



Original Article

Daytime somnolence in adult sleepwalkers



Alex Desautels^{a,b}, Antonio Zadra^{a,c}, Marc-Antoine Labelle^{a,c}, Yves Dauvilliers^d, Dominique Petit^{a,e}, Jacques Montplaisir^{a,e,*}

^a Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Cœur, Montreal, Canada

^b Department of Medicine, Université de Montréal, Service of Neurology, Hôpital du Sacré-Coeur, Montreal, Canada

^c Department of Psychology, Université de Montréal, Montreal, Canada

^d National Reference Network for Narcolepsy, Sleep-Disorders Center, Department of Neurology, Hôpital Gui de Chauliac, Inserm U1061, UM1, Montpellier, France

^e Department of Psychiatry, Université de Montréal, Montreal, Canada

ARTICLE INFO

Article history:

Received 7 February 2013

Received in revised form 9 April 2013

Accepted 10 April 2013

Available online 29 August 2013

Keywords:

Parasomnia

Sleepwalking

Somnolence

Quantitative EEG

Epworth Sleepiness Scale

Slow-wave activity

ABSTRACT

Objectives: Sleepwalkers often complain of excessive daytime somnolence (EDS). Our retrospective study aimed to document the presence of EDS in a substantial sample of sleepwalkers and to explore the contribution of other sleep disorders, nocturnal sleep disruption, and sleep depth to the alteration of their daytime vigilance.

Methods: Seventy adult sleepwalkers and 70 control subjects completed the Epworth Sleepiness Scale (ESS). Sleepwalkers also were studied for one night in the sleep laboratory. We compared the sleep profiles of 32 somnolent vs 38 nonsomnolent sleepwalkers and investigated the relationship between ESS scores and sleep-related variables.

Results: No differences were found in polysomnographic (PSG) parameters. Slow-wave activity (SWA) also was similar in the two subgroups. Sleepwalkers' ESS scores were not correlated with their body mass index (BMI) or periodic limb movements during sleep (PLMS) index, but they tended to be negatively correlated with indices of respiratory events.

Conclusions: The EDS reported by adult sleepwalkers does not appear to be explained by the presence of concomitant sleep disorders or PSG signs of nocturnal sleep disruption. These results raise the possibility that EDS is part of the sleepwalking phenotype and that it is linked to its underlying pathophysiology.

© 2013 Elsevier B.V. All rights reserved.

1. Introduction

Somnambulism (or sleepwalking) is defined by the American Academy of Sleep Medicine as “a series of complex behaviors that are usually initiated during arousals from slow-wave sleep (SWS) and culminate in walking around with an altered state of consciousness and impaired judgment.” Episodes generally develop from sudden but incomplete arousals from SWS and less often from stage 2 sleep [1–4]. No major differences in the percentage of sleep stages or in sleep stage distribution have been observed between sleepwalkers and age-matched controls. The main distinguishing findings are the increased number of awakenings and electroencephalographic (EEG) microarousals (MA), specifically occurring in the SWS of sleepwalkers even on nights without episodes [2,5,6]. Some studies also found decreased slow-wave activity (SWA) in sleepwalkers' first sleep cycles of the night [6,7] and a different time course of their δ activity decay across the night [2].

Despite the documented increase in sleepwalkers' nocturnal awakenings during SWS and their reduced SWA, there is a paucity of information on sleepwalkers' daytime functioning. Recently a large survey of the general population reported that somnambulism was associated with sleepiness [8]. Additionally a laboratory study of 10 adults who consulted a sleep clinic for chronic somnambulism revealed that they reported excessive daytime somnolence (EDS) [9], even after a night without sleepwalking episodes. In fact, the sleepwalkers had significantly lower mean sleep latencies on the Multiple Sleep Latency Test (MSLT) than matched control subjects. Moreover, 7 of the 10 sleepwalkers had a mean latency below 8 min, which is the accepted threshold for clinical somnolence [10].

