

Review Article

Sleep Architecture in Head Injury – An Entity Slowly Coming Out of its Shell

Munakomi S*

Department of Neurosurgery, College of Medical Sciences, Chitwan, Nepal

*Corresponding author: Sunil Munakomi, College of Medical Sciences, Chitwan, Nepal

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Introduction

Traumatic Brain Injury (TBI) is a silent epidemic out crying its impact on a global front [1]. It is not only accountable for early and late consequences on the patients but it also heightens the emotional burden onto their families and also cripples the economy of any nations. One of the neglected aspects of the consequences of TBI is its impact on the sleep architecture and the outcome thereafter. This epiphenomenon has now slowly been brought out of its shell [2].

Implications of sound sleep

Sound and effective hours of sleep have paramount implications in clearing away waste metabolites in the brain through the G-lymphatic pathways. This is prudent for timely restoration of the internal milieu inside the brain for its efficient functioning [3]. Sleep promotes enhanced Cerebrospinal Fluid (CSF) to Interstitial Fluid (ISF) exchange [3]. Studies have even verified that lateral and supine positions are more favorable for this clearance process [4]. However, there is certain timeframe within the sleep cycle that bears immense importance with regard to its role in physiological restoration and waste disposal. Studies have proven that the N3 phase is the most crucial in this aspect wherein presence of Slow Wave Sleep (SWS) decreases the Cerebral Metabolic Rate of Oxygen (CMR02) thereby minimizing its metabolic demand [5]. This can have a huge hand in silencing the metabolically stormy brain during early phase of TBI. Moreover, Growth hormone pulsatile release during sleep accelerates tissue healing and hastens recovery [6]. So is true for the release of cortisol during hours of sleep which combats physiological disequilibrium inside our body. In a nutshell, proper sleep reenergizes the tiring brain.

Sleep-a tightly monitored physiology

The sleep-wake cycle in our body is timely regulated via fine tuning and interplay between process C that maintains wakefulness and process S that governs sleep phase [7]. These are guarded through the pre-optic region of the hypothalamus. Circadian rhythm of the sleep wake cycle is the consequence of periodic change in the expressions of Period gene and Cryptochrome gene (together referred as clock genes). Supra-chiasmatic nucleus through its relays on the Dorso-medial nucleus in the thalamus coordinates this sleep wake system [7]. The control system in the Pons allows for the timely switch from Non Rapid Eye Movement (NREM) to Rapid Eye Movement (REM)

Abstract

Herein we enlighten on interplay between sleep and head injury and its impact to the patient. We recommend on implementing sleep hygiene in management of these patients as this approach can prove to be a prodigy in our attempt to nullify the short and long term consequences of fragmented sleep cycle.

Keywords: Sleep cycle; Brain trauma; Waste clearance

during the sleep [8]. Overall this tightly monitored system has further connections to the Reticular Activating System (RAS) as well the forebrain circuits.

Impact of trauma on the sleep hygiene

Sleep architecture in ICU patients is grossly fragmented [9]. Most of the patients lack sleeps in the N3 phase that is most fruitful for the restorative mechanism. They characteristically have difficulty for sleep initiation and continuity. Major subsets of patients managed in the Intensive Care Unit (ICU) spend most hours of their sleep in the nonconsolidated and light N1 and N2 NREM phases [9].

There are various factors that ameliorate sleep hygiene in the ICU such as noise, lights, medications and the frequent physical interplay during patient care [10]. Moreover, trauma can have impact on the hypothalamic pituitary axis thereby affecting the system that monitors circadian sleep rhythm [11]. It disrupts the tightly regulated physiological process between different centers within the brain.

TBI in early phase is metabolically stormy. There is interplay of different collaborative processes such as excito-toxicity, apoptosis, parthanatos, lactate storm etc that further insults the already traumatized brain. This adds up the physiological dysregulation

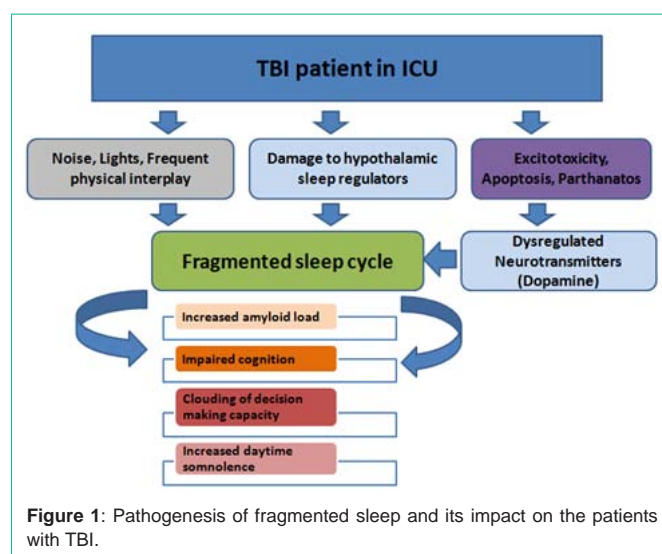


Figure 1: Pathogenesis of fragmented sleep and its impact on the patients with TBI.

including that of the neurotransmitters involved in the sleep wake mechanism.

TBI itself leads to increased amyloid load within the brain [12,13]. There are dispositions of the B-amyloid plaques within the paravascular lymphatic pathways. This clogs the clearance pathways thereby summing up the waste load. This can have a paramount effect on protracting recovery and increases odds of negative consequences to the patients.

Disrupted sleep predisposes day time somnolence, cognitive impairment, and clouding of decision making capabilities. All these protract recovery time and add up the risk of additional accidents.

Conclusion

The pathogenesis regarding the fragmented sleep cycle and its outcome on the patients with TBI has been summarized on Figure 1. Effective implementation of sleep hygiene will therefore increase the chances of better outcome in patients with TBI [10]. But it demands major change in the patient care culture currently being implemented. Nurses and the treating doctors should be educated of the importance of sleep hygiene and its impact on the patient with head injury.

Further research should provide more insights on the temporal relationships between the increased waste load (B amyloid deposits) following TBI and its impact upon the clearance mechanism (G-lymphatic pathways) especially during sleep. This can certainly be a silver lining in our quest to manage traumatic brain storm.

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