but as a will have co-exist, in is alem

Multiple sclerosis, tropical spastic para-
pareis and HTLV-I infection

A course without remissions(s) and re-
lapse(s) was the one clinical feature that
distinguished human T-cell lymphotrophic
virus type-I (HTLV-I) associated tropical
spastic paraparesis (TSP) invariably from
multiple sclerosis (MS) in the series of
patients reported by Rudge et al.\textsuperscript{2} We
do not consider this to be an absolute rule
since we have reported a patient with HTLV-I
associated TSP who did indeed manifest such a
pattern of illness.\textsuperscript{3}

We should perhaps emphasise\textsuperscript{4} that our
patient was an African, born and raised in
Swaziland where the very occurrence of MS
is quite uncertain. Also, corticosteroids were
not given, an intervention that might have
corresponded to the relapsing-remitting case
in a case of HTLV-I associated myelopathy
described recently by McKendall et al.\textsuperscript{5}

\textbf{BOOK REVIEWS}

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This is a compact volume in the series “Clinicians Guide to Nuclear Medicine” which is edited by the second author. The

Dr Poser and his colleagues have not
attempted any type of epidemiological study
to show a relationship between trauma and
MS. The evidence he cites for a positive
relationship is largely speculative and con-
ducnal. While we agree with him that a
"perfect" epidemiological study in MS is
difficult to design and execute, this fact
should not deter those who would design
reasonable studies to find out what actually
happens in this illness.

Breakdown of the blood-brain barrier pre-
cedes symptoms and other MRI signs of new
113:1477–89.

2 Schumacher GA, Beebe G, Kihler RF, et al.
Problems in experimental trials of therapy
in multiple sclerosis; report on the panel
on the evaluation of experimental trials of thera-
py in multiple sclerosis. Ann NY Acad Sci
1965;122:552–68.

Neuropathological features of Alz-
heimer’s disease in non-demented Par-
kinsonian patients

I was interested to read about the two cases
reported by Daniel and Lees.\textsuperscript{1} Both
of the patients had neuropathological features
of Alzheimer’s disease and nigral loss but clin-
ically had dopa-responsive Parkinsonism.
However, one of the interesting features of
their condition that may help distinguish such
patients from those with idiopathic Parkinson’s
disease, is the speed with which they
develop involuntary movements related
to therapy. In both of their cases the patients
developed abnormal movements within 12
and six months of starting oral levodopa.
This may relate to their combined pathology
of nigral loss with striatal plaques and neurofil-
ament tangles. It will therefore be of interest
to see if patients who develop early dyskinetic
movements with levodopa have similar neu-
ropathology.

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University College, Oxford

1 Daniel SE, Lees AJ. Neuropathological features
of Alzheimer’s disease in non-demented Park-
insonian patients. J Neurol Neurosurg Psy-