CONCUSSION

DR WILLIAM A. EARDLEY - NEUROLOGY

DISCLOSURE

PERSONAL DISCLOSURE:

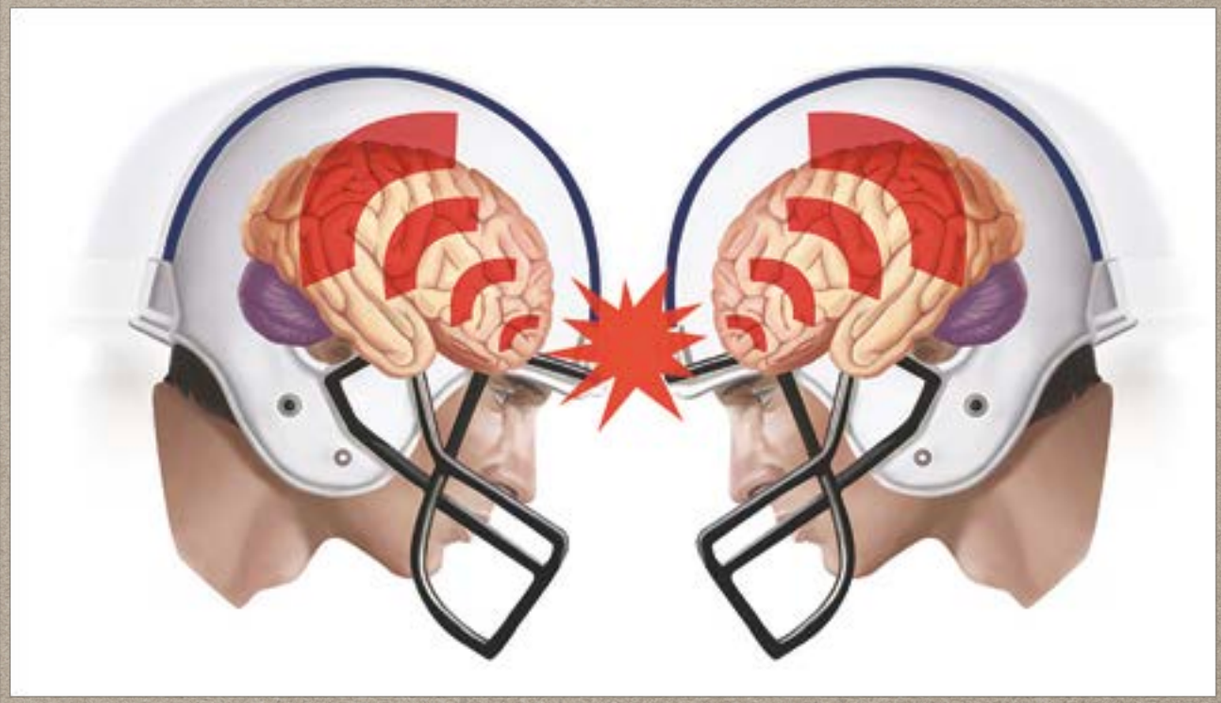
- I HAVE NO CURRENT OR PAST RELATIONSHIPS WITH COMMERCIAL ENTITIES.

COMMERCIAL SUPPORT DISCLOSURE:

- THIS LEARNING ACTIVITY HAS RECEIVED FINANCIAL SUPPORT FROM THE NANAIMO DIVISION, NANAIMO MEDICAL STAFF ENGAGEMENT SOCIETY, AND THE PRACTICE SUPPORT PROGRAM.

OBJECTIVES

- Discuss what a concussion is
- Discuss the physiology of a concussion
- Discuss persistent symptoms & post concussive syndrome



DEFINITION

- “Concussion denotes the acute neurophysiological event related to blunt impact or other mechanical energy applied to the head, neck or body (with transmitting forces to the brain), such as from sudden acceleration, deceleration or rotational forces.”
- Concussion is a clinical syndrome of neurocognitive or behavioral dysfunction resulting from a biomechanically induced alteration of brain physiology.

