Traumatic Brain Injury
Basics and Beyond

Thursday June 19, 2014

Tad Gorske, Ph.D.
Assistant Professor
Division of Neuropsychology and
Rehabilitation Psychology
Department of Physical Medicine and Rehabilitation
University of Pittsburgh School of Medicine
Acknowledgements

• I want to thank Dr. Camiolo-Reddy from the UPMC Department of Physical Medicine and Rehabilitation and Dr. Michael Collins from the UPMC Sports Medicine Concussion Program for giving me permission to use information from their slide presentations.
Traumatic Brain Injury
Definition of Traumatic Brain Injury

- Closed head injury (CHI) – Skull intact, brain not exposed.
- Penetrating head injury (PHI) – Open head injury where skull and dura are penetrated by an object.
- Vascular insults (due to stroke, anoxia, etc. will also be included for today’s purposes.)
Head Injury Morphology: Blunt versus Penetrating

Blunt Injury
• Coup: adjacent to the area struck by an object
• Countercoup injuries: usually due to acceleration of the brain, striking the calvarium or skull base of the opposite side

Penetrating
• Bladed weapons, low and high velocity projectiles
• Injury from GSW results from energy transfer from the projectile to impacted tissue
Coup and Contrecoup Injury
• Centers for Disease Control TBI Definition
  – Craniocerebral trauma, specifically, an occurrence of injury to the head (arising from blunt or penetrating trauma or from acceleration/deceleration forces) that is associated with any of these symptoms attributable to injury: decreased level of consciousness, amnesia, other neurologic or neuropsychological abnormalities, skull fracture, diagnosed intracranial lesions, or death.

Manly and Maas (2013) JAMA.

• Traumatic brain injury (TBI) is a multifaceted condition, not an event.
• Broadly defined as an alteration in brain function or other evidence of brain pathology caused by an external force that can occur in traffic, at home, at work, during sports activities, and on the battlefield (pg. 473).
Prevalence of TBI (Center for Disease Control)

- Associated with:
  - 52,000 deaths annually;
  - 275,000 hospitalizations – nonfatal TBI
  - 1,365,000 ER visits
  - Estimated 1.7 million TBI’s occurring in the US annually.
In 2010, about 2.5 million emergency department (ED) visits, hospitalizations, or deaths were associated with TBI—either alone or in combination with other injuries—in the United States.

- TBI contributed to the deaths of more than 50,000 people.
- TBI was a diagnosis in more than 280,000 hospitalizations and 2.2 million ED visits. These consisted of TBI alone or TBI in combination with other injuries. (Source CDC).
Rates of TBI hospitalization and death by age group

Rates per 100,000

Age Group

0-4 5-14 15-24 25-34 35-44 45-54 55-64 65-74 75+

Rates per 100,000
• Estimated cost of TBI was $260 billion spent in the United States
Proportion of TBI related hospitalizations and deaths (Source: CDC, National Center for Injury Prevention and Control)

- Falls: 40.5%
- Assaults: 10.7%
- MVA/Traffic: 14.3%
- Struck by/Against: 15.5%
- Other/Unknown: 19.0%
Mechanism of Brain Injury

• **Primary Injury:** Events that occur at the moment of injury, although possibly preventable are not reversible
  – Damage that results from shear forces; seen in the initial minutes/hours after the insult
  – Cortical disruption
  – Axonal Injury
  – Vascular Injury
  – Hemorrhage

• **Secondary Injury:** Damage sustained minutes, hours, or days after primary injury
  – Evolution of brain damage
  – Post traumatic ischemia
  – Excitotoxicity
  – Cell Death
  – Axonal Injury
  – Cerebral Swelling
  – Inflammation/regeneration
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.
Closed Head Injury

- Resulting from falls, motor vehicle crashes, etc.
- Focal damage and diffuse damage to axons
- Effects tend to be broad (diffuse)
- No penetration to the skull
Open Head Injury

- Results from bullet wounds, etc.
- Largely focal damage
- Penetration of the skull
- Effects can be just as serious
Effects on Brain Structure
Common areas of TBI occurrence
Head Injury Morphology: Cerebral Contusions

