

# Seizure Threshold in Electroconvulsive Therapy: III. A Long-Term Study

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## Abstract

There has been a dearth of long-term studies investigating the seizure-threshold changes in patients receiving electroconvulsive therapy (ECT). This study aimed to determine changes in seizure threshold over acute, continuation, and maintenance ECT (Phases I, II, and III). Twenty schizophrenic patients were estimated to have a seizure threshold by the dose-titration method. All patients had a rise in seizure threshold at the end of Phase I with  $185 \pm 196$  per cent increments. Ten patients had a further threshold-increase at the sixth month (Phase II,  $n = 20$ ), and four at the twelfth month (Phase III,  $n = 14$ ). The overall threshold-increases of Phases II and III were  $370 \pm 342$  per cent and  $416 \pm 427$  per cent, respectively. Seizure-threshold increases were robust during acute ECT, and tended to reach a plateau over the continuation and maintenance phases.

**Key word :** Electroconvulsive Therapy, Schizophrenia, Changes in Seizure Threshold, Acute and Maintenance Treatments, Anticonvulsant Effects

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Several lines of evidence suggest that electroconvulsive therapy (ECT) possesses powerful anticonvulsant properties. Post et al<sup>(1)</sup> demonstrated that repeated courses of electroconvulsive

shock in rats were able to block the development of amygdala-kindled seizures. In humans, ECT has been used to treat patients with seizure disorders and their associated behavioral problems shortly

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after its inception(2-5). Recent work has also demonstrated a progressive increase in seizure threshold over the treatment course(6-11).

At the present time, a number of studies quantify seizure threshold over 6-8 ECT sessions (7,8,11-14) and only one estimates up to 20 treatments(15). There has been no study of threshold-change over long-term ECT treatment. Our study aimed to determine seizure-threshold changes from the beginning of acute ECT to the continuation (C-ECT) and maintenance (M-ECT) courses, and factors that might predict the increment.

## METHOD

### Subjects

Twenty patients with DSM-IV schizophrenia(16), with psychotic exacerbation and with a history of prior responsiveness to ECT, were referred for ECT because of failed neuroleptic treatment. The study consisted of 3 phases: Phase I-acute treatment, Phase II -C-ECT (6 months), and Phase III- M-ECT (1 year). Subjects had participated in one of four consecutive research protocols and inclusion/exclusion criteria are described elsewhere (15). Patients were excluded if they had received treatment with depot neuroleptics or ECT during the prior six months and who had received medicines that inhibit seizures (e.g., antiepileptics, benzodiazepines, beta-blockers). All patients were withdrawn from psychotropic medicines at least 5 days prior to the start of ECT. Flupenthixol was prescribed with a fixed dosage schedule: 12 mg/day during the first week then increased to 24 mg/day depending on tolerability. Benzhexol (4-10 mg/day) was used to control extrapyramidal symptoms, with dosage titrated on a clinical basis. The dosages of both medicines were kept constant, after eight weeks of study, throughout all 3 phases. No other medicines were prescribed.

### ECT technique

Methods of ECT administration are fully described elsewhere(15). Briefly, thiopental (2-4 mg/kg), succinylcholine (0.5-1 mg/kg), and atropine (0.4 mg) were used, with dosage in consecutive treatments based on anesthetic response. Ketamine (1 mg/kg) was used as a replacement in patients for whom seizure duration was shorter than our criterion for seizure adequacy (described below) at the maximal charge settings of the ECT devices. Patients were oxygenated from the administration

of anesthetic agent until the resumption of spontaneous respiration. The MECTA SR1 and Thymatron DGx were used. Each patient received treatment with the same ECT device throughout all 3 phases. The bitemporal bilateral electrode placement was used exclusively.

