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1148 • STROKE MEDICINE Clinical examination in stroke disease Blood pressure and cardiac auscultation Higher cerebral function Speech and language Attention and neglect Abbreviated mental test Cranial nerve function Neck stiffness/pain Visual fields Nerve palsy, e.g. 3rd, 6th, 7th or 12th General appearance Conscious level Posture: leaning to one side? Facial symmetry

Gait Able to weight-bear? Ataxic Hemiparetic gait pattern Sensory system Touch sensation Cortical sensory function: sensory inattention or neglect Joint position sense

Motor system Muscle bulk Abnormal posture or movements Tone Strength, including pronator drift Co-ordination Tendon reflexes Plantar reflexes

Hemiparetic posture Left pronator drift Visual field defect Mitral stenosis Extensor plantar reflex Fanning of toes Up Atrial fibrillation Left facial (7th nerve) palsy Pulse Rate and rhythm Loud Loud OS A2P2 MDM

Clinical examination in stroke disease • 1149

Rapid assessment of suspected stroke Rosier scale Can be used by emergency staff to indicate probability of a stroke in acute presentations: Unilateral facial weakness +1 Loss of consciousness −1 Unilateral grip weakness +1 Seizure −1 Unilateral arm weakness +1 Unilateral leg weakness +1 Speech loss +1 Visual field defect +1 Total (−2 to +6); score of > 0 indicates stroke is possible cause Exclusion of hypoglycaemia • Bedside blood glucose testing with BMstix Language deficit • History and examination may indicate a language deficit • Check comprehension (‘lift your arms, close your eyes’) to identify a receptive dysphasia • Ask patient to name people/objects (e.g. nurse, watch, pen) to identify a nominal dysphasia • Check articulation (ask patient to repeat phrases after you) for dysarthria Motor deficit Subtle pyramidal signs: • Check for pronator drift: ask patient to hold out arms and maintain their position with eyes closed (see opposite) • Check for

clumsiness of fine finger movements Sensory and visual inattention • Establish that sensation/visual field is intact on testing one side at a time • Retest sensation/visual fields on simultaneous testing of both sides; the affected side will no longer be felt/seen • Perform clock drawing test (see below) Truncal ataxia • Check if patient can sit up or stand without support General examination Skin • Xanthelasma • Rash (arteritis, splinter haemorrhages) • Colour change (limb ischaemia, deep vein thrombosis) • Pressure injury Eyes • Arcus senilis • Diabetic retinopathy • Hypertensive retinopathy • Retinal emboli Cardiovascular system • Heart rhythm (?atrial fibrillation) • Blood pressure (high or low) • Carotid bruit • Jugular venous pulse (raised in heart failure, low in hypovolaemia) • Murmurs (source of embolism) • Peripheral pulses and bruits (?generalised arteriopathy) Respiratory system • Signs of pulmonary oedema or infection • Oxygen saturation Abdomen • Palpable bladder (urinary retention) Locomotor system • Injuries sustained during collapse • Comorbidities that influence recovery, e.g. osteoarthritis Clock drawing test A An image drawn by a doctor. B An image drawn by a patient with left-sided neglect. A B

1150 • STROKE MEDICINE Functional anatomy and physiology The main arterial supply of the brain comes from the internal carotid arteries, which supply the anterior brain through the anterior and middle cerebral arteries, and the vertebral and basilar arteries (vertebrobasilar system), which provide the posterior circulation to the posterior cerebral arteries. The anterior and middle cerebral arteries supply the frontal and parietal lobes, while the posterior cerebral artery supplies the occipital lobe. The vertebral and basilar arteries perfuse the brainstem, mid-brain and cerebellum (Fig. 26.2). The functions of each of these Cerebrovascular disease is the third most common cause of death in high-income countries after cancers and ischaemic heart disease, and the most common cause of severe physical disability. It includes a range of disorders of the central nervous system (Fig. 26.1). Stroke is the most common clinical manifestation of cerebrovascular disease and results in episodes of brain dysfunction due to focal ischaemia or haemorrhage. Subarachnoid haemorrhage (SAH) and cerebral venous thrombosis (CVT) will be discussed separately, since their pathophysiology, clinical manifestations and management are distinct from those of stroke. Vascular dementia is described on page 1191. Fig. 26.1 A classification of stroke disease. Stroke (acute, focal brain dysfunction due to vascular disease) Total anterior circulation stroke (TACS) (15%) Partial anterior circulation stroke (PACS) (30%) Lacunar stroke (LACS) (20%) Posterior circulation stroke (POCS) (20%) Intracerebral haemorrhage (10%) Subarachnoid haemorrhage (5%) Central venous thrombosis (<1%) Anterior (carotid) circulation (65%) Posterior (vertebrobasilar circulation) (20%) Brain parenchyma (10%) Subarachnoid space (5%) Venous system Infarct (85%) Arterial (>99%) Haemorrhage (15%) Infarct (often develops secondary haemorrhage) Venous (<1%) • Embolism (cardiac, major vessels) • Thrombosis in situ Clinical classification Site of lesion Pathology Vascular system Common pathophysiology • Thrombosis in situ • Thrombosis • Embolism (cardiac) • Vascular degeneration • Aneurysm • Arteriovenous malformation • Aneurysm • Arteriovenous malformation • Vascular degeneration • Thrombosis in situ Fig. 26.2 Arterial circulation of the brain. A Horizontal view. B Lateral view. Anterior (carotid) circulation Posterior (vertebrobasilar) circulation Anterior cerebral artery (ACA) Posterior communicating artery (PCoA) Posterior cerebral artery (PCA) Small perforating vessels Anterior communicating artery (ACoA) Basilar artery (BA) Vertebral arteries (VA) Internal carotid arteries (ICA) Middle cerebral artery (MCA) PCA PCoA BA VA ICA ACA MCA A B

