Neuropsychological Evaluation

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Disclosures

- No Financial Disclosures
- Employee of Frazier Rehab & Neuroscience Institute, part of KentuckyOne Health

Agenda

- Neuropsychological Assessment
- Dementia, Mild Cognitive Impairment, Mild Traumatic Brain Injury
- Depression, Pseudo-dementia and Anxiety

Case #1

What is Neuropsychology?

- Study of brain-behavior relationships
- Behavioral, emotional, cognitive, and functional effects of brain injury and neurological disorders
- Licensed clinical psychologist – not a neurologist or M.D.
  - Trained in science of cognitive and behavioral assessment
  - We use a different set of diagnostic “tools”
- Integration of psychometric test data and other methods

Practice & Roles of a Neuropsychologist

Who make us who?

- ~80% doctoral-level Clinical/Counseling Psychologists with 2-year post-doc in Clinical Neuropsychology
- ~80% work with adults, at least for part of their practice
- ~42% institution-based, ~22% private practice, ~25% both institution/private practice, ~10% post-doctoral

Where do we practice?

- ~40% Institution/Medical
- ~27% Private Practice
- ~26% Combined Institution/Private Practice

Practice & Roles of a Neuropsychologist

What do we do?
- ~85% clinical/administrative, 8% teaching/training, 7% research
  *Assessment / interview / consultation / feedback / intervention

- Collaborate with other specialists
  - Neurology, PM&R, Rehab teams (OT, PT, ST, Psych)
  - Neurology, Primary Care, Psychiatry, Physical are top referral in that order of frequency*
  - Surgical Conferences

Common Clinical Issues Referred to our Neuropsychology Clinic
- Memory Loss/Dementia
- Trauma
- Parkinson's disease
- Stroke
- Seizures
- Tumor
- Pre-surgical
- Psychiatric

Neuropsychological Assessment: Components
- Integration process, including:
  - clinical interview
  - collateral information/interview
  - paper and pencil standardized, normed, objective tests of multiple cognitive domains
  - computer tests
  - behavioral observations
  - medical history & medications
  - neuroimaging
  - psychiatric history

- What I tell my patients:

Top 5 Conditions Referred for Neuropsychological Evaluations*

<table>
<thead>
<tr>
<th>Rank</th>
<th>2005</th>
<th>2010</th>
<th>2015</th>
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<tbody>
<tr>
<td>1</td>
<td>Traumatic Brain Injury</td>
<td>Traumatic Brain Injury</td>
<td>Traumatic Brain Injury</td>
</tr>
<tr>
<td>2</td>
<td>ADHD</td>
<td>ADHD</td>
<td>ADHD</td>
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<tr>
<td>3</td>
<td>Learning Disorder</td>
<td>Elderly dementias</td>
<td>Elderly dementias</td>
</tr>
<tr>
<td>4</td>
<td>Elderly dementias</td>
<td>Learning Disorder</td>
<td>Seizure Disorder</td>
</tr>
<tr>
<td>5</td>
<td>Stroke</td>
<td>Other medical/neurological</td>
<td>Other medical/neurological</td>
</tr>
</tbody>
</table>

*Includes respondents who evaluate patients across the full lifespan (i.e., pediatric and adult)

Neuropsychological Assessment: Purposes
- Differential diagnosis
  - Part of the diagnostic work up
  - Complementary to imaging, EEG, labs, etc.
  - Not diagnostic alone
- Establish baseline...
  - for tracking of disease burden/progression, if any (MCI, AD, etc.)
  - for effectiveness of medical intervention or treatment (TBI, stroke)
  - for early detection of symptoms, especially if subtle
- If early in recovery: identify deficits for rehab (Treatment planning).
- Later in recovery: prognosis, lasting effects
- Functional ability – “Life decision points” (RTW, Drive, Independence, FDA, living arrangement), identify compensatory strategies to leverage
- Pre-surgical and post-surgical assessment
  - DAS and WAIS evaluations
  - Consent, risk factors for poor outcome, understanding of surgical outcomes/procedure, treatment compliance, etc.
- Educational: intervention/community support
- Forensic

NPE: Domains Measured
- Intelligence
- Language
- Academic Skills
- Visuospatial Abilities
- Attention & Executive Functioning
- Memory and Learning
- Motor/Sensory
- Psychological / Emotional / Stress
- Functional Status
- Health Behaviors
NPE: Unique Features

