



Axial CT with injection of contrast, showing two recent areas of hypodensity in the right frontal and left frontoparietal regions corresponding to pial vessel infarcts in the territory of the right and left middle cerebral artery.

artery,⁴ the proximal carotid "stump"³ or, in the iatrogenic circumstances of this patient, the aorta. Periorbital directional Doppler, however, demonstrated normal flow in the ophthalmic artery. Secondly, embolisation through extracranial-intracranial anastomosis could be responsible but it is unlikely as these are considered too narrow to allow an embolus responsible for such a large left hemispheric infarction to pass. A third possibility is embolisation of thrombotic material breaking off from the distal soft "white tail" of the thrombus located in the left internal carotid artery.² This hypothesis is lacking support: there was arteriographic evidence of internal carotid artery occlusion for at least six years and a "soft white tail" has little chance of persisting for six years after occlusion of the internal carotid artery. Fourthly, infarctions might result from haemodynamic alterations in blood flow, but at onset there was no evidence of haemodynamic attacks with a low flow state during the transluminal angioplasty. Furthermore, the two ischaemic areas were not similar to those described in watershed infarcts.⁶ Therefore evidence for cortical low flow infarcts in this patient is lacking.

We believe that the most likely cause of the left hemispheric infarction is an embolism across the circle of Willis, in this case embolisation through the anterior communicating artery caused by thrombotic material broken away from thrombi located either in the aorta or the contralateral, stenosed right internal carotid artery where thrombotic material was floating in the lumen. This hypothesis is strongly supported by the presence of left and right hemispheric infarcts of the same age. Embolism across the circle of Willis seems the only plausible mechanism for left hemispheric infarction in our patient.

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- 1 Coté R, Tayler DW, Barnett HJM. Internal carotid occlusion: a prospective study. *Stroke* 1983;14:898-902.
- 2 Finkelstein S, Kleinman GM, Cuneo R, Baringer JR. Delayed stroke following carotid occlusion. *Neurology* 1980;30:84-8.
- 3 Barnett HJM, Peerles SJ, Kaufmann JCE. "Stump" of internal carotid artery: a source of further cerebral ischemia. *Stroke* 1978;9:448-56.
- 4 Gertler JP, Cambria RP. The role of the external carotid endarterectomy in the treatment of ipsilateral internal carotid occlusion: collective review. *J Vasc Surg* 1987;6:158-67.
- 5 Georgiadis D, Grosset DG, Lees KR. Transhemispheric passage of microemboli in patients with unilateral internal carotid artery occlusion. *Stroke* 1993;24:1664-6.
- 6 Weiller C, Ringelstein EB, Reiche W, Buell U. Clinical and haemodynamic aspects of low-flow infarcts. *Stroke* 1991;22:1117-23.

MATTERS ARISING

Elementary visual hallucinations in migraine and epilepsy

We would like to add a cautionary note to the highly interesting study by Panayiotopoulos¹ on the different elementary visual hallucinations in migraine and epilepsy. The paper concludes that visual hallucinations in occipital epileptic seizures are predominantly multicoloured as opposed to predominantly black and white patterns in migraine.

To be able to reach this conclusion, there needs to be certainty that the diagnosis was correct. This is most likely the case for the patients with epilepsy as in all there was either evidence of spike and slow wave activity or a structural occipital lobe lesion. The group of patients assigned to the migraine group are, however, not clearly defined. The appreciable difficulty in being able to differentiate between migraine and epilepsy is stated but too little is said about the possibility of false diagnosis in the migraine group. So it is possible that some of the patients diagnosed as having migraine actually have occipital epilepsy. This would in turn falsify the conclusion of the study.

To illustrate the difficulty of ascribing a diagnosis of migraine to patients without evidence of spike and slow wave activity or a structural occipital lobe lesion we refer to a patient we described earlier² who experienced visual hallucinations (distorted vision and false colours). She was repeatedly diagnosed as having migraine. Doppler sonography of the posterior cerebral arteries during symptoms showed increased blood flow velocity typical of local autoregulatory

hyperperfusion due to increased neuronal activity. This enabled the diagnosis of migraine to be excluded and a diagnosis of occipital epilepsy to be established. Ictal EEG was non-specifically slowed.

As we do not know how many of the migraine group in Panayiotopoulos's study really had migraine, we urge caution in the interpretation and application of the proposed conclusion.

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- 1 Panayiotopoulos CP. Elementary visual hallucinations in migraine and epilepsy. *J Neurol Neurosurg Psychiatry* 1994;58:1371-4.
- 2 Wilder-Smith E, Nirrko A. Contribution of concurrent Doppler and EEG in differentiating occipital epileptic discharges from migraine. *Neurology* 1991;41:2005-7.

Panayiotopoulos replies:

In my report on elementary visual hallucinations in migraine and epilepsy I thought that I was unduly overemphasising that visual partial epileptic seizures may be misdiagnosed as migraine and the need for a precise description of the visual hallucinations in these two conditions. If anything, I was biased stressing the possibility of falsely diagnosing migraine instead of epilepsy rather than the other way round. Two out of the four illustrative cases were selected to demonstrate this diagnostic error.

Therefore, I thank Wilder-Smith for his letter, which reassured me that my above fears were unfounded as he stresses the same point—namely, that visual partial seizures may be misdiagnosed as migraine. He goes one step further however, arguing that some of my patients diagnosed with migraine may have had occipital epilepsy. I do not think that this mistake was made because in all 50 patients the diagnosis of migraine was based on strict clinical criteria, a long follow up, response to treatment, and not only on a normal or equivocally abnormal EEG. In particular, all 47 patients with classic migraine had the characteristic migrainous visual prodrome lasting 5–20 minutes before the onset of mainly unilateral headache characteristic of migraine. Not a single patient in the migraine group had any suggestion of epileptic seizures, which, given my special interest in these conditions,^{1,2} I would be able to recognise.

The author also wishes to discuss his published case which, like my cases, was misdiagnosed as migraine. I did not cite his report because although the "coloured" visual hallucinations of this patient were consistent with my findings, misdiagnosis was not indicated and previous attacks were monocolourally described as "migraine". More clinical details along the lines of my report and previous reports¹ from Wilder-Smith would be more enlightening. The patient had clusters of "15-30 second attacks of distorted vision and false colours" associated with simultaneous and equally brief ictal EEG changes. The diagnosis of visual partial seizures should be clear and if these were of acute onset in adult life, MRI instead of Doppler would be more appropriate. More confidence in the clinical symptoms, which is the main point of my report, may have avoided the need for further investigations and delaying treatment.