New and emerging treatments for stroke

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Acute stroke is a treatable condition. Over the last 10 years, the benefit of a number of acute strategies has become established and evaluation of other potentially beneficial interventions is underway. This review will discuss the evidence-based management of the acute stroke patient, consider barriers to provision of such care and update the reader on emergent strategies, which may form part of stroke management in the future.

Keywords: stroke; thrombolysis; neuroprotection; haemostatic therapy

Introduction

Stroke is the single biggest cause of major disability in the UK [1] and is the third leading cause of death in most Western countries. The condition has a profound effect on patients and relatives and is associated with a vast economic burden [2, 3]. In the UK and other countries, these costs are on the rise and consistently consume ~5% of health care resources [3], costing the NHS and wider economy ~£7 billion per year [4]. Despite this, there are depressingly few effective treatments for acute stroke, with organized stroke care, early aspirin and thrombolytic treatment being the only proven therapeutic strategies.

The gross deficiencies in stroke care in the UK have been extensively highlighted in recent reports from the National Audit Office [5] and the House of Commons Public Accounts Committee [4]. Hopefully, this official acknowledgement will raise the profile of stroke care and research, lead to more widespread introduction of proven effective therapies and aid the development of new treatments. This review will highlight the new and emerging treatments for acute stroke, in particular those which may soon be part of routine clinical care. It will focus upon advances in imaging techniques, development and implementation of reperfusion strategies, neuroprotection and therapeutic hypothermia, advances in the management of intracerebral haemorrhage and the potential role of surgery in the treatment of stroke.
Importance of rapid assessment—the narrow therapeutic time window

Infarct volume increases in the first few hours after onset of ischaemic stroke, with the ischaemic penumbra gradually being subsumed into the area of infarction. The penumbra is the region where blood supply is significantly reduced, but energy metabolism is maintained because of collateral flow [6]. Its viability depends on the severity and duration of ischaemia, and if blood flow is swiftly restored, some penumbral tissues may be saved. However, during the time lag to reperfusion, a physiological cascade occurs, which places penumbral tissue at further risk. Cellular metabolism becomes anaerobic leading to acidosis, and sodium–potassium transporters become dysfunctional causing a rise in intracellular osmolarity and cytotoxic oedema. Intracellular calcium increases and ultimately the process becomes self-perpetuating with further rises in intracellular calcium, possibly because of release of intracellular glutamate [7]. Increased calcium concentration within cells enhances injury through activation of lipase, protease and free radical generation. Blood–brain barrier integrity is reduced [8], and if breached, blood components can enter the interstitial space causing vasogenic oedema and increasing the risk of haemorrhagic transformation of infarcted tissue. Furthermore, even if reperfusion occurs, the penumbra is vulnerable to the effects of reperfusion injury [9] as further free radical formation and release of harmful neurotransmitters may be provoked.

In the context of intracerebral haemorrhage, it has recently been recognized that early haemorrhage growth occurs in a significant number of patients [10, 11]. In a study of 103 patients, all of whom presented within 3 h of onset and underwent serial brain imaging with X-ray computed tomography (CT), substantial haemorrhage growth occurred in 28% of patients [11]. Haemorrhage growth was defined as a 33% increase in volume, occurred particularly within the first hour, and was associated with clinical deterioration (Fig. 1).

Thus, for both ischaemic and haemorrhagic stroke, there are therapeutic targets, which are likely to exist only in the early hours after stroke. This of course necessitates rapid assessment and investigation. However, the National Sentinel audit found that only 30% of those suspected stroke patients who were deemed to require urgent brain imaging (within 30 min) received a scan on the same day. Also, many ambulance services do not treat potential stroke patients as the highest category of emergency. Reducing delays to assessment and imaging to increase the numbers presenting within the first hours after onset of stroke is therefore crucial and will have a significant impact on patient outcomes. Such delays are multifactorial and will require comprehensive strategies to
increase public awareness and improve ambulance, emergency medicine and radiology services, whilst ensuring that all patients have equity of access to stroke unit care.

**Brain imaging techniques in suspected acute stroke**

The choice of brain imaging modality lies between CT and magnetic resonance imaging (MRI). All patients with suspected acute stroke should have an urgent brain scan to enable rapid differentiation of ischaemic from haemorrhagic stroke. Emergency scanning must be performed in those presenting within 3 h, with severe headache, fever, exposure to, or expected use of, anticoagulants and abnormal parameters of blood coagulation. Importantly, however, it must be recognized that there is no rationale for waiting to image patients—it delays treatment initiation and is the least cost-effective approach [12].

