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Review Article

Screening for sleep dysfunction after traumatic brain injury

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ABSTRACT

Numerous studies on the high prevalence of sleep disorders in individuals with traumatic brain injury (TBI) have been conducted in the past few decades. These disorders can accentuate other consequences of TBI, negatively impacting mood, exacerbating pain, heightening irritability, and diminishing cognitive abilities and the potential for recovery. Nevertheless, sleep is not routinely assessed in this population. In our review, we examined the selective screening criteria and the scientific evidence regarding screening for post-TBI sleep disorders to identify gaps in our knowledge that are in need of resolution. We retrieved papers written in the English-language literature before June 2012 pertinent to the discussion on sleep after TBI found through a PubMed search. Within our research, we found that sleep dysfunction is highly burdensome after TBI, treatment interventions for some sleep disorders result in favorable outcomes, sensitive and specific tests to detect sleep disorders are available, and the cost-effectiveness and sustainability of screening have been determined from other populations. The evidence we reviewed supports screening for post-TBI sleep dysfunction. This approach could improve the outcomes and reduce the risks for post-TBI adverse health and nonhealth effects (e.g., secondary injuries). A joint sleep and brain injury collaboration focusing on outcomes is needed to improve our knowledge.

1. Introduction

Traumatic brain injury (TBI) has been defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force," [1] and is a leading cause of death and disability in adults and children in Canada and the United States [2]. The sleep and wakefulness cycles often become disturbed with brain injury, irrespective of the severity [3–5]. This disturbance occurs through various pathophysiologic mechanisms resulting from direct and indirect trauma to the brain areas responsible for sleep-wake regulation regulation; conceivably, damage to these areas can lead to hypothalamus and neurotransmitter dysfunction. Furthermore, pain resulting from neck or back injury or other medical or psychiatric disorders caused by brain trauma and subsequent medications may affect sleep. Once sleeping problems develop they lead to additional confusion, frustration, and depression [6], further impairing the individual. A recent study of 29,640 US Navy and Marine Corps men with blast-related TBI found that sleep problems mediated the effect of a positive TBI screen on the development of posttraumatic stress disorder and depression [7], highlighting the significance of early identification and treatment of sleep dysfunction following injury. Moreover, individuals with sleep complaints are more sensitive to pain and may increase their medication requests, increasing sleep disruption and establishing a pathologic reverberating loop [8,9]. High comorbidity of chronic pain with head and other trauma has been reported, with headaches as the primary complaint in postconcussive syndrome. The incidence of headaches is a whopping 90% in the immediate period following the accident [10], with 44% of patients experiencing ongoing problems 6 months after injury [11]. Moreover, seizures and epilepsy, important medical and neurologic TBI sequelae, have an established relationship with sleep dysfunction. Sleep deprivation is a seizure trigger [12] and recent studies on the relationship between sleep disturbances and epilepsy emphasize the significance of early diagnostic and therapeutic management of sleep dysfunction in individuals with TBI, given its influence in posttraumatic seizure development [13–15].

In the past two decades, numerous researchers have studied the relationship between TBI and sleep disturbance, with coinciding findings and conclusions: sleep dysfunction is prevalent in this population and is an integral component in health following injury. It is crucial that the evidence is acknowledged and that steps are taken to integrate the screening and management of sleep complaints and the associated wakefulness disturbances into TBI clinical programs.

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Presently, most individuals with TBI do not undergo sleep dysfunction screening or assessment during their treatment and rehabilitation phase. This constitutes a knowledge-to-practice gap that requires intervention to ensure an evidence-based approach to the evaluation and rehabilitation of individuals with TBI. Our review first aimed to appraise the selective screening criteria and examine the scientific evidence regarding sleep disorder screening following TBI; second, we aimed to identify gaps in our knowledge that are in need of resolution.

2. Search strategy and selection criteria

We searched PubMed using *exp sleep** OR *sleep disorders*, AND *exp brain injuries*, *craniocerebral trauma*, *coma*, *post-head injury*, *head injuries*, *penetrating*, *intracranial hemorrhage*, *traumatic*, OR *exp skull fractures*. The appropriate truncations were included. Only papers written in the English-language literature published before June 2012 were used. The reference lists of relevant review articles were scanned for published studies that may have been overlooked during the electronic literature search. We emphasized experimental studies, prospective epidemiologic studies, and nonrandomized and randomized trials. An additional literature search was performed using the PubMed database for findings on cost-effectiveness from other study populations.

