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Monitoring of Antidepressant Response to ECT With Polysomnographic Recordings and the Dexamethasone Suppression Test

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Abstract. Ten patients treated with electroconvulsive therapy (ECT) only were followed with serial sleep polysomnographic recordings and dexamethasone suppression tests (DSTs). Both biological correlates of depression showed improvement with ECT. The use of serial sleep measures and serial DSTs in monitoring the clinical response to ECT is discussed.

Key Words. Major depressive disorder, electroconvulsive therapy, sleep electroencephalogram, dexamethasone suppression test.

Electroconvulsive therapy (ECT) continues to be one of the most effective forms of treatment for severe, psychotic, or medication-nonresponsive major depressive disorder (MDD) (*APA Task Force Report #14*, 1978). Although the use of ECT declined with the development of antidepressant medications, interest in this treatment modality has renewed in recent years (*Consensus Development Conference Statement*, National Institutes of Health, 1985). While the literature on biological correlates of affective disorders has grown in size and complexity, published studies of ECT patients are relatively few. This scarcity of studies concerning patients undergoing ECT probably reflects the practical difficulties of conducting research in severely ill patients. Nevertheless, some reports indicate that both the dexamethasone suppression test (DST) (Carroll, 1972; Dysken et al., 1979; Albala et al., 1981; Papakostas et al., 1981; Coryell and Zimmerman, 1983; Yerevanian et al., 1983; Nemeroff and Evans, 1984; Coryell, 1986; McAllister and Price, 1986; Grunhaus et al., 1987) and sleep electroencephalography (EEG) (Grunhaus et al., 1985; Hoffman et al., 1985) may be valuable in monitoring the antidepressant effects of ECT.

Initial reports were enthusiastic about the ability of the DST to monitor response

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to ECT, suggesting that favorable response to ECT would be accompanied by normalization of the DST (Carroll, 1972; Dysken et al., 1979; Albala et al., 1981). Those failing to respond to the treatment would continue to show the nonsuppressive response. More recent communications (Coryell, 1986; Grunhaus et al., 1987) have been tempered by caution, but do not rule the DST out as a potentially useful tool in monitoring ECT response.

Sleep polysomnography has also been studied extensively in affective disorders, and many changes believed to be characteristic of MDD have been described (Kupfer, 1976; Kupfer et al., 1978; Feinberg et al., 1982). These abnormalities appear to be more frequent in the severely depressed or psychotically depressed patients who are often referred for ECT. In these situations, patients may show even more disrupted sleep with frequent arousals and early morning awakening, very short rapid eye movement (REM) latency or sleep onset REM periods (SOREM), and high REM density (Thase et al., 1986). Surprisingly, very few modern studies have used serial studies of the sleep electroencephalogram (EEG) to monitor the progress of patients undergoing ECT. Grunhaus et al. (1985) and Hoffmann et al. (1985) have described the normalization of sleep EEG abnormalities in MDD patients after ECT.

The present report replicates and expands our previous study on the changes of sleep parameters with ECT. Interestingly, 2 of the current sample of 10 patients were considered nonresponders to treatment; these patients did not show normalization of the pretreatment abnormal biological correlates.

Methods

Ten inpatients (four males and six females, ages ranging from 44 to 77 with a mean of 61.5 years) from the Clinical Studies Unit of the University of Michigan Hospitals were included in the study. All were evaluated according to our standard protocol, which includes: (1) 12-14 days of drug-free observation; (2) several diagnostic interviews by senior psychiatrists and the complete Schedule for Affective Disorders and Schizophrenia (Spitzer and Endicott, 1975); (3) a diagnostic family interview corroborating anamnestic information; (4) a review of all past medical records as available; and (5) a complete medical and laboratory workup. At the end of the observation period, diagnosis was made by consensus of all clinicians involved using the Research Diagnostic Criteria (RDC) (Spitzer et al., 1978).

