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# Topographical EEG Mapping in a Case of Recurrent Sleep Terrors

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*Sleep terrors are characterized by marked CNS arousal and typically occur during stage 3-4 sleep within the first NREM cycle. Studies of the EEG during sleep terrors suggest that delta power and synchrony in the EEG may be important physiological markers of sleep terror presence and intensity. An EEG mapping study was undertaken with a single participant who experienced three sleep terror episodes in the laboratory. A one-minute section of EEG was sampled immediately prior to the onset of each of the three sleep terrors. Similar EEG sections were taken from 10 healthy sex- and age-matched controls. The sleep terrors and control (normative) data were then compared topographically with z-scores (z-mapping). The z-maps indicated that all three sleep terrors contained more total and delta power in central and frontal areas than the control EEG sections. Moreover, relative delta power in these areas for the three sleep terrors was proportional to the subjective intensity of the episode. Although this pre-arousal EEG pattern may be related to ongoing slow-wave sleep mentation that may sometimes trigger sleep terror episodes, its functional significance remains an open question. The results demonstrate the utility of EEG mapping for the quantification of brain activation during sleep terror attacks and suggest that discrete activity profiles are identifiable for different types of dreaming-related arousal.*

**KEY WORDS:** EEG mapping; arousal; sleep terrors; parasomnias.

## INTRODUCTION

Many types of unusual sleep states accompanied by sleep mentation (e.g., lucid dreaming, anxiety dreams, sleep terrors) are characterized by increased CNS arousal (Nielsen & Zadra, 1997; Zadra & Nielsen, 1995). These unusual states are typically accompanied by strong emotions and eventual awakening. EEG mapping may be appropriate for quantifying gross changes in cortical activation during such sleep

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states. The present research focuses on the topographical EEG mapping of sleep terrors.

Sleep terrors, sometimes called "pavor nocturnus" in children and "incubus attacks" in adults, are characterized by a loud piercing scream or cry for help, intense autonomic activation (e.g., heart rates doubling or nearly tripling), inconsolability, and overwhelming anxiety or acute panic. In many episodes, there is precipitating dream imagery that can range from a brief frightening thought or image to more elaborate dreamlike mentation (Fisher, Kahn, Edwards, Davis, & Fine, 1974; Kahn, Fisher, & Edwards, 1991; Schenck, Milner, Hurwitz, Bundlie, & Mahowald, 1989). Although some of this mental content can be related to "post-arousal" events (e.g., fear of dying associated with autonomic activation), there exist numerous examples of imagery occurring during "pre-arousal" events (e.g., Fisher, Kahn, Edwards, Davis, & Fine, 1974). Following arousal, the research participant is disoriented, confused, unresponsive, and sometimes delusional; full consciousness is usually not attained for several minutes after the onset of the attack. The participant is partially to totally amnesic for the event upon regular awakening in the morning.

Several studies have shown that sleep terrors are most likely to occur during stage 3-4 sleep within the first NREM cycle (e.g., Broughton, 1968; Gastaut & Broughton, 1965; Fisher, Kahn, Edwards, & Davis, 1974; Schenck, et al., 1989). This is when EEG delta activity is both most frequent and most continuous. Of the many findings reported in Fisher's early studies (see Fisher, Kahn, Edwards, & Davis, 1973a; 1973b; 1974; Fisher, Kahn, Edwards, Davis & Fine, 1974), one of the most salient is that the severity of the sleep terror, as assessed by heart rate increase and maximum heart rate after arousal, is proportional to the duration of the preceding stage 3-4 sleep episode. This is true both for spontaneously occurring sleep terrors and for sleep terrors induced precipitously by sounding a loud buzzer. The presence of EEG slow wave synchronization (i.e., continuous high voltage delta waves) during NREM sleep also occurs more frequently in sleep terror participants than in controls (Fisher, Kahn, Edwards, & Davis, 1974; Halász, Ujszászi, & Gádoros, 1985). Finally, EEG activity during the sleep terror typically shows a regular delta pattern that gives way to theta and alpha activity (Keefauver & Guilleminault, 1994). These EEG findings together suggest that delta power and synchrony in the EEG may be important physiological markers of sleep terror presence and intensity.

