Hippocrates
(460-377) BC

- First do no harm.
- No head injury is too trivial to ignore
- Sport is the preserver of health
Concussion: Update 2017

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Neurology Consultants of Kansas
Concussion: Update 2017

1. What is Concussion and why is it important?
2. Our role in concussion
3. CTE/Chronic Traumatic Encephalopathy
4. What’s new?
Concussion Definition

- It is a form of **traumatic brain injury** that occurs when the brain is violently jarred back and forth or rotated inside the skull as a result of a blow to the head or body.
The Human Brain
The Human Brain

- About 4 pounds of tissue
- About 100 billion nerve cells
- Each touching 1000 - 100,000 other neurons
- Over several thousand miles of axons
- Using 100 trillion synaptic connections
- Each firing 10 - 1000 times a second
- Estimated memory Capacity 2.5 Petabytes/2.5 million gigabytes
Wheat Head/Neuron

- Dendrites
- Cell body
- Axon
Wheat Heads/Neurons
Gray-White Interface

Gray Matter

White Matter
Vulnerable Brain Tissues

- Gray Matter
  - The cellular layer where the majority of electrical impulses are generated
  - This contains memory, cerebral processing and integration, motor initiation, etc.
Vulnerable Brain Tissues

- White Matter

  Neurons are connected by axons (long projections of nerve cells resembling insulated wiring) which connect neurons to other neurons.
Anatomy of Concussion

- The cervical spine allows the head to rotate to avoid blunt trauma.
- Rotational forces can be the most damaging forces during injury.
Inertial Loading
Primary Mechanism of Concussion

Rotational Acceleration and deceleration
Axonal Strain/Shear
Neurometabolic Cascade Following mTBI

- Axonal Injury-strain/shear
  - Membrane disruption
  - Calcium influx
  - Neurofilament compaction
  - Microtubule disassembly
  - Axon swelling and possible cell death
Neurometabolic Cascade Following mTBI

- Cell Body
  - Depolarization
  - Release excitatory neurotransmitters - glutamate
  - Inc. membrane pumping
  - Hyperglycolysis to inc. ATP
  - Lactate accumulation
  - Calcium sequestration
  - Mitochondrial dysfunction
  - Dec. ATP production
  - Enzyme activation
  - Apoptosis
Micro-anatomy of Concussion

- Shear / Strain Injury
  This occurs when differing densities/tissue planes are put through linear and rotational acceleration and deceleration, leading to plane-shifts.
At Time Of Impact

Normal function
T-1

Massive depolarization
T=0
Concussion -
Massive Unsynchronized Depolarization
Immediate Signs of Concussion

- Appears dazed or stunned
- Is confused about assignments
- Forgets plays
- Is unsure of game, score, or opponent
- Moves clumsily or has slurred speech
Immediate Signs of Concussion

- Answers questions slowly
- Loses consciousness
- Shows behavior or personality changes
- Any amnesia
By definition a CT/MRI scans are within normal limits.
The Problem

- Motor function
- Sensory Function
- Memory
- Concentration
Biggest Fear

Second Impact Syndrome
Second Impact Syndrome

- Second concussion occurs while still symptomatic/healing from previous head injury
- Loss of consciousness is not required
- Second impact more likely to cause brain swelling and other widespread damage
- Possibly secondary to damaged autoregulation
- Can be fatal - 50% mortality in severe cases, 100% morbidity
- High risk of long term cognitive impairment
Will You Recognize Concussion?
Will You Recognize Concussion In Real Time?
Kansas Sports Concussion Partnership

www.KansasConcussion.org
KSCP Pocket Card

Concussion Recognition >

SIGNS OBSERVED BY OTHERS
• Appears dazed or stunned
• Is confused about assignment
• Forgets plays
• Is unsure of game, score, or opponent
• Moves clumsily or has slurred speech
• Answers questions slowly
• Loses consciousness
• Shows behavior or personality changes
• Cannot recall events prior to hit
• Cannot recall events after hit

