

Nonpharmacological management of sleep disturbances after traumatic brain injury

Alphonsa Thomas and Brian D. Greenwald*

JFK Medical Center-Johnson Rehabilitation Institute, Edison, NJ, USA

Abstract.

BACKGROUND: Sleep plays an integral role in several physiologic functions such as cognition and functional ability. Sleep disturbances are common after brain injury and can interfere with rehabilitation and recovery. There are several pathways by which sleep is initiated, and thus various ways to target common complaints as well.

OBJECTIVES: To review alternative and non-pharmacological treatment approaches to sleep disorders following TBI.

METHODS: The authors present a review of the literature on various alternative treatment methods for treating sleep disorders, and discuss evidence for safety and efficacy.

CONCLUSIONS: Typical management include conservative measures and pharmacologic treatment. This article provides a review of the available nonpharmacologic treatment options for sleep disorders in patients with traumatic brain injury. There are a number of non-pharmacological treatment methods available for treatment of sleep disorders in this population.

Keywords: Sleep, traumatic brain injury, nonpharmacologic

1. Introduction

Proper sleep is one of the mainstays of treatment when promoting the recovery of brain injured patients. Without it, there can be observed impairments in cognition, physical functioning, and it can lead to stagnation of neurorecovery. The anatomy of sleep is an intricate process and involves the subtleties of environmental factors to complex cortical and sub-cortical connections (Barshikar & Bell, 2017). The brain regulates sleep via several pathways and connections and it is when these are disrupted that we see sleep abnormalities arise.

Sleep disturbances are a common occurrence after a traumatic brain injury (TBI). They can appear as soon as 24 hours after injury and continue for several years (Zollman, Larson, Wasek-Throm, Cyborski, & Bode, 2012). The prevalence of sleep disorders ranges from 30 to 84% in the TBI population. It is

estimated that about 68% of patients in rehabilitation units demonstrate some form of sleep disturbance (Barshikar & Bell, 2017). A meta-analysis performed on a group of TBI patients showed that the most common sleep disturbances were insomnia (50%), poor sleep maintenance and sleep efficiency (49–50%), delayed sleep onset (36%), early morning awakenings (38%), and nightmares (27%) (Mathias & Alvaro, 2012). Another meta-analysis by Grima et al. looked at pooled polysomnography data which showed that TBI patients had poor sleep efficiency, shorter total sleep duration and greater wake after sleep onset time. Although the sleep architecture for the control and TBI patients were similar, the data suggested that TBI patients spent less time in REM sleep (Grima, Ponsford, Rajaratnam, Mansfield, & Pase, 2016).

Traumatic brain injury patients with sleep disturbances have been found to have longer inpatient hospital stays, higher cost of rehabilitation, and higher rates of functional disability (Lim et al., 2013). Despite this, sleep-wake cycle disorders are not thought to be static after TBI. Based on a prospective study of patients with moderate to severe TBI in

*Address for correspondence: Brian D. Greenwald, MD, Medical Director of Center for Brain Injuries, JFK Medical Center-Johnson Rehabilitation Institute, 65 James Street, Edison, NJ 08820, USA. Tel.: +1 732 321 7000 /Ext: 62018; Fax: +1 732 321 7733; E-mail: Brian.Greenwald@hackensackmeridian.org.

acute rehabilitation, 84% were found to have sleep disorders on admission, 63% after 3 weeks and 59% at 4 weeks post-injury (Barshikar & Bell, 2017; Nakase-Richardson et al., 2013). Given the high incidence of sleep disturbances in the TBI population, the optimization of sleep is cornerstone to promoting neurorecovery. There are many medications and other less invasive strategies to help promote sleep. This article will discuss the non-pharmacologic treatments options to help assist those with sleep disorders after traumatic brain injury.

