Matters arising

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 anecdotal. 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.


Multiple sclerosis, tropical spastic paraparesis and HTLV-I infection

An article without misprints(s) and a relapse(s) was the one clinical feature that distinguished human T-cell lymphotropic virus type-I (HTLV-I) associated tropical spastic paraparesis (TSP) invariably from multiple sclerosis (MS) in the series of cases reported by Rudge et al.1 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.2 We should perhaps emphasise 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 contributed to the relapsing-remitting course of a case of HTLV-I associated myelopathy described recently by McKendall et al.3


Rudge et al reply:

It is true that there are patients with HTLV-I positive tropical spastic paraparesis, who do not show a progressive course, but they are exceptional. In fact, the major clinical problem is differentiating patients with multiple sclerosis from those with TSP when the course is progressive. Such a situation arises in HTLV-I endemic areas where the two conditions co-exist, for example Brazil. Obviously a small proportion of the population will have HTLV-I antibodies in their serum, but as a rule in TSP the titres are higher, often dramatically so, than in the asymptomatic carriers, or patients with unrelated illnesses.

In the black South African patient described by Gledhill et al. the titres of HTLV-I antibodies in the serum and CSF were extremely high and the diagnostic category into which he fell viz: TSP or MS, is heavily dependent upon the weight one gives to an episode of unilateral visual failure that was irreversible and the improvement observed. Whatever the diagnosis, this case is exceptional.


Neuropathological features of Alzheimer's disease in non-demented Parkinsonian patients

I was interested to read about the two cases reported by Daniel and Lees.1 Both of the patients had neuropathological features of Alzheimer's disease and nigral loss but clinically had dopa responsive Parkinsonism. However, one of the interesting features of their condition that may help distinguish such patients from those with idiopathic Parkinsonian 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 levadopa. This may relate to their combined pathology of nigral loss with striatal plaques and neurofibrillary tangles. It will therefore be of interest to see if patients who develop early dyskinetic movements with levadopa have similar neuropathology.

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Focus on Parkinson's Disease.

This account of the papers presented at a postgraduate conference concerning Parkinson's disease and extrapyramidal syndromes held at Tremizzo, Italy in April of 1990 comprehensively covers the pathophysiology of akinesia and hyperkinnesia, the aetiology of Parkinson's disease, the multiple system atrophies and current problems and potential developments in therapy. All but 17 of the 56 contributors are from Italy and thus to a considerable degree the book represents an authoritative Italian statement concerning this important group of movement disorders. It is a most impressive account and the balanced content reflects not only the excellence of the contributors but also the editorial skills of Professors Caraceni and Nappi.

The text is gratifyingly free of the irritating defects often associated with precipitate publication of proceedings and manages to be both comprehensive and succinct. Particularly impressive is an account of the renaissance of interest in the subthalamic nucleus in the organization of basal ganglia function by Rondot, the biochemical aspects of the Lewy body disease by Gerlach and Reiderer, and Duvoisin's critique of genetic and epidemiological factors. Other impressive surveys cover the neuropsychology of the parkinsonian syndromes, primary autonomic failure, single photon emission computer tomography and the management of late complications of Parkinson's disease. Perhaps because of the risk of the appearance of second thoughts, the chapter on adenial medial implants is not the strongest or most critical but does illustrate the breadth of topics covered. The book is presented in Italy and not in the series so elegantly produced and the print is agreeable to read.

Gerald Stern