Precipitating factors of somnambulism

Impact of sleep deprivation and forced arousals

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ABSTRACT

Objective: Experimental attempts to induce sleepwalking with forced arousals during slow-wave sleep (SWS) have yielded mixed results in children and have not been investigated in adult patients. We hypothesized that the combination of sleep deprivation and external stimulation would increase the probability of inducing somnambulistic episodes in sleepwalkers recorded in the sleep laboratory. The main goal of this study was to assess the effects of forced arousals from auditory stimuli (AS) in adult sleepwalkers and control subjects during normal sleep and following post-sleep deprivation recovery sleep.

Methods: Ten sleepwalkers and 10 controls were investigated. After a baseline night, participants were presented with AS at predetermined sleep stages either during normal sleep or recovery sleep following 25 hours of sleep deprivation. One week later, the conditions with AS were reversed.

Results: No somnambulistic episodes were induced in controls. When compared to the effects of AS during sleepwalkers' normal sleep, the presentation of AS during sleepwalkers' recovery sleep significantly increased their efficacy in experimentally inducing somnambulistic events and a significantly greater proportion of sleepwalkers (100%) experienced at least one induced episode during recovery SWS as compared to normal SWS (30%). There was no significant difference between the mean intensity of AS that induced episodes during sleepwalkers' SWS and the mean intensity of AS that awakened sleepwalkers and controls from SWS.

Conclusions: Sleep deprivation and forced arousals during slow-wave sleep can induce somnambulistic episodes in predisposed adults. The results highlight the potential value of this protocol in establishing a video-polysomnographically based diagnosis for sleepwalking.

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GLOSSARY

AS = auditory stimuli; **PLMS** = periodic leg movements during sleep; **PSG** = polysomnography; **SWS** = slow-wave sleep.

Somnambulism (sleepwalking) is considered a "disorder of arousal"[1] characterized by motor activity, impaired judgment, misperception and relative unresponsiveness to environmental stimuli, and variable retrograde amnesia.[2,3] This parasomnia affects up to 4% of adults[4,5] and is a leading cause of sleep-related violence and self-injury.[6-11] Episodes generally arise from impaired awakenings out of slow-wave sleep (SWS; stages 3 and 4) and sometimes from stage 2 sleep.[8,12,13] Investigating somnambulism with objective instruments such as polysomnography (PSG) and audiovisual monitoring is difficult as episodes rarely occur in the sleep laboratory.[1,14,15]

Two groups of factors have been described as facilitating or precipitating sleepwalking in predisposed individuals: those that deepen sleep (e.g., neuroleptics, sleep deprivation) and factors that fragment sleep (e.g., stress, arousing stimuli).[2,16] Sleep deprivation, which

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Figure 1 Study design for sleepwalkers and control subjects

5 sleepwalkers	Sleep period 1 (Baseline)	Sleep deprivation (25 hrs)	Sleep period 2 (Recovery)	One week to one month	Sleep period 3 (Normal with AS)	One week	Sleep deprivation (25 hrs)	Sleep period 4 (Recovery with AS)
5 sleepwalkers	Sleep period 1 (Baseline)	Sleep deprivation (25 hrs)	Sleep period 2 (Recovery)	One week to one month	Sleep deprivation (25 hrs)	Sleep period 3 (Recovery with AS)	One week	Sleep period 4 (Normal with AS)
5 controls	Sleep period 1 (Baseline)	One day to one week	Sleep period 2 (Normal with AS)	One week	Sleep deprivation (25 hrs)	Sleep period 3 (Recovery with AS)		
5 controls	Sleep period 1 (Baseline)	One day to one week	Sleep deprivation (25 hrs)	Sleep period 2 (Recovery with AS)	One week	Sleep period 3 (Normal with AS)		

intensifies pressure for SWS and augments subjects' arousal threshold,[17-19] has been shown to increase the frequency of somnambulistic behaviors in the laboratory.[20-22] Some data also support the idea that forced arousals can precipitate sleepwalking. Early studies of a few young sleepwalkers found that behavioral events could be induced by standing the child on his or her feet during SWS.[1,23-25] Two episodes were also triggered during SWS in one of four children by calling his name.[24] In one study of sleep terror patients, sounding a loud buzzer during SWS induced sleep terrors in two of four patients.[26] However, these limited findings have not been replicated in almost 40 years and, to our knowledge, the efficacy of forced awakenings in inducing somnambulism has never been evaluated in adult patients.

