Arousal Reactions in Sleepwalking and Night Terrors in Adults: The Role of Respiratory Events

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Study Objectives: The aim of the study was to determine the role of respiratory events, assessed by means of esophageal pressure monitoring, during arousals from slow wave sleep in adult patients with parasomnias.

Design: N/A

Setting: N/A

Patients: Ten patients with parasomnias (sleepwalking, night terrors, or both) and 10 control subjects matched for gender and age underwent 3 consecutive nights of polysomnography.

Interventions: N/A

Measurements and Results: By increasing sleep fragmentation, esophageal pressure monitoring has a deleterious effect on sleep architecture in patients with parasomnias and in control subjects. Respiratory events occur more frequently in parasomniacs than in controls. Respiratory effort seems to be responsible for the occurrence of a great number of arousal reactions in parasomniacs and is involved in triggering the parasomnia episodes.

Conclusion: Sleep-disordered breathing seems to be frequently associated with parasomnias during slow wave sleep, emphasizing the utility of performing esophageal pressure monitoring in cases of sleep walking or night terrors.

Keywords: Sleep walking, night terrors, parasomnias, respiratory events, esophageal pressure.

INTRODUCTION

SLEEP WALKING AND NIGHT TERRORS ARE PARASOMNIAS CHARACTERIZED BY A SUDDEN AROUSAL FROM SLOW WAVE SLEEP (SWS), EITHER IN THE FIRST OR IN SUBSEQUENT SLEEP CYCLES.[1-4] This arousal is associated with a dissociated reaction between cortical activity and an elaborated motor activity in sleepwalking and between cortical activity and an autonomic discharge, including tachycardia, tremor, and sweating, in night terrors. The increase of the sleep density, assessed by the slow wave activity [power density of the electroencephalographic (EEG) delta band between 0.75 Hz and 4.5 Hz], during the SWS episode preceding the occurrence of a parasomnia may partially explain this dissociation.[4] To occur, SWS parasomnia requires not only a pressure increase in the SWS, but also an enhancement in the intervening SWS arousal. The sleep of patients who sleep walk and have night terrors is fragmented by frequent brief arousals or microarousals that occur primarily during SWS.[4-9] These frequent SWS interruptions, which cause the patient to be unable to sustain deep sleep (even after 38 hours of sleep deprivation[3]) can be considered as a polysomnography (PSG) characteristic of sleepwalking and night terrors.[10-11]

Two case reports[12-13] have described the use of nasal continuous positive airway pressure resulting in sleepwalking or night terrors caused by SWS rebound. A previous report[14] postulated that SWS parasomnia could be triggered by sleep apnea. Moreover, sleepwalking and night terrors occur significantly more often in children with sleep apnea than in normal children,[14-16] so it may be assumed that respiratory events could be responsible for the fragmented sleep of parasomniac patients.

SUBJECTS AND METHODS

Subjects

Patients with parasomnias were recruited at the sleep disorders outpatient clinic of the University Hospital; control subjects were recruited through public advertisement. The Ethics Committee of the University Hospital approved the study, and all subjects signed a consent form and were paid for their participation.

The diagnosis of sleepwalking or night terror was determined according to the *International Classification of Sleep Disorders*.[1] To be classified as parasomniac, the subjects must have reported sleepwalking, night terrors, or both several times a month and had a sleep history of sleepwalking or night terrors that was confirmed by a third person (bed partner or parent) or by the general practitioner. To be included in this study, patients should previously have had at least one episode of parasomnia documented by video and PSG. Ten parasomniacs, 4 male and 6 female aged 22 to 40 years (28.6 years ± 1.8) free of psychotropic drugs for at least 1 month, were selected for the study. Frequency of reported sleepwalking or night terrors episodes were either several times a month (1 subject), several times a week (5 subjects), or every night (4 subjects) since childhood or adolescence (mean age of onset = 7.8 years ± 1.2 years). Mean body weight was 23.2 ± 0.8 kg/m² (range 19.2-25.9 kg/m²). Ten normal subjects who were "good sleepers" served as controls; during a selection night of PSG, they exhibited apnea and periodic limb movement indexes less than 5 and a sleep efficiency index greater than 90 %. They were sex and age (28.4± 2.01) matched to parasomniacs. None were obese (body mass index less than 26), and none had taken psychotropic drugs for at least 1 month.

