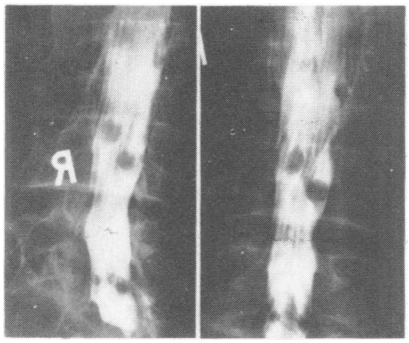

Figure *Metrizamide radiculogram showing numerous rounded filling defects in close association with the roots of the cauda equina.*

months prior to presentation. Hearing in the right ear had deteriorated in association with progressive double vision on looking to the right as well as intermittent right sided facial paraesthesae. Initial examination revealed a partial 3rd and complete 6th and 8th cranial nerve lesions on the right, in addition to a mild pyramidal distribution paraparesis with bilateral extensor plantar responses. Sensation in the right leg was diminished to all sensory modalities. Investigations at that time including an enhanced CT scan were unremarkable apart from an elevated CSF protein of 4·83 g/l which contained no malignant cells. A clinical diagnosis of an intrinsic brain stem glioma was made, and he was treated with prednisolone for four weeks. The patient continued to deteriorate to the point where he was wheelchair bound and had developed a complete right ptosis. On readmission the principal new findings were the complete loss of all lower limb reflexes and a sensory level to pin prick in the lower dorsal region. A repeat CT scan revealed obstructive hydrocephalus but no other definite pathology even after contrast enhancement. Two further CSF examinations failed to detect malignant cells but a metrizamide myelogram showed multiple intradural, extramedullary, rounded lesions, in close association with the roots of the cauda equina. These were thought initially to be metastases or neurofibromata (fig). At this stage, a review of the 3rd CT scan revealed a small area of pathological enhancement in the vermis, indenting the roof of the 4th ventricle. Therefore a posterior fossa exploration was performed, which revealed numerous black streaks on the arachnoid, biopsies of which confirmed a diagnosis of malignant melanomatosis. Tumour mass was seen on the surface of the right cerebellar hemisphere. Despite careful fundoscopy and clinical examination no evidence of other melanotic lesions were found. Melanuria was not detected. The patient died two months later despite craniospinal irradiation. There was no post mortem examination.

It is likely that this tumour originated in the posterior fossa leptomeninges and spread into the substance of the brain stem and the cerebellum as well as via the CSF to the cauda equina. It is surprising that despite clear evidence of multiple meningeal lesions and of supposed CSF tumour spread, that repeated examinations of the CSF for malignant cells were negative. Although in isolated cases melanoma cells have been detected in CSF,[8] no information is available on the detection rate, as has been published for gliomas and secondary carcinomatisis.[9] "Coal black" CSF was obtained in one case of spinal cord primary melanoma. Post mortem examination of this case revealed numerous tumour nodules throughout the cauda equina region.[10] Abnormal CT findings have been reported in four cases of primary leptomeningeal melanomatisis.[6 11 12] In three cases dense meningeal enhancement was reported, whilst in the fourth case CT revealed a low density frontal lesion without meningeal enhancement despite subsequent surgical evidence of melanotic meningeal invasion.[12] In the present case, in spite of extensive posterior fossa meningeal involvement with tumour, only a small area of contrast enhancement was seen in the vermis on review of the final CT scan.

The diffuse pattern of meningeal melanomatosis reported in the current case and seen in various other cases of primary nervous system melanomas is in sharp contrast to that found in metastatic melanomata. In a recent series of 33 cases of cerebral metastatic melanomatosis, 20 showed a solitary space occupying lesion, 12 multiple lesions and in only one case was diffuse meningeal disease observed.[13] When melanomata exclusive to the nervous system present with spinal cord or root symptoms an early preoperative diagnosis is usually made.[3] However, intracranial presentations as in our case are difficult to diagnose and often remain undiagnosed premortem[1-2] despite modern techniques.

We thank Dr JN Blau for permission to report this case.

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Subarachnoid haemorrhage and nasal vasoconstrictor abuse.

Sir: Recently, several arterial abnormalities attributed to abuse of nasal sympathomimetic drugs have been described. Fendler *et al*[1] reported the case of a woman with arteriographic changes in renal arteries and severe hypertension after taking fenoxazoline HCL in nasal spray for

five years. Dezellus *et al*[2] observed a patient with obstruction of the arteries of the lower limbs after daily use of nasal descongestants for seven years. We present a woman with subarachnoid haemorrhage and irregularities of the intracranial vessels probably induced by chronic abuse of nasal vasoconstrictors.

