# Dynamics of Slow-Wave Activity During the NREM Sleep of Sleepwalkers and Control Subjects

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**Study Objective:** To compare the number and distribution of awakenings from slow-wave sleep (SWS) and both the power and dynamics of EEG slow-wave activity (SWA) in sleepwalkers and controls. Somnambulism is considered to be a disorder of arousal from NREM sleep and related to anomalous SWS and SWA. Power spectral analyses have never been used to quantify patients' SWA across sleep cycles.
**Design:** N/A
**Setting:** N/A
**Patients:** A polysomnographic study was performed on 15 adult sleepwalkers and 15 age- and sex-matched controls.
**Interventions:** N/A
**Measurements & Results:** Sleepwalkers had a significantly greater number of awakenings from SWS than did control subjects. Controls showed a greater decrease in SWA across NREM cycles. Sleepwalkers had a significantly lower level of SWA during the first NREM period, where most awakenings take place.
**Conclusion:** Sleepwalkers appear to suffer from an abnormality in the neural mechanisms responsible for the regulation of SWS.
**Key words:** Sleepwalking; parasomnias; EEG; slow-wave sleep; slow-wave activity

## INTRODUCTION

SLEEPWALKING (SOMNAMBULISM) IS ONE OF THE PARASOMNIAS, A GROUP OF CLINICAL DISORDERS CHARACTERIZED BY ABNORMAL MOTOR, VERBAL, OR EXPERIENTIAL EVENTS THAT OCCUR DURING SLEEP. Although sleepwalking may occur from lighter sleep stages, behavioral manifestations almost always arise from a sudden but incomplete arousal from slow-wave sleep (SWS; stages 3 and 4 sleep), and are considered to be a "disorder of arousal" from non-rapid eye movement (NREM) sleep.[1-4] Somnambulistic episodes tend to occur in the first third of the night and are usually characterized by confusion, automatic behaviors, decreased responsiveness to external stimuli, and amnesia of the event on the following morning.[4-7] The actual behavioral manifestations can range from relatively simple movements (e.g., sitting up in bed, picking at the bed covers, quiet walking about) to more complex acts (e.g., getting dressed, climbing ladders, driving motor vehicles) including frantic, agitated attempts to run or escape .[8-14]

Sleepwalking occurs in 1% to 15 % of the general population[15,16] and is more frequent in children and young adolescents (4% to 17%) than in adults (1% to 4%).[15, 17-21] Since full-blown episodes of somnambulism rarely occur in the sleep laboratory,[3,8,22] the diagnosis of sleepwalking is largely based on the individual's history.

Analyses of sleep macrostructure (e.g., sleep architecture, cyclic patterns of sleep stages) show no significant differences between adult somnambulistic patients and control subjects,[11,23-25] except for one study showing a greater number of arousals out of SWS in sleepwalkers compared to controls.[22]

Spectral analysis of the EEG is a reliable method of assessing the dynamics of different sleep EEG frequency bands across the night. EEG slow-wave activity (SWA; spectral power on the 0.75-4.5 Hz band) is a quantitative measure of slow-wave sleep dynamics and a good indicator of the integrity of the homeostatic process underlying sleep regulation.[26-29] EEG slow-wave activity shows a global declining trend during sleep.[30-32] Since somnambulistic patients have repeated awakenings from early NREM sleep cycles, interruptions would be expected in the initial buildup of SWA. However, spectral analyses have never been performed to quantify somnambulistic patients' SWA

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across sleep cycles.

The goals of this study were to compare the power as well as the dynamics of EEG SWA during sleepwalkers' and controls' NREM sleep in relation to the number of awakenings from SWS and to assess the distribution of awakenings across sleep episodes. Since SWA is more prominent during the first NREM cycle than during any other sleep cycle, it was hypothesized that awakenings from early SWS would disrupt the buildup of SWA. A greater number of awakenings from early SWS was expected to occur in somnambulistic patients than in controls. Consequently, it was hypothesized that the degree of SWA disruption during the first NREM period would be significantly greater among sleepwalkers.

