



Classical Stroke Syndromes

Localising the lesion from the bedside

Territories · Cortical and lacunar syndromes · Thalamus · Brainstem · Cerebellum · Posterior circulation

About this guide

From the artery to the deficit and back: the territories of the anterior and posterior circulation, the cortical, lacunar, thalamic, brainstem and cerebellar syndromes, the posterior-circulation traps, and twelve localisation cases with worked answers.

What changed. First edition. Written up and greatly expanded from the author's December 2024 teaching lecture of the same name; the lecture's illustrations are replaced by original diagrams.

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A teaching reference, not a protocol. Doses and thresholds are given to aid recognition; check them against current local guidance and prescribing information before acting on them.

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Part I

The method

Why syndromes still matter · The rules of localisation · Bamford's four

Imaging tells you where the infarct is. The examination tells you where it is *before* the scanner, when the scan is normal, when the patient is in a district hospital with no MRI, and when the deficit does not match the picture. A stroke syndrome is a cluster of deficits that one artery's territory produces, and it is the fastest route from the bedside to the vessel – and from the vessel to the treatment.

This guide grew out of a teaching session for interns in December 2024. It works in one direction and then the other: from the artery to the deficit, so that the territories make sense, and from the deficit back to the artery, which is what you do on the ward.

1.1 Five questions that localise

- 1. Is there a cortical sign?** Aphasia, neglect, a field defect, apraxia, gaze preference or cortical sensory loss puts the lesion in the hemisphere surface – a branch occlusion, usually embolic. Their absence with a dense motor or sensory deficit points to a deep, small-vessel lesion.
- 2. Which parts of the body, and in what proportion?** Face, arm and leg equally: internal capsule or pons. Face and arm more than leg: lateral convexity (MCA). Leg more than arm: medial surface (ACA).
- 3. Are the findings crossed?** A cranial nerve deficit on one side with long-tract signs on the other is brainstem until proved otherwise; the cranial nerve tells you the level and the side.
- 4. Is consciousness reduced?** One hemisphere rarely does it acutely unless the infarct is huge. Early coma means the brainstem reticular formation, both thalami, or a malignant mass effect.
- 5. Which side are the eyes looking?** A hemispheric lesion drives the eyes towards itself (away from the weak side); a pontine lesion drives them away (towards the weak side); a seizure drives them away from the focus.

● The two halves of the brainstem

Medial brainstem syndromes take the motor pathway and the cranial nerve nuclei that sit near the midline – III, IV, VI and XII. Lateral syndromes take the sensory pathways, the sympathetic fibres and the nuclei that sit laterally – V, VII, VIII, IX and X – plus the cerebellar connections. Medial means weakness; lateral means numbness, Horner, ataxia and vertigo.

1.2 The Oxfordshire classification

Bamford's 1991 classification (the Oxfordshire Community Stroke Project, OCSP) is simple, reproducible at the bedside before any scan, and it predicts outcome. Every clinician who admits strokes should be able to apply it in thirty seconds.

Syndrome	Criteria	Usual cause · one-year outlook
Total anterior circulation (TACS)	All three: new higher cortical dysfunction (aphasia, neglect, visuospatial); homonymous hemianopia; motor and/or sensory deficit in at least two of face, arm, leg.	Proximal MCA or carotid occlusion. Worst outlook: most patients dead or dependent at one year.

Syndrome	Criteria	Usual cause · one-year outlook
Partial anterior circulation (PACS)	Two of the three TACS components; or higher cortical dysfunction alone; or a motor or sensory deficit more restricted than a LACS (e.g. one limb, or face and hand).	Branch occlusion, usually embolic. Highest early recurrence.
Lacunar (LACS)	Pure motor, pure sensory, sensorimotor, or ataxic hemiparesis, involving at least two of face, arm, leg — with no cortical sign, no field defect and no brainstem sign.	Small penetrating artery disease. Lowest early mortality.
Posterior circulation (POCS)	Any of: crossed cranial nerve and long-tract signs; bilateral motor or sensory deficit; disordered conjugate eye movement; cerebellar signs without ipsilateral long-tract signs; isolated homonymous hemianopia or cortical blindness.	Vertebrobasilar territory. Best functional outlook, but a high early risk in basilar disease.

Table 1.2 Bamford et al., Lancet 1991. Add a final letter after imaging: TACI for an infarct, TACH for a haemorrhage.

Part II

The arteries

Circle of Willis · Anterior and posterior circulation · Territories and border zones

2.1 From the neck to the circle

Two internal carotid arteries carry about 80% of the brain's blood; two vertebral arteries carry the rest, joining at the pontomedullary junction to form the basilar artery. The **circle of Willis** links them: the anterior communicating artery joins the two anterior cerebrals, and each posterior communicating artery joins a carotid to a posterior cerebral. A complete, symmetrical circle is the exception — in most people one segment or more is small or absent — which is why the same occlusion produces a mild deficit in one patient and a catastrophe in another.

