Nocturnal Eating: Sleep-Related Eating Disorder or Night Eating Syndrome? A Videopolysomnographic Study

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Study Objectives: To describe the clinical and videopolysomnographic characteristics of nocturnal eating episodes in sleep-related eating disorder.

Design: Descriptive study of outpatients prospectively enrolled in 2 sleep centers.

Setting: Videopolysomnographic recordings done in the sleep laboratory.

Patients: Thirty-five consecutive drug-free patients with nocturnal eating.

Interventions: N/A.

Measurements and Results: Clinical interviews disclosed abnormal compulsory nocturnal eating episodes in all patients associated with a clinical report of sleepwalking (in 1), somniloquy (in 5), restless legs syndrome (in 8), and periodic limb movements during sleep (in 4). Videopolysomnography documented 45 episodes of nocturnal eating in 26 patients. Eating always occurred after complete awakenings from non-rapid eye movement sleep and only in 1 patient from REM sleep and was characterized by electroencephalographic alpha activity with no dissociated features of state-dependent sleep variables. Patients interviewed during the eating episodes were fully conscious and remembered the events the next day. Pathological periodic limb movements during sleep index was recorded in 22 and restless legs syndrome dyskinesias in 5 patients. Recurring chewing and swallowing movements during sleep were a feature in 29 patients, associated in about half of the events with electroencephalographic arousals.

Conclusions: In our patients, eating episodes occurred with normal consciousness and recall. Chewing or swallowing movements during sleep occurred frequently, resembling rhythmic masticatory-muscle activity in bruxism patients. The presence of periodic limb movements during sleep and chewing activity, the reported efficacy of dopaminergic medications, and the compulsory food-seeking behavior all argue for a dopaminergic dysfunction underlying the pathogenesis of sleep-related eating disorder.

Keywords: Sleep-related eating, night eating syndrome, eating disorders, arousal disorder, polysomnography, chewing rhythmic movements, periodic limb movement disorder.

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INTRODUCTION

SLEEP-RELATED EATING DISORDER (SRED) IS A HETEROGENEOUS SYNDROME COMBINING CHARACTERISTICS OF BOTH EATING AND SLEEP DISORDERS, in which nocturnal arousals from sleep are followed by rapid and compulsory eating behavior. In the case series already reported, individuals displayed reduced or, more rarely, full awareness during the episodes and usually had subsequent amnesia for the nocturnal eating episodes. Patients were predominantly women with personal histories of other parasomnias and with a chronic course of the disorder.[1-3] Polysomnographic (PSG) recordings showed, in addition to the eating episodes, evidence for parasomnias during slow-wave sleep, such as sleepwalking and sleep apnea, periodic limb movements during sleep (PLMS), restless legs syndrome (RLS), and narcolepsy.[1,3-5] However, the relationship between SRED and other categories of sleep-related eating, in particular the night eating syndrome (NES), remains unclear.[3] The presence of impaired consciousness and subsequent amnesia for the eating episodes has been considered by some authors[3,6] to be the major differentiating feature of SRED versus NES. This has been, however, by no means a universal finding, and the PSG characteristics of the eating episodes are still to some extent controversial. PSG data are indeed scant and not routinely obtained in studies of SRED or NES, and only 3 relatively large series of polygraphically documented patients are available.[1,3,4,7]

We performed a videopolysomnographic (VPSG) evaluation of 35 consecutive patients with nocturnal eating and report here the clinical and VPSG characteristics of the abnormal eating episodes, the patterns of abnormal motor behavior, and their relationship to sleep structure.

MATERIAL AND METHODS

Patients

Thirty-five consecutive outpatients (21 women; mean age: 44±12.7 years, range: 24-77; mean body mass index: 28.5±4.6; mean age at onset of SRED: 39.6±13.9 years) were prospectively enrolled between March 2001 and June 2003 at our centers for sleep disorders (Table 1). Nocturnal eating with consequent body-weight increase was the main clinical condition for which all patients sought referral. All patients underwent a semistructured clinical interview regarding their nocturnal eating episodes, sleep habits, history of parasomnias, history of psychiatric disorders, and physical and neurologic examination.