3. Screening as a secondary prevention tool

Screening is a secondary prevention tool, the primary goal of which is to prolong life, decrease morbidity, and improve the outcome and quality of life—all using the available resources. According to the Canadian Task Force on Preventive Health Care [16,17], the selective screening criteria for the presence of existing disease in a specific population subgroup are: (1) the targeted disease must be sufficiently burdensome to the population that a screening program is warranted; (2) efficacious treatment for the target illness must be available; (3) early detection must improve disease outcome; (4) accurate (i.e., sensitive and specific) screening tests should be available; (5) diagnosis and treatment facilities should be available; and (6) cost, feasibility, and acceptability of screening and early treatment should be established.

4. Analysis

4.1. Nature of sleep (and wake) disorders following TBI: incidence, prevalence, and impact

The International Classification of Sleep Disorders lists 84 sleep disorders under eight major categories, each possessing a well-understood history and a description of disorders that can be successfully treated [18]. Many of these disorders are sufficiently burdensome after TBI [19]. Specifically, this applies to insomnias [20,21], sleep-related breathing disorders (SRBD) [22], hypersomnia not due to breathing disorders [3,23], circadian rhythm sleep disorders (CRSD) [24–26], sleep-related movement disorders [27], and other sleep disorders [28].

4.1.1. Insomnia

Fichtenberg et al. [21] reported the increased insomnia incidence following acute TBI (e.g., 4 months after injury) relative to the general population. Thirty percent of TBI patients were found to have insomnia, with both sleep initiation difficulties and sleep duration limitation being more than three times as common compared to that in the general population [20]. The study also reported that TBI patients with insomnia had poorer vocational outcomes, more behavioral problems, cognitive and communicative dysfunctions, and a higher incidence of anxiety and depression. Insomnia with shortened sleep time was the most biologically severe phenotype of the disorder, associated with cognitive-emotional and cortical arousal; activation of the limbic and stress systems; and high risk for hypertension, diabetes mellitus, neurocognitive impairment, and mortality [29]. A sleep duration of less than 5 h also was shown to be associated with an increased risk for falls [30], lower grip strength, and slower walking speed in a population of elderly men [31]; poorer cognitive performance in the Nurses' Health Study Cohort [32]; and obesity in individuals older [33] and younger than 65 years [34].

Other than these general insomnia patterns, some factors specifically related to TBI could explain the increased risk for insomnia. Although the presence of insomnia following injury has not been found to be related to time since injury, injury severity appears to play a role. Several studies have reported more insomnia complaints in individuals with milder TBI, possibly due to their greater awareness of changes to sleep since injury [35,36]. Another explanation is that mild TBI has been shown to increase cortical arousability, which could be due to hypocretin (orexin) neurotransmitter dysregulation because of the injury. This hyperarousability causes sleep fragmentation and increases the amount of lighter stages of sleep, leading to the feeling of being unrested or unrefreshed on awakening, and therefore feeling greater dissatisfaction with sleep [37]. It also could be from increased intracranial pressure during the night after the injury, which was shown to be associated with reduced sleep quality [38]. Lastly behavioral and psychosocial factors such as irregular sleep–wake pattern, consumption of caffeine and other substances, excessive worrying at night, and extended time in bed including naps, also may contribute to insomnia, which may be challenging but necessary to address.

4.1.2. SRBD

Higher SRBD prevalence in TBI populations ranges from 25% to 35% [22,27] compared to 6% in the general population [39]. Recent systematic reviews have demonstrated an increased risk for serious adverse effects in patients with untreated SRBD [40]. Acute cardiovascular events have been associated with SRBD, and an extremely high odds ratio for myocardial infarction in patients with obstructive sleep apnea (OSA, a form of SRBD) has been observed [41], as well as axonal damage because of OSA [42]. A recent neuropsychologic study of TBI patients with OSA revealed cognitive domain deficits in executive functioning and attention [43]. It also has been reported that cognitive performance deterioration is significantly correlated with increased severity of nocturnal breathing irregularity, magnitude of nocturnal hypoxemia, and the extent of sleep disruption in patients with SRBD for decades in the general population [44,45].

The other effects of SRBD include exacerbation of epilepsy through sleep disruption and deprivation, hypoxia, and decreased cerebral blood flow [46]. Because head trauma also has been cited as a cause of epilepsy with the relative risk for being diagnosed with the seizure disorder increasing 2- and 7-fold after mild and severe head injury, respectively [47], it is difficult to dismiss these relationships as coincidental. Therefore, it would make sense to consider sleep factors in posttraumatic epileptogenesis. Seizures can also often cause repetitive periods of apnea, which can closely mimic the conditions of obstructive or central apneas [48]. Clinical differentiation between sleep disorders and epileptic events after TBI should be considered in any recurrent, stereotyped, and unusual events during sleep reported by a TBI patient.

Recently, the effect of untreated OSA on cognitive decline in TBI patients was uncovered: TBI patients with OSA performed significantly worse on neuropsychologic testing than those without