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THE TREATMENT OF INSOMNIA THROUGH THE USE OF ELECTROSLEEP: AN EEG STUDY

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In order to test whether electrosleep is an effective therapeutic procedure, its effects were assessed on the most common of sleep disorders, sleep onset insomnia. The present research was designed to avoid the inadequacies of previous investigations by a) using a double blind procedure; b) using objective evaluation procedures (EEG recordings); and c) using the recommended number and distribution of treatments. Additionally, subjective sleep evaluation and personality-psychopathology data were obtained.

Ten volunteers with objectively established insomnia were randomly assigned to one of two experimental conditions. These conditions were electrosleep treatment and simulated electrosleep treatment. Initially, each subject (*S*) spend 3 successive nights in the sleep laboratory for the EEG-EOG monitoring of his night-time sleep patterns. This was followed by a series of 24 daytime "treatments," each 15 minutes in duration. Following this phase, each *S* again spent 2 nights in the sleep laboratory, at which time post-treatment effects were measured. After 14 no-treatment days, each *S* returned to the sleep laboratory for follow-up measurement.

On the basis of EEG measures, the actual treatment group exhibited a statistically significant decline in the latency of sleep onset, percentage of total bed time awake, and percentage of total sleep time in stage 1 sleep. A significant increase in the percentage of total sleep time in stage 4 and total delta sleep was found. Additionally, subjectively reported latencies of sleep onset declined significantly. These post-treatment results were maintained at follow-up. Significant differences were not found in the simulated treatment group.

Possible group differences in age, chronicity of insomnia, psychopathology, sex, participation in psychotherapy, placebo reactivity and suggestibility are discussed in terms of a "selective" placebo effect. These alternatives are discounted before concluding that electrosleep is truly an effective therapeutic procedure in the treatment of sleep onset insomnia.

Electrosleep has been described as "a state of consciousness grossly indistinguishable from ordinary sleep, produced by the direct action of a weak rhythmic current on the brain of a co-operating subject in a nondistracting environment" (2, p. 9). To initiate this state various electrosleep devices manufactured in the United States, Europe and Japan have been used.

The impetus for electrosleep research has

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come from the theoretical and experimental work of Pavlov. He states, "foreign stimuli which may be repeated without further consequence for the animal also through their own effects alone lead to the development of an inhibitory condition in the cortex" (8, p. 99). This cortical inhibition (sleep) protects the cortical cells from working beyond their capacity. Furthermore, the overworking of cortical cells is related to neurosis and various functional disorders (20).

Electrosleep treatments are typically 30 to 60 minutes in duration, for a period of 5 to 10 days, with no side effects being reported. This may be contrasted with pharmaceutically induced sleep therapy in which treatments are continuous over a much longer period and have associated adverse side effects. These effects have included alterations of the "normal" sleep patterns, that is, a reduction in rapid eye movement (REM) and delta (primarily stage 4) sleep. Psychophysical and biochemical changes such as drug withdrawal, dependence, and addiction are also encountered (3). Thus, there is a great need for a therapeutic procedure, without associated side effects, to deal with the ever-increasing rate of sleep disturbances, now a major health problem in groups of all ages.

In an uncontrolled study, Rosenthal and Wulfsohn (11) have reported the effects of electrosleep therapy on patients registered in a psychiatric "medication clinic." *Ss* were given 10 daily treatments following clinical ratings of anxiety, depression and sleep disturbances on a 7-point scale and evaluation on the Zung Self Rating Depression Scale. Similar evaluation procedures were used following treatments. The initial treatment was 10 minutes in duration, the second treatment, 20 minutes, and the remaining treatment, 30 minutes. Following treatment *Ss* were allowed to lie quietly or sleep for the remainder of the 1-hour period. The results indicated that 7 *Ss* had "relatively total remission" of symptoms, 2 *Ss* had "partial remission" of symptoms and 1 *S* was a "treatment failure." The most marked change was on the clinical sleep disturbance and anxiety self-ratings.

Due to the fact that the *Ss* in this study were undergoing "psychiatric treatment" for 15 to 30 minutes on a monthly or bimonthly basis and were suddenly switched to a 60-minute period on a daily basis, it was reasoned that the effects of the treatment could be attributed to suggestion. In a single blind study (12) *Ss* were assigned to treatment and simulated treatment conditions. The evaluation procedures used in this study were identical to those employed in the previous study. In the treatment condition 9 *Ss* had "relatively total remission" of their symptoms, 1 *S* had "partial remission" and 2 *Ss* were "treatment failures." In the simulated treatment condition 4 *Ss* exhibited an "improvement" whereas 2 *Ss* were unchanged. However, those who did show an improvement did not display the "marked improvement" found in the actual treatment condition. It was concluded that improvement can be attributed partially to suggestion.

