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Original Article

# Auditory arousal responses and thresholds during REM and NREM sleep of sleepwalkers and controls

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ABSTRACT

*Background:* It has been suggested that sleepwalkers are more difficult to awaken from sleep than are controls. However, no quantified comparisons have been made between these two populations. The main goal of this study was to assess arousal responsiveness via the presentation of auditory stimuli (AS) in sleepwalkers and controls during normal sleep and recovery sleep following sleep deprivation.
*Methods:* Ten adult sleepwalkers and 10 age-matched control subjects were investigated. After a screening night, participants were presented with AS during slow-wave sleep (SWS), REM, and stage 2 sleep either during normal sleep or daytime recovery sleep following 25 h of sleep deprivation. The AS conditions were then reversed one week later.
*Results:* When compared to controls sleepwalkers necessitated a significantly higher mean AS intensity (in dB) to induce awakenings and arousal responses during REM sleep whereas the two groups' mean values did not differ significantly during SWS and stage 2 sleep. Moreover, when compared to controls sleepwalkers had a significantly lower mean percentage of AS that induced arousal responses during REM sleep while the opposite pattern of results was found during SWS.
*Conclusions:* The data indicate that sleepwalkers have a higher auditory awakening threshold than controls, but only for REM sleep. These findings may reflect a compensatory mechanism of the homeostatic process underlying sleep regulation during sleepwalkers' REM sleep in reaction to their difficulties maintaining consolidated periods of NREM sleep.



## 1. Introduction

Somnambulism (sleepwalking) is a non-rapid eye movement (NREM) sleep parasomnia characterized by behavioral manifestations of varying degrees of complexity and duration, usually initiated out of slow-wave sleep (SWS; stages 3 and 4 sleep), and occasionally from stage 2 sleep [1,2]. Most episodes are characterized by mental confusion, impaired judgment, misperception and relative unresponsiveness to the environment, and variable retrograde amnesia [1]. Somnambulism is considered a "disorder of arousal" [3] as affected individuals experience difficulties transitioning from NREM sleep to full awakening. This parasomnia is common and affects up to 4% of adults [4,5]. Whereas the occurrence of sleepwalking in children is frequently viewed as a relatively benign condition, somnambulism in adults can result in injury to the sleeper or to others [6–11].

Although sleep architecture is generally preserved in patients suffering from somnambulism [6,12], their sleep is characterized by NREM instability [13–15]. In addition, sleepwalkers experience a greater number of spontaneous awakenings and micro-arousals out of SWS [6,14,16–20]. However, whether or not sleepwalkers also show a differential arousal response (e.g., awakening or micro-arousal threshold) in reaction to external sensory stimulation has not been the subject of quantified comparisons.

It has been suggested that sleepwalkers are pathologically deep sleepers difficult to awaken from NREM sleep [21,22]. One pilot investigation of four somnambulistic patients found that they were more difficult to awaken than controls from NREM sleep using both meaningless (loud noises) and meaningful (their names) auditory stimulus (AS) [21]. However, this study was descriptive in nature, limited to a small group of children, and did not include

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quantified comparisons between groups. In healthy participants, auditory awakenings thresholds are higher in SWS than in stage 2 sleep or REM sleep, with generally comparable thresholds between stage 2 and REM sleep [23–25]. Auditory awakening thresholds are also known to increase after sleep deprivation when recovery sleep is initiated during the night [26,27] and are considered a good indicator of the sleep homeostatic process [27,28]. The main goal of the present study was to investigate if, in fact, somnambulistic patients have higher awakening thresholds than controls across different sleep stages by assessing the effects of AS in both groups during normal sleep and following 25 h of sleep deprivation.

## 2. Methods

Polysomnographic (PSG) data from a study [29] on the experimental induction of sleepwalking was used to assess the effects of AS in sleepwalkers and controls during normal sleep and daytime recovery sleep following 25 h of sleep deprivation. Since detailed information on the participants, sleep-deprivation protocol, and materials used has been previously published [29], this information is presented succinctly.