No patient had cognitive deterioration (Mini-Mental State Examination scores were always ≥ 24) or physical illnesses, includ-

Disclosure Statement

This was not an industry supported study. Drs. Vetrugno, Manconi, Ferini-Strambi, Provini, Plazzi, and Montagna have indicated no financial conflicts of interest.

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Table 1—Demographic and Clinical Characteristics of Patients

Characteristic	
Sex	
Men	14
Women	21
Age, y	
At observation	44 ±12.7
At onset of SRED	39.6±13.9
Body mass index, kg/m^2	28.5±4.6
Frequency of nocturnal eating	Every or almost nightly
Nocturnal eating episodes per night by history, no.	Patients, no.
1	2
2-5	25
>5	8
Positive family history for	
Nocturnal eating	2
Insomnia	2
Psychiatric disorders plus nocturnal eating	2
Associated features by history	
Sleepwalking	1
RLS	8
PLMS	4
Sleep talking	5
Mood depression	14
Chronic hepatitis B	2
Gastritis	2
Systemic hypertension	2

Data are presented as number, except age and body mass index, which are presented as mean ± SD. SRED refers to sleep-related eating disorder; RLS, restless legs syndrome; PLMS, periodic limb movements of sleep.

ing alcohol and drug abuse, diabetes mellitus, thyroid and other metabolic/endocrine disorders, were absent. Mood was assessed by means of the Yesavage or Beck Depression scales.[8,9] Concurrent daytime eating disorders were also absent; in particular, no patient reported daytime binge eating or bulimia, as assessed by specific clinical interview. All patients were drug free at the time of the VPSG examination.

VPSG Evaluation

All patients underwent 1 night of VPSG evaluation. The sleep laboratory was equipped with a food-and-drink dinner table positioned at the bedside with staples chosen and brought in by the patients themselves. Sleep recordings and scoring were done according to standard methods[10] and included electroencephalogram (EEG) (C3-A2; C4-A1, O2-A1); right and left electrooculogram; chin superficial electromyogram (EMG); and surface EMG of the orbicularis oculi, orbicularis oris, masseter, sternocleidomastoideus, biceps brachii, biceps femoris, and tibialis anterior muscles. Oronasal airflow and thoracic and abdominal respiratory efforts were monitored with a strain gauge, and oxygen saturation (SaO_2) was monitored with a finger pulse oximeter.

Sleep structure and sleep efficiency (the percentage of total sleep time/time in bed) were evaluated. Arousals with arousal index (the number of arousals per hour of sleep) and PLMS with PLMS index (PLMS-I, the number of PLMS per hour of sleep) were calculated according to Carskadon and Coleman's criteria.[11,12] A PLMS-I greater than 5 throughout the night sleep was considered pathologic. An abnormal breathing event during sleep was defined as apnea (a complete cessation of airflow lasting 10 seconds or more) or hypopnea (a discernible reduction in respiratory airflow accompanied by a decrease of at least 4% in SaO_2), differentiating obstructive from central sleep apnea if the thoracic and abdominal respiratory efforts respectively continued or not. A respiratory disturbance index (the average number of apnea-hypopnea episodes per hour of sleep) greater than 5 was chosen as a criterion for sleep-disordered breathing.

RESULTS

Anamnestic Data of the Patients

There was a preponderance of young women, (mean age at onset of nocturnal eating was 39.6±13.9 years, range 17-77), and nocturnal eating episodes were reported every night, or almost every night, often many times per night (Table 1). Patients complained that they woke up from sleep after having regularly fallen asleep at night and felt a compulsive desire to eat because of an "inner" feeling. They denied that the feeling was like being hungry. After having eaten, usually a few snacks, they would go back to bed, quickly falling asleep, only to wake up again in the night with the same sensation. This could recur several times per night. A need to drink was not usual, and no accidents occurred during the eating episodes. Additional reported categories of sleep disorders were sleepwalking in 1 patient, RLS in 8, PLMS in 4, and sleep talking in 5. Fourteen patients reported complaints indicative of depressed mood, as assessed by Yesavage scale[8] and Beck Depression Inventory scale.[9] There was a positive family history of nocturnal eating in 2 patients, insomnia in 2 patients, and psychiatric disorders (depression, eating disorders) plus nocturnal eating in 2 patients (Table 1). Neurologic examination and brain magnetic resonance or computed tomographic imaging, done in all patients, were always normal.

