Matters arising

High dose intravenous methyl prednisolone in acute exacerbations of multiple sclerosis

Sir: Like Dowling, Bosch and Coole,[1] Buckley, Kernard and Swash,[2] we use high dose intravenous methyl prednisone in acute exacerbations of multiple sclerosis.

Thirteen patients (10 women and three men) aged 15–41 years were treated by intravenous methyl prednisolone, 1 g daily for 5 days. As we were afraid of a relapse at the beginning, two patients received ACTH afterwards. Three patients received Imuran before the exacerbation and this drug was continued later. Ten patients improved very quickly, often during the perfusions and to a great extent. For example, a bedridden patient was able to walk, another patient's vision improved from 2/10 to 10/10 by the end of the treatment. In the three other cases, improvement was slower and less marked but no failures occurred. Nine of the patients had already been treated by ACTH for previous exacerbations. In all these cases, methyl prednisone was more effective: improvement was quicker and more marked.

No complications due to the treatment were noted, except a little asthenia. We did not observe any relapses immediately or during the first month. Only one patient had another exacerbation (vertigo) 45 days after the perfusion, in a new area: neither hospitalisation nor corticoids were required. We do not think that corticoid treatment is required after the perfusions.

So, our results were very good and rapid in all cases of acute exacerbations of multiple sclerosis. This allows a very much shorter hospitalisation period (usually a week for a known case); this is very useful for both the patients and society in general. Effectiveness does not seem to depend on the age of the patient or that of the disease. Our very favourable assessment of high dose methyl predisolone for a short time in acute exacerbations of multiple sclerosis needs to be confirmed by other studies on larger groups. A longer follow up period is required in order to judge the effectiveness and the absence of secondary complications of this treatment more exactly.

JY GOAS
JL MARION
A MISSOUM
Neurology Department,
University Hospital (Gueôde),
55 Rue Jules Gueôde,
29279 Brest Cedex,
France

References

[1] Dowling PC, Bosch VV, Cook SD. Possible beneficial effect of high dose intravenous steroid therapy in acute demyelinating disease and transverse myelitis. *Neurology (Minneap)* 1980;**30**:33–6.

[2] Buckley C, Kennard C, Snash M. Treatment of acute exacerbations of multiple sclerosis with intravenous methyl prednisolone. *J Neurol Neurosurg Psychiatry* 1982;**45**:179–80.

Stevens replies:

We are grateful for Dr Flor-Henry's careful analysis of our findings in the two studies of telemetered EEGs of schizophrenic patients. Although nothing would have given more satisfaction than to find some consistent specific localisable derangement of cerebral electrical activity during abnormal behaviours of schizophrenia, our interpretation of the data did not allow us to specify which was the abnormal hemisphere for the few statistically significant changes emerging from our extensive studies of scalp EEG in schizophrenic patients.

In the first study, (Stevens *et al*, 1979) patients showed a decrease in left temporal power but an increase in right temporal slow activity during hallucinations. Was this evidence of faulty activation of the speech area on the left or abnormality from the homologous area on the right? True, schizophrenic patients in contrast to normals demonstrated inappropriate activation of the left hemisphere during spatial tasks—but was this due to failure of activation of an abnormal right hemisphere? True again, anecdotal information illustrated from five patients in the first study showed predominance of left temporal abnormality but in our second study (Stevens and Livermore, 1982) in which we attempt quantitative analysis of power spectra of the data of many of the same patients plus additional subjects, the complex mix of findings appears to implicate both hemispheres in the unusual information processing suggested by the EEG data. Thus, as Flor-Henry notes, the catatonic schizophrenics had more right temporal slow activity and the paranoids more left; alpha-suppression in the left temporal lobe during abnormal behavioural or subjective events could as well represent inappropriate failure to suppress on the right, as was also seen in the failure of schizophrenic patients to suppress right temporal alpha during performance of spatial tasks.

Finally, carbamazepine, and also sodium valproate have both been shown to have important therapeutic and preventive effects in mania.[1–3] We found that these anticonvulsants agents are not useful in and may even exacerbate schizophrenia, one of the few pharmacologic distinctions between these disorders, a clue worth pursuing in the investigation of underlying etiologies of the disease and mechanisms of drug actions.

JANICE R STEVENS

References

[1] Ballenger J, Post R. Therapeutic effects of carbamazepine in affective illness: a preliminary report. *Commun Psychopharmacol* 1978;**2**:159–75.

[2] Emrich HM, Zerseen DV, Kissling W, Moller H-J, Windorfer A. Effect of sodium valproate on mania: the GABA-hypothesis of affective disorders. *Arch Psychiat Nervenkr* 1980;**229**:1–16.

[3] Okuma T, Kishimoto A, Inoue K, *et al.* Antimanic and prophylactic effects of carbamazepine (Tegretol) on manic depressive psychosis: a preliminary report. *Folia Psychiat Neurol Jpn* 1973;**27(4)**:283–97.

Subacute sensory neuropathy with remission: an association with lymphoma

Sir: Sagar and Read[3] have reported a case of sensory neuropathy (as described by Denny-Brown) during Hodgkin's disease which progressively disappeared while the lymphoma were being successfully treated. Given the usually prolonged severity of such a neuropathy, they suggested that the underlying tumour was most likely related to its prognosis. In his thesis,[2] one of us had recounted two similar cases.

A 48-yr-old woman in whom Hodgkin's disease (stage 2 type 2) was diagnosed in January 1977, developed neurological symptoms before treatment which consisted of violent and painful paraesthesiae in the limbs and such difficulty in standing and walking that the patient was soon bedridden. Tendon jerks had completely disappeared; proprioceptive sensibility in the limbs was greatly reduced and there was thermal hypoaesthesia in a "glove and stocking" distribution; there was slight distal weakness; CSF was normal; electrophysiological examination showed partial denervation but conduction velocity was normal. She was treated with CCNU, vincristine, and procarbazine from February to October 1977. From the first month there was an improvement and walking became possible. Despite the persistance of serious distal paraesthesiae and impaired

High dose intravenous methyl prednisolone in acute exacerbations of multiple sclerosis.

J Y Goas, J L Marion and A Missoum

*J Neurol Neurosurg Psychiatry*1983 46: 99
doi: 10.1136/jnnp.46.1.99

Notes