STROKE and transient ischaemic attacks are intrinsically linked and, although they warrant discussion as separate entities, they have a shared pathophysiological basis that includes them under the single umbrella of cerebrovascular disease.

Stroke is Australia’s second most common killer after coronary heart disease, and a leading cause of disability. In 2011, Australians will experience about 60,000 new and recurrent strokes, which equates to one stroke every 10 minutes. Strokes cost Australia an estimated $2.14 billion a year, underscoring the magnitude of the adverse public health impact and the need to improve outcomes after stroke.

Ischaemic stroke is defined as the sudden onset of neurological deficit as a result of ischaemia. Transient ischaemic attack (TIA) is defined as a transient neurological event that lasts from less than 24 hours to one that typically lasts less than one hour, and that is not associated with changes on neuro-imaging.

Within the first 12 months after an initial stroke, 8-12% of patients experience a second stroke, and patients who have TIA have a 5-7% risk of stroke in the following week. Clearly not only is optimal management of an index event essential, but it is also paramount to address the overall vascular risk to prevent recurrent strokes.

Transient ischaemic attack and stroke prevention

**Background**

**Transit from www.australiandoctor.com.au**

**Inside**

- Epidemiology, risk factors and aetiology
- Clinical features
- Investigations
- Treatment
- Secondary prevention after stroke or TIA

**The authors**

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- **PROFESSOR CHRIS LEVI**, director, acute stroke services, John Hunter Hospital; and director, centre for brain and mental health research, University of Newcastle and Hunter Medical Research Unit, Newcastle, NSW.
Epidemiology, risk factors and aetiology

STROKE is divided into ischaemic (comprising of athero-thrombotic and embolic), and haemorrhagic. About 85% of strokes are ischaemic, and about 15% are haemorrhagic. In this article we concentrate on the mechanisms underlying ischaemic stroke.

Mechanisms of brain ischaemia include:
- Thrombosis of an artery supplying the brain, related to in-situ processes within that artery.
- Focal occlusion of a cerebral artery.
- Global hypoperfusion caused by a systemic process such as shock.

It has become more useful to think about the vascular system as a single entity, where there are epidemiological data linking cerebral, coronary, and peripheral vessels, and the aetiologi- cal risk factors underlying pathology in them are similar (see above).

A recent publication reported the prevalence of common atherosclerotic risk factors in a cohort of patients with stroke or TIA. The mean age ± standard deviation was 73 ± 15 years. Of these:
- 15% had previously diagnosed diabetes; 10% were diagnosed with diabetes during admission.
- 62% had previously diagnosed hypertension; another 7% were diagnosed during admission.
- 88% of patients had dyslipidaemia.
- 35% had all three risk factors concurrently.

Clinically the division of strokes into age groups is useful aetologically. Strokes in young patients (often defined as those under 40) are associated with distinct aetiological factors, in particular, thrombophilic disorders and valvular heart disease. Young patients warrant more extensive investigation to exclude some of these less common conditions.

Clinical features

STROKE can be subclassified by the pathological process and the vascular distribution affected. Defining the overall pathological process is critical for making decisions regarding thrombolysis and inpatient therapy, and for prognosis.

Identifying the clinical features of a stroke and using the Oxfordshire Ischaemic Stroke Subtypes definitions (Table 1) may help identify the vascular distribution of the stroke. Unfortunately, interobserver agreement on the vascular territory is at best fair.

Thus, history and physical examination remain important in the clinical diagnosis of strokes. In a community-based study of diagnosis accuracy, clinical diagnosis by primary care physicians practicing in an emergency setting had a sensitivity of 92% for stroke and TIA.

Clinically several factors significantly increase the probability of a stroke being haemorrhagic, including:
- Coma.
- Neck stiffness.
- Seizures accompanying the neurological deficit.
- Diastolic blood pressure >110 mmHg.
- Vomiting.
- Headache.

However, intracerebral haemorrhage and ischaemic stroke can only be reliably differentiated using neuro-imaging, emphasising the need for early referral for non-contrast or advanced modality CT scan or perfusion-diffusion MRI if available (see Investigations, page 22).

The National Institute of Health Stroke Scale (NIHSS) is a commonly used classification of stroke severity at the time of presenta- tion. It provides a structured neuro-logical examination with diagnostic and prognostic value. This scale is particularly useful in front-line work at EDs and other pre-hospital settings.

