Asymptomatic carotid stenosis: spare the knife

Should patients without any symptoms but with a carotid bruit avoid investigation and surgical treatment for the noise in their neck? The answer should be based on answers to several questions. Is the bruit caused by a "significant" internal carotid atheromatous lesion? Can the presence of the lesion be confirmed without risk to the patient? If there is a significant lesion what is the risk of stroke and what are the risks of surgery?

There are no published randomised controlled trials of the value of carotid endarterectomy in these patients, though at least one study is in progress. We therefore have to advise patients on the balance of risks of surgery versus no surgery using uncontrolled data. If patients start considering surgery they may initiate a medical cascade over which they have little or no control, involving them in ever increasing risk, anxiety (for both patient and doctor), and expense.[1] The cascade may begin quite eaily: the patient, a hypertensive middle aged man, consults his general practitioner with non-specific symptoms; the general practitioner happens to auscultate the neck and hears a carotid bruit; the general practitioner wonders whether the bruit indicates an increased risk of stroke; the patient asks the general practitioner about the importance of the bruit; the patient becomes worried and returns later to ask to be referred to a vascular surgeon for advice; the surgeon decides that he must do a carotid angiogram before making a decision; the angiogram shows an atheromatous lesion at the origin of the left internal carotid causing a 60% diameter stenosis. Finally the surgeon performs a prophylactic carotid endarterectomy; unfortunately the patient has a stroke causing a right hemiplegia shortly after the operation. How often do such disasters occur? Does the bruit justify the risk of angiography and surgery?

About 1500 carotid endarterectomies are performed in the United Kingdom every year, mostly for symptomatic carotid disease—that is, transient ischaemic attack or minor ischaemic stroke (P Morris, personal communication)—so that this cascade is likely to be rare here. Nevertheless, the potential is likely to be greater in the United States of America, where many more operations are performed each year (85 000 in 1982[2] and now estimated at 130 000-180 000 for 1986[3]), many for asymptomatic carotid stenosis.

Prospective studies on representative samples of people without previous stroke or transient ischaemic attack who have a carotid bruit suggest that their annual risk of stroke is about 1-2%,[4-6] though only three studies have examined the risk in patients with a proved internal carotid stenosis.[6-8] But not all of these strokes were necessarily due to thromboembolism from an atheromatous plaque in the internal carotid artery: most of the strokes in patients with asymptomatic bruit or stenosis occur in arterial territories unrelated to the carotid artery with the bruit or stenosis.[4-8] Of the ipsilateral strokes, a few are due to primary intracerebral haemorrhage. Of the ischaemic strokes, some are due to thromboembolism from carotid atheroma, others are due to disease of the small penetrating arteries of the brain, and still others are due to embolism from the heart. The annual risk of ipsilateral ischaemic thromboembolic stroke may be as low as 0·1% per year.[3] We do not know with certainty whether asymptomatic patients with "tight" stenosis run a greater risk of stroke than those with a lesser stenosis. More importantly, the annual risk of myocardial infarction and death from non-cerebral vascular diseases is higher (4-10%) than the risk of stroke, and the risk of serious cardiovascular events increases with the severity of the carotid stenosis.[4-8]

Despite these considerations, some patients with a bruit may, knowing that they might have a carotid stenosis, feel worried enough to consider angiography and possibly even surgery. Non-invasive ultrasound screening can reasonably reliably identify the patients with normal or minimally diseased arteries, who may then be spared the risks of angiography. Unfortunately, the facilities for ultrasound assessments are not widely available in the United Kingdom. The risk of angiography varies from centre to centre, depending on the age and general vascular state of the patient and the radiological facilities and skills available locally.[9 10] Cerebral angiography should be performed only in centres with neurological and neuroradiological skills; there the risk of any complication is 5-12%, of any neurological complication 5-15%, of permanent neurological impairment 0·2-5·3%, of acute renal failure 0·1%, and of death about 0·05%.[9 10]

The risks of carotid endarterectomy vary widely, with perioperative stroke or death rates of 1-5% reported from specialist centres and of 11-21% from community hospitals.[11] These do not compare favourably with the risks of no surgery described above. The evidence from the two randomised trials of carotid endarterectomy in symptomatic patients is not encouraging either[12 13]; neither showed a definite benefit from surgery and indeed one was stopped prematurely because the surgical complication rate was unacceptably high.[13]

Two other different groups of patients are sometimes considered for prophylactic carotid endarterectomy for an asymptomatic carotid stenosis: patients about to undergo major surgery, particularly cardiac surgery, and patients with transient ischaemic attacks or stroke found to have a stenosis contralateral to the symptomatic carotid artery. In the first group the evidence is against surgery[14] and in the second the data are inadequate to make a clear decision.