- Use of objective, standardized administration of valid psychometric tests based on matched normative comparisons with individuals of similar age, education, sex, SES to obtain derived standardized scores
  - Sensitivity and specificity of test
  - Valid/reliable psychometric properties
  - Standardized
  - Base rates
  - Compare data to estimated premorbid ability
  - Cultural considerations
- Identify nature and localization of cognitive/cerebral dysfunction based on a pattern (constellation) of scores/profile consistent with known conditions (perhaps not detectable by imaging)
- Pediatric to geriatric populations
- Performance validity and symptom validity testing


NPE: Performance and Symptom Validity Testing

Change in cognition – especially in an individual with a family history of dementia or history of dementia risk factors
Change in personality
Functional decline
Fall bedside MMSE / SLUMS screenings
Reversible causes are all negative
While most referrals come from neurology, family practice providers may be first health care professionals to hear of symptoms
Need to be able to cooperate with testing (SSL / ESL limited norms)

When to Refer?

- Change in cognition – especially in an individual with a family history of dementia or history of dementia risk factors
- Change in personality
- Functional decline
- Fall bedside MMSE / SLUMS screenings
- Reversible causes are all negative
- While most referrals come from neurology, family practice providers may be first health care professionals to hear of symptoms
- Need to be able to cooperate with testing (SSL / ESL limited norms)

Please discuss nature of referral, what to expect, purpose – evaluations will be of different lengths, depending on age and referral questions – will be long!

NPE: Physician Satisfaction

Tremont et al., TCM, 16(4), 2002
- Perceptions of hospital-based neuropsychology levels
  - Most common RFP: differential / confirmatory diagnosis
  - High level of satisfaction and agreement with report / results
  - 94.3% found the report very useful; 97% agreed with diagnoses / recommendations; 94% would refer again

Temple et al., TCM, 21, 2006
- Most common RFP: differential / confirmatory diagnosis
  - High level of satisfaction and agreement with report / results
  - 517 AMA member surveyed; multiple physician specialty / practice settings
  - 94% agreement with diagnostic impressions across all specialties
  - 88% found NPE useful across all specialties; 87% would refer in the future
  - 83% found useful; 85% would refer in future

What will you get?

3-7 PAGE REPORT
DIAGNOSIS
FUNCTIONAL ABILITY
STRENGTHS
WEAKNESSES
RECOMMENDATIONS
PROGNOSIS FOR RECOVERY

1. Schroeder, et al, American Family Physician, 2019

Secondary Gain & Symptom Exaggeration:
Trends in Malingering Research
Patient Satisfaction


70% overall satisfaction with evaluation process n=129

Bodin, et. al, TCN, 21(6), 2007. Parent satisfaction with pediatric neuropsychological evaluation

82% either mostly or very satisfied n=177

Dementia
Diagnostic Criteria, Types, Evaluation, Management

Case 2

What is Dementia?
- Major Neurocognitive Disorder (DSM-5)

Impairment in cognition that causes loss of independence in activities of daily living
Dementia is a “syndrome” — a group of symptoms — with multiple causes

DSM-5
A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (aphasia, apraxia, agnosia, memory, etc.) — based on:
1. Concern of the individual, informant, or the clinician that there has been a significant decline in cognitive ability;
AND
2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, other quantified clinical assessment.
B. The cognitive deficits interfere with independence in instrumental ADLs
C. Not delirium
D. Not another mental disorder to explain symptoms

Dementia:
Types & Causes
- Alzheimer’s disease
- Atypical Frontal Variant AD
- Posterior Cortical Atrophy (PCA)
- Vascular
- Parkinson’s disease
- Parkinson’s Plus syndromes
- Lewy bodies disease
- Frontotemporal Lobar Degeneration (FTD)
- Alcohol-Related Dementias
- Huntington’s disease
- Mixed Dementias

