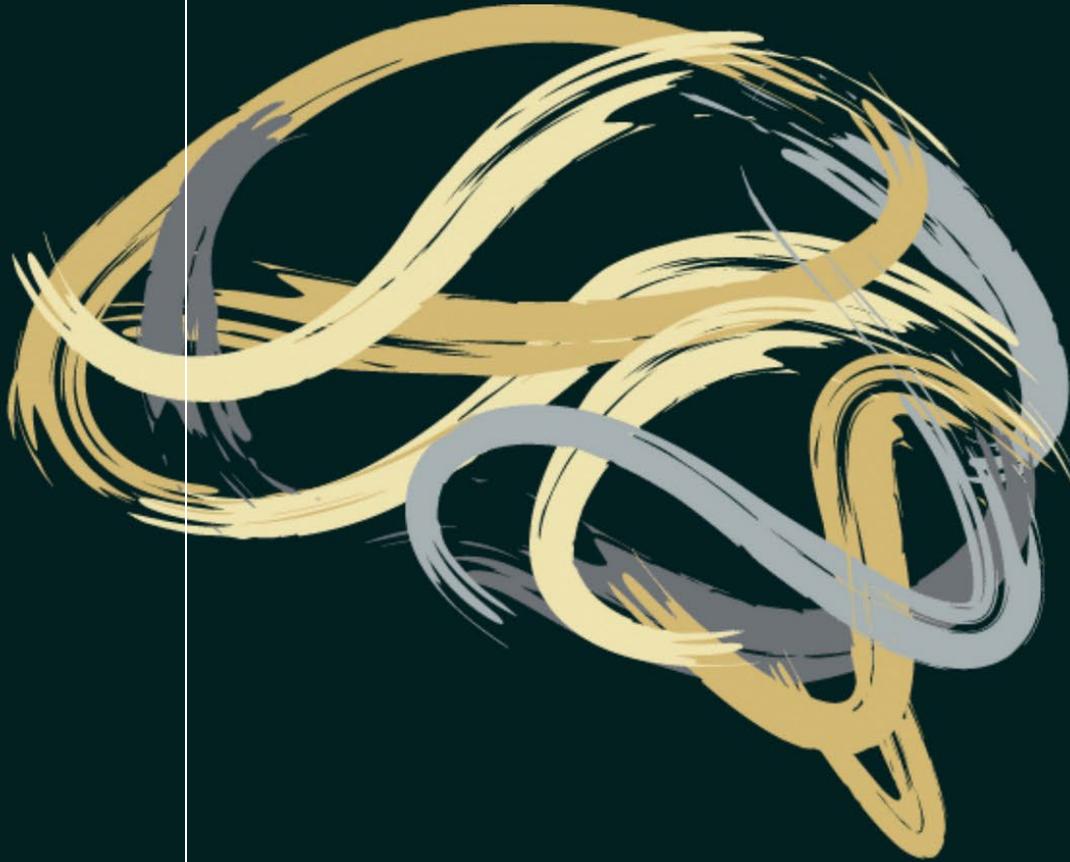




Department of Psychiatry

SCHOOL OF MEDICINE

UNIVERSITY OF COLORADO ANSCHUTZ MEDICAL CAMPUS



# The Invisible Injury: Identity Disruption and Recovery in Adults with Brain Injury

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Thida Thant, MD and Holly Gerber, PhD

University of Colorado School of Medicine, Department of Psychiatry

Psychiatric Consultation for the Medically Complex Clinic

EASY Consultation

# Disclosures

We do not have any relevant financial relationships with ineligible companies.

We are employed by the University of Colorado and will discuss some clinical services/resources we are involved in.

# Overview & Learning Objectives

After this session participants will be able to:

- Identify common psychological struggles in adult patients living with brain injury
- Discuss practical interventions to help support adult patients living with brain injury
- Review available and accessible resources for both patients living with brain injury and professionals providing care to adult patients with brain injury.



# Background

- 2023 National Health Interview Survey: 3% (~ 9, 757,000) of Americans self-reported experiencing a TBI in the prior year
  - CDC: >200,000 TBI –related hospitalizations in 2020 and almost 70,000 TBI-related deaths in 2021.
- Despite this, many TBI patients struggle to access effective, timely care post-injury, especially for mental health effects.
- Permanent neuropsychiatric disabilities affect an estimated 80,000 to 90,000 patients who suffer a TBI.
  - Psychosocial and psychological impairments --> substantial disability & significant family stress
- Even more mild TBIs (i.e., without an associated hospital stay) may result in neuropsychiatric sequelae.

*“Many times, patients will come to us and say: ‘I’m not the same person I was before my brain injury.’ This is where we focus on re-scripting their narrative. We might have to remind them that we can’t necessarily get them back to who they were before the injury, but we can get them better than they are now.”*

- Dr. Durga Roy, ACLP 2024

# Case

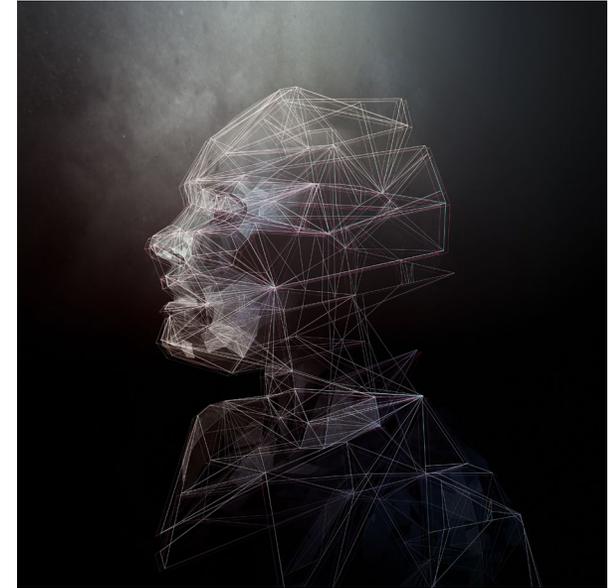
David is a 45-year-old man who presents to your office with complaints of memory issues and depression. His medical history includes multiple prior concussions from playing sports throughout high school and college but a year ago he suffered from a TBI following a severe car accident for which he had a prolonged medical admission and recovery.

Since the accident, he describes poor sleep, depressed mood and struggles with motivation. He has been unable to return to work due to his cognitive struggles which he finds frustrating and finds himself withdrawing from his wife and friends. He finds himself more irritable, which was never an issue before.



# Common psychological struggles associated with brain injury

- Risks and vulnerabilities to adjustment
- "Good days and bad days" with cognition and fatigue
- Emotional Dysregulation
  - o Irritability
  - o Emotional lability
- Mood, anxiety, and post-traumatic reactions
- Identity and Role Disruption
  - o Grief for "old self"
  - o Loss of social roles
  - o Relationship strain
- "I don't look injured"
- Impaired Insight
- Suicide Risk



Jonasson et al. (2018). Mental fatigue and impaired cognitive function after an acquired brain injury, *Brain and Behavior*, 8(8), doi: 10.1002/brb3.1056

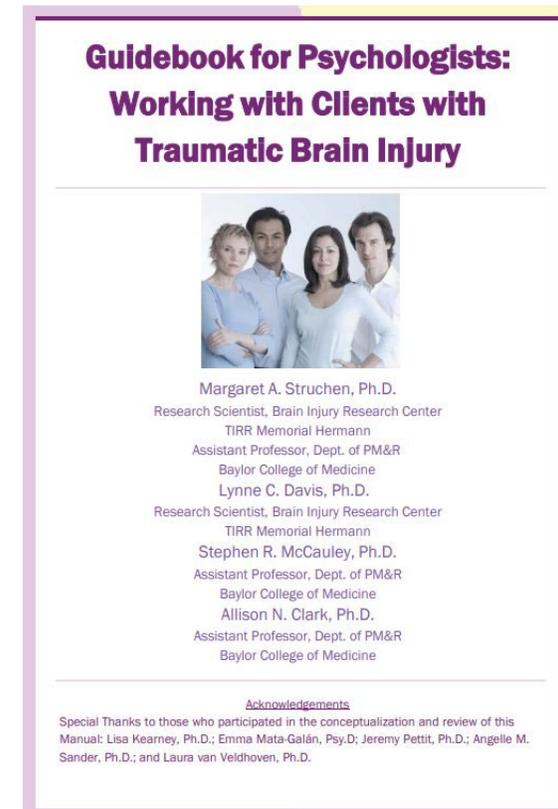
Howlett, Nelson, & Stein (2023). Mental health consequences of traumatic brain injury, *Biological Psychiatry*, 91(5), 413-420. doi: 10.1016/j.biopsych.2021.09.024

Villa, Causer, & Riley (2021). Experiences that challenge self-identity following traumatic brain injury: a meta-synthesis of qualitative research, *Disability Rehabilitation*, 43(23), doi: [10.1037/rep0000462](https://doi.org/10.1037/rep0000462)



