psychotic versus non-psychotic depression has not been systematically studied. We therefore examined B12 and folate levels in unmedicated patients with well-defined major depression to determine the association between psychotic depression and serum levels of these vitamins.

Of 53 patients presenting to the Mood Disorders Clinic with a major depressive episode as defined by Research Diagnostic Criteria (Spitzer *et al*, 1977) generated from the Schedule for Affective Disorders and Schizophrenia — Lifetime Version (Spitzer *et al*, 1978), five patients (9%) had psychotic depression and 48 patients had major depression alone. The mean B12 concentration, measured by radio-immunoassay (Quantphase, Bio-rad, California), for the psychotic group was 181.6 ± 57.3 pmol/l (range 107.0 ± 266.0 pmol/l), while the mean B12 for the non-psychotic group was $316{\cdot}7 \pm 105.4$ pmol/l (range 139.0–574.0 pmol/l) (normal range = 110–630 pmol/l). Using *t*-tests, there was a statistically significant difference between the two groups for B12 levels ($t = 2.8$, d.f. = 51, $P < 0.01$) but not for folate levels. Furthermore, when several clinical and behavioural variables such as age at onset and duration of depressive illness were entered into a multiple regression with B12 as the dependent variable, the presence or absence of psychosis contributed significantly to variance in B12 ($R^2 = 0.13$, d.f. = 48, $P < 0.01$).

Patients with psychotic depression may have a lower B12 level than non-psychotic patients. This confirms previous findings that low B12 is associated with mental disturbance (Shovron *et al*, 1980). This is the first report of which we are aware of a specific association between psychotic depression and lower B12. We have previously shown (Levitt & Joffe, in preparation) that B12 is not associated with duration of current depression or weight and appetite changes in depression. In addition, B12 depletion may take many months. It is therefore unlikely that nutritional deficit secondary to the anorexia of current depression is primarily responsible for the lower B12. Although low B12 may sometimes result from low folate, we did not find a significant difference in folate levels between the psychotic and non-psychotic depressives. Another possible explanation for this difference is that lower B12 predisposes to the development of psychotic symptoms during a depressive episode. This hypothesis needs to be tested on a large population with repeated measures of B12 after recovery.

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References

EVANS, D. L., EDELSON, G. A. & GOLDEN, R. N. (1983) Organic psychosis without anaemia or spinal cord symptoms in patients with vitamin B12 deficiency. *American Journal of Psychiatry*, **140**, 218–221.

SHOVRON, S. D., CARNEY, M. W. P., CHANARIN, I. & REYNOLDS, E. H. (1980) The neuropsychiatry of megaloblastic anaemia. *British Medical Journal*, **281**, 1036–1038.

SHULMAN, R. (1967) Vitamin B12 deficiency and psychiatric illness. *British Journal of Psychiatry*, **113**, 252–256.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1977) *Research Diagnostic Criteria for a Selected Group of Functional Disorders (ED 3)*. New York: New York State Psychiatric Institute.

SPITZER, R. L. & ENDICOTT, J. (1978) *Schedule for Affective Disorders and Schizophrenia — Lifetime Version (SADS-L)* (3rd edn). New York: New York State Psychiatric Institute.

Clinical Dementia Rating

SIR: The Washington University Clinical Dementia Rating (*Journal*, 1982, **140**, 566–572) has been widely adopted. A revision of this staging scale was published in a Letter to the Editor (*Journal*, 1984, **145**, 339).

In order to describe more precisely the rating of questionable dementia (CDR 0.5), our group has recently published a second revision (*Mount Sinai Journal of Medicine*, 1988, **55**, 87–96). Because this change may be of interest to your readers, the newest version is offered here (Table I).

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Delusional AIDS and Depression

SIR: In high-risk subjects (drug addicts, homosexuals, anancastic or paranoid personalities) depressive states have been observed in which patients held the delusional belief of having AIDS (Miller *et al*, 1985; Fleming, 1986). We report a case of a patient showing a delusional idea of death from AIDS.

