## **Traumatic Brain Injury**

Lorenzo Mannelli Nuclear Medicine

#### UW Nuclear Medicine Conference – DS1226 CME Disclosure Statement



## I, Dr. Lorenzo Mannelli have no financial relationships to disclose.



## **Traumatic Brain Injury**

## **Common Data Elements in Radiologic Imaging of Traumatic Brain Injury**

Ann-Christine Duhaime, MD, Alisa D. Gean, MD, E. Mark Haacke, PhD, Ramona Hicks, PhD, Max Wintermark, MD, Pratik Mukherjee, MD, PhD, David Brody, MD, Lawrence Latour, PhD, Gerard Riedy, MD, Common Data Elements Neuroimaging Working Group Members, Pediatric Working Group Members

Arch Phys Med Rehabil Vol 91, November 2010

#### Metabolic imaging of mild traumatic brain injury

A. P. Lin • H. J. Liao • S. K. Merugumala • S. P. Prabhu • W. P. Meehan III • B. D. Ross

Brain Imaging and Behavior (2012) 6:208-223

J Head Trauma Rehabil Vol. 27, No. 3, pp. 216–221 Copyright © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins

Ethical Implications of Neuroimaging in Sports Concussion

J. Valerio, MD. MSc: J. Illes. PhD

Psychotic Disorder Due to Traumatic Brain Injury: Analysis of Case Studies in the Literature

Daryl Fujii, Ph.D. Daniel C. Fujii

J Neuropsychiatry Clin Neurosci 24:3, Summer 2012

**Cerebral blood flow and the injured brain: how should we monitor and manipulate it?** Armagan Dagal<sup>a</sup> and Arthur M. Lam<sup>b</sup>

Current Opinion in Anesthesiology 2011, 24:131-137

Investigating white matter injury after mild traumatic brain injury David J. Sharp and Timothy E. Ham

Current Opinion in Neurology 2011, 24:558-563

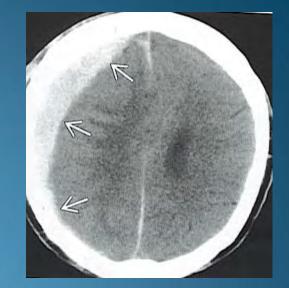
# Why imaging?

- Visualize, categorize, location, nature, and degree of damage
- Triage for acute interventions and determining prognosis
- Long-term outcome

Characterization of basic processes

Mass lesions
Brain swelling
Cerebral blood flow

## CT



- NCT has a pivotal role in the rapid initial assessment of patients with head trauma and in follow-up
- NCT: early surgical management + prognosticate clinical outcome.
- Parameters: skull fracture, epidural or subdural hematoma, subarachnoid hemorrhage, brain parenchymal hematoma/contusion, brain swelling and edema, mass effect, midline shift, subfalcine herniation, effacement of the basilar cisterns

# Marshall and Rotterdam scores

- Incorporate acute CT findings to predict broad categories of outcome.
- Practical and useful

Patient's characteristics	Admission	Clinical course	Outcome
Biological constitution Genotype	Injury details Type (eg. closed, penetrating),	Biological response to injury Metabolomics	Mortality
	cause		Glasgow outcome scale
Demographic factors Age, ethnic origin	Clinical severity	Change in admission variables Clinical severity, change in CT,	(extended)
nget ennie blight	Intracranial (GCS/pupils), extracranial (AIS/ISS)	biomarkers, laboratory values	Health-related quality of life
Socioeconomic status and education		Other predictors Secondary insults, clinical monitoring (ICP, brain tissue PO <sub>1</sub> , evoked potentials)	Hearth-related quality of the
	Secondary insults Systemic (hypoxia, hypotension, hypothermia), intracranial (neuroworsening, seizures)		Neuroimaging
Medical history			
			Neuropsychological assessment
	CT characteristics		, L.

