Published November 1, 2010

Cervical Spine Injuries: Beyond Whiplash

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Cervical spine injuries (CSI) are an uncommon but important consequence of blunt trauma. Between 0.9 and 3% of blunt trauma victims sustain some type of CSI, resulting in 12,000 new cases of spinal cord injury (SCI) in the United States every year. [i] The lifetime burden of SCI is tremendous, and the legal ramifications of missed SCI are considerable (Table 1).

- 231,000-311,000 persons living with SCI
- 12,000 new cases per year
- **41% motor vehicle collisions**, 27% falls, 15% violence, 8% sports, rest are other/ unknown
- Most common sports with SCI: Football, diving, gymnastics, skiing, wrestling, rugby, surfing, and equestrian events
- Average age of SCI in 1970: **28.7 years**
- Average age of SCI in 2005: **40 years**
- 81% **male** patients
- 66.2% Caucasian, 27% African-American, 8% Hispanic, 2% Asian
- Type of neurologic injury:
  - ➡ 38.3% Incomplete Tetraplegia
  - ➡ 22.9% Complete Paraplegia
  - ➡ 21.5% Incomplete Paraplegia
  - ➡ 16.9% Complete Tetraplegia
- Lifetime health care costs (*Estimates do not include indirect costs like loss in wages, benefits, and productivity, which average $65,384 per year in 2009 dollars*):
  - ➡ 50-year-old paraplegic: $745,951
  - ➡ 25-year-old high tetraplegic (C1-4): $3,273,270
- Most common cause of death for SCI patients in 1970: **Renal failure**
- Most common cause of death for SCI patients in 2009: **Pneumonia**, septicemia, and pulmonary embolism

Table 1. Summary of SCI Facts and Figures (United States) [1]

This article provides an analysis of cervical spine injuries, focusing on the fundamentals as well as the standard practices and mythology related to pre-hospital management, Emergency Department (ED) evaluation, and pharmacotherapy. The following will be addressed:

- Cervical anatomy
- Unstable cervical fractures
- Incomplete spinal cord syndromes
- The debate over cervical collars
- Spinal versus neurologic shock
- Blood pressure management
- Steroid administration in SCI
- NEXUS and Canadian C-spine Rules
- Imaging of the Cervical Spine
- Airway issues

As you progress through this article, consider the following nightmare (actual) case:

A 45 year-old male (unrestrained driver) arrives in the ED following a single vehicle MVC. EMS reports marked damage to the car. He received 1 mg of lorazepam en route due to his agitation. On arrival, the patient is somewhat intoxicated, but speaking (indeed yelling) clearly, emphasizing his desire to have the straps/collar removed and to sit up. His initial vital signs are normal except for a heart rate of 112. His secondary exam demonstrates a 4 cm laceration to his left eyebrow with surrounding contusion. He also complains of tingling in his arms and legs, but he has normal strength. He has mild midline neck pain.

He receives a head CT, plain radiography (3 views) of his cervical spine, chest x-ray, and pelvis x-ray. All of these studies are normal, though the cervical x-rays are of poor quality.

Upon re-examination of the patient, he now has slightly decreased strength in his legs, more so than his arms. While still supine, he begins to vomit, is noted to aspirate, and seizes. Bag mask ventilation is initiated, followed by intubation using direct laryngoscopy. In order to perform this, the cervical collar is removed, an assistant maintains in-line stabilization, and the physician performing the intubation uses a gentle head tilt/chin lift maneuver. The endotracheal tube is placed easily.

His blood pressure drops to 70/40 mm Hg after the intubation, and the physician immediately orders a normal saline bolus, which, after 1 liter, brings the blood pressure back to 110/70 mm Hg. Heart rate is 100 bpm. A FAST exam is negative. The patient is sent back to CT in order to obtain views of the cervical/thoracic/lumbar spine, chest, abdomen, and pelvis. The patient is noted to have a bilateral facet dislocation of C6-C7.

During his hospital course, the weakness progresses and the patient remains permanently paraplegic. A Morbidity and Mortality conference a few weeks later criticizes the Emergency Physician for:

- Modality of cervical spine imaging
- Positioning maneuver for intubation (head tilt/chin lift)
- Choice of direct laryngoscopy for intubation
- Failing to initiate steroids

Remind me: how many cervical vertebrae are there?

Cervical spine anatomy is tricky. The spinal cord is approximately 42 to 45cm in length, ranging from the superior border of the atlas to the second lumbar vertebra. [ii] Both the C1 and C2 vertebrae have distinct anatomy. C1, or the "atlas," has a ring-like structure, with no body. It lives up to its mythological namesake (the "Atlas" of Greek mythology who supported the heavens), as it collaborates with C2 (the "axis") to connect skull to spine, allowing flexion, extension, and rotatory movements of the head. C2 possesses the peculiar odontoid process ("dens"), which arises from the vertebral body. C2, including the dens, is the most commonly fractured cervical vertebra (Table 2).

- NEXUS project involving 34,069 patients with blunt trauma
- Substudy of 818 (2.4% of all patients) with 1,496 distinct CSI to 1,285 different C-spine structures
- Top 3 fracture levels:
  1. **C2** (24%; 16% non-odontoid, 8% odontoid)
  2. C6 (20%)
  3. C7 (19%)
- Top 3 dislocation/subluxation levels:
  1. **C5-C6** (25%)
  2. C6-C7 (23%)
  3. C4-C5 (16%)
- Top 3 anatomic location of fractures in C3-7:
  1. **Body**
  2. Spinous process
  3. Lamina
- Percentage of all CSI considered clinically insignificant: **29%**

Table 2. Statistics on Blunt Traumatic Cervical Spine Injury [3]

Below C2, consider the anatomy in terms of two columns, the anterior and posterior columns, which are commonly referenced by spine surgeons. Table 3 lists the anatomic components of each, while Figure 1 illustrates the column anatomy.

Anterior Column	Posterior Column
Vertebral bodies	Spinal canal
Intervertebral disks	Pedicles
Anterior longitudinal ligament	Transverse processes
Posterior longitudinal ligament	Articulating facets
	Laminae
	Spinous processes
	Nuchal ligament complex (supraspinous, interspinous, and infraspinous), capsular ligaments, and ligamentum flavum)

Table 3. Anterior and posterior columns of the cervical spine.

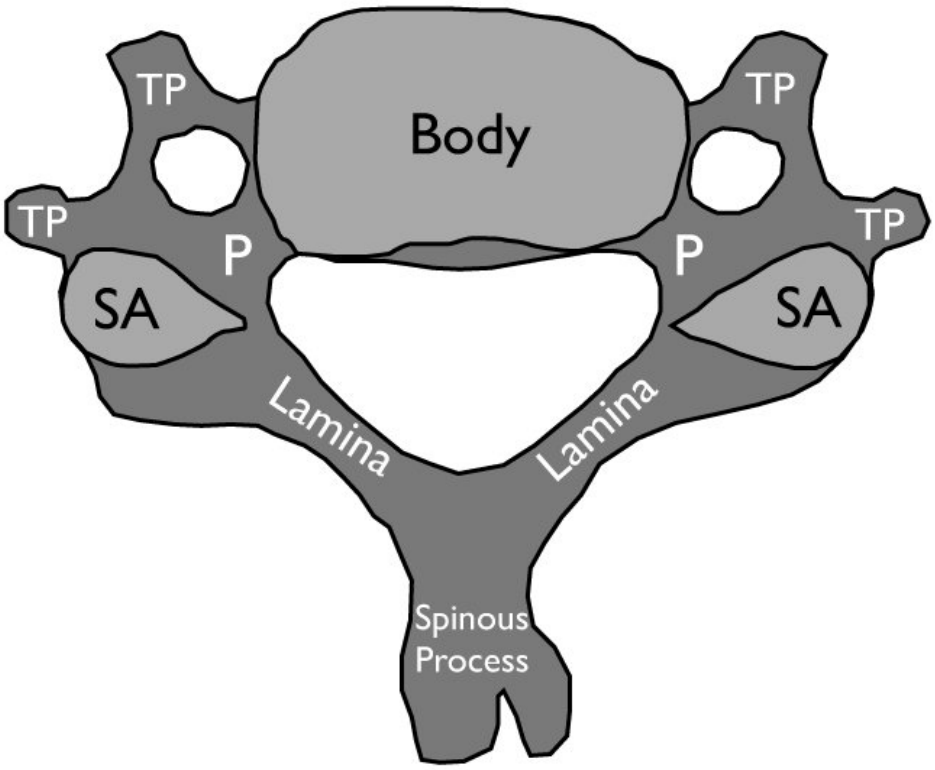


Figure 1. Anatomy of the cervical vertebra. TP=transverse process. SA=superior articular process. P=pedicle.

The anterior column stabilizes the spine in extension, and the posterior column coordinates flexion. Therefore, extension injuries commonly lead to anterior column disruption, and flexion injuries typically result in posterior column pathology.

Is the fracture unstable?

Stability is one of the most important determinations in the initial evaluation of cervical spine injuries. Disposition is based in large part on the answer to the question: *Is this cervical spine injury stable or unstable?*

Stable fractures occur more often. The most common fractures from a substudy of the National Emergency X-Radiography Utilization Study (NEXUS), which enrolled over 34,000 patients, are presented in Table 2. Table 4 lists both stable and unstable cervical spine injuries.

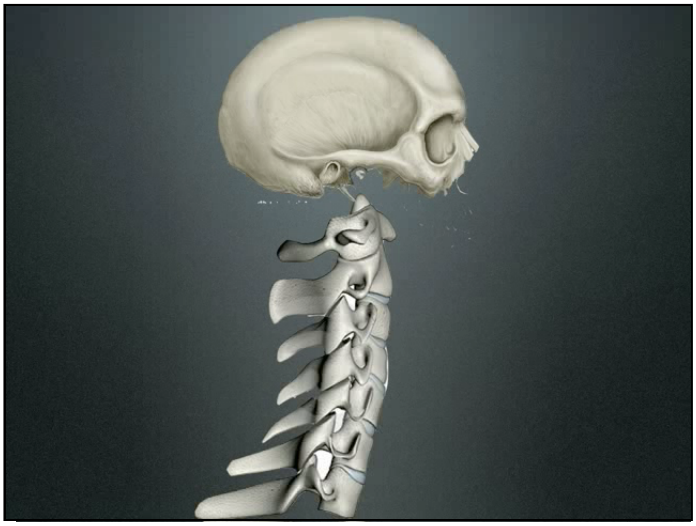
Stable Cervical Spine Injury	Unstable Cervical Spine Injury
Spinous process fracture	Atlanto-occipital joint dislocation
Wedge compression fracture (≤25% loss of height)	Atlanto-axial joint dislocation
Isolated avulsion fracture (no ligamentous injury)	Flexion or extension teardrop fracture
Type 1 odontoid fracture	Bilateral facet dislocation
End-plate fracture	Odontoid fracture, with lateral displacement fracture
Simple osteophyte fracture	Posterior neural arch fracture of C1
Transverse process fracture	Hangman's fracture (C2)
Unilateral facet dislocation	Jefferson fracture (C1)
Clay shoveler's fracture	Pure spinal subluxation

Table 4. Common stable and unstable cervical spine injuries.

