ECT WITH BENZODIAZEPINES

DEAR SIR,

One aspect may be added to Kendell's excellent review of ECT (*Journal*, **139**, 265–83, 1980), *viz.* the increasing use—or rather misuse—of benzodiazepines together with ECT. Since there is strong evidence that cerebral seizure activity is essential for the antidepressive effect and since benzodiazepines have anticonvulsant properties, the combination is irrational. As shown by Strömgren *et al* (1980) patients with benzodiazepines have shorter average seizure duration and more submaximal seizures compared to patients of similar age without benzodiazepines. Accordingly, the benzodiazepine–ECT combination had a lower antidepressive effect, which was shown by three more treatments being required on an average.

The same issue is valid for at least one of the new batch of comparisons between simulated and real ECT (Johnstone *et al*, 1980). In this all patients had a benzodiazepine hypnotic every night and additional diazepam was prescribed if required. The long half life of these drugs guarantees an anticonvulsive effect. There was no recording of the seizures and an assurance that a fit was always evoked is meaningless since it may well have been submaximal.

If so called real ECT is given in an irrational way any comparison between such treatment and simulated ECT is misleading. With sufficiently high doses of benzodiazepines real ECT becomes simulated ECT. The negative interaction of ECT and benzodiazepines should be made known in all treatment centers.

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References

JOHNSTONE, E. C., DEAKIN, J. F. W., LAWLER, P. *et al* (1980) The Northwick Park electroconvulsive therapy trial. *Lancet*, *i*, 1317–20.

KENDELL, R. E. (1981) The present status of electroconvulsive therapy. *British Journal of Psychiatry*, **139**, 265–83.

STRÖMGREN, L. S., DAHL, J., FJELDBORG, N. & THOMSEN, A. (1980) Factors influencing seizure duration and number of seizures applied in unilateral electroconvulsive therapy. *Acta Psychiatrica Scandinavica*, **62**, 158–65.

SMOKING PROFILES OF PATIENTS ADMITTED FOR NEUROSIS

DEAR SIR,

We would like to respond to the letter from M. R. Eastwood (*Journal*, January 1982, **140**, 102).

He says, "Examination of Table III shows that the only significant difference between contrast groups with respect to age at starting to smoke was due to the disproportionately small number of neurotics in the older group". Precisely. The disproportionately small number in the older group is because those who are smokers have started in a much younger age group, leaving only a small number to start later. Only 49.7 per cent of males in the general population have started to smoke by 17 years, but 63.7 per cent of the neurotic males have done so. This obviously leaves a much smaller proportion of neurotics to start smoking later—only 8.1 per cent against 22.5 per cent of the general population. Of course, nearly all those (both neurotics and general population) who are going to smoke have taken up the habit by the age of 30—very few take up smoking after this age (Lee, 1976).

The same holds true for females—23.6 per cent of the general population start below 17 years but 33.3 per cent of neurotics do so. However, this does leave a larger proportion of neurotic women to start smoking after eighteen years.

There is an error in Table VI. In fact, 60 per cent of the surgical group and 78 per cent of the neurotic group of smokers inhaled deeply—the raw figures are actually 7.0 and 41.0

We agree that smoking is not a panacea for neuroticism. However, it would seem that neuroticism is a factor in the initiation and continuation of the smoking habit.

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Reference

LEE, P. N. (ED.) (1976) *Statistics of Smoking in the United Kingdom*. London: Tobacco Research Council.

ALCOHOLISM, DEPRESSION AND PLASMA FOLATE

DEAR SIR,

There are numerous reports of reduced folate concentration in the serum of patients with a depressive illness (Carney, 1967; Hunter *et al*, 1967). In a previous study we found that nearly one-quarter of depressive patients had a low serum folate on admission to hospital and that these patients responded less well to treatment than did patients with normal serum folate (Reynolds *et al*, 1970). In a recent study on outpatients with affective disorders maintained on lithium, we found that those with a low serum folate concentration had significantly higher morbidity than patients with higher folate levels (Coppen and Abou-Saleh, 1982).

These findings prompted us to examine serum folate concentration in patients admitted to a Regional Alcoholism Unit.

