Objectives

- Understand the physical, cognitive, and emotional complications of a mild brain injury
- Identify appropriate management strategies to maximize outcomes in those who have suffered a mild TBI
- Appreciate the rehabilitation process and resources available to facilitate return to pre-morbid state
According to the CDC, an estimated 1.7 million people sustain a traumatic brain injury (TBI) each year. Of them:

- 52,000 die
- 275,000 are hospitalized
- 1.365 million, nearly 80%, are treated and released from an emergency department
- 80,000 of these survivors will live with significant disability

• About 75% of TBIs that occur each year are non-sports related concussions or other forms of mild TBI
TBI Introduction

- 5.3 million have TBI-related disabilities (2% of population)
- 90% are mild TBI’s (GCS>13)
- Economic Impact:
  - $9.2 Billion in lifetime medical costs ($4.2 million/survivor)
  - $51.2 Billion in productivity losses
TBI Introduction

• 50% of hospitalized TBI’s are from transportation accidents (most common cause <75yo)
• Incidence has decreased by 38% between 1980 and 1994
• 50% of people sustaining TBI are intoxicated at time of injury
TBI Introduction

• Most common cause >75yo: falls
• Fall prevention: address polypharmacy, balance impairments, orthostatic hypotension, minimize sedating medications
Introduction

• **1.6 to 3.8 MILLION** sports-related mild TBI’s/concussions occur annually
  – Most (90%) recover in 7-10 days
  – Prolonged recovery may be due to:
    • Non-physiologic Post-Concussive Syndrome (PCS)
    • Prolonged Concussion (Physiologic PCS)
  – Non-sports related concussions
    • Most recover in 3 months
    • 33% may have prolonged symptoms
  – Soldiers in Combat
    • 5-10% had LOC or altered consciousness
    • Caused by blasts, falls, MVA’s, wounds
Definition of a mild TBI/concussion:

• A traumatic injury to tissues of the brain as a result of a violent blow, shaking, spinning, or acceleration/deceleration forces that results in a complex pathophysiological process affecting the brain
• Glasgow Coma Scale (GCS) Score of 13-15
• May or may not have loss of consciousness (LOC)
Severity- Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Glasgow coma scale</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>to speech</td>
<td>3</td>
</tr>
<tr>
<td>to pain</td>
<td>2</td>
</tr>
<tr>
<td>none</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>orientated</td>
<td>5</td>
</tr>
<tr>
<td>confused</td>
<td>4</td>
</tr>
<tr>
<td>inappropriate</td>
<td>3</td>
</tr>
<tr>
<td>incomprehensible</td>
<td>2</td>
</tr>
<tr>
<td>none</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor response</strong></td>
<td></td>
</tr>
<tr>
<td>obeys commands</td>
<td>6</td>
</tr>
<tr>
<td>localises to pain</td>
<td>5</td>
</tr>
<tr>
<td>withdraws from pain</td>
<td>4</td>
</tr>
<tr>
<td>flexion to pain</td>
<td>3</td>
</tr>
<tr>
<td>extension to pain</td>
<td>2</td>
</tr>
<tr>
<td>none</td>
<td>1</td>
</tr>
<tr>
<td><strong>Maximum score</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>15</td>
</tr>
</tbody>
</table>
Risk Factors for TBI

• Males:Females = 2:1 (except in sports)
• 15-34 years old → less than 5 years old/
greater than 60 year old
• Low socioeconomic status
• Low cognitive function
• History of hospital admissions for intoxication
Sports with Highest Incidence of Concussions

• Football
• Wrestling
• Soccer - girls
• Soccer – boys
• Girls basketball – 3 times higher incidence than boys basketball
• Overall, females are 2-3 times more likely to get a concussion than males
• Children and teens are more likely to get a concussion and take longer to recover than adults.
Mild TBI/Concussions: How Do They Occur?

• Primary Injury
  – Shifting of the brain
  – Results in contusions or bruising of the brain
Mild TBI/Concussions: How Do They Occur?

