Post Concussion Syndrome: Cortisol, Allostatic Load, and “Neurologic Exhaustion.”

Alternate Title: Post Concussion Syndrome “It’s all in their head”

Dr Kristian Goulet
Assistant Professor University of Ottawa
Medical Director: CHEO Concussion Clinic, the Pediatric Sports Medicine Clinic of Ottawa and the Eastern Ontario Concussion Clinic
“Disclosures”

• Received payment from Nyco Med, Purdue and Jannssen

• This “message” is about the relationship between Mild Traumatic Brain Injury (concussion) in *children* and PCS not:
  – Moderate to Severe Brain Injury
  – Second impact syndrome
  – Chronic Traumatic Encephalopathy

• This presentation used to be 220 slides
• I could also make a presentation with 220 slides explaining why PCS is entirely due to pure organic contributions.
• I use an incredible amount of analogies
Objective

1. To quickly describe what a concussion is at the clinical and pathophysiological level (Dispel the Great Concussion Mystery)
2. Explain why this is distinct from, and only indirectly related to, Post Concussion Syndrome (PCS)
3. Outline what I believe is the true etiology of PCS
   - 8 yrs and 2500 concussions to realize Concussions \( \Rightarrow \) Post Concussion Syndrome
4. To propose an informal algorithm for working up patients with PCS
5. Explore possible treatment options
6. Not confuse, anger or bore anyone
What is a Concussion?

• Concussion is defined as
  ➢ “A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces”
  ➢ My Definition: Any mental status change following trauma that cannot be attributed to extracranial factors or a preexisting condition

1. Short-lived.
   – Indeed MOST patients are symptom-free within 10 days.

2. Resolution typically follows a sequential course; symptoms may be prolonged.
   ✓ +/- LOC

3. Functional disturbance NOT a structural injury.

4. 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.

5. Due to contact and/or acceleration/deceleration forces of the head.

Concussion vs Post Concussion Syndrome

- Amnesia
- Disorientation
- Appearing dazed
- Acting confused
- Memory Issues
- Emotional Liability
- Physical incoordination
- Seizure
- Slow verbal responses
- Personality changes
- Hallucinations
- Drowsiness
- Sadness
- Headache
- Dizziness
- Nausea or vomiting
- Vision changes
- Photophobia
- Phonophobia
- Feeling “out of it”
- Difficulty concentrating
- Tinnitus
- Nothing at all initially

Incidence? Waljas et al 2014 found 31% of healthy controls met criteria for PCS
Iverson 2006: 90% of patients with depression met self-report criteria for a PCS
Symptom Etiology Changes Over Time

A great study by McNally et al 2013 concluded:

- Injury characteristics predict PCS in the first month following mild TBI but show a decreasing contribution over time.

- In contrast, noninjury factors are more consistently related to persistent PCS at 3 months.

- Psychological and psychiatric symptoms tend to evolve over time, often weeks to months.

- Anxiety and affective symptoms may complicate the clinical picture if the initial symptoms do not rapidly dissipate.

- Irritability, insomnia and worry complicate and amplify pain (headache).

Macleod 2010 state that:

- Rarely are the organic factors persistent
  - they may increase in intensity if accompanied by psychiatric morbidity.

- Incorrect diagnosis, fears of brain damage and over-solicitous medical attention may cause iatrogenic persistence of symptoms.

- Psychosocial factors amplify and maintain symptomatology, which may still retain a pseudoneurological signature.

Lishman (1988) also propose that organic factors are chiefly relevant in the earlier stages, whereas long-continued symptoms are largely due to “neurotic developments”.


