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Sleep EEG of patients with obsessive-compulsive disorder

Fritz Hohagen[1], Stephanie Lis[1], Stephan Krieger[1], Gaby Winkelmann[1], Dieter Riemann[1], Rosemarie Fritsch-Montero[1], Eibe Rey[2], Joseph Aldenhoff[2], Mathias Berger[1]

[1] Psychiatric Department, University of Freiburg, Hauptstrasse 5, D-79104 Freiburg, Germany
[2] Central Institute of Mental Health, Mannheim, Germany

Summary. Twenty-two patients suffering from an obsessive and compulsive disorder (OCD) according to DSM-III-R were investigated by polysomnographic sleep EEG recordings under drug-free conditions and compared to age- and sex-matched healthy controls. Sleep efficiency was significantly lower and wake % SPT was significantly increased in the patient group compared to healthy subjects. Sleep architecture did not differ among the two samples. Especially REM sleep measures, in particular, REM latency did not differ among the groups. No positive correlation was found between sleep variables and rating inventories for obsession and compulsions (Y-BOCS), depression (Hamilton) and anxiety (CAS). A secondary depression did not influence sleep EEG variables. The results of this study contradict the assumption that OCD patients show REM sleep and slow wave sleep abnormalities similar to those shown by patients with primary depression.

Key words: Obsessive and compulsive disorder – Sleep EEG – REM sleep

Introduction

Obsessive compulsive disorder (OCD) is a chronic and potentially disabling illness characterized by recurrent thoughts, ideas or repetetive, relatively stereotyped behaviors that the patient feels driven to perform and recognizes as ego dystonic. Recent evidence suggests that the prevalence of OCD is greater than commonly believed, with current estimates of lifetime prevalence as high as 2.0–3.0% in the general population (Robins et al. 1984). For more than 100 years neurobiological factors have been thought to play an important role in the pathogenesis of obsessive and compulsive symptoms (Tuke 1894). There is a growing body of evidence which supports the view of a frontal cortex/basal ganglia dysfunction in OCD. Many authors reported the manifestation of obsessive and compulsive symptoms in specific neurological conditions with an underlying dysfunction of basal ganglia, i.e. in cases of encephalitis lethargica Economo (Bender 1935; Schilder 1938), Gilles-de-la Tourette syndrome (Pauls et al. 1986), Sydenham's chorea (Rapoport 1988, 1991) and after bilateral lesions of nucleus pallidus (Laplane et al. 1984). Furthermore, the remarkable success of psychosurgical sectioning of pathways between the frontal lobe and basal ganglia (for overview, see Khanna 1988) supports the hypothesis of a basal ganglia/frontal lobe dysfunction in OCD. Recently, PET studies in patients suffering from OCD showed that OCD patients had higher levels of glucose metabolism in areas of the frontal lobe and in the cingulate pathway which connects the frontal lobe and the basal ganglia (Baxter et al. 1987; Swedo et al. 1989).

Several lines of evidence point to the involvement of the neurotransmitter serotonin in the pathophysiology of obsessive-compulsive disorder. Clomipramine, a potent serotonin reuptake blocker (Thoren et al. 1980; Marks et al. 1980; Montgomery 1980; Ananth et al. 1981; Insel et al. 1983; Flament et al. 1985), and the new selective 5-HT re-uptake blockers fluoxetine (Fontaine and Chouinard 1985; Turner et al. 1985) and fluvoxamine (Perse et al. 1987; Price et al. 1987; Goodman et al. 1989a, 1990) have been found to be effective while various tricyclics acting on noradrenergic transmission and benzodiazepines have not proven to be useful in the treatment of OCD. Biochemical studies on platelet serotonin levels in children with OCD (Flament et al. 1987) and challenge tests with meta-chloro-phenyl-piperazine (mCPP), a nonselective 5-HT receptor agonist (Zohar et al. 1987; Charney et al. 1988; Hollander et al. 1991), add more evidence for the involvement of a central nervous serotonergic deficit in OCD.

