E: Can you see it?
P: Yes, I can. It's very strange.
E: How many legs do you have?
P: Two.
E: Do they both work?
P: Yes, they do. It's the third hand that's peculiar. I don't want to talk about it.

As in the previous patient we have reported,[1] the supernumerary phantom occurs in the context of severe left hemiplegia and sensory loss associated with left neglect. In the earlier case, however, the lesion was a subcortical haemorrhage and there was no visual field deficit. Cognitively, both patients were fully aware that their left arm was paralysed and of the resulting handicap. Likewise, in both patients, there was a firm conviction of the "reality" of the third limb in conjunction with (rational) bafflement by the anomaly of the experience. Both men well realised that others would find their claim unbelievable, and were hence disinclined to discuss the issue at length. Why reduplication phenomena are typically (but not invariably) consequent on right hemispheric pathology[3] remains to be determined.

In reports of supernumerary phantoms prior to our own, the patients were usually seen in the acute phase, and it is generally assumed that, like anosognosia, the phenomenon remits rapidly. This patient, by contrast, shows that, like "ordinary" phantoms, the experience of a supernumerary limb can persist.

P W HALLIGAN
Rivermead Rehabilitation Centre, Oxford, UK
J C MARSHALL
University Department of Clinical Neurology, The Radcliffe Infirmary, Oxford, UK

Correspondence to: Dr J C Marshall, Neuropsychology Unit, University Department of Clinical Neurology, The Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE, UK.

1 Halligan PW, Marshall JC, Wade DT. Three arms: a case of supernumerary phantom limb after right hemisphere stroke. *J Neurol Neurosurg Psychiatry* 1993;**56**:159–66.
2 Weinstein EA, Kahn RL, Malitz S, Rosanski J. Delusional reduplication of parts of the body. *Brain* 1954;77:45–60.
3 Rogers MJC, Franzen MD. Delusional reduplication following closed-head injury. *Brain Injury* 1992;**6**:469-76.

Visually induced paroxysmal nausea and vomiting as presenting manifestations of multiple sclerosis

The protean manifestations and paroxysmal symptoms in multiple sclerosis are well described.[1] We report a patient with clinically definite multiple sclerosis whose first symptoms of the disease were paroxysmal nausea and vomiting induced by visual perception of movement. Closure of his eyes or cessation of the movements led to a remarkably abrupt termination of symptoms.

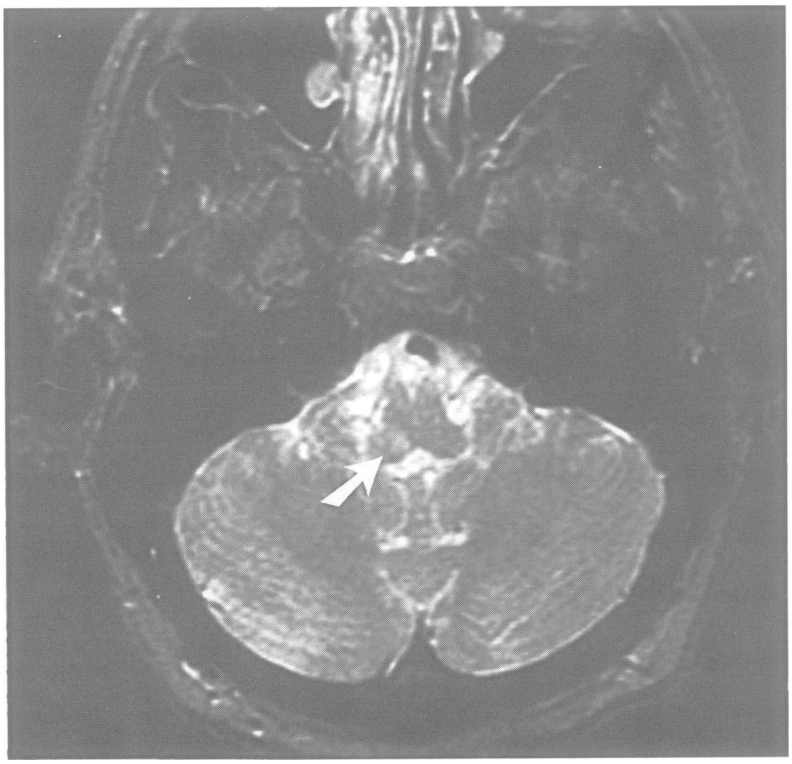
A 47 year old man with the diagnosis of multiple sclerosis was seen in our clinic in August 1993 because of severe paroxysmal nausea and vomiting. These symptoms were induced by perception of any kind of movement in the patient's field of vision. The symptoms would begin abruptly with intense nausea and if the triggering movements persisted, vomiting would soon follow. Movements of any kind (people walking, watching a person getting up from a chair) would all lead to these symptoms. Interestingly, these would occur even if the patient was standing still or lying down in bed. Closure of his eyes or the cessation of movements would abruptly terminate symptoms. These symptoms occurred paroxysmally, lasted three to four hours, and remitted spontaneously. They dated to 1968 when he was first diagnosed with probable multiple sclerosis based on the paroxysmal symptoms and clinical examination. Neurological examination then had shown horizontal gaze evoked nystagmus, generalised hyperreflexia, and bilateral extensor plantar responses. A careful review of the patient's history and records showed paroxysmal nausea and vomiting as the initial manifestations of his disease. Subsequently he had had several such episodes besides other exacerbations including cerebellar ataxia, paraesthesiae and optic neuritis, leading to the diagnosis of clinically definite multiple sclerosis in 1984.

