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FAMILY AND SOCIAL FACTORS IN THE COURSE OF SCHIZOPHRENIA

DEAR SIR,

In a recent letter (*Journal*, January 1977, **130**, p 102) Dr R. D. Scott and his colleagues levelled a number of criticisms at our work on Expressed Emotion (EE) in the relatives of schizophrenic patients. Unfortunately their thinking on the issues they raise is muddled. In order to clear up any confusion their letter may have engendered we will deal briefly with the points raised.

(1) They quote a figure of 27 per cent first admissions in the sample reported in the 1972 paper. This figure actually refers to first episode of illness, by no means the same as first admission. In fact, the proportion of first admissions in this sample was 59 per cent. The corresponding proportion in the sample reported in the 1976 paper was 57 per cent.

(2) They assert that 'the pattern of outcome in schizophrenia is usually quite apparent by two years and often by one year after first breakdown', and use this to detract from the predictive value of EE. In the first place, their statement is incorrect, as it is actually easier to predict outcome at the time of first admission than on readmission (Brown *et al*, 1966, p 142). Secondly, we had taken pains to demonstrate that the association between High EE and relapse remained significant when other factors contributing to relapse were controlled for. To satisfy any doubts Dr Scott and his colleagues may still harbour about this issue we have now reanalysed the pooled data from our two studies after separating them into first admissions and readmissions. For first admissions the relapse rates are: high EE, 38 per cent; low EE, 13 per cent ($P < \cdot 01$), and for readmissions: high EE, 69 per cent; low EE, 17 per cent ($P < \cdot 01$). The degree of association between EE and relapse can be expressed by the coefficient gamma, which is ·62 for first admissions and ·83 for readmissions. Hence EE is highly predictive of relapse in schizophrenic patients regardless of whether it is measured at the first admission or on any subsequent admission.

(3) They claim that by studying first admissions they can avoid retrospective bias and determine how parental attitudes to the patient arise even before the first manifestations of illness. This is an old fallacy which runs through the literature on the parents of schizophrenics, and has been criticized on a number of counts (Hirsch and Leff, 1975).

(4) They have attempted to derive a measure of EE from a self-rating paper-and-pencil technique. This is not comparable with our measures of EE, which entail as much emphasis on tone of voice as on content of speech.

(5) They state that 'In patients with good outcomes, the test score thought to be associated with EE decreases dramatically during the follow-up period.' Brown and his colleagues pointed out in the 1972 paper that all the indices of EE are decreased when measured at follow-up, with criticism showing the greatest reduction. Evidently measures of EE vary considerably over time, being elevated at the time of a crisis and falling as it subsides. However, it is inferred that when measured at a crisis point EE represents some characteristic of the relative's behaviour towards the patient which does endure over time and which influences the patient's tendency towards relapse. This supposition has been greatly strengthened by a recent study (Tarrier *et al*, in press) of the psychophysiological responses of schizophrenic patients to their relatives measured *in their own homes*. These measures, in particular the frequency of spontaneous fluctuations of the GSR, distinguish between patients living in high EE and low EE homes. The physiological measures in the patients were made over two years after the measurement of the relatives' EE. This provides strong support for a relationship between EE and an enduring emotional atmosphere in the home.

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BEHAVIOUR THERAPY

DEAR SIR,

Readers of Dr Hafner's paper (*Journal*, 1976, 129, p378–83) might wonder whether the conclusions drawn come from the data presented. He states that 'About 18 per cent of patients were adversely affected by the treatment programme', . . . that 'barely two-thirds of the 39 patients benefited usefully from treatment, and the emergence of fresh symptoms was a significant problem', and 'it is inescapable that a proportion of patients who received a standard symptomatic treatment were worse one year later'.

Tables I and II in the paper show improvement in all three groups on 4 of the 5 measures given. Although we are told that at one-year follow-up Group 3 was worse on 3 out of 4 measures, excluding symptoms directly treated, these are not specified, and the tables supplied actually depict slight *improvement* which is probably within chance levels (MHQ 56·8 to 55·3, FSS 74·8 to 70·5, self-dissatisfaction 13·5 to 12·1); on the only measure which seemed to imply a deterioration this change was within chance limits (spouse-dissatisfaction 7·0 to 8·2, $P < 0·1$). On what criteria, therefore, were Group 3 worsened, apart from a non-significant change on a rating scale of spouse-dissatisfaction whose reliability and validity has not been presented, and despite improvement in 4 other measures? To proceed from a non-significant change on 1 out of 5 measures which is in the opposite direction to all other measures given, and then to draw conclusions about 'worsening' seems illogical.

This aside, the definition of 'symptom emergence' is questionable: 'an increase over pre-treatment scores on any scale of the MHQ of Fear Survey Schedule on more than one of the 5 post-treatment assessments.' Fluctuations in severity of agoraphobia without treatment have been so well documented that 'fresh' symptoms need to be clearly beyond the natural fluctuation. Nevertheless, patients who before treatment had rather poorer marriages exposed themselves significantly less to the phobic situation, and after treatment did less well than other patients.

I agree with Dr Hafner's point that we should seek evidence of worsening in wide areas of a patient's functions after direct treatment of a specific problem. In a study of group exposure (flooding) for agoraphobics this was done (Hand *et al*, 1974): marital and interpersonal relationships for a group of 25 patients as a whole *improved* significantly, and the gain continued at follow-up. Seven patients had to be offered help for marital difficulties; however, two-thirds of the 21 married patients regarded their marriages as unsatisfactory *before* treatment. This emphasizes that far from treatment being responsible for making marital discord 'emerge' or become 'substituted' for phobias, improvement in phobias and obsessions spills over as improvement in other areas. In a prospective follow-up study of phobic disorders 4 years after treatment, patients who improved most in their phobias showed least subsequent depression, and the group as a whole showed no worsening of general anxiety, obsessions, depersonalization, work, social, sexual, family, or other relationships (Marks, 1971).

Briefly, Dr Hafner's data illustrate the well-documented phenomenon that patients who improve after behavioural treatment for a main problem, tend to improve in other areas, thus negating traditional ideas about symptom substitution.

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DEAR SIR,

Reading Dr Hafner's article, we are deeply concerned at the methodology employed and the lack of understanding of the principles of behaviour therapy shown by the author. We will very briefly support our criticism.

Methodology

(a) There is a lack of information concerning the treatment involved. Who took the patients into the frightening situations; was the exposure graded (gradual approach) or was the flooding technique used?

(b) The patients are divided on the basis of results arising from two questionnaires which are then transformed into a score and used for statistical analysis. It is not clear whether the increases within