Neurosurgical Trauma

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Epidemiology

- Average incidence of clinically important head injury in children is about 185 per 100,000 with the incidence generally dropping with increasing age
  - Boys are injured at a rate of approx twice that of girls

- In the US, TBI annually impact 475,000 children and causes over 2,600 TBI related deaths, 37,000 hospitalizations, and 435,000 ED visits

- Cost of treatment for TBI approximate $2.6 billion annually

- 85% of the brain injuries sustained in childhood are mild and non-life threatening

- Severity and mechanism of brain injury are determinants of outcome
  - Most common mechanism resulting in head injury in children is a fall
  - Inflicted injury is the leading cause of severe brain injuries in very young children
  - In older children, severe brain injury is most commonly seen in relation to MVCs
Pathophysiology

- Primary neurologic injury—immediate disruption of neuronal, axonal, supportive measures, and vascular tissues
  - Magnitude and location
  - Major determinant of injury outcome
  - Triggers cascade of intracellular and extracellular biochemical changes

- Secondary neurologic injury—result of a cascade of biochemical, cellular, and metabolic responses to direct injury which worsens in patients who develop hypoxia, hypotension, or both
  - New damage in the region of the primary injury and in areas of previously uninjured brain or spinal cord
Systemic Responses

- Systemic reaction seen after brain or spinal cord injury include alterations in blood pressure and respirations

- Initially following brain injury, CBF appears to be decreased in children
  - Hypo perfusion in conjunction with increased metabolic demand makes the brain more susceptible to secondary insults such as hypoxemia and hypotension
  - Cerebral perfusion may be particularly dependent on maintaining adequate BP because cerebral autoregulation is often impaired
  - Release of excitatory neurotransmitters cause neuronal damage

- Cerebral swelling
- Hyperthermia
- Hyperglycemia
- Post traumatic seizures
- Expanding local hemorrhages can cause compression of adjacent vessels and tissues resulting in ischemia
Therapeutic Strategies

• Essential therapeutic strategies for brain and spinal cord injuries are based on preventing ischemic injury by aggressive support of intravascular volumes and blood pressure.

• Effective, supportive, and preventative therapy should begin as quickly after the injury as possible.

• Support of systemic blood pressure, reduction of ICP to assure cerebral perfusion, maintaining normal ventilation, removal of compressive lesions and the prevention of the deleterious complications are mainstay of management.
Classification

- Severity of traumatic brain injury is typically defined by the initial Glasgow Coma Scale or Pediatric Glasgow Coma score

- Mild (GCS 13-15)
- Moderate (GCS 9-12)
- Severe (GCS<9)

<table>
<thead>
<tr>
<th>Sign</th>
<th>Glasgow Coma Scale</th>
<th>Pediatric Glasgow Coma Scale</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>To command</td>
<td>To sound</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Verbal response</td>
<td>Oriented</td>
<td>Age-appropriate vocalization, smile, or orientation to sound, interacts (coos, babbles), follows objects</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Confused, disoriented</td>
<td>Cries, irritable</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>Cries to pain</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>Moans to pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Motor response</td>
<td>Obey commands</td>
<td>Spontaneous movements (obeys verbal command)</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localizes pain</td>
<td>Withdraws to touch (localizes pain)</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Withdraws</td>
<td>Withdraws to pain</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion to pain</td>
<td>Abnormal flexion to pain (deorticile posture)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Abnormal extension to pain</td>
<td>Abnormal extension to pain (decerebrate posture)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Best total score</td>
<td></td>
<td></td>
<td>15</td>
</tr>
</tbody>
</table>
Spectrum of TBI

- Focal injuries (Usually immediately apparent on CT):
  - Contusions
  - Lacerations
  - Traumatic hematomas
  - Localized damage resulting from expanding masses, shifts, and distortions of the brain

- Diffuse injuries (Less striking changes on early neuroimaging):
  - DAI (cerebral concussions)
  - Global ischemia
  - Systemic hypoxia
  - Diffuse brain swelling
  - Diffuse vascular injury
Focal Brain Injury

