naturally and was placed strategically; as near as can be determined from the CT scan, it was contiguous with the site of the nucleus ventralis intermedius. Clinical recovery from the effects of the infarct on its primary site, the internal capsule, was complete. It would seem, therefore, that the lesion produced in the thalamus, that is, at the edge of the infarct, was very small indeed, or was very subtle. Its effectiveness in curing the tremor suggests that the therapeutic part of surgical lesions lies in the most lateral part of the nucleus.

To our knowledge this is the only reported case of its kind. It lends further support to the effectiveness of surgically induced lesions in the management of severe ET.

RODERICK DUNCAN, IAN BONE, IAN D MELVILLE, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF

References

Bromocriptine therapy in striatonigral degeneration

Sir: Striatonigral degeneration is a neuro-pathological entity within the group of "multiple system atrophy" degenerative diseases. Neuropathologically it is characterised by severe atrophy of the putamen and moderate atrophy of the substantia nigra. Clinically the patient with striatonigral degeneration presents with an atypical Parkinsonian syndrome, in which muscular rigidity predominates over the other symptoms; the disease also progresses more rapidly than idiopathic Parkinson's disease.1–3

Most of the patients with striatonigral degeneration do not respond to levodopa therapy.4–5 The use of lisuride or pergoline, both dopamine receptor agonists, has not improved patients with multiple system atrophy.5–7

We treated a striatonigral degeneration patient with levodopa and bromocriptine. As far as we know, there are no previous reports on bromocriptine therapy for striatonigral degeneration. Our patient, a 69 year old Caucasian male, complained in 1981 of weakness of his right arm and right leg. There was a history of alcohol abuse; the family history did not include any neurological disorders. Neurological examination at that time revealed a right central facial paresis, slight weakness of the right arm and leg, symmetrical deep-tendon reflexes and no Babinski signs. He exhibited cogwheel rigidity of the right arm and a short-stepped gait. EEG and CT were normal. The diagnosis was minor stroke and a mild, atypical Parkinsonian syndrome. In addition to acetosal the patient received amantadine which did not relieve his Parkinsonian symptoms. In 1983 his illness had progressed, the patient was more rigid and moderately disabled as far as walking was concerned (Hoehn and Yahr scale 3). He was put on a therapeutic levodopa regimen in the outpatient clinic but developed severe hypotension, which made hospitalisation necessary in December 1983. Clinically the patient obviously improved on levodopa; unfortunately within about one month his condition deteriorated again (Hoehn and Yahr scale 4–5). Levodopa was discontinued and bromocriptine (Parlodel 15 mg three times daily) was prescribed. He improved markedly and was, upon discharge, in good condition (Hoehn and Yahr scale 1–2). Ten months later he experienced exacerbation of his disease with progressive hypokinesia and rigidity, without tremor. From that time onwards all medication failed and the patient deteriorated slowly. He died in October 1985 of an aspiration pneumonia, 4 years after the onset of his Parkinsonian symptoms.

Postmortem examination revealed bronchopneumonia. The external appearance of the brain was unremarkable. There was a severe atrophy of the putamen and depigmentation of the substantia nigra. Microscopic examination revealed a marked neuronal loss with dense fibrillary gliosis in the putamen; an abnormal brown pigment had been deposited in the glial cells. There was a moderate neuronal loss in the substantia nigra; no Lewy bodies were found. The pons, medulla oblongata and cerebellum, showed no significant changes. The neuropathological diagnosis was striatonigral degeneration.

The diagnosis of striatonigral degeneration must be considered when a patient presents with rigidity as the initial symptom, there is no tremor and the rate of deterioration is more rapid than one would expect in idiopathic Parkinson's disease. Most striatonigral degeneration patients do not respond to levodopa. In our case, levodopa had a beneficial effect which, however, lasted for only one month. The patient improved surprisingly soon after the administration of bromocriptine and remained in a good condition for a period of 10 months.

There is no explanation for the fact that levodopa fails to be effective in striatonigral degeneration. Probably as a result of degeneration of putamen and substantia nigra, the drug is not able to reach enough dopamine receptors to improve the Parkinsonian symptoms. Bromocriptine bypasses the degenerating presynaptic nigrostriatal neurones and affects directly on the post-synaptic receptors of the remaining neurones of the striatum in the striatonigral degeneration. If the diagnosis striatonigral degeneration is likely and there is no response to levodopa after a period of 3 months, we advise on the basis of our experience with one patient, medication with bromocriptine.

RB VAN LEEN, WWM PERKINS
Department of Neurology, Westeinde Hospital, Lijnbaan 32, Postbox 4, 2501 CK The Hague, The Netherlands

References