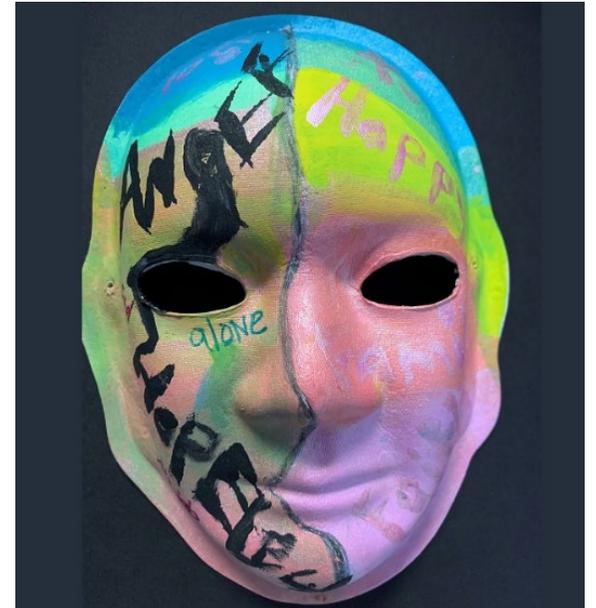
# How to elicit patients' feelings about living with BI

- Validation and normalization first
- Ask about function over abstract emotional inquiry
- Anchor questions to specific contexts
- Ask about identity disruption directly
- Other tools and strategies that may be helpful
  - Shorter prompts to reduce cognitive fatigue
  - Slow pacing and repetition
  - Emotion lists or scales



# Coping with identity changes after BI

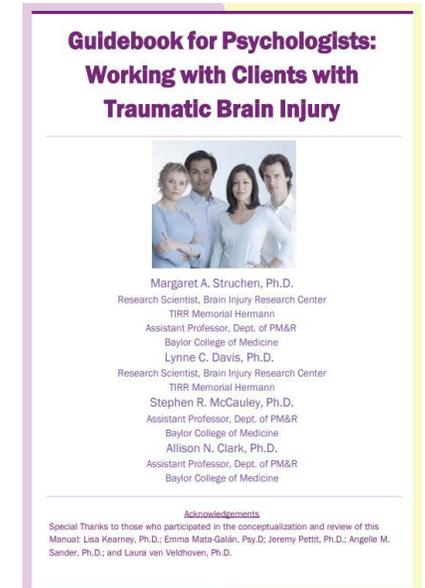
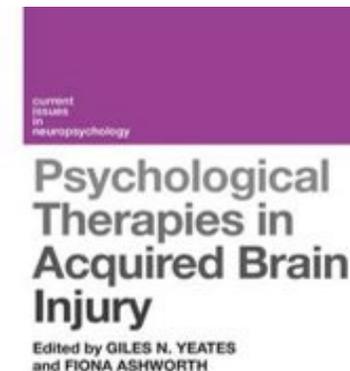
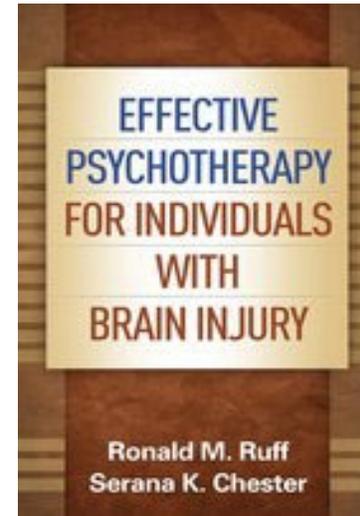
- Grief reaction
- Narrative therapy or identity reconstruction work
- Acceptance-based approaches (ACT)
- Values-guided goal setting
- Peer support groups for survivors
- Other therapies (e.g., music, art, equine)



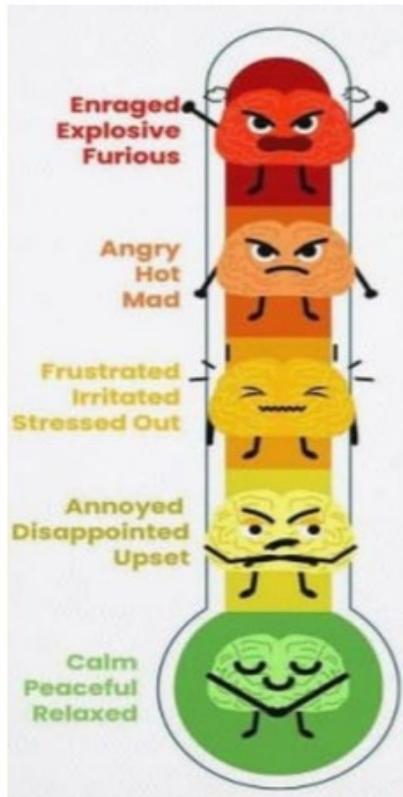
<https://blogs.und.edu/und-today/2025/01/unmasking-brain-injury/>

# Emotional and Behavioral Regulation

- Modify existing therapeutic modalities
  - Concrete
  - Simplified worksheets
  - Visual aids
  - Experiential
- Structured Problem-Solving Therapy
- Involve family members
  - Coping Ahead & DICE Framework



**1. STOP** Notice how I am feeling



**2. SLOW DOWN:** Lower the volume of my feelings



**3. THINK:** Why am I feeling this way?



Overstimulated



Change in my process



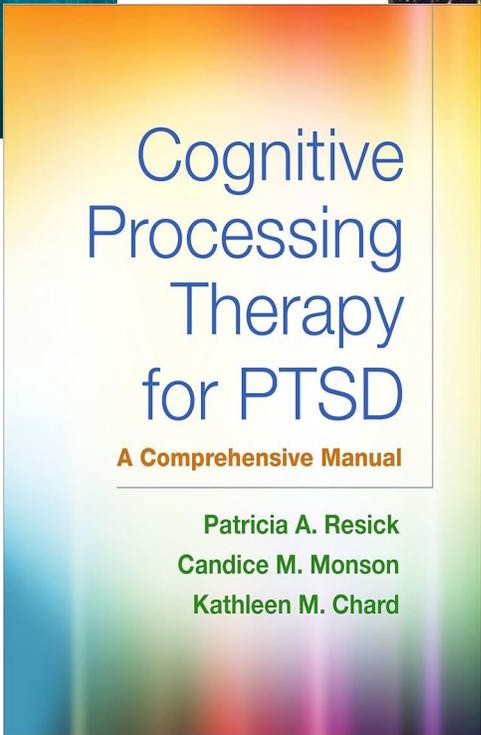
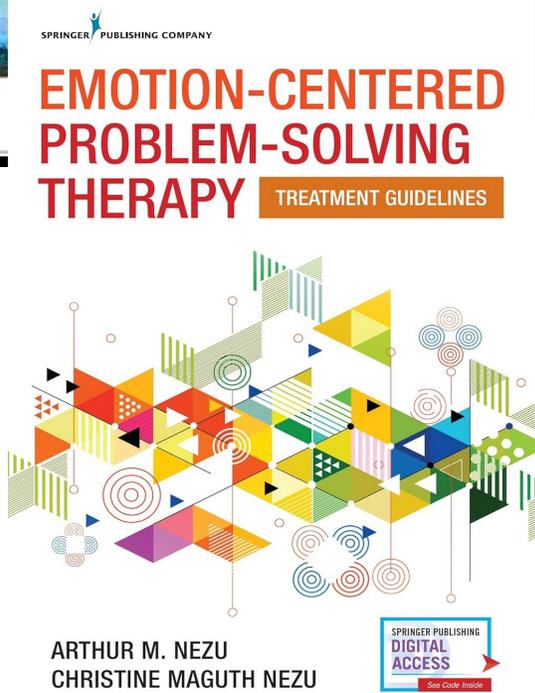
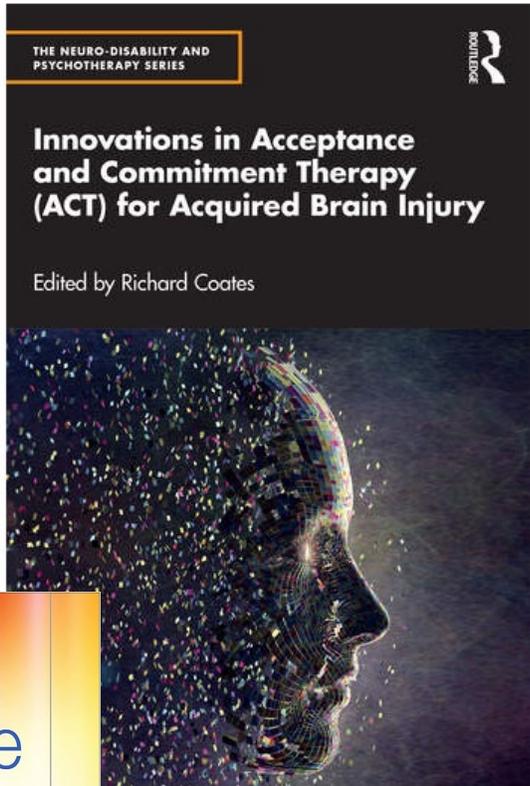
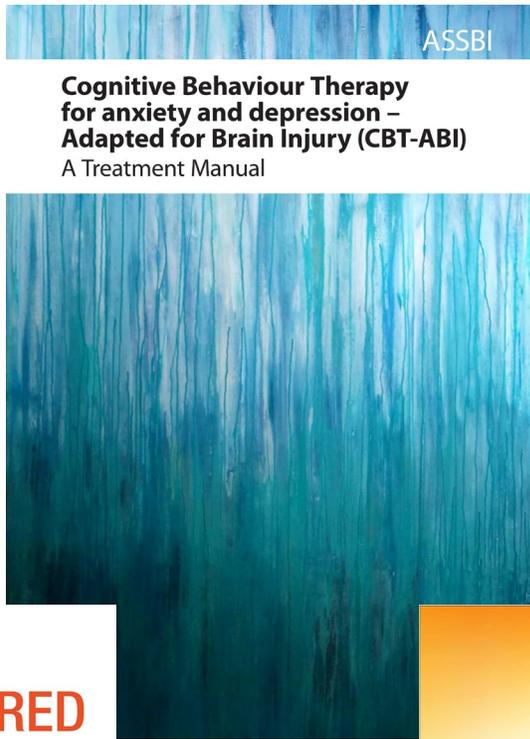
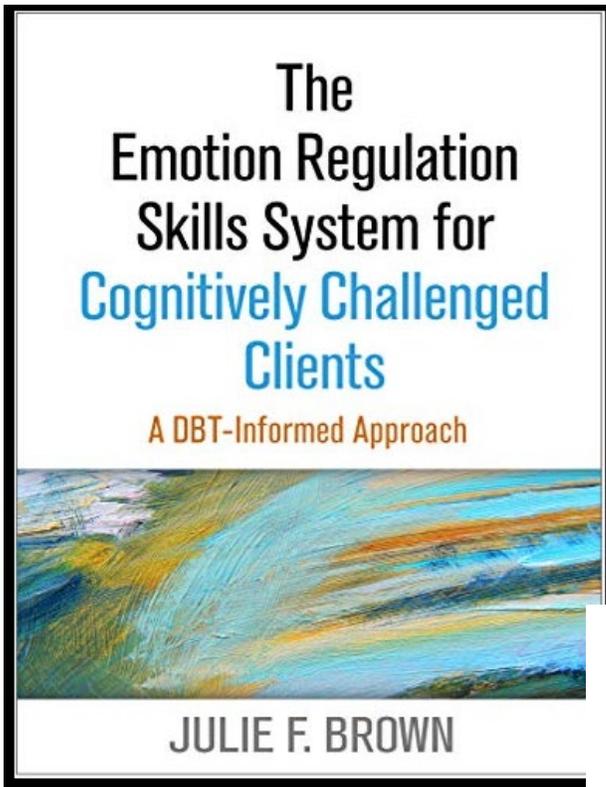
Not being understood



