Correspondence to: Dr Kapoor, Department of Clinical Neurology, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK.

1 Tsuji I, Nakajima F, Morimoto J, Nounaka Y. The sexual function in patients with spinal cord injury. *Urol Int* 1961;**12**:270-80.
2 Brindley GS. The actions of parasympathetic and sympathetic nerves in human micturition, erection and seminal emission and their restoration in paraplegic patients by implanted electrical stimulators. *Proc R Soc Lond* 1988;**B235**:111–20.
3 Yeates WK. Ejaculation and its disorders. *Arch Ital Urol* 1990;**62**:137–48.
4 Marson L, McKenna KE. The identification of a brainstem site controlling spinal sexual reflexes in male rats. *Brain Res* 1990;**515**: 303–8.
5 Levine SB. Marital sexual dysfunction: ejaculation disturbances. *Ann Intern Med* 1976; **84**:575–9.
6 Yarkony GM. Enhancement of sexual function and fertility in spinal cord-injured males. *Am J Phys Med Rehabil* 1990;**69**: 81–7.

Abnormal evoked potentials in Miller-Fisher syndrome: further evidence of combined peripheral and central demyelination

We read with interest the short report by Ferrer *et al*[1] about a patient with Miller-Fisher syndrome in whom CNS demyelination was documented by MRI a few years later.[1]

We would like to direct attention to an additional case supporting the possibility of combined central and peripheral demyelination in this syndrome.

A 66-year-old previously healthy woman was referred for diplopia of 24 hours duration, an unsteady gait, and distal paresthesias in the upper limbs. Examination showed bilateral ptosis, complete external ophthalmoplegia, and severe axial and limb ataxia. On the second day of admission to hospital, progressive proximal muscle weakness developed accompanied by generalised areflexia. The plantar responses were flexor. All sensory modalities were intact. Routine laboratory examinations, CT of her brain, and EEG were normal. The sterile acellular cerebrospinal fluid obtained on the fifth day of admission to hospital was clear and under normal opening pressure, with a protein concentration of 0·75 g/l and no oligoconal bands.

Electrodiagnostic studies disclosed prolonged distal motor latencies, slow conduction, and prolonged F-responses in the median, ulnar, common peroneal, and posterior tibial nerves. Needle electromyography indicated active symmetrical generalised denervation. The visual evoked potentials (VEPs) were remarkable for prolongation of the p100 peaks (right 126 ms; left 128 ms; normal 100–110 ms), which retained their normal amplitude. Brain stem auditory evoked potentials (BAEPs) showed an impaired wave pattern with prolongation of N_3, N_5, and brain stem conduction time.

Within one week and with only conservative treatment, spontaneous improvement was noted in the ptosis and horizontal eye movements, but there was still partial limitation of vertical gaze.

Neurological examination after three months showed a full range of eye movements, normal muscle strength, and deep tendon reflexes. The VEPs and BAEPs had returned to normal.

The aetiology and pathogenesis of the Miller-Fisher syndrome are not well outlined, particularly the location of the pathological changes. Most workers favour a peripheral origin,[2] although others suggest brain stem inflammatory lesions[3] or a combination of central and peripheral demyelination.[4] Abnormal BAEPs have been described in only a few cases.[5] Results from MRI have usually been normal,[6] except in the reports of Giroud *et al*[7] and Ferrer *et al*.[1]

Abnormal BAEPs and VEPs, together with evidence of demyelination on brain MRI, support the hypothesis that in the Miller-Fisher syndrome there is a combination of peripheral and central demyelination. Considering the dissimilarities between central and peripheral myelin, however, this conclusion leaves the immunological mechanisms by which this process is mediated unclear.

H GOLDBERG-STERN
E MELAMED
N GADOTH
Department of Neurology, Beilinson Medical Center, Petah Tiqva 49100, Israel

Correspondence to: Dr Goldberg-Stern, Department of Neurology, Beilinson Medical Center, Petah Tiqva 49100, Israel.

1 Ferrer X, Ellie E, Larriviere M, Delepanque B, Laguery A, Julien J. Late central demyelination after Fisher's syndrome: MRI studies. *J Neurol Neurosurg Psychiatry* 1993; **56**:693–9.
2 Jamal AJ, Ballantye JP. The localization of the lesion in patients with acute ophthalmoplegia, ataxia and areflexia. (Miller-Fisher syndrome). *Brain* 1988;**111**:95–114.
3 Meienberg O. Lesion site in Fisher syndrome. *Arch Neurol* 1984;**41**:250–1.
4 Ropper AH. Proposed mechanism of ataxia in Fisher's syndrome. *Arch Neurol* 1983;**40**: 537–8.
5 Rudolph SH, Montesions C, Shanzer S. Abnormal brain stem auditory evoked potentials in Fisher syndrome. [abstract] *Neurology* 1985;**35**(suppl 1):70.
6 Ropper AH. Three patients with Fisher syndrome and normal MRI. *Neurology* 1988;**38**:1630–1.
7 Giroud M, Mousson C, Chalopin JM, Rifle G, Dumas R. Miller-Fisher syndrome and pontine abnormalities on MRI: a case report. *J Neurol* 1990;**237**:489–90.