## METHODS

Subjects were 15 sleepwalkers (5 males/10 females; mean age = 25.1±4.6 yrs; age range: 19—39 yrs) and 15 age- and sex-matched controls (mean age = 24.7±5.3 yrs; age range: 18—37 yrs). Table 1 presents a description of each sleepwalker. All patients underwent a standard psychiatric and neurological investigation. None of the patients reported a history of psychiatric disorders, severe head trauma, diurnal or nocturnal seizures, current use of psychotropic medications or of other medications known to influence sleep or the EEG. Participants were screened for the presence of sleep apneas and periodic limb movements during sleep (PLMS). Subjects with an index of respiratory events (apneas, hypopneas/hour of sleep) greater than 10 or a PLMS index greater than 10 were excluded from the study. For both groups, only subjects with comparable sleep parameters entered the study.

### Polysomnography

Subjects underwent one night of continuous polysomnographic recording in the sleep laboratory. Electrodes were placed according to the international 10-20 system for the following montage: C3/A2, O2/A1, left and right electrooculogram (EOG) and chin electromyogram (EMG). A Grass polygraph (sensitivity 7.0 V/mm, bandpass 0.3-100 Hz) was used to amplify signals. The signals were also relayed to a PC computer where they were digitized at a sampling rate of 128 Hz and filtered with a digital filter having an upper cutoff frequency of 64 Hz. Twenty-second epochs from the C3/A2 lead were used to visually score sleep stages according to established criteria.[33] Awakenings were scored when sleep stages were interrupted by stage 0 for at least 50% of one epoch[33] and were tabulated separately for each sleep stage.

### EEG Spectral Analyses

In order to obtain data which were comparable with most studies investigating sleep regulatory mechanisms [29,32,34] and since it is the standard derivation for sleep scoring,[33] spectral analyses were performed on the C3/A2 derivation using a commercial software package (Eclipse 3.0, Stellate Systems). Fast Fourier transforms (FFTs) were computed on four-second mini-epochs with a cosine window tapering yielding a spectral resolution of 0.25 Hz. Mini-epochs containing artifacts were rejected by visual

**Table 1—**Patients' characteristics

| Patient | Gender | Age at Consultation | Episode frequency | Self-injurious or aggressive behavior | Association with sleep terrors |
|---|---|---|---|---|---|
| FD | F | 37 | 1-2/month | No | No |
| BS | F | 25 | Few times/week | No | No |
| SD | M | 27 | Few times/month | Purposeless aggression, minor injuries | No |
| PL | M | 22 | Five times/week | No | No |
| SB | F | 22 | 2/month | No | Yes |
| KW | F | 24 | 2 times/week | Minor injuries | Yes |
| ER | M | 18 | Few times/year | Minor injuries | Yes |
| NH | F | 23 | 1-3 times/week | No | No |
| CH | F | 26 | Few times/month | No | No |
| AH | M | 30 | 1-4 times/night, 5 nights/week | Aggressive behavior toward objects, minor injuries | Yes |
| BG | M | 27 | Few times/week | No | No |
| CG | F | 21 | Few nights/week, more than one episode within one night is frequent | No | No |
| GF | F | 21 | Few times/month | No | No |
| IP | F | 23 | 3-4 times/week | No | No |
| CC | F | 20 | 1-2 times/month | No | No |

**Table 2—**Polysomnographic data for sleepwalkers and controls

| | Sleepwalkers | | Controls | | | |
|---|---|---|---|---|---|---|
| Sleep variables | Mean | S.D. | Mean | S.D. | t-value | P |
| Sleep latency (min) | 12.8 | 14.5 | 13.5 | 8.5 | -0.16 | 0.88 |
| REM latency (min.) | 103.2 | 43.6 | 94.7 | 27.8 | 0.63 | 0.53 |
| Sleep efficiency (%) | 93.3 | 5.7 | 95.1 | 3.2 | -1.08 | 0.29 |
| % stage 1 | 8.0 | 3.9 | 9.3 | 2.9 | -1.08 | 0.29 |
| % stage 2 | 59.2 | 6.0 | 54.8 | 4.4 | 2.27 | 0.03 |
| % stages 3+4 | 12.6 | 5.6 | 15.1 | 4.7 | -1.30 | 0.20 |
| % stage REM | 20.3 | 4.6 | 20.8 | 2.9 | -0.40 | 0.69 |
| Total sleep time (min) | 459.2 | 45.8 | 470.3 | 53.4 | -0.61 | 0.55 |

inspection and treated as missing data in order to preserve sleep continuity. Five consecutive four-second epochs were averaged to maintain a correspondence with the 20-second epochs of sleep scoring. Power spectral analysis of SWA (0.75 - 4.50 Hz) was performed for the first four NREM cycles. In order to investigate EEG SWA dynamics and to compare sleep cycles between subjects, each NREM episode was divided into 20 equal intervals, and each REM episode into five equal intervals.

