Pediatric Concussion: where is the evidence?

Dr. Sami AL Farsi, MD
Senior consultant, PEM
Director, Child Health Department
Royal Hospital, Oman
DISCLOSURE

I do not have any relevant financial relationship with commercial interest to disclose.
Objectives

- Discuss the pathophysiologic basis for mild traumatic brain injury
- Describe the evidence base behind cognitive and physical rest after pediatric mild traumatic brain injury
- Explain appropriate return to activity guidelines to patients and families
CONCUSSIONS

- Headache
- Confusion
- Blurry Vision
- Sickness

I feel weird!
Epidemiology:

- More than 600,000 cases of pediatric head trauma present to EDs annually
- Highest reported rates of concussion in patients aged 12 to 17 years
- Males in early adolescence are at the highest risk for concussion (55-60%)
- Falls are the most common cause in children younger than 10 years of age.
- Sports-related concussions are the most common cause in children ages 10 years and older.
- The true incidence of concussion ~ significantly higher than estimated (diagnosed and treated outside an ED)
- Diagnosis of concussion has increased dramatically over the past decade (doubled)*
Pediatric Concussion

- Prospective, cross-sectional study @ tertiary care pediatric emergency department
- Ages 5 through 17 who presented with a head injury
- 495 children
- Mean age was 10.1 years, 62.2% were male
- Emergency physicians diagnosed concussion in 40.4%
- 89.5% Met the international criteria (Zurich consensus statement)

J Pediatr 2015;166:1214-20
Definition of Concussion
Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016


PREAMBLE
The 2017 Concussion in Sport Group (CISG) consensus statement is designed to build on the principles outlined in the previous statements1–4 and to develop further conceptual understanding of sport-related concussion (SRC) using an expert consensus-based approach. This document is developed for physicians and healthcare providers who are involved in athlete care, whether at a recreational, elite or professional level. While agreement exists on the principal messages conveyed by this document, the authors acknowledge that the science of SRC is evolving and therefore individual management and return-to-play decisions remain in the realm of clinical judgement. Articles were screened by the expert panels for the Berlin meeting. The details of the search strategies and findings are included in each of the systematic reviews.

The details of the conference organisation, methodology of the consensus process, question development and selection on expert panellists and observers is covered in detail in an accompanying paper in this issue.5 A full list of scientific committee members, expert panellists, authors, observers and those who were invited but could not attend are detailed at the end of the summary document. The International Committee of Medical Journal Editors conflict of interest declaration for all authors is provided in Appendix 1.
Definition

- Mild traumatic brain injury*

- A complex pathophysiological process induced by biomechanical forces

- Caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.
Definition

- Results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously

- Result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury*
Definition

The clinical signs and symptoms cannot be explained by drug, alcohol, or medication use, other injuries (such as cervical injuries, peripheral vestibular dysfunction, etc) or other comorbidities (eg, psychological factors or coexisting medical conditions)
Pathophysiology
Metabolic cascade occurs after a concussion
Sheer force and rotational deformity, rather than direct trauma, lead to mechanical damage to neurons and axons
Influx of calcium interferes with mitochondrial function and causes temporary cell dysfunction
Cerebral blood flow, which delivers glucose, decreases in response to injury, leading to an imbalance in energy supply and demand*
1. Widespread depolarization and neurotransmitter release

2. Potassium efflux

3. Calcium in the cell impairs ATP production in mitochondria, worsening energy crisis

4. Calcium influx also causes axonal swelling and decreased axonal function

Adolesc Med. 2015; 26: 491–506.
Clinical Manifestation and Diagnosis
Concussion

Headache  
Fatigue  
Amnesia  
Vomiting  
Decreased reaction time  
Knocked out  
Lack of concentration  
Inappropriate emotions

Seeing stars  
Slurred speech  
Lack of orientation  
Blurred vision  
Difficulty sleeping  
Nausea  
Lack of energy  
Loss of consciousness  
Irritability  
Dizziness  
Reduced coordination  
Getting your “bell rung”  
Easily distracted

