Current Practice

PRACTICAL NEUROLOGY

Management of Major Strokes

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The word stroke emphasizes the rapidity of onset but not the cause of the illness. Its clinical evolution is exemplified by maximum disability in a few hours and some, albeit slight, evidence of improvement within 48 hours. The neurological deficit may be hemiplegia, with or without aphasia, or tetraplegia with ocular and bulbar signs, depending on whether parts of the brain within the territory of supply of the carotid or basilar arteries are most affected. There will have been transient loss of consciousness in many cases and in a few prolonged coma. Most patients have cerebral vascular disease, but in some, 2% to 5% in various series, the stroke occurs in association with conditions such as brain tumour or is an integral part of another disease as in neurosyphilis. Error in diagnosis is promoted when patients with cerebral vascular disease present with headache and signs progressing over days—features suspicious of a tumour. Conversely, patients without vascular disease may have an illness with sudden onset and some recovery. Even within the cadre of cerebral vascular disease the distinction between thrombosis, haemorrhage, and embolism is often not possible. Thus a stroke progressing over days is not uncommon in carotid artery occlusion; cerebral haemorrhage may not be associated with loss of consciousness; cerebral embolism need not have an abrupt onset or a clinically recognized source for the embolus. The need for a continual and flexible assessment of each patient is thus apparent.

Preventive Treatment

One aspect of prevention that should be emphasized is the danger of sustained awkward neck postures in the middle-aged and elderly. Patients have been seen whose stroke may have been precipitated by sleeping on a too short sofa (a chaise-pas-assez-longue syndrome) or in a chair. The contraindications to the various oral contraceptive preparations are not yet fully appreciated, but probably these drugs should not be prescribed for women with a history of hemiplegic migraine.

First Few Hours

When the diagnosis of infarction or haemorrhage is certain a few patients, conscious, with hemiplegia, and with excellent home conditions, can be nursed at home. The doctor should have been assured by a relative that the patient has been in stable health for the previous months. The patient should be seen again within 12 hours to confirm that he can swallow and that there is no retention of urine.

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Hypothermia.—Strokes which are the result of ischaemic infarction tend to occur in the early morning, though the patient may state that his illness started when he woke some hours later. A lower body temperature during sleep is a normal diurnal variation, but this will be greatly exaggerated when, after a stroke, the patient becomes immobile in a cold environment, perhaps having fallen unconscious just after attempting to rise out of bed. The rectal temperature should always be taken with a low-reading thermometer. If the reading is below 95° F. (35° C.) the patient should be wrapped in blankets, taken to hospital, and nursed in a warm cubicle. Hydrocortisone (100 mg.) can be given by intravenous drip.

Respiratory difficulty may arise during the transport of the patient. At home and in the ambulance the mouth should be cleaned with care, a clear airway established, and the patient placed in a prone-lateral position and accompanied in the ambulance by a trained person. The quip of an anaesthetist colleague is very true: "If the patient sees heaven he will soon be in it."

Management in Hospital

The principles are as follows:

(1) Limit brain damage by immediate attention to the body systems. Do not overlook an aetiology for which early or special treatment and investigation is required.

(2) Be vigilant when after 48 hours there is no distinct clinical indication of improvement.

(3) Know the scope, limitations, and hazards of the more specialized investigations.

Limiting Brain Damage and Early Treatment

Around the destroyed area there is a considerable amount of non-functioning brain capable of recovery. In cerebral infarction this tissue must be given the maximum chance of survival until the regional anastomotic circulation facilitates a less critical cerebral blood flow. Since 1890 carbon dioxide has been recognized as a most potent agent in increasing cerebral blood flow. There are conflicting views on whether blood flow in the affected cerebral hemisphere is actually increased by carbon dioxide in the first week after a stroke. Studies of regional cerebral blood flow suggest that there is already maximum vasodilatation in the ischaemic area and that carbon dioxide inhalation therapy would dilate vessels in the normal area with the possibility of a shunt of blood away from the site of damage. Partial oxygen pressure in the brain-damaged areas is of profound importance, but regional and general regulation of oxygen within the brain is complex in disease and

not yet sufficiently clear for guidance to be given to the physician. Intermittent humidified oxygen inhalation, 20 minutes per hour, especially where there are respiratory complications, should be given by a mask which allows an approximate O_2 concentration of 25% to 40%. An accompanying heart lesion would have serious consequences, and some studies suggest that 20% of patients with strokes have had a previous myocardial infarction. A history of cardiac pain is rarely obtained, partly because of loss of memory and communication. However, electrocardiographic change without infarction may occur when the brain-stem is damaged. There may be an established or a paroxysmal cardiac arrhythmia. Low blood pressure (below 100 mm. Hg systolic) would decrease cerebral blood flow, but in strokes it is more often found to be normal or elevated. Hypotension is treated by elevating the foot of the bed, controlling cardiac failure and arrhythmia, while in a few patients corticosteroids may be needed. The physician should be alert for respiratory complications, and careful nursing and care of the skin are important.

