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Narcolepsy type 1 and hypersomnia associated with a psychiatric disorder show different slow wave activity dynamics

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The aim of the study was to compare electrophysiological parameters of night sleep in narcolepsy type 1 and hypersomnia associated with a psychiatric disorder. Forty-four patients: 15 with narcolepsy type 1, 14 with hypersomnia associated with a psychiatric disorder and 15 age- and sex-matched controls participated in the study. The study subjects filled in the Athens Insomnia Scale (AIS) and the Beck Depression Inventory (BDI). The severity of daytime sleepiness was quantified subjectively using the Epworth Sleepiness Scale (ESS) and the Stanford Sleepiness Scale (SSS), and objectively using the Multiple Sleep Latency Test (MSLT). All subjects underwent polysomnography (PSG) on the two consecutive nights. The data from the second night was analysed. The slow wave activity (SWA, 1–4 Hz) was calculated for the three consecutive sleep cycles, and topographic delta power maps were plotted. In contrast to narcoleptics, psychiatric hypersomniacs had undisturbed nocturnal sleep, high sleep efficiency, normal non-rapid eye movement (NREM) and rapid eye movement (REM) sleep proportions, normal REM latency and sleep latencies on MSLT and PSG. The subjective and objective sleepiness was significantly higher in narcolepsy group than in psychiatric hypersomnia group. In all the study groups SWA was the most prominent in frontal areas, while the greatest between-group differences were found in the central areas. There were significant differences between the groups in SWA in the second NREM episode. The highest SWA was observed in the hypersomnia group, while the lowest in the narcolepsy group. Psychiatric hypersomniacs and controls did not differ in the SWA exponential decline over consecutive NREM episodes, whereas narcoleptics exhibited a steeper dissipation of sleep pressure from the first to the second NREM episode. In conclusion, narcolepsy type1 and hypersomnia associated with psychiatric disorder differ in the SWA dynamics. Narcoleptics presented with the altered dynamics of sleep homeostasis, whereas psychiatric hypersomniacs showed normal nocturnal sleep and normal sleep homeostasis.

Key words: hypersomnia, narcolepsy, hypersomnolence, slow wave activity, sleep homeostasis.

INTRODUCTION

According to the third revision of the International Classification of Sleep Disorders (ICSD-3), narcolepsy type 1 (N1) and hypersomnia associated with a psychiatric disorder (HPD) belong to the group of Central Disorders of Hypersomnolence. They are characterized by excessive daytime sleepiness (EDS) that is not caused by a disturbed sleep, abnormalities of respiration during sleep or misaligned circadian rhythms (American Academy of Sleep Medicine 2014). EDS occurs in 5–15% of the general population (Partinen and Hublin 2011).

Narcolepsy type 1 (narcolepsy with cataplexy, NC, according to ICSD-2) (American Academy of Sleep Medicine 2005) is a homogenous condition caused by a deficiency of hypothalamic hypocretin transmission. The disorder is characterized by EDS, signs of rapid eye movement (REM) sleep dissociation, like cataplexy, sleep paralysis, hypnagogic hallucinations and fragmented nocturnal sleep (American Academy of Sleep Medicine 2014). Total sleep time over 24 hour period in narcoleptics is normal but its distribution is altered. The patients exhibit more daytime sleep and more wakefulness during sleep than controls (Broughton et al. 1988). It is postulated that the severe hypocretin deficiency results in the destabilization of hypothalamic control of sleep and wakefulness and causes sleep-wake transitions instabilities (Saper et al. 2001, Sorensen et al. 2013).

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On the contrary, hypersomnia associated with a psychiatric disorder is a heterogenous condition with EDS, excessive nocturnal sleep, or excessive napping. Associated psychiatric disorders include mood disorders, conversion or somatoform disorders, and other mental disorders. There is no concordance between subjective and objective sleepiness. The mechanisms through which the sufferers express their psychiatric symptoms as excessive sleepiness is still unknown (American Academy of Sleep Medicine 2014). Hypersomnia is highly prevalent in various mood disorders and is associated with increased risk of depressive relapse and suicide. Hypersomnia is often treatment resistant. Despite its significant impact on natural history of mood disorders, the origins, clinical manifestations, course and sequelae of hypersomnia associated with a psychiatric disorder are poorly understood (Kaplan and Harvey 2009, Plante 2015). Polysomnography (PSG) studies in depression show disturbances of sleep continuity, reduction of slow wave sleep (SWS) and disinhibition of REM sleep (Kupfer and Foster 1972, Benca et al. 1992, Riemann et al. 2001).