2. Diagnosis

Identifying the root cause of sleep disturbances requires some detective work, and a proper history can assist with this. Target areas that should be focused on include: environmental factors, medical comorbidities, primary sleep disorders, medication use, stimulant use, and mood/psychiatric factors. A patient's typical routine for bed prior to their injury and at the current time should also be evaluated. Polysomnography is regarded as the gold standard for diagnosing sleep disturbances. It entails monitoring sleep patterns through the use of electroencephalography (EEG), electromyography (EMG) and electrooculography (EOG). Other diagnostic tools include sleep diaries and activity monitors such as wrist actigraphs.

3. Treatment

Similar to the management of many disorders in medicine, the approach to sleep should start with conservative measures with progression to more aggressive options if needed. The initial management of sleep should involve ruling out possible causes of disturbed sleep and modifying them. Examples of such factors would be pain, bowel/bladder issues and mood changes. It is after these likely culprits are ruled out that further intervention can be made.

3.1. *Sleep hygiene and behavioral therapy*

Sleep hygiene should be considered for all those with sleep disorders. It is a relatively low cost and low risk approach to management of sleep that patients should be able to easily implement it into their general routine. While it is a very broad term, sleep hygiene can be broken down into 4 domains:

sleep homeostatic factors, circadian factors, medication/drug effects, and arousal in sleep setting (Bogdanov, Naismith, & Lah, 2017). Sleep homeostatic factors entails items such as regular exercise. Studies have shown that TBI patients who performed aerobic exercise on a regular basis compared to those who did not had improved quality of sleep (Bogdanov et al., 2017).

Behavioral modification aims to alter a patient's negative associations with sleep. Techniques include stimulus control, sleep restriction, relaxation techniques, and cognitive behavioral therapy (Ouellet, Beaulieu-Bonneau, & Morin, 2015). Cognitive behavioral therapy (CBT) is considered the first line approach for management of insomnia. It helps identify, challenge, and alter dysfunctional beliefs and attitudes about sleep. Table 1 provides a brief summary of each form of therapy involved. CBT has been shown to be successful and even superior to pharmacologic treatment in the treatment of insomnia within the general population (Jacobs, Pace-Schott, Stickgold, & Otto, 2004; Ouellet, Beaulieu-Bonneau, & Morin, 2015). Its use has also been found efficacious in the traumatic brain injury population (Nguyen et al., 2017). Treatment using these approaches have been found to help improve sleep onset and efficiency while decreasing nighttime awakenings (Ouellet, Beaulieu-Bonneau, & Morin, 2015).

3.2. *Light therapy*

Ocular light exposure of certain wavelengths has been used as a stimulant and as an anti-depressant. It is known to stimulate circadian, neuroendocrine and neurobehavioral responses (Sinclair, Ponsford, Taffe, Lockley, & Rajaratnam, 2013). Light exerts its effects on the brain via a non-image forming photoreceptor system that is separate from rods and cones (Sinclair et al., 2013; Wu et al., 2018). Studies show that exposure to blue or blue-enriched light have the greatest improvements in level of alertness and mood. It has also been shown to improve sleep efficiency, increase sleep time, and reduce nocturnal awakenings (Wu et al., 2018).

A study by Sinclair et al. looked at this specifically in the TBI population. Patients received 4 weeks of blue light therapy for 45 min/day at home. Findings showed reduced fatigue and daytime sleepiness with a trend toward baseline after stopping treatment (Sinclair et al., 2013). The study also found that the magnitude of reduction in both fatigue and daytime sleepiness with the blue light therapy was greater

Table 1
Components of CBT (Ouellet, Beaulieu-Bonneau, & Morin, 2015)

