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Slow wave activity and slow oscillations in sleepwalkers and controls: effects of 38 h of sleep deprivation

ROSEMARIE PERRAULT[1,2], JULIE CARRIER[1,2], ALEX DESAUTELS[1], JACQUES MONTPLAISIR[1,3] and ANTONIO ZADRA[1,2]

[1]Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Cœur, Montréal, Québec, Canada, [2]Department of Psychology, Université de Montréal, Montréal, Québec, Canada and [3]Department of Psychiatry, Université de Montréal, Montréal, Québec, Canada

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Correspondence
Antonio Zadra PhD, Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Cœur de Montréal, 5400 Gouin Boulevard Ouest, Montréal, Québec H4J 1C5, Canada.
Tel.: 514-343-6626;
fax: 514-343-2285;
e-mail: antonio.zadra@umontreal.ca

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SUMMARY

Sleepwalkers have been shown to have an unusually high number of arousals from slow wave sleep and lower slow wave activity (SWA) power during the night than controls. Because sleep deprivation increases the frequency of slow wave sleep (SWS) arousals in sleepwalkers, it may also affect the expression of the homeostatic process to a greater extent than shown previously. We thus investigated SWA power as well as slow wave oscillation (SWO) density in 10 sleepwalkers and nine controls at baseline and following 38 h of sleep deprivation. There was a significant increase in SWA during participants' recovery sleep, especially during their second non-rapid eye movement (NREM) period. SWO density was similarly increased during recovery sleep's first two NREM periods. A fronto-central gradient in SWA and SWO was also present on both nights. However, no group differences were noted on any of the 2 nights on SWA or SWO. This unexpected result may be related to the heterogeneity of sleepwalkers as a population, as well as our small sample size. SWA pressure after extended sleep deprivation may also result in a ceiling effect in both sleepwalkers and controls.

INTRODUCTION

Somnambulism (or sleepwalking) is a common parasomnia characterized by complex behaviours usually initiated during arousals from slow wave sleep (SWS). Although adult somnambulistic patients and controls do not differ significantly in terms of overall sleep architecture, sleepwalkers experience an unusually high number of arousals and brief microarousals out of SWS (Blatt *et al.*, 1991; Espa *et al.*, 2000; Gaudreau *et al.*, 2000; Pilon *et al.*, 2008). Sleepwalkers also have lower overall EEG slow wave activity (SWA: spectral power in the 0.75–4.5 Hz band) than controls, with the greatest difference occurring during the first non-rapid eye movement period (NREMP) (Espa *et al.*, 2000; Gaudreau *et al.*, 2000; Guilleminault *et al.*, 2001). Given that SWA is a good indicator of the expression of the homeostatic process, these findings suggest that normal SWA build-up may be impeded by sleepwalkers' frequent arousals from SWS, especially during the first two sleep cycles, when most of their awakenings from SWS occur. More recently, we found that the density of slow wave oscillations (SWO), defined as high-amplitude (>75 μV) waves with a peak frequency of approximately 0.7–0.8 Hz, increased significantly immediately preceding the onset of somnambulistic episodes (Jaar *et al.*, 2010), but SWO have yet to be investigated in relation to sleepwalkers' overall NREM sleep.

Sleep deprivation is a powerful tool to assess sleep homeostasis, and both SWA and SWO can be modulated by homeostatic sleep pressure. For instance, recovery sleep in normal subjects is associated with increases in SWA and SWO density and fewer awakenings out of NREM sleep (Bersagliere and Achermann, 2010; Borbely *et al.*, 1981; Carrier *et al.*, 2011). However, sleepwalkers show a different physiological response to sleep deprivation, as their recovery sleep is characterized by increased awakenings from SWS (but not from Stage 2 or REM sleep) in comparison to baseline values (Zadra *et al.*, 2008). Furthermore, sleep deprivation also increases the frequency of somnambulistic events recorded during sleepwalkers' recovery sleep (Joncas *et al.*, 2002; Zadra *et al.*, 2008). One hypothesis is that this increase in sleepwalkers' arousals out of SWS alters the dynamics of their SWA and SWO in comparison to baseline

recordings. We thus investigated SWA and SWO in sleepwalkers compared to controls by evaluating the effect of enhanced homeostatic pressure on their recovery sleep.