SYMPTOMS REPORTED BY YOU
• Headache
• Nausea or vomiting
• Balance problems or dizziness
• Double or fuzzy vision
• Sensitivity to light or noise
• Feeling sluggish or drowsy
• Feeling foggy or groggy
• Concentration or memory problems
• Confusion

Concussion Quick Facts >

1. ALWAYS remove athletes immediately after suspecting a concussion. Do NOT allow return to play the same day with a concussion.
2. Athletes do NOT have to be knocked out to have a concussion. 90% of concussions occur without a loss of consciousness.
3. CT scans don’t diagnose concussions. Everyone with a concussion has a normal CT scan.
4. It is OK to let someone fall asleep after being hit in the head. With careful monitoring, rest and sleep will be helpful.
5. “Warm up for Return” is a graded process that requires a minimum of five days.
6. 9 out of 10 athletes will be back to normal within two weeks. They may miss a few games.
7. Kansas law requires a physician’s signature (MD/DO) to “Return to Play.”
8. Athletes who return to full contact too early risk Second Impact Syndrome, a rare but devastating brain injury that may result in death.
9. Concussions can affect driving, school work, sleep, emotions, relationships and self worth.
10. The “game plan” is not just about returning an athlete to their sport; it is about returning the person back to their life.
It’s the Law!

Getting the athlete back in the game

A flowchart for the evaluation, recognition, and management of sports-related head injuries:

1. **Signs & Symptoms of Concussion?**
   - Yes: **Remove athlete from play**
   - No: **Signs & Symptoms of Concussion?**

2. **Serious Concerns?**
   - Yes: **ER/CT Head**
   - No: **Start SCORE Card**

3. **Start SCORE Card**
   - **Give Athlete Concussion Guides and SCORE Card 1**
   - **Athlete’s Symptoms**
     - **SCORE Card 1**
     - Administered by medical professional, coach, trainer, or designated school official. Athlete takes test, and score is reviewed by medical examiner.
   - **Medical Assessment**
     - **SCORE Card 2**
     - Administered by physician or medical professional. Athlete may access to the athlete’s medical records.
   - Repeat both assessments until no symptoms and normal.

4. **Do active symptoms/problems continue?**
   - Yes: **Consider referral to concussion specialist**
   - No: **Start Warm Up for Return**

5. **Start Warm Up for Return**
   - **SCORE Card 3**
   - If any symptoms occur, stop and rest for the day. Repeat step once symptom-free.
   - **STEP 1 Increase intensity**
   - **STEP 2 Add intensity**
   - **STEP 3 Add coordination and cognitive load**
   - **STEP 4 Restore confidence and assess functional skills**

6. **Warm up completed. Symptom free?**
   - No: **Consider referral to concussion specialist**
   - Yes: **Release for Competition & Practice**

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**Kansas Sports Concussion Partnership**

www.KansasConcussion.org
## Score Card - Page 1

### Athlete's Symptoms Score Card

**Name:**

**Date of Birth:**

**Date of Injury:**

**Sport:**

**Position:**

**School:**

**Primary Care Physician:**

**Parent/Guardian notified:**

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### Symptom Evaluation

**How do you feel now?** Score the symptoms:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>1</th>
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<td>3. Neck pain</td>
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<td>5. Dizziness</td>
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<td>7. Balance problems</td>
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<td>10. Fatigue or low energy</td>
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<td>11. Don’t feel right</td>
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<td>12. Feeling slowed down</td>
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<td>13. Feeling like in a fog</td>
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<td>14. Difficulty concentrating</td>
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<td>15. Difficulty remembering</td>
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<td>16. Confusion</td>
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<td>17. More emotional</td>
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<td>19. Sadness</td>
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<td>20. Nervous or anxious</td>
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<td>21. Difficulty falling asleep</td>
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</table>

**For Office Use Only**

- **Total # of Symptoms:** 22 max.
- **Symptom Severity Score:** 132 max.
- **Total # of Balance Errors:** 36 max.
- **Total Cognition Score:** 24 max.