Koegler *et al.* (5) evaluated the effects of electrosleep therapy on 16 psychiatric patients who represented 11 different diagnostic classifications. Evaluation procedures consisted of clinical checklist ratings filled out by the patient, a friend or relative of the patient, treatment psychiatrists, and the patient's regular therapist. Additionally, the MMPI was administered prior to and following the treatment.

The results indicated that sleep symptoms (insomnia) were "markedly improved" in 7 *Ss* and "improved" in 4 *Ss*. Five *Ss* did not exhibit sleep symptomatology. Nonsleep symptoms (psychological disturbance) were "markedly improved" in 5 *Ss*, "improved" in 8 *Ss* and "not improved" in 3 *Ss*. Though no statistical analysis or control procedures were employed, it was concluded that a) electrosleep therapy

creates an improvement in sleep patterns for most patients with moderate or severe sleep symptoms; b) significant improvement of nonsleep symptoms often occurred concurrently with the relief of sleep symptoms; and c) a close dependence of the patient on the operator occurred.

It should be noted that studies using EEG measurements as outcome criteria have not as yet been reported, whereas EEG measurements during treatment have been reported (7, 9). Here EEG tracings revealed that sleep potentials did not appear immediately at treatment onset. In 13 recordings the first sleep potentials appeared within 5 to 10 minutes and in the 3 remaining recordings after 10 minutes. In an undescribed control group, sleep potentials appeared 5 to 10 minutes after the start in 9 recordings and after 10 minutes in 10 recordings. In a frequency comparison of EEG states during treatment, deep sleep patterns were not observed, whereas stage 1-REM was the most frequently observed. Additionally, the EEG tracings showed that patients' statements of having slept correlated significantly better with objective data than statements of not having slept.

In considering this brief survey of electrosleep literature, it seems apparent that any further investigation of the effects of electrosleep therapy must deal with the inadequacies of previous research, particularly the lack of proper control and evaluation procedures. There is the question of the number of treatments necessary to show an effect. It has been recently commented that electrosleep may not be beneficial if less than 20 treatments are used and that these effects are cumulative and thus should be more pronounced under massed practice conditions.[2] These two additional considerations have not been investigated in the existing literature.

The research presented here has been designed to avoid the inadequacies of previous work by a) using a double blind procedure to eliminate the possibility of effects due to suggestion; b) using objective evaluation procedures, namely EEG records of the pre- and post-treatment sleep; and c) using the recommended number and distribution of treatments.

[2] A personal communication from Jack Snyder, Roche Pharmaceutical Company.

HYPOTHESES

The following hypotheses were investigated: if electrosleep is truly an effective therapeutic procedure a) the latencies of sleep onset in post-treatment sleep patterns should be significantly different from those latencies found in the pretreatment sleep patterns of people suffering from sleep onset insomnia. More specifically, the post-treatment latencies of those *Ss* receiving the actual treatment should be significantly shorter than their pretreatment latencies. No significant difference between the pre- and post-treatment latencies should be evidenced for those *Ss* receiving the simulated treatment. b) A follow-up test made to assess the stability of the effect of electrosleep therapy should show a maintenance of this effect over a no-treatment follow-up interval. Finally, c) it would be expected that the objective improvement predicted in hypotheses a and b could be accompanied by a confirmatory significant change in the subjective sleep reports of those *Ss* participating in the actual treatment condition. Conversely, if a) there is a significant change in the sleep patterns of *both* the actual and simulated treatment groups, an "objective" placebo effect will have been substantiated; b) there is no significant change in the post-treatment sleep patterns, but there is a significant change in the subjective reports of *both* the actual and simulated treatment groups, a "subjective" placebo effect will be substantiated.

METHOD

SUBJECTS

Local newspaper advertising was used to solicit people who have chronic trouble fall-

ing asleep (insomniacs) to participate in research on an experimental treatment for insomnia. Forty volunteers then completed a preliminary sleep questionnaire (Sleep Questionnaire 1) pertaining to their "normal" sleep habits. This information was used to select those with a subjectively established insomnia—those whose reported latency to sleep onset was 60 minutes or more at least 3 times a week. Ten volunteers were then randomly selected. Each potential *S* was required to spend 3 nights for the monitoring of his sleep in the sleep laboratory. The first 2 nights were considered as adaptation nights. The third session was used as the baseline for all *Ss*. Those *Ss* whose records indicated that they did not reach stage 2 sleep within 20 minutes and stage 4 sleep within 60 minutes were classified as insomniacs and continued in the study. *Ss* who failed to meet this criterion were replaced by a random selection from the remaining *Ss*. At the completion of the study each *S* was paid 25 dollars.

