# Evidence-based guidelines for early stroke management

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***Stroke disease is the commonest neurological emergency encountered by the junior medical team. We have reviewed the literature to produce a series of substantiated guidelines to assist the admitting doctor in managing early stroke care optimally.***

Stroke is a common medical emergency. Almost 25% of men and 20% of women at 45 years of age can expect to have a stroke if they live to be 85 years old (Bonita, 1992). Concepts and management of patients with stroke have changed considerably in recent years and there is evidence that optimum care is inconsistently applied (Lindley et al, 1995a,b). Although many reviews of stroke care are available, their format has been largely discursive, concerned with informing rather than providing clinical guidelines aimed at reducing variability in clinical practice.

Clinical guidelines are effective in improving the quality and outcomes of patient care (Grimshaw and Russell, 1993; Effective Health Care, 1994) and reducing variations in practice. Our aim has been to develop evidence-based guidelines for early stroke care, principally to assist decision-making for the admitting junior doctor. These guidelines cover the first 24 hours of care, when inconsistent clinical decision-making is common and may not be easily corrected later.

## SCOPE OF THE GUIDELINES

The guidelines are based on the World Health Organization (WHO) definition of stroke:

> **'A syndrome of rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin.' (WHO MONICA Project Principal Investigators, 1988)**

They are not therefore comprehensive for transient ischaemic attack (TIA) management. We have also excluded 'young' patients with stroke (arbitrarily defined as <45 years), as the underlying causal mechanisms are more varied and conventional vascular risk-factors less prominent (Martin et al, 1997). Inevitably there is some blurring of the selected timeframe and some of the guidelines go beyond the first 24 hours (e.g. management of atrial fibrillation (AF) or hypertension). This is simply to reflect natural clinical processes and to provide guidelines which are clinically relevant. However, certain important but less immediate stroke issues such as nutritional support for persisting dysphagia, and the whole area of rehabilitation, have not been addressed.

Finally, we have assumed an acute medical ward or a medical admission unit as the setting in which these guidelines will be applied. Although well-coordinated stroke care in stroke rehabilitation units provides superior outcome to stroke care on general medical wards (Stroke Unit Trialists Collaboration, 1997), the benefit of specialized acute stroke units is unclear (Bath et al, 1996).

## METHOD

A series of Medline (1966–1998) searches using key words and subject headings was used to identify relevant articles. This was supplemented by searches of the Stroke Module of the Cochrane Library up to June 1998. The information obtained and the resulting guideline has been classified according to accepted levels of evidence:

- Level A: Based on randomized controlled trials (RCT) or meta-analysis of RCTs
- Level B: Based on robust experimental or observational studies
- Level C: Based on expert opinion.

Wherever possible we have sought RCT evidence and especially systematic reviews of RCTs for our guidelines, as this method provides the least biased and most precise information.

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A guidelines development group was convened to review the evidence. This comprised consultant staff from neurology, care of the elderly, general medicine, radiology and public health medicine.

Recommendations were based on the strength of the evidence and its applicability to local circumstances. Informal consensus methods were used to agree recommendations.

Review of the guidelines was undertaken by consultant medical staff in the hospital. A 3-month period for piloting the guidelines took place. They were subsequently reappraised and modified in the light of feedback from medical staff. Further review is scheduled in 1999.

## HISTORY AND EXAMINATION

A stroke clerking proforma has been developed as a basis for clinical audit (Royal College of Physicians, 1994). Although this approach can improve the completeness of patient assessment (Davenport et al, 1995), it is unclear whether improved management and outcomes are achieved.

### History

Despite advances in neuroradiology the diagnosis of stroke remains primarily a clinical one. The WHO definition of stroke emphasizes the importance of a history which uncovers a sudden onset neurological deficit. If the patient is unable to give a history (reduced consciousness, dysphasia, dementia), then a witness account should be specifically sought as this improves the precision of the diagnosis (Sandercock et al, 1985; Allen, 1993). Indeed, a history and examination has a sensitivity of 95% (only 5% false negative diagnosis) but a more variable specificity of 66–97% (3–34% false positive diagnosis), with the higher percentage representing more experienced clinicians (Ebrahim, 1990).

It is good practice while taking the history to identify the person behind the stroke by understanding their pre-stroke lifestyle, housing and family structure. Pre-stroke disability can be assessed using the Barthel Index (Mahoney and Barthel, 1965). This background is valuable in informing the appropriateness of potential treatments and promotes a process of support and interest which patients value during the crisis stage of stroke (Pound et al, 1995).

