

This is particularly true with rapid tumour enlargement, as may occur with haemorrhage into the tumour.⁷ Other possible causes of a sixth nerve palsy related to pituitary adenoma are hydrocephalus due to obstruction of the foramen of Monro by tumour, and a coexistent cerebral aneurysm.^{8,9}

Bromocriptine, a dopamine agonist, is a potent inhibitor of the synthesis and release of prolactin. It may dramatically reduce the size of large prolactinomas and there have been several reports of improvement of visual fields and extraocular movements after its administration.^{10,11} The tumour shrinkage is most likely related to reduction in cell size. There is no evidence of widespread tumour necrosis, vascular injury, platelet aggregation or thrombosis after treatment with bromocriptine.^{10,11}

Our patient developed a sixth nerve palsy in association with a rapid reduction in prolactinoma size, including its extrasellar components. There was no evidence of localised tumour expansion or haemorrhage, hydrocephalus, cerebral aneurysm, recent viral infection or other identifiable causes for the nerve palsy. Although simultaneous independent disease is a possibility, we consider that sixth nerve damage was more likely to have been related to rapid decompression or shift in position of the nerve caused by tumour shrinkage. Hence, the appearance of a sixth nerve palsy during treatment of a large prolactinoma with bromocriptine may signify either rapid tumour expansion or, more rarely, a sudden decrease in tumour size.

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Accepted 15 June 1986

No enophthalmos in Horner's syndrome

Sir: Enophthalmos is a controversial feature in Horner's syndrome. Some regard it as mere illusion created by narrowing of the palpebral fissure.¹ We investigated the presence of this sign in a prospective series of patients with oculosympathetic dysfunction.

Thirteen patients with unilateral oculosympathetic dysfunction were examined with a "Krahn" exophthalmometer. This instrument makes it possible to measure the exact distance between the anterior surface of the cornea and the lateral margin of the orbit. The patients were examined while sitting. The readings were "blind", owing to an error of calibration, which caused an artificial difference between the right and left eye, and of which the examiner was unaware. We excluded all intracranial or intraorbital diseases that could affect the position of the eyeball other than by oculosympathetic denervation. Horner's syndrome had been present in these patients from a few months up to more than ten years. The diagnosis of unilateral oculosympathetic dysfunction was based on previously published criteria involving serial photography of the pupil.²

The exophthalmometric readings in the 13 patients with oculosympathetic dysfunction gave an average of 16.2 mm on the side of oculosympathetic dysfunction and 15.8 mm at the normal side (after correction for the error of calibration). In five cases there was no difference at all, three cases showed enophthalmos (0.5 mm or 1.0 mm) and five showed exophthalmos (1 mm or 2 mm). Four patients had Horner's syndrome for more than ten years, one for at least five years, the rest for two years or less.

Enophthalmos should no longer be regarded as a part of Horner's syndrome. In his original description Horner mentioned only in passing that the position of the eyeball seemed slightly inward ("sehr unbedeutend zurückgesunken").³ The casualness of this remark contrasts with the completeness of his description of miosis and ptosis and with his measurements of the temperature of the face. Later writers, however, have included enophthalmos among the main features of the syndrome, and Horner's chance remark has been perpetuated into the textbooks of the present day.⁴⁻⁶ Our measurements fail to show even the slightest relationship between enophthalmos and oculosympathetic dysfunction, not even in patients with miosis and ptosis of more than 10 year's standing. Similar findings have recently been reported by Lepore⁷ and Nielsen.⁸

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Accepted 17 January 1986

Aphemia as a first symptom of multiple sclerosis

Sir: Speech disorders are common in the course of multiple sclerosis, but aphasia is rare.¹⁻³ We describe a patient with aphemia, that is, an articulation disorder caused by a left hemisphere lesion, as a first symptom of multiple sclerosis.

A 29 year old right handed female medical student complained of speech problems and of a loss of strength in the right hand, which had developed insidiously in 5 days. The speech difficulties mainly consisted of an altered inflection. Writing was unimpaired, but less skilfully performed. She had no other symptoms and her medical history was uneventful. Apart from contraceptives no medication was taken. Neurological examination disclosed a minimal weakness of the right arm and leg. Cranial nerves, coordination, sensory testing, and reflexes were normal, with both plantar responses flexor. There were no signs of bucco-facial apraxia. There was no dysphagia, increase of facial reflexes or lability of emotional expression. Her speech was monotonous with a loss of interpunction and emotional expression. Neuropsychological examination did not show any language disorder. Conversational speech was fluent with a normal syntax and vocabulary, without paraphasias. Comprehension of spoken and written language was excellent. Confrontation naming, series speech, reading aloud, writing, and repetition of spoken language were entirely normal. No ideational or ideomotor apraxia was present. Visuospatial skills, tactile perception and memory functions were unimpaired. There was no attentional or planning disturbance.

