

# ERABI

EVIDENCE-BASED REVIEW  
of moderate to severe  
ACQUIRED BRAIN INJURY

## Clinical Guidebook

### 5. Mental Health Following Acquired Brain Injury

Pavlina Faltynek MSc, Laura Rees PhD, Ali Bateman MD, Shannon Janzen MSc,  
Robert Teasell MD FRCPC



## Table of Contents

<b>5.1 Introduction to Neurobehavioural Disorders Post ABI</b> .....	3
<b>5.2 Clinical Presentation of Neurobehavioral Sequelae</b> .....	5
5.2.1 Depression .....	5
5.2.2 Anxiety Disorders .....	6
5.2.3 Agitation and Aggression .....	7
5.2.4 Addictive Behaviours .....	8
5.2.5 Suicidal Ideation .....	10
<b>5.3 Outcome Measures and Assessments</b> .....	11
5.3.1 Agitated Behavior Scale .....	11
5.3.2 Overt Behavior Scale .....	12
5.3.3 Patient Health Questionnaire .....	13
5.3.4 Hospital Anxiety and Depression Scale .....	14
5.3.5 Generalized Anxiety Disorder 7-Item Scale .....	15
<b>5.4 Criteria for Diagnosis</b> .....	15
<b>5.5 Interventions for Mental Health Disorders Following an ABI</b> .....	16
5.5.1 Interventions for Depression .....	17
5.5.1.1. Non-Pharmacological Interventions .....	18
5.5.1.2 Pharmacological Interventions .....	21
5.5.2 Anxiety Disorders .....	22
5.5.2.1 Non-Pharmacological Interventions .....	22
5.5.2.2 Pharmacological Interventions .....	23
5.5.3 Interventions for Agitation and Aggression .....	23
5.5.3.1 Non-pharmacological Interventions .....	23
5.5.3.2 Pharmacological Interventions .....	24
5.5.3.3 Use of Restraints .....	26
5.5.4 Addictive Behaviors .....	27
5.5.5 Suicidal Ideation .....	28
<b>5.6 Case Study</b> .....	30
<b>5.7 References</b> .....	34

## 5. Mental Health Following Acquired Brain Injury

### By the end of this chapter you should know:

- The most common mental health concerns following an ABI.
- How to identify neurobehavioral sequelae in individuals with an ABI.
- Common assessments for evaluating these deficits and dysfunctions.
- Evidence-based interventions for treating depression, anxiety, and other challenging behaviors.
- Potential case management scenarios.

### 5.1 Introduction to Neurobehavioural Disorders Post ABI

One of the many functional domains that can be affected by an acquired brain injury is mental health. Mental health refers to our mood, personality, emotional regulation, and behavior (McAllister, 2013). Following an acquired brain injury (ABI), individuals may suffer from mood disorders such as major depression, post-traumatic stress disorder and generalized anxiety. Challenging behaviours such as agitation and aggression, as well as addictive behaviours such as substance abuse, may also become a significant problem post-ABI. Mental health issues are associated with worsening of other ABI sequela and poorer outcomes over all (Bedard et al., 2003; Berthier et al., 2001; Jorge, 2005).

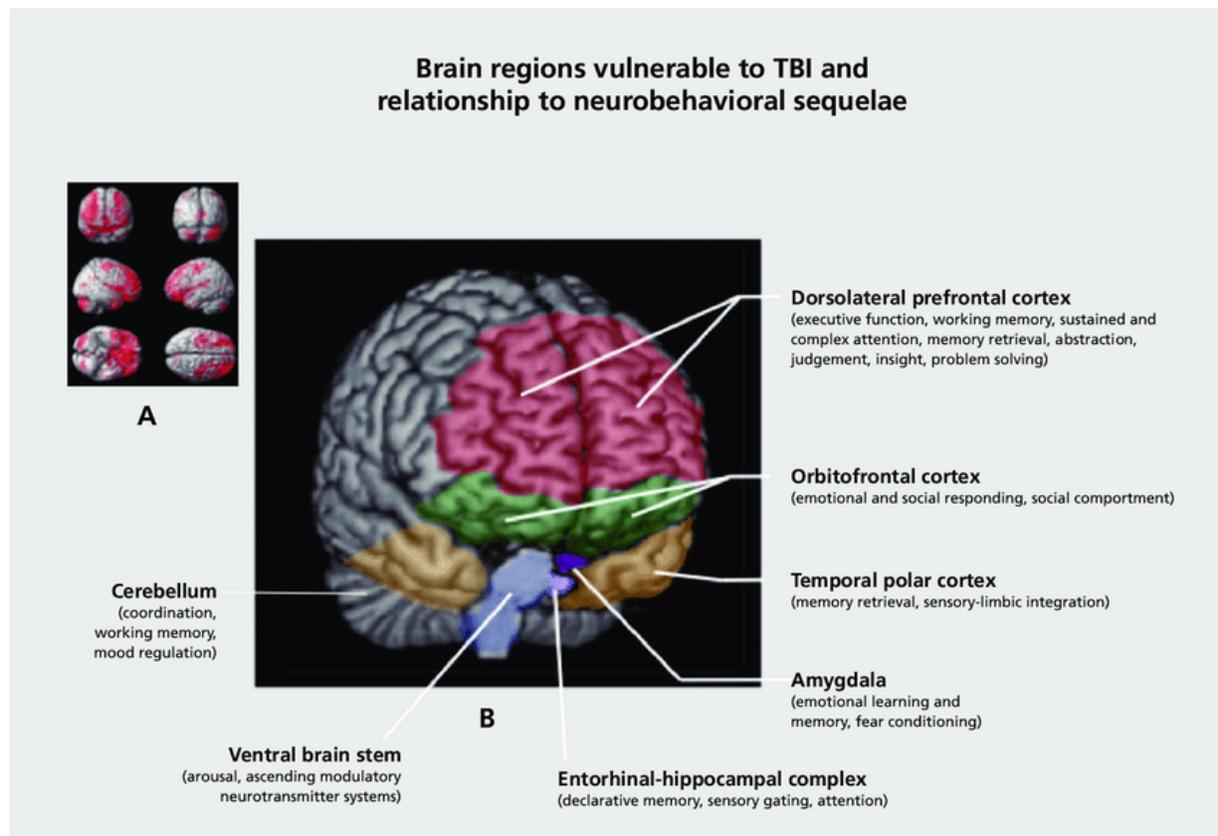


Figure 5.1 Brain regions typically damaged in traumatic brain injuries (A); Brain regions associated with neurobehavioral sequelae and their relationship to commonly damaged regions of the brain ((A) Adapted from ref 112: Bigler E. Structural imaging In: Silver J, McAllister T, Yudofsky S, eds. Textbook of Traumatic Brain Injury. Washington DC: American Psychiatric Press; 2005:87. Copyright © American Psychiatric Press, 2005. (B) Adapted from ref 111: Arciniegas DB, Beresford TP. Neuropsychiatry: an Introductory Approach. Cambridge, UK: Cambridge University Press; 2001:58. Copyright © Cambridge University Press, 2001; (McAllister, 2011)).

Changes in mood and emotional states result from alterations to the flow of excitatory and inhibitory neurotransmitters such as epinephrine, norepinephrine, dopamine, acetylcholine, and serotonin. Three major frontal and subcortical circuits have been identified that help modulate human emotion and behavior: the dorsolateral frontal-subcortical circuit, the lateral orbitofrontal-subcortical circuit, and the anterior cingulate-subcortical circuit. The name for each circuit comes from its location in the frontal cortex (McAllister, 2013). The relevant pathway of each circuit is illustrated in Figure 5.2.

When trying to understand the complex interplay between the external expression of emotion and internal regulatory mechanisms, it is useful to think of the brain as a vast network of overlapping neural circuitry connecting the frontal cortex, temporal cortex, hippocampus, thalamus, and globus pallidus (McAllister, 2013).

**Q1. Which 3 regions of the brain have been associated with the presence of mood or emotional disorders in individuals with an ABI?**

1. The dorsolateral frontal-subcortical region.
2. The lateral orbitofrontal-subcortical region.
3. Anterior cingulate-subcortical region.

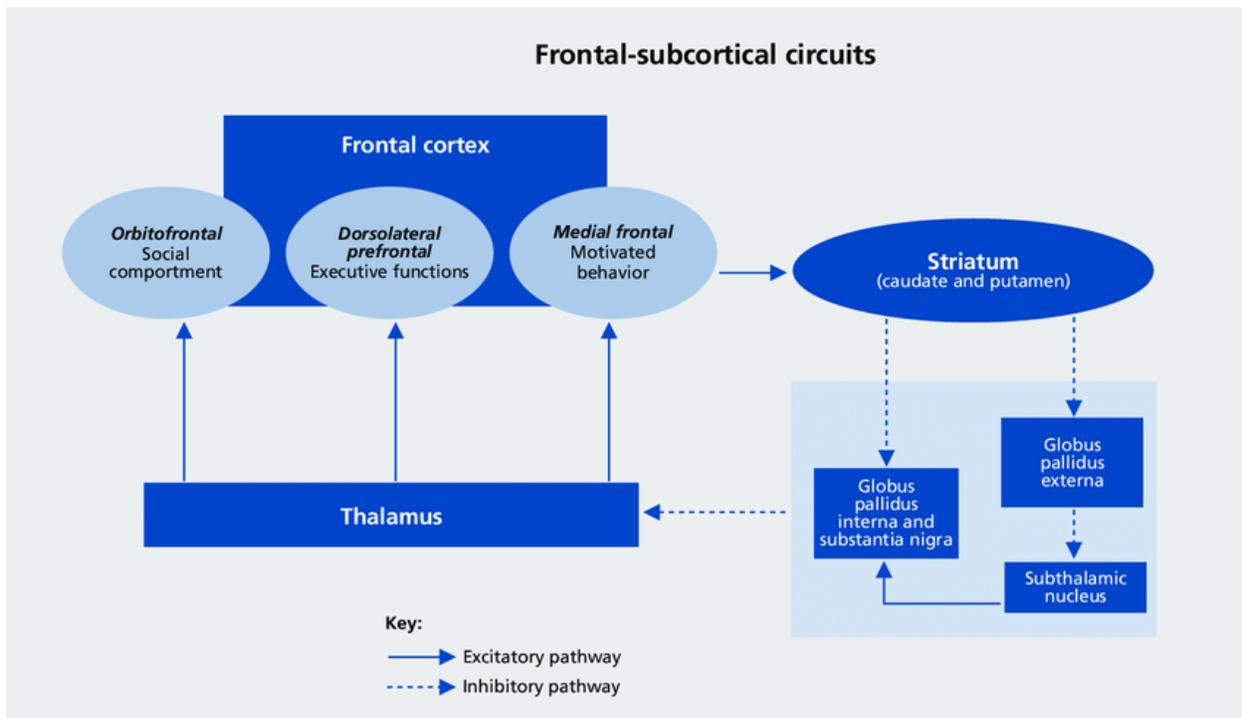


Figure 5.2 Diagram of neural circuits related to neurobehavioral sequelae following an ABI (Adapted from ref 111: Arciniegas DB, Beresford TP. Neuropsychiatry: an introductory Approach. Cambridge, UK: Cambridge University Press; 2001:58. Copyright © Cambridge University Press, 2001; (McAllister, 2011)).

## 5.2 Clinical Presentation of Neurobehavioral Sequelae

When assessing any potential neurobehavioral impairments following an ABI, it is important to consider the potential impact of comorbid executive dysfunction, cognitive-communication impairment, and cognitive difficulties on how a patient may present. Individuals may not present with the typical features outlined in the DSM diagnostic criteria that is often used for psychiatric diagnosis and therefore a broader approach as to how psychiatric disorders may present is recommended (McAllister, 2013). Some specific considerations for the presentation of neuropsychiatric disorders in ABI populations are presented below (McAllister, 2013).

### Considerations for the presentation of neuropsychiatric disorders (McAllister, 2013):

- Challenges with speech or language may influence the presentation of sequelae.
- Impaired memory, time distortion, and poor self-monitoring may impact observable signs.
- Executive dysfunction may result in unpredictability.
- Routine dependence; individuals with an ABI may become reliant on a routine as part of their recovery and become frustrated when that routine is altered.
- The reliability of an individual's statements about oneself may be influenced by comorbid cognitive difficulties.

### 5.2.1 Depression

#### Q2. When do the symptoms of depression begin to become apparent following an ABI?

1. Depression in those who have sustained an ABI may be a reaction to the injury and/or the result of the pathophysiological changes that have taken place.
2. For some, depression will develop within months of the injury.
3. For others, depression may develop later, and it will be months or years before the clinical symptoms of depression are observed and individuals are treated.

**Depression is the most common mood disorder diagnosed in persons who sustain an ABI** (Jean-Bay, 2000; Jorge, 2005; Seel et al., 2010; Underhill et al., 2003). Studies have suggested the development of depression may be related to the location of injury, a pre-existing mental health condition, personality type, family support, social support, psychological stressors, and/or neurochemical imbalances (Bay & Covassin, 2012; Bombardier et al., 2016; Cnossen et al., 2016; Jorge, 2005; Ownsworth & Oei, 1998; Rosenthal et al., 1998; Sigurdardottir et al., 2013). Identifying and treating depression if present is important, as depression has been associated with poorer functional recovery and quality of life following ABI (Anke et al., 2015; Hudak et al., 2012). Common signs and symptoms of depression are outlined below.

### **Common signs and symptoms of depression following an Acquired Brain Injury:**

- Feeling low, sad, or hopeless consistently over at least two weeks.
- Disinterest in pleasurable activities, or activities that were previously enjoyed.
- Feelings of worthlessness, excessive guilt, and failure.
- Disturbances in sleep or appetite.
- Social withdrawal.
- Lethargy.
- Thoughts of death or suicide.
- Difficulty concentrating.

