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The Role of Sleep and Sleep Deprivation in Epileptic Seizures

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Abstract

Background. Sleep is a fundamental physiological process that regulates brain network stability and neuronal excitability. In patients with epilepsy, sleep and the sleep–wake cycle are closely linked to the occurrence of epileptiform discharges and seizure susceptibility. Disturbances in sleep architecture, sleep deprivation, and irregular sleep–wake rhythms are recognized as modifiable factors influencing seizure occurrence, disease course, and diagnostic yield.

Aim of the study. The aim of this study was to summarize current evidence on the relationship between sleep physiology, sleep deprivation, and epileptiform brain activity, with particular emphasis on the mechanisms linking sleep stages to seizure generation and the clinical relevance of sleep EEG in epilepsy.

Materials and methods. A narrative review of the literature was conducted using PubMed, Google Scholar, and selected academic sources.The analysis included original research articles, clinical studies, systematic reviews, and expert consensus papers published from 2010 through 2025. Articles were selected based on their relevance to the relationship between sleep physiology and epileptiform brain activity, with priority given to peer-reviewed publications in English-language journals.

Results. The reviewed evidence shows that non–rapid eye movement (NREM) sleep, particularly slow-wave sleep, promotes neuronal synchronization and interictal epileptiform discharges, while rapid eye movement (REM) sleep suppresses epileptic activity. Sleep deprivation lowers the seizure threshold by disrupting the excitatory–inhibitory balance and destabilizing sleep–wake transitions. EEG recordings performed during sleep or after sleep deprivation significantly increase the diagnostic yield of epileptiform abnormalities. Irregular

sleep–wake rhythms and fragmented sleep have been shown to increase seizure risk, even without a reduction in total sleep duration.

Conclusion. Sleep significantly influences epileptiform brain activity and seizure occurrence through stage-dependent neurophysiological mechanisms. Sleep deprivation and irregular sleep patterns represent clinically important seizure-provoking factors, while sleep EEG and sleep deprivation EEG are valuable diagnostic tools in epilepsy. Recognizing sleep as a modifiable factor is important for epilepsy diagnosis, patient education, and comprehensive disease management.

Keywords: sleep; epilepsy; epileptiform discharges; NREM sleep; REM sleep; sleep deprivation; EEG

1. Introduction

Epilepsy represents a spectrum of neurological disorders characterized by a long-term predisposition to generate epileptic seizures and subsequent brain damage [1]. According to the International League Against Epilepsy (ILAE), epilepsy can be diagnosed after two or more unprovoked seizures separated by more than 24 hours, after a single unprovoked seizure associated with a high risk of recurrence (>60%), or upon identification of a specific epilepsy syndrome [1].

Epilepsy is one of the most common chronic neurological disorders which affects 1% of the population. The disease affects people at any age but is found to peak at two periods: during childhood and in older adults [2].

The pathophysiology of epilepsy is associated with an imbalance between excitatory and inhibitory mechanisms of the central nervous system, resulting in pathological hypersynchronization. This imbalance is mediated by alterations in ion channel function, dysregulation of GABAergic and glutamatergic neurotransmission, and reorganization of neuronal networks that contribute to the formation and persistence of epileptic foci [3].

The etiology of epilepsy is diverse and includes structural, genetic, metabolic, immunological, infectious, and unknown causes [2]. Regardless of the cause, many patients are affected by external factors such as sleep deprivation, irregular circadian rhythm, or sleep disorders, which may lower the seizure threshold and promote the occurrence of seizures.

Sleep is a physiological state essential for the proper functioning of the central nervous system. It plays a key role in regulating neuronal excitability, synaptic plasticity, memory consolidation, and maintaining brain metabolic homeostasis [4]. During sleep, significant changes in brain bioelectrical activity occur, influencing the degree of neuronal synchronization and modulating susceptibility to epileptic seizures [5].

