The Role of the Headache Specialist in the Management of Sports Concussion

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Introduction

• Challenges in Headache Medicine.
• Treatment strategies for medication overuse headache.
• Neurotoxins: A review of the evidence.
• Hormones and Headaches.
• Sports Concussion.
• Headache – Basic Science.
Sports Concussion Facts for the Headache Specialist

- Headache far and away the most common symptom.

- Most concussion symptoms are short lived and do not require treatment.

- Sports Concussion are usually managed by PCP’s

- 27 States have passed laws

- Headache is the most common reason for referral to a specialist.
Sports Concussion Epidemiology

- Estimated 1.6 to 3.8 million Sports Related Concussions Annually in the US.
- Each year 60K High School Athletes sustain concussions.
- Most common cause of head trauma in children under 18, and the second most common cause in adults 18-65.
- What sport has the highest reported concussion rate???
Sports Concussion Epidemiology

- At the high school level Football has the highest incidence among males and Soccer among females.

- 60% of all high school football and 40% of all female soccer players.

- In all amateur sports Ice Hockey has the highest incidence.

- 4% of all college football players are estimated to have suffered a concussion during their career.

- 5 out of every 7 deaths from football related injuries are deemed to be secondary to head trauma.
Definition

- At present there is no uniform accepted definition of sports related concussion.
- Lack of a uniform definition creates significant problems when attempting to perform research studies.
- Sports Related Concussion and Mild Traumatic Brain injury (MTBI) are often used synonymously.
- The consensus statement from the Zurich 2008 summit suggested there was a difference between concussion and mTBI.
Definition

- **AAN Definition**: “A trauma-induced alteration in mental status that may or not involve a loss of consciousness. Confusion and amnesia are the hallmarks of concussion. The confusional episode may occur immediately after the blow to the head, or several minutes later.”

- **Zurich 2008 Definition**: “A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.”

- **Conidi Definition**: “a prolonged transient alteration in neuronal function caused by a blow to the athletes head, and/or body with transmission of force to the head, with rotational and/or translational (i.e. angular and lateral) movement of the head resulting in neurological symptoms that resolve sequentially over time.”
Immediate Signs and Symptoms

- Vacant stare (befuddled facial expression)
- Delayed verbal and motor responses (slow to answer questions follow instructions)
- Confusion and inability to focus attention.
- Disorientation (walking in the wrong direction, unaware of time, date and place)
- Slurred or incoherent speech.

When in doubt...sit out!!
Immediate Signs and Symptoms

- Headache
- Gross observable decrease in coordination (stumbling, trouble with tandem gait).
- Emotions out of proportion to circumstances
- Memory deficits (exhibited by the athlete repeatedly asking the same question that has already been answered, or inability to memorize and recall 3 of 3 words or 3 of 3 objects in 5 minutes)
- Any period of loss of consciousness
Intermediate Signs and Symptoms

- Headache
- Dizziness, loss of equilibrium or vertigo
- Lack of awareness of surroundings
- Nausea or vomiting
Late Signs and Symptoms

- Persistent low grade and sometimes even severe headache, (may include photo/phono phobia).
- Light-headedness.
- Poor attention, concentration and memory.
- Heat Intolerance.
- Easy fatigability Sleep Disturbances.
- Irritability and low frustration tolerance.
- Anxiety and/or depressed mood.
- *Sleep disturbance.
- *Decreased reaction time and balance.
Mechanism

- Several theories none universally accepted.

- Rotational, Angular, and/or Lateral forces cause rotation of the cerebral hemispheres around the upper brainstem.

- A direct blow to the head is not required.
Pathophysiology

- Understanding the pathophysiology is essential in developing evidence based RTP guidelines.

- Based mainly on rat percussion and some fMRI studies.

- Sports concussion pathophysiology and migraine pathophysiology share many common denominators.

- Post concussive deficits based on temporary neuronal dysfunction, not cell death.

- Three phases; acute, intermediate and late.
Disruption of neural membranes and axonal stretching result in abrupt and indiscriminant release of neurotransmitters and unchecked ion fluxes.

Excitatory neurotransmitters; NMDA and Glutamate trigger neuronal depolarization with K+ efflux and Ca++ influx.

