five years. Dezellus *et al*² observed a patient with obstruction of the arteries of the lower limbs after daily use of nasal descongents 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. A traumatic lumbar puncture released uniformly blood-stained cerebrospinal fluid, with xanthochromic supernatant. The opening pressure was 200 mm H₂O. 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 saccular 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, CO₂, 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 descongents 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

and well and there were no abnormal neurological signs. Serum biochemistry and electroencephalogram were normal as was computed tomography of the head. There has been no recurrence during three months follow-up.

The clinical history, substantiated by several witnesses, resembles the cases of transient global amnesia as described by Fisher¹ and Shuping² but only once reported in childhood.³ Whilst drowsiness is not usually a feature it was nevertheless in keeping with the time of onset of the attack in this case and he was easily aroused from sleep. Temporal lobe ischaemia resulting from vascular disease has been proposed as the mechanism of transient global amnesia⁴⁻⁶ but this patient's age and normal physical examination make vascular disease unlikely. It has been proposed that in younger patients transient global amnesia could be a manifestation of migraine with bilateral temporal lobe ischaemia³ but in the present case there was no history of headaches and no family history of migraine. Exposure to the cold, and in particular immersion in cold water, has provoked attacks of transient global amnesia in adults.^{2,7} The mechanism by which cold exposure can produce a disorder of temporal lobe function is unknown. As transient global amnesia may follow painful stimuli, emotional stress and sexual intercourse it has been suggested that the disorder is caused by a focal hippocampal seizure triggered by an essentially emotional event.⁷ However, amnesia has also been produced in rats and mice by sudden immersion in ice cold water^{8,9} suggesting a more direct physiological mechanism. In the present case it seems likely that considerable cooling occurred when the patient removed his anorak and sat resting with his inner clothing wet with sweat and this cooling may have been the provoking factor.

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