Table Serum antibodies to cysticercus and mycobacterial antigens

Group	Cysticercus antibodies		Mycobacterial antibodies	
	OD 492 nm (mean, 2 SD)	Positive/total	OD 492 nm (mean, 2 SD)	Positive/total
Control	0.01, 0.02	0/36	0.16, 0.12	3/56
Systemic active Tb	0.01, 0.03	0/26	1.3, 0.4	31/31
CNS Tb	0.03, 0.09	1/29	0.76, 0.5	13/18
Neurocysticercosis	0.67, 0.5	19/22	0.12, 0.09	0/32
Epilepsy	0.04, 0.11	2/39	0.18, 0.2	1/38
Disappearing CT lesions	0.46, 0.4	12/38	0.09, 0.1	2/38

Antimycobacterial and anticysticercus antibodies in sera were measured by ELISA as described earlier. The mean + 2 SD of control OD values in each case were taken as cut-off values to determine positivity rates.

culomas and treated these patients with antituberculous drugs. After this report, in India, most patients who presented with epilepsy and an enhancing ring or disc lesion have been treated for tuberculosis. Ahuja and Mohanta⁸ in a prospective study of late onset epilepsy investigated 253 patients all of whom had CT. There were 23 patients with a space occupying lesion shown by CT. The lesion disappeared in 11 patients with antituberculous treatment. The authors concluded that these were tuberculomas. Goulatia *et al*¹ presented evidence that most of these lesions disappeared when treated with anticonvulsant drugs alone, thus casting doubt on the tuberculoma hypothesis. However, the nature of the underlying lesion remained a matter of speculation. Sethi *et al*² speculated that it could be focal encephalitis of obscure origin.

Since cysticercus is well known to produce epilepsy and a similar CT morphology in cysticercosis has been described, we investigated our patients for evidence of cysticercosis. Approximately one third of our patients showed evidence of antibodies against cysticercus in serum. Ghosh *et al*³ (reported at the 38th Annual Conference of the Neurological Society of India, 1988) have performed stereotaxic biopsy in 14 such patients. Biopsy revealed evidence of parasitic granuloma in 10 patients of which six were definite cysticercus. None of the patients showed evidence of tuberculosis. It is our contention that the CT lesions could be produced by more than one underlying pathology and cysticercosis is an important cause. Our current hypothesis is that these patients have a "micro" lesion producing an epileptogenic focus and since the lesion is small it is not detected on CT scan in the interictal period. During a seizure there is a breakdown of the blood brain barrier (BBB) around the lesion which produces the enhancing lesion. As the BBB is repaired, the lesion regresses. We have seen five patients in

whom the lesion disappeared and reappeared at the same or different site after a flurry of seizures, to disappear again when the seizures were controlled. This observation would support the concept of breakdown in BBB. Though tuberculomas can have similar image morphology on the CT scan, it seems that it is not the cause of "disappearing lesions" in any significant number of patients.

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References

- 1 Goulatia RK, Verma A, Mishra NK, Ahuja GK. Disappearing CT lesion in epilepsy. *Epilepsia* 1987;28:523-7.
- 2 Sethi PK, Kumar BR, Madan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. *J Neurol Neurosurg Psychiatry* 1985;48:866-9.
- 3 Jaykumar PN, Taly AB, Mohan PK. Transient computerised tomographic (CT) abnormalities following partial seizures. *Acta Neurol Scand* 1985;72:26-9.
- 4 Bhatia S, Tandon PN. Solitary 'Microlesions' in CT: A clinical study and follow up. *Neurology (India)* 1988;36:139-49.
- 5 Tandon PN, Bhargava S. CNS tuberculosis: lesions learnt from CT studies. *Neurology (India)* 1980;28:207-12.
- 6 Chawla TC, Atreyi M, Jailkhani BL. Immunohistochemical characterization of saline extracted antigens of mycobacterium tuberculosis H 37

Ra, Int. Arch. Allerg Appl Immunol 1986; 80:221-3.

