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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Reference

- 1 Herderschee D, Stam J, Derix MMA. Aphemia as a first symptom of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1987;50:499.

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

Matters arising

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