Neurologic and Neurosurgical Emergencies in the ICU
Overview

- Review definitions of altered consciousness/coma
- Review cranial nerve exam for mid/hind brain injury
- Review physiology of intracranial pressure and basis for monitoring
- Review Dx and Tx of Status epilepticus
- Acute stroke intervention
- Subarachnoid hemorrhage
Altered Consciousness and Coma

Consciousness requires arousal (coming from the brainstem reticular formation) and content (the cerebral hemispheres).

Alterations in consciousness stem from:
- Disorders affecting the reticular formation
- Disorders affecting both cerebral hemispheres
- Disorders affecting the connections between the brainstem and the hemispheres
Altered Consciousness and Coma

Definitions

- Delirium: classically, altered awareness with motor and sympathetic hyperactivity, often with sleeplessness, hallucinations, and delusions
  - More recently used to describe any acute change in consciousness short of coma, as a synonym for encephalopathy
- Obtundation: the patient appears to sleep much of the day but has some spontaneous arousals
Altered Consciousness and Coma

- **Stupor:** the patient lies motionless unless aroused but will awaken with stimulation; localizes or withdraws from noxious stimuli

- **Coma:** the patient makes no understandable response to stimulation but may display abnormal flexor (decorticate) or extensor (decerebrate) posturing
Altered Consciousness and Coma

- Examining the patient with altered consciousness:
  - ABCs - insure adequate oxygenation and blood pressure before proceeding
  - Be certain that the blood glucose is at least normal
  - If there is any reason to suspect thiamine deficiency, administer 100 mg thiamine IV
Altered Consciousness and Coma

The purpose of the coma examination is to determine whether the upper brainstem is functioning.

- Brainstem dysfunction means immediate imaging.
- Bilateral hemispherical dysfunction leads initially to metabolic or toxic diagnoses.

Four domains to examine:
- Pupillary responses
- Extraocular movements
- Respiratory pattern
- Motor responses
Parasympathetic control of pupil size
Sympathetic control of pupil size
Control of Horizontal Eye Movements

- Neck stretch receptors
- III
- VI
- VIII
- MLF
- Neck stretch receptors
- III
- VI
- VIII
Spontaneous horizontal conjugate eye movements prove that the brainstem centers for eye movement are intact.
- These overlap the portion of the reticular formation necessary for consciousness.
- Therefore, coma in a patient with roving horizontal conjugate eye movements is not due to brainstem dysfunction.
If there are no spontaneous eye movements, attempt to trigger them.

- In the absence of cervical spine disease, test cervico-ocular reflexes ("dolls’ eyes"):  
  - Turning the head to the right should cause the eyes to go left, and vice versa.  
  - Same meaning as spontaneous movements regarding the brain stem  
  - Partial responses mean a problem involving the brainstem or cranial nerves (use the diagram to determine where the problem lies).
Vestibulo-ocular testing (“cold calorics”)

- Check for tympanic membrane perforation first
- 50 - 60 mL ice water in one extra-ocular canal using soft tubing (e.g., from a butterfly; do not use an IV catheter, which can penetrate the tympanic membrane)
- Tonic deviation of both eyes toward cold ear indicates intact brainstem function.
- Wait for one ear to warm up before testing the other ear.
Assessing Eye Movements

Nystagmus away from the cold ear is due to cortical correction of the brainstem-induced eye movement and means the patient is not comatose.
Respiratory Patterns in Coma

- Cheyne – Stokes respiration: bilateral hemispheral dysfunction
- Central reflex hyperpnea: midbrain dysfunction causing neurogenic pulmonary edema
  - rarely see true central neurogenic hyperventilation with this lesion; central hyperventilation is common with increased ICP
Respiratory Patterns in Coma

- Apneustic respiration (inspiratory cramp lasting up to 30 sec): pontine lesion
- Cluster breathing (Biot breathing): pontine lesion
- Ataxic respiration: pontomedullary junction lesion
Motor Responses

- Defensive, avoidance, or withdrawal - indicative of cortical function (the patient is not comatose)
- Flexor (decorticate) posturing - the cortex is not in control of the spinal cord, but the midbrain (red nucleus) is
- Extensor (decerebrate) posturing - the midbrain is not in control but the pontomedullary region (vestibular nuclei) is
- Going from flexion to extension indicates worsening; extension to flexion, improvement
Increased Intracranial Pressure