- Most are associated with impact related mechanisms, can also result in skull fractures
- 20% of head injured children who are admitted to the hospital have skull fractures
  - Majority will not require any treatment
  - Serve as an indicator of both the mechanism and severity of the head injury
  - Associated with a higher likelihood of developing an expanding intracranial hematoma or harboring a significant brain injury
- The most common focal injury resulting from non penetrating mechanisms is a cerebral contusion
- Clinical presentation depends mostly upon the extent of the initial injury, amount of associated hemorrhage resulting in mass effect, and location in the brain
- Many are neurologically silent
- When symptomatic, usually cause focal neurologic deficit or seizures
Focal Brain Injury

- Traumatic intracerebral hematomas are unusual lesions in the pediatric age group
  - Seem to be related to disruption of central arterial blood vessels
  - Associated with more severe mechanisms of injury and with more profound neurologic dysfunction
  - Part of DAI in many cases
  - Differentiated from traumatic hematomas by lack of contact with the surface of the brain

- Penetrating injuries
  - Can result in focal contusions, intracerebral hemorrhages, and cerebral lacerations
  - Usually silent but deeper penetrations are more likely to be symptomatic
  - May become symptomatic in delayed fashion from the expansion of intracerebral hemorrhage, recognition of CSF fistula, or by the appearance of symptoms indicated infection
  - Strongly associated with direct cerebrovascular injury
Diffuse Brain Injury

- Characterized by general disturbances of neuronal function that begin immediately at the time of injury, while showing general preservation of the brain structure on early CT.

- Direct result of energy dissipation within the substance of the brain or as the result of systemic insult: Angular or translational accelerations or decelerations.

- Exist on a continuum from extremely mild with transient physiologic disturbances of neurology function (cerebral concussions) to DAI which is more damaging and ultimately lethal.

- The amount of neuronal disruption is roughly proportional to the amount of energy dissipated in the brain substance.

- Appearance on CT can be as subarachnoid hemorrhage, small but widespread intracerebral hemorrhages and intraventricular hemorrhage.
Diffuse Brain Injury

- Cerebral concussions
  - Graded set of clinical syndromes with increasing disturbances in the level and content of consciousness
  - Confusion without amnesia
  - Confusion associated with amnesia of varying depth and duration
  - Loss of consciousness with and without transient sensorimotor paralysis or disturbances of respiration or circulation

- DAI
  - Most common cause of prolonged coma from mechanical brain injury
  - Unconscious from the time of injury and remain so for a prolonged period
  - Not uncommon to note pupillary changes, skewed gaze and decerebrate posturing
  - Appearance on CT varies on the severity of injury and degree of associated hemorrhages
Inflicted Injuries

- The most common cause of severe and life threatening brain injury in infants is inflicted injury.
- Infants presenting with alteration of consciousness, retinal hemorrhages, and acute intracranial hemorrhages on CT scans are likely to have suffered NAT.
- Additional findings of new or healing skeletal fractures, unexplained bruises, apnea, seizures or other solid organ injuries.
- Thorough evaluation by a multidisciplinary team should be performed whenever there is suspicion that an injury is inflicted.
Initial Assessment of TBI

- First establish a working diagnosis of the type and severity of the injury that directs the selection of initial therapies and planning and coordination of other diagnostic studies and management of associated systemic injuries
  - Determine the severity of the injury by scoring the level of consciousness (GCS)

- Second is to establish a baseline to measure the effects, both positive and deleterious, of the therapies or interventions
  - Identify signs of impending herniation
    - Hypertension with bradycardia or tachycardia
    - Unequal or fixed and dilated pupils
    - Abnormal breathing pattern
    - Hemiparesis
    - Decorticate, decerebrate or absent motor response to pain
Initial Assessment of Severe TBI

- **Neurologic assessment that includes:**
  - Level of consciousness
  - Pupillary examination for size, reactivity and symmetry
  - Extra ocular movements
  - Funduscopic examination
  - Brainstem reflexes
  - Deep tendon reflexes
  - Response to pain

