Letters

Cerebral infarction on CT scans in patients with transient ischaemic attacks relates to severity of angiographic disease at the carotid bifurcation

Sir: The current reappraisal of the effectiveness of carotid endarterectomy in the management of patients with transient ischaemic attacks (TIAs) associated with carotid stenosis has highlighted a controversy over the relevance of the severity of vessel narrowing. Physicians differ widely in their approach to the management of visible atheromatous change in the carotid artery, particularly when the lesion has produced a stenosis of less than 60%.

Until the prospective data from the European Carotid Endarterectomy trial, or that beginning in North America, become available, the evidence on the pathological importance of the severity of bifurcation disease has to be more indirect.

We considered that a study of the incidence of "silent" cerebral infarction as revealed by CT scanning in patients with TIAs without residual deficit whose angiograms showed different degrees of arterial wall disease might be of value. Previous publications have included patients with residual deficit,^{1,2} or have been based on Doppler studies.³ None of our patients in this study had had a previous stroke.

The CT scans and angiograms of 69 patients with TIAs were compared. CT scans were noted to show infarcts, or atrophy or to be normal. Angiograms were characterised as showing a normal bifurcation, wall irregularity, stenosis with narrowing of the lumen of over 20% or occlusion at the carotid bifurcation. The degree of stenosis was calculated by comparing the residual lumen with the widest part of the carotid bulb.

The results are shown in the table, from which it can be seen that the results of scanning and the severity of atheromatous changes at the carotid bifurcation are related (Chi square 18.2, $p = 0.006$).

Infarction was commonest with carotid occlusion (Chi square 11.3, $p < 0.001$). Amongst those with stenosis there was evidence that infarction was commoner with severe stenosis ($\geq 90\%$) than with moderate stenosis ($\leq 75\%$). Thus two of the seven patients with severe stenosis had infarcts on their CT scans whilst none of the five with moderate stenosis did. Overall seven of the 17 patients with tight stenosis or occlusion had CT scan evidence of infarction whilst only three of the remaining 50 did (Chi square 12.4, $p < 0.001$). There was no evidence of a relationship between extent of arterial disease and presence or absence of cerebral atrophy on the scans (Chi square 3.0, $p = 0.59$), although atrophy was rarest with normal angiograms.

These findings show that infarction in TIA patients is commonest with severe atheromatous disease. This perhaps supports a conservative approach towards lesser degrees of stenosis and plaque formation. No evidence was found of the reported paradoxical protection from atrophy with severe carotid narrowing.³

There is some evidence of impaired autoregulation in the presence of carotid occlusion or tight stenosis,⁴ and in some patients of impaired vasodilator reserve.⁵ This suggests that the cause for infarction in these patients may relate to haemodynamic effects. Perhaps the hemisphere is more vulnerable to the effects of embolism if its haemodynamic reserve is impaired. If so, might such patients have the best rationale for endarterectomy or revascularisation?

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References

- 1 Perrone P, Candelise L, Scotti G, De Grandi C, Scialfa G. CT evaluation of patients with

transient ischaemic attacks: correlation with clinical and angiographic findings. *Eur Neurol* 1979;18:217-21.

- 2 Goldenberg G, Reisner T. Angiographic findings in relation to clinical course and results of computed tomography in cerebrovascular disease. *Eur Neurol* 1983;22:124-30.

- 3 Nicolaidis AN, Papadakis K, Grigg M, Al-Katoubi A, Williams MA, Deacon DFS. The significance of silent cerebral infarction and atrophy. In: Bernstein EF, ed. *Amaurosis Fugax*. New York: Springer Verlag, 1987 (in press).

- 4 Mendelow AD, Graham DI, Tour UI, Fitch W. The haemodynamic effects of internal carotid artery stenosis and occlusion. *J Neurosurg* 1987;66:755-63.

- 5 Gibbs JM, Wise RJ, Leenders KL, Herold S, Frackowiak RSJ, Jones T. Cerebral haemodynamics in occlusive carotid artery disease. *Lancet* 1985;i:933-4.

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Subarachnoid haemorrhage from spinal tumour (in the absence of spinal symptoms or signs)

Sir: Spinal tumour is an uncommon cause of subarachnoid haemorrhage and should be considered particularly when cerebral angiographic studies are negative and spinal symptoms and signs are present.¹ Cases have been reported where the predominant symptoms and signs suggest an intracranial rather than spinal origin of haemorrhage.²⁻⁴ We report a case with recurrent subarachnoid haemorrhage from spinal tumour in the absence of spinal symptoms or signs; this has not previously been described.

A 31 year old man had sudden onset of severe occipital headache, photophobia and vomiting. These symptoms persisted for 4 days. In the 2 years preceding this illness he had had 6 episodes of throbbing occipital headache of sudden onset which were less severe, of shorter duration and were unaccompanied by vomiting. On examination he was alert and co-operative. He had neck stiffness and a positive Kernig's sign. There were no other abnormal physical signs. CT head scan showed minimal dilatation of the lateral ventricles. At lumbar puncture, pressure was elevated (22 cm), the cerebrospinal fluid (CSF) uniformly blood-stained with xanthochromia. Four-vessel carotid and vertebral angiography was normal. He made an uncomplicated recovery and was discharged from hospital 3 weeks after admission.

