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 Horner[7]). 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").

H ACKERMANN*
W ZIEGLER†
W H OERTEL‡
*Department of Neurology, University of Tübingen,**
Department of Neuropsychology, Max-Planck-Institute for Psychiatry, München,†
Department of Neurology, Klinikum Großhadern, University of München.‡
Federal Republic of Germany.

Address for correspondence:
Dr H Ackermann,
Department of Neurology,
University of Tübingen,
Liebermeisterstr. 18–20
D-7400 Tübingen, FRG

References

1 Souques MA. Palilalie. *Rev Neurol* 1908;**16**: 340–2.
2 Critchley M. On palilalia. *J Neurol Psychopathol* 1927/28;**8**:23–32.
3 Brain R. Speech Disorders: *Aphasia, Apraxia and Agnosia.* Washington, D.C.: Butterworth, 1961.
4 Pick A. Die Palilalie, ein Teilstück striärer Motilitätsstörungen. In: Pick A, ed. *Die neurologische Forschungsrichtung in der Psychopathologie. Beihefte zur Monatsschrift für Psychiatrie und Neurologie,* Heft 13. Berlin: Karger, 1921:178–224.
5 Weingarten K. Tics. In: Vinken PJ, Bruyn GW, eds. *Diseases of the Basal Ganglia. Handbook of Clinical Neurology, Vol. 6.* Amsterdam: North-Holland Publishing Comp, 1968: 782–808.
6 Boller F, Albert M, Denes F. Palilalia. *Br J Dis Comm* 1975;**10**:92–7.
7 LaPointe LL, Horner J. Palialaia: A descriptive study of pathological reiterative utterances. *J Speech Hear Dis* 1981;**46**:34–8.
8 Tissot R, Constantinidis J, Richard J. Pick's Disease. In: Vinken PJ, Bruyn GW, Klawans HL, eds. *Neurobehavioural Disorders. Handbook of Clinical Neurology, Vol. 46.* Amsterdam: Elsevier Science Publishers, 1985: 233–46.
9 Ludlow CL, Rosenberg J, Salazar A, Grafman J, Smutok M. Site of penetrating brain lesions causing chronic acquired stuttering. *Ann Neurol* 1987;**22**:60–6.
10 Mumenthaler M. *Neurology 12 ed.* Stuttgart, New York: Thieme, 1983.
11 Rosenbek J, Messert B, Collins M, Wertz RT. Stuttering following brain damage. *Brain Lang* 1978;**6**:82–96.
12 Fahn S. Fluctuations of disability in Parkinson's disease: Pathophysiology. In: Marsden CS, Fahn S, eds. *Movement Disorders. Vol. 2.* London: Butterworth Scientific, 1982: 123–45.
13 Esteguy M, Bonnet AM, Kefalos J, Lhermitte F, Agid Y. Le test a la L-Dopa dans la maladie de Parkinson. *Rev Neurol (Paris)* 1985;**141**:413–5.
14 Marsden CD, Parkes JD, Quinn N. Fluctuations of disability in Parkinson's disease: Clinical aspects. In: Marsden CE, Fahn S, eds. *Movement Disorders. Vol 2.* London: Butterworth Scientific, 1982:96–122.
15 von Economo C, *Die Encephalitis lethargica. Ihre Nachkrankheiten und ihre Behandlung.* Berlin: Urban and Schwarzenberg, 1929.
16 Marie P. A singular trouble with speech: Palilalia (dissociation of voluntary speech and automatic speech). *Le Monde Medical* 1925;**664**:329–44. Reprinted in: Cole M, Cole M, eds. *Pierre Marie's Papers on Speech Disorders.* New York: Hafner, 1971.
17 Critchley M. *Aphasiology and Other Aspects of Language.* London: Edward Arnold, 1970.
18 McLeod P, Posner M. Privileged loops from percept to act. In: Bouma H, Bouwhuis D, eds. *Attention and Performance X.* Hillsdale: Erlbaum, 1984.
19 Friedrich FJ, Glenn CG, Marin OSM. Interruption of phonological coding in conduction aphasia. *Brain Lang* 1984;**22**:266–91.
20 Kent RD, LaPointe LL. Acoustic properties of pathologic reiterative utterances: A case study of palilalia. *J Speech Hear Res* 1982;**25**:95–9.