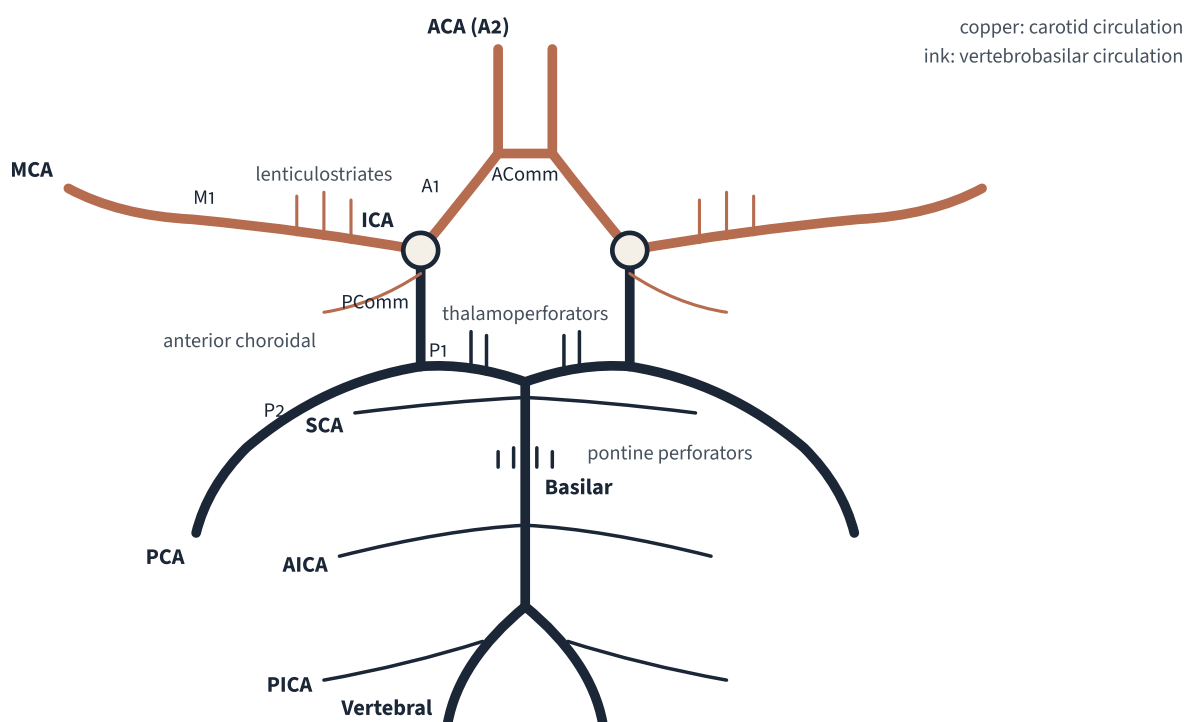


Figure 1. The circle seen from below, frontal lobes at the top. The perforating arteries — lenticulostriate, anterior choroidal, thalamoperforating and pontine — are end arteries: occlude one and nothing replaces it, which is why small deep infarcts produce such pure syndromes.

2.2 What each artery supplies

Artery	Territory	Signature deficit
Ophthalmic (ICA)	Retina and optic nerve	Transient monocular blindness (amaurosis fugax); central retinal artery occlusion
Anterior choroidal (ICA)	Posterior limb of internal capsule, optic tract, lateral geniculate body, medial temporal lobe	Hemiplegia, hemisensory loss and a field defect without cortical signs
ACA	Medial frontal and parietal lobes to the parieto-occipital sulcus; anterior four-fifths of the corpus callosum	Leg more than arm weakness; abulia; incontinence; callosal disconnection
Recurrent artery of Heubner (ACA)	Head of caudate, anterior limb of internal capsule	Face and arm weakness with abulia
MCA cortical	Lateral frontal, parietal and temporal lobes; insula	Face and arm more than leg; aphasia or neglect; hemianopia
Lenticulostriates (MCA M1)	Putamen, globus pallidus, internal capsule, corona radiata	Lacunar syndromes; striatocapsular infarction
PCA cortical	Occipital lobe, inferomedial temporal lobe, splenium	Homonymous hemianopia; alexia, memory or visual-recognition deficits
Thalamic perforators (PComm, P1, P2)	Thalamus and rostral midbrain	Sensory, arousal, memory and eye-movement syndromes (\$5)
Basilar perforators	Paramedian pons	Pure motor hemiparesis; ataxic hemiparesis; locked-in state if bilateral
SCA	Superior cerebellum, dorsolateral upper pons	Limb ataxia, dysarthria
AICA	Anterior inferior cerebellum, lateral lower pons, inner ear (labyrinthine artery)	Vertigo with hearing loss, facial palsy
PICA (or vertebral)	Lateral medulla, inferior cerebellum	Lateral medullary syndrome; vertigo and truncal ataxia

