TBI/Concussion: Overview, Clinical Aspects, and Evolving role for Neuroimaging

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Potential Conflicts of Interest

Speaker for: Amgen, Avanir, Depomed, Eli Lilly, Oxtellar, Promius, and Teva Pharmaceuticals.

Grant and research support from Amgen, Avanir, Eli Lilly, Gammacore, Impax, Teva, and Dent Family Foundation.
Concussion

**Definition:** A complex pathophysiological process affecting the brain, induced by biomechanical forces. Can be caused by a direct blow or indirect forces. It involves impairment with neurologic dysfunction. Traditional neuroimaging is usually normal. (CT scan)
Concussion as a mild TBI?

- Traumatic brain injury grading classification has been abandoned for a more individualized care approach.

- Presentation/recovery is often paradoxical:
  - Moderate to severe TBI – better recovery
  - Mild repetitive TBI – prolonged recovery

- Could this be the difference between diffuse vs focal injury OR acute vs chronic?

Povlishock, JT and Katz, D The Jrn of Head Trauma Rehabilitation:Jan-Feb 2005, vol 20, Issue 1, p 76-94
Case Study:

- 12 yr old female previously healthy, Level 8 gymnastic.
- She got too much power going into a vault and fell off head first onto the concrete.
- LOC 3-4 min. Memory loss for info prior to the event that day and for everything until that evening at immediate care.
- PE: 2 days later in office. Mild headache, 4/10 daily, worse with exertion but overall ok. Some unsteadiness on tandem gait.
MRI brain series

FLAIR and T1 – right frontal and superior anterior temporal WM changes and encephalomalacia

SWI hypointensities
right frontal
One week later pt comes in with notable bruising behind the left ear. She did not communicate this concern until 1 week later. CT did not show fracture. “Battles sign” – risk CSF leak
<table>
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<tr>
<th>Technique/Modality</th>
<th>Principal Application in TBI</th>
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<tr>
<td><strong>Structural</strong></td>
<td></td>
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<tr>
<td>CT</td>
<td>Intra/extra-axial hemorrhage, skull fracture, cerebral edema, herniation</td>
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<td>MRI</td>
<td></td>
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<tr>
<td>FLAIR</td>
<td>Contusion, nonhemorrhagic DAI, subarachnoid hemorrhage</td>
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<tr>
<td>DWI, ADC</td>
<td>DAI, cerebral edema</td>
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<td>STIR</td>
<td>Orbital or calvarial trauma</td>
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<tr>
<td>GRE, SWI</td>
<td>Microhemorrhages (hemorrhagic DAI) from shearing</td>
</tr>
<tr>
<td>DTI</td>
<td>White matter integrity and connectivity</td>
</tr>
<tr>
<td>VBM</td>
<td>Atrophy, ventriculomegaly</td>
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<td><strong>Functional</strong></td>
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<td>fMRI</td>
<td>Neuronal activation during functional tasks inferred from BOLD signal</td>
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<td>CT/MR perfusion</td>
<td>Quantitative cerebral perfusion</td>
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<tr>
<td>MR spectroscopy</td>
<td>Neuronal loss, edema, inflammation, hypoxia</td>
</tr>
<tr>
<td>FDG-PET</td>
<td>Metabolic changes, task-related metabolism</td>
</tr>
</tbody>
</table>

Abbreviations: ADC, apparent diffusion coefficient; BOLD, blood oxygen level–dependent; CT, computed tomography; DAI, diffuse axonal injury; DTI, diffusion tensor imaging; DWI, diffusion-weighted imaging; FDG-PET, [18F]-2-fluoro-2-deoxy-D-glucose–positron emission tomography; FLAIR, fluid-attenuated inversion recovery; fMRI, functional magnetic resonance imaging; GRE, gradient-recalled echo; MRI, magnetic resonance imaging; STIR, short tau inversion recovery; SWI, susceptibility-weighted imaging; TBI, traumatic brain injury; VBM, voxel-based morphometry.
CT imaging should not be used to diagnose sports related concussion but might be obtained to rule out more serious TBI such as an intracranial hemorrhage in athletes with a suspected concussion who have loss of consciousness, posttraumatic amnesia, persistently altered mental status (Glasgow Coma Scale <15), focal neurologic deficit, evidence of skull fracture on examination, or signs of clinical deterioration. (Level C)

To CT or Not to CT??

