Management of acute ischaemic stroke

Ischaemic strokes comprise the majority of strokes in clinical medicine. With the widespread use of thrombolytic therapy within 3 hours of presentation the long-term disability as a result of ischaemic stroke has improved markedly. This article summarizes the clinical management of ischaemic strokes including recent advances.

Stroke is a common cause of disability and death in developed countries and up to two decades ago it was seen as a difficult management issue for physicians, leading to a sense of hopelessness with consequent unnecessary increase in disability and mortality. With increased acceptance of thrombolysis since the late 1990s there has been a dramatic increase in research in the overall management of strokes, particularly ischaemic strokes. This article reviews the management of ischaemic stroke, highlighting some of the important developments of the last decade.

Initial assessment

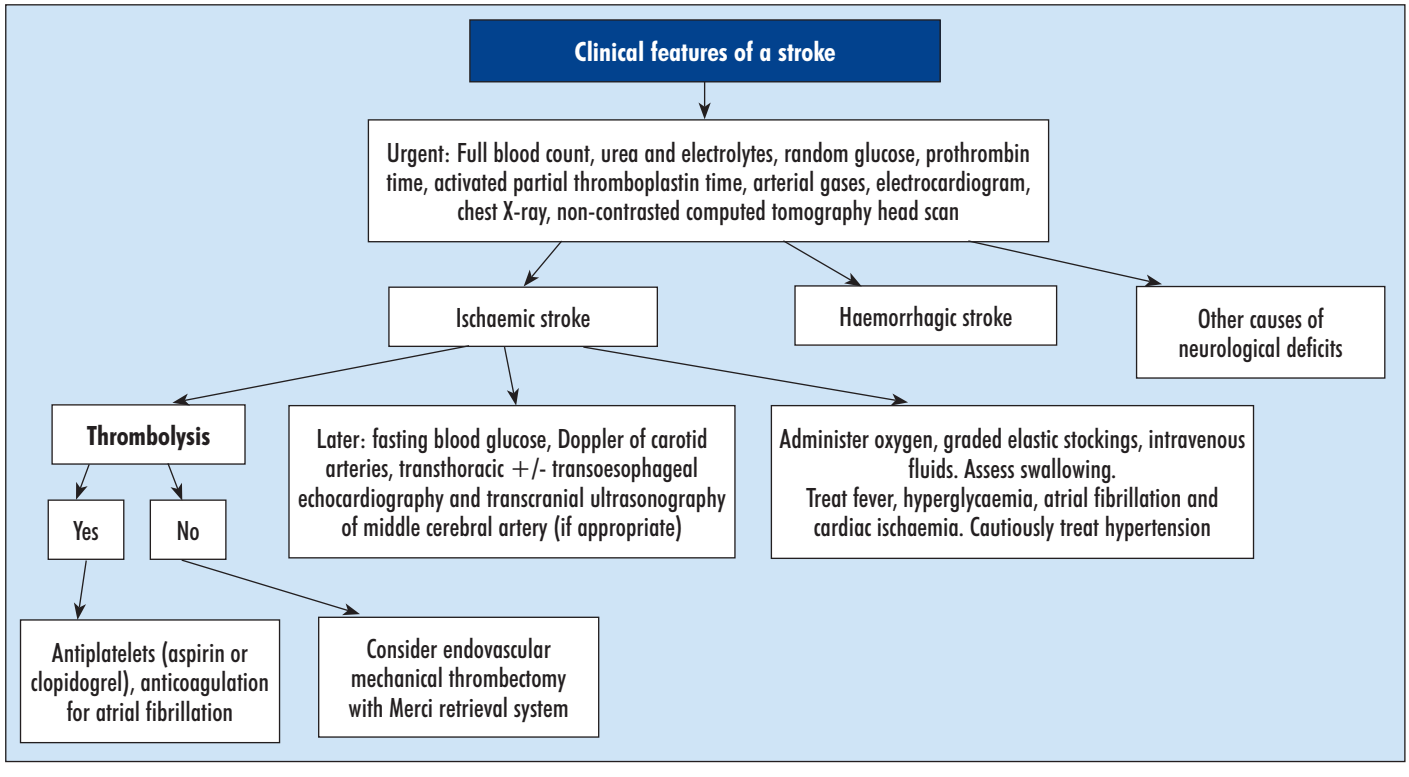
Ischaemic strokes comprise the majority of presenting strokes. Errors in the diagnosis of strokes can occur when the patient presents with unrecognized seizure, or where there are metabolic or toxic causes. At presentation it is critical to assess the patient comprehensively, taking a detailed history from the patient and/or other significant person who was present at the time of the event, noting the neurological deficit and ultimately deciding the vascular territory involved in the clinical event.