APPARATUS

An Electrodorm 1 was used to produce the electrosleep treatments. An eight-channel Beckman Dynograph was used to record EEG patterns. A Centrallab, PA 4001, 23 position rotary switch was employed in the construction of the double blind apparatus.

DESIGN

Subjects were randomly assigned to one of two experimental conditions. These conditions were electrosleep treatment and simulated treatment. The independent variables in the two-way analysis with repeated measures were: type of treatment (actual and simulated) and type of sleep session (pretreatment, post-treatment, and follow-up). The main dependent variable obtained from the EEG records was the latency of sleep onset in minutes, that is, the time to the first sleep spindle or "K" complex, whichever came first, 2-minute artifact method. The EEG records were analyzed in terms of percentage of total sleep time spent in each sleep stage and the percentage of total bed time *Ss* were awake.

Two control procedures were introduced. First, through the information obtained from Sleep Questionnaire 1 it was possible to schedule treatments in a fashion that would standardize the treatment-post-treatment interval, that is, the last electrosleep treatment was administered to all *Ss* 10 hours prior to their reported bed time as indicated on this questionnaire.

Since it was reasoned that a standardized awakening time, bed time, or amount of sleep could extend or restrict *Ss*' "normal" sleep patterns and thus produce experimental artifacts (REM deprivation, REM satiation *etc.*) which could confound post-treatment results, *Ss* were allowed to follow their "normal" sleep habits. Thus, standardization was not employed between *Ss*, but was employed within *Ss* to insure comparative accuracy of the repeated measures.

Second, "blind" procedures were employed throughout this research. A double blind treatment procedure was employed while EEG records were scored "blindly."

PROCEDURE

Each volunteer completed a questionnaire (Sleep Questionnaire 2) pertaining to his sleep habits for the preceding week, the Cornell Index, and the MMPI. Those *Ss* selected were instructed to refrain from taking any medication or drugs during the course of the study.

On the day following the baseline pretreatment phase, the electrosleep treatment phase was initiated. The electrosleep stimuli were applied to the area above the eyes (through electrodes resembling a sleep mask) and the nape of the neck. Gauze strips saturated with water were placed between the electrodes and the skin. The treatment phase consisted of a total of 24 treatments, the first being 5 minutes in duration, the second, 10 minutes, and the remainder 15 minutes in duration. During the

first two treatments *Ss* remained quietly in the same position for the remainder of the 15-minute period. Treatments were administered at 8 to 10 hours prior to the *Ss* normal bed time. At the end of this phase, *Ss* were asked to complete a second Sleep Questionnaire 2 and the Cornell Index.

The following instructions were used: "You are to lie quietly during the treatment. Shortly, as the current is increased, you will feel a tingling or prickling sensation over your eyes. Report this immediately. When this sensation habituates or subsides, please report that too. This sensation is necessary to determine the proper machine settings. Finally, you will be required to report and describe any visual imagery and/or sensations that you experience during the treatment." One *S* in each group found it more comfortable to sit rather than lie down.

After the tingling sensation was reported, the following procedures were conducted: a) the output current of the Electrodorm 1 was increased 0.1 mA; b) a cover was placed over the output meter to guard against *E* seeing a change in output with the institution of the double blind procedure; and c) a rotary switch on a separate control box was rotated. This switch was rotated to a predetermined position (position 2–23) which had been previously supplied to *E*. Of the 23 total positions only the first was known by *E* to be "live," that is, position 1 was used only during the initial "adjustment" period. Of the remaining 22 positions 6 were "live" and would continue the treatment, whereas the remaining positions terminated the current and resulted in a simulated treatment. Positions were prewired without *E*'s knowledge. Since *S* adapts to the "adjustment" tingle very quickly, neither *E* nor *S* can differentiate between actual and simulated treatment after this "tingle" period.

Ten hours following the last treatment, *Ss* were monitored during their night-time sleep on 2 successive nights. Here post-treatment effects were obtained. After 14 "off" days the *Ss* again slept in the laboratory for 2 successive nights. Here follow-up effects were obtained. Again Sleep Questionnaire 2 and the Cornell Index were administered. In both instances the record from the 2nd night was used for the purposes of analysis.

At the completion of the experiment all *Ss* were informed of the purpose of this investigation. Those who participated in the simulated treatment condition were offered a series of electrosleep treatments if they so desired. Two of the 5 *Ss* requested 10 "legitimate" treatments.

SCORING CRITERIA

The EEG records were scored by the criteria outlined by Rechtschaffen and Kales (10).

RESULTS

PRETREATMENT SLEEP MEASURES

To determine if any initial group differences existed on sleep measures, all pretreatment sleep measures for the actual and the simulated treatment groups were compared through the use of the Mann Whitney *U* test. No significant differences were found between groups on any of the pretreatment objective or subjective sleep measures ($p > .05$).

