Spinal Trauma

Spinal stability: is the ability of the spine under physiologic loads to limit displacement. The **three-column concept** is one of the theories of spinal stability and it suggest *that All the fractures and the fracture-dislocations categorized under instability if there is disruption in at least two of the three columns.* These 3 columns are classified as; **(1) Anterior column** includes the anterior longitudinal ligament, anterior part of the intervertebral disk & anterior part of the vertebra (2) **Middle column** includes the posterior part of the disk & the vertebra, the posterior longitudinal ligament and perhaps the pedicle (3) **Posterior column** includes the interspinal, supraspinal ligaments and ligamentum flava and the apophyseal joints.

Clinically vertebral body compressions of more than 50% are commonly considered unstable. Evaluation of the posterior elements should include an over all view of spinal alignment and spinal curves. Increase distance between spinous processes often indicates disruption of the posterior column.

Level of spinal cord injury: The motor part of the spinal segment can be assessed examination of the key muscle groups. The sensation by sensory level

Upper limbs	Lower limbs
Elbow flexors [C5]	Hip flexors [L2]
Wrist extensor [C6]	Knee extensors [L3]
Elbow extensor [C7]	Ankle dorsiflexor [L4]
Finger flexors [C8]	Long toe extensor [L5]
Finger adductors [T1]	Ankle planter flexor [S1]

Completeness of lesion: this includes complete and incomplete lesions of the spinal cord.

(A) **Incomplete lesion:** any residual motor or sensory function more than 3 .segments below the level of the injury

Signs of incomplete lesion include; (1) Sensation (including position sense) or voluntary movement in the lower limbs. (2) Sacral sparing [sensation around the anus, voluntary rectal sphincter contraction or voluntary toe flexion]. (3) An injury does not qualify as incomplete with preserved sacral reflexes alone.

Types of incomplete lesion include

(1)Central cord syndrome: there is a disproportionately grater motor deficit in the upper limbs than in the lower limbs. It is usually results from .hyperextension injury in the presence of osteophytic spurs

(2)Brown-Sequard syndrome (cord hemisection): there are (a) ipsilateral motor paralysis (due to corticospinal tract lesion) and loss of posterior column function (proprioception and vibratory sense) below lesion level, (b) contralateral loss of pain & temperature sensation (spinothalamic tract leasion) .below lesion level. It is usually a result of penetrating trauma

(3) Anterior cord syndrome: there are (a) paraplegia or (if the lesion is higher than C7) quadriplegia. (b) dissociated sensory loss below the lesion (loss of spinothalamic tract function and preservation of posterior column function). It may be results from occlusion of anterior spinal artery or from anterior cord compression (by .(dislocated bone fragment or by traumatic herniated disc
(4) Posterior cord syndrome: produce pain and paresthesias in the neck, upper .arms and torso. It is relatively rare
(5)Conus medullaris syndrome: the conus medullaris is found between the spinal levels ofT11 and L2. There are both upper and lower motor neuron signs in the lower limbs which tend to be symmetrical with flaccid rectal tone and urinary retention
Cauda equine syndrome: is an injury to the nerve roots arising from the conus .medullaris fracture or acute disk herniation) extending from L2 and below
Patient presented with asymmetrical lower motor neuron signs in the lower limbs with loss of bladder and bowel control.
B) Complete lesion: there is no preservation of motor and, or sensory function 3 segments bellow the level of the lesion.

Spinal shock: traumatic injuries to the spinal cord interrupt or temporarily damage a number of descending and ascending pathways. The most common initial presentation of a complete spinal cord injury with respect to reflex and autonomic function is a period of areflexia and flaccidity. This phase is gradually replaced by hypertonia, exaggerated reflexes and in many cases spasticity. The transition may last days to weeks. The immediate onset of hyperreflexia and spasticity is uncommon and their presence is a bad prognostic sign. The period of transition in reflex and .autonomic function is often referred to as spinal shock

Neurogenic shock: is condition characterized by hypotension and bradycardia resulting from interruption of the sympathetic nervous system pathways within the spinal cord. The incidence of significant neurogenic shock increases with injuries above T6 because unopposed vagal tone slows the heart and creates lower systemic .vascular resistance, resulting in venous pooling.
:Primary and Secondary Injury Mechanisms
Primary injury is the damage that results at time of accidence

Mechanical force	**Mechanism of injury**
Impact + Persisting compression	Burst fracture Fracture-dislocation Disk rupture
Impact alone (temporary compression)	Hyperextension
Distraction	Hyperflexion
Laceration, transection	Burst fracture Laminar fracture Fracture-dislocation Missile

Secondary injury is the damage that results from the events of primary injury.

