Course Objectives:

A. Define concussion and the current understanding of concussion pathophysiology
B. Become familiar with procedures to identify concussion on site or when presenting hours-days post injury
C. Be able to utilize standard decision making for return to activity
D. Gain an understanding of what a persistent post concussion syndrome is and the factors that complicate recovery
E. Be able to devise an appropriate treatment plan for the patient with a persistent post concussion syndrome

PART 1 ACUTE CONCUSSION

A. Introduction and Overview
B. Overview of Issue
   1. Statistics: 2010 – 2.5 million ED visits, hospitalizations or deaths associated with TBI
   2. Leading causes are falls, unintentional blunt trauma, MVAs and assaults (CDC)
   3. Estimates by CDC place totals at 1.6 -3.8 million each year; 300,000 are estimated as sports-related (approximately 25% are football players)
   4. Evolution of concussion knowledge
   5. Young brains vs. mature brains
C. Iowa Law: Senate File 367
   1. Defines licensed healthcare professional
   2. Outline requirements for State of Iowa
D. Terminology
   1. Traumatic Brain Injury (TBI): TBI is defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force (Menon et al., 2010)
   2. Mild Traumatic Brain Injury (mTBI): form of TBI
   3. Concussion: type of traumatic brain injury (TBI) that results from a bump, blow or jolt to the head (or hit to the body) that causes the brain to move about in the skull. (CDC) OR “Trauma-induced alteration in mental status
that may or may not involve loss of consciousness” NATA 2014 position statement

4. Sports Concussion: Zurich Statement

E. Mechanism of Injury - Biophysics

i. Primary Injury – Impact event
   1. Blow to head, face, neck or elsewhere on the body
   2. Modern day athletes are larger and impact velocities are increased (known)
   3. Highest strain forces imparted at the deep mid-brain level, 10 milliseconds following impact (extrapolated from laboratory models based on NFL computerized video analysis)
   4. Cumulative number of head impacts best correlates with the potential for concussion occurrence or chronic effects (measured as dose over a lifetime)

ii. Secondary Injury – Subsequent pathophysiological process
   1. Includes immediate and delayed cellular events
   2. Area of brain affected determines function impaired
   3. Signs and symptoms

F. On-site responder – Using case studies throughout
   1. Suspect a concussion? Remove from play
   2. Evaluation & Assessment – tools to use
   3. Decision Making Process
   4. Next-steps – home instructions, talking with parent/guardian

G. Skills session – Evaluation of a suspected concussion
   1. Symptom assessment
   2. SCAT or SCAT 2
   3. Neurological Assessment
   4. Balance testing – BESS testing

H. Return to Learn/Work (RTL) & Return to Play/Activity (RTP)
   1. Initial response to School/Work and Play/Activity
   2. Graduated RTL & RTP protocols
   3. RTL should happen BEFORE RTP

References


b. Dashnaw ML, Petragalia AL, Bailes JE. An overview of the basic science of concussion and subconcussion: where we are and where we are going. *Neurosurg Focus*, 2012;33 (6): E5.


f. McAvoy K. REAP The Benefits of Good Concussion Management. HCA-Healthone LLC, 2ed, 2013. Available at RockyMountainHospitalForChildren.com


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**Resources and Links**

CDC – Heads up program

Search “heads up”; toolkits are available for a variety of individuals

http://www.cdc.gov/concussion/sports/resources.html#3

REAP Program by Karen McAvoy, PsyD

http://www.sportsconcussions.org/REAP.html

Rocky Mountain Youth Sports Medicine Institute: Center for Concussion

**Concussion management manual** You must create an account with Issuu to download, but it is definitely worth your time.


Connecticut Concussion Task Force has some very specific information for parents & guardians, school staff, student athletes, kids & teens, healthcare providers, and coaches & athletic staffs as you begin to think about ‘Return-to-Learn’ protocols.

http://www.connecticutconcussiontaskforce.org/


Traumatic Brain Injury Networking Team Resource Network [www.COKidswithbraininjury.com](http://www.COKidswithbraininjury.com) . This website provides guidance to school psychologists and related service providers through assessments and interventions for students with a traumatic brain injury.