- CHRONIC ILLNESS
Who gets PCS (There is no G Threshold)

- Stress/anxiety
- **High Achieving Teenagers**
- Women
- Those who live alone
- Smart/Not smart
- The Young/The Old
- **Those who get injured**
- Minorities
- Those with litigation involved (McNally et al 2013)

- MacLeod et al suggested
  - Physiological changes in the brain due to the *initial direct or indirect effects*
  - Depression due to the initial direct or indirect effects of concussion
    - Depression due to other factors
  - Chronic pain
  - Sleep disturbance and fatigue
  - Anxiety (generalized or specific or trauma-related)
  - Life stress, family and marital stress, and other psychosocial problems
  - Litigation stress
  - Numerous neurological conditions
  - Numerous psychiatric conditions
  - Malingering
“Concussion Cycle” & Stress/Cortisol Effect and Neurologic Exhaustion

My argument is that it is not the aforementioned chemicals that cause PCS but something else that perpetuates the symptoms:

Injury, Depression, Anxiety, Sleep, Pain, Organic cause, Healthy Patient, Illness, SEIDS, Cortisol Effect

- **Concussion**
  - Headache, trouble concentration, sleep issues, missed school, missed sport

- **Leading to Neurochemical Alterations and “Vital Exhaustion”**
Allostatic Load and Neurologic Exhaustion
Things At Play

1. Stress/Cortisol Response in Goulet Terms:
   I. My 8 year old self
   II. My Medical School Career
   III. Last Thursday Night

2. Empty Gas tank (unrefreshing sleep) “Duress”

3. Overtraining syndrome-use of antidepressants/Try and turn the table on stress

4. Prehistoric times: Prolonged levels of stress drives down serotonin/dopamine-
   1. phono/photo

5. Allostasis and Psychoneuroimmunology study of brain-behavior-immune interrelationships-
   microglial
Determining the Potential Contributors to PCS

- Organic/“Physiogenesis”
  - Persistent Neurometabolic Anomaly ie “concussion”
  - Actual Brain Damage(?)
  - VestibuloOccular Reflex Dysfunction
  - Neck Injury
  - TMJ
  - Neuroendocrine Dysfunction/Hormonal
  - Autonomic Dysfunction
  - Sleep
  - Infectious
  - Autoimmune

- Psychologic/“Psychogenesis”
  - Anxiety-Flight or Flight
  - Depression
  - Stress/Duress
  - PTSD
  - Iatrogenic
  - Premorbid Psych Functioning
  - Malingering
  - Coping Strategies
  - Support Structures
If All “Organic” Causes are Ruled Out, One Must Then Equally Rule Out the Psychologic Contribution.

- It's hard to treat someone who is depressed/stressed who doesn’t believe it (or who’s parent doesn’t believe it)
  - *that is where the ALLOSTATIC EXPLANATION WORKS SO WELL*
Overtraining Syndrome, SEIDS, Fibromyalgia and PCS

- I see many similarities between PCS and Overtraining Syndrome and SEIDS (IL-6)
- In both conditions the body/nervous systems are exhausted and then the brain and body both break down

Armstrong and VanHeest 2002 in their article The unknown mechanism of the overtraining syndrome: clues from depression and psychoneuroimmunology
  - There is no objective biomarker for OTS and the underlying mechanism is unknown
  - that OTS and clinical depression involve remarkably similar signs and symptoms, brain structures, neurotransmitters, endocrine pathways and immune responses.
  - Novel recommendations are proposed for the treatment of overtrained athletes with antidepressant medications, and guidelines are provided for psychological counselling.

Good Old Days Effect and How it Effects Incidence

Are some Post Concussive Patients actually already recovered?

- In a unique study Brooks et al 2014, they propose that some patients with post concussive symptoms overestimate their premorbid state.
  - This results in patients believing they are not recovered when in fact that have reached baseline measures.
- The "good old days" bias is present in pediatric mTBI by 1-month post-injury, influences retrospective symptom reporting, and has measureable implications for determining recovery in research and clinical practice.
- These other studies showed similar finding about attrition and overestimating previous premorbid state.
Attribution/Expectation of Symptoms

- The tendency to attribute common current symptoms to a past concussion has been called ‘expectation as aetiology’ and was first coined by Mittenberg et al. 1992
  - The individuals with head injury reported 60% fewer symptoms pre-injury relative to the base rate in the healthy controls, AGAIN overestimating their premorbid state

- Mittenberg et al. proposed
  - “the expectation of symptoms after head injury results in the misattribution of common symptoms and complaints to the injury and minimization of preinjury symptoms and minimization of other aetiologies, such as stress.”

- Bleanger et al 2013 based on their study of 91 patients with mTBI found
  - “Those who attribute their symptoms to TBI are more likely to report greater symptom severity overall.
  - Taken together, knowledge, self-efficacy, and attributions contribute independently to PCS severity.