### Statistical Analyses

T-tests for independent samples were used to compare sleep variables and awakenings from each sleep stage between the two groups. A two-way analysis of variance (ANOVA) with one independent factor (Group) and one repeated measure (NREM cycle) was performed to evaluate the time course of SWA across NREM cycles. Alpha levels were adjusted with Huynh-Feldt correction for sphericity, and results were considered significant when $p \leq 0.05$. Contrast analyses were used to decompose the interaction effects and identify the nature of significant results. A two-way ANOVA with one independent factor (Group) and one repeated measure (each half of NREM cycle 1) was performed to investigate SWA dynamics during the first NREM cycle.

## RESULTS

### All-Night Sleep Parameters

In the sleepwalker group, of the five behavioral manifestations that occurred during the laboratory sleep study, only one occurred in stage 2, the other four arose out of SWS. The polysomnographic data for the adult sleepwalkers and controls are summarized in Table 2. Sleepwalkers had a higher percentage of stage 2 sleep (t(28)=2.27; p=0.03). No other significant difference was found on any of the other sleep variables indicating that both groups had comparable sleep architecture.

The number of awakenings from each sleep stage for the somnambulistic and control groups are presented in Table 3. Overall, both groups of subjects experienced more awakenings from stages 1 and 2 sleep than from stages 3 and 4. However, sleepwalkers had a significantly greater number of awakenings from SWS (stage 3 and 4 sleep) than did controls (t(20.96)=2.74; p=0.01). There were no significant between-group differences for the number of awakenings from other sleep.

**Table 3—**Arousals from different sleep stages in sleepwalkers and controls

| | Sleepwalkers | | Controls | | | |
|---|---|---|---|---|---|---|
| | **Mean** | **S.D** | **Mean** | **S.D** | **t-value** | **P** |
| Stage 1 | 8.4 | 4.9 | 8.5 | 4.8 | -0.07 | 0.94 |
| Stage 2 | 10.3 | 5.7 | 9.3 | 6.6 | 0.41 | 0.68 |
| Stages 3 + 4 | 2.4 | 1.9 | 0.9 | 1.0 | 2.75 | 0.01 |
| REM | 3.0 | 2.1 | 4.3 | 3.5 | -1.20 | 0.24 |

### Spectral Analyses

The absolute SWA power for sleepwalkers' and controls' four NREM cycles is presented in Figure 1. Both groups evinced a global decline in SWA across each NREM cycle. A two-way ANOVA showed a significant Group X NREM cycle interaction (F3,84 = 2.90, e = 0.84, p<0.05), indicating that the decrease in SWA across NREM cycles was greater in controls than in sleepwalkers. Contrast analyses revealed a significant between-group difference for the first NREM cycle (p<0.03), indicating a greater level of SWA in the control group than in sleepwalkers at the beginning of the night. There were no statistically significant differences between the two groups on the remaining three NREM cycles.

The dynamics of SWA across the first four NREM cycles are illustrated in Figure 2. Since the most important difference between sleepwalkers' and controls' SWA occurred within the first NREM cycle, the intracycle dynamics of SWA were further investigated. A two-way ANOVA revealed a significant Group by Half of NREM