Forty-one patients suffering from chronic alcoholism and who satisfied the criteria for the diagnosis of alcohol dependence syndrome (WHO ICD-9, 1978) were studied. These patients were admitted when they were 3–6 weeks abstinent from alcohol after having been detoxified as inpatients in other wards or as outpatients. During their stay they received group psychotherapy and social rehabilitation. While they were being treated they completed the Beck Depression Inventory (Beck *et al*, 1961), and the Severity of Alcohol Dependence Questionnaire (Stockwell *et al*, 1979). Blood was taken on the same day for the estimation of plasma folate. Plasma folate was estimated using a Quanta-Count Folate Kit (Bio-Rad Laboratories, Watford, U.K.).

In line with a previous report (Theano, 1977), the plasma folate of 41 alcoholic patients (mean ± S.E. = 6.9 ± 0.5 ng/ml), did not differ from that of a control group of 60 normal subjects with no history of alcoholism or psychiatric illness (7.0 ± 0.3 ng/ml). However, by dividing the patients into those with a folate level above ⩾7 ng/ml) and below the mean (<7 ng/ml) a striking difference in depressive morbidity was revealed (Table I). The mean morbidity score (BDI) for the low folate group was approximately twice as high as that for the high folate group; the SADQ scores did not differ significantly. There was no difference in the liver function tests in these two groups.

TABLE I

Plasma folate, Beck Depression Scores (BDI) and Severity of Dependence Questionnaire (SADQ) scores in alcoholic patients

Plasma folate	n	BDI	SADQ
<7 ng/ml	23	*21.0±2.2	33.2±2.7
⩾7 ng/ml	18	10.8±1.9	30.1±2.9

(Results as means ± S.E.).

* Patients with serum folate <7 ng/ml had significantly higher BDI scores. (P <0.001).

The more severely depressed patients may have had a poorer diet than the less depressed patients, and this may account for their lower plasma folate levels. In addition, lower folate levels may decrease brain 5-hydroxytryptamine (5-HT) formation as it has been shown that rats fed a folate-deficient diet show a decreased 5-HT synthesis (Botez *et al*, 1979).

There is a good deal of evidence that 5-HT is decreased in depression (Coppen, 1967) and, hence, the low plasma folate in these patients may, in turn, contribute to the severity of their affective disturbance.

Replacement therapy with folic acid should be undertaken with physiological doses (300 μg a day) rather than with the larger doses that are normally given. Animal work on folate deficient rats has shown that large pharmacological doses of folic acid inhibit 5-HT formation. Furthermore, it should be noted that pharmacological doses of folic acid have been reported to cause mental disturbances in man (Hunter *et al*, 1970).

The relationship between plasma folate concentration and depressive symptomatology is intriguing and merits further investigation.

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References

BECK, A. T., WARD, C. M., MENDELSON, M., MOCK, J. & ERBAUGH, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–71.

BOTEZ, M. I., YOUNG, S. M., BACHEVALIER, J. & GAUNTHIER, S. (1979) Folate deficiency and decreased brain 5-hydroxytryptamine synthesis in man and rat. *Nature*, **278**, 182–3.

CARNEY, M. W. P. (1967) Serum folate values in 423 psychiatric patients. *British Medical Journal*, *iv*, 512–16.

COPPEN, A. (1967) The biochemistry of affective disorders. *British Journal of Psychiatry*, **113**, 1237–64.

—— & ABOU-SALEH, M. T. (1982) Plasma folate and affective morbidity during long-term lithium therapy. *British Journal of Psychiatry* (in press).

HUNTER, R., JONES, M., JONES, T. G. & MATTHEWS, D. M. (1967) Serum B_{12} and folate concentrations in mental patients. *British Journal of Psychiatry*, **113**, 1291–5.

—— BARNES, J., OAKLEY, H. F. & MATTHEWS, D. M. (1970) Toxicity of folic acid given in pharmacological doses. *Lancet*, *i*, 61–3.

REYNOLDS, E. H., PREECE, J. M., BAILEY, J. & COPPEN, A. (1970) Folate deficiency in depressive illness. *British Journal of Psychiatry*, **117**, 287–92.

STOCKWELL, T., HODGSON, R., EDWARDS, G. & RANKIN, H. (1979) The development of a questionnaire to measure severity of alcohol dependence. *British Journal of Addiction*, **74**, 79–87.

THEANO, G. (1977) Do alcoholics need vitamins? *British Journal of Psychiatry*, **131**, 326–7.