Brain Injury Alliance Iowa – [www.biaia.org](http://www.biaia.org)

Concussion Information on the home page


Iowa Athletic Trainers’ Society – [www.iowaats.com](http://www.iowaats.com)
PART 2 PERSISTENT CONCUSSION

A. How do neurons work?
   1. To survive neuron must have:
      a. Fuel
      b. Other nutrients
      c. Stimulation
      d. No inflammation
   2. Fuel is related to both supply and demand
      a. Supply - oxygen, glucose. Anemia and dysglycemia are both very significant issues for brain function
      b. The brain is very metabolically active - meaning it has a higher oxygen demand per unit of tissue than other tissues in the body...AND
      c. The brain does not store any fuel. Neurons get their energy via a “hand to mouth” system.

   NOTE: An area or circuit in the brain that has “normal” cerebral blood flow, may become damaged or dysfunctional if the area has a high demand (is working hard) and the “normal” flow is just not enough to keep up.

   3. Vitamins, minerals and other nutritional factors both directly and indirectly effect brain function.
      a. Neurotransmitters
      b. Components of neuronal and extra neuronal structures
      c. Balance in the intra and extracellular environment
      d. Signaling in immune and fuel supply pathways

   4. Stimulation
      a. Use it or lose it
      b. If neurons have abundance of fuel and other nutrients, stimulation can make them grow, become more fuel efficient and more stable
      c. Plasticity and Windup

   5. Inflammation
      a. The brain should be a sterile environment with no “debris”
      b. There is a mostly separate immune system within the brain that is regulated to keep it from phagocytizing itself
      c. Microglia - Rest, Primed, Active

B. Head/neck trauma & concussion
   1. Brain is affected directly..... but how much?
   2. Imaging is negative, but there is disruption of the system
      i. Mechanical- axonal, BBB, etc
ii. Chemical leakage into extracellular environment  
iii. Cerebral Blood flow changes  
iv. Immune responses  
v. Demand increases

3. The secondary affect of concussion:

SIMULTANEOUS ISSUES
I. Neurons become fragile, spill some excitatory contents (glutamate), Ca++ enters (Mg+ exits), neurons become much more sensitive and fire, increasing demand in a compromised system, neurons shift between unstable firing and failure, apoptosis is promoted.

II. Microglia are primed and activated by spillage into extracellular environment, phagocytosis of debris begins, dysfunctional/inefficient neurons produce proinflammatory chemical signals, microglia are further activated and identify neurons themselves as targets.

III. Blood brain barrier is disrupted mechanically and chemical/physiological processes, brain’s environment is no longer sterile and WBCs enter.

IV. Dysafferentation is occurring via multiple mechanisms, so stimulation is altered via both receptor signaling (spinal and other joint/muscle mechanoreceptors, auditory, labyrinthine) and signal reception (central integration of signals is altered, changing the ability of neurons to tolerate signaling and producing abnormal perceptual and motor responses)

V. Genetic, innate expression is now responding to a pathological environment.

C. Concussion Recovery- Uncomplicated vs Complicated
   1. Uncomplicated occurs when injury is not severe AND person is very healthy AND lacks tendency for expression of neurodegeneration
   2. Complicated occurs when any of many factors interfere with recovery

D. MANAGING THE PATIENT WITH PERSISTANT POST CONCUSSION SYNDROME
   1. Be methodical & patient; minimize variables
   2. Look at labs
3. Investigate possible TBI
4. Use exam to identify neural circuitry of deficit
5. Establish objective measures
6. TREATMENT
   a. Focus on metabolic stability (fuel, hormones, etc)
   b. Go slow and get patient (family) commitment to the long term plan
   c. Modify ADL’s- food, screen time, sleep, environmental exposures
   d. Neuroprotective supplementation- plant phenol antioxidants
   e. Graded, rehab specific to patient- incorporate adjustments into this
7. Schedule formal re-evaluation using objective measures
8. Utilize other specialists to assist with areas listed above if needed.
9. Communicate
   a. At ROF be clear about expectations
   b. Provide hurdles early on
   c. Risk of future issues
   d. Send initial and progress reports to other providers

REFERENCES:


