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Recurrent intracranial haemorrhage in Behcet disease

Sir: I read with interest the contribution by Nagata entitled "Recurrent intracranial haemorrhage in Behcet disease".¹ Nagata attributes the intracranial haemorrhages principally to hypertension and hypertensive arterial changes. I do not think that atherosclerotic embolic disease however has been excluded as a potentiating factor for such a rapid flurry of intracranial accidents. Diffuse disseminated atheroembolisation is a disorder wherein atheromatous fragments chronically shower the peripheral vasculature in small to large numbers, producing radiographic and clinical pictures that are often indistinguishable from lacunar stroke syndrome. Moreover there is a high incidence of gastrointestinal ulceration and inflammation which is often indistinguishable histopathologically from a vasculitis. In addition there is a predilection for the basal ganglia as we recently reported in a study of the neuro-ophthalmic manifestations of diffuse disseminated atheroembolisation.² I would be most interested to learn if this patient's abdominal aorta had the characteristic "brittle" appearance associated with diffuse disseminated atheroembolisation and if review of the gastrointestinal and kidney slides showed cholesterol clefts in any of the small arterioles.

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- ¹ Nagata K. Recurrent intracranial haemorrhage in Behcet disease. *J Neurol Neurosurg Psychiatry* 1985;48:190-1.
- ² Coppeto JR, Lessell S, Lessell IM, Greco TP, Eizenberg MS. Diffuse disseminated atheroembolism; three cases with neuro-ophthalmic manifestation. *Arch Ophthalmol* 1984;102:225-8.

Nagata replies:

In answer to the letter from Dr Coppeto, there was no pathological evidence of a disseminated atheroembolism in our case. As I have reported, perivascular inflammatory changes in the intracerebral small vessels were in an active stage that could cause organic changes of the vessel wall, and

there was much greater evidence of hypertensive vascular changes which are closely associated with hypertensive intracerebral haemorrhage. Although there were small softening foci scattered in the cerebral white matter and in the pons, we did not find cholesterol emboli in these small ischaemic lesions. The abdominal aorta did not have a "brittle" appearance of atherosclerosis. The ulceration and erosion of the gastrointestinal tract were much different from those due to vasculitis or disseminated embolism. There were no cholesterol clefts in the small arteries of the intestine or kidneys as far as we investigated. In addition, the onset age of this patient was much younger than that of reported cases with histopathological features of disseminated atheroembolism. Moreover, in most of the reports concerning intracranial disseminated atheroembolism, the intracranial accidents occurred as a form of cerebral infarction. All three cases of diffuse disseminated atheroembolism reported by Coppeto *et al* (1984) also showed ischaemic cerebrovascular process. As far as the haemorrhagic lesions in this patient are concerned, it is very hard to substantiate that they were associated with disseminated atheroembolism, which was not proved in any other organs in this patient.

The spoon test for assessing sudomotor autonomic failure

Sir: I read with interest the reported new "Spoon test" by SA Tsementzis and ER Hitchcock.¹ This test describes the use of an English kitchen soup spoon but we in the former colonies are familiar with the test using an American teaspoon. This was reported by Doctor Ernest Bors in his book² as follows: "The autonomic deficit can be assessed by determining skin sweating at rest. The simplest clinical examination consists of letting the convexity of a teaspoon glide by its own weight over the skin in a zigzagging fashion. When the spoon starts sticking to the skin, the level of skin moisture has been reached. It is necessary to keep the spoon dry and to exclude contact sweating caused by skin folds (axilla, elbow, inguinal region, flexor surface of the knee) or by bed blankets."

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References

- ¹ Tsementzis SA, Hitchcock ER. The spoon test: a simple bedside test for assessing sudomotor autonomic failure. *J Neurol Neurosurg & Psychiatry* 1985;48:378-80.
- ² Bors E, Comarr AE. History and Examination. In: *Neurological Urology: Physiology of Micturition, Its Neurological Disorders and Sequelae*. Baltimore, University Park Press 1971:170.

Hitchcock writes:

Although unaware of the work at the time we presented our paper it has been brought to our attention that the same test was described more than 20 years ago by Bors.¹ We did not in our paper therefore attribute, as we should have done, the test to this author but we would like the reference to be generally known and we append it at the end of this letter.

The fact that the test has already been described in no way detracts from presenting the paper which made a critical examination of the accuracy of a test which we believe is so useful it deserves reviving.

Reference

- ¹ Bors E. Simple methods of examination in paraplegia: I. The spoon test. *Paraplegia* Vol II 1964;1:17-9.

Delayed onset dystonia

Sir: Burke, *et al*¹ wrote about eight patients with dystonia appearing one to 14 years after a cerebral insult. Although previous examples of delayed-onset dystonia can be found in the literature, the clinical importance of this phenomenon had not been adequately emphasised. The comprehensive review of the literature of Burke *et al* failed, however, to quote an earlier description and pathophysiological discussion of delayed-onset dystonia. In 1966, one of us (LBB) reported² two young patients with axial and bilateral upper limb dystonia in whom the diagnosis of idiopathic torsion dystonia had been suggested in view of the normal early development of the dystonia and absence of additional neurological signs. A positive history of perinatal anoxia in one and of prolonged and complicated labour in the second patient were the only diagnostic clues in these cases.