Investigations • 1151

Neuroimaging Computed tomography (CT) scanning is the mainstay of emergency stroke imaging. It allows the rapid identification of intracerebral bleeding and stroke ‘mimics’ (i.e. pathologies other than stroke that have similar presentations), such as tumours. Magnetic resonance imaging (MRI) is used when there is diagnostic uncertainty or delayed presentation, and when more information on brain structure and function is required (Fig. 26.4). Contraindications to MRI include cardiac pacemakers and claustrophobia on entering the scanner. CT angiography (CTA) and CT perfusion are now being used to characterise the cerebral circulation and areas of ischaemia better (p. 1072). Vascular imaging Various techniques are used to obtain images of extracranial and intracranial blood vessels (Fig. 26.5). The least invasive is ultrasound (Doppler or duplex scanning), which is used to image the carotid and the vertebral arteries in the neck. In skilled hands, reliable information can be provided about the degree of arterial stenosis and the presence of ulcerated plaques. Blood flow in the intracerebral vessels can be examined using transcranial Doppler. While the anatomical resolution is limited, it is improving and many centres no longer require formal angiography before proceeding to carotid endarterectomy (see below). Blood flow can also be detected by specialised sequences in MR angiography (MRA) or CTA but the anatomical resolution is still not as good as that of intra-arterial angiography, which outlines blood vessels by the injection of radio-opaque contrast intravenously or intra-arterially. The X-ray images obtained can be enhanced by the use of computer-assisted digital subtraction or spiral CT. Because of the significant risk of complications, intra-arterial contrast angiography is reserved for use when non-invasive methods have provided a contradictory picture or incomplete information, or when it is necessary to image the intracranial circulation in detail, e.g. to delineate a saccular aneurysm, an arteriovenous malformation or vasculitis. Blood tests These identify underlying causes of cerebrovascular disease, e.g. blood glucose (diabetes mellitus), triglycerides and cholesterol (hyperlipidaemia) or full blood count (polycythaemia). Erythrocyte Fig. 26.3 Venous circulation of the brain. Posterior Superior sagittal sinus Cavernous sinus Transverse sinus Jugular vein Anterior Fig. 26.4 Acute stroke seen on computed tomography (CT) scan with corresponding magnetic resonance imaging (MRI) appearance. A CT may show no evidence of early infarction. B A corresponding image seen on MRI diffusion weighted imaging (DWI) with changes of infarction in the middle cerebral artery (MCA) territory (arrows). A and B, Courtesy of Dr A. Farrell and Prof. J. Wardlaw. A B areas of the brain are described on page 1064. Communicating arteries provide connections between the anterior and posterior circulations and between left and right hemispheres, creating protective anastomotic connections that form the circle of Willis. In health, regulatory mechanisms maintain a constant cerebral blood flow across a wide range of arterial blood pressures to meet the high resting metabolic activity of brain tissue; cerebral blood vessels dilate when systemic blood pressure is lowered and constrict when it is raised. This autoregulatory mechanism can be disrupted after stroke. The venous collecting system is formed by a collection of sinuses over the surface of the brain, which drain into the jugular veins (Fig. 26.3). Investigations A range of investigations may be required to answer specific questions about brain structure and function and about the function of the vascular system.

 are initially reduced but then become increased with a spastic pattern of increased tone (see Box 25.14, p. 1082). Upper motor neuron weakness of the face (7th cranial nerve) is often present. Speech disturbance Dysphasia and dysarthria are the most common presentations of disturbed speech in stroke (p. 1087). Dysphasia indicates damage to the dominant frontal or parietal lobe (see Box 25.2, p. 1066), while dysarthria is a non-localising feature that reflects weakness or incoordination of the face, pharynx, lips, tongue or palate. Visual deficit Visual

loss can be due to unilateral optic ischaemia (called amaurosis fugax if transient), caused by disturbance of blood flow in the internal carotid artery and ophthalmic artery, leading to monocular blindness. Ischaemia of the occipital cortex or post-chiasmic nerve tracts results in a contralateral hemianopia (p. 1088). Visuo-spatial dysfunction Damage to the non-dominant cortex often results in contralateral visuo-spatial dysfunction, e.g. sensory or visual neglect and apraxia (inability to perform complex tasks despite normal motor, sensory and cerebellar function; p. 1086), sometimes misdiagnosed as delirium. sedimentation rate (ESR) and immunological tests, such as antineutrophil cytoplasmic antibodies (ANCAs, p. 992), may be required when vasculitis is suspected. Genetic testing for rarer inherited conditions, such as CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy), may be indicated. Lumbar puncture Lumbar puncture (p. 1077) is reserved for investigation of SAH. Cardiovascular investigations Electrocardiography (ECG; p. 448), including ECG monitoring and echocardiography (p. 451), may reveal abnormalities that may cause cardiac embolism in stroke. Presenting problems Most vascular lesions develop suddenly within a matter of minutes or hours, and so should be considered in the differential diagnosis of patients with any acute neurological presentation. Weakness Unilateral weakness is the classical presentation of stroke and, much more rarely, of CVT. The weakness is sudden, progresses rapidly and follows a hemiplegic pattern (see Fig. 25.17, p. 1082). There is rarely any associated abnormal movement. Reflexes Fig. 26.5 Different techniques for imaging blood vessels. A Doppler scan showing 80% stenosis of the internal carotid artery (arrow). B Three-dimensional reconstruction of CT angiogram showing stenosis at the carotid bifurcation (arrow). C MR angiogram showing giant aneurysm at the middle cerebral artery bifurcation (arrow). D Intra-arterial angiography showing arteriovenous malformation (arrow). A–D, Courtesy of Dr D. Collie. A B C D

However, if and when these homeostatic mechanisms fail, the process of ischaemia starts, and ultimately leads to infarction unless the vascular supply is restored. As the cerebral blood flow declines, different neuronal functions fail at various thresholds (Fig. 26.7). Once blood flow falls below the threshold for the maintenance of electrical activity, neurological deficit develops. At this level of blood flow, neurons are still viable; if blood flow increases again, function returns and the patient will have had a Fig. 26.6 Homeostatic responses to falling perfusion pressure in the brain following arterial occlusion. Vasodilatation initially maintains cerebral blood flow (A), but after maximal vasodilatation further falls in perfusion pressure lead to a decline in blood flow. An increase in tissue oxygen extraction, however, maintains the cerebral metabolic rate for oxygen (B). Still further falls in perfusion, and therefore blood flow, cannot be compensated; cerebral oxygen availability falls and symptoms appear, then infarction (C). Symptoms Infarction Cerebral oxygen extraction Cerebral blood volume Cerebral oxygen metabolism Blood flow Perfusion pressure Vasodilatation A B C Time (mins/hrs) 26.1 Risk factors for stroke Fixed risk factors • Age • Gender (male > female except at extremes of age) • Race (Afro-Caribbean > Asian