According to the Two-Process Model of Sleep Regulation, the sleep propensity is jointly determined by a homeostatic and a circadian process. The homeostatic process (Process S) increases gradually during wakefulness, which reflects an increasing sleepiness, and declines exponentially during sleep. A reliable electroencephalography (EEG) marker of Process S is slow wave activity (SWA, 1–4 Hz), an indicator of sleep homeostasis and sleep intensity (Borbély and Tobler 2011). In depression, the build-up of process S is deficient, which results in SWS reductions during sleep and shorter REM sleep latency. After sleep deprivation the process S is restored to the normal level, which causes an instant but short antidepressant effect (Borbély 1987, Borbély and Wirz-Justice 1982). Although sleep in depression has been widely studied there is still limited data on sleep in psychiatric hypersomnia. Dolenc and others (1996) found the reduction of SWS in patients with dysthymia and hypersomnia. Plante and colleagues (2012) demonstrated the significant reduction of SWA in patients with major depressive disorder (MDD) and hypersomnia. It suggests the insufficient build-up of the process S during wakefulness in psychiatric hypersomnia. Similar mechanism is postulated in idiopathic hypersomnia (Sforza et al. 2000).

In narcolepsy, PSG studies consistently show a disrupted night sleep with frequent brief awakenings, arousals and an increased amount of stage 1 sleep (Overeem et al. 2001, Roth et al. 2013, American Academy of Sleep Medicine 2014). The destabilization of hypothalamic control of sleep and wakefulness results in frequent, undesired sleep-wake state transitions (Saper et al. 2001, Sorensen et al. 2013). Khatami and others (2007) found that narcoleptic subjects exhibited a steeper declining trend of SWA from the first to the second sleep episode in comparison to controls, which results from an insufficient non-rapid eye movement (NREM) intensity.

The differential diagnosis of hypersomnias is a challenging task because the diagnostic criteria overlap (Vgontzas et al. 2000, American Academy of Sleep Medicine 2014). There are no definite tests to confirm HPD and a broad differential diagnosis is needed to rule out other causes of sleepiness. There are limited studies using objective measures of sleep propensity in psychiatric hypersomnia. EDS in mood disorders is rather a subjective complaint than an objective finding. Besides, depressive symptoms are frequently noted in central disorders of hypersomnolence, for example in narcolepsy (Dauvilliers et al. 2013), which makes the differential diagnosis of various hypersomnias more difficult.

Therefore, we aimed to evaluate electrophysiological parameters of central disorders of hypersomnolence. To the best of our knowledge, there are no studies on sleep EEG brain mapping in patients with hypersomnia associated with a psychiatric disorder compared to patients meeting criteria of narcolepsy type 1 and healthy controls. Furthermore, we evaluated sleep parameters, daytime sleepiness, subjective difficulties with night sleep, depressive feelings and psychosocial impact in the two groups of patients.

METHODS

Participants

The study was performed in twenty nine outpatients meeting ICSD-3 (American Academy of Sleep Medicine 2014) criteria for central disorder of hypersomnolence, 15 subjects with narcolepsy type 1 (narcolepsy with cataplexy, 6 men and 9 women, N1) and 14 with hypersomnia associated with a psychiatric disorder (6 men and 8 women, HPD), recruited from Sleep Disorders Clinic, Medical University of Warsaw. Psychiatric diagnoses were established according to the International Classification of Diseases, 10th Edition (ICD-10) criteria (World Health Organization 1993). Exclusion criteria included: somatic disorders, misusing substances or drugs, psychotic or dementia disorders, other sleep disturbances causing sleepiness as breathing-related sleep disorders or insufficient sleep syndrome.

The control group consisted of 15 healthy age- and sex-matched volunteers, recruited from the employees of the Nowowiejski Hospital in Warsaw and the Institute of Cardiology in Warsaw (C).

Both healthy volunteers and patients were required to be free of any prescription or nonprescription drugs

for at least 2 weeks prior to the study. Sleep habits were confirmed by sleep diaries. All subjects gave informed consent before participating. The study was approved by the University Bioethics Committee.

Procedure

Prior to entering the study, each subject underwent psychometric assessment including the Beck Depression Inventory (BDI, Beck et al. 1961). The disruption of night sleep was quantified by means of 8-item version of the Athens Insomnia Scale (AIS, Soldatos et al. 2000, 2003, Fornal et al. 2011), the severity of daytime sleepiness was assessed subjectively using the Epworth Sleepiness Scale (ESS, Johns 1991) and the Stanford Sleepiness Scale (SSS, Hoddes et al. 1973), and objectively using the Multiple Sleep Latency Test (MSLT, Carskadon 1986).