Very Controversial!!

Canadian CT head Rule:

Minor head injuries that involve loss of consciousness (LOC), definite amnesia, or witnessed disorientation in patients with a Glasgow Coma Scale (GCS) score of 13-15

High Risk: GCS <15 at 2 hrs, suspected fracture, vomit > 2 xs, > 65 yrs

Medium Risk: Retrograde amnesia (prior to injury) > 30 min, dangerous mechanism.
To CT or Not to CT??

New Orleans Criteria:
Minor head injury with a GCS score of 15.

- Will require CT if any of the following conditions were met:
  - Severe headache, vomiting, >60 years, drug or alcohol intoxication, persistent anterograde amnesia, visible trauma above the clavicle, and/or seizure.
CT Radiation Risks

- Dose dependent
- Younger patient = increased radiation associated disease (cancer) and risk of developmental impairment.
- The younger the age the higher the cell turn over ("Choosing Wisely Campaign").

CT positive findings study

1772 patients

Grp A (1453) - headache only symptom – 6.2% Abnml
Grp B (726) - headache plus symptoms – 13.2% Abnml

CT findings

- 45 professional boxers - serial CT head scans.
- 6 (13%) - evidence of "progressive brain injury."
- 3 boxers progressive cortical atrophy
  (1 with bilateral parieto-occipital encephalo-malacia)
- 3 boxers - cavum septum pellucidum (CSP)
- Progressive CT changes associated with having greater than 10 losses


BLEEDS – Subdural, Epidural, Subarachnoid

1.) **Subdural**

- Extra-axial bleed between the arachnoid and dural junction.
- Stretching of cortical bridging veins, can cross sutures.
- 10-30% of chronic subdural have repeated bleeds.
- **young people** = trauma or bleeding disorder
- **older people** = minor head injury or spontaneous

- Osborn, A. “Osborn’s Brain Imaging, Pathology and Anatomy” Ch 1 Trauma

CT axial – 26 yo MVA
Subdural bleeds on MRI

"Courtesy of Allen D. Elster, MRIquestions.com"
Subdural hematoma

- **Acute** = CT hyperdense (60%), mixed (40%)
- **Subacute** = CT isodense 7-10 days, blooming artifact (hypointense) on GRE/SWI on MRI
- **Chronic** = CT hypodense, loculated

- **Hygroma** (arachnoid tear → subdural CSF)
- **Effusion** (clear fluid after meningitis)
- **Empyema** (pus)

Osborn, A “Osborn’s Brain Imaging, Pathology and Anatomy” Ch 1 Trauma
BLEEDS – Subdural, Epidural, Subarachnoid

2.) Epidural

- Extra-axial bleed between the skull and dura.
- Laceration of the artery (90%), vein (10%).
- Skull fracture in 90-95%.
- Rare = 1-4% TBI, 50% have lucid interval.
- Hyperdense egg shape, “Swirl sign”

- Osborn, A “Osborn’s Brain Imaging, Pathology and Anatomy” Ch 1 Trauma


https://pubs.rsna.org/doi/abs/10.1148/radiology.218.2.r01fe09433?journalCode=radiology
3.) **Subarachnoid (SAH)**

- Bleeding, between the pia mater and arachnoid space.
- **Most commonly** between 25 to 65 yrs, increasing in frequency with age.
- **M/C** extra-axial hemorrhage
- Traumatic SAH > aneurysmal SAH
- Adjacent to cortical contusions
- Superficial sulci > basal cisterns

*Courtesy of Allen D. Elster, MRIquestions.com*
Intraparenchymal Hemorrhage

3D CT reconstruction

Depressed left frontal and orbital fracture.
**Case Presentation:** 59 yr old healthy, professional man. Seen for follow up after a syncopal episode while visiting another state. Pt fell in a public bathroom with unknown time of LOC. Was seen in local ER and then had F/U MRI.
Case Presentation: Seemed fine for 6 months but continued to be very active, playing golf and traveling overseas. Came to the office for opinion due to recent cognitive decline. It was now affecting his job. Exam: slowed deliberate speech, downplayed symptoms, confused at times, looked fatigued, he could not look to the left (neglect of left), right pronator drift.
29 yo MVA with left frontal DAI, previous rt frontal shunt and SWI, now with seizures
CT Perfusion in Cerebral Contusions

- Full Extent of Cerebral Contusions Seen Earlier
- Small Hemorrhagic Focus vs Extensive Brain Perfusion Abnormality

M Wintermark. Admission Perfusion CT: Prognostic Value in Patients with Severe Head Trauma. Radiology July 2004
MRI and mTBI

- **Versatile** – many modalities to use for different aspects of damage.
- **Controversy** - regarding the utility of conventional MRI in mTBI or concussion, cost.