Diagnostic Criteria

Glasgow Coma Scale

RESPONSE	SCORE
Eye opening	
No eye opening	1
To pain	2
To speech	3
Spontaneously	4
Best verbal response	
None	1
Incomprehensible sounds	2
Inappropriate words	3
Patient confused	4
Patient oriented	5
Best motor response	
None	1
Extensor response to painful stimulus	2
Flexion to painful stimulus	3
Withdraws from pain	4
Localizes to pain stimulus	5
Obeys commands	6

Based upon: 1) loss of consciousness; 2) posttraumatic amnesia; 3) disorientation and confusion at initial assessment

- **Mild** TBI: <30 min LOC; <24 hours amnesia; GCS: 13-15 (not below 13 at 30 minutes)
- **Moderate** TBI: 30 minutes-24 hours LOC; 24 hours-7 days amnesia; GCS: 9-12
- **Severe TBI**: >24 hours LOC; >7 days amnesia; GCS: 3-8

DSM-V: 3 Part Test

First: Meet diagnostic criteria for“Mild Neurocognitive Disorder”

- A. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains based upon **both**:
    - 1 . Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function
    - 2. A modest impairment in cognitive performance, preferably **documented by standardized neuropsychological testing** or, in its absence, another **quantified clinical assessment**.
- B. The cognitive **deficits do not interfere with capacity for independence** in everyday activities
    - i.e., complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required.
- C. The cognitive deficits do not occur exclusively in the context of a delirium.
- D. The cognitive deficits are **not better explained by another mental disorder** (e.g., major depressive disorder, schizophrenia).

Second: There must be evidence of a traumatic brain injury

ONE OF THE FOLLOWING REQUIRED:

- 1. Loss of consciousness
- 2. Post-traumatic amnesia
- 3. Disorientation and confusion
- 4. Neurological signs (e.g. neuroimaging demonstrating injury; an new onset of seizures; a marked worsening of a pre-existing seizure disorder; visual field cuts; anosmia; hemiparesis)

Third: The neurocognitive disorder must present immediately after the occurrence of the traumatic brain injury or immediately after recovery of consciousness and persists past the acute post-injury period.

American Congress of Rehabilitation Medicine

- A patient with mild traumatic brain injury is a person who has had a traumatically induced **physiological disruption of brain function**, as manifested by at least one of the following:
  - Any period of loss of consciousness
  - Any loss of memory for events immediately before or after the accident
  - Any alteration in mental state at the time of the accident (e.g. feeling dazed, disoriented, or confused) and focal neurological deficits that may or may not be transient
- But where the severity of the injury does not exceed the following
- loss of consciousness of approximately 30 minutes or less
- after 30 minutes, an initial Glasgo Coma Scale of 13-15; and
- post traumatic amnesia not greater than 24 hours

American Academy of Neurology: Diagnosis by Degree of Certainty

Definite

- Concussion is the only explanation for the clinical presentation.
- Clear loss of consciousness from a witnessed biomechanical trauma
- In the absence of a clear loss of consciousness, the observation of posturing

Probable

- Concussion is the most likely cause of the clinical presentation.
- While other possible explanations exist, they are deemed less likely.
- The traumatic insult was clearly defined by witnesses or is identifiable on video.

Possible

- Concussion is not the most likely cause of the clinical presentation.
- Other possible explanations are identified, such as migraine, dehydration, or viral illness.
- The presumed traumatic insult was not witnessed or is difficult to describe.

SIGNS AND SYMPTOMS

- Patients often appear and behave similarly to those having an acute migraine headache or toxic-metabolic encephalopathy.
- Physical symptoms of imbalance and dizziness reminiscent of patients with central or peripheral causes of vertigo.
- Transient memory dysfunction similar to that seen in transient global amnesia.
- Mental status very similar to that seen in patients with a wide variety of mood disorders.

SYMPTOMS CAN ALSO BE CATEGORIZED INTO SUBCATEGORIES OF:

- Physical/somatic;
- Cognitive/mental status;
- Affective/behavioral and
- Sleep

PHYSICAL/SOMATIC

- Headache
- Nausea/vomiting
- Dizziness
- Incoordination
- Imbalance
- Photophobia
- Phonophobia
- Slurred speech
- Numbness/tingling
- Blurred vision/diplopia/flashing lights
- Tinnitus
- Weakness

COGNITIVE/MENTAL STATUS

- Amnesia
- Confusion
- Difficulty remembering
- Inattention
- Language difficulties
- Slowed thinking
- Disorientation
- Vacant stare
- Loss of consciousness
- Feeling “slowed down”
- Feeling “in a fog” or “dazed”
- Difficulty concentrating