2.3 Territories and border zones on an axial slice

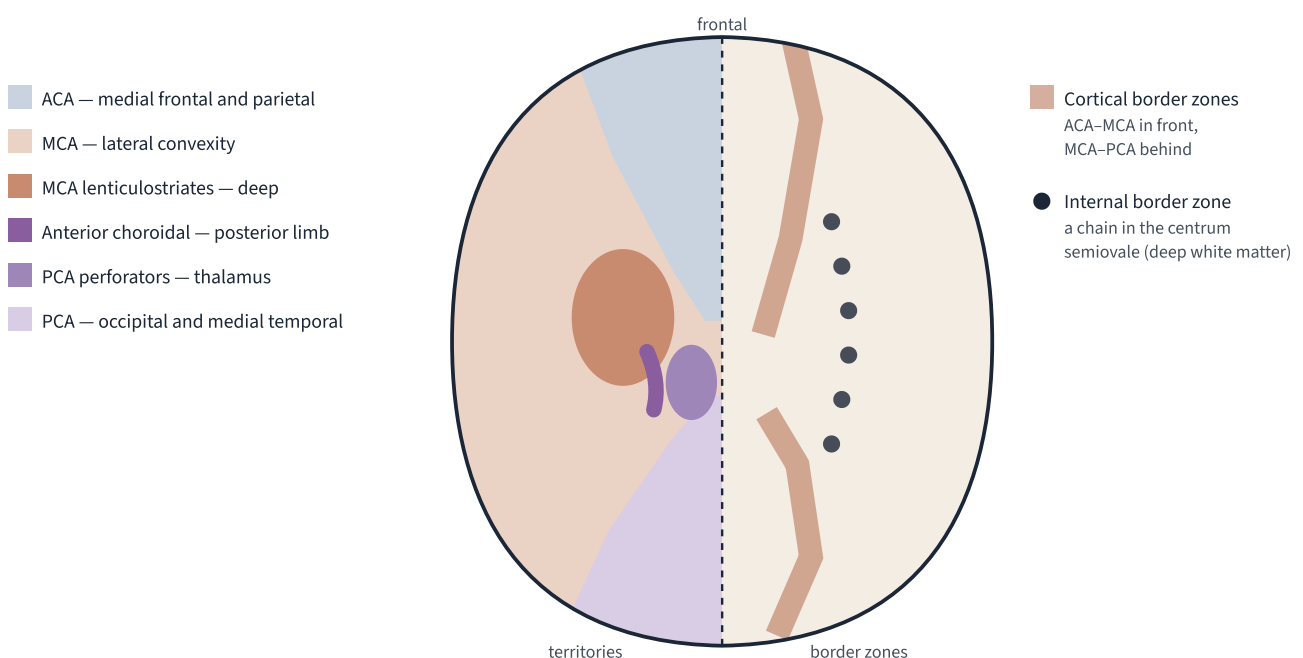


Figure 2. A schematic axial section at the level of the basal ganglia. Territories (left half) vary between people and should be read as tendencies, not borders; border zones (right half) are where perfusion is lowest when pressure falls.

Part III

The anterior circulation

Carotid · Anterior cerebral · Middle cerebral · Anterior choroidal · Border zones

3.1 Internal carotid artery

Carotid disease announces itself in two ways. **Embolism** from a stenosed or dissected carotid sends clot downstream, most often to the MCA. **Haemodynamic failure** distal to a severe stenosis or occlusion starves the border zones when blood pressure falls.

- **Transient monocular blindness** – a curtain descending over one eye for minutes – is a carotid TIA and carries a high early stroke risk. A central retinal artery occlusion is a stroke of the eye and needs the same urgent work-up.
- **Limb-shaking TIA** – brief, repetitive, involuntary jerking of the contralateral arm or leg on standing or exertion – is haemodynamic carotid insufficiency, not epilepsy.
- **Carotid dissection** – ipsilateral head, face or neck pain, a partial Horner syndrome (ptosis and miosis, sweating spared), sometimes lower cranial nerve palsies or pulsatile tinnitus, then a contralateral hemispheric stroke.
- **Acute occlusion** with a poor circle produces the full MCA picture, often with ACA territory as well.

3.2 Anterior cerebral artery

ACA infarcts are uncommon (a few per cent of ischaemic strokes) because the anterior communicating artery so often rescues the territory. When it happens, the pattern is distinctive enough to be recognised from the end of the bed.

Deficit	Why
Weakness and sensory loss of the leg, more than the arm; face spared	The leg area of the motor and sensory strips lies on the medial surface. The proximal arm is often weak too; the hand and face are spared.
Abulia, apathy, slowness, mutism	Medial frontal lobe and cingulate gyrus; worst with bilateral infarction.
Urinary incontinence	Medial frontal micturition centre — the patient is unconcerned by it.
Grasp and groping reflexes	Released frontal reflexes in the contralateral hand.
Transcortical motor aphasia	Dominant supplementary motor area: little spontaneous speech, intact repetition.
Callosal disconnection	Left-hand apraxia, left-hand agraphia and tactile anomia — the right hemisphere is cut off from left-hemisphere language.
Alien hand	Frontal type (medial frontal): groping, grasping, compulsive use of objects by the contralateral hand. Callosal type: intermanual conflict, one hand undoing what the other does.

● **When both anterior cerebrals go**

If both A2 segments arise from one A1, or a single (azygos) A2 supplies both hemispheres, one occlusion infarcts both medial frontal lobes: paraparesis that looks spinal, incontinence and akinetic mutism. The same picture follows vasospasm after a ruptured anterior communicating aneurysm. Always examine the arms and the face before calling a paraparesis "cord".

3.3 Middle cerebral artery

The MCA is the commonest site of embolic occlusion and the source of the most disabling syndromes. Its stem (M1) gives the lenticulostriate arteries before dividing, usually into a superior and an inferior division (M2), whose cortical branches (M3–M4) spread over the convexity.

Level	Dominant (usually left) hemisphere	Non-dominant (usually right)
M1 stem, before the lenticulostriates	Contralateral hemiplegia (face and arm worse than leg), hemisensory loss, homonymous hemianopia, eyes and head deviated towards the lesion — plus global aphasia (left) or neglect, anosognosia and dysarthria (right). Risk of malignant oedema.	
Superior division	Face and arm weakness; Broca's (non-fluent) aphasia with apraxia of speech; gaze preference	Face and arm weakness; neglect, motor impersistence, flat prosody
Inferior division	Wernicke's (fluent) aphasia , often with little or no weakness — mistaken for confusion or psychosis; superior quadrantanopia	Neglect, constructional and dressing apraxia, agitated confusional state; superior quadrantanopia
Angular gyrus branch	Gerstmann syndrome : agraphia, acalculia, finger agnosia, right–left disorientation; alexia with agraphia; anomia	Spatial disorientation, constructional apraxia
Deep (lenticulostriate)	Striatocapsular infarction: hemiparesis of face, arm and leg, little sensory loss; a cortical sign suggests a large deep infarct with poor collaterals. Smaller occlusions give lacunar syndromes (§3.5).	