**Studies examining depression following ABI have noted that depression or depressive symptoms can begin within the first three months of injury** but the risk of developing depression post-ABI remains high for years after injury and may therefore present much later. Depression occurring within the first year has been noted in 27%-61% of individuals with ABI (McAllister, 2013). The risk for depression is high post-ABI and remains this way for decades following injury (Hoffman et al., 2010). A meta-analysis conducted by Osborn et al. (2014) reported that 21% to 43% of individuals have depression within the first five years of TBI; after five years, the prevalence then stabilizes to approximately 22%. These rates are high compared to the general population, where rates are around 7% (Thompson, 2019). A recent study of 827 individuals with a TBI found that 56% experienced depression within three months of injury and consistent with earlier literature, individuals with depression post-TBI had worse outcomes overall (Singh et al., 2018). One of the suspected factors influencing the high rates of depression after traumatic brain injury is the significant overlap between the areas of the brain moderating depressive symptoms and the regions of the brain most affected by a TBI, including the dorsolateral prefrontal cortex, anterior cingulate gyrus, and ventrolateral prefrontal cortex. (Brody et al., 2001; Drevets & Raichle, 1992; Liotti & Mayberg, 2001; McAllister, 2013).

Distinguishing between depression and behavioural sequelae resulting from an ABI can be challenging as there can be overlap between symptoms. For example, disturbances in appetite may be due to depression or due to impaired smell and taste resulting from the ABI. Diminished ability to perform everyday tasks may be driven by depression or from post-injury executive dysfunction. An increase in irritability and behavioural issues may be symptoms of either depression or brain injury (Fleminger et al., 2003). Moreover, the development of depression can exacerbate neurobehavioural sequelae of ABI (Fleminger et al., 2003).

### **5.2.2 Anxiety Disorders**

Anxiety is a subjective sensation of apprehension that may be accompanied by signs that are part of the fight or flight response such as restlessness, tension, tachycardia, or shortness of breath. Anxiety can be related to cognitive impairment resulting from injury or it may be related to the psychological trauma of the injury itself. Anxiety disorders are common following ABI and can be disabling. These disorders may be generalized or may be limited to a specific circumstance or phobia.

**Anxiety or anxiety disorders have been reported to occur in 4% to 28% of individuals who sustain an ABI** (Alway et al., 2012; Anke et al., 2015; Deb et al., 1999; Fann et al., 1995; Gould et al., 2014; Hart et al.,

2016; O'Donnell et al., 2008; Osborn et al., 2015; van Reekum et al., 1996). Several studies have reported a 2.3-fold increased risk of anxiety disorders in ABI populations compared to non-ABI populations (van Reekum et al., 2000), as well as higher rates of comorbid depression. The most common anxiety disorders affecting persons who have sustained an ABI are Post-Traumatic Stress Disorder (PTSD), Obsessive Compulsive Disorder (OCD), and Generalized Anxiety Disorder (GAD). These disorders and their clinical presentation are outlined in Table 5.1. OCD has been reported to affect approximately 6.4% of persons with an ABI (McAllister, 2013), while Post-traumatic Stress Disorder (PTSD) is experienced at slightly higher rates of 14%-20% of persons with an ABI (Whelan-Goodinson et al., 2009).

**Table 5.1 Description of the clinical features associated with post-ABI anxiety disorders (American Psychiatric Association, 2013).**

Anxiety Disorder	Description
Generalized anxiety disorder (GAD)	Excessive anxiety and/or worry that is difficult to control, feelings of restlessness, fatigue, irritability, muscle tension, and feeling on edge are also typical. This can be accompanied by sleep disturbances.
Obsessive compulsive disorder (OCD)	Presence of invasive and unwanted obsessions, compulsions, or both that cause significant distress. Obsessions are recurrent and persistent thoughts, urges, or images. Compulsions are described as repetitive behaviours or mental acts (such as praying) that an individual feels compelled to complete.
Post-traumatic stress disorder (PTSD)	After experiencing a traumatic event, the individual has recurrent, involuntary, and intrusive memories and/or dreams about the event. They may also experience significant psychological distress in the form of flashbacks or re-experiencing the traumatic event, or may have involuntary or hypervigilant responses to cues that symbolize the traumatic event. Avoidance of stimuli associated with the traumatic event and/or changes in cognition or mood associated with the time of the event are also possible symptoms of PTSD.

### 5.2.3 Agitation and Aggression

#### **Q3. Who might be the first person(s) to point out disruptive behavior like agitation or aggression following an ABI?**

1. Family members, caregivers, partners, and friends might be the first to notice a change in behavior related to agitation and/or aggression. Agitation and aggression are usually the least tolerated behaviors as they can cause significant emotional disruption and harm to the individual and all of those in the circle of care.

Agitation and aggression occur in approximately 33% to 54% of patients with TBI (Janzen et al., 2014; Sabaz et al., 2014). Agitation is generally defined as restlessness, impulsiveness, edginess, distractibility, wandering, and/or non-compliance, while aggression is defined as physical or verbal violence that may put the individual and others at risk for injury (Eisenberg et al., 2009). The occurrence of these behaviours have been associated with several factors, including younger age (Jean-Bay, 2000; Nott et al., 2006; Wolffbrandt et al., 2013), frontal lobe lesions (Tateno et al., 2003; Warriner & Velikonja, 2006), premorbid

major depression (Bakchine et al., 1989; Jean-Bay, 2000; Kim & Humaran, 2002; Nott et al., 2006; Sabaz et al., 2014; Tateno et al., 2003), and premorbid substance abuse (Sabaz et al., 2014; Tateno et al., 2003). During rehabilitation and the severity/duration of these challenging behaviours can have a negative impact on functional outcomes (Jean-Bay, 2000; Singh et al., 2014).

**Agitation is characterized as “heightened activity with non-purposeful behavior” and is often accompanied by disorientation, post-traumatic amnesia (PTA), disinhibition, aggression, and akathisia** (Nott et al., 2006). In the acute period (14 days) following a moderate to severe ABI, agitation is considered a normal phase of recovery and is most strongly associated with frontal lobe lesions (Cifu, 2010). Resolution of agitation typically coincides with emergence from PTA (J. Van Der Naalt, 2000; Nott et al., 2006), with 45% of individuals having agitation limited to the PTA period (Fugate et al., 1997). Approximately half of patients will experience persistent agitation beyond the emergence from PTA (Fugate et al., 1997). Agitation following an ABI can also stem from delirium, an acute confused state arising from the interplay of multiple factors including severe medical illness that is typically reversible or transient with appropriate treatment (Fleminger et al., 2003; Oddo et al., 2016a, 2016b). As such, screening for reversible medical contributors to agitated behaviour, such as infection, electrolyte disturbance, metabolic derangements, pain, and medication use is important. Although aggression is influenced by premorbid factors such as history of depression, substance abuse, and poor social functioning, there appears to be no such relationship for agitation (Tateno et al., 2003). Agitation is associated with comorbid psychiatric problems (Reyes et al., 1981), such as increased anxiety and depression (Levin & Grossman, 1978), increased length of stay, poorer cognitive functioning at discharge (Bogner et al., 2001), and delayed return to work (Denny-Brown, 1945).

Agitation and aggression represent challenging injury sequelae as both of these conditions can significantly impact an individual’s ability to participate in therapy. **As the intensity of both of these behaviours increase, so does the need for urgent comprehensive intervention** (Karol, 2013). Agitation and aggression are typically identified by restlessness, fits of anger, and/or stubbornness (Ciurli et al., 2011). These symptoms are often exacerbated by external cues or stimuli, which is why the majority of interventions for agitation and aggression include counselling and creating strategies with caregivers and therapists to reduce the number of triggers for these behaviours (Karol, 2013). Aggressive behaviour typically falls within two common scenarios: an individual may have difficulty moderating the intensity (emotional regulation) or duration of a behavior, and/or disinhibition may prevent the individual from stopping an inappropriate or dangerous behaviour in the first place (Karol, 2003). The former scenario of aggressive behaviour highlights an individual’s inability to moderate their behaviour, while the latter centres on the inability to inhibit inappropriate behaviour.

## 5.2.4 Addictive Behaviours

**Q4. What is the most commonly abused substance following an ABI?**

1. Alcohol

Research has shown that substance abuse occurs more frequently in those who have sustained an ABI than members of the general public (Taylor et al., 2003). Prevalence of substance abuse and addiction has been identified as approximately 4.6% in the general population (American Addiction Centers, 2019;

Spitzer et al., 2006). But the precise rates of pre- and post-injury substance abuse differ across the literature: **44-79% reported having an alcohol addiction at time of injury, while 12-38% reported having a drug addiction** (Andelic et al., 2010; Bombardier et al., 2002; Kolakowsky-Hayner et al., 2002; Kwok et al., 2013; Taylor et al., 2003; West et al., 2009). It has been reported that as many as 40% of persons with a TBI meet the criteria for substance abuse or dependence as defined by the DSM-5 (Hibbard et al., 1998).

**TBI patients who abused alcohol pre-injury were ten times more likely to demonstrate problematic alcohol use post injury** (Bombardier et al., 2003). The number of alcoholic drinks per week pre-injury reported in a sample of TBI patients was in the 84<sup>th</sup> percentile of average American alcohol consumption (Bombardier et al., 2002). Among those who sustain their ABI in a motor vehicle collision, one of the leading causes of brain injury, almost half were found to be intoxicated at the time of injury (DeLambo et al., 2008; Wehman et al., 2000; West et al., 2009). Studies suggest that alcohol and substance abuse decline within the first year of injury, with as many as 72% seeing a reduction in alcohol use with up to 36% abstaining (Bombardier et al., 2003; Jorge, 2005; Kelly et al., 1997; Ponsford et al., 2007), but of those who returned to drinking two years post injury are likely to consume more than before the injury (Bombardier et al., 2002; Ponsford et al., 2007). Individuals who have a history of substance abuse prior to ABI may initially reduce the extent or frequency of their substance use post-injury, evidence suggests these persons are likely to ultimately resume using (Bogner & Corrigan, 2013). Previous studies have found that approximately one third of individuals who sustain an ABI have a history of substance abuse (Bogner & Corrigan, 2013). However, this number may represent an underestimation of the true rate of substance abuse following ABI, as persons with a history of substance abuse are more likely to be lost to follow-up (Corrigan et al., 1997; Corrigan et al., 2003).

According to the DSM-5, substance abuse is categorized by the compliment of behaviours related to the use of that substance (American Psychiatric Association, 2013). Behaviors indicative of substance abuse are presented below in Table 5.2 along with categories of substances which can be abused. In this instance, individuals may be taking substances (prescription, illegal, or otherwise) for longer periods of time and higher dosages than recommended.



**Clinical Tip!**

If substance abuse behaviors are observed, a management strategy should be discussed as soon as possible with the individual and family.

**Table 5.2 Potential behavioral indicators of substance abuse, as well as commonly abused substances ((American Psychiatric Association, 2013); <https://www.mayoclinic.org/diseases-conditions/drug-addiction/symptoms-causes/syc-20365112>).**

Substances vulnerable to abuse	Potential behavioral indicators of substance abuse
<ul style="list-style-type: none"> <li>• Alcohol</li> <li>• Opiates</li> <li>• Cannabis</li> <li>• Prescription Medication</li> <li>• Hallucinogens</li> <li>• Stimulants/Sedatives</li> </ul>	<ul style="list-style-type: none"> <li>• Extended use and increased dosage compared to standard use</li> <li>• Multiple failed attempts to control or eliminate use of substance</li> <li>• Significant amount of time invested in obtaining, using, or recovering from the substance</li> <li>• Intense cravings or urges for the substance</li> <li>• Frequent use of substance impedes fulfillment of obligations</li> <li>• Risky use of substance (using in dangerous places, using despite known risks, using despite acknowledgement of substance abuse)</li> </ul>

	<ul style="list-style-type: none"> <li>• Continued substance use despite resulting social and interpersonal impairment</li> <li>• Seeking the substance from multiple sources</li> <li>• Experiencing withdrawal symptoms when the substance is not taken</li> <li>• Purchasing excessive amounts of the substance without reasonable means</li> </ul>
--	--

Persons who sustain an ABI are also at risk for other addictive behaviours, namely problem gambling. In a population-based matched case-control study, persons with a prior history of TBI had higher subsequent risk of problem gambling, with 5.4% reporting problem gambling behaviours compared to 3.5% of matched controls without a history of brain injury (Bhatti et al., 2019). This was most pronounced for middle aged men (35-64 years old), those with a history of smoking or alcohol use, and in persons with a history of two or more TBIs.

### 5.2.5 Suicidal Ideation

Suicidal ideations are the thoughts or considerations of suicide that when left unattended can lead to distress and attempted suicide. **23-28% of individuals report suicidal ideation after sustaining a TBI** (Mackelprang et al., 2014; Simpson & Tate, 2002; Tsaousides et al., 2011). Compared to the general population, persons who have sustained a TBI are twice as likely to commit suicide, even after controlling for substance abuse and comorbid psychiatric conditions (Ahmedani et al., 2017). Risk factors for suicide post-ABI include being male (Wisco et al., 2014), comorbid diagnosis of depression, anxiety, or PTSD (Tsaousides et al., 2011), greater number of sustained TBIs (Wisco et al., 2014), perceived burdensomeness (Bryson et al., 2017), and substance abuse history (Simpson & Tate, 2005). Age at time of injury is not associated with suicidal ideation (Mackelprang et al., 2014; Simpson & Tate, 2002). Persons with ABI are also more likely to have pre-morbid characteristics that increase their risk of suicide compared to the general population, such as being male and having a history of substance abuse (Ahmedani et al., 2017; Bahraini et al., 2013; Simpson & Tate, 2007).

Suicidal ideation does not appear to dissipate over time following an ABI. Elevated suicidal ideation at one year post TBI is associated with continual elevation of ideation at five years post-injury (Fisher et al., 2016). The risk for suicidal ideation and attempt remains high at 20 years after TBI (Fisher et al., 2016), emphasizing the importance of screening for and addressing suicidality.

If suicidal ideation is not addressed, the risk of suicide attempts is high (Simpson & Tate, 2007) and is further increased when emotional distress is present (Gutierrez et al., 2008; Simpson & Tate, 2002). Emotional disturbance and substance abuse history increase the risk for attempted suicide by a factor of 21, compared to individuals with no history (Simpson & Tate, 2005). Within their lifetime, 26% of individuals post-TBI attempt suicide, and half of these individuals make more than one attempt (Simpson & Tate, 2002, 2005).

Thoughts of suicide or suicidal ideation must be taken seriously and addressed. Below is a comprehensive list of indicators of suicidal ideation. Although many of these symptoms can be caused by the ABI itself, it is important to remember to assess the individual as a whole, as the number of indicators increases so does the risk of potential suicide. If suicidality is an active concern or potentially threatening a person's safety, the person should be sent to the emergency department for evaluation.