- Most common: frontal and temporal poles adjacent to bony surfaces of the skull base
- Contusions of the grey white junction associated rotational/acceleratory vectors and more force
- Clinically, contusions can range from asymptomatic lesions to “massive pulped areas of brain” with resulting herniation
Head Injury Morphology: Cerebral Contusions
Head Injury Morphology: Epidural Hemorrhage

• Usually:
  – Middle fossa via laceration of the middle meningeal artery
  – Lenticular shaped
  – Bounded by suture lines where the pericranial layer of dura attaches to the skull
  – Associated with an overlying skull fracture (often)

• Classically described with an associated lucid interval prior to clinical deterioration, only about 50% of patients present this way

• If detected early they are usually associated with a good outcome and have a mortality of less than 10%
Head Injury Morphology: Epidural Hematoma
Head Injury Morphology: Acute Subdural Hemorrhage

• Usually associated with a much **poorer prognosis**
  – b/c they represent much more severe underlying brain damage

• Torn bridging veins or from associated contusions hemorrhaging into the subdural space

• Subdural hemorrhages tend to follow the shape of the convexities and tend to cover the entire hemisphere
Head Injury Morphology: Subdural Hematoma
Head Injury Morphology: Subarachnoid Hemorrhage

- Usually caused by trauma
- Occurs locally over convexities affected by coup type of injuries or frontotemporal poles affected by countercoup injuries

- Traumatic subarachnoid hemorrhage can result in hydrocephalus
Head Injury Morphology: Diffuse axonal injury

- Pathologic term referring to changes occurring in the brain post TBI as a result of rotational forces

- DAI is the most commonly cited cause, in the absence of hemorrhages or ICP elevations, of prolonged depressed consciousness and of severe disability in brain injured individuals
Head Injury Morphology:
Diffuse axonal injury

• Three grades for DAI are widely used:
  – Grade 1: scattered throughout the white matter of the brain
  – Grade 2: requires the presence of a corpus callosum lesion
  – Grade 3: lesion in the dorsolateral quadrants of the rostral brain stem
    • Grade 3 lesions nearly always result in patients being in a coma or a vegetative state until death
TBI: A *biological* event within the brain

- Tissue damage
- Bleeding
- Swelling
- Cell death
- Stroke
- Seizure
- Other multiple medical complications
Physiologic Response to Brain Injury: Brain Edema

- Intracranial pressure is determined by the volume of three elements in the skull:
  - the brain parenchyma volume
  - the blood volume
  - the CSF volume

- Compensatory mechanisms as ICP rises:
  - initial passive release of CSF into the spinal canal
  - an innate compliance of the craniospinal axis that allows for an increase in intracranial contents without an initial increase in intracranial pressure

- Once the compensatory reserve is exhausted, there is a precipitous rise in ICP
Intracranial Pressure Monitoring

ICP monitoring in persons with:

- severe TBI and abnormal CT scan
- normal head CT if age >40, motor posturing, SBP <90 mmHg
Assessment of TBI

How many fingers am I holding up?
TBI: Changes in **functioning**

- **Injured Brain**
  - Leads to problems in functioning
  - Loss of consciousness/coma
  - Other changes due to the TBI
  - Post-traumatic amnesia (PTA)
How to measure “severity”? 

- Duration of loss of consciousness
- Initial score on Glasgow Coma Scale (GSC)
- Length of post-traumatic amnesia
- Rancho Los Amigos Scale (1 to 10)
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<td></td>
<td>Abnormal flexion pain</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Extension to pain</td>
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<td></td>
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C = eyes closed due to swelling
T = tracheostomy or endotracheal tube

Usually record best arm
F = Fit
Rancho Scale

• Level I - No Response
  Level II - Generalized Response
  Level III - Localized Response
  Level IV - Confused/Agitated
  Level V - Confused, Inappropriate Non-Agitated
  Level VI - Confused, Appropriate
  Level VII - Automatic, Appropriate
  Level VIII - Purposeful, Appropriate
Mild injury

