# Clinical and Polysomnographic Features of Sleep-Related Eating Disorder

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***Background:*** Sleep-related eating disorder is a recently described clinical syndrome that combines characteristics of both eating and sleep disorders. Nocturnal partial arousals are followed by rapid ingestion of food and subsequent poor memory for the episode. Only two case series examining this disorder have been published, and both are from the same sleep disorders center in a general hospital.

***Method:*** The author describes 23 consecutive cases of sleep-related eating disorder that presented to the Sleep Disorders Center at McLean Hospital. All patients were administered a standardized clinical sleep disorders evaluation followed by a semistructured interview to elicit information regarding characteristics of sleep-related eating disorder. Polysomnographic evaluation was performed on all patients with clinical histories of sleep-related eating disorder.

***Results:*** Eighty-three percent (N = 19) of the 23 patients were female. For most of the patients, the disorder had begun in adolescence (mean ± SD = 21.6 ± 10.9 years) and had been chronic, with a mean duration of 15.8 ± 11.2 years. Nearly all patients reported eating on a nightly basis (1–6 times per night), and all episodes followed a period of sleep. All patients described their eating as "out of control," and two thirds stated that they "binged" during the night. Over 90% (21/23) reported their state at the time of nocturnal eating as "half-awake, half-asleep" or "asleep," and over 90% reported "consistent" or "occasional" amnesia for the event. Nearly half (11/23) of the sample were given a polysomnographic diagnosis of somnambulism. Thirty-five percent (8/23) had a lifetime eating disorder diagnosis.

***Conclusion:*** Sleep-related eating disorder appears to be a relatively homogeneous syndrome combining features of somnambulism and daytime eating disorders. However, no current nosology accurately characterizes these patients. Physicians should be aware of the existence of the disorder and the value of referring patients with sleep-related eating disorder to a sleep disorders center.

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Sleep-related eating disorder is a recently described clinical syndrome[1] that combines characteristics of both eating and sleep disorders. The behavior consists of partial arousals from sleep, usually within 2 to 3 hours of sleep onset, and subsequent ingestion of food in a rapid or "out of control" manner. Individuals will at times eat unusual combinations of food, inedible substances, or eat in a sloppy manner, and will later describe their state of consciousness at the time as "half-awake" or "asleep." If they are interfered with during the episode, they may become agitated or angry. Recollection for the episode the following morning is frequently significantly impaired. Thus, the behavior combines the compulsive eating of bulimia nervosa or binge-eating disorder with the disordered arousal, confusional behavior, and amnesia of a parasomnia.

Investigations of both eating disorders and sleep disorders populations have found notable rates of sleep-related eating disorder. We administered a self-report questionnaire to 132 outpatients and 24 inpatients with eating disorders and found that 9% of the former and 17% of the latter described eating during nocturnal confusional arousals.[2] Gupta[3] interviewed 32 female patients with bulimia and reported that 10 had nocturnal binge eating at least 2 to 3 times per month. The behavior was described as "purposeful," but with partial amnesia, and was worse during exacerbation of their daytime eating disorder. Most had histories of parasomnias, but no polysomnography was performed. Other small case series[4] have also described patients with a daytime eating disorder and nocturnal eating.

Schenck et al., in two separate reports,[5,6] reviewed over 7 years' worth of patients presenting to a sleep disorders clinic and found 38 patients with sleep-related eating disorder (0.5% of all referrals in the initial sample). Over 70% of patients with sleep-related eating were given a diagnosis of sleepwalking, 13% had restless legs syndrome/periodic leg movements of sleep, and 10% had obstructive sleep apnea, all based on polysomnographic data. Only

5% had a diagnosis of a current or past eating disorder (all anorexia nervosa).

I assessed the clinical and polysomnographic features of 23 consecutive patients with sleep-related eating disorder in an attempt to better describe the syndrome and its association with both eating and sleep disorders.

