FR2-144. Traumatic Brain Injury: Opportunities for Success

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Objectives

Following this session attendees should be able to:
1. Differentiate types of traumatic brain injury.
2. Describe management of traumatic brain injury.
3. Discuss outcomes from traumatic brain injury.
Disclosures

None relevant to this presentation

Vince Vacca May 31, 2013
“If the human brain were so simple that we could understand it, we would be so simple that we couldn’t”
Emerson Pugh, The Biological Origin of Human Values (1977)

The human brain:
• Consists of 100 billion neurons & around 100,000 miles of blood vessels.
• Each neuron has somewhere between 1,000 and 10,000 synapses, equaling about 1 quadrillion synapses.
• There are more than 100,000 chemical reactions happening in the human brain every second.

<table>
<thead>
<tr>
<th>SENSE</th>
<th>MINIMUM STIMULUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vision</td>
<td>A candle flame seen at 30 miles on a dark, clear night</td>
</tr>
<tr>
<td>Hearing</td>
<td>The tick of a clock at 20 feet under quiet conditions</td>
</tr>
<tr>
<td>Taste</td>
<td>One teaspoon of sugar in 2 gallons of water</td>
</tr>
<tr>
<td>Smell</td>
<td>One drop of perfume diffused into the entire volume of six rooms</td>
</tr>
<tr>
<td>Touch</td>
<td>The wing of a fly falling on your cheek from a distance of 1 centimeter</td>
</tr>
</tbody>
</table>
Normal human consciousness

- is defined as the presence of a wakeful arousal state and the awareness and motivation to respond to self and/or environmental events.
- The core areas for maintaining an awake state appear to be in the midbrain and pons.
- These areas activate the central thalamus and basal forebrain. Neurol Clin. 2011
Approximately 1.7 million Americans have a TBI every year.

- This leads to 1.3 million visits to the emergency department, 275,000 hospitalizations, and 52,000 deaths.
- 124,000 remain permanently disabled.
- 50% die from TBI within 2 hours.
- Between ages 14 and 34, there is a high incidence of motor vehicle–related TBI.
- Patients older than 75 have the highest TBI-related mortality.

Critical Care Neurology June 2012; AACN Advanced Critical Care Volume 24 Number 1 January – March 2013
Every 21 seconds a person in the US sustains an TBI

- People who have had a TBI are
  - 1.8 times more likely to report subsequent binge drinking,
  - 1.5 times more likely to be depressed,
  - 2.3–4.5 times more likely to develop Alzheimer’s,
  - 7.5 times more likely to die,
  - 29 times more likely to develop epilepsy.

- Not only are there great personal costs, but there are also profound negative societal consequences; for example, an estimated 87% of prison inmates have incurred a head injury in their lifetime.

Transl Stroke Res. 2011
After mild and moderate head injuries a range of post-concussion symptoms (PCS) are often reported by patients.

Full recovery from PCS usually occurs within three months of the injury.

A significant minority, however, continue to experience symptoms beyond this time.

To date, no means of identifying these patients early after injury has been reported.
Traumatic Brain Injury (TBI)

- There are 1.7 million documented TBI’s annually, with estimates closer to around 3.8 million.
- As the number of participants in youth sports continues to grow, the incidence of brain injury is proportionally increasing as well.
- There is a greater awareness of potential short- and long-term sequelae of athletes who suffer brain injuries such as increased propensity for re-injury, cognitive slowing, early onset Alzheimer's, second impact syndrome, and chronic traumatic encephalopathy (CTE). Rehabil Res Pract. 2012
National TBI Estimates

www.cdc.gov/traumaticbraininjury/statistics.html

Mar 18, 2013

- Of the 1.7 million TBI’s in the USA annually ~ 75% of are concussions or other forms of “Mild TBI”.
- Each year, on average, traumatic brain injuries (TBIs) are associated with an estimated 275,000 hospitalizations, and 52,000 deaths in the US.
Traumatic brain injury (TBI) has long been called a “silent epidemic”

- Many survivors of TBI are left with long term disabilities, and even a “Mild” TBI can leave people with:
  - cognitive impairments,
  - difficulty concentrating,
  - fatigue,
  - headaches.
- The financial expense to the US has been estimated to exceed $56 billion annually. 
  
Front Neurol. 2013
Pre-Hospital management of TBI

• An airway must be established, and arterial oxygen saturation below 90% and systolic blood pressure below 90 mm Hg must be avoided.

• End-tidal carbon dioxide must be maintained between 35 mm Hg and 40 mm Hg.

• Volume status should be assessed and resuscitation begun.

TBI surveillance using the common data elements for traumatic brain injury: a population study.

- About 56.1% of subjects were discharged directly from the ED.
- Of those who were admitted, over half (51%) ended up with an ICU stay, were predominantly those with moderate or severe injury.

The neurological and behavioral sequelae of mTBI develop over the course of days to months after the initial trauma.

- Most behavioral recovery is observed during the first 6 months after injury.
- However, this recovery often plateaus or declines between 6–12 months after the TBI.

Of the Reported 173,285 of TBI’s sports- and recreation-related TBIs among children and adolescents.

- 25% of TBI survivors develop depression,
- 22% develop substance abuse,
- 14% develop post-traumatic stress disorder,
- 9% develop generalized anxiety disorder,
- 9% develop panic disorder,
- 6.4% develop obsessive-compulsive disorder,
- 4.2% develop bipolar affective disorder,

- all of which are increases from the respective prevalence in the general population.

