Concussion
Occupational Brain Injury & Assessment

NAOEM 2016

Jennifer M Zumsteg, MD

Assistant Professor
Physical Medicine and Rehabilitation (PM&R)
Board Certified, Brain Injury Medicine (BIM)
Fellowship Director, Brain Injury Medicine
Harborview – c1931
Financial Disclosures

• No conflicts for this lecture

• Research funding
  – UW TBI Model System: National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR)
Learning Objectives

• Overview of challenges in TBI
• Framework for TBI diagnosis
• Clinical assessment tools
• Strategies for return to activity
• Tips for managing common symptoms
Suggested Broad Resources


Case

- Mr. TBI is a 25yo office manager who fell and struck his head 2 weeks ago...
  - Scalp hematoma
  - Confused / repeats same questions
  - Initial head CT normal
  - Reports dizziness, headaches, irritability, sleep changes, and cognitive impairment
Case

• Mr. TBI is a 25yo office manager who fell and struck his head 2 weeks ago...
  – Did he even have a TBI?
  – Can he work now? In what capacity?
  – Assessment and treatment tools for dizziness, headaches, irritability, sleep changes, and cognitive impairment?
TBI Diagnosis
Define TBI

Injury to the Head
*Direct or Indirect Force*
- Reasonable mechanism
- Subjective/objective report
- Imaging findings

Decreased LOC / Amnesia
- Subjective/objective report

Objective Neuro/psych Findings
- Data collection...

http://www.cdc.gov/ncipc/pub-res/tbi_congress/05_references_appendix.htm
Define TBI

Injury to the Head

• Reasonable mechanism
• Subjective/objective report
• Imaging findings

Decreased LOC / Amnesia

• Subjective/objective report

Objective Neuro/psych Findings

• Data collection

Observed?

Intoxicated?

Hypotensive?

Baseline symptoms?

http://www.cdc.gov/ncipc/pub-res/tbi_congress/05_references_appendix.htm
Neurological Abnormalities

• Your excellent exam
• History / observation

• Examples:
  – motor function
  – sensory function
  – reflexes
  – abnormalities of speech
  – seizures

...acutely following head trauma.
Psych/Behavioral Abnormalities

• Mental status *exam*
  • Neuropsychological *exam*

• Examples:
  – Disorders of mental status
    • Disorientation, agitation, confusion
  – Other changes in cognition, behavior, or personality
Post-traumatic Symptoms

• The majority of patients with mild TBI will have no symptoms within several weeks

• Early reassurance and education in mTBI helps with recovery
Key Principles of Treatment

• Validate the experience of the person (impairment and subjective complaints)
• Do not assume psychological factors as the primary problem and source of disability
• Incorporate successful functional tasks into therapy program to allow patient to re-build sense of control
Key Principles of Treatment

- Treat physical, psychological, and cognitive problems
- Address the complaints with concrete action
- Begin the process of sorting out primary problems (physical or cognitive) from secondary (psychological) problems
Diagnose TBI

• **Clinical** diagnosis
  1. Reasonable mechanism of injury
  2. LOC / dazed / amnesia at the time of injury
  3. Objective neuro/psychological abnormalities

• Differentiate from
  *Whiplash and Headache*
Diagnose TBI

• **Reasonable** mechanism of injury
  
  A. Blunt trauma
  
  B. Penetrating trauma
  
  C. Acceleration / deceleration
    - MVC
    - Collision (sports, transportation injuries)
  
  D. Blast
Reasonable Mechanism

• Symptoms alone do NOT make the diagnosis
TBI Severity
Epidemiology: Severity

• Mild: 80 – 90 %

• Moderate: 5 - 10%

• Severe: 5 - 10%
Mild TBI

• Lack agreement on definition
  – WHO, CDC, ACRM, other workgroups

• Severity is based on initial injury, not outcome
  – Glasgow Coma Scale (GCS)
  – Duration of post-traumatic amnesia
Mild TBI - Definition

- Acute brain injury from external forces, with
  - Confusion OR disorientation OR LOC <30min OR PTA < 24 hours
  - AND/OR transient neurological abnormalities
  - AND GCS 13-15, 30 minute post-injury