- **MacKenzie et al 2002:**
  - “Longitudinal studies of concussion conventional MRI studies may have a higher utility over time as axonal degeneration evolves. “
  - Retrospectively evaluated the volume of brain parenchyma with mild and moderate TBI.

- Brain atrophy evident at an average of 11 months after trauma.

- Subjects with LOC had increased brain parenchyma loss.

- Volume loss is presumed secondary to the neurodegenerative cascade of axonal degradation after the injury.

Early FLAIR Important

Magnetic Resonance Imaging Improves 3-Month Outcome Prediction in Mild Traumatic Brain Injury

Esther L. Yuh, MD, PhD,1,2 Pratik Mukherjee, MD, PhD,1,2 Hester F. Lingsma, PhD,3

Et al Annals of Neurology 2013

Moen et al, J Neurol Neurosurg Psychiatry 2012

3 Months

12 Months

6 Months
# Automatic Atrophy Quantification


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## General Morphometry Report

<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>LH Volume (cm³)</th>
<th>LH Volume (% of ICV)</th>
<th>RH Volume (cm³)</th>
<th>RH Volume (% of ICV)</th>
<th>Asymmetry Index (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forebrain Parenchyma</td>
<td>520.22</td>
<td>31.02</td>
<td>491.11</td>
<td>28.28</td>
<td>0.70</td>
</tr>
<tr>
<td>Cortical Gray Matter</td>
<td>254.08</td>
<td>10.78</td>
<td>237.58</td>
<td>14.10</td>
<td>10.79</td>
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<tr>
<td>Lateral Ventricle</td>
<td>25.11</td>
<td>1.50</td>
<td>31.11</td>
<td>1.85</td>
<td>-21.34</td>
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<tr>
<td>Inferior Lateral Ventricle</td>
<td>2.31</td>
<td>0.14</td>
<td>2.63</td>
<td>0.15</td>
<td>-0.00</td>
</tr>
<tr>
<td>Hippocampus</td>
<td>2.81</td>
<td>0.17</td>
<td>3.12</td>
<td>0.19</td>
<td>-10.25</td>
</tr>
<tr>
<td>Amygdala</td>
<td>1.02</td>
<td>0.06</td>
<td>1.28</td>
<td>0.08</td>
<td>-22.72</td>
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<tr>
<td>Caudate</td>
<td>3.83</td>
<td>0.23</td>
<td>3.79</td>
<td>0.23</td>
<td>1.10</td>
</tr>
<tr>
<td>Putamen</td>
<td>4.87</td>
<td>0.29</td>
<td>4.41</td>
<td>0.28</td>
<td>6.60</td>
</tr>
<tr>
<td>Pallidum</td>
<td>0.89</td>
<td>0.00</td>
<td>0.85</td>
<td>0.00</td>
<td>4.00</td>
</tr>
<tr>
<td>Thalamus</td>
<td>8.72</td>
<td>0.52</td>
<td>8.12</td>
<td>0.48</td>
<td>7.09</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>58.25</td>
<td>3.47</td>
<td>57.61</td>
<td>3.44</td>
<td>1.10</td>
</tr>
</tbody>
</table>

*The Asymmetry Index is defined as the difference between left and right volumes divided by their mean (in percent).
CT and T2 coronal MRI – frontal contusion
MRI - SWI vs GRE in mTBI

- Gradient echo MRI technique sensitive to hemorrhage that results from DAI and deoxyhemoglobin in venous blood. Susceptibility weighted imaging is superior.

- **Tong et al:**
  - Compare MRI -SWI and GRE in a peds and adolescents with suspected DAI from TBI.
  - SWI identified significantly more small hemorrhagic lesions than the GRE.
  - Degree of SWI correlates negatively with patient outcomes.
  - SWI and acute injury increase the ability to identify small intracranial hemorrhagic lesions.
  - **SWI 3 to 6 x more sensitive.**


MRI - SWI in Peds TBI study: Beauchamp et al (2013)

- SWI lesions detected at all levels of injury (mild to severe)
- 19% patients with negative CT or no CT had SWI lesions.
- Primary location in frontal lobes.
- Volume of lesions inversely correlated with intellectual function even 6 mos out.


