CONCUSSION/CTE PRESCRIPTION

READERS SUMMARY:

WHAT REALLY IS A CONCUSSION?
HOW DO I TREAT A CONCUSSION IF IT HAPPENS?
WHAT THE MILITARY,NFL, NHL,MLB, NBA and NCAA SHOULD DO NOW?
WHAT EVERY PARENT NEEDS TO KNOW NOW?

Today marks the fourth blog in this series. Today also marked another early death in the NFL community. This time Orlando "Zeus" Brown was found dead in Baltimore. He was a mammoth offensive tackle who played in the NFL from 1994 to 2005. After hearing about it today I decided to post this blog tonight. I think this information is critical and needs to be considered by everyone at risk right now.

Concussion is a trauma-induced alteration in mental status that may or may not involve loss of consciousness. Headache, confusion and amnesia are the hallmarks of concussion. The confusional episode and amnesia may occur immediately after the blow to the head or several minutes later. I have also seen symptoms appear several days later from concussions especially in the younger patient and in the multiply concussed patient.

Sources of Concussion:

Blasts
Vehicle crashes
Projectiles
Falls
Sports injuries

Symptoms of concussion:

· Headaches or neck pain that do not go away;
· Slowness in thinking, speaking, acting, or reading;

· Getting lost or easily confused;
· Feeling tired all of the time, having no energy or motivation;
· Mood changes (feeling sad or angry for no reason);
· Changes in sleep patterns (sleeping a lot more or having a hard time sleeping);
· Difficulty remembering, concentrating, or making decisions;· Light-headedness, dizziness, or loss of balance;· Urge to vomit (nausea);· Increased sensitivity to lights, sounds, or distractions;
· Blurred vision or eyes that tire easily;· Loss of sense of smell or taste and or ringing in the ears.

Many use descriptive language in trying to relay information about what a concussion is. Many call it a “brain bruise” This is very inaccurate. There are no findings on imaging studies in most concussions. The best way to understand concussion biologically is that it is an energy disturbance caused by the abruptness of the trauma inflicted to the brain. Many studies of a concussed brain show blood flow changes on functional MRI’s and on PET scans. If EEG’s are also done on patients often times there is low voltage findings on the study signifying decreased energy efficiency that leads to acute changes in the neurocircuitry of the surrounding neurons. This injury depletes neurons of energy and this puts the area of injury under much higher risk to future damage because of the accumulation of the release of excitotoxic neuro chemicals released by the cells injured. When these chemicals are released by the damage neurons there is a simultaneous release of magnesium and intracellular zinc and calcium. The calcium release can result in cell death if the injury response is not contained by the brain’s endogenous immune response and by the microglia and surrounding astrocytes. These cells re-uptake the toxic chemicals released and try to contain the release of the the metal ions locally to limit damage. Concussion severity seems to correlate quite well to the release of these substances and the resulting decrease in cerebral blood flow

to these areas.

According to a recent statement by Dr. Robert Stern, co-director of CSTE, new evidence shows" that 85{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} of concussions require about three weeks of recovery" (Abel, D., 2010). This is a longer time period than anything currently in the literature for return to play guidelines. I personally believe this statement does not come close to going far enough for those with severe concussions or those who are multiply concussed.

You also must realize that currently in 2011 there is no "level one data" on concussion risk, or treatment. Therefore there are no uniform agreed treatments. With that as a background one must entirely focus on the known biochemistry and pathophysiology to make best "guesstimates" on how one should proceed once the concussion has occurred.

HOW DO YOU PREVENT A CONCUSSION?

1. Avoidance of activities where there is risk for head trauma.
2. Avoiding situations where there are many angular momentum forces
3. Avoid severe flexion or extenuation of the neck and head.
4. If you are involved in a high risk activity you should consider limiting all glucose consumption for 6 hours prior to the activity.
5. You should maintain a ketogenic diet, loaded with medium chain triglycerides 6 hours before the at risk behavior to create a protective terroir for the brain if injury were to occur.
6. A properly fitted mouthguard should be worn at all times to avoid traumatic impact of the mandibular condyles into the base of the temporal lobes. Many concussed patient have tongue lacerations because they are not wearing mouth guards and their concussion can be quite severe because the hippocampus