AFFECTIVE/BEHAVIORAL

- Emotional lability
- Anxiety
- Mania
- Nervousness
- Sadness/depression
- Drowsiness
- Irritability
- Fatigue/lethargy

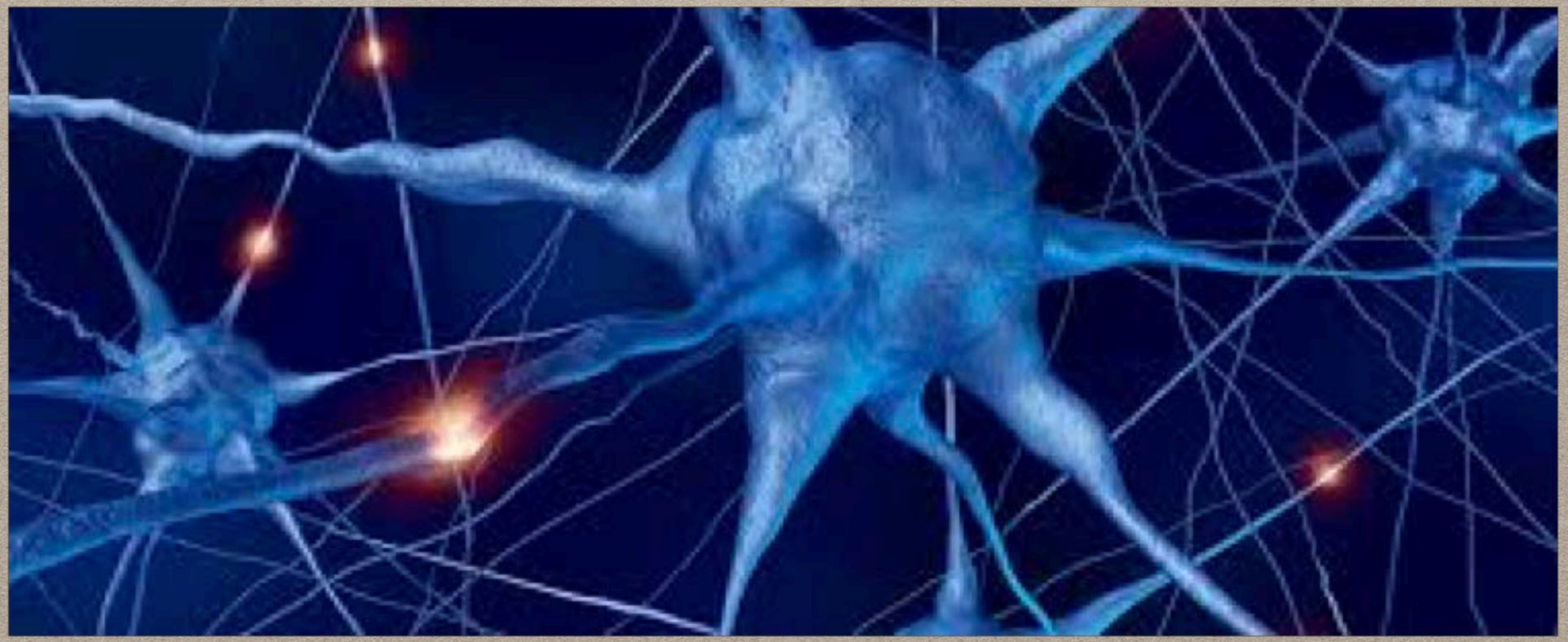
SLEEP

- Increased latency/trouble falling asleep
- Frequent waking
- Sleeping more than usual
- Sleep-related breathing disorder
- Circadian rhythm disorder
- Parasomnias

ACUTE PHYSICAL SIGNS ON EXAMINATION

- Impaired conscious state or brief loss of consciousness
- Confusion
- Vacant stare/glassy eyed
- Amnesia: retrograde or anterograde
- Slow to answer questions or follow directions
- Easily distracted/poor concentration
- Poor coordination or balance
- Unsteady gait
- Personality change
- Inappropriate emotion (laughing or crying)
- Slurred speech

PHYSIOLOGY OF CONCUSSION



- Concussion can be considered a diffuse pathologic process that produces brain network dysfunction.
- The diffuse nature of the injury produces clinical syndromes that often involve the disruption of functional brain networks, such as those responsible for memory, balance, and vestibular control.
- Impairment is due to functional rather than physical injury.

