

**Northwest Community EMS System
EMP Training Program
SPINAL CORD TRAUMA
Connie J. Mattera, M.S., R.N., EMT-P**

Reading assignment: Caroline
SOP: Spine trauma
Lab manual: All spine motion restriction procedures

Knowledge objectives

Upon completion of the class and study questions, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Describe the incidence, morbidity, and mortality of spinal injuries in the trauma patient.
2. Discuss the anatomy and physiology of structures related to spinal injuries including those in the following areas: cervical, thoracic, lumbar, sacrum, coccyx, head, brain, spinal cord, nerve tract(s), and dermatomes.
3. Predict spinal injuries based on mechanism of injury.
4. Explain the pathophysiology of spinal injuries.
5. Describe the assessment findings associated with spinal injuries.
6. Sequence the management of spinal injuries.
7. Identify the need for rapid intervention and transport of the patient with spinal injuries.
8. Integrate principles of pathophysiology to the assessment of a patient with a spinal injury.
9. Formulate a field impression based on the assessment findings.
10. Develop a patient management plan based on the field impression.
11. Describe the pathophysiology of traumatic spinal injury related to the following:
 - a. Spinal shock
 - b. Spinal neurogenic shock
 - c. Tetraplegia/paraplegia
 - d. Incomplete cord injury/ cord syndromes:
 1. Central cord syndrome
 2. Anterior cord syndrome
 3. Brown-Sequard syndrome
12. differentiate between traumatic and non-traumatic spinal injuries based on the assessment and history.

Spinal Cord Trauma

I. Introduction

- A. A spinal cord injury is a transient or permanent injury of varied etiology to the vertebral column, its neural or ligament structures resulting in either complete or incomplete pathologic changes.
- B. This presentation reviews spinal anatomy and physiology, injury demographics and epidemiology, and the essential elements of assessment and initial management of spinal cord injury (SCI). The EMT's responsibility to independently assess and stabilize these patients is limited. However, the ultimate outcome may be critically dependent upon proper care. Careful attention to a meticulous baseline assessment and appropriate resuscitation techniques is necessary to prevent further harm from occurring that could lead to deterioration, preventable morbidity, and/or mortality.

II. Epidemiology of spinal trauma

A. Incidence

- 1. There have not been any studies of the overall incidence of SCI in the U.S. since the 1970s so it is unclear if the numbers have changed in recent years. Data from states that do have SCI registries suggest the rate is about 40 cases/million population, excluding those who die at the scene. Given the overall US population, there are approximately 11,000-12,000 new SCIs per year (UAB, 2008).
- 2. It is estimated that an additional 3000, or 20 persons/million, suffer SCI but expire prior to hospital admission.
- 3. Spinal cord injury occurs in over 2.6% of all severe multiple trauma victims and in 7.5% to 8.9% of serious head-injured patients (GCS < 10).

B. Prevalence - There are approximately 255,702 persons living with SCI.

C. Demographics

- 1. **Age range:** Trend since 2000 is towards an older mean age at the time of injury (39.5 years) (UAB, 2008). Percentage of persons older than 60 yrs at injury has increased from 4.7% prior to 1980 to 11.5% since 2000 (Ntl SCI database, 2008).
- 2. **Gender:** Since 2000, 77.8% are male. 4:1 ratio mostly due to etiology of injury (UAB, 2008).
- 3. **Ethnicity:** Although the majority of SCI patients are white, the incidence is trending higher among non-whites.
- 4. **Time of greatest injuries:** Holidays and weekends between 12:00 and 5:00 AM after leaving bars and parties. Almost 65% are associated with alcohol and/or drug abuse.

D. Etiology of spinal cord dysfunction/damage

The National Spinal Cord Injury Database has been in existence since 1973 and captures data from about 13% of new SCI cases in the US. Since its inception, 25 federally funded model SCI care systems have contributed data. As of June 2000, the database contained information on more than 24,332 persons with traumatic spinal cord injuries. The database recognizes 38 separate causes of injury which are classified into five major categories:

- 1. Motor vehicle crashes (42%) since 2005
- 2. Falls - 27.1% and increasing
- 3. Acts of violence (primarily GSW) 15.3% since 2005 and declining since 2000.
- 4. Recreational/sporting activities such as diving into less than 3 ft. of water (66% of sports-related accidents) 7.4% and decreasing
- 5. Others (Diseases such as polio, spina bifida, Friedreich's Ataxia): (6.9%) (UAB, 2008)

- E. **Morbidity and mortality:** Depends on the age at injury, time since they were injured, and injury severity. Mortality ranges from 2 - 16 times greater for SCI than for other injuries, usually within the first 24 hours. Rate increases with age. Life expectancy has improved significantly from median length of post-injury survival in the 70's of 17-25 years to as high as 30-40 years (UAB, 2008). Mortality during first few months after SCI is perhaps as high as 20%. However, those who survive have relatively low excess mortality when compared with non-spinal cord injured population.

Causes of death: In the past, the leading cause of death in SCI patients was renal failure. Today, it is pneumonia, pulmonary emboli, and septicemia (UAB, 2008).

III. Anatomy of vertebral column and spinal cord

- A. The spine consists of a supporting skeletal structure (vertebral column) and a central nervous system pathway (spinal cord). Each segment of the vertebral column represents a complex arrangement of joints, pivots, restraints, and activators held together by ligaments that provide support, protection of neural elements and fluidity of movement to the body. The vertebral column is rigid enough to protect neural structures yet flexible to allow fluidity of movement in six degrees of freedom: flexion and extension; right and left side bending; right and left rotation.

B. Vertebral column

1. The **vertebral column** is a hollow skeletal tube composed of 31-33 irregular bones that attach to the head, ribs and pelvis. It provides a major portion of the axial skeleton. The spinal column is generally 4 to 5 inches shorter in females than in males and normally withstands injury up to 1,000 ft/lbs. of force.

2. **Vertebral structures:** Although each is slightly different, cervical, thoracic and lumbar vertebrae have a similar structure:

- a. **Body:** Anterior weight-bearing structure made of cancellous bone surrounded by a layer of hard, compact bone that provides support and stability. Size varies by location, increasing in height with each successive segment.

- b. **Lamina:** The posterior plates fuse to form spinous processes that make up the foramen of the spinal canal.

- c. **Pedicles:** Thick, lateral bony struts connect the vertebral body with spinous and transverse processes and also help make up the spinal foramen.

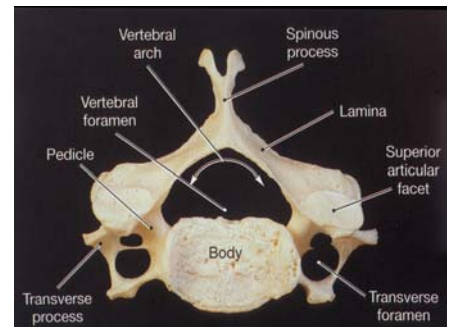
- d. The inferior border of each pedicle contains an **intervertebral foramen**, (notch) that allows the exit of a peripheral nerve root and spinal vein and the entrance of a spinal artery on each side and at each vertebral junction.

- e. **Transverse spinous process:** Junction of each pedicle and lamina on each side of a vertebra. These project laterally and posteriorly and form points of attachments for various muscles and ligaments.

- f. **Posterior spinous process:** Posterior spiny arch or tail formed by the fused lamina that also becomes an attachment site for muscles and ligaments.

- g. Each vertebra articulates with another through superior and inferior **facet joints**, that are the pivots of the spinal column. Their anatomic configuration determines the pattern of motion at any given level of the spine.

- (1) Cervical facets are oriented 45° to the end plate that allows rotation coupled with lateral bending.



- (2) Lumbar facets are oriented perpendicular to the end plate to allow bending in the sagittal plane.

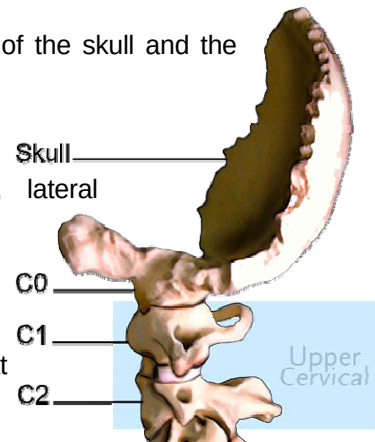
h. **Vertebral foramen/spinal canal**

- (1) Opening extending through the entire spinal column in which the spinal cord sits. It conforms to the various spinal curvatures and changes in shape and size at the different levels:
- (2) At C1 and C2 there is relatively more space between the cord and the interior of the vertebral column. At C3 the canal is rather large and triangular, allowing about 1 cm of clearance (95% occupied).
- (3) The thoracic region is small and circular with the least amount of clearance (5-6 mm). Stress on the cord here is likely to result in irreversible injury.
- (4) The lumbar region is triangular again with 1 cm clearance (65% occupied) as the cord narrows to a point below L1 before giving rise to the cauda equina.

C. **Anatomical divisions of vertebral column**

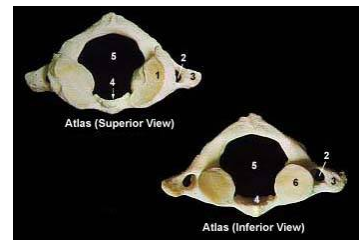
1. **Cervical vertebrae:** 7 bones between the base of the skull and the shoulders; 8 nerve roots

- a. The relatively heavy head (16 to 22 lbs.) sits on the fragile c-spines that are designed to allow flexion, extension, lateral bending, and turning of the head.
- b. Smaller than the other vertebrae, they allow greater movement. Yet the pathways in the cervical cord are critical to life functions. Rapid deceleration that propels the head forward and backward can result in devastating SCI.

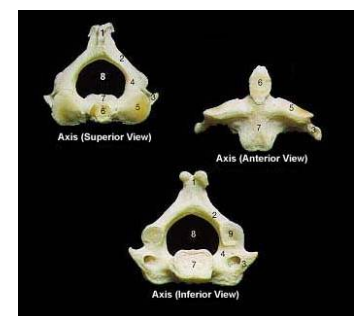


c. **Unique bones in the upper cervical area**

- (1) **Atlas:** Like the Greek God, Atlas, who held the world on his hand, the spine atlas (C1) consists of two lateral masses and two arches (no body or spinous processes) that support the head through secure interfaces with the occipital bone and C2. This is considered the "joint above" when splinting the spine. No disc between skull and C1.



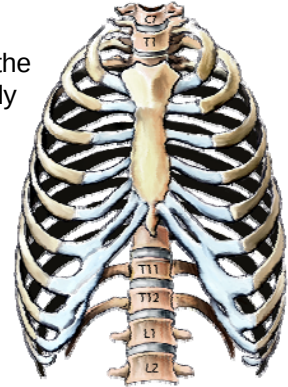
- (2) **Axis:** C2, also called the epistropheus, is most striking because of a small bony "tooth", the odontoid process or **dens**, which rises perpendicularly from the midportion of the upper body surface. The dens provides a pivot point that permits head rotation to the left and right and the ability to nod the head. No disc between C1 & C2.



- d. The lower four cervical vertebrae have rather smooth bodies that are broader from side to side than front to back. C7 is distinctive due to its prominent posterior spinous process that can be felt at the base of the neck, just above the shoulders.
- e. Just anterior to the articular facets are the transverse processes through which runs an oval foramen that is a passageway for the vertebral artery, vein and sympathetic nerves.

2. **Thoracic vertebrae**

- a. Rib cage and spinous processes limit motion of the thoracic spine. The motion allowed is mostly rotation.
- b. 12 bones, 12 nerve roots
- c. Junctional vertebrae - adjacent regions are more prone to injury
 - (1) C6 to T2
 - (2) T11 to L2
- d. Floating ribs at T11 and T12
- e. Body - progressive increase in mass from T1 to T12
- f. Pedicles - small diameter
- g. Laminae - vertical, with "roof tile" arrangement
- h. Spinous processes - long, overlapping, projected downward; larger because they are associated with the muscles holding the upper body erect and with the movement of the thoracic cage during ventilation
- i. Intervertebral foramen - larger, less incidence of nerve compression



3. **Lumbar vertebrae**

- a. 5 bones, 5 nerve roots
- b. Body - L1 to L5 progressive increase in mass as they carry the majority of the upper body's weight
- c. Pedicles - longer and wider than thoracic; oval shaped
- d. Spinous processes - horizontal, square shaped
- e. Transverse processes - smaller than in thoracic region
- f. Intervertebral foramen - large, but with increased incidence of nerve root compression
- g. Spinal foramen - large to allow for cauda equina and nerve roots
- h. L5 articulates with the sacrum, which attaches to the pelvis
- i. The main motion of the lumbar spines is bending forward and extending backwards with some bending to the side



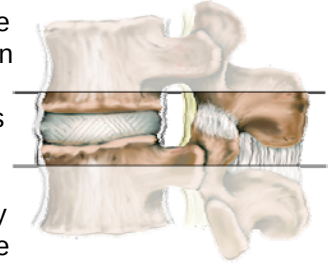
4. **Sacral vertebrae**

- a. 5 fused vertebrae that form the posterior plate of the pelvis; 5 nerve roots
- b. The articulation with the pelvis occurs at the sacroiliac joints on the right and left lateral surfaces of the sacrum and permits no movement. This forms the "joint below" for purposes of splinting the spine.

5. **Coccyx:** 3-5 fused vertebrae that are vestiges of a tail. They either have none or only one nerve root. Injury is usually clinically insignificant.

6. The vertebral column **curves** naturally after infancy in order to allow an upright posture and movement. The cervical and lumbar areas have concave curves while the thoracic and sacral regions have convex curves. These curves strengthen the spine and permit a greater range of motion.

- D. **Intervertebral discs:** Most vertebrae are separated and cushioned by intervertebral discs composed of cartilage and pulposi that limit bone wear, act as shock absorbers, and serve as the fibrocartilaginous joint of the motion segment. They are the largest avascular structures in the body. Discs are present at levels C2-C3 to L5-S1 and make up about 25% of the total length of the spinal column. As we age, they degenerate, lose water content, and thin out, accounting for height loss as we get older. Most axial loads are transmitted through the discs, especially with flexion. They are designed to allow compressive and rotational motion. The axis of spine rotation is usually on the posterior 1/3 of the disc.

