Acute Stroke and Transient Ischaemic Attack in the Emergency Department

Evidence based review

Written for the College of Emergency Medicine e-learning programme in collaboration with Doctors.net.uk
Stroke Classification

Stroke can initially be classified according to its pathological origins:

**Ischaemic (69%):**
- caused by thrombus building up within an artery
- embolism from an extracranial site
- hypoperfusion due to reduced cerebral blood flow

**Haemorrhagic (19%):**
- more common in young patients
- intracerebral vessel origin in 13%
- subarachnoid vessel origin in 6%

**Uncertain cause (12%)**

**Thrombosis**

A thrombus usually forms around atherosclerotic plaques causing narrowing of the vessel lumen and results in turbulent blood flow through the area of stenosis. This turbulent flow may lead to intimal disruption or plaque rupture, both of which activate the clotting cascade. This causes platelets to adhere to the plaque surface, where they eventually form a fibrin clot.

As the lumen of the vessel becomes more occluded, ischaemia develops distal to the obstruction resulting in a TIA or stroke. Alternatively, the fibrin clot may embolise distally.

The commonest areas are at arterial branch points, where there is already increased flow turbulence. Thrombosis occurs in various sizes of arteries, resulting in different syndromes. Lipohyalinosis (build-up of fatty hyaline matter in the blood vessel as a result of high blood pressure and ageing) and microatheroma are the main causes of small vessel ischaemic disease, and typically result in lacunar infarcts (small “lake-like” infarcts) that are found at autopsy in affected patients.

Arterial dissection may also lead to thrombus formation eg in trauma or arteritis.

**Embolus**

An embolus occurs when dislodged thrombus travel distally and occlude vessels downstream. The usual sites of origin are:

**Cardiac:**
- turbulence around the valves – due to endocarditis or a prosthetic valve
- turbulence within the chambers – atrial fibrillation (AF)
- ventricular aneurysm
- dilated cardiomyopathy
- congestive cardiac disease
- atrial myxoma
- post-myocardial infarction incidence of stroke is 2-3%, most occurring within 1 month

**Distal sites embolising through a patent foramen ovale**

Atherosclerotic lesions in the aortic arch, carotid arteries, and vertebral arteries

**Systemic hypoperfusion**

Any cause of systemic hypotension such as cardiac arrhythmias, septic shock or massive blood loss can cause a stroke. A reduction in cerebral perfusion pressure activates the cerebral autoregulatory system. As the small arterioles constrict in an attempt to maintain pressure, ischaemia can develop in the distal branches of the vascular tree. An area of the brain that lies between two major vascular supplies (eg the middle and anterior cerebral arteries) is known as a watershed area. These areas are especially prone to ischaemia during episodes of systemic hypotension.

**Other causes of ischaemic stroke include:**
- hyperviscosity syndromes – polycythaemia, multiple myeloma, sickle cell disease
- arteritis – in rheumatoid arthritis, systemic lupus erythematosus, polyarteritis nodosa etc
- venous thrombosis
- dissection

**Haemorrhagic stroke**

Intracerebral haemorrhage is the result of the rupture of a vessel within the brain parenchyma. It can be divided into:

**Primary:**
- hypertension
- amyloid angiopathy (the deposition of amyloid protein in vessel walls causing them to become more rigid, fragile, and prone to rupture)

**Secondary:**
- aneurysm
- arteriovenous malformations
- neoplasms
- trauma
- anticoagulation
- use of thrombolytics
- haemorrhagic conversion of an ischaemic stroke

Normal blood flow is 50 mL/min per 100 g brain tissue. This can reduce to nearly 20 mL/min whilst still maintaining normal function. However, if perfusion drops to less than 20 mL/min, function is impaired. Within seconds, an ischaemic cascade begins. The central area of infarction is irreversibly damaged - 1.9 million neurones die for each minute that a stroke is left untreated. This is, however, surrounded by the ischaemic penumbra, the area with potential for reversibility. This is therefore the target for treatment.
An exponentially increasing cycle is generated in the ischaemic area where the loss of adenosine triphosphate (ATP) leads to calcium influx, causing the release of multiple neurotransmitters, including glutamate. This activates excitatory receptors on other neurones, which in turn release further calcium, compounding the situation. Enzymes are released which break down the cell membrane. An inflammatory response is generated, involving free radicals, nitric oxide and cytokines which cause further neuronal damage. Therefore, without treatment or neuronal protective measures, the ischaemic penumbra becomes permanently damaged. This can occur as early as a few hours after the original ischaemic insult.

