Traumatic Brain Injury (TBI), Post-concussion Symptoms (PCS), and Psychiatric Conditions

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Epidemiology in US
- Incidence: 100-300 per 100,000 hospitalized (4-5 times this number treated as outpt)
- 70-90% of all hospital-treated TBI is Mild
- Age: 15-24 (peak range)
- Sex: 2:1 (peak for females: age >75 yrs)
- Costs: > $1 Billion per year

Current TBI Landscape
1. TBI a major public health problem worldwide
2. > 80% classified as MTBI
3. Young/old, male, minorities of low SES at risk
4. Nothing “mild” about the societal impact of MTBI
5. What works for moderate/severe may not for MTBI
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TBI: “Signature Injury” of Iraq War

For the first time in U.S. Military history, head injuries outnumber chest and abdominal wounds.

- **In-Theater**
  - ~ 20% of all deployed soldiers have TBI
  - ~75% of all blast injuries have TBI
  - >90% are nonpenetrating MTBI

- **Stateside**
  - >50% of all evac’s to WRAMC
  - 45% of all blasts Rx’d at WRAMC
  - 66% of all wounded who do not RTD have TBI

Huge volumes in VA system

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Challenges in Defining & Diagnosing TBI

- Classifying TBI severity an imperfect science
- Varied emphasis on acute injury characteristics
- Limitations of traditional methods (GCS)
- Limited reliability, validity, predictive power of classification methods
- Uncertainties remain regarding variables influencing functional, social, cognitive, and occupational outcomes

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Mild Brain Injury

- American Congress of Rehabilitation Medicine defines mTBI as:
  1. Traumatically induced disruption of brain function.
  2. LOC < 30 min or alteration of consciousness manifested by incomplete memory of event or being dazed and confused.
  3. PTA no longer than 24 hrs.
  4. May or may not have focal neurologic findings.

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Indicators of mTBI

- LOC = 0-30 min
- Post-Traumatic Amnesia (PTA) = minutes to 24 hrs
- Glasgow Coma Scale (GCS) = 13-15
- Acute Clinical presentation = appear stunned or dazed, drowsy or indifferent, disoriented, trouble with complex commands, HA, nausea, or vomit.

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Severity

- The American Academy of Neurology grading system:
  1. Grade 1: confusion w/o LOC, symptoms clear in 15 min.
  2. Grade 2: confusion w/o LOC, symptoms lasting longer than 15 min.
  3. Grade 3: LOC

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Biomechanics:

- “Theoretical” force is just that: theoretical
- Injury can only be defined by change in neurological status: “could have” does not equal “did”
- Some evidence to suggest that there is a threshold of mechanical force for injury, but:
- This is not a strong predictor of injury, and does not predict clinical outcome

Adopted from McCrea, 2010
**Pathophysiology**

- Generally considered diffuse.
- Any alteration in consciousness suggests widespread neuronal dysfunction. Neuronal damage has traditionally been described in terms of primary injury (e.g., contusion or axonal shearing) and of secondary injury (e.g., hyperglycolysis, acidosis).
- Animal models show axonal distortion and degeneration.
- Axonal changes typically evolve over 12 to 24 hours.
- Neuronal recovery can be seen over periods of weeks to 3 months, perhaps mirroring recovery processes.

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**Diffuse Axonal Injury - what is it?**

- Diffuse Axonal Injury (DAI) is a microscopic diagnosis—it cannot be made definitively in a living patient!
- Involves widespread axonal transection and neuronal degeneration.
- In grades II-III, macroscopic lesions may be evident, but it is unclear if macroscopic lesions (corpus callosum, brainstem) imply DAI.
- DAI is not obligatory even in fatal TBI.
- Adams et al., 1999: 26% (N=434)
- Bennet et al., 1995: 42% (N=100)
- If less than 50% of pts who eventually die from severe TBI have DAI, what % might be expected in mTBI?
  Adapted from Alexander (1995)

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**Neuropathophysiology - take home**

- In vivo work essentially all from animal models, human brains may be different.
- Little evidence of significant permanent cell loss.
- Evidence of disrupted homeostasis—interestingly, this follows a time pattern and course similar to that observed in clinical recovery of concussed patients.

