Matters arising

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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References

Hosokawa replies:

We have reported the common hypometabolism of the striatum in five patients with chorea due to different underlying diseases: Huntington’s disease, choreoan- tochytosis, 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 with SLE with central nervous system involvement (unpublished data); therefore, the values in the cortex are not appropriate as a reference.

References

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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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’s disease

Sir: The data in the letter from Wilcock and Esiri,4 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

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References