Key summary points: C2 was the most commonly injured, dislocation and subluxation injuries typically occurred in the lower cervical spine, and almost a third of cervical fractures were deemed clinically insignificant. [iii]

The following are a few of the more frequently described, *unstable* cervical injuries [iv, v]:

Atlanto-occipital dissociation/dislocation (AOD). AOD is typically caused by a hyperextension injury with distraction. Injury occurs to the ligaments between the occipital condyles and the atlas (C1). Simply put, the spine and the skull move independently, resulting in significant (and potentially repetitive) cord injury (Click on image to play video).



AOD is thought to occur in up to 2-3% of all cervical spine fractures. [iii, v] Nearly 20% of patients with acute traumatic AOD will present with a normal neurological exam. It occurs more commonly in children, and is usually fatal due to respiratory arrest. Plain films are only around 60% sensitive for this finding. CT is the imaging modality of choice. On lateral films, the most striking abnormality is an increased distance from the occiput to the atlas, which typically should not exceed 5mm. Moreover, the presence of soft tissue swelling on a lateral radiograph should be a red flag. *Figures 2a and 2b*

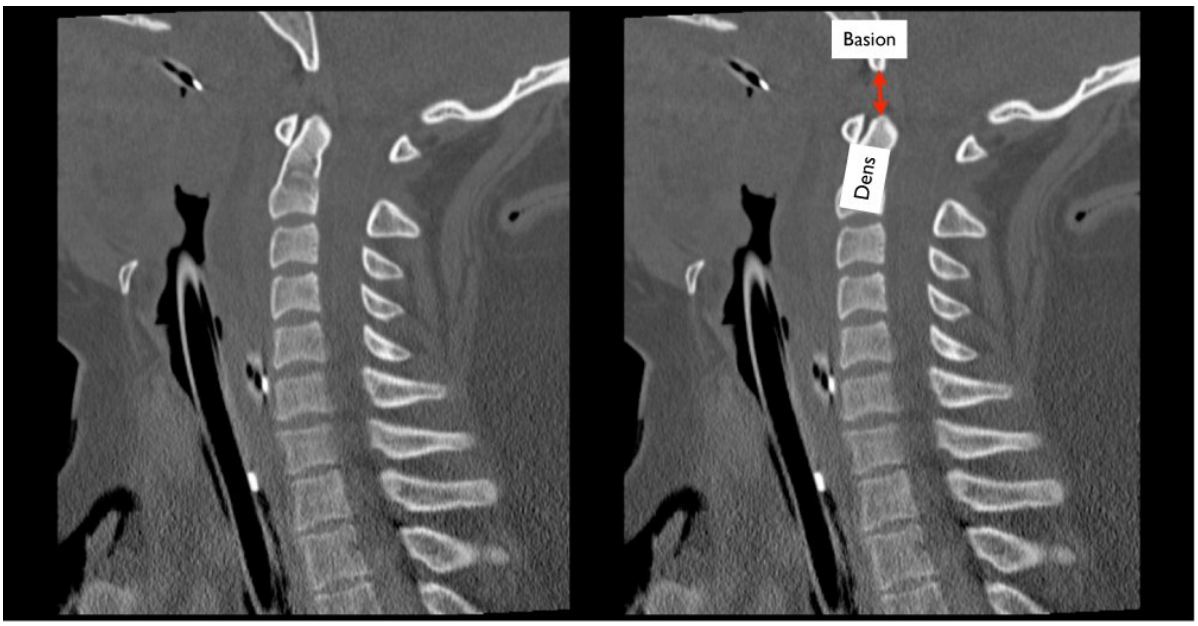


Figure 2a. *Atlanto-occipital dislocation.* Sagittal reformation image, demonstrating widening (red arrow) between the dens and the basion of the skull. (Courtesy of C.E. Smith, MD, Vanderbilt University)

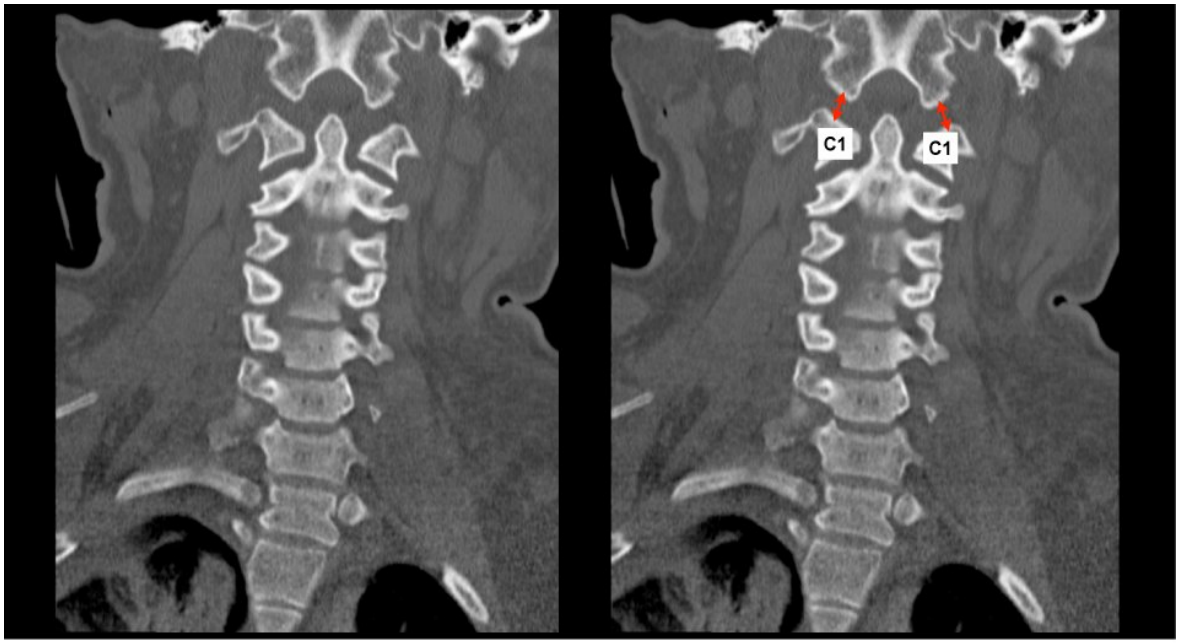


Figure 2b. *Atlanto-occipital dislocation*. Coronal reformation image, demonstrating widening (red arrows) between C1 and the base of the skull. (Courtesy of C.E. Smith, MD, Vanderbilt University)

Bilateral facet dislocation. An extreme *flexion* injury resulting in a high degree of anterior subluxation. In this injury, the inferior articulating facets of the upper vertebra pass upward and over the superior facets of the lower vertebra. The annulus fibrosus, anterior longitudinal ligament, and posterior ligamentous complex are all disrupted. The result is anterior displacement of the vertebral body by more than ½ of the diameter (on lateral projection), while the superior facets lie anterior to the inferior facets. This is a very unstable injury. *Figure 3*.

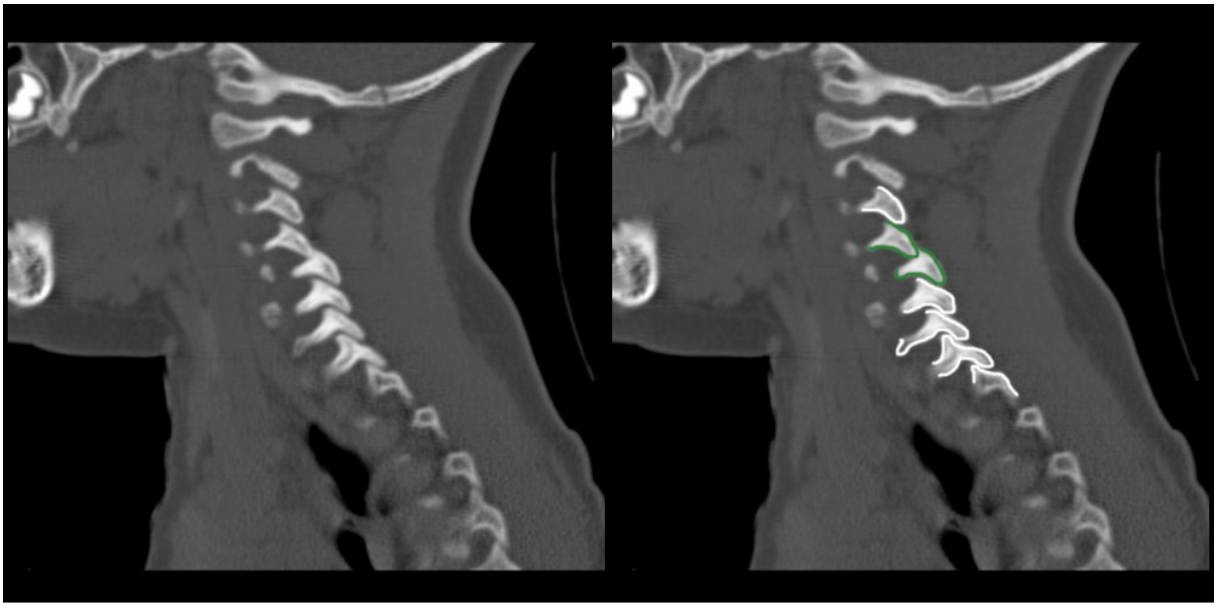


Figure 3. *Bilateral facet dislocation.* Sagittal reformation image, showing a "jumped" facet, or facet dislocation, as indicated by the green outline of the affected segments. Note the contrast to the normal anatomy of the cervical vertebrae outlined in white. (Courtesy of C.E. Smith, MD, Vanderbilt University)

Hangman's fracture (or traumatic spondylolysis of C2). A *hyperextension* injury resulting in bilateral fractures of the pedicles of C2. Typically a result of a sudden deceleration, with the skull, atlas, and axis violently hyperextended. Good news: the spinal canal is quite wide at this level. Bad news: the lesion is still unstable. *Figures 4a and 4b*.

