Sleep, 17(4):339–344

Nocturnal Eating Syndrome in Adults

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Summary: Ten adult subjects were referred to our sleep disorders center complaining of difficulty in maintaining sleep due to frequent and recurrent awakenings to eat or drink. All patients manifested more than one episode per night, characterized by compulsive food seeking and a return to sleep only after adequate food intake. Food-seeking drive was described as an urgent abnormal need to swallow food and was associated with an absence of real hunger. Six subjects showed an elective nighttime intake of carbohydrates, and in all cases only edible substances were injested. The patients were always fully awake during the episodes and could clearly recall them in the morning. Polysomnographic investigation showed low levels of sleep efficiency, a high number of awakenings and a strict relation between nocturnal eating episodes and nonrapid eye movement (NREM) sleep. The average length of each episode was 3.5 minutes. The "eating latency", that is the interval between awakening and chewing start, was shorter than 30 seconds in 50% of the episodes. No medical, hormonal or neurological disorders were found during clinical and laboratory investigations. Body mass index was abnormally high in six patients. Anorexia nervosa and bulimia were carefully excluded. Various psychiatric disturbances were found in nine subjects, who were nevertheless well-functioning adults. Concurrent dyssomniac disorders, such as narcolepsy or periodic leg movements occasionally associated with restless legs syndrome, were diagnosed in five patients. Six of the seven subjects treated with d-fenfluramine (15–30 mg daily at bedtime) and followed for 6–15 months showed a drastic reduction in the number of nocturnal eating episodes and a decreased amount of total calories and disappearance of sweet food election. **Key Words:** Sleep—Eating disorders—Nocturnal awakenings—Compulsive behavior.

According to the recent International Classification of Sleep Disorders (1), the nocturnal eating (drinking) syndrome (NES) is characterized by recurrent awakenings associated with an inability to return to sleep without eating or drinking.

Nocturnal eating syndrome was first described in 1955 by Stunkard et al. in a group of adult obese patients with nocturnal hyperphagia, insomnia and diurnal anorexia (2). These authors suggested that in these patients the disorder could be intimately linked to the pathogenesis of their obesity and could represent a response to stressing factors (2). However, in spite of the worldwide relevance of obesity and the increasing interest dedicated to eating disorders, NES is still an underrated topic. In addition, the International Classification of Sleep Disorders states that the disorder is "primarily a problem of infancy and early childhood" (1). This paper highlights the clinical findings, polysomnographic features and therapeutic strategies for 10 subjects with adult onset of NES, in which obesity was not a mandatory feature.

Accepted for publication February 1994.
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MATERIALS AND METHODS

Within an 18-month period (October 1990–March 1992), 10 subjects (six males and four females) between 23 and 62 years of age (mean 41.1 years) were referred to our sleep disorders center complaining of recurrent nighttime awakenings associated with compulsive feeding behavior. As supplementary major complaints, four subjects reported chronic insomnia and one reported excessive daytime sleepiness. Lifetime sleep–wake schedules were investigated by means of a standardized comprehensive questionnaire.

Routine blood and urine assay; serial blood levels of glucose, insuline, cortisol and growth hormone; oral glucose tolerance tests; thyroid hormones dosage (T3-T4-TSH); and abdominal echography were performed for all subjects, and gastroscopy was performed in three patients who complained of daytime gastric discomfort. Subjects were considered overweight when body mass index (BMI) exceeded 25 in males and 23.8 in females.

