Definition- Traumatic Brain Injury (TBI)

- A traumatically induced physiologic disruption of brain function with the presence of any one of the following:
  - Loss of consciousness
  - Anterograde or retrograde memory loss
  - Alteration of mental status
  - Focal neurologic deficit

- American Congress Rehabilitation Medicine
Definition- Mild TBI (MTBI)

• Loss of consciousness must be less than 30 minutes
• GCS 13-15
• Post traumatic amnesia not greater than 24 hours
• This definition is inclusive of a broad range of injuries
Definition of Concussion

• A complex pathophysiological process affecting the brain induced by traumatic biomechanical forces
  – (Consensus Statement on Concussion in Sport- 2009)

• This is Mild Traumatic Brain Injury!
  – Controversy over terminology reflects struggle for appropriate assessment / Rx
Concussion - definition cont

- Due to direct blow or by an impulsive force
- Rapid symptom onset followed by spontaneous resolution
- Clinical symptoms largely reflect dysfunction, rather than structural damage
- Results in graded set of symptoms, with or without loss of consciousness
- Standard structural neuroimaging is normal
Complicated MTBI

- GCS 13-15 with positive CT scan
- Higher incidence of cognitive and psychosocial symptoms at 6 months
- Similar functional recovery to moderate TBI
Epidemiology

• TBI Incidence- CDC estimate (Langlois et al)
  – 1.5 million per year
  – 250K hospital admits
  – 50K rehab admits
  – Likely underestimated, as taken from ED data and reliant on coding

• 80% Mild

• Sports concussion estimated at 1.6-3.8 million per year
Pathophysiology

- Mechanical trauma to the brain
- Rotational and/or acceleration/deceleration forces differentially affect brain tissues
- Complex cascade of neurochemical/metabolic events
- Disruption of neuronal cell membranes and axonal stretching with indiscriminate ion flux
Pathophysiology

- Widespread release of neurotransmitters, particularly excitatory
- Diffuse, relative suppression of neurons ensues
- Restoration of ionic balance requires high levels of glucose metabolism
- Lactate produced, can contribute to cerebral edema
Pathophysiology

- Changes in cerebral blood flow
- Neuronal dysfunction without significant acute cell death
- Results in post concussive metabolic vulnerability
Imaging

• Acute consideration: CT scan
• Not required for majority of patients
• Standard CT and MRI normal in 99% MTBI
Imaging

• Risk factors associated with need of neurosurgical intervention:
  – GCS < 15 after 2 hours
  – Suspected skull fracture
  – Vomiting
  – Age > 65
  – Prolonged retrograde / anterograde amnesia
  – Intoxication
Imaging

• No defined clinical role for PET or SPECT-experimental, looking at metabolic effects
• Diffusion tensor imaging visualizes the integrity of white matter tracts- early studies showing correlation with symptoms post MTBI
• Magnetic Resonance Spectroscopy also under study
Diagnosis

- EEG not yet sensitive or specific enough
- Serum markers pursued, none are yet promising:
  - Neuron enolase
  - S100 proteins
  - Tau protein
  - Alpha II- Spectrin
Recovery

- Majority of individuals become asymptomatic within 3 months
- 8-33% continue to report distressing symptoms
Post Concussion Syndrome

- Cognitive
- Physical
- Emotional / Behavioral
- No specific predictors for development
Post Concussive Syndrome

- DSM IV criteria
- Formal testing evidence of attention / memory problems
- 3 or more of the following causing disturbed function:
  - Fatigue
  - Sleep disorder
  - HA
  - Vertigo
  - Irritability/Aggression
  - Anxiety, Depression or Lability
  - Personality change
  - Apathy
Post Concussive Syndrome

• Risks for developing
  – Female, >40 years
  – Litigation / Compensation
  – Previous TBI, psychiatric illness
  – Hx EtOH
  – Poor pre-injury cognitive ability
  – Low SE status, poor psychosocial function
Somatic

- HA
- Vertigo
- Blurred vision
- Photophobia
- Phonophobia
- Poor sleep
- Decreased sense of smell
Headache

- Most common somatic symptom (25-78%)
- Prevalence, duration and severity of headache is greater in MTBI than severe TBI
- May be of tension or migraine pattern
- Often correlates with fatigue, both physical and cognitive, indicating need for activity modification
Headache

