The Physiatrist: The Quarterback of Rehabilitation After Brain Injury

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What is a Physiatrist?

A brain injury is complex and multi-faceted. It requires special attention and focus. The Physiatrist not only cares for the patient but also interfaces with the Payor, the Case Managers, Adjustors, Attorneys, multiple Medical Specialists, and the Medical Directors of insurance companies.

- Physiatrists maximize what a patient can do and assist the patient in adapting to what he or she cannot. A physiatrist should be consulted when pain, weakness, or disability is preventing a patient from achieving their desired level of independence.

- Physiatrists work in a variety of environments including inpatient, outpatient, and consulting roles. They can practice solo, in groups, hospitals, and academic settings.

- In addition to management used in general medical practice, physiatrists prescribe therapeutic exercise, prosthetics / orthotics, and adaptive devices to treat patients of all ages.

- Physiatrists facilitate physiologic adaptation to disability to prevent complications or deterioration secondary to disabling conditions.

- The goal of the physiatrist is to provide medical care to patients with pain, weakness, numbness, and loss of function so that they can maximize their physical, psychological, social, and vocational potential

Incidences of TBI

- 2.4 million people, including 475,000 children, sustain a TBI in the U.S. each year.
- 5.3 million individuals live with life-long disability as a result of TBI.
- 52,000 people will die. 275,000 people will be hospitalized.
- 1.365 million people will be treated and released from an emergency department.
- TBIs are caused by falls (35%), car crashes (17%), workplace accidents (16%), assaults (10%), and other causes (21%).
- About 75% of TBIs that occur each year are

  concussions or other forms of mild traumatic brain injury (MTBI).

Traumatic Brain Injury

- Scope of the Problem
  - 1.4 million emergency department visits
    - 124, 000 expected to have long-term disability
  - 3.17 million with long-term disability

Langlois JA et al. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention, 2004

Selassie A, et al. Incidence of Long term disability following traumatic brain injury hospitalizations, United States, 2003. J Head Trauma Rehabil 2008;23:123-131

Zaloshnja E, et al. Prevalence of long-term disability from traumatic brain injury in the civilian population of the United States, 2005. J Head Trauma Rehabil 2008;23:394-400

Scope of the Problem

- Changing demographics
  - 2000 census: 35 million ≥ 65 years
  - 2050: > 86 million

–US Census Bureau 2004

FACTS AND STATISTICS

- 10% of all contact sport athletes sustain concussions yearly.
- 63% of all concussions occur in football. Estimated that up to 20% of football players will sustain a concussion per season.
- An athlete who sustains concussion is 4-6 times more likely to sustain a second concussion.
- “Bell ringers” or mild concussions account for 75% of all concussive injuries.
- Effects of concussion are cumulative in athletes who return to play prior to complete recovery.
- The best way to prevent problems with concussion is to manage them effectively when they occur.
- No athlete should return to play while experiencing symptoms of concussion.

ON-FIELD SIGNS/SYMPTOMS OF CONCUSSION

Concussion Signs	Concussion Symptoms
• Appears dazed	• Headache
• Confused about play	• Nausea
• Answers question slowly	• Balance problems
• Personality/behavior change	• Double vision
• Forgets plays prior to hit	• Photosensitivity
• Retrograde amnesia	• Feeling sluggish
• Forgets plays after hit	• Feeling foggy
• Anterograde amnesia	• Change in sleep pattern
• Loss of consciousness	• Cognitive changes

LATER SIGNS OF CONCUSSION: Post-Concussion Syndrome

- Decreased Processing Speed
- Short-Term Memory Impairment
- Concentration Deficit
- Irritability/Depression
- Fatigue/Sleep Disturbance
- General Feeling of “Fogginess”
- Academic Difficulties


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54
54


HAMILTON
ELLIS
DEFOOR

SECOND IMPACT SYNDROME

Occurs in athletes with prior concussion following relatively minor second impact
- Second impact has been shown to occur up to 14 days post-injury
- Athlete returns to competition before resolution of symptoms

Catastrophic increase in intracranial pressure
- Vasomotor paralysis, edema, massive swelling, herniation, death

Most often occurs in athletes <21 years old
- Neuro-chemical processes appear to differ in developing brain

"…in an adult trauma patient, acute injury is not just a brief physiological setback to a healthy life, but rather signals significant long-term mortality in a large number of patients."

Davidson GH et al. Long-term survival of adult trauma patients. JAMA 2011;305:1001-1007

Proposition

- TBI is not just an event, similar to a broken bone that will heal over time

- TBI is a chronic disease, potentially impacting – Life expectancy
  - Cognitive changes over time
  - Psychiatric disorders
  - Seizures
  - Neuroendocrine disorders
  - Neurodegenerative disorders

Definition: A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function as manifested by at least one of the following:

- Any period of loss of consciousness;
- Any loss of memory for events immediately before or after the accident;
- Any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused);
- Local neurological deficit (s) that may or may not be transient but where the severity of the injury does not exceed the following:
  - **Loss of consciousness of approximately 30 minutes or less**
  - **After 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and**
  - **Posttraumatic amnesia (PTA) not greater than 24 hours**

Erichsen, J.E., 1866

First to describe a syndrome of multiple symptoms following minor or severe head injury.

Reglar, J. 1879

Noted an increase in PCS with introduction of compensation system in the U.S.

Syndrome due to fright, long inactivity, and a desire for financial gain.