There is no reliable way to determine if the abrupt onset of neuro-logical deficits represents reversible ischaemia without subsequent brain damage, or if the ischaemia will result in permanent damage to the brain (ie, a stroke). Therefore, patients presenting with reversible neurological symptoms suggestive of a TIA should be managed with the same sense of urgency as for an established stroke and should be referred urgently for appropriate investigations and management.

Indications for admission to hospital for patients with TIA follow the guidelines set out by The National Institute of Neurological Disorders and Stroke (NINDS) score. This is clearly modifiable for individual facilities and the availability of clinical and radio- logical support services.

In general practice it is useful to be aware of conditions that share signs and symptoms with stroke and that should be included in the differential diagnosis when assessing patients with a suspected stroke. Hypoglycaemia, migraines and seizures may present with some stroke-like features. A known history of cognitive impairment, non-neurological abnormal physical findings, and the decreased level of consciousness in patients with suspected stroke should suggest to the clinician that the presentation may not be due to stroke.

A diagnosis of TIA is paramount to decrease the chance of an established stroke developing. On diagnosis of a probable TIA, the ABCD2 score (see box right) is a simple scoring system that can be used to stratify patients who need urgent specialist assessment within 24 hours (ABCD2 score >4), and those who are suitable for outpatient assessment if tests can be performed within 48 hours (ABCD2 score <4). Patients not admitted for investigation should be reviewed in a TIA clinic if available. If not available, the investigations described in the next section should be performed.

People with crescendo TIA (two or more TIAS in a week) should be treated as being at high risk of stroke, even if their ABCD2 score = 3.

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People with crescendo TIA (two or more TIAS in a week) should be treated as being at high risk of stroke, even if their ABCD2 score = 3.

Patients who have had a TIA but who present late (more than one week after their last symptom has resolved), should be treated as though they are at lower risk of stroke.

<table>
<thead>
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<th>Table 1: Oxfordshire Ischaemic Stroke Subtypes</th>
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<td>Stroke subtype</td>
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<td>Total anterior circulation infarct (TACI)</td>
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<td>Lacunar infarct (LACI)</td>
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<td>Partial anterior circulation infarct (PACI)</td>
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<td>Posterior circulation infarct (POCI)</td>
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ABCD2 score

ABCD2 is a prognostic score used to identify people at high risk of stroke after a TIA. It is calculated based on:

A: Age (≥60 years = 1 point)
B: Blood pressure at presentation (≥140/90 mmHg = 1 point)
C: Clinical features:
- Unilateral weakness = 2 points
- Speech disturbance without weakness = 1 point
D: Duration of symptoms:
- ≥60 minutes = 2 points
- ≤59 minutes = 1 point
D: Diabetes = 1 point

Total scores range from 0 (lowest risk) to 7 (highest risk)
HOW TO TREAT

TRANSIENT ISCHAEMIC ATTACK AND STROKE PREVENTION

Investigations

DISTINGUISHING BLEEDING FROM ISCHAEMIA

- CT is considered sufficiently sensitive for detecting mass lesions, such as neoplasm or abscess, as well as for detecting acute haemorrhage.

Non-contrast CT

- Non-contrast CT images can be acquired rapidly with modern multi-detector scanners and at sub-millimetre image resolution. It is considered sufficiently sensitive for detecting mass lesions, such as a neoplasm or abscess, as well as for detecting acute haemorrhage. However, it may not be sensitive enough to detect an ischaemic stroke, especially if it is small, acute, or in the posterior fossa (ie, brainstem and cerebellar areas).

- Early features of ischaemia on CT include local cortical gyral swelling and sub-ependymal (sparing) secondary to cytotoxic oedema. Another early radiological sign is the hyperdense ‘middle cerebral artery sign’, in which a hyperintensity is present in the absence of bleeding and ischaemic stroke, especially if it is small, acute, or in the posterior fossa (ie, brainstem and cerebellar areas).

- Non-contrast CT after 72 hours. This is normal non-contrast CT on stroke in patients with a high pre-test probability of stroke. Therefore this may not apply when MRI is used in the broad range of unselected patients presenting with suspected acute stroke usually seen in routine clinical practice.

