Objective: Somnambulism affects up to 4% of adults and constitutes one of the leading causes of sleep-related violence and self-injury. Diagnosing somnambulism with objective instruments is often difficult because episodes rarely occur in the laboratory. Because sleep deprivation can precipitate sleepwalking, we aimed to determine the effects of 25 hours of sleep deprivation on the frequency and complexity of somnambulistic episodes recorded in the laboratory.

Methods: Thirty consecutive sleepwalkers were evaluated prospectively by video-polysomnography for one baseline night and during recovery sleep after 25 hours of sleep deprivation. Ten sleepwalkers with a concomitant sleep disturbance were investigated with the same protocol.

Results: Sleepwalkers experienced a significant increase in the mean frequency of somnambulistic episodes during postdeprivation recovery sleep. Postsleep deprivation also resulted in a significantly greater proportion of patients experiencing more complex forms of somnambulism. Sleep deprivation was similarly effective in 9 of the 10 patients presenting with a comorbid sleep disturbance. Combining data from all 40 patients shows that whereas 32 episodes were recorded from 20 sleepwalkers (50%) at baseline, recovery sleep resulted in 92 episodes being recorded from 36 patients (90%).

Interpretation: The findings support the view that sleepwalkers suffer from a dysfunction of the mechanisms responsible for sustaining stable slow-wave sleep and suggest that these patients are particularly vulnerable to increased homeostatic sleep pressure. Strong evidence is provided that 25 hours of sleep deprivation can be a valuable tool that facilitates the polysomnographically based diagnosis of somnambulism in predisposed patients.

Somnambulism (sleepwalking) is a common arousal parasomnia that affects up to 4% of adults. Behavioral manifestations of varying degrees of complexity and duration arise from incomplete awakenings, usually from slow-wave sleep (SWS: stage 3 and 4 sleep) and sometimes for stage 2 sleep. Most episodes are characterized by misperception and relative unresponsiveness to the environment, mental confusion, and variable retrograde amnesia. Sleepwalkers often consult because of a history of aggressive and/or injurious behaviors during sleep. Moreover, there has been a sharp increase in the number of studies relating somnambulism to homicides, attempted homicides, filicides, suspected suicides, and other sleep-related behaviors with medicolegal implications.

Unlike most sleep disorders, sleepwalking is diagnosed primarily or exclusively by the patient’s clinical history. Sleep laboratory investigations are helpful in ruling out other disorders (eg, nocturnal seizures, rapid eye movement [REM] sleep behavior disorder), but no diagnostic sleep protocol exists to confirm the diagnosis. The principal difficulty in diagnosing somnambulism with objective instruments such as polysomnography (PSG) is that behavioral events rarely occur in the sleep laboratory. Moreover, when they do occur, laboratory episodes are less complex than what is described for the patient’s home environment. Several polysomnographic features including frequent arousals from SWS, presence of hypersynchronous delta waves, and diminished slow-wave activity have been proposed as indirect evidence supporting the diagnosis, but these variables lack sensitivity and specificity.

The sleep of somnambulistic patients is characterized by non–rapid eye movement (NREM) sleep instability, including an inability to maintain consolidated periods of SWS. Because sleep deprivation intensifies pressure for SWS, we previously hypothesized that it should heighten sleepwalkers’ inability to sustain SWS and increase behavioral manifestations, and clinical reports have suggested that sleep deprivation can facilitate somnambulism in predisposed individuals. However, the use of sleep deprivation to increase epi-
sodes in the laboratory has yielded mixed results. Two studies found that 24 and 38 hours of sleep deprivation significantly increased the frequency of somnambulistic events in sleepwalkers, whereas one study did not find an increase in the number of episodes after 36 hours of sleep deprivation. These inconsistencies may be due, in part, to the limited number of patients investigated (7–10 patients). In addition, differences exist in the proportion of men and women investigated across studies, but sex effects have yet to be explored.

We sought to evaluate the effects of sleep deprivation on the frequency and complexity of somnambulistic events in a large sample of male and female sleepwalkers. Although 38 hours of sleep deprivation has yielded the most promising results to date, this protocol is overly demanding for many research and clinical laboratories. We therefore opted for 25 hours of sleep deprivation with recovery sleep being initiated in the morning, a procedure known to significantly increase SWS during recovery sleep. Secondary aims were to investigate the efficacy of sleep deprivation in facilitating behavioral episodes as a function of sex, as well as in sleepwalkers presenting with a concomitant sleep disturbance.