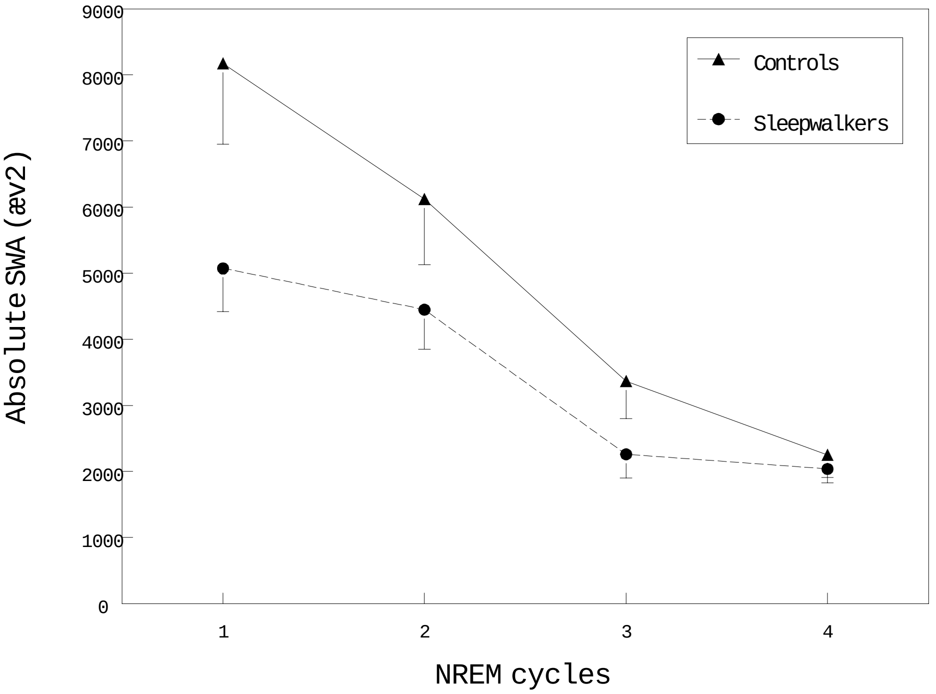

**Figure 1—**Absolute SWA across four NREM cycles for sleepwalkers and controls subjects

cycle 1 interaction effect (F1,18 = 6.75, p<0.02). Contrast analyses highlighted the different dynamics of SWA for both groups. Specifically, SWA power was similar for sleepwalkers and controls during the first half of NREM cycle 1 (p=0.43), but was significantly higher in the control group during the second half of the first NREM cycle (p<0.001).

## DISCUSSION

The adult somnambulistic patients in this study were found to have a significantly higher number of awakenings from SWS than did age - and sex-matched control subjects. No significant differences were found between the two groups for number of awakenings from other sleep stages. These results indicate that an important feature of sleepwalking is the presence of a high number of awakenings in stages 3 and 4 sleep.

Consistent with most previous reports,[11,22-25] we found no major differences between sleepwalkers and controls in their overall sleep architecture. Only one of the eight traditional sleep parameters investigated was significantly different between the two groups; sleepwalkers had a greater percentage of stage 2 sleep. Although people with a history of somnambulism are sometimes described as having excessively deep sleep,[5] our polysomnographic results confirm that they do not have more slow-wave sleep than controls (see Table 2). One study even showed a selective decrease in SWS for a subgroup of sleepwalkers with a serious violence toward others compared to other subgroups of sleepwalkers.[34]

Consistent with our hypothesis, somnambulistic patients were found to have significantly lower SWA power than control subjects during the first NREM cycle. As a result, the rate of the decrease in SWA across NREM cycles was greater in control subjects than in sleepwalkers. These results suggest that sleepwalkers' frequent awakenings from SWS interfere with the normal buildup of their SWA. Consequently, somnambulistic patients have lower EEG SWA than controls, especially during the first NREM cycle. The buildup of SWA at the beginning of the first NREM cycle was similar for both groups, but SWA fell prematurely in sleepwalkers (see Figure 2). In fact, SWA power was significantly lower among sleepwalkers during the second half of the first NREM cycle; the period during which most awakenings from SWS occur. In subsequent NREM cycles, however, awakenings from SWS are less frequent and SWA can resume its buildup. This would account for the absence of significant differences in the two groups' SWA across the three other NREM cycles. The pattern of SWA observed during the first NREM cycle among adult sleepwalkers suggests a lack of integrity in the homeostatic process underlying the regulation of slow-wave sleep. Whether or not significant disruptions in SWA also occur in younger populations of sleepwalkers remains an open question.

