Management of Acute Spinal Cord Injury

Shawn Song, MD
SCI Fellow
Department of Rehabilitation Medicine
University of Washington/VA Puget Sound

PMR Review Course: March 2015

Topics to Cover

• In the field:
  – Immobilization
• In the ER/ICU:
  – Neurogenic and spinal shock
  – Neuroprotection
  – Diagnostic imaging
  – Clinical exam
  – Timing of surgery

Mr. MC: At the Scene

• 25 y/o M involved in motocross accident.
  – (+) LOC per bystanders, EMS called.
  – No spontaneous movement of UE/LE’s observed by EMS.
  – Breathing spontaneously.
  – Suspected R humeral fx.

• Medical issues in ICU/floor:
  – Neurological comorbidities
  – MSK
  – CV
  – Respiratory
  – Heme: DVT/PE prophylaxis
  – Skin
  – Bowel/bladder
  – Endocrine: Hypercalcemia
Immobilization

• **Who?** Any patient with potential SCI (AMS, intoxicated, suspected fx or distracting injury, focal neuro deficit, spinal pain).
• **How?** Combination of rigid cervical collar with supportive blocks on a backboard with straps to secure entire spine.
• **Until?** Definitive treatment.

Mr. MC: Field → ER

• Patient immobilized and transported to HMC with suspected SCI.
• In the ER:
  – Patient noted to be hypotensive to 80/50’s, bradycardic to 40’s with T of 96.2 F.
  – Breathing spontaneously with good sats (for now).
• **Next steps?**
  – CV stabilization
  – Imaging
  – Neuroprotection
  – Surgery
  – Clinical exam

Neurogenic Shock

• **Definition:** Triad of hypotension, bradycardia and hypothermia.
• **Pathophysiology:** Secondary to loss of sympathetic activity below lesion.
  – Loss of vascular tone → blood pools in venous compartment.
  – Loss of sympathetic input to heart (T1-T4) results in unopposed vagal tone → bradycardia and reduced myocardial contractility.

Treatment of Hypotension

• **Aggressive fluid resuscitation.**
• **Vasopressors with both α- and β-adrenergic actions to counter the loss of sympathetic tone AND provide chronotropic support to the heart (dopamine, NE, PE).**
Bradycardia

- Due to unopposed vagal tone.
- Stimulation of vagally innervated tissue can further decrease HR (tracheal suctioning, defecation).
- Most pronounced 2-3 weeks post-injury.
- Treatment:
  - Mostly education, atropine IV prior to suctioning.
  - Transvenous pacing, pacemaker rarely necessary.

Temperature Dysregulation

- Secondary to inability to redirect blood from periphery to core → excessive heat loss/gain.
- Results in poikilothermia (body assumes temperature of ambient environment).
- Be aware of environmental temp, facilitate cooling/warming.

Treatment of Neurogenic Shock

- **Goal:** Maintain spinal cord perfusion.
- Uncontrolled studies suggest improved outcomes with maintenance of MAP > 85 for 7 days post-SCI.
- AANS/CNS guidelines recommend maintenance of MAP between 85-90.

Spinal vs. Neurogenic Shock

- Spinal shock:
  - **Definition:** Loss of sensory, motor, and reflex function of the SC below lesion.
- Neurogenic shock:
  - *The cardiovascular manifestation of spinal shock.*
Stages of Spinal Shock

- **Stages**:
  - Phase I (0-1 day post-injury): Flaccid paralysis with absent DTR's. Abnormal reflexes may be present.
  - Phase II (1-3 days post-injury): DTR's still absent, but abnormal reflexes become stronger.
  - Phase III (4 days – 1 month post-injury): Early hyper-reflexia. DTR's return, bradyarrhythmias and hypotension usually improve, AD can emerge.
  - Phase IV (1-12 months post-injury): Hyper-reflexia/spasticity emerge. AD can become stronger.


Neuroprotection: Steroids

- Administration of methylprednisolone for the treatment of acute SCI is **NOT** recommended.
- There is NO Class I/II medical evidence supporting clinical benefit.
- There IS Class I/II/III evidence that high-dose steroids are associated with SE’s.