Secondary events	Description
Systemic effects	Neurogenic shock
Damage to the cord microcirculation	Mechanical disruption of capillaries and venules
Biochemical changes	Free radical production and lipid peroxidation
Electrolyte shift	Increased intracellular calcium
Edema	Cytotoxic edema
Loss of energy metabolism	Decrease ATP production

The end result of the secondary events is decreasing local blood supply at site of cord injury which results in further ischemic damage. If these changes are not corrected, the resulting biochemical changes from the infarcted area lead to ischemic changes in the adjacent areas and vicious circle will continue. The successful use of steroids in spinal cord injury is strong evidence for the beneficial interruption or prevention of secondary mechanism of injury, especially those related to lipid peroxidation and the .production of oxygen-free radicles

Prognostic factors for recovery: about 3% of patients with complete injuries on initial exam will develop some recovery within 24 hours. The persistence of a complete spinal cord injury beyond 24 hours indicates that no distal function will recover. In contrast, most patients who enter the hospital with an incomplete neurologic injury attain some degree of recovery. The level and the degree of incomplete injury also provide important prognostic information. Cervical injuries have a higher potential for recovery than do thoracic or thoracolumbar injuries. The less the severe the injury, the more likely it is that the patient will recover. Younger patients fare much better than their older counterparts in terms of regaining neurologic function after spinal cord .injury

Management

The major causes of death in spinal cord injury are aspiration and shock. Management proceeds in association with routine treatment of other injuries:

(A) Management in the field (at time of accident)

(1)Immobilization prior to and during extrication from vehicle and transport to prevent active or passive movements of the spine (place the patient on back-board with sandbags on both sides of the head and 3 inch strip of adhesive tape from one side of the back-board to the other across the forehead immobilizes the spine, a rigid cervical collar may be used as supplement.

(2)Maintain blood pressure (a) pressors (dopamine) (b) fluid as necessary to .(replace losses (c) Military anti-shock trousers (prevent venous pooling).
(3)Maintain oxygenation (by mask or if the patient need intubations; by .nasotracheal method to avoid neck movement.
(4)Brief motor exam: ask patient to (a) move arms (b) move hands (fingers) (c) move leg (d)move toes.

(B) Management in the hospital

(1)Immobilization: maintain backboard / head - strap to facilitate transfers to CT table..etc

(2)Treatment of neurogenic shock (hypotension) by (a) dopamine (b) careful hydration (c) atropine for bradycardia

(3)Maintaining oxygenation
(4)NG tube for suction: prevent vomiting and aspiration, and decompresses abdomen (which can interferes with respirations if distended (paralytic ileus)

(5)Indwelling (Foley) urinary cat
(6)Temperature regulation
(7)(Correction of electrolytes (hypokalemia due to increase aldosterone
(8)More detailed neurological examination.

(9)Radiologic evaluation: **(I) Cervical spine**: AP, open —mouth and lateral X-ray was taken while in rigid collar; if all 7 vertebrae and C7-Tljunction are adequately visualized and are normal, and if the patient has no neck pain and is neurologically intact, then we can remove the collar and no further radiological exam is needed. But, if the patient had neurological deficit and was complaining from neck pain or tenderness or if there are abnormal X-ray findings then further studied are needed (oblique for cervical spine X-ray, flexion-extension views, CT scan or MRI).
(II) Thoracic and lumbosacral spine AP and lateral X-ray view for all trauma patients with RTA, complaining of back pain or unconscious. If there are positive X-ray finding or if there is neurological deficit MRI of the spine is indicated

(10)Medical treatment specific to spinal cord injury: using methylprednisolone within first 8 hours of injury to counteract secondary phenomena especially those related to lipid peroxidation and production of oxygen-free radicals(methylprednisolone 30mg/kg bolus dose infusion in first quarter of an hour,thene a gap three quarter of an hour followed by maintenance of 5.4mg/kg/h in the next 23 hour. No role to give methyprdnisolone after 8 hour from the time of injury.

(11)Surgical treatment: surgery is indicated for (a) deformity correction (b) stabilization of the spine and (c) decompression of the neurological elements.