(excludes more severe injuries, effects from other injuries, intoxication)

CDC, 2003
WHO, 2004
## Glasgow Coma Scale (GCS)

<table>
<thead>
<tr>
<th></th>
<th>Score</th>
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</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>Response to verbal command</td>
<td>3</td>
</tr>
<tr>
<td>Response to pain</td>
<td>2</td>
</tr>
<tr>
<td>No eye opening</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>No verbal response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td></td>
</tr>
<tr>
<td>Obeys commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizing response to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdrawal response to 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>No motor response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
</tr>
</tbody>
</table>

The GCS is scored between 3 and 15, 3 being the worst and 15 the best. It is composed of three parameters: best eye response (E), best verbal response (V), and best motor response (M). The components of the GCS should be recorded individually; for example, E2V3M4 results in a GCS score of 9. A score of 13 or higher correlates with mild brain injury; a score of 9 to 12 correlates with moderate injury; and a score of 8 or less represents severe brain injury.
TBI Severity Using GCS

• **Mild**: 13-15
  – Mild-complicated: 13-15 with CT or MRI findings

• **Moderate**: 9-12

• **Severe**: 3-8 = Coma
  • Intubate...
It’s worse than mild TBI if...

• LOC greater than 30 minutes
• Amnesia (PTA) greater than 24 hours
• Initial GCS ≤ 12
• (Cranial nerve injuries)
  – CN I commonly injured after mild TBI
• Any imaging findings on head CT or MRI

• Concussion sub-scale severity rating - limited use clinically
Post-Concussion Syndrome
Clinical practice guidelines for mild traumatic brain injury and persistent symptoms

Shawn Marshall MD MSc FRCP  Mark Bayley MD FRCP  Scott McCullagh MD FRCP  Diana Velikonja PhD CPsych  Lindsay Berrigan PhD

Abstract

Objective To outline new guidelines for the management of mild traumatic brain injury (MTBI) and persistent postconcussive symptoms (PPCS) in order to provide information and direction to physicians managing patients' recovery from MTBI.

Quality of evidence A search for existing clinical practice guidelines addressing MTBI and a systematic review of the literature evaluating treatment of PPCS were conducted. Because little guidance on the management of PPCS was found within the traumatic brain injury field, a second search was completed for clinical practice guidelines and systematic reviews that addressed management of these common symptoms in the general population. Health care professionals representing a range of disciplines from across Canada and abroad were brought together at an expert consensus conference to review the existing guidelines and evidence and to attempt to develop a comprehensive guideline for the management of MTBI and PPCS.

Main message A modified Delphi process was used to create 71 recommendations that address the diagnosis and management of MTBI and PPCS. In addition, numerous resources and tools were included in the guideline to aid in the implementation of the recommendations.

Conclusion A clinical practice guideline was developed to aid health care professionals in implementing evidence-based, best-practice care for the challenging population of individuals who experience PPCS following MTBI.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3303645/
Post-Concussive Syndrome (ICD10)

• A. History of head trauma with loss of consciousness preceding symptom onset by a maximum of 4 wk.

• B. Symptoms in 3 or more of the following symptom categories:
  • headache, dizziness, malaise, fatigue, noise intolerance;
  • irritability, depression, anxiety, emotional lability;
  • subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment;
  • insomnia;
  • reduced alcohol tolerance; and
  • preoccupation with above symptoms and fear of brain damage with hypochondriacal concern and adoption of sick role.
Post-Concussive Syndrome (DSM)

- (A) history of TBI causing "significant cerebral concussion"
- (B) objective cognitive deficit in attention and/or memory
- (C) presence of at least 3 of 8 symptoms (fatigue, sleep disturbance, headache, dizziness, irritability, affective disturbance, personality change, apathy) that appear after injury and persist for 3 months
- (D) symptoms that begin or worsen after injury
- (E) interference with social role functioning
- (F) exclusion of dementia due to head trauma and other disorders that better account for the symptoms.
MRI?