Aphasia	Fluency	Comprehension	Repetition	Naming	Lesion
Broca	Non-fluent	Relatively intact	Impaired	Impaired	Left inferior frontal gyrus and beneath
Wernicke	Fluent, paraphasic	Impaired	Impaired	Impaired	Left posterior superior temporal gyrus
Conduction	Fluent, phonemic errors	Intact	Severely impaired	Impaired	Left supramarginal gyrus, arcuate fasciculus
Global	Non-fluent	Impaired	Impaired	Impaired	Large left MCA infarct
Transcortical motor	Non-fluent	Intact	Preserved	Impaired	Left SMA or ACA–MCA border zone
Transcortical sensory	Fluent	Impaired	Preserved	Impaired	Left MCA–PCA border zone
Anomic	Fluent	Intact	Intact	Impaired	Anywhere; least localising

Table 3.3 Repetition is the pivot: impaired in the perisylvian aphasias, preserved in the transcortical ones. Subcortical (thalamic, striatocapsular) aphasias tend to preserve repetition too.

◆ Malignant MCA infarction

- A complete MCA infarct swells over two to five days; without surgery about four in five die. Watch for falling consciousness, a dilating pupil and new contralateral signs.
- Decompressive hemicraniectomy within 48 hours roughly halves mortality and increases survival with moderate disability in patients up to 60 (pooled DECIMAL, DESTINY and HAMLET). Over 60 it saves lives but most survivors are severely disabled (DESTINY II) – a decision to share with the family early, before the swelling forces it.
- The dominant hemisphere is not a contraindication; aphasia often improves more than expected.

3.4 Anterior choroidal artery

A small branch of the distal carotid that supplies the posterior limb of the internal capsule, the optic tract and lateral geniculate body, and part of the medial temporal lobe. Occlusion gives the triad of **hemiplegia, hemisensory loss and homonymous hemianopia** – the visual defect sometimes a characteristic sectoranopia from the lateral geniculate body – with cognition, language and attention preserved. That combination, without cortical signs, is the clue; the full triad is present in a minority.

3.5 Lacunar syndromes

Lacunes are small (under 15–20 mm) deep infarcts from occlusion of a single penetrating artery, most often from lipohyalinosis or microatheroma at its origin. C. Miller Fisher described the classic syndromes; the five below account for most of them. They share what they lack: no aphasia, no neglect, no field defect, no reduced consciousness.

Syndrome	Features	Usual sites
Pure motor hemiparesis	Face, arm and leg weak to a similar degree; no sensory loss. The commonest.	Posterior limb of internal capsule; basis pontis; corona radiata
Pure sensory stroke	Numbness of face, arm and leg on one side, sometimes splitting the midline exactly.	Ventral posterior thalamus
Sensorimotor stroke	Both, without cortical signs.	Thalamocapsular junction
Ataxic hemiparesis	Weakness (often leg more than arm) with ataxia out of proportion to it, on the same side.	Basis pontis; internal capsule; corona radiata
Dysarthria–clumsy hand	Dysarthria, facial weakness and a clumsy hand. A variant of ataxic hemiparesis.	Basis pontis; genu of internal capsule

◆ Capsular warning syndrome

Repeated, stereotyped episodes of pure motor or sensorimotor deficit – several in hours – from a failing single perforator. A large proportion go on to a completed capsular or pontine infarct. Admit, do not reassure, and treat urgently; the role of thrombolysis and dual antiplatelets is a decision for the stroke team.

3.6 Border-zone (watershed) infarction

Border zones lie where two arterial territories meet, at the end of each supply. They infarct when perfusion falls – cardiac arrest, profound hypotension, or a severe carotid stenosis that has used up its reserve – and the pattern names the cause.

- **Anterior (ACA–MCA) cortical border zone** – weakness of the proximal arm and shoulder, sparing face and hand; bilaterally, the "**man in a barrel**" after cardiac arrest: arms weak, legs and face relatively spared. Dominant side: transcortical motor aphasia.
- **Posterior (MCA–PCA) border zone** – transcortical sensory aphasia (dominant), visuospatial disturbance, lower quadrantanopia; bilaterally, **Balint syndrome** (§4.2).
- **Internal border zone** – a chain of small infarcts in the centrum semiovale parallel to the lateral ventricle, strongly linked to carotid occlusion or severe stenosis. Look for it on MRI; it predicts recurrence.

Part IV

The posterior cerebral artery

Vision · Reading · Memory · Recognition

4.1 One posterior cerebral artery

The PCA usually arises from the top of the basilar; in about one person in five it comes mainly from the carotid through a large posterior communicating artery (the fetal pattern), so a carotid embolus can produce a PCA stroke. Its proximal segment (P1) gives perforators to the midbrain and thalamus; its cortical branches supply the occipital lobe, the inferomedial temporal lobe and the splenium.

Deficit	Detail
Contralateral homonymous hemianopia	Often with macular sparing — the occipital pole has a dual MCA–PCA supply. Patients may be unaware and present with "bumping into things" or a car accident. Small or upper-bank lesions give quadrantanopias.
Alexia without agraphia (left PCA)	Left occipital cortex plus the splenium: the patient writes a sentence and cannot read it back. Usually with a right hemianopia; colour naming is often impaired too.
Memory impairment	Hippocampal and parahippocampal infarction: verbal memory on the left, visual on the right; severe and persistent if bilateral.
Visual recognition (right or bilateral)	Prosopagnosia (inability to recognise faces — fusiform gyrus, usually bilateral or right), achromatopsia, topographical disorientation.
Thalamic and midbrain signs	If the occlusion is proximal (P1): contralateral sensory loss, vertical gaze palsy, third nerve palsy, reduced arousal (§5).
Confusional state	Particularly with right or bilateral infarcts; often mistaken for delirium.