Up to now, surprisingly few studies have been done to investigate the sleep of patients with obsessive compulsive disorder. Insel et al. (1982) reported a significantly decreased total sleep time with more awakenings, less stage 4 sleep and shortened REM latencies in OCD patients compared to a group of age- and sex-matched normal subjects. These abnormalities, especially the finding of shortened REM latencies in OCD patients, resembled

Correspondence to: Fritz Hohagen

those of an age-matched group of depressed patients. Recently, another study showed normal REM latencies in OCD patients (Walsleben et al. 1990). Thus, the question still remains open, whether OCD patients show REM sleep abnormalities comparable to those generally observed in depressed patients, for whom shortened REM latency, prolongation of the first REM period and heightened REM density are among the most remarkable and widely documented biological abnormalities (for a review, see Gillin et al. 1984).

Results from animal studies support the hypothesis that REM sleep is promoted by cholinergic cells located mainly in the pontinoreticular formation which show increased activity during REM sleep and that REM sleep is inhibited by noradrenergic and serotonergic neurons located in the locus coeruleus and in the raphe nuclei respectively, which show decreased activity during this sleep stage (Hobson et al. 1975; Hobson et al. 1986). Thus, the finding of shortened REM latency can be interpreted as an indicator of a cholinergic hyperactivity or a hypoactivity of the aminergic neurotransmitter system. A hypothesized central nervous serotonergic deficit is postulated in OCD patients. This deficit should lead to an attenuated inhibition of REM sleep and a shortening of REM latencies in the sleep EEG.

The aim of the present study was to investigate in a large sample whether patients suffering from obsessive-compulsive disorder show REM sleep abnormalities under baseline conditions. The results of such a study could contribute to a better understanding of the psychobiological aspect of the disorder.

Patients and methods

Selection of subjects

We investigated 22 patients suffering from an obsessive-compulsive disorder according to DSM-III-R (age 38.7 ± 11.0 years, 12 females, 10 males). All patients were inpatients of our clinic. Diagnoses were established by means of a structured clinical interview, the SCID (Spitzer et al. 1984; German translation by Wittchen et al. 1988). The Yale-Brown Obsessive Compulsive Scale (Y-BOCS) Goodman et al. 1989b,c; German translation by Büttner-Westphal and Hand 1991) rating had to exceed 16 points. In no case was the obsessional disorder secondary to affective illness or schizophrenia. In addition to the obsessive-compulsive disorder which in any case was the principal diagnosis which led to admission at the hospital, seven patients suffered from a secondary major depressive disorder according to DSM-III-R. The depressive syndrome was considered secondary if the depressive symptoms developed in the course of the underlying obsessive-compulsive disorder and did not precede the OCD symptoms according to the clinical judgement. Additionally to the principal diagnosis of OCD, one patient fullfilled diagnostic criteria of a current generalized anxiety disorder according to DSM-III-R.

Depressive mood was evaluated by means of the 21-item Hamilton scale (Murphy et al. 1982) with a mean Hamilton score of 17.9 ± 9.1, and obsessions and compulsions were rated by means of the 10-item Y-BOCS with a mean value of 25.2 ± 6.4. Anxiety was rated by means of the Clinical Anxiety Scale (CAS) (Snaith et al. 1982) with a mean value of 10.1 ± 5.0.

All patients had been drug-free for at least 7 days with a mean drug-free period of 14.6 ± 6.7 days. Seven patients had never received any psychoactive medication before, in four patients the drug-free interval had been longer than one year. Table 1 displays the psychoactive drugs taken by the eleven patients prior to entering the study and the drug-free period before being investigated by means of the sleep EEG. Prior to the sleep EEG recordings, all patients had been controlled by drug screening.

Table 1. Psychoactive medication taken by the patients prior to inclusion in the sleep-EEG recording ($n = 11$)

Psychoactive drugs (dose/day)	Drugfree period mean ± SD
Patient 1: Sulpiride 50 mg	28
Thioridazine 25 mg	23
Patient 2: Fluoxetine 20 mg	19
Patient 3: Clomipramine 150 mg	10
Chlorprothixene 50 mg	10
Patient 4: Fluspirilene 6 mg	23
Patient 5: Oxazepam 0.5 mg	17
Fluvoxamine 150 mg	17
Sulpiride 200 mg	17
Patient 6: Clomipramine 75 mg	10
Diazepam 0.25 mg	10
Patient 7: Oxazepam 20 mg	21
Chloral hydrate 2000 mg	14
Doxepine 50 mg	8
Patient 8: Doxepine 50 mg	8
Patient 9: Clozapine 400 mg	7
Patient 10: Dipotassium clorazepate 30 mg	12
Patient 11: Fluvoxamine 100 mg	24
Whole patient group ($n = 11$)	14.6 ± 6.7

Three female patients were on hormonal contraceptives at the time of the sleep EEG recording.