Treatment is aimed at both restoring flow to the ischaemic penumbra and also at preserving and protecting as many neurones as possible. Time is critical. Many measures are only effective early on in the generation of the ischaemic cascade.

### Anatomy of the Circle of Willis

Knowledge of the arterial blood supply to the brain is important to appreciate the various presentations and significance of cerebrovascular disease.

The Circle of Willis is supplied anteriorly by the two internal carotid arteries which form the anterior cerebral artery and the middle cerebral artery. Posteriorly it is supplied by the two vertebral arteries which form the basilar artery, which in turn divides to form the posterior cerebral arteries.

![Anatomy of Circle of Willis](image)
Key

A: anterior cerebral artery – supplies frontal and parietal lobes and most of corpus callosum
B: anterior communicating artery
C: middle cerebral artery – supplies frontal, parietal, temporal, anterior occipital lobes and basal ganglia and internal capsule
D: posterior communicating artery
E: posterior cerebral artery – supplies occipital lobe
F: superior cerebellar artery
G: basilar artery
H: anterior inferior cerebellar artery
I: vertebral artery
J: anterior spinal artery
K: posterior inferior cerebellar artery

Risk factors for stroke

Many risk factors are modifiable. About 20,000 strokes per year could be prevented with better control of blood pressure, cardiac arrhythmias, improved statin use and smoking cessation (Department of Health (DH), 2007).

Previous stroke

There is a 10% risk that people who have had a stroke will have another in the first year, and a 30-43% risk of a second stroke within 5 years (The Stroke Association, 2006).

Transient ischaemic attack (TIA)

TIA is defined as stroke symptoms and signs that resolve within 24 hours, though in practice, the symptoms usually last a few minutes or hours at most. These patients have a high risk of stroke in the near future – overall there is a 20% risk in the following 4 weeks.

Beware of TIA mimics – the commonest including:

- syncope
- migraine
- anxiety
- seizures/ post-ictal
- acute confusion
- global confusion
- transient amnesia

There are two main questions to be asked when examining a patient with neurological symptoms that have fully resolved and where TIA mimics are less likely:

- Is the patient at high risk or low risk of subsequent stroke?
- Are the symptoms within the carotid territory or the posterior circulation?
Risk stratification

Risk stratification is achieved using the ABCD2 scoring tool:

| A: age ≥60 | 1 point |
| B: blood pressure ≥140/90 (either one of these values reaching 140 or 90) | 1 point |
| C: clinical symptoms | 2 points |
| ● unilateral weakness | 1 point |
| ● only speech affected | 0 points |
| ● other neurological deficit | |
| D: duration of symptoms | 2 points |
| ● ≥1 hour | 1 point |
| ● 10 – 59 minutes | 0 points |
| ● <10 minutes | |
| D: diabetes | 1 point |

Table 1. ABCD2 scoring tool (Johnston et al., 2007)

A score of 4 or more should be considered high risk. Patients with crescendo TIA i.e two or more episodes of neurological deficit in a week are automatically high risk, even if their ABCD2 score is 3 or less. Scores in relation to risk of stroke:

<table>
<thead>
<tr>
<th>ABCD2 score</th>
<th>2-day risk of stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 or 1</td>
<td>0%</td>
</tr>
<tr>
<td>2</td>
<td>1 – 2%</td>
</tr>
<tr>
<td>3</td>
<td>up to 3%</td>
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<tr>
<td>4</td>
<td>2 – 5%</td>
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<tr>
<td>5</td>
<td>3 – 7%</td>
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<tr>
<td>6</td>
<td>4 – 14%</td>
</tr>
<tr>
<td>7</td>
<td>up to 50%</td>
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</tbody>
</table>

Table 2. ABCD2 scores in relation to 2-day risk of stroke
The next thing to determine is whether the symptoms are predominantly of carotid origin (usually around 80% of cases) or vertebrobasilar:

<table>
<thead>
<tr>
<th>Territory</th>
<th>Symptoms</th>
</tr>
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<tbody>
<tr>
<td>Carotid</td>
<td>Weakness of leg and arm</td>
</tr>
<tr>
<td></td>
<td>Clumsiness</td>
</tr>
<tr>
<td></td>
<td>Amaurosis fugax</td>
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<tr>
<td></td>
<td>Recognition problems</td>
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<td></td>
<td>Sensory deficit paralleling weakness</td>
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<tr>
<td></td>
<td>Hemianopia</td>
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<tr>
<td></td>
<td>Dysphasia</td>
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<tr>
<td>Vertebrobasilar</td>
<td>Vertigo</td>
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<tr>
<td></td>
<td>Facial parasthesia</td>
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<tr>
<td></td>
<td>Diplopia</td>
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<tr>
<td></td>
<td>Bilateral spasticity</td>
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<tr>
<td></td>
<td>Nystagmus</td>
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<tr>
<td></td>
<td>Dysarthria</td>
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<tr>
<td></td>
<td>Visual field deficits</td>
</tr>
</tbody>
</table>

Table 3. Typical carotid and vertebrobasilar symptoms of TIA

General features such as confusion and amnesia are common.

If the symptoms correlate with the carotid territory, carotid imaging is required (by Doppler ultrasound, computed tomography angiography (CTA) or magnetic resonance angiogram (MRA)) to determine whether there is significant narrowing within the carotid arteries. This can be treated by carotid endarterectomy, an operation which is performed either under general or local anaesthetic to clear the obstructing plaque from the carotid lumen.

Significant narrowing is defined as 50-99% according to the North American Symptomatic Carotid Endarterectomy Trial criteria (Ferguson et al, 1999) or 70-99% according to the European Carotid Surgery Trialists’ Collaborative Group criteria (ECST, 1998).

All patients should also receive best the medical care, which includes:

- **antiplatelet agents** (all patients should receive 300 mg aspirin, continued as low dose 75 mg indefinitely, but if aspirin alone is ineffective, it should be combined with dipyridamole)
- control of **blood pressure**
- lowering of **cholesterol**
- **diet** and lifestyle advice

The remaining question is now “How quickly do patients need investigating?” Patients who are at high risk on the ABCD2 scale or have crescendo symptoms should be investigated within 24 hours. This includes carotid imaging. If patients are deemed to be less of a risk, they should still be investigated within a week.

If significant carotid stenosis is found, there is slight controversy over how quickly an endarterectomy should be performed. The National Stroke Strategy (DH, 2007) recommends a maximum of 48 hours, but the National Institute for Health and Clinical Excellence (NICE, 2008) states that assessment for endarterectomy should occur within a week and surgery within 2 weeks. Note that there is a small risk of stroke peri and postoperatively. The sooner the surgery is performed, the greater the risk. Overall, 3% of asymptomatic and 6% of symptomatic patients will have a stroke or die post-endarterectomy.

Other complications of endarterectomy include haematoma compressing the trachea, and hyperperfusion syndrome which is due to the sudden increase in blood flow distal to the now-clear stenosis. This manifests as headache, contralateral neurological deficits and seizures. Although it usually occurs within the first week, it can occur up to a month postoperatively and could present acutely to the emergency department (ED).
If it is not clear about the diagnosis of TIA, which vascular territory is involved, or the cause of the symptoms, the aim is to perform diffusion weighted MRI (DWI) or MRA within 24 hours. This is clearly ambitious. However, patients who have normal DWI MRI, irrespective of their ABCD2 are significantly less likely to have a disabling ischaemic stroke at 90 days (Asimos et al., 2009).

Many EDs now have pathways in place to fast track high-risk TIA patients. Optimising medical care and providing urgent surgical intervention in those appropriate could reduce the number who go on to have a stroke by 80%. The EXPRESS trial (Luengo-Fernandez et al., 2007) also showed this would reduce further hospital admissions and bed days, saving £624 per patient, a total saving of 290,000 hospital bed days and £68 million in acute care costs.