0-20 minute loss of consciousness GCS = 13-15
PTA < 24 hours

Moderate injury

20 minutes to 6 hours LOC GCS = 9-12
PTA = 30m – 24 hours

Severe injury

> 6 hours LOC GCS = 3-8
PTA = >1 day
What Happens as the Person with Moderate or Severe Injury Begins to Recover After Injury?
Recovery and Plasticity

- Plasticity refers to the ability of the brain to recover and regenerate.
- Controversial idea; definition and mechanisms are not clear
- Idea that the CNS is a dynamic system capable of reorganization in response to injury
Determining Recovery Potential

• Some guidelines
  – Lower Glasgow Coma Scale (GCS) Score;
  – Longer coma duration (greater than 4 weeks);
  – Longer duration of Post Traumatic Amnesia (PTA) (good recovery unlikely when >3 months);
  – Older age assoc. with worse outcomes
  – Neuroimaging features (presence of SAH, cisternal effacement, significant midline shift, EDH or SDH on acute care CT = worse outcomes).
Cognitive Impairments after TBI
Effects on Brain Structure
Common areas of TBI occurrence
Cognitive Impairments after TBI

- Post Traumatic Amnesia
- Information processing and attention;
- Anosognosia (unawareness of deficits);
- Intellectual functioning
- Memory
- Confabulation and delusions
- Spatial Cognition
- Chemical Senses (Olfaction and Taste)
- Executive Functions
- Social Cognition and Behavior
Concussions
Concussions

- Synonymous with mild TBI;
- Disruption of neuronal functioning;
  - Metabolic dysfunction at the level of the neuron,
  - Results in a neuronal energy crisis,
  - Leads to interference in cell communication,
  - Further injury or stress can lead to cell damage or death,
  - It takes many days for the neuron to return to normal,
Concussion management

• Acute
  – Rule out more serious intracranial pathology *CT, MRI, neurologic examination primary diagnostic tests*

• Post Injury
  – Prevent against Second Impact Syndrome
  – Prevent against cumulative effects of injury
  – *Less biomechanical force causing extension of injury*
  – Prevent presence of **Post-Concussion Syndrome**
How long for recovery?

• Range of 1-17 days depending on the research.

• Collins et al. (2006) Neurosurgery:
  – 134 High School Male FB athletes
  – 40% recovery by week 1; 60% by week 2; 80% by week 3; remainder remain an anomaly.
Post Concussive Sx

• Physical
  – Migraine/Headaches
  – Visual disturbance
  – Dizziness
  – Noise/Light sensitivity
  – Nausea

• Cognitive
  – Attention/concentration
  – Memory
  – Fogginess
  – Fatigue
  – Cognitive Slowing
Post Concussive Sx

• Neuropsychiatric
  – More emotional
  – Sadness
  – Nervous
  – Irritable

• Sleep Disturbance
  – Difficulty falling asleep
  – Increased sleep more than usual
Computerized Neuropsychological Testing

- IMPACT, CogSport, CANTAB, Headminder,
- Baseline – Concussion – First Follow up – Follow as needed
Concussion/MTBI Issues and Controversies

• About 75% of head injuries are concussions/MTBI (Centers for Disease Control).

• Historical trends have increased TBI/Concussion awareness
  – “Decade of the Brain (90 – 99);
  – Increased media attention to sports concussion;
  – US Military actions in Middle East
This Terminally Ill Former Player Is The New Face Of The NFL's Concussion Issue
Concussion/MTBI Issues and Controversies

• MTBI is a common complaint in workman’s compensation, disability, litigation;
• MTBI is becoming a common outpatient clinical complaint;
• In an MTBI group of disability claimants, about 40% produced invalid neuropsychological test results (Green, 2001).
The Controversy of Prolonged Post Concussive Sx (PCS)

• 90% of athletes demonstrate symptom recovery in 10 days or less.

• In some studies 2.5% are symptomatic after 45 days (McCrea, 2013).

• Studies show no residual cognitive impairment after 7 days (McCrea, 2013).

