Hypersynchronous Delta Waves and Somnambulism: Brain Topography and Effect of Sleep Deprivation

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Study Objectives: Hypersynchronous delta activity (HSD) is usually described as several continuous high-voltage delta waves (≥ 150 µV) in the sleep electroencephalogram of somnambulistic patients. However, studies have yielded varied and contradictory results. The goal of the present study was to evaluate HSD over different electroencephalographic derivations during the non-rapid eye movement (NREM) sleep of somnambulistic patients and controls during normal sleep and following 38 hours of sleep deprivation, as well as prior to sleepwalking episodes.
Design: N/A.
Setting: Sleep disorders clinic.
Patients: Ten adult sleepwalkers and 10 sex- and age-matched control subjects were investigated polysomnographically during a baseline night and following 38 hours of sleep deprivation.
Interventions: N/A.
Measurements and Results: During normal sleep, sleepwalkers had a significantly higher ratio of HSD over the time spent in stage 2, 3 and 4 on

frontal and central derivations when compared with controls. Sleep deprivation resulted in a significant increase in the ratio of the time in HSD over the time in stage 4 on the frontal lead in both groups and on the central lead in controls. There was no evidence for a temporal accumulation of HSD prior to the episodes.
Conclusions: HSD shows a clear frontocentral gradient across all subjects during both baseline and recovery sleep and has relatively low specificity for the diagnosis of NREM parasomnias. Increases in HSD after sleep deprivation may reflect an enhancement of the homeostatic process underlying sleep regulation.
Keywords: Sleepwalking, parasomnias, delta activity, hypersynchronous delta, electroencephalogram, sleep deprivation
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INTRODUCTION

SOMNAMBULISM (SLEEPWALKING) IS A PARASOMNIA CHARACTERIZED BY BEHAVIORAL MANIFESTATIONS OF VARIOUS DEGREES OF COMPLEXITY AND duration occurring during non-rapid eye movement (NREM) sleep.[1] Somnambulistic episodes generally arise from sudden but incomplete arousal from slow-wave sleep (SWS; stage 3 and 4 sleep) during the first third of the night[2-4] but may also occur out of stage 2 sleep or later during the night.[5-7] Somnambulism is considered to be a "disorder of arousal,"[4,8] as affected individuals appear to be caught between NREM sleep and full awakening.

One of the more controversial findings regarding the sleep electroencephalogram (EEG) of somnambulistic patients is the presence of hypersynchronous delta activity (HSD), usually described as continuous high-voltage (> 150-uV) delta waves occurring during SWS or immediately prior to an episode. This activity was first noted prior to sleepwalking events by Jacobson et al.[2] Subsequent studies in adult parasomniacs have yielded mixed results,

with sleepwalking or sleep terror episodes being occasionally,[5,9] often,[10] or always[11,12] associated with HSD. These inconsistencies may be due in part to the age and clinical history of the patients[13,14] and methodologic differences in the identification and quantification of HSD.[15-17] Schenck et al,[17] for example, found that most behavioral and nonbehavioral arousals from SWS in adult patients were not preceded by a delta-wave build-up and that only 15.5% were preceded by delta-wave clusters, but no comparisons were made with more-traditional definitions of HSD. More recently, 1 investigation found that the frequency of HSD in the EEG of adult sleepwalkers was dependent on the derivation investigated (presence of a frontocentral gradient).[15]

To help resolve this controversy and fill the void in knowledge concerning the specificity and sensitivity of HSD for somnambulism, the present study used the polysomnographic data collected by Joncas et al[7] to systematically evaluate HSD during the NREM sleep of somnambulistic patients and controls recorded during normal sleep and following 38 hours of sleep deprivation (a condition known to increase the frequency of somnambulistic episodes recorded in the laboratory). Specifically, differences in the occurrence of HSD were investigated over 5 different derivations (F3, C3, P3, T3, and O1) throughout participants' NREM sleep (stage 2, 3, 4) and immediately prior to somnambulistic episodes. HSD was assessed with several variables, including the most frequently described methods,[11,13] and with Schenck et al's[17] criteria for delta-wave build-up and delta-wave clusters.

It was hypothesized that sleepwalkers would have more HSD than control subjects and that HSD would increase during recovery sleep following a fronto-occipital gradient. Secondary goals were to investigate the temporal and topographic distribution of HSD prior to somnambulistic episodes and to assess HSD prior to somnambulistic behaviors as a function of episode complexity.

METHODS

Detailed information on the participants, sleep-deprivation protocol, and materials used has been published.[7] This information is thus presented succinctly.

Subjects

Ten adult sleepwalkers (3 men, 7 women, mean age: 25.1 years, SD: 4.1) and 10 sex and age-matched controls were investigated (mean age: 25.2 years, SD: 3.6). Sleepwalkers reported a minimal average of 2 episodes per month over the past 6 months with the episodes not being of a traumatic, neurologic, or pharmacologic origin. Exclusion criteria for all participants consisted of the following: (1) the presence of another sleep disorder and/or an index (number per hour of sleep) greater than 10 for respiratory events or periodic leg movements during sleep (PLMS); (2) the presence of a major psychiatric disorder; (3) the presence or history of a neurologic disorder; and (4) the use of drugs that could influence the sleep EEG. The protocol was accepted by the hospital's ethics committee.