A non-contrast CT brain is the most widely available test and has excellent sensitivity for identification of haemorrhage early after onset. Its sensitivity is considerably less than that of MRI for the identification of ischaemic change within the first 24 h, but a normal scan is accepted as consistent with ischaemia if in association with relevant clinical signs. It is increasingly recognized that the sensitivity of CT for detection of haemorrhage falls dramatically after 7 days, thereafter MRI becomes the...
investigation of choice [13]. While an important point, this will rarely be an issue in the management of patients with acute stroke.

Newer CT- and MRI-based techniques are available which have the potential to increase both diagnostic sensitivity and specificity and increase the numbers of patients receiving treatments such as thrombolysis. MRI perfusion and diffusion imaging can distinguish ischaemic brain tissue at risk of infarction from the infarct core and thereby identify potentially salvageable brain tissue. Diffusion imaging is based upon the premise that during brain ischaemia, energy-dependent sodium/potassium ATPase pumps become dysfunctional leading to cytotoxic oedema. The movement of water molecules, or apparent diffusion, is reduced in such oedematous areas, which can be detected by fast MRI scanning. While such lesions show as hyperintense areas on diffusion images and provide a measure of extent of tissue injury, they may still appear normal on conventional MRI or CT images. As such, diffusion imaging is of value in the rapid identification of cerebral infarction and in the differentiation of new from old infarction (Fig. 2). Perfusion imaging measures the amount of MRI contrast, which enters the brain and allows maps of cerebral blood volume, cerebral blood flow and mean transit time to be created. This therefore provides a measure of the total

![Fig. 2](https://academic.oup.com/bmb/article-abstract/77-78/1/87/324775)
ischaemic area. Thus, areas that exhibit change in the perfusion image but not the diffusion sequence may represent the ischaemic penumbra—
isaemia but not yet injured tissue, which may be potentially salvageable [14]. Furthermore, the perfusion image parameters alone provide useful
information; areas with normal cerebral blood volume but reduced cere-
bral blood flow may survive, but areas of reduced cerebral blood vol-
ume are less readily salvageable.

Similar data can be gained during CT perfusion scanning, although
MRI is generally regarded as superior. Again, repeated scanning of an
area of the brain is performed as a bolus of intravenous contrast passes
through it allowing estimation of cerebral blood flow, mean transit time
and cerebral blood volume. As with MRI, there is some evidence that
these parameters can be used to predict whether areas of ischaemic brain
can survive [15].

It is hypothesized that such techniques will help direct treatment
strategies—if potentially salvageable areas of tissue are found, throm-
bolytic and other therapies may still prove beneficial, regardless of
time from onset. This will be discussed in more detail later, but these
developments make it crucial that access to more rapid and sophisti-
cated imaging is granted.

Management of acute stroke

Management of acute stroke [and transient ischaemic attack (TIA)]
serves a dual purpose—to reduce the high risk of recurrent stroke and to
reduce the burden of disability in established stroke. Secondary prevent-
ative measures continue to improve and have been reviewed elsewhere
[16]. Acute treatment of stroke involves control of physiological vari-
bles, strategies to reperfuse the ischaemic area, measures to reduce
growth of primary intracerebral haemorrhage (haemostatic therapy),
protection of the vulnerable, yet salvageable, ischaemic penumbra (neu-
roprotectant therapy) and surgery.

All patients should be cared for in a dedicated acute stroke unit, which
in itself saves lives and significantly improves functional outcomes [17,
18]. By definition, a stroke unit is an area and environment of organized
and multidisciplinary care, which ensures access to specialist medical,
nursing and allied staff and treatment.