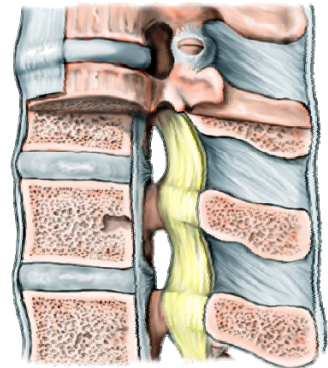


Herniated discs Stress on the vertebral column may cause the gelatinous center of the disc to bulge or extend into the spinal canal causing central cord or nerve root signs. Bending beyond 15° or torsion beyond 20° are the primary causes of disc failure. This is often due to improper lifting and occurs most commonly at L-4, 5. Degenerative disease increases the risk.

- E. **Spinal ligaments:** The vertebrae are connected by 40 muscles and bands or sheets of tough, fibrous, **ligaments** that connect bones, cartilage, or other structures and tether the base of the skull to the pelvis and hold the column in alignment so the cord is not stretched or pinched. They become active when stressed to maximum range of motion to protect the joints from being hyperflexed. If the vertebral column sustains normal to 60% flexion or up to 70% extension, there is no stretching of the cord. Flexion or extension beyond those limits predisposes the ligaments and cord to injury.

Significant ligaments

1. **Anterior longitudinal:** Ribbon that runs anterior to vertebral body. It is responsible for resistance to hyperextension.
2. **Posterior longitudinal:** Runs along the posterior body within the vertebral canal. It prevents hyperflexion.
3. Others: Capsular, supraspinous, interspinous, cruciform, accessory atlantoaxial, and ligamenta flavum (criss-crosses between laminae). All resist flexion and add to strength, stability and articulation.
4. **Ligament changes:** All ligaments are subject to **creep** (asymptomatic elongation of the fibers subjected to a constant force over time); **stress-relaxation** (decrease in stress in a material subjected to constant deformation over time); and **hysteresis** (energy loss resulting from cyclic loading and unloading).



F. Spinal cord and nerves

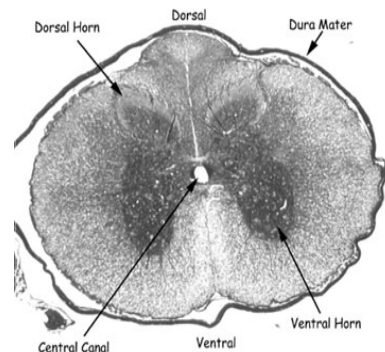
1. The spinal cord serves as a unique nerve center in its own right as well as a transmitter of motor and sensory nerve impulses between the brain and the body controlling movement, posture, respiration, heart rate, heat regulation, circulation, bowel, bladder, and sexual function. If the cord is severed (physically or physiologically), the bridge is blown out of the road and nerve transmissions are disrupted.
2. **Tissue characteristics:** Composed of central and peripheral nervous system tissue.
3. **Topography**
 - a. In a fetus, the cord fills the entire length of the vertebral column. However, as we mature, the length of the cord does not keep pace with the growth of the vertebral column, thus peripheral nerve roots are pulled into the spinal foramen. In adults, the cord is about 18" long and extends from the

brainstem (medulla) as it exits from the skull at the foramen magnum to the lower border of the 1st or 2nd lumbar vertebrae.

- b. It is firm, cord-like structure that is approximately $\frac{3}{4}$ inch in diameter at the top but narrows to a point at the bottom (*conus medullaris*) prior to giving rise to paired nerve branches called the "**cauda equina**" or horse's tail that descend inferiorly through the spinal canal and exit through the remaining lumbar and sacral vertebrae. Any injury below L2 may spare certain nerve roots and affect others.
 - c. The cord is wider in the cervical and lumbar regions. The *cervical enlargement* is located between C3 or C4 through T1. This bulge contains the nuclei for the upper extremities. The *lumbar enlargement* is located between T9 through T12 and contains the nuclei for the lower extremities.
 - d. The cord and peripheral nerve roots are encased by the same **meninges** (dura, arachnoid, and pia) as the brain. The dural sac is firmly attached to the base of the skull and continues down to S2 where an extension of the pia mater continues past the conus medullaris as the *filum terminale* and connects the spinal cord with the coccyx. Denticulate ligaments are lateral extensions of pia mater that fuse with arachnoid mater. These attachments help position the cord centrally within the spinal canal yet permit the column to move around the cord (Allen & Harper, 2003).
 - e. Cerebral spinal fluid surrounds the cord in the subarachnoid space. The central canal also contains CSF. There is a reservoir of CSF in the lumbar region called the spinal cistern. This is where CSF is most safely removed for a spinal tap.
 - f. The cord is also surrounded by a protective layer of epidural fat and blood vessels
4. **Histology:** Although the detailed anatomy of the spinal cord is complex, knowledge of its basic anatomy allows one to appreciate the extent of neurologic damage and the rationale for the various assessment components.

a. **Nerve pathways: Ascending and descending spinal cord tracts**

A cross section of the cord reveals an H- or butterfly-shaped central core of gray matter (anterior [ventral] and posterior [dorsal] horns) that is composed of neural cell bodies and synapses. Surrounding the gray matter on each side are three bundles or columns of peripheral white matter composed of myelinated ascending and descending fiber pathways (axons). Messages are relayed to and from the brain through these pathways.



The tracts are named for their source of origin to their point of termination, e.g., corticospinal, spinothalamic. When someone experiences a SCI, the pathways are damaged (functionally or anatomically) and the processes controlled by that tract are disrupted.

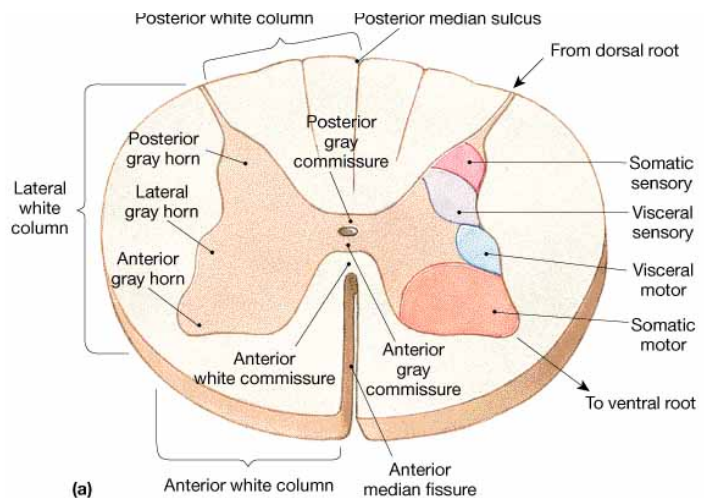
b. **The cord is divided into an anterior 2/3rds and a posterior 1/3**

(1) **Anterior portion**

- (a) **Central gray** - The posterior (dorsal) horns carry sensory input and the anterior (ventral) horns innervate the motor nerve of that segment.

(b) **Spinothalamic tracts - ascending**

- (i) Lateral and anterior tracts that originate in the spinal column (spino) and terminate in the thalamus (thalamic) in the brain.
- (ii) Lateral tracts carry pain and temperature. Each fiber crosses in the cord shortly after the arrival of the incoming sensory nerve so injury causes contralateral (opposite side) deficits.
- (iii) Anterior tracts carry crude touch and pressure perception to the brain.



- (a) **Spinocerebellar tracts** (anterior and posterior) function to coordinate impulses necessary for muscular movements by carrying impulses from muscles in the legs and trunk to the cerebellum.

(b) **Corticospinal (Pyramidal) tracts - descending**

- (i) Brings motor commands from the frontal lobe to the anterior horn cells and causes voluntary movement at that level - **corticospinal tracts**.
- (ii) **Lateral tracts** (80%) cross (*decussate*) in the medullary pyramids. So damage to the cord causes ipsilateral (same side) deficits below the lesion. The anterior tract descends uncrossed.

(c) **Extrapyramidal:** Specialized sensory functions

(2) **Posterior portion**

- (a) **Fasciculus gracilis and cuneatus:** Dorsal or posterior columns carry the conscious perception of position sense (**proprioception**), vibration, light touch, deep pressure (massage), two-point discrimination, and stereognosis (recognition of the form of objects by touch) to the brain from the skin, muscles, tendons and joints.
- (b) Fibers cross after entering the brain at the medulla. Thus, injury causes disruption on the same side of the body.

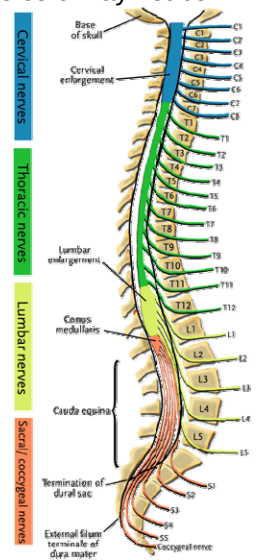
5. **Spinal nerves**

- a. The cord has 31 pairs of spinal nerves attached to it, with each pair arising from a different segment (Allen & Harper, 2003). They convey information from peripheral sensory receptors to the spinal cord and information from

- b. Spinal nerves connect to the cord via a **posterior** (dorsal root) and an **anterior** (ventral) root and are called **mixed nerves** because each contains sensory and motor axons.
- c. **Motor neurons**
 - (1) **Upper motor neurons:** Originate in the brain and terminate in the ganglia at each segmental level of the cord. They synapse with lower motor neurons arising in the spinal cord. Their function is to carry the messages back and forth from the brain to the spinal nerves. UMN injuries occur above the level of the anterior horn cell.
 - (2) **Lower motor neurons:** Originate in ganglia of the cord and terminate in an effector. These 31 pairs of nerves exit and enter at each vertebral level and communicate with specific areas of the body. Each pair has an anterior nerve root (motor) and a posterior nerve root (sensory). LMN injuries at or below the anterior horn cells.

6. **Neurological divisions:** Each part of the cord is divided into specific neurologic segments with higher levels controlling the upper parts of the body and lower segments controlling the lower parts of the body. The consequences of injury reflect this organization (NINDS, 2007). The anatomic levels of the cord may not be adjacent to the levels of the vertebral bodies that contain them. Therefore, the bony level of injury can be different from the neurologic level of impairment.

- a. **Cervical** (8 nerve roots): Each level contributes to different functions in the scalp, neck, shoulders, and arms.
- b. **Thoracic** (12 nerve roots): Upper thoracic nerves supply muscles of the chest that help in breathing and coughing. Lower thoracic nerves provide abdominal muscle control; also contains nerves of the sympathetic nervous system.
- c. **Lumbar** (5 nerve roots): Hip flexors and legs; sensation anterior legs
- d. **Sacral** (5 nerve roots): Bowel and bladder control, sexual function; sensation in posterior legs and rectum
- e. **Coccyx:** 1 nerve root
- f. Nerve roots often converge in a cluster of nerves called a **plexus** (or braiding) that permits peripheral nerve roots to rejoin and function as a group.



- (1) **Cervical plexus:** First 5 cervical nerve roots. The cervical plexus forms the Phrenic nerve (C3-5) that moves the diaphragm.
- (2) **Brachial plexus** joins the nerves controlling the upper extremities (C5-T1). Main nerves arising from this plexus are the axillary, median, musculocutaneous, radial and ulnar.
- (3) **Lumbar plexus:** L1-L4. Supplies the skin and muscles of the abdominal wall, external genitalia and part of the lower limbs. Major nerves are the femoral and obturator nerves.

- (4) **Sacral plexus:** L4-S4. Supplies the buttocks, perineum, and most of the lower limbs. Major nerves are the pudendal and sciatic. The sciatic is composed of the tibial and common fibular (common peroneal) nerves (Allen & Harper, 2003).

G. **Blood supply to the cord**

1. The spinal cord needs a constant source of oxygenated blood. Fed by:
 - a. One anterior spinal artery provides perfusion to the anterior 2/3 of cord
 - b. Two posterior spinal arteries that perfuse the posterior 1/3 of cord
2. Compromise to these vessels may cause paraplegia or tetraplegia from the same mechanism as a stroke.

H. **Autonomic nervous system** - See neuro A&P handout for more detail

1. **Sympathetic nervous system:** Controlled by the temporal lobe and the hypothalamus. It must send information along tracts that go through the brain stem and cervical cord before exiting at the thoracic and lumbar levels of the spine to reach target structures. Thus it is called the **thoraco-lumbar system**.
 - a. **Two main receptors**
 - (1) **Alpha** receptors: Constrict vessels, bronchioles
 - (2) **Beta** receptors: Dilate vessels, bronchioles; increase heart rate, strength of contraction, and speed of conduction.
 - b. SNS stimulation is also responsible for sweating, pupil dilation, shunting of blood from the periphery to the core, temperature regulation and autoregulating BP when the patient changes position.
 - c. When spine injury is at or above T6, the sympathetic nerves below the injury become disconnected from the nerves above they cease to function temporarily. They continue to operate automatically once the period of spinal shock is over.
 - d. Anything that stimulates the sympathetic nerves can cause them to overreact resulting in *autonomic dysreflexia*, a potentially lethal complication of SCI.
2. **Parasympathetic (Cranio-sacral system)**
 - a. Fibers carry signals to organs of the abdomen, heart, lungs, and skin above the waist that arise from the brainstem (**Vagus nerve**) and very high spinal cord. Nerves that supply the reproductive organs, pelvis and leg begin at the sacral level (S2, 3, 4).
 - b. **Cardiac:** Vagal stimulation slows heart rate and conduction. The Vagus nerve travels from its origins outside of the medulla to the heart via the carotid arteries – not through the cord. Thus, vagal tone is intact following cervical injury. When dysreflexia occurs, parasympathetic nerves attempt to control the rapidly increasing BP by slowing down the heart rate.
 - c. **GI/GU:** Disruption of lower PNS fibers results in loss of bowel/bladder tone and sexual function. (S2, 3, 4...urine, feces, semen hit the floor).

I. **Predisposition to injury**

1. Spine lesions are caused by direct and indirect forces
2. Cervical and thoraco-lumbar segments are most frequently injured since they are areas of highest spine mobility and the points at which the normal vertebral curves change direction. In younger adult patients, the greatest mobility occurs between C4 to C7. The most common spine injuries (about 50%) occur at C5 followed by C4 and C6 (UAB, 2008). In patients older than 65, degenerative stiffening of the spine

limits movement. The most mobile segment and the region of most injuries in these patients is C1 to C2 (Lomoschitz et al, 2002).

