

Increased delta power and discrepancies in objective and subjective sleep measurements in borderline personality disorder

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Abstract

Background: Previous studies have shown depression-like sleep abnormalities in borderline personality disorder (BPD). However, findings in BPD are not unequivocal for REM dysregulation, as well as for a decrement of slow wave sleep and sleep continuity disturbances. Earlier findings in sleep EEG abnormalities in BPD may have been confounded by concomitant depressive symptoms.

Methods: Twenty unmedicated female BPD patients without current comorbid major depression and 20 sex- and age-matched control subjects entered the study. Conventional polysomnographic parameters and for the first time sleep EEG spectral power analysis was performed on two sleep laboratory nights. Subjective sleep parameters were collected by sleep questionnaires in order to assess the relationship between objective and subjective sleep measurements.

Results: BPD patients showed a tendency for shortened REM latency and significantly decreased NonREM sleep (stage 2). Spectral EEG analysis showed increased delta power in total NREM sleep as well as in REM sleep in BPD patients. Subjective ratings documented drastically impaired sleep quality in BPD patients for the two weeks before the study and during the two laboratory nights.

Conclusion: Not-depressed BPD patients only showed tendencies for depression-like REM sleep abnormalities. Surprisingly, BPD patients displayed higher levels of delta power in the sleep EEG in NREM sleep than healthy control subjects. There was a marked discrepancy between objective and subjective sleep measurements, which indicates an altered perception of sleep in BPD. The underlying psychological and neurobiological mechanisms of these alterations are still unclear and need to be clarified in future studies including interventions on a pharmacological and cognitive-behavioral level.

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1. Introduction

Borderline personality disorder (BPD) is a frequently diagnosed personality disorder with a prevalence of 1–2% in the general population (Gunderson and Zanarini,

1987) encompassing a variety of symptoms such as aversive inner tension, self-injurious behavior and affective dysregulation. Furthermore, disturbances of sleep continuity and nightmares are frequently encountered in BPD (Asaad et al., 2002).

Up to now 10 sleep-EEG studies have been published in BPD. Some of the earlier studies did not explicitly control for concomitant major depression in BPD. Therefore, in these studies findings of depression-like sleep abnormalities such as shortened REM latency

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(Bell et al., 1983; McNamara et al., 1984; Reynolds et al., 1985; Lahmeyer et al., 1988) as well as sleep continuity disturbances (Bell et al., 1983) and increased REM density (Bell et al., 1983; McNamara et al., 1984) might be interpreted as a consequence of the concomitant depression. Six studies explicitly measured and reported concomitant depression and four of these studies reported reduced REM latency (Akiskal et al., 1985; Asaad et al., 2002; Battaglia et al., 1993; Benson et al., 1990) suggesting a common biological origin for affective disorders and BPD. In one study (Battaglia et al., 1999), young never-depressed BPD patients had higher REM density during the first REM period extending the view that REM density in the first REM period could be a marker of liability to affective disorders (Modell et al., 2002). In one recent study REM latency did not differentiate BPD and MDD and normal controls (De la Fuente et al., 2001).

To our knowledge, there is no study which investigated the microstructure of sleep in BPD. Recently, higher theta and delta power in stage 4 have been found in male antisocial patients with BPD comorbidity compared to controls (Lindberg et al., 2003a). This is in contrast to almost all other studies of spectral sleep EEG power in psychiatric populations (with the exception of patients with panic disorders), where mainly reductions of delta power have been described (especially in depressed patients; for overview see Riemann et al., 2001).

One sleep study reported more concomitant sleep complaints in BPD than in healthy controls (Asaad et al., 2002). However, earlier polysomnographic studies in BPD did not provide data on questionnaires validated to indicate the presence of sleep disturbances (e.g., Pittsburgh Sleep Quality Index, PSQI, (Buysse et al., 1989)).

In the present study, we aimed to assess whether the subjective ratings of sleep disturbances in patients with BPD are associated with objective polysomnographic findings and whether alterations of the sleep EEG spectral power can be observed in nondepressed female BPD patients when compared with healthy controls.

2. Materials and methods

2.1. Subjects

Twenty female patients with BPD meeting the diagnostic criteria of DSM-IV entered the study. In all patients the core symptoms of emotional instability and impulsivity were reported already in adolescence. The patient sample was restricted to female sex in order to avoid sex influences and to guarantee stronger homogeneity of the samples. Diagnosis of BPD was confirmed by assessment of the appropriate segment

of the structured Clinical Interview for DSM-IV (SCID-II) and comorbid Axis-I-disorders were assessed by the SCID-I (First, 1997). The last episode of any other psychiatric lifetime diagnosis had to have ended at least six months prior to investigation in the sleep laboratory. Patients with comorbid current major depression and alcohol or substance abuse and dependency were excluded from the trial. The study protocol was approved by the local ethical committee of the University of Freiburg. Written informed consent was obtained from patients and healthy subjects prior to study participation.

BPD patients were compared with a healthy control group which included 20 exactly age- and gender-matched subjects from our database. All patients and control subjects were free of psychotropic medication for at least two weeks prior to investigation, which was controlled by urine drug screening prior to the first night in the sleep laboratory (subjects with positive results were excluded from the study). None of the subjects took contraceptive medication during the study and for at least four weeks prior to the investigation. Menstrual cycle was not controlled in the study. Demographic and clinical characteristics of the 20 patients are given in Table 1.

Healthy controls were assessed by two experienced psychiatrists with semi-structured interviews to exclude any kind of psychopathology. Furthermore, results of an EEG, ECG and routine blood testing (blood count, renal, hepatic and thyroid function) had to be within normal limits.

2.2. Sleep recordings

Patients and controls spent two nights in the sleep laboratory. The first night served for adaptation and for the exclusion of clinically relevant periodic leg movements (PLMS) and sleep apnea syndrome. A PLMS index $>5/h$ (with arousal) and an apnea/hypopnea-index (AHI) $>5/h$ were considered as exclusion criteria.

Polysomnography (PSG) encompassed EEG (C3-A2, C4-A1), horizontal and vertical eye movements and submental EMG. Musculus anterior tibialis and respiration (oral/nasal air flow and thoracic/abdominal respiratory effort and oxygen saturation) were recorded only during the first night in the sleep laboratory.

All recordings were carried out from 11 p.m. to 7 a.m. All sleep recordings were scored in 30-s epochs, according to Rechtschaffen and Kales criteria (1968) by experienced raters “blind” to the experimental conditions (group and night) of the recordings.

The following sleep parameters were determined: total sleep time (min); sleep efficiency (in %, i.e., total sleep time/time spent in bed $\times 100$); latency to stage 2

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