## Symptoms and Signs

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**SUBARACHNOID HAEMORRHAGE**

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**PATIENT QUESTIONS**

5.53 What are the symptoms of TIA and stroke? 139
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5.1 What are focal neurological symptoms and signs?

These are clinical features (see Box 5.1) that arise from a disturbance in an identifiable focal area of the brain, for example unilateral weakness (corticospinal tract) or clumsiness (cerebellum), unilateral sensory loss (spinothalamic tract), speech disorder (dominant hemisphere) and double vision (oculomotor pathways). They are caused by focal cerebral ischaemia or haemorrhage.

**BOX 5.1 Focal neurological and ocular symptoms**

**Motor symptoms**
Weakness or clumsiness of one side of the body, in whole or in part (hemiparesis)
Simultaneous bilateral weakness (paraparesis, quadriparesis)*
Difficulty swallowing (dysphagia)*
Imbalance (ataxia)*

**Speech or language disturbances**
Difficulty understanding or expressing spoken language (dysphasia)
Difficulty reading (dyslexia) or writing (dysgraphia)
Difficulty calculating (dyscalculia)
Slurred speech (dysarthria)*

**Sensory symptoms**

**Somatosensory**
Altered feeling on one side of the body, in whole or in part (hemisensory disturbance)

**Visual**
Loss of vision in one eye, in whole or in part (transient monocular blindness or amaurosis fugax)
Loss of vision in the left or the right half or quarter of the visual field (hemianopia, quadrantanopia)
Bilateral blindness
Double vision (diplopia)*

**Vestibular symptoms**
A spinning sensation (vertigo)*

**Behavioural or cognitive symptoms**
Difficulty dressing, combing hair, cleaning teeth, etc.; geographical disorientation; difficulty copying diagrams such as a clock, flower or intersecting cubes (visual–spatial–perceptual dysfunction)
Forgetfulness (amnesia)*

* As an isolated symptom, this does not necessarily indicate transient focal cerebral ischaemia, because there are many other potential causes.
5.2 **What are non-focal neurological symptoms and signs?**

Non-focal symptoms (see Box 5.2) are not neuroanatomically localising, for example light headedness, faintness, ‘dizziness’, generalised weakness and drop attacks.

**BOX 5.2 Non-focal neurological symptoms**

- Generalised weakness and/or sensory disturbance
- Lightheadedness
- Faintness
- ‘Blackouts’ with altered or loss of consciousness or fainting, with or without impaired vision in both eyes
- Incontinence of urine or faeces
- Confusion
- Any of the following symptoms, if isolated*
  - A spinning sensation (vertigo)
  - Ringing in ears (tinnitus)
  - Difficulty swallowing (dysphagia)
  - Slurred speech (dysarthria)
  - Double vision (diplopia)
  - Loss of balance (ataxia)

* If these symptoms occur in combination, or with focal neurological symptoms, they may indicate focal cerebral ischaemia.

Non-focal symptoms alone should not be interpreted as a transient ischaemic attack (TIA) or stroke because they are seldom due to focal cerebral ischaemia or haemorrhage.

However, the neurological symptoms are not always easy to categorise. Sensory and motor disturbances in a pseudo-radicular pattern (such as wrist drop or tingling in two or three fingers) may reflect focal neurological dysfunction; so may cognitive changes such as amnesia but these can be difficult to characterise and quantify, particularly when transient. Vertigo, confusion and dysarthria may reflect either focal or non-focal pathology, depending on whether other definitely focal neurological symptoms occur concurrently and whether they occur in the right milieu.

5.3 **What are the clinical features (symptoms and signs) of TIA and stroke?**

The clinical features of a TIA and stroke are the symptoms and signs of a sudden loss of function of a focal part of the brain (i.e. focal neurological dysfunction) (see Table 5.1). They are determined by the site of the brain
TABLE 5.1 Neurological symptoms during transient ischaemic attacks

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Proportion*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral weakness, heaviness or clumsiness</td>
<td>50</td>
</tr>
<tr>
<td>Unilateral sensory symptoms</td>
<td>35</td>
</tr>
<tr>
<td>Slurred speech (dysarthria)</td>
<td>23</td>
</tr>
<tr>
<td>Transient monocular blindness</td>
<td>18</td>
</tr>
<tr>
<td>Difficulty speaking (dysphasia)</td>
<td>18</td>
</tr>
<tr>
<td>Unsteadiness (ataxia)</td>
<td>12</td>
</tr>
<tr>
<td>Dizziness (vertigo)</td>
<td>5</td>
</tr>
<tr>
<td>Homonymous hemianopia</td>
<td>5</td>
</tr>
<tr>
<td>Double vision (diplopia)</td>
<td>5</td>
</tr>
<tr>
<td>Bilateral limb weakness</td>
<td>4</td>
</tr>
<tr>
<td>Difficulty swallowing (dysphagia)</td>
<td>1</td>
</tr>
<tr>
<td>Crossed motor and sensory loss</td>
<td>1</td>
</tr>
</tbody>
</table>

* Percentage of 184 - the proportion of patients with TIA with various focal neurological symptoms from the Oxfordshire Community Stroke Project; many patients had more than one symptom (e.g. weakness as well as sensory loss) and no patient had isolated dysarthria, ataxia, vertigo, diplopia or dysphagia.

that has been damaged by ischaemia or haemorrhage, the extent of the damage, and the activities in which the patient is engaged at the time of the TIA or stroke. The latter is particularly relevant for patients with a TIA because the neurological symptoms during a brief episode of ischaemia can only reflect what the patient was doing at the time of the ischaemic event. As many hours of wakefulness are spent in an alert state with eyes open, a keen sensorium, an upright posture, and often speaking or reading, it is not surprising that most of the symptoms that patients with TIA experience are a loss of speech and/or a loss of motor, somatosensory and/or visual function on one side of the body. Other, more transient activities, such as swallowing and calculation, are less frequently reported.

5.4 How are focal neurological symptoms and signs elicited quickly and accurately?

- The key points to elicit about focal neurological symptoms are shown in Box 5.3.

- As patients may use different terms to describe a symptom, often because of differences in their language or culture, it is important to establish that your interpretation is the same as the patient’s. A common example is the interchangeable use of terms such as ‘heaviness’ and ‘numbness’ by patients to describe motor and sensory deficits. If you are unsure exactly what the patient means, ask the patient, ‘Try to
describe what you mean in another way’, or paraphrase their description by asking, ‘What do you mean by that?’.

■ When a patient complains of loss of vision in one eye, the visual loss may be monocular or a homonymous hemianopia. If the visual loss was transient, it is important to ask the patient whether they closed each eye in turn during the attack and, if so, whether they could be sure that the blindness involved only one eye, or a part of both eyes. If the visual loss is persistent (i.e. still present), it is crucial to test the visual fields. Although conventional confrontation methods can be difficult, if not impossible, in patients who are drowsy, dysphasic or cognitively impaired, other methods may help determine whether there is an abnormality, such as stimulating the patient to look in each direction by doing something ‘interesting’ in different fields of vision, for instance following the examiner’s face rather than a finger or a pen.

■ Papilloedema is very uncommon in acute stroke; if present, suspect other conditions such as a brain tumour, subdural haematoma and cerebral venous sinus thrombosis.

■ The best screening tests of motor function in the arm and leg are a ‘pronator’ drift of the outstretched arm from the horizontal with the eyes closed and rapid tapping of the foot against the examiner’s hand respectively, but neither test is very specific. The most sensitive clinical test of corticospinal function is probably impairment of fine finger movements (e.g. repetitively tapping the index finger and thumb together) or rapid hand movements.

■ The anatomical extent of the weakness and its functional consequences (i.e. can the patient grip, or walk?) are more important to assess than grading the severity using a motor scale.

■ Examination of the sensory system is a notoriously unreliable part of the neurological examination but sensory symptoms are usually quite accurate (particularly for pain and temperature, less so for

---

**BOX 5.3  Key points to elicit about focal neurological symptoms**

1. **Nature** - Was the deficit of the motor, somatosensory, visual and/or other system?
2. **Quality** - Was there a loss of function (e.g. weakness or numbness) or a gain of function (e.g. jerking, paraesthesiae)?
3. **Anatomical distribution** - For example, did the deficit involve the face, arm or leg, or the face, arm and leg?
4. **Onset** - Was it sudden, stuttering or gradual?
5. **Evolution** - For example, did the deficit recover, stabilise or progress?
proprioception), even when no deficit is identified on examination.

Irrespective of the motor and sensory deficit identified by examining the patient in bed, it is essential to see whether the patient can sit up, get off the bed and walk - provided there is no risk to the patient or clinician. Patients with no motor or sensory deficit on examination in the bed may still be unable to walk because of severe gait ataxia, or neglect; cerebellar signs may be missed if the patient’s gait is not tested. Indeed, cerebellar infarction may also be misdiagnosed as ‘labyrinthitis’, or even as upper gastrointestinal disease if nausea and vomiting (secondary to vertigo) are prominent.

