Assessing the acute spinal cord injury patient

Introduction

Timely intervention in a spinal cord injury has been documented to decrease morbidity and mortality. It is therefore necessary to know how to recognize and approach the acute spinal cord injury. The concept of “time is spine” highlights the importance of goal-directed interventions that should be accomplished in a timely manner. There is no cure for the spinal cord injury however, building on the findings from current research, some novel approaches are predicted to substantially improve short-term and long-term functional recovery.

In this short document we will revisit briefly the pathomechanics and pathophysiology of the acute spinal cord injury. We will also discuss the patients that were seen at the PGH in a 10 year period from the standpoint of a hospital setting. Lastly, we will discuss the necessary steps that a clinician has to undertake to maximize the recovery potentials of these patients.

Pathomechanics

Injury to the spine is dependent also on two elements of load transmission, the amount of force applied and the position of the body during the application of force. The specific anatomical structure and location of injury that the spine sustains is dependent in the mechanism of injury applied. It is also sensitive to the initial position of the head relative to the spine, the rate of loading and the degree to which the neck is constrained also affect the tolerance. Individual parameters, including the presence of preexisting pathology or deformities, as well as the geometry and bone density, also affect biomechanics.

Penetrating or direct injury occurs, when the force is directly applied to the spinal cord. This is commonly seen in the penetrating injuries to the spine, like gunshot and stabbing injuries. In contrast, blunt injury to the spinal cord occurs when the force is transmitted through the body causing the most susceptible area to fail. Blunt injuries are by far, the more common causes of spinal cord injury. This type of injury occurs when the forces acting on the spine cannot be tolerated thus causing the spine to fail.

The common blunt mechanisms for injuring the spinal cord are summarized as follows: axial deformation, axial rotation/torsion, segmental translation and combined mechanisms (1). This corresponds to the classification used in spinal cord injuries.

1. Axial deformation has two possible variants, compression and distraction (tension). Compression forces act on the sagittal plane that causes a flexion on one side of the spine and extension on the other side. Distraction or elongation, often occurring at the cervical spine, act on the longitudinal axis.

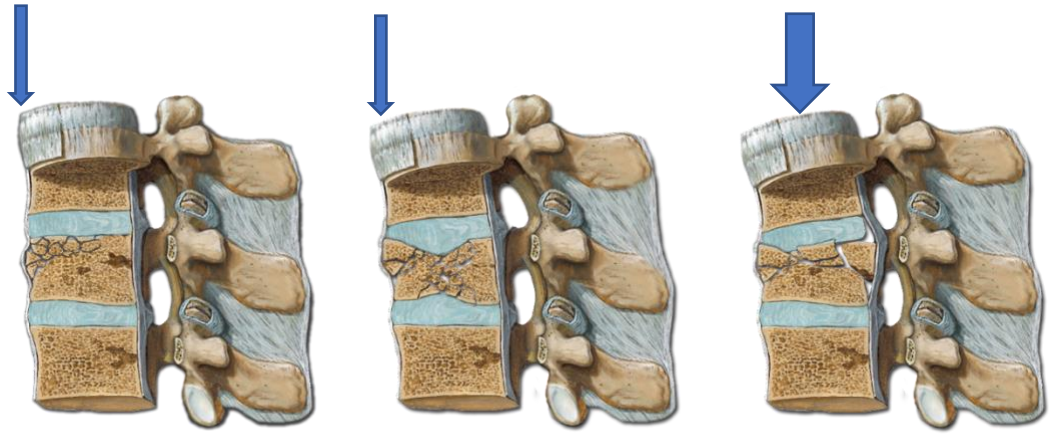


Figure 1.1: Axial deformation via compression
Eccentric deformation with central axial spinal pillar unaffected resulting in (a) superior wedge fracture, (b) vertebral body collapse and without instability. And thru centric compression and bony retropulsion resulting in a (c) burst fracture with instability.