> European) • Previous vascular event: Myocardial infarction Stroke Peripheral vascular disease • Heredity • Sickle cell disease • High fibrinogen Modifiable risk factors • Blood pressure • Cigarette smoking • Hyperlipidaemia • Diabetes

mellitus • Heart disease: Atrial fibrillation Congestive cardiac failure Infective endocarditis • Excessive alcohol intake • Oestrogen-containing drugs: Oral contraceptive pill Hormone replacement therapy • Polycythaemia Ataxia Stroke causing damage to the cerebellum and its connections can present as an acute ataxia (p. 1086) and there may be associated brainstem features such as diplopia (p. 1088) and vertigo (p. 1086). The differential diagnosis includes vestibular disorders (p. 1104). Headache Sudden severe headache is the cardinal symptom of SAH but also occurs in intracerebral haemorrhage. Although headache is common in acute ischaemic stroke, it is rarely a dominant feature (p. 1080). Headache also occurs in cerebral venous disease. Seizure Seizure is unusual in acute stroke but may be generalised or focal (especially in cerebral venous disease). Coma Coma is uncommon, though it may occur with a brainstem event. If present in the first 24 hours, it usually indicates a subarachnoid or intracerebral haemorrhage (see Box 10.26, p. 194). Stroke Stroke is a common medical emergency. The incidence rises steeply with age, and in many lower- and middle-income countries it is rising in association with less healthy lifestyles. About 20% of stroke patients die within a month of the event and at least half of those who survive are left with physical disability. Pathophysiology Of the 180–300 patients per 100 000 population presenting annually with a stroke, 85% sustain a cerebral infarction due to inadequate blood flow to part of the brain, and most of the remainder have an intracerebral haemorrhage (see Fig. 26.1). Cerebral infarction Cerebral infarction is mostly caused by thromboembolic disease secondary to atherosclerosis in the major extracranial arteries (carotid artery and aortic arch). About 20% of infarctions are due to embolism from the heart, and a further 20% are due to thrombosis in situ caused by intrinsic disease of small perforating vessels (lenticulostriate arteries), producing so-called lacunar infarctions. The risk factors for ischaemic stroke reflect the risk factors for the underlying vascular disease (Box 26.1). About 5% are due to rare causes, including vasculitis (p. 1040), endocarditis (p. 527) and cerebral venous disease (see below). Cerebral infarction takes some hours to complete, even though the patient’s deficit may be maximal shortly after the vascular occlusion. After the occlusion of a cerebral artery, infarction may be forestalled by the opening of anastomotic channels from other arterial territories that restore perfusion to its territory. Similarly, reduction in perfusion pressure leads to compensatory homeostatic changes to maintain tissue oxygenation (Fig. 26.6). These compensatory changes can sometimes prevent occlusion of even a carotid artery from having any clinically apparent effect.

 of the excitatory neurotransmitter glutamate into the extracellular fluid. Glutamate opens membrane channels, allowing influx of calcium and more sodium into the neurons. Calcium activates intracellular enzymes that complete the destructive process. The release of inflammatory mediators by microglia and astrocytes causes death of all cell types in the area of maximum ischaemia. The infarction process is worsened by anaerobic production of lactic acid (Fig. 26.8) and consequent fall in tissue pH. There have been attempts to develop

neuroprotective drugs to slow down the processes leading to irreversible cell death but so far these have proved disappointing. The final outcome of occlusion of a cerebral blood vessel thus depends on the competence of circulatory homeostatic mechanisms, the metabolic demand, and the severity and duration of the reduction in blood flow. Higher brain temperature, e.g. in fever, and higher blood glucose have both been associated with a greater volume of infarction for a given reduction in cerebral blood flow. Subsequent restoration of blood flow may cause haemorrhage into the infarcted area (‘haemorrhagic transformation’). This is particularly likely in patients given antithrombotic or thrombolytic drugs, and in patients with larger infarcts. Radiologically, a cerebral infarct can be seen as a lesion that comprises a mixture of dead brain tissue that is already undergoing autolysis, and tissue that is ischaemic and swollen but recoverable (the ‘ischaemic penumbra’). The infarct swells with time and is at its maximal size a couple of days after stroke onset. At this stage, it may be big enough to exert mass effect both clinically and radiologically; sometimes, decompressive craniectomy is required (see below). After a few weeks, the oedema subsides and the infarcted area is replaced by a sharply defined fluid-filled cavity. Intracerebral haemorrhage Intracerebral haemorrhage causes about 10% of acute stroke events but is more common in low-income countries. It usually results from rupture of a blood vessel within the brain parenchyma but may also occur in a patient with SAH (see transient ischaemic attack (TIA). However, if blood flow falls further, a level is reached at which irreversible cell death starts. Hypoxia leads to an inadequate supply of adenosine triphosphate (ATP), which leads to failure of membrane pumps, thereby allowing influx of sodium and water into cells (cytotoxic oedema) and release Fig. 26.8 The process of neuronal ischaemia and infarction. (1) Reduction of blood flow reduces supply of oxygen and hence adenosine triphosphate (ATP). H+ is produced by anaerobic metabolism of available glucose. (2) Energy-dependent membrane ionic pumps fail, leading to cytotoxic oedema and membrane depolarisation, allowing calcium entry and releasing glutamate. (3) Calcium enters cells via glutamate-gated channels and (4) activates destructive intracellular enzymes (5), destroying intracellular organelles and cell membrane, with release of free radicals. Free fatty acid release activates pro-coagulant pathways that exacerbate local ischaemia. (6) Glial cells take up H+, can no longer take up extracellular glutamate and also suffer cell death, leading to liquefactive necrosis of whole arterial territory. (AMPA = α-amino-3-hydroxy-5-methyl-4isoxazolepropionic acid receptor; NMDA = N-methyl-D-aspartate; NO = nitric oxide) ↓Oxygen +glucose Anaerobic metabolism ↓ATP H+ Ca2+ Na+/K+ ATPase↓ Lipid peroxidases Proteases NO synthase Ca2+ Na+ AMPA NMDA Glutamate ↓Glutamate uptake by glia H+ K+ Glia Fe + H Water Na+ Oedema Free radicals Ca2+

Thromboxane Prostaglandins Free fatty acids Depolarisation Fig. 26.7 Thresholds of cerebral ischaemia. Symptoms of cerebral ischaemia appear when the blood flow has fallen to less than half of normal and energy supply is insufficient to sustain neuronal electrical function. Full recovery can occur if this level of flow is returned to normal but not if it is sustained. Further blood flow reduction below the next threshold causes failure of cell ionic pumps and starts the ischaemic cascade, leading to cell death.

