Sleep Medicine 64 (2019) 85–91

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Searching food during the night: the role of video-polysomnography in the characterization of the night eating syndrome

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ARTICLE INFO

Article history:
Received 24 April 2019
Received in revised form
19 June 2019
Accepted 24 June 2019
Available online 5 July 2019

Keywords:
Night eating syndrome
Sleep-related eating
Videopolysomnography
Diagnostic criteria

ABSTRACT

Objectives: To describe the video-polysomnographic (VPSG) features of the night eating syndrome (NES), exploring the existence of potential subtypes.
Methods: In this study, 20 consecutive patients with NES according to the most recent diagnostic criteria underwent an overnight VPSG. None of them presented with a sleep-related eating disorder (SRED). VPSG recordings were reviewed identifying all eating episodes. For each episode, eating latency (time delay from awakening to food intake), eating duration (time between eating onset to eating offset) and sleep latency after eating offset (time delay from eating offset to sleep) were calculated. Total episode duration was considered as the time between awakening and sleep latency after eating offset.
Results: Ten patients fulfilled the A1 core criterion for NES (evening hyperphagia with consumption of at least 25% of the daily caloric intake after the evening meal); within this group, eight patients also fulfilled the A2 criterion (at least two episodes of nocturnal eating per week) and were thus included in the evening hyperphagia (EH) subgroup. The remaining 10 patients satisfied only the A2 core criterion for NES, constituting the nocturnal ingestion (NI) subgroup. We recorded 20 eating episodes, seven in the EH group and 13 in the NI group. In the EH subgroup, three eating episodes occurred before sleep onset, one after an awakening from non-rapid eye movement (NREM) stage 1 sleep, two from NREM stage 2 and one from REM sleep. All 13 NI episodes occurred after an awakening from sleep (1 from NREM stage 1 sleep, 8 from NREM stage 2 and four from NREM stage 3). In EH patients, eating latency, total episode duration and sleep latency after eating offset were significantly longer than in NI patients.
Conclusion: Our VPSG data from a case series of 20 patients referred to our center for nocturnal eating indicate potential different NES subtypes. This distinction may have an impact on patients' treatment and follow-up.

1. Introduction

Nocturnal feeding is common in eating disorders such as bulimia nervosa and binge eating disorder (BED), in which abnormal eating is present throughout day and night. Two specific categories of abnormal eating occur only during the night: (a) night eating syndrome (NES) and (b) sleep-related eating disorder (SRED) [1,2].

NES, first described in 1955 [3] as a disorder characterized by morning anorexia, evening hyperphagia and insomnia, is currently included in the Diagnostic and Statistical Manual of Mental Disorders, V edition (DSM-V) [4]. NES is not uncommon, with a

Abbreviations: AHI, apnea/hypopnea index; BMI, body mass index; DSM-V, Diagnostic and Statistical Manual of Mental Disorders, V edition; EH, evening hyperphagia; NES, night eating syndrome; BED, binge eating disorder; NI, nocturnal ingestion; OSAS, obstructive sleep apnea syndrome; PLMS, periodic limb movements during sleep; RLS, restless legs syndrome; SRED, sleep-related eating disorder; VPSG, video-polysomnography.

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https://doi.org/10.1016/j.sleep.2019.06.018
1389-9457/

prevalence of about 1.5% in the general population [5,6], which increases up to 3.8–7% in individuals with diabetes [7], up to 20% in psychiatric patients [8] and in candidates for weight loss surgery [9]. Since its first description by Stunkard et al. [3], NES diagnostic criteria have been revisited, now including six items, all of which need to be fulfilled (See Box 1. Proposed NES Diagnostic Criteria). The core criterion requires one or both between evening hyperphagia (criterion A1) and/or at least two nocturnal awakenings per week with food ingestion (criterion A2), associated with full awareness (criterion B) [10].

Since the international NES working group proposed these criteria, possible subtypes of the disorder (ie, the evening hyperphagia and nocturnal ingestion subtypes) have been hypothesized. The same working group proposed that researchers involved in this field provide their contribution towards a more thorough understanding of such phenotypes by presenting hands-on clinical and, if available, PSG data pointing at any segregation of the proposed criteria [10,11].

