THE MANAGEMENT OF THE ACUTE PHASE OF STROKE ILLNESS

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Early management of the stroke patient in hospital has been the subject of two audit meetings of the Department of Medicine in Lancaster. Case notes of recent admissions with stroke were critically reviewed and evaluated against the advice given in the King's Fund Forum Consensus Statement of 1988[1], along with more evidence from the Oxford Community Stroke Project [2]. Following further discussion by consultants and junior staff it has been possible to produce a protocol for management of these patients. The essence of the brief protocol is given in the accompanying tables.

INTRODUCTION

Stroke illness carries a very high mortality with about half the victims dying in the early weeks of the illness[3]. The majority of the survivors will have permanent significant disability and mortality after the first year is of the order of ten per cent per annum, deaths being from cardiac and cerebral events [4]. With this in mind, the management of acute stroke naturally evolves around the four objectives of enhancing the chances of immediate survival, maximising the natural recovery process, minimising the subsequent disability and preventing further strokes or premature death from other cardiovascular events.

HOSPITALISATION

Neither the audit, nor this paper based on it, attempts to explore the case for the hospitalisation of strokes, though it is difficult to envisage in particular the investigations and nursing management being successfully achieved in the average community setting. Hospitalisation certainly facilitates our first objective, though with skilled and well-resourced community care the latter three objectives may be achieved without it. In practice the decision to hospitalise stroke patients is not the result of scientific soul-searching but is a pragmatic response either to circumstances surrounding the acute event, e.g. a 999 call, or to the perceived social circumstances, e.g. living alone, infirm carer.

INITIAL ASSESSMENT

The History (Table 1)

Table 1 – History Points

Details of onset and time sequence
Sources of information
Recent head injury
Past medical history
Age
Domestic background
Pre morbid health status

The aim in taking the history is to answer the question: 'is this truly a stroke or is it some other type of acute cerebral and neurological disturbance?' Information may have to be obtained from sources other than the patient such as relatives, or witnesses to the acute event, or it may be largely circumstantial, being obtained from neighbours, policemen who have broken in or ambulance crews. Special points in the history-taking are to clarify a clear time sequence of events and to seek any history of a recent head injury. The past medical history is particularly relevant for establishing the probability of a stroke and for predicting future events (Table 2).

Table 2 – Past Medical History

Myocardial infarction/Ischaemic heart disease
Treated hypertension
Valvular heart disease
Peripheral vascular disease
Diabetes
Previous strokes/Transient ischaemic attacks
Alcohol and smoking habits
Drug history (especially hypertensive and diabetic drugs)

The age of the patient is relevant to the diagnosis as stroke becomes increasingly common as age advances, and conversely classical stroke from cerebrovascular atherosclerosis is rare below the age of 55 years.

It is important to collect additional information about the patient's domestic arrangements and his primary carer's health status, and to have some clear idea about the patient's pre-morbid level of activity, independence, mental state and continence, so as to provide a base line against which progress in recovery and rehabilitation can be measured.

THE EXAMINATION

It is important to examine the patient carefully and fully at the onset and to repeat the process as the stroke proceeds because the neurological features may change markedly in the first 48-72 hours. Functional neurological assessment is as important as noting discrete neurological deficits (Tables 3 and 4).

Table 3 – Discrete Neurological Deficts

Conscious level
Cranial nerve lesions
Visual fields
Facial asymmetry
Power deficit in muscle groups
Abnormal reflexes

Table 4 – Functional Neurological Assessment

Speech – expressive and receptive
Swallowing
Co-ordinated movement, eg. feeding
Balance
Continence
Apraxias
Inattention
Denial of disability
Emotional lability

Evidence of other cardiovascular disease should be looked for since its presence influences the probability of stroke, its prognosis, and the likelihood of recurrence (Table 5).

Table 5 – Cardiovascular Features

Hypertension
Atrial fibrillation
Cardiomegaly
Heart murmurs
Aneurysms
Carotid abnormalities
Absent peripheral pulses

Finally, any other abnormal clinical features should be noted, in particular the presence of co-existing musculo-skeletal disability such as arthritis, or chronic cardio-pulmonary insufficiency, both of which are likely to have adverse effects on rehabilitation.

DIAGNOSIS

The majority of clinical presentations that look like a stroke are a stroke, but the diagnosis should aim to define the situation precisely. It is not possible to differentiate clinically between thrombosis, haemorrhage and embolism. The last may often be inferred from circumstantial evidence but the first two can only be differentiated by C.T. brain scan. It is useful to stage the stroke process and also to form some idea of prognosis, which the patient and his relatives will ask about. Finally if the diagnosis does not 'hang true' consider other possibilities (Table 6).

Table 6 – Alternative Diagnoses

Post ictal
Hypoglycaemic
Overdose
Subarachnoid haemorrhage
Space-occupying lesion including blood collections
Cerebral infections – bacterial, viral, protozoal and abscesses
Arterio-venous malformations

INVESTIGATION

Most strokes require relatively simple routine investigation with more complex procedures being reserved for selected cases. Lumbar puncture and C.T. brain scan are not routine but indicated occasionally to clarify diagnostic uncertainty (Table 7), (Table 8)[1].