### Electrical stimulus dosing strategy

Seizure threshold was defined, in Phase I, as the lowest electrical intensity that produced an adequate seizure, i.e., a motor seizure lasting at least 30 seconds plus electroencephalogram (EEG) evidence of a cerebral seizure. In both Phases II and III, a duration of motor seizure was lowered to 25 seconds for such criterion. Initial seizure threshold was estimated by our titration schedule (Table 1) at the first two treatments. The first level of electrical intensity (10%) was administered to all patients. In a case of missed or short seizures, stimulus intensity was increased by one level; and up to 4 stimulations with an interval of at least 40 seconds between each without giving additional thiopental. At the second session, stimulus intensity 5 per cent lower was used; if an adequate seizure was not elicited, a prior value was adopted as initial threshold. Wherever, such a seizure could be elicited, this value was used. For all subsequent treatments, electrical intensity was increased by one level when missed or short seizures were attained.

In Phase I, seizure threshold was re-estimated at the seventh, fourteenth, and twentieth sessions. Starting with the patient's prior threshold dose, if an adequate seizure did not occur, a 50 per cent increase in stimulus dose (from a prior threshold to the current dose) was administered. In a case of missed or short seizures, a 75 per cent increase was used; if this still failed, the most recent stimulus intensity was adopted as the threshold. This dosing strategy was also used in both Phases II and III.

A 3-week stabilization period(17,18) was used as a response criterion in Phase I and as a method for terminating ECT during acute treatment. Patients who passed this 3-week period [and with Brief Psychiatric Rating Scale(19) (BPRS) scores  $\leq 25$ ] were ECT responders. In Phase II, subjects received C-ECT with a fixed schedule: 4 weekly followed by 10 biweekly treatments. Seizure threshold was quantified at the first treatment, at the third and sixth months. For Phase III, the M-ECT schedule varied between 2-4 weeks depending on

**Table 1. Our dosing schedule for MECTA SR1 and Thymatron DGx. Initial and successive treatments (25-100% increments)**

Level*	MECTA SR1					Thymatron DGx	
	Pulse width	Frequency	Duration	Current	Charge (mC)	%	Charge (mC)
1	1.0	40	1.25	0.6	60	10	50.4
2	1.0	40	2.0	0.75	120	20	100.8
3	1.0	60	2.0	0.75	180	30	151.2
4	1.2	60	2.0	0.8	230.4	40	201.6
5	1.0	90	2.0	0.8	288	50	252
6	1.4	90	2.0	0.8	403.2	70	352.8
7	2.0	90	2.0	0.8	576	100	504
Extra level**							
1	1.0	40	0.5	0.8	32	5	25.2
2	1.0	40	1.5	0.7	84	15	75.6
3	1.0	90	1.0	0.8	144	25	126
4	1.0	60	2.0	0.8	192	35	176.4
5	1.2	70	2.0	0.75	252	45	226.8
6	1.2	90	2.0	0.8	345.6	60	302.4
7	1.6	90	2.0	0.8	460.8	80	403.2
8	1.8	90	2.0	0.8	518.4	90	453.6

\* Increase by one level is recommended for either dose titration at the first treatment or using in subsequent sessions.

\*\* An extra level is only used at the second treatment.

patient's clinical condition. Seizure threshold was estimated at the ninth and twelfth months.

### Statistical analysis

Seizure threshold data were analyzed after log transformation. Differences between groups on single, continuous variables were evaluated with *t*-tests or analysis of variances (ANOVA). Paired *t*-tests were used to assess the differences of thresholds. Relations between continuous variables were examined with the Pearson's product-moment correlation. The degree to which variables could predict seizure threshold was examined by a stepwise multiple regression analysis. Values are given as mean  $\pm$  SD. SPSS (1996 SPSS Inc.) was used for all analyses.

### RESULTS

Table 2 shows the clinical profile of 20 patients in our study. Fourteen patients received ECT with MECTA SR1 and 6 with Thymatron DGx. All patients completed Phases I and II, and 14 finished Phase III. Seizure threshold and seizure duration at each estimation are summarized in Table 3. Table 4 presents seizure threshold as a function of gender and the ECT devices.