- The underlying pathophysiology of concussion centers on membrane leakage, ionic flux, indiscriminate glutamate release, and energy crisis.
- An indiscriminate release of potassium and influx of sodium and calcium occurs, generating a physiologic state resembling the spreading depression described in migraine.
- At the same time, depolarized neurons trigger a widespread release of glutamate.
- The membrane ionic pumps deplete intracellular energy creating an energy crisis.
- Simultaneously, an initial decrease in cerebral blood flow occurs. Uncoupling, which is a mismatch, worsens the metabolic dysfunction.
- Stretching of axons can also result in impaired transport and connectivity.


POST_CONCUSSION
SYNDROME
NEUROPSYCHOLOGICAL
POSTCONCUSSIVE

DELAYED SYMPTOMS

- Signs and symptoms of concussion may take several hours to develop or may not be noted by the patient until further cognitive or physical exertion.

POST CONCUSSIVE SYNDROME

- The majority of patients will have symptoms resolve within 1-3 months. Approximately 15% will have symptoms that persist past this time.
- It is debated whether PCS even exists as an entity. Symptoms associated with post-concussion syndrome are non-specific and common in healthy populations.
- For those that do believe it exists, there remains ongoing debate as to whether persistent symptoms are best attributed to biological or psychosocial factors.
- Individuals with PCS are often depressed, whether from neurologic insult related to the concussion or in reaction to cognitive and somatic changes resulting from the concussion.
- Prolonged postconcussive symptoms are highly associated with non-mTBI psychological factors
- Relevant definitions: ICD-10 and DSM-V

Diagnostic Criteria for Post-Concussion Syndrome (ICD-10)

Note: ICD-10 criteria approach prolonged postconcussive symptoms as a somatic symptom disorder (e.g., neuropsychological testing results may be normal, hypochondriasis may be present)

- A. History of **head trauma with loss of consciousness** preceding symptom onset by a **maximum of 4 weeks**.
- B. Symptoms in **3 or more of the following** symptom categories:

1. Complaints of unpleasant sensations and pains, such as headache, dizziness (usually lacking the features of true vertigo), general malaise and excessive fatigue, or noise intolerance.

2. Emotional changes, such as irritability, emotional lability, both easily provoked or exacerbated by emotional excitement or stress, or some degree of depression and/or anxiety.

3. Subjective complaints of difficulty in concentration and in performing mental tasks, and of memory complaints, without clear objective evidence (e.g., psychological tests) of marked impairment.

4. Insomnia.

5. Reduced tolerance to alcohol.

6. Preoccupation with the above symptoms and fear of permanent brain damage, to the extent of hypochondriacal, overvalued ideas, and adoption of a sick role.

DSM-V

- “Postconcussional disorder" eliminated in most recent update.
- Instructs psychiatrists to diagnose either major or mild neurocognitive disorder due to traumatic brain injury, depending on the extent of cognitive and functional deficit.
- The DSM-V criteria are oriented toward prolonged postconcussive symptoms as a neurological disorder (must have objective evidence of cognitive difficulties).

RISK FACTORS

PRE-DISPOSING RISK FACTORS

- Adolescents and elderly patients are at higher risk
- History of previous physical limitations
- Confounding effects of other health-related issues, e.g., pain medications, disabling effects of associated injuries, emotional distress
- Being a student
- Presence of life stressors at the time of the injury
- Lack of social supports
- Psychiatric history
- Past history of migraines
- Female gender
- History of prior concussions/head injuries
- Attentional, learning, or developmental disorders

PERI-INJURY FACTORS

Concussions resulting from car collisions, falls, assaults and sports injuries are commonly associated with post-concussion syndrome as compared against military personnel suffering similar head injuries.

POST-INJURY FACTORS

- Migraines
- Diagnosis of major depressive disorder or PTSD
- A low level of resilience
- Cognitive distortions such as the "good old days" bias
- Negative expectations molded by contextual factors after diagnosis ("nocebo effects")
- Litigation
- Not returning to work or significant delays in returning to work following the injury
- High number of symptoms reported early after injury
- Post-traumatic amnesia (PTA) or other memory problems after injury
- Early onset of pain and in particular headache within 24 hours after injury
- Reduced balance or dizziness during acute stage
- Presence of nausea after injury
- Skull fracture

DOES CAUSATION REALLY MATTER?