TABLE 1. *Clinical features*

Patient	Sex	Age	BMI[a]	Clinical features	Promoting events	Predisposing factors	Associated complaints	Drugs
1	M	44	30.3	Two to three eating episodes per night with a marked sweet food election lasting 9 months. Normal diurnal food intake.	Diet	—	Chronic insomnia	Chronic intermittent use of benzodiazepines (10 years); discontinuous use of antidepressants (10 years)
2	F	45	23	Four to seven eating episodes per night lasting 10 years. Diurnal hyporexia.	Traumatic life event	—	Daytime fatigue	—
3	M	46	39.5	Three to four eating episodes per night lasting 16 years. Normal diurnal food intake.	—	Peptic ulcer	—	—
4	F	23	22.2	Three eating episodes per night with a marked sweet food election lasting 5 years. Diurnal hyporexia.	—	Poor sleep hygiene	Daytime sleepiness, sleep attacks and fragmented nocturnal sleep (lasting 10 years)	—
5	F	30	23.2	Two to three eating episodes per night with obsessional behaviors and a marked sweet food election lasting 5 years. Diurnal hyporexia.	Delivery	—	—	—
6	F	49	28.5	Two to three eating episodes per night lasting 2 years. Diurnal overeating.	Traumatic life event	—	Chronic insomnia (lasting 2 years)	—
7	F	32	32.1	Eight to ten eating episodes per night with a marked sweet food election lasting 10 years. Diurnal hyporexia.	Traumatic life event	—	Sleep disruption and daytime fatigue	—
8	M	62	31.5	Two eating episodes per night lasting 10 years. Normal diurnal food intake.	Shift work	Drug-induced gastritis; nightwork	Daytime fatigue	Chronic intermittent use of benzodiazepines (15 years)
9	M	40	24.2	Two to three eating episodes per night with a mild sweet food election lasting 10 years. Normal diurnal food intake.	Traumatic life event	Ernia jatus	Chronic insomnia (lasting 10 years)	Chronic intermittent use of benzodiazepines (10 years)
10	F	40	28.8	Two to three eating episodes per night with a marked sweet food election lasting 5 years. Diurnal overeating.	Traumatic life event	Poor sleep hygiene	Chronic insomnia (lasting 5 years)	—

[a] Body mass index (BMI) normal values: males 20–25; females 18–23.8.

All patients underwent neurological examination completed by enhanced computerized tomographic scan or NMR.

Psychiatric investigations including the diagnostic checklist for DSM-III-R (3) and the Eating Attitude Test (EAT-26) (4) were carried out on all patients. The Minnesota Multiphasic Personality Inventory (MMPI), the Hamilton Rating Scale for Depression (HRSD) and for Anxiety (HRSA) were administered to seven subjects.

Each subject underwent an audiovisually controlled polysomnogram in a partially soundproof recording chamber at the Sleep Disorders Center of the University of Parma. The night recording time always lasted 500 minutes. Besides electroencephalographic monitoring, the polysomnograms assessed eye movements, muscle tone, anterior tibialis muscle activity and heart rate. Patients were asked to bring food to the sleep laboratory according to their nocturnal eating habits. After each recording, all subjects filled out a multisection questionnaire for a self-evaluation of the previous night of sleep.

TABLE 2. *Psychiatric investigations*

Patient	Clinical evaluation (DSM-III-R)	MMPI	EAT-26[a]	HRSD[b]	HRSA[c]
1	Panic disorder with agoraphobia, major depression	Hysteria, depression, schizophrenia	Normal	21	100
2	Generalized anxiety disorder	nd[d]	Normal	nd	nd
3	Panic disorder with agoraphobia	nd	Normal	nd	nd
4	Normal	Depression, hypochondria, psychastenia	Normal	14	49
5	Generalized anxiety disorder	Hysteria, paranoia, social introversion	Normal	15	68
6	Panic disorder	nd	Normal	nd	nd
7	Agoraphobia without panic disorder	Hysteria, depression, psychastenia	Normal	20	116
8	Panic disorder with agoraphobia, dysthymia	Depression, hysteria, schizophrenia	Normal	20	60
9	Panic disorder	Depression, hysteria, psychastenia	Normal	29	37
10	Agoraphobia without panic disorder	Depression	Normal	16	40

[a] EAT-26 = Eating Attitude Test (short version).
[b] HRSD = Hamilton Rating Scale for Depression (normal values ≤15).
[c] HRSA = Hamilton Rating Scale for Anxiety (normal values ≤50).
[d] nd = not done.

