2015 Health Care Symposium

Current advances and trends in the diagnosis and treatment of MTBI and PTSD that result from traumatic injuries

May 28, 2015

AGENDA

- Simon Muller: Welcome
- Dr. Keegan: MTBI: Identification and Evaluation
- Dr. Moscovitch: MTBI and Sleep Disorders
- Dr. Debert: MTBI
- Dr. Pachet: Risk Factors for a Protracted Outcome after MTBI
- Dr. Mundin: Brain Structure and Function in PTSD
- Dr. Keiser: Current Clinical Concepts in Post-Concussion Syndrome and Treatment
- Jackie Halpern, Q.C.: Questions for the Panel
- Simon Muller: Closing Remarks
Mild Traumatic Brain Injury
Identification and Evaluation

John F. Keegan Ph.D.
Clinical Neuropsychologist

Mild Traumatic Brain Injury (MTBI)

Definition of Mild Traumatic Brain Injury
Pathophysiology
Diagnostic Process
Alternate Sources of Impairment
Recovery from Brain Injury
Recovery from Personal Injury
Definition of Mild Traumatic Brain Injury

- American Congress of Rehabilitation Medicine
- World Health Organization

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:

1. any period of loss of consciousness;
2. any loss of memory for events immediately before or after the accident;
3. any alteration in mental state at the time of the accident (e.g. feeling dazed, disoriented or confused); and
American Congress of Rehabilitation Medicine 2

4. focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 50 minutes or less;
- after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13/15; and
- posttraumatic amnesia (PTA) not greater than 24 hours.

World Health Organization Task Force

“MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury.”
Concussion:
Another Term for Mild Brain Injury

- Graded levels of concussion (Colorado Medical Society guidelines)
  - Grade 1
    - Transient confusion
    - No loss of consciousness
    - Mental status abnormalities resolve in less than 15 minutes
  - Grade 2
    - Transient confusion
    - No loss of consciousness
    - Mental status abnormalities last more than 15 minutes
  - Grade 3
    - Any loss of consciousness, brief or prolonged, seconds or minutes

Criteria for Diagnosis

Although an assessment of GCS score just after 30 minutes post-injury remains the ideal, our proposed definition permits diagnostic use of a GCS score assessed by a qualified healthcare provider at the first opportunity.
Mild Brain Injury Assumptions

- Tissue deformation likely insufficient to result in axonal shearing
- Secondary injury sequelae not likely present (e.g. anoxia, hemorrhage)
- No additional accident-related brain sequelae are present (e.g. fat emboli)
- Mild brain injuries are at one end of a continuum of brain injury, and thus methods are needed to clinically identify the likelihood that a particular head injury has resulted in a mild traumatic brain injury

Biomechanical Bases of Injury

- Tissue deformation consequential to acceleration/deceleration injuries
- Acceleration and deceleration result minimally in the stretching of selected axons
- Hemorrhagic contusions and axonal shearing are indicia of more severe brain injury, and need not occur in a mild brain injury
- Rotational injuries (chin free to rotate into chest) produce the most significant evidence of behavioral suppression, as demonstrated in baboons (Genarelli)
Normal Brain

Injury Simulation
Coup Contrecoup

Morphological Change
(i.e. Tissue Deformation)

- Does not necessarily result in axonal shearing
- The degree of tissue deformation and the rate of deformation determine the degree of axonal damage
- Axonal damage in the absence of shearing can result in destruction of the axon
- Destruction of the axon is evident with the classic formation of axonal swelling (i.e., retraction ball)
- Axonal damage probably occurs as a consequence of vulnerable axons which, when stretched, result in changes in axoplasmic transport
- Changes in axoplasmic transport ultimately result in destruction of the axon
- The degree of damage is a function of the number of axons destroyed, which is a consequence of the rate and degree of tissue deformation
Neuronal Shearing

Neuropsychological Methods

*History of case*

*Clinical interview*

Behavioral observations

Observations of significant others

Neuropsychological tests
Assessment Goals

Quantify impairments
Diagnose disease/syndrome
Measure process of change
Measure medication effects
Measure lesion effects
Determine ultimate impairment
Recommendations for care

