Traumatic Brain Injury (TBI): The Science of Concussions

*It’s all in your head*

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No disclosures
Incidence: an increasing health burden

- From 2007–2013 rates of ER visits increased by 47%*
- 1.7 million in 2006
- 2.4 million in 2010
- 2.8 million in 2013
- No specific data for work related
- Under reported as estimated up to 25% not seen or evaluated
- MVC third leading cause of TBI (CDC)
- Injuries to brain account for 30% of injury related deaths

* All data from CDC
TBI causes:

- Falls number one cause
- Struck by or against an object number two cause
- MVC third leading cause of TBI (CDC)
- All of these may occur in the workplace
Definition

- An external mechanical force causes brain dysfunction *
- A form of acquired brain injury, occurs when a sudden trauma causes damage to the brain.**
- TBI is a nondegenerative, non congenital insult to the brain from an external mechanical force with an associated diminished or altered state of consciousness. ***
- There are at least an additional twenty definitions and none have LOC as part of definition

- Loss of consciousness not required to have a TBI
- Studies done with Glasgow Coma Scale of 13–15 and defines as mild
  *Mayo Clinic
  **National Institute of Neurologic Disorders and Stroke
  ***Medscape
Definition

- Non specific
- External/acquired
- Force
- Brain dysfunction
- Brain damage
- Multiple areas affect function
- Symptoms include “dazed, saw stars, white lights” as initial presentation
Concussion

- Closed head injury with an immediate temporary loss of brain function that involves movement of the head
- Is sometimes used in lieu of TBI
- Is also non specific
- No two snowflakes are alike; neither are any two TBI/concussion
Forces
Anatomy of a head injury

There are two types of concussions, defined by the impact forces that create them: linear and rotational. In both kinds, the neurons, brain and central nervous system cells can be stretched and torn.

**Linear**
1. Direct impact stops the head’s forward motion.
2. The brain keeps moving, colliding with the inside of the skull and injuring the frontal lobe.
3. The brain recoils, crashing into the back of the skull and injuring the occipital lobe.

**Rotational**
A lateral impact, like a cross-punch in boxing, spins the brain on its axis, stretching or tearing neurons.

Source: Centers for Disease Control and Prevention

R.L. REBACH/STAFF ARTIST
Types of forces impact

- Rotational very important
- Post concussion advanced imaging shows changes in mid brain structures which are impacted by rotation*
- This includes corpus callosum
- Biomarkers also elevated in these areas

* 12th International Brain Injury Congress March, 2017
Cingulate gyrus, amygdala, hippocampus have changes in post concussion imaging
Functions

- Cingulate gyrus: emotion formation and processing, learning and memory
- Amygdala: emotions and motivations
- Hippocampus: memory, spatial navigation
- Changes correlate with symptoms
Anatomic changes

- Post concussion changes include inflammation, structural changes, and gliosis*
- Biomarkers confirm these changes but not statistically significant

* multiple studies
Structural changes after one concussion


confirm a pattern of differences in regional volume
Structure loss correlates with symptoms

Figure 4:

A. Bar graph shows that patients with MTBI showed significantly ($P < .01$) elevated scores on clinical symptom scales of depression, anxiety, and PCS compared with control subjects at both initial and 1-year follow-up visits. Higher scores on clinical symptom scales = more severe symptoms; error bars = standard errors of the mean. Average scores were also higher at 1-year follow-up than at the initial visit in patients with MTBI. B. Bar graph created after ANOVA with Friedman test after accounting for six confounding factors (age, sex, injury site, mechanism of injury, loss of consciousness duration, and time between injury and initial imaging) shows significantly elevated depression scores with least-square estimates in patients at follow-up compared with the initial visit ($P = .001$).
Energy crisis at the molecular level along the magnesium pathway (2001)

Acute inflammation that lasts 14–16 days at a minimum and likely lasts longer with current data at 30 days

Energy crisis, decreased cerebral blood flow, axonal injury

*Giza CC¹, Hovda DA. The new neurometabolic cascade of concussion
Neurosurgery. 2014 Oct;75 Suppl 4:S24–33
Force of movements
from Colello
Neurometabolic Cascade of mTBI

- K+
- Ionic flux
- Cell Death
- Protease activation
- Energy Crisis
- Ca2+
- Altered neurotransmission
- Axonal injury
- Glutamate
- ADP
- ATP
- Mito