## METHOD

Between July 1991 and July 1996, 23 patients were seen at the McLean Hospital Sleep Disorders Center who were given a diagnosis of sleep-related eating disorder. All patients were referred for evaluation of a sleep disorder and were administered a standardized clinical sleep disorders evaluation followed by a semistructured interview to elicit information regarding characteristics of sleep-related eating disorder. Fourteen patients also completed the Pittsburgh Sleep Quality Index[7] and a sleep diary for 1 week prior to evaluation. In many cases, the patient's bed partner or members of the family of origin were also interviewed. Current and lifetime DSM-III-R[8] clinical diagnoses were noted from the admissions and discharge summaries for inpatient referrals from McLean Hospital (N = 5). Thorough psychiatric evaluations, including questions relevant to DSM-III-R-defined eating disorders, were performed on all patients by the author, a board-certified psychiatrist.

Polysomnographic evaluation was performed on all patients with clinical histories of sleep-related eating disorder. This consisted of electroencephalography (EEG) ($C_4$–$A_1$ and $O_1$–$A_2$), submental and anterior tibialis electromyography (EMG), electrocardiogram (ECG), and, where clinically indicated, respiratory monitoring (nasal and oral airflow, abdominal and chest effort, and oximetry). All patients were videotaped. Patients were encouraged to bring food with them to the sleep laboratory, so that they could have it at the bedside during the night. Patients were put to bed within 1 hour of their customary bedtime and had at least 6½ hours of total recording time, prior to spontaneous awakening in the morning. Polysomnographic studies were scored according to standard criteria.[9]

## RESULTS

Characteristics of the 23 patients are presented in Table 1. Nearly all were women, the majority of whom were in their mid-20s to mid-40s. For most of the patients, the disorder had begun in adolescence or early adulthood, and over 60% had had sleep-related eating disorder for over 12 years. For 3 patients, onset had occurred before the age of 10. Most of the patients were unable to identify precipitating events for their nocturnal eating, although sleep-related eating began in 2 patients after diets, in another after sobriety from alcohol, and in 1 after the death of her father. Sixty-one percent (14/23) had sleep-related eating disorder as their primary reason for referral to the sleep disorders center, whereas the remaining 39% (N = 9) described it in the course of the evaluation for another primary complaint (2 for somnambulism, 3 for insomnia, 1 for fatigue, 1 for nightmares, and 2 for sleep apnea). Six patients had one or more first-degree relatives with sleep-related eating disorder.

Table 1. Characteristics of 23 Patients With Sleep-Related Eating Disorder

| Characteristic | Value |
|---|---|
| Female, % (N) | 82.6 (19) |
| Age at presentation (mean ± SD y) | 37.0 ± 8.4 |
| Age at onset (mean ± SD y) | 21.6 ± 10.9 |
| Body mass index (kg/m$^2$) | 27.4 ± 7.5 |
| History of parasomnia, % (N) | 78.3 (18) |
| Family history of sleep-related eating disorder, % (N) | 26.1 (6) |
| Frequency of sleep-related eating (mean ± SD episodes/wk) | 13.0 ± 8.7 |
| State of alertness, % (N) | |
| Mostly asleep | 43.5 (10) |
| Half-awake, half-asleep | 47.8 (11) |
| Mostly awake | 8.7 (2) |
| Amnesia, % (N) | |
| Consistent | 47.8 (11) |
| Occasional | 43.5 (10) |
| Rare | 8.7 (2) |
| Daytime eating disorder, % (N) | 34.8 (8) |
| Bulimia nervosa | 13.0 (3) |
| Anorexia nervosa | 13.0 (3) |
| Binge-eating disorder | 8.7 (2) |
| "Bingeing" during nocturnal eating, % (N) | 65.2 (15) |

Seventy percent (16/23) of the patients were taking psychotropic medications at the time of the consultation and during the sleep study, and these may have influenced the clinical presentation as well as the polysomnographic results. Thirty-nine percent (N = 9) of the whole sample were taking benzodiazepines, 61% (N = 14) antidepressants, and 13% (N = 3) antipsychotics. Thirty-nine percent (N = 9) of all patients were taking multiple medications.