Diagnostic Process
Define Brain Injury

- GCS<15
- EMS +
- Witness +
- Hospital Admitting +
- Imaging +
- Late self report +
- Early self report +

Brain Injury

Possible Brain Injury

No Brain Injury

History of Case

- Academic records
- Work performance evaluations
- Examinations for discovery
- Medical records
- Patient interview
- Questionnaires from significant others
Clinical Interview

- Determination of affect and mood
  - evaluation of language

- Evaluation of thought process
  - evaluation of defenses

- Evaluation of level of cooperation

Neuropsychological Tests

- Malingering
- Arousal
- Processing Speed
- Attention
- Sensory Functions
- Language
- Memory
- Spatial Abilities

- Abstract Reasoning
- Motoric Functions
- Integrative functions
- General Intellect
- Academic Level
- Emotional States
- Emotional Traits
Observations

Behavioral Observations
- observations of neuropsychologist
- observations of neuropsychological technician

Observations of Significant Others
- Home - Work
- Recreation - Surveillance

Impairment 1

Neurologically Based
- consistent test results
- proportionate to medical records
- confounds absent
Impairment 2

Non-Neurologically Based
- inconsistent test results
- disproportionate to medical records
- confounds present

Sources of Non-Neurologically Based Impairment 1

Physically Based Confounds
- medication effects
- acute or chronic pain
- unrelated issues
Sources of Non-Neurologically Based Impairment 2

Psychologically Based Confounds
- emotional states
- emotional traits
- malingering

Malingering

Conscious act of deception

Motive for deception unimportant

Normative vs. chance level performance
Malingering of Psychopathology

- Rare Symptoms
- Indiscriminate item endorsement
- Blatant symptoms
- Improbable symptoms

Malingering of Cognitive Impairment

- Symptom validity testing
- Magnitude of error
- Performance curve
Recovery from Brain Injury 1

- There is consistent evidence that adults experience symptoms, especially headache, in the acute stage and during the first month after MTBI. Although symptoms are common after MTBI, they are not unique to this type of injury since they are also evident in chronic pain patients, in other types of injuries and in healthy controls.

Recovery from Brain Injury 2

- The stronger studies, utilizing appropriate control groups and controlling for confounding factors, suggest that post-concussion symptoms are largely resolved within 3 months to a year. However, some individuals experience persistent symptoms after MTBI, and several studies have attempted to identify reasons for this. There is evidence that some of the observed longstanding post-concussion symptoms may be attributable to factors other than the MTBI. Studies that examine the relationship between litigation and/or compensation issues and slower recovery after MTBI consistently report an association between them.
Recovery from Personal Injury

Post-concussion symptoms should be assessed in the light of the background prevalence of these symptoms and with attention to other possible contributing factors. Few studies, for example, have adequately assessed the role of psychological distress and depression after an injury, medication effects or pain from associated injuries in the etiology of symptoms in the acute stage of MTBI.

Sleep Medicine and mTBI

By
Adam Moscovitch MD, FRCPC, DABSM, DABPN
Medical Director
Sleep & Fatigue Institute
Assoc. Clinical Professor
The University of Calgary
Background

- Diplomate, American Boards of Psychiatry & Neurology
- Diplomate, American Board of Sleep Medicine
- Diplomate, American Board of Forensic Medicine & Forensic examiners
- 1988: Fellow, Sleep Apnea Research Group, U. of Calgary
- 1989: Visiting scholar, Stanford Sleep Disorders Clinic & Research Center
- 1990-91: National Institute of Aging (NIA) Research Fellow, Stanford
- Clinical and Research focus on fatigue and its impact on workplace safety, including insurance and medico-legal consequences
- 2004: Outstanding Clinical Investigator, Dept. of Psychiatry, U of Calgary
- 1999-2005 Principal investigator, NAFMP for Commercial motor vehicle transportation; Expert Panel member on review of HOS regulations, Transport Canada. Consultant, US Departments of Transportation, Justice
- Over 600 IMEs, predominantly on complex insurance/legal cases of overlap between Pain, Sleep, Psychiatric conditions, Fibromyalgia/CFS