K+ flow through the neuron and axon, leading to energy crisis and subsequent cell death. The cascade involves calcium influx, altered neurotransmission, and axonal injury.
Chronic Pathophysiology

- Cell Death
- Altered Proteolysis
- Abnormal Protein
- Protein Aggregate
- Energy Crisis
- Toxic Accumulation: Intra- or Extracellular
- Inflammation
- Glutamate
- Altered neurotransmission
- Axonal injury
Inflammation

- Acute inflammation
- Sub acute inflammation
- Chronic inflammation
- Subset of individuals with neurodegeneration longitudinally*

Time course of recovery

- At six months 46% recovery*
- At 12 months between 60% and 90% recovered with many studies showing 90%*
- Commonly accepted is 75% recovery
- Recovery rates correspond to ongoing pathophysiology, types of forces, and systems affected

- 12th International Brain Injury Congress, March, 2017
Significant number of individuals perform poorly on neuropsychological measures at 12 month interval post mild TBI

Linked to post concussion symptoms, mood, and self reported cognitive outcomes

Longer trajectory for recovery

What systems affected commonly

- Decreased cerebral blood flow
- Visual convergence with VOD and accommodation
- Autonomic nervous dysfunction on both sympathetic and parasympathetic tracts
- Always a psychiatric component
- Sleep
- Otic
- Higher cortical functions such as memory, focus with neurocognitive deficits
Assessment protocol

Concussion / Mild TBI

Initial Assessment

>3 weeks, no improvement

Secondary Assessment

Graded Treadmill Test

No symptoms at rest

No symptoms

Recovered

Return to Play Protocol

Physiological PCD

Vestibulo-ocular PCD

Cervicogenic PCD

Symptoms at rest

Symptom onset or exacerbation

Vestibulo-ocular symptoms

Cervicogenic symptoms

No symptom Exacerbation
Common symptoms

- Use questionnaire routinely
- Includes Neurobehavioral Symptom Inventory (NSI)
- Rivermead
- ACE
- Buffalo concussion inventory
- 22 or more symptoms
NSI

- Used by Department of Defense in concussion evaluation
- Skepticism comes as all are deemed subjective
- Commonality of symptoms

*Department of the Army, Defense and Veterans Brain Injury Center, Information Paper, May, 2014  original pub J of Head Trauma, 1995
# Neurobehavioral Symptom Inventory

**Table 1: NSI Symptoms**

<table>
<thead>
<tr>
<th>Item Number</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Feeling Dizzy</td>
</tr>
<tr>
<td>2</td>
<td>Loss of Balance</td>
</tr>
<tr>
<td>3</td>
<td>Poor coordination, clumsy</td>
</tr>
<tr>
<td>4</td>
<td>Headaches</td>
</tr>
<tr>
<td>5</td>
<td>Nausea</td>
</tr>
<tr>
<td>6</td>
<td>Vision problems, blurring, trouble seeing</td>
</tr>
<tr>
<td>7</td>
<td>Sensitivity to light</td>
</tr>
<tr>
<td>8</td>
<td>Hearing difficulty</td>
</tr>
<tr>
<td>9</td>
<td>Sensitivity to noise</td>
</tr>
<tr>
<td>10</td>
<td>Numbness or tingling on parts of my body</td>
</tr>
<tr>
<td>11</td>
<td>Change in taste and/or smell</td>
</tr>
<tr>
<td>12</td>
<td>Loss of appetite or increased appetite</td>
</tr>
<tr>
<td>13</td>
<td>Poor concentration, can’t pay attention, easily distracted</td>
</tr>
<tr>
<td>14</td>
<td>Forgetfulness, can’t remember things</td>
</tr>
<tr>
<td>15</td>
<td>Difficulty making decisions</td>
</tr>
<tr>
<td>16</td>
<td>Slowed thinking, difficulty getting organized, can’t finish things</td>
</tr>
<tr>
<td>17</td>
<td>Fatigue, loss of energy, getting tired easily</td>
</tr>
<tr>
<td>18</td>
<td>Difficulty falling or staying asleep</td>
</tr>
<tr>
<td>19</td>
<td>Feeling anxious or tense</td>
</tr>
<tr>
<td>20</td>
<td>Feeling depressed or sad</td>
</tr>
<tr>
<td>21</td>
<td>Irritability, easily annoyed</td>
</tr>
<tr>
<td>22</td>
<td>Poor frustration tolerance, feeling easily overwhelmed by things</td>
</tr>
</tbody>
</table>
Etiology of symptoms

