know whether patients who 'fell by the wayside' during their long wait were deterred from seeking psychiatric help on later occasions.

I feel we should be circumspect before we too eagerly accept long waiting-lists as blessings in disguise.

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THE CLASSIFICATION OF DEPRESSIONS

DEAR SIR,

Professor Kendell's review (1) of the current state of the controversy surrounding the classification of depressions was extremely welcome. I should like to add one point concerning the dimensional/categorical part of the argument and suggest that the pattern of aetiology should not be assumed identical to that derived from studies concerned largely with clinical presentation. If we consider the clinical picture in depression, while a dimensional hypothesis is plausible for some of the features, notably mood disturbance, it is much less so for others, such as nihilistic delusions or auditory hallucinations, which seem to have no equivalents in normal states, or for the rapid changes in mental state that occur with acute onset or with swings from depression to hypomania. These 'discontinuous' features tend to occur in those whom Kendell designates Type A depressives, and it is noteworthy that in cluster analyses, where a substantial proportion of variables are related to clinical features, such a group of patients tends to be reliably identified (e.g. 2, 3), whereas Type B patients either fail to 'cluster' or appear as more than one group. It has been suggested that Type B depressions may be dimensional and continuous with normal states, while Type A are categorical and represent a pathological form with a separate and presumably discrete causation. Yet relevant studies reveal little evidence of major differences in the aetiology of the different forms of depression. It is plausible to suppose that in all depressions, one or more constitutional factors are implicated, together with stressful environmental features, and that all these are continuously distributed. It will be noted that in those studies in which a relatively large proportion of variables concerned with aetiology were included—notably Kendall's own (4, 5)—the separation of Type A depression has been less easy to demonstrate.

Can we then suppose that similar and continuously distributed causal factors can lead to either a continuous or a discontinuous symptom pattern? This is theoretically possible, as can be seen if one considers the analogy of the electric switch; and there are many instances in biology where continuous forces normally have continuous effect but at extreme intensities demonstrate a new and discontinuous effect. This model has recently received more prominence with the provision of a mathematics to deal with it (6), and is rather unfortunately known as 'catastrophy theory', The evidence for such a model of depression is limited; but it seems worthwhile to bear in mind that classification based on patterns of symptoms may be of limited value in the description of aetiology.

REFERENCES

1. KENDELL, R. E. (1976) The classification of depressions: a review of contemporary confusion. *British Journal of Psychiatry*, **129**, 15–28.
2. PAYKEL, E. S. (1971) Classification of depressed patients: a cluster-analysis-derived grouping. *British Journal of Psychiatry*, **118**, 275–88.
3. EVERITT, B. S., GOURLAY, A. J. & KENDELL, R. E. (1971) An attempt at validation of traditional psychiatric syndromes by cluster analysis. *British Journal of Psychiatry*, **119**, 399–412.
4. KENDELL, R. E. (1968) *The Classification of Depressive Illnesses*. Maudsley Monograph No. 18. London: Oxford University Press.
5. —— & GOURLAY, J. (1970) The clinical distinction between psychotic and neurotic depression. *British Journal of Psychiatry*, **117**, 257–60.
6. THOM, R. (1975; French edition 1972) *Structural Stability and Morphogenesis*. Reading, Mass.: W. A. Benjamin.

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LONG-TERM EFFECTS OF BEHAVIOUR THERAPY

DEAR SIR,

The introduction of behavioural techniques has led to a wave of therapeutic enthusiasm, but there are few long-term follow-up studies, and it is for this reason that I would like to report on two patients whom I treated ten years ago, using the method of systematic desensitization.

