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Perspective

Inter-relationship between Sleep and Epilepsy

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DESCRIPTION

Sleep and epilepsy are interrelated and are closely related with each other since antiquity. Until 1980s, Polysomnography (PSG) was primarily used to clarify diagnostic questions about epilepsy. By exploiting the potential capacity of sleep to activate subtle or muted paroxysmal abnormalities during wakefulness, and to avoid inducing latent seizures by central nervous system stimulants, clinical environments dedicated to Electroencephalography (EEG) were converted into rudimentary and temporary sleep laboratories. However, the patient only had to fall asleep to reach the deepest stages of non-rem sleep, so recording was limited to 30-60 minutes, or up to an hour and a half, to attempt rapid capture. The same is true for the Rapid Eye Movement (REM) stage. Even today, especially in the field of pediatrics, video EEG is usually performed in the morning after sleep deprivation when epilepsy is suspected.

Sleep has a profound effect on epilepsy, non-REM sleep promotes epilepsy activity, and REM sleep suppresses it. Evidence of the effects of sleep on epilepsy exists not only in the structure of sleep, but also in its microstructure. Analysis of the Cyclic Alternating Pattern (CAP), an EEG marker of unstable sleep, shows that epileptic activity does not increase uniformly during non-rem sleep, but increased epileptic activity is a CAP-A1 subtypes that consist of recurrent EEG bursts of slow-wave activation.

In clinical practice, it is generally accepted that lack of sleep can cause seizures and increase the likelihood of detecting certain epilepsy-like abnormalities with standard EEG. This is because cortical excitability increases with time of awakening. However, this statement only applies if a particular epilepsy subtype is considered. Sleep deprivation in generalized epilepsy is known to lead to more frequent seizures, especially in juvenile myochronic epilepsy. In addition, sleep deprivation protocols aimed at inducing certain epileptic-like abnormalities with standard EEG have been most useful in general epilepsy situations. On the other hand, blind use in all forms of epilepsy or localized epilepsy did not add more value than the subsequent standard EEG. According to previous research studies, only five related studies are based on focal epilepsy. Two of the five studies showed no clear association between sleep deprivation and seizure risk. The only randomized study conducted by the epilepsy monitoring unit in 84 patients with preoperative epilepsy found no effect of sleep deprivation on the development of seizures. Interestingly, recent data on localized drug-resistant epilepsy suggest that increasing sleep time by 1.6 hours may reduce the risk of seizures in the next 48 hours by 27%.

CONCLUSION

Hours can be spend to analyzing traces to look for paroxysmal abnormalities or signal changes to determine if a patient is a carrier of epilepsy syndrome. Starting from these premises of detail and dynamic assumptions, a new frontier of research continues to investigate the effects of epilepsy on the process of circuit plasticity and memory integration, as well as bidirectional interactions between arousal and epilepsy-sensitive mechanisms that occur during or that are modulated by sleep. Overlapping and distinction of parasomnia in Non-Rapid Eye Movement (NREM), Sleep-related Hypermotor Epilepsy (SHE) and other sleep disorders is also worth revisiting from a variety of perspectives, perhaps the time is ripe to also include Sleep-Related Epilepsies (SRE) on the list of sleep disorders. Finally, close attention should be paid to the effects of epilepsy-related sleep disorders on the biological autonomic nervous system and the acute and long-term effects of Anti-Seizure Medication (ASMs) on sleep structure.

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