Material

Polygraphic recordings were conducted on a 32-channel Grass polygraph (sensitivity at 7 μV, bandpass at 0.3-100 Hz; Grass Instruments, Quincy, Mass). Signals were relayed to a personal computer, digitized at a sampling rate of 128 Hz, and digitally filtered with an upper cutoff frequency of 64 Hz. Two sleepwalkers and 4 controls were recorded with a sampling rate of 256 Hz. The EEG recordings and electrode placement were performed according the 10-20 system (F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6) with a linked-ear reference and C3-A2. Electromyograms, electrooculograms, and electrocardiograms were also recorded. During the screening night, electromyography of the anterior tibialis was recorded for screening of PLMS. Respiration was monitored using an oronasal thermistor and a thoracic strain gauge. Oxygen saturation was recorded with a finger pulse oximeter, and snoring with a snoring microphone. Twenty-second epochs from the C3/A2 lead were used to visually score sleep stages according to established criteria.[18]

Procedure

Participants were recorded for 3 nights, including an initial screening night to ensure that they were free of any major sleep disorder. The second night served as a baseline recording. One week later, subjects returned to the laboratory for the 38-hour sleep-deprivation protocol and spent the night as well as the following day under constant supervision. Their sleep was recorded during their recovery night, and subjects were informed that they could sleep as long as they wanted. To control for any habituation effect to the recording procedure, 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.

Scoring of HSD

HSD was scored with a slight modification of the criteria outlined in previous studies.[11,13,19] Specifically, during NREM sleep, an HSD event was scored if it contained at least 5 seconds of continuous high-voltage ($\geq$ 150 μV) delta waves (1-3 Hz). The minimal interval needed without HSD to score 2 consecutive HSD events was at least 1 second. Although some studies of HSD have used a duration criteria of 10 seconds, we opted for a more liberal criteria of 5 seconds while taking into account HSD duration. Total time spent in HSD was tabulated separately for stage 2, stage 3, stage 4 and SWS (stage 3 and 4 sleep). Initial analyses revealed that the time spent in HSD was significantly greater during stage 4 sleep than during stage 2 and stage 3, but the 40 hours of sleep deprivation also significantly increased time spent in stage 4 (see Joncas et al).[7] Consequently, this measure of HSD did not allow us to determine if the observed increase in HSD during recovery sleep was simply a by product of increased stage 4 sleep or if it was related to individual differences in the duration of different sleep stages. To take these concerns into account, the ratio of total time spent in HSD over of the time spent in stage 2, stage 3, stage 4, and SWS was used as the measure of HSD during NREM sleep.

Scoring of HSD was performed independently on the scoring channel (C3/A2) as well as on the following leads: F3, P3, T3, and O1, each with a linked-ear reference. Since no HSD activity was found on the T3 lead, this derivation was excluded from further analyses. HSD was scored by a trained research assistant who was blind to the participants' group (sleepwalker or control) and condition (baseline or recovery sleep).

The temporal distribution of HSD was evaluated during the 5-minute window immediately preceding each somnambulistic episode arising from SWS. The time spent in HSD was tabulated separately for each minute in the 5-minute windows. Two additional variables were also scored and analyzed: (1) the number of HSD events occurring during the 10 seconds (HSD-10s) prior to somnambulistic episodes and (2) the number of HSD events occurring during the 30 seconds (HSD-30s) prior to the episodes. To ensure that the research assistant responsible for the scoring of HSD during NREM sleep remained blind to the participants' group and condition, the scoring of HSD prior to somnambulistic episode was performed by the first author (MP) after training by a certified polysomnography technologist.

Scoring of Delta-Wave Build-up and Delta-Wave Clusters

Delta-wave build-up and delta-wave clusters prior to somnambulistic episodes were scored according to the criteria established by Schenck et al.[17] Specifically, a delta-wave build-up was scored if the highest-amplitude delta wave occurred in the 10 seconds immediately preceding a somnambulistic episode, as compared with the 11- to 30-second prearousal period, whereas a delta-wave cluster was scored if 2 or more consecutive highest-amplitude delta waves occurred in the 10 seconds immediately preceding a somnambulistic episode, as compared with the 11- to 30-second period. Scoring was performed independently on the scoring channel (C3/A2) and on the following additional leads (F3, P3, O1, all with linked-ear reference) by the first author.

To assess whether the complexity of the somnambulistic episodes was differentially related to HSD, to delta-wave build-up, or to delta-wave clusters, each episode was scored on a 3-point scale: 1 for simple behaviors (e.g., playing with the bed sheets), 2 for complex behaviors (e.g., attempting to leave the bed or sitting up in bed), and 3 for actually leaving the bed (see Joncas et al[7] for details).

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Statistical Analyses

Statistical analyses were performed using a commercial software package (SPSS Software 12.0, SPSS Inc. Chicago, IL). Between-group comparisons for the ratio of total time spent in HSD over time spent in specific sleep stages (computed separately for stage 2, 3, 4 and SWS) were performed with a $2 \times 2 \times 4$ analysis of variance (ANOVA) with 1 independent factor (group) and 2 repeated measures (night and derivation). A $2 \times 2 \times 3$ ANOVA with 1 independent factor (group) and 2 repeated measures (night and sleep stage) was performed separately on the F3, C3, P3, and O1 leads to evaluate potential differences between sleep stages.