- **Laboratory studies:**
  - Blood glucose
  - Type and cross
  - ABG/VBG
  - CBC
  - Serum electrolytes and osmolality
  - AST/ALT
  - UA
  - PT, aPTT, INR
Initial Assessment of Severe TBI

- All pediatric patients with severe TBI should undergo unenhanced CT of the head
  - Some severely injured patients may initially have a normal CT scan but generally have significant neurologic findings on physical examination

- Children injured in accidents with high energy mechanisms that result in isolated chest, abdominal or skeletal injuries should undergo brain CT prior to anesthetic administration of the institution of narcotic analgesia or sedation

- All patients with severe TBI require imaging of the cervical spine and should not have spine injury excluded based upon imaging alone

- MRI imaging is more sensitive for DAI and associated cerebral edema but not necessary for initial stabilization and treatment

- May also require abdominal CT to evaluate for intra-abdominal injury with significant blunt multiple trauma and unreliable abdominal examination
Early Management of Severe TBI

- Initial management of children with severe TBI includes supporting airway, breathing and circulation, to prevent secondary injury caused by hypoxia or hypotension and measures to prevent and treat intracranial HTN.

- Advanced airway management is warranted to maximize oxygenation and ventilation and protect against aspiration of gastric contents for:
  - Decreasing level of consciousness (GCS\(\leq 8\)) or rapidly falling
  - Signs of respiratory failure
  - Hemodynamic instability

- Maintain cervical spine immobilization

- Hyperventilation (PaCO\(_2<35\)) may cause cerebral ischemia due to cerebral blood flow, should maintain between 35 and 40mmHg

- Optimize cerebral perfusion by managing intracranial pressures
Early Management of Severe TBI

- Treat hypovolemic shock with isotonic solutions with the goal of attaining a state of normal circulating volume
- Temperature management to prevent fever but avoid hypothermia
- Head of bed elevation
- Use of steroids is not recommended
- Feeding patients by the 5\textsuperscript{th} day to obtain basic caloric intake decreases mortality
Basic measures for stabilization of children with elevated ICP and interventions for impending herniation or sustained elevations:

- Early involvement of a neurosurgeon should occur for all children with signs of herniation or symptomatic intracranial hypertension, whenever possible.

### Baseline stabilization and management for increased ICP

1. If not already performed for herniation, secure the airway using ETI with Cricothyrotomy in tracheal patients.
2. Maintain normal oxygenation and blood pressure; avoid hyperventilation. For intubated patients, maintain PaCO₂ at 25 to 40 mmHg unless sign of herniation.
3. Elevate the head of the bed 15 to 30 degrees and keep the head midline.
4. Maintain normal body temperature using antipyretics or cooling blankets as needed.
5. Administer dexamethasone 0.5 mg/kg (maximum dose 15 mg) every 6 hours to patients with conditions causing vasogenic edema such as brain tumors, intracranial hematomas, cerebral edema, and other central nervous system processes.
6. Administer prophylactic anticonvulsants (e.g., benzodiazepines, phenytoin, or phenobarbital) to patients with severe traumatic brain injury, depressed skull fractures, or parenchymal abnormalities.
7. During mechanical ventilation, avoid high positive inspiratory pressure (IPPV) and end expiratory pressures (PEEP) as long as oxygenation and ventilation is maintained.
8. Insure sedation and analgesia with continuous infusions of midazolam and morphine.
9. Maintain paralysis with a continuous infusion of vecuronium.
10. Administer bicarbonate 1 mEq/kg, 2 to 3 minutes before suction of the orotracheal tube.

### Signs of impending herniation?
- Hypertension with brady- or tachycardia
- Anorexia (unusual refusal)
- Severe headache
- Convulsions
- Abnormal breathing pattern
- Hypotonia
- Decrease or decrease posture

### Intracranial monitoring indicated (any one of the following):
- Traumatic brain injury with ICP C6 or
- Signs of impending herniation
- Underlying condition that will require ongoing ICP monitoring?