Increased intracellular calcium triggers further neuronal depolarization and further release of excitatory neurotransmitters and still further release of K+ into the extracellular space.
Acute Phase

- Excitation is followed by a wave of neuronal suppression (i.e. Spreading Depression).
- Activation of membrane pumps in an effort to restore homeostasis.
- Results in increased glucose utilization.
- Increased glycolysis leads to increased lactate production.
- Increased lactate results in neuronal dysfunction via metabolic acidosis, membrane damage, alterations in blood brain barrier permeability, and cerebral edema.
Intermediate Phase

- Uncoupling of glucose metabolism and cerebral blood flow.
- Calcium influx, Mitochondrial Dysfunction and Delayed Glucose Hypo-metabolism.
- Calcium accumulation can persist for 2-4 days.
- Excess calcium is sequestered in mitochondria resulting in impaired metabolism and energy failure.
Pathophysiology

- Cerebral glucose use is diminished by 24 hours and can last up to 2-4 weeks post injury (average recovery 10 days).

- Intracellular Magnesium levels are immediately reduced and remain for up to 4 days.

- Axonal stretching results in membrane disruption and axonal swelling.
Late Phase

- Delayed Cell Death, Persistent Calcium Accumulation and Neurotransmitter Alterations.

- Elevated intracellular calcium can lead to over activation of enzymes and free radical production resulting in cell death.

- Post concussion alterations in NMDA, adrenergic, cholinergic, and GABAergic neurotransmission can result in long term deficits in memory and cognition, even in the setting of minimal anatomic damage.
Pathophysiology

- In the first 30 minutes when the system is stretched to its max., the brain may be unable to respond to a second stimulus induced increase in cerebral glucose metabolism.

- Increased intracellular calcium after a second physiological stimulus can lead to protease activation and programmed cell death.

- Alterations in NMDA receptor composition can persist for one week post injury and a second injury in this period can lead to further impairment of excitatory neurotransmission.
Physiology

• Concussion takes on average 10 to 14 days to recover and some studies suggest 2-4 weeks.

• Repeat Concussions during the post injury period, when the cell is most vulnerable can have catastrophic consequences.

• In rare cases the second impact syndrome which triggers an uncontrolled metabolic process resulting in uncontrolled brain swelling and in most cases death.

• What would have happened if a second hurricane hit New Orleans just after Katrina?
Diagnostic Studies

- Most professional, college and some high school sports teams use standardized sideline evaluation (SCAT-2).

- A number of professional organizations use computerized pre and post concussion testing.

- Balance testing (BESS & Plate) in specialized sports concussion clinics.

- Formal neuropsychological testing for prolonged cognitive symptoms.

- Neuroimaging (DTI, fMRI, MRSpect).

- Electrophysiologic (Q-EEG) and Biomarkers.
Management of Associated Symptoms

- Most concussions are managed by primary care physicians i.e. FP and Peds.

- Most symptoms are short lived, do not require treatment, headache is the exception.

- The neurologist will commonly encounter patients with prolonged symptoms.

- These patients require more involved treatment, which may include additional education, academic and work related accommodations, physical/vestibular therapy, cognitive rehabilitation, psychotherapy and pharmacological treatment.
Management of Associated Symptoms

- Psychological management usually involves monitoring and treating depression (unrecognized)

- Pharmacological management; treating episodic and prolonged symptoms, shortening the duration of symptoms.

- Prolonged symptoms require a multidisciplinary approach.

- Shortening the duration of symptoms can have catastrophic effects as medications can mask unresolved concussion symptoms.
Pharmacological Management

• There are NO evidence based studies on the pharmacological treatment of associated symptoms in sports related concussion.

• Use one medication to treat multiple symptoms.

• Medications should be easily titrated and weaned.

• Medications need to be discontinued and the athlete monitored off of all such medications prior to RTP.
Pharmacological Management of Elite Athletes

- Take into account governing bodies rules and regulations for banned substances.

- Most governing bodies (NHL) uses WADA guidelines, NFL and MLB independent.

- Classes of Banned Substances; Anabolic steroids, growth factors, peptide hormones, oxygen transport enhancers, hormone antagonists (masking agents), Beta blockers, beta agonists, diuretics, glucocorticoids, Amphetamines, narcotics, cannabinoids.