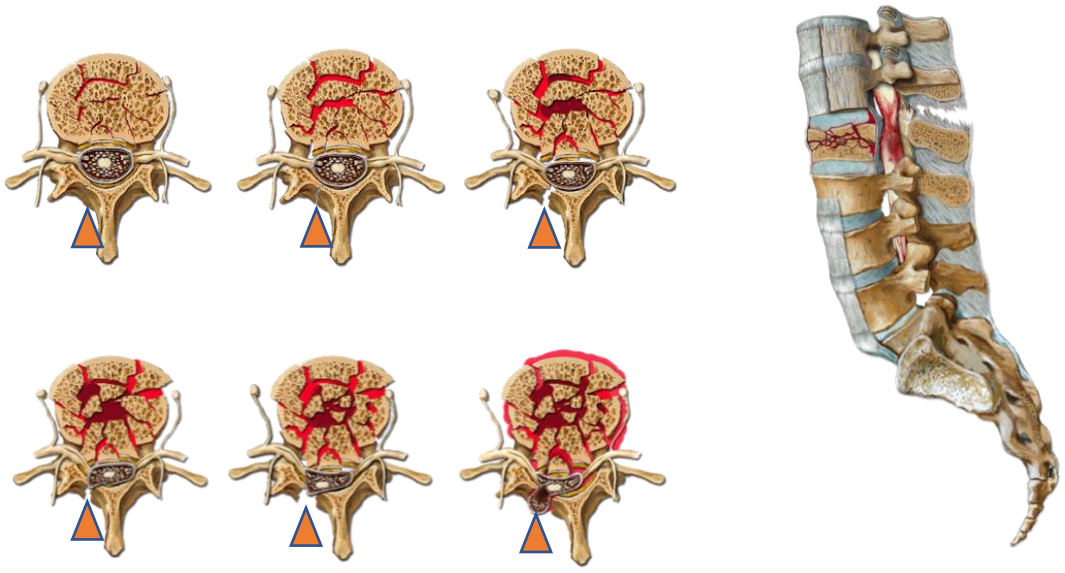


Figure 1.2: Axial deformation – Severity of compression
Concentric deformation with central axial spinal pillar unaffected resulting in (a) vertebral body with right laminar fracture, (b) vertebral body collapse/burst fracture with <50 retropulsion, (c) laminar fracture displacement (d) >50 bony retropulsion (e) progression of laminar displacement and protrusion of thecal sac, (f) extrusion of thecal sac, dural tear considered (g) sagittal representation of the severe cord compression secondary to a burst fracture. Bony instability, hematoma formation and cord contusion contributes to cord injury.

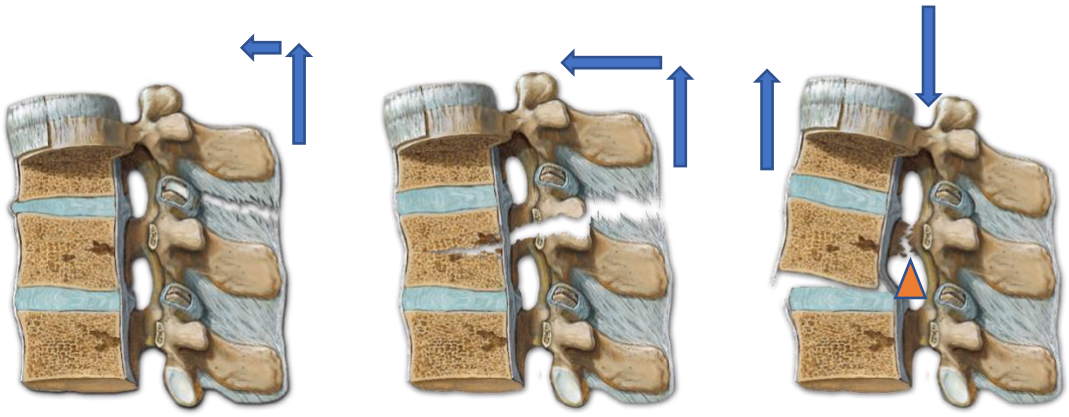


Figure 1.2: Axial deformation via distraction, flexion or extension
Eccentric deformation and flexion with central spinal pillar unaffected resulting in (a) posterior ligamentous injury and facet distraction, with involvement of the central pillar resulting in (b) Chance fracture or transpedicular transverse shear vertebral body fracture and posterior ligamentous injury. And (c) thru centric distraction and extension resulting in ligamentous/disc injury anteriorly with pedicular fracture.