**Guideline 1 (level B):**
**A careful history is reliable in the diagnosis of stroke. Patient and/or their family should be informed about the diagnosis of stroke and given the opportunity of asking questions.**

**TABLE 1.**
**The Oxford classification of cerebral infarction**

| | | |
|---|---|---|
| TACI (Total anterior (= carotid) circulation infarct | Hemi motor and sensory deficit | |
| | Hemianopia | |
| | Cortical dysfunction | Dysphasia |
| | | Visuospatial disturbance |
| PACI (Partial anterior (= carotid) circulation infarct | Any two of the above (e.g. dysphasia and hemiplegia) or an isolated cortical dysfunction (e.g. dysphasia) | |
| LACI (Lacunar infarction) | Pure motor hemiplegia | |
| | Pure sensory loss | |
| | Motor and sensory loss | |
| POCI (Posterior circulation infarction) | Vertigo, diplopia, ataxia, isolated hemianopia | |

**TABLE 2.**
**Outcomes according to cerebral infarct clinical syndromes**

| | 30-day mortality | 3-month recurrence | 6-month dependence |
|---|---|---|---|
| TACI | 39% | Low | 39% |
| PACI | 4% | Very high | 34% |
| LACI | 2% | Low | 26% |
| POCI | 7% | High | 18% |

LACI = Lacunar infarction; PACI = Partial anterior (= carotid) circulation infarct; POCI = Posterior circulation infarction; TACI = Total anterior (= carotid) circulation infarct

### Examination

The purpose of examination at presentation is to further secure the diagnosis of stroke, specify the impairments, identify the subtype of ischaemic stroke (Bamford et al, 1991; Anderson et al, 1994) (*Table 1*) and identify co-existing conditions such as chronic respiratory disease, cardiac failure and peripheral vascular disease. Stroke subclassification is important since it gives useful indication of prognosis for early mortality, recurrence and functional outcome (*Table 2*). Examination at maximal neurological deficit also identifies associated important features in early management, e.g. impaired consciousness, impaired swallow, initial blood pressure (BP), presence of AF, fever and hypoxia. The former two issues are discussed here and the others later in the text.

**Consciousness:** Impaired consciousness can be quantified using the Glasgow Coma Score. A reduced coma score is associated with a worse outcome in acute stroke (Oxbury et al, 1975). Although sepsis and metabolic factors may contribute to drowsiness, impaired consciousness generally indicates the presence of cerebral oedema with associated raised intracranial pressure (Roper and Shafran, 1984).

**Swallowing:** Unsafe swallowing may affect up to a third of stroke patients on admission. About a

half will recover within the first week but about 10% will have persisting impairment at 1 month (Barer, 1989). An immediate assessment of swallowing in conscious patients is important to determine the risk of aspiration. Testing the gag reflex is unhelpful as it does not discriminate between safe and unsafe swallowing (Horner et al, 1988; Davies et al, 1995).

Videofluoroscopy, widely accepted as the 'gold standard' for swallowing assessment, is impractical for routine use in the acute stroke setting. A simple bedside evaluation is the preferred approach and should be routinely employed using a standardized swallowing assessment (Splaincard et al, 1988; Kidd et al, 1993; Smithard et al, 1996), one example of which is given in *Figure 1*. Using a pulse oximeter to detect desaturation during swallowing as a predictive marker for aspiration has been described but needs further study (Zaidi et al, 1995; Collins and Bakheit, 1997).

**Guideline 2 (level B):**
**Examination at presentation should describe the neurological impairments and identify the stroke sub-type. Assessment of consciousness and swallowing should always be recorded.**

## CARDIOPULMONARY RESUSCITATION STATUS

After the history and examination, the most senior doctor available should make a decision on the cardiopulmonary resuscitation (CPR) status of the patient. The pre-stroke disability and lifestyle assessment is important in informing the CPR status. Resuscitation is rarely successful (and therefore generally inappropriate) in patients who have impaired consciousness from a major stroke (Schneider et al, 1993).

**Guideline 3 (level B):**
**CPR status should be clearly recorded in the case notes with a date for review.**

## INVESTIGATIONS

The investigation of acute stroke should be directed towards delineating the type of stroke (infarct or haemorrhage), candidate causes and uncovering risk factors for future vascular events (*Table 3*). A full blood count will detect anaemia (exacerbates cerebral hypoxia) and conditions associated with vascular stasis (erythrocytosis or thrombocytosis). Measurement of plasma viscosity or erythrocyte sedimentation rate may direct the physician towards a vasculitis, particularly cranial arteritis. Patients with no vascular risk factors and a history of venous or arterial thrombosis should be investigated for a thrombophilia (clotting screen, protein C & S). Blood glucose and cholesterol are discussed later in the text.