Our study aimed to better document the presence of EDS in a larger sample of subjects consulting for sleepwalking and to use polysomnography to assess the contribution of nocturnal events, nocturnal sleep disruption, and poor sleep depth to sleepwalkers' EDS.

2. Methods

2.1. Subjects

Seventy sleepwalkers (40 women, 30 men; mean age, 33.1 ± 10.1 years; age range, 17–60 years) were included in our

* Corresponding author. Address: Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Cœur de Montréal, 5400 Boul. Gouin Ouest, Montréal, Québec H4J 1C5, Canada. Tel.: +1 514 338 2693; fax: +1 514 338 2531.

E-mail address: jy.montplaisir@umontreal.ca (J. Montplaisir).

study. All subjects were investigated for at least one night in the sleep laboratory and completed the Epworth Sleepiness Scale (ESS) [11]. None of the subjects had sleep-disordered breathing or concomitant neurologic or psychiatric disorders, and none of the subjects were taking psychotropic medications. Seventy control subjects (42 women, 28 men; mean age, 35.5 ± 14.0 years; age range, 20–56 years) without a sleep disorder only completed the ESS for comparisons purposes.

2.2. Data collection

The ESS is a self-administered scale in which the subject is asked to rate his or her chance of dozing off in each of the 8 daily life situations based on a 4-point scale (0–3). Thus the total score can range from 0 to 24. The cutoff point for pathologic EDS was established as a score of greater than 10 [12]. The body mass index (BMI) was obtained for every subject. Information regarding familial history of sleepwalking was obtained from the subjects during the clinical interview. A positive family history was defined as the presence of current or prior sleepwalking in at least one first-degree relative of the patient.

Polysomnographic (PSG) recordings were performed between 2007 and 2011 using a 32-channel Grass polygraph (sensitivity, $7 \mu\text{V}/\text{cm}$; bandpass, 0.3–100 Hz). Signals were relayed to a PC, digitized at a sampling rate of 256 Hz using commercial software (Harmonie, Stellate Systems, Montreal, Canada). PSG recordings and electrode placement were performed according to the international 10–20 system and included electrooculograms, submental electromyography, electrocardiogram, and EEG, which were recorded with a linked-ear reference (or a linked-mastoid reference). Surface electromyography of the bilateral anterior tibialis also was used to quantify periodic limb movements during sleep (PLMS) according to the standard method [13]. Respiration was monitored using an oronasal cannula and a thoracoabdominal plethysmograph. Oxygen saturation was recorded with a finger pulse oxymeter. None of the subjects had an index of respiratory events (number of apneas + hypopneas) greater than 15 per hour of sleep.

The scoring of the sleep stages was performed by trained technicians according to the standard method [13]. MA were scored on the central and occipital leads according to the standard criteria [13]. The MA index represents the number of MA per hour of sleep. In addition to these indices, the following sleep architecture variables were retained for analysis: sleep latency, sleep duration, sleep efficiency, total number of awakenings, percentage of time spent in each sleep stage, duration (in minutes) of SWS, percentage of SWS in each third of the sleep period, number of awakenings during SWS, all-night MA index, MA in SWS index, and number of somnambulistic episodes during the PSG investigation.

Finally, the SWA (0–4.5 Hz) during nonrapid eye movement (NREM) sleep and also during SWS in the first two sleep cycles was measured in two subgroups of sleepwalkers: 15 subjects with an ESS score below 7 (9 women, 6 men; mean age, 31.3 years) and 15 subjects with an ESS score greater than 12 (9 women, 6 men; mean age, 33.4 years). Five subjects in each subgroup had the linked-mastoid reference and 10 subjects in each group had the linked-ear reference. Spectral analyses were computed with a commercial software package (Stellate Systems, Montreal, Canada) on the C3 to A2, F3, and F4 leads using fast Fourier transform algorithms with cosine tapering on 4-s artifact-free sections, yielding a spectral resolution of 0.25 Hz.