Disclosure

<table>
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<tr>
<th>Grants/research support:</th>
<th>Neurocrine Biosciences, Wyeth, Novartis, Bristol-Myers Squibb, Smith Kline, Pfizer, Neuroscience Pharma, Merck Frost, Takeda, Hoffman-La Roche, Vella, Lundbeck, Aventis, Organon, Servier, Sanofi-Aventis, Cephalon, Draxis, Eli-Lilly, Janssen, Sepracor, Otsuka</th>
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<td>Speaker’s bureau/honoraria:</td>
<td>Pfizer, Bristol Myers Squibb, Valeant, Eli Lilly, Paladin</td>
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Why does TBI cause sleep disturbance?

- **Acute phase of TBI**
  - Disturbances of sleep are thought to be due to the biological changes in the intracranial environment

- **Chronic phase of TBI**
  - Disturbances of sleep are believed to be also related to the psychiatric and psychosocial stressors, and chronic pain in this patient population

Sleep problems in context

- TBI
- mTBI
- INJURY
- Psychiatric Factors (Depression, Anxiety, PTSD)
- Pain
TBI and Sleep

• Following TBI, 36 – 70 % of patients will develop a sleep disturbance
• The impact of TBI on the sleep-wake cycle is related to three factors:
  – The injury itself
  – The neuropsychiatric conditions associated with TBI
  – The pharmacologic management of the injury

TBI and Sleep

• Sleep disorders play a large role in the ability of patients to recover from TBI, and are a negative prognostic indicators, if not addressed
Potential causes of sleep-wake disturbance following TBI

**Pathophysiological factors**
- Structural damage to structures important for sleep
- Hormonal and neurotransmitter alterations (reduced Hypocretin levels)
- Pain
- Medications

**Psycho-social factors**
- Environment (e.g. hospital)
- Stress
- Poor sleep Hygiene
- Thoughts, attitudes, beliefs

---

**Picture of Normal Sleep**

8-hour EEG recording in a healthy person

Hypnogram in a Patient

Interaction of Circadian Rhythm and Sleep
Spectrum of most common sleep-wake disturbances seen following TBI

- Insomnias
- Disorders causing Excessive Daytime Sleepiness
  - Post-traumatic hypersomnia
  - Sleep apnea (Obstructive or Central)
  - Narcolepsy
- Circadian Rhythm Sleep Disorders
- Fatigue

Potential impact of sleep-wake disturbances

- Insomnia 30-40%
- Fatigue 30-75%
- Sleepiness 14-55%

- Concentration
- Memory
- Attention
- Vigilance
- Pain
- Irritability
- Anxiety
- Depression

Rehabilitation
Return to work
Quality of life
**Insomnia**

- Difficulty initiating or maintaining sleep, early morning awakening and non refreshing sleep
- Following TBI, early and middle insomnias are very common

**Insomnia in mild to severe TBI**

- Moderate to severe insomnia
- Present 5.7 nights/week
- Appears within a few weeks after accident
- Average duration: 6 years
- 41.6 % receiving treatment

Ouellet, Beaulieu-Bonneau, & Morin (2006) *Journal of Head Trauma Rehabilitation*

Average time since injury: 7.8 years
N=452
Hypersomnia

- Excessive sleepiness with either prolonged sleep episodes or daytime sleep episodes
- Hypersomnia is more commonly associated with the chronic phase of TBI
- A major cause of decreased productivity following TBI

Differentiating fatigue from sleepiness

<table>
<thead>
<tr>
<th>FATIGUE</th>
<th>EXCESSIVE DAYTIME SLEEPINESS</th>
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<tr>
<td>Subjective feeling of weariness, depleted energy</td>
<td>Physiological drive to sleep</td>
</tr>
<tr>
<td>Multidimensional (e.g. mental, physical)</td>
<td>Measurable signs:</td>
</tr>
<tr>
<td>No objective measure</td>
<td>Yawning</td>
</tr>
<tr>
<td></td>
<td>Eyes drooping</td>
</tr>
<tr>
<td></td>
<td>Reduced alertness</td>
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<td>Can be measured in a sleep laboratory (MSLT)</td>
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Sleep Study Results
87 TBI subjects
Age 38 +/-15 years