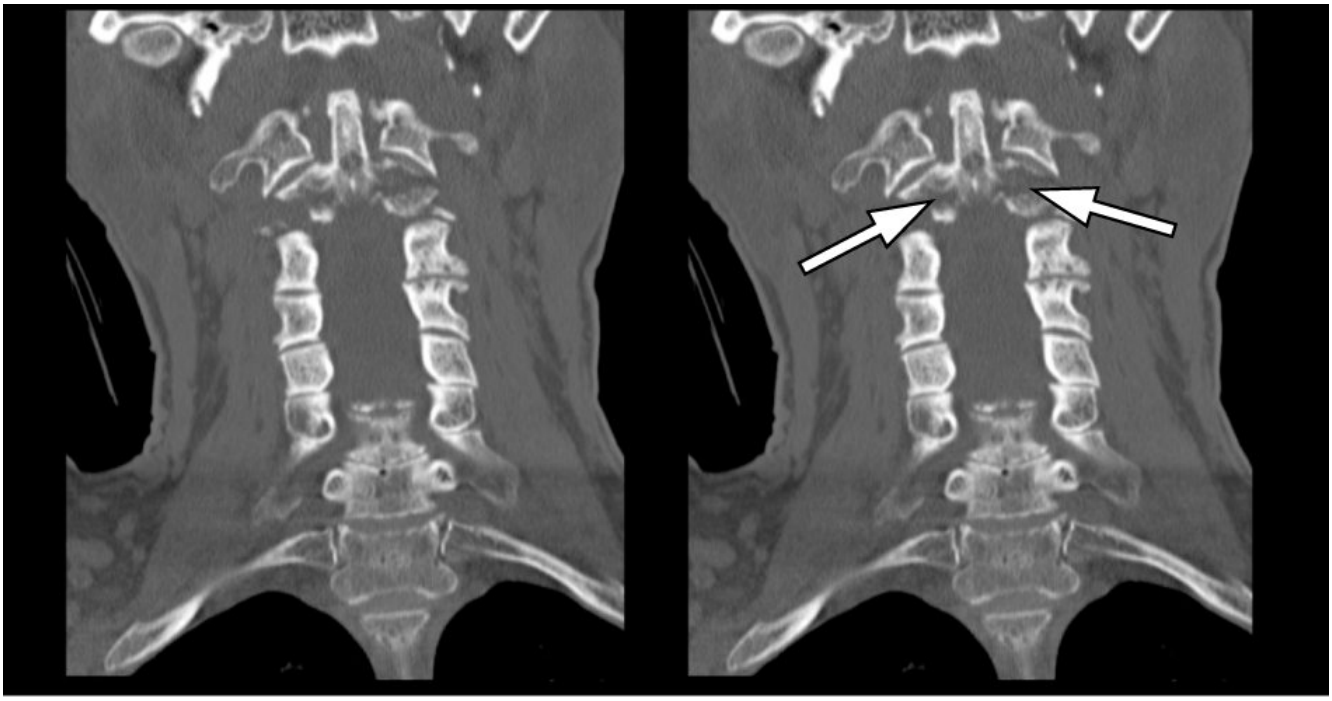


Figure 4a. *Hangman's fracture.* Coronal reformation image, with fractures to the body, lateral masses, and pedicles of C2 (white arrows). (Courtesy of C.E. Smith, MD, Vanderbilt University)

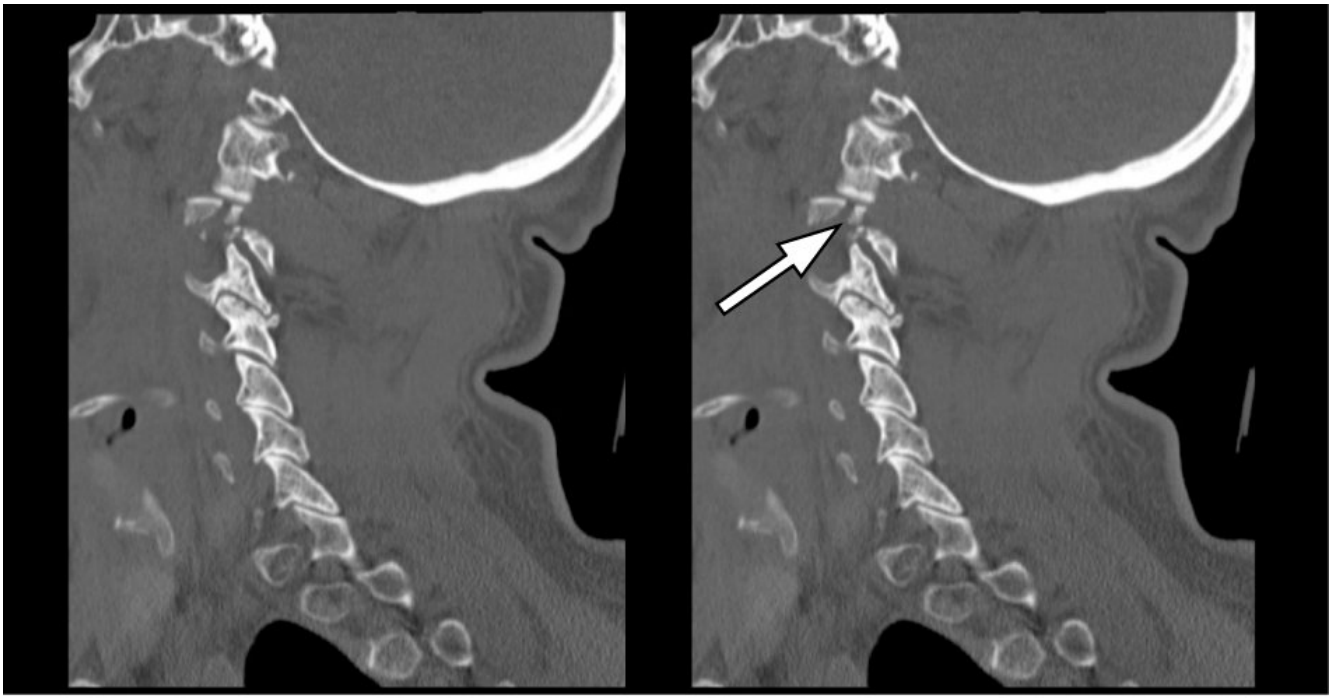


Figure 4b. *Hangman's fracture*. Sagittal reformation image, with arrow indicating C2 body and pedicle fractures (white arrow). (Courtesy of C.E. Smith, MD, Vanderbilt University)

Jefferson fracture (burst fracture of the ring of C1). C1 fractures account for around 9% of cervical spine fractures, though they are only rarely associated with neurologic deficits. They are accompanied by other cervical fractures in around one-half of cases. [vi, vii] The Jefferson fracture is an example of a *vertical compression* injury to C1, with fractures of the anterior and posterior arches of the atlas and a disruption of the transverse ligaments. The force from the occipital condyles pushes the lateral masses outward. Very unstable. *Figures 5a and 5b.*

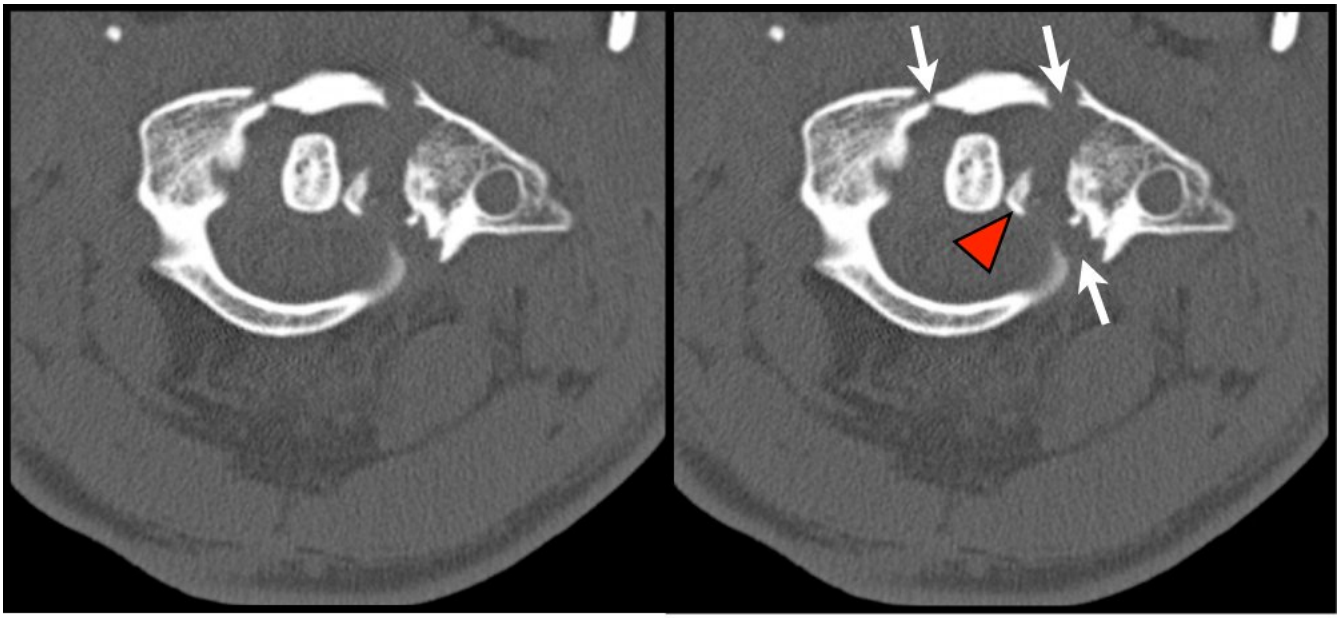


Figure 5a. *Jefferson fracture*. Axial reformation image, notable for multiple fractures of the ring of C1, indicated by the white arrows. Red arrowhead shows a probable avulsion fracture from injury to the transverse ligament. (Courtesy of C.E. Smith, MD, Vanderbilt University)