This athlete is symptom free and has normal exams. I authorize him/her to start “Warm Up for Return.”

**Name:**

**Date:**

**Signature:**

Go to Score Card 3 to start your “Warm Up for Return.”
# Medical Assessment Score Card

**Date of Birth**

**Date of Injury**

**Sport**

**Position**

**School**

**Primary Care Physician**

A project sponsored by Kansas Medical Society

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## Cognition/Balance Assessment

### Immediate Memory

- **Exam 1:** 4-6-7
- **Exam 2:** 6-5-6
- **Exam 3:** 5-4-3
- **Exam 4:** 5-7-5
- **Exam 5:** 7-3-1

Total score for all 5 trials. 15 points possible.

### Concentration

- **Trial I:** 6-3-2-1, 6-2-1-5, 3-5-2-6
- **Trial II:** 3-4-5-6, 1-3-2-4, 6-5-1-2
- **Trial III:** 4-2-1-3, 3-4-2-1, 4-1-2-3
- **Trial IV:** 3-4-2-5, 4-1-2-3, 1-3-4-5

Score 1 pt. if entire sequence is correct. 4 pts. possible.

### Balance Errors

- **I. Double Leg Stance:** Stand together with hands on hips and eyes closed. Should remain stable for 30 seconds. Count number of times that person moves out of that position.
- **II. Single Leg Stance:** Stand on non-dominant foot (opposite dominance by asking). Which foot would you use to kick a ball? The dominant leg should be held off the floor a few inches and maintain stability for 30 seconds with hands on hips and eyes closed. Count number of times that person moves out of their position. If person stumbles out of this position, have them open eyes and return to the start position and continue balancing. Start timing when they are set and have their eyes closed.
- **III. tandem Stance:** Stand heel-to-toe with the non-dominant foot in back. Weight should be evenly distributed across both feet, should maintain stability for 30 seconds with hands on hips and eyes closed. Count number of times that person moves out of that position. If athlete stumbles out of this position, have them open eyes and return to the start position and continue balancing. Start timing when they are set and have their eyes closed.

Beginning counting errors only after the athlete has assumed the proper start position.

- **Stance I:** 4 or more errors. 20 points possible.
- **Stance II:** 2 or more errors. 10 points possible.
- **Stance III:** no errors. 10 points possible.

Total # of Balance Errors (30 max.)

## Delayed Recall

- **Exam 1:** Score one point for each word remembered (5 points possible).

## Total Cognition Score

Add the three individual (+1) scores. Transfer total score to athlete's SCORE Card. (24 total points possible)
## Symptoms Of Concussion

<table>
<thead>
<tr>
<th>Physical</th>
<th>Cognitive</th>
<th>Emotional</th>
<th>Sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA Photo/Phonophobia</td>
<td>Concentration</td>
<td>Irritability</td>
<td>Hypersomnia</td>
</tr>
<tr>
<td>Nausea/Vomiting</td>
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</tr>
<tr>
<td>Dizziness</td>
<td>Memory</td>
<td>Depression</td>
<td>Insomnia</td>
</tr>
<tr>
<td>Lightheadedness</td>
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<tr>
<td>Balance problems</td>
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<tr>
<td>Blurry Vision</td>
<td>Feeling slowed</td>
<td>Mood lability</td>
<td>Trouble falling</td>
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<tr>
<td></td>
<td>down</td>
<td></td>
<td>asleep</td>
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<tr>
<td>Loss of energy</td>
<td>Inability to</td>
<td>Anxiety</td>
<td>Trouble staying</td>
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<td></td>
<td>multitask</td>
<td></td>
<td>asleep</td>
</tr>
</tbody>
</table>
## Multidisciplinary Symptoms
Requires Multidisciplinary Treatment