Education

- Wasldron et al 2015 found that educating undergrad students about a positive recovery may result in less symptom persistence.

- Nygren de Boussard et al found that early, reassuring educational information is beneficial after MTBI.

- Kemp et al 2013 found that the terminology used by clinicians can impact the recovery of concussions.

  
  
Education:

• Miller et al. found that a single psychoeducational session is a key factor in preventing or shortening PCS
  – Provide supportive reassurance
  – Educate about the symptoms of concussion,
  – Describe the expected recovery course
  – Emphasize the appropriate attribution of symptoms to benign aetiologies,
  – Explain the gradual resumption of activities can be highly effective in reducing symptoms.
It is Not the Injury But the Presence of Injury

- Meares et al. Mild traumatic brain injury does not predict acute postconcussion syndrome. (n 175)
  - Diagnosis of acute PCS was not specific to mTBI (mTBI 43.3%; controls 43.5%).
  - Pain was associated with acute PCS in mTBI.
  - Females were 3.33x more likely (95% CI 1.20 to 9.21).
  - The strongest effect for acute PCS was a previous affective or anxiety disorder (OR 5.76, 95% CI 2.19 to 15.0).
  - These results were confirmed at the 3 month mark

- Yeates et al. (2009) studied 2 subgroups of children,
  - 1 group of children orthopedic injury and the other of children with mild TBI,
  - They found that persistent PCS unrelated to brain insult

  - After 1 year, the prevalence of PCS is similar between children w MTBI and children w orthopedic injuries
  - Children with MTBI experience greater rates of psychiatric illness during the 3 years after their injury.

- Asarnow et al., 1995 and Bijur & Haslum 1995 studied behavioral outcomes after injury:
  - They found that there were not more behavioural problems in children with mTBI relative to children with other traumatic injuries.
Depression and PCS

• “Depression is the best example of a psychiatric condition that can seriously complicate our understanding of recovery following mild head injury”

• Prevalence of 13% -77% and is a risk factor for poor recovery1-6

• Maeres et al 2011 found that preinjury depression/anxiety and acute post-traumatic stress (at 5-days post-injury) were predictive of post-concussive symptoms at 3-months.

• mTBI Patients who are depressed report lower QoL, lower satisfaction with life and poor memory abilities.

• Depressed patients have more severe PCS compared to non-depressed TBI patients
  – headache, blurred vision, dizziness, and memory impairment8,9

• Similarly Hutchinson found that pre-existing psychosocial and/or learning disabilities resulted in significantly longer recovery times.

• A great recent review by Broshek et al 2015 demonstrates that pre-morbid and concurrent anxiety increases the risk PCS.
  – propose that neurobiological abnormalities may serve as a pre-disposition for the expression of affective distress following a traumatic brain injury (i.e. diathesis/stress model).

Depression and PCS

• In a review by Busch and Alpern 1998 they conclude that MTBI is the triggering event for a set of pathophysiological changes and a concomitant depressive episode in a vulnerable population.

• Directional relationship between depression and mtbi:

Vassallo et al found that remote psychiatric difficulties, individuals with mood, anxiety, and conduct disorders were 2.5, 1.6, and 1.7 times, respectively, more likely to sustain TBI

• Very interestingly, mTBI subjects have higher rates of depression, postconcussive syndrome and poor global outcome than those with more severe TBI10

• Uomoto and Esselman reported:
  – 95% of individuals that had sustained a MTBI reported chronic pain while only
  – 22% of those having suffered a moderate- to-severe TBI did so11

• Kumar found that preexisting depression is a risk factor for
  – poor affective/behavioral, cognitive, and mental health-related QOL outcomes at 3 months
Anxiety and Concussions:

- We already know that pre-existing Depression and Anxiety are the greatest predictors of PCS

- Bryant and colleagues studied 1,084 patients with mTBI for 1 yr. They found:
  - generalized anxiety disorder 13.4%, posttraumatic stress disorder 13.0%, agoraphobia 12.8%, social phobia 9.0%, OCD 4.0%.