Disorders of speech and language are not synonymous; some patients may have slurred speech with normal language function (i.e. a disorder of articulation; dyarthria), and others may have normal articulation (and even speech) with abnormal language function causing difficulty in reading and writing. Because dysphasia is a disturbance of understanding and/or expression of spoken and/or written language, it is important to recognise that reading and writing (as well as speech) are also important language functions that should be assessed. The assessment of language function may be difficult or impossible in those with severe deafness and/or confusion. In these patients, it is important to beware of diagnosing dysphasia, particularly if other symptoms and signs suggest isolated non-dominant hemisphere dysfunction.

Visual–spatial–perceptual dysfunction is often best detected by asking the patient to draw a clock, copy a drawing of intersecting pentagons, and observing how the patient responds to the environment and carries out tasks around the ward, such as interacting with others (e.g. neglecting the left hemispace), dressing (i.e. putting on a shirt and taking it off), eating their food, reading a newspaper and writing a sentence.

Clinicians assessing stroke patients should be able to identify cognitive disorders in general terms, without having to resort to detailed assessment batteries, in order to facilitate diagnosis, localisation of the lesion and basic management. Few cognitive functions are absolutely specific for a single area of the brain and few tests are absolutely specific for a single aspect of higher cerebral function. The terms short- and long-term memory are used loosely by clinicians and often rather differently by neuropsychologists. In patients with stroke, it may be easier to distinguish anterograde amnesia from retrograde amnesia, and to observe how the patient is responding to nursing and rehabilitation instructions (i.e. are they learning and carrying over new information from day to day?).
5.5 How sudden is the onset of focal neurological symptoms in patients with TIA and stroke?

Most symptoms of an ischaemic event in the brain arise suddenly and are maximal at onset, without intensification or spread. The onset is usually so abrupt that the patient can describe exactly what they were doing at the time of onset. Occasionally, the symptoms may worsen gradually or in a stepwise fashion, but nonetheless their onset is usually sudden. If the patient cannot recall the precise onset of the symptoms, but is quite aware of the symptoms, the diagnosis of TIA and stroke is in doubt.

5.6 Can anything precipitate a TIA or stroke?

The onset of symptoms of TIA and stroke is seldom associated with a precipitating event. Although the circadian variation in stroke onset (more strokes occur during the morning, especially between 0800 and 1000 hours, than later) suggests some form of predisposition, it remains unexplained. Nevertheless, it is important to ask the patient exactly when the symptoms began and what they were doing at the time. This is because symptoms of low flow to the brain or eye (‘haemodynamic’ TIA or stroke) can be precipitated by a change in posture, neck turning, exposure to bright or white light, a hot bath, a heavy meal (postprandial hypotension), exercise, sexual activity, hypotensive drugs, general anaesthesia, cardiac arrest or cardioversion, in people with severe carotid and vertebrobasilar occlusive disease and a compromised collateral cerebral and ocular circulation. These symptoms sometimes need to be distinguished from symptoms of hypoglycaemia (precipitated by a large carbohydrate meal) and seizures (provoked by exposure to bright flashing lights).

Vigorous physical activity and coitus have also been associated with haemorrhagic stroke, particularly subarachnoid haemorrhage (SAH). However, apart from isolated case reports, there is no evidence that vigorous physical activity, coitus or stress precipitate TIAs and ischaemic stroke.

Occasionally, the aura of a previously experienced and otherwise unremarkable attack of migraine with aura persists for days or longer, the computed tomogram (CT) is normal or shows infarction, and the cause is attributed to arterial occlusion that may be due to vasospasm. The simultaneous occurrence of a TIA/stroke and a migraine attack may be coincidental and due to chance (both conditions are common; the prevalence of migraine in the general population is about 10% and that of ischaemic cerebrovascular disease is about 0.8%); causal (migraine may predispose to cerebral ischaemia by leading to platelet activation, arteriolar constriction and dehydration, or cerebral ischaemia may trigger off a migraine attack); or a misdiagnosis (e.g. carotid or vertebral artery dissection may cause
headache and a neurological deficit due to thromboembolism that is misinterpreted as migraine); or a syndrome suggestive of stroke or migraine may be a manifestation of another disease such as mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (MELAS) or an arteriovenous malformation.

The last trimester of pregnancy and the puerperium is a time when otherwise healthy young women may be predisposed to stroke as a result of paradoxical embolism from the venous system of the legs or pelvis, intracranial haemorrhage due to eclampsia, a ruptured arteriovenous malformation, and intracranial venous sinus thrombosis.

5.8 How reliable and accurate is the clinical diagnosis of TIA by general practitioners?

The clinical diagnosis of TIA by general practitioners (GPs) is not very accurate. In the Oxfordshire Community Stroke Project (OCSP), 512 patients were referred by their GP or attending hospital doctor with a diagnosis of possible TIA of whom 317 (62%) were considered by the OCSP neurologists not to have had a TIA but to have suffered from something else (see Table 5.2)'1. In another study, 30% of the patients originally...
TABLE 5.2 Diagnosis of suspected TIA by general practitioners in the community

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients (n = 512)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA</td>
<td>195 (38%)</td>
</tr>
<tr>
<td>Not TIA</td>
<td>317 (62%)</td>
</tr>
<tr>
<td>Migraine</td>
<td>52 (10%)</td>
</tr>
<tr>
<td>Syncope</td>
<td>48 (9%)</td>
</tr>
<tr>
<td>Possible TIA*</td>
<td>46 (9%)</td>
</tr>
<tr>
<td>‘Funny turn’†</td>
<td>45 (9%)</td>
</tr>
<tr>
<td>Isolated vertigo</td>
<td>33 (6%)</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>29 (6%)</td>
</tr>
<tr>
<td>Transient global amnesia</td>
<td>17 (3%)</td>
</tr>
<tr>
<td>Lone bilateral blindness*</td>
<td>14 (3%)</td>
</tr>
<tr>
<td>Isolated diplopia</td>
<td>4 (0.8%)</td>
</tr>
<tr>
<td>Drop attack#</td>
<td>3 (0.6%)</td>
</tr>
<tr>
<td>Intracranial meningioma</td>
<td>2 (0.4%)</td>
</tr>
<tr>
<td>Miscellaneous97</td>
<td>24 (5%)</td>
</tr>
</tbody>
</table>

* Possible TIA was diagnosed in patients in whom the clinical features were not sufficiently clear to make a diagnosis of definite TIA or anything else.
† ‘Funny turn’ was used to describe transient episodes of only non-focal symptoms not due to any identifiable condition (e.g. isolated and transient confusion).
‡ Lone bilateral blindness was later considered to be a TIA, after following these patients and noting their similar prognosis to patients with definite TIA.
# Sudden transient loss of postural tone causing a fall to the ground.
97 For example, hypoglycaemia, entrapment neuropathy, demyelination, subdural haematoma, psychogenic.

classified by their doctors as having TIAs were reclassified as not having TIAs when their records were reviewed by a stroke specialist. However, the accuracy of the diagnosis of TIA by GPs is being compared in these instances only with the diagnosis of TIA by a neurologist. So, how accurate is the diagnosis of TIA by a neurologist? (See Q 5.9.)

5.9 How reliable and accurate is the clinical diagnosis of TIA by neurologists?

The problem of accurately diagnosing TIA is not unique to GPs and hospital doctors. Experienced neurologists also show considerable interobserver variability in the diagnosis of TIA. Kraaijeveld et al investigated the interobserver agreement for the diagnosis of cerebral TIA amongst eight senior and interested neurologists from the same department who interviewed 56 patients with suspected TIA in alternating
pairs. The diagnosis was based on internationally accepted criteria. The agreement rates were corrected for chance (kappa statistic). Both neurologists agreed that 36 patients had a TIA and 12 had not, but they disagreed about eight patients (= 0.65; for perfect agreement = 1.0) (see Table 5.3). Therefore, the interobserver reliability of the diagnosis of TIA is not very good.

5.10 Why is the clinical diagnosis of TIA unreliable?

The clinical diagnosis of TIA is unreliable because there is no diagnostic test and the diagnosis depends entirely on the history - on an accurate:

■ recollection and communication of the symptoms by the patient
  (which is a difficulty if the patient delays going to the doctor after an attack or is forgetful - a particular problem in the elderly)

■ interpretation of the symptoms by the doctor (several studies have shown that clinicians differ in the interpretation of even isolated elements of the history\(^{2-4}\))

■ application of the symptoms to the diagnostic criteria for TIA (which lack detail).

The likelihood and accuracy of the diagnosis of a TIA increases with the abruptness of the onset of symptoms, the certainty that the neurological symptoms were focal, and the age and vascular risk factor profile of the patient. If the patient is young and free of vascular risk factors, the probability of a TIA is small.

