microbial colonization that occurred in this patient. When not maintained properly, they can become colonized by multiple organisms with potentially pathogenetic antigens that are readily aerosolized when the device is activated.

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REFERENCES

Sleep-related Eating Disorder as a Cause of Obstructive Sleep Apnea*

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A patient with obesity resulting from sleep-related eating disorder demonstrated signs and symptoms of obstructive sleep apnea (OSA). Incarceration restricted access to food during the night, leading to weight loss and clinical improvement. Release from prison allowed recurrence of unrestricted sleep-eating, recurrent obesity, and documented OSA. Successful treatment of sleep-related eating disorder can result in improvement in coexisting OSA.

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OSA = obstructive sleep apnea

Sleep-related eating disorder is best defined as the nocturnal occurrence of involuntary, often disinhibited eating during sleep; the subject is either totally unaware of the behavior or behaves in an involuntary, automatic manner despite awareness of the nocturnal eating behavior. In most reports of the syndrome, the majority of subjects are overweight based on body mass index (BMI). 1,2 Despite the well-known relationship between obesity and obstructive sleep apnea (OSA), to our knowledge, there has been no previous documentation of a relationship between nocturnal sleep-related eating disorder, consequent obesity, and development of OSA. We therefore report a patient with significant obesity on the basis of nocturnal sleep-related eating, who developed OSA. Clinical improvement of sleep apnea correlated with cessation of nocturnal eating, achieved through a drastic yet effective form of behavior modification.

CASE REPORT

The patient is a 34-year-old white man without significant medical problems or preexisting sleep disorders. There was no history of childhood sleepwalking or night terrors, nor was there a family history of parasomnias. He was involved in a motorcycle accident in 1979, suffering nasal and mandibular injury. After recuperation, the patient was noted to snore more heavily than before the accident, and would intermittently awaken with a snort. Witnessed apneas were not observed. Many of these awakenings were associated with the patient walking into the kitchen and gorging himself on food. Specifically, the patient would usually drink ½ to 1 gallon of milk at a time, with copious quantities of cookies, breads, or leftovers. He would then return to bed and fall asleep. This behavior was typically repeated two to three times per night. He reported no recollection of any nocturnal binging, and became aware of the nocturnal activity by finding bits of food in his bed or crumbs in his beard or by being observed by his wife. The sleep-eating behavior did not exist prior to the motorcycle accident.

Over the subsequent year, the patient gained 40.5 to 45 kg, which was attributed to nocturnal eating. His daytime caloric intake actually decreased in an attempt to lose weight. During this time, his wife noted progressively heavy snoring, now accompanied by episodes of struggling to breathe during sleep, lasting approximately 30 s and terminated by gasping or choking. These episodes would frequently be associated with awakenings and sleep-eating.

In 1981, the patient was incarcerated for 1.5 years, during which time he lost 36 kg. The weight loss was believed to be the direct result of the inaccessibility to food during nocturnal awakenings while the patient was in his prison cell. On release from prison, the patient's wife noted decreased snoring and a significant reduction in witnessed apneas. The nocturnal sleep-eating persisted, however, still associated with nocturnal binging, slovenly behavior while eating, and no recollection by the patient. He gradually gained approximately 40 kg, and demonstrated resumption of severe snoring and witnessed apneas.

In 1991, the patient presented to the Rhode Island Hospital Sleep Disorders Center for evaluation of the sleep-eating, snoring, and witnessed apneas. He had been diagnosed as having hypertension during the previous five years. He denied nocturnal alcohol use or illegal drug abuse. He also denied significant daytime hypersomnolence, but noted an eight- to nine-month history of impotence. The BMI at the time of evaluation was 37.5 kg/m. 2. The patient underwent full polysomnography to better evaluate his sleep disorder. Monitoring was conducted using 2 channel EEG, electro-oculogram (EOG), and submental, intracranial, and anterior tibialis electromyogram (EMG) by surface electrodes. Airflow was detected using nasal and oral thermistors. Arterial oxygen saturation was recorded with a pulse oximeter, and respiratory effort was detected by impedance bands around the chest and abdomen. The sleep study demonstrated OSA. The apnea-hypopnea index was 25 episodes per hour, associated with oxygen desaturation to 81 percent. There was no evidence of seizure activity by the two-channel EEG, nor was significant nocturnal myoclonus or REM-associated behavior observed. No abnormal nocturnal behavior was noted during the study.