Nocturnal eating was characterized as unremitting and driven. Nearly all patients ate on a nightly basis from 1 to 6 times per night, although the exact number of episodes varied from night to night, and uncertainty regarding the exact number of episodes per night was frequent. All patients reported that nocturnal eating followed a period of sleep, although time of night varied between and within patients. Two thirds of patients self-described their current nocturnal eating as "bingeing," whereas 2 had "binged" only in the past. The remaining patients reported that their eating was "out of control," but did not describe it as a binge. Only 1 patient identified any history of purging after sleep-related eating. All patients reported eating mostly "sweets," rarely cooking, and commonly leaving the area messy. One patient had attempted to consume fingernail polish; a number had eaten frozen foods. Unusual combinations of food (e.g., peanut butter and steak) were sometimes reported. Morning anorexia or restriction of

daytime eating was common as a response to nocturnal eating. No differences in the frequency of sleep-related eating or the presence of bingeing between medicated and unmedicated patients were observed.

Given the limitations of the language in describing states of awareness, patients were restricted to the choices of "mostly asleep," "half-awake, half-asleep," or "mostly awake" to describe their level of consciousness during the nocturnal eating episodes. Nearly all (21/23) of the patients reported that they were "mostly asleep" or "half-awake, half-asleep." These states were variable from episode to episode within individual patients, but the modal state during nocturnal eating could be readily identified by all patients. Five reported being told that they were very irritable if prevented from eating. Nearly all of the patients described at least occasional amnesia for the episodes, and the types and quantity of food consumed often had to be reconstructed from evidence upon awakening in the morning (e.g., missing food, messy kitchen, food in bed). One patient had amnesia only while taking lorazepam. Otherwise, no differences in level of consciousness during sleep-related eating or subsequent amnesia were observed between medicated and unmedicated patients. Patients who described themselves as more asleep during the episode were more likely to more consistently report amnesia: 70% (7/10) of those who reported being asleep during sleep-related eating consistently described amnesia, while only 36% (4/11) who stated they were "half-awake, half-asleep" consistently described amnesia.

Eight patients had a current or past history of a daytime eating disorder: 3 with anorexia nervosa, 3 with bulimia nervosa, and 2 with binge-eating disorder. Forty-two percent of the women (8/19) and 25% of the men (1/4) were overweight (body mass index > 25.0). Most of the patients (78%, 18/23) had preexisting histories of sleep disorders (9 with somnambulism alone, 3 with nocturnal enuresis until their early teens, 2 with both somnambulism and night terrors, 2 with night terrors and nocturnal enuresis, and 2 with all three behaviors). Nearly all patients (N = 17) discontinued other sleepwalking behaviors once sleep-related eating became established as a regular occurrence.

All but 1 of the patients had abnormal polysomnographic results. Eleven patients had multiple abrupt awakenings from slow-wave sleep (mean = 5.5; range, 3–11) with or without overt behavioral manifestations and were thus given diagnoses of somnambulism. Of the 7 patients who were medication-free, 4 were diagnosed with somnambulism. Five patients aroused from non-REM sleep (3 from slow-wave sleep) and ate food, which they had brought with them. All eating that occurred in the laboratory setting was during EEG-defined wakefulness. Two patients had awakenings with a scream or intense motor activity (1 from stage II and the other after a late morning episode of stage IV).