### ICP monitor placed by neurosurgeon

### ICP >20 mmb/hg?

### Harnett 0.25 to 1 g/kg
- OR
- 1% saline 3 to 5 mL/kg
- Hyperosmolar to 40% NaCl 30 to 35 mmb/hg

### ICP >20 mmb/hg?

### Acute obstructive hydrocephalus on neuroimaging?

### Emergent placement of an external ventricular drainage device and drainage of CSF by a neurosurgeon

### Neuroimaging shows lesions amenable to neurosurgical treatment?

### Baseline stabilization and management (refer to table):  
- Maintain additional therapies required to control ICP
- Treat underlying cause

### Potential actions determined in consultation with a neurosurgeon and patient tolerance:
- Rehaut/Other coma
- Decompress cranectomy
- Moderate hypothermia (core body temperature 33.5 to 34°C)
- Hyperventilation to 25 to 30 mmHg with monitoring of central oxygenation

### Emergency operation
Early Management of Severe TBI

- Focal injuries that may require neurosurgical intervention must quickly be identified and surgically managed:
  - Epidural hematomas
  - Acute subdural hematomas with associated midline shift
  - Penetrating brain injuries

- In patients whose brain injury is not amenable to surgical correction, further care is focused on preventing hypoxia or hypotension and monitoring for and treating intracranial HTN
Management of Minor Brain Injuries

- The vast majority of children with head injury have trivial, minor, or minimal primary brain injuries
- These children will most likely recover without any intervention, however there are a small fraction who harbor enlarging hematomas or are in the early stages of brain swelling
- Focus of evaluating is to identify patients at risk of neurologic deterioration or delayed complications and prevent either from occurring
- Early neurologic imaging may identify findings that the patient is at risk for deterioration regardless of their level of consciousness
  - Cisternal compression, hemorrhagic shear, contusion or small traumatic hematomas
  - Patient are candidates for frequent reassessments, repeat neuroimaging, ICP monitoring, therapies to control intracranial dynamics
Minor Head Trauma Definitions

- **Children <2 y/o:**
  - History of physical signs of blunt trauma to the scalp, skull or brain in an infant that is alert or awakens to voice or light touch

- **Children >2 y/o:**
  - GCS 14-15 at initial examination, no abnormal or focal neurologic exam findings, no physical evidence of skull fracture

- **Mild traumatic brain injury:** associated with brief LOC, disorientation, or vomiting (GCS 13-15)
  - Concussions are defined as trauma induced brain dysfunction without demonstrable structural injury on standard neuroimaging

- **Clinically important TBI:** are at higher risk of intracranial injury after minor head trauma
  - Presence of intracranial injury (epidural hematoma, SDH, contusion) associated with neurosurgical intervention (surgery or ICP monitor), intubation, hospitalization >48 hrs due to head injury, death
  - Or depressed skull fracture warranting operative evaluation
  - Or basilar skull fracture, hemotympanum, CSF otorrhea or rhinorrhea
Clinical Decision Rules

For infants and children with minor head trauma, absence of high risk signs or symptoms of clinically important TBI it is suggested that management decisions be guided by the use of the Pediatric Emergency Care Applied Research Network (PECARN)

**PECARN rule: Findings associated with very low risk of significant traumatic brain injury in children:**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Clinical criteria</th>
</tr>
</thead>
</table>
| <2          | Normal mental status  
Normal behavior per routine caregiver  
No LOC  
No severe mechanism of injury  
No nonfrontal scalp hematoma  
No evidence of skull fracture |
| ≥2 to 18 | Normal mental status  
No LOC  
No severe mechanism of injury  
No vomiting  
No severe headache  
No signs of basilar skull fracture |
Clinical Decision Rules

- Children <2 y/o are more difficult to assess, may be asymptomatic despite having a ciTBI, are at risk for abusive head trauma, and are more prone to skull fractures

Clinical findings associated with a high risk for clinically important traumatic brain injury in children

<table>
<thead>
<tr>
<th>Clinical findings that warrant urgent neuroimaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suspicion of child abuse (infants)</td>
</tr>
<tr>
<td>Persistent vomiting</td>
</tr>
<tr>
<td>Seizure</td>
</tr>
<tr>
<td>Altered mental status*</td>
</tr>
<tr>
<td>Prolonged loss of consciousness</td>
</tr>
<tr>
<td>Bulging fontanelle (infants)</td>
</tr>
<tr>
<td>Focal neurologic findings</td>
</tr>
<tr>
<td>Skull fracture</td>
</tr>
</tbody>
</table>
Clinical Decision Rules

