

ERABI

EVIDENCE-BASED REVIEW
of moderate to severe
ACQUIRED BRAIN INJURY

Clinical Guidebook

13. Fatigue and Sleep Disorders Post Acquired Brain Injury

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Fatigue and Sleep Disorders Post ABI

By the end of this chapter you should know:

- The most common sleep disorders following an acquired brain injury (ABI)
- How to identify fatigue and sleep disorders in individuals with an ABI
- How and when to screen for/test fatigue and sleep disorders in individuals with an ABI
- Potential case management scenarios

13.1 Introduction

Fatigue and sleep disorders following acquired brain injury (ABI) are common and persistent, occurring across a range of brain injury severities (Cronin & O’Loughlin, 2018). Given the impact these symptoms can have on quality of life, activities of daily living, social functioning and the course of recovery in rehabilitation, this is an important area in the field of neurorehabilitation. This guidebook presents a clinical overview for the assessment, diagnosis and management of fatigue and sleep disorders following an ABI.

13.2 Fatigue

Fatigue has numerous definitions and classifications, reflecting multiple perspectives from patients to individuals involved in healthcare and research. However, clinically, *fatigue* is commonly defined as *feelings of physical and/or mental exhaustion during or after usual activities, or feelings of inadequate energy to begin activities* (Aaronson et al., 1999; Chen, 1986).

13.2.1 Classification

Fatigue is typically classified as *physical, psychological or mental* (summarized in Table 1). Briefly, physical fatigue is associated with reduced performance in physical tasks, while psychological fatigue is associated with depression, anxiety, and other psychological conditions (Bell, 2015). Distinct from psychological fatigue, mental fatigue is associated with reduced performance in cognitive tasks (i.e., attention, concentration and memory; Bell, 2015). Physical, psychological and mental fatigue are not mutually exclusive; they often appear together and may overlap (Figure 1).

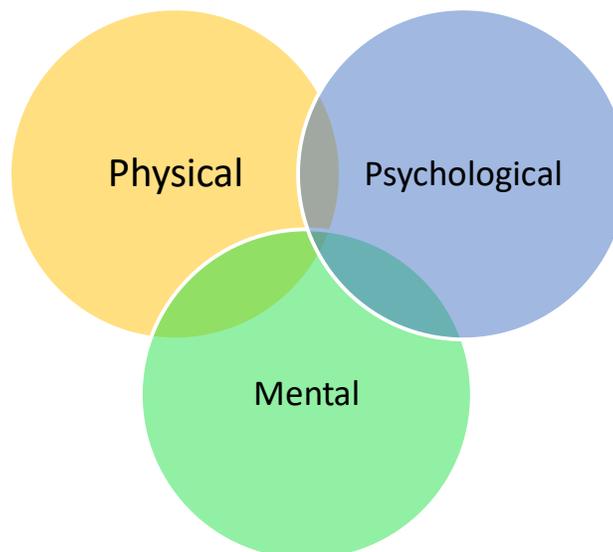


Figure 1: Classification of Fatigue

Table 1: Classification of Fatigue (modified from Bell, 2015).

Type of Fatigue	Definition	Example
Physical	Related to reduced performance in physical tasks	"I'm tired and I need rest. I'm dragging today."
Psychological	"A state of weariness related to reduced motivation, prolonged mental fatigue or boredom that occurs in situations such as chronic stress, anxiety or depression" p.1 (Lee et al., 1991)	"I just can't get motivated to do anything. Being depressed wears me out; I just don't feel like doing anything."
Mental	Related to reduced performance in cognitive tasks	"After a while, I just can't concentrate anymore. It's hard to stay focused. My mind goes blank."

Q1: How is fatigue defined? How is it classified?

Fatigue is defined as *feelings of physical and/or mental exhaustion during or after usual activities, or feelings of inadequate energy to begin activities* (Aaronson et al., 1999; Chen, 1986).

Fatigue is classified as physical, psychological or mental. Patients can experience fatigue in one or more of these domains.

13.2.2 Epidemiology

Fatigue is one of the most common symptoms following brain injury (Duclos et al., 2014b), with an estimated prevalence of 32-73% post traumatic brain injury (TBI) (Englander et al., 2010; Ponsford et al., 2012b; Silver et al., 2018) relative to 29% in the general population (Silver et al., 2018). Unfortunately, the absolute prevalence is difficult to determine due to variation in how fatigue is defined between studies (Aaronson et al., 1999), differences in data collection methods and varying time points of assessment (Silver et al., 2018). In one study, 43% of patients with TBI reported fatigue as their first symptom experienced, and a significant proportion identified fatigue as their most disabling symptom (Belmont et al., 2006a). Although fatigue is more common among women in the general population (3:2, female: male), fatigue post TBI does not appear to differ between men and women (Borgaro et al., 2004; Ziino & Ponsford, 2005).

The aspects most frequently cited as challenging by patients with post-traumatic fatigue include: "difficulty to perform a complex task without tiredness," "difficulty to hold attention during an entire activity," "difficulty not to go back to sleep during daytime" and "difficulty not to take nap during daytime" (Borgaro et al., 2004). As a result of this, not only is fatigue burdensome, it is often associated with poor outcomes (Belmont et al., 2006a; Ponsford et al., 2011). In fact, a number of studies have reported fatigue's negative effects on social, physical and cognitive functioning (Ziino & Ponsford, 2006), participation in everyday activities (Cantor et al., 2008) and role in increased work-related and other disabilities (McCrimmon & Oddy, 2006). Although this likely has a dramatic economic impact on the healthcare system, no studies to date have examined the medical costs and losses due to fatigue after brain injury.

Q2. What is the prevalence of fatigue following TBI?

32-73% of patients with TBI experience fatigue

13.2.3 Risk Factors

A variety of risk factors may increase one's susceptibility to fatigue following ABI. Some of these factors include: anxiety and depression, sleep difficulties, side effects of medication, pain, physical difficulties, cognitive difficulties, hormonal changes, diet, coping skills, environment, social roles and other medical conditions (Ouellet et al., 2015). However, these risk factors have not been well established, due to the subjective nature of fatigue, variation in research methodologies, individual patient characteristics and timing of presentation/diagnosis. To complicate things further, many of these risk factors are labile, changing throughout care. In light of this, Figure 2 summarizes the risk factors that may contribute to fatigue over time.

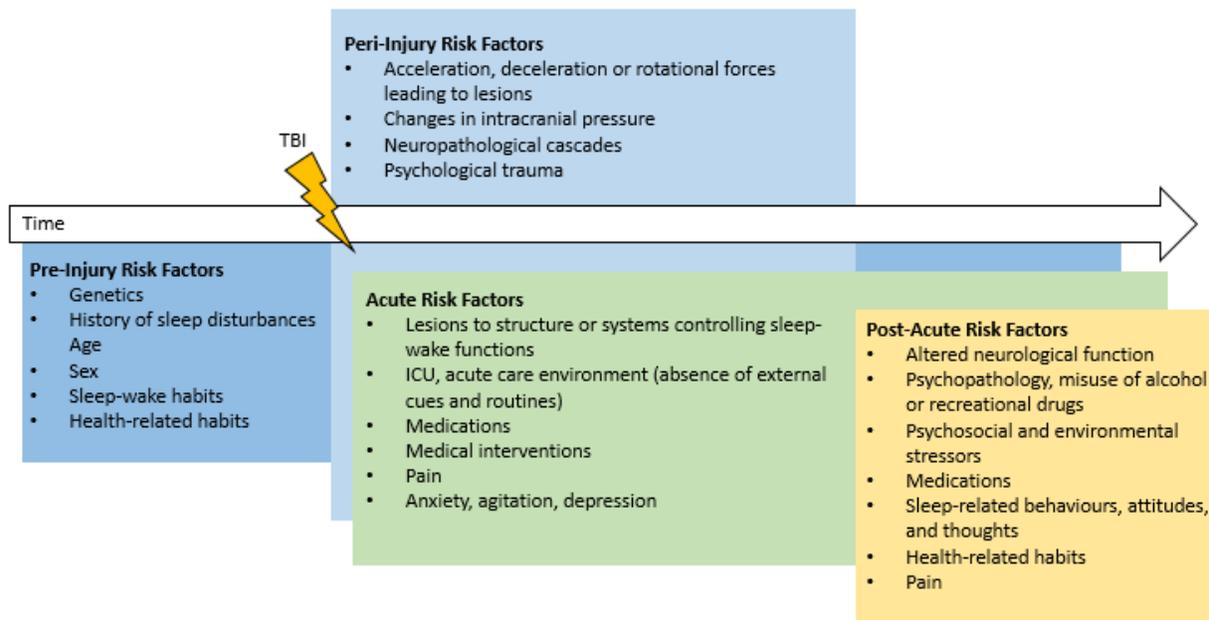


Figure 2: Risk Factors Associated with Fatigue Over Time. Modified from (Ouellet et al., 2015).

Pre-Injury Risk Factors

Risk factors prior to the injury that may contribute to fatigue post TBI include: genetics (Wang et al., 2017), history of sleep disturbances (Lichstein et al., 1997), older age (Avlund, 2010; Schwarz et al., 2003; Westerlund et al., 2010), female gender, poor sleep hygiene or health-related habits (Herman, 2018).

Peri-Injury Risk Factors

At the time of injury, several risk factors associated with the injury and experience of the event itself may contribute to fatigue. Primary injury as a result of acceleration, deceleration, or rotational forces has been found to contribute to fatigue (Schönberger et al., 2017), as well as the resulting secondary injury due to neuropathological cascades (Ouellet et al., 2015). Further, experiencing a TBI can be a traumatising event itself leading to psychological distress and fatigue (Kumar et al., 2018).

Acute Risk Factors

In the acute phase of the injury, several risk factors directly or indirectly related to the brain injury may contribute to fatigue; this includes: damage to structures or systems involved in sleep-wake function (Schönberger et al., 2017), hospital environment (Chiu et al., 2014; Duclos et al., 2014a; Ouellet et al., 2015), medications (Mollayeva et al., 2014), pain (Beaulieu-Bonneau & Ouellet, 2017; Englander et al., 2010), anxiety, agitation or depression (Beaulieu-Bonneau & Ouellet, 2017; Englander et al., 2010).

Post-Acute Risk Factors

Finally, over the long-term, a variety of psychological, environmental, and behavioural risk factors may interact to contribute to fatigue; this includes: altered neurological function (Ouellet et al., 2015; Ouellet et al., 2019), psychopathology or substance abuse (Mollayeva et al., 2014; Ouellet et al., 2019), psychosocial and environmental stressors (Bay & Xie, 2009; Bruijijel et al., 2018; Ouellet et al., 2019), medications (Mollayeva et al., 2014; Ouellet et al., 2019), sleep-related behaviours, attitudes, and thoughts (Ouellet et al., 2015; Ouellet et al., 2019), health-related habits (Ouellet et al., 2015; Ouellet et al., 2019) and pain (Ouellet et al., 2019; Sigurdardottir et al., 2013).