To be included in the study, patients were required to (1) meet the diagnosis of MDD, (2) have been free from psychotropic drugs for at least 12 days, (3) not have an illness or have received medications that would preclude the sleep-EEG recordings, (4) have been treated with ECT only without other medications than those required for the anesthesia/muscular relaxation process, and (5) have agreed to participate in serial sleep EEG recordings and blood drawing for the DST. The clinical indications for ECT were severe depression, presence of delusional features, and either previous poor response to antidepressant medications or previous good response to ECT.

Unilateral nondominant ECT was administered three times a week with a Medcraft instrument. Treatment parameters were set to achieve seizure activity (recorded with the cuff method) of at least 25-sec duration. Restimulation was performed whenever seizures were absent or too short. Standard anesthetic pretreatment included the use of a quaternary anticholinergic agent (glycopyrrolate), a short-acting barbiturate (methohexital), and a muscle relaxant (succinylcholine). The length of the ECT course was determined by clinicians without knowledge of the research data. No antidepressant, antipsychotic, or sedative hypnotic agents were administered concurrently with the treatments. One patient required 5 mg of diazepam

for post-ECT agitation; the post-ECT sleep recordings in this patient were performed 12 days from the last dose of minor tranquilizers.

Two consecutive nights of sleep were recorded following the drug-free period with subjects sleeping in their own hospital bed (patients 2 and 4 in Table 1 could tolerate only night 2 of recording). Placement of electrodes and scoring were done according to the method of Rechtschaffen and Kales (1968). The following definitions were applied when studying the sleep recordings: *time asleep* (TS-A): time (min) from sleep onset to end of sleep minus awake; *total recording period* (TRP): time (min) from beginning of recording ("lights out") to end of recording; *awake*: time (min) awake between sleep onset and end of sleep; *delta sleep*: stages III and IV; *early morning awakening* (EMA): time (min) awake between end of sleep and arising (end of recording); REM *activity* (RA): the sum of the eye movement scores during REM sleep for the entire night, based on a score of 0-8 for each min of REM sleep; REM *density*: REM activity/REM time (RA/RT); REM *latency*: time (min) from sleep onset to onset of stage REM; REM *latency minus awake* (RLMA): REM latency minus any time awake between sleep onset and REM onset (min); REM *percent* (REM%): RT/(TS - A) $\times$ 100%; REM *time* (RT): time (min) spent in stage REM; *sleep efficiency*: (TS - A)/TRP $\times$ 100%; *sleep latency*: time (min) from beginning of recording to sleep onset; REM *period*: a period containing at least three 1-min epochs of REM sleep, with not more than 30 min of non-REM sleep between any two epochs of REM; *sleep onset*: $\geqslant$ 3 min of continuous REM sleep, or 10 consecutive min of sleep with the first min being stage 2, 3, 4, or REM sleep and at most 1 min of time awake during this 10-min period.

Two nights of post-ECT sleep EEG recordings (patient 7 in Table 1 could tolerate only 1 night of recording) were performed following the same guidelines as the pre-ECT tracings, with the patient sleeping in his own bed and before beginning maintenance psychotropics. Post-ECT sleep recordings were done 1-4 days after the last ECT.

It is unclear whether the medications used during the ECT procedure affect sleep EEG recordings. Glycopyrrolate and succinylcholine do not cross the blood-brain barrier and therefore would not be expected to have central effects. Methohexital, on the other hand, could have definite effects on sleep EEG recordings. To circumvent this problem, we recorded our patients at least 36 hours after the anesthesia. Nevertheless, the possibility that repeated anesthesia, however brief, might affect sleep EEG recordings needs to be kept in mind.

The clinical status of the patients was rated weekly with the 17-item Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1960). We defined treatment outcome as follows: good response—final HRSD $\leqslant$ 10; poor response—final HRSD $>$ 10. Ratings were performed within 48 hours of the sleep recordings. Clinicians had no knowledge of sleep EEG results throughout the treatment period, and sleep EEG raters were requested to refrain from inquiring about the patients' clinical progress while they were performing and scoring the sleep EEG recordings.