A 26-year-old woman was hospitalised because of stupor, aphasia, and right hemiparesis. Three days before she had had palpitations, followed shortly by sudden occipital headache, mixed aphasia, mental confusion and difficulty in moving her right arm and leg. Since her adolescence the patient had suffered occasional attacks of migraine but had never taken ergotamine compounds. Five years before admission "sinusitis" was diagnosed and she started to take increasing doses of xylomethazoline HCL in nasal spray. In the last two years inhalations took place every hour and a 10 mg bottle was consumed every 5 to 6 days. On admission the patient showed nuchal rigidity. Fundi were normal. The blood pressure was 95/60 mm Hg, pulse rate 55/min and temperature 36·5°C. There was no evidence of external trauma. The CT scan was normal. Atraumatic lumbar puncture released uniformly blood-stained cerebrospinal fluid, with xanthochromic supernatant. The opening pressure was 200 mm H_2O. Three days later the patient was alert and no focal signs were elicited. On the seventh day the headache subsided and the patient was asymptomatic.

Transfemoral four-vessel cerebral angiography was carried out on the ninth day after admission. Partial and irregular narrowing of the vessels alternated with mild dilatations. Alterations were multifocal and more marked on the first segments of both anterior cerebral arteries and the vertebrobasilar system. There was no vessel size predilection. The carotid arteries were normal. No sacular aneurysm was discovered. Cerebral circulation time was normal. Haematocrit, haemoglobin, white blood cell count, differential and platelet count were all normal. The coagulation profile was normal. Sodium, potassium, chloride, CO_2, calcium, phosphorus, total protein, albumin, total bilirubin, alkaline phosphatase, lactic dehydrogenase, creatine kinase, uric acid, blood sugar, VDRL, FTA and antinuclear antibody were all normal. Electrocardiogram and echocardiogram were unremarkable. The patient has remained asymptomatic save occasional menstrual headaches during the following 18 months.

No case of cerebral arteriopathy or subarachnoid haemorrhages associated with chronic abuse of nasal vasoconstrictors has been reported previously. That a nasal sympathomimetic is the cause of this syndrome is only speculative, but is strongly suggested by comparing our case with others closely related. In the report of Fendler *et al*[1] the patient developed arteriographic stenosis and dilatations of renal arteries which disappeared after the drug was discontinued. In the case reported by Dezellus *et al*[2] with occlusion of the arteries of the lower limbs ("Buerger type"), the trophic injuries and the intermittent claudication subsided when the use of fenoxazoline HCL nasal spray was discontinued. Only exceptionally has arterial beading been reported as one type of cerebral vascular spasm in subarachnoid haemorrhage.[3] In view of the clinical condition of the patient when arteriography was carried out and the widespread arteriopathy it is unlikely that the arterial abnormalities in our case were caused by subarachnoid haemorrhage alone. The recurrent migraine may be an additional factor.

Our case is comparable to the cerebral arterial beading and intracranial haemorrhage in the amphetamine abuse patients.[4] Pathologically, this sympathomimetic drug could induce necrotising angiitis indistinguishable from polyarteritis nodosa.[5] The anomalies of our case could be explained as a complication of chronic vasospasm. Myonecrosis in vessel walls has been described in experimental catecholamine-induced chronic cerebral vasospasm.[6] If our observation is confirmed, a more restricted policy in the use of nasal descongestants should be recommended.

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Juvenile transient global amnesia

Sir: Transient global amnesia was originally described by Fisher and Adams as a prolonged memory loss of undetermined cause.[1] It is characterised by sudden onset of amnesia lasting less than 24 hours with awareness both of identity and predicament during the attack. Retrograde amnesia subsides when the attack ends but there is persistent amnesia for the period of the attack. Cases with head injury, epilepsy, psychiatric illness or progressive memory loss are excluded from the diagnosis.[2] We report here a case of transient global amnesia in a 16-year-old boy.

A 16-year-old boy was brought to the hospital having had memory loss of abrupt onset and lasting nine hours. The boy set out on an all-night hike with scouts on a mild though wet evening and was wearing seven layers of clothing including a nylon anorak. His behaviour was completely normal until midnight when, after walking two miles, the party stopped for a rest. He was perspiring excessively and his inner clothing was wet with sweat so he took off the anorak and sat resting by the road-side for 25 minutes. During this time he became cold and started shivering. He was noted to be unusually quiet though he responded appropriately to questions. When the party moved off again he continued to complain of the cold. Though rational and well orientated in conversation he seemed reluctant to continue walking and at 3 am he was taken by car to a friend's home where he fell asleep. He awoke at 6 am when his conversation was again normal except that he was asking about a school friend whom he should not have expected to be present on the hike. He fell asleep once more waking at 9 am whereupon he had no recollection of any event after halting for the road-side rest at midnight and this amnesia has persisted. There was no history of head injury or alcohol or drug abuse and no past history or family history of migraine or epilepsy. On examination he appeared fit