• Analgesia overuse complicated 42% in one study
• Other post traumatic causes
  – TMJ
  – Occipital neuralgia
  – Trigeminal nerve injury
  – Dysesthesia over scalp injury
  – CSF leak
  – SAH
  – Carotid or vertebral artery disruption
Headache Treatment

• Avoid overuse of analgesics and risk of rebound headaches
• Be aware of side effects of medications—somnolence, dizziness, cognitive impairment, nausea etc
• Treat whiplash
  – Therapy
  – Address spasms
  – Physical modalities
• Anti-inflammatories—judiciously
• Migraine meds
  – Prophylactic and abortive strategies
• Neuropathic pain meds
  – Amitriptyline
  – Neurontin
  – AEDs
Headache Treatment

- DHEA and metaclopramide infusions
- Occipital nerve blocks
- Botox injections
- Biofeedback
- Aromatherapy
Vertigo

• Usually resolves in 7 days
• Etiologies:
  – Labyrinthine concussion- position related
  – Cervicogenic- controversial, neck restrictions
  – Perilymphatic fistula- Rupture of oval or round window with sudden unilateral sensorineural hearing loss, acute persistent vertigo and ataxia w/gradually improving course
  – Endolymphatic hydrops “Meniere’s”—disturbance of fluid transport
  – Temporal bone fx with CN VIII involvement
• ENT referral for severe and/or persistent symptoms
• Balance therapy helpful and labor intensive. Typical course completion may take 6-8 months
• Techniques designed to facilitate accommodation
Pain

- Often myofascial
- Coexists with physical, cognitive and emotional dysfunction
- Early education and intervention
- Can pursue Pain Psychology evaluation
Cognitive

- Memory Impairment
- Difficulties in concentration
- Decreased speed of processing
- Difficulties with learning
- Fatigue
Cognition

- Education, reassurance
- Staged return to vocational activities
- Cognitive rehabilitation measures to develop effective compensatory strategies
- Respect for role of fatigue
Cognition

- Formal testing not indicated in all patients
- Skill of practitioner a large factor in validity
- Baseline testing very helpful (often there for professional athletes)
- Sensitivity / ceiling effect
- Guidance for cognitive strategies and return to work
Sleep Disturbance

• Often multifactorial:
  – Physiologic alterations in arousal
  – Pain
  – Psychologic factors
  – Pre-injury factors
Sleep Disturbance

• Sleep hygiene
  – Routine
  – Caffeine, nicotine
  – Environment
Sleep disturbance

• Medications- early use appropriate
  – Trazadone 50-200 mg qhs
  – Sonata / Lunesta
  – Ambien ? Cognitive side effects
  – Avoid benzodiazepines secondary to addictive and depressive properties
  – Rozerem under study. Targets circadian rhythm through melatonin receptor agonist mechanism
Psychological

- Depression
- Anxiety
- Irritability
- Sleep disturbance
- Fatigue
- More common in those with history of psych disorders
Early Intervention

- Education in the emergency department regarding probable acute symptoms very helpful in outcomes.
- Reassurance of natural history for symptom resolution in majority of patients.
- Decreased symptoms at 3 months in 202 adults and pediatric patients who received education on concussion and post concussive syndrome (Ponsford et al)
Risk of Multiple Concussions

- In multiple studies, a history of prior concussion was associated with a higher rate of subsequent concussion.
- Symptom duration was longer in those with prior concussions.
- There may be long term cognitive sequelae.
Dementia Pugilistica

- Classically renowned neurodegenerative disorder
- First recognized in 1928- associated with multiple concussive blows in boxers
- Dementia
- Movement disorders- Parkinsonism
Second Impact Syndrome

- Second often minor blow in patient who is still symptomatic from a recent concussion
- Diffuse cerebral swelling—often fatal
- Prevalence and Incidence remain unknown
- 1st described in 1973 by Richard Schneider
- Approx 20 reported cases all <20 yo, 1998 review 17 cases, has been demonstrated in animals
- Proposed mechanism—disordered cerebral autoregulation—cerebrovascular congestion—malignant cerebral edema
Second Impact Syndrome

- Proposed mechanism is disordered cerebral autoregulation
  - Autoregulatory failure noted in 20-30% mTBI and 80% severe TBI
  - Inability to respond to blood pressure gradient changes normally
  - Traumatic catecholamine surge
  - Cerebrovascular congestion-malignant cerebral edema
Chronic traumatic encephalopathy

