conditioning techniques are of the disease-attacking sort, and should be as specific as possible, while all relationship therapies, from psycho-analysis onwards, are non-specific.

5. *Prognosis.* It is within the proper exercise of the doctor's authority to decide how far the sufferer's environment (as contrasted with his disorder) affects the prognosis. Environmental stress, whether social or other, affects prognosis whenever specific treatments for disease are less than 100 per cent successful. In psychiatry this means almost always.

These comments should suffice to show that the kind of depersonalized model described by these authors cannot be an exclusive source of the doctor's authority. Medicine may be, as they say (p. 955) a 'dirty, rough business', but it is still, at least on this side of the Atlantic, concerned with real human beings as well as models.

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DEAR SIR,

In reference to our paper, 'Laing's Models of Madness', we quite agree with Dr. Mathers that our description of the medical model in our original paper is 'limited'. In another of our papers on this topic 'Models of Alcoholism' (1) we attempted to deal with the problem of limitation. We said:

> 'The models are abstractions, or "ideal types". The reality from which they are abstracted is extremely complex, and in order to make models which can be compared the complexity must be reduced to manageable proportions. In doing so, we are aware that we have necessarily distorted the reality which is experienced by the proponents of the various points of view. We trust that the exercise of constructing and contrasting models will prove sufficiently useful to compensate for the inevitable distortions occasioned by this method.

A model is only a point of view or theory arranged in such a way that it can be compared with some other point of view or theory. We are in the process of collecting all the many and varied points of view about schizophrenia which we can find. We hope to encourage others to do the same. We would be particularly pleased if someone whose model we have described would say to us: 'You have got my model quite wrong. In the dimension of aetiology, it really ought to read . . .' We feel it would then be possible to have much more focused discussions of actual differences in opinion than we have had so far.

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CLASSIFICATION OF DEPRESSIVE ILLNESS

DEAR SIR,

I should like to report the findings from an attempt to replicate Kendell's discriminant analysis of the features of depressive illness (1968), using data from item-sheets completed on patients admitted to the Professorial Psychiatric Unit, the University of Melbourne.

This unit provides training facilities over a six-month period for postgraduates in the third year of their appointment to the State Mental Health Service. As part of their duties these postgraduates, of equivalent status to registrars in the British system, had to complete an item-sheet whose design was largely influenced by the Maudsley 'tem-sheet. The appearance of Kendell's monograph provided an opportunity for a test of the value of this method of collecting data and a fortuitous chance to replicate the basic study, as all the sixty items selected by Kendell were included in this item-sheet, and were recorded by trainee psychiatrists as in the Maudsley study.

Kendell's choice of discriminant analysis was determined by his preference for a linear canonical variate capable of handling data dichotomized as coming from patients with either psychotic or neurotic depression. The procedure in summary was to calculate the percentage frequency (p) with which each of the sixty items occurred in the two diagnostic categories; to calculate the standard error of the difference between the two percentages for each item, and to use the critical ratio (CR) with its positive or negative sign as the diagnostic weighting. The formula for the critical ratio (from which, incidentally, the square root has been omitted in the monograph) is

$$CR = \frac{p^1 - p^2}{\sqrt{\frac{p^1 (100 - p^1)}{N_1} + \frac{p^2 (100 - p^2)}{N_2}}}$$

where p^1 is the percentage frequency for the item in the psychotic depression group and p^2 for the neurotic depressive group. The number of cases in each group is designated N_1 and N_2.

A Fortran IV computer programme was written to solve this formula, and the raw data for the sixty items were punched on IBM cards for all the admissions to the unit in 1967. The programme was keyed to select and discriminate the two categories of depression and yielded $N_1 = 20$ and $N_2 = 53$. These numbers are small in comparison to Kendell's groups of 391 psychotic and 250 neurotic depressives, but the smaller N is taken into account in the formula and was expected to produce smaller values of the critical ratios. As in Kendell's study, the programme went on to recalculate the score for each patient using the obtained critical ratios as weighings. From these it produced means and standard deviations for each group, and wrote out frequency distributions and estimates of the degree of misclassification.

Results

As predicted, the smaller N produced critical ratios smaller than those reported by Kendell. The Maudsley figures ranged from 7·29 to −5·51, while in this study the range was from 2·45 to −3·1. As a rough check of the degree of agreement between the two sets of critical ratios independent of magnitude, the product moment correlation coefficient was found to be +0·5 (significant at the 0·001 level). Kendell lists 22 items as keyed in the negative direction, and the present study found 23. (The negative direction gives the weighting towards neurotic depression.)

This initial agreement with Kendell's findings was maintained when the diagnostic indices were applied to the two groups. The weighted score mean for the psychotic group was 9·48 (S.D. 2·95), Kendell's 9·5). For the neurotic depression group the mean was 1·23 (S.D. 3·92), Kendell's −1. The weighted score means for the two diagnostic groups were significantly different ($p<0·001$). Again, as Kendell found, no significant sex difference occurred within either group.

Using levels of 1 S.D. below the psychotic and 1 S.D. above the neurotic mean, the diagnostic indices showed a misclassification rate of 15 per cent for psychotic depression and 23 per cent for neurotic depressions. Kendell's overall misclassification rate was 27 per cent (19 per cent for psychotics and 40 per cent for neurotic depressives).

The preliminary analysis confirmed Kendell's method of deriving weighted scores from discriminant analysis and using these to load the item to produce a diagnostic index.

The question of bimodality (argued out by your reviewer K. Hope) was then examined. This is the crux of the argument, as it is on this that Kendell claims that 'the analysis fails . . . to provide any evidence of a qualitative difference between the psychotic and neurotic forms of depression'. The observed distribution of the diagnostic index scores for the total group of patients was compared with the expected proportions of the normal distribution curve and found to be significantly different ($\chi^2 = 24·78$; $p = 0·01$; 11 d.f.). From this the only conclusion is that the distribution is not normal.