SRED is an non-rapid eye movement (NREM) sleep parasomnia characterized by recurrent episodes of dysfunctional and involuntary eating and drinking that occur after an arousal during the main sleep period [12–15]. The presence of impaired consciousness and subsequent amnesia for the eating episodes has been considered the major differentiating feature of SRED versus NES. Both NES and SRED patients present sleep disturbances. Difficulties in initiating and maintaining sleep, decreased sleep efficiency and total sleep time, poor sleep quality are commonly found among NES patients as well as periodic limb movements during sleep (PLMS) and bruxism [16]. Conversely, SRED is frequently associated with other sleep disorders (particularly parasomnias, restless legs syndrome (RLS) and obstructive sleep apnea syndrome [OSAS] [13,15]. Several studies described the features of SRED nocturnal eating episodes by means of video-polysomnographic (VPSG) recordings, while NES episodes have only been partially defined so far [14,17].

The aim of this work consists in describing clinical and VPSG features of nocturnal eating episodes and their possible relationship with sleep structure in 20 consecutive patients referred to our sleep center and our Unit of Metabolic Diseases and Clinical Dietetics with a diagnosis of NES, following the putative existence of different NES subtypes.

Box 1
Proposed night eating syndrome diagnostic criteria [10].

A. The daily pattern of eating demonstrates a significantly increased intake in the evening and/or nighttime, as manifested by one or both of the following:
   1. At least 25% of food intake is consumed after the evening meal
   2. At least two episodes of nocturnal eating per week
B. Awareness and recall of evening and nocturnal eating episodes are present.
C. The clinical picture is characterized by at least three of the following features:
   1. Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week
   2. Presence of a strong urge to eat between dinner and sleep onset and/or during the night
   3. Sleep onset and/or sleep maintenance insomnia are present four or more nights per week
   4. Presence of a belief that one must eat in order to initiate or return to sleep
   5. Mood is frequently depressed and/or mood worsens in the evening.
D. The disorder is associated with significant distress and/or impairment in functioning.
E. The disordered pattern of eating has been maintained for at least three months.
F. The disorder is not secondary to substance abuse or dependence, medical disorder, medication, or another psychiatric disorder.

2. Materials and methods

2.1. Patients

This prospective study included 20 outpatients (13 women; mean age: 48 ± 12 years, range: 28–64; mean body mass index [BMI]: 30 ± 9 kg/m^2, range: 21–53; mean age at onset of nocturnal eating: 36 ± 12 years, range: 20–59) complaining of nocturnal eating with consequent body weight increase enrolled from January 2014 to January 2017. Fourteen patients were referred to our sleep center and six patients were referred to our Unit of Metabolic Diseases and Clinical Dietetics. All 20 patients were evaluated using the same procedure. They underwent a physical and neurological examination and two separated semi-structured face-to-face clinical interviews with a sleep disorders specialist and with an eating disorders expert. The first interview regarded medical history, with particular attention to sleep habits, nocturnal eating episodes and the presence of neurological disorders. The second interview was conducted to carefully exclude the presence of lifetime and current daytime eating disorders. In particular, binge eating disorder (BED) was excluded by using the Binge Eating Scale (BES). The presence of NES and/or SRED was investigated using the latest set of diagnostic criteria for NES together with the International Classification of Sleep Disorders, Third Edition (ICSD-3) criteria for SRED [10,18]. Food consumption during episodes had been previously assessed by a registered dietitian based on dietary recall. Based on the presence of the A1 and A2 core current diagnostic criteria for NES [10], the patients were divided into two subgroups. Patients satisfying the A1 and, if present, A2 criteria formed the evening hyperphagia (EH) subgroup, while patients satisfying only the A2 core criterion constituted the nocturnal ingestion (NI) subgroup (Table 1). All patients were free of medication for at least one week prior to VPSG and gave their written consent for the study.

2.2. VPSG recording

All patients underwent a full-night VPSG. The sleep laboratory was equipped with food and drinks on a dinner table next to the bed, with items chosen and brought in by the patients themselves. VPSG included electroencephalogram (EEG) (C3-A2; C4-A1; O2-A1); right and left electrooculogram; chin superficial electromyogram; surface electromyogram of orbicularis oculi, orbicularis oris, masseter, sternocleidomastoideus, biceps brachii, and tibialis anterior muscles; chest and abdominal respirograms; and oxygen saturation. Recordings were done with a synchronized closed-circuit audio-video monitoring beginning at dinnertime and ending at the morning wake-up. Light-out time was based on individual habitual bedtime ranging between 22:00 and 23:30. Sleep stages, arousals with arousal index, PLMS with PLMS index, and apnea-hypopnea index (AHI) were scored according to the standards of the American Academy of Sleep Medicine (AASM) criteria [19].

