nerve palsy were present. Perimetry showed full visual fields and visual acuity was 6/9 in both eyes. Magnetic resonance imaging showed appreciable tumour recurrence. Overtly haemorrhagic and necrotic pituitary adenoma was identified at operation and later verified histologically. Two months after this procedure the left third nerve palsy had begun to resolve.

Haemorrhage within pituitary adenomas is a common finding either at the time of operation or on subsequent histopathological examination and is often subclinical. It has been suggested that in cases of subclinical haemorrhage, tissue necrosis is an uncommon finding whereas it is common in those presenting with clinical apoplexy. Repeated minor haemorrhage within vascular adenomas probably accounts for the clinically occult group, whereas swelling secondary to ischaemic oedema or haemorrhagic infarction in an adenoma which has undergone necrosis, is proportionately the more likely sequence of events in cases of clinical apoplexy. The second mechanism seems to have been responsible for the three cases presented here, where extensive necrosis was uniformly seen. The absence of severe headache, obtundation, and altered visual acuity accords with the finding that whereas ophthalmoplegia is commonly seen in pituitary apoplexy, defects in visual acuity are more often associated with impaired consciousness.

The mortality and morbidity of untreated classical pituitary apoplexy is high, whereas the results of expedient surgical intervention for pituitary apoplexy are generally good. Misdiagnosis of pituitary apoplexy even in its more typical presentation is common, however, and it is therefore important that those involved in the initial evaluation of patients are aware of the variability in clinical presentation and the need for early and appropriate radiological evaluation. Moreover, the syndrome combining visual field deficits and visual acuity with high diagnostic accuracy with excellent anatomical detail particularly in the coronal plane (figure).

The case for urgent surgical decompression of the acute or threatened visual field loss is well defined and many advocate conservative management in this instance. Steroid treatment and close clinical observation is certainly associated with spontaneous improvement in some cases, although there do not seem to be any reliable predictive criteria on which such patients can be identified. The clinical course is unpredictable and the possibility of rapid deterioration with blindness or death must be borne in mind and weighed against the surgical alternative. The transphenoidal approach in experienced hands is well tolerated and the surgery ensures immediate decompression of the parasellar structures and affords the opportunity to obtain tissue for histological evaluation to aid subsequent management. Extravasation of CSF into the sphenoid sinus may occur as the sole manifestation of infarction or haemorrhage in pituitary adenomas. Early recognition of this “non-apoplectic” mode of presentation allows prompt neurosurgical management, hopefully averting permanent ophthalmoplegia and the possibility of life threatening upward extension of the pituitary mass, while permitting definitive treatment of the underlying adenoma.

**Excitotoxic amino acid neurotransmitters are increased in human cerebrospinal fluid after subarachnoid haemorrhage**

Experimental evidence suggests a role for glutamate neurotoxicity in many neurodegenerative disorders, but few clinical studies have been conducted to substantiate this hypothesis. We have measured excitatory and inhibitory amino acid neurotransmitters including glutamate, aspartate, taurine, and alanine in human CSF in the acute stage of subarachnoid haemorrhage in relation to clinical outcome.

The study protocol was approved in advance by the local ethics committee and informed consent was obtained from a relative. Ten patients who had a subarachnoid haemorrhage due to a ruptured cerebral aneurysm were included in this study (table) and their ruptured aneurysm was clipped within 48 hours of ictus. A ventricular catheter was placed, CSF was intermittently drained to maintain the intracranial pressure below 20 mm Hg, and CSF samples were collected from day 3 to day 8 after the haemorrhage and for at least two days after operation. Samples were deproteinised and stored at -80°C. Analysis of amino acids in CSF samples were collected and stored in the same way from patients without neurological disorders who underwent spinal taps for spinal anaesthesia.

The CSF samples were diluted 10 times with distilled water and 30 μl aliquots were used. Amino acids were measured three times for each CSF sample by high performance liquid chromatography (HPLC) with electrochemical detector (ECD-100; EICOM, Kyoto, Japan) after a 4-6 mm (diameter) × 150 mm reverse phase pre-column (EICOM, Kyoto, Japan) and a 250 mm reverse phase pre-column (EICOM, Kromasil, Japan) derivatisation procedure with o-phthalaldehyde and ethylmercuricant. The liquid phase was 0-1 mol phosphate buffer (pH 6-0) with a 30% methanol gradient. Standards of aspartate, glutamate, taurine, and alanine in concentrations of 9, 90, and 900 pmol (WakoJuyunyu, Tokyo, Japan) were added to each sample for quantitative analysis. The retention time for glutamate, aspartate, and alanine was determined by running a full blank sample and a full calibration curve. The retention time for glutamate was 3.0 min, aspartate 3.2 min, and alanine 3.4 min.