METHODS

Polysomnography

Each subject underwent 3 overnight PSG recordings in the sleep laboratory. Subjects went to sleep at their usual bed time (10:30 PM to 12:00 PM) and were awakened at their customary time of arousing (7:00 AM to 8:00 AM). The first night was a selection and habituation night: PSG was performed with 8 EEG channels in order to reveal possible epileptic patterns. The second night served as a baseline night in order to assess pos-

sible effects of esophageal pressure monitoring on sleep parameters and was performed with limited montage: 2 EEG channels (C3/A2 and C4/A1), 1 chin electromyogram channel, 2 electrooculogram channels, and 1 electrocardiogram channel. The third night was devoted to the assessment of respiratory events. Measurements of thoracic and abdominal movements were performed using piezoelectric bands, and oral airflow, using of thermocouples. Respiratory effort was assessed by means of esophageal pressure monitoring

Esophageal pressure monitoring

Esophageal pressure monitoring was performed using a catheter mounted with a esophageal pressure transducer[17] (Gaeltec LTD, Scotland). A 2.1-millimeter silicone tube containing one pressure transducer was carefully inserted through one nostril into the pharynx and esophagus. The pressure transducer was placed in the esophagus approximately 35 centimeters from the nostril.

Sleep parameters analyses

Visual scoring of sleep stages was carried out according to Rechtschaffen and Kales[18] criteria, with the assistance of an integrated digital filtering analysis system (Neurop CEA-INSERM), which was particularly useful in determining the proportion of each EEG band per epoch and allowed for more reliability in scoring sleep stages, particularly for SWS.[19]

Arousal reactions

Arousal reactions were computed according to American Sleep Disorders Association criteria.[20] The main features of arousal scoring included:

- An EEG arousal was characterized by an abrupt shift in EEG frequency that included a combination of theta, alpha, and frequencies greater than 16 Hz but no spindles.
- At least 10 seconds of continuous sleep were needed before the arousal reaction.
- The arousal reaction must have lasted at least 3 seconds and less than 15 seconds.
- The occurrence of simultaneous EMG activity together with the arousal reaction was necessary in rapid eye movement (REM) sleep only.

In addition, the total number of SWS interruptions was scored. The occurrence of one epoch scored as stage 0, or one arousal reaction, or one hypersynchronous high-voltage delta waves arousal (HSDWA) characterized the interruption of SWS. The HSDWA, a typical sleep EEG pattern observed in parasomnia, was defined as the occurrence of continuous hypersynchronous high-voltage (>150 µvolts) delta waves (1-3 Hz) in stages 3 and 4 sleep associated with increased muscle activity and a duration of at least 10 seconds.

Arousal reactions and HSDWA were scored separately by 2 of us (FE and AB); when differences occurred, the final decision was made after discussion.

Differences in arousal reactions and HSDWA between baseline night and esophageal pressure monitoring night were also studied in order to look for a possible effect of the esophageal catheter on the sleep of both parasomniacs and controls.

Respiratory events analyses

Respiratory events were analyzed exclusively according to esophageal pressure (Pes) measurements. The main features were defined as following:

- Pes crescendo was defined as the sequence of progressively more-negative peak end inspiratory pressure, terminated by a sudden change in pressure to a less negative level.
- To be counted, the crescendo must have lasted 10 seconds or longer (typically at least 3 breaths or more)
- Delta Pes crescendo was the difference between the values of the negative peak inspiratory pressure of the last breath (the largest Pes value) and the value of the negative peak inspiratory pressure of the first breath of the crescendo (the smallest value).
- The delta pressure was defined as the differential pressure between expiration and the more-negative peak end inspiratory pressure.

All respiratory events were captured through use of a DC cursor that easily identified the most-negative and most-positive digitized Pes data points for the identified breath.

