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SUPPLEMENT

Slow Wave Sleep: Does it Matter?

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Sleep is a behavior observed in all living organisms and is necessary for survival. In rodents, total sleep deprivation results in a large rise in energy expenditure and consequent weight loss, despite increased food intake and — unless sleep deprivation is halted — it leads to death, usually in 2–3 weeks.[1] Death associated with chronic sleep deprivation has also been reported in other species.[2] In humans, the rare hereditary condition, fatal familial insomnia, which is associated with a progressive inability to sleep, invariably leads to death, with a time course of between 8 and 72 months.[3,4] In the absence of a disorder, total sleep deprivation does not occur over prolonged periods of time. This attests to the homeostatic drive for sleep; beyond a few days, heroic measures must be taken to keep humans awake.

Although less dramatic, the effects of sleep disturbances may be of more practical use in helping us to understand the consequences of normally occurring voluntary sleep loss and sleep loss associated with sleep disorders. In a recent large study of working adult Americans, 16% of respondents reported sleeping less than 6 hours per night on work days.[5] Chronic sleep restriction is associated with an increase in sleep propensity — even in inconvenient and dangerous situations — a deterioration of daytime performance, including memory, and a number of physiologic consequences, including adverse effects on endocrine functions and immune responses[6] and an increase in the risk of obesity and diabetes.[7]

In order to understand the consequences and effects of sleep disturbances and how these may be ameliorated, it is important to be able to analyze the neurobiology of sleep. Today, sleep is characterized using polysomnography. Using this technique, it has been shown that sleep consists of two distinct components: rapid eye movement (REM) sleep and nonREM (NREM) sleep. Standardized methods for characterizing normal sleep were first published in 1968 by Allan Rechtschaffen and Anthony Kales.[8] REM sleep is characterized by rapid eye movements and a low-amplitude, mixed-frequency EEG.[8] It accounts for approximately 18–22% of total sleep time, with the percentage of REM sleep decreasing slightly with age in adults.[9] NREM sleep is characterized by the presence of K complexes, sleep spindles, and slow wave activity (SWA), and is arbitrarily divided into four stages of sleep by Rechtschaffen and Kales, with stage 1 considered "light sleep" and stages 3 and 4 considered "deep sleep."[8]

Stage 3 sleep has been defined as an epoch of sleep containing more than 20% SWA, while stage 4 sleep has been defined as an epoch of sleep containing more than 50% SWA.[8] Together, NREM sleep stages 3 and 4 are often known as slow wave sleep (SWS). SWS is thought, by some investigators, to play an important role in cerebral restoration and recovery in humans[10,11] and to be involved in the maintenance and consolidation of sleep.[12] However, the exact nature and role of SWS are not clearly understood and there is still much to learn about SWS generation and its physiologic functions. Some of the key questions that need to be answered are: What is the relationship between SWS and other sleep parameters, such as sleep continuity, sleep duration, daytime sleep propensity, and daytime functioning? Does SWS vary with respect to age, sex, race, or basal sleep duration? What are the neurophysiologic and neuroanatomic origins of SWS? Does SWS have unique functions? Is it possible to influence SWS with pharmacologic agents and what are the consequences of doing so? Many of these important questions are addressed in the following articles, which were first presented at a satellite symposium entitled "Slow Wave Sleep (SWS): Does it Matter?" held at the 22nd Annual Meeting of the Associated Professional Sleep Societies (APSS) in Baltimore on June 10, 2008. In these articles, the authors review what is currently known about SWS. In his article, Derk-Jan Dijk discusses the nature of SWA and SWS and how they are regulated, before going on to evaluate the factors affecting SWS and the correlation of SWS with sleep continuity, sleep propensity, and daytime functioning. Giulio Tononi then delves deeper into the electrophysiologic nature of SWA, its regulation, and the relationship between SWA and synaptic strength, before Matthew Walker outlines evidence supporting a role for SWS in memory processing. Finally, James Walsh examines the impact of hypnotic agents on SWS and the consequences of enhancing SWS. Against this background, the question of whether SWS truly has a restorative and refreshing role will be considered, together with the suggestion that enhancing SWS may be of benefit to patients who experience insomnia in its many forms.

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