VPSG Findings

Architecture of Nocturnal Sleep

On average, sleep structure was characterized by increased non-rapid eye movement (NREM) light sleep stages 1 and 2 (60.5%±8.2%, range: 44%-78.3%; n.v. ~ 50%), decreased NREM deep sleep stages 3-4 (17.9%±8.1%, range: 0%-35%; n.v. ~ 25%) and rapid eye movement (REM) sleep (21.6%±5.8%, range: 0%-31.7%; n.v. ~ 25%). Mean sleep latency was 8.6±5.9 minutes (range: 1-23); REM sleep latency, 108.5±72 minutes (range: 29-379); and sleep efficiency was reduced (76.2%±18.5%, range: 11%-96%; n.v. > 85%). Mean arousal index was 19±11.3 (range: 4-48). PLMS were observed in 24 patients, with a mean PLMS-I of 26.7±18.7 (range 5-73); 22 patients had a PLMS-I greater than 5. In 5 patients, VPSG showed motor restlessness with involuntary limb movements while awake and resting dyskinesia, typical of RLS, associated with pathologic PLMS-I in 4 patients, and with a PLMS-I of 5 in 1 patient. Sleep efficiency was particularly reduced in these patients who had VPSG features of RLS. Snoring not associated with obstructive sleep apneas [respiratory disturbance index < 5 (range 0-3); mean SaO_2 throughout sleep: 96% (range 95-97%) never peaking below 90%] was present in 11 patients (Table 2-please log on to http://www.journalsleep.org/ to view this table.).