### Chest X-ray

A clinical policy of investigating all patients with acute stroke with a chest X-ray is unsupportable. A study (Sagar et al, 1996) of 435 patients pre-

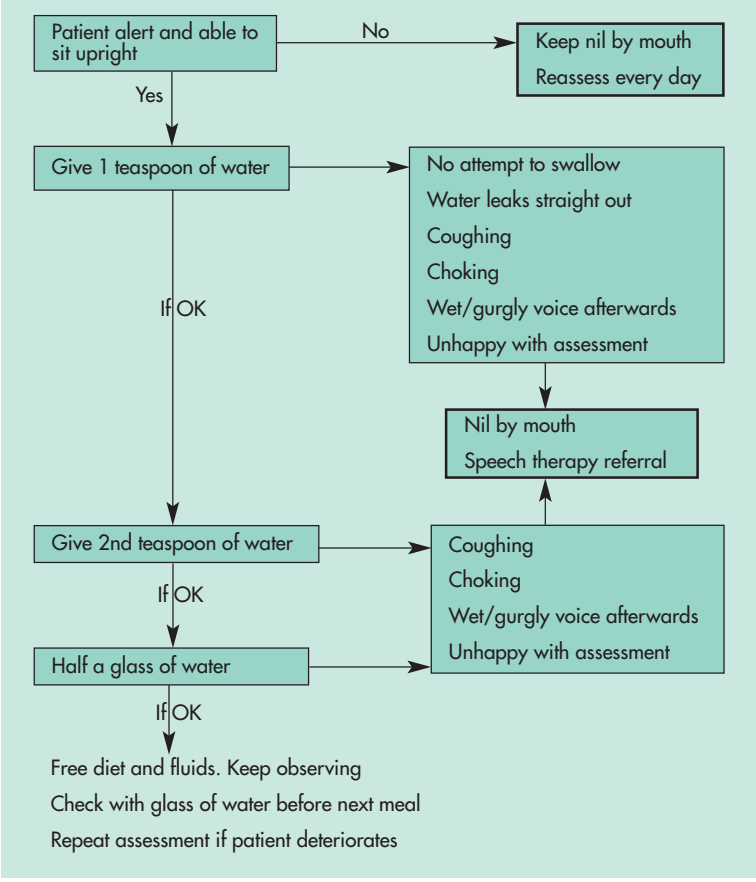


*Figure 1. Standardized swallowing assessment.*

**TABLE 3.**
**Investigations in suspected cases of acute stroke**

| | |
|---|---|
| Blood | Full blood count |
| | Urea and creatinine |
| | Erythrocyte sedimentation rate/plasma viscosity |
| | Blood gases (if hypoxia suspected) |
| | Blood sugar |
| | Cholesterol |
| | Thrombophila screen* |
| Radiological/ cardiological | Chest X-ray* |
| | Electrocardiogram |
| | Head computed tomography scan |
| | Echocardiography* |
| | Carotid duplex Doppler* |

* Selected patients only (see text)

senting with a new stroke reported a chest X-ray abnormality in only 16% and this contributed to changed clinical management in only 4%. Although the study was limited by its retrospective design, and did not assess how a normal chest X-ray appearance might influence management, it seems sensible to request the investigation only in the presence of specific indications such as weight loss or unexplained chest symptoms.

**Guideline 4 (level B):**
**A routine chest-X-ray is unnecessary.**

### Electrocardiogram

Stroke and heart disease share common epidemiological and clinicopathological features. A cardiac cause of death following stroke is not uncommon during the first month (Ebrahim, 1990). An electrocardiogram (ECG) will reliably identify AF (see later) and can indicate pre-existing ischaemic heart disease (e.g. Q waves), both of which have important implications for secondary vascular prevention or for rehabilitation (Roth, 1994).

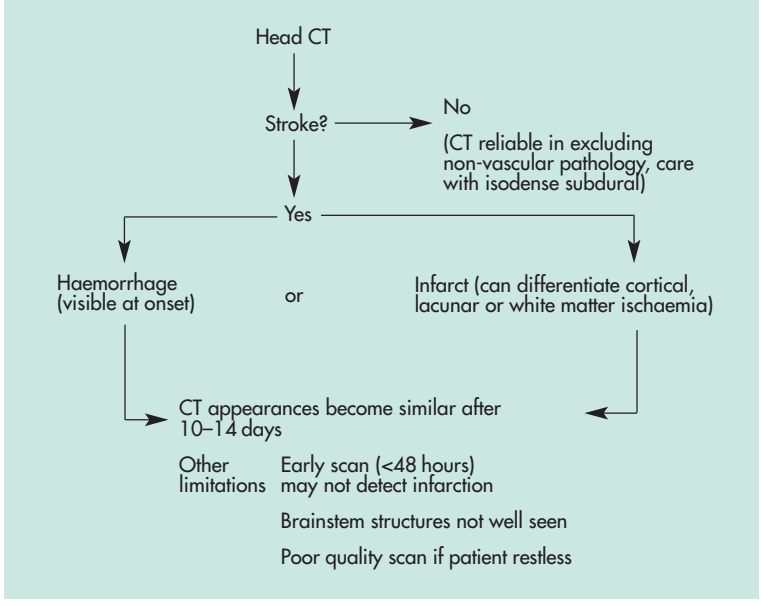


*Figure 2. Contribution and limitations of head computed tomography (CT) in acute stroke.*