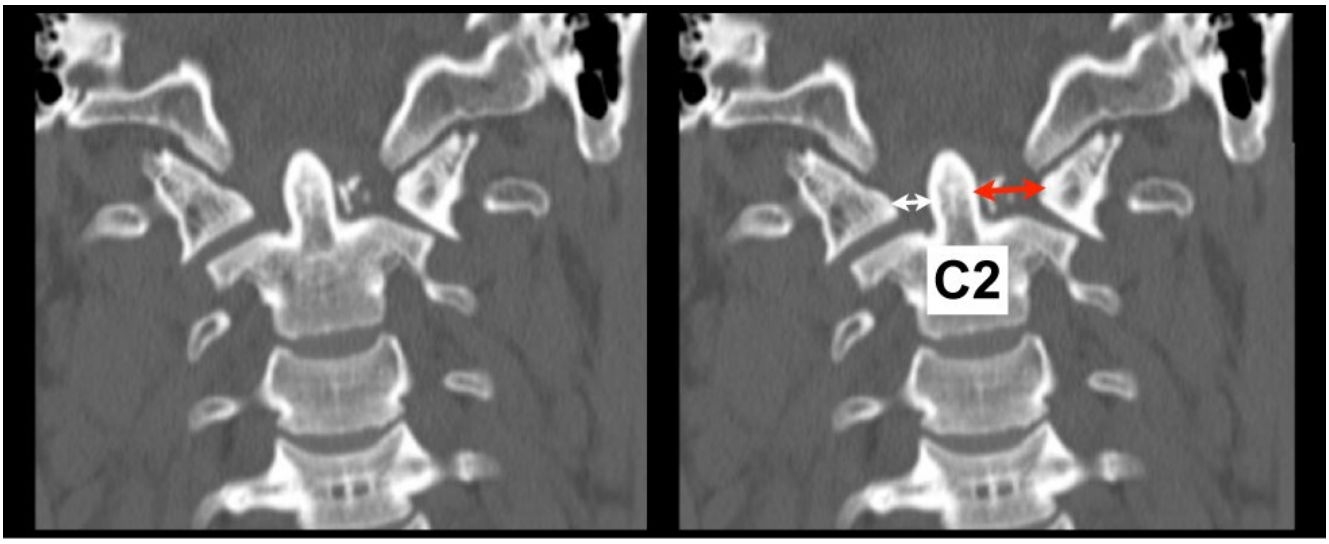


Figure 5b. *Jefferson fracture*. Coronal reformation image with noted asymmetry to the lateral masses of C1 with regard to C2. Red arrow on the patient's left demonstrates widening compared to the corresponding white arrow. (Courtesy of C.E. Smith, MD, Vanderbilt University)

Odontoid fractures. Odontoid fractures account for 5-15% of cervical spine fractures. These fractures are accompanied by a neurologic deficit in 25% of cases and death in 5% to 10% of cases. [vii] Type I odontoid fractures involve an avulsion of the tip of the dens at the insertion site of the alar ligament. While stable in isolation, this pathology is also seen with AOD. Type II fractures, the **most common** (~ 60% of odontoid fractures), occur at the base of the dens. These injuries produce a high degree of non-union when treated non-surgically. Type III fractures extend into the body of the axis. Non-union is also a common outcome. In type II/III fractures, the fractured segment can displace posteriorly and lead to SCI. *Figure 6.*

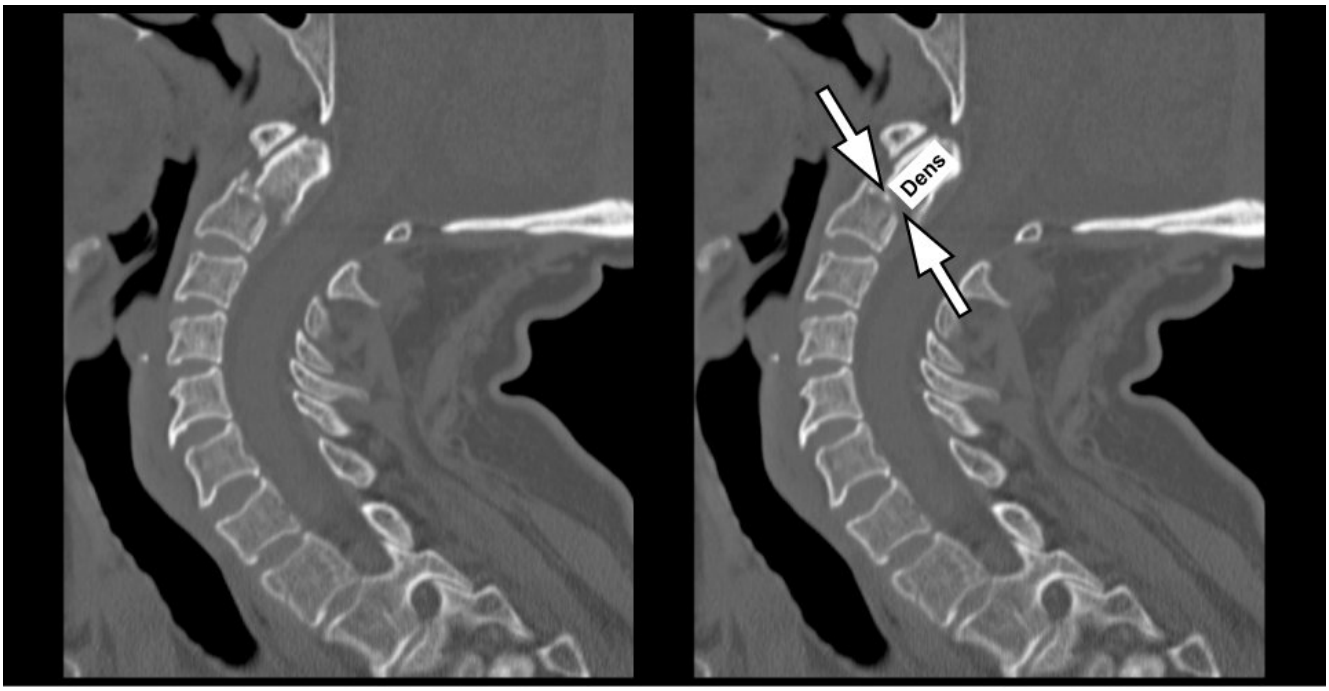


Figure 6. *Type 2 odontoid fracture*. Sagittal reformation image with fracture to the base of the dens of C2 (white arrows). (Courtesy of C.E. Smith, MD, Vanderbilt University)

Flexion/Extension teardrop fractures. In *flexion* teardrop, severe flexion forces cause an anterior-inferior wedge fracture of the vertebral body. *Posterior* column injury results in ligamentous disruption and common neurologic injury. In *extension* teardrop, severe extension forces cause the anterior longitudinal ligament to avulse the anterior-inferior corner of the vertebral body away from the vertebra (often C5-7). Radiographic appearance between flexion and extension teardrops is similar. Depending on the degree of ligamentous injury, the injury may be stable or very unstable. *Figure 7.*

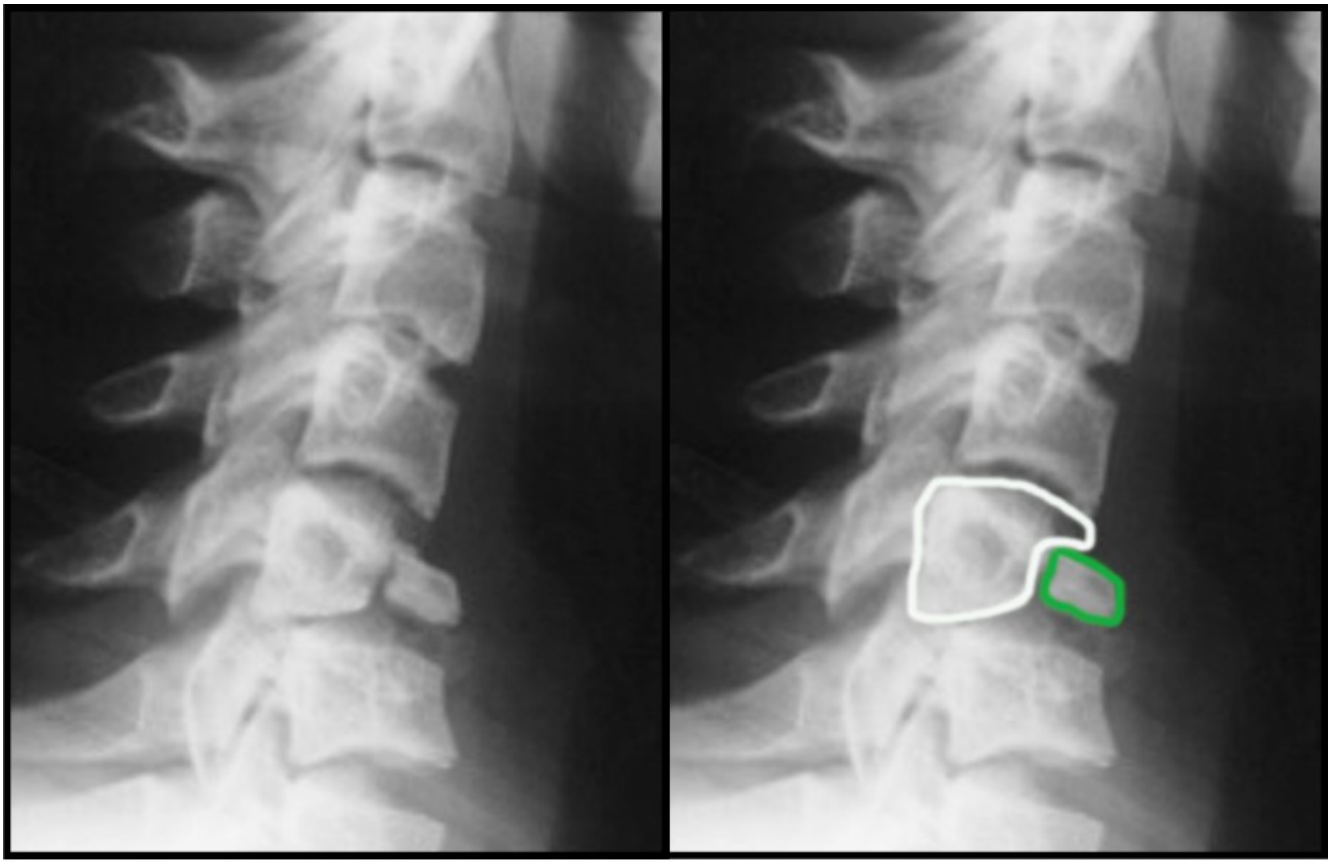


Figure 7. *Flexion teardrop fracture*. Lateral cervical spine radiograph demonstrating a teardrop fracture, as outlined in green.

Which tract is which?

Only three specific clinical syndromes - central cord syndrome, Brown-Sequard syndrome, and the anterior cord syndrome - account for over 90% of incomplete SCI.