Sensitivity to light  
Ringing in ears  
Sensitivity to noise  
Feelings of sadness
DIAGNOSIS OF CONCUSSION

- The diagnosis of structural brain injury has been excluded
- Concussion can still be difficult to identify
- Currently no objective measure with which to make the diagnosis
- The majority of concussions do not result in dramatic symptoms such as loss of consciousness
- A concussion can go unrecognized by both the patient and medical providers
- Cognitive diagnostic tools have been developed to aid in the identification of a concussion*
The Standardized Assessment of Concussion (SAC) and the Sport Concussion Assessment Tool 3 (SCAT3) are the most commonly used cognitive screens.

SCAT3 increasingly has been used. It assesses subjective symptoms, includes cognitive assessment, and evaluates coordination and balance.

- SCAT3_5 to 12.full.pdf
- SCAT3_13 and above.full.pdf

SCAT3 can be time-consuming and ideally it is used as a tool for repeat assessment.

It is not an ideal test to perform in the ED.
Many acutely concussed children are too symptomatic to complete neurocognitive testing, and exposing children to these tests in the acute setting may exacerbate symptoms.

The utility of neurocognitive testing acutely is somewhat controversial.

Lower raw scores on neurocognitive testing in the ED do not predict prolonged recovery.

More useful than raw data is comparison between a patient's baseline testing and his or her performance after an mTBI.

The most important use of such testing is likely in trending scores as a patient recovers and determining clearance for return to sports.
Neurocognitive testing, in conjunction with history and physical examination findings, may play a role in concussion diagnosis but should serve as an adjunct and not a primary diagnostic tool.
Clinical manifestation

- Concussions cause a constellation of functional symptoms in the absence of structural damage.
- Symptoms can begin with the trauma or in the subsequent 24-48 hours and may evolve slowly over days to weeks.
- Headache is the most commonly reported symptom.
- Immediate loss of consciousness occurs in < 10% of cases.
- Early symptoms of concussion may also include dizziness, amnesia to the event, confusion, nausea, and vomiting.
## Concussion Symptom*

<table>
<thead>
<tr>
<th>Physical</th>
<th>Cognitive</th>
<th>Emotional</th>
<th>Sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>Feeling mentally foggy</td>
<td>Irritability</td>
<td>Drowsiness</td>
</tr>
<tr>
<td>Nausea</td>
<td>Feeling slowed down</td>
<td>Sadness</td>
<td>Sleeping either more or less</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Difficulty with concentration</td>
<td>Emotional lability</td>
<td>than usual</td>
</tr>
<tr>
<td>Balance problems</td>
<td>Difficulty with memory</td>
<td>Nervousness</td>
<td>Trouble falling asleep</td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blurry vision</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Photophobia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phonophobia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numbness/tingling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
History

- When and how the injury happen?
- What where the initial and current symptoms?
- Activity level since the injury (in relation to school work, electronic devices and exercise)
- Sleep
- Previous medical evaluation during this injury
- Attending school?
History:

- ADHD, Learning disability or dyslexia
- Reading, vision, speech problems
- Glasses or contact lenses
- Anxiety, depression or migraine
- Motion sickness
Physical exam

