the peripheral nerves or motor neurons. Excessive responses may indicate spinal cord damage. Reflexes should be checked bilaterally.

Reflex	Discussion
Biceps (C5)	Test by tapping biceps tendon, which should cause the muscle to contract, flexing the elbow.
Triceps (C7)	Test by tapping triceps tendon, which should cause extension of the elbow.
Patellar (L4)	Test by tapping patellar tendon, which should cause extension of the leg.
Achilles (S1)	Tap Achilles tendon, which should cause plantar flexion.
Jaw jerk	Exaggerated response indicates injury at or above the pons.
Deltoid (C5)	Usually associated with hyperreflexia.
Pectoral, Superficial abdominal (T9-T12)	Pectoral reflex usually associated with hyperreflexia. Presence of hyperreflexia and loss of

	superficial abdominal reflex, and Babinski indicates injury to spinal cord or conus medullaris.
Bulbo- or cliterocavernositis (S3-S4)	May be retained with complete injury but lost during period of spinal shock, reappearing when shock resolves.
Anal wink (S5)	The anal wink reflex is tested by stroking the skin about the anus, causing a reflex contraction of the anal sphincter. It is graded as absent or present with absence indicating disruption of the reflex arc.
Babinski	Graded as present or absent. The Babinski response (toe moves toward the top of the foot and other toes fan out) is normal in infants but abnormal after age 2. If present, it indicates damage to nerve paths connecting the spinal cord and the brain.

ASIA Impairment Scale (AIS)

The American Spinal Injury Association (ASIA) Impairment Scale (AIS), incorporating the

International Standards for Neurological Classification of Spinal Cord Injury (ICOS) and modified in 2010, is completed after the following are determined:

- **Sensory levels** (right and left): Based on testing of dermatomes.
- **Motor levels** (right and left): Based on manual muscle testing.
- **Single neurological level:** Based on the lowest segment in which sensory and motor function are normal on both sides (the most cephalad of the sensory and motor levels).
- **Complete/Incomplete** (absence or presence of sacral sparing): Complete injury is indicated by no voluntary anal contraction, sensory scores of 0 for S4 and S5, and no sensation of deep anal pressure. Otherwise, it is incomplete.

Grade	AIS
A= Complete	No sensory or motor function preserved in the sacral segments (S4-S5).
B = Sensory incomplete	Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5 (light touch, pin prick at S4-S5: or deep anal pressure (DAP)), AND no motor function is preserved more than three

	levels below the motor level on either side of the body.
C = Motor incomplete	Motor function is preserved below the neurological level**, and more than half of key muscle functions below the single neurological level of injury (NLI) have a muscle grade less than 3 (Grades 0-2).
D = Motor Incomplete	Motor function is preserved below the neurological level**, and at least half (half or more) of key muscle functions below the NLI have a muscle grade > 3.
E = Normal	If sensation and motor function as tested are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI or observable deficits does not receive an AIS grade.

When assigning the AIS grade those receiving a grade of C or D must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body. Present standards allow even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B versus C).

NOTE: When assessing the extent of motor sparing below the level to distinguish between AIS B and C, the **motor level** on each side is used; whereas to differentiate between AIS C and D (based on proportion of key muscle functions with strength grade 3 or greater), the **single neurological level** is used.

Diagnostic radiography

Canadian C-spine rule

If patients are alert with no symptoms of vertebral/spinal cord injury and GCS of 15 and no drug or alcohol use, the Canadian C-spine rule is often used to determine the need for radiography.

1. High risk factors that mandate radiography (YES):

- Age \geq 65
- Dangerous mechanism
 - Fall from \geq 3 feet/5 stairs.
 - Axial load to head (diving).

- Motor vehicle accident, high speed (>62 MPH), rollover, ejection. Rollover, ejection.
- Motorized recreational vehicles.
- Bicycle collision.
- Paresthesia in extremities.

2. Low risk factors that allow safe evaluation of ROM with NO radiography:

- Simple rear-end motor vehicle accident, EXCLUDING:
 - Pushed into oncoming traffic.
 - Hit by bus/large truck.
 - Rollover.
 - Hit by high-speed vehicle.
- Sitting position in ED.
- Ambulatory at any time after injury.
- Delayed onset of neck pain.
- Absence of midline c-spine tenderness.

3. Additional criteria to above (2) to evaluate need for radiography:

- Able to actively rotate neck 45° right and left—**NO** radiography.
- Unable to actively rotate neck 45° right and left—**YES** radiography.