- Intermediate risk patients may be managed with close observation for 4-6 hours after injury or may be evaluated immediately by head CT

### Moderate risk findings of clinically important traumatic brain injury in children

**Clinical finding warranting observation or neuroimaging**

<table>
<thead>
<tr>
<th>Clinical Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting that is self-limited</td>
</tr>
<tr>
<td>Loss of consciousness that is uncertain, or isolated and very brief (less than a few seconds)</td>
</tr>
<tr>
<td>Headache (older, verbal child)</td>
</tr>
<tr>
<td>History of lethargy or, in infants, irritability, now resolved</td>
</tr>
<tr>
<td>Behavioral change reported by caregiver (infants)</td>
</tr>
<tr>
<td>Injury caused by high-risk mechanism of injury*</td>
</tr>
<tr>
<td>Scalp hematoma (particularly nonfrontal) in infants younger than 2 years of age</td>
</tr>
<tr>
<td>Skull fracture more than 24 hours old (nonacute)</td>
</tr>
<tr>
<td>Unwitnessed trauma of concern¶</td>
</tr>
<tr>
<td>Age younger than three months with nontrivial trauma</td>
</tr>
</tbody>
</table>
Minor Head Trauma Management

- Low risk of ciTBI based upon clinical findings or according to a clinical decision rule can be discharged home without neuroimaging
  - Risk of ciTBI for those who meet very low risk by PCARN rule is approx 0.5 to 3 per 1000

- Not low risk:
  - Who are observed without neuroimaging be discharged home rather than undergo hospital admission if they show improvement in initial symptoms during an observation period lasting 4-6 hours post injury and meet all criteria for safe discharge
  - Who have normal neuroimaging, normal level of consciousness and meet all discharge criteria can be discharged rather than hospital admission

- Discharge criteria:
  - No suspicion of inflicted injury, easily arousable with light touch with normal neuro exam, return to baseline level of function, tolerating oral intake of fluids, no extracranial injuries warranting admission, capable caretakers
Early Complications of TBI

- Acute complications include those related to skull fractures, infectious processes related to cranial penetration and CSF fistulas and acute neurologic complications.
- Simple, non depressed or minimally depressed skull fractures will heal spontaneously.
- Widely diastatic or cranial burst fractures in young children are indications of dural injury and are not likely to heal without surgical reconstruction.
- Basilar skull fractures place the patient at increased risk for infection/meningitis and associated with cranial neuropathies (olfactory, optic, auditory, vestibular, or facial nerve injuries).
  - Bilateral orbital ecchymosis or swelling, signs of midface or orbital fractures, hemotympanum, otorrhagia, or Battle sign.
Early Complications of TBI

- One of the most common complications of brain injury, even in mild injury, is epilepsy.
  - Risk factors include younger age and increasing injury severity.
  - Early seizures defined as those that occur within the first week of injury.

- Post concussion syndrome: shortly after what seems like a mild cranial impact injury, the child exhibits the acute onset of pallor, diaphoresis, and impair responsiveness.
  - CT normal.
  - Resolves as rapidly as it occurs.
Outcomes of TBI

- With the exception of infants suffering inflicted head injuries, the overall mortality from head injury in childhood is roughly half of that reported for similarity severe head injury in adults
  - Mortality <5% for all levels of injury severity
  - <20% for those defined as having severe injuries based on GCS or other injury severity scoring

- Factors related to poor outcome include: high energy mechanisms, structural injury, swelling and shift on admission CT scans, persistent or resistant elevations in ICP, presence of chest or abd injuries, systemic complications

- TBI is the leading cause of acquired disability in childhood

- Children who have suffered severe brain injuries are likely to show persistent adverse effects on intellectual function, memory, attention, language, and behavior

- Inflicted brain injuries are associated with the highest morbidity and mortality approaching 40%
Concussion

- Initial management focuses on avoidance of additional head injury through removal of competition, restriction of physical activity and neurocognitive rest.
- Full physical rest until the patient has no symptoms of concussion or return to baseline on standardized assessment tools.