Table Comparison of angiography and CT scans in patients with TIAs

Angiography Carotid bifurcation	n	CT scan		
		Atrophy	Infarction	Neither
Normal	22	4	1	17
Irregular	23	9	2	12
Stenosis	12	3	2	7
Occlusion	10	4	5	1
Total	67	20	10	37

Three months later he presented again with severe, throbbing, occipital headache of gradual onset over 7 days, and vomiting. He had neck stiffness, Kernig's sign was negative, physical examination was normal.

Fig Lumbar myelogram showing an intradural tumour at L3/4.

CT scan was normal. Lumbar puncture showed uniformly bloodstained CSF with xanthochromia. In view of recurrent haemorrhage and after careful review of the previous negative angiographic studies, despite the absence of any spinal symptoms or signs, myelography was considered to be a necessary investigation. This defined a round, intradural tumour at the level of the 3rd and 4th lumbar vertebrae (fig 1). At laminectomy a tumour was removed. Histology showed this to be an angioblastic meningioma with associated haemosiderin pigment, indicative of previous haemorrhage. The patient remains well, when reviewed 6 months after operation.

In the investigation of subarachnoid haemorrhage the cause may not be determined in between 21% and 43% of cases. When blood is seen on CT scanning predominantly in the mesencephalic cisterns rather than the basal cisterns and when four-vessel cerebral angiography is normal, a benign mesencephalic type of haemorrhage should be suspected. Bleeding in such cases is likely to be of venous or capillary origin rather than arterial. It is believed approximately 50% of cases of subarachnoid haemorrhage with normal four-vessel angiography are of this type, and the prognosis is good.³

Spinal subarachnoid haemorrhage is uncommon and is reported in only 0.05% to 0.6% of patients with subarachnoid haemorrhage.^{2,6} A spinal subarachnoid haemorrhage is usually due to spinal arteriovenous malformation (AVM).^{7,8} Fifty-six cases of spontaneous subarachnoid haemorrhage from spinal tumour have been recorded in the literature. Most tumours have been in the cauda equina (46 cases), 33 of which have been ependymoma, and eight neuroma or neurofibroma.^{2-4,8-12} Spinal subarachnoid haemorrhage usually presents with severe back pain (le coup de poignard of Michon^{13,14}) and root pain at the level of the tumour or AVM. Frequently there is weakness and loss of tendon reflexes in the lower limbs. Haemorrhage from both spinal tumour and arteriovenous malformation often occurs in association with physical effort, and false localising intracranial signs and hydrocephalus may occur.^{2-4,8}

The case presented is unusual and important since there was no back pain or root pain consequent on 2 episodes of subarachnoid haemorrhage. It is uncommon for spinal meningioma to bleed and we have only been able to find two previous cases in the literature.^{14,15} In one of these the haemorrhage caused sudden headache, but in the second case the headache was progressive over a period of 2 weeks. In both

there was a history of severe low back pain.

Spinal tumour is an uncommon cause of subarachnoid haemorrhage but should be suspected when cerebral angiographic studies are normal and when spinal symptoms or signs are present. CT head scan may or may not show evidence of blood. When blood is seen predominantly in the mesencephalic rather than the basal cisterns then a benign mesencephalic haemorrhage should be suspected. In other circumstances a spinal subarachnoid haemorrhage should be considered and if haemorrhage is recurrent investigated even in the absence of spinal symptoms or signs.

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References

- 1 Brismar J, Sunbarg G. Subarachnoid haemorrhage of unknown origin; prognosis and prognostic factors. *J Neurosurg* 1983;58:349-54.
- 2 Halpern J, Feldman S, Peysere E. Subarachnoid haemorrhage with papilloedema due to spinal neurofibroma. *Arch Neurol Psychiatr* 1958;79:138-41.
- 3 Luxon LM, Harrison MJ. Subarachnoid haemorrhage and papilloedema due to cervical neurilemmoma. *J Neurosurg* 1978;48:1015-8.
- 4 Iob I, Andrioli GC, Rigobello L, Salar G. An unusual onset of spinal cord tumour: subarachnoid haemorrhage and papilloedema. *Neurochirurgia (Stuttg)* 1980;23:112-6.
- 5 Van Gijn J, Van Dongen KJ, Vermeulen M, Hijdra A. Perimesencephalic haemorrhage: non-aneurysmal and benign form of subarachnoid haemorrhage. *Neurology* 1985;35:493-7.
- 6 Sahs AL, Peret GE, Locksley HB, Nishioka H. *Intracranial Aneurysms and Subarachnoid Haemorrhage*. Philadelphia: JB Lippincott, 1969.
- 7 Henson RA, Croft PB. Spontaneous spinal subarachnoid haemorrhage. *Q J Med* 1956;25:53-66.
- 8 Djindjian M, Djindjian R, Houdart RM, Hurth M. Subarachnoid haemorrhage due to intraspinal tumours. *Surg Neurol* 1978;9:223-9.