3. The next area of greatest mobility occurs at the thoraco-lumbar junction. A T12 - L1 bony injury is the second most commonly seen injury in the US (30%). A vertebral column injury at this level may allow "root escape" of L1 - L4 as the L1 nerve exits at the T10 vertebral level. Thoracic spines are injured less commonly since the spine is more rigid and is stabilized by rib attachments.
4. Lumbar fractures constitute (15%) of SCI and commonly occur due to improperly worn seat belts.

IV. Biomechanic mechanisms of injury

The mechanism of injury (MOI) has predictive value in identifying a SCI patient. Acute neurologic injury occurs when excessive energy is deposited in the spinal cord or its vascular structures (Hauswald et al, 1998).

- A. **Flexion:** Excessive forward motion of the head. Normal to 60% of flexion causes no stretching of cord. Hyperflexion is seen in severe deceleration. Greater stress occurs at C5-6 causing bilateral facet dislocations. May cause wedge fractures of anterior vertebrae, stretching or rupturing of interspinous ligaments, compression injury to the spinal cord, disruption of the disc with forward bilateral facet dislocations, and fracture of the pedicle.
- B. **Flexion with rotation:** Causes unilateral facet dislocation, fracture of the vertebra, or rupture of supporting ligaments.
- C. **Vertical compression/axial loading:** These usually stable injuries result primarily from transmission of forces along the line of the axial skeleton, such as might occur when landing on the feet or buttocks in a fall. The vertebral body is compressed and/or shattered, producing a "burst" fracture and fragments of bone may become embedded in the cord resulting in an anterior cord syndrome. It may also occur at the level of C1 as a result of a diving injury. The ring fractures in several places and "opens up" creating severe instability.
- D. **Hyperextension:** Excessive backward extension of the neck (as with a fall onto the forehead or rear impact collision) may rupture the anterior longitudinal ligament while the posterior column resists further displacement. Normal to 70% of extension results in no stretching of cord. Extreme cord stretch may occur as the head arcs backwards. Often seen in the elderly. The neurological deficit is usually incomplete resulting in a *Central Cord Syndrome*. It may also disrupt the intervertebral discs or compress the interspinous ligament.
- E. **"Whiplash":** Sudden hyperextension of the spine with prolongation of the neck stretches the ligaments from the force of the lower body moving forward as the head moves backward and downward. Hyperextension produces the greatest injury since forward flexion is limited as the chin strikes the chest and lateral flexion is limited as the head hits the shoulder.
- F. **Distraction injuries:** A distraction injury results from force applied to the spinal axis to distract or pull it apart (hanging). This may cause stretching of the cord or its supporting ligaments. The most common site of injury is in the cervical area. A drop of 18" greater than the patient's height causes irreversible CNS injury.
- G. **Pathological fractures** in those with osteoporosis or metastatic disease
- B. **Lateral bending:** Often caused by a direct blow to the side of the body. Causes varying degrees of posterior and lateral ligament damage, lateral compression of the vertebral body, and lateral displacement of the vertebra.
- C. **Penetrating wounds:** The incidence of gunshot-related SCI is accelerating. The most common open spine wounds are caused by missile injuries or stabbings. Points of special emphasis:
 1. Assume association with drugs/alcohol until proven otherwise

2. A bullet passing through the vertebral canal usually results in complete neurological deficit. Anatomic disruption to cord may be due to the blast effect (injury to sensitive neural tissue produced by the energy of the missile) or by direct injury of neural tissue as the bullet enters the spinal canal. Fragments of bone and/or disc disruption occasionally cause injury.
3. Search for penetrating thoracic and lumbar visceral injury. Hemopneumothorax, acute abdomen or great vessel injury take precedence for treatment.
4. Assess for CSF drainage from the wound.

V. **Structures affected**

- A. **It is important to remember that a patient can have trauma to the spine, ligaments or discs without cord injury and cord injury without bony injury.**
- B. **Ligaments:** Disruption of ligaments removes the stability of the spinal column allowing abnormal vertebral motion.
- C. **Bones:** Any of the vertebral bony elements may be disrupted. If moved out of their normal articulations, the bones can sublux (incomplete dislocation) or dislocate. Injury forces may fracture a spinous or transverse process, the pedicles, laminae, or vertebral body.
- D. **Discs:** With compression, these may herniate and compress the cord or nerve roots.
- E. **Cord substance:** See biochemical changes
- F. **Vessels:** Anterior spinal arteries are frequently compressed by an anterior dislocation injury creating a perfusion deficit in the anterior 2/3 of the cord.

VI. **Pathophysiology - biochemical changes in the cord (Nice to know only)**

- A. Neurologic lesions are dynamic (Hauswald et al, 1998). In many cases, the majority of damage may not occur due to the primary injury at the time of trauma but due to the **secondary injury** events over a period of hours. Trauma sets off a chain of reactions in the spinal cells wherein the tissue destroys itself. Most immune cells rarely enter the spinal cord. When the cord is damaged by trauma or disease, these cells flood to the area attempting to eliminate debris and releasing regulatory chemicals that may be beneficial or harmful (NINDS, 2007). Secondary neurochemical events involve the release of highly reactive free oxygen radicals, prostaglandins, and excitatory amino acid derivatives such as glutamate and aspartate, neurotransmitters that bind to at least four classes of receptors causing secondary damage from overexcited nerve cells. Glutamate and aspartate levels rise to six times normal baselines, resulting in concentrations that can kill neurons for up to one hour after injury. Limb dysfunction can occur when the cord is exposed to excitatory amino acids.
- B. Secondary insult also results from calcium- and sodium-mediated cell injury, inflammation, and apoptosis or genetically programmed cell death (Fehlings, 2001). **Receptors involved in secondary cord damage**
 1. Kainite and quisqualate receptors control channels for Na influx and K efflux from cells
 2. N-methyl-D-aspartate (NMDA) receptor has voltage-dependent channel for Na and K, and a channel for Ca influx that is regulated by Mg and zinc.
 3. Initial cellular swelling is attributed to Na influx and subsequent disintegration results from Ca influx. This type of cell death, where the cells swell and break open, is **necrosis**.
 4. Some cells commit suicide in a process called **apoptosis**. These damaged cells eliminate themselves without releasing harmful substances into the surrounding area, thus sparing their neighbors (NINDS, 2007).
 5. Antagonists such as phencyclidine, Ketamine, Mg, dextrorphan, and MK-801 have been studied to see if they would decrease secondary neurologic injury.

- C. Other substances that may cause neuronal damage are opioid peptides, such as endorphins. They have been implicated in the hypotension that occurs after SCI. For this reason, some physicians order naloxone. However, after injury there are a large number of kappa receptors that are insensitive to naloxone and the NASCIS II study failed to show any improved recovery using this agent.
- D. Further injury is caused by activation of membrane phospholipases that cause lipid hydrolysis, destroying cell membrane integrity. Enzyme activity produces free radicals (lipid peroxides), leukotrienes and prostanooids. Thromboxane A₂ increases soon after injury causing an inflammatory response that may persist for 18 hours, decreasing tissue perfusion.
- E. **End result:**
 - 1. Death of nerve cells within the spinal cord
 - 2. Disruption of nerve pathways
 - 3. Demyelination or loss of insulation around the axons

VII. **Primary and secondary spinal tissue injuries**

- A. **Concussion:** Constitutes 3% to 4% of all SCI. They often occur in patients with previous stenosis and are characterized by a temporary and spontaneously reversible physiologic disruption with transient deficits for 24-48 hours but no identifiable neuropathologic changes noted on exam of the cord. Considered an incomplete injury, they may be present in patients with simple compression fractures or in those who have no radiologic evidence of fracture. It is hypothesized that the dysfunction is due to a short-duration shock wave or pressure wave within the cord. Hyperreflexia without spasticity may be seen in this syndrome.
- B. **Cord contusion:** Bruising of the cord caused by fractures, dislocations, or direct trauma. It is associated with swelling, some tissue damage, and vascular leakage. Vascular or hemorrhagic disruption may cause temporary to permanent loss of function despite normal x-rays. Will take longer to recover than a concussion.
- C. **Laceration:** Tearing of cord substance as a result of a projectile or bone entering the spinal cord. They are likely to result in hemorrhage into the cord tissue, swelling due to the injury and disruption of some portions of the cord and their associated communication pathways.

They may be classified as complete or incomplete depending on the degree of damage.
- D. **Hemorrhage**
 - 1. Bleeding in or around cord as supplying vessels are torn or stretched.
 - 2. Bleeding alters neurochemistry, which further increases cord damage.
 - 3. Injury is related to the amount of hemorrhage.
- E. **Vascular interruption** of primary spinal cord vessels results in cord ischemia. If corrected early, the ischemic changes may be reversed. Necrosis from prolonged ischemia leads to permanent loss of function.
- F. **Transection**
 - 1. True transection is rarely seen. "Physiologic transection" due to vascular interruption is common.
 - 2. The cord necroses quickly. Loss of function may occur within four hours after traumatic insult. Transection totally interrupts all cord impulse conduction between the brain and the body. Incomplete transections present as cord syndromes.
- G. **Pinching of cord:** May occur with or without vertebral displacement
- H. **Stretching of cord:** Follows a distraction injury

VIII. **Vertebral column injuries** (*Nice to know only*)

- A. **Specific fractures**

1. **Simple:** Involves spinous/transverse processes; alignment is intact; some cord compression.
2. **Compression:** Body of vertebrae is wedged/compressed anteriorly usually from hyperflexion; cord compression possible.
3. **Comminuted (Burst):** Body of vertebra shatters and may be driven into the cord.
4. **Teardrop:** Small bone fragment breaks from anterior edge of vertebra and lodges within the cord. Surgery is necessary to remove the fragment. Cord damage is present if the fragment has penetrated cord substance.
5. **Cervical fractures involving C1 or C2**
 - a. **Jeffersonian ring fracture:** Rare fx of C1 where body splits into several parts at the thin anterior arch and the weak posterior arch near the vertebral arteries causing outward displacement of the lateral masses.
 - (1) Generally neurologically intact, initially
 - (2) If fracture fragments migrate, fatal injuries may occur
 - b. **Atlanto-occipital dislocation:** Rare avulsion of C1 body from occipital bone; immediately fatal.
 - c. **Odontoid (dens) fractures:** Severe vertical compression at C2
 - (1) Type 1: top) may have no nerve damage
 - (2) Type 2: isthmus)
 - (3) Type 3: into body of C2
 - (4) Results in both anterior and posterior displacement. Cord damage is possible. The spinal canal widens in this area, which allows a greater degree of mobility than in the thoracic area.
 - (5) May be missed on plain films. If suspected, take open mouth views, window CT or cervical MRI if necessary.
 - (6) Rx: Immobility (halo) and, in some cases, bed rest
 - d. **Hangman's fracture:** Bilateral avulsion fracture through arch of C2 with body of C2 separated from supporting posterior elements.
 - (1) Generally neurologically intact, initially
 - (2) Treated with immobility (halo) and bed rest
- B. **Dislocation:** Ligamentous damage may allow one vertebra to move with respect to the next, disrupting alignment and allowing stretching and kinking of the cord. Rotational force may cause facet joints, especially cervical, to slide over one another and cause "twisting" of the cord. The neck may become "locked" in this position.
 1. Considered "unstable" if two or more inflection points are disrupted.
 2. Spinal cord injury probable
- C. **Subluxation:** Partial/incomplete dislocation of one vertebra over another
 1. Initial mechanism of injury may cause subluxation, then vertebra can realign spontaneously due to repelling forces of the trauma
 2. Patient sustains SCI with initial subluxation or may have transient deficits
 3. Complete SCI possible with spontaneously resolved subluxation
- D. **Stable vs. unstable fractures**
 1. **Stable:** "The ability of the spine under physiologic loads to maintain relationships between vertebrae in a way that there is neither damage nor subsequent irritation to the spinal cord or nerve roots, AND in addition, there is no development of incapacitating deformity or pain due to structural changes."

2. Fractures are more stable if there is limited damage to supporting structures or bony alignment is preserved.
3. **Any injury is treated in the field as unstable** with the potential for worsening deficits.
4. Stable injuries usually pose no immediate spinal cord compromise, but potential for deficit remains; therefore, many Spinal Centers now perform early spinal fusion.

IX. **Manifestations of spinal cord injury**

A. Depends on the type and level of injury

B. **Spinal shock**

1. The term **spinal shock** refers to the temporary local neurological condition that occurs immediately after the spinal injury. Swelling and edema of the cord begins within 30 minutes after the insult creating the effect of a physiologic transection with disruption of nerve conduction. Physiologic transection or disruption of spinal cord conduction is not necessarily associated with anatomical transection.
2. Severe **pain** may be present just above the level of injury due to a zone of heightened sensitivity.
3. The patient presents with **flaccid paralysis**, no motor tone; flaccid sphincters and absent reflexes below the level of injury. They **cannot perceive any pain, temperature, touch, proprioception or pressure below the level of the lesion**. There is also **impaired thermoregulation**, absent somatic/visceral sensations below the lesion, bowel distension and loss of peristalsis (adynamic ileus).
4. May see varying degrees of spinal shock. Acute SCI does not always mean complete functional loss.
5. Spinal shock usually **subsides in hours to weeks** depending on the individual as spinal neurons regain some excitability and perianal reflexes return
 - a. Anal reflex: Anal sphincter puckers upon rectal examination
 - b. Bulbocavernosus reflex: Muscle contraction occurs as the glans penis is squeezed or the urinary catheter is pulled.
 - c. Spasticity usually supersedes the flaccid state after several weeks in areas where no function has returned.