Subjects and Methods

Subjects

Subjects were 29 adults and 1 adolescent (12 male and 18 female subjects; mean age, 28.6 years; standard deviation, 7.2; age range, 16–47 years) consecutively referred to the Sleep Disorders Clinic of the Hôpital du Sacré-Cœur by their physician between August 2003 and March 2007 for suspected somnambulism. Patients underwent a semistructured clinical interview and a physical examination to determine specific sleep complaints and other psychiatric or physical symptoms, and were then prospectively evaluated with a sleep deprivation protocol as part of our clinical investigation. Patients included in the study had to report a clinical history (including over the past 6 months) of somnambulism and were then prospectively evaluated with a sleep deprivation protocol as part of our clinical investigation. Patients included in the study had to report a clinical history (including over the past 6 months) of somnambulism or somnambulism and sleep disorders that were not of a traumatic, neurological, or medication-induced origin, and to receive a final diagnosis of sleepwalking according to the International Classification of Sleep Disorders.

Thirteen other patients similarly referred to our sleep disorders clinic over this period were excluded from the study according to the following exclusion criteria: a presence or history of neurological disorders including epilepsy (n = 4), meeting Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria for a major psychiatric disorder (n = 5), having a history of drug addiction or abuse (n = 2), and presenting with an uncertain or atypical clinical history of somnambulism (n = 2). Nine other adult sleepwalkers and one adolescent met all of the inclusion and exclusion criteria but presented with a concomitant sleep disorder. Seven (6 men, 1 woman; mean age, 30.6 years; standard deviation, 10.1; age range, 16–43 years) had a periodic leg movements during sleep (PLMS) index greater than 10, and three (2 men, 1 woman; mean age, 38.0 years; standard deviation, 9.2; age range, 30–48 years) were diagnosed with mild sleep apnea syndrome. These 10 patients were investigated separately from the main sample of 30 patients to assess the protocol’s generalizability. None of 40 patients used medications that could influence the sleep electroencephalogram (EEG), sleep architecture, motor activity during sleep, or daytime vigilance. The study was approved by the hospital’s ethical and scientific committee, and consent was obtained from each patient and from the adolescent’s parents.

Procedures

All patients underwent one night of continuous PSG recording in the sleep laboratory. Depending on patients’ habitual sleep/wake cycle, lights off was between 22:00 and no later than 24:00, and wake time occurred between 6:00 and 8:00. After the whole-night baseline recording, patients were instructed to go about their regular daytime activities as usual but were forbidden from taking naps. They returned to the laboratory in the evening for the sleep deprivation protocol and spent the remainder of the night under constant supervision. Recovery sleep was scheduled the next morning, 1 hour after their previous wake time (after 25 hours of wakefulness). Patients were told they would be allowed to sleep as long as they wished but were prohibited from consuming alcohol, caffeine, or other stimulating substances the day before and during all laboratory procedures.

Materials

PSG recordings were conducted on a 32-channel Grass polygraph (sensitivity at 7 μV, bandpass at 0.3–100 Hz; Grass Instruments, Quincy, MA) and digitized at a sampling rate of 256 Hz. EEG recordings and electrode placement were performed according to the international 10–20 system (Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6, Fz, Cz, Pz) with a linked-ear reference and included electrocorticograms, submental electromyography, surface electromyography of the bilateral anterior tibialis, and an electrocardiogram. Respiration was monitored using an oronasal canula and a thoracoabdominal plethysmograph, whereas oxygen saturation was recorded with a finger pulse oximeter. Twenty-second epochs of PSG were used to score sleep stages according to standard criteria. Arousals were defined as a transition from any sleep stage to stage of wakefulness. Microarousals were scored on the C3/A2 lead according to the American Sleep Disorder Association’s criteria, and the microarousal index (number of microarousals per hour of sleep) was determined. All patients were continuously video-taped during both sleep assessments.