Railway Hysteria

Railway traumas can produce a shock to the nervous system causing neurasthenia or hysteria.

Symptoms included:

- **Sleeplessness**
- **Depression**
- **Inability to work**
- **Tinnitus**
- **Vasomotor disturbance**
- **Eye Strain**
- **Spinal pain and twitches**
- **Irritability**
- **Memory disturbance**
- **Headache**
- **Nervousness**
- **Excessive swearing**
- **Enlarged pupils**
- **Pulse irregularities**

Post-Concussion Syndrome

- **Symptoms persist after a *mild* TBI at a rate of 5-20% at 1 year**
- **Chronic pain was increased in *mild* TBI as compared to moderate or severe**
  - ***83% mod-severe TBI had no pain***
  - ***5% mild TBI had no pain and 15% had pain in 4 or more places***

Functional Outcome After Mild TBI

- **Extent of brain damage**
- **Persistent symptoms of injury**
- **Personality style of person**
- **Family and social support systems**
- **Job and home requirements**
- **Age and medical factors**
- **Legal status**
- **Adequacy of medical response to injury**

Mild TBI: Early Preventative Intervention

- **Intervene immediately after injury**
- **Give information and education**
- **Actively manage a gradual process of return to functioning**
- **Whenever possible, negotiate a gradual return to work**
- **Involve the patient's family or significant other**
- **Provide referral or treatment for specific symptoms that do not improve spontaneously.**

Treatment of Persistent Post-traumatic Symptoms

- **Validate the experience of the person.**
- **Do not prematurely confront emotional factors as primary.**
- **Reestablish the shaken sense of self.**
- **Involve the family.**
- **Begin the process of sorting primary for secondary deficits.**
- **Medical intervention for depression and anxiety, headaches, pain in the neck and back, dizziness, vestibular dysfunction, sleep disturbance, and anger management.**

Treatment of PCS

Survey of 165/432 Neuropsychologists
Typical Complaints of PCS

- Poor concentration – 95.1%
- Poor Memory – 89%
- Irritability – 85.9%
- Headache – 85.3%
- Fatigue – 74.8%
- Depression – 71.2%

Treatment of PCS

- Anxiety – 62%
- Dizziness – 50.9%
- Blurry / double vision – 31.5%
- Light sensitivity – 27.6%
- Sound sensitivity – 27.6%
- Other – 16.6%

Medical Issues of PCS

- Headache
- Sleep disturbance
- Diet
- TMJ
- Musculoskeletal complaints
- Iatrogenesis
- Sexual dysfunction

Chronic Daily Headache

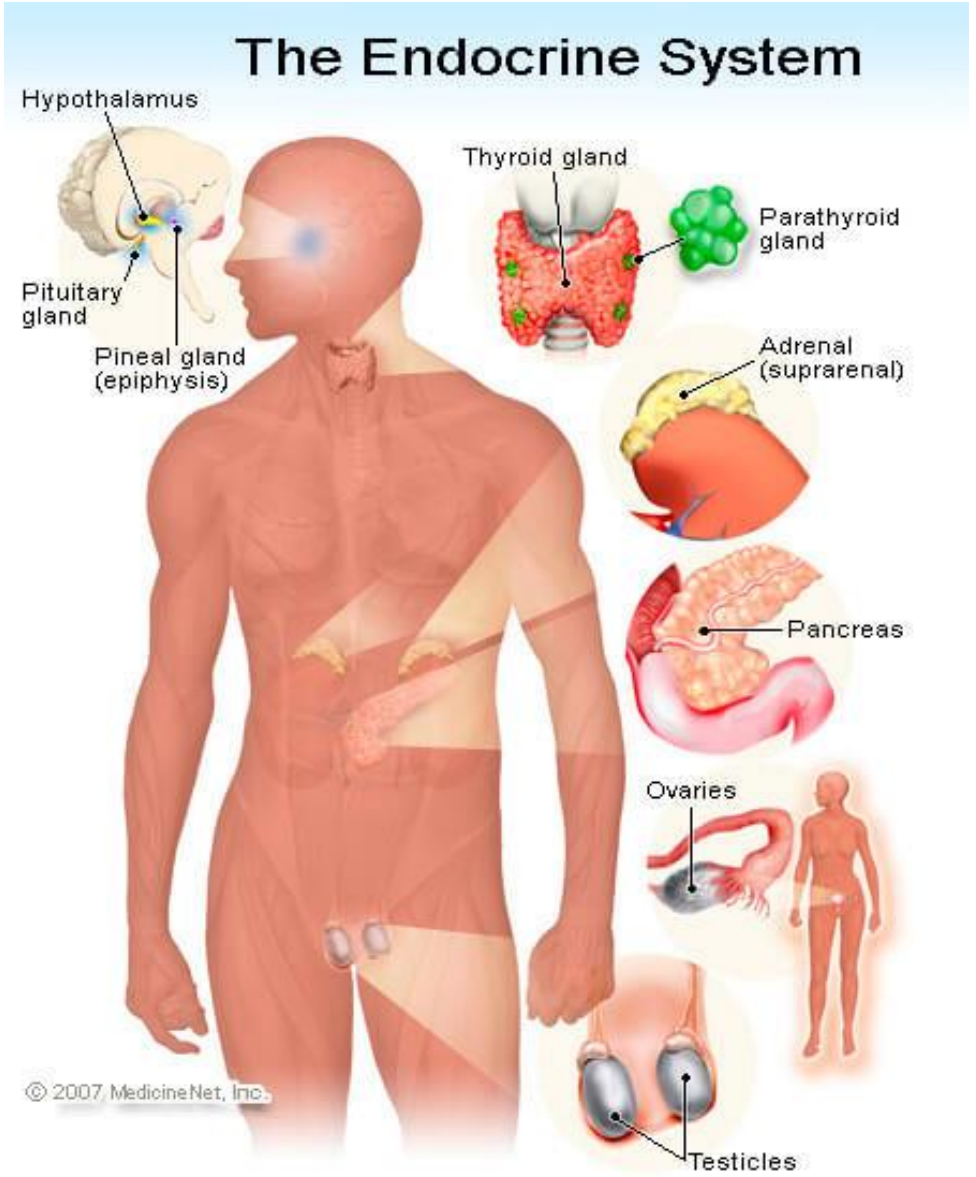
- Generalized headache pain
- Associated with substance abuse, withdrawal, or rebound
- Commonly associated with caffiene, alcohol, nicotine, many analgesics, many antihistamines
- Non-steroidal anti-inflammatory drugs