Polysomnograms were visually scored according to standard criteria (5). The number and duration of nocturnal eating episodes were assessed as well as the total amount of calories injested during the night.

Following the recording night, seven patients agreed to be treated chronically with d-fenfluramine, 15–30 mg daily at bedtime. Throughout the treatment period, self-evaluation of nocturnal awakenings and total nighttime amount of injested food were monitored by a sleep log and a specific diary.

RESULTS

Table 1 summarizes the main clinical features. The average duration of the NES symptomatology was 7.4 years (range 9 months–16 years), with a referred mean number of eating-related awakenings per night of 3.4 (range 2–10). The mean age of onset of the disorder was 33.7 years (range 18–52). In eight patients, possible promoting factors such as traumatic life events, nightwork, delivery or strict diet could be related to the onset of the disorder. Poor sleep hygiene was detected in two patients.

All subjects reported being fully awake during the nocturnal episodes and exhibiting compulsive behaviors to obtain food, which were sometimes associated with aggressive conduct against constraints. The food-seeking drive was described as an urgent abnormal need to swallow food, with an absence of real hunger. Only edible substances were chosen. Striking amounts of food could be injested during the night. One patient described her typical nocturnal meal as composed of six slices of cake, two fruits, one piece of cheese and two cups of milk. Six subjects showed an elective nighttime intake of carbohydrates and sweet food. In two patients, the nocturnal eating behavior ceased whenever they slept away from home. Four subjects complained of hyporexia and two of overeating as being abnormal daytime dietary habits. A long history of taking benzodiazepines or antidepressant drugs was reported by three patients.

Medical data. No metabolic or hormonal disorders were identified, except in patient 8, who showed a mild glucose intolerance. In the three patients that underwent gastroscopy one showed evidence of ernia jatus, another drug-induced gastritis and another ulcer. However, antacid drugs were ineffective in reducing nocturnal awakenings. BMI was abnormally high in six patients; in five of these the weight gain had appeared only after the onset of NES.

Neurological data. Neurological and neuroimaging findings were unremarkable.

Psychiatric data (Table 2). Clinical assessment according to DSM-III-R criteria revealed panic disorder in five patients, associated with agoraphobia in three cases, generalized anxiety disorder in two patients and agoraphobia without panic disorder in two subjects. Apart from the peculiar nocturnal behavior, no obsessive-compulsive disorder was diagnosed. The EAT-26 showed normal scores in all patients. Both bulimia and anorexia nervosa were carefully excluded. Traits of depression and hysteria were revealed in most cases by the MMPI, and the HRSD and the HRSA showed pathological scores in five and four patients, respectively. Nevertheless, all patients were generally well-functioning adults, and apart from the intermittent use of psychotropic drugs in three subjects (Table 1), none of the patients was ever admitted to a hospital for psychiatric disturbances.

Polysomnographic data (Tables 3 and 4). Nocturnal recordings showed a variable number of eating-related awakenings (range 2–8 per night), which were quickly

TABLE 3. *Polysomnographic data*

Patient	Nocturnal eating episodes	Additional features
1	Two episodes during light NREM sleep (mean duration 2.5 minutes) in the first half of the night	Periodic chewing movements
2	Three episodes during light NREM sleep (mean duration 5 minutes) in the first third of the night	–
3	Three episodes during NREM sleep (mean duration 5.6 minutes) in the first third of the night	Periodic chewing and swallowing movements Periodic leg movements
4	Three episodes during NREM sleep (mean duration 3.3 minutes)	SOREMPs[a]
5	Three episodes during NREM sleep (mean duration 3.6 minutes)	–
6	Three episodes during light NREM sleep (mean duration 2.6 minutes) in the first half of the night	–
7	Six episodes during NREM sleep (mean duration 3.3 minutes)	Periodic chewing and swallowing movements Periodic leg movements
8	Two episodes during light NREM sleep (mean duration 4 minutes) in the first half of the night	Periodic leg movements
9	Three episodes during light NREM sleep (mean duration 2 minutes) in the first half of the night	–
10	Eight episodes during NREM sleep (mean duration 4 minutes)	Periodic chewing and swallowing movements Periodic leg movements

