Sleep Pattern in Patients with OCD

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Abstract

The aim of this work was twofold: to study polysomnographic findings in patients suffering from Obsessive Compulsive Disorder (OCD), in an attempt to clarify if there are sleep variables specific for OCD; and to determine the possible relationship of OCD to other psychopathological disorders, with special emphasis on Major Depressive Disorder (MDD). Nineteen patients suffering from OCD, were assessed by means of standardized sleep questionnaire, the Yale Brown Obsessive Compulsive Scale (Y-BOCS) for the assessment of severity of obsessive compulsive symptoms, and all night polysomnography. Our results revealed a significant decrease in sleep efficiency index (SEI), REM per cent, REM latency, and SWS latency among OCD patients. Those results indicate that depression has no effect on severity of OCD and that the co-morbidity of depression with OCD has no significant effect on the sleep measure. The study concludes that sleep changes recorded in patients with OCD can be primary in origin and are independent of an associated major depressive disorder.

Introduction: The study of Obsessive Compulsive disorder is an example of the positive impact of modern research on mental disorders. As recently as the 1980s, Obsessive Compulsive disorder was considered an uncommon disorder, hardly responsive to treatment. It is now recognized that it is more prevalent that previously believed and that it may be very responsive to treatment (Kaplan et al., 1994). Life time prevalence rated of OCD vary considerably between countries. The lowest rates were observed in Taiwan (Yeh et al., 1985) varying between 0.5 and 0.9% and in India (Khanna et al., 1993), where the rates were 0.6%. In north and Central America, the lifetime prevalence rates ranged between 2.6 and 3.2% (Degonda et al., 1993), while the ECA study (Karon and Gording, 1991) found a one year prevalence rate of 1.65% for OCD. Okasha (1990) found that the prevalence rate of OCD, in a psychiatric out patients Egyptian study, was 2.3%.

The international (ICD-10) and American (DSM-IV) classification systems are sufficiently similar, so as to expect substantial concordance between diagnosis based on the two systems (Foa & Kozak, 1994). The two nosologies are on the whole convergent for OCD (Foa & Kozak, 1994).

The introduction of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), like other OCD rating scales, provided a specific measure of the severity of OCD symptoms of OCD, that is not influenced by the type of obsessions and compulsions present. Thus, Y-BOCD provides a structured tool for the phenomenological study of OCD symptoms (Okasha, et al., 1994).

Over the past decade psychopharmacological studies suggested a novel strategy for investigating the possible etiology of Obsessive-Compulsive disorder (Zohar&Insel 1987a), and a new method of treatment (Insel&Murhpy, 1981).

The major hypothesis regarding the pathophysiology of this disorder postu-

lates an involvement of 5-hydroxyl typtamine or serotoin (5-HT), on the basis of an almost exclusive response of OCD patients to 5-HT reputake inhibitors, while tricycle antidepressants, with less marked activity on 5-HT uptake, are less effective (Zohar&Insel 1987). However, because about 40% of patients do not respond to SRIs, it is possible that other mechanisms may be involved (Goodman et al., 1990). Moreover, SRIs are also effective in other psychiatric disorders, and this shared response raises the issue of the specificity.

Recently, a number of biological research was conducted in an attempt to identify biological mechanism specific for the disorder and the possible relationship of OCD to other forms of psychopathology, mainly to major depressive disorder and anxiety disorders.

The search for biological markers in OCD is a relatively new approach, as until recently, OCD has been considered to be of psychological origin. Nevertheless, this approach turned out to fruitful (Weizman et al., 1991). Eight types of biological markers were assessed in patients with OCD(Weizman et al., 1991): Dexamethasone Suppression test (DST), Rapid Eye Movement (REM) latency and density, growth hormone response to clonidine, platelet 3H-imiprammine binding, platelet sertonin uptake, cerebrospinal fluid (CSF) measure of 5-hydroxyl-indolactic acid(5HIAA) and 3-methoxy-4-hydroxyl phenyl-glycol (MHPG).

The special emphasis on the relation of OCD to major depressive disorder has been on a number of biological investigations that pointed to a similarity in pathophsiology between the two disorders.

