NEUROLOGICAL MANAGEMENT

Stroke and transient ischaemic attacks

Peter Humphrey

The management of stroke is expensive and accounts for about 5% of NHS hospital costs. Stroke is the commonest cause of severe physical disability. About 100 000 people suffer a first stroke each year in England and Wales. It is important to emphasise that around 20–25% of all strokes affects people under 65 years of age. The annual incidence of stroke is two per 1000.¹ About 10% of all patients suffer a recurrent stroke within one year. The prevalence of stroke shows that there are 250 000 in England and Wales. Each year approximately 60 000 people are reported to die of stroke; this represents about 12% of all deaths. Only ischaemic heart disease and cancer account for more deaths. This means that in an "average" district health authority of 250 000 people, there will be 500 patients with first strokes, with a prevalence of about 1500.

Transient ischaemic attacks (TIAs) are defined as acute, focal neurological symptoms, resulting from vascular disease, which resolve in less than 24 hours; most settle in less than 30 minutes. The incidence of TIAs is a quarter that of stroke.

Over the past 20 years, mortality from stroke has fallen both in the United Kingdom and United States by about a quarter. There has also been a fall in the incidence of stroke.² This is probably real but may be partly accounted for by the reclassification of stroke. The more successful treatment of hypertension is also likely to be relevant, but is unlikely to be a complete explanation as this improvement had also been seen during 1950–60, before the treatment of hypertension was widely practised.

Stroke is not a diagnosis. It is merely a description of a symptom complex thought to have a vascular aetiology. It is important to classify stroke according to the anatomy of the lesion, its timing, aetiology, and pathogenesis. This will help to decide the most appropriate management.

Classification of stroke

Many neurologists have described vascular syndromes in erudite terms. Most of these are of little practical use. A broadbased anatomical knowledge is important, however, as this has significance in pathogenesis and management.

ANATOMY

Carotid v vertebrobasilar arterial territory

Carotid—Classifying whether a stroke is in the territory of the carotid or vertebrobasilar arteries is important, especially if the patient makes a good recovery. Carotid endarterectomy is of proven value in those with carotid symptoms. Carotid stroke usually produces hemiparesis, hemisensory loss, or dysphasia. Apraxia and visuospatial problems may also occur. If there is a severe deficit, there may also be an homonymous hemianopia and gaze palsy. Episodes of amaurosis fugax or central retinal artery occlusion are also carotid events.

Horner's syndrome can occur because of damage to the sympathetic fibres in the carotid sheath. This especially follows carotid dissection.

After an internal carotid occlusion, there may be exaggerated pulsation in the branches of the external carotid artery (especially the superficial temporal artery). Increased collateral bloodflow through this artery shunts blood via the orbital vessels into the ophthalmic artery and then into the circle of Willis in an attempt to compensate for the internal carotid occlusion, patients thus performing their own extracranial-intracranial anastomosis. This is an almost universal finding on ultrasonography in internal carotid artery occlusion. Sometimes the collateral flow is so marked that an orbital bruit is heard and the superficial temporal artery on the side of the occlusion becomes tender and painful. This can mimic temporal arteritis. It is particularly important that the temporal artery is not biopsied or a major collateral source of blood supply will be obliterated.

Vertebrobasilar—The terminal branches of this system are formed by the posterior cerebral artery. Ischaemia of its territory usually produces unilateral field defects. Bilateral symptoms are not uncommon, with complete blindness or bilateral visual hallucinations, such as an impression of frosted glass or water running across the whole field of vision.

Sometimes amnesic symptoms may be seen. In most patients, however, transient global amnesia is no longer thought to be a TIA.³

The posterior cerebral artery also supplies part of the thalamus: infarction here produces sensory impairment over the contralateral

side of the body. This may be accompanied by a very unpleasant pain which may be spontaneous or induced by light touch (thalamic pain) and often only reaches its peak some months after the stroke.

The brainstem signs after vertebrobasilar ischaemia depend on the level of the lesion.⁴ Midbrain ischaemia may result in pupillary changes with impaired vertical gaze or oculomotor nerve dysfunction. Damage to the pons produces horizontal gaze palsy with facial weakness or sensory loss. In either case a tetraparesis or hemiparesis may occur.

A wide range of other syndromes is reported to follow ischaemia of localised areas of the brain. The basic pattern is one of ipsilateral cranial nerve palsies and cerebellar disturbance combined with contralateral paresis or sensory loss which may affect the face, arm and leg, or arm and leg, depending on the level in the brainstem at which this occurs. Horner's syndrome may be seen.

In the "locked-in" syndrome, patients appear to be unconscious but are actually fully conscious. They can only move their eyes vertically; sometimes they can move their eyelids. It is good practice to introduce oneself to patients who appear to be unconscious, and immediately ask them to move their eyes before accepting that they are truly unconscious.

One word of caution—carotid artery dissection often presents with ipsilateral Horner's syndrome and contralateral hemiparesis. It has also been described with ipsilateral cranial nerve palsies (especially affecting nerves IX–XII) because the expanded carotid artery damages these nerves in the neck. Classical teaching would have mistakenly put this vascular syndrome in the vertebrobasilar territory.