The Post-Traumatic Headache

- The Patient notes the onset of headache within 24 hours
- Usually consists of a bilateral pressure-like sensation located in the back or front of the head, and can be one sided.
- Complaints increased with exercise, bending over, coughing or rapid movement of the head. Symptoms decrease with rest, relaxation and sleep.
- Migraine like symptoms such as photophobia, nausea and vomiting.
- 20% report symptoms after two years.

SPECIFIC MEDICAL CONDITIONS

Pituitary Dysfunction



Pituitary Dysfunction

Authors	Sample	Years Post Injury	Assessment Method	Outcome
Bushnik 2007	64 with varying TBI severity	10	Serum determination and glucagon stimulation for GH	Severe GHD: 39% Moderate GHD: 27% Central Hypothyroidism: 19% 40%deficient in 1 axis 44% deficient in 2 9% deficient in .2
Bondanelli 2004	50 with Mild-severe TBI	12-64 months (22% >5 years post)	Pituitary screen including growth hormone response to stimulation tests.	54% developed pituitary dysfunction within 5 years post-injury Hypogonadism: 14% Central Hypothyroidism: 8% Prolactin abnormality: 16% Partial GHD: 20% GHD: 8% Older age associated with GHD ($p<.05$); Negative correlation with age & GH response to GH stimulation tests

Pituitary Dysfunction

- Literature suggests
  - Common
  - Older individuals at increased risk
  - Can occur at various times post-injury
- May contribute to post-TBI morbidity
- Suggested guidelines for screening

IOM Report

- The committee concludes, on the basis of its evaluation, that there is sufficient evidence of an association between moderate or severe TBI and endocrine dysfunction, particularly hypopituitarism.

- The committee concludes, on the basis of its evaluation, that there is sufficient evidence of an association between moderate or severe TBI and growth hormone insufficiency.

Neurodegenerative Diseases and TBI: Is there an association?

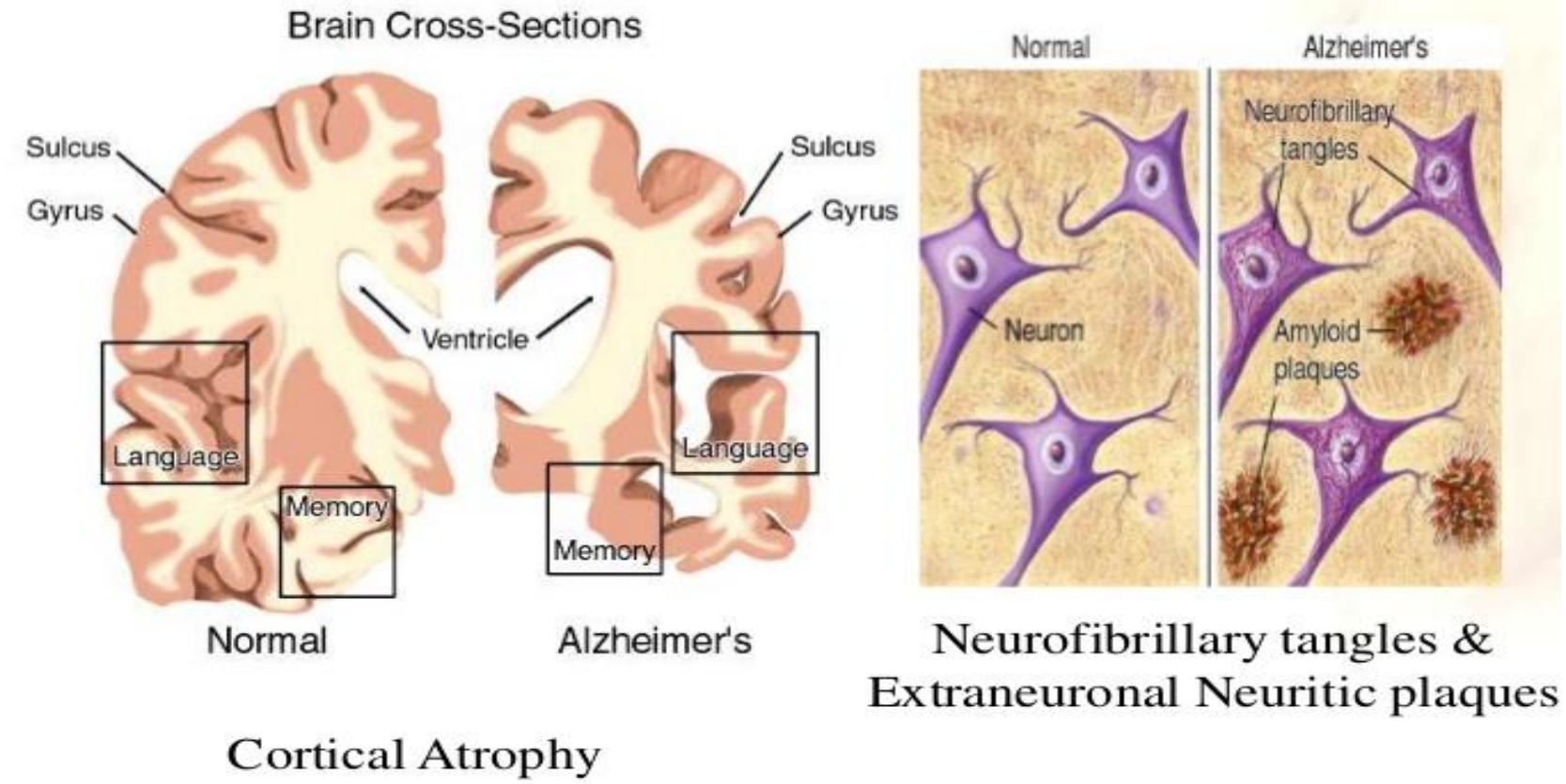
- Dementia of the Alzheimer Type
- Parkinson's Disease
- Multiple Sclerosis
- ALS