Before being included in the study, a careful physical examination, an electroencephalogram, an electrocardiogram, routine blood tests and computer tomography of the brain revealed no abnormal findings. All patients gave their written consent prior to being included in the study.

Healthy volunteers

Twenty-two healthy subjects were age-matched to the patient group and investigated under the same study conditions. 10 were female, 12 were male, the mean age was 38.6 years ± 13.0. There was no significant age difference between the patient group and the healthy volunteers ($t = 0.02$, $P \leq 0.98$). Before being included in the study, all subjects were carefully screened for physical and mental health. Each subject underwent a physical examination, an electroencephalogram, an electrocardiogram, drug screening, routine blood tests and an extensive psychiatric interview. Only subjects who were free of medical and psychiatric disorders and had no family history of psychiatric disorders were included. Only subjects with a regular sleep-wake cycle similar to the lights out (11.00 p.m.) and lights on (7.00 a.m.) schedule in our laboratory were included. All subjects received detailed information about the experimental procedures and gave their written consent. They were paid for their services.

Sleep recordings

Sleep was recorded between lights out (11.00 p.m.) and lights on (7.00 a.m.) by means of standard procedures. Horizontal EOG, submental EMG, EEG (= C3–A2, C4–A1) were recorded at a pa-

per speed of 10 mm/sec and scored blindly by two experienced raters according to standardized criteria (Rechtschaffen and Kales 1968). Interrater reliability for sleep recordings in our sleep laboratory is checked bimonthly. Coefficients of agreement vary between 0.80–0.90. After one adaptation night the subsequent night was evaluated for sleep variables.

Records were evaluated for the following sleep parameters:

1. Sleep efficiency – ratio of total sleep time (TST) to time in bed(TIB) × 100%.
2. Stages wake, 1, 2, 3, 4, slow wave sleep (SWS, stages 3 and 4 combined), and REM, all expressed in percent of sleep period time (SPT = time from sleep onset till final awakening).
3. Latencies, i.e. time from the beginning of the record to the first epoch of stage 2 (sleep onset latency) and from sleep onset to the first epoch of stage REM (REM latency) in minutes. In addition, a strict definition was adopted for REM latency, i.e. the first REM period must be at least 3 min in length (REM latency 3 min).
4. Number of awakenings during SPT (at least one epoch of stage wake).
5. Early morning awakening (EMA) in minutes, that is, length of time between the last epoch of stages 2, 3, 4, and REM occurring during the record and lights on in the morning.
6. REM density for each REM period and for the whole night (REM density is defined as the ratio of 3-s mini-epochs including eye movements to the total number of 3s mini-epochs per REM period × 100%).

Hamilton, Y-BOCS and CAS were correlated to sleep variables to determine the impact of psychopathology on sleep. To determine the impact of a secondary depression on sleep variables, the 7 OCD patients suffering from a secondary major depressive disorder according to DSM-III-R were compared to 7 age-matched OCD patients from our sample not suffering from a secondary depressive syndrome. The mean age of the OCD patients with secondary depression was 36.1 ± 10.7 years (2 were male, 5 were female). The mean age of the control group was 38.1 ± 14.6 years (4 males, 3 females). Furthermore, the whole sample was split into 2 groups according to the Hamilton rating: 1 group showing Hamilton ratings < 18 (n = 12, 6 females, 6 males, mean age 38.8 ± 11.0), the other group showing Hamilton ratings ≥ 18 (n = 10, 4 males, 6 females, mean age 38.5 ± 11.7). There were no significant differences between the groups concerning age.