**TABLE 4.**
**Framework for head computed tomography (CT) scan**

| Indication | Comment |
|---|---|
| Uncertain diagnosis of stroke | A head CT scan will reliably exclude non-vascular pathology. Care is needed to detect an isodense subdural haematoma |
| Current or contemplated antiplatelet or anticoagulation treatment | Main indication in practice |
| Suspected cerebellar haematoma | Potential neurosurgical intervention |
| Possible carotid endartectomy | To exclude haemorrhagic stroke and structural lesion mimicking mini-stroke |
| Suspected subarachnoid haemorrhage | Potential neurosurgical intervention |

**Guideline 5 (level B):**
**All conscious stroke patients should have an ECG.**

### Head computed tomography

Head computed tomography (CT) as applied to stroke has evolved and become absorbed into clinical practice without systematic evaluation. There is, however, no shortage of 'expert opinion' which generally supports a low threshold clinical policy to scan (Royal College of Physicians, 1990; US National Stroke Association Consensus Statement, 1993; Lindley et al, 1995a), but controversy exists (Wardlaw and Allison, 1994). A policy of 'routine' head CT scanning is a rather intellectually lazy approach. It is better to understand the advantages and limitations (*Figure 2*) of the investigation and to adopt a system of intelligent clinical questioning pertinent to individual patients with suspected stroke. A useful framework provided by the King's Fund Forum (1988) in their consensus statement is given in *Table 4*.

In practice the main clinical issue dealt with by a head CT scan is in the important distinction between cerebral infarction and haemorrhage. This allows rational and safer use of anticoagulation or antithrombotic treatments. Clinical scoring systems cannot reliably distinguish between the two pathologies (Weir et al, 1994).

**Guideline 6 (level C):**
**Patients should have a head CT scan if there is uncertainty about the diagnosis of stroke or if anticoagulation or antiplatelet treatment is contemplated.**

### Head magnetic resonance imaging

To develop acute stroke treatments with the potential to limit cerebral damage, the detection of early ischaemic changes seen on magnetic resonance imaging (MRI), but not by head CT has been considered an advantage. MRI is also able to demonstrate smaller lesions and image the posterior fossa, brainstem and lacunar lesions more clearly than CT. However, studies comparing CT and MRI provide unclear implications for clinical practice (Imakita et al, 1988; Mohr et al, 1995). Newer techniques such as diffusion-weighted MRI have promise (Lutsep et al, 1997) and may be incorporated into routine practice in the future.

**Guideline 7 (level B):**
**There is no indication for routine MRI in acute stroke.**

### Echocardiography

A cardio-embolic source for cerebral infarction is increasingly sought, particularly in partial anterior and posterior circulation infarcts (PACI

and POCI) where the risk of recurrent stroke is high. An echocardiogram can detect sources of emboli (e.g. thrombus, myxoma, vegetation) or conditions associated with higher risk of stroke (e.g. dilated left ventricle, AF and dilated left atrium, mitral stenosis).

The benefit of transoesophageal echocardiography (TOE) over transthoracic echocardiography (TTE) in acute stroke has been widely debated. TOE provides better anatomical information (e.g. left atrial thrombus, patent foramen ovale, vegetations) but is more invasive (and therefore more hazardous). It can also be a difficult procedure in the context of a disabling stroke. The largest study comparing the two methods favours TTE initially (Leung et al, 1995) and a recent review reached the same conclusion (American College of Cardiology/American Heart Association, 1997). Unfortunately the clinical yield of echocardiography in stroke is very low and routine echocardiography cannot be recommended (Leung et al, 1995; Chambers et al, 1997). It is more sensible to select patients for this investigation first, by whether a positive scan would change clinical management (usually initiation of anticoagulation), and second where clinical features or an abnormal ECG suggests cardiac disease.