PRETREATMENT PERSONALITY-PSYCHOPATHOLOGY MEASURES

To determine if groups differed in "presence" or "degree" of psychopathology, Cornell Index scores (see Table 6) were scored in accordance with method B of the Cornell Index manual, distributed to a 2 × 2 contingency table, and analyzed through the use of Fisher's exact probability test. Method B uses a cut-off score of 13. Through the use of Siegel's (14) tables, no significant difference between groups was found ($p > .05$). Additionally, the Mann Whitney *U* test revealed no significant dif-

ferences in Cornell Index raw scores between the two groups ($U = 4.5$, $.096 < p < .152$).

The Spearman Rank Correlation Coefficient revealed no significant relationship between the Cornell Index raw scores and the pretreatment latency of sleep onset (see Table 2) in the actual treatment group ($r_s = .550$), in the simulated treatment group ($r_s = .300$), and in both groups ($r_s = .188$). Additionally, no significant relationship was found between Cornell Index raw scores and the pretreatment percentage of total bed time awake (see Table 3) in the actual treatment group ($r_s = .800$), in the simulated treatment group ($r_s = .300$), and in both groups ($r_s = .381$). Thus, this measure indicates no group differences in "degree" and/or "presence" of psychopathology. Furthermore, "degree" of "psychopathology" was not related to "degree" of sleep disorder.

The MMPI was analyzed through the use of Fisher's exact probability test. T scores above 70 were used as cut-off scores. One of 5 *Ss* in the actual treatment group and 4 of 5 *Ss* in the simulated treatment group exhibited these elevated T scores. Through the use of Siegel's (14) tables, no significant difference between groups was found ($p > .05$). A further analysis was conducted on the various subscales of the MMPI. Table 1 indicated that, through the use of the Mann Whitney technique, groups differed on the Pd (psychopathic deviate), Sc (schizophrenia), and Ma (hypomania) scales. In each instance the simulated treatment group exhibited a higher mean T score. Through the use of Fisher's test, these scores were then evaluated for group differences in "presence" of psychopathology. T scores above 70 were used as cut-off scores. On all four scales, significant differences between groups were not found ($p > .05$). Therefore, though a statistical bias did exist on Pd, Sc and Ma, this was not interpreted as a *clinically* significant finding.

TREATMENT EFFECTS-SLEEP MEASURES

To test hypotheses a and b, the Friedman Two-Way Analysis of Variance by Ranks was employed to analyze the pretreatment, post-treatment, and follow-up measures. Tables 2–4 indicate that on the basis of EEG measures the actual treatment group exhibited a significant decline in time to

TABLE 1

Pretreatment MMPI T scores for Actual and Simulated Treatment Groups

Group	Subject	L	F	K	Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma	Si	AI	TMAS[a]
Actual treatment	1	50	73	49	65	58	56	64	80	62	60	73	57	66	57.3	17
	2	40	50	48	44	46	60	48	55	62	53	51	63	40	45.5	15
	6	46	50	57	44	47	54	57	37	53	48	44	55	38	45.8	6
	7	50	53	55	47	46	53	46	65	44	38	40	45	40	33.1	8
	8	46	53	53	60	53	66	64	41	47	44	42	63	50	31.3	12
	$\bar{X}$	46.4	55.8	52.4	52.0	50.0	57.8	56.2	55.6	53.6	48.6	50.0	56.6	46.8	42.60	11.6
Simulated treatment	3	50	70	61	58	47	53	88	32	88	55	83	63	48	44.3	14
	4	53	68	49	78	75	81	69	32	79	55	77	63	52	49.8	29
	5	43	68	57	57	45	63	67	39	59	53	68	65	53	33.7	14
	9	40	83	46	70	80	70	73	41	65	76	67	68	70	90.0	42
	10	50	60	49	47	77	60	69	78	56	73	78	65	61	104.8	23
	$\bar{X}$	47.2	69.8	52.4	62.0	64.8	65.4	73.2	44.4	69.4	62.4	74.6	64.8	56.8	64.52	24.4
Analysis	U^b	10.5	4	12.5	6.5	7.5	7	0	7.5	4	3.5	2	2	5	7	4
		.670			.222	.310			.310		.056					
	p^c	$< p < .842$	.096	1.000	$< p < .310$	$< p < .420$	.310	.008	$< p < .420$	.096	$< p < .096$	.032	.032	.150	.310	.096

[a] Raw scores are provided as T scores are not available for this scale.
[b] Mann Whitney *U*.
[c] Two-tailed.

sleep onset (latency), percentage of total bed time awake, and percentage of total sleep time in stage 1 sleep. Though a significant increase was evidenced in percentage of total sleep time in stage 4 sleep, a further analysis revealed a significant increase in total delta sleep. This latter analysis was performed to equalize any scoring inadequacies in differentiating stage 3 and stage 4 sleep. On subjective measures, a significant decline in reported time to sleep onset (latency) was reported by the actual treatment group (Table 5). No significant differences were exhibited by the simulated treatment group.