Alzheimer’s Dementia: Epidemiology

Most common form of dementia:
- AD accounts for 60% - 80% of all dementias (Yaffe & Hazzouri, 2017; Alz. Assoc., Alz. & Dementia, 2018(14): 367-429)
- AD prevalence decade:
  - 11% of U.S. adults over age 65 (Herbert, et al., 2013, Neurology, 80(19): 1778-83)
  - 32% of adults over 85 (Herbert, et al., 2013, Neurology, 80(19): 1778-83)
- 1.7 million in U.S. with AD (Alz. Assoc., Alz. & Dementia, 2018(14): 367-429)
- One person every 65 seconds develops AD
- 6-8%
- Incidence of AD doubles every 5 years after age 65 (Matthews, 2017, In The behavioral neurology of dementia.)
- Early onset AD: typically faster decline, greater genetic component, less common
Vascular Dementia

Vascular cause alone accounts for about 10-20% of all dementias (Yaffe, K., In The behavioral neurology of dementia, 2017; Alz. Assoc., Alz. & Dementia, 2018(14): 367-429)

- Large vessel disease caused by single or multiple critical or subcritical infarctions
  - ACA, PCA, MCA distributions ex.; ischemic borderzone, watershed areas
  - Thalamus, caudate, basal ganglia, etc.
- Small vessel disease
  - Lacunar infarction
  - Periventricular and deep white matter
- "Subcortical vascular dementia" – dementia secondary to ischemia caused by small vessel disease

- Temporal relationship between vascular event and cognitive decline
  - Usually, but not always, a step-wise decline
  - Depression, anxiety are common
  - Vascular Mild Cognitive Impairment - VaMCI

Mixed AD/Vascular Dementia is Common

About 50% of individuals with AD pathology also have vascular brain changes (Alz. Assoc., 2018; Schneider, et al., 2007)

  - 86% of their patients meeting criteria for AD were also found to have evidence of other possible dementing disorders, including cerebrovascular disease
- Bowler, Munoz, and Hachinski (1998)
  - In 22 patients with dementia: 81% of patients were diagnosed clinically with AD; however, at autopsy, only 44% of cases had pure AD, without any other causative causes of dementia.
- Crystal et al. (2000)
  - Of 56 patients meeting diagnostic criteria for AD, 28 had pure AD, while the remaining 28 also displayed a wide variety of cerebrovascular lesions.

Neuropathology of Mixed Dementias

Schneider, et al., 2007
141 subjects
80 had AD pathology on autopsy (57%)
37 had AD pathology alone (44%)
43 had AD pathology plus additional pathology (14%):
- 58% also had vascular infarcts
- 28% also had PD/LBD pathology
- 9% also had both vascular + PD/LBD
50 had clinical dementia (35%): Of these, most common neuropathology was AD + vascular (38%), followed by AD path. alone (30%)

FTD: Neuroanatomical Pathology

Leading cause of early onset dementia (Pang & Miller, 2017, In The Behavioral Neurology of Dementia)

- In patients <65, 20-50% have FTD
- 60% have onset between ages 45-64 (Kopman & Roberts, 2011, J. Mol Neuroscience, 45(3): 330-5)
- FTD more common in men (Rathore et al., Neurology, 2002; 58(1))
- High heritability – 40% with family history (Goldman et al., Neurology, 2005; 65(11), 1817-9)

Two Variants

- Language
- Behavioral
Mild Cognitive Impairment (MCI) - Peterson

- Transitional stage between normal aging and dementia
- Prevalence: 15-20% over age 60
- Roughly (5-15%) of MCI cases per year convert to dementia, most commonly the amnestic type progressing to AD
- Often, but not always, a prodromal phase of AD

Diagnosis:
- Mild but measurable cognitive decline exists, beyond normal aging...
- But it is not severe enough to cause loss of independence in daily living activities
- Diagnosis requires cognitive complaint
- Intact general cognitive ability

Peterson, Continuum (Minneap Minn) 2016, 22(2), 404-418
Roberts & Knopman, Clinical Geriatric Med., 2013(29), 753-72

Dementia: Evaluation

- History/Clinical exam
- Labs: Reversible Causes
- Imaging
- Cognitive, Adaptive & Psychological Screening
- Neuropsychological Assessment

Evaluation: History/Interview

- Interview is revealing....
- Is it really memory loss, or is it something else???
- Word-finding problems - very common in normal aging
- Slower processing speed - common as well “Not as sharp as I was before.”
- Attention problems - multifactorial causes
- Fatigue/tiredness/stress/weds, etc.
- Repeats self in interview
- Anosognosia
- Caregiver - Informant clues
- Collateral information gathering on ADLs – important to measure ADL’s
- Onset, Precipitating Event, Frequency, Progression