Lacunar

These small, deep microinfarcts described by Fisher are commonly seen in hypertensive and diabetic patients.⁵ They rarely occur in patients with carotid artery stenosis. It is important to recognise these lacunar syndromes because of their good prognosis and different pathogenesis. Lacunar infarcts commonly present as pure motor stroke, pure sensory stroke, sensorimotor stroke, or ataxic hemiparesis (table 1). Acute focal movement disorders may also be lacunar. Patients must have either complete face, arm and leg or major face and arm or leg involvement. Those with more restricted deficits—for example, weak hands only—are not included. These are considered to be partial, anterior circulation infarcts in the cortex.

Table 1 Common lacunar infarcts

Clinical type	Site of lesion
Pure motor hemiplegia	Internal capsule, pons, cerebral peduncle
Pure hemi-anaesthesia	Thalamus
Ataxic hemiparesis	Pons, internal capsule
Dysarthria/clumsy hand syndrome	Pons, internal capsule

Sometimes multiple lacunar infarcts occur. In such patients, there is often, but by no means always, a history of preceding minor stroke. The resulting syndrome is of a pseudo-bulbar palsy with dementia, dysarthria, small stepping gait (*marche à petits pas*) unsteadiness, and incontinence.

Bamford and colleagues⁶ have used the Oxford Community Stroke Data to classify strokes clinically into—(a) lacunar infarcts; (b) total anterior circulation infarcts; (c) partial anterior circulation infarcts; and (d) posterior circulation infarcts (vertebrobasilar)—(a) and (b) are classified as carotid artery territory stroke.

Total anterior circulation infarct (TACI) presents with a combination of higher cerebral dysfunction deficit (dysphasia, dyscalculia, and visuospatial disorder), homonymous visual field defect and motor or sensory deficit, or both, of at least two areas of the face, arm, and leg. Partial anterior circulation infarcts (PACI) present with only two of the three components of the TACI syndrome with higher cerebral function alone or with a motor/sensory deficit more restricted than those classified as lacunar infarcts—for example, confined to one limb, or to face and hand but not to the whole arm.

Patients with posterior circulation infarct (POCI) present with any of the symptoms described in the section on vertebrobasilar disease.

Using these simple clinical criteria, it proved possible to classify most strokes into one of these four different categories. This may be important as the prognosis, aetiology, and risk of recurrent strokes varies in the different groups. The TACI group had a very poor prognosis, with high mortality but a low recurrence rate, presumably as most of the carotid territory had been destroyed by the infarct. The PACI group had a good prognosis but a high early recurrence rate, as this type of stroke is frequently embolic, probably from internal carotid artery atheroma. These patients have much to lose if a second stroke occurs. The lacunar group had an intermediate prognosis but a low risk of recurrence. This type of stroke is rarely due to embolic disease but follows microvascular thrombosis or haemorrhage, often as a result of hypertension with or without diabetes.

The POCI group had a good prognosis but high early recurrence rate. This Oxford Community study dispels the notion that brainstem strokes in general have a poor prognosis in the acute phase: it also emphasises the significant risk of recurrence and the need to give advice about risk factors, and offer early medical treatment to those with posterior circulation infarcts.

Subclavian steal syndrome

This syndrome is largely an irrelevance. Subclavian stenosis is common in asymptomatic patients. The classic syndrome of brainstem ischaemia on exercising the arm is rarely present. It has a very low risk of stroke (under 2% annually).⁷ It can usually be

diagnosed by measuring the blood pressure in both arms. It is not necessary to consider any form of surgical intervention unless there are intractable vertebrobasilar TIAs. Angioplasty is a less invasive option.

Border zone infarcts

Sometimes infarction follows a generalised reduction in cerebral blood flow. This is most commonly seen after a cardiac arrest or hypoxic damage during cardiac surgery. Ischaemia is then especially marked in the border zone between the territory of individual arteries because here perfusion is least. The parieto-occipital zone is the area most often affected, where border zone infarcts produce visual field defects (often partial and easily missed on routine examination), reading difficulties, visual disorientation, and constructional apraxia.⁸ In the frontal border zone, slowing up, pathological grasp reflexes, gait disturbances, and incontinence may occur.

The clinical description is therefore helpful in formulating an opinion about the anatomical site, aetiology, prognosis, and risk of recurrence. This may well have management consequences.

TIMING AND PATHOGENESIS

The timing of events is important in our understanding of pathogenesis. Most TIAs are embolic and usually arise from the internal carotid artery or from the heart. A small percentage are haemodynamic—these usually occur when there is severe, widespread occlusive disease.