- Standard neurologic examination (GCS)
- Evaluate for vestibular and oculomotor dysfunction, very common postconcussive deficits
- It can be performed in children as young as 6 years
- ~80% of concussed patients show at least 1 vestibular or oculomotor abnormality
- It helping prognosticate for patients with mTBI
- Vestibular deficits are more prevalent in children with prolonged symptoms
<table>
<thead>
<tr>
<th>Physical Examination Element</th>
<th>How to Perform Examination</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysmetria</td>
<td>Finger-nose-finger, examiner's finger moving horizontally, 10 repetitions</td>
<td>Slow reaction time, past-pointing, intention tremor</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Examiner's finger moving horizontally, progressively more rapidly, stopping centrally</td>
<td>&gt;1 beat of nystagmus at center of visual field</td>
</tr>
<tr>
<td>Smooth pursuits</td>
<td>Examiner's finger moving horizontally, progressively more rapidly</td>
<td>Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue)</td>
</tr>
<tr>
<td>Fast saccades</td>
<td>Examiner's fingers shoulder-width apart (horizontal) and forehead-chin distance (vertical), 30 repetitions</td>
<td>Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue, fogginess)</td>
</tr>
<tr>
<td>Gaze stability</td>
<td>Patient fixes gaze on examiner's thumb while nodding yes and then shaking head no side to side, 30 repetitions</td>
<td>Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue, fogginess)</td>
</tr>
<tr>
<td>Near-point convergence</td>
<td>Patient holds pen with letters at arm's length, brings toward nose until becomes double</td>
<td>Letters become double at &gt;6 cm from the tip of the nose</td>
</tr>
<tr>
<td>Gait/balance testing</td>
<td>Tandem heel-toe gait forward and backward with eyes open and closed</td>
<td>Raises arms for stability or widens gait, extreme truncal swaying</td>
</tr>
</tbody>
</table>
Vestibulo-Ocular Examination for Concussion

A

B
NEUROIMAGING AND LABORATORY TESTING

- Diagnosis is made by history and physical examination
- Not require neuroimaging
- CT brain, Multiple studies have developed decision models to help rule out clinically significant intracranial hemorrhage
- MRI has little role in the immediate diagnosis of concussion
- Future: functional MRI may be have role rehabilitate patient with prolong recovery
- Lab test: No role currently
- Lab test future: that there may be a role in the future for GFAP, among other potential candidate markers (including S100 calcium binding protein B, myelin basic protein, and ubiquitin C-terminal hydrolase-L1), as potential indicators for more severe concussion
# PECARN (Decision Rules for Avoiding CT in Children with Head Trauma)

<table>
<thead>
<tr>
<th>&lt; 2 years of age</th>
<th>&gt; 2 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal mental status</td>
<td>Normal mental status</td>
</tr>
<tr>
<td>No scalp hematoma except frontal</td>
<td>No loss of consciousness</td>
</tr>
<tr>
<td>Loss of consciousness &lt; 5 seconds</td>
<td>No vomiting</td>
</tr>
<tr>
<td>Non-severe injury mechanism*</td>
<td>Non-severe injury mechanism*</td>
</tr>
<tr>
<td>No palpable skull fracture</td>
<td>No signs of basilar skull fracture</td>
</tr>
<tr>
<td>Normal behavior</td>
<td>No severe headache</td>
</tr>
</tbody>
</table>

*Severe mechanism defined as motor vehicle collision with patient ejection, death of a passenger, rollover; pedestrian or cyclist without helmet struck by motorized vehicle; fall > 1.5 m for children (> 2 years), struck by motorized vehicle; fall > 1.5 m for children (> 2 years), fall > 0.9 m for < 2 years; or head struck by high-impact object*
Management
Most consensus and agreement statements
Till they become symptom free
Promote recovery by minimizing brain energy demands
The exact amount and duration of rest is not yet well defined in the literature and requires further study
Brief period of rest during the acute phase (24–48 hours) after injury
Patients can be encouraged to become gradually and progressively more active while staying below their cognitive and physical symptom-exacerbation thresholds (i.e., activity level should not bring on or worsen their symptoms)
COGNITIVE AND PHYSICAL REST

- Emergency setting
- No significant differences between the 2 groups at baseline
- Randomized a group of 88 patients
- Aged 11 to 22 years
- 5 days of strict rest (no work, no school, and no physical activity) VS 1 to 2 days of rest followed by a stepwise return to activity*
- Participant prescribed strict rest had a slower resolution of their symptoms and had a higher symptom burden during the first 10 days mainly emotional symptoms