Accepted 12 September 1988.

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 chlordiazepoxide, 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 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 Saundby[1] described a young woman with recurrent migraine and ophthalmoplegia. Eight years later Charcot[2] 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

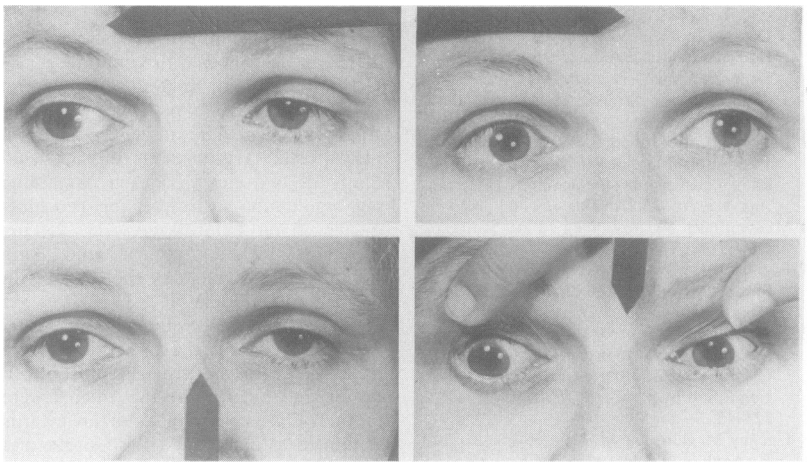

Fig 1 *Patient on day after admission attempting maximum gaze in the directions shown by arrows. Appearance in primary position of gaze identical to that on attempted upgaze (lower left).*

Walsh and O'Doherty[3] 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 hypoglossal[10] 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[3] or occlusion of the small vessels supplying them[11] 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 convergence[12] or convergence and upgaze[13] 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[3] 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.

DO HUTCHINSON
I MACG DONALDSON*
Department of Neurology, Christchurch Hospital and Department of Medicine, Christchurch School of Medicine, Christchurch, New Zealand

*To whom correspondence should be addressed.

References

1 Saundby R. A case of megrin, with paralysis of the third nerve. *Lancet* 1882;**ii**:345–6.
2 Charcot JM. Sur un cas de migraine ophtalmoplegique. *Progr Med* 1890;**15**:83–86, 99–102.
3 Walsh FB O'Doherty DS. A possible explanation of the mechanism of ophthalmoplegic migraine. *Neurology*. 1960;**10**:1079–84.
4 Friedman AP, Harter DH, Meritt HH. Ophthalmoplegic migraine. *Arch Neurol* 1962; 7:320–7.
5 Loewenfeld IW. Pupillary deficit in "ophthalmoplegic migraine". In: Glaser JS, ed. *Neuro-ophthalmology* St Louis: The CV Mosby Company, 1980:180–200.
6 Harris M. Ophthalmolplegic migraine and periodic migrainous neuralgia, migraine variants with ocular manifestations. *Ophthamic Seminars* 1976;**1**:413–50.
7 Bramwell E. A note upon the aetiology of recurrent and periodic ocular palsy and ophthalmoplegic migraine. *Trans Ophthal Soc UK* 1934;**54**:214–21.
8 Patrikios JS. Ophthalmoplegic migraine. *Arch Neurol. Psychiatr* 1950;**63**:902–917.
9 Mackay RP. Ophthalmoplegic migraine. *Am J Ophthalmol Ser* 1929;**12**:889–95.
10 D'Astros L. L'Attaque d'Ophtalmolpleguique migraineuse. *Presse Med* 1898;**6**:40–1.
11 Vijayan N. Ophthalmoplegic migraine: ischaemic or compressive neuropathy? *Headache* 1980;**20**:300–4.
12 Duane A. Recurrent paralyses of the eye. Muscles with especial reference to Ophthalmolplegic migraine. *Arch Ophthalmol* 1923; **52**:417–35.
13 Vogelsang K. Zur Klinik der Ophthalmoplegischen migraine. Migraine. *Z Augenheilk* 1932;**77**:328–33.
14 Miller NR In: *Walsh and Hoyt's Clinical Neuroophthalmology*, Williams and Wilkins, Baltimore. Vol 2, 4th ed. 1985:569.

Accepted 6 February 1989

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