### Indicators of potential suicidal ideation and risk (Nordqvist, 2018):

- Feeling or appearing to feel trapped or hopeless.
- Feeling intolerable emotional pain.
- Having or appearing to have an abnormal preoccupation with violence, dying, or death.
- Having mood swings, either happy or sad.
- Talking about revenge, or guilt, or shame.
- Being agitated, or in a heightened state of anxiety .
- Experiencing changes in personality, routine, or sleeping patterns.
- Consuming more drugs or alcohol than usual, or starting drinking when they had not previously.
- Engaging in risky behavior, such as driving carelessly or taking drugs.
- Getting their affairs in order and giving things away.
- Getting a hold of a gun, medications or substances that could end a life.
- Experiencing depression, panic attacks, or impaired concentration.
- Increased isolation.
- Talking about being a burden to others.
- Psychomotor agitation, such as pacing around a room, wringing one's hands, and removing items of clothing and putting them back on.
- Saying goodbye to others as if it were the last time.
- Seeming to be unable to experience pleasurable emotions from normally pleasurable life events such as eating, exercise, social interaction, or sex.
- Severe remorse and self criticism.
- Talking about suicide or dying, expressing regret about being alive or ever having been born.

## 5.3 Outcome Measures and Assessments

### 5.3.1 Agitated Behavior Scale

The Agitated Behavior Scale (ABS) was developed to specifically assess agitation during the acute phase of acquired brain injury (Bogner, 2000), and can be accessed [HERE](#). This tool is designed to be executed repeatedly over time to assess agitation over the course of recovery, and is available for free. The ABS has been validated in TBI populations and the psychometrics of the ABS support its use with an interrater reliability of  $r=0.92$ , and Chronbach's  $\alpha=0.83-0.92$  (Corrigan, 1989).

The ABS should be administered by a nurse or a clinician and should take place over at least a 30-minute observation period. The ABS consists of 14 questions regarding observable behaviors and asks you to rank the intensity to which that behavior is present (Figure 5.3). Scores range from 14-56, with specific cut-offs for brain injury as follows: within normal limits =  $\leq 21$ , mild agitation = 22-28, moderate agitation = 29-35, severe agitation =  $\geq 35$ .

- 1 = absent: the behavior is not present.
- 2 = present to a slight degree: the behavior is present but does not prevent the conduct of other, contextually appropriate behavior. (The individual may redirect spontaneously, or the continuation of the agitated behavior does not disrupt appropriate behavior. )
- 3 = present to a moderate degree: the individual needs to be redirected from an agitated to an appropriate behavior, but benefits from such cueing.
- 4 = present to an extreme degree: the individual is not able to engage in appropriate behavior due to the interference of the agitated behavior, even when external cueing or redirection is provided.

**Figure 5.3 The rating scale for observable behaviors for the ABS.**

### **5.3.2 Overt Behavior Scale**

The Overt Behavior Scale (OBS) has nine sections which are designed to identify the frequency and intensity of agitation, aggression, and anti-social behaviors over the last three months (Kelly et al., 2006). The assessment involves summing how many items the individual presents with, the severity of each of the behaviours, and the impact of these behaviours. Interrater reliability of the OBS is considered high ( $r=0.97$ ). Figure 5.4 shows a section of the OBS pertaining to physical aggression and the associated scoring. Recently, Kelly et al. (2019) published a validated self-report version of the OBS, which is also available for use for free [HERE](#). Although the scoring of the OBS is guided (based on frequency of behaviors), the interpretation of those scores is often subjective. This is due to the interplay based on the frequency of behaviors and the severity of the behaviors which are present (impact). For example, an individual may frequently slam doors, but the negative impact of that behavior is relatively low compared to a single instance of physical aggression towards another or themselves which required immediate intervention. For this reason, both the frequency and impact of each behavior should be taken into consideration when evaluating the severity of challenging behaviors.

# PHYSICAL AGGRESSION

Severity	Levels Tick each level that is a problem (✓)	Frequency	Impact
		1 = < 1/month	1 = no impact
		2 = 1/month or more	2 = minor
		3 = 1/week or more	3 = moderate
		4 = 1/day	4 = severe
		5 = multiple daily	5 = extreme

## Physical aggression against objects

Has the client shown any physical aggression against objects?

NO (go to next behaviour)

YES (rate the subscale below)

Slams doors, scatters clothing, makes a mess in clear response to some antecedent.

1




Throws objects down (without some other person at risk of being hit by the object), kicks furniture without breaking it, marks the wall.

2




Breaks objects, smashes windows.

3




Sets fire, throws objects dangerously (i.e., some other person is at risk of being hit by the object(s) thrown but is not actually hit) *If the object thrown does hit someone score this as Physical aggression against other people.*

4




Figure 5.4 The physical aggression portion of the Overt Behaviors Scale and examples of scoring categories.

### 5.3.3 Patient Health Questionnaire

The Patient Health Questionnaire (PHQ-9) is a 9-item self-administered questionnaire which focuses on depressive symptoms and can be overseen by any healthcare provider (Kroenke et al., 2001). This assessment tool is free to use (accessed [HERE](#)) and asks individuals to identify during the last two weeks the extent to which they've experienced depressive symptoms (Figure 5.5). The "Guide for Interpreting PHQ-9 Scores" (Figure 5.6) provides a breakdown of scores to facilitate interpretation (Kroenke et al., 2001). The majority of actions recommended for score ranges on the PHQ-9 involve clinical judgement to determine the appropriateness of treatment except above a score of 15, at which point treatment is recommended.

Over the last 2 weeks, how often have you been bothered by any of the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed? Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

**Figure 5.5 The 9 questions included in the PHQ-9 assessing depressive symptoms.**

Guide for Interpreting PHQ-9 Scores		
Score	Depression Severity	Action
0 - 4	None-minimal	Patient may not need depression treatment.
5 - 9	Mild	Use clinical judgment about treatment, based on patient's duration of symptoms and functional impairment.
10 - 14	Moderate	Use clinical judgment about treatment, based on patient's duration of symptoms and functional impairment.
15 - 19	Moderately severe	Treat using antidepressants, psychotherapy or a combination of treatment.
20 - 27	Severe	Treat using antidepressants with or without psychotherapy.

**Figure 5.6 Reference for the interpretation of scores for the PHQ-9.**

### 5.3.4 Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale (HADS) is another free assessment tool which can be used to determine the severity of depression and anxiety symptoms specifically in ABI populations (Zigmond & Snaith, 1983). This test is typically self-administered. Both sections of the assessment (anxiety and depression) use the same scoring range to determine the severity of the symptoms: 0-7 “Normal”, 8-10 “Borderline Abnormal”, 11-21 “Abnormal”. Questions are framed both positively and negatively such, “I still enjoy the things I used to enjoy”, and “I get sudden feelings of panic”. A pdf of the HADS can be accessed [HERE](#).

### 5.3.5 Generalized Anxiety Disorder 7-Item Scale

The Generalized Anxiety Disorder 7-item Scale (GAD-7) (Spitzer et al., 2006) is an open source 7-item questionnaire which can be self-administered, or clinician administered to assess the frequency of which an individual feels symptoms of anxiety. The assessment is graded on a scale of 0-3, with the sum score ranging between 0 and 21. Scores ranging from 0-5 are considered indicative of mild anxiety, 6-10 is considered moderate, and scores from 11-21 are indicative of severe anxiety (Spitzer et al., 2006). The GAD-7 has been shown to have a sensitivity of 89% and specific of 82% when using a score of 10 as the threshold (Spitzer et al., 2006). Figure 5.7 shows the GAD-7 which can be accessed [HERE](#).

Over the last 2 weeks, how often have you been bothered by the following problems?	Not at all sure	Several days	Over half the days	Nearly every day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it's hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3
<i>Add the score for each column</i>	+	+	+	
<b>Total Score (add your column scores) =</b>				

Figure 5.7 The GAD-7

### 5.4 Criteria for Diagnosis

**Q5. What resources are available to help you determine a diagnosis for mood and behavioral disorders following an ABI?**

1. Diagnostic and Statistical Manual of Mental Disorders-5.
2. Clinical assessment tools (ABS, PHQ-9, OBS, HADS, Beck Depression Inventory, Depression Anxiety & Stress Scale, GAD7, Structured Clinical Interview (from DSM)).
3. Colleagues with significant clinical experience.

Mental health and neurobehavioural challenges can be difficult to diagnose following an ABI for a variety of reasons. There may be comorbid factors which impact the presentation of disorders and individuals may have pre-existing conditions which can also influence mood and behavior. Like methods used for the diagnosis of cognitive disorders, multiple factors need to be considered in order to result in the most accurate picture of an individual’s emotional and behavioral functioning.

With the exception of agitation and aggression, the common diagnostic criteria used for depression, anxiety disorders, addictive behaviors, and suicidal ideation in individuals with an ABI are those listed in the DSM-5. Table 5.3 provides a list of the relevant criteria and the page number on which they can be found. Although the DSM-5 provides the most definitive criteria for mental health and neuropsychological disorders, it is important to note that this diagnostic criterion may not always be appropriate for an individual with an ABI as comorbid deficits may conflict with the expression, interpretation, and function of behavior or thoughts in individuals.

**Table 5.3 Page number of diagnostic criteria in the DSM-5**

Topic	Page Number
Depression	p. 160
Generalized Anxiety Disorder (GAD)	p. 222
Obsessive Compulsive Disorder (OCD)	p. 237
Post-traumatic Stress Disorder (PTSD)	p. 271
Substance Abuse (Alcohol, cannabis, hallucinogens, opioids)	p. 483 (490, 509, 520, 541)
Suicidal Behavior Disorder (SBD) and Nonsuicidal Self-Injury	p. 801-803

\*SBD and suicidal ideation are not considered synonymous, for indicators of suicidal ideation see section 5.2.5 above.

With respect to diagnosing agitation and aggression following an ABI, the thresholds provided by the ABS in section 5.3.1 are useful in confirming the presence of significant agitation or aggression: within normal limits =  $\leq 21$ , mild agitation = 22-28, moderate agitation = 29-35, severe agitation =  $\geq 35$ .

## 5.5 Interventions for Mental Health Disorders Following an ABI

Interventions for mental health or neurobehavioral disorders should be considered when an individual is not progressing as expected (Ontario Neurotrauma Foundation, 2015). Prior to any interventions, a neurobehavioral assessment should be completed by a licensed practitioner and should include comprehensive screening for comorbid illnesses, such as cognitive dysfunction, metabolic disorders, medication side effects, and communication impairment (Ontario Neurotrauma Foundation, 2015). The Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI indicates any intervention following an ABI should be designed to address pre-injury factors (such as lifestyle or addiction), injury related factors (such as severity), and post-injury factors (such as coping, pain, or sleep disturbances) to encourage the best possible outcome (Ontario Neurotrauma Foundation, 2015).

Educating the patient and family on existing neurobehavioral disorders and mental health concerns should also be a priority (Ontario Neurotrauma Foundation, 2015). Family and a person’s care team can be an excellent resource to help identify changes in mood, thoughts, or behaviour following an ABI. As a critical part of the support system, they should be provided with the appropriate information and tools to best support an individual with an ABI.

In 2015, the International Committee on Mental Health in Cystic Fibrosis created an algorithm (Figure 5.6) for the treatment of depression and anxiety for those with cystic fibrosis (Quittner et al., 2015). Although not developed for ABI, the clinical approach in this algorithm has broad relevance and utility, and has been included for that reason.

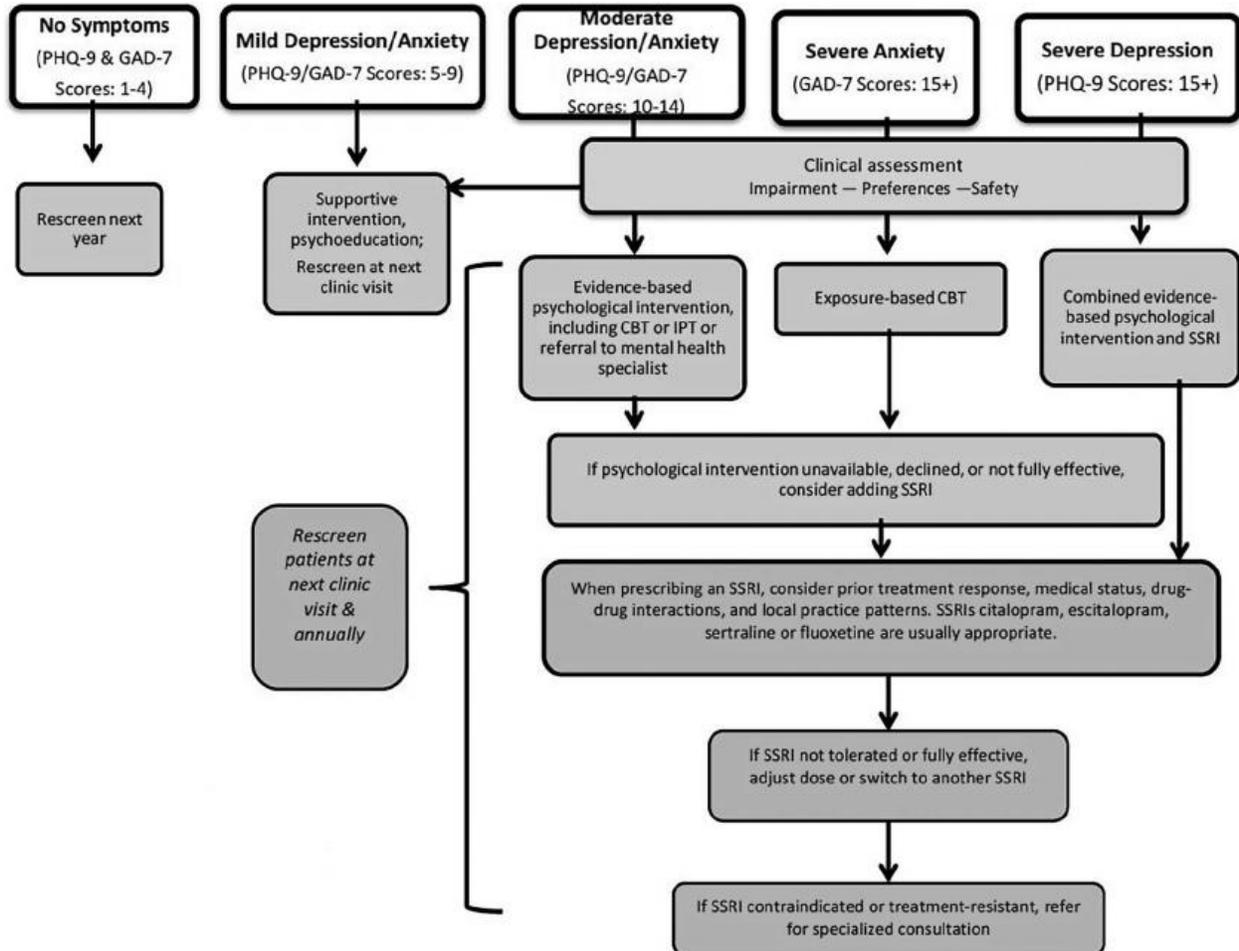


Figure 5.6 A potential management strategy for the management of depression and anxiety following an ABI, taken from the International Committee on Mental Health in Cystic Fibrosis (Quittner et al., 2015).

