thiamazole 30 mg daily improved all the symptoms, including meralgia paraesthetica.

A year later, the left lateral cutaneous nerve of the thigh sensory conduction velocity had improved to 58·8 m/s with normal amplitude (3·6 μV).

To discover the frequency of localised sensory disturbances in hyperthyroidism, we studied clinically 20 patients with Graves' disease, including these two cases (10 hyperthyroid patients, 10 euthyroid, after treatment). Symptoms consistent with mononeuropathy such as localised sensory disturbance or muscle weakness, were present in nine (45%). These symptoms were dysaesthesia, paraesthesia or hypoaesthesia on fingers in five patients, hypoaesthesia or dysaesthesia on the lateral aspects of the thigh in seven patients, and bilateral foot drop in one patient (case 1). Tinel's sign of median nerve was present in seven of 10 hyperthyroid patients and five of 10 euthyroid patients. A positive Tinel's sign was found in 60% of thyrotoxic patients but also in 14·5% of 282 normal controls (Chi-square $=23{\cdot}6$, $p < 0{\cdot}00001$).

Our two cases demonstrated a combination of mononeuropathy and thyrotoxicosis. These mononeuropathies were confirmed by nerve conduction studies and improved following treatment for thyrotoxicosis. In addition, the denervation findings and low amplitude evoked reponses without conduction block of the peroneal nerve in case 1 suggest mono-axonopathy associated with thyrotoxicosis. The dissociation between complete foot drop and no conduction block of the peroneal nerve supports this possibility.

In euthyroid patients, the frequency of both the symptoms and Tinel's sign diminished with the duration of treatment.

In a detailed electrophysiological study[3] of patients with thyrotoxicosis, loss of functioning motor units with normal conduction velocities demonstrated motor neuron dysfunction and the remarkable capacity of motor neurons to resume normal function. Individual nerves are more sensitive to mechanical damage if a generalised peripheral neuropathy is present.[4] It seems likely to us that the fragility of nerve axons associated with hyperthyroidism predisposes to mononeuropathies.

S IJICHI
K NIINA
M TARA
F NAKAMURA
Division of Internal Medicine, Kagoshima Municipal Hospital, Kagoshima
N IJICHI
S IZUMO
M OSAME
The Third Department of Internal Medicine, Faculty of Medicine, Kagoshima University, Kagoshima, Japan

Correspondence to: The Third Department of Internal Medicine, Faculty of Medicine, Kagoshima University, 1208–1 Usuki-cho Kagoshima 890, Japan

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Correction:
This letter was printed in the August issue with only one MRI image.

MRI of thoracic cord in tropical spastic paraparesis

Tropical spastic paraparesis (TSP) is a disease occurring in Afro-Caribbeans following HTLV-1 retro-virus infection. There is some evidence that the geographical and ethnic distribution of HTLV-1 illness is even wider[1] and HTLV-1 associated myelopathy (HAM) in Japan is probably the same disorder. Abnormalities are found on MRI of the brain in both TSP[2,3] and HAM.[4] High signal areas are found in the brain similar to those in multiple sclerosis (MS), though they tend to be less extensive. The thoracic cord (on which the brunt of the pathological process falls) has been examined in only three patients, one of whom had atrophy.[2] Since the clinical picture of TSP may resemble that of progressive MS, we have made a systematic comparison of the MRI characteristics of the thoracic cord in the two conditions.

Nine patients with TSP who were born in the Caribbean were compared with an age and sex matched group of European white patients with clinically definite MS,[5] all of whom had a progressive spastic paraparesis. Disability was scored using the Kurtzke Disability Status Scale.[6] The patients with TSP were anti-HTLV1 positive and had HTLV-1 genome integrated into leucocyte DNA. Eight were female. The mean age was 53 years (range 43–65 years), the mean symptom duration was 12 years (range 1·5–23 years), and the mean Kurtzke disability score was seven (range five to eight). The mean age of the MS patients was 42 years (range 35 to 53 years), the mean symptom duration was 11 years (range seven to 17 years), and the mean Kurtzke disability score was five (range 4 to 6). The spine was imaged by a Picker 0·5T superconducting machine with T1 weighted ($SE_{500\,40}$) 5 mm contiguous parasagittal slices using a surface coil. All MS patients and five TSP patients had additional T2-weighted sequences ($SE_{1500\,80}$, 5 mm contiguous parasagittal slices) to detect abnormal signal. Images were reported without knowledge of the individual diagnosis by one of the authors (EPGH du B).