5.11 What are the differential diagnoses of TIA of the brain?

**MIGRAINE AURA (WITH OR WITHOUT HEADACHE)**

■ young to middle-aged patients

■ positive symptoms (visual scintillations, tingling)
spread of symptoms to adjacent areas over minutes
■ symptoms resolve gradually and usually within 20–60 minutes
■ headache (often unilateral and pulsatile) and nausea usually accompany or follow the neurological symptoms
■ past or family history of migraine is common
■ vascular risk factors are uncommon
■ recurrences are usually stereotyped and may be reduced with migraine prophylaxis.

**PARTIAL (FOCAL) EPILEPTIC SEIZURES**
■ positive symptoms (e.g. limb jerking, tingling)
■ symptoms arise over seconds to 1–2 minutes (not abruptly)
■ symptoms spread or march to adjacent areas over several seconds
■ symptoms usually resolve quickly within a few minutes but can be prolonged for hours
■ antecedent partial seizure symptoms may be present (e.g. epigastric discomfort, nausea)
■ impaired awareness (i.e. complex partial seizure) or secondary generalisation with a tonic-clonic convulsion or loss of consciousness may occur
■ persistent focal neurological signs may be present after symptoms resolve
■ recurrences are usually stereotyped and respond to antiepileptic drugs.

**TRANSIENT GLOBAL AMNESIA**
■ abrupt onset of loss of anterograde episodic memory for verbal and non-verbal material
■ usually accompanied by repetitive questioning
■ resolves within 24 hours (and usually a few hours) leaving a dense amnesic gap for the duration of the attack
■ no clouding of consciousness, loss of personal identity or ability to recognise familiar individuals or places, other focal neurological symptoms, or epileptic features
■ recurrent attacks are exceptional
■ the diagnosis is all but impossible if there is no witness available.

**LABYRINTHINE DISORDERS**
(benign recurrent vertigo, benign paroxysmal positional vertigo, acute labyrinthitis)
■ vertigo is the only neurological symptom (with secondary nausea and ataxia).
**METABOLIC DISORDERS**
(hypoglycaemia, hyperglycaemia, hypercalcaemia, hyponatraemia)
- hypoglycaemic attacks may recur at regular times and can be excluded with appropriately timed tests of blood glucose levels.

**HYPERVENTILATION, ANXIETY OR PANIC ATTACKS, SOMATISATION DISORDER**
- consider reproducing the symptoms (e.g. forced hyperventilation).

**INTRACRANIAL STRUCTURAL LESION**
(meningioma, tumour, giant aneurysm, arteriovenous malformation, chronic subdural haematoma)
- usually cause recurrent stereotyped events; exclude with CT or MRI of the brain.

**ACUTE DEMYELINATION (MULTIPLE SCLEROSIS)**
- usually subacute onset in young adults; exclude with MRI of the brain.

**SYNCOPE**
- a non-focal neurological symptom
- often a precipitating circumstance.

**DROP ATTACKS**
- usually in middle-aged women
- onset when standing or walking
- legs give way and patient falls to the ground with otherwise preserved neurological function and consciousness throughout
- recovery is immediate unless the patient is injured.

**MONONEUROPATHY/RADICULOPATHY**
- lower motor neuron signs.

**MYASTHENIA GRAVIS**
- fatiguability.

**CATAPLEXY**
- brief muscle weakness precipitated by excitement or emotion (e.g. laughter).
5.12 What are the differential diagnoses of TIA of the eye (amaurosis fugax)?

**RETINAL DYSFUNCTION**

**Vascular**

- **Retinal migraine** - gradual ‘build-up’ of transient monocular visual impairment that is usually incomplete and associated with ‘positive’ visual symptoms (e.g. scintillations) lasting up to 1 hour. A pulsatile headache or orbital pain may coexist.

- **Anterior-middle cranial fossa dural arteriovenous malformation** - may rarely cause transient monocular blindness (TMB), probably because of transient lowering of retinal arterial pressure associated with shunting of blood away from the ophthalmic artery to the malformation.

- **Central or branch retinal vein thrombosis** - may present with attacks of TMB but the fundoscopic appearance is characteristic, with engorged retinal veins and multiple retinal haemorrhages (see Fig. 5.1).

  ![Fig 5.1 Fundus of a patient with central retinal vein thrombosis showing the characteristic engorged retinal veins and multiple retinal haemorrhages.](image)

- **Retinal haemorrhage** - if small or located in the periphery of the retina may cause sudden loss of vision in one eye.

**Non-vascular**

- **Paraneoplastic retinopathy** - painless, brief (seconds to minutes) episodes of monocular dimming of the central field of vision, and overwhelming visual glare and photosensitivity when exposed to bright light.

- Ophthalmoscopy usually reveals an attenuated calibre of retinal arterioles; electroretinography demonstrates abnormal cone- and rod-mediated responses; and antiretinal antibodies may be present in the serum. Progressive visual loss evolves, during which time a small-cell carcinoma of the lung often declares itself.

- **Phosphenes** - flashes of light and coloured spots which are induced by eye movement in a dark environment and occur in the absence of luminous stimuli. They may occur following mechanical pressure on
the normal eyeball (stimulating the retina), in a healthy dark-adapted closed eye after a voluntary eye movement/saccade (flick phosphenes), or with disease of the visual system at any site (e.g. recovery phase of optic neuritis).

- **Lightning streaks of Moore** - brief, recurrent, stereotyped vertical flashes of light in the temporal visual field of one eye elicited by eye movement. Common in elderly people when in a dark environment. They are a photopsia (subjective sensation of sparks or flashes of light), which may be caused by collapse of the posterior vitreous with its detachment from the retina (with age), and triggered by the transient mechanical forces of eye movement. Benign.

- **Chorioretinitis.**

## OPTIC NERVE DISORDERS

### Vascular

- **Anterior ischaemic optic neuropathy** due to arteritis (e.g. giant cell), atherothrombosis or malignant arterial hypertension - may cause abrupt onset of TMB, but usually the visual disturbance is in the form of an altitudinal field defect (loss of either the upper or lower half of the field of one eye) rather than as if a ‘curtain has descended or ascended over the whole eye’, because the optic nerve is supplied by an upper and lower division of the posterior ciliary artery. Shortly after the onset of blindness, fundoscopy may reveal distended veins, a swollen optic disc (or part of the disc), variable pallor of the disc, flame-shaped haemorrhages at or near the disc and, occasionally, cotton wool spots (see Fig. 5.2). Systemic upset, tender temporal arteries, and a raised erythrocyte sedimentation rate (ESR) are clues to arteritis, and an increased blood pressure and ophthalmoscopic features of retinal arteriolar disease, optic disc oedema and retinal haemorrhages point to the diagnosis of malignant hypertension.

![Fig 5.2 Fundus of a patient with anterior ischaemic optic neuropathy showing swelling and variable pallor of part of the optic disc, flame-shaped haemorrhages near the disc, distended veins and cotton-wool spots.](image-url)
Non-vascular

- **Papilloedema** (see Fig. 5.3) - patients with papilloedema from any cause may experience transient visual blurring or obscurations, with or without photopsia. The visual loss in chronic papilloedema is often postural, occurring as patients rise from bed or chair, and may involve either eye alone or both eyes together. The explanation may be transient optic nerve ischaemia secondary to a relative decrease in orbital blood flow secondary to the raised cerebrospinal fluid (CSF) pressure in the subarachnoid space around the optic nerve with an increase in pressure in the veins draining the optic nerve head.

- **Optic neuritis and Uhthoff’s phenomenon** - patients with optic nerve demyelination (most commonly due to multiple sclerosis) may experience transiently decreased vision in one or both eyes, usually after exercise or exposure to heat (Uhthoff’s phenomenon).

- **Dysplastic coloboma.**

**EYE AND ORBIT**

Transient changes in the ocular media or intraocular pressure can cause transient monocular visual disturbance, but the most common causes (listed below) can usually be excluded from the ophthalmological examination.

- Anterior (aqueous humour) chamber and posterior chamber (vitreous) haemorrhage.

- Raised intraocular pressure (glaucoma): may cause transient monocular visual impairment, but usually associated with recurrent attacks of pain in the eye and forehead, which may be precipitated by sitting in the dark, mydriatics or emotional upset. Other features include cloudiness of the cornea, discoloration of the iris, a dilated pupil and circumcorneal injection. An arcuate scotoma and pallid cupped disc are characteristic of narrow-angle glaucoma, which is often familial. Tonometry is
necessary to confirm raised intraocular pressure.

- Reversible diabetic cataract
- Lens subluxation
- Orbital tumour (e.g. optic nerve sheath meningioma): may cause gaze-evoked loss of vision, but the time course of the blindness is limited to the duration of gaze in the affected direction; the visual acuity usually returns to normal within about 30 seconds of the eye returning to the primary position of gaze. The loss of vision possibly relates to compression of blood vessels surrounding or supplying the optic nerve.