Evaluation: Treatable/Reversible Causes of Dementia-like Symptoms

- Psychiatric
  - Pseudo-dementia
- Metabolic
  - Hepatic Encephalopathy
  - Hypothyroidism
  - Vitamin Deficiency (B12, folic acid)
- Infectious
  - Herpes Encephalitis
  - AIDS
- Medications
  - Anti-cholinergic Effects
- Systemic/Other
  - OSA
  - Tumor – Paraneoplastic syndrome
  - NPH
  - Inflammatory/autoimmune
Dementia Screening

- MMSE: cutoff score <24
- SLEMS: <26; <24 with less than 12 year education
- MoCA: <26
- Clock Drawing
- Cross Drawing
- Functional Activities Questionnaire (FAQ)
- Neuropsychiatric Inventory (NPI)

Dementia Screening: Clock Drawings

Dementia: Functional & Psychiatric Screening

Dementia: Why Neuropsychological Assessment?

- Several organizations stress importance of neuropsychological assessment in diagnosis and management of dementia (Schneider, et al., 2019)
  - National Institute on Aging-Alzheimer’s Association Workgroup
  - European Federation of Neurological Societies – European Neurological Society
  - ICD-10 & DSM-5:
    - NPE is preferred method of documenting cognitive dysfunction
  - NPE should be considered in most cases, especially in mild symptoms and in those who are young or have atypical features (Boeve & Miller, 2017, TBND)

  Complementary part of diagnostic workup of differential diagnosis:
  - Normal aging vs. MCI vs. Dementia (if so, type) vs. Depression vs. Somatic
  - Medications Necessary?
  - Functionality
  - Global ability
  - Specific cognitive deficits, compared to baseline
  - “Constellation” of scores to differentiate type of dementia
  - In turn, determines treatment, expectations
    - Driving, living arrangement, guardianship, retirement, POA, etc.
    - Tailored compensatory strategies / recommendations
    - Psychological overlay/pseudodementia
    - Establishes baseline: clarify diagnosis/progression with serial assessment
### NPE Patterns

<table>
<thead>
<tr>
<th></th>
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<th>FTD</th>
<th>VAD</th>
<th>PDD</th>
<th>Depression</th>
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<td>Language</td>
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<td>Visuospatial</td>
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<td>▼</td>
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<tr>
<td>Ex. Functioning</td>
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<tr>
<td>Memory</td>
<td>▼</td>
<td>▼</td>
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<tr>
<td>Depression/Psych</td>
<td>▼</td>
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</table>

*Semantic fluency/naming

### Differential Diagnosis

**Alzheimer's Disease**
- Language
- Visuospatial
- Ex. Functioning
- Memory
- Depression/Psych

**Dementia**
- Functional Impairment Absent
- Lack of Progression
- Demencia Absent
- Parkinsonism Present
- Visual Hallucinations Early in Disease
- REM Sleep Disorder
- Fluctuating Attention
- Age < 65
- Behavioral Disorders Early in Disease
- Spinal Muscular Atrophy
- Memory Abilities Variable
- Imaging Evidence of Vascular Disease
- Lack of Progression

**Consider**
- MCI
- Lewy Body Disease
- Fronto-temporal Dementia
- Vasc Dem

### Dementia: Management

**Medications**
- Vary depending on type of dementia: Acetylcholinesterase inhibitors indicated for AD dementia, less effective in FTD. Namenda for moderate to severe AD dementia (mixed results with PDD); avoid older antipsychotics with UBO, Nuplazid for PDD w/ psychosis
- Avoid medications with anticholinergic properties
- Minimize psychiatric medications with possible adverse cognitive side-effects — Benzodiazepines, Opioids

**Weak efficacy of antidepressants, antipsychotics, and mood stabilizers for management of psychiatric symptoms in AD.** (Onyike & Rabins, 2017, In the Behavioral Neurology of Dementia)

**Inconsistent support for high intake of vitamins C, E, B, and folic to lower risk of AD (Luchinger & Mayeux, 2004, Lancet Neurology, 3(10), S79-87).**

### Dementia: Medications to Avoid

- Exercise!!!
- Manage vascular risk factors
- Eat healthy
- Study!