- Autonomic dysfunction causes orthostasis and systemic fatigue
- Decreased cerebral blood flow causes headache and systemic fatigue
- Psychiatric component of irritability, depression, anxiety caused by stress of dysfunctional pathways and physical dysfunction
Objective testing

- ImPACT test FDA approved
- CNS Vital Signs similar to ImPACT not FDA approved
- BrainScope 300 FDA approved quantitative EEG FDA approved
- CT scan: screening for hemorrhagic lesions only positive 9.6% of the time. Negative does not mean no concussion
- Glasgow Coma Scale (GSC) is triage screening test in ER does not reflect concussion severity
Objective Biomarkers

- Tau protein levels disappointing results and not used
- S100B and Ubiquitin C-terminal hydrolase-L1 are correlated with concussion and mid brain structural changes but not to statistically significant level
- Other biomarkers research
Biomarkers after TBI
multiple mechanisms of action and sites
Biomarkers effects

- Ionic imbalance, energy depletion, cell death
- Disruption of cytoskeleton through increase in extracellular glutamate and intra-axonal calcium
- In term activates many substances that trigger cleavage of NFs and α-spectrin
- Calcium activates transcription factors that upregulate inflammatory mediators such as TNF-α and IL-1β

*Adrian, et. al., Biomarkers of Traumatic Brain Injury: temporal changes in body fluids eNeuro 2016 Dec 21;3(6) 0294–16
Biomarker levels time course
Neurodegeneration: Gliosis


**Figure 1**

Illustration of the glial contributions to secondary injury mechanisms associated with neurodegeneration following traumatic brain injury.
Clinically translatable biomarkers for traumatic brain injury. *In vivo* magnetic resonance spectroscopy (1H-MRS) and glial-specific serum biomarkers may provide the link needed to branch the basic and clinical research arenas.
Objective examination

- EOM
- Tandem walk and stance
- DTRS but not useful as rarely lateralizing signs
- Orthostatic BP readings
- VOD maneuvers
- Mini mental status not particularly useful as multifactorial input
Objective testing: ImPACT

- FDA approved 8/16 for testing of brain baseline and recovery
- Domains tested: verbal memory, visual memory, reaction time, processing speed
- Algorithms built in for age, effort
- Is reliable, valid, sensitive/specific
- To take sample test go to: www.impacttestonline.com/impacttestdemo
- Additional data: go to http://www.impacttest.com/training/events/page/baseline for free courses on testing
BrainScope 300

- FDA approved Dec., 2016 for screening and need for CT quantitative EEG 99% accurate
- Off label use post concussion for longitudinal deficits with 12 programs build in including Rivermead, ACAT, etc.
Vestibuloculomotor testing

- Objective examination
- Cannot have volitional component

Initial Presentation

- Headaches
- Fuzzy thinking
- Personality changes
- Short term memory
- Fatigue
- Dizziness
- Vague sense of abnormalities
- Photosensitivity and hyperacusis
- Lack of socialization
Effects

- Impaired neurocognitive such as thinking or memory
- Movement: dizziness, vertigo
- Sensation for vision/hearing
- Psychiatric/psychological such as anxiety, depression, irritability/anger
- Socialization withdrawal
Time course

- Post concussion may be subtle and not readily apparent
- Peer reviewed study showed symptoms can start up to two weeks after initial injury but may be longer
- Post traumatic epilepsy occurs within two years 80% of the time but can go as long as 12 years
- 11.5% of post traumatic epilepsy at five years
Headaches and Cognition

- Type of Headaches:
  - Post concussive/global
  - Migraine
  - Tension/musculoskeletal

- Cognitive
  - Fuzzy thinking
  - Short term memory
  - Concentration
  - Just don’t feel right
Psychiatric

- Reactive depression
- Irritability
- Anger
- Interpersonal issues
- Anxiety
- Sleep issues
- Frequently not reported by injured except as “my....said I’m.......”
Psychiatric (2)

- A single concussion increases the risk of a Major Depressive Disorder (MDD) by 750%
- Both suicide and homicide need to be screened at initial and subsequent visits
- Self reported depressive symptoms rather than cognitive complaints are associated with objective executive function*