4.2 Both posterior cerebral arteries

- **Cortical blindness** – loss of vision with normal pupillary reflexes and fundi, because the pathway to the midbrain is intact.
- **Anton syndrome** – cortical blindness denied: the patient confabulates what they "see" and walks into furniture.
- **Balint syndrome** – bilateral parieto-occipital damage, classically from the posterior border zones: simultanagnosia (seeing one element of a scene at a time), optic ataxia (misreaching under visual guidance) and ocular apraxia (difficulty directing gaze voluntarily).
- **Severe amnesia** from bilateral medial temporal infarction.

● **An embolus that travelled up the basilar**

Bilateral PCA infarction, often with thalamic and midbrain involvement, is the footprint of an embolus lodged at the top of the basilar. Look for the cardiac source and image the whole posterior circulation.

Part V

The thalamus

Four arteries, four syndromes

The thalamus is small, but its four vascular territories produce four quite different syndromes – and bilateral paramedian infarction is a cause of coma that is easy to miss on an early CT.

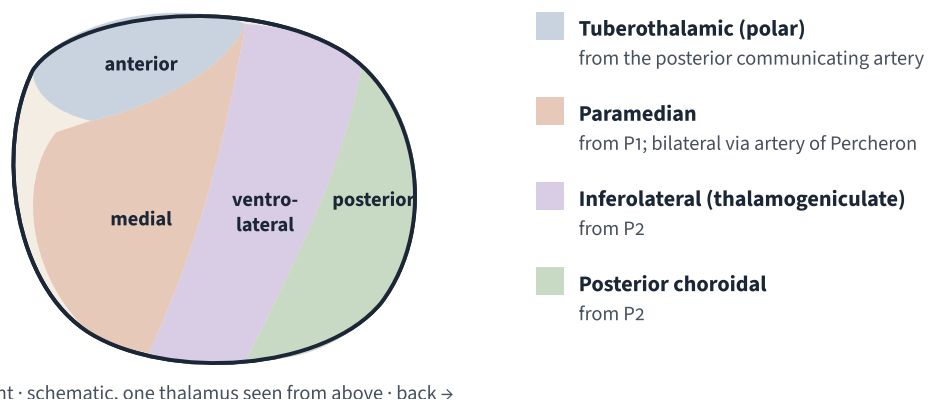


Figure 3. The four thalamic territories (after Schmahmann, Stroke 2003). Boundaries vary between individuals.

Territory	Syndrome
Tuberothalamic	Apathy, perseveration, personality change and anterograde amnesia — verbal on the left, visual on the right; emotional facial paresis. Often mistaken for a frontal lesion or a primary psychiatric illness.
Paramedian	Reduced arousal and hypersomnolence at onset; vertical gaze palsy; amnesia and apathy afterwards. Artery of Percheron: a single trunk supplying both paramedian thalami (and sometimes the rostral midbrain) — sudden coma or stupor with vertical gaze palsy and a normal early CT. Ask for MRI with diffusion.
Inferolateral	Contralateral sensory loss of all modalities; hemiataxia; sometimes mild hemiparesis. Weeks later, in some, central post-stroke pain (Dejerine–Roussy): burning, allodynic pain in the numb side.
Posterior choroidal	Field defects from the lateral geniculate body (quadrantanopia, sectoranopia); variable sensory loss; aphasia or neglect from the pulvinar.

Part VI

The brainstem

Crossed signs · Midbrain · Pons · Medulla

6.1 Reading a crossed syndrome

Eponymous brainstem syndromes are less important than the method that generates them, because real infarcts rarely respect the textbook boundaries. Find the cranial nerve sign – it gives the level and the side of the lesion. Then add the long tracts: motor (medial), spinothalamic (lateral), medial lemniscus (medial), sympathetic (lateral) and cerebellar connections.

Level	Medial (paramedian)	Lateral
Midbrain nerves III (and IV)	Oculomotor nucleus and fascicle Cerebral peduncle (corticospinal) Red nucleus, superior cerebellar peduncle fibres	Spinothalamic tract Trochlear nucleus (dorsal) Dorsal midbrain: vertical gaze, pupils, lid (Parinaud)
Pons nerves V, VI, VII, VIII	Corticospinal tract (basis pontis) Abducens nucleus and fascicle MLF, PPRF (horizontal gaze) Facial nerve fascicle	Trigeminal nuclei · facial nucleus Vestibular and cochlear nuclei Middle cerebellar peduncle Spinothalamic · sympathetic
Medulla nerves IX, X, XI, XII	Pyramid (corticospinal) Medial lemniscus Hypoglossal nucleus and fascicle (tongue)	Spinal trigeminal nucleus · nucleus ambiguus · vestibular nuclei Inferior cerebellar peduncle Spinothalamic · sympathetic

Figure 4. Medial lesions take the motor pathway and the midline nuclei (III, IV, VI, XII); lateral lesions take sensation, the sympathetic fibres, the cerebellar connections and the lateral nuclei (V, VII, VIII, IX, X).

6.2 Midbrain

Syndrome	Lesion	Ipsilateral	Contralateral
Weber	Ventral midbrain: cerebral peduncle and third nerve fascicle	Third nerve palsy	Hemiparesis, including the lower face
Claude	Tegmentum: third nerve fascicle and red nucleus / cerebellar outflow	Third nerve palsy	Ataxia, tremor
Benedikt	Tegmentum and peduncle combined	Third nerve palsy	Ataxia, involuntary movements, hemiparesis
Dorsal midbrain (Parinaud)	Pretectum and posterior commissure	Upgaze palsy, convergence–retraction nystagmus on attempted upgaze, light–near dissociation of the pupils, lid retraction (Collier's sign). Also from pineal tumours and hydrocephalus.	