IOM concludes insufficient evidence

Dementia of the Alzheimer Type (DAT)

CNS Degenerative disorders...

Alzheimers Disease:



DAT

Significant association	Increased odds (but not reaching significance)
• Plassman 2000* • Schofield 1997** • French 1985 • Broe 1990* • Heyman* • Guo 2000***	• Amaducci 1986 • Broe 1990 • No significance • Guskiewicz 2005

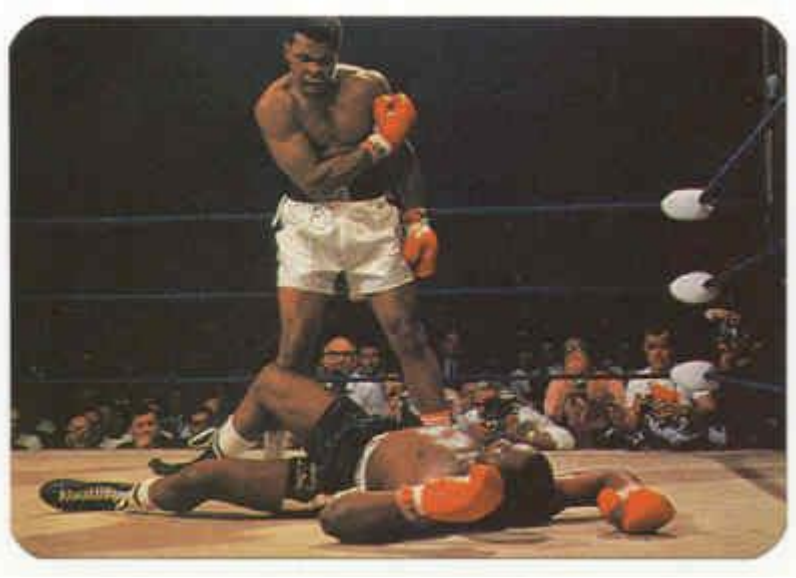
*Moderate to Severe TBI
**LOC > 5 minutes
***+LOC

TBI and Risk of Dementia of the Alzheimer Type IOM

- The committee concludes, on the basis of its evaluation, that there is sufficient evidence of an association between moderate or severe TBI and dementia of the Alzheimer type.

- The committee concludes, on the basis of its evaluation, that there is limited/suggestive evidence of an association between mild TBI (with LOC) and dementia of the Alzheimer type.

- The committee concludes, on the basis of its evaluation, that there is inadequate/insufficient evidence to determine whether an association exists between mild TBI (without LOC) and dementia of the Alzheimer type.




AP

Cumulative Hippocampal Damage Following Repeated Injury

- Differential damage attributable to mild, moderate, and severe levels of stretch injury.
- Neuron-specific enotase (NSE) and S-100 beta protein were evaluated following repeated injury compared to single injury.
- Both glial cells and neurons exhibit increased signs of damage following repeated injury.

Parkinson's Disease

TBI and Long-Term Risk of PD

Bower 2003	**N=196 with PD. Matched to non-PD control Rochester Epidemiology Project**	**Hx of TBI more frequent in men with TBI (OR, 6.0)** **HX TBI more frequent in all cases of PD (OR, 4.3)** **No increased risk with mTBI without LOC**
Goldman 2006	93 male twin pairs discordant for PD	TBI associated with increase risk of PD (OR, 3.0) Risk greater with multiple TBI (OR, 4.3) Small# of twins concordant for PD indicated earlier onset with TBI
Taylor 1999	140 with PD 147 controls	Four factors associated with PD •TBI (OR, 6.23) Family Hx of PD (OR, 6.08) •Family history tremor (OR, 3.97) •History of depression (OR, 3.01)

TB and risk of PD – IOM Report

- The committee concludes, on the basis of its evaluation, that there is sufficient evidence of an association between moderate or severe TBI and parkinsonism.