Feeling out of Control

Something else? Pain? Activity too hard?

<b>DESCRIBE BEHAVIOR:</b>	<b>IDENTIFY CAUSE:</b>
<b>WHAT HELPS TO REDUCE EMOTION/BEHAVIOR</b>	<b>WHAT WORSENS/ CONTRIBUTES</b>
<b>PLAN TO TRY:</b>	<b>EVALUATE: WHAT WORKED/DIDN'T</b>



# Post-concussive Syndrome

- Typically follows *mild* TBI
- Symptoms may be caused, worsened, or maintained by multiple etiologies
- Strong association with
  - Anxiety
  - Depression
  - Sleep Disturbance
  - Expectation Effects
- Treatment Emphasis
  - Psychoeducation on expected recovery
  - Graded return to activity & reduction of avoidance
  - CBT-based interventions
  - Treat comorbid mental health disorders

# Back to the case



David is a 45-year-old man with presents to your office with complaints of memory issues and depression. His medical history includes multiple prior concussions from playing sports throughout high school and college but a year ago he suffered from a TBI following a severe car accident for which he had a prolonged medical admission and recovery.

Since the accident, he describes poor sleep, depressed mood and struggles with motivation. He has been unable to return to work due to his cognitive struggles which he finds frustrating and finds himself withdrawing from his wife and friends. He finds himself more irritable, which was never an issue before.

What psychological struggles might David be dealing with?

What non-pharmacological interventions (if any) would you consider for David?

# Neuropsychiatric Sequelae of Brain Injury (BI)

**Personality changes**

**Mood changes**

**Anxiety**

**Psychosis**

**Agitation**



# Remember David?

David is a 45-year-old man who presents to your office with complaints of **memory issues and depression**. His medical history includes multiple prior concussions from playing sports throughout high school and college but a year ago he suffered from a TBI following a severe car accident for which he had a prolonged medical admission and recovery.

Since the accident, he describes **poor sleep, depressed mood and struggles with motivation**. He has been unable to return to work due to his cognitive struggles which he finds frustrating and finds himself **withdrawing** from his wife and friends. He finds himself **more irritable**, which was never an issue before.



# Pharmacologic Management for Neuropsychiatric Sequelae of BI

- Generally: Medications effective for primary psychiatric disorders in non-TBI patients are similarly effective in TBI patients.
  - le. Pharmacologic intervention usually parallels that of primary psychiatric syndromes in post-stroke and post-TBI neuropsych conditions
- BUT! This population is often more sensitive to medications and side effects so:
  - “start low, and go slow”
  - But avoid undertreating - being overly cautious may lead to inadequate medication trials if doses remain too low or medications are stopped too early
  - Goal is a well-tolerated titration & adequate duration

- DLPFC: Executive functioning (working memory, decision making, problem-solving, mental flexibility)
- Orbitofrontal cortex: social behaviors, ability self-monitor/self-correct
- Anterior cingulate: motivation, reward behaviors
- Hippocampus, amygdala: Association with PTSD?

**Table**

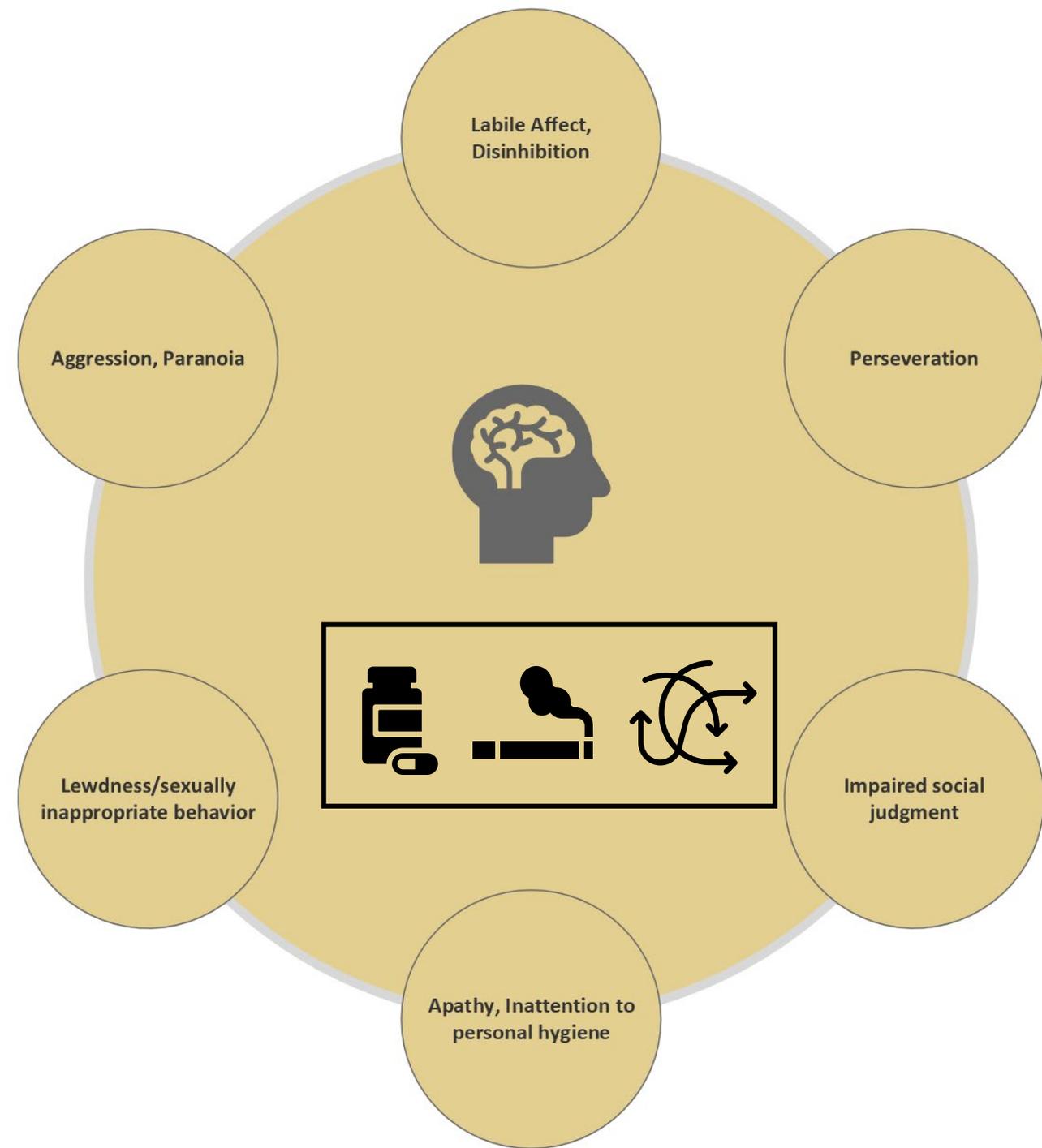
**Psychiatric symptoms of stroke by anatomic location**