C. **Neurogenic (spinal vascular) shock**

1. Temporary **loss of the autonomic function** of the cord at the level of injury which controls cardiovascular function. **Marked hemodynamic and systemic effects** within 30 minutes of injury, especially in high cervical lesions, due to **disruption of the sympathetic nervous system** fibers.
2. Lasts 4-6 weeks
3. Flaccid paralysis, absent reflexes
4. Loss of pain, touch, pressure below the level of injury
5. **Hypotension** is due to absent or impaired peripheral vascular tone (loss of SNS alpha receptor stimulation) allowing blood to pool in the enlarged vascular space causing a relative hypovolemia. With decreased cardiac preload, the ventricles do not stretch adequately, thus reducing the strength of contractions (Starling's law) and decreasing cardiac output. Systolic pressures are usually in the range of 80-100 mmHg. Venous pooling in the lower extremities makes the patient extremely sensitive to sudden position changes when going from a supine to a sitting position.
6. **Bradycardia** is due to unopposed vagal tone as the Vagus nerve runs outside of the cord and is not damaged in cervical lesions. The adrenal gland loses its SNS control and does not produce epinephrine or norepinephrine. These hormones are responsible for increasing HR.

7. **Anhidrosis** (lack of sweating) is due to disrupted SNS innervation to sweat glands. Skin is warm, dry and flushed below the lesion.
8. Absent/impaired thermoregulation: **Hypothermia** results as dilated blood vessels allow radiant loss of body warmth.

X. **Patient assessment**

- A. Since most SCI's result from some form of rapid deceleration trauma, it is not surprising that they are often accompanied by multiple associated injuries. Even in the absence of these injuries, SCI affects all body systems and predisposes patients to many complications.
- B. Although it is unknown how much spinal motion is permissible without harm during transport and during the initial workup (Hauswald et al, 1998), the current principles of spine trauma management include recognition of the injury, appropriate splinting/motion restriction, and reducing the chances of a secondary injury, i.e., aspiration pneumonia, hypovolemic shock due to a missed injury and inadvertently moving the patient with an unrecognized spine injury in a manner that may make it worse.

C. **Scene size up**

1. Determine scene safety. If unsafe, control and/or correct the hazard.
2. If a potential crime scene, make efforts to preserve the integrity of possible evidence.
3. Determine the total number of patients, which can be very difficult in calls after dark. Passengers may be ejected due to impact velocities, run from the site due to fear, or wander away in confusion as a result of head trauma. Call for additional help if needed; begin triage; activate local MCI plan if indicated.
4. Prepare and take essential equipment to scene: long spine board with immobilization devices/straps; airway & oxygen equipment; trauma box; PPE/BSI as needed.
5. **Determine mechanism of injury (MOI):** Assess type of energy imparted to patient: mechanical, thermal, electrical
 - a. Assess speed/trajectory of impact
 - b. Blunt/penetrating?
 - c. Flexion/extension/rotation/distraction?
 - d. Height of a fall
 - e. Restraints used or an airbag deployed?
 - f. The degree of vehicular damage
 - g. Patient's position in the vehicle
6. Classify the MOI as positive, uncertain, or negative as one decision point in deciding if spine motion restriction will be necessary.
 - a. **Positive (+) mechanisms** strongly suggesting the need for full spine motion restriction pending patient complaint and/or physical exam findings:
 - (1) High velocity crash (≥ 40 mph) with vehicle deformity > 20 inches
 - (2) Intrusion into passenger compartment > 12 inches
 - (3) Fall from $2\frac{1}{2}$ -3 times the patient's height (20 feet)
 - (4) Penetrating trauma near the spine
 - (5) Ejection from a moving vehicle
 - (6) Motorcycle crash > 20 mph or w/ separation of rider from bike
 - (7) Diving injury
 - (8) Auto-pedestrian or auto-bicycle > 5 mph impact
 - (9) Pedestrian thrown or run over
 - (10) Death of occupant in same passenger compartment
 - (11) Rollover crash

- b. **Uncertain mechanisms** that MAY OR MAY NOT indicate the need for spine motion restriction depending on patient reliability and physical exam findings:
 - (1) Moderate to low velocity MVC (< 40 mph); pt ambulatory at scene w/o evidence of + MOI
 - (2) MVC or other trauma patient with an isolated injury without positive assessment findings for SCI
 - (3) Isolated minor head laceration/injury without positive mechanism for spine injury – person trips and falls and hits their head
 - (4) Syncopal event in which patient was already seated or supine
 - (5) Syncopal event in which a patient was assisted to a supine position by a bystander
 - c. In **negative mechanisms**, the injury is obviously superficial or very remote from the spine, i.e., an extremity, and is insufficient to cause SCI. Some researchers suggest these MOIs do not require immobilization (Domeier et al, 1997; Goth, 1995).
- D. **Initial (primary) assessment:** The exam of any patient with suspected SCI must be tailored to their level of consciousness, reliability and MOI. If positive and/or uncertain MOI, it must be completed with the patient in a neutral position without any movement of the spine. Apply manual motion restriction to the head and neck while instructing the patient not to move unless you ask them to. The neck and trunk must not be flexed, extended or rotated. Determine whether the patient is deteriorating by questioning them about limb movement and sensation immediately after injury and comparing baseline findings to repeated physical assessments.
- 1. **General impression:** Overall look while approaching the patient(s); determine age and gender. Observe the position in which the patient is found. There are some positions quite characteristic of spinal cord injury such as a **tilt to the head** or an **arm up in a hold-up or prayer position**.
 - 2. **Determine if clinically evident immediate life threat exists.**
 - a. #1 priority: Save the life! Normalize vital signs; normal blood oxygen levels
 - b. #2: Manage the spine
 - 3. **Level of consciousness: AVPU** (ATLS discounts AVPU and strongly recommends early GCS)
 - 4. If they are combative and thrashing about, sedation and restraints may be necessary early for the patient's protection and spine stabilization.
 - 5. **Airway assessment/control**
 - a. If the patient is unable to talk, or is making gurgling, snoring or grunting noises, the airway is impaired. The tongue, secretions blood, vomitus, edema, foreign bodies or improperly inserted airways may occlude the oropharynx. A retropharyngeal hematoma associated with injury of the upper cervical spine (C2) may also impinge on the airway.
 - b. **Open and control the airway** starting with the least and progressing to the most invasive methods.
 - (1) Gain airway control while maintaining the head and neck in neutral alignment. Vertebrae that are still aligned, but injured can become unstable at any moment and can damage or sever the spinal cord causing permanent neurologic dysfunction
 - (2) Clear the mouth and suction as needed. Frequent suctioning is sometimes necessary in patients with c-spine injuries to maintain airway clearance and to prevent atelectasis. Limit suction

application to 10 seconds to prevent vagal stimulation and hypoxia. Preoxygenate well before and after each suction attempt.

- (3) **Manual methods:** Use modified jaw thrust or chin lift maneuvers. If these are successful in opening the airway, maintain it with an OPA or NPA depending on the presence or absence of a gag reflex and facial injuries.
- (4) If apneic, perform orotracheal intubation with **in-line stabilization**.
- (5) If patient has an impaired airway with protective airway reflexes intact or responds to pain, consider need for **drug-assisted intubation with in-line stabilization**. Premedicate with Lidocaine 1.5 mg/kg if head injury is present; benzocaine spray if gag reflex is intact. Sedate with midazolam and etomidate.
- (6) While blind nasotracheal intubation is sometimes preferred in spontaneously ventilating patients with mid to lower cervical spine injuries, it may result in extensive bleeding of the nasal mucosa and the posterior pharynx with resulting aspiration of blood and hypoxia. Success rates are not as good on first attempt as with sedated in-line intubation and *Klebsiella* may seed itself in the sinuses from obstruction of the sinus ostia. Advanced Trauma Life Support (ATLS, 2008) guidelines do not advocate its use as a primary airway access maneuver in trauma patients. This is not the preferred method of airway access unless patient entrapment makes other intubation routes impossible.
- (7) If the airway cannot be secured by intubation and the patient is unconscious with no gag reflex and cannot be ventilated adequately with a BVM device, attempt insertion of an alternate airway (King LTS-D). If that fails, perform a cricothyrotomy.

6. **Ventilations/gas exchange**

- a. Ventilatory assessment is often performed simultaneously with the airway inspection. In a patient who is complaining of neck or back pain, priorities include an examination of the respiratory system.
- b. Major ventilatory muscles include the diaphragm, intercostal, abdominal, and accessory muscles (sternocleidomastoid and scalenus)
 - (1) The diaphragm is innervated by the Phrenic nerve (C4) with small contributions from C3 and C5 (cervical plexus). [**C3, 4, and 5 keep the diaphragm alive!**] Lesions occurring at or above C3-4 may lead to paralysis of the diaphragm, apnea, and the need for assisted ventilations.
 - (2) Lesions occurring below C4 may allow partial or complete diaphragm function but suboptimal ventilatory function due to loss of the intercostal muscles that are innervated by T1-8 and may be responsible for as much as 35% of effective ventilations.
 - (3) The abdominal muscles are innervated by T9-12. Lesions above T12 paralyze the abdominal muscles and interfere with forceful expiration and coughing. These patients are susceptible to atelectasis, pneumonia and acute bronchial obstruction.
- c. Ask a cooperative patient to take a deep breath to assess muscle function and symmetry of chest expansion while you evaluate their pattern of speech. A patient with a lesion above T2 will only use their diaphragm and accessory muscles in the neck to ventilate. Intercostal muscles will have lost their nerve stimulation.

- d. Assess the general rate and depth of ventilations and capnography if available. Hypercapnia due to poor ventilation and hypoxia will worsen SCI and rescuers must be alert to the need for ventilatory assistance. A RR greater than 35 per minute may indicate borderline pulmonary function.
- e. **DO NOT ASSUME THAT A VENTILATING PATIENT WILL REMAIN THAT WAY!** Within 30-60 minutes following trauma, the injured spinal cord may swell due to the biochemical changes and ascend the lesion by two nerve roots. An injury that started at C5 or 6 may worsen to a loss at C3 or 4 and impair or eliminate ventilations.
- f. Early recognition of **hypoxia** is critical. The patient should be observed for signs of mental status and skin changes suggesting hypoxia. Monitor pulse oximetry on room air and after O₂ administration.
- g. **Interventions**
 - (1) If the patient is hypoventilating, assist ventilations with a BVM. Even in the absence of hypoxemia, intubation and ventilatory assistance is indicated if the patient shows signs of impending ventilatory failure.
 - (2) O₂ 12-15 L/NRM or BVM at 10-12 BPM
 - (3) Monitor chest excursion. If using a BVM, give just enough tidal volume to see the chest rise.

7. **Circulatory assessment for hemodynamic stability**

- a. Assess perfusion by comparing radial and carotid pulses for presence, general rate, quality, regularity, & equality along with skin color, temperature, and moisture. Most importantly, monitor the patient's LOC.
- b. Persistent hypotension in SCI is generally due to life-threatening hemorrhage -look for a site of blood loss. Neurogenic shock worsens hypovolemic shock as peripheral vasodilation (due to loss of sympathetic tone) prevents shunting of blood to vital organs.
- c. Monitor cardiac rhythm and intervals
- d. **Interventions**
 - (1) If no carotid pulse: initiate CPR
 - (2) Avoid sudden patient movements to prevent abrupt decreases in the BP
 - (3) Control external bleeding with direct pressure/pressure dressings
 - (4) Determine the need for vascular access based on the patient's hemodynamic status and the need for IV fluids and/or medications. Maintain adequate hydration and volume status to keep systolic BP at 90 unless accompanied by penetrating trauma. General hemodynamic resuscitation includes IV lines of NS or LR on a large bore catheter. Warm fluids if possible.
 - (5) Volume resuscitation may be used to increase stroke volume. However, in pure **neurogenic shock**, not associated with other injury, vagolytic drugs such as **atropine 0.5 mg rapid IVP** and pressor agents such as **dopamine in alpha doses** (10-20 mcg/kg/min) are preferred to overhydrating the patient with its resulting damage to the lungs and brain. Carefully monitor the patient's response to vasopressors. It may be less than expected since the sympathetic nervous system is compromised.

- (6) Control pelvic bleeding by wrapping the patient with a sheet, pelvic binder or PASG. May also consider applying PASG if VS do not respond to fluid challenges or meds to provide increased peripheral vascular resistance in the lower extremities if vasopressors are not available.
- 8. **D: Assess disability** (Brief neuro exam to determine immediate neurological life-threat)
 - a. Initial pupil assessment for size, shape, equality, and reactivity to light
 - b. If altered sensorium consider possible causes. Obtain blood glucose. If < 70: **DEXTROSE** at an age-specific concentration and amount.
 - c. GCS if not already done
 - d. Ability to move all 4 extremities
- 9. **E: Expose all injuries:** Undress to assess; recover the patient to maintain body warmth.

The target body temperature in patients with acute spinal injury is undergoing study. Traditionally, it has been held that maintaining normal body temperature is essential for trauma patients. Hypothermia impairs their ability to unbind oxygen from hemoglobin. It also predisposes the patient to adult respiratory distress syndrome (ARDS) and disseminated intravascular coagulation (DIC), a potentially lethal complication where patients clot and bleed at the same time. However, successful outcomes following controlled cooling in patients with return of spontaneous circulation following cardiac arrest and in those with a stroke offer the possibility of improved outcomes for patients with SCI.
- 10. **Transport decision:** A decision must be made whether to complete the focused history and PE on-scene or to transport immediately with further assessments and interventions enroute.
 - a. **The actual or potentially unstable patient should be transported as soon as possible based on impairment of the ABCs.** Attempt to keep on-scene times as short as possible (BTLS goal: 10 minutes or less).
 - b. **Level I:** SCI with neuro deficit, in combination w/ injury to another system, or hemodynamic instability; penetrating SCI
 - c. **Nearest TC Level I or II:** Suspected isolated SCI; hemodynamically stable
- E. **Focused history and physical exam (secondary assessment)**
 - 1. Positive or uncertain mechanism of injury?
 - a. **NO:** Focused assessment specific to cc/injury; VS; SAMPLE history.
 - b. **YES:** While continuing to stabilize the spine, perform a **rapid trauma assessment**.
 - 2. **Establish neurological reliability for exam**
 - a. Evaluate the patient's **reliability** before performing a focused or detailed assessment
 - b. **To be considered reliable**, the patient must appear calm, cooperative, alert, free of impairments, alert and oriented, and perform cognitive functions appropriately
 - c. **Unreliable patients:** Patients who are experiencing an acute stress reaction, brain injury, intoxication from drugs and/or alcohol, abnormal mental status, distracting injuries, or communication problems cannot provide a reliable exam. **An unreliable patient always means an unreliable neuro exam.** Inebriated, confused and multiply injured patients

are especially likely to have clinically unapparent spine lesions with very subtle presentations.