- The committee concludes, on the basis of its evaluation, that there is limited/suggestive evidence of an association between mild TBI (with LOC) and parkinsonism.

Role of the Vestibular System

- Detects linear and angular head acceleration
- Helps gaze stabilize images in visual field
- Postural controls helps maintain balance
- Spatial orientation or perception of body movement

Vestibular Dysfunction

- **Symptomatology**
- **End of organ pathology**
  - Eng
    - Perilymphatic fistula
    - Cupulolithiasis
- **Brainstem pathology**
  - Vestibulocerebellar pathways
  - Vestibulo-ocular pathways
  - Vestibulospinal pathways

Central Vestibular Lesions

- **Gives rise to vertigo, nystagmus, and imbalance.**
- **Look for other anatomic correlates due to brainstem. "neighborhood" signs i.e., ipsilateral sensory loss in the face or to contralateral sensory loss below the neck or to both, hoarseness, ipsilateral hemiataxia, and facial paralysis.**

Lesions of the Vestibular Nerve

- **Moderate and severe TBI may have temporal bone fractures that make up 22% of skull fractures. There is transection of the nerve due to fracture.**
- **Gives rise to vertigo, nystagmus, hearing loss and imbalance.**

- **Persons with documented peripheral vestibular disorders, excluding persons with central vestibular and Hx of TBI**

- **Pre-morbid psychiatric Dx in 29% (7% depression and most common)**

- **Follow-up: Psychiatric Dx emerged within six months of onset of vestibular symptoms**
  - **Depression – 43%**
  - **Anxiety/panic disorder – 28%**
  - **Phobic states – 29%**

- **Standard treatment of vestibular symptoms was ineffective unless psychiatric symptoms were first controlled.**

Eagger et al.,1992

Lesions of the Labyrinth

- **Secondary to temporal bone fractures there can be displacement of the inner-ear sense organ. There is a rupture of the cochlear or membranous labyrinth and or semicircular canals.**
- **Gives rise to violent vertigo (with nausea and vomiting), complete loss of hearing, ear pain and drainage.**
- ***Perilymphatic fistlas*; if trauma to the external auditory canal there may be loss of fluid into the middle ear cavity.**
- **Gives rise to straining or sneezing that can precipitate vertigo and fluxations in hearing.**

BPPV–Benign Paroxysmal Positional Vertigo

- A peripheral vestibular syndrome seen after head trauma
- Vertigo lasts 30-90 seconds and is violent brought on by assuming a characteristic position.
- Hearing loss and ringing in the ear 9tinnitus) are not common.
- Nystagmus seen after a latency of 5-10 seconds
- Nystagmus disappears with repetition

Vestibular Rehabilitation

- ***Liberatory maneuvers*;** designed to "liberate" the plugs of otolith debris from the semicircular canals in BPPV
- ***Habituation exercises*;** designed to decrease the vertigo associated with motion.
- ***Balance retraining*;** improving the maintenance of the center of gravity irrespective of the source of the problem.
- ***Gaze Stabilization*;** retraining the VOR vestibloocular reflex. The use of special lenses or vision therapy.
- ***Psychotherapy*;** this can often prevent the anxiety from dizziness developing into a major problem.
- ***Medication*;** antihistamines, antidepressants, diuretics and benzodiazepines.

Cognitive Load and Gait Stability in MTBI

- **N = 28 subjects (14MTBI, grade II; 14 matched normals)**
- **Compared gait stability across two concurrent tasks**
  - **Reaction time task**
  - **Continuous sequential question and answer task**

- **Subjects with MTBI adopted a slower, more conservative gait strategy**
  - **Varied with task complexity**