### 2.1. Subjects

Ten adult sleepwalkers (four men, six women, mean age: 26.3 years, SD: 5.3) and ten normal controls (three men, seven women, mean age: 25.6 years, SD: 3.2) were investigated in the sleep laboratory. Exclusion criteria for all participants consisted of: (1) the presence of another sleep disorder or an index (number per hour of sleep) greater than five for respiratory events (apnea–hypopnea index) or greater than ten for periodic leg movements during sleep; (2) the presence of any major psychiatric disorder; (3) the presence or history of any neurological 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) transmeridian travel or night work in the three months preceding the study. All sleepwalkers were referred to the hospital's Sleep Disorders Clinic by a physician prior to their participation. 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.

### 2.2. Procedures

After a baseline night used to screen for other sleep disorders, participants were randomly assigned to either normal or recovery sleep conditions with AS. One week later the normal versus recovery sleep AS conditions were reversed. During normal sleep with AS, lights off was between 22:00 and 24:00 and wake time between 6:00 to 8:00 depending on participants' habitual sleep-wake cycle. Recovery sleep with AS was scheduled one hour after their previous wake time (i.e., following 25 h of wakefulness) and participants were allowed to sleep as long as they wanted to. All participants were prohibited from consuming alcohol, caffeine, or other stimulating substances the day prior to and during all laboratory procedures.

### 2.3. Material

Polysomnographic (PSG) recordings were obtained by means of a 32-channel Grass polygraph (sensitivity at 7 μV/cm, 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 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 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 oxymeter. Twenty-second epochs of PSG were used to score sleep stages according to standard criteria [30].

### 2.4. Auditory stimuli (AS)

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 1000 Hz) presented in ascending intensities of 10 dB (from 40 to 90 dB) with a minimal interval of one minute between two stimuli. AS were presented in the targeted sleep stage after at least one 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 ear-phones inserted into both ears and stimulus presentations were carried out with Neuroscan (Neurosoft Inc., Sterling, USA). The 1st and 2nd groups of AS were presented during SWS in the first and second NREM–REM sleep cycles whenever possible. The 3rd and 4th groups of AS were presented during REM and stage 2 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 to a limited number of subjects and was not included in the present analysis. During baseline sleep the group of AS from REM sleep was delivered in the second sleep cycle for eight sleepwalkers and eight controls, and in the third sleep cycle for the remaining subjects. During recovery sleep the group of AS from REM sleep was delivered in the second sleep cycle for six sleepwalkers and five controls, in the third sleep cycle for two sleepwalkers and two controls, and in the fourth sleep cycle for two sleepwalkers and three controls. During stage 2 sleep, the group of AS was delivered during the third or the fourth sleep cycles for most of the participants during both sleep periods.

Experimentally induced awakenings were defined as a transition from any sleep stage to stage of wakefulness for at least 10 s within 3 s after the AS. In sleepwalkers, experimentally induced transitions from NREM sleep to somnambulistic episodes were also categorized as an induced awakening to avoid unjustified data exclusion. This decision was based on the fact that, in some sleepwalkers, the presentation of AS during their SWS only induced somnambulistic episodes as opposed to complete awakenings and that the mean intensity of AS that induced somnambulistic episodes versus awakenings in patients was not significantly different [29]. Experimentally induced micro-arousals were scored on the C3/A2 lead according to the American Sleep Disorder Association's criteria [31] within 3 s after the AS. Induced arousal responses refer to the induced awakenings and micro-arousals. Induced arousal responses were scored independently by certified sleep technicians and the first author. The Kappa inter-rater reliability test indicated a high degree of concordance between scorers ($K = 0.943$). The mean intensity of AS required to induce awakenings or arousal responses and the mean percentage of AS followed by awakenings or arousal responses were calculated for each subject for SWS, REM sleep, and stage 2 sleep. If participants were not awakened by the 90 dB AS a score of 100 dB was assigned for the analysis. A similar procedure has been used in other studies [27].