Respiratory events were scored separately by 2 of us (FE and AB); when differences occurred, the final decision was made after discussion.
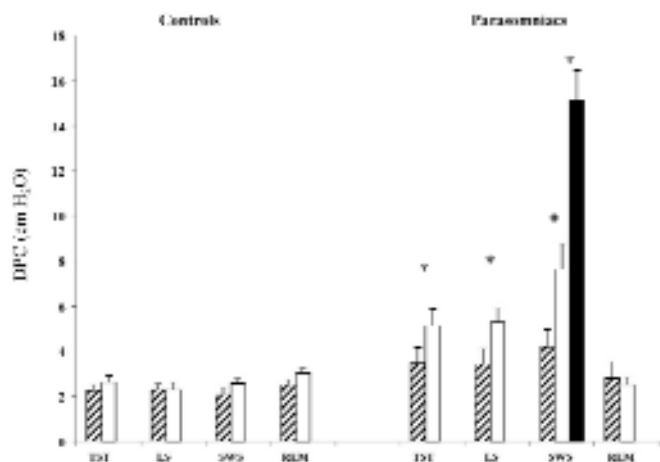
Sleep parameters	Controls Baseline (SEM)	EPM (SEM)	Parasomniacs Baseline (SEM)	EPM (SEM)	Group (p)	Anova Night (p)	Group* Night (p)
Sleep-onset latency	8.0 (2.2)	14.2 (2.3)	11.7 (2.9)	24.8 (12.2)	1.0 (ns)	2.94 (ns)	0.38 (ns)
Total sleep time	450.0 (17.5)	404.2 (12.5)	422.8 (18.6)	362.2 (20.3)	2.68 (ns)	17.44 (0.0006)	0.34 (ns)
Wake time after sleep onset	12.5 (1.9)	58.4 (15.5)	32.7 (7.8)	79.6 (11.6)	4.05 (0.05)	18.95 (0.0004)	0.01 (ns)
Sleep efficiency	95.6 (0.4)	87.7 (3.0)	92.6 (1.8)	81.7 (2.8)	3.86 (0.065)	17.6 (0.0005)	0.43 (ns)
Number of awakenings <1 min	11.3 (0.6)	16.4 (1.7)	11.4 (0.9)	24.7 (2.9)	6.18 (0.02)	25.16 (0.0001)	5.0 (0.03)
Number of awakenings >1 min	3.8 (0.6)	6.3 (2.2)	8.8 (1.7)	11.2 (1.5)	10.96 (0.004)	1.96 (ns)	0.01 (ns)
Arousal index	4.0 (0.2)	12.3 (0.4)	15.4 (1.2)	30.3 (1.3)	30.12 (0.0001)	22.23 (0.0002)	0.64 (ns)
Number of arousals from SWS	4.1 (1.0)	3.8 (0.5)	10.5 (2.8)	9.5 (1.4)	7.4 (0.01)	1.8 (ns)	0.5 (ns)
Number of HSDWA	0.2 (0.1)	0.1 (0.1)	5.1 (1.3)	3.9 (0.9)	0.6 (ns)	1.4 (ns)	0.2 (ns)
Stage 1 (min)	19.8 (0.7)	38.1 (5.7)	24.6 (3.1)	40.6 (5.1)	0.61 (ns)	23.69 (0.0001)	0.11 (ns)
Stage 2 (min)	239.0 (12.3)	181.3 (10.5)	197.2 (15.3)	159.6 (10.6)	4.47 (0.04)	29.13 (0.0001)	1.29 (ns)
SWS (min)	92.8 (8.6)	104.2 (6.7)	114.3 (16.5)	101.5 (7.8)	0.64 (ns)	0.01 (ns)	1.69 (ns)
REM sleep (min)	98.4 (12.9)	80.5 (6.9)	79.2 (10.8)	60.4 (8.9)	2.91 (ns)	4.56 (0.04)	0.01 (ns)
Stage 1 (%)	11.5 (4.7)	9.8 (1.8)	5.9 (0.9)	11.3 (1.4)	0.61 (ns)	0.42 (ns)	1.6 (ns)
Stage 2 (%)	53.0 (1.5)	44.7 (1.9)	47.5 (3.8)	44.4 (2.2)	1.22 (ns)	6.18 (0.02)	1.3 (ns)
SWS (%)	21.1 (2.2)	25.7 (1.3)	27.2 (3.8)	27.4 (1.4)	2.25 (ns)	1.24 (ns)	1.06 (ns)
REM sleep (%)	21.4 (2.2)	20.8 (1.5)	18.6 (1.8)	16.2 (1.9)	3.52 (ns)	0.73 (ns)	0.22 (ns)
REM sleep latency	79.8 (4.6)	96.5 (16.8)	87.5 (9.8)	142.1 (11.3)	4.94 (0.04)	10.59 (0.004)	2.99 (ns)

