The Neuro-ophthalmology of Concussion

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Dr. Messner has received honoraria from Carl Zeiss Meditec. He is on the speakers’ bureau for Carl Zeiss Meditec.

Dr. Leong is employed by King-Devick Test, LLC as the Director of Research.

Chris Nowinski

Trainers rushed to Johnson’s aid. Minutes later, they led him from the field. Upon reaching the sideline, Johnson fell to his knees and clutched his head. Never there was a defining image of the epidemic of concussions sweeping the NFL, Johnson embodied it.

What happened next shows the NFL’s inability to improve safety and a $30 million donation to the National Institutes of Health to study brain trauma — still cannot claim to have a coherent, effective way of dealing with concussions. We lack adequate tools for diagnosing them on the field.

Just 11 minutes and 44 seconds after taking a hit that should have ended his game, Johnson returned to the field. In the fourth quarter, after all, and the Lions were down 14 points to a division rival. There was no question “he’d play,” Johnson told Detroit’s WXYT-FM the following day. “You get concussed, you gotta keep on playing.”

The NFL’s Rush to Build Unbreakable Players

Even more stunning, Johnson and his own coach can’t agree on what happened. Four days after the Sept. 30 matchup, Johnson announced he’d finished the game with a concussion. Not so, insists Lions head coach Jim Schwartz, who asserted at a press conference that Johnson “was thoroughly checked” on the sideline and cleared to resume playing.
**Concussion**

- Complex pathophysiological process resulting from traumatic biomechanical forces
- Functional rather than structural injury
- Mild traumatic brain injury (mTBI) – Diffuse axonal injury
- Loss of consciousness in less than 10%


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**Epidemiology**

- 300,000 to nearly 4 million per year
- Problem of under-reporting and debate regarding definition
- Multiple studies suggest rate on rise
- Girls have a higher rate of concussion
- Boys’ High school football followed by girls’ soccer lead the list
- Nearly 85% of concussions may go undiagnosed

*JAMA*, October 27, 2010

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**State concussion bill closer to reality**

House panel agrees on plan to protect kids

By: Jim Siegel
The Columbus Dispatch – Saturday, June 1, 2012 7:10 AM

An ally to educate coaches and ensure that young Ohio athletes are pulled from competition when they show concussion-like symptoms passed a House committee yesterday after 10 revisions and several months of debate.

There has been general agreement that more needs to be done to protect young athletes from the dangers of concussions, but legislators have struggled to balance issues regarding liability and risk.

House members still expressed reservations yesterday.

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**Concussive Sports Injuries**

- School sport concussions drawing national attention
- Collegiate, high school
- NFL interest in chronic traumatic encephalopathy
- 20 to 30% of Alzheimer’s patients report head trauma vs. 8 to 10% of controls

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**High School Concussions** (per 100,000)

- Football: Between 60 and 76
- Gir’s soccer: Between 33 and 35
- Boys’ lacrosse: Between 20 and 31
- Boys’ soccer: Between 20 and 31
- Boys’ wrestling: Between 17 and 23
- Girls’ basketball: Between 16 and 18
- Softball: Between 11 and 16
- Boys’ basketball: Between 11 and 21
- Girls’ field hockey: Between 10 and 24
- Cheerleading: 11
- Girls’ volleyball: Between 5 and 8
- Boys’ baseball: Between 4 and 6


**Hockey Concussion Education Project (HCEP)**

- 67 male hockey players (ages 16-21)
- Prospective, physician-observed surveillance for concussion (2009-2010)
- 17 players sustained 21 concussions (25%)
- 5/17 (29%) sustained a second concussion
- 15/17 (88%) players admitted to prior concussion
- Concussion by position:
  - 71% forwards
  - 29% defensemen

*Neurological Focus 2010*

**Challenges: Recognition and Management of Concussion**

1. Wide variety of symptoms or delayed onset
   - easily “missed” by coaches, medical staff
2. Denial of symptoms (“I feel fine”)
   - Metabolically-mediated euphoria (McKee 2010)
3. Most current diagnostic/management protocols not evidence-based
4. Traditional neurological / radiological studies (CT, MRI, EEG) normal with mTBI
   - Metabolic vs. structural changes

**Youth Sports & Concussion**

- Children are more susceptible to head injury than adults and require a longer period of recovery

