Depression in Parkinson's disease

We were surprised at the choice of controls used by Huber et al in their study which recently showed little relationship between depressive features and the progression of the disease. They recruited their 'controls' from carers of Parkinsonian patients, either spouses of the patients or members of a support group, which we have previously criticised with respect to studies which have had similar methodological problems.

We have studied a group of 100 Parkinsonian patients and their carers using the Hospital Anxiety and Depression Scale which avoids many of the somatic elements of the Hamilton or Beck inventories, and has been well validated for the physically ill. We found a close relationship not only between the levels of depression in patient and carer, but also for features of anxiety. We have used controls of similar disability who were unrelated to the patients, and shown no difference in depressive features between Parkinsonian and non-Parkinsonian patients. This leads us to believe that in contrast to the argument proposed in this paper, and traditional teaching, much of the 'depression' seen not only in Parkinson's disease but also in other chronically disabling diseases (such as stroke) is a non-specific feature of disability. Variations in anxiety is a much more specific and clinically relevant psychological feature of this intriguing disease.

We would hope that recognition of these psychological features will be reflected not only in therapy for the patient, but also in the consideration of their management.

Episodic paroxysmal hemicrania

The report by Blau and Engel highlights the episodic nature of paroxysmal hemicrania and suggests that patients can be divided into three groups: A) those who begin with the chronic phase; B) those who remain episodic and C) those who start episodic and then progress to chronicity.

The report prompts me to detail the history of a female patient presented at the age of 43 with a three year history of episodic left sided paroxysmal hemicrania. At worst, her attacks occurred up to 20 times per day with a maximum duration of 10–15 minutes. At best, they occurred twice per day. The initial cluster period was four to six months with four to six months of relief from the attacks. Investigation showed no abnormality and a diagnosis of paroxysmal hemicrania was made and she showed a dramatic response to indomethacin.

The initial episodic phase of the disease lasted four years and in the fourth year, following the diagnosis being established at the end of the third year, she had two periods of therapy with indomethacin of five months each.

She then went into a three year period where she required daily therapy because of the chronicity of her symptoms, the attacks occurring virtually daily. She then re-entered an episodic phase of the disease which has now lasted for two years.

The patient's history would suggest therefore that patients with episodic disease can have protracted chronic phases and then return to an episodic pattern and that this pattern should be recognised as part of the spectrum of paroxysmal hemicrania.