Loss of Slow-Wave Sleep

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A 40-year-old Hispanic American male, who weighed 165 lb and was 5’11” tall, consented to participate in a 3-day sleep protocol and was admitted to the UCLA General Clinical Research Center. The patient complained of fatigue during the day and difficulty initiating sleep, along with 3 to 4 awakenings per night. He denied snoring and having any difficulty breathing during sleep. A 2-week sleep diary confirmed his sleep-wake pattern. The patient also denied any history of head injury, epilepsy, or anoxia and the use of any over-the-counter or prescription medication use for the previous month. A diagnosis of current depression was ruled out, though he reported having been treated for depression with Paxil 2 years ago, when he took his last dose of Paxil. The patient reported alcohol consumption of 10 drinks per day, 12 days per month, with last his drink 18 days prior to study admission to the research center.

Sleep Study

Overnight polysomnography was carried out over 3 nights using a computerized system (Sonomologica, Flaga hf, Medical Devices, Iceland). The first night assessed the presence of sleep apnea and nocturnal myoclonus. The patient’s apnea-hypopnea index was within normal limits (< 1.5/h), as was his periodic leg movement index (< 10/h). Oxygen saturation fluctuated between 91% and 95% throughout the night. During the third night, polysomnography showed that the patient slept for 403.5 minutes, with a sleep onset of 39.9 minutes and an overall sleep efficiency of 84.1%. Out of his total sleep time, he spent 12.1% (49 minutes) in stage 1, 61.7% (249 minutes) in stage 2, and 26% (105.5 minutes) in rapid eye movement sleep (Figure 1). No stage 3 or 4 sleep was noted. The patient experienced spontaneous arousals and microarousals throughout the night, with an index of 7.2.

What is the diagnosis?

![Figure 1](image-url) — Hypnogram delineating sleep stages scored in accordance to Rechtschaffen and Kales criteria. REM, S1, S2, S3, S4 refer to rapid eye movement sleep and sleep stages 1, 2, 3, and 4, respectively.

Disclosure Statement

This was not an industry supported study. Drs. Eljammal, Valladares, and Irwin have indicated no financial conflicts of interest.

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Alcohol dependence in protracted abstinence (> 14 days since last alcoholic drink)

People with alcohol dependence, as compared with matched controls, have evidence of loss of slow-wave sleep, decreased total sleep time, and sleep fragmentation, which persist years after abstinence. The effect of alcohol on sleep is further influenced by ethnicity: African American alcoholics have been shown to have a greater loss of slow-wave sleep than do European American alcoholics. The mechanisms that contribute to loss of slow-wave sleep in alcohol dependence are not known, although vagal tone during presleep wakefulness, as measured through heart rate variability, positively correlates with slow-wave sleep in people with alcohol dependence. Given the potential effects of relaxation techniques and other sleep hygiene practices on increasing vagal tone, it is possible that these strategies have the potential to ameliorate some of the negative effect of alcohol dependence on sleep depth. The loss of slow-wave sleep can also be rooted in a myriad of conditions. Some of these conditions include head injury, epilepsy, anoxia, depression, and aging and the first-night effect.

Though the effects of head injury or trauma on sleep have not been well documented, studies have shown that traumatic brain injury can precipitate insomnia. Furthermore, electroencephalographic (EEG) studies during wakfulness have shown EEG attenuation due to minor concussions. Epilepsy, anoxia, and depression have all been noted to cause slow-wave sleep attenuation. Moreover, as we age, sleep becomes less robust, more fragmented, and attenuated, which is part of natural EEG deterioration. Aside from these medical conditions, external environmental stressors, such as “the first-night effect” can play a role in the manipulation of sleep by decreasing the amount of slow-wave sleep.

Follow-Up

The patient was diagnosed as having alcohol dependence, according to Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria. Since he was abstinent for more than 14 days, he had entered a period of protracted abstinence, during which the possibility of acute withdrawal symptoms, such as seizures, anxiety, vomiting, hand tremor, hallucinations, autonomic hyperactivity, and psychomotor agitation, had abated. Furthermore, sleep analysis from the third night provided a good representation of the patient’s stable and normal sleep. The patient was discharged and advised to practice good sleep hygiene.

Though EEG attenuation can be caused by various conditions, importance should be placed on finding the cause because complications may arise from the underlying condition. Although many of the conditions that lead to EEG attenuation can not be fully treated and are chronic in nature, good sleep hygiene practices can improve and help to preserve sleep from further deterioration. Such hygiene practices include keeping a regular sleep-wake schedule and refraining from the following: consuming caffeinated beverages after lunch or alcohol near bedtime, smoking in the evening, forcing sleep, planning for the next day at bedtime, going to bed hungry, and exercising with 4 to 5 hours prior to bedtime.

Clinical Pearl

1. Patients with alcohol dependence commonly complain of disturbed sleep, not only during periods of drinking, but also during acute and prolonged withdrawal.
2. Patients with alcohol dependence report difficulty falling asleep and maintaining sleep.
3. Polysomnography studies show an attenuation of slow-wave sleep in patients with alcohol dependence.
4. Other pathologic causes of loss of slow-wave sleep are depression, head injury, epilepsy, and anoxia.

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