*Kirkwood MJ, et al., Pediatrics 2006*

**“Structure vs. Function”**

- Concussion is a metabolic rather than a structural abnormality
  - Detectable abnormalities {N-acetylaspartate (NAA)} on MR spectroscopy

*Vagnozzi R, et al., Brain 2010*

**Cognitive Impairment in Children with TBI (Impaired Neuroplasticity and Brain Development)**

*Scherwath A, et al., Brain June 2011*
WWI

- Advent of trench warfare and continuous shelling
- 10% overall fatalities due to shrapnel/indirect trauma
- Term “shell shock” used to describe psychiatric findings

Major Frederick Mott

- British neuropathologist who proposed studying brains of deceased WWI soldiers
- Questioned link between “shell shock” and organic brain disease
- In 1917, British Army bans further use of term “shell shock”

Combat-Related TBI

- Most deployed soldiers report one or more blast injuries
- TBI proposed as “signature injury” of modern combat
- Limited data on prevalence
- Potential link to post-traumatic stress disorder (PTSD)

mTBI in Soldiers Returning from Iraq

- Survey of 2525 Army soldiers 3-4 mos after return from deployment
  - 4.9% reported injury with LOC
  - 43.9% with PTSD
  - 10.3% injury with altered mental status
  - 27.3% with PTSD


WWII

- Finnish psychiatrist Dr. Harry Federley adopts term LMF (Lacks Moral Fibre) for combat-related neuroses
- German psychiatrist Dr. Rudolph Brickenstein: “...that if a soldier did break down and could not continue fighting, it was a leadership problem, not one for medical personnel or psychiatrists. Breakdown (he said) usually took the form of unwillingness to fight or cowardice.”

PTSD & Post-Traumatic Headache

- Retrospective analysis of 270 soldiers with post-traumatic headache
  - 105 (39%) met criteria for PTSD

Rosenthal JF et al. Headache 2013
Biomechanics of Concussion

…“Brain in a Box”

- Direct injury to brain at point of contact (coup)
- Injury opposite the point of impact (contrecoup)
- Rotational forces with shearing/stretching of axons
- "wash-over" effect (blast injuries)


Neurology of Concussion

- Many symptoms not captured by cognitive testing
  - Photophobia: occipital lobe, brainstem
  - Blurred vision: frontal, temporal lobes
  - Convergence insufficiency / accommodative dysfunction: frontal lobe, brainstem
  - Diplopia and vertigo: brainstem, cerebellar paths

Vision/Oculo-Motor

T1WI 3T scanner

Neuronal Injury Following Concussion
The underlying pathophysiology of concussion appears to be shearing forces causing diffuse axonal injury (DAI).

Douglas Smith, 2010, Scientific American: p 70

Frontal lobe and Temporal lobe

Axons of the Frontal and Temporal lobes are particularly susceptible in Traumatic Brain Injuries—DAI

In the normal
- Ca++ into the neuron
- Glutamate released (neurotransmitters)

Glutamate is principal excitatory neurotransmitter in the brain; And it has to be present in just the right amounts inside and outside the neuron

In the normal neuron...
The concentration of intracellular glutamate is approx 16 times that of extracellular concentration

It's regulated--excess amounts removed—actively transported into glia (glutamate transporters)

Trauma causes DAI and widespread release of glutamate—there is reverse glutamate transport—glutamate builds up in the extracellular space where it is toxic to neurons
Trauma causes widespread release of neurotransmitters especially glutamate—excitotoxicity.

- The excess glutamate overstimulates its receptor (NMDA) on postsynaptic cell
- Too much Calcium flows into the cell—leads to calcium cascade that is toxic to the cell—Cell Death

N-methyl-D-aspartate (NMDA) receptor

Above a certain concentration Calcium is toxic

Also:
- When calcium moves into the neuron
  - K⁺ moves out of neuron
  - Out of glia

Nerve impulse...

While K⁺ is essential to conducting the nerve impulse...

Too much K⁺ causes random firing (depolarization) of neurons

To get back to normal, the neuron has to work extra hard—requires energy!!!

Uses more and more energy (ATP)

“energy crisis”
In addition, the excess Ca++ that moved into the neuron damages mitochondria (the power plants) normally, mitochondria make the energy (ATP).