**Guideline 8 (level B):**
**There is no indication for routine echocardiogram in acute stroke.**

### Carotid duplex Doppler scan

A carotid duplex Doppler scan is a reliable non-invasive investigation for carotid artery stenosis provided it is performed by an experienced operator (Humphrey, 1995). There is good evidence that symptomatic patients with severe stenosis (about 80% or greater stenosis) benefit from endarterectomy by an experienced surgical team whereas patients with moderate stenosis do not (North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991; European Carotid Surgery Trialists' Collaborative Group, 1998). Only patients with similar characteristics to those recruited to the RCTs and who are therefore suitable candidates for carotid endarterectomy should be investigated (Brown and Humphrey, 1992):

1. Patients with a minor ischaemic stroke in the carotid artery territory and little persisting disability
2. Patients with a more substantial stroke in the carotid territory who have made a good recovery
3. Patients who are otherwise medically fit.

**Guideline 9 (level A):**
**Carotid duplex Doppler scan is indicated for selected patients only.**

## MANAGEMENT

### General care and support

**Fever:** The presence of fever in acute stroke is accompanied by a worse prognosis (Azzimondi et al, 1995; Reith et al, 1996). At present it is not known whether the relationship is causal or a reflection of stroke severity. Obviously a fever should prompt a search for an infection source. Antipyretics, although often used as a symptomatic treatment, are not known to influence outcome.

**Hypoxia:** The level of oxygen saturation may be critical in the potentially salvageable ischaemic penumbra zone surrounding the area of neuronal death. Hypoxia is exacerbated by seizures, cardiac arrhythmia and pneumonia, all of which are associated with a worse cognitive outcome (Moroney et al, 1996). Correction of hypoxia seems sensible, although there is no evidence to suggest that outcome is improved.

Cerebral oxygen delivery might also be improved by haemodilution or by hyperbaric oxygen treatment. Haemodilution using venesection and/or plasma expanders aims to reduce haematocrit levels, thereby increasing cerebral blood flow but a systematic review shows no evidence for efficacy (Asplund et al, 1997). Hyperbaric oxygen is similarly ineffective (Anderson et al, 1991; Nighoghossian et al, 1995).

**Guideline 10 (level B):**
**Arterial blood gases should be checked if hypoxia if suspected.**

**Cerebral oedema:** Therapeutic strategies to reduce cerebral oedema complicating acute stroke have been investigated but none have proved successful. Cerebral oedema related to tumours (vasogenic oedema) responds well to corticosteroids but cerebral oedema complicating acute stroke (cytotoxic oedema) does not. There is no effect on survival or improvement in functional outcome (Tellez and Bauer, 1973; Poungvarin et al, 1987; Qizilbash et al, 1997).

The two hyperosmolar agents, mannitol and glycerol, have also been evaluated but studies involving mannitol have largely been confined to the context of neurosurgery and animal models (Paczynski et al, 1997). A meta-analysis of RCTs investigating glycerol provides insufficient evidence to support its use, as, although there is a trend towards improved short-term outcome, the estimate of effect is statistically inconclusive (Rogvi-Hansen and Boysen, 1997).

**Guideline 11 (level A):**
**Corticosteroids or hyperosmolar agents to reduce cerebral oedema in acute stroke are unproven treatments and should not be used.**

**Blood pressure:** Hypertensive encephalopathy and aortic dissection associated with hypertension may present with an acute stroke syndrome and both require urgent attention to BP control. However, for most patients with raised BP detected immediately after stroke onset, BP will fall spontaneously during the first 10 days (Wallace and Levy, 1981; Morfis et al, 1997). This is a particularly vulnerable period as cerebrovascular autoregulation is impaired and injudicious reduction in BP might cause further damaging hypoperfusion in the ischaemic penumbra tissue. The converse, important clinical consideration is that low BP needs prompt correction of hypovolaemia and withdrawal of hypotensive medication.

There is unequivocal evidence for hypertension treatment in primary stroke prevention (Collins and MacMahon, 1994), but hypertension treatment in stroke survivors is much less clear and will remain so until the results of current RCTs in this area become available (Neal et al, 1996). Simple extrapolation from the primary prevention trials to stroke survivors is unwise as although patients have higher absolute risk of further vascular events and therefore greater potential benefit, BP reduction in patients with possible stenotic vascular segments may be harmful by inducing hypoperfusion events. Reassuring observational data were obtained from the UK TIA aspirin trial (1991) where a direct and continuous relationship was found between systolic and diastolic BP and further stroke such that a 5 mmHg reduction of diastolic BP was associated with about a third fewer recurrent strokes.