It has been suggested that the probability of recording somnambulistic events in predisposed individuals can be increased by combining factors that deepen and fragment sleep.[27,28] The main goal of the present study was to verify this hypothesis under controlled conditions by combining 25 hours of sleep deprivation with the presentation of auditory stimuli (AS) in sleepwalkers and controls. It was predicted that 1) forced arousals during SWS would induce somnambulistic episodes in sleepwalkers but not in controls and 2) sleep deprivation would increase the efficacy of forced arousals.

METHODS

Subjects. Ten adult sleepwalkers (4 men, 6 women, mean age: 26.3 years, SD: 5.3) and 10 normal controls (3 men, 7 women, mean age: 25.6 years, SD: 3.2) were investigated. Exclusion criteria for all participants consisted of 1) the presence of another sleep disorder[3] or an index (number per hour of sleep) greater than five for respiratory events (apnea-hypopnea index) or greater than 10 for periodic leg movements during sleep (PLMS); 2) the presence of any major psychiatric disorder[29]; 3) the presence or history of any neurologic disorder or auditory deficits; 4) the use of medications that could influence the sleep EEG, sleep architecture, motor activity during sleep, or daytime vigilance; and 5) a transmeridian traveling or night work in the 3 months preceding the study. All sleepwalkers were referred to the Sleep Disorders Clinic by a physician prior to their participation. To be included, sleepwalkers had to report a clinical history (including over the past 6 months) of somnambulism that was not of a psychologically traumatic, neurologic, or medication-induced origin. Eight patients reported sleepwalking since childhood, 1 since adolescence, and 1 since late adolescence and episode frequency ranged from once per month to a few times per week. The protocol was approved by the hospital's ethics committee. All subjects provided written consent prior to the study and received financial compensation for their participation.

Procedures. Figure 1 illustrates the study design. Sleepwalkers were recorded during one baseline night which was used to screen for other sleep disorders. Lights off was between 22:00 and 24:00 and wake time between 6:00 to 8:00 depending on patients' habitual sleep-wake cycle. After the baseline recording, patients were instructed to go about their regular daytime activities but were forbidden from taking naps. They returned to the laboratory in the evening for the sleep deprivation protocol and remained under constant supervision. Recovery sleep was scheduled the next morning, 1 hour after their previous wake time (i.e., following 25 hours of wakefulness). Patients were prohibited from consuming alcohol, caffeine, or other stimulating substances the day prior to and during all laboratory procedures. One to 4 weeks later, sleepwalkers returned to the sleep laboratory for a third PSG recording and were randomly assigned to either normal or recovery sleep conditions with AS. One week later, the AS conditions (during normal or recovery sleep) were reversed. Controls underwent the same protocol with the exception of first recovery sleep period without AS. Thus, sleepwalkers underwent four PSG recordings and controls three PSG recordings, including two recordings with AS in both groups.

Materials. PSG recordings were conducted on a 32-channel Grass polygraph (sensitivity at 7 μV, bandpass at 0.3–100 Hz). Signals were relayed to a PC, digitized at a sampling rate of 256 Hz, and digitally filtered with an upper cutoff frequency of 100 Hz using commercial software (Harmonie, Stellate Systems, Montréal, Canada). EEG recordings and electrode placement were performed according 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 electro-oculograms, submental electromyography, surface electromyography of the bilateral anterior tibialis, and an electrocardiogram. During all baseline recordings, respiration was monitored using an oronasal canula and a thoracoabdominal plethysmograph while oxygen saturation was recorded with a finger pulse oximeter. Twenty-second epochs of PSG were used to score sleep stages according to standard criteria.[30] 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[31] and the microarousal index (number of microarousals

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Table 1 Sleep variables and number of awakenings per sleep stages during normal and recovery sleep with auditory stimuli (AS) in sleepwalkers and control subjects

