Figure Cerebellar atrophy, mainly of the folia with 4th ventricle dilatation.

who had a broad based gait since she was 45 with pronounced bilateral dysmetria on finger-nose test. On CT (fig) we found marked cerebellar atrophy, mainly of the folia, with fourth ventricle enlargement. All her six sons and daughters who had myotonia, diagnosed on clinical and electromyographic grounds, had no cerebellar signs and two of them had normal CT scans. Heredoataxia and other causes of cerebellar atrophy, such as drugs, alcohol and neoplastic disease were excluded.² The exact meaning of these findings is unknown.

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Aphemia as a first symptom of multiple sclerosis

Sir: I must take exception to the diagnosis of multiple sclerosis given the patient described

by Herderschee *et al* in the *J Neurol Neurosurg Psychiatry*.¹

The history and findings do not fulfill the Poser *et al* criteria for laboratory supported definite multiple sclerosis for the important reason that dissemination in time, as well as dissemination in space, is not present. All the parameters cited in support of the diagnosis of multiple sclerosis, that is the CSF findings, the CT and MR images and, of course, the resolution of symptoms are all compatible with acute disseminated encephalomyelitis, a diagnosis which on the basis of the information provided seems considerably more likely.

This is, unfortunately, a common and distressing problem in differential diagnosis, compounded by the still not widely known fact that all the ancillary supporting data, viz the elevated CSF IgG, the presence of oligoclonal bands in the CSF, periventricular areas of attenuation with or without contrast enhancement, and areas of increased signal intensity by MR, can be seen equally in acute disseminated encephalomyelitis as in multiple sclerosis.

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Herderschee *et al* reply:

The criticism by Dr Poser of the diagnosis of multiple sclerosis in our patient is based upon his denial of the presence of dissemination in time and in space. The emergence of new lesions has, admittedly, not been demonstrated because no MR scan was made in the early stage of the disease.

The following facts, however, are against the alternative diagnosis of acute disseminated encephalomyelitis: (1) there was no history of antecedent infection or vaccination;¹ (2) systemic symptoms (fever, headache, nausea) which are included in all descriptions of acute disseminated encephalomyelitis, were absent.² Our patient was in an excellent general condition; (3) the EEG was entirely normal;³ (4) the CT scan in acute disseminated encephalomyelitis may be normal, but if abnormal it usually shows extensive and multiple cortical enhancing lesions or subcortical non enhancing lesions, or both.⁴

Ancillary investigations may not distinguish between a first attack of multiple sclerosis and acute disseminated encephalomyelitis, but clinical data and circumstantial evidence do. Considering the clinical presentation and results of ancillary investigations the diagnosis of acute disseminated encephalomyelitis is improbable. In our opinion multiple sclerosis is the most likely diagnosis.

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Palatal myoclonus influenced by neck posture

Sir: We read with great interest the communication on palatal myoclonus influenced by head posture.¹ Jacobs *et al*² had previously reported two cases of palatal myoclonus, one of which varied with neck posture and the other the myoclonus disappeared after two years. We report a patient with posture-related palatal myoclonus which remitted after three months.

A 52-year-old man presented with a three-month history of a clicking noise emanating from his throat, occurring only in certain neck postures. He had no problems with hearing or swallowing. On examining his throat with his neck flexed anteriorly and to the right, a clicking noise appeared associated with rhythmic contractions of the throat. The palatal myoclonus disappeared with his head in the midline position. It failed to appear in any other neck posture. His CT scan showed mild frontal atrophy. Three months later, his palatal myoclonus had remitted. Various manoeuvres including breath holding, squeezing the hands, curling the toes, moving joints and hyperventilation are known to exacerbate palatal myoclonus. However, reports of precipitation by certain head and neck movements are rare, our case being the third, and the first in which the myoclonus eventually disappeared. Palatal myoclonus is always associated with a disturbance of the Guillain Mollaret triangle with subsequent olivary hypertrophy.³ The

inferior olivary nucleus would seem to play a crucial part in the pathophysiology of palatal myoclonus. It receives afferent fibres from a wide range of structures, including the cerebral cortex,⁴ caudate nucleus, red nucleus, cerebellum and spinal cord.⁵ The gracile and cuneate nuclei project into the anterior cerebellum directly and indirectly via the basilar pons and inferior olive.⁵

As Corbin and Williams¹ have suggested, afferent impulses triggered by neck posture could, by projections to the inferior olive, influence the reverberating circuit which underlies palatal myoclonus. If this is the case, the projections seem influential in only a small proportion of patients with palatal myoclonus, and a particular head posture is not relevant in this respect. Conceivably,

subtle changes in palatal myoclonus triggered by head posture are more common, but only detectable by electrophysiological techniques.

