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A Controlled Comparison of Simulated and Real ECT

By J. LAMBOURN and D. GILL

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

Pre-treatment Hamilton rating	*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				
64	56	+	+	Placebo	7	Amitriptyline	2	+++
50	28	++	++	N.K.	—	Imipramine	Lost	Lost
54	26	++	++	Placebo	—	Amitriptyline	42	+
52	18	++	+++	N.K.	—	0	16	++
72	2	+++	+++	Active	—	0	14	+++
32	12	++	+++	N.K.	—	0	0	+++
36	2	+++	++	N.K.	—	0	Lost	Lost
52	10	+++	+++	N.K.	—	Amitriptyline	10	+++
42	8	+++	+++	Active	3	Amitriptyline	10	+++
46	8	+++	+++	N.K.	—	0	14	+++
58	16	+++	++	N.K.	—	Amitriptyline	Lost	Lost
52	56	0	0	N.K.	5	0	14	+++
30	20	+	++	N.K.	5	Clomipramine	20	+
58	46	+	+	N.K.	5	Clomipramine	16	+++
76	56	+	+	N.K.	6	Amitryptiline	30	++
22	12	++	+	Placebo	—	Dothiapen	20	+
796	Total 376	+++ 6	+++ 6	Correct 2 Incorrect 3	31 ECTs 6 patients	10 patients	208	+++ 8
50	Mean 24			N.K. 11			16	
				Simulated ECT group				
70	38	++	+	N.K.	—	0	10	+++
32	8	+++	+++	Placebo	—	Clomipramine	14	++
48	43	+	+	N.K.	7	0	8	+++
38	38	0	+	N.K.	7	0	14	++
54	68	0	0	N.K.	5	Amitriptyline	8	+++
72	40	++	+++	N.K.	—	Amitriptyline	Lost	Lost
46	18	++	++	N.K.	4	Amitriptyline	4	+++
60	12	+++	+++	Active	—	0	30	++
56	6	+++	++	Placebo	—	Imipramine	0	+++
58	62	0	0	Placebo	—	0	50	+
66	18	+++	+++	Active	4	0	Lost	Lost
56	26	++	+++	Active	4	MRC Trial Tab	Lost	Lost
74	31	++	++	Active	—	Amitriptyline	12	+++
70	70	0	0	Placebo	5	0	10	+++
30	10	+++	+++	Active	—	Mianserin	0	+++
34	10	+++	++	N.K.	—	Amitriptyline	4	+++
864	Total 498	+++ 6	+++ 6	Correct 4 Incorrect 5	36 ECT's 7 patients	9 patients	164	+++ 9
54	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. l. 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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References

BARTON, J. L., MEHTA, S. & SNAITH, R. P. (1973) The prophylactic value of extra ECT in depressive illness. *Acta Psychiatrica Scandinavica*, **49**, 386–92.

BRILL, N. Q. (1959) Relative effectiveness of various components of electroconvulsive therapy: an experimental study. *Archives of Neurology and Psychiatry*, **81**, 627–35.

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–74.

CRONHOLM, B. & OTTOSSON, J-O. (1960) Experimental studies of the therapeutic action of electroconvulsive therapy in endogenous depression. *Acta Psychiatrica et Neurologica*, **35**, (145), 69–97.

HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

KREITMAN, N., SAINSBURY, P., MORRISSEY, J., TOWERS, J. & SCRIVENER, J. (1961) The reliability of psychiatric assessment: an analysis. *Journal of Mental Sciences*, **107**, 887–908.

LANCSTER, N. P., STEINERT, R. R. & FROST, I. (1958) Unilateral electroconvulsive therapy. *Journal of Mental Science*, **104**, 221–7.

MEDICAL RESEARCH COUNCIL (1965) Clinical trial of the treatment of depressive illness. *British Medical Journal*, *i*, 881–6.

MCDONALD, I. M., PERKINS, M., MARJERRISON, G. & PODILSKY, M. (1966) A controlled comparison of amitriptyline and electroconvulsive therapy in the treatment of depression. *American Journal of Psychiatry*, **122**, 1427–31.

ROBIN, A. A. & HARRIS, J. A. (1962) A controlled study of imipramine and electroplexy. *British Journal of Psychiatry*, **108**, 217–19.

ROYAL COLLEGE OF PSYCHIATRISTS (1977) Memorandum on the use of electroconvulsive therapy. *British Journal of Psychiatry*, **131**, 261–72.

SIEGEL, S. The Wilcoxon matched-pairs signed-ranks test. In *Non-parametric Statistics for the Behvioural Sciences* (ed. H. F. Harlow). New York: McGraw-Hill Book Company Inc.