6.3 Pons

Syndrome	Lesion	Ipsilateral	Contralateral
Millard–Gubler	Ventral caudal pons: corticospinal tract, VI and VII fascicles	Lateral rectus palsy, lower motor neurone facial palsy	Hemiparesis sparing the face
Foville	Dorsal caudal pons: PPRF or abducens nucleus, VII fascicle	Horizontal gaze palsy to the side of the lesion; facial palsy	Hemiparesis
One-and-a-half	PPRF or abducens nucleus plus the ipsilateral MLF	No horizontal movement of the ipsilateral eye; the contralateral eye can only abduct, with nystagmus. Add a facial palsy and it is "eight-and-a-half".	
Lateral inferior pontine (AICA)	Lateral pons, middle cerebellar peduncle, inner ear	Deafness and tinnitus, vertigo, facial palsy, facial numbness, Horner, ataxia	Pain and temperature loss in the body
Paramedian pontine	Basilar perforator	Pure motor hemiparesis, ataxic hemiparesis or dysarthria–clumsy hand — the pontine lacunes.	
Locked-in	Bilateral ventral pons — basilar occlusion	Quadriplegia and anarthria with preserved consciousness, vertical eye movements and blinking. Ask the patient to look up and down before deciding that they are in a coma.	

6.4 Medulla

The **lateral medullary syndrome** (Wallenberg) is the commonest brainstem stroke syndrome and the most rewarding to examine, because almost every finding has a named structure behind it. The usual cause is occlusion or dissection of the intracranial **vertebral** artery rather than PICA itself.

Finding	Structure
Vertigo, nausea, vomiting, nystagmus, lateropulsion	Vestibular nuclei
Ipsilateral loss of pain and temperature in the face; facial pain	Spinal trigeminal nucleus and tract
Contralateral loss of pain and temperature in the body	Spinothalamic tract
Dysphagia, hoarseness, ipsilateral palatal and vocal cord weakness; hiccups	Nucleus ambiguus (IX, X)
Ipsilateral Horner syndrome	Descending sympathetic fibres
Ipsilateral limb and gait ataxia	Inferior cerebellar peduncle, cerebellum
What is absent	Weakness (the pyramid is medial) and loss of joint position sense (the medial lemniscus is medial)

◆ Dysphagia kills in Wallenberg's

Aspiration pneumonia is a leading cause of death after lateral medullary infarction. Nil by mouth until a formal swallow assessment; consider early nasogastric feeding. A vertebral dissection may also extend or embolise — image the vessel.

Other medullary syndromes	Lesion	Findings
Medial medullary (Dejerine)	Pyramid, medial lemniscus, XII fascicle — vertebral or anterior spinal artery branches	Ipsilateral tongue weakness (deviates towards the lesion); contralateral hemiparesis sparing the face; contralateral loss of joint position and vibration sense
Hemimedullary (Babinski–Nageotte)	Medial and lateral medulla	Lateral medullary syndrome plus contralateral hemiparesis
Opalski	Lateral medulla extending below the pyramidal decussation	Lateral medullary syndrome with ipsilateral hemiparesis

Part VII

The cerebellum and the basilar artery

Three cerebellar arteries · Top of the basilar · Dizziness that is a stroke

7.1 Cerebellar infarction

Artery	Features	Trap
PICA	Vertigo, vomiting, nystagmus, truncal and gait ataxia — the patient cannot sit or stand unaided — with little limb ataxia in the medial branch territory	Isolated vertigo that looks peripheral; examine gait, not just the limbs
AICA	Vertigo with acute hearing loss and tinnitus, facial palsy, facial numbness, Horner, limb ataxia	Sudden deafness with vertigo is not always labyrinthitis; it can herald basilar occlusion
SCA	Limb ataxia and dysarthria; vertigo less prominent	Often part of a top-of-basilar embolus

◆ The cerebellar infarct that swells

Large cerebellar infarcts swell over two to four days, compressing the fourth ventricle and brainstem: headache, vomiting, falling consciousness, new gaze palsy. Obstructive hydrocephalus and brainstem compression need urgent neurosurgical review; suboccipital decompressive craniectomy is life-saving, and a ventricular drain alone risks upward herniation.

7.2 Basilar occlusion and the top of the basilar

Basilar occlusion is the most lethal ischaemic stroke and among the most often missed. It may begin with a prodrome of fluctuating diplopia, dysarthria, vertigo or hemiparesis over hours or days, or present abruptly with coma.

- **Top-of-the-basilar syndrome** (Caplan, 1980) — an embolus at the bifurcation infarcts the rostral midbrain, thalami and occipital lobes: vertical gaze palsy, pupillary abnormalities, somnolence, vivid visual hallucinations (peduncular hallucinosis), memory loss and field defects or cortical blindness — often with little weakness.
- **Mid-basilar occlusion** — pontine syndromes up to locked-in state.
- **Warning signs** — reduced consciousness with any eye-movement abnormality, bilateral or alternating limb signs, and a "stroke" that fluctuates.

Treatment has changed

Two Chinese trials, ATTENTION and BAOCHE (2022), showed that thrombectomy improves outcome after basilar occlusion. The 2026 AHA/ASA guideline makes thrombectomy a Class 1 recommendation for basilar occlusion with NIHSS ≥ 10 within 24 hours. The syndrome you recognise at the bedside is the one you send for angiography.

7.3 Acute vertigo: stroke or labyrinth?

Most acute vertigo is peripheral, but a cerebellar or brainstem stroke can present with vertigo alone, and early MRI diffusion misses a meaningful fraction of small posterior-circulation infarcts in the first 24–48 hours. In a patient with continuous vertigo and spontaneous nystagmus (the acute vestibular syndrome), the HINTS examination performed by a trained examiner is more sensitive than early MRI (Kattah et al., 2009).