Sometimes it is possible to identify patients with haemodynamic TIAs clinically. Embolic TIAs usually occur for no apparent cause. Haemodynamic TIAs, however, may have a trigger; for instance, standing, exercising, lowering blood pressure, eating, and straining have all been described as precipitating events. Haemodynamic amaurosis fugax may be triggered by a bright light or sunlight. Unlike patients with embolic amaurosis fugax who usually describe a shutter or black shadow descending across the visual field, those with haemodynamic attacks often initially describe increased contrast between black and white and then whiteness of vision before their vision goes. Haemodynamic amaurosis fugax is often more gradual than embolic amaurosis. Sometimes haemodynamic TIAs may be preceded by symptoms of presyncope such as dizziness and faintness. Finally, they may occur many times a day over a considerable period of time: this is unusual in embolic TIAs.

Although haemodynamic TIAs are rare compared with embolic TIAs, it is important to identify them, as it is unlikely that antiplatelet therapy will help these symptoms. Reconstructive vascular surgery is more likely to be appropriate.⁹

Stroke is usually secondary to thromboembolic disease. About 15% of all strokes are haemorrhagic. Five per cent of these are secondary to subarachnoid haemorrhage and

10% to intracerebral haemorrhage (table 2).

Thromboembolic stroke accounts for 85% of all stroke.¹⁰ It is usually possible on clinical grounds to differentiate those with subarachnoid haemorrhage. The management of subarachnoid haemorrhage is discussed elsewhere.¹¹

The clinical differentiation of thromboembolic disease and intracerebral haemorrhage is difficult. Various authors have attempted to develop a clinical score.¹² Early loss of consciousness, early vomiting, bilateral extensor plantars, and marked elevation of blood pressure all suggest haemorrhage. TIAs and the presence of peripheral vascular disease suggest thromboembolic disease.

No clinical score is sufficiently accurate, however, to allow reliable differentiation. CT, provided that it is performed within two weeks of the first symptom, is the only reliable method. It should be used in patients with stroke because the proper assessment and treatment of thromboembolism and haemorrhage differ.

What percentage of strokes are thrombotic and embolic is even more difficult to ascertain. It is estimated that about half of all thromboembolic strokes are embolic (table 3) and half thrombotic. The percentage of all strokes caused by other factors such as primary hypoperfusion (see above, border zone infarcts), vasospasm, and arteritis is small.

Table 2 Causes of cerebral haemorrhage

- Hypertension
- Berry aneurysm
- Perimesencephalic haemorrhage
- Arteriovenous malformation
- Anticoagulants
- Bleeding into tumour
- Mycotic aneurysm
- Coagulation disorders
- Arteritis
- Amyloid angiopathy
- Drug abuse—cocaine
- Venous thrombosis

Table 3 Cardiac sources of emboli

- Left atrium:
 - Thrombus (usually secondary to atrial fibrillation)
 - Myxoma
 - Paradoxical embolism
- Mitral valve:
 - Rheumatic endocarditis
 - Infective endocarditis
 - Marantic endocarditis
 - Prosthetic valve
 - Mitral valve prolapse
 - Mitral annulus calcification
- Left ventricle:
 - Thrombus—myocardial infarction, cardiomyopathy
- Aortic valve:
 - Rheumatic endocarditis
 - Infective endocarditis
 - Marantic endocarditis
 - Bicuspid valve
 - Aortic sclerosis and calcification
 - Prosthetic valve
 - Syphilitic aortitis
- Congenital cardiac disorders
- Cardiac surgery:
 - Air embolism
 - Platelet/fibrin embolism

Accuracy of diagnosis

The differential diagnosis of TIA and stroke includes epilepsy, migraine, tumour, demyelination, syncope, subdural haematoma, malignant hypertension, hyperventilation, hypoglycaemia, and giant intracerebral aneurysm.¹³

Focal motor seizures may be mistaken for TIAs, especially in patients with a very severe carotid stenosis, in whom the jerking of the limbs occurs as part of the TIA. Focal sensory seizures are even more difficult to distinguish, although the march of the sensory symptoms in a focal seizure may be helpful.

Migraine occasionally presents diagnostic difficulties. The slow build up of a migrainous aura, which often lasts 20 to 30 minutes, would be unusual in a TIA. Visual migraine often consists of positive visual symptoms, such as scintillating scotomas, unlike the blackness of amaurosis fugax. The presence of a typical migrainous headache is unlikely in a TIA. Headache occurs in 16% of patients with TIAs.¹⁴

The United Kingdom TIA Study Group has recently presented their data on tumours mimicking TIAs. Patients who present with sensory or jerking TIAs, loss of consciousness, or speech arrest should all be suspected of having a tumour until proven otherwise.¹⁵

Demyelination is usually suspected because of the age of the patient, past history of previous attacks, and a more gradual onset of hemiparesis compared with that seen in a vascular hemiparesis.

Subdural haematomas rarely present with vascular-like symptoms. They do, however, present a particular diagnostic difficulty. The diagnosis of a carotid TIA is usually reasonably consistent,^{16,17} but vertebralbasilar TIAs are more variable. It is important to be wary of labelling the following as TIAs: loss of consciousness; dizziness; mental confusion; incontinence of faeces or urine; or bilateral loss of vision with reduced level of consciousness. These are all often secondary to hypoperfusion, following primary cardiac disease.