Repetitive mild Traumatic Brain Injury

- NAA* is also a source of energy for Neuron to perform all its functions
- NAA* is found 100-fold more in neurons than any other tissue

NAA cerebral levels are decreased for weeks after concussion.

With (*H-MRS)

<table>
<thead>
<tr>
<th>Energy</th>
<th>NAA</th>
<th>ATP</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 hrs</td>
<td>↓ 35%</td>
<td>↓ 57%</td>
</tr>
<tr>
<td>15 hrs</td>
<td>↓ 46%</td>
<td>↓ 45%</td>
</tr>
</tbody>
</table>

After first concussion

Longhi et al., Neurosurg: 2005: 56 (2): 364-74

Vagnozzi et al., Neurosurgery 2008: 62
Vagnozzi et al., Brain: 2010: 133; 3232-3234

Mitochondria

- They make NAA too!
- It is also a source of energy for Neuron to perform all its functions
- But there's a decrease in function of mitochondria
- So decreased energy

One more finding re Calcium...

Ca++ is toxic... Above certain levels
When Calcium moves into axon stimulates enzymes (calpain, caspase) which digest brain Spectrin that forms the neuronal cytoskeleton.

Axonal transport

Microtubules are made up of spherical proteins bound together by tau proteins.

Tau proteins stabilize microtubules. They are abundant in neurons of CNS, and not much elsewhere.

Frontal and medial temporal lobes of the brain have been implicated a lot in mTBI literature.

We'll look at Normal functions of these areas-- Show correlation with dysfunction... With tauopathy.

Frontal lobe

Prefrontal cortex 9, 10, 11, 12

Executive function Personality

Frontal and medial temporal lobes of the brain have been implicated a lot in mTBI literature.

We'll look at Normal functions of these areas-- Show correlation with dysfunction... With tauopathy.

Executive Function

- Anatomical substrate of personality
- Seat of the intellect; abstract thought
  - Attention/concentration
  - Planning for future
  - Consequences of behavior

Limbic Lobe
(emotional brain)

- Parahippocampal gyrus
  - hippocampus*
  - amygdala (in uncus)

- hippocampus is ‘in’ Parahippocampal gyrus (memory)

Executive Function

- Anatomical substrate of personality
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Limbic System

- Archicortex—first cortex:
  Preservation of Self and Preservation of Species

- Anatomical substrate of emotion; including mood
- Part of limbic system is important in memory
  - hippocampus
  - Many connections with olfactory cortex

- Many connections with prefrontal cortex
- Part of limbic system is in temporal lobe (temp lobe at risk in TBI)

University of Nevada

cc = corpus callosum
Prefrontal cortex (via axons) impacts the Limbic system. In the normal state, Limbic system (emotion) sometimes overrides Executive function (prefrontal cortex), but the huge impact that the Neocortex (new brain) has on limbic brain, prevails. Therefore frontal lobe lesions may occur.

Damage either/or their axonal connections
- Cognitive impairment, executive dysfunction
- Loss attention, concentration
- Impulsivity
- Depression, mood swings
- Explosivity and aggression
- Memory loss
- Dementia
- Profound short term mem loss, paranoia

Cingulate
Anterior Cingulate cortex can be divided
Dorsal: cognitive function
Prefrontal cortex; motor areas;
FEFs (Frontal Eye Fields)
Ventral: emotion function
Limbic system

Therefore frontal lobe lesions:
- executive dysfunction
- emotional dysfunction
- problems with eye movements

Longitudinal Changes in Brain Volume Following TBI
- Longitudinal, segmentation analysis of TBI patients (one concussion)
- Post hoc analysis @ 1 year
- Atrophy of cingulate gyrus, isthmus of cingulate gyrus and pre-cuneus
- Correlation with neuro-cognitive, behavioral & visual-motor abnormalities

Concussion Signs and Symptoms (May be Delayed Minutes to Days)
- Physical: headache, dizziness, vision changes
- Cognitive: memory impairment, concentration
- Emotional: irritability, sadness
- Sleep disorder


Yongxia Zhou, Andrea Klerans, Damon Kenul, Yulin Ge, Joseph Rath, Joseph Reaume, Robert I.Grossman, and Yvonne W. Lui

