HOW I WOULD MANAGE A TRANSIENT CEREBRAL ISCHAEMIC ATTACK (TIA) IN A 48-YEAR-OLD HEADMASTER

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A transient cerebral iscahemia attack (TIA) is defined as an acute *focal* loss of neurological function with complete recovery within 24 hours. The diagnosis of TIA is essentially clinical and depends entirely on good history-taking, supplemented where possible by the account of family and friends. TIAs are due to temporary vascular occlusion by small emboli, and symptoms differ according to whether the blockages occur in the territory of the basilar or carotid arteries (Table 1). If this territorial distinction can be made initially, it will help in guiding further investigation. A full history will also include inquiry into previous illnesses and an assessment of cardiovascular risk factors.

TABLE 1
Features distinguishing carotid and vertebral territory TIAs (by definition all symptoms are transient).

Carotid	Unilateral arm, leg or facial weakness Uniocular blindness (*amaurosis fugax*) Hemianopic visual loss
Vertebral	Bilateral or altitudinal visual loss Vertigo Vomiting Dysarthria

Physical examination will include looking for residual neurological signs (usually none) and a search for potential sources of emboli. Emboli may originate in the heart, aorta or neck arteries. As a general rule, cardiac sources are more important in younger, and arterial in older, patients. The cardiac lesions associated with a TIA are not necessarily the same as those causing emboli and stroke. Mitral valve prolapse, atrial septal defects and atrial septal aneurysms, and minor aortic valve disease are relatively common predisposing causes, whereas mitral stenosis rarely, if ever, presents as a TIA. Carotid disease may or may not be associated with a carotid bruit. Subclavian arterial stenosis usually does produce a bruit, and may also be associated with different blood pressure readings in the two arms Aortic atheroma focus change has no such clinical signs.

TIAs and hypertension all strongly associated, presumably because hypertension is a marker for general, and aortic, atheroma. TIAs are rarely associated with abnormalities of blood coagulation as in thrombophilia, systemic lupus erythematosus and thrombotic thrombocytopenic purpura (TTP). In the author's experience, cardiac arrhythmias are seldom associated with a TIA unless there is concomitant severe arterial stenosis.

Simple investigations will include blood count, plasma viscosity, glucose, cholesterol and anti-nuclear antibodies, ECG and chest radiography. The three essential specialist investigations are a brain scan, duplex ultrasound studies of the carotid and vertebral arteries, and an echocardiogram in patients with a murmur or aged under 65.

I would always want to do either a CT, or preferably an MRI (Magnetic Resonance Imaging), brain scan. This may show an unsuspected space occupying lesion, demyelinating plaques or, quite commonly, multiple previous silent infarcts.

Must do:

- **CT, or preferably an MRI, brain scan.**
- **Duplex ultrasound studies of the carotid and vertebral arteries.**
- **Transoesophageal echocardiography (in patients with a murmur or under 65).**

Duplex ultrasound, provided it is carried out by a skilled and experienced operator, has high sensitivity and specificity for detecting carotid and proximal vertebral artery lesions, and has largely superseded angiography. Digital subtraction angiography with an aortic injection of contrast may still have a role in investigating vertebrobasilar and some carotid disease, although even in these instances one would probably now prefer to proceed to an intravenous contrast enhanced MRI angiogram.[1,2]

The situation with regard to echocardiography depends partly on the age of the patient and partly on the clinical circumstances. Younger patients, say under 65, are more likely to have a cardiac source for emboli causing TIAs: this is often a clinically 'silent' lesion such as atrial septal aneurysm. If a cardiac lesion is identified it is very likely to alter management. Patients over 65 frequently have minor cardiac lesions whose discovery seldom affects management. My current policy is not to request routine echocardiograms in patients over 65 unless they have had TIAs in multiple arterial territories or had other evidence of a cardiac lesion. High quality echocardiography is essential and, in a headmaster of 48, I would probably insist on a transoesophageal echo as this provides a better view of the mitral valve and atrial septum.