VALENTINE, M., KEDDIE, K. M. G. & DUNNE, D. (1968) A comparison of techniques in electro-convulsive therapy. *British Journal of Psychiatry*, **114**, 989–96.

WILSON, I. C., TAYLOR VERNON, J., GUIN, T. & SANDIFER, M. G. (1963) A controlled study of treatments of depression. *Journal of Neuropsychiatry*, **4**, 331–7.

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PAPERS AND SHORT REPORTS

Electric convulsion therapy in depression: a double-blind controlled trial

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Abstract

The therapeutic effect of simulated and real bilateral electric convulsion therapy was examined in a double-blind, randomised trial on 22 patients with primary depressive illness. Each treatment was given twice weekly for three weeks and the results assessed by the psychiatrist using a visual analogue rating scale, nurses using a nine-point rating scale, and the patients themselves using the Beck Depression Inventory.

With all three methods of assessment patients given the real treatment substantially improved ($p < 0.001$), whereas those given simulated treatment showed little change. Three weeks after substituting real treatment for simulated, however, these patients were also significantly improved (psychiatrist's rating $p < 0.001$; nurses' rating $p < 0.005$; Beck inventory $p < 0.005$).

These findings confirm the value of electric convulsion therapy in severe depressive illness and strongly suggest that the convulsion is important for the therapeutic effect.

Introduction

Electric convulsion therapy has been used for over 40 years in the treatment of depressive illness. Though at its introduction the evaluation of treatment by double-blind controlled trials was uncommon, several investigations over the years have shown electric convulsion therapy to be of considerable value in this condition.[1]

The importance of the convulsion in the therapeutic effect has been the subject of recent and conflicting reports. In one study[2] the effects of giving either two real or two simulated doses of electric convulsion therapy were compared, and despite both groups receiving concomitant antidepressant treatment, a clear advantage for electric convulsion therapy was reported. A second[3] study also found a significant advantage of eight real over eight simulated treatments, but a third investigation[4] failed to find any significant difference between six real and simulated treatments.

In view of these conflicting investigations I report a double-blind comparison of simulated and real electric convulsion therapy in patients suffering from primary depressive illness.

Patients and methods

Twenty-five inpatients with primary depressive illness[5] were selected for the study and gave their consent for the investigation. One patient in the electric convulsion therapy group and one in the simulated group were withdrawn from the trial in the first week because of concern about their lack of improvement. Another patient in the real electric convulsion therapy group could not complete the Beck Depression Inventory[6] and was withdrawn from the trial in the first week.

The remaining 22 patients, admitted to the trial after at least one week's assessment as an inpatient, were randomly allocated to receive either real or simulated electric convulsion therapy. The mean age of the real electric convulsion therapy group was 52.0 ± 3.3 years and of the simulated group 53.3 ± 6.9 years. Table I shows the sex distribution.

TABLE I—*Psychiatrist's rating of patients receiving real and simulated electric convulsion therapy (visual analogue rating scale). Results expressed as means ± SEM*

Group	M	F	Base	Week 1	Week 2	Week 3	After treatment
Electric convulsion therapy	6	5	67·9 ± 4·7	47·4* ± 4·2	34·1* ± 3·2	19·5* ± 5·4	15·8* ± 4·6
Simulated electric convulsion therapy	7	4	70·7 ± 6·5	70·8 ± 5·9	68·9 ± 4·2	63·4 ± 5·2	72·2 ± 5·4

*Significant reduction from baseline: $p < 0.001$ (method of paired comparisons).
Change in rating for real electric convulsion therapy group significantly greater than corresponding change in simulated group: one week $p < 0.02$; two weeks, three weeks, and after treatment $p < 0.001$.

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Treatment was given twice weekly, and its nature was kept blind both to the patients and to the assessors. All patients were premedicated with 80 mg atropine subcutaneously. The anaesthetic agent was Althesin (alphadolone) and the muscle relaxant suxamethonium. Electric convulsion therapy was administered from a Transycon machine using 40 joules with double-sided unrectified waveform and bilateral anterior temporal placement of the electrodes. All patients were given 50 mg amitriptyline at night, and no other medication was prescribed.

After six treatments, if judged therapeutically desirable, patients were switched to the other treatment for a further six treatments.

Patients were assessed by the psychiatrist in charge using a visual analogue rating scale, one end being absent morbidity, and the other end the most severe depressive illness. The psychiatrist performed the rating the day after each treatment and again five days after the sixth and final treatment. Nurses rated the patients daily on a nine-point scale from "very much worse" to "very much better." The patients rated themselves on the Beck Depression Inventory before starting treatment and the day after each second treatment.