Increased oxygen extraction Failure of electrical function Failure of ionic pumps Potassium efflux Sodium influx Cerebral blood flow mL/100 g/min Symptoms Cell death

such as abnormal movement). Provided there is a clear history of this, the chance of a brain lesion being anything other than vascular is 5% or less (Box 26.3). If symptoms progress over hours or days, other diagnoses must be excluded. Delirium and memory or balance disturbance are more often due to stroke mimics. Transient symptoms, e.g. syncope, amnesia, delirium and dizziness, do not reflect focal cerebral dysfunction but are often mistakenly attributed to TIA (see Fig. 10.3, p. 182, and Box 26.4). Campaigns to raise public awareness of the emergency nature of stroke exploit the fact that weakness of the face or arm, or disturbance of speech is the most common presentation. The clinical presentation of stroke depends on which arterial territory is involved and the size of the lesion (see Fig. 26.1). These will both have a bearing on management, such as suitability for carotid endarterectomy. The neurological deficit can be identified Fig. 26.9 CT scans showing intracerebral haemorrhage. A Basal ganglia haemorrhage with intraventricular extension. B Small cortical haemorrhage. A and B, Courtesy of Dr A. Farrell and Prof. J. Wardlaw. A B 26.3 Differential diagnosis of stroke and transient ischaemic attack ‘Structural’ stroke mimics • Primary cerebral tumours • Metastatic cerebral tumours • Extradural or subdural haematoma • Demyelination • Peripheral nerve lesions (vascular or compressive) • Cerebral abscess ‘Functional’ stroke mimics • Todd’s paresis (after epileptic seizure) • Hypoglycaemia • Migrainous aura (with or without headache) • Focal seizures • Ménière’s disease or other vestibular disorder • Conversion disorder (p. 1202) • Encephalitis 26.2 Causes of intracerebral haemorrhage and associated risk factors Disease Risk factors Complex small-vessel disease with disruption of vessel wall Age Hypertension High cholesterol Amyloid angiopathy Familial (rare) Age Impaired blood clotting Anticoagulant therapy Blood dyscrasia Thrombolytic therapy Vascular anomaly Arteriovenous malformation Cavernous haemangioma Substance misuse Alcohol Amphetamines Cocaine below) if the artery ruptures into the brain substance as well as the subarachnoid space. Haemorrhage frequently occurs into an area of brain infarction and, if the volume of haemorrhage is large, it may be difficult to distinguish from primary intracerebral haemorrhage both clinically and radiologically (Fig. 26.9). The risk factors and underlying causes of intracerebral haemorrhage are listed in Box 26.2. Explosive entry of blood into the brain parenchyma causes immediate cessation of function in that area as neurons are disrupted and white-matter fibre tracts are split apart. The haemorrhage itself may expand over the first minutes or hours, or it may be associated with a rim of cerebral oedema, which, along with the haematoma, acts like a mass lesion to cause progression of the neurological deficit. If big enough, this can cause shift of the intracranial contents, producing transtentorial coning and sometimes rapid death (p. 1127). If the patient survives, the haematoma is gradually absorbed, leaving a haemosiderin-lined slit in the brain parenchyma. Clinical features Both acute stroke and transient ischaemic attack (TIA) are characterised by a rapid-onset, focal deficit of brain function and can be considered as a spectrum of symptoms from transient (TIA) to persistent (stroke). The typical presentation occurs over minutes, affects an identifiable area of brain and is ‘negative’ in character (i.e. abrupt loss of function without positive features 26.4 Characteristic features of stroke and non-stroke syndromes (‘stroke mimics’) Feature Stroke Stroke mimics Symptom onset Sudden (minutes) Often slower onset Symptom progression Rapidly reaches maximum severity Often gradual onset Severity of deficit Unequivocal May be variable/uncertain Pattern of deficit Hemispheric pattern May be non-specific with delirium, memory loss, balance disturbance Loss of consciousness Uncommon More common

 hypoxia or severe systemic infection. The combination of severe headache and vomiting at the onset of the focal deficit is suggestive of intracerebral haemorrhage. General examination may provide clues to the cause and identify important comorbidities and

complications. Several terms have been used to classify strokes, often based on the duration and evolution of symptoms: • Transient ischaemic attack (TIA) describes a stroke in which symptoms resolve within 24 hours – an arbitrary cut-off that has little value in practice, apart from perhaps indicating that underlying cerebral haemorrhage or extensive cerebral infarction is extremely unlikely. The term from the patient's history and (if it is persistent) the neurological examination. The presence of a unilateral motor deficit, a higher cerebral function deficit such as aphasia or neglect, or a visual field defect usually places the lesion in the cerebral hemisphere. Ataxia, diplopia, vertigo and/or bilateral weakness usually indicate a lesion in the brainstem or cerebellum. Different combinations of these deficits define several stroke syndromes (Fig. 26.10), which reflect the site and size of the lesion and may provide clues to the underlying pathology. Reduced conscious level usually indicates a large-volume lesion in the cerebral hemisphere but may result from a lesion in the brainstem or complications such as obstructive hydrocephalus, Fig. 26.10 Clinical and radiological features of the stroke syndromes. The top three diagrams show coronal sections of the brain and the bottom one shows a sagittal section. The anatomical locations of cerebral functions are shown with the nerve tracts in green. A motor (or sensory) deficit (shown by the areas shaded red) can occur with damage to the relevant cortex (PACS), nerve tracts (LACS) or both (TACS). The corresponding CT scans show horizontal slices at the level of the lesion, highlighted by the arrows. Combination of: Hemiparesis Higher cerebral dysfunction (e.g. aphasia) Hemisensory loss Homonymous hemianopia (damage to optic radiations) Leg Total anterior circulation syndrome (TACS) Clinical syndrome Common symptoms Common cause CT scan features Arm Higher cerebral functions Face Optic radiations Middle cerebral artery occlusion (Embolism from heart or major vessels) Isolated motor loss (e.g. leg only, arm only, face) Isolated higher cerebral dysfunction (e.g. aphasia, neglect) Mixture of higher cerebral dysfunction and motor loss (e.g. aphasia with right hemiparesis) Leg Partial anterior circulation syndrome (PACS) Arm Higher cerebral functions Face Optic radiations Occlusion of a branch of the middle cerebral artery or anterior cerebral artery (Embolism from heart or major vessels) Pure motor stroke – affects two limbs Pure sensory stroke Sensory-motor stroke No higher cerebral dysfunction or hemianopia Leg Lacunar syndrome (LACS) Posterior circulation stroke (POCS) (lateral view) Arm Higher cerebral functions Visual cortex Cerebellum Cranial nerve nuclei Face Optic radiations Thrombotic occlusion of small perforating arteries (Thrombosis in situ) Homonymous hemianopia (damage to visual cortex) Cerebellar syndrome Cranial nerve syndromes Occlusion in vertebral, basilar or posterior cerebral artery territory (Cardiac embolism or thrombosis in situ)