EEG data and visual scoring

All participants were studied on two consecutive nights with laboratory-based PSG. The first adaptative night was used to rule out sleep apnea, periodic limb movements and other sleep disorders. On the second night, EEG signal was recorded from 23 derivations following an extended version of the international 10–20 system (with additional electrodes at Fpz and Oz) versus reference electrode positioned between Fz and Cz. The impedance of the electrodes was kept below 10 kΩ. The signals were sampled at 128 Hz and filtered between 0.15 and 30 Hz. Sleep stages were visually scored according to the Rechtschaffen and Kales criteria (1968), in 20 s segments, from C3-A2 derivation. Directly after the second night of PSG, the consecutive five sessions of routine diagnostic MSLT were conducted (Carskadon 1986).

EEG data and spectral analysis

EEG data from the second nights of PSG recordings were subjected to spectral analyses. First, the power spectra were calculated using a fast Fourier transform algorithm (FFT) in 4 seconds segments (Hamming widow) for the whole sleep. FFT was performed for frequencies between 1 and 35 Hz (data from C3-A2 derivation). The data were plotted to make individual EEG-chronospectrograms. Second, the EEG-chronospectrograms were compared to individual hypnograms and the three consecutive NREM-REM sleep cycles were marked according to Feinberg and Floyd criteria (1979). The peak values of SWA were visually detected and matched with hypnograms to determine the last 240 seconds of artefact-free EEG recordings of SWS (stage 3 or 4) for the three consecutive sleep cycles in every subject. The EEG was converted to an average reference, and spectral analysis was performed for 240 s of the peak value of SWA in the three consecutive sleep NREMs for 21 channels. Maps of absolute delta power (1–4 Hz) were plotted for the sleep cycles.

We used REMbrandt®7.2 Sleep Diagnostic Software, The Source Advanced Analysis software package (ASA 4; ANT Software BV) and topoplot function of EEGLAB software for the quantitative analysis of the EEG.

Statistical analysis

Normal distribution in the groups was checked with Kolmogorov-Smirnov test. We used one-way ANOVA test and *post-hoc* Duncan test for comparisons between groups for variables with normal distribution. For variables with non-normal distribution we used nonparametric Kruskal-Wallis test, and the t-test for comparisons between the two groups.

Comparisons between the 3 groups for the 3 consecutive sleep cycles were performed using one-way ANOVA test and *post-hoc* Duncan test. Changes in the value of power in subsequent sleep cycles and the percentage of power decrease were analyzed for two derivations (Fz, Cz) using two-way analysis of variance for repeated measures (rANOVA, groups * sleep cycles).

All statistical calculations were performed using program STATISTICA 12.0 (Stat Soft, Krakow, Poland).

The results of the study are presented as arithmetic mean and standard error (±SE). We considered $p<0.05$ as statistically significant.

RESULTS

Demographic and clinical characteristics

There were no significant differences between groups in mean age (N1 – mean age 34.4±2.97, range 17–53 years; HPD – mean age 27.7±2.39, range 21–56 years; C – mean age 32.2±2.42, range 22–51 years; $F_{2,41}=1.7$, $p=0.2$).

Narcoleptics were less educated than HPD and C. Their education was shorter (10.87 years±0.93) comparing to HPD (13.86±0.8) and C (15.67 ±0.7, N1 *vs.* C $p<0.001$, N1 *vs.* HPD $p<0.05$; *post-hoc* Duncan test).

4 of 15 patients with N1 and 3 of 14 patients with HPD were unemployed. Five narcoleptics complained about job loss because of daytime sleepiness. 9 of 15 narcoleptics and 2 of 14 hypersomniacs claimed that they had sleep-related accidents. In 5 narcoleptics and in 1 hypersomniac the accident occurred during driving. In 4 of 15 narcoleptics and 1 of 14 hypersomniac an

accident occurred while operating house equipment, and in 5 of 15 narcoleptics an accident occurred at work. Three narcoleptics had several accidents under various circumstances.

All the HPD patients had ICD-10 (World Health Organization 1993) psychiatric diagnosis: 5 subjects were diagnosed with dysthymic disorder, 1 subject with major depressive disorder, single episode, in partial remission, 3 subject with mixed anxiety and depressive disorder, 6 subjects with somatoform disorders and 1 subjects with dissociative disorder. 12 patients had one and 2 patients obtained two psychiatric diagnoses. We also found that 4 of 15 narcoleptics had additional psychiatric diagnosis: 3 subjects were diagnosed with major depressive disorder and 1 subject with adjustment disorder.

Psychometric tests

The BDI results differed significantly between the groups ($F_{2,41}$=10.51, p<0.001), N1 and HPD subjects obtained higher scores than C subjects (N1 – 15±2.29 *vs.* C – 2.47±0.8, p<0.001, HPD – 12.14±2.65 *vs.* C – 2.47±0.8, p<0.01, *post-hoc* Duncan test).