Adolescents are at their peak of physical strength

- resilience, and immune function, yet mortality rates among 15–24 year olds are more than triple the mortality rates of middle school children.
- Adolescents are more vulnerable to nicotine, alcohol, and other drug addictions because the limbic brain regions that govern impulse and motivation are not yet fully developed.
- During puberty, the increases in estrogen and testosterone bind receptors in the limbic system, which not only stimulates sex drive, but also increases adolescents’ emotional volatility and impulsivity.
A significant portion of brain growth and development

- occurring in adolescence is the construction and strengthening of neurocircuitry and pathways
- The frontal lobes are involved in movement control, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, and social and sexual behavior.
- The prefrontal cortex, remains in a process of continuous reconstruction, consolidation, and maturation during adolescence.

Neuropsychiatr Dis Treat. 2013
Dopamine influences brain events that
- Control movement, emotional response, and the ability to experience pleasure and pain.
- Its levels decrease during adolescence, resulting in mood swings and difficulties regulating emotions.

Serotonin plays a significant role in mood alterations, anxiety, impulse control, and arousal.
- Its levels also decrease during adolescence, and this is associated with decreased impulse control.

Melatonin regulates circadian rhythms and the sleep–wake cycle. The body’s daily production of melatonin increases the requirement for sleep during adolescence.
Brain, endocrine and immune system function together

- to maintain homeostasis and prevent disease.
- Multi-organ damage following TBI can lead to increased numbers of infiltrating inflammatory cells and levels of cytokines in the brain.
- The leaky BBB allows the passage of inflammatory molecules and cells into and out of the injured brain initiating a cascade of responses in the brain and other organs.

J Neuroinflammation. 2012
The limbic system

• is a group of structures located deep within the cerebrum.
  – It is composed of the amygdala, the hippocampus, and the hypothalamus.
• These brain regions are involved in the expression of emotions and motivation, which are related to survival.
  – The emotions include fear, anger, and the fight or flight response.
• The limbic system regulates functions related to memory storage and retrieval of events that invoke a strong emotional response.

Neuropsychiatr Dis Treat. 2013
The prefrontal cortex (PFC) offers an individual the capacity to exercise good judgment when presented with difficult life situations.

- Neuroimaging studies have revealed that when interacting with others and making decisions, adolescents are more likely than adults to be swayed by their emotions.

Neuropsychiatr Dis Treat. 2013
Steinberg studied risk-taking behavior in teens

• and how this was influenced by their peers.
• He used a driving simulation game in which he studied teens deciding on whether or not to run a yellow light, and found that when teens were playing alone they made safer decisions, but in the presence of friends they made riskier decisions.

Neuropsychiatr Dis Treat. 2013
According to a report by the Centers for Disease Control and Prevention

- the major cause of death among the teenage population is due to injury and violence related to sex and substance abuse. Neuropsychiatr Dis Treat. 2013
- Injuries are the leading cause of death between the ages of 1 and 44 years.
- Traumatic brain injury (TBI) accounts for 30% of the mortality associated with injury-related death.

Critical Care Neurology June 2012
Monro-Kelli: The contents of the Non-Expandable cranial vault are Brain, CSF, and Blood (arterial and venous).

- In an adult, the brain = 80%, CSF and blood each = 10%.
- An increase in the volume of one compartment or appearance of a new mass lesion must result in a decrease in the volume of the other compartments.

June 2012; Volume 18(3) Critical Care Neurology
Normal Rate of Cerebral Blood Flow

- through the brain of the adult person averages 50 to 65 mL’s / 100 grams of brain tissue / minute.
- For the entire brain, this amounts to 750 to 900 mL’s / min, or ~15% of the resting cardiac output.
- And consumes 20% of available oxygen.

Continuum Lifelong Learning Neurol 2012
Cerebral Perfusion Pressure (CPP) is derived from MAP - ICP.

• CPP can only be determined when ICP measurement is also being performed.

• The current guideline recommendation is to use ICP and CPP-guided therapy.

• A CPP less than 50 mm Hg is associated with poor outcome even if it occurs only periodically.

• Overall goal for CPP should be 50 mm Hg to 70 mm Hg.
• Once intracranial compensatory mechanisms are exhausted, small increases in intracranial volume can cause large increases in ICP
• How can you know where your patient is on the Brain Compliance curve?
• Normal ICP
CBF dys-autoregulation

- also occurs as a result of severe TBI and is associated with poor outcome.
- Dys-autoregulation can be focal or diffuse, and its presence predisposes tissue to hyper-perfusion and vasogenic edema.
- The state of autoregulation can be determined by simple observation of changes in ICP with spontaneous changes in MAP.
- With loss of autoregulation, ICP increases linearly, with increases in MAP even within the postulated autoregulation range.
The forces of impact determine the nature of primary injury from TBI.

- The mechanisms implicated are acceleration-deceleration (motor vehicle accidents, falls), rotational (motor vehicle accidents, falls), crush (struck-by events, motor vehicle accidents), and missile (gun shot, shrapnel).
- Focal lesions include subdural hematomas (SDHs), cerebral contusions with intracerebral hemorrhage, epidural hematoma (EDH), and traumatic subarachnoid hemorrhage (tSAH).
- Diffuse lesions include tSAH and diffuse axonal injury (DAI) due to shear forces.
- The mainstay of reducing primary injury is prevention.