Sleep and epilepsy are linked. Sleep influences the frequency and nature of epileptic seizures, while epilepsy itself and its treatment can lead to disturbances in sleep structure and reduced sleep quality [4,6]. Sleep disorders, such as insomnia, sleep fragmentation, irregular circadian rhythm, and sleep deprivation, are common in patients with epilepsy and are among the most important factors inducing epileptic seizures [4,7]. Sleep deprivation leads to a lowered seizure threshold, increased epileptiform activity on the EEG, and impaired seizure control [7]. Therefore, sleep has significant diagnostic and therapeutic implications in the care of patients with epilepsy.

The aim of this paper is to discuss the role of sleep and sleep disorders in the induction of epileptic seizures with particular emphasis on sleep deprivation as a major seizure-provoking factor based on the current scientific literature.

2. Physiology of Sleep: Basic Concepts

Sleep is a dynamic and regulated biological process necessary for normal brain function. During sleep, the brain remains highly active, with ongoing interactions between cortical and subcortical networks that influence neuronal excitability. Normal sleep shifts between two main states, NREM and REM sleep, which repeat in cycles throughout the night. Each state is associated with distinct patterns of brain activity. Differences in neuronal activity and synchronization between NREM and REM sleep have important implications for seizure generation [8,9]. During the night, sleep occurs in repeated cycles of about 90 minutes, each beginning with NREM sleep and followed by REM sleep. Sleep starts with the lighter stages of NREM sleep (N1 and N2) and then moves into deep slow-wave sleep (N3), after which the first REM episode appears. In the later part of the night, there is less deep NREM sleep and more REM sleep, especially toward the morning [10]. The mechanisms underlying this relationship are discussed in detail in the following sections.

3. Sleep architecture and modulation of epileptiform activity

NREM sleep is divided into three stages (N1–N3), representing progressively increasing depth of sleep. Stage N1 represents the transition from wakefulness to sleep, whereas stage N2 is

characterized by sleep spindles and K-complexes, reflecting thalamocortical regulation and reduced responsiveness to external stimuli. Stage N3, also referred to as slow-wave sleep, represents the deepest stage of sleep and is characterized by high-amplitude, low-frequency delta activity on EEG [8]. Slow-wave sleep constitutes a key physiological state linking sleep to epileptiform activity. During N3 sleep, large populations of cortical neurons engage in highly synchronized firing, producing the characteristic slow oscillations observed on EEG.

From an epileptological perspective, this widespread synchronization is of particular relevance. Increased and unstable neuronal synchrony lowers the seizure threshold and facilitates both the generation and propagation of epileptiform discharges. Accordingly, NREM sleep—and SWS in particular—constitutes the physiological state most strongly associated with activation of interictal epileptiform activity. Consistent EEG evidence demonstrates that interictal spikes, sharp waves, and spike–wave complexes occur more frequently during NREM sleep than during wakefulness, with the strongest activation observed during slow-wave sleep across a wide range of epilepsy syndromes [6,13,14,15]. Importantly, this effect reflects genuine state-dependent changes in cortical excitability rather than improved signal detection alone.

In contrast, REM sleep is characterized by low-amplitude, desynchronized EEG activity resembling wakefulness, rapid eye movements, and generalized muscle atonia. Despite behavioral sleep, REM sleep is associated with high levels of cortical activation and a distinct neurochemical environment, dominated by increased cholinergic and reduced monoaminergic activity. These neurophysiological and neurochemical features promote cortical desynchronization, increase the seizure threshold, and limit the initiation and spatial propagation of epileptic activity. As a result, seizures during REM sleep are relatively rare [4,6,9,16].

4. Sleep deprivation as a trigger for epileptic seizures

Sleep deprivation is a well-known and clinically important trigger of epileptic seizures. It refers to any reduction or disruption of normal sleep relative to physiological need. This includes not only total sleep loss, but also shortened sleep duration, frequent nocturnal awakenings, fragmented sleep, and irregular sleep–wake timing that disrupts circadian rhythms. All of these patterns can disturb the brain's ability to maintain stable neuronal activity and increase susceptibility to seizures in people with epilepsy [22].