- No primary disorder 47
  54%
- OSA 20
  23%
- PTH 10
  11%
- PLMS 6
  7%
- Narcolepsy 5
  6%

Sleep-Wake Schedule Disturbances

- The displacement of sleep from its original circadian pattern-Mismatch between sleep schedule and body clock
- Patients are unable to go to sleep or stay awake at a time they desire
- When they do fall asleep, the duration and pattern of sleep are normal
- Disturbance results from damage to the suprachiasmatic nucleus of the hypothalamus, aggravated by social factors
- Treated predominantly by Phototherapy
Additional references

• Ayalon et al “Circadian rhythm sleep disorders following mTBI” Neurology. 2007;68:1136-1140
• Schreiber et al “Long lasting sleep patterns in adult patients with mTBI and non mTBI subjects. Sleep Medicine. 2008;9:481-487
• Sullivan et al “Characterizing sleep reported sleep disturbance after mTBI” Neurotrauma. 2015;32(7) 474-486
• Huang et al “Sleep duration and sleep quality following acute mTBI: A propensity score analysis”. Behav. Neurology. 2015
Latest Advances in Mild Traumatic Brain Injury

McLeod Law LLP - 2015 Health Care Symposium

Chantel Debert MD MSc FRCPC CSCN
Physical Medicine and Rehabilitation

Take Home Messages

- COMPLETE REST is not best
- Look at the BIG PICTURE not just the head
- REPETITIVE mTBI may lead to long term sequelae

"It's a concussion, Son—you're sitting out the next siege."
Epidemiology

- mTBI ~600 per 100,000 population
- Calgary Data
  - 9456 head injuries in ER in 2013
  - ~20% revisit rate to the ER within 30 days
  - ~1200 mTBI were seen in the sport concussion clinic
  - ~2000 pediatric head injuries to ACH
- ~3 million annually
- Gross under estimation
- 5-9% of all sports related injuries

HOW & WHO?

YEARS

0 5 15 24 65
Does this patient have a mTBI?

American Congress of Rehabilitation Medicine:

- A traumatically induced physiological disruption of brain function resulting from the head being struck or striking an object or the brain undergoing an acceleration and deceleration movement, as manifested by at least one of the following: any period of loss of consciousness up to 30 min; post-traumatic amnesia not exceeding 24 h; any period of confusion or disorientation; transient neurological abnormalities, including focal signs, seizures, and intracranial lesions not requiring surgery; a GCS score of 13–15 (ie, ranging from confusion to normal consciousness on examination within 30 min after presentation).

Treating mTBI
Management

- Patients should **not** sit in a dark room and do nothing.
- 1-2 days of rest and then gradual return to previous mental and physical activity is recommended.


Management

- Initially:
  - Limit mental and physical activities that cause symptoms, but not complete rest
  - Sleep when needed
  - No ETOH
  - No sleeping medications, aspirin or anti-inflammatory medications
  - No driving until medically cleared
Prognosis

- **Sport Concussion**
  - 80-90% resolve within 7-14 days

- **Mild TBI**
  - Symptoms will usually resolve by 12 weeks
  - Pre-injury neuropsychiatric disorders are strong indicators of persistent post traumatic symptoms
  - Usually 1 month off work on average

Meares et al. 2011
McCrory et al. 2012
Levin et al. 2015

What if the patient does not improve within 1-3 months????
Educate, Educate, Educate

- Education on symptoms & positive outcomes
- Empower patient for self management
- Provide sleep hygiene information
- Teach tools for managing stress
- Encourage & closely monitor return to normal previous activities


Treat what you see...think past the head

MENTAL HEALTH

NEUROENDOCRINE DYSFUNCTION

COGNITION

HEADACHE

SLEEP DIFFICULTIES

DIZZINESS
If patients have not improved by 3 months post injury or is complex consider referring to a multidisciplinary PCS treatment program

In Calgary you can refer to the brain injury program. Referral letters and very good educational handouts can be found at:

http://www.albertahealthservices.ca/cbi.asp

What about patients with multiple head injuries...