Table 2. Polysomnographic Features of 23 Patients With Sleep-Related Eating Disorder

| | Medication-Free (N = 7) | | Medicated (N = 16) | |
|---|---|---|---|---|
| Variable | Mean | SD | Mean | SD |
| Total sleep time (TST) (min) | 392.4 | 51.9 | 352.4 | 52.7 |
| Latency to stage 1 (min) | 9.3 | 5.8 | 10.5 | 10.1 |
| Sleep efficiency (TST/total recording time) | 88.3[a] | 8.8 | 80.2 | 8.6 |
| Stage 1 % | 11.0 | 4.0 | 16.5 | 10.1 |
| Stage 2 % | 48.9 | 6.1 | 55.0 | 15.6 |
| Stages 3 and 4 % | 21.6 | 10.0 | 12.5 | 10.0 |
| Stage REM % | 20.4 | 6.6 | 12.4 | 9.9 |
| REM latency (min, time from Stage 1 to REM) | 115.1 | 60.1 | 142.7 | 91.5 |
| Arousal index (arousals/h) | 18.3 | 5.8 | 22.0 | 12.6 |
| Slow-wave sleep awakening index[b] | 2.8 | 1.6 | 2.5 | 1.5 |
| Apnea + hypopnea index (apnea + hypopnea/h of sleep) | 4.6 | 8.3 | 10.8 | 25.5 |
| Periodic leg movement index (periodic leg movements/h of sleep) | 2.4 | 4.7 | 9.2 | 12.1 |

[a]Significantly different from medicated group, t = 2.1, p = .05.
[b]Number of direct excursions from stages 3 or 4 to > 15 s awake/h of stages 3 and 4.

In general, patients had little difficulty falling asleep but had significant sleep disruption (Table 2). Eight had sleep efficiencies below 80%, mostly comprised of nocturnal awakenings (only 1 had a sleep-onset latency longer than 20 minutes). Arousal indices ranged from 6 to 53 per hour with an average of 21/hour. Six patients had periodic leg movements of sleep, with indices ranging from 12 to 30 events (with arousal) per hour. Three patients had sleep apnea (apnea/hypopnea indices greater than 10 events/hour), although only 1 patient had significant apnea, with an apnea/hypopnea index of 90 events/hour. One patient had a sleep-onset REM episode.

Follow-up treatment data were not available for most of the patients. However, in those patients who were followed up, medication treatment of sleep-related eating disorder was only of mixed benefit. Two had permanent improvement with clonazepam, 1 additional patient had only temporary benefit. One patient had temporary improvement with trazodone. One patient had temporary benefit from fluoxetine. Three had improvement with carbidopa-levodopa, although improvement was only temporary in 1 patient. One improved with behavioral measures (blocking the door to the kitchen). One worsened with clomipramine, and another worsened with phenelzine.

## DISCUSSION

These data confirm and extend many of the features of sleep-related eating disorder described in previous studies. Similar to the population in previous reports,[3,5,6] our patients were predominantly female, with histories of other parasomnias (especially somnambulism), who de-

scribed chronic, compulsive nocturnal eating in a state usually described as partially or fully asleep. Most patients demonstrated primary sleep disorders on the basis of polysomnography, particularly multiple slow-wave sleep awakenings and/or periodic leg movements of sleep. The similarity of our sample to those previously published suggests that sleep-related eating presents clinically as a relatively homogeneous disorder, although it may have multiple possible etiologies.

There is no diagnostic category in the DSM-IV that accurately characterizes these patients. The presence of a reduced level of consciousness with amnesia is consistent with the DSM-IV description of sleepwalking disorder (307.46). However, most of our patients exhibited no somnambulistic behaviors other than nocturnal eating, and although eating is listed in DSM-IV as one of the possible activities that the somnambulist may perform, there is no mention of this behavior as a stereotyped form of sleepwalking. The preponderance of females in our sample, as well as in previous published cases, is inconsistent with the roughly equal sex distribution of somnambulism.[10] On the other hand, the age at onset of the behavior in the teens or early 20s in the majority of our sample is consistent with findings in other studies of parasomnias,[11,12] although it would be considered late by traditional standards.[13] Similarly, the description of multiple episodes per night of sleep-related eating is consistent with somnambulism.[14]