Critical Care Neurology June 2012
Time is Brain

Densely packed, intricately patterned, substrate of mind and awareness, the human brain is a wonder of nature.

- the average number of neurons in the human forebrain is 22 billion.
- In patients experiencing a typical large vessel acute ischemic stroke, 120 million neurons, 830 billion synapses, and 714 km (447 miles) of myelinated fibers are lost each hour.
- In each minute of ischemia, 1.9 million neurons, 14 billion synapses, and 12 km (7.5 miles) of myelinated fibers are destroyed.
- Compared with the normal rate of neuron loss in brain aging, the ischemic brain ages 3.6 years each hour without treatment.

Stroke. 2006; 37: 263-266
Although the consequences of mTBI are not readily appreciated

- it can still cause infrastructural damage to the brain and secondary axonal injury and shows symptoms like cognitive or intellectual deficits and behavioral and personality changes even six months after injury.

- In most patients suffering from mild brain injury, the symptoms disappear within six months but many others suffer in a variety of ways that may be underappreciated and treated inadequately or improperly.

Diffuse brain swelling after TBI is more common in pediatric patients and results from mechanisms different from adults.

- Cerebral white matter (WM) consists largely of densely packed myelinated neuronal axons, and efficient cognitive processing relies on the integrity of these pathways.

- The possibility that the mechanisms, forces, biomechanics, and pathophysiological responses to concussive brain injury differ between adults and children needs to be further explored, because such differences could change the assessment and management of pediatric athletes who sustain Sports-Related Concussion (SRC).

The primary injury of TBI causes DAI, & leads to cerebral edema, intracranial hematoma, elevated ICP, reduced CPP, and cerebral ischemia.

- Therapeutic efforts focus on reducing the secondary insults of hypoxia, hypercapnea, systemic hypotension (Even a single episode of hypotension is a powerful predictor of outcome following TBI) and intracranial hypertension.
The functional outcome of TBI varies widely. Diffuse Axonal Injury (DAI).

- DAI was the only radiologic variable predicting an unfavorable outcome.
- Thus, it is important to identify DAI by applying MRI in the acute phase. DAI - One of the most common primary traumatic brain injuries in patients with severe head trauma (up to 48% in one series)
- Impaired consciousness is usually greater in patients with DAI than in patients without DAI
- Frequent cause of poor clinical outcome in patient with head injuries J Clin Neurol. 2012
"Diffuse axonal injury Imaging"

- Diffuse
- Bilateral
- Majority of lesions (80%) are multiple
- Occur at the gray-white matter junction
- Lesions are most frequently ovoid, larger centrally than peripherally
- Frequently involved are the:
  - Frontal and temporal lobes
  - Caudate nuclei
  - Thalamus
  - Internal capsule
- More frequently associated with hemorrhage
- MRI is the preferred method of study

- Damage occurs at time of injury and then when secondary swelling occurs
- Immediate loss of consciousness
- Most, but not all, have no period of lucidity
- Brainstem functions remain intact so it is rarely a cause of death
Some key points about DAI:

• If the head CT is negative, and all recreational drugs have worn off and the patient still doesn’t wake up, DAI is likely.
• MRI can confirm the diagnosis
• Slow recovery of consciousness or failure to recover correlates with death
• Hyperglycemia and the presence of a subdural also are highly correlated with mortality
• Bottom line: The diagnosis of DAI can generally be made clinically with the assistance of head CT.
• Supportive care, avoidance of complications and early therapy and rehab are the best treatments we have to offer.

Once the brain suffers mechanical insult, the injury process evolves over time and includes

- The secondary, *nonmechanical injury phase*, - progressive, lasts from hours to days, significantly contributing to neurological disabilities.
- Injury to the cerebral vasculature breaks the blood–brain barrier (BBB), allows entry of immune cells and stimulates inflammatory reactions.
- The molecular events result in apoptosis, inflammation, altered plasticity and neuronal regeneration.
- The complex nature of acute and chronic inflammatory reactions may aggravate the pathologic outcome or promote the repair process.
- Also, multi-organ damage in trauma patients can lead to elevated circulatory levels of inflammatory cytokines that may contribute to the post-TBI pathogenesis of the brain and cause multiple organ dysfunction syndrome (MODS) and death.

Post-injury symptomatology

- included the occurrence of loss of consciousness (LOC), the duration of LOC, an alteration of consciousness (AOC), post-traumatic amnesia (PTA), seizure, and vomiting.

- An AOC was considered to be present if the patient reported any of the following: feeling dazed or confused, having difficulty thinking, or if the neurologic exam revealed a decreased (altered) mental status.

- Amnesia has been shown to be most predictive of neurocognitive deficits.


Perm J. 2012 Spring; 16(2): 54–56.
Brain Injury Secondary Cascade

- Among the causes of neurologic dysfunction, structural causes such as ischemic stroke (AIS), intracerebral hemorrhage (ICH), subarachnoid hemorrhage (aSAH), and traumatic brain injury (TBI) carry the worst prognosis and are the greatest challenge to critical care specialists based on the interaction between hypoxemia and secondary neurological insults.

