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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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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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.