Treatment with routine antiemetics had always failed and over the course of years, the patient had learned to control his symptoms by closing his eyes or having the inciting movements stopped if possible. This turned out to be a consistent cure for his symptoms although they clearly affected his professional and social life. The patient's medical history was otherwise unremarkable.

General physical examination and review of systems including the gastrointestinal system were normal. Neurological examination showed bilaterally decreased olfaction, decreased gustation over the entire tongue, a pale right optic disc, generalised hyperreflexia with extensor plantar responses, horizontal lateral gaze nystagmus, and moderate impairment of tandem walking. During the course of examination, the patient experienced severe nausea, which he attributed to the examiner's movements. He subsequently vomited and then closed his eyes, which led to cessation of nausea and vomiting. Resumption of the examination led to their recurrence and this time, on the patient's request, the examiner remained stationary in his seat, which also resulted in the resolution of symptoms.

Routine serology was negative including rapid plasma reagin, angiotensin converting enzyme, and Lyme titres. Analysis of CSF showed oligoclonal bands with total protein of 67 mg/dl (normal 15–45 mg/dl), but normal cell count and myelin basic protein concentration. Cultures of CSF were negative. An ECG showed sinus bradycardia at a rate of 50/minute; EEG was normal. Auditory and somatosensory evoked potentials were normal. Pattern visual evoked potentials were abnormal on the right, suggestive of a lesion anterior to the optic chiasm, and normal on the left. Brain MRI showed multiple areas of high signal on the T2 weighted images seen in the left optic radiation and throughout the posterior portion of the midbrain and pons, near the collicular plate, and the floor of the fourth ventricle near the area postrema (figure). Cervical MRI was normal.

Paroxysmal symptoms have been reported as the initial manifestations of multiple sclerosis.[2] Vomiting has been reported as a prominent symptom in the disease, especially in the newly diagnosed adolescent population in the early stages.[3] We report paroxysmal nausea and vomiting induced by visual perception of movement as presenting symptoms of multiple sclerosis. These symptoms could be abruptly terminated by cessation of movements or closure of eyes. This was the easiest and



Axial second echo of T2 (TR/TE = 2000/85) shows high signal in the right area postrema consistent with a multiple sclerosis plaque (arrow). (Signa 1·5 T GE Medical Systems, Milwaukee, WI, USA.)

most effective cure for the patient. Routine antiemetics had minimal or no effect on his symptoms. This clinical syndrome, to the best of our knowledge is the first of its kind in the neurological literature.

Vomiting has been associated with a chemoreceptor trigger zone in the area postrema and a vomiting centre, both located in the medulla oblongata.[4] The location and existence of the vomiting centre is, however, controversial. A possible anatomical pathway from the retina to the vomiting centre and the chemoreceptor trigger zone in the area postrema may explain the symptomatology in our patient. Retinal ganglion cells project to the primary visual cortex (Brodmann area 17) for visual perception. Efferents from the visual cortex project to the superior colliculus, which is known to send efferents to the pontine and medullary reticular formation,[5] reticular formation being the site of the vomiting centre. As visualised in brain MRI (figure) a large lesion occupied the posterior portion of the brainstem including the medulla. Ephaptic spread, arguably the most accepted explanation of paroxysmal symptoms in multiple sclerosis,[6] from this lesion could certainly involve the vomiting centre and the chemoreceptor trigger zone in the area postrema. Furthermore, involvement of the nucleus of the tractus solitarius could also lead to nausea and vomiting as it is reciprocally connected to the area postrema. Despite the plausibility of this explanation, one needs to bear in mind that clinical symptoms and lesions seen on MRI in multiple sclerosis do not always correlate.

Curiously, despite the protean manifestations of paroxysmal symptoms in multiple sclerosis, paroxysmal nausea and vomiting have never been reported as manifestations of multiple sclerosis. The differential diagnosis of paroxysmal vomiting is complex and among the many causes, a psychogenic basis has also been emphasised. In one report, intractable hiccups were reported as manifestations of multiple sclerosis.[7] In the same paper, the authors quoted several surveys emphasising a psychogenic basis of intractable hiccups, thereby raising the possibility of misdiagnosing multiple sclerosis as a conversion reaction. We agree with their point of view as paroxysmal nausea and vomiting without neurological symptoms may easily mislead the clinician towards a psychogenic aetiology, if other tests are negative. The possibility of multiple sclerosis should be considered when evaluating patients with paroxysmal symptoms such as nausea and vomiting.