For the first 24 hours after a stroke, unless there is obvious dehydration, the intake of fluid may reasonably be limited by the patient's ability to swallow. When the ability to swallow is unlikely to recover quickly feeding by an oesophageal tube is necessary. A rectal examination should always be done on the first day for impacted faeces and prostatic enlargement, and a bladder regimen initiated. Catheter drainage should, if possible, be avoided. A full blood count, serum electrolytes, cholesterol, urea, blood glucose, erythrocyte sedimentation rate, serum B_{12} and folic acid, and the Wassermann reaction should be determined on admission, and some of these tests may need to be repeated at intervals.

Treatment of patients who are deeply unconscious from any type of cerebral vascular disease has been most discouraging. Hypoglycaemia, spontaneous or induced, though rare, must never be overlooked. The result of the blood sugar taken on admission should be quickly sought. Empirical treatment on admission with intravenous 10% dextrose and an intravenous vitamin preparation is not always unrewarding. Fulminating meningitis and acute haemorrhagic leucoencephalitis can have an apoplectic onset, and when a patient is in coma neck stiffness may be slight. If the patient has a high temperature (without signs of brain-stem damage) and there is a high polymorphonuclear leucocytosis in the blood, these diagnoses should be carefully considered.

Anticoagulants

A relatively young person (under 55 years, for instance) who has rheumatic heart disease, is conscious, and has had an abrupt onset to the stroke should be given an anticoagulant drug such as warfarin sodium as early as possible in the illness and continued for some months or indefinitely. Cerebral embolism does occur in rheumatic patients in regular cardiac rhythm. Hypertension, some neck stiffness, and marked impairment of consciousness might suggest cerebral haemorrhage and would contraindicate this treatment. Unfortunately, a clear cerebrospinal fluid can be found with cerebral haemorrhage, but a clear fluid is an added assurance before starting anticoagulants. Atrial fibrillation without rheumatic heart disease is not an indication for anticoagulant drugs in older patients but could be considered in the younger.

The occurrence with sudden onset over a few days of discrete episodes of retinal occlusion or aphasia or so-called "step-ladder" or "stuttering" increase in limb weakness is strong, though not conclusive, evidence of ischaemic or embolic cerebral vascular disease and anticoagulation for some weeks is reasonable. In the past, anticoagulant therapy was advised for patients with hemiparesis progressing over days (stroke in evolution), but the differential diagnosis between infarction, cerebral haemorrhage, and a tumour is very difficult. For this reason anticoagulant treatment should not be given without advice from a neurological surgeon or physician. However, where there are signs (ophthalmoplegia, dysarthria, and dysphagia) indicating progressing involvement of the brain-stem, and the patient is conscious, vertebrobasilar artery occlusion (from thrombosis or emboli from atheromatous plaques) and not haemorrhage is likely. In this situation subcutaneous heparin, 7,500 units six-hourly (each abdominal quadrant in turn) should be given for 48 hours and warfarin sodium for a few weeks. General contraindications to anticoagulants include renal and liver disease, bleeding diastheses, and hypertension (diastolic pressure above 110 mm. Hg), and one should be cautious about even short-term use when the patient is over 65 years. The use of a fibrinolytic drug has so far not been satisfactory in the treatment of completed stroke.

Surgical Treatments

In the small group of relatively young patients with strokes but without rheumatic carditis, hypertension, or any predisposing general disease cerebral angiography should be performed. A haematoma from an angioma or aneurysm which has bled intracerebrally may be treated surgically. In the occasional patient who improves but slowly over a period of a week and then complains of headache, and has increasing neurological signs, aspiration of an intracerebral clot may lead to dramatic improvement. A disturbing problem is hemiplegia around the time of the puerperium in a patient without eclampsia. Anticoagulant drugs are not advisable. The diagnoses of the stroke in four such patients seen in recent years were cortical cerebral thrombophlebitis, occlusion of the middle cerebral artery, internal carotid artery occlusion in the neck, and an intracerebral haematoma. The emergent recommendation is initially to assume and treat cerebral oedema and epilepsy if present; if after some days there is unsatisfactory progress, cerebral angiography should be performed. Surgery for the removal of clots in the neck vessels has not been successful treatment in major strokes.

