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Posterior circulation ischaemic stroke

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Cite this as: *BMJ* 2014;348:g3175
doi: 10.1136/bmj.g3175

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About 20-25% (range 17-40%) of the 150 000 ischaemic strokes in the United Kingdom each year affect posterior circulation brain structures (including the brainstem, cerebellum, midbrain, thalamuses, and areas of temporal and occipital cortex), which are supplied by the vertebrobasilar arterial system.[1] Early recognition of posterior circulation stroke or transient ischaemic attack (TIA) may prevent disability and save lives, but it remains more difficult to recognise and treat effectively than other stroke types. Delayed or incorrect diagnosis may have devastating consequences, including potentially preventable death or severe disability, if acute treatment or secondary prevention is delayed.[2] The annual adjusted incidence of posterior circulation infarction was estimated at 18 per 100 000 person years (95% confidence interval 10/100 000 to 26/100 000) in an Australian study.[3] Preceding posterior circulation TIA or other transient brainstem symptoms, particularly if recurrent, signal a high risk of impending ischaemic stroke and should prompt specialist urgent referral for further management.[4] New acute treatment options and stroke prevention strategies specific to the posterior circulation are important areas of active research.

This review aims to demonstrate the importance and challenges of recognising and treating posterior circulation stroke, including the key differences between posterior and anterior circulation stroke.

What is posterior circulation ischaemic stroke?

Posterior circulation ischaemic stroke is a clinical syndrome associated with ischaemia related to stenosis, in situ thrombosis, or embolic occlusion of the posterior circulation arteries—the vertebral arteries in the neck, the intracranial vertebral, basilar, and posterior cerebral arteries, and their branches (fig 1). Common sites of occlusion cause characteristic clinical patterns and syndromes (figs 1 and 2).

SOURCES AND SELECTION CRITERIA

We searched PubMed up to November 2013 with the terms "posterior circulation," "stroke," "ischaemic," and "vertebrobasilar," targeting full text English language studies published since 1990. We also searched the reference lists of the identified articles and our own files. Only papers published in English, or with an English abstract, were reviewed. The final selection of references was based on our judgment of relevance to the topic of this review.

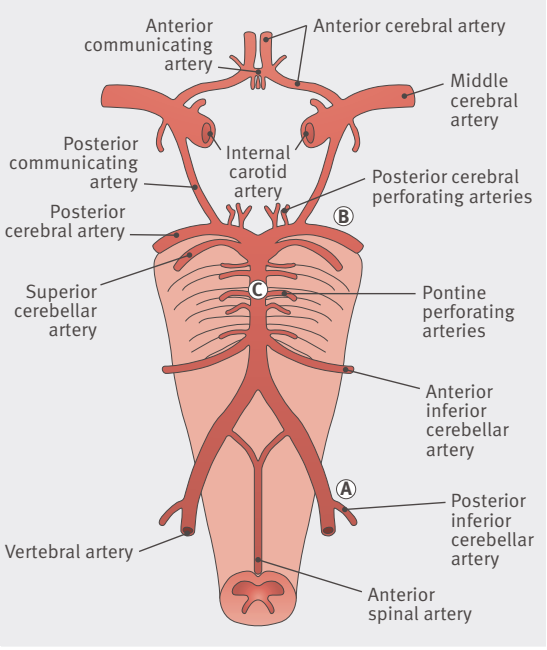

Fig 1 | Anatomy of the vertebral and basilar arterial circulation and circle of Willis. (A) Site of posterior inferior cerebellar artery occlusion; (B) site of posterior cerebral artery occlusion; (C) site of pontine perforating artery occlusion

SUMMARY POINTS

- Posterior circulation stroke accounts for 20-25% (range 17-40%) of ischaemic strokes
- Posterior circulation transient ischaemic attacks may include brief or minor brainstem symptoms and are more difficult to diagnose than anterior circulation ischaemia
- Specialist assessment and administration of intravenous tissue plasminogen activator are delayed in posterior circulation stroke compared with anterior circulation stroke
- The risk of recurrent stroke after posterior circulation stroke is at least as high as for anterior circulation stroke, and vertebrobasilar stenosis increases the risk threefold
- Acute neurosurgical input may be needed in patients with hydrocephalus or raised intracranial pressure
- Basilar occlusion is associated with high mortality or severe disability, especially if blood flow is not restored in the vessel; if symptoms such as acute coma, dysarthria, dysphagia, quadriparesis, pupillary and oculomotor abnormalities are detected, urgently seek the input of a stroke specialist

