WORK FOR ALL

Any country could solve the problem of unemployment by learning from the Chinese—all that is necessary is a stock of picks, shovels, and baskets. As an example, workers on a commune who could not afford machinery moved three million cubic metres of earth over seven winters to deepen their fishponds. Research had shown that more fish could be bred in the same area of water if the depth was greater, and this proved to be the case. Their increased income will help them achieve the goal of farm mechanisation by 1980. Chairman Mao's roots went deep into Chinese soil—he was very much a man from the land. He saw China as primarily an agricultural country, and he was determined to give the peasant a status equal to that of the worker, both blue- and white-collar types. The Government guarantees the farmer's income. It buys his produce, and sells to the consumer; it fixes the price of his farm machinery, fertiliser, and pesticides. The income of the farm worker is roughly equal to that of a factory hand, though the city dweller possibly benefits in relation to the amenities of urban life.

There is great disagreement in China about what we would call marginal payments. In a detailed briefing at a commune near Shanghai we were told that all workers received 12 yuan (£3) a week, this being sufficient on which to live, if simply. Points were allocated for heavy work, long hours, and skills. Each worker's points were published monthly, and at the end of the year the commune's profits were shared in proportion to the points awarded through the year. In answer to a question the commune spokesman insisted that this was not a bonus, and in the sense that points had to be earned by work it was not, though undoubtedly it was a margin of some sort.

A large body of opinion in China opposes all types of marginal payment, on the basis that they constitute a bourgeois tendency, while many believe that, in the face of the need for

constantly increased production, material incentives are necessary. This argument is likely to be thrashed out at the highest level, the Central Committee of the Party, before it is resolved, and might form part of another debate on the direction of the mass line. As, however, the Chinese give themselves until the year 2000 to achieve their four modernisations—in agriculture, industry, science and technology, and defence—a decision may be deferred until production goals have been won.

Improved living conditions

There has been a revolutionary change for the better in the living standards of the ordinary Chinese, yet they are still well below the expectations of the average Western family. Private cars are unknown. There is a shortage of power—public buildings and private homes are usually not heated (though, fortunately for visitors in winter, Western style hotels are). Industrial air pollution is conspicuous. I should think that many houses and factories built since liberation will before too long be demolished and replaced by something better, and if this is so the Chinese have 20 or 30 years' hard work ahead of them.

Perhaps people work better if they have a major challenge, a conspicuous objective, can believe in what they are doing, and have faith in their leadership. (The Chinese may have lost some of their faith during the Great Leap Forward and the Cultural Revolution, but these events are receding into history.) This may just be another way of saying that it is easier to make hard conditions good than good conditions excellent. To a Westerner, China's lack of privacy and lack of the opportunity to differ could make life intolerable, yet we found much there to admire. A State that could combine the best of Western ways with the best that China has to offer would provide a good life for its citizens.

*If I Had . . .***If I had a transient ischaemic attack at the age of 55**

J M S PEARCE

British Medical Journal, 1978, **1**, 969-971**Definition**

Transient ischaemic attack means a transient disorder of cerebral function most commonly manifested as hemiparesis, loss of visual or speech function, lasting less than 24 hours, with complete recovery.

History (fictitious)

"It is only a funny attack of migraine," I thought as the shadow appeared in the upper part of my right field of vision while I sat at lunch one day. The shadow descended like a

shutter darkening the vision, and dropping relentlessly downwards until the lamb cutlet in front of me disappeared when I covered my other eye. The descent of this spectre took only a minute, and then a strange numbness and tingling affected the lips on the left side of my face, and five minutes later I felt a similar sensation in my fingertips and in my foot. My fingers became clumsy, the fork in the left hand missed its target, which was clearly perceived through the left eye. A momentary feeling of panic supervened but was quickly dispelled by the brain's incredible capacity for self-deception which enabled me to recall the papers I had written years before on complicated migraine; not forgetting, of course, attacks of common migraine years before which had faded as increasing maturity had replaced the enthusiastic impulsiveness of youth.

