Management of Concussion & Post-Concussion Syndrome

Vernon B. Williams, MD
October 26, 2017

PLEASE STAND BY – WEBINAR WILL BEGIN AT 12:00 PM PST
FOR AUDIO: CALL 866-740-1260 / ACCESS CODE: 764-4915#

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CONCUSSION DISCUSSION:
Content, Concepts, and Context

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California State Athletic Commissioner
Consultant to Brain Treatment Center
DISCUSSION ELEMENTS

- Pre-Injury Concepts
- Acute Event
- Individualized Symptom Management
- Return to Work
- Retirement Considerations
- Chronic Conditions
SPORTS CONCUSSION

When in Doubt—Sit It Out!

Helmets Save Lives

UP TO 3.8 million

sports/recreation concussions each year

What is a Concussion?

A condition resulting from the stunning, damaging, or shattering effects of a hard blow to the head.

The First 10 Days

If you are within ten days of having a concussion, there is a greater risk for another one.

Glossary

 Concussion is recognized as a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness (LOC). Estimates of sports-related mild traumatic brain injury (mTBI) range from 1.6 to 3.8 million affected individuals annually in the United States, many of whom do not obtain immediate medical attention. The table summarizes the currently available data for the overall concussion rate (CR) and the CRs for 5

HEAD TRAUMA Followed by ANY of These?

- Headache
- Dizziness
- Confusion
- Memory Loss
- Difficulty Concentrating

Access resources on how to identify and seek treatment for sports concussion at AAN.com/view/concussion

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Summary of evidence-based guideline update: Evaluation and management of concussion in sports


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Comprehensive to American Academy of Neurology

guideline.on.com

ABSTRACT

Objective: To update the 1997 American Academy of Neurology (AAN) practice parameter regarding sports concussion, focusing on 4 questions: 1) What factors increase the concussion risk? 2) What diagnostic tools identify those with concussion and those at increased risk for severe/longitudinal early impairments, neurologic catastrophe, or chronic neurobehavioral impairment? 3) What clinical factors identify those at increased risk for severe/longitudinal early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment? 4) What interventions enhance recovery, reduce recurrent concussion risk, or diminish long-term sequelae? The complete guideline on which this summary is based is available as an online data supplement to this article.

Methods: We systematically reviewed the literature from 1995 to June 2012 for pertinent evidence. We assessed evidence for quality and synthesized into conclusions using a modified Grading of Recommendations Assessment, Development and Evaluation process. We used a modified Delphi process to develop recommendations.

Results: Specific risk factors can increase or decrease concussion risk. Diagnostic tools help identify individuals with concussion including graded symptom checklists, the Standardized Assessment of Concussion, neurocognitive assessments, and the Balance Error Scoring System. Specific symptomatology, concussive history, and younger age identify those at risk for postconcussive impairments. Risk factors for recurrent concussion include history of multiple concussions, particularly within 1.0 years after initial concussion. Risk factors for chronic neurobehavioral impairments include concussion exposure and APoE e4 genotype. Data are insufficient to show that any intervention enhances recovery of disabilities long term or reduces postconcussion. Practice recommendations are presented for preparticipation assessment, management of suspected concussion, and management of diagnosed concussion. Neurology 2013;100

Supplemental data at www.neurology.org

2013 American Academy of Neurology

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Additional References: The guideline is available at www.neurology.org.
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**Terminology**

**Concussion**

**PCS**
Zurich 2012: Concussion is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be utilized in defining the nature of a concussive head injury include:

1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an “impulsive” force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms

NATA: concussion as a “trauma-induced alteration in mental status that may or may not involve loss of consciousness.” The terms “ding,” “getting one’s bell rung,” “clearing the cobwebs,” and other such phrases in reference to concussive injuries are antiquated, minimize injury severity, and should not be used to refer to concussion or mild traumatic brain injury.

International Rugby League: Concussion is a brain injury caused by either direct or indirect forces to the head. Concussion typically results in the rapid onset of short-lived impairment of brain function. Loss of consciousness occurs in less than 15% of concussion cases and whilst a feature of concussion, loss of consciousness is not a requirement for diagnosing concussion. Concussion results in a disturbance of brain function rather than damage to structures such as blood vessels, brain tissue or fractured skull. Typically standard neuroimaging such as MRI or CT scan is normal.