- Check with governing body prior to treatment.
Management of Headache

- Most common symptom of concussion (88% of all athletes).

- Abortive and prophylactic therapy.

- Acute post traumatic migraine most common IHS subtype.

- No Class one evidence

- Apply current headache management principals.
Management of Headache

- OTC medications are first line
- Triptan’s for non responders.
- DHE-45 and Ketorolac for triptan non responders or patients with rapid onset of symptoms.
- Avoid narcotics and butalbital/caffeine containing compounds.
Management of Headache

- Preventative therapy for patients with > 6-8 headache days per month.
- TCA’s are the mainstay (Avoid Amitriptyline)
- AED’s not widely used
- Avoid cardiac medications i.e. beta blockers and calcium channel blockers.
- Leukotriene receptor antagonists.
- Memantine
- Vitamins and Minerals.
Management of Headache

- 15% will develop chronic post traumatic headache (attributed to mild head injury).
- Most likely to be referred by sports medicine physician.
- Require a multi-disciplinary approach.
- Best referred to a tertiary headache center.
- Avoid onabotulinum toxin (Botox®)
Return to Play

- Clinical signs are manifestations of neuronal dysfunction.
- Completely asymptomatic and off all medications.
- Normal neurological exam and all pre-concussion testing returned to baseline.
- Guidelines are not evidence based.
- Zurich 2008 used most widely.
- AAN outdated
2008 Zurich Return to Play Guidelines

• Stepwise process to RTP.

• Based on full clinical and cognitive recovery.

• Athlete can proceed to the next level if asymptomatic at the current level.

• At least 24 hrs. between steps, 1 week minimum to RTP.

• Symptoms return, athlete drops back to the previous asymptomatic level.

• No athlete should return to play on the same day.

• Zurich does not accurately reflect physiology.
### 2008 Zurich Return to Play Guidelines

#### Graded Return to Play Protocol

<table>
<thead>
<tr>
<th>Rehabilitation stage</th>
<th>Functional exercise at each stage of rehabilitation</th>
<th>Objective of each stage</th>
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<tbody>
<tr>
<td>1. No Activity</td>
<td>Complete physical and Cognitive Rest.</td>
<td>Recovery</td>
</tr>
<tr>
<td>2. Light aerobic exercise</td>
<td>Walking, Swimming, Stationary Bike, HR&lt;70% Maximum</td>
<td>Increased heart rate</td>
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<tr>
<td>3. Sport Specific Exercise</td>
<td>Skating or Running Drills without contact.</td>
<td>Add Movement</td>
</tr>
<tr>
<td>4. Non Contact training</td>
<td>More complex Drills without contact.</td>
<td>Exercise, coordination and cognitive load.</td>
</tr>
<tr>
<td>5. Full Contact</td>
<td>Normal Training</td>
<td>Restore confidence</td>
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<tr>
<td>6. Return to Play</td>
<td>Game Play</td>
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</table>
1. Any athlete suspected of suffering a concussion should be removed from participation until evaluated by a physician.

2. No athlete should be allowed to participate in sports if they are still experiencing symptoms of a concussion.

3. Following a concussion a physician and preferably a neurologist should be consulted prior to clearing the athlete to return to participation.

4. A certified coach or athletic trainer should be present at all sporting events, including practices, where risk of concussion is involved.

5. Education efforts should be maximized to improve the understanding of concussion by all athletes, parents, and coaches.

6. An athlete must be completely asymptomatic (cognitively and physically), and participate in non-contact game situations (without re-occurrence of symptoms), and complete a graded protocol prior to RTP.
RTP Children

- Children age 10 and younger and high school athletes recover slower.
- More complex neuro-anatomical and neuro-chemical make up.
- Physiological changes take longer to recover.
- Age appropriate symptom checklists and parental input.
- Neuropsychological testing is not appropriate for young children.
RTP Children

- Cognitive Rest
- Activity to tolerance with symptoms > 4 weeks.
- Physiological studies suggestive of 2-4 week recovery time.
- Prolonged Graded RTP that mirrors brain physiology.
- 72 Hours between steps.