Statistics

For descriptive statistics of sleep variables, mean and standard deviation (SD) were calculated. For comparison between the group of patients and the control group a t-test (two-tailed) was chosen. In addition, a Pearson correlation coefficient was used to correlate sleep variables and rating scores on depression, obsessional and anxiety inventories for each of the patients with OCD. The level of significance for all calculations was set at 5%.

Results

Table 2 displays the sleep variables for obsessive-compulsive patients and normal subjects. Concerning sleep continuity, sleep efficiency was significantly lower and wake % SPT was significantly increased in the patient group compared to healthy controls. Furthermore, the number of awakenings and early morning awakening (EMA) were increased in the patient group compared to the healthy controls. Nevertheless, neither result reached the level of significance. Sleep architecture did not differ between the two samples. Likewise, REM sleep measures, in particular REM latency, did not differ between the groups. Only the duration (min) and the density (%) of the first REM period were increased, without reaching the level of significance.

Table 2. Sleep variables of patients with OCD (n = 22) compared with healthy controls (n = 22). Mean ± SD, t-tests (two-tailed)

	Controls	OCD	t	P
Sleep continuity				
Sleep period (min)	442.4 ± 23.3	440.3 ± 28.0	0.27	0.792
Sleep efficiency (%)	92.1 ± 5.4	86.9 ± 9.4	2.23	0.033
S2-latency (min)	20.0 ± 18.5	21.8 ± 17.3	–0.24	0.809
Time awake %SPT	3.2 ± 3.6	7.1 ± 8.3	–2.04	0.050
No. awakenings	9.4 ± 6.0	14.5 ± 10.5	–1.95	0.060
EMA (min)	2.6 ± 5.5	9.0 ± 13.7	–2.00	0.055
Sleep architecture				
Stage 1 %SPT	4.1 ± 2.2	5.0 ± 3.0	–1.10	0.277
Stage 2 %SPT	58.6 ± 7.5	54.3 ± 8.5	1.74	0.089
Stage 3 %SPT	7.0 ± 5.3	6.3 ± 5.2	0.43	0.670
Stage 4 %SPT	2.4 ± 3.8	2.0 ± 4.9	0.29	0.772
SWS %SPT	9.5 ± 8.1	8.4 ± 8.9	0.42	0.680
REM sleep				
Rem latency (min)	67.9 ± 25.5	61.0 ± 26.2	0.89	0.380
REM latency (3 min)	88.6 ± 46.8	78.0 ± 53.9	0.70	0.491
REM %SPT	23.7 ± 5.1	24.0 ± 6.3	–0.16	0.873
REM density (% total)	23.1 ± 10.1	26.7 ± 10.2	–1.16	0.252
Duration 1st REM (min)	16.0 ± 9.0	26.0 ± 21.7	–1.99	0.057
Density 1st REM (%)	17.4 ± 10.8	23.9 ± 14.6	–1.75	0.088
1st REM interval (min)	83.8 ± 33.7	85.2 ± 20.4	–0.16	0.870
Duration 2nd REM (min)	27.7 ± 15.3	24.7 ± 11.5	0.72	0.474
Density 2nd REM (%)	23.4 ± 12.9	27.0 ± 12.0	–0.96	0.342
2nd REM interval (min)	72.1 ± 20.0	76.4 ± 20.1	–0.69	0.496
Duration 3rd REM (min)	32.5 ± 14.2	31.3 ± 17.5	0.25	0.804
Density 3rd REM (min)	24.1 ± 11.0	28.2 ± 13.1	–1.12	0.269
3rd REM interval (min)	58.0 ± 12.7	63.6 ± 25.4	–0.86	0.398

Sleep variables were correlated with rating data for depression (Hamilton Rating), obsession and compulsions (Y-BOCS) and anxiety (CAS) in all patients suffering from OCD. No positive correlation could be found between sleep variables and any of these rating scores.