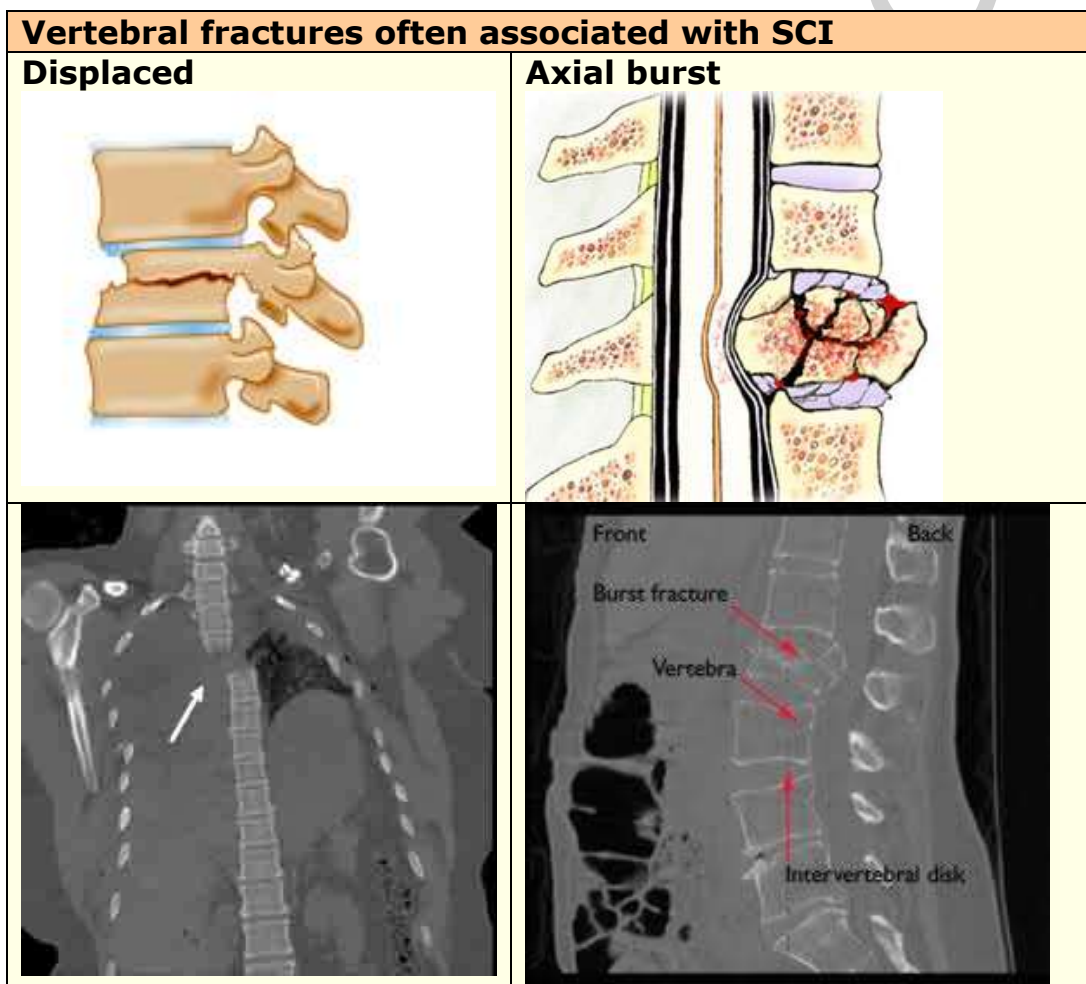
Radiologic assessment

Cervical spine: While the simple radiograph (x-ray) is probably the most commonly used radiologic assessment, it is less than 90% sensitive in detecting fractures while the CT scan is about 96% specific, so the CT scan is the radiologic assessment of choice. However, combined they are specific at about 99%, so CT is often done in conjunction with at least a lateral C-spine x-ray, but protocols may differ from one institution to another.

Fractures that extend horizontally in the axial plane, parallel to the imaging slice, may be missed by CT. The most commonly missed fractures occur at C1, C2 and C7 to T1. MRI is indicated for patients with neurological deficits or significant fractures requiring surgical reduction.

Thoracolumbar spine: The most common sites of injury are T12 to L1. AP and lateral radiographs usually provide good assessment. CT provides a closer evaluation of bone anatomy, especially if the x-rays are not clear, and MRI provides visualization of the spinal cord and nerves, helping to identify spinal cord and ligamentous injuries. All patients with neurologic deficits should have an MRI.

Lumbar spine: Spinal cord injuries from lumbar fractures are rare although injuries to the conus medullaris or cauda equina may occur. AP and lateral x-rays are usually done first but CT may be done to further evaluate burst fractures. MRI and myelography are indicated with neurological injury.



Pathophysiology of spinal cord injury

Spinal cord injuries may range from contusion, laceration, and compression to complete transection, and impairment may be temporary or permanent. SCIs may be further categorized as **primary**,

from the initial trauma, and **secondary**, from a chain of events that results in destruction of myelin and axons.

Immediately after injury, axonal transmission is interrupted and decreased spinal blood flow can result in ischemia. Initially, injury is more severe to gray matter than to white.

Within a few minutes, hemorrhages can begin to occur in the gray matter and **within 30 minutes**, central neuronal necrosis is evident and nerve fibers are edematous.

By 4 hours, the gray matter shows marked necrosis and increasing necrosis in the white matter as well. **By 8 hours**, the axons have become maximally edematous and axonal necrosis is occurring along with vesicular degeneration. **By \leq 24 hours**, permanent damage can occur.

If the cord has not suffered irreparable damage, these secondary effects may be reversed with prompt and effective treatment within the first crucial hours so that partial damage does not become permanent.

Management

Shock

Differentiating between hypovolemic and neurogenic shock is especially critical during initial management although elements of both may be present. **Neurogenic shock** can occur with both incomplete or complete blunt and penetrating spinal cord injuries and result in impairment of the autonomic nervous system controlling the cardiovascular system. Injuries above T1 may cause disruption of the entire sympathetic nervous system and those below T1 may cause varying degrees of disruption.

Neurogenic shock is typically characterized by hypotension and warm dry skin caused by lack of vascular tone (vasodilation) resulting in hypothermia from loss of cutaneous heat. This causes a relative hypovolemia because it reduces venous blood return to the heart. Bradycardia is common but is not always present.

Other indications of autonomic dysfunction can include ileus, urinary retention, and loss of anal sphincter tone.