Neuroprotection: Cooling

- **Theory**: Hypothermia leads to ↓ apoptosis, mitochondrial dysfunction, metabolic demand, cell membrane injury, inflammation.
- **Evidence**:
  - Two recent small studies have shown improved rates of conversion from complete to incomplete injuries at 1 year after injury (43% conversion rate) following cooling in the acute period.


Diagnostic Imaging

- Image the entire spine as ~20 % pts will have non-continuous spine fx.
- CT is preferred modality.
- 3v XR of entire spine, if CT unavailable.
  - To image C1-C2: Open mouth view.
  - To image C7-T1: Swimmer’s view.
- Perform MRI of known areas of SCI.
Neurologic Exam
- ASIA exam should be performed and documented ASAP and after any surgery.
  - Ideally, daily x 3d to monitor progression of deficits.
  - Prognostic value.
- Also make note of reflexes (DTRs, DPR, BC) as these can indicate the stage of spinal shock.

Timing of Surgery
- Paucity of literature to determine if early (<12h) versus late decompression is beneficial for recovery.
- “Consider early surgical spinal canal decompression in the setting of a deteriorating SCI as a practice option that may improve neurological recovery.”


Mr. MC: ER → ICU
- MAP>85 after IVF and dopamine gtt.
- NOT given methylprednisolone.
- CT reveals b/l C4-5 jumped facets, MRI shows increased T2 signal with punctate hemorrhage at level of C4-5.
- Initial ASIA exam reveals C4 AIS A level.
- Taken to OR for C4-5 lami, C3-C6 PSIF <24hrs after injury, admitted to neuro ICU post-op.

Mr. MC: ICU (HD#3)
- Confused, but appropriate. Still in PTA.
- BP in 100/60's, HR in 70's. Has not been mobilized out of bed yet.
- Extubated with good O2 saturation.
- Has non-blanching erythema over sacrum.
- Foley remains in, absent BCR on exam.
- No BM's since admission.
- Rehab consult obtained by ICU team (finally!)
Medical problem list

- Neurological
- MSK
- CV
- Respiratory
- Heme: DVT/PE prophylaxis
- Skin
- Bowel/bladder
- Endocrine: Hypercalcemia

Neurological complications: TBI

- Screen for TBI:
  - Incidence of concomitant SCI/TBI: 25-59%.
  - Evaluate problem solving, attention, and memory skills.
  - Integrate Speech/Language Pathology and/or SCI Psychology during early.

Prognosis in SCI+TBI

- Evidence is mixed:
  - Several studies show worse functional outcomes with dual diagnosis.1,3,4
  - Newer study shows equivalent functional outcomes.2
  - Multiple studies show longer LOS during acute rehab in patients with dual diagnosis.2,3,4

MSK Complications

- 64% of SCI pts have concomitant injuries.
  - Bony fractures/dislocations.
  - Muscle/nerve injuries.
  - Amputations.
  - Skin compromise: burns, open wounds.

Orthostatic Hypotension

- ↓ SBP >/= 20mmHg, ↓ DBP >/= 10mmHg upon assumption of upright position.
- Due to loss of vasomotor tone and expanded vascular bed.
- Can contribute to secondary neurologic injury and complicate/delay rehab.
- Incidence of 60% during 1st month post-injury.
- Most common in patients with motor complete cervical SCI.


Orthostatic Hypotension: Treatment

- Slow mobilization in reclining w/c or tilt table
- Compressive devices: abdominal binder, elastic stockings
- Medications: Midodrine, fludrocortisone.
- Very limited evidence for pharmacologic and non-pharmacologic interventions (but we use them anyways).


Autonomic Dysreflexia

- Bradycardia and neurogenic shock common.
- Autonomic dysreflexia less common in the acute period.
  - Requires intact spinal reflexes and will usually not be seen until later stages of spinal shock.

Pathophysiology of Respiratory Dysfunction in Acute SCI

- Inspiratory muscle weakness
- Expiratory muscle weakness
Inspiratory Muscles

- Diaphragm:
  - Innervated by C3-C5 via the phrenic nerve.
  - Provides 60% of vital capacity.