	Normal sleep with AS		Recovery sleep with AS		*p* Value		
	SW	Controls	SW	Controls	Group	Night	Interaction
Sleep latency, min	17.2 (17.5)	17.2 (24.8)	2.9 (2.1)	4.9 (7.4)	NS	0.013	NS
REM latency, min	139.9 (73.3)	112.9 (70.3)	89.9 (31.7)	96.3 (103.0)	NS	0.039	NS
Total sleep time, min	448.2 (57.7)	429.6 (72.6)	393.0 (62.4)	347.4 (114.4)	NS	<0.001	NS
Sleep efficiency, %	93.1 (3.7)	89.9 (10.5)	90.2 (5.3)	82.8 (18.8)	NS	0.005	NS
% Stage 1	9.9 (4.8)	9.2 (4.9)	9.0 (3.7)	10.8 (8.0)	NS	NS	NS
% Stage 2	62.8 (5.1)	56.7 (7.7)	62.7 (9.1)	55.6 (7.5)	0.035	NS	NS
% SWS	8.2 (4.9)	12.9 (7.2)	11.7 (8.4)	16.8 (6.4)	NS	0.015	NS
% REM	19.2 (6.3)	21.2 (6.9)	16.6 (5.3)	16.9 (8.1)	NS	0.04	NS
Awakenings from stage 1	12.3 (8.3)	13.5 (13.1)	15.7 (8.8)	18.1 (22.8)	NS	NS	NS
Awakenings from stage 2	18.1 (4.2)	16.2 (4.4)	18.7 (6.2)	14.5 (7.2)	NS	NS	NS
Awakenings from SWS	2.6 (3.0)	3.1 (2.6)	5.0 (3.9)	2.7 (1.8)	NS	NS	0.023
Awakenings from REM	6.7 (4.3)	7.0 (5.9)	3.5 (2.4)	5.3 (5.4)	NS	0.003	NS
Microarousal index (nb/h)	11.9 (5.3)	8.7 (5.0)	8.0 (4.3)	6.7 (3.8)	NS	<0.001	NS

Numbers in parentheses denote SD.
SW = sleepwalkers; SWS = slow-wave sleep; NS = not significant.

per hour of sleep) was determined. All patients were continuously videotaped during both sleep assessments.

Auditory stimuli. Sleep periods with AS included four to five separate groups of AS. Each group contained a maximum of six stimuli (three seconds of a pure sound at 1,000 Hz) presented in ascending intensities of 10 dB (from 40 dB to 90 dB) with a minimal interval of 1 minute between two stimuli. AS were presented in the targeted sleep stage after at least 1 minute of stable EEG and EMG until an EEG arousal, a behavioral episode, or the maximum of six AS was reached. When an AS resulted in an arousal or a behavioral episode, the remaining stimuli from the group were presented if the subject returned to the targeted sleep stage during the same sleep cycle. The AS were delivered by earphones inserted into both ears and stimulus presentations were carried out with Neuroscan (Neurosoft Inc., Sterling). The first and second groups of AS were presented during SWS in the first and second NREM-REM sleep cycles whenever possible. The third and fourth groups of AS were presented during stage 2 and REM sleep in the second or subsequent sleep cycles. Subjects with abundant SWS later in the sleep period were also presented with an additional group of AS during SWS. This supplementary group of AS was presented in one sleepwalker during normal sleep and in three sleepwalkers and two controls during recovery sleep.

Scoring of somnambulistic episodes. Behavioral movements identified on participants' PSG recordings were investigated 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.[21] The video recording of each episode was then independently reviewed by two of the authors (M.P., A.Z.) for final classification as a somnambulistic episode. Somniloquy alone was not considered as a partial manifestation of somnambulism. The Kappa inter-rater reliability test indicated a high degree of concordance between the two raters for the presence or absence of somnambulistic episodes (K = 0.873).

Statistical analyses. Between-group comparisons of sleep variables for both sleep periods with AS were performed with a 2 × 2 analysis of variance with one independent factor (group) and one repeated measure (sleep period). A Freedman Test was performed to compare the frequency of somnambulistic behaviors in sleepwalkers during sleep periods with and without AS. The Wilcoxon rank test was used to compare the frequency of episodes in sleepwalkers during sleep periods with AS, as well as the mean percentage of individual AS trials that induced an episode. Chi-squares were used to compare the proportion of patients experiencing at least one episode during PSG recordings. All analyses were performed using SPSSv15 (SPSS, Chicago, IL).

RESULTS

Baseline recordings indicated that all subjects were free of any major sleep disorder. There were no significant differences between sleepwalkers and controls on standard PSG variables, indicating that both groups had comparable sleep architecture. No EEG epileptiform activity or behavioral stereotypies were observed during any of the PSG recordings, including post-sleep deprivation investigations.