[Click here to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI on Neurobehavior and Mental Health](#)

### 5.5.1 Interventions for Depression

Interventions for depression fall into two broad categories: non-pharmacological and pharmacological. The interventions with the strongest evidence that have been identified as clinically relevant by our reviewers are discussed below. For treatment of mental health concerns, as with other categories of injury sequelae that have effective non-pharmacological and pharmacological interventions, appropriate

interventions should be discussed with the individual in question and their views on treatment should be taken into consideration whenever possible.

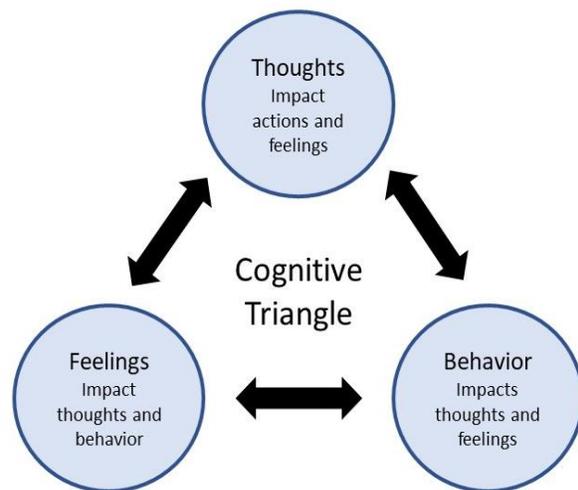
### 5.5.1.1. Non-Pharmacological Interventions

#### Cognitive-Behavioral Therapy

**Q6. What is Cognitive-Behavioral Therapy? Name a strategy that falls into this category.**

1. Cognitive-Behavioral Therapy (CBT) is a form of psychotherapy where individuals learn strategies to identify, question, change, and accept thoughts, beliefs or emotions which cause them significant distress in their daily life.
2. Radical acceptance. Radical acceptance is a cognitive strategy whereby an individual attempts total and complete acceptance of a situation and is no longer fighting a reality they cannot change.

Strong evidence in the ABI literature supports the use of CBT, with multiple RCTs demonstrating its efficacy (Section 8.2.4.1 ERABI). CBT also includes strategies to improve coping, relaxation, organization, and graded exposure to stimuli. **CBT for the treatment of depression following an ABI is recommended by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI.** An excellent resource for CBT is *A Therapist's Guide to Brief Cognitive Behavioral Therapy* by Cully and Teten (2008), which can be accessed [HERE](#). Figure 5.7 shows the cognitive triangle which is often referenced in CBT to help individuals better understand the relationships between thoughts, emotions, and actions. One of the goals of CBT is often to break the negative cycle of thoughts, behaviours and emotions which can prevent individuals from full recovery.



**Figure 5.7 The cognitive triangle which is often used to help individuals understand the connection between thoughts, feelings, and behaviors.**

[Click HERE to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI with respect to interventions for the management of depression](#)

## Key Study

Author/ Year/ Country/ Study Design/ N	Methods	Outcomes
<p><a href="#">Ponsford et al.</a> (2016) Australia RCT PEDro=7 N<sub>Start</sub>=75, N<sub>End</sub>=51</p>	<p><b>Population:</b> TBI. <i>CBT+MI Group (n=26):</i> Mean Age=46.69 yr; Gender: Male=18, Female=8; Mean Time Post Injury=4.88yr; Mean GCS=10.43. <i>CBT+NDC Group (n=26):</i> Mean Age=39.88 yr; Gender: Male=20, Female=6; Mean Time Post Injury=3.58 yr; Mean GCS=10.48. <i>WC Group (n=23):</i> Mean Age=39.87 yr; Gender: Male=17, Female=6; Mean Time Post Injury=2.61 yr; Mean GCS=8.23.</p> <p><b>Intervention:</b> Participants diagnosed with depression and/or anxiety were allocated to receive cognitive behavioural therapy (CBT) with either motivational interviewing (CBT+MI) or non-directive counseling (CBT+NDC), or to a waitlist control (WC). MI and NDC were each delivered for 3 wk, followed by 9 wk of CBT, with three CBT booster sessions 21-30 wk from baseline. Assessments were conducted at baseline, 3 wk, 12 wk, 21 wk, and 30 wk.</p> <p><b>Outcome Measure:</b> Depression, Anxiety &amp; Stress Scale (DASS), Hospital &amp; Anxiety Depression Scale (HADS), Sydney Psychosocial Reintegration Scale 2 (SPRS-2).</p>	<ol style="list-style-type: none"> <li>1. All groups demonstrated significant improvements on the DASS-Depression, HADS-Anxiety, and SPRS-2 over time.</li> <li>2. On the DASS-Depression, there was a significantly greater reduction in score over time in CBT+MI versus WC (<math>p&lt;0.005</math>) but not CBT+NDC versus WC; there was no significant difference between CBT+MI and CBT+NDC.</li> <li>3. On the HADS-Anxiety, there was a significantly greater reduction in score over time in CBT+NDC versus WC (<math>p&lt;0.05</math>) but not CBT+MI versus WC; there was no significant difference between CBT+MI and CBT+NDC.</li> <li>4. On the SPRS-2, there was no significant difference in improvement between groups over time.</li> <li>5. Higher baseline DASS-Depression and HADS-Anxiety scores were significantly associated with greater response to treatment (<math>r=0.34</math>, <math>p&lt;0.05</math> and <math>r=0.37</math>, <math>p&lt;0.05</math>, respectively).</li> <li>6. When combining CBT+MI and CBT+NDC groups, there were significantly greater improvements on HADS-Anxiety (<math>p&lt;0.05</math>), DASS-Depression (<math>p&lt;0.005</math>), and SPRS-2 (<math>p&lt;0.05</math>) in the combined group versus WC.</li> </ol>

CBT is frequently used to treat depression as a variety of strategies are available to help individuals whose sources of distress result from different causes. In a large randomized control trial (RCT) by Ponsford et al. (2016), the authors found that CBT combined with motivational interviewing resulted in a significantly greater reduction in depressive symptoms compared to those in the wait-list control group. In this study, higher baseline depression scores were significantly correlated with greater benefits as a result of treatment. A brief list of common CBT strategies is presented in Table 5.4.

**Table 5.4 Examples of Cognitive Behavioral Strategies adapted from (Rector, 2010).**

Strategy	Description
Journaling	Recording information about your moods, thoughts, sources of distress, intensity of feelings, and responses to them.
Identifying cognitive distortions	Identifying beliefs or views which are causing significant distress and challenging them.
Cognitive restructuring	Finding ways to restructure harmful beliefs, values and thoughts so that they do not negatively impact your life.
Progressive muscle relaxation	Involves focusing on relaxing sequential muscle groups one at a time.

Interoceptive exposure	Allowing yourself to experience negative feelings and emotions in order to decrease your fear about experiencing them.
Questioning negative thoughts	This strategy involves identifying and questioning automatic negative thoughts to evaluate if they are truly based in reality.
Self-monitoring	Being aware of when and how you experience distress in your daily life in order to better evaluate the causes of your distress and your reaction to them.

### Mindfulness-Based Stress Reduction

#### Q7. What components are typically included in mindfulness-based stress reduction (MBSR)

1. MBSR typically incorporates mindful meditation, body awareness, and yoga with the goal of promoting relaxation and stress management.

Mindfulness-Based Stress Reduction usually takes place in weekly group setting (Crane, 2002), where participants are asked to focus in a non-judgemental and accepting way on their negative emotions, pain, thoughts, and sensations (Fjorback et al., 2011). The theory behind MBSR states that improved self-observation will lead to improved coping skills (Baer, 2003). MBSR has been shown to significantly reduce psychological distress and improve physical health (Williams et al., 2001). **Similar to CBT, MBSR is also recommended by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI for the treatment of depression following an ABI and has been shown to be effective in persons who sustain an ABI** (Bedard et al., 2003; Bedard et al., 2012; Bedard et al., 2014). A meta-analysis of 72 studies concluded that MBSR is effective for individuals experiencing depression for the first time, as well as for those who have relapsed from recovering from depression (Fjorback et al., 2011). However, multiple sources indicate that MBSR should not be considered an exclusive treatment for depression but part of a complement of therapies designed to reduce depression symptoms overall (Fjorback et al., 2011; Kabat-Zinn, 2000). Although specific strategies can be used in MBSR, general mindfulness and yoga practices are also used to ease depression and anxiety and can be helpful.

#### Key Study

Author/ Year/ Country/ Study Design/ N	Methods	Outcomes
<a href="#">Bedard et al.</a> (2014) Canada RCT PEDro=6 N=76	<p><b>Population:</b> TBI; Gender: Male=42, Female=34; Mean Age=46.5 yr; Mean Time Post Injury=4.25 yr.</p> <p><b>Intervention:</b> Participants were diagnosed with depression. The treatment group (n=38) received 1.5 hr weekly sessions of mindfulness-based cognitive therapy for 10 wk. The control group (n=38) received usual care. Assessments were conducted at baseline, 10 wk, and 3 mo follow-up.</p> <p><b>Outcome Measure:</b> Beck Depression Inventory II (BDI-II), Patient Health</p>	<ol style="list-style-type: none"> <li>1. Following treatment, the treatment group showed significantly greater reduction in BDI-II scores than the control group (<math>p=0.029</math>), which was maintained at the 3 mo follow-up.</li> <li>2. No significant between-group differences on PHQ-9 and SCL-90-R were found (<math>p&gt;0.05</math>).</li> <li>3. Neither PHLMS nor TMS reached significance in demonstrating increases in mindfulness for the treatment group (<math>p&gt;0.05</math>).</li> </ol>

Author/ Year/ Country/ Study Design/ N	Methods	Outcomes
	Questionnaire 9 (PHQ-9), Symptom Checklist 90 Revised (SCL-90-R). Philadelphia Mindfulness Scale (PHLMS), Toronto Mindfulness Scale (TMS).	

### 5.5.1.2 Pharmacological Interventions

Multiple pharmacological agents have been found to effectively treat depression and major depressive disorder in individuals with an ABI. **The Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI recommend the use of selective serotonin reuptake inhibitors (SSRIs) as a first-line pharmacological treatment for the management of depression** (Ontario Neurotrauma Foundation, 2015). The pharmacological interventions which have been shown to be efficacious in the treatment of depression following an ABI are presented below in Table 5.5.

**Table 5.5 Pharmacological treatments which have been shown to be effective in the treatment of depression specifically in those with an ABI.**

Drug	Description	Reference
Methylphenidate	Methylphenidate is a central nervous stimulant which has moderate evidence in support of its use for ABI populations. Methylphenidate may be used after SSRIs have been shown to be ineffective.	(INESSS-ONF, 2015; Lee et al., 2005; Spitzer et al., 2006)
Sertraline	Sertraline is an SSRI which has been shown to be effective at doses starting at 25mg and increasing to a daily dose of 50-200mg for individuals with an ABI.	(Lamontagne et al., 2017)
Citalopram	Citalopram is also an SSRI, with moderate evidence for its use in ABI populations. Recommended dosages start at 10mg, and increase to a daily dose of 20-40mg.	(INESSS-ONF, 2015; Perino et al., 2001; Rapoport et al., 2008).

The mechanism of action for SSRI's is shown in Figure 5.8. SSRIs block the reabsorption of serotonin by the pre-synaptic nerve which releases it. **By blocking reabsorption, increased amounts of serotonin in the synaptic space can act on receptor sites on the post-synaptic nerve.** SSRIs are described as safe, usually with only mild side-effects such as drowsiness, insomnia, diarrhea, nausea, and reduced libido. Although SSRIs are usually well tolerated, potential drug interactions exist and should be screened for when initiating or adjusting medications

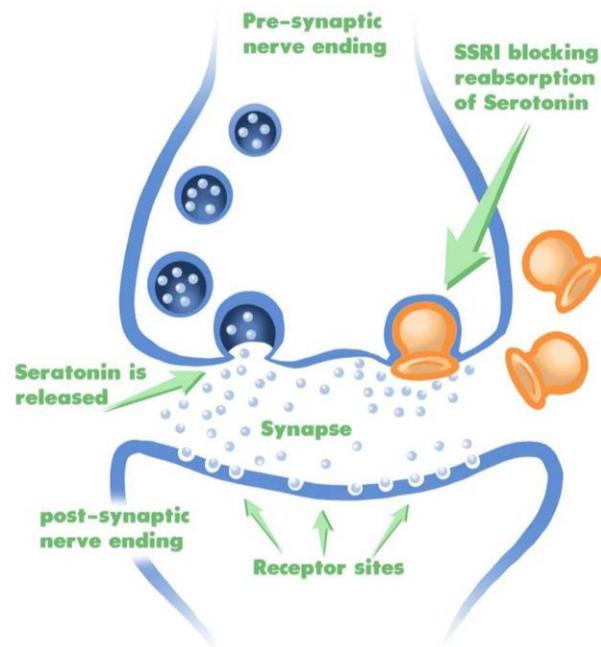


Figure 5.8 An illustration of the mechanism of action for SSRIs

## 5.5.2 Anxiety Disorders

### 5.5.2.1 Non-Pharmacological Interventions

**Q8. What is the relationship between the non-pharmacological interventions recommended for depression and those recommended for anxiety?**

1. CBT is the primary non-pharmacological intervention recommended for the treatment of both depression and anxiety following an ABI.

