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DEAR SIR,

Some acutely psychotic patients have raised serum creatine kinase (CK) levels.

Is this finding of any significance in understanding the pathogenesis or course of acute psychosis?

Does it provide a useful diagnostic or predictive method?

I believe that the answer to both these questions is 'Probably, no', and none of Professor Meltzer's interesting observations convinces me otherwise. Cunningham *et al.* (1974) have also concluded that observed serum CK elevations are due to 'non-psychiatric factors' and that serum CK estimation was not useful as a diagnostic or predictive test.

Professor Meltzer refers to his earlier proposal that a similar mechanism underlies serum CK increases in patients with acute psychoses and patients with acute brain diseases. It may well be that similar non-specific factors operate. Serum CK elevations occur in unconscious patients without local brain disease, as in hepatic coma (Schiavone and Kaldor, 1965) and drug overdose (Wright *et al.*, 1971) when leakage of the enzyme from muscle due to local damage and catabolism is probably responsible. In acutely psychotic patients a number of non-specific factors may operate additively to produce such a rise. Hyperactivity is only one such factor; profound hypoactivity, change in appetite and many others should also be considered. Professor Meltzer's own observation that psychiatric patients with more 'florid psychotic symptoms' have higher serum CK levels would be in line with such a view and the fact that such patients require higher doses of medication is in itself hardly surprising. Serum CK levels are simply a reflection of the rate of leakage of the enzyme from striated and cardiac muscle where it is present in large amounts. Muscle diseases and myocardial damages are examples of conditions in which serum CK elevations are specifically related to the underlying disease process. Acute cerebral diseases, drug overdose, walking from London to Brighton, deprivation of sleep and acute psychoses are probably conditions in which non-specific factors are responsible for increased release of the enzyme from muscle.

Professor Meltzer does not mention the possible diagnostic or predictive usefulness of serum CK estimations in his letter, although he has advocated it in the past (Meltzer, 1969). Even on the basis of the findings he quotes in his letter, the test would have insufficient sensitivity and his observation that raised serum CK levels are more likely in patients with florid psychotic symptoms confirms my own finding that those patients who do display such elevations pose few diagnostic problems as far as differentiating between psychotic and non-psychotic illness is concerned.

Finally, I must apologize for the drafting error to which Professor Meltzer has drawn attention. The finding of Smith *et al.* (1970) was that 25 out of 300 healthy ambulant males had serum CK levels above 100 I.U./L (not, as I wrote, 300 I.U./L, which is clearly inconsistent with the following sentence). The statistical comparison was not, of course, based on the findings of Smith and his colleagues but the fact that over 8 per cent of a group of healthy men had 'raised serum CK levels' is relevant to the discussion.

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EFFECTS OF SMALL ELECTRICAL CURRENTS UPON DEPRESSIVE SYMPTOMS

DEAR SIR,

We read with interest the paper of Nias and Shapiro in your issue for October 1974 on the effects of small electrical currents upon depressive symptoms. Their findings of similar effects produced by opposite

current polarity has prompted us to review our original data on normal subjects (Sheffield and Mowbray, 1968), to see if any of our subjects showed a consistently opposite reaction to the others in the trial. In our study, scores on the 'Clyde Mood Scale' (the closest parameter to that measured by Nias and Shapiro), showed an apparent effect of the current which was not statistically significant. However, on analysing individual scores in our data there was no individual who reacted consistently in an opposite direction to the general trend for each item.

Another interesting point is that we also encountered the same difficulties regarding the itching under one electrode which made double blind conditions of the trial a little more difficult to control. Surprisingly in our case it was consistently the positive electrode applied to the forehead and not the negative electrode as described by Nias and Shapiro.

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HALOPERIDOL IN THE TREATMENT OF STUTTER

DEAR SIR,

Recent trials of haloperidol in the treatment of stutter (Refs 1, 2, 3) have evoked widespread interest and as expected have been followed by cautionary tales. Faced with a stutterer demanding to be put on the 'new' treatment, what should the therapist do in the light of current knowledge?

The position at present seems to be this—these three trials were all controlled studies, but samples were small. The results seem to bear a direct relationship to the mean dosage employed for each trial and for how long it was given (see Table overleaf).

In our own study (1) the dosage was increased in weekly steps to a final dose of 4·5 mg. daily at the beginning of the third week. This dose was maintained for six weeks. The first assessment, however, was made at the end of the fourth week, after patients had taken the maximum dose for two weeks. (The significant results obtained at four weeks were maintained at eight weeks but had not improved further). Three-year follow-up of this two-month study showed stutterers to have maintained some improvement—in one dimension of three measured the improvement was significant.

Swift, Swift and Arellano in their three-week study reached a peak dose of 3·5 mg. daily, which was only maintained for one week before the assessment of progress was made. Results showed significant improvement in 6 out of 7 patients with stutter, all of whom relapsed within two weeks of discontinuing the trial.

Quinn and Peachey in their three-week study gave a mean dose of 2·5 mg. daily but do not say whether this was given throughout the three-week trial period. Four out of 18 patients were substantially improved and 6 others improved in lesser degrees, but none of their results reached statistical significance.

Although these studies were not strictly comparable, the following comments can be made:

1. The effective dose of haloperidol in the treatment of stutter in most cases seems to be 3·5 mg. daily.

2. The maximum effect seems to be reached after two weeks on the effective dose. Maintenance dose, however, may be lower.

3. Individual response to the drug is variable. It is probably prudent to build up to the more effective dose by weekly increments.

4. The incidence of side effects is high and calls for weekly supervision of patients during the first 4 or 5 weeks.

5. Clinical impression suggests treatment should be continued at least two months—our three-year follow-up of patients whose stutter had been treated successfully with haloperidol for two months (though not cured) showed that the improvement had been maintained, although in only one dimension of three measured did this reach statistical significance.

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