subjects. Close collaboration between biochemists and clinicians might clarify this issue.

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DEPRESSIVE ILLNESSES IN LATE LIFE

DEAR SIR,

In his recent paper Dr. Post (*Journal*, October 1972, pp. 393–404) considered the distribution of the scores of 92 depressed patients on the Newcastle diagnostic index (Carney *et al.*, 1965). He found that this distribution did not depart significantly from normality and concluded that there was, therefore, no evidence from this data that a dichotomy exists between unipolar and bipolar affective psychoses on the one hand and neurotic depressions on the other.

However, Dr. Post's distribution, and his value of χ^2 obtained ($\chi^2 = 11 \cdot 3$, d.f. $= 8$, $P = \cdot 2$) suggests that the corresponding population distribution may not be normal, and that additional data might well have produced a significant departure from normality. Accordingly I asked Dr. Kendell to let me have the distribution of 130 depressed patients on the Newcastle index, referred to by him in his 1968 paper (Kendell, 1968). Dr Kendell has kindly given me this distribution, which is reproduced below together with Dr. Post's, with the sum of the two and with the expected normal frequencies.

Although Dr. Post's patients were all over sixty, and Dr. Kendell's patients were younger, the means of the two distributions are very similar (3·86 and 3·79 respectively), so are their variances (10·69 and 10·39), and so are their distributions ($\chi^2 = 4 \cdot 98$, d.f. $= 10$, $P = \cdot 89$). It is therefore reasonable to add them together, and any departure from unimodality found in the combined distribution cannot be due to differences between Drs. Kendell and Post, or their data. The two sets of data are also similar in that there is a distinct dip at the score of 5 in both distributions. The distribution of the summed frequencies in the Table definitely departs from normality ($\chi^2 = 24 \cdot 5$, d.f. $= 11$, $P = \cdot 011$). (Dr. Peter Britton has kindly checked this result for me). Thus the hypothesis that the population distribution of depressed patients is normal can be rejected. But this does not necessarily mean that the population is non-unimodal; in general the distribution might be skewed, or flat, rather than bimodal.

The present data, however, appear to be non-unimodal. In particular, frequency of only 16 at the score of 5 is considerably less than either of the two adjacent frequencies (26 and 27). If the population distribution is unimodal, the frequency at score 5 should be at least a third of the sum of the three frequencies at scores 4, 5 and 6, since the scores of 4 and 6 have the highest frequencies and are therefore between the points of inflexion, if they exist. The sum of the sample frequencies at the three scores is 69. Thus the expected frequency at score 5 (given the unimodal hypothesis) is 23 or more. But it is only 16, and the exact probability of obtaining this frequency, or less, given an expected frequency of 23, is only ·045 —a significant result. Thus the hypothesis that the population frequency at score 5 is a third or more can be rejected. This frequency must, therefore, be less than a third of the sum of the frequencies at scores 4, 5 and 6. So it may be concluded that the population distribution is not unimodal.

The distribution of depressed patients is, therefore, neither normal nor unimodal. This is an important finding, because the data upon which it is based are certainly not biased in such a way as to generate a spurious departure from unimodality; it is clear that neither Dr. Post nor Dr. Kendell favour a bimodal view of depression. Dr. Kendell (1968, p. 21) concluded that the bimodal distribution obtained by Carney *et al.* (1965) might well be due to 'the strength of their original convictions and the pernicious influence of the halo effect, rather than any characteristics inherent in the patients'. This suggestion of Kendell's can now perhaps be rejected.

There is one further point which needs to be considered. Dr. Post (1972, p. 402) states 'it should in any case seem obvious that an analysis of data

Score	−3	−2	−1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	Total
Post's data	1	1	6	11	6	9	10	8	6	13	9	6	2	3	0	0	1	92
Kendell's data	1	5	9	10	11	8	14	18	10	14	14	10	1	3	1	1	0	130
Sum	2	6	15	21	17	17	24	26	16	27	23	16	3	6	1	1	1	222
Expected	5·8	5·6	9·1	13·3	18·7	23·3	26·6	27·1	25·5	22·0	16·4	12·0	7·8	4·4	2·4	1·1	0·9	222·0

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classified as either "black" or "white" (with an occasional allowance made for "grey"), even though referring to complex phenomena (like "personality", "precipitating factors" or "course independent of events"), would lead to equally simple and clear-cut results, e.g. the existence of a dichotomy'.

This is only true when the items of the scale are perfectly or very highly correlated amongst themselves. If the items are independent, or only slightly correlated, then, in view of the central limit theorem, the distribution of the summed scores will tend to normality, as Maxwell (1971) has pointed out (see also Guilford, 1956, p. 452). The average intercorrelation of the items of the Newcastle diagnostic index is only about ·18. Thus, given an homogeneous population, the distribution of this index will tend to be normal, not bimodal. This is also true of the Depressive Category-Type Scale (average intercorrelation = ·14) on which Garside *et al.* (1971) and Sandifer *et al.* (1966) found bimodal distributions. If this were not so, then such scales as those of the EPI and MPI would tend to have bimodal distributions, whereas in fact they have unimodal distributions.

Thus the finding that Kendell's and Post's data, when added together, are inconsistent with both the normal and unimodal hypotheses clearly indicates that there are at least two distinct populations of depressed patients. These populations, of course, may overlap to some extent, but they are nevertheless distinct in the sense that the majority of patients can be classified as belonging to particular groups.

Finally, as Dalén, a lucid exponent of Popper's ideas, has recently (1972) pointed out, a theory or hypothesis can be *dis*proved, but 'nothing can prove a theory is *true*: collecting facts which are favourable to a theory does not lead to any conclusive result'. The unimodal hypothesis of depression is a satisfactory hypothesis in that it is capable of being disproved. But it cannot be proved, as Drs. Kendell and Post have tried to do. Indeed, when their separate data are increased by adding them together, the resulting distribution is inconsistent with the unimodal hypothesis, as were the data of Carney *et al.* (1965), Sandifer *et al.* (1966), Fahy *et al.* (1969), Gurney (1971) and Garside *et al.* (1971). Thus six sets of data, collected by three independent groups at different places, are all inconsistent with the unimodal hypothesis of depression. Is it not now disproved?

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SUSTAINED RELEASE AMITRIPTYLINE (LENTIZOL) IN DEPRESSIVE ILLNESS

Dear Sir,

As Medical Director to the Company responsible for the production of sustained-release amitriptyline (Lentizol), and having been associated with Dr. Haider in the study reported in the *Journal* (May 1972, **120**, 521–2), I feel that there are several points which require comment in the letter from Dr. Arthur Rifkin *et al.* in the October 1972 issue of the *Journal*, **121**, 457. I am sure that Dr. Haider himself will wish to reply personally to this letter, but as he is now resident in Pakistan and there may be some delay before his reply is received I should like to make the following comments:

1. At the present time, to my knowledge, there are no published clinical trials demonstrating that ordinary amitriptyline given in a single daily dose is efficacious. It seems that the authors of this letter feel that the new sustained form of amitriptyline, which is a recognized advance in the formulation of the drug, should be matched against ordinary amitriptyline given in an as yet unproved dosage

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