TABLE 2

EEG Latencies of Sleep Onset in Minutes at Pretreatment, Post-treatment, and Follow-up Measurement

Group	S	Pre-	Post-	Follow-up
Actual treatment	1	100.5	12.0	5.0
	2	80.5	14.0	10.0
	6	65.5	16.0	8.5
	7	33.5	3.0	2.0
	8	24.0	8.0	5.5
	$\bar{X}$	60.8	10.6	6.2
				$\chi r^2 = 10.00$
				$p = .00077$
Simulated treatment	3	66.0	23.0	37.0
	4	73.0	69.0	45.0
	5	37.5	27.0	14.5
	9	51.0	64.0	21.0
	10	75.0	111.0	62.0
	$\bar{X}$	60.5	58.8	35.9
				$\chi r^2 = 5.20$
				$p = .093$

TABLE 3

Per cent of Total Bed Time Awake at Pretreatment, Post-treatment, and Follow-up Measurement

Group	S	Pre-	Post-	Follow-up
Actual treatment	1	24.46	2.54	0.67
	2	17.23	2.58	2.47
	6	22.82	11.84	7.40
	7	6.18	1.66	0.60
	8	25.98	2.34	1.10
	$\bar{X}$	19.334	4.192	2.448
				$\chi r^2 = 10.00$
				$p = .00077$
Simulated treatment	3	18.18	12.45	18.22
	4	25.58	25.68	11.46
	5	16.75	9.26	6.19
	9	6.61	18.05	5.90
	10	19.36	27.06	15.98
	$\bar{X}$	17.296	18.500	11.550
				$\chi r^2 = 2.80$
				$p = .367$

TREATMENT EFFECTS-PERSONALITY-PSYCHOPATHOLOGY-MEASURES

The Friedman Two-Way Analysis of Variance by Ranks was employed to analyze the pretreatment, post-treatment, and follow-up measures on the Cornell Index. For total Cornell Index scores no significant difference was found for either the actual ($\chi r^2 = 1.30$, $.522 < p < .691$) or the simulated treatment group ($\chi r^2 = 0.70$, $.691 < p < .954$) (Table 6). Corrected Cornell Index scores yielded similar nonsignificant results for the actual treatment ($\chi r^2 = 3.10$, $.182 < p < .367$) and the simulated treatment group ($\chi r^2 = 0.10$, $.954 < p < 1.00$) (Table 6). This latter analysis omitted questions 29 "Do you often have difficulty in falling asleep?" and 65 "Do you frequently get up tired in the morning?". It was reasoned that a variation in these insomnia-related questions could confound the general "psychological disturbance" score.

RELATIONSHIP OF SUBJECTIVE AND OBJECTIVE SLEEP MEASURES

The Spearman Rank Correlation Coefficient was used in order to compare various subjective and objective (EEG) sleep measures. In the actual treatment group the relationship between the two measures of time to sleep onset was significant at the pretreatment measures ($r_s = .900$, $p < .05$) and nonsignificant at the post-treatment ($r_s = .238$) and follow-up measures ($r_s = .575$). In the simulated treatment group, all

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TABLE 4

Per cent of Total Sleep Time for Seven Sleep Stages Taken at Pre-treatment, Post-treatment, and Follow-up Measurement