Distracting injuries are defined as possible long-bone fractures, rib fractures, pelvic fractures or clinically significant abdominal pain. If these are present with an uncertain or positive mechanism of injury, patients should be or remain immobilized.

- d. **The patient whose examination is unreliable or who is in coma must have continuous spine protection until the presence of an injury can be excluded radiographically** (ATLS, 2008).
 - e. If combative and thrashing about, sedation and restraints may be necessary for the patient's protection.
3. A reliable patient with a history of trauma should be asked if they have any midline or lateral spine **pain** or if they experience any pain when they attempt to move their head or neck. An unreliable patient or one with cervical lesions may be unable to discern pain and may have associated injuries to the face, head, chest, abdomen, and musculoskeletal system. Therefore, a thorough exam of the entire body is indicated.
 - a. The pain associated with SCI may be **radicular** or associated with nerve root compression. Patients may describe waves of stabbing or sharp pain or a band of burning pain at the point where normal feeling stops. Prepare patient for unusual sensory experiences common in SCI; phantom pains, limbs "floating", hot skin, feet twisted or moving, or burning pains in the arms. Reassure patient that unusual sensory experiences are common.
 - b. If any back or spine pain is present, spine motion restriction must be maintained.
 - c. Use touch to convey concern; pay attention to level of actual sensory losses and touch them where they can still feel it.
 4. Rapidly inspect and palpate head, neck, chest, abdomen, pelvis, all extremities, and back for DCAP-BLS; TIC; PMS (deformity, contusion, abrasion, puncture wounds, bruising, laceration, swelling; tenderness, instability, crepitus; pulses, motor and sensory integrity).
 5. Obtain baseline VS; monitor at least every 15 min. and more frequently depending on the degree of compromise and instability. Compare and contrast VS and PE findings to suspect hypovolemic vs. neurogenic shock.
 6. SAMPLE history

F. Detailed physical exam enroute

1. **HEENT:** All patients with injuries above the clavicles must be assessed for SCI. Kaups and Davis (1998) concluded that indirect spine injury does not occur in patients with isolated GSW to the head and therefore they do not need cervical immobilization that could complicate airway management. In addition to assessing for local trauma, examine for a characteristic head tilt or inability to move spontaneously.
2. **Pulmonary system/thorax:** Most frequently occurring associated injuries are to the torso including fractured ribs, sternum, clavicle, scapula or pelvis.
 - a. Reassess symmetry of chest wall movement, work of breathing, use of accessory muscles, and breath sounds. Retraction of neck muscles is a sign of poor ventilation and may indicate respiratory decompensation. Paradoxical ventilations may indicate diaphragmatic impairment due to SCI.

- b. Partial paralysis of the diaphragm can occur in cervical injury; one side only may be affected.
 - c. Evaluate for thoracic aortic tear as patient may exhibit signs of paraplegia due to interrupted vascular supply to the thoracic cord.
 - d. Re-assess oxygenation via mental status, SpO₂, and skin color and ventilatory status with capnography.
- 3. **Cardiovascular system**
 - a. Reassess for hemodynamic compromise/shock: LOC, feeling of dizziness, and presence of ectopic beats or escape rhythms (junctional or ventricular).
 - b. Continuously monitor ECG for severe bradycardia which may require a pacemaker if fluid therapy and drugs are ineffective.
- 4. **GI System**
 - a. Exam is unreliable in the presence of a neuro deficit.
 - b. Inspect for signs of trauma, contour. Severe gastric distention impairs the ability to ventilate.
 - c. Auscultate bowel sounds in at least one quadrant for one minute. Anticipate physiologic ileus in acute stage. Patient is at risk for vomiting.
 - d. Palpate all four quadrants for guarding and/or rigidity. Patient may be insensitive to pain and will not develop a rigid abdomen due to absence of muscle tone.
- 5. **GU System:** Assess for blood at the urinary meatus, rectum, and vagina. Note any signs of loss of bowel or bladder control. Males: Assess for scrotal swelling/ecchymosis. If present, suspect pelvic fracture. Assess for **priapism** (sustained erection).
- 6. **Extremities:** Inspect for wounds, deformity, edema/swelling and angulation. Palpate for deformities, tenderness, instability, crepitus, false motion or resistance to attempted motion suggesting fracture. Observe for abnormal position of the arms (hold-up). Protect paralyzed limbs.
- 7. **Vertebral assessment**
 - a. While maintaining spine alignment, palpate over each posterior spinous process for **pain, point tenderness**, and/or **deformity**. Begin at the neck and work towards the pelvis. Pain occasionally radiates to the arms, about the chest and abdomen, or into the lower extremities. The absence of pain or tenderness along the spine in a neurologically normal (reliable and negative PE) patient without distracting injuries virtually excludes the presence of a significant SCI (Domeier et al, 1997).
 - b. Other diagnostic signs and symptoms include: prominence of spinous processes, pain on attempted motion, edema, ecchymoses, visible deformity and paravertebral muscle spasms.
- 8. **Skin:** Assess skin integrity and body temperature. Quadriplegics (tetraplegics) may be **poikilothermic** (body temperature assumes environmental temperature) due to absent input from periphery for temperature control. Temperature may decrease to 95.6° - 96° F or may elevate to 101°-102° F. In neurogenic shock the skin is warm, dry, and flushed due to vasodilation and absence of sweating (anhidrosis).
 - a. **Problem:** Skin break down is a major complication of SCI occurring as a result of pressure, primarily over the bones of the buttocks (particularly ischial tuberosities and the trochanters at the hip) that are in contact with the spine boards. **Two hour window** on the spine board before skin

breakdown begins. Thirty-two percent get a skin lesion within 24 hours of injury. There are blood distribution shifts to the skin and subcutaneous tissues in addition to changes in muscle tone and sensation. Normal skin and soft tissue elasticity is lost. Increased stiffness, vascular alterations, and alterations in muscle tone combine to significantly reduce the skin's ability to withstand pressure (UAB, 2008).

b. **Interventions**

- (1) Pad bony prominences that lie against the spine board to prevent pressure sores. May use an inflated BP cuff to cushion small of back, behind knees, etc.
- (2) At the hospital, patients should be removed from hard spine boards as soon as possible (after injury is ruled out) to prevent the development of decubitus ulcers and persistent restlessness. Unconscious patients are at greater risk for this complication (ATLS, 2008).

9. **Neurological exam**

a. **Level of consciousness:** Consider head injury, chemical impairment, hypoxia, hypoglycemia, and/or hypothermia as sources of decreased LOC

- (1) Mental status exam: awareness; orientation
- (2) Quantify responsiveness with Glasgow coma score: Do not score the patient as having no motor response if limbs are paralyzed. Ask them to blink or move some facial muscle that would be innervated by a cranial nerve.
- (3) Sometimes the presence of SCI is obscured by other trauma; especially when the patient is unconscious.

b. **Cranial nerve assessment:** especially for high cervical injuries

- (1) In high cervical lesions (C2, 3, 4), traction effect is possible on these nerves, so function may be diminished or absent

Observe for a drooping eyelid (ptosis) and a small pupil (Horner's Syndrome) that indicates injury to C3.
- (2) Assess function of glossopharyngeal (IX), vagus (X), accessory (XI) and hypoglossal (XII) nerves.
- (3) Accessory muscle function can be confused with shoulder shrug ability (CN XI).

c. The **purpose of the focused neurologic evaluation** in the field is to establish the

- (1) baseline levels of the lesion for later comparisons;
- (2) completeness of the lesion; and
- (3) type of cord syndrome, if lesion is incomplete.

d. **Neurologic level of injury:** There is frequent discrepancy between the bony and neurologic levels of injury due to the way the spinal nerves enter between the vertebrae and then ascend inside the spinal canal before attaching to the spinal cord. Use the body as its own control comparing results from the left and right sides and/or upper vs. lower extremities.

- (1) **Motor exam:** Motor components of spinal nerves innervate specific tissues and muscles of the body in regions called **myotomes**. As the body matures, some muscles merge and control is not as specific as it is with dermatomes.

- (a) Bilaterally assess each major motor group from the top down to identify the lowest spinal segment associated with normal voluntary motor function, then determine the extent of function in segments below this level. This becomes the initial neurologic level of injury. Monitor for possible ascending lesion, i.e., worsening deficits, small decreases in motor ability or altered respiratory patterns, especially with cervical injuries.
- (b) Ask the patient to simultaneously flex (C5) then extend (C7) both elbows then wrists (C6); abduct the fingers and keep them open against the examiner's resistance and then close (adduct) the fingers and attempt to open them against the examiner's resistance (T1). As an alternative maneuver, have the patient curl all four fingers while the examiner applies opposing pull with their fingers to determine strength against resistance. This will test the finger flexors (C8).
- (c) In the lower extremities, ask the patient to bend and extend the knees then plantar flex the feet and ankles like pressing down on a gas peddle of a car (S1, 2) and dorsiflex the toes to gravity and against resistance (L5).

The presence of paraplegia or quadriplegia (tetraplegia) is presumptive evidence of spinal instability (ATLS, 2008).

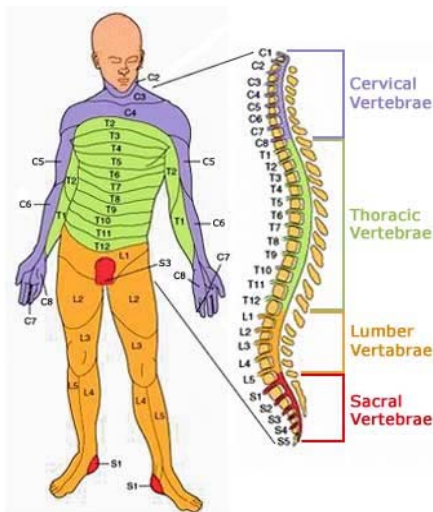
(d) **Landmark myotomes:**

- (i) C3-5: Diaphragm
- (ii) C5: Elbow flexors: biceps, brachialis, brachioradialis
- (iii) C6: Wrist extensors
- (iv) C7: Elbow extensors: triceps
- (v) C8: Finger flexors: flexor digitorum profundus to middle finger
- (vi) T1: Hand intrinsics: interossei; small finger abductors (abductor digiti minimi)
- (vii) T2-7: Intercostal muscles
- (viii) L2: Hip flexors: Iliopsoas
- (ix) L3: Knee extensors: Quadriceps
- (x) L4: Ankle dorsiflexors: Tibialis anterior
- (xi) L5: Long toe extensors: Extensor hallucis longus
- (xii) S1: Ankle plantarflexors (Gastronemius, Soleus)
- (xiii) S4-5: Anus, bowel/bladder

- (e) Motor integrity in an unconscious patient is evaluated by assessing their response to a pain stimulus. C-spine injury is likely if an unconscious patient grimaces, vocalizes, or opens his eyes in response to pinching the border of the trapezius muscle but does not move his limbs. If a patient moves both arms but neither leg in response to pain, suspect a SCI below the cervical level. Pain response should be tested at several locations (including points with cranial nerve distribution such as the supraorbital ridge or earlobe) before assuming absence of a response.
- (f) If motor exam cannot be completed due to local injury, the entire exam is unreliable and spine motion restriction is required.

(2) **Sensory exam**

- (a) Sensory components of the spinal nerves innervate specific and discrete areas of the body surface called **dermatomes**. They are distributed from the occiput of the head (C2) to the buttocks and rectum (S5).
- (b) Question the patient about abnormal sensations they may be experiencing, e.g., "pins and needles", electric shock, or hyperacute pain to touch (hyperesthesias). As with the motor exam, determine the lowest level of normal sensation as well as any areas of intact "spared" sensation below this level.
- (c) **Conscious patient:** Evaluate light touch by using a cotton swab on a broken wooden stick or a partially opened paperclip. Bilaterally assess sensory integrity from the feet up, making sure to compare equality on both sides. With their eyes closed, touch the patient, alternating between the sharp and fuzzy or dull ends of the stimulus. Ask the patient to localize the touch and ask if they can distinguish between sharp and dull sensations. Touch the back of the patient's legs to evaluate for sacral sparing and move up the front of the legs, anterior abdomen and chest, then the hands and arms. Draw a mark on the patient's body the level at which he first reports that he can feel the touch. Presence of any abnormal sensation indicates the need for spine motion restriction.



Landmark dermatomes

- C2: Occipital protuberance on back of head
- C3: Supraclavicular fossa
- C5: Lateral side of the antecubital fossa
- C6: Thumb and medial index finger (6 shooter)
- C7: Middle finger (the bird...)
- C8: Little finger
- T2: Apex of axilla
- T4: Nipple line (4th ICS)**
- T10: Umbilicus**
- L1: Inguinal line
- L2: Mid-anterior thigh
- L3: Medial aspect of the knee
- L5: Dorsum of the foot
- S1-3: Back of leg
- S4-5: Perianal area

- (3) **Proprioception:** Evaluate the patient's ability to appreciate limb position sense while the eyes are closed by moving the thumb and great toe up or down. Position sense is mediated by the posterior (dorsal columns) of the cord.

10. **Deep tendon reflex exam:** Not usually assessed in the field. Reflexes are usually absent initially. They return several hours to weeks post-injury. If reflexes are intact, preservation of motor and sensory activity in the same spinal cord segments is likely. Once cord reorganization begins, the bulbocavernosus reflex (external anal sphincter function) is the main indicator of future bowel, bladder, and sexual functioning.

