before that time, led me to propose a nervous system model for schizophrenia which was essentially 'vertical' in conception, as distinct from the 'horizontal' models dictated by hemisphere research. The crucial feature of the model was that schizophrenia is essentially a state of central nervous imbalance which may take one of two major forms, depending on the direction of 'dissociation' of CNS function. One is led to wonder whether the many examples of cerebral asymmetry now being reported in schizophrenia are merely another manifestation of such imbalance and are in themselves not fundamental to the disease. The problem of replication experienced in much earlier psychophysiological research on schizophrenia was, I suspect, not due to investigators neglecting the hemisphere 'dimension', but to other methodological inadequacies, such as their almost universal use of medicated patients—an error which it is encouraging to see Gruzelier and Manchanda did not commit.

GORDON CLARIDGE

University of Oxford,
Department of Experimental Psychology,
South Parks Road,
Oxford OX1 3UD

LOW AND HIGH ENERGY ECT

DEAR SIR,

In their paper on the therapeutic effects of low and high energy ECT (*Journal*, October 1982, **141**, 357–66), Drs Robin and de Tissera base their conclusion that energy dosage is crucially related to therapeutic response in ECT on the fact that average convulsing time as measured by the naked eye was closely similar for their low energy pulse, high energy pulse and high energy sinusoidal wave ECT groups.

It has been clearly shown, however, that the naked eye measurement of ECT seizure activity is a totally inadequate measure of the brain seizure activity. Blachly and Gowing (1966) found that EEG evidence of seizure activity persisted long after muscular evidence of the seizure had stopped. Sørensen *et al* (1981) found that electromyographically monitored seizure activity lasted between 43 per cent and 89 per cent of the duration of the EEG-monitored seizure and that the EMG/EEG ratio varied widely between treatments in individual patients. Christensen and Koldbaek (1982) found that only 26 per cent of the variance in clinically observed seizure duration could be accounted for in terms of EEG-monitored seizure duration.

Robin and de Tissera discount Maletzky's (1978) work relating EEG seizure duration and the therapeutic efficacy of ECT on the grounds that Maletzky 'was essentially counting treatments in an unnecessarily elaborate way'. This view is incompatible with Maletzky's observation that 'several patients receiving 2–3 stimulus presentations, but with very long subsequent seizures, generally improved, whereas several other patients with many seizures but each of short duration, failed to improve . . .'. In view of the established clinical efficacy of Fluorothyl-induced seizures (Small, 1974) in which electrical energy is not involved at all, and the demonstration by Rosenthal, Macey and Timiras (1962) of a linear relationship between log stimulus intensity and seizure duration in rats receiving electroshock, it seems likely that true seizure duration, rather than energy dosage, determined clinical outcome in Robin and de Tissera's patients.

We have previously suggested (Berrios and Katona, 1982) that EEG-monitored seizure duration may be a useful mediating variable in studies correlating input variables with the clinical outcome of ECT. The studies cited above have used the MECTA apparatus. An alternative method which does not necessitate replacing the ECT apparatus is the EEG protection unit described in our paper (details available from authors).

Energy dosage may indeed prove to be of importance in ECT but until information is available relating energy dosage to EEG-monitored seizure duration the case against the central therapeutic role of induced seizure activity in ECT remains unproved.

C. L. E. KATONA
G. E. BERRIOS

Fulbourn Hospital,
Cambridge CB1 5EF

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