The 1 mg overnight DSTs were performed following the guidelines of Carroll et al. (1981). All patients had been drug free for 10-14 days before the DST, and no exclusion criteria for the DST were present in any of the patients. Blood was drawn at 8 a.m., 4 p.m., and 11 p.m. the day after the administration of 1 mg dexamethasone. The results presented are the maximal postdexamethasone cortisol levels observed among the three samples. DSTs were performed on different days than the sleep recordings.

The pre- and post-ECT sleep EEG results were compared by paired t tests. One of the patients was a nonresponder, and another patient relapsed 3 weeks after the end of the ECT course in spite of appropriate tricyclic antidepressant maintenance. These two patients were considered nonresponders to treatment in the analysis.

Results

Table 1 shows the demographic, clinical, and laboratory information of the patients. All were diagnosed as MDD endogenous subtype according to the RDC, and three of them were also psychotic according to the RDC. The number of ECT treatments

Table 1. Sample description

Patient	Age (years)	Sex	Diagnosis	Pre-ECT scores HRSD	DST	REM L	REM D	Post-ECT scores HRSD	DST	REM L	REM D	No. ECTs
1[1]	61	F	MDD-ED Psychosis	25	10	21.5	4.0	3	3.7	74	2.3	14
2	60	F	MDD-ED	26	11	0	2.7	8	2.9	44	1.9	11
3	73	F	MDD-ED	30	2.5	27	4.1	8	4.8	45	2.8	8
4	77	F	MDD-ED	38	13.1	37.5	2.8	1	4.1	335	1.4	9
5[2]	42	M	MDD-ED Psychosis	28	2.5	45	2.2	4	1.3	4	1.9	5
6	55	F	MDD-ED	22	13.2	75	3.2	5	2.3	112	2.5	13
7[2]	47	M	MDD-ED Psychosis	40	9.5	0	3.0	31	0.5	5	4.1	14
8	57	F	MDD-ED Bipolar	20	1.0	5	3.6	0	0.5	66	1.8	10
9	74	M	MDD-ED	30	2.1	0	2.1	6	0.9	59	2.8	7
10	69	M	MDD-ED	35	2.1	6	2.7	9	2.3	34	2.5	5

Abbreviations. ECT = electroconvulsive therapy. F = female. M = male. HRSD = Hamilton Rating Scale for Depression. DST = dexamethasone suppression test. REM L = rapid eye movement sleep latency. REM D = rapid eye movement sleep density. MDD-ED = major depressive disorder, endogenous subtype.

1. Previously reported by Grunhaus et al. (1985).
2. Categorized as treatment nonresponder. Patient 5 relapsed 3 weeks after final ECT despite adequate tricyclic regimen.

ranged between 5 and 14, with a mean of 9.6; nine of the ECT series were unilateral and one was bilateral. The HRSD shows that all patients were significantly depressed and that all but one showed a dramatic response to ECT. A paired *t*-test analysis for the HRSD scores showed a very significant difference between the pre-and post-ECT scores ($p < 0.001$), with the mean for the pre-ECT being 26.8 and that for the post-ECT being 7.7 with a standard deviation of 10.8.

The DST results show that 5 of the 10 patients were nonsuppressors before ECT and that all patients were suppressors following the course of ECT. Paired-*t* analysis on the log-transformed values of the maximal postdexamethasone plasma cortisol showed a significant drop for the post-ECT time point ($p < 0.01$) (mean pre-ECT log-transformed cortisol was 1.8; mean post-ECT log-transformed cortisol was 1.0; the standard deviation was 0.7). Pre-ECT REM latency and REM density scores were consistent with those reported for MDD endogenous patients with the exception of patient 6, who showed a REM latency within normal range, but high REM density. The striking change observed with ECT on the sleep parameters is seen individually in Table 1 and in more detail with group means in Table 2.