2.3. Statistical analyses

To determine if the distribution of sleepwalkers' scores on the ESS was significantly different from that of nonsleepwalkers, *t* tests

were used to compare the group of 70 sleepwalkers to a group of 70 control subjects (distributions were normal). To fully explore the relationship between EDS and other variables, two statistical approaches were taken. First sleepwalkers were divided into two groups based on their ESS score. Based on established cutoff points, sleepwalkers with an ESS score between 0 and 10 were considered nonsomnolent and those with an ESS score ≥ 11 were classified into the somnolent group. The *t* tests were used to compare the two subgroups on all variables. The second approach consisted of using correlational analyses with Pearson product moment correlation coefficients to assess if a linear relationship existed between ESS scores and the other variables of interest. Finally Mann–Whitney *U* tests were used to compare the mean SWA of the two subgroups of 15 subjects. In all statistical analyses, the significance level was set at $P < .05$.

3. Results

The mean ESS score for the 70 sleepwalkers was significantly higher than that of controls (9.1 ± 4.5 vs 5.8 ± 3.4 ; $P = .000003$). Thus nearly half (45.7%) of the subjects who consulted a sleep disorders center for sleepwalking without concomitant depression reported EDS, as indexed by a score greater than 10 on the ESS. The distribution of ESS scores per group is shown in Fig. 1.

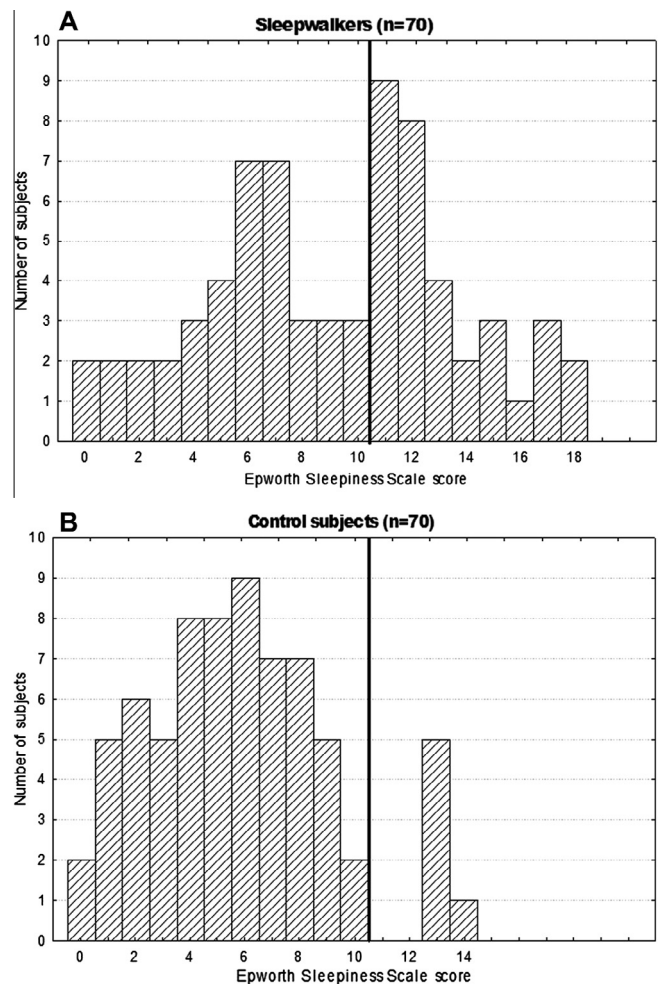


Fig. 1. Distribution of Epworth Sleepiness Scale scores in 70 adult sleepwalkers (A) and 70 adult controls (B). The bold vertical line represents the cutoff point for pathologic somnolence.

Table 1
Sleep profiles of nonsomnolent and somnolent sleepwalkers.