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Cognitive Sequelae

- Short-Term effects (within 1 week): processing speed, attention, reaction time, and memory.
- Asymptomatic individuals without LOC can show impaired processing speed.
- Rapid recovery course; usual recovery course is 1-3 months.
- If persistent tend to be in domains of memory, attention, and processing speed.
- Complications such as: skull fx, contusions, hematomas, litigation, and mood/psych issues. More likely to lead to persistent cognitive problems (processing speed, verbal and recognition memory, and verbal fluency).

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Cognitive Sequelae (cont.)

- The long-term effects of mTBI remains a controversial area.
- Clear association with mTBI and increased rates of depression, anxiety, and PTSD have been found.
- Presence of these d/o serve to accentuate or increase degree of distress associated with lingering symptoms.
- Successful treatment of co-morbid conditions can result in significant reduction of post-injury complaints.

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Behavioral Sequelae

- Two broad forms:
  1. Acute Neuropsychiatric as part of natural course.
  2. Increased vulnerability to psychiatric d/o during subsequent recovery.
- Postconcussive Symptoms = constellation of symptoms experienced subsequent to brain injury.

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PCS

- The most common PCS can be grouped into three categories.
  1. Cognitive (decreased memory, attention, and concentration).
  2. Somatic (HA, fatigue, insomnia, dizziness, tinnitus, and sensitivity to light and noise).
  3. Affective (depression, irritability, and anxiety).

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Word of Caution

- It is not clear if there is a postconcussive “syndrome” per se.
- Common symptoms that occur in a given individual as a function of his or her particular injury and relevant premorbid factors.
- Subjective complaints in several domains are common, it is not clear that it is helpful to conceptualize the sequelae of TBI as a syndrome, as it may send one down the wrong treatment path.

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PCS

- If one considers the multiple symptoms to be a syndrome with a common underlying mechanism (neural damage, depression, malingering), one tends to attribute multiple symptoms to a single etiology (PCS) and look for treatments to ameliorate the syndrome.
- If one views the symptoms as having many different mechanisms (albeit the same initiating event), then one tends to take a more careful look at the typology of each symptom and is therefore better positioned to properly diagnose and treat the different sources of distress.

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Severity of subjective distress and disability is subject to a variety of influences (premorbid level, psychosocial stress, compensation/litigation, and psychiatric complications).

TBI in general and mTBI appear to increase the risk for developing a variety of psychiatric d/o that contribute to significant disability following the injury. Andrews, 2016

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Psychiatric conditions

- Personality Changes
- Psychotic syndromes
- Depression
- Mania
- Anxiety
- PTSD

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Personality Changes

- Primary psychiatric illness usually have gradual onset.
- Trauma occurs suddenly and devastatingly.
- May impair awareness of changes.
- Disorderliness, suspiciousness, argumentativeness, isolation, disruptiveness, and anxiousness can all become pronounced after Traumatic injury.

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Personality changes (cont)

- Vulnerability to the prefrontal and frontal regions in BI gives rise to changes known as “frontal lobe syndrome.”
- Cognitive functions are sometimes preserved while personality changes abound.
- Common disturbances: impaired social judgment, labile affect, lewdness, loss of empathy, increased risk-taking, lack of concern for future.

Behavioral syndromes related to frontal lobe damage

- **Orbitofrontal syndrome**: impulsivity, disinhibition, hyperactivity, distractibility, and mood lability.
- **Dorsolateral area**: slowness, apathy, and perseveration. Negative symptoms akin to symptoms associated with schizophrenia (blunted affect, emotional & social withdrawal, passivity, and lack of spontaneity).
- **Inferior orbital & anterior temporal lobes**: rage, violent and aggressive behaviors.

DSM-5 diagnostic criteria for personality change

A. Persistent personality disturbance that represents a change from previous personality.

B. Evidence from history, examination, or lab findings that the disturbance is direct consequence of general medical condition.