Test	Reassuring — peripheral	Dangerous — central
Head impulse	Abnormal: a corrective saccade on turning towards the affected ear	Normal head impulse in a patient with acute vertigo
Nystagmus	Unidirectional horizontal, beats away from the affected ear, suppressed by fixation	Direction-changing on eccentric gaze; purely vertical or torsional
Test of skew	No vertical refixation on alternate cover	Skew deviation — vertical misalignment
Plus: hearing	—	New unilateral hearing loss with vertigo suggests AICA infarction

Table 7.3 Any one dangerous feature makes the presentation central until proved otherwise. HINTS applies only to continuous vertigo with nystagmus — not to brief positional episodes, which are tested with the Dix–Hallpike manoeuvre — and should not be used in a patient who is not dizzy now. Also check gait: inability to stand unaided is central.

Part VIII

From syndrome to scan and treatment

Mimics and chameleons · Imaging · What changes in the first hours

8.1 Mimics and chameleons

Looks like a stroke — is not	Clue
Seizure with post-ictal (Todd) paresis	Witnessed seizure, tongue biting, deficit improving over hours; gaze away from the weak side at onset
Hypoglycaemia	Any focal deficit — check the glucose before anything else
Migraine with aura	Positive symptoms that march over minutes; history of similar attacks
Functional disorder	Positive signs (Hoover's sign, give-way weakness, midline-splitting sensory loss with inconsistency) — a diagnosis to make on positive features, not by exclusion
Space-occupying lesion, subdural haematoma	Progression over days; headache; a deficit that does not fit one artery
Sepsis unmasking an old stroke	Old deficit re-emerging with fever or metabolic upset

Looks like a stroke — is not	Clue
Bell's palsy, peripheral vertigo	Forehead involved (Bell's); positive head impulse and positional history (vertigo)
Is a stroke — does not look like one	Clue
Isolated vertigo	Normal head impulse, central nystagmus, skew, inability to walk (§7.3)
Acute confusion	Right MCA inferior division, PCA or thalamic infarction — look for neglect and field defects
Fluent nonsense	Wernicke's aphasia mislabelled as psychosis
Sudden amnesia	Thalamic, hippocampal or fornix infarction (transient global amnesia resolves within 24 hours)
Isolated hand weakness	Small cortical infarct in the "hand knob" of the precentral gyrus — mimics a radial or ulnar palsy
Coma	Basilar occlusion or artery of Percheron infarction

8.2 Imaging — what the syndrome asks of the scanner

- **Non-contrast CT** excludes haemorrhage and shows established infarction; early ischaemic change is scored with ASPECTS in the MCA territory. It is insensitive in the posterior fossa.
- **CT angiography** from the aortic arch to the vertex in every candidate for thrombectomy — the syndrome predicts the occlusion, the angiogram confirms it.
- **CT perfusion or MRI** selects patients in later windows by showing salvageable tissue.
- **MRI with diffusion** is the test of choice for small, brainstem and posterior-fossa infarcts — but can be falsely negative in the first day.

8.3 What changes in the first hours (2026 AHA/ASA)

- Intravenous thrombolysis with **tenecteplase or alteplase** within 4.5 hours is Class 1; between 4.5 and 9 hours it is reasonable when imaging shows salvageable tissue (Class 2a).
- **Thrombectomy** for anterior large-vessel occlusion within 24 hours in selected patients, now including large infarct cores (ASPECTS 3–5); for **basilar occlusion** with NIHSS ≥ 10 within 24 hours (Class 1). Medium and distal vessel occlusions have no recommendation for thrombectomy.
- After reperfusion, **do not lower systolic pressure below 140 mmHg** (Class 3: harm).
- Lacunar syndromes are not a reason to withhold thrombolysis; the capsular warning syndrome is a reason to act quickly.

Thresholds change: read the current guideline before acting, and the updates page for what has moved.

Part IX

Localisation cases

Twelve patients, from the bedside to the artery

Case 1

A 74-year-old woman with atrial fibrillation wakes unable to move her right arm. Her right face droops, the arm is plegic, the leg is mildly weak, and she produces only a few effortful words, though she follows commands. Eyes deviated to the left.

- A. Left internal capsule lacune
- B. Left MCA superior division
- C. Left ACA
- D. Left PCA

B. Face and arm more than leg, non-fluent aphasia with comprehension preserved, gaze towards the lesion: left MCA superior division, embolic from the atrial fibrillation. A lacune would not cause aphasia or gaze deviation.

Case 2

A 68-year-old man is brought by his family for "confusion". He talks fluently in long sentences that make no sense, does not follow commands, and is unaware of any problem. Power is normal.

- A. Delirium
- B. Left MCA inferior division
- C. Right MCA superior division
- D. Acute psychosis

B. Wernicke's aphasia from an inferior-division embolus, often with little or no weakness. Test repetition and naming; look for a right upper quadrantanopia.

Case 3

A 59-year-old has sudden weakness of the right leg; the right arm is slightly weak at the shoulder, the hand and face are normal. He is slow, says little, and is incontinent without concern.

- A. Left ACA
- B. Spinal cord infarction
- C. Left MCA
- D. Right pontine lacune

A. Leg more than arm with abulia and incontinence – medial frontal, left ACA territory. A cord lesion would not cause abulia, and examination of sensory levels would differ.

Case 4

A 62-year-old hypertensive man has weakness of the left face, arm and leg to an equal degree. Sensation, vision, language and attention are normal.

- A. Right MCA cortical branch
- B. Pure motor lacunar stroke — right internal capsule or pons
- C. Right ACA
- D. Right thalamus

B. Face, arm and leg equally, no cortical signs, no sensory loss: pure motor hemiparesis from a lacune in the posterior limb of the internal capsule or the basis pontis. Still a candidate for thrombolysis.