Mild Traumatic Brain Injury: Longitudinal Regional Brain Volume Changes

Original Research - Neuroradiology
For checked items
Full Text • Figures Only • Rights and Permissions

Volume Following TBI
- Longitudinal, segmentation analysis of TBI patients (one concussion)
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Complications of Concussion

- Second-impact syndrome (SIS)
- Postconcussion syndrome (PCS)
- Chronic traumatic encephalopathy (CTE)

Second-Impact Syndrome

- Sustaining a second concussion before “recovery” from first concussion
- Majority in pediatric/adolescent populations (< 20 yrs.)
- Enhancement of cerebrovascular congestion (?)
  - Diffuse cerebral edema
  - Death (within minutes to days!)


Postconcussion Syndrome

- 3 months duration of three or more of following:
  - Fatigue
  - Disordered sleep
  - Irritability / aggressiveness
  - Anxiety / depression
  - Personality changes / apathy
  - Impaired attention / memory
  - Impaired eye tracking

Diagnostic and Statistical Manual of Mental Disorders. Fourth Edition
Heitger MH et al. Brain 2009

Crosby progressing slowly; return not set
Tuesday, January 25, 2011
By Dave Molinari, Pittsburgh Post-Gazette

Montclair High School football player Ryne Dougherty died on Oct. 15, 2008, two days after collapsing in a junior varsity game against Don Bosco Prep.
The family of a Montclair High School football player who died two days after collapsing in a 2008 junior varsity game agreed Monday to settle its lawsuit against the school and the township’s Board of Education for $2.8 million, the family’s lawyer said.

Nj.com September 9, 2013

Chronic Traumatic Encephalopathy (CTE)

- Historical evidence of progressive neurologic disability in boxers:
  - “punch drunk” (Martland 1928)
  - “dementia pugilistica” (Millspaugh 1937)
- Signs & symptoms:
  - Memory disturbance, confusion, behavioral/personality changes, Parkinsonism and speech/gait abnormalities
- Average age of diagnosis = 43 yrs.
- Average age of death = 54 yrs.

Neuropathology of CTE

- Atrophy of cerebral hemispheres, temporal lobe, mammillary bodies & brainstem
- Ventricular dilatation
- Fenestration of septum pelucium
- **Marked accumulation of tau-immunoreactive astrocytes**


Mr. Duerson’s Clinical History

- Long-standing complaints of headaches since NFL and onward.
- Over the ~5 years prior to death, he had worsening short-term memory difficulties, as well as problems with language and “vision”
- Increasingly out of control:
  - Short fuse
  - Hot tempered
  - Physically abusive
  - Verbally abusive

Dave Duerson

Dave Duer son
• Co-Captain of 2010 Penn Football Team
• Began playing football at 9 years old
• Committed suicide April 26, 2010, at the age of 21
• No history of concussion
• No history of mental illness
• Mentioned doing poorly in two classes to his parents the day before hanging himself in his off-campus apartment

Owen Thomas

Clinical “Spectrum” of CTE
• Boston University study of 36 male subjects with histopathologically documented CTE
• Retrospective interviews with next-of-kin
  – 61% Behavioral/mood disturbances (younger age)
  – 31% Cognitive impairment (older age)

Owen Thomas

18 y/o male with CTE

Stage 1: headache and loss of attention and concentration
Stage 2: depression, explosivity and short-term memory loss
Stage 3: loss of executive dysfunction and cognitive impairment
Stage 4: dementia, word-finding difficulty and aggression


CTE is a disease of progressive neurologic & psychologic dysfunction with increasing CNS deposition of tau related to repetitive head blows over time.

The Search for Surrogate Biomarkers

- PET scans of retired NFL players reveals FDDNP signals in areas of histopathologic documentation of tau (Small GW, et al. Am J Geriatr Psychiatry 2013)
- OCT abnormalities in veterans with TBI vs. age-matched controls (Kardon R, et al. ARVO 2013)

CTE Masquerades

- Alzheimer disease
- Progressive supranuclear palsy
- Parkinsonism
- Amyotrophic lateral sclerosis (Lou Gehrig’s Disease)

54 Veterans with TBI 25 Age-Matched Normal Veterans

The Search for Surrogate Biomarkers (cont.)