15/15 of N1 and 12/14 of HPD subjects filled in the AIS. They complained of difficulties with night sleep and their AIS scores were higher than those of healthy controls ($F_{2,39}$=10.19, p<0.001, N1 – 9.6±1.22 *vs.* C – 3.67±0.44, p<0.01, HPD – 10.5±1.6 *vs.* C – 3.67±0.44, p<0.001, *post-hoc* Duncan test).

Analyses of daytime sleepiness between the N1 and the HPD groups showed a significant difference. The N1 group scored significantly higher than the HPD group on the ESS (20.06±0.93 *vs.* 14.92±0.7, t=4.32, p<0.001) and on the SSS (5.46±0.34 *vs.* 3.57±0.34, t=3.94, p<0.001).

PSG parameters

The comparison of PSG parameters demonstrated significant differences between narcolepsy and the two remaining groups (Table I). N1 exhibited significantly shorter sleep latency, more stage 1 sleep, more wake during sleep, less stage 2 and stage 3+4 sleep comparing to HPD and C. Moreover, N1 showed a significantly shorter REM sleep latency than the other groups, whereas REM sleep duration and percentage did not differ.

The mean sleep latency measured using MSLT was significantly shorter in N1 than in HPD and C ($F_{2,41}$=91.37, p<0.001; N1 – 2.86±0.45, HPD – 12.14±0.72, C – 14.49±0.75; N1 *vs.* C p<0.001, N1 *vs.* HPD p<0.001, HPD *vs.* C p<0.05, *post-hoc* Duncan test). N1 showed significantly more Sleep Onset REM Periods (SOREMPs) than HPD and C subjects ($H_{2,41}$=39.28, p<0.001; N1 – 3±0.28, HPD – 0.07±0.07, C – 0; N1 *vs.* C p<0.001, N1 *vs.* HPD p<0.001, Kruskal-Wallis test).

EEG spectral analysis

In all the groups the predominance of EEG delta activity in the three NREM sleep episodes was observed in frontal areas. The delta power decreased across the

Table I. Polysomnographic parameters of night sleep in narcolepsy group – N1, hypersomnia group – HPD, and control subjects – C [ANOVA $_{df\ 2,41}$ – (F), Kruskal-Wallis test $_{df\ 2,41}$ – (H)]. Means ±Standard Errors (SE) are shown. * p<0.05 for N1 *vs.* HPD, # p<0.05 for N1 *vs.* C (post-hoc)

Group / Sleep parameters	N1 (n=15) Mean ±SE	HPD (n=14) Mean ±SE	C (n=15) Mean ±SE	F/H, p
Sleep latency (min)	2.56±0.46*#	15.72±3.9	16.78±3.12	F=7.87 p<0.01
Total sleep time (min)	401.21±17.03	418.99±14.63	385.74±10.16	NS
Sleep efficiency (%)	88.26±2.69	94.24±2.42	94.2±1.35	NS
REM sleep latency (min)	21.75±11.42*#	75.96±8.57	86.51±8.79	H=17.39 p<0.001
stage 1 of NREM sleep (%)	18.52±4.06*#	4.69±1.05	5.04±1.2	F=9.54 p<0.001
stage 2 of NREM sleep (%)	39.19±2.13*#	50.72±2.06	50.52±1.14	F=13.25 p<0.001
stages 3+4 of NREM sleep (%)	15.69±1.54*#	19.44±1.33	21.43±0.58	F=5.87 p<0.05
REM sleep (%)	26.55±2.58	25.14±1.36	22.79±1.17	NS
Wake after sleep onset (%)	13.17±3.93*#	5.86±3.71	3.15±1.63	H=9.31 p<0.05