The temporal distribution of HSD before somnambulistic episodes was assessed with a 2×5 ANOVA with 2 repeated measures (night and 1-minute intervals) using each derivation separately for time spent in HSD during the 5-minute windows preceding the somnambulistic episodes. Since 1 patient did not have an episode during the study, this subject's data were excluded from this analysis. To evaluate the temporal distribution of HSD during the 5-minute windows prior to the episodes from SWS during recovery sleep as a function of episode complexity, the complexity of the episodes (simple versus more complex) was considered as a between-group factor. Accordingly, a 2×5 ANOVA with 1 independent factor (complexity) and 1 repeated measure (1-minute interval) was performed for time spent in HSD during the 5-minute windows prior to the somnambulistic episodes on each derivation.

A Greenhouse-Geisser correction for sphericity was applied to repeated ANOVAs when required, but the original degrees of freedom are reported. Contrast analyses were used to decompose interaction effects, and pairwise (posthoc) comparisons for main effects were performed with Bonferroni adjustments for multiple comparisons.

Some data points for ratio of total time in HSD over time in specific sleep stages were more than 3 standard deviations above the mean for 1 sleepwalker and 1 control. To reduce their impact on the distribution, these values were changed to 1 unit larger than the next most extreme score in the distribution, as described by Tabachenick and Fidell.[20]

χ^2 Analyses were used to compare the distribution of delta-wave build-up, delta-wave clusters, HSD-10s, and HSD-30s for somnambulistic episodes recorded during baseline sleep versus recovery sleep, between simple episodes and complex episodes for each derivation, and for differences across derivations. A Yate correction was applied for values less than 5.

RESULTS

Effects of Sleep Deprivation on Somnambulistic Episodes

The effects of the 38 hours of sleep deprivation on the polysomnographic data of sleepwalkers and control subjects, as well as on the frequency of somnambulistic episodes, have been previously reported.[7] The screening night indicated that all subjects were free of any major sleep disorder, revealed no signs of epileptic activity (such as spikes or spike and waves), and showed that sleepwalkers' sleep architecture was comparable with that of the controls.[7] In particular, there was (1) no indication of sleep-disordered breathing, as sleepwalkers had a mean apnea-hypopnoea index per hour of sleep of 0.3 (SD: 0.2) and controls an index of 0.3 (SD: 0.5) and (2) no indication of PLMS, as sleepwalkers had a mean PLMS index per hour of sleep of 2.0 (SD: 3.1) and controls an index of 2.6 (SD: 3.5). A total of 7 somnambulistic episodes (5 from SWS, 2 from stage 2) were recorded from 4 sleepwalkers during the baseline night, and 37 episodes (30 from SWS, 7 form stage 2) from 9 sleepwalkers were recorded during recovery sleep. None of the controls had any behavioral manifestations on either of the 2 nights. Sleep deprivation also increased the complexity of the somnambulistic episodes evaluated on a 3-point scale; whereas none of the baseline episodes were more complex than type 1, 8 of the behavioral manifestations from recovery sleep (all from SWS) were of type 2 or 3.

Ratio of Total Time in HSD Over Time in Specific Sleep Stages

Eight of the 10 control subjects and all 10 sleepwalkers had at least 1 HSD event at baseline, whereas 9 of the controls and all of the sleepwalkers had at least 1 HSD event during recovery sleep. Figure 1 presents the mean ratio ($\pm$ SEM) of the total time in HSD over the time spent in sleep stages 2, 3, 4, and SWS for each derivation (F3, C3, P3, O1) during the baseline and recovery sleep of sleepwalkers and controls.

A significant night $\times$ derivation $\times$ group interaction was found for the ratio of time in HSD over the time spent in stage 2 ($F_{3,54} = 3.61$, P = .019; see Figure 1-A), stage 3 ($F_{3,54} = 5.31$, P = .003; see Figure 1-B), stage 4 ($F_{3,54} = 4.64$, P = .032; see Figure 1-C), and SWS ($F_{3,54} = 5.35$, P = .016; see Figure 1-D). Contrast analyses revealed a significant main effect of group for the ratio of the time in HSD/stage 2 ($F_{1,18} = 4.59$, P = .046) and in HSD/stage 3 ($F_{1,18} = 4.47$, P = .049), reflecting that these ratios were greater in the group of sleepwalkers when compared with controls. The ratio of HSD/stage 4 and HSD/SWS was also significantly higher in sleepwalkers than controls but only during normal sleep on the F3 and C3 derivations (P values < .01). Contrast analyses also revealed that the ratio of time in HSD/stage 2 and in HSD/stage 3 was significantly lower during sleepwalkers' recovery sleep on F3 and C3 (P values < .05), while no significant differences were found for controls between the 2 nights on any derivation for these 2 ratios of HSD. The ratio of the time in HSD/stage 4 and HSD/SWS was higher during recovery sleep on F3 in both groups (P values < .01) and on C3 in controls (P values < .01).