Table 1—Mean polysomnographic data in parasomniacs and in controls during base line (BL) and esophageal pressure monitoring night (EPM). Group = controls versus parasomniacs; Night = EPM night versus BL night; G*EPM = Group by Night interaction; SWS, slow wave sleep; HSDWA= hypersynchronous delta waves arousal; REM, rapid eye movement

Figure 1—Delta Pes crescendo (DPC) in controls and parasomniacs followed by an arousal (white bar), a parasomnia (black bar) or not followed by an arousal (hatched bar). TST = total sleep time, SWS = slow wave sleep, LS = light sleep (stages 1 & 2)
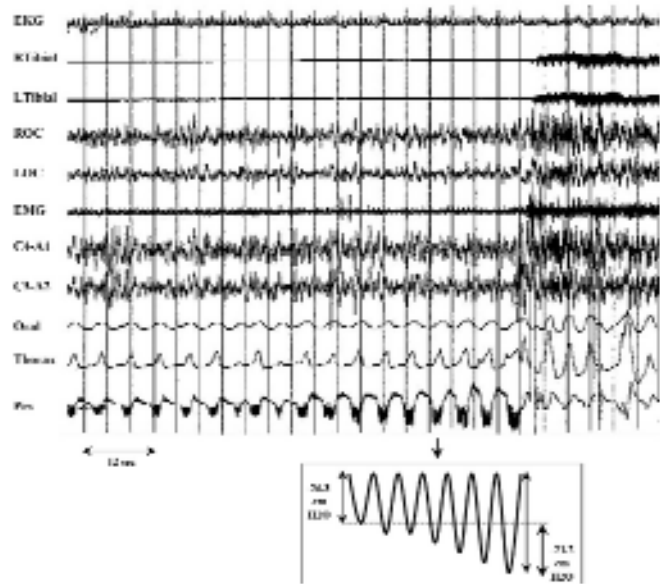

Figure 2—Recording of an episode of parasomnia. EKG = electrocardiogram, R tibial = right tibial electromyography, L tibial = left tibial electromyography, ROC = right ocular movement, LOC = left ocular movement, EMG = chin electromyography, C4-A1 and C3-A1 electroencephalography channels, Oral = oral airflow, Thorax = thoracic respiration, Pes = esophageal pressure.

	Controls	range	Parasomniacs	range	F	p
TST	5.6 (0.8)	(1.1-8)	22.9 (4.2)	(5.4-52.3)	16.79	< 0.01
With arousal (AR)	2.4 (0.4)	(0.5-4.6)	15.2 (3.2)	(2.6-40.6)	15.97	< 0.01
Without arousal (N-AR)	3.2 (0.5)	(0.6-5.9)	7.7 (1.7)	(0.9-15.7)	6.16	0.02
NREM sleep	4.9 (0.7)	(0.5-6.8)	24.1 (5.1)	(6.3-63.7)	13.74	< 0.01
With arousal (AR)	2.1 (0.3)	(0.3-3.6)	16.2 (4.1)	(3-50.3)	11.84	< 0.01
Without arousal (N-AR)	2.8 (0.5)	(0.2-5.1)	7.9 (1.8)	(0.6-15.5)	7.19	0.01
REM sleep	7.9 (1.6)	(0.8-14.8)	20.2 (3.6)	(1.5-34.6)	9.47	< 0.01
With arousal (AR)	3.6 (1.2)	(0-11.6)	11.6 (2.5)	(0-24.4)	8.52	< 0.01
Without arousal (N-AR)	4.3 (1.2)	(0-12.7)	8.6 (1.9)	(0.8-17.9)	3.41	ns

Table 2—Respiratory disturbance indexes associated (AR) or not associated with arousal (N-AR) during the total sleep time (TST), the non–rapid-eye-movement (NREM) sleep, and the rapid-eye-movement (REM) sleep.
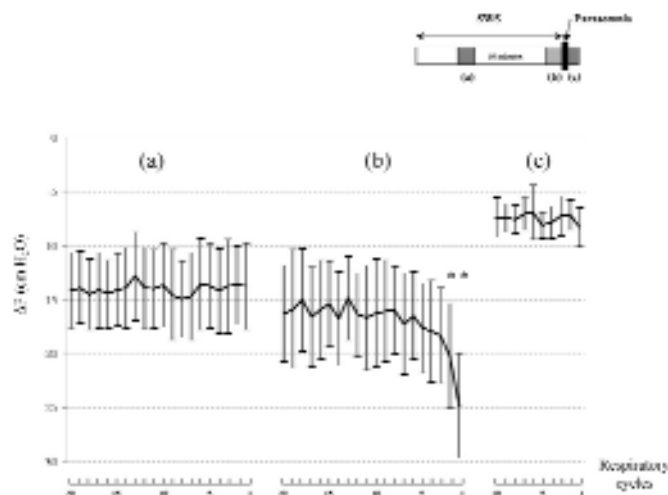
Statistical analysis