• Secondary Injury “Neurometabolic Cascade”
  – Series of ionic, metabolic, and pathophysiological events accompanied by axonal injury
  – Increased energy required to recover + decreased cerebral blood flow/mitochondrial dysfunction = ENERGY MISMATCH and thus post-traumatic vulnerability
  – More pronounced in youth
Diffuse Axonal Injury

- Focal damage and diffuse damage to axons
- Effects tend to be broad (diffuse)

Figure 1-16. Axonal shearing may occur in acceleration as well as deceleration injuries. The nerve fiber may be stretched or completely severed, producing the manifestations of diffuse head injury.
Diffuse Axonal Injury

• Leading cause of morbidity (cognitive, behavioral, and arousal deficits) in TBI

• Occurs during acceleration/deceleration events (MVA’s) exposing the brain to stretch and torque forces → pathophysiologic process lasting days → axonal swelling and detachment

• CT/MRI often unremarkable except for some petechial hemorrhages
Diffuse Axonal Injury
Physical Symptoms

- Headache - most common complaint
- Nausea/vomiting
- Balance problems
- Dizziness
- Visual Problems
- Fatigue
- Sensitivity to light and/or noise
- Numbness/tingling
- Dazed/stunned
Emotional Symptoms

• Irritable
• Sadness
• Labile
• Nervousness
Sleep Symptoms

• Drowsiness
• Sleep more than usual
• Sleep less than usual
• Difficulty falling asleep
Cognitive Symptoms

• Difficulty concentrating
• Memory problems
• Forgetting information or conversations
• Confusion
• Repeats questions

• Feeling sluggish
• Feeling “foggy”
• Answers questions slowly
Mild TBI/Concussion Assessment

• Assess “A,B,C’s”
• Exclude cervical spine injury and more serious brain injury (send to ED)
• Mild TBI’s typically are NOT associated with obvious structural damage to the brain → CT or MRI scans usually are normal

• When to order imaging:
  – Deteriorating mental status
  – Focal neurological findings
  – Worsening symptoms
  – GCS< 15
  – Suspected Skull Fracture
  – >2 Vomiting episodes
  – > 30min Pre-traumatic amnesia
  – Bleeding risk
  – Intoxication
Concussion Sideline Assessment

The Standardized Assessment of Concussion
Obtain Pre-Season Baseline Score; Compare with Post-Concussion Score

**NAME OF ATHLETE:**
- Age: [ ]
- Sex: [ ]
- Examiner: [ ]
- Nature of Injury: [ ]
- Date of Exam: [ ]

**I. ORIENTATION**
- Month: [ ]
- Date: [ ]
- Day of Week: [ ]
- Year: [ ]
- Time: [ ]
- Orientation Total Score: [ ]

**II. IMMEDIATE MEMORY**
All 3 trials are completed regardless of score on trial 1 & 2; score equals sum across all 3 trials

<table>
<thead>
<tr>
<th>List</th>
<th>Trial 1</th>
<th>Trial 2</th>
<th>Trial 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
</tr>
<tr>
<td>Apple</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
</tr>
<tr>
<td>Saddle</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
</tr>
<tr>
<td>Bubble</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
</tr>
</tbody>
</table>
- Total Immediate Memory Recall: [ ]

Note: Do not inform the subject that delayed recall will be tested.

**III. NEUROLOGICAL SCREENING**
Recollection of injury (pre- or post-traumatic amnesia)
- Strength: [ ]
- Sensation: [ ]
- Coordination: [ ]

**IV. CONCENTRATION**
Digits Backwards: If correct, go to the next string length. If incorrect, read second trial. Stop after incorrect on both trials
- 1-9-3 | 2-7-9 | 0 1 |
- 3-1-5 | 2-7-9 | 0 1 |
- 6-2-9-7-1 | 5-2-8-6-1 | 0 1 |
- 7-1-8-4-6-2 | 5-2-1-3-8-1 | 0 1 |
- Months of the Year in Reverse Order: Athlete must recite calendar year from correct
- Total Concentration Score: [ ]