The exact incidence of cervical SCI clinical syndromes is unclear. One series from 2007 noted that SCI clinical syndromes (including lumbo-sacral syndromes) accounted for 20.9% of the 839 consecutive admissions of patients with acute SCI. [viii] The most common incomplete cord syndrome is central cord syndrome, representing 44% of *all* incomplete SCI syndromes. Extrapolating from the available data (Figure 8), central cord syndrome is shown to be even more prevalent in the incomplete **cervical** spinal syndromes (65%), followed by Brown-Sequard (25%) and anterior cord syndrome (8%).

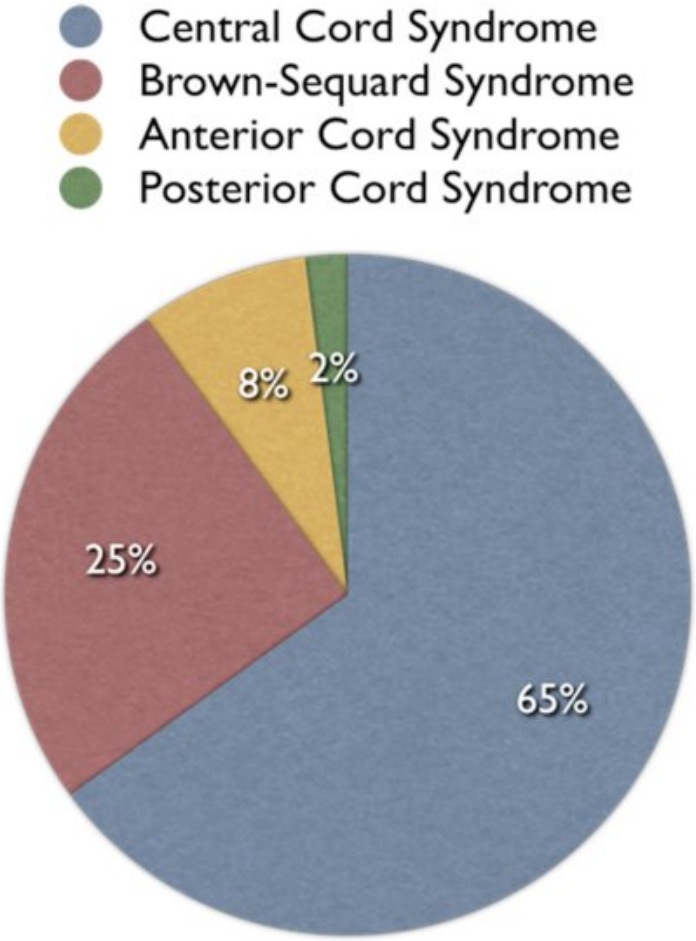


Figure 8. Distribution of the incomplete spinal cord syndromes of the cervical spine. (Extrapolated from data provided by McKinley et al. [8])

Central cord syndrome classically occurs in older patients with pre-existing degenerative disease. However, the mean age of patients in most series is less than 50 years, consistent with the age distribution of SCI in general. One series found that one-half of the patients were under 30 years old at the time of the injury. [ix] In the same study, the most common cervical spine injuries precipitating the central cord syndrome were fracture-dislocation and compression fracture. The mechanism of injury is generally a combination of spondylolysis and a hyperextension injury, which precipitates cord compression by bone spur migration and buckling of the ligamentum flavum. This results in contusion to the central portion of the cord, affecting the central gray matter. The deficits tend to have a characteristic pattern (the mnemonic "MUD" is often described):

- **M**otor deficits are greater than sensor
- **U**pper extremities are affected more than the lower extremities
- **D**istal portions of the limbs (i.e. the hands) are affected more than proximal portions

It is unclear whether the topographic location of arm and leg fibers within the corticospinal tract, or the proportion of fibers dedicated to the upper extremities produces the pattern of central cord syndrome deficits. [x] In the latter hypothesis, injury to the corticospinal tract, which

is thought to *more* commonly subserve hand/arm function, would produce a disproportionate neurologic deficit. Also of note, while sensory deficits are not a "classic" feature, patients can experience a variety of sensory abnormalities: impaired or absent perianal sensation, hyperpathia or excessive sensory response, and Lhermitte's sign (electric shock paresthesias). *Figures 9a and 9b demonstrating normal anatomy and central cord pathology, respectively.*

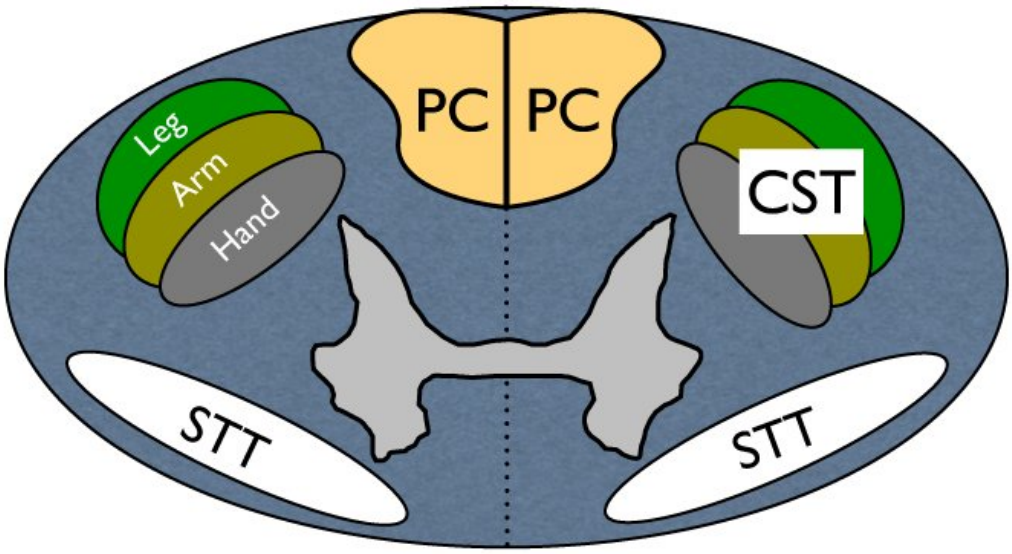


Figure 9a. Normal cervical spinal cord. (PC=Posterior Columns. CST=Cortico-spinal tract. STT=Spino-thalamic tract)

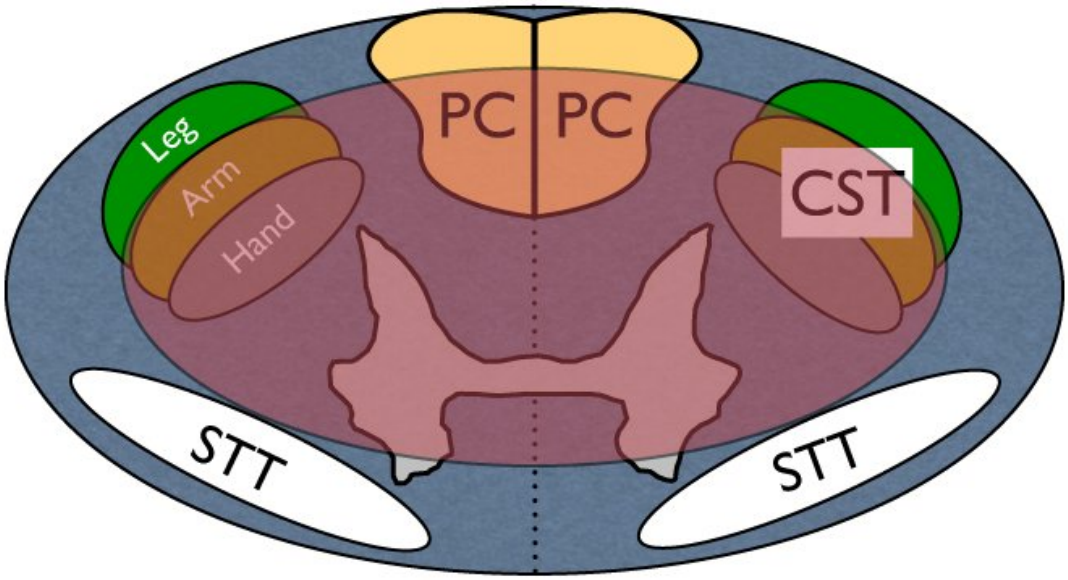


Figure 9b. Central cord syndrome.

Brown-Sequard syndrome results from hemisection of the spinal cord. In most cases, this is the result of penetrating trauma or lateral mass fractures. The lesion produces ipsilateral motor loss (and loss of proprioception) as well as contralateral loss of sensitivity to pain and temperature below the level of injury. Bowel and bladder function is generally maintained, as is

unilateral motor strength. The “pure” form of Brown-Sequard syndrome is uncommon. A “relative” syndrome of motor and sensory deficits is more common. [xi] *Figure 9c.*

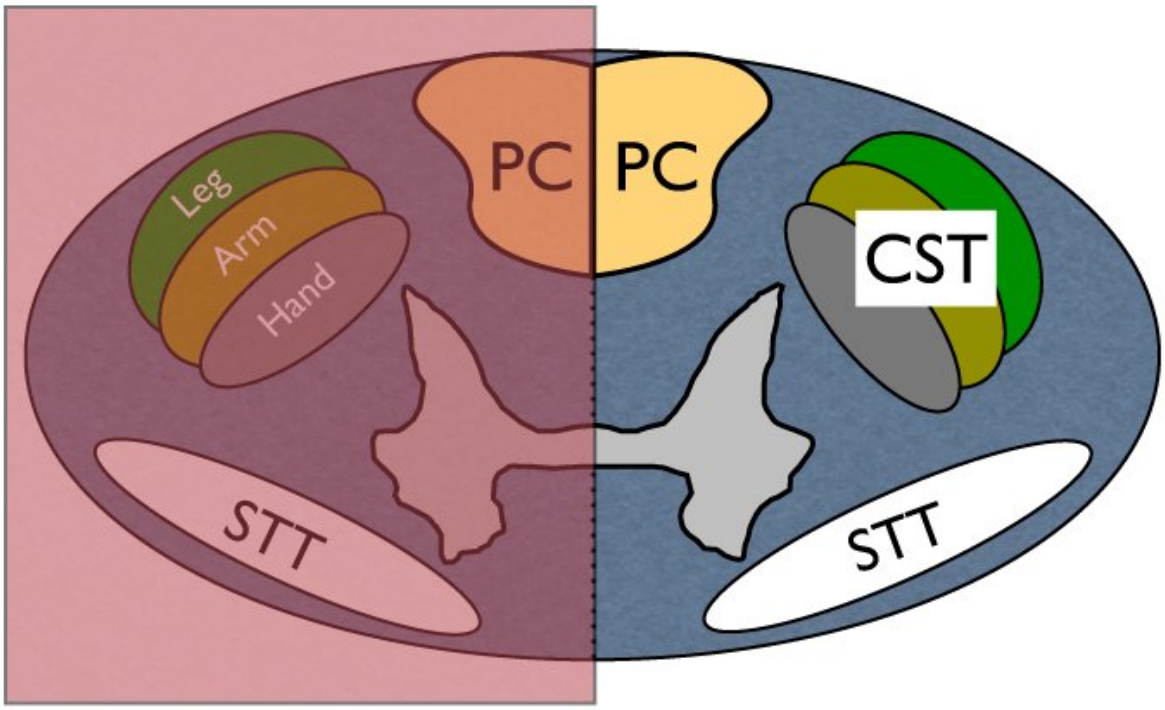


Figure 9c. Brown-Sequard syndrome.