Ratio of the Time HSD Over the Time Spent in Sleep Stages 2, 3, and 4 Per Derivation

There was a significant night $\times$ sleep stage interaction for the ratio of time in HSD over the time spent in sleep stages 2, 3, and 4 on the frontal ($F_{2,36} = 5.51$, P = .027) and central ($F_{2,36} = 5.87$, P = .016) derivations while a significant main effect of sleep stage was found on the parietal ($F_{2,36} = 20.12$, P < .0001) and occipital derivations ($F_{2,36} = 12.61$, P = .002). There was also a night $\times$ group interaction on the F3 ($F_{1,18} = 5.56$, P = .03) and C3 derivations ($F_{1,18} = 4.97$, P = .039). Contrast analyses of the night $\times$ sleep stage interaction on F3 and C3 showed that, on both nights, the ratio of HSD/stage 4 was greater than the ratio of HSD/stage 3 and of HSD/stage 2 (F3, P values < .001; C3, P values < .004), and that the ratio of HSD/stage 3 was in turn greater than HSD/stage 2 (F3, P < .002; C3, P < .017). However, sleep deprivation significantly increased only the ratio of HSD/stage 4 (F3, P < .001; C3, P < .01). Contrast analyses of the night $\times$ group interaction on F3 and C3 showed that the ratio of time in HSD over time in specific sleep stages (2, 3, and 4) was significantly higher in
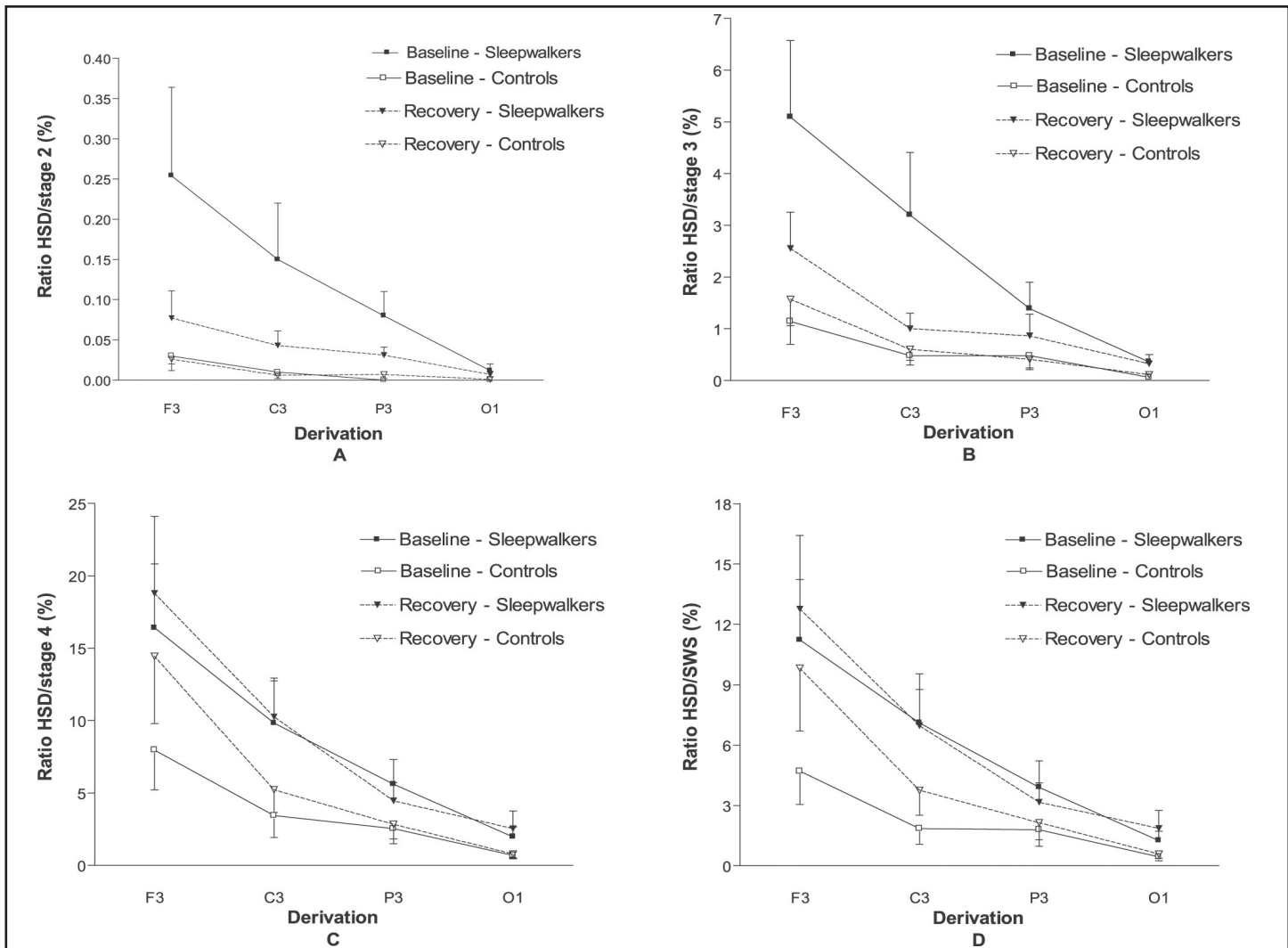
Figure 1—Mean ratio of the time in hypersynchronous delta activity (HSD) over the time spent in stage 2 (panel A), 3 (panel B), 4 (panel C) and slow-wave sleep (panel D) for the F3, C3, P3, and O1 derivations during baseline and recovery sleep for sleepwalkers and control subjects. Note: The scale of the ratio values presented on the Y axis of each panel varies in accordance to the magnitude of the results.

sleepwalkers during baseline sleep (P values < .01), but not during recovery sleep. Pairwise comparisons revealed that on the P3 and O1 derivations, the ratio of HSD/stage 4 was greater than the ratio of HSD/stage 3 and of HSD/stage 2 (P values < .007) and that HSD/stage 3 was greater HSD/stage 2 (P < .019). A very similar pattern of overall results was obtained when total time in HSD and the number of HSD events were investigated per sleep stage and topographically.