Other risk factors for stroke and TIA:

**Hypertension**

It is estimated this causes 50% of ischaemic strokes. Hypertension is defined as blood pressure greater than or equal to 140/90. It is found in around a third of men and women in England, but the majority is not on any treatment and of those who are, nearly 60% are still hypertensive (British Heart Foundation Health Promotion Research Group, 2006).

**Smoking**

Ten per cent of stroke deaths are attributable to smoking (Health Select Committee, 2000). It raises the risk of stroke by two to four times of that of a non-smoker (Goldstein et al., 2006). It takes 5 years after stopping to reduce the risk of stroke down to that of a non-smoker.

**Diabetes**

This increases the risk of stroke by two to three times (Diabetes UK, 2004). It has been diagnosed in around 3-4% of women and men in England.

**Hypercholesterolaemia**

High levels of cholesterol (>5 mmol/L) significantly increase the risk of stroke (The Stroke Association, 2006). The aim is to give statin therapy to all people with a greater than 20% risk of cardiovascular disease.

**Diet**

Salt increases blood pressure and may also have a direct effect on stroke risk, and the average daily intake is far higher than the recommended level. Eating fruit and vegetables reduces the risk of stroke. Wholegrain foods reduce stroke risk specifically in women (The Stroke Association, 2006).
Alcohol

In men, drinking more than 35 units per week doubles the risk of dying from stroke compared with non-drinkers (The Stroke Association, 2006).

Recreational drugs

Cocaine, amphetamines, ecstasy and other recreational drugs increase the risk of both ischaemic and haemorrhagic stroke. This especially holds true for younger patients with arteriovenous malformations and haemorrhagic stroke (The Stroke Association, 2009a).

Exercise

Moderate activity reduces the risk of stroke by up to 27% (The Stroke Association, 2006).

Socioeconomic factors

Low socioeconomic groups have an increased risk of stroke (Department of Health (DH), 2007).

Atrial fibrillation (AF)

AF is found in 15% of stroke patients (The Stroke Association, 2006). Ischaemic heart disease and left ventricular hypertrophy may lead to AF or be independent risk factors.

Non-modifiable risk factors

Sex

Under the age of 75, more men have strokes than women. However, women are one-and-half times more likely to die from stroke than men (The Stroke Association, 2009b). Women aged 45-54 have a higher risk of stroke than men of the same age. Also, women are more likely to inherit an increased risk of stroke than men.

There are special considerations in women. Some are modifiable, others less so. The relationship between hormones, i.e. oestrogen and progesterone, and stroke is mixed. Oestrogen may reduce atherosclerotic narrowing and help keep high-density lipoprotein (HDL) cholesterol high, thereby reducing the risk of stroke in women, especially those who are pre-menopausal. However, the oral contraceptive pill (COC) causes the risk of stroke to increase – the higher the oestrogen dose, the higher the risk. This is especially true of the third-generation COC which increases clotting tendency. The effect is compounded by smoking. It should be noted, however, that the overall risk is still very small.

In pregnancy, as the circulating blood volume increases, so does the risk of clotting and stroke. The risk of stroke is maximal in the last trimester of pregnancy and for 6 weeks postpartum.

Pre-eclampsia may affect the arteries, causing narrowing and therefore ischaemia, or more rarely, an intracerebral bleed.
HRT increases the risk of stroke in women who are already at high risk, eg personal history of stroke or ischaemic heart disease. HRT leads to higher blood pressure and abnormal clotting.

Women aged 20-44 who have migraines or a family history of migraine have an increased risk of ischaemic stroke. This is compounded if they are hypertensive, smokers or taking the COC.

**Age**

Stroke is more common over the age of 55. The risk doubles roughly every decade.

**Family history**

This increases the risk, possibly due to the familial tendencies of diabetes and hypertension.

**Ethnicity**

Afro-Caribbeans are two times more likely to suffer a stroke than those of Caucasian ethnicity, and tend to have strokes at a younger age. African, Afro-Caribbean and South Asian people have higher rates of hypertension, diabetes and sickle cell disease (The Stroke Association, 2006).

**Other**

Conditions which result in hyperviscosity such as polycythaemia and sickle cell disease are risk factors. Hyperhomocysteinuria is also an independent risk factor.