**Guideline 12 (level B):**
**Record BP at least twice a day. Correct low BP where possible. Defer any reduction in acute BP for 10 days unless hypertensive encephalopathy or aortic dissection is present.**

**Atrial fibrillation:** Patients presenting with an ischaemic stroke and non-rheumatic AF have a higher mortality (early and late) (Framingham Study, 1996), and are at high relative risk (12%) of a further stroke in the year after the event. The European Atrial Fibrillation Trial (EAFT, 1993) recruited patients presenting with TIA or minor ischaemic stroke within the previous 3 months and demonstrated that anticoagulation with warfarin reduced the risk of a further stroke by 67% and aspirin by 14%. Patients with major strokes (needing some assistance to walk and for personal care) were excluded. Major complicating bleeding events were low in the trialists hands (warfarin 2.8%/year; aspirin 0.9%/year) probably because of careful patient selection and fastidious international normalized ratio (INR) control.

Thus, where there is no contraindication, and the patient consents, warfarin should be used for secondary prevention and, in other circumstances, aspirin. Subsequent work has indicated that the target INR should be 2–4: an INR of less than 2 has little treatment effect and greater than 5 carries a high risk of a bleeding complication (EAFT, 1995; Stroke Prevention in Atrial Fibrillation Investigators (SPAF) III, 1996). When to commence warfarin is not known but waiting at least 48 hours, and possibly longer for larger infarcts, has been advised (Sherman et al, 1995).

**Guideline 13 (level A/B):**
**Patients with AF and ischaemic stroke resulting in mild disability who have no anticoagulation contraindication should be treated with warfarin (INR 2–4) after waiting at least 48 hours. Aspirin should be used in other circumstances.**

**Blood glucose:** A glucose level greater than 8 mmol/litre is an independent predictor of poor outcome after acute stroke (Weir et al, 1997). The outcome of patients presenting with acute myocardial infarction and random blood sugars greater than 11 is improved by intensive insulin therapy (Malmberg et al, 1995, 1997), but it is not yet known whether a similar intensive approach to hyperglycaemia management in acute stroke is beneficial.

**Guideline 14 (level B/C):**
**Measure blood sugar on admission for all patients. If high, or patient known to have diabetes, monitor blood sugars at 6-hourly intervals.**

**Cholesterol:** Epidemiological studies do not support the notion of cholesterol as an independent risk factor for cerebrovascular disease (Prospective Studies Collaboration, 1995) and there are no intervention studies examining cholesterol lowering in patients presenting with stroke. However, a meta-analysis of trials investigating cholesterol reduction by a statin for patients with ischaemic heart disease has demonstrated an overall stroke risk reduction of 30% (Blaun et al, 1997; Herbert et al, 1997). Many patients presenting with stroke will have co-existing ischaemic heart disease and will therefore fulfil the entry criteria of these coronary secondary prevention trials. A reduction of baseline total cholesterol of 4.8 mmol/litre has been associated with benefit (Sacks et al, 1996).

**Guideline 15 (level A):**
**Stroke survivors with a background of ischaemic heart disease and reasonable medium term prognosis (2–3 years) with a cholesterol greater than 4.8 mmol/litre should be treated with a statin.**

**Prevention of early complications:**

*Deep vein thrombosis:* Detailed studies have shown that deep vein thrombosis (DVT) complicates stroke in about 50% of patients during the first 10 days (Kalra et al, 1995), but pulmonary embolus is much less common (6–16% of patients; Sandercock et al, 1993). Although heparin, particularly low molecular weight heparin, is effective in preventing DVT complicating acute stroke (Sandercock et al, 1993; Counsell and Sandercock, 1997), the risk of haemorrhagic complications, particularly intracerebral haemorrhage, has been less well understood.

The International Stroke Trial (IST, 1997) showed that subcutaneous heparin (12500 IU or 5000 IU twice daily) is associated with a significant excess of extracranial and intracranial haemorrhages and only a small and inconclusive reduction in pulmonary embolism confined to the higher dose heparin regimen, for which haemorrhagic complications were greater.

Graduated compression stockings have proven benefit in the prevention of DVT in surgical patients but have not yet been evaluated in stroke (Clagett et al, 1992; Wells et al, 1994). They should not be used if peripheral arterial insufficiency exists as ischaemic skin damage may result.

**Guideline 16 (level A/C):**
**Subcutaneous heparin to prevent DVT should be avoided. Graduated stockings are of uncertain effectiveness but should not be used in patients with concomitant peripheral vascular disease.**

*Pressure sores:* Pressure sores are a preventable early complication of an acute stroke which has resulted in immobility. Prevention involves assessment of risk and rapid deployment of special low pressure mattress systems which are more effective than standard hospital mattresses (Effective Health Care, 1995).