Not all Survive TBI

Natasha Richardson
1963-2009
The Brain Trauma Foundation

- Endorses very elaborate yet clear guidelines for the critical care management of a patient with severe TBI.
- The main principles of therapy are to ensure adequate cerebral perfusion and oxygenation while brain swelling and injury improve.
- Continuum Lifelong Learning Neurol 2012;18(3):532–546

- A sustained ICP greater than 20 mm Hg is considered harmful and is associated with poor outcome.
- Higher ICP causes secondary ischemia to the brain.
- ICP-directed therapy should be used to maintain ICP less than 20 mm Hg.
- TBI guidelines recommend that ICP be monitored in all patients with a severe TBI (GCS score less than 9)
Concussion or mild traumatic brain injury (mTBI) is one of the most common neurologic disorders accounting for approximately 90% of all brain injuries sustained. Such injuries are a common occurrence in athletes with an estimated 1.6–3.8 million sport-related concussion annually in the USA. This can be seen as a gross underrepresentation of the true number as many athletes do not seek medical attention or vocalize their symptoms.

The term mild TBI (mTBI) is now used in place of concussion according to the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO).

- CDC defines a mTBI as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head.
- The American Academy of Neurology (AAN) defines mTBI as a biomechanically induced brain injury resulting in neurologic dysfunction.
- mTBI results in a constellation of physical, cognitive, emotional, and/or sleep-related symptoms and may or may not involve a loss of consciousness (LOC).
- Duration of symptoms is highly variable and may last from several minutes to days, weeks, months, or even longer in some cases. Rehabil Res Pract. 2012
mTBI and concussion are classified by transient loss of consciousness, amnesia, altered mental status

- Glasgow Coma Score of 13 to 15, and focal neurologic deficits following an acute closed head injury.
- Most patients recover quickly, with a predictable clinical course of recovery within the first one to two weeks following traumatic brain injury.
- Persistent physical, cognitive, or behavioral post-concussive symptoms may be noted in 5 to 20 percent of persons who have mTBI.
- Physical symptoms include headaches, dizziness, and nausea, and changes in coordination, balance, appetite, sleep, vision, and hearing.
- Cognitive and behavioral symptoms include fatigue, anxiety, depression, and irritability, and problems with memory, concentration and decision making.

Am Fam Physician. 2012
mTBI (Concussion) is caused primarily by a rotational acceleration of the brain.

- TBI affects the developing brain differently than the fully developed brain.
- Weaker neck muscles and larger head may be a more important issue for female athletes, it is highly likely that soccer is the sport with the greatest concussion risk (multiple Class I studies).
Teammates attempted to rouse Tim Tebow after the hit. "I think it's a concussion," Florida coach Urban Meyer said afterward. "I think he'll be all right. "He took a pretty good shot."
Solid White is Skull Bone
White Mixed With Gray is Diffuse Subarachnoid Blood
Dilated Temporal Horn

Axial CT

Epidural hematoma

Coronal CT

A

B

A

B

C

D

Coronal CT

Red arrow indicates
Blue arrow indicates
Therapies to Lower ICP

• The head of the bed should be elevated 30 to 45 degrees.
• This maximizes cerebral venous drainage via jugular veins.
• However, the patient should be inclined only if the spine has been deemed stable.

• Venticulostomy
• Hyperosmolar therapy
• Sedation and analgesia
• Hyperventilation
• Muscle relaxation
• Hypothermia
• Barbiturate coma
• Decompressive craniectomy
What do we do?

• Assess: LOC, RASS, CAM, GCS, NIHSS
• Pupils/Reactions – Pupillometer
• EOM’s/Gaze
• Visual fields
• Motor strength
• Sensory
• Cranial Nerves (Pawtucket)
• Brain Stem Reflexes
Glasgow Coma Scale (Injury & Function)

- The motor component of the GCS score is a powerful predictor of outcome and contains most of the predictive power of the score.
- "Flexing to pain" or worse, requires intubation for airway protection.
- GCS does not just measure brain injury but also brain function which can be affected by many (Non-Relevant to the injury) conditions.
A GCS score of:
- 13 or higher correlates with a mild brain injury,
- 9 to 12 is a moderate injury,
- 8 or less is a severe brain injury.
- GCS of 3 w/ severe TBI and clinical indications of herniation: bilateral fixed, non-reactive pupils = grim prognosis

• GCS – has value however: your patient can be drunk and score (8 or less) and be “fine” the next day or........your patient can sustain a mTBI (and unless you see him/her at the scene) score a 15 and be dead in hours.
• That’s why the history leading up to and immediately after injury is so important.
• That information will guide next steps beyond simple “neuro assessment”.

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
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</thead>
<tbody>
<tr>
<td><strong>Eye Opening</strong></td>
</tr>
<tr>
<td>Spontaneous</td>
</tr>
<tr>
<td>To verbal command</td>
</tr>
<tr>
<td>To pain</td>
</tr>
<tr>
<td>No response</td>
</tr>
<tr>
<td><strong>Verbal Response</strong></td>
</tr>
<tr>
<td>Oriented and converses</td>
</tr>
<tr>
<td>Disoriented and converses</td>
</tr>
<tr>
<td>Inappropriate words</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
</tr>
<tr>
<td>No response</td>
</tr>
<tr>
<td><strong>Motor Response</strong></td>
</tr>
<tr>
<td>Obey verbal commands</td>
</tr>
<tr>
<td>Localizes pain</td>
</tr>
<tr>
<td>Withdraws from pain (flexion)</td>
</tr>
<tr>
<td>Abnormal flexion in response to pain (decorticate rigidity)</td>
</tr>
<tr>
<td>Extension in response to pain (decerebrate rigidity)</td>
</tr>
<tr>
<td>No response</td>
</tr>
</tbody>
</table>
TBI surveillance using the common data elements for traumatic brain injury: a population study.