Since very little is known about the characteristics of the EEG that precede sleep terror episodes, a pilot EEG mapping study was undertaken with a single participant who experienced repeated sleep terror episodes in the laboratory. The goal was to pilot test EEG mapping procedures for their sensitivity in detecting elevations in delta power. It was also thought that cortical regions where sleep terror related changes in delta power are maximal might be revealed with this approach, providing clues as to the pathophysiology of this disorder.

## METHODS

A 32-year-old female participant (M) who demonstrated recurrent sleep terrors in the sleep laboratory was studied and compared with 10 healthy sex- and age-

matched controls with no history of parasomnias. M slept three consecutive nights in the sleep laboratory and controls two consecutive nights. All participants were in good health with no prior or current use of medication.

Participants were monitored for eye movements, heart rate, and respiration rate. In addition, EEG was recorded at 128 Hz from a 19-channel referential electrode montage from the 10-20 system (Jasper, 1958): Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, O1, O2, T3, T4, T5, T6, Fz, Cz, and Pz, with a common reference.

## EEG Z-Mapping

M experienced a sleep terror during slow wave (Stage 3-4) sleep within the first NREM cycle on each of her three nights in the laboratory. A one-minute artifact-free section of EEG (i.e., 15 sections of 4 seconds each) was sampled immediately prior to the onset of each of the three sleep terrors, subjected to amplitude spectral analysis (FFT), and filtered with a Hanning window. EEG power was calculated for total power (0.75-31.00 Hz) and for each of five component frequency bands: delta (0.75-3.75 Hz), theta (4.00-7.75 Hz), alpha (8.00-12.75 Hz), beta1 (13.00-20.25 Hz), and beta2 (20.50-31.00 Hz).

Similar artifact-free sections were drawn from each control participant's stage 3-4 sleep during the first NREM cycle. Five control sections came from night 1 and five from night 2. The five control sections from each of the two nights were matched for the total amount of time M spent in stage 3-4 sleep, as well as for the amount of continuous stage 4 sleep prior to the onset of the sleep terror on nights 1 and 2, respectively. These 10 control sections were averaged. The sleep terrors and control (normative) data were then compared topographically using z-scores (z-mapping). The data were represented visually by interpolating the z-scores for each of the 19 electrodes and displaying these scores as 2D gray scale maps.

## Subjective and Objective Intensity Ratings

Following each sleep terror, M was asked to rate the intensity of the episode on a 9-point scale and then was questioned about the presence of any mentation preceding or during the terror. In addition, changes in heart rate were calculated by comparing the participant's baseline heart rate (mean rate for the 2 minutes before arousal) to the highest heart rate within any 15-second period during the first 75 seconds of the post-arousal period. Changes in respiration rates were calculated in a similar fashion.

# RESULTS

M experienced three sleep terrors, one on each of three consecutive nights. The three episodes occurred during slow-wave sleep within the first NREM sleep cycle. The time spent in stage 3-4 sleep prior to the onset of the sleep terrors was 46.1 minutes on night 1, 60.9 minutes on night 2, and 50.3 minutes on night 3. The

amount of continuous stage 4 sleep that preceded each episode was 3.5 minutes on night 1, 8.2 minutes on night 2, and 2.8 minutes on night 3. All three episodes began with a sudden yell and continued with gross motor activity, including sitting up in bed and pointing at a wall (night 2) and sitting up while placing her hand in her mouth and trying to spit (night 3). Mentation was clearly recalled following two of the three episodes (nights 2 and 3). Following the terror on night 2, M reported that there had been a man sitting in a chair beside her bed and that he had wanted to harm her. Subsequent to the sleep terror on night 3, M reported that her mouth had been filled with large insects which she was quickly trying to grab with her hands and spit out—but without success. M had only vague recall following her first sleep terror about choking on something. Vocalizations (i.e., moaning) were noted prior to the onset of her third sleep terror.

M provided subjective ratings of the intensity of each sleep terror and reported that the episodes had been much less intense than were her typical sleep terrors at home. Pre- to post-arousal changes in heart and respiration rate, as well as the participant's rating of the intensity of each sleep terror are presented in Table 1.