Not only should the assessment with history and clinical examination be comprehensive, but some pertinent investigations need to be promptly requested. Blood investigations needed immediately after clinical assessment include full blood count, urea and electrolytes, prothrombin time, activated partial thromboplastin time, blood glucose and arterial gases if hypoxia is suspected (*Figure 1*). Electrocardiogram, chest X-ray and computed tomography (CT) scan of the head (non-contrast) will also be required when the blood tests are ordered (*Figure 2*). Once the diagnosis has been established, Doppler ultrasonography of the common carotid and internal carotid arteries will need to be performed as well as transthoracic echocardiography and, if required, transoesophageal echocardiography.

The initial assessment must also document the severity of the neurological deficit as set out in the National Institute of Health Stroke Scale (NIHSS), which is now widely used in established stroke units (Brott et al, 1989) (*Table 1*). This scale is based on neurological deficits

Dr Husayn Al Mahdy *is Consultant Physician, Queen Elizabeth Hospital, London SE18 4QX*

Figure 1. Management of acute ischaemic stroke.



found on clinical examination and should take no more than 15 minutes to document. This scale predicts the functional outcome after a period of rehabilitation. A score of less than 10 predicts a favourable outcome whereas a score greater than 20 predicts a poor outcome (Adams et al, 1999).

Urgency of CT scan

A CT scan of the head needs to be undertaken immediately to establish the aetiology of the stroke and determine suitability for thrombolysis in cases that present within 3 hours of onset of symptoms without contraindications (*Table 2*). A full blood count reveals co-existing infection or other haematological problems that would influence initial management of the patient. Urea and electrolytes are important for assessing hydration and electrolyte abnormalities in those taking diuretics, which are commonly prescribed for hypertension. In addition, many patients presenting with strokes are taking anticoagulants and assessment of prothrombin time might identify early those with probable haemorrhagic infarcts or with primary intracerebral haemorrhage and provide a guide for the reversal of anticoagulation. In those eligible for thrombolysis this can provide a safer mechanism of administration of anticoagulants.

Other coexisting clinical issues

Those who present with hypoglycaemia will easily be identified by blood glucose measurement, and this may influence the ultimate diagnosis of the presentation. In those with hyperglycaemia appropriate management can be undertaken in addition to stroke management. On the other hand, an electrocardiogram is essential in the initial evaluation to determine whether cardiac ischaemia

Figure 2. Non-contrast computed tomography scan of head showing a hypodense area in the left parietal lobe depicting cerebral infarction of the left middle cerebral artery distribution. Note the associated effacement of sulci.

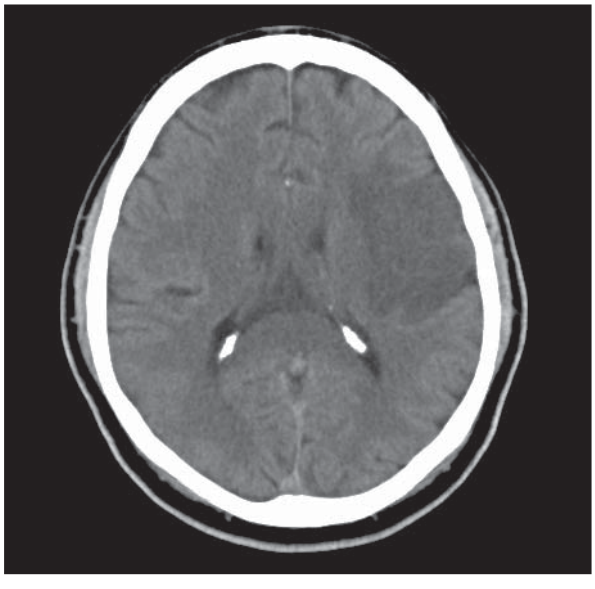


Table 1. National Institutes of Health Stroke Scale (NIHSS)