We agree with Burke *et al* in that

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delayed-onset dystonia following perinatal anoxia probably has been underestimated in the past. Thus, 8% (5 of 40) of our patients with torsion dystonia as the only clinical abnormality, had a positive history of complicated labour, but only one patient had suffered severe asphyxia. The practical problem one faces in the other four patients is to decide whether they actually have delayed-onset dystonia or if expression of their dystonia was facilitated by perinatal anoxia. In this relatively small number of patients we have noticed the coexistence of dystonia with other abnormal movements in the form of postural tremor (three patients) ballism (two patients) and reflex or action myoclonus (three cases). The possible diagnostic importance of these findings will only be judged adequately after extensive clinical-epidemiological data about the incidence of other movement disorders in primary torsion dystonia is collected. To our knowledge such information is not yet available. For the time being in the absence of a definitive marker for torsion dystonia, the role of anoxia as an aetiological or predisposing factor in patients with pure dystonia and normal laboratory tests will be difficult to assess.

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- ¹ Burke RE, Fahn S, Gold AP. Delayed-onset dystonia in patients with "static" encephalopathy. *J Neurol Neurosurg Psychiatry* 1980;43:789-97.
- ² Barraquer Bordas L. Sobre la evolución clínica de algunos síndromes extrapiramidales de instauración infantil. *Pres Med Argent* 1966;53:123-9.

We are grateful to Obeso and his colleagues for bringing to our attention two probable cases of delayed-onset dystonia due to perinatal anoxia. We would certainly agree that there have been a number of clear descriptions of this phenomenon prior to our report of 1980. We would also agree that the incidence of this occurrence is probably underestimated; since our original report we have seen numerous additional cases.

We strongly concur with Obeso *et al* that

this form of dystonia can be clinically indistinguishable from idiopathic (primary) torsion dystonia. Thus, like Marsden and Harrison¹ we believe that a diagnosis of idiopathic torsion dystonia cannot be made in the presence of a history of an abnormal birth. In the absence of markers for the primary dystonias, diagnosis can be difficult where there is some mild or questionable birth complication or development delay. Given the clinical similarity between delayed-onset and primary dystonia, it was our policy in a recent study of the clinical course of autosomal dominant torsion dystonia among non-Jews to exclude from consideration any individual within a pedigree who had a history of frank birth injury.² Such an exclusion criterion is needed to insure a pure sample of primary dystonics, even within a family group.

Not only will the discovery of markers for the primary dystonias assist differentiation between these two conditions, but also the development of measures of the degree of birth asphyxia. In addition, it is possible that magnetic resonance imaging may prove useful by allowing visualisation of striatal pathology, which would be anticipated in dystonia due to perinatal asphyxia.

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- ¹ Marsden CD, Harrison MJG. Idiopathic torsion dystonia: a review of 42 patients. *Brain* 1974;97:793-810.
- ² Burke RE *et al*. The clinical course of autosomal dominant torsion dystonia. *Neurology (Cleveland)* 1985;35 (Suppl 1):273.

Book reviews

The Psychopharmacology of Epilepsy. Edited by Michael R Trimble. (Pp 184; £21.50.) Chichester: John Wiley & Sons Ltd, 1985.

Everyone involved in the care of patients with epilepsy is aware of the striking association between epilepsy and psychiatric disorder, which is most common in patients with complex partial epilepsies.

Hughlings Jackson believed that "epilepsy was the cause of insanity in 6% of the insane". Would that things were so black and white! This book, whilst short, explores the shades of grey in some depth. It is a multi-author volume. The Editor contributes both an introductory review and a conclusion. The meat in the sandwich consists of a number of chapters, some of which represent review articles, others of which contain some original investigative data. The topics include interesting chapters on the kindling model of epilepsy which explores both the neurochemical basis, and animal behavioural changes which occur during the process. Subsequent chapters review the roles of anti-epileptic drugs in psychopathology, and the associations between depression and psychosis and epilepsy. The practical problems of how to treat psychiatric disorder in patients with epilepsy are considered and perhaps the most useful chapter documents the difficulty of defining the extent to which antidepressant drugs can cause seizures. Finally the possibility that anticonvulsant drugs may have psychotropic properties is explored. A considerable amount has been written concerning the relationship between epilepsy and psychiatric disorder over recent years. This book is a useful summary of the field, but readers should not expect any clear answers to the many questions and problems they face in everyday clinical practice. One area that one was sorry to see receive little attention was the question of whether criminal, violent, or generally antisocial behaviour may occur as a sub-clinical ictal phenomenon. Clinical neurologists rarely accept this but will be well aware that many of their psychiatric colleagues seem to hold a diametrically opposed view as judged by the number of referrals of such patients who have some form of EEG abnormality!

When one accepts that no specific psychiatric disorder can be associated with epilepsy, and that biological factors, drugs