Recordings were reviewed identifying all eating episodes. For each episode, sleep stage of onset, eating latency (the time delay

Table 1
Patients' answers to the items of the newly proposed NES diagnostic criteria (A-F), divided into two subgroups: Evening Hyperphagia group (EH) and Nocturnal Ingestion group (NI) [10].

	EH	NI
A1) At least 25% of food intake is consumed after the evening meal	10	0
A2) At least two episodes of nocturnal eating per week	8	10
B) Awareness and recall of evening and nocturnal eating episodes are present	10	10
C1) Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week	8	6
C2) Presence of a strong urge to eat between dinner and sleep onset and/or during the night	10	10
C3) Sleep onset and/or sleep maintenance insomnia are present four or more nights per week	9	2
C4) Presence of a belief that one must eat in order to initiate or return to sleep	6	5
C5) Mood is frequently depressed and/or mood worsens in the evening	7	3
D) The disorder is associated with significant distress and/or impairment in functioning	10	3
E) The disordered pattern of eating has been maintained for at least 3 months	10	10
F) The disorder is not secondary to substance abuse or dependence, medical disorder, medication, or another psychiatric disorder	10	10

between awakening to food intake), eating duration (the time between eating onset to eating offset) and sleep latency after eating offset (the time delay between eating offset to resume sleep) were calculated. Total episode duration was defined as the time between the awakening followed by eating and the resumption of sleep after eating offset.

2.3. Statistical analysis

Continuous variables are presented as range and mean ± standard deviation (SD), and categorical variables as absolute frequency and relative frequency (%). Comparison between EH and NI subgroups were made by Wilcoxon Signed Ranks Test. Statistical significance was set at $p < 0.05$. Analyses were performed using Stata software, version 12 (Stata Corp, College Station, TX, USA).

3. Results

3.1. Clinical data

The EH subgroup comprised 10 patients (seven women and three men; mean age: 56 ± 5 years, range: 46–63; mean BMI: 37 ± 8 kg/m^2, range: 28–53). Neurological and neuroradiological examinations were normal in all of them. The mean age of EH onset was 44 ± 11 years (range: 21–59). In six patients, EH onset was closely related to a particularly stressful life event, including pregnancy and menopause. The average duration of symptoms was 13 ± 11 years (range: 2–41). Eight patients reported EH every night, reporting to be fully awake and conscious during the eating episodes. All patients except one were obese: three moderately (BMI: 30–34 kg/m^2), three severely (BMI: 34–39.9 kg/m^2) and three extremely (BMI ≥ 40 kg/m^2). Four patients reported suffering from hypertension, two from diabetes and two from previous myocardial ischemia. Seven patients reported depressed mood and anxiety and nine reported insomnia. None of the patients had a history of NREM parasomnias. Two patients presented a positive family history for NREM parasomnias and three for nocturnal eating episodes.

The NI subgroup included 10 patients (six women and four men; mean age: 39 ± 11 years, range: 28–64; mean BMI: 24 ± 3 kg/m^2, range: 21–29). Neurological status and neuroimaging were normal in all of them. The mean age of NI onset was 28 ± 5 years (range: 20–36), and the mean duration of symptoms was 10 ± 8 years (range: 1–23). In four patients, NI appeared closely to a particularly stressing life event. All patients reported NI every night, often many times per night, reporting to be fully awake and conscious during the nocturnal episodes. Only four patients out of 10 were overweight (BMI, 25–30 kg/m^2). One patient suffered from hypertension; three reported depressed mood and two of them maintenance insomnia. Four patients presented a positive family history for nocturnal eating. The comparison between the two subgroups showed EH patients were significantly older ($p = 0.007$) with a higher mean BMI ($p = 0.0006$) and a later eating disease onset ($p = 0.009$). Clinical data are summarized in Table 2.

