Matters arising

Sir: Navarro and colleagues1 report a patient with bilateral Parkinsonism secondary to a corpus callosal glioma. Their patient had signs of long-tract disease and other hemisphere dysfunction to point to the need for further investigation. We report the case of a 74 year old lady who developed bilateral Parkinsonism secondary to an intrinsic cerebral tumour without such clues. She had a long history of anxiety and depression and for 2 years had complained of vague headaches. During this time she also complained of dizziness for which prochlorperazine was prescribed. Within 4 days of starting this she suffered an unwitnessed episode of loss of consciousness which was attributed to the medication, which was promptly withdrawn. She had no more episodes of loss of consciousness. For 6 months she had noted slowness of movement, unsteadiness while walking and a resting tremor affecting both hands. Examination (by three examiners) showed bilateral impulsive facies, a resting tremor (4–6 Hz) affecting both hands but worse on the right and cogwheel rigidity in all limbs but worse in the arms. There was no right arm swing during walking and she had difficulty turning around corners. There were no clear symptoms or signs of cortical dysfunction. No signs referable to the long tracts were found and there were no signs of raised intracranial pressure.

The 4 days of treatment with prochlorperazine (8 months previously) were not thought to be responsible for the Parkinsonism.

Fig  CT Scan following injection of intravenous contrast medium shows an enhancing space occupying mass in the right hemisphere with contra-lateral hydrocephalus.
Matters arising

She was thought to have Parkinson's disease but when she became rapidly unconscious (3 days after the above examination) CT scan (fig) revealed a large left hemisphere intrinsic tumour with contra-lateral hydrocephalus. The patient continued her rapid deterioration and died. Unfortunately a post-mortem examination was not obtained.

Our patient's Parkinsonian features were more prominent in her arms, suggesting that hydrocephalus was not the mechanism responsible for the Parkinsonism and that direct infiltration of tumour into the contra-lateral basal ganglia may have contributed. This case illustrates that unlike Navarro et al's patient, tell-tale symptoms or signs of cerebral tumour are not always present in a patient with Parkinsonism secondary to intrinsic brain tumour.

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Reference

Double-blind controlled trial of azathioprine in the treatment of multiple sclerosis

Sir: In 1983 the Medical Research Council Working Party on Trials of Immunological Treatment in Multiple Sclerosis concluded that existing trials did not determine whether azathioprine is effective in the treatment of multiple sclerosis. Consequently we embarked on a double-blind controlled multicentre trial of treatment with azathioprine lasting for three years. Between April 1983 and October 1984, 354 patients were randomly allocated to azathioprine or placebo treatment groups. The results are being reviewed periodically by an impartial audit committee because if an overwhelmingly significant effect were observed during the course of the trial we would have an ethical obligation to stop. The major outcome criteria which the drug would have to fulfill to establish its effectiveness would be differences between the two groups in change in Kurtzke disability status score or ambulation index. We would also be interested in changes in individual items of the Kurtzke functional scale score, particularly visual function which is almost entirely independent of the ambulation index and only a minor component (compared with pyramidal function) of the Kurtzke disability status scale.

The large number of patients in the trial and the small standard deviation of change in Kurtzke disability status after two years (n = 230, mean = 0.29, standard deviation = 0.73) will permit some analysis of subgroups. There are many possible hypotheses which could be tested, many of which will be significant by chance alone. Consequently before data entry is complete and any subgroup analyses are performed we wish to publish the hypotheses which we will be testing. At entry patients were classified according to set criteria as:

1. clinically definite having at least two episodes and two clinical lesions or two episodes and one clinical and one subclinical lesion,
2. laboratory supported definite having at least two episodes, one clinical lesion and oligoclonal bands or IgG increase in the CSF and
3. currently progressive having two necessarily separate lesions (of which one might be subclinical) and oligoclonal bands or IgG increase in the CSF.

Groups 1 and 2 will be analysed together and group 3 separately. The subgroup of group 3 who have had a progressive course from onset will be analysed separately from those who developed a progressive course after initial relapses and remissions. Patients treated early in their disease (within 2 or 5 years of onset) will be analysed separately from those who treated later. Patients with relatively mild disease at entry into the trial (Kurtzke disability status scale score less than 5) will be analysed separately from those with more severe disease (scores 5–6). Of particular interest will be whether azathioprine is effective in those patients who have a malignant disease course in whom scores of 5–6 have been achieved within five years from onset. Other aspects which merit investigation are possible differences in responsiveness between males and females and younger and older patients. We will also evaluate whether the effectiveness of treatment is related to HLA status, in particular to the presence or absence of DR2 antigen. The frequency of relapses will also be compared but we consider this analysis less important. We intend to make the results available as soon as possible after collection of the last data in October 1987.

The British and Dutch Multiple Sclerosis Azathioprine Trial Group

The participating centres are the neurology departments in the following hospitals: Aberdeen Royal Infirmary; Queen Elizabeth Hospital, Birmingham; Newmarket General Hospital; University Hospital of Wales; Walsgrave Hospital, Coventry; Dundee Royal Infirmary; Royal East Sussex Hospital, Hastings; Ipswich Hospital; Walton Hospital, Liverpool; Pembury Hospital, Pembury; West Hill Hospital, Dartford; The Radcliffe Infirmary, Oxford; Southampton General Hospital; Pinderfields General Hospital, Wakefield; Addenbrooke's Hospital, Cambridge; Brook General Hospital, Guy's Hospital, National Hospital for Nervous Diseases, London; Manchester Royal Infirmary; Royal Victoria Infirmary, Newcastle upon Tyne, Norfolk & Norwich Hospital; Academisch Ziekenhuis, Groningen, The Netherlands.

A list of participants can be obtained from the British and Dutch Multiple Sclerosis Azathioprine Trial Office, Department of Neurology, Guy's Hospital, London SE1 9RT, UK.

References

Computed tomographic findings of brain and skull in myotonic dystrophy

Sir: We read with interest the paper by Avrahami et al. about computed tomographic findings in myotonic dystrophy. Brain atrophy has been described in these patients; however, cerebellar atrophy associated to this disorder has not yet been described.

We have recently seen a patient aged 54 yr with a family history of myotonic dystrophy

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74 year old lady who developed bilateral parkinsonism secondary to an intrinsic cerebral tumour.
H S Pall and A Williams

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