CBT is recommended for the treatment of anxiety and depression following an ABI. **CBT is the only non-pharmacological intervention for anxiety supported by both the ABI literature and the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI (Ontario Neurotrauma Foundation, 2015).** Please see section 5.5.1.1. for discussion on CBT.

### 5.5.2.2 Pharmacological Interventions

**Q9. What pharmacological interventions are recommended for the treatment of anxiety following an ABI?**

1. SSRIs are the only pharmacological intervention recommended for the treatment of anxiety following an ABI.

There are many areas of ABI rehabilitation where there is not enough literature to exclusively support a specific intervention; pharmacological interventions for anxiety is one of these areas. Although there are no studies meeting the ERABI inclusion criteria that evaluate pharmacological interventions for anxiety, **the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI, through clinical consensus, support the use of SSRIs for the management of anxiety following an ABI.** Given the tolerability of SSRIs and rates of comorbid depression among those with an ABI and anxiety, SSRIs should be used as a first line of treatment for the pharmacological management of anxiety. **Benzodiazepines are not recommended for the treatment of anxiety as potential side effects can impact arousal, cognition, and motor coordination** (Ontario Neurotrauma Foundation, 2015). For more information on SSRIs please see section 5.5.1.2 (Pharmacological interventions for depression) of this chapter.

[Click here to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI recommendations for anxiety disorders](#)

### 5.5.3 Interventions for Agitation and Aggression

In the following sections it should be noted that the non-pharmacological interventions highlighted are not appropriate for individuals still in PTA, however the pharmacological interventions are.

#### 5.5.3.1 Non-pharmacological Interventions

##### Anger Management Programs

**Q10. What types of therapies or strategies can be included in anger management programs?**

1. Anger management therapy.
2. Self-management training.
3. Cognitive behavioral therapy.
4. Individual or group-based therapies.
5. Social skills training.

A variety of interventions attempting to improve aggression and agitation have been studied in the ABI literature. CBT, self-management therapy, and anger management therapy have been shown to be effective components of anger management programs following an ABI (Abouafia-Brakha et al., 2013;

Burke et al., 1988; Hart et al., 2012; Medd & Tate, 2000; O'Leary, 2000; Walker et al., 2010). Counselling is thought to be an effective intervention for a variety of reasons: it can help give insight into triggers of negative behaviors, help explore thoughts and motivations for behavior, and also restore control to the individual in managing their care, which can be essential (Karol, 2013). Counselling should be considered as part of the treatment strategy wherever individuals are behaviorally and cognitively able to participate.

### Key Study

Author/ Year/ Country/ Study Design/ N	Methods	Outcome
<a href="#">Walker et al.</a> (2010) Australia Pre-Post N=52	<p><b>Population:</b> TBI; Mean Age=32.3 yr; Gender: Male=40, Female=12; Mean Time Post Injury=4.1 yr; Injury Severity: Severe.</p> <p><b>Intervention:</b> Participants received 2 hr sessions of group-based CBT focused on anger management, 1 x/wk for 12 wk. Assessments were conducted at baseline, 12 wk, and 3-16 mo follow-up.</p> <p><b>Outcome Measure:</b> State-Trait Anger Expression Inventory (STAXI).</p>	<ol style="list-style-type: none"> <li>At post treatment, there were significant reductions in trait anger (<math>p=0.002</math>), anger expression-out (<math>p=0.003</math>), and anger control (<math>p=0.005</math>), but not in state anger or anger expression-in.</li> <li>At follow-up (<math>n=31</math>), the improvements from baseline were maintained, but there were no further improvements from post treatment.</li> </ol>

Cognitive behavioral therapies focusing on anger management have been shown to be the most effective at reducing aggression following an ABI (Aboulafia-Brakha et al., 2013; Medd & Tate, 2000; Walker et al., 2010). This is in line with expert consensus as it is recommended that aggression and agitation therapies be as individualized as possible in order to target the root cause of the behavior (Karol, 2013). CBT is a flexible intervention that provides a variety of strategies which can be tailored to meet the needs of an individual, allowing for a personalized care. Details on specific CBT strategies can be found in section 5.5.1.1 (Non-pharmacological interventions for depression) of this chapter. Unfortunately, no specific anger management training interventions are recommended by guideline groups to our knowledge.

### Social Skills Training

Moderate evidence supports social skills training to improve aggression following an ABI. Social skills training aims to reduce the frequency and intensity of socially unacceptable behaviors and help individuals begin to socially interact in a positive manner (Karol, 2013). Social skills training is a type of behavior therapy that can be delivered in a group or individualized format and is often one component of a larger treatment strategy. Social skills training can span a large spectrum of behavioral skills, from making eye contact, to language sensitivity and communication (Karol, 2013).

An RCT by McDonald et al. (2008), found that social skills training significantly improved partner directed behaviour compared to social non-therapy groups, and waitlist controls. In addition, some of these effects were maintained at one-year follow-up post-treatment.

### 5.5.3.2 Pharmacological Interventions

Recommended pharmacological interventions for aggression and agitation following an ABI are dependent upon the severity of the observed behavior and the danger that it poses to the individual and others. The diversity of behaviours typical of post-ABI agitation makes research for treatment efficacy

difficult to conduct, resulting in limited literature. Agitation may impair recovery by creating a disruptive and/or unsafe environment for rehabilitation or community living (Rosati, 2002). This section outlines specific interventions and the level of agitation severity for which they are appropriate. The use of these medications for the management of agitation and aggression post-ABI is off-label according to Health Canada.

**A treatment algorithm for the Pharmacological Management of Agitation and Aggression Following TBI by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI can be found [HERE](#).**

**Q11. What pharmacological interventions are recommended for the management of severe aggression and agitation following an ABI?**

1. Intramuscular benzodiazepines for severe life-threatening agitation or aggression (Ontario Neurotrauma Foundation, 2015).
2. Oral neuroleptic medications such as quetiapine, ziprasidone, olanzapine, and risperidone for severe aggression which threatens patient or staff safety.

Severe aggression and agitation can be life threatening (Karol, 2013). Typically, as aggression severity increases the comprehensiveness of the treatment approach should also increase, which may involve combining pharmacological treatments and optimizing non-pharmacological interventions (Karol, 2013). In the setting of severe, life-threatening aggression, or behaviour that threatens patient or staff safety, **an ideal medication should have “a rapid onset of action, achieve maximal effect with a single dose, cause minimal adverse effects, and allow the patient to resume normal daily activities as quickly as possible without causing protracted sedation or cognitive impairments”** (Stanislav & Childs, 2000). Medications used on an as-needed basis, such as intramuscular benzodiazepines and oral neuroleptic medications, should be short-acting for this reason. For persistent agitation, or for persons frequently requiring pharmacologic treatment for agitation or aggression, the use of scheduled daily medications outlined below should be considered (Ontario Neurotrauma Foundation, 2015).

For non-life-threatening aggression, the goals of therapy are to reduce the frequency and intensity of agitated behaviours while minimizing cognitive effects and sedation. Medications with the best evidence are those that are taken daily: beta-blockers (pindolol and propranolol) and anti-epileptic medications (valproate and carbamazepine) for persons with comorbid seizures (Ontario Neurotrauma Foundation, 2015). These medications reduce the intensity and frequency of agitation, and may be used in conjunction with as-needed medications for severe agitation or aggression.

**Q12. What medications can be used to treat non life-threatening aggression?**

1. Propranolol can be used with a recommended maximum daily dose of 420-520mg/day (Ontario Neurotrauma Foundation, 2015).
2. Pindolol can also be used with a recommended maximum daily dose of 40-100mg/day (Ontario Neurotrauma Foundation, 2015).
3. For those with comorbid seizures, valproate (750-2250mg/day) or carbamazepine (200-1200mg/day) can be used (Ontario Neurotrauma Foundation, 2015).

**Q13. What medications are recommended by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI for the management of agitation in persons with impaired arousal or attention?**

1. Methylphenidate.
2. Amantadine.

[Click here to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI recommendations for pharmacological interventions for agitation and aggression](#)

Although pharmacological agents may be necessary for the management of agitation or aggression, and can be effective, the benefits of counselling and non-pharmacologic strategies in appropriate individuals should not be underestimated. Counselling is an excellent adjunct to pharmacological therapy as it can help provide insights into triggers and thought patterns, as well as improve individual autonomy.

### 5.5.3.3 Use of Restraints

**Q14. What is the definition of a restraint? What types of restraints exist?**

1. A restraint can be either chemical, physical, or both and is designed to limit negative behavior either through sedation or through immobilization.
2. Chemical restraints can include (but are not limited to) beta blockers, antidepressants, anticonvulsants, psychostimulants, propranolol, neuroleptics, valproic acid, and anti-Parkinson's agents (Busch & Shore, 2000; Gregory Jr & Bonfiglio, 1995; McNett et al., 2012).
3. Physical restraints can take the form of bed rails, feeding trays, hand tying, chest straps, seat belts, ankle/wrist restraints, and jacket restraints (Busch & Shore, 2000; Gregory Jr & Bonfiglio, 1995; Marks, 1992; Morrison et al., 1987).

**There is insufficient evidence to support the use of restraints in ABI/TBI populations** (Duxbury & Wright, 2011). Studies have identified a variety of reasons as to why restraints are used in some scenarios but not others. Some individuals indicate that restraints are used as a fall-prevention measure (Kow & Hogan,

2000; Minnick et al., 2007; Mion et al., 1996; Sandhu et al., 2010; Schleenbaker et al., 1994; Suen et al., 2006), despite a lack of evidence to support this. Others have found that the values, beliefs, and education of nurses and the demographic factors of the patient influence the use of restraints (Evans & FitzGerald, 2002; Ludwick et al., 2008). The use of restraints in hospitalized patients may increase the rates of clinical agitation. Although there may be instances where restraint use is the only viable option remaining in managing challenging behaviors, potential biases and motivations should be examined carefully before concluding restraints are the appropriate course of action.

The use of restraints remains controversial and is not meant to be a part of standard of care practice. Policies related to the use of restraints often state that the use of restraints should meet the following criteria: (1) be individualized and offer as much dignity to the individual as the situation allows, (2) be humanely and professionally administered, (3) have safety protocols in place, (4) patient must be monitored, (5) careful documentation of the type of restraint, the reason for it, and the means for observation while in the restraint, (6) the method or choice of restraint must be the least restrictive option available (American Nurses Association, 2012; College of Nurses of Ontario, 2009; Ministry of Health and Long Term Care for the Province of Ontario, 2001; St. Joseph's Health Care, 2012). In accordance with provincial legislation, the College of Nurses for Ontario suggests that the following information is to be recorded when using restraints: type used, alternatives considered and used, date and time of application, reason given to patient, significant patient behaviours, and patient response.

#### 5.5.4 Addictive Behaviors

Best practices outlined by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI state that all individuals should be screened at time of injury for substance use as a factor in the acquired brain injury (Ontario Neurotrauma Foundation, 2015). Three management principles for substance use disorders following ABI are: (1) management of any substance use disorder should be simultaneous and not sequential to other rehabilitation intervention, (2) substance use should not be a justification for exclusion from other rehabilitation interventions, and (3) incentives should be used by clinicians in order to engage individuals fully in the intervention (Ontario Neurotrauma Foundation, 2015).

[Click here to access the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI for Substance Use](#)

Screening for substance use and other addictive behaviours, such as gambling, may help support individuals and families in receiving more timely interventions. Unfortunately, there is limited evidence to guide interventions for reducing and eliminating substance abuse or gambling among those with an ABI.

**No single intervention has been found to consistently reduce or eliminate substance use in individuals post-ABI** (Bogner & Corrigan, 2013; Corrigan & Bogner, 2007; Corrigan et al., 2005; Corrigan & Mysiw, 2013). Bogner and colleagues (2013) proposed a model of care strategy when approaching the treatment and management of substance use disorders specifically within the ABI population. The Four Quadrant Model (Bogner & Corrigan, 2013) proposes four unique situations varying with the severity of the ABI and substance use. This model considers the stages of ABI rehabilitation that an individual might be moving through and proposes a harmonious strategy based on ABI and substance use severity. Table 5.6 outlines strategies that may provide benefit for substance use when combined with other treatments, such as participation in substance use programs, to improve the likelihood of successful remission from substance

use disorders. In addition to the strategies outlined below, multiple ABI and mental health networks have come together to develop the Substance Abuse and Brain Injury (SUBI) project and create materials in support of the management of those with concurrent brain injury and substance abuse. SUBI developed free materials that are available for both clinicians and individuals with ABI.

[Click here to go to the Substance Abuse and Brain Injury \(SUBI\) Project website](#)

**Table 5.6 Strategies which have been suggested to improve the likelihood of cessation or reduction of substance use.**

Strategy	Description
Motivational interviewing	Motivational interviewing consists of identifying and eliciting an individual's intrinsic motivation for reducing their substance abuse, and then participating in behaviors which clearly promote change. This can involve joint decision making, education, and acknowledgement of the individual's autonomy.
Financial incentive	Providing some form of financial compensation for participation in substance use programs or treatments. There is moderate evidence to support the use of this strategy.
Intensive case management	This strategy involves providing higher than normal amounts of support, coordination, monitoring, and resources than is typically done in an effort to actively reduce and eliminate substance abuse.
Reduction of logistical barriers	The reduction of logistical barriers includes eliminating or reducing physical or organizational barriers which would impact access to treatment, such as transportation, clinical staff, and cost of participation.

Although there are pharmacological interventions which are used in non-ABI populations to treat substance abuse, these have not been evaluated for efficacy in persons who have sustained an ABI. Some of the pharmacological interventions available for the treatment of opioid abuse are naltrexone and methadone, and for the treatment of alcohol abuse are acamprosate and disulfiram (Corrigan & Mysiw, 2013). There are no guidelines for the use of pharmacotherapy for addiction following an ABI, and treatment may have side-effects (Toce et al., 2018). When pharmacological intervention is appropriate, it is best combined with some form of behavioral intervention as well (Corrigan & Mysiw, 2013). Additional resources for information and education on substance abuse following an ABI can be found [HERE](#) at the Acquired Brain Injury Partnership Project.