AAN: a form of mild traumatic brain injury (TBI), is a common consequence of trauma to the head in contact sports. A clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness (LOC).

American Medical Society for Sports Medicine: Concussion is defined as a traumatically induced transient disturbance of brain function and involves a complex pathophysiologic process. Concussion is a subset of mild traumatic brain injury that is generally self-limited and at the less severe end of the brain injury spectrum.

Department of Defense/VA: A traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event:

- Any period of loss of or a decreased level of consciousness (LOC)
- Any loss of memory for events immediately before or after the injury (post-traumatic amnesia [PTA])
- Any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.) (Alteration of consciousness/mental state [AOC])
- Neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient
- Intracranial lesion. External forces may include any of the following events: the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement without direct external trauma to the head, a foreign body penetrating the brain, forces generated from events such as a blast or explosion, or other forces yet to be define
Typically, standard neuroimaging such as MRI or CT scan is normal.

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Multiple Definitions

Wrong Metric? (Sub-Concussive)

Sport vs Civilian vs Military
Pre-Injury Concepts
KEY POINT

Every game plan starts with a “philosophy”:

• *All* concussions are serious
• Management starts long *before* injury/symptoms
• *Removal* of symptomatic athletes from play
• Recognition of *unique/individual* aspects
Neurometabolic Changes

PERIOD OF VULNERABILITY
Another concussion during this period can lead to irreparable damage or death.

DEMAND FOR GLUCOSE

MISMATCH

BLOOD FLOW
METABOLIC RATE FOR OXYGEN

0 days
-1
0
1
2
3
4
5
6
7
8
9
10
11
12
13

150%
100
(Normal)
50
0
Neurometabolic Cascade

- Glutamate binds NMDA
- K+ out
- Ca++ in → cell death
- Na+/K+ pump very active
- Increased lactate
- Inflammatory response
- Glucose hypermetabolism
- Decreased cerebral blood flow
- Cellular energy crisis = postconcussive vulnerability of brain

Axonal Injury

- Stretch injury breaks microtubules
- Mechanical failure of axonal cytoskeleton
- Transport catastrophe
- Primary physical injury (stretch and relaxation/breaking tubules) followed by secondary chemical insult (co-accumulation of Amyloid Beta, Amyloid Precursor Protein, Tau, and Neuofilament)
1. Mechanical damage
2. Chemical damage

Catastrophe!!

Stretch injury breaks microtubules

Tang-Schomer et al., FASEB 2010

Microtubule Ruffling Site
Dynamic Loading Microtubule Shearing Postinflammation Stress
MT Breaking Pantol Relaxation
Restricted Transport
Longer Term and Chronic or Delayed Changes

- Delayed Glucose Hypometabolism
  initial hyperglycolysis followed by low glucose use (5-10 days in animals, up to 4 weeks in humans)

- Neurotransmitter Alterations
  alterations in glutamatergic, adrenergic, and cholinergic systems (late loss of cholinergic neurons leads to learning and spatial memory deficits in animals)

- Delayed Cell Death and Persistent Calcium Accumulation
Concussion Suspected/Reported

Athlete Self-Report

Relies on athlete buy-in
Concussion Suspected/Reported

Report From 3rd Party

Requires educated stakeholders
Concussion Suspected/Reported

Personal Observation

Recognize physical and behavioral signs:

- Obvious
- Subtle
Acute Concussion

- ABC’s
- Exam
- Disposition
- Follow-up
Check for Red Flags

- Loss of Consciousness
- Neurological deficits
- Loss of bowel or bladder control
- Vomiting
- C-Spine Injury
- Skull fracture/defect
"RED FLAGS" (What to do and check for)

- L.O.C. > 1 minute (prolonged)
- Combative/Agitated? Abnormality of speech?
- Excessively drowsy? Severe or worsening headache?
- Slow or slurred speech? Agitated? Confused?
- C-spine tenderness/pain with movement?
- Abnormal ocular movements? Unequal pupils? Diplopia?
- Visual field loss? Weakness? Seizure activity?
- Black eyes or bruising behind ears? CSF leak ears/nose?
Immediate/On Field or Sideline Assessment

- Maddocks or Modified Maddocks (venue, half, scored last, played last week, won last game?)

