

# Hypersomnolence: Unraveling the causes

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**E**stablishing a diagnosis of hypersomnia—recurrent episodes of excessive daytime sleepiness (EDS) or prolonged nighttime sleep—requires a stepwise assessment. We describe a complex case of an older adult who presented with multiple potential causes of hypersomnolence.

**CASE REPORT**

**Persistent daytime sleepiness**

Mr. W, age 63, is a veteran with a medical history significant for severe obstructive sleep apnea (OSA), insomnia, restless leg syndrome, hypertension, and major depressive disorder. He reported long-standing EDS that was causing functional and social impairment. Mr. W's EDS persisted despite the use of continuous positive airway pressure (CPAP) therapy. A download of his CPAP compliance summary revealed both optimal CPAP adherence (>7-hour usage for 95%) and control of OSA (Apnea Hypopnea Index <5). His Epworth Sleepiness Scale (ESS) score remained at 20 out of 24. Another clinician had previously prescribed modafinil to treat Mr. W's EDS, which was presumed to be related to sleep apnea. At the time of assessment, Mr. W was taking modafinil, 200 mg every morning, without significant relief of his daytime somnolence. Laboratory results revealed normal liver function tests, electrolytes, and hormonal levels, and a urine toxicology was negative. Mr. W said he constantly rubbed his legs to ease his bilateral leg movement. He reported both sensory and motor components, and relief with movement and absence of sensations in the morning.<sup>1</sup> Gabapentin was initiated and titrated to a therapeutic dose to stabilize these symptoms.

Further contemplation led the treating clinician to investigate sleep deprivation or insomnia as potential causes of Mr. W's daytime somnolence. Mr. W also reported occasional insomnia symptoms. To probe for the culprit of daytime sleepiness, actigraphy wrist monitoring was performed and showed no persistent insomnia or circadian rhythm disturbances.<sup>2</sup> Medication reconciliation revealed Mr. W was taking 2 medications (fluoxetine and modafinil) that made him alert, but because he took these in the morning, it was unlikely that they were affecting his sleep. Upon review of his sleep habits, Mr. W's naps were rare and unrefreshing during the day and he was not drinking excessive amounts of caffeinated beverages.

The diagnostic uncertainty led the treating clinician to order a polysomnography sleep study (PSG) with Multiple Sleep Latency Test (MSLT), which revealed a mean sleep latency of 4.1 minutes with no rapid eye movement (REM) periods during his PSG nor next-day napping.<sup>3</sup> The PSG showed sleep fragmentation with a sleep efficiency of 90%. The results indicated residual sleepiness secondary to OSA.

Next, the clinician prescribed dextroamphetamine, 25 mg/d, which lowered Mr. W's

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**Disclosures**

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ESS score by 2 points (18 out of 24). The clinician presumed that if the stimulant worked, the diagnosis would more likely fit the criteria for residual sleepiness from OSA, rather than idiopathic hypersomnia (IH). Due to a lack of efficacy and adverse effects, the patient was tapered off this medication.

Mr. W reported that he experienced sleepiness during his service in the military at age 23. He also said he did not feel refreshed if he napped during the day.

To address the hypersomnia, he was prescribed off-label sodium oxybate. Sodium oxybate was efficacious and well tolerated; it was slowly titrated up to 9 g/d. After taking sodium oxybate for 2 months, Mr. W's ESS score diminished to 6. Currently, he reports no functional impairment. A repeat actigraphy showed minimal sleep fragmentation and a strong normal circadian rhythm.

**Identifying hypersomnia**

Idiopathic hypersomnia should be considered when a patient's excessive sleep or EDS are not better explained by another sleep disorder, other medical or psychiatric disorders, or the use of illicit drugs or medications.<sup>4</sup> Idiopathic hypersomnia is characterized by EDS that occurs in the absence of cataplexy and is accompanied by no more than 1 sleep-onset REM (SOREM) period on an MSLT and the preceding PSG combined. The differential diagnosis includes narcolepsy, sleep apnea, and other conditions; most importantly, insufficient sleep syndrome must be carefully considered and excluded.

In IH, evidence of hypersomnia must be demonstrated by an MSLT showing a mean sleep latency of <8 minutes or by PSG or wrist actigraphy showing a total 24-hour sleep time of >660 minutes.<sup>4</sup> A prolonged and severe form of sleep inertia, consisting of prolonged difficulty waking up with repeated returns to sleep, irritability, automatic behavior, and confusion, often occurs in IH but is not pathognomonic.<sup>4</sup>

Naps are long—often 60 minutes—and described as unrefreshing by 46% to 78% of patients.<sup>4</sup> Sleep efficiency on polysomnography is usually high (mean 90% to 94%). Self-reported total sleep time is lon-

ger than in controls and is >10 hours in at least 30% of patients.<sup>4</sup> Unfortunately, symptoms and certain objective findings of IH are not unique to the disorder and are considered ubiquitous.

For Mr. W, a diagnosis of narcolepsy was unlikely due to his MSLT results. Patients with narcolepsy have cataplexy (REM dissociation) and/or at least 2 SOREM periods on MLST, or at least 1 SOREM period on MLST in conjunction with a SOREM on the preceding PSG,<sup>4</sup> which Mr. W did not exhibit. Patients with narcolepsy typically take refreshing naps lasting 15 to 30 minutes. Although not unique to narcolepsy, common findings include hypnagogic hallucinations and sleep paralysis. Patients with narcolepsy typically do not have sleep inertia but, when seemingly awake, have lapses in vigilance sometimes in combination with automatic behavior, such as writing gibberish or interrupting a conversation with a completely different topic. Another characteristic PSG finding is moderate to severe sleep fragmentation, which may be due to associated periodic limb movements or instability in sleep/wake transitions.<sup>5</sup> Mr. W had no history of traumatic brain injury that would suggest hypersomnolence secondary to a brain injury.

Among medical conditions, OSA is the predominant cause of EDS, but this, too, was unlikely for Mr. W because the CPAP therapy reports indicated excellent chronic use and effect. His apnea/hypopnea index was low, and the lowest oxygen saturation recorded on his pre-MSLT PSG using CPAP was 93%. Subjectively, Mr. W reported no choking, gasping, or snoring while receiving CPAP therapy.

Restless leg syndrome was excluded because after receiving gabapentin, both Mr. W and his wife reported improvement in his leg movements.

Although patients with mood disorders such as depression have normal MSLT results, Mr. W reported no excessive time lying in bed awake, which patients with depression often describe as fatigue and sleepiness. In addition, Mr. W's score on



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the Clinically Useful Depression Outcome Scale indicated he was not depressed.

Mr. W's clinician prescribed off-label sodium oxybate to address his EDS. Its potential benefit in this case may be related to its activity on gamma-aminobutyric acid (GABA<sub>B</sub>) receptors and its effects in prolonging slow-wave sleep, which has restorative properties. This treatment's effectiveness in this patient was surprising and without precedent. Because the causes of IH often are not precisely defined, we do not recommend administering a trial of this medication without stepwise exclusion of other causes of sleepiness as demonstrated in Pagel's algorithm "Diagnosis

and Management of Conditions That Cause Excessive Daytime Sleepiness,"<sup>6</sup> available at [www.aafp.org/afp/2009/0301/p391.html](http://www.aafp.org/afp/2009/0301/p391.html).

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**We do not recommend administering sodium oxybate without first excluding other causes of excessive sleepiness**