### 5.5.5 Suicidal Ideation

**Q15. What non-pharmacological interventions have been shown to significantly reduce the sense of hopelessness in individuals with an ABI?**

1. Group cognitive behavioral interventions.

Unfortunately, no non-pharmacological interventions have demonstrated benefit for suicidality following ABI. **However, some studies have shown that CBT can produce a significant reduction in feelings of hopelessness** (Brenner et al., 2017; Simpson et al., 2011). Two RCTs have examined group-based cognitive based therapy in an attempt to reduce suicidal ideation following an ABI. Neither study produced

significant changes on the Beck Scale for Suicide Ideation (Brenner et al., 2017; Simpson et al., 2011). Although these studies demonstrated improved feelings of hopelessness, they did not reduce other symptoms associated with suicidal ideation such as depression.

Signs and symptoms of suicidal ideation are described in section 5.2.5 (suicidal ideation). In clinical practice, managing suicidal ideation involves managing its associated signs and symptoms, such as depression, hopelessness, agitation and aggression, and ensuring patient safety. This may require having patients assessed in the emergency department if risk is imminent, counselling patients and families about the need to seek emergency medical care if suicidal ideation escalates or there is concern of harm, ensuring access to support (e.g. crisis line, supportive friend or family member), and/or contracting to safety and scheduling regular follow-up. Reducing the likelihood of suicide completion may still occur even if the rates of suicidal ideation do not appear to decrease. **Being vigilant of signs and symptoms of suicidal ideation, screening for mood disorders, and treating depression and anxiety may reduce the incidence of suicidal ideation and completion rate in ABI populations.**

### Key Study

Author/Year/ Country/Study Design/ N	Methods	Outcomes
<a href="#">Brenner et al.</a> (2017) Australia RCT PEDro=6 N <sub>Start</sub> =44, N <sub>End</sub> =35	<p><b>Population:</b> TBI. <i>Intervention Group (n=15):</i> Mean Age=47.7 yr; Gender: Male=13, Female=1, Transgender=1. <i>Waitlist Group (n=20):</i> Mean Age=54.6 yr; Gender: Male=19, Female=1, Transgender=0.</p> <p><b>Intervention:</b> Participants were randomized to receive a manualized, small-group cognitive behavioural intervention focused on alleviating hopelessness or to a waitlist. The intervention was 2 hr and delivered weekly for 10 wk. Participants were crossed over to the alternate intervention after 10 wk. Assessments occurred at baseline, 10 wk, and 20 wk.</p> <p><b>Outcome Measure:</b> Beck Hopelessness Scale (BHS), Beck Depression Inventory (BDI), Beck Scale for Suicide Ideation (BSSI).</p>	<ol style="list-style-type: none"> <li>1. After controlling for baseline BHS scores, the intervention group had significantly lower hopelessness post intervention compared to those on the waitlist (p=0.03); these reductions were maintained at follow-up.</li> <li>2. The waitlist group demonstrated significant reductions on the BHS (p=0.01) and depression (p=0.003) after completing the intervention.</li> <li>3. There were no significant between-group differences for the BDI or BSSI.</li> </ol>

## 5.6 Case Study

### Patient Snapshot:

#### Mr. J...

Is a 42-year-old male who was involved in a high speed MVC resulting in a moderate brain injury and orthopedic injuries (fracture to his right tibia and fibula and right wrist). He was admitted to your outpatient Rehabilitation Program for ABI 10-months post-injury for further therapy.

**Lifestyle Factors:** Mr. J has a history of a previous MVC (two years past) that resulted in a number of orthopedic injuries and chronic pain. He has completed a BSc and had just recently returned to work following recovery from the 1<sup>st</sup> MVC. He is recently single and has a supportive family who live in another city. He currently uses medical marijuana to manage pain and assist with sleep.

**Medical History:** Mr. J had an initial GCS of 12, and his duration of post-traumatic amnesia was about four hours and has since resolved. An MRI showed a diffuse axonal injury and cognitive screening at the time was suggestive of mild impairment. He had an open reduction and internal fixation for his tibia and fibula fractures and closed reduction of his wrist fracture. There is no history of alcohol or substance abuse, and he has chronic neuropathic pain.

**Signs & Symptoms:** Mr. J reported persisting symptoms of somatic pain (secondary to fractures) and chronic headache, light and noise sensitivity, subjective impairment of memory and executive functions, low mood and “mood swings” in which he is very irritable. Upon admission Mr. J expressed that his main goal was to improve his functional independence, irritability, and low mood.

Mr. J's previous care provider has acknowledged his neurobehavioral challenges and referred him to you in your out-patient clinic. What do you do next?



Complete a neuroendocrine screen to rule out any underlying causes of his neurobehavioral challenges, complete your own neuropsychological assessments, and communicate with other members of his care team in order to coordinate treatment.

**\*Note:** Mr. J's motor, sensory, and pain management is continued in the Motor and Sensory Case Study which is a part of Chapter 6 of this guidebook.

**Q1. What are some considerations to keep in mind while addressing Mr. J's neurobehavioral deficits?**

1. Confirm whether or not Mr. J has a previous mental health diagnosis, or a history of medication use related to mental health disorders. Previously tried effective medications can be helpful in treatment planning.
2. Recognize that there is a relationship between chronic pain and low mood.
3. Monitor his overall quality of mood and use targeted questions to screen for suicidality (i.e. Have you thought about hurting yourself recently? Have you had thoughts of suicide?).
4. Communicate with his other care team regularly to be aware of his treatments and medications, as these may influence your choice of treatment.
5. Discuss patient preferences of treatment (pharmacological vs. non-pharmacological) to collaboratively develop a treatment plan.
6. Mr. J has expressed that he will continue to use medical marijuana during his recovery.

**Q2. What screening tools can you use to further examine the extent of Mr. J's neurobehavioral concerns?**

1. Take a history and perform a mental status examination.
2. Patient Health Questionnaire-9 (PHQ9).
3. Hospital Anxiety and Depression Scale (HADS).
4. Geriatric Depression Scale (not appropriate in a patient of Mr. J's age).
5. Patient Health Questionnaire 9 Modified for Teens (not appropriate in a patient of Mr. J's age).

**As depression and anxiety are distinct but often comorbid, you conduct both the PHQ9 and HADS.** Mr. J's score on the PHQ9 is 13, while his HADS score is 9, indicating moderate depression, and mild anxiety, respectively. You also take a history to explore Mr. J's mood and anxiety symptoms, and perform a mental status examination. You conclude that Mr. J is experiencing symptoms consistent with depression, and that he has no history of mania. The "mood swings" he experiences are characterized by frustrated outbursts, having less patience ("a short fuse") even with trivial things, and episodes of feeling profoundly sad. He denies symptoms suggestive of post-traumatic stress disorder (PTSD). At present, he denies suicidal or self-harm ideation.

**Q3. Based on these results, what potential therapeutic treatments can you recommend to Mr. J?**

1. Cognitive behavioral therapy (CBT) with a referral to a clinical psychologist.
  2. Pharmacotherapy for depression and anxiety.
  3. Combination CBT and pharmacotherapy.
- \*Mr. J has the option to use these therapies simultaneously or sequentially based on his preferences and the severity of his symptoms.

**\*Note: You hear from Mr. J's other treating physician that he is being treated with gabapentin for his pain.**

Based on Mr. J's desire to manage his mood as quickly as possible you both agree that he will be referred to a clinical psychologist for CBT and start pharmacotherapy simultaneously. Being mindful of the fact that Mr. J is using medical marijuana and already on gabapentin you prescribe him venlafaxine, which may also have benefits for his pain management.

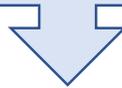
### Therapy Breakdown:

CBT → Mr. J attends CBT with a clinical psychologist once weekly

#### Q4. What other considerations should you be mindful of with regards to Mr. J's treatment and progress?

1. Regularly follow-up with Mr. J to monitor potential suicidal ideation and have him create a safety plan.
2. Recognize that it may take up to 4-6 weeks to see the effects of the venlafaxine, however side effects may present right away so explain to Mr. J what to expect.

**You've been treating Mr. J for 6 weeks now, and at your follow-up he explains that he feels the medication isn't working, although he feels he's gained some benefits from attending CBT. What do you do next?**



Mr. J's pain is being managed sufficiently and he's expressed that he believes CBT is relieving some of the negative mood. You agree that he'll remain on venlafaxine for 6 more weeks and if he still feels there's no added benefit from the venlafaxine at that time you'll reassess and switch medications.



#### Clinical Tip!

Listen to the patient and their constellation of symptoms in order to better understand their compliment of symptoms and the potential neuropsychological factors contributing to them.

**It's been 3 months since you started treating Mr. J and you're checking in again...**



Mr. J has seen some progress with respect to this mood, anxiety, and depression and is now attending CBT once bi-weekly. However, he still feels that his progress is too slow and that he would like to try a different medication. He's neither expressed nor shown any indications of suicidality during your treatment of him and is looking forward to going back to work once the remainder of his motor symptoms resolve.

**Q6. Based on Mr. J's wishes, what other treatment options can you offer him?**

1. Sertraline.
2. Citalopram.
3. Methylphenidate.
4. Duloxetine.
5. Fluoxetine.
6. Paroxetine.

\*There is limited evidence to guide antidepressant therapy after acquired brain injury. These treatment options are listed in no particular order. When prescribing antidepressant therapy, treatment choices are based on the same principles as managing depression in a non-brain injured population.

You prescribe Mr. J sertraline and schedule your next follow-up session for 3 months as he's progressing well.

**It's been 3 months since you started Mr. J on sertraline and you're checking in again...**



Mr. J seems very happy with the progress that he's made, both neuropsychologically and with his comorbid motor deficits. He seems engaged and motivated to continue to work to improve his progress. He asks how long he can expect to continue on sertraline. You recommend at least 6 months of treatment before a trial of discontinuation. You explain that a longer course of treatment may be needed if his symptoms return during the trial of discontinuation. You also recommend considering postponing a trial of discontinuation if it coincides with attempted return to work or other important milestones to avoid changing too many things at once.

**Conclusion:**

Based on the stabilization of Mr. J's mood, his reduction in pain, your review of the notes from the clinical psychologist, and his continued progress (emotionally and physically) you discharge him from your care with the understanding that his family physician will continue his neurobehavioral management.

## 5.7 References

- Aboulafia-Brakha, T., Greber Buschbeck, C., Rochat, L., & Annoni, J. M. (2013). Feasibility and initial efficacy of a cognitive-behavioural group programme for managing anger and aggressiveness after traumatic brain injury. *Neuropsychological rehabilitation, 23*(2), 216-233.
- Ahmedani, B. K., Peterson, E. L., Hu, Y., Rossom, R. C., Lynch, F., Lu, C. Y., Waitzfelder, B. E., Owen-Smith, A. A., Hubley, S., Prabhakar, D., Williams, L. K., Zeld, N., Mutter, E., Beck, A., Tolsma, D., & Simon, G. E. (2017). Major physical health conditions and risk of suicide. *American Journal of Preventive Medicine, 53*(3), 308-315.
- Alway, Y., McKay, A., Ponsford, J., & Schönberger, M. (2012). Expressed emotion and its relationship to anxiety and depression after traumatic brain injury. *Neuropsychological rehabilitation, 22*(3), 374-390.
- American Addiction Centers. (2019). Alcohol and Drug Abuse Statistics Retrieved from <https://americanaddictioncenters.org/rehab-guide/addiction-statistics>
- American Nurses Association. (2012). Position statement: Reduction of patient restraint and seclusion in health care settings. Retrieved from <http://www.nursingworld.org/restraintposition>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders -5* (S. K. K. Schultz, E. A. Ed.). Washington, DC: American Psychiatric Association
- Andelic, N., Jerstad, T., Sigurdardottir, S., Schanke, A. K., Sandvik, L., & Roe, C. (2010). Effects of acute substance use and pre-injury substance abuse on traumatic brain injury severity in adults admitted to a trauma centre. *J Trauma Manag Outcomes, 4*, 6.
- Anke, A., Andelic, N., Skandsen, T., Knoph, R., Ader, T., Manskow, U., Sigurdardottir, S., & Roe, C. (2015). Functional Recovery and Life Satisfaction in the First Year after Severe Traumatic Brain Injury: A Prospective Multicenter Study of a Norwegian National Cohort. *Journal of Head Trauma Rehabilitation, 30*(4), E38-E49.
- Baer, R. A. (2003). Mindfulness Training as a Clinical Intervention: A Conceptual and Empirical Review. *Clinical Psychology: Science and Practice, 10*(2), 125-143.
- Bahraini, N. H., Simpson, G. K., Brenner, L. A., Hoffberg, A. S., & Schneider, A. L. (2013). Suicidal ideation and behaviours after traumatic brain injury: A systematic review. *Brain Impairment, 14*(1), 92-112.
- Bakchine, S., Lacomblez, L., Benoit, N., Parisot, D., Chain, F., & Lhermitte, F. (1989). Manic-like state after bilateral orbitofrontal and right temporoparietal injury: Efficacy of clonidine. *Neurology, 39*(6), 777-781.
- Bay, E., & Covassin, T. (2012). Chronic Stress, Somatic and Depressive Symptoms Following Mild to Moderate Traumatic Brain Injury. *Archives of Psychiatric Nursing, 26*(6), 477-486.
- Bedard, Felteau, M., Mazmanian, D., Fedyk, K., Klein, R., Richardson, J., Parkinson, W., & Minthorn-Biggs, M.-B. (2003). Pilot evaluation of a mindfulness-based intervention to improve quality of life among individuals who sustained traumatic brain injuries. *Disability & Rehabilitation, 25*(13), 722-731.
- Bedard, M., Felteau, M., Marshall, S., Campbell, S., Gibbons, C., Klein, R., & Weaver, B. (2012). Mindfulness-based cognitive therapy: benefits in reducing depression following a traumatic brain injury. *Advances in mind-body medicine, 26*(1), 14-20.
- Bedard, M., Felteau, M., Marshall, S., Cullen, N., Gibbons, C., Dubois, S., Maxwell, H., Mazmanian, D., Weaver, B., Rees, L., Gainer, R., Klein, R., & Moustgaard, A. (2014). Mindfulness-based cognitive therapy reduces symptoms of depression in people with a traumatic brain injury: Results from a randomized controlled trial. *Journal of Head Trauma Rehabilitation, 29*(4), E13-E22.