In order to investigate the effect of an enhanced pressure of the homeostatic process on the dynamics of SWA and SWO in sleepwalkers and controls, the present pilot study used the polysomnographic (PSG) data collected from both groups by Joncas *et al.* (2002) during normal sleep and following 38 h of sleep deprivation. The following predictions were tested: (a) sleepwalkers would show a lower SWA and SWO density at baseline compared to controls and (b) that sleep deprivation would augment SWA and SWO density in both groups, but less so in sleepwalkers.

METHODS

Participants

Ten adult sleepwalkers (three men, seven women, mean age: 25.1 $\pm$ 4.1) and 10 sex- and age-matched controls (mean age 25.2 $\pm$ 3.6) were investigated. Patients were referred to our Sleep Disorders Center prior to their participation, were free of other sleep disorders, major psychiatric or neurological disorders and did not take medications known to influence sleep architecture or sleep electroencephalography (EEG). The frequency of patients' somnambulistic episodes varied from a few times per month to a few times per week, and in all but one of the patients the disorder began during childhood. None of the 20 participants had been included in our previous studies of EEG spectral analysis in sleepwalking (Gaudreau *et al.*, 2000; Jaar *et al.*, 2010). The study was approved by the hospital's ethics and scientific committee and signed consent was obtained from each participant.

Material

Polysomnographic (PSG) recordings were conducted on a 32-channel Grass polygraph (sensitivity at 7 μV, bandpass at 0.3–100 Hz). Signals were digitized at a sampling rate of 128 Hz and filtered digitally with an upper cutoff frequency of 64 Hz. EEG recordings and electrode placement were performed according the 10–20 system with a linked-ear reference. Twenty-second epochs from the C3/A2 lead were used to score sleep stages. A sleep cycle was defined as a NREM sleep episode lasting at least 15 min followed by an REM episode lasting at least 5 min, except for the first REM episode.

Procedures

The PSG recordings were conducted over 3 nights, including an initial screening night to ensure that they were free of any major sleep disorder. The second PSG recording night served as a baseline recording. One week later, subjects returned to the laboratory for the 38-h sleep deprivation protocol and spent the night as well as the following day under constant supervision. During this period, participants were prohibited from taking any stimulating substances (caffeine, nicotine, etc.). Their sleep was recorded during their recovery night and subjects informed that they could sleep as long as they wanted. To control for any habituation effect to the recording procedure, baseline and recovery sleep conditions were counterbalanced, as one-half of the subjects had the sleep deprivation on their third and last visit, while the other half had it on their second stay with the baseline recording occurring on the third visit. Participants were videotaped continuously throughout the 3 recording nights.

Spectral analyses for SWA were computed with a commercial software package on F3 and C3 using a fast-Fourier transform (cosine tapering) on 4-s artefact-free sections. Epochs containing artefacts were considered as missing data to preserve sleep continuity. An in-house software package was used to detect SWO automatically on artefact-free derivations F3 and C3, according to published criteria (Carrier *et al.*, 2011): (1) negative peak <-40 μV; (2) peak-to-peak amplitude >75 μV; (3) duration of negative deflection > 125 ms and < 1500 ms; and (4) duration of positive deflection <1000 ms. SWO density was defined as the number of SWO per minute of NREM sleep. To control for differences in sleep duration between baseline and recovery conditions, mean sleep SWA and SWO density included the first four NREM periods.

The impact of sleep deprivation on SWA and SWO in sleepwalkers and controls was assessed with three-way analyses of variance (ANOVAS) with one independent factor (group) and two repeated measures (night and derivation). Changes in SWA and SWO across NREM periods were investigated separately for C3 and F3, with three-way ANOVAS with alpha levels adjusted for non-sphericity (Huynh–Feldt). Due to a corrupt data file, PSG recordings from one control were unusable; analyses were thus performed on 10 sleepwalkers and nine controls. Results were considered significant when $P \leq 0.05$.

