artery, the proximal carotid artery "stump" or, in the iatrogenic circumstances of this patient, the aorta. Peribarital directional Doppler, however, demonstrated normal flow in the ophthalmic artery. Secondly, embolisation through extracranial arterial 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. 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 transcluminal angioplasty. Furthermore, the two ischaemic areas were not similar to those described in watershed infarcts. 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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MATTERS ARISING

Elementary visual hallucinations in migraine and epilepsy

We would like to add a cautionary note to the highly interesting study by Panayiotopoulos et al 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 earlier who experienced visual hallucinations (distorted vision and false colours). She was repeatedly diagnosed as having migraine. Doppler sonography of the posterior cerebral arteries during symptomatic showed increased blood flow velocity typical of all 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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Panayiotopoulos replies:

In my report on elementary visual hallucinations in migraine and epilepsy I thought that I was unduly overemphasising that visual parietal 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 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 migraine can be misdiagnosed as migraine because of his temporal retardation leading to wrong diagnosis. He would 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 prodromi, which are usually present 20 minutes before the onset of main uni-lateral 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, 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 monocularly described as "migraine". More clinical details along the lines of the lines of my report and previous reports 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.
Antiganglioside antibodies in the CSF of patients with motor neuron disease and Guillain-Barré syndrome

In a recent report in this Journal Stevens et al described increased titres of antiganglioside antibodies (AGAs) in the CSF of patients with amyotrophic lateral sclerosis.1 They concluded that patients with amyotrophic lateral sclerosis have raised CSF IgM antibodies to all gangliosides except asialo GM1 (A-GM1), due to a chronic intrathecal immune response. The authors did not, however, evaluate other motor neuron disorders related to amyotrophic lateral sclerosis and with sometimes borderline diagnosis.2 We have studied AGA reactivity in the CSF of 23 patients whose diagnosis included (a) four strictly defined patients with amyotrophic lateral sclerosis; (b) 13 patients with lower motor neuron signs, from which six had a syndrome of multifocal motor neuropathy with conduction block and two had overactive tendon reflexes in limbs, with weak, wasted, twitching muscles, Romberg's sign or ankle clonus; and (c) the patient with Guillain-Barré syndrome and three patients with chronic inflammatory demyelinating neuropathy. Thirty three subjects were tested as controls, including 28 patients with other neurological disease and 10 people whose CSF was normal and in whom irrelevant diseases, such as migraine or tensional headache, were found after later studies (normal controls).

Serum and CSF were assayed for antibodies to gangliosides GM1, GD1b, GD1a, and A-GM1 by enzyme linked immunosorbent assay (ELISA) according to the method described by Noble-Orazi et al.3 Results were expressed as the mean absorbance obtained from the well coated with ganglioside minus the absorbance obtained from a bovine serum albumin coated well. Results were considered positive when this difference exceeded 0.1. Concentrations of AGA were considered to be increased if this titre was higher than 3 SD from the mean of the results obtained by Noble-Orazi et al.4 Results were expressed as the mean absorbance obtained from the well coated with ganglioside minus the absorbance obtained from a bovine serum albumin coated well. Results were considered positive when this difference exceeded 0.1. Concentrations of AGA were considered to be increased if this titre was higher than 3 SD from the mean of the results obtained by Noble-Orazi et al.4

The authors report significantly increased antibody titres and evidence of intrathecal immunoglobulin synthesis in the CSF of patients with amyotrophic lateral sclerosis,1 Guillain-Barré syndrome, and other motor neuron disorders. They conclude that CSF immunoreactivity to AGM1, GD1b, and GM1 in the CSF of patients with amyotrophic lateral sclerosis and lower motor neuron disease, as well as from Guillain-Barré syndrome. They conclude that CSF immunoreactivity to AGM1, GD1b, and GM1 is specific for these disorders. Although they interpret their data as affirmative for an intrathecal immunological process that is typically raised in this disease,1 they report antibody spectra differing from those in our sample of patients with amyotrophic lateral sclerosis. On closer scrutiny, this seems not to be the case, as anti AGM1 IgM antibodies do appear in CSF of nine of 35 patients of our previously reported sample. Anti-AGM1 antibodies are not, however, part of the panel of antibodies that are typically raised in this disease.1 Although the comparative approach of Iniguez et al is up to date, due to the small sample size the results are difficult to interpret in terms of specificity and sensitivity— for example, the CSF-IGM and the IgG index of their patients are raised (which was not the case in our study) but are not reported as significant due to large within-group variation. The results within the three

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