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Medicine in Old Age

Diseases of the Motor System

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British Medical Journal, 1973, 4, 664-666

The Registrar General's *Statistical Review of England and Wales for the Year 1970* shows that 19% of all deaths in the over-65s were directly attributed to diseases of the nervous system. In addition, the 4% of all deaths in the same age group attributed to diseases of the arteries must include a proportion with central nervous system involvement. Mortality figures reflect morbidity (see fig.); every geriatric department contains more patients in this disease category than in any other. Though patients over 65 constitute only 12% of the total population, they occupy over one-third of the total N.H.S. beds—or a total of roughly 80,000 excluding psychiatric hospitals. Taken together, these facts indicate that elderly patients with diseases of the central nervous system must make formidable demands on the resources of the N.H.S. Moreover, with an annual rise of 100,000 in the over-65s this demand must continue to increase.

Disease of the Pyramidal System

By far the commonest disease of the central nervous system encountered is a stroke, varying from a disabling hemiplegia to a transient ischaemic cerebral episode with rapid recovery. A stroke with paralysis warranting admission to hospital is more common than acute appendicitis; a general practitioner with a list of 3,000 will see five new strokes each year and will have a similar number of disabled stroke survivors on his list. The annual stroke incidence is about two per 1,000 of the total population—which means 110,000 new stroke presentations each year in England and Wales. Three-quarters of all stroke patients are over the age of 65. In the United Kingdom 130,000 patients have appreciable impairment from a stroke and 93,000 of these are severely handicapped.¹

The onset of a stroke due to cerebral thrombosis or embolism is usually first noticed after a period of sleep or rest. The patient may rise from bed to go to the lavatory and promptly falls owing to paresis of the leg. Relatives may become alarmed because the patient is vacant and confused after an afternoon

nap; when the doctor arrives the patient appears normal again. This latter episode is typical of transient ischaemic cerebral episodes, which may recur with frequency.

The patient with a major stroke has motor weakness of both the arm and leg; initially there is flaccid muscle tone with

Semilogarithmic plot by age and sex of estimates of incidence and mortality in the Oxford Record Linkage area in 1963 for all types of stroke combined. (Acheson, R. M., and Fairbairn, A. S., *British Medical Journal*, 1970, 2, 621.)

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sluggish reflexes, but after a few days the tone increases and the jerks become brisk. The plantar response is extensor. The motor weakness is rarely equal in both arm and leg, one limb having more pareses than the other, depending on the arterial territory maximally involved. Occlusion of the anterior cerebral artery results in maximum motor weakness in the contralateral leg; occlusion of the middle cerebral artery produces paralysis maximum in contralateral arm and face, with aphasia should the dominant hemisphere be involved.

The last two decades have seen a revolution in the management and rehabilitation of the patient with a stroke.² In the early weeks after a stroke the mortality is about 50%; of the survivors, half regain independence in daily living activities outside hospital. Beyond question the best results are obtained by admitting all patients to special stroke units, where ancillary forces can be concentrated and where the patient's morale is maintained by a team spirit with patients pacing each other in the race to recovery; this is the best therapy to combat fatalism.

Most patients with uncomplicated hemiplegia spend two to three months retraining to stand and walk. Improvement in motor power can still be expected for up to eight months. Patients who progress to the point of social rehabilitation at home must be followed up for at least two years in the outpatient department or in day hospital. Careful neurological analysis of every stroke patient pays great dividends in successful rehabilitation. For most patients recovery is slow but persistent; for those with delayed recovery it is imperative to identify the barriers to progress;³ these include dysphasia, cortical sensory deficit, impaired muscle joint position, or an associated psychiatric disturbance. A discussion with the physiotherapist and occupational therapist on the reasons for delayed recovery is essential.

When discharge is planned it is prudent for the occupational therapist to visit the house and advise on what help for the handicapped⁴ is required in the way of a supporting rail, ramp, or aids to daily living in the kitchen.