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- Non-contrast CT after 72 hours. This is normal non-contrast CT on stroke in patients with a high pre-test probability of stroke. Therefore this may not apply when MRI is used in the broad range of unselected patients presenting with suspected acute stroke usually seen in routine clinical practice.

- CT cannot be performed on people with certain types of medical devices (eg, pacemakers) or in those with claustrophobia or delirium, which in the scanner degrades the image resolution. The increased sensitivity of multimodal MRI sequences (TI- and T2-weighted and fluid attenuated inversion recovery) take the mean effective dose from a brain scan of 2.7mSv, while additional CT angiogram and CT perfusion scans take the mean effective dose to 13mSv — about five times the exposure of an unenhanced CT brain. However, in a single CT — even with prolonged exposure to radiation with the new modalities — this is unlikely to be of any clinical relevance. It is not currently an issue that concerns physicians, as CT of the brain involves relatively low radiation exposure.

- The volume (ie, size) of the lesions identified by diffusion weighted imaging and perfusion weighted imaging MRI may not only serve a diagnostic purpose but may also predict initial clinical severity, final infarct size and late clinical outcome in anterior-circulation stroke syndromes, thus serving prognostic purpose as well.

- Sometimes MRI shows white-matter hyperintensities. These have recently been associated with an increased risk of subsequent stroke, dementia and death.

- Not all patients with TIA need immediate brain imaging. Patients should be assessed by a specialist before a decision on imaging is made. Brain imaging after suspected TIA is recommended when:

  - The vascular territory affected is uncertain (anterior or posterior circulation) and the patient is being considered for carotid endarterectomy.
  - The pathology underlying the patient’s neurological symptoms is uncertain, eg, alternative diagnoses may include migraine, epilepsy or tumour.
  - Intracerebral haemorrhage needs to be excluded, for example, patients taking anticoagulants or with long duration of symptoms (eg, for subdural bleed symptoms can be for hours, days or weeks).

CT angiography

- Because many patients are undergoing non-contrast CT scanning during the early phase of diagnosis, there is a trend to use CT angiogra-
phly to document vascular pathology. CT angiography not only allows non-invasive assessment of the intracranial and extracranial circulation, but can also exclude dissection and arteriovenous malformations that may present with neurological signs and symptoms.

**Duplex Doppler scan**

Duplex Doppler examination of the carotid arteries should be carried out as soon as possible (figure 1). This is often limited by the availability of ultrasoundographic resources. The role of carotid endarterectomy and carotid stenting in the prevention of early stroke recurrence in patients with ipsilateral carotid artery stenosis has increased the urgency of this investigation. The results are given in the form of Duplex velocities in the different carotid territorial arteries and may allow differentiation of the infract core and the penumbra area (potentially salvageable parenchyma), leading to extension of the therapeutic window. Much of the literature addresses the use of IPA within three hours of stroke onset, as described in the NINDS trial. The 3rd European Cooperative Acute Stroke Study (ECASS III) showed that the extension of the treatment window to 4.5 hours is efficacious and safe in selected patients.

On the basis of an absolute increase in favourable outcomes of 10% between the IPA placebo and placebo groups in ECASS III, among patients who actually received treatment according to the protocol, the number needed to treat to produce one additional recovery outcome is only 10. The number needed to treat to achieve a measurable advantage in functional outcome score is probably only four.

The introduction of aspirin should probably be delayed for 24 hours after thrombolysis due to increased risk of haemorrhagic transformation of the stroke. It is important to note that although thrombolysis is an exciting development in the management of acute stroke, most patients will not receive acute reperfusion therapy because of delayed presentation or lack of local facilities and expertise, especially in regional and rural centres. Currently fewer than 10% of patients receive thrombolysis. Extension of the therapeutic window may allow for the development of models of management that would include air or road transport to facilities with thrombolysis capacity. This may resolve some of the challenges of offering equitable care to regional or rural communities where thrombolysis is not currently available.

**EGG**

An electrocardiogram is essential to rule out arrhythmia, especially atrial fibrillation, and other findings associated with established valvular heart disease or diagnosed with transient arterial disease.

**Echocardiogram**

If any evidence of cardiac disease is present, echocardiography will further delineate the pathology and exclude intracardiac thromboembolic sources. Echocardiography may also point to potential left-to-right shunts to explore the aortic arch for atheroclerotic plaques.