• We did note, for instance, that mTBI had unexpected amounts of pathology on CT (21% had bleeds, 13% had fractures), neurosurgical intervention rates (3.8%), and death rates (0.8%).

• This higher than expected acuity in this group may indicate the GCS scale was an underestimation of illness or that the medical community’s assumptions about “mild” TBI may be underestimating the disease process.

NIH-Stroke Scale: stroke-specific quantitative (0-42) scale assesses:

- Level of consciousness, (Brainstem, either/both hemisphere)
- Language function, (Left hemisphere)
- Neglect, (Right hemisphere)
- Visual field, (Either hemisphere, occipital lobe)
- Eye movements, (Brainstem, both hemispheres)
- Facial palsy, (Either hemisphere)
- Motor strength, (Either hemisphere, basal ganglia, brainstem)
- Sensory function, (Thalamus, basal ganglia)
- Coordination (Cerebellum)
- Associated with final outcome after a stroke in terms of length of stay, survival, and discharge destination.
- Weighted toward signs caused by anterior-circulation infarction
- Scores differ between hemispheres as well

<table>
<thead>
<tr>
<th>NIH STROKE SCALE ITEM</th>
<th>Scoring Definitions</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>3a. LOC</td>
<td>0=alert and responsive 1=arousable to minor stimulation 2=arousable only to painful stimulation 3=reflex responses or unarousable</td>
<td></td>
</tr>
<tr>
<td>4b. LOC Questions--Ask pt's age and month. Must be exact.</td>
<td>0=Both correct 1=One correct (or dysarthria, intubated, foreign lang) 2=Neither correct</td>
<td></td>
</tr>
<tr>
<td>4c. Commands--open/close eyes, grip and release non-paretic hand, (Other 1-step commands or mimic ok)</td>
<td>0=Both correct (ok if impaired by weakness) 1=One correct 2=Neither correct</td>
<td></td>
</tr>
<tr>
<td>5a. Best Gaze--Horizontal EOVM by voluntary or Doll's.</td>
<td>0=Normal 1=partial gaze palsy; ahhn gaze in 1 or both eyes 2=Forced eye deviation or total paresis which cannot be overcome by Doll's.</td>
<td></td>
</tr>
<tr>
<td>6a. Visual Field--Use visual threat if nec. If monocular, score field of good eye.</td>
<td>0=No visual loss 1=Partial hemianopia, quadrantanopia, extinction 2=Complete hemianopia</td>
<td></td>
</tr>
<tr>
<td>7a. Facial Palsy--If stuporous, check symmetry of grimace to pain.</td>
<td>0=Normal 1=partial paralysis, flat NLF, asymm smile 2=partial paralysis (lower face=UMN) 3=complete paralysis (upper &amp; lower face)</td>
<td></td>
</tr>
<tr>
<td>8a. Motor Arm--arms outstretched 90 deg (sitting) or 45 deg (supine) for 10 secs. Encourage best effort. Circle paretic arm in score box</td>
<td>0=No drift x 10 secs 1=Drift but doesn't hit bed 2=Some antigravity effort, but can't sustain 3=No antigravity effort, but even minimal mt counts 4=No movement at all x=unable to assess due to amputation, fusion, fx, etc.</td>
<td></td>
</tr>
<tr>
<td>9a. Motor Leg--raise leg to 30 deg supine x 5 secs.</td>
<td>0=No drift x 5 secs 1=Drift but doesn’t hit bed 2=Some antigravity effort, but can’t sustain 3=No antigravity effort, but even minimal mt counts 4=No movement at all x=unable to assess due to amputation, fusion, fx, etc.</td>
<td></td>
</tr>
<tr>
<td>10a. Limb Ataxia--check finger-nose-finger ; heel-shin; and score only if out of proportion to paralys</td>
<td>0=No ataxia (or aphasic, hemiplegic) 1=ataxia in upper or lower extremity 2=ataxia in upper AND lower extremity x=ataxia in upper AND lower extremity</td>
<td></td>
</tr>
<tr>
<td>11a. Sensory--Use safety pin. Check grimace or withdrawal if stuporous. Score only stroke-related losses.</td>
<td>0=Normal 1=mod-mild unilateral loss but pt aware of touch (or aphasic, confused) 2=Total loss, pt unaware of touch. Coma, bilateral loss</td>
<td></td>
</tr>
<tr>
<td>12a. Best Language--Describe the cookie jar picture, name objects, read sentences. May use repeating, writing, stereognosis</td>
<td>0=Normal 1=mod-mild aphasia; (diff but partly comprehensible) 2=severe aphasia; (almost no info exchanged) 3=mute, global aphasia, coma. No 1 step commands</td>
<td></td>
</tr>
<tr>
<td>13a. Dysarthria--read list of words</td>
<td>0=Normal 1=mild-mild; slurred but intelligible 2=severe; unintelligible or mute x=intubation or mech barrier</td>
<td></td>
</tr>
<tr>
<td>14a. Extinction/Neglect--simultaneously touch patient on both hands, show fingers in both vis fields, ask about deficit, left hand.</td>
<td>0=Normal, none detected. (vis loss alone) 1=Neglects or extinguishes to double simultaneous stimulation in any modality (vis, aud, sens, spatial, body parts) 2=profound neglect in more than one modality</td>
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</table>

- **Stroke severity scores:**
  - 0 to 5: mild stroke
  - 6 to 15: moderate stroke
  - 16 to 20: moderate to severe stroke
  - > 20 severe stroke
- NIHSS <5 most strongly associated with D/C home
- NIHSS 6-13 most strongly associated with D/C to rehab
- NIHSS >13 most strongly associated with D/C to nursing facility

Schlegel et al., 2003; 2011 American Heart Association
This concussion assessment tool contains an assessment of orientation, memory, concentration, balance & symptoms.