Anxiety Sensitivity

- Is considered a personality trait
- fearful response ass with an individual’s own bodily sensations
- arises from the belief that these sensations are signs of impending harmful consequences (NOCEBO?)
- has been demonstrated to influence how individuals perceive pain following a concussion **
- Heightened anxiety sensitivity may contribute to how clinical populations, (especially mTBI), perceive their injuries. ***


Negative cognitive loop

- In this model, the initial concussion symptoms disrupt cognition
  - in vulnerable individuals, these transient symptoms can cause anxious and fearful reactions,
  - which can cause further cognitive disruption (in flight or flight mode)
- This alternating pattern of cognitive slips followed by anxiety which exacerbates cognitive disruption creating greater levels of anxiety can create a dysfunctional feedback loop.


When cognitive symptoms persist the individual may develop a “shaken sense of identity” \(^1\), Extra Motivated TEEN

Kay et al propose a model that psychological distress

- can create further cognitive compromise by suppressing attention, mental efficiency, learning and memory,
- this creates cognitive symptoms above and beyond those accounted for by the concussion.
- avoidance of anxiety provoking situations (school avoidance!),

The anxiety and avoidant behaviours may also lead to depression.

The psychological overlay accumulates and intensifies and may become more disabling than the initial injury.

Kay et al. also identified psychosocial factors that can affect recovery, including work or school demands, as well as pressure from teammates and coaches to return to competition.

**THE CONCUSSION CYCLE**


Anxiety & the “Evolutionary Faux Pas”

- The way I see it and explain it to child is that teenagers are an evolutionary “faux pas”
- This is one of the most stressful times in their lives and they have very little coping strategies in place

Sources of stress:
- School
- Work
- Career Aspirations
- College University
- Sport
- Very Little Autonomy
- Bullying
- Body Image
- Sexual Orientation
PTSD

- Carelson et al 2010 looked at 836 patients who had confirmed TBI,
  - 63.9% also had PTSD and 35.6% were given a diagnosis of an anxiety disorder other than PTSD.
- PTSD following mTBI has been linked to increased PCS *
- PTSD in children is driving the presence of pain, and not vice versa.**
- PTSD is strongly associated with CDH, suggesting that traumatic stress may lead to headache chronication.

- Sawyer et al 212 prospectively enrolled individuals within one week of mTBI who were hospitalized. Participants were assessed at baseline, 3, 6, and 12 months
  - Univariate regression revealed a significant ass btwn PTSD and worse recovery (p<.001).
  - Headache is more common in persons with PTSD
  - “Further research is needed to examine whether PTSD symptoms exacerbate headaches, or whether problematic headache symptoms exacerbate PTSD.”

---


---

A Longitudinal Study of Headache Trajectories in the Year Following Mild TBI: Relationship to PTSD Symptoms.


FEAR and PCS

- In an interesting study using a rat model Reger et al. induced head injury.
  - They found that injured rodents demonstrated increased fear conditioning and an over-generalization of learned fear to conditioned and novel conditions.
  - Flight or Fight?

- Broshek et al 2015 discuss the implication of fear of concussion in the context of sports related concussion.
  - fear of re-injury,
  - fear of being perceived as weak,
  - fear of losing or not achieving a desired role,
  - fear of isolation
  - loss of affiliation with the team
  - fear of losing financial stability (income or scholarship),
  - pressure from teammates coaches etc
- They contend that patients can be functionally disabled by their concern about their symptoms

THIS DRIVES THE PERPETUAL FIGHT FLIGHT RESPONSE

- Emotional Intelligence Goleman 2006

To Summarize Thus Far

- What I believe happens is that many children at this age often barely able to cope with all of those stressors
- They then have a head injury where they miss school (get stressed and socially isolated), have their sport taken away from them (depressed), and have to deal with constant pain (effecting mood, ability to concentrate etc)
  - ALLOSTATIC LOAD
- This becomes too much for them and they become psychologically/emotionally exhausted and neurochemical changes ensure
  - VITAL EXHAUSTION/NEUROLOGY EXHAUSTION
- This is the true source of many of their symptoms
- Not the Sodium Potassium Glutamate etc imbalance that we know a concussion to be
Dopamine: The Unification of the Organic and Psychologic

- Bales et al 2015 calls Dopamine the Gatekeeper of cognition
- DA is critical for a number of physiologic functions including hormone secretion, movement control, motivation, emotion, and cognitive processing (Jackson and Westlind-Danielsson, 1994; Floresco and Magyar, 2006).