Anterior cord syndrome is due to hyperflexion injuries which result in cord compression. Typically, there is an insult (bony fragment or herniated disk) to the anterior two-thirds of the spinal cord. The posterior columns are spared. Patients experience immediate, complete motor paralysis with hypoesthesia and/or hyperesthesia below the level of the injury. Position, touch, and vibratory sensations are maintained. Unfortunately, the syndrome portends a poor prognosis for functional improvement. *Figure 9d.*

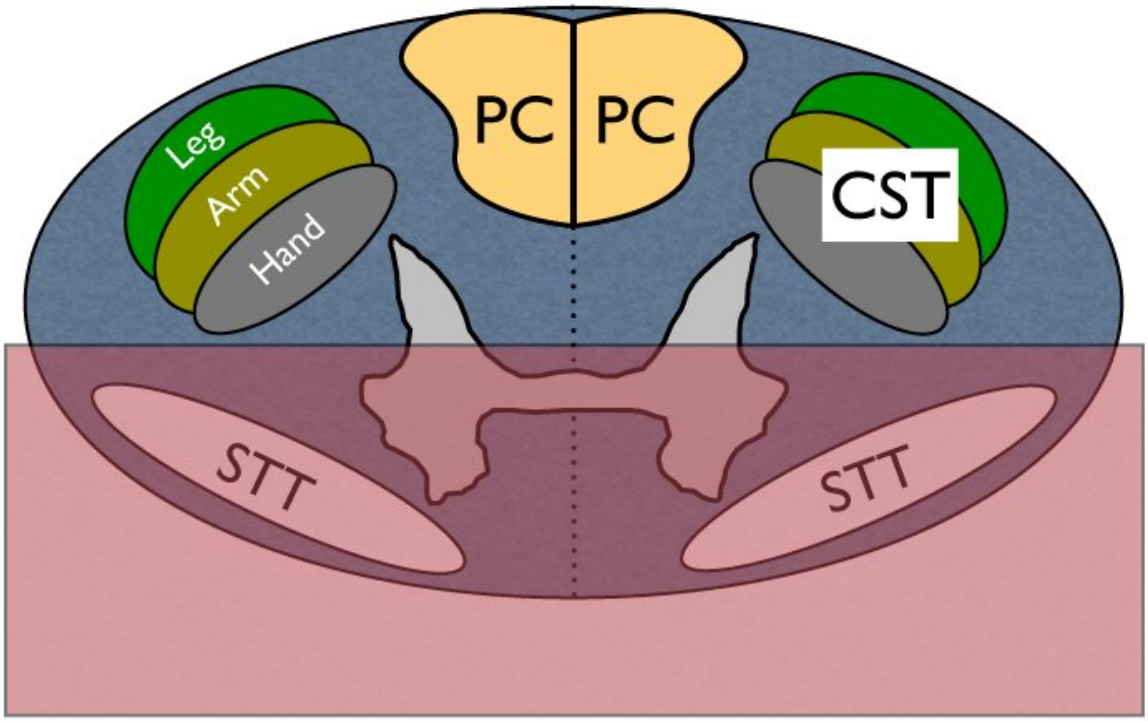


Figure 9d. Anterior cord syndrome.

Hypotension and SCI: Spinal shock, neurogenic shock, or neither?

In the multi-trauma patient with apparent neurologic deficits and hypotension, identifying the source of the "shock" is tantamount. Unfortunately, determining the cause is almost as difficult as sorting through the varied terminology. When asked the question of whether the hypotension is due to a neurologic process, the default answer should be ***NO***. Even in the setting of CSI and apparent spinal cord injury, the shock state is more likely to be due to underlying hemorrhage. More extensive imaging, including bedside ultrasound, may elucidate the specific cause of hypotension. [xii, xiii]

With either physiologic or anatomic transection of the spinal cord, patients may develop temporary loss of all or most spinal reflex activity (including motor and sensory function) below the injury. Flaccid paralysis is observed. These findings are termed ***spinal shock*****.** The return of spinal reflexes in 24-48 hours marks the end of spinal shock, and the neurologic deficits that remain are generally permanent.

Arterial hypotension may or may not occur in this process, depending on the degree to which autonomic reflexes are affected. With injury to the paravertebral ganglia and a loss of sympathetic tone, blood pressure will lower. Cervical lesions may result in a loss of thoracolumbar vascular tone. SCI with severe hypotension that requires aggressive management (including vasopressors) generally occurs in only 20-30% of cases. One study, though, reported persistent bradycardia in 100% of patients with a severe cervical injury, while 68% of patients developed hypotension (35% requiring vasopressors). [xiv] Interestingly, the presence or absence of sympathetic tone (based on blood pressure measurements) does not predict the patient's prognosis or differentiate complete from incomplete lesions. [xv, xvi, xvii] That said, when SCI is accompanied by spinal shock, the overall prognosis tends to be worse.

Neurogenic shock is an amalgam of clinical findings that occur in the setting of spinal shock. The most common signs include hypotension and bradycardia, though hypothermia is also known

to occur. Disruption of sympathetic outflow leads to decreased vascular resistance (hypotension), unopposed vagal tone (bradycardia), and blood pooling in the periphery with associated heat loss (hypothermia). *Tachycardia* and hypotension in the multi-trauma patient are more suggestive of a hypovolemic etiology.

What if my patient remains hypotensive? Studies demonstrate that aggressive blood pressure and hemodynamic management may improve outcomes. [xviii, xix] Cord ischemia is likely to contribute to ongoing neuronal injury/deficit. Since hypotension (systolic < 90 mm Hg) and hypoxia (paO2 <60mmHg) appear to increase morbidity and mortality in traumatic brain injury, some experts recommend maintaining a high mean arterial pressure (MAP 85-90 mm Hg) to improve neurologic outcome in acute traumatic SCI. These recommendations are based on data from studies looking at aggressive hemodynamic management for 5-7 days after injury, with an emphasis on early intervention. [xviii, 20-24] However, these studies were all *non-randomized case series*, in which every patient (no control group) was treated "aggressively" to maintain a high MAP. This Class III data is not able to assign *causation* regarding the improved outcomes after hemodynamic management. That said, appropriate resuscitation in the neurologically injured trauma patient is important, and hypotension is likely to be harmful.

What vasopressor should I use? No particular vasopressor has been shown to be more effective than another. [xiv, xviii] While the clinical setting may favor a particular agent (e.g. dopamine in a hypotensive, bradycardic SCI patient), all of the vasopressors studied improved blood pressure in neurogenic shock. As for target mean arterial pressure, the studies typically used a MAP < 85 mm Hg as the criterion for initiating therapy, though this is by no means a well-studied or definitive standard. In fact, the most recent study by Bilello et al [xx] describes initiation of vasopressors only when hypotension (systolic pressure < 90 mm Hg) was refractory to intravenous fluid administration. No further intervention was required above a systolic blood pressure of 90 mm Hg. Though lacking support from randomized studies, chronotropic agents like atropine are thought to be effective in treating bradycardia due to SCI.

In summary, avoid hypotension, aggressively resuscitate with crystalloid, and consider vasopressor support particularly if the patient remains hypotensive (<90mmHg). [xxi]

Steroids in SCI - What's the latest?

Before you read any further, let's cut to the chase: ***The jury is still out on the issue of steroids in SCI***. This article will *not* be a position statement in the debate over steroids. Instead, the literature will be briefly reviewed, and the reader can make up his/her own mind on the topic.

The debate will be summarized through a brief timeline regarding a few of the most recognized studies (and reviews) of methylprednisolone (**MP**):

1990/1992 - National Acute Spinal Cord Injury Study (NASCIS) 2 trial was published in the *New England Journal of Medicine*. At 1 year after injury, there were no differences in neurologic recovery in the MP group compared to placebo. A subgroup treated *within* 8 hours did appear to benefit (average increase in motor score 5.2 compared to placebo). [xxii, xxiii] What this motor improvement actually means is difficult to extract from the data, and is beyond the scope of this article. The precise clinical improvement denoted by a 5-point gain generally depends on whether the improvements are concentrated in a few myotomes, or scattered over a number of myotomes. In the former scenario, a C5 quadriplegic might be reclassified as a C7 quadriplegic (better with self-care and transfers). In the latter scenario, there would be no clinically significant functional improvement. [xxiv]

1997 - NASCIS 3 trial was published in *JAMA* [xxv], randomizing patients into three treatment groups (no placebo). Motor improvements were observed at 6 months for the MP infusion

groups: 3.4 for 24 hour infusion, 2.8 for 48 hour infusion. At 1 year, the difference in raw motor scores between the 48 hour and 24 hour MP protocol between 3-8 hours was only 1 point.

1998 - An assessment of the NASCIS 2 and 3 trials questioned the statistical analysis, randomization, and clinical endpoints used in the studies. [xxiv] Moreover, the significance of the clinical "gains" was questioned and the higher incidence of sepsis and pneumonia in patients treated with MP was noted.

2000 - A prospective, randomized trial from France, looking at nimodipine, MP, or both versus no medical treatment showed no difference between the treatment and control groups. [xxvi]

2000 - Cochrane review: "High dose methylprednisolone steroid therapy is the only pharmacological therapy shown to have efficacy in a Phase Three randomized trial when it can be administered within eight hours of injury." [xxvii] The review noted that the NASCIS findings were replicated in a Japanese study, but not in the French study referenced above. Also of interest is that the lead author for the Cochrane review was a major contributor to the NASCIS series.