RESULTS

The effects of sleep deprivation on the behavioural manifestations, as well as on the polysomnographic data of sleepwalkers and controls, have been reported previously (Joncas *et al.*, 2002). A total of seven behavioural manifestations (five from SWS, two from Stage 2) were recorded from four sleepwalkers at baseline and 37 episodes (30 from SWS, seven from Stage 2) from nine sleepwalkers during recovery sleep. None of the controls had any behavioural manifestations on either of the 2 nights. Sleepwalkers had a significantly higher number of arousals from SWS compared to control subjects on both nights, but there was no significant effect of sleep deprivation on arousals.

Analyses of night-time SWA revealed a main effect for both night and derivation, with significantly greater spectral power during the recovery night compared to baseline ($F_{(1,16)}$ =14.44, $P < 0.05$), and on F3 compared with C3 ($F_{(1,16)}$ =42.60, $P < 0.05$). There was no significant group × night interaction, indicating that sleepwalkers and controls did not react differently to sleep deprivation in their overall SWA rebound.

Fig. 1 depicts the dynamics of SWA on the central derivation across each NREMP for baseline and recovery nights in sleepwalkers (top) and controls (bottom). Results for SWO density are presented in Fig. 2. On both F3 and C3, there was a significant ($P < 0.05$) night × NREMP interaction, indicating that the typical decrease in SWA across NREMP was greater during the recovery night when compared to baseline. Tests for simple main effects revealed that SWA power was significantly greater during the recovery night, but only for the second NREMP ($P < 0.049$).

There was a significant night × derivation interaction for night-time SWO density ($F_{(1,17)}$=13.57, $P < 0.002$). Simple main effects revealed that night-time SWO density was significantly greater in F3 compared to C3, and that this difference was magnified during recovery sleep ($P < 0.001$). There was no significant group effect for SWO density.

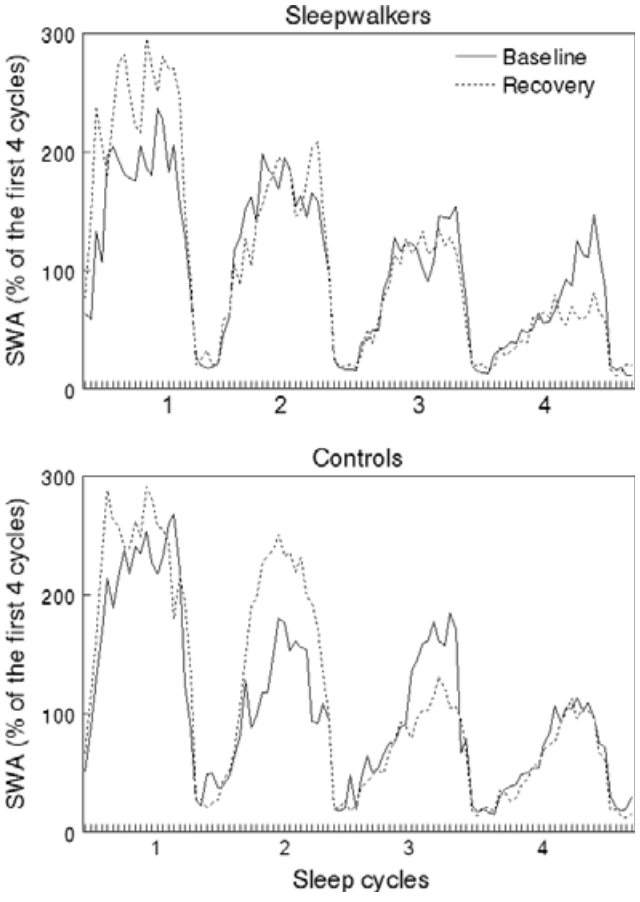

Figure 1. Dynamics of slow wave activity (SWA) across the first four sleep cycles in sleepwalkers and controls during baseline and recovery sleep.