Accessory Inspiratory Muscles

- Stabilize chest wall during inspiration:
  - Sternocleidomastoid (C1-2).
  - Scalenes (C4-8).
  - Pec major (C7-T1).

Consequences of Weak Inspiratory Muscles

- Injury below C3 leads to flaccid paralysis of accessory muscles acutely.
  - With loss of chest wall stabilization, contraction of diaphragm causes chest wall to contract rather than expand.
  - Max inspiratory force can decrease by 70%.
- Abd musculature paralysis also places diaphragm at mechanical disadvantage.

Consequences of Weak Inspiratory Muscles

- Inadequate lung expansion, leading to:
  - Reduced compliance (stiff lungs) within hours of injury.
  - Atelectasis.
  - Ventilatory failure, immediate or delayed.
Expiratory Muscles

- Expiration is passive: diaphragm relaxes.
- Active expiration is needed for coughing.
- Thoracic innervation: weak in all tetraplegic patients, leading to impaired cough:
  - Internal intercostals (T1-T8)
  - Abdominals (T7-T12)

Bronchial Hyper-responsiveness

- Increased bronchial responsiveness and reversible airflow limitation seen in tetraplegics.
  - Reflects unopposed vagal tone to bronchioles mediating bronchostriction.
  - Treated in the acute period with anticholinergic agents (ipratropium) and beta-agonists (metaproterenol).

Respiratory complications

- Atelectasis, PNA, and respiratory failure are the most common complications.
  - Secondary to inability to clear secretions and adequately ventilate lungs.
- 84% incidence of any complication with C1-C4 level of injury.
- 60% incidence with lower injury levels (C5-8 and thoracic levels).

Techniques to Clear Secretions

- Manually assisted coughing (“quad cough, tussive squeeze”)
- Deep breathing and coughing
- Glossoharyngeal breathing
- Positioning (supine, Trendelenberg)
- Chest PT
- Mechanical in-exsufflator
- Inspiratory spirometry
- CPAP, BiPAP
- Intermittent positive pressure breathing (IPPB) “stretch”
- Intrapulmonary percussive ventilation (IPV)
- Bronchoscopy

Mr. MC: ICU (HD#3)

- During rehab consult, you notice RR has increased from 15 to 25 in past 12 hrs.
- Patient with mild difficulty with full sentences.
- CXR with atelectasis.
- You ask ICU team for ABG.

Signs of Impending Respiratory Failure

- ↑ Anxiety
- SOB
- ↑ Temp
- Change in RR
- ↑HR
- Change in secretions
- ↓ VC
- ↓ Peak expiratory flow rate (esp with cough)

Respiratory Failure

- Definition: $pO_2 < 50$ or $pCO_2 > 50$ on ABG.
- Onset at time of injury or develops over first few days post-injury (usually 3-4 days).
  - Overall, 75% of tetraplegics require mechanical ventilation in the acute phase.
- Vital capacity and ABG should be monitored frequently.

### Consider Intubation if...

- $pO_2 < 50$ or $pCO_2 > 50$ on ABG.
- $VC < 10-15cc/kg$ of ideal body weight (or downtrending).
- Intractable atelectasis on serial CXR.
- PNA.
- Deteriorating vitals, peak expiratory flow rate, FEV$_1$, or NIF.

### Mechanical Ventilation Protocol

- Consider use of protocol that includes ↑ TV to resolve or prevent atelectasis:
  - Initial setting of 12-15ml/kg.
  - Not for use with acute lung injury or ARDS.
  - Has been shown to reduce incidence of atelectasis and ↓ weaning times.

*Guidelines for ventilation available in Pulmonary Management of SCI chapter, Kirshblum, Spinal Cord Medicine.*

### Weaning

- Consider using progressive ventilator-free breathing (PVFB) over synchronized intermittent mandatory ventilation (SIMV)
  - Small studies show that PVFB provides a greater chance for successful weaning compared to SIMV among C3 and C4 tetraplegics.