Treatment for neurogenic shock

- Rapid administration of crystalloids to maintain mean arterial pressure at 85-90 mm Hg for at least 5 to 7 days. Overhydration must be avoided, however, because it may result in increased edema of the cord or pulmonary edema.
- Placing of pulmonary artery catheter to monitor fluid overload.
- Vasopressors (dopamine, dobutamine) if hypotension persists after administration of fluids.
- Atropine as indicated for persistent bradycardia (<45 bpm).

Orthostatic hypotension is very common in those with injuries above T7 in the first two weeks after injury, and BP remains unstable. While BP slowly returns to preinjury levels, periods of orthostatic hypotension may still occur, interfering with mobility. With quadriplegia/tetraplegia, even slight elevations of the head may cause the BP to fall precipitously. Tilt tables may help to alleviate orthostatic hypotension by gradually elevating the head.

About 70% of high cervical spinal cord injury patients will exhibit severe bradycardia and hypotension and about 16% may suffer cardiac arrest, so avoiding hypotension and hypoxia is critical to survival.

Hypovolemic shock occurs when the total circulating volume of fluid decreases, leading to a fall in venous and a decrease in stroke volume and cardiac output. This usually results in generalized arterial vasoconstriction and decreased tissue perfusion although neurogenic shock may impair this response.

Typical symptoms include anxiety, pallor, cool clammy skin, delayed capillary refill time, hypotension, increased respiratory rate, and weak, thready pulse. Identifying the cause of fluid loss is essential to reestablishing adequate intravascular fluid volume.

Treatment for hypovolemic shock

- Administration of blood, blood products, autotransfusion, colloids (such as plasma protein fraction), and/or crystalloids (such as normal saline).
- Medications may include vasopressors, such as dopamine.

Treatment for hypovolemic shock should help cord perfusion and treat neurogenic shock as well, but (as noted above) care must be taken to prevent overhydration.

Spinal shock, a concussive injury, occurs in about 50% of those with acute spinal cord injury and must be differentiated from neurogenic shock. Spinal shock is a temporary neurologic syndrome characterized by decreased reflexes, sensory impairment, and flaccid paralysis below the level of the injury. Spinal shock can last for a few days or even months and may mask residual neurologic function. Reflexes below the level of injury are depressed or absent and those above the level of injury are usually unaffected.

Spinal shock occurs in 4 phases:

1. Loss/weakening of reflexes. Persists for about 1 day.
2. Return of some reflexes. The bulbocavernosus reflex usually returns first.
3. Hyperreflexia.
4. Resolution.

Respiratory complications

Any cervical injury can result in respiratory compromise. Total loss of respiratory muscle function occurs with injuries above C4. Levels C3 to C5 (phrenic nerve) innervate the diaphragm, so any lesion above this level requires immediate ventilation. Even with injuries below this level, in which diaphragmatic breathing can occur, hypoventilation is common because of paralysis of intercostal muscles, which decreases vital capacity and tidal volume, especially if hemorrhage or edema affects the function of the phrenic nerve, causing diaphragmatic paralysis. Oxygen is administered to maintain a high partial pressure of oxygen (PaO₂). Paralysis of the abdominal muscles decreases the ability to force expirations, cough, or clear secretions.

Usually the vital capacity of patients with cervical cord injuries is \leq 30% in the initial period after injury although this may improve as muscles become more spastic, preventing collapse of the chest wall.

Those with complete injuries at C5 and above are usually intubated early and a tracheostomy performed to facilitate mechanical ventilation while lower cervical injuries may be initially ventilated with orotracheal intubation. Patients with injuries at C4 or higher will require mechanical ventilation at discharge (MVDC) as will about half of patients with C5 injuries and some patients with C6 injuries.

Patients with intubation and ventilation must be monitored carefully as artificial airways provide access for microorganisms, so chest

physiotherapy and bronchial hygiene are important. Patients must be carefully monitored for pulmonary edema resulting from increased sympathetic nervous system activity (which shunts blood to the lungs) and fluid overload.

Immobilization may lead to deep vein thrombosis (see below), which can result in pulmonary embolism (PE). The risk of death from PE is more than 200 times greater with spinal cord injury patients than others. Symptoms of PE include dyspnea, tachypnea, tachycardia, anxiety, chest pain, fever, rales, cough, and hemodynamic instability.

Diagnosis includes ABG analysis with hypoxemia, hypocarbia, and respiratory alkalosis common findings. ECG may show abnormalities. Spiral CT and pulmonary angiograms may confirm diagnosis. Echocardiogram can show emboli in central arteries and show cardiac hemodynamic status.

PE treatment

- Anticoagulation therapy can include unfractionated or low-molecular weight heparin (LMWH).
- Oxygen.
- Vasopressors, such as dobutamine or dopamine.
- Diuretics, antiarrhythmics as indicated.
- Analgesia if necessary (morphine).
- Thrombolytic therapy, such as alteplase, may be considered with massive PE and hemodynamic instability.
- Vena cava filter may be considered if anticoagulation is contraindicated or ineffective.

High dose steroids

There remains some controversy about the use of high dose steroids after spinal cord injury. While studies show that patients with incomplete or suspected incomplete blunt injuries to the spinal cord exhibit improved neurological function if high dose steroids, typically methylprednisolone, are administered within 8 hours of injury, other studies show that length of hospitalization is increased and costs significantly higher.

Steroids should not be administered with penetrating injuries because of the risk of infection. Protocols may vary, but high dose steroids is usually considered an option, taking potential side effects into consideration, and is part of the protocol in some trauma centers.