Several of those investigations used the Dexamethasone Suppression Test (DST). In a study carried out in a mixed population of non depressed inpatients and outpatients affected by OCD, about 25% had abnormally high cortisol levels following administration of 1 mg of Dexamethasone given at 11 p.m. (Insel et al, 1982). a 25% incidence of nonsuppersion approximates the sensitivity of the DST for endogenous depression in some centers and is somewhat higher than the incidence of DST nonsuppresion in normals. However, the significance of this test in OCD is diminished, as two report described normal DST responses in OCD (Monterio et al., 1986).

Along another dimension, a study carried out on drug free OCD patients showed that nine of a total of 10 exhibited decreased TSH response (Weizman et al., 1988). The blunted TSH response to TRH stimulation indicated some dysfunction of the hypothalamic-pituitary throid axis, and suggested a link between OCD and mood disorders. However, reduced TSH response to THR has also been found in other neuropsychiatric disorders, including panic disorders (PD), eating disorders, personality disorder, mania, alcoholism, schizophrenia, and cocaine abuse (Khan et al., 1988), which again questions the specificity of this test.

The clondine-stimulated release of growth hormone (GH) allows us to evaluate dynamically the status of alpha 2-adrenorceptor. In our study of nine OCD patients and nine healthy volunteers, the patients showed a significant blunted GH response following this challenge with clonidine; GH increase of greater than 3 ng/ml following clonidine were observed only in two (out of nine) patients with OCD, compared with five (out of nine) normal controls (Siever et al., 1983). However, baseline GH levels were not significantly difference between the two

groups. Similar to the DST, the issue of specificity of the blunted GH response remains controversial, as non depressed postmenopasusal women and schizaffective patients also show attenuation similar to that seen among depressives (Siever et al., 1983).

Using the somatosensory averaged evoked response, Shagass and colleagues (1980) have reported an abnormal pattern in the middle latency waves of the somatosensory averaged evoked response (AER) that appears specifically in OCD and not in other psychiatric disorders, including depression (Shagass et al., 1980). In summary, four out of eight biological markers studied in MMD and OCD are similar in both conditions, three are not and one is doubtful.

The four biological markers that are similar are DST non suppression, blunted growth hormones response to clonidine, decreased REM latency and decreased platelet imipramine binding. However, two out of the four biological markers that are similar in OCD and MDD are coupled to dissimilar findings. In MDD the increased RE density is coupled with decreased REM latency, while in OCD the decrease in REM latency is not associated with alteration in REM density. Similarly the decrease imipramine binding in MDD is accompanied by reduced serotonin uptake, whereas in OCD the reduced imipramine binding is not accompanied by diminished sertoin uptake.

This lack of correlation between the changes in REM latency and density on the one hand and imipramine binding and serotonin uptake on the other hand in patients with OCD further emphasizes the biological difference, rather then the similarity, between MDD and OCD.

The other similar biological markers are neuroendocrine biological markers (DST, GH response to clonidine and TSH response to TRH). All are present in a variety of psychiatric and non-psychiatric conditions and thus do not appear to be specific. Another finding, the elevates GFS 5-HIAA level in patients with OCD, is opposite to the reduced or normal CFS 5-HIAA level reported in MDD.

Insel and Coworkers (1982), studied the sleep pattern of 14 OCD patients with all-night EEG recording, and found that most of the sleep difficulties were subjectively connected with compulsive activities at night.

Most of the sleep EEG studies found that patients with OCD had decreased REM latency compared to age-matched normal controls and depressed patients, although in a study by Insel et al., (1982), the OCD patients were significantly less depressed than the primary affective group.

Sleep abnormalities have also been reported in other psychiatric disorders including anxiety disorders, schizophrenia, alcoholism and dementia. the REM latency has been investigated with varying results: some studies have reported reduced REM latency in depression, schizophrenia, OCD, borderline personality disorder (Benca et al., 1992). However, the specificity of shortened REM latency for depression remains controversial (Weizman et al., 1991).

This paper will be concrned only with the sleep electroencephalograph as a possible marker in patients with OCD. There is a paucity of information regarding the subjective and objective sleep of patients with Obsessive Compulsive disorder . The triad of short REM latency, increased REM density and disturbed temporal distribution of REM has been suggested as being specific to endogenous depressive illness (Vogel, 1981).