In conclusion, the balance of evidence is at present against angiography and surgery for patients with asymptomatic carotid bruit or stenosis.[15] This conclusion may change when more data are available, especially on the relation between the degree of carotid stenosis and the risk of ipsilateral thromboembolic stroke and on the risks and benefits of surgery. When such data are published and when facilities for ultrasonic assessment of the extracranial arteries are more widely available physicians and surgeons in the United Kingdom may adopt the policy (practised in some centres of the United State of America and Europe) of angiography and surgery for asymptomatic patients at high risk, selected by non-invasive methods.

Nevertheless, surgery on the carotid artery will only ever be a small part of the management of such patients. Death and serious disablement from diseases of the heart and the peripheral arteries are more likely than stroke; hence careful management of heart disease and vascular risk factors will always be the mainstay of management. The use of low dose aspirin or β blockers, or both, in such patients has given promising but unproved results. Large scale randomised trials should be undertaken.

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Diagnosing pulmonary thromboembolism

About a third of patients with untreated symptomatic pulmonary embolism die,[1-3] but with treatment the mortality is only 8%.[4 5] Nevertheless, pulmonary embolism is diagnosed in only 29% of episodes,[6] mostly because of the varied manifestations. What are the manifestations and how can we improve our diagnostic rates?

The classic triad is dyspnoea, pleural pain, and haemoptysis, but these occur in only a fifth of the patients with major pulmonary embolism.[7] Pulmonary embolism affects the circulation and these effects may be misdiagnosed as being due to heart disease.[8] Thromboembolism often causes a mild fever but occasionally is associated with a very high temperature leading to diagnostic confusion.[4] Mild fever, diaphragmatic pleurisy, and an increased serum bilirubin concentration may also give rise to confusion since these may be due to cholecystitis. Again, pulmonary embolism may cause pulmonary oedema,[8] bronchospasm,[9] alveolar collapse, pulmonary infarction, and pleural effusions and therefore lead the doctor to misdiagnose asthma, bronchitis, or pneumonia.

Similar confusion may arise from the results of investigations, which again give non-specific results. In several large series of patients with a clinical diagnosis of pulmonary embolism and an abnormal isotope perfusion scan pulmonary arteriography showed that only 17-50% had pulmonary embolism.[4 10 11] Pulmonary arteriography is the gold standard for diagnosing pulmonary embolism; it is safe,[12] accurate,[13 14] and reproducible, with close agreement in the interpretation of the results between independent observers.[12] Nevertheless, this investigation is much underused, and the first special procedure a doctor is likely to ask for is an isotope perfusion scan. The results of chest radiography, electrocardiogram, and measurement of the partial pressure of the arterial blood gases are of no diagnostic value.[10]

Abnormalities of isotope perfusion scans may be due to many lesions, of which pulmonary embolism is one, but abnormalities indistinguishable from those due to pulmonary embolism occur in 5% of normal healthy volunteers.[15] An abnormal result of an isotope perfusion scan demands further investigation, although a normal result virtually excludes the possibility of pulmonary embolism.[10 16 17] Many workers believe that an isotope scan showing normal ventilation in the area of diminished perfusion is diagnostic of pulmonary embolism. This is true only if the perfusion defect is segmental or larger.[11 16] With smaller defects the probability of pulmonary emboli being present is only 18-63%.[11 16] Other conditions clearly cause mismatching of ventilation and perfusion (V/Q).[18]

The presence of similar abnormalities of both ventilation and perfusion is often taken as indicating a disorder other than pulmonary embolism. Nevertheless, pulmonary emboli affect ventilation[9 10] and hence may give rise to matched ventilation and perfusion defects. No fewer than a third of patients with matched ventilation and perfusion defects shown on isotope scans have pulmonary emboli. Unfortunately many clinicians rely almost entirely on this technique in diagnosing pulmonary embolism,[19] and the resultant total error (false positives and false negatives) is unacceptable at 21%.[16]

In the few patients who are too ill for pulmonary arteriography ascending phlebography should be considered. The presence of venous thrombosis suggests that cardiorespiratory symptoms are due to pulmonary embolism, but a third of patients with pulmonary embolism have no evidence of this.[11]

My scheme for diagnosing pulmonary embolism is in four parts. Firstly, consider the possibility of pulmonary embolism in all cases. Secondly, perform an isotope perfusion scan. If the result is normal dismiss the diagnosis of pulmonary embolism unless the clinical suspicion is overwhelming. If this shows segmental or larger defects then an isotope ventilation scan is indicated. If the abnormally perfused area shows normal ventilation then the diagnosis of pulmonary embolism is accepted unless surgery or thrombolytic treatment is contemplated. Lastly, in all other circumstances pulmonary arteriography is indicated, with particular attention being paid to the abnormal areas shown on the perfusion scan.

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