C. Not better accounted for by another medical condition.

D. Does not occur exclusively during the course of delirium.

E. Causes clinically significant distress or impaired social, occupational, or other functioning.
Subtypes
- Labile type: affective lability
- Disinhibited type: poor impulse control
- Aggressive type: aggression, rage outburst
- Apathetic type: marked apathy and indifference
- Paranoid type: suspiciousness or paranoid ideation
- Combined type
- Unspecified type

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Psychiatric disorders
- Studies show that psychiatric d/o are common following Traumatic injuries.
  - Fann et al. (1995) found 26% major depression, 24% generalized anxiety, 14% dysthymia, 8% substance abuse.
  - Deb et al. (1999) found psychiatric d/o in 21.7% vs. 16.4% in hospitalized population. Higher rate of depression (13.9% vs 2.1%) & panic d/o (9.0% vs 0.8%).
  - Factors associated with psych d/o include: preinjury alcohol use, lower cognitive scores, fewer yrs of education.

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Psychiatric (cont.)
- Hibbard et. al. (1998) found major depression (61%), substance abuse (28%), and PTSD (19%) were the most common psych complaints.
- Several studies suggest a higher than expected rate of preinjury psychiatric disorders (77%-88%) and substance abuse (22%-30%).
- Fann et. al. (1993) found 50% reported preinjury history of psych problems.
- Mt. Sinai Medical Center study found 51% had pre-TBI psych d/o (depression & substance abuse), at rates more than twice those reported in community samples.

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Psychotic Syndromes

- Even with severe injuries, relatively rare occurring in 0.07%-9.8%.
- Estimated 15% of pts with schizophrenia have hx of BI.
- Group of pts with schizophrenia had significantly greater hx of BI with LOC before age 10 than did pts with mania or depression.
- Cognitive deficits following TBI in conjunction with psychosis increase risk for homelessness.
- Malaspina et al. (2001) reported that even mTBI can interact with genetic vulnerability to increase the risk of developing mental illness in general and schizophrenia in particular.

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Affective changes

- Depression is common.
- Adjustment to change in abilities (sadness, grief).
- Careful diagnostic evaluation is required to distinguish grief reactions, sadness, and demoralization from major depression.
- Pre-morbid mood disturbance and substance abuse should be assessed.
- Common scenario suggested is premorbid depression leading to poor concentration, to substance abuse, to risk taking (or overt suicidal behavior), which contribute to MVA and BI.

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Depression

- Beware symptom overlap.
- Many postconcussive symptoms (e.g. subjective slowing, irritability, fatigue, and sleep disturbance) can be consistent with depressive syndrome, even when patients may not endorse explicit depressed mood.
- Gfeller et al. (1994) found a relationship b/t depression, increased rates of PCS, and impaired performance on cognitive measures.
- Prior hx or family hx of depression may be greater risk for developing symptoms subsequent injury.

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Risk of suicide
- Studies have consistently reported increased risk of suicide following TBI.
- Silver et al. (2001) found those with BI report higher frequency of attempts than those without (8.1% vs 1.9%).
- Combination of depression, disinhibition and impulsivity.
- Medical team, family, and caregivers must work closely to monitor suicide risk on a regular basis.

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Mania
- Manic episodes and bipolar d/o have been reported to occur after TBI.
- Occurrence is less frequent than depression.
- One study found bipolar d/o occurred in 1.6% of those with BI, but was not significant when social demographic factors and quality of life were considered.
- Different from primary or idiopathic mania, in that may have higher rate of relapse and higher percentage of irritable and violent behavior.

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Mania (cont.)
- Commonly patents may have both personality changes secondary to injury and a manic syndrome.
- Damage to basal region of right temporal lobe.
- Studies of secondary mania with other neurological causes suggest that genetic predisposition may be an important factor in the expression of manic and/or bipolar syndromes.

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Anxiety

- Epstein & Ursano compiled results of 12 studies from 1942-1990.
- Out of 1,199 pts, 29% were diagnosed with clinical anxiety after TBI.
- Hibbard et al. found: PTSD 18%, OCD 14%, Panic d/o 11%, generalized anxiety 8%, phobic d/o 6%.
- All of which were more frequent after TBI compared with before TBI.