3.2. Polysomnographic data

On average, sleep efficiency (SE) was reduced, NREM light sleep was increased and REM sleep decreased in both subgroups. Mean REM sleep latency was increased, especially in EH patients (Table 3). The mean PLMS index was pathological in both subgroups (normal value, NV < 15) but only five EH patients and two NI patients had a PLMS Index > 15. No patients showed symptoms related to RLS nor presented any recurrent activity of the masseter and orbicularis oris muscles [20]. Six EH patients presented with OSAS: four mild (AHI <15), one moderate (AHI >15) and one severe (AHI >30). Only 2 NI patients had a mildly pathological AHI. Polysomnographic data are summarized in Table 3.

3.3. Eating episodes

We recorded 20 eating episodes, seven in EH patients and 13 in NI patients. All NI patients presented at least one eating episode during VPSG while eating episodes occurred only in five out of 10 EH patients. In the EH subgroup, three eating episodes occurred before sleep onset. In particular, one 6-minute-long episode occurred after dinner, before the lights off signal; the other two (lasting 6 and 8 minutes respectively) occurred during relaxed wakefulness, respectively at 56 and 109 minutes after lights off. The remaining four eating episodes occurred after an awakening from sleep (one episode from NREM stage 1 sleep, two episodes from NREM stage 2 and one from REM sleep). The mean eating latency after awakening from sleep was 12 ± 7 minutes (range: 3–20), the mean eating duration was 7 ± 2 minutes (range: 5–10) and the mean sleep latency after eating offset was 31 ± 15 minutes (range:

Table 2
Patients' clinical data divided into two subgroups: Evening Hyperphagia group (EH) and Nocturnal Ingestion group (NI).

	EH	NI
Patients (n)	10	10
Male (n)	3	6
Female (n)	7	4
Age (mean ± SD)	56 ± 5	39 ± 11
BMI (mean, Kg/m^2 ± SD)	37 ± 8	24 ± 3
Age at onset (mean ± SD)	44 ± 11	28 ± 5
Symptoms duration (mean ± SD)	13 ± 11	10 ± 8

n, number; SD, standard deviation.

Table 3
Sleep parameters and features of the eating episodes occurring after sleep onset in Evening Hyperphagia (EH) and Nocturnal Ingestion (NI) patients' group.

	EH	NI	p
Total sleep time, min	290 ± 105 (84–419)	297 ± 46 (201–348)	0.743
Time in bed, min	444 ± 152 (222–688)	383 ± 26 (362–433)	0.624
Sleep efficiency, % (NV > 85)	66 ± 21 (35–95)	75 ± 10 (55–86)	0.513
WASO, min	70 ± 41 (19–141)	86 ± 35 (48–155)	0.390
Sleep latency, min (NV < 30)	22 ± 40 (1–134)	7 ± 8 (1–28)	0.388
Sleep Stage 1, % (NV 2–5)	13 ± 5 (5–22)	16 ± 7 (4–28)	0.347
Sleep Stage 2, % (NV 45–55)	53 ± 12 (22–67)	46 ± 7 (34–54)	0.102
Sleep Stage 3, % (NV 15–25)	22 ± 14 (7–55)	23 ± 13 (8–41)	0.967
Sleep Stage REM, % (NV 20–25)	10 ± 6 (1–23)	12 ± 5 (1–21)	0.374
REM latency, min (NV 60–120)	183 ± 99 (71–374)	105 ± 58 (43–246)	0.122
Arousal Index (NV < 15)	14 ± 6 (3–22)	14 ± 9 (4–28)	0.870
PLMS Index (NV < 15)	19 ± 27 (0–80)	21 ± 18 (0–46)	0.563
AHI (NV < 5)	9 ± 10 (0–34)	4 ± 3 (0–10)	0.325
Eating latency, min	12 ± 7 (3–20)	1.4 ± 0.12 (0.07–3)	0.003*
Eating duration, min	7 ± 2 (5–10)	5 ± 3 (1–12)	0.147
Sleep latency after eating offset, min	31 ± 15 (18–55)	10 ± 15 (1–57)	0.009*
Total eating episode duration, min	46 ± 16 (28–66)	18 ± 15 (4–57)	0.012*

NV, normal value; REM, rapid eye movement; PLMS, Periodic Limb Movements during Sleep; AHI, Apnea–Hypopnea Index; * = statistical significance $p \leq 0.05$; values are expressed as a mean ± standard deviation; the range is in the brackets.