Location		Symptoms
<b>Frontal lobe</b>	Lateral frontal lobe	Aprosodia, executive dysfunction, lack of empathy, poor self-regulation, trouble with attention
	Superior and inferior medial cortices	Apathy, lack of motivation, altered self-regulation, altered emotional processing, disinhibition
	Basal forebrain	Confabulation, reduced motivation, delusions (Capgras syndrome, reduplicative paramnesia)
	Orbital cortex	Personality changes, impulsivity, poor social judgment, reduced empathy, altered self-regulation, lack of goal-directed behavior
	Subcortical	Personality changes, reduced emotions, poor empathy, irritability
<b>Parietal lobe</b>	Dominant	Language deficits
	Nondominant	Neglect/inattention of the opposite side, denial of symptoms, anosodiaphoria, aprosodia, flat affect, personality changes
<b>Occipital lobe</b>		Visual hallucinations (colors, scotomas, metamorphopsia, palinoptic images, complex hallucinations, Charles Bonnet syndrome)
<b>Other locations</b>	Midbrain or thalamus	Peduncular hallucinosis
	Subthalamic nucleus	Hemiballismus, mood and behavioral disturbances, personality changes, hyperphagia
	Caudate	Dorsolateral caudate: abulia, psychic akinesia, decreased problem-solving ability, reduced abstract thinking, diminished spontaneity  Ventromedial caudate: disinhibition, disorganization, impulsiveness, affective symptoms with or without psychosis

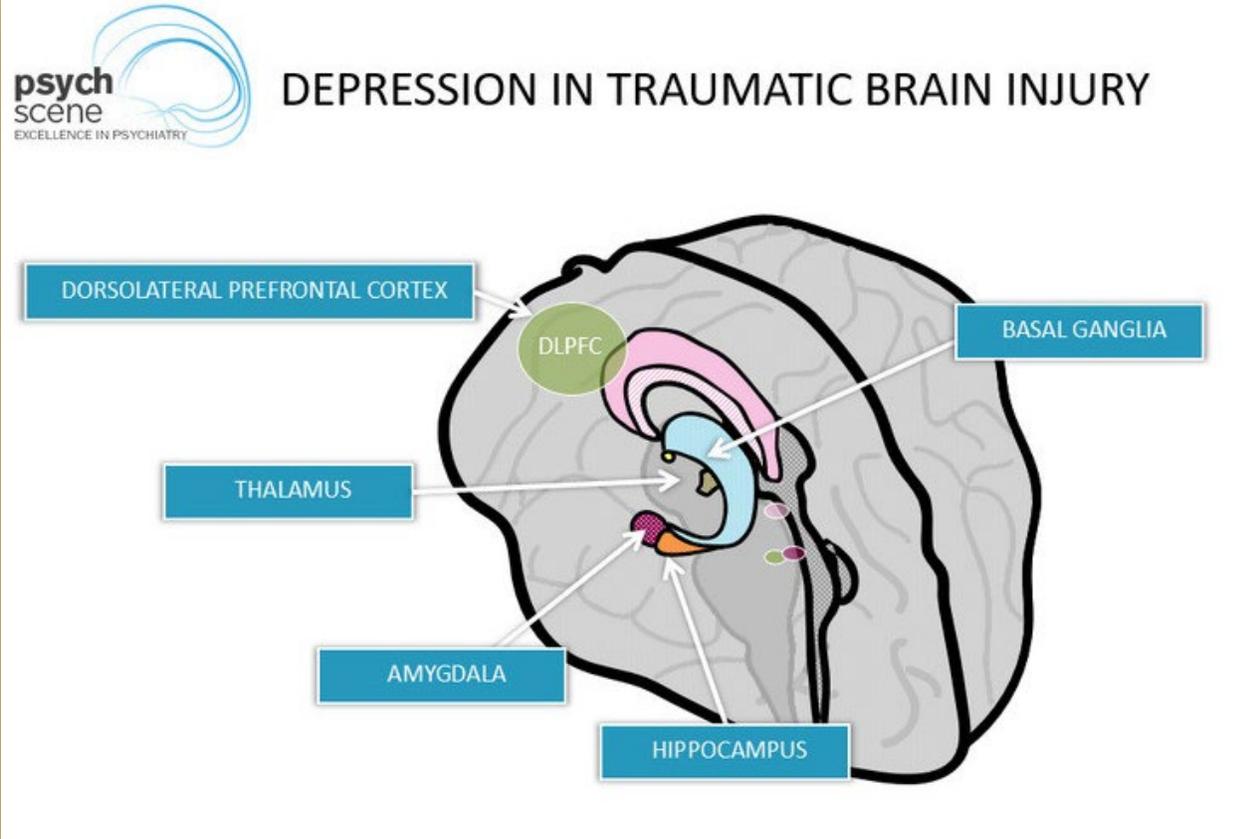
**Source:** References 1-10

# Personality Changes in BI

- Prior 30-year study found that almost  $\frac{1}{4}$  of patients following TBI acquired a personality disorder diagnosis following brain injury
- May have limited insight into personality changes
- Consider including family members and caregivers in the evaluation treatment planning



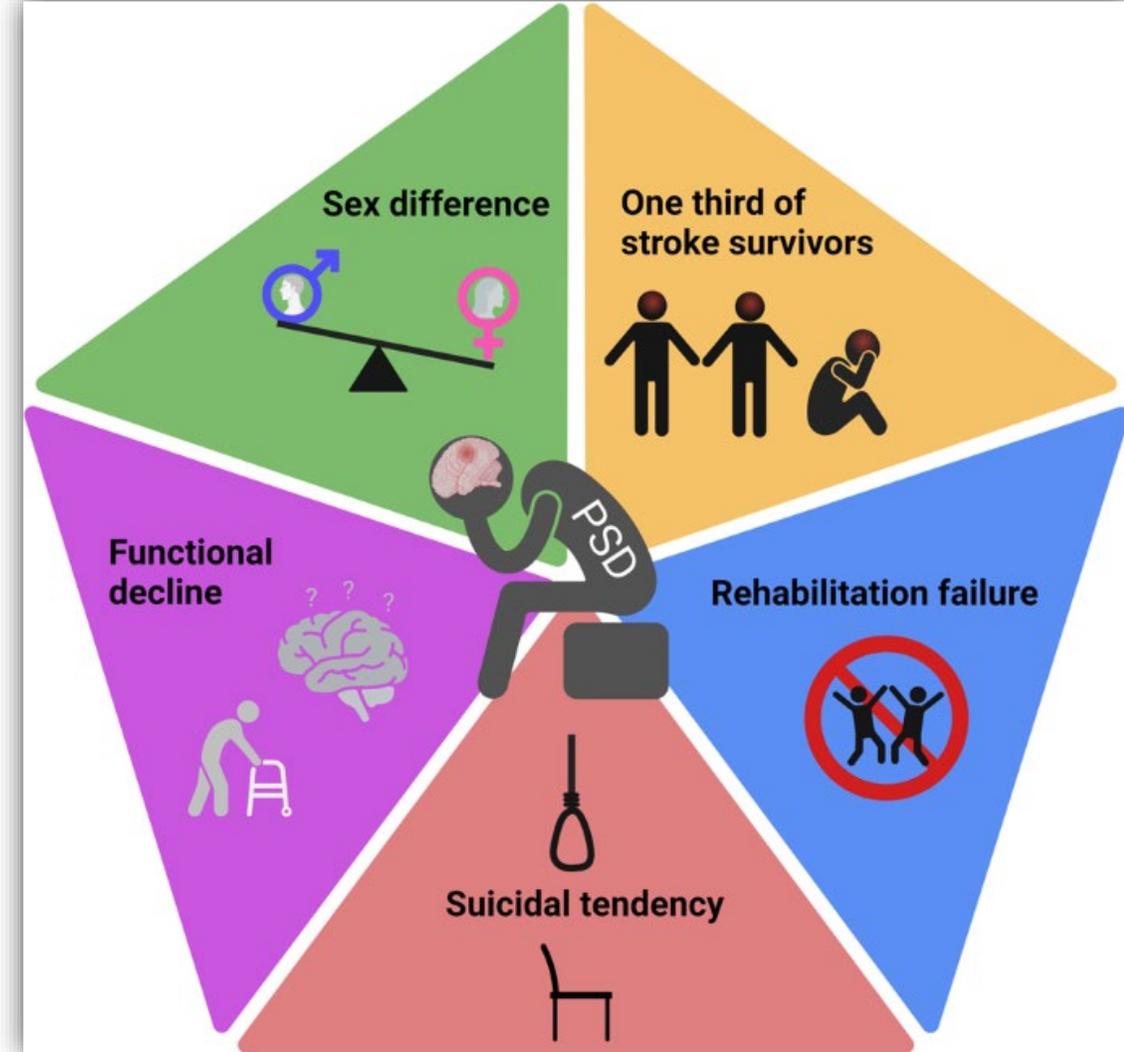
# Depression & TBI



- 26–77% in mild TBI (rate increases with injury severity)
- Predictive & risk factors: Pre-morbid substance abuse, poor functioning, lower education, unstable work history, Neuroanatomic/physiologic changes, lost capabilities, changes in roles, and psychosocial/financial stressors
  - Complicated by: Overlap with cognitive impairment & personality changes
- Associated with poor outcome across multiple domains
- Prominent signs/symptoms: Fatigue, distractibility, anger, irritability, and rumination.
- Suicide risk: Up to 15% of individuals make an attempt in the 5 years after TBI.
  - Risk factors: Intense despair, hopelessness, worthlessness, loss of sense of integrity, relationship breakdown, isolation, prominent insomnia and chronic headache
  - Combination of depression and disinhibition associated with frontal lobe injury