One-way analyses: group (controls vs parasomniacs), night (baseline night vs esophageal pressure monitoring night) and two-way analyses (group by night, group by arousal reaction), analysis of variance (ANOVA), and chi-square tests (comparison of respiratory disturbances between parasomniacs and controls) were performed using SAS/STAT software release 8.2.

RESULTS

Episodes of parasomnia

Six episodes of parasomnia were recorded in 6 of the 10 subjects during the esophageal pressure monitoring night—night terrors in 2 cases, sleepwalking in 1, and elementary episodes in 3. During these elementary episodes, subjects abruptly sat up in bed, looked around wildly, spoke unintelligibly, lay back down, and returned to sleep. All episodes of parasomnia occurred in SWS stage 4: three during the first non-REM (NREM) cycle, 2 during the second, and 1 during the third.

Finally, the EEGs of all patients revealed no epileptic activity, such as spikes or spikes and waves.

Polygraphic data

Baseline night: Table 1 summarizes parameters derived from visual computer-aided PSG scoring for the baseline night and esophageal pressure monitoring and ANOVA results comparing 2 groups: parasomniacs versus control subjects for both nights—the baseline night versus esophageal pressure monitoring night. The sleep of parasomniacs was characterized by major fragmentation. During the baseline night, the waketime after sleep onset (WASO), the number of awakenings greater than 1 minute, the number of arousal reactions per hour (arousal reaction index), and the number of arousal reactions from SWS were significantly increased, and the sleep efficiency was significantly decreased in parasomniacs compared to controls. The HSDWA were relatively frequent in parasomniacs and absent or very rare in controls. Stage 2 sleep was significantly decreased and the REM latency significantly increased in parasomniacs compared to controls.

Esophageal pressure monitoring night: Esophageal pressure monitoring enhanced sleep fragmentation in both parasomniacs and controls. The WASO, the arousal index, stage 1 sleep duration and percentage, and REM sleep latency were significantly increased. The total sleep time (TST), sleep efficiency, stage 2 sleep duration and percentage, and REM sleep duration were decreased; however, the number of awakenings that lasted less than 1 minute was increased only in parasomniacs, and the number of arousals from SWS and the number of HSDWA were not different in parasomniacs compared to controls.

Respiratory events: As shown in Table 2, all the respiratory event indexes except respiratory events not associated with arousal reactions in REM sleep were significantly higher in parasomniacs than in controls. The respiratory events associated with arousal reaction percentage was significantly increased in parasomniacs compared to controls either in TST (68.2% versus 42.5%; $X^2 = 6.89$; p = 0.0087) or in NREM sleep (68.8% versus 42.9%; $X^2 = 5.47$; p = 0.0193).

As assessed by the interaction between groups and arousal reaction (F value = 11.78; p = 0.02), the delta Pes crescendo was significantly increased in respiratory events associated with arousal reactions in parasomniacs. This increase was far more dramatic when the crescendo preceded an episode of parasomnia (Figure 1). Moreover, all episodes of parasomnia were preceded by a Pes crescendo, as documented by the example in Figure 2. The delta pressure was significantly greater during the last two breaths before the occurrence of the parasomnia than during the 20 breaths assessed in SWS 10 minutes before the parasomnia and during the 18 breaths assessed immediately before the occurrence of the parasomnia (Figure 3).

Figure 3—Delta pressure (P) values in parasomniacs measured 10 minutes before the occurrence of an episode of parasomnia (a), immediately before an episode of parasomnia (b) and immediately after an episode of parasomnia (C)
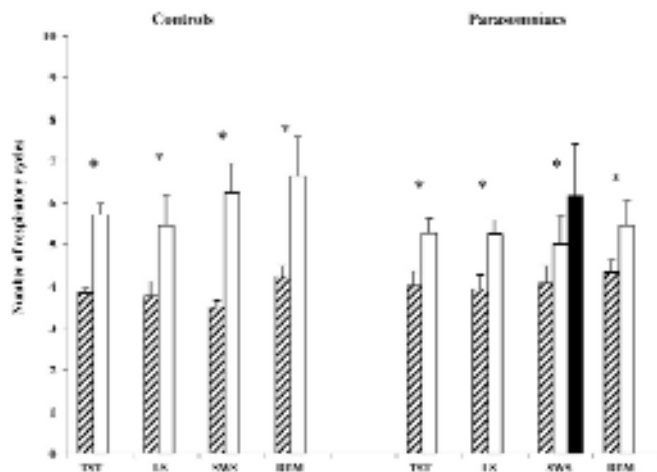

