Original Article

Electroencephalographic slow waves prior to sleepwalking episodes

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ABSTRACT

Objective: Recent studies have suggested that the onset of sleepwalking episodes may be preceded by fluctuations in slow-wave sleep electroencephalographic characteristics. However, whether or not such fluctuations are specific to sleepwalking episodes or generalized to all sleep–wake transitions in sleepwalkers remains unknown. The goal of this study was to compare spectral power for delta (1–4 Hz) and slow delta (0.5–1 Hz) as well as slow oscillation density before the onset of somnambulistic episodes versus non-behavioral awakenings recorded from the same group of sleepwalkers. A secondary aim was to describe the time course of observed changes in slow-wave activity and slow oscillations during the 3 min immediately preceding the occurrence of somnambulistic episodes.
Methods: Twelve adult sleepwalkers were investigated polysomnographically during the course of one night.
Results: Slow-wave activity and slow oscillation density were significantly greater prior to patients' somnambulistic episodes as compared with non-behavioral awakenings. However, there was no evidence for a gradual increase over the 3 min preceding the episodes.
Conclusions: Increased slow-wave activity and slow oscillation density appear to be specific to sleepwalking episodes rather than generalized to all sleep–wake transitions in sleepwalkers.

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1. Introduction

Sleepwalking, also known as somnambulism, is a non-rapid eye movement (NREM) parasomnia involving behaviors of varying complexity, usually initiated during arousals from N3 sleep, also known as slow-wave sleep (SWS) [1,2], during the first sleep cycle of the night [2,3]. The symptoms and manifestations that characterize sleepwalking can vary greatly both within and across patients, but most episodes are characterized by misperception and relative unresponsiveness to the environment, impaired judgment, perceived threat or agitation, and variable retrograde amnesia [4].

Whereas sleepwalkers' overall sleep architecture and cycling among sleep stages is essentially the same as that of controls [2,3,5], sleepwalkers show an unusually elevated number of spontaneous awakenings and electroencephalogram (EEG) arousals occurring out of SWS, even on nights without episodes [3,6,7]. Furthermore, sleepwalkers' increased number of arousals is limited to SWS as they do not show a greater number of awakenings from other sleep stages

in comparison with controls [3]. Other documented abnormalities in sleepwalkers' SWS include disturbances in sleep intensity as measured quantitatively by slow-wave activity (SWA: spectral power between 0.5 and 4.5 Hz) [3,5] as well as atypical patterns in the cyclic alternating pattern rate, a measure of NREM sleep instability [8–10]. Taken together, these findings suggest that somnambulism could be due to a dysfunction in SWS regulation (see [2] for a review).

Although a majority of polysomnographic (PSG) studies of sleepwalkers have examined general all-night sleep characteristics, increasing attention has been paid to the study of sleep EEG variables immediately preceding the onset of somnambulistic episodes. To date, the study of SWA and slow oscillations (SO; EEG waves >75 μV, <4 Hz) prior to episode onset appears to be the most promising in advancing our understanding of pathophysiological mechanisms underlying this parasomnia. One study of 11 patients with sleepwalking and/or sleep terrors (six with sleep terrors only) found that SWA during the 2 min immediately preceding a parasomniac episode is greater than SWA measured 10 min prior to the parasomniac episode [6]. SWA during the 2 min prior to a parasomniac episode was also greater than that during the 2 min preceding an arousal without behavioral manifestations. However, nine of the 15 episodes analyzed in this study were sleep terrors and there was no indication as to how many of the 15 episodes and 150 arousal reactions analyzed came from each of the 11 subjects investigated. It is thus possible

Table 1
Patient characteristics.

