cured by decompression of the Chiari malformation shown by a second myelogram. Three patients with hypercapnia, one with frank papilloedema and haemorrhages, have led to a greater awareness of this cause of headache and of the sleep apnoea syndrome which may accompany it.

Of the 36 patients with "other" cause for *blackout,* drugs were judged responsible in 23 and there were six cases of postural hypotension induced by antihypertensive therapy. Surprisingly only five patients had cardiac arrhythmia (Holter monitoring was negative in other suspects) and four had cough syncope. Nine had episodes of hypoglycaemia, either insulin-induced or reactive after abdominal surgery. Of neurological causes of blackout nine had transient ischaemic attacks, five idiopathic drop attacks and three the narcolepsy tetrad. No clue to these organic causes appeared in the referral letters. Twenty of 110 patients with an unremarkable story of simple fainting had a photosensitive response during EEG. The abnormality was limited to a polyspike-and-wave burst on photic stimulation at 10–30 Hz, not continuing beyond the time of the stimulus and usually shorter, and always suppressed by occlusion of one eye by the patient's own hand.

The yield of organic disease from the patients with *dizzy spells* was unexpectedly high. Among the 33 whose dizzy spell was due to central vertigo was one patient with cerebral tumour, nine with an episode of demyelination and the rest had either vertebro-basilar disease or minor epilepsy. Benign paroxysmal positional nystagmus and vestibular neuronitis were poorly recognised but relatively common disorders. Curiosities were two cases of Lermoyez' syndrome[2 3] (allergy, deafness and tinnitus culminating in a vertiginous bout from which the patient emerged with restored hearing) and one with the disputed syndrome known as Tumarkin's otolithic crisis:[4] a farmer with a long history of active middle-ear infection was looking over the rails at his cattle when he experienced "crynnu"—a goose walking over his grave—then found himself on the ground.

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References

[1] Fitzpatrick R, Hopkins A. Referrals to neurologists for headaches not due to structural disease. *J Neurology Neurosurg Psychiatry* 1981;**44**:1061–7.
[2] Lermoyez M. Le vertige qui fait entendre (Angiospasme labyrinthique). *Presse médicale* 1919;**27**:1–3.
[3] Eagle WW. Lermoyez's syndrome—an allergic disease. *Ann Otol Rhinol Laryngol* 1948;**57**:453–64.
[4] Turmarkin A. The otolithic catastrophe: a new syndrome. *Br Med J* 1936;**2**:175–7.

Erythrocyte deformability in multiple sclerosis

Sir: Brunetti *et al*[1] have recently reported an elevation in whole blood viscosity in 36 patients with multiple sclerosis when compared with unmatched normal controls. In the absence of a significant difference in plasma viscosity or haematocrit between the two groups the authors attributed the rise to a reduction in erythrocyte deformability although this property was not examined. Ernst[2] however, has pointed out that the relative viscosity does not always correlate with red cell deformability which moreover may have been affected by the steroid therapy the patients had recently received.

We have measured erythrocyte deformability in multiple sclerosis by microfiltration using a modification[3] of the method of Reid *et al.*[4] Samples from 15 patients (11 female and 4 male) with established disease in remission were compared with healthy age and sex matched controls. Subjects with a raised ESR, fasting blood sugar or fasting lipids were excluded as were those patients receiving steroids or other medication thought to influence red cell deformability.

No significant difference in the mean red cell deformability was detected between the patient and control groups, in either native plasma or saline (table).

Table *Mean deformability index*

	Plasma	*Saline*	*HCT*
Multiple sclerosis	0·54 ± 0·07	0·72 ± 0·09	40·7 ± 4·1
Controls	0·48 ± 0·06	0·71 ± 0·10	43·3 ± 2·6
Student's paired *t* test	NS	NS	NS

There are several possibilities that may explain our failure to confirm Brunetti's conclusion. The administration of steroids may have influenced the red cell deformability as suggested by Ernst. Alternatively red cell deformability may be reduced in this condition but only during relapse. Finally the existence of a raised whole blood viscosity may not necessarily imply the presence of reduced red cell deformability.

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References

[1] Brunetti A, Ricchieri S, Patrissi G, Girolami A, Tavolato B. Rheological and fibrinolytic findings in multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1981;**44**:340–3.
[2] Ernst E. Letter. *J Neurol Neurosurg Psychiatry* 1982;**45**:185.
[3] Pollock S, Harrison MJG. Red cell deformability is not an independent risk factor in stroke. *J Neurol Neurosurg Psychiatry* 1982; **45**:369–71.
[4] Reid H, Barnes A, Lock P, Dormandy J, Dormandy T. A simple method for measuring erythrocyte deformability. *J Clin Pathol* 1976;**29**:855–8.

Notice

The IXth International Congress of Neuropathology will be held in Vienna, 5–10 September, 1982. Address for enquiries: Dr H Lassmann, c/o Vienna Medical Academy, Alser Str 4, A–1090 Vienna, Austria.

A one day symposium entitled "The Guillain-Barré syndrome: recent advances" will be held at the historic Abbaye de Royaumont (near Paris) France on 20 September, 1982. For details and registration, contact Prof Gerard Said, Service de Neurologie, Centre Hospitalier de Bicêtre, 94270 Le Kremlin-Bicêtre, France.