- Retinal deposition of hyperphosphorylated tau (McKee A. personal communication 2012)
- Potential for OCT and other visual tests as surrogate biomarkers of CTE
Why Do We Need a Rapid Sideline Test for Concussion?

• Detecting early signs of concussion may improve outcomes in athletes with mild closed head trauma
• Possible devastating long-term disability
• Following a concussion, you are 3 times more likely to have another one
• Need an easy objective test since qualified personnel not always available


New Rapid Sideline Tests

Concussion Tests: 2 Types

• Testing for diagnosis: King-Devick (K-D) test, Standardized Assessment of Concussion (SAC), SCAT3, MACE
• Testing for management: ImPACT, other computerized testing

Levels of Evidence

Expert Opinion and Clinical Observation are the LOWEST forms of evidence

What is the Evidence?

• Management is largely guided by expert opinion or retrospective data
• Levels of evidence in medicine: 5 levels
  – Highest: prospective study, established criteria
  – Lowest: expert opinion
• Formal research testing is the only way to get evidence that a test works!

Initial Assessment: SCAT3
SCAT3 for Sideline Testing?

- Evaluation of symptoms, orientation, memory & balance
- Takes 20 minutes
- Not all components validated
- Validation of SAC using prior definitions of concussion / only tests cognitive domain
- Requires medical professionals

Concussion Anatomy

- Cerebral cortex, brainstem, cerebellum
- All pathways vulnerable in concussion

Maruta, 2010: J Head Trauma Rehabil
King-Devick Concussion Screening Test

Objective, Remove-From-Play Sideline Assessment
Test of rapid number naming & eye movements

- Speed & error compared to baseline time
  - BEST of two trials to determine Baseline Time
  - Retest after suspected head injury

*INCREASED time or ERRORS → Concussion

*Can be administered by coaches/parents (non-medical professional)
*A tool for athletes for remove-from-play game time decisions

Why Test Eye Movements?
- Vision testing >50% of the brain’s pathways
- Impaired eye movements are a proven indicator of suboptimal brain function
- May capture dysfunction not observed in cognitive testing

King-Devick Test in Scientific Publications

King-Devick Test in Scientific Publications

King-Devick Test in Scientific Publications

King-Devick Test in Scientific Publications

Comparison and utility of King-Devick and ImPACT® composite scores in adolescent concussed patients.

“King-Devick Test as a rapid sideline screening test for concussion that can be effectively administered by non-medically trained laypersons.”

Effectiveness of Concussion Assessment and Management. Systematic Review

King-Devick Test as a concussion screening test administered by sports parents.

“King-Devick Test as a rapid sideline screening test for concussion that can be effectively administered by non-medically trained laypersons.”


The King-Devick test as a concussion screening test administered by sports parents.

“King-Devick Test as a rapid sideline screening test for concussion that can be effectively administered by non-medically trained laypersons.”
Sports-Related Concussions in Youth: Improving the Science, Changing the Culture.

“The King-Devick Test… such tools as well as balance tests may be used either by trained responders as part of an acute sideline or in-field assessment or by health care providers during subsequent clinical evaluation”

In the Clinic: Concussion

“For the oculomotor examination, an additional test that may be used is the King–Devick test, which is a tool that rapidly assesses eye movement and can be used in the office or in the same as to determine alterations in eye movements associated with acute concussion. This has been found to be particularly useful in sports in which head injuries are common. The King–Devick test is a 1-minute test in which the athlete is asked to read single digits displayed on cards; any slowing of time to complete the test, ideally compared with the athlete’s baseline, is suggestive of concussion.”


“How can the SCAT2 be improved?

Future research should consider the efficacy for inclusion of vision tests such as the King Devick Test and clinical reaction time tests. Recent studies suggest that these may be useful additions to the sideline assessment of concussion.