<table>
<thead>
<tr>
<th>Most common symptoms</th>
<th>Treatment options</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA</td>
<td>Rx based on phenotype</td>
</tr>
<tr>
<td>Neck pain</td>
<td>Physical therapy</td>
</tr>
<tr>
<td>Autonomic</td>
<td>Start with non-pharmacologic Rx</td>
</tr>
<tr>
<td>Balance</td>
<td>Vestibular therapy</td>
</tr>
<tr>
<td>Oculomotor</td>
<td>Vision therapy</td>
</tr>
<tr>
<td>Cognitive</td>
<td>Cognitive rehab</td>
</tr>
<tr>
<td>Emotional</td>
<td>Biofeedback/CBT/therapy</td>
</tr>
<tr>
<td>Insomnia</td>
<td>Biofeedback/Melatonin</td>
</tr>
</tbody>
</table>
Return-To-Activity

- Largely symptom free
- Exertional protocol
- Return-to-learn goals met
- Back to prior baseline testing scores
- Sport specific return-to-play protocol
Risk Factors For Prolonged Recovery

- Pre-injury HA history
- Developmental history and learning disabilities
- Psychiatric history
- Concussion history
  - Number of prior concussions
  - Symptom duration
  - History of reduced threshold for concussion
Return-to-Play

- **Day 1** - Light aerobic exercise. No weight lifting.
- **Day 2** - Running /swimming. No helmet or equipment.
- **Day 3** - No contact drills in full uniform, light weights.
- **Day 4** - Participate in full-contact practice/training.
- **Day 5** – Return to competition or game play
Athlete’s Concussion Guide

ATHLETE’S CONCUSSION GUIDE

Definition >
A concussion is the most common form of head injury suffered by athletes. It is a form of traumatic brain injury that occurs when the brain is violently jarred back and forth or rotated inside the skull as a result of a blow to the head or body. This can "stun" the brain cells or even result in their death. You do not have to lose consciousness to suffer a concussion.

Any athlete in motion is at risk for a concussion. This may occur in any sport to boys and girls alike. Symptoms may appear immediately or develop over several days. They may last a few days to several months and interfere with schoolwork and social life.

Management >
How to know if you have a concussion?
If you have any of the signs or symptoms listed under the "Recognition" section of this guide, you may have a concussion. Do not play through a concussion because it’s not worth the risk to your health and your life.

What do I do if I think I might have a concussion?
Stop playing right away! Don’t game a bump. Move or fall to your head. Talk a nerve, coach or athlete_info about your symptoms. You should be immediately removed from practice or games to avoid further injury. If symptoms develop at home or school, immediately tell a parent/guardian, teacher or coach.

What happens when I report a possible concussion?
An athletic trainer, coach, FFT, paramedical or physician will evaluate you as soon as you report your symptoms. If there are no complications and you are cleared to return, you can return to the game or practice as soon as possible. If you do not have a concussion, you will not be able to play your sport until cleared by a physician.

What do I need to do after I am allowed to return to play?

Concerns >
What are possible complications from a concussion?
While 5% of athletes are back to their baseline within 7-10 days, there are 10% of athletes who will experience prolonged symptoms or other complications.

This is called Post-Concussive Syndrome and occurs when symptoms from a concussion are prolonged. Difficulty with concentration, memory, and postural instability can be common symptoms. Talk with the physician if symptoms last longer than a couple of weeks. A referral to a specialist for further evaluation and treatment may be required.

What might happen if the athlete returns to play before symptoms are gone?
If an athlete returns to play before symptoms have resolved, the risk of returning to a second concussion, which could develop Chronic Traumatic Encephalopathy (CTE). This syndrome can cause various brain abnormalities or other health problems even after the athlete’s retirement.

Better to miss a game or two than the whole season or your whole life!