is often severely impacted by this type of impact. For athletes or soldiers who need to verbalize to act a voice controlled system should be developed using nanotechnology so they can wear the mouthguard. This should not be compromised due to the severe risks of what hippocampal damage can lead too.
7. All head gear should be mandatory and contain motion and impact detectors to help doctors understand injury vectors to predict injury and severity. (IMPACT evaluation, Miltary Anam evaluation; Parallel biomarker study; Helmets with helmet blast sensors and cooling.) All helmets should have a cooling system built within the helmet to keep the head temperature cooler than normal for the neuro-protective effects of cold temperatures as biomarkers
8. Cooling shirts should be designed and worn to protect the posterior and lateral neck. This allows for a constantly controlled temperature across the vertebral and carotid arteries to mitigate risk before concussion occurs. If this is not worn, Ice packs should be immediately placed and secured to this area for any traumatic risk. Body and head cooling techniques should be used on the sideline, bench or in military theaters.
9. Prior to onset of high risk behavior one should consider supplementing with 25 mgs of zinc, 400 mgs of Magnesium. Ingestion of coconut oil in packet form should be considered as soon as help arrives to improve the neuronal energy deficit and limit neuronal damage. All patents at risk should have baseline neuro-cognitve batteries made mandatory. The younger the patient the more the testing will need to be enforced.
10. All at risk patients should avoid all MSG, artificial sweeteners, and excito-toxins to limit risk of neuronal damage pre and post injury occurrence.

WHAT SHOULD BE DONE ONCE A CONCUSSION OCCURS?

A. All people at risk should undergo fMRI pre and post injury/deployment as well as in game/theater scanning.

Functional MRI/PET scanning should be required in all front line military and sports venues.
B. Resting fMRI/PET scanning should be able to distinguish mTBI, PTSD as it has with 80-90{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} accuracy distinguished from neurodegenerative disorders. All fMRI scanners should have minimum 3.0 tesla MR scan capability. All scanning needs to be done during waking hours (sleep deprivation causes negative scan effects).
C. Ketogenic packets, supplements available at all at risk events. Strict avoidance of all glucose and fructose drinks or foods during at risk activities to limit risk.
D. All patients should be removed from all activities and assessed by a neurosurgeon/neurologist with correct skill set. No exceptions should be made.
E. Avoid excessive bright light in the eyes due to thalamic sensitivity/ lateral geniculate nucleus. Patients need DHA replacement. Real foods high in DHA are a better choice than DHA supplements because DHA needs to be in the SN-2 position to get into the brain.

WHAT IS THE IDEAL STUDY FOR CONCUSSION AND CTE RISK?

A. The ideal study of at risk patients: Combine/Preseason/Pre Deployment scans for baseline with formal neuro-cognitve battery.

B. Organ donor status made mandatory to allow postmortem exam of at risk patients. Allow for concussion history; Thorough neuropsychology evaluation; Genetic Testing for ApoE 4 allele

C. Scanning in dedicated scanner before at risk activity begins (3 Tesla MRI magnet fixed scanner)

D. Parallel biomarker study modeling the VA’s program for the military. (Banyan Biomarkers)

E. Deployment/In Game injury: Scanned in theater/ City post concussion (3T fixed scanner) with Anam evaluation Parallel

biomarker study, review of Helmet sensor and vehicle blast sensor data to assess risk.

F. Post Deployment/Post Game management: Repeat scanning post deployment in exact same scanner as pre-deployment/game assessment. Complete neuropsychology evaluation with sequential brain examination using neurocognitive testing parameters like IMPACT/KIA Continued surveillance in civilian status through Veteran's Administration Parallel biomarker Study

WHAT ARE THE SYMPTOMS OF IMPENDING CTE FOR THOSE AT RISK TO BE AWARE OF?

The clinical symptoms associated with CTE vary in severity depending on which clinical stage the individual is in (McKee, A.C., et. al., 2009). Initial early symptoms include the following:

– Deterioration in attention, concentration, memory, poor decision making
– Disorientation to person, place or time.
– Confusion that waxes and wanes. This is dramatic when the patient is intoxicated.
– Dizziness by spatial of visual cues.
– Headaches become very common. Cervical pain is also a common feature.
– Lack of insight, lack of empathy, and a personality change is apparent to everyone who knows them
– Poor judgment involves use of drugs, alcohol, risky behavior,
– Overt dementia becomes obvious to friends and family.
– Slowed muscular movements with increased activity.
– Staggered gait and loss of balance is common.
– Impeded speech: Word finding difficulty is the first sign one usually reports.
– Tremors and fibrillations in major muscle groups
– Vertigo at rest or during motion