I managed to leave the table and get to my favourite armchair, noting with some satisfaction that my left arm and leg could still move, although they felt like heavy lumps of lead and had lost more than a little of their customary agility and dexterity. Some 10 minutes later the shutter started to lift in my right eye and the heaviness had started to lessen in the limbs.

Department of Neurology, Hull Royal Infirmary, Hull HU3 2JZ
J M S PEARCE, MD, FRCP, consultant neurologist

As I cursed my fate, I was pleased to note that my vocabulary and articulation were not impaired. Within the half hour the whole incident had resolved, the headache never materialised.

But what should I do next? Some hours of introspection convinced my rational mind that this was a transient ischaemic attack (TIA) and no ordinary migraine. Apart from this latter disorder my health had been good except for the self-inflicted ills of the occasional cigarette and a degree of obesity thoroughly deserved and acquired with great enjoyment.

Orthodox management

Presented with such a patient, examination and investigation aim at discovering reversible causes of the disorder. The most important is hypertension. The purpose of investigation is to prevent further ischaemic attacks which may culminate in a stroke or death. The risk is greater in the first two months: 40% of patients with TIAs will have a major stroke within three years. Assessment includes three different aspects (see table).

Primary assessment of TIA or stroke

Brain	(a) Where is the lesion in the brain? (b) Is it haemorrhage or infarct—thrombotic or embolic? (c) How large is it? (d) Could it be other than an infarct—for example, benign mass? (e) Were there previous lesions in the brain?
Vessels	(a) Is the lesion thrombotic or embolic? (b) If embolic, where is it from? (c) Could there be an underlying arteritis? (d) Is there abnormal intravascular coagulation? (e) Is the lesion in small vessels inaccessible, or in neck vessels accessible to surgery?
Systemic factors	Is the patient: (a) Hypertensive on serial recordings? (b) Diabetic? (c) Anaemic or with a high packed cell volume? (d) Cardiac source of embolism: thrombus, arrhythmia, myxoma, or floppy valve. (e) Hyperlipidaemic? (f) Syphilis (now rare in this context). (g) Collagenosis, polyarteritis, systemic lupus, giant cell arteritis?

The lesion in the brain—This is normally not seen as a consequence of TIA, the clinical recovery indicating ischaemia rather than permanent infarction. Chest and skull x-ray films, supplemented by isotope or CAT scanning, will show infarcts or haemorrhages in gross examples of stroke. The site of the lesion, its size and the visualisation of other older and unsuspected lesions may significantly influence subsequent management. Before CAT scanning and angiography diagnostic errors were frequent; thus one important role is to confirm the pathology so that a tumour, primary or metastatic, which may present as a stroke in 10-15% of patients will not be overlooked. Subdural haematoma, angioma, and meningioma are particularly likely to mimic a stroke or TIA. In older subjects, previous areas of infarction or haemorrhage may be shown by a CAT scan, and the cerebral atrophy of Alzheimer's disease, occurring independently, may be an important factor in deciding against radical treatment.

Lesions in the vessels—Hypertension and diabetes produce small-vessel disease. This affects smaller arteries and arterioles arising from the middle cerebral and posterior cerebral arteries deep in the brain to produce microaneurysms and lacunar infarction of 2-10 mm in the basal ganglia, internal capsule, and pons. Angiography is not indicated in many of these patients, but, because both disorders can accelerate the development of atheroma, the occurrence of carotid stenosis should not be overlooked. The presence of an arterial bruit in the neck or attacks of amaurosis fugax (as described in my imaginary case history) are clear indications of carotid disease and angiography should be performed in these cases. Younger patients, including women taking oral contraceptives, usually need angiograms.

The vogue for arch aortograms to show both vertebral and carotid vessels has in some degree receded. This trend is probably correct because very few patients with proved vertebral artery disease are suitable for surgery, although some vascular surgeons insist on full visualisation of all four vessels so that they know the efficiency of the collateral circulation available before they embark on a carotid thromboendarterectomy. The minimal requirement, therefore, is bilateral carotid angiograms to show the presence of occlusion or

stenoses. In experienced hands the morbidity of carotid angiography is less than 1%, and the mortality rate a tiny fraction of 1%.