18–55) (Fig. 1). The mean total episode duration was 46 ± 16 minutes (range: 28–66) (Table 3).

All 13 NI episodes occurred after an awakening from sleep (one from NREM stage 1 sleep, eight from NREM stage 2 and four from NREM stage 3). Mean eating latency was 1.4 minutes ±7 seconds (range: 4 seconds-3 minutes); mean eating duration was 5 ± 3 minutes, (range: 1–12) and the mean sleep latency after eating offset was 10 ± 15 minutes (range: 1–57). The mean total episode duration was 18 ± 15 minutes (range: 4–57) (Table 3).

In both subgroups, patients were fully conscious during the eating episodes, clearly identified and recalled their eating episodes as usual. During the entire episodes, EEG was characterized by normal alpha activity with electrooculogram and heart and breathing rate typical of wakefulness. Patients were questioned by technicians, who were always present during the VPSG, whenever eating occurred and they all correctly answered to the questions on the episodes, on the time of occurrence, on the place in which they were, and on the reasons for eating. The technicians used a pre-prepared list of questions. All the patients clearly identified and recalled all of their eating episodes the morning after when, before stopping the video recording, the technician, as usual, actively but generically asked to the patients what happened during the night.

Eating and manipulation of food during the episodes was appropriate. No binge behaviors were detected. In particular, as usually reported by the patients, during the recorded eating episodes, we did not observe any rapid and/or large ingestion of food nor purging during or after VPSG. Only in the EH group, patients often performed other actions besides eating, such as doing crosswords, getting up and walking, also outside the recording room, or stretching (Fig. 2).