Institute of Medicine (US), National Research Council (US). Washington (DC): National Academies Press (US); 2013


King-Devick Test in Scientific Publications

According to the Research

- High test retest reliability (ICC = 0.97)
- Can screen for witnessed and un-witnessed/un-reported concussions
- Quick, objective sideline test
- Can be administered by non-medical professionals
- Shown to be robust to fatigue
- Able to detect visual and cognitive function in:
  - Hypoxia
  - Parkinson’s Disease
  - Multiple Sclerosis
  - Extreme Sleep Deprivation
  - Learning Disability

Source: Galetta, K et al. Neurology 2011; J Neuro Sci 2011-13; Curr Neurol Neurosci 2012

K-D Test For Witnessed and Un-Witnessed, Un-Reported Concussion

Amateur Rugby League New Zealand Pilot Study:

- Participants: 50 athletes over 12 matches
- Concussions: 5
  - 3 witnessed/un-reported
  - 2 un-witnessed/un-reported (detected on post-game assessment with King-Devick test)

Follow-up Study:

- Participants: 37 athletes over 24 matches
- Concussions: 22
  - 5 witnessed/un-reported
  - 17 un-witnessed/un-reported (detected on post-game assessment with King-Devick test)


Concussion Sideline Assessment: Composite Measure?

- Standardized Assessment of Concussion (SAC)
- Balance Error Scoring System (BESS)
- King-Devick Test

Collegiate Football, Soccer & Lacrosse

Prospective longitudinal study of sideline concussion screening

- 52% of concussions identified using SAC (> 2pt worsening)
- 79% of concussions identified using KD
- 89% of concussions identified combining: KD & SAC
- 100% of concussions screened using a composite of sideline tests: KD test, SAC, BESS

Layperson Test Administrators

- Cohort of amateur boxers
- Laypersons administered pre-fight and post-fight K-D Test
- Masked to head trauma status
- Matches watched by ringside physician and boxing trainer
- Athletes with suspected head trauma received MACE administered by ringside physician
- Athletes with concussion compared to athletes screened using K-D Test
- Masked layperson K-D Testers accurately identified concussed athlete due to worsening in K-D Test compared to baseline (3.2 seconds + increased errors)
- 6 boxers in multiple bouts showed no worsening of K-D times
- Scores were not affected by fatigue


When is it acceptable for an athlete to return to play following a concussion?

Graduated Return to Play Protocol

<table>
<thead>
<tr>
<th>Rehabilitation Stage</th>
<th>Functional Exercise at Each Stage of Rehabilitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) No activity</td>
<td>Complete physical, cognitive rest</td>
</tr>
<tr>
<td>(2) Light aerobic exercise</td>
<td>Walking, swimming, stationary bike</td>
</tr>
<tr>
<td>(3) Sport-specific exercise</td>
<td>Running drills in soccer, skating drills in hockey, etc.</td>
</tr>
<tr>
<td>(4) Noncontact drills</td>
<td>More complex training drills, may start resistance training</td>
</tr>
<tr>
<td>(5) Full-contact practice</td>
<td>With medical clearance, participate in normal training activities</td>
</tr>
<tr>
<td>(6) Return to play</td>
<td>Normal game play</td>
</tr>
</tbody>
</table>

AAN Clinical Practice Reference Sheet for Clinicians, 2011.

Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012

How can the SCAT2* be improved? (Sport Concussion Assessment Tool)

Future research should consider the efficacy for inclusion of vision tests such as the King Devick Test and clinical reaction time tests. Recent studies suggest that these may be useful additions to the sideline assessment of concussion.


*Now SCAT3

Future Research: Visual Structure & Functional Abnormalities with Contact Sports Athletes (VICTORS)

- Study sites:
  - Illinois Eye Institute @ Illinois College of Optometry
  - NYU Langone Medical Center @ NYU School of Medicine
  - Center for the Study of Traumatic Encephalopathy @ BU School of Medicine

VICTORS study (cont.)

• Cross sectional & longitudinal analysis of ocular structure and functional findings among retired NFL players as compared to age-matched norms
• Testing protocol:
  – SD-OCT (RNFL & GCC)
  – Low contrast acuity
  – King Devick

Questions to be Answered

• What are the best tests to diagnose and manage concussions?

• Can we use ocular morphology and visual-motor testing as surrogate biomarkers for CTE?

• In what ways can we lead the effort to reduce effects of concussion?

There’s No Such Thing as a Tough Brain

Mike Haynes, far left  Mike Webster (1952-2002), far right
Antonio, third from left  Mike Webster (1952-2002), far right
Jim Murray, third from right  Mike Webster (1952-2002), far right
Mike Haynes, second from right  Mike Webster (1952-2002), far right
Mike Haynes, far right  Mike Webster (1952-2002), far right

NFL Hall of Fame Class of 1997