- GCS: Best eye opening (1-4), verbal (1-5), and motor (1-6) responses (<13 = ER)

- C-spine Assessment: Pain-free at rest? Full active ROM? Normal motor/sensory exam?
Locker-Room or Office/Clinical Assessment

- Symptoms (Graded Symptom Checklist)
- Cognitive Screening
  - Orientation
  - Memory (5/10; Immed./Delayed)
  - Concentration (Digits/Months)
- Neurological Examination
  - C.N.'s, C-spine, Coord., Balance

SCAT 5: Focuses on Cognitive. No autonomic or Behavioral Assessment
What's New - Office Assessment?

- Transition to Evidence-Based Physical Examination
- Cervical strength and proprioception
- Autonomic assessment
- Static and dynamic visual acuity
- Screen for behavioral, psychiatric, and substance abuse d/o's
- Technology (BNA, QEEG, DTI, fMRI, MEG, Sleep)
Individualized Symptom Management
Symptom Management/RTP

H&P

Testing

Symptom Management

Return to Play
Approach: Cognitive Restructuring
Nocebo Effect/Response

The *expectation* of sickness and the affective states associated with such expectations *cause sickness in the expectant*.
Media and Anecdotes

- Most anecdotal stories and sensational cases distort framing of real (relative) risk.

- Anecdotes glorified in media.

- “Chief complaint CTE”
  - 14 year old attempted suicide
  - 25 year old former college football player
Clinical impairments in attention, memory, processing speed and reaction time may be driven by underlying impairments in neurotransmission or axonal dysfunction resulting in impaired network connectivity.
Watchful waiting might not work best.

- Traditional default position of concussion management.
- Doesn’t violate Grading Scale Guidelines
Approach: Individualized Management

- Spectrum/Continuum
- Age
- Circumstance
Approach: Stratified Care
Definition of Population Sub-Groups

Susceptibility

Prognosis

Response to Treatment
Characteristics
Genetic and Clinical Biomarkers
Severity of Sx.'s in 1st 24-48hrs. consistently associated w/ worse outcome

Complicating Factors

**Postconcussion Syndrome**: symptoms last weeks to months after injury
- Longer recovery (>21 days) predictors: dizziness at time of injury, more cognitive or migraine symptoms, headache lasting >60 hr, 3+ symptoms at initial presentation, and presence of fatigue / tiredness / fogginess

**Second Impact Syndrome**

**Chronic Traumatic Encephalopathy**

Herring et al., 2011
Vagnozzi et al., 2010
Giza & Hovda, 2001

<table>
<thead>
<tr>
<th>Table 2 Concussion modifiers</th>
<th>McCrory et al., 2013</th>
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</thead>
<tbody>
<tr>
<td><strong>Factors</strong></td>
<td><strong>Modifier</strong></td>
</tr>
<tr>
<td>Symptoms</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td>Duration (&gt;10 days)</td>
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<tr>
<td></td>
<td>Severity</td>
</tr>
<tr>
<td>Signs</td>
<td>Prolonged loss of consciousness (LOC) (&gt;1 min), Amnesia</td>
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<tr>
<td>Sequence</td>
<td>Concussive convulsions</td>
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<tr>
<td>Temporal</td>
<td>Frequency—repeated concussions over time</td>
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<td></td>
<td>Timing—injuries close together in time</td>
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<td>‘Recency’—recent concussion or traumatic brain injury (TBI)</td>
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<tr>
<td>Threshold</td>
<td>Repeated concussions occurring with progressively less impact force or slower recovery after each successive concussion</td>
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<tr>
<td>Age</td>
<td>Child and adolescent (&lt;18 years old)</td>
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<tr>
<td>Comorbidities and premorbidities</td>
<td>Migraine, depression or other mental health disorders, attention deficit hyperactivity disorder (ADHD), learning disabilities (LD), sleep disorders</td>
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<tr>
<td>Medication</td>
<td>Psychoactive drugs, anticoagulants</td>
</tr>
<tr>
<td>Behaviour</td>
<td>Dangerous style of play</td>
</tr>
<tr>
<td>Sport</td>
<td>High-risk activity, contact and collision sport, high sporting level</td>
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</tbody>
</table>
Approach: Treatable Conditions
Post-Concussion Headache