Clinical Tip!

Key modifiable risk factors for post-traumatic fatigue include:

- Sleep hygiene practices (see section 13.2.10)
- Exercise and physical activity (see section 13.2.10)
- Substance use or abuse (such as alcohol, nicotine, caffeine, and recreational drugs)
- Medications (see section 13.2.9)
- Untreated medical conditions (such as obstructive sleep apnea, obesity hypoventilation syndrome, hypopituitarism, hydrocephalus, anemia, depression, anxiety, agitation, PTSD, nightmare disorders)
- Untreated pain

13.2.4 Etiology

The underlying causes of fatigue following an ABI are not clear. Fatigue is likely multifactorial, arising from one or more pathophysiological processes (i.e., structural damage, endocrine or biochemical abnormalities), as well as psychosocial and non-neurological mechanisms. The following section describes the etiology of fatigue following an ABI (summarized in Figure 3).

Pathophysiological Factors

Non-pathophysiological fatigue from sleep deprivation, overwork or overexertion is easily remedied by sleep, rest or a period of recovery (DeLuca, 2005). However, following an ABI, many patients may experience feelings of fatigue that are not remedied by sufficient rest and recovery. This *pathophysiological fatigue* is often a result of a variety of different factors.

Structural Damage

In neuropathological causes of fatigue, damage may occur to either the central (CNS) or peripheral nervous system (PNS) directly (due to injury) or indirectly (due to neuropathological cascades). Injury to the CNS may result in neurological dysfunction that leads to fatigue, which is often referred to as *central fatigue* (Silver et al., 2018). Depending on which area of the brain is affected, this can lead to different types of fatigue experienced by patients with ABI. If damage occurs to areas of the CNS that are responsible for attention, concentration and memory (i.e., prefrontal cortex and hippocampus), this may lead to cognitive difficulties and *mental fatigue* (Silver et al., 2018). In contrast, if damage occurs to areas of the CNS that are responsible for hormonal regulation and production (i.e., hypothalamus or pituitary gland) this may lead to neuroendocrine disorders and *physical fatigue* (Silver et al., 2018).

Neuroendocrine Disorders are discussed in Chapter 10 of the ERABI clinical guidebooks and can be accessed [here](#).

Physical fatigue may also arise from damage to areas of the brain involved with movement and coordination (motor cortex and cerebellum). *Peripheral fatigue* may arise if the CNS is unable to adequately communicate with the PNS, or if the PNS is unable to communicate with muscle (i.e., impaired neuromuscular transmission at the motor endplate) or if muscles are unable to utilize energy required for activity, resulting in muscle deconditioning and *physical fatigue* (Silver et al., 2018).

Biochemical Alterations

Indirect injury, resulting from ABI, activates neuropathological cascades leading to alteration of biochemical and cellular pathways. In particular, levels of tyrosine and other essential amino acids have been found to decrease over time (Aquilani et al., 2003). As tyrosine and tryptophan are precursors to the neurotransmitter serotonin, they play an important role in regulating fatigue (Zasler et al., 2012). Disruption of this biochemical pathway, as a result of ABI, may lead to increased fatigue (Aquilani et al., 2003).

Sleep Disturbances

Although it would seemingly make sense to link disorders of sleep with fatigue (Clinchot et al., 1998), this relationship remains inconclusive (Fellus & Elovic, 2007). Sleep disturbances can exacerbate fatigue; however, fatigue may also manifest independent of sleep disorders (Ouellet et al., 2015).

Physiological Factors

Physiological causes of fatigue include physical deconditioning, sleep deprivation, excessive energy consumption and depletion of hormones or neurotransmitters, often resulting in *physical fatigue* (Silver et al., 2018).

Psychosocial Factors

In addition to pathophysiological factors, psychosocial stressors, comorbid psychopathologies and behavioural factors may contribute to the development of fatigue. Adjusting to new cognitive and physical limitations following ABI can be challenging and often leads to depression, anxiety, stress or reduced motivation, resulting *psychological fatigue* (DeLuca, 2005). Additionally, behavioural factors such as poor sleep hygiene, caffeine consumption or use of alcohol and drugs may also lead to fatigue (Zasler et al., 2012).

Non-Neurological Factors

Non-neurological causes of fatigue include medications, other medical conditions, as well as pain and may result in *mental, physical or psychological fatigue* (Silver et al., 2018).

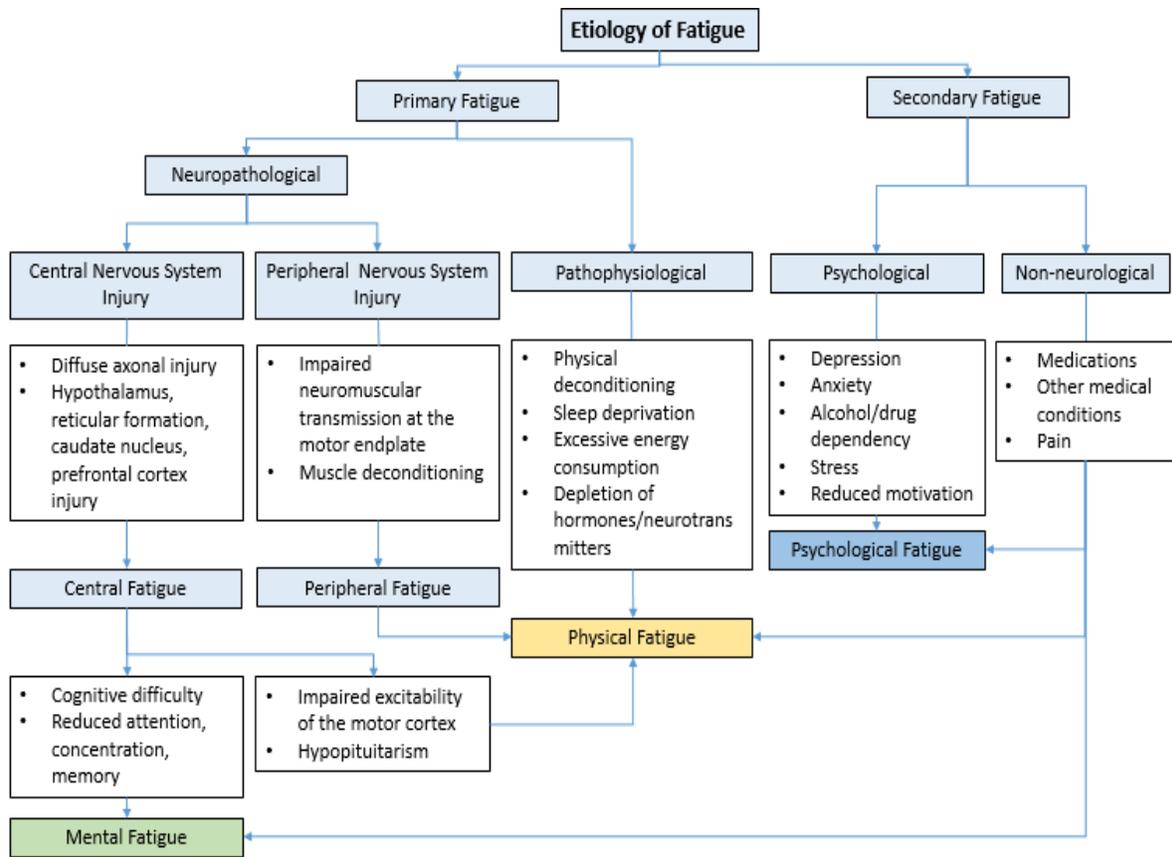


Figure 3: Etiology of Fatigue

13.2.5 Timing of Onset

Patients may experience fatigue within a few days or weeks following injury and these symptoms may persist for some time after (Hutchison et al., 2009; Ouellet & Morin, 2006). In a systematic review, Mollayeva et al. (2014) found that 17-47% of individuals with *mild TBI* report significant fatigue between two days and three months post-injury, with lower percentages reported for longer time since injury. However, research relating to timing of onset of fatigue in *moderate to severe TBI* is limited. Information from studies including all severity levels suggests that about 46% suffer from fatigue at one-year post-injury (Belmont et al., 2006b; Bushnik et al., 2008; Olver et al., 1996; Ouellet & Morin, 2006). Several studies have found a significant proportion of patients experience fatigue well beyond one year following TBI (Olver et al., 1996; Ouellet & Morin, 2006; Ziino & Ponsford, 2005), with the incidence of fatigue increasing, rather than decreasing, over time in these studies (Ponsford & Ziino, 2003; Ponsford et al., 2012a; Ziino & Ponsford, 2005). For example, in a population of patients who sustained mild to severe TBIs, an average of eight years earlier, 68% were found to be significantly fatigued (Ouellet & Morin, 2006). Furthermore, a prospective study by Ponsford et al. (2014) found that 70% of patients with mild to severe TBI reported significant fatigue 2 years post-injury, while slightly fewer reported fatigue at five years (60%) and 10 years (57%) following TBI. Although there is conflicting evidence as to whether or not fatigue increases over time for persons with moderate or severe TBI, these studies illustrate the significant proportion of patients with mild, moderate, or severe TBI who experience persistent post-traumatic fatigue.

Q3. Which patients with TBI should be assessed for fatigue? When should you evaluate fatigue in patients with TBI?

All individuals who have sustained an ABI should be assessed for fatigue and sleep disorders. The assessment for fatigue should include a focussed history, physical examination, assessment of medications, medical conditions, and sleep hygiene. Clinicians should consider structural abnormalities, affective disorders, sleep disorders, metabolic disorders, electrolyte abnormalities, neuroendocrine abnormalities and medication side effects or polypharmacy as potential treatable contributors to fatigue. Treatment should be directed towards the cause of the fatigue.

There are no specific guidelines on when you should screen for fatigue. However, evaluating patients for fatigue and its impact on function and recovery should take place at regular intervals during the acute, subacute, and chronic phases of injury, including during inpatient rehabilitation and afterwards. Clinicians should also consider fatigue or sleep disorders as part of the differential when evaluating patients for cognitive or behavioural changes, or if there are concerns from patients and/or caregivers about the level of fatigue.

13.2.6 Signs and Symptoms

Symptoms of fatigue may present at any time following brain injury and are highly variable patient to patient. Patients may experience different levels of severity and pervasiveness, with some individuals experiencing symptoms of fatigue all the time and others only upon mental or physical exertion. For example, a patient's level of fatigue may also be influenced by how much they are doing physically and cognitively, and whether they are taking time to rest periodically or pace themselves. For other patients, fatigue may be pervasive and unrelated to activity.