Does your patient have chronic traumatic encephalopathy?
Chronic Traumatic Encephalopathy

- Dementia Puglistica or Punch Drunk Syndrome
- CTE was first described in boxers in 1928
- Currently a POST-MORTEM diagnosis

What is CTE?

- Progressive neurodegenerative disease
- Believed to be caused by repetitive trauma to brain including concussions and sub-concussive blows/unrecognized concussions.
- Symptoms begin most often years or decades after the brain trauma and usually continue to worsen.

What CTE is not....

- Prolonged post-concussion syndrome
- Not just cumulative effects of concussion
Diagnosis of CTE
Post Mortem

**CHRONIC TRAUMATIC ENCEPHALOPATHY**
Tau Protein: Amygdala (McKee et al. 2009)

Healthy Brain  Football Player  Boxer

Normal 65 yo male  45 yo Football player committed suicide  73 yo boxer with extreme dementia pugilistica  

Thank you!!!
Questions?
chantel.debert@albertahealthservices.ca
http://www.albertahealthservices.ca/cbi.asp
References
16. Silverberg, N. D. & Jerson, G. L. Is rest after concussion "the best medicine?": recommendations for activity resumption following concussion in athletes, civilians, and military service members. J. Head Trauma Rehabil. 28, 250–9
MTBI: Risk Factors for Protracted Recovery

Arlin Pachet, Ph.D, R.Psych., ABPP
Board Certified Clinical Neuropsychologist

May 28, 2015

Presentation Objectives

- MTBI defined
- Expected outcomes after MTBI
- Risk factors
MTBI Defined

- WHO definition
- ACRM
- Key pathognomonic indicator is mental status change
- GCS
- PTA
- RA

MTBI Recovery Expectations

- Take home message: MTBI does not equate to permanent cognitive impairment after uncomplicated MTBI
- Recovery is typically full and brisk in uncomplicated MTBI and usually complete within days to a few months at most.
MTBI: Common Persisting Complaints

- Headache
- Dizziness
- Cognitive slowing and cognitive inefficiency
- Irritability
- Fatigue
- Visual changes
- Depression and anxiety

Risk factors for protracted outcome after MTBI

- Injury variables: may predispose a MTBI client to more prolonged recovery and/or to persistent problems on a neurologic basis.

- Subject variables: may predispose a MTBI client to more prolonged symptomology on a psychological basis.
Risk factors for protracted outcome after MTBI

Injury Variables (brain injury variables)
- Persistent symptoms have been reported to increase with the presence of intracranial lesion.
- Prior brain injury: factors of cumulative effect and “brain reserve capacity”.
- Remote history of concussion? Does that matter?

Risk factors for protracted outcome after MTBI

Subject Variables (non-brain injury variables)
- Substance dependence
- Financial incentive
- Social difficulties
- Stress
- Pre-injury maladaptive coping
Risk factors for protracted outcome after MTBI

Subject Variables (non-brain injury variables)
- Psychiatric history
- Older age
- Pain
- Premorbid cognitive issues such as a neurodevelopmental disorder (e.g., LD and ADHD)
- Financial incentive
- Pre-injury maladaptive coping

Risk factors for protracted outcome after MTBI

Subject Variables (non-brain injury variables)
- Family support or lack there of
- Pre and post accident social difficulties and stress
- Pre-injury migraines
- Over-medicalization
- Genetics (e.g., APOE)?
Risk factors for protracted recovery

- Emergence and persistence of a cluster of symptoms following a MTBI
- Symptoms are not diagnostically specific

Contact Information

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Brain Structure and Function in PTSD

Prepared by:

Joann Mundin, MD, FRCPC, MSc
Psychiatrist

for presentation at:
Health Care Symposium 2015
Current advances and trends in the diagnosis and treatment of MTBI and PTSD that result from traumatic injuries
May 28, 2015

Disclosures
1. I have received honoraria from Otsuka
2. Affiliation with Caleo Health
3. Medicolegal work:
   - 60% defence
   - 40% plaintiff
4. Most of the images in these slides belong to other people. I use them when I teach, and give full credit to their rightful owners.
Background

1. Psychiatrist since 2003
2. Treating and non-treating practice
3. First love in psychiatry was emergency room (sick v. stressed)
4. At least 500 non-treating assessments since I started to keep track in 2011
5. Large component of my referrals ask about PTSD
6. I have assessed patients and examinees who have developed PTSD from a range of traumas, including sexual assault, catastrophic collisions, catastrophic non-collision accidents, workplace accidents in the oil patch and animal assault. I have assessed many examinees who believed they sustained PTSD, but actually did not. I have treated patients with PTSD. I have collaborated with psychologists in the treatment of PTSD.
History of PTSD

Image from World War I taken in an Australian dressing station near Ypres in 1917. The wounded soldier in the lower left of the photo has a dazed thousand-yard stare, a frequent manifestation of "shell-shock" or combat stress disorder.
Between 1916 and 2013, the concept of PTSD became diluted and inaccurate:

- Bullying is a form of psychological and emotional rape because of its intrusive and violational nature
- Prolonged Bullying can cause Complex Post Traumatic Stress Syndrome

Trauma that breaks the brain (not just the mind)

1. Exposure to war as a combatant or civilian
2. Threatened or actual physical assault (e.g., physical attack, robbery, mugging, childhood physical abuse)
3. Threatened or actual sexual violence (e.g., forced sexual penetration, alcohol/drug-facilitated sexual penetration, abusive sexual contact, noncontact sexual abuse, sex trafficking)
4. Being kidnapped, being taken hostage, terrorist attack, torture, incarceration as a prisoner of war, natural or human-made disasters
5. Severe motor vehicle accidents.
6. A life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events (e.g., waking during surgery, anaphylactic shock).
DSM-5 ONLY TALKS ABOUT THE MIND

FROM THE PERSPECTIVE OF THE MIND: THERE ARE 636,120 WAYS TO HAVE PTSD

636,120 Ways to Have Posttraumatic Stress Disorder.
Isaac R. Galatzer-Levy
Richard A. Bryant. Perspectives on Psychological Science November 2013 vol. 8 no. 6 651-662
THERE ARE YEARS STRONG EVIDENCE FROM THE BASIC SCIENTISTS THAT SHOWS THE EFFECTS OF STRESS ON THE BRAIN

PTSD is associated with major changes to the brain; This is not merely an emotional (mind) reaction to an event.

<table>
<thead>
<tr>
<th>Neurotransmitters</th>
<th>Effect</th>
</tr>
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<tbody>
<tr>
<td>Hypothalasium</td>
<td>almond-shaped and unsuitable response to stress</td>
</tr>
<tr>
<td>Hypothalasium-rotunda</td>
<td>abnormal stress encoding and fear processing</td>
</tr>
</tbody>
</table>

Image from: www.pixshark.com

Table 1. Summary of neurobiological features with identified abnormalities and functional implications in patients with post-traumatic stress disorder.

- CORT: corticotropin-releasing hormone
- CRH: corticotropin-releasing hormone
- ACTH: adrenocorticotropic hormone
- TSH: thyroid-stimulating hormone
- DHEA: dehydroepiandrosterone
- POMC: pro-opiomelanocortin
- CRH: corticotropin-releasing hormone

PTSD (AND MOST MENTAL ILLNESS) = BURNT OUT BRAIN CELLS

Images from: 1. biology.stackexchange.com 2. fashions-cloud.com
Severe stress leads to dendritic retraction

Front. Behav. Neurosci., 29 March 2012
Toward a limbic cortical inhibitory network: implications for hypothalamic-pituitary-adrenal responses following chronic stress

Image from: Neuroexia.com

Acosta et al Neuroinflammation after chronic stress and TBI. PLOS 1 December 2013 | Volume 8 | Issue 12
HIPPOCAMPUS IS WHERE IT'S AT.