### Dementia: Management

- Refer/treat sleep disorders, dehydration, pain etc.
- Correct hearing and vision loss, to the extent possible
- Promote regular sleep-wake cycles
- Caregiver respite/support/groups
- Future planning—dependent on dementia type and course
- Psycho-education, when necessary
- Driving evaluation / Safety / Supervision
- Manage vascular risk factors
- Leverage strengths from NPE
- Serial neurocognitive testing in MCI or unclear etiology
- Behavioral Management:
  - Reassurance, distraction, structure, redirection
  - Safety
  - Don’t argue

**References**
Traumatic Brain Injury: Definition

- **CDC**: a disruption in the normal function of the brain caused by a bump, blow, or jolt to the head, or by a penetrating head injury.
- **ACRM**: an alteration of brain functioning, or other evidence of brain pathology, caused by an external force.

TBI: Incidence Rates

- **CDC 2013**: About 2.8 million TBI-related emergency department (ED) visits, hospitalizations, and deaths occurred in the United States.
  - 52,000 deaths
  - 282,000 hospitalizations
  - 2.5 million ED visits

TBI: Pathophysiology

- **Primary Injuries**
  - **Focal**
    - Skul fracture
    - Contusion – coup contre coup
    - Penetrating injury
    - Intracranial hematrage
  - **Diffuse**
    - Diffuse Axonal Injury

- **Secondary Injuries**
  - Cascade of associated biochemical and molecular events
    - Neurochemical and electrochemical imbalances:
      - Abnormal levels of neurotransmitters: calcium and sodium influx, mitochondrial overload
      - Excess excitatory neurotransmitter release: glutamate = excitotoxicity
    - Impaired brain metabolism:
      - Change in cerebral perfusion
      - Fluctuating glucose metabolism
    - Biomolecular degradation:
      - Free radical production
      - Axonal swelling, myelin degradation
    - Inflammation:
      - Release of cytokines, nitric oxide
      - Cerebral edema
        - Secondary to cellular swelling due to ionic imbalance
        - Cell death, unscheduled apoptosis, and cytotoxicity

TBI: Definition by Injury Severity

<table>
<thead>
<tr>
<th>Classification</th>
<th>Initial GCS</th>
<th>PTA</th>
<th>Loss of Consciousness</th>
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</thead>
<tbody>
<tr>
<td>Mild</td>
<td>13 – 15</td>
<td>&gt;24 hr.</td>
<td>0 – 30 min.</td>
</tr>
<tr>
<td>Moderate</td>
<td>9 – 12</td>
<td>24 hr. – 7 days</td>
<td>30 min. – 24 hr.</td>
</tr>
<tr>
<td>Severe</td>
<td>3 – 8</td>
<td>&gt;7 days</td>
<td>&gt;24 hr.</td>
</tr>
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</table>
Mild TBI: Definition

American Congress of Rehabilitation Medicine (ACRM)

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);
4. Focal neurologic deficit(s), which 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 min, an initial GCS of 13-15
- And PTA not greater than 24 hours

(Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the ACRM, 1993)

Mild TBI: Outcome & Prognosis

Vast majority of TBIs are mild: 70-80% (Kraus, et al., 1996; Zasler et al., 2007; Williams et al., in Traumatic Brain Injury)

It is well established in literature that most patients recover fully, cognitively, within days, weeks or at the latest, three months post-injury. (Binder et al., 1997; Binder, Rohling & Larrabee, 1997; Carroll et al., 2005; McCrea et al., 2000; Schretlen & Shapiro, 2003; Carroll et al. - WHO, Collaborative Centre Task Force on Mild TBI; 2004; Belanger & Vanderploeg, 2005; Iverson et al., 2005; Ruff et al., 2009; McCrea, et al., 2009; Rohling et al., 2011)

Typical absence of objective evidence on neuroimaging.