# MRI



# MRI

- Conventional MRI findings are similar to CT, but MRI is more sensitive
- Conventional MR: T1-weighted imaging, T2-weighted imaging, and T2 FLAIR
- Volume quantification: quantitative marker of tissue damage
- Sensitivity increases with magnetic field (T2\* GRE) micro bleeds
- Unconventional MRI: susceptibility-weighted imaging, perfusion-weighted imaging, microstructural white matter tract integrity (DTI), metabolic activity (MR spectroscopy), and hemodynamic brain function (fMRI).

### Mild Traumatic Brain Injury (mTBI)

## **Clinical definition**

#### • Mild Traumatic Brain Injury:

- Post-traumatic amnesia not greater than 24 hours
- after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13–15
- loss of consciousness of <30 minutes</li>
- any loss of memory for events immediately before or after the accident; any alteration in mental state at the time of the accident, and focal neurological deficit(s) that may or may not be transient.

#### CT and conventional MR are normal

#### Epidemiology of Mild TBI

Public Health Problem
 Accounts for more than 75% of all brain injuries.
 Frequently underdiagnosed.

Potentially detectable by: SPECT, PET, MRS (unconventional MRI)

#### **Clinical features of mTBI**

- Common causes were MVA (70%), sporting injury (<14%) and falls (5-10%).
- Natural hx of TBI is toward spontaneous improvement within 3 months of injury.
- Post traumatic amnesia is considered one of the more sensitive markers for those who will suffer ongoing symptoms.
- GCS has often found to correlate poorly with degree of persisting deficit.

#### Sports-related concussion

- 1.6 to 3.8 million each year in the United States
- immediate and long-term neurological problems: headaches, dizziness, behavioral changes, and problems with memory and attention

### Acute and long-term sequelae

- Headache, dizziness, sensitivity to noise/light, insomnia, chronic pain, depression, & fatigue
- 83% report fatigue within 1st 7 days
- 75% report persistent fatigue 6 months
- 32-73% endure fatigue 5 yrs

#### **Cognitive Fatigue**

 Common feature of many complaints such as MS; usually subjective.

Motor fatigue: decline in strength during sustained contractions.

 Cognitive fatigue: decline in cognitive performance over a single testing session in a task requiring sustained attention

PASAT used to measure cognitive fatigue in MS

#### **Cognitive Fatigue**

- PASAT in MS
- Percent decline in performance; the ratio of the number of correct responses for the first 20 items to the last 20: MS 5% decline, normals no decline.
- Some improvement in performance for the first 2-3 trials.

# How to quantify fatigue (role for imaging?)

- Objective neurological/neuropsychological findings vs. subjective experience of mental fatigue
- PASAT as objective measure for producing mental fatigue targeting information processing

#### <sup>99m</sup>Tc-HMPAO SPECT imaging

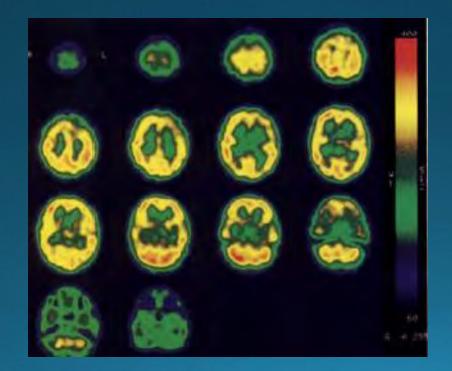
- Studies brain blood flow
- <sup>99m</sup>Tc-ECD and <sup>57</sup>CoCl<sup>2</sup> have also been used.
- SPECT in acute to sub-acute mTBI (92 pts): decreased perfusion in frontal and parietal lobes of adults (children, temporal lobe) – no significant correlation with symptoms
- SPECT in chronic mTBI: decreased perfusion in frontal and parietal lobes – tests at rest or during stress

# SPECT in chronic sports-related head injury

 Hypoperfusion in the prefrontal poles, temporal poles, occipital lobes, anterior and posterior cingulate gyri, and hippocampus – no controls

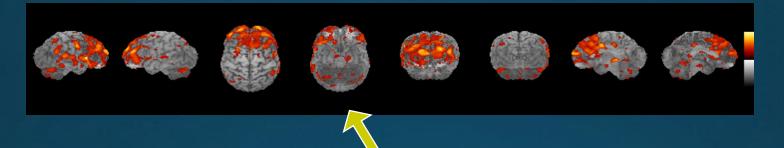
### Role of SPECT in mTBI

• Absence of SPECT abnormalities = prognosis of good recovery (one month, 3 months, 6 months and 12 months after injury).

