than "fantastic" in nature. Formal psychological testing revealed a 35 point discrepancy between verbal and performance IQ (vIQ = 131, pIQ = 96). Premorbid IQ (NART) was estimated at 121, thus indicating a decline in general intellectual ability. Recognition memory for words (39/50) and for faces (30/50) was considerably impaired. Performance on an easy recognition memory test for pictures was weak (Camden A 26/30) while on a similarly difficult test performance was at a chance level (Camden B 12/30). There was some evidence of a nominal dysphasia (McKenna 19/30).

Laboratory investigations showed a normal full blood count (with a MCV of 88), red cell transketolase, U + E + creat., LFTs and gamma GTs, B12 and folate, Iron studies, TFTs and VDRL. CSF analysis revealed 17 white blood cells (99% lymphocytes) with a protein of 0·57 mg/l. Oligoclonal bands were present. An EEG showed a low amplitude record with no other abnormal features, and CT of the brain was consistent with a moderate degree of generalised cerebral atrophy. MRI of the brain demonstrated extensive periventricular high signal change on T2 weighted sequences. Multiple high signal foci were seen in the deep white matter of both hemispheres and the overall picture was that of demyelination. Visual evoked responses were abnormal on the left, showing an anterior visual pathway lesion also compatible with demyelination. Sensory evoked potentials were likewise abnormal.

A dual diagnosis of alcohol related cognitive impairment and definite multiple sclerosis was made and a course of IV Parentrovite given. The patient was transferred to his local psychiatric hospital with the recommendation that abstinence from alcohol should be enforced as far as possible.

The patient presented with a profound memory deficit together with a more widespread decline in cognitive abilities as evidenced by a substantial drop in IQ and mild nominal dysphasia. In addition, the history of neurological disability and the results of the investigations made it possible to diagnose multiple sclerosis.

The cognitive abnormalities found in this patient are in many ways similar to those described by Cutting[1] in patients with slow onset Korsakoff's or alcoholic dementia, in whom memory difficulties occur on a background of more widespread cognitive deterioration. Alcohol abuse and associated nutritional deficiencies are considered to be the cause of this picture without invoking the presence of additional aetiological factors. Recently Lishman[2] has put forward a hypothesis that the Wernicke-Korsakoff pathology may also be responsible for a primary alcoholic dementia by its involvement with certain key forebrain nuclei, (the nucleus basalis of Meynert), thus compromising acetylcholine production and producing a clinical picture similar to Alzheimer's disease.

Our patient, on the other hand, had evidence of dysphasia and a very large verbal-performance discrepancy (WAIS) not often found in Korsakoff's syndrome. Similar findings emerged in a recent study by Jacobson and Lishman[3] in which only two out of 38 Korsakoff patients had both the severe memory and intellectual decline exhibited by our patient, and whose verbal-performance discrepancies on IQ (namely 31 and 33 respectively) matched those of our patient (Jacobson, Lishman, personal communication). Thus the possibility remains that some of the cognitive deficits present in our patient were the result of demyelination rather than alcohol related pathology. When compared with normal subjects matched for age and education, multiple sclerosis patients have been found to have significantly lower IQ's.[4] Grant *et al*[5] in a study of 43 patients with multiple sclerosis, most of whom were in the early or middle phases of the disease, found memory to be impaired in a sizeable proportion of cases, and came to the conclusion that memory loss may be prevalent early on in the natural history of the disease. In both Korsakoff's syndrome[1] and multiple sclerosis[6] verbal IQs decline more than performance IQs and it could be argued that the combination of multiple sclerosis and Korsakoff's syndrome in the same patient may have contributed to the large discrepancy between the two IQ subsets, and to the unusual cognitive findings in general. However, even if multiple sclerosis is likely to have been significant, it is unlikely to explain the bulk of the deficits, as it is uncommon for such a profound degree of abnormality to be present in multiple sclerosis patients without severe neurological impairment, although cases of rapidly progressive dementia have been described.[7]

In summary, this case presented with a number of interesting features. Theoretically, alcohol alone could have accounted for the clinical and psychometric findings. However, the atypical findings and the patient's patchy recall of a previously suspicious neurological deficit, prompted careful clinical and laboratory investigation that unearthed a second significant pathology. This highlights the importance of looking for other pathologies in patients with alcohol related brain damage (and perhaps in multiple sclerosis also), if the presentation or clinical picture is in any way unusual.

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Accepted 26 August 1988

Cerebral infarction after excessive use of nasal decongestants

Sir: Sympathomimetic agents are frequently used as anoretics, nasal decongestants and cold preparations. Neurological side effects have included seizures,[1-2] cerebral haemorrhage[3] and neuropsychiatric symptoms.[4-6] Two cases of cerebral infarction following prolonged use of oral phenylpropanolamine[7] (PPA) and one case after a single dose of PPA[8] have been reported. We report two patients with stroke presumably due to prolonged use of nasal decongestants.

