forms of depressive illness, but we have consistently failed to do so.

R. E. KENDELL.
FELIX POST.

The Maudsley Hospital,
Denmark Hill,
Camberwell,
London SE5 8AZ.

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CREATINE PHOSPHOKINASE ACTIVITY IN PSYCHIATRIC PATIENTS

DEAR SIR,

Gosling *et al.*, in your *Journal* (1), found that half of their newly-admitted psychotic patients had raised creatine phosphokinase (CPK) activity. This was specifically associated with the psychosis: none of their neurotic control patients showed an elevated CPK.

We have investigated newly-admitted female patients to the psychiatric ward of the Johannesburg General Hospital during a period of 30 days. This ward admits unselected cases across the whole psychiatric spectrum. Patients were only excluded from this investigation if they had received intramuscular chlorpromazine, as this can raise CPK values (2). The CPK assay was carried out by means of the *accu-zyme enzyme reagent* kit. Normal range for females is 0 to 30 I.U. Clinical assessment of the patient's condition was carried out independently by two psychiatrists, who were asked to allocate the patient to a psychotic or non-psychotic group. Where there was disagreement on the allocation (3 cases), the cases were omitted (the CPK levels for these three cases were within normal limits).

Details are set out in the following table:

TABLE

	Age	Clinical diagnosis	CPK I.U.
		Psychotic group	
1.	56	Manic-depressive psychosis (depressed phase)	29·9
2.	51	Manic-depressive psychosis (manic phase)	28·7
3.	49	Schizo-affective disorder	21·1
4.	36	Schizophrenia	30·4
5.	57	Presenile dementia	23·4
6.	16	Schizophrenia	26·7
7.	23	Schizo-affective disorder	28·4
8.	32	Schizophrenia	65·0
		Non-psychotic group	
1.	60	Anxiety state and hypochondriasis	29·3
2.	65	Personality disorder and alcoholism	24·6
3.	22	Neurotic depression	56·5
4.	31	Anxiety state	58·4
5.	19	Personality disorder and epilepsy	31·8
6.	46	Personality disorder	53·6
7.	70	Toxic confusional state	31·0
8.	21	Neurotic depression	28·4
9.	28	Neurotic depression	26·6
10.	25	Dissociative hysterical illness	30·0

The mean CPK level of the psychotic group was 31·7 (S.D. 13·8) and of the non-psychotic group 37·0 (S.D. 13·4). This difference is not statistically significant.

We have therefore failed to confirm that raised CPK activity indicates the presence of psychotic illness. In those cases showing abnormal levels the elevations were small. We suggest that patients manifesting elevated CPK levels may share a common factor, but that this is not the presence *per se* of psychosis. This factor may be a subclinical myopathy (3), a possibility further supported by the finding (4) that the raised serum CPK is of the muscle isoenzyme type. Furthermore, it is interesting that Meltzer (4) noted a familial tendency, in that some parents of psychotic patients had continuous CPK elevations while the patients themselves only demonstrated these intermittently.

J. P. LOEBEL.
A. H. ROBINS.

Department of Psychiatry,
Johannesburg General Hospital and the
University of the Witwatersrand,
Johannesburg, South Africa.

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TARDIVE DYSKINESIA

DEAR SIR,

I am writing in reference to the article on Tardive Dyskinesia published in your December 1972 issue (**121**, 605–12). This is a subject which continues to command interest. Part of the problem is that dyskinetic phenomena may occur even early in treatment. I have seen them within six months of instituting neuroleptic treatment. However, in these instances it disappears with the discontinuation of medication. The question of the incidence of permanent neurological impairment remains an unanswered one, as does the treatment. Three points seem to be of importance in this area:

(i) Tardive dyskinesia disappeared in three manic patients (erroneously diagnosed as schizophrenics and treated with neuroleptics) when they were changed to lithium therapy. In other non-manic cases, improvement of symptoms took place when the patients were given lithium. These have been uncontrolled studies, but are of sufficient interest to warrant further investigation.

(ii) In the past year I have seen several other patients, not schizophrenics, who presented with severe tardive dyskinesia when receiving neuroleptics. One was a patient who had received 100 mg. of thioridazine daily for 10 years. He had a well-marked bucco-lingo-masticatory syndrome which disappeared when medication was withdrawn. More important was the appearance of a crippling tardive dyskinesia in a neurotic patient who had received no more than 10 mg. of trifluperazine daily for less than a year. It did not remit upon withdrawal of medication. I have also seen two involutional depressives who were treated with neuroleptics, both of whom were unable to dress themselves or function in the outside world because of their dyskinesias. These observations have important implications in terms of the use of neuroleptics for treating neurotic conditions and particularly for treating depressions. There is a considerable literature suggesting that neuroleptics are a good treatment for certain types of depression, and it has also been stated that neuroleptics are the treatment of choice for 'agitated depression' (1). On the basis of the above-listed cases, I would say that neuroleptics are contraindicated in the treatment of such patients, since there are no data to suggest that these are better than antidepressant medication, which certainly does not produce tardive dyskinesia.

(iii) The third and last point relates to why some patients on small dosages of medication should develop this syndrome. The last three of the patients mentioned above received medication for a short period of time only, and certainly did not ingest anything like the large amounts that chronic schizophrenics may have received. All were female and all were of Eastern European Jewish background. The question therefore, arises as to whether, as with the congenital dystonias, Eastern European Jews may have an increased susceptibility to this syndrome. It has even been suggested that the administration of small amounts of neuroleptics may be a suitable way to detect the heterozygous carriers of congential dystonias (2).

GEORGE M. SIMPSON.

Research Center,
Rockland State Hospital,
Orangeburg,
N.Y. 10962, U.S.A.

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BENIGN MYALGIC ENCEPHALOMYELITIS

DEAR SIR,

May I be permitted to draw attention to the fact that the paper entitled 'A controlled follow-up of cases involved in an epidemic of Benign Myalgic Encephalomyelitis' by Drs. McEvedy and Beard (*Brit. J. Psychiat.* (Feb. 1973), **122**, 141), is merely a prolongation of the thesis they submitted through the columns of the *British Medical Journal* of 3 January 1970. In a letter to the *B.M.J.* (1970, **1**, 362) Drs. N. Compston, H. Dimsdale, A. T. Richardson and myself pointed out that while a diagnosis of hysteria had been seriously considered at the time of the outbreak, the occurrence of fever in 89 per cent, of lymphadenopathy in 79 per cent, and of ocular palsy in 19 per cent, rendered it quite untenable. In the same issue Dr. E. D. Acheson, who had personal experience of cases at the Middlesex Hospital, stated that he too had considered a possible diagnosis of hysteria but for similar reasons had ruled it out. Most important evidence favouring our view that the condition was infective in origin was the occur-