Scoring of Somnambulistic Episodes

Behavioral movements identified on patients’ PSG recordings were investigated by the second author (M.P.) by examining the accompanying time-synchronized video recordings. As in our previous work, particular attention was given to behavioral episodes characterized by clumsy, stereotyped, or repetitive movements; by confusion, agitation, or disorientation during the event; and to episodes accompanied by somniloquy. The video recording of each episode was then independently reviewed by two of the authors (A.Z., M.P.) for...
final classification as a somnambulistic episode, and each episode’s complexity was independently scored on a three-point scale shown to have good interrater reliability. A complexity level of 1 was scored when the episode was characterized by simple behaviors (eg, playing with the bed sheets or the electrodes wires) or a change in bodily position (eg, turning and resting on one’s hands while staring about) with observable confusion. Type 2 events consisted of more complex behaviors such as sitting up in bed, resting on one’s knees, or trying to get out of bed. Any event during which the patient left the bed was scored as a 3. Somniloquy alone was not considered as a partial manifestation of somnambulism. The frequency of each type of episode was tabulated. The interrater reliability test indicated a high degree of concordance for the presence or absence of somnambulistic episodes (0.906), as well as for the complexity index (0.840). Discrepancies were resolved by discussion. Apneas, hypopneas, legs movements, and PLMS were also analyzed in temporal relation to each somnambulistic event.

Statistical Analysis
Wilcoxon rank tests were used for between sleep periods comparisons of sleep variables, including frequency of somnambulistic episodes. χ² tests were used to compare the proportion of patients experiencing at least one episode during the two sleep periods. An analysis of variance with one independent factor (sex: male and female) and one repeated measure (sleep periods: baseline and recovery sleep) was performed to investigate sex differences in the frequency of somnambulistic episodes recorded. All analyses were performed using SPSS v15 (SPSS, Chicago, IL).

Results
The average frequency of somnambulistic episodes reported by the main group of 30 sleepwalkers varied from a few times a year to a few times per week. The patients’ estimated monthly prevalence of episodes was 9.7 ± 7.7. Twenty-two patients (73%) reported sleepwalking onset during childhood, 6 (20%) in adolescence and 2 (7%) in adulthood. Fifteen patients (50%) reported a history of self-injuries caused by sleep-related behaviors. A history of sleep terrors was reported by 18 patients (60%), although only somnambulism was observed in the sleep laboratory.

Standard PSG variables for baseline and recovery sleep investigations are presented in Table 1. Sleep deprivation resulted in a significant increase in time spent in SWS and in the percentage of SWS. Patients’ recovery sleep also showed significant reductions in several PSG variables, including sleep latency, REM latency, total sleep time, the percentage of stage 1 sleep, time spent in stage 1, 2, and REM sleep, as well as in the microarousal index.

The Figure shows the mean number of awakenings per patient from different sleep stages observed during baseline and recovery sleep. When compared with baseline values, recovery sleep showed significant reductions in the number of awakenings from stage 2 sleep and from REM sleep, whereas the number of awakenings from SWS increased significantly (all p values < 0.01).

Table 1. Sleep Variables from Baseline and Recovery Sleep in Sleepwalkers

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline Night</th>
<th>Recovery Sleep</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep latency, min (SD)</td>
<td>13.5 (8.7)</td>
<td>4.3 (5.4)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>REM latency, min (SD)</td>
<td>121.1 (47.0)</td>
<td>79.9 (56.2)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Sleep efficiency, % (SD)</td>
<td>89.7 (9.1)</td>
<td>89.4 (9.8)</td>
<td>0.754</td>
</tr>
<tr>
<td>Total sleep time, min (SD)</td>
<td>436.2 (56.2)</td>
<td>362.8 (85.3)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Total wake time, min (SD)</td>
<td>49.3 (42.3)</td>
<td>42.0 (39.5)</td>
<td>0.136</td>
</tr>
<tr>
<td>Stage 1, min (SD)</td>
<td>35.8 (11.9)</td>
<td>22.7 (10.9)</td>
<td>&lt;0.003a</td>
</tr>
<tr>
<td>Stage 2, min (SD)</td>
<td>269.6 (40.6)</td>
<td>212.6 (64.1)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>SWS, min (SD)</td>
<td>42.0 (27.6)</td>
<td>61.7 (37.5)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>REM, min (SD)</td>
<td>88.9 (30.4)</td>
<td>65.8 (31.0)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Stage 1, % (SD)</td>
<td>8.4 (3.2)</td>
<td>6.6 (3.1)</td>
<td>&lt;0.003a</td>
</tr>
<tr>
<td>Stage 2, % (SD)</td>
<td>61.8 (6.1)</td>
<td>58.2 (10.0)</td>
<td>0.084</td>
</tr>
<tr>
<td>SWS, % (SD)</td>
<td>9.9 (6.7)</td>
<td>17.7 (11.3)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>REM, % (SD)</td>
<td>19.9 (5.2)</td>
<td>17.6 (6.9)</td>
<td>0.072</td>
</tr>
<tr>
<td>Microarousal indexb (SD)</td>
<td>9.6 (3.8)</td>
<td>6.7 (3.3)</td>
<td>&lt;0.001a</td>
</tr>
</tbody>
</table>

