extant scales is a serious drawback to rational choice and we still claim to have provided some useful information on which to base this choice. We do not think our claims were over bold or our study unduly flawed.

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FOLATE, AFFECTIVE MORBIDITY AND LITHIUM THERAPY

DEAR SIR,

In their paper (*Journal*, July 1982, **141**, 87–9) Coppen and Abou-Saleh report significantly lower plasma folate concentrations in the lithium-treated patients than in the control subjects. Unfortunately, however, the validity of their observation is impaired in the absence of the pre-lithium folate values. The baseline data are important particularly because there is some evidence to suggest an interaction between lithium and folate metabolism (Herbert and Colman, 1980; Prakash *et al*, 1981). Besides, the control group does not appear to have been matched with the sample. The authors have also not commented on their findings of folate concentrations in the unipolar patients (N = 81) who not only constituted a larger but also more important subgroup of the sample because folate deficiency has been reported more frequently in depression than mania (Shulman, 1979).

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DEAR SIR,

Dr Prakash's comment that the validity of our observation is impaired in the absence of the pre-lithium folate values seems unjustified. The underlying assumption is that lithium therapy *per se* could have caused the relative reduction in plasma folate concentration in these patients, but evidence supporting such a contention can only be described as anecdotal. Nevertheless, we fail to see how such an assumption could explain the observed association between low folate concentrations and high affective morbidity in these patients.

As regards his second point, we agree that these patients were not perfectly matched with the control group. Age makes no contribution to low plasma folate levels observed in psychiatric patients (Carney, 1979). In our patients, low, medium and high plasma folate groups had similar sex distributions with proportions of males to total of 37 per cent, 33 per cent and 40 per cent respectively.

As regards the last point, our data suggest that plasma folate levels are also reduced in mania.

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SCHIZOPHRENIA AND LATERALIZATION OF GALVANIC SKIN RESPONSE

DEAR SIR,

Perhaps I may be allowed to comment on the recent paper by Gruzelier and Manchanda (*Journal*, November 1982, **141**, 488–95) in which they report that the direction of lateralization of the galvanic skin response differentiates two forms of schizophrenia, a retarded, emotionally withdrawn form and a type characterized by florid delusional symptoms and emotional reactivity. In their discussion the authors comment that such a subdivision has rarely in the past produced 'decisive psychophysiological and behavioural differences'.

Fifteen years ago, in my book *Personality and Arousal*, I demonstrated an almost identical dichotomy of the schizophrenias, revealed in the clustering of certain psychophysiological and psychological test measures in drug-free patients. I draw attention to this not to detract from the results reported by Gruzelier and Manchanda—which are indeed impressive—but to illustrate that the clinical typology they describe can be arrived at without reference to the notion of hemisphere dysfunction which is currently capturing interest as a possible neurophysiological basis for the psychotic states. My own work, which was carried out

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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.

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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.

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