Treatment with methylprednisolone

- Initial dose: 30 mg/kg IV over 45 minutes (within 8 hours of injury).
- Follow-up dose: 5.4 mg/kg/hr. IV over 23 hours (continuous drip) if administered within 3 hours of injury and over 48 hours if administered after 3 hours of injury.
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Traction/Surgical reduction

With suspected vertebral or spinal cord injury, the spine should be immediately immobilized. For those

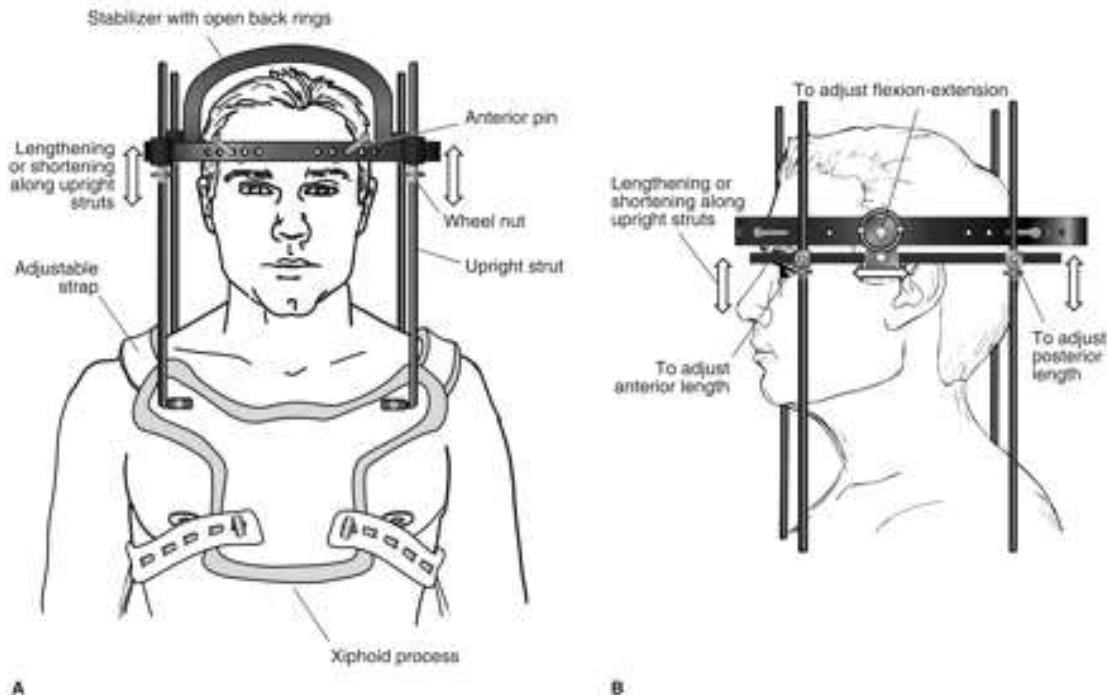
with cervical injuries and spinal misalignment, such as from displaced fractures, cervical tongs and traction should be applied immediately even if there is no evidence of neurological deficit to prevent further injury.

Gardner Wells tongs



Cervical fractures are usually reduced and the spine aligned with some form of skeletal traction or halo device. A range of tongs is available. The Gardner Wells tongs (see illustration above) do not require predrilled holes in the skull, but the Crutchfield and Vinke tongs are inserted after predrilled holes are created under local anesthetic.

Halo device



With cervical traction, the patient's neck must be maintained in neutral position and the weights must hang freely. The amount of traction depends on the patient's size and the amount of displacement.

Injuries to the thoracic and lumbar areas are usually treated with surgical repair followed by immobilization with fitted brace without traction because these areas are less unstable than the cervical area.

Deep vein thrombosis

Immobility increases the risk of deep vein thrombosis (DVT), which in turn increases the risk of pulmonary embolus (PE). The risk of DVT ranges from 39% to 100% and for pulmonary embolus 4% to 10%.

The patient must be routinely assessed for signs of DVT by daily measurements of thigh and calf as increased circumference may indicate DVT. The usual symptoms of pain and Homan's sign (calf pain with dorsiflexion of foot and ankle with knee extended) are missing after spinal cord injury. An early indication of DVT may be a low-grade fever. Highest risk occurs in the first 14 days, peaking at days 7 and 10.

Diagnostic procedures include the D-dimer test with a negative finding ruling out DVT but a positive finding inconclusive. Doppler

ultrasonography is the imaging method of choice to diagnose DVT with specificity at 98 to 100%. Because of the high incidence of DVT in spinal cord injury, routine prophylaxis should be provided.

DVT prophylaxis	
Mechanical interventions.	<ul style="list-style-type: none"> • External pneumatic compression devices. • ROM (adjunctive, not effective alone). •
Anticoagulation	<p>Note: Anticoagulation therapy is recommended for 8 weeks after injury for those with uncomplicated complete SCI and for 12 weeks for completed injuries with risk factors. Anticoagulation therapy can include:</p> <ul style="list-style-type: none"> • Unfractionated heparin. • Low-molecular weight heparin (LMWH). • Warfarin (for long-term therapy).
Surgical intervention	<ul style="list-style-type: none"> • A vena cava filter may be placed to prevent PE if anticoagulation is contraindicated or if anticoagulation is not successful. • Thromboembolectomy may be indicated in some circumstances to ensure venous patency.