Sleep helps maintain a balance between excitatory and inhibitory processes in the brain. During normal sleep, inhibitory systems mediated by gamma-aminobutyric acid (GABA)—provide a stabilizing influence on neuronal networks. Sleep deprivation leads to reduced GABAergic

inhibition, weakening these stabilizing mechanisms and making neurons more prone to hyperactivity and synchronization, which are key features of epileptiform activity. This reduction in inhibitory control has been observed in both clinical and experimental studies of sleep-deprived epileptic models [11,18,20]. In parallel, sleep deprivation promotes enhanced synchronization of neuronal networks. Following periods of insufficient sleep, compensatory recovery sleep often occurs with an increased proportion of NREM sleep, including slow-wave sleep, a physiological state characterized by widespread neuronal synchronization that favors the emergence of epileptiform activity [6,19].

Sleep deprivation is also frequently accompanied by circadian rhythm disruption. Long-term monitoring studies suggest that inconsistency in sleep-wake timing is more strongly associated with seizure occurrence than changes in total sleep duration, emphasizing the clinical importance of maintaining stable sleep–wake patterns in people with epilepsy [26,28,32]. Together, these findings indicate that sleep deprivation lowers seizure threshold not through a single mechanism, but via converging effects on inhibitory control, network synchronization, and circadian regulation, all of which contribute to increased epileptiform activity and seizure susceptibility [18,19,21,23].

The strong activating effect of sleep deprivation on epileptiform activity has important diagnostic implications. Recording EEG after sleep deprivation is a well-established strategy to improve the detection of interictal epileptiform discharges, in patients with suspected epilepsy and normal or inconclusive routine wake EEG recordings [5,6,22]. By increasing sleep pressure, sleep deprivation promotes rapid sleep onset during EEG recording and increases the proportion of NREM sleep, a state known to facilitate epileptiform discharges [5,7,22]. During wakefulness, epileptiform activity may remain functionally suppressed due to cortical desynchronization, stronger inhibitory control, and the neuromodulatory environment characteristic of the waking state. High levels of monoaminergic and cholinergic tone during wakefulness promote neuronal desynchronization and stabilize cortical networks limiting the emergence of hypersynchronous epileptiform discharges [9,11]. During sleep particularly NREM sleep this stabilizing influence is reduced. The transition from wakefulness to NREM sleep is associated with increased neuronal synchronization, altered neuromodulatory balance, and reduced inhibitory control, which together remove this functional “masking” effect and reveal an underlying predisposition to pathological synchronization. As a result, interictal epileptiform discharges become more frequent and more readily detectable on EEG during sleep, particularly during NREM stages [6,7,15]. Accumulating evidence indicates that the

activation of epileptiform discharges on EEG is not related to sleep itself, but rather to sleep instability and transitions between sleep stages [7].

Despite its diagnostic utility, sleep deprivation is not a benign intervention. Prolonged wakefulness may provoke clinical seizures during or shortly after EEG recording, especially in patients with poorly controlled epilepsy or heightened sensitivity to sleep loss. Therefore, the use of sleep deprivation as an activation method requires careful patient selection, appropriate monitoring, and clear safety recommendations to minimize clinical risk [5,22].

5. Sleep disorders in epilepsy

Sleep disturbances are the most common non-seizure-related complaints reported by patients with epilepsy and represent an important clinical issue that affects both seizure control and quality of life. Growing evidence shows that the link between epilepsy and sleep disorders goes beyond the short-term triggering of seizures by sleep deprivation. Instead, it involves persistent disturbances in sleep quality, sleep continuity, and circadian regulation. This relationship is bidirectional and forms a "vicious circle," in which epilepsy disrupts normal sleep, while impaired sleep, in turn, increases vulnerability to epileptic seizures [6,25–27].

Epidemiological studies show that sleep disorders affect approximately 30% to over 60% of patients with epilepsy. Symptoms such as insomnia, excessive daytime sleepiness, and sleep fragmentation occur significantly more frequently in patients with epilepsy than in the general population [6,24]. Sleep disturbances have been documented across all epilepsy types and age groups and may be present even in patients with well-controlled seizures. Moreover, alterations in sleep continuity and stability have also been observed in drug-naïve patients and during the early stages of epilepsy, suggesting that sleep disorders are not solely a consequence of long-standing disease or antiseizure medication, but may reflect intrinsic abnormalities in sleep regulation associated with epilepsy itself [17,29].