<table>
<thead>
<tr>
<th>Symptoms of concussion recorded by the High School Reporting Information Online injury surveillance system (HS RIO)</th>
<th>No. of concussions resulting in symptom</th>
<th>Percentage of concussions resulting in symptom*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>508</td>
<td>93</td>
</tr>
<tr>
<td>Dizziness/unsteadiness</td>
<td>406</td>
<td>75</td>
</tr>
<tr>
<td>Difficulty concentrating</td>
<td>308</td>
<td>57</td>
</tr>
<tr>
<td>Confusion/disorientation</td>
<td>250</td>
<td>46</td>
</tr>
<tr>
<td>Vision changes/sensitivity to light</td>
<td>204</td>
<td>38</td>
</tr>
<tr>
<td>Nausea</td>
<td>157</td>
<td>29</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>144</td>
<td>27</td>
</tr>
<tr>
<td>Amnesia</td>
<td>132</td>
<td>24</td>
</tr>
<tr>
<td>Sensitivity to noise</td>
<td>103</td>
<td>19</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>58</td>
<td>11</td>
</tr>
<tr>
<td>Irritability</td>
<td>50</td>
<td>9</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>25</td>
<td>5</td>
</tr>
<tr>
<td>Hyperexcitability</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>41</td>
<td>8</td>
</tr>
</tbody>
</table>
Concussion

- Cognitive rest: children and adolescents returning to school after a concussion warrant monitoring of symptoms and academic adjustments as needed, if symptoms are exacerbated by work or environment.

### Potential adjustments for concussion symptoms in the school setting

<table>
<thead>
<tr>
<th>Sign/symptom</th>
<th>Potential adjustments in school setting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>- Permit frequent breaks</td>
</tr>
<tr>
<td></td>
<td>- Identify aggravators and reduce exposure to them</td>
</tr>
<tr>
<td></td>
<td>- Provide rests, planned or as needed, in nurses office or quiet area</td>
</tr>
<tr>
<td>Dizziness</td>
<td>- Allow student to put head down if symptoms worsen</td>
</tr>
<tr>
<td></td>
<td>- Give student early dismissal from class and extra time to get from class to class to avoid crowded hallways</td>
</tr>
<tr>
<td>Visual symptoms (light sensitivity, double vision, blurry vision)</td>
<td>- Reduce exposure to computers, smart boards, videos</td>
</tr>
<tr>
<td></td>
<td>- Reduce brightness on the screens</td>
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<tr>
<td></td>
<td>- Allow the student to wear a hat or sunglasses in school</td>
</tr>
<tr>
<td></td>
<td>- Permit use of audiocassettes or books</td>
</tr>
<tr>
<td></td>
<td>- Turn off fluorescent lights as needed</td>
</tr>
<tr>
<td></td>
<td>- Seat student closer to the center of classroom activities (blurry vision)</td>
</tr>
<tr>
<td></td>
<td>- Cover one eye with patch/tape one lens if glasses are worn (double vision)</td>
</tr>
<tr>
<td>Noise sensitivity</td>
<td>- Allow the student to have lunch in quiet area with a classmate</td>
</tr>
<tr>
<td></td>
<td>- Limit or avoid band, chair, or shop classes</td>
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<tr>
<td></td>
<td>- Avoid noisy gyms and organized sports practices/games</td>
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<tr>
<td></td>
<td>- Permit the use of earplugs</td>
</tr>
<tr>
<td></td>
<td>- Give student early dismissal from class and extra time to get from class to class to avoid crowded hallways during pass time</td>
</tr>
<tr>
<td>Difficulty concentrating or remembering</td>
<td>- Avoid testing or completion of major projects during recovery when possible</td>
</tr>
<tr>
<td></td>
<td>- Provide extra time to complete nonstandardized tests</td>
</tr>
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<td></td>
<td>- Postpone standardized testing (may require formal academic accommodation)</td>
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<tr>
<td></td>
<td>- Consider one test per day during exam periods, as tolerated</td>
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<tr>
<td></td>
<td>- Consider the use of preprinted notes, notetaker, scribe, or reader for oral test taking</td>
</tr>
<tr>
<td>Sleep disturbances</td>
<td>- Allow for late start or shortened school day to catch up on sleep</td>
</tr>
<tr>
<td></td>
<td>- Allow rest breaks</td>
</tr>
</tbody>
</table>
Concussion

