that with patients whose mother-tongue was not English, translated versions of standardized English questionnaires could be given by computer and remain accurate and valid.

The use of microcomputers is of special interest in psychiatry, as a large part of diagnosis depends on explicit answers to questions. There is a growing interest in structured interview techniques (for example, the Present State Examination of Wing *et al*). In the United States many workers have shown that even disturbed psychiatric patients are amenable to computer interview (for example, Williams, 1975).

At the present time, we are engaged in a number of studies looking at applications of the computer in different psychiatric populations:

The assessment and treatment of phobic disorders;
The assessment of severity of depression;
The assessment of suicidal ideation;
Designing a program for general history taking;
The assessment of elderly patients;
The assessment of the deaf psychiatric patient;
The assessment of intelligence.

We would be interested to hear from other workers engaged in similar work.

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PSYCHIATRIC ILLNESS AND HYPERCALCAEMIA

Dear Sir,

Dr Weizman and his colleagues in their paper on Hypercalcaemia-Induced Psychopathology in Malignant Diseases (*Journal*, October 1979, **135**, 363–6) have missed what appears to me to be the most important conclusion from their data, namely, that the psychiatric disturbances in patients with hypercalcaemia are in most cases organic brain syndromes (confusional states).

In the first place I note that they describe Case 1 in their table as "anxiety, depression" but report that he had mild symptoms and that in the admission examination he showed memory disturbance. Although there sometimes may be an apparent disturbance of memory in *severe* affective disorders this is not the case here and the diagnosis is clearly an organic brain syndrome. Secondly, although it seems that the descriptions of Cases 2 and 3 may have been interchanged in the table in error, even so *each of* these patients had an organic brain syndrome, and the paranoid symptoms in Case 3 are to be regarded as part of this and of no diagnostic importance per se. Thus at least 5 of their patients with psychiatric disturbance suffered from organic brain syndromes. (Might one not, perhaps, be excused for suspecting that Cases 4 and 6 described in the table as "depression" and "severe depression" respectively might also have been suffering from organic syndromes?).

Finally I note that they state that "patient no. 1 was suspected to suffer from a psychogenic anxiety depressive syndrome reactive to malignancy until hypercalcaemia was found". The diagnosis of an anxiety state on the one hand or a confusional state on the other does not depend upon the level of calcium but upon a correct evaluation of the clinical features. I find it difficult to see how anyone could ascribe "cognitive dysfunction . . . to emotional reactions to the basic disease". From the information given it seems to me that if the clinical features had been correctly evaluated then "potentially harmful treatment with . . . drugs" could have been prevented long before the diagnosis of hypercalcaemia.

A sound clinical basis is essential for proper research in psychiatry.

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UP-TO-DATE RECORDS OF LONG-STAY PATIENTS

Dear Sir,

Assessment of long-stay psychiatric patients is often made difficult because the past records are inadequate. It may be impossible for staff having no previous knowledge of the patients to extract an adequate account of the clinical course and treatment from unorganized handwritten notes.

I would like to suggest a cumulative type of record making; in essence, a brief typewritten statement of

essential information, to be updated at each reassessment by the multidisciplinary team, and incorporating the contributions of all disciplines.

New members of staff, or those dealing with emergencies, would need to read only a single typed sheet to acquire a grasp of the case.

Forms with headings could be used for recording information, e.g.:

> Name, ward—date of birth—borough of origin—dates of and reasons for admissions—treatments and special precautions, e.g. drug sensitivity—general progress—other medical/surgical conditions. Present mental state (date of examination)—nursing dependency—occupational ability—links with family—social activities—diagnostic formulation. Current treatment/rehabilitation programme and aims.

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CLINICAL ASPECTS OF THE INTERACTION OF LITHIUM AND STIMULANTS

DEAR SIR,

Recently it has been shown that lithium can prevent some aspects of the response to amphetamine (Van Kammen and Murphy, 1975) and methylphenidate (Huey *et al*, 1977). However, others have questioned the uniformity and degree of the lithium block of stimulant effects (Angrist and Gershon, 1979; Wald *et al*, 1978). We present here two cases where lithium did *not* prevent stimulant effects in humans and it is suggested that clinicians should remain open about the nature of lithium interaction or lack of it with other psychoactive drugs in clinical situations.

Case 1. A 47-year-old married man with a 27-year history of bipolar manic-depressive illness was stabilized on lithium therapy with a marked elimination of manic attacks. Mild chronic outpatient depressions persisted and were not responsive to addition of amitriptyline up to 150 mg or nialamid to 125 mg for 4 weeks each. After cessation of previous antidepressants but while remaining on lithium with plasma levels of 0.6–0.7 mEq/L, methylphenidate 10 mg each morning led to rapid remission of depressive symptoms of low mood, lack of energy, loss of sex interest and psychomotor retardation. The methylphenidate was continued for three months and then discontinued with no return of symptoms. Lithium did *not* block the antidepressant effect (Bassik and Schoonover, 1977) of methylphenidate.

Case 2. A 57-year-old divorced man with a 24-year history of bipolar manic-depressive illness was a lithium responder. He suffered from a bucco-facial-lingual masticatory syndrome diagnosed as tardive dyskinesia due to neuroleptic usage before institution of lithium prophylaxis. As part of a research protocol using the concept of receptor sensitivity modification treatment of tardive dyskinesia (Friedhoff, 1977), the patient received 125 mg L dopa and 12.5 mg carbidopa daily for 3 days. On the fourth day the dose was raised to 250 mg L dopa plus 25 mg carbidopa. On the following day the patient developed typical manic syndrome with pressure of speech, aggressiveness, hyperactivity, and absence of need to sleep. The lithium blood level was 1.04–1.1 mEq/L. The effect of L-dopa to induce mania in bipolar patients has been previously noted (Murphy *et al*, 1971) but the phenomenon's occurrence in the presence of therapeutic lithium levels is important. This same patient had previously developed a transient (8 hours) manic attack in a research design employing 30 mg of intravenous methylphenidate, despite therapeutic lithium levels then (Wald *et al*, 1978).

In summary, lithium coverage does not necessarily prevent either positive or negative consequences of arousal-inducing drugs.

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