Patient	Gender	Age at recording	Episode frequency	Presence of concomitant sleep terrors	Presence of a positive family history of somnambulism
1	F	25.3	1/month	Possible	Yes
2	F	33.5	±daily	No	No
3	M	22.3	Daily	Nil	No
4	F	19.4	Daily	Yes	Possible
5	F	18.3	Two times per week	No	No
6	F	20.5	Daily	No	Yes
7	M	39.9	±Daily	Yes	Possible
8	F	32.1	Few times/month	No	No
9	F	35.2	Few times/week	No	No
10	M	19.2	Three or four times/week	No	Yes
11	F	22.2	Two times/week	No	Yes
12	F	40.5	Four or five times/week	No	Yes

that data from some subjects were given a disproportionate weight in comparison with others. A subsequent study [5] analyzed spectral power in delta (2.25–4 Hz) and in slow delta (0.75–2 Hz) bandwidths during the 32 s immediately preceding somnambulistic episodes in 12 adult sleepwalkers. They found an increase in slow delta power density during the 4–16 s (12 s total) preceding episode onset as compared with the 28–32 s segment preceding an episode. However, the statistical analyses focused only on the 4–16 s time window before the episodes, leaving out the 16–28 s time period. Moreover, analyses of SWA beyond 32 s prior to an episode may be necessary to detect more gradual changes in EEG signals. More recently, Jaar et al. [11] investigated the sleep EEG prior to somnambulistic episodes recorded out of SWS during daytime recovery sleep following 25 h of sleep deprivation, finding an increase in SWA during the 20 s prior to episode onset. This study was also the first to investigate SO in sleepwalkers. It revealed an abrupt increase in SO density in the 20 s immediately preceding episode onset. However, the specificity and generalizability of the study's findings were limited by the fact that the data were collected during daytime recovery sleep following 25 h of sleep deprivation and that no comparable control periods were investigated.

The aim of the present study was to investigate patterns of SWA and SO density prior somnambulistic episodes recorded during normal sleep in comparison with patterns observed prior to non-behavioral awakenings collected from the same subjects. PSG data collected during normal overnight recordings from adult sleepwalkers were used to compare EEG patterns observed during the 3 min preceding episode onset (a longer time window than previously investigated the literature) with those observed from normal awakenings without behavioral manifestations while focusing on fluctuations in the spectral power of SWA (0.5–1 Hz), delta (1–4 Hz) and slow delta (0.5–1 Hz) bandwidths and on SO density. It was hypothesized that SWA, delta, slow delta, and SO density would be greater before somnambulistic episodes than before non-behavioral awakenings collected from the same sleep stage and NREM sleep cycle. Since postulated increases in SWA may occur gradually, the time course of observed changes in SWA and SO during the 3 min preceding somnambulistic episodes was also investigated.

2. Methods

2.1. Subjects

Subjects were 12 adult sleepwalkers (three men, nine women; mean age, 27.4 years; SD, 8.4) referred to the Sleep Disorders Clinic of the Hôpital du Sacré-Coeur by their physician for suspected somnambulism. All patients reported a clinical history (including over the previous 6 months) of somnambulism that was not of a traumatic, neurological, or medication-induced origin, and received a final diagnosis of SW according to the International Classification of Sleep Disorders [4]. Exclusion criteria consisted of: (1) the presence of another sleep disorder [4] or an index (number per hour of sleep) >5 for respiratory events (apnea–hypopnea index) or >10 for periodic leg movements during sleep; (2) a history of neurological or psychiatric disorders; (3) a history of drug addiction or abuse; and (4) the use of medications that could influence the sleep EEG, sleep architecture, motor activity during sleep, or daytime vigilance. The 12 patients included in the present study were selected on the basis of having experienced, while undergoing a whole-night PSG in the sleep laboratory, at least one spontaneous somnambulistic episode in addition to one non-behavioral awakening during the same sleep stage within the same NREM period with a minimum of 5 consecutive minutes of sleep separating the two events. The study was approved by the hospital's ethics and scientific committee, and informed consent was obtained from each patient. Table 1 presents the clinical characteristics of these 12 patients.

2.2. Materials

PSG recordings were conducted on a 32-channel Grass polygraph (sensitivity, 7 μV/cm; bandpass, 0.3–100 Hz). Signals were digitized at a sampling rate of either 256 Hz using commercial software (Harmonie, Stellate Systems, Montréal, QC, Canada). EEG recordings and electrode placement were performed according the 10–20 system (Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6, Fz, Cz, Pz; linked ears), with left and right electro-oculogram, chin EMG, pulse oxymetry, nasal/oral thermistor, and electromyogram leg electrodes. All patients were continuously videotaped during sleep assessment and sleep stage data scored according to established criteria [12].

