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Letter to the Editor

Polysomnography in Sleep Maintenance Insomnia Patients

BARRY KRAKOW, MD, JESSICA KRAKOW, BA, and FRITZ EBERLE, MBA

Letter to the Editor in response to Russell Rosenberg's article on "Sleep maintenance insomnia: Strengths and weaknesses of current pharmacologic therapies." *Ann Clin Psychiatry* 2006;18:49–56

TO THE EDITOR:

Sleep maintenance insomnia is the most pervasive sleep complaint in the general population, and Rosenberg's timely review on deficits in pharmacologic therapies for this vexing condition highlights challenges facing primary care physicians, sleep specialists, and psychiatrists (1). As Rosenberg reports, "currently used medications fall short when it comes to safely and effectively addressing sleep maintenance problems" (1).

Why these medications fail is an essential question that must be addressed to optimize sleep therapy. Two pathophysiological models for middle-night insomnia offer some insights: (1) *hyperarousal theory*, in which the sleeping brain via innate influences or other stimuli provokes increased EEG arousal activity or lowered arousal thresholds from sleep; (2) *learned insomnia theory*, in which a brief arousal from sleep is followed by increased cognitive activity, resulting in a long waking period. Each paradigm or the combination supposedly explains sleep maintenance insomnia, yet neither pharmacologic agents nor cognitive behavioral techniques (CBT) (1), the most common therapies, cure insomnia.

We believe these strategies are only partially successful because increased arousal activity and a lowered arousal threshold from sleep may also be caused by physiological disorders of sleep respiration—sleep-disordered breathing (SDB) (2). Having worked with several thousand sleep maintenance insomnia patients, we find a sizable majority show visible sleep fragmentation due to SDB events on the EEG tracings of the polysomnogram (PSG) (2). Many breathing-related arousals immediately preceded longer bouts of sleeplessness.

We theorize that hypnotics fail because they do not decrease SDB-driven sleep fragmentation. Arousals therefore persist in hypnotic treated patients, and eventually, SDB-triggered breakthrough awakenings worsen insomnia (2). In severe cases, patients and prescribing physicians respond by increasing dosing or dosage of hypnotics, leading to dependency. Conversely, CBT lessens the cognitive activity after the arousal, leading to shorter awakenings, but CBT still does not address the underlying breathing disruption.

The two most common forms of SDB are obstructive sleep apnea (OSA) and a variant, upper airway resistance syndrome (UARS). Relationships between SDB and insomnia are not well-described, due to the lack of testing with both PSG and advanced respiratory monitoring systems in most insomnia studies. Indeed, as Rosenberg states, the American Academy of Sleep Medicine does not recommend routine sleep studies for insomnia (1). Yet, at centers using PSG to research sleep respiration in insomnia-related work, SDB rates were well above 50% in primary insomniacs, crime victims, post-menopausal women, sexual assault survivors, fibromyalgia patients, and disaster survivors. Often, UARS events were more frequent than classic apneas and hypopneas.

Authors are associated with the Sleep & Human Health Institute and Maimonides Sleep Arts & Sciences, Ltd., Albuquerque, NM, USA.

Financial Disclosure: Dr. Krakow owns and operates two commercial websites, www.sleeptreatment.com and www.nightmaretreatment.com, which market books and services for the treatment of insomnia and nightmares.

Address correspondence to Barry Krakow, MD, Sleep & Human Health Institute, 6739 Academy Road NE, Ste. 380, Albuquerque, NM 87109. E-mail: bkrakow@sleeptreatment.com

Guilleminault and colleagues' time series study of 94 UARS patients bolsters the theory of SDB as a precursor to insomnia and subsequent hypnotic use (3). Four years after initial PSG diagnosis, all 94 patients were reassessed. After the initial testing, nearly all patients' insurance carriers rejected their UARS diagnoses, payment was denied for positive airway pressure (PAP) therapy, and patients had not returned to the sleep center for care. Regarding sleep, not only had insomnia worsened, but also, use of prescribed hypnotics twice per week or more rose from 11% to 61% of the sample. Repeat PSG reconfirmed UARS or worsening to OSA in all patients (3).

Several case series and two randomized controlled trials (4,5) show that SDB treatments for insomnia patients reduce both SDB and insomnia symptoms. In our clinical work, we routinely observe that PAP therapy cures sleep maintenance insomnia. For these reasons, we include SDB in the differential diagnosis of all sleep maintenance insomnia patients and test the vast majority with PSG and advanced respiratory monitoring systems.

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