Systemic Disease

Giant cell (cranial) arteritis, subacute bacterial endocarditis, and atrial myxoma among many diseases do, though rarely, present as strokes. The cranial arteries should be palpated daily for tenderness and pulsatility and a sedimentation rate estimated twice weekly. If this is persistently high and there is some anaemia and no evidence of infection, prednisone 15 mg. thrice daily should be given even without an arterial biopsy. For the diagnosis of bacterial endocarditis and myxoma of the atrium, unusual acumen, perspicacity, discernment, and luck are needed. Cerebral thrombosis occurs in neurosyphilis, and a search for previous documents and serological tests should never be omitted.

Why Patients with Strokes are Not Improving after 48 Hours

Between 24 and 48 hours after the onset the patient should be showing either distinct improvement in consciousness or in the paralysis, or at the very least no worsening. Some reasons for delay in recovery, which may occur in various combinations, are shown in Table I.

TABLE I.—*Why Patients with Stroke Are Still Not Improving 48 Hours After Onset*

Head injury sustained at time of apoplexy
Hypothermia and hyperpyrexia
Respiratory complications including pneumonitis
Alteration in respiratory rate with hypoventilation or hyperventilation
Injudicious medication including insulin and morphine
Electrolyte imbalance and uraemia
Renal tract or other infections
Myocardial infarction and cardiac arrhythmia with or without hypotension
Associated disease, particularly the anaemias, diabetes, and polycythaemia
Cerebral oedema including that associated with severe hypertension
Epilepsy developing after a stroke
A further or extending cerebral infarct, embolus, or haemorrhage
Not cerebrovascular disease but "stroke" caused by cerebral tumour, abscess, or subdural haematoma.

Signs of head injury should be sought. Cerebral oedema as well as other causes of increased intracranial pressure is

suggested when there is increasing clouding of consciousness, the development of bilateral signs where previously there was hemiplegia, and the blood pressure is rising. Treatment can be started with oral glycerol 1 g./kg. body weight given four-hourly and by a steroid preparation such as dexamethasone 4 mg. intramuscularly six-hourly. When signs of cerebral oedema develop acutely (or there is evidence of cerebral oedema and the patient is to be conveyed for a long journey by ambulance to a specialist unit) 200 ml. of 25% mannitol intravenously given over 20 minutes and 8 mg. of intravenous dexamethasone is effective. This has superseded 50% glucose, which irritates veins and may damage renal tubules. If the diastolic blood pressure still remains consistently over 125 mm. Hg and is continuing to rise, intramuscular reserpine should be given in a dose which would lower the level to between 110 and 120 mm. Hg. A portable *x*-ray examination of the chest would help to indicate the nature of any respiratory complication (a metastatic lung neoplasm may present with hemiplegia, and a radiograph of the chest should always be done at some stage). Epilepsy should be treated with phenytoin sodium and phenobarbitone. The distinction between further or extending cerebrovascular disease and a non-vascular cause of stroke is crucial as the justification for any hazardous investigation.

Special Investigations

A brief critique of the more specialized investigation is presented in Table II.

TABLE II.—*Special Investigations in the Management of Strokes*

Test	Designation	Value	Limitations
Straight radiograph skull	1	Detects a pineal shift	Pineal may not be calcified
Echoencephalogram	1	Easy bed-side test. May be repeated frequently	Capricious results unless performed by one expert
Thermography		Asymmetry of frontal skin temperatures suggests carotid artery occlusion	Many false negatives
E.E.G.	1	Tumour can be suspected from serial records	Non-discriminative in first few weeks after stroke
Brain scan	2	Safe, most helpful, reliable procedure when tumour is still suspected several weeks after stroke	Unhelpful in differential diagnosis in first few weeks. Availability restricted
Cerebral circulation transit time	2	If normal prognosis said to be good	Gives no information of regional blood flow
Lumbar puncture	*2	Will diagnose subarachnoid haemorrhage, meningitis, syphilis	Can precipitate coning even when papilloedema is absent
Cerebral angiography	*3	Gives aetiological as well as anatomical information (i.e. subdural haematoma, angioma, or tumour may be diagnosed)	May need general anaesthetic. More dangerous when there is cerebral oedema

* Discussed in text.
1 A safe non-traumatic and generally available test which could be routinely done.
2 An easy test to perform but either some danger or facilities limited.
3 A highly skilled procedure which is potentially hazardous.

