studied by Plioplys *et al* were found in Western blots of frontal cortex and, therefore, should not be confused with the Purkinje cell specific reactivity of the antibody found in patients with paraneoplastic cerebellar degeneration.

Other examples of autoantibodies specific for neurological paraneoplastic syndromes include: (1) an antineuronal antinuclear antibody which has been found in sera from patients with small cell lung cancer and neurological paraneoplastic diseases but not in any of more than 400 controls⁴ and (2) an autoantibody that reacts with retinal ganglion cells which only have been found in patients with small cell lung cancer and retinal degeneration.^{6,7}

Finally, Plioplys *et al* used total homogenates of different parts of the brain in their Western blots which, in our experience, have yielded an especially high incidence of non-specific banding.² Non-specific immunoreactivity can be significantly reduced by using isolated neurons.²

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Plioplys replies:

Drs Anderson and Posner have pointed out further references that anti-neuronal antibodies may play a pathogenic role in the production of neurological diseases. Some of their own results were in fact mentioned in the text of our article, particularly the 34/38K and 62/64K bands associated with paraneoplastic cerebellar degeneration.

The purpose of our study was to investigate the presence of circulating anti-central nervous system (CNS) antibody activity in neurologic diseases. It should be noted that Drs Anderson and Posner specifically and exclusively refer to "anti-neuronal" antibodies. We used Western blots made from whole human brain because we wanted to investigate the potential presence of antibodies recognising antigenic epitopes in neurons and in other CNS cells, such as glia. There is no a priori reason why neurological autoimmune processes should be limited to neurons. In a population study there are definite advantages in investigating not only anti-neuronal activity specifically, but anti-CNS activity more generally. It should be noted that in our article the expression "anti-CNS" was used exclusively, and the term "anti-neuronal" was never used. "Neurons" and "CNS" are not synonyms.

As the results of our study indicated, the incidence of background banding was not high—an overall incidence of 30% for the entire studied population. Of those that had banding, 65% had only one band. Thus, when investigating anti-CNS antibody activity, whole brain Western blots indeed may be very useful, and in our hands do not have excessive background staining.

Currently I am completing an extensive survey of anti-CNS (not anti-neuronal) antibody activity in childhood neurological diseases with a total studied cohort of over 400 children. Some of the results have been submitted in abstract form for presentation and the completed manuscripts will be ready soon. In these studies, as in the previous one, Western blots of whole brain were used, in addition to acetone-fixed frozen tissue sections. Unlike the published results with the adult study, in this investigation there were quite significant positive correlations observed for a number of neurological and rheumatic diseases.

Since these results will be published in abstract form soon, and shortly will be submitted in manuscript form for publication, do not think that it would be appropriate to discuss details of the results here. However one result can be mentioned (it has been presented in part in abstract form, *Can J Neurol Sci* 1986;13:167). That concerns the opsoclonus-myoclonus syndrome of Kinsbourne. In investigating the sera from four children with definite and two with probable Kinsbourne's syndrome, anti-cerebellar reactivity was detected in three of them. In one child during the acute phase of illness there was an anti-cerebellar specific antigen identified with a molecular weight of 62K. Convalescent sera obtained two years later revealed no immunoreactivity. In another child, during the acute phase there was a 43K band detected which was not cerebellar specific. In a third child, during convalescence there were two bands identified which were cerebellar specific, 27K and 35K.

On the basis of cerebellar specificity on Western blots, and on the basis of the identified molecular weights, these results are highly significant ones. In the two cases that displayed specific anti-cerebellar reactivity the identified antigen molecular weights corresponded to Anderson and Posner's described 32/34K and 62/64K anti-cerebellar reactivity in paraneoplastic cerebellar degeneration. It should be noted that in our extensive childhood population study on cerebellar Western blots, the incidence of banding in the 32/34K range is 0.5% and in the 62/64K range was completely absent. These results give credence to two hypotheses concerning Kinsbourne's disease: (1) this disease may be on the basis of an antibody-mediated autoimmune disease process; (2) the primary CNS site that is affected may be the cerebellum.

The cited Kinsbourne's syndrome results, along with the results that will be forthcoming from this laboratory, give convincing evidence that investigations of anti-CNS immunoreactivity using whole brain homogenates in preparing Western blots is a very useful technique for investigating neurologic disease processes.

Positron emission tomography in cases of chorea with different underlying diseases

Sir: We read with interest the study of Hosokawa *et al*¹ who reported abnormalities of striatal local cerebral metabolic rate for glucose (LCMRglc) in five patients with chorea

of various aetiologies. The hypometabolism of the striatum, visually appreciated in four patients and present on quantitative analysis in the fifth, was used to support their conclusion that dysfunction of the striatum is the common pathophysiological disturbance in chorea of diverse causes. By inference, one is left to assume that all patients with chorea would be expected to show hypometabolism of the striatum on 18F fluorodeoxyglucose (FDG) positron emission tomography (PET).

