CME

The value of sleep deprivation as a diagnostic tool in adult sleepwalkers

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Abstract—*Objective:* Adult somnambulism can result in injury to the sleeper and to others. Attempts to induce sleepwalking episodes in the sleep laboratory have yielded mixed results.[1,2] Having shown that sleepwalkers have lower slow-wave activity power than control subjects,[3] the authors hypothesized that an enhanced pressure of the homeostatic process underlying sleep regulation could affect the disorder's characteristics even further. Therefore, the effects of 38 hours of sleep deprivation in adult sleepwalkers and control subjects were investigated. *Methods:* Ten adult somnambulistic patients and 10 sex- and age-matched control subjects were studied in the sleep laboratory. After a screening night, participants were monitored during 1) one night of baseline recording, and 2) one recovery night in which subjects slept ad libitum immediately after the sleep deprivation protocol. Behavioral manifestations were assessed for frequency and complexity using a 3-point scale of increasing complexity. *Results:* None of the control subjects had any behavioral manifestations on either of the two nights. Conversely, sleepwalkers showed a significant increase in the frequency and complexity of the somnambulistic episodes during the recovery night compared with baseline. Somnambulistic patients had a greater number of awakenings from slow-wave sleep than control subjects on both nights, but there was no significant increase during the recovery night. *Conclusion:* Sleep deprivation can be an effective tool for inducing somnambulistic episodes in the laboratory, thereby facilitating the diagnosis of sleepwalking.

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Sleepwalking is considered a disorder of arousal,[4] characterized by a disorientation in time and space, a slowing in speech and mentation, poor response to stimulation, and memory impairment for the event.[5,6] Although usually considered a benign condition in children, sleepwalking in adults often is characterized by potentially injurious behavioral manifestations. Several reports relate sleepwalking episodes to homicides.[7-11] Thus, a clear diagnosis is vital with respect to the potential dangers associated with adult sleepwalking as well as for choosing the most appropriate treatment. Sleepwalking rarely occurs in the sleep laboratory and that the incidence of somnambulistic episodes is less in the laboratory than in the patient's normal environment[4,6,12,13] constitutes a potential obstacle in establishing a polysomnographically-based diagnosis.

Data exist to support the idea that sleep deprivation could be a good diagnostic tool for adult somnambulism. Sleepwalkers awake from slow-wave sleep (SWS) more often than control subjects.[14] Recent studies show that as a result of these awakenings, sleepwalkers have a reduction of slow-wave activity compared with controls.[3,15] The pathophysiology of sleepwalking can be conceptualized as an inability to maintain consolidated SWS. Forty hours of sleep deprivation induces a significant pressure for SWS.[16] Therefore, one may hypothesize that sleep deprivation would heighten sleepwalker's inability to sustain SWS, leading to greater sleep fragmentation and to an increase in the frequency of behavioral manifestations in the laboratory. One group having assessed the effects of sleep deprivation in sleepwalkers found that it increased the frequency of complex behavioral episodes.[1] However, they did not observe the sleep characteristics usually associated with a recovery from sleep deprivation. Using a similar protocol, other researchers did not find an increase in the number of episodes among adult sleepwalkers after 36 hours of sleep deprivation.[17] The effects of sleep deprivation on somnambulism remain unclear.

This study was designed to assess the effects of prolonged sleep deprivation on sleep and nocturnal behavioral manifestations in the laboratory in sleepwalkers and control subjects. Based on our pilot investigation,[18] it was hypothesized that sleep deprivation would increase the frequency and complexity of the behavioral manifestations and the number of awakenings from SWS.

Materials and methods. *Subjects.* Ten adult sleepwalkers (3 men, 7 women, mean age: 25.1, SD: 4.1) and 10 sex- and age-matched control subjects were studied (mean age: 25.2, SD: 3.6). All sleepwalkers were patients referred to the Sleep Disorders Clinic prior to their participation in the study. For inclusion in the study, sleepwalkers had to report a minimum of 2 episodes on average per month over