Case Report: R.M. is a 32-year-old heterosexual male with no previous personal or family history of psychiatric disorder. At the age of 30 he developed the fear of being affected by AIDS because he occasionally experienced cephalgia, vomiting, and diarrhoea; repeated routine blood tests were always negative. But the patient remained unconvinced, and at the age of 32 he applied for admission to an infectious diseases unit. HIV antibody testing gave negative results. Nevertheless, the patient remained deluded, convinced of his infection, and decided to await death: he therefore stopped work and took to his bed. This behaviour was

Table I. Clinical Dementia Rating

Impairment	*None* 0	*Questionable* 0.5	*Mild* 1	*Moderate* 2	*Severe* 3
Memory	No memory loss or slight inconstant forgetfulness	Consistent slight forgetfulness; partial recollection of events; 'benign' forgetfulness	Moderate memory loss; more marked for recent events; defect interferes with everyday activities	Severe memory loss; only highly learned material retained; new material rapidly lost	Severe memory loss; only fragments remain
Orientation	Fully oriented	Fully oriented except for slight difficulty with time relationships	Moderate difficulty with time relationships; oriented for place at examination; may have geographical disorientation elsewhere	Severe difficulty with time relationships; usually disoriented in time, often to place	Oriented to person only
Judgement and problem solving	Solves everyday problems well; judgment good in relation to past performance	Slight impairment in solving problems, similarities, differences	Moderate difficulty in handling problems similarities, differences; social judgment usually maintained	Severely impaired in handling problems, similarities, differences; social judgment usually impaired	Unable to make judgments or solve problems
Community affairs	Independent function at usual level in job, shopping, business and financial affairs, volunteer and social groups	Slight impairment in these activities	Unable to function independently at these activities though may still be engaged in some; appears normal to casual inspection	No pretence of independent function outside home Appears well enough to be taken to functions outside a family home	Appears too ill to be taken to functions outside a family home
Home and hobbies	Life at home, hobbies, intellectual interests well maintained	Life at home, hobbies, intellectual interests slightly impaired	Mild but definite impairment of function at home; more difficult chores abandoned; more complicated hobbies and interest abandoned	Only simple chores preserved; very restricted interests, poorly sustained	No significant function in home
Personal care	Fully capable of self-care		Needs prompting	Requires assistance in dressing, hygiene, keeping of personal effects	Requires much help with personal care; frequent incontinence

Score only as decline from previous usual level due to cognitive loss, not impairment due to other factors.

maintained for about two months. Then, suddenly, the patient showed a pantoclastic episode: he destroyed furniture in his flat, fouled the walls with faecal matter, and developed persecutory ideas focused on his relatives.

On admission he appeared autistic, passive (fairly catatonic), and completely lacking in psychopathological insight. He believed himself to be suffering from AIDS and to be very close to death. Pharmacological treatment was started with haloperidol (6 mg i.m.) and clonazepam (3 mg i.m.), but after 4 months, and despite increase in neuroleptic dose, the clinical picture did not show any appreciable change. Neuroleptic therapy was then discontinued, and treatment with imipramine (100 mg daily, p.o.) was started. After a few days the patient became to doubt the irreversibility of his condition, his catatonic attitude improved, and he started to be more active, loquacious, and interested in personal relationships. Subsequently he became critical of his previous psychopathological situation and started to make plans for the future. Seven months after admission the patient had completely recovered and was discharged on maintenance therapy of 50 mg daily p.o. of imipramine. After twelve months the patient resumed work on the same regime.

This patient seems to represent an extreme expression of fear about AIDS and its peculiarity is in the lack of risk factors and the absence of previous psychopathological disorder. At the time of acute onset of symptoms the patient was diagnosed

as suffering from schizophreniform disorder (DSM–III), but antidepressant treatment dramatically improved the clinical picture (where haloperidol did not) supporting a diagnosis of major depressive disorder, in spite of its atypical symptomatology.