### 5.13 What is the role of imaging the brain by CT in the differential diagnosis of TIA?

The main purpose of cranial CT or MRI in patients with suspected TIA is to detect an underlying structural intracranial lesion, such as arteriovenous malformation, meningioma, subdural haematoma (see Fig. 5.4), which may present like a TIA. It is not to detect low-density lesions (presumed infarcts) or to exclude primary intracerebral haemorrhage (PICH), as in patients with stroke (see Q 5.23), because definite PICH only very rarely causes focal neurological symptoms lasting less than 24 hours.

![Fig 5.4 Plain cranial CT scan showing a rim of high signal adjacent to the right temporal and occipital cortex due to a subdural haematoma.](image)
5.14 What is the yield of CT brain imaging in patients with suspected TIA?

Limited data indicate that the yield of CT for detecting structural lesions is about 1% in patients with suspected TIA.\(^5\) With such a low yield, routine CT imaging of every patient who has had a single TIA must be considered very carefully. The small minority of ‘TIA’ patients with structural intracranial lesions who will be missed by not performing CT will probably continue to have symptoms (and so return to the doctor) and their outcome is unlikely to be altered by a short delay in diagnosis.

It seems that the small yield of structural brain lesions from CT is almost always in patients with carotid territory ‘TIAs’ and there is some evidence that performing CT in patients with vertebrobasilar territory TIAs and transient monocular blindness is a waste of resources.\(^6\)

Data on the cost-effectiveness of CT and MRI in patients with TIA are very few and there is a need for a methodologically sound, prospective, multicentre study of this question, particularly in view of the considerable cost implications of a policy of ‘CT or MRI for all suspected TIA patients’.

5.15. Which patients with suspected TIA should have CT brain imaging?

Brain imaging by CT should probably be reserved for patients:
- with more than one TIA of the brain, particularly if they are in the carotid territory
- being considered for carotid endarterectomy (to avoid operating on someone with a symptomatic meningioma, for example).\(^7\)

5.16 Should patients with TIA have a plain or a contrast CT brain scan?

CT for patients with a suspected TIA should initially be a non-contrast study.

5.17. When is a contrast CT or MRI brain scan indicated in patients with a TIA?

Contrast CT or MRI and MR angiography is indicated if brain tumour, giant aneurysm or arteriovenous malformation (AVM) is suspected as the cause of transient focal neurological symptoms (see Fig. 5.5). MRI is also indicated for patients who continue to have suspected vertebrobasilar TIAs despite optimal medical therapy, and in whom cranial CT is unhelpful and a structural abnormality in the posterior fossa is still suspected. Although more expensive, MRI of the posterior fossa (and also the cerebral
hemispheres) is superior to CT for detecting infarcts and, of more relevance for management, for demyelinating and structural lesions. However, even MRI is not 100% sensitive in detecting brainstem infarcts.

5.18 What is the role of EEG in patients with suspected TIA?

Electroencephalography (EEG) is indicated in patients with suspected TIA when the clinical diagnosis of TIA is in doubt and partial (focal or localisation related) seizures are a possibility (e.g. transient positive neurological symptoms that progress with or without loss of consciousness). About 35% of all patients with epilepsy consistently have epileptiform discharges on the waking interictal EEG, 50% do so on some occasions with repeated recording sleep-deprived recordings, and about 15% never do. These figures vary according to the type of epilepsy; amongst patients who present with a first seizure, epileptiform abnormalities on the EEG are present in a higher proportion with idiopathic generalised seizures than with partial seizures.

The results of EEG should be interpreted cautiously. Besides the modest sensitivity of EEG in distinguishing epileptic seizures from non-epileptic events such as TIA (i.e. only a modest proportion of patients with epilepsy have an abnormal EEG), further difficulty arises as a result of the poor specificity of EEG abnormalities (all too frequently patients with non-epileptic events such as TIA are reported to have an abnormal EEG).
DIAGNOSIS OF STROKE

5.19 How is the diagnosis of stroke made?

Symptoms and signs in the diagnosis of stroke

Like the diagnosis of TIA, the diagnosis of stroke is also clinical and depends crucially on an accurate history, taken from the patient, carer or witness. To decide whether the symptoms and signs are due to a vascular event of the brain, ensure that:

- The neurological symptoms and signs are focal (i.e. neuroanatomically localising) rather than non-focal
- The focal neurological symptoms are negative in quality (i.e. loss of function) rather than positive (i.e. muscle paralysis rather than jerking, numbness rather than pins and needles, blindness rather than visual hallucinations)
- The onset of the focal neurological symptoms was sudden
- The focal neurological symptoms were maximal at onset (i.e. evolving over minutes in all of the affected body parts) rather than progressive (evolving over hours to days, and migrating from one body part to another).

Useful websites can be found at the following locations:

http://www.strokeaha.org
http://www.dcn.ed.ac.uk/spgm

If all these criteria are met, the likelihood of a vascular disturbance (ischaemia or haemorrhage) of brain function is high. The likelihood is even greater if the ‘milieu’ is appropriate (e.g. an elderly patient with prolonged exposure to several vascular risk factors). About 80% of stroke patients have at least one vascular risk factor, and most are elderly; stroke is uncommon (but not that rare) in young people.

The exception to these criteria is the minority of patients with SAH who present with headache but have no focal neurological symptoms or signs (neck stiffness is not invariable and may not occur for several hours). As the clinical features of SAH may differ substantially from those of intracerebral haemorrhage and infarction, they are discussed separately (see Q 5.48).

5.20 How accurate is the clinical diagnosis of stroke?

The clinical diagnosis of stroke is accurate about 80–85% of the time, depending on the time since stroke onset and the experience of the examiner.9,10
5.21 **What are the common pitfalls in the clinical diagnosis of stroke?**

The clinical diagnosis of stroke is often most difficult in the hyperacute phase (e.g. within 6 hours of onset) when symptoms and signs may change rapidly. This and other difficulties in the diagnosis are shown in *Box 5.4.*

**Box 5.4 Pitfalls in the diagnosis of stroke**

- within 6 hours of onset (i.e. in the hyperacute phase)
- onset of symptoms is uncertain (e.g. because of coma, dysphasia, confusion, no witness)
- neuroanatomical localising value of the symptoms is uncertain (e.g. confusion, amnesia, coma)
- symptoms are positive in nature (e.g. movement disorders such as hemiballismus caused by lesions of the subthalamic nucleus of Luys in the midbrain)
- symptoms are progressing (rather than recovering) over hours or even days
- patient does not recognise that there is a problem at all (e.g. patients with isolated visual–spatial–perceptual dysfunction such as hemispatial neglect or geographical disorientation - the so-called ‘inobvious stroke’).

In these situations the clinical diagnosis of stroke is less certain and further investigations are usually required quite urgently to exclude alternative diagnoses that may require different and immediate treatment (e.g. hypoglycaemia, non-convulsive epileptic seizures, brain infection, subdural haematoma) *(see Q 5.22).*

5.22 **What are the differential diagnoses of stroke?**

**Differential diagnosis of stroke (in order of frequency of occurrence in general practice)**

- Metabolic/toxic encephalopathy (hypoglycaemia, non-ketotic hyperglycaemia, hyponatraemia, Wernicke-Korsakoff syndrome, hepatic encephalopathy, alcohol and drug intoxication)
- Functional/non-neurological (e.g. hysteria)
- Epileptic seizure (postictal Todd’s paresis) or non-convulsive seizures
- Hemiplegic migraine
- Structural intracranial lesion (e.g. subdural haematoma, brain tumour, arteriovenous malformation)
5.23 What is the role of imaging the brain by CT in the diagnosis of stroke (versus not stroke)?

1. To exclude non-vascular intracranial pathology (e.g. subdural haematoma, brain tumour) as the cause of the focal neurological symptoms and signs.
2. To confirm the presence of recent cerebral infarction, or intracerebral or SAH which is relevant to the clinical presentation.

5.24 What is the yield of early CT of the brain in identifying a relevant stroke lesion?

Early CT imaging of the brain, done within the first few days of stroke, identifies intracerebral haemorrhage in all cases, SAH in about 95-97% and cerebral infarction in about two-thirds. The lower yield for cerebral infarction is because:

- CT may have been done early
- the infarct is too small to be imaged in this way
- the infarct is obscured by artefact (particularly in the posterior fossa)
- the resolution of the CT is not very good.

However, even within 5 hours of symptom onset, CT will show abnormalities of cerebral infarction in about 50% of cases.11

5.25 Does a normal CT scan mean that the patient has not had a stroke?

No. The absence of a visible infarct on CT does not mean the patient has not had a stroke. A patient with a clinical diagnosis of stroke and an early CT brain scan that is either normal or shows a relevant hypodense lesion, consistent with infarction (see Qs 5.26-5.30), is classified as having an ischaemic stroke.