**Other tests**

Selected patients may require more intensive investigation, including lumbar puncture, EEG and toxicology screens, usually when an alternative diagnosis is suspected.

## Investigations in development

Current studies are focusing on the role of biomarkers in the diagnosis of stroke. Measurement of biomarkers related to the aetiology of cardio-embolic stroke, such as B-Natriuretic peptide and D-dimer proteins, may further advance our understanding of stroke. The future may also see the introduction of new biomarkers that may have predictive potential to detect or prevent recurrent stroke.

### Secondary prevention after stroke or TIA

#### CHADS2 VAS score for the use of warfarin

1. Congestive heart failure (score = 1)
2. Hypertension (score = 1)
3. Age >75 (score = 2)
4. Diabetes (score = 1)
5. Stroke and/or TIA (score = 2)
6. Vascular disease (score = 1)
7. Sex (female gender score = 0)

**Warfarin**

Warfarin remains the gold standard for therapy in patients who have AF and meet the risk–benefit criteria of the new CHADS2 VAS score. This is a comprehensive scoring tool based on the factors shown in the box above. A score of 1 or above recommends the use of aspirin or nothing, 1 the use of aspirin or warfarin, and a score >1 the use of warfarin for secondary prevention in patients with AF.

Many of the so-called low-risk patients may actually benefit from the use of vitamin K antagonist such as warfarin.

#### CHA2DS2-VASc score for the use of warfarin

1. Congestive heart failure (score = 1)
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6. Vascular disease (score = 1)
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**Warfarin**

Warfarin remains the gold standard for therapy in patients who have AF and meet the risk–benefit criteria of the new CHADS2 VAS score. This is a comprehensive scoring tool taking into account the factors shown in the box above. A score of 2 or above recommends the use of aspirin or nothing, 1 the use of aspirin or warfarin, and a score >1 the use of warfarin for secondary prevention in patients with AF.

The ACTIVE study recruited patients in AF at high risk of stroke who could not take warfarin. The addition of clopidogrel to aspirin reduced the risk of ischaemic stroke (absolute risk reduction 1.9%) but this benefit was counteracted by an increase in bleeding (absolute risk increase 1.3%).

### Clopidogrel

**ClotAD**

ClotAD is a powerful platelet inhibitor with a well-established role in coronary artery disease. In the setting of secondary prevention of stroke, the CAPRIE study showed a reduction of stroke in those treated with clopidogrel versus aspirin (number needed to treat 200 per annum). However, this beneficial effect was limited to those with peripheral vascular disease.

**MATCH Study**

The MATCH study showed that the addition of clopidogrel to aspirin reduced the risk of ischaemic stroke (absolute risk reduction 1.9%) but this benefit was counteracted by an increase in bleeding (absolute risk increase 1.3%). There was no benefit from the addition of clopidogrel to aspirin in the CHARISMA Study.

**ClotAD in stroke**

ClotAD is recommended for patients with aspirin intolerance or allergy, or for those who cannot tolerate clopidogrel.

### Cardiologist revisualisation

Symptomatic extracranial internal carotid artery stenosis poses a high short-term risk of ischaemic cerebral infarction of up to 20-50% in the first three months. Cardiologist revisualisation is an integral part of the interventional strategy in patients who meet the current criteria for carotid artery revascularisation. The criteria for intervention include symptomatic carotid stenosis of >70% and severe intermittent claudication (systolic velocity >210cm/s).

The procedures currently used in the UK are percutaneous transluminal angioplasty (PTCA) and/or stenting, which are performed under local anaesthesia. The procedure is usually carried out as an outpatient. A recent study has shown a delay in the diagnosis of stroke. Measurement of biomarkers related to the aetiology of cardio-embolic stroke, such as B-Natriuretic peptide and D-dimer proteins, may further advance our understanding of stroke. The future may also see the introduction of new biomarkers that may have predictive potential to detect or prevent recurrent stroke.
HOW TO TREAT

Transient ischaemic attack and stroke prevention

from previous page

available include surgical carotid endarterectomy (CEA) and percutaneous angioplasty and stenting. Studies have suggested the benefit of CEA or stenting diminishes with the time from the index event, emphasising the need for urgent and early vascular surgery if the patient meets the criteria for intervention.