Patients with fear of AIDS should be carefully evaluated because of the relationship with depressive disorders as indicated in this case. Pharmacotherapy appears to be of paramount importance for diagnostic discrimination in psychiatry, as in other branches of medicine.

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References

FLEMING, B. (1986) Psychiatric disorders associated with fear of AIDS. *The Ulster Medical Journal*, **55**, 84–85.

MILLER, D., GREEN, J., FARMER, R. & CARROL, G. (1985) A 'Pseudo-AIDS' syndrome following from fear of AIDS. *British Journal of Psychiatry*, **146**, 550–551.

Mianserin and Mania

SIR: We would like to report the changes we observed in a woman suffering from Huntington's chorea after she was treated with mianserin.

Case Report: Mrs M. A., aged 53, has been suffering from Huntington's chorea for eight years. She has mild to moderate choreiform movements of the upper limbs, face, and neck and she has been known to suffer from moderate to severe depression with a history of three previous suicide attempts with overdoses. There has been no clinical or psychological evidence of cognitive impairment. She has been on a long-stay psychiatric ward for three years and her depression has been treated with tricyclic antidepressants in the past. At the time of starting mianserin she was on no other medication. Over the preceding two months her appetite had been poor, and she had lost approximately 4 kg in weight. She also had polydipsia, but no evidence of glycosuria. She was clearly suffering from depression and was making reference to hopelessness and suicide. Mianserin was given at a dosage of 20 mg b.d. Mianserin was chosen because of its appetite stimulant and antidepressant properties and for being free from anticholinergic side-effects.

Seven days later her appetite had improved and the polydipsia had disappeared. The choreiform movements were noted to have increased. After ten days treatment the movements had become severe and incapacitating, making her restless, overactive, and exhausted to the point that she was unable to stand up. She was also found to be having bouts of uncontrollable laughter for no understandable reason.

Mianserin was stopped, with a noticeable reduction in overactivity 12 hours later; the choreiform movements began to decrease 24 hours after mianserin was stopped. Two weeks following initiation of treatment, the motor activity and choreic movement have continued to improve although these have not returned to the pre-treatment state. The appetite has remained good and there has been no recurrence of polydipsia. She has remained free from depression and she has not further expressed suicidal thoughts. It is of interest to note that her behaviour improved and that her choreiform movements decreased with the discontinuation of mianserin and without further need to treat her with sedatives or neuroleptic medication. We would point out that the patient had never manifested excitable behaviour before during her in-patient treatment over the last three years and there had never been any such reactions to tricyclic antidepressants or other medications.

Coppen *et al* (1977) reported 6 out of 13 patients with bipolar affective illness, treated with mianserin, to have become manic. In this study the patients had previously been treated with lithium carbonate before the changeover to mianserin. The paper does not state clearly whether the patients were suffering from any affective illness at the time when they were started on mianserin. Three of the 13 patients had left the trial at their own request because of drowsiness, and only four patients completed the full three months. It is not stated in the paper how long the manic symptoms persisted and whether mania needed further treatment. Mianserin was given at a dose of 20 mg t.i.d. in this trial, and the trial appears to be designed to investigate the possible prophylactic properties of mianserin in bipolar affective disorder. Our experience with Mrs M.A. is very different to the patients described by Coppen *et al* (1977). It illustrates the possible stimulating properties of mianserin in a patient who had never manifested manic illness in the past and who appeared to have been depressed because of the physical and mental disabilities of Huntington's chorea. It is of interest to note that her depressive symptoms were replaced by overactivity, restlessness, and laughter, while there was a considerable increase of choreic movements. Discontinuation of mianserin was followed by reduction of motor activity and choreic movement. We wonder if mianserin can be particularly effective in treatment of depression associated with organic brain disease, provided that its haemopoietic side-effect is adequately monitored?

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