PTSD vs. Psychiatric

- PTSD has specific criteria in DSM V
- Clinical depression has specific criteria
- Reaction to a traumatic event causing a TBI is NOT PTSD
Restorative sleep important in brain recovery
Sleep disorders occur between 30% – 70% of time post concussion and may be up to 100% depending on author
Sleep disorders include insomnia, periodic limb movement disorders (PLMD) and can include frank narcolepsy
Polysomnogram may be needed to determine
It’s all in your head: common misconceptions

- Symptoms occur close to injury
- No objective data to evaluate
- No psychiatric component
- Recovery should be rapid
- There is no defined mechanism of injury to brain structures
- Negative CT means not concussed
- Glasgow Coma Scale normal of 15/15 means not concussed
Symptoms

- Commonality of some symptoms
- Symptoms are individually variable
- Symptoms can occur some time after the date of injury
- Sleep disorders occur in 75% of TBI cases and may or may not be related by patient
If you’ve seen one concussion

- You’ve seen one concussion
Preexisting conditions

- Prior history of ADHD in childhood
- Prior history of headaches
- Prior psychiatric issues/diagnoses including PTSD
- Seizure disorder
- Prior neurologic disorders
Differences in response to concussion

- Age
- Gender
- Prior history of ADHD in childhood
- Prior concussions may or may not be relevant
- There are at least eight anti-inflammatory cytokines in brain and act at variable rate
Return to work

- Early return to work helpful for the brain
- High risk for second concussion in 14–16 day window prohibits for some occupations such as roofer, law enforcement, firefighters
- Criteria includes: working at unprotected heights, weaponized
Red flags

- Used by the Guidelines published by the American College of Occupational Medicine for MSK but not TBI
- CDC does have red flags
- Governmental Agency (GA) guides is support for standard of care
Role of Imaging

- Routine imaging (CT, MRI) only $+10\%$ of time as resolution insufficient to see axonal changes
- Neurosurgeons do not generally evaluate patients with negative imaging
- New technology: Diffusion Tensor Imaging
Diffusion Tensor Imaging

- Not readily available
- Not used in practice
- Research use
- White matter changes, axonal injury
The circled front part of a DTI brain scan shows the most common site for tearing in the white matter in concussions. The standardized CAT and MRI brain scans may appear to be normal in concussion patients, but these techniques do not pick up tears in the white matter. White matter links areas of gray matter to produce thinking functions of the brain, such as attention and memory. If the white matter is damaged by a concussion, the connections get disrupted and attention and memory are affected. Scientists who are part of the BTF research consortium have shown white matter damage which correlates with attention and memory problems. A new technology called DTI, or diffusion tensor imaging, uses advanced software to get more detailed information from an MRI and allows us to obtain images of the white matter.

source: brain trauma foundation
The Holy Grail of TBI is measurement: studies as to biomarkers with tau protein implicated and localized activation of calpain *

“Diffuse axonal injury, a major component of traumatic brain injury, is characterized by a sequence of neurochemical reactions initiated at the time of the trauma and resulting in axonal degeneration and cell death.”

DTI correlates with biomarkers?

Traumatic brain injury, neuroimaging, and neurodegeneration.

Department of Psychology, Brigham Young University Provo, UT, USA; Neuroscience Center, Brigham Young University Provo, UT, USA; Department of Psychiatry, University of Utah Salt Lake City, UT, USA; The Brain Institute of Utah, University of Utah Salt Lake City, UT, USA.


Source: PubMed

ABSTRACT Depending on severity, traumatic brain injury (TBI) induces immediate neuropathological effects that in the mildest form may be transient but as severity increases results in neural damage and degeneration. The first phase of neural degeneration is explainable by the primary acute and secondary neuropathological effects initiated by the injury; however, neuroimaging studies demonstrate a prolonged period of pathological changes that progressively occur even during the chronic phase. This review examines how neuroimaging may be used in TBI to understand (1) the dynamic changes that occur in brain development relevant to understanding the effects of TBI and how these relate to developmental stage when the brain is injured, (2) how TBI interferes with age–typical brain development and the effects of aging thereafter, and (3) how TBI results in greater frontotemporolimbic damage, results in cerebral atrophy, and is more disruptive to white matter neural connectivity. Neuroimaging quantification in TBI demonstrates degenerative effects from brain injury over time. An adverse synergistic influence of TBI with aging may predispose the brain injured individual for the development of neuropsychiatric and neurodegenerative disorders long after surviving the brain injury.
Current research shows white matter changes occur longitudinally in TBI
White matter changes are non specific
White matter changes occur commonly in cerebrovascular disease from hypertension and diabetes but not 100%
Careful correlation between white matter and function
Cannot simply dismiss white matter as caused by other disease
Advanced imaging