Locations of MicroBleeds Correlated to Cognitive Function

Short Term Memory Loss due to Lesions in Hippocampus, Putamen and Globus Pallidus

DWI (Diffusion weighted imaging) in mTBI

- Measures mobility of water molecules
- **Areas of high diffusion** hypointense DWI and hyperintense ADC (vasogenic edema)
- **Acute restricted diffusion** hyperintense DWI and hypointense ADC (cytotoxic edema)
- Decreased ADC is seen in acute and subacute phase DAI.
- Mean whole brain ADC can predict outcome in TBI and can predict duration of coma or functional outcome in patients with severe TBI.

Advanced Neuroimaging of Mild Traumatic Brain Injury Laszlo L. Mechtler, MD a, b, *, Kalyan K. Shastri, MD, MSc, Kevin E. Crutchfield, MD c Neurol Clin 32 (2014) 31–58
Diffuse Axonal Injury - 29-year-old woman with DAI, presenting with loss of consciousness after motor vehicle accident. A. FLAIR image shows a hyperintense lesion in the left middle cerebellar peduncle to cerebellar hemisphere due to DAI. B. DWI shows hyperintense lesions in the corpus callosum and bilateral internal capsules, which is typical findings of DAI.
MRS - Magnetic resonance spectroscopy

MRS = noninvasive assess the integrity of cellular structures by measuring cerebral metabolic changes.

Degrees of irreversible cellular death altering levels of N-acetylaspartate (NAA), total creatine, and total choline.

**Increased** choline levels: myelin injury and cell membrane degradation.

**Decreased** NAA levels: are related to axonal injury.

**Creatine levels**: energy metabolism and mitochondrial function.

Advanced Neuroimaging of Mild Traumatic Brain Injury Laszlo L. Mechtler, MD a, b, *, Kalyan K. Shastri, MD, MSb, Kevin E. Crutchfield, MD c Neurol Clin 32 (2014) 31–58

• 12 college athletes with age matched controls within 6 days of injury.
• NAA/Cr levels were significantly reduced in primary motor cortex in concussed athletes.


• Sports related concussions 14 people
• MRS at 3 days, 15 days, and 30 days
• Decreased NAA/Cr levels at day 3, modest recovery at day 15 and normalization at day 30.
• Athletes reported normalization at day 3
### Table 1
Common neurometabolite alterations in mTBI

<table>
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<th>Neurometabolite</th>
<th>Role in/Marker of</th>
<th>Alteration in TBI</th>
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<tbody>
<tr>
<td>NAA</td>
<td>Neuronal/axonal integrity</td>
<td>Reduced</td>
</tr>
<tr>
<td>Cr</td>
<td>Cellular energy metabolism</td>
<td>Constant</td>
</tr>
<tr>
<td>Cho</td>
<td>Membrane synthesis/repair</td>
<td>Increased</td>
</tr>
<tr>
<td>Lac</td>
<td>Anaerobic glycolysis</td>
<td>Increased</td>
</tr>
<tr>
<td>Glx</td>
<td>Excitatory neurotransmitters</td>
<td>Increased</td>
</tr>
<tr>
<td>Ins</td>
<td>Inflammation (glial cells)</td>
<td>Increased</td>
</tr>
</tbody>
</table>

**Abbreviations:** Cho, choline; Cr, creatine; Glx, glutamate/glutamine; Ins, myoinositol; Lac, lactate; mTBI, mild traumatic brain injury; NAA, N-acetyl aspartate; TBI, traumatic brain injury.
Normal MRS

Myo-inositol - Choline - Creatine - Glutamine/glutamate - N-acetyl aspartate - Lipids/lactate
Fig. 4. Comparison of proton magnetic resonance spectra from a young patient with mTBI (A, B) and a healthy control subject (C, D) showing significant alteration of NAA and Cho in b1 compared with spectra a1, c1, and d1. The b1 voxel is located near injury seen on patient’s T2 MRI in the left splenium. (Adapted from Govind V, Gold S, Kaliannan K, et al. Whole-brain proton MR spectroscopic imaging of mild-to-moderate traumatic brain injury and correlation with neuropsychological deficits. J Neurotrauma 2010;27(3):483–96; and Reprinted from Toledo E, Lebel A, Becerra L, et al. The young brain and concussion: imaging as a biomarker for diagnosis and prognosis. Neurosci Biobehav Rev 2012;36(6):1510–31; with permission.)
Based on modulation of image intensity by oxygenation state of blood.

