Journal, 1978, **1**, 560–61), I think Dr Dann is mistaken in thinking that the stresses of university life, or indeed of any busy working life, including that of a doctor in general practice, are necessarily precipitants of breakdown. Of course, the student's psychiatric illness must be adequately treated to start with, and regular psychiatric follow-up provided during the university term. Likewise, the psychiatrist should make sure the student is keen to continue study, and in conjunction with the university tutor should discuss the suitability of particular courses, which are not always wisely chosen.

In these circumstances students can and do acquit themselves very successfully, and it would be very unfair to bar them from taking their degrees, as well as wasteful to society to throw away their education in mid-stream and fail to use their trained abilities. Continued medication is no barrier to success, in fact it may be a pre-requisite. Dr Dann's experience as a student health centre medical officer is quite different from mine as a psychiatrist and I think it would be a terrible act of blind prejudice against our patients if his advice were followed to exclude all who have suffered a psychotic breakdown from a university career.

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CEREBRAL DYSFUNCTION IN SCHIZOPHRENIA

DEAR SIR,

Abrams, Redfield and Taylor (*Journal*, September, 1981, **139**, 190–94) are to be congratulated upon their careful study. In strict logic we must agree with their conclusion that their results were "consistent with a variety of studies demonstrating significant cerebral dysfunction in carefully diagnosed schizophrenic patients". However, another way of putting it would be that the WAIS is not very good at distinguishing demented patients from schizophrenic patients. On the WAIS, patients with organic brain disease come out as more similar to schizophrenics than to affectives.

This is not evidence that there is cerebral dysfunction in schizophrenia. It is evidence that the WAIS is not a specific diagnostic tool.

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SERUM DOPAMINE-BETA-HYDROXYLASE IN MANIC-DEPRESSIVE PSYCHOSIS

DEAR SIR,

Recent studies of manic-depressive psychosis have focussed on the function of certain fluids in the human body whose significance seems to be problematical. For example, a low level of urinary excretion of methoxy-4-hydroxyphenylethylene-glycol was reported by Maas (1974); cerebrospinal fluid 5-hydroxyindoleacetic acid levels were found to be lower in depression by Banki (1977); but studies of serum dopamine-beta-hydroxylase (S–DBH) have attracted less publicity since Shopsin's work (1972). Shopsin argued that there might be a relationship between the activity of S–DBH in the blood on the one hand, and the dynamics of mental disorder on the other; but neither he nor the researchers coming in his wake were able to perceive one. Shopsin's work was based on the correlation of levels of activity of S–DBH in a statistically significant number of blood samples. He did not perceive the possible variation of S–DBH over a period of time, misled, perhaps, by the negative results of Weinshilbaum's study (1971) of a normal subject's S–DBH variation.

This is an interim notification of results obtained in Toyoake, Japan in 1980 in a series of experiments designed to test the seemingly unpromising hypothesis that the S–DBH activity of manic-depressive psychosis measured over a period of time—as opposed to that of normal subjects—actually does change, and change, we suggest, in an arresting and consistent way.

Our strategy focussed on the alternating manic and depressive phases of psychosis. We followed 4 cases of circular manic-depressive psychosis over their clinical course (Nomura *et al*, 1981). The results indicated that S–DBH was high in the manic phase, decreases with clinical improvement, and becomes low in the depressive phase. Especially, S–DBH in two of our cases was in the manic phase almost twice that of the depressive phase. Each subject received lithium carbonate and/or antidepressants which are known to influence S–DBH. However, the fluctuation patterns of S–DBH did not always parallel the doses of medication. Furthermore, the hypothesis that the physical activity of the patients influenced the results can be discounted, because all blood samples were collected immediately after the patients woke early in the morning. It is therefore possible to speculate that the changes of S–DBH observed were due to physiological variations related to the clinical states the patients were in. On this evidence, we suggest that the imputed sympathetic activity level is high in the manic phase and low in the depressive phase. It remains to be seen, however, whether these changes reflect the activity of catecholamine in the brain, as proponents of the so-called catecholamine hypothesis of affective disorder might wish.

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ANTIPARKINSONIAN MEDICATION IN DEPRESSED SCHIZOPHRENICS ON DEPOT NEUROLEPTICS

DEAR SIR,

Recent work has confirmed the need for antiparkinsonian medication in neuroleptic treated schizophrenia (Manos *et al*, 1981) and suggested that 68 per cent of patients given placebo developed extrapyramidal side-effects. In another report Johnson (1981) indicated a tendency for depressive-type symptoms to improve when these patients were given orphenadrine 50 mg twice daily. However, he considered that some of the symptoms may have been neuroleptic drug-induced akinesia rather than depressive in origin.

This report relates to the outcome of a single blind cross-over study comparing sustained release benzhexol (Artane Sustets) with procyclidine hydrochloride in schizophrenia treated with depot neuroleptics. The results from 33 patients are available. They consist of 18 men and 15 women with a mean age of 47 years who were receiving depot neuroleptics for chronic schizophrenia. Nineteen patients had been prescribed fluphenazine decanoate and 14 fluphenthixol decanoate. Before entry into the study the patients were not receiving antiparkinsonian drugs. On recruitment the patients were either prescribed sustained release benzhexol 15 mg as a single morning dose or procyclidine hydrochloride 5 mg 3 times daily. On completion of 7 days' treatment the patients prescribed benzhexol were given procyclidine for 7 days and those commenced on procyclidine were given benzhexol. Extrapyramidal signs and symptoms were recorded on day 0, 7 and 14 on a 4 point scale as being absent, mild, moderate or severe. The data analysis showed that the 2 groups of patients were comparable at entry to the study and that there was no significant difference between benzhexol and procyclidine in the management of rigidity, tremor or akathisia. Both drugs were found to be equally effective.

Of 9 patients in the benzhexol-treated group reporting depressive symptoms initially, 7 were found to be improved after one week as compared with only 4 of 9 patients on procyclidine. This result, though interesting, did not reach statistical significance, but the trend observed here accords with that noted by Johnson in patients treated with orphenadrine. Could it be that there is a case for offering schizophrenic patients on neuroleptics an antiparkinsonian rather than antidepressant drug when they present with depressive type symptoms?

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POSITIVE AND NEGATIVE SCHIZOPHRENIC SYMPTOMS

DEAR SIR,

Crow in his discussion papers (1980, 1981), and Wing (1978), have not been explicit about their use of the concept of positive and negative symptoms. This concept has its origins in Hughlings Jackson's theory of evolution and dissolution of the nervous system (Jackson, 1894; Levin, 1936). Crow and Wing designate delusions, hallucinations and motility disorders as positive symptoms, believing that they predominate in acute attacks, whether at onset or later. Volitional defect, withdrawal, and flattening of affect are described as negative symptoms and they predominate in the quiescent phases of the chronic stage of the illness. The two categories of symptoms are presumed to reflect ". . . different underlying pathological processes" (Crow, 1980), within the group of schizophrenias. Supporting evidence is afforded by different responses to drug therapy and by other physical measurements.

Positive symptoms in the Jacksonian sense are the result of damage to healthy mental life. This damage leads first to negative symptoms. They are to be found during acute attacks, in the loss of selective attention, the loss of the capacity to discriminate the bodily and