Item	Name	Response	Patient score
1a	Level of consciousness	0 = Not alert, arousable 1 = Not alert, obtunded 2 = Unresponsive	
1b	Questions	0 = Answers both correctly 1 = Answers one correctly 2 = Answers neither correctly	
1c	Commands	0 = Performs both tasks correctly 1 = Performs one task correctly 2 = Performs neither task	
2	Gaze	0 = Normal 1 = Partial gaze palsy 2 = Total gaze palsy	
3	Visual fields	0 = No visual loss 1 = Partial hemianopia 2 = Complete hemianopia 3 = Bilateral hemianopia	
4	Facial palsy	0 = Minor paralysis 1 = Minor paralysis 2 = Partial paralysis 3 = Complete paralysis	
5a	Left motor arm	0 = No drift 1 = Drift before 10 seconds 2 = Falls before 10 seconds 3 = No effort against gravity 4 = No movement	
5b	Right motor arm	0 = No drift 1 = Drift before 10 seconds 2 = Falls before 10 seconds 3 = No effort against gravity 4 = No movement	
6a	Left motor leg	0 = No drift 1 = Drift before 5 seconds 2 = Falls before 5 seconds 3 = No effort against gravity 4 = No movement	
6b	Right motor leg	0 = No drift 1 = Drift before 5 seconds 2 = Falls before 5 seconds 3 = No effort against gravity 4 = No movement	
7	Ataxia	0 = Absent 1 = One limb 2 = Two limbs	
8	Sensory	0 = Normal 1 = Mild loss 2 = Severe loss	
9	Language	0 = Normal 1 = Mild aphasia 2 = Severe aphasia 3 = Mute or global aphasia	
10	Dysarthria	0 = Normal 1 = Mild 2 = Severe	
11	Extinction and inattention	0 = Normal 1 = Mild 2 = Severe	

Adapted from Brott et al (1989)

is associated with cerebral infarction, in which case these will be managed simultaneously. In patients with atrial fibrillation there will be great suspicion that the ischaemic stroke was the result of an embolic phenomenon and anticoagulation at a later date could be considered. The cardiac events, as demonstrated by electrocardiogram, can be explored further by an echocardiogram that provides detailed information about the structural integrity of the myocardium and its valves. In cases of significant myocardial damage demonstrated by transthoracic echocardiogram associated with low ejection fraction of the left ventricle, the overall prognosis tends to be poor.

Clinical management

Thrombolysis

Thrombolysis is the most important aspect of the initial management of acute ischaemic stroke (National Institute of Neurological Disorders and Stroke, 1995). It has received widespread acceptance in clinical practice and there is now optimism that such an active approach will result in good long-term outcomes of decreased morbidity and mortality. Up to 20 years ago ischaemic strokes were seen by physicians as a disease entity from which good recovery was not possible, and inevitably leading to premature death. With the trials on thrombolysis in the 1990s and successful use of tissue plasminogen activator in acute stroke as described in the National Institute of Neurological Disorders and Stroke (1995) trial the whole focus of management of ischaemic strokes changed rapidly. The use of recombinant tissue-type plasminogen activator (rt-PA) given within 3 hours of onset of neurological deficit as an intravenous infusion of 0.9 mg/kg body weight (maximum of 90 mg), with the initial 10% of the dose being given as a bolus and the rest in the following 60 minutes, is now the standard treatment, provided the entry criteria in *Table 2* are met (Adams et al, 2005). The aim is to restore blood supply to the affected area in the cerebrum to salvage the ischaemic penumbra (Adams et al, 2003). Those who have thrombolysis within 90 minutes of the clinical event tend to do better. However, symptomatic intracranial haemorrhage is a major complication of this treatment and its prevention is the chief aim of the entry requirements.