# Post-Stroke Depression

- Risk factors: History of depression, pre-stroke functional impairment, living alone, post-stroke social isolation, personal losses and possibly female gender
- Lesion location? (More controversial)
- Complicated by: Overlap between depression and other post-stroke sequelae like flattened affect, monotonous tone of voice, amotivation, decreased insight
  - Communication barriers like aphasia
  - Helpful to focus on specific diagnostic criteria like in the DSM-5 (guilt, worthlessness, hopelessness, anhedonia, suicidality)



# Pharmacologic Treatment of Depression in BI

- Often undertreated but early/effective intervention important given impact on recovery
- Antidepressants: Generally shown to be effective
  - Positive studies for SSRIs, SNRIs and nortriptyline
    - Mild – mod PSD: SSRIs generally tx of choice (efficacy, side effect profile, cardiovascular safety)
- Buspirone: Found to be helpful for depression, anxiety, irritability, aggression  
Very favorable safety profile
- ECT: Appears to be an effective treatment for PSD (high response rates, low complication rates but beware cognitive side effects!)

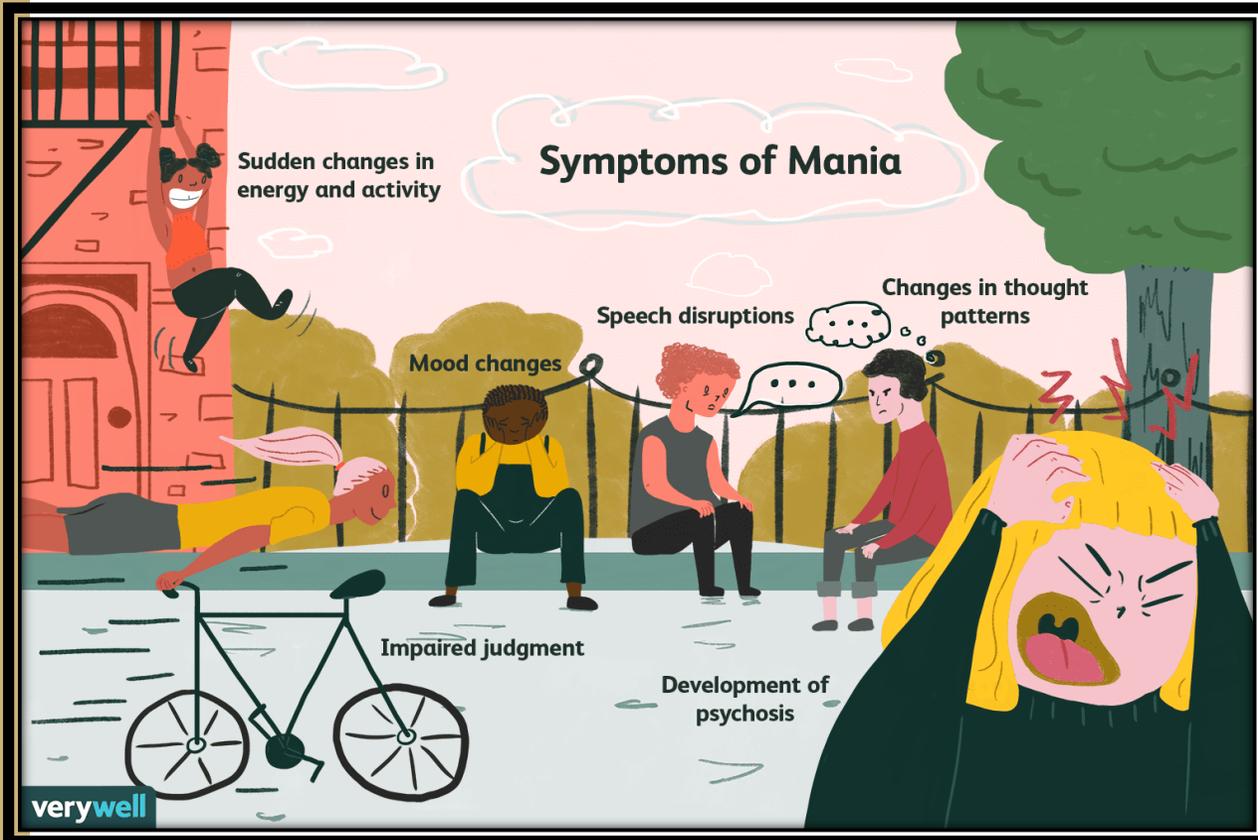
- Positive evidence for psychostimulants (ie. methylphenidate and dextroamphetamine) but have not been studied under placebo-controlled, double-blind conditions
  - Retrospective studies: Response rates 47-80%
- Can be helpful for apathy, depressed mood, cognitive symptoms, fatigue
- Mostly considered in more severe cases impairing MDM capacity, nutritional intake, ability to participate in rehabilitation
- Beware paradoxical reaction, agitation, paranoia, irritability

**Table 20-2**

### **Guidelines for the Use of Psychostimulants to Treat Depression**

1. Consider possible (relative) contraindications to psychostimulant use:
  - a. history of ventricular arrhythmia
  - b. recent myocardial infarction
  - c. congestive heart failure with reduced ejection fraction
  - d. poorly controlled hypertension
  - e. tachycardia
  - f. concurrent treatment with MAOIs.
2. Initiate treatment with a morning dose of 5 mg methylphenidate or dextroamphetamine (2.5 mg in frail elderly or medically tenuous patients).
3. Check vital signs and response to treatment in 2–4 hours (the period of peak effect).
4. If the initial dose is well tolerated and effective throughout the day, continue with a single daily morning dose.
5. If the initial dose is well tolerated and effective for several hours, with a loss of effect in the afternoon, give the same dose twice per day (in the morning and the early afternoon).
6. If the initial dose is well tolerated but is without significant clinical effect, increase dose by 5 mg per day until a clinical response is achieved, intolerable side effects arise, or 20 mg dose is ineffective (i.e., a failed trial).
7. Continue treatment throughout the hospitalization; stimulants can usually be discontinued at discharge.

# Mania in BI



- Similar to mania from bipolar disorder (ie. flight of ideas, pressured speech, a decreased need for sleep, grandiosity, and associated psychotic symptoms)

## TBI

- Increased rates of mania compared to the general population
  - Predisposing factors: Family history of affective illness, lesion/injury location (right temporal lobe , right orbitofrontal cortex injuries)
- Seizures also more common in this group --> EEG important + anticonvulsant mood stabilizers often preferred

## Post-Stroke

- Rarer (< 1% of patients).
- Lesions in the right orbitofrontal cortex, right basal temporal cortices, dorsomedial thalamus, and head of caudate appear to be associated most often with post-stroke mania
- Right hemisphere strokes > left?

# Psychosis in BI

- Psychosis 2/2 TBI considered relatively rare (rates between 0.7% and 9.8%)
  - Controversy over timeline between injury to onset of symptoms (immediately vs up to years later)
  - Relation with frontal & temporal lobe injuries; post-traumatic seizures
  - Symptoms can overlap with cognitive impairment and behavioral changes
  - Patients with primary psychotic disorders also found to have higher incidence of brain injury (exact relationship unclear)
- Post-stroke psychosis is also uncommon (~1–2%).
  - Usually have right temporoparietal lesions
    - Temporal lobe damage --> complex partial seizures + associated psychosis?
  - High rate of associated seizures.

# Pharmacologic Treatment of Mania in BI

- Mania should be treated using standard agents and following the same principles as treatment of mania from primary bipolar disorder.