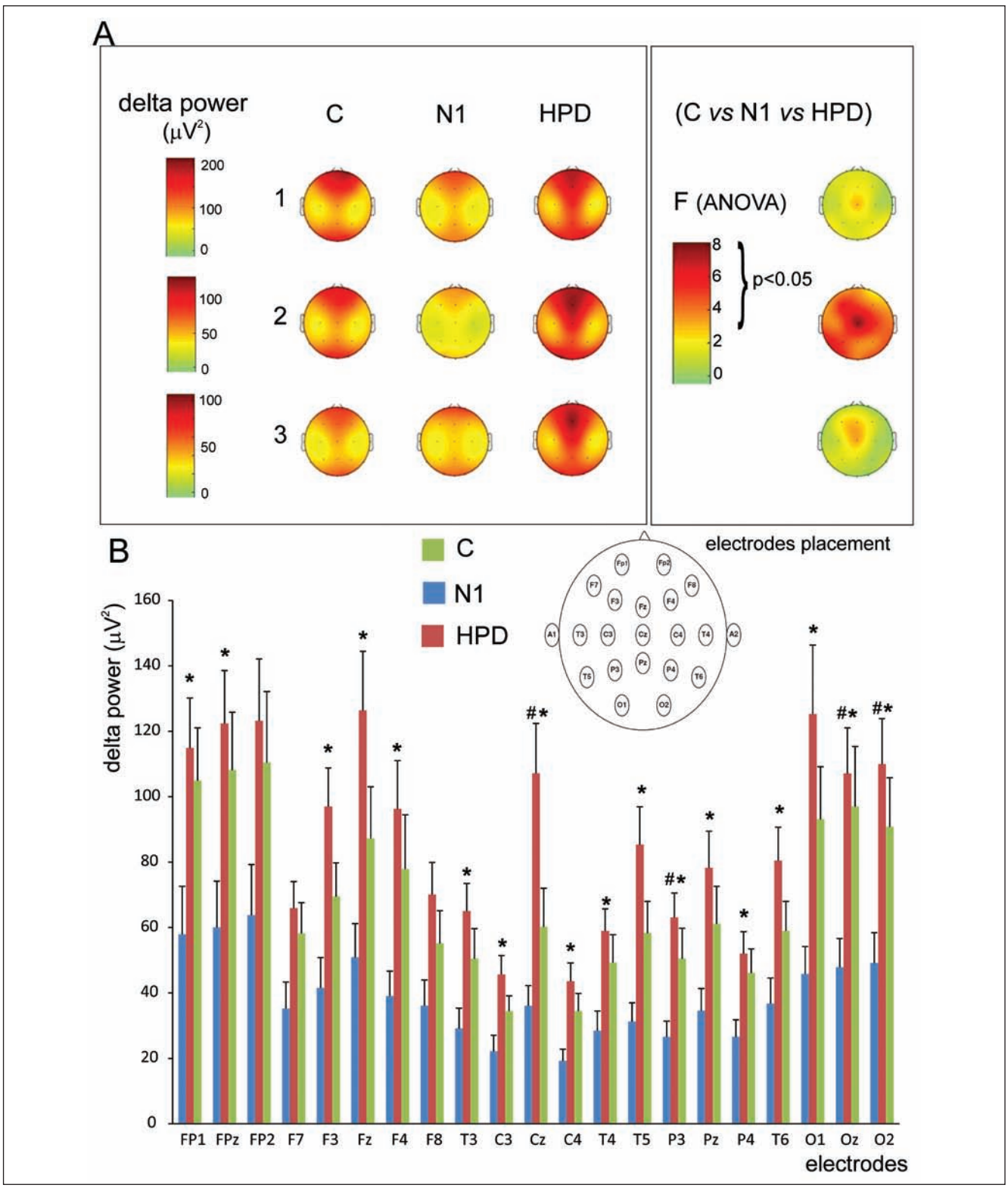

Fig. 1. (A). The topographic distribution of delta power in consecutive NREM episodes (1, 2, 3) in control subjects (C, n=15), Narcolepsy type 1 (N1, n=15) and Hypersomnia associated with a psychiatric disorder (HPD, n=14). The last column shows the F values for C-N1-HPD comparison by one-way ANOVA; $p<0.05$ for F equal or more than 3.32. Significant between group differences were found in the second NREM episode in 18 out of 21 EEG derivations (detailed data is shown in B). (B). The mean values of absolute delta power from 21 EEG derivations in the second NREM episode in control subjects (C, n=15, green bars), Narcolepsy type 1 (N1, n=15, blue bars) and Hypersomnia associated with a psychiatric disorder (HPD, n=14, red bars). The comparison of SWA in the three groups (ANOVA, *post-hoc* Duncan) from EEG derivations. * $p<0.05$ (N1 *vs.* HPD), # $p<0.05$ (N1 *vs.* C).

three consecutive NREMs in the HPD and C groups. In the N1 group the delta power decreased from the first to the second episode and then increased from the second to the third episode in almost all EEG derivations (18 of 21). The topographic distribution of SWA and the maps of between groups differences in the three consecutive NREM episodes are depicted on Fig. 1A.

There were significant differences between the groups in delta power in the second NREM episode. The highest level of SWA was observed in the HPD group, the intermediate level in controls and the lowest in the N1 group. The greatest differences were present in the central areas (Fig. 1B).

The predominance of SWA was observed in the three groups in frontal areas and the greatest between group differences were seen in central areas. Thus the Fz and Cz leads were selected for the comparison of SWA dissipation over three consecutive NREM episodes.