Evolution of HSD Prior to Somnambulistic Episodes

Figure 2 depicts the mean values (± SEM) for the evolution of the time spent in HSD during the first 5 minutes immediately prior to somnambulistic episodes recorded during baseline and recovery sleep across the F3, C3, P3, and O1 derivations. These episodes refer to the spectrum of behaviours that may or may not culminate in frank somnambulism in the sleep laboratory.

A significant main effect of night was found for the frontal ($F_{1,8}$ = 8.79, P = .02) and central derivations ($F_{1,8}$ = 9.43, P = .02) for the time spent in HSD. No significant differences were found per minute of sleep for any derivation and there was no significant night × minute interaction for the time in HSD (P values > .05).

Figure 3 presents the mean values (± SEM) for the evolution of the time spent in HSD as a function of complexity of the somnambulistic episodes arising during recovery SWS. An analysis of variance performed on each derivation revealed no significant differences per minute of sleep, per episode as a function of complexity, and no significant complexity × minute interaction (P values > .05).

HSD-10s and HSD-30s Prior to Somnambulistic Episodes

Table 1 presents the number and proportion of somnambulistic episodes arising from SWS that were preceded by HSD-10s or HSD-30s during baseline and recovery sleep for the F3, C3, P3, and O1 derivations and as a function of episode complexity. There was no significant difference in the proportion of episodes preceded by HSD-10s and HSD-30s during either night on any derivation (P > .05). HSD-10s and HSD-30s (both nights combined) were found in a significantly greater proportion on F3 than on other leads (HSD-10s, P < .00001; HSD-30s, P < .00001). HSD-30s were also found in a significantly greater proportion on C3 than on P3 and O1 (P = .002). During recovery sleep, we also observed an HSD event that lasted 10 seconds after the onset of
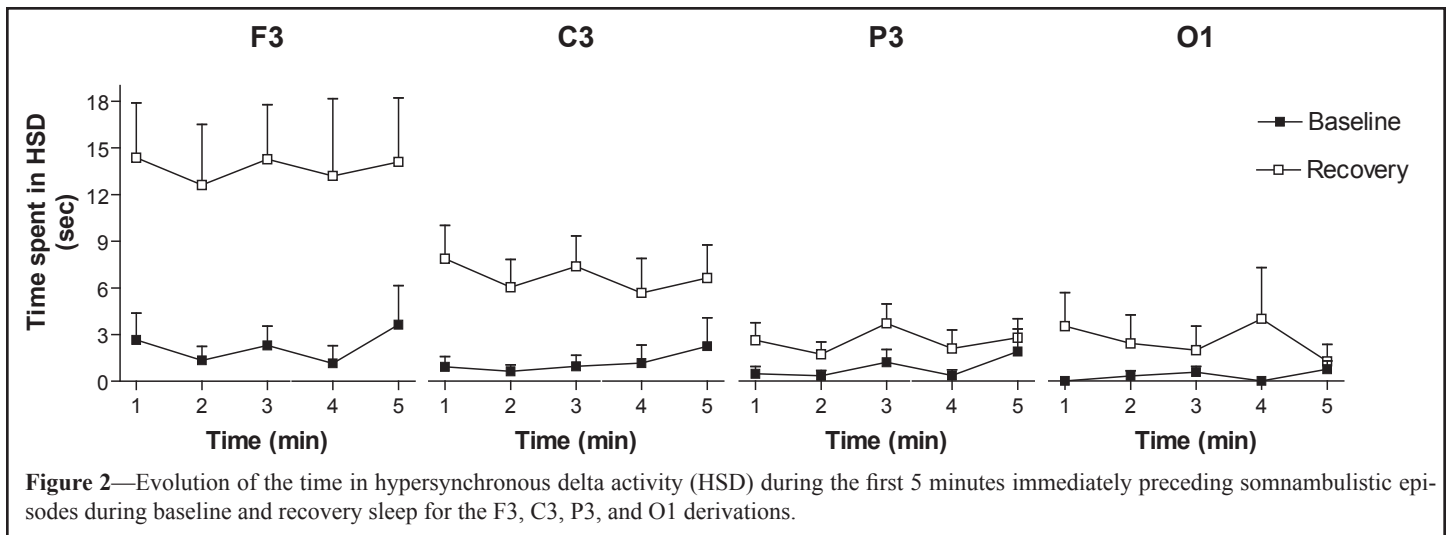
Figure 2—Evolution of the time in hypersynchronous delta activity (HSD) during the first 5 minutes immediately preceding somnambulistic episodes during baseline and recovery sleep for the F3, C3, P3, and O1 derivations.
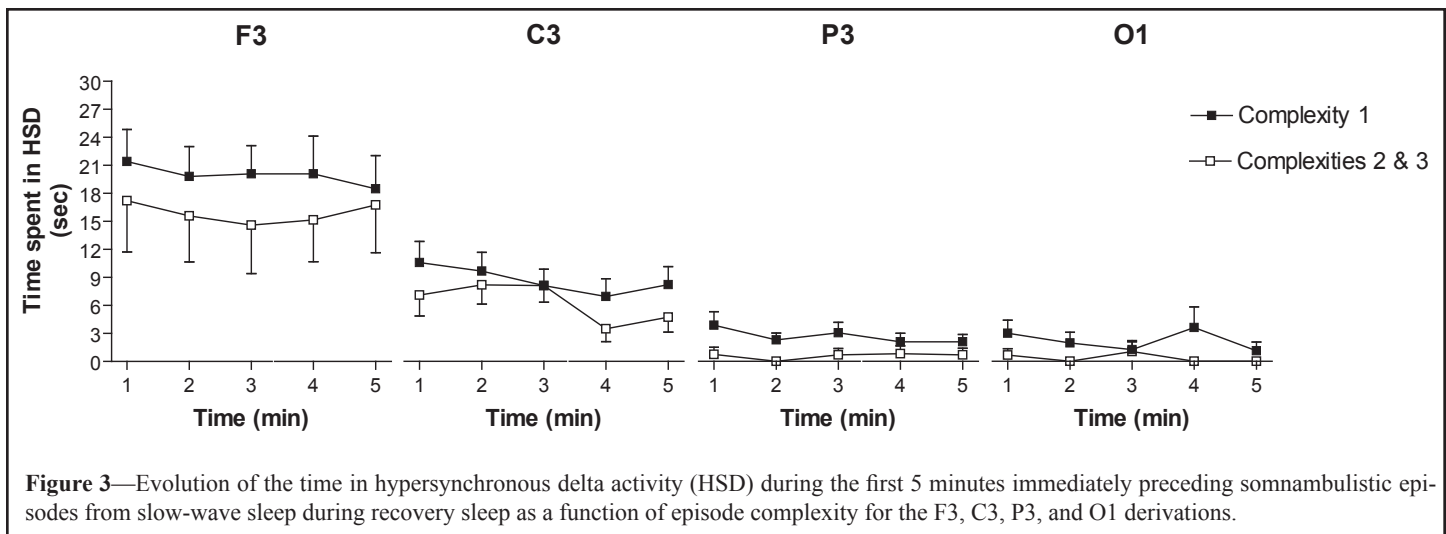

