Today's Treatment

Diseases of the central nervous system

Cerebral atherosclerosis, transient ischaemic attacks, Menière's disease, and disorders of balance

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The apparently unrelated disorders of cerebral atherosclerosis, transient ischaemic attacks, Menière's disease, and disorders of balance may be conveniently considered together as conditions with a primarily vascular aetiology which cause increasing disability with advancing years.

Cerebral atherosclerosis

CHRONIC BRAIN FAILURE

The concept of senility due to faulty cerebral plumbing has long since outlived its usefulness. Senility has never been a substitute for a proper diagnosis and there is no evidence that the pathological changes normally associated with chronic brain failure in old age are secondary to atherosclerosis.¹ The typical, insidious, slowly progressive dementia of old age is associated with a diffuse Alzheimer-like degeneration, and there is no relationship between the degree of Alzheimer degeneration and the state of the cerebral vessels. The diagnosis of organic brain syndrome secondary to cerebral atherosclerosis requires the presence of focal neurological signs and symptoms, and a step-wise progression, and is reinforced by the co-existence of hypertension. The typical pathological finding in true atherosclerotic dementia is widespread small brain infarcts. Many of the infarcts affect the cortex and are presumably responsible for the observed mental deterioration. Therefore drugs with a reputed psychoanabolic effect, whose only pharmacological action is vasodilatation, are of no value in the treatment of organic brain disease.

In a few patients, however, various indices of mental function improve after treatment with some preparations—for instance, cyclandelate (Cyclospasmol), naftidrofuryl (Praxilene),² and dihydroergotoxine mesylate (Hydergine). It is suggested that they have a direct action on the metabolism of cerebral cells. They should be withdrawn if there is no improvement in two months, as improvement thereafter is extremely unlikely.

ACUTE CONFUSIONAL STATES

Since the brain is such a sensitive indicator of oxygen deprivation many systemic upsets in diffuse atherosclerosis,

which compromises cerebral circulation, will result in an acutely confused state. At least 15% of the patients admitted to hospital with a diagnosis of dementia will improve when the underlying condition is treated. For this reason, many doctors are unwilling to use the term "dementia" which suggests an irreversible process with a poor prognosis, and so encourages therapeutic nihilism. Conditions which may give rise to confusional states are inappropriate drug treatment, depression, intracranial space-occupying lesion, respiratory infection, myocardial infarction, thyroid disease, B₁₂ folate deficiency, and hypokalaemia. Thus in a confused old person, urea, electrolytes cardiac enzymes, thyroid function, and serum B₁₂ and folate should be investigated. An electrocardiogram should be recorded, and chest and skull x-ray pictures taken. All underlying systemic conditions must be treated before the stable mental state is assessed.

In patients with mild confusion in whom restlessness causes problems at home or in hospital sedation with a phenothiazine such as promazine 25 mg three times a day or thioridazine 12.5 mg two to four times a day may modify behaviour with very few side effects. We have found chlormethiazole edisylate (Heminevrin) 250 mg three times daily to be useful at home. Confused patients will accept these preparations more readily in syrup form. Over-enthusiastic sedation will expose an old person to grave dangers: drowsiness will make an ambulant patient liable to fall and in a bedridden patient will predispose to pressure sores, deep venous thrombosis, incontinence, or dehydration. The more time that is devoted to the patient the less need there will be for sedation. Contact with familiar attendants, a bright environment, and as few changes in surroundings as possible are together usually as valuable as tranquillising drugs.

Transient ischaemic attacks

Transient ischaemic attacks have been defined as the sudden occurrence of usually repeated episodes of sensory or motor impairment caused by temporary inadequacy of blood flow to localised areas of the brain that disappear completely within 24 hours.³ Most last for less than one hour and some for only a few minutes. Their clinical importance lies in the fact that they often warn of an impending completed stroke. Over one-third of patients with transient ischaemic attacks will have a major stroke within five years.⁴

The clinical picture depends on whether the carotid or the vertebrobasilar territory is affected. An important type of attack occurring in the carotid territory is amaurosis fugax, in which the vision of one eye is lost for a short time after interruption of flow in the ophthalmic artery, which is a branch of the internal carotid. Attacks involving the carotid circulation are more likely

to be followed by a cerebrovascular accident than those arising in the vertebrobasilar system.⁵

Most transient ischaemic attacks are caused by extracranial vascular disease, notably of the carotid tree near the bifurcation. Areas of atheroma may give rise to a stenosis or, more commonly, may ulcerate, become covered with friable thrombus, and provide a ready source of emboli. At first it seems strange that repeated embolism may reproduce identical transient neurological deficits; but flow in vessels is laminar, which means that emboli entering the circulation at the same point will tend to be carried to the same destination. Emboli thrown off from mural thrombus in the heart may also cause transient ischaemic attacks.

The major problem in patients who have multiple areas of arterial stenosis in extracranial vessels may be a failure of perfusion through collateral vessels. Inadequate flow may be secondary to systemic hypertension, reduced cardiac output, or increased blood viscosity. Anything that affects the quality of the cerebral perfusate (anaemia, anoxia, hypoglycaemia) may precipitate transient ischaemic attacks in susceptible individuals.