To investigate the impact of a secondary depression on sleep variables, severity of obsession and compulsions and anxiety, we compared the subgroup of seven OCD patients suffering from a secondary major depressive disorder according to DSM-III-R to 7 age-matched OCD patients from our sample not showing a secondary depression. The patient group with a secondary major depressive disorder showed significantly higher scores on the Y-BOCS (30.4 ± 3.1) compared to the OCD patients not showing a depression (22.0 ± 7.6) (t = 2.7, $P \leq 0.05$). Additionally, they showed higher scores in the anxiety ratings (CAS: 14.6 ± 3.7 vs. 5.9 ± 4.1; t = 4.2, $P \leq 0.001$). Regarding the sleep variables, no significant differences could be found between the two patient groups (Table 3).

The same was true when subdividing the patient sample into one patient group with Hamilton score ≥ 18 and another patient group with a Hamilton score < 18. The patients with Hamilton Scores ≥ 18 showed higher ratings

Table 3. Sleep variables of OCD patients with a secondary major depressive disorder ($n = 7$) compared with OCD patients without a secondary major depressive disorder ($n = 7$)

	Without MDD	With MDD	t	P
Sleep continuity				
Sleep period (min)	454.4 ± 29.5	448.5 ± 18.8	0.44	0.667
Sleep efficiency (%)	90.1 ± 11.9	85.7 ± 7.8	0.82	0.431
S2-latency (min)	12.0 ± 12.1	20.8 ± 15.5	–1.18	0.263
Time awake %SPT	6.7 ± 10.9	9.1 ± 8.7	–0.45	0.660
No. awakenings	11.8 ± 7.6	17.2 ± 10.7	–1.09	0.299
EMA (min)	4.8 ± 9.0	5.8 ± 9.0	–0.21	0.840
Sleep architecture				
Stage 1 %SPT	4.1 ± 2.6	4.9 ± 2.1	–0.64	0.533
Stage 2 %SPT	55.5 ± 9.4	56.5 ± 5.0	–0.24	0.818
Stage 3 %SPT	5.3 ± 4.1	3.0 ± 3.8	1.09	0.299
Stage 4 %SPT	2.5 ± 6.6	1.0 ± 2.6	0.57	0.585
SWS %SPT	7.8 ± 9.5	4.0 ± 6.3	0.89	0.393
REM sleep				
Rem latency (min)	57.3 ± 14.4	66.7 ± 32.2	–0.66	0.529
REM latency (3 min)	72.7 ± 37.9	81.5 ± 69.1	–0.29	0.776
REM %SPT	24.9 ± 5.8	24.6 ± 7.2	0.09	0.934
REM density (% total)	26.3 ± 9.7	28.4 ± 9.9	–0.39	0.701
Duration 1st REM (min)	16.7 ± 7.3	26.1 ± 13.0	–1.65	0.131
Density 1st REM (%)	20.2 ± 9.7	29.2 ± 14.3	–1.38	0.197
1st REM interval (min)	75.9 ± 18.0	76.2 ± 7.8	–0.05	0.963
Duration 2nd REM (min)	22.7 ± 6.9	18.8 ± 7.7	1.00	0.339
Density 2nd REM (%)	26.9 ± 10.3	29.7 ± 10.7	–0.50	0.625
2nd REM interval (min)	69.3 ± 14.9	79.1 ± 27.0	–0.84	0.422
Duration 3rd REM (min)	26.9 ± 10.9	43.2 ± 23.7	–1.65	0.135
Density 3rd REM (min)	26.3 ± 14.9	30.8 ± 12.0	–0.62	0.550
3rd REM interval (min)	69.9 ± 39.4	63.5 ± 20.2	0.37	0.718

Table 4. Sleep variables of OCD patients with HAMD scores < 18 ($n = 12$) compared with OCD patients with HAMD scores ≥ 18 ($n = 10$)