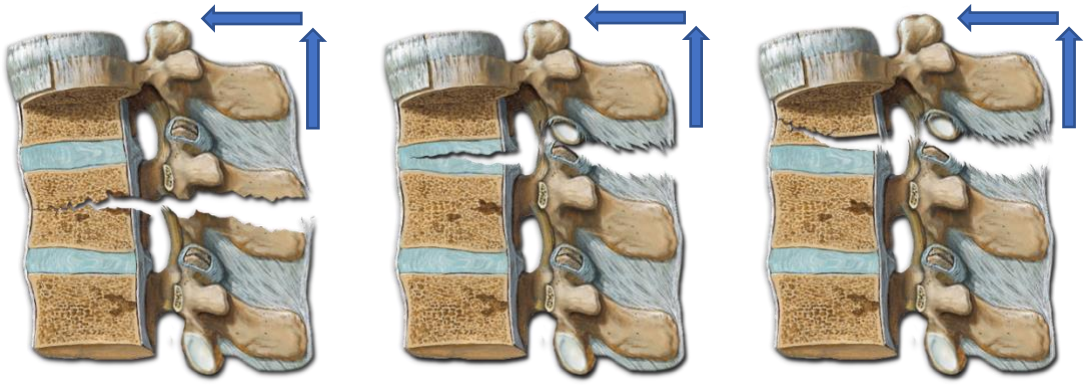


Figure 1.3: Axial deformation via distraction and flexion
Eccentric deformation and flexion with central spinal pillar unaffected resulting in (a) a mainly bony injury of spinous process, laminar and transverse vertebral body fracture, (b) no bony involvement, shearing thru posterior ligaments, the facet joint and disc, or (c) a combination of both, shearing thru the posterior ligaments, the facet joint, disc and vertebral body.

2. Torsion or axial rotation is a rotational force acts on the transverse plane
3. Segmental translation is a shearing injury mechanism acting on the transverse plane.
4. Combined injuries are usually secondary to the resolution of forces applied to the body, it is very difficult to get the force to be applied at a specific anatomic plane.

Pathophysiology

The injury sustained from spinal cord trauma may be divided into primary and secondary. The degree or severity of neurologic injury derives from the initial injury itself as a result of the direct mechanical trauma to the cells and microvasculature of the spinal cord.(2) The secondary injury is the so-called “second hit”, this is a cascade of pathophysiologic processes that render more injury, ironically as the body tries to compensate. Secondary spinal cord injury occurs in phases. These phases are the immediate phase, the acute phase and subacute phase. (3) There is not much that can be done for the primary injury, however, the secondary injury presents an opportunity for the healthcare worker to positively alter the natural course of the injury.

Spinal cord pathology is dependent on two physiologic time frames: The initial or primary trauma and the subsequent secondary insult from ischemia, hemorrhage and edema. A cascade of physiologic, extracellular biochemical, and intracellular insults comprises the secondary injury phase. (4)

Damage and disruption of neurons results in increased extracellular glutamate concentration hastening cell death. There would also be an influx of cytokines, vasoactive peptides, and peripheral inflammatory cells due to the injury of the spinal cord barrier allowing increased cord edema and inflammation. (5) Later in the cascade, cell death is propagated by the influx of cytotoxic by-products. (6) Glial scar formation forming a physical barrier to contain the injury. Numerous mechanisms further restrict regeneration and plasticity to facilitate neurologic recovery.

Spinal shock is defined as the complete loss of motor and sensory function below the level of injury. The end of spinal shock indirectly indicates the prognosis of the injury, by identifying if the injury is complete or not. Spinal shock occurs in phases: (1) initial phase/period of areflexia as a result of lost stimulation and increased inhibition of the descending supraspinal tracts, (2) return of cutaneous/bulbocavernosus reflex which occurs between day 1 to 3 due to denervation sensitivity, (3) period of hyperreflexia due to axon-mediated synapse growth and lastly, (4) period of spasticity and hyperreflexia due to continued synapse growth.

Injury to the sympathetic tracts from cervical and upper thoracic injuries may result to neurogenic shock (bradycardia and hypotension) because of unopposed parasympathetic outflow. Hypoperfusion, hypoxemia and hypercarbia may be observed due to decreased innervation of the primary (diaphragm) and secondary (intercostals and abdominal) muscles of respiration. This predisposes to an increased risk for pneumonia due to impaired coughing and clearance of secretions.

Care for the spinal cord injured patient

Immediate

Immediate or emergent care for traumatic injuries of the spine should not differ from the care given to all trauma patients. It must be assumed that the victims have cervical trauma if they are unconscious during extrication. The hierarchy of airway breathing, and circulation remain the cornerstone of initial management. The cervical spine should be stabilized by a rigid collar during extrication.

Airway management of cervical spine patients is challenging but should be addressed. A patient who is combative or with a high risk for aspiration, extra care is needed when performing intubation. Endotracheal intubation should be performed via the oral route with a rapid sequence induction with manual in-line stabilization. The airway management team demands a physician who is experienced in airway control techniques.