Neuroimaging Brain imaging with either CT or MRI should be performed in all patients with acute stroke. Exceptions are where results would not influence management, such as in the advanced stage of a terminal illness. CT remains the most practical and widely available method of imaging the brain. It will usually exclude non-stroke lesions, including subdural haematomas and brain tumours, and will demonstrate intracerebral haemorrhage within minutes of stroke onset (see Fig. 26.9). However, especially within the first few hours after symptom onset, CT changes in cerebral infarction may be completely absent or only very subtle. Changes often develop over time (see Fig. 26.13) but small cerebral infarcts may never show up on CT scans. For some purposes, a CT scan performed within 24 hours is adequate TIA traditionally also includes patients with amaurosis fugax, usually due to a vascular occlusion in the retina. • Stroke describes those events in which symptoms last more than 24 hours. The differential diagnosis of patients with symptoms lasting a

few minutes or hours is similar to those with persisting symptoms (see Box 26.3). The term ‘minor stroke’ is sometimes used to refer to symptoms lasting over 24 hours but not causing significant disability. • Progressing stroke (or stroke in evolution) describes a stroke in which the focal neurological deficit worsens after the patient first presents. Such worsening may be due to increasing volume of infarction, haemorrhagic transformation or increasing cerebral oedema. • Completed stroke describes a stroke in which the focal deficit persists and is not progressing. When assessing a patient within hours of symptom onset, it is not possible to distinguish stroke from TIA unless symptoms have already resolved. In clinical practice, it is important to distinguish those patients with strokes who have persisting focal neurological symptoms when seen from those whose symptoms have already resolved. Investigations Investigation of acute stroke aims to confirm the vascular nature of a lesion, distinguish infarction from haemorrhage and identify the underlying vascular disease and risk factors (Box 26.5). Risk factor analysis Initial investigation includes a range of simple blood tests to detect common vascular risk factors and markers of rarer causes, along with an ECG and brain imaging. Where there is uncertainty about the nature of the stroke, further investigations are indicated. This especially applies to younger patients, who are less likely to have atherosclerotic disease (Box 26.6). 26.5 Investigation of a patient with an acute stroke Diagnostic question Investigation Is it a vascular lesion? CT/MRI Is it ischaemic or haemorrhagic? CT/MRI Is it a subarachnoid haemorrhage? CT/lumbar puncture Is there any cardiac source of embolism? ECG Holter monitoring Echocardiogram What is the underlying vascular disease? Duplex ultrasound of carotids MRA CTA Contrast angiography What are the risk factors? Full blood count Cholesterol Blood glucose Is there an unusual cause? ESR Serum protein electrophoresis Clotting/thrombophilia screen (CT = computed tomography; CTA = computed tomographic angiography; ECG = electrocardiogram; ESR = erythrocyte sedimentation rate; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging) 26.6 Causes and investigation of acute stroke in young patients Cause Investigation Cerebral infarct Cardiac embolism Echocardiography (including transoesophageal) Premature atherosclerosis Serum lipids Arterial dissection MRI CTA Reversible cerebral vasoconstriction syndromes MRI CTA Thrombophilia Protein C, protein S Antithrombin III Factor V Leiden, prothrombin Homocystinuria (p. 369) Urinary amino acids Methionine loading test Antiphospholipid antibody syndrome (p. 977) Anticardiolipin antibodies/lupus anticoagulant Systemic lupus erythematosus ANA Vasculitis (e.g. primary angiitis of the central nervous system) ESR CRP ANCA CADASIL CARASIL MRI brain Genetic analysis Skin biopsy Mitochondrial cytopathy Serum lactate White cell mitochondrial DNA Muscle biopsy Mitochondrial molecular genetics Fabry’s disease Alpha-galactosidase levels Sickle cell disease Sickle cell studies Neurovascular syphilis Syphilis serology Primary intracerebral haemorrhage AVM MRI/MRA Drug misuse Drug screen (amphetamine, cocaine) Coagulopathy PT and APTT Platelet count Subarachnoid haemorrhage Saccular (‘berry’) aneurysm MRI/MRA AVM MRI/MRA Vertebral dissection MRI/MRA (ANA = antinuclear antibody; ANCA = antineutrophil cytoplasmic antibody; APTT = activated partial thromboplastin time; AVM = arteriovenous malformation; CADASIL/CARASIL = cerebral autosomal dominant/recessive arteriopathy with subcortical infarcts and leucoencephalopathy; CRP = C-reactive protein; CTA = computed tomographic angiography; ESR = erythrocyte sedimentation rate; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; PT = prothrombin time)

 disability and handicap through rehabilitation, and reducing the risk of recurrent stroke or other vascular events. With TIA there is no persisting brain damage and disability, so the priority is to reduce the risk of further vascular events. Supportive care Rapid