Our results suggest that spectral analyses of SWA across NREM cycles can contribute to our understanding of the disorders of arousal. Delineating patterns of SWA associated with various parasomnias could help clarify their underlying pathophysiology. Given the likelihood that results of our study could be used in medico-legal settings, it is worth noting that the presence or absence of a decrease of SWA

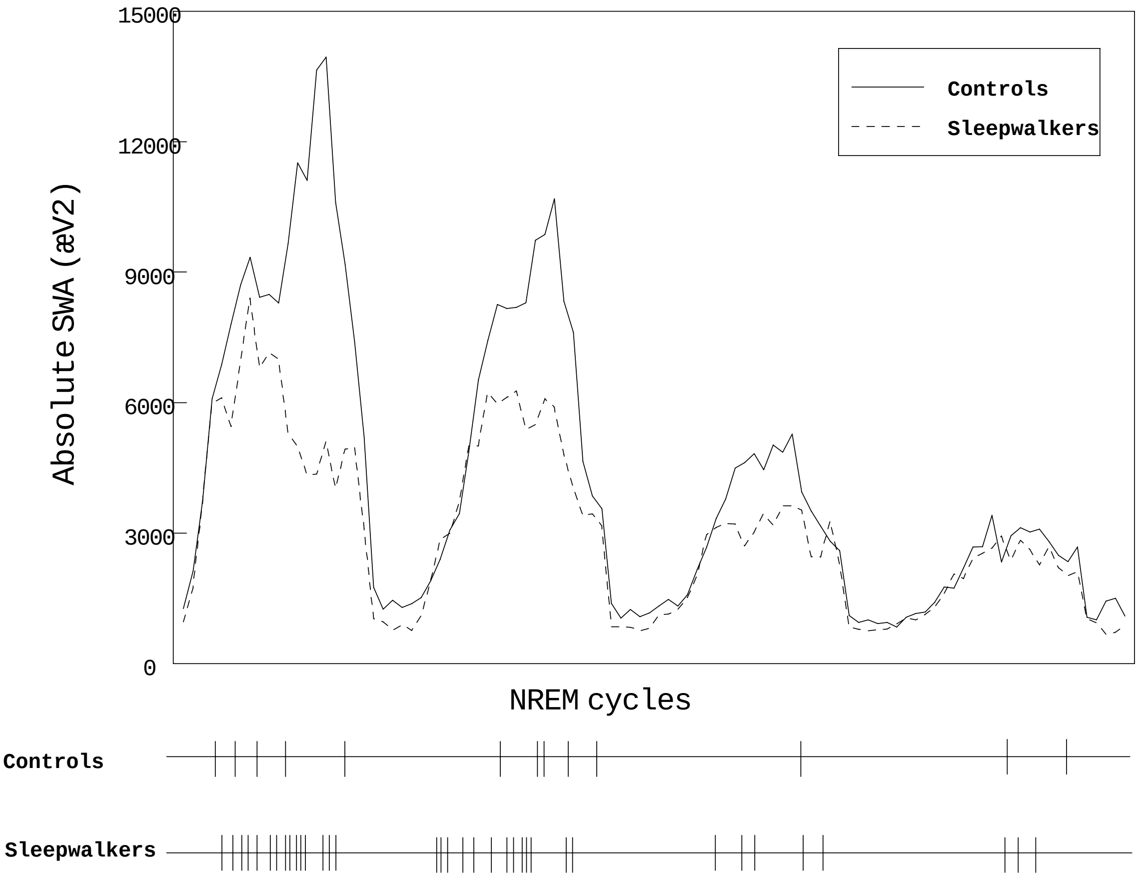

**Figure 2—**Dynamics of SWA over four consecutive sleep cycles in sleepwalkers and control subjects. The distribution of awakenings from stages 3 and 4 are represented by vertical marks on a line corresponding to the time course of the four NREM cycles.

early in the night and of awakenings from SWS in a given individual does not conclusively establish or refute a tendency toward sleepwalking. The diagnostic value of SWA and awakenings from SWS could be further assessed by submitting them to manipulations influencing homeostatic features of sleep regulation. For instance, sleep deprivation is a factor which augments SWA[26,27,35] as well as the frequency and intensity of somnambulistic episodes.[36-38] Assessing the differential effects of such a challenge in sleepwalkers could help determine these variables' diagnostic and theoretical utility. Taken together, the results suggest an abnormality in the neural mechanisms responsible for the regulation of SWS in somnambulistic patients. To what extent this presumed dysfunction in the regulation of SWS is similar to what has been found in other types of sleep disorders[39] and whether or not is related to the genetic predisposition to somnambulism[20,40,41] remains an open question.

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