Catena et al, 2006

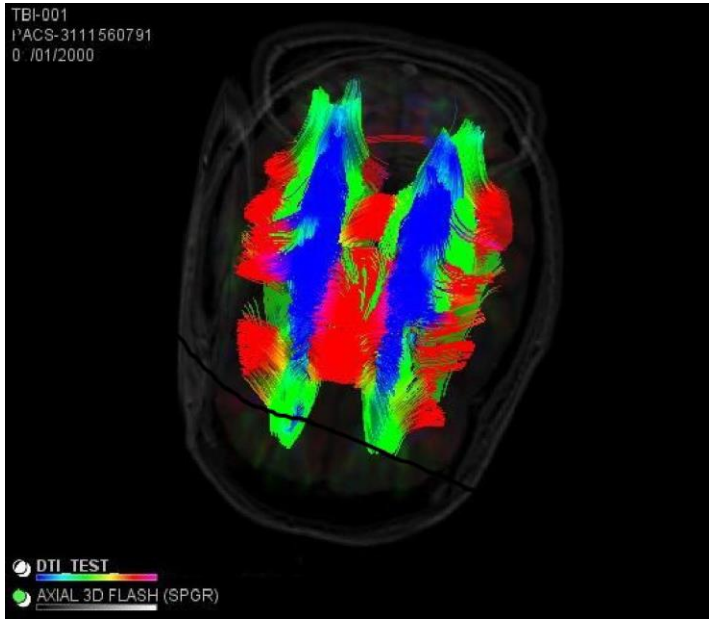
Brain Atrophy and MTBI

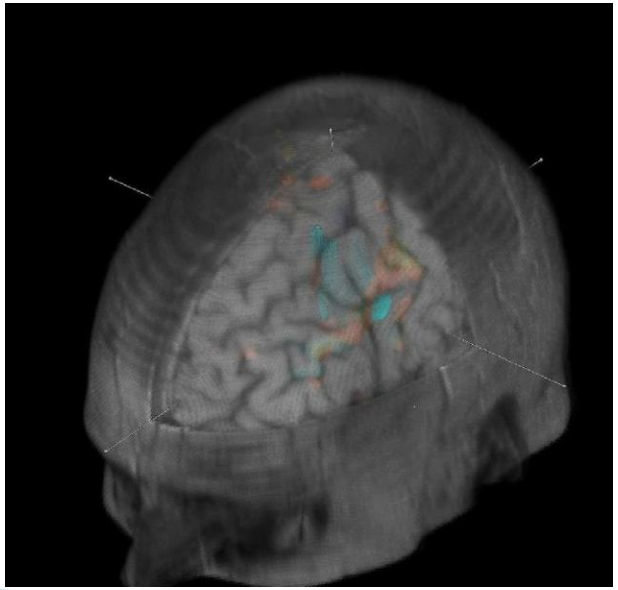
- Whole brain atrophy is evident in mild or moderate TBI at about 11 months post-injury.

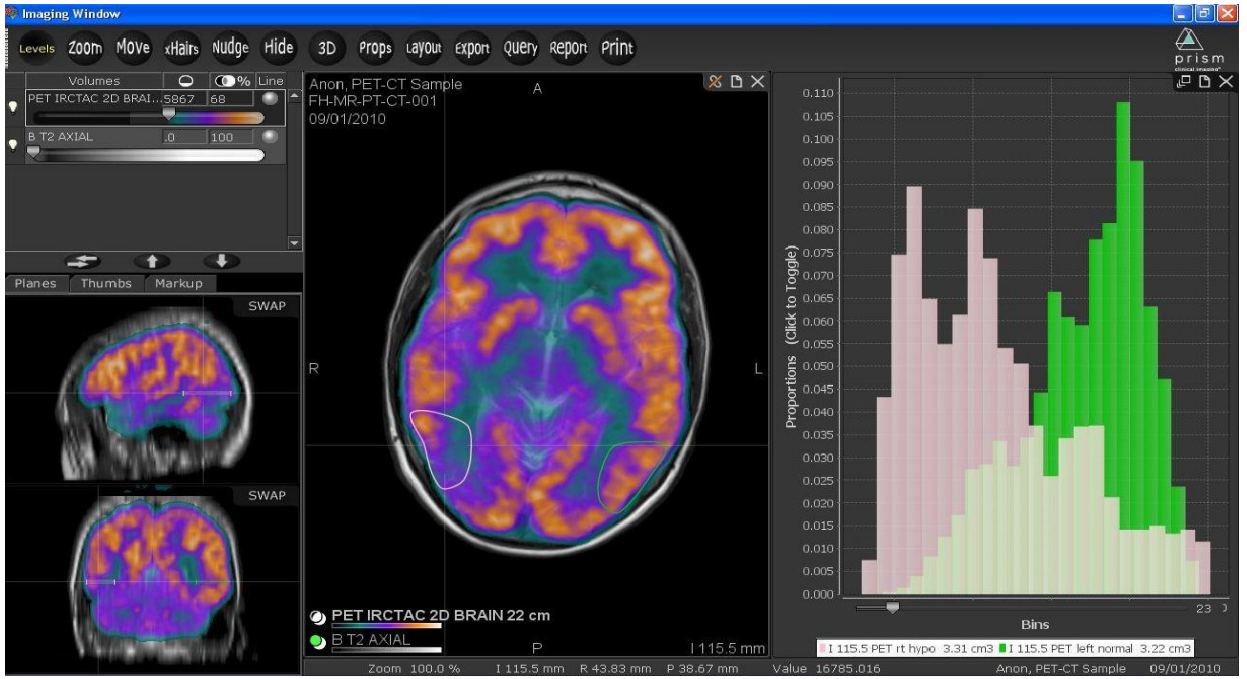
- % change in volume of brain parenchyma is greater in LOC than non-LOC

Repetitive MTBI and Encephalopathy

- Autopsy examination of retired professional football player
- Symptoms of cognitive impairment, a mood disorder, and Parkinsonian symptoms
- No family Hx of AD or any head trauma outside football
- “Chronic traumatic encephalopathy was evident with many diffuse amlyoid plaques as well as sparse nerofibrillary tangles and tau-positive neuritic threads in neocotical areas/”