Two-way rANOVA revealed significant difference in the relative delta power change in time between groups for Cz derivation (groups N1, HPD, C × sleep episode 2–3, $F_{2,40}$=3.33, p<0.05) and for Fz derivation (groups N1, HPD, C × sleep episode 2–3, $F_{2,40}$=3.54, p<0.05). *Post-hoc* analysis showed that N1 had significantly lower relative SWA than HPD for Cz and Fz derivations in the second NREM episode. N1 had significantly lower relative SWA

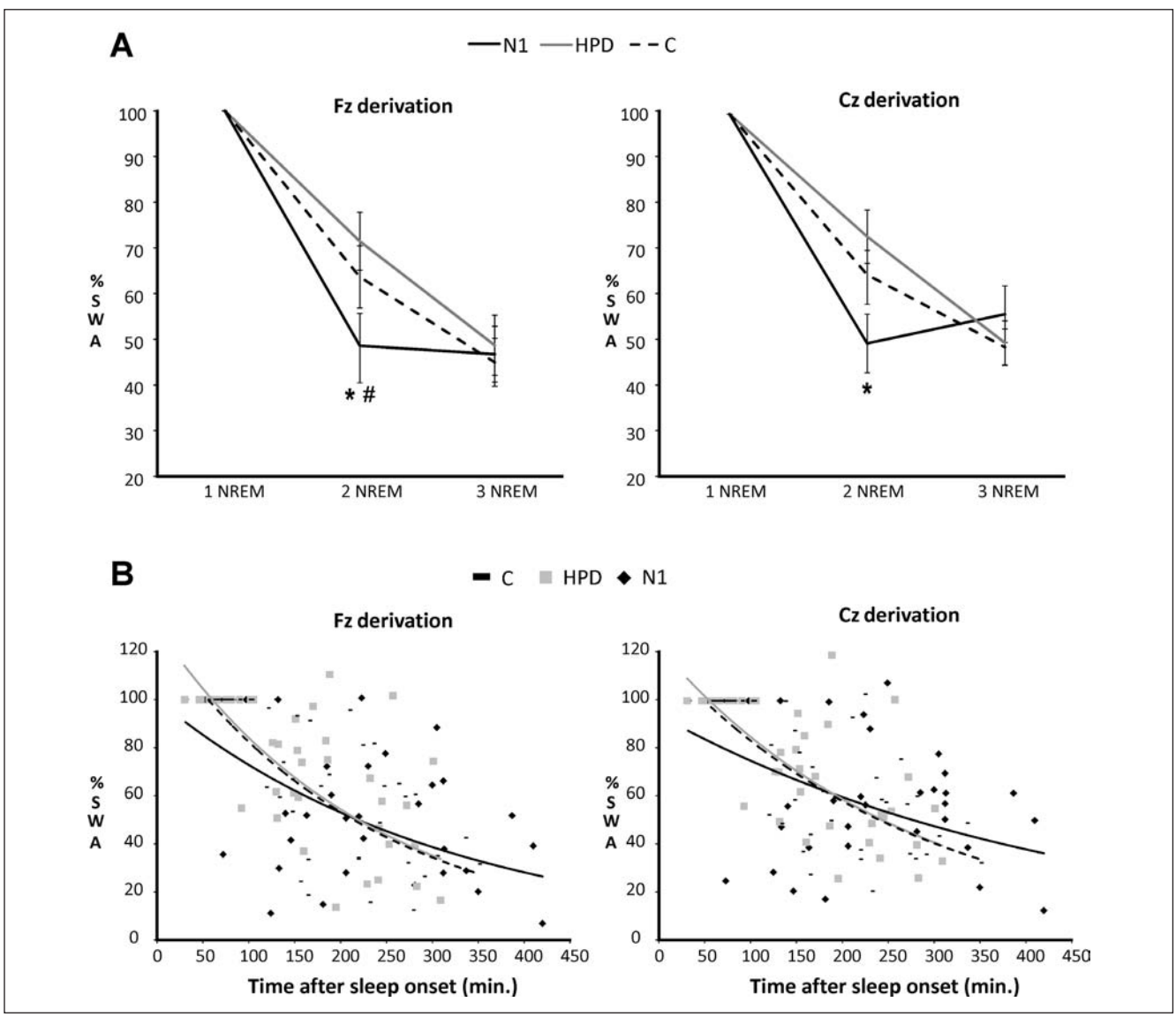

Fig. 2. The percentage change of slow-wave activity (SWA) across three consecutive NREM sleep episodes for Cz and Fz derivations in three groups: narcolepsy type 1 (N1, n=15, black lines, black diamonds), hypersomnia associated with psychiatric disorder (HPD, n=14, grey lines, grey squares) and control subjects (C, n=15, dashed lines, black rectangles). SWA values are showed as a relative to the SWA value of the first NREM episode (100%). (A) shows the averaged group values of SWA ±SE in the three NREM episodes. * p<0.05 for N1 *vs.* HPD, # p<0.05 for N1 *vs.* C (ANOVA, *post-hoc* Duncan). (B) shows the individual values of SWA in the time after sleep onset. The curves represent the trend lines for SWA change.

than C for Fz derivation in the second NREM episode. In the N1 group in the Cz derivation there was a decrease of SWA followed by an increase of SWA, while in the Fz derivation SWA did not change significantly between the second and the third NREM episodes. HPD and C did not differ in the SWA exponential decline over NREM episodes, whereas N1 exhibited a steeper sleep pressure dissipation from the first to the second NREM episode than HPD and C (Fig. 2).