Monitoring and control of physiological variables

Arterial hypertension occurs in as many as 80% of patients following
acute stroke [19]. It is associated with a poor outcome [20], but it may
represent a protective response—falls in blood pressure may lead to infarct extension because of impaired cerebrovascular autoregulation following acute stroke [21]. There is some evidence that a J-shaped relationship exists with arterial hypotension also being associated with a poor outcome [19]. Such uncertainty also surrounds intracerebral haemorrhage where hypertension may contribute to haemorrhage growth. At present, guidelines suggest lowering blood pressure in the presence of encephalopathy or of aortic aneurysm with renal involvement and withholding antihypertensive treatment unless systolic blood pressure is >220 mmHg or diastolic blood pressure is >120 mmHg. This leaves a great deal of uncertainty in the majority of patients, and it is unclear whether prior antihypertensive therapy should be discontinued in the acute phase and at what thresholds of blood pressure, we should intervene and the treatment targets that we should aim for. Fortunately, several large clinical trials are now underway and should address these issues [Controlling Hypertension and Hypotension Immediately Post-Stroke Trial (CHHIPS trial), Continue or Stop post-Stroke Antihypertensives Collaborative Study (COSSACS) and Efficacy of Nitric Oxide in Stroke Trial (ENOS trial)].

Elevated blood glucose in the acute phase following ischaemic stroke is associated with a poor outcome [22], regardless of the presence of pre-existing diabetes. In a recent systematic review of non-diabetic patients, stress hyperglycaemia defined as blood glucose of >6 or 7.1 mmol/l was strongly predictive of increased hospital mortality [relative risk (RR) = 3.28, 95% confidence interval (CI) = 2.32–4.64] and poor functional outcome (RR = 1.41, 95% CI = 1.16–1.73) [23]. This may be because elevated blood glucose increases brain lactate production, which is associated with increased infarct size [24], may reduce the efficacy of thrombolytic therapy [25] and may increase the risk of haemorrhagic transformation of infarcted tissue. Randomized controlled trials, such as the Glucose Insulin in Stroke trial (GIST-UK), are in progress to assess efficacy of intervention to lower elevated blood glucose, but in the meantime, guidelines suggest that glucose-containing fluids are avoided after acute stroke and either that ‘markedly elevated’ levels should be lowered or that levels should be maintained within normal limits [26, 27]. Either glucose–potassium–insulin or sliding scale regimens can be used.

Fever has also been associated with a poor outcome following acute stroke [28], possibly because of a detrimental effect on intracerebral metabolism, increased free radical production [29] or changes in blood–brain barrier function [30]. Guidelines suggest the maintenance of temperature within normal limits, using antipyretic agents if required, but it is again unclear whether this improves clinical outcomes.
Reperfusion strategies in acute ischaemic stroke

Intravenous thrombolytic therapy

Thrombolytic therapy with tissue plasminogen activator (rt-PA) is the only licensed treatment for acute ischaemic stroke in Europe. It must be administered within 3 h of symptom onset. The first evidence of efficacy was published in 1995, and licence was granted for use in the USA in 1997. In Europe, however, a conditional licence was first granted in 2002, and uptake has remained disappointingly slow. As many as two-thirds of otherwise eligible patients miss out on treatment because of delay in presentation and/or early misdiagnosis [31]. The UK performs particularly badly and currently holds the 15th place in the European league table of thrombolysis use: 238 patients (around 0.2% of stroke incidence) were treated in 2005. A target of ensuring delivery of thrombolysis to 5% of stroke patients has recently been set, and only a few isolated UK centres achieve this.

Intravenous thrombolytic therapy is clearly beneficial as shown in a recent pooled meta-analysis of the major thrombolysis studies [32]. The analysis considered 2775 patients treated within 6 h of ictus. The odds of favourable outcome were 2.8 (95% CI = 1.8–9.5) for treatment within 90 min and 1.6 (95% CI = 1.1–2.2) for treatment between 91 and 180 min. Benefit was still apparent for patients treated between 181 and 270 min (odds ratio = 1.4, 95% CI = 1.1–1.9). The rate of significant intracerebral haemorrhage was 5.9% in those treated with t-PA compared to 1.1% in those treated with placebo. However, only a small number of these were of clinical significance, and this risk of haemorrhage is already accounted for in the calculation of odds of favourable outcome. These results tell us that the chances of being free of handicap after stroke are increased nearly 3-fold by thrombolytic treatment, provided it is administered within 90 min of onset. Smaller but still significant benefits are seen up to 4.5 h. The number needed to treat (NNT) to achieve an excellent outcome (and avert one case of death or dependency) following treatment is ~7 [32]. The NNT to achieve a reduction in disability is much lower, at ~3, with a number needed to harm of 30 [33]. Thus, for every 100 patients treated with t-PA within 3 h, 32 will achieve a better outcome despite approximately three who will suffer significant haemorrhagic change.