Table 2. Entry criteria of patients for thrombolysis

1.	A diagnosis of ischaemic stroke
2.	Computed tomography scan of head shows hypodensity of no more than one third of the cerebral hemisphere
3.	Patient and/or relatives informed of potential risks and benefits of thrombolysis. Consent granted
4.	Onset of symptoms less than 3 hours previously
5.	No history of trauma or previous stroke in the previous 3 months
6.	No myocardial infarction in the previous 3 months
7.	No gastrointestinal or urinary tract haemorrhage in the previous 3 weeks
8.	No major surgery in the previous 2 weeks
9.	No arterial puncture at a non-compressible site in the previous 7 days
10.	No history of previous intracranial haemorrhage
11.	Blood pressure less than 185 mmHg systolic and less than 110 mmHg diastolic
12.	No evidence of active bleeding or fracture on examination
13.	If taking warfarin then international normalized ratio is ≤1.7
14.	If receiving heparin in the previous 24 hours activated partial thromboplastin time must be in the normal range
15.	Platelet count ≥100 x 10^6/litre
16.	Blood glucose > 2.7 mM
17.	No seizure activity

From Adams et al (2005)

However, thrombolytic therapy has been shown to provide benefit in cases of acute ischaemic stroke up to 4.5 hours from the onset of symptoms in a trial organized by the European Cooperative Acute Stroke Study (Hacke et al, 2008). Those with severe stroke with NIHSS score of over 25, on oral anticoagulants and previous stroke with diabetes mellitus were excluded from the study. The proportion of patients with symptomatic intracranial haemorrhage was no greater than expected, but as the patients in this trial were highly selected and thus not representative of patients routinely given thrombolysis, care needs to be exercised in interpreting these data.

Patients receiving thrombolysis should have their vital signs monitored every 15 minutes for the first 2 hours, every 30 minutes for the next 6 hours and then hourly for the following 16 hours. Anticoagulants and antiplatelet drugs should be avoided for the first 24 hours. Individuals not eligible for thrombolysis, either as a result of abnormal coagulation (international normalized ratio> 1.7, activated partial thromboplastin time >45 seconds or platelets <100x10^6/litre) or beyond the therapeutic window of 3 hours, can have the occluded intracranial artery re-opened by a Merci retriever catheter for thrombectomy (Smith et al, 2005; Nogueira et al, 2009) as a primary procedure akin to primary angioplasty in acute myocardial infarction. Such patients are suitable for mechanical thrombectomy for up to 8 hours after the onset of symptoms. The process involves cerebral angiography following CT scan of the head to locate the occluded artery and guide the thrombectomy catheter. Good neurological outcome and improved survival have been observed in these studies. However, intracranial haemorrhage is the main complication of this procedure, occuring in about 5% of cases. At present this procedure is not routine practice in stroke units. Interestingly trials have also shown positive results in the use of this technique as a rescue procedure in non-canalized intracranial arteries after thrombolysis.

Other investigations

Doppler ultrasonography of the common carotid and internal carotid arteries are important in evaluating patients who would benefit from endarterectomy of the

relevant significantly stenosed artery, as demonstrated in the European Carotid Surgery Trialist Group (1998) and North American Symptomatic Carotid Endarterectomy Trial Collaborators (1991). Both trials came to similar conclusions; those with greater or equal to 70% stenosis of the relevant internal carotid artery benefit from carotid endarterectomy rather than medical treatment with aspirin. However, the measurement of degree of stenosis of the internal carotid artery was different in these two trials and awareness of the degree of stenosis of the case involved is required, depending on which criteria were used in the assessment of that stenosis. The timing of surgery has been debated extensively, but it is now realized that early intervention is desirable as the longer the wait, the lower the likelihood of preventing further strokes. The National Institute for Health and Clinical Excellence (2008) advocates that surgery be undertaken within a week of the acute stroke. Transcranial ultrasonography (*Figure 1*) has been successfully used as a non-invasive method of determining the patency of thrombolytic therapy over time (Demchuk et al, 2001). This is useful in predicting outcome and may prove valuable in guiding other mechanical thrombectomy or embolectomy devices, such as the Merci retriever catheter (Smith et al, 2005), in suitable cases when intravenous thrombolysis has been unsuccessful.