The behavior also meets many of the criteria for binge-eating disorder (a proposed diagnosis for DSM-IV), such as binge eating, lack of control over the episode, and associated distress. The absence of compensatory activities (e.g., purging) is also consistent with binge-eating disorder and distinguishes the syndrome from bulimia nervosa. Over one third of my patients had a history of daytime eating disorders. The finding that sleep-related eating disorder appears to occur in highest frequency in patients with eating disorders[2] suggests that the two disorders may have a common underlying pathophysiology. Similarly, the exclusiveness of eating during the nocturnal episodes suggests the importance of this behavior for these patients. For some patients, sleep-related eating disorder may, in fact, be the concurrence of a diurnal eating disorder with somnambulism. For other patients, the altered control present during somnambulism might permit the expression of an otherwise latent or subclinical eating disorder. Further work to delineate eating attitudes in this population is under way.

The familiality of sleep-related eating disorder demonstrated in my series, as well as in that of Schenck and colleagues',[6] is consistent with both eating disorders and somnambulism. Somnambulism is thought to be at least 10 times more prevalent in first-degree relatives of sleepwalkers than in the general population.[15] Similarly, both anorexia nervosa[16] and bulimia nervosa[17] have strong familial representation.

The International Classification of Sleep Disorders (ICSD)[13] includes a disorder called *nocturnal eating (drinking) syndrome*, which is characterized by rapid binge eating occurring either before sleep onset or immediately after an arousal from sleep. In distinction to sleep-related eating disorder, the individual maintains full awareness during the episode and has no subsequent amnesia for the nocturnal eating episode.[18–20] Primary sleep disorders and eating disorders are specifically excluded by this diagnosis. Few studies of nocturnal eating syndrome have inquired as to the history of parasomnias, and few patients have been tested polysomnographically. It is not even clear from some reports whether patients have been questioned as to their level of alertness during the episode or whether there was subsequent amnesia (other than the study of Spaggiari et al.[18]). Obesity is commonly seen in such patients, and the frequency of daytime eating disorders depends on the source of the sample. Similar to my findings on sleep-related eating disorder,[2] rates of nocturnal eating syndrome have been found to be below 1% in overweight controls without eating disorders and about 15% in patients with binge-eating disorder.[21]

Whether sleep-related eating disorder and nocturnal eating syndrome are two distinct nocturnal eating disorders or on a continuum of severity is unclear. Both the rapid, compulsive binge eating associated with nocturnal awakenings and the chronic course are similar in the two disorders. However, the major differences in the state of alertness and associated level of recall distinguish the two syndromes. In fact, many of my patients reported significant variability in their level of alertness during nocturnal eating, between individual episodes both within and between nights. For instance, they might report being partially awake during one of the night's episodes, and asleep and amnesic for another episode the same night; or they might have had a preponderance of fully asleep episodes in their teens or 20s and become more alert during nocturnal eating as they got older. Thus, sleep-related eating disorder and nocturnal eating syndrome may be similar syndromes with varying degrees of awareness based on the extent of arousal from sleep.

Another sleep disorder that shares some features with sleep-related eating disorder is the Kleine-Levin syndrome.[22] In this disorder, patients (predominantly adolescent males) will exhibit attacks of profound sleepiness, hypersexuality, irritability, and overeating lasting days to weeks. The distinctions between sleep-related eating disorder and the Kleine-Levin syndrome are clear, and no patients met criteria for this disorder.

Polysomnography was of value in documenting primary sleep disorders in the majority of patients. Roughly 50% (11/23) of my sample were given a laboratory diagnosis of somnambulism, based on an excess number of awakenings from slow-wave sleep with or without overt behavioral manifestations.[12,23,24] This figure is somewhat

less than the 70% of Schenck and colleagues' cumulative sample[6] of sleep-related eating disorder patients who were given this diagnosis. This disparity may be due to the different sources of patient referrals and locations of sleep laboratories: ours in a psychiatric hospital and theirs in a general hospital.