Single symptoms such as vertigo, diplopia, dysphagia, dysarthria, loss of balance, tinnitus, sensory symptoms confined to one part of one limb or face, amnesia, drop attacks, and scintillating scotomas, should always be interpreted cautiously when they occur in isolation. They may, however, be consistent with TIAs, especially if they occur together or with other more definite symptoms of TIAs. The reliability of the diagnosis can be improved if clear cut criteria in plain language are used in the assessment of TIAs.¹⁷

In the diagnosis of stroke, the false positive rate with no investigations is between 1% and 5%, if a careful history is taken of the event.¹⁰ It is important to emphasise that CT is no more accurate than clinical opinion¹³; this is probably because some events that are clinically strokes are mistakenly diagnosed on CT as tumours, a diagnosis that is not substantiated with time.

Risk factors

Age is the most important risk factor for stroke. Hypertension is the most important treatable risk factor.¹⁸ The risk of stroke after a TIA is 30% in five years, the highest risk being in the first year.¹⁹ Other proven risk factors include cardiac disease, diabetes mellitus, smoking,²⁰ and hypercholesterolaemia.²¹ High cholesterol levels are also a major risk factor for heart disease which will be the cause of death in most patients with cerebrovascular disease.

Alcohol, taken in excess, is probably a risk factor for cerebrovascular disease, especially haemorrhage. Raised homocysteine and fibrinogen levels may be independent risk factors for vascular disease.^{22,23}

It is not known if obesity, stress, or physical activity have any part to play in the aetiology of stroke—if so, it is likely to be small.

Investigations

Few basic investigations are necessary for most patients with TIAs. Measurements should include a full blood count, erythrocyte sedimentation rate, urea and electrolyte levels, glucose and cholesterol levels. Many physicians request a chest radiograph and ECG, although it is debatable whether these are necessary if there are no symptoms of cardiac disease.

Patients with carotid TIAs or stroke with recovery should also be assessed with Doppler/duplex ultrasonography to detect carotid stenosis—this is highly accurate but very dependent upon the operator's skills²⁴⁻²⁶; our own experience has shown that most radiology departments setting up Doppler/duplex ultrasound services are highly inaccurate, and all such units should have their results substantiated either by angiography or a proven ultrasound service.

Table 4 lists other tests that should be considered.

Treatment

MEDICAL

Vascular risk factors

High blood pressure after acute stroke is common, often settles spontaneously, and does not need to be treated in most people. Treatment should be started only if hypertensive encephalopathy is considered to be likely (systolic more than 230 mmHg; diastolic over 130 mmHg) or the patient has had a proven cerebral haemorrhage and the blood pressure is markedly elevated. It is also important to check the blood pressure in all patients with stroke—one or two months after discharge from hospital, as a significant number will show a persistent rise in blood pressure that is severe enough to require treatment, even though their blood pressure was satisfactory when they were discharged.

Hypertension is the most important risk factor for stroke. The risk of stroke rises exponentially as diastolic blood pressure increases in the range 70–100 mmHg. A

Table 4 Additional investigations in patients with TIAs

Thyroid function tests (especially when cholesterol is raised or the patient is in atrial fibrillation)

CT if there is:

- doubt about the diagnosis especially if the stroke onset was gradual or the history unclear
- a need to exclude cerebral haemorrhage (ideally all strokes)
- cerebellar stroke with a deteriorating level of consciousness

Cerebral angiography

- (Digital subtraction/magnetic resonance) may be indicated if the patient is a candidate for carotid endarterectomy
- To look for evidence of arteritis
- In cerebral haemorrhage

Echocardiography if:

- abnormal cardiac findings are present, suggestive of valvular heart disease or evidence of recent myocardial infarction, or left ventricular aneurysm either clinically or on chest radiograph or ECG.
- all young patients with stroke (<50 yrs) if no clear cut cause is found
- multiple territory stroke for no clear cause
- antiphospholipid syndrome or systemic lupus erythematosus? Libman-Sacks endocarditis
- positive blood cultures

Blood cultures if the patient is febrile, to detect subacute bacterial endocarditis

Temporal artery biopsy to detect arteritis

24-Hour ECG in case of arrhythmia (very rarely necessary: grossly over used)

Work up for young patients with stroke (see table 6)

7.5 mmHg rise in diastolic pressure within the range 70–110 mmHg is associated with a doubling in the risk of stroke.

This underlines the importance of blood pressure control. There is a risk of precipitating hypotension in a small number of patients, especially the elderly; this is often overstated as a reason for not being more aggressive in the treatment of high blood pressure, especially isolated systolic hypertension.

In the population at large, a modest fall of 5 mmHg in mean diastolic pressure, achievable by reducing the mean daily salt intake by 50 mmol/l, might reduce overall stroke mortality by 22%.²⁷ This would have a greater effect on the total number of strokes than just treating high blood pressure in people with diastolic pressures of over 100 mmHg.