	Nonsomnolent sleepwalkers (n = 38)		Somnolent sleepwalkers (n = 32)		t test
	Mean	SD	Mean	SD	P value
Age at PSG, y	34.3	10.6	31.6	9.5	.27
Gender	21 W/17 M		19 W/13 M		.73
Positive family history	75.0% (24/32)		74.1% (20/27)		.93
ESS score	5.7	2.7	13.2	2.3	.000001
BMI	26.0	4.1	26.1	4.9	.86
Hypopnea index	0.8	1.4	0.6	1.2	.60
Apnea index	1.2	3.1	0.7	0.7	.30
AHI	2.0	3.9	1.3	1.7	.32
PLMS index	10.4	12.7	9.4	10.2	.74
PLMS-MA index	3.5	2.8	3.2	2.5	.68
MA	10.3	5.3	9.0	5.0	.31
Sleep latency	14.6	10.9	15.6	17.3	.77
Sleep duration	428.3	41.7	426.2	57.3	.86
Sleep efficiency	89.3	6.8	90.0	6.9	.67
Awakenings, n	27.0	10.7	25.7	10.0	.61
N1%	11.6	5.1	10.3	4.7	.26
N2%	54.2	7.5	52.0	7.9	.23
REM sleep%	18.8	4.2	20.0	4.9	.29
SWS%	15.4	9.0	17.8	8.7	.26
• First third%	8.9	5.3	10.0	4.6	.36
• Second third%	4.8	3.8	5.3	3.5	.54
• Third third%	1.7	2.5	2.5	3.4	.30
SWS duration (min)	66.1	38.5	74.6	34.0	.34
Awakenings from SWS, n	2.8	2.3	3.7	2.6	.17
MA index in SWS, n/h	3.5	3.0	3.1	2.4	.58
Episodes during PSG, n	1.2	1.7	1.3	1.9	.98

Abbreviations: PSG, polysomnography; y, years; SD, standard deviation; W, women; M, men; ESS, Epworth Sleepiness Scale; BMI, body mass index; AHI, apnea-hypopnea index; PLMS, periodic limb movements during sleep; PLMS-MA, periodic limb movements during sleep with microarousals; n, number; N1, stage 1 sleep; N2, stage 2 sleep; REM, rapid eye movement sleep; SWS, slow-wave sleep; min, minutes; n/h, number per hour.

3.1. Dichotomized ESS scores

In the first set of analyses, sleepwalkers were divided into two subgroups based on their score on the ESS: 38 subjects had a score ≤ 10 and 32 subjects had a score > 10 . Subgroups were similar in age and proportion of men and women (Table 1). The proportion of subjects with a positive family history also was similar in the two subgroups. The index of respiratory events was not significantly dif-

ferent in the two subgroups. Moreover, these values were well within reference range, and thus had little clinical significance. All of the other sleep parameters, including indices of sleep fragmentation (i.e., PLMS index, total number of awakenings, MA index, number of awakenings from SWS, MA in SWS, number of somnambulistic episodes during PSG), were similar in both subgroups. In addition, they were all within reference range (Table 1).

3.2. Correlations between ESS score and other variables

Sleepwalkers' scores on the ESS were not correlated with their BMI, PLMS index, or PLMS-MA index. Oddly ESS tended to be negatively correlated with the index of respiratory events ($n = 69$; $r = -0.23$; $P = .06$), indicating that the sleepier subjects had lower indices of respiratory events, mainly fewer apneas in our sample. Therefore, concomitant sleep disorders do not explain the EDS reported by sleepwalkers.

ESS was not significantly correlated with sleep duration, sleep efficiency, sleep latency, total number of awakenings, MA index, MA during SWS index, number of awakenings during SWS, total time spent awake, or stage 1 sleep percentage. ESS scores also were not related to the number of somnambulistic events recorded during the night. ESS scores were weakly negatively correlated with total percentage of stage 2 sleep ($r = -0.24$; $P = .045$). The scores tended to be positively correlated with total percentage of SWS ($r = 0.19$; $P = .12$) and with percentage of SWS in the last third of the night ($r = 0.23$; $P = .059$), indicating that the subjects who reported more daytime sleepiness actually tended to have a little more (not less) SWS.