*PVFB protocol available in Pulmonary Management of SCI chapter, Kirshblum, Spinal Cord Medicine.*

### Tracheostomy

- Common in cervical SCI:
  - 75% in complete cervical SCI.
  - 50% in incomplete cervical SCI.
- Risk factors: AIS A injury, age>45 yrs, comorbid lung disease, smoking history.
- Early tracheostomy (<7d) may be associated with quicker extubation and shorter LOS.

DVT in Acute SCI

- High incidence in acute SCI: 18-100%.
  - Historically, PE is 3rd most common cause of death in acute SCI.
  - Significantly ↓ incidence with ppx.
- Highest risk within first 2 weeks post-injury (but rare <72 hours post-injury).
- Overall risk declines after 8 weeks (<6%).

Risk Factors of DVT in SCI

- Motor complete > incomplete.
- Paraplegia > tetraplegia.
- Male > female.
- Unaffected by age, except >60 y/o have higher rates of pulmonary embolism (PE).

Pathophysiology of DVT

- Due to paralysis and loss of venous muscle pumping
- Reduced fibrinolytic activity and ↑ factor VIII activity

DVT Prophylaxis

- SCDs in first 2 weeks.
  - If not placed within 72hrs of injury, then screen for DVT.
- LMWH (dalteparin 5000U SC qd) started within 72hrs is 1º ppx.
  - If no active bleeding, coagulopathy, or e/o head injury.
### Duration of DVT Prophylaxis

<table>
<thead>
<tr>
<th>Motor incomplete</th>
<th>Treatment</th>
<th>Duration of ppx</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>UH 5000 units SC q12h or LMWH</td>
<td>ASIA C x 8 weeks, ASIA D until ambulating or at d/c</td>
</tr>
<tr>
<td>Motor complete – uncomplicated</td>
<td>LMWH</td>
<td>8 wks</td>
</tr>
<tr>
<td>Motor complete – complicated (LE fx, &gt;70 y/o, obesity, prior thrombosis, heart failure, cancer)</td>
<td>LMWH</td>
<td>12 wks</td>
</tr>
</tbody>
</table>

### DVT: IVC Filters

- Placement indicated if there is a contraindication to anticoagulation.
  - If contraindication is temporary, consider temporary filter.
- No evidence to support routine placement of IVC filters.
- Filter complications: Migration, perforation, cough-assist restrictions, lifetime anticoagulation, ↑ risk of DVT?

### DVT Treatment in SCI

- LMWH as effective as IV unfractionated heparin.
  - Dalteparin 200 units/kg/day SC, max 18,000 units/dose.
  - Overlap with warfarin until INR 2-3 x 48 hours.

### Duration of DVT Treatment

- Duration of treatment:
  - Initial DVT: 3 months.
  - Initial PE: 6 months.
  - Recurrent DVT: 6-12 months.
  - Recurrent PE: Lifetime.
Neurogenic Skin

- **Mechanism of injury:** Local soft tissue ischemia due to prolonged and increased pressure over bony prominences.
  - 70 mmHg pressure continuously over 2 hrs results in tissue damage.
- **Incidence:** 36.5% during acute rehab.
  - Most common locations were sacrum (43%), heel (19%), and ischium (15%).


Pressure Ulcers: Deep Tissue Injury

Discolored intact skin due to damage of underlying soft tissue.

Pressure Ulcers: Stage I

Non-blanchable erythema.

Pressure Ulcers: Stage II

Partial thickness loss of dermis.
Pressure Ulcers: Stage III

Full thickness loss of dermis.

Pressure Ulcers: Stage IV

Full thickness loss of dermis with exposed bone, tendon, or muscle.

Pressure Ulcers: Unstageable

Full thickness loss of dermis with unknown depth due to slough or eschar.

Prevention of Pressure Ulcers

- Protect in transport and handling:
  - Immobilization, traction, restricted positioning.
- Assess skin at risk regularly.
- Place pt on pressure reduction mattress.
- Rotating bed if spine unstable (but assess positioning in bed).
- Turn q2 hrs when stabilized.
- Maintain area under patient clean and dry.
- Assess nutritional status.
- Early ulcers may delay acute rehab!
New Pressure Ulcer Resource

“Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline.”
Consortium for SCI Medicine, 2014

Bowel Management

• Initiate appropriate, regularly scheduled bowel program.
  – PO/PR meds.
  – +/- digital stimulation.
  – Staff training!