All behavioral manifestations arising out of patients' SWS (stage N3) were visually inspected on the accompanying time-synchronized video recordings and attention paid to behavioral episodes characterized by clumsy, stereotyped or repetitive movements, confusion, agitation or disorientation during the event, and episodes accompanied by (but not limited to) somniloquy. The selected somnambulistic episode occurred during the first NREM period for nine participants and in the second NREM period for the other three subjects. For each patient, one non-behavioral awakening was selected that matched the patient's episode occurrence in terms of sleep cycle and sleep stage (with the restriction that a minimum of 5 min of continuous sleep separated the two events). Non-behavioral awakenings were defined as a transient interruption of sleep, identifiable when ≥50% of an epoch contained alpha (8–13 Hz) activity or low-voltage, mixed (2–7 Hz) frequency activity [12]. In five cases the somnambulistic episode occurred before the arousal, and in seven cases the order was reversed.

Table 2
Polysomnographic data.

Sleep variables	Mean	SD
Sleep latency (min)	16.3	11.9
SWS latency (min)	28.6	35.7
R latency (min)	107.6	38.3
Sleep efficiency (%)	90.0	4.9
% N1	8.3	3.0
% N2	53.6	6.4
% N3	17.3	6.2
% R	20.9	4.4
Total sleep time (min)	420.3	35.1
Awakenings (total no.)	31.0	11.4
Awakenings (per hour of sleep)	4.1	1.4
Awakenings from N3 (total no.)	4.6	1.9
Mean time of SW episode onset after sleep onset (min)	97.7	49.8
Mean time on onset of awakening after sleep onset (min)	98.1	76.3

Abbreviations: SD, standard deviation; SWS, slow-wave sleep; R, rapid eye movement (REM) sleep; N1, non-REM sleep stage 1; N2, non-REM sleep stage 2; N3, non-REM sleep stage 3.

Spectral analyses for SWA were computed with a commercial software package on C3 using a fast Fourier transform (cosine tapering) on 4 s artifact-free sections. Epochs containing artifacts were considered as missing data to preserve sleep continuity. An in-house software package was used to automatically detect SO on artifact-free derivation C3 according to published criteria: (1) negative peak <-40 μV; (2) peak-to-peak amplitude >75 μV; (3) duration of negative deflection >125 ms and <1500 ms; and (4) duration of positive deflection <1000 ms. SO density was defined as the number of SO per minute [13].

Fluctuations in SWA (0.5–4 Hz), delta (1–4 Hz), slow delta (0.5–1 Hz) and SO density were assessed with analysis of variance with two repeated measures (event type: SW episode vs arousal and sequential time windows prior to episode onset: 3 to 2 min before episodes, 2 to 1 min, 1 min to 32 s, and 32 s to episode onset. Alpha levels were adjusted for non-sphericity (Huynh–Feldt). To assess finer time-related fluctuations, the minute immediately preceding each SW episode and awakening was divided into 15 windows of 4 s each and the resulting time segments analyzed with a repeated measures ANOVA with two measures (event type: SW episode vs arousal, and time window: 15 windows of 4 s each leading up to the event). $P \le 0.05$ was considered significant.

3. Results

PSG variables (mean and SD) for the adult sleepwalkers are presented in Table 2. Mean onset of SW episodes and awakenings (calculated as time after sleep onset; see Table 2) did not differ [$F(1,11) = 0.06, P > 0.05$]. SWA was significantly higher before SW episodes compared with non-behavioral awakenings [event type effect: $F(1,11) = 14.8, P < 0.01$]. There was no significant main effect for time window or significant interaction between time window and event type, indicating that, for both event types, SWA spectral power did not significantly differ between the four time windows retained for the analysis. Figure 1 illustrates the dynamics of SWA across each time window (3 min, 2 min, 1 min and 32 s) for somnambulistic episodes and non-behavioral awakenings. Delta and slow delta were also significantly higher before SW episodes than non-behavioral awakenings with no gradual change over the 3 min preceding events [event type effect: $F(1,11) = 9.99, P < 0.01$; and $F(1,11) = 22.88, P < 0.01$ for delta and slow delta, respectively]. Finally, SW episodes were preceded by a greater SO density than non-behavioral awakenings [event type effect: $F(1, 11) = 10.11, P < 0.01$]. No significant time window effect or interaction between event type and time window was found for SO.

