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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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