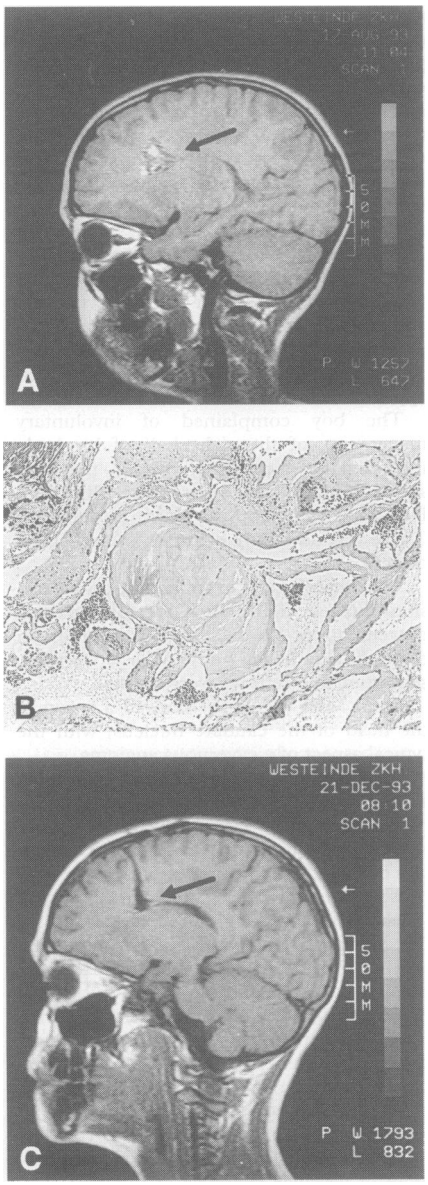

(A) Typical aspect of cavernous angioma in the head of the caudate nucleus (MRI image, SE TR 520/TE 25, after intravenous contrast). (B) Cavernous angioma with anastomosing vascular channels, with fibrotic walls and organised thrombus (centre). There is no pre-existing brain tissue between the vessels (haematoxylin-eosin, originally × 100). (C) Control MRI after removal of the cavernous angioma, showing a postoperative defect.

become symptomatic between the ages of 20 and 40 years.

The natural history of sporadic cavernous angiomas is not well known. Two retrospective review studies of presumed cavernous angiomas, detected by MRI, showed that the most frequent presenting symptoms were seizure with 1·5% per person/year exposure, and haemorrhage, with estimated annual bleeding risks of 0·8%, and 0·25% per person/year respectively.[5 6] The risk of recurrent appreciable bleeding in CVMs that presented with haemorrhage is generally suggested to be larger, perhaps comparable with that in classic arteriovenous malformations, and may prove fatal. In children Scott *et al* recommend surgery if the lesion is safely accessible, is currently symptomatic, or shows evidence of having bled in the past.[4]

Results of operation in previously reported cases of CVMs in the basal ganglia presenting with (progressive) hemiparesis, however, have generally been poor.[2] Stereotactic localisation with CT guidance and microsurgical techniques have facilitated surgery in deep paraventricular or basal ganglia lesions, offering now a better perspective in these cases. This is the second case report describing effective surgical management in resolving a movement disorder due to a cavernous angioma of the basal ganglia, without complications. This indication for removal of a CVM should be considered in subsequent cases.

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MATTERS ARISING

The nature of apraxia in corticobasal degeneration

We read with interest the report by Leiguarda *et al*[1] about the nature of apraxia in corticobasal degeneration. The authors concluded that ideomotor apraxia is the most frequent type of apraxia in corticobasal degeneration. We disagree with them about the conclusion, and would like to comment on apraxia in corticobasal degeneration.

We have experienced four patients with corticobasal degeneration, two of whom were reported elsewhere.[2] These four patients presented consistently with asymmetric limb-kinetic apraxia, but with neither ideomotor apraxia nor ideational apraxia. Unlike ideomotor apraxia and ideational apraxia limb-kinetic apraxia is defined as a breakdown of previously skillful movements, manifested by difficulty in making fine finger movements.[3 4] These four patients also had difficulty in making gestures and using objects on the side of the greater clumsiness. Such apraxic disorders could not be considered ideomotor apraxia or ideational apraxia, because limb-kinetic apraxia can cause clumsiness in all praxic acts on the side contralateral to the lesion.[4]

The discrepancy between the report of Leiguarda *et al* and ours may partly be due to the heterogeneity of corticobasal degeneration or varieties of duration of the illness. On the other hand, the following possibilities may account for the differences. Firstly, as mentioned, limb-kinetic apraxia might induce a disorder of symbolic action, which mimicked ideomotor apraxia, leading to the authors' conclusion. Secondly, limb-kinetic apraxia and ideomotor apraxia might coexist. Limb-kinetic apraxia usually occurs on the side contralateral to the lesion, whereas ideomotor apraxia occurs bilaterally. Thus it is possible, as reported by Leiguarda *et al*, that only ideomotor apraxia is detectable on the side of least clumsiness.