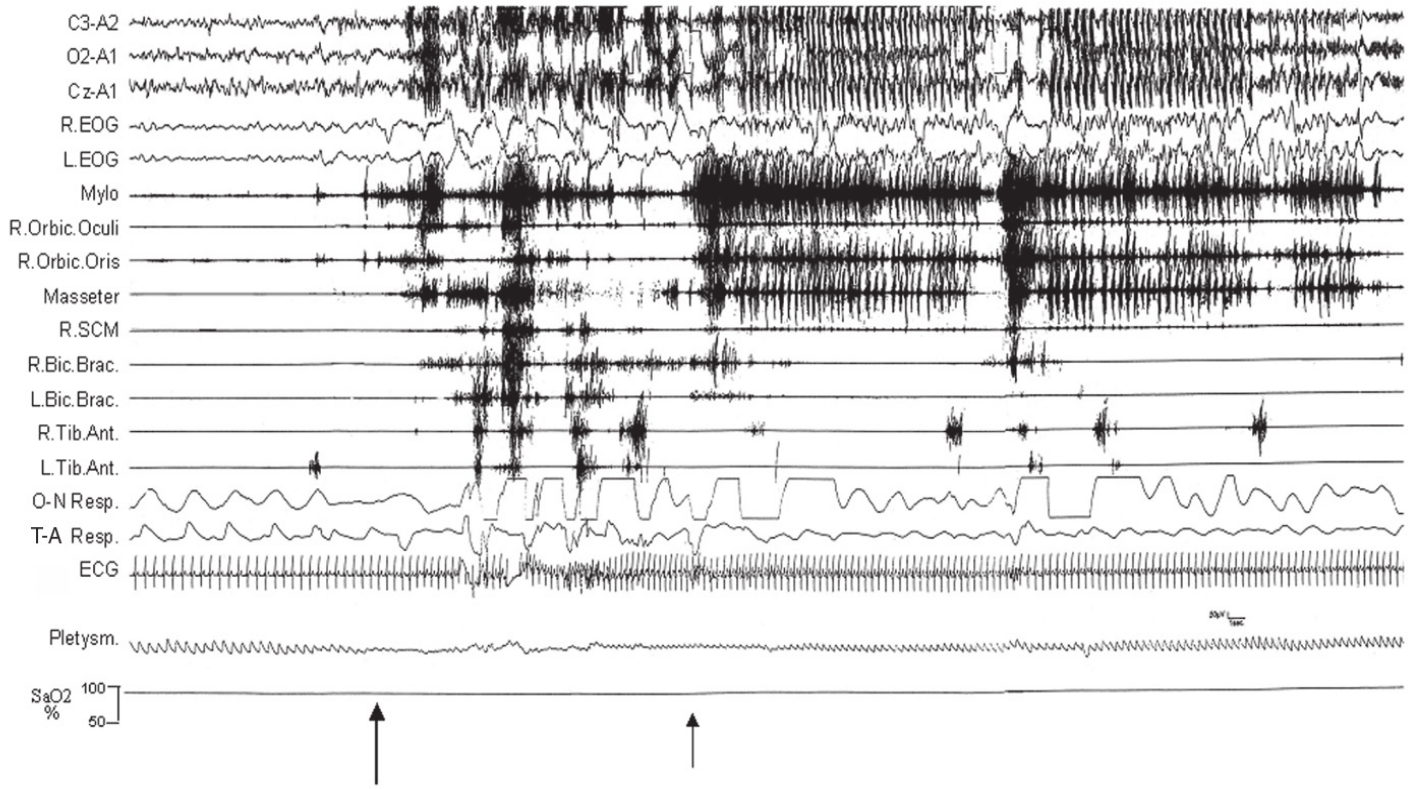

Figure 1—Sleep-related eating episode in patient 1. There was a short delay between awakening (long arrow) and the beginning of eating-related muscle activity (short arrow). EOG refers to electrooculogram; Mylo, mylohyoideus; Orbic. Oculi, Orbicularis oculi; Orbic. Oris, orbicularis oris; SCM: sternocleidomastoideus; Bic. Brac., biceps brachii; Tib. Ant., Tibialis Anterior; O-N Resp., oral-nasal respirogram; T-A Resp., thoracoabdominal respirogram; ECG, electrocardiogram; Pletysm, plethysmogram; SaO_2: oxygen saturation.

VPSG Features of the Eating Episodes

During VPSG, eating never occurred prior to sleep onset. Twenty-six patients were observed getting out of bed, after having fallen asleep, to eat the food placed at the bedside (Figure 1). Fourteen patients displayed 1 episode of nocturnal eating, 7 patients had 2, 3 patients had 3, and 2 patients had 4 episodes (Table 2). Eating episodes always arose after awakening from NREM sleep, only in 1 patient from REM sleep, and without any close relationship with PLMS or respiratory-related arousals (Table 2). In particular, eating episodes in 14 patients occurred after awakening from stage 2 NREM sleep; in 3 patients from stage 4 NREM sleep; in 5 patients from stages 2, 3, and 4 NREM sleep; in 1 patient from REM sleep; whereas the episodes occurred in 2 during prolonged intrasleep wakefulness. EEG activity recorded during the episodes was always characterized by normal alpha activity with electrooculogram and heart and breathing rates typical of wake (Figure 1). Mean time delay from awakening during the night to food intake (eating latency) was 7.1 minutes, with a wide range from 0.33 to 81 minutes and with a mean eating duration of 6.2 minutes (range: 0.41-20). The mean time delay between wake-up and beginning to eat was 8.9 minutes for episodes occurring during stage 2 NREM, and 1.9 minutes for those during stages 3-4 NREM sleep. Time delays were consistently longer in those patients with associated RLS, probably indicating an effect of RLS disturbances on sleep structure causing prolonged wakefulness (Table 2). Patients were questioned whenever eating occurred, and, apart from a transitory slowness of mentation and behavior related to sleep inertia, all patients were conscious during the episodes, independently from the time delay between awakening and eating. As a reason for eating, patients reported feeling a compulsion to eat and specifically denied feeling hungry or thirsty. Nocturnal eating behavior, including manipulation of food, was appropriate. All patients clearly identified and recalled all of their eating episodes the morning after. Sleep latency from resumption of quiet resting posture after nocturnal eating was 14 minutes (range: 0.5-107).