There are important differences between posterior and anterior circulation stroke. The differences include the value of screening instruments, optimum diagnostic modalities, and clinical features (table).[5-9] The face arm speech test (FAST), a widely used prehospital stroke recognition screening instrument, is less sensitive for detecting posterior circulation stroke than for anterior circulation stroke (the carotid territory, including the anterior and middle cerebral arteries and their branches).[5] It can be difficult to determine the vascular territory of an acute ischaemic clinical syndrome on purely clinical grounds, but this knowledge may be needed to determine the most appropriate acute treatment and prevention strategy.[10] However, computed tomography (CT), the standard brain imaging modality in hyperacute stroke, has limited sensitivity in posterior circulation stroke.

Although in the past posterior circulation ischaemia was considered to have a lower recurrence risk than anterior circulation ischaemia, current data suggest that the risk is at least as high, if not higher.[1]

What causes posterior circulation stroke?

The most common causes of posterior circulation stroke are occlusion or embolism from large artery vertebrobasilar atherosclerosis or dissection, and embolism from the heart.[11 12] In a large US hospital registry study of 407 patients with posterior circulation stroke, embolism was the most common mechanism (40% of patients); large artery occlusive lesions caused haemodynamic brain ischaemia in 32%; and the remainder of strokes were attributed to in situ small vessel occlusion, other identified mechanisms, or unknown causes.[12] Of the strokes attributed to embolism 24% had cardiac source, 14% were caused by to artery-to-artery embolism, and 2% had multiple sources of potential embolism.[12] Recent population based and hospital observational studies have shown a threefold increased risk of stroke after posterior circulation TIA or minor stroke in patients with symptomatic vertebrobasilar stenosis than in those without stenosis.[6 13 14] Dissection of the extracranial vertebral artery is also an important cause of stroke, especially in young patients; it may be painless and usually occurs without a clear history of trauma. In a systematic review of vertebral artery dissection the most common symptoms were dizziness or vertigo (58%), headache (51%), and neck pain (46%). The annual incidence of spontaneous vertebral artery dissection is estimated at 1-1.5 per 100 000 per year.[15] Less common causes include vasculitis and dolichoectasia (elongation and tortuosity) of the vertebral and basilar arteries. In younger people, dolichoectasia may be a clue to Fabry's disease, a rare X linked inherited multisystem lysosomal storage disorder.[16]

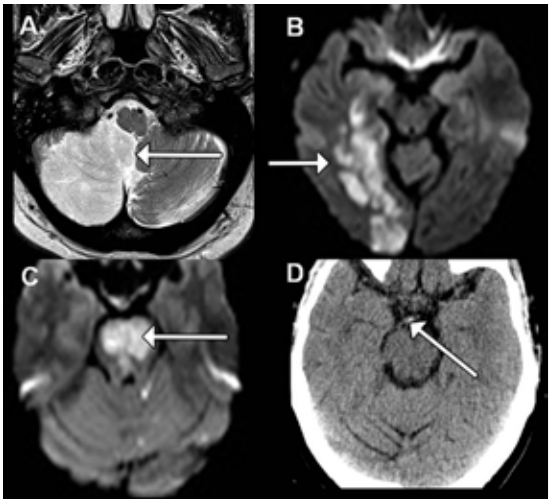

Fig 2 | Imaging findings associated with the sites of occlusion shown in fig 1. (A) Full right posterior inferior cerebellar artery territory infarct (arrow) shown on T2 weighted magnetic resonance imaging (MRI); (B) acute right posterior cerebral artery territory infarct (arrow) shown on diffusion weighted MRI; (C) acute bilateral pontine infarction (arrow) as a result of acute basilar occlusion shown on diffusion weighted MRI; (D) axial computed tomography scan showing bright (hyperdense) region (arrow) consistent with an acute basilar thrombus