- This tool is intended to be used in conjunction with your clinical judgment.
- If ANY significant abnormality is found, a conservative, "safety first" approach should be adopted.
- An athlete suspected of sustaining a concussion is a "No Go" and does not return to play in the same game or practice.
ANY OF THE FOLLOWING ARE OBVIOUS SIGNS OF DISQUALIFICATION (i.e. "No Go"):  

1) LOC or unresponsiveness? (for any period of time) If so, how long? ______________ Y N  
2) Confusion? (any disorientation or inability to respond appropriately to questions) Y N  
3) Amnesia (retrograde / anterograde)? If so, how long? ____ Y N  
4) New and/or persistent symptoms: see checklist? (e.g. headache, nausea, dizziness) Y N  
5) Abnormal neurological finding? (any motor, sensory, cranial nerve, balance issues, seizures) or Y N  
6) Progressive, persistent or worsening symptoms? If so, consider cervical spine and/or a more serious brain injury (See box below) – Next slide for us.
Neurological Screen for Cervical Spine and/or More Serious Brain Trauma

- Deteriorating mental status? Y/N
- Any reported neck pain, cervical spine tenderness or decreased range of motion? Y/N
- Pupil reaction abnormal or pupils unequal? Y/N
- Extra-ocular movements abnormal and/or cause double vision? (difficulty tracking and/or reading) Y/N
- Asymmetry or abnormalities on screening motor or sensory exam? Y/N
<table>
<thead>
<tr>
<th>Mydriasis</th>
<th>Miosis</th>
</tr>
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<tbody>
<tr>
<td>CN III damage</td>
<td>Carotid Artery injury in neck or skull base</td>
</tr>
<tr>
<td>Unilateral = Transtentorial/Uncal herniation</td>
<td>Horner’s Syndrome – Miosis w/ ipsilateral ptosis and anhydrosis</td>
</tr>
<tr>
<td>Seizure/Post-Ictal</td>
<td>Hypothalamic, cervicothoracic or direct orbital injury</td>
</tr>
<tr>
<td>Atropine/Sympathomimetics</td>
<td></td>
</tr>
<tr>
<td>Injury Location</td>
<td>Abnormality</td>
</tr>
<tr>
<td>-----------------------------------------------------</td>
<td>--------------------------------------------------</td>
</tr>
<tr>
<td>Cavernous Sinus / Supra-Orbital Fissure</td>
<td>CN III, IV, &amp; VI</td>
</tr>
<tr>
<td>Transtentorial (Uncal) herniation</td>
<td>CN III</td>
</tr>
<tr>
<td>Raised ICP (false localizing sign)</td>
<td>Isolated Abducens(6) palsy</td>
</tr>
<tr>
<td>Frontal eyes field (brodman’s area 8)</td>
<td>Ipsilateral tonic conjugate deviation</td>
</tr>
<tr>
<td>Seizure involving frontal eyes field</td>
<td>Conjugate deviation to contralateral side</td>
</tr>
<tr>
<td>Occipital lobe injury (unilateral)</td>
<td>Hemianopsia + ipsilateral conjugate gaze preference</td>
</tr>
</tbody>
</table>
## Brainstem Reflexes

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Location and Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial Palsy - unilateral</td>
<td>CN VII – basilar skull fx’s.</td>
</tr>
<tr>
<td>Corneal Reflex</td>
<td>Rostral Pontine</td>
</tr>
<tr>
<td>Dolls’ Eyes</td>
<td>Vestibulo-ocular CN VIII</td>
</tr>
<tr>
<td>Cold Calorics</td>
<td>COWS – Normal Coma – ipsilateral deviation Stuporour/Obtunded – nystagmus to contralateral</td>
</tr>
<tr>
<td>Gag &amp; Cough</td>
<td>CN’s IX &amp; X, and Brainstem swallowing centers</td>
</tr>
<tr>
<td>Respiratory variation (Cheynes-Stokes)</td>
<td>B/L hemispheric/diencephalic injury to upper pons</td>
</tr>
<tr>
<td>Ataxic breathing</td>
<td>Medullary respiratory generator center</td>
</tr>
</tbody>
</table>
# Deep Tendon and Superficial Reflexes

- **DTR’s exaggerated after TBI due to cortical dis-inhibition**
- **Decreased/Absent if Spinal Cord Injured**
- **Asymmetric DTR’s – Unilateral brain/spinal injury**