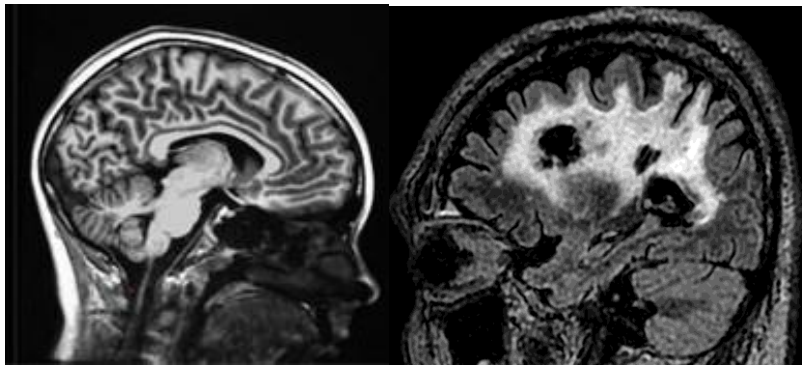




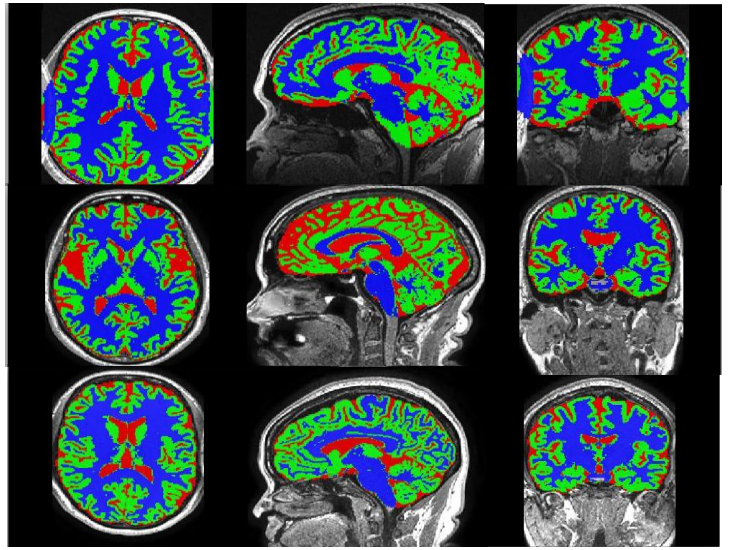
Courtesy Jim Reuss

PRISM

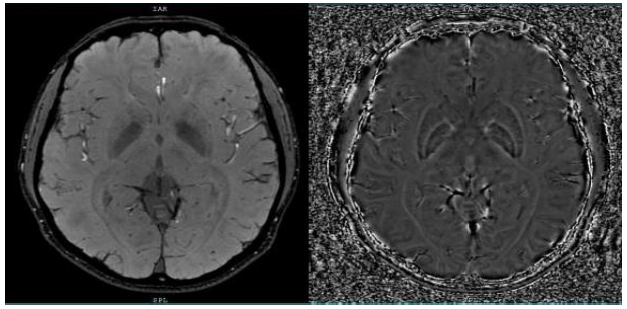
Preliminary Images



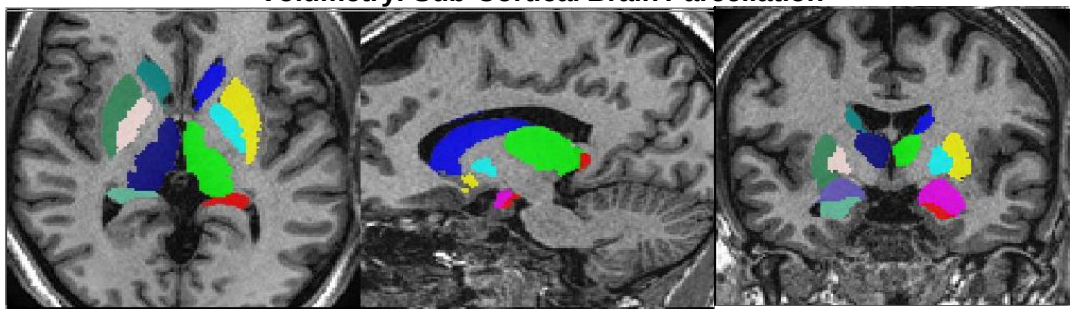
Anatomical: T1 & T2 FLAIR



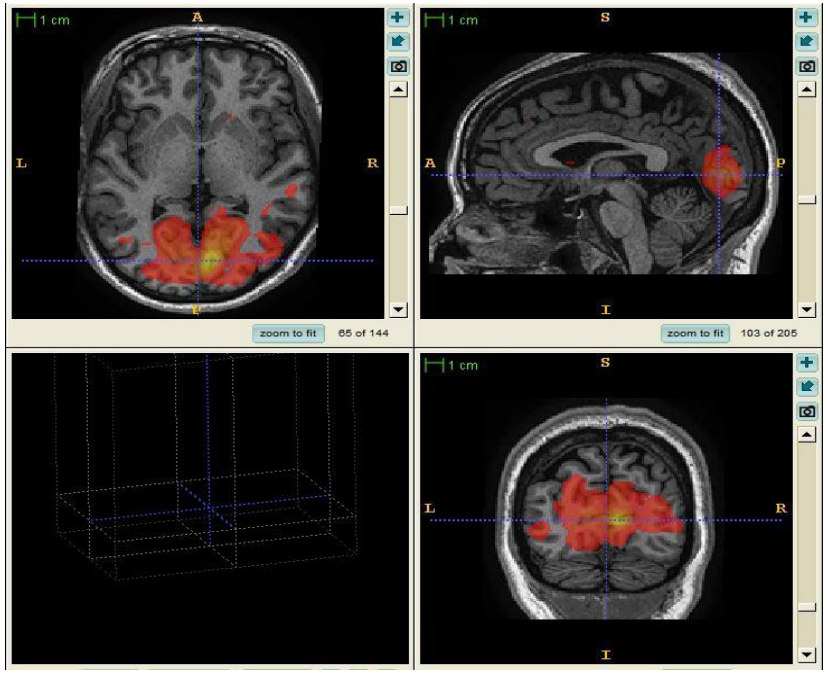
Volumetry: Sub-Cortical Brain Parcellation