Stimulus control
Recommendations designed to reassociate the bed and bedroom with sleep, rather than anxiety or other stressors. Reinforcing a regular sleep-wake schedule.
Sleep restriction
Limiting the time spent in the bed or bedroom for only sleep (go to bed only when sleepy, no reading or problem solving in bed). As the patient progresses this restriction is loosened until a desired sleep duration is attained.
Cognitive therapy for insomnia
Psychotherapeutic method aimed at identifying and evaluating any negative attitudes or beliefs that may reinforce insomnia (excessive worrying, misconceptions about sleep).
Sleep hygiene education
General recommendations and education about environmental (room temperature, light, noise) and lifestyle factors (exercise, diet, drugs, alcohol) that impact sleep.
Fatigue management
Method of education to increase awareness regarding fatigue, how to manage it, and to create ways to conserve energy (pacing, planning daily activities).

than that reported in a comparable study which studied modafinil 100–200 mg daily for 6 weeks (Sinclair et al., 2013)

3.3. *Chiropractic and osteopathic manipulative treatment*

There are limited studies on the use of chiropractic and osteopathic manipulative treatments (OMT) for the management of insomnia. Research on chiropractic treatment suggests that spinal manipulative therapy excites GABA-ergic inhibitory neurons, promotes relaxation of the mind, and non-rapid eye movement sleep (Kingston, Raggio, Spencer, Stalaker, & Tuchin, 2010). Different pathways have been suggested for how the therapy works, but no clear studies have been performed to confirm them.

Cranial/craniosacral therapy is a subset of osteopathic treatment. It is a form of noninvasive manual therapy that aims to correct any restrictions within the craniosacral system (CSS). The CSS is comprised of meninges, the bones attached to meninges, glia and CSF (Wetzler, Roland, Fryer-Dietz, & Dettmann-Ahern, 2017). The free and fluid movement between these structures are the focus behind the treatments therapeutic effects. The primary respiratory mechanism (PRM) is the driving force behind the motion between the cranial bones. The motion that is produced can be palpated by trained osteopaths (Cutler, Holland, Stupski, Gamber, & Smith, 2005). Cutler et al. studied the use of cranial manipulation, specifically compression of the fourth ventricle (CV4) technique, and its effects on sleep latency. The group hypothesized that the CV4 technique helped reduce sleep latency by decreasing sympathetic tone and results were consistent with this. The authors note

that despite the results, mechanisms behind this relationship requires further research (Cutler et al., 2005; Kingston et al., 2010).

Wetzler et al. used a combination of different OMT styles (craniosacral therapy, visceral manipulation, and neural manipulation) to assess its therapeutic effect on concussion recovery. Visceral and neural manipulation target restrictions in the tissue, organs and nerves of the body. It promotes freedom of movement, normal vascularity, tone, and pressures (Wetzler et al., 2017). The results of the study showed a significant reduction in pain around the head/neck region and sleep time was doubled. Hours of sleep averaged 2 hours on the first day of treatment and increased to 4 hours at the end of treatment with continued increase at 3 month follow-up. Treatment also helped to reduce the fatigue and anxiety in the study group as well (Wetzler et al., 2017).

3.4. *Acupuncture*

Acupuncture has been noted to have a beneficial effect on sleep. Zollman et al. performed a pilot study on TBI patients with chronic insomnia and acupuncture. Despite scant use of sleep aids in both arms of the study, results showed that those treated with acupuncture were able to taper off these medications within the first week of intervention. The control group and treatment group did not show a difference in total sleep time, but those with acupuncture reported a higher quality of sleep. This effect was noted to last at least one month after discontinuing treatment. Cognitive function improved to a significant degree in the treatment group. Interestingly, this improvement does not appear to correspond to the data found. There was no difference in total sleep time

between both arms of the study, and despite less medication use in the treatment group this finding did not reach clinical significance. This might suggest that acupuncture might have other indirect effects in the TBI population (Zollman et al., 2012).