Treating all hypertensive patients would reduce the mortality of stroke by 15%. This compares with aspirin, which reduces the overall incidence of stroke by 1–2%, and carotid endarterectomy, which reduces the overall incidence by 0.5%.²⁸ Recent data suggest that inadequate monitoring and treatment of high blood pressure is common and is the most important, avoidable risk factor.²⁹ These figures emphasise that treating high blood pressure will do more for stroke prevention than any other treatment, either surgical or medical.

Advice about tobacco smoking is clearly important. Good control of diabetic symptoms is also to be encouraged, although there are no data proving that good control reduces the risk of stroke.

There is no consensus about the value of cholesterol lowering drugs. There is, however, no doubt that the lower the cholesterol, the lower the chance of a heart attack.

Prescription costs for lipid lowering drugs are increasing by 20% per year. Drug trials have as yet shown no clear benefit from lipid lowering drugs, although the United Kingdom Healthy Heart Study is about to start, in which 20 000 subjects are expected to be recruited.

A small number of patients, who have a packed cell volume of over 50, need assessment for polycythaemia followed by appropriate treatment.

Acute stroke

In recent years there has been much debate over the value of stroke units. A recent overview leaves little doubt that patients treated in stroke units do better than those treated in general medical wards.³⁰ There is no specific reason for this: it may just be an organisational matter rather than due to any one specific treatment.

More difficult is the question of whether patients do better at home or in hospital. The recent papers from Gladman *et al*,³¹ and Young and Forster,³² have suggested that home treatment may be better. More work needs to be done on this. If patients are kept at home, it is clearly important that they should still be investigated in the most appropriate manner. Trials comparing home treatment with stroke units are needed. We should not assume that hospital is better. Motivation and “do it yourself” physiotherapy are probably greatly enhanced by staying at home, provided that adequate support from social services, paramedical teams, and the family is available.

There is no proven medical treatment for acute stroke. Dextran has been shown not to be beneficial. The trials of calcium antagonists, steroids, and glycerol are inconclusive,³³ and this is an area of active research.

The International Stroke Trial (IST) is comparing heparin, aspirin, and placebo. The Multicentre Acute Stroke Trial (MAST) is assessing thrombolysis. These trials have recently been reviewed.³⁴ Despite the lack of any proven treatment, it is one of the most exciting frontiers in the field of acute neurology. There is no doubt that successful treatment will be found: the increase in, and interest created by, large multicentre trials is rightly unstoppable. Stroke units make these trials much easier to perform. We have only to see how the ISIS trials have transformed the care of myocardial infarction to know that we must encourage stroke units to investigate treatments for this most debilitating disease.

TIA and stroke with recovery

There is no doubt that aspirin reduces the risk of stroke and death in patients with TIAs by approximately 25%.³⁵ The exact dose is unclear. The evidence is best for doses of around 300 mg. Some believe smaller doses (of 37.5 or 75 mg) may be adequate but it is possible that these trials may be too small, and that a difference between 37.5 mg and larger doses may have been missed because of a type II statistical error.

Ticlopidine is also an effective antiplatelet agent, perhaps more effective than aspirin.³⁶ It is available in the United States but is not on general release in the United Kingdom. Unfortunately it sometimes causes skin rash, diarrhoea, and reversible neutropenia—patients, therefore, need more careful monitoring. A related drug, clopidogrel, is now undergoing trials comparing it with aspirin.

Warfarin is indicated for definite cardiac emboli. Lone atrial fibrillation has now been added to this list. The European Atrial Fibrillation Study shows that warfarin reduces the risk of subsequent stroke by 60–70% compared with placebo in patients who have had an episode of cerebral ischaemia. The annual risk of serious bleeding was only 3% with 0.2% intracranial bleeds.³⁷

I also use warfarin if a patient has had several TIAs that were not controlled by aspirin. I give warfarin for six to 12 months and then switch back to aspirin, provided no further ischaemic events occur. There are no good data to support this as yet but trials are being performed in the United States, Italy, and The Netherlands comparing aspirin with warfarin.

A very common question is when to start warfarin after a definite stroke. The risk of recurrent emboli is high after the first event but there is the danger of secondary haemorrhage into an infarct if warfarin/heparin is started too early. The Cardiac Embolism Study Group has shown that the risk of secondary haemorrhage is very low 11 days after the initial event. The risk is also very low in the first 11 days if the infarct is small or the deficit mild. I therefore start anticoagulants immediately if the deficit is mild but delay for 11 days if the deficit is severe (for example, severe hemiparesis, sensory loss and dysphasia with a large infarct visible on CT. Clearly on the first day, CT may be negative and the decision then has to be made on the severity of the clinical deficit only).³⁸

SURGICAL TREATMENT

In 1954 the first carotid endarterectomy was performed. The annual risk of stroke in patients with a carotid stenosis who have had a TIA is about 10%. The surgical risk of carotid endarterectomy varies from 1% to 25%. It is not surprising therefore that, for 37 years, it was not known if this operation was worthwhile. It was only with the publication of the European Carotid Surgery Trial (ECST) and the North American Trial (NASCET) in 1991 that the true value of this operation became known.³⁹

The incidence of serious complications in ECST was 3.7% and in NASCET 2.1%. Patients were randomised to surgery and medical treatment or best medical treatment. In the group with 70–99% stenosis, there was a highly significant benefit from surgery—there were 75% fewer strokes in those treated with carotid endarterectomy. Clearly the lower the surgical complication rate, the sooner the patient benefits from surgery.