Urinary retention

With spinal shock, the bladder is atonic and can become overdistended but as this subsides, the bladder may become hyperirritable with reflex emptying. During these phases, an indwelling catheter is placed for continuous drainage; however, this increases the risk of infection. Once the patient's condition is stabilized and IV fluids are no longer necessary, intermittent catheterization should begin.

Thermoregulation

Thermoregulation is impaired with spinal cord injury, resulting in poikilothermism (adjustment of the body temperature to the environmental temperature) because the peripheral temperature sensations cannot reach the hypothalamus, which controls temperature.

Additionally, the ability to sweat or shiver to control temperature is impaired below the level of injury. The higher the injury, the greater the problem with thermoregulation.

Temperature must be monitored carefully and the environment adjusted as needed. Subnormal temperatures and hypothermia (<35° C/95° F) are common. Warming blankets may be necessary to maintain adequate temperature but heavy covers should be avoided. If fever occurs, cooling blankets may be necessary. Overexposing the body, such as during bathing, should be avoided

Pressure sores

Immobility increases the risk of skin breakdown and pressure sores over bony prominences.

Patients should be turned at least every two hours, but should not lie directly on the hipbone but should be positioned at the 30-degree position on a pressure relieving surfaces.

Proper body alignment and support should be provided. The skin should be checked at each time the patient is turned for any indications of skin irritation or breakdown. Those able to sit must do weight shifts at least every 15 minutes.

Orthotic devices may be used to raise the head off the bed for those who must stay in the supine position. Minimal air loss beds help reduce pressure. Donut-shape devices should be avoided. Patients on Stryker frames should be rotated on a regular scheduled basis.

Gastrointestinal problems

Injury above T5 results in hypomotility, which increases the risk of ileus and gastric distention. This is

often treated with an NG tube and intermittent suctioning, but this increases the risk of gastric irritation.

Common signs of bleeding are often lacking, so monitoring blood pressure and blood counts can help to identify bleeding. Stress ulcers may develop because of increased release of hydrochloric acid in the stomach. Peak occurrence of stress ulcers is 6 to 14 days after injury.

Treatment for GI problems

Stress ulcers	Histamine H2-receptor blockers (Ranitidine, famotidine). Proton pump inhibitors (omeprazole, lansoprazole)
Delayed gastric emptying	Metoclopramide (Reglan®).

When bowel sounds are good, the NG tube may be removed but those with cervical injuries must be evaluated for swallowing prior to

beginning feedings. If the patient is unable to take oral feedings, enteral feedings or total parenteral nutrition (TPN) may be indicated to provide nutritional support.

Because of less voluntary control over the bowel, a neurogenic bowel occurs. Initially, with spinal shock and with injuries at levels T12 or lower, the bowel is areflexic with decreased sphincter tone, but as reflexes return to normal, sphincter tone is increased and reflex defecation occurs. A regular bowel-training program should be initiated to control bowel movements and prevent incontinence.

Metabolic abnormalities

Electrolyte levels must be monitored closely as NG suctioning can lead to metabolic alkalosis, especially affecting sodium and potassium levels. Decreased perfusion of tissues may cause acidosis.

Nutrition is especially important, as loss of body weight is common, usually about 10% or more with increased loss of nitrogen. After a severe spinal cord injury, hypermetabolic and protein catabolic states occur with prolonged loss of nitrogen and muscle mass, so malnutrition can occur within 2 to 3 weeks. Nutritional needs are greater than normal and a diet high in protein with positive nitrogen balance is necessary to prevent skin breakdown and muscle atrophy.

Autonomic dysreflexia

Autonomic dysreflexia occurs with central cord lesions above T7 when a painful stimulus occurs below the spinal cord injury. Autonomic dysreflexia is common after resolution of spinal shock and the return of reflexes. Normally, the autonomic nervous system maintains homeostasis through a negative feedback between the sympathetic and parasympathetic nervous systems so that one is active while the other is not:

- **Sympathetic:** Dilates pupils, increases heart rate, and constricts vessels. The sympathetic nerves leave between T5 and L2 with major output at the splanchnic outflow (T5-T6).
- **Parasympathetic:** Constricts pupils, decreases heart rate, and dilates vessels. The parasympathetic nerves leave the CNS at the midbrain and base of the brain (cranial nerves) and the lumbar area.

Because of the spinal cord injury, the two systems work independently, so if a painful stimulus ascends to the splanchnic outflow, a sympathetic response occurs below the level of injury with vasoconstriction, hypertension and severe headache, but the parasympathetic system cannot respond to counteract this through the feedback loop, so the brainstem stimulates the vagus nerve to slow the heart and dilate vessels above the injury; however, the parasympathetic response cannot travel below the injury. This is a life-threatening condition that must be resolved immediately.

Autonomic dysreflexia may be triggered by any sensory stimulation although distended bladder or rectum is the most common precipitating factor. Other triggers include ingrown toenail, pressure sores, sunburn, restrictive clothing, and sexual intercourse.

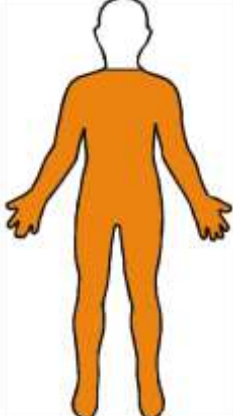
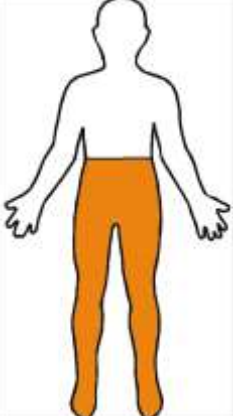
Symptoms include:

- Increase in BP by 20-40 mm Hg systolic (often up to 300 mm Hg).
- Blurred vision,
- Pounding headache.
- Piloerection.
- Below lesion: Vasoconstriction, pallor.
- Above lesion: Marked diaphoresis, flushing.
- Bradycardia.
- Nasal congestion.
- Anxiety, restlessness.

