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A Controlled Comparison of Simulated and Real ECT

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SUMMARY **Two groups of 16 patients with depressive psychosis took part in a controlled evaluation of electro-convulsive therapy (ECT). One group received six brief pulse unilateral shocks under conventional anaesthesia and muscle relaxation; the second group underwent the same procedure without receiving shocks. Outcome was assessed by a separate investigator using the Hamilton Rating Scale for Depression under double-blind conditions. The results showed that this form of ECT was only superior to the control treatment for one item in the scale, a finding which could have occurred by chance. The results suggest that the ECT pre-treatment procedure has an important therapeutic effect. This casts some doubt on current views of the effectiveness of electro-convulsive therapy in general, and of brief pulse unilateral ECT in particular.**

Introduction

Electroconvulsive therapy (ECT) was introduced to psychiatry before controlled trials of treatment were widely used. Although it has been in clinical use for over 40 years and is accepted as a highly effective therapy, particularly for depressive psychosis, there is little proof that either the passage of electricity or the resultant convulsion are the important components of treatment.

Four methods have been used to investigate the efficacy of ECT, Cronholm (1960) found the effectiveness of ECT with normal fit length superior to that of ECT where fit length had been shortened. Lancaster (1958), during his study of unilateral ECT, found that in 32 cases where ECT had failed to produce a convulsion the improvement in depression scores was significantly less. Others have compared ECT favourably with pharmacotherapy (Robin, 1962; Medical Research Council, 1965), and active ECT has been compared with a simulated procedure in which shocks have not been given (Wilson, 1963; McDonald, 1966; Brill, 1959).

Only the last-mentioned method takes account of the non-specific therapeutic effect of the ECT procedure, which is independent of the shock itself. Unfortunately, the results found were conflicting and open to criticism.

Wilson compared ECT plus imipramine; ECT plus placebo imipramine; placebo ECT plus imipramine; placebo ECT plus placebo imipramine. The author himself admitted the inadequate size of the double placebo group, which contained only six patients, two of whom made a good recovery, and another did well. In the epicrisis, reference was made to ECT and imipramine proving equally effective in equivalent dosage, but the author could not assess their superiority over placebo.

McDonald performed a similar study but with only four in the simulated ECT group. In his paper, their outcome was concealed in the data of those who received placebo amitriptyline, but as a combined group they did worse than those who received amitriptyline or real ECT ($P < 0.05$).

Using a mixed diagnostic group, Brill found no statistically significant difference in outcome with straight ECT; ECT plus succinylcholine; ECT plus thiopentone; thiopentone alone; nitrous oxide anaesthesia alone. This was true for the depressed patients in the group also. Because of these doubts, another comparison of

active and simulated ECT was deemed necessary.

Method

Patients

Following Ethical Committee approval, and having obtained informed consent, all right-handed patients with a diagnosis of depressive psychosis referred for ECT at Knowle Hospital were screened. Both in-patients and out-patients were included, but those with other psychiatric or organic disorder were excluded, as were those who had received ECT within the preceding three months.

Procedure

Psychotropic drugs (see Table I), except benzodiazepine hypnotics, were stopped the night before the first treatment. Allocation of each patient to a simulated or active ECT group was made by a constrained random procedure based on age (over or under 45) and sex, so that the two groups were balanced for these variables. All patients received a standardized anaesthetic regime (with dose modified for extremes of physique) of methohexitone sodium, 70 mgm; suxethonium cation, 50 mgm; and atropine 1.2 mgm intravenously. All patients then received four ventilations with oxygen before the electrodes were applied to the right temporo-parietal position described by Lancaster (1958). The only difference in treatment given to the placebo group was that they did not receive an electrical stimulus. Those in the active ECT group received a brief pulse stimulus of approximately 10 Joules from an Ectron Duopulse Mk. 4, which was checked electrically and mechanically before and at the conclusion of the project. This was noted to produce a bilateral modified convulsion on every occasion. Patients in both groups were then ventilated until spontaneous respiration had been established.

The control group, therefore, received an elaborate procedure involving loss of consciousness, nursing care and attention, and the expectation of a beneficial outcome. The treatments were given three times weekly and referring doctors were at liberty to withdraw any patient from the study if adequate improvement had not been achieved.

Assessments

These included:—

(i) the Hamilton Rating Scale for Depression (Hamilton, 1960), completed by D.G. (who was blind to which treatment was being given) prior to and one day after 6 treatments and again one month later.

(ii) a global assessment of improvement by the referring doctor one day after 6 treatments,

(iii) days in hospital and treatments received in the month of follow up.

As a final check that the code had not been broken, referring doctors were asked to state which treatment they thought their patients had received.

Results (Table II)

The scores on the Hamilton Scale were found to be skewed, so non-parametric statistics were used in the analysis. The Wilcoxon matched-pairs signed-rank test (Siegal, 1956) was used, and as the hypothesis did not predict the direction of the result a two-tailed test was appropriate.