admission of patients to a specialised stroke unit facilitates coordinated care from a specialised multidisciplinary team, and has been shown to reduce both mortality and residual disability amongst survivors. For every 1000 patients managed in a stroke unit, an extra 50 will avoid death or long-term disability, compared to those managed in general wards. Consideration of a patient's rehabilitation needs should commence at the same time as acute medical management. Dysphagia is common and can be detected by an early bedside test of swallowing. This allows hydration, feeding and medication to be given safely, if necessary by nasogastric tube or intravenously. In the acute phase, a checklist may be useful (Box 26.8) to ensure that all the factors that might influence outcome have been addressed. In recent years, many services have developed hyperacute stroke units (HASUs) to ensure that patients are given immediate access to these interventions, as well as urgent medical treatments. The patient's neurological deficits may worsen during the first few hours or days after their onset. This may be due to extension of the area of infarction, haemorrhage transformation of an infarction, or the development of oedema with consequent mass effect. It is important to distinguish these patients from those who Fig. 26.11 Emergency management of stroke. *Varies with patient selection and delay in treatment. (NNT = number needed to treat to avoid one death or long-term disability) Haemorrhagic stroke Neuroimaging Ischaemic stroke Reverse coagulation abnormality Eligible for urgent reperfusion therapy? Yes (20%) No (80%) Aspirin (NNT 80) Intravenous thrombolysis (NNT 9–20)* and/or Mechanical thrombectomy (NNT 8–10)* Acute stroke unit care (NNT 20) Identify cause and plan secondary prevention Clinical diagnosis of stroke (Box 26.4) 26.7 Indications for immediate CT/MRI in acute stroke • Patient on anticoagulants or with abnormal coagulation • Consideration for reperfusion (thrombolysis) or immediate anticoagulation • Deteriorating conscious level or rapidly progressing deficits • Suspected cerebellar haematoma, to exclude hydrocephalus but there are certain circumstances in which an immediate CT scan is essential (Box 26.7). Even in the absence of changes suggesting infarction, abnormal perfusion of brain tissue can be imaged with CT after injection of contrast media (i.e. CT perfusion scanning). This can be useful in guiding immediate treatment of ischaemic stroke. MRI is not as widely available as CT and scanning times are longer. However, MRI diffusion weighted imaging (DWI) can detect ischaemia earlier than CT, and other MRI sequences can also be used to demonstrate abnormal perfusion (see Fig. 26.4). MRI is more sensitive than CT in detecting strokes affecting the brainstem and cerebellum, and, unlike CT, can reliably distinguish haemorrhagic from ischaemic stroke even several weeks after the onset. CT and MRI may reveal clues as to the nature of the arterial lesion. For example, there may be a small, deep lacunar infarct indicating small-vessel disease, or a more peripheral infarct suggesting an extracranial source of embolism (see Fig. 26.10). In a haemorrhagic lesion, the location might indicate the presence of an underlying vascular malformation, saccular aneurysm or amyloid angiopathy. More recently, CTA is being used to show vessel occlusion suitable for clot retrieval (see later). Vascular imaging Many ischaemic strokes are caused by atherosclerotic thromboembolic disease of the major extracranial vessels. Detection of extracranial vascular disease can help establish why the patient has had an ischaemic stroke and, in selected patients, may lead on to specific treatments, including carotid endarterectomy to reduce the risk of further stroke (see below). The presence or absence of a carotid bruit is not a reliable indicator of the degree of carotid stenosis. Extracranial arterial disease can be non-invasively identified with duplex ultrasound, MRA or CTA (see Fig. 26.5), or occasionally by intra-arterial contrast radiography as above. Cardiac investigations Approximately 20% of ischaemic strokes are due to embolism from the heart. The most common causes are atrial fibrillation, prosthetic heart valves, other valvular abnormalities and recent myocardial infarction. These may be identified by clinical examination

and ECG, but a transthoracic or transoesophageal echocardiogram is also required to confirm the presence of a clinically apparent cardiac source or to identify an unsuspected source such as endocarditis, atrial myxoma, intracardiac thrombus or patent foramen ovale. Such findings may lead on to specific cardiac treatment. Management Management (Fig. 26.11) is aimed at identifying the cause, minimising the volume of brain that is irreversibly damaged, preventing complications (Fig. 26.12), reducing the patient's

mannitol or artificial ventilation. Surgical decompression to reduce intracranial pressure should be considered in appropriate patients. Reperfusion (thrombolysis and thrombectomy) Rapid reperfusion in ischaemic stroke can reduce the extent of brain damage. Intravenous thrombolysis with recombinant tissue Fig. 26.12 Complications of acute stroke. Nurse semi-erect Avoid aspiration (nil by mouth, nasogastric tube, possible gastrostomy) Complications Prevention Treatment Epileptic seizures Maintain cerebral oxygenation Avoid metabolic disturbance Anticonvulsants Depression and anxiety Maintain positive attitude and provide information Antidepressants Painful shoulder Avoid traction injury Shoulder/arm supports Physiotherapy Physiotherapy Local glucocorticoid injections Chest infection Antibiotics Physiotherapy Appropriate aperients and diet Constipation Appropriate aperients Avoid catheterisation if possible Use penile sheath Urinary infection Antibiotics Frequent turning Monitor pressure areas Avoid urine damage to skin Pressure sores Nursing care Pressure-relieving mattress Maintain hydration Early mobilisation Heparin (for high-risk patients only) Deep vein thrombosis/ pulmonary embolism Anticoagulation (exclude haemorrhage first) are deteriorating as a result of complications such as hypoxia, sepsis, epileptic seizures or metabolic abnormalities that may be reversed more easily. Patients with cerebellar haematomas or infarcts with mass effect may develop obstructive hydrocephalus and some will benefit from insertion of a ventricular drain and/or decompressive surgery (see Fig. 26.11). Some patients with large haematomas or infarction with massive oedema in the cerebral hemispheres may benefit from anti-oedema agents, such as Blood glucose • Check blood glucose and treat when levels are ≥11.1 mmol/L (200 mg/dL) (by insulin infusion or glucose/potassium/insulin (GKI) • Monitor closely to avoid hypoglycaemia Temperature • If pyrexic, investigate and treat underlying cause • Control with antipyretics, as raised brain temperature may increase infarct volume Pressure areas • Reduce risk of skin breakdown: Treat infection Maintain nutrition Provide pressure-relieving mattress Turn immobile patients regularly Incontinence • Check for constipation and urinary retention; treat these appropriately • Avoid urinary catheterisation unless patient is in acute urinary retention or incontinence is threatening pressure areas Mobilisation • Avoid bed rest Airway • Perform bedside screen and keep patient nil by mouth if swallowing unsafe or aspiration occurs Breathing • Check respiratory rate and give oxygen if saturation < 95% Circulation • Check peripheral perfusion, pulse and blood pressure, and treat abnormalities with fluid replacement, anti-arrhythmics and inotropic drugs as appropriate Hydration • If signs of dehydration, give fluids parenterally or by nasogastric tube Nutrition • Assess nutritional status and provide supplements if needed • If dysphagia persists for >48 hrs, start feeding via nasogastric tube Medication • If dysphagic, consider other routes for essential medications Blood pressure • Unless there is heart or renal failure, evidence of hypertensive encephalopathy or aortic dissection, do not lower blood pressure abruptly in first week as it may reduce cerebral perfusion. Blood pressure often returns towards patient's normal level within days 26.8 How to manage a patient with acute stroke