TREATMENT

General—Careful attention should be given to the exclusion of any systemic condition interfering with the quantity or quality of cerebral blood. Hypertension should be carefully controlled unless there is a surgically accessible major vessel stenosis which should be dealt with before the blood pressure is lowered. Special care should be exercised when using hypotensive agents in the elderly since they may appreciably reduce cerebral blood flow.

Surgical—Since angiography is not without risk it should only be undertaken in those patients who are considered fit for surgical intervention. If there is an operable stenosis endarterectomy should be performed, whether or not blood flow is thought to be reduced, since it is likely to be a source of emboli. The most usual area for surgery is the carotid bifurcation.

Medical—Long-term anticoagulants reduce the likelihood of cerebral infarction in patients suitable for surgery. Anticoagulation for one year may give the lesion a chance to heal by the epithelium growing over the ulcerated area, and the anticoagulant may then be withdrawn gradually with no recurrence of symptoms in many cases. This may be of particular advantage in the treating of the elderly patient with transient ischaemic attacks, when co-operation in drug taking may be unreliable. Anticoagulants are contraindicated in patients with severe hypertension, duodenal ulcers, liver disease, and alcoholism, or a mental state such that they cannot be relied on to take treatment regularly.

When platelets come into contact with intima damaged by atheroma platelet adenosine diphosphate (ADP) is released and irreversible platelet aggregation takes place, forming friable thrombus. Salicylates and other anti-inflammatory drugs are believed to block platelet ADP release. Aspirin 600 mg daily reduces frequency of attacks by reducing platelet "stickiness." In many centres it is now the usual medical treatment in transient ischaemic attacks.

Menière's disease

Menière's syndrome (paroxysmal vertigo with tinnitus and sensorineural hearing loss) may result from a variety of conditions, such as polyarteritis nodosa, syphilis, and trauma. The idiopathic form of this disorder is referred to as Menière's disease, and is associated with the finding of hydrops of the endolymphatic sac. The common underlying mechanism in these conditions is probably episodic labyrinthine ischaemia. An identical clinical picture may be caused by a blocked eustachian tube during an upper respiratory infection.

MANAGEMENT

General—The first objective in treatment must be the control of any predisposing condition. A salt-restricted diet and total avoidance of tobacco are usually advised. Treatment for Menière's disease should achieve either (a) suppression of vestibular function, (b) reduction of pressure in the endolymphatic space, or (c) vasodilatation within the labyrinthine circulation.

Medical—Objective assessment is difficult because of the heavy psychological overlay often present, but two forms of treatment, betahistine hydrochloride (Serc) and diuretic treatment, are more effective than a placebo in the control of vertigo.⁶ Betahistine hydrochloride has a specific vasodilator effect on labyrinthine circulation in pharmacological dosage.⁷ Because of its histamine-like action it should be avoided in acute gastric conditions, and should not be used with antihistamines. Chlorthalidone and hydrochlorothiazide has been used to try to reduce the fluid retention in Menière's disease. Psychotropic drugs which specifically suppress vestibular function, such as cinnarizine (Sturgenon) and prochlorperazine edisylate (Stemetil) are widely used. They may cause troublesome postural hypotension and balance upsets in the elderly.

Surgical—In unilateral disease in which medical treatment has failed, and hearing in the affected side is so impaired that no attempt need be made to conserve it, labyrinthectomy is the treatment of choice. When useful auditory function remains destruction of the vestibular labyrinth alone by ultrasound or cryotherapy may save hearing. Ultrasound may have a beneficial effect on endolymphatic hydrodynamics.

Treatment of acute attack—In many patients strict bed rest while the attack lasts is the only measure that affords any relief. Dimenhydrinate (Dramamine), if necessary given intramuscularly, may be useful when given early in an attack.

Disorders of balance

Balance disorders form a major contribution to disability in the elderly and the phase "falling about at home" often seems to be the sine qua non of geriatric referral. Disturbances of balance can present a considerable diagnostic challenge which is best faced from a simplistic view of the physiology of balance.

POSTURAL FIXATION

Postural mechanisms depend on information from proprioceptive, visual, and labyrinthine afferents and the integration of that information and appropriate motor responses by the ganglia, cerebellum, and cerebral cortex. The disturbances of balance associated with the involvement of the posterior column, visual upsets, and vestibular lesions are familiar. The areas of central integration, however, may be implicated in many cases. Cerebellar disease from any cause is associated with postural imbalance, most typically in unilateral lesions, in which the patient stumbles towards the affected side. In Parkinsonism involvement of the basal ganglia results not only in abnormal posture with a tendency to fall backwards but also in the loss of righting reflexes. Occasionally elderly patients are unable to stand, and if supported are unable to walk, although there is no demonstrable paralysis and they are able to perform pedalling movements when lying on their backs. This apraxia of gait is often the clinical end-point of a dominant hemisphere lesion which leaves no residual paresis.