	HAMD < 18	HAMD ≥ 18	t	P
Sleep continuity				
Sleep period (min)	435.2 ± 30.3	446.2 ± 25.2	–0.91	0.374
Sleep efficiency (%)	90.0 ± 8.4	83.2 ± 9.5	1.74	0.098
S2-latency (min)	20.3 ± 20.3	22.6 ± 13.7	–0.32	0.755
Time awake %SPT	4.4 ± 4.8	10.3 ± 10.5	–1.63	0.128
No. awakenings	14.2 ± 11.2	14.8 ± 10.1	–0.12	0.905
EMA (min)	7.3 ± 13-3	11.0 ± 14.6	–0.62	0.545
Sleep architecture				
Stage 1 %SPT	5.5 ± 3.4	4.5 ± 2.6	0.73	0.472
Stage 2 %SPT	52.5 ± 10.1	56.5 ± 5.8	–1.16	0.259
Stage 3 %SPT	8.2 ± 4.8	4.1 ± 4.9	1.94	0.067
Stage 4 %SPT	3.1 ± 6.2	0.7 ± 2.2	1.25	0.232
SWS %SPT	11.4 ± 9.8	4.9 ± 6.4	1.87	0.077
REM sleep				
Rem latency (min)	59.0 ± 26.3	63.3 ± 27.3	–0.37	0.715
REM latency (3 min)	70.0 ± 36.8	87.7 ± 70.3	–0.72	0.486
REM %SPT	25.0 ± 6.0	22.8 ± 6.9	0.78	0.446
REM density (% total)	26.4 ± 10.3	27.1 ± 10.5	–0.16	0.878
Duration 1st REM (min)	30.3 ± 26.3	20.9 ± 13.9	1.07	0.299
Density 1st REM (%)	22.1 ± 15.5	26.0 ± 14.0	–0.63	0.539
1st REM interval (min)	83.0 ± 19.1	87.9 ± 22.6	–0.54	0.595
Duration 2nd REM (min)	25.1 ± 9.8	24.3 ± 13.7	0.16	0.877
Density 2nd REM (%)	27.2 ± 13.8	26.9 ± 10.2	0.04	0.965
2nd REM interval (min)	75.2 ± 19.1	77.6 ± 22.2	–0.27	0.792
Duration 3rd REM (min)	26.5 ± 10.4	37.1 ± 22.7	–1.36	0.198
Density 3rd REM (min)	27.2 ± 12.2	29.5 ± 14.6	–0.40	0.691
3rd REM interval (min)	56.5 ± 15.5	72.3 ± 33.1	–1.33	0.211

on the Y-BOCS (28.8 ± 5.1 vs. 22.3 ± 6.0, t 2.7, $P \leq 0.01$) and higher scores on the CAS (12.6 ± 4.6 vs. 8.1 ± 4.6, t - 2.3, $P \leq 0.05$). No significant difference could be found concerning sleep variables comparing both patient groups (table 4).

Discussion

The principal aim of this study was to investigate whether patients with OCD can be differentiated by means of the sleep EEG from healthy controls and whether these patients show sleep EEG changes, especially REM sleep abnormalities, similar to those shown by depressed patients. In depressed patients the REM sleep disinhibition at the beginning of the night is currently interpreted as an inbalance of cholinergic/aminergic neurotransmitter systems (Berger et al. 1989; Gillin et al. 1984). While in depression a hyperactivity or receptor supersensitivity of the cholinergic system or a hypoactivity of the aminergic system is assumed, a serotonergic deficit is postulated in obsessive-compulsive disorder (Insel et al. 1983, 1984; Charney et al. 1988; Goodman et al. 1990). Experimental studies in animals demonstrated the importance of the serotonergic system for the regulation of REM sleep. It is well documented that serotonergic "REM off" neurons located in the dorsal raphé nuclei inhibit cholinergic "REM on" neurons located mainly in the giganto-cellular field of the pontine reticular formation (for overview, see Steriade and McCarley 1990). According to the reciprocal interaction model of NON-REM/REM-sleep regulation (Hobson et al. 1975; Hobson et al. 1986), a central nervous deficit of the serotonergic system may lead to shortening of REM latency by relative hyperactivity of the cholinergic system.