- Berthier, M. L., Kulisevsky, J., Gironell, A., & López, O. L. (2001). Obsessive-compulsive disorder and traumatic brain injury: Behavioral, cognitive, and neuroimaging findings. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 14(1), 23-31.
- Bhatti, J. A., Thiruchelvam, D., & Redelmeier, D. A. (2019). Traumatic brain injury as an independent risk factor for problem gambling: a matched case-control study. *Social Psychiatry and Psychiatric Epidemiology*, 54(4), 517-523.
- Bogner, J. (2000). *The Agitated Behavior Scale*. Retrieved from
- Bogner, J., & Corrigan, J. D. (2013). Interventions for substance misuse following TBI: A systematic review. *Brain Impairment*, 14(1), 77-91.
- Bogner, J. A., Corrigan, J. D., Fugate, L., Mysiw, W. J., & Clinchot, D. (2001). Role of agitation in prediction of outcomes after traumatic brain injury. *Am J Phys Med Rehabil*, 80(9), 636-644.
- Bombardier, C. H., Hoekstra, T., Dikmen, S., & Fann, J. R. (2016). Depression trajectories during the first year after traumatic brain injury. *Journal of Neurotrauma*, 33(23), 2115-2124.
- Bombardier, C. H., Rimmele, C. T., & Zintel, H. (2002). The magnitude and correlates of alcohol and drug use before traumatic brain injury. *Arch Phys Med Rehabil*, 83(12), 1765-1773.
- Bombardier, C. H., Temkin, N. R., Machamer, J., & Dikmen, S. S. (2003). The natural history of drinking and alcohol-related problems after traumatic brain injury. *Arch Phys Med Rehabil*, 84(2), 185-191.
- Brenner, L. A., Forster, J. E., Hoffberg, A. S., Matarazzo, B. B., Hostetter, T. A., Signoracci, G., & Simpson, G. K. (2017). Window to Hope: A Randomized Controlled Trial of a Psychological Intervention for the Treatment of Hopelessness Among Veterans With Moderate to Severe Traumatic Brain Injury. *J Head Trauma Rehabil*.
- Brody, A. L., Barsom, M. W., Bota, R. G., & Saxena, S. (2001). Prefrontal-subcortical and limbic circuit mediation of major depressive disorder. *Semin Clin Neuropsychiatry*, 6(2), 102-112.
- Bryson, C. N., Cramer, R. J., & Schmidt, A. T. (2017). Traumatic brain injury and lifetime suicidality: Applying the interpersonal-psychological theory perspective. *Death Studies*, 41(7), 399-405.
- Burke, W. H., Wesolowski, M. D., & Lane, I. (1988). A positive approach to the treatment of aggressive brain injured clients. *International Journal of Rehabilitation Research*, 11(3), 235-241.
- Busch, A. B., & Shore, M. F. (2000). Seclusion and restraint: A review of recent literature. *Harvard Review of Psychiatry*, 8(5), 261-270.
- Cifu, D., Caruso, D. . (2010). *Traumatic Brain Injury*. New York: Demos Medical Publishing.
- Ciurli, P., Formisano, R., Bivona, U., Cantagallo, A., & Angelelli, P. (2011). Neuropsychiatric disorders in persons with severe traumatic brain injury: prevalence, phenomenology, and relationship with demographic, clinical, and functional features. *J Head Trauma Rehabil*, 26(2), 116-126.
- Cnossen, M. C., Scholten, A. C., Lingsma, H. F., Haagsma, J., & Polinder, S. (2016). Predicting major depression and posttraumatic stress disorder after traumatic brain injury: A systematic review. *Brain Injury*, 30(5-6), 517.
- College of Nurses of Ontario. (2009). The Standard of Care-Practice Standard. Retrieved from <http://www.cno.org>
- Corrigan, J. D. (1989). Development of a scale for assessment of agitation following traumatic brain injury. *J Clin Exp Neuropsychol*, 11(2), 261-277.
- Corrigan, J. D., & Bogner, J. (2007). Interventions to promote retention in substance abuse treatment. *Brain Injury*, 21(4), 343-356.
- Corrigan, J. D., Bogner, J., Lamb-Hart, G., Heinemann, A. W., & Moore, D. (2005). Increasing substance abuse treatment compliance for persons with traumatic brain injury. *Psychol Addict Behav*, 19(2), 131-139.
- Corrigan, J. D., Bogner, J. A., Mysiw, W. J., Clinchot, D., & Fugate, L. (1997). Systematic bias in outcome studies of persons with traumatic brain injury. *Arch Phys Med Rehabil*, 78(2), 132-137.

- Corrigan, J. D., Harrison-Felix, C., Bogner, J., Dijkers, M., Terrill, M. S., & Whiteneck, G. (2003). Systematic bias in traumatic brain injury outcome studies because of loss to follow-up. *Arch Phys Med Rehabil*, 84(2), 153-160.
- Corrigan, J. D., & Mysiw, W. J. (2013). Substance Misuse Among Persons with Traumatic Brain Injury In K. D. Zasler ND, Zafonte RD. (Ed.), *Brain Injury Medicine: Principles and Practice* (2nd ed., pp. 1315-1328). New York: Demos Medical Publishing.
- Crane, B. (2002). Full catastrophe living; using the wisdom of your body and mind to face stress, pain and illness. Jon Kabat-Zinn, 1996 Publisher: Piatkus, London ISBN: 0-749-915-854. *Spirituality and Health International*, 3(2), 52-52.
- Cully, J. A., & Teten, A. L. (2008). *A Therapist's Guide to Brief Cognitive Behavioral Therapy* Vol. 1.
- Deb, S., Lyons, I., Koutzoukis, C., Ali, I., & McCarthy, G. (1999). Rate of psychiatric illness 1 year after traumatic brain injury. *American Journal of Psychiatry*, 156(3), 374-378.
- DeLambo, D. A., Chandras, K. V., Homa, D., & Chandras, S. V. (2008). Psychiatric disabilities and substance abuse: Applications for rehabilitation professionals. *Compelling counseling intervention: Celebrating VISTAS'fifth anniversary*, 149-159.
- Denny-Brown, D. (1945). Disability arising from closed head injury. *JAMA*, 127, 429-436.
- Drevets, W. C., & Raichle, M. E. (1992). Neuroanatomical circuits in depression: implications for treatment mechanisms. *Psychopharmacol Bull*, 28(3), 261-274.
- Duxbury, J., & Wright, K. (2011). Should nurses restrain violent and aggressive patients? *Nursing times*, 107(9), 22-25.
- Eisenberg, M. E., Im, B., Swift, P., & Flanagan, S. R. (2009). Management of Traumatic Brain Injury-Related Agitation. *21(3-4)*, 215-229.
- Evans, D., & FitzGerald, M. (2002). Reasons for physically restraining patients and residents: A systematic review and content analysis. *International Journal of Nursing Studies*, 39(7), 735-743.
- Fann, J. R., Katon, W. J., Uomoto, J. M., & Esselman, P. C. (1995). Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *American Journal of Psychiatry*, 152(10), 1493-1499.
- Fisher, L. B., Pedrelli, P., Iverson, G. L., Bergquist, T. F., Bombardier, C. H., Hammond, F. M., Hart, T., Ketchum, J. M., Giacino, J., & Zafonte, R. (2016). Prevalence of suicidal behaviour following traumatic brain injury: Longitudinal follow-up data from the NIDRR Traumatic Brain Injury Model Systems. *Brain Injury*, 1-8.
- Fjorback, L. O., Arendt, M., Ørnbøl, E., Fink, P., & Walach, H. (2011). Mindfulness-Based Stress Reduction and Mindfulness-Based Cognitive Therapy – a systematic review of randomized controlled trials. *Acta Psychiatr Scand*, 124(2), 102-119.
- Fleminger, S., Oliver, D. L., Williams, W. H., & Evans, J. (2003). The neuropsychiatry of depression after brain injury. *Neuropsychological Rehabilitation*, 13(1-2), 65-87.
- Fugate, L. P., Spacek, L. A., Kresty, L. A., Levy, C. E., Johnson, J. C., & Mysiw, W. J. (1997). Definition of agitation following traumatic brain injury: I. a survey of the brain injury special interest group of the american academy of physical medicine and rehabilitation. *Arch Phys Med Rehabil*, 78(9), 917-923.
- Gould, K. R., Ponsford, J. L., & Spitz, G. (2014). Association between cognitive impairments and anxiety disorders following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 36(1), 1-14.
- Gregory Jr, H. H., & Bonfiglio, R. P. (1995). Limiting restraint use for behavior control: the brain injury rehabilitation unit as a model. *Maryland medical journal (Baltimore, Md. : 1985)*, 44(4), 279-283.
- Gutierrez, P. M., Brenner, L. A., & Huggins, J. A. (2008). A preliminary investigation of suicidality in psychiatrically hospitalized veterans with traumatic brain injury. *Archives of Suicide Research*, 12(4), 336-343.

- Hart, T., Fann, J. R., Chervoneva, I., Juengst, S. B., Rosenthal, J. A., Krellman, J. W., Dreer, L. E., & Kroenke, K. (2016). Prevalence, Risk Factors, and Correlates of Anxiety at 1 Year after Moderate to Severe Traumatic Brain Injury. *Arch Phys Med Rehabil*, 97(5), 701-707.
- Hart, T., Vaccaro, M. J., Hays, C., & Maiuro, R. D. (2012). Anger self-management training for people with traumatic brain injury: A preliminary investigation. *Journal of Head Trauma Rehabilitation*, 27(2), 113-122.
- Hibbard, M. R., Uysal, S., Kepler, K., Bogdany, J., & Silver, J. (1998). Axis I psychopathology in individuals with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 13(4), 24-39.
- Hoffman, J. M., Bell, K. R., Powell, J. M., Behr, J., Dunn, E. C., Dikmen, S., & Bombardier, C. H. (2010). A randomized controlled trial of exercise to improve mood after traumatic brain injury. *PM and R*, 2(10), 911-919.
- Hudak, A. M., Hynan, L. S., Harper, C. R., & Diaz-Arrastia, R. (2012). Association of depressive symptoms with functional outcome after traumatic brain injury. *J Head Trauma Rehabil*, 27(2), 87-98.
- INESSS-ONF. (2015). *Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI*. Retrieved from Ontario: <https://braininjuryguidelines.org/modtosevere/>
- J. Van Der Naalt, A. H. V. Z. W. J. S. J. M. M. (2000). Acute behavioural disturbances related to imaging studies and outcome in mild-to-moderate head injury. *Brain Injury*, 14(9), 781-788.
- Janzen, S., McIntyre, A., Meyer, M., Sequeira, K., & Teasell, R. (2014). The management of agitation among inpatients in a brain injury rehabilitation unit. *Brain Injury*, 28(3), 318-322.
- Jean-Bay, E. (2000). The biobehavioral correlates of post-traumatic brain injury depression. *The Journal of neuroscience nursing : journal of the American Association of Neuroscience Nurses*, 32(3), 169-176.
- Jorge, R. E. (2005). Neuropsychiatric consequences of traumatic brain injury: A review of recent findings. *Current Opinion in Psychiatry*, 18(3), 289-299.
- Kabat-Zinn, J. (2000). Indra's net at work: The mainstreaming of Dharma practice in society *The psychology of awakening: Buddhism, science, and our day-to-day lives*. (pp. 225-249). York Beach, ME, US: Samuel Weiser.
- Karol, R. (2003). Neuropsychosocial intervention; the practical treatment of severe behavioral dyscontrol after acquired brain injury (Vol. 27). Portland: Ringgold Inc.
- Karol, R. (2013). Principles of Behavioral Analysis and Treatment. In K. D. Zasler ND, Zafonte RD. (Ed.), *Brain Injury Medicine: Principles and Practice* (2nd ed., pp. 1053-1066). New York: Demos Medical Publishing.
- Kelly, D. F., McArthur, D. L., Levin, H., Swimmer, S., Dusick, J. R., Cohan, P., Wang, C., & Swerdloff, R. (2006). Neurobehavioral and quality of life changes associated with growth hormone insufficiency after complicated mild, moderate, or severe traumatic brain injury. *Journal of Neurotrauma*, 23(6), 928-942.
- Kelly, G., Simpson, G. K., Brown, S., Kremer, P., & Gillett, L. (2019). The Overt Behaviour Scale-Self-Report (OBS-SR) for acquired brain injury: exploratory analysis of reliability and validity. *Neuropsychological rehabilitation*, 29(5), 704-722.
- Kelly, M. P., Johnson, C. T., Knoller, N., Drubach, D. A., & Winslow, M. M. (1997). Substance abuse, traumatic brain injury and neuropsychological outcome. *Brain Inj*, 11(6), 391-402.
- Kim, E., & Humaran, T. J. (2002). Divalproex in the management of neuropsychiatric complications of remote acquired brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 14(2), 202-205.
- Kolakowsky-Hayner, S. A., Gourley Iii, E. V., Kreutzer, J. S., Marwitz, J. H., Meade, M. A., & Cifu, D. X. (2002). Post-injury substance abuse among persons with brain injury and persons with spinal cord injury. *Brain Injury*, 16(7), 583-592.