- DAergic pathways in the CNS can be divided into two predominant systems:
  - (1) the nigrostriatal pathway and
  - (2) the mesocorticolimbic pathway projecting to the prefrontal cortex (PFC), hippocampus, amygdala, and nucleus accumbens (Alexander and Crutcher, 1990; Graybiel, 1990)

- DA receptors are abundantly expressed in brain areas known to be damaged after TBI, (prefrontal frontal cortex and striatum), which are important for cognitive function

- DAergic system dysfunction within the PFC has been strongly tied to attentional and cognitive symptoms associated with schizophrenia and attention deficit hyperactivity disorder (ADHD; Heilman et al., 1991; Tassin, 1992; Knable et al., 1997; Tanaka, 2006).

- Dopamine levels have been shown to fluctuate greatly with head injury (McIntosh et al., 1994).
  - Donnemiller et al. (2000) used single photon emission computed tomography (SPECT) to show that striatal DAT binding is decreased in patients 4–5 months after severe TBI, even in cases where no anatomical evidence of direct striatal injury exists

- The efficacy of DA receptor agonists suggests that TBI patients benefit from the promotion of central DAergic transmission.
  - This could be a sign that DA release is suppressed after injury, that DA uptake is over active, or some combination of the two.
  - Alternatively, it might be the case that DA activity remains normal after injury, but that basal DA activity is inadequate in the face of the injury-induced disruptions. Bales et al (Donnemiller et al., 2000).

As quoted by Persistent cognitive dysfunction after traumatic brain injury: A dopamine hypothesis James W. Balesa,b,c,d,e, Amy K. Wagnera,b,c,f, Anthony E. Klina,b,c,d,f,g, and C. Edward Dixona,b,c,e,f,* Neurosci Biobehav Rev. 2009 July ; 33(7): 981–1003.

- Persistent cognitive dysfunction after traumatic brain injury: A dopamine hypothesis James W. Balesa,b,c,d,e, Amy K. Wagnera,b,c,f, Anthony E. Klina,b,c,d,f,g, and C. Edward Dixona,b,c,e,f,* Neurosci Biobehav Rev. 2009 July ; 33(7): 981–1003.

- 10.1016/j.neubiorev.2009.03.011
Dopamine: PFC the Unification of the Organic and Psychologic

- The PFC and hippocampus, are essential for memory formation.
- Mild traumatic brain injury often involves damage to the prefrontal cortex due to shearing forces of the frontal regions against the skull.
  - Effects on the PFC include decreased glucose metabolism (Fontaine et al., 1999), changes in frontal lobe blood flow during memory tasks (Ricker et al., 2001), and hypoactivation with memory tasks (Sanchez-Carrion et al., 2008).
  - Gould et al 2014 and Kinsella et al 1988 hypothesizes anxiety from head injury is secondary to focal and diffuse injury that can perturb the inhibitory functioning of the prefrontal cortex and lead to over-activation of the amygdala and other subcortical limbic structures.
  - PTSD involves an exaggerated response of the amygdala-resulting in impaired regulation by the medial prefrontal cortex.
- Koenigs et al 2008 propose that the neurobiological basis of anxiety disorders may be due to a complex interaction between PFC and the amygdala
- TBI causes an imbalance in excitatory and inhibitory components of these circuits.

- In terms of Microglia and Dopamine...we know that microglial can cause both healing and damage to the brain. Its activity is increased by head injury and stress.
- In a study by Brew et al 2015 they found that dopamine can be protective to the developing brain as it was associated with decreased apoptosis, oxidative stress and decreased neuroinflammation in white matter and caudate nucleus