PSG variables and awakenings per sleep stage from normal and recovery sleep with AS for sleepwalkers and controls are presented in table 1. Analysis of variance revealed a group × sleep period interaction for the number of awakenings from SWS [$F(1,18) = 6.15$, $p = 0.023$]. Simple effects revealed a significant increase in the number

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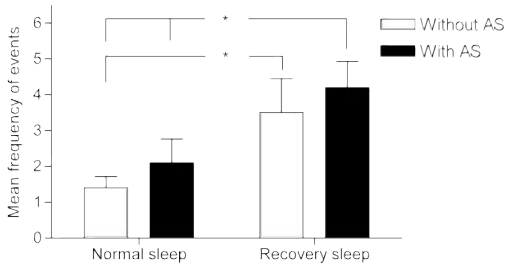
Table 2 Characteristics of somnambulistic events during sleep periods with and without auditory stimuli (AS)

	Baseline sleep	Normal sleep with AS	Recovery sleep without AS	Recovery sleep with AS
Total no. of episodes	14	21	35	42
No. (%) of episodes experimentally induced	—	10 (48)	—	25 (60)
No. (%) of patients with at least one spontaneous episode	8/10 (80)	6/10 (60)	10/10 (100)	7/10 (70)
No. (%) of patients with at least one induced episode	—	5/10 (50)	—	10/10 (100)
No. (%) of episodes arising from SWS	12 (85.7)	18 (85.7)	30 (85.7)	37 (88.1)
No. (%) of episodes arising from stage 2 sleep	2 (14.3)	3 (14.3)	5 (14.3)	5 (11.9)

SWS = slow-wave sleep (stage 3 and 4 sleep).

Figure 2 Mean frequency (SEM) of somnambulistic events recorded in sleepwalkers across sleep periods with and without auditory stimuli (AS)



Differences are significant at a level of $p < 0.05$.

of awakening from SWS in sleepwalkers during recovery sleep as compared to normal sleep ($p = 0.019$) whereas controls did not differ across the two sleep periods. A significant sleep period effect was found for several variables including a reduction in sleep latency [$F(1,18) = 7.66$, $p = 0.013$], in REM latency [$F(1,18) = 4.94$, $p = 0.039$], in total sleep time [$F(1,18) = 20.19$, $p < 0.001$], in sleep efficiency [$F(1,18) = 10.30$, $p = 0.005$], in the percentage of REM sleep [$F(1,18) = 4.90$, $p = 0.040$], in the number of awakenings from REM sleep [$F(1,18) = 12.30$, $p = 0.003$], and in the microarousals index [$F(1,18) = 18.22$, $p < 0.001$] during recovery sleep as compared to normal sleep. Sleep deprivation significantly increased the percentage of SWS [$F(1,18) = 7.30$, $p = 0.015$]. A significant group difference indicated that sleepwalkers had a higher percentage of stage 2 sleep during both sleep periods [$F(1,18) = 5.18$, $p = 0.035$].

The characteristics of spontaneous and induced somnambulistic episodes recorded from sleepwalkers are presented in table 2. None of the control subjects experienced behavioral manifestations that were suggestive of somnambulism during uninterrupted sleep or following forced arousals. No significant group differences were observed between sleepwalkers and controls for the mean number of AS delivered during SWS, stage 2 sleep, and REM sleep (all p values > 0.05).

Figure 2 presents the mean frequency of all somnambulistic events recorded across sleepwalkers' four PSG assessments. Freedman test revealed a significant effect of sleep periods ($p = 0.006$). Post hoc comparisons revealed that the number of sleepwalking episodes significantly increased during recovery sleep with AS when compared to baseline ($p = 0.005$) and to normal sleep with AS ($p = 0.043$). Recovery sleep without AS also significantly increased the mean frequency of episodes when compared to baseline recordings ($p = 0.047$) (see figure 2).

