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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- 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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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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References

- 1 Gijn J van. Relief of common migraine by exercise. *J Neurol Neurosurg Psychiatry* 1987;50:1700-1.
- 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

Schiff and Glee. The sections were examined and the number of plaques, tangles and cells showing granulovacuolar degeneration (GVD) in the hippocampus was rated on a four point scale; none, some, moderate and many.

The results are shown in the table and show evidence indicating differences between the ratings for the left and right hippocampus. Chi-square tests show the differences between left and right to be highly significant for plaques, tangles and GVD ($p < 0.0004$ for all three measures). There was no consistent difference between counts on one side compared with the other.

Table

	% Agreement	% Disagreement
Plaques	65.45	34.55
Tangles	61.82	38.18
GVD	69.09	30.91

Our results derived from a larger sample of aged subjects but assessed with a less rigorous method of quantification are similar to the results reported by Wilcock and Esiri for Alzheimer cases. They emphasise the need for caution in making correlations between pathological and biochemical measurements obtained from the two hemispheres. The pathological and biochemical data in adjacent areas need to be established before adjacent sampling techniques are adopted for pathological and biochemical correlations in controls and demented of the Alzheimer type.

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- 2 Miller AKH, Alston RL, Mountjoy CQ, Corsellis JAN. Automated differential cell counting on a sector of the normal human hippocampus: the influence of age. *Neuropathol Appl Neurobiol* 1984;**10**:123-41.

Book reviews

Cranial and Spinal Magnetic Resonance Imaging: An Atlas and Guide. By David L Daniels and Victor M Houghton. (Pp 350; \$150.00.) New York: Raven Press, 1987.

The accurate interpretation of pathology in magnetic resonance imaging requires both an in depth knowledge of normal anatomy and also an appreciation of the appearance of normal and abnormal tissue on different sequences. So an atlas dealing with this subject should play an essential part in the background reading of everyone likely to come into contact with MR imaging.

Cranial and Spinal Magnetic Resonance Imaging: An Atlas and Guide sets out to fulfil this requirement. It is divided into 11 chapters. The first is devoted to a summary of the physical principles of MRI and assumes that the reader is already familiar with the subject. To those who are experienced the information provides a useful summary of the practical aspects of image interpretation. However, those commencing the subject will find the information rather too limited to be really useful. Chapter 2 deals with the appearance of most of the common pathological conditions providing guidelines to the interpretation of the MR images. Chapters 3 to 11 fulfil the essential purpose of the book. Each commences with a short description of the anatomy followed by selected cryomicrotome sections and the equivalent MR scan slices in the three orthogonal planes. Most chapters end with further examples of pathology. Chapters 4 and 5 deal with the deep cerebral structures and limbic system in particular detail and those who have been fortunate enough to hear Professor Naidich lecture on this subject will recognise his contribution in them. Chapters 6, 7 and 8 deal with the pituitary region, posterior fossa and petrous anatomy respectively and the last 3 chapters are devoted to the spine.

The imaging has been undertaken on a General Electric Signa system operating at 1.5 Tesla and as would be expected the anatomical detail is good. The reproduction and annotation of the scans and cryomicrotome slices, which are in colour, is excellent. Most images have been undertaken using T1 and T2 weighted spin echoes with a slice thickness of 3 or 5 mm but reference is also made to fast imaging with gradient echoes in the cervical spine.

As is to be expected with a multiauthor book there is some unnecessary repetition in the text but this has been kept to a minimum. The book is well produced and contains a wealth of detail. Nevertheless it is not cheap and at \$150 will not easily find its way into individual ownership. However, it should be available in libraries as a reference to all those, surgeons, physicians and radiologists alike, who have anything to do with the interpretation of MRI scans and anyone who is prepared to spend this sort of money will find it a very rewarding book.

DPE KINGSLEY

Intracranial Tumors in Infancy and Childhood: Basic Research Diagnosis and Treatment. (Progress in Experimental Tumor Research Vol 30.) Edited by N Kageyama, K Nagoya, K Takakura, F Epstein, H Hoffman, L Schut (Pp 378; £135.00.) Basel: Karger, 1987.

The International Publishers, Karger are skilled in the alchemy of turning symposia into books. Paediatric neurosurgeons already know this from *Concepts in Paediatric Neurosurgery* that records the annual meetings of the American Society for Paediatric Neurosurgeons. That series is already up to seven but this present volume is number 30 in Karger's series, *Progress in Experimental Tumor Research*. It is a record of the International Symposium on Paediatric Oncology (ISPN) that was held in Toba, Japan, in November 1985. Neurooncology in this context is confined to those tumours whose primary diagnosis and management is most likely to come within the province of the neurosurgeon. Those interested in neuroblastoma or the neurological complications of lymphoma and leukaemia (not to mention the problems of spinal cord compression from extra-dural malignant disease) will be disappointed.

The volume opens with some chapters on basic research in neurooncology before passing on to individual tumours and their management. Much of the material presented overlaps with that already published in the *Concepts of Paediatric Neurosurgery* series, but this is perhaps not surprising because the Toba conference contributors are derived almost exclusively from the United States and Japan. Again, it is not surprising that the Japanese provide most of the articles on the management of pineal tumours and details of their extensive experience is always interesting to read.