Treatment for autonomic dysreflexia

- Immediately elevate the head of the bed to 45 degrees or sit patient upright.
- Investigate and identify cause and alleviate.
- Immediately catheterize to relieve distended bladder or if catheter is in place, check for kinks or blockage.
- Apply topical anesthetic ointment to rectal area prior to digital exam to check for fecal impaction.
- Loosen clothes, remove shoes, and relieve all skin stimuli.
- Monitor BP frequently.
- If symptoms persist after stimulus is relieved, administer an α -adrenergic blocker or vasodilator (such as nifedipine).

Levels of injury and functional abilities

Quadriplegia/Tetraplegia	Paraplegia
 <p>Paralysis involving all 4 extremities.</p>	 <p>Paralysis involving the lower extremities.</p>

Quadriplegia/Tetraplegia

Patients with complete injury to the cervical spine (C1 to C8) will have paralysis of all 4 limbs. Depending on the level of injury, patients may have some sensation and motor activity in the shoulders and upper arms

Injury Level	Functional abilities
C1	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Little or no control of head or neck and requires continuous ventilation. Injuries are often fatal. May be able to use voice or sip-n-puff controlled devices, such as electric wheelchairs and computers. Dependent for all personal care.
C2/C3	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Head and neck sensation and some control of neck. Requires mechanical ventilation but may be independent of ventilation for short periods. Can use voice or sip-n-puff controlled devices, but remains dependent for all personal care.
C4	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Good sensation and

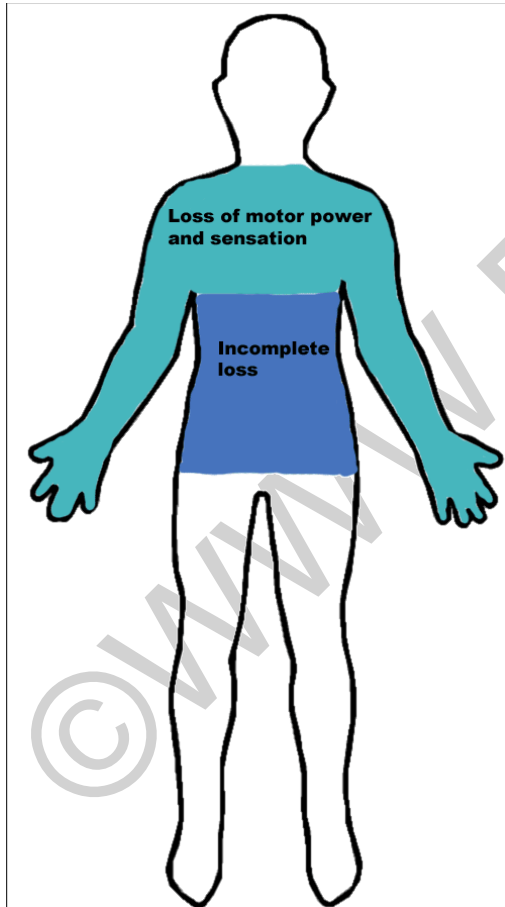
	movement of head and neck and some shoulder elevation. Diaphragm movement allows for independent ventilation. May be able to eat and manage some activities with adaptive sling but needs mouth, head, or shoulder controlled electric chair and dependent for most personal care.
C5	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Good strength and movement of head, neck, and shoulders and has elbow flexion, which allows some independence in eating and dressing. Can use electric or modified manual wheelchair for mobility.
C6	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Full innervation of shoulder and has wrist extension or dorsiflexion, so can be independent in eating, dressing, and elimination with minimal assistance. Can use and transfer to manual or electric wheelchair.
C7, C8	Vagus nerve domination of heart, respiration, circulation, and all organs below level of injury. Full extension of elbow and wrist plantar flexion with some control of fingers, allowing independence in eating, dressing, and elimination. Can use and transfer to manual wheelchair.

Paraplegia

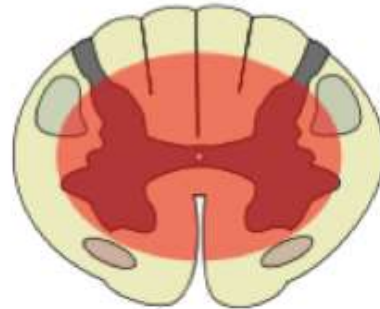
Injury to the thoracic or lumbar spine results in varying levels of paralysis of the lower extremities with weak to good trunk stability, depending on the level of injury.

Injury Level	Functional abilities
T1 to T5	Sympathetic innervation to heart, vagus nerve domination of all vessels and organs below injury. Full use of hands and fingers and use of intercostal and thoracic muscles. Independent in personal care, transfers, wheelchair use.
T6 to T10	Vagus nerve domination of leg vessels. As for T1-T5 and control of abdominal muscles with moderate to good balance with trunk muscles and independence in personal care, transfers, wheelchair use.
T11 to L5	Vagus nerve domination of leg vessels at L1-L2 and partial vagus nerve domination of leg vessels, GI and GU organs.