**Management of stroke**

There are three main components of integrated stroke services:

1. Primary and secondary prevention
2. Acute care
3. Early and continuing rehabilitation. This should be provided by an appropriately skilled multidisciplinary team (MDT)

**Diagnosis**

The first point at which a stroke needs to be recognised is by the patient themselves, or their families. The Department of Health launched the ACT F.A.S.T. campaign on 9th February 2009 (Department of Health, 2009). This is a 3-year campaign to promote public awareness of stroke, advertising on TV, radio, online and in print. F.A.S.T. is a system developed in Newcastle for paramedics to use in the rapid recognition of stroke. It has a 79% pick-up rate.
The FAST system comprises four key points:

F  Face – is there new weakness of one side of the face?
A  Arm – can the patient hold both arms up?
S  Speech – is there new slurring of speech?
T  Time to call an ambulance if these tests are positive

The next tool in the rapid recognition of stroke is the Recognition Of Stroke In the Emergency Room (ROSIER) scale (Nor et al, 2005), again developed in Newcastle. It has been shown to be 92% sensitive and 96% specific for stroke identification:

Is there a NEW, ACUTE onset (or on awakening from sleep) in the following:

- asymmetrical facial weakness: Yes (+1 pt) No (0 pts)
- asymmetrical arm weakness: Yes (+1 pt) No (0 pts)
- asymmetrical leg weakness: Yes (+1 pt) No (0 pts)
- speech disturbance: Yes (+1 pt) No (0 pts)
- visual field defect or ophthalmoplegia: Yes (+1 pt) No (0 pts)
- loss of consciousness or syncope: No (0 pts) Yes (-1 pt)
- any seizure activity: No (0 pts) Yes (-1 pt)

Total score………… (-2 to +5)

Stroke is unlikely, but not excluded if total score is less than 0.

We still need to beware of stroke mimics – these can be particularly convincing in both symptoms and signs. The commonest include hypoglycaemia, post-ictal state, infection, tumours, toxins/metabolic disturbances and demyelinating conditions.

**Approach to the patient with stroke – history and examination**

Most ischaemic events start suddenly, which means the patient or their family should be able to tell when symptoms began or what they were doing when symptoms started. Symptoms also tend to be maximal at onset. Some patients wake with symptoms, so for the purposes of treatment times, the time that should be documented is when they were last observed free of neurological deficit.

Care should be taken to ensure the symptoms described are of new onset. This can be particularly difficult in the presence of a previous cerebrovascular event which has some residual deficit. Often, the patient’s perception of their new deficit can be quite different from that of their family, so multiple opinions should be sought. This is especially true where a patient has demonstrated a degree of confusion, memory loss, or has long-term memory problems. Trauma should not be ruled out. Evidence should also be sought of possible stroke mimics, and of past medical history and drug history to identify possible risk factors.

Examination includes a full general examination, paying particular attention to any cardiovascular signs including carotid bruits, bilateral blood pressure and the presence of atrial fibrillation. It is important to establish a baseline neurological deficit, involving:

- Glasgow Coma Scale (GCS), (Teasdale and Jennett, 1974)
- cranial nerve problems
- communication
• limb tone, power, sensation, reflexes with plantars, coordination
• any inattention or neglect and co-ordination

Often, signs can be elicited that correspond with the symptoms described, which in turn can have important prognostic significance. There are four main stroke syndromes:

**Total anterior circulation stroke (TACS)** (NB significantly poorer prognosis)
A triad of:
• weakness/ numbness of at least two of the face, arm and/or leg
• homonymous visual field defect
• dysphasia, dyscalculia or visuospatial disorder

**Partial anterior circulation stroke (PACS)**
Two of the three TACS criteria:
• isolated dysphasia, dyscalculia or visual field defect or limb weakness
• weakness of one limb only

**Posterior circulation stroke (POCS)**
• brainstem or cerebellar symptoms or signs
• ipsilateral cranial nerve palsy with contralateral motor or sensory signs
• bilateral motor/ sensory symptoms
• defect in conjugate gaze
• isolated hemianopia

**Lacunar stroke (LACS)**
• pure motor weakness of face, arm and/or leg
• pure sensory deficit of face, arm and/or leg
• sensorimotor – combination of above
• no cortical signs or symptoms
• ataxic hemiparesis, ie weakness and clumsiness

The Europe-wide method of assessing stroke severity is known as the National Institutes of Health Stroke Severity (NIHSS) Scale [pdf]. This is a way of quantifying symptoms and signs of stroke on a numerical scale, thus producing an idea of the baseline deficit, with which all further improvements or deteriorations can be compared.