PSEUDOBULBAR PALSY

During recovery from a stroke a second cerebrovascular accident may occur on the opposite side. If both areas of vascular occlusion are in the region of the internal capsule then pseudobulbar palsy may result. Pseudobulbar palsy occurs in patients who are hypertensive and is characterized by emotional lability, a spastic tongue, brisk jaw jerk, and an upper motor neurone lesion in all four limbs; death results from aspiration pneumonia. The emotional lability is distressing for patient and relatives but alleviation can be obtained with imipramine⁵ or amitriptyline.⁶

BRAIN STEM ISCHAEMIA

Vascular insufficiency or occlusion in the posterior cerebral circulation results in one of the brain stem syndromes. Here the patient is prostrated with severe giddiness, double vision, vomiting, and ataxia. On examination, there is nystagmus, intention tremor in the ipsilateral limbs, and numbness in the ipsilateral face and contralateral side of the body. Ipsilateral involvement of a cranial nerve may be present. Despite the alarming onset the prognosis is good.

MOTOR NEURONE DISEASE

As in younger people, motor neurone disease is characterized by weakness and wasting in all four limbs, fasciculation being maximum in the shoulder and pelvic girdles; all limb reflexes are brisk, the plantars are extensor and there is no sphincter disturbance. However, unlike the younger age groups, the prognosis is much more favourable with survival for more than a

decade. Even with bulbar involvement there is a relatively better prognosis.⁷

Disease of the Extrapyramidal System

In 1817 an East London general practitioner, Dr. James Parkinson, published *An Essay on the Shaking Palsy*, giving a lucid description of the clinical manifestations of disease of the extrapyramidal system. Every general practice contains several patients with Parkinsonism easily remembered both by their appearance and by the "repeat prescription" list. Parkinsonism or paralysis agitans is due to disease of the basal ganglia. Primary degeneration of unknown aetiology is the commonest cause, with degeneration secondary to vascular disease or to encephalitis lethargica being much less frequent. Drugs can cause Parkinsonism, the chief offenders being methyl dopa, reserpine, chlorpromazine, and the phenothiazines; haloperidol is notable for causing the rapid production of Parkinsonian features. Parkinsonism caused by drugs is dose-dependent. Carbon-monoxide intoxication and manganese poisoning are often quoted but rarely seen aetiologies of Parkinsonism, but most geriatric departments have an ex-boxer with this disease.

Clinically Parkinsonism is recognized at a glance, from the fixed expressionless gaze, infrequent blinking, and festinating walk in small steps with bent attitude. Roughly half the patients exhibit the coarse compound tremor, which may affect any or all of the limbs as well as the jaw. The tremor is present at rest but disappears when the patient voluntarily uses the limb or falls asleep; anxiety worsens the tremor. The limb reflexes are normal. The postencephalitic type of Parkinsonism, with seborrhoeic skin, drooling saliva, and oculogyric crisis, is rarely seen because there are now few survivors of the 1920 epidemic.

Treatment

The introduction of L-dopa represented a major advance in therapeutics. Unfortunately, not all patients are suitable for this therapy; it is a good working rule that to benefit a patient must be clear in the head and be prepared to tolerate the nausea during the "build up" period until the improvement is obvious.⁸ Initial dosage must be small—for example, L-dopa 0.25 mg thrice daily after food. Dosage may be increased by 0.5 g every fourth day, but if nausea and vomiting cannot be tolerated then reversion to a smaller dose with a slower "build up" must be made. Nausea may also be reduced by using one of the slow release preparations such as Broca Dopa temp tabs. Months rather than weeks should be the aim in reaching the optimum dosage of 5 g daily, always remembering that maximum improvement may take six months to occur. Once a patient has increased mobility, a sense of wellbeing and renewed vigour for daily living activities she will not hesitate to persist with L-dopa. Patients frequently receive tonics in the form of polyvitamin preparations; pyridoxine (vitamin B₆) cancels the clinical action of L-dopa and since it is present in many vitamin mixtures care must be taken to avoid it. Like all powerful drugs, L-dopa has serious side effects: depression, anxiety, or confusion may force withdrawal of the drug. Abnormal involuntary movements may occur affecting the face, neck, and spinal muscles or even causing rhythmic movements of the abdominal wall. Cardiac arrhythmia and hypotension are hazards in patients with pre-existing coronary artery disease. Fortunately all the toxic effects are dose-dependent. The introduction of L-dopa decarboxylase inhibitors promises the maximum benefit of L-dopa with minimum toxic effects.