Insomnia and sleep fragmentation are the most prevalent sleep complaints in epilepsy. Patients report difficulties initiating or maintaining sleep, frequent nocturnal awakenings, and non-restorative sleep. These disturbances may result from nocturnal seizures, interictal epileptiform activity, anxiety related to seizure anticipation, or adverse effects of antiseizure medications [6,25]. Clinically, sleep fragmentation is particularly relevant because it disrupts normal sleep architecture, reduces slow-wave sleep, and increases instability of NREM sleep—conditions known to facilitate epileptiform activity and lower the seizure threshold. As a result, insomnia and fragmented sleep not only impair daytime functioning but may also directly contribute to poorer seizure control [6,25].

Nocturnal seizures exert a substantial impact on sleep quality and sleep architecture through several mechanisms. Seizures occurring during sleep frequently induce abrupt cortical arousal, autonomic activation, and transitions to wakefulness, leading to fragmentation of sleep continuity. In addition, the postictal period is often characterized by prolonged alterations in arousal regulation and delayed re-entry into stable sleep, further reducing sleep efficiency [6,27]. Patients are often unaware of nocturnal seizures, and their presence is suggested only by indirect signs such as morning headaches, confusion upon awakening, unexplained injuries, urinary incontinence, or pronounced excessive daytime sleepiness [34].

6. Long-term effects of epileptiform activity and seizures on brain function

While single, brief seizures rarely cause permanent structural brain damage, prolonged seizures or recurrent epileptic activity can lead to neuronal injury, network reorganization, and long-term functional impairment [1,2]. Experimental and clinical studies indicate that seizure-related brain damage arises from overlapping mechanisms, including excitotoxicity, metabolic stress, and pathological synaptic plasticity [3]. Sleep plays a critical modulatory role in these processes and may serve as an important neuroprotective factor by regulating neuronal excitability, synaptic plasticity, and the expression of epileptiform activity [6,25].

During seizures, excessive and synchronous neuronal firing leads to pathological glutamatergic activation and intracellular calcium overload. This process triggers excitotoxic cascades involving oxidative stress, mitochondrial dysfunction, and activation of apoptotic pathways, ultimately leading to neuronal loss [3,20]. Animal studies of prolonged seizures report selective neuronal loss in vulnerable brain regions such as the hippocampus, neocortex, and thalamus, even in the absence of systemic hypoxia [20,27].

These acute injury mechanisms are increasingly thought to contribute to subsequent, longer-term reorganization of neural circuits following recurrent seizures.

Beyond acute neuronal injury, recurrent seizures induce long-term reorganization of neural networks, including loss of inhibitory interneurons, reactive gliosis, and aberrant synaptic remodeling (e.g., mossy fiber sprouting). These changes promote network hyperexcitability and epileptogenesis and are pronounced following status epilepticus, which carries the highest risk of irreversible brain injury and is clinically associated with hippocampal atrophy, cognitive decline, and the development of drug-resistant temporal lobe epilepsy [1,27].

Brain vulnerability to seizure-induced injury is age-dependent. In the developing brain, severe or prolonged seizures may disrupt critical processes such as synaptogenesis, myelination, and

network maturation, resulting in persistent cognitive and behavioral consequences even when overt neuronal loss is limited [19,21].

Together, these findings indicate that epilepsy is not only a disorder of recurrent seizures but also a condition associated with progressive alterations in brain network integrity. Early and effective control of seizures and epileptiform activity is essential to preserve cognitive function and long-term brain stability [1,2].

7. Clinical Implications: sleep, lifestyle and seizure control

Growing evidence indicates that lifestyle-related factors, particularly sleep habits, play an important role in seizure control and overall disease management in patients with epilepsy. While pharmacotherapy remains the cornerstone of treatment, non-pharmacological strategies focus on sleep hygiene, regular circadian rhythms, physical activity, and stress management. These methods have gained increasing recognition as essential components of comprehensive epilepsy care [29,30].