In contrast to this hypothesis, the sleep EEG of OCD patients did not show the postulated abnormalities. Under drug-free conditions, the sleep of patients with OCD was slightly impaired in comparison to that of healthy probands, showing a significantly lowered sleep efficiency and increased wake % SPT. This finding is in line with data reported by Insel et al. (1982) and Walsleben et al. (1990). Apart from these rather unspecific differences which may be due to stronger irritability of patients regarding the sleep-recording procedure, no significant abnormalities could be found compared to the group of age-

and sex-matched healthy subjects. Especially REM sleep and slow wave sleep were unchanged in comparison to the controls. These findings contradict the results of an earlier study done by Insel et al. (1982), who reported a decrease of slow wave sleep and shortening of REM latency in obsessive-compulsive patients showing many similarities to the sleep of patients suffering from primary depression. The authors mentioned that in contrast to depressed patients, who showed a reduction of REM latency in the first as well as in the subsequent night, the OCD patients had normal REM latencies in the adaptation night and showed a reduction of REM latency in the second night. Nevertheless, as in our study only the post-adaptation night was evaluated, this "first night effect" for REM latency cannot account for the controversial results of these two studies.

Another explanation for the discrepant findings could be differences concerning age. It is well known that age has a strong influence on REM sleep parameters. Nevertheless, the patient sample investigated by Insel and coworkers (1982) was younger (mean age 36.4 ± 4.2 years) than our patient sample (mean age 38.7 ± 11.0 years). As REM sleep abnormalities in depressed patients become more pronounced with increasing age (Riemann et al. in press; Gillin et al. 1981; Lauer et al. 1991), our older sample should have shown more pronounced REM sleep abnormalities compared to the younger patient group if these could have been expected at all in view of the slight age difference. This was not the case.

A further explanation for the discrepant findings concerning REM sleep in our study compared to the data reported by Insel and co-workers in 1982 could be the different assessment of diagnoses. In contrast to the latter study, a structured clinical interview (SCID) was used to establish the diagnoses of OCD. Thus, the use of the structured clinical interview according to DSM-III-R may have strengthened the validity of the OCD diagnoses, excluding all patients suffering from a primary affective disorder. In fact, Insel et al. reported that some of their patients had family histories of affective disorder in first-degree relatives, and one patient had a history of a circumscribed episode of major depressive disorder without obsessive symptoms several years before the sleep EEG investigation. Thus, it cannot be ruled out that some of the patients investigated by Insel et al. suffered from a primary depression.

Another problem which has to be discussed is whether psychoactive drugs taken prior to the sleep EEG recordings may have influenced the sleep EEG variables. All patients investigated in present study were drug-free for at least 7 days with a mean drug-free period of 14.6 ± 6.7 days. However, several studies have demonstrated that a one-week drug wash-out period is sufficient to exclude confounding effects of prior psychoactive medication on EEG sleep variables (Berger et al. 1983; Lauer and Pollmächer 1992). Thus, a confounding drug effect on the sleep EEGs of OCD patients in the present study can largely be ruled out.

The results of our study are in line with another recent study in a smaller patient group which also found normal REM latencies under baseline conditions in patients with OCD (Walsleben et al. 1990).

No significant association was found between psychometric tests and sleep variables. A secondary depression did not have any impact on sleep in OCD patients. This result is in accordance with earlier studies done by our group which did not find an association between REM sleep abnormalities and a secondary depression in patients suffering from anxiety and schizophrenic disorders (Hohagen et al. 1991). A secondary major depressive disorder was only associated with a more pronounced intensity of the obsessive and compulsive disorder. Patients with a secondary depression according to DSM-III-R or with Hamilton ratings ≥ 18 showed significantly higher ratings on the Y-BOCS compared to patients not presenting a secondary depressive syndrome.

The question has been raised whether there is a psychobiological relationship between OCD and primary depression (Insel et al. 1984). Some of the features of the syndrome overlap with those found in primary affective disorder. Depression, often to a severe degree, may frequently complicate OCD. Dexamethasone non-suppression and blunted growth hormone response to clonidine have been found in depressed patients as well as in patients suffering from OCD (Insel et al. 1984). On the other hand, OCD shows an earlier (childhood) onset and a chronic course in contrast to the adult onset and episodic course of primary depression. Furthermore, the sex distribution and the response to pharmacological treatment are different in OCD and primary affective disorder (Goodman et al. 1990). The sleep EEG is a useful tool to investigate whether there is a biological link between different psychiatric disorders (Berger et al. 1989). The results of our study contradict that OCD patients show REM and slow wave sleep abnormalities similar to those of patients suffering from primary depression. Our findings strongly support the view of a nosological boundary between OCD and primary depression.

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