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### 2.5. Statistical analyses

Between-group comparisons for AS intensity and percentage of trials that induced awakenings and arousal responses for both sleep periods were performed with a $2 \times 2 \times 3$ GLM repeated measures analysis with one between-factor (group) and two within-factors (sleep period and sleep stage). Chi-squares were used to compare the proportion of sleepwalkers and controls that experienced at least one induced awakening or arousal response during baseline or recovery sleep with Yate's correction. Analyses were performed using PASW Statistics 18 (SPSS, Chicago, IL, USA).

## 3. Results

Baseline recordings (without AS) indicated that all subjects were free of any major sleep disorder and showed that sleepwalkers' sleep architecture was comparable to that of the controls. Basic PSG variables along with the effects of the AS and 25 h of sleep deprivation on the PSG data of sleepwalkers and controls, as well as on the frequency of somnambulistic episodes have been detailed elsewhere [29]. It should be noted, however, that no significant differences were found between the mean intensity of AS that induced somnambulistic events during sleepwalkers' SWS and the mean intensity of AS that fully awakened sleepwalkers and controls from SWS.

Table 1 presents the mean intensity (in dB) of AS that induced awakenings and arousal responses during SWS, stage 2, and REM sleep recorded from sleepwalkers and controls during normal and recovery sleep periods. There was no significant group × sleep period × sleep stage interaction for these two variables. A significant group × sleep stage interaction was found for the mean intensity of AS that induced an awakening ($F[2, 36] = 7.68$, $p = 0.002$) and an arousal response ($F[2, 36] = 7.66$, $p = 0.002$). Simple effect contrasts revealed that sleepwalkers had a significantly higher AS intensity that induced awakenings ($F[1, 18] = 9.62$, $p = 0.006$) as well as arousal responses ($F[1, 18] = 9.34$, $p = 0.007$) during REM sleep (during both sleep periods) when compared to controls. Sleepwalkers and controls did not differ significantly during SWS or stage 2 sleep. A significant effect for sleep period was also found for the mean intensity of AS that induced awakenings ($F[1, 18] = 6.55$, $p = 0.020$), revealing a significant reduction in the intensity of AS that induced awakenings during recovery sleep as compared to normal baseline sleep in both groups (for SWS, stage 2, and REM sleep).

The mean percentage (±SEM) of AS that experimentally induced awakenings during both sleep periods for sleepwalkers and controls are presented in Fig. 1. There was a significant group - sleep period × sleep stage interaction ($F[2, 36] = 4.84$, $p = 0.014$) for the mean percentage of experimentally-induced awakenings. Contrast analyses indicated that, when compared to controls, sleepwalkers had a significantly lower mean percentage of AS that lead to awakenings ($F[1, 18] = 6.51$, $p = 0.020$) during REM sleep. Analyses also revealed that a significantly higher percentage of AS induced awakenings during sleepwalkers' recovery SWS sleep in comparison to controls ($F[1, 18] = 18.97$, $p < 0.001$) and a significant increase in the percentage of AS that induced awakenings during sleepwalkers' recovery SWS as compared to their normal