Biological Markers in Obsessive-Compulsive Disorder (OCD) and Maor Depressive Disorder (MDD) (Weizman et al., 1991)

Markers	MDD	MDD	Similarity of OCD to MDD
Endorinal DST nonsuppression	(1)+	(2,3,4)+ (5)-	+
Blunted growth hormone response to clonidin	(6)+	(7)+	+
Blunted TSH response to TRH	(8)+	(9)+	+
Electrophysiologic Decresed REM latency Increased REM density	(10)+ (12)+	(11)+ (11)-	+ -
Platelet Reduced 3H-imipramine binding	(13)+	(14)+	+
Reduced serotonin uptake	(15)+	(14,15)-	-
CSF amine metabolites 5-HIAA increased MHPG decreased	(16)- (18)±	(15)+ (17)± (17)-	- ±

1. Carroll et al., 1981; 2. Insel & Goodwin, 1983; Cottraux et al., 1994; 4. Asberg et al., 1982; 5. Monterio et al., 1986; 6. Siever & Uhde, 1984; 7. Siever et al., 1983; 8. Loosen & Prange 1982; 9. Weizman et al., 1988; 10. kuffer et al., 1978; 11. Insel et al., 1982; 12. Foster et al., 1976; 13. Meltzer et al., 1983; 14. Weizman et al., 1986; 15. Insel et al., 1985; 16. Van Praag, 1982; 17. Thoren et al., 1980; 1. Jimerson & Berrettini, 1985.

Aim of work

Studying the polysomnographic findings in patients suffering from obsessive compulsive disorder , in an attempt to identify any possible OCD-specific sleep and to clarify the possible relationship of OCD to other forms of psychopathoogy, mainly to major depressive disorder.

Material and Method

1. Sample

a- Nineteen patients suffering from OCD and fulfilling the diagnosis criteria ICD-10). All patients were free from any psychotropic medication for at least two weeks before the assessment. Patients with physical disorder that may have an effect on sleep, as well as patients having other psychiatric diagnosis and patients

with sleep apnea syndrome were excluded from the study.
b- A control group of 18 subjects matched for age and sex with the OCD group, with no history of psychiatric or medical illness.
Assessment:
a- All diagnosis were based on the ICD-10 criteria.
b- Patients were assessed by means of the Arabic version of Y-BOCS for symptomatology and severity of OCD-symptoms. The Arabic version of Y-BOCS was translated by two senior psychiatrists at the Institute of Psychiatry, AIN Shams University.
c- Both patients and controls were subjected to a standardized sleep questionnaire (including an interview with relatives, physical examination as well as all night polysomnography,)PSG), by sleep Trace 2000. The first PSG assessment was a full-montage, measuring electroencephalogram (EEG), electro-occulogram (EOG), respiratory events, oxygen saturation, electro-cardiogram (ECG), and body position.
d- Statistical analysis using the mean, standard deviation and T-test cases and control along different measured parameters. Correlation coefficient was used as a measure of correlation between the Y-BOCS and other measured parameters.

Results:

1. Demographic data and comorbidity:
Our sample included 18 OCD patients, 9 males(50%) and 9% females (50%) almost in a ratio of 1:1. Ages of the patients ranged between 21 and 51 years, with a mean age of 34.44±7.79 years. Five patients had a past history of depressive disorder, five other patients had a family history of depressive disorder, and one patient had both past history of depression and family history of depressive disorder, i.e. one third of patients has a co-morbid depressive disorder.

2- Y-BOCS scores
Severity rating showed that scores on the Y-BOCS obsessive subscale ranged between moderate to severe, with a mean value of [13.33±2.93]. Scores on the Y-BOCS compulsive subscale were moderate, with a mean value of[12.11±1.9]. The majority of patients (16/8=88.9%) were rated as moderate on the total Y-BOCS with a mean value of [26.00±2.87], while two patients were rated as severe on the total Y-BOCS with a mean value of [31.5±0.5] (Table 1).

3- Sleep questionnaire
Assessment by the sleep questionnaire revealed that sleep complaints were present in 6 patients (33%) , 5 of whom had difficulty in falling asleep as well as interrupted sleep and one had prolonged sleep hours. Only one patient attributed his sleep difficulty to a compulsive ritual of checking the doors and gas outlets.