aWilcoxon rank test.
bMeasured as number of arousals per hour of sleep.
SD = standard deviation; REM = rapid eye movement; SWS = slow-wave sleep.

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There were no significant sex differences or night
recovery sleep (100% of men vs 83% of women).

During both sleep periods. There were no significant
mean frequency of somnambulistic episodes, and that
increase in the mean number of somnambulistic episodes
recorded, and (3) a significantly greater proportion of
patients experiencing one more complex (type 2 or 3)
episode. The ratio of sleepwalking episodes arising
from SWS to the number of awakenings from SWS
was also significantly greater during recovery sleep.

Sex Comparisons
There was no significant difference in men’s and wom-
en’s estimated monthly prevalence of sleepwalking ep-
isodes (10.3 ± 7.1 vs 9.7 ± 8.3, respectively) or in the
proportion of patients reporting a history of sleep-
related injuries (50% for both sexes). Significant main
effects of night (F [1,28] = 18.05; p < 0.001) and sex
(F [1,28] = 5.85; p = 0.022) were found for mean
episode frequency, demonstrating that sleep depriva-
tion significantly increased both men’s and women’s
mean frequency of somnambulistic episodes, and that
male patients experienced a greater number of episodes
during both sleep periods. There were no significant
night × sex interactions. A significantly greater propor-
tion of men (75%) than women (33%) experienced at
least one behavioral episode during baseline sleep (p =
0.025) but no significant difference was observed dur-
ing recovery sleep (100% of men vs 83% of women).

Sex Comparisons

Sleep-Disordered Patients
There were no significant sex differences or night ×
sex interactions for any of the sleep variables presented
in Table 1 or the Figure.

Sleep-Disordered Patients
Table 3 summarizes the characteristics of the episodes
recorded from the 10 sleepwalkers presenting with a
comorbid sleep disturbance. Of the 7 sleepwalkers with
PLMS, 5 experienced a total of 8 somnambulistic epi-
isodes at baseline and 7 had a total of 21 episodes dur-
ing recovery sleep. Sleep deprivation significantly in-
creased the mean frequency of the episodes (1.0 ± 0.8
vs 3.0 ± 1.4; p < 0.05), whereas significantly decreas-
ing patient’s mean PLMS index (19.4 ± 4.3 vs 7.2 ±
5.0; p < 0.05). Of the 29 episodes recorded from these
patients, only 3 events at baseline were preceded within
90 seconds by an individual leg movement part of a
PLMS, and no leg movements were observed 10 sec-
onds before onset of any of the episodes. No episodes
were experienced by the three sleep apnea patients at
baseline, whereas two episodes from two patients were
recorded during recovery sleep. None of these events
was temporally related to an apnea or a hypopnea.

Discussion
The results provide important clinical data on the use
of sleep deprivation as a diagnostic tool for somnam-
bulism. When compared with the 30 sleepwalkers’
baseline assessment, recovery sleep after 25 hours of
sleep deprivation resulted in a significant increase in
the frequency of somnambulistic episodes recorded, as
well as in the proportion of sleepwalkers experiencing
at least one episode under laboratory conditions. Sleep
depprivation was similarly effective in somnambulistic
patients presenting with PLMS and with mild sleep ap-
ea syndrome. Combining data from all 40 patients
investigated shows that whereas 32 behavioral episodes
were recorded from 20 sleepwalkers (50%) at baseline,
recovery sleep resulted in 92 episodes being recorded
from 36 patients (90%). Approximately 80% of the
somnambulistic behaviors recorded during baseline
sleep occurred out of SWS. The remaining 20% of ep-
isodes occurred out of stage 2 sleep, a finding consist-
tent with previous studies.3,17,26 Sleep deprivation re-
sulted in a small, but not statistically significant,
increase of the number of episodes occurring out of
SWS.