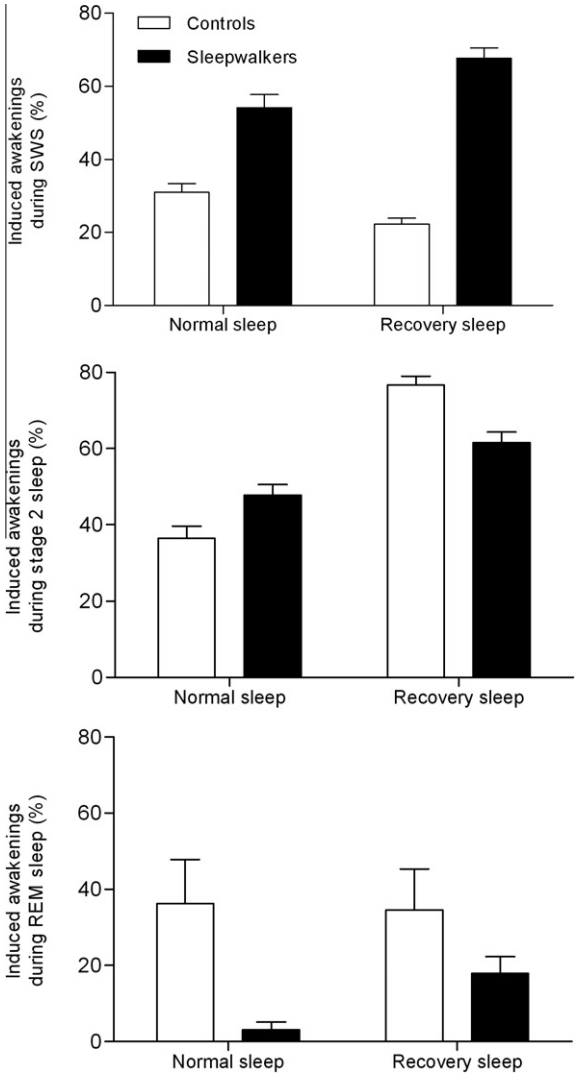


**Fig. 1.** Mean percentage (%) (±SEM) of auditory stimuli (AS) intensity that induced awakenings during SWS, stage 2, and REM sleep in sleepwalkers and controls during normal and recovery sleep.

**Table 1**
Mean intensity (in dB) of auditory stimuli (AS) that induced awakenings or arousal responses (awakenings and micro-arousals) during SWS, stage 2, and REM sleep in sleepwalkers (SW) and controls during normal and recovery sleep.

| Mean AS intensity (in dB) that induced: | Normal sleep | | Recovery sleep | |
|---|---|---|---|---|
| | SW | Controls | SW | Controls |
| Awakenings during SWS | 53.8 (11.1) | 56.3 (13.5) | 53.4 (10.1) | 59.8 (14.4) |
| Arousal responses during SWS | 52.3 (9.9) | 53.6 (8.6) | 52.7 (8.4) | 57.4 (9.9) |
| Awakenings during stage 2 | 55.3 (11.7) | 63.0 (20.7) | 51.0 (7.9) | 48.1 (12.6) |
| Arousal responses during stage 2 | 53.9 (9.6) | 57.8 (21.0) | 50.0 (6.4) | 48.7 (12.3) |
| Awakenings during REM | 94.1 (8.6) | 70.0 (22.0) | 80.6 (13.3) | 67.2 (22.5) |
| Arousal responses during REM | 80.4 (16.2) | 64.0 (19.4) | 80.2 (13.7) | 63.1 (19.1) |

Numbers in parentheses denote SD.
SW = sleepwalkers.
SWS = slow-wave sleep.

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sleep ($F[1, 9] = 12.03$, $p = 0.007$). Control participants did not differ significantly across the two sleep periods during SWS. Finally, there was a significant sleep period effect for the mean percentage of AS that induced awakenings during stage 2 sleep (for both groups) ($F[1, 18] = 11.57$, $p = 0.003$).

The mean percentages (±SEM) of AS that experimentally induced arousal responses during both sleep periods for sleepwalkers and controls are presented in Fig. 2. There was no significant group × sleep period × sleep stage interaction for the mean percentage of induced arousal responses during sleep. A significant group × sleep stage interaction was found. Contrast analyses revealed that, when compared to controls, sleepwalkers had a greater mean percentage of AS that induced arousal responses during SWS ($F[1, 18] = 7.54$, $p = 0.013$) and a significantly lower mean percentage of AS that resulted in arousal responses ($F[1, 18] = 7.73$, $p = 0.012$) during REM sleep. There was no significant group difference for the mean percentage of AS that induced arousal responses during stage 2 sleep.

A significantly lower proportion of sleepwalkers (20%) than controls (80%) experienced at least one induced awakening during baseline REM sleep ($\chi^2$ [1, $N = 20$] = 1.88, $p = 0.03$). There was no other significant difference in the proportion of sleepwalkers and controls experiencing at least one induced awakening or arousal response during baseline or recovery sleep.