Because of the danger of hypertensive crisis monoamine-oxidase inhibitor drugs must never be administered at the same time as L-dopa. Sympathomimetic agents must also be avoided.

Conventional anticholinergic drugs such as orphenadrine

50 mg thrice daily may safely be given to supplement therapy with L-dopa. In patients unsuitable for L-dopa therapy the anticholinergic drugs may still be useful, but it is important to remember that they can precipitate acute glaucoma or acute urinary retention. Amantadine has been disappointing in treatment of the patient with Parkinsonism but is always worth a trial where the other two lines of therapy have failed.

HEMIBALLISMUS

A vascular lesion of the subthalamic nucleus results in sudden onset of violent choreic movements in the contralateral half of the body. The violence of the movements rapidly exhausts the patient and involvement of the pharyngeal muscles may result in aspiration pneumonia. Tetrabenazine is effective in relieving the movements but the movements can be abolished only with thalamotomy.

Lower Motor Neurone Lesions

Polyneuropathy represents the commonest lower motor neurone lesion of old age and is characterized by bilateral flaccid muscle weakness maximum in the distal limb muscles, always worse in the legs. There may also be sensory loss of the stocking-and-glove type. Most cases of peripheral neuropathy are mixed motor and sensory. Asymmetry is most unusual in either motor or sensory component.

The clinical signs are weakness, sluggish or absent reflexes, wasting of the muscles, and tenderness of the muscles and soles of the feet; tenderness is a prominent feature of alcoholic neuropathy but may occur in the acute stage of any neuropathy. The sensory loss usually involves all modalities of sensation. Plantar responses are flexor or absent.

Diabetes mellitus represents the most likely cause of a polyneuropathy in any old person. Routine urine testing cannot rule out the diagnosis—nor can one estimation of the blood sugar level. A glucose tolerance test should be performed in all cases when both urine tests and blood sugar levels are inconclusive. Most elderly diabetics are obese and management of the polyneuropathy aims at arresting its progress by good diabetic control, preferably by diet, supplemented if required by a

sulphonylurea or a diguanide preparation. Diabetic peripheral neuropathy may be purely sensory and may affect only muscle joint and vibration sensations—hence the description of this condition as “diabetic pseudotabes.” These patients may develop perforating ulcers in the feet and, with vascular impairment, great care must be taken in chiropody.

Polyneuropathy may be the presenting manifestation of a carcinoma, especially carcinoma of the bronchus, occurring long before the primary neoplasm is obvious. Herpes zoster, even a mild and limited infection, may be complicated by polyneuropathy.

Deficiency of vitamin B₁₂ either in Addisonian pernicious anaemia or in a malabsorption syndrome may result in polyneuropathy, but this may be associated with posterior column signs to cause subacute combined degeneration. Systemic therapy with vitamin B₁₂ will reverse the lesion in the lower motor neurone but will only arrest the lesion in the posterior column.

The extent of alcoholism is probably underestimated in elderly women, who prefer to avoid drinking in pubs. In addition to peripheral neuropathy, these patients may develop a gross memory defect or become very confused: hence Korsakoff's psychosis may be diagnosed as senile dementia. The value of Parenterovite is questionable, but it seems to provide psychotherapy for the officiating physician and a jolt to the sobering patient.

Drugs which may cause polyneuropathy are the inorganic arsenicals, gold, sulphonamides, nitrofurantoin, and isoniazid.

Patients with polyneuropathy of any aetiology will become disabled and unable to walk. While the primary aetiology is being treated ambulation must be maintained or regained with physiotherapy.

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