DISCUSSION

A new finding of the study is that psychiatric hypersomniacs and narcoleptics differ in the SWA exponential decline over NREM sleep episodes.

Our study confirmed a profound psychosocial impact of narcolepsy. Narcoleptic patients were less educated than hypersomniacs and controls. One third of the N1 group lost a job because of EDS, and one third had a sleep-related automobile accident. This is in line with previous studies showing lower quality of life, higher frequency rates of automobile accidents and decreased simulated driving performance in narcolepsy (Broughton et al. 1981, George et al. 1996, Findley et al. 1999).

BDI scores indicated a moderate depression in the N1 and the HPD groups according to the validation of the Polish version of the inventory (Parnowski and Jernajczyk 1977). Self-reported depressive symptoms in patients with sleep disorders were also reported in the previous study, with the mean BDI score for narcoleptics of 10 or more (Mosko et al. 1989, Daniels et al. 2001, Vandeputte and Weerd 2003, Dauvilliers et al. 2009). In our study, BDI scores in the HPD group were lower than in the N1 group, and lower than in nonorganic hypersomnia in the study by Kofmel and others (2014). In our HPD group only 6 of 14 subjects met mood disorders criteria. The frequency of low mood disorders in our HPD group is in line with HPD differentiation into two subtypes i.e., mood disorders and a conversion disorder or somatic symptom disorder, according to ICSD-3 (American Academy of Sleep Medicine 2014).

The subjective and objective sleepiness

In our study, narcolepsy and hypersomnia subjects scored higher in the AIS than controls, which reflects subjective complaints on night sleep. Narcoleptics typically report disruption of nocturnal sleep such as an inability to maintain sleep (Aldrich 1992, Overeem et al. 2001, Dauvilliers et al. 2007, American Academy of Sleep Medicine 2014). The N1 group showed an elevated AIS score, which was coherent with PSG results and reflected the disrupted night sleep. In contrast, the HPD group complained about the poor quality of sleep despite normal PSG results.

The severity of subjective and objective daytime sleepiness was significantly higher in the N1 group than in the HPD group. The N1 group obtained higher scores in the sleepiness scales than the HPD group. The N1 group had severe subjective sleepiness according to Mitler and colleagues (2000), which was in line with objective measures in our study. Namely, the N1 group showed significantly shorter night sleep latency, REM latency, mean sleep latency in MSLT, and more SOREMS in MSLT in comparison to the HPD group and controls. The results confirm severe EDS in the N1 group, which is consistent with previous study by Dauvilliers and others (2001) and ICSD-3 (American Academy of Sleep Medicine 2014). In contrast to severe subjective and objective sleepiness in the N1 group, psychiatric hypersomniacs exhibited a moderate subjective sleepiness (Mitler et al. 2000) with no indicators of objective sleepiness, the mean sleep latency in MSLT in the HPD group was within the reference range for healthy. The discrepancy between objective and subjective findings is typical for HPD (American Academy of Sleep Medicine 2014). It is worth noting that although the MSLT is a widely used tool to measure the sleep propensity for diagnostic purposes it has several limitations. For example, the mean sleep latency of less than 8 minutes is a cut-off to define a pathological sleepiness (American Academy of Sleep Medicine 2014), while the meta-analysis by Plante and colleagues (2017) showed that the sleep propensity in patients with psychiatric hypersomnolence is similar to normal values, with only 25% of patients having the mean sleep latency below 8 minutes. It is unknown whether patients with HPD are essentially sleepy, or if they suffer from the lack of energy. Moreover, the differentiation between EDS and fatigue in HPD is particularly difficult and it is unknown to what extent those two symptoms overlap (Ohayon 2008).