In the ECST trial, the crossover was at approximately five months and in NASCET three months. All fit patients with a tight symptomatic stenosis should therefore be offered surgery. They should be told the risk of stroke—that is, 10% annually, the local operative risk, and advised that operation reduces the risk of stroke to 2–3% annually. Patients can then make up their own minds. If the surgical risk is over 10%, then the benefit is lost: all units should aim for less than 5%.

There are a possible 5000 candidates for carotid endarterectomy in England and Wales: operation in this group would prevent 500 strokes in the first year. It is not a cheap form of treatment but, for the individual with a carotid TIA and a tight stenosis, surgery reduces the risk of a stroke by 75%.

The average district general hospital in the United Kingdom serving a population of 250 000 would expect to have about 20 patients who are fit, symptomatic, and have a carotid stenosis of more than 70%. The value of this operation is dependent on low operative mortality and morbidity. I believe that there should be a small number of designated surgeons in each region who do this operation. Each should do at least 50 operations per year and the results should be independently audited.

It is important to appreciate that this operation is only for patients with recent carotid symptoms, such as amaurosis fugax, hemiparesis, hemisensory loss, and dysphasia. Just how much this operation has been overused is emphasised by the report in 1988 (before the results of the ECST and NASCET trials) which showed that only 35% of patients had this operation for appropriate reasons in a sample of 1302 patients in the United States.⁴⁰

Ultrasonography/angiography

None of these surgical trials included the angiographic risk. Although the risk of stroke after carotid angiography is generally quoted as 1%,⁴¹ it is almost certainly higher in patients with carotid stenosis, leaving approximately 2% with a permanent disability.⁴² Doppler/duplex ultrasonography is undoubtedly the best screening test but it is very dependent on the skill of the operator. Some units operate on the results of ultrasonography alone; unfortunately, obtaining information on the intracranial circulation is difficult with ultrasound. Combined with magnetic resonance angiography (MRA), ultrasound gives highly accurate information about both the carotid artery in the neck, as well as an angiographic picture of the whole intracerebral circulation.

Our policy is to operate on the basis of an entirely non-invasive work up with ultrasound and MRA if both tests agree (Young *et al*⁴³). We have found that, in patients with 70–99% stenosis, the tests agree in 96%: we only pursue digital subtraction angiography in the other 4%. If MRA proves to be less dependent on the operator and becomes widely

available, it may replace the need for ultrasonography; however, this would require an enormous expansion in MRA to all district general hospitals.

In expert hands, a bruit is the best clinical guide to detect an underlying internal carotid stenosis.^{44,45} The bruit is lost, however, in very tight stenoses (false negative).

A false positive bruit is not infrequent in the presence of either a contralateral occlusion, external carotid stenosis, or just internal carotid atheroma. Furthermore, if the presence of a bruit is to be of value, it needs to be useful to those who are making the initial assessment. The presence or absence of a bruit mentioned in the referral letter to a cerebrovascular clinic showed a specificity of 70% and sensitivity of 57% for patients with 70–99% stenosis (Davies and Humphrey^{45a}). On this basis, many patients with a carotid stenosis would be denied surgery if only those with a bruit were referred.

All patients with carotid TIA or who have recovered from stroke should have carotid ultrasonography in a department with a proven track record. I no longer listen for a bruit; if I wish to detect a carotid stenosis I use ultrasonography.

My own personal work up for carotid endarterectomy is therefore a careful history, simple examination of the cardiovascular system (occasionally I examine the neurological system!), routine blood tests, chest radiography, and ECG. This, combined with Doppler/duplex ultrasonography, is all done at the first clinic visit. If the patient is found to have a carotid stenosis and is prepared to take the risk of surgery, then an urgent MRA is booked in the outpatient department.

CT/MRI

I do not routinely perform CT or MRI on these patients. The value of CT was evaluated in a prospective study of 469 patients being considered for carotid endarterectomy—the cost was considerable and the results did not alter management.⁴⁶ In this increasingly cost conscious health service, we need to look for value for money.

Tumour “TIAs”¹⁵ are rare and can often be suspected on clinical grounds—patients with speech arrest, pure sensory TIA, black-outs, and jerking during their attacks should all raise the suspicion of alternative pathology. CT need only be performed in this group.

Assessment for surgery

I remain convinced that a neurologist or physician with a major interest in vascular disease should perform the initial assessment. These are not patients who should be referred primarily to the vascular surgeons. In our cerebrovascular clinics, of the 25 new patients we see each week, only two or three on average meet all the criteria for carotid endarterectomy. The differential diagnoses recently seen in our clinic include migraine, epilepsy, hyperventilation, tumours, Parkinson's disease, and motor neuron disease, to

name but a few. In a population of a million people, there are about 50 to 100 who are candidates for carotid endarterectomy each year: this compares with around 15 000 with asymptomatic carotid stenosis. The scope for inappropriate surgery is substantial. It is unreasonable to expect a vascular surgeon to differentiate these other conditions.