Comparison of anterior and posterior circulation ischaemic stroke[5-9]

Aspects	Anterior circulation (carotid territory)	Posterior circulation (vertebrobasilar territory)
Clinical recognition tools		
Prehospital triage tools and scores, such as FAST*	High sensitivity: >90%	Moderate sensitivity: ~60%
Imaging		
Computed tomography	Moderate sensitivity	Poor sensitivity
Magnetic resonance imaging	Very good to excellent sensitivity (>95%)	Very good sensitivity (>80%)
Clinical features†		
Isolated hemianopia	+	++
Quadrantanopia	–	+
Pupil abnormalities	+ (Horner's syndrome)	+++ (may be bilateral)
Diplopia	–	+++
Focal (unilateral) sensorimotor	+++	++
Bilateral sensorimotor	–	+++
Unsteadiness/ataxia	+	++
Vertigo	±	+++
Dysarthria	++	++
Dysphasia	+++	+ (thalamic infarcts)
Coma	+	+++
	Coma unusual, unless there is mass effect and raised intracranial pressure (for example, as a result of large middle cerebral artery stroke); rare as an initial hyperacute presenting symptom; somnolence may occur	Coma well recognised in thalamic and brainstem ischaemia and may be an acute presenting symptom
Acute management		
Intravenous tissue plasminogen activator time window (h)	4.5	4.5 (but used up to 24 in basilar occlusion)
Endovascular treatment	Benefit not proved	Benefit not proved but often considered for basilar occlusion, especially if it has not responded to intravenous treatment
Neurosurgical intervention	Hemicraniectomy indicated for malignant middle cerebral artery syndrome	External ventricular drainage or posterior decompression indicated for hydrocephalus in acute infarction with mass effect
Stroke risk in symptomatic large vessel disease by 90 days after transient ischaemic attack or stroke	18%	Almost 25%

*FAST=face arm speech test.
†Scores represent the estimated relative likelihood of each symptom being present in anterior and posterior circulation ischaemic stroke.

Box 1 | Common symptoms seen in posterior circulation ischaemia

- Motor deficits (weakness, clumsiness, or paralysis of any combination of arms and legs, up to quadriplegia, sometimes changing from one side to another in different attacks)[17]
- "Crossed" syndromes, consisting of ipsilateral cranial nerve dysfunction and contralateral long motor or sensory tract dysfunction are highly characteristic of posterior circulation stroke[18]
- Sensory deficits (numbness, including loss of sensation or paraesthesia in any combination of extremities, sometimes including all four limbs or both sides of the face or mouth)
- Homonymous hemianopia—a visual field defect affecting either the two right or the two left halves of the visual fields of both eyes
- Ataxia, imbalance, unsteadiness, or disequilibrium
- Vertigo, with or without nausea and vomiting
- Diplopia as a result of ophthalmoplegia
- Dysphagia or dysarthria
- Isolated reduced level of consciousness is not a typical stroke symptom but can result from bilateral thalamic or brainstem ischaemia (especially from rostral basilar artery occlusion)

Similar to other forms of cerebrovascular and cardiovascular disease, the risk factors for posterior circulation strokes include hypertension, smoking, hypercholesterolaemia, atrial fibrillation, and coronary artery disease.

What are the clinical symptoms and signs of posterior circulation ischaemia?

Posterior circulation ischaemia can be challenging to recognise, particularly in patients with a TIA, which may have resolved by the time of presentation. However, there are some characteristic clinical patterns (box 1).