OMAR A KHAN
GEORGE M SANDOZ
MICHAEL J OLEK
Department of Neurology, Medical College of Virginia, Richmond, Virginia, USA
ARNOLD J KUTA
Division of Neuroradiology, Department of Diagnostic Radiology, Medical College of Virginia, Richmond, Virginia, USA

Correspondence to: Dr Omar A Khan, Maryland Center for Multiple Sclerosis, Department of Neurology, University of Maryland Hospital, 22 South Greene Street, Baltimore, MD 21201, USA.

1 Osterman PO, Westerberg CE. Paroxysmal attacks in multiple sclerosis. *Brain* 1975;**98**: 189–202.
2 Twomey JA, Espir MLE. Paroxysmal symptoms as the first manifestations of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1980; **43**:296–304.
3 Sindern E, Haas J, Stark E, Wurster U. Early onset MS under the age of 16: clinical and paraclinical features. *Acta Neurol Scand* 1992;**86**:280–4.
4 Davis CJ, Lake-Bakaar GV, Grahame-Smith DG. *Nausea and vomiting: mechanisms and treatment.* Heidelberg: Springer-Verlag, 1986.
5 Williams PL, Warwick R, Dyson M, Bannister LH, eds. *Gray's anatomy.* 37th ed. London: Churchill Livingstone, 1989:985–7.
6 Ekbom KA, Westerberg CE, Osterman PO. Focal sensory-motor seizures of spinal origin. *Lancet* 1968;i:67.
7 McFarling DA, Susac JO. Hoquet Diabolique: Intractable hiccups as a manifestation of multiple sclerosis. *Neurology* 1979;**29**: 797–801.

MATTERS ARISING

Progressive supranuclear palsy: neuropathologically based diagnostic clinical criteria

Collins *et al*[1] provide a valuable set of criteria to aid in the clinical diagnosis of progressive supranuclear palsy. They include as a prerequisite the absence of family history: this is based on their own series of 12 patients who did not have a positive family history and the fact that progressive supranuclear palsy is considered to be sporadic. We have reported a family with autosomal dominant progressive supranuclear palsy, one member of whom would have fulfilled the criteria of Collins *et al*[1] were it not for the family history.[2] Details on the other family members were insufficient to apply the criteria but members of the family showed classic neuropathological changes at necropsy. Thus, progressive supranuclear palsy shares with many other neurodegenerative diseases, such as Alzheimer's and Pick's disease, a phenotype common to both sporadic and autosomal dominant cases. Whereas the classification of such cases as separate diseases or subtypes is arguable, the current usage in the field of Alzheimer's disease is to consider autosomal dominant familial cases as a subtype. The prerequisite of an absent family history may unnecessarily exclude cases of familial progressive supranuclear palsy.

M N ROSSOR
J BROWN
Department of Neurology, The National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK

Correspondence to Dr M N Rossor.

1 Collins SJ, Ahlskog JE, Parisi JE, Maraganore DM. Progressive supranuclear palsy: neuropathologically based diagnostic clinical criteria. *J Neurol Neurosurg Psychiatry* 1995; **58**:167–73.
2 Brown J, Lantos P, Stratton M, Roques P, Rossor M. Familial progressive supranuclear palsy. *J Neurol Neurosurg Psychiatry* 1993; **56**:473–6.

NOTICES

First European Forum of quality improvement in health care. QEII Conference Centre, London. 7–9 March 1996.

This first European Forum will allow the exchange of ideas on quality improvement in health care and provide education. The forum will consist of plenary lectures, parallel seminars and workshops and discussions and short educational courses.

The themes of the first forum are:

- The fundamentals of continuous quality improvement
- Achieving patient orientation
- Leadership and managing organisational change
- Improved quality and reducing costs
- The importance of measurements
- Involving everybody in quality improvements
- Professional education for quality
- The politics of quality.

For more information contact: Clare Moloney, BMA Conference Unit, BMA House, Tavistock Square, London WC1H 9JP. Fax: 0171 383 6663. Tel: 0171 383 6478.

World Federation of Neurosurgical Societies: awards to young neurosurgeons

The World Federation of Neurosurgical Societies will give five awards to young neurosurgeons for the best papers submitted for presentation at the XI International Congress of Neurological Surgery to be held in Amsterdam, Netherlands on 6–11 July 1997. This will be open to all neurosurgeons born after 31 December 1961. Each award will consist of an honorarium of US $1500, a certificate, and complete waiver of registration fees along with accommodations for the Congress. The papers will be judged by a committee and must contain original, unpublished work on basic research orclinical studies related to neurosurgery.

Young neurosurgeons should submit eight copies of the manuscript (not more than 10 double spaced typewritten pages exclusive of figures and tables) to:

Albert L Rhoton Jr MD, Chairman
WFNS Young Neurosurgeons Committee,
Department of Neurological Surgery,
University of Florida Medical Center,
PO box 100265; 1600 SW Archer Road,
Gainesville, Florida 32610-0265, USA

The submission should be accompanied by a supporting letter from the Head of the candidate's neurosurgical department. The last date for submission is 1 October 1996.