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Book reviews

Parkinson's Disease. (*Advances in Neurology Vol 45*). Edited by Melvin D Yahr, Kenneth J Bergmann. (Pp 680; \$113.50.) New York: Raven Press, 1986.

Here is another conference symposium in the grand style. But it is one with a difference—thanks to the shrewd foresight of its well known editors we are spared the exhaustive exegesis of unintelligible technology which exudes like some opaque excrescence from so many modern conferences. We are not subjected to a morass of badly written, hastily compiled conclusions padding out the very latest numerical and graphic data in ill-digested form. Each of the contributors has presented an overview of current developments, illustrated by a sample of his recent investigations.

Over 500 participants from 28 countries took part, we are told, but mercifully not all were required to share the fruits of their ephemeral knowledge with readers of this volume. There are four major themes: epidemiological and aetiological factors; biochemical and ultrastructural pathology; motor and non-motor aspects of Parkinson's disease; and, current and future approaches to therapy.

The result is a truly up to date, lucid review of the subject, heavily laden with modern techniques and seminal data which will appeal not only to research workers, but to the jobbing clinical neurologist who seeks further understanding of this intriguing and still bewildering complex of motor and neuropsychological disorder. It is well

presented with good illustrations and tabular material. The authors provide valuable information over almost the entire field so that it is invidious to pick out some of the many topics which are important, yet which so frequently escape attention in less comprehensive anthologies. The editors are to be congratulated on managing to distil such a useful compendium which (price apart) deserves a wide readership.

JMS PEARCE

Congenital Disorders and Infections and Cancer. Handbook of the Spinal Cord Vols 4 & 5. Edited by Robert A Davidoff. (Pp 848; \$149.75 USA & Canada; \$179.50 others.) New York: Marcel Dekker, 1987.

These are the final two volumes in a series of five which extensively cover the anatomical, physiological, biochemical, pharmacological and clinical aspects of spinal cord function and dysfunction. It is a multi author series and as such the quality of the chapters shows some variability. Volumes 4 and 5 cover congenital and traumatic disorders in part I and infections and cancer in part II. In total there are twenty chapters with seven hundred and seventy nine pages including an extensive author and subject index.

The first chapter sets the clinical tone of the book and illustrates with many references the basic spinal cord syndromes as they present in practice. The following four chapters are concerned with spinal cord trauma including the pathophysiological mechanisms occurring during injury, and

spinal cord regeneration. The chapter on spinal cord trauma and its early clinical management is very good. It has, perhaps, a slight bias towards operative intervention but nevertheless places this in its true context and is not blind to the substantial evidence in the literature which indicates that early intervention rarely improves neurological function with the exception of a few uncommon clinical situations. It includes the use of computed tomography and MRI which is not yet freely available to Spinal Injury Centres in the UK. Whether this high technology in radiological investigation will make any difference to the ultimate outcome, remains to be seen. The chapter on neurophysiology of spinal spasticity was written at the theoretical and investigational level. I was disappointed that there was no clinical section dealing with the problems of management of painful spinal spasticity following traumatic paraplegia.

There is an adequate but not over-inspiring chapter on cervical spondylosis and stenosis which covered most aspects of this common condition. However, major omissions concerned the natural history and progression of the disease process and there was little or no mention of MRI in cervical degenerative disease. I found this slightly surprising considering the author works in a major unit in San Francisco. Nevertheless the illustrations were of high quality and the chapter was extremely well referenced.

For the remainder of part I my interest waned slightly when confronted with an over-view of inherited disorders of the spinal cord, neural tube defects and autonomic nervous system diseases of the spinal cord. Nevertheless these provide a valuable source