It is evident that the sleep terror from night 2 received the highest rating, followed by nights 3 and 1 which were rated as being quite similar to one another. It is of interest to note that the participant's ratings of the sleep terrors parallel the changes in heart rate from pre- to post-arousal periods. Furthermore, consistent with the findings reported by Fisher et al. (1973a), the intensity of the sleep terror was proportional to the total amount of stage 3-4 sleep and of continuous stage 4 sleep preceding each episode.

Spectral analyses were performed on the 60-second EEG segment preceding each of the three sleep terrors and compared with the average of the spectral maps from the control group. The spectral analyses showed that all three sleep terrors contained more total power than the control EEG segments. The largest differences occurred in central and frontal areas. Spectral analyses of the component frequency bands revealed that these differences were largely due to elevated delta power. The elevated delta power is illustrated in Figure 1, which presents the spectral maps of the most and the least intense of the three episodes as well as the averaged map for the controls. As shown in the figure, the most intense sleep terror contains more delta power than the least intense sleep terror and both show greater delta power than the control map.

**Table 1.** Heart Rate and Respiratory Changes Before and After Arousal and Subject's Rating for Each Sleep Terror

| Night | Baseline HR | ST* HR | HR Change | Baseline RR | ST* RR | RR Change | Subject's Rating |
|---|---|---|---|---|---|---|---|
| Night 1 | 69 | 90 | 21 | 16 | 20 | 4 | 4 |
| Night 2 | 72 | 102 | 30 | 15 | 20 | 5 | 8 |
| Night 3 | 63 | 88 | 25 | 16 | 20 | 4 | 5 |

Note: *ST = Sleep Terror.

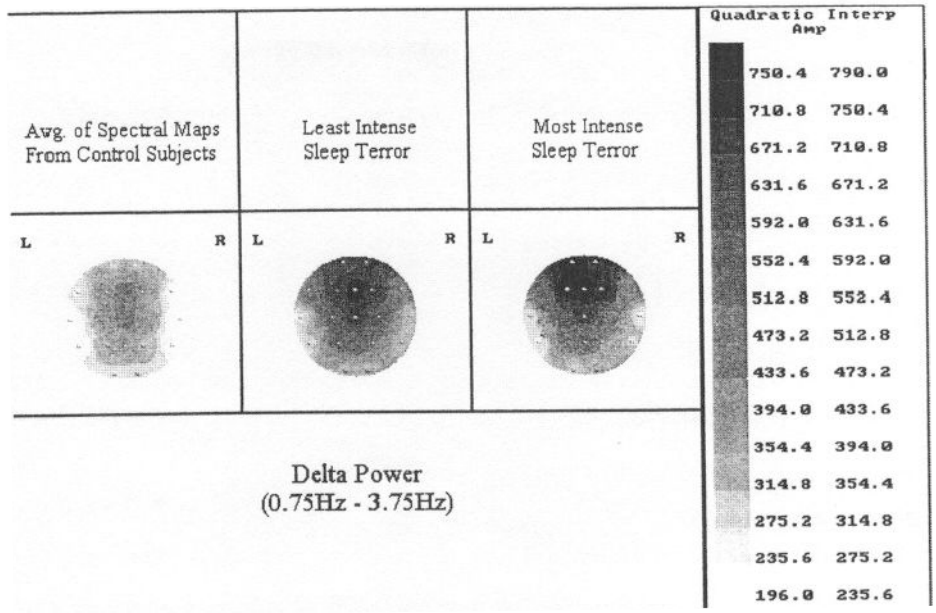


**Fig. 1.** Spectral maps of the most and the least intense sleep terrors and an averaged control group.

To better quantify the magnitude of the observed differences in the spectral analyses, the sleep terrors and control (normative) data were compared topographically with z-mapping. Figure 2 presents the z-scores for delta power of each of the three sleep terrors when compared to the averaged control data. These maps indicate that all three sleep terrors contained significantly ($z > 1.96$, $p < .05$) more delta power in central and frontal areas than the control EEG sections and that these differences are of even greater magnitude over the left than the right frontal regions. Moreover, relative delta power in these areas for the three sleep terrors is proportional to the subjective intensity of the episode. Mapping of the other frequency bands showed that the control EEG sections had slightly but non-significantly more beta1 and alpha power in parietal and occipital regions. No differences were noted in the beta2 and theta frequency bands.