Mindfulness meditation has also led to reductions in total wake time, presleep arousal time and insomnia severity index (Barshikar & Bell, 2017). There is some research into the neural mechanism of this form of meditation. There is evidence that regions of the brain such as the insula, putamen, somatosensory cortex, parts of the anterior cingulate cortex, and prefrontal cortex are activated with mindfulness meditation. Research also shows that the activity within the amygdala can be adjusted which help to reduce anxiety and depression. Routine meditation appears to lead to improved emotional control and regulation (Zou, Wu, & Fan, 2016).

3.5. *Diet*

The goal of nutritional interventions are to modulate neurotransmitter activity within the brain that are associated with the sleep-wake cycle. Some of these are orexin, serotonin (5-HT), gaba-aminobutyric acid (GABA). Dietary changes help to supplement the brain with the building blocks needed to augment these neurotransmitters (Halson, 2014).

Branched chain amino acid (BCAA) supplementation into the diet of mice with TBI and sleep disorders have been found to restore sleep-wake regulation and orexin neuronal activity (Elliott et al., 2018; Lim et al., 2013). BCAAs are precursors to glutamate and GABA synthesis within the brain, both of which act as mediators of orexin neuronal activity (Elliot et al., 2018). TBI decreases the density of glutamate immuno-gold labeling within nerve terminals who make an excitatory synaptic contact onto orexin neurons. Orexin producing neurons are located within the lateral hypothalamus and produce the neuropeptides orexin-A (hypocretin-1) and orexin-B (hypocretin-2). These neuropeptides bind to G-protein coupled receptors and project throughout the cerebral cortex, limbic system and brainstem. It is an important component of sleep-wake regulation. Low levels are found to be consistent with narcolepsy post-TBI and exogenous administration helps promote wakefulness (Elliot et al., 2018).

The production of 5-HT is dependent on the availability of its precursor amino acid, L-tryptophan (Trp) (Halson, 2014). Trp is the least abundant of amino acids. Therefore, ingestion of other forms of protein

that increase large neutral amino acids (LNAA) can decrease the uptake of Trp into the brain. There are several ways by which brain Trp can be increased. There is limited research in the TBI population on the effect of a carbohydrate diet on sleep but the available studies allude to some benefit. The ingestion of carbohydrate helps to increase brain Trp via insulin which drives LNAA into skeletal muscle (Halson, 2014). A high carbohydrate diet has been found to enhance sleep-onset latency, increase REM sleep, decrease light sleep and wakefulness. A high protein diet appears to result in improved sleep quality through fewer wake episodes (Halson, 2014).

3.6. *Melatonin*

Melatonin is a hormone that is secreted from the pineal gland and plays an integral role in the sleep wake cycle. 5-HT is a precursor to the synthesis of melatonin (Halson, 2014). Prior research has shown that patients with TBI have lower melatonin levels in the evening and night compared to their healthy counterparts, which suggests that TBI disrupts the melatonin pathway. A study by Grima et al, showed 42% less melatonin overnight in the TBI group compared to controls. The patients averaged 6 years post-injury which suggests that this disturbance in the melatonin pathway is an ongoing impairment (Grima, Ponsford, Hilaire, Mansfield, & Rajaratnam, 2016). Besides interruption of melatonin synthesis, using mouse models, it has also been found that TBI likely lowers the level of melatonin receptor subtypes MT1 and MT2 (Osier et al., 2017). Supplementation with melatonin has shown to improve subjective sleep quality and actigraphic sleep efficiency. It was also found that melatonin reduced self-reported anxiety, fatigue, improved self-perceived vitality and mental functioning (Grima et al., 2018).

3.7. *Herbs and other nutritional remedies*

There are several options of homeopathic and dietary supplements available as sleep aids. Research using these agents in the brain injury population is scarce. Valerian is an herb that is thought to induce a calming effect by binding to GABA A receptors, and attenuating the excitability of the nervous system (Halson, 2014). Dosage varies from 300 to 600 mg a day. These preparations are not FDA regulated, and therefore the content and concentration can vary (Fernández-San-Martín et al., 2010). A meta-analysis performed on the use of valerian in insomnia

showed that the herb would be effective for subjective improvement of insomnia (greater sleep quality), but further studies on its objective improvements are wanted (Fernández-San-Martín et al., 2010).