	<p>Range from able to ambulate short distances with assistance to full ambulation, often with crutches or long leg braces, depending on level.</p> <p>L1-L3, Control of hip flexors, hip abductors, good sitting balance.</p> <p>L2-L4, Knee extension.</p> <p>L3-L4 Independent ambulation with short leg braces and canes.</p> <p>L4-L5, Knee flexion and ankle dorsiflexion.</p>
S1 to S5	<p>Full control of leg, foot, and ankle. Able to ambulate independently with or without assistance and has normal to impaired function of bowel and bladder.</p> <p>S2-S4, Innervation of perineal muscles with bowel, bladder control and sexual function.</p>



Central Cord Syndrome

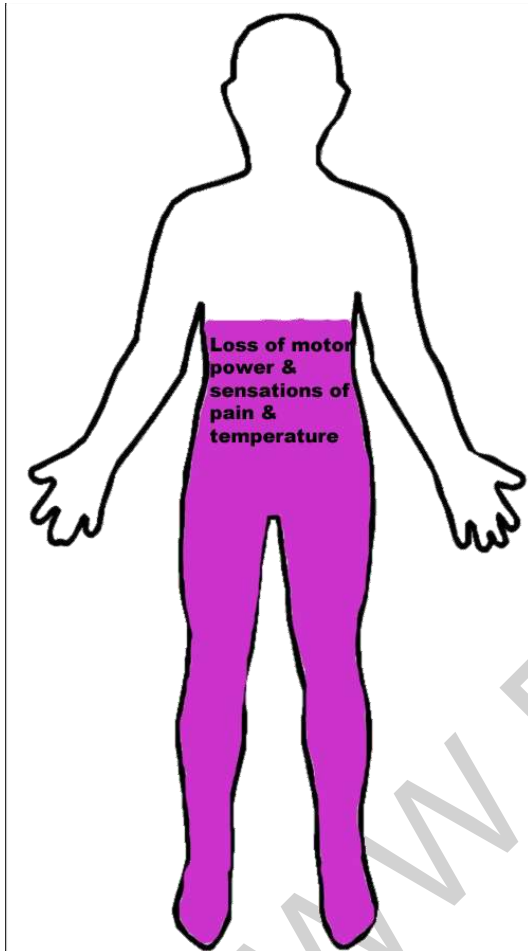


Central cord syndrome is the most frequent type of incomplete lesion, primarily of the cervical spine, most often occurring in elderly patients with pre-existing cervical spondylosis.

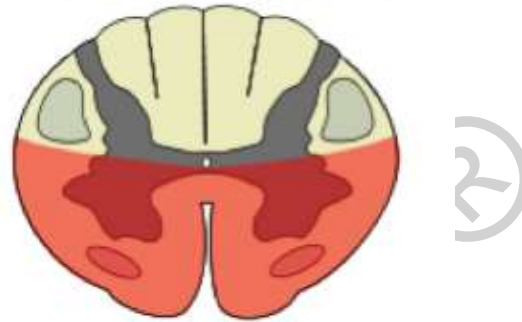
In younger patients, CCS may occur with high force trauma. CCS results from hyperextension that compresses the spinal cord anteriorly, resulting in bleeding and/or edema and injuring the central gray matter.

Loss of motor functions in the upper extremities is more pronounced than in the lower extremities, although there may be some gait impairment, with various sparing of sensations below the level of injury and varying degrees of bowel/bladder impairment and retention.

Symptoms may improve with conservative treatment, including immobilization of the cervical spine, and therapy to improve muscle function



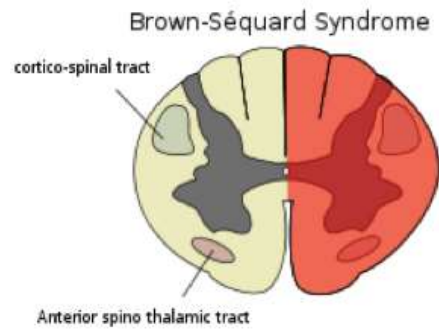
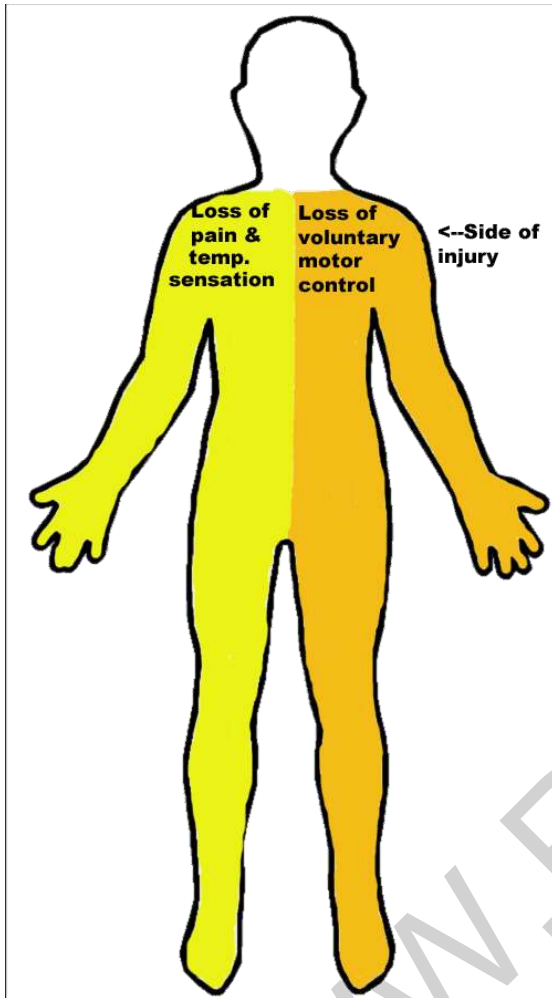
Anterior Cord Syndrome



Anterior cord syndrome is the most severe of the cord syndromes, with the worst prognosis for recovery, with improvement rare (10-15%) if there is no evidence of progressive reduction of symptoms within 24 hours.