4- Sleep EEG
Analysis of the sleep EEG in both patients and control groups showed that the sleep efficiency index (SEI) was significantly lower in patients than in the control group (the mean value was 61.28±8.64 for the patients and 77.06±8.13 for the control) ($p<0.001$). REM per cent (REM time) was significantly lower in patients than in the control group ($p<0.05$) (REM % was 22.65±3.39 in patients compared to 26.2±4.4 in the control group). Slow wave sleep (SWS) latency was significantly lower in patients than in the control group (p,0.05). (Table 2)

Regarding the REM latency, it was significantly lower in patients compared to the control group (p<0,.01 (REM latency as 61.57±9.71 in patients while it was 75.6±9.36 in the control.
Both stage I & stage II percentage were significantly higher in patients than the control group ($p<0.001$ and $p<0.05$ respectively.

Table (1) Profile of OCD patients (n=18)

Variable	Mean Value
Age	34.44±7.79
Y-BOCS	
Obsessive subscale	3.33±2.93 (moderate to severe)
Compulsive subscale	12.11±1.94 (moderate)
Total score	26.00±2.87 (moderate)
Sleep Variables	
Sleep Efficiency Index (SEI)	61.28±8.64
Stage I%	5.27±1.39
Stage II%	53.27±3.48
Stage III%	9.32±1.96
Stage IV%	10.85±2.71
SWS%	20.96±3.39
REM %	22.65±3.80
REM latency	61.57±9.71
SWS latency	50.25±12.83
Number of Arousals	19.87±9.07

Table (2) Sleep Measures in OCD Patients and Control Subjects

Sleep Variables	OCD patients Mean value	Controls Mean value	t	Level of Signficant
Sleep efficiency Index (SEI)	61.28±8.64	77.06±8.13	4.81	p<0.001
Stage I%	5.27±1.39	2.83±1.06	5.202	p<0.001
Stage II%	53.27±3.48	52.77±4.09	0.326	p>0.05
Stage III%	9.32±1.96	8.04±1.82	2.353	p<0.05
Stage IV%	10.85±2.71	10.79±2.98	0.053	p>0.05
SWS%	20.69±3.39	18.83±4.81	1.083	p>0.05
REM%	22.65±3.80	26.2±4.40	2.145	p>0.05
REM latency	61.57±9.71	75.6±9.63	3.683	p<0.01
SWS latency	50.25±12.83	61.2±14.74	1.971	p<0.05
No. of Arousals	19.78±9.07	11.4±4.84	3.187	p<0.01

p<0.05= insignificant; p>0.05= signficant; p<0.01-high signficant; p<0.001 very high signficant

NB: SEI, REM percentage; REM latency and SWS latency were signficantly lower in OCD patients than in control, while stageI and stageIII percentagees and the number of arousals were signficantly higher among OCD patients than controls.

Table(3) Correlation between obsessive, compulsive and total Y-BOCS scores and the age and sex of patients

Variable	Y-BOCS Obsessive subscale	Y-BOCS Compulsive subscale	Y-BOCS Total score
Age	0.4545 p<0.05 (Sig)	0.0979 p>0.05 (N.S.)	0.3814 p>0.05 (N.S.)
Sex	Mean a 14.7' 11.8 p<0.05 (Sig)	Mean a11.7' 12.4 p>0.05 (N.S.)	Mean a 27.6 '24.3 p<0.05 (Sig)

*Negative values mean inverse correlation, while positive values mean positive correlation
*p>0.05= non significant value (N.S.) * p>0.05= significant value

Table(4) Correlation between obsessive, compulsive and total Y-YBOCS value and sleep variables

Sleep Variable	Y-BOCS Obsessive subscale	Y-BOCS Compulsive subscale	Y-BOCS Total score
Sleep Efficiency Index (SEI)	-0.04520 p>0.05 (N.S.)	0.0332 p>0.05 (N.S.)	-0.7091 p>0.05(Sig)
Stage I%	0.5411 p>0.05(Sig)	-0.1846 p>0.05 (N.S.)	-0.3443 p>0.05 (N.S.)
Stage II%	0.2872 p>0.05 (N.S.)	-0.2722 p>0.05 (N.S.)	0.1941 p>0.05 (N.S.)
Stage III%	0.1465 p>0.05 (N.S.)	0.0522 p>0.05 (N.S.)	-0.3660 p>0.05 (N.S.)
Stage IV%	-0.5496 p>0.05(Sig)	-0.0662 p>0.05 (N.S.)	-0.03447 p>0.05 (N.S.)
SWS%	-0.2069 p>0.05 (N.S.)	-0.2112 p>0.05 (N.S.)	-0.3051 p>0.05 (N.S.)
REM%	-0.3718 p>0.05 (N.S.)	-0.1127 p>0.05 (N.S.)	-0.1294 p>0.05 (N.S.)
REM latency	-0.5091 p>0.05(Sig)	-0.2960 p>0.05 (N.S.)	-0.8479 p>0.05(Sig)
SWS latency	0.4974 p>0.05(Sig)	0.3231 p>0.05 (N.S.)	0.3147 p>0.05 (N.S.)
Number of Arousals	0.4834 p>0.05(Sig)	-0.006 p>0.05 (N.S.)	0.4477 p>0.05 (N.S.)