Other Resources >
Kansas City: www.kscsp.org

KSHPA Guidelines: www.kshpa.org

Concussion Care: www.concussioncare.org

KCHA Guidelines: www.kcha.org

Centers for Disease Control and Prevention: www.cdc.gov/concussion/headsUp/youth.html

Kansas Sports Concussion Partnership: www.kansassconcussion.org

Athlete’s CEI © 2011 Page 1

KSCP
Kansas Sports Concussion Partnership

Quick Facts >
1. ALWAYS remove athlete immediately after suspecting a concussion. Do NOT allow return to play the same day with a concussion.
2. Athletes do NOT have to be removed if they were involved in a traffic collision but have a normal CT scan.
3. CT scans don’t diagnose concussions. Everyone with a concussion should have a normal CT scan.
4. It is OK to let someone fall asleep after being hit in the head, with normal cognitive monitoring, rest and sleep will repair.
5. "Warm up for Return" is a gradual process that requires a minimum of five days.
6. 0 out of 10 athletes will be back to normal within two weeks. They may take a few years.
7. Kansas law requires a physician’s signature (MD/DO) to "Return to Play.”
8. Athletes who return to full contact too early risk Second Impact Syndrome, a rare but devastating brain injury that may result in death.
9. Concussions can affect driving, schoolwork, sleep, emotions, relationships and self worth.
10. The “game over” is not just about returning an athlete to their sport; it is about returning the person back to their life.

Recognition >
Recognition is based on the following:

• Appearance
• Nausea or vomiting
• Balance problems or dizziness
• Dizziness
• Sensitivity to light or noise
• Feeling dizzy or off balance
• Concentration or memory problems
• Confusion

SCORING Card >
This first aid care professional that evaluates you will fill out a concussion evaluation tool called a SCORING Card. If this first person is not your physician or you will not during recovery, make sure you get a copy of SCORING Card to take to your physician appointment. This will contain important information that your physician will use to monitor your progression and return you to the game as soon as possible.

Return to Play >
When can I start playing sport again?
Returning to your sport is a step by step process. Once you have no symptoms or signs of concussion and have achieved normal results on SCORING Card & a medical physician will start your Return to Play. This process should be monitored by an athletic trainer or designated school official.

One you have completed the progression back to play, you will be authorized to start the "Warm up for Return." At any time that you do not feel quite right, tell your doctor, parent, coach or athletic trainer immediately.

After I am symptom free, what is the progression back to play?
This is a progression as a warm up for return to your sport. Taking at least 24 hours for each step before moving on to the next step is recommended.

If any symptoms recur, report this right away and stop for the day. You must be completely symptom free before starting the progression again.

Step 1: Light aerobic exercise. Carrying walking or riding an exercise bike. No weight training.

Step 2: Running a gun or on the field. No helmet or equipment should be used.

Step 3: Non-contact training skills and full equipment. Light resistance training or light weight training.

Step 4: Fall contact training under supervision of certified athletic trainer.

Step 5: Return to competition or game play.

You must be seen by a physician (MD/DO) during the warm up progression to be legally released for return to competition or practice.

Concerns >
What are possible complications from a concussion?
While 5% of athletes are back to their baseline within 7-10 days, there are 10% of athletes who will experience prolonged symptoms or other complications.

This is called Post-Concussive Syndrome and occurs when symptoms from a concussion are prolonged. Difficulty with concentration, memory, and postural instability can be common symptoms. Talk with the physician if symptoms last longer than a couple of weeks. A referral to a specialist for further evaluation and treatment may be required.

What might happen if the athlete returns to play before symptoms are gone?
If an athlete returns another time after still recovering from a first concussion, he/she could develop Chronic Traumatic Encephalopathy (CTE). This syndrome can cause various brain abnormalities or other health problems even after the athlete’s retirement.