- The volume of the skull is a constant (Monro-Kellie hypothesis) which contains:
  - Brain
  - Blood
  - CSF
- An increase in the volume of any of these or the introduction of alien tissue (e.g., tumor) will raise ICP.
Increased Intracranial Pressure

- Initially, the ICP rises slowly as volume is added (CSF and then blood exits the skull)

- But as the volume increases to rise, compliance worsens and the pressure rises rapidly:
  - This impairs arterial blood flow, producing ischemia
  - Focal increases in volume also cause herniation from high pressure compartments to lower pressure ones.
    - Cerebral Perfusion Pressure = MAP – ICP (or CVP)
      - Keep CPP > 60, ICP < 20
      - Keep Brain O2 (discussed later) > 20
Increased Intracranial Pressure

- The standard theory of coma due to rostro-caudal brainstem movement has been supplanted by Ropper’s lateral shift theory.
- Shift is often heralded by a third cranial nerve palsy (usually causing a dilated pupil before failure of extra-ocular movements).
Herniation
Standard Model

Inferred force vector causing transtentorial herniation

diencephalon

midbrain

pons

uncus

midline

temporal lobe
cavernous sinuses

third cranial nerves

third nerve palsy from compression

midbrain

cistern obliterated

temporal lobe

uncus

Standard Model
Current Model

Force vector displacing diencephalon laterally

diencephalon
midbrain
pons
temporal lobe
uncus
cistern widened
midline

Society of Critical Care Medicine
Current Model

- Cavernous sinuses
- Third cranial nerves
- Third nerve palsy from stretch
- Cistern widened
- Midline
- Uncus
- Temporal lobe
brain abscess

enlarged cistern

Bleck et al, 2000
Coma Scales

- Standard: Glasgow coma scale (GCS)
<table>
<thead>
<tr>
<th>Adult</th>
<th>Pediatric</th>
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<tbody>
<tr>
<td><strong>Eye opening</strong></td>
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<tr>
<td>Spontaneous 4</td>
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<tr>
<td>Reaction to speech 3</td>
<td>Reaction to speech 3</td>
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<tr>
<td>Reaction to pain 2</td>
<td>Reaction to pain 2</td>
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<tr>
<td>No response 1</td>
<td>No response 1</td>
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## Adult and Pediatric GCS

<table>
<thead>
<tr>
<th>Adult</th>
<th>Pediatric</th>
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<tr>
<td><strong>Best motor response</strong></td>
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<td>Flexion 3</td>
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<tr>
<td>Extension 2</td>
<td>Extension 2</td>
</tr>
<tr>
<td>No response 1</td>
<td>No response 1</td>
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</table>
## Adult and Pediatric GCS

### Adult Verbal

<table>
<thead>
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<th>Score</th>
<th>Description</th>
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<tbody>
<tr>
<td>5</td>
<td>Oriented</td>
</tr>
<tr>
<td>4</td>
<td>Confused</td>
</tr>
<tr>
<td>3</td>
<td>Inappropriate words</td>
</tr>
<tr>
<td>2</td>
<td>Incomprehensible sounds</td>
</tr>
<tr>
<td>1</td>
<td>No response</td>
</tr>
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</table>
Monitoring Sedation Status over Time in ICU Patients: Reliability and Validity of the Richmond Agitation-Sedation Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Combative</td>
<td>Overtly combative, violent, immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Very agitated</td>
<td>Pulls or removes tube(s) or catheter(s); aggressive</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated</td>
<td>Frequent nonpurposeful movement, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Restless</td>
<td>Anxious but movements not aggressive or vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>Drowsy</td>
<td>Not fully alert, but has sustained awakening</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(eye opening/eye contact) to voice (&gt;10 seconds)</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation</td>
<td>Briefly awakens with eye contact to voice (&lt;10 seconds)</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation</td>
<td>Movement or eye opening to voice (but no eye contact)</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation</td>
<td>No response to voice, but movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable</td>
<td>No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>

Verbal stimulation

Physical stimulation

Procedure for RASS Assessment
1. Observe patient
   - Patient is alert, restless, or agitated. Score 0 to +4
2. If not alert, state patient’s name and say to open eyes and look at speaker.
   - Patient awakens with sustained eye opening and eye contact. Score -1
   - Patient awakens with eye opening and eye contact, but not sustained. Score -2
   - Patient has any movement in response to voice but no eye contact. Score -3
3. When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
   - Patient has any movement to physical stimulation. Score -4
   - Patient has no response to any stimulation. Score -5
Increased Intracranial Pressure