Lumbar Puncture.—This should not be considered as a routine test in a hospital without a neurological or neurosurgical unit. Lumbar puncture will lead to the diagnosis of subarachnoid haemorrhage, meningitis, and syphilis, and is therefore necessary when these are suspected ; it should be performed before starting anticoagulant drugs. It is less widely realized that a lumbar puncture may precipitate deterioration in patients with brain tumours, even in the absence of papilloedema. The investigation should therefore not be done as a method of distinguishing between vascular and neoplastic causes of stroke. Estimation of the pressure of the cerebrospinal fluid is not always easy, and an elevation of protein and even of the leucocytes occurs in both.

Cerebral Angiography.—In skilled and experienced units morbidity from the test is low. Carotid artery angiography is a partial examination, and aortic arch and brachial or axillary arteriograms are required for examination of the vertebrobasilar artery system. Angiography should be performed only after consultation with the neurological physician and especially the surgeon who might be expected to treat any lesion demonstrated or any adverse outcome of the test.

Aftercare and Rehabilitation

One must be prepared to put in a great deal of effort and resourcefulness without expecting quick or spectacular success. There are many factors determining success, which range from the degree of brain damage in the patient to the number of skilled staff available (Table III). In some patients the residual deficit is not clearly defined even three weeks after the onset of the stroke, and one must avoid a too hasty conclusion that any intellectual disability is likely to be permanent. Nevertheless, at about three weeks a rehabilitation programme should be planned for each patient, based on a careful assessment of physical, intellectual, and social circumstances.

Postural Hypotension.—Brain-stem damage and recumbency will predispose to postural hypotension. During rehabilitation the blood pressure sitting and standing must always be compared with that in the reclining position. Random checks for postural hypotension should be made as the patient is allowed to sit up for long intervals. If postural hypotension is present a period of "vasomotor training" with a tilting bed is needed and an abdominal binder and ephedrine 30 mg. b.d. sometimes help.

Hypertension.—Clear evidence of malignant hypertension and persistent severe hypertension in the younger mobile patient are the only particular indication for treatment of patients with hypotensive drugs. Considerable experience with the drug chosen is needed to reduce blood pressure moderately and avoid postural hypotension.

Physiotherapy.—This starts with the patient's admission. Gentle passive movement of the limbs through a complete range of movement several times daily should prevent contractures. The development and degree of spasticity are very variable. The techniques of "proprioceptive neuromuscular facilitation" propounded by Mrs. Berta Bobath have been found valuable. Patterns of movement, especially those initiated from the trunk, are encouraged early and there is less emphasis initially on the unaffected limbs. Some of the exercises in this system are too strenuous for the ill and fragile patient. There is correct attention towards the development of the positive supporting reaction in the hemiparetic lower limb, and "bed-ending" exercises will

TABLE III.—*Factors on Which Patient's Prognosis and Eventual Placement will Depend*

Patient			Home and Hospital		
Physical	Higher Integrative Functions	Past Attainments	Relatives	Hospital Services	Community Services
Degree of paralysis Discriminative sensory loss Joint position sense loss Presence of "positive supporting reactions" Postural hypotension Other causes of postural vertigo Hypertension Hearing and seeing Cardiac insufficiency Respiratory reserve Incontinence of urine	Dementia Language disturbance Neglect and denial of hemiparesis Motor perseveration Memory loss for immediate events Loss of postural fixation Disturbance of body image Apraxia Catastrophic reactions Depression "Don't want to do it"	Occupation Hobbies and skills	Willingness to help Intelligence Suitable housing	A well-equipped assessment and rehabilitation unit A day hospital	Home help Home physiotherapy Special appliances

demonstrate the effectiveness of its return. Sensory deficit, denial of disease, and other "mental barriers" often lead to more disability than loss of power. Swelling of the legs results from immobility and the influence of gravity. Physiotherapy, tilting chairs, and planned posture are required, and diuretics should be used only where there are systemic causes of oedema.