7 Diwan AA, Coker-Vaum M, Brown P, *et al*. Enzyme linked immunosorbent assay (ELISA) for the detection of antibodies to cysticerci of *Tania Solium*. *Am J Trop Med Hyg* 1982;78:492-6.

8 Ahuja GK, Mohanta A. Late onset epilepsy. A prospective study. *Acta Neurol Scand* 1982; 66:216-26.

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Exercise induced diplopia as a presentation of midline cerebral tumour

Sir: We report a patient who presented with exercise induced diplopia caused by a midline cerebral tumour. To our knowledge this presentation has not been previously reported for any neurological disease, including intracerebral malignancy.

A 22 year old white, male, telephone engineer presented to his general practitioner in April 1986 with a 10 day history of exercise induced diplopia. These symptoms had occurred on three occasions and lasted between 15 and 90 minutes. Twice diplopia was noticed while playing football and once while undergoing physical training. The symptoms subsided 5 minutes after cessation of exercise. he reported that the images were displaced vertically and were more divergent on upward gaze. When not exercising he had no diplopia, headache or other symptoms related to the nervous system. Examination in April 1986 revealed no ophthalmological or neurological abnormality. He was normotensive.

Between June and August 1986 he was investigated and during this period his episodes of diplopia became more frequent and occurred with lesser degrees of activity. He was observed undergoing vigorous exercise, when no abnormalities of extra-ocular muscle function were noted but the symptoms of diplopia were reproduced. CT of the head with contrast merely revealed some possible enlargement of the ventricular system which was not considered to be definitely abnormal. Lumbar puncture revealed a pressure of 18 cm of cerebrospinal fluid with normal protein levels, immunoglobulin electrophoresis and cytology. Following these investigations no definite conclusions were reached but follow-up with a repeat of CT was arranged.

By December 1986 he had deteriorated and complained of diplopia occurring much of the time, a worsening of memory, headache, inco-ordination and drowsiness. On re-

admission he was noted to have poor memory, fine horizontal nystagmus, bilateral restriction of upward gaze and an inability to walk heel to toe. Fundoscopy was normal as was the rest of his neurological and general examination. A repeat of CT revealed a midline mass lesion obliterating most of the third ventricle and growing into the left lateral ventricle. The lateral ventricles were more prominent than on the previous scan. Histological examination of a small fragment of the tumour following burr-hole biopsy revealed the features of an astrocytoma. The fragment was considered too small for grading.

The patient initially responded to dexamethasone and local radiotherapy and in August 1987 a further CT head scan again revealed the midline mass lesion but indicated some shrinkage. Following this, his condition gradually deteriorated and he died in November 1988.

There seems little doubt that this man's exercise induced diplopia resulted from his midline astrocytoma. At presentation this tumour was presumably producing a small elevation of the cerebrospinal fluid pressure as demonstrated by the possible enlargement of the ventricular system seen on CT scan, and the pressure recorded at lumbar puncture. As the illness progressed, raised intracranial pressure became more pronounced. It seems likely that the onset of diplopia during exercise was due to a further elevation of intracranial pressure produced by exertion.

Exercise induced diplopia is not a well recognised presenting feature of midline cerebral tumours although disturbances of gaze are not unusual.¹ A "Medline" computer search back to 1966 using "exercise," "sports" and "diplopia" as key words revealed no publications on the association.

The case does provide circumstantial evidence that, in humans, exercise tends to elevate intracranial pressure. It is possible, however, that this pressure elevation only occurs when pathological abnormalities exist.

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- Walton J. *Brain's Diseases of the Nervous System*. Oxford: Oxford University Press, 1985.

Effect of tropatepine, an anticholinergic drug, on regional cerebral blood flow in patients with Parkinson's disease.

