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 com-
plete. 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 ther-
apeutic 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.

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Bromocriptine therapy in striatonigral
degeneration

Sir: Striatonigral degeneration is a neu-
ropathological entity within the group of
"multiple system atrophy" degenerative dis-
ases. Neuropathologically it is character-
ised by severe atrophy of the putamen and
moderate atrophy of the substantia nigra.
Clinically the patient with striatonigral
degeneration presents with an atypical Par-
kinsonian syndrome, in which muscular
rigidity predominates over the other symp-
toms; the disease also progresses more rap-
 idly 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 pergolide,
both dopamine receptor agonists, has not
improved patients with multiple system
atrophy.6 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 stria-
onigral 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 neuro-
logic disorders. Neurological examination at
that time revealed a right central facial pares-
sis, slight weakness of the right arm and leg,
symmetrical deep-tendon reflexes and no
Babinski signs. He exhibited cogwheel rigi-
dity of the right arm and a short-stepped gait.
EEG and CT were normal. The diagnosis
was minor stroke and a mild, atypical Par-
kinsonian syndrome. In addition to acossal
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 dete-
riorated again (Hoehn and Yahr scale 4-5).
Levodopa was discontinued and bro-
ocriptine (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 Octo-
ber 1985 of an aspiration pneumonia, 4
years after the onset of his Parkinsonian
symptoms.

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

The diagnosis of striatonigral degener-
ation must be considered when a patient
presents with rigidity as the initial symp-
tom; there is no tremor and the rate of deter-
ioration is more rapid than one would expect
in idiopathic Parkinson's disease. Most stria-
onigral 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 admin-
istration of bromocriptine and remained in
very 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 degen-
eration 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 neu-
rons and acts directly on the post-synaptic
receptors of the remaining neurons of the
striatum in the striatonigral degeneration
patient. If the diagnosis striatonigral degen-
eration 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.

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