expressiveness for male and female, congruence for slow and fast speakers, and objective focused movements for fast speakers). Furthermore, the interview with the stranger was apparently later than the interview with the spouse and some degree of recovery would have been expected.

If anything, these figures show that depressive behaviour is *not* consistently reduced by changing the social environment.

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SERUM CREATINE KINASE IN ACUTE PSYCHOSIS

DEAR SIR,

In a recent report in the *Journal* (1974, **125**, 280), Harding reported that 5 out of 34 acutely psychotic patients had increased serum creatine phosphokinase (CPK) activity (>100 IU/L). They compared this result to the study of Smith *et al.* (1970), in which, according to Harding, 25 normal subjects were found to have serum CPK levels greater than 300 IU/L. Since this latter figure is far in excess of any previous report of serum CPK activity in the normal population (Rosalki, 1967; Meltzer, Elkun and Moline, 1969), I checked the report of Smith *et al.* (1970) and found that serum CPK levels greater than 300 IU/L were found in only two of 296 subjects and none had serum CPK levels between 200–300 IU/L.

It is unfortunate that Harding did not report his data in acutely psychotic patients in relation to the time of onset of their psychotic symptoms. He states only that the onset was less than one month before admission. We have found that the incidence of increased serum CPK levels at admission in those psychotic patients with onset of gross psychotic symptoms less than seven days before admission (54/98) is significantly greater than those with onset greater than seven days (35/105; Chi-square, Yates' correction $= 8{\cdot}892$, $p < 0{\cdot}005$). The incidence of CPK increase in psychotic patients with symptoms greater than two weeks in our series is about 15 per cent, which is similar to the find of Harding (1974). The mean duration of the serum CPK increase in psychotic patients in our series is $3{\cdot}8 \pm$S.D. $3{\cdot}1$ days. Thus, duration of illness is a key factor in studying serum CPK levels in psychotic patients, just as it is in patients with myocardial infarction and cerebrovascular accidents, head injuries or infections (Roe *et al.*, 1972; Dubo *et al.*, 1967).

Harding believes that I have not sufficiently attended to the effects of activity on serum CPK activity in my previous studies and cites the finding of Griffith *et al.* (1966) that an 87 km. walk raised serum CPK activity markedly. Such massive activity is not characteristic of psychotic patients in my experience. Harding himself states that some psychotic patients with decreased motor activity had increased serum CPK levels. We have reported the same (Meltzer, 1969). We have specifically studied the effects of exhaustive isotonic exercise (Meltzer and Moline, 1969) and isometric exercise (Goode, D. and Meltzer, H. Y., in preparation) and found relatively small increases in serum CPK activity compared to those at the time of an acute psychotic period.

The increases in serum CPK levels in acute psychotic patients are comparable to those in patients with a variety of acute brain diseases in duration, magnitude, source, and percentage of patients with increases (Dubo *et al.*, 1967; Wolintz *et al.*, 1969). In these latter patients, there is no possibility that increased motor activity is the cause of the increases in serum CP levels. We have proposed that a similar mechanism underlies the serum CPK increase in patients with acute psychoses and patients with known acute brain diseases (Meltzer, 1969). Our current studies suggest that the psychiatric patients with increased serum CPK levels have more florid psychotic symptoms, require higher doses of medication and longer stay in hospital than those without increased serum CPK levels.

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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

https://doi.org/10.1192/bjp.126.5.489 Published online by Cambridge University Press