– Deafness that can come one abruptly

The individual may progress through three stages of the disease beginning in the first stage with affective disturbances and psychotic symptoms. The emotional lability and change in personality are standouts of this stage. As the disease progresses to stage two, the individual may suffer from social instability, erratic behavior, severe memory loss, and the initial symptoms of Parkinson's disease (McKee, A.C., et.al., 2009) . Many functional movement disorders can be seen at this stage of the disease progression. Family members should look for them to alert the doctors of the progression. Alcoholism and drug abuse are particularly common in people developing CTE from their concussions. Generally, the more pathways of inflammation that the patient sustains at this time the quicker the symptoms tend to progress. This can be from obesity, drugs, alcohol, or environmental toxins. Sometimes, the symptoms progress rapidly when their are acute spikes in cortisol, insulin, or in ingestion of excitotoxins. A new trauma or life stress can also precipitate a serious neurological decline.

The 3rd and final stage consists of a progressive deterioration to dementia and may have other symptoms including the signs associated with Alzheimer's disease or Parkinson's disease, speech difficulties, gait abnormalities, dysarthria (speech disorder characterized by neuromuscular weakness or lack of control of facial muscles), dysphagia (difficulty swallowing), and ptosis (drooping eyelid) (McKee, A.C., et.al., 2009).

How is CTE distinguished from other neurodegenerative tauopathies, (i.e. Alzheimer's Disease)

A. CTE usually Involves superficial cortical layers, specifically neocortical layers 1 and 2.
B. Patchy distribution in frontal and temporal cortices is the rule in CTE and not in AD or PD.

C. Propensity for injury in sulcal depths as opposed to surface gyri – Deposition of Î²-amyloid occurs in <50{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} of cases – Marked accumulation of tau immunoreactive astrocytes (concentrated around glutamate receptors of the neocortex)
D. Superficial cortical laminae: layers II and III Perivascular Patchy, irregular Prominent glial tangles
E. Key MRI findings: Often greatest at sulcal depths, Dot like tangles, spindle-shaped neuritis, subcortical white matter defects on STIR or T2 imaging. Dilated ventricles due to cerebral atrophy, fenestrated septum pellucidum is extremely common in this group, atrophic mammillary bodies (due to memory loss), loss of thalamic volume, loss of neocortex volume, Loss of medial temp lobe structures resulting in profound memory changes.
F. Decreased Apo E and AÎ² spinal fluid concentrations in acute injury settings. Most patients do not have CSF examined but this should be considered by neurosurgeon/neurologists rendering independent assessments in severe persistent concussion symptoms. Regular MRI/CT is worthless in initial assessments. Functional MRI, connectome mapping and Diffusion tensor imaging should be considered gold standard imaging testing for this group of patients. (tractographic imaging)
G. Auto antibodies can show up 5-7 days post injury and can persist for years in at risk patients. This should be mandatory testing for all at risk patients post injury for their lifetime. (4 biomarkers in clinical testing)

Conclusions:

fMRI, through connectome mapping, will detect organic changes in functional neural architecture after significant mTBI. These can all be made available in most cities supporting professional athletics of in the military theater. For younger patients parents should assess whether these treatment options exist in their areas before consenting to allowing

participation in at risk activities.

The presence of these changes should serve as a warning that repeated exposure to concussive forces may lead to the development of chronic traumatic encephalopathy as the patient ages. This tool should be used in all cases in my opinion. Cost is steep but the cost of CTE is steeper to the patient and to their family due to its debilitating and possibly lethal results.

The pairing of RfMRI with other biomarker studies will provide cross validation of methods and the development of less complex methods of detection of significant mTBI, i.e., easily obtained serum biomarkers. (Banyan Biomarkers)
Dietary and supplement regimens should be instituted at the time of injury. Neurosurgical or neurology consultation should be mandatory in all cases and decision tree analysis should be uncoupled from the employment/deployment situation in all cases.
Everyday more data is released causing me to update this Rx.

CITES:
http://journals.lww.com/neurosurgery/Abstract/2011/07000/Emerging_Histomorphologic_Phenotypes_of_Chronic.20.aspx

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Thalamus Damage and Persisting Concussion Symptoms Radiology. Tang L, Ge Y, Sodickson DK, Miles L, Zhou Y, Reaume J, Grossman RI.
http://www.cdc.gov/Concussion/