If present = “complicated” mTBI, with outcome similar to moderate TBI

Mild TBI: Favorable Course of Cognitive Recovery

Schretlen & Shapiro, 2003

McCrea et al., 2009

Binder et al. (1997)

11 studies

Beyond acute stage of recovery, small effect size on cognitive performance (d = -.12)

Schretlen and Shapiro (2003)

39 studies; 48 comparisons of mTBI patients (n=1716) vs. controls (n=1164)

Moderate effect size for cognitive impairment in acute stage of injury  (1st 6 days, d = -.41)

But full cognitive recovery typically attained after 1-3 months (d = -.08)


Cognitive deficits first few days post injury (attention, processing speed and memory recall)---largely resolve by 3 months.

Children: do not have higher rates of subsequent behavioral or academic problems

Mild TBI not a significant risk factor for seizures, tumor, or dementia (or is inconclusive)

Frencham et al. (2005), follow-up to Binder et al. (1997)

17 studies  - 1993 to 2003

Small effect sizes across all stages post-injury adults

Effect tends toward zero at 3 months post-injury, comparable to non-head injury patients

Belanger et al. (2005)

39 studies, including 1463 mtbi patients and 1191 controls

No residual neuropsychological impairment after 3 months post-injury (d = .04).

Large effect sizes for litigants (d = .78). Litigation associated with stable or worsening of cognitive functioning over time.

Mild TBI: Outcome, Iverson, 2005

"Permanent cognitive, psychological, or psychosocial problems due to the biological effects of this injury should be considered uncommon in trauma patients and rare in athletes." (p. 301)

"Under most circumstances, we should anticipate good recovery following an mTBI. Patients and athletes should be reassured." (p. 311)

"Very mild concussions likely produce virtually no permanent damage to cells resulting in long-term symptoms or problems...." (p. 303)

"In prospective studies using adequate methodology, mTBIs are not associated with measurable cognitive decrements after a few weeks or months after injury." (p.303)

Postconcussive Symptoms

Controversial, ill-defined, non-specific syndrome

- Some studies say as high as 10-15% but probably too high due to methodological and sample characteristics
- Probably more like 3-5%

Fatigue Irritability Insomnia
Headaches Dizziness/vestibular
Problems concentrating Forgetfulness
Light or sound sensitivity Pain

A complex interaction of cognitive, physical, and emotional complaints

Postconcussive Symptoms: Non-specificity of Symptoms

- PCS symptoms are so non-specific that they have little diagnostic specificity – not unique to mTBI
  - Headache, dizziness, irritability, concentration problems are very common in healthy individuals and college students, without TBI (Sawchyn et al., 2000; Courney et al., 1988)
  - Chronic pain patients endorse PCS symptoms at high rate; 37% depression, 31% anxiety, 30% memory problems 81% endorsed 3 or more PCS in both groups, 25% endorsed 6 or more (Iverson & McKraken, 1997)
- Non-TBI personal injury claimants endorse PCS symptoms at high rate, comparable to mTBI claimants (Less-Haley & Brown, 2001)

- Symptoms of PCS, by themselves, can result in cognitive impairment (Glenn, 2012, in: Traumatic Brain Injury, Tsao, JW ed.)
- “Complaints are not diagnostic of traumatic brain injury neuropathology.” (Larrabee et al., 2013, p. 231)

PCS: Factors Prolonging Recovery

- Iverson, 2005: “In these [few trauma] patients, recovery can be incomplete and can be complicated by preexisting psychiatric or substance abuse problems, poor general health, concurrent orthopedic injuries, or comorbid problems (e.g., chronic pain, depression, substance abuse, life stress, unemployment, and protected litigation.” (p. 301)
  - Structural damage - macroscopic or microscopic (complicated mTBI)
  - Chronic health issues: pain, sleep disorder, migraines
  - Pre-injury or co-existing psychological factors and/or somatization
  - Symptom exaggeration / malingering
  - Misattribution, symptom expectations, response bias
  - Litigation/companation
  - Demographics (less education, female, older age)
  - PTSD

mTBI/PCS: Overlap with PTSD

PTSD
Re-experiencing sx
Nightmares
Intrusive thoughts
Shame/Guilt

Insomnia
Irritability
Depression/Anxiety
Fatigue
Problems
Memory problems

mTBI/PCS
Dizziness
Light/sound sensitivity
Blurred vision

Secondary Gain and Symptom Exaggeration

Base rates of insufficient effort and malingering increase dramatically in workers’ compensation, litigation, and other secondary gain contexts

