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.

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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.

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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:

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- Achieving patient orientation
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- The importance of measurements
- Involving everybody in quality improvements
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- 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.