• Pathologic finding of tau protein in brain tissue of athletes with history of multiple concussions
• Clinical symptoms: memory loss, violent outbursts, mood disturbance, cognitive decline, eventual movement abnormalities
  – (McKee A et al)
Blast Exposure

- Very common in current conflicts in Iraq and Afghanistan
- Rand self report study estimates 300K individuals may have sustained TBI
- Complex setting for mechanism of injury: primary blast wave, physical forces, other injuries
Blast Exposure

- Mild TBI in blast setting results in higher proportion of symptomatic individuals
- Psychological factors difficult to elucidate
Types of Blast Injury

- Primary - barotrauma
- Secondary - effects of projectiles
- Tertiary - from structural collapse and displacement
- Quaternary - explosion related injuries, not from the above mechanisms
Primary

• Over pressurization or under pressurization relative to atmospheric pressure. The wave dissipates quickly, causing the greatest risk of injury to those closest to the explosion.
• Air filled organs and air-fluid interfaces are susceptible
• Rupture of tympanic membranes, pulmonary damage and rupture of the colon.
• Brain injury via concussion or air embolism.
• Eye injuries- globe rupture, serous retinitis
The tympanic membrane (TM) can rupture with only 5 psi over atmospheric pressure.

Other organs are damaged at far higher pressure gradients, 56-76 psi.

If there is no rupture of the TM, primary blast injury of other organs is unlikely.

Pulmonary barotrauma or "blast lung" is the most common fatal primary injury - pulmonary contusion or systemic air embolism.
Brain Injuries

• LOC and contusions were previously considered secondary or tertiary injuries, but with widespread use of body armor, CNS damage is increasingly attributed to the direct effects of blast.
• Direct concussive force of the blast wave?
• Effect of a sudden rush of blood volume into, then out of, the intracranial system?
Brain Injuries

- Mild to moderate brain injuries may initially go undetected, particularly if there are other life threatening injuries present, or if there is a setting of mass casualty.
- First responders must address life threatening conditions as a priority.
- Many witnessing a traumatic event can be psychologically stunned - hard to distinguish from altered mental status of concussion.
Post traumatic stress reactions

- Blast injury survivors are returning with high rates of PTSD. Many recall the scene of the blast, being wounded, wondering if they would survive, and seeing others killed or severely injured.
- Individuals with TBI and PTSD often present with similar complaints-
Mild TBI and PTSD: Overlapping Symptoms and Diagnostic Clarification

<table>
<thead>
<tr>
<th>Mild TBI</th>
<th>PTSD</th>
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<tbody>
<tr>
<td>Insomnia</td>
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<tr>
<td>Impaired memory</td>
<td>Impaired memory</td>
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<tr>
<td>Poor concentration</td>
<td>Poor concentration</td>
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<tr>
<td>Depression</td>
<td>Depression</td>
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<tr>
<td>Anxiety</td>
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<tr>
<td>Irritability</td>
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<tr>
<td>Fatigue</td>
<td>Emotional Numbing</td>
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<tr>
<td>Headache</td>
<td>Flashbacks/Nightmares</td>
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<tr>
<td>Dizziness</td>
<td>Avoidance</td>
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<tr>
<td>Noise/Light intolerance</td>
<td></td>
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</tbody>
</table>
Potential Clinical Presentation

**PTSD**
- Flashbacks
- Nightmares

**TBI**
- Headaches
- Dizziness

Intersections:
- Attentional problems
- Depression
- Irritability
- Anxiety
Blast Exposure

• Recent VA Clinical Practice guideline emphasize importance on focusing on symptom management
• Challenge of classifying this syndrome with previously understood clinical presentations
• Categorization / disability process very controversial
Return to Work

- Consideration of workplace demands
- Multitasking
- Vertigo / Heights
- Cognitive Headache
- Safety / Well being of others
- Necessity of accuracy
Return to Work

• Define tasks and consider modification
  – Part time trial
  – Decreased stress for evaluation or performance
  – Decreased “busy work”
  – More time to complete tasks
  – Organization support
  – Memory cues

• Environmental modification
  – Lighting
  – Ear plugs
  – Decreased competing stimulation
Summary

• Approximately 25% of traditional MTBI patients will develop post-concussion syndrome
• Blast exposure syndrome may have very different physiology and natural history
• Imaging and markers still under investigation
• Early education best Rx and decreases late symptoms
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