We have recently reported our experience with FDG PET scanning in four patients with chorea secondary to systemic lupus erythematosus (SLE).² In contrast to the findings of Hosokawa *et al.*,¹ we found no reduction in striatal LCMRglc; indeed there was a slight increase in the ratio of striatal to cortical glucose metabolism.

The pathophysiology of chorea in SLE remains uncertain. Pathological study in a small number of SLE chorea patients has not revealed a consistent distribution of potential causative lesions and the striatum has not been invariably involved in these cases. This does not exclude the possibility that primary or secondary striatal dysfunction is the cause of SLE chorea. Nor does normal LCMRglc exclude a disturbance in function of striatal neurons. However, our results do indicate that striatal FDG hypometabolism is not the PET correlate of chorea.

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- 1 Hosokawa S, Ichiya Y, Kuwabara Y, *et al.* Positron emission tomography in cases of chorea with different underlying diseases. *J Neurol Neurosurg Psychiatry* 1987;50:1284-7.
- 2 Guttman M, Lang AE, Garnett ES, *et al.* Regional cerebral glucose metabolism in SLE chorea: further evidence that striatal hypometabolism is not a correlate of chorea. *Movement Disorders* 1987;2:201-10.

Hosokawa replies:

We have reported the common hypometabolism of the striatum in five patients with chorea due to different underlying

diseases: Huntington's disease, choreoacanthocytosis, sporadic progressive chorea and dementia, pseudo-Huntington form of dentato-rubro-pallido-luysian atrophy (DRPLA) and hemichorea.¹ The findings were obvious both by visual inspection of PET images and by quantitative analysis of LCMRglc values. Therefore, we concluded that dysfunction of the striatum is relevant to the genesis of chorea in these patients. We do not intend to exclude the possibility of different pathophysiological mechanisms of chorea with other underlying diseases such as systemic lupus erythematosus (SLE), as pointed out by Drs Lang and Garnett. However, they should be cautious about the interpretation of their results that striatal hypometabolism is not the PET correlate of SLE chorea, since they mainly analysed striatum/cortex ratios in their patients instead of analysing absolute values of LCMRglc.² We have occasionally observed cortical hypometabolism in patients of SLE with central nervous system involvement (unpublished data); therefore, the values in the cortex are not appropriate as a reference.

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- 2 Guttman M, Lang AE, Garnett ES, *et al.* Regional cerebral glucose metabolism in SLE chorea: further evidence that striatal hypometabolism is not a correlate of chorea. *Movement Disorders* 1987;2:201-10.

Relief of common migraine by exercise

Sir: In a recent letter, van Gijn¹ proposed a remarkable self-treatment for migraine attacks, viz. performing strenuous exercise. The advice was based on an anecdotal observation in a patient who suffered from headache attacks, which most migrainologists probably would not have diagnosed as migrainous. Although suggestive, attacks of "dull, boring headache in the left temple," lasting for one to three days, not accompanied by severe nausea and/or vomiting, phonophobia or photophobia or prodromal symptoms, and of such "severity" as to still allow the patient to run a few miles, certainly do not suffice to make a diagnosis of migraine.² A family history of "similar headaches" also fails to support the diagnosis. In conclusion, exercise therapy may have

relieved this patient's headaches, but to extrapolate this observation to migraine, in my opinion, would be quite premature.

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- 2 Blau JN. Towards a definition of migraine. *Lancet* 1984;i:444-5.

Van Gijn replies:

I thank Dr Ferrari for his interest in my clinical note. He is correct in suggesting that patients with migraine who are too ill to do anything should not be advised to go for a run. It was my purpose to draw attention to the benefits of exercise in a type of vascular headache clearly related to migraine, not to discuss the degree of relationship. By the way, what is a migrainologist?

Asymmetry of pathology in Alzheimer disease

Sir: The data in the letter from Wilcock and Esiri,¹ although the size of the sample was small, demonstrates the differences found between specimens taken from the left and right hand sides of the brain in Alzheimer's disease.

Differences between the left and right side have earlier been shown in respect of the neurons in the H1 Sommer sector of the normal human hippocampus.² The letter from Wilcock and Esiri caused us to re-examine data from a series of 56 subjects aged 59 to 103 years, who had not been assessed cognitively in life and whose brains had been collected for a biochemical investigation of the temporal lobes. The temporal lobe from each side was removed and weighed and the medial temporal region was then dissected and weighed. There was no significant difference between the weight of the left and the right (paired *t* test. 8-82, SD 1-4: 8-75, SD 1-52: *t* = 0-34, df 53, *p* = 0-73). Sections were taken from the anterior hippocampus posterior to the pes and stained with haematoxylin and eosin, Nissl, Periodic Acid