There is little doubt that the risk of a stroke is highest in the first six months after the initial event and, if the time from first symptom to assessment, investigation, and surgery takes several months, then we are failing to meet the needs of many patients. In the United Kingdom we need to assess these patients within a few days of their symptoms and prepare them for surgery, if appropriate, within two weeks after TIAs and six to eight weeks after recovery from stroke. This will require changes in organisation, more neurologists with an interest in vascular disease, and perhaps more vascular surgeons.

Hydrocephalus

Hydrocephalus is a complication of cerebellar strokes (both haemorrhage and infarction) which may be amenable to surgical treatment.

Asymptomatic bruits

These are common in the elderly population (approximately 7% over the age of 65). The annual risk of ipsilateral stroke is approximately 2%. Surgery is of no proven value, although trials are in progress.

OTHER ASPECTS

Emotional

Psychiatric factors are very important; few doctors have sufficient time to address these fully. Depression and anxiety are common: with reassurance, especially about the risk of recurrence and advice about treatment to prevent further events, this will often improve. Counselling the spouse and close family is also important.⁴⁷

Emotionalism is also common: it is present in both bilateral and unilateral strokes. It is usually sufficient to explain to the patient that this is a physical symptom which will improve with time. Small doses of amitriptyline (10–25 mg) may be beneficial.⁴⁸

Epilepsy

Early epilepsy occurs in about 10% of all patients with stroke. It should be energetically treated with, for example, intravenous phenytoin, as the cerebral metabolic rate doubles during a fit. Late epilepsy after a stroke is a common cause of epilepsy in the elderly population. It is rare in the individual patient, however, and indicates that the validity of the diagnosis of cerebrovascular disease should be reassessed.

Dysphagia

This is common, even after unilateral strokes. It usually improves but predisposes to aspiration, chest infection, dehydration, and death. As it can be assessed by simply asking the

patient to drink 50 ml of water, this test should be mandatory in all patients with stroke.⁴⁹

Thalamic pain

This is more common than is generally appreciated; it frequently starts weeks after the patient is discharged from hospital. It is a cause of great distress and may be helped by a variety of strategies.⁵⁰

Other common problems

After acute stroke, deep venous thrombosis occurs in more than 50% of paretic legs, although a relatively small number develop symptomatic pulmonary embolism. It can usually be managed with elasticated stockings. Pressure sores, septicaemia (often secondary to urinary tract or chest infection), and hyperglycaemia may arise. Frozen shoulder is a common problem and can markedly slow recovery.

Prognosis

The risk of a stroke after TIA is approximately 30% in five years. It is highest in the first year: in patients with carotid stenosis it is about 10–12%. The figures for stroke are similar. It is therefore important to reassure all patients that a second stroke is not imminent. Even patients with bilateral internal carotid occlusions only have a recurrent stroke risk of 13% per year.

Death in most patients with TIAs and stroke is caused by cardiac arrest.⁵

After an acute stroke, 20–30% of patients die. A poor prognosis is associated with reduced consciousness, conjugate gaze palsy, signs of severe brainstem dysfunction, pupillary changes, and incontinence persisting beyond the first few days. Strokes resulting in cognitive impairment such as apraxia and neglect, and visuospatial dysfunction also carry a poor prognosis for recovery.

One year after a stroke, 33% of patients will be dead, 22% dependent, and 45% independent. Most recovery occurs in the first few weeks; less occurs in months three to six and even less (but still useful recovery) occurs in months six to 12. It is known that some symptoms, such as hemiplegic leg, can improve over a long period of time, but others often do not improve unless there is early recovery—for example, retinal infarction, homonymous hemianopia, and isolated spinothalamic sensory loss. In the hemiplegic hand, if there is no active hand grip after three weeks, there is unlikely to be much improvement. It is crucial to take the natural history of disability into account when planning rehabilitation.

Six months after a stroke, almost half the patients will be physically independent, 15% will have speech problems, 11% will be incontinent of urine and 7% incontinent of faeces, and 33% will still need assistance with feeding.

Stroke in the young

Stroke in youth is often not caused by premature atheroma (table 5). It should be investigated by a neurologist. The most common causes are emboli from the heart, carotid or vertebral dissection, antiphospholipid syndrome, arteritis, cerebral venous thrombosis, and premature atherosclerotic or hypertensive vascular disease. These require active assessment and treatment (table 6).⁵² It is likely that cerebral venous thrombosis should be treated by anticoagulation.⁵³

Care of patients with TIA and stroke

In England and Wales there are approximately 100 000 patients with first stroke each year and 25 000 with initial TIAs.

