

it for me to reject the traditional classification without sufficient evidence. My use of depressives who had not experienced a manic phase *may* not be defensible according to some evidence, but equally it may be entirely defensible, and Dyson and Mendelson's paper I take as strengthening my position. My understanding of imipramine is that in spite of its pharmacological similarity to chlorpromazine it is *not* noted for its tranquillizing effects. Even if Dr. Silverman finds its anti-manic properties unremarkable, those writers quoted in my paper certainly do betray a note of surprise at its effectiveness.

6. It is absurd to object that because one paper reporting the use of the digit-symbol test does not reflect depressive retardation neither will a measure of reaction time. Several different factors have been isolated among psychomotor measures (Seashore, 1951). The digit-symbol test involves a continuous response, while simple RT involves 'speed of initiating a single response'. The differential properties of psychomotor tests in relation to clinical status have been reported elsewhere (Court and Cameron, 1963). That reaction time provides a suitable measure for judging severity of illness may be concluded from my own work (Court, 1964), and it would be difficult to be more positive than King (1968) in a recent review—'a synthesis of the empirical findings reported by many different investigators, working with quite varied hypotheses, populations and procedures, makes clear the following themes: psychomotor retardation is found at all three levels of function (sub-factors) tested among the psychoses; most noticeably among the schizophrenias, but present in depression and mania as well. The degree of slowing follows closely clinical estimates made of the severity of disorder. . . .'

7. I respect Dr. Silverman's observations relating to the appearance of depression following the administration of haloperidol. While the point may prove an embarrassment to the model, I would prefer to await scientific documentation of this before rejecting the view that spontaneous fluctuations of the illness account for his observations. Hitherto it has appeared that the neuroleptics do not induce depression.

8. Finally, Sir, since my proposal is by no means conclusively established, may I allude to one final piece of evidence which has come to my notice since preparing the paper for publication. It follows that, with the model proposed, any improvements in the treatment of manic-depressive depression should lead to a corresponding decline in the incidence of mania. Older clinicians speak of such a decline, and this has been documented by Lachman and Abrams

(1963) who show a dramatic decline in admissions of manic patients to Bellevue Hospital during the years 1948–62. They did not find any single causal factor to explain this, but it could be argued that a high percentage of patients who would previously have developed manic episodes were effectively treated while depressed and never developed the more extreme reaction. In other words, the decline of mania arises from the improved treatment of recurrent depression.

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CHROMOSOME ABNORMALITIES IN PSYCHIATRIC PATIENTS

DEAR SIR,

Anders *et al.* (*Journal*, September 1968, p. 1167) found a significantly higher frequency of hypermodal cells and acentric chromosome fragments in psychiatric patients, compared with a control group. The authors mention that these abnormalities might be causally related to the mental illness of these patients.

An increased frequency of hypermodal cells as well as chromosome fragments and breaks is, however, likely to be due to treatment with psychotropic drugs and not causally related to the mental illness. We found a significantly higher frequency of hypermodal cells, breaks and chromosome fragments in psychiatric patients treated with psychotropic drugs compared with a control group of patients not treated with such drugs.

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DEAR SIR,

In reply to the letter by Nielsen *et al.*, we agree with their suggestion that some of our observations might be related to the psychotropic drugs being taken by these patients.

However, these patients were not taking LSD and in any case the evidence regarding LSD and chromosome breakage is controversial, and with regard to the other psychotropic drugs not-proven.

We believe that our explanation of an ageing effect is the more likely one.

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BIRTH ORDER AND DEPRESSION

DEAR SIR,

In his analysis of data relating birth order to depression. Professor Grosz (*Journal*, December 1968, p. 1555) notes that there seems to be a deficit of youngest-born among sibships of size three. But this deficit is

based on the expectation of equal numbers in each birth rank. There are two reasons why, on the null hypothesis, one would expect fewer representatives in the higher birth ranks from sibships of a given size:

1. The population in this country has been increasing for many years. Hence, if at a given time f_1 is the number of first-born of a given age, drawn from sibships of size n , and if f_2 is the number of second-born of the same age and also drawn from sibships of size n , then $f_1 > f_2 > \dots > f_n$.

2. Grosz explicitly states that he excluded sibships in which deaths had occurred. But the sibs of a youngest-born are more likely than those of an oldest-born of the same age to have suffered death—more years have been at risk. So in excluding sibships containing deaths, Grosz would be expected to have discarded disproportionately many youngest-born depressives.

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DEAR SIR,

Dr. James's argument is inappropriate and irrelevant to my study, as significant deficits of youngest-born among sibships of size three were found only among depressive patients. They were not found among the control samples of patients suffering from schizophrenia and anxiety.

Now, granted that it is possible, even though improbable, that the two considerations mentioned by Dr. James account for the findings only among the depressives and that for some reason they do not apply to the other two groups, they would still leave unexplained why among the depressives themselves the youngest-born were significantly under-represented only among patients with two differently-sexed sibs but not among patients with two same-sexed sibs.

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THE NEUROSYPHILITIC PSYCHOSIS TODAY

DEAR SIR,

Dr. Dewhurst's article (*Journal*, January 1969, p. 31) re-emphasizes that the improved treatment of