Identifying signs and symptoms of fatigue in patients can be challenging given the subjective nature of fatigue and its many domains. Moreover, patients may not specifically attribute their symptoms to fatigue. A careful evaluation of the signs and symptoms of fatigue in each of the physical, mental and emotional categories can be helpful.

Physical Symptoms
<ul style="list-style-type: none">• Yawning• Heavy eyelids• Eye-rubbing• Nodding off or head drooping• Headaches, nausea, or upset stomach• Slowed reaction time• Lack of energy, weakness, or light headedness

Mental Symptoms
<ul style="list-style-type: none">• Difficulty concentrating• Lapses of attention• Failure to communicate important information• Failure to anticipate events or actions• Making mistakes on well-practiced tasks• Forgetfulness• Difficulty thinking clearly• Poor decision making

Emotional Symptoms
<ul style="list-style-type: none">• More quiet or withdrawn than normal• Lack of motivation• Irritable• Low motivation• Heightened emotional sensitivity

13.2.7 Clinical Assessment

Throughout the acute, sub-acute and chronic phases following an ABI, it is important to periodically assess patients for the development of signs and/or symptoms of fatigue. This requires screening for symptoms of fatigue and arranging for diagnostic testing and appropriate treatment when warranted. However, assessment of fatigue is challenging as no measurement instruments have been developed to specifically assess fatigue following an ABI. Likewise, screening for fatigue involves subjective reporting and careful interpretation, as fatigue is multifaceted in nature.

The following sections will outline an approach to identifying and managing fatigue clinically.

The ONF-INESSS guidelines recommend that all individuals who have sustained a traumatic brain injury be assessed for fatigue and offered appropriate treatment (ONF-INESSS, 2015).

13.2.8 Screening

Several scales have been used to screen for fatigue. The most frequently used instruments for evaluating fatigue include the Fatigue Severity Scale (FSS), Fatigue Impact Scale (FIS), Visual Analogue Scale-F (VAS-F), Global Fatigue Index (GFI) and Epworth Sleepiness Scale (ESS) (summarized in Table 2). None of these scales were not specifically developed for those with ABI, instead they were developed for patients with other chronic illnesses such as Human Immunodeficiency Virus or Multiple Sclerosis (Armutlu et al., 2007; Fish et al., 2007). Of these, the FSS has been validated in patients with ABI (Dittner et al., 2004; Ziino & Ponsford, 2005). All of the screening tools have limitations; they are neither diagnostic nor a substitute for a comprehensive clinical assessment.

Table 2: Common scales used for clinical screening of fatigue

Scale Author, Year	Initial Population	Specified Fatigue Subscales	Time Frame	Purpose	Score Interpretation
Fatigue Severity Scale (Krupp et al., 1989)	MS, lupus, healthy	None	Not stated	Measures severity of fatigue, impact on activities and lifestyle.	Score range: 1-7 Higher score=more severe fatigue
Modified Fatigue Impact Scale (Fisk et al., 1994)	MS	Physical, cognitive, psychosocial	Past 4 weeks	Measures impact of fatigue on life function.	Score range: 0-84 Higher score=more severe fatigue
Visual Analog Scale – Fatigue (Lee et al., 1991)	Sleep disordered and healthy	Energy, fatigue	Not stated	Measures severity of fatigue.	0-4 No fatigue 5-44 Mild 45-74 Moderate 75-100 Severe
Global Fatigue Index (Belza, 1995)	Rheumatoid Arthritis	Degree, severity, distress, impact on ADLs, timing	Not stated	Measures severity and impact of physical and mental fatigue.	Score range: 1-50 Higher score=more severe fatigue
Epworth Sleepiness Scale (Johns, 1991)	Sleep disorders, healthy controls	Daytime sleepiness	Not stated	Measures daytime sleepiness	0-10 Normal 11-14 Mild Sleepiness 15-17 Moderate 18-24 Severe

The ONF-INESSS guidelines recommend the use of questionnaires for assessment of fatigue following TBI (ONF-INESSS, 2015).

13.2.9 Diagnosis

Fatigue resulting from an ABI is diagnosed on the basis of exclusion of reversible or treatable causes of fatigue. As such, a systematic approach guided by a thorough clinical history should be used to rule out other causes of fatigue. The clinical history should involve characterization the patient’s symptoms of fatigue (e.g., onset, duration, impact of physical activity, cognitive activity and rest on symptoms), relationship to medications or substance use, mood or anxiety symptoms, and neuroendocrine or metabolic symptoms. If improvement in fatigue is observed with a change in activity or environment, it may be related to apathy or boredom. Similarly, if fatigue improves with rest, it may be related to a lack of sleep. In addition, risk factors for fatigue (as discussed in section 13.2.3) should be evaluated. It is essential to conduct a thorough review of medications, as many commonly used in the rehabilitation of ABI may contribute to fatigue (see Table 3). Assessment of fatigue should also involve a comprehensive assessment of sleep by history, including collateral history from a bed partner or caregiver if available. This should include sleep history, review of sleep hygiene practices, and sleep diaries which may form the basis for clinical decision-making regarding the need for formalized sleep disorder diagnostic testing such as nocturnal polysomnography (PSG) or actigraphy.

Table 3: Common Medications Associated with Fatigue
(adapted from Zasler et al., 2012).

Medications Frequently Associated with Fatigue	Medications Commonly Used in TBI Rehabilitation
Antiepileptics	Levetiracetam Carbamazepine Valproate
Antihistamines	
Antipsychotics, typical and atypical	Olanzapine Quetiapine Clozapine
Corticosteroids	
Antiarrhythmics	Propranolol Verapamil
Antidepressants	SSRIs Trazadone Tricyclic antidepressants
Antiemetics	Phenergan Zofran

Findings from the clinical history should guide the physical exam to rule out specific causes of fatigue. A physical examination should be performed to assess for new or changing neurologic features, such as those suggestive of hydrocephalus, and for signs of endocrine or metabolic abnormalities. Signs and symptoms of cardiopulmonary disease, autoimmune disease and malignancy should be considered and

Table 4: Differential Diagnosis of Fatigue
(adapted from Zasler et al., 2012).

Etiology	Possible Conditions
Cardiopulmonary	Heart failure Acute myocardial ischemia Atrial fibrillation COPD Obstructive sleep apnea
Metabolic	Anemia Iron deficiency without anemia Vitamin D deficiency
Endocrine	Diabetes mellitus Hypothyroidism Hyperthyroidism Hypopituitarism Addison disease
Neurologic	Stroke Restless leg syndrome Multiple sclerosis Parkinson disease
Psychiatric	Depression Alcohol dependence Drug dependency
Infectious	HIV Tuberculosis Toxoplasmosis Cytomegalovirus Lyme disease Brucellosis Epstein Barr Virus
Gastrointestinal	Celiac disease Primary biliary cirrhosis
Neoplastic	Myelodysplastic syndrome Chronic myeloid leukemia Non-Hodgkin lymphoma Hodgkin lymphoma Other malignancy
Other	Primary insomnia Medication-induced fatigue Chronic renal disease Fibromyalgia Systemic lupus erythematosus Chronic fatigue syndrome

investigated further (common causes of fatigue and recommended items for evaluation are listed in Table 4).

Initial investigations should be determined based on the history and physical examination. Where appropriate, laboratory tests may include complete blood count (CBC), electrolytes, fasting blood glucose or HbA1c, C-reactive protein (CRP), pregnancy test, a workup for neuroendocrine causes of fatigue (see chapter 10 of the ERABI clinical guidebooks), or blood levels of anti-seizure medications. For patients with new or worsening neurological abnormalities on examination, consideration should be made for neuroimaging investigations such as CT or MRI.

13.2.10 Management

The management of fatigue is complicated by its multifaceted nature and as of yet a definitive treatment does not exist. Initial management should begin with addressing comorbidities or contributing causes identified on history, physical examination, and investigations. If psychological distress is the underlying cause of fatigue, referral to psychiatric care or counselling and/or antidepressants may be necessary. Additionally, any underlying medical illnesses, endocrine abnormalities or sleep disorders should be addressed. Following the management of comorbid conditions, non-pharmacological interventions such as behavioural modifications and psychoeducation should be considered next. Once conservative measures fail and modifiable factors have been addressed, pharmacological management should be considered.

The following section will provide an overview of the non-pharmacological and pharmacological interventions available for the management of fatigue following an ABI. An overview of these interventions are summarized in Table 5.

Non-Pharmacological Interventions

Behavioural Interventions

CBT

The goal of CBT is to modify behavioural factors that perpetuate fatigue. Ngyuen et al. (2017) divides CBT for fatigue into six modules that focus on psychoeducation, daily schedule organization, cognitive restructuring, sleep interventions, strategies for physical and mental fatigue, as well as relapse prevention (summarized in Table 6). However, only one study has evaluated the efficacy of this method in a population with moderate-to-severe TBI. In a small randomized controlled trial, Ngyuen et al. (2017) found that CBT significantly improves daily fatigue levels in patients with moderate-to-severe TBI and improvements were maintained for a period of two months following therapy. Although promising, further research is necessary to determine the efficacy of CBT for managing fatigue following ABI. The ONF-INESSS guidelines do not make any recommendations about CBT for fatigue.

Table 5: Summary of Interventions for Fatigue Following ABI

Non-pharmacological Interventions	Pharmacological Interventions
<ul style="list-style-type: none"> • CBT • Psycho-education • Pacing • Sleep hygiene • Mindfulness therapy • Lifestyle management • Exercise 	<ul style="list-style-type: none"> • Methylphenidate • Modafinil

Table 6: Components of CBT for Fatigue Following TBI (Nguyen et al., 2017)

Module	Content	Aims
1	Psychoeducation CBT framework	Shift unhelpful beliefs about symptoms. Encourage understanding of link between sleep habits and activity levels with symptom maintenance.
2	Reorganize daily schedules	Initiate pacing of tasks, incorporating optimal number of rest breaks, improving sleep hygiene practices.
3	Graded activity Cognitive restructuring	Integrate meaningful activity depending on goals and available energy. Modify appraisals on rest and sleep practices.
4	Sleep interventions	Behavioural interventions (stimulus control, bedtime restriction), relaxation techniques.
5	Strategies for physical and mental fatigue	Environmental modification, restructuring tasks. Cognitive strategies for information processing and time pressure management.
6	Review techniques Relapse prevention	Consolidate treatment gains.