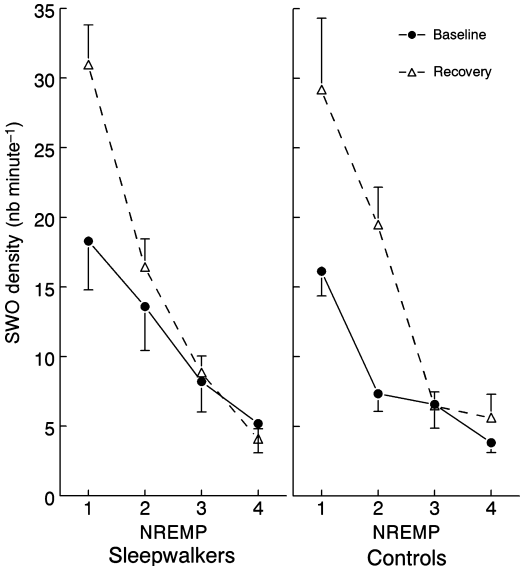

Figure 2. Slow wave oscillation (SWO) density (+standard error of the mean) across the first four sleep cycles in sleepwalkers and controls during baseline and recovery sleep.

Significant night × NREMP interactions were found for SWO density on C3 and F3; ($P < 0.02$). For both derivations, SWO density was significantly greater during the recovery night's first two NREMPs compared with baseline, without significant group differences.

DISCUSSION

This study used sleep deprivation to investigate SWA dynamics and SWO density in sleepwalkers and controls. Consistent with previous findings, SWA and SWO density was greater during recovery sleep compared to the baseline night, with a fronto-central gradient underlying SWA expression. However, no significant differences in absolute SWA were found between sleepwalkers and controls at baseline. Although this result is based on a small sample, and should therefore be considered with caution, it should be noted that significant differences between sleepwalkers' and controls' baseline level of SWA have been reported, with sample sizes ranging from 11 to 15 subjects per group (Espa *et al.*, 2000; Gaudreau *et al.*, 2000; Guilleminault *et al.*, 2001). This unexpected result may also be related to the heterogeneity of sleepwalkers as a population. In our previous study (Gaudreau *et al.*, 2000), somnambulistic episodes as well as awakenings from SWS occurred mainly during the second half of the first sleep cycle, resulting in a significant decrease in SWA for that time-period. By contrast, the SWS awakenings and episodes recorded from the patients in the present study were distributed more equally across the night (only 45% occurred during sleepwalker's first sleep cycle), possibly accounting for the lack of a significant decrease in their SWA at baseline.

Sleep deprivation augments SWA, but as it also increases behavioural manifestations in sleepwalkers, a lesser increase

in SWA during their recovery sleep was predicted in comparison to controls. Although there was an indication of this diminished increase in SWA for cycle 2 in sleepwalkers (see Fig. 1), our findings indicate that sleepwalkers and controls did not differ significantly in terms of SWA and SWO in their response to sleep deprivation. It is possible that SWA pressure after 38-h sleep deprivation creates a ceiling effect, masking between-group differences. When part of this pressure is relieved after the first cycle, differences may begin to emerge during subsequent cycles.

It is well established that even on nights without episodes, both adult and children sleepwalkers show an increased cyclic alternating pattern (CAP) rate, a measure of NREM instability which expresses the organized complexity of arousal-related phasic events in NREM sleep (e.g. Guilleminault *et al*., 2005; Zucconi *et al*., 1995). The study of CAP allows for an analysis of the time structure of SWS activity that cannot be captured with measures of SWA. It has also been suggested that the atypical CAP parameters observed in patients with NREM sleep parasomnias lead to recurrent SWS fragmentation, thereby contributing to the occurrence of SWS parasomnia (Bruni *et al*., 2008; Guilleminault, 2006; Guilleminault *et al*., 2006). How sleepwalkers' CAP-related parameters react to sleep deprivation remains to be determined, but the systematic study of sleepwalkers' CAP-related indices, in conjunction with measures of SWA and SWO, could help to refine our understanding of the physiological processes underlying their NREM instability.

