**Minor TBI and Concussion**

**Summary Handout**

* Epidemiology
	+ 2.5 Million traumatic brain injuries every year in USA.
		- 75-95% of these are “mild”
		- 166 deaths a day due to TBI
	+ In developed countries caused by:
		- Motor Vehicle Accidents (20-45%)
		- Falls (30-38%)
		- Recreational accidents (10%)
		- Assault (5-17%)
	+ Sports are large contributor to minor TBI
		- Estimated (not reported) 1.6-3.8 million sport related concussions every year
		- Likelihood of athlete in contact sport experiencing concussion: 20% per season
		- Increased frequency in:
			* Males aged 15-34
			* Lower socioeconomic status, lower cognitive function and history of substance abuse
* Minor Head Injury - Based on the GCS score 30 minutes after injury.
	+ GCS = 14-15 = Mild Head Injury
	+ Also cannot have LOC > 30 min or other focal neuralgic deficit
	+ GCS = 9 – 13 = Moderate Head Injury
	+ GCS < 9 = Severe Head Injury
* Concussion Definition
	+ Concussion specifically describes the pathophysiological state which causes ***symptoms*** involved in minor TBI. It usually has rapid-onset and is short lived impairment of serologic function.
		- i.e the clinical symptoms of a TBI
	+ Mild TBI is “traumatically induced physiological disruption of brain function”
	+ Any LOC, loss of memory or any alteration in mental state at time of incident
	+ Caused by more than just direct force to the head or neck
		- Quick head movement can be transmitted from large blow to body
		- Rapid onset of symptoms which resolves spontaneously
		- May worsen over minutes to hours
	+ Clinical symptoms due to ***functional changes*** rather than structural anomalies
		- Neuroimaging will be normal
		- If imaging obtained and have contusion and hemorrhage the primary diagnosis is no longer mild TBI
			* May still develop post-concussion syndrome
	+ Concussion previously graded based on presence and length of loss of consciousness
	+ This grading system is no more
	+ Brief loss of consciousness does not predict long term course or outcome
	+ Absence of LOC doesn't mean “milder” injury and cannot justify earlier return to play
* Concussion Pathophysiology
	+ Large blow (to head or body) causes rotational acceleration of brain which causes micro shearing forces at the neuronal level
	+ Forces cause:
	+ Neuronal depolarization
		- Opening of Na/K+channels leads to influx of sodium and efflux of potassium. Calcium dependent neurons release glutamate which further depolarizes neurons.
	+ Lactic acid accumulation
		- The large areas of depolarization as mentioned above require lots of ATP to move the ions through the Na/K pumps. Increase in glycolysis locally causes consumption of glucose and therefore release of lactic acid.
	+ Decreased cerebral blood flow
		- Areas affected by the trauma have decreased blood flow. This is problematic as these ares also have consumed much of the glucose as mentioned above. This last days to weeks and contributes to pronged symptoms.
	+ Other theories exist and is not fully understood currently
* Sideline Assessment
	+ Various sideline tools exist to assess symptoms of concussion
	+ Standardized concussion assessment (SAC)
	+ Sport Concussion Assessment Tool (SCAT)
	+ Lower scores associated with concussion **but no cutoff has been established for diagnosis**
	+ **Clinical diagnosis** with no gold standard
	+ These tools are not mandatory but can provide valuable information for follow-up
	+ Some argue there should be pre-season baseline testing performed as:
		- 21-47% of males with psych or migraines and 33-72% of girls with history of psych, ADHD or trauma would score high enough to be considered concussed
* ED Assessment
	+ Evaluate for other injuries
	+ Consider imaging following usual decision rules - Canadian or PECARN
	+ Routine imaging is not indicated
		- CT or MRI won’t show evidence of concussive brain injury
		- Only obtain if concerned about other structural intracranial injury
	+ Signs and symptoms of concussion:
		- May be no symptoms at time of presentation
		- Confusion
		- Blank stare
		- Emotional liability
		- Poor focus
		- Delayed verbal responses
	+ Don’t forget to examine other causes of AMS; even in setting of head trauma
		- Dehydration
		- Hypoglycemia
		- Migraine
		- Psyciatric Disorders
* Early Complications of Concussion
	+ Seizure
		- Post-traumatic seizure possible in < 5%
		- Reflects changes from the trauma rather than epilepsy
		- Most common in fist 24 hours post injury
		- Nonetheless at higher risk of developing epilepsy in future
		- Seizure prophylaxis not warranted
		- Higher risk if have other intracranial pathology
	+ Unless some other indication as mentioned previously or there is suspicion for other intracranial pathology, no need to routinely image concussion - although may consider in case of first time seizure even in post traumatic setting
* Natural Course of Concussion - Pediatrics
	+ Pediatric EM Concussion Team study
		- Prospective cohort of 2700 children from 9 EDs in Canada
			* Interested in determining symptom resolution
		- Youngest children recovered first (5-7 years)
			* 80% symptom free at 2 weeks; most in 1 wk.
