with pseudotumor cerebri associated with obesity and focal neurological deficits including right lower motor neuron type facial palsy and hemiparesis. The right hemiparesis and cranial nerve deficits recovered completely after treating the pseudotumour cerebri with lumbar puncture, steroids and Diamox. Even though men with PTC are less likely to be obese than women, they tend to be more obese than control subjects and should be counselled on weight reduction diets.⁷ If focal neurological deficit disappears with treatment of pseudotumour cerebri including diuretics, weight reduction, steroids, lumboperitoneal shunting procedure or optic nerve sheath decompressions, it should be considered as a part of pseudotumour cerebri rather than as a casual finding. Patients with atypical pseudotumour cerebri should be followed closely.

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MATTERS ARISING

Cerebral blood flow and transient global amnesia

Fujii *et al*¹ reported regional cerebral blood flow (rCBF) and metabolism studies by positron emission tomography (PET) in four patients who experienced transient global amnesia (TGA). The authors concluded that TGA does not depend on persistent ischaemia. Unfortunately, the PET scans were performed one to five months after the episodes of TGA. CBF studies are rarely performed during TGA because the amnesic episodes last for a short period of hours. The authors pointed out that "further studies on cerebral circulation and metabolism are needed especially in the early stage or during the attack".

We have reported² measurements of CBF

in TGA using Xenon¹³³ inhalation technique with calculation by the initial slope index method (gamma-camera Toshiba CGA 202). Five patients were studied after the TGA (respectively 72, 2, 24, 40 and 5 hours after its end). CBF studies were also carried out during TGA for the first two cases: one in the middle and the other in the last third of the attack. During TGA, total CBF was decreased, with focal reduction of flow in the right temporal lobe (case 1) or in the left inferior temporo-frontal lobe (case 2). No localised or diffuse decreases were observed in CBF measured after the attacks of TGA in all five cases.

Treig *et al*³ presented reversible unilateral hypoperfusion in the mediobasal-temporal regions of three patients with TGA. In two other cases,^{4,5} PET studies during TGA showed similar and reversible abnormalities with reduction of rOEF in mesial temporal lobes.

All these data are in accordance with the assumption by Fujii *et al*¹ that "TGA is caused by reversible circulatory and/or metabolic disturbance" which leaves no permanent sequela. Our observations² confirm that TGA results from transient abnormalities, in regions responsible for the development of amnesia, such as in the temporal lobes.

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Dr Fujii *et al* reply:

We thank Drs Croisile and Trillet for their informative comments on our paper. We agree that studying regional cerebral blood flow and metabolism during both acute and chronic stages of transient global amnesia (TGA) is important. We are also interested in changes in local concentration of neurotransmitters in several acute and chronic disorders. We hope to have the opportunity to receive such information during attacks of TGA in the near future.

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Aneurysm re-rupture: Doppler evidence of first phase vasospasm?

The recent excellent demonstration of transcranial Doppler waveform changes occurring around the time of re-rupture of an intracranial aneurysm¹ suggests that the development

of zero diastolic flow coincides with aneurysm rupture. It appears more likely that rupture occurred during the early part of the Doppler trace labelled "10 seconds before rupture". This would explain the gradually declining diastolic, and to a lesser extent systolic, velocities during this trace.

These changes are not mentioned by the authors. The time course of development of intracranial circulatory arrest would become less "instant" than they imply, but would remain "immediate". The connecting Doppler trace between the first and second given would be of interest, and might show a continued decline of flow velocities—the "mirror-image" of the gradual recovery of flow velocities seen in the later traces. The changes shown may represent the first phase of so-called biphasic spasm, and if development of the recovery from this phase can occur as quickly as this (within 5–6 minutes), it is not surprising that angiography often fails to identify this process.²

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- 1 Steinmetz H, Hassler W. Reversible intracranial circulatory arrest in acute subarachnoid haemorrhage. *J Neurol Neurosurg Psychiatry* 1988;51:1355-6.
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Dr Steinmetz replies:

I agree with the helpful comment of Dr Grosset that aneurysmal rupture probably took place shortly before the point marked in our illustration.¹ The transient circulatory arrest was indeed preceded by a gradual decline of flow velocity over some seconds. However, it is unlikely that this reflects vasospasm since acute arterial narrowing should have caused at least an initial flow acceleration in an affected segment. Wilkins² reviewed arteriograms obtained during aneurysm rupture, and also failed to demonstrate acute vasospasm.

The transcranial Doppler tracings at the time of subarachnoid haemorrhage¹ corresponded to those in angiographically proven intracranial circulatory arrest.^{3,4} They match the findings of Nornes, who measured an immediate intracranial pressure increase to arterial pressure levels in ongoing subarachnoid haemorrhage.⁵ His concept of an acute brain tamponade, which is supported by our data,¹ is sufficient to explain the arrest of the bleeding. Although ensuring clot formation, this severe haemorrhagic-ischaemic event may determine the outcome in at least some patients with subarachnoid haemorrhage.

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