ACS is characterized by loss of pain, temperature, and motor function below the level of injury but with retention of light touch, proprioception, and vibration sensation.

ACS may occur as the result of acute disc herniation or hyperflexion injuries associated with fracture dislocation of vertebra. In many cases, the ACS may result from compression of and injury to the anterior spinal artery, which nourishes the anterior two-thirds of the spinal cord. ACS is the second most common cervical cord injury.



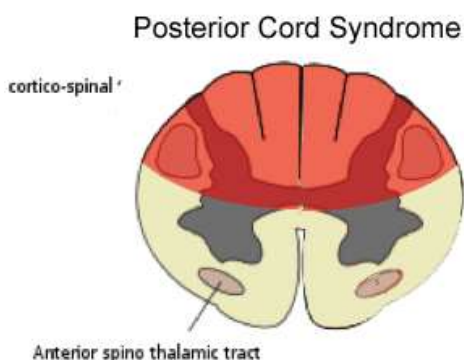
Brown-Séquard syndrome (or lateral cord syndrome) is rare but has a better prognosis than the other syndromes.

The lesion on one side of the spine is caused by transverse hemisection of the spinal cord (most often from a stabbing or missile injury), fracture-dislocation of a unilateral articular process, or acute ruptured disc. BSS is most common in the cervical region.

Stabbing injuries, the most common, may also result in severe hemorrhage.

While symptoms may vary, BSS is characterized by ipsilateral paralysis or paresis with ipsilateral loss of touch, pressure, and vibration sensation below the level of the lesion and contralateral loss of pain and temperature 2 to 3 levels below the level of the lesion. The Babinski sign is usually found ipsilateral to the lesion although abnormal reflexes and the Babinski sign may not be evident with acute injury.

Posterior cord syndrome



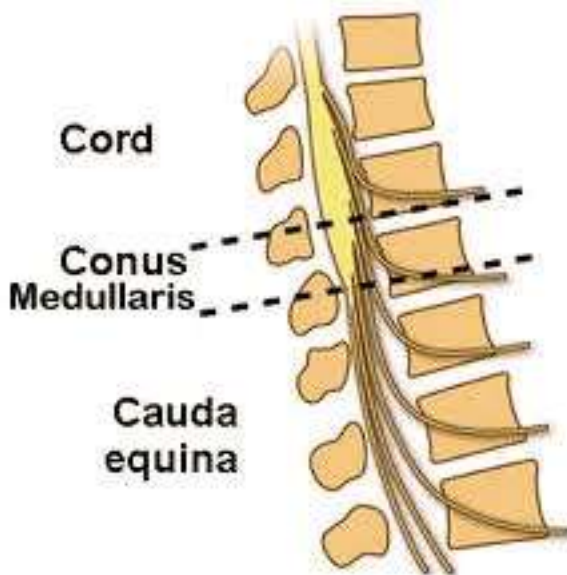
Posterior cord syndrome is very rare and usually results from cervical hyperextension injuries that damage the dorsal areas of the cord.

PCS is characterized by loss of deep pressure, deep pain, and proprioception below the level of the lesion but with normal motor function and other pain and temperature sensations intact. Prognosis is good.

Conus medullaris syndrome

CMS affects the upper motor neurons and may result in hyperreflexia and/or reflex stunning with loss of the bulbocavernosus reflex. Onset of symptoms is often sudden and bilateral with numbness localized to perineal area.

Patients typically develop symmetric hyperreflexic distal paresis of lower extremities. Impotence is frequent as well as sphincter dysfunction (an early sign) that results in urinary retention and overflow incontinence as well as fecal incontinence.



Cauda equina syndrome

CES usually results from penetrating or compressive injury below the level of the spinal cord. CES syndrome affects the nerve roots, so it is classified as a peripheral nerve disorder, but the effects may be permanent.

Because numerous nerve fibers can be affected, patients may exhibit a range of symptoms, including bowel and bladder dysfunction, saddle paresthesias, sciatica (unilateral or bilateral) and varying degrees of

lower extremity motor weakness and sensory loss. Patients may exhibit asymmetric areflexic paraplegia.

Onset of symptoms is often gradual and unilateral with urinary retention a late indication. Patients may develop erectile dysfunction and lack of sensation in pubic/genital area (glans penis/clitoris). Operative intervention to relieve compression may allow for recovery although there remains some controversy about the timing of decompressive surgery.

Conclusion

Adequate treatment in the initial period after trauma is critical to the long-term outcome for patients. Many of the problems identified in the initial assessment and management of the patient will remain chronic issues, so patient education and training are essential. Once the patient has stabilized, the long rehabilitation process begins with the goal preventing complications while allowing the patient as much independence as possible.

Patients often experience profound grief at the loss of body function and disturbed body image and may experience the same stages as those faced with death: denial, anger, bargaining, depression, and acceptance. While early care is focused primarily on physical needs, recognizing the patients' emotional needs and providing support is equally important.

Ventilator-dependent patients with intact phrenic nerves may receive phrenic nerve stimulators or diaphragmatic pacemakers. With rehabilitation, many patients can learn to live independently or with minimal assistance.

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