There is an ongoing debate about the role of these two procedural alternatives. A recent systematic review concluded that carotid endarterectomy was superior to carotid artery stenting for short-term outcomes but the difference was not significant for intermediate-term outcomes. This difference in benefit was mainly for non-disabling stroke.

During the peri-procedural period, there was a higher risk of stroke with stenting and a higher risk of MI with endarterectomy.

The majority of patients with asymptomatic high-grade carotid stenosis (figure 6) has been a topic of active debate, with proponents on both sides. In 2009 the European Society of Vascular Surgery published guidelines recommending CEA in asymptomatic men under 75 with 70-99% stenosis, if the perioperative stroke and death rate is <3%. CEA should be considered in younger, fit patients.

Figure 6: CT angiogram showing carotid stenosis.

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Figure 6: CT angiogram showing carotid stenosis.

Cranial Doppler showing micro-emboli may help risk-stratify this cohort to a higher-risk group and thus more likely to benefit from CEA.

As medical therapy improves, the annual rate of stroke in medically treated patients may well drop to under 1% and the risk-benefit ratio for CEA may well change in favour of optimal medical therapy in asymptomatic patients.

Risk-factor management

Physicians should keep in mind that other common clinical interventions, including blood pressure control, lipid lowering, glucose control in diabetes, and smoking cessation, are each about three times more effective than aspirin in preventing future stroke.

The target-driven management of hypertension in patients after cerebrovascular events is the single most important treatable risk factor for recurrent stroke, with about 60% of strokes attributed to hypertension. A recent meta-analysis has shown an average decrease in blood pressure of 5.8 mmHg reduced the incidence of stroke by 42% over a 2-5 year follow-up.

BP targets for cerebrovascular disease have not been specifically defined, but it is not unreasonable to use established a BP target of ≤140/90 mmHg. The BP should be lowered gradually over several months to minimise side effects, including hypotensive symptoms due to failure of autoregulation of cerebral blood flow. The choice of antihypertensive agents should be individualised to the patient’s comorbidities, with diuretics, ACEIs, ARBs and calcium-channel blockers being favoured.

Smoking-cessation programs should be encouraged, as the patient’s health will significantly benefit beyond its effect on stroke prevention.

A meta-analysis has shown the benefit of statins in reducing the rates of stroke and cardiovascular events is independent of cholesterol lowering. Statins, given early benefits in trials of acute coronary syndrome, a crucial proportion is probably justified and may also increase the rates of long-term use.

Falls make up a significant number of complications after stroke. The only intervention shown to be effective in reducing falls is vitamin D for female stroke survivors in an institutional setting.

Ongoing support for patients

It is important to appreciate the toll that a stroke places not only on the patient’s wellbeing, but also on their family and carers. Studies have demonstrated that the stress places families at risk for developing depression, poor quality of life and health problems. They need support and links with community-based stroke support groups. Social workers or community health nursing facilities may need to be accessed to ensure appropriate and adequate management of the family and avoid carer burnout.

THIS case highlights the variability in management of patients who present to a regional or rural centre. A 62-year-old man was brought in to a GP’s surgery by his son. His son had a history of heavy cigarette smoking. His BP at the surgery was 158/86 mmHg, with a past history of atrial fibrillation. He was seen by the doctor on call an hour later. He was admitted to a high-dependency unit with an altered mental state. He was seen by the neurologist and was given intravenous Asasantin and rosuvastatin were offered. His neurological status showed some improvement, with power 3/5 and some word formation, but with persistent dysarthria.

Figure 6: CT angiogram showing carotid stenosis.

Fasting LDL-C level was 3.4 mmol/L, fasting glucose level was normal and BP remained moderately elevated. Despite careful introduction of thickened fluids and bolus-handling exercises he had a mild aspiration that was documented on chest X-ray and managed conservatively.

Telemetry monitoring did not reveal any evidence of arrhythmia. Duplex Doppler studies of the carotid arteries showed only minor stenosis (<50%) atherosclerotic disease bilaterally, and trans-thoracic echocardiogram showed left ventricular hypertrophy.

Asassinat and rosuvastatin were introduced and the patient was restarted on his background antihypertensives, which included an ACE inhibitor-diuretic fixed combination. This controlled his BP to ≤140/90 mmHg.

He was admitted to the stroke unit for rehabilitation by the multidisciplinary team led by the local rehabilitation physician.