**V. EXERTIONAL MANEUVERS**
- Jumping Jacks: [ ]
- Sit-ups: [ ]
- Push-ups: [ ]
- Knee-bends: [ ]

**VI. DELAYED MEMORY RECALL**

<table>
<thead>
<tr>
<th>List</th>
<th>Trial 1</th>
<th>Trial 2</th>
<th>Trial 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow</td>
<td>0 1 0 1</td>
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</tr>
<tr>
<td>Bubble</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
<td>0 1 0 1</td>
</tr>
</tbody>
</table>
- Total Delayed Memory Recall: [ ]

**SUMMARY OF TOTAL SCORES**
- Orientation: [ ]
- Immediate Memory: [ ]
- Concentration: [ ]
- Delayed Memory Recall: [ ]
- Overall Total Score: [ ]

If score is below baseline, DO NOT return to play.

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Disadvantages to “Quick” Assessments

- Reduced sensitivity to complexity of mild concussions
- Overemphasis on loss of consciousness
- Lack of emphasis on neurocognitive dysfunction
- Not adequately sensitive to the individualized nature of a concussion
Comprehensive Neuropsychological Assessments

- Verbal and non-verbal intellectual functions
- Attention/concentration
- Processing speed
- Memory & new learning capacity
- Language processing
- Visual Spatial Processing
- Sensory & Motor Functions
- Executive Functions
  - Mental Flexibility
  - Fluency
  - Adaptive Problem Solving
- Emotional & Personality Functioning
Neurocognitive Assessments

- Comprehensive testing takes 6-8 hours
- Costly
- Relatively more vulnerable to practice effects with repeated use
- Turn around on results can be several days to a week
Importance of Neurocognitive Assessment

• Testing reveals the presence of cognitive deficits in patients who may otherwise be symptom-free
• Increased accuracy of diagnosis
• Increased likelihood of appropriate early intervention
• Increased quality of return to play decision
Computer Administered Screening Tools

• Takes approximately 30 minutes to complete
• Baseline tests are relatively inexpensive
• Reports generated immediately
• Tests designed to have multiple forms since each is comprised of randomly generated test (reduces the problem of practice effects)
• Validity checks are included
Computer Administered Screening Tools

• Pre-injury baseline data can be used for comparison purposes with post-injury testing
• Athlete can be compared to themselves
• Normative comparisons are available
• Data can be integrated with assessment of signs and symptoms
ImPACT Testing

- Useful screening tool
- Not a substitute for comprehensive neuropsychological testing
- Can be administered to athletes 11 to 59 years of age
- Has established validity
- Sensitive and specific for concussions
ImPACT Testing

- ImPACT is the most widely used neurocognitive screening tool for concussions
- Used by the NFL, NHL, MLB, NCAA, Professional Soccer
- Military is investigating possibility of implementation
- Available in 13 different languages
ImPACT Testing

• Verbal memory
• Visual memory
• Visual motor speed

• Reaction time
• Impulse control
• Symptom score
Management of Concussions

• Early diagnosis
• Rule out more serious brain injury
• Prevent long-term cumulative and chronic sequelae- PCS, 2\textsuperscript{nd} Impact Syndrome, CTE
• Rest the Brain!
  – Restriction of BOTH physical and cognitive activities such as exercise, rigorous classes, homework, videogames—and even driving!
Post-Concussion Syndrome Introduction

• 1.6 to 3.8 MILLION sports-related mild TBI’s/concussions occur annually
  – Most (90%) recover in 7-10 days
  – Prolonged recovery may be due to:
    • Non-physiologic Post-Concussive Syndrome (PCS)
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  – Non-sports related concussions
    • Most recover in 3 months
    • 33% may have prolonged symptoms
  – Soldiers in Combat
    • 5-10% had LOC or altered consciousness
    • Caused by blasts, falls, MVA’s, wounds
Post-Concussion Syndrome - Introduction

• After 3 weeks of symptoms, patients will worry about when/if they will recover.
• By 6 weeks, PCS can alter lives.
Post-Concussion Syndrome

• **WHO ICD-10 Criteria**: 3 or more of the following lasting > 2 weeks:
  – Headache
  – Dizziness
  – Fatigue
  – Irritability
  – Insomnia
  – Cognitive Deficits (Attention/Memory)
Post-Concussion Syndrome

• Other symptoms:
  – Photophobia
  – Hyperacusis
  – Feeling “foggy”
  – Blurry vision
  – Depressed/anxious mood
Risk Factors for PCS

• History of previous concussions
• Female sex
• History of psychiatric illness
• History of cognitive dysfunction
• Age – younger the athlete, longer the recovery
• History of migraine headaches
• ** Severity is not a risk factor!
Differential Diagnoses

• Depression
• Somatization
• Chronic Fatigue/Pain
• Cervical Injury/Myofascial Pain
• Vestibular Dysfunction
• Visual-spatial deficits
• Combination of above
Post-Concussion Syndrome

• Imaging is usually negative/equivocal
• Often a “disconnect” between observable signs & symptoms and neurocognitive deficits
• Can have measurable pathophysiology
  – fMRI- activation in the dorsolateral prefrontal cortex; de-activation of medial frontal cortex
  – SPECT scan- persistent abnormalities of brain blood flow
  – Neurochemical imbalances- Serum S100B
  – Electrophysiologic findings- QEEG
• Quantitative postural instability usually associated with physiologic PCS
Post-Concussion Syndrome- Theories

• Anatomic/Mechanical
  – Acceleration/deceleration → shearing → neuronal depolarization → nerve cell death
  – Diffusion tensor imaging (DTI), QEEG, SPECT demonstrate focal cortical dysfunction, blood-brain-barrier disruption, and reduced global/regional cerebral blood flow

• Physiologic
  – Decreased cerebral circulation
  – Higher resting and post-exertion HR
  – Increased sympathetic activity/ lower parasympathetic activity
  – Central autoregulation is disturbed → cardiorespiratory control of ventilation disrupted → symptoms exacerbated with exertion
Post-Concussion Syndrome - Theories

- Neurometabolic (Figure adapted from Hovda et al)
  - Metabolic hyperactivity followed by metabolic depression
  - Full metabolic recovery may be prolonged compared to actual symptoms
Diagnostic Testing

- **History**
  - Predisposing factors, other accompanying injuries

- **Physical Exam**
  - MMSE (assess concentration/memory), CN exam (EOM), gait/balance testing, assessment of cervical spine

- **Standardized treadmill test using the Balke protocol to differentiate physiologic PCS from non-physiologic PCS**

- **Neuropsychological testing**
  - Full Battery- may last 4-8 hours
  - IMPACT testing- used widely

- **Imaging- usually negative**
When to Seek Treatment

• Persistent mild TBI symptoms require individualized evaluation and treatment
• Treatment should be focused on symptomatic and functional improvement
Treatment

- All mild TBI’s are different
- Should not be treated the same
- Individualized treatment necessary
Treatment/ Rehabilitation

• Initial treatment- Relative physical and cognitive rest
• Education, education, education!!
• A comprehensive mild TBI/Post-Concussion Day Rehab Program is an ideal environment to treat those with PCS
  – Rehabilitation specialists equipped to handle all of the varying degrees of a mild TBI/concussion/PCS
  – Specialists with expertise to address all symptoms with an individualized approach
The Types of Treatment

The treatment team should include:

• Patient
• Family
• Clinical neuropsychologist
• Physician
• Nursing rehab specialist
• Physical therapist
• Occupational therapist
• Speech therapist
• Case manager/social worker
• Consultants: Neuro-optometry, Psychiatry, Neurology
Rehabilitation Specialists

• Medical management by a board-certified physiatrist specializing in concussions and brain injury
  – Coordinates treatment team
  – Can provide medical management
  – Can consult specialists as needed
  – Can oversee return-to-play; return-to-work; return-to-school with follow-up appointments as needed
Medical Management

- Insomnia
  - Educate regarding sleep hygiene
  - Minimize nighttime interruptions
  - Avoid naps if possible
- Medications
  - Trazodone, TCAs, mirtazapine
  - Gabapentin, TCAs for HA
  - Melatonin, valerian root: OTC
  - Avoid diphenhydramine, benzodiazepines, zolpidem if possible
Medical Management

- Headaches - usually multi-factorial
  - Migraines - TCA, fioricet, propranalol, NSAIDs, “triptans,” antiseizure medications, botulinum toxin using migraine protocol, acupuncture
- Greater occipital neuralgia - greater occipital nerve blocks with steroid
- Myofasical neck pain - myofascial release, massage, trigger point injections, acupuncture
- Comprehensive pain management program may be necessary in refractory cases
Medical Management

- Cognitive (attention and memory) deficits
  - “Start low and go slow”
  - Provide an adequate therapeutic trial
  - Perform continuous reassessments and define an objective behavioral target
  - Monitor for side effects
  - Consider drug augmentation
  - Change strategy if symptoms get worse
  - Consider half lives of meds
Medical Management

• Attention/Concentration deficits
  • Methylphenidate, Amantadine
  • (Ar)modafinil if arousal complicates symptoms

• Memory deficits
  • Donepezil, CDP-choline

• Emotional Lability/Depression
  • SSRI’s, TCA’s, Antiepileptic agents
Rehabilitation Specialists

• Comprehensive neuropsychological assessment provided by neuropsychologists specializing in brain injury

• Counseling, emotional support and education regarding recovery from brain injury for patients and families

• Cognitive behavioral therapy/neurofeedback
  – Identifies/changes patterns of maladaptive thinking and behavior to help with coping

• Availability to interface with employer/educational staff should work accommodations or an IEP be indicated
Goals of rehabilitation

• Return to play
• Graduated return to school
• Graduated return to work
• Prevention of long-term sequelae
  – Second Impact Syndrome (SIS)
  – Slower recovery from future neurologic insults
  – Permanent neurocognitive impairment
  – Neurodegenerative disease- Chronic Traumatic Encephalopathy (CTE)
  – Depression
Second Impact Syndrome

– A 2\textsuperscript{nd} TBI/concussion that occurs before metabolic homeostasis (recovery) is restored
– Rare and disputed diagnosis
Second Impact Syndrome

• Following a mild TBI, the risk of a 2\textsuperscript{nd} concussion is 2-3 times more likely
• Following a 2\textsuperscript{nd} mild TBI, risk is 9 times more likely a patient will experience a 3\textsuperscript{rd} concussion
• Controversy whether SIS is a complication of recurrent concussion, diffuse cerebral swelling (a recognized complication of TBI in children), or occult subdural injuries.
• Patients under the age of 21 are at increased risk, especially athletes (at increased risk for multiple traumatic impacts)
Second Impact Syndrome

• Behavior and personality changes
• Depression and suicide
• Parkinson’s Disease
• Speech issues
• Trouble walking
• Dementia
• Even death from brain swelling
Progressive degenerative neurological process found in some athletes who sustain multiple concussions and sub-concussive blows. This early degenerative process is characterized by cerebral atrophy and increased levels of tau protein, as well as cognitive impairment (dementia) and, in some cases, depression.

McKee /Cantu, 2009; Omalu,/DeKosky 2005.
CHRONIC TRAUMATIC ENCEPHALOPATHY
Tau Protein: Amygdala (McKee et al. 2009)
PCS- Returning to Play

• Graduated return to play protocol
• Progress from one stage to the next only if athlete is asymptomatic
Returning to Play - Stages

1) No activity: Physical and cognitive rest

2) Light aerobic exercise: Walking, swimming, stationary bike (no resistance training)

3) Sport-specific exercise: Running, skating – No head impact exercises
4) Non-contact training drills, like passing drills (football) – begin resistance training
5) Full contact practice after MD clearance
6) Return to play
Summary

1) Ongoing mild TBI symptoms are caused by prolonged concussion or another process (cervical injury, migraine HA, depression/anxiety, chronic pain, vestibular dysfunction, visual-spatial deficits, or combination of above)

2) Pathophysiology of PCS may reflect anatomic, neurometabolic, and physiologic causes

3) Individualized treatment with early education, a comprehensive mild TBI/Post-concussion Day Rehab Program, and aerobic exercise therapy can be effective
References

Questions

Thank you.

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