### Phase I study

Initial seizure threshold estimated by our titration schedule was  $85.4 \pm 39.6$  mC. There was a substantial variability in thresholds, ranging from 25.2 to 180 mC (714%); and thresholds of male patients tended to be higher than female patients ( $t = 1.46$ ,  $p = 0.16$ ). Initial threshold estimated with the MECTA SR1 (9 females, 5 males) was significantly higher than the Thymatron DGx (5 females, 1 male;  $t = 3.15$ ,  $df = 18$ ,  $p = 0.006$ ). All patients seized at the first session. Average number of stimulations were  $1.7 \pm 0.7$  (range: 1-3). Initial seizure threshold had an inverse relation with the ECT devices ( $r = 0.6$ ,  $p = 0.006$ ; MECTA = 1, Thymatron = 2), and direct relation with thiopental ( $r = 0.53$ ,  $p = 0.016$ ). Stepwise multiple regression revealed that both the ECT device ( $\beta = 0.52$ ,  $t = 3.08$ ,  $p = 0.007$ ) and thiopental ( $\beta = 0.44$ ,  $t = 2.61$ ,  $p = 0.018$ ;  $F = 9.98$ ,  $p = 0.001$ ) contributed to initial threshold, and accounted for 48.6 per cent of the variance.

As shown in Table 3, the thresholds estimated at the seventh, fourteenth, and twentieth sessions of Phase I were  $171.2 \pm 72.9$  ( $n = 20$ ),  $276.0 \pm 149.6$  ( $n = 11$ ), and  $360.0 \pm 305.5$  mC ( $n = 2$ ), respectively. Average number of stimulations at each session were  $2.6 \pm 0.5$ ,  $2.2 \pm 1.2$ , and only one, res-

pectively. The magnitude of seizure-threshold increase of Phase I was  $184.5 \pm 195.8$  per cent (range: 40-860%). There were no significant differences in threshold-increase between either male or female

patients ( $t = 0.33$ ,  $df = 18$ ,  $p = 0.74$ ) or ECT devices ( $t = 0.89$ ,  $df = 18$ ,  $p = 0.39$ ). Seizure-threshold increase was inversely related to succinylcholine ( $r = 0.45$ ,  $p = 0.048$ ). There was a marked reduction in

**Table 2. Patient characteristics (n = 20).**

Variable	mean $\pm$ SD	range
Age (yr)	$31.7 \pm 7.0$	18-43
Sex	14 female, 6 male	-
Education (yr)	$9.9 \pm 4.1$	4-16
Onset (yr)	$20.4 \pm 4.5$	13-32
Duration of illness (yr)	$11.9 \pm 5.8$	3-25
Current episode duration (yr)	$1.4 \pm 1.4$	1mo-4yrs
Prior failure of adequate neuroleptic trials	$3.7 \pm 1.4$	2-7
No. of psychiatric admissions	$5.9 \pm 4.5$	1-15
No. of ECT treatments	$11.9 \pm 4.4$	7-23
Dosage of flupenthixol (mg)	$22.8 \pm 3.1$	12-24
BPRS scores		
at entry	$50.1 \pm 9.2$	37-67
% of reductions	$72.4 \pm 12.6$	51.2-94
GAF scores		
at entry	$32.1 \pm 5.2$	25-45
% of increments	$62.5 \pm 28.6$	23.1-121.4
Succinylcholine (mg)	$23.8 \pm 5.0$	12.5-87.5
Thiopental (mg)	$129.7 \pm 16.9$	100-150