The characteristics of all induced somnambulistic behaviors are presented in table 3. When compared to regular night sleep with AS, recovery sleep with AS resulted in 1) a significantly greater proportion of patients experiencing one or more induced episodes during SWS ($p = 0.005$), 2) a significant increase in the mean frequency of induced episodes during SWS ($p = 0.04$), and 3) a significant increase in the mean percentage of AS trials that induced an episode during SWS ($p = 0.035$). No significant differences were found when this mean percentage was compared across sleep cycles within the same night. The intensity of the AS that induced somnambulistic episodes from SWS was similar during the two sleep periods and ranged from 40 dB to 70 dB during nor-

Table 3 Characteristics of induced somnambulistic events in sleepwalkers during normal and recovery sleep with auditory stimuli (AS)

	Normal sleep with AS	Recovery sleep with AS	*p* Value
Total no. of induced episodes during SWS	7	23	—
No. (%) of patients experiencing at least one induced episode during SWS	3/10 (30)	10/10 (100)	0.005
Mean (SD) frequency of induced episodes during SWS	0.7 (1.3)	2.3 (1.2)	0.040
Mean percentage of AS trials that induced an episode during SWS	19.8 (37.1)	57.3 (31.7)	0.035
Mean (SD) intensity in dB of the AS that induced episodes	48.6 (12.1)	53.5 (11.5)	NS
Total no. of induced episodes during stage 2	3	2	—
No. (%) of patients experiencing one induced episode during stage 2	3/10 (30)	2/10 (20)	NS
Mean percentage of AS trials that induced an episode during stage 2	4.8 (9.4)	2.9 (6.2)	NS

SWS = slow-wave sleep (stage 3 and 4 sleep); NS = not significant.

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mal sleep and from 40 dB to 80 dB during recovery sleep. Three somnambulistic events were also induced after forced awakening from stage 2 during normal sleep and two during recovery stage 2 sleep. No episodes were induced during REM sleep. There were no significant differences between the mean intensity of AS that induced full awakenings during sleepwalkers' SWS at baseline (53.8 ± 9.2 dB) and recovery sleep (55.0 ± 13.1 dB) and controls' SWS at baseline (51.4 ± 10.1 dB) and recovery sleep (52.2 ± 10.0 dB).

DISCUSSION Our results indicate that forced arousals during SWS can experimentally induce somnambulistic episodes in adult sleepwalkers. Moreover, and as predicted, sleep deprivation significantly increased the forced arousals' efficacy. In fact, sleep deprivation nearly tripled the percentage of individual AS trials presented during SWS that induced a behavioral episode (approximately 57% vs 20%). The results from the adult sleepwalkers' normal sleep is consistent with a study[24] of four children in which 7 behavioral episodes were induced in 38 attempts (18%) by standing the children up during SWS. In addition, post-deprivation recovery sleep significantly increased the frequency of induced episodes during SWS when compared to normal sleep, as well as the proportion of sleepwalkers experiencing at least one induced episode during SWS. Recovery sleep resulted in 23 induced episodes from all 10 patients during SWS while only 7 behavioral events were induced in 3 sleepwalkers (30%) during normal SWS.

Our results also demonstrate that sleep deprivation alone is more effective than forced awakenings alone in increasing the probability of recording behavioral events in the sleep laboratory. However, as previously suggested,[27,28] combining a factor that deepens sleep (sleep deprivation) with one that fragments it (AS) yields a greater number of episodes in the laboratory. The fact that none of the controls experienced somnambulistic manifestations indicates that these factors alone or in combination do not lead to sleepwalking, but rather that they increase the probability of such events among those so predisposed. The data thus suggest that this combined protocol can be used as a diagnostic tool or to investigate neural correlates of actual somnambulistic episodes.

These findings are consistent with the view of somnambulism as a disorder of arousal.[1] Data from both sleep recordings with AS highlight the fact that when the pressure for SWS is increased, sleepwalkers experience greater difficulty in transitioning from SWS to another sleep stage or to full cortical arousal following forced awakenings. This, in turn, can propel patients toward somnambulism. This abnormal arousal reaction during SWS may arise from the selective activation of thalamocingulate circuits and the persisting inhibition of other thalamocortical arousal systems.[32] The observation that five episodes were also induced during stage 2 sleep (where delta waves can occupy up to 20% of the EEG record) in 4 of the 10 patients suggests that sleepwalkers' atypical reactions can also be evoked during stage 2.