Table 2. Mean sleep variables pre- and post-ECT

	Sleep variables	Pre-ECT	Post-ECT	SD	Paired *t*
Sleep continuity measures	TRP	429.3 (424.3)[1]	453 (465.3)	47.5 (32.6)	NS (**)
	EMA	12.2 (12.6)	12.7 (15.4)	15.3 (15.5)	NS (NS)
	Sleep latency	51.6 (54.5)	32.6 (36)	24.6 (26.1)	* (NS)
	Arousals	8.8 (10.1)	5.7 (5.1)	8.5 (8)	NS (NS)
	Awake	92.2 (105)	30.6 (31.1)	104.1 (113.9)	NS (NS)
	TSA	278 (257)	375.9 (381.5)	97 (85.1)	** (**)
	A.TSA%	21.9 (24.5)	9 (9.4)	11 (11.1)	** (**)
	Sleep efficiency	64.4 (60.3)	83 (81.7)	16.5 (16.9)	** (**)
Sleep architecture	Stage 1	67.4 (66.6)	77 (63.1)	55.5 (32.6)	NS (NS)
	Stage 2	143 (128.1)	207.5 (220.6)	79.2 (58.1)	* (**)
	Stage 3	4.5 (5.6)	9.2 (9.5)	13.6 (14.7)	NS (NS)
	Stage 4	0 (0)	0.2 (0.12)	0.4 (0.4)	NS (NS)
	Delta	4.5 (5.6)	9.4 (9.6)	13.5 (14.4)	NS (NS)
	Delta %	1.6 (2.0)	2.3 (2.7)	4.2 (4.5)	NS (NS)
	Stage 1%	28 (29.9)	20.9 (17.3)	18.1 (12.3)	NS (*)
REM measures	REM latency	19.8 (20.1)	78.6 (60.3)	77.2 (16.1)	* (***)
	RLMA	17.1 (16.8)	75.5 (58.1)	72.2 (13.3)	* (***)
	REM%	22.8 (22.6)	22.1 (23.2)	8.6 (5.1)	NS (NS)
	REM time	62.5 (57.1)	82 (88.1)	34.4 (17.8)	NS (***)
	REM activity	193.8 (190)	175.7 (191.3)	101.5 (91.6)	NS (NS)
	RA.TSA	0.7 (0.7)	0.5 (0.5)	0.3 (0.3)	* (NS)
	REM density	3 (3.2)	2.2 (2.2)	0.9 (0.9)	** (**)

Abbreviations. ECT = electroconvulsive therapy. REM = rapid eye movement. TRP = total recording period. EMA = early morning awakening. TSA = time spent asleep. A.TSA% = ratio of awake time after sleep onset to total min of actual sleep %. RLMA = REM latency minus awake. RA.TSA = ratio of total REM activity to time asleep.

* = $p < 0.05$. ** = $p < 0.01$. *** = $p < 0.001$.

1. Values in parentheses correspond to treatment responders only ($n = 8$).

The specific sleep EEG measurements are presented in Table 2. The scores for the treatment responders ($n = 8$) are presented in parentheses following the mean for the whole group. Sleep continuity measurements improved significantly after ECT, particularly sleep latency ($p < 0.05$), time asleep ($p < 0.01$), and % awake ($p < 0.05$), and Stage 1% significantly decreased ($p < 0.05$). REM-related measures were the most dramatically affected by ECT, particularly when the analysis was performed with the treatment responders alone. REM latency, REM latency minus awake, REM time, and REM density all changed significantly ($p < 0.05$ or 0.01) when the whole group was considered and even more so for some variables ($p < 0.001$) when just responders were considered.

Discussion

This study confirms our previous report (Grunhaus et al., 1985) and that of Hoffmann et al. (1985) on the favorable effects of ECT on sleep measures of MDD patients. These effects are seen at all levels, including sleep continuity, sleep architecture, and REM-related sleep measurements. Of the two nonresponders, one showed no clinical change, normalized his DST, yet showed no change in REM latency and REM density. Patient 5 (Table 2) received only five ECTs and showed a dramatic positive response, but REM values were more abnormal in the post-ECT recordings. As noted before, this patient relapsed within 3 weeks of ending ECT. The DST results for this patient showed normal suppression both before and after treatment.

