Matters arising

Respiration and sleep in Parkinson's disease

Sir: We read with interest the report by Apps *et al.*¹ We should like to describe our own recent experience reported elsewhere,² which has some similarities but also some important differences.

There is some evidence that patients with Parkinson's disease die more frequently during sleep than those with other neurological diseases,³ suggesting possible respiratory insufficiency. Flow-volume loop abnormalities have also been described in awake patients which are consistent with reversible upper airways obstruction.⁴ For these reasons we decided to document sleep physiology in 10 patients with idiopathic Parkinsonism and to compare them with 10 patients with post-encephalitic Parkinsonism, in whom sleep abnormalities have long been recognised,⁵ and 20 age- and sex-matched controls. Bedside screening tests of autonomic function were performed in all subjects and no abnormalities were present.

Like Apps *et al.*, we noted a significant reduction in the duration and proportion of REM sleep in our patients, which was most marked in those with post-encephalitic Parkinsonism (Group 3); less marked in the six patients with Hoehn & Yahr stage III-IV patients with idiopathic Parkinsonism receiving optimal levodopa therapy (Group 2); and least marked in four patients with mild (stage I-II), untreated idiopathic Parkinsonism (Group 1). However, all patients had more frequent episodes of obstructive and central sleep apnoea than controls, most frequent in Group 2 (see table). Apnoeic episodes were predominantly (>80%) obstructive/mixed in nature, but central apnoeic episodes were also recorded in all patient groups and were of similar frequency in groups 2 and 3. In addition, one patient in Group 1 had recurrent episodes of obstructive sleep apnoea in REM and non-REM sleep for more than two hours each night, accompanied by arterial oxygen desaturation of 5-25%, and therefore fulfilled the accepted criteria⁶ for the sleep apnoea syndrome. Interestingly this patient was not

obese, consistently had a normal flow-volume loop when awake, and had no other respiratory abnormalities. This is of particular significance since both Parkinson's disease and obstructive sleep apnoea syndrome are said to be associated with abnormalities of the flow-volume loop in many patients.

Whilst we acknowledge that there may be differences of methodology and of semantics in defining "significance" of apnoeic episodes⁷ between our own study and that of Apps *et al.*, our own data suggest that significant respiratory abnormalities during sleep may be just as frequent in idiopathic and post-encephalitic Parkinsonism of comparable severity. This raises important questions about possible more extensive neurological deficits, for example affecting the brainstem, in Parkinson's disease. Further studies are clearly warranted.

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Cisplatin neuropathy with Lhermitte's sign

Sir: Like Dewar *et al.*¹ and Thompson *et al.*² we have seen Lhermitte's phenomena in three patients with cancer in whom the only drug in common was cisplatin and who do not appear to have metastatic disease. Our patients had no obvious signs of a myelopathy and only mild signs of a sensory neuropathy. The Lhermitte's phenomenon improved even when the neuropathy did not. As suggested, it may be that the dorsal column demyelination seen in the one pathologically examined case³ is all secondary to damage in the dorsal root ganglia and an example of central distal axonopathy.¹ We write to report another symptom in this situation that is normally associated with a primary demyelination in the spinal cord: exacerbation of symptoms after exercise. This patient is aged 38 years and developed an undifferentiated teratoma in his anterior mediastinum. He was treated with surgery and chemotherapy that included cisplatin. At the end of the course he described Lhermitte's phenomenon that lasted for a few weeks, and also a more permanent tingling in his feet. He said that when he ran about a hundred yards his feet would become numb and he would become unsteady. Examination at rest showed absent reflexes, no definite sensory loss and his gait was normal. After exercise he made mistakes on joint position sense testing of his toes and became unsteady, and this was worse when he closed his eyes. The same phenomenon did not occur in a hot bath. His symptoms had not progressed over 12 months and so we do not believe he has metastases to his cervical cord and think that they are related to his cisplatin therapy.

Unlike Lhermitte's phenomenon which is rather non-specific for all sorts of causes of posterior column dysfunction, worsening of symptoms on exercise in this fashion is very unusual except in multiple sclerosis and it is of interest that it happens in this situation.

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Table Respiratory abnormalities in normals and patients during sleep

Subjects	Mean no of apnoeic* episodes	% of apnoeic episodes of obstructive/mixed nature	Mean no of desaturation† "dips"
Normals (n = 20)	10.8	9	0.7
Group 1: PD untreated (n = 4)	24	93	3
Group 2: PD treated (n = 6)	48.5	83.5	26
Group 3: PEP (n = 10)	29.7	81	9

*Significant apnoea is defined as a cessation of nasal airflow for greater than or equal to 10 s.⁶

†Significant arterial oxygen desaturation "dips" defined as a greater than or equal to 4% fall in oxygen saturation.⁶

PD = Parkinson's disease. PEP = post-encephalitic Parkinsonism.