- Concerning mechanism of injury

- Unexplained neuro, MSK and/or psych findings on exam

- Symptoms not improving
American Society of Neuroradiology

- Advanced imaging still just for research
- Very difficult to apply clinically
- Stay tuned...
  DTI and other modalities quite promising...
Pathophysiology
Definition

- **Cause of injury**: a description followed by the CDC that would allow for public health tracking and interventions

- **Mechanism of injury**: description used medically to help us describe to each other the type(s) of external forces that were exerted on the brain

- **Pathophysiology of injury**: description of the injury that is seen in the brain from the sub-cellular to gross anatomy range
Leading Causes of TBI

- Falls, 40.5%
- Struck by/against, 15.5%
- Unknown/Other, 19.0%
- Motor vehicle traffic, 14.3%
- Assaults, 10.7%
Work Related TBI

• Washington
Cause of Injury 1988-1990 (n=301)

- Fall from elevation
- Struck by
- Highway MVC
- Fall same level
- Struck against
- Caught in/under
- Highway, non-MVC
- Explosion

Cause of Injury 16-24 years  (n=273, 1998-2008)

- Falls
- MVC
- Struck by/against
- Other
- Machinery

J Safety Res. 2013 Jun; 45: 133–139.
Pathophysiology

Primary Injury

• Displacement and direct injury from physical forces
  – Injury → hours

Secondary Injury

• Due to cellular and inflammatory response
  – Hours → days / weeks
## Pathological Injury Classification

<table>
<thead>
<tr>
<th></th>
<th>Diffuse Brain Injury</th>
<th>Focal Brain Injury</th>
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<tbody>
<tr>
<td>Primary Brain Injury</td>
<td>- Diffuse axonal injury</td>
<td>- Focal cortical contusion</td>
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<tr>
<td></td>
<td>- Petechial white matter hemorrhage with diffuse vascular injury</td>
<td>- Intracerebral hemorrhage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Extracerebral hemorrhage</td>
</tr>
<tr>
<td>Secondary Brain Injury</td>
<td>- Delayed neuronal injury</td>
<td>- Delayed neuronal injury</td>
</tr>
<tr>
<td></td>
<td>- <strong>Diffuse brain swelling</strong></td>
<td>- Focal brain swelling</td>
</tr>
<tr>
<td></td>
<td>- Diffuse ischemic injury</td>
<td>- Focal ischemic injury</td>
</tr>
<tr>
<td></td>
<td>- Diffuse hypoxic injury</td>
<td>- Focal hypoxic injury</td>
</tr>
<tr>
<td></td>
<td>- <strong>Diffuse metabolic dysfunction</strong></td>
<td>- Regional metabolic dysfunction</td>
</tr>
</tbody>
</table>

Symptoms and
Anatomic Correlates
Vulnerable Areas

• Direct pressure / Contusion
  – Inferior frontal lobe (orbitofrontal)
  – Anterior temporal lobe (temp poles)
• Diffuse axonal injury
  – Corpus collosum
  – Central white matter
  – Brainstem
Inferior frontal lobe (orbitofrontal)

• Olfactory recognition

• Reward pathways
  • Behavioral choice
  • Learning
  • Memory
  • Control of emotion

• Emotional inhibition
  • Decision making
  • Executive function
  • Many projections
Anterior Temporal Lobes

• Semantic memory
  – Knowledge of...
    • Objects
    • People
    • Words
    • Facts

• Contribute to semantic information
  – Visual, auditory, emotional
Diffuse Axonal Injury

• Connections across the brain
  – Corpus collosum
  – Central white matter Brainstem

• Arousal, attention ➔ functional memory
Event – Helpful Info

• Incident history details
  – forces to the head and/or body
• LOC? Amnesia? Confusion?
  – duration
• Glasgow Coma Scale on presentation
• Other complicating factors
  – Intoxication, hypotension
• Symptoms at / near time of injury
  • Within 4 weeks
Assessment & Treatment

- Cognitive Changes
- Depression and Anxiety
- Insomnia
- Balance/Dizziness
- Headaches