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**Treatment of acute stroke**

The crucial point here is stroke is treatable. Rapid, coordinated stroke care is shown to reduce mortality and improve outcome in terms of disability severity.
General treatment

Airway: In patients with reduced GCS, the airway may become problematic. Patients with stroke may have altered swallowing and gag reflexes anyway, further compromising the airway.

Breathing: Oxygen should be applied if saturations drop below 95%, bearing in mind patients with type 2 respiratory failure or those with depressed respiratory effort secondary to their neurological deficit. The commonest causes of hypoxia in a stroke patient are:

- partial airway obstruction
- hypoventilation
- aspiration
- atelectasis

Circulation: Stroke patients need intravenous (IV) access, cardiac monitoring and a 12-lead ECG to look for risk factors such as AF, acute MI (occurs in 3%) or arrhythmia (occurs in 4%). An acute neurological event can itself result in cardiac dysfunction causing neurogenic pulmonary oedema.

Blood should be taken for:

- full blood count – looking for anaemia, polycythaemia, thrombocytosis, thrombocytopenia, sepsis, leukaemia
- urea and electrolytes – looking for uraemia, hyponatraemia
- clotting screen – looking for coagulopathy. This will alter the ability to thrombolysie. If the cause of the neurological deficit is found to be an intracerebral haemorrhage, the coagulation screen and platelets are invaluable, especially if the patient has a reversible condition such as an overdose of warfarin
- random blood sugar or nearside glucose testing – hypoglycaemia is a common stroke mimic and high sugar is known to adversely affect the outcome of stroke
- cholesterol

Imaging in acute stroke

The primary imaging modality in the UK is computed tomography (CT) scanning. This is sufficient to determine whether a stroke is due to ischaemia or haemorrhage. Indications for immediate non-contrast brain CT scanning (ie the next available slot within “office” hours or within an hour if out of hours) are:

- potentially eligible for thrombolysis or early anticoagulation
- taking anticoagulants
- known bleeding tendency
- GCS <13
- unexplained progressive or fluctuating symptoms
- papilloedema, neck stiffness or fever
- severe headache with onset of neurological deficit

All other acute stroke patients should have a CT scan within 24 hours. It has been shown that hospitals that perform CT scanning within 48 hours of admission in their stroke patients have an 8% lower incidence of death in hospital (The Stroke Association, 2006).
While non-contrast CT is still the commonest form of imaging, there are far more sophisticated investigations available which are able to provide more information on the size and area of the ischaemic core and surrounding penumbra.

Non-contrast CT rarely shows any changes within the first few hours of ischaemia, but after 6 to 12 hours, there is sufficient oedema to show as a hypodense area. Hypodensity on the initial CT early on is associated with poor outcome.

CT perfusion scanning: IV contrast is used to show perfusion in areas of the brain – hypodensity corresponds with the ischaemic penumbra, giving an impression of the degree of viability.

CT angiography: a reduction in perfusion appears hypodense, corresponding with a filling defect in a cerebral artery. It is also more likely to reveal a subarachnoid haemorrhage than non-contrast CT.

Magnetic Resonance Imaging (MRI): as MRI becomes more available, this will provide more accurate, earlier diagnosis and prognosis. As well as the standard MRI, there is also diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI). MRI is far more sensitive at showing the size of an infarct in the early stages: cellular oedema is visible on DWI within just 10-15 minutes of symptom onset. DWI can also show small areas of ischaemia in areas where CT is less sensitive, i.e. the cerebellum and brainstem. MR angiography can also show vascular defects.