5.26 What do primary intracerebral haemorrhage and subarachnoid haemorrhage look like on CT?

Intracerebral and subarachnoid blood appear immediately as a white area...
5.27 Does the appearance of the haemorrhage evolve with time on CT brain scan?

Yes. With time, the CT appearance of a white, hyperdense area of haemorrhage becomes less dense and, after a few days to a few weeks (depending on its size), it becomes isodense with surrounding brain tissue (‘fogging’) and may be difficult to see. It is the smaller haemorrhages that may become isodense within days. Thereafter the haemorrhage becomes hypodense and may be mistaken for an old infarct (see Fig 5.8). This is one of the main reasons why CT must be done early, within the first few days of stroke: to identify intracerebral haemorrhage reliably if it has occurred.

5.28 What does a very recent cerebral infarct look like on CT brain imaging?

In the hyperacute stage of an ischaemic stroke (i.e. within the first few
hours), the CT image often appears normal. However, within 3 hours of onset of middle cerebral artery territory infarction there are usually subtle changes in the ischaemic brain parenchyma which are easily overlooked by the inexperienced or untrained observer. These include loss of outline (obscuration) of the lentiform nucleus and loss of visualisation of the insular ribbon, due to loss of normal grey-white matter differentiation (at the basal ganglia-white matter and cortex-white matter interfaces) (see Fig. 5.9). Other features include effacement of the overlying cortical sulci and

**Fig 5.8** Plain cranial CT scan of the patient in Fig 5.6, performed 14 days after the initial CT scan, now showing only a small area of low intensity, which looks like an infarction but actually represents resolution of haemorrhage, in the right posterior putamen. A follow-up scan was performed because the original tomogram was missing, and the radiologist described the lesion as an infarct in the right posterior putamen. This emphasises the importance of performing CT early, within the first week (and preferably within the first hours to days), in patients with suspected stroke in order to document the pathology clearly and optimise diagnosis and treatment.

**Fig 5.9** Plain cranial CT scan performed 3 hours after the onset of acute right frontal lobe ischaemic stroke showing loss of outline (obscuration) of the right lentiform nucleus, loss of visualisation of the insular ribbon, effacement of the overlying cortical sulci and compression of the lateral ventricle due to focal brain swelling.
Therefore, the hyperdense artery sign is probably a reasonably reliable indicator of an occluded cerebral artery when the hyperdensity is visible at a distance from the carotid siphon, for example in the proximal middle cerebral artery (MCA) or its branches, and particularly in younger patients. An absent sign is certainly not a reliable indicator of a patent artery.
5.30. Does the appearance of a cerebral infarct evolve with time on CT?

Yes. As the infarct ‘progresses’ with time during the first few days, it becomes more clearly demarcated and more hypodense (black) and well defined. The ischaemic white matter becomes hypodense with respect to normal white matter, and the abnormal grey matter becomes even more hypodense so that overall the whole lesion appears darker than the surrounding brain.¹⁸

Swelling of the infarct is usually maximal around days 3–5 and this gradually subsides during the second and third week. However, occasionally infarct swelling can occur very rapidly, within the first 24 hours, to cause brain herniation, but generally only with very extensive infarcts. The amount of infarct swelling and the rate at which it appears vary between patients, for reasons that are not well understood.

Small infarcts probably appear later than large ones, because there is less tissue altering its density, so that lacunar infarcts are less likely to show up in the first 24 hours and sometimes do not do so at all. Small infarcts in the brainstem and cerebellum are particularly difficult to visualise with CT because of artefacts arising from the petrous bones; this is probably less problematic with modern scanning technology and thinner scan sections.¹⁹

5.31 What is ‘fogging’ on CT brain images?

The so-called ‘fogging effect’ usually occurs during the second week after stroke onset, when the hypodense infarct on CT gradually increases in density, sometimes becoming isodense.²⁰ The infarct is then indistinguishable from normal brain and may be overlooked at this time.²¹ It is less pronounced in large infarcts, but may lead to underestimation of infarct size. It does not occur in all infarcts and the timing of occurrence after stroke onset may vary. The ‘fogging effect’ may last for up to 2 weeks and then the infarct becomes progressively more hypodense.

5.32 What does an old cerebral infarct look like on CT?

Eventually a sharply demarcated, atrophic, hypodense (similar to CSF) defect remains. Old brain infarcts are usually atrophic, very hypodense (similar to CSF) and sharply demarcated from surrounding normal brain (see Fig 5.11).

Although these features generally make it possible to ‘age’ infarcts, it is not always possible to tell with absolute certainty how old an infarct is. This is important to consider when trying to ascribe particular clinical symptoms to lesions seen on CT.
5.33 What features of a low-density lesion on CT distinguish arterial infarcts from other pathologies?

Brain infarcts caused by arterial occlusion are usually easy to diagnose from their site, shape and density, in association with appropriate clinical features. However, other pathologies can occasionally produce similar appearances, which may be confusing.

VENOUS INFARCTS

Venous infarcts, although uncommon, are frequently misdiagnosed as arterial infarcts, intracerebral haemorrhages or tumours on CT. They are typically of low density and may be wedge-shaped, like arterial infarcts, but the key differentiating features are:

- they often do not quite fit the usual site of an arterial infarct
- they are much more swollen for their size than an equivalent-sized arterial infarct
- there may be swelling in the hemisphere beyond the low-density area
- they often contain haemorrhage.

The haemorrhage is typically in the centre of the low-density area and may be patchy and finger-like in distribution, whereas in arterial infarcts the haemorrhage is usually around the edges. The high density of a thrombosed cortical vein or sinus may also be visible (hyperdense artery
sign). After intravenous contrast, there may be an ‘empty delta’ sign in the venous sinus and serpiginous enhancement at the edges of the infarct (see Fig. 5.12).

**VIRAL ENCEPHALITIS**

Viral encephalitis due to herpes simplex virus is usually focal and typically involves the medial temporal lobes. However, it is sometimes more widespread, affecting the frontal and temporal lobes, or temporal-parietal lobes, and can look exactly like an infarct (see Fig. 5.13). In these cases the clues to encephalitis are usually associated clinical features of subacute (as opposed to acute) onset, fever and progression (if not treated with aciclovir) rather than improvement. It is essential that the CT scan is reviewed with all the clinical information and, if in doubt, other diagnostic tests are done, such as EEG or MRI.

**PURULENT CEREBRITIS**

Purulent cerebritis can look like an infarct, although the lesion is usually not wedge-shaped and involves more white matter than cortex; the clinical picture should allow the distinction to be made.

**TUMOURS**

Occasionally, a metastasis to the cerebral cortex may mimic an infarct on
CT, particularly if there is a lot of white matter oedema. However, metastases are lower in density than an infarct, and administration of intravenous contrast may reveal an enhancing cortical nodule (see Fig. 5.5). If there is still doubt, repeat the CT scan a few weeks later because infarcts usually get smaller whereas tumours usually stay the same size or increase in size.

5.34 Does intravenous contrast help?

The only indication for performing CT with intravenous contrast in a patient with suspected stroke is when the plain (non-contrast) CT scan, and clinical features, are suggestive of a structural intracranial lesion mimicking a stroke, such as a brain tumour, abscess or AVM. These lesions enhance with contrast (see Fig. 5.5).

If the patient has had an ischaemic stroke, the contrast usually has little effect on the appearance of the infarct in the first week after onset. However, in the second and third weeks more striking contrast enhancement occurs, frequently corresponding with the time of maximal blood–brain barrier breakdown and positivity of radioisotope scans. The mechanism is probably a combination of blood–brain barrier breakdown, neovascularisation and impaired autoregulation, and the resulting appearance on CT is referred to as ‘luxury perfusion’. The tendency to enhance with contrast gradually resolves over the following few weeks.
5.35 What does haemorrhagic transformation of the infarct look like on CT brain scan?

Recent haemorrhage in the infarct produces areas of increased density relative to both normal brain and infarcted tissue, and presumably contributes to any swelling (see Fig. 5.14).

5.36 Should all patients with suspected stroke have CT imaging?

Yes, if excluding a relevant non-vascular intracranial pathology and identifying the pathological cause of the stroke is likely to influence management (e.g. by clarifying aetiology, prognosis or treatment). This is almost invariably the case, unless the patient is terminally ill and requiring full nursing care before the onset of suspected stroke; optimising the diagnosis improves patient management.23

5.37 Are there any advantages of MRI over CT brain imaging in the diagnosis of stroke?

MRI of the brain is more sensitive than CT for detecting cerebral infarction, particularly small deep infarcts (e.g. lacunar infarcts) and those occurring in the posterior fossa. Furthermore, certain MRI techniques, such as diffusion weighted imaging (DWI), are very sensitive at highlighting the recent ischaemic lesion and may be particularly helpful when conventional MRI (e.g. T2-weighted imaging) shows several areas of abnormality. However, even MRI can be normal in clinically definite stroke.24
5.38 When is an MRI scan of the brain preferable to CT in patients with suspected stroke?