Landscape
- Most frequently experienced concussion related symptom (as high as 88%)
- Can be acute (< 3 months) or chronic (> 3 months)
- Emergent/catastrophic: Secondary causes (ICH, increased ICP) must be excluded

Common Sub-Types
- Tension
  - Abortive: simple analgesics, aspirin, acetaminophen, NSAID's
  - Preventive: amitriptyline best efficacy
- Migraine
  - Abortive: non-specifics, NSAID's, simple analgesics, combination analgesics
  - Preventive: Propranolol, timolol, valproic acid, amitriptyline, topiramate
- Cluster
  - Abortive: 100% O2 via facemask, subQ sumatriptan or DHE
  - Preventive: verapamil, lithium, topiramate
- Mixed
- Trigemocervical Nucleus (area of upper cervical cord where sensory fibers in descending tract of trigeminal nucleus caudalis are believed to interact with sensory fibers from upper cervical roots)

COMMONLY MISSED
- Cervicogenic
- Whiplash

Special Treatment Considerations
- Supplements: Fever Few, Riboflavin
- Medrol, Dosepak, Toradol
correcting the sleep-wake cycle

**SETTING THE CIRCADIAN RHYTHM**

**MORNING ROUTINE:** To maximize your time with us, it is important to improve the quality of the patient’s sleep. Oddly enough, proper sleep begins with waking up properly. To achieve this, the patient must start their morning waking early enough to receive at least 45 minutes (1 hour ideally) of exposure to the morning sky between the hours of 7:00-11:00 AM. Why 7:00-11:00 am? It is during this time a very specific wavelength of light is emitted as the sun reflects off of the atmosphere. Sorry, glasses and windows of every type block the light we are looking for.

**EVENING ROUTINE:** In the evening we ask that the patient gets to sleep around 9:00 PM to ensure the individual is sleeping between the hours of 10:00 PM and 2:00 AM. Our research has shown that the rest provided to the brain during this time frame proves to be one of the most important elements in allowing treatment to be effective.

**MORNING ROUTINE:**
7:00am-11:00am
Morning exposure for at least 45 minutes during this period.

**EVENING ROUTINE:**
10:00pm-2:00am
Sleep during this 4-hour period is critical for efficacious treatment results.

**MORNING LIGHT = PROPER SLEEP**

When activated by morning sunlight (450-500nm) wavelength, the suprachiasmatic nucleus (SCN) delivers inhibitory information to the paraventricular nucleus (PVN). This is the start of a complex biological process that culminates in the pineal gland which regulates and suppresses melatonin secretion in the brain. Once this process begins, roughly 12 hours later suppression of melatonin will cease, leading to drowsiness and sleep.

1. Morning sunlight between the 450-500nm wavelength hits the eye. This morning light is integral for setting your circadian rhythm.

2. The supranucleus (SCN) receives this light and passes inhibitory signals on to the preganglionic sympathetic neurons of the spinal cord.

3. Pineal gland receives feedback from the paraventricular nucleus (PVN) and regulates melatonin secretion in the brain.

**Center for Sports Neurology**
Visual Impairment after TBI

Evaluation Protocol

Visual Pathway Assessment

Sensory Fusion Assessment

Ocular Motility Assessment

motor and sensory aspects functions of visual system

Patient Complaints and Clinical Observations

visual function changes occur in 4 domains

Landscape

visual function changes occur in 4 domains

- person often unaware or has difficulty describing
- Person may have developed partial/incomplete compensatory strategies

Also consider reduced or sensitivity function
- optic nerve
- visual hemi fields
- accommodative

Oculomotor control

Central visual processing

Decreased Acuity

- decreased acuity
- low contrast
- difficulty reading

Visual Field Deficit

- difficulty identifying objects
- confusion with orienting
- difficulty navigating