Orofacial Masticatory Activity During VPSG

In 29 patients, VPSG showed recurrent EMG activity of the masseter and orbicularis oris muscles, which, when checked against the videorecordings, could be defined as repetitive masticatory and swallowing behavior. This activity could occur either as tonic EMG or as phasic bursts of masseter and/or orbicularis oris and oculi muscles, less than 250 milliseconds in duration, reminiscent of faciomandibular jerks (Figures 2 and 3). We scored this EMG activity whenever it lasted more than 3 seconds and with 3 or more consecutive bursts of EMG activity on the masseter according to Lavigne et al.[13] Such EMG activity was absent during wakefulness and present throughout all sleep stages but mainly during stages 1 and 2 of NREM sleep, with a mean chewing/swallowing movements index of 27.2±14.3 bursts per hour (range: 8-72) (Table 2). Masseter EMG activity was often associated with EEG arousals visible on the EEG (defined as a brief change in fast EEG activity or slow delta and K-complex bursts) and with transient electrocardiogram acceleration. A mean of 116.2 masticatory/swallowing movements per patient per night was recorded (range: 11-322), which were associated with EEG arousal in 48% of the episodes (range: 18.3-87.6).

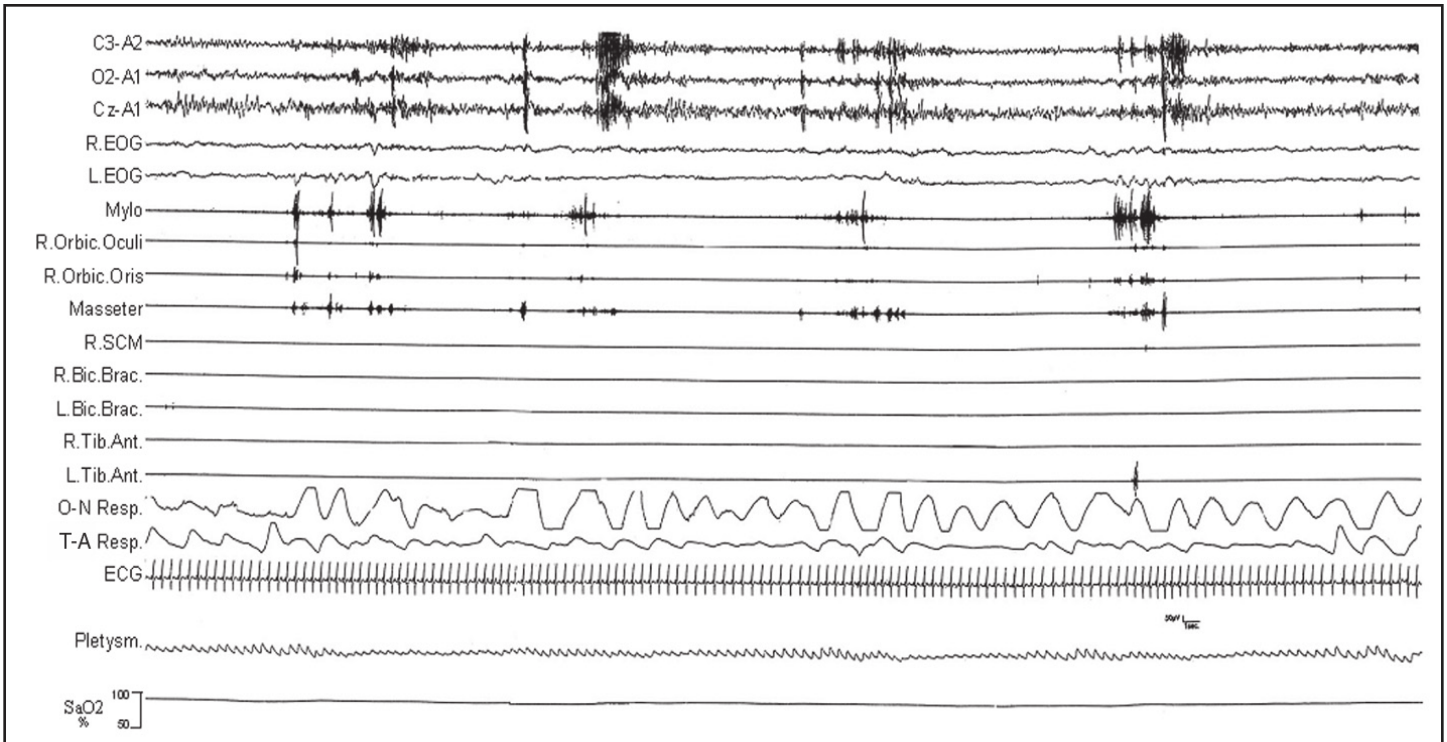

Figure 2—Repetitive masticatory movements during non-rapid eye movement sleep stage 2 in patient 1 involving masseter, orbicularis oris, orbicularis oculi and mylohyoideus muscles. Abbreviations as per Figure 1.

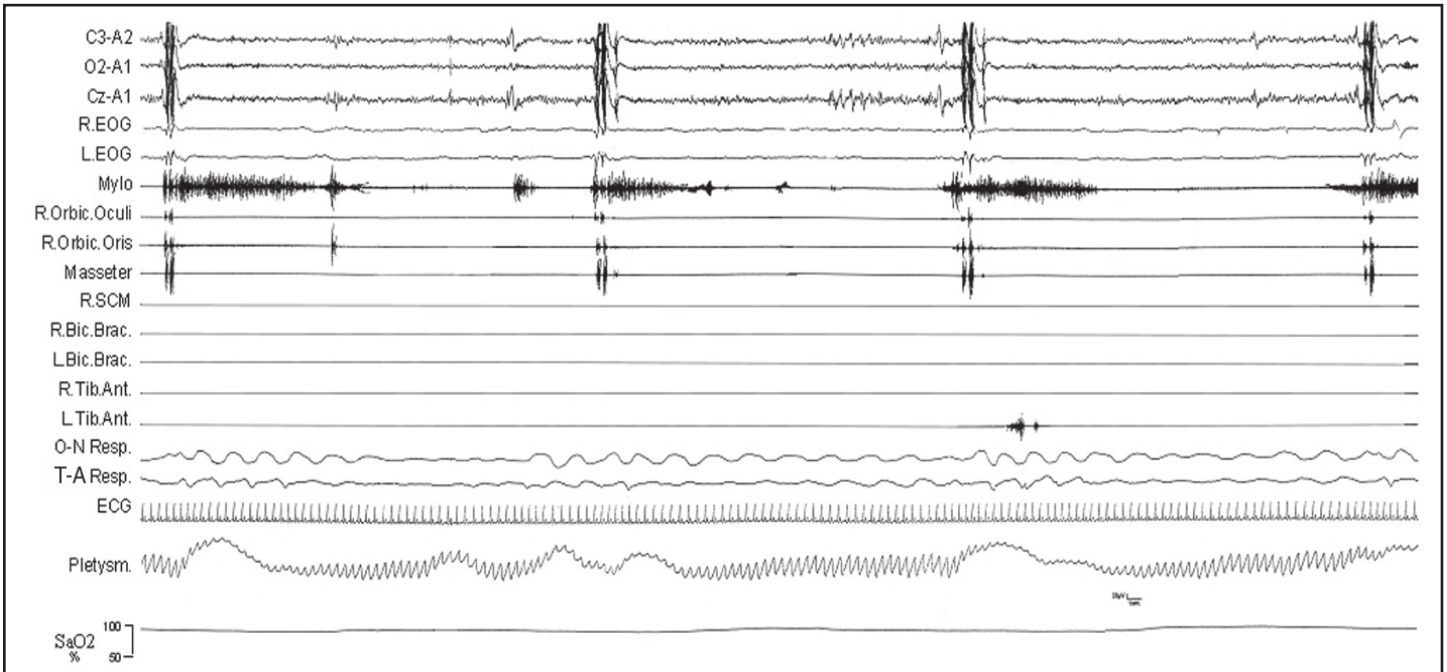

Figure 3—Myoclonic repetitive jerks involving faciomandibular muscles during non-rapid eye movement sleep stage 2 in patient 4. Transient tonic electromyogram activity in the mylohyoideus muscle follows the faciomandibular jerks. Abbreviations as per Figure 1.