Better to miss a game or two than the whole season or your whole life!
“It is suspected that repeated blows to the head can play a role in the formation of lesions or injuries in the long-term. In other words, it is more harmful to sustain regular blows to the head which would, on their own, go unnoticed, then to sustain the occasional, acute and symptomatic injury. It is therefore essential to avoid training sessions concentrated purely on heading the ball as the risks are compounded-as opposed to in a match.”

1998
Effects of Recurrent Concussion

- Increased number of symptoms
- More severe symptoms
- Greater risk of injury
- Longer recovery
- Slower return to activity
- Greater cumulative impairment
Definition of Concussion

Clinical Syndrome
-AAN 1997

Injury
-CISG 2012
Delayed Side Effects

- Depression
- MCI / Mild Cognitive Impairment
- Dementia
- CRSD/Circadian Rhythm Sleep Disorder
- CTE/Chronic Traumatic Encephalopathy
Punch Drunk
Dementia Pugilistica
Chronic Traumatic Encephalopathy
“Concussion”
Dec 25, 2015
Dr. Ann McKee MD
Neuropathologist
Boston University School of Medicine

- Dx with CTE by March 2016
  - NFL
    - 90 of 94 or 96%
  - College
    - 45 of 55 or 82%
  - High School
    - 6 of 26 or 23%
Chronic Traumatic Encephalopathy/CTE Neuropathology

- Gross pathology
  - Cerebral atrophy
  - Enlarged lateral and third ventricles
  - Cavum septum pellucidum with fenestrations
  - Thinning corpus callosum
  - Atrophy of diencephalon and mammillary bodies
  - Depigmentation of the locus ceruleus and substantia nigra
Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

Jama.2017;318(4):360-370
July 25, 2017

MACROSCOPIC pathology of a football player: The top image is a normal brain. Severe stage IV CTE is shown, as evidenced by cavum septum pellucidum (1); severe cerebral atrophy (2); dilated lateral ventricles (3); and dilated third ventricle (4).
Microscopic Pathology
- Deposition of hyperphosphorylated tau (p-tau)
  - NFT’s neurofibrillary tangles (NFT’s)
  - Thorned astrocytes (TA)
  - Dotlike neurites
- Perivascular location
- Predilection for depths of the sulci
CTE
Neuropathology
CTE
Neuropathology
Dr. Bienick/Mayo Clinic

- Brain bank with >7,000 brains
- Narrowed it down to those who played amateur contact sports
- Settled on 66 who played primarily HS and college football, but others who were active in boxing, rugby, wrestling, basketball and soccer
- 198 control brains
They found CTE in 21 of 66 – 32% or almost 1/3

No CTE was found in the 198 controls
CTE
Chronic Traumatic Encephalopathy

- It’s neuropathology is unique
- Primary driver is exposure to repetitive impacts
- More common than recently appreciated
- Once initiated, it is progressive
CTE
Chronic Traumatic Encephalopathy

- It’s neuropathology is unique
- Primary driver is exposure to repetitive impacts
- More common than recently appreciated
- **Once initiated, it is progressive!**
“Tauopathies”

• Frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17)
• Alzheimer's disease
• Aging
• Progressive supranuclear palsy
• Pick's disease
• Argyrophilic grain dementia
• Corticobasal degeneration
• Progressive subcortical gliosis
• Amyotrophic lateral sclerosis/parkinsonism-dementia complex
• Diffuse neurofibrillary tangles with calcification
• Dementia pugilistica (Chronic traumatic encephalopathy)
• Tangle-only dementia
• Down syndrome
• Gerstmann-Strausssler-Scheinker disease
• Hallervorden-Spatz disease
• Creutzfeldt-Jakob disease
• Multiple system atrophy
• Niemann-Pick disease type C
• Prion protein cerebral amyloid angiopathy
• Subacute sclerosing panencephalitis
• Myotonic dystrophy
• Non-guamanian motor neuron disease with neurofibrillary tangles
• Postencephalitic parkinsonism
• Meningioangiomatosis
• Tuberous Sclerosis
Burden of Disease
Burden of Disease
Burden of Disease
Burden of Disease
Burden of Disease
Burden of Disease
CTE
Chronic Traumatic Encephalopathy