Sir: Despite the recent discovery of alterations in numerous central neurotransmitters and peptides in Parkinson's disease,¹ only dopaminergic or anticholinergic agents have been found to be active therapeutic agents. Previous studies have shown that acute administration of dopaminergic drugs (levodopa^{2,3} or bromocriptine⁴) induced a significant increase in rCBF in Parkinson's disease. However, as far as we know, no study has investigated the effects of anticholinergic drugs on rCBF in Parkinsonians. In this study, we investigated the influence of tropatepine, an anticholinergic agent commonly used in the treatment of Parkinson's disease.

Fifteen patients with idiopathic Parkinson's disease (10 men and five women, mean age: 63.5, SD 8.1 years), stage II to IV on the modified Hoehn and Yahr's staging were included in this study. All these patients were regularly treated with levodopa (plus dopa decarboxylase inhibitor). Tropatepine hydrochloride was injected acutely by intramuscular route at a dose of 10 mg, which is the dose commonly used in clinical practice to counteract the extrapyramidal effects of neuroleptic drugs. A first rCBF measurement was made with SPECT (Tomatic 64, Medimatic, Copenhagen) (for description see⁵) at time 0 (that is, at 11 a.m. before drug injection). The second measurement was performed at time 90 min, that is at the mean maximal peak plasma level of tropatepine. PCO₂ was measured and different regions of interest (ROIs) were determined as previously described.² The changes were evaluated before and after tropatepine by paired *t* test and the level of significance was *p* < 0.05.

There was no significant difference in PCO₂ before and after tropatepine (39.2, SD 3.1 versus 39.9, SD 3.1 mmHg). Under basal conditions in Parkinsonians, mean rCBF was 50.6, SD 11.4 ml/100 g/min which is a normal value for such a population under our experimental conditions.^{2,4}

Acute administration of tropatepine did not significantly change total CBF (50.7, SD 10.7 ml/100g/min) or rCBF in any ROI. The effect on extrapyramidal symptoms were not investigated and no side effect was observed except in one patient in stage II who developed 60 min after tropatepine a confusional state which spontaneously disappeared after 4 hours.

The study allows two conclusions to be made. First, it confirms our previous find-

ings^{2,4,6} that, under our experimental conditions, the total CBF of non-demented Parkinsonians (especially regularly treated ones) does not exhibit major changes in comparison with normal subjects. We failed to find any decrease in the frontal pattern, unlike Bes *et al.*⁷

Secondly, our results demonstrate that acute administration of an anticholinergic drug, in contrast to levodopa^{2,3} or bromocriptine,⁴ failed to change total or rCBF in Parkinsonians. In fact a physiological role for cholinergic mechanisms in the regulation of CBF has been suggested: binding studies showed the presence of muscarinic cholinergic receptor sites in cerebral blood vessels⁸ and cholinergic agents (like acetylcholine or physostigmine) were found to increase CBF.⁹ Soremin *et al.*¹⁰ suggested that, in rabbits, the cholinergic cerebral vasodilatation does not depend on cerebral metabolic activation and involved muscarinic receptors located beyond the blood brain barrier. Few studies have investigated the effects of anticholinergic drugs in man: using individual detectors and the [133 Xe] inhalation technique, Honer *et al.*¹¹ recently found that high doses of another anticholinergic drug, scopolamine reduced global CBF and especially frontal cortex perfusion in normal subjects. Although we used a therapeutic and effective dose (see the patient with the induced side effect) of tropatepine, we were unable to find similar results in Parkinsonians.

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References

- Javoy-Agid F, Ruberg M, Hirsch E, *et al.* Recent progress in the neurochemistry of Parkinson's disease. In: Fahn S, Marsden CD, Jenner P, Teychenne P, eds. *Recent Developments in Parkinson's disease 1986*; New York: Raven Press, 67-83.
- Montastruc JL, Celis P, Agniet A, *et al.* Levodopa-induced regional cerebral blood flow changes in normal volunteers and patients with Parkinson's disease. Lack of correlation with clinical and neuropsychological improvements. *Movement Disorders* 1987;2:279-89.