It was initially feared that the risk of haemorrhage would be greater when rt-PA was used out with research settings. However, a rigorous audit of outcomes in several thousand European patients has confirmed that safety is at least as good as in trials, with low rates of symptomatic significant haemorrhage (unpublished data). Experienced centres still, however, achieve the best results.
The risk of haemorrhage should not deter use of thrombolytic therapy, but precautions must be taken to minimize its risk. The factors associated with an increased risk of haemorrhage are increasing age, extensive early infarct change on brain imaging, diabetes mellitus (DM), elevated blood glucose, DM and a history of previous stroke, and a low platelet count [34]. Those with higher baseline stroke severity may also have a higher risk of haemorrhage but equally may derive greatest benefit from treatment. Exclusion criteria are similar to those for thrombolysis in myocardial infarction, although patients with a blood pressure of >180/110 mmHg, or those who require treatment to attain a satisfactory blood pressure, are generally not treated. There are also concerns regarding its use in those with DM and a history of previous stroke. Mild systemic bleeding can also occur, and there is a risk of angiooedema of ∼1%, which is typically mild [35]. In practice, inexperienced stroke specialists usually err on the side of excessive caution, and as a result, many patients with milder strokes are deprived of the opportunity of cure.

**Intra-arterial thrombolysis**

Intra-arterial thrombolysis involves direct catheterization of an occluded artery and local administration of thrombolytic agents (rt-PA or urokinase). This appears to be an effective therapy for confirmed middle cerebral artery (MCA) or basilar artery occlusion. For example, in the Prolyse in Acute Cerebral Thromboembolism (PROACT II) trial [36], where treatment was initiated within 6 h of onset, reperfusion rates in those with confirmed MCA occlusion were significantly higher following intra-arterial thrombolysis with urokinase (66% compared with 18% in controls, $P < 0.001$). Clinical outcomes were also significantly improved with more patients living independently at day 90 (40% compared with 25% of controls, $P = 0.043$, relative risk reduction 58%).

Basilar artery occlusion carries a grave prognosis with a high mortality rate—perhaps in excess of 70%. While randomized controlled trial evidence is lacking, several case series have been published which suggest reduced mortality rate following intra-arterial thrombolysis [37, 38]. However, a recent systematic analysis suggested reperfusion rates, and clinical outcomes were similar with both intra-arterial and intravenous treatment [38] emphasizing the need for administration of either form of thrombolysis in this devastating condition.

Catheter-based techniques may allow lower systemic doses of thrombolytic agents to be used and the use of mechanical clot disruption and retrieval. This would be a particular advantage in those with a significant haemorrhage risk or in those unsuitable for intravenous rt-PA. In a
recent small series of 12 patients with basilar artery occlusion [39], six patients underwent successful mechanical recanalization. The remainder required intra-arterial thrombolysis, but where successful, mechanical recanalization afforded shorter treatment times and avoidance of the risks of thrombolytic therapy. A further larger study included 151 patients with ischaemic stroke and any treatable intracranial or extracranial artery occlusion who were deemed unsuitable for rt-PA [40]. Patients underwent embolectomy using a novel retrieval device within 8 h of symptom onset; a high rate of vascular recanalization was seen (46% compared with 18% in a historical control sample).

These techniques are promising, and although randomized controlled evidence is lacking, they represent a real alternative for those with major stroke who are unsuitable for rt-PA. Further study is required, but the limited availability and need for specialist neuroradiology staff may hinder introduction of these techniques into routine practice.

**Extending the window**

As mentioned above, there is evidence from existing randomized data that thrombolytic therapy is effective as late as 4.5 h after onset of symptoms. This is the subject of an ongoing randomized controlled trial (placebo-controlled trial of alteplase (rt-PA) in acute ischaemic hemispheric stroke where thrombolysis is initiated between 3 and 4 h after stroke onset—ECASS III), which will assess efficacy of thrombolysis in patients with ischaemic stroke treated between 3 and 4.5 h from onset. The possibility of extending the time-window further, to 6 h after onset of symptoms, is also being evaluated by a large randomized controlled trial (International Stroke Trial-3).