Other sleep aids include hops, passionflower, kava, lavender, skullcap, lemon balm, lysine, magnesium, St. John's wort, magnolia bark, GABA and 5-hydroxytryptamine. The research on these supplements are finite, and even more so in the TBI population. They are commonly found in over the counter sleep remedies (Halson, 2014).

3.8. *Miscellaneous*

A randomized control study looked at the efficacy of home-based warm footbaths on sleep disturbances in TBI. Results showed reduced latency of sleep and waking after sleep onset in those patient who used the footbath. The role of warm-sensitive neurons are thought to be an important role in temperature and sleep regulation (Chiu, Lin, Chiu, & Chen, 2017). The study notes that a relatively high distal skin temperature has been implicated in the initiation of sleep through the proposed mechanism of modulating the firing rate of warm-sensitive neurons in the preoptic area and anterior hypothalamus. These neurons are postulated to increase their firing rate with the initiation of sleep, and thus increasing skin temperatures leads to a similar rise in firing rate leading to sleep (Chiu et al., 2017). The promotion of relaxation is also a contributing factor to this.

Cranial electrotherapy stimulation (CES) is an FDA approved treatment method to decrease insomnia, but the use of this treatment within the TBI population is lacking. It first attained in approval for marketing in 1979 for the treatment of anxiety, depression and insomnia. Treatment involves the use of a small electronic device that sends weak, pulsed electrical stimulation to the brain via electrodes on the scalp (Kirsch & Nichols, 2013). The neurophysiologic effects and overall relaxation from the use of CES arises due to the release of neurotransmitters such as beta-endorphins, adrenocorticotrophic hormone, serotonin, melatonin, norepinephrine, cholinesterase and decreased serum cortisol levels. Functional magnetic resonance imaging shows that CES causes cortical deactivation, similar to those produced by anxiolytic medications (Kirsch & Nichols, 2013). Is also thought to work on the reticular activating system, thalamus, hypothalamus and limbic systems. The effects of this treatment are thought to be cumulative. Specifically in insomnia

patients, results can vary widely from immediate benefit to improvement 2 months after treatment (Kirsch & Nichols, 2013).

A rather controversial alternative to management of sleep disorders is the use of cannabinoid products. The endocannabinoid system is thought to serve as a link between the circadian sleep-wake cycle. The two most researched compounds are delta-9 tetrahydrocannabinol (THC) and cannabidiol (CBD). Research on THC suggests that it provides a short-term sleep benefit, but that chronic use can lead to habituation, a less pronounced circadian rhythm, daytime sleepiness, delayed onset sleep latency and negative mood and memory impairments (Babson, Sottile, & Morabito, 2017). CBD has a lower addiction profile compared to THC. At low doses it has been shown to be stimulating while at higher doses can be sedative. Noted effects on sleep disorders include increase in total sleep time (Babson et al., 2017). A review of the literature thus far, reveals that the information we have on the use of cannabinoids and their utility is mixed and further research needs to be done to gain a better grasp of its effects (Babson et al., 2017). While there are some studies that show some benefit with use, caution must be taken in the TBI population as use of these compounds can cause further cognitive impairments.

4. Conclusion

The management of sleep disorders after TBI is of utmost importance as it lends itself to neurorecovery. Failure to obtain adequate rest has been associated with impaired cognition and physical function. Using conservative measures prior to the institution of pharmacologic agents help to prevent the unwanted side effects of mediations. There are several options to choose from based on patient preferences and clinical judgement. The optimization of sleep helps brain injured patients maximize their limited time in rehabilitation without the stressors from lack of sleep impeding them.

Conflict of interest

There are no disclosures or conflicts of interest reported by the authors. No funding was received by the authors for this work.

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