Comparison of Isolated Nocturnal Eating Versus Nocturnal Eating Associated With Another Disorder

In order to verify for possible causal relationships and in an attempt to classify our patients according to the *International Classification of Sleep Disorders-2* diagnostic criteria (Table 2), we divided the whole sample of patients into those with isolated nocturnal eating and those with associated medical or psychiatric diseases or even isolated sleep-related symptoms. All of our patients fulfilled *International Classification of Sleep Disorders-2* criteria A, Bii, and Bvi. (criterion A. calling for recurrent episodes of involuntary eating and drinking occurring during the main sleep period; criterion Bii for insomnia related to sleep disruption from repeated episodes of eating, with a complaint of nonrestorative sleep, daytime fatigue, or somnolence; and criterion Bvi. for adverse health consequences from recurrent binge eating of high-caloric foods). However only 22 patients (63%) had other mental or medical disorder or isolated sleep-related symptoms associated with the nocturnal eating, whereas 13 patients (37%) did not (Table 2). When comparing these 2 groups with respect to the

characteristics of the eating episodes and the VPSG findings, patients with nocturnal eating but without associated disorders were significantly younger, had higher total sleep time, better sleep efficiency, a lower number of eating episodes per night, and lower PLMS-I and chewing-swallowing index (all p values < .05). The VPSG features of the eating episodes were however the same.

DISCUSSION

Nocturnal eating episodes in our patients were reportedly often associated with isolated symptoms during sleep or sleep disorders such as somniloquy, sleepwalking, RLS, PLMS, and snoring. Videopolygraphically, increased PLMS-I was found in 22 patients and RLS in 5; 4 of the RLS patients also had increased PLMS-I. No patient had VPSG features of obstructive sleep apnea syndrome. Overall, nocturnal eating was associated with either anamnestic or polygraphically documented sleep disorders in 24 of 35 patients. Our clinicodemographic data confirm previously reported features,[2,3] including female sex predominance (21/35).

In our case series, sleep structure was characterized by a prevalence of light NREM sleep and reduced sleep efficiency, some of these findings probably relative at least in part to a first-night effect. However, all sleep stages were represented in most patients, and there was a relatively normal cycling of the different sleep phases. Dissociated features of abnormal NREM or REM sleep were never observed, even during the eating episodes recorded in 26 patients. Therefore, sleep macrostructure was substantially normal, and the truly abnormal finding was a decreased sleep efficiency. All eating episodes occurred during EEG-defined wakefulness, and patients, upon being questioned, appeared to be awake and conscious even when the delay between waking up and eating was very short. All patients had a clear recall of the episodes in the morning. For these particular features, our findings are comparable with those of Spaggiari et al,[7] in which all of the patients had normal consciousness levels while eating. Spaggiari et al's patients, however, were reported as having NES.[7] Full consciousness was present in only 21% of Schenck et al's SRED series[4] and in 9% of Winkelman's cases,[3] and, indeed, clouding of consciousness has been considered the hallmark of SRED versus other nocturnal eating disorders such as NES.[6] Such a discrepancy calls for an explanation, and foremost, in our opinion, is the fact that our patients were all drug free, whereas most patients in the other case series were sometimes on heavy psychotropic medications (70% of Winkelman's series, moreover observed in a psychiatric setting) or substance abuse. Therefore, confusion and amnesia for the episodes, and even probably the careless and sloppy eating behavior reported in the literature may relate, at least in part, to the confounding effects of medications or substance abuse. Moreover, medication may also interfere with state-dependent features, thus originating dissociated states of NREM and REM sleep. Another factor worth considering is that the level of consciousness after awakening physiologically depends upon the duration and depth of preceding sleep and intervening wakefulness (so-called sleep inertia). In our patients, the impairment of consciousness during the eating episodes seemed indeed not to go further than what is normally due to sleep inertia. An alternative explanation for these findings of normal versus abnormal consciousness level during the eating episodes is that we are dealing with 2 (or more) different populations of patients, a concept also embodied in the original definition of SRED as a heterogeneous syndrome.[1] The difficulties with classification of SRED on the basis of impaired consciousness during the eating episodes are also highlighted in the revised *International Classification of Sleep Disorders-2* classification, which acknowledges that eating episodes in SRED may be accompanied by either clear or clouded consciousness.[14] Based on our patients' series, while suggesting that SRED probably represents a heterogeneous syndrome that may conceivably occur with medications and in drug-naïve patients, we conclude that impaired levels of consciousness cannot be taken as the 1 feature clearly typifying SRED, in comparison with the other NES.