The speech difficulty was characterised by hesitation in starting a sentence and in pronouncing words starting with a consonant. She did not simplify certain phonemes, but rather gave the impression of stuttering although there was no actual repetition of phonemes. The speech difficulty was not

constant for a certain word or sound, but changed during conversation. Inflection was radically altered. She could not sing a scale or simple songs because of the dysprosody, but humming was normal. The patient was definitely concerned about her symptoms.

Extensive blood examination disclosed no abnormality. There was no serological evidence of viral or treponemal infection. A contrast enhanced CT scan of the brain on admission was normal. Cerebrospinal fluid (CSF) contained 73 erythrocytes, 16 lymphocytes, and 2 monocytes per mm³. Total protein was 0.35 g/l with an IgG index of 1.58 (normal less than 0.60) and a normal albumin ratio. Electrophoresis showed oligoclonal bands confirming intrathecal IgG synthesis EEG and evoked potential studies (visual, sensory and auditory) were normal. Within a few days the weakness disappeared and in 2 weeks there was a resolution of the speech disorder. A second CT scan now showed a hypodense area with central contrast enhancement in the frontal white matter of the left hemisphere (fig a). She was discharged in good condition and resumed her studies without signs of recurrent neurological deficit during a follow-up of one year. After 6 months magnetic resonance imaging (MRI) showed multiple paraventricular lesions (fig b). Spontaneous recovery, CSF findings, and lack of any other explanation were suggestive of multiple sclerosis. In combination with the MRI findings 6 months later the criteria for laboratory supported definite multiple sclerosis were fulfilled.^{4,5}

Abnormal speech with normal propositional language can be subdivided into three groups according to the kind of articulatory disorder and the accompanying neurological symptoms and signs: dysarthria, aprosodia, and aphemia. In dysarthria the articulation disorder is basically a simplification of articulation with articulatory distortions.⁶ In the aprosodies the affective components of speech are impaired after right hemisphere lesions.⁷ The third group, called aphemia or apraxia of speech comprises articulation disorders which have caused much debate. Different authors have stressed aphasic, dysarthric, or apraxic aspects, and named this group of articulation disorders accordingly.⁸ Basically aphemia concerns involvement of a purely expressive aspect of spoken language, and as such it is difficult to distinguish from motor aphasia.⁹ Propositional language however is entirely normal. There has been discussion about the interpretation of phonematic errors as *aphasic* or *apraxic*.¹⁰ The inconsistency of the articulatory problems is used as a distinction from dysarthria and to stress the apraxic aspect.⁶ A frequent concomitant finding is oral or facial apraxia.¹¹ The symptoms of our patient fit in with the picture of aphemia. A left-sided lesion of the posterior part of the inferior frontal lobe and especially the deep white matter of the anterior limb of the internal capsule is the most consistent pathological finding. The aetiology is nearly always vascular or traumatic.⁶ In our patient a lesion became visible on the CT scan which extended into the area of the

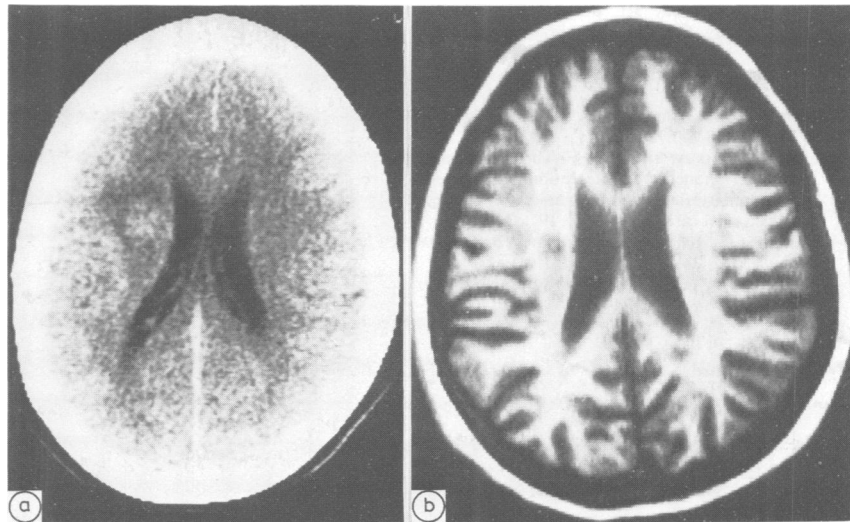


Fig (a) Contrast enhanced CT scan at the level of the cella media, (b) axial MRI scan, 6 months later, at the level of the cella media. (TR 1400 ms; TE 32 ms; TI 400 ms).