Because the posterior circulation supplies the brainstem, cerebellum, and occipital cortex, symptoms often include dizziness, diplopia, dysarthria, dysphagia, disequilibrium, ataxia, and visual field deficits. Acute onset "crossed" deficits—cranial nerve territory symptoms on one side and sensory or motor deficits of the opposite arm and leg—are virtually diagnostic of posterior circulation ischaemia.[7] In a large single centre observational study of 407 patients, the most common posterior circulation symptoms were dizziness (47%), unilateral limb weakness (41%), dysarthria (31%), headache (28%), and nausea or vomiting (27%). The most common signs were unilateral limb weakness (38%), gait ataxia (31%), unilateral limb ataxia (30%), dysarthria (28%), and nystagmus (24%).[18]

In practice it can be difficult to distinguish between posterior and anterior circulation stroke because some common syndromes (such as hemiparesis) are not specific for one or the other (table).[7 10]

Vertigo (a feeling of true movement relative to the environment) and "dizziness" are common symptoms in general practice and the emergency room and present a particular challenge.[19] It is crucial to elicit exactly what a patient means by dizziness (true feeling of rotation, dissociation between the patient and the environment, or presyncopal symptoms). Urgently refer all patients with acute vertigo and any other focal neurological symptoms for specialist assessment.

Which other disorders can mimic posterior circulation ischaemic stroke?

Acute peripheral vestibular dysfunction can mimic stroke in general practice or the emergency department. It typically causes isolated vertigo with no other brainstem symptoms or signs and is more common than stroke. The head impulse or Dix-Hallpike tests may help in the diagnosis of peripheral vestibular disturbance.[19] Acute intracranial haemorrhage, subarachnoid haemorrhage, and tumour can mimic ischaemic stroke, further highlighting the importance of prompt imaging. Basilar migraine, which may have aura features including vertigo and diplopia, as well as severe occipital headache, can resemble acute stroke, and should always be excluded, especially if it is the patient's first presentation.[21]

Toxic or metabolic disturbances may initially present with features resembling cerebrovascular disease. These include drugs of misuse or prescribed drugs (such as anticonvulsants), hypoglycaemia, central pontine myelinolysis, and post-infectious disorders, such as antibody associated disorders (for example, Miller Fisher syndrome, which causes ophthalmoplegia, ataxia, and areflexia).[21]

Posterior reversible encephalopathy syndrome can cause posterior circulation ischaemia, which results in visual disturbance, seizures, and other focal symptoms. This syndrome has a predilection for the posterior circulation and is usually associated with hypertension.

Which clinical syndromes are caused by posterior circulation stroke?

Although ischaemia can occur anywhere in the vertebrobasilar territory, a large registry study from the United States suggested that infarcts most often include the distal territory (rostral brainstem, superior cerebellum, occipital and temporal lobes).[12] Several posterior circulation clinical syndromes are highly localising and are important for all doctors who look after acute stroke patients to recognise (fig 2; box 2, see bmj.com).

How is posterior circulation ischaemic stroke diagnosed?

The diagnosis of posterior circulation ischaemic stroke is based on rapidly developing clinical signs of focal (or occasionally global) disturbance of cerebral function, with no apparent cause other than that of vascular origin.[24]

An index of suspicion for posterior circulation stroke should be maintained in patients presenting with acute neurological symptoms. In the initial assessment phase it is important to establish the onset and tempo of symptoms and establish whether the patient has experienced typical or characteristic posterior circulation stroke symptoms such as acute diplopia, visual field disturbance, or swallowing difficulties.

Diagnostic tools such as the recognition of stroke in the emergency room (ROSIER) scale may help medical staff in the emergency department rapidly recognise acute stroke because this tool includes assessment of visual fields.[25]

Posterior circulation stroke is diagnosed on the basis of history and clinical examination, assisted by imaging. Assessment by a specialist stroke team with admission to a stroke unit is the optimum approach. Assessment in the emergency department for homonymous visual field deficits; eye movement abnormalities (including simple labyrinthine

TIPS FOR NON-SPECIALISTS

Careful history taking is needed to identify patients with posterior circulation stroke, who may present with recurrent, stuttering, or progressive symptoms, which may include altered level of awareness (not a typical stroke symptom but seen in bilateral thalamic ischaemia)

Clinical signs that may help identify a posterior circulation stroke include the presence of homonymous visual field deficits, eye movement abnormalities, Horner's syndrome, or gait ataxia

Previously ambulant patients with acute focal neurological symptoms leading to acute loss of balance should never be discharged without ensuring they can walk if stroke is a possible explanation. Always consider a posterior circulation stroke if a patient is uncharacteristically disabled for the amount of alcohol reportedly consumed