A 35 year old white man presented to the emergency department complaining of sudden speech disturbances and weakness of the right arm and leg. His past history revealed a chronic allergic rhinitis. He had used a total of 15 mg of oxymethazoline every 3 days in nasal decongestants for 20 years. He was taking no other medication, and had no

history of migraine, hypertension, diabetes or cardiovascular disease. He was not a smoker and drank alcohol infrequently. At the time of hospital admission, neurological findings included the following: right facial, arm and leg paresis, right Babinski's sign and a moderate right hemisensory loss. His speech was non-fluent; he was not able to repeat short phrases or name objects. Comprehension was intact. There were no other neurological findings. His general examination was normal. Blood pressure was 110/70 mm Hg. The following examinations were either normal or negative: complete blood count, coagulation screening tests, sodium, potassium, renal and hepatic biochemical tests, total protein and serum electrophoresis, total cholesterol, HDL, LDL, triglicerides, lipidogram, serum glucose, anticardiolipin, antinuclear and anti-DNA antibodies, cryoglobulins, hepatitis B surface antigen, complement levels, RPR and FTA-ABS tests, chest radiograph, electrocardiogram and echocardiogram. Cerebrospinal fluid examination revealed no abnormalities. CT of the brain showed an ischaemic infarct in the middle cerebral artery territory. A conventional arteriogram showed a thrombotic occlusion of the left Silvian artery.

The second case, a 40 year old white man was admitted to the hospital because of a sudden onset of speech disorder and weakness of the right arm and leg. His past history revealed a chronic allergic rhinitis. He had taken a total of 10 mg of Phenoxazoline every 5 days in nasal decongestants for 15 years. He was taking no other medication, and had no history of migraine, hypertension, diabetes or cardiovascular disease. He did not smoke or drink alcohol. On examination he appeared well. Blood pressure was 120/75 mm Hg. A left cervical bruit was heard. The remainder of his general examination was unremarkable. His speech was non-fluent. He was not able to repeat simple phrases and had mild difficulty with object naming. Other significant findings included mild right facial, arm and leg paresis, brisk right muscle stretch reflexes, and an extensor plantar response on the right. The following examinations were either normal or negative: complete blood count, coagulation screening tests, renal and hepatic biochemical tests, total protein and serum electrophoresis, total cholesterol, HDL, LDL, triglicerides, lipidogram, serum glucose, anticardiolipin, antinuclear and anti-DNA antibodies, cryoglobulins, hepatitis B surface antigen, complement levels, RPR and FTA-ABS tests, chest radiograph, electrocardiogram and echocardiogram. Cerebrospinal fluid was normal. CT of the brain showed an ischaemic infarct in the middle cerebral artery territory. Digital intravenous substraction arteriogram showed an occluded left internal carotid artery at the level of the bifurcation.

Phenoxazoline and oxymetazoline are sympathomimetic agents belonging to the group of imidazolines with alfa-receptor activity. Therapeutically, they are used in nasal spray or drops for the treatment of allergic rhinitis and as vasoconstrictive eye-drops. The local nasal application of these drugs has been reported to have cardiovascular or central nervous system untoward effects.[6 9-10] There have also been reports of the association between oral PPA and brain infarction[7-8] and myocardial infarction after intravenous amphetamine administration[11] and oral PPA.[12]

These ischaemic complications have been attributed to three mechanisms: (1) the production of a necrotising angeitis,[4 5] (2) the development of a vascular spasm[7] and hypertension.[13-15]

Johnson *et al*[7] postulated vasospasm as a possible mechanism in two patients with cerebral infarction that developed after chronic use of PPA but in whom there was neither hypertension nor demonstrable angiographic abnormality. Edwards *et al*[8] postulated an idiosyncratic, transient episode of accelerated hypertension that produced a lacunar infarction in the distribution of the left lenticulostriate arteries in a patient without hypertension on admission or any angiographically demonstrable abnormality. In our second case, arteriography showed a complete occlusion in the left internal carotid artery at the level of bifurcation. This is one of the angiographic findings in extracranial occlusive disease related to atherosclerosis. Our patient had no risk factors for atherosclerosis: hypertension, smoking, diabetes mellitus, hyperlipidaemia, coronary artery disease, previous transient ischaemic attacks or leg claudication. Nevertheless it has been reported in young patients without known risk factors for cerebral atherosclerosis who had typical lesions found on arteriography.[16] In the first case arteriography showed an occlusion of the left Silvian artery; there were no other angiographic signs suggestive of atherosclerosis. Emboli are more common in the middle cerebral artery distribution, and are more common in the left than in the right hemisphere. This patient had no risk factors for atherosclerosis or potential source of emboli.