**Guideline 17 (level A/B):**
**Patients should be routinely assessed for pressure sore risk and those with high risk provided with a specialized pressure lowering system.**

**Management of cerebral infarction**

Occlusion of a cerebral artery results in a central area of severe focal ischaemia surrounded by an area of moderate ischaemia where the neuronal cell membrane is intact but the cell becomes electrically silent and synaptic transmission fails (ischaemic penumbra). A complex and incompletely understood process (Pusinelli, 1992) may further damage this area of relative ischaemia, extending the final infarct size. Pharmacological modification of the ischaemic penumbra process is an attractive goal but no drug has yet proved clinically successful (Langhorne and Stott, 1995). Modification of associated cerebral oedema has also been unsuccessful (see above). Only antithrombotics and anticoagulants are currently of proven benefit, in relation to prevention of further vascular events (secondary prevention) rather than direct acute infarct-modifying effects.

**Antiplatelet therapy:** Aspirin is the most studied agent. A dose of 75–150 mg/day inhibits platelet cyclo-oxygenase and reduces the risk of further stroke and other vascular events by 22%, about 40 vascular events avoided per 1000 patients treated over 3 years (Antiplatelet Trialist Collaboration, 1994). Larger doses of aspirin are no more effective but are associated with more side-effects, especially gastropathy (Weil et al, 1995).

Two large studies (CAST, 1997; IST, 1997) have shown additional benefit when aspirin is started within 48 hours of stroke onset (10 deaths or recurrent strokes per 1000 patients treated). Both studies committed patients to an early head CT scan to exclude intracerebral haemorrhage, when antiplatelet agents are contraindicated. Rapid onset of action requires a loading dose of at least 150 mg which can be given orally or rectally. Aspirin should be avoided if there is a history of aspirin hypersensitivity, active peptic ulcer or recent gastrointestinal bleeding, or if the patient is already taking an anticoagulant. The relative benefit of aspirin in each stroke sub-type (TACI, PACI, LACI or POCI) is inconclusive (CAST, 1997; IST, 1997). This is not surprising as there is variation in the natural history of cerebrovascular disease: even when the first event is lacunar, further events may be embolic (Kappelle et al, 1995; Yamamoto and Bogousslavsky, 1998).

There is less information available for other antiplatelet drugs such as dypiridamole (Antiplatelets Trialist Collaboration, 1994), ticlopidine (Hass et al, 1989; Antiplatelets Trialist Collaboration, 1994) and clopidogrel (CAPRIE, 1996). They have similar efficacy to aspirin and can be used as (more expensive) alternatives. These agents have different modes of action to aspirin and therefore synergy is possible (Diener et al, 1996), but not yet well established.

**Guideline 18 (level A):**
**Aspirin should be given immediately in conscious patients with no contraindication provided a head CT scan result will be quickly available to exclude intracerebral haemorrhage.**

**Anticoagulant therapy:** *Warfarin:* The role of warfarin in stroke in the presence of AF is now well defined (see above) but its use in the presence of sinus rhythm has been more uncertain. The

Stroke Prevention in Reversible Ischaemia Trial (SPIRIT, 1997) compared low dose aspirin (30 mg) with warfarin (INR 3–4.5) in patients who had had TIA or minor stroke within the last 6 months and were in sinus rhythm. The trial was stopped early because of a significant excess of haemorrhagic complications in the warfarin group. *Heparin:* The efficacy and safety of early anticoagulation was unclear until IST (1997) addressed this issue. Low dose heparin (5000 IU twice daily) was associated with a small but significant reduction in early death or stroke (12 fewer events per 1000 patient treated) and a slight but non-significant excess of haemorrhagic complications. However, the observed treatment effect was no greater than that seen with aspirin. A medium-dose heparin regimen (12500 IU twice daily) had the same treatment benefit but with significantly greater hazard. In the sub-group of patients with AF, the balance of clinical benefit to hazard did not support the use of subcutaneous heparin.

**Guideline 19 (level A):**

**Avoid subcutaneous heparin.**

**Thrombolysis:** One trial using tissue plasminogen activator in highly selected patients, in circumstances which would be difficult to replicate in routine practice, has reported a significant reduction in poor functional outcome (NINDS rt-PA Stroke Study, 1995). However, a systematic review and statistical summary of all available evidence indicates considerable caution is needed. The meta-analysis findings (Wardlaw et al, 1997) show uncertain clinical benefit at the expense of greater hazard (increased symptomatic intracranial haemorrhage).

**Guideline 19 (level A):**

**Avoid thrombolysis.**

**Treatment of cerebral haemorrhage:** The main initial management of cerebral haemorrhage is supportive care as outlined above. Surgical evacuation of a supratentorial intracerebral haemorrhage is controversial and probably best avoided (Hankey and Hon, 1997; Prasad and Shrivastava, 1997). Surgery for a infratentorial (cerebellar) haemorrhage is more widely accepted (Shenkin and Zavala, 1982; Hankey and Hon, 1997). The main indication is deteriorating or depressed conscious state.