BOLD (Blood Oxygen Level Dependent) image intensity based on local balance of oxygenated and deoxygenated hemoglobin.

Deoxyhemoglobin is a natural magnetic resonance contrast agent.

Neuronal activation = increased blood flow out of proportion to O2 consumption reducing deoxyhemoglobin.

Totally noninvasive. Without radiation.

Involves a cognitive task to assess function --- Time consuming!
fMRI – Functional MRI

- Two studies: HS football players (mostly linesmen with inc subconcussive CHI), no clinical symptoms of concussion = worsened neurocognitive tests and changes on fMRI (Breedlove et al., 2012; Talavage et al., 2014).

- Former NFL players exhibited functional hypoconnectivity during resting state fMRI and hyperactivation of brain regions during cognitive tasks in fMRI compared to controls (Hampshire et al., 2013). Overcompensation?

Resting-State Functional Connectivity Alterations Associated with Six-Month Outcomes in Mild Traumatic Brain Injury

Eva M. Palacios,1 Esther L. Yuh,1,2 Yi-Shin Chang,1 John K. Yue,2,3 David M. Schnyer,4 David O. Okonkwo,5 Alex B. Valadka,6 Wayne A. Gordon,7 Andrew I. P. Maas,5 Mary Vassar,2,3 Geoffrey T. Manley,2,8 and Pratik Mukherjee1,9

Jrn Neurotrauma 2017
Functional MRI Changes from Trauma

- **Even if No Concussion:**
  - Mid-Season Cognitive Changes

- **After Concussion**
  - Specific Areas of Increased and Decreased Activation
  - Compensation
  - Some studies have suggested we don’t lose function but cannot recruit networks – change connectivity – thalamus has been implicated.

*Talavage et al. (2010)*; *Chen et al. (2007)*

Brain activation during a verbal working memory task.
MRI Perfusion without Contrast: Arterial Spin Labeling (ASL)

- Global, regional and diffuse CBF Reduction in PCS
  - Posterior Cingulate Cortices, Thalamus, Frontal Cortex
  - Associated with Neurocognitive Changes
  - Structural lesions both focal and diffuse can effect absolute CBF in chronic TBI

Compared CBF maps assessed in 18 concussed football players obtained within 24 h and at 8 days after injury with a control group of 19 matched non-concussed football players.

Concussed athletes = decrease in CBF at 8 days compared to control.

Scores on (Sport Concussion Assessment Tool 3, SCAT3) and cognitive measures (Standardized Assessment of Concussion [SAC]) demonstrated significant impairment but returned to baseline levels at 8 days.
DTI – Diffusion Tensor Imaging

- Powerful and tool for evaluating brain structure, especially white matter
- Exploits water’s differential diffusion along versus across axons
- Provides information on axonal direction and integrity
- Images modified for sensitivity to water movement in different directions
- Fractional Anisotropy:
  - Diffusion Directionality – 6 directions
  - Intact Axons: Linear Restriction
• College football players – decrease in WM integrity between pre-season and post-season measures and deleterious effects persist chronically after 6 months of no-contact rest (Bazarian et al., 2014).

• Chronic structural neuronal damage with DTI in recent military veterans (Iraq and Afghanistan) with blast exposure, even in the absence of a clinically evident concussion (Taber et al., 2015).
DTI studies found correlations between the extent of WM damage and severity of TBI \cite{Matsushita2011}, number of TBI \cite{Davenport2012}, and impaired cognitive function \cite{Salmond2006, Miles2008, Niogi2008}.

Several studies showed no brain damage on MR and CT scans at the time of initial examination. Acute studies utilizing DTI immediately following head injury have produced conflicting results \cite{Mayer2010, Henry2011}.
DTI is better in assessing chronic WM changes.

- Acute trauma = increased FA, Chronic = decreased FA

“in frontal and temporal regions, indicating loss of myelin and degenerative changes in the corpus callosum, superior longitudinal fasciculus, internal capsule, fornix, and insula.”