- Useful in research and includes PET, functional MRI
- Not readily available in practice
- Not useful except to document changes as can use other tools clinically
Treatments

- Many and varied depending on area involved
- Rx most frequently used is GABA agent as useful for headache and overall brain function
- Tricyclic for sleep and headaches, not given in high enough doses to treat depression and at therapeutic levels has side effects which can confuse clinical course
- Headache depending on type e.g. Topamax, Depakote, Tegretol
- Quantified exercise (aquatic, treadmill)
- Ice topically
- Sleep hygiene
Primary treatment

- Gabapentin 300 mg per day recommended by Brain Injury Association of America
- Exercise with defined parameters
- Need to restore CBF but too much exercise detrimental to brain
- Vestibular
- Neurocognitive
- Breathing
- Psychotropics
Therapy

- Occupational therapy for: neurocognitive, oculomotor, vestibular components
- Physical activity: rest is no longer considered appropriate
- Circulation to brain declines with ten days of lack of physical activity
- Defined structured environment helps both physical and psychological recovery
Ancillary therapies

- Cognitive behavioral therapy (CBT)
- Meditation
- Biofeedback
- Yoga
- Breathing exercises
- Acupuncture, acupressure
Objective testing: biomarkers

- Holy grail
- In process
- Not there yet
- No defined biomarker for brain injury that is reliable or consistent
Case example of severe vs mild

- Driving from work call
- High speed collision on 495
- Unconscious at the scene and intubated in the field
- Permanent total even with marked improvement two years post injury
- Diffuse axonal injury as pathophysiology
FINDINGS: Multiple zones of hemorrhagic and nonhemorrhagic diffuse axonal injury are noted throughout the bilateral cerebral hemispheres as well as the posterior body and splenium of the corpus callosum. The brainstem and cerebellum appear spared. There is diffuse subarachnoid hemorrhage filling multiple bilateral cerebral sulci and interpeduncular cistern. There has been interval development of bilateral frontal CSF intensity extra-axial fluid collections, each measuring approximately 5 mm maximum thickness. There is no hydrocephalus. The basal cisterns remain patent. The major intracranial flow voids are unremarkable. There is mild-moderate paranasal sinus opacification including layering fluid components. The nasopharynx is nearly completely filled with fluid. There is mild bilateral mastoid fluid signal. Incidentally noted is an expansile hyperintense T1/T2 mass within the right petrous apex measuring 1.7 cm AP x 1.7 cm wide x 1.9 cm tall.

IMPRESSION:
1. Multiple areas of hemorrhagic and nonhemorrhagic diffuse axonal injury throughout the cerebral hemispheres and involving the corpus callosum with diffuse subarachnoid hemorrhage as above.
2. Bilateral frontal CSF intensity collections as above, likely representing subdural hygromas.
Occupational medicine approach

- Defined protocol with algorithms
- Evidence based
- Supported by objective data
- Supported by medical literature
- Comprehensive to multiple components
- Psychological component acknowledged and addressed
- Early return to work helps brain recovery with some limits on physical activities e.g. lifting
Summary: the brain is in your head

- Significant incidence in general population, signature injury of Gulf War 2
- Science confirms brain inflammation and damage
- Objective data documents brain injury and recovery
- Multiple therapies needed for total treatment
- Employers need education about brain injury and longitudinal symptoms and need for treatment which may be lifelong
Thank you

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Case study 1 DOI 12/16/16

- Seat belted driver rear ended no LOC occipital scalp struck seat rest
- Immediate headache over left eye
- Seen at Rightime Concussion did nothing
- Non restorative sleep
- Photosensitivity and hyperacusis
- Continued headache and blurred vision
- Much more anxious increased irritability
- First seen 2/9/17
Treatment

- Gabapentin 200 mg/day
- Aquatic therapy
- UVA sunglasses and anti glare CPU screen
- Ice
- Topamax 50 mg bid (prior history of migraine)
Clinical course: typical concussion

- 2nd visit 3/9/17
- “It’s like night and day”
- HA minimal
- Irritability better
- Sent for TOS evaluation for possible seat belt injury
- Final visit: 5/4/17
- HA gone, memory intact, irritability at baseline except when loud noises
- PMD to determine continued Topamax
Case Study 2  DOI 12/25/16