A follow-up non-contrast CT scan of the head is essential in those who deteriorate. Neurologically, such deterioration suggests extension of the infarction or its haemorrhagic transformation (particularly those who have had thrombolysis). In these cases antiplatelet drugs may need to be stopped temporarily.

For those whose initial CT scan of the head does not convincingly show any discernible changes, a follow-up CT scan a few days later would be helpful in confirming the diagnosis and the territory involved, or alternatively a magnetic resonance image of the head with the relevant angiogram would help elucidate the nature of the neurological deficit.

Other clinical interventions

Studies in other clinical aspects of care have also advanced understanding over the past decade. For example, administration of regular subcutaneous low molecular weight heparin or unfractionated heparin as prophylaxis for deep vein thrombosis during the immobility phase of stroke has been shown to transform an ischaemic stroke to a haemorrhagic one thereby complicating the clinical picture (Sandercock et al, 2004). Therefore, early mobilization and use of physical methods in the form of graded elastic stockings (Grandi et al, 2005) are preferred in stroke units for the prevention of deep vein thrombosis. However, the evidence for such physical modalities in preventing thromboembolism is inconclusive. Deep vein thrombosis or pulmonary embolism still occur despite the use of such physical therapies, and hence the need for anticoagulation arises. This has to be balanced with the risk of haemorrhagic transformation of the infarction.

Further studies in the safe use of anticoagulants are urgently needed, taking into account the extent of ischaemic brain, to safely treat those who sustain thromboembolism within a few days of a stroke. In those presenting with atrial fibrillation with strong suspicion of an embolic cause of acute infarction, anticoagulation must be withheld in the acute phase and can be introduced after 14 days (International Stroke Trial Collaborative Group, 1997).

Aspirin should be administered at a dose of 300 mg daily orally (Chinese Acute Stroke Trial Collaborative Group, 1997), or via a nasogastric tube in the acute phase and continued for 14 days when the dose can be reduced to a maintenance dose of 75 mg or 150 mg depending on the country's pharmaceutical formulary. It is tempting to combine aspirin with clopidogrel to provide more effective antiplatelet activity, but such a combination leads to haemorrhagic transformation of the cerebral infarct (Diener et al, 2004). Those who are intolerant to aspirin can be given clopidogrel, although there are no large studies comparing the use of clopidogrel with aspirin in the acute phase.

It is not uncommon for patients to present with swallowing difficulties after an acute ischaemic stroke. Swallowing should be assessed in every individual with acute stroke and this can easily be done at the bedside and in those judged to have doubtful or defective swallowing reflex. Nutrition via a nasogastric tube is essential to promote effective rehabilitation, and also facilitates administration of oral medications. Usually swallowing problems can be treated by changing the consistency of the diet, and with time these generally improve, allowing a return to a normal diet. Swallowing assessment can be undertaken by a speech therapist and in difficult cases, especially when silent aspiration is suspected, video fluoroscopy can be arranged with the hospital radiology department. In the event of an extended period of defective swallowing, despite modification of the diet, a percutaneous endoscopic gastrostomy tube is better than a nasogastric tube for providing nutrition and medications directly into the stomach (James et al, 1998).