**Guideline 20 (level A):**

**Patients with a cerebellar haematoma and depressed conscious level should be referred urgently to a neurosurgeon.**

## KEY POINTS

- A carefully-taken history is reliable in accurately diagnosing stroke.
- The stroke subtype, conscious level and an assessment of swallow should be recorded in all patients.
- Investigations should be critically selected to assist individual patient management rather that a policy of 'blanket tests.
- Immediate post-onset hypertension settles spontaneously in most patients and does not need treatment.
- Early aspirin treatment in ischaemic stroke is beneficial but subcutaneous heparin should be avoided.
- Warfarin is superior to aspirin for secondary prevention when atrial fibrillation is present, but patient need careful selection.

## CONCLUSIONS

Clinical guidelines are 'systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances' (Field and Lohr, 1992). Guidelines have the capability to change clinical practice and affect patient outcomes (Effective Health Care, 1994). Stroke is a condition eminently suited to guideline-based management because it is common, has a widely dispersed evidence base, and injudicious interventions readily contribute to a detrimental outcome.

These guidelines are based on desirable attributes (Effective Health Care, 1994), particularly a strong and explicitly linked relationship to the supportive evidence. The simplicity and clarity should maximize their implementation by the target audience: the admitting junior doctor. To further facilitate implementation, the guidelines are summarized in a simple flow diagram (*Figure 3*) which draws together the principle of structured assessment, careful individual patient focused investigation and considered treatment. In this way an individualized care pathway can be constituted to optimum clinical outcome and promote efficient use of resources. HM


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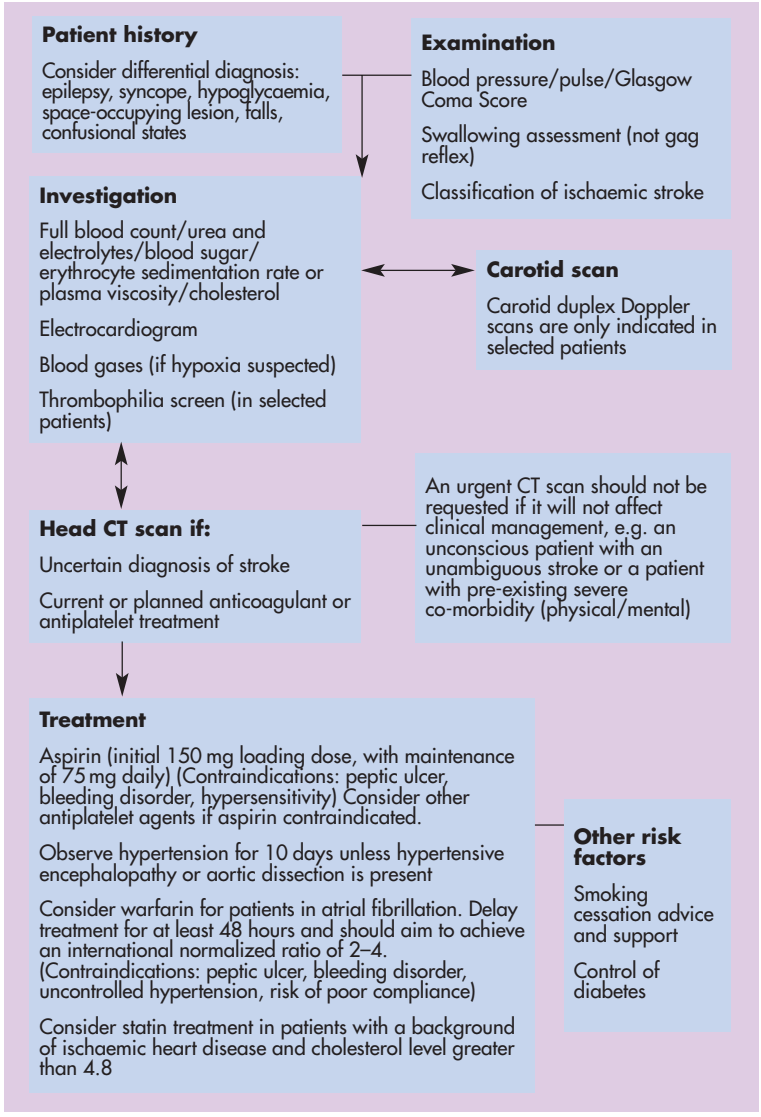


***Figure 3. Summary of clinical guidelines for early management of stroke. CT = computed tomography.***

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