• World Health Organization (2004) – Persistent PCS symptoms may be attributable to non-injury factors (demographic, psychosocial, medical, situational).
Factors related to PCS

- PCS sx are nonspecific to concussions. Sx found in psychiatric, orthopedic, stress related illness, and multiple other psychological and physical injuries (Larsen, et al., 2013).

- Other factors associated w/prolonged PCS include secondary injuries, suboptimal effort, malingering, psychogenic, iatrogenic factors.
What about Biomarkers?

• Significant functional connectivity abnormalities found after acute MTBI resolve by 7 weeks (McCrea, 2013).

• DTI abnormalities found in acute MTBI were largely normalized by 3-5 months (McCrea, 2013).
Other sources of PCS error

• Psychological
  – PCS patients have high levels of depression, anxiety, and stress response.
  – Attribution error – Patients erroneously attribute sx to brain injury factors, increase stress level, exacerbate otherwise mild symptoms.
Other sources of PCS error

• Iatrogenic
  – Ex. Patient sustains a concussion due to MVA between 1-2 years prior to being seen for neuropsychological assessment
    • Seen multiple providers, records indicate “post concussive symptoms.”
    • Given care and treatment for PCS.
    • Educated on PCS.
    • Given letters to be off work/away from school due to PCS.
  • Remember your Elizabeth Loftus!
Bender and Matuszewicz (2013)

• Iatrogenic error: Provider-Sources
  – “..authoritative sources (e.g., doctors) can erroneously diagnose mTBI and cause false memories to form.” (pg. 115)
  – Injury note transferred in record and accepted at face value.
  – Way information is collected (ie. checklists).
  – Patients often forced to “prove” their symptoms.
Bender and Matusewicz (2013)

- Iatrogenic error: Patient sources
  - “compensation neurosis” in context of legal proceedings.
  - Outright malingering.
  - Factors that can lead to compensation neurosis include: unwarranted suggestions of long-term injury from attorneys and/or family; prolonged litigation; personality characteristics involving dependence, avoidance, or exaggeration; rationalization; and retaliation.
Advent of Symptom Validity Tests (SVT’s).

• Basic principle of SVT’s:
  – Easy enough that legitimate neurological groups will pass them. Exceptions are profound cognitive impairment such as dementia.
  – SVT’s are sensitive to effort. Are not sensitive to brain dysfunction with the exception of dementia.
  – Failure on SVT’s means that performance is suspicious for poor effort.
  – SVT research has led many to question previous research findings and observations.
Data from the clinic.
Clinic Data, Neuropsychological Tests

MTBI

TBI

Vocabulary Matrix
Below black line represents abnormal performance area.

\[ * = \leq .001 \]

\[ .008 \]

\[ .15 \]
Comprehensive Rehabilitation

- Physical Therapy
- Occupational Therapy
- Speech Therapy
- Medical Management
- Psychological/Neuropsychological
- Emotional/Psychiatric Management as appropriate
- Family Support
- Case Management
Nonpharmacological Treatment

• Environmental management
  – Reduce over-stimulation
  – Facilitate adaptive engagement with the environment
  – Support existing strengths and psychosocial resources

• Educational interventions for persons with TBI and their families

• Symptom-targeted PT/OT/SLP
The Role of Neuropsychological Assessment: Historical Perspective

Period of Neuropsychological Localization

Period of Neurocognitive Evaluation

Current Period??
Technician / Artist

• Neuropsychologists are challenged to expand their roles from a purely technical endeavor to a more holistic perspective.
• Cognitive theorist, functional anatomist
Technician / Artist

• Neuropsychologists are challenged to expand their roles from a purely technical endeavor to a more holistic perspective.