There is preliminary evidence that the use of MRI- or CT-based diffusion and perfusion scanning allows safe use of thrombolytic therapy up to 9 h after onset in selected patients. In the Desmoteplase in Acute Ischaemic Stroke (DIAS) trial [41], reperfusion rates following treatment with desmoteplase (at a dose of 125 μg/kg) were significantly higher when compared with placebo (71 versus 19.2%, \(P = 0.012\)), and clinical outcomes were also improved (favourable outcome in 60 versus 22.2%, \(P = 0.009\)). These results are supported by the more recent Dose Escalation of Desmoteplase for Acute Ischaemic Stroke (DEDAS) trial [42] where reperfusion rates and clinical outcomes were also improved in those treated with 125 μg/kg of desmoteplase. A further phase III study (DIAS II) is now underway and also incorporates a CT-based entry criterion.

If these trials suggest that the time-window can be safely extended, it may aid achievement of the target of thrombolysis delivery to 5% of stroke patients.
Neuroprotectant strategies

Neuroprotectant drugs aim to save ischaemic brain tissue by damping down the potentially harmful molecular processes in the penumbra. This may prolong the life of the penumbra, maintain blood–brain barrier integrity, reduce oedema and haemorrhagic transformation and reduce reperfusion injury. Such strategies may also be of benefit in the perihaemorrhage region in intracerebral haemorrhage, but to date, no neuroprotectant drugs have reached routine clinical practice.

However, evidence has recently emerged that NXY-059, a novel free radical trapping agent [43], may be an effective treatment. In the Stroke–Acute Ischaemic–NXY-059 (Cerovive) Treatment (SAINT I) trial [44], 1772 patients with acute ischaemic stroke were randomized to receive NXY-059 or placebo within 6 h of ictus. Treatment significantly improved disability at 3 months after stroke as measured by the modified Rankin scale; odds of improvement 1.2 (95% CI = 1.01–1.42) following NXY-059 compared to placebo. There was no reduction in mortality, but 4.4% more became asymptomatic (modified Rankin score of 0) and 3.7% more were able to walk without help (modified Rankin score of <4). A further intriguing result was a reduction in the rate of haemorrhagic transformation amongst those who received rt-PA and NXY-059 (2.5 versus 6.4% with placebo, P = 0.036). Trends to improvement were seen for secondary endpoints comprising other functional and disability scores, although statistical significance was not reached. Importantly, however, the trial was not powered to study these, and the mRs is widely regarded as the favoured disability endpoint in acute stroke trials [45, 46]. These results are encouraging; despite the modest treatment effect, the drug could be given to a large number of acute stroke patients, perhaps without the need for prior brain imaging, and there are few adverse events. Thus, if efficacy is confirmed by the ongoing SAINT II trial, the treatment could bring considerable reductions in dependency and in the economic cost of stroke.

Therapeutic hypothermia

The role of therapeutic hypothermia is being examined, and while safety and efficacy data are only beginning to emerge, induced hypothermia may represent a new treatment strategy in acute stroke [47]. There are several mechanisms by which hypothermia may convey a neuroprotectant mechanism. It will decrease cellular metabolism [48], limit cytotoxic and excitatory cascades, reduce free radical formation and suppress blood–brain barrier breakdown [49]. Mechanisms of cooling include surface cooling, which, while able to attain reductions in temperature
New and emerging treatments for stroke

[50], has disadvantages of patient discomfort. It may also necessitate sedation and paralysis to reduce shivering if more moderate degrees of hypothermia are required. The preferable, yet more invasive, approach is to use intravascular cooling via a central venous heat exchange catheter. This is typically inserted via a femoral vein into the inferior vena cava, is able to achieve target temperature rapidly and has few side effects [51]. It also allows greater control over rewarming, which may be associated with increases in intracranial pressure if performed rapidly [52].

There is a wealth of data to show that therapeutic hypothermia is effective in animal models of cerebral ischaemia, particularly with transient ischaemia where cooling was initiated within 1 h [53–55]. Less effect is seen in permanent ischaemia models. Therefore, like thrombolytic therapy, it is likely that therapeutic hypothermia will only prove effective if initiated early after onset and in patients with reperfusion (either spontaneous or iatrogenic). Mild hypothermia has been similarly effective to deeper levels in animal models [56] and is more feasible. The duration of hypothermia has varied in studies but in practical terms is likely to be around 24 h.

Evidence of efficacy in humans is lacking, although a recent case report [57] and some human feasibility studies [52, 58] provide prima facie evidence of efficacy. There is a need for further work to clarify efficacy, optimal degree and duration of hypothermia and the best rewarming strategy. Hopefully, trials such as The Nordic Cooling Stroke Study and Controlled Hypothermia in Large Infarction Trial will clarify the role of this promising and physiologically sensible treatment approach.