The simplest check was to examine the extent to which the separate distributions for the two diagnostic categories showed a good fit with the expected proportions of the normal curve. For the psychotic group $\chi^2 = 4·19$; $p<0·5$; 5 d.f. and for the neurotic group $\chi^2 = 11·12$; $p = 0·2$; 9 d.f. Neither distribution is significantly different from normal, and it could be argued that the absence of normality in the distribution of scores for the two groups combined arises because it is composed to two approximately normally distributed groups with no appreciable overlap. In other words the findings support the bimodality of the distribution.

Following Kendell again, the item weightings calculated from the 1967 data were used to discriminate the diagnostic categories in the 1968 admissions. In this year 33 patients were diagnozed as suffering from psychotic depression and 52 from neurotic depression. The degree of misclassification was found to be 54 per cent overall (62 per cent for the psychotic group and 48 per cent for the neurotic group), an unsatisfactory level of discrimination.

There is unfortunately no way in which an appeal to the data can resolve the discrepancy between these two studies. The most likely source of error is an underlying one—the data themselves. Kendell went to some length to calibrate and to correct for unreliability introduced by the registrar observers, and it may well be that our smaller number of registrars (twelve in any one year) did not permit random errors to be cancelled out. On the face of it, it is unlikely that this source of error can ever be overcome to a degree sufficient to permit it to be used to resolve the question of discrimination of clinical categories.

Granted the smaller numbers, this study initially showed sufficient agreement with Kendell to make it a replication, but subsequently failed to support his finding of a lack of bimodality. This may confirm Hope's argument for a roughly even chance occurrence of bimodality.

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ACUTE PSYCHOTIC EPISODES IN PATIENTS TREATED WITH FLUPHENAZINE ENANTHATE

DEAR SIR,

Fluphenazine enanthate (F.E.) is a new type of neuroleptic drug obtained by combining a fatty acid with fluphenazine (a phenothiazine of the piperazine group) prepared in sesame oil. When it is administered intramuscularly or subcutaneously the therapeutic agent (fluphenazine) is gradually released over a period of two weeks, and because of this it can be prescribed in a dosage of 1 ml. every two weeks (Kinross-Wright *et al.* 1963). Clinical data reported by several investigators who have used it to treat schizophrenic patients show that its pharmacological and therapeutic effects are comparable to fluphenazine hydrochloride (Kurland *et al.* 1964) and that it is particularly useful for the treatment of acute schizophrenic reactions (Kline and Simpson, 1964).

My own clinical experience confirms the favourable opinions of it for the treatment of chronic schizophrenic patients, but at the same time casts some doubt on its usefulness in preventing the occurrence of acute psychotic episodes.

The following illustrative cases belong to a group of 25 chronic schizophrenic patients suffering from delusions and/or hallucinations and treated on an ambulatory basis with F.E.:

(1) A 25-year-old Negro male with a history of schizophrenia, paranoid type, of long duration, was started on F.E. on 13 December, 1967. At the time of his first clinical evaluation he was in good contact, well related, friendly, co-operative and normally talkative. However, abstract thinking impairment and delusional ideas were easily elicitable, and he admitted experiencing frequent auditory hallucinations. He received 1 cc. of F.E. weekly for the first three weeks and 1 cc. every two weeks thereafter. At the end of February 1968 he was greatly improved, his symptoms had completely disappeared, and he returned to work. He continued to attend the psychiatric clinic regularly, and on 22 April he received, as scheduled, 1 ml. of F.E. Four days later he was brought back to the clinic by his brother because he had become argumentative, extremely delusional and acutely hallucinating, and was admitted to hospital.

(2) A 35-year-old Puerto Rican seaman with a history of schizophrenic reaction, paranoid type, of at least one year's duration. While at sea, he began to suffer from auditory hallucinations of such intensity and frequency that he was discharged from duty. For about a year he was treated with various phenothiazines, but at the time of his referral to our clinic his auditory hallucinations were still continuous and troublesome. He was started on F.E. on 10 April, 1968, 1 ml. weekly for the first three weeks and 1 ml. every two weeks thereafter. On 6 June he received his sixth dose; at that time the auditory hallucinations had completely disappeared and his mental condition was considered much improved. But a week later he was brought in to the emergency room by a relative because of a sudden recurrence of severe, threatening auditory hallucinations. He was admitted to a psychiatric ward.

(3) A 29-year-old Puerto Rican woman with a history of schizophrenic reaction, paranoid type, characterized by impaired abstract thinking, poor judgment, lack of insight, ideas of reference and auditory hallucinations. At the time of referral she was symptom-free, as she had responded favourably to other phenothiazine therapy, but as she was felt to require maintenance pharmacotherapy, and as she often neglected to take the prescribed oral medications, she was started on F.E. treatment. She received her first dose of 1 ml. on 1 May, 1968, and 1 ml. every two weeks thereafter. One week after receiving her fourth dose she again developed ideas of reference, auditory hallucinations and verbal aggressiveness towards her husband. One ml. of F.E. was given immediately, and this was repeated on 25 June and 2 July, but there was practically no improvement and she was therefore put on to a different drug regimen.

These three clinical cases show that F.E. may not prevent the occurrence of acute psycnotic episodes, and that in such circumstances its therapeutic effect may be of limited value.

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STEREOTACTIC TREATMENT OF PARKINSONISM

DEAR SIR,

The clinical findings and the results of psychological testing of patients submitted to stereotactic treatment of parkinsonism reported by D. Asso *et al.*