• Cognitive theorist, functional anatomist, psychotherapist, family therapist, emotional adjustment, viewing the person from a holistic perspective.
Holistic Neuropsychological Principles

• Empower patients and families to take an active role in the treatment process;
• Believe people with neurological disabilities are more like people without neurological disabilities (ie. *Go beyond the brain*);
• Convey honesty and caring in personal interactions to form a foundation for a strong therapeutic relationship;
• Develop practical plans for rehabilitation; explain rehabilitation techniques in understandable language;
Holistic Neuropsychological Principles

• Help patients and families understand neurobehavioral sequelae of brain injury and recovery;
• Recognize change is inevitable and help families cope with change;
• Every patient is important, treat with respect;
• Remember that patients and families have different perspectives regarding treatment approaches;
• Be willing to refer if appropriate.
Existential Issues in TBI

- Awareness of change;
- Emotions;
- Struggle of acceptance;
- Struggle to make sense and find meaning;
- Struggle to reclaim/find a sense of self
TBI Recovery Challenges

• Knowledge of deficits
• Adapting to deficits
• Grieving and Coping (Denial, anger, bargaining, depression, acceptance).
• Learning and re-learning
• Integrating knowledge into the self
• Re-discovering meaning and a sense of purpose
“...But be that as it may, those of us who did make it have an obligation to build again. To teach to others what we know, and to try with what's left of our lives to find a goodness and a meaning to this life.”
(Quote from the movie “Platoon”, 1986)
How neuropsychological assessment addresses these challenges

1. Knowledge of deficits
2. Adapting to deficits
3. Grieving and Coping (Denial, anger, bargaining, depression, acceptance).
4. Learning and re-learning
5. Integrating knowledge into the self
6. Re-discovering meaning

1. Provides information on cognitive functioning.
2. Presents potential ameliorative strategies.
3. Does not directly address.
5. Presents one aspect of the person (cognition).
6. Does not directly address.
How collaborative neuropsychological assessment addresses these challenges

1. Knowledge of deficits
2. Adapting to deficits
3. Grieving and Coping (Denial, anger, bargaining, depression, acceptance).
4. Learning and re-learning
5. Integrating knowledge into the self
6. Re-discovering meaning

1. Provides information on cognitive functioning and seeks individual application.
2. Presents potential ameliorative strategies and seeks out the individuals own resources for change.
3. Address a person’s experience and reactions to information provided; balances education and the I-Thou interaction.
4. Cognitive rehabilitation and remediation and works to motivate internalization.
5. Presents one aspect of the person (cognition) and considers it within the context of the whole person.
6. Looks toward the future and what all this means for the person.
Methods of Collaborative Neuropsychology

• Demystify the neuropsychological assessment process: Provide feedback report; explain session purpose; facilitate collaboration and empathic understanding
• Answer what the individual wants to know (If you can).
• Explain how strengths and weaknesses are determined.
• Ensure an understanding of the information provided.
Methods of Collaborative Neuropsychology

• Ensure the information relates to the persons experience;
  Or if it doesn’t
• Explore the discrepancy.
• Summarize what has been discussed.
• Make suggestions
• Look to the future.
Applications In Brain Injury (BI) Rehabilitation

- Developing rapport
- Encouraging discussion/elaboration
- Demystify neuropsychology/brain
- Facilitate insight/awareness
- Provide markers of progress
Applications In Brain Injury Rehabilitation

• Demystify experience (ie. Why is ______ happening to me?)
• Educate on brain injury recovery (Expect the worst but hope for the best)
• Provide individualized recommendations for adaptation/remediation
• Addressing Existential issues
Strengths of CTNA in BI

• Evaluation of progress, ie. Does recovery seem to be on the right track;
• Develop personalized recommendations for rehabilitation;
• Insight and awareness (more difficult with anosognosia);
• Helps to explain experience;
• Strengths and challenges for goals;
• Balance hope and painful realities;
• Finding meaning and exploring the integration of mind and spirit
Case Example
Contact Information

Tad T. Gorske, Ph.D., Assistant Professor
Division of Neuropsychology and Rehabilitation Psychology
Department of Physical Medicine and Rehabilitation
Clinical Neuropsychology Services
Mercy Hospital-Building D
Room G138
1400 Locust Street
Pittsburgh, PA 15219
Phone: 412-232-8901
Fax: 412-232-8910
garskett@upmc.edu
http://www.rehabmedicine.pitt.edu/
http://www.linkedin.com/in/tadgorske