A familiar but little-understood phenomenon in old people who have been confined to bed is the tendency to attempt to balance leaning backwards about 10° off plumb. They seem to be misinformed about the true nature of the vertical and will insist that they are upright. Exercises on a tilting table or raised heels may help those patients.

REFLEX ANTIGRAVITY SUPPORT

The erect position may be maintained by occasional bursts of activity in the leg and trunk muscles which act to correct any sway from the vertical. This is under reflex control of centres in the pons and medulla and brain stem ischaemia may be the cause of the drop attacks described by Sheldon,⁸ in which there is a sudden loss of tone in the legs. The commonest predisposing factor is postural hypotension but in some patients vertebrobasilar insufficiency plays a part. Postural hypotension occurs in 30% of old people over the age of 75 living at home. In severe cases the two most important factors are drugs with a hypotensive effect and cerebrovascular disease. Many old people who are unwilling to stand and are labelled unco-operative will be found to have unsuspected orthostatic hypotension.

MANAGEMENT OF RECURRENT FALLS

An awareness of the hazards facing the unsteady elderly person in his own home and a willingness to encourage preventive measures will avert occasional catastrophes. The old lady with a fractured femur should excite clinical curiosity. Why did she fall? Does she have undiagnosed disease or is her house

dangerous? A trusted general practitioner or health visitor will be able to suggest simple home improvements: a light in the dark passageway, no loose mats on slippery floors, a handrail on both sides of the stairs, beside the toilet, and bath, etc. Physiotherapy, including instruction in the use of a walking aid, will help to overcome the lack of confidence that is so often a major problem after a fall. Finally, attention must be given to current drug treatment. Many drugs, especially sedatives, will produce hypotension in the elderly and it should be understood that many of the preparations used indiscriminately for "dizziness" are sedatives and will only make the situation worse.

References

- ¹ Hackinski, V C, *et al*, *Lancet*, 1974, 2, 207.
- ² Judge, T G, in *Geriatric Medicine*, ed W F Anderson and T G Judge, p 107. London, Academic Press, 1974.
- ³ World Health Organisation, *Cerebrovascular Diseases: prevention, treatment and rehabilitation*, Technical Report Series no 469. Geneva, WHO, 1971.
- ⁴ Hass, W K, *Medical Clinics of North America*, 1972, 56, 1281.
- ⁵ Marshall, J, *Quarterly Journal of Medicine*, 1964, 33, 309.
- ⁶ Hinchcliffe, R, *Acta Oto-laryngologica*, 1972, 305, suppl p 10.
- ⁷ Martinez, D M, *Acta Oto-laryngologica*, 1972, 305, suppl p 29.
- ⁸ Sheldon, J H, in *Medicine in Old Age*, ed J N Agate, p 199. London Pitman Medical, 1966.

Talking with the Juniors

Angry moderates

FROM A SPECIAL CORRESPONDENT

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"We are angry moderates." "The unions, the patients—in fact the whole hospital community—are with us." These two comments highlighted for me two hours or so of conversation with some London-based junior doctors that took place before the Government's latest proposals for resolving the contract impasse (8 November, p 358). Like their provincial colleagues whom I had met a week or so earlier (18 October, p 157), none of them showed any political intent or extremist approach to the dispute. They were simply and absolutely "fed up with being pushed around," as one young surgeon put it.

"I don't mind how long I work," another surgical registrar declared, who had obviously accepted that medicine demanded unreasonable hours. But he expected a decent salary to compensate for the uncertainty of the training years and the domestic disruption. "I don't want to be worrying about the gas bill in the midst of a major abdominal operation." This group were certainly worrying about their gas bills, but as most of them were married this was not surprising (many families are worried about their household bills in 1975). But the point they made with some passion was that they would tolerate insecurity and a modest income if at the end of their long training the rewards were reasonable. Clearly, however, a consultant post in 1975 is not the attraction it was in 1965. Indeed, the plight of today's young consultants was recognised: they composed the sandwich generation—who had arrived to find their status a mirage,

professional satisfaction hard to achieve, and money for everything, including their salaries, scarce. Consultants are not unique in the community in suffering these frustrations, but the medical registrar thought that the doctors, as the only well-organised professional group, were being looked to by other professions to defend professional independence and to hold out against a decline in standards.

These registrars were all jealous of their professional freedom and were not happy about the new contract on this score—nor about the Government's attitude to independent practice—though they conceded reluctantly that it had seemed to be the only way of obtaining a decent financial reward in a state service. No one opposed the NHS; in fact, they wanted it to succeed, though the doctor from India was bitter about the misuse of overseas doctors by the Service. He had come to England with a good postgraduate qualification, yet had faced great difficulties in obtaining a suitable training post. "All we get offered is service posts in geriatric or psychiatric hospitals doing work that could be equally well done by nurses."

Clinical responsibility

This remark started an argument about what should be done by doctors in training. Several of the participants were from teaching hospitals and seemed reasonably satisfied with their training. Certainly they carried a great deal of clinical responsi-