Systemic factors—Much the most important is hypertension, which for practical purposes implies a sustained diastolic pressure of over 100 or a systolic pressure of over 180 mm Hg. The passion for obtaining a basal blood pressure reading is unrealistic. Frequent casual recordings, or, better still, 24-hour monitoring during daily activities are more valuable in borderline cases. The important contributory roles of a high packed cell volume, or anaemia, necessitate a full blood picture. A raised sedimentation rate may be an indication of polyarteritis, systemic lupus erythematosus, or giant cell arteritis. Fasting and postprandial blood glucose will show contributory diabetes, and fasting plasma cholesterol and triglyceride concentrations should be estimated. Meningovascular syphilis and general paresis are now extremely rare in the context of TIAs, but VDRL, WR, and TPHA tests are still often performed routinely.

Clinical examination should include a search for peripheral vascular disease and symptomatic coronary heart disease, whose presence may well deter the surgeon, and, if so, angiography is unnecessary because there are no therapeutic consequences. Since most TIAs result from embolism rather than thrombosis, not only should a carotid stenosis be sought as a source of embolism, but also cardiac lesions—of which intermittent atrial fibrillation, paroxysmal atrial and ventricular tachycardia, bacterial endocarditis, and mural thrombus over the site of an infarct are the most important. Of patients presenting with TIAs, 40% have an abnormal ECG. In many instances of TIAs, carotid angiograms are normal and embolism from a paroxysmal cardiac arrhythmia is an important cause, though often very difficult to prove. All patients should have a routine ECG with a prolonged rhythm strip, but in selected cases it is prudent to obtain ECG monitoring for 24 to 48 hours.

TIAs in vertebrobasilar territory usually display distinctive clinical features. If these occur in a normotensive person without evidence of coexistent carotid artery disease most neurologists would not advise angiography. A search for contributory systemic factors should, however, be undertaken. The role of cervical spondylosis in compressing vertebral arteries may be important, though if other indications of cervical myelopathy are absent it does not usually require treatment. If brain stem ischaemia is associated with exercise of the arm—the subclavian steal syndrome—a four-vessel arch angiogram via femoral or brachial artery may lead to successful disobliteration of a subclavian stenosis.

If I were the victim

Should I have an isolated TIA, I would first contact my general practitioner and would try to be a good and compliant patient. I doubt, however, whether I would succeed. I would hope he would refer me to a neurologist, preferably of my choice, and I would readily submit to a full clinical examination and for a detailed search for the contributory systemic factors. This might include sacrificing 50 ml of blood and a sample of urine and having ECGs and inpatient cardiac monitoring. I would willingly have chest x-ray films and CAT scanning, which would no doubt eliminate the exaggerated fears of other lesions. But the major decision relates to angiography. On balance, I would submit to bilateral carotid angiograms, performed by a neuroradiologist of repute, with a good anaesthetist.

If the angiography showed a stenosis of the carotid bifurcation in the neck, and if other tests had failed to prove widespread arterial disease, then I hope that sense would overcome apprehension leading to successful carotid thromboendarterectomy. I would choose both surgeon and anaesthetist with care.

If, by contrast, the results of systemic investigations and angiography were normal, I would feel relieved—such patients in my experience having a much smaller recurrence rate. I would probably take aspirin, 600 mg twice daily, to reduce platelet aggregability and subsequent thromboembolism. Should angiograms show a total occlusion or extensive atheroma of cerebral vessels and the carotid syphon, the choice would be between watch and do nothing, aspirin, and anticoagulants. The efficacy of the last is least unconvincing in TIAs, and, though I am a devoted anti-anticoagulator, recurrence of TIAs (especially

if they increased in frequency or duration) would persuade me to accept such treatment—provided that there was no operable stenosis or reversible factor such as hypertension. Their benefit probably lasts only two or three months and certainly if they have no clinically impressive effect they should gradually be discontinued after such a period.