Psychoeducation

The goal of psychoeducation is to educate patients and caregivers about the sequelae of ABIs, such as a reduced ability to perform tasks that may result in fatigue. Accordingly, patients may learn compensatory techniques to adjust to their new cognitive and physical limitations. This may involve a variety of interventions such as behavioural modifications, pacing, sleep hygiene or changes to lifestyle management strategies. The ONF-INESSS guidelines do not make any recommendations about TBI education.

Pacing

Those who are suffering from fatigue may benefit by performing important activities when they feel they are at their best (Lezak, 1978). Energy conservation and pacing are two ways an individual is encouraged to overcome or deal with his or her levels of fatigue following brain injury (Fellus & Elovic, 2007). Many patients find that simple tasks require more concentration and effort than they did previously and, as a result, they tire more easily (Lezak, 1978). As part of their rehabilitation, individuals may be taught or re-

taught how to prioritize their commitments and are encouraged to recognize their abilities and limitations (Fellus & Elovic, 2007). For some this may come easily, but for others it may require more education or other interventional programs (2007). Although pacing is a concept that has been accepted by health care professionals and encouraged within the ABI population, its benefits have not yet been studied with this group and, as a result, the treatment effects of pacing strategies are not known.

The ONF-INESSS guidelines recommend that energy conservation strategies should be considered in the treatment of fatigue for individuals with traumatic brain injury (ONF-INESSS, 2015).

Sleep Hygiene

Sleep hygiene involves education about behavioural patterns and environmental factors that disrupt sleep. Sleep support strategies should be provided, including information about avoiding caffeine, screen time before bed and maintaining a daily schedule. A full list of suggested strategies may be found in the guidelines for concussion/mild traumatic brain injury [HERE](#).

The ONF-INESSS guidelines recommend that sleep hygiene practices should be considered in the treatment of fatigue for individuals with traumatic brain injury (ONF-INESSS, 2015).



Clinical Tip!

Key elements of sleep hygiene include:

- Going to bed and getting up at the same time every day, including weekends
- Avoiding naps, or taking naps for no longer than 20 minutes if necessary
- Avoiding caffeine, alcohol and nicotine
- Regular exercise Avoiding heavy exercise and heavy meals for several hours before bedtime
- Keeping bedroom quiet, dark, and at a comfortable temperature
- Keeping stress and work out of the bedroom
- No TV or computer while in bed
- If unable to sleep, don't lie in bed; get up and do a relaxing activity for a short while

Mindfulness-Based Therapy

Mindfulness-based cognitive therapy combines cognitive behavioural therapy methods with mindfulness meditation practices such as yoga, body scan and sitting meditation or mindfulness classes. It is thought to reduce fatigue by lessening the impact of maladaptive coping strategies, behavioural, emotional or cognitive patterns. Few studies have evaluated the efficacy of mindfulness-based therapy on fatigue following ABI. In a randomized controlled trial of 11 participants with TBI, Johansson et al. (2012) found significant improvements in subjective measures of mental fatigue (mental fatigue scale). Similarly, in a follow-up study using a live, interactive, online mindfulness-based therapy program, Johansson et al. (2015a) found significant improvements in subjective measures of mental fatigue (mental fatigue scale). However, further research is necessary to determine the efficacy of mindfulness-based therapy on fatigue in a population with ABI. The ONF-INESSS guidelines do not make any recommendations about mindfulness-based therapy for fatigue.

Lifestyle Interventions

Lifestyle Management Strategies

Lifestyle changes can include anything from diet modifications to self-care to exercise. Although this approach intuitively makes sense, there are challenges when attempting to compare studies as the breadth of interventions and outcomes is significantly larger than in most areas of research.

In a small (n=8) pre-post study by Stubberud et al. (2017), participants underwent 36 hours of programming focusing on lifestyle factors, adaptive coaching, and goal management training. The intervention significantly reduced fatigue at posttest and 3-month follow-up, but not at 9-month follow-up. Sleepiness was significantly reduced at posttest, 3-month follow-up, and 9-month follow-up. The authors also reported a significant improvement on anxiety scores on the Hospital Anxiety and Depression Scale. The ONF-INESSS guidelines do not make any recommendations about lifestyle management strategies for fatigue.

Exercise

Exercise may improve fatigue and has significant benefits for cardiovascular health, general well-being, emotional and immune system functioning. In a randomized controlled trial, Kolakowsky-Hayner et al. (2017) found that exercise (a community walking program) positively improved fatigue in a population with TBI. Importantly, this effect was maintained over an 8-month follow-up period.

The ONF-INESSS guidelines recommend that exercise should be considered in the treatment of fatigue for individuals with traumatic brain injury (ONF-INESSS, 2015).

Complementary and Alternative Medicine

Light Therapy

The goal of light therapy is to shift waking or bedtimes towards a more desirable sleep-wake schedule. Typically, light therapy involves a person being exposed to a short wavelength light (430-475 nm; blue wavelength light) upon awakening. The theoretical basis for light therapy is using light to alter melatonin production and secretion. Photosensitive retinal ganglion cells respond to blue light and transmit signals to hypothalamic nuclei to suppress the production of melatonin, leading to increased daytime alertness and earlier onset of evening sleep (Bajaj et al., 2017). However, light therapy has not been well studied in a population with ABI. Sinclair et al. (2014) conducted a RCT examining the effectiveness of light therapy, comparing blue and yellow light therapy to a control group. Only the blue light therapy demonstrated significantly decreased fatigue ($p < 0.001$) and daytime sleepiness ($p < 0.01$) compared to the control group but these improvements did not persist at follow-up.

The ONF-INESSS guidelines recommend that light therapy should be considered in the treatment of fatigue for individuals with traumatic brain injury (ONF-INESSS, 2015).

Pharmacological Interventions

Stimulant medications, including methylphenidate and modafinil, have been used in the pharmacological management of fatigue (see Table 15 in pharmacological management of sleep disorders).

Methylphenidate

Methylphenidate is a neurostimulant commonly used to treat narcolepsy and attention deficit hyperactive disorder in children (Weber & Lutschg, 2002). Methylphenidate increases dopamine and norepinephrine within the brain. A case control study by Lee et al. (2005) reported that methylphenidate may be effective in reducing excessive daytime sleepiness in patients with mild to moderate TBI. In a

randomized controlled trial, a population with moderate TBI experienced significant improvements in mental fatigue (assessed with the mental fatigue scale) with methylphenidate in a dose-dependent manner (Johansson et al., 2015b).

The ONF-INESSS guidelines recommend considering short-term treatment with methylphenidate to reduce excess daytime sleepiness in individuals with traumatic brain injury (ONF-INESSS, 2015).

Modafinil

Modafinil, a wakefulness promoting agent, has been approved to address excessive daytime sleepiness (EDS) (Jha et al., 2008). The precise mechanism of action of modafinil is unknown. Modafinil is approved for use in narcolepsy and sleeping difficulties associated with shift work (US Modafinil in Narcolepsy Multicenter Study Group, 1998, 2000). Studies exploring the use of modafinil for fatigue and excessive daytime sleepiness in patients with other conditions, such as TBI, Parkinson's disease, multiple sclerosis, and post-polio syndrome, provide inconsistent results (Sheng et al., 2013).

The ONF-INESSS guidelines do not make any recommendations about the use of modafinil for the treatment of fatigue following TBI.

13.3 Sleep Disorders

Sleep disorders are one of the most common and persistent sequelae of ABI. Patients in both the acute and chronic phases of rehabilitation, regardless of injury severity, may present with insomnia, excessive daytime sleepiness, an increased need for sleep, or sleep fragmentation (Ouellet et al., 2015). Numerous studies have identified that patients with ABI may underestimate their sleep disturbance and/or its impact on their day-to-day function (Imbach et al., 2016b; Imbach et al., 2015). Therefore, understanding these sleep disorders to effectively identify and treat patients with ABI is important and can complement other efforts to promote maximal rehabilitation and functional recovery.

In this section, a review of the clinical features, diagnosis and management of the most common sleep disorders following ABI are discussed.

13.3.1 Neurobiological Basis of Sleep

Sleep is a complex and vital process that is regulated by a variety of neurochemicals and brain regions. Particular regions within the brain that have been implicated in regulating sleep-wake function include the brain stem, basal forebrain and hypothalamus (American Psychiatric Association, 2013). Additionally, a variety of hormones (including melatonin), endogenous products and neurotransmitters (such as serotonin and acetylcholine) have been found to play an important role in regulating sleep (American Psychiatric Association, 2013). A full review of the neurological basis of sleep and sleep cycles is beyond the scope of this guidebook. For further information, please refer to standard textbooks on this subject (Ward, 2001).

13.3.2 Epidemiology

Sleep disorders are estimated to occur in 30-70% of individuals following an ABI and can substantially impact a patient's physical, cognitive and social abilities, ability to participate in rehabilitation programs, and activities of daily living. There are many different sleep disorders experienced by patients with ABI including insomnia, sleep apnea, hypersomnia, parasomnias, narcolepsy or circadian rhythm disorders (Ouellet et al., 2019).

Table 7: Prevalence of Common Sleep Disorders Following TBI (Ouellet et al., 2015; Rao et al., 2015)

Sleep Disorder	Prevalence General Population	Prevalence TBI
Insomnia	6%	30-70%
Sleep apnea	2-15%	23-36%
Post-traumatic hypersomnia	N/A	20-28%
Parasomnias	4-67%	25%
Restless legs syndrome	2-7%	13-17%
Narcolepsy	0.02 -0.04%	3-5%
Circadian rhythm sleep-wake disorders	Unknown	Unknown

Understanding how prevalent sleep disorders are post brain injury is challenging for a number of reasons. The actual prevalence is difficult to ascertain as many milder injuries may go unreported. In addition, the vast majority of patients who have experienced an ABI are never investigated for sleep disorders. Nevertheless, several studies provide some insight and in most cases, individuals with TBI tend to experience greater levels of sleeping disorders than the general population (See Table 7).

The rest of this chapter will provide an overview of the most common sleep disorders following an ABI; however, a full description of all sleep disorders is beyond the scope of this guidebook. For further information, please refer to the [International Classification of Sleep Disorders](#).

Q4. What are the three most common sleep disorders following an ABI?

1. Insomnia
2. Sleep apnea
3. Hypersomnia

13.3.3 Risk Factors

Many of the risk factors reported for fatigue in section 13.2.3 are also applicable to sleep disorders. Risk factors for sleep disorders often evolve over time depending on a variety of factors (see Figure 2 in fatigue risk factor section). Although risk factors exist for sleep disorders in the general population (See Table 8), little information is available specifically for persons with ABI. In one study, Hou and colleagues found that severe TBI (as measured with the GCS), less education and the presence of residual symptoms (i.e., headache or dizziness) were significantly associated with an increased risk of developing sleep disorders (Hou et al., 2013). However, loss of consciousness and cause of injury were not associated with an increased risk of sleep disorders (Hou et al., 2013).