Specific treatment for intracerebral haemorrhage

Supportive treatment is indicated as for all types of stroke. Approximately 15% of cases of intracerebral haemorrhage are associated with warfarin. These patients have a higher risk of death and disability [59] and require rapid reversal of anticoagulation [60]. No specific licensed treatment exists for the majority with spontaneous intracerebral haemorrhage, but there are some exciting developments. Recombinant activated factor VII (rFVIIa) is a licensed treatment for bleeding in haemophiliacs who are resistant to factor VIII replacement. It is a powerful initiator of haemostasis even in patients with normal coagulation. It has been widely used in emergency surgery and trauma with considerable success. A recent clinical trial suggests that rFVIIa can limit early haemorrhage growth with ensuing reductions in mortality and disability, provided treatment is administered within 4 h of onset [61]. Three-month mortality was 18% in the treatment groups compared with 29% in the placebo group (a relative risk reduction of 38%, \( P = 0.02 \)). Because of
its procoagulant effect, treatment may transiently promote thrombo-
sis. Thromboembolic events were slightly more common with treat-
ment (7 versus 2%, \( P = 0.12 \)), but these risks must be kept in
perspective—most of the events were minor and not associated with
permanent harm, whereas the mortality of untreated intracerebral
haemorrhage exceeds 30% and only \(
\sim 20\% \) regain functional independence [62]. A trial to explore the risk–benefit ratio further should report
shortly [Recombinant Factor VIIa in Acute Intracerebral Haemorrhage
(FAST) trial].

**Surgical treatment of acute stroke**

Complete MCA infarction is associated with brain oedema, increased
intracranial pressure and a risk of transtentorial herniation and death
(Fig. 3). Medical therapy does little to improve mortality rates, which
may be as high as 80%. Decompressive hemicraniectomy can relieve
increased intracranial pressure and prevent herniation. Several case series
and a recent systematic review suggest that this improves mortality [63],
perhaps to as low as 30%. There is also some evidence that pre-emptive
hemicraniectomy should be considered in patients with complete MCA
infarction [64, 65]. The recent Decompressive Surgery for the Treatment

![Fig. 3](https://academic.oup.com/bmb/article-abstract/77-78/1/87/324775)

**Fig. 3** X-ray computed tomogram of a patient with massive cerebral infarction. Note
the swollen, oedematous brain with midline shift and compression of the adjacent
hemisphere.
of Malignant Infarction of the Middle Cerebral Artery (DESTINY) trial [66] showed a significant mortality reduction in those with severe MCA stroke involving at least 2/3 of its territory following surgery compared to standard conservative treatment (46.7 versus 88.2%). While this evidence clearly shows that hemicraniectomy is a viable treatment in some patients, further work is required to help identify those most likely to deteriorate despite medical treatment and define the optimal timing of treatment; for example, a recent work suggests that the highest risk lesions are those involving additional vascular territories to the MCA [67]. More importantly, robust randomized controlled evidence is required to show not just reduced mortality but that surviving patients have reasonable functional outcomes. At least three randomized controlled trials are in progress, aiming to recruit 35 [Decompressive Craniectomy in Malignant Middle Cerebral Artery Infarcts (DECIMAL)], 56 [Hemicraniectomy for Malignant Middle Cerebral Artery Infarcts (HeMMI)] and 112 [Hemicraniectomy after MCA Infarction with Life-threatening Edema Trial (HAMLET)] patients, respectively. Larger studies may be required to assess functional outcomes.

Conclusion

Numerous promising advances have been made over the recent years. In particular, if results of the SAINT II, DIAS II and FAST trials confirm those of previous studies, there is a very real possibility that “late” thrombolytic therapy, neuroprotectant strategies and haemostatic therapies for ICH will soon become part of routine stroke care. Combined with the increasing political attention, these developments will hopefully ensure reductions in both mortality and morbidity.

All of these treatments require rapid access to brain imaging, which is unavailable in many centres. However, imaging is ultimately required in all stroke patients, and it is increasingly clear that immediate or emergency imaging is the most cost-effective approach. More immediate access to imaging for stroke patients is therefore both logical and in the interest of patients and will help ensure maximum efficacy of new and current treatments.

References


New and emerging treatments for stroke


