Posterior Circulation Stroke:

Vascular Anatomy and Clinical Recognition for Emergency Clinicians

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How to Use This Review

This review examines posterior circulation stroke, focusing on vascular anatomy, territory-specific clinical presentations, and recognition of stroke patterns that commonly present with vertigo or dizziness.

The document follows a structured clinical format with numbered sections, integrated callout boxes for rapid reference, summary tables, and a references section. It is designed both as a learning resource and a quick-reference tool for practising clinicians.

 Key Point: *Foundational concepts and summary statements that anchor the core scientific content of each section.*

 Clinical Insight: Clinically relevant observations derived directly from the evidence — for direct application in assessment and diagnosis.

 Clinical Pearl: High-yield, memorable clinical points — the take-home messages most likely to influence management or examination performance.

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I. Introduction

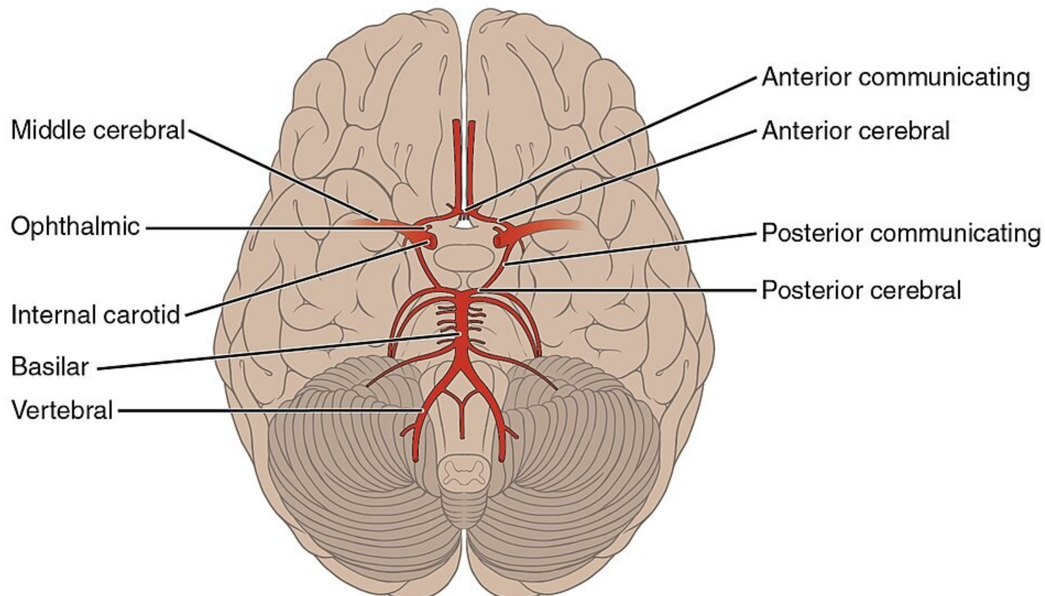

Figure 1. Inferior (basal) view of the brain showing the Circle of Willis and vertebrobasilar system. Anterior circulation (ICA -> MCA, ACA, ophthalmic) and posterior circulation (vertebral -> basilar -> PCA) converge at the arterial circle.
Source: Wikimedia Commons

Posterior circulation stroke accounts for approximately 15–20% of all ischaemic strokes but causes disproportionate morbidity and mortality because frontline recognition is delayed or missed [4,5,11]. Small infarcts in the cerebellum or brainstem commonly present with vertigo, nausea and gait unsteadiness — a pattern indistinguishable from peripheral vestibular disease at first glance [2,3].

The clinical challenge is the overlap between benign peripheral vestibular disease and dangerous posterior-circulation ischaemia [2,8]. A systematic approach anchored on vascular risk assessment, the HINTS+ examination, careful gait testing and liberal use of MRI-DWI is essential to close the diagnostic gap in the emergency department [2,5,8].

Compared with anterior-circulation stroke, posterior-circulation events are missed at initial presentation in roughly 35% of cases — more than three times the rate of anterior-circulation misses [2,4,11]. The consequences include preventable death from cerebellar oedema or basilar occlusion, and lifelong disability from delayed reperfusion [3,4].
The reasons for under-recognition are well described: clinicians underweight 'isolated' vestibular symptoms, posterior-fossa CT is insensitive in the first 24 hours, and the bedside vestibular examination is rarely performed competently outside vestibular-medicine practice [2,7,11].