- Kow, J. V., & Hogan, D. B. (2000). Use of physical and chemical restraints in medical teaching units. *CMAJ*, *162*(3), 339-340.
- Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606-613.
- Kwok, C., McIntyre, A., Janzen, S., Sequeira, K., & Teasell, R. (2013). Drug and alcohol abuse among individuals with acquired brain injury. *CJAM Canadian Journal of Addiction Medicine*, *4*(2), 14-19.
- Lamontagne, M.-E., Truchon, C., Kagan, C., Bayley, M., Swaine, B., Marshall, S., Kua, A., Allaire, A.-S., Marier Deschenes, P., & Gargaro, J. (2017). *INESSS-ONF clinical practice guidelines for the rehabilitation of adults having sustained a moderate-to-severe TBI*.
- Lee, H., Kim, S. W., Shin, I. S., Yang, S. J., & Yoon, J. S. (2005). Comparing effects of methylphenidate, sertraline and placebo on neuropsychiatric sequelae in patients with traumatic brain injury. *Human Psychopharmacology*, *20*(2), 97-104.
- Levin, H. S., & Grossman, R. G. (1978). Behavioral Sequelae of Closed Head Injury: A Quantitative Study. *Archives of Neurology*, *35*(11), 720-727.
- Liotti, M., & Mayberg, H. S. (2001). The role of functional neuroimaging in the neuropsychology of depression. *J Clin Exp Neuropsychol*, *23*(1), 121-136.
- Ludwick, R., Meehan, A., Zeller, R., & O'Toole, R. (2008). Safety work: Initiating, maintaining, and terminating restraints. *Clinical Nurse Specialist*, *22*(2), 81-87.
- Mackelprang, J. L., Bombardier, C. H., Fann, J. R., Temkin, N. R., Barber, J. K., & Dikmen, S. S. (2014). Rates and predictors of suicidal ideation during the first year after traumatic brain injury. *American journal of public health*, *104*(7), e100-107.
- Marks, W. (1992). Physical restraints in the practice of medicine. Current concepts. *Arch Intern Med*, *152*(11), 2203-2206.
- McAllister, T. (2011). *Neurobiological consequences of traumatic brain injury* (Vol. 13).
- McAllister, T. (2013). Emotional and Behavioral Sequelae of Traumatic Brain Injury. In N. Zasler, Katz, D., Zafonte, R. (Ed.), *Brain Injury Medicine; Principles and Practice* (2nd ed., pp. 1034-1052). New York: Demos Medical Publishing.
- McDonald, S., Tate, R., Togher, L., Bornhofen, C., Long, E., Gertler, P., & Bowen, R. (2008). Social Skills Treatment for People With Severe, Chronic Acquired Brain Injuries: A Multicenter Trial. *Arch Phys Med Rehabil*, *89*(9), 1648-1659.
- McNett, M., Sarver, W., & Wilczewski, P. (2012). The prevalence, treatment and outcomes of agitation among patients with brain injury admitted to acute care units. *Brain Injury*, *26*(9), 1155-1162.
- Medd, J., & Tate, R. L. (2000). Evaluation of an anger management therapy programme following acquired brain injury: A preliminary study. *Neuropsychological rehabilitation*, *10*(2), 185-201.
- Ministry of Health and Long Term Care for the Province of Ontario. (2001). *Patient Restraints Minimization Act*. Retrieved from <http://www.e-laws.gov.on.ca/html/source/statutes/english/2001>.
- Minnick, A. F., Mion, L. C., Johnson, M. E., Catrambone, C., & Leipzig, R. (2007). Prevalence and variation of physical restraint use in acute care settings in the US: Clinical scholarship. *Journal of Nursing Scholarship*, *39*(1), 30-37.
- Mion, L. C., Minnick, A., & Palmer, R. (1996). Physical Restraint Use in the Hospital Setting: Unresolved Issues and Directions for Research. *Milbank Quarterly*, *74*(3), 411-433.
- Morrison, J., Crinklaw-Wiancko, D., King, D., Thibeault, S., & Wells, D. L. (1987). Formulating a restraint use policy. *Journal of Nursing Administration*, *17*(3), 39-42.
- Nordqvist, C. (2018). What are suicidal thoughts? Retrieved from <https://www.medicalnewstoday.com/kc/suicidal-thoughts-ideation-193026>.
- Nott, M. T., Chapparo, C., & Baguley, I. J. (2006). Agitation following traumatic brain injury: An Australian sample. *Brain Injury*, *20*(11), 1175-1182.

- O'Donnell, M. L., Bryant, R. A., Creamer, M., & Carty, J. (2008). Mental health following traumatic injury: Toward a health system model of early psychological intervention. *Clinical Psychology Review, 28*(3), 387-406.
- O'Leary, C. A. (2000). Reducing aggression in adults with brain injuries. *Behavioral Interventions, 15*(3), 205-216.
- Oddo, M., Crippa, I. A., Mehta, S., Menon, D., Payen, J.-F., Taccone, F. S., & Citerio, G. (2016a). Optimizing sedation in patients with acute brain injury. *Critical care, 20*(1), 128.
- Oddo, M., Crippa, I. A., Mehta, S., Menon, D., Payen, J.-F., Taccone, F. S., & Citerio, G. (2016b). Optimizing sedation in patients with acute brain injury. *Critical care (London, England), 20*(1), 128.
- Ontario Neurotrauma Foundation. (2015). Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI. 1rst. Retrieved from <https://braininjuryguidelines.org/modtosevere/>
- Osborn, A. J., Mathias, J. L., & Fairweather-Schmidt, A. K. (2014). Depression following adult, non-penetrating traumatic brain injury: A meta-analysis examining methodological variables and sample characteristics. *Neuroscience and Biobehavioral Reviews, 47*, 1-15.
- Osborn, A. J., Mathias, J. L., & Fairweather-Schmidt, A. K. (2015). Prevalence of Anxiety Following Adult Traumatic Brain Injury: A Meta-Analysis Comparing Measures, Samples and Postinjury Intervals. *Neuropsychology., 06*.
- Ownsworth, T. L., & Oei, T. P. (1998). Depression after traumatic brain injury: conceptualization and treatment considerations. *Brain Inj, 12*(9), 735-751.
- Perino, C., Rago, R., Cicolin, A., Torta, R., & Monaco, F. (2001). Mood and behavioural disorders following traumatic brain injury: Clinical evaluation and pharmacological management. *Brain Injury, 15*(2), 139-148.
- Ponsford, J., Lee, N. K., Wong, D., McKay, A., Haines, K., Alway, Y., Downing, M., Furtado, C., & O'Donnell, M. L. (2016). Efficacy of motivational interviewing and cognitive behavioral therapy for anxiety and depression symptoms following traumatic brain injury. *Psychol Med, 46*(5), 1079-1090.
- Ponsford, J., Whelan-Goodinson, R., & Bahar-Fuchs, A. (2007). Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury, 21*(13-14), 1385-1392.
- Quittner, A., Abbott, J., M Georgiopoulos, A., Goldbeck, L., Smith, B., E Hempstead, S., Marshall, B., Sadoska, K., & Elborn, S. (2015). *International Committee on Mental Health in Cystic Fibrosis: Cystic Fibrosis Foundation and European Cystic Fibrosis Society consensus statements for screening and treating depression and anxiety* (Vol. 71).
- Rapoport, M. J., Chan, F., Lanctot, K., Herrmann, N., McCullagh, S., & Feinstein, A. (2008). An open-label study of citalopram for major depression following traumatic brain injury. *Journal of Psychopharmacology, 22*(8), 860-864.
- Rector, N. A. (2010). *Cognitive Behavioral Therapy: An information guide*. Retrieved from Canada:
- Reyes, R. L., Bhattacharyya, A. K., & Heller, D. (1981). Traumatic head injury: restlessness and agitation as prognosticators of physical and psychologic improvement in patients. *Arch Phys Med Rehabil, 62*(1), 20-23.
- Rosati, D. L. (2002). Early polyneuropharmacologic intervention in brain injury agitation. *American Journal of Physical Medicine and Rehabilitation, 81*(2), 90-93.
- Rosenthal, M., Christensen, B. K., & Ross, T. P. (1998). Depression following traumatic brain injury. *Arch Phys Med Rehabil, 79*(1), 90-103.
- Sabaz, M., Simpson, G. K., Walker, A. J., Rogers, J. M., Gillis, I., & Strettles, B. (2014). Prevalence, comorbidities, and correlates of challenging behavior among community-dwelling adults with

- severe traumatic brain injury: A multicenter study. *Journal of Head Trauma Rehabilitation*, 29(2), E19-E30.
- Sandhu, S. K., Mion, L. C., Khan, R. H., Ludwick, R., Claridge, J., Pile, J. C., Harrington, M., Winchell, J., & Dietrich, M. S. (2010). Likelihood of ordering physical restraints: Influence of physician characteristics. *Journal of the American Geriatrics Society*, 58(7), 1272-1278.
- Schleenbaker, R. E., McDowell, S. M., Moore, R. W., Costich, J. F., & Prater, G. (1994). Restraint use in inpatient rehabilitation: Incidence, predictors, and implications. *Arch Phys Med Rehabil*, 75(4), 427-430.
- Seel, R. T., MacCiocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major depression after moderate to severe tBI. *Journal of Head Trauma Rehabilitation*, 25(2), 99-112.
- Sigurdardottir, S., Andelic, N., Røe, C., & Schanke, A. K. (2013). Depressive symptoms and psychological distress during the first five years after traumatic brain injury: Relationship with psychosocial stressors, fatigue and pain. *Journal of Rehabilitation Medicine*, 45(8), 808-814.
- Simpson, G. H., & Tate, R. (2002). Suicidality after traumatic brain injury: Demographic, injury and clinical correlates. *Psychol Med*, 32(4), 687-697.
- Simpson, G. H., & Tate, R. (2005). Clinical features of suicide attempts after traumatic brain injury. *Journal of Nervous and Mental Disease*, 193(10), 680-685.
- Simpson, G. K., & Tate, R. L. (2007). Suicidality in people surviving a traumatic brain injury: Prevalence, risk factors and implications for clinical management. *Brain Injury*, 21(13-14), 1335-1351.
- Simpson, G. K., Tate, R. L., Whiting, D. L., & Cotter, R. E. (2011). Suicide prevention after traumatic brain injury: A randomized controlled trial of a program for the psychological treatment of hopelessness. *Journal of Head Trauma Rehabilitation*, 26(4), 290-300.
- Singh, R., Mason, S., Lecky, F., & Dawson, J. (2018). Prevalence of depression after TBI in a prospective cohort: The SHEFBIT study. *Brain Inj*, 32(1), 84-90.
- Singh, R., Venkateshwara, G., Nair, K. P. S., Khan, M., & Saad, R. (2014). Agitation after traumatic brain injury and predictors of outcome. *Brain Injury*, 28(3), 336-340.
- Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A Brief Measure for Assessing Generalized Anxiety Disorder: The GAD-7. *Arch Intern Med*, 166(10), 1092-1097.
- St. Joseph's Health Care. (2012). Minimal Use of Restraints of Voluntary Patients and Residents Retrieved from [http://intra.sjhc.london.on.ca/policy/search\\_res.php](http://intra.sjhc.london.on.ca/policy/search_res.php)
- Stanislav, S. W., & Childs, A. (2000). Evaluating the usage of droperidol in acutely agitated persons with brain injury. *Brain Injury*, 14(3), 261-265.
- Suen, L. K. P., Lai, C. K. Y., Wong, T. K. S., Chow, S. K. Y., Kong, S. K. F., Ho, J. Y. L., Kong, T. K., Leung, J. S. C., & Wong, I. Y. C. (2006). Use of physical restraints in rehabilitation settings: Staff knowledge, attitudes and predictors. *Journal of Advanced Nursing*, 55(1), 20-28.
- Tateno, A., Jorge, R. E., & Robinson, R. G. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 15(2), 155-160.
- Taylor, L. A., Kreutzer, J. S., Demm, S. R., & Meade, M. A. (2003). Traumatic brain injury and substance abuse: A review and analysis of the literature. *Neuropsychol Rehabil*, 13(1-2), 165-188.
- Thompson, V. L. S. (2019). Moving Beyond Mental Illness to Mental Health and Wellbeing. *Health*.
- Toce, M. S., Chai, P. R., Burns, M. M., & Boyer, E. W. (2018). Pharmacologic Treatment of Opioid Use Disorder: a Review of Pharmacotherapy, Adjuncts, and Toxicity. *J Med Toxicol*, 14(4), 306-322.
- Tsaousides, T., Cantor, J. B., & Gordon, W. A. (2011). Suicidal ideation following traumatic brain injury: prevalence rates and correlates in adults living in the community. *Journal of Head Trauma Rehabilitation*, 26(4), 265-275.
- Underhill, A. T., Lobello, S. G., Stroud, T. P., Terry, K. S., Devivo, M. J., & Fine, P. R. (2003). Depression and life satisfaction in patients with traumatic brain injury: A longitudinal study. *Brain Injury*, 17(11), 973-982.

- van Reekum, R., Bolago, I., Finlayson, M. A., Garner, S., & Links, P. S. (1996). Psychiatric disorders after traumatic brain injury. *Brain Inj*, *10*(5), 319-327.
- van Reekum, R., Cohen, T., & Wong, J. (2000). Can traumatic brain injury cause psychiatric disorders? *J Neuropsychiatry Clin Neurosci*, *12*(3), 316-327.
- Walker, A. J., Nott, M. T., Doyle, M., Onus, M., McCarthy, K., & Baguley, I. J. (2010). Effectiveness of a group anger management programme after severe traumatic brain injury. *Brain Inj*, *24*(3), 517-524.
- Warriner, E. M., & Velikonja, D. (2006). Psychiatric disturbances after traumatic brain injury: Neurobehavioral and personality changes. *Current Psychiatry Reports*, *8*(1), 73-80.
- Wehman, P., Targett, P., Yasuda, S., & Brown, T. (2000). Return to work for individuals with TBI and a history of substance abuse. *NeuroRehabilitation*, *15*(1), 71-77.
- West, S. L., Graham, C. W., & Cifu, D. X. (2009). Rates of Alcohol/Other Drug Treatment Denials to Persons With Physical Disabilities: Accessibility Concerns. *Alcoholism Treatment Quarterly*, *27*(3), 305-316.
- Whelan-Goodinson, R., Ponsford, J., Johnston, L., & Grant, F. (2009). Psychiatric disorders following traumatic brain injury: their nature and frequency. *J Head Trauma Rehabil*, *24*(5), 324-332.
- Williams, K. A., Kolar, M. M., Reger, B. E., & Pearson, J. C. (2001). Evaluation of a Wellness-Based Mindfulness Stress Reduction Intervention: A Controlled Trial. *American Journal of Health Promotion*, *15*(6), 422-432.
- Wisco, B. E., Marx, B. P., Holowka, D. W., Vasterling, J. J., Han, S. C., Chen, M. S., Gradus, J. L., Nock, M. K., Rosen, R. C., & Keane, T. M. (2014). Traumatic brain injury, PTSD, and current suicidal ideation among Iraq and Afghanistan U.S. veterans. *Journal of traumatic stress*, *27*(2), 244-248.
- Wolffbrandt, M. M., Poulsen, I., Engberg, A. W., & Hornnes, N. (2013). Occurrence and severity of agitated behavior after severe traumatic brain injury. *Rehabil Nurs*, *38*(3), 133-141.
- Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatr Scand*, *67*(6), 361-370.