Neuropsychiatric Aspects of Disturbed Sleep

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Summary. Various types of sleep disruption or even disorganization are often caused by brain dysfunction secondary to traumatic brain injury, encephalitis, cerebral stroke, etc., depending on the brain regions mainly affected. Thus, lesions of the pontine tegmentum result in reduction of REM sleep, whereas lesions of the lower brainstem cause elimination of slow-wave sleep. Moreover, clinical observation shows that some neurological patients present with somnolence whereas others exhibit sleeplessness and some others a disruption of the normal circadian alternation of sleep and wakefulness. On the other hand, primary sleep disorders such as fatal familial insomnia, narcolepsy, and REM sleep behavior disorder are often associated with neuropathological or neurophysiological alterations and are accompanied by neuropsychological manifestations. Also, organicity, mainly in the form of neurodevelopmental aberrations, is strongly suggested in many cases of sleepwalking and night terrors. Finally, serious cognitive and psychosocial consequences of curtailed or otherwise disturbed sleep are often observed in sleep deprivation, chronic insomnia, obstructive sleep apnea syndromes, and narcolepsy. Therefore, to optimize the management of patients with sleep disorders, it is important to specify the exact nature and the degree of any coexisting neuropsychiatric manifestations.

Keywords. Sleep, Sleep disorders, Neurological diseases, Neuropsychiatry

Introduction

Sleep is a behavioral state that is characterized by reduced responsiveness to environmental stimuli, a high degree of immobility and changes of autonomic functions such as body temperature, heart rate, and blood pressure. The traditional concept of brain inactivity during sleep alternating with brain activation during wakefulness has been disproven since the introduction of the EEG in the study of sleep by Loomis et al. (1937), who was the first to note in the late 1930s the presence of various sleep stages. About 20 years later, a distinct sleep stage characterized by rapid eye movements (REM sleep) was discovered and associated with dreaming and muscle atonia (Aserinsky and Kleitman 1953; Dement and Kleitman 1957; Jouvet and Michel 1959).
Finally, in 1968 the standardized criteria for the identification of sleep EEG stages, still in use today, were established (Rechtschaffen and Kales 1968).

The main distinction of the sleep stages is between the REM and the non-REM (NREM) sleep. NREM sleep is further subdivided in four stages (stage I to stage IV), mainly on the basis of the progressive synchronization of the EEG that is generally characterized by theta-frequency waves of relatively low amplitude in stages I and II, gradually changing to high-amplitude delta waves in stages III and IV. Because of the preponderance of slow frequencies in the EEG during sleep stages III and IV, these two stages taken together are called slow-wave sleep (SWS). During REM sleep, the EEG is desynchronized to resemble that of wakefulness or stage I. In contrast to wakefulness and stage I, muscle tone in REM is very low and the electro-occulogram (EOG) shows the characteristic rapid eye movements. The sequence of sleep stages during the night follows a periodic pattern that is called a sleep cycle. In the first sleep cycle of nocturnal sleep of a young adult, sleep stages I to IV follow each other progressively and, about 90 min after the onset of sleep, the first REM period occurs. After the first REM period, which lasts about 15 min, stage II reoccurs and subsequent cycles resemble the first, although the duration of SWS is progressively shorter and that of stage II and REM is longer (Kales 1969; Bixler et al. 1986).

Sleep regulation is maintained through two basic mechanisms. One depends on the length of wakefulness and the accumulation of “sleepiness” and the other on the circadian (i.e., within the 24-h period) alternation of light and darkness (Borbely et al. 1983). The main brain region that is associated with the circadian regulation of sleep is the suprachiasmatic nucleus of the anterior hypothalamus, which is directly connected to the retina and is, thus, influenced by the light–dark cycle. Apart from this structure, other regions of the diencephalon are also associated with sleep regulation, whereas REM is mainly influenced by structures in the pontine brain stem (Siegel 1990). The main neurotransmitter systems implicated in sleep–wakefulness regulation are the cholinergic, which is particularly active during REM sleep, and the two aminergic, which are quite active during wakefulness; relative equilibrium between these systems is believed to produce the alternation of REM and non-REM sleep (Hobson 1983). These views, however, can be considered rather simplistic; based on many studies over the years, almost all neurotransmitters and neuromodulators as well as various neuropeptides have been found to be implicated in sleep regulation.

By its proper nature, sleep is a neurobehavioral phenomenon. Thus, as is expected, there is an intimate relationship of disturbed sleep to various neuropsychological manifestations. In this review chapter, the neuropsychiatric aspects of disturbed sleep are briefly presented, with the focus on (a) certain neurological disorders that are frequently associated with disturbances of the sleep–wake cycle; (b) brain dysfunction in specific sleep disorders; and (c) the cognitive and psychological consequences of disturbed sleep.

**Neurological Disorders Associated with Disturbances of the Sleep–Wake Cycle**

Among the neurological disorders, those frequently associated with sleep disturbances are traumatic brain injury, encephalitis, cerebral stroke, dementia, and epilepsy.