<table>
<thead>
<tr>
<th>Normal Babinski</th>
<th>Intact descending corticospinal inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive Babinski</td>
<td>Interrupted inhibition pathways</td>
</tr>
<tr>
<td>Neuro-Diagnostic Evaluation</td>
<td></td>
</tr>
<tr>
<td>----------------------------</td>
<td></td>
</tr>
<tr>
<td><strong>CT</strong></td>
<td></td>
</tr>
<tr>
<td>1. Penetrating head trauma</td>
<td></td>
</tr>
<tr>
<td>2. Basilar/depressed skull fx</td>
<td></td>
</tr>
<tr>
<td>3. Post-traumatic seizure</td>
<td></td>
</tr>
<tr>
<td>4. Severe Head Injury</td>
<td></td>
</tr>
<tr>
<td>Especially when a/w:</td>
<td></td>
</tr>
<tr>
<td>Altered Level of Consciousness</td>
<td></td>
</tr>
<tr>
<td>Focal Deficits</td>
<td></td>
</tr>
<tr>
<td>Persistent Headaches /emesis</td>
<td></td>
</tr>
<tr>
<td><strong>MRI</strong></td>
<td></td>
</tr>
<tr>
<td>Superior to CT in subacute and chronic phases of injury to detect contusions/shearing in white matter.</td>
<td></td>
</tr>
<tr>
<td>Essential for Spinal Cord</td>
<td></td>
</tr>
<tr>
<td><strong>Cerebral Angiography</strong></td>
<td></td>
</tr>
<tr>
<td>Carotid/vertebro-basilar dissections/occlusions and aneurysms (pseudo-aneurysms)</td>
<td></td>
</tr>
</tbody>
</table>
Assessment Findings

- CN III indicative of expanding intracranial ‘lesion’ because herniation of medial temporal lobe directly damage CN III = Pupil Dilation/Impaired reaction to light – Ipsilateral to injury (unless bilateral)

- Evaluation of eye movements is prognostic: 3rd and 6th nerve palsies are common indicators of raised ICP.

- Abnormal eye movements may result from brainstem dysfunction, damage to CN’s III, IV, VI or VIII

- Absent eye movements w/ low level of responsiveness indicates poor prognosis

- Limb weakness/deficit – useful sign but not so much for localizing site of lesion (Kernohans Notch) 

- Right hemisphere transtentorial herniation, causes a Kernohan's notch in the left cerebral peduncle which results in right-sided motor impairment.
- This phenomenon can be clinically confusing since it is a secondary condition caused by a primary condition in the opposite hemisphere of the brain = false localizing sign.
Assessment Findings

- Post traumatic amnesia can reflect the extent/severity of the TBI and can last for weeks
- Headache & Vomiting – common following TBI but if prolonged may indicate intracranial hematoma
- Consider hyper-extension injury to cervical spine w/ frontal lacerations/bruising a/w TBI
- Rhinorrhea/Otorrhea, conjunctival hemorrhage, Battles/Raccoons can all indicate basal skull fracture and risk for meningitis

Indications for brain computed tomography scan after minor head injury (MHI).

• WE SUGGESTED THAT ABNORMAL BRAIN CT SCAN RELATED TO THE TRAUMA AFTER MHI CAN BE PREDICTED BY THE PRESENCE OF ONE OR MORE OF THE FOLLOWING RISK INDICATORS:
  – Headache,
  – vomiting,
  – LOC,
  – Amnesia,
  – Alcohol intoxication.

• Any patient with these indicators following MHI, must be considered as a high-risk MHI. J Emerg Trauma Shock. 2011
In game three - 6/6/2011 Stanley Cup Finals, #18 Nathan Horton was hit with late check to the head by Vancouver Canucks defenseman Aaron Rome.
Horton was later diagnosed with a **severe concussion** and missed the remainder of the 2011 Stanley Cup Finals

- Convulsive Concussion – LOC ~ 40 seconds, woke w/ complete amnesia
- Didn’t know his trainer or where he was
- CT normal (Need an MRI)
- The Bruins went on to win the series in game seven, and Horton hoisted the Stanley Cup in Vancouver on June 15.
There is increasing evidence showing that TBI is associated with neurodegenerative diseases like Alzheimer’s disease (AD), Parkinson’s disease (PD), multiple sclerosis (MS), and amyotrophic lateral sclerosis (ALS).

- Epidemiological data indicates a single TBI event may trigger or accelerate the onset of Alzheimer’s disease (AD) in later life.
- Repetitive mTBI has been associated with progressive neurodegeneration.
- Since, Rudelli et al. reported a case of classic AD pathology in a 38 year old severe head trauma patient, both tau pathologies and beta-amyloid (Aβ) plaques were identified in survivors of single TBI.
- Subsequently, cases of AD-like pathology including neurofibrillary tangles and Aβ deposition were reported in head trauma victims, including boxers, irrespective of age. J Neuroinflammation. 2012
The traumatic tearing of neuronal connections (axonal shearing) disconnects or impairs cortical circuitry, thalamic circuitry, or both, contributing to cognitive impairment and dementia.
• The 43-year-old linebacker died May 2 of a self-inflicted gunshot wound.

• If a 'Grade 1' concussion means you see stars after a hit, Plummer says “he's had 1,000 in his career, and his ex-teammate, Junior Seau, had 1,500”.

• Plummer played beside Seau at linebacker in four years with the San Diego Chargers. Robert Klemko, USA TODAY

• Seau was diagnosed with chronic traumatic encephalopathy, or CTE, after his death.
"In the 1990s, I did a concussion seminar. They said a Grade 3 concussion meant you were knocked out, and a Grade 1 meant you were seeing stars after a hit, which made me burst out in laughter," Plummer told the San Jose Mercury News. "As a middle linebacker in the NFL, if you don't have five of these (Grade 1 effects) each game, you were inactive the next game.