Generally, no FDA-approved treatments specific to TBI
Key Scales Available...

http://www.tbims.org/combi/list.html
Treatment

• Cognitive Changes
  – Speech Therapy (SLP) – cognitive therapy
  – Medications
  – Neuropsychological testing

• Depression and Anxiety
  – Medications; psychotherapy; exercise

• Insomnia
  – Sleep hygiene; medication; exercise
Treatment

• Balance/Dizziness
  – Medical assessment
  – Physical therapy; ENT assessment

• Headaches
  – Consider whiplash; Physical therapy, chiropractic
  – Migraine vs. tension vs. other
  – Medications, exercise, avoid triggers
    • Accommodate sensory overstimulation
TBI Principles of Treatment

- With meds...

  - Start LOW...go SLOW...but GO!

  - Think about multiple effects of medications
  - Use side effects to your advantage
Cognitive Changes

• Screening tools ➔ Many limitations
  – Montreal Cognitive Assessment (MoCA)
    • 3 versions, many languages
    • [http://www.mocatest.org/](http://www.mocatest.org/)

  – ** The Executive Interview (EXIT-25) **

  – Moss Attention Rating Scale
    • [http://tbims.org/combi/mars/](http://tbims.org/combi/mars/)
Classic Profile of MTBI:

- Slowed mental processing time
- Impaired complex memory
- Impaired complex attention
Neuropsychological Assessment
Testing Options

** Board Certified Neuropsychologist **

→ Computerized: volume, cost, alternate tests
  • no info on auditory processing or verbal memory, no recall memory, no observation
Neuropsychological Assessment

• Most convincing if baseline data exists as comparison points
  – Athletics may have baseline data
    – ImPACT – Immediate Post Concussion Assessment and Cognitive Testing...
      adult norms not clearly available
    • Baseline school data may be helpful
    • Structured interview helpful
When to Refer for NPE

• Individualized, goal directed
• Education level and Language
  – Same primary language, 6th grade education
• Reasonable functional plateau
• Tolerate testing
• Minimize interference factors
  – Mood, pain, sleep, motor impairments, meds
Cognitive Changes - Meds

• Need to fit overall impairments / goals
  – Methylphenidate 1\textsuperscript{st} line for attention
  – Amantadine used for many applications
  – Side effects of medications
  – Treat other factors known to interfere with thinking
    • Pain, sleep, mood...
Emotional Changes

• Agitation / Restlessness / Labile
  – Agitated Behavior Scale: [http://www.tbims.org/combi/abs/](http://www.tbims.org/combi/abs/)
  – Overt Behavior Scale: [http://www.tbims.org/combi/obs/index.html](http://www.tbims.org/combi/obs/index.html)
Depression and Anxiety

• Depression

• Anxiety

• PTSD
  – Civilian Checklist: [http://www.mirecc.va.gov/docs/visn6/3_PTSD_CheckList_and_Scoring.pdf](http://www.mirecc.va.gov/docs/visn6/3_PTSD_CheckList_and_Scoring.pdf)
Depression Treatment

JOURNAL OF NEUROTRAUMA 26:2383–2402 (December 2009)
© Mary Ann Liebert, Inc.
DOI: 10.1089/neu.2009.1091

Treatment for Depression after Traumatic Brain Injury:
A Systematic Review

Jesse R. Fann,¹ Tessa Hart,² and Katherine G. Schomer³

Abstract
The aim of this systematic review was to critically evaluate the evidence on interventions for depression following traumatic brain injury (TBI) and provide recommendations for clinical practice and future research. We reviewed pharmacological, other biological, psychotherapeutic, and rehabilitation interventions for depression following TBI from the following data sources: PubMed, CINAHL, PsycINFO, ProQuest, Web of Science, and Google Scholar. We included studies written in English published since 1980 investigating depression and depressive symptomatology in adults with TBI; 658 articles were identified. After reviewing the abstracts, 57
Depression Treatment

• **Psychotherapy** (cognitive behavioral therapy)