Atrophy of the thoracic cord was seen in six of nine patients with TSP and five of nine patients with MS. Three of five patients with TSP who had T2-weighted images of thoracic cord had diffuse high signal and all three had atrophy (fig). Five of nine with MS had high signal return on T2 weighted images, one of whom did not have atrophy. The pattern of high signal was diffuse in two and focal or patchy in three (fig).

These results confirm the previous MRI finding of atrophy in the thoracic cord in a proportion of patients with TSP. However, a similar degree of atrophy is seen as frequently in patients with MS who had a progressive spastic paraparesis, a finding compatible with pathological studies where cord atrophy is present in 72% of patients with MS at necropsy.[7] There was some difference in the pattern of high signal seen in the two groups, with more diffuse and uniform high signal in TSP and focal or patchy high signal in MS. However, these differences in the MRI findings are slight and a reliable distinction between the two conditions cannot be made on these grounds.

AG KERMODE
P RUDGE
AJ THOMPSON
EPGH DU BOULAY
WI MCDONALD
The Multiple Sclerosis NMR Research Group, Institute of Neurology, Queen Square, London

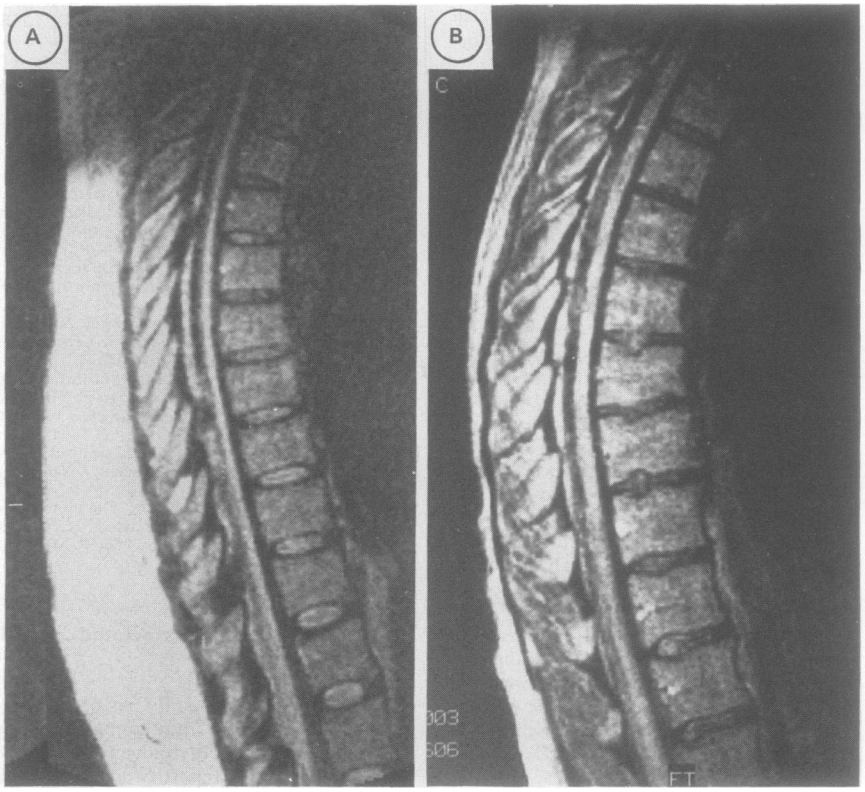

Figure The MRI on the left shows diffuse high signal with atrophy of the thoracic cord in TSP. The right shows the patchy high signal typically seen in MS. ($SE_{1500/80}$, 5 mm sagittal slices).