 Clinical Pearl: Posterior circulation stroke causes 15–20% of all strokes but is often missed because it presents with vertigo.

II. Vertebrobasilar Anatomy

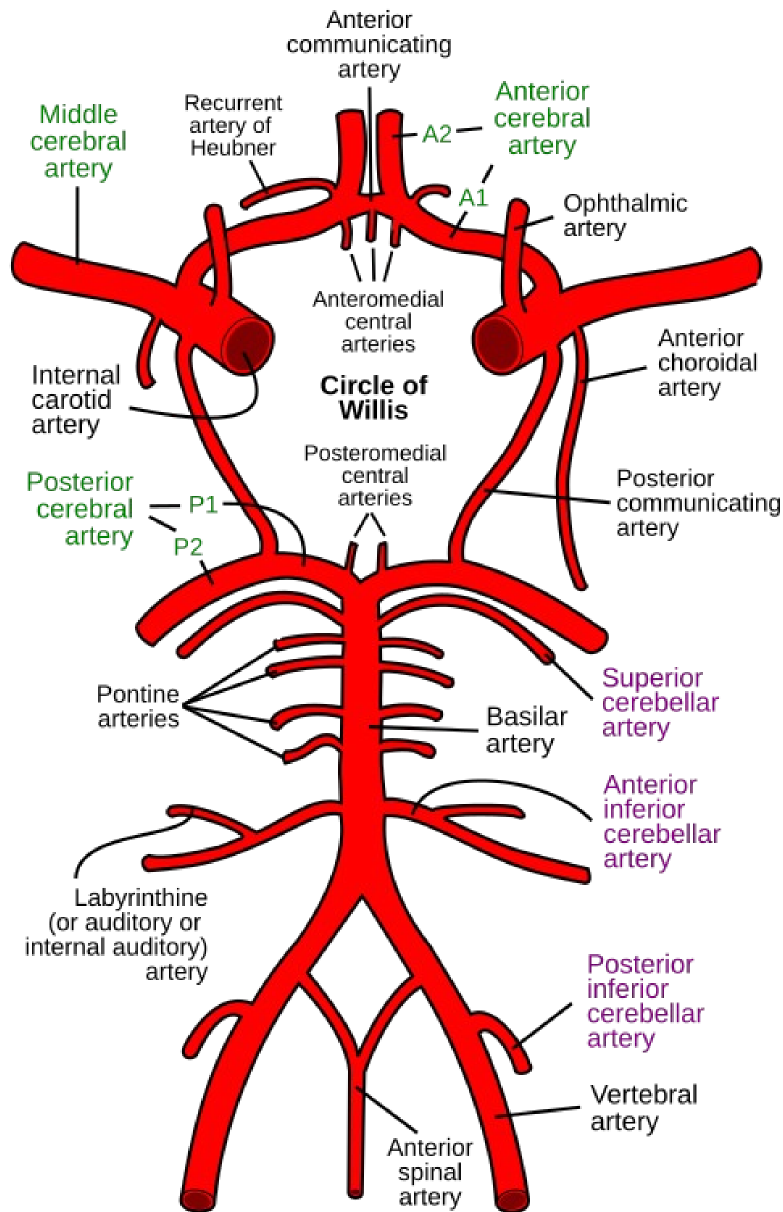

Figure 2. Schematic of vertebrobasilar and Circle of Willis anatomy. The paired vertebral arteries form the basilar, giving off pontine perforators, AICA, and SCA, before terminating as the PCAs. PICA arises directly from the vertebral arteries; the labyrinthine artery typically arises from AICA.
Source: Wikimedia Commons

The two vertebral arteries ascend through the cervical transverse foramina and unite at the pontomedullary junction to form the basilar artery [5,6]. Along their course the vertebrals give off the posterior inferior cerebellar arteries (PICA), which supply the lateral medulla and posterior-inferior cerebellum [3,5] [5].

The basilar artery runs along the ventral pons and gives rise to the anterior inferior cerebellar arteries (AICA) — supplying the lateral pons and inner ear via the labyrinthine artery — and the superior cerebellar arteries (SCA) before terminating in the paired posterior cerebral arteries (PCA), which supply the midbrain, thalamus and occipital cortex [5,6,7]. Occlusion of any of these vessels produces territory-specific syndromes that the emergency clinician must recognise [6].

Table 1. Posterior Circulation Arterial Supply

