Letters

Paraplegia following oral hypotensive treatment of malignant hypertension

Sir: A 41 year old smoker was admitted to hospital with a two year history of headache and a five week history of painless haematuria. On examination his blood pressure was 200/140 mmHg supine and he had a grade IV hypertensive retinopathy. That evening he was given enalapril 2.5 mg and atenolol 100 mg. The following morning he complained of incontinence and severe weakness of his legs. On examination his blood pressure was 100/60 mmHg supine; there were no signs of an abdominal aneurysm and peripheral pulses were equal. He had absent abdominal reflexes and a spastic paraparesis with upgoing plantars. Pain sensation over the legs was impaired but temperature sensation was normal to routine tests. A brain CT scan was normal.

The acute paraplegia in this patient was probably due to infarction of the thoracic cord in the watershed zone between the anterior, circumflex and posterior spinal arteries following the rapid decrease in mean arterial blood pressure. There was no evidence to suggest a dissection of the aorta. We are not aware of any previous cases of paraplegia occurring in this setting although it has been reported following the hypotension of myocardial infarction and cardiac arrest.¹ The cerebral complications of rapid reduction in mean arterial blood pressure using intravenous therapy in malignant hypertension have been well documented.² This case suggests that the spinal cord, like the brain, is unable to autoregulate blood flow during rapid and extensive reductions of blood pressure in severe hypertension, and is a further reminder of the dangers of hypotensive treatment, even using oral agents.

P BROWN

M GROSS

M HARRISON

Department of Neurology,
Middlesex Hospital,
London W1N8AA, UK

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AIDS and catatonia

Sir: There have been several reports of the psychiatric complications of acquired immune deficiency syndrome (AIDS). In many of these cases psychiatric symptoms antedate neurological or immunodeficiency symptoms and the only evidence for AIDS may be a positive HTLV-III.¹

We report here a 19 year old hispanic male, who presented in a catatonic state and was discovered to be HTLV-III positive. The patient had no previous psychiatric history and his behaviour started deteriorating two months prior to admission into the hospital. During this time, the patient became progressively more lethargic and withdrawn, refused to speak or eat, and would remain motionless for hours at a time. Homosexuality was not confirmed but the family gave a history of a two-month incarceration one year prior to hospitalisation. At time of admission he was noted to have marked seborrhoea, mydriasis, intermittent lachrimation and oropharyngeal candidiasis. He was incontinent for faeces and urine, had a decreased gag reflex and showed increased muscular tone and brisk reflexes in all extremities. He was initially treated with 30 mg of haloperidol for two months with minimal response and a very slow clinical recovery of movement and speech. The patient remained with a residual deficit. Laboratory findings was normal. The lumbar puncture revealed no evidence of an infectious process. CT scan of the brain revealed diffuse cortical atrophy. EEG showed diffuse cortical slowing. The positron emission tomography scans showed increased blood flow and glucose metabolism in the basal ganglia (caudate, putamen) and right temporal cortex.

Acute onset of illness, lack of previous psychiatric illness, lack of a family history for psychoses along with the cortical atrophy, the cerebral metabolic abnormalities and positive HTLV-III suggest an AIDS encephalopathic process.

Damage to the CNS has been postulated to occur secondary to opportunistic infections, malignancies or from direct invasion of the CNS by the HTLV-III.^{2,3} In some of these cases, the psychiatric features may be more prominent than the neurological ones and may occur without manifestations of immunodeficiency.⁴ Predilection of some viruses for behaviorally related brain areas (temporal lobes, orbital frontal cortex, hippocampal gyrus) account for the high incidence of psychiatric symptoms in some patients with viral encephalitis.⁵ Furthermore, viral encephalitis presenting with

catatonic syndrome have been found to affect mostly the limbic system (medial temporal cortex). Catatonic features have also been observed in humans in lesions of the basal ganglia.⁶ Careful investigation of the brain areas most affected by HTLV-III may provide insight into the neuropsychiatric implications observed in the AIDS patient.

HTLV-III in cerebrospinal fluid⁷ in conjunction with mental state examination, CT scan and EEG may help identify encephalopathy in patients at risk for AIDS.

NORA D VOLKOW

ANDREW HARPER

DEANNA MUNNISTERI

JEFFREY CLOTHIER

University of Texas Health

Science Center in Houston,

PO Box 20708,

Houston, TX 77030,

USA

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Hemiballism and CT-documented lacunar infarct in the lenticular nucleus

Sir: Hemiballism is a rare condition usually due to an infarction or a haemorrhage in the contralateral subthalamic nucleus.¹ However, in several cases,² this nucleus has been intact at post-mortem examination. Only a few cases of hemichorea-hemiballism have been reported in which a contralateral vas-

cular lesion was documented by CT scan.³⁻⁵ We report a patient with reversible hemiballism related to a CT scan documented lacunar infarct in the lateral part of the contralateral lenticular nucleus.

