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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Reference

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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