*Pediatrics. 2015 Feb;135(2):213-23
Medication

- Acetaminophen and ibuprofen (acute setting)

- Not helpful during the recovery phase and should not be continued after ED discharge

- Avoid narcotics to prevent changes in mental status or neurologic exam
Medication

- Nausea is also a common problem associated with concussion. Non-sedating antiemetics (Ondansetron)

- Other medications (gabapentin, topiramate, and amitriptyline) outside ED by specialist for ostconcussive syndrome
Disposition

Patients can be discharged safely from the ED if the following criteria are met:

- Return to baseline level of function with a GCS of 15
- Tolerance of oral intake without significant nausea or vomiting,
- No other injuries warranting admission
- Reliable caretakers
Disposition

Discharge instructions should outline strict return precautions:

- Inability to awaken the child
- Seizure
- Vomiting that begins 4-6 hours after the injury or continued vomiting
- Change in mental status
- New unsteady gait or clumsiness/incoordination
Disposition

Follow-up with a physician or an athletic trainer should be arranged within 48 hours for symptom re-evaluation and initiation of progression to return to activity if appropriate.
## Graduated return-to-school strategy

<table>
<thead>
<tr>
<th>Stage</th>
<th>Aim</th>
<th>Activity</th>
<th>Goal of each step</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Daily activities at home that do not give the child symptoms</td>
<td>Typical activities of the child during the day as long as they do not increase symptoms (e.g., reading, texting, screen time). Start with 5–15 min at a time and gradually build up.</td>
<td>Gradual return to typical activities</td>
</tr>
<tr>
<td>2</td>
<td>School activities</td>
<td>Homework, reading or other cognitive activities outside of the classroom</td>
<td>Increase tolerance to cognitive work</td>
</tr>
<tr>
<td>3</td>
<td>Return to school part-time</td>
<td>Gradual introduction of schoolwork. May need to start with a partial school day or with increased breaks during the day.</td>
<td>Increase academic activities</td>
</tr>
<tr>
<td>4</td>
<td>Return to school full time</td>
<td>Gradually progress school activities until a full day can be tolerated.</td>
<td>Return to full academic activities and catch up on missed work</td>
</tr>
</tbody>
</table>
# Graduated return-to-sport (RTS) strategy

<table>
<thead>
<tr>
<th>Stage</th>
<th>Aim</th>
<th>Activity</th>
<th>Goal of each step</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Symptom-limited activity</td>
<td>Daily activities that do not provoke symptoms</td>
<td>Gradual reintroduction of work/school activities</td>
</tr>
<tr>
<td>2</td>
<td>Light aerobic exercise</td>
<td>Walking or stationary cycling at slow to medium pace. No resistance training</td>
<td>Increase heart rate</td>
</tr>
<tr>
<td>3</td>
<td>Sport-specific exercise</td>
<td>Running or skating drills. No head impact activities</td>
<td>Add movement</td>
</tr>
<tr>
<td>4</td>
<td>Non-contact training drills</td>
<td>Harder training drills, eg, passing drills. May start progressive resistance training</td>
<td>Exercise, coordination and increased thinking</td>
</tr>
<tr>
<td>5</td>
<td>Full contact practice</td>
<td>Following medical clearance, participate in normal training activities</td>
<td>Restore confidence and assess functional skills by coaching staff</td>
</tr>
<tr>
<td>6</td>
<td>Return to sport</td>
<td>Normal game play</td>
<td></td>
</tr>
</tbody>
</table>
Graduated return-to-sport (RTS) strategy

- Initial period of 24–48 hours of both relative physical rest and cognitive rest is recommended before beginning the RTS progression
- There should be at least 24 hours (or longer) for each step of the progression
- If any symptoms worsen during exercise, the athlete should go back to the previous step
Recovery

- Clinical recovery is defined functionally as a return to normal activities, including school, work and sport, after injury.