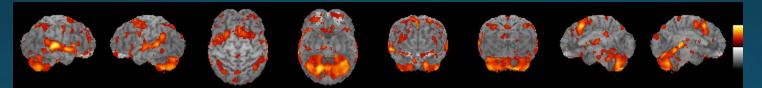


#### Active (PASAT) SPECT

#### **mTBI**



#### Healthy controls

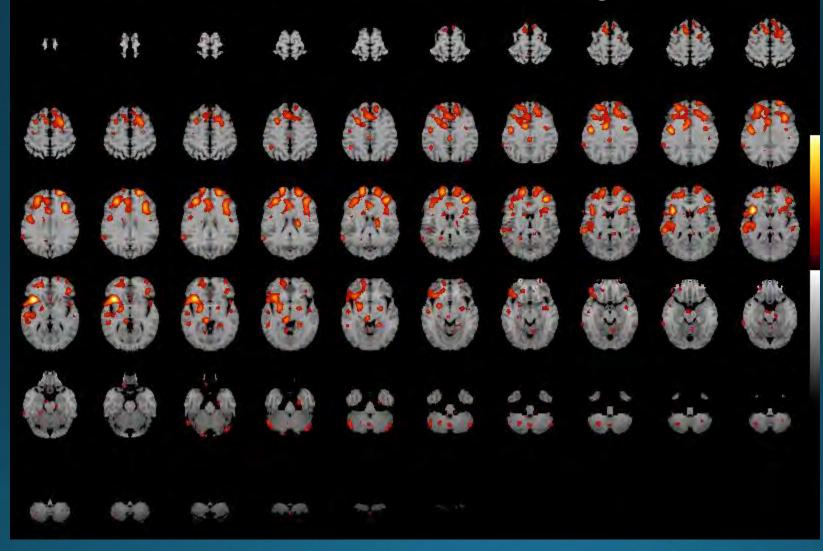




#### Healthy Controls



#### Mild Traumatic Brain Injury

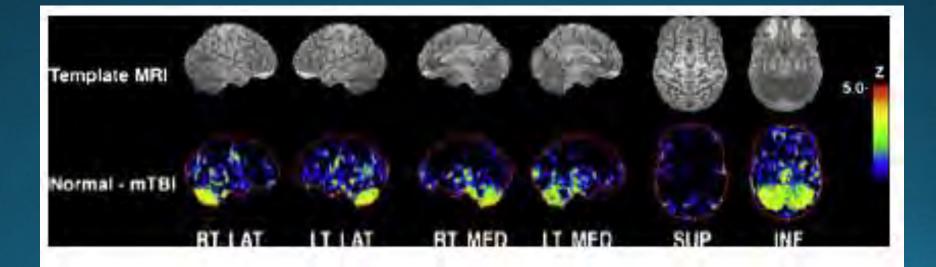


### PET in mTBI

- Decreased FDG uptake in medial temporal, posterior temporal, and posterior frontal cortex in mTBI subjects compared to controls.
- Increases in FDG uptake in the anterior temporal and anterior frontal cortex.

# PET in chronic repetitive head injury

- Boxers: hypometabolism in posterior cingulate cortex, parieto-occipito frontal lobes and cerebellum
- Veterans: hypometabolism in cerebellum, vermis, pons, and medial temporal lobe



## MRS

- Lipid = trauma
- Lactate = poor perfusion/outcome
- N-acetyl aspartate (NAA) = presence of suggests good function
- Glutamate and glutamine (Glx): correlated with outcome (astrocytes)
- Choline (Cho): marker of damage
- ml (myo-inositol): marker of damage (membrane/astrocytes)
- Cr (creatine): reference peak