3.3. SWA and ESS scores

There were no between-group differences in SWA during either of the first two NREM sleep cycles (or for the first two cycles pooled) derived from the C3-A2, F3, and F4 leads (Fig. 2). There also were no between-group differences for any cycle or lead when only the SWA in SWS of the first two cycles was considered.

4. Discussion

Our study revealed that a significant number of adult sleepwalkers reported EDS. This finding does not apply to childhood

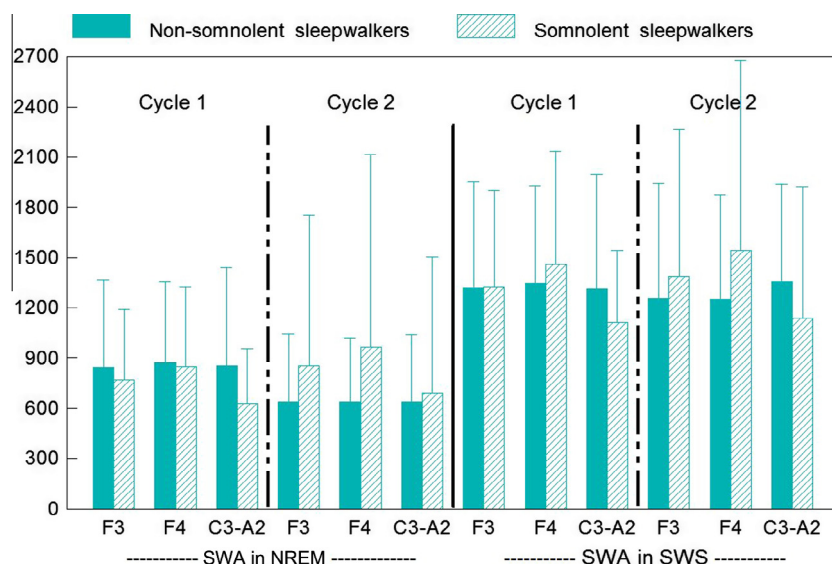


Fig. 2. Slow-wave activity (0–4.5 Hz) during nonrapid eye movement sleep for sleep cycles 1 and 2 and during slow-wave sleep only for cycles 1 and 2 in nonsomnolent and somnolent sleepwalkers.

somnambulism, which is much more prevalent but usually does not require any intervention, given its transitory and relatively harmless nature. This finding suggests that the widespread notion of adulthood somnambulism as a parasomnia without consequences on daytime functioning should be reconsidered. Moreover, the findings indicate the somnolence associated with somnambulism does not appear to be the direct result of sleep impairment. Indeed no significant correlations were found between ESS scores and any of the key sleep-related variables. On the contrary, there was evidence to suggest that sleepwalkers who reported the most severe EDS tended to have more rather than less SWS.

Several studies have reported that the arousals caused by sleep apneas can be a trigger for sleepwalking episodes [8,14,15]. The EDS noted in our study did not result from the presence of a sleep apnea syndrome, as none of the subjects had an apnea–hypopnea index (AHI) greater than 15 and only three subjects had an AHI between 10 and 15. If anything, the more somnolent sleepwalkers (ESS >10) tended to have a lower AHI than nonsomnolent sleepwalkers (ESS ≤10) and ESS tended to be negatively correlated with AHI. In fact, the three subjects with an AHI between 10 and 15 were all in the nonsomnolent group. However, these AHI values were not clinically significant, as they were well within the range of normal scores.