*Negative values mean inverse correlation, while positive values mean positive correlation
*p>0.05= non significant value (N.S.) * p>0.05= significant value

The patients had a higher number of arousal than the control group (p<0,01), and there was no significant difference in all sleep measures between both sexes (p>0.05).

5- Correlation between Y-BOCS and measured parameters

A significant correlation was found between the total Y-BOCS scores and female sex. As regards the Y-BOCS obsessive subscale scores, a significant correlation was found between age and female sex. No significant correlation was detected between the Y-BOCS compulsive subscale and any of those two variables. (Table 3)

Regarding the correlation between the Y-BOCS scores and the sleep measures, the correlation coefficient test showed a significant positive correlation between the Y-BOCS obsessive subscale on the one hand and the following sleep measure on the other. Stage I percent, SWS latency and the number of arousals (p<0.05). A significant inverse correlation was found between the Y-BOCS compulsive subsclae and stage IV percent and REM latency (p<0.05). No significant correlation was detected between the Y-BOCS compulsive subscale and any of the sleep measurements (p>0.05). As regard the total Y-BOCS scores, there was a significant inverse correlation with the sleep efficiently index (SEI) and the REM latency (p<0.05). (Table 4)

In order to assess the effect of depression on the sleep measures in patients with OCD the 18 patients were divided into 2 groups: OCD patients with depression (5 out of 18 = 28%) and OCD patients without depression (2 out of 18= 67%). Both groups were compared regarding the sleep measures. No significant differences were detected between the sleep measures in both groups (p>0.05) indicating that an associated de-

Table(5) Correlation between obsessive, compulsive andpresence of depression or family history of depression in OCD patients

Variable/Mean	Y-BOCS Obsessive subscale	Y-BOCS Compulsivesubscale	Y-BOCS Total score
Depression 5 out of 18	14.8±3.2	13.2±1.3	28.3.8
No Depression 13 out of 18	12.7±2.7 p>0.05 N.S.		25.2±2 c
Family history of Depression 5 out of 18	13.4±3.0	p>0.05 N.S.	26.4±1.3
No Family history of Depression	13.3±3.0 p>0.05 N.S.		25.8±3.3 p>0.05 N.S.

pressive disorder did not have an effect on the sleep measures of OCD patients. Assessment for the correlation between the severity rating scales on the total Y-BOS scores and its subscale on the one hand and the presence or absence of comorbid depression or the family history of depression in patients with OCD on the other hand revealed that both depression and family history of depression had no effect on the severity of OCD. (Table 5)

Discussion: The findings in the present study suggest that the sleep profiles in patient with OCD are different of those of normal subjects. Also, the study did not find any characteristics or specific complaints in OCD patients. Only 33% of those patients had sleep complaints, either interrupt sleep, difficulty to fall asleep or hypersomnia. Only one patient had attributed his insomnia to the compulsive rituals. This finding is contrary to that of the study of Insel et al., (1982), who found that most of the sleep difficulties of OCD patients were subjectively connected with compulsive activities. Also, they reported sleep complaints in about 79% of OCD patients.

The most significant characteristics of sleep EEG in patients with OCD were decreased sleep efficiency index (SEI) ($p<0.001$), decreased REM latency ($p<0.01$) and increased number of arousal ($p<0.01$). The above findings were matched with that of Rappaport et al., (1981) and Insel et al., (1982).

Hohagen et al., (1994) in their study of sleep EEG of patients with OCD, found that (SEI) was significantly lower and the numbers of arousals were significantly higher in the patient group compared to healthy subjects, which is compatible with the findings of our study. However, in contrast to our findings, they found that REM latency did not differ among the groups. The results of Hoghagen et al., (1994) contradict the assumption that OCD patients show REM sleep and slow sleep abnormalities similar to those shown by patients with primary depression.

