1983, but was then lost to follow-up. By March 1984 she had not left her flat for four months, felt "unable to face the outside world", was floridly thyrotoxic, and needed a large dose of diazepam (and much persuasion) to get her to hospital by taxi. Medical treatment and a graded behavioural regime led to a full recovery. By the end of May 1984 she was both euthyroid and "enjoying the great outdoors".

The second patient, a 31 year-old female bank clerk, was successfully treated with carbimazole for thyrotoxicosis in 1978. She remained well, without medication, until December 1983 when she developed feelings of shakiness and weakness, episodes of depersonalisation, and began to avoid going out because of "panic attacks". All investigations, including a TRH test, proved negative. A combination of propranolol, behavioural advice and support led to near-complete remission of agoraphobic symptoms over four months.

In contrast to Dr Weller's case, both these patients developed agoraphobia *after* being diagnosed as thyrotoxic, and it is quite understandable that such behaviour might develop in certain individuals. It may be that the negative results reported in my second patient and the initial presentation of Dr Weller's patient were simply false negatives, although his "appropriate serum samples" may not have included a T3 level or TRH test. It may be that our present tests of thyroid function are simply too insensitive to subtle hormonal changes.

Nevertheless, there certainly seems scope for a detailed investigation of the relationship between hyperthyroidism and agoraphobia. Given their often similar physical symptoms the wonder is that this has not been done!

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MITRAL VALVE PROLAPSE AND ANXIETY DISORDERS

Dear Sir,

The relationship between mitral valve prolapse (MVP) and anxiety disorders remains an enigma. The majority of the literature on this area has stemmed from the USA with reports in the late 1970's that the prevalence of MVP in certain anxiety disorders, namely panic attacks/disorder and agoraphobia was considerably higher than expected (Pariser *et al*, 1979; Kantor *et al*, 1980 and Gorman *et al*, 1981).

Several recent papers have cast doubt on this, finding a prevalence of MVP in anxiety disorders of zero or within the normal range (Shear *et al*, 1984 and Hickey *et al*, 1983). For example a study by Bass and Wade (Bass *et al*, 1984) on 99 cardiac patients presenting with chest pain reported that of the 31 patients who had normal coronary arteries, 12 had a psychiatric diagnosis of anxiety/phobic neurosis and none of these 31 patients had any echocardiographic evidence of MVP.

As part of a study of anxiety disorders we have investigated 19 psychiatric out-patients for the presence of MVP. Using the Research Diagnostic Criteria (Spitzer *et al*, 1978) the patients were made up of 10 with panic disorder (4 of whom fulfilled criteria for agoraphobia and 1 for social phobia), 5 with phobic disorder only (4 agoraphobia and 1 social phobia) and 4 with generalized anxiety disorder. None of these patients had any evidence of MVP by either clinical or 2D echocardiographic criteria (reference for criteria used, Wann *et al*, 1983).

This appears to be further evidence that there is no increased prevalence of MVP in the anxiety disorders. In fact, considered with the study of Bass and Wade (1984) it suggests that anxious patients may even have a lower incidence of MVP than the normal population.

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RELAXATION AND DEPERSONALISATION

DEAR SIR,

In a sample of forty anxious patients treated over the past few years with Jacobsen's progressive relaxation, there were seven who reported becoming distressed by the technique. A paradoxical outcome of this nature has been termed 'relaxation-induced anxiety' (Heide & Borkovec, 1983). Looking retrospectively at the clinical notes, it struck me that these seven could be singled out as reporting depersonalisation syndrome, prior to treatment. In a further retrospective investigation, the seven adverse responders were administered the 'Self Alienation Questionnaire' (Dixon, 1963) which purports to measure depersonalisation. As a group, they scored significantly higher self-ratings of 'Self-Alienation' than ten randomly selected control subjects who responded favourably to the relaxation procedure (adverse patients' mean = 32; controls' mean = 22; P = .05).

The questionnaires were administered post-treatment, which produces methodological problems in that treatment outcome may have flavoured response to the questionnaire items. Nonetheless, there is tentative evidence here that the presence of relaxation may even distress depersonalised patients, presumably by exacerbating feelings of unreality.

I wonder if any other reader has noticed adverse reactions to relaxation technique in depersonalised subjects? If so, the presence of depersonalisation may suggest that relaxation-orientated methods are contra-indicated.

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NORADRENALINE AND TARDIVE DYSKINESIA

DEAR SIR,

We read with interest the article by Jeste *et al* (*Journal*, February 1984, **144**, 177–80) in your Journal in which attention was drawn to findings supporting increased noradrenergic activity in tardive dyskinetic patients. The conclusions of the authors are in our opinion premature and even perhaps misleading. Most of the CSF samples for noradrenaline estimation were taken from schizophrenic in-patients, some of whom were receiving neuroleptic treatment and others who had been free of such treatment for at least 6 months.

Increased noradrenaline has been shown by other workers to occur in CSF samples of schizophrenic subjects (Hornykiewicz, 1982) as well as in certain cases in samples from some subcortical areas (Hornykiewicz, 1982).

Tardive dyskinesia is an abnormal movement disorder, reported in patients usually on long-term neuroleptic therapy. The dopamine theory implicating postsynaptic receptor hypersensitivity, has been the most widely accepted explanation of this condition. In view of the strong interrelationship between the dopamine and noradrenaline systems (for instance, damage to noradrenergic pathways in the prefrontal cortex prevents the development of denervation supersensitivity to D_1 dopamine receptors in the affected area (Hornykiewicz, 1982)), it is likely that chronic dopamine blockade leads to a compensatory noradrenergic hyperactivity.

The fact that beta-blockers have been shown to ameliorate tardive dyskinesia does not exclude the possibility that this condition involves other neurotransmitter systems, for instance Gaba-ergic drugs have also been useful in the therapy of this condition (Korsgaard, 1976).

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TREATMENT OF NEUROLEPTIC MALIGNANT SYNDROME

DEAR SIR,

In recent letters from Dr Jan Scott (*Journal*, January 1984, **143**, 98) and Dr P. D. White (*Journal*, April 1984, **144**, 437) in response to Dr Rosemarie V. Cope's letter (*Journal*, August 1983, **143**, 202–23) on neuroleptic malignant syndrome, various treatments are mentioned including dantrolene and bromocriptine.