[a] SOREMPs = sleep onset REM periods.

followed by food intake and subsequent return to sleep. The mean amount of calories injested during the night turned out to be 1,200 kcal (range 500–2000 kcal). The mean duration of each episode was 3.5 minutes (range 1–9 minutes). The "eating latency", that is, the time interval between awakening and chewing start, was shorter than 30 seconds in 50% of the episodes (Fig. 1). The 10 polysomnograms included a total number of 65 intrasleep awakenings, the majority of which (55%) were accompanied by food injestion. All eating episodes were strictly related to nonrapid eye movement (NREM) sleep (Fig. 2). In particular, 80% of the episodes aroused directly from stages 1 and 2, whereas the remaining 20% originated from slow-wave sleep. Each patient could clearly recall in the morning questionnaire every single eating episode of the previous night. Sleep efficiency index ranged from 66 to 80% (mean 74%). Latency to sleep onset was longer than 20 minutes in four patients, and latency to rapid eye movement (REM) sleep was longer than 90 minutes in all cases. Three patients showed values of slow-wave sleep below 15%, whereas in all but one case the percentage of REM sleep ranged from 20 to 30%. No periodic appearance of the eating episodes could be identified. Four polysomnograms showed irregular recurrence of chewing and swallowing movements throughout all sleep stages. Narcolepsy was diagnosed in one patient, and periodic leg movements with or without a restless legs syndrome was diagnosed in four.

Pharmacological data. The mean follow-up time lasted 11 months (range 6–15 months). Out of the seven patients treated with d-fenfluramine, one recovered completely. A drastic improvement of at least a 50% reduction in nocturnal eating episodes, associated with a >70% decrease of total calorie intake and a disappearance of sweet food election, occurred in five subjects. Only one patient dropped out because of asthenia and drowsiness. The use of d-fenfluramine brought no substantial modification to daytime eating habits.

DISCUSSION

The adult subjects described in this clinical study complained of difficulty maintaining sleep due to frequent and recurrent awakenings to eat or drink. All patients manifested more than one episode per night, characterized by nocturnal food seeking and return to

TABLE 4. *Main polysomnographic data*

Patient	SL	REM-L	AWAKE	TST	SE	S1+S2	S3+S4	REM
1	43	195	6	369	74	209 (57%)	49 (13%)	111 (30%)
2	31	108	6	369	74	201 (55%)	83 (22%)	85 (23%)
3	2	91	7	387	77	255 (66%)	50 (13%)	82 (21%)
4	2	197	5	402	80	213 (53%)	94 (23%)	95 (24%)
5	28	201	5	331	66	205 (62%)	53 (16%)	73 (22%)
6	20	122	7	370	74	239 (65%)	56 (15%)	75 (20%)
7	11	96	6	400	80	276 (69%)	60 (15%)	64 (16%)
8	20	208	9	362	72	252 (70%)	33 (9%)	77 (21%)
9	22	126	6	390	78	215 (55%)	86 (22%)	89 (23%)
10	12	230	8	313	63	166 (53%)	65 (21%)	82 (26%)

SL: sleep latency; REM-L: latency to REM stage; AWAKE: number of awakenings; TST: total sleep time; SE: sleep efficiency index; S1, S2, S3, S4, REM: stages 1, 2, 3, 4 and REM. Apart from AWAKE, all parameters are expressed in minutes. The percentages in parentheses are referred to TST.