Investigate posterior circulation transient ischaemic attack symptoms urgently to avoid preventable disability or death. Use rapid access transient ischaemic attack services or stroke specialist assessment if available, and use magnetic resonance imaging in the acute phase, especially if the diagnosis is unclear, because this modality has high sensitivity for identifying ischaemic lesions

Consider transferring patients at risk of deterioration in the acute phase of posterior circulation ischaemic stroke to a neuroscience centre because they may need urgent neurosurgery for mass effect or hydrocephalus

tests such as the head thrust/impulse test (http://content.lib.utah.edu/cdm/singleitem/collection/ehsl-dent/id/6)); and looking for Horner's syndrome (ptosis, small pupil (miosis), and anhydrosis on the same side), bilateral small or fixed pupils, and ataxia may aid early diagnosis.[18 19]

All cases of suspected stroke require urgent brain imaging with CT or magnetic resonance imaging (MRI) to exclude haemorrhage. If a patient is a candidate for thrombolysis therapy, brain and vessel imaging with a technique such as CT angiography is essential to identify basilar artery occlusion. It should be performed without delay, because minimising the time between stroke onset and the start of thrombolysis is associated with a good outcome.

Current international guidelines recommend MRI for assessing TIA, including those in the posterior circulation. It can help diagnose disorders that mimic stroke and TIA, can help verify vascular territory, and diffusion weighted imaging abnormalities independently predict early stroke risk after TIA.[29 30] MRI provides the greatest diagnostic yield when performed as soon as possible (certainly within a few days) of symptom onset, especially in minor stroke or TIA.[31] To help differentiate stroke from rare mimic disorders, such as encephalitis, further investigation with lumbar puncture (if no clinical or radiological contraindications are present) may be necessary if fever or atypical imaging features are identified.

How is posterior circulation stroke managed?

Similar to other stroke and acute neurological emergencies, stabilisation and resuscitation of patients with acute phase posterior circulation stroke are crucial. Careful assessment of airway, breathing, and circulation is also crucial before transfer in patients who may be at risk of deterioration during inter-hospital transportation, with input from an anaesthetics team if indicated.

Thrombolysis

The large ECASS3 randomised controlled trial found that intravenous tissue-type plasminogen activator (tPA) may be used in patients with posterior circulation stroke who meet the eligibility criteria, within 4.5 hours of symptom onset.[32] Results from randomised controlled trials in ischaemic stroke showed that intravenous alteplase (recombinant tPA) improves functional outcome using the modified Rankin score (a functional outcome score) at three months. Unfortunately, specialist assessment and intravenous administration of tPA are slower in patients with posterior circulation stroke compared with those with anterior circulation stroke, probably because of delayed or missed diagnosis.[33 34] Case series have shown prolonged door to needle time in patients with posterior circulation stroke; one observational study of 237 patients showed a mean time of 156.2 min (standard deviation 23.2) in the posterior circulation group versus 141.1 min (30.7) in the anterior circulation group; P=0.01.[33] Another study showed a prolonged door to needle time, but no prolonged stroke specialist to needle time.[34]

As with anterior circulation events, the administration of tPA in the posterior circulation carries a risk of haemorrhage, anaphylaxis, or angio-oedema. On the basis of its licence for use, contraindications to tPA include any intracerebral haemorrhage, known or suspected central nervous system lesion with high likelihood of haemorrhage after tPA (such as brain tumour, abscess, vascular malformation, aneurysm, contusion, or endocarditis), and clinical presentation suggestive of subarachnoid haemorrhage even with normal CT results. Other contraindications are uncontrolled hypertension (systolic blood pressure >180 mm Hg or diastolic blood pressure >110 mm Hg at start of tPA treatment), history of intracranial haemorrhage, active internal bleeding, fracture, acute trauma, stroke, serious head trauma, intracranial or intraspinal surgery in past three months, or bleeding disorder.[32]