**Table 3. Seizure threshold data of all 3 phases\*.**

	Seizure threshold (mC)	Seizure duration (sec)	
		motor	EEG
<i>Phase I</i>			
Initial threshold (n = 20)	85.4 ± 39.6 (25.2-180)	53.4 ± 19.4 (30-106)	61.9 ± 21.6 (32-110)
Seventh session (n = 20)	171.2 ± 72.9 (50.4-288)	41.1 ± 12.1 (25-72)	48.9 ± 14.9 (27-85)
Fourteenth session (n = 11)	276.0 ± 149.6 (84-576)	45.2 ± 20.1 (30-100)	57.6 ± 41.5 (35-180)
Twentieth session (n = 2)	360.0 ± 305.5 (144-576)	49.0 ± 24.0 (32-66)	58.0 ± 31.1 (36-80)
Last estimates (n = 20)	223.3 ± 136.4 (50.4-576)	41.3 ± 11.4 (30-68)	47.4 ± 12.6 (34-80)
% change (n = 20)**	184.5 ± 195.8 (40-860) <sup>a</sup>	-17.4 ± 22.6 (-61-22) <sup>b</sup>	-18.3 ± 21.7 (-55-7) <sup>c</sup>
<i>Phase II (n = 20)</i>			
First treatment	226.2 ± 137.2 (50.4-576)	40.4 ± 8.6 (30-57)	47.7 ± 14.6 (30-81)
Third month	286.7 ± 173.7 (50.4-576)	46.2 ± 16.6 (20-85)	55.5 ± 19.3 (25-89)
Sixth month	354.0 ± 198.1 (50.4-576)	43.2 ± 16.9 (26-82)	48.8 ± 18.0 (30-95)
% change	74.1 ± 113.4 (0-400) <sup>d</sup>	7.8 ± 37.0 (-45-100)	-4.1 ± 24.2 (-49-52)
Total change (%)	369.9 ± 341.9 (40-1300) <sup>e</sup>	-11.6 ± 40.3 (-71-85)	-16.6 ± 27.2 (-67-28) <sup>f</sup>
<i>Phase III (n = 14)</i>			
Ninth month	313.9 ± 199.1 (100.8-576)	42.2 ± 25.2 (24-100)	48.6 ± 30 (24-105)
Twelfth month	313.9 ± 199.1 (100.8-576)	39.7 ± 15.1 (20-71)	56.4 ± 37.9 (23-158)
% change	13.0 ± 28.1 (0-100)	-9.5 ± 26.8 (-39-39)	10.1 ± 44.4 (-37-116)
Total change (%)	416.1 ± 426.8 (50-1500) <sup>g</sup>	-26.2 ± 31.4 (-78-42) <sup>h</sup>	-11.0 ± 45.4 (-74-76)

\* Values are shown in mean  $\pm$  SD (range).

\*\* Calculated from the first to last estimates.

a:  $t = 7.98$ ,  $df = 1, 19$ ,  $p < 0.0001$ ; b:  $t = 3.05$ ,  $df = 1, 19$ ,  $p = 0.007$ ; c:  $t = 3.36$ ,  $df = 1, 19$ ,  $p = 0.003$

d:  $t = 3.44$ ,  $df = 1, 19$ ,  $p = 0.003$ ; e:  $t = 8.43$ ,  $df = 1, 19$ ,  $p < 0.0001$ ; f:  $t = 2.86$ ,  $df = 1, 19$ ,  $p = 0.01$

g:  $t = 6.54$ ,  $df = 1, 13$ ,  $p < 0.0001$ ; h:  $t = 3.0$ ,  $df = 1, 13$ ,  $p = 0.01$