Group	Sleep Stage	S	Pre-	Post-	Follow-up
Actual treatment	1	1	43.42	1.86	1.02
		2	1.71	1.99	0.55
		6	10.62	33.58	7.99
		7	6.90	5.67	2.55
		8	1.06	5.40	0.92
		$\bar{X}$	12.742	9.700	2.606
					$\chi r^2 = 7.60$
					$p = .024$
	REM	1	7.12	18.07	14.75
		2	26.35	19.23	18.36
		6	9.48	10.45	7.02
		7	24.45	19.60	24.18
		8	10.03	9.00	18.00
		$\bar{X}$	15.486	15.270	16.462
					$\chi r^2 = 0.40$
					$p = .954$
	REM + A	1	7.12	18.07	14.75
		2	26.35	19.23	18.36
		6	19.82	10.45	18.91
		7	24.45	20.06	24.18
		8	15.83	21.80	18.00
		$\bar{X}$	18.714	17.992	18.840
					$\chi r^2 = 0.40$
					$p = .954$
	2	1	22.42	40.40	34.58
		2	50.72	54.91	48.77
		6	50.11	37.13	42.69
		7	48.43	53.29	48.80
		8	57.26	50.00	47.68
		$\bar{X}$	45.788	47.146	44.504
					$\chi r^2 = 3.60$
					$p = .182$
	3	1	17.08	20.30	14.57
		2	13.57	17.37	19.86
		6	19.41	18.47	28.85
		7	15.05	15.01	14.45
		8	24.27	9.60	26.35
		$\bar{X}$	17.876	16.150	20.816
					$\chi r^2 = 0.40$
					$p = .954$
	4	1	9.96	19.37	31.68
		2	7.64	6.50	12.46
		6	0.00	0.37	1.56
		7	5.17	5.97	10.06
		8	1.58	13.20	7.05
		$\bar{X}$	4.870	9.082	12.562
					$\chi r^2 = 6.40$
					$p = .039$
	Delta[a]	1	27.04	39.67	46.25
		2	21.21	23.87	32.32
		6	19.41	18.74	30.41
		7	20.22	20.98	24.47
		8	25.85	22.80	33.40
		$\bar{X}$	22.746	25.212	33.770
					$\chi r^2 = 7.60$
					$p = .024$

TABLE 4—*Continued*

Group	Sleep Stage	*S*	Pre-	Post-	Follow-up
Simulated treatment	1	3	12.75	16.60	8.69
		4	3.65	2.03	1.62
		5	5.46	1.86	2.11
		9	8.88	9.39	7.90
		10	5.03	3.36	2.44
		$\bar{X}$	7.154	6.648	4.552
					$\chi r^2 = 5.20$
					$p = .093$
	REM	3	14.53	13.97	21.96
		4	5.21	12.20	12.95
		5	19.54	37.66	13.98
		9	25.43	24.91	22.21
		10	28.10	17.67	16.12
		$\bar{X}$	18.562	21.282	17.444
					$\chi r^2 = 0.40$
					$p = .954$
	REM + A	3	14.53	13.97	21.96
		4	25.78	12.20	12.95
		5	19.54	37.66	18.73
		9	25.43	65.96	22.21
		10	28.10	17.67	17.63
		$\bar{X}$	22.676	29.492	18.696
					$\chi r^2 = 1.60$
					$p = .522$
	2	3	55.39	58.89	49.60
		4	27.34	45.73	56.15
		5	47.41	45.08	37.47
		9	51.39	4.87	61.04
		10	56.83	58.30	50.38
		$\bar{X}$	47.672	42.574	50.928
					$\chi r^2 = 0.40$
					$p = .954$
	3	3	14.38	9.88	14.69
		4	33.85	29.67	11.97
		5	18.68	7.24	13.72
		9	7.08	5.41	1.63
		10	9.88	14.66	7.76
		$\bar{X}$	16.774	13.372	9.954
					$\chi r^2 = 2.80$
					$p = .367$
	4	3	2.93	0.66	5.06
		4	9.38	10.37	17.31
		5	8.91	8.16	27.97
		9	7.22	24.37	7.22
		10	0.16	6.01	19.79
		$\bar{X}$	5.720	9.914	15.470
					$\chi r^2 = 4.10$
					$.124 < p < .182$
	Delta[a]	3	17.31	10.54	19.75
		4	43.23	40.04	29.28
		5	26.59	15.40	41.69
		9	14.30	29.78	8.85
		10	10.04	20.67	27.55
		$\bar{X}$	22.294	23.286	25.424
					$\chi r^2 = 0.40$
					$p = .954$

[a] Delta sleep is comprised of the sum of stages 3 and 4.

Follow-up
8.69
1.62
2.11
7.90
2.44
4.552
$\chi r^2 = 5.20$
$p = .093$
21.96
12.95
13.98
22.21
16.12
17.444
$\chi r^2 = 0.40$
$p = .954$
21.96
12.95
18.73
22.21
17.63
18.696
$\chi r^2 = 1.60$
$p = .522$
49.60
56.15
37.47
61.04
50.38
50.928
$\chi r^2 = 0.40$
$p = .954$
14.69
11.97
13.72
1.63
7.76
9.954
$\chi r^2 = 2.80$
$p = .367$
5.06
17.31
27.97
7.22
19.79
15.470
$\chi r^2 = 4.10$
$.124 < p < .182$
19.75
29.28
41.69
8.85
27.55
25.424
$\chi r^2 = 0.40$
$p = .954$

measures, pretreatment ($r_s = .675$), post-treatment ($r_s = .350$) and follow-up ($r_s = .700$), were nonsignificant (Tables 2 and 5).