- Develops insidiously years after trauma
- Progresses slowly over decades
- 20% of athletes are still active in a sport
- Average onset 14 yrs after retirement
- Mean duration of course is 15 yrs
- Age of clinical symptom onset 19-83 yrs
Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

Jama.2017;318(4):360-370
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HEMISPHERIC brain sections showing tau pathology in a normal brain, mild CTE, and severe CTE. Bottom row: Microscopic images of tau immunostained paraffin-embedded sections (bottom; tau neurofibrillary tangles appear dark red).
CTE
Chronic Traumatic Encephalopathy

- Onset begin in one or more distinct domains
  - Mood - depression
  - Behavior - explosivity, verbal and physical violence, loss of control, impulsivity, paranoia, and rage
  - Cognitive - Memory, executive functioning, impaired attention
  - Motor - dysarthria, dysphagia, coordination problem, parkinsonism
Tiaina Baul Seau Jr.

- 2 Super Bowls
- 12 Pro Bowls
- No reported concussions
- Suicide at age 43
- Diagnosis CTE
fMRI
During A Visual Memory Test

TELLTALE A sequence of fMRIs from a “functionally impaired” player showed that brain activity (orange) declined noticeably, especially in the frontal lobe, then rebounded after the hitting stopped.
fMRI
During A Visual Memory Test
fMRI During A Visual Memory Test

Pre-Season | Season #1 | Season #2
Concussion Is Not The Scariest Part?

- We have 100 billion nerve cells
- 0.1% would be 100 million cells
  - Let’s say that is the average threshold of injury to identify concussion on the sideline
Concussion Is Not The Scariest Part!

What would you call an athlete with only 70,000,000 of their cells knocked off line?

“NORMAL”

Cleared to return to game
fMRI
During A Visual Memory Test

Pre-Season  |  Season #1  |  Season #2
New Terms In The Literature

- “Subconcussive” Head Impacts
- “Subconcussive” Head Trauma
Biomarkers

- Neuroprotein S-100
  - Astroglia/shwann cells
- Neuro specific enolase (NSE)
  - Neuronal cytoplasm
- Tau - microtubular binding protein
- SBDP-145/SPDP 120
  - Axons
- UCHL-1
  - Cytoplasm
- MAP-2
  - Dendrites
- NF-L
  - Axons
Diffusion Tensor Imaging
DTI
Diffusion Tensor Imaging

Control

Boxer
Susceptibility-Weighted MRI

SWMRI

Figure 4.
Susceptibility-weighted image (SWI) example. Comparison of (a) T2-weighted, (b) SWI filtered phase, (c) processed magnitude, and (d) maximum intensity projection images on patient with traumatic brain injury, acquired on 3 T TRIO Siemens system. SWI has the following acquisition parameters: echo time/repetition time (TR/TE): 29/20 ms, flip angle: 15˚, bandwidth: 120 Hz/pixel, 8-channel phased array coil with a parallel imaging factor of two, field of view (FOV): 256 × 256 mm², slice thickness: 2 mm, acquisition matrix: 512 × 416 × 64, spatial resolution: 0.5 × 0.5 × 2 mm³. T2-weighted image acquired with T2 fast spin echo with TR/TE: 5000/113 ms, FOV: 256 × 256 mm², slice thickness: 2 mm, acquisition matrix: 320 × 320. Red arrows label multiple possible microhemorrhages invisible on both T1- and T2-weighted images (some not labeled). In this case, SWI data clearly demonstrate multiple possible microhemorrhages in brain.
Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

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July 25, 2017

Findings –

- In a convenience sample of 202 deceased players of American football from a brain donation program
- CTE was diagnosed in 177 players across all levels of play (87%)
CTE diagnosis break down

0 of 2 pre-high school – 0%
3 of 14 high school - 21%
48 of 53 college – 91%
9 of 14 semi-professional – 64%
7 of 8 CFL – 88%
110 of 111 NFL – 99%
CTE severity
Jama.2017;318(4):360-370
July 25, 2017

- All 3 High school pts had mild severity
- College - 56% severe
- Semiprofessional – 56% severe
- Professional – 86 % severe
Clinically what does that mean?