One strength of the present study is the 38-h duration of sleep deprivation, which allowed all participants to be recorded during nocturnal sleep periods before and after sleep deprivation. However, this sleep deprivation protocol is very demanding and the present pilot results are based on a relatively small sample size, with statistical limitations including conservative statistical power. Observed effect sizes were small to medium (between 0.13 and 0.3), and power analyses reveal that approximately 40–50 subjects would have been be required for differences in SWA and SWO parameters recorded on both nights to reach statistical significance. Finally, while recovery sleep initiated during the night did not increase our patients' arousals significantly compared to baseline (see Joncas *et al*., 2002), sleep deprivation with recovery sleep initiated in the morning has been shown to increase the number of SWS awakenings significantly in sleepwalkers, but not in controls (Pilon *et al*., 2008). Taken together, these findings 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. The study of SWA, SWO and CAP rate during sleepwalkers' daytime recovery sleep is suggested as a next step in this line of research.

REFERENCES

Bersagliere, A. and Achermann, P. Slow oscillations in human non-rapid eye movement sleep electroencephalogram: effects of increased sleep pressure. *J. Sleep Res*., 2010, 19: 228–237.

Blatt, I., Peled, R., Gadoth, N. and Lavie, P. The value of sleep recording in evaluating somnambulism in young adults. *Electroencephalogr. Clin. Neurophysiol*., 1991, 78: 407–412.

Borbely, A. A., Baumann, F., Brandeis, D., Strauch, I. and Lehmann, D. Sleep deprivation: effect on sleep stages and EEG power density in man. *Electroencephalogr. Clin. Neurophysiol*., 1981, 51: 483–495.

Bruni, O., Ferri, R., Novelli, L., Finotti., E., Milano, S. and Guilleminault, C. NREM sleep instability in children with sleep terrors: the role of slow wave activity interruptions. *Clin. Neurophysiol*., 2008, 19: 985–992.

Carrier, J., Viens, I., Poirier, G. *et al.* Sleep slow wave changes during the middle years of life. *Eur. J. Neurosci*., 2011, 33: 758–766.

Espa, F., Ondzé, B., Deglise, P., Billiard, M. and Besset, A. Sleep architecture, slow wave activity, and sleep spindles in adult patients with sleepwalking and sleep terrors. *Clin. Neurophysiol*., 2000, 111: 929–939.

Gaudreau, H., Joncas, S., Zadra, A. and Montplaisir, J. Dynamics of slow-wave activity during the NREM Sleep of sleepwalkers and control subjects. *Sleep*, 2000, 23: 755–760.

Guilleminault, C. Hypersynchronous slow delta, cyclic alternating pattern and sleepwalking. *Sleep*, 2006, 29: 14–15.

Guilleminault, C., Poyares, D., Aftab, F. A. and Palombini, L. Sleep and wakefulness in somnambulism: a spectral analysis study. *J. Psychosom. Res*., 2001, 51: 411–416.

Guilleminault, C., Lee, J. H., Chan, A., Lopes, M.-C., Huang, Y. and da Rosa, A. Non-REM sleep instability in recurrent sleepwalking in pre-pubertal children. *Sleep Med*., 2005, 6: 515–521.

Guilleminault, C., Kirisoglu, C., da Rosa, A. C., Lopes, C. and Chan, A. Sleepwalking, a disorder of NREM sleep instability. *Sleep Med*., 2006, 7: 163–170.

Jaar, O., Pilon, M., Carrier, J., Montplaisir, J. and Zadra, A. Analysis of slow-wave activity and slow-wave oscillations prior to somnambulism. *Sleep*, 2010, 33: 1511–1516.

Joncas, S., Zadra, A., Paquet, J. and Montplaisir, J. The value of sleep deprivation as a diagnostic tool in adult sleepwalkers. *Neurology*, 2002, 58: 936–940.

Pilon, M., Montplaisir, J. and Zadra, A. Precipitating factors of somnambulism: impact of sleep deprivation and forced arousals. *Neurology*, 2008, 70: 2284–2290.

Zadra, A., Pilon, M. and Montplaisir, J. Polysomnographic diagnosis of sleepwalking: effects of sleep deprivation. *Ann. Neurol*., 2008, 63: 513–519.

Zucconi, M., Oldani, A., Ferini-Strambi, L. and Smirne, S. Arousal fluctuations in non-rapid eye movement parasomnias: the role of cyclic alternating pattern as a measure of sleep instability. *J. Clin. Neurophysiol*., 1995, 12: 147–154.