The one part of my own medicine to which I would closely adhere is the strict supervision and control of high blood pressure. If I were so affected I would try hard to be what is popularly known as a “compliant customer.” Symptomatic or not, with signs of left ventricular strain or hypertrophy or not, I would accept a strict regimen because the achievement of normal blood pressure is usually possible nowadays without serious side effects. This, above all other mentioned factors, can enormously improve the outlook, in direct proportion to the level of control achieved. One would try to acquire a passive

frame of mind, accepting the necessity of regular “check-ups”: retinoscopy, ECG, and a chat with a trusted colleague without becoming too preoccupied or introspective. In fact, I would try to emulate the best of my own patients.

Selected further reading

- Carter, A B, *Lancet*, 1970, **1**, 485.
 Fisher, C M, *Archives of Neurology (Chicago)*, 1970, **22**, 13.
 Gunning, A G, *et al*, *Quarterly Journal of Medicine*, 1964, **33**, 155.
 Hutchinson, E C, and Acheson, E J, *Strokes: Natural History, Pathology and Surgical Treatment*. London, W B Saunders, 1975.
 Marshall, J, *The Management of Cerebrovascular Disease*, 3rd edn. Oxford, Blackwell, 1976.
 Pearce, J, and Aziz, H, *British Medical Journal*, 1969, **4**, 78.
 Ross Russell, R W R, *Cerebral Arterial Disease*. Edinburgh, Churchill, 1976.

Today's Treatment

Diseases of the respiratory system

Bronchiectasis and cystic fibrosis

MARGARET E HODSON

British Medical Journal, 1978, **1**, 971-973

Bronchiectasis

Bronchiectasis is characterised by dilatation of the bronchi. This dilatation may be generalised or confined to a single segment or lobe of the lung, and it may be tubular, fusiform (spindle shaped), or saccular (cystic).

Aetiology

Bronchiectasis usually starts in childhood but may begin in adult life. The commonest cause is damage to the bronchial tree after infections such as tuberculosis, bacterial or viral pneumonia, and bacterial infections complicating measles or pertussis. Bronchiectasis may be associated with certain congenital disorders—for instance, Kartagener's syndrome (a condition also characterised by dextrocardia and sinusitis), Williams-Campbell syndrome (bronchomalacia caused by congenital absence of cartilage distal to the first division of the peripheral bronchi), and sequestered lung segments. Obstruction of the bronchi due to carcinoma, tuberculous stenosis, or inhaled foreign bodies may cause pulmonary collapse and subsequent infection leading to bronchiectasis. Recurrent sinus infections may also lead to it.

Bronchiectasis may complicate a more widespread disorder such as cystic fibrosis or some immunological deficiency such as hypogammaglobulinaemia. Bronchiectasis that characteristically affects the proximal bronchi may occur in allergic bronchopulmonary aspergillosis.

Symptoms and diagnosis

The symptoms of bronchiectasis are cough and sputum production. There may be associated haemoptysis and the symptoms of a more generalised bronchitis such as wheeze and dyspnoea. The patient may have poor general health and finger clubbing. Crepitations may be heard over the areas of retained secretions. Bronchiectasis can often be diagnosed from the chest radiograph if a lateral and penetrated film are taken as well as the routine posterior-anterior view. Ring or tubular shadows may be seen, or abnormalities of the vascular pattern. The diagnosis can be confirmed and the full extent of the disease defined by bronchography, though this is an unpleasant investigation and is justified only if the patient is a candidate for surgical treatment.

Treatment

Treatment may be medical or surgical. If a patient has moderate or severe symptoms, is otherwise fit, has good lung function, and has bronchographic evidence of localised disease then he should be offered surgical resection of the affected lobe. If the remaining lung function is reasonable patients can stand resection of one lobe, the left lower lobe and lingula, or the right lower and middle lobes. In carefully selected cases excellent results can be obtained and a lifetime of medical treatment avoided. All these patients should receive intensive medical treatment before operation.

Most cases of bronchiectasis are unsuitable for surgery either because the patient has poor lung function or because the disease is widespread. The most important aspect of medical treatment is regular postural drainage of the affected segments or lobes. To drain the basal bronchi the patient may raise the foot of the bed; special tipping beds are available for this purpose. At home a patient may use a pile of pillows.

Brompton Hospital, London SW3 6HP

MARGARET E HODSON, MD, MRCP, senior medical registrar