Normal function of the hippocampus
Hippocampus shrinks in PTSD

As a result:
- Poor control of stress responses
- Poor control of memory
- Poor context for fear conditioning

THE PREFRONTAL CORTEX SHRINKS IN PTSD

Anterior cingulate cortex image from: mybrainnotes.com
Normal effects of ACG in the prefrontal cortex

- Regulation of blood pressure
- Regulation of heart rate
- Management of reward anticipation
- Decision making
- Empathy
- Impulse control
- Emotion

Amygdala gets dragged into this mess?

<table>
<thead>
<tr>
<th>Role of the amygdala</th>
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<tbody>
<tr>
<td>- Memory</td>
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<tr>
<td>- Decision making</td>
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<tr>
<td>- Emotional reactions</td>
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Image from: brainconnectino.com
THE TREATMENT OF PTSD MUST INVOLVE REGROWTH OF THE SHRUNKEN BRAIN CELLS

Are there any ways to regrow PTSD damaged brain cells?

1. Evidence based MEDICATION

2. Evidence based COUNSELLING:

3. Time!
Most people with PTSD will recover, but those who pursue treatment will recover faster.
PTSD and medication

1. Sertraline
2. Paroxetine
3. (Prazocin)

PTSD and counselling: CBT

- My opinion: CBT offers the highest yield in terms of counselling through the management of sleep interfering symptoms.


- This is a critical issue because of the effects that insomnia has on the health of brain cells:

Image from: webmd.com
Normal and disrupted sleep

Purple = brain cell garbage

This image from a mouse brain shows the fluid drained (purple) and glia cells (green) flushed out into the brain's vasculature.
CBT focussed on managing the stressors associated with PTSD

Images from: adapted-therapies.com and friendshipcircle.org
CBT might regrow broken brain cells

Increase in prefrontal cortical volume following cognitive behavioural therapy in patients with chronic fatigue syndrome

Floris P. de Lange, Anda Koers, Joke S. Kalkman, Gjis Bleijenberg, Peter Hagoort, Jos W. M. van der Meer, and Ivan Toni

In patients who are receiving treatment, the average duration of symptoms is approximately 36 months; in patients who are not receiving treatment, the average duration of symptoms rises to 64 months.


Time: Most people with PTSD will recover, but those who pursue treatment will recover faster.
BEWARE OF JUNK SCIENCE AND PARTIAL INFORMATION WHEN IT COMES TO PTSD

THERE ARE DECADES OF GOOD PROOF, SO WHY DON’T WE TALK MORE ABOUT THE BASIC SCIENCE OF PTSD?

www.genesinlife.org
1. Basic science is hard to translate in general
2. Basic science is hard to translate in specifics (cross examination)
3. The research techniques that could be used in humans are slowly coming into daily use.
4. Not every trauma causes PTSD

Translation of Basic Science

The estimated volume of OX6 positive cells was examined using Cavalieri estimator probe of the unbiased stereological cell technique [26] in analyzing the cortex, striatum, thalamus, cerebral peduncle, corpus callosum, and cerebellum areas such as white matter (WM), granular cell layer (GCL), and molecular layer (ML). Ki67 and DCX positive cells were counted within the subgranular zone (SGZ) in both hemispheres (ipsilateral and contralateral), using the optical fractionator probe of unbiased stereological cell counting technique. The sampling was optimized to count at least 300 cells per animal with error coefficients less than 0.07. Each counting frame (100 X 100 mm for OX6, Ki67, and DCX) was placed at an intersection of the lines forming a virtual grid (125 X 125 mm), which was randomly generated and placed by the software within the outlined structure.

Influence of Post-Traumatic Stress Disorder on Neuroinflammation and Cell Proliferation in a Rat Model of Traumatic Brain Injury

Acosta et al
MOST TRAUMAS WILL NOT CAUSE PTSD

Likelihood that a trauma will cause PTSD?