- 24 yo female seat belted driver MVC no LOC
- ER same day CT negative
- First concussion visit: 1/18/17
- Symptoms systemic fatigue, foggy thinking, HA, inability to concentrate, active suicidal ideation w/o intent
- PMH: chronic PTSD, bipolar, ADHD in childhood
- “I’m having thoughts I know I shouldn’t have”
- Psychosocial stressors: living arrangements, job
Clinical course

- Same day referral for psychiatric evaluation but not processed
- Gabapentin 100 mg bid
- ImPACT score 0.43 (normal 0.00 to 0.70 with mean of 0.34)
- Aquatic therapy
- Xanax 0.25 bid and hs
- Off work until 2/2/17 then four hours/day
- Symptom exacerbation when did RTW
Increasing anxiety with psychosocial stressors
Systemic fatigue, neurocognitive difficulties
Non restorative sleep
HA gone
Felt better with aquatics
Suicidal ideation gone, reactive depression continues
Change gabapentin to 200 mg q hs
Add Paxil 20 mg q am
Third visit: 4/5/17

- Panic attacks with Paxil
- New psychiatrist: new Rx, significant improvement on Lamictal
- Back at work full time, sleep now restorative
- No neurocognitive or other symptoms
- New job offers
- Discharged from active care
- Continue gabapentin for another month as Lamictal titrated
Concussion complications

- Pre existing psychiatric dx including PTSD and bipolar
- One concussion increases risk of Major Depressive Disorder (MDD) by 750%
- ALWAYS a psychiatric component to a concussion: irritability, anxiety, depression
- Compounded by lack of socialization and non restorative sleep which are part of concussion
- Sleep disorders occur in 75% of patients up to and including narcolepsy *

*bibliography available upon request
Systemic fatigue from autonomic dysfunction and decrease in CBF

Frequently have exacerbations with RTW and must adjust physical activity to accommodate

Early return to work is beneficial to brain recovery

Brain recovery lags behind clinical recovery so continued Rx

Psychiatrists also use gabapentin when treating acute PTSD
Known differences in concussion response

- Gender
- Prior ADHD in childhood
- Pre-existing psychiatric conditions including PTSD
- Age
Case Study 3  DOI 11/4/16

- Works as Correctional Officer and ran into co-worker while running causing him to fall to floor and strike head
- Witnessed LOC and then intermittent recurrent LOC with confusion
- Taken to UMMS Shock Trauma GCS 13/15
- CT negative
- Observed and improved with d/c home
- Follow up 2 weeks with HA Rx amitriptyline
- Told all symptoms would resolve in another two weeks
First visit: 11/30/16

- ImPACT 11/23/16 negative 0.23
- ImPACT 11/28/16 positive 0.08
- HA 5/7 days, non restorative sleep, photosensitivity, dizziness, does not feel safe driving, irritable/moody, more aggressive, emotional lability, blurred vision
- Rx: sleep hygiene, gabapentin 100 mg tid, increase amitriptyline to 25 mg q hs
- Off work: not safe in prison environment
- Ophthalmology consult
Cognitive function and memory better
HA better qod now
Continued feeling of unsafe
Photosensitivity and hyperacusis
Non restorative sleep
New vertigo
Wife noticed improvement
Add vestibular therapy
May need brief course of Provigil
Increased Gabapentin to 300 mg q hs
Xanax
Third visit: 2/15/17

- HA much improved
- Referred to neuro ophthalmologist by ophthalmologist
- Better in all domains but not normal
- Take Xanax
- Continue gabapentin
- Increase amitriptyline to 50 mg q hs
- RTW on limited schedule
- ImPACT on 2/2/17 increased to 0.21
- MRI Brain two non specific foci of hyperintensity
Fourth visit: 4/5/17

- Did RTW on light duty
- Some unusual feeling only occur at work
- All Rx continued
- RTW full duty one month from light duty
- He feels he is close to baseline function
Recurrent vertigo may be benign positional versus VOD
Add meclizine
Increase Xanax
Vestibular therapy
Continue rx
Sixth visit: 7/5/17

- Dizziness/vertigo
- Did not get ENT evaluation
- No change with start of Xanax taper
- Continue other Rx
Concussion course

- Mild TBI defined by GCS of 13–15/15 on presentation
- Expectation of resolution created issues
- Multiplicity of factors for continued symptoms including non restorative sleep
- New symptoms can occur some time removed from initial brain trauma
- ImPACT documents continued improvement objectively
- May have to be definitive about RTW