- Mittedberg et al. (2002)
  - Survey of 388 U.S. neuropsychologists regarding 33,531 annual cases. Estimated rates of probable malingering:
    - Personal injury litigation = 29%
    - Disability = 30%
    - Criminal = 25%
    - Dental considerations = 45%
  - Based on conditions:
    - Mild head injury = 30%
    - Traumatic/chronic fatigue = 25%
    - Chronic pain = 31%
    - Neurotoxicity = 27%
    - Electrical injury = 20%
Secondary Gain, Disability, and Persisting Symptoms

WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury (Carroll et al., 2004, p. 102):

“Although the evidence indicates good recovery for most adults sustaining MTBI, where symptoms and disability are persistent, compensation and litigation factors are important, and exploratory studies suggest that prior health, age and life stressors are also determinants of poorer outcome.”

TBI: Neuropsychological Evaluation

- When to administer?
- Outline extent of deficits to inform rehabilitation and recovery
- Degree of psychological distress present
  - Medication? Psychotherapy?
- Symptom validity
- Education regarding injury specifics and expected outcome
- Functional determination
  - Return to work/school?
  - Return to drive?
  - Live independently?
- If disability:
  - Functional or academic accommodations necessary?

Depression and Anxiety

Diagnosis, Management

Sadness vs. “Clinical” Depression

Sadness
- Normal
- Temporary
- Does not Impair Functioning

Depression
- Clinical Term
- Constellation of Symptoms
- Longer Duration
- Possibly Continuous/ Recurrent
- Not Always Reactive
- Impairs Functioning
- Continuum of Severity

Major Depressive Disorder - DSM-5

Either:
- Depressed mood or
- Anhedonia

And at least four of the following in a consecutive two-week period:
- Weight/appetite change
- Sleep disturbance
- Fatigue
- Psychomotor retardation or agitation
- Worthlessness
- Concentration problems
- Suicidal ideations or passive thoughts of dying

Specifiers: Mild, Moderate, or Severe, with/without psychotic features

“Depression is a thief... it steals your life away. When you should be out participating in life, you’re not.”

“It’s like a heavy blanket weighing you down... What am I living for?”

“Like a black hole inside of me that I can’t fill, no matter what.”

“I’m stuck in the bottom of a well... it’s totally dark.”
Depression: Symptoms

Physical Symptoms
- Fatigue/Loss of energy
- Sleep Disturbance
- Appetite changes
- Restlessness
- Sexual Dysfunction

Affective Symptoms
- Depressed mood
- Anhedonia
- Hopelessness
- Helplessness
- Worthlessness
- Loneliness/Empathy
- Apathy
- Isolation/Withdrawal

Cognitive Symptoms
- Decreased concentration
- Slowed thinking (bradyphrenia)
- Suicidality
- Indecision
- Forgetfulness
- Pessimistic thinking

Pseudodementia vs. Dementia

Depression
- Complain of terrible memory!!!
- But memory recall and recognition often good, if not WNL
- Oriented better
- Language intact
- Visuospatial intact
- Executive skills diminished but not severely
- Inconsistent performance – effortful processing
- Careless mistakes
- Performance improves with encouragement
- A lot of “Don’t know” answer
- Faster onset and faster progression (sometimes)
- Or prolonged course without adaptive decline
- Still could be an early sign of a dementing process emerging

Dementia
- May not complain or realize cognitive decline
- Memory consolidation/recognition impaired (DAT)
- Less oriented
- Language change, apraxia, agnosia
- Apraxia, perseveration
- Executive skills worse, depending on dementia type
- Confabulated/distorted/perseverative mistakes
- Performance does not improve
- Incorrect answers
- Slow insidious onset

Anxiety Disorders
- Generalized Anxiety Disorder
- Specific Phobias
- Panic Disorder
- Agoraphobia
- Social Anxiety Disorder
- PTSD
  - Now classified as a Trauma-Related Disorder in DSM-5

Screening Tools
- GDS
- BDI-II
- BAI
- PHQ-7
- GAD-7

Treatment: Medications
- SSRI
- SNRI (Effexor, Wellbutrin)
- Tricyclic antidepressants (amitriptyline)
  - But more potential for adverse cognitive SE
  - Collaboration with psychiatry

Treatment: CBT

Thoughts

Behavior

Emotions
Questions?