CORRESPONDENCE

CREATININE PHOSPHOKINASE IN ACUTE PSYCHOSIS

To the Editor:

There is disagreement as to whether or not serum creatinine phosphokinase (CPK) levels are elevated in acute psychotic disorders (Weiner, 1980). A recent case of ours suggests the possibility of viewing the problem in a new way.

A 32-year-old housewife was admitted on December 12, 1980, displaying incoherence of speech, restlessness, persecutory and other delusional ideas, and feelings that her thoughts were being broadcast. A diagnosis of schizophrenia was established. Remission was promptly induced with a drip infusion of haloperidol, and subsequently maintained with oral haloperidol, and later with chlorpromazine, until February 8 (Day 57), when she suddenly developed a second acute episode with agitation, incoherence of speech, and the delusion that the doctors and nurses in the hospital were disguised patients, and that the patients were disguised doctors and nurses. This episode was again successfully handled with a drip infusion of haloperidol.

The serum CPK level in this case was periodically measured (Fig. 1). It is interesting to note that CPK was at the high level of 287 IU/liter (normal range 25-120 IU/liter) on the day of admission, and then, as expected, gradually dropped to the normal range. But it did not rise at all during her second episode of acute schizophrenia.

This case suggests the possibility that an elevated CPK level may be found in an initial acute episode but not in a subsequent episode. A systematic research project is now in progress to test this hypothesis.

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January 26, 1983
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REFERENCE


SCHIZOPHRENIA, AMINO ACIDS, AND CELIAC DISEASE

To the Editor:

Several years ago I wrote a letter to Biological Psychiatry about a possible clue to the etiology of schizophrenia and celiac disease (Manowicz, 1978) pointing out that the amino acid levels in schizophrenic patients as reported by Perry et al. (1969) are correlated positively with amino acid levels in celiac disease patients as reported by Douglas and Bueth (1969) after adjustments are made for control values. This is of interest because of the theory postulated by Dohan and Gruberberger (1973) that there may be common factors in the etiology of these two diseases.

Simpson (1982) has published further analyses of these data indicating that the observed positive correlation can be attributed to the inclusion of glutamic acid in the