TREATMENT

TIAs are by definition self-limiting. The scope of this treatment is to prevent more devastating sequels such as stroke. The European carotid surgery trial has provided guidance on which patients with carotid stenosis will benefit from carotid artery surgery and which will do as well on a

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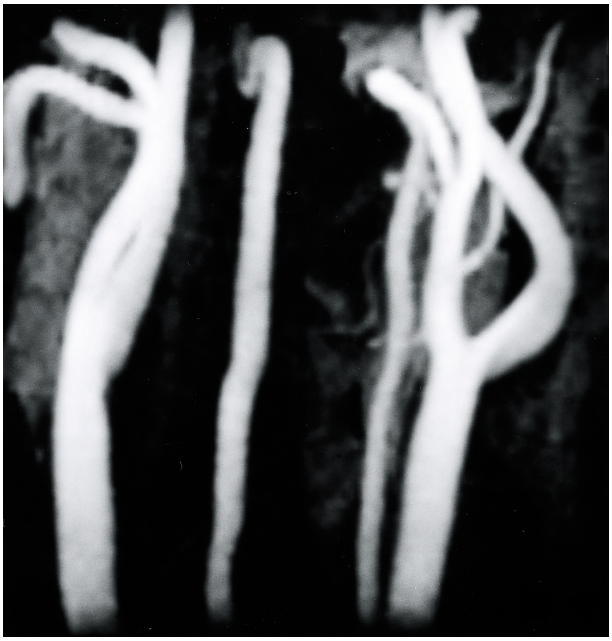

FIGURE 1
Single image from a magnetic resonance angiography study of the vertebral and carotid arteries. Once the images have been acquired, they can be studied in many planes and from any angle (Courtesy of Dr Richard Keal.)

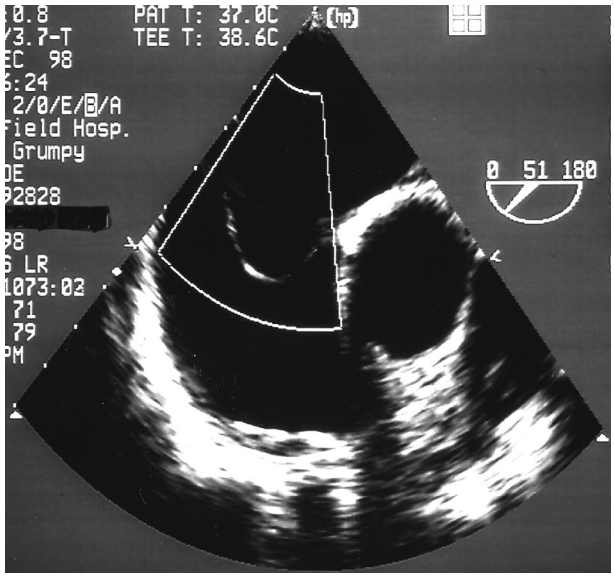

FIGURE 2
Transoesophageal echocardiogram showing a large atrial septal aneurysm. (Courtesy of Dr Richard Keal.)

more conservative regimen of aspirin.[3] The dose of aspirin is not critical although 150 mg/day is commonly used. The effiicacy of aspirin may be further enhanced by adding dipyrimadole.[4] Clopridogrel is an alternative for those unable to tolerate aspirin.[5] I would be keen to recommend endarterectomy for an otherwise fit young patient who has had a definite recent carotid territory TIA, has a tight stenosis in the appropriate carotid artery, and can be operated on in a unit with a good track record. Angioplasty may one day be an alternative.

Aspirin will also reduce the risk of further TIA and the very small risk of stroke in mitral valve prolapse. Atrial septal defects or atrial septal aneurysms are usually dealt with surgically in a young patient, although percutaneous closure with the Amplatzer device is now an attractive alternative.[7] Anticoagulation with Warfarin is used by some cardiologists and neurologists for patients with recurrent TIA, but clinical trials evidence for its efficacy is lacking.

Treatment summary:

- **Aspirin (or clopidogrel).**
- **Correct risk factors.**
- **Surgery for severe carotid stenosis.**
- **? ASD/PFO closure.**

Obviously, the aim of investigation is to find and treat a 'high-risk' lesion. Remember, however, that one can never be sure that such a lesion was actually the source of a particular embolus. Realistically, the most likely outcome of investigations is that they will be negative. The implications of this need to be carefully explained to the patient. The patient should be advised to take aspirin, and attempts are made to control any cardiovascular risk factors, including hypertension, high cholesterol and diabetes: he can be reassured that the risk of a stroke or other serious outcome is low. Whilst one can never totally exclude the possibility of a stroke, the longer the time lapse from the original TIA, and the more successful the patient is in reducing risk factors, the less is the likelihood that this will occur.

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