2002 - Hadley **[xxviii]** reviewed the relevant steroid literature and voiced concerns regarding the NASCIS 2 and 3 trials, including the lack of measure of functional significance to the motor gains, dependence on post-hoc analyses, and no analysis of surgical treatment.

2003 - The Canadian Association of Emergency Physicians declared that the evidence was insufficient to support the use of steroids in SCI as a treatment *standard* or as a *guideline* for treatment. It was simply a treatment *option*. [xxix]

2006 - Questionnaire survey of surgeon members of the North American Spine Survey, which reported that while 91% used the MP steroid protocol, only 24% actually believed it worked. **[xxx]**

Summary:

- Few randomized, controlled trials have studied MP versus placebo in acute SCI.
- There were some modest motor benefits observed in both complete and incomplete SCI patients treated with MP within 8 hours. Incomplete lesions tended to improve more than complete lesions.
- Many criticisms have been made of the NASCIS studies.
- The complications of steroid use, including pulmonary, gastrointestinal, infectious, and time to discharge, are legitimate. These concerns are particularly relevant to older patients.

Take-home point for Emergency Physicians:

- This is not a decision you will make in isolation. The determination to use steroids should be made in conjunction with your trauma team or referral trauma center.

Do Patients Pith Themselves?

Is cervical immobilization necessary in the pre-hospital setting? The default response, based on textbooks and ATLS courses, is a resounding ***YES***. In fact, this issue is somewhat controversial.

Consider the following case:

> *An 82 year-old male with history of ankylosing spondylitis has a documented C6 fracture after a fall. He is placed in a hard cervical collar and later progresses to quadriplegia and death.* [xxxi]

Cervical collars may indeed protect the spine, but they also *produce* harm in certain patient populations and contexts. Disease entities such as rheumatoid arthritis or ankylosing spondylitis are known to cause a fixed flexion deformity. *Recommendation*: Avoid changing the "usual alignment" of the cervical spine.

Cervical collars have been associated with a number of other problems: [xxxii, xxxiii]

- Increased intracranial pressure
- Pressure sores
- Ventilator-associated pneumonia
- Reduced tidal volumes
- Dysphagia
- Pain

The main push for cervical immobilization found its roots in population statistics (1970s and 1980s). During the 1970s, most patients (55%) referred to regional spinal cord injury centers arrived with complete neurological lesions. In the 1980s, however, most spinal cord-injured patients (61%) arrived with incomplete lesions. [xxxiv] This improvement in the neurological status of patients was attributed to the development of emergency medical services (EMS) initiated in 1971, and the care (including spine immobilization) rendered by EMS personnel before the patient reached the hospital.

One question is whether immobilization actually *immobilizes*. In a study of 6 fresh, elderly cadavers, surgical lesions were made to the cervical spine (rendering unstable motor segments). Stress was then applied to the models in the collars. No effective limitation of movement was found, despite the collars. In fact, in most loading modes, displacement was less with *no* collar. [xxxv]

A study out of the University of New Mexico performed a retrospective chart review that compared its own neurologic outcomes to those of a sister hospital in Malaysia. [xxxvi] New Mexico boarded and collared all of their patients (334) en route to the hospital, while Malaysia immobilized none of theirs (120). Neurologic disability was less for the Malaysian patients (21% in New Mexico versus 11% in Malaysia). Several limitations exist to the study, including the nature of the injuries, the patients excluded from the study, and the lack of "injury matching" based on Injury Severity Scores.

A number of articles serve as counterpoints to the New Mexico experience. These studies, in general, speculate that the development of secondary neurologic deficits is a ***result*** of delayed diagnosis, and hence inadequate management (including proper immobilization). A few of the articles are summarized in Table 5. [xxxvii-xxxviii, xxxix]

Reid et al.	• Prospective study of 253 patients with 274 spinal injuries • Delayed diagnosis in 22.9% cervical and 4.9% thoracolumbar injuries • Secondary deficit was 10.5% in the delayed diagnosis group versus 1.4% in those whose fractures were identified on initial screening
Ravichandran et al.	• British series of trauma patients; 4.3% missed spinal injuries • 10/15 patients deteriorated neurologically
Davis et al.	• Retrospective analysis of cervical spine injuries at 6 trauma centers • 740 patients, delayed/missed diagnosis in 34 (4.6%) • 10/34 (29%) developed permanent sequelae as a "result" of these delays
Rogers et al.	• Observational experience of the author, an orthopedic surgeon, over a 10 year span (1940-1950) • 77 patients with 87 cervical fractures • 1 in 10 patients with worsening neurologic deficit after the injury

Table 5. Studies of delayed diagnosis and neurologic deterioration. [92-95]

The backboard, or "long spine board," is an effective means of moving patients in the pre-hospital setting, but plays little role in the ED management of cervical spine injuries. There is no evidence to suggest that maintaining patients on spine boards provides any added benefit after arrival to the ED. [xl]

In summary:

- There is *no Class I or Class II* medical evidence to support spinal column immobilization in all patients after trauma.
- Although immobilization of an unstable cervical spinal injury makes good sense, and *Class III* evidence reports neurological worsening with failure of adequate spine immobilization, no case-control studies or randomized trials address the effect of spine immobilization on clinical outcomes after cervical spinal column injury.
- Cervical spine immobilization is still considered the standard of care until more definitive research arrives.

A 2001 Cochrane review stated, "The effect of spinal immobilization on mortality, neurological injury, spinal stability and adverse effects in trauma patients remains uncertain," and emphasized the need for large prospective studies to validate decision criteria for spinal immobilization [xli].

Does secondary neurologic injury equal poor hospital (ED) management?

Primary and secondary injury is difficult to both characterize and differentiate. A complex cascade of events follows the SCI. The initial damage is due to the mechanical impact to the cord, causing hemorrhage and edema. Secondary degeneration then follows, with further hemorrhage, edema, and tissue hypoperfusion.

Class III evidence suggests neurologic deterioration when injuries are missed. "Secondary" neurologic injuries are observed to occur in 10-29% of patients who are initially un-diagnosed. Subacutely, a neurologic deficit either manifests or simply worsens. Did the delay in diagnosis promote further cord trauma and progressive neurologic deficits? In fact, evidence suggests that neurologic deterioration occurs even when management is appropriate. Somewhere between 2-10% of patients with early diagnosis and standard-of-care management will still suffer deterioration (i.e. an ascending myelopathy). This progressive, post-injury process is reportedly linked to vascular dysfunction (vasospasm, hemorrhage), cord edema, inflammation, and apoptosis. [xlii, xliii, xliv, xlv, xlvi]

Neurologic deterioration may simply represent a natural progression of disease, rather than a product of delayed diagnosis or poor medical care. Cases of missed and/or delayed diagnosis with neurologic deterioration, however, produce a rather complicated medico-legal situation. **[xlvii]** For now, strict spinal precautions remain the standard for all CSI patients arriving in the Emergency Department.

Imaging the C-spine: *When* and *How*?

Recommendation: Employ a clinical decision rule when considering cervical spine imaging. The two main decision rules regarding C-spine imaging are the NEXUS criteria and the Canadian C-spine Rule (CCR). [xlviii, xlix] Both rules have been extensively validated and are excellent decision rules. The relative strength of each has been roundly debated. [l] (Tables 6 and 7)

No radiography if:

- No posterior midline cervical spine tenderness
- No evidence of intoxication
- Normal level of alertness
- No focal neurological deficit
- No painful distracting injuries

C-spine tenderness = pain on palpation of the posterior midline neck from the nuchal ridge to the prominence of the first thoracic vertebrae
Intoxication = recent history by patient or observer, odor of alcohol, slurred speech, ataxia, dysmetria or other cerebellar findings, any behavior consistent with intoxication
Alertness = GCS 15; must be oriented to person, place, time, events; remember three objects at five minutes; no delayed or inappropriate response to external stimuli.
Focal deficit = any focal neurological finding on motor or sensory examination
Distracting injury = any condition through by the clinician to be producing pain sufficient to distract the patient forma second (neck) injury. Long-bone fracture, visceral injury requiring surgical consultation, large laceration, degloving injury, or crush injury, large burns, etc.

Table 6. NEXUS Low Risk Criteria (NLC) Algorithm for screening of neck injuries. [54]

No radiography if:

- If NO high-risk mechanism
- Age < 65
- NO paresthesias in extremities
- Presence of at least one low-risk factor
- Able to rotate the neck actively 45 degrees left and right

Applies to alert patients (GCS 15), stable condition.

High-risk mechanism: fall from > 3 feet or 5 stairs, axial load to the head (diving), MVC at high speed (>110km/hr or 62mi/hr) or with rollover or ejection; collision involving motorized recreational vehicle; bicycle collision; being hit by a bus, large truck, or high-speed vehicle.

Low-risk factors: Simple rear-end motor vehicle, collision or sitting in the ED or ambulatory, delayed onset of neck pain or absence of midline cervical tenderness.

Table 7. Canadian C-Spine Rule. [55]

If a patient, based on NEXUS or CCR, needs imaging, should it be a plain radiograph or computed tomography? Regarding CT versus plain films, the American College of Radiology takes the position that CT should be the primary screening study for any patient who meets criteria for imaging based on either CCR or NEXUS. [li] This position is supported by a number of studies that demonstrate a low sensitivity of plain films for identifying clinically significant fractures. [lii, liii, liv, lv, lvi] One study by Nunez et al. [lvii] showed that up to 40% of C-spine fractures were missed on plain films. One-third of these were "clinically significant" or unstable.

In these studies, however, the frequency of *inadequate* plain films reached 30-40%. No wonder there was an alarming number of "missed" fractures. One study specifically looked at the issue of inadequate plain films, noting incomplete or inevaluable cervical spine radiographs in more than **one-third** of patients imaged. [lviii] The take-away is that poor plain radiographs equals missed injuries. In these cases, CT is necessary.

The decision to perform plain films or CT should also take into account the likelihood of other injuries. It is rare to have an isolated CSI; 80% of CSI are accompanied by other trauma, in particular head injuries and other spine injuries. [lix] In a recent retrospective chart review of the National Trauma Data Bank between 2002-06 (more than 2.7 million traumas), cervical spine injuries were reported in 6.7% of facial fractures and 7.0% of head injuries. Conversely, facial fractures and head injuries were observed in 13.5% and 40.2%, respectively, of patients with CSI. [lx]

The flip side of an increasing reliance on CT imaging of the cervical spine involves increasing medical costs and radiation exposure. A great deal of recent literature addresses the radiation dose of various CT studies, and the associated lifetime attributable risk of cancer. [lxi] Depending

on the source, estimates can vary. Smith-Bindman et al. [lxii] reported the median effective doses of radiation for various scans at 4 Northern California institutions. There was a great deal of variation in radiation exposure within and across institutions. The authors documented even *higher* doses of radiation than previously thought or reported in the literature.