Hypotension occurs in the spine trauma patient. This is usually caused by hemorrhage or neurogenic shock. Maintenance of normal blood pressure also prevents secondary ischemic changes. A target MAP of 85 mmHg should be maintained in the first 5 days. Vital signs are often confusing in acute spinal cord injury because of an increased incidence of associated injuries. A diligent search for occult sources of hemorrhage must be made. The common areas where occult hemorrhage may be present include the chest, abdomen, retroperitoneum and fractures of the pelvis or long-bones. Appropriate investigations, including radiography or computed tomography (CT) scanning, are required. In the unstable patient, diagnostic peritoneal lavage or bedside focused abdominal sonography for trauma (FAST) ultrasonographic study may be required to detect intra-abdominal hemorrhage.

Once hemorrhage is ruled out as a cause for hypotension, initial treatment of neurogenic shock focuses on fluid resuscitation. Judicious fluid replacement with isotonic crystalloid solution to a maximum of 2 L is the initial treatment of choice. Overzealous crystalloid administration may cause pulmonary edema, because these patients are at risk for the acute respiratory distress syndrome (ARDS).

Once the primary resuscitation is complete and stable, splinting and transport of the injured patient becomes a critical part of avoiding long term disabilities. The current recommendation is that a cervical collar should be put on a suspect cervical injury patient. The head is initially stabilized by the first person on the scene. A collar is then applied with supportive blocks on either side of the head. The head can be immobilized further with adhesive. Sandbags, newspapers and cartons may be used to stabilize the patient's neck though the stability it offers at best is questionable .

While the head is stabilized by the initial person on the scene, the patient should be transferred to a rigid spine board. Transfer can be accomplished either using a log roll with subsequent spine board insertion or a 6-person lift (lift-and-slide) technique. When the patient is seen supine, the 6-person lift is recommended, using a scoop stretcher

placed under the patient, who is lifted about 4 to 6 in (10 to 15cm) while the spine board is slid underneath. The patient is then lowered down onto the spine board and strapped to it. At least 3 straps should be used to secure the torso, pelvis, and legs. The head should be secured with towels, blankets, or commercial head immobilizers and then secured to the board with tape. Once the patient is secured to the spine board and strapped down (body first, followed by the head), transport can begin.

It is unfortunate that in our present setting, most patients are brought to the hospital late and without immobilization. Most of what is described as what should be immediate care or onsite care actually occurs at the emergency room.

Early Acute

Once the spine patient is stabilized and transferred detailed examination can begin. The ASIA impairment scale or the International standards for classification on SCI (ISNICSCI) must be done. This helps because this aids in the communication between the care management team. It can help in predicting prognosis and in the enrollment of clinical trials.

The role of steroids for treating acute spinal cord injury remains controversial. Methylprednisolone sodium succinate (MPSS) works by up-regulating the anti-inflammatory cytokines. The net decrease in inflammation decreases the “second hit” effect. This has been proven in animal studies. The human trials however have mixed results. As of this writing MPSS is not recommended in most protocols. (7-10)

Blood pressure augmentation can be achieved by using vasopressors. An arterial line is required for continuous monitoring. The target of maintaining the mean arterial pressure (MAP) >85 mmHg has been shown to improve outcome.(11) The increase in pressure seeks to decrease ischemia in the damaged structures and increase perfusion to the high risk areas.(12, 13)

Early surgical decompression of ongoing spinal cord compression has been proven to positively affect outcome.(14) Retrospective studies showed early surgery to improve neurologic recovery with no increase in complications if compared to late surgery. (14-17) Specifically, for cervical dislocation, early and rapid close reduction of the spine with traction is imperative. Surgery follows reduction to regain spinal stability. Early stabilization of an unstable spine also aids in the early mobilization of the patients.

Deep venous thrombosis has been reported to be highest in the spinal cord injury patient. Physical interventions like compressive stockings and early mobilization may not be feasible in the multiply injured patient. The timing for the pharmacologic treatment remains controversial.

Subacute-Chronic

The increase in life expectancy among SCI patients have been largely attributed to the prevention of complications. In the subacute phase, focus should be on the preservation/maximization of function and prevention of complications. Early mobilization seeks to maintain what function remains. It can also help prevent the deleterious effects of prolonged bed rest.

Autonomic dysreflexia is a potentially lethal complication of SCI in lesions at or above T6. This unopposed hypertension will cause a cascade of autonomic disarray.(18) It presents as an imbalance of sympathetic tone causing hypertension, flushing of the skin above the lesion and ironically at times, bradycardia. Autonomic dysreflexia can be prevented by searching for possible noxious stimuli that will incite the event. Common examples are pressure sores, urinary tract infection and bladder distention.