The choice of CT or MRI will depend on local availability, cost and effectiveness. If CT is available, it should be performed as soon as possible in all patients because it is the best technique for diagnosing or excluding early intracerebral haemorrhage and is essential to image suspected SAH; MRI is no substitute and can fail to image SAH. MRI will more often confirm the site of cerebral infarction suspected clinically but, as yet, this is rarely necessary; the immediate priority is to exclude intracerebral haemorrhage. So MRI is not necessarily better than CT. Furthermore, CT is an excellent technique for ill, confused patients - as many stroke patients are - and MRI is a more difficult technique to apply. Circumstances in which brain MRI is preferable to CT are shown in Box 5.5.

Box 5.5 Circumstances where MRI is preferable to CT

- When more than 10 days has elapsed since stroke onset, CT shows a low-density area that could have been infarction or resolving haemorrhage and it is essential to know whether the stroke was ischaemic or haemorrhagic
- When CT is negative and it is crucial to be able to localise the infarct (this happens only very occasionally); MRI is more likely to image the lesion
- Arterial dissection is a suspected cause of cerebral infarction.

5.39 What does a recent cerebral infarct look like on MRI (Fig 5.15)?

The earliest changes of cerebral ischaemia detected by routine MRI (i.e. not with spectroscopy, perfusion or diffusion imaging) at various times after stroke onset are:

- **Minutes**: loss of the normal flow void in the symptomatic artery within minutes of onset (the MR equivalent of the hyperdense artery sign on CT)
- **3 hours**: swelling of the ischaemic brain on T1-weighted images, but without signal change on T2-weighted images
- **8 hours**: signal changes on T2-weighted images
- **16 hours**: signal change on T1-weighted images.

Large infarcts are often visible on routine T1- and T2-weighted imaging within 6 hours, but small cortical and subcortical infarcts may never become visible. Infarcts of any size are more often and more quickly visible...
on fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI), but currently these are not in widespread routine use.

5.40 Does the appearance of infarction on MRI evolve with time?

As with CT, MRI also shows that the infarct swells.

In the second week after infarct onset, MRI T2-weighted images often show a diffuse increase in signal (brightness) of gyri overlying the infarct, probably as a result of neovascular capillary proliferation or loss of autoregulation in leptomeningeal collaterals. It remains visible for up to 8 weeks after onset. A similar appearance is seen on CT, attributed to areas of breakdown of the blood–brain barrier, and corresponds to the gyriform petechial haemorrhages seen at autopsy.

Infarcts do not usually show enhancement with intravenous contrast until the second week after the stroke. Thereafter they generally show marked contrast enhancement around the edges (and within the infarct if sufficient contrast is administered) for several weeks after the stroke.

5.41 Is there a fogging effect on MRI (as there is with CT)?

Yes, probably. In the second to third week after onset, some infarcts increase in signal on T1-weighted MRI, decrease in signal on T2-weighted images, and become isodense with normal brain on MRI (as they may do on CT - the ‘fogging’ effect). As the infarcts may have lost most of their mass effect at this stage, they may be difficult to identify.
5.42 **What is the cause of the fogging effect on MRI?**

It is probably due to diffuse petechial haemorrhage from leaky capillaries, with diapedesis of red blood cells. On CT, the red blood cells cause a diffuse increase in Hounsfield numbers, raising the low density of the lesion to that of normal brain parenchyma.

5.43 **What are the late appearances of infarction on MRI?**

After several weeks, the infarcted brain appears as an area with similar signal characteristics to CSF, i.e. bright on T2 and dark on T1, with an ex vacuo effect on the surrounding brain. Other long-term effects of ischaemic stroke seen on MRI include wallerian degeneration, visible as atrophy and low intensity in the white matter of the brainstem, and the late effects of any haemorrhagic transformation.

5.44 **Can MRI differentiate new from old infarcts?**

Yes. Diffusion-weighted imaging (see Q 5.45) is an MRI technique that shows recent infarcts as an area of increased signal for up to several weeks after the stroke (see Fig. 5.16).

5.45 **What is diffusion-weighted MRI?**

DWI signals reflect the mobility of water molecules within tissues (e.g. brownian motion). In ischaemic tissues, energy failure leads to impairment of cell membrane function (e.g. the sodium-potassium ATP pump), which results in movement of water into the cell (cytotoxic oedema).
Intracellular water restricts diffusion and produces high signal on DWI images.

The major potential advantage of DWI in acute ischaemic stroke is the rapid appearance of abnormal signal soon after onset of blood flow impairment. In animal models, affected cerebral tissues exhibit high signal within 14 minutes of vessel occlusion. In stroke patients, initial studies have shown alteration of water diffusibility in the suspected infarcted tissue, which varied both within the lesion and with time.31

5.46 What is perfusion-weighted MRI?

Perfusion-weighted MRI aims to measure the patency of, and degree of blood flow through, the cerebral microcirculation. This can be achieved by examining the magnetic properties of flowing blood (‘arterial spin tagging’) or a bolus of contrast agent, such as gadolinium, which has been injected intravenously.

5.47 What is the role of EEG in the differential diagnosis of stroke?

In patients with suspected stroke EEG helps to determine whether there is a seizure focus when the clinical diagnosis is in doubt (e.g. in those with suspected postictal paresis or suspected non-convulsive status epilepticus who may present with the sudden onset of a confusional state) and the CT scan is normal or shows a lesion that is not typical of an infarct or PICH, and if there is any suspicion of Creutzfeldt-Jakob disease or herpes encephalitis (e.g. clinical deterioration with new neurological signs, particularly myoclonus and cognitive decline, or hemiparesis, dysphasia and fever).

The EEG is not useful for diagnosing stroke: stroke is a clinical diagnosis and has no specific EEG features.32

The usual EEG findings in acute stroke are a localised reduction of normal cortical rhythms and a major surrounding slow-wave abnormality with individual waves of less than 1 Hz. Focal EEG slowing, however, is not specific; it indicates only the presence and side of the lesion. Although the EEG may help distinguish between small deep (lacunar) and cortical infarction, the clinical features and brain CT or MRI are more effective tools for doing this.33,34
5.48 What are the clinical features of subarachnoid haemorrhage?

Clinical features of subarachnoid haemorrhage

- Headache is the cardinal clinical feature of SAH and is the only symptom in about one-third of patients. It usually arises suddenly, 'like a blow on the head' or 'an explosion inside the head', reaching a maximum within seconds. It is the most severe headache the patient has ever had, and is initially generally diffuse and poorly localised. However, after minutes to hours the headache spreads to the back of the head, neck and back as blood tracks down into the spinal subarachnoid space. Previous similar headaches, due to presumed previous, unrecognised 'warning leaks' are most uncommon
- Nausea and vomiting: common at the outset
- Photophobia
- Neck stiffness: takes 3–12 hours to develop
- Brudzinski's sign - forward flexion of the neck evokes flexion at the hip and knee
- Limitation of straight leg raising
- Kernig's sign - passive extension of the knee with the hip flexed elicits pain in the back and leg and resistance to hamstring stretch
- Altered consciousness - at least 60% of patients
- Intraocular preretinal subhyaloid haemorrhage, usually near the optic disc (see Fig. 5.17)
- Focal neurological signs - oculomotor (IIIrd cranial) nerve palsy suggests posterior communicating artery aneurysm; dysphasia and hemiparesis suggest intracerebral extension of the SAH
- Fever - but the pulse rate remains disproportionately low (compared with fever due to infection in which the pulse rate also rises)
- Epileptic seizures - about 10% of patients.

5.49 What are the differential diagnoses of SAH?

The sudden onset of a severe headache may be due to several other conditions, such as ‘thunderclap headache’, cerebellar or intraventricular haemorrhage, benign orgasmic or exertional cephalalgia, and migraine, which may sometimes start suddenly rather than gradually (see Box 5.6).

Although most (75%) individuals with a sudden severe headache have not had a SAH, they must all be investigated to exclude it. Not uncommonly, the history of onset of headache is unclear, and the differential diagnosis is even wider.
### Box 5.6 Differential diagnosis of sudden unexpected headache

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<thead>
<tr>
<th>With neck rigidity</th>
<th>Without neck rigidity</th>
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<tbody>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>Migraine</td>
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<tr>
<td>Acute painful neck conditions</td>
<td>Thunderclap headache</td>
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<tr>
<td>Meningitis/encephalitis</td>
<td>Benign orgasitic cephalalgia</td>
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<tr>
<td>Cerebellar stroke</td>
<td>Benign exertional headache</td>
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<td>Intraventricular haemorrhage</td>
<td>Pituitary apoplexy</td>
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<tr>
<td>Pituitary apoplexy</td>
<td>Reaction whilst on monoamine oxidase inhibitors</td>
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<td>Recent head injury</td>
<td>Phaeochromocytoma</td>
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<td>Expanding intracranial aneurysm</td>
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<td>Acute obstructive hydrocephalus</td>
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### 5.50 How is the diagnosis of SAH made?