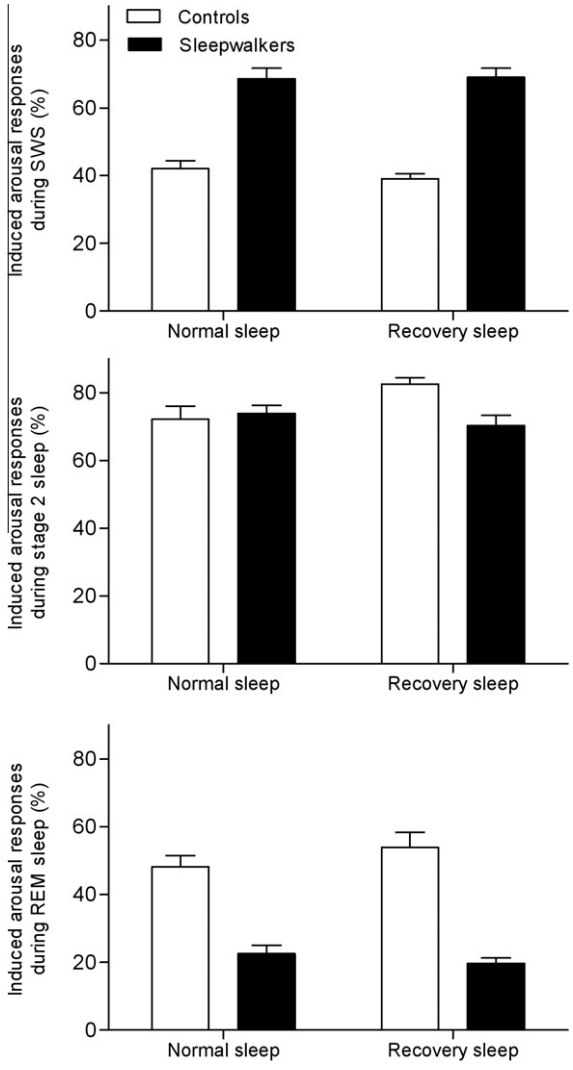


**Fig. 2.** Mean percentage (%) (±SEM) of auditory stimuli (AS) intensity that induced arousal responses (awakenings and micro-arousals) during SWS, stage 2, and REM sleep in sleepwalkers and controls during normal and recovery sleep.

## 4. Discussion

The present study shows that adult patients with a history of sleepwalking have a significantly higher auditory awakening and arousal threshold than healthy controls, but only for REM sleep. This finding was consistent across all REM sleep variables investigated during normal sleep as well as recovery sleep following 25 h of sleep deprivation (see Table 1 and Figs. 1 and 2). Hence, when compared to controls, sleepwalkers showed both a higher intensity of AS required to induce awakenings or arousals and a lower percentage of AS that induced awakenings or arousals during the two periods of sleep recordings. In addition, 80% of the control participants experienced at least one induced awakening from AS during baseline REM sleep as compared to only 20% of the sleepwalkers. One unexpected finding was that the two groups' auditory awakening and arousal thresholds did not differ significantly for SWS and stage 2 sleep. This contrasts with clinical impressions [22,32] as well as a pilot study of four somnambulistic children suggesting that they were more difficult to awaken from NREM than were controls [21]. However, more recent investigations of auditory awakening thresholds with quantified measures have shown that normal children are in fact very difficult to awaken during SWS [25,33,34]. Whether somnambulistic children differ from controls as do adult sleepwalkers on the basis of their awakening thresholds from REM sleep, or simply from NREM sleep, remains to be determined. Also unknown is whether the use of meaningful AS (e.g., participant's name) would have yielded differing arousal thresholds across groups or sleep stages when compared to the pure tone AS used in the present study.