- Current guidelines recommend providing patients supportive treatments, regardless of whether their symptoms (headache, depression, etc.) is a primary result of head injury, secondarily caused, or was a pre-morbid condition worsened following injury.
- While lawyers will want clarity on causation, medically speaking - etiology only matters for those patients who may have to retire from a sport, or change other activities because of the likelihood of recurrent prolonged symptoms if future head injuries occur.

GENERAL RECOMMENDATIONS

- Try non-pharmacologic treatments first. Many medications used for one symptom may make others worse (i.e. opiates on overuse headache; antidepressants on sleep or concentration, hypnotics on memory, etc.).
- Psychosocial factors play a significant role in evolving or persistent symptoms. Some believe they are the sole cause. If symptoms are not improving within the first month patients should be referred on to a psychologist or psychiatrist for assessment and recommendations.
- If symptoms are worsening, it is important to rule out entities like subdural hematoma, dissection, etc.
- Most patients make a complete recovery despite what they do. Some take a bit longer, but overall, patients should be provided a positive prognosis.
- The days of “brain rest” are over. Patients should be encouraged to get back to their normal routines, including work and especially cardiovascular exercise, as soon as possible with few exceptions.


THANK
YOU

CLINICAL VIGNETTE

HEADACHE AND SLEEP DIFFICULTIES

PATIENT

43F was driving home from work when she had brought her vehicle to a stop. Just then she noticed headlights approaching in her rear view mirror. There was a sudden jolt and the back of her head struck the headrest. There was no loss of consciousness or amnesia. She exited her vehicle and noticed her bumper was pushed in, though otherwise her car was in driveable condition. The other driver immediately said her tail light did not work and a discussion ensued. They eventually exchanged insurance information. When the patient arrived home, she was very upset.

That evening, the patient started to feel increasing neck stiffness and her sleep was very interrupted. The next day, she visited her GP to discuss the accident. Her GP found cervical neck tenderness and diagnosed her with likely whiplash. She was prescribed naproxen and tramacet prn, which she began taking regularly. The next week, she began complaining of headaches, ongoing insomnia and worsening whole body pain. Her GP gave her a medical leave note for 1 week. The following week, she had no meaningful improvements and went to the ER where they arranged an xray of her neck which showed age expected degenerative changes and a CT head which was normal. She was prescribed 30 percocet for breakthrough pain and another note for 2 weeks off from work.

When the patient returns to see her GP, 2 weeks later, she relays how the ER doctor tells her that her "back is shot" and she suffered a concussion. In addition to headaches and insomnia, she is now also noticing worsened whole body pain, photophobia, memory problems, head fog, tinnitus, mood lability and balance difficulties.

…She does not think she will be able to return to work because of these symptoms.

APPROACH TO HEADACHE

- First step: Characteristics of headache: Severe, 15/10, pressure, holocephalic, photophobia, constant/no fluctautions, daily, no red flags, no exacerbating factors. Needing percocet, naproxen daily now "just to function".
- Questions:
  - Does this mimic a primary headache disorder?
  - Does this help to guide management?
  - What is the etiology? Is this post concussive syndrome?

SEMIOLOGY: NON-SPECIFIC

- Differential Diagnosis:
  - Headache attributed to trauma or injury to the head and/or neck
  - Headache attributed to psychiatric disorder, including somatoform disorder
  - Headache attributed to a substance or its withdrawal
  - Headache associated with sleep disorder
  - Headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structure
  - Headache attributed to cranial or cervical vascular disorder
  - Centralized pain disorder
  - Conversion disorder
  - Fibromyalgia
  - Malingering

APPLICATION

- Why not post concussive syndrome?
- Does differential help guide management? Yes. It means further assessments are necessary to rule out competing causes.
- It will be very difficult to discuss treatment options until the causes are better understood. It is not unusual for patients to have multiple contributing causes.
- Treatments may have uncertain benefit and side effects may be troublesome.

APPROACH TO INSOMNIA

- 3 P's
  - Predisposing factors
  - Precipitating factors
  - Perpetuating factors
- Management: Address factors. What is causing the awakenings? Does this patient require a sleep study?
- Cognitive behaviour therapy for insomnia.
- Short course of sedatives, doxepin and zolpidem. Possible role for gabapentin if coexisting anxiety and pain, but longer duration of action can cause intolerable side effects.


THANK
YOU