		- 8-12 years old 80% symptom free at 4 weeks
		- 13-18 year old boys 50% no symptoms at 4 weeks
			* In this age group only less than 50% of girls had resolution of symptoms by 12 weeks
		- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6583432/>
* Natural Course of Concussion - Adults
	+ Data on adults not as well understood
		- Studies which do exist show peak of post-concussive symptoms 7-10 days
		- 50% achieve resolution around 4 weeks
		- Most cases completely resolved in three months
		- Some patients have symptoms for >1 year
		- <https://pubmed.ncbi.nlm.nih.gov/22496195/>
* Concussion Recovery
	+ “Rest is best” - No longer the recommendation
		- Initial 48 hour period of rest followed by return to light activity
	+ RCT in kids looked at light aerobic activity after 48 hours of rest
		- Those in aerobic group recovered 4 days faster as compared control group (stretching)
		- This does not mean return to sport play
* Return to learn! - Returning to school should be prioritized in children
	+ Slow return - based on symptoms should occur outside initial 48h period of rest
	+ Rest beyond 48h has shown no benefit and may prolong course
		- 48 hours of cognitive rest versus 5 days showed worse outcomes and prolonged recovery in those resting for 5 days.
	+ Screen time - prohibited in first 24-48 hours
* Return to Play
	+ Consensus Statement on Concussion in Sport from 2012
		- Graded return to activity with escalating levels of activity and contact
		- If have any symptoms, return to prior level of activity where symptoms did not exist
		- Attempt progression every 24 hours
	+ The idea with returning to athletic play is that an athlete should be symptom free by time they return to full contact and activity. That being said the progression to this full activity should take time and occur after initial period of 24-48 hours of rest. This allows the time for brain to “rest” and allow for identification of symptoms which may peak around day 7-10 but allow for quicker progression if an athlete is not experiencing symptoms.
		- https://pubmed.ncbi.nlm.nih.gov/23479479/
	+ Return to Play - Children
		- Based on these protocols there is a **minimum of five days**
		- For children and adolescents it is recommended and some professional organizations say it should be ***required*** a child see healthcare professional before returning to play.
			* This can be a primary care, athletic trainer, neurosurgeon or any other health care professional who is well versed in post concussive symptoms.
			* For adolescent athletes with prolonged symptom course or who have had more than one concussion, some experts recommend staying at each stage for 1-2 weeks before progression.
		- In children with prolonged symptoms or who have had more than one concussion the RTP time may be weeks - months
		- Recovery goals should be tailored to the unique needs of patient
* Second Impact Syndrome
	+ Feared complication with second brain injury while in recovery from first
		- The reason the guidelines are conservative, especially with children is the feared complication of second impact syndrome. With repeat head injury in the recently concussed athlete the brain has lost much of its auto-regulatory functions in regards to intracranial and cerebral perfusion pressure. The metabolic changes last up to 10 days after the initial injury. With a second impact death can occur within 2-5 minutes and causes death much faster than those from an acute epidural hematoma. Because brain swelling is more significant in children initially as compared to adults this is why experts are more conservative in children returning to play.
			* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2672291/
	+ Metabolic changes from first injury disrupt auto-regulatory mechanisms
		- Potential for diffuse swelling, brain herniation and death
		- Death occur within a few minutes
	+ Treat like other cases of cerebral edema
		- Hypertonic saline and intubation for airway protection