• SSRIs
  – Better response
  – Fewer side effects

• Citalopram and sertraline considered 1st line

• Others:
  – Seizure threshold: avoid bupropion
  – More side effects: TCAs challenging
Apathy

• Apathy Evaluation Scale
  – http://www.tbims.org/combi/aes/index.html
Insomnia – To Do

** Sleep Hygiene **

• Exercise

• Melatonin
  – Sleep cycle: 0.5 mg po 5 hours before desired sleep
  – Sleep onset: 1-10mg po qhs

• Trazodone as needed: 25-125mg po qhs

• Consider further sleep evaluation – fatigue?
  – Other organic causes of insomnia / fatigue...
Insomnia / Fatigue

• Insomnia Severity Index:

• Epworth Sleepiness Scale:
  – http://epworthsleepinessscale.com/about-epworth-sleepiness/
Insomnia - Avoid

• Anticholinergic meds (increase confusion)
  – antihistamines
• Benzodiazepines (amnesia, rebound)
• Caffeine
• Alcohol
Balance & Dizziness

• Peripheral vs. Central

• Oculomotor and CNS issues — often complex

• Consider referral to ENT / Neuro-otology
UW Balance & Dizziness Center

• Neuro-otology consultation

• Central and peripheral dizziness and balance testing:
  – http://depts.washington.edu/coursejo/
Headaches
Post-traumatic Headaches

By definition ➔ start within 7 days

http://ihs-classification.org/en/
Headache after TBI

- Post-traumatic headache (PTH) is one of the most common persisting symptoms after injury, with prevalence ranging from 30-90% of patients in studies to date. PTH occurs more commonly in those with lesser severity brain injury.
  - Lahz & Bryant, 1996

- PTH is the salient physical symptom after blast exposure in military populations.
  - Hoge et al., 2008, Theeler & Erickson, 2009

- PTH can be chronic, with 18-22% persisting past one year.
  - Lew et al., 2006
• Patients with PTH reported greater functional impairment than those with non-traumatic headache.
  — Marcus, 2003

• Athletes with headaches following concussion performed worse on cognitive testing than those without.
  — Mihalik et al., 2005

• ICH criteria do not contribute to treatment planning and do not account for latency of PTH following trauma.
  — Theeler & Erickson, 2009

• Primary headache =
  Idiopathic, classic features

• Secondary headache =
  Defined by underlying cause
General Treatment Approach

• Headache episodes = episodic treatment
  – Abortive therapy (short acting meds)

• 2+ headache days per week
  – At risk for medication rebound or overuse headache
  – Consider prophylactic therapy (daily meds)
  – Goal is decrease frequency and severity of HA

• Treat associated symptoms (nausea)
Treatment

• Treat subtype of headache AND potential triggers
  – Migraine (abortive and prophylactic therapy)/stress, food, neck dysfunction, sleep disorder, medication overusage)
  – Tension type (physical therapy, analgesics, behavioral)
  – Cervicogenic (diagnostic and therapeutic blocks, therapy, NSAIDs)

• Always evaluate for potential pain mediators and treat (depression, insomnia)

Migraine Meds

• Abortive / Acute
  – Acetaminophen, NSAIDs, Exedrin
  – Triptans (try different formulations)

• Prophylactic
  – AEDs:
    • Valproic acid
    • Topiramate
  – B-blockers:
    • Metoprolol, propranolol, timolol


Tension Meds

• Abortive / Acute
  – 1\textsuperscript{st} NSAIDs, ASA, Acetaminophen
  – 2\textsuperscript{nd} Excedrin (especially if severe)

• Prophylactic
  – 1\textsuperscript{st} Amitriptyline
  – 2\textsuperscript{nd} Mirtazapine or Venlafaxine
  – Other TCAs

RTW & Brain Injury

“Because of this complexity and the unique nature of each patient with TBI, prediction of RTW for individual patient is not feasible.”