Several studies have shown that the sleep of somnambulistic patients is characterized by NREM instability, including an increased number of spontaneous arousals from SWS [13–20]. In line with these results, our findings indicate that the mean percentage of AS that experimentally-induced arousal responses during SWS is significantly higher in sleepwalkers compared to controls. This finding indicates that sleepwalkers are particularly vulnerable to the presentation of AS during SWS. In addition, our results suggest that a noteworthy interplay of NREM and REM sleep dynamics may also be observed in sleepwalkers. Our data reveal an opposing pattern of results between sleepwalkers and controls for the mean percentage of AS that induced arousal responses during SWS and REM sleep from both the normal and recovery sleep periods. When compared to controls, sleepwalkers had a significantly higher percentage of AS that induced an arousal response from SWS combined with a significantly lower percentage of AS that induced an arousal response from REM sleep. These contrasts are similar to findings by Joncas et al. [19] in their investigation of 40 h of sleep deprivation in sleepwalkers and controls. Specifically, they found that, when compared to baseline sleep recordings, sleepwalkers experienced a significantly greater number of spontaneous awakenings from SWS, as well as a significant decrease in the number of spontaneous awakenings from REM during nocturnal recovery sleep, whereas the control subjects did not differ across the two nights.

These results suggest that a compensatory mechanism involving the homeostatic process underlying sleep regulation is at work during sleepwalkers' REM sleep in reaction to their difficulties maintaining consolidated periods of NREM sleep. This hypothesis is consistent with other findings. EEG slow-wave activity (SWA; spectral power in the 0.75 and 4.5 Hz band, also called delta frequency band) is a quantitative measure of sleep dynamics, an indicator of sleep intensity [35,36], and reflects the accumulation

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of sleep pressure as a function of prior time awake and its dissipation during sleep [35]. Sleepwalkers' NREM instability interferes with the build-up of SWA across the night and, when compared to healthy controls, they have significantly lower overall SWA during NREM sleep [14,15,17,18]. Studies on the effects of sleep deprivation on EEG activity have shown that recovery sleep enhances delta frequency power during both NREM and REM sleep [37,38]. We thus propose that sleepwalkers' sleep fragmentation (including the experience of partial episodes) increases their homeostatic sleep pressure, which in turn results in increased pressure for SWA across the sleep episode, including during REM sleep, as a compensatory mechanism.

This working hypothesis is also consistent with the significant greater percentage of delta frequency band (approximately 22%) during total sleep time found in adults with sleep terrors or sleepwalking in comparison to controls [17]. Furthermore, this significantly greater percentage of delta frequency was not statistically significant when the analyses were solely performed during SWS [17]. Further investigations of the homeostatic sleep regulation of sleepwalkers' NREM as well as REM sleep are required to elucidate the nature and extent of their interplay in parasomniacs.

Studies have shown that auditory awakening thresholds increase during stage 2 sleep in healthy participants after sleep deprivation when recovery sleep is initiated during the night (i.e., when sleep is initiated during the usual circadian timing for sleep propensity) [26,27]. When compared to our baseline recordings, 25 h of sleep deprivation with recovery sleep being initiated in the morning significantly decreased the intensity of AS required to induce awakenings from SWS, REM and stage 2 sleep in patients as well as controls. These results may be attributable to circadian effects as daytime recovery sleep is known to increase sleep fragmentation and reduce sleep efficiency since the sleep episode occurred at a circadian time of increasing wake propensity [39–41]. Interestingly, we found no significant difference between the two sleep periods for the intensity of AS that induced arousal responses during these sleep stages.

In summary, the present study was the first to use quantified measures to investigate the auditory awakening thresholds of sleepwalkers in comparison to controls. While two groups' auditory arousal thresholds did not differ significantly for SWS and stage 2 sleep, sleepwalkers did have a higher auditory arousal threshold from REM sleep. We suggest that sleepwalkers' higher auditory awakening threshold from REM sleep reflects a compensatory mechanism related to the homeostatic process of sleep regulation. While NREM sleep instability constitutes a well documented core feature of sleepwalkers' PSGs, the present findings indicate that basic REM sleep parameters in sleepwalkers should, at the very least, be reported, as they have long been completely ignored in most PSG studies of parasomniacs. The study of homeostatic aspects of sleep regulation during both NREM and REM in sleepwalkers may also help specify how and why the sleep of parasomniacs differs from that of healthy controls.

## Conflict of Interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: doi:10.1016/j.sleep.2011.10.031.

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