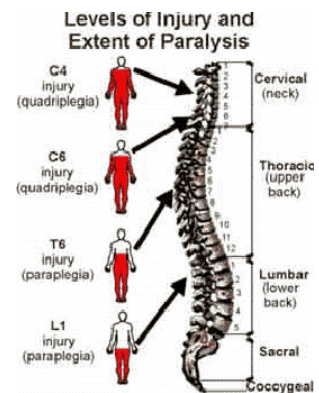
G. **Clinical exam findings suggesting positive (+) spine injury:** (Think Ps)

1. **Pain** in neck or spine (patient complaint)
 2. **Painful movement**
 3. Spine **tenderness/pain/deformity to palpation**
 4. **Paralysis/paresis**; abnormal motor exam
 5. **Paresthesias** (upper and/or lower extremities): tingling, numbness, burning
 6. Abnormal **perception/response** to pain stimulus (sharp/dull; pin prick)
 7. **Ptosis** (Horner's Syndrome)
 8. **Priapism**
 9. **Position abnormality**: head tilt, arms in hold-up or prayer position
 10. **Proprioception deficit**
 11. **Poikilothermia**
 12. **Parasympathetic dysfunction** in lower sacral nerves: loss of bowel/bladder tone flaccidity of intra-abdominal muscles, loss of peristalsis
- Plus...
13. Diaphragmatic breathing
 14. Neurogenic shock: Decreased BP, bradycardia, decreased RR, warm, *dry* skin with systemic hypothermia
 15. Head trauma with altered mental status
 16. Absence of sweating below level of injury (anhydrosis)

H. **Neurologic extent of injury (complete versus incomplete lesions)**

1. A complete evaluation of both sensory and motor levels at the hospital will determine the neurological level of SCI. The patient will be categorized according to classifications published in the *International Standards for Neurological and Functional Classification of Spinal Cord Injury*, revised in 1996 and endorsed by the American Spinal Injury Association (ASIA). Generally, the higher the injury, the more extensive the impairment.

- a. C1 to T1 nerve roots: **Tetraplegia (formerly quadriplegia)** (51.6%). Patients experience loss of feeling and/or movement in the head, neck, upper chest, arms, hands, and/or fingers plus the losses of paraplegia.
- b. T2 or below: **Paraplegia** (46.3%). These patients experience losses in the chest, abdomen, hips, legs, feet, toes, and rectum.



2. If the cord is severely damaged at the injury site, there is generally **complete** loss of sensation and voluntary muscle control in areas innervated more than two levels below the bony lesion persisting more than 48 hours.
3. Recovery after a complete injury is usually minimal, although neurological function may improve by several grades. If there is any evidence of motor or sensory function in the S4/S5 area on rectal exam, one of three **incomplete** injury classifications is given according to the ASIA Impairment Scale:
 - a. **A = Complete:** No preservation of sensorimotor function below the level of injury including sacral segments S4-5. Both sides of the body are equally affected.
 - b. **B = Incomplete: Preserved sensation only** - Sensory but no motor function is preserved below the neurological level; extends through sacral segments S4-5. Preservation of any demonstrable sensation excluding phantom sensations.
 - c. **C = Incomplete: Motor function is preserved** below the neurological level and the majority of key muscles below the neurological level have a muscle grade < 3. Sensory function may or may not be preserved. A

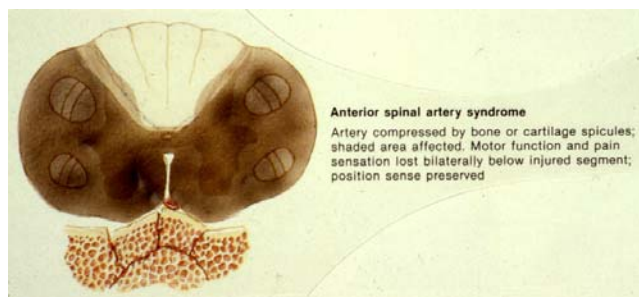
person may be able to move one limb more than another and may have more functioning on one side of the body than the other.

- d. **D = Incomplete:** Motor function is preserved below the neurological level and the majority of key muscles below the neurological level have a muscle grade greater than or equal to 3.
- e. **E = Normal:** Sensory and motor function is normal
- f. Groupings by incidence (UAB, 2008)
 - (1) (PI) Incomplete paraplegia (18.5%)
 - (2) (PC) Complete paraplegia (23%)
 - (3) (QI) Incomplete tetraplegia (34.1%)
 - (4) (QC) Complete tetraplegia (18.3%)

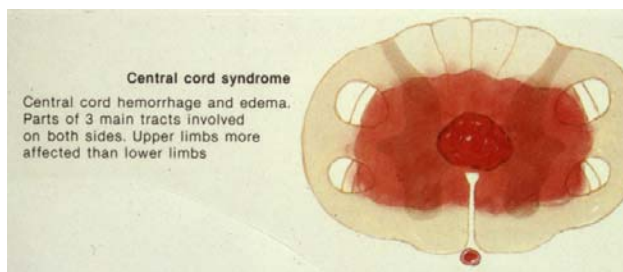
I. **Spinal cord syndromes:** incomplete injuries

1. **Anterior cord syndrome**

- a. **Mechanism of injury:**
Occurs with flexion injuries or fractures
- b. **Pathophysiology:**
Interruption of the anterior spinal artery by occlusion or spasm and the posterior ligaments with resultant loss of perfusion to the anterior 2/3rds of the cord. Prognosis for recovery is poor.
- c. **Physical findings**
 - (1) Paralysis and loss of pain, temperature, and touch below the lesion due to involvement of the corticospinal and spinothalamic tracts.
 - (2) Preserves light or crude touch, two-point discrimination, position sense, vibration, and deep pressure mediated by the dorsal columns.

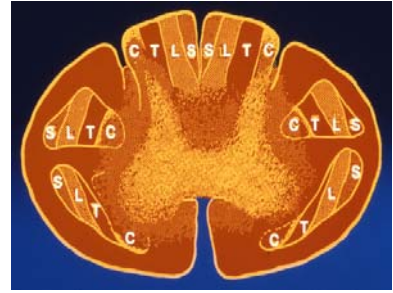


2. **Central cord syndrome**



- a. **Mechanism of injury**
 - (1) Hyperextension injury causing damage, hemorrhage or edema to central cervical segments. Usually no fracture or bony disruption, but tears occur to the anterior longitudinal ligament.
 - (2) Central cord lesions usually occur following a "smash" or "pinch" type injury of short duration and with a less violent force. The "pinch" results from bony spurs, ligamentous flavum and intervertebral discs and are more prevalent in the older population with preexisting conditions such as spondylosis and stenosis due to arthritic changes.

- b. **Pathophysiology:** Motor fibers in the cord are distributed in a unique fashion. Those that are most needed (cervical) are most central in the cord. Those that are less critical are distributed towards the surface. In central cord syndrome, the more centrally located (cervical, thoracic) motor and sensory tracts are affected. Lateral fibers to the lower extremities remain intact.
- c. **Physical findings:** Patient presents with a greater loss of function in the upper extremities than lower and a variable sensory loss of pain and temperature. May have bowel and bladder dysfunction. ("They can dance, but they can't play the piano.")
- d. **Treatment:** Patient may be placed in halo traction on a Roto-rest bed at the hospital while ligaments heal or be placed in a Jewett brace if a thoracic injury.
- e. **Usually good prognosis.** May have some residual loss in hands.
3. **Posterior cord syndrome:** Associated with extension injuries to the C-spine with pure injury to the dorsal column and is rarely seen. This injury produces dysfunction in the dorsal columns resulting in a decrease in light touch, proprioception, and vibration leaving motor and most sensory function intact. With visual cues, will rehab well.
4. **Brown-Sequard syndrome** – rare; generally good prognosis
- a. **Mechanism of injury:** Most common with penetrating trauma and often due to a cervical lesion.
- b. **Pathophysiology:** Hemi-transection of the cord with complete damage to all spinal tracts on the involved side.
- c. **Clinical presentation**
- (1) Injury to corticospinal motor tracts (that have crossed in the medulla) causes an ipsilateral (same side) motor loss below the lesion. Damage to posterior columns that do not cross causes loss of touch, position, pressure and vibration on the same side below the lesion.
 - (2) Injury to ascending spinothalamic (sensory) fibers that cross immediately after entering the cord causes contralateral (opposite side) sensory loss of pain and temperature below the level of the lesion.
 - (3) Most significant residual deficit is in ipsilateral upper extremity.
- d. You may encounter a Brown-Sequard and Central Cord Syndrome in same patient.
5. **Root syndromes:** Can involve any area although they are more common in the cervical and lumbar areas and are considered to be distinct from SCI. Compression of nerve root(s) on one side at isolated levels occurs due to disk herniation or subluxation of a vertebral body. Patient presents with specific deficits at level of compression.

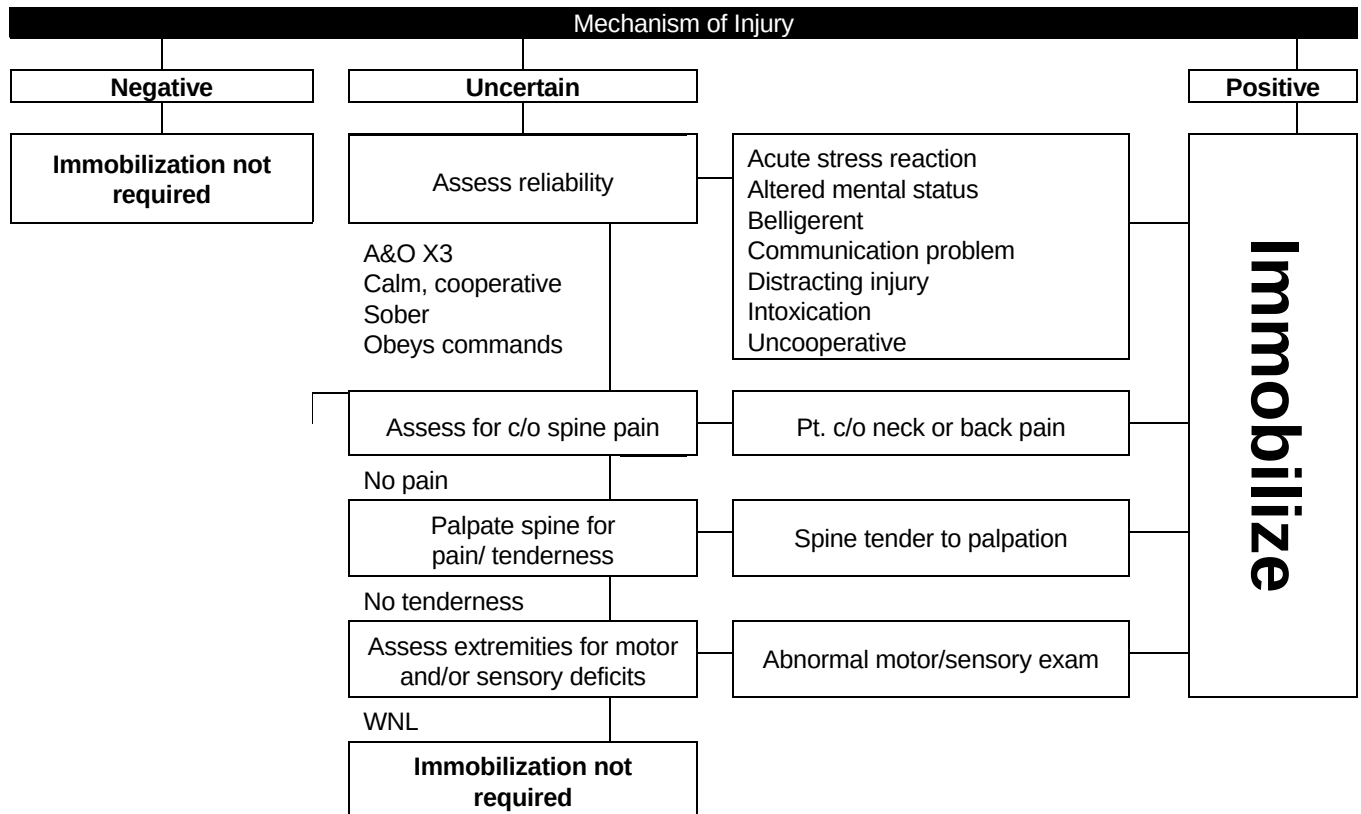


6. **Horner's syndrome**
 - a. Seen with high cervical lesions. They involve damage to the cervical portion of sympathetic chain.
 - b. Characterized by ptosis, pupillary constriction (miosis), and anhydrosis (inability to sweat) on the same side as the injury.
7. **Conus medullaris syndrome:** Injury of the sacral cord (conus) and lumbar nerve roots within the neuronal canal, which usually results in an areflexic bladder, bowel, and lower limbs. Sacral segments may occasionally show preserved reflexes, i.e., bulbocavernosus and micturition reflexes.
8. **Cauda equina syndrome:** Injury to the lumbosacral nerve roots within the neural canal resulting in areflexic bladder, bowel, and lower limbs.

XI. **Treatment of the patient with suspected spinal cord injury**

- A. Since most SCIs result from some form of rapid deceleration trauma, it is not surprising that they are often accompanied by multiple associated injuries. Even in the absence of these injuries, SCI affects all body systems and predisposes patients to many complications.
- B. Dress wounds, splint fractures if time permits
- C. Seizure/vomiting precautions
- D. Assess and treat for pain.
- E. **Spine motion restriction**
 1. Although it is unknown how much spine motion is permissible without harm during transport and during the initial workup (Hauswald et al, 1998), the current principles of spine trauma management include recognition of the injury, appropriate splinting, and reducing the chances of a secondary injury, i.e., aspiration pneumonia, hypovolemic shock due to a missed injury and inadvertently moving the patient with an unrecognized spine injury in a manner that may make it worse.
 2. **Application of spine motion restriction – decision tree**
 - a. Traditional belief held that immobilization is generally protective and that patients with possible spine fractures will have a higher incidence of neurologic injuries if immobilization is not carried out.
 - b. Conservative approaches to applying spine immobilization without consideration of the patient's physical exam are not without problems. Immobilization is uncomfortable or painful at the occipital prominence and lumbosacral areas. It causes a significant restrictive effect on pulmonary function in healthy non-smoking adults, poses an increased risk for aspiration and skin breakdown, can augment intracranial pressure (ICP) from application of cervical collars, may adversely prolong scene times, and constitutes a potentially unnecessary expense (Orledge & Pepe, 1998; McHugh & Taylor, 1998).
 - c. The decision to immobilize **depends to a large extent upon three factors:** Mechanism of injury, patient reliability, and neuro exam findings (EAST, 2000). Mechanism of injury is sometimes excluded as a determining factor for spine motion restriction.
 - (1) **Full spine motion restriction is generally indicated for the following:**
 - (a) Positive mechanism of injury
 - (b) Uncertain MOI in an unreliable patient
 - (c) Positive physical exam

- (2) A person who has sustained trauma with the potential for cervical spine injury may be managed **without spine motion restriction** ONLY IF:
- (a) there is no significant mechanism of injury,
 - (b) the individual is alert and oriented (calm and cooperative),
 - (c) the individual does not show evidence of intoxication or chemical impairment,
 - (d) the individual does not have a significant "painful, distracting injury" or craniofacial trauma,
 - (e) the individual does not complain of pain in the neck or spine,
 - (f) the individual does not have neurological deficits suggesting SCI (motor or sensory deficits in the extremities),
 - (g) there is no tenderness on palpation of the spine, and
 - (h) the individual can move the neck in a full range of motion without pain.
- d. It is important for EMS personnel to document all steps of the process completely and thoroughly. Reassess the patient regularly. Clearly document patient reliability, mechanism of injury and negative physical exam findings if no immobilization is applied.
- e. **When in doubt, treat as a "positive spine injury" and apply complete spine motion restriction.**



F. Spine splinting principles

1. Primary goal is to prevent further injury
2. The spine should be considered one long bone for splinting purposes with a joint at either end (head and pelvis). It is impossible to isolate and splint at only one

injury site. Secondary spine injuries may be preventable with proper immobilization.