Medication Class	Notes
Anticonvulsant mood stabilizers (ex. Valproic acid, carbamazepine)	<ul style="list-style-type: none"><li>• May be preferable (esp. In patients with co-morbid seizures)</li><li>• Patients with BI may have more side effects with Li</li></ul>
Neuroleptics/antipsychotics	<ul style="list-style-type: none"><li>• May be particularly helpful if presence of agitation/aggression</li><li>• Patients with BI have increased risk of EPS (ie. dystonias, akathisia, parkinsonism)</li><li>• Atypical (2nd gen) preferred over typical (1st gen)</li></ul>
Benzodiazepines	<ul style="list-style-type: none"><li>• May be helpful in acute phase</li><li>• Risk of disinhibition in BI</li></ul>

# Pharmacologic Treatment of Psychosis in BI

- Monitor for EPS
- Similar to mania management in BI: Typically prefer atypical > typical
- Use with caution/monitor closely: Antipsychotics with significant anticholinergic affects and/or those that more significantly decreased seizure threshold (ie. Clozapine)

# Anxiety in BI

TBI	Post-Stroke
<p data-bbox="183 444 1207 496">Most commonly reported: GAD, PTSD</p> <ul data-bbox="183 522 1100 568" style="list-style-type: none"><li data-bbox="183 522 1100 568">• 11%: GAD co-morbid with depression</li></ul> <p data-bbox="183 819 1233 1011">Some evidence that early intervention with CBT for acute stress disorder may prevent PTSD after mild TBI.</p> <ul data-bbox="183 1036 1172 1133" style="list-style-type: none"><li data-bbox="183 1036 1172 1133">• Impaired memory of traumatic event a/w lower rates of PTSD?</li></ul>	<p data-bbox="1324 444 2415 568">1/4 of PS patients meet criteria for GAD in acute post-stroke period</p> <ul data-bbox="1324 594 2191 691" style="list-style-type: none"><li data-bbox="1324 594 2191 691">• 3/4 of these patients have co-morbid depression</li></ul> <p data-bbox="1324 802 2288 993">A/w negative impact on functional recovery &amp; impaired ADL for up to 3 years post-stroke</p> <ul data-bbox="1324 1019 2018 1065" style="list-style-type: none"><li data-bbox="1324 1019 2018 1065">• PSD + PSA appears additive</li></ul>

# Pharmacologic Treatment of Anxiety in BI

- Generally can be treated like any primary anxiety syndrome.
- SSRIs are effective in the treatment of a variety of anxiety disorders
  - Often treatment of choice if co-morbid PSD
- Benzodiazepines:
  - Can be considered for isolated/infrequent anxiety
  - Monitor for ataxia, sedation, paradoxical disinhibition
  - Does not treat co-morbid depression



# Other post-stroke phenomena & Medications

	<b>Catastrophic Reaction</b>	<b>Pseudobulbar affect</b>
<b>Symptoms/Notes</b>	<ul style="list-style-type: none"><li>• Symptom collection: Desperation &amp; frustration</li><li>• Rates in acute phase: 3 – 20%</li><li>• ¾ also have PSD</li><li>• Risk factor: personal &amp; fam history of psychiatric disorders</li><li>• A/w anterior sub-cortical lesions &amp; left cortical lesions</li></ul>	<ul style="list-style-type: none"><li>• Frequent/easily triggered spells of laughing or crying</li><li>• 15% in post-stroke patients</li><li>• Milder cases: Briefer, can occur with appropriate changes in mood</li><li>• Severe cases: May be inappropriate to context and/or incongruent with mood</li><li>• Can cause: Embarrassment, social withdrawal, decreased QoL</li></ul>
<b>Rx Treatment</b>	<ul style="list-style-type: none"><li>• Consider antidepressants</li><li>• May improve with treatment of co-morbid PSD</li></ul>	<ul style="list-style-type: none"><li>• Consider antidepressants</li><li>• Nuedexta (dextromethorphan and quinidine)</li></ul>

What pharmacological interventions (if any) would you consider for David?



# Available Resources



University of Colorado  
Anschutz Medical Campus

## Psychiatric Consultation for the Medically Complex Program (PCMC)

- Time-limited (only 1 – 6 sessions with the psychiatrist), not for ongoing or long-term care
- Trainee-based clinic
- Generally need to be seen in person at least once but otherwise can do telehealth
- Patients must be located in Colorado at time of visit
- Offers group & individual therapy, cognitive screening and psychiatric evaluation and medication management
- Can refer via Epic:
  - Amb ref to psychiatry --> click PCMC option
  - For "referring to" field: Thida Thant for medication management or Holly Gerber for group/individual therapy
  - If patient does not meet criteria (or has exclusionary criteria), triage coordinators may schedule them in the general clinic

## About PCMC

The outpatient PCMC Program specializes in brief intervention services for patients who are typically referred by physicians in other medical specialties. PCMC services typically include evaluations and short-term treatment to assess mental health concerns and complications arising secondary to medical disorders or their treatment, assessment for potential organ transplantation or surgical interventions, evaluation for our chronic medical illness group, and psychopharmacological consultations in medically complex patients. Common comorbid medical conditions include diseases such as post-acute sequelae of COVID-19 (PASC), post intensive care syndrome (PICS), movement disorders, cancer, epilepsy, traumatic brain injury, neurocognitive disorders and endocrine dysfunction. PCMC is now also offering brief individual psychotherapy as well as a variety of group therapy offerings.

### Specialty Areas

- Patients who are experiencing psychiatric symptoms or complications secondary to medical disorders or treatments such as:
  - Endocrine disorders
  - Neurological disease (ie. stroke, TBI, Parkinson's disease, dementia, multiple sclerosis, epilepsy)
  - Deep-brain stimulator surgery
  - HIV
  - Transplant
  - Cancer
  - Post acute sequelae of COVID-19 (PASC or Long-Haulers syndrome)
  - Post intensive care syndrome (PICS)
- Patients interested in process and skill building psychotherapy groups for:
  - Post acute sequelae of COVID-19 (PASC or Long-Haulers syndrome)
- Coping with chronic illness
- Patients interested in brief psychotherapy to address challenges associated with medical disorders or treatments such as:
  - Coping with and adjusting to new medical diagnoses
  - Coping with progression or complication of pre-existing medical conditions
  - Traumatic injuries (burn, spinal cord injury, etc.)
  - Medical trauma as a result of treatment for medical conditions

# How to decide who/when to refer to PCMC?

Also typically unable to accept primary FND or somatic symptom disorder patients due to time-limited nature of clinic

## Who we see

- Patients who are experiencing psychiatric symptoms or complications secondary to medical disorders or treatments such as endocrine disorders, neurological disease (ie. stroke, TBI, Parkinson's disease, dementia, multiple sclerosis), deep-brain stimulator surgery, HIV, transplant or cancer.
- Patients who may benefit from specialized psychopharmacological consultations such as medically complex or geriatric patients.
- Patients who are interested in participating in a 9-week psychoeducational and skill building group about coping with chronic medical illness or psychotherapy groups for COVID-19 survivors
- Post-ICU and post-COVID patients experiencing associated mental health difficulties including the need for cognitive screening
- Patients who would benefit from brief psychotherapy to address challenges associated with medical disorders or treatments such as:
  - Coping with and adjusting to new medical diagnoses
  - Coping with progression or complication of pre-existing medical conditions
  - Traumatic injuries (burn, spinal cord injury, etc.)
  - Medical trauma as a result of treatment for medical conditions

## Who would *not* benefit?

- Patients who will need long-term psychiatric care exceeding 1-6 medication sessions or brief psychotherapy
- Patients seeking general psychiatric care
- Patients with high psychiatric case management needs or who require involuntary outpatient treatment
- Patients experiencing acute safety concerns that would be better served by emergency evaluation





Time-limited format (9 sessions) focused on skill-building and education

Curriculum Overview	
1: Chronic illness education, living with chronic illness, introduction to mindful breathing	6: Social support and connection, meditation practice
2: Values clarification, ongoing mindfulness practice	7: Trauma, resilience & gratitude in chronic illness, meditation practice
3: Emotion identification and problem solving, body scan practice	8: Meaning-centered living, meditation practice
4: "Unhelpful thoughts" intro, meditation practice	9: Final reflection and wrap-up
5: Communication in chronic illness, meditation practice	

**Coping with Illness Therapy Group**

The word cloud includes the following terms: Active, Balance, Values, Intellectual, Nutrition, Time, Sleep, Commitment, Exercise, Wellness, Insight, Support, Spirituality, Healthy, Lifestyle, Learn, Physical, Vitality, Involved, Vegetables, Relaxation, Persistence, Mindfulness, Walking, Preventive, Habits, Social, Choices, Fruits, Eating, Fitness.

Workbook and facilitator guide available for free download [here](#)



Effective Access and Support from psychiatry  
Healthcare Consultation Line



Colorado Pediatric Psychiatry  
Consultation & Access Program



PERINATAL MENTAL HEALTH & SUBSTANCE USE  
CONSULTING + ACCESS PROGRAM

**Mission:** To increase mental health identification and support strategies for primary and other healthcare providers. Increase provider-to-provider mental health consultation, education, and support services to frontline healthcare workers treating patients across the lifespan.

