

gastrointestinal tract even in the absence of localising symptoms or signs.

TP ENEVOLDSON,
JA BALL,
JM MCGREGOR,
Department of Neurology,
St Thomas' Hospital,
London SE1 7EH, United Kingdom

- 1 McLeod JG, Walsh JC. Peripheral neuropathy associated with lymphomas and other reticulosos. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. *Peripheral Neuropathy*, 2nd Ed. London: WB Saunders, 1984: 2192-203.
- 2 Moore RY, Oda Y. Malignant lymphoma with diffuse involvement of the peripheral nervous system. *Neurology (Minneapolis)* 1962;12: 186-91.
- 3 Croft PB, Urich H, Wilkinson M. Peripheral neuropathy of sensorimotor type associated with malignant disease. *Brain* 1967;90: 31-66.
- 4 Blanchard BM. Peripheral neuropathy (non-invasive) associated with lymphoma. *Ann Intern Med* 1962;56:774-8.
- 5 Schold SC, Cho ES, Somasunderam M, Posner JB. Subacute motor neuropathy; a remote effect of lymphoma. *Ann Neurol* 1979;5: 271-87.

We thank Dr RW Ross Russell for permission to report one of his patients, Mr KG Burnand for performing the surgery, and Professor RACHughes for reporting the nerve biopsy.

Dysphagia due to a pharyngeal mucocoele mimicking myasthenia

Paranasal mucocoeles on occasion may cause neurological deficits.^{1,2} By contrast, mucocoeles of the oropharynx and trachea rarely produce neurological complications.³ We report a case of progressive dysphagia in a myasthenic patient caused by an aryepiglottic mucocoele.

A 29 year old right handed black woman with congenital myasthenia gravis, and a history of depression, presented to the emergency department two days after developing suicidal thoughts and abruptly discontinuing her medication which included pyridostigmine. In addition to her psychiatric symptoms, she complained of headaches, fatigue, diplopia, dysarthria and dysphagia. The dif-

ficulty she had in swallowing was progressive over several weeks, equal for solids and liquids, and accompanied by the sensation of a painless "lump in the throat". Although she had experienced swallowing difficulties in the past related to myasthenia, her current dysphagia was qualitatively different, although she could not elaborate further. Her history was significant with a thymectomy at the age of nine and numerous admissions for chronic upper respiratory infections and myasthenia. She had a long history of depression and a personality disorder.

The head and neck examination was normal without evidence of masses. Neurological examination revealed bilateral ptosis and dysarthria. The oropharynx was clear, the gag reflex was intact and she swallowed water without difficulty. Volitional motor testing was unreliable. The rest of the neurological examination was normal. An MRI of the head demonstrated a mass in the hypopharynx (fig). A left ary-epiglottic cyst was removed by direct laryngoscopy which proved to be a mucocoele on microscopic sections. Post operatively, the dysphagia resolved and the myasthenia was controlled with pyridostigmine.

A mucocoele is a mucus gland retention cyst which typically arises in the cranial sinuses, although other head and neck structures may be involved. Predisposing factors for the development of a mucocoele include trauma, structural abnormalities, and chronic infections.⁴ Neurological complications may arise depending on the location of the mucocoele. Paranasal mucocoeles may cause a variety of cranial neuritides¹ and suprasellar extension may mimic a pituitary adenoma.² By contrast, upper respiratory tract mucocoeles may cause airway compromise, but neurological manifestations are distinctly rare.³

A clinician may not be overtly concerned with a depressed patient complaining of a "lump in the throat" or a myasthenic patient experiencing bulbar symptoms while off anticholinesterase medications. This case illustrates the difficulty inherent in the psychiatric patient with an organic complaint which may be further confounded by an established diagnosis. Mucocoele should be included in the differential diagnosis of the

predisposed patient who develops cranial neurological symptoms.

SYLVAIN S JUNGER, ROBERT B WRIGHT,
Rush-Presbyterian-St Luke's Medical Center,
1653 West Congress Parkway, Chicago,
Illinois 60612, United States

- 1 Johnson LN, Hepler RS, Yee RD, Bazdorf U. Sphenoid sinus mucocoele mimicking diabetic ophthalmoplegia and retrobulbar neuritis. *Am J Ophthalmol* 1986;102:111-5.
- 2 Abia AA, Maroon JC, Wilberger JE, Kennerdell JS, Deeb ZL. Intracellar mucocoele simulating pituitary adenoma: Case report. *Neurosurgery* 1986;18:197-9.
- 3 Redmond FA, Rogers CS. Acute airway compromise due to tracheal mucocoele. *Ann Emerg Med* 1987;16:445-6.
- 4 Manusco AA, Hanafey WN. *Computed tomography and magnetic resonance imaging of the head and neck*. Baltimore: Williams and Wilkins, 1985:22-4.