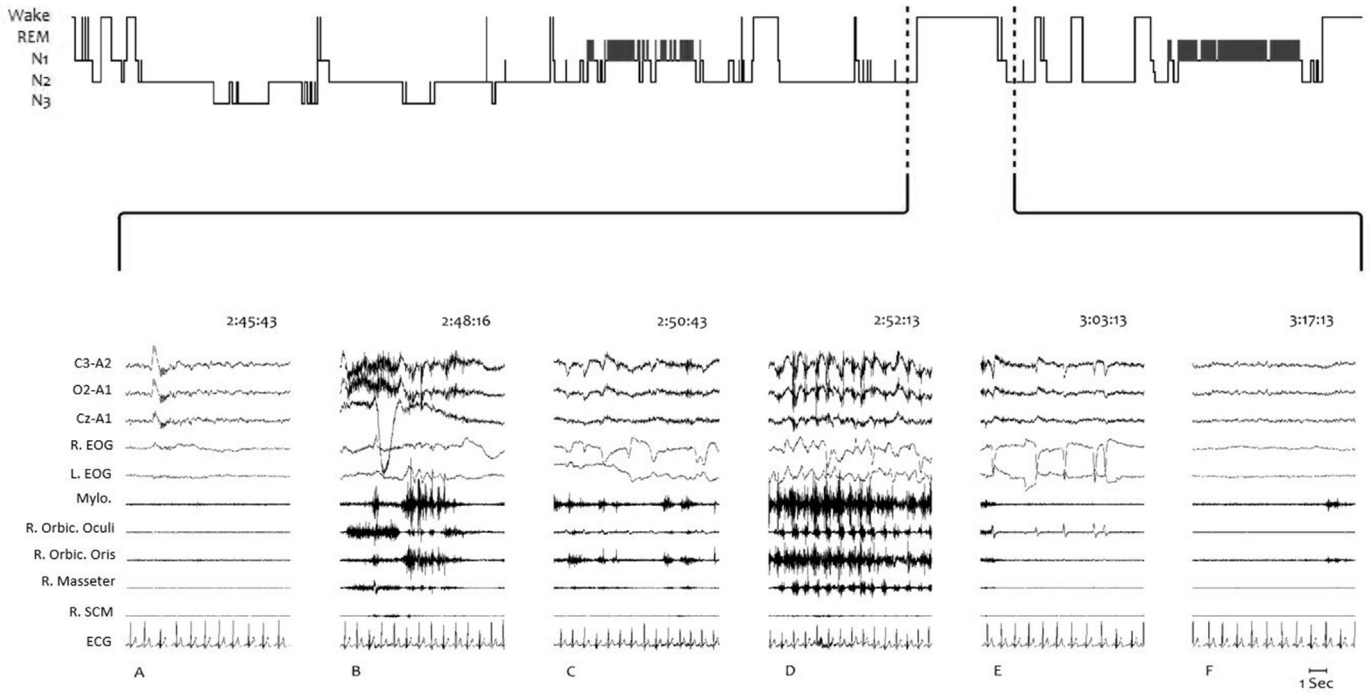

Fig. 1. Hypnogram (top) and polysomnographic excerpts (bottom) of a nocturnal eating episode in a patient with Evening Hyperphagia (EH). Dotted and full black lines represent the onset and offset of the eating episode. Hypnogram shows frequent awakenings and a prolonged REM sleep latency. Polysomnographic excerpts documented a prolonged eating latency from the awakening (B) from NREM stage 2 of sleep preceding the episode (A) and the beginning of eating (D).

Comparing the episode features in the two subgroups, the mean eating latency and the mean sleep latency after eating offset were significantly longer in the EH subgroup ($p = 0.003$ and $p = 0.009$ respectively). In addition, the total episode duration was significantly longer in the EH subgroup ($p = 0.012$), although the eating duration was similar in both ($p = 0.147$) (Table 3).

4. Discussion

NES has been described as a combination of an eating disorder, a sleep disorder and a mood disorder [21,22]. Our VPSG data of a case series of 20 patients diagnosed with NES according to the most recent criteria [10] seemed to confirm the existence of at least two different subtypes of NES: the EH and NI. The EH subgroup comprised patients who ate especially between dinner and sleep onset, but who could also eat during the night. The NI subgroup included patients who ate exclusively during an awakening from sleep.

The two subgroups showed different VPSG features. In EH patients, VPSG documented a long eating latency after infra-sleep awakenings and a long sleep latency after eating offset. On the other hand, patients of the NI subgroup presented eating episodes only after having fallen asleep and never before sleep onset, consumed food briefly within awakenings, and showed shorter sleep latencies after eating offset. From a clinical point of view these data could be of relevance. In fact, although episodes of eating during the night represent a common feature of EH and NI subgroups of patients overlapping the two conditions, they present with different features. The overlap in fact could be overcome asking the patient not only if nocturnal awakenings for eating are present but also gathering information on the features of the episodes. In particular, it is necessary to investigate the time delay between awakening from sleep and the beginning of eating and the time delay between the end of eating and the sleep latency after eating offset. EH patients have a longer eating latency, total episode duration, and sleep latency after eating offset than NI. Moreover, during the eating episodes EH patients perform other actions besides eating, such as walking around in the different rooms of the house, also before eating.

Of interest, this EH pattern might be attributed to a sleep-onset and sleep-maintenance insomnia, according to the hypothesis reported by Manni and colleagues [23]. The authors identified two different types of nocturnal eating behaviors (excluding nocturnal eating in the context of daytime eating disorders, eg, binge eating): (1) nocturnal eating that shared the features of the typical eating behavior of NES, namely compulsive feeding shortly after an infra-sleep awakening; and (2) nocturnal eating consisting of sporadic intake of food at variable distance from infra-sleep awakenings (aspecific nocturnal eating). Even in their experience, the eating behavior of these latter cases seemed more similar to a "killing time" activity, resembling the typical behaviors of insomniacs trying to recapture sleep (eg, doing crosswords, getting up and walking, stretching, etc.).

Alternately, the NI pattern of ingestion holds some of the characteristics typical of an arousal disorder in which the eating behavior is due to an “inner” desire facilitating eating episodes just after an awakening from sleep. It is our experience that in predisposed people sleep may sometimes become a favorable time for the emergence of complex, apparently goal-directed behaviors, characterized by compulsive features (eg, searching for food or smoking) [24,25].

Fig. 2. A frame of photographic sequences of nocturnal eating episodes in a patient with Eating Hyperphagia (EH). Patient is sleeping before the eating episode (02:45); after the awakening she calls the technician for assistance (02:47) leaves the bed and stays outside the recording room for some minutes (02:48 to 02:50). Then she comes back, sits on the bed looking at the food and starts eating (02:52). At the end, the patient lays down resuming a quiet posture before sleeping (02:56).