**Contact Us:** **1-888-910-0153** (call for consultation or resources) or  
[COMAP@cuanschutz.edu](mailto:COMAP@cuanschutz.edu)

- CoPPCAP – [info@coppcap.org](mailto:info@coppcap.org)
- EASY – [easyconsultation@cuanschutz.edu](mailto:easyconsultation@cuanschutz.edu)
- PROSPER – [prosper@ucdenver.edu](mailto:prosper@ucdenver.edu)

**Things to remember:**

- FREE service for medical providers & navigators (including BH providers)
- Support any provider in CO
- Payor blind (i.e. insurance status not a factor)
- Not taking over patient care
- Not an emergency service
- Grant and gift funded



# CO-MAP Components

## Psychiatric Consultation:

- Access line for free provider-to-provider consultation
- E-consults; asynchronous contact

## Education:

- ECHOs & other educational forums
- Website/Toolkits for resources

## Referral & Support:

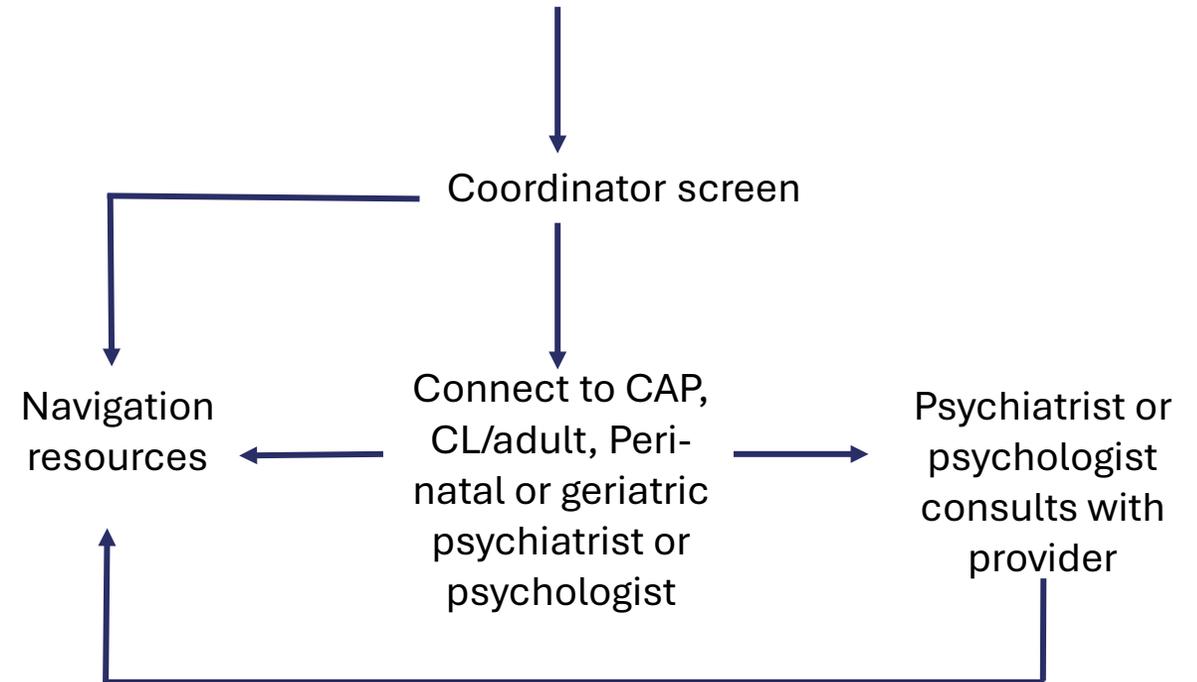
- Curated mental health resources & other community supports

University providers/team members with Epic access can also contact us via Epic!

- Send staff message with patient chart attached and consult question to "CO-MAP" inbasket pool

Non-UCH providers can contact us via our [website consult form](#)

Call, eMail or eConsult  
from provider/clinician



Call Us Now Toll-FREE:

1-888-910-0153

# In Summary

- Neuropsychiatric symptoms after stroke and TBI are common, can mimic primary psychiatric conditions and can include mood changes, anxiety, personality changes, agitation, psychosis
  - Can have significant impact on the long-term outcome of both TBI & post-stroke patients
- A multidisciplinary and rehabilitative approach is important and should include families/caregivers
- When it comes to psychological considerations
  - Identity is shaped by personal experience and sociocultural context; brain injury can disrupt both how individuals see themselves and how they function within their social world.
  - Communication style, treatment plans, and therapy approaches need to be tailored to a patient's unique abilities and needs
- When it comes to medications:
  - “start low and go slow” but ensure adequate dosing and duration of medication trials
  - Minimize polypharmacy
- Providers can access curbside consultation through CO-MAP for medically complex patients or outreach the PCMC clinic if considering a referral





**QUESTIONS?**

**CASES?**

**Contact information:**

[Thida.thant@cuanschultz.edu](mailto:Thida.thant@cuanschultz.edu)

[Holly.gerber@cuanschultz.edu](mailto:Holly.gerber@cuanschultz.edu)

# Supplementary Slides



University of Colorado  
Anschutz Medical Campus

<b>Class</b>	<b>Highlights</b>	<b>*DDI = drug-drug interaction</b>
Selective serotonin reuptake inhibitors (SSRIs)		
Sertraline (Zoloft)	Tends to cause GI SE, relatively low risk of DDI	
Escitalopram (Lexapro)	Similar to citalopram, generally well tolerated, low risk of DDI, smaller dosing range*	
Citalopram (Celexa)	Low risk of DDI, more dosing restrictions, QTc (black box)	
Fluoxetine (Prozac)	Long half-life (helpful in intermittent adherence), high risk for DDI, can be activating	
Paroxetine (Paxil IR, CR)	High anticholinergic & antihistaminic burden (sedation, weight gain, dry mouth), short half-life (higher risk of discontinuation syndrome), high risk for DDI, not recommended in pregnancy, problematic in the elderly	
Fluvoxamine (Luvox)	Short half-life, not well tolerated, DDI	
Serotonin norepinephrine reuptake inhibitors (SNRIs)		
Venlafaxine (Effexor IR, XR)	Less sedating, adjunct in chronic pain, can be problematic in patients with HTN (inc BP & HR), IR formulation a/w more side effects and discontinuation syndrome	
Desvenlafaxine (Pristiq)	Similar to venlafaxine	
Duloxetine (Cymbalta)	Less sedating, FDA approved for fibromyalgia & DM related neuropathic pain, shorter half-life, increased risk of DDI	
Other		
Bupropion (Wellbutrin IR, SR, XL)	Increase seizure risk (IR/SR especially contraindicated in seizure, eating disorder), not recommended for anxiety disorders or depression with anxiety, no serotonin effect or sexual SE, XL supposed to be better tolerated, DA & NE effect	
Mirtazapine (Remeron)	Serotonin & NE effect, less risk of sexual SE, lower doses more a/w sedation/sleepiness, may be helpful in anxiety, has dissolvable form	

# References



- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. American Psychiatric Association: Arlington; 2013.
- Andersen G. Treatment of uncontrolled crying after stroke. *Drug Ther.* 1999;6:105–111.
- Astrom M. Generalized anxiety disorder in stroke patients: a three-year longitudinal study. *Stroke.* 1996;27:270–275.
- Bartoli F, Lillia N, Lax A, et al. Depression after stroke and risk of mortality: a systematic review and meta-analysis. *Stroke Res Treat.* 2013;2013:862978.
- Bouvy P, van der Wetering B, Meerwaldt J, et al. Buspirone: neuropsychiatric effects. *J Head Trauma Rehabil.* 1991;6:90–92.
- Bryant RA, Moulds M, Guthrie R, et al. Treating acute stress disorder following mild traumatic brain injury. *Am J Psychiatry.* 2003;160:585–587.
- Carota A, Rossetti AO, Karapanayiotides T, et al. Catastrophic reaction in acute stroke: a reflex behavior in aphasic patients. *Neurology.* 2001;57:1902–1905.
- Castillo CS, Schultz SK, Robinson RG. Clinical correlates of early-onset and late-onset poststroke generalized anxiety. *Am J Psychiatry.* 1995;152:1174–1179.
- Centers for Disease Control and Prevention. (2024, October 29). TBI data. Centers for Disease Control and Prevention.
- Chemerinski E, Levine SR. Neuropsychiatric disorders following vascular brain injury. *Mt Sinai J Med.* 2006;73:1006–1014.
- Corcoran C, McAllister TW, Malaspina D. Psychotic disorders. In: Silver JM, McAllister TW, Yudofsky SC. *Textbook of Traumatic Brain Injury.* American Psychiatric Publishing: Washington, DC; 2011.
- Critical Care and Traumatic Brain Injury - Academy of Consultation-Liaison Psychiatry. (2024, May 31). Academy of Consultation-Liaison Psychiatry.
- Cummings JL, Mendez MF. Secondary mania with focal cerebrovascular lesion. *Am J Psychiatry.* 1984;141:1084–1087.
- Currier MB, Murray GB, Welch CC. Electroconvulsive therapy for poststroke depressed geriatric patients. *J Neuropsychiatry Clin Neurosci.* 1992;4:140–144.
- Deb S, Crownshaw T. The role of pharmacotherapy in the management of behavior disorders in traumatic brain injury patients. *Brain Inj.* 2004;18:1–31.
- Deb S, Lyons I, Koutzoukis C, et al. Rate of psychiatric illness one year after traumatic brain injury. *Am J Psychiatry.* 1999;156:374–378.
- Dikman SS, Bombardier CH, Machamer JE, et al. Natural history of depression in traumatic brain injury. *Arch Phys Med Rehabil.* 2004;85:1457–1464.
- Duncan KR. Neuropsychiatric symptoms after stroke. *Current Psychiatry.* 2022;21(9).
- Elovic EP, Lansang R, Li Y, et al. The use of atypical antipsychotics in traumatic brain injury. *J Head Trauma Rehabil.* 2003;18:177–195.
- Fujii DE, Ahmed I. Risk factors in psychosis secondary to traumatic brain injury. *J Neuropsychiatry Clin Neurosci.* 2001;13:61–69.
- Goyal R, Sameer M, Chandrasekaran R. Mania secondary to right-sided stroke responsive to olanzapine. *Gen Hosp Psychiatry.* 2006;28:262–263.