Seau's family has agreed to have his brain studied for the effects of concussions.

Former Chicago Bears safety Dave Duerson last year also died of a self-inflicted gunshot wound to the chest. He left a suicide note asking his family to donate his brain to Boston University, where researchers learned he suffered from a neurodegenerative disease linked to concussions.
Recently researchers have begun to explore the inter-relationship between homelessness and (TBI).

- TBI often occurs among young persons, affecting prime working years.
- Evidence, predominantly from individuals with more severe TBI, suggests that cognitive, physical and emotional consequences may persist and place individuals at risk for social failure.
- TBI is associated with low subsequent employment rates which can contribute to a downward spiral to homelessness.
- It is also suggested that in the homeless population, cognitive impairment may increase the risk of remaining homeless, illustrating the potential for TBIs to contribute to the chronicity of homelessness.

The findings of the few studies identified pointed to high rates of TBI among people who are homeless.

- Two studies that explored the relationship between homelessness and TBI revealed that for the majority of participants, the first incidence of TBI occurred before the onset of homelessness, suggesting that TBI may be a risk factor for homelessness.

- Certain health conditions are common to both the homeless and TBI populations.
  - the rate of mental disorders among individuals who are homeless is high and is estimated to be between 80%-90% in countries such as the US and Canada.
  - psychiatric disorders are also highly prevalent in individuals post-TBI, with estimates of rates ranging between 18%-65%
William P. Meehan III, MD

- is Director of the Micheli Center for Sports Injury Prevention, Director of the Sports Concussion Clinic at Boston Children’s Hospital, Director of Research for the Brain Injury Center at Boston Children’s Hospital, and an Assistant Professor of Pediatrics at Harvard Medical School.
- He is a member of the American College of Sports Medicine and the American Medical Society for Sports Medicine.
- He serves on the Section on Emergency Medicine for the American Academy of Pediatrics, the Advisory Board of the Sports Legacy Institute, and on the Advisory Committee for Sports Head Injuries for the Commonwealth of Massachusetts Department of Public Health.
- He is the team physician for the Northeastern University Men’s Ice Hockey Team.
A few mTBI facts – Dr. Meehan

- Nearly one fourth of concussed athletes (23.5%) had resolution of their symptoms within 24 hours of the injury.
- Of all athletes concussed, most (77.9%) had resolution of their symptoms within 7 days of injury, while 19.2% of concussed athletes had symptoms that lasted longer than 1 week but resolved within 1 month.
- Only 2.8% of concussed athletes had symptoms that lasted longer than 1 month.
- Female athletes appeared more likely to have symptoms lasting more than 7 days from the time of injury (26.4% vs 19.6%, P = .05).

- We found that concussion accounts for nearly 15% of all sport-related injuries in US high schools.
- Returning to play before symptom resolution can be associated with poor outcomes, such as second-impact syndrome.
Rules for Student Athletes

- Once a student athlete is placed under the care of the Athletic Training Staff he/she must be released by a member of the staff prior to practice/competition.
- Clearance for participation may be withheld if the student athlete fails to show for treatment/rehabilitation or keep off campus referrals.
- Failure or negligence in following rehabilitation or therapy prescribed by the athletic trainers, physicians or orthopedic surgeon shall render a student athlete liable to suspension from practice and/or competition.
- Coaches will be kept apprised of all prescribed rehabilitation and therapy.
NCAA – mTBI (Concussion)

• First, they should be removed from play as soon as a concussion is suspected and evaluated by an appropriate health-care professional, such as a certified athletic trainer, team physician or a health care professional experienced in concussion evaluation and management.

• Athletes with a concussion are not allowed to return to the game that day.

• They should not return to play until all symptoms have been resolved during rest and exertion.

• A health care professional with experience in evaluating concussions should clear the student-athlete before returning to play is considered.

www.ncaa.org/wps/wcm/.../Concussion
Post-TBI challenges

- Cognitive
- Slower processing of information
- Loss of short-term memory
- Poor planning, organizing skills
- Setting goals
- Completing tasks
- May act impulsively
- Difficulty solving problems

- Communication
- Trouble finding words
- Difficulty understanding written & verbal communications
- Difficulty spelling, reading, writing
- Inappropriately interrupting conversations
- Inability to follow conversation
- Using inappropriate tone of voice
- Unable to process subtleties/nuances in language (e.g. difference between tongue-in-cheek and seriousness)
What happens when students with TBI go to college?

• Injured before college
• Lower graduation rates (National Longitudinal Transition Study, Wagner et al., 2005)
• Individualized Education Program (IEPs) & transition teams are important to successful college entry (Glang et al., 2008)
• About half are linked to campus disability services (Tobis & Glang, 2008)
• Are the needs of students w/ TBI the same as those w/ learning disabilities?

• Difficulty with academics, like studying, homework, tests
• Problems making decisions
• Difficulty with relationships
• Physical impairment: arm/hands (writing)
• Mood changes
• Anger
• Physical impairment: legs (walking)
• Substance/alcohol abuse
• Memory problems
• Dizziness
• Headaches
• Attention problems
• Fatigue
• Organization problems
• Depression
• Difficulty maintaining friendships
Selected References

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