Management

• Make plans to correct the underlying pathophysiology if possible.
• Airway control and prevention of hypercapnea are crucial:
  - When intubating patients with elevated ICP use thiopental, etomidate, or intravenous lidocaine to blunt the increase in ICP associated with laryngoscopy and tube passage.
• ICP monitoring usually needed to guide therapy
• Cortical Oximetry (Lycox catheter) may be beneficial
  • Measures O2 tension in parenchyma in area of catheter
  • Keep Brain O2 > 20
Increased Intracranial Pressure

Posture and head position
- Avoid jugular vein compression
  - Head should be in neutral position
  - Cervical collars should not be too tight
- Elevation of the head and trunk may improve jugular venous return.
Hyperventilation (PaCO$_2$ 30-35 mmHg) works by decreasing blood flow and should be reserved for emergency treatment and only for brief periods.

- The major determinant of arteriolar caliber is the extracellular pH, not actually the PaCO$_2$, but this is the parameter we can control.
Intact Auto-regulation

![Graph showing cerebral blood flow vs. arterial blood pressure in the zone of autoregulation with pressures for passive dilatation and constriction.]

Defective Auto-regulation

Cerebral blood volume compartment

Arterial blood pressure (mm Hg)

Cerebral blood flow (ml/100 g/min)
Increased Intracranial Pressure

- Pharmacologic options
  - Mannitol 0.25 gm/kg q4h (may need to increase dose over time)
  - Hypertonic saline (requires central line)
    - 3%
    - 7.5%
    - 23.4% (30 mL over 10 min)
  - Steroids only for edema around tumors or abscesses (not for use in trauma or cerebrovascular disease)
**Increased Intracranial Pressure**

**Sedation**
- Benzodiazepines
- Propofol
- Barbiturates

Works by decreasing cerebral metabolic rate, which is coupled to blood flow
- Requires autoregulation, which often fails in patients with elevated ICP
- Often causes a drop in MAP, impairing cerebral perfusion and thus requiring vasopressors (e.g., norepinephrine)
Increased Intracranial Pressure

- High-dose barbiturates
  - E.g., pentobarbital 5 – 12 mg/kg load followed by infusion to control ICP
  - Very rarely needed
Increased Intracranial Pressure

Surgical options

- Resect mass lesions if possible
- Craniectomy
  - Lateral for focal lesions
  - Bifrontal (Kjellberg) for diffuse swelling
Classification of Neurogenic Respiratory Failure

- **Oxygenation failure** (low PaO₂)
  - primary difficulty with gas transport
  - usually reflects pulmonary parenchymal disease, V/Q mismatch, or shunting

- Primary neurologic cause is neurogenic pulmonary edema.
Neurogenic Pulmonary Edema

A state of increased lung water (interstitial and sometimes alveolar):
• as a consequence of acute nervous system disease
• in the absence of
  - cardiac disorders (CHF),
  - pulmonary disorders (ARDS), or
  - hypervolemia
Causes of Neurogenic Pulmonary Edema

**Common**
- SAH
- head trauma
- intracerebral hemorrhage
- seizures or status epilepticus

**Rare**
- medullary tumors
- multiple sclerosis
- spinal cord infarction
- Guillain-Barré syndrome
- miscellaneous conditions causing
- intracranial hypertension
- many case reports of other conditions
Classification of Neurogenic Respiratory Failure

Ventilatory failure (inadequate minute ventilation [VE] for the volume of CO₂ produced):

- In central respiratory failure, the brainstem response to CO₂ is inadequate, and the PaCO₂ begins to rise early.
- In neuromuscular ventilatory failure, the tidal volume begins to fall, and the PaCO₂ is initially normal (or low).
Causes of Neurogenic Ventilatory Failure

Most common causes are:
- Myasthenia gravis
- Guillain-Barré syndrome
- Critical illness polyneuropathy, myopathy
- Cervical spine disease

Many rarer causes
Management of Neurogenic Ventilatory Failure

Airway protection and mechanical ventilation
- Don’t wait for the PaCO₂ to rise

Specific therapies
- Myasthenia: IgIV, plasma exchange
- Guillain-Barré: plasma exchange, IgIV
- Critical illness polyneuropathy, myopathy: time
Status Epilepticus