- **Return to learn**: Once patients can concentrate on a task and tolerate visual and auditory simulation for 30-45 min they may return to school with academic adjustments.

- **Because cognitive recovery may lag behind physical recovery**, clinicians frequently use neurophysiological testing to help determine when the patient has fully recovered from a concussion.

- **Return to play**: Fully complete the consensus graduated return to play protocol after an individualized period of cognitive and physical rest before clearance for competition.

- **Graded return to play protocol to begin after:**
  - Successful return to school
  - Symptom free and off prescribed medications to treat concussion
  - Normal neurologic exam
  - Back at baseline balance and cognitive performance measures
##  Graduated return to play protocol

<table>
<thead>
<tr>
<th>Rehabilitation stage</th>
<th>Functional exercise at each stage of rehabilitation</th>
<th>Objective of each stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No activity</td>
<td>Complete physical and cognitive rest</td>
<td>Recovery</td>
</tr>
<tr>
<td>2. Light aerobic exercise</td>
<td>Walking, swimming or stationary cycling keeping intensity &lt;70 percent MHR; no resistance training</td>
<td>Increase HR</td>
</tr>
<tr>
<td>3. Sport-specific exercise</td>
<td>Skating drills in ice hockey, running drills in soccer; no head impact activities</td>
<td>Add movement</td>
</tr>
<tr>
<td>4. Non-contact training drills</td>
<td>Progression to more complex training drills, eg, passing drills in football and ice hockey; may start progressive resistance training</td>
<td>Exercise, coordination, and cognitive load</td>
</tr>
<tr>
<td>5. Full contact practice</td>
<td>Following medical clearance, participate in normal training activities</td>
<td>Restore confidence and assess functional skills by coaching staff</td>
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<tr>
<td>6. Return to play</td>
<td>Normal game play</td>
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Six-day return to play protocol. Each day the athlete makes a stepwise increase in functional activity, is evaluated for symptoms, and is allowed to progress to the next stage each successive day if asymptomatic.
Spine and Spinal Cord Injury

- Spinal cord injuries in children are rare but the consequences of such injuries can be devastating.

- Major therapeutic efforts are the same as for brain injury with the aim to prevent new primary injury and ameliorate secondary injury:
  - Maintaining anatomic alignment of the vertebral column during the period of resuscitation and evaluation.
  - Supporting blood pressure and oxygenation.
Epidemiology

- Less than 10% of spinal injuries occur in children each year
- Spinal fractures account for 1-2% of all pediatric fractures
- <20% of spinal injuries in children occur below the cervical spine
- Children <8 y/o are more susceptible to injury of the upper cervical spine
- Younger patients may also incur fractures of the growth plate and ligamentous injuries
- Most common injuries in older children are vertebral body and arch fractures, usually in the lower cervical spine
- Most common causes of spinal injuries include falls, athletic activities, child abuse, diving accidents, and motor vehicle trauma
Anatomy

- Pediatric spine has several properties that allow significant self reducing displacement of the vertebral column permitting a wider range of flexion, extension and rostrocaudal distraction
  - Elasticity of the joint capsules and ligaments
  - Shallow and horizontally oriented facet joints
  - Anterior wedging of the vertebral bodies
  - Poorly developed uncinate processes
  - Larger heads and weaker cervical musculature

- 10-15% of spinal injuries in children involve skip lesions with vertebral or cord injuries at multiple levels
Mechanism of Injury