Figure 3—Evolution of the time in hypersynchronous delta activity (HSD) during the first 5 minutes immediately preceding somnambulistic episodes from slow-wave sleep during recovery sleep as a function of episode complexity for the F3, C3, P3, and O1 derivations.

1 sleepwalking episode, but HSD was not observed during any of the other postarousal EEG recordings.[21]

There was no significant difference in the proportion of episodes preceded by HSD-10s or HSD-30s during simple episodes (type 1) as compared with relatively more complex ones (type 2 and 3) (P > .05).

Delta-Wave Build-up and Delta-Wave Clusters Prior to Somnambulistic Episodes

The number and the percentage of somnambulistic episodes preceded by delta-wave build-up or by a delta-wave cluster during baseline and recovery sleep on the F3, C3, P3, and O1 derivations as a function of episode complexity are presented in Table 2. There was no significant difference in the proportion of episodes preceded by delta-wave build-up or by delta-wave clusters during baseline and recovery sleep for any of the derivations (P values > .05). Delta-wave build-up (both nights combined) occurred significantly more frequently on F3 than on other derivations (P = .01), but no significant differences were found between derivations for delta-wave clusters.

There was no significant difference in the proportion of episodes preceded by delta-wave build-up or delta-wave clusters during simple episodes (type 1), as compared with relatively more complex ones (types 2 and 3) (P values > .05).

DISCUSSION

HSD During NREM Sleep

The main goal of this study was to assess the topography of HSD during the NREM sleep of adult sleepwalkers and control subjects during baseline and recovery sleep. As hypothesized, sleepwalkers had a significantly higher ratio of HSD/stage 2 and HSD/stage 3 on both study nights. One unexpected result was that sleepwalkers' ratio of HSD/stage 4 and HSD/SWS was significantly higher only during baseline recordings on frontal and central derivations, due principally to the presence of HSD during NREM sleep of adult subjects who were free of any major sleep disorder. That HSD was found in 8 of the 10 controls during baseline recording and in 9 of the 10 after sleep deprivation contrasts with previous reports indicating that high-amplitude delta activity is a normal occurrence in the sleep of children under the age of 9[22,23] but extremely rare[13,19] or over absent[11] in adults.

Our finding that HSD occurs frequently in the sleep EEG of adult somnambulistic patients during NREM sleep that is unaccompanied by sleepwalking episodes has been previously reported.[13, 24,25] However, our results from both study nights refine these observations by showing that HSD is significantly more prevalent during stage 4 sleep, as compared with stage 2 and 3, and that sleep deprivation results in a significant increase in the ratio of time in HSD/stage 4 sleep on the frontal derivation for sleepwalkers and on the frontal and central ones for control subjects. The

Night	HSD-10s, no. (%)				HSD-30s, no. (%)			
	F3	C3	P3	O1	F3	C3	P3	O1
Baseline night (n=5)	2	0	0	0	2	1	1	0
Complexity 1	(40)	(0)	(0)	(0)	(40)	(20)	(20)	(0)
Recovery night (n=30)	13	3	2	1	26	16	5	5
Complexities 1, 2 & 3	(43.3)	(10)	(6.7)	(3.3)	(86.7)	(53.3)	(16.7)	(16.7)
Both nights combined (n=35)	15	3	2	1	28	17	6	5
Complexities 1, 2 & 3	(42.9)	(8.6)	(5.7)	(2.9)	(80)	(48.6)	(17.1)	(14.3)
Recovery night Complexity 1 (n=22)	10	2	2	1	19	11	5	4
	(45.5)	(9.1)	(9.1)	(4.5)	(86.4)	(50)	(22.7)	(18.2)
Both nights combined Complexity 1 (n=27)	12	2	2	1	21	12	6	4
	(44.4)	(7.4)	(7.4)	(3.7)	(77.8)	(44.4)	(22.2)	(14.8)
Recovery night Complexities 2 & 3 (n=8)	3	1	0	0	7	5	0	1
	(37.5)	(12.5)	(0)	(0)	(87.5)	(62.5)	(0)	(12.5)

Table 1—Somnambulistic Episodes from Slow-Wave Sleep Proceeded by HSD-10s or HSD-30s as a Function of Episode Complexity

HSD-10s refers to the hypersynchronous delta activity (HSD) events occurring during the 10 seconds prior to somnambulistic episodes; HSD-30s refers to the HSD events occurring during the 30 seconds prior to somnambulistic episodes.