Transient sixth-nerve palsy as the first presentation of acute leukaemia

It is not unusual for the cranial nerve to be affected early in the course of acute lymphoblastic leukaemia (ALL) and this is considered to be the result of meningeal infiltration.[1 2] Unless specifically treated, the neurological deficit is usually progressive. We report here the case of a patient with transient sixth-nerve palsy as a presentation of ALL.

A 43-year-old man was admitted with an acute onset of diplopia. He had a three month history of malaise and weight loss. He had previously had a systemic evaluation, which was normal except for a Westergren erythrocyte sedimentation rate of 40 mm/h, a transient disturbance in liver function tests (aspartate transaminase, alanine transaminase) and a constant increase in lactate dehydrogenase. Serological tests for hepatitis were negative.

On examination a complete left sixth-nerve palsy was found. The rest of the general and neurological examination was normal. A complete blood count and film, blood glucose concentration, thyroid function tests, antinuclear factor, serum immunoglobulins, immunoelectrophoresis and antibodies to acetylcholine receptor were all within normal limits. Lumbar puncture yielded a clear acellular fluid with 530 mg/l protein and 3·6 mmol/l glucose. Computed tomography of his brain gave normal results. MRI of his brain showed homogenous replacement of the skull bone marrow by an enhancing material with extension to the left cerebello-pontine angle and left cavernous sinus. A bone marrow specimen was therefore obtained and showed extensive infiltration with immature lymphoid cells, compatible with ALL.

A week after admission a gradual, spontaneous improvement in eye abduction was noted. Several days later, before any treatment (and specifically without steroid treatment), his eye movements were full in all directions, and there was complete resolution of the diplopia. The patient was treated with a standard chemotherapy protocol for adult ALL. An Ommaya reservoir was inserted and intrathecal methotrexate and cytarabine were administered with cranial radiotherapy. The patient is currently in complete remission 12 months after the diagnosis of ALL.

Cranial nerve palsies as the first presentation of lymphoproliferative disorders, although uncommon, have already been reported.[1 3] There is a single case report of transient third-nerve palsy in primary meningeal lymphoma.[4] We believe that this is the first description of a patient with a systemic lymphoproliferative disorder, namely ALL, presenting with a spontaneously resolving ocular motor deficit. The cause of the left sixth-nerve palsy in our patient was most probably leukaemic involvement of the left cavernous sinus, as was shown by MRI. The transitory nature of the neurological dysfunction may be due to transient pressure by leukaemic cells on the cranial nerve itself or on the vasa nervorum, with subsequent ischaemia. The exact mechanism of transiency remains obscure, however. To conclude, cranial nerve palsy led to the diagnosis of ALL with minimal systemic clues. Spontaneous remission of a neurological deficit should not mean that an underlying malignancy can be ruled out.

LEA AVERBUCH-HELLER
Department of Neurology, Hadassah University Hospital and Hebrew-Hadassah Medical School, Jerusalem, Israel.
SHMUEL GILLIS
Department of Haematology, Hadassah University Hospital and Hebrew-Hadassah Medical School, Jerusalem, Israel.
TAMIR BEN-HUR
Department of Neurology, Hadassah University Hospital and Hebrew-Hadassah Medical School, Jerusalem, Israel.

Correspondence to: Dr Averbuch-Heller, Department of Neurology, Hadassah University Hospital, POB 12000, IL-91120, Jerusalem, Israel.

1 Wiernik PH. Acute leukemias. In: De Vita VT, Hellman S, Rosenberg SA, eds. *Cancer. Principles and practice of oncology.* Philadelphia: Lippincot, 1989:1817.
2 Lister TA, Whitehouse JM, Beard ME, *et al.* Early central nervous system involvement in adults with acute non-myelogenous leukemia. *Br J Cancer* 1977;**35**:479–83.
3 Lossos A, Averbuch-Heller L, Reches A, Abramsky O. Complete unilateral ophthalmoplegia as the presenting manifestation of Waldenstrom's macroglobulinemia. *Neurology* 1990;**40**:1801–2.
4 Galetta SL, Sergott RC, Wells GB, *et al.* Spontaneous remission of a third-nerve palsy in meningeal lymphoma. *Ann Neurol* 1992;**32**:100–2.