Table 8: Common Risk Factors of Sleep Disorders in the General Population

Sleep Disorder	Risk Factors In General Population
Insomnia (LeBlanc et al., 2009)	<ul style="list-style-type: none"> Advanced age Chronic disease Medications Sex – women are at increased risk Psychological factors Lifestyle behaviours
Sleep-related breathing disorders (Burman, 2017)	<ul style="list-style-type: none"> Excess weight Narrowed airway Hypertension Chronic nasal congestion Smoking Diabetes Sex – men are at increased risk Family history Asthma
Narcolepsy (Scammell, 2019)	<ul style="list-style-type: none"> Age (10-30yr) Family history
Circadian rhythm disorders (Zhu & Zee, 2012)	<ul style="list-style-type: none"> Advanced age Family history Lifestyle habits Environment or occupation (shift work) Other medical conditions Sex – men are at an increased risk

Q5. What are the risk factors for sleep disorders?

- Injury severity (greater severity has higher risk)
- Level of education
- Residual symptoms

13.3.4 Etiology

The underlying causes of sleep disorders in individuals with an ABI are not well known. However, it likely involves a complex interaction between pathophysiological processes (i.e., structural damage or neurochemical alterations), psychological, environmental (i.e., noises or lights) and social factors. In light of this, the following section outlines the pathophysiological, environmental and psychosocial factors that may contribute to the development of sleep disorders following an ABI.

The ONF-INESSS guidelines recommend that clinicians should consider the possibility of sleep disorders related to traumatic brain injury as a cause of cognitive and other behavioural changes (ONF-INESSS, 2015).

Pathophysiological Factors

Structural Damage

Primary or secondary damage to areas of the brain that regulate the sleep-wake cycle (e.g., suprachiasmatic nuclei of the hypothalamus, reticular activating system, pontine nuclei) may cause sleep disorders (Gabor et al., 2001; Mahowald, 2000; Makley et al., 2009; Zasler et al., 2012). The type of sleep disorder that arises depends on which sleep-regulating region on the brain is injured (Viola-Saltzman & Watson, 2012). Posttraumatic hypersomnia may arise from damage to areas of the brain involved in the

maintenance of wakefulness, such as the brainstem reticular formation, posterior hypothalamus and the area surrounding the third ventricle (Viola-Saltzman & Watson, 2012). Excessive daytime sleepiness may be aggravated by damage to the ascending reticular activating system (Ouellet et al., 2019). Hypersomnia may also be exacerbated by lesions to high cervical cord regions, which are known to precipitate sleep disordered breathing (particularly obstructive sleep apnea) (Guilleminault et al., 2000; Leduc et al., 2007; Viola-Saltzman & Watson, 2012). Sleep apnea may also arise from injuries to other parts of the body, as is the case with orofacial fractures that result in obstructive sleep apnea (Zasler et al., 2012). Insomnia is likely a result of damage to the inferior frontal and anterior temporal regions, which includes the basal forebrain (an area involved in sleep initiation). These regions are often affected by coup-contrecoup brain injuries that result in damage to the base of the skull in areas of bony irregularities (especially the sphenoid ridges) (Viola-Saltzman & Watson, 2012). Circadian rhythm disorders may arise from damage to the suprachiasmatic nucleus and/or its output tracts.

Neurochemical Alterations

In addition to structural damage, ABI may result in alterations to neurochemicals that are involved in sleep regulation, resulting in sleep disorders (Cronin & O’Loughlin, 2018). The most common of these are orexin and melatonin, described below.

Orexin

Orexin, which is also referred to as hypocretin, is a hypothalamic neuropeptide that regulates wakefulness through excitation of several downstream monoaminergic and cholinergic wake-promoting systems (Lim & Baumann, 2019). Low levels of orexin are found in most cases of narcolepsy with cataplexy (Dauvilliers et al., 2003). Similar low levels of orexin have been found in patients with moderate-to-severe TBI, which may account for the prominence of excessive daytime sleepiness and sleep fragmentation observed within this population (Baumann et al., 2005; Baumann et al., 2009). However, alterations in orexin signalling cannot account for all sleep abnormalities, as levels often return to baseline in the chronic phases of injury (Baumann et al., 2007).

Melatonin

Melatonin is an important hormone that regulates sleep-wake cycles. It is primarily produced in the pineal gland and is released into the bloodstream following a circadian pattern, rising in the evening and peaking overnight (Lim & Baumann, 2019). Importantly, several studies have found that melatonin secretion and circadian regulation are dysregulated following a brain injury, in both the acute and chronic phases (Grima et al., 2016; Naseem & Parvez, 2014; Seifman et al., 2014; Shekleton et al., 2010). Seifman and colleagues (2014) found significantly lower levels of serum melatonin in patients with acute TBI when compared to healthy controls. Likewise, in another study of 23 patients with chronic TBI (>6 months), melatonin production was decreased when compared with age and sex matched controls (Shekleton et al., 2010). Several studies have demonstrated that melatonin supplementation can be helpful for patients with a history of moderate to severe ABI and insomnia (Grima et al., 2018; Shekleton et al., 2010). For further information, please refer to the Neuroendocrine Guidebook chapter.

The ONF-INESSS guidelines recommend considering the use of melatonin 2-5 mg for insomnia after traumatic brain injury (ONF-INESSS, 2015).

Environmental and Psychosocial Factors

Throughout recovery, environmental factors may play a significant role in the development of sleep disorders. Circadian rhythms are often disrupted by a lack of external environmental cues that synchronize biological rhythms with 24-hour light-dark cycles (also known as zeitgebers) (Zasler et al., 2012). In the acute phase, this may result from frequent medication administration, pain or anxiety related to hospitalization, noise, lack of access to windows or natural light, lack of routine and bright lights (Frieze et al., 2007; Zasler et al., 2012). However, environmental disruptions beyond the acute care setting may also interfere with sleep-wake patterns. For example, when reintegrating into the community it may be difficult to adjust to a new living or sleeping environment or regular sleep-wake routines (e.g., fixed bedtime or regular meal times) (Zasler et al., 2012).

In addition to environmental factors, psychosocial stressors, comorbid psychopathologies and behavioural factors may contribute to the development of sleep disorders. Following ABI, individuals must adjust to new cognitive and physical limitations in order to reintegrate into their social and familial roles. Unsurprisingly, this is often a significant source of stress, leading to anxiety and difficulties trying to fall asleep or awaken (Morin, 1993). To further complicate the matter, insomnia, hypersomnia and excessive daytime sleepiness may be exacerbated by comorbid mood and anxiety disorders, such as depression and generalized anxiety disorder (Ashman et al., 2004; Fann et al., 2002). Substance abuse, major depression and dysthymia may also exacerbate excessive daytime sleepiness or hypersomnia (Krahn, 2005). Behavioural factors such as poor sleep hygiene, caffeine consumption or use of alcohol or drugs may also affect sleep-wake patterns leading to disordered sleep (Zasler et al., 2012).

13.3.5 Timing of Onset

Sleep disturbances may arise in the acute, sub-acute or chronic phases following an ABI and can often persist for years following injury (Ouellet et al., 2015). Although there is limited research in the acute phase following an ABI, sleep-wake patterns are often disrupted within the first few days following TBI (Ouellet et al., 2019). In the acute inpatient rehabilitation setting, 84% of patients with moderate-to-severe TBI experience sleep-wake disturbances at admission, while 66% continue to have disturbances one month following injury (Nakase-Richardson et al., 2013; Silver et al., 2018). In particular, patients in the intensive care unit (ICU) suffer from frequent awakenings, prolonged sleep-onset latency and significant decreases in deep, slow-wave and rapid eye movement sleep (REM) (Frieze et al., 2007; Gabor et al., 2001). Within the first few days, patients are also likely to experience hypersomnolence (Billiard & Podesta, 2013; Chiu et al., 2013; Sommerauer et al., 2013). Although environmental factors play a role in sleep disturbances in the acute setting, the hospital environment is not the sole cause (Duclos et al., 2016). Many of these disturbances likely arise from the brain injury and its sequelae.

Unfortunately, sleep disturbances often persist well beyond the acute time frame, leading to sleep disorders that have been reported by patients in the chronic phase, including up to eight years following injury (Ouellet et al., 2006; Silver et al., 2018). A meta-analysis of 21 studies found that 50% of patients in the chronic phase of TBI experience sleep disturbances and 25-29% were diagnosed with a sleep disorder (Mathias & Alvaro, 2012). Furthermore, a large cohort of outpatients undergoing rehabilitation with and without TBI were objectively evaluated for sleep disturbances using actigraphy in the chronic phase of recovery. Patients with TBI were found to have higher levels of daytime sleepiness and delayed sleep onset (Imbach et al., 2016a; Silver et al., 2018).

Sleep disorders are prevalent throughout a patient’s injury trajectory and have the potential to undermine recovery, rehabilitation and outcomes. Consequently, it is important to routinely screen for sleep disturbances in order to assess the impact on recovery and outcome, as well as treatment needs.

13.3.6 Signs and Symptoms

The main features and most common symptoms of sleep disorders post ABI are presented in Table 9.

Table 9: Common Sleep Disorders and Associated Symptoms (Zasler et al., 2012)

Insomnia	
<i>Main Feature</i>	<ul style="list-style-type: none"> Dissatisfaction with the quality or quantity of sleep
<i>Common Symptoms</i>	
Subjective complaints of difficulty initiating sleep, maintaining sleep (frequent awakenings or difficulty returning to sleep), early morning awakenings (with insufficient sleep duration) and/or nonrestorative sleep	
Sleep-related breathing disorders	
<i>Main Feature</i>	<ul style="list-style-type: none"> Altered respiration during sleep
<i>Common Symptoms</i>	
Daytime sleepiness, snoring which may be interrupted by apneas (observed gasping during sleep), slow or absent breathing, frequent awakenings to restart breathing, restless and nonrestorative sleep, and/or morning headaches	
Narcolepsy	
<i>Main Feature</i>	<ul style="list-style-type: none"> Rare disorder characterized by recurrent unplanned daytime sleep episodes Tetrad of classic symptoms: daytime sleepiness, cataplexy (i.e., episodic loss of muscle function), hypnagogic hallucinations (i.e., dream-like experiences while falling asleep, dozing or awakening) and sleep paralysis (i.e., transitory, inability to talk or move upon awakening).
<i>Common Symptoms</i>	
Post-traumatic hypersomnia	
<i>Main Feature</i>	<ul style="list-style-type: none"> Hypersomnia because of medical condition (TBI) when other primary sleep disorders have been ruled out
<i>Common Symptoms</i>	
Excessive daytime sleepiness, increased sleep duration	
Circadian rhythm sleep disorders	
<i>Main Feature</i>	<ul style="list-style-type: none"> Mismatch between one’s sleep-wake rhythm and the 24-hour environment. In addition to sleep-wake cycle disturbances, melatonin secretion and body temperature rhythms can be disrupted.
<i>Common Symptoms</i>	
Sleep disturbances when trying to conform with conventional times (inability to fall asleep or remain asleep); normal sleep quality and duration when choosing the preferred schedule	

13.3.7 Clinical Assessment

Given the prevalence and impact of sleep disorders on quality of life, mental and physical health, individuals presenting with sleep complaints following an ABI should be systematically assessed. Clinical assessment should include a combination of both subjective and objective tools. Subjective tools include a clinical history including sleep history, medications, self-report questionnaires and sleep diaries completed by the patient. Objective tools used include actigraphy, polysomnography, multiple sleep latency test and maintenance of wakefulness test.