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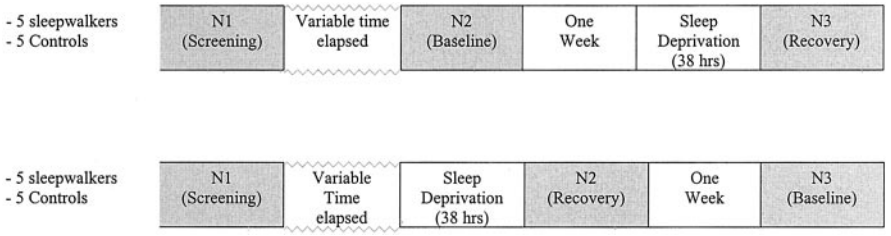

Figure 1. The study design illustrates how participants were recorded for three nights and had to refrain from daytime naps for 1 week before each polysomnographic recording.

the past 6 months that were not of a traumatic, neurologic, or pharmacological origin. All subjects reported being in good health. Exclusion criteria consisted of the following: 1) the presence of another sleep disorder (e.g., narcolepsy, sleep apnea syndrome, periodic leg movements during sleep [PLMS], REM sleep behavior disorder), an index criterion greater than 10 was used for apnea, hypopnea, and PLMS; 2) the presence of a major psychiatric disorder (e.g., schizophrenia, depression); 3) the presence or history of a neurologic disorder (e.g., epilepsy, concussion); and 4) use of drugs that could influence sleep architecture or the EEG (e.g., antidepressants, lithium, benzodiazepines). The protocol was accepted by the Ethics Committee of the Hôpital du Sacré-Coeur de Montréal. All participants signed a consent form and were remunerated for their participation.

Material. Polygraphic recordings were conducted on a 32-channel Grass polygraph (sensitivity at 7 μV, bandpass at 0.3–100 Hz; Grass Instruments, Quincy, MA). Signals were relayed to a PC, digitized at a sampling rate of 128 Hz, and digitally filtered with an upper cutoff frequency of 64 Hz (2 patients and 4 controls were recorded with a sampling rate of 256 Hz). EEG recordings and electrode placement were performed according the 10–20 system (F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6) with a linked-ear reference. Electromyograms (EMG), electro-oculograms (EOG), and EKG also were recorded. During the screening night, EMG of the anterior tibialis was recorded for screening of PLMS. Respiration was monitored using an oronasal thermistor and a thoracic strain gauge. Oxygen saturation was recorded with a finger pulse oximeter (Biox III, Ohmeda, Boulder, CO). Subjects were continually monitored by an assistant during the period of sleep deprivation. In addition, all subjects were fitted with a portable device (Handy Sleep; Glonner, Planegg, Germany) with C3, O2, EMG, and EOG to verify that they did not sleep during their period of sleep deprivation. Twenty-second epochs from the C3/A2 lead were used to visually score sleep stages according to established criteria.[19]

Procedure. Figure 1 illustrates the study design. Participants were recorded for three nights and had to refrain from taking daytime naps for 1 week before each polysomnographic recording. All participants came for an initial screening night (Night 1) to ensure that they were free of any major sleep disorder. Then they were scheduled for a second night of polysomnographic recording, which took place between 1 and 6 weeks after the initial screening night. The second night served as a baseline night and subjects were recorded with a complete EEG montage (19 electrodes). Lights off was at 23:00 and wake time was at 7:00. One week later, subject returned to the laboratory for the sleep deprivation protocol. The 38 hours of sleep deprivation proceeded as follows: after their normal morning awakening, subjects were instructed to go about their day as usual but were forbidden from taking any naps. Then they came to the laboratory in the evening where the ambulatory device was installed and spent the remainder of the night and the following day under constant supervision. Participants were prohibited from taking any stimulating substances (caffeine, nicotine) during this period. Subjects were recorded with the full montage during their recovery night (Night 3) and were told that they could sleep as long as they wished. To control for laboratory adaptation effects, half of the subjects had the sleep deprivation on their third and last visit, whereas the other half had it on the second visit with the baseline recording occurring on the third night, one week after the recovery night.