MANAGEMENT

The nursing needs are simple but time-consuming and are summarised in Table 9. The physiotherapist needs to be working with the nursing staff at this early stage. Feeding and fluid administration can be difficult initially, some degree of incoordinate swallowing is common but fortunately tends to improve with time. Intravenous fluid may be needed but rarely I.V. feeding. Deeply unconscious strokes present a special dilemma since they cannot be fed orally and have a poor prognosis as regards survival and future recovery. It is very important to involve the relatives in any difficult decisions which have to be made.

Table 7 – Investigations

Routine:	Full blood count, erythrocyte sedimentation rate biochemical profile, blood glucose, electro-cardiogram, chest X-ray
Special:	Cerebral spinal fluid examination, computerized tomographic brain scan
In selected patients:	
	Syphilis serology
	Sickling
	Cholestrol/lipid levels
	Carotid ultrasound → Carotid angiograms
	Echocardiography
	Skull X-ray

Table 8 – Major Indications for C.T. Brain Scanning in Stroke

Uncertain diagnosis of stroke
Current or contemplated anticoagulant therapy (to exclude haemorrhagic stroke)
Cerebellar hamatoma suspected
Carotid endarterectomy proposed
Suspected subarachnoid haemorrhage
Young patient

Table 9 – Nursing Management

Prevention and treatment of:	Deep venous thrombosis
	Pulmonary emboli
	Chest infection
	Induced spasticity
	Contractures
	Joint trauma, especially the hemiplegic shoulder

There have been many attempts to find medical regimes which can influence beneficially the outcome of stroke but as yet there are no specific drugs which have been shown to do this. Glycerol, naftidrofuryl, nifedipine (a calcium channel blocker) and many other drugs are currently being assessed but there is no indication for the routine use of any of these outside controlled clinical trials [1]. There is a case for specific drug treatment in strokes occurring secondary to certain other conditions (Table 10). The question of anticoagulation is still unresolved: it is traditional to anticoagulate patients in atrial fibrillation with known rheumatic heart disease but no trials have ever been done, and patients with non-rheumatic atrial fibrillation require a C.T. brain scan if anticoagulants are contemplated, because 10% of these patients will in fact have had haemorrhagic strokes, not embolic ones. Even so the evidence that anticoagulation is helpful to the other 90% is not conclusive [5].

SECONDARY PREVENTION

Management of the acute phase includes considering whether any steps can be taken to prevent a further stroke

Table 10 – Treatments for Secondary Stroke

Steroids for giant cell arteritis
Platelet reduction in thrombocythaemia
Viscosity reduction in hyperviscosity syndromes
Anticoagulants for patients with prosthetic heart valves if not already anticoagulated (Always check that patients who are supposed to be anticoagulated really are)
Neurosurgery for aneurysms and arterio-venous malformations

occuring in the immediate or longer term. In this area nothing is certain but the best evidence exists for anti-platelet therapy, and moderate evidence for blood pressure control (Table 11)[1]. Anticoagulation has not been shown to prevent recurrence in all causes of atrial fibrillation. There is an undoubted increased risk of stroke in patients with rheumatic mitral stenosis and atrial fibrillation but a controlled trial to show that anticoagulation reduces the risk has not been done and probably never will be, and the practice of anticoagulating these patient is based on "common sense". (Table 12)[5].

Table 11 – Secondary Prevention

Strong evidence:	Anti-platelet treatments, e.g. Aspirin 150 – 300 mg. daily. Ideally only after C.T. brain scan has confirmed a non-haemorrhagic stroke. Starting treatment after a one-month interval has been advocated as a compromise when no scan has been done. Reduction of polycythaemic/poly-thrombocythaemic conditions.
Moderate evidence:	Reduction of hypertension (but not rapidly at the time of the stroke). Normalisation of lipids?
Probable benefit:	Cessation or reduction of: alcohol obesity
Not yet proven:	Carotid endarterectomy

Table 12 – Anticoagulation, Atrial Fibrillation and Stroke Prevention

Atrial fibrillation with an ischaemic electrocardiogram	– benefits minimal cerebral atherosclerosis likely to co-exist
Atrial fibrillation with no ischaemic electrocardiogram or valve disease ('lone' atrial fibrillation)	– no benefit because no increased risk
Paroxysmal atrial fibrillation without valve disease	– no benefit because no increased risk
Thyrotoxic atrial fibrillation	– no benefit because no increased risk
Elective cardioversion	– anticoagulation reduces embolic stroke risk
Atrial fibrillation with rheumatic mitral stenosis	– increased stroke risk, anticoagulants 'presumed' to reduce embolic events

CONCLUSION

This protocol provides a rational framework based on the best evidence at present available within which we should treat acute stroke patients in hospital. It will undoubtedly need modifying in the light of future trial results but for the moment it gives us a standard against which we can audit the management of these patients.

REFERENCES

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FURTHER READING:

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