Cervical spine CT applies a high radiation dose to the thyroid, which is quite radiosensitive. Rybicki et al. [lxiii] reported a marked increase (14-fold) in radiation to the thyroid when CT replaced plain radiographs of the cervical spine. A number of studies tout a lower-dose protocol for CT of the cervical spine, without notable differences in image quality. [lxiv, lxv]

Use common sense when imaging: Rather than whisk every NEXUS+ or CCR+ patient to CT, others advocate an additional decision rule to guide imaging. The hope is to identify a "low risk" population in whom plain films would be adequate. CT scans would then be used primarily in a "high risk" population. One clinical decision rule by Hanson et al. addresses this issue. [lxvi] Specifically, the authors sought to identify a select group of adult patients who were at greater than 5% risk for cervical spine fracture. The study group included 4,285 patients: 3,685 had "low risk" factors and underwent plain radiography, while 601 patients with "high risk" factors underwent CT (Table 8).

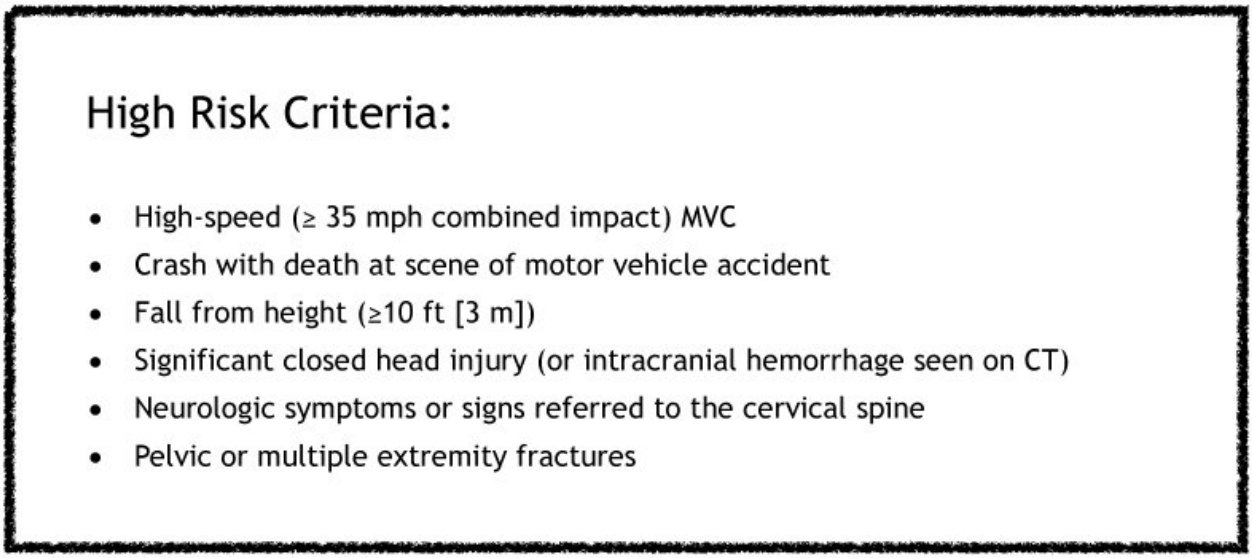


Table 8. Hanson et al. "High-Risk" Criteria. [72]

When validated, the group identified as "high risk" by the decision rule had a CSI rate of 8.7% by CT. In the "low risk" group who underwent plain radiographs alone, the incidence of CSI was 0.2%. While this study deserves further validation, it does reflect an important component of emergency care: assigning pre-test and post-test probabilities to various disease presentations.

MRI, the Emergency Department, and CSI: The role of MRI in the Emergency Department work-up of CSI is widely debated. There are few clear indications for emergent MRI in CSI patients. MRI is appropriate in patients with objective neurologic deficits, despite negative plain radiography and CT. This is especially true in the pediatric population with clinical features of spinal cord injury without radiographic abnormality, or SCIWORA. Beyond that, MRI is not considered a primary imaging modality in most CSI patients.

An awake, neurologically intact patient with a negative MDCT study has an extremely low risk of clinically significant CSI. This includes patients with persistent, severe midline cervical pain. [lxvii] In this patient population, there is little if any role for MRI. The likelihood of a clinically significant ligamentous injury in this population is so low that cervical immobilization is rarely beneficial.

Excluding cervical spine injuries in obtunded patients, however, remains controversial. While some studies suggest that MDCT identifies all unstable injuries in obtunded/unreliable patients, a recent meta-analysis noted that MRI altered management in 6% of cases (5% with continued collar immobilization, 1% required surgical stabilization) in which the CT was negative. [lxviii, lxix, lxx, lxxi] Ligamentous injuries were the most commonly "missed" abnormality. The meta-analysis was limited by the retrospective nature of the majority of the studies, as well as the heterogeneity of study design, patient population, and data analysis.

Summary:

- MRI is unnecessary if the CT is negative in neurologically intact trauma patients with neck pain (resolved or persistent).
- The role of MRI in obtunded patients with a negative CT is controversial; the decision to pursue further imaging will generally occur in an inpatient setting, not in the ED.

CSI and the emergency airway: Is it more of the same?

There is a variety of dogma that governs management of the airway in a patient with documented or suspected CSI. Much of this is unsubstantiated. We will discuss:

- Manual in-line stabilization
- Spinal movement during airway management
- Direct laryngoscopy and other advanced airway adjuncts in CSI

Manual in-line stabilization

In-line stabilization describes a maneuver to limit head and neck movement during airway management. Typically, an assistant crouches next to the patient, holding both mastoids and cradling the head. Studies clearly demonstrate that while *overall* spinal movements may be restricted by this maneuver, there may be little restriction of movement at the injured segment. [lxxii, lxxiii, lxxiv] Compared to full immobilization (collar, tape, and sandbags), the largyngoscopic view with in-line-stabilization was superior. [lxxv, lxxvi] In-line stabilization remains the standard of care.

When does the cervical spine move the most? Which airway technique is best?

When evaluating each step of emergency airway management, it turns out that the intubation itself (i.e. direct laryngoscopy, or DL) does ***not*** produce the most cervical spine movement. In one study of eight recently deceased trauma victims, various intubation procedures were performed including mask ventilation, oral intubation, and nasal intubation. Maximum cervical spine displacement, in fact, occurred with mask ventilation. [lxxvii] Another study of unstable spine cadaver models looked at chin lift, jaw thrust, and head tilt maneuvers, as well as placement of oral and esophageal airways. [lxxviii] Head tilt (downward displacement of the head with extension of the head and neck) produced no significant changes. Chin lift and jaw thrust resulted in the *same* amount of distraction, while disc space enlargement was similar among the various airways. The presence of a cervical collar did not effectively immobilize the neck for any of the maneuvers.

Another cadaver study looking at C3 posterior injuries showed similar displacement with a variety of airway interventions (facemask ventilation, DL, fiberscope-guided nasal intubation, laryngeal mask airway, and Combitube insertion). The least displacement occurred with fiberoptic nasal intubation. Most maneuvers produced 1.7-3.7 mm of displacement. [lxxix] *The clinical significance of any of these small spinal movements is unknown.*

Head-to-head comparisons of rigid fiberoptic scopes and DL have also been done. When evaluating the Bonfils intubation fiberscope to DL, slightly more spinal movement occurred with DL. The Bonfils scope produced superior laryngoscopic views. [lxxx] When the Glidescope was compared to DL in collared patients, the laryngoscopic view was again better with the fiberoptic video larygnoscope. [lxxxi] The Glidescope decreased spinal movement to some extent, but only at the C2-5 segment. Motion at the other segments was similar. [lxxxii] Importantly, no difference in spinal movement has been noted based on the type of DL blade used. [lxxvi, lxxxiii]

So, **in summary**, when compared to DL, rigid fiberoptic scopes:

- Produce better intubation views.
- Increase the time to intubation.
- Slightly decrease cervical spine movements. The clinical significance of these decreased movements is unknown.

Is direct laryngoscopy really O.K. for intubating patients with cervical spine injuries?
The outcomes literature regarding CSI patients and airway management is pretty straightforward:

- *No differences in neurologic deterioration in CSI patients have been observed whether direct laryngoscopy, blind nasal intubation, or fiberoptic bronchoscopy was used for airway management.* [lxxxiv, lxxxv, lxxxvi, lxxxvii]

Even in the *rare* case study that documents neurologic deterioration after intubation, there is no definitive evidence that the intubation maneuver produced the deficit. With an appreciation of secondary degeneration and the natural course of SCI, it is not surprising that a few of the patients studied would have progressive neurologic deficits over time.

Should we use an advanced airway technique just "to be safe"? Anesthesiologists are known to favor flexible fiberoptic bronchoscopes. [lxxxviii] The literature, however, is sparse regarding this technique in the setting of trauma. [lxxxix] It is also not a standard approach for an emergent "crash" airway. Moreover, in one study of patients undergoing *elective* cervical spine surgery, up to 12% were noted to desaturate (mean saturation 84%) during bronchoscope-facilitated intubation. [xc]

For now, the most appropriate airway technique in CSI remains debatable. The best approach in patients with airway compromise and a concern for CSI is to employ the technique with which you are most familiar.

Returning to our nightmare case described at the introduction:

Are the criticisms fair?
Modality of cervical spine imaging: CT of the cervical spine would have been more appropriate in this case, as he met "high risk" criteria. Also, the plain radiographs were "inadequate", and deserved further imaging, which was eventually performed. *Fair criticism.*

Positioning maneuver for intubation: No evidence exists to suggest a benefit of jaw thrust over head tilt/chin lift. Spinal movement is the same for the various positioning maneuvers. The assistant appropriately maintained in-line stabilization. *Unfair criticism.*

Choice of DL for intubation: No evidence exists to support one intubation maneuver over another. While there may be a *slight* decrease in spinal movement with fiberoptic intubation, the clinical significance of this is debatable. Moreover, issues of desaturation and time to intubation

must be weighed into the decision of which technique to use. The most skilled airway expert using the most familiar approach is best. *Unfair criticism.*

Failing to initiate steroids: This decision is not made in isolation. The evidence does not clearly demonstrate a significant benefit to steroids, but the long-term management of SCI patients is not in the Emergency Department. The decision to initiate steroids should be in conjunction with your trauma team or referral trauma center. *Unfair criticism.*

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