27 players with mild severity
- 96% had behavior or mood symptoms or both
- 85% had cognitive symptoms
- 33% had signs of dementia
Clinically what does that mean?

84 participants with severe CTE
- 89% had behavior or mood symptoms or both
- 95% had cognitive symptoms
- 85% had signs of dementia
Of the 111 CTE cases with standardized informant reports:
- 85% of mild cases showed progressive clinical course
- 100% of severe cases showed progressive clinical course
Further Questions that may influence outcomes

- Age of first exposure
- Duration of play
- Player position
- Cumulative hits
- Linear /rotational acceleration of hits
Age of first exposure to football and later-life cognitive impairment in former NFL players

Julie M. Stamm et al.
Age of First exposure to Football...
Julie M. Stamm et al. 2015

- 42 NFL players
- Divided by exposure above or below age 12
- Controlling for
  - Total years played, age at eval, estimated IQ, memory impairment and executive function
- WCST, NAB-LL, WRAT-4

Players in the <12 age group performed significantly worse on all measures
214 American football players
- 43 played only through high school
- 103 played only through college
- 68 played in NFL

Telephone administered testing
- Depression, behavior regulation, apathy, executive functioning

Results from former players who started before and after age 12 were compared
Participation in Youth football before age 12
- Increased problems with behavior regulation, apathy and executive functioning by **two fold**
- Increased depression by **three fold**

Results independent of
- Years played, total concussions, or how far you went playing football
# of active Football players

- **NFL**: 1,800
- **College**: 54,250
- **High School**: 1,140,000
- **Youth Football**: >10,000,000
Pitt State Gorilla
Zack Langston
Pitt State Gorilla
Zack Langston

- Suffered from
  - behavior and mood swings
  - Memory loss
  - Anxiety
  - Impaired judgement
  - Depression
  - Impulse control problems
  - paranoia
Zack Langston

- Took his own life  
  Feb 2, 2014

- June 2, 2017  
  Langston Family sues NCAA and MIAA

- As of Oct. 2016 there are 43 NCAA class-action law suits regarding handling of concussions
Wisconsin Longitudinal Study in 1957

- 10,317 HS Male graduates
- 2,692 had data on football participation and still in study in 2017
  - 834 played football
  - 1858 did not
Association of Playing High School Football With Cognition and Mental Health Later in Life
JAMA Neurol. 2017;74(8):90-918

- **Outcome** -
  - No difference in Cognition
  - No difference in Depression
Editorial
Reassuring News About Football and Cognitive Decline? – Not So fast
JAMA August 2017

Limitations

- No data on years played, position played, or level of play
- No concussion/injury history
- Fairly simple measurement tools
- Outcomes measured at single time points
There Is Still Much We Do Not Know

- The true prevalence or incidence of CTE
- How much room between mTBI/concussion and incurable degenerative disease?
  - Emotional issues, depression, anxiety, PTSD, etc.
- Risk factors for CTE
  - ApoE-4 Allele
  - Other genetic risks?
VICIS Zero1 Helmet
Q-Collar
by Q30 Innovations
Is This Ridiculous?
Is This Ridiculous?
Knowledge Transfer (KT)

Brandi Chastain vows to donate her brain to Boston University for research.
Our Children—Our Future
Knowledge Transfer (KT)

“When in doubt, Sit them out”

www.KansasConcussion.org