The following sections will outline an approach to identifying and managing sleep disorders.

The ONF-INESSS guidelines recommend that all individuals who have sustained a traumatic brain injury be assessed for sleep disorders and offered appropriate treatment (ONF-INESSS, 2015).

13.3.8 Screening

A variety of self-reported questionnaires exist to evaluate various aspects of sleep disorders following ABI (Table 10). Scores obtained from these questionnaires are not a substitute for assessment and do not provide a diagnosis; however, they can identify persons at risk of sleep disorders and/or track the effects of interventions.

Table 10: Common Self-Reported Questionnaires for the Assessment of Sleep Disorders

Item	Questionnaire	Author, Year	Purpose	Score Interpretation
Sleep quality	Pittsburgh Sleep Quality Index	(Buysse et al., 1989)	Measures quality and patterns of sleep within seven domains	>5 Poor sleep quality
	Insomnia severity index	(Bastien et al., 2001)	Screening for insomnia	0-7 No insomnia 8-14 Mild 15-21 Moderate 22-28 Severe
Circadian preferences	Morningness-eveningness questionnaire	(Horne & Östberg, 1976)	Preference for morning or evening tendencies	>59 Morning type 42-58 Intermediate <41 Evening type
	Sleep timing questionnaire	(Monk et al., 2003)	Assessment of habitual sleep timing	No score.
Daytime sleepiness	Epworth Sleepiness scale	(Johns, 1991)	Measures general level of daytime sleepiness	≥10 excessive daytime sleepiness
	Stanford Sleepiness Scale	(Shahid et al., 2011)	Alternative to Epworth, measures daytime sleepiness	≥4 excessive daytime sleepiness
Sleep apnea	STOP-BANG	(Chung et al., 2016)	Screening for obstructive sleep apnea (S noring, T ired, O bserved apneas, high blood P ressure, B ody mass index, A ge, N eck size, male G ender)	0-2 Low risk 3-4 Intermediate risk 5-8 High risk
	Berlin Questionnaire	(Netzer et al., 1999)	Screening for obstructive sleep apnea	≥2 positive categories indicates high likelihood of sleep disordered breathing

13.3.9 Diagnostic Testing

The choice of diagnostic method for sleep disorders depends on a variety of factors, including accessibility, the suspected diagnosis, patient characteristics, and the importance of evaluating sleep in a patient's typical environment (Silver et al., 2018). Depending on these characteristics, a wide variety of diagnostic tools are available for use such as polysomnography, actigraphy and multiple sleep latency tests (Silver et al., 2018). Guidance on the indication and use of these tests is available but is not specific to ABI (Silver et al., 2018). A summary of diagnostic tests used for the analysis of sleep disturbances following ABI is reported in Table 11.

The ONF-INESSS guidelines recommend common objective assessments for sleep disorders including: polysomnography, actigraphy, multiple sleep latency tests and maintenance of wakefulness tests (ONF-INESSS, 2015).

Table 11: Summary of Tools for the Assessment of Sleep Disorders following ABI

Assessment Method	Description
Sleep Diary	<ul style="list-style-type: none"> • Self-monitor nature, severity, and frequency of sleep difficulties • Several days of monitoring is necessary to gather clinically relevant information
Self-Reported Questionnaires	<ul style="list-style-type: none"> • Assess nature and severity of symptoms • <i>Insomnia</i>: Insomnia Severity Index or Pittsburgh Sleep Quality Index • <i>Excessive daytime sleepiness</i>: Epworth Sleepiness Scale • <i>Circadian rhythms</i>: Morning-Eveningness Questionnaire or Sleep Timing Questionnaire • <i>Sleep apnea</i>: STOP-Bang, Berlin Questionnaire
Polysomnography	<ul style="list-style-type: none"> • Gold standard to diagnose obstructive sleep apnea, limb movement disorders and narcolepsy • Combination of EEG, electro-oculography (measures eye movements) and electromyography (measures limb movements)
Multiple Sleep Latency Test And Maintenance of Wakefulness Test	<ul style="list-style-type: none"> • Objective measures of daytime sleepiness performed in a specialized setting
Actigraphy	<ul style="list-style-type: none"> • Wristwatch-like device worn over extended period of time to measure motor activity, heart rate, and sleep-wake schedule

Sleep Diaries

A sleep diary can be a helpful tool to identify factors that may affect sleep-wake patterns and can assist in monitoring any changes in sleep before or after treatment (Zasler et al., 2012). Information obtained from the sleep diary may help guide treatment recommendations (i.e., changes in sleep hygiene or sleep-wake schedules) (Zasler et al., 2012). It is recommended that patients record at least two consecutive weeks of sleep diary entries to obtain a good overview of sleep patterns (Aaronson et al., 1999). Although sleep diaries are helpful in diagnosing sleep disorders, studies comparing diaries with actigraphy have found some reporting bias (Nakase-Richardson et al., 2016b). In this sense, actigraphy may be a useful adjunct to sleep diaries.

Polysomnography

Polysomnography (PSG) is considered the gold standard to diagnose obstructive sleep apnea, periodic limb movement disorders and narcolepsy (Ouellet et al., 2015). PSG provides information on sleep architecture, sleep efficiency and physiological parameters during sleep allowing for comprehensive assessment of sleep staging and arousals (Lim & Baumann, 2019). If testing for obstructive sleep apnea, home sleep apnea testing may be an alternative to in-laboratory PSG where available (Lim & Baumann, 2019). Conversely, a clinical history with or without sleep diary or self-reported questionnaires may suffice in patients presenting with a primary complaint of insomnia or circadian rhythm disturbance and PSG is not likely necessary (Lim & Baumann, 2019).

Actigraphy

Actigraphy devices are usually worn on the wrist or waist and record periods of rest and activity using an accelerometer to detect motion. They can be used as an objective measure of sleep-wake patterns to diagnose sleep disorders including insomnia and circadian rhythm disorders and are often used as an adjunct to data obtained from a sleep diary; however, they are not widely available clinically (Lim & Baumann, 2019). Recent studies have validated the use of actigraphy in patients with TBI across all levels of injury severity and cognitive impairment (Nakase-Richardson et al., 2016a). Further research is necessary to validate this measure in individuals with spasticity, paresis, depression, and/or agitation (Lim

& Baumann, 2019; Silver et al., 2018). As such, caution should be used when considering actigraphy as the sole tool to assess sleep in these patient populations (Lim & Baumann, 2019).

Multiple Sleep Latency Test

The multiple sleep latency test (MSLT) is a validated objective of excessive daytime sleepiness that is based on the premise that patients with a higher degree of sleepiness will have a shorter sleep latency. It is used to quantify daytime sleepiness and differentiate pathological sleep abnormalities from subjective complaints of sleepiness and post-TBI fatigue (Zasler et al., 2012). Using standardized conditions that eliminate or minimize external stimulating factors, patients are monitored for a period of time (typically 40 minutes) to determine their propensity to fall asleep. Essentially a napping test, normally alert individuals may fall asleep within 12 minutes on the test; individuals who fall asleep within <5 minutes have a positive test for excessive daytime sleepiness (Littner et al., 2005). The MSLT is the gold standard test for excessive sleepiness, and has been validated for narcolepsy and idiopathic hypersomnia, but not specifically for post-traumatic hypersomnia (Littner et al., 2005). Similar to the PSG, the MSLT may not be appropriate for all patients presenting with sleep-wake disturbances. The MSLT is not an appropriate test for ruling out obstructive sleep apnea (Lim & Baumann, 2019).

Maintenance of Wakefulness Test

The maintenance of wakefulness test (MWT) is an alternative test for excessive daytime sleepiness (Zasler et al., 2012). Similarly to the MSLT, mean sleep onset latency is recorded for all nap opportunities. The MWT is also based on the same premise of the relationship between sleep latency and sleepiness; however, in the MWT, patients are challenged to stay awake under standardized conditions designed to promote sleepiness: patients rest in a dark room with minimal external stimulation, and individuals who fall asleep in <8 minutes are deemed to be excessively sleepy (Littner et al., 2005). This test is less useful to diagnose narcolepsy. However, as it measures one's aptitude to stay awake, it is particularly relevant for the TBI population to infer variations in daytime vigilance or fluctuations in cognitive function (Arand et al., 2005; Castriotta et al., 2009).

The normal values for the MSLT and the MWT have been derived from comparing patients with narcolepsy to normal patients; therefore, these timing cut-offs may not be appropriate to diagnose post-traumatic excessive daytime sleepiness. Neither the MSLT nor the MWT have been validated in patients with brain injury.

13.3.10 Diagnosis

Sleep disorders resulting from an ABI should be diagnosed with a comprehensive evaluation to rule out comorbid medical conditions, psychiatric disturbances or environmental factors.

Findings from the clinical history should guide the physical exam and use of problem-focused laboratory (if necessary) and/or diagnostic testing (as indicated above) or referral to a sleep specialist.

Clinical guidelines and practice parameters are available for the diagnosis of circadian rhythm disorders (Morgenthaler et al., 2007), insomnia (2006; Sateia et al., 2017), obstructive and central sleep apnea (Aurora et al., 2012), hypersomnia (Morgenthaler et al., 2007), and parasomnias (Zak et al., 2010a; Zak et al., 2010b), although they are not specific to patients with a history of TBI. Diagnostic criteria according the DSM-5 and potential differential diagnoses are presented in Table 12.



Clinical Tip!

