selves may be sufficient to give rise to obsessive-compulsive behaviour.³ The behavioural symptoms appeared in our patient after an interval of days (obsessions) to weeks (compulsions, apathy) after the putaminal stroke and progressed over subsequent months. Likewise, patients have been described who presented obsessive-compulsive behaviour or "psychic akinesia" (apathy, lack of motivation) after a period varying from days to years after basal ganglia lesions, often with a progressive worsening over time in the initial phase.^{3,4,7} The behavioural changes in our patient occurred about 23 years after the onset of Parkinson's disease. Interestingly, although Parkinson's disease is one of the most common neurological disorders affecting the basal ganglia, reports concerning the occurrence of obsessive-compulsive behaviour in Parkinson's disease are surprisingly scanty.¹

Neuropsychological examination disclosed in this patient a widespread cognitive impairment, with a severely reduced capacity to control attentional resources.

In our patient, compulsive verbal iterations could contribute to her marked breakdown of attentional control, which in turn might partially account for her poor cognitive performance. In dual task paradigms when a secondary task (for example, counting from 1 to 5 repeatedly) is performed concurrently with the task of interest (primary task), performance on one or both tasks will deteriorate if the processing demands of the two tasks exceed the available attentional resources. In this patient, the incessant verbal iterations might act as a sort of articulatory suppression "secondary" task that, during various "primary" cognitive tasks, could interfere with the operation of a limited capacity processing system, such as the supervisory attentional system or the central executive component of the working memory model.

The putamen receives projections from the supplementary motor area, and, in turn, projects back to this area through the internal segment of the globus pallidus and the nucleus ventralis lateralis of the thalamus. Because the left supplementary motor area seems to be involved in motor planning of speech,⁸ the association in our patient between obsessive-compulsive behaviour mainly characterised by verbal iterations and a dysfunction in the basal ganglia-frontal loop system in the dominant hemisphere for language is of interest.

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Down's syndrome and Parkinson's disease

Almost all patients aged over 40 with Down's syndrome develop dementia consistent with Alzheimer's disease with senile plaques and neurofibrillary tangles. There is some overlap between Alzheimer's and Parkinson's disease, with severe dementia occurring in about 15% of patients with Parkinson's disease. Rigidity and, less commonly, other parkinsonian features occur in some patients with Alzheimer's disease. The relatively new concept of Lewy body dementia has created even greater problems in delineating the two entities. A pathological-anatomical link between the genetically determined chromosomal aberration Down's syndrome and Parkinson's disease would be an argument in favour of genetic influences in Parkinson's disease, and of shared pathogenetic links between Alzheimer's and Parkinson's diseases. The pathological hallmarks of Parkinson's disease are Lewy bodies, with severe neuronal loss in the ventrolateral tier of the pars compacta of the substantia nigra. Pathological studies have suggested that cortical neurofibrillary tangles and neuritic plaques are associated more often with Lewy bodies in the brain stem than could be explained on the basis of chance.¹ However, in an extensive search for Lewy bodies in 121 patients with Alzheimer's disease and 273 controls without Parkinson's disease, Gibb *et al* were not able to establish such an association.² There are some reports of parkinsonian signs in Down's syndrome,³ and of improvement after treatment with levodopa.^{4,5} Bodhireddy *et al*⁶ described a patient with Down's syndrome with diffuse Lewy body disease and Alzheimer's disease. Raghavan *et al*⁷ found two patients with nigral Lewy bodies but no cell loss in 23 patients with Down's syndrome. They argued that the presence of Lewy bodies in two out of 23 cases (8.7%) in this age group (with the expectation of incidental Lewy bodies being less than 1%) is probably not coincidental. However, in 14 cases of Down's syndrome, Gibb *et al* found no nigral Lewy bodies.⁸

We examined haematoxylin and eosin stained sections from the substantia nigra of 27 patients with Down's syndrome, aged 25 to 70, with a mean age of 47.8 years. Ten patients were aged between 50 and 59, five between 60 and 69, and one patient was 70 years old. The clinical diagnosis of Alzheimer's disease is difficult to ascertain in mentally retarded patients. It was based on above all the appearance of memory deficits but also, although usually later, increased orientation difficulties. Alzheimer's disease was suspected in 21 of our 22 patients over 30 years of age. No Lewy bodies were found, but in all cases of clinically suspected Alzheimer's disease nigral neurons contain-

ing tangles were easily identified. One patient, a woman, who died aged 56, was examined one year before death, at a neurological department. She was judged to be parkinsonian with rigidity and given medication (benserazid/levodopa). At postmortem no Lewy bodies were found in the nigra; there was nigral cell loss but not to the degree usually seen in Parkinson's disease.

The frequency of incidental Lewy bodies between the sixth and ninth decade has been reported by Gibb and Lees⁹: after compilation of five other studies, the percentage in the age group 50 to 59 was 1.8% and between 60 to 69 it was 10.7%. For the whole group, aged between 50 and 69 Lewy bodies were found in 32 out of 437 patients (7.3%).

As there were no Lewy bodies in the tissues examined, our study suggests that there is no link between Parkinson's disease and Down's syndrome. This is contrary to the opinion of Raghavan *et al*, but agrees with the findings of Gibb and Lees. A compilation of these two studies with our own, only including patients aged between 50 and 69, gives two patients out of 46 (4.3%) with Lewy bodies. This result, compared with 7.3% among controls, makes it highly unlikely that there is an increased frequency of Lewy bodies in patients with Down's syndrome.

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