**Table 4. Seizure threshold as a function of gender and ECT devices\*.**

	Gender		ECT devices	
	Female (n = 14)	Male (n = 6)	MECTA SRI (n = 14)	Thymatron DGx (n = 6)
<i>Phase I (n = 20)**</i>				
Initial threshold	78.8 ± 43.6	100.8 ± 24.7	98.6 ± 36.5	54.6 ± 29.5 <sup>a</sup>
Last estimates	209.1 ± 144.1	256.4 ± 121.8	272.2 ± 134.4	109.2 ± 37.9 <sup>b</sup>
% increase in threshold	194.3 ± 218.3	161.8 ± 114.5	210.0 ± 225.2	125.0 ± 88.0
<i>Phase II (n = 20)</i>				
First treatment	213.3 ± 145.6	256.4 ± 121.8	276.3 ± 133.9	109.2 ± 37.9 <sup>c</sup>
Sixth month	367.0 ± 201.7	323.6 ± 204.2	386.9 ± 193.7	277.2 ± 203.5
% increase in threshold	96.9 ± 127.7	20.8 ± 40.1	42.7 ± 62.9	147.2 ± 171.4
Overall increases (%)	432.2 ± 373.7	224.6 ± 212.3	313.0 ± 239.1	502.8 ± 515.2
<i>Phase III (n = 14)***</i>				
Ninth & twelfth months	353.9 ± 206.6	167.2 ± 56.9	328.8 ± 212.5	294.0 ± 197.6
% increase in threshold	16.6 ± 31.1	8.5 ± 16.4	19.0 ± 40.1	
Overall increases (%)	507.6 ± 440.1	80.7 ± 26.9	301.1 ± 288.7	569.4 ± 554.6

\* Seizure thresholds are expressed in mean ± SD, and in millicoulombs (mC).

\*\* Phases I & II, classified by gender & ECT devices as-MECTA: 9 female, 5 male; Thymatron: 5 female, 1 male.

\*\*\* Phase III-there were 11 female, 3 male; 8 patients treated with MECTA, and 6 with Thymatron. And, classified by gender & ECT devices as-MECTA: 6 female, 2 male; Thymatron: 5 female, 1 male.

a:  $t = 3.15$ ,  $df = 18$ ,  $p = 0.006$ ; b:  $t = 3.58$ ,  $df = 18$ ,  $p = 0.002$ ; c:  $t = 3.63$ ,  $df = 18$ ,  $p = 0.002$ .

seizure duration over acute ECT (motor:  $-17.4 \pm 22.6\%$ ,  $t = 3.05$ ,  $df = 1,19$ ,  $p = 0.007$ ; EEG:  $-18.3 \pm 21.7\%$ ,  $t = 3.36$ ,  $df = 1,19$ ,  $p = 0.003$ ).

### Phase II study

All patients received C-ECT combined with flupenthixol. Seizure thresholds estimated at the first treatment, third and sixth months were  $226.2 \pm 137.2$ ,  $286.7 \pm 173.7$ , and  $354.0 \pm 198.1$  mC, respectively. Average number of stimulations were  $1.1 \pm 0.2$  (1-2),  $1.9 \pm 1.2$  (1-4), and  $1.7 \pm 1.2$  (1-4), respectively. Ten patients had a further rise in thresholds ( $74.1 \pm 113.4\%$ , range 0-400%;  $t = 3.44$ ,  $df = 1,19$ ,  $p = 0.003$ ). Females tended to have more threshold-increase than male patients ( $t = 2.01$ ,  $df = 18$ ,  $p = 0.06$ ). Rise in seizure threshold was directly related to ECT devices ( $r = 0.59$ ,  $p = 0.028$ ; MECTA = 1, Thymatron = 2), but inversely related to number of acute ECT ( $r = 0.55$ ,  $p = 0.04$ ) and to BPRS scores at Phase I entry ( $r = 0.58$ ,  $p = 0.03$ ). Stepwise multiple regression revealed only the ECT device ( $B = 0.59$ ,  $t = 2.5$ ,  $p = 0.028$ ;  $F = 6.26$ ,  $p = 0.028$ ) contributed to the threshold-increase of Phase II, and accounted for 28.8 per cent of the variance. There were no significant changes in seizure duration (motor:  $t = 0.81$ ,  $p = 0.43$ ; EEG:  $t = 0.37$ ,  $p = 0.7$ ).