Microbleeds: SWAN (Mag + Phase)



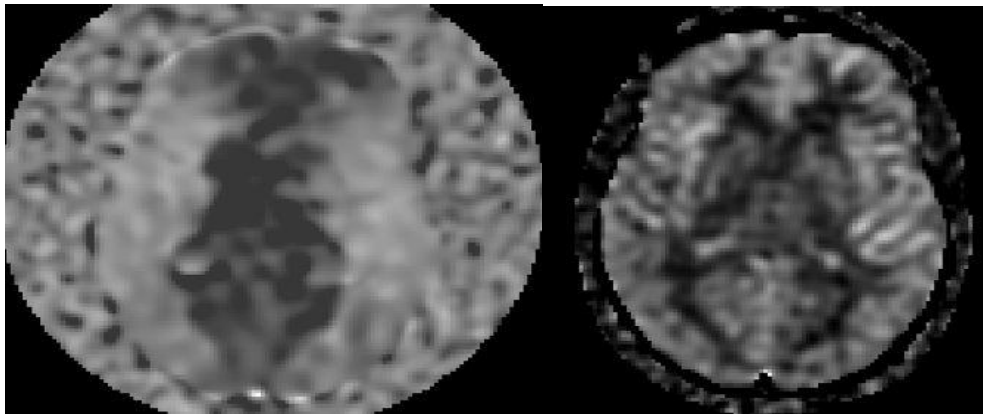
Preliminary Images



Functional: rs-fMRI

TT Maps

CBF Maps



Perfusion: 3D ASL

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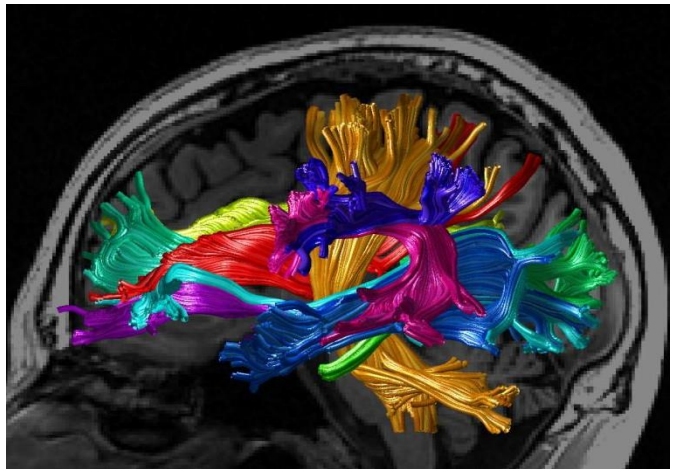
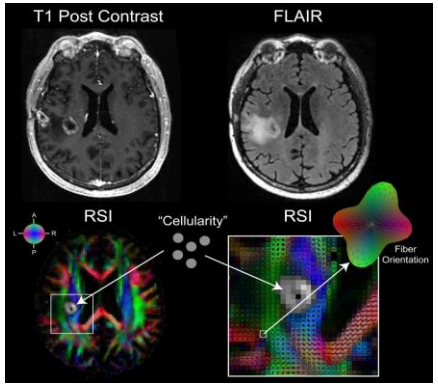
RSI

Preliminary Images

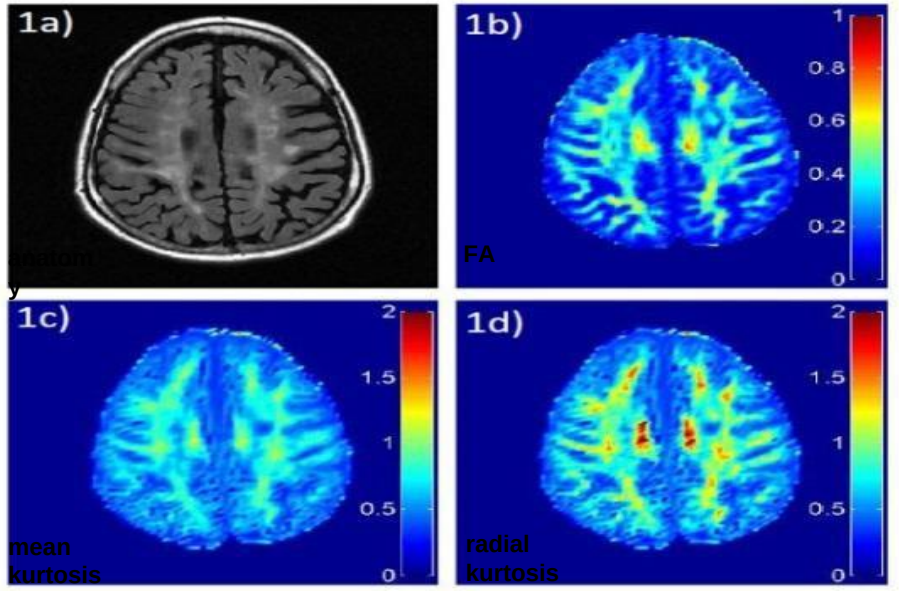


Structural: Multi-Band Diffusion



RSI

Kurtosis



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