Specific treatment of stroke

Stroke units

All patients should be admitted to a stroke unit. This is a specialist area with multidisciplinary staffing and equipment for both neurological and physiological monitoring of patients and their subsequent early rehabilitation. General functions include:

- full monitoring: neurology and physiology
- critical care, especially in the first 24 hours and if complications should occur, with input from appropriate specialists
- physiotherapy: respiratory and mobility
- speech and language, including swallowing assessment
- nutrition screening
- communication between the patient, their family and the services

Stroke units are the most effective “intervention” to reduce death or disability. They benefit all patients, but especially those with the most severe symptoms. This is due to relatively simple measures including prevention of aspiration, swallowing assessment and early rehabilitation, with early discharge planning. Since most stroke progression occurs in the first 24 hours, this is the crucial time to be monitored by the specialist team.

Blood pressure (BP) control

Hypertension is not normally treated in the acute setting due to the risk of reduction in cerebral perfusion pressure or watershed hypoperfusion. However, it should be considered in:

- patients who would otherwise be candidates for thrombolysis, whose BP is >185/110
- intracerebral haemorrhage with systolic BP >200
- hypertensive encephalopathy or nephropathy
- hypertensive cardiac failure or myocardial infarction
- pre-eclampsia or eclampsia

Rapid reduction in BP is likely to be harmful, causing watershed ischaemia. Therefore, controlled reduction with a slow bolus of IV labetalol or a titrated infusion of IV nicardipine is more appropriate.

**Tight glycaemic control**

High glucose levels result in reduction of reperfusion in thrombolysis and extension of the infarcted territory, resulting in a poor outcome. Therefore, blood sugar should be maintained between 4 and 11 mmol/L.

**Prevention of pyrexia**

High temperature increases ischaemic injury within the brain and is therefore associated with a poor outcome. Paracetamol should be given for pyrexia. Conversely, mild hypothermia preserves neurons.

**Early swallowing assessment**

Difficulty in swallowing after stroke predisposes to aspiration pneumonia. This increases mortality, therefore early assessment of dysphagia is essential. Any problems identified on a simple examination should result in specialist opinion within 24 hours and nasogastric feeding.

**Haemorrhagic stroke**

In haemorrhagic stroke, an accurate haemoglobin, platelet count and clotting function screen is essential. For patients who are taking warfarin, the INR should be brought below 1.5. This can be achieved with IV vitamin K, which takes effect within 6-8 hours, and IV prothrombin complex concentrate which works instantaneously. Note, the prothrombin complex wears off within a few hours, so patients will bleed unless also given vitamin K.

**Drug therapy**

**Thrombolytics**

In order to deliver a stroke thrombolysis service, the ED concerned must:
- be able to deliver thrombolytic therapy in less than 180 minutes since onset of signs
- be able to exclude haemorrhage by prompt CT
- fulfill National Institute of Neurological Disorders and Stroke (NINDS) criteria
- be registered with SITS–MOST (Safe Implementation of Thrombolysis in Stroke Monitoring Study) – this is the Europe-wide monitoring/audit study (*Wahlgren et al.*, 2008)
When considering medication in the treatment of acute stroke, the first question concerns suitability for thrombolysis. This is time-critical – the time of onset of symptoms must be known. Therefore those waking with symptoms are automatically excluded.

The initial evidence shows that for suitable patients who are treated within the first three hours after onset of symptoms, the percentage who are independent and well (modified Rankin score less than 2) at three months increases from 38% to 50%. The percentage who suffer haemorrhagic transformation changes from about 1% to 6%. However, overall, the percentage who die as a result of the stroke remains unchanged at 8-12% at 30 days. The number needed to treat is 7 (Wahlgren et al., 2008).

This benefit is only maintained by excluding those at highest risk of either death or haemorrhage. If the NIHSS score is very high, benefit will not be seen as these patients are likely to die anyway. Conversely, if the NIHSS score is low, the risk of bleeding outweighs any benefit as these patients are likely to remain independent. Older patients are also more likely to haemorrhage, so the age of 80 is the usual cut-off.

More recently, the European Cooperative Acute Stroke Study (ECASS) III trial (Hacke et al., 2008) has shown no increase in mortality or bleeding risk up to 4.5 hours post-symptom onset. And in centres enrolled in the IST-3 trial, enrolment can occur in the over 80s and up to 6 hours post-onset. This does not mean patients can be left longer – the quicker they are treated, the better the outcome.