A detailed clinical history should examine a variety of factors including:

- Pre-injury and current sleep-wake patterns
- Frequency, duration, severity, development and fluctuations of night-time sleep difficulties or daytime sleepiness
- Contributing factors (i.e., pain, environment, comorbid conditions)
- Impact of sleep-wake disturbances and associated distress
- Use of prescription and over the counter medications, as well as other substances (e.g., natural products, caffeine, alcohol, or recreational drugs)
- Core symptoms of sleep disorders (e.g., snoring, breathing interruptions, hypnagogic hallucinations, nightmares, limb movements)
- Collateral history (from a family member, bed partner, or roommate) may be helpful when available

Table 12: Diagnostic criteria and possible differential diagnoses of common sleep disorders following ABI (Rao et al., 2015)

Disorder	Diagnostic criteria	Differential Diagnosis
Insomnia	Significant distress or impairment ≥ 3 nights per week for ≥ 3 months; not attributable to a comorbidity, substance abuse, or medication effect.	Medications (e.g., psychostimulants, corticosteroids, anticonvulsants, antidepressants), pain, psychological or medical comorbidities, environmental factors or life habits, other sleep disorders (e.g., sleep apnea), neuroendocrine disorders (e.g., growth hormone deficiency, hyperthyroidism).
Hypersomnolence	A main sleep period ≥ 9 hours that is non-restorative; difficulty in feeling fully awake on abrupt awakening; significant distress or impairment, frequency of ≥ 3 times per week for ≥ 3 months.	Medications (e.g., CNS depressants, opioids or other pain medications, anticonvulsants, anti-emetics, antihistamines, antidepressants, anxiolytics, beta-blockers, anti-spasticity medications, muscle relaxants), psychological or medical comorbidities, substances (e.g., cannabis, alcohol), age-related increased need for sleep (e.g., adolescents), other sleep disorders (e.g., sleep apnea), neuroendocrine disorders (e.g., hypothyroidism).
Narcolepsy	≥ 3 times per week for >3 months; cataplexy, orexin deficiency	Insufficient sleep syndrome (chronic sleep deprivation), medications (see above), other sleep disorders (e.g., sleep apnea, hypersomnia, Kleine-Levin syndrome).
Breathing-related sleep disorders	Nighttime breathing disturbances and negative daytime consequences (e.g., fatigue); for obstructive sleep apnea, this is often defined as ≥ 15 obstructions per hour of sleep.	Breathing-related sleep disorder is an umbrella term that encompasses central sleep apnea, obstructive sleep apnea, obesity hypoventilation syndrome, and sleep-related hypoventilation.
Circadian rhythm sleep-wake disorders	A persistent misalignment of the circadian system; excessive sleepiness or insomnia, clinically significant distress or impairment.	Environmental influences on circadian rhythm (e.g., shift work, inappropriate sleep environment, residing at extremely high or low latitudes), medications (e.g., CNS depressants or stimulants), substances (e.g., amphetamines, cannabis, alcohol), age-related increased/decreased need for sleep (e.g.,

		adolescents, elderly), other sleep disorders (e.g., sleep apnea).
Restless legs syndrome	Symptoms persisting for ≥ 3 months and occurring ≥ 3 nights per week; disturbed night-time sleep and daytime sleepiness.	Medications (antihistamines, dopamine antagonists such as anti-emetics or antipsychotics, lithium, antidepressants such as SSRIs and TCAs), substances (caffeine), akathisia, peripheral vascular disease Secondary causes of restless legs syndrome include iron deficiency, pregnancy, chronic kidney disease, and peripheral neuropathy.
Parasomnia	Varies with different parasomnias (see DSM-5).	

13.3.11 Management

The management of sleep disorders is disorder-specific and includes non-pharmacological and/or pharmacological approaches. Very few treatments have been validated in patients with post-traumatic brain injury sleep disorders. The following section provides an overview of both pharmacological and non-pharmacological interventions available for the management of sleep disorders following an ABI (summarized in Table 13)

Table 13: Summary of Interventions for Common Sleep Disorders Following ABI

Sleep Disorder	Non-pharmacological Interventions	Pharmacological Interventions
Insomnia	CBT** TBI education Sleep hygiene** Acupuncture Regular exercise** Avoid caffeine, other CNS stimulants	Melatonin** Melatonin agonists Trazodone** Benzodiazepines& Nonbenzodiazepine receptor agonists& Tricyclic antidepressants Other sedative antidepressants Orexin blockers Re-evaluate contributory medications
Sleep-related breathing disorders	Non-invasive positive pressure ventilation (NIPPV, such as CPAP or BiPAP) TBI education Sleep position (lateral decubitus) Weight loss (if overweight) Avoid alcohol, other CNS depressants Oxygen therapy Surgery for upper airway obstruction	Morning modafinil (100 to 400 mg) and armodafinil (150 to 250 mg) are approved for obstructive sleep apnea and excessive daytime sleepiness in patients who also use CPAP Re-evaluate contributory medications Consider referral to sleep specialist
Narcolepsy	TBI education	Stimulants Consider referral to sleep specialist
Hypersomnia	TBI education Caffeine and power naps	Methylphenidate** Modafinil and armodafinil Amantadine Re-evaluate contributory medications
Circadian rhythm sleep-wake disorders	TBI education Sleep hygiene Light therapy	Melatonin**
Restless legs syndrome	Iron supplementation (if iron deficient) Avoid caffeine, nicotine, alcohol Exercise program (if peripheral vascular disease)	Dopamine agonists (pramipexole) Levodopa/carbidopa Gabapentin Re-evaluate contributory medications
Parasomnia	Sleep hygiene	Dopamine agonists Sedative-hypnotics

**i indicates recommended by ONF Guidelines; & indicates ONF Guidelines recommend against use

Non-Pharmacological Interventions

Non-pharmacological management options include lifestyle (e.g., exercise), and behavioural interventions (e.g., cognitive behavioural therapy (CBT) and sleep hygiene), respiratory and surgical therapy, as well as complementary and alternative medicine (e.g., acupuncture or bright light therapy). Non-pharmacological interventions may be initiated simultaneously with pharmacologic strategies during management of comorbidities.

Lifestyle Interventions

Relaxation Strategies

Very limited evidence exists examining the use of relaxation strategies for sleep disturbances following an ABI. In a crossover RCT, Chiu et al. (2017) evaluated the effect of a warm footbath each evening on sleep latency and efficiency in a population with TBI. However, participants did not show significant improvements in total sleep time or sleep efficiency, despite a significant reduction in the number of times participants woke after sleep onset and sleep onset latency in the warm footbath group compared to controls. Future long-term studies with a larger sample size are needed to determine the impact of relaxation strategies, such as a warm footbath, on sleep in individuals with ABI.

The ONF-INESSS guidelines do not make any recommendations about relaxation strategies for the treatment of sleep disorder in individuals with TBI.

Behavioural Interventions

Cognitive Behavioural Therapy

Cognitive behavioural therapy (CBT) may be used for the management of primary (unrelated to other medical conditions) or secondary (related to comorbid medical or psychiatric conditions) insomnia. In the acute phase, CBT has been shown to be as effective as pharmacological agents and in the chronic phase has been shown to be even more effective than pharmacological agents (Morin et al., 2006; Murtagh & Greenwood, 1995; 湖, 1994). The goal of CBT is to modify behavioral factors that perpetuate insomnia, for example, sleep habits and dysfunctional beliefs about sleep. CBT for insomnia has five main components: stimulus control, sleep restriction, cognitive therapy, sleep hygiene education and fatigue management (summarized in Table 14). Using this method, Ouellet and colleagues found statistically significant and clinical improvements in insomnia in 73% of patients with mild-to-severe TBI (Ouellet & Morin, 2007).

The ONF-INESSS guidelines recommend that CBT should be considered in the treatment of sleep disorders for individuals with traumatic brain injury (ONF-INESSS, 2015).

Table 14: Treatment Components of CBT for Patients with Insomnia Following ABI (Ouellet & Morin, 2007)

Component of CBT	Key Methods
Stimulus control	Behavioural recommendations that reinforce associations between the bed, bedroom and bedtime with sleep rather than feelings of frustration, anxiety or tension.
Sleep restriction	Behavioural method limiting the time spent in bed to the actual sleep time, as assessed through daily sleep logs.
Cognitive therapy	Psychotherapeutic method designed to identify and alter dysfunctional beliefs or attitudes related to sleep (e.g., excessive worrying, unrealistic expectations or misconceptions about sleep).
Sleep hygiene	Education about health habits (e.g., diet, exercise or substance use) and environmental factors (e.g., light, noise or temperature) that impact sleep.
Fatigue management	Psychoeducative method to improve recognition and management (e.g., pacing and scheduling activities) of symptoms of fatigue, as well as change dysfunctional beliefs about fatigue and rest.

Sleep Hygiene

Sleep hygiene involves education about behavioural patterns and environmental factors that disrupt sleep. A full list of suggested strategies may be found in the guidelines for concussion/mild traumatic brain injury [HERE](#).

The ONF-INESSS guidelines recommend that sleep hygiene should be considered in the treatment of sleep disorders for individuals with traumatic brain injury (ONF-INESSS, 2015).

Respiratory and Surgical Therapy

In the general population, obstructive sleep apnea is often managed with alcohol avoidance, non-invasive positive pressure ventilation therapy (e.g., CPAP), proper sleep positioning (lateral decubitus, avoidance of supine position), dental or nasal appliances, and weight loss. The effectiveness of these interventions has not been reported for patients with ABI specifically, although many of these non-pharmacologic strategies address the underlying contributors to OSA. CPAP prevents soft tissues in the upper airway from collapsing by delivering constant pressure throughout the respiratory cycle (Zasler et al., 2012). In general, compliance with CPAP machines is often challenging for patients, with varying usage rates reported (40-84%) (Russell, 2014). Surgical management of obstructive sleep apnea is usually only offered for individuals who do not find CPAP therapy effective after at least 3 months (Silver et al., 2018).

The ONF-INESSS guidelines do not make any recommendations about respiratory therapy and surgical therapy for the treatment of sleep disorder in individuals with TBI.

Complementary and Alternative Medicine

Acupuncture

Few studies have examined the benefits of acupuncture in a population with ABI. In a small randomized trial, Zollman et al. (2012) found that acupuncture had no effect on objective measure of sleep; although, it did improve subjective measures of sleep quality and cognitive function. In another controlled trial, weekly sessions of acupuncture for five weeks led to short-term improvements in objective and subjective measures of sleep; however, improvements were not maintained at follow-up (Huang et al., 2018). A number of studies have demonstrated the effectiveness of acupuncture in treating insomnia within

healthy individuals and various other patient populations; however, many of these studies have methodological limitations (Zhao, 2013). Further research is necessary to determine the efficacy of acupuncture in a population with ABI.