Scoring of somnambulistic episodes. To our knowledge, no scale has been developed to score nocturnal behavioral manifestations. Thus, a scale was constructed to quantify the complexity of sleepwalking episodes. We began with a standard definition of a somnambulistic manifestation summarized in the American Sleep Disorder Association manual[5] and detailed in various papers including Nino-Murcia and Dement.[6] We looked specifically for episodes that were characterized by clumsy or repetitive movements, confusion or disorientation during the event, and sometimes accompanied by somniloquy. A 3-point scale then was used to classify the complexity of each behavioral manifestation. A complexity level of 1 was scored when the episode was characterized by a change in bodily position (e.g., turning and resting on one's hands) or any simple behaviors (e.g., playing with the bed sheets). Type 2 episodes were scored if the patient executed a complex behavior such as sitting up in bed, resting on his or her knees, or trying to get out of bed. Any event during which the subject left the bed was scored as 3. The frequency of each type of episode also was tabulated. Arousals were defined as a transition from any sleep stage to stage W (wakefulness). The video recordings of every subject were scored blind by one of the authors (S.J.) for the presence of somnambulistic events. All sleepwalking episodes then were reviewed and scored independently by two of the authors (A.Z., S.J.) for complexity. The Kappa inter-rater reliability test indicated a high degree of concordance between the two raters ($K = 0.778$). Discrepancies were resolved by discussion.

Statistical analyses. Between-group comparisons of the sleep variables for both nights were performed with a 2×2 analysis of variance with one independent factor (group) and one repeated measure (night). Contrast analyses were used to decompose significant interaction effects. Analyses of variance (group × night) also were performed to compare the number of awakenings from different sleep stages. The Wilcoxon test was used to compare the frequency and the complexity index of the behavioral events. The complexity index was calculated for each subject by

Table 1 *Sleep variables from baseline and recovery nights for sleepwalkers and control subjects*

	Baseline		Recovery		*p* Value		
Sleep variables	SW	Control	SW	Control	Group	Night	Interaction
Sleep latency, min	9.3	21.4	2.6	1.8	NS	0.01	NS
	(10.45)	(29.9)	(2.2)	(2.0)			
REM latency, min	94.4	97.2	107.3	66.7	NS	NS	0.004
	(26.9)	(33.5)	(37.9)	(25.4)			
Sleep efficiency, %	94.3	94.9	96.6	98.2	NS	0.049	NS
	(3.5)	(7.4)	(3.0)	(1.3)			
Stage 1, min	31.3	31.3	13.2	11.3	NS	<0.001	NS
	(14.9)	(9.45)	(9.2)	(5.1)			
Stage 2, min	247.8	244.4	203.9	213.2	NS	<0.001	NS
	(36.2)	(41.8)	(31.1)	(43.6)			
Stage 3 + 4, min	40.6	47.1	71.7	68.2	NS	<0.001	NS
	(14.8)	(22.4)	(15.3)	(20.5)			
REM, min	99.8	92.9	79.9	90.8	NS	0.049	NS
	(28.6)	(31.7)	(16.9)	(28.5)			
Stage 1, %	6.9	6.9	2.9	2.5	NS	<0.001	NS
	(3.4)	(2.6)	(2.1)	(1.1)			
Stage 2, %	53.9	53.1	46.2	47.1	NS	0.004	NS
	(7.8)	(9.1)	(5.6)	(7.8)			
Stage 3 + 4, %	8.8	10.0	16.4	15.2	NS	<0.001	NS
	(3.0)	(4.2)	(4.1)	(4.7)			
REM, %	21.6	20.1	18.0	19.9	NS	NS	NS
	(5.7)	(6.2)	(3.2)	(5.1)			

Numbers in parentheses denote SD.

SW = sleepwalkers; NS = not significant.

summing the number of events multiplied by their complexity level at baseline and after sleep deprivation.

Results. The frequency of somnambulistic episodes reported by the patients varied from a few times per month to a few times per week. Two patients reported frequently having more than one episode within the same night. Three patients reported a history of aggressive nocturnal behaviors that resulted in self-injuries. Sleepwalking began during childhood in all but one of the patients. Both sleepwalking and sleep terrors were reported by one subject, although only somnambulistic events were noted in the sleep laboratory. Two of the sleepwalkers reported having the occasional nightmare.