Another problem is represented by whether nocturnal eating episodes should be associated or not with another sleep disorder to be classified as SRED, as originally proposed.[1,2] The new *International Classification of Sleep Disorders-2* [14] states, on the contrary, that SRED cannot be diagnosed in the presence of another sleep disorder, medical or neurologic disorder, mental disorder, or medication use or substance use disorder that better explain the disturbance (diagnostic criterion C.). This last criterion is difficult to apply, given our current ignorance about the causative mechanism of SRED.

Our patients with isolated nocturnal eating had indeed significantly lower mean age, higher total sleep time and sleep efficiency, and fewer eating episodes per night and PLMS-I and chewing-swallowing index, when compared with the patients with nocturnal eating associated with another sleep, medical, or psychiatric disorder. However, such differences could, at least in part, be explained by the nature of the associated sleep disorders, such as periodic limb movement disorder and RLS, whereas the VPSG characteristics of the nocturnal eating episodes remained the same in the 2 groups of patients. Therefore, although PSG sleep characteristics differ whether patients with nocturnal eating have some other associated disorder, the causative link between the latter and the nocturnal eating and whether we are dealing with 2 (or more) truly nosologically different populations remains unclear.

A remarkable VPSG feature in our patients possibly differentiating SRED from other nocturnal eating disorders was that compulsory eating occurred after having fallen asleep and never prior to sleep onset. Such behavior has also been reported in all previous case series,[2,3,5,7] though not in the original paper on NES by Stunkard et al.[15]

A peculiar motor event observed in 29 out of 35 of our patients was regularly recurring chewing and swallowing movements throughout all sleep stages, especially during light NREM sleep, with trigeminal and facial-muscle EMG involvement. This has been briefly mentioned in previous SRED series but without PSG documentation.[4,7] Such EMG activity in our patients was reminiscent of the spontaneous rhythmic masticatory-muscle activity (RMMA) with or without tooth-grinding observed during sleep in normal subjects and especially in sleep bruxism.[13,16] RMMA may be associated with chewing, swallowing, and breathing and occurs in relation to transient activation in the cortex and autonomic nervous system during sleep.[17,18] Trigeminal-facial EMG activity was periodic in our patients, linked to EEG arousals in about half of the events, and often coexistent with PLMS. This multidistrict simultaneity, e.g., orbicularis oculi/oris-masseter-tibialis anterior, of the phasic motor events is suggestive of a generalized motor disinhibition, probably of a subcortical motor-pattern generator that phasically fires during sleep.[19] Intriguingly, an increased PLMS-I was present in 22 of our patients. This high recurrence of

PLMS (63%) and RMMA (83%) in nocturnal eating suggests the presence of common pathophysiologic factors, at least in a subset of patients, possibly related to involvement of the dopaminergic system, which has been implicated in the origin of both the PLMS and the RMMA.[17,20] Moreover, pharmacologic manipulation of the dopaminergic system is effective both for reducing PLMS and for curtailing nocturnal eating.[4,20,21] The dopaminergic system is implicated in reward mechanisms at the mesolimbic level, and its dysfunction plays a role in compulsive behaviors, including food-seeking behavior.[22] It is therefore appropriate to suggest a pathogenic role for the dopaminergic system in nocturnal eating disorders, based on the beneficial effects of dopaminergic agents in SRED,[4,21,23] its association with increased motor activity (RMMA, PLMS, RLS) purportedly of dopaminergic origin, and the compulsive food-seeking episodes. SRED could be conceived of as a disorder whereby sleep-related motor and autonomic arousal processes may happen to stimulate the appetitive-feeding pathways and elicit a time-inappropriate compulsive feeding behavior. Our findings also emphasize the importance of VPSG in the evaluation and characterization of unusual behaviors arising from sleep.

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