A previously healthy 57 year old man was admitted 12 hours after the sudden onset of uncontrollable, rapid and large amplitude movements of the right limbs. Proximal joints were mainly affected by stereotyped abduction-inward rotation movements at the shoulder and flexion-extension movements at the hip. Neurological examination revealed no other abnormality. The blood pressure was 170/110 mm Hg, equal in both arms. The heart was normal at clinical and electrical examination. Cervical arteries were normal at Doppler and echotomography. Treatment with haloperidol, 2mg three times a day, was started on admission. The ballistic movements disappeared within 6 hours. The only residual anomaly was a slight muscular hypotonia of the right limbs. Three days after admission, haloperidol was discontinued without recurrence of the dyskinesia. Two unenhanced CT scans (6 mm thick slices) were performed on the 5th and 25th days after clinical onset. A small hypodense area in the lateral part of the left lenticular nucleus was suspected on the first CT scan. This hypodense area was larger and better defined on the second CT scan (fig). The patient was discharged on antihypertensive therapy and, at one year follow-up, he had no further symptoms.

Fig Unenhanced CT scan (performed 25 days after onset): low density area in the lateral part of the left lenticular nucleus (arrow).

In this patient with hemiballism, CT scan disclosed a small low-density area in the lateral part of the contralateral lenticular nucleus. The increase in size of the hypodensity at two successive examinations was consistent with a lacunar infarct of the same date as the clinical symptoms. Review of the pathological data in cases of ischaemic hemichorea-hemiballism from the literature reveals that most of them were related to lacunar infarcts in the subthalamic nucleus,⁶ in its afferent and/or efferent pathways,⁷ in the striatum² or in the thalamus.⁶ Therefore, hemichorea-hemiballism has been included in the group of clinical syndromes most commonly caused by lacunar infarcts.⁸ In our experience, most patients with this syndrome have normal CT scans. However, the CT scan underestimates the extent of vascular lesions, at least as judged by magnetic resonance imaging, which will probably detect more often the lesion responsible for this syndrome.

Lacunar infarcts result from occlusion of perforating branches of the large cerebral arteries. The cause of the occlusion is most frequently lipohyalinosis or microatheroma, involving the walls of the perforating arteries,⁸ two arteriopathies encountered in long-standing hypertension. However, hemichorea-hemiballism caused by embolisation from the heart⁶ or by atheromatous disease of internal carotid or posterior cerebral arteries have been reported.⁷ Transient hemichorea-hemiballism, as the main or sole manifestation of TIAs, has also been described in relation to basilar⁹ or internal carotid¹⁰ atheromatous disease. Consequently, assessment of a patient with this syndrome should be the same as for any other stroke, and should include requisite investigations to specify the mechanism of the causal vascular injury. Spontaneous outcome of hemiballism is unpredictable; dyskinesias may last days, months or even years.¹⁶ Neuroleptic therapy usually induces a dramatic improvement in dyskinesias¹¹ but spontaneous recovery can be masked by this treatment. Therefore, neuroleptic therapy should be discontinued rapidly to detect whether or not recovery of dyskinesias have occurred.

JEAN-LOUIS MAS*

MICHEL LAUNAY†

CHRISTIAN DEROUESNE*

Clinique des Maladies du
Système Nerveux*

Service de Neuroradiologie,†

Hôpital de la Salpêtrière,

47 Bld de l'Hôpital,

75651 Paris, Cedex 13,

France

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Parkinson's disease in twins

Sir: Debate on the inheritance of Parkinson's disease continues. It is not uncommon for a patient to say that another family member has been similarly affected. However, Parkinson's disease is a common illness, particularly in those over the age of 60 years, and benign essential tremor which is often mis-interpreted as Parkinson's disease, is even commoner. Duvoisin *et al*¹ personally examined a large number of relatives of both patients and of their spouses, but found approximately equal numbers of secondary cases of Parkinson's disease in both groups. This evidence against any major genetic contribution to the aetiology of the disease was given strong support by a subsequent study of Parkinson's disease in twins.² When twins, one of whom had definite Parkinson's disease, were examined, only one of the 43 pairs of monozygotic twins was definitely concordant for Parkinson's disease, and none of 19 dizygotic pairs was concordant. Despite this evidence, however, others³ have subsequently continued to suggest that inheritance plays a part in Parkinson's disease, at least in some fami-