Therefore, in patients who are not overtly at risk of secondary haemorrhage or bleeding elsewhere eg major surgery in the preceding 3 months or previous intracerebral haemorrhage, they should have a CT brain performed as soon as possible. They need to have platelet levels and clotting checked – the latter takes a minimum of 20 minutes on the analyser, so if clotting is likely to be normal, thrombolysis should not be delayed awaiting the results. The main contraindications to thrombolysis on CT are bleeding or an obvious infarct greater than a third of the middle cerebral artery territory.

Alteplase is the only thrombolytic licensed for use in acute stroke. It should only be administered by those with specialist training in stroke thrombolysis.

**Antiplatelet medication**

In patients who are not suitable for thrombolysis, they should, wherever possible, receive 300 mg aspirin. A previous history of dyspepsia should be countered with a proton pump inhibitor in addition to the aspirin. In case of severe intolerance to aspirin, clopidogrel 300 mg can be given instead.

**Complications of thrombolysis**

**Haemorrhagic transformation**

Ischaemic damage within the brain results in friability of the parenchyma. It is therefore much more susceptible to bleeding. Symptomatic haemorrhage is defined as haemorrhage seen on CT scan with a drop in the NIHSS score of 4 or more points. It occurs in around 1% of stroke patients without the addition of thrombolytics. With this medication, 6% suffer symptomatic bleeding (Wahlgren et al., 2008).

Symptoms include headache, nausea and vomiting, new neurological symptoms or signs, fall in GCS or acute hypertension. If haemorrhage is suspected, the alteplase should be stopped if still running, and any clotting deficit corrected. Neurosurgical opinion should then be sought if appropriate.
Hypotension

This is often transient. Oxygen should be given, a downward head tilt and possibly a fluid challenge.

Uncontrolled hypertension

Two readings should be taken, 5-10 minutes apart. The target BP should be <185/110. The reduction in BP should be gradual.

Anaphylaxis

Assess ABCs for shock and treat as appropriate, though never with intramuscular medication.

Palliation

Not all patients will survive: 45,000 patients die each year as a result of stroke. Occasionally, it is apparent from the outset that there is no hope of recovery, and in some cases it is not appropriate to move the patient from the ED. In these instances, good communication with family is imperative. Arrangements will need to be made for patient’s dependents if necessary; the patient may have had no previous disability and have had a dependent partner.

Further care

Multi-faceted stroke rehabilitation involves:

- mobility and movement
- communication
- everyday care eg dressing, washing etc
- depression
- swallowing
- nutrition
- cognition
- vision and perception
- continence
- relationships

Currently, only about half of patients who suffer a stroke receive appropriate rehabilitation in the first 6 months after discharge. Of the younger individuals, three-quarters want to return to work. A third of patients have problems with communication and a further third develop depression. Early, coordinated specialist rehabilitation reduces mortality and long-term disability, be it on a stroke unit or as part of an organised, early supported discharge scheme.
Disability is defined on the Modified Rankin Scale:

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No symptoms</td>
</tr>
<tr>
<td>1</td>
<td>No significant disability, though still has some symptoms – able to carry out all usual activities</td>
</tr>
<tr>
<td>2</td>
<td>Slight disability – unable to carry out all previous activities, but still self-caring</td>
</tr>
<tr>
<td>3</td>
<td>Moderate disability – needs help, but can walk without assistance</td>
</tr>
<tr>
<td>4</td>
<td>Moderately severe disability – needs help with walking and self-care</td>
</tr>
<tr>
<td>5</td>
<td>Severe disability – bedridden, incontinent and needs constant care</td>
</tr>
<tr>
<td>6</td>
<td>Dead</td>
</tr>
</tbody>
</table>

Table 4. Modified Rankin Scale (Rankin, 1957)

The final question that could be asked of the emergency physician concerns future driving. After TIA or stroke due to either ischaemia or intracerebral haemorrhage, patients must not drive for 1 month (DVLA, 2010). After this period, if there is no residual deficit, or only a mild deficit which does not affect limbs or visual fields, driving may be resumed without notifying the DVLA.

If a patient experiences multiple TIAs over a short period, they should not drive until 3 symptom-free months have passed.

Any seizures occurring within 24 hours of a stroke or TIA may be treated as provoked, so do not necessarily mean an automatic year-long ban. Each case is treated on an individual basis.