The overall outcome for the 32 patients in this study was quite good, only 5 failing to make any improvement after six 'treatments' given over a period of two weeks. These 5 patients all improved during the one-month follow-up period, and although 6 other patients were lost from the study one can conclude that the prognosis of depressed patients in an active treatment program is good. The contribution of spontaneous remission during this study remains an unknown factor because of the lack of a totally untreated control group.

Discussion

In this group of patients suffering depressive psychosis, six brief pulse unilateral ECT's did not produce a significantly superior therapeutic effect when compared with a simulated procedure. There could be several reasons for these results other than a conclusion that the electrical stimulus/convulsion component of ECT is an unimportant part of the ECT procedure. The diagnosis of depressive psychosis

TABLE I

Demographic and pre-treatment assessment

	Male	Female	Age	Previous admissions for depression	Previous courses of ECT	Carney prognostic index depressive psychosis	Carney ECT indicator good	Carney ECT indicator bad	Anti-depressant medication prior to study	Pre-treatment Hamilton rating
Real ECT group										
		*	38	1	1	*			0	64
	*		60	4	3			*	75 mg. 1/52 Imipramine	50
		*	55	5	3				100 mg. 12/52 Amitriptyline	54
		*	44	4	1				0	52
		*	56	3	2				75 mg. 2/52 Imipramine	72
	*		50	0	0			*	0	32
	*		53	0	0	*	*		0	36
	*		36	4	4	*			0	52
		*	59	10	10		*		0	42
		*	52	0	0	*		*	0	46
		*	64	2	1	*			75 mg. 4/52 Maprotiline	58
		*	60	1	0	*			75 mg. 24/52 Imipramine	52
	*		69	2	2				45 mg. 8/52 Phenelzine	30
	*		44	6	0	*	*		2 mg. 2/52 Flupenthixol	58
		*	68	1	1	*	*		0	76
	*		63	1	1	*	*		100 mg. 4/52 Imipramine	22
Total	7	9	871	44	29	9	5	3	8	796
Mean			54.4	2.75	1.8					50
Simulated ECT group										
		*	42	1	1	*	*		100 mg. 8/52 Dothiepen	70
	*		60	5	2	*	*		75 mg. 4/52 Clomipramine	32
		*	63	2	2				0	48
		*	37	6	5			*	0	38
		*	54	7	5	*			150 mg. 12/52 Amitriptyline	54
	*		54	3	1	*			0	72
	*		56	1	0	*	*		0	46
	*		41	3	3	*	*		0	60
		*	52	3	1	*			0	56
		*	59	2	2	*			0	58
		*	63	0	0	*	*		0	66
		*	60	1	1		*		0	56
	*		52	0	0	*			150 mg. 12/52 Amitriptyline	74
	*		43	1	0	*			200 mg. 6/52 Amitriptyline	70
		*	66	1	0	*	*		0	30
	*		54	0	0		*		0	34
Total	7	9	856	36	23	12	8	1	5	864
Mean			53.5	2.25	1.4					54

TABLE I—Continued

Assessments after six treatments						*Assessment after a further month*		
	Hamilton rating	Hamilton Outcome improvement 1– 33%+ 34– 66%++ 67–100%+++	Referring Doctors' global assessment of outcome	Referring Doctors' attempt to break blind code	Extra ECT after study	Extra antidepressant after study	Hamilton rating	Hamilton improvement 1– 33%+ 34– 66%++ 67–100%+++
Real ECT group								
	56	+	+	Placebo	7	Amitriptyline	2	+++
	28	++	++	N.K.	—	Imipramine	Lost	Lost
	26	++	++	Placebo	—	Amitriptyline	42	+
	18	++	+++	N.K.	—	0	16	++
	2	+++	+++	Active	—	0	14	+++
	12	++	+++	N.K.	—	0	0	+++
	2	+++	++	N.K.	—	0	Lost	Lost
	10	+++	+++	N.K.	—	Amitriptyline	10	+++
	8	+++	+++	Active	3	Amitriptyline	10	+++
	8	+++	+++	N.K.	—	0	14	+++
	16	+++	++	N.K.	—	Amitriptyline	Lost	Lost
	56	0	0	N.K.	5	0	14	+++
	20	+	++	N.K.	5	Clomipramine	20	+
	46	+	+	N.K.	5	Clomipramine	16	+++
	56	+	+	N.K.	6	Amitryptiline	30	++
	12	++	+	Placebo	—	Dothiapen	20	+
Total	376	+++ 6	+++ 6	Correct 2 Incorrect 3	31 ECTs 6 patients	10 patients	208	+++ 8
Mean	24			N.K. 11			16	
Simulated ECT group								
	38	++	+	N.K.	—	0	10	+++
	8	+++	+++	Placebo	—	Clomipramine	14	++
	43	+	+	N.K.	7	0	8	+++
	38	0	+	N.K.	7	0	14	++
	68	0	0	N.K.	5	Amitriptyline	8	+++
	40	++	+++	N.K.	—	Amitriptyline	Lost	Lost
	18	++	++	N.K.	4	Amitriptyline	4	+++
	12	+++	+++	Active	—	0	30	++
	6	+++	++	Placebo	—	Imipramine	0	+++
	62	0	0	Placebo	—	0	50	+
	18	+++	+++	Active	4	0	Lost	Lost
	26	++	+++	Active	4	MRC Trial Tab	Lost	Lost
	31	++	++	Active	—	Amitriptyline	12	+++
	70	0	0	Placebo	5	0	10	+++
	10	+++	+++	Active	—	Mianserin	0	+++
	10	+++	++	N.K.	—	Amitriptyline	4	+++
Total	498	+++ 6	+++ 6	Correct 4 Incorrect 5	36 ECT's 7 patients	9 patients	164	+++ 9
Mean	31			N.K. 7			12.6	