Zassler, Brain Injury Medicine p1349.
Work Considerations

• Avoid return to work same day of injury
• Consider no driving, no alcohol
• Directed by symptoms, job, capacity...
Risk Factors
Frequently found to contribute to poor work outcomes:

- More severe injury
- Higher age at injury
- Male
- Lower level of pre-injury education or occupational status
- Less social support
- Significant functional/cognitive impairments
- Minority group membership
- History of substance abuse

Zassler, Brain Injury Medicine p1349
Approach to Progression of Activity

• Sample model from sports concussion and return to play
Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012

Paul McCrory, Willem H Meeuwisse, Mark Aubry, Bob Cantu, Jiří Dvorák, Ruben J Echemendia, Lars Engebretsen, Karen Johnston, Jeffrey S Kutcher, Martin Raftery, Allen Sills, Brian W Benson, Gavin A Davis, Richard G Ellenbogen, Kevin Guskiewicz, Stanley A Herring, Grant L Iverson, Barry D Jordan, James Kissick, Michael McCrea, Andrew S McIntosh, David Maddocks, Michael Makdissi, Laura Purcell, Margot Putukian, Kathryn Schneider, Charles H Tator, Michael Turner

PREAMBLE
This paper is a revision and update of the recommendations developed following the 1st (Vienna 2001), 2nd (Prague 2004) and 3rd (Zurich 2008) International Consensus Conferences on Concussion in Sport and is based on the deliberations at the 4th International Consensus Conference on Concussion in Sport, Zurich, Switzerland, November 2012.

SECTION 1: SPORT CONCUSSION AND ITS MANAGEMENT
The Zurich 2012 document addresses the sport concussion and its management issues raised in the previous Vienna 2001, Prague 2004 and Zurich 2008 documents and applies the consensus questions and clinical guidance approach to current knowledge and practice. Further, it provides an update on research, guidelines and awareness of brain injury following concussion.
# Return to Play — *minimum* 5-7 days

<table>
<thead>
<tr>
<th>No activity</th>
<th>Physical/cognitive rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light aerobic exercise</td>
<td>Walking/swimming/stationary bicycle</td>
</tr>
<tr>
<td></td>
<td>&lt;70% max HR</td>
</tr>
<tr>
<td></td>
<td>No resistance training.</td>
</tr>
<tr>
<td>Sport specific exercise</td>
<td>No head impact activities</td>
</tr>
<tr>
<td>Non contact training drills</td>
<td>Increase complexity and cognitive load. Resistance training</td>
</tr>
<tr>
<td>Full contact practice</td>
<td></td>
</tr>
<tr>
<td>Return to play</td>
<td>Normal game activities</td>
</tr>
</tbody>
</table>

Advance next stage when asymptotic at least 24 hours
Injured Worker

• Consider progressive structured program such as work hardening

• During functional capacity evaluation, request information about ability to follow directions, distractibility, emotional status, and other cognitive skills

• Comprehensive outpatient day programs
  – Treatment environment that also understands cognitive changes
Summary
Case

- Mr. TBI is a 25yo office manager who fell and struck his head 2 weeks ago...
  - Scalp hematoma
  - Confused / repeats same questions
    - GCS 14
  - Initial head CT normal
  - Reports dizziness, headaches, irritability, sleep changes, and cognitive impairment
Case

• Mr. TBI is a 25yo office manager who fell and struck his head 2 weeks ago...
  – Did he have a TBI?
  – Can he still work? In what capacity?
  – Assessment and treatment tools for dizziness, headaches, irritability, sleep changes, and cognitive impairment?
Case - Diagnosis

• Did Mr. TBI have a TBI?
  
✓ Mechanism of injury
✓ Change in consciousness
✓ Neuro/psych/behavioral symptoms and signs
Take Home Points

• Most mild TBI gets better within weeks
• Persistent TBI symptoms can benefit from treatment
• Resources available for those who don’t get better quickly
• Cognitive, mood and other changes after TBI are functionally important for work
More on PM&R

Physical Medicine and Rehabilitation

- UW Medicine Rehabilitation – Clinical Care
  - http://www.uwmedicine.org/services/rehabilitation
- UW Department Rehabilitation Medicine
  - http://rehab.washington.edu/
- Academy - AAPM&R
  - https://www.aapmr.org/practice/marketing/Pages/playback-pmr-sandel-video.aspx
The University of Washington’s Department of Rehabilitation Medicine strives to improve health, functional performance, and participation in life’s activities for people who have disabling conditions and diseases.
Supplemental Info
Primary Injury

• Diffuse Axonal Injury
  – Most commonly affect **white matter** in areas including **brainstem, corpus callosum, basal ganglia, thalamus** and the **cerebral hemispheres**

Diffuse axonal injury

CT scan of the brain showing diffuse axonal injury (DAI). Note the deep shearing-type injury in or near the white matter of the left internal capsule (arrow).