- Cervical spine injury can occur through flexion, extension, vertical compression, rotation or a combination of these.
- Most result from direct compression or disruption of the cord by fracture fragments or subluxed vertebrae.
- Hyperflexion injuries are the most common and may cause wedge fractures of the anterior vertebral body with disruption of the posterior elements.
- Hyperextension injuries may cause compression of the posterior elements and disruption of the anterior longitudinal ligament.
- Axial loading may cause burst or comminuted fractures of the C1 in the upper cervical spine or of the vertebral bodies in the lower cervical spine.
- Rotational injuries may cause fracture or dislocation of the facets.
Evaluation of Injury

- Must have an increased index of suspicion based on injury mechanism and neurologic presentation
- Classic triad of symptoms includes local pain, muscle spasm, and decreased range of motion of the neck
- Transient or persistent paresthesias or weakness
- Tenderness, swelling, ecchymosis, or palpable defect posteriorly along the spinous processes
- Injury above the T6 level may present in spinal or neurogenic shock (hypotension and bradycardia)
- Neurologic examination: flaccid muscle tone, muscle strength, isolated sensory deficit, deep tendon reflexes, rectal tone, level of paralysis
Clinical Spectrum of Injury

- Spine injuries are assessed in terms of immediate and late stability
  - Immediate stability refers to the risk of new or further neurologic injury while bearing physiologic loads without immobilization
  - Late stability implies the potential to heal with proper immobilization based on specific injury pattern
Clinical Spectrum of Injury

- The cardinal sign of a spinal cord injury is neurologic dysfunction below an anatomic spinal motor or sensory level.

- Spinal cord injury generally presents as symmetrical flaccid paralysis with sensory loss at the same anatomic level.

- Cervical spinal cord injuries can cause profound systemic hypotension by disruption of sympathetic pathways below the level of the injury.

- Neurogenic shock → bradycardia with hypotension, vasodilation despite hypothermia.
  - Paradoxic respirations, priapism, Horner syndrome, inability to sweat.
Injury without Radiographic Abnormality

- Spinal cord injury without radiographic abnormality (SCIWORA)
  - No evidence of fracture or ligamentous instability on plain xray or CT

- Injury of children, likely related to biomechanical properties of the juvenile spine

- Many will develop neurologic deficits hours to day after injury

- Immobilization of the cervical spine markedly reduces the incidence

- Most patient swill have evidence of spinal cord and/or ligamentous or other soft tissue injury on MRI
Initial Management

- Mainstay of management of spine injury is immobilization of the entire spinal axis in the field
  - Use of appropriately sized cervical collar
  - Children <8 y/o, proper immobilization may require special boards as the disproportionately large head places them in flexion when placed on a neutral board

- The number of repeat radiographs required to ascertain that pediatric cervical spine is free of injury after head trauma has been show to be significantly decreased when the initial CT of the cervical spine is performed at the time of head CT

- MRI is very sensitive at detecting ligamentous disruption and instability not seen on plan radiographs or CT and demonstrates extent of actual damage to the spinal cord
Early Management

- Extremely important to aggressively support systemic perfusion and oxygenation
- Commonly have gastric dilation which can affect respiration, place NG tube for decompression
- May need intubation to support respiration if high cervical injury
- Resuscitation with intravascular volume and vasopressor
- Early surgical therapy is rarely needed and considered for neurologic deterioration occurring in the face of irreducible subluxation or compression from bone fragments, extruded disk material, or enlarging hematoma
Complications

- Skin breakdown, infections, DVT, autonomic dysreflexia, contractures, spasticity, neurogenic bladder and bowel, and progressive spine deformity

- Single major acute complication of spinal cord injury in children is respiratory with respiratory failure being the most common cause of death in the acute phase of injury
Outcomes

- Mortality of spinal cord injury in childhood has been reported at 28% (majority appear to have occurred at the scene)
- Outcomes are related to the level and severity of injury
- Although limited improvement over time may be seen with complete injuries, full recovery is not expected
- While children with incomplete spinal cord injuries have a good chance of showing significant functional improvement and even complete recovery
- Staggering costs of long term care
References


- UptoDate
  - Severe traumatic brain injury in children: Initial evaluation and management
  - Minor head trauma in infants and children: Evaluation and management
  - Concussion in children and adolescents: Management