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- 9 Kormos RL, Tucker WS, Bilbao JM, Gladstone RM, Bass AG. Subarachnoid haemorrhage due to a spinal cord haemangioblastoma: case report. *Neurosurgery* 1980;6:657-60.
- 10 Muhtaroglu U, Strenge H. Rezidivierende subarachnoidalblutung bei spinalem Neurinom. *Neurochirurgia (Stuttg)* 1980; 23:151-5.
- 11 Heimberger K, Schnaberth G, Koos W, Pendl G, Auff E. Spinal cavernous haemangioma (intradural-extramedullary) underlying repeated subarachnoid haemorrhage. *J Neurol* 1982;22:289-93.
- 12 De-Divitiis E, Mairuri F, Corriero G, Donzelli R. Subarachnoid haemorrhage due to a spinal neurinoma. *Surg Neurol* 1985;24: 187-90.
- 13 Michon P. Le coup de poignard rachidien. Essai sur les haemorrhagies meningees spinales. *Press Med* 1928;36:964-6.
- 14 Nassar SI, Correl JL. Subarachnoid haemorrhage due to spinal cord tumours. *Neurology* 1968;18:87-94.
- 15 Roger H, Paillas JE, Duplay J. Hemorragie meningee spino-cerebrale revelatrice d'une tumeur de la queue de cheval chez deux jeunes sujets. *Bull Mem Soc Med Hop* 1949;65:37-40.

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Operculum syndrome from brain abscess in a left-hander

Sir: The operculum syndrome is a cortical type of pseudo-bulbar paralysis resulting from lesions in the opercula of both hemispheres.¹ An ill-understood unilateral type is also described. Previously reported cases have been caused by occlusive vascular lesions, neoplasms and meningoencephalitis.¹ We wish to record a case which is unique in that the operculum syndrome resulted from a nocardial abscess in the right hemisphere of a left-handed patient who recovered following treatment of the abscess.

A 67 year old Asiatic man presented in April 1985 with difficulty in swallowing for one week and severe speech difficulty for 2 days. He had previously been treated for pulmonary tuberculosis and mild hypertension. There was no previous history of any neurological illness. On examination he was almost mute, being only capable of grunts and hisses. His face was expressionless, with bilateral ptosis. Both sides of the lower face were paralysed but he was able to wrinkle his forehead. The pupils and eye movements were normal. The fundi showed

only mild hypertensive changes. Jaw movements were very weak on both sides. Saliva drooled from his lips. His pharynx, palate and tongue did not move voluntarily. There was mild upper limb weakness of pyramidal distribution, slightly more on the left side. Motor and sensory findings were otherwise normal. A chest radiograph showed the appearance of long-standing fibrotic tuberculosis in the right upper lobe, with possible activity.

The clinical diagnosis was the operculum syndrome, probably the result of atherosclerotic cerebral infarction. Apart from a white cell count of $11.7 \times 10^9/l$ and ESR of 46 mm/h, blood investigations were normal. Intravenous edrophonium hydrochloride (Tensilon) had no effect. Computed tomography (CT) showed a large area of irregular low density in the fronto-central part of the right hemisphere, with slight effacement of the right lateral ventricle (fig a). Following intravenous contrast an area of ring enhancement was defined in the more superficial part of this area (fig b). The left hemisphere was normal except for undue prominence of the Sylvian fissure, probably representing atrophy in the adjacent cortex. Sulci over the convexity of the left hemisphere were more prominent than on the right side.

A right fronto-temporal craniotomy was performed and a superficial mass of 3 to 4 cm in diameter was excised from the region of the operculum. This contained yellowish

pus. The histology of the operation specimen showed a pyogenic abscess with surrounding gliosis. Gram-positive branching filamentous organisms were identified within the exudate. The features were considered to be those of cerebral abscess due to nocardia. There was no evidence of tuberculosis or malignant tumour.

After operation the patient was given tuberculostatic drugs and intravenous penicillin and sulphadiazine. There was transient increase in the weakness of the left arm but this, and other neurological functions improved over the ensuing 3 weeks. Recovery was longest delayed in the movements of the left lower face and the tongue. Some dysarthria persisted. At no time was there evidence of impaired emotional control. Further CT, 18 days after operation, showed some mixed density abnormality in the right fronto-temporal area, but all evidence of the abscess had disappeared. The left Sylvian fissure remained prominent.

During the patient's convalescence we concluded that his speech disorder was anarthria of cortical type. With the help of the patient and his family we learned that he was primarily left-handed though he had been forced to use his right hand at school. As a result he was able to write with either hand. Tests, based on those described by Subirana,² revealed strong left-handed predominance and preference for the left foot and eye. Two of his five children are left handed.

After a course of antibiotics and con-

Fig (a) CT scan showing area of reduced density in the right hemisphere with slight effacement of the right lateral ventricle. Note the slight prominence of the left Sylvian fissure (b) CT scan, at a level 20 mm above (a), showing the ring density which appeared with contrast enhancement.