familial, chronic progressive neurological disorder of middle and later life, of unknown aetiology, characterised by marked supranuclear opthalmoplegia affecting vertical eye movements initially, pseudobulbar palsy, dysarthria, dystonic rigidity of the neck and upper trunk, and dementia/frontal lobe syndrome (Price, 1978). Other cerebellar and pyramidal symptoms may occur (Lishman, 1978). PSP was first described by Steele, Richardson & J. Olszewski (1964) as a clinical syndrome separate from Parkinsonism, and is sometimes known as the Steele-Richardson-Olszewski syndrome. Diagnosis is mainly by clinical progression of the disease (Haldeman, Goldman, Hyde & Pribram, 1981). The largest reported series is by Jackson, Jankovic and Ford (1983) who found 16 cases of PSP in 415 patients with an initial diagnosis of Parkinsonism. We have been unable to find any case report in a British psychiatric journal in the last ten years.

A 48 year-old divorced woman with seven children was referred to a psychiatrist with a three year history of changing personality, depression, loss of weight, difficulty in speaking normally, increasing lack of competence in the home, occasional lapses of memory, a tendency to fall and difficulty in using her hands efficiently. She was admitted to a psychiatric ward for assessment. Mental state examination revealed no psychotic features, she was correctly orientated for time, place and person, and memory was apparently normal on testing. She had insight into the gradual changes in herself, and was distressed and depressed by them. She was referred to a neurologist, who detected severely impaired vertical eye movements, mask-like facies, axial rigidity, dystonia, dysarthria and hyper-reflexia. On the basis of these and her mental changes, the diagnosis of Steele-Richardson-Syndrome was made, supported by CT Scan (Jackson *et al* (1983), Haldeman *et al* (1981) which showed moderate cerebral atrophy.

During the next 9 months, the patient deteriorated rapidly. She became incontinent, disinhibited, often undressed in public, required to be bathed and dressed, fell frequently, and developed periodic inflammatory changes in her eyes. Nonetheless she remained fully orientated, and answered questions rationally. This combination of depression, disinhibition and need for nursing care, together with intact awareness of her surroundings and retained insight, created a management problem, as she was not appropriately placed in a general psychiatric ward, or psychogeriatric unit (at the age of 48). Special arrangements were made for her transfer to Part III Accommodation with Psychogeriatric Day Hospital attendance. Soon after her transfer she developed respiratory complications and was admitted to a general medical ward, where she died 11 days later. Death was attributed to pulmonary embolism. No post mortem was done.

The following features are of interest.

1. The case presented as a psychiatric disorder.
2. Although her behaviour suggested pre-senile dementia, cognitive functions and insight remained relatively intact, suggesting predominantly frontal lobe damage.
3. The age of onset was 45 (usual range 51–68 years, average 59.3 years) (Jackson *et al*, 1983).
4. The disease progressed rapidly to death 9 months after diagnosis (average duration 4.4 years (Lieberman *et al*, 1982) or 5.4 years (Jackson *et al*, 1983)).

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B. DAS

Lynfield Mount Hospital,
Bradford BD9 6DP

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AGORAPHOBIA AND HYPERTHYROIDISM

DEAR SIR,

Having recently seen two patients with agoraphobic and thyrotoxic symptoms, I was struck by Dr Weller's letter (*Journal*, May 1984, **144**, 553–4) reporting this association. It is surprising that there seems to be no literature on this specific relationship at all, even though thyroid function tests are now mandatory (excessively so) in the assessment of anxiety states.

My first patient, a 41 year-old housewife, was treated for thyrotoxicosis between 1978 and February

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!

T. H. Turner

St Bartholomew's Hospital,
West Smithfield,
London EC1A 7BE

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.

Michael P. Chan
George A. Hibbert
John Watkins

University Department of Psychiatry,
Warneford Hospital,
Oxford OX3 7JX

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