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Anxiety

- Significant overlap b/t many postconcussive symptoms and core symptoms in generalized anxiety d/o (GAD).
- HA, dizziness, blurred vision, irritability, and sensitivity to light and noise.
- It is less clear how many pts actually experience anxiety and how many have diagnosable anxiety disorders.

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PTSD

- In general pop, lifetime prevalence of PTSD is 7.8%.
- 9.2% risk of developing PTSD after exposure to trauma, highest for assualtive violence.
- 10% to 25% with TBI patients.
- Increasing awareness of relationship b/t PTSD and brain injury.
- PTSD and acute brain response are not uncommon after serious MVA.
- Sleep disturbance, decreased reactivity, exaggerated startle response, daytime flashbacks, and avoidant behaviors.
- Studies have shown TBI groups with PTSD were significantly more symptomatic than TBI without PTSD.
- Studies suggest PTSD can amplify postconcussive symptoms after mTBI and complicate recovery.

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PTSD Risk Factors
- Pre-trauma Risk – Sex and marital status are strongest predictors of PTSD (female and previously married are highest risk)
- Trauma-related risk – Men, combat, and witness to death/injury. Women, rape, and sexual assault
- Posttraumatic risk – Lack of support, additional stressors (stronger than pre-trauma factors)
- Poor cognition – pre-trauma cognitive deficits (less able to cope)
- TBI increase risk after 6 months if acute stress d/o, symptoms of depression/anx within 1 week of injury (or previous history)

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Clinical Presentation
- Symptoms seen both in post-TBI and PTSD
  - Noise sensitivity
  - Fatigue
  - Anxiety
  - Insomnia
  - Poor concentration, memory
  - Irritability
  - Symptoms are not diagnostic as high base rates in general pop

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Evaluation
- Symptom Checklists – Self-report questionnaires (screening only). Poor specificity
- Structured Interviews – “gold standard”
  - Extended time to complete (30 to 120 minutes)
  - Structured Interview for PTSD (SI-PTSD);
  - Clinician-Administered PTSD Scale for DSM-5 (CAPS-5);
  - PTSD Symptom Scale – Interview (PSS-I);
- Neuropsychological Examination

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Management

- Psychopharmacology where cognitive impairment makes psychotherapy unrealistic.
- SSRI more effective than older meds.
- Trazodone helpful with nightmares.
- Antipsychotics can interfere with cognition and cognitive recovery.
- Sedatives not recommended in PTSD (for long term use).

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Management

- CBT and exposure therapy (limited by memory and cognitive deficits if present).
- Referral to PTSD Specialists can be helpful. (Association for Behavioral and Cognitive Therapist, DOD/VA)

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Treatment Issues

- Proper evaluation is imperative.
- Significant effort should be expended to clarify premorbid history.
- Prior H (medical history, substance abuse, education, work history, psychiatric history).
- Neuropsychological assessment helps identify cognitive strengths and weaknesses and patterns of performance helpful in determining treatment decisions, educational and occupational recommendations.
- Also, NP aids determination of baseline functioning and track recovery (is treatment working?).

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Treatment (cont.)
- Psychopharmacological treatment
- Behavioral
- Psychological
- Social interventions

Behavioral & Cognitive
- Behavioral methods used in response to aggressive outbursts and maladaptive social behaviors.
- Behavioral modification was found to be 75% effective in dealing with disturbed behavior after severe BI.
- Current cognitive therapies focus on teaching the pt new strategies to compensate for lost or impaired functions.

Psychological & Social
- Psychological issues revolve around:
  1. Premorbid psychopathology
  2. Response to traumatic event
  3. Reaction to deficits from BI
  4. Issues related to potential recurrence of BI
Conclusions

- Traumatic Brain Injury can lead to emotional damage in patient and family.
- Must increase efforts devoted to prevention.
- Advocate a multidisciplinary and multidimensional approach to the assessment and treatment of neuropsychiatric aspects.
- Proper management is imperative for successful return to activities.

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Questions

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References


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