Sleep deprivation also significantly increased the pro-
portion of sleepwalkers experiencing at least one com-
plex episode. The data from the 40 sleepwalkers indi-
cate that although only 5 episodes from 5 patients
(12.5%) recorded at baseline were sufficiently complex
to be scored as type 2 or 3, there was a total of 22 such
episodes recorded from 14 patients (35%) during
recovery sleep. In most clinical investigations, the ob-
servation of a relatively simple behavioral episode in
the laboratory may be inconclusive. By yielding a
greater number of episodes with a wider range of
complexity, sleep deprivation can facilitate the video-
polysomnographically based diagnosis of somnambu-
lism and its differentiation from other disorders.

Our findings concord with those of Joncas and col-
leagues,17 who found that 38 hours of sleep depriva-

tion significantly increased the frequency of somnambulistic episodes observed in 9 of their 10 patients. However, our data indicate that a considerably shorter period of sleep deprivation resulting in daytime recovery sleep is equally effective in facilitating a range of somnambulistic behaviors. When compared with baseline recordings, both the 38- and 25-hour sleep deprivation protocols resulted in marked decreases in the number of awakenings from stage 1 sleep, from stage 2 sleep, and from REM sleep. However, only the 25-hour sleep deprivation protocol significantly increased the number of awakenings from SWS. This suggests that sleepwalkers are particularly vulnerable to increased homeostatic sleep pressure after sleep deprivation when recovery sleep is initiated at a circadian time of increasing wake propensity.21 Consistent with this view, one recent study 27 showed that when compared with baseline recordings, sleep deprivation with recovery sleep being initiated in the morning significantly increased the number of SWS awakenings in sleepwalkers but not in control subjects.

Our results demonstrated that the ratio of sleepwalking episodes from SWS to the number of SWS arousals also significantly increased during recovery sleep as compared with baseline (82 vs 42%, respectively). Hence, the increased number of episodes recorded after sleep deprivation was not accompanied by a proportional increase in the number of arousals. Sleep deprivation thus has a differential impact on sleepwalkers' SWS in that its priming effect is greater for somnambulism than for typical EEG arousals. In addition, sleepwalkers' increased difficulty in passing from SWS to another sleep stage or to full cortical arousal after sleep deprivation may partially explain why they are

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Baseline Sleep</th>
<th>Recovery Sleep</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of episodes</td>
<td>24</td>
<td>69</td>
<td>—</td>
</tr>
<tr>
<td>Number of patients with at least one episode</td>
<td>15/30 (50%)</td>
<td>27/30 (90%)</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Number of patients with at least one episode of type 2 or 3</td>
<td>4/30 (13%)</td>
<td>11/30 (37%)</td>
<td>&lt;0.05a</td>
</tr>
<tr>
<td>Mean (SD) frequency of episodes</td>
<td>0.80 (0.96)</td>
<td>2.30 (2.12)</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Number of episodes arising from stage 2 sleep</td>
<td>5/24 (21%)</td>
<td>8/69 (12%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Number of episodes arising from SWS</td>
<td>19/24 (79%)</td>
<td>61/69 (88%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Ratio of number of episodes from SWS to number of awakenings from SWS</td>
<td>0.42 (0.55)</td>
<td>0.82 (0.95)</td>
<td>&lt;0.012b</td>
</tr>
</tbody>
</table>

*aChi-square. bWilcoxon rank test.
SD = standard deviation; SWS = slow-wave sleep.