At the time of discharge he had regained some function of his right arm and good improvement in his gait. His speech remained dysphasic, supportive community-based allied health interventions were started and he was referred to his GP for follow-up.

Authors’ case study

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References

Available on request from julian.mclaffin@redbusiness.com.au

Further reading

• Muntag M, et al. ‘We’re just sick people, nothing else…’: factors contributing to elderly stroke patients’ satisfaction with rehabilitation. Clinical Rehabilitation 2008; 22: 825-35

Revised by cljr 1/3/11

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1. Anderson`s Hints.pdf 1/2/11, 12:02 PM

HOW TO TREAT
Transient ischaemic attack and stroke prevention

Background

How to Treat Quiz

Transient ischaemic attack and stroke prevention — 4 February 2011

1. Which TWO statements are correct?
   a) Transient ischaemic attack (TIA) is defined as a transient neurological event that lasts less than 24 hours and is associated with changes on neuro-imaging
   b) Patients with TIA have a 5-7% risk of stroke in the following week
   c) Most strokes are ischaemic in origin
   d) Strokes in patients under 40 years are mostly due to cardiac atherothrombosis

2. Which TWO statements are correct?
   a) Dysphasia, dyscalculia and visual–spatial
   b) Ipsilateral cranial nerve palsy with d) Using currently available neuro-imaging, the characteristic of a partial anterior circulation infarct
   c) The sensitivity of CT to detect ischaemic symptoms is limited
   d) Carotid revascularisation should be considered in this setting.

3. Which TWO statements are correct?
   a) In patients with suspected stroke, prior cognitive impairment makes a diagnosis of stroke more likely
   b) If a TIA lasts for 90 minutes, subsequent stroke is more likely than if the TIA lasts for 60 minutes
   c) With people too far or TIA in a week are at high risk of stroke, even if other key risk factors are not present.
   d) Using currently available neuro-imaging, the absence of an identifiable infarct excludes ischaemic pathology

4. Which TWO statements are correct?
   a) CT scans may not detect an ischaemic stroke, especially if it is small, acute, or in the posterior fossa
   b) The sensitivity of CT to detect cerebral haemorrhage increases after the first week following the onset of symptoms
   c) The sensitivity of CT to detect ischaemic stroke decreases in the 48-72 hours after the onset of symptoms
   d) Using MRI or CT perfusion scanning, identification of a perfusion deficit is an important part of early stroke management

5. Which THREE statements are correct?
   a) MRI has documented that patients presenting with TIA actually have ischaemic infarcts in about 50% of cases
   b) Brain imaging after a suspected TIA is recommended if there is uncertainty regarding alternative diagnoses such as haemorrhage, migraine, tumour or epilepsy
   c) The combination of MRI and activated thromboplastin time are useful in patients with TIA and probable stroke
   d) Echocardiography can exclude intracardiac sites of thromboembolism in a patient with stroke

6. Which THREE statements are correct?
   a) Thrombolysis using tissue plasminogen activator (tPA) is given within 4.5 hours of the onset of an ischaemic stroke
   b) Thrombolysis has decreased mortality from stroke
   c) Thrombolysis improves the time to recovery and the functional outcomes for ischaemic stroke patients
   d) Good outcomes for stroke patients are independent of early admission to a stroke unit

7. Which TWO statements are correct?
   a) Prevention of aspirin by adequate swallowing assessments and feeding protocols is an important part of early stroke management
   b) Aspirin as secondary prevention following stroke or stroke does not decrease morbidity or mortality due to ischaemic stroke
   c) Full-dose dipyridamole may be poorly tolerated
   d) Low-dose dipyridamole is as effective as full-dose treatment

INSTRUCTIONS

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points. We no longer accept quizzes by post or fax. The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

ONLINE ONLY

CDP QUIZ UPDATE

The RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2011-13 triennium. You can complete this online along with the quiz at www.australiandoctor.com.au. As this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

NEXT WEEK

The next How to Treat aims to shed some light on the classification, aetiology, and clinical behaviour of cardiomyopathy, the diagnostic techniques currently used for investigation, and latest therapeutic options available. The author is Dr Giovanna Zingarelli, staff specialist cardiologist, Gosford Hospital, and (conjoint) senior lecturer, University of Newcastle, NSW.