- According to a 2008 study, in Canada, 76 of 100 of us will encounter a trauma bad enough to place us at-risk for PTSD.

- However, only 9 in 100 of us might go on to develop PTSD in our lifetime. Those people who go on to get PTSD are often exposed to at least 4 or more traumatic events.

- The most common forms of trauma resulting in PTSD included:
  1. Unexpected death of a loved one
  2. Sexual assault
  3. Seeing someone badly injured or killed.

HOW CAN I IDENTIFY MY PATIENT/CLIENT WITH PTSD?

1. You must know the DSM-5
2. You must assess every item of the DSM-5
3. You must not introduce your bias to the assessment
4. You must not rely on symptom checklists.
DSM-5 terminology is the accepted language in the mental health community in Canada.

- Mental health experts should be communicating with the diagnostic language that is currently spoken by the majority of the mental health community in Canada.

- A mental health expert should identify instances where non-standard language has been used to describe a mental health condition. It is not always possible to translate non-standard data into currently accepted terminology.
Remember there are over 600,000 ways to have PTSD.

**ASSESS EVERY DIAGNOSTIC ITEM FOR PTSD IN THE DSM-5**

A PTSD expert report should be lengthy and detailed.

- A medical expert's assessments for PTSD should be lengthy because they have to consider every one of the DSM-5 diagnostic criteria.

- There are 636,120 ways to have PTSD. If your experts don’t look for all of them, the diagnosis could be missed. If a medical expert only looks at some of criteria, then a diagnosis could be stated where one does not exist.
DO NOT INTRODUCE YOUR BIAS INTO THE ASSESSMENT

"You have a very bad case of transference."

The person’s response involved intense fear, helplessness, or horror...

…no longer appears in the DSM-5.
This is important.
key points

1. The individuals who have PTSD need to be identified, because they have a brain disorder that needs medical treatment.

There is a subgroup of Canadian individuals (approximately 10%) who appear to be particularly vulnerable and develop full-syndromal PTSD. This disorder seems to have significant morbidity and impairment in social and occupational functioning and, along with its associated sequelae, is quite chronic in a large proportion of affected individuals. It is therefore paramount for Canadian primary care and mental health workers, as well as other front-line individuals, to become better equipped in recognizing and treating PTSD.
2. PTSD is treatable. There is evidence based treatment for PTSD.

- PTSD experts should be telling you which treatments are evidence-based, mainstream treatments, accepted in the wider mental health community and which ones are not.

- It is unfortunately too common that evidence-based treatment is not offered to patients with PTSD, for a variety of reasons.

3. MENTAL HEALTH INJURIES ARE OFTEN FORGOTTEN (UNDERSTANDABLY SO) IN THE INITIAL MANAGEMENT OF CATASTROPHIC PHYSICAL HEALTH INJURIES.

That is because of basic principles of triage in a life or limb threatening emergency.
Thank you for your interest!

Current Clinical Concepts in Concussion Diagnosis and Management

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Assistant Professor of Clinical Neurology, Carrick Institute

www.carrickinstitute.com  (321) 868-6464
The power of the brain is in its ability to COMMUNICATE
No one is impervious to the effects of a brain injury.
Routine concussion evaluations are incomplete

“Accumulating evidence indicates that cognitive testing should be viewed as one of several complementary tools necessary for a comprehensive assessment of concussion”

Ellemberg, 2009
Rotational accelerations are greatest predictor of injury

Rowson, Steven, et al. 2012
Poorer subconscious oculomotor function in the Post Concussive Syndrome (PCS) group supports the notion that 
**PCS is not merely a psychological entity, but also has a biological substrate.**

Heitger, 2009
Adaptation vs. Healing
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References

Neurophysiology of gait: from the spinal cord to the frontal lobe. Takakusaki K 2013

Central Processing

Motor Command

Sensory Input

Motor Output
Questions?
Thank You for attending the 2nd Annual Health Care Symposium!