Urine output increased to 2000 ml in the next 24 hours, and there was no further decrease in renal function. The patient was discharged from the hospital two weeks later.

**Nerve conduction studies**

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Latency (ms)</th>
<th>Velocity (m/s)</th>
<th>Amplitude (μV)</th>
<th>Minimum F-latency (ms)</th>
<th>SNAP amplitude (μV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>4.0</td>
<td>44.3</td>
<td>70.0±7.0</td>
<td>30.4</td>
<td>34.0</td>
</tr>
<tr>
<td>Ulnar</td>
<td>8.0</td>
<td>54.4</td>
<td>67.7±7.1</td>
<td>28.9</td>
<td>31.2</td>
</tr>
<tr>
<td>Peroneal</td>
<td>3.9</td>
<td>46.1</td>
<td>41.4±7.1</td>
<td>57.2</td>
<td>25.1</td>
</tr>
<tr>
<td>Tibial</td>
<td>3.9</td>
<td>43.9</td>
<td>11.6±9.5</td>
<td>52.7</td>
<td>25.1</td>
</tr>
</tbody>
</table>

**Sensory**

- **Rural:** 3.2, 43.8 μV
- **Median:** 3.0, 46.7 μV
- **Ulnar:** 2.9, 48.3 μV
- **Radial:** 2.0, 50.0 μV

**SNAP =** Sensory nerve action potential; **R =** right.

**Letters to the Editor**

Ebolism across the circle of Willis

Ebolism is one of the proposed causes of ischaemic strokes delayed in onset after internal carotid artery (ICA) occlusion. The embolus may derive from a "tail" of thrombus that lies at the top of the ICA, or from thrombus from the ipsilateral extracranial carotid artery passing through the pial vessels of the ophthalmic artery. Another mechanism could be embolisation across the internal carotid artery to supply the ophthalmic artery through the anterior communicating artery. A 74 year old right handed man underwent right carotid endarterectomy six years earlier for right transient cerebral ischaemia. Conventional arteriography showed a stenosis greater than 70% of the right internal carotid artery and an occlusion of the left internal carotid artery. Six years later a right renal artery stenosis was discovered that required transluminal angioplasty. During the procedure, the auxiliary catheter was removed through the aorta, the patient sustained a right hemispheric infarction by embolic migration through the anterior communicating artery.

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artery, the proximal carotid "stump" 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-necranial 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 infarct 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 transuminal 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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SERGE TIMSIT
DOMINIQUE LAAENGH-MASSONI
RACHID MANAI
GERALD RANCUREL

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.

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Letters to the Editor

PANAYIOTOPoulos' report: In my report on elementary visual hallucinations in migraine and epilepsy I thought that we 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 arguments were not 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 his 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 phenomena (20-30 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, 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 intended and previous attacks were monocularly described as migraine.

More clinical details along 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.

1 Panayiotopoulos CP. Elementary visual hallucinations in migraine and epilepsy. J Neurol Neurosurg Psychiatry 1978;41:1171-1

MATTERS ARISING

Elementary visual hallucinations in migraine and epilepsy

We would like to add a cautionary note to the highly interesting study by Panayiotopoulos' 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 migrainous 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 migrainous 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 symptoms showed increased blood flow velocity typical of focal intracranial 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' study really had migraine, we urge caution in the interpretation and application of the proposed conclusion.

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Panayiotopoulos replies:

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Embolism across the circle of Willis.

K Wegener, S Timsit, D Laaengh-Massoni, R Manaï, G Rancurel and E Kieffer

J Neurol Neurosurg Psychiatry 1995 58: 517-518
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