 haemorrhage. The potential gain from good secondary prevention can be expressed as the number needed to treat (NNT) to avoid a recurrent stroke. Patients with ischaemic events should be put on long-term antiplatelet drugs (NNT 100) and statins (NNT 60) to lower cholesterol. For patients in atrial fibrillation, the risk can be reduced substantially (NNT 15) by using oral anticoagulation with warfarin to achieve an international normalised ratio (INR) of 2–3. The newer direct oral anticoagulants (such as dabigatran, rivaroxaban and apixaban) are now widely used, offering improved safety and effectiveness at increased drug cost. The risk of recurrence after both ischaemic and haemorrhagic strokes can be reduced by blood pressure reduction, even for those with relatively normal blood pressures (NNT 50). Carotid endarterectomy and angioplasty A small proportion of patients with a carotid territory ischaemic stroke or TIA will have more than 50% stenosis of the carotid artery on the side of the brain lesion. Such patients have a greater than average risk of stroke recurrence. For those without major residual disability, removal of the stenosis has been shown to reduce the overall risk of recurrence (NNT 15), although the operation itself carries about a 5% risk of stroke. Surgery is most effective in patients with more severe stenoses (70–99%) and when it is performed within the first couple of weeks after the TIA or ischaemic stroke. Carotid angioplasty and stenting are technically feasible but have not been shown to be as effective as endarterectomy for the majority of eligible patients. Endarterectomy of asymptomatic carotid stenosis has been shown to reduce the subsequent risk of stroke but the small absolute benefit does not justify its routine use. Unusual causes A minority of strokes are caused by arterial dissection of the carotid (carotid dissection) or vertebral artery (vertebral artery dissection). The presenting history often includes minor injury and face or neck pain. After confirmation on angiography (MRA or CTA), treatment is with either antiplatelet drugs or anticoagulation. Reversible vasoconstriction syndromes require good physiological control (particularly blood pressure). Subarachnoid haemorrhage Subarachnoid haemorrhage (SAH) is less common than ischaemic stroke or intracerebral haemorrhage (see Fig. 26.1) and affects about 6/100 000 of the population. Women are affected more commonly than men and the condition usually presents before the age of 65. The immediate mortality of aneurysmal SAH is about 30%; survivors have a recurrence (or rebleed) rate of about 40% in the first 4 weeks and 3% annually thereafter. Some 85% of cases of SAH are caused by saccular or ‘berry’ aneurysms arising from the bifurcation of cerebral arteries (see Fig. 26.2), particularly in the region of the circle of Willis. The most common sites are in the anterior communicating artery (30%), posterior communicating artery (25%) or middle cerebral artery (20%). There is an increased risk in first-degree relatives of those with saccular aneurysms, and in patients with polycystic kidney disease (p. 405) and congenital connective tissue defects such as Ehlers–Danlos syndrome (p. 970). In about 10% of cases, SAHs are non-aneurysmal haemorrhages (so-called peri-mesencephalic haemorrhages), which have a very characteristic appearance on CT and a benign outcome in terms of mortality and recurrence. 26.9 Anticoagulation in old age • Increased risk: older age and previous stroke increase the risk of stroke in the presence of atrial fibrillation, and bleeding risk with anticoagulation. • Falls risk: elderly patients are more prone to falls, including head injuries, which increase bleeding risk. • Monitoring of therapy: fine adjustments to daily dose of warfarin may be more difficult, as may monitoring of therapy. • Impact of comorbidities: the presence of conditions such as chronic renal impairment, diabetes or heart failure may affect the risk:benefit ratio, and decisions to use anticoagulation must be weighed carefully. Decision aids have been developed to assist (see ‘Further information’). plasminogen activator (rt-PA) increases the risk of haemorrhagic transformation of the cerebral infarct with potentially fatal results. The main contraindications are bleeding risk (recent haemorrhage, anticoagulant therapy) and delay to treatment; the earlier

treatment is given, the greater the benefit. However, if given within 4.5 hours of symptom onset to carefully selected patients, the haemorrhagic risk is offset by an improved overall outcome. Recently mechanical clot retrieval (thrombectomy) in patients with a large-vessel occlusion can greatly improve the chances of avoiding disability (see Fig. 26.11). Aspirin In the absence of contraindications, aspirin (300 mg daily) should be started immediately after an ischaemic stroke unless rt-PA has been given, in which case it should be withheld for at least 24 hours. Aspirin reduces the risk of early recurrence and has a small but clinically worthwhile effect on long-term outcome (see Fig. 26.11); it may be given by rectal suppository or by nasogastric tube in dysphagic patients. Heparin Anticoagulation with heparin has been widely used to treat acute ischaemic stroke in the past. While it reduces the risk of early ischaemic recurrence and venous thromboembolism, it increases the risk of both intracranial and extracranial haemorrhage. Furthermore, routine use of heparin does not result in better long-term outcomes, and therefore it should not be used in the routine management of acute stroke. It is unclear whether heparin might provide benefit in selected patients, such as those with recent myocardial infarction, arterial dissection or progressing strokes. Intracranial haemorrhage must be excluded on brain imaging before considering anticoagulation. Coagulation abnormalities In those with intracerebral haemorrhage, coagulation abnormalities should be reversed as quickly as possible to reduce the likelihood of the haematoma enlarging. This most commonly arises in those on warfarin therapy. There is no evidence that clotting factors are useful in the absence of a clotting defect. Management of risk factors The approaches used are summarised in Figure 26.13. The average risk of a further stroke is 5–10% within the first week of a stroke or TIA, perhaps 15% in the first year and 5% per year thereafter. The risks are not substantially different for intracerebral

present at onset if there is an associated intracerebral haematoma. A third nerve palsy may be present due to local pressure from an aneurysm of the posterior communicating artery, but this is rare. Fundoscopy may reveal a subhyaloid haemorrhage, which represents blood tracking along the subarachnoid space around the optic nerve. Investigations CT brain scanning and lumbar puncture are required. The diagnosis of SAH can be made by CT but a negative result does not completely exclude it, since small amounts of blood in the subarachnoid space cannot be detected by CT (see Fig. 26.14). Lumbar puncture should be performed 12 hours after symptom onset if possible, to allow detection of xanthochromia (p. 1077). If either of these tests is positive, cerebral angiography (see Fig. 26.5) is required to determine the optimal approach to prevent recurrent bleeding. Around 5% of SAHs are due to arteriovenous malformations and vertebral artery dissection. Clinical features SAH typically presents with a sudden, severe, ‘thunderclap’ headache (often occipital), which lasts for hours or even days, often accompanied by vomiting, raised blood pressure and neck stiffness or pain. It commonly occurs on physical exertion, straining and sexual excitement. There may be loss of consciousness at the onset, so SAH should be considered if a patient is found comatose. About 1 patient in 8 with a sudden severe headache has SAH and, in view of this, all who present in this way require investigation to exclude it (Fig. 26.14). On examination, the patient is usually distressed and irritable, with photophobia. There may be neck stiffness due to subarachnoid blood but this may take some hours to develop. Focal hemisphere signs, such as hemiparesis or aphasia, may be Fig. 26.13 Strategies for secondary prevention of stroke. (1) Lower blood pressure with caution in patients with postural hypotension, renal impairment or bilateral carotid stenosis. (2) Other statins can be used as an alternative to simvastatin in patients on

warfarin or digoxin. (3) Warfarin and aspirin have been used in combination in patients with prosthetic heart valves. (4) The combination of aspirin and clopidogrel is indicated only in patients with unstable angina or those with a temporary high risk of recurrence (e.g. carotid stenosis). (ACE = angiotensin-converting enzyme; BP = blood pressure; CT = computed tomography; ECG = electrocardiogram; MRI = magnetic resonance imaging; TIA = transient ischaemic attack; U&Es = urea and electrolytes) Antiplatelet drugs4 • Aspirin 300 mg at once then 75 mg daily • Clopidogrel 75 mg daily is effective alternative Lower cholesterol2 if total cholesterol