7.1. Importance of sleep hygiene in epilepsy

Sleep hygiene includes behavioral and environmental practices that promote proper sleep duration, continuity, and alignment of the sleep–wake rhythm with the internal circadian clock. In patients with epilepsy, unhealthy sleep habits, including irregular sleep times, short sleep duration, evening screen exposure, or stimulant use, may worsen sleep disturbances and increase seizure risk [19,29].

Clinical reviews suggest that improving sleep hygiene may contribute to better seizure control, enhanced daytime functioning, and improved quality of life, even in patients receiving optimal antiseizure medication therapy [29,31]. Key sleep hygiene recommendations for patients with epilepsy and their clinical relevance are summarized in Table 1.

Table 1. Sleep hygiene guidelines for patients with epilepsy and their clinical significance [5,6,12,22,26,29,32].

Sleep Hygiene Element	Recommendation	Clinical Significance in Epilepsy
Consistent Sleep–Wake Schedule	Maintaining regular bedtimes and wake-up times every day, including weekends	Stabilizes the circadian rhythm and supports predictable neuronal excitability; variability in sleep timing correlates with seizure risk more than total sleep duration [26,32]
Adequate Sleep Duration	Ensuring sufficient individual sleep time (typically 7–9 hours for adults)	Insufficient sleep and chronic fragmentation increase epileptiform activity and lower seizure threshold [5,22]
Structured Pre-Sleep Routine	Engaging in relaxing, regular activities before bed (e.g., reading, relaxation techniques, breathing exercises)	Reduces cortical hyperarousal and pre-sleep anxiety, which can trigger seizures [29]
Optimal Sleep Environment	Quiet, dark, cool, and comfortable bedroom; reducing light and screen exposure	Improves sleep continuity and efficiency; reduces fragmentation that triggers nocturnal seizures [6]
Limiting Stimulants and Evening Substances	Avoiding caffeine, nicotine, alcohol, and heavy meals before sleep	Preserves proper sleep architecture, especially slow-wave sleep, important for neuronal excitability modulation [12]
Balanced Daytime Rest and Activity	Short, early naps instead of long and late afternoon naps	Maintains homeostatic sleep pressure and circadian stability, which may reduce seizure susceptibility [33]

8. Conclusion

People with epilepsy frequently experience sleep disturbances that can severely affect their seizure control and quality of life. Accumulating evidence demonstrates that sleep is not a passive state but an active neurobiological process that plays a fundamental role in regulating neuronal excitability, network synchronization, and the expression of epileptiform activity. Insufficient or disrupted sleep increases seizure susceptibility, whereas maintaining regular and stable sleep patterns supports seizure stability.

The relationship between sleep and epilepsy is bidirectional. Specific sleep stages differentially modulate epileptiform activity: non–rapid eye movement (NREM) sleep promotes neuronal synchronization and facilitates the occurrence of epileptiform discharges, while rapid eye movement (REM) sleep is associated with relative suppression of seizure initiation and propagation. Sleep deprivation and irregular sleep–wake rhythms further exacerbate cortical excitability, lower seizure threshold and increase seizure frequency. These mechanisms highlight sleep as a key modulator of seizure occurrence across epilepsy syndromes.

Taken together, these findings support a broader conceptualization of epilepsy as a dynamic network disorder, in which sleep–wake regulation constitutes an integral determinant of seizure susceptibility and long-term brain stability. Sleep represents a modifiable and clinically meaningful factor in epilepsy management. Routine assessment of sleep quality, sleep regularity, and comorbid sleep disorders should be integrated into epilepsy care alongside pharmacological treatment.

Addressing sleep-related factors through sleep hygiene education, lifestyle interventions, and targeted treatment of sleep disorders offers a low-cost, non-invasive strategy with potential benefits extending beyond seizure reduction, including improvements in cognitive functioning, emotional regulation, and overall quality of life.

Future research should focus on further elucidating the mechanisms linking sleep regulation to epileptogenesis and seizure dynamics, with particular emphasis on individualized sleep–seizure relationships identified through long-term EEG monitoring, wearable sleep technologies, and chronobiological approaches.

Disclosure

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