The patient refused nasal continuous positive airway pressure (CPAP), and instead underwent correction of nasal septal deviation.
which resulted in decreased snoring but did not reduce the presence of witnessed apneas. The abnormal nocturnal behavior also persisted after surgery. He had not been compliant with a combination of carbidopa and levodopa (Sinemet), which was prescribed for control of sleep-eating. The patient was unavailable for follow-up before a repeated sleep study could be performed, and before any response to the combination drug product could be determined.

**Discussion**

In the largest and most extensive published report of patients with sleep-related eating disorder, clinical and polysomnographic data were gathered on 19 subjects. The characteristics of our patient's nocturnal disorder were similar to characteristics of many of the patients in that report: onset was related to acute stress of a traumatic motorcycle accident; sleep-eating was disinhibited, excessive, and slovenly; and there was no awareness or recollection of nocturnal eating by the patient. Like other reported patients with this syndrome, this patient had no evidence of a diurnal eating disorder, and in fact, many such patients attempt strict daytime dieting with or without exercise to compensate for nocturnal eating. Despite such measures, most patients with this syndrome are overweight, probably on the basis of excessive nocturnal caloric intake.

Surprisingly, there have been no reports of OSA directly resulting from sleep-related eating, despite the common occurrence of nocturnal gluttony and consequent obesity. In the report of Schenk et al., various sleep disorders coexisted in patients with sleep-related eating disorder, including classic sleepwalking, periodic movements of sleep, and narcolepsy. Only two subjects had documented OSA, both of mild severity. Body habitus was not described, and no temporal relationship was reported between onset of sleep apnea and possible weight gain from sleep-eating.

The patient described in this report demonstrates a clear relationship between weight gain and the subsequent development of clinical characteristics of OSA. It is possible that the patient's OSA may have resulted directly from nasal and mandibular injury causing increased upper airway resistance. Arousal from sleep apnea could then precipitate atypical nocturnal behavior, including sleep-related eating, with subsequent weight gain. This scenario is unlikely, however, as overt witnessed apneas were not observed until significant weight gain had occurred. Furthermore, a brief prison term, with restriction of the patient's access to food during the night, resulted in significant weight loss and clinical resolution of OSA. Once freed from the confines of the jail cell, sleep-eating behavior resumed, weight gain recurred, and OSA developed, documented by overnight sleep monitoring. Therefore, this patient's course suggests that sleep-related eating resulted initially from stress and trauma of the motorcycle accident. This then led to significant weight gain and to the development of OSA. The actual causal relationship between OSA and sleep-related eating would have been better resolved had the patient been compliant with specific treatment for either disorder (nasal CPAP or drug combination, respectively). For example, resolution of OSA following successful suppression of sleep-related eating with the drug combination and subsequent weight loss would suggest the atypical nocturnal behavior as the primary disorder. Such follow-up was not available in this patient's case.

Drastic behavior modification for the current patient in the form of incarceration achieved total control of caloric intake, with substantial weight loss and clinical remission of OSA. These results would probably not have been the case with daytime dietary indiscretion, which could easily have persisted despite imprisonment. Such stringent control over nocturnal eating is obviously impractical, even under more voluntary conditions. Similar success may also be achieved in a more clinically appropriate manner by using pharmacologic agents. Benzodiazepines have been found useful, similar to the experience in sleepwalking. Dopaminergic agents have also been found useful in altering food consumption patterns both clinically and in animal studies.

In summary, patients with sleep-related eating disorder should be carefully screened for coexisting sleep apnea. We propose that sleep-related eating may be the primary disorder, leading to extreme weight gain and the development of OSA. In such patients, supervised pharmacologic treatment of sleep-eating would then achieve improvement in weight gain and related OSA. Because benzodiazepine use could possibly worsen OSA by preferentially reducing pharyngeal muscle tone, dopaminergic agents should be the initial treatment of choice. As sleep-eating behavior may respond dramatically to such pharmacologic and/or behavior modification therapy, certain patients with sleep-related eating disorder may demonstrate improvement in comorbid OSA as well as in the primary somnambulistic disorder.

**References**