QUALITATIVE DATA

These data are of a subjective nature and were reported continually throughout the study on Sleep Questionnaire 3. Table 7 contains an "analysis" of these data at pre-treatment, post-treatment, and follow-up measures. As one can readily see, a trend toward less difficulty in falling asleep is reported by the actual treatment group. Furthermore, they report being more rested the following morning. However, in terms of these *subjective reports* similar, though not identical, trends toward *improvement* are in fact reported by the simulated treatment group. This, then, clarifies the questionable results in earlier research.

At the completion of the study, all *Ss* within the actual treatment group felt they had benefited from the electrosleep treatments. In the simulated treatment group 3 *Ss* felt they had benefited from the electrosleep treatments, 1 *S* "benefited through the 1st week of treatments, but then something happened, it didn't seem to get any better," and 1 *S* felt no benefits.

The types of sensations and visual imagery that were "experienced" during the electrosleep "treatments" indicated that the simulated treatment group had a more bizarre experience. For example, they reported, "no stinging today, my head was sort of throbbing;" "I'm really dizzy;" "big electric shock, blue flash, and bad feeling in my mouth;" "a strong surge of power in the beginning followed by the usual prickling sensation, light-headedness off and on . . . a real trip," whereas, the actual treatment *Ss* commented, "a warmth over the eyeballs;" "prickling sensation on eyelids and base of neck;" "felt prickling on eyes and back of neck, but then it dropped off."

Furthermore, the *Ss* in the simulated treatment group seemed to experience a more extreme sensation and for a longer duration of time throughout the "treatment." That is, on occasion they experienced sensations to the very end of the "treatment," whereas the actual treatment group did not. Of greater importance, these reports indicate that the double blind procedure was successfully carried out. Neither *E* nor *Ss* were really able to differentiate between the two types of treatments. This is further substantiated by the fact that when the *Ss* in the simulated treatment group were informed that they had not received actual treatments, they had difficulty believing it because of "all they felt."

Information from Sleep Questionnaire 3 to some extent reveals the treatment process. In the simulated treatment group a marked variability on time to sleep onset and *Ss*' restfulness was continually reported. In the actual treatment group similar variability is also seen, but only during the early phase of treatments. However, within this latter group, at a mean of 15.6 days variability declined sharply.

TABLE 5

Subjectively Reported Latencies of Sleep Onset in Minutes at Pretreatment, Post-treatment, and Follow-up Measurement

Group	*S*	Pre-	Post-	Follow-up
Actual treatment	1	120.0	13.0	10.0
	2	90.0	15.0	20.0
	6	45.0	15.0	15.0
	7	60.0	15.0	10.0
	8	15.0	15.0	10.0
	$\bar{X}$	66.0	14.6	13.0
				$\chi r^2 = 6.70$
				$.024 < p < .039$
Simulated treatment	3	60.0	30.0	30.0
	4	45.0	15.0	15.0
	5	20.0	15.0	10.0
	9	30.0	30.0	20.0
	10	45.0	70.0	45.0
	$\bar{X}$	40.0	32.0	24.0
				$\chi r^2 = 4.30$
				$.124 < p < .182$

TABLE 6

Cornell Index Scores at Pretreatment, Post-treatment, and Follow-up Measurement

	Group	*S*	Pre-	Post-	Follow-up
Total	Actual treatment	1	19	13	12
		2	4	0	0
		6	6	7	6
		7	5	3	6
		8	13	14	11
		$\bar{X}$	9.4	7.4	7.0
					$\chi r^2 = 1.30$
					$.522 < p < .691$
	Simulated treatment	3	6	6	6
		4	44	36	39
		5	7	6	6
		9	43	54	49
		10	22	20	18
		$\bar{X}$	24.4	24.4	23.6
					$\chi r^2 = 0.70$
					$.691 < p < .954$
Corrected[a]	Actual treatment	1	17	13	12
		2	3	0	0
		6	5	5	4
		7	4	2	5
		8	12	12	10
		$\bar{X}$	8.2	6.4	6.2
					$\chi r^2 = 3.10$
					$.182 < p < .367$
	Simulated treatment	3	4	4	5
		4	42	34	37
		5	5	6	5
		9	41	52	47
		10	20	18	16
		$\bar{X}$	22.4	22.8	22.0
					$\chi r^2 = 0.10$
					$.954 < p < 1.000$

[a] Questions 29 and 65 (insomnia-related questions) have not been included in this computation.

UNUSUAL FINDINGS

Four of 5 *S*s in both the actual and simulated treatment groups revealed sleep deprivation effects (ambiguous REM) at least once over the 7 laboratory nights. Additionally, 1 *S* revealed ambiguous REM characterized by sleep spindles in both the EEG and EOG patterns on three occasions. This finding suggests that *S*s were "true" insomniacs rather than "functional" insomniacs.