The underlying pathophysiology of sleep-related eating disorder is unclear. One hypothesis is that an internally generated stimulus (e.g., periodic leg movement, apnea) may produce a partial arousal, which, if occurring at the right time (probably non-REM sleep), in a predisposed individual, may be associated with a nocturnal eating episode. This mechanism has been proposed as a cause of somnambulism[23] and may similarly produce sleep-related eating. Of note, about 25% of my sample and 20% of Schenck and colleagues' patients had periodic leg movements of sleep. One of my patients had an episode of periodic leg movements of sleep that was clearly associated with an arousal from stage 4 sleep and a subsequent episode of nocturnal eating. Treatments directed toward decreasing the numbers of periodic leg movements of sleep or their associated arousals (levodopa) have been among the most successful treatments for sleep-related eating disorder.[6]

The differential diagnosis of nocturnal eating consists of sleep-related eating disorder, in which nocturnal eating occurs with altered levels of awareness; nocturnal eating syndrome, in which nocturnal eating occurs during full alertness; binge-eating disorder or bulimia nervosa with nocturnal eating, in which nocturnal eating with full alertness is combined with a daytime eating disorder; dissociative disorder with nocturnal eating, in which an individual eats at night and has altered levels of awareness, but has other evidence of a dissociative disorder (e.g., posttraumatic stress disorder, multiple personality disorder); and Kleine-Levin syndrome, in which compulsive eating is combined with hypersexuality, behavioral abnormalities, and excessive somnolence.

In summary, current nosologies address either the sleep disorder or the eating disorder features of sleep-related eating disorder. None of them combines the confusional arousal aspect (the sleepwalking) with the compulsive eating characteristics, which together appear to be the hallmark of sleep-related eating disorder. In this way, sleep-related eating disorder has characteristics of both an eating disorder and a sleep disorder, without being limited to one or the other. For these reasons, I believe that sleep-related eating disorder should be afforded diagnostic recognition independent of both somnambulism and daytime eating disorders.

The conclusions of the current study are limited by a number of factors. Many of my patients were either inpatients at a psychiatric hospital or referred by psychiatrists. This may have increased the associated psychopathology of the patients, and, in particular, may overestimate the percentage of sleep-related eating disorder patients with comorbid eating disorders. In fact, over one third of my patients had a lifetime history of a daytime eating disorder, whereas only 5% of Schenck and colleagues' sample had such a diagnosis. The fact that 60% of the patients were referred for evaluation of sleep-related eating disorder as their primary complaint may similarly bias the sample toward a more severely affected group. However, the similarity of this group to previously published samples as well as to unselected nonpatient samples[2] suggests that this is not the case. The interviews were performed by a nonblinded clinician; however, the nature of the disorder is generally so stereotyped and unusual that the results appear to be reliable. The polysomnographic data were confounded by the presence of psychiatric medications in over two thirds of the sample. Most antidepressants prolong REM latency and reduce the amount of REM sleep.[25] Similarly, benzodiazepines suppress slow-wave sleep.[26] Although I found few significant differences between the medication-free and medicated patients, my sample of unmedicated patients was small. The presence of a non–sleep-related eating disorder comparison group may have shed more light on the polysomnographic data.

Despite these limitations, this study demonstrates that sleep-related eating disorder is a relatively homogeneous disorder, associated with significant chronicity and morbidity. Physicians should be aware of the existence of the disorder and the value of referring patients with sleep-related eating disorder to a sleep disorders facility.

*Drug names:* carbidopa-levodopa (Sinemet), clomipramine (Anafranil), clonazepam (Klonopin), fluoxetine (Prozac), levodopa (Larodopa), lorazepam (Ativan and others), phenelzine (Nardil), trazodone (Desyrel and others).