3. **Spine motion restriction** means manual stabilization of the head and neck in an **eyes forward position** unless pain/resistance to movement is encountered, followed by the application of an appropriately sized rigid cervical collar (unless contraindicated); axial alignment of the head and torso splinted to a long spine board using head blocks or towel rolls to limit lateral head movement, forehead straps or tape to limit neck flexion; and backboard straps or cravats to secure the patient to the board (shim sides of patient if necessary).
4. Spine motion restriction may be applied to a supine, standing, or a seated patient.
5. The use of **cervical collars** has become somewhat controversial; however, many neurosurgeons and orthopedists still recommend the use of a properly fitted rigid cervical collar as an adjunct with lateral immobilization devices.
6. **Positioning:** Neutral positioning allows for the most space for the cord thus reducing cord hypoxia and reducing excess pressure on the tissue. No pillows should be placed under the patient's head, however, MRI studies reveal that the adult cervical spinal canal is largest and the c-spines are anatomically aligned if the head is elevated 2 cm by padding under the occiput with a folded towel or pad. Pediatric patients have a relatively larger head and need padding under the torso to maintain alignment and prevent neck flexion if immobilized on an adult spine board. Some peds spine boards are constructed with a recessed portion for the head or torso padding.
7. **C-collar measurement:** Determine the correct size by placing your fingers parallel to the patient's neck, measuring from the top of the trapezius muscle to the bottom of the chin. Transfer that measurement to the c-collar and size the collar to approximate that height.
8. Patients on the ground should be log-rolled into a supine position. Log roll or use a scoop stretcher to place a supine patient on a long-spine board. Unconscious patients with the head passively rotated to one side should be maintained in that position. If the patient is in respiratory distress, attempt to bring the head into axial alignment using *gentle* traction. Stop all movement if resistance is encountered. Ask conscious patients to turn their own head if there are no overt signs of neuro deficit. If the patient experiences any neck pain or tingling in the arms or hands, stop all movement. Immobilize as found.
9. Stable patients in vehicles after an MVC without cardiorespiratory compromise and who are presenting with head/neck pain or tenderness to palpation; head injury; any neurovascular impairment; spine deformity on palpation; and/or positive MOI suggesting possible SCI (starred windshield) should be manually immobilized with a rescuer's hands to the head prior to placing a c-collar and a vest-type extrication device (**KED**) for extrication onto a long spine board.
10. Unstable patients in vehicles with cardiorespiratory compromise or those where the scene is unsafe should be **rapidly extricated** onto a long spine board.
11. Once positioned on the long spine board, padding should be used to fill all voids and limit movement of the patient. Apply lateral immobilization devices to the head in the form of commercial devices or a blanket roll. Secure patient to the board using straps over the shoulders and across the chest, over the pelvis and legs.
12. Ambulatory patients complaining of neck pain or tenderness to palpation should be immobilized using the **standing backboard technique**.

G. **Helmeted patients**

1. **Special assessment needs:** Fit of helmet and movement within the helmet; ability to gain access to airway
2. Types of helmets
 - a. Sports
 - b. Motorcycle
3. **Helmet removal guidelines:**
 - a. Type of helmet influences the techniques used for removal
 - b. Fitted **helmets** should be left on the patient and removed in the ED unless the patient is experiencing airway/ventilatory impairment that cannot be corrected with the helmet in place. Never attempt to remove a helmet alone. It takes a minimum of two persons.
 - c. **Indications for leaving helmets in place prior to C-spine clearance**
 - (1) Good fit with little or no head movement within the helmet
 - (2) No impending airway or breathing problems
 - (3) No interference with ability to assess airway and ventilations
 - (4) Removal may cause further injury
 - (5) Proper spine immobilization can be performed with helmet in place
 - d. **Remove helmets immediately if any of the following occur**
 - (1) The helmet does not immobilize the patient's head
 - (2) You cannot securely immobilize the patient with the helmet in place
 - (3) The helmet prevents airway access or maintenance
 - (4) The helmet prevents assessment of anticipated injuries
 - (5) Cardiac arrest

H. **Pharmacotherapy**

1. Short-acting, reversible **sedatives** are recommended for the acutely agitated patient after a correctable cause of the agitation has been excluded, i.e., hypoxia. Considerable clinical judgment on the part of the EMT-P is required. The ability to control and protect the airway is required, as well as the capability to provide controlled ventilations
2. The use of **steroids** is very controversial. They are not used in the NWC EMSS. Steroids do not help the primary injury to neurons or glial cells. However, neural tissue does not tolerate inflammation well. Inflammatory processes set up an environment that does not support tissue repair and poses a potential chemical barrier to tissue regrowth, regeneration, and repair. Cavities develop weeks after injury and may extend an intense inflammatory reaction to areas not directly injured which becomes the process targeted by high dose steroid therapy (Fitch, 2007).
 - a. **Current review of the international literature shows that the majority of patients with SCI are NOT getting steroids (Fitch, 2007).**
 - (1) **National Association of EMS Physicians:** Evidence for use of high-dose steroids for spinal cord injury remains inconclusive and not standard of care for out-of-hospital emergency medical care.
 - (2) Canadian Spine Society and Canadian Neurosurgical Society: There is insufficient evidence to support the use of high-dose methylprednisolone within eight hours following an acute closed spinal cord injury as a treatment standard or as a guideline for treatment. Treatment option for which there is weak clinical evidence.

- (3) AANS and Congress of Neurological Surgeons (Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries): Insufficient evidence to support as treatment standard or treatment guidelines; treatment with methylprednisolone for 24 or 48 hours recommended as option in treatment of patients with acute SCI (but should be undertaken only with knowledge that evidence suggesting harmful side effects more consistent than any suggestion of clinical benefit) (Fitch, 2007).
3. Latest investigational intervention includes the initiation of induced **hypothermia** immediately following injury to decrease metabolic demands of the tissues and prevent cellular destruction. However, this is very controversial and the benefits have not been proven in randomized controlled prospective studies.

II. **Complications of spinal cord trauma**

- A. **Aspiration:** Need to be intubated if ability to preserve airway is impaired
- B. **Respiratory arrest** due to ascending cervical lesion: monitor patient's speech and ability to ventilate. Never leave patient alone. Keep a BVM by patient if C5 lesion or above is suspected.
- C. **Respiratory insufficiency:** Cervical lesions preserve diaphragm but lose intercostal muscles, which impair cough and deep breathing.
- D. **Tension pneumothorax:** From barotrauma of overventilating the patient.
- E. **Deep vein thrombosis; pulmonary embolus** from immobility
- F. **Pneumonia:** Due to immobility and poor respiratory muscle control with loss of coughing and respiratory protective reflexes.
- G. **Atelectasis:** Encourage deep breathing and assisted coughing. Splint abdomen with pillows.
- H. ARDS if allowed to remain in neurogenic shock
- I. **Autonomic dysreflexia (AD)**
 1. This is a potentially life-threatening condition most commonly associated with injuries above T4-6 as spinal shock resolves. Usually a late complication of upper motor neuron dysfunction, but can present acutely. The patient has experienced a disconnection between the body below the injury and the control mechanisms for BP and heart function. It does not occur in those with lower motor neuron dysfunction or spinal cord infarcts that destroy preganglionic sympathetic fibers.
 2. **Pathophysiology**
 - a. The patient experiences a massive, uninhibited, uncompensated cardiovascular response due to irritation, pain or some stimulation of the sympathetic NS below the level of injury. The irritated area sends a signal upward, but it is not able to reach the brain. An uncontrolled continuous lower motor neuron reflex action takes place.
 - b. The stimulus causes massive uninhibited activation of the sympathetic nervous system resulting in vasoconstriction. This is evidenced by cool, pale extremities, systolic BPs greater than 200 mmHg and diastolic BPs of 130 mmHg or above.
 - c. Hypertension results in a pounding headache and carotid sinus/aortic arch stimulation. Cerebral, carotid and aorta baroreceptors sense the hypertension and trigger the vasomotor center in the medulla to activate the parasympathetic nervous system.
 - d. Vagal compensation causes bradycardia and vasodilation of peripheral and viscera vessels **above** the level of the lesion, but those below remain constricted. Selective vasodilation results in flushed, diaphoretic skin and nasopharyngeal and neck vessel congestion

3. **Precipitating factors:** Over-distention of bladder due to obstructed urine outflow from spasm or kinked urinary catheter; bladder infection; severe constipation/fecal impaction or excess gas; or other noxious stimuli like surgical procedures, i.e., line insertions; skin stimulation from extreme heat or cold, pressure areas, decubitus ulcers, ingrown toenail or pain/pressure from being left too long in one position; or pregnancy.
4. **Signs and symptoms**
 - a. Sudden severe hypertension (SBP can go up to 300 mmHg)
 - b. Headache, generally pounding
 - c. Nasal congestion
 - d. Pupillary dilation; spots before the eyes or blurred vision
 - e. Anxiety, apprehension, feeling of impending doom
 - f. Bradycardia (usually)
 - g. Rebound hypotension
 - h. Flushing and sweating **above** the level of injury
 - i. Pilomotor erection; "goose bumps" **above** the lesion
 - j. Chills without fever
 - k. Bronchospasm
 - l. Seizures, stroke and death if not treated
5. **Immediate interventions:** (UAB, 2008)
 - a. Sit patient up and take BP in each arm. Repeat BP q. 3 minutes and between steps. Normal SBP for a person with a SCI above T6 can be in the 90-110 mmHg range. If the BP is elevated over 150 mmHg, treat until a cause is found and eliminated.
 - b. Monitor VS q. 5 minutes until stable
 - c. **Look for noxious stimuli below level of injury**
 - (1) **Check bladder for distention:** If indwelling urinary catheter is already in place, inspect for kinks, folds, constrictions or obstructions. Irrigate or replace the catheter if allowed by local protocols to insure patency.
 - (2) **Check skin:** Remove constrictive clothing. Examine for pressure ulcers. Reposition patient. Examine seat cushion and wheelchair for sharp or hard objects. Remove sharp objects from pockets or on seat cushion. Examine for insect bites. Reduce irritation from cuts, bites or burns with cold packs. Evaluate environmental temperature.
 - (3) Do symptoms change as environmental and patient's temperature change? Evaluate recent surgical sites. Observe for ingrown toenails.
 - (4) **Evaluate for gastrocolic irritation:** Was a tube feeding given recently? Too rapid? Too cold? Too large a volume?
 - d. **Gender specific**
 - (1) **Males:** Genitalia pinched? Condom catheter too tight? Reflexogenic erection? Remove condom catheter and clothing.
 - (1) **Females:** Menstrual cramping?
 - d. Reduce BP with vasodilators only if noxious stimulus cannot be removed or removal was ineffective in aborting symptoms
- I. **Psych sequelae**
 1. Denial: Many believe surgery will cure them
 2. Anger
 3. Depression
 4. Coping

XII. Non-traumatic spinal conditions

A. Low back pain

1. Affected area
 - a. Between lower rib cage and gluteal muscles
 - b. May radiate to thighs
2. 1% of acute low back pain is sciatica
 - a. Usual cause is in the lumbar nerve root
 - b. Pain accompanied by motor and sensory deficits, e.g., weakness
3. 60% - 90% of population experience some form of low back pain
 - a. Affects men and women equally
 - b. Women over 60 years old report low back pain symptoms more often
4. Most cases of LBP are idiopathic; precise diagnosis difficult
5. Causes
 - a. Tension from tumors
 - b. Disk prolapsed
 - c. Bursitis
 - d. Synovitis
 - e. Rising venous pressure
 - f. Tissue pressure due to degenerative joint disease
 - g. Abnormal bone pressure
 - h. Problems with spinal mobility
 - i. Inflammation caused by infection; osteomyelitis
 - j. Fractures
 - k. Ligament strains
6. Risk factors
 - a. Occupations requiring repetitious lifting
 - b. Exposure to vibrations from vehicles or industrial machinery
 - c. Osteoporosis
7. Anatomical considerations
 - a. Pain from innervated structures -Varies from person-to-person
 - b. Disk has no specific innervation; compresses cord if herniated
 - c. Source of pain in L-3,4,5, and S-1 may be interspinous bursae
 - d. Anterior and posterior longitudinal ligaments, and other ligaments are richly supplied with pain receptors
 - e. Muscles of spine vulnerable to sprains/strains

B. Degenerative disc disease

1. Common for patients over 50 years of age
2. Causes
 - a. Degeneration of disc
 - b. Biomechanical alterations of disc
 - c. Narrowing of the disk
3. Results in variable segment stability

C. Spondylolysis

1. Structural defect of spine involves the lamina or vertebral arch
2. Usually occurs between superior and inferior articulating facets
3. Heredity a significant factor
4. Rotational fractures common at affected site

D. Herniated intervertebral disc

1. Also called herniated nucleus pulposus
2. Tear in the posterior rim of capsule enclosing the gelatinous center of the disc
3. Causes
 - a. Trauma
 - b. Degenerative disk disease
 - c. Improper lifting -Most common cause
4. Men ages 30 - 50 years are more prone than women
5. Commonly affects L-5, S-1 and L-4, L-5 discs
6. May also occur in C-5, C-6, and C-7
7. S&S
 - a. Pain usually occurs with straining: Coughing or sneezing
 - b. May have limited range of motion in lumbar spine
 - c. Tenderness upon palpation
 - d. Alternations in sensation, pain, and temperature
 - e. Due to nerve root pressure
 - f. Cervical herniations may include
 - (1) Upper extremity pain or paresthesia; Increasing with neck motion
 - (2) Slight motor weakness may also occur in biceps and triceps

E. Spinal cord tumors

1. Causes
 - a. Compression of the cord
 - b. Degenerative changes in the bone/joints
 - c. Interrupted the blood supply
2. Manifestations are dependent upon
 - a. Tumor type
 - b. Location

F. Management of non-traumatic spine pain

1. Primarily palliative to decrease any pain or discomfort from movement.
2. Seek OLMC order for muscle relaxant
3. May elect to immobilize to aid in comfort; full spine motion restriction is not required unless condition is a result of trauma

XIII. Summary of key points

- A. The keys to appropriate management of patients with SCI lie in attending to life-threatening injuries, avoiding unnecessary movement of the spinal column, carefully documenting patient reliability, MOI, patient history and physical exam findings, interventions, and responses to interventions. Initial assessments are used as a baseline upon which changes in the patient's neurological status are compared.
- B. Historically, destroyed neurons have been permanently lost with no restoration of function. Exciting research evaluating the efficacy of hypothermia, stem cells, gangliosides, opiate receptor antagonists, NMDA receptor antagonists, calcium antagonists, thyrotropin releasing hormone, and intercostal nerve transplants supports a cautiously optimistic future for spine regeneration.
- C. Despite the marvels of modern research, prevention is still the key to reducing the number of SCI, including public education relative to wearing seat belts, helmets, enforcing DUI laws, instructing parents in the use of child restraint devices, training children to jump rather than to dive when testing the depth of water and outlawing such practices as spear tackling in high school football.