## DISCUSSION

The results demonstrate the utility of EEG mapping procedures for the quantification of brain activation during sleep terror attacks. In a single participant, EEG mapping produced reliable evidence of localized elevation in delta power prior to sleep terror episodes. Spectral analyses showed that all three sleep terrors were characterized by more total and delta power than the control EEG segments. Moreover, z-mapping of the EEG revealed increased delta power in central and frontal

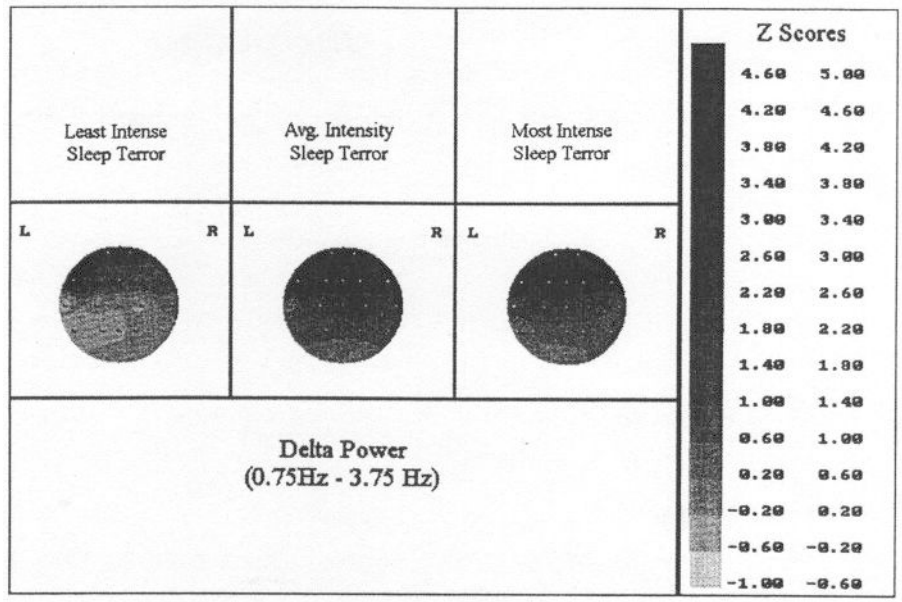


**Fig. 2.** z-maps for each of three sleep terrors.

regions—increases that were associated with the intensity of the episode. Even though there were no notable changes in autonomic variables in the pre-arousal period, there did exist a distinctive EEG pattern that preceded the onset of the sleep terror and that was related to its severity.

The elevated levels of delta power reported here are not due to the presence of a generalized, hypersynchronous symmetric delta pattern in the EEG, which has been observed prior to the onset of sleep terror and sleep walking episodes (Fisher, Kahn, Edwards, & Davis, 1974; Halász, et al., 1985). Although such EEG patterns were noted during portions of M's periods of slow wave sleep, they were absent from the EEG sections selected for the spectral analyses. Thus, even though sleep terrors are conceptualized as a "disorder of arousal" (Broughton, 1968), our preliminary findings suggest that their severity is related to the amount of delta power in central, frontal, and more particularly left frontal regions. This pre-arousal EEG pattern may be related to ongoing slow-wave sleep mentation that, as suggested by some authors (e.g., Fisher, Kahn, Edwards, & Davis, 1974; Kahn, Fisher, & Edwards, 1991), may sometimes trigger sleep terror episodes. Finally, it is also possible that there is a relatively gradual "buildup" of delta activity that contributes to the onset and intensity of sleep terrors.

The functional significance of the EEG patterns associated with the onset of sleep terrors is unclear. There has been considerable debate about the lateralization of function in the expression of emotions. Some reviews suggest that the right hemisphere is more critically involved in processing negative emotions and the left hemisphere in processing positive emotions (e.g., Tucker, 1981; Silberman &

Weinbartner, 1986), while others propose a right hemisphere dominance for all types of emotional expression (e.g., Borod, 1992; Kolb & Whishaw, 1996). How differential patterns of slow-wave EEG activity are related to various proposed brain mechanisms for the generation and processing of affect remains an open question.

The present study, together with our previous results on the EEG mapping of REM nightmares (Nielsen & Zadra, 1997), illustrates how quantitative EEG mapping techniques may be applied to the study of unusual dreaming states. These results suggest that discrete activity profiles are identifiable for different types of dreaming-related arousal.

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