each item were used, or differences between the total. One cannot add scores based on different scales.

(c) The author claims that half of the patients in Group 3 did improve, while the other half got worse. If this was the case, it would have been essential to re-examine the original assessment of the six patients who deteriorated.

(d) It is not stated whether the tests were related or unrelated. It is hoped that the related test was used.

Theoretical assumptions

In the Introduction, the author contrasts behaviour therapy with psychotherapy and claims to investigate standard treatment for agoraphobia. It is a known fact that there is no standard treatment for any phobia, least of all in agoraphobia, where fear of going out is usually only one of the many complaints and not necessarily the most important one.

What Dr Hafner does is to disregard individual differences and to administer a package to a heterogeneous sample. It is within the author's right to administer his treatment programme, but to suggest that this is standard behaviour therapy practice, and to make far-reaching conclusions on the basis of his study, is not acceptable. Behaviour therapy as a technology which leads to psychiatric diagnosis followed by a package deal treatment has a very limited application. It is essential to tailor treatment to the individual on the basis of behaviouristic analysis, which is guided by learning principles derived from relatively well-established findings in experimental psychology. Such analysis will lead to different treatments for the same diagnostic categories and will lead to the possibility of treatment of any complaint whether 'symptomatic' or 'underlying'.

The only difference between psychotherapy and behaviour therapy is in our view, the way both assessment and treatment are structured. Behaviour therapy would soon come to an unhappy end if treatment for agoraphobics consisted of nothing else but taking people on walks.

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DEAR SIR,

I respond to the letters of Dr Stern and of Drs Meyer and Reich. In my paper I suggest (p 378) that the resolution of the question of 'symptom substitution' was '. . . hindered by dogmatic and exaggerated statements on both sides, as well as by a lack of reliable data'. The letters to which I am responding seem to demonstrate the truth of this. They appear to be used mainly as vehicles for putting forward the authors' own views on behaviour therapy, at the price of idiosyncratic interpretations of my paper.

For example, Dr Stern quotes me as saying 'barely two-thirds of the 39 patients benefited usefully from treatment . . .' without mentioning that the context of this half-sentence (p 382) clearly indicates that this is one possible viewpoint, and not my own.

Drs Meyer and Reich suggest that my therapy '. . . consisted of nothing else but taking people on walks', even though I stated (p 379) that '. . . each patient's post-treatment programme took into account progress made during treatment and allowed for any revision of initial treatment goals judged to be necessary'. They misconstrue my use of the term 'a standard intensive symptomatic treatment programme' as implying that the programme used is the treatment of choice for all agoraphobics, even though I clearly indicate my reservations about this type of treatment approach (p 382): 'If a standard group treatment programme makes a proportion of patients worse, can it be justified ethically . . .?'

Regarding their comment under (a), a detailed description of the treatment is given in the separate report mentioned in the text and listed in the references.

Space permits a response to only two specific criticisms of my data analysis. Regarding comment (b) of Drs Meyer and Reich, it seemed more accurate to use the total incidence of reported fresh symptom emergence as a basis for forming the three groups, rather than scores on just one of the symptom questionnaires. Once the three groups were formed, the questionnaires were analysed separately, as is shown clearly in Table I.

Regarding paragraph 2 of Dr Stern's letter, contrary to his statement that I have not specified the symptoms directly treated, these are described in some detail (p 381) as being reflected in the phobic anxiety scale of the MHQ and the A and B scales of the FSS.

Both letters appear to reflect an inability to acknowledge that behaviour therapy, whether based on exposure in real life or on so-called 'behaviouristic analysis', can make a proportion of people worse. It is naturally (as it was for me) painful for any therapist to acknowledge this, particularly if he wishes to emphasize the virtues of his own treatment approach in relation to that of others. But only by admitting that behaviour therapy may have adverse effects can the explanation for these be sought, and treatment modified.

In my study, those patients who deteriorated or failed to improve usefully were often married to

people who appeared to have a vested interest in their spouse's disability and who firmly resisted attempts to explore this aspect of the problem. Treating such patients alone seems inappropriate to me now, since a large part of the real problem lies outside them: namely, in the inability of their spouses to acknowledge their role in maintaining the patients' symptoms, and in the spouses' reluctance to undergo therapy in relation to this.

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LITHIUM THERAPY AND OPHTHALMIC GRAVES' DISEASE

DEAR SIR,

We were interested to read Dr Rosser's paper (*Journal*, January 1976, **128**, p 61). We have recently had a patient treated with lithium carbonate who developed ophthalmic Graves' disease (Rundle and Wilson, 1945). This condition is characterized by classical exophthalmos and an abnormal hypothalamico-pituitary axis with normal levels of tri-iodothyronine and thyroxine and absence of other signs of hyperthyroidism. In thyroid disorder exophthalmos indicates an auto-immune process and occurs in this condition in classical Graves' disease and occasionally in Hashimoto's disease.

Case history. The patient is 35. His mother and one brother had required thyroidectomy for thyrotoxicosis, and another brother had exophthalmos. His first admission was in 1956 and there have been fifteen further admissions; most of these were for over-activity, over-talkativeness, lack of sleep and grandiose ideas. A diagnosis of manic depressive psychosis was made.

He was first treated with lithium carbonate in March 1967, at which time there was no mention of any abnormality of thyroid status or of abnormality of the eyes. He was noted to have exophthalmos, with a rather prominent thyroid, at admission in May 1968. This admission became necessary 'a few days' after he had ceased to take lithium carbonate; treatment with this was immediately restarted. No further mention of his thyroid state occurs in the notes until 1973. During the intervening period he received lithium therapy except for periods of two months and of one month during 1968. In 1973, during a further admission following his discontinuing all medication including lithium, his protein-bound iodine level was recorded as 9·2 μg/100 ml and he was noted to have moderate exophthalmos. A physician's opinion at that time was that he was euthyroid and that his mildly raised protein-bound iodine was a reflection of over-activity, although this level in most laboratories would be taken to indicate hyperthyroidism. After this he was not treated with lithium until June 1975. In March 1976 his eye signs became more prominent; there was some enlargement of the thyroid, but no other signs of thyrotoxicosis. He was over-active, aggressive, talkative and grandiose at this time, but had been on lithium continuously for nine months. Levels of tri-iodothyronine and thyroxine were within the normal range. However, there was a reduced response of thyroid-stimulating hormone (TSH) to thyrotrophin-releasing hormone (TRH). Values of TSH before, 20 minutes after and 60 minutes after intravenous injection of TRH were recorded as $<0\cdot5$, 2·8 and 1·6 milliunits per litre. This was despite the known effect of lithium in enhancing the release of TSH after TRH injection and indicates an abnormal hypothalamico-pituitary axis: hence a diagnosis of ophthalmic Graves' disease was made (Hall *et al*, 1970).

Discussion. This case is of interest taken alongside those of Rosser. We infer that eye signs did not occur in her cases. Nevertheless, in this case there may have been an element of the rebound phenomenon she suggests in the episodes of 1968 and 1973 occurring after self-withdrawal from lithium therapy. This explanation would not account for the exacerbation in March 1976. The case certainly indicates that a 'forme fruste' of hyperthyroidism may occur despite lithium therapy. It may be that this was Graves' disease in a predisposed subject in which the known effects of lithium were successful in minimizing the output of tri-iodothyronine and thyroxine. It is also conceivable that lithium withdrawal may have stimulated an auto-immune process, and it would certainly be of interest to look at antithyroid antibodies in patients on lithium therapy. Added circumspection might be advisable in the lithium therapy of patients with a marked family history of thyroid disorder.

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