Table 3. Characteristics of Somnambulistic Events Recorded during Baseline and Recovery Sleep in 10 Sleepwalkers Presenting with Periodic Leg Movements during Sleep or Mild Sleep-Disordered Breathing

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Baseline Sleep</th>
<th>Recovery Sleep</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of episodes</td>
<td>8</td>
<td>23</td>
<td>—</td>
</tr>
<tr>
<td>Number of patients with at least one episode</td>
<td>5/10 (50%)</td>
<td>9/10 (90%)</td>
<td>0.051</td>
</tr>
<tr>
<td>Mean (SD) frequency of episodes</td>
<td>0.80 (1.03)</td>
<td>2.3 (1.64)</td>
<td>&lt;0.017a</td>
</tr>
<tr>
<td>Number of episode arising from stage 2 sleep</td>
<td>2/8 (25%)</td>
<td>4/23 (17%)</td>
<td>0.64</td>
</tr>
<tr>
<td>Number of episode arising from SWS</td>
<td>6/8 (75%)</td>
<td>19/23 (83%)</td>
<td>0.64</td>
</tr>
</tbody>
</table>

*Wilcoxon rank test.
SD = standard deviation; SWS = slow-wave sleep.
more likely to experience more complex episodes during recovery sleep. This hypothesis is consistent with observations indicating that other factors that deepen sleep such as young age, fever, hyperthyroidism, and neuroleptics are susceptible of facilitating or triggering sleepwalking in predisposed individuals.

To our knowledge, sex differences have not been examined in laboratory investigations of sleepwalking, including for the effects of sleep deprivation as a priming agent. Epidemiological studies of parasomnias have found comparable prevalence rates of sleepwalking in men and women. The monthly frequency of sleepwalking reported by our male and female patients was equivalent. By contrast, PSG recordings showed that during normal sleep laboratory investigations, men experience a greater number of episodes than do women, and that sleep deprivation significantly increases this baseline frequency in both sexes. Why male individuals are more likely than female individuals to experience somnambulistic behaviors in the sleep laboratory remains unclear, but sex effects should be considered in future investigations.

One shortcoming of this study is that control participants were not directly investigated. However, none of the control subjects recorded in previous studies of the effects of sleep deprivation on sleepwalking, including in our laboratory, and none of the healthy participants included in similar 25-hour sleep deprivation protocols conducted in our research center experienced behavioral manifestations suggestive of somnambulism. This indicates that sleep deprivation alone does not lead to sleepwalking, but rather that it increases the probability of recording somnambulistic behaviors in predisposed individuals.

Sleepwalking has been described in association with other sleep disorders, particularly sleep apnea syndrome and PLMS. Our patient sample indicates that a majority of adult sleepwalkers referred to a sleep disorders clinic by their treating physician for suspected somnambulism do not suffer from comorbid sleep disorders. Moreover, only 2 of the 124 episodes recorded from our patients were preceded within 30 seconds by an apnea, a hypopnea, or a PLMS, and none within 10 seconds.

Because the results of our study could be used in medicolegal settings, it is important to note that the observation of behavioral events in the sleep laboratory after sleep deprivation is not in and of itself sufficient to confirm a diagnosis of somnambulism in a medicolegal context. To minimize the possibility of malingering, we strongly suggest that EEG patterns recorded during actual behavioral episodes be investigated with an appropriate EEG montage. Schenck and colleagues found that three postarousal EEG patterns characterized the first 10 seconds of most SWS arousals in adults with sleepwalking/sleep terrors. A subsequent study showed that approximately 80% of the EEG tracings recorded during somnambulistic events are readable and can be classified according to the three postarousal patterns that Schenck and colleagues identified. Furthermore, EEG patterns can show differential relations to behavioral complexity, and episodes arising out of stage 2 can be classified according to the same patterns that characterize episodes from SWS.

In summary, the data support the hypothesis that via its homeostatic pressure for increased SWS, sleep deprivation strongly facilitates the occurrence of somnambulistic episodes in predisposed patients. These results are consistent with the view that sleepwalkers’ pathophysiology involves a dysfunction of the mechanisms responsible for sustaining stable SWS. Used as a diagnostic tool, sleep deprivation shows a high sensitivity for somnambulism and may be clinically useful with a wider range of somnambulistic patients than previously reported. The data also provide empirical support for the clinical recommendation that sleepwalkers maintain a regular sleep cycle and avoid sleep deprivation.

This research was supported by the Canadian Institutes of Health Research (MOP77613, A.Z., J.M.).

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