1. A 30-year old married woman who had been treated for her frigidity and housebound features (Kraft, 1967) was delighted with the treatment outcome; she was very much more cheerful, no longer moody, and could cope adequately with main roads and crowded places. Further she was pleased to report that during these ten years there had been a great improvement in her relationship with her husband. However, she was only prepared to speak to me on the telephone and refused to come and see me, because she felt that this might lead to a recurrence of her

old symptoms. For similar reasons, she was reluctant to lose weight. She explained to me: 'I must know where you are. If you disappeared suddenly, I would go into a panic. Coming to see you would mean going back, the dependence on you. I would prefer not to. I have a horrible feeling if I came to see you I would do something silly, go to pieces, return to symptoms, like going back to see old boy friends. I prefer to forget about that.'

Quite clearly, the patient may have made a good symptomatic recovery but basic problems of panic and the need for extra marital relationships had not been resolved. It would indeed have been surprising if this had been achieved by systematic desensitization alone.

2. A 20-year-old single male Drinamyl addict (Kraft, 1968) was equally delighted with his treatment result, and was proud to report that he no longer had any need for addictive drugs, though he did admit to smoking hashish when he was on holiday abroad, which was frequent. Whereas he was quite incapable of doing any sort of work at the time when he began treatment, he had now built up a successful business. He told me that he had put on four stones in weight, which he attributed to excessive drinking when he was working at a brewery, but he had no insight into the mechanisms involved. He found the follow-up interview extremely complicated, and on leaving he said that he hoped that there would never be a need for a further visit.

These are just two examples from my series, but they illustrate the sort of treatment result one may expect from behaviour therapy when given alone. Both patients showed symptom substitution in the form of weight gain, and the transference was not resolved in either case. It is for this reason that I have come to the conclusion that behavioural techniques should be used as part of a much wider psychotherapeutic programme, in which the patient is offered psychoanalytically-oriented psychotherapy as well as behaviour therapy.

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REFERENCES

KRAFT, T. (1967) Treatment of the housebound housewife syndrome. *Psychotherapy and Psychosomatics*, 15, 446-53.
—— (1968) Successful treatment of a case of Drinamyl addiction. *British Journal of Psychiatry*, 114, 1363-4.

DEATH DUE TO ISONIAZID (INH) AND PHENYTOIN

DEAR SIR,

The dangerous interactions that can occur between anticonvulsant and antituberculous drugs, particularly iso-nicotinic acid hydrazide, have become widely recognized during the past ten years. INH interferes with the bio-transformation of hydantoinates in the liver, resulting in a rise in serum levels especially in patients who are slow acetylators of INH. The protean manifestations of 'phenytoin encephalopathy' can be difficult to recognize, particularly in chronic epileptics who may have existing neuro-psychiatric syndromes (Johnson, 1975).

We wish to report briefly on a fatal outcome from such a reaction:

A 47-year-old hospitalized subnormal epileptic had been treated with phenytoin and phenobarbitone for 20 years. At the age of 45 she developed tuberculous adenitis and was treated with I.N.H. and streptomycin. Over the next year, she developed a bizarre neuro-psychiatric state with choreiform movements, and died. At a coroner's court death due to 'phenytoin toxicity' was recorded (serum phenytoin 2 days post mortem 8 mg/100 ml). This was attributed to interaction between the anticonvulsant and antituberculous drugs.

Clinicians treating epileptics who are taking anticonvulsant drugs should be aware of this potentially dangerous interaction which can result if antituberculous drugs have to be simultaneously administered. Determination of the patient's acetylator status and regular monitoring of the serum phenytoin levels are essential if dangerous complications of interaction are to be avoided.

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REFERENCE

JOHNSON, J. (1975) Epanutin and Isoniazid Interaction, *British Medical Journal*, 152.

A COMPILATION OF PAPERS FOR THE USE OF POSTGRADUATE STUDENTS OF PSYCHIATRY

DEAR SIR,

Copies are still available of this compilation, which was prepared by the Clinical Tutors' Sub-Committee and was printed on their behalf by John Wyeth Laboratories; those wanting copies should write to John Wyeth Laboratories, Huntercombe Lane South, Taplow, Maidenhead, Berks.

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