Hoffmann et al. (1985) mention that one of their patients did not respond to ECT clinically but nevertheless showed improved sleep recordings. They suggest that this disparity may be due to an effect of ECT on sleep mechanisms regardless of treatment response. In our sample, the evidence is different: those patients who failed to respond to ECT showed a corresponding lack of improvement on most of the sleep parameters explored. Hoffmann et al. do not mention their definition for treatment response, but their raw HRSD scores suggest that some of the patients would have been categorized as either partial responders or nonresponders by more standardized methods of defining treatment response (Coryell, 1982; Greden et al., 1983). At any rate, studies with larger samples are needed to clarify this point.

Our sleep findings support previous published reports of the favorable effects of ECT on sleep parameters (Green and Stajduhar, 1966; Hawkins et al., 1967; Zarcone et al., 1967; Van de Castle and Hawkins, 1969; Mendels et al., 1974). Very few reports using conventionally accepted methods of sleep recording had been published until our report (Grunhaus et al., 1985) and that of Hoffmann et al. (1985). Methodological differences, such as the definitions of sleep parameters and the length of the drug-free period preceding the EEG recording, make comparisons with previous studies very difficult. Nevertheless, Green and Stajduhar (1966) reported a case of a psychotically depressed patient whose dream latency showed a significant delay after ECT. Our findings basically corroborate this report.

As mentioned, we found significant improvements in some of the sleep EEG parameters of patients undergoing ECT. Thus, sleep EEG findings appear to be state dependent and to improve with change in the clinical condition. Rush et al. (1986)

and Puig-Antich et al. (1985) have reported, both in adults and adolescents, that sleep EEG findings during a depressive episode are not necessarily state related. These authors studied depressed patients before treatment with antidepressants and after withdrawal from antidepressants following recovery. They reported that sleep EEG abnormalities persisted after the withdrawal from antidepressant medication. These authors suggest that sleep EEG abnormalities in their patients might reflect an underlying trait feature, which in turn may be related to a higher risk for depression. Some of the differences between the study we are reporting and those by Rush et al. and Puig-Antich et al. may relate to the severity of the depression and the degree of decreased REM latency. Our patients were markedly depressed, and seven of them had REM latencies below 10 min. The patients of Rush et al. and Puig-Antich et al. were less severely ill and had longer REM latencies.

The DST was initially proposed as an effective diagnostic tool in MDD. More recently, it has come under sharp criticism, and its applicability in the diagnosis of MDD has been questioned (Arana et al., 1985; Stokes, 1987). Nevertheless, its potential use for the monitoring of antidepressant response is still viewed positively (Arana et al., 1985), even though some contradictory evidence has been published. To our knowledge, the validity of the DST in a very sick population of depressives has yet to be evaluated prospectively. Patients referred for ECT may constitute a good sample for this study. It may well be that in groups with a higher sensitivity for the DST, such as severely depressed or psychotic patients, the applicability of the DST in monitoring treatment response may be greater. In this sample of patients, DST findings normalized in all of the initial nonsuppressors, including a non-responder to ECT.

The practical applications of the sleep findings we have reported are difficult to assess. On the one hand, ECT is a major psychiatric procedure performed on severely ill patients. The need for laboratory tests that could monitor antidepressant response is particularly great for this group of patients. On the other hand, sleep EEG measures have many restrictions, both economical and clinical (e.g., the length of the drug-free period and patient compliance), that limit their clinical applicability. Our preliminary findings suggest that it may be worthwhile, particularly in those patients with a severe illness, to monitor sleep EEG change occurring with ECT. Prospective study of larger samples of patients, including a followup period after hospital discharge and comparisons with age-matched controls, may provide us with the information necessary to make a more reliable judgment on this very important question.

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