The diagnosis of SAH is frequently suspected from the clinical history and examination, but requires confirmation by CT (see Fig. 5.7), which is positive in at least 95% of cases when performed within 1–2 days of onset.

If SAH is suspected but the CT scan appears normal, it is important to look carefully at the interpeduncular cistern, ambient cisterns, quadrigeminal cistern, the region of the anterior communicating artery and posterior inferior cerebellar artery, the posterior horns of the lateral
ventricles and the cortical sulci (see Fig. 5.18). If blood is present in these sites, it may be isodense or slightly hyperdense, and hence the normally hypodense cisterns and sulci may be difficult to see and seem ‘absent’.

If the early CT scan is still considered ‘negative’, as it will be in 3–5% of cases of SAH, a lumbar puncture is required to identify the characteristic yellowish colour (xanthochromia) of the centrifuged CSF due to the breakdown products of haemoglobin (oxyhaemoglobin and bilirubin).

### 5.51. When should lumbar puncture be performed in patients with suspected SAH and negative CT findings?

Lumbar puncture should be done only after at least 12 hours have elapsed since the onset of headache to enable a reliable distinction to be made between a traumatic tap and haemorrhage in the subarachnoid space. This is because it takes up to 12 hours for red cells to lyse and haemoglobin to be broken down to oxyhaemoglobin and bilirubin, which after centrifugation of the CSF results in a yellowish colour of the supernatant (xanthochromia), and on spectrophotometry can be recognised by characteristic absorption bands. Because xanthochromia is the only reliable proof that haemorrhagic spinal fluid has not resulted from the trauma of the puncture itself, it is crucial that the CSF is not examined within 12 hours of onset of headache; no xanthochromia within 12 hours
5.52 What are the common pitfalls in the diagnosis of SAH?

Because SAH is uncommon, with an annual incidence of six per 100 000, GPs encounter it only once in every 8 years on average, and therefore rarely think of it. When they do, it is usually in a patient with sudden onset of severe headache (because this is the cardinal clinical feature of SAH), but only one in eight patients whose only symptom is sudden severe headache will have SAH; the other seven have mostly innocuous conditions such as migraine.

Other reasons for misdiagnosis of SAH include:

- Failure to appreciate the spectrum of presentations of SAH - the diagnosis has to be considered with atypical presentations such as coma, seizure, delirium and focal stroke
- Failure to understand the limitations of CT of the brain - no extravasated blood is seen in about 2–5% of patients with SAH having CT within 12 hours of onset, or in 50% of those imaged with CT at 1 week.
- Failure to perform lumbar puncture at the appropriate time and correctly interpret CSF findings.37,38

5.53 What are the symptoms of TIA and stroke?

Every stroke is different, and people who have strokes are affected in different ways. The symptoms of stroke depend on the part of the brain that is affected by a lack of blood flow and the size of the damaged area. They also vary in severity from complete recovery within 24 hours (a TIA) to a prolonged, and even persistent, deficit due to permanent damage to a part of the brain (stroke).
The symptoms of stroke usually come on suddenly, and are noticed suddenly. However, if they come on during sleep they are not noticed until the affected person wakes up. It is the suddenness of the onset of the symptoms that usually stamps them as being due to a stroke, as opposed to a condition such as migraine or brain tumour. So, if the symptoms come on slowly, and also gradually get worse over a few days, weeks or months, they are unlikely to be due to stroke. The common symptoms of stroke are described below.

**Muscle weakness or paralysis**
Stroke may cause a lack of muscle strength in any group of muscles in the body, but most commonly causes weakness of the muscles of the face, hand, arm and leg on one side (hemiparesis). Weakness on the right side of the body (right hemiparesis) is due to impaired function of the left side of the brain (by infarction or haemorrhage into the brain). Weakness on the left side of the body is due to impaired function of the right side of the brain. At least half of patients with stroke have some form of hemiparesis.

Mild weakness may sometimes involve only a difficulty in controlling movement, rather than a difficulty of actual movement.

**Loss of sensation (feeling)**
Stroke may cause a loss or alteration of feeling in any part of the body but, like weakness, most commonly causes a loss of feeling (numbness) of the skin of the face, hand, arm and leg on one side of the body (hemisensory loss or hemianaesthesia). Sensory impairment on the right side (hemianaesthesia) is due to impaired function of the left side of the brain (by infarction or haemorrhage into the brain). Sensory impairment on the left side of the body is due to impaired function of the right side of the brain. About half of patients with stroke have some form of hemianaesthesia.

**Difficulty with speech**
Stroke may cause slurred speech by causing weakness of the muscles of the face, mouth and throat. As a result, it is difficult to articulate sounds. This is called dysarthria. Speech content is normal, however.

Stroke may also cause difficulty with speech (dysphasia) in the form of difficulty:
- understanding what is said to you, like listening to a foreign language (receptive dysphasia)
- finding the right words and speaking fluently (expressive dysphasia)
- understanding what is written (i.e. difficulty reading - dyslexia)
- writing.

Dysphasia (disturbance of language) is caused by impaired function of the side of the brain that is dominant for language. The left side of the brain (hemisphere) is dominant for language in almost all individuals who are right-handed. The right side of the brain (hemisphere) is dominant for language in about half of individuals who are right-handed and half of individuals who are left-handed.
Consequently, a stroke involving the left side of the brain (left hemisphere) commonly gives rise to a loss of speech and to a loss of muscle strength and feeling on the right side of the body. This is because part of the left side of the brain controls speech (in most individuals) and an adjacent part of the left side of the brain controls movement on the right side of the body. The same major blood vessel supplies blood to these areas and, if blocked, causes loss of function of both parts of the left hemisphere of the brain.

**Visual symptoms**

Stroke and TIA can cause a loss of vision in the whole visual field of one eye (monocular blindness), in half of the visual field of each eye (homonymous hemianopia), or double vision (diplopia).

Loss of vision in one eye that comes on suddenly and resolves within 24 hours (often 5–10 minutes) is usually due to a transient ischaemic attack of the eye (a temporary lack of blood supply to one eye) and is commonly called amaurosis fugax.

Homonymous hemianopia is a loss of vision to one side, so that the patient cannot see anything to one side (either the left side or the right side) with either eye.

It can be difficult to tell the difference between transient or brief episodes of monocular blindness and homonymous hemianopia unless the patient closes each eye in turn during the episode of blindness and notes whether all the vision in one eye, or the left or right half of the vision in both eyes, is affected.

Double vision occurs when one eye is looking in one direction, and the other eye is looking in another direction, so that the brain receives images of two different objects at the same time from the two eyes looking in different directions. It occurs when the visual axis of each eye is different, in the vertical, horizontal or, indeed, any plane. This is usually caused by damage to one of the nerves that control the muscles of the eyeball.

**Dizziness**

Stroke and TIA involving the nerves originating in the balance organ in the inner ear (labyrinth) may cause a feeling of spinning called vertigo. When the world around appears to be moving (when it actually is not), this makes us feel nauseated (sometimes with vomiting) and unsteady on our feet (ataxic).

**Headache**

Stroke and TIA are not usually painful. However, headache may arise if the membrane covering the brain (the meninges) or the blood vessels in the brain are stretched or irritated.

The meninges may be stretched by swelling of a part of the brain, as occurs soon (minutes to hours) after a large bleed into the brain, or a few days after a large infarct, when swelling (oedema) of the brain is maximal. Bleeding over the surface of the brain (subarachnoid haemorrhage) characteristically causes a very severe headache, because the blood directly irritates the pain-sensitive meninges that cover the brain.
Occasionally a stroke may be caused by a tear in the inside lining of the wall of an artery (dissection) to the brain, which may lead to blockage of the artery (and ischaemic stroke) and also cause quite severe pain in the head or neck, wherever the arterial wall is torn.

**Vomiting**

Vomiting after stroke is not common, but can be caused by:

- Other symptoms of the TIA or stroke, such as vertigo. Vertigo is a sensation of rotation or spinning such that the individual feels as if he or she - or the world around them - is spinning around. This may occur if a TIA or stroke involves the nerves in the brainstem that receive information from the balance organ (labyrinth) in the inner ear. Vertigo causes both a feeling of nausea and vomiting, and also unsteadiness walking (ataxia)
- Direct involvement of the vomiting centre in the base of the brain (medulla)
- Raised pressure inside the head (commonly due to a large bleed or infarct of the brain) which is transmitted to the vomiting centre in the base of the brain (medulla).