Types of status epilepticus:
  • Convulsive
  • Nonconvulsive
Status Epilepticus

Definition
• Typically diagnosed after 30 min of either:
  - Continuous seizure activity
  - Intermittent seizures without recovery between
• **Don’t wait for 30 min to treat:**
  - Seizures become more difficult to treat the longer they last.
  - More systemic complications occur (e.g., aspiration).
  - Most seizures end spontaneously within 7 min in adults and 12 min in children:
    • These are reasonable points to start treating to terminate seizures in order to prevent the establishment of status.
Status Epilepticus

Initial treatment
- Lorazepam (Ativan) IV 0.1 mg/kg
- Alternatives:
  - Propofol
    - Phenobarbital IV 20 mg/kg
    - Valproate IV 20 - 30 mg/kg
- If IV access cannot be established,
  - Midazolam (Versed)(buccal, nasal, IM)

Failure of the first drug given in adequate dosage constitutes refractory status.
Status Epilepticus

Treatment of refractory status (RSE)

- Midazolam 0.2 mg/kg loading dose with immediate infusion 0.1 – 2.0 mg/kg/hr
  - Must have EEG monitoring and demonstrate seizure suppression
  - After 12 hours free of seizures attempt to taper
  - May need other drugs (e.g., phenytoin, phenobarbital) to prevent recurrence

- Other options for RSE
  - Propofol
  - Pentobarbital
Acute Stroke Intervention

Intravenous thrombolysis is indicated for patients with:
- A clinical diagnosis of ischemic stroke
- A CT scan excluding intracerebral hemorrhage
- Onset of symptoms less than 3 hours before starting treatment
- No contraindications

- rt-PA 0.9 mg/kg (up to 90 mg)
  - 10% bolus, remainder over 60 min
Acute Stroke Intervention

- Between 3 and 6 hours, intra-arterial therapy may be an option.

- No role for acute heparin in evolving or completed stroke
  - May be needed later for secondary prevention in patients with atrial fibrillation.
Intracerebral Hemorrhage

- Hypertensive hemorrhages occur in the:
  - Putamen
  - Thalamus
  - Pons
  - Cerebellum

- Patients with hemorrhages elsewhere, or without a history of hypertension, need to be worked up for underlying vascular lesions or a bleeding diathesis.
For supratentorial hemorrhage, the major determinant of survival is hemorrhage volume:

- < 30 mL usually survive
- > 60 mL frequently die

Patients with cerebellar hemorrhages often benefit from surgical evacuation:

- Proceed before cranial nerve findings develop.
Intracerebral Hemorrhage

Management remains controversial

- Airway control
- Lowering mean arterial pressure may limit hemorrhage growth
- Correct coagulopathy
- Surgical intervention not routinely useful
  - May be helpful with superficial lesions
Subarachnoid Hemorrhage

Most commonly due to trauma and then ruptured aneurysm

Present with sudden headache, often diminished consciousness
  • Focal findings suggest intracerebral hemorrhage, which may occur due to dissection of blood from the bleeding aneurysm into the cortex.
Current Management Strategies for SAH

- Early definitive aneurysm obliteration
- Induce hypertension and increase cardiac output to treat vasospasm if it develops (after aneurysm is clipped)
  - Biggest risk between days 3-7 following bleed
- Nimodipine or nicardipine to relieve or ameliorate the effects of vasospasm
Current Management Strategies for SAH

- Interventional neuroradiologic techniques (e.g., angioplasty and intra-arterial verapamil or nicardipine infusion) to treat vasospasm
- Ventricular drainage to treat hydrocephalus
Complications of Aneurysmal SAH

- Rebleeding
- Cerebral vasospasm
- Volume disturbances
- Osmolar disturbances
- Seizures

- Arrhythmias and other cardiovascular complications
- CNS infections
- Other complications of critical illness
Aneurysmal Rebleeding

- Risk of rebleeding from unsecured aneurysms:
  - about 4% on the first post-bleed day
  - about 1.5% per day up to day 28

- Mortality of rebleeding following the diagnosis of SAH exceeds 75%.