Hippocampus and Dopamine

• Hippocampus, and limbic structures have albeen shown to be sensitive to damage after TBI (Dixon et al., 1987; Lighthall, 1988; Lighthall et al., 1989, Stuss et al., 1985; Levin, 1990; Ponsford and Kinsella, 1992; McDowell et al., 1997).
  – The hippocampus, which is also critical for cognitive function does not have a high level of DA receptor expression, but is dependent on DA activity to modulate function
• Mild TBI models show diffuse white matter damage, cortical cell loss, and hippocampal cell loss (Hicks et al., 1993; Sanders et al., 2001),
  – DA receptors in the hippocampus are the physiologic basis for memory formation and consolidation (Li et al., 2003; Lemon and Manahan-Vaughan, 2006).
  – In experimental TBI the hippocampus is exquisitely sensitive to both acute apoptotic events and excitotoxicity (Kotapka et al., 1991; Hicks et al., 1993; Dietrich et al., 1994; Smith et al., 1994).
  – Damage to the hippocampus causes reproducible deficits in spatial and temporal memory processing (Buckley, 2005).
  – Bremmer et al. found a connection between reduced hippocampal volumes and in trauma-exposed populations.
• Sapolsky propose high levels of glucocorticoids can decrease hippocampal volume (STRESS RESPONSE).
• Levita et al. show decrease hippocampal may cause anxiety
• Decreased hippocampal volume has also been associated with depressive episodes, and increased volumes ass w symptom resolution

References:
- Verstes RP. Interactions among the medial prefrontal cortex, hippocampus and midline thalamus in emotional and cognitive processing in the rat. Neuroscience 2006;142:1-20.

Disentangling Mild Traumatic Brain Injury and Stress Reactions (NEJM) Richard A. Bryant, Ph.D.
Serotonin and Head Injury

- Vecht et al., 1975 found levels of dopamine and serotonin were reduced in the cerebrospinal fluid of patients with TBI.

- Mobayed M, Dinan TG found patients who were depressed following mTBI have blunted prolactin response to buspirone,
  - They propose there is altered serotonin function and this is what differentiates depressed vs non depressed TBI patient.

- More recently Booji 2015 looked at serotonin and head Injury
  - Serotonin plays an important role in the etiology of depression.
  - Serotonin is also crucial for brain development.
  - Animal studies have demonstrated that early disruptions in the serotonin system affect brain development and emotion regulation in later life.

- A plausible explanation is that environmental stressors reprogram the serotonin system through epigenetic processes by altering serotonin system gene expression.

- This in turn may affect brain development, including the hippocampus, a region with dense serotonergic innervations and important in stress-regulation.

- These results suggest that serotonin transporter methylation may be involved in physiological gene-environment interaction in the development of stress-related brain alterations.

- Booji L¹, Syzd M², Carballedo A², Frey EM³, Morris D¹, Dymov S², Vaisheva F², Ly V², Fahey C³, Meaney J³, Gill M³, Frodl T¹ DNA methylation of the serotonin transporter gene in peripheral cells and stress-related changes in hippocampal volume: a study in depressed patients and healthy controls. *PLoS One.* 2015 Mar 17;10(3).

Sleep from Rao et al 2015

• In a great review by Rao et al:
  – Sleep disturbances are common after TBI, and disrupt recovery.
  – Sleep disturbances can be secondary to brain injury or caused by secondary factors
    • medical comorbidities, pain, medication side effects, stress etc.
  – They identify sleep issues like insomnia, sleep apnea, hypersomnia, circadian rhythm sleep–wake cycle disorders and parasomnias

• Ouellet et al found 50% of TBI patients have been found to experience chronic insomnia.
  – This study also found Milder severity of injury, severe depressive symptoms, severe pain, and fatigue have been found to be predictors of insomnia

• In a more recent study Towns et al 2015 found
  – 92% of patients with mTBI reported poor sleep.
  – Sleep quality significantly accounted for the variance in PCS, \( p < 0.001 \),

• They Conclude
  – While sleep is associated with PCS severity, psychological distress is a more potent predictor

Studies by Ponsford et al 2012, Fogelberg et al 2012 and Mathis and Alvaro 2012 sleep problems after TBI have been linked to anxiety, depression, cognitive difficulties, and trouble with rehab.


Ouellet MC, Beaulieu-Bonneau S, Morin CM. Insomnia in patients with traumatic brain injury: frequency, characteristics, and risk factors. J Head Trauma Rehabil 2006; 21 (3) 169-182
The Concept of Resilience/Adaptation

- Resilience is defined as the ability to rebound and recover after insult.
  - It has been proposed that those with less resilience are more likely to develop PCS \(^1, 2\)
- Losoi et al found that: Resilience prevents fatigue after mtbi
- Merritt el al: 142 military: higher resilience reported fewer PCS and PTSD-related symptoms
- Sullivan et al 2015: perceived psychological resilience was the strongest predictor of PCS-like symptomatology
  - even more than a history of mtbi.

