

MMPI (Hypochondriasis, Depression, Hysteria). Our age at onset effects were found on the Psychopathic Deviate scale and three of the so-called MMPI psychotic scales. As Kogeorgos *et al* used a measure which seems to be particularly sensitive to neurotic symptomatology (Crown-Crisp Experiential Index), one might reasonably expect to find little similarity between our results.

We speculated that there was a complex interplay between seizure type, age at onset, and duration of disorder in the predisposition to specific types of psychopathology. Further work will be needed to support or refute that particular contention.

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BENZODIAZEPINES AND EFFECTIVENESS OF ECT

DEAR SIR,

We note the remark in Dr Elia's letter (*Journal*, March 1982, **140**, 322) concerning the Northwick Park ECT trial that "Johnstone *et al*, write that improvement scores were similar in patients with and without diazepam". Since no such statement appears in our paper it seems that the puzzlement Dr D'Elia has experienced arises more from the discrepancy between the findings of the trial and his own expectations than from an obscurity in our account. In fact the statement quoted earlier in Dr D'Elia's letter that "improvement scores were similar in patients with and without diazepam" is correct. Eighteen of the 62 patients who finished the course (8 on real ECT and 10 on simulated) received diazepam either as 5 mg thrice daily or as diazepam 10 mg in occasional doses to relieve distress. All the patients in the trial received a benzodiazepine hypnotic (nitrazepam) and this is clearly stated.

If Dr D'Elia wishes to reject the conclusions we have drawn from our study on the grounds that hypnotic/sedative medication was not discontinued for the period of convulsive therapy he will find few studies of ECT which he will be able to accept. Many authors are vague on this issue. We have tabulated information on concomitant medication from a number of studies (Table).

TABLE

Concomitant drug therapy (sedative, anxiolytic or anti-depressant drugs given in addition to trial therapy) administered in trials of ECT

Information not given:

- Miller *et al* (1953)
Brill *et al* (1959)
Wittenborn *et al* (1962)
Wilson *et al* (1963)
McDonald *et al* (1966)
Smith *et al* (1967)
Cronin *et al* (1970)

Information incomplete:

- Harris and Robin (1960) —sodium amytal 3-6 gr when sedation was required
Robin and Harris (1962) —groups shown retrospectively to be comparable for 'additional treatment'
Fahy *et al* (1963) —barbiturate hypnotics were given for severe insomnia
Halliday *et al* (1968) —some patients given antidepressants of unstated dose and others not; no statement on other drugs; barbiturate sedation as required
Freeman *et al* (1978) —some patients given antidepressants of unstated type and dosage and others not; no comment on hypnotics or other sedatives
Lambourn and Gill (1978) —psychotropic drugs, except benzodiazepine hypnotics, were withdrawn
Taylor and Fleming (1980) —attempt made to withdraw additional medication apart from benzodiazepine hypnotics

Detailed information provided:

- Cronholm and Ottosson (1960) —insulin (16 cases); phenobarbitone 25 mg+0.16 g opium tincture three times per day (31 cases); promethazine (5 cases); meprobamate (3 cases); chlorpromazine (3 cases); amylobarbitone (1 case); no concomitant drugs (28 cases)
Herrington *et al* (1974) —an unsuccessful attempt was made to avoid hypnotic or sedative medication. A benzodiazepine hypnotic was finally given in 4 cases and diazepam in doses up to 30 mg/day in 11 cases
West (1981) —amitriptyline 50 mg at night given to all patients

Thus the view that hypnotic/sedative medication has in some way obscured the efficacy of ECT in the

Northwick Park trial can hardly be supported by evidence from trials that have been conducted without such medication. We frankly doubt that those who like Dr D'Elia have criticised our trial on these grounds maintain the consistency of their view by withdrawing from benzodiazepine or other sedative medication all those patients under their care whom they decide to treat with ECT.

However, the important point should not be overlooked that this objection does not in any case overcome the central problem posed by our findings to those who are unwilling to accept them. The limited (though significant) difference between real and simulated ECT groups depends not upon the fact that our real ECT patients did less well than those in comparable studies (which is not the case) but upon the fact that the simulated ECT patients improved almost as much.

Inappropriate sample selection could account for this finding and in the paper to which Dr D'Elia refers Kendell states that he considers the limited past ECT experience of our sample may indicate that our selection criteria were too wide. Recent detailed assessment of our findings (which we will shortly submit for publication) does not support this view.

We reiterate the conclusion expressed in our paper that electrically induced convulsions offer a significant advantage over simulated treatment in severe depressive states but this advantage is of lesser magnitude and more transient than has sometimes been claimed. It is clear to us that there are many like Dr D'Elia and Professor Kendell who are unwilling to accept our conclusions although the basis of their criticisms remains to us unconvincing.

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