- Rebleeding is more frequent in:
  - patients with higher grades of SAH
  - women
  - those with systolic blood pressures over 170 mmHg
Volume and Osmolar Disturbances

Reported in about 30% of SAH patients

Most common problem is cerebral salt wasting
- SIADH should not be diagnosed in the period of risk for vasospasm.
- Acute SAH patients should never be allowed to become volume depleted.
- The primary problem is excess of natriuretic factors, with secondary water retention to attempt to maintain volume (converse of SIADH).
Volume and Osmolar Disturbances

Prophylaxis: maintain adequate salt intake
- (e.g., 3L+ saline/d)
- some use mineralocorticoid supplementation

If hypo-osmolality occurs, need to increase the osmolality of the fluids administered to exceed that of the urine excreted
- hypertonic saline (1.8% - 3%) as needed
- some also give supplemental salt enterally
Conclusion

• Brain losses auto-regulation for up to 7 days post-injury
  – CPP can be affected directly
  – Tx guided by cortical oximetry and ICP monitoring

• Minimize secondary insults
  – Keep CPP > 60 (or SBP > 100)
  – Keep PCO2 30-35

• Time = Brain in injury and CVA
  – Surgical intervention ASAP when needed
Head Trauma
Hypoxia and hypotension are the 2 major causes of secondary CNS injury following head trauma.

Even in the best intensive care units, these complications occur frequently.

Preventing hypoxia and hypotension could have the greatest effect of any currently available treatment for head trauma.
Fluid Thresholds and Outcome from Severe Brain Injury

Retrospective study (from the NIH multicenter hypothermia trial data) of the effect on GOS of ICP, MAP, CPP, and fluid balance at 6 months after injury

Univariate predictors of poor outcome:
- ICP > 25 mm Hg
- MAP < 70 mm Hg or
- CPP < 60 mm Hg and fluid balance < -594 mL

**Fluid Thresholds and Outcome from Severe Brain Injury**

*Conclusions:* Exceeding thresholds of ICP, MAP, CPP, and fluid volume may be detrimental to severe brain injury outcome.

Fluid balance lower than -594 mL was associated with an adverse effect on outcome, independent of its relationship to intracranial pressure, mean arterial pressure, or cerebral perfusion pressure.
Diffuse Axonal Injury

- An active process triggered by the injury that takes about 24 hours to develop in humans
- May occur without any radiographic abnormality
- Frequently seen in areas of radiographically apparent “shear injury”
  - this latter finding usually occurs at the grey-white junction
- Is a major cause of long-term disability
Oxygenation Monitoring

- **Jugular bulb catheter**
  - jugular venous blood oxygen saturation
    - A-V differences in saturation, content, lactate

- **Direct cortical oxygen sensors (Licox)**
Management

Resuscitation and airway management

• avoid hypoxia and hypotension
• concomitant cervical spine lesions
• methods of intubation
  - orotracheal with inline stabilization
    • no nasal tubes (tracheal or gastric)
  - fiberoptic
• posture and head position
  - effects on ICP and CPP
Management

- Antiseizure drugs
  - phenytoin 20 mg/kg
  - only for the first week for patients without seizures

- Free radical scavengers
  - potential future therapies

- Nutrition and GI bleeding prophylaxis

- Thromboembolism prophylaxis
<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>% of pts</th>
<th>Good/moderate</th>
<th>Severe/vegetative</th>
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<td>5.9</td>
<td>0.0</td>
<td>94.1</td>
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<td>23.7</td>
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<td>52.0</td>
<td>13.5</td>
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<td>Diffuse injury III (swelling)</td>
<td>Cisterns compressed or absent, shift 0 – 5 mm, no high or mixed density lesion &gt; 25 cm³</td>
<td>20.5</td>
<td>16.4</td>
<td>49.7</td>
<td>34.0</td>
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<td>6.2</td>
<td>37.6</td>
<td>56.2</td>
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<td>Any lesion surgically evacuated</td>
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<td>22.8</td>
<td>38.4</td>
<td>38.8</td>
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<td>High or mixed density lesion &gt; 25 cm³ not surgically evacuated</td>
<td>4.8</td>
<td>11.1</td>
<td>36.1</td>
<td>52.8</td>
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<td>Brainstem injury</td>
<td>(no brainstem reflexes by physical exam)</td>
<td>0.4</td>
<td>0.0</td>
<td>33.3</td>
<td>66.7</td>
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### Life Expectancy for Persons who survive the first 24 hours