Oculomotor Dysfunction

Visual Inattention

Subtle
- only falls visual task, not middle
- only fails verbal task, not middle
Vestibular Impairment after TBI

Training
- Gaze Stability
- Eye/Head Motion
  - Balance

Testing
- BESS
- Head Thrust Test
- Visual Acuity Test
- Nystagmus Test
- Dizziness Handicap Inventory

Landscape
- Dizziness, balance problems reported as early symptom in 55%/43% of sports concussion
- Vestibular dysfunction of specific concern in those with delayed/prolonged recovery
- Often responsive to Vestibular Rehabilitation

Potential Post-Traumatic Issues
- Dizziness
- Postural Instability
- Fogginess
- Balance Dysfunction
- Oculomotor Dysfunction
- Motion Intolerance
- Headache/Migraine
- Perilymphatic Fistula
- Temporal bone fracture
- Impaired Balance
- Blurry Vision
- Difficulty Focusing
- Difficulty in busy environments
- Headache
- Neck Pain
- Auditory (tinnitus, fullness)

Subjective Symptoms
- Dizziness
- Postural Instability
- Fogginess
- Balance Dysfunction
- Oculomotor Dysfunction
- Motion Intolerance
- Headache
- Neck Pain
- Auditory (tinnitus, fullness)
Approach: Multi/Inter-Disciplinary
General Return to Work Principles

- Recovery and return recommendations should be individualized.
- Avoid "cocoon therapy".
- Provide symptom based accommodations.
- Role for graded exercise after acute (one week or so) period.
Post-Concussive Dysfunction Both Metabolic and Functional May Have Two Phases

- **Early:** Associated with vulnerability to second/worsened injury
  Benefit to cognitive and physical rest (protection from excessive stimulation.)

- **Late:** May benefit from *controlled* stimulation.
Evidence in Support of Conservative Approach

Metabolism, Rest, and Return to Play

- Metabolic Abnormalities May Be Sub-Clinical
- Metabolic Abnormalities Last Longer (45 days vs 30 days) if Second Concussion Within Days of First Concussion
- No Clear Linkage To Clinical Symptoms or Neurocognitive Measures
Evidence in Conflict of "Complete Rest Until Symptom-Free"

Graded Exercise

Animal evidence suggests that exercise in concussed rodents worsens outcome when the exercise occurs acutely after injury, however, if instituted >1 week after injury the exercise then confers benefits in terms of recovery (Griesbach 2004, 2007).

Human evidence is limited to a couple Class III studies that, while intriguing, require replication prospectively and with more objective measurement of activity levels. One human study suggests moderate levels of activity were associated with best outcome (Majerske 2008).
Retirement Considerations.
Patterns
Abnormal Exam
Abnormal Imaging
Desire
Number of Events?

Change Jobs?
Change Position?
Chronic Conditions
Persistent Concussion Symptoms >10 days?

Post Concussion Syndrome > 3 months?
Chronic Traumatic Encephalopathy (CTE)
  - Pathologic/Anatomic Diagnosis
  - pTau at depths of sulci; Distinct from AD

Traumatic Encephalopathy Syndrome (TES)
  - Neurocog. c/o x 2 yrs+
  - Hx. Head Impact Exposure
  - Progressive emotional, motor, & behavioral dysfx.
  - Delayed onset distinguishes from PCS (8-10 yr. latency)
Symptoms

**Neurologic**

**Non-Neurologic**
Longer Term and Chronic or Delayed Changes

- Delayed Glucose Hypometabolism
  initial hyperglycolysis followed by low glucose use (5-10 days in animals, up to 4 weeks in humans)

- Neurotransmitter Alterations
  alterations in glutamatergic, adrenergic, and cholinergic systems (late loss of cholinergic neurons leads to learning and spatial memory deficits in animals)

- Delayed Cell Death and Persistent Calcium Accumulation
- **Stretch injury** breaks microtubules
- **Mechanical failure of axonal cytoskeleton**
- **Transport** catastrophe
- **Primary physical injury** (stretch and relaxation/breaking tubules) followed by **secondary chemical insult** (co-accumulation of Amyloid Beta, Amyloid Precursor Protein, Tau, and Neuofilament)
Symptoms

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Non-Neurologic
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