- Resolution of post-concussion-related symptoms and a return to clinically normal balance and cognitive functioning.
Predictors of prolonged symptoms

- Vary across studies
- Female sex
- Increased number of initial symptoms
- Older age (adolescent vs child)
- Loss of consciousness and amnesia
- Premorbid conditions including previous concussion, learning difficulties, and psychiatric illness
Clinical Risk Score for Persistent Postconcussion Symptoms Among Children With Acute Concussion in the ED

- Prospective, multicenter cohort study
- Ages 5 to 18 from 9 pediatric EDs within Canada between 2013 and 2015
- Purpose: To derive and validate a clinical risk score in children to stratify PPCS risk after a concussion
- 2584 patients who presented within 48 hours of an acute head injury and who followed-up 28 days later were enrolled in both a derivation and validation cohort
- 30% met criteria for PPCS in the derivation cohort, & 33% in the validation cohort
- The variables included in the PPCS risk score model included female sex, age 13 years or older, fatigue, sensitivity to noise, headache, physician-diagnosed migraines, prior concussion with symptoms lasting longer than a week, answering questions slowly, and 4 or more errors on the Balance Error Scoring System tandem stance

JAMA. 2016;315(10):1014-1025
Clinical Risk Score for Persistent Postconcussion Symptoms Among Children With Acute Concussion in the ED

- Each patient could score a total of 12 points in the model.
- Low risk patients (<3 points) had a negative likelihood ratio of 0.36
- High risk patients (>9 points) had a positive likelihood ratio of 3
- Allowing higher risk individuals to be identified and treated as early as possible

JAMA. 2016;315(10):1014-1025
Persistent symptoms

Failure of normal clinical recovery, symptoms that persist beyond expected time frames (ie, >10–14 days in adults and >3-4 weeks in children)

A detailed clinical assessment is required to identify specific primary and secondary pathologies that may be contributing to persisting post-traumatic symptoms.

Treatment should be individualized and target-specific medical, physical and psychosocial factors identified on assessment

Cases better be managed in a multidisciplinary collaborative setting, by healthcare providers
Complications (Post concussion syndrome)

- Most patients with a concussion will have a spontaneous, sequential resolution of their symptoms within 7 to 10 days.
- Some patients have a prolonged recovery with sustained symptoms (PCS).
- Persistent headache, dizziness, cognitive impairment, and psychological symptoms lasting from 6 weeks to 3 months after a concussion.
- Treatments by an experienced practitioner (e.g., Tricyclic antidepressants for postconcussive headache).
- Patients presenting to the ED with signs and symptoms of PCS should be referred to a pediatric neurologist, neuropsychologist, sports medicine physician, or other specialist with expertise in head injury.
Complications (Second Impact Syndrome)

- Rare but feared complication of concussion
- Sustains a second impact before the symptoms and pathophysiological changes from the first injury have fully cleared
- Cerebral swelling and herniation
- Pediatric and adolescent patients at the highest risk of this condition
- Need to be monitored closely for deterioration of neurologic state after one or more concussive events
COMPLICATIONS (Chronic traumatic encephalopathy)

- Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease caused by repetitive head trauma.

- Manifestations include mood disturbances, parkinsonism, ataxia, dysarthric speech, poor concentration, attention and memory loss, and behavioral outbursts.
SUMMARY

- Concussion is subset of mTBI
- Full medical assessment with a thorough history from both the patient and witnesses to the event
- The physical exam should pay special attention to the head, neck, and neurologic exam, including mental status, gait, balance, Oculo-vestibular examination and evidence of associated extracranial injury
- Most symptoms resolve within 7-10 days
SUMMARY

- Diagnosis by History and PE
- No role of neuroimaging and lab test
- Cognitive and physical rest till they become symptom free
- Refer if Persistent symptoms ( >10–14 days in adults and > 3-4 weeks in children)
- Graduated return-to- school and sport strategies
- Pay attention to complication PCS, CTE and second impact syndrome
THANKS