Orthostatic hypotension due loss of vascular tone because of decrease sympathetic regulation, reflex vasoconstriction and upregulation of nitric oxide may also be observed in these patients.(19) Midodrine, volume expanders, and salts may be used to increase peripheral tone.(18) At the onset, hypotensive episodes may occur more frequently and may gradually improve over time.

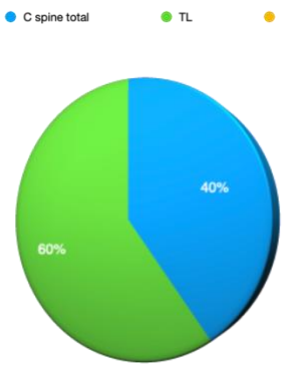
Inability to urinate or prolonged catheter use can cause complications. In spite of all the advances in modern medicine it remains as the second most common cause of death among the SCI patients. A urinary tract infection is defined as “as a colony count of 105 colony forming units per milliliter or greater, with a fever of 38° and two symptoms including, over distention of the bladder, lower abdominal pain, increased urinary incontinence, increased spasticity, autonomic hyperreflexia, and increased sweating or malaise”. Present strategies include clean catheterization programs, using of different maneuvers to promote voiding (eg Crede's maneuver) and regular monitoring. Catheter dependency is common and use of prophylactic antibiotics may increase the chance of having resistant bacteria and worse, prostatitis, epididymitis, urethritis.(20) Intermittent catheterization is preferred as it reduces the risk of infections. Sacral nerve stimulation and anticholinergics (oxybutynin hydrochloride) may be used for neurogenic detrusor muscle over-activity. (21, 22)

Insensate skin can present with life threatening complications. A pressure ulcer is defined as “a localized area of tissue necrosis that tends to develop when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time." Pressure ulcers occur in about a third of SCI patients in their lifetime. Regular wound inspection and prevention interventions are useful in preventing this complication. Please see http://sci.washington.edu/info/pamphlets/msktc-pressure_relief.asp.

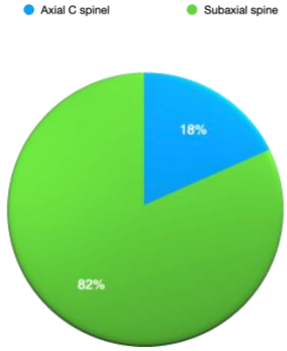
Figure 5. Primary and Seondary Injury to the Spine. Pathophysiology and Management

Quick facts from PGH

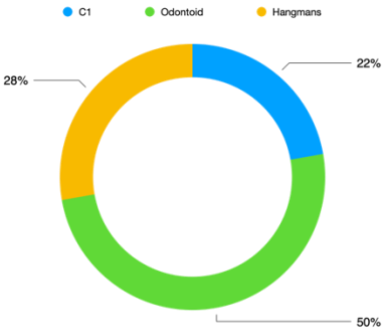
The Philippine General Hospital experience. A 10 year review of the census for the Spine division of the Department of Orthopedics was done. In 10 years, The total trauma cases to the spine was 247. There were 98 documented to have cervical spine trauma and 159 thoracolumbar injuries.



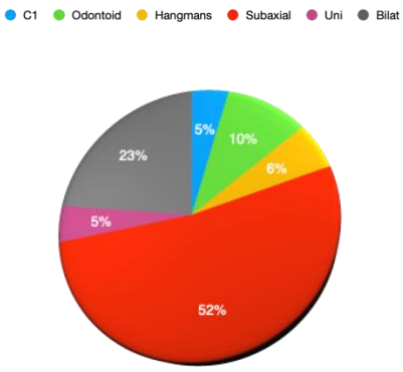
Cervical spine trauma was mostly due to vehicular accidents and falls. From the 98 patients, 18 injuries were at the axial cervical injuries.



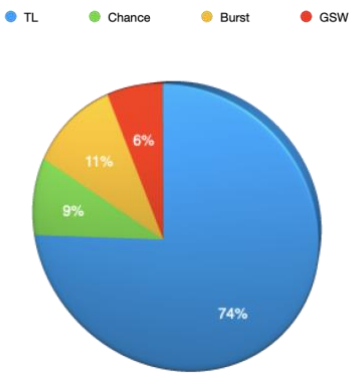
There were 80 subaxial injuries. The 18 injuries at the axial spine were 4 C1 fractures, 9 odontoid fractures and 5 Hangman's fractures.



Majority of the subaxial injuries (n=66) were burst and compression fractures. There were 4 unilateral facet dislocations and 20 bilateral facet dislocations.



For thoracic and lumbar injuries, majority (n=110) were unspecified fractures. There were 14 Chance fractures, 16 burst fractures and 9 gunshot wounds.



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