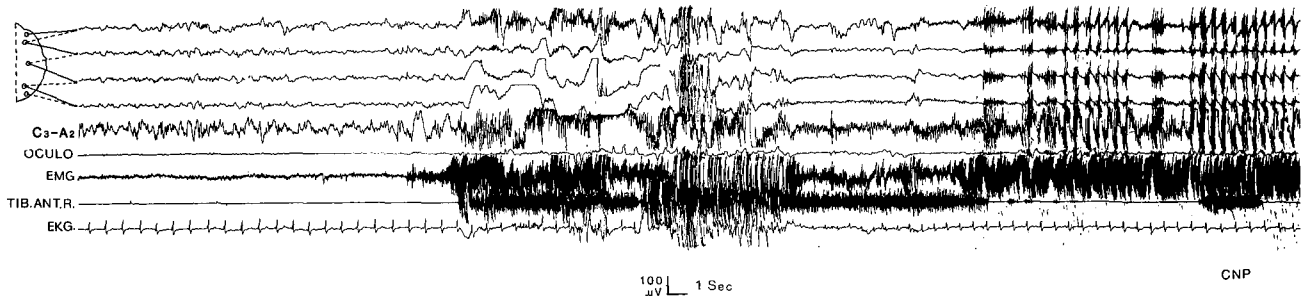

FIG. 1. Eating episode originating from slow-wave sleep. The sudden awakening is followed within 30 seconds by chewing movements (artifacts on the EEG leads). Oculo, electrooculogram; EMG, electromyogram; Tib Ant R, anterior right tibialis muscle; EKG, electrocardiogram.

sleep only after adequate food intake. Medical, neurological and laboratory investigations revealed no peculiar findings, and psychiatric disorders such as anorexia nervosa and bulimia were excluded. Polysomnograms were characterized by low levels of sleep efficiency and a high number of awakenings. Overweight and predisposing factors such as poor sleep hygiene, shift work or gastric discomfort were frequent clinical findings. No definite sex difference could be established. On the basis of these clinical data all the subjects met the main diagnostic criteria for a severe form of NES (Table 5).

Our cases, however, included supplementary features that may be considered of major diagnostic relevance. Although the International Classification of Sleep Disorders indicates among the diagnostic criteria of NES the absence of any other sleep disorder producing difficulty in maintaining sleep, five of our patients showed the coexistence of a dyssomniac disorder such as narcolepsy or periodic leg movements. Nevertheless, even in these cases, the majority of intrasleep awakenings were strictly and exclusively related to compulsive food seeking. In spite of the widely accepted statement that NES is primarily a problem of early childhood, in all our patients the onset of NES occurred during adulthood. Bulimia and anorexia nervosa were carefully excluded on the basis of DSM-III-R and EAT-26 criteria. In five of the six patients with abnormally high BMI values, weight gain was directly associated with the onset of sleep-related eating. Various psychiatric disturbances were found in nine of our 10 patients, and yet they were well-functioning adults. All nocturnal awakenings aroused from NREM sleep, and subjects retained memory of the eating episodes the next morning. Finally, the compulsive behavior of food seeking associated with an absence of real hunger, the food peculiarity, the short eating latency and the prompt return to sleep after food intake represent further specific features that may permit us to distinguish NES from other nocturnal dietary habits, especially those observed among insomniac patients. Table 6 is a list integrating the standardized diagnostic criteria of NES with these additional findings. According to these data, NES in adults could be described as a compulsive NREM sleep-related eating disorder, different from bulimia, anorexia nervosa or any other known psychiatric illness (3).

The drastic improvement in most of the patients treated with d-fenfluramine, a drug that increases 5-HT postsynaptic availability, confirms that serotonergic systems play a prominent role in the multifactorial control of appetite. In particular, the relation between sleep and appetite suggests that special attention should be paid to the hypothalamus, not only because the