Case 5

A 45-year-old woman has vertigo, vomiting and hoarseness after neck pain for two days. Examination: left ptosis and miosis, reduced pain sensation on the left face and the right arm and leg, left limb ataxia, power normal.

- A. Right lateral medulla
- B. Left lateral medulla — left vertebral dissection
- C. Left medial medulla
- D. Left lateral pons

B. Ipsilateral face and contralateral body pain-and-temperature loss, ipsilateral Horner and ataxia, hoarseness, no weakness: left lateral medullary syndrome. Neck pain suggests vertebral dissection. Keep nil by mouth until the swallow is assessed.

Case 6

A 70-year-old man has a right third nerve palsy with a dilated pupil and weakness of the left face, arm and leg.

- A. Weber syndrome — right ventral midbrain
- B. Right uncal herniation only
- C. Claude syndrome
- D. Left Weber syndrome

A. Ipsilateral third nerve with contralateral hemiparesis: right cerebral peduncle (Weber), PCA or top-of-basilar perforators. In a patient with a falling GCS and a hemispheric mass, the same combination means uncal herniation — context decides.

Case 7

A 66-year-old woman suddenly cannot read her newspaper, though she writes a letter normally and cannot read it back. She has a right homonymous hemianopia.

- A. Left MCA superior division
- B. Left angular gyrus
- C. Left PCA with splenial involvement
- D. Right PCA

C. Alexia without agraphia: left occipital cortex plus the splenium disconnects the intact right visual cortex from left-hemisphere language. Angular gyrus lesions cause alexia *with* agraphia.

Case 8

A 58-year-old man is found unresponsive. He opens his eyes to voice, then drifts off. Vertical eye movements are absent; horizontal movements are full. Limbs move to pain. CT on arrival is normal.

- A. Metabolic encephalopathy
- B. Bilateral paramedian thalamic infarction — artery of Percheron
- C. Right MCA infarction
- D. Non-convulsive status epilepticus

B. Hypersomnolence with vertical gaze palsy and a normal early CT: bilateral paramedian thalamic ($\pm$ rostral midbrain) infarction from an artery of Percheron. MRI with diffusion confirms it; EEG is still reasonable if the picture is uncertain.

Case 9

A 63-year-old has had continuous vertigo with nausea for six hours. Horizontal nystagmus beats left on left gaze and right on right gaze. The head impulse test is normal in both directions. He cannot stand without support.

- A. Vestibular neuritis — reassure and discharge
- B. Benign paroxysmal positional vertigo
- C. Posterior circulation stroke until proved otherwise
- D. Ménière's disease

C. Direction-changing nystagmus, a normal head impulse during continuous vertigo, and inability to stand are all central features. A normal early MRI does not exclude a small infarct.

Case 10

A 52-year-old resuscitated after a cardiac arrest can move his legs and face normally but cannot lift either arm at the shoulder; grip is relatively preserved.

- A. Bilateral ACA infarction
- B. Cervical cord lesion
- C. Bilateral ACA–MCA border-zone infarction
- D. Bilateral lacunes

C. "Man in a barrel": proximal arm weakness from bilateral anterior border-zone infarction after global hypoperfusion. Check reflexes and sensory level to exclude the cord.

Case 11

A 49-year-old woman has sudden vertigo, right-sided deafness and tinnitus, right facial weakness including the forehead, right facial numbness and right limb ataxia.

- A. Right lateral medullary syndrome
- B. Right AICA infarction (lateral inferior pons)
- C. Labyrinthitis
- D. Bell's palsy with vestibular neuritis

B. Hearing loss plus facial palsy, facial numbness and ataxia on one side: AICA territory, including the labyrinthine artery. Image the basilar – AICA infarction can herald basilar occlusion.

Case 12

A 71-year-old cannot see but insists the room is "a bit dark". He describes the doctor's tie incorrectly and walks into the door. Pupils react briskly; fundi are normal.

- A. Bilateral optic neuropathy
- B. Anton syndrome — bilateral occipital (PCA) infarction
- C. Functional visual loss
- D. Pituitary apoplexy

B. Cortical blindness with denial and confabulation. Brisk pupils and normal fundi place the lesion behind the lateral geniculate body. Look for a top-of-basilar embolus and its source.

Appendix

Quick reference and sources

For the ward and for checking

A One-line localisers

Leg more than arm

ACA, or parasagittal; check the face and the bladder

Face and arm more than leg	MCA cortical
Face, arm and leg equal, no cortical sign	Internal capsule or pons (lacune)
Crossed signs	Brainstem; the cranial nerve gives the level and the side
Hemianopia alone	PCA (or optic radiation)
Hemiplegia + hemianopia, no cortical sign	Anterior choroidal artery
Hearing loss + vertigo	AICA — think basilar
Hypersomnolence + vertical gaze palsy	Paramedian thalami — artery of Percheron
Proximal arms, bilateral	Anterior border zones after hypotension
Alexia without agraphia	Left PCA with splenium
Blind, unaware, normal pupils	Bilateral occipital — Anton

B Abbreviations

ACA	anterior cerebral artery
AComm	anterior communicating artery
AICA	anterior inferior cerebellar artery
ASPECTS	Alberta Stroke Program Early CT Score
ICA	internal carotid artery
MCA	middle cerebral artery
MLF	medial longitudinal fasciculus
NIHSS	National Institutes of Health Stroke Scale
OCSP	Oxfordshire Community Stroke Project
PCA	posterior cerebral artery
PComm	posterior communicating artery
PICA	posterior inferior cerebellar artery
PPRF	paramedian pontine reticular formation
SCA	superior cerebellar artery
SMA	supplementary motor area
TIA	transient ischaemic attack

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