Letters

since most communications were not based upon detailed phonetic characterisation of changes along repetitions. An exception is a case report of assumed simple schizophrenia,²⁰ documenting that there was no consistent decrease in either word duration and intensity along repetition trains (see also LaPointe and Horner⁷). Presumably, our finding of an improvement in articulatory and vocal performance along repetition trains does not reflect voluntary control, since this would not conform with the patient's inability to interrupt the repetition loop. A more plausible explanation would rather ascribe the observed resetting of phonetic parameters to spontaneously occurring shifts of psychomotor drive ("energising").

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Ophthalmoplegic migraine with bilateral involvement

Sir: Ophthalmoplegic migraine is characterised by attacks of unilateral headache and transient dysfunction of one or more ocular motor nerves. To our knowledge, bilateral involvement in a single attack has not previously been described.

A 37 year old Caucasian woman had experienced migraine attacks approximately

twice monthly since aged 12 years. Usually 2 hours scintillating photopsia was replaced by left frontal headache, with vomiting, diarrhoea and occasional tingling of hands and feet. Her mother experienced frequent similar episodes. The patient suffered a typical attack. At its onset she took her usual therapy of two capsules, each containing 1 mg ergotamine tartrate, 5 mg prochlorperazine, 5 mg chlorthalidone, 250 mg paracetamol, 250 mg aspirin and 8 mg codeine. The following day she awoke with left ptosis and diplopia.

Visual acuity was 6/6 in each eye, visual fields were full and the optic fundi were normal. There was moderate left ptosis. Both saccadic and pursuit eye movements were abnormal. Gaze to left, right and downwards was limited to about 10° (fig). Upgaze was impossible, and convergence severely limited. Abduction was slightly greater than adduction on right lateral gaze, although eyes were otherwise conjugate. No improvement occurred with oculoccephalic testing. Both pupils were 5 mm diameter and unresponsive to light and accommodation. The remainder of a full neurological and general medical examination was normal.

Blood count, ESR, automated biochemistry, treponemal serology, red cell transketolase and chest radiograph were normal. Computed tomography of brain with and without contrast enhancement was unremarkable, as was bilateral carotid and left vertebral angiography. Cerebrospinal fluid contained no cells and oligoclonal banding was absent, although protein was 0.69 g/l (0.15-0.40) and IgG: albumin ratio 0.29 (0-0.10). Nerve conduction studies and visual evoked potentials were normal.

One week later, ptosis and headache had largely resolved, pupil reactions to light and convergence had returned, and range of eye movements had increased. Ocular movements, however, had become more dysconjugate with adduction being 5° greater than abduction on left and right lateral gaze, and depression of the left eye being 5° less than the right on downgaze.

By 10 weeks the ophthalmoplegia had resolved. Pizotifen was then administered and resulted in reduced headache frequency. There has been no recurrence of ophthalmoplegia after five years.

In 1882 Saundby¹ described a young woman with recurrent migraine and ophthalmoplegia. Eight years later Charcot² labelled a similar case as "migraine ophtalmoplegique". Many early cases, however, were subsequently discovered to have structural intracranial pathology, prompting development of diagnostic criteria. Those of

Fig 1 Patient on day after admission attempting maximum gaze in the directions shown by arrows. Appearance in primary position of gaze identical to that on attempted upgaze (lower left).

Walsh and O'Doherty³ state that there should be (1) a history of typical migraine, (2) ophthalmoplegia involving one or more nerves on one side or alternating sides, and (3) exclusion of other causes by arteriography, surgery or necropsy.

The condition is uncommon, affecting approximately 1 in 600 migraine sufferers.⁴ Patients are usually under 30 years of age, and have longstanding migraine. Following a headache, which is almost always periorbital, ptosis and oculomotor palsy develop. Pupillary paralysis is almost invariable.^{4,5} Recovery occurs over days to months but recurrences may result in permanent deficit. Abducens nerve is involved alone in approximately 10% of cases,⁶ and occasionally oculomotor palsy is accompanied by trochlear,⁷ trigeminal,⁸ facial,⁹ or hypoglossal¹⁰ nerve involvement.

Ocular paralysis usually seems to result from a peripheral lesion, but the pathophysiology has been debated. Possible mechanisms include direct pressure on ocular motor nerves³ or occlusion of the small vessels supplying them¹¹ due to carotid artery oedema. Angiographic narrowing of the ipsilateral intracavernous carotid has been demonstrated in this disorder.³

Ophthalmoplegic migraine involving episodic paralysis of convergence¹² or convergence and upgaze¹³ has rarely been reported. These instances suggest that ocular paralysis occasionally results from brainstem involvement.

The current case fulfils established criteria for diagnosis of ophthalmoplegic migraine. There was no definite evidence of other diseases which cause ophthalmoplegia and

the minor CSF protein abnormalities were non-specific. In particular there were no other features suggesting multiple sclerosis or Miller-Fisher syndrome.

Of specific interest was the bilateral involvement. While there have been isolated reports³ of ocular paralysis alternating sides in successive migraine attacks, bilateral involvement in a single episode has not, to our knowledge, been reported. Although no lesion was demonstrated, central pathology was considered unlikely as such a lesion would have to involve both midbrain and pons, and other clinical evidence of this was lacking. In addition, both levator palpebrae superioris muscles are believed to be innervated by a single midline nucleus and involvement of this would produce bilateral ptosis.¹⁴ Finally, the recovery of eye movements proceeded asymmetrically. We thus suspect ophthalmoplegia resulted from bilateral peripheral lesions, possibly within the cavernous sinuses.

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Seizures as the initial manifestation of paralytic rabies

Sir: Typical phobic spasms in the form of aerophobia and hydrophobia are helpful signs in making a diagnosis in patients with encephalitic rabies. These signs, however, do not necessarily persist throughout the whole clinical course of the diseases. They may present in only half of the cases of paralytic rabies.¹ The clinical diagnosis of rabies can thus be extremely difficult. We here describe a patient with rabies who had paralysis resembling the Guillain Barré syndrome (GBS) and who initially presented with absence like seizures. She had no aerophobia