Reproduced with permission from: J Claude Hemphill II, MD and Nicholas Phan, MD, FRCSC.

Case courtesy of Dr Alexandra Stanislavsky, Radiopaedia.org
Primary Injury: DAI

- Affects the brain on macroscopic and microscopic level
  - Macro: Hemorrhage from tearing of blood vessels
  - Micro: Increases cell membrane permeability
Primary Injury: DAI

• Disruption of axonal transport impairment and axonal swelling, followed by detachment over a period of time

Primary Injury

• Impact depolarization
  – Massive surge of extracellular potassium and glutamate release (excitatory) and leads to excitotoxicity (secondary injury)
Secondary Injury

Secondary Injury

• Cascade of biochemical, cellular, and molecular events that occurs **hours to days** after the initial impact

• Mechanisms include:
  – Ischemia
  – Secondary cerebral swelling
  – Axonal injury
  – Inflammation
Neurotransmitter Dysfunction
Neurotransmitter Dysfunction

• Acute alterations of cerebral neurotransmitter levels seen after the injury possibly from stretching and straining forces to the brain

• Excess neurotransmitter ("neurotransmitter storm") is thought to contribute to the early neuropathophysiology of TBI, including mild TBI
Why care about neurotransmitters?

- **Glutamate**: mediator of **excitatory** signals for normal brain function
- **Dopamine**: **motor** movement, **arousal**, **mood**
- **Norepinephrine**: attention
- **Serotonin**: **cognitive** function & stabilizing and modulating brain function
- **Acetylcholine**: **memory** & **motor** function
Ddx “Dizziness”

• Vertigo
• Balance impairment
• Vision changes
• Pre-syncope/syncope
• Associated with psychiatric diagnoses
• Other associations
  – Hyperventilation
  – Headache
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<tbody>
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<td>Primary care</td>
<td>Dizziness clinic</td>
<td>Dizziness clinic</td>
<td>Emergency room</td>
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<tr>
<td>Patients</td>
<td>100</td>
<td>102</td>
<td>2222</td>
<td>125</td>
</tr>
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<td>Average age</td>
<td>46</td>
<td>-</td>
<td>48</td>
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<td>Cause, percent</td>
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<td>Vertigo</td>
<td>54</td>
<td>46</td>
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<td>50</td>
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<tr>
<td>Benign positional vertigo</td>
<td>16</td>
<td>12</td>
<td>17</td>
<td>-</td>
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<tr>
<td>Vestibular neuronitis</td>
<td>4</td>
<td>4</td>
<td>10</td>
<td>1</td>
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<tr>
<td>Other vestibular</td>
<td>10</td>
<td>9</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>Central</td>
<td>10</td>
<td>7</td>
<td>4</td>
<td>7</td>
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<tr>
<td>Migraine</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nonspecific</td>
<td>10</td>
<td>10</td>
<td>-</td>
<td>-</td>
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<td>Psychiatric disorder</td>
<td>16</td>
<td>9</td>
<td>21</td>
<td>11</td>
</tr>
<tr>
<td>Presyncope</td>
<td>6</td>
<td>4</td>
<td>-</td>
<td>14</td>
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<tr>
<td>Disequilibrium</td>
<td>2</td>
<td>16</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>1</td>
<td>23</td>
<td>-</td>
<td>5</td>
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<td>Multicausal</td>
<td>13</td>
<td>12</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Unknown</td>
<td>8</td>
<td>9</td>
<td>19</td>
<td>10</td>
</tr>
</tbody>
</table>