The night sleep parameters

Comparison of sleep parameters demonstrated worse sleep quality in the N1 group than in the HPD group and controls. The N1 group presented with significantly more stage 1 sleep, less stage 2, 3+4 sleep and more wake during sleep when compared with the HPD group and controls. This indicates a decrease of slow-wave sleep and an increase of wakefulness and light sleep in our N1 group. Our results are in line with previous studies on disturbed nocturnal sleep in narcolepsy (Overeem et al. 2001, Roth et al. 2013, American Academy of Sleep Medicine 2014). The HPD group had normal, undisturbed nocturnal sleep, high

sleep efficiency, normal NREM and REM sleep proportions in comparison to controls. There are only a few PSG studies in HPD. Similarly to our results, Nofzinger and others (1991) didn't find sleep disturbances in patients with hypersomnia in bipolar depression. Billiard and colleagues (1994) found that the main pathology among subjects affected with hypersomnia associated with mood disorders was anergia facilitating or mimicking sleep. On the contrary, Dolenc and others (1996) found the abnormal macrostructure of sleep in 12 patients with dysthymia and hypersomnia. Similarly, Vgontzas and colleagues (2000) showed a disrupted nocturnal sleep in patients with psychiatric hypersomnia.

SWA

The topographic maps of NREMs revealed the highest SWA in frontal regions in the three studied groups. The predominance of EEG power in frontal regions in NREMs is well documented in literature (Tinguely et al. 2006).

The lowest SWA was observed in the N1 group, the intermediate in controls and the highest in the HPD group (see Fig. 1), however the difference was significant only in the second NREM episode.

Narcoleptics had the lowest SWA in spite of the highest level of EDS measured objectively and subjectively. Similarly, Pizza and others (2013) and Guilleminault and others (1998) found lower SWA in narcoleptics than in controls, suggesting the impairment of process S in narcolepsy. Insufficient sleep pressure may compromise consolidation and maintenance of sleep. The lower level of SWA and the insufficient NREM sleep intensity in the N1 group are coherent with the sleep disruption in PSG. Moreover, the N1 group presented with different SWA dynamics than HPD group and control subjects. The N1 group showed the steeper decline of SWA from the first to the second NREM episode. Similar findings were reported by Khatami and colleagues (2007) who found the steeper decay of SWA in NC, which reflected the inability to consolidate sleep and the insufficient NREM sleep intensity. After sleep deprivation SWA was increased and sleep fragmentation was postponed in NC, which indicates the preserved homeostatic regulation in NC (Khatami et al. 2008).

In our study the HPD group had the highest SWA. Significant differences between the three groups occurred in the second NREM episode (see Fig. 1B). The HPD group didn't show the sleep changes typical for depression. This is coherent with a moderate level of depressive feelings in the group. There are contradictory reports with regard to changes in sleep in depression. Peterson and Benca (2011) didn't find typical depression sleep changes in bipolar depression, atypical depression and in young patients with depression. PSG results and high SWA in our HPD group resemble sleep after deprivation, except of normal sleep latencies and normal SWS sleep proportions. Schwartz and others (2001) found higher SWA in NREMs and sleep profiles resembling sleep after deprivation in patients with seasonal affective disorder. There is some evidence of the depressogenic effect of NREM sleep in major depression (Beersma and van den Hoofdakker 1992). On the other hand, there is a hypothesis of depression that attributes the sleep changes in depression to the deficiency of Process S (Borbély 1987). The reduction of SWA in MDD and hypersomnia was demonstrated by Plante and others (2012) and in idiopathic hypersomnia by Sforza and colleagues (2000). It is assumed that the Process S–deficiency plays a role in the pathophysiology of hypersomnia. Our study shows that sleep pressure discharging in the HPD group and in controls was the same. It indicates the high sleep pressure, high sleep intensity and normal sleep homeostasis in HPD. Higher SWA is an objective finding that may confirm sleepiness in our HPD group. Further research is needed to find the mechanism through which the hypersomniacs express their psychiatric symptoms into excessive sleepiness.

To summarize, we compared PSG data and power spectra of hypersomnia associated with a psychiatric disorder and narcolepsy type 1. We found discrepancies between objective and subjective findings in the HPD group. Despite complaints of EDS, HPD subjects had normal sleep latencies in MSLT and in PSG, undisturbed nocturnal sleep and high sleep efficiency. In contrast, the N1 group presented with disturbed nocturnal sleep with a decrease of slow-wave sleep and an increase of wakefulness and light sleep. The HPD and the N1 groups differed in the SWA exponential decline over NREM episodes. The N1 group exhibited a steeper dissipation of sleep pressure from the first to the second NREM episode than the HPD group and controls. The discharging of sleep pressure in the HPD group and in controls was the same. The study indicates low sleep intensity and altered homeostatic sleep regulation in N1. In contrast, the HPD group exhibited normal sleep homeostasis, high sleep pressure and high sleep intensity. Additionally, we confirmed a profound psychosocial impact of narcolepsy. We also found elevated subjective measures of disturbed sleep and elevated measures of depression in the HPD and the N1 groups.

CONCLUSION

Our study shows that narcolepsy type 1 and hypersomnia associated with a psychiatric disorder differs in the SWA dynamics.

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