3. Sullivan KA, Edmed SL, Allan AC, Smith SS, Karlsson LJ. The role of psychological resilience and mTBI as predictors of postconcussional syndrome symptomatology. Rehabil Psychol. 2015 May;60(2):147-54
Coping Strategies and Cognition

• It has been postulated that a lack of coping skills or neural reserve influence the development of PCS in children (Dennis, Yeates, Taylor, & Fletcher, 2007)

• Woodrome et al. 2011 go as far as to say that
  – “interventions designed to prevent or ameliorate post-concussive symptoms should focus on identification of children with mild TBI who use maladaptive coping strategies”
  – They identify emotion-focused strategies and problem-focused disengagement as detrimental


Is PCS Due to The Pain?

- Uomoto and Esselman report that 95% of individuals that had sustained a MTBI reported chronic pain
  - only 22% of those having suffered a moderate- to-severe TBI did so
  - “These results underscore the high frequency of chronic pain problems in the mild head injury population and implicate the need for avoiding the mislabeling of symptoms such attentional deficits or psychological distress as attributable only to head injury sequelae in those with coexisting chronic pain.

- Recently Weyer et al 2013 in their study of chronic pain and PCS found that
  - high chronic pain exacerbates the emotional aspect of PCS.

- Taylor et al 2010 propose that physical discomfort or difficulties in adjusting to the effects of injury, are likely to contribute to PCS after mTBI.

- Pain is known to be associated with a variety of cognitive, emotional, and behavioral symptoms (Beaupre, De Guise, McKerral, 2012).

- Hart et al in their review report those with chronic pain have impairments on measures of attention, processing speed, and psychomotor speed were often present in the chronic pain

- Eccleston and Crombez 1999 propose pain may compete with other attentional demands, leading to difficulties with attention

---

Post Concussion Syndrome: Bringing it Home

• At the end of the day It starts with a concussion/organic injury but at some point (1 month/three month?) the influence changes-its just hard to tell when
• Perpetual flight or flight
• In large part its almost inconsequential as you cant treat a concussion anyway-you manage a concussion.
• You have to rule out all other causes and treat symptoms
• You can treat mood and optimize mental health, sleep and eating
• Anxiety stress, and pain issues cause the prolongation of symptoms and must be addressed
• What I believe is that one cannot separate mind from body and when one is “broken” the other will eventually break (Overtraining syndrome)

• Physical injury can lead to psychological injury which then in turn can lead back into physiologic injury.

• There is a strong tie between physical and psychological symptoms
  – the more one has of one, the more one has of the other (simon et al 1999)

Workup/Treatment

• All About the History
• Physical Exam
• At 4 weeks
  – Blood work
  – Ensure all possible physical contributors are dealt with
  – Get a sense of how full their plate is

• 3-6 months consider imaging looking for Chiari or evidence of previous brain bleed

• Starts with supplements
• Eating, sleeping, and drinking optimization
• Education
• Break the cycle
  – Elavil vs other headache treatments
  – Adjust the neurochemicals
    • Stimulants or Antidepressants
Treatment
MPH and TBI

Johansson B, Wentzel AP, Andréll P, Rönnbäck L, Mannheimer C.
➢ Methylphenidate can be a treatment option for long-term mental fatigue and cognitive impairment after a TBI.

➢ Methylphenidate decreased mental fatigue for subjects suffering a traumatic brain injury, the treatment is considered to be safe and is recommended, starting with a low dose.

Methylphenidate reduces mental fatigue and improves processing speed in persons suffered a traumatic brain injury.
Johansson B, Wentzel AP, Andréll P, Mannheimer C, Rönnbäck L.
➢ Methylphenidate was generally well-tolerated and it improved long-lasting mental fatigue and processing speed after traumatic brain injury.
"Now this is not the end. It is not even the beginning of the end, but it is, perhaps, the end of the beginning.” November 1942