The results of all three assessments were presented before treatment (baseline) and thence weekly the day after each second treatment.

TABLE II—*Beck Depression Inventory scores in patients treated with real and simulated electric convulsion therapy. Figures are means ± SEM*

Group	No	Base	Week 1	Week 2	Week 3
Electric convulsion therapy	11	26·6±2·8	21·5*±3·3	15·3**±2·4	10·8**±2·6
Simulated electric convulsion therapy	11	24·1±3·5	23·9 ±3·6	21·7 ±3·4	22·2 ±3·8

Significant reduction from baseline: *$p<0.05$; **$p<0.001$ (method of paired comparisons).
Change in Beck score for real electric convulsion therapy group significantly greater than corresponding change in simulated group; one week $p<0.05$; two weeks $p<0.01$; three weeks $p<0.002$.

TABLE III—*Nurses' rating scale for patients treated with real and simulated electric convulsion therapy. Figures are means ± SEM*

Group	No	Base	Week 1	Week 2	Week 3
Electric convulsion therapy	11	5·9±0·4	3·0**±0·4	1·6**±0·2	1·2**±0·1
Simulated electric convulsion therapy	11	5·1±0·4	3·8* ±0·4	4·6 ±0·5	4·2 ±0·6

Significant reduction from baseline: *$p<0.05$; **$p<0.001$ (method of paired comparisons).
Change in nurses' rating for real electric convulsion therapy group significantly greater than corresponding change in simulated group: one week $p<0.05$; two and three weeks $p<0.001$.

TABLE IV—*Psychiatrist's, nurses', and Beck rating scales of change for 10 of 11 patients completing course of simulated and subsequently crossed over to real electric convulsion therapy. Figures are means ± SEM*

Rating by:	No	Base	Week 1	Week 2	Week 3	After treatment
Psychiatrist	10	73·4±5·8	50·3*±5·9	31·9***±4·9	14·5***±4·0	11·9***±3·3
Nurses	10	3·8 ±0·4	3·8 ±0·4	2·3* ±0·2	1·6** ±0·2	
Beck Scale	10	21·1 ±3·6	17·6* ±3·8	13·9* ±3·6	10·7** ±3·2	

Significant reduction from baseline: *$p<0.05$; **$p<0.005$; ***$p<0.001$.

Results

Tables I, II, and III show the ratings during the first three weeks of treatment. There was a highly significant and clinically important improvement in the electric convulsion therapy group, whether rated by the psychiatrist, nurses, or the patients themselves. The effects were apparent after one week of treatment. The patients treated by simulated electric convulsion therapy showed little clinical change during this period and differed significantly from the real electric convulsion therapy group in this respect.

Ten of the 11 patients receiving simulated electric convulsion therapy were switched to the alternative treatment. None of the electric convulsion therapy group was switched ($p<0.005$). Table IV gives the results in the switched group. This group of patients responded significantly to real electric convulsion therapy.

Discussion

The results of this trial are very strong evidence that the convulsion is an important part of the therapeutic effect of electric convulsion therapy. The patients, who had had at least one month's history of depression[5] and one week's assessment as an inpatient before admission to the trial, showed little improvement on simulated electric convulsion therapy but made a substantial improvement when given real electric convulsion therapy.

All trials on antidepressant measures tend to differ because of variation in patient selection, but there is now strong evidence from the results of these studies that the convulsion is important in the effect of electric convulsion therapy. It is perhaps relevant that the study showing no difference[4] used unilateral rather than bilateral electric convulsion therapy.

In this study electric convulsion therapy was shown to be an excellent treatment of severe depressive illness. The question of further treatment after the initial study was not investigated. It should be emphasised, however, that the relapse rate after electric convulsion therapy or after six weeks' treatment with an antidepressant remains high,[7–10] and that the optimal time for treatment after apparent recovery is six months. After electric convulsion therapy patients will remain vulnerable, and it is prudent to administer for six months either antidepressants or lithium.[11] If the depression is the patient's third or more episode then the question of prophylactic lithium should be considered.[12]

References

[1] Crow TJ, Johnstone EC. Electroconvulsive therapy: efficacy, mechanism of action, and adverse effects. In: Coppen A, Paykel ES, eds. *Psychopharmacology of affective disorders*. Oxford: Oxford University Press, 1979:108-22.