Raised intracranial pressure from a large middle cerebral artery infarction and the resulting cerebral oedema may require surgical decompression by hemicraniectomy (*Figure 3*) in order to salvage the surrounding viable brain tissue (Juttler et al, 2007). This improves clinical outcome with reduced mortality, but patients tend to be left with significant long-term neurological deficits requiring long-term personal care. On the other hand, cerebellar infarctions that lead to acute secondary hydrocephalus warrant surgical decompression with an intraventricular catheter and prognosis in these cases tends to be better (Matthew et al, 1995) (*Figures 4a* and *b*). When hypoxia is suspected in the acute setting, the cause needs to be established and rectified, e.g. respiratory tract infection or left heart failure, and supplemental oxygen is essential to avoid extension of cerebral infarction or exacerbate myocardial ischaemia if present. Oxygen saturation needs to be maintained at >95% (Ronning and Guldvog, 1999).

The presence of fever in cases where a source of sepsis has been excluded is an occasional consequence of the ischaemic stroke probably as a result of excessive metabolic demands and excessive release of neurotransmitters (Hajat et al, 2000). Efforts should be made to reduce the temperature using antipyretics as sustained fever leads to increased morbidity and mortality.

Myocardial infarction and atrial fibrillation may complicate clinical features (Myers et al, 1982; Lane et al, 1992) during the early presentation of ischaemic stroke. Thus patients must be continuously monitored in the first 48 hours of presentation and prompt adequate treatment of resulting cardiac failure provided. Should anticoagulation be required in those with atrial fibrillation then a wait of 2 weeks is essential (International Stroke Trial Collaborative Group, 1997). It is also not unusual for hyperglycaemia to complicate the management of cerebral infarction. This may be the first presentation of diabetes mellitus or a reaction to the neurological insult. Cerebral infarction associated with hyperglycaemia tends to have a poor outcome (Burns et al, 1999; Parsons et al, 2002), principally because of the reduced volume of salvageable tissue. The initial treatment of hyperglycaemia should be with an intravenous infusion of insulin. After 48 hours a review of the insulin requirements and the presenting clinical features will dictate whether long-term hypoglycaemic therapy is required and in what form.

Figure 3. A large left middle cerebral and anterior cerebral artery infarction producing a mass effect with midline shift to the right and compression of the left lateral ventricle. This infarction will lead to death if a left hemicraniectomy is not undertaken.

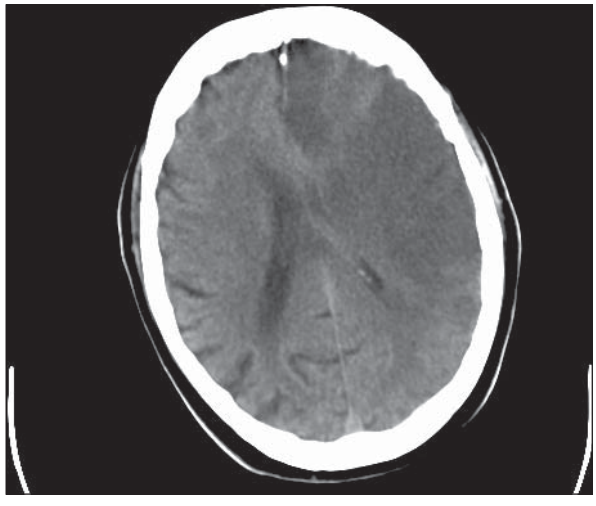
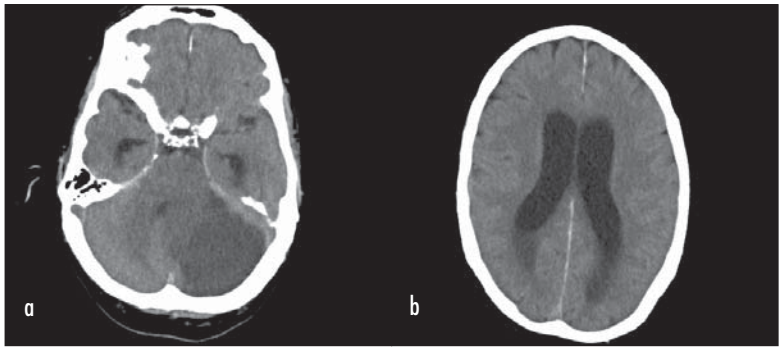


Figure 4 a and b. Left cerebellar infarction leading to acute secondary hydrocephalus that required emergency shunting. In these cases CSF shunting is undertaken for a short period waiting for the infarction to organize itself and restore the circulation of CSF.