![Illustration of FA changes in concussion](image-url)
Fig. 6. Shown are the ICBM-81 white matter regions, colored to indicate the number of publications reporting white matter abnormalities (regions with no abnormal findings in the literature are not shown).


ACR = anterior corona radiate
GCC = genu corpus callosum
PlIC = Post limb int capsule
SCC = splenium Corpus callosum
PET

- Requires injection of radioactive compound.
- 18F-2fluoro-2-deoxy-D-glucose (FDG) most common
- Brain utilization of glucose – metabolism.
- No acute studies – all chronic
- Pricey as research tool
- Inconsistent results but decreased activation correlated with cognitive testing.

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Relative Sensitivity

MR Spectroscopy (global decreased NAA)
Diffusion Tensor Imaging – (anisotropy)
Magnetic Susceptibility (SWI or GRE)
Apparent Diffusion Coefficient (ADC)
Diffusion Weighted Imaging (DWI)
FLAIR
Conventional MR (T2W > T1W)
CT
Skull Radiogram
# Methods

**Clinical Interviewer**

All patients were seen by Jennifer Williams McVige MD, board certified in pediatric neurology and certified in UCNS headache medicine and neuroimaging, as well as her team of the two nurse practitioners and one physician assistant.

**Patients**

- Age: 1-78 years
- Patients included in the study were stratified into three age-groups:
  1. Pediatric = <13 yrs (86)
  2. Adolescent = 13-18 yrs (274)
  3. Adult = 18 yrs and > (350)

**Criteria (Inclusion)**

- Inclusion (710)
- A total of 870 patients were screened and 710 were included for the study
- Inclusion criteria consisted of patients who had neuroimaging prescribed and completed (610 MRI and 451 CT)

**Criteria (Exclusion)**

- Exclusion (160)
- 160 Patients were excluded if neuroimaging was not ordered or completed AND if there was a lack of imaging within 2 yrs of TBI

**Time Course**

January 2012 – May 2016

Approved by the Western Institutional Review Board
Figure 2. CT Imaging Prior to DENT
Chart shows 410 CT scans completed before DENT. Most prescribed by emergency room or urgent care (322 out of 410 total).

Figure 3. Volume of Neuroimaging Ordered Vs. Volume of Discovered Abnormalities
This graph shows the number of MRIs and CTs ordered compared to number of abnormalities found from each imaging type. 46% more MRIs are ordered but 3.43x more abnormalities were discovered from MRIs vs. CTs.
Patient Examples

Image 1. Encephalomalacia
14 yr F hit in head with tree causing intracranial hematoma and CSF leak. Post craniotomy image with encephalomalacia.
Cause TBI?: No From TBI?: Yes Prolong Recovery?: Yes

Image 2. White Matter Changes
35 yr F history of benign intracranial hypertension and concussion. White matter changes consistent with head trauma and incidental choroid plexus cysts.
Cause TBI?: No From TBI?: Yes Prolong Recovery?: Yes

Image 3. Chiari Malformation
13 yr F neck injury and concussion. Incidental finding Chiari Malformation with syrinx. Subsequent suboccipital decompression.
Cause TBI?: No From TBI?: Yes Prolong Recovery?: Possible

Image 4. White Matter Changes and Brain Bleed
22 yr F pushed out of moving vehicle. Microhemorrhage on SWI in right temporal region. Injury not seen on FLAIR.
Cause TBI?: No From TBI?: Yes Prolong Recovery?: Yes

Image 5. Multiple Sclerosis
34 yr F with multiple falls. Imaging revealed incidental finding of Multiple Sclerosis.
Cause TBI?: Possible From TBI?: No Prolong Recovery?: Yes

Image 6. Glioma
17 yr F sustained a concussion caused by a seizure. Incidental finding of low grade glioma in the right medial temporal region.
Cause TBI?: Possible From TBI?: No Prolong Recovery?: Possible
Abnormalities on MRI believed to cause TBI (definite and probable) were NOT associated with prolonged recovery ($p = 0.8399$).

Incidental abnormalities on MRI believed to prolong recovery (definite and probable) were NOT associated with prolonged recovery ($p = 0.2870$).

Abnormalities on MRI as a result of TBI (definite and probable) were associated with prolonged recovery ($p = 0.0396$).
Thank you
Any questions?