Acute endovascular therapy

Acute endovascular therapy (intra-arterial clot removal or lysis) has been used in acute basilar occlusion because of the high likelihood of death or severe disability in the absence of recanalisation.[35-37] Evidence from a systematic review of published case series reporting the outcome of basilar artery occlusion after intravenous or intra-arterial thrombolysis showed that only 2% of 420 patients had a good outcome in the absence of basilar artery recanalisation.[38] However, intra-arterial therapy has not been proved to be of benefit. In a large international registry study of 592 patients with basilar occlusion, no significant difference was detected in outcome, as defined by modified Rankin score at one month after intravenous versus intra-arterial therapy.[35] Data from the Basilar Artery International Cooperation Study BASIC registry found similar outcomes in 347 patients with a severe deficit (coma, locked-in syndrome, or tetraplegia) when treated with intra-arterial thrombolysis or intravenous thrombolysis (relative risk 1.06, 95% confidence interval 0.91 to 1.22).[35] An ongoing randomised controlled trial is investigating the value of early intra-arterial therapy in basilar occlusion (www.basicstrial.com).

The time window for treatment for basilar occlusion may be longer than for other stroke types, and although treatment within 4.5 hours is desirable, it may be reasonable to consider treatment (intravenous or endovascular) up to 24 hours from onset.[39] The usefulness of emergency angioplasty

ADDITIONAL EDUCATIONAL RESOURCES

Resources for healthcare professionals

European Stroke Organisation (www.eso-stroke.org)
—Useful source of European stroke guidelines (free, registration not required)

Virtual Stroke University (www.stroke-university.com)
—Useful source of expert lectures on all aspects of stroke (free, registration not required)

Internet Stroke Centre (www.strokecenter.org/professionals)
—Independent source of educational and clinical trial information (free, registration not required)

Royal College of Physicians (www.rcplondon.ac.uk/resources/stroke-guidelines)
—UK national clinical guidelines for stroke (free, registration not needed)

American Heart Association (http://stroke.ahajournals.org/content/44/3/870)
—Guidelines for the early management of patients with acute ischemic stroke (free, registration not needed)

Resources for patients

Stroke Association (www.stroke.org.uk)
—Important source of information about stroke for patients and carers; provides an online patient forum

National Institute for Health Research Stroke (www.crn.nihr.ac.uk/focus_on/stroke)
—Research information

UK Stroke Forum (www.ukstrokeforum.org)
—A coalition of more than 30 organisations committed to improving stroke care in the UK

Bauby J-D. The diving bell and the butterfly: a memoir of life in death. Random House, 1997.
Autobiographical short book written by a journalist using his eye movements to communicate his account of life after he had a pontine stroke that caused quadriparesis, leaving him "locked in"

ARNI Institute (www.arni.uk.com/)
—Focuses on functional rehabilitation and exercise training after stroke

Different Strokes (www.differentstrokes.co.uk)
—A charity specifically for young patients with stroke

or stenting of the extracranial vertebral arteries in unselected patients is not yet well established.[39]

Neurosurgery

Neurosurgical intervention (including external ventricular drainage or decompression) may be lifesaving in large volume cerebellar infarction with falling level of consciousness attributable to raised intracranial pressure or acute hydrocephalus.[41-43] A large infarction of the cerebellum is often followed by delayed swelling. Although the early symptoms may be limited to impaired function of the cerebellum, oedema can cause brain stem compression and can rapidly progress to loss of brain stem function. Emergency posterior fossa decompression with partial removal of the infarcted tissue may be lifesaving. However, data come from case series—evidence from randomised controlled trials is lacking. In a case series of 52 patients with space occupying cerebellar infarction defined by computed tomographic criteria, 39 patients developed signs of brain stem compression and 41 developed a disturbance of consciousness.[42] Twenty one of the patients with decompressive surgery who were in an advanced clinical state (stuporous or comatose with posturing and cardiovascular or respiratory instability) before surgery were reported to recover well, compared with none of the patients who did not have surgery.[42]

Which investigations are needed after treatment of posterior circulation stroke?