The polysomnographic finding of this study showed increased stage I and II per cent. These findings are different from those obtained by Insel et al., (1982) and Rappoport et al., (1981) who found a significant increase in stage IV per cent in patients with OCD. Our study found no significant difference between patient and control group regarding this variable ($p>0.05$).

Our results support those of Rappoport et al., (1981) who found a significant decrease in REM latency time compared to the healthy group ($p<0.05$).

The changes in the sleep measures were not different between both sexes. These changes have been observed in OCD patients with and without sleep complaints, indicating that those changes are less likely to be considered as the result of sleep difficulty secondary to psychiatric sympotomatology.

The effect of OCD severity, as measured by the Y-BOCS, on the sleep measures did not show a significant correlation between the Y-BOCS compulsive subscale and the sleep measures ($p>0.05$). On the other hand there was a significant correlation between Y-BOCS obsessive subscale and Y-BOCS total scores and some of the sleep measures, namely SEI, number of arousal and REM latency. Our findings differ from other studies of sleep patterns in OCD patients as Insel et al., (1982) and Hogagen et al., (1994) mentioned that the severity had no relation to the sleep measures in OCD patients.

OCD patients who had comorbid depression disorder were compared with OCD patients without depression. Our results suggest that depression had no significant effect on sleep measures in OCD patients ($p>0.05$) and the same was found as regards the family history of depression. Rappoport et al., (1981) suggested that REM latency may be decreased in patients with OCD, independent of the coexistence of depressive sympotomatology. Insel et al. (1982) found significant decrease in REM latency in patients with OCD. However, they found that, the other REM indices (i.e. REM density, number of REM periods and the total REM time) were not significantly different between patients with OCD and normal control subjects. They mentioned that the presence of depressive symptoms did not account for the short REM latency in OCD.

Because the reduced REM latency found in OCD patients has been most often associated with major depression, the NIHM team compared their findings in OCD patients with a group of aged-matched patients with major depression. There were few significant differences between the two groups: they did not differ on REM latency, REM efficiency, number of REM periods and total REM time, although there was a trend for the obsessive-compulsive patients to have lower REM density compared with depressive patients.

Marassiti et al. (1994) on discussing the biological dissection of obsessive-compulsive disorder, summarized the sleep EEG studies on OCD as that patients with OCD resemble patients with major depressive disorder in only one variable i.e. decreased REM latency, while they are identical to normal subjects on the other variable, i.e. increased REM density.

In conclusion it seems that the sleep EEG studies in different psychiatric disorders confirm the recent concept about the sleep markers as having little specificity. It seems that the triad of REM latency, increased REM density and disturbed temporal distribution of REM has to be reconsidered again. No solitary sleep variable seems to single out as absolutely specific for any particular psychiatric disorder. What is to be considered here is the shared dysfunction in biological markers suggesting a common brain abnormality which might explain the similarity of symptoms across different disorders.

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أنماط النوم لدى المصابين باضطراب الوسواس القهرى

الهدف من هذه الدراسة ذو شقين: الأول، دراسة ما يمكن الكشف غنه باستخدام رسام النوم لدى المرضى الذين يعانون من باضطراب الوسواس القهرى، فى محاولة لتوضيح ما إذا كان هناك متغيرات خاصة بالنوم فى هذا الأضطراب ، الثانى تحديد العلاقات المحتملة بين اضطراب الوسواس القهرى و غيره من الأضطرابات النفسية خاصةا لإكتئاب الجسيم. و قد اختير ١٩ مريضا يعانون من اضطراب الوسواس القهرى تم تقسيمهم بواسطة مقياس بال-بروان(Y-BOCS)لتحديد درجة شدة اعراض الوسواس القهرى وكذلك بواسطة رسام النوم طيلة الليل. وقد بينت النتائج وجود انخفاض ملحوظ فى درجة النوم (REM) و فى النسبة المئوية لحركات العين السريعة (SEI)و فى درجة الكمون لحركات العين السريعة. و(SWS) لدى هؤلاء المرضى. و تدل هذه النتائج على أن ا لإكتئاب ليس له تاثير على شدة الوسواس القهرى و أن تزامل ا لإكتئاب و الوسواس القهرى ليس له تاثير على مقياس النوم. و انتهت الدراسةإلى تغيرات النوم المسجلة لدى مرضى الوسواس القهرييمكن أن تكون ذات طبيعة أولية و مستقلة عن أى اضطراب اكتئابى مصاحب لها.