Magargal *et al* described in 1985 the first case of thrombosis of one of the branches of the retinal artery in a young patient with no other past medical history than the chronic abuse of a nasal spray with oxymetazoline. They found an increase platelet aggregation with ADP and epinephrine and suggested that the alpha-adrenergic agent was the cause.[17]

Our two patients may constitute examples of such pathogenic mechanism. Irrespective of the underlying pathophysiology, the timing, the relationship between exposure and reaction and the exclusion of alternative aetiologies[18] make us suspect a relationship between chronic use of sympathomimetic agents by the nasal route and the ischaemic complications presented by these two patients.

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Calcitonin for pseudoclaudication in lumbar spinal stenosis

Sir: "Pseudoclaudication" or intermittent claudication of the cauda equina is closely related to lumbar spinal stenosis (LSS), for which the only known effective treatment is surgical decompression by wide laminectomy (averaging three vertebrae), with an optimum success rate of 66 to 85%.[1,2]

The pathogenesis is still controversial; however, many consider vascular insufficiency of the nerve roots as a main causative mechanism.[1,3] The observed beneficial effects on pseudoclaudication in patients with Paget's disease of bone, who have been treated with calcitonin,[4] raises the possibility that this hormone may be useful in pseudoclaudication in non-Paget's patients, owing to its effectiveness in lowering the metabolic activity of the bone and, consequently, its blood supply.[5,6] Such a mechanism may permit more blood to reach neural tissues.

A single clinical trial using calcitonin for the treatment of pseudoclaudication in non-Paget's LSS patients showed promising results.[3]

Based on these data, we administered calcitonin to six patients suffering from neural pseudoclaudication. The diagnosis of LSS was considered when the patient had a combination of pseudoclaudication, unlimited "bicycling" ability while supine, no signs of peripheral vascular disease, and the demonstration of lumbar spinal canal stenosis on spinal CT scan and/or myelography. The aetiology of LSS was either osteoarthritic-spondylotic or combined with discopathy.

All patients were manual workers. Their mean age was 63 years and their chief complaint was intermittent claudication when walking. Duration of symptoms was 4–8 months in three patients and 3–10 years in the others.

Other complaints were low back pain, paraesthesia in one or both legs, muscle cramps, standing discomfort, and in two patients, episodes of urinary incontinence.

A variety of analgesics and different modes of physical therapy were tried and considered not helpful.

Neurological examination revealed decreased or absent ankle reflexes in five patients, decreased or absent patellar reflexes in two patients, weakness of one leg in three patients and positive straight leg raising in one.

Clinical and electromyographic evidence of peripheral neuropathy was present in four patients. Impaired glucose tolerance test was found in two patients and decreased serum vitamin B-12 in one.

Each patient was treated with intramuscular injections of Salmon calcitonin (Miacalcic, Sandoz) 100 I.U., 4 times a week for 4 weeks.

Each patient was asked to determine his comfortable walking distance by measuring the distance he was able to walk, at the same time of day, along a certain route at a steady pace. The patients documented their walking ability several days before treatment was started, during the second week, and on the last few days of each treatment period.

Clinical assessment was performed by one of the authors (J.S.) before and after each treatment period. Results of treatment and follow-up are summarised in the table which documents the time that initial and maximal response was reached in each patient as well as maximal walking distance. Since most of the patients needed more than one course of calcitonin injections, the time of repeated treatment periods is also shown (this is indicated in months after the first treatment course).

This unselected group of elderly patients, who suffered from symptoms due to moderately severe LSS, were also suffering from chronic diseases typical for this age group, such as diabetes mellitus, atherosclerotic cardiovascular diseases. However their poor quality of daily life was determined by their LSS. All patients refused surgical intervention but agreed to participate in the clinical trial with intramuscular calcitonin.

The response to calcitonin was favourable in five out of six patients. Four patients showed a rapid initial response. The only non-responding patient suffered from severe LSS with complete myelographic block at L_{3-4} level. After the "failure" of the medical approach he agreed finally to be operated on and benefited from improvement following surgery.

Surprisingly, four of our patients suffered concomitantly from peripheral neuropathy due to different causes. Their "neuropathic" symptoms and conduction time were not altered by calcitonin.

Table *Therapeutic effects of calcitonin in LSS patients*

Patient No.	*Age*	*Walking distance*		*Initial Response (days)*	*Maximal Response (days)*	*Repeated Courses of Calcitonin (months)*
		Before Treatment (metres)	*After Calcitonin (metres)*			
1	70	10	3000	10	30	11,22
2	55	500	1500	8	120	3
3	64	20–30	500	28	90	3
4	64	100–300	1500–5000	8	30	—
5	50	30	30	8(temp)	—	—*
6	74	50–100	400	10	30	3

*referred for surgery.