Analyses on the final minute (divided into 15 consecutive 4 s windows) preceding episodes and awakenings revealed that SWA was significantly greater before SW episodes compared with non-behavioral arousals [event type effect: $F(1,11) = 12.64, P < 0.01$], but no significant temporal pattern was found. Delta and slow delta showed a similar course, being greater within the minute prior to somnambulistic episodes as compared with the minute before non-behavioral awakenings [event type effect: $F(1,11) = 9.94, P < 0.01$], without a time window effect. SO density showed a similar pattern with greater values observed within the minute prior to the somnambulistic episodes as compared with normal awakenings [$F(1,11) = 14.22, P < 0.01$].

4. Discussion

The main goal of the present study was to compare EEG fluctuations in SWA and SO density preceding somnambulistic episodes with those observed prior to non-behavioral awakenings. Consistent with our first hypothesis, we found that somnambulistic episodes, compared with non-behavioral awakenings, were preceded by enhanced SWA power density. This elevated SWA is likely attributable to the concomitant increase in SO density, as previously suggested by Jaar et al. [11], who also found that increased SWA prior to somnambulistic episodes was accompanied by an increase in SO density but not in SO amplitude. As SWA and SO are believed to be related to sleep depth [14] our results support the idea that enhanced sleep intensity, reflected through SWA and SO during sleepwalkers' NREM sleep, plays a role in episode occurrence during patients' normal sleep and that this effect is specific to their somnambulistic events.

Several possible mechanisms might explain these results. Slow oscillations between prolonged hyperpolarization and relatively short depolarization phases of thalamocortical and cortical neurons have been hypothesized to protect sleep integrity by inhibiting afferent stimulations during sleep [15]. Observed increases in sleepwalkers' SO density could thus reflect a process whereby the cortex, challenged by an internal or external afferent thalamocortical stimulus, actively tries to block the arousal stimulus through a
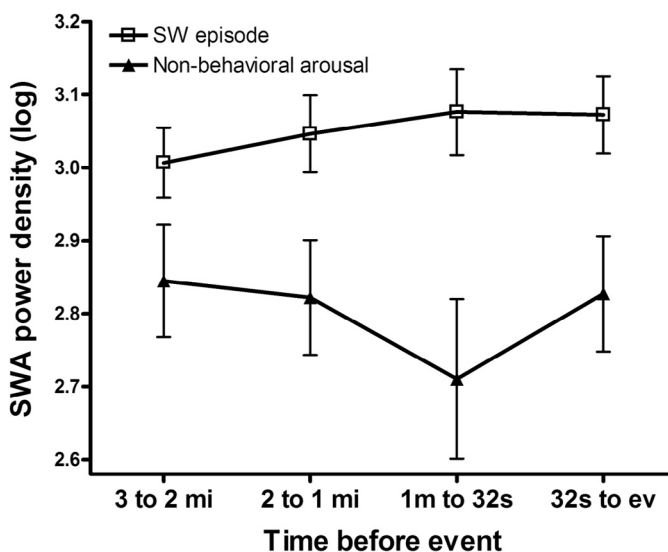

Fig. 1. SWA dynamics over the 3 min preceding SW episodes and arousals. SWA, slow wave activity. The x-axis shows the three time segments chosen for analysis, namely 3 to 2 min prior to the event (somnambulistic episode, illustrated by a solid line with squares, and normal awakenings without behavioral manifestations, illustrated by the solid line with triangles), 2 to 1 min prior to the event, 1 min to 32 s before the event, and 32 s until the onset of the event.