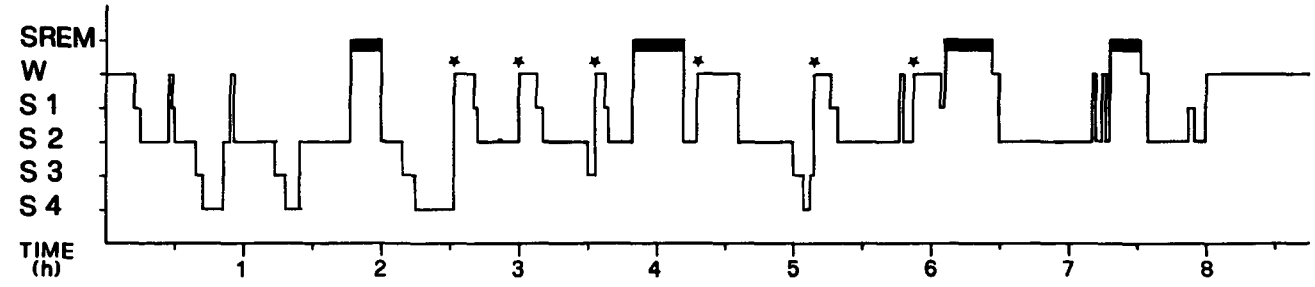

FIG. 2. Sleep histogram of an adult subject affected by nocturnal eating syndrome (patient 7). All intrasleep episodes of compulsive eating (black stars) arise from NREM sleep. Besides the increased number of awakenings, the regular organization of sleep in stages and cycles is preserved. W, wakefulness; S1, S2, S3, S4, NREM sleep stages; SREM, REM sleep.

TABLE 5. *Diagnostic criteria for nocturnal eating syndrome*[a]

A. A complaint of difficulty in maintaining sleep
B. Frequent and recurrent awakenings to eat or drink
C. Sleep onset is normal following ingestion of the desired food or drink
D. Polysomnographic monitoring demonstrates an increase in the number or duration of awakenings
E. No evidence of psychiatric or medical disorders to account for the complaint (e.g. bulimia, hypoglycemia)
F. Absence of any other sleep disorder producing difficulty in maintaining sleep

[a] From the International Classification of Sleep Disorders.

effects on feeding are mediated by receptors in the paraventricular nucleus and other hypothalamic sites (6), but especially because of the intrinsic chronobiological properties of this structure. Oral activities such as drinking, eating and smoking seem to be influenced by a 90–100 minute rhythm (7,8), which recalls the cyclic alternation of NREM and REM sleep (9) and the Basic Rest Activity Cycle (10). The demand for food is closely related to REM sleep in babies (11), whereas this periodic feeding behavior is generally suppressed in adults. Oswald and Adam (12) reported an adult case with nocturnal episodes of eating chronologically linked to REM sleep. Roper (13) described a woman with episodes of sleepwalking and unconscious overeating, during which she could injest even inedible substances. Gupta reported the presence of sleep-related eating disorder in 10 out of 32 female patients with bulimia nervosa (14). Schenck et al. (15) polygraphically analyzed 19 subjects affected by heterogeneous sleep-related eating episodes. However, most of the patients suffered from somnambulism and experienced confusional-amnestic arousals with automatic behaviors. In an updated paper, the same group reported additional categories of sleep-related eating disorders (16). Recently, Winkelman et al. (17) described nocturnal binge eating in 15 patients, most of them female and most with concurrent psychiatric disorders. However, half of all cases described regular amnesia for the behavior, and less than half of all cases had a daytime eating disorder. The present report provides a framework for NES in a group of adults all sharing common clinical features (Table 6).

TABLE 6. *Clinical features common to all 10 patients*

A. A complaint of difficulty in maintaining sleep
B. Frequent and recurrent awakenings to eat or drink
C. Food-seeking drive described as urgent abnormal need to swallow food with absence of real hunger
D. Compulsive feeding behavior
E. Food peculiarity (always edible substances)
F. Inability to return to sleep without eating
G. Full waking during the episodes and clear recall of them in the morning
H. Polysomnographic evidence of low sleep efficiency index and high number of awakenings
I. Strict relation between nocturnal episodes and NREM sleep
J. Short "eating latency" and quick return to sleep after eating
K. No medical, hormonal or neurological disorders
L. Absence of bulimia or anorexia nervosa

The 10 subjects described in this clinical study represented 5% of the total number of out-patients that were referred to our sleep disorders center in an 18-month period. This statistical consideration suggests that NES, if carefully sought for, may be much more common than usually believed.

Acknowledgements: We thank Dr. Manrico Guerra for his valuable advice on dietetic and endocrinological issues.

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