The sleep variables from both nights for the two groups are shown in table 1. To facilitate comparisons between pre- and postdeprivation nights, a sample of EEG data were selected from each subject's recovery night, beginning at lights out, that was equal in length to the total sleep time during the baseline recording. Mean total sleep time (in minutes) at baseline was 460.2 (SD: 23.1) for the sleepwalkers and 462.8 (SD: 49.9) for the control subjects. A Student's *t*-test for independent samples revealed no significant difference in the duration of the sleep deprivation between sleepwalkers (mean: 2306, SD: 104.8) and control subjects (mean: 2286.1, SD: 77.2). There was a group × night interaction for REM latency ($F_{1, 18} = 10.84$, $p = 0.004$). Contrast analyses revealed no differences on the baseline night but a difference for the recovery night ($F_{1, 18} = 7.89$, $p = 0.012$) indicating that sleepwalkers had a longer REM latency than control subjects. A night effect was found for several variables including a reduction in sleep latency ($F_{1, 18} = 7.59$, $p = 0.013$), in time spent in stage 1 ($F_{1, 18} = 52.24$, $p < 0.001$), in stage 2 ($F_{1, 18} = 17.66$, $p < 0.001$), and in REM sleep ($F_{1, 18} = 4.43$, $p = 0.049$), as well as in the percentage of stage 1 ($F_{1, 18} = 38.93$, $p < 0.001$) and stage 2 sleep ($F_{1, 18} = 10.56$, $p = 0.004$). There also was an increase in sleep efficiency ($F_{1, 18} = 4.43$, $p = 0.049$), time spent in SWS ($F_{1, 18} = 27$, $p < 0.001$), and the percentage of SWS ($F_{1, 18} = 27.99$, $p < 0.001$). No significant group differences were found on any of the other sleep variables across the 2 nights.

Figure 2 illustrates the number of awakenings from different sleep stages during baseline and recovery nights for both groups. Analyses of variance revealed a night effect, reflecting a decrease in the number of awakenings from stage 1 ($F_{1, 18} = 8.85$, $p = 0.002$) and stage 2 ($F_{1, 18} = 19.33$, $p = 0.0001$) during the recovery night compared with baseline, but no group differences were observed. There was a group × night interaction for the number of awakenings from REM sleep ($F_{1, 18} = 7.99$, $p = 0.01$). Contrast analyses revealed a decrease in sleepwalkers'

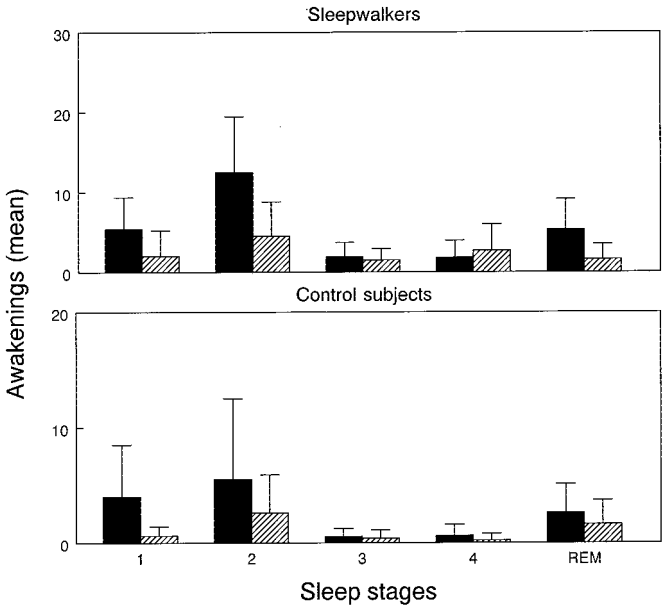

Figure 2. Awakenings per sleep stages for sleepwalkers and control subjects. Contrast analyses revealed a decrease in sleepwalkers' number of awakenings from REM on the recovery night (▨) compared with baseline (■), whereas control subjects did not differ across the two nights.

number of awakenings from REM on the recovery night compared with baseline ($F_{1, 18} = 30.01$, $p < 0.001$), whereas control subjects did not differ across the two nights. A group effect was found for the number of awakenings from stage 3 ($F_{1, 18} = 12.42$, $p = 0.002$) and stage 4 ($F_{1, 18} = 8.92$, $p = 0.008$). These results reflect the expected higher number of transitions from SWS to wakefulness in sleepwalkers when compared with control subjects. However, there was no significant night effect indicating that sleep deprivation did not increase sleepwalkers' number of awakenings from SWS.

Contrary to the sleepwalkers, none of the controls experienced a nocturnal manifestation that was suggestive of a somnambulistic episode. Table 2 presents the characteristics of the behavioral episodes observed in our patient group. There was an increase in the mean number of episodes on the recovery night compared to baseline levels ($T = 0$, $p = 0.011$). In fact, sleep deprivation resulted in a five-fold increase in the total number of somnambulistic episodes recorded in the laboratory. There was also an increase in the complexity of the episodes recorded during the recovery night as compared to baseline ($T = 0$, $p = 0.007$). On both nights, approximately 80% of the sleepwalking episodes occurred out of SWS with the rest occurring out of stage 2 sleep. During baseline recordings, none of the patients experienced an episode more complex than type 1. Conversely, four patients had episodes that were either of type 2 or 3 during the recovery night resulting in a total of eight complex manifestations recorded after sleep deprivation.

Table 2 *Characteristics of somnambulistic events recorded during baseline and recovery nights*

Characteristics	Baseline night	Recovery night	*p* Value
Total episodes	7	37	—
Mean (SD) frequency	0.7 (0.95)	3.7 (4)	0.011
Complexity index (mean)	0.7	4.6	0.007
Percentage arising from stage 2	18.8	19.6	—
Percentage arising from stage 3	25	23.9	—
Percentage arising from stage 4	56.2	56.5	—

Discussion. The main goal of the current study was to assess the value of sleep deprivation as a diagnostic tool for adult somnambulism. The 38 hours of sleep deprivation resulted in a significant increase in the frequency of somnambulistic episodes during recovery sleep. The data thus indicate that sleep deprivation increases the probability of recording actual clinical events in the sleep laboratory and obtaining a polysomnographically-based diagnosis. Sleep deprivation also increased the complexity of the behavioral manifestations evinced by the sleepwalkers. Although all of the episodes recorded at baseline were relatively simple, eight behavioral manifestations observed during the recovery night were complex enough to be classified as either type 2 or type 3. The complexity of the somnambulistic events observed in the laboratory after sleep deprivation more closely resembles the patients' description of the episodes experienced in their home environment.

As expected, most (approximately 80%) of the behavioral manifestations recorded at baseline and on the recovery night occurred out of SWS. That approximately 20% of the episodes occurred out of stage 2 sleep is consistent with previous studies.[20-22]

A salient characteristic of somnambulistic patients' sleep EEG is their unusually high number of awakenings from SWS. Because this feature is considered an intrinsic component of sleepwalkers' pathophysiology, an increase in the frequency of awakenings from SWS was expected during the recovery night, resulting in poorly consolidated sleep. Although sleepwalkers had a greater number of transitions from SWS to wakefulness than controls, there was no significant increase in the number of transitions after sleep deprivation. As seen in figure 2, although control subjects have less fragmented sleep across all sleep stages after sleep deprivation, sleepwalkers show a small (but nonsignificant) increase in the number of awakenings from stage 4 sleep during their recovery sleep. This differs from the typical increase in sleep consolidation found in normal subjects after an extended period of sleep deprivation.[23] More importantly, this observation contrasts with the fivefold increase in the number of behavioral events observed in the laboratory during sleepwalkers' postdeprivation recovery night. Although overt behavioral manifestations and awakenings from SWS may both constitute common features of the arousal disorders, the fact that they respond

differentially to an enhanced homeostatic pressure suggests different underlying pathophysiologic mechanisms. The longer REM latency in sleepwalkers on the recovery night suggests that the pressure for deep sleep is maintained for a longer period of time during the first non-REM period as a result of the multiple sleep disruptions.

Our results support the hypothesis that sleep deprivation can be used as diagnostic tool for adult somnambulism. Sleep deprivation effectively increases the frequency and complexity of somnambulistic events during recovery nights, thereby facilitating a polysomnographically-based diagnosis. Although the occurrence of one relatively simple episode may not be sufficient to establish the diagnosis of somnambulism, the observation and recording of a greater number of episodes with a wider range of complexity would be invaluable in establishing the diagnosis. That none of our control subjects experienced any nocturnal behavioral manifestations in the laboratory demonstrates that sleep deprivation alone does not lead to somnambulistic episodes, but rather that it increases the probability of somnambulistic behaviors among those so predisposed.

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