- Hackett ML, Anderson CS, Hourse A, et al. Interventions for treating depression after stroke. *Cochrane Database Syst Rev.* 2008;(4):CD003437.
- Hackett ML, Kohler S, O'Brien JT, et al. Neuropsychiatric outcomes of stroke. *Lancet Neurol.* 2014;13:525–534.
- Hackett ML, Yapa C, Parag V. Frequency of depression after stroke: a systematic review of observational studies. *Stroke.* 2005;36:1330–1340.
- Hama S, Yamashita H, Shigenobu M, et al. Depression or apathy and functional recovery after stroke. *Int J Geriatr Psychiatry.* 2007;22:1046–1051.
- Howlett, Nelson, & Stein (2023). Mental health consequences of traumatic brain injury, *Biological Psychiatry*, 91(5), 413-420. doi: 10.1016/j.biopsych.2021.09.024
- Jeong S, Chokkalla AK, Davis CK, et al. Post-stroke depression: epigenetic and epitranscriptomic modifications and their interplay with gut microbiota. *Mol Psychiatry.* 2023;28:4044–4055.
- Jonasson et al. (2018). Mental fatigue and impaired cognitive function after an acquired brain injury, *Brain and Behavior*, 8(8), doi: 10.1002/brb3.1056
- Kim E, Bijlani M. A pilot study of quetiapine treatment of aggression due to traumatic brain injury. *J Neuropsychiatry Clin Neurosci.* 2006;18:547–549.
- Klein E, Caspi Y, Gil S. The relation between memory of the traumatic event and PTSD: evidence from studies of traumatic brain injury. *Can J Psychiatry.* 2003;48:28–33.
- Kohler S, Verhey F, Weyerer S, et al. Depression, non-fatal stroke and all-cause mortality in old age: a prospective cohort study of primary care patients. *J Affect Disord.* 2013;150:63–69.
- Koponen S, Taiminen T, Portin R, et al. Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. *Am J Psychiatry.* 2002;159:1315–1321.
- Kraus FJ, Chu LD. Epidemiology. In: Silver JM, McAllister TW, Yudofsky SC. *Textbook of Traumatic Brain Injury.* 2nd ed. American Psychiatric Publishing: Washington, DC; 2011.
- Kuipers P, Lancaster A. Developing a suicide prevention strategy based on the perspectives of people with brain injuries. *J Head Trauma Rehabil.* 2000;15:1275–1284.
- Levine DN, Finklestein S. Delayed psychosis after right temporoparietal stroke or trauma: relation to epilepsy. *Neurology.* 1982;32:267–272.
- Lieberman A, Benson DF. Control of emotional expression in pseudobulbar palsy. A personal experience. *Arch Neurol.* 1977;34:717–719.
- Lipsey JR, Robinson RG, Pearlson GD, et al. Nortriptyline treatment of poststroke depression: a double-blind study. *Lancet.* 1984;1:297–300.
- Masand P, Murray GB, Pickett P. Psychostimulants in poststroke depression. *J Neuropsychiatry Clin Neurosci.* 1991;3:23–27.
- Morris PLP, Robinson RG, Raphael B. Emotional lability after stroke. *Aust N Z J Psychiatry.* 1993;27:601–605.
- Morris, S.D. (2004). Rebuilding identity through narrative following traumatic brain injury. *J of Cognitive Rehabilitation.* Retrieved from: <https://www.neuropsychonline.com/loni/jcrarchives/vol22/V22I2Morris.pdf>
- Morrison JH, Molliver ME, Grzanna R. Noradrenergic innervation of the cerebral cortex: widespread effects of local cortical lesions. *Science.* 1979;205:313–316.
- *Neuropsychiatry of Traumatic Brain Injury (TBI)-Head Injury and Psychiatry.* (n.d.). Psych Scene Hub.
- Ouimet MA, Primeau F, Cole MG. Psychosocial risk factors in poststroke depression: a systematic review. *Can J Psychiatry.* 2001;46:819–828.
- Parikh RM, Robinson RG, Lipsey JR, et al. The impact of post-stroke depression on recovery of activities of daily living over two year follow-up. *Arch Neurol.* 1990;47:1000–1003.

- Pourcher E, Filteau M, Bouchard R, et al. Efficacy of the combination of buspirone and carbamazepine in early posttraumatic delirium (letter). *Am J Psychiatry*. 1994;151:150–151.
- Quinn, D. K., Mayer, A. R., Master, C. L., & Fann, J. R. (2018). Prolonged Post concussive Symptoms. *The American journal of psychiatry*, 175(2), 103–111. <https://doi.org/10.1176/appi.ajp.2017.17020235>
- Rabins PV, Starkstein SE, Robinson RG. Risk factors for developing atypical (schizophreniform) psychosis following stroke. *J Neuropsychiatry Clin Neurosci*. 1991;3:6–9.
- Ratey J, Leveroni C, Miller A, et al. Low-dose buspirone to treat agitation and maladaptive behaviour in brain-injured patients: two case reports (letter). *J Clin Psychopharmacol*. 1992;12:362–364.
- Rapoport MJ, McCullagh S, Streiner D, et al. The clinical significance of major depression following mild traumatic brain injury. *Psychosomatics*. 2003;44:31–37.
- Robinson RG. Mood disorders secondary to stroke. *Semin Clin Neuropsychiatry*. 1997;2:244–251.
- Robinson RG, Boston JD, Starkstein SE, et al. Comparison of mania with depression following brain injury: causal factors. *Am J Psychiatry*. 1988;145:172–178.
- Robinson RG, Jorge RE. Mood disorders. In: Silver JM, McAllister TW, Yudofsky SC. *Textbook of Traumatic Brain Injury*. American Psychiatric Publishing: Washington, DC; 2011.
- Sander, A. M., Clark, A. N., Arciniegas, D. B., Tran, K., Leon-Novelo, L., Ngan, E., ... Walser, R. (2021). A randomized controlled trial of acceptance and commitment therapy for psychological distress among persons with traumatic brain injury. *Neuropsychological Rehabilitation*, 31(7), 1105–1129. <https://doi.org/10.1080/09602011.2020.1762670>
- Seel RT, Kreutzer JS, Rosenthal M, et al. Depression after traumatic brain injury: a National Institute on Disability and Rehabilitation Research Model Systems multicenter investigation. *Arch Phys Med Rehabil*. 2003;84:177–184.
- Silver JM, Arciniegas DB, Yudofsky SC. Psychopharmacology. In: Silver JM, McAllister TW, Yudofsky SC. *Textbook of Traumatic Brain Injury*. American Psychiatric Publishing: Washington, DC; 2011.
- Silverberg, N. D., Iverson, G. L., & Caplan, B. (2013). Cognitive-behavioral management of persistent post-concussive symptoms. *Journal of Head Trauma Rehabilitation*, 28(4), 299–304.
- Simpson G, Tate R. Clinical features of suicide attempts after traumatic brain injury. *J Nerv Ment Dis*. 2005;193:680–685.
- Starkstein SE, Fedoroff JP, Price TR, et al. Catastrophic reaction after cerebrovascular lesions: frequency, correlates, and validation of a scale. *J Neurol Neurosurg Psychiatry*. 1993;56:189–194.
- Starkstein SE, Mayberg HS, Berthier ML, et al. Secondary mania: neuroradiological and metabolic findings. *Ann Neurol*. 1990;27:652–659.
- Starkstein SE, Robinson RG, Price TC. Comparison of cortical and subcortical lesions in the production of post-stroke depression matched for size and location of lesions. *Arch Gen Psychiatry*. 1988;45:247–252.
- Stengler-Wenzke K, Muller U. Fluoxetine for OCD after brain injury. *Am J Psychiatry*. 2002;159:872.
- Stern TA. *Massachusetts General Hospital Handbook of General Hospital Psychiatry*. 2018.
- van Reekum R, Cohen T, Wong J. Can traumatic brain injury cause psychiatric disorders? *J Neuropsychiatry Clin Neurosci*. 2000;12:316–327.
- Villa, Causer, & Riley (2021). Experiences that challenge self-identity following traumatic brain injury: a meta-synthesis of qualitative research, *Disability Rehabilitation*,
- Wijeratne C, Malhi GS. Vascular mania: an old concept in danger of sclerosing? A clinical overview. *Acta Psychiatr Scand*. 2007;116(suppl 434):35–40.
- Waltzman D, Black LI, Daugherty J, Peterson AB, Zablotsky B. Prevalence of traumatic brain injury among adults and children. *Ann Epidemiol*. 2025;103:40–47.