Incontinence of Urine.—This may be present in some patients who are not demented, and its cause is often elusive. Urinary infections should be controlled, and if a two-hourly bladder regimen is not successful much thought is needed, and urological advice should be sought for a safe urinary appliance or other treatment. Very rarely focal epilepsy is the cause, when phenytoin 100 mg. is useful.

Language Disturbances.—An important mental barrier to recovery is aphasia. It is also distressing to the patient and his relatives and visitors. Patients who have a degree of mixed cerebral dominance tend in general to recover language better than those with strong left hemisphere dominance. Expressive aphasia with good comprehension is eventually less of a handicap than word fluency with poor comprehension. Some reading difficulties may be dyslexic, while some are related more to scanning or visual field defects. It is essential not to misdiagnose dementia when in fact there is mainly a loss of language. The speech therapist has an important role in treating aphasia. The aphasic patient may learn to communicate in a faculty that has been preserved, such as by miming or drawing. It is known that one musician composed a great work even when he had expressive and receptive aphasia.

Depressive illness may retard progress in rehabilitation and any psychiatric aspects that compound the effects of brain damage require urgent treatment. Vasodilators (cyclandelate) are occasionally used, but their effectiveness is still in doubt.

Community and Hospital Services.—These, when well planned and staffed by dedicated people, are very useful. The physician should review the needs of each of his patients before they leave hospital. The home facilities should be inspected, and hand-rails, ramps, and toilet modifications made, and special appliances (Zimmer aid, chair, etc.) provided. A day hospital aids follow up, and can provide continued help from speech, occupational, and physiotherapists.

Envoi

The patient who has had a stroke is currently being treated by general physicians, geriatricians, and neurological physicians and surgeons. The long-term supervision is often taken over by the geriatric physician and the psychiatrist. Prognosis in terms of the number of patients who return to their previous employment has not been assessed, though 60% of patients should achieve self-care and some independence. This outline of management is based on experience of admissions to hospitals, the siting of specialist units, and the provision of aftercare in this regional area. It is suggested in this paper that referral for specialist advice and admission to a specialist unit are usually needed for progressing strokes and where there is evidence of an expanding intracranial lesion. It is most important to assess all the body systems within the first few days of a stroke. Treatment is active, but not confined to the nervous system.

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ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Unequal Legs

Q.—*Is there any surgical operation to equalize the legs in cases of children in which one leg is congenitally shorter than the other and in which the shortage appears to be due to a defect in one tibia?*

A.—There are a number of operations for leg equalization in children. The defect in the tibia is not specified, but if this is partial absence there is likely to be considerable deformity of the foot. In such cases this overrides the problem of shortening. A defect of the tibia alone does not often give rise to notable leg shortening.

Leg equalization is possible by operative lengthening of the short tibia. This is an operation which over recent years has been demonstrated as a successful and safe method. It is usually performed between the ages of 8 and 14, and at least 2 in. (5·1 cm.) can be gained. When necessary the operation can be repeated. The operation has technical difficulties and needs an experienced surgeon to undertake it, but is then a safe procedure.

In an older person or in a tall child operative shortening of the normal, long leg is safe and easy. Not unnaturally it is unattractive to the patient but is frequently used. The third available method is by arresting the epiphyses of the long leg at an appropriate time to make the legs equal. It is probably the least suitable of the three methods mentioned.

Atypical Facial Pain with Cervical Spondylosis

Q.—*What is the reason for bizarre symptoms in the forehead and face, suggestive of sinusitis, in association with cervical spondylosis? What is the best management for this type of spondylosis?*

A.—It is assumed that the term cervical spondylosis is used to describe patients in whom radiographs of the cervical spine show evidence of degenerative change in the apophysial joints and discs, and whose neck movements in general are mildly restricted. That excludes patients with an acute mechanical torticollis and those with overt signs of spinal cord involvement. Frontal and facial pain is not a common symptom in patients with cervical spondylosis thus defined.

Campbell and Lloyd[1] made a careful assessment of 40 patients with atypical facial pain, and concluded that it was most likely to be due to stimulation of the superior cervical sympathetic ganglion with secondary liberation of histamine. They could not accept that cervical spondylosis was the main cause. Their patients gained greatest benefit from immobilization of the neck in a plaster collar.

Atypical facial pain differs from migraine in that it lasts much longer or is continuous and is not associated with visual disturbance. Focal neurological signs, particularly a third-nerve palsy, would suggest a carotid aneurysm and call for an arteriogram. Careful investigation should exclude local causes