<table>
<thead>
<tr>
<th>Age at Injury</th>
<th>No SCI</th>
<th>Motor Functional at any Level</th>
<th>Para</th>
<th>Low Tetra (C5-C8)</th>
<th>High Tetra (C1-C4)</th>
<th>Ventilator Dependent at any Level</th>
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</thead>
<tbody>
<tr>
<td>20 yrs</td>
<td>57.2</td>
<td>51.6</td>
<td>45.2</td>
<td>39.4</td>
<td>33.8</td>
<td>16.2</td>
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<tr>
<td>40 yrs</td>
<td>38.4</td>
<td>33.5</td>
<td>27.8</td>
<td>23.0</td>
<td>18.7</td>
<td>7.2</td>
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<tr>
<td>60 yrs</td>
<td>21.2</td>
<td>17.5</td>
<td>13.0</td>
<td>9.6</td>
<td>6.8</td>
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</table>

### Life Expectancy for Persons who survive at least 1 year post-injury

<table>
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<tr>
<th>Age at Injury</th>
<th>No SCI</th>
<th>Motor Functional at any Level</th>
<th>Para</th>
<th>Low Tetra (C5-C8)</th>
<th>High Tetra (C1-C4)</th>
<th>Ventilator Dependent at any Level</th>
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<tr>
<td>20 yrs</td>
<td>57.2</td>
<td>52.5</td>
<td>46.2</td>
<td>41.2</td>
<td>37.1</td>
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<tr>
<td>40 yrs</td>
<td>38.4</td>
<td>34.3</td>
<td>28.7</td>
<td>24.5</td>
<td>21.2</td>
<td>13.7</td>
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<tr>
<td>60 yrs</td>
<td>21.2</td>
<td>18.1</td>
<td>13.7</td>
<td>10.6</td>
<td>8.4</td>
<td>4.0</td>
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</table>
About 40% of persons with paraplegia and 30% of persons with tetraplegia (quadriplegia) eventually return to work.
ASIA IMPAIRMENT SCALE

□ A = Complete: No motor or sensory function is preserved in the sacral segments S4-S5.

□ B = Incomplete: Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

□ C = Incomplete: Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

□ D = Incomplete: Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

□ E = Normal: motor and sensory function are normal
Complete SCI

- Loss of all function below the level of the lesion
- Typically associated with spinal shock
Types of Incomplete SCI

- Central cord syndrome
- Anterior cord syndrome
- Brown-Sequard syndrome
- Spinal cord injury without radiologic abnormality (SCIWORA)
Central Cord Syndrome

- Typically results from an extension injury
- Greater impairment of upper than lower extremity function
- Urinary retention
- Sparing of sacral sensation
Anterior Cord Syndrome

Due either to:

- Compression of the anterior portion of the cord by a vertebral body
- Anterior spinal artery occlusion

Presents with preservation of dorsal column function (vibration and position sense)
Brown-Sequard Syndrome

- Hemisection of the cord
- Usually due to penetrating injury
Spinal Cord Injury Without Radiologic Abnormality (SCIWORA)

- No bony abnormalities on plain film or CT
  - MRI may show abnormalities
- Usually in children; symptoms may be transient at first
- Should probably lead to immobilization to prevent subsequent development of cord damage
Secondary Injury

After the initial macroscopic injury, secondary injuries are an important cause of disability:

- Movement of unstable spine
- Vascular insufficiency
- Free radical induced damage
**Neural Control of Blood Pressure and Blood Flow**

- Complete lesions above T1 will therefore eliminate all sympathetic outflow.
- Lesions between T1 and T6 will preserve sympathetic tone in the head and upper extremities but deny it to the adrenals and the lower extremities.
- Lesions between T6 and the lumbar cord will preserve adrenal innervation but denervate the lower extremities.
“Spinal” shock

- Actually refers to the acute loss of tendon reflexes and muscle tone below the level of a spinal cord lesion.
- However, neurogenic hypotension is very common and can be profound with spinal cord lesions above T1:
  - In the series of Vale et al, 40% of patients with complete cervical spinal cord lesions were in neurogenic shock on presentation.
- Hypotension in spinal shock is typically accompanied by bradycardia, reflecting loss of cardiac sympathetic efferents and unopposed vagal tone:
  - These patients are unable to mount a tachycardic response to volume depletion.
  - Because of their vasodilation they are warm, but may still have elevated venous lactate concentrations.
CNS Disturbances Affecting the Cardiovascular System