Evaluation of the results was performed by the Student's t test.

**Letters to the Editor**

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<td><strong>Excitotoxic amino acid neurotransmitters are increased in human cerebrospinal fluid after subarachnoid haemorrhage</strong></td>
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Summary of patients with subarachnoid haemorrhage: clinical profiles, and amino acid neurotransmitter (AANT) concentrations in CSF

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<th>Day of sampling</th>
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Mean (SD) Control (n = 16): 1.9 (1.7)* 5.0 (3.6)* 8.6 (4.3) 105.3 (44) 31.9 (62).1

* p < 0.05 vs controls.

outcome requires study of a larger population of patients but our preliminary results provide a rational basis for such studies and for trials of excitoxious amino acid antagonists.

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Incidence of internal carotid artery dissection in the community of Dijon

Dissection of the cervical internal carotid artery is one of the major causes of ischaemic stroke in patients under 50 years old. Reliable epidemiological data are not numerous. Only one defined population study on internal carotid artery dissection has been published.1

We examined the occurrence of spontaneous internal carotid artery dissection in the population of the city of Dijon from 1985 to 1993, by means of the stroke Registry of Dijon2 which records all first strokes, and also asymptomatic stenosis, occlusion, and dissection of internal carotid artery occurring in the population of the city of Dijon. The diagnosis was ascertained by an ultrasonic Doppler examination of the cervical internal arteries, and a carotid angiography. Patients with a history of cervical trauma were not included. For the calculation of incidence, the whole population of Dijon was considered to be at risk, with 135,000 inhabitants in 1985 and 150,000 inhabitants in 1992, and was used as the denominator for the study.

From 1985 to 1993, 36 internal carotid artery dissections were diagnosed. The average annual incidence for all age groups was 2.5 per 100,000 population (95% confidence interval 1.9–3.9). There were no statistically significant annual variations (three cases in 1985, four in 1986, three in 1987, five in 1988, four in 1989, five in 1990, three in 1991, four in 1992, five in 1993). The mean age was 39.9 (7–5) years for the 21 women, 43.7 (5–7) years for the 15 men. All had headache or neck pain; 19 patients presented with cerebral ischaemic symptoms, four with retinal ischaemic symptoms; one patient had had a subarachnoid haemorrhage. Occlussylpatropic palsy was noted in 12 patients, and 12th cranial nerve palsy in one. Isolated headache was the single symptom in three cases (8%). The diagnosis of dissection was suspected with ultrasound in 34 cases (with demonstration of a double lumen in eight cases), and proved with angiography in all cases, with MRI in only the last four patients. No necropsy cases of spontaneous cervical internal carotid artery dissection were found during this period. No bilateral cervical internal carotid artery dissections, or associated vertebral artery dissection were found. Arterial hypertension was seen in 19 cases. Dysplastic cervical or retinal arteries were found in 15 cases. Association with tobacco and pill consumption was present in 15 out of 21 female cases. Evolution was marked by slight neurological sequelae in four cases. Headache resolved in all patients. Recurrent arterial dissections were not found at follow up ranging in the three months to nine years.

These 36 cases represent 2% of 1784 patients with stroke collected in this population of Dijon and 10% of the 356 stroke patients under 50 years old. This confirms that internal carotid artery dissection is a major cause of cerebral infarction in those under 50 years old.1

The average annual incidence of spontaneous internal carotid artery dissection for all age groups was 2.5 per 100,000 inhabitants, similar to that in the city of Rochester.1 This compares with 2.2 per 100,000 for aneurysmal subarachnoid haemorrhage in Dijon.3

Before ultrasound was introduced many cases remained undiagnosed. Biller et al.1 reported a spontaneous cervical internal carotid artery dissection in 0.3% of 4531 patients with acute stroke and Bogousslavsky et al.4 reported dissection in 2.5% of 1200 patients with acute stroke. A community based study in Rochester, Minnesota, 1987–92 gave a similar incidence of 2.5 per 100,000.1

Headache, ischaemic cerebrovascular disease, and oculussylpatropic palsy were the most common manifestations of spontaneous internal carotid artery dissection. Some internal carotid artery dissections do not present with ischaemic stroke (10% in the study of Shievink et al.4) in the present study.

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