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Glossary

Apoptosis	Programmed cell death or “cell suicide”; a form of cell death in which a controlled sequence of events (or program) leads to the elimination of cells without releasing harmful substances into the surrounding area. Many types of cell damage can trigger apoptosis, and it also occurs normally during development of the nervous system and other parts of the body. Technically, the term apoptosis refers only to the structural changes that cells go through, and programmed cell death refers to the complete underlying process, but the terms are often used interchangeably.
Ascending pathways	Nerve pathways that go upward from the spinal cord toward the brain and carry sensory information from the body.
Astrocytes (meaning “star cells” due to their shape)	The largest and most numerous supporting, or glial, cells in the brain and spinal cord. They contribute to the blood-brain barrier, help regulate the chemical environment around cells respond to injury, and release regulatory substances that influence nerve cells.
Autonomic dysreflexia	A potentially life-threatening increase in BP, sweating, and other autonomic reflexes in reaction to bowel impaction or some other stimulus.
Axon	Long nerve fibers that conduct electrical impulses away from nerve cell bodies. They contact other nerve, muscle, and gland cells at synapses and release neurotransmitters that influence those cells.
Blood-brain barrier/ blood-spinal-cord barrier	Barriers, largely formed by endothelial cells that line blood vessels, that restrict the entry of circulating substances and immune cells into the brain and spinal cord. Trauma may compromise these barriers and contribute to further damage in the brain and cord. These barriers also prevent entry of some potentially therapeutic drugs.
Central cord syndrome	Affects the cervical region of the cord and results from focused damage to the corticospinal tracts. Patients often spontaneously and rapidly recover a great amount of function within days or weeks after injury.
Complete injury	Used to describe an absence of sensory and motor function in the lowest sacral segment.
Corticospinal tracts	Nerve fibers that carry signals from the motor control areas of the brain's cerebral cortex (frontal lobe) down the spinal cord.
Dendrites	Tree-like branches emerging from nerve cell bodies that receive signals from other nerve cells at synapses.
Dermatome	Refers to the area of the skin innervated by the sensory axons within each segmental nerve (root). There are 28 dermatomes on each side of the body
Descending pathways	Nerve pathways that go down the spinal cord and allow the brain to control movement of the body below the head.
Dorsal	Refers to the back of an organism, like the dorsal fin of a fish.
Free radicals	Highly reactive chemicals that attack molecules crucial for cell function by capturing electrons and modifying chemical structures.
Glia	Supporting cells of the nervous system. They far outnumber nerve cells. They provide physical support, respond to injury, regulate the chemical composition surrounding cells, participate in the blood-brain/blood-spinal cord barriers, form the myelin insulating nerve pathways, help guide neuronal migration during development, and exchange metabolites with neurons. They may also produce substances that help and hinder regeneration in the spinal cord. Major types: astrocytes, oligodendrocytes, and microglia.
Gray matter	Parts of the brain and cord composed mainly of cell bodies and dendrites. The gray matter of the spinal cord lies in a butterfly-shaped region in the center of the cord while the gray matter of the brain forms the cortex (bark) around the outside of the brain.

Incomplete injury	Partial preservation of sensory and/or motor function found below the neurological level, including the lowest sacral segment. Sacral sensation includes sensation at the anal mucocutaneous junction as well as deep anal sensation. The test of motor function is the presence of voluntary contraction of the external anal sphincter upon digital examination.
Interneurons	Neurons confined wholly within the spinal cord, as compared to sensory and motor neurons whose axons project outside of the cord. Interneurons help integrate sensory information and generate coordinated muscle commands.
Myelin	Electrically insulating coating around axons that gives white matter its whitish appearance. Myelin increases the speed and reliability of signal transmission along nerve fibers. In the CNS, oligodendrocytes create the myelin. In the PSN, Schwann cells generate myelin.
Myotome	Refers to the collection of muscle fibers innervated by the motor axons within each segmental nerve (root). There are 10 myotomes on each side of the body.
Necrosis	A type of cell death in which the cells swell and break open, release their contents and can damage neighboring cells and provoke inflammation.
Neurological level, sensory level, and motor level	The first of these terms refers to the most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body. In fact, the segments at which normal function is found often differ by side of the body and in terms of sensory vs. motor testing. Up to four different segments may be identified in determining the neurological level: R-sensory; L-sensory; R-motor; L-motor. It is strongly recommended to separately record each segment rather than a single level as this can be misleading. When the term sensory level is used, it refers to the most caudal segment of the spinal cord with normal sensory function on both sides of the body. The motor level is similarly defined with respect to motor function.
Neurotransmitter	Chemicals released by nerve cells at synapses that influence the activity of other cells. They may excite, inhibit, or otherwise influence the activity of cells.
Oligodendrocytes	A type of glial cell that produces myelin around axons in the CNS, but also produces substances that inhibit the regeneration of axons in the adult CNS.
Paraplegia	Refers to impairment or loss of motor and/or sensory function in the thoracic, lumbar or sacral (but not cervical) segments of the spinal cord, secondary to damage of neural elements within the spinal canal. With paraplegia, arm functioning is spared, but, depending on the level of injury, the trunk, legs and pelvic organs may be involved. The term is used in referring to cauda equina and conus medullaris injuries but not to lumbosacral plexus lesions or injury to peripheral nerves outside the neural canal.
Plasticity	The ability of the nervous system to change with experience.
Receptors	Molecules, usually found on the surfaces of cells, that enable cells to respond to neurotransmitters, hormones, and other messenger molecules. They may act directly by opening ion channels in the cell membrane that are part of the same receptor molecule, or indirectly by activating second messenger systems that go on to affect various processes in the cell. The term receptor also refers to cells or structures that receive sensory information, such as pain receptors and light receptors in the eye.
Schwann cells	Glial cells in the PNS that wrap nerve fibers with myelin and also secrete regulatory factors.
Skeletal level	Refers to the level at which, by radiographic examination, the greatest vertebral damage is found.
Spinal cord segments	Divisions of the spinal cord along its length. Each spinal segment sends a pair of motor and sensory nerves to the body. Higher segments control movement and sensation in the upper parts of the body, while lower segments control lower parts of the body.
Spasticity	A state of increased muscular tone in which abnormal stretch reflexes intensify muscle resistance to passive movements.

Synapse	The functional connection between a nerve cell axon and target cells, which may be other nerve cells, muscle cells, or gland cells. At the synapse, the axon releases a chemical neurotransmitter that diffuses across a tiny gap and binds to receptors that then change the target cell's behavior. Synapses may be excitatory (increasing a target cell's electrical activity), inhibitory (reducing a target cell's activity), or have more complex influences (such as adjusting the sensitivity of cells to other signals).
Tetraplegia (preferred to quadriplegia)	Refers to impairment or loss of motor and/or sensory function in the cervical segments of the spinal cord due to damage of neural elements within the spinal canal. Tetraplegia results in impairment of function in the arms as well as in the trunk, legs, and pelvic organs. it does not include brachial plexus lesions or injury to peripheral nerves outside the neural canal.
Ventral	Toward the front of the body
White matter	Areas of the brain and spinal cord that contain mainly nerve fibers rather than nerve cell bodies and dendrites (gray matter). The myelin insulating covering of axons gives the whitish appearance. White matter in the spinal cord is located in the outer portion, while it is on the inner portions of the brain.
Zone of partial preservation (ZPP)	Refers to those dermatomes and myotomes caudal to the neurological level that remain partially innervated. When some impaired sensory and/or motor function is found below the lowest normal segment, the exact number of segments so affected should be recorded for both sides as the ZPP. Only used with complete injuries.

NINDS, 2007

Study Questions

1. List the five main anatomical divisions of the vertebral column from top to bottom including the number of bones and nerve roots for each division.

Division	# Bones	# Nerve roots

2. Name the distal portion of the spinal cord that is composed of nerve roots that look like a horse's tail.

3. Blood supply to the anterior 2/3 of the cord is provided by the _____ artery.
Blood supply to the posterior 1/3 of the cord is provided by two _____ arteries.

4. The sympathetic nervous system fibers exit from the _____ and _____ areas of the spinal cord.

5. **True or false:** The vagus nerves exit from the cervical area of the cord to innervate the heart.

6. Which injury is described as a spontaneously reversible physiologic disruption of the cord with transient deficits for 24-28 hours but no identifiable neuropathologic changes noted on exam?

Which injury results in bruising of the cord caused by fractures, dislocations, or direct trauma?

7. Explain the anticipated changes to the vital signs **and their cause** if a patient presents in neurogenic shock:

VS changes	Cause
BP:	
P:	
Temp:	

8. Which findings are necessary for a patient to be considered reliable?

9. Give two examples of distracting injuries

10. Under what circumstances may a SCI patient need to be sedated and restrained?

11. Why should a paramedic anticipate impairment of the airway if a patient presents with a C2 fracture without neurological deficit?

12. If a SCI patient is awake with an impaired airway or has a deteriorating GCS, what airway access procedure should be attempted?

13. Lesions above C3-4 may lead to _____ and the need for mechanical ventilation.

14. Describe the type of ventilations that would probably be exhibited by a patient with a lesion at the level of C6.

Why is this patient at risk for respiratory arrest during the first hour after injury?

15. What is the first drug of choice to treat the abnormal pulse rate associated with neurogenic shock?

16. What drug and dose might be used as a vasopressor to support perfusion and BP if the heart rate is normal and fluid challenges fail to correct the BP abnormality in neurogenic shock?

17. What is the potential benefit to be gained by inducing hypothermia in a patient with an acute SCI?

18. Why should a patient with paralysis of the legs be evaluated for an aortic tear?

19. Why is the clinical abdominal exam an unreliable indicator of possible intraperitoneal bleeding in an acutely injured patient with tetraplegia?

20. When assessing the vertebrae, what findings are significant for SCI on inspection and palpation?

21. Why is there an absence of sweating below the level of a cervical lesion?

22. Describe the motor exam maneuvers a patient should be asked to perform to assess the three major myotomes in the upper extremities:
- Elbow: _____
- Wrist: _____
- Hand: _____
23. Describe the maneuvers a patient should be asked to perform to assess the lumbar and sacral motor nerve segments in the lower extremities:
- _____
- _____
24. How should a paramedic assess sensory integrity in a patient with suspected SCI?
25. If a patient with suspected SCI presents with paralysis of the legs, full motor and sensory sparing of the hands and arms and a sensory deficit at the nipple line, what is the probable level of injury?
- | | | | |
|----|-----|----|------|
| A. | C-7 | C. | T-10 |
| B. | T-4 | D. | C-4 |
26. How should a patient with SCI be assessed for sacral sparing?
27. How should a patient with SCI be assessed for deficits in proprioception?
28. List at least five physical exam findings that suggest spinal cord injury. (Think Ps)
- _____
- _____
- _____
- _____
- _____
29. Tetraplegia is caused by a neurologic level of injury that involves nerve roots _____ or above.
30. List the three factors upon which the decision to provide spine motion restriction depends:
-
-
-
31. Full spine motion restriction is indicated if the following is present:
-
-
-
32. No spine motion restriction is indicated if the following conditions are met:

33. An ambulatory adult presents c/o pain after a heavy piece of pipe fell on the back of his neck. He has point tenderness to palpation at C-7, full motor strength in the hands and a burning sensation in his thumbs and index fingers. How should spine motion restriction be accomplished?
34. What is the critical dimension when measuring a patient for a cervical collar?
35. Why is it so important to pad bony prominences in a patient who is placed on a long spine board?
36. Under what circumstances may a patient be removed from a vehicle using rapid extrication?
37. Under what circumstances should a patient be removed from a vehicle using the KED?
38. Should helmets be removed or left in place prior to radiologic clearance of the c-spine if the airway can be secured and the helmet fits securely?
40. Which of these is present in a patient with Anterior Cord Syndrome?
Paralysis / sparing of motor function
Loss / sparing of the sensory function of pain / temperature
Loss / sparing of the sensory function of light touch and position sense
- Which of these is present in a patient with Brown Sequard Syndrome?
Ipsilateral / contralateral motor deficits
Ipsilateral / contralateral sensory deficits
41. What causes autonomic dysreflexia?
42. List three of the presenting S&S of autonomic dysreflexia.
-
-
-
43. List two interventions for autonomic dysreflexia.
-
-