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Journal search and commentary

Article reviewed: Behavioral and neuroendocrine characteristics of the night-eating syndrome[☆]

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Category

Insomnia, parasomnia

Objectives

(a) Determine behavioral features to establish the diagnosis of nocturnal eating syndrome (NES).

(b) Evaluate neuroendocrine characteristics of NES.

Study design

Two separate studies, behavioral and neuroendocrine, both use between group comparisons of NES patients vs. controls without NES matched for weight, age and sex.

Study population

NES subjects were selected from all responding to newspaper adds who met the same criteria in both studies: morning anorexia, evening overeating (>50% of daily food intake after evening meal but before end of sleep period), and insomnia. Subjects were excluded with serious emotional or physical illness including diabetes but not obesity.

Study samples

Behavioral study: ten NES patients and ten matched controls all overweight, mostly female.

Neuroendocrine study: 12 NES subjects (seven overweight) and 21 controls (ten overweight) all female.

Methods

Behavioral study: Motion sensors documented nocturnal awakenings and a diary was used to determine subjective mood and calories consumed by time of day.

Neuroendocrine study: After an overnight fast subjects were studied for 24 h in a clinical research ward with blood samples taken every 2 h from an indwelling catheter for glucose, insulin, melatonin, leptin and cortisol. Meals and light exposure were carefully controlled and matched for the groups.

Results

The behavioral study found that night eaters compared to controls had many more awakenings from sleep most (52%) with eating, ate somewhat more (about 25%) total daily calories with slightly

[☆] Birketvedt GS, Florholmen J, Sundsfjord J, Østerud B, Dinges D, Bilker W, Stunkard A. Behavioral and neuroendocrine characteristics of the night-eating syndrome. J Am Med Assoc 1999;282:657–663.

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less consumed in the day and considerably more after dinner time (74 vs. 37% of daily intake) with a high carbohydrate to protein ratio (7:1) for nocturnal eating. Mood was lower and decreased more at nighttime for night eaters.

The neuroendocrine study found that night eaters compared to controls had more awakenings from sleep with a significant decrease in normal increases in melatonin and leptin during the night and overall higher cortisol levels except for the early morning (04:00 and 06:00 h). There were no differences for phase nor for pre- and post-prandial glucose and insulin.

Conclusion

The authors concluded that the criteria identified a diagnostic syndrome with both disordered eating, sleep, and possibly mood accompanied by a matching loss of neuroendocrine changes which could lead to disrupted nocturnal sleep (melatonin) and less nocturnal appetite suppression (leptin). The authors argue that the corticotropin-releasing hormone (CRH) may be increased by the less than expected leptin increases at night and that the increased CRH may contribute to the melatonin decreases at night. A model is proposed for stress exacerbating the disorder by increasing CRH.

Comment

Despite the relatively small sample sizes in these two studies the agreement in the results for patients diagnosed in the same manner from two separate populations provides impressive support for this as a distinct diagnostic category. This must be compared to the Nocturnal Eating Syndrome defined in ICSD diagnostic coding manual of the American Academy of Sleep Medicine as a disorder where after a nocturnal awakening eating is required before the patient is able to return to sleep. This diagnosis has been focused mostly on young children who are nursing. It has been somewhat awkwardly extended to include adults where the compulsion to eat before returning to sleep is documented and usually occurs with partial amnesia for the nocturnal eating. But this disorder in adults should probably be considered one of the arousal disorders and classified as a parasomnia. In contrast to these other two disorders, the requirement of eating for return of sleep was not included in the diagnosis used in this study. The behavioral study showed that almost half of the large number of nocturnal awakenings occurred with a return to sleep without eating and in the neuroendocrine study nocturnal eating was not allowed. Thus this study documents for adults a night eating syndrome which has different behavioral characteristics than that seen for the nocturnal eating disorder in children. Both the behavioral and neuroendocrine studies indicate that the night-eating disorder described in this article is a circadian rhythm disorder more than arousal or extrinsic sleep disorder. The relationship between these three conditions: nocturnal eating disorder in children, night eating in adults, and the special cases of adults who have some amnesia for nocturnal events remain to be explored further but at this point it seems likely they represent three separate disorders, one an extrinsic sleep disorder, another a disorder of circadian rhythms and the last a parasomnia or arousal disorder.

The major problems with these studies are the small sample sizes and for the endocrine study the changes introduced by the forced change in eating times for the night eaters but not controls. The neuroendocrine differences do not establish causation, since it is not clear whether these cause or result from the disorder, but the results offer potential considerations for future evaluations and treatments. Overall, this article defines for sleep medicine an adult circadian disorder of eating and sleep deserving of consideration especially when evaluating insomnia patients.