The ONF-INESSS guidelines do not make any recommendations about acupuncture for the treatment of sleep disorder in individuals with TBI.

Light Therapy

The goal of light therapy is to shift waking or bedtimes towards a more desirable sleep-wake schedule. Typically, light therapy involves a person being exposed to a short wavelength light (430-475 nm; blue wavelength light) upon awakening. Further information regarding light therapy can be found in section 13.2.10.

The ONF-INESSS guidelines recommend that light therapy should be considered in the treatment of sleep disorders for individuals with traumatic brain injury (ONF-INESSS, 2015).

Pharmacological Interventions

Pharmacological management options include melatonin, antidepressants, benzodiazepines, non-benzodiazepines, and neurostimulants (summarized in Table 15).

Table 15: Summary of Pharmacological Interventions for Sleep Disorders Following ABI. Modified from (Silver et al., 2018).

Class	Drug and Dose	Impact on Sleep and Arousal	Side Effects and Cautions
		Indications in TBI	
Melatonin and Agonists	Melatonin 0.5-5 mg *doses of 2-5mg are recommended by the ONF Guidelines	↓ SL, ↑ SE Sleep initiation and/or maintenance	Headache, confusion, vivid dreams
	Ramelteon 8 mg	↓ SL, ↑ TST Sleep initiation and/or maintenance	Drowsiness, fatigue, dizziness
Orexin Receptor Antagonist	Suvorexant 5-20 mg	↓ SL, ↑ TST, ↑ SE Sleep initiation and/or maintenance	Somnolence, fatigue, headache, dry mouth
Antidepressants	Trazodone 50-150 mg *Note that the ONF-INESSS Guidelines recommend the use of trazodone (25-100mg) for insomnia post traumatic brain injury.	↓ SL, ↑ TST Sleep initiation and/or maintenance	Dizziness, dry mouth, nausea, blurry vision, drowsiness, hypotension, rebound insomnia, psychomotor impairments, QT prolongation
	Mirtazapine 15 mg	↓ SL, ↑ TST, ↑ SE Sleep initiation and/or maintenance	Drowsiness, dry mouth, weight gain, dizziness, impaired driving performance

Tricyclic Antidepressants	Doxepin 3-6 mg	↓ SL, ↑ TST Sleep initiation and/or maintenance	Headache, somnolence, sedation, dry mouth
	Nortriptyline 10-20 mg	↓ SL, ↑ TST Sleep initiation and/or maintenance	Headache, somnolence, sedation, dry mouth, dizziness, orthostatic hypotension, urinary retention
Benzodiazepines *note that the ONF Guidelines recommend against the use of these medications; if their use is necessary, the ONF Guidelines recommend limiting use to 7 days	Temazepam 7.5-30 mg	↓ SL, ↑ TST, ↓ nocturnal awakening	Rebound insomnia, altered sleep architecture, dependence, tolerance, amnesia, paradoxical aggression
Nonbenzodiazepine Sedatives ("Z drugs") *note that the ONF Guidelines recommend against the use of these medications; if their use is necessary, the ONF Guidelines recommend limiting use to 7 days	Zolpidem 1.75-12.5 mg	↓ SL, ↑ TST, ↓ SE Sleep initiation and/or maintenance Short-term use	Amnesia and impaired motor skills, rebound insomnia, rare somnambulism, somnolence, dizziness
	Zaleplon 5-10 mg	↓ SL Sleep initiation and/or maintenance Short-term use	Less likely to cause next-day impaired psychomotor skills
	Eszopicone 2-3 mg	↓ SL, ↑ TST, ↑ SE, ↑ quality of sleep Sleep initiation and/or maintenance Short- and long-term use	Amnesia and impaired motor skills, relatively long half-life, bitter taste, dry mouth, dizziness, somnolence
Stimulants	Methylphenidate 5-15 mg *Note that that ONF-INESSS Guidelines recommend short-term use of methylphenidate to reduce excessive daytime sleepiness in individuals with TBI.	↓ Fatigue, ↑ arousal Hypoarousal, excessive daytime sleepiness, narcolepsy	Arrhythmias, heart rate and blood pressure changes, seizures, weight loss
	Amantadine 50-400 mg	↑ Arousal Hypoarousal	Arrhythmias, agranulocytes, headaches
	Modafinil 200 – 400 mg	↑ Arousal Excessive daytime sleepiness, narcolepsy	Headache, ↑ heart rate and blood pressure, ↑ liver enzymes
	Armodafinil 150 – 250 mg	↑ Arousal Excessive daytime sleepiness, narcolepsy	Headache, ↑ heart rate and blood pressure

SE = Sleep efficiency, SL = sleep latency, TST = total sleep time

Q6. What are the clinical features of sleep disorders that might indicate a need for testing or referral to a sleep specialist?

- Features suggestive of narcolepsy (cataplexy, hypnagogic hallucinations, sleep paralysis, excessive daytime sleepiness)
- Features suggestive of obstructive sleep apnea or other breathing-related sleep disorders (consider polysomnography and/or referral to a sleep specialist)
- Features suggestive of restless legs syndrome (consider testing ferritin, creatinine, and/or other investigations suggested by physical examination)
- Patients with symptoms refractory to nonpharmacologic and/or pharmacologic management that are interfering with function or causing distress

13.4 Case Study

Patient Snapshot:

Mr. J...

42-year-old male involved in a high speed motor vehicle collision (MVC) resulting in a moderate brain injury and orthopedic injuries (fracture to his right tibia and fibula and right wrist) 6 months ago. He is currently living in the community and you are following him for his brain injury.

Lifestyle Factors: Mr. J had a previous MVC (two years past) that resulted in a number of orthopedic injuries and chronic pain. He is recently single and has a supportive family who live in another city. He and his family do not believe there was a history of snoring before his injury. He has used medical marijuana to manage pain and assist with sleep since before his brain injury. He continues to do so.

Medical History: Mr. J had an initial Glasgow Coma Scale score of 12, and his duration of post-traumatic amnesia was approximately four hours. An MRI has 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 abuse, and he has chronic neuropathic pain.

Signs & Symptoms: Mr. J describes significant fatigue and difficulty with sleep. Mr. J tried melatonin but did not find this helpful. He is currently taking sertraline and gabapentin.

You have previously assessed Mr. J's fatigue from a neuroendocrine perspective (see chapter 10) and have addressed his low mood with pharmacologic (venlafaxine, sertraline) and nonpharmacologic (CBT) strategies. What do you do next?



Review Mr. J's current medications.

Review Mr. J's substance use including alcohol and other depressants, caffeine and other stimulants.

Assess Mr. J for signs and/or symptoms of sleep and/or fatigue dysfunction.

***Note: Mr. J's neurobehavioural, neuroendocrine, and motor/sensory impairment management is continued in the Neurobehavioral, Neuroendocrine and Motor/Sensory Case Studies which are part of Chapters 5, 10 and 6 of this guidebook, respectively.**

Q1. What are some questions to ask Mr. J to screen for sleep dysfunction?

1. How long does it take you to fall asleep most nights?
2. Once you fall asleep, are you able to stay asleep? If not, what (if anything) wakes you from sleep?
3. Do you ever fall asleep during the day without meaning to?
4. Has anyone ever told you that you snore or that you wake from sleep gasping for air?
5. Do you ever wake up with a headache in the morning?

Mr. J reports daytime fatigue, occasional daytime naps, difficulty falling asleep and difficulty staying asleep. He denies other significant symptoms suggestive of mood or neuroendocrine dysfunction. He is

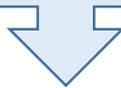
currently single, but states that previous partners and family members have not reported that he snores. He denies morning headaches. In addition to his medications (sertraline and gabapentin), he uses approximately 1 g of edible cannabis to fall asleep; this is sometimes effective.

Q2. What are some tools you can use to assess Mr. J's sleep and fatigue?

1. Pittsburgh Sleep Quality Index
2. Epworth Sleepiness Scale
3. Fatigue Severity Scale
4. STOP-Bang Questionnaire for Obstructive Sleep Apnea

You determine that Mr. J has issues failing in both sleep and fatigue domains. Screening questions and the STOP-Bang questionnaire do not support a likely diagnosis of obstructive sleep apnea. You provide education about post-ABI changes to sleep and fatigue. You review Mr. J's sleep hygiene practices and make recommendations for improvement (see section 13.2.10). You also recommend he try CBT for fatigue and sleep.

You ask Mr. J to try to incorporate some of the sleep hygiene strategies you have recommended. He also plans to pursue sleep and fatigue-specific CBT. You ask him to return in 3 months to assess his progress.



Mr. J reports some improvements in his sleep quality and consistency with the introduction of sleep hygiene practices and CBT strategies but he continues to experience delayed sleep initiation and bothersome daytime fatigue. You discuss the role that regular physical exercise and pacing and planning may have in improving energy levels and sleep quality. You also present pharmacological options. At this time, Mr. J would prefer to continue with nonpharmacologic strategies.

Q3. If, in future, Mr. J wishes to try pharmacologic options for his delayed sleep initiation, what medication options could you consider?

1. Re-evaluating Mr. J's current medications (sertraline and gabapentin)
 2. Trazodone (25-100mg nightly)* (beware serotonin syndrome, as Mr. J is on sertraline)
 3. Mirtazepine* (beware serotonin syndrome, as Mr. J is on sertraline)
 4. Benzodiazepines or non-benzodiazepine sedative hypnotics could be considered for short-term use (<7days) but are not recommended by the ONF Guidelines
- NB: Mr. J has already tried melatonin, which is recommended by the ONF Guidelines.

Q4. If, in future, Mr. J wishes to try pharmacologic options for his daytime fatigue, what medication options could you consider?

1. Methylphenidate can be considered for excessive daytime sleepiness (ONF Guidelines)
2. Modafinil or amantadine are other potential options (although there is limited evidence in patients with ABI)

13.5 Summary

In summary, ABI may lead to pathological dysfunction of a variety of processes that regulate fatigue and sleep-wake functions, resulting in persistent non-physiological fatigue and/or sleep disorders. Although sleep disturbances and fatigue were presented separately from each other in this guidebook, it is important to note that they are not mutually exclusive. Symptoms of one disorder or a combination may present at any time throughout injury and across all severities or mechanisms of injury. For those recovering from an ABI, fatigue and sleep disorders are highly disruptive and dramatically interfere with one's physical, cognitive and social abilities. Consequently, it is important for clinicians to perform comprehensive assessments of patients' sleep and/or fatigue including assessing potential contributing causes or remediable factors affecting a patient's sleep or fatigue. Although there is little ABI-specific evidence for the management of sleep and fatigue dysfunction, clinicians may rely on evidence from the general population as a starting point for addressing many of these conditions.

13.6 References

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