Taken together, these findings point to EDS as an intrinsic characteristic of sleepwalkers, which possibly is indicative of a general impairment in their daytime functioning. Findings from other studies also support this view of somnambulism. First we previously showed that subjects had EDS as measured objectively by the MSLT in a small group of sleepwalkers [9]. Second Oudiette et al. [16] reported pathologic ESS scores in nearly half of their 43 patients presenting with NREM sleep parasomnia. Third a single-photon emission computed tomography study performed during wakefulness in sleepwalkers and controls revealed hypoperfusions in the sleepwalkers' frontopolar cortex, superior and middle frontal gyri, superior and inferior temporal gyri, and angular gyrus [17]. Finally a transcranial magnetic stimulation study during wakefulness demonstrated hypoexcitability of some cortical GABAergic and cholinergic inhibitory circuits in sleepwalkers [18].

No data exist for possible neural substrates of EDS associated with adult somnambulism. However, it is now widely accepted that sleepwalking is strongly influenced by genetic factors. The between-subgroup difference in somnolence could be due to variability in patients' genetic profile. For example, it has been shown that certain polymorphisms in the adenosine deaminase gene, *ADA*, and in *PER3* in humans affect SWS duration and NREM sleep EEG (SWA) [19,20]. Interestingly, a certain polymorphism of *PER3* also affects the waking EEG. Individuals carrying the *PER3*^{5/5} allele not only have more slow waves in NREM sleep but also have more θ and α activity during wakefulness compared to individuals with the *PER3*^{4/4} allele. This finding could potentially account for the association between greater SWS and less alert wakefulness in a subgroup of sleepwalkers.

4.1. Clinical significance and study limitations

Our study documented the presence of EDS in subjects with sleepwalking, and nearly half of the 70 investigated subjects exceeded the clinical threshold of 10 on the ESS. Furthermore, sleepwalkers' EDS did not appear to be caused by sleep fragmentation, short sleep duration, or poor sleep quality or depth. We suggest that EDS is part of the phenotype associated with sleepwalking and that it should be considered in the clinical assessment and management of adult somnambulism. Although the ESS was developed over 20 years ago and continues to be widely used to assess EDS resulting from a variety of etiologies, further testing of daytime vigilance is needed to elucidate the functional significance

of the EDS experienced by sleepwalkers. Because measures of alertness and sleepiness often do not correlate with one another, additional and objective assessments, including MSLT, psychomotor vigilance task, event-related potentials, among others, should be considered to better delineate which specific aspects of alertness are most impaired in sleepwalkers.

Financial disclosures

This study is not industry sponsored.

Dr. Desautels received a research grant from GlaxoSmithKline. Dr. Dauvilliers has received funds for speaking and board engagements with UCB, Cephalon, Jazz, Novartis and Bioprojet. Dr. Montplaisir received research grants/support from Merck and GlaxoSmithKline, served as an advisor for Sanofi-Aventis, Servier, Merck, Jazz Pharmaceutical, Valeant Pharmaceutical, and Impax Laboratories, and received honoraria for speaking engagements from Valeant Pharmaceutical and Otsuka Pharmaceutical.

All of these disclosures represent financial activities outside the submitted work.

Dr. Zadra, Mr. Labelle, and Dr. Petit report no disclosures.

Author contributions

Dr. Desautels: data analysis, interpretation, and first draft of the manuscript.1.

Dr. Zadra: study concept and design, interpretation, and critical revision of the manuscript for important intellectual content.

Mr. Labelle: acquisition of data.

Dr. Dauvilliers: critical revision of the manuscript for important intellectual content.

Dr. Petit: acquisition of data, data analysis, and critical revision of the manuscript.

Dr. Montplaisir: study concept and design, critical revision of the manuscript for important intellectual content, and study supervision.

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: <http://dx.doi.org/10.1016/j.sleep.2013.04.029>.

