since most communications were not based upon detailed phonetic characterisation of changes along repetitions. An exception is a
case report of assumed simple schizophrenia,
documenting that there was no consistent
trend in either word duration and intensity along repetition trains (see also
LaPointe and Horner'). 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 character-
ised 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, diarr-
hoea 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 prochlor-
perazine, 5 mg chlor Diazepoxide, 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 oculocephalic
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 trans-
ketolase 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 dys-
conjugate with addition 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 ophthalm-
oplegia 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 oph-
thalmoplegique". Many early cases, however,
were subsequently discovered to have struc-
tural intracranial pathology, prompting
development of diagnostic criteria. Those of
Walsh and O'Doherty 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. Patients are usually under 30 years of age, and have longstanding migraine. Following a headache, which is almost always peri-orbital, ptosis and oculomotor palsies develop. Pupillary paralysis is almost invariable. Recovery occurs over days to months but recurrences may result in permanent deficit. Abducens nerve is involved alone in approximately 10% of cases, and occasionally oculomotor palsy is accompanied by trochlear, trigeminal, facial, or hypoglossal 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 nerves or occlusion of the small vessels supplying them due to carotid artery oedema. Angiographic narrowing of the ipsilateral intracavernous carotid has been demonstrated in this disorder. Ophthalmoplegic migraine involving episodic paralysis of convergence or convergence and upgaze 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 reports 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. 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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Letters

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. 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.
Ophthalmoplegic migraine with bilateral involvement.

D O Hutchinson and I M Donaldson

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