Our NI ingestion group was also similar to the patients described by Spaggiari and colleagues [26], in whom the interval between awakening and chewing start was shorter than 30 seconds in 50% of the episodes with a prompt return to sleep after food intake. As in the cohort described by Spaggiari and colleagues, in our setting all eating episodes occurred during EEG-defined wakefulness, and patients, when questioned, appeared to be awake and conscious, even when the delay between waking up and eating was very short [26]. Indeed, dissociation of states were never observed in our recordings, not even during the eating episodes.

In our case series, sleep macrostructure was substantially normal, all sleep stages were represented and there was a relatively normal cycling of the different sleep phases. Sleep structure was characterized by a prevalence of light NREM sleep and reduced SE. Of note, almost all our EH patients (in comparison to only two out of 10 NI patients) were positive for the criterion of sleep-onset and/or sleep-maintenance insomnia $\geq$4 nights per week (Table 1).

Finally, EH and NI subgroups also differ for demographic and clinical characteristics, being NI patients younger, not necessarily obese and with a shorter disease duration. Thus, even if NES has classically been associated with increased food intake causing unwanted weight gain and obesity [27,28], this finding was not completely reproducible in our cases. However, the different eating patterns of our subgroups was not investigated in detail in this work, and it deserves consideration in future studies. Some authors did report that the association between NES and obesity remains controversial, finding no relationship between the two conditions [5,6]. Moreover, the general presence of NES does not help identify obese individuals with specific medical complications [29]. Additionally, patients with NES are significantly more likely to meet the diagnostic criteria for depression and anxiety disorders [29], especially in the evening [3,30]. The lack of any psychiatric evaluation including a detailed psychometric assessment to provide a psychopathological profile of the NES patients was a relevant limitation of our study. Further studies are warranted to provide these evaluations especially considering the potentially different NES subtypes in larger groups of patients. Finally, we cannot exclude that a possible "first-night effect" could have influenced our VPSG results, including possible differential first-night effects on the two subgroups of NES patients, which should be explored in future studies.

5. Conclusions

NES is an abnormal eating behavior, which appears to segregate to the evening or sleeping hours. NES may indeed coexist with BED. This was not true in our series, corroborating the hypothesis that potentially different NES phenotypes might actually exist. In particular, we identified EH and NI subgroups. The EH subgroup comprises patients who ate especially between dinner and sleep onset, but who could also eat during the night. The NI subgroup included patients who ate exclusively during an awakening from sleep. Although nocturnal eating episodes represent a common overlap between the two subgroups of patients, our VPSG recordings demonstrated that eating episode features, especially eating latency, episode duration and sleep latency after eating onset did significantly differ between EH and NI patients. EH patients show a longer eating latency, a longer total episode duration and a longer sleep latency after eating offset than NI. Moreover, during the eating episodes EH patients performed other actions before and besides eating (eg, walking around and stretching, exhibiting proneness to insomnia).

We recommend searching these items actively in order to improve the clinical evaluation of NES patients. Instrumental data might be a useful clinical tool to segregate patterns within NES subtypes, and we recommend using the added value of VPSG data. As previously suggested [29], identifying diverse NES subtypes could impact on treatment and follow-up; as an example, EH patients might respond more promptly to a combined pharmacologic and cognitive-behavioral approach, whereas these strategies could not be equally effective in NI patients. Due to the small sample size and lack of any psychiatric evaluation providing a psychopathological profile of NES patients our conclusions must be considered preliminary and further studies must be carried out to confirm them.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Acknowledgements

We thank Cecilia Baroncini for language editing.

Conflict of interest

Dr. Loddo, Dr. Zanardi, Dr. Caletti, Mr. Mignani, Dr. Petroni and Dr. Chiaro report no disclosures.

Dr. Provini received honoraria for speaking engagements or consultancies from Sanofi, Bial, Fidia, Vanda Pharmaceuticals Ltd, Zambon, Eisai Japan and Italfarmaco.

Prof. Marchesini received honoraria from Sanofi, Merck Sharp & Dome, participated in the advisory Board of Lilly, Gilead, has received research support from Sanofi, Lilly, Novo Nordisk, Gilead, Genfit, Janssen, Glaxo.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2019.06.018.

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