Night	Delta-wave build-up, no. (%)				Delta-wave cluster, no. (%)			
	F3	C3	P3	O1	F3	C3	P3	O1
Baseline (n=7)	6	4	3	3	1	1	1	1
Complexity 1	(85.7)	(57.4)	(42.9)	(42.9)	(14.3)	(14.3)	(14.3)	(14.3)
Recovery sleep (n=37)	27	23	18	15	7	4	1	1
Complexities 1, 2 & 3	(73)	(62.2)	(48.6)	(40.5)	(18.9)	(10.8)	(2.7)	(2.7)
Both nights combined (n=44)	33	27	21	18	8	5	2	2
Complexities 1, 2 & 3	(75)	(61.4)	(47.7)	(40.9)	(18.2)	(11.4)	(4.5)	(4.5)
Recovery sleep Complexity 1 (n=29)	21	18	17	10	5	3	1	1
	(72.4)	(62.1)	(58.6)	(34.5)	(17.2)	(10.3)	(3.4)	(3.4)
Both nights combined Complexity 1 (n=36)	27	22	20	13	6	4	2	2
	(75)	(61.1)	(55.6)	(36.1)	(16.7)	(11.1)	(5.6)	(5.6)
Recovery sleep Complexities 2 & 3 (n=8)	6	5	1	5	2	1	0	0
	(75)	(62.5)	(12.5)	(62.5)	(25)	(12.5)	(0)	(0)

Table 2—Somnambulistic Episodes Preceded by Delta-Wave Build-up or by Delta-Wave Clusters as a Function of Episode Complexity

HSD-10s refers to the hypersynchronous delta activity (HSD) events occurring during the 10 seconds prior to somnambulistic episodes; HSD-30s refers to the HSD events occurring during the 30 seconds prior to somnambulistic episodes.

significant effect of sleep deprivation on the ratio of HSD/stage 4 is more pronounced in control subjects and possibly suggests the presence of a ceiling effect in sleepwalkers.

Although sleepwalkers have been described as having a greater number of arousals during SWS preceded by EEG slow-wave synchronization,[6,11,19,26] our results show that HSD also occurs frequently in the NREM sleep of sleepwalkers and controls without the presence of arousal reactions. Furthermore, despite the significant increase in HSD during stage 4 after sleep deprivation, there was no significant difference in the number of awakenings from SWS between the 2 nights in both groups.[7] The data further demonstrate that HSD can be observed during stage 2 sleep.

HSD events may be viewed by some in relation to the cyclic alternating pattern which expresses the organized complexity of arousal-related phasic events in NREM sleep.[27,28] Specifically, 1 cyclic alternating pattern subtype (A1) could overlap with HSD, as it is characterized by synchronized EEG patterns (e.g., delta bursts), by low delta power and a frontal predominance.[29] In addition, when compared with controls, adult sleepwalkers show an increased cyclic alternating pattern rate, and in the A1 subtype in particular.[6] However, these 2 types of EEG activity are distributed differently during sleep and show differential reactions to sleep deprivation. While the number of A1 events remain unchanged across the first 3 sleep cycles[30] and after 40 hour of sleep deprivation,[31] supplemental analyses of our data (not reported in the results section) revealed that all HSD ratios occurred significantly more frequently during the first third of the night than during subsequent sections, and during the second third compared with the last third for both sleep periods. Our data also showed that, unlike A1 events, HSD increases during stage 4 after sleep deprivation.

The functional significance of HSD remains unclear. Early studies suggested that HSD in sleepwalkers was related to central nervous system immaturity.[2,3,25] The aforementioned results speak against this hypothesis. The data suggest rather that HSD is related to the expression of the homeostatic process underlying sleep regulation. According to the widely recognized 2-process model of sleep regulation, the sleep-wake cycle is regulated by the interaction of 2 processes: a circadian process and a homeostatic process.[32,33] The intensity and dynamic of SWS and slow-wave activity (SWA) (spectral power in the 0.75- to 4.5-Hz band) are considered to represent the expression of the homeostatic process and are typically present in greater amount at the beginning of the night and decline exponentially as the night progresses.[34,35] Furthermore, data indicate that the increase in SWA is primarily due to an increased amplitude of delta waves[36] and that this amplitude is greater at the beginning of the night and decreases as sleep progresses.[37] These findings suggest that higher-amplitude delta waves are associated with the higher homeostatic pressure present early during the sleep period.