- It is tempting to treat this hypotension with volume expansion, even if the patient is not volume depleted.
  - Initially this is appropriate as venous return is frequently reduced.
  - However, this must be pursued cautiously.
- If the patient is conscious, making urine, and the venous lactate is decreasing, the MAP is probably adequate.
- Neurogenic pulmonary edema is common in patients with cervical spinal cord lesions, complicating their management.
- These patients commonly develop pulmonary vascular redistribution, interstitial edema, increased AaDO₂, and on occasional alveolar edema at PCWPs in the 18 - 20 mmHg range:
  - May provide important clues to the mechanisms of NPE
Management of Cardiovascular Shock After Spinal Cord Injury

Always suspect associated injuries:
- Usual symptoms and physical findings may be absent due to the spinal cord injury.

Volume resuscitation cannot be guided solely by physical findings:
- Hypotension and bradycardia will persist regardless of the volume of saline or colloid administered.

Replace the missing adrenergic tone with $\alpha$-agonists (phenylephrine or norepinephrine depending on heart rate).
March 2002
Spinal Perfusion Pressure Management

Developed by analogy to cerebral perfusion pressure management

- Attempt to prevent cord ischemia by raising blood pressure.
  - Assumes that the same secondary injury mechanisms (hypotension and hypoxia) worsen the outcome from spinal cord injury as in head injury
  - NASCIS II and III provide an inference that oxygen-derives free radicals worsen outcome after spinal cord injury.
Vale et al applied cerebral perfusion pressure management principles to 77 patients with cervical and thoracic cord injuries.

- Place PA catheters and arterial lines
- Maintained MAP > 85 mmHg
  - Used “fluids, colloids, and vasopressors”
  - Did not specify how much of what
Spinal Perfusion Pressure Management

30% of patients with complete cervical injuries were able to walk at 1 year
- 20% had regained bladder function

“Much better than historical controls or reports in the literature”
In a series 67 patients with penetrating injuries of the cord, only 7% of patients presented with neurogenic shock:

- 74% of patients had significant blood loss, felt to explain their hypotension.
CNS Disturbances Affecting the Cardiovascular System

Autonomic dysreflexia:

- Patients with lesions above T5 may develop hypertension and profuse sweating in response to a distended viscus (usually the bladder).
- Presumably represents adrenal release of catecholamines via spinal cord pathways not being controlled by brainstem centers.
Lesions above or at C4
- Phrenic nerve failure

Lesions between C4 – T6
- Loss of parasternal intercostal contraction causes chest wall to sink during inspiration, decreasing the tidal volume
- Loss of sympathetic innervation to the lungs can also prompt bronchospasm (imbalance of parasympathetic and sympathetic tone).
Management

**ABCs**
- If intubation needed, use in-line stabilization
  - Direct laryngoscopy vs. fiberoptic
- Maintain blood pressure with volume, packed RBCs, vasopressors as needed

**Prevent secondary injury**
- Log-rolling

**Consider concomitant head injury**
Management

Pharmacologic

- Methylprednisolone 30 mg/kg bolus then 5.4 mg/kg/h for 23 – 47 hours depending on latency from the injury
  - Starting 0 – 3 hours from injury: 23 hours duration
  - 3 – 8 hours: 47 hours
  - After 8 hours, do not start
  - Although there is still debate about its efficacy, this is often considered the “standard of care.”
  - Not likely to be an anti-edema effect, since tirilazad (a non-glucocorticoid free radical scavenger) is equivalent.
Blood Pressure

- No standards or guidelines
- Options:
  - Avoid or correct hypotension (systolic BP < 90 mmHg)
  - Maintaining MAP between 85 and 90 mmHg for the first 7 days is recommended
DVT Prophylaxis

Standards

- Either:
  - LMW heparin, rotating bed, adjusted dose heparin (1.5 x control aPTT), or a combination of these, or
  - Low-dose unfractionated heparin plus sequential compression devices or electrical stimulation

Guidelines

- Low-dose unfractionated heparin alone is insufficient.
- Oral anticoagulation alone probably not indicated
DVT Prophylaxis

Options

- 3-month duration of prophylaxis
- Use IVC filters for patients failing anticoagulation or intolerant of it