DISCUSSION

The three major hypotheses of this investigation were confirmed. Specifically, a) the post-treatment latencies of sleep onset in the actual treatment group were significantly shorter than the pretreatment latencies; b) this effect was maintained over a no-treatment interval; and c) the subjective sleep report of those *S*s changed accordingly. Furthermore, no such effects were exhibited by the simulated treatment group. Neither of the placebo hypotheses were confirmed.

Of prime importance is that the decline in wakefulness, both in amount and frequency, as judged by the percentage of total bed time awake and number of spontaneous awakenings, and stage 1 or "light" sleep was

offset by an increase in delta (primarily stage 4 sleep) or "deep" sleep. That is, the increased percentage of total sleep time was "deep" sleep. This, then, counteracted another characteristic of insomnia, suppression of stage 4 sleep.

Finally, though this study presents strong evidence regarding the efficacy of electrosleep, we must first consider the possibility of any "selective" placebo effect or confounding variable in the data before concluding that electrosleep is an effective therapeutic procedure in the treatment of sleep onset insomnia. Simply, is there some logical alternative explanation to account for the improvement following the electrosleep treatments in only the actual treatment group?

One could hypothesize that the results obtained were attributed to group differences in age, chronicity of insomnia (long term *vs.* short term) or a family history of insomnia. However, on all available information none of these variables could have confounded the final outcome. These variables as well as marital status, sex, previous use of sleep "medication," and participation in psychotherapy were well "matched." Where appropriate, a statistical analysis using Fisher's exact probability test reveals no significant group differences ($p > .05$). It must be further noted that all those in the actual treatment group did improve. This was not the case in the simulated treatment group.

Though a pretreatment statistical bias on three subscales (Pd, Sc and Ma) seems to indicate that the relative level of psychopathology is higher in the simulated treatment group, it is difficult to attribute the outcome of this study to effects of psychopathology. First, this bias is not clinical in nature. Second, sleep deprivation effects were exhibited in both groups, therefore negating the possibility of functional insomnia in either group.

Finally, is it possible that one group was more suggestible or more prone to placebo reactivity? That is, could the simulated treatment group have been less prone? Though the area of placebo reactivity has been given considerable attention, little in the way of identifying the placebo reactor has been uncovered. However, there is some consensus that placebo reactors are significantly ($p < .05$) more anxious than nonreactors. This has been the only replicated finding (1, 6, 13, 16–18).

Within the research presented here two MMPI anxiety indices were considered, Welsh's (19) anxiety index (AI) and Taylor's (15) Manifest Anxiety Scale (TMAS). Though no significant differences were found between groups on either of these two

TABLE 7

Qualitative Data (Response Frequencies) Obtained from Sleep Questionnaire 3 at Pretreatment Post-treatment, and Follow-up Measures

Last night, how much difficulty did you have falling asleep initially?

	Pre-	Post-	Follow-up
Actual treatment			
No difficulty	0	2	3
Very little difficulty	2	3	2
Quite a bit of difficulty	3	0	0
Much difficulty	0	0	0
Simulated treatment			
No difficulty	0	2	2
Very little difficulty	2	2	2
Quite a bit of difficulty	3	1	1
Much difficulty	0	0	0

How rested did you feel this morning?

	Pre-	Post-	Follow-up
Actual Treatment			
Very rested	0	3	3
Moderately rested	1	2	2
Not very rested	3	0	0
Not rested at all	1	0	0
Simulated treatment			
Very rested	0	1	1
Moderately rested	1	2	2
Not very rested	0	0	0
Not rested at all	4	2	2

indices (Table 4), the simulated treatment group did score higher on both. Mean AI T score for the simulated treatment group was 64.52 and for the actual treatment group was 42.60. Mean TMAS raw score for the simulated treatment group was 24.4 and for the actual treatment group was 11.6. This would indicate that on the basis of previous findings the simulated treatment group would be more "prone" to placebo reactivity than the actual treatment group. If this is the case, this lends further support to electrosleep being an effective actual treatment rather than a placebo effect.

Thus, only two alternatives appear in the data: a) the obtained effects are solely attributable to electrosleep; or b) electrosleep is a placebo which interacts at some level with psychopathology. However, the latter is negated by the fact that the simulated treatment group was more placebo "prone."

Since we can now conclude that electrosleep is effective in the treatment of chronic sleep onset insomnia, it remains for further research to determine why it is effective. It has been suggested by Koegler *et al.* (4) that: a) a conditioning process is performed at some level; or b) that the explanation lies in the direct action of the electricity on the brain. The former is beclouded by the magnitude of the interval between the treatment and bed time and the latter due to the questionable penetrating power of the current. Thus, though both are questionable, perhaps the latter hypothesis may be the more tenable.

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