Figure 4—Number of breaths in esophageal pressure (Pes) crescendo preceding an arousal (white bar) or a parasomnia (black bar) or not preceding an arousal (hatched bars).

In both groups, the number of breaths was greater in respiratory events associated with arousal reaction events than in respiratory events not associated with arousal reaction events. However, as shown by the interaction between groups and arousal reaction (F value = 11.30; p = 0.005), the number of breaths in respiratory events associated with arousal reaction events was greater in controls (by 48%) than in parasomniacs (by 31%). The number of breaths in the Pes crescendo associated with parasomnias was significantly higher than in the Pes crescendo associated with arousal reactions only (Figure 4).

DISCUSSION

Esophageal pressure monitoring has a deleterious effect on sleep architecture by increasing sleep fragmentation in both parasomniac patients and control subjects. Esophageal pressure can be assessed during sleep by 3 methods: esophageal manometry via balloon,[23] esophageal manometry via water-filled catheter, [24] and catheter pressure transducers.[16] Two previous studies have examined the effect of esophageal pressure monitoring on sleep parameters. In one study, esophageal pressure monitoring was performed with catheter pressure

transducers[25] and in the other, with water-filled catheters[26]; no effect was observed on the sleep architecture of obstructive sleep apnea patients. The method we used for monitoring esophageal pressure (catheter pressure transducer) is identical to the one used in the first study and does not cause more discomfort than the method used in the second study. The main difference between our study and the previous studies may concern the population investigated. In our study, subjects were either "good sleeper" control subjects or parasomniac subjects with more fragmented sleep than normal subjects but far less than the apneic patients in the other studies. In both prior studies, sleep was more fragmented during the baseline night than in our study. For example, sleep efficiency was lower (78.1% and 83.4%) in prior studies[25-26] than in our study (95.6% in controls subjects and 92.6% in parasomniac subjects). If we look at the differences between control subjects and parasomniacs in our own study, we note that esophageal pressure monitoring increases the arousal index of controls (by 186.04 %) more significantly than that of parasomniacs (by 96.75 %). It can be supposed that in subjects with major sleep fragmentation, a floor effect can mask the impact of esophageal pressure monitoring.

Another important finding of this study is that respiratory events leading to arousals are more numerous in parasomniacs than in controls and are involved in the triggering of the parasomnia episodes. The respiratory effort—as evidenced by the delta Pes crescendo, the delta pressure, and the number of breaths—seems to be responsible for the arousal reactions in parasomniacs. The delta Pes crescendo is higher in respiratory events associated with an arousal reaction than in respiratory events not associated with arousal reaction, and delta pressure is increased during the last breaths preceding the occurrence of a parasomnia. A significant rise of the spectral delta band power density has been shown in upper airway resistance syndrome over the period from 6 to 2 seconds before the end of the respiratory event, and this rise has been interpreted as a compensatory mechanism of the central nervous system for permitting the sleep to continue.[22] Not only do episodes of parasomnia occur most often in SWS (6 out of 6 in our study), but they are also preceded by a significant increase in delta power density.[4,27] It may be supposed that abnormal sleep density, as observed in parasomniacs,[4] can be reinforced by respiratory events leading to a very high arousal threshold, which is responsible for the confused state that is characteristic of the parasomnia episodes. This mechanism cannot explain the occurrence of all episodes of somnambulism or night terrors, however, and the intervening arousal enhancement during SWS can be induced by several other factors: stressful situations,[28] alcohol use, and the presence of DQB1*05 HLA antigen.[29] Sleepwalking and night terrors are often seen in association with sleep disordered breathing, and the treatment of obstructive sleep apnea or upper airway resistance can reduce or eliminate episodes of parasomnia.[14] These results emphasize the utility of performing esophageal pressure monitoring in cases of sleep walking or night terrors.

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