[2] Freeman CPL, Basson JV, Creighton A. Double-blind control'

electroconvulsive therapy (ECT) and simulated ECT in depressive illness. *Lancet* 1978;i:738-40.
[3] Johnstone EC, Deakin JFW, Lawler P, *et al.* The Northwick Park electroconvulsive therapy trial. *Lancet* 1980;ii:1317-20.
[4] Lambourn J, Gill D. A controlled comparison of simulated and real ECT. *Br J Psychiatry* 1978;133:514-9.
[5] Feighner JP, Robins E, Guze S, Woodruff R, Winokur G, Munoz R. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry* 1972;26:57-63.
[6] Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry* 1961;4:561-71.
[7] Imlah NW, Ryan E, Harrington JA. The influence of antidepressant drugs in the response to electroconvulsive therapy and on subsequent relapse rates. *Journal of Neuropsychopharmacology* 1965;4:439-42.
[8] Kay D, Fahy T, Garside R. A seven-month double-blind trial of amitriptyline and diazepam in ECT treated depressed patients. *Br J Psychiatry* 1970;117:667-71.
[9] Mindham RHS, Howland C, Sheppard M. An evaluation of continuation therapy with tricyclic antidepressants in depressive illness. *Psychol Med* 1973;3:5-17.
[10] Coppen A, Ghose K, Montgomery S, Rao VAR, Bailey J, Jørgensen A. Continuation therapy with amitriptyline in depression. *Br J Psychiatry* 1978;133:28-33.
[11] Perry A, Tsuang MT. Treatment of unipolar depression following electroconvulsive therapy. *Journal of Affective Disorders* 1979;1:123-9.
[12] Coppen A, Noguera R, Bailey J, *et al.* Prophylactic lithium in affective disorders. *Lancet* 1971;ii:275-9.

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Policy for prevention of Asian rickets in Britain: a preliminary assessment of the Glasgow rickets campaign

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Abstract

Evidence of continuing hospital admissions of patients with Asian rickets and osteomalacia led to a further attempt to provide more effective preventive measures for the Glasgow Asian community. Dose-response studies showed that the equivalent of 10 μg of vitamin D daily would provide effective prophylaxis, and a general practice survey showed that self-administered vitamin D supplements would reduce the prevalence and severity of Asian rickets. A multidisciplinary working group devised a preventive campaign based on the free issue of vitamin D supplements on demand to children who required them. Supported by a health education programme for community health personnel and the Asian community, the first 16 months of the campaign produced an eight-fold rise in the issue of supplements to older Asian children and a 33% increase in their issue to infants of all ethnic groups.

Because more children are receiving vitamin D supplementation the campaign seems likely to reduce the prevalence of Asian rickets in Glasgow.

Introduction

Rickets and osteomalacia remain common among Asians in Britain,[1-4] but prophylactic measures have been poorly organised and no account of an effective preventive campaign has been published. We describe the encouraging results of a campaign initiated by the Greater Glasgow Health Board in March 1979.

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Population data

PATIENTS DISCHARGED FROM HOSPITAL WITH NUTRITIONAL RICKETS AND OSTEOMALACIA

Altogether 138 Asians were discharged from Glasgow hospitals with a diagnosis of nutritional rickets or osteomalacia during 1968-78 (table I, fig 1). The Asian population of the city is estimated to have increased from about 8000 to 14 000 in this time. The annual incidence

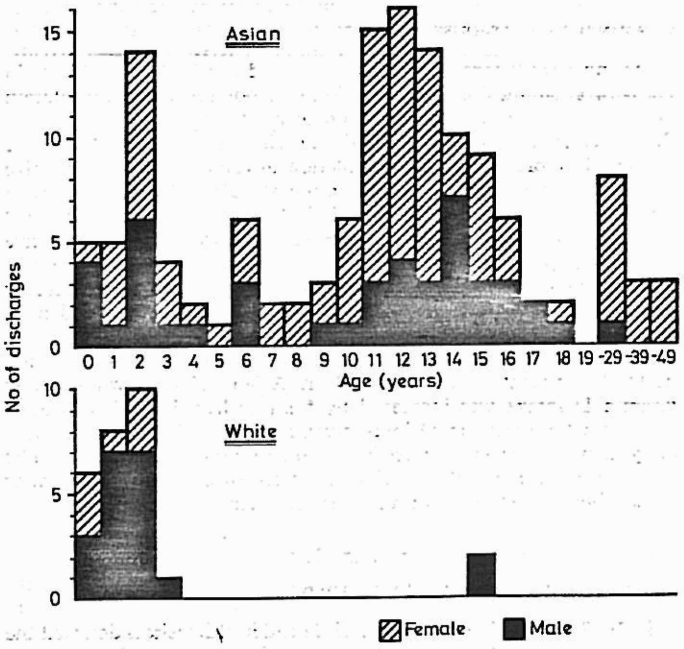

FIG 1—Numbers of white and Asian patients discharged from all Glasgow hospitals with nutritional rickets and osteomalacia between 1968 and 1978. Each case record was examined.