Identification of the underlying mechanism or risk factors is an important aspect of stroke prevention because it has implications for optimum preventive treatment—such as anticoagulation for atrial fibrillation. Therefore, general and cardiac investigations recommended by international stroke guidelines should be carried out to help identify modifiable risk factors and guide secondary prevention strategies. Tests include electrocardiography, renal and liver function tests, full blood count including platelets, and the measurement of glucose, lipids, serum electrolytes, prothrombin time, international normalised ratio, and activated partial thromboplastin time.[29 43 44]

Some stroke patterns, specifically isolated posterior cerebral artery infarction and top of the basilar syndrome, are often associated with cardioembolism—more than 40% of posterior cerebral artery infarcts were attributed to a cardioembolic mechanism in the New England registry. These clinical syndromes therefore merit detailed assessment for a cardioembolic source including atrial fibrillation through cardiac rhythm monitoring.[12] Prolonged monitoring (with a prolonged ambulatory tape or an implantable device) increases the rate of detecting atrial fibrillation in patients in whom no other stroke mechanism or risk factor is identified but a cardioembolic source is suspected.[45] Echocardiography is recommended in selected patients, such as those with evidence of cardiac disease or suspected cardiac, aortic, or paradoxical embolism.[29 44] Further specialist serum investigations for systemic disease that predisposes to arterial thrombosis may be needed in patients in whom no clear cause is identified.[43 44]

What treatment is recommended after posterior circulation stroke?

Current international guidelines recommend secondary prevention with lifestyle modification and drugs, including antiplatelet agents, lipid lowering drugs, and blood pressure control to a target of less than 80 mm Hg/140 mm Hg.[29 44 46] Antiplatelet agents should be started once haemorrhage has been excluded and 24 hours has elapsed in patients who have received thrombolysis. Clopidogrel alone (or aspirin and dipyridamole) is recommended for long term secondary prevention of thromboembolic events.[44] In patients with indications for anticoagulation (such as atrial fibrillation), treatment should be started when the potential benefit outweighs the risk of harm by haemorrhagic transformation of the infarct generally about two weeks after an acute ischaemic stroke.[44] In patients at high risk of ischaemic stroke, such as those with symptomatic vertebrobasilar stenosis, dual antiplatelet treatment should be considered. A recent randomised trial in 5170 Chinese patients found that short term use of clopidogrel and aspirin when given within 24 h of minor stroke in any territory or onset of high risk TIA reduced the risk of recurrent stroke.[47]

The SPARCL randomised controlled trial included all subtypes of ischaemic stroke and showed that atorvastatin 80 mg per day reduced non-fatal or fatal stroke after stroke and TIA; however, there is a paucity of randomised controlled trial data for the hyperacute phase in the hours and days after TIA or stroke.[9 48]

Antihypertensive treatment and targets should be in accordance with guidelines for comorbid diseases such as diabetes. Evidence to support the use of antihypertensives in patients with stroke comes from the PROGRESS study,

QUESTIONS FOR FUTURE RESEARCH

What are the optimal secondary prevention strategies for posterior circulation stroke, including pharmacological treatments, or endovascular approaches for symptomatic vertebrobasilar stenosis?

Does acute endovascular therapy have a role in basilar artery occlusion?

Should a longer time window be used for thrombolysis in the posterior circulation?

a randomised controlled trial of a perindopril based regimen in 6105 patients (including those with and without hypertension) with previous stroke or TIA, which showed a 28% relative risk reduction for stroke.[46]

Which patients are at highest risk of deterioration or recurrence after posterior circulation minor stroke or TIA?

Patients with basilar occlusion may have a stuttering onset, with fluctuating or resolving symptoms that initially present as TIA but progress after vessel occlusion to devastating brainstem stroke.[21] Between 55% and 63% of patients with basilar artery occlusion have prodromal TIAs, minor strokes, or other symptoms, which are more common with atherosclerotic than embolic occlusions.[22] Patients with acute basilar artery occlusion have high mortality rates of 41-95% in natural history studies or studies of intravenous thrombolysis, with mortality rates being highest when there is no recanalisation.[22 32] In survivors with poor recanalisation, severe disability (for example, the locked-in syndrome) is common. In a single centre case series of 50 consecutive patients with angiographically confirmed basilar artery occlusion treated with intravenous thrombolysis, none of those with failed recanalisation who survived were living independently at three months.[49] For patients with symptomatic vertebrobasilar stenosis, the risk of recurrent stroke is almost 25% in the first 90 days.[6]