## REFERENCES

1. Schenck CH, Mahowald MW. Review of nocturnal sleep-related eating disorders. Int J Eat Disord 1994;15:343–356
2. Winkelman JW, Herzog D, Atala K, et al. Epidemiology of sleep-related eating disorder in psychiatric and nonpsychiatric populations. Presented at the 147th annual meeting of the American Psychiatric Association; May 25, 1994; Philadelphia, Pa
3. Gupta MA. Sleep-related eating in bulimia nervosa: an underreported parasomnia disorder [abstract]. Sleep Research 1991;20:182
4. Guirguis WR. Sleepwalking as a symptom of bulimia. BMJ 1986;293: 587–588
5. Schenck CH, Hurwitz TD, Bundlie SR, et al. Sleep-related eating disorders: polysomnographic correlates of a heterogeneous syndrome distinct from daytime eating disorders. Sleep 1991;14:419–431
6. Schenck CH, Hurwitz TD, O'Connor KA, et al. Additional categories of sleep-related eating disorders and the current status of treatment. Sleep 1993;16:457–466
7. Buysse DJ, Reynolds CF, Monk TH, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Research 1989;28:193–213
8. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised. Washington, DC: American Psychiatric Association; 1987
9. Rechtschaffen A, Kales A. A Manual of Standardized Terminology: Techniques and Scoring System for Sleep Stages of Human Subjects. Los Angeles, Calif: Brain Information Service/Brain Research Institute, U of Ca-

lif; 1968
10. Thorpy MJ. Disorders of arousal. In: Thorpy MJ, ed. Handbook of Sleep Disorders. New York, NY: Marcel Dekker; 1990:531–549
11. Schenck CH, Milner DM, Hurwitz TD, et al. A polysomnographic and clinical report on sleep-related injury in 100 adult patients. Am J Psychiatry 1989;146:1166–1173
12. Blatt I, Peled R, Gadoth N, et al. The value of sleep recording in evaluating somnambulism in young adults. Electroencephalogr Clin Neurophysiol 1991;78:407–412
13. Diagnostic Classification Steering Committee. International Classification of Sleep Disorders: Diagnostic and Coding Manual. Rochester, Minn: American Sleep Disorders Association; 1990
14. Kavey N, Whyte J, Resor SR, et al. Somnambulism in adults. Neurology 1990;40:749–752
15. Kales A, Soldatos CR, Bixler EO, et al. Hereditary factors in sleepwalking and night terrors. Br J Psychiatry 1980;137:111–118
16. Strober M, Lampert C, Morrell W, et al. A controlled family study of anorexia nervosa: evidence of familial aggregation and lack of shared transmission with affective disorders. Int J Eat Disord 1990;9:239–253
17. Kendler KS, MacLean C, Neale M, et al. The genetic epidemiology of bulimia nervosa. Am J Psychiatry 1991;148:1627–1637
18. Spaggiari MC, Granella F, Parrino L, et al. Nocturnal eating syndrome in adults. Sleep 1994;17:339–344
19. Aronoff NJ, Geliebter A, Hashim SA, et al. The relationship between daytime and nighttime food intake in an obese night-eater [abstract]. Sleep Research 1992;21:26
20. Stunkard AJ, Grace WJ, Wolff HG. The night-eating syndrome. Am J Med 1955;19:78–86
21. Greeno CG, Wing RR, Marcus MD. Nocturnal eating in binge eating disorder and matched-weight controls. Int J Eat Disord 1995;18:343–349
22. Billiard M. Other hypersomnias. In: Thorpy MJ, ed. Handbook of Sleep Disorders. New York, NY: Marcel Dekker; 1990:353–371
23. Broughton R. Sleep disorders: disorders of arousal? Science 1968;159:1070–1080
24. Halasz P, Ujszaszi J, Gadoros J. Are microarousals preceded by electroencephalographic slow wave synchronization precursors of confusional awakenings? Sleep 1985;8:231–238
25. Sharpley AL, Cowen PJ. Effect of pharmacologic treatments on the sleep of depressed patients. Biol Psychiatry 1995;37:85–98
26. Gaillard JM. Benzodiazepines and GABA-ergic transmission. In: Kryger MH, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. 2nd ed. Philadelphia, Pa: Saunders; 1994:349–354