> 3.5 mmol/L (135 mg/dL) with simvastatin 40 mg nocte, after checking liver function tests Anticoagulation if no contraindications. Warfarin with target INR 2–3 (or 3.5 if mechanical prosthetic valve). Direct oral anticoagulants (DOACs) offer safe, effective alternative Consider • Cardioversion • Anti-arrhythmic Carotid endarterectomy Refer if > 70% stenosis on symptomatic side Carotid duplex Lower BP1 if BP > 130/70 mmHg 1–2 weeks after onset • Thiazide diuretic • ACE inhibitor (check U&Es) • Other agents Lifestyle • Smoking cessation • Lower salt intake • Lower fat intake • Lower excess alcohol intake • Increase exercise • Lose excess weight Haemorrhagic Sinus rhythm If contraindications to anticoagulation, e.g. bleeding, falls, binge drinking, poor adherence Thyroid function tests and echocardiogram Atrial fibrillation ECG Ischaemic Single typical TIA Stroke or atypical or multiple cerebral TIAs CT brain scan within 24 hours of onset; MRI if later than 7 days

1162 • STROKE MEDICINE (focal or generalised). The deficit can increase if spreading thrombophlebitis occurs. Investigations and management MR venography demonstrates a filling defect in the affected vessel. Anticoagulation, initially with heparin followed by warfarin, is beneficial, even in the presence of venous haemorrhage. In selected patients, endovascular thrombolysis has been advocated. Management of underlying causes and complications, such as persistently raised intracranial pressure, is important. About 10% of cerebral venous sinus thrombosis, particularly cavernous sinus thrombosis, is associated with infection (most commonly Staphylococcus aureus), needing antibiotic treatment. Otherwise, the treatment of choice is anticoagulation. Further information Websites eso-stroke.org European Stroke Organisation guidelines. nhs.uk/actfast FAST (face, arms, speech, time) campaign to raise public awareness of the emergency nature of stroke. nice.org.uk/guidance National Institute for Health and Care Excellence CG180 ‘Tools and resources’ includes a patient decision aid – Atrial fibrillation: medicines to help reduce your risk of a stroke – what are the options? rcplondon.ac.uk/resources/stroke-guidelines Royal College of Physicians of London clinical guideline. stroke.cochrane.org Systematic reviews of stroke treatments. stroketraining.org Stroke Training and Awareness Resources. Fig. 26.14 Investigation of subarachnoid haemorrhage. (CSF = cerebrospinal fluid; CT = computed tomography) Emergency CT Traumatic lumbar punctures do not cause xanthochromia in that specimen Negative (<10% of subarachnoid haemorrhage) Shows subarachnoid haemorrhage Blood/xanthochromia CSF (after 12 hours) If CT and CSF at 12 hours are negative the patient has not had a subarachnoid haemorrhage Refer to neurosurgeons Resuscitate Nimodipine 60 mg 26.11 Clinical features of cerebral venous thrombosis Cavernous sinus thrombosis • Proptosis, ptosis, headache, external and internal ophthalmoplegia, papilloedema, reduced sensation in trigeminal first division • Often bilateral, patient ill and febrile

Superior sagittal sinus thrombosis • Headache, papilloedema, seizures • Clinical features may resemble idiopathic intracranial hypertension (p. 1133) • May involve veins of both hemispheres, causing advancing motor and sensory focal deficits Transverse sinus thrombosis • Hemiparesis, seizures, papilloedema • May spread to jugular foramen and involve cranial nerves 9, 10 and 11 26.10 Causes of cerebral venous thrombosis Predisposing systemic causes • Dehydration • Pregnancy • Behçet's disease (p. 1043) • Thrombophilia (p. 922) • Hypotension • Oral contraceptive use Local causes • Paranasal sinusitis • Meningitis, subdural empyema • Penetrating head and eye wounds • Facial skin infection • Otitis media, mastoiditis • Skull fracture Management Nimodipine (30–60 mg IV for 5–14 days, followed by 360 mg orally for a further 7 days) is usually given to prevent delayed ischaemia in the acute phase. Insertion of platinum coils into an aneurysm (via an endovascular procedure) or surgical clipping of the aneurysm neck reduces the risk of both early and late recurrence. Coiling is associated with fewer perioperative complications and better outcomes than surgery; where feasible, it is now the procedure of first choice. Arteriovenous malformations can be managed either by surgical removal, by ligation of the blood vessels that feed or drain the lesion, or by injection of material to occlude the fistula or draining veins. Treatment may also be needed for complications of SAH, which include obstructive hydrocephalus (that may require drainage via a shunt), delayed cerebral ischaemia due to vasospasm (which may be treated with vasodilators), hyponatraemia (best managed by fluid restriction) and systemic complications associated with immobility, such as chest infection and venous thrombosis. Cerebral venous disease Thrombosis of the cerebral veins and venous sinuses (cerebral venous thrombosis) is much less common than arterial thrombosis. However, it has been recognised with increasing frequency in recent years, as access to non-invasive imaging of the venous sinuses using MR venography has increased. The main causes are listed in Box 26.10. Clinical features Cerebral venous sinus thrombosis usually presents with symptoms of raised intracranial pressure, seizures and focal neurological symptoms. The clinical features vary according to the sinus involved (Box 26.11 and see Fig. 26.3). Cortical vein thrombosis presents with focal cortical deficits such as aphasia and hemiparesis (depending on the area affected), and epilepsy

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Revision #1
Created 2026-01-08 16:24:56 UTC by Omar Ayman
Updated 2026-01-08 16:24:56 UTC by Omar Ayman