The level of blood glucose at which insulin therapy should be initiated is debateable, but the American Stroke Association (Adams et al, 2003) recommends intervention when blood glucose is equal or greater than 11 mM.

Treatment of hypertension in the acute setting is controversial. There are no good studies to guide initial management and one can only proceed with what is common practice in most established stroke units. In most instances blood pressure tends to decline with time as patients become accustomed to the stroke unit or as pain or fever is controlled. Those who persist or have markedly elevated blood pressure warrant treatment if systolic blood pressure exceeds 220 mmHg or the diastolic pressure is greater than 120 mmHg (Powers, 1993).

However, if hypertension leads to cardiac compromise, e.g. pulmonary oedema or hypertensive encephalopathy, cautious blood pressure reduction is essential, to levels slightly less than those indicated. Either labetalol or glyceryl trinitrate infusions can be used intravenously, depending on individual circumstances. Lowering blood pressure is critical in the initiation of thrombolysis and 24 hours after it, and in such circumstances blood pressure must be maintained at no more than 185 mmHg systolic and 110 mmHg diastolic (Adams et al, 2005) (*Table 2*).

Younger patients with ischaemic stroke

Younger patients who present with ischaemic strokes pose a specific challenge. Those under 55 years of age should be screened for patent foramen ovale. The presence of foramen ovale in the population is around 25% (Hagen et al, 1984) and in most cases it does not cause any significant pathology. It is suggested that patent foramen ovale predisposes to ischaemic stroke by paradoxical embolism and additional procoagulant conditions predispose such patients to ischaemic stroke (Botto et al, 2007). However, it is becoming evident that patients over the age of 50 years classed as having cryptogenic ischaemic strokes are more likely to have patent foramen ovale than previously thought (Hande et al, 2007). Of particular importance are cases of atrial aneurysm associated with patent foramen ovale, where the association of patent foramen ovale with ischaemic strokes is even stronger.

Closure of patent foramen ovale may lead to reduced propensity to ischaemic stroke, but it is recommended that investigations for procoagulants be undertaken. In those with coexisting procoagulants, persisting atrial aneurysm or atrial fibrillation post-closure of the patent formane ovale, long-term anticoagulation is appropriate (Krumsdorf et al, 2004).

Stroke patients should be managed in dedicated stroke units from the time of admission. These units are staffed by experienced nurses, physiotherapists, occupational thera-

pists, speech therapists and clinical psychologists as well as dedicated stroke physicians or neurologists. The beneficial effects of stroke units are widely recognized (European Stroke Initiative, 2004), leading to significant reduction in mortality, dependency and need for institutionalization compared to such patients being managed in an acute medical ward. Complications in the acute phase are easily recognized and dealt with, thus improving outcomes.

Conclusions

With the safe clinical application of thrombolytic therapy in the mid-1990s, coupled with recent advances in other areas of management in the last decade and the setting up of effective stroke units, clinical outcome for patients with ischaemic stroke has improved markedly. Ischaemic strokes are no longer a hopeless clinical scenario. Patients who present early for thrombolytic therapy can look forward to a good prognosis. **BJHM**

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KEY POINTS

- Thrombolysis in the management of acute ischaemic stroke has been an important advance.
- Thrombolysis needs to be undertaken within 3 hours of an acute ischaemic event.
- Those not eligible for thrombolysis within the time window can be managed by mechanical thrombectomy.
- Hemicraniectomy is an important part of dealing with cerebral oedema as a result of a large middle cerebral artery infarction.
- Those with cerebellar infarction need to be monitored closely for resulting raised intracranial pressure in which case urgent decompression with an intraventricular catheter will be required to deal with acute hydrocephalus.
- Further studies are required regarding the control of acute hypertension after an acute ischaemic stroke.
- Cardiac and respiratory complications require adequate treatment.