TABLE II

Hamilton rating scale for depression—scores before and after six treatments: ECT versus placebo (absolute change in scores)

Hamilton Rating Scale for Depression	Mean change		Wilcoxon test		
	ECT	Control	Z	p. 1. Tail	N/T
Total Score	25.86	23.12	−0.31	0.38	16/62
Items					
1. Depressed mood	3.0	2.5	0.31	0.38	12/35
2. Guilt	2.0	1.76	0.43	0.33	12/35.5
3. Suicide	3.38	3.32	0.59	0.28	15/21.5
4. Insomnia (initial)	1.12	1.26	−0.22	0.41	11/30.5
5. Insomnia (middle)	0.38	1.62	−1.957	0.025	13/17.5*
6. Insomnia (delayed)	1.12	1.50	−0.40	0.34	11/28.5
7. Work and interest	2.62	2.12	0.08	0.47	12/38
8. Retardation	0.76	1.40	−1.19	0.12	8/9.5
9. Agitation	1.26	0.62	1.16	0.12	11/20
10. Anxiety (psychic)	2.76	1.76	0.91	0.18	13/32.5
11. Anxiety (somatic)	2.5	0.62	1.61	0.0537	12/18.5
12. Somatic symptoms gastro-intestinal	1.26	1.38	−0.31	0.38	6/9
13. Somatic symptoms general	1.12	0 62	0.90	0.18	12/27.5
14. Genital symptoms	0.88	1.26	−0 56	0 29	10/22
15. Hypochondriasis	0.62	−0.50	1.86	0.031	7/3*

* P. Two-tail significant at the 0.10 level.

might have been inaccurate, but we relied on the referring consultants' diagnosis, and as 77 per cent reliability has been found between psychiatrists using this criterion (Kreitman, 1961) this procedure was felt to be justified. The selection of out-patients might reflect the referring consultants' opinion that these had a better prognosis than patients admitted, and the randomization of two out-patients to the placebo group but not the active treatment group might be argued to have introduced a bias. This is not borne out, as the two out-patients made only a mean 38 per cent improvement and therefore slightly disadvantaged the placebo group. It could also be argued that only mildly depressed patients were referred for the study. As all the patients receiving ECT were screened, and only six patients fulfilling the research criteria did not enter the study, this is difficult to defend. The possibility was examined that a sub-group of patients did well but their responses were masked by our presentation of mean results; the distribution of good responses was similar between the groups, and no clinical features distinguished them. The Carney diagnostic index for depressive illness (1965) was found to predict the outcome of treatment poorly in both groups. It has been argued that unilateral ECT is less effective than bilateral ECT (Royal College of Psychiatrists, 1977), and despite argument to the contrary (D'Elia, 1975), it is impossible to generalize the results of this study to include other techniques of administration. Valid criticism can be made that assessment after only two weeks was too early to allow the full therapeutic effect of ECT to develop, and that the arbitrary application of six treatments was not ideal (Barton, 1973). The referring clinicians were, however, able to add extra ECT or medication afterwards, and there was no difference in outcome between the groups one month later. That part of the study was unfortunately not blind, and it is difficult to interpret the findings meaningfully owing to the loss of six patients in that time.

Overall improvement on the Hamilton Scale showed a small trend in favour of ECT, and it is possible that if a larger sample of patients had been treated this difference would have been significant. Nevertheless, only two of the individual items in the scale were significant, one in favour of ECT and one in favour of the

control treatment, results which could have occurred by chance.

The implication of these findings is that the effectiveness of unilateral brief pulse ECT shown in previous investigations is due in large part to the attendant procedures associated with the administration of an anaesthetic and the mystique associated with an unusual form of treatment. Further studies with simulated ECT are therefore indicated to explore this apparent placebo effect, particularly in patients treated over a longer period, using a range of stimulus parameters and electrode placements.

In a recently published study (Freeman, 1978) it was found that bilateral ECT using a sinusoidal stimulus waveform was significantly superior to a simulated ECT placebo. If the interpretations of both that study and the one presented here are correct, then the equipotency of unilateral and bilateral ECT, given with both sinusoidal and brief pulse stimuli must be seriously re-examined.

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