**Drowsiness or unconsciousness**

Stroke and TIA do not commonly cause a loss of consciousness. However, as with vomiting, drowsiness and loss of consciousness can be caused by:

- Complications of the stroke (e.g. an epileptic seizure), although the drowsiness is usually transient, recovering after a few minutes to hours
- Direct involvement of the consciousness centre in the brainstem (midbrain, pons) by a strategically located bleed or infarct
- Raised pressure inside the head (commonly due to a large bleed or infarct of the brain) which is transmitted directly or indirectly to the consciousness centre in the upper brainstem (mid brain, pons).

**5.54 What should you do if you or someone else has a stroke?**

If someone suddenly loses function of a particular part of the body, which is thought to be a stroke, you should immediately call for an ambulance to transport the person to the nearest hospital. At the onset of a stroke it is uncertain whether the symptoms will resolve quickly within a few hours (a TIA) or whether they will persist (a stroke).

If the symptoms have resolved by the time the person arrives at the hospital, he or she can be assessed and discharged home with appropriate investigations, treatment and follow-up. If the symptoms persist, the person should be assessed as a medical emergency and considered for treatments that rescue damaged brain cells, prevent complications of stroke (e.g. swallowing fluid and food down the wrong way and into the lungs, causing pneumonia) and minimise a recurrence of stroke (and the occurrence of heart attack).

Sometimes, the symptoms are not due to a stroke or TIA but to another
medical condition such as migraine, epileptic seizure, fainting attack, or inflammation or tumour of the brain.

5.55 What tests will the doctors do after a TIA or stroke?

After asking about the onset and nature of your symptoms (the history) and examining your head, eyes, neck, chest, heart and limbs (physical examination), the doctor will arrange for some tests to be done to define the nature and cause of the stroke further, as described below.

Blood tests

Blood tests are to check the concentration of red blood cells, glucose (sugar) and cholesterol (fat) in the blood. A high blood glucose or cholesterol concentration may indicate underlying diabetes mellitus or high cholesterol levels, which may predispose to hardening of the arteries (atheroma). The test involves a needle being inserted into a vein in the arm.

Electrocardiography (ECG)

This is a test to check the rhythm of the heart - whether it is regular or irregular (e.g. atrial fibrillation) - and whether there is evidence of a previous heart attack or the effect of high blood pressure on the heart. It involves placing a series of electrodes on to the skin of the chest and recording the electrical activity in the heart, and is not at all painful.

Echocardiography

This is usually performed when the stroke is caused by a blocked artery to the brain (an ischaemic stroke) and when the cause of the blocked artery is thought to be a problem with the heart (i.e. a blood clot has formed in a chamber of the heart or on a valve in the heart, and has broken off and lodged downstream in a blood vessel in the neck or the brain). The doctor is usually suspicious of the heart as a source of the blood clot if the heart is beating irregularly (e.g. atrial fibrillation), if there is an abnormal sound when the doctor listens to the heart (e.g. a heart murmur), or if the ECG is abnormal.

The test involves putting some jelly on the chest and then placing a probe on the chest which emits sound waves. The jelly is to ensure good contact between the probe and the skin. The sound waves bounce off the heart and are recorded by the probe; they are then converted into images of the structure of the heart. The test is therefore an ultrasound examination of the heart. It is safe, non-invasive and painless.

CT brain scan

Computed tomography (CT) of the brain is a special kind of X-ray imaging of the brain. Its main role is to exclude other possible causes of a stroke (e.g. a brain tumour) and to distinguish between stroke caused by a blocked blood vessel and that due to a burst blood vessel. The test involves lying still on a bed while the scanner emits X-rays at your head. It does not usually involve any injections and takes only 10–15 minutes.
**MRI brain scan**

Magnetic resonance imaging (MRI) is sometimes required to obtain a more detailed image of the brain (particularly the back part of the brain) or to image the blood vessels in the brain without the need for an injection of contrast into the blood vessels. The test involves lying still on a bed while the scanner uses magnetism to image the brain. It does not use X-rays or usually involve any injections. About 5–10% of people cannot tolerate the test because they find it too claustrophobic or noisy. It usually takes 15–30 minutes.

**Carotid ultrasonography**

This examination is usually performed when the TIA or stroke is caused by a blocked artery to the brain (an ischaemic stroke) and the cause of the blockage is thought to be a problem with the carotid artery at the front of the neck (i.e. a blood clot has formed in a major blood vessel in the neck, and has broken off and lodged downstream in a blood vessel in the brain). The doctor is usually suspicious of the artery in the neck as a source of the clot if the stroke is caused by a blocked artery in the brain that is a branch of the carotid artery in the neck (a carotid territory TIA or ischaemic stroke) and if there is an abnormal sound when the doctor listens to the neck (a bruit).

The test involves putting some jelly on the neck and then placing a probe on the neck which emits sound waves. The sound waves bounce off the carotid artery in the neck and are recorded by the probe, and are then converted into images of the structure of the carotid artery and the inside of the carotid artery. It detects any narrowing of the artery that takes blood to the eye and brain. It is a useful test not only to determine the cause of the ischaemic stroke but also for deciding whether to perform an operation that cleans out the artery in the neck (carotid endarterectomy) to reduce the risk of future strokes.

**Angiography**

An angiogram is an image of the inside of the blood vessels and can be obtained non-invasively by means of magnetic resonance imaging (MRI), often at the time of an MRI scan of the brain (see above). However, the resolution of the images is not optimal. The best quality image of the inside of blood vessels is obtained by contrast catheter angiography. This involves injecting a local anaesthetic into the skin of the groin, then inserting a fine plastic tube (catheter) into the major blood vessel in the groin (femoral artery) and passing the catheter up the femoral artery into the aorta in the abdomen and chest, and then up the carotid arteries in the neck. A special dye (radio-opaque contrast) is then injected into the blood vessels in the neck and a rapid sequence of X-ray images is taken of the skull.

The angiogram shows not only the bones of the skull but also the dye (contrast) in the vessels, as it passes quickly through the brain (to be excreted in the urine). Because a catheter is inserted into the main blood vessels to the brain, and contrast dye is injected into these arteries, there is a very small risk (0.5%) that the catheter could damage the inside of one of the arteries or the contrast could irritate the inside lining of the arteries, and lead to a stroke.
REFERENCES


The Stroke Association

In the UK, the Stroke Association provides practical support, including telephone helplines, publications and welfare grants, to people who have had strokes, their families and carers. At local levels, the Stroke Association provides:

- Family support workers - people who offer emotional support and advice to families of people who have had a stroke, and to people affected by stroke who live alone
- A community service called Dysphasia Support - volunteers work to improve communication skills with people who have lost the ability to speak, read or write.

The national headquarters of the UK Stroke Association can be contacted at:

Stroke Association
Stroke House
123–127 Whitecross Street
London EC1Y 8JJ
Tel: 020 7566 0300
Website: http://www.stroke.org.uk

Different Strokes

Different Strokes is run by and for younger people who have had a stroke. Their helpline is staffed by young stroke survivors, and a national counselling network is available. Local branches exist which organise regular exercise classes.

Different Strokes
Sir Walter Scott House
PO Box 5082
Milton Keynes MK5 7ZH
Tel: 01908 236 033
Website: http://www.differentstrokes.co.uk

Local stroke clubs

Local stroke clubs are emerging rapidly. They take many different forms and may be organised by local stroke survivors or carers, district nurses, general practitioners, or local branches of the Stroke Association or Different Strokes. If interested, it is best to contact the local GP, members
of the patient’s specialist stroke team or, in the UK, the *Health Information Service* (freephone in UK: 0800 66 55 44).

**Carer groups**
- **Carers National** - Tel: 020 7490 8818
- **Relatives and Residents Association** - provides help and advice for people in long-term care facilities. Tel: 020 7916 6055

**Specific disability groups**
- **Action for Dysphasia Adults (ADA)**
  1 Royal Street
  London SE1 7LL
  *Tel: 020 7262 9572*

- **Continence Foundation**
  The Basement
  Doughty Street
  London WC1N 2PH
  *Tel: 020 7404 6875*
  *Helpline: 020 7831 9831*

- **SPOD (association to aid the sexual and personal relationships of people with a disability)**
  286 Camden Road
  London N7 OBJ
  *Tel: 020 7607 8851*
APPENDIX 2
Medical websites

American Stroke Association
*Website:* [http://www.strokeaha.org](http://www.strokeaha.org)

Chest, Heart and Stroke, Scotland
*Medical helpline:* 0845 077 6000

Cochrane Stroke Review Group
*Website:* [http://www.dcn.ed.ac.uk/csrg](http://www.dcn.ed.ac.uk/csrg)

European stroke organisation
[http://www.eurostroke.org](http://www.eurostroke.org)

Canadian stroke consortium:
[http://www.strokeconsortium.ca](http://www.strokeconsortium.ca)

International Stroke Thrombolysis Register for Safe Implementation of Thrombolysis in Stroke (SITS)
[http://www.acutestroke.org](http://www.acutestroke.org)