It is therefore crucial to be able to identify which patients are at highest risk of early recurrent stroke, both for triage purposes and for optimum management. If ongoing studies of vertebrobasilar stenosis demonstrate the efficacy of endovascular treatment, identification of high risk patients may be increasingly necessary.[50 51] Unfortunately, no specific dedicated prediction rule for identifying patients at highest risk of stroke after posterior circulation TIA or minor stroke currently exists. The ABCD2 clinical prediction score (Age, Blood pressure, Clinical symptoms (such as speech disturbance or weakness), Duration of symptoms and Diabetes) for use in TIA has not been specifically validated in vertebrobasilar territory TIA. However, one observational hospital based series showed that 30% of patients with recurrent posterior circulation events within the first 90 days after stroke or TIA were not identified as being high risk using the ABCD2 score.[5 52 53]

Registry data from New England in the US have shown an overall 30 day mortality of 3.6% in posterior circulation stroke, with embolic mechanism, distal territory location, and basilar artery occlusive disease carrying the worst prognosis.[12]

What is on the horizon for posterior circulation stroke?

Outstanding research questions remain regarding acute phase management, secondary prevention, and risk prediction.

Treatment time windows in acute basilar occlusion and different treatment strategies should be tested against each other in randomised trials. The BASICS trial—a randomised controlled multicentre open label phase III intervention trial with blinded outcome assessment, investigating the efficacy and safety of additional intra-arterial treatment (within six hours of symptom onset) after intravenous thrombolysis in patients with basilar artery occlusion—is currently recruiting patients.

The management of posterior circulation large artery disease in patients with vertebrobasilar stenosis, especially among patients with TIA and minor stroke, is an area of active research and interest. Current studies include a multicentre randomised controlled open prospective clinical trial of vertebral artery stenting versus best medical treatment.[50 51]

Thanks to Annemarie O'Flynn (general physician) for her comments on the review article.

Contributors: Both authors helped plan and write the article and in the literature search. DW is also responsible for the illustrations and is guarantor.

Competing interests: ÁM reports salary funding from the Health Service Executive (HSE) Ireland/Dr Richard Steeven's scholarship, and has received unrestricted educational grants towards travel and accommodation costs for attendance at conferences from Boehringer Ingelheim, Pfizer Healthcare, Lundbeck, and Biogen Idec. DW reports educational grants from Allergan; he also receives research support from the Stroke Association and the British Heart Foundation. Part of this work was undertaken at UCLH/UCL which received a proportion of funding from the Department of Health's NIHR Biomedical Research Centres funding scheme.

Provenance and peer review: Commissioned; externally peer reviewed.

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ANSWERS TO ENDGAMES, p 40

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ANATOMY QUIZ

Anteroposterior left vertebral angiogram

A: Basilar artery
B: Left posterior cerebral artery
C: Left superior cerebellar artery
D: Left anterior inferior cerebellar artery
E: Left posterior inferior cerebellar artery
F: Left vertebral artery

STATISTICAL QUESTION

Clinical trials: units of randomisation

The episode of acute asthma (answer *b*) was the unit of randomisation.

PICTURE QUIZ

A student with macrocytic anaemia

1 A hypersegmented neutrophil with a red cell fragment, red cell anisopoikilocytosis, and a megaloblastic nucleated red blood cell.
2 In view of her diet and short history, folate or vitamin B_{12} deficiency, with folate deficiency being the most likely.
3 Give intramuscular vitamin B_{12}, oral folic acid, and oral iron (to stop depletion of stores) and monitor potassium and reticulocytes. Transfusion should be avoided if possible.
4 Dietary deficiency, malabsorption (such as coeliac disease, inflammatory bowel disease, and pernicious anaemia), and drugs (such as methotrexate).
5 Vitamin B_{12} or folate deficiency, hypothyroidism, liver disease, alcoholism, drugs that cause folate deficiency (for example, methotrexate), haemolysis, and bone marrow disorders (for example, myelodysplasia).