Vertigo

Origin of VERTIGO

Middle English, from Latin vertigin-, vertigo, from vertere to turn

First Known Use: 15th century
Dix Hallpike maneuver

With the patient sitting, the neck is extended and turned to one side. The patient is then placed supine rapidly, so that the head hangs over the edge of the bed. The patient is kept in this position and observed for nystagmus for 30 seconds. In patients with benign paroxysmal positional vertigo, nystagmus usually appears with a latency of a few seconds and lasts less than 30 seconds. It has a typical trajectory, beating upward and torsionally, with the upper poles of the eyes beating toward the ground. After it stops and the patient sits up, the nystagmus will recur but in the opposite direction. Therefore, the patient is returned to upright and again observed for nystagmus for 30 seconds. If nystagmus is not provoked, the maneuver is repeated with the head turned to the other side. If nystagmus is provoked, the patient should have the maneuver repeated to the same (provoked) side; with each repetition, the intensity and duration of nystagmus will diminish.
In patients with benign paroxysmal positional vertigo due to canalithiasis, the particle repositioning maneuver encourages the calcium carbonate debris to migrate toward the common crus of the anterior and posterior canals and exit into the utricular cavity. Step 1 is the standard Dix-Hallpike positioning test.

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nystagmus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direction</td>
<td>Unidirectional, fast component toward the normal ear; never reverses direction</td>
<td>Sometimes reverses direction when patient looks in the direction of slow component</td>
</tr>
<tr>
<td>Type</td>
<td>Horizontal with a torsional component, never purely torsional or vertical</td>
<td>Can be any direction</td>
</tr>
<tr>
<td>Effect of visual fixation</td>
<td>Suppressed</td>
<td>Not suppressed</td>
</tr>
<tr>
<td><strong>Other neurologic signs</strong></td>
<td>Absent</td>
<td>Often present</td>
</tr>
<tr>
<td><strong>Postural instability</strong></td>
<td>Unidirectional instability, walking preserved</td>
<td>Severe instability, patient often falls when walking</td>
</tr>
<tr>
<td><strong>Deafness or tinnitus</strong></td>
<td>May be present</td>
<td>Absent</td>
</tr>
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### Dix-Hallpike maneuver for positional nystagmus: Findings in central versus peripheral vertigo

<table>
<thead>
<tr>
<th>Latent period before onset of positional nystagmus</th>
<th>Peripheral disorder</th>
<th>Central disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 to 20 seconds</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Duration of nystagmus</td>
<td>Less than 1 minute</td>
<td>Greater than 1 minute</td>
</tr>
<tr>
<td>Fatiguability</td>
<td>Fatiguing with repetition</td>
<td>Nonfatiguing</td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>Only one type, may change direction with gaze</td>
<td>May change direction with a given head position</td>
</tr>
<tr>
<td>Intensity of vertigo</td>
<td>Severe</td>
<td>Less severe, sometimes none</td>
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</table>
Other Peripheral Dizziness

• Labyrinthine concussion
  – Hearing loss and continuous vertigo → general imbalance
  – Generally improves quickly
  – Rx: Time; Vestibular therapy

• Perilymphatic fistula
  – Disruption of middle and inner ear boundaries
  – Controversial
  – Hearing loss, vertigo and tinnitus
  – Symptoms may vary with pressure/valsalva
Clinical features of central versus peripheral vertigo

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Dizziness

Treatment:

• Treat other issues: Visual correction, motor control, medication side effects
  – Vestibular therapy
  – Compensatory strategies
  – Otologist evaluation
  – Medication treatment
    • Antihistamines, phenothiazines, benzodiazepines
      – Concern for adverse effects on cognition in TBI
References

• Centers for Disease Control [http://www.cdc.gov/traumaticbraininjury/data/](http://www.cdc.gov/traumaticbraininjury/data/)
• Up to Date, headings listed each slide
  – Chapter 30: Conceptualizing Outcome from Mild TBI and Chapter 81: Return to Work after TBI
Other References and Readings