Regarding the underlying mechanism of apraxia in corticobasal degeneration, Leiguarda *et al* attributed ideomotor apraxia to dysfunction of the supplementary motor area. However, the role of the supplementary motor area in motor acts still remains controversial. The supplementary motor area may play an important part, as well as the motor cortex, in execution of complex finger movements and may not work as a supramotor centre.[5] It seems likely that the apraxic disorders arise from another cortical lesion. Neuropathological studies have shown that the sensorimotor cortex is predominantly involved in corticobasal degeneration.[6 7] With SPECT, we showed that cerebral blood flow was mainly decreased in the unilateral perirolandic cortices in all four patients. The perirolandic cortical hypoperfusion could account for contralateral limb-kinetic apraxia, as a lesion in the sensorimotor cortex induces limb-kinetic apraxia on the contralateral side.[4] We therefore consider that limb-kinetic apraxia is the most frequent type of apraxia in corticobasal degeneration, even if ideomotor apraxia or ideational apraxia may exist.

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Leiguarda et al reply:

We appreciate Okuda and Tachibana's comments about our paper on apraxia in corticobasal degeneration[1] because they enable us to clarify the status of limb-kinetic apraxia, a point which is particularly relevant for the clinical interpretation of the apractic disorders in patients with corticobasal degeneration.

Limb or melokinetic apraxia (originally called "innervatory apraxia" by Kleist[2]) was considered by Liepmann to be a form of limb apraxia due to the loss of "kinaesthetic-innervatory engrams" secondary to "sensorimotor" damage.[3] The disorder is characterised by clumsiness in the performance of fine motor acts by the hand contralateral to the hemispheric damage. All types of movements including gestures, regardless of whether the patient creates or imitates them, become "uncouth, clumsy, inexpert, and preceded by fruitless attempts which only bring the wrong muscles into play."[4]

The status of limb-kinetic apraxia has been debated[5] for over three decades. Most authors have refused to consider symptoms connected with limb-kinetic apraxia as apractic.[6] Geschwind disregarded it completely[7] and Rothi *et al* failed to include limb-kinetic apraxia in their neuropsychological model of limb praxis.[8] Brain concluded that it is simply a partial symptom of pyramidal tract lesion,[9] a view also shared by Ajuriaguerra and Tissot,[10] Hecaen and Rondot,[6] and Mesulam.[11] De Renzi in particular contends that "limb-kinetic apraxia has never been described with sufficient accuracy to be distinguishable from a mild form of paresis and to gain acceptance by neurologists."[12] In support, monkeys with lesions restricted to the corticospinal tract show similar errors.[13]

This may be an extreme view of limb-kinetic apraxia. Lipmann's definition of apraxia may be summarised as a deficit in the performance of purposeful skilled movements, in the absence of elementary motor (weakness, akinesia, abnormal posture, or tone) or sensory deficits, or of impaired comprehension or memory.[3] The disruption of movement seen in lesions of the corticospinal pathway, or as seen in Parkinson's disease, can seldom be fully explained by weakness, akinaesia, abnormal posture, or tone. There is additional breakdown of the movement pattern or formula—Liepmann's innervatory engram—that suggests a higher motor disorder or apraxia. This is exactly what is seen to a pronounced degree in corticobasal degeneration, particularly in the initially affected limb. To this extent we agree with Okuda and Tachibana.

We deliberately employed standardised tests for ideomotor and ideational apraxia, however, and concentrated on the less affected limb. We did not explore the contentious topic of limb-kinetic apraxia, because it is such an uncertain area. Nevertheless, we agree that patients with corticobasal degeneration characteristically exhibit a higher order motor deficit in their more affected limb, which we would be happy to call limb-kinetic apraxia if others would allow the term! Our study also shows that many patients with corticobasal degeneration likewise fail tests for ideomotor apraxia, a failure that we do not think can be explained by limb-kinetic apraxia alone.

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Transient epileptic amnesia—a clinical update and a reformulation

In relation to the interesting article by Kapur[1] on transient epileptic amnesia, I would like to remind your readers of some of the published work related to such findings. Thus experimental studies, using either intracarotid sodium amylobarbitone or electrical stimulation for diagnostic purposes on epileptic patients, have shown associations between the temporal lobe of the hemisphere dominant for speech and both memory and consciousness.[2,3] It is important to keep this in mind when discussing the anatomical and pathophysiological basis of amnesic phenomena, transient or otherwise.

EA SERAFETINIDES

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NOTICE

The winter meeting of the **British Neuropsychiatric Association** will take place in the Conference Theatre London Zoo, on 20 January 1995. The subject will be *the neuropsychiatry of vascular disease*. For further information please contact Sue Garrett, Administrative Assistant BNPA, 17 Clocktower Mews, London N1 7VV, UK. Telephone/fax 071-226 5949.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiratory date, and your full name.

Manual of Psychiatric Emergencies (third edition). Edited by STEVEN E HYMAN and GEORGE E TESAR. Publisher: Churchill Livingstone, Edinburgh 1993. (Pp 354; £21.95.) ISBN 316387282.

When the Editor sent this little book to me for review my first response was "Oh dear, another psychiatric vade-mecum". Other texts about psychiatric emergencies suggest that authors often have difficulty finding enough to say about psychiatric emergencies, with the result that what emerges is a short textbook of psychiatry rather than a text more precisely focused upon emergencies. That can only be done from the perspective of a casualty officer. That is exactly what these authors have achieved by compiling the views of some thirty contributors most of whom, one suspects, have had significant experience of being on-call in a busy modern general hospital.

The initial section is a series of contributions providing practical and explicit guidance about the assessment of different presenting problems; The Emergency Psychiatric Evaluation, with special emphasis upon neuropsychiatric evaluation, Crisis, The Suicidal Patient, the Violent Patient, Acute Grief and Disaster Victims, Families