The increased number of arousals during SWS in sleepwalkers in comparison with that of controls[6,7,11-13,26,38] and the presence of somnambulistic episode per se results in significant sleep fragmentation and interferes with the normal build-up of SWA.[11,12,38] As has been suggested by others,[11,39] this sleep fragmentation and resulting sleep deprivation increases the pressure for sleep and for SWS in particular. We hypothesize that the higher sleep pressure from sleepwalkers' homeostatic process results in the generation of delta waves with increased amplitude that appear during stage 2 sleep and reach maximal intensity during stage 4. The significant increase in HSD during stage 4 observed after sleep depriva-

tion may thus result from both sleep pressure and increased sleep fragmentation (i.e., increased frequency of behavioral episodes) in sleepwalkers and from sleep deprivation in controls. All HSD ratio variables occurred significantly more frequently during the first third of the night than during subsequent sections and during the second third compared with the last third for both sleep periods. This demonstrates that HSD occurs more frequently at the beginning of the sleep period and that HSD shows an overnight decline as, has been previously demonstrated for SWA.[34,35]

This hypothesis is also consistent with the finding of HSD and high-amplitude delta waves during the SWS of adults with sleep-disordered breathing, another sleep-disordered population characterized by considerable sleep fragmentation and sleep deprivation.[14,40] Taken together, these results provide strong evidence that the sleep EEG of adult sleepwalkers contains an unusually high proportion of HSD during SWS but that HSD also occurs in the SWS of normal controls and of patients with sleep-related respiratory disorders.

The topographic data for all HSD-related variables revealed a robust frontocentral gradient in the expression of these forms of delta activity. These findings are in accordance with our hypothesis given the frontal predominance in EEG power for SWA during normal sleep[41,42] and the fact that the rebound in SWA induced by sleep deprivation (i.e., sleep pressure) is more pronounced over the frontal regions of the brain.[43-45] Delta-wave counts and delta amplitude have also been found to be greatest in frontal regions.[46]

Our topographic results also have important methodologic implications, since montages and leads used to assess HSD have greatly varied across studies. When scored simultaneously on more than 1 derivation, HSD will be found in varying amounts depending on the topographic location of the leads selected. Moreover, the derivation used to score HSD is not always clearly identified, and the duration and amplitude components of HSD are not always specified by researchers. These methodologic shortcomings in the study of HSD add to those highlighted by Schenck et al.[17]

HSD Prior to Somnambulistic Episodes

A secondary aim of this study was to investigate the temporal and topographic distribution of HSD prior to somnambulistic episodes. There was no evidence for a temporal accumulation of HSD during 5-minute windows immediately preceding the episodes recorded at baseline and no differential effect between simple episodes and more complex ones. These findings are consistent with those of Schenck et al,[17] who found that most behavioral and nonbehavioral arousals from SWS in adults with sleepwalking or sleep terrors were not preceded by delta-wave build-up, by electromyogram activation, or by an acceleration of the electrocardiogram. Sleep deprivation, however, significantly increased the time spent in HSD on the frontal and central derivations during the 5-minute windows prior to the episodes. In accordance with our hypothesis, this could reflect augmented sleep pressure following 40 hours of sleep deprivation.

None of the somnambulistic episodes from SWS were preceded by an HSD event on C3 in the 10 seconds prior to their onset (HSD-10s), and only 20% were preceded by an HSD event in the 30 seconds (HSD-30s). Even though we used a more liberal criteria for HSD and a longer period prior to clinical events (i.e., 30 seconds), these results are in opposition to reports that all epi-

sodes of arousal disorders are preceded by HSD[11,12] but are consistent with the finding that somnambulism is only occasionally associated with HSD.[5,9,13] Sleep deprivation resulted in a small, but not statistically significant, increase in the proportion of episodes preceded by HSD-10s and HSDA-30s on most derivations, and no significant differences were found between simple and relatively more-complex episodes.

Using Schenck et al's[17] operationalizations for EEG delta-wave build-up and delta-wave clusters, approximately 60% of the somnambulistic episodes recorded on C3 were preceded by delta-wave build-up, whereas less than 15% showed evidence of delta-wave clusters. These figures are comparable with those reported by Schenck et al.[17] Sleep deprivation did not influence the proportion of episodes preceded by delta-wave build-up or clusters on any derivation. Furthermore, these variables did not discriminate between simple behavioral events (type 1) and more-complex ones (types 2 and 3). These findings are consistent with those of Schenck et al,[17] who found no significant differences in delta-wave build-up or delta clusters between arousals with or without vigorous behaviors. Finally, our topographic analyses revealed frontal predominance for delta-wave build-up but not for delta clusters.

CONCLUSION

To our knowledge, this is the first study to have systematically investigated the occurrence and topographic distribution of HSD with an array of measures both during normal sleep and following sleep deprivation. The most salient findings are: (1) when compared with the sleep EEG in control subjects, HSD occurs more frequently during sleepwalkers' sleep EEG; (2) sleep deprivation leads to increased HSD during stage 4 sleep in both groups; (3) HSD recorded in patients and controls shows a clear frontocentral gradient during both baseline and recovery sleep; and (4) there is no evidence that somnambulistic episodes are immediately preceded by a build-up in HSD or by any HSD-related variables. These findings reinforce the results from previous studies[14,17] in demonstrating that regardless of how it is measured, HSD has low specificity for the diagnosis of NREM parasomnias. We suggest that HSD may be related to the expression of the homeostatic process underlying sleep regulation. If so, then the occurrence of HSD would be expected to be preferentially related to factors that deepen sleep, such as young age, hyperthyroidism, fever, sleep fragmentation, or sleep deprivation. Further studies are required to investigate the exact neurophysiologic mechanisms underlying HSD and its relationship to SWA.

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