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SERUM CREATINE KINASE IN ACUTE PSYCHOSIS

DEAR SIR,

In recent years there have been many published reports of increased levels of serum CPK during acute psychotic episodes. The interpretation of these findings is controversial. Meltzer (1976) thinks that the increased serum CPK values are caused by some as yet undefined processes which are also causative for the acute episode. Other authors (Cunningham *et al*, 1975; Harding, 1975) think that the rise in CPK values is due to factors which are not directly connected with the acute psychotic episode, and that therefore the serum CPK levels are of value neither diagnostically nor predictively. Soni (1976) very recently proposed that the increase in serum CPK in psychotic states is related to the increased psychomotor activity, but points out that the relationship is not a simple one and depends on the amount of muscular effort the subject is accustomed to make.

We have approached this problem from the observation of Meltzer that in the first-degree relatives of patients with acute psychotic disease one finds small but persisting elevations in serum CPK levels (75–140 IU/litre). Even though it is difficult to connect this finding with the return to normal levels in the psychotics after a few weeks and with the increased levels seen in chronic patients, the study of relatives rather than of patients offers the advantage of providing a correction factor for the non-psychiatric factors. It must also be remembered that there are genetic factors in psychosis. We have therefore measured the serum CPK levels, under standard conditions, in bloods taken over a period of two months from six patients with primary affective disorders—during a normothymic period—and in at least two of their first-degree healthy relatives. Our data do not agree quantitatively with those of Meltzer, since our values ranged between 20 and 85 IU/litre. Qualitatively they do agree with his findings in four of our six families, in which both relatives had values higher than their respective ill relatives. Of the two remaining families, in the first, one relative had a higher CPK level and one a lower level than the ill member, and in the second, both had lower levels than the ill member.

Obviously, these data indicate that further studies must be carried out with more sophisticated experimental designs taking into account the differing homogeneous diagnostic clusters among the acute psychotic subjects, at least differentiating between PAD subjects and those with the various types of schizophrenia. Undoubtedly it will be of value to study the first-degree relatives along with the subjects, but attention must also be paid to any non-psychiatric differences that may interfere with the interpretation of the results.

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MONOSYMPTOMATIC HYPOCHONDRIASIS

DEAR SIR,

Bebbington (1) has described the heterogeneous nature of conditions subsumed under this heading, and has stated the generally accepted view that such disorders have a uniformly poor prognosis. We believe that one important sub-group may be amenable to successful treatment. Riding and Munro (2) and Reilly (3) have found that mono-