TIA

Most TIAs will be managed by general practitioners. All reasonably fit patients with carotid events aged under 80 years should be referred to a neurologist or physician with an interest in vascular disease, to investigate the possibility of carotid stenosis, provided the patient is prepared to take the risk of operation. All patients should be told the local surgical risk. The results should be independently audited to ensure a low complication rate.

I believe that patients under 50 years of age with TIAs should receive specialist opinion.

Table 5 Causes of stroke in the young

Common:
Premature atherosclerosis
Cardiac embolism
Dissection—carotid or vertebral
Antiphospholipid syndrome including Sneddon's syndrome
Migraine
Arteritis (including postinfective e.g. ophthalmic zoster)
Venous thrombosis
Pregnancy
Uncommon:
Fibromuscular dysplasia
Drug abuse especially cocaine, heroin, amphetamine
Late effect of radiotherapy
Moya moya syndrome
Takayasu's syndrome, Behçet's syndrome
Amyloid angiopathy
Homocystinuria
Fabry's disease
Pseudoxanthoma elasticum, Marfan's syndrome, Ehlers-Danlos syndrome
Haematological causes
Mitochondrial cytopathy
Syphilis
AIDS
Neoplastic angioendotheliosis

Table 6 Additional tests in young patients with stroke

- MRI/MRA*
- Echocardiography*
- Serology for syphilis*
- Lupus anticoagulant, antinuclear factor*
- Anticardiolipin antibody*
- Conventional angiography
- Haemoglobin electrophoresis
- Haematological opinion including pro-antithrombin III
- 24-Hour ECG
- Screening tests for homocystinuria
- Lumbar puncture
- Brain biopsy/meningeal biopsy
- White blood cell α -galactosidase
- Muscle biopsy
- HIV screen

*Should be performed in all young patients with stroke.

I spend much time reassuring patients and refuting the diagnosis of vascular disease, usually with enormous relief to the patient. Patients with TIAs not controlled by aspirin or causing diagnostic difficulty should also be seen for specialist opinion.

STROKE

This is more difficult—the first question is whether the patient should be admitted to hospital. This is partly dependent on the severity of the deficit and age but, more often, on social factors such as the presence of carers and support services. Whatever policy is followed, patients should be investigated appropriately and, ideally, all should have CT.

I have no doubt that there will be treatment for acute stroke soon (as in myocardial infarction), and that acute stroke units will be needed to administer this. Acute investigations will all be performed at the same time. After a short period in such a unit (perhaps 24–72 hours), there are likely to be three options. For patients who have a mild deficit and will do well in any case, they can go home, needing only a small amount of domiciliary services. For the severely disabled who will not do well whatever is done, these patients may be managed at home or in nursing homes or other long stay institutions. It is a waste of time and resources putting these people through a long and arduous rehabilitation programme which will do nothing except lead to frustration and disappointment for staff, patient, and family alike. Realistic goals must be set at all times and the patient and carers must understand what these are.

The rehabilitation of those patients with intermediate disability will be discussed in a separate article in this series.

Clinical criteria to identify these groups are slowly being formulated. Where doubt exists, the patient should be assumed to be able to benefit from rehabilitation. New data are beginning to identify patients at an early stage who will not benefit from rehabilitation.

A decision is needed whether rehabilitation is best delivered at home or in hospital. It is also necessary to ascertain what aspects of disability respond to physiotherapy, speech therapy, and occupational therapy. Could one type of generalised therapist deliver most of this care and advice, only calling on a more specialised service if necessary? This would certainly simplify a lot of domiciliary care.

Rehabilitation research needs to answer questions such as this to provide a useful, long term answer about the role of these therapists. Multicentre trials with simple protocols are just as relevant to rehabilitation as drug trials.

I suspect that rehabilitation is best managed at home: one hour daily (at best) of inpatient physiotherapy will be trivial compared with the amount of “physiotherapy” motivated patients will do in their own home. The two most important factors affecting length of stay in hospital are stroke severity and the absence of a carer at home.⁵⁴ Routine

follow up every three to six months may help to reverse or slow down the late decline in mobility seen after stroke.⁵⁵ An integrated stroke service must be coordinated by a team leader. In hospital, this will be the consultant in charge of the stroke unit. In the community, the integrated stroke service liaising with the general practitioner will organise the necessary service. We do not want two completely separate services, one led by the hospital and one by the general practitioner. Large, randomised trials with detailed costings are needed to find out what is the most effective method of delivering optimal care. In addition, all therapists must be aware of their role as counsellors.⁵⁶

It is essential that we use the charities fully. In the United Kingdom, the Stroke Association is becoming more and more active, and a host of support clubs are springing up. Booklets about many aspects of stroke from TIA to wheelchairs, and from epilepsy after stroke to stroke in the young are available.

Delivering stroke care is expensive; it uses a large percentage of the NHS budget and we need to deliver care in the most efficient and cost effective manner. No view, however steeped in tradition, should be exempt from proper clinical assessment with large, properly conducted trials. The progress made in the past 10 years tells us this must be the way forward; it is exciting to answer questions that everyone involved in the care of patients with stroke will face daily. The ultimate beneficiary is the patient.

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