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Introduction

It is astonishing that, each and every living being undergo a passive mode in a particular time point of its circadian rhythm, which is called 'sleep'. Apparently it looks like a passive period but, several activities occur during this process like: memory consolidation, health restoration, energy strengthening etc. Though, exactly how sleep occurs is still a mystery, researchers have postulated that, sleep is needed for optimal health and wellbeing of body because, during sleep restoration and rejuvenation takes place, muscle growth increases, injured tissue gets repaired and hormone synthesis optimizes.

Therefore, any disruption in the sleep routine amounts to poor Quality of Life (QoL). "Journal of Sleep Disorders & Therapy" is dealing with multiple tantalizing findings that have been showcased in this issue; one of the more crucial findings is that Obstructive Sleep Apnea (OSA) is a crucial risk factor in cardiovascular disease (CVD). Therefore, an indirect mode of mitigating CVD is by keeping OSA in check, or by treating the OSA itself. Such a strategy opens up new avenues for curing CVD. Thus, the understanding of disease etiology can be leveraged for treating the disease.

The concept of sleep has undergone several modifications and revisions over the years, with different groups coming up with different hypotheses. The study of sleep received a big boost from technological advances such as the invention of electroencephalogram by Hans Berger in 1929, and the invention of the ARAS (Ascending Reticular Activating system). Among the more modern definitions of sleep was, 'the state where both the cortex and the thalamus are deactivated'. These days it is widely accepted that sleep is not merely a passive state in which the brain is quiescent, vital cortical functions are constantly taking place during certain stages of sleep. Normal sleep is compartmentalized into the cyclic alternations of Rapid Eye Movement (REM) and NREM (non-REM) sleep stages, of which NREM occupies ~75% to 80% of the complete sleep cycle in humans. Boniface [1], has authored a review on NREM, which discusses the connection between substrates, neurotransmitter systems and generation and behaviour of NREM sleep.

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by sporadic apneas and hypopneas, leading to nocturnal events such as hypoxemia sleep fragmentation. These events negatively affect patients' daytime activities and the patient suffers from mood-swings, fatigue, sleepiness, and reduced productivity. These impairments result in reduced quality of life (QoL). The incidence of OSA is increasing throughout the World, with the patients incurring high health care costs. Hessmann et al. [2], conducted trials on 627 patients suffering from OSA. The patients were provided with standardized questionnaires in order to assess daytime sleepiness, co-morbid disorders, and depressive symptoms. HrQoL was evaluated using the EuroQol-Instrument and influencing factors analyzed. The authors observed that OSA had a significant impact on the HrQoL.

The Oxford Sleep Resistance test (OSLER) is a useful tool for measuring the ability to stay awake. However, it is still not clear if OSLER can be used to detect physiological sleep onset as it relies on behavior such as frequency of blinking. The OSLER test works by estimating the Sleep Onset Latency (SOL) which is a measure of the time taken by the test subject to fail to respond to 7 successive visual stimuli. As of today, OSLER has not been validated against electroencephalography (EEG) recordings. Jobin et al. [3], conducted a comparative analysis of the OSLER values and the EEG-determined SOL readings (EEG-SOL) in order to analyze if OSLER values can translate into EEG readings in sleep disorder patients. The authors observed that in most trials, even though the patients failed OSLER, no sleep was recorded in EEG. Therefore, the sleep onset in the OSLER cannot be used to accurately detect EEG sleep onset.

As sleep influences both the acquisition and the consolidation of memory, sleep deprivation is frequently linked to cognitive dysfunctions. Chanana and Kumar [4], investigated the plausible mechanistics involved in the induction of cognitive dysfunction in response to sleep deprivation. Towards this, various neurological, biochemical and histopathological assays were performed in 24-hour sleep deprived mice. The authors observed significantly compromised cognitive functions in mice subjected to 24 hour sleep deprivation cycle on alternate days over a period of 21 days. These animals also demonstrated elevated aceytlcholinesterase activity, increased oxidative stress, impaired mitochondrial enzyme activity, and altered histopathology of hippocampal and thalamo-cortical regions of the mouse brain. Thus, suggesting that the interplay between the acetylcholinesterase activity, oxidative stress, and mitochondrial impairment might influence the survival of hippocampal and cortical neurons thereby precipitating sleep deprivation induced cognitive impairments.

Obesity and pulmonary hypertension (PH) frequently coexist in clinical patients. Further, multiple studies have demonstrated the higher prevalence of Sleep related breathing disorders (SRBD) in patients with pulmonary hypertension and vice versa. SRBD is an umbrella term encompassing various sleep related pathologies such as obstructive sleep apnea (OSA) and obesity hypoventilation syndrome (OHS). An increasing number of patients are afflicted with pulmonary hypertension (PH) rooted in alveolar hypoventilation. Severe PH defined by a mean pulmonary artery pressure (PAP) exceeding 25 mmHg is frequently observed in patients with OHS. In this issue, Dursunoglu [5] presented the case report of an obese man afflicted with severe pulmonary hypertension and excessive daytime sleepiness, who responded only to Oxygen plus AVAPS treatment.

Obstructive sleep apnea syndrome (OSA) and Rapid eye movement behavior disorder (RBD) have different pathophysiologies. In this issue, Park et al. [6], hypothesized that RBD patients with a history of OSA have more stable sleep compared to patients having OSA alone and sought to verify the same by cardiopulmonary coupling (CPC). Towards this, polysomnography (PSG) was conducted on 138 subjects having OSA without RBD (n=32), OSA with RBD (n=26), RBD alone (n=29), and normal control (n=51). The results supported the authors' hypothesis of RBD plus OSA patients having more stable sleep thereby, suggesting that RBD may have a cushioning effect on OSA.

Cardiovascular disease (CVD) is the leading cause of mortality in the World. Traditional risk factors underlying CVD include diabetes, hypertension, obesity, and dyslipidemia. Obesity has not only increased the rate of incidence of CVD, but also brought in an additional risk factor to the CVD risk factor spectrum: Obstructive Sleep Apnea (OSA). In this issue, Sharma et al. [7], have authored a review discussing the ever increasing pandemic of obesity and OSA, in the perspective of CVD. The authors also highlighted the therapeutic scheme for reducing OSA as a method for achieving reduced CVD.

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