### Phase III study

Fourteen patients completed Phase III. Seizure thresholds estimated at the ninth and twelfth months were equal ( $313.9 \pm 199.1$  mC). Average number of stimulations at the ninth month was  $1.4 \pm 0.6$  (1-3). Only 4 patients had a further threshold-increase ( $13.0 \pm 28.1\%$ , range 0-100%). There were no significant changes of seizure duration (motor:  $t = 1.59$ ,  $p = 0.14$ ; EEG:  $t = 1.1$ ,  $p = 0.29$ ).

An overall increase in seizure threshold at the end of Phases II and III were  $369.9 \pm 341.9$  per cent and  $416.1 \pm 426.8$  per cent, respectively (Table 3). The magnitude of threshold-increase of Phase I tended to be higher than Phase II ( $184.5 \pm 195.8\%$  vs  $74.1 \pm 113.4\%$ ;  $t = 2.06$ ,  $df = 1,19$ ,  $p = 0.053$ ), and was much higher than Phases III ( $184.5 \pm 195.8\%$  vs  $13.0 \pm 28.1\%$ ;  $t = 2.88$ ,  $df = 1,13$ ,  $p = 0.013$ ). There was no significant difference in the threshold-increase between Phases II and III ( $t = 1.59$ ,  $df = 1,13$ ,  $p = 0.14$ ). Seizure duration became somewhat shorter by the end of Phases II and III (Table 3).

There were no differences in threshold-increases at the end of Phases II and III, either as a function of sex ( $t = 1.26$ ,  $p = 0.22$ ; and  $t = 1.63$ ,  $p = 0.13$ ) or the ECT devices ( $t = 0.86$ ,  $p = 0.42$ ; and

$t = 1.18$ ,  $p = 0.26$ ). An overall increase in thresholds of Phase II had no relation with any variables. Only thiopental was negatively related with an overall threshold-increase of Phase III ( $r = 0.56$ ,  $p = 0.037$ ).

## DISCUSSION

We reported a prospective study investigating seizure-threshold rise in 20 patients with schizophrenia, received bilateral ECT over the acute and maintenance treatments. The magnitude of threshold-increases at the ends of Phases I, II, and III were  $184.5 \pm 195.8$ ,  $369.9 \pm 341.9$ , and  $461.1 \pm 426.8$  per cent, respectively (Table 3). Threshold-increase appeared to reach a plateau during Phase III. There was a substantial reduction in seizure duration over the ECT course.

Overestimation of seizure threshold was of critical concern in our study. In an attempt to avoid using too weak an electrical stimulation in treating patients with treatment-refractory schizophrenia, our criteria for seizure adequacy were set at  $\geq 30$ s of motor seizures in Phase I, and  $\geq 25$ s in Phase II and III. This criterion is much longer than the usual recommendations (20-25s(20); 15s of motor, and/or 25s of EEG seizures(21)). Thus, it is evident that our criterion for seizure adequacy was excessive, and might inevitably lead to the spuriously high threshold values at later treatments in Phase I. Nonetheless, our conservative dose-titration strategies in quantifying initial thresholds and subsequent estimations might attenuate this problem.

Initial seizure thresholds, the last estimates of Phase I, and the first Phase II estimates, quantified with the MECTA were higher than with the Thymatron. The results may be explained by two reasons. *First*, at each level of an electrical intensity of our dose-titration schedule, a stimulus charge always had a specific reference to the maximal settings of each device. Thus, a stimulus charge of the MECTA was always higher than the Thymatron at all levels. *Second*, there was a different gender ratio of subjects who received ECT with each device. There were more male patients with MECTA (5) than Thymatron (1). Seizure threshold is known to be higher in males than females(14,22-25).

Thiopental was found to have significant effects on both initial seizure threshold and an overall threshold-increase at the end of Phase III. The result might be explained by an anticonvulsant effect of barbiturate anesthetic(20). Succinylcholine exerts its muscle relaxation effect in a dose-related fashion. Since motor seizure duration was our principal criterion in quantifying seizure threshold, succinylcholine might have an inverse relation with the threshold-increase.