Bladder Management

• Foley catheter during acute period:
  – Allows for adequate hydration.
  – Preferable to intermittent catheterization during the acute phase of injury.
  – Prophylactic antibiotics for UTI prevention not recommended.

Hypercaldemia of Immobilization

• Pathophysiology: Immobilization after SCI triggers ↑ bone resorption and ↓ PTH.
• Incidence: 24% of children and young adults with SCI.
  – Most commonly seen 4-8 weeks after injury.
• Risk factors:
  – Male sex, adolescence, motor complete injury, higher neurologic level, longer immobilization.
Treatment of Hypercalcemia

- Similar to hypercalcemia of other causes:
  - Hydration
  - Diuretics
  - Calcitonin
  - Bisphosphonates

- Most patients respond to single dose of pamidronate 60mg IV.


Thanks for your attention!!!

Questions?

Resources


Practice Question #1

1. Which treatment is NOT standard management for acute care of spinal cord injury?
   a) Prophylaxis for venous thromboembolism within 72 hours.
   b) Volume resuscitation to optimize spinal cord perfusion.
   c) High-dose methylprednisolone as a neuroprotective agent.
   d) Assessment and treatment of spinal and neurogenic shock.
Answer
c) The Consortium for Spinal Cord Injury Medicine Clinical Practice Guidelines consider the use of high-dose methylprednisolone to be a treatment option rather than a standard. The effectiveness of high-dose methylprednisolone as a neuroprotective agent has been questioned, and it has been associated with increased risk of infection, gastrointestinal bleeding, and steroid myopathy.

Practice Question #2
2. Regarding spinal shock in acute SCI:
   a) Duration of spinal shock is correlated with long-term outcome.
   b) Reflex activity typically returns over the course of days.
   c) A reliable ASIA classification can be performed during spinal shock.
   d) It is more common in tetraplegia than in paraplegia.

Answer
c) Spinal shock is a condition in which upper motor neuron sensory motor loss is associated with areflexia below the level of injury. It is a poorly defined phenomenon. Reflex activity can often be detected by electrophysiologic study when it is not clinically apparent. Reflex activity typically returns over the course of weeks or months. The presence of spinal shock is of marginal prognostic significance. A reliable ASIA classification can be carried out when spinal shock is present.

Practice Question #3
3. Functional outcomes after the use of methylprednisolone in persons with penetrating spinal cord injury as compared with blunt injury are:
   a) Markedly improved.
   b) Better.
   c) Unchanged.
   d) Worse.
**Practice Question #4**

4. You are called to the neuro ICU to evaluate a patient with new SCI; you determine that the patient has sustained a C7 ASIA A spinal cord injury. Which change in the respiratory system would be expected?

a) Residual volume will decline to 30% of predicted value.
b) Pulmonary function will not improve after the first 2 weeks post-injury.
c) Expiratory reserve volume increases 40% 6 weeks post-injury.
d) Vital capacity of 60% predicted value may be obtained within the first 6 months post-injury.

**Practice Question #5**

5. A 22-year-old woman with a C5 ASIA class A spinal cord injury sustained in a car crash 2 weeks ago complains of lightheadedness, dizziness, and nausea during her physical therapy session. In response to her therapist’s call, you recommend:

a) Sitting the patient up and loosening tight garments.
b) Placing the patient in Trendelenburg position.
c) Using elastic abdominal binders and elastic stockings.
d) Adjustment of HALO vest.
c) Orthostatic hypotension (OH) is a decrease in blood pressure that results from a change in body position toward the upright posture. Symptoms include lightheadedness, dizziness, nausea. This form of hypotension is most likely to occur in persons with high levels of injury. Treatment involves daily tilting with gradual change to upright posture. Elastic binders help compress the abdomen, thus limiting blood accumulation in the abdominal vasculature. Elastic stockings limit blood accumulation in lower extremities. Patients must be adequately hydrated. Salt tablets, 1 gram 4 times daily, ephedrine, 20–30mg up to 4 times daily, Florinef, and Midodrine may be used as pharmacologic adjuncts.