Trazodone is ineffective in essential tremor

The pathophysiology of essential tremor (ET) is not known. No gross structural changes in the nervous system have been found in ET,¹ so it is presumed that it results from an abnormality in some aspect of neuronal function, for example, changes in transmitter levels. In a single study using positron emission tomography, increased glucose metabolism was found in the inferior olive in patients with ET.² It has been suggested that rhythmic activity of neurons in the inferior olive may play a role in the generation of symptomatic tremor in humans.³ Olivary neurons receive a serotonergic input,⁴ and a recent paper reported a good result from the serotonin agonist trazodone hydrochloride (Molipaxin), 150 mg/day, in two patients with ET.⁵ The study was not, however, placebo controlled and no objective quantitation of tremor was used. We therefore carried out a double-blind, placebo controlled trial of trazodone in ET.

Fourteen patients with ET, not taking any form of medication, were entered into the study. Nine patients (mean age 60 years, range 41-74 years) completed the study and five withdrew for personal reasons. Patients received, in random order, trazodone 50 mg twice daily for one week followed by 50 mg three times a day for three weeks or a matching placebo. At the end of each treatment period, tremor was assessed in the laboratory 1.5 hours after the morning dose. Postural tremor of the hands (arms supported to the wrists with hands held horizontally in pronated posture) was evaluated by 1) accelerometry, according to a method previously described,⁶ 2) clinical rating on a scale from 0 (no visible tremor) to 5 (gross, self-injuring tremor), 3) patients' self-rating from +5 (large increase in tremor), through 0 (no change), to -5 (large reduction in tremor), 4) manual performance test (spiral tracing) scored for accuracy by three assessors on a scale from 0 (smooth tracing, no errors) to 5 (very tremulous tracing, many errors).

Results showed no statistically significant differences between trazodone and placebo on any of the measures used. Median tremor magnitude (in milli-g, where $g = 981 \text{ cm/s}^2$) was 23.4 (placebo), 29.7 (trazodone); mean scores for clinical rating (placebo/trazodone respectively) were 2.3/2.3; self rating 0.2/0.0;

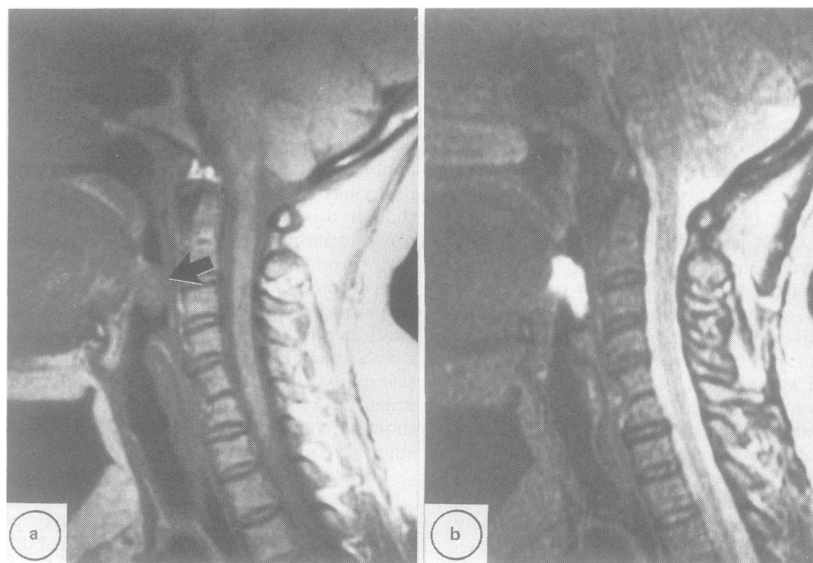


Fig Magnetic resonance image of the head. A T1 weighted image reveals a mass in the hypopharynx (arrow). B The mass demonstrates increased signal intensity on a T2 weighted image.