References

- [1] Kavey NB, Whyte J, Resor Jr SR, Gidro-Frank S. Somnambulism in adults. *Neurology* 1990;40:749–52.
- [2] Espa F, Ondze B, Deglise P, Billiard M, Besset A. Sleep architecture, slow wave activity, and sleep spindles in adult patients with sleepwalking and sleep terrors. *Clin Neurophysiol* 2000;111:929–39.
- [3] Joncas S, Zadra A, Paquet J, Montplaisir J. The value of sleep deprivation as a diagnostic tool in adult sleepwalkers. *Neurology* 2002;58:936–40.
- [4] Pilon M, Montplaisir J, Zadra A. Precipitating factors of somnambulism: impact of sleep deprivation and forced arousals. *Neurology* 2008;70:2284–90.
- [5] Blatt I, Peled R, Gadoth N, Lavie P. The value of sleep recording in evaluating somnambulism in young adults. *Electroencephalogr Clin Neurophysiol* 1991;78:407–12.
- [6] Gaudreau H, Joncas S, Zadra A, Montplaisir J. Dynamics of slow-wave activity during the NREM sleep of sleepwalkers and control subjects. *Sleep* 2000;23:755–60.
- [7] Guillemainault C, Poyares D, Aftab FA, Palombini L. Sleep and wakefulness in somnambulism: a spectral analysis study. *J Psychosom Res* 2001;51:411–6.
- [8] Ohayon MM, Mahowald MW, Dauvilliers Y, Krystral AD, Leger D. Prevalence and comorbidity of nocturnal wandering in the U.S. adult general population. *Neurology* 2012;78:1583–9.
- [9] Montplaisir J, Petit D, Pilon M, Mongrain V, Zadra A. Does sleepwalking impair daytime vigilance? *J Clin Sleep Med* 2011;7:219.
- [10] van den Hoed J, Kraemer H, Guillemainault C, Zarcone Jr VP, Miles LE, Dement WC, et al. Disorders of excessive daytime somnolence: polygraphic and clinical data for 100 patients. *Sleep* 1981;4:23–37.

- [11] Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540–5.
- [12] Johns MW. Sensitivity and specificity of the multiple sleep latency test (MSLT), the maintenance of wakefulness test and the epworth sleepiness scale: failure of the MSLT as a gold standard. *J Sleep Res* 2000;9:5–11.
- [13] Iber C, Ancoli-Israel S, Chesson A, Quan SF, Medicine ftAAoS. *The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications*. 1st ed. Westchester: American Academy of Sleep Medicine; 2007.
- [14] Espa F, Dauvilliers Y, Ondze B, Billiard M, Besset A. Arousal reactions in sleepwalking and night terrors in adults: the role of respiratory events. *Sleep* 2002;25:871–5.
- [15] Guilleminault C, Kirisoglu C, Bao G, Arias V, Chan A, Li KK. Adult chronic sleepwalking and its treatment based on polysomnography. *Brain* 2005;128(pt. 5):1062–9 [published online ahead of print April 7, 2005].
- [16] Oudiette D, Leu S, Pottier M, Buzare MA, Brion A, Arnulf I. Dreamlike mentations during sleepwalking and sleep terrors in adults. *Sleep* 2009;32:1621–7.
- [17] Dang-Vu TT, Labelle MA, Petit D, Soucy JP, Zadra A, Montplaisir J. Functional neuroimaging of sleepwalking. *Sleep* 2012;35 (abstract supplement).
- [18] Oliviero A, Della Marca G, Tonali PA, et al. Functional involvement of cerebral cortex in adult sleepwalking. *J Neurol* 2007;254:1066–72.
- [19] Retey JV, Adam M, Honegger E, Khatami R, Luhmann UF, Jung HH, et al. A functional genetic variation of adenosine deaminase affects the duration and intensity of deep sleep in humans. *Proc Natl Acad Sci U S A* 2005;102:15676–81 [published online ahead of print October 12, 2005].
- [20] Viola AU, Archer SN, James LM, Groeger JA, Lo JC, Skene DJ, et al. PER3 polymorphism predicts sleep structure and waking performance. *Curr Biol* 2007;17:613–8 [published online ahead of print March 8, 2007].