cortico-thalamic feedback response. Sleepwalkers' inability to fully awaken during somnambulistic episodes could therefore result from these antagonistic influences on the thalamus [5]. This effect is supported by the notion that internal or external factors which tend to fragment sleep (eg, sleep apnea, auditory or proprioceptive stimuli in the sleeper's environment) can facilitate the occurrence of somnambulistic episodes in predisposed individuals [16,17] and that sleep deprivation, which normally leads to consolidated sleep in normal controls, significantly increases the frequency of non-behavioral arousals [18] and somnambulistic episodes [19], as well as SWA and SO density [20] in sleepwalkers. However, the present results cannot be viewed as providing strong support for this view, as the somnambulistic episodes observed in our study occurred spontaneously and thus were not related to any clear exogenous stimuli.

One important finding from the present study is that, even within the sample of sleepwalkers, different EEG patterns were associated with non-behavioral arousals versus somnambulistic episodes, warranting further exploration of the electrophysiological processes by which somnambulistic episodes arise. Future studies should aim to determine the role of sleep intensity in the occurrence of somnambulistic episodes, for example by comparing spontaneous episodes arising out of stage N3 versus stage N2 sleep, or by comparing the EEG patterns associated with experimentally induced episodes (eg, via auditory stimulation [16]), full awakenings, and trials during which stimulations do not induce an arousal. It would also be of great interest to examine somnambulistic episodes in relation to sleep spindles, a brain wave known to be involved in protecting sleep integrity against activating stimuli [15].

Contrary to our second hypothesis, no gradual increases in SWA or SO density were found over the four broad time windows selected prior to episode occurrence, nor within the final minute immediately preceding episodes or arousals. Hence, we did not find evidence for a gradual build-up in enhanced sleep pressure over several minutes prior to episode onset, as was suggested by Espa et al. [6], nor did we observe peak in the final 20 s before the episode, as documented in previous studies [5,11]. This unexpected result could be due to the heterogeneity of sleepwalkers as a population. In fact, considerable variations exist in the disorder's phenotypic expression (eg, age of onset, episode frequency and complexity, comorbidity with other parasomnias, etc.) and patient characteristics (eg, age, gender, family history). However, somnambulism is typically assumed to represent a uniform condition across studies, whereas the potential effects of phenotypic variations on PSG-based variables and their expression in sleepwalkers remain to be investigated.

As an example, 10 out of 12 episodes analyzed in the present study were relatively simple manifestations (eg, sitting up in bed, staring about, moving one's arms around), whereas two were of intermediate complexity (eg, trying to get out of bed). None of the episodes involved a patient actually making it over the bed's guard rails and attempting to leave the sleep chamber. Moreover, even relatively simple episodes can vary in terms of behavioral and emotional intensity (eg, agitated versus calm events) as well as duration. Information on episode complexity is rarely reported in the literature [6,8,11], but episodes recorded following a sleep deprivation protocol may be more complex or agitated on average, as sleep deprivation has been shown to increase both the frequency and complexity of somnambulistic episodes in predisposed individuals [16,21]. The extent to which SWA and SO activity prior to episode onset show differential patterns as a function of event complexity or effects of sleep deprivation remains to be determined.

The proportion of women and men in our sample (nine women, three men) may also have influenced our results. Whereas significant sex-related differences in measures of SWA and SO have been documented in the sleep of normal controls [13,22,23] (eg, higher SWA in women than in men), such sex effects have yet to be explored in PSG investigations of patients presenting with a history of NREM parasomnias.

Although the results of the present study help clarify the mechanisms by which somnambulistic episodes differ from normal arousals, more work is needed to delineate the functional significance of SWA and SO in relation to the occurrence of somnambulistic episodes. Differentiating patterns of SWA and SO before episodes associated with various subtypes of somnambulism (eg, in terms of event complexity, in people with and without a family history for the disorder, in people with childhood versus adult onset) could also be helpful in clarifying pathophysiology of the disorder.

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Conflicts of interest

None declared.

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