The magnitude of seizure-threshold increases was modest in Phases II and III. There is evidence that increase of interval between treatments may result in a relatively unchanged seizure duration(26), and thus might explain our results.

Severity of psychosis, as reflected in the BPRS scores at Phase I entry, was able to predict the threshold-increase of Phase II. This is parallel to the findings of Shapira et al(14) in depressive patients, and our prior report(15). Also, it may suggest that less functional patients may have greater inhibition of functional activity at the outset and less responsivity to ECT.

Another limitation of our study is a concomitant neuroleptic use, which may have effects on seizure threshold. Flupenthixol is  $\sim 1.5$  times more potent than haloperidol. The dosage range 12-24 mg/day used in our study is equal to  $\sim 800$ -1,600 mg of chlorpromazine equivalence. Its effect on seizure threshold has not been reported. By using a fixed titration schedule, its effect on seizure threshold may be minimized. Furthermore, we did not find such an effect in our prior study(15).

The scientific merit of our study is limited by a small number of sample patients, criteria for seizure adequacy, dose-titration strategies and our study design.

In summary, this prospective study investigated the seizure-threshold change over long-term ECT. The magnitude of threshold increase was large. Rise in threshold was robust during acute ECT and tended to reach a plateau in the maintenance phase.

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## ปริมาณไฟฟ้าที่ใช้ในการรักษาด้วยไฟฟ้า : III. การศึกษาระยะยาว†

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การใช้ปริมาณไฟฟ้าที่เหมาะสมในการรักษาด้วยไฟฟ้าเป็นสิ่งที่มีความสำคัญมาก ในปัจจุบันยังไม่มีการศึกษา ระยะยาวของปริมาณไฟฟ้าที่เพิ่มขึ้นระหว่างการรักษาด้วยไฟฟ้า คณะผู้วิจัยได้ทำการศึกษาการเพิ่มขึ้นของปริมาณไฟ ในระหว่างการรักษาด้วยไฟฟ้าตลอดช่วงระยะเวลา 1 ปีในผู้ป่วยจิตเภทเรื้อรังจำนวน 20 คน

พบว่าค่าเฉลี่ยของปริมาณไฟฟ้าขั้นต่ำที่ใช้ในการเริ่มต้นการรักษา มีค่าเท่ากับ 85 มิลลิคูลอมบ์และมีค่าแตกต่างกัน ในระหว่างผู้ป่วยถึง 7 เท่า ปริมาณไฟฟ้าขั้นต่ำที่เพิ่มขึ้นเมื่อสิ้นสุดการรักษาระยะแรกมีค่าเฉลี่ยเท่ากับ  $185 \pm 196\%$ , ที่ระยะ เวลา 6 เดือนเท่ากับ  $370 \pm 342\%$ , และที่ระยะเวลา 1 ปีเท่ากับ  $416 \pm 427\%$

การศึกษานี้พบว่ามีการเพิ่มขึ้นอย่างมากของปริมาณไฟฟ้าขั้นต่ำที่ใช้ในการรักษาระยะแรก มีการเพิ่มขึ้นในปริมาณ ปานกลางที่ระยะเวลา 6 เดือน และมีค่าเกือบคงที่ที่ระยะเวลา 1 ปีของการรักษา สิ่งที่ยาขานนี้มีประโยชน์มากในการรักษา ผู้ป่วยด้วยการใช้ปริมาณไฟฟ้าที่เหมาะสมในช่วงต่าง ๆ ของการรักษาด้วยไฟฟ้า

**คำสำคัญ :** การรักษาด้วยไฟฟ้า, โรคจิตเภท, การเปลี่ยนแปลงปริมาณไฟฟ้าต่ำสุดตลอดช่วงการรักษา

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