Some authors have suggested that sleepwalkers are pathologically deep sleepers difficult to awaken.[25,33] One study[25] found that sleepwalkers are more difficult to awaken than controls from NREM sleep using both meaningless and meaningful AS. However, this study did not include quantified comparisons between the two groups. More recently, it has been argued that sleepwalkers can also be more easily arousable from sleep than are control subjects.[34] Our data reveal that the mean intensity of AS that induced somnambulistic episodes during sleepwalkers' SWS is comparable to the mean intensity of AS that induced full awakenings during both sleepwalkers' and controls' SWS (approximately 50 dB). This intensity is also consistent with studies having investigated auditory arousal thresholds in normal adults' SWS.[17,18] These findings thus suggest that sleepwalkers are neither more easily nor more difficult to "awaken" from SWS than are controls but rather that sleepwalkers suffer from an atypical and distinct arousal reaction.

Since the same intensity of AS administered within the same sleep stage can trigger either a somnambulistic episode or a full awakening, sleepwalkers' arousal response is likely modified by other processes. We suggest that AS which induce episodes are likely preceded by increases in EEG slow-wave activity (spectral power in 0.75 to 4.5 Hz), an indicator of sleep intensity.[35] Spontaneous somnambulistic episodes have been shown to be immediately preceded by an increase of slow-wave activity[13] or low delta power (0.25–2.0 Hz),[36] a process that may reflect cortical reaction to brain activation. Furthermore, the fact that sleep deprivation results in increased amounts of SWS and slow-wave activity[35] could account for the increased efficacy of AS in inducing episodes during patients' recovery sleep.

One common finding in normal PSG investigations of sleepwalkers is that they typically experi-

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ence an unusually high number of SWS arousals.[13,14,37] When compared to baseline recordings, we found that 25 hours of sleep deprivation, with recovery sleep being initiated in the morning, significantly increased the number of SWS awakenings in sleepwalkers but not in controls. This finding differs from a previous study of adult sleepwalkers showing that 38 hours of sleep deprivation, with recovery sleep being initiated during the night, did not increase their number of SWS awakenings.[21] These results indicate that sleepwalkers are particularly vulnerable to increased homeostatic sleep pressure following sleep deprivation when sleep is initiated at a circadian time of increasing wake propensity.[38,39]

Significant effects of homeostatic and circadian processes were also found in both groups' daytime recovery sleep in comparison to their baseline sleep. Past findings[39] suggest that the observed increase in SWS and reduction in sleep latency can be attributed to elevated homeostatic pressure following sleep deprivation while circadian effects are likely responsible for the observed reduction in total sleep time, in REM latency, and in sleep efficiency.

When a somnambulistic episode was recorded in the present study, we did not intervene unless the patient's behavior was overly agitated or represented a risk of injury (e.g., attempting to climb over the bed rails). The decision to intervene was made during four episodes from four patients, including three induced episodes. In each case, the patient reported mentation involving perceptual, cognitive, and affective dimensions that accompanied the episode which was motivated by an intrinsic sense of emergency or underlying logic. The phenomenologic contents described corresponded to the observed behaviors. For instance, one patient who quickly removed his pillow and frantically examined the back of his bed reported that he believed that his newborn had fallen behind the bed. Similarly, in the three induced episodes, patients reported hearing a voice asking them to carry out a precise action that precipitated the behavior. For example, one patient suddenly looked up at the ceiling with a fearful expression, started pointing about with one hand, and then proceeded to remove her electrodes with agitation. She later reported hearing someone tell her to tear away the electrodes or she was going to suffocate because the electrodes were attached to the ceiling. Although we do not believe that patients' mental content precipitates somnambulism in and of itself, these observations raise important questions as to the role that phenomenologic experiences play in how somnambulistic episodes unfold and merit further investigations.

Taken together, the data support the hypothesis that via its homeostatic pressure for increased SWS, sleep deprivation facilitates the occurrence of somnambulism in predisposed individuals and that this effect can be augmented by incorporating forced arousals. The findings are consistent with the suggestion that the combination of factors that deepen sleep and that fragment sleep increases sleepwalkers' probability of experiencing an episode. The results thus support the view that the pathophysiology of this disorder of arousal involves a dysfunction in the mechanism responsible for sustaining consolidated periods of SWS.[13,21,37] The tested protocol shows a high sensitivity in adult sleepwalkers thereby suggesting its use for diagnostic purposes. Additional research, however, is required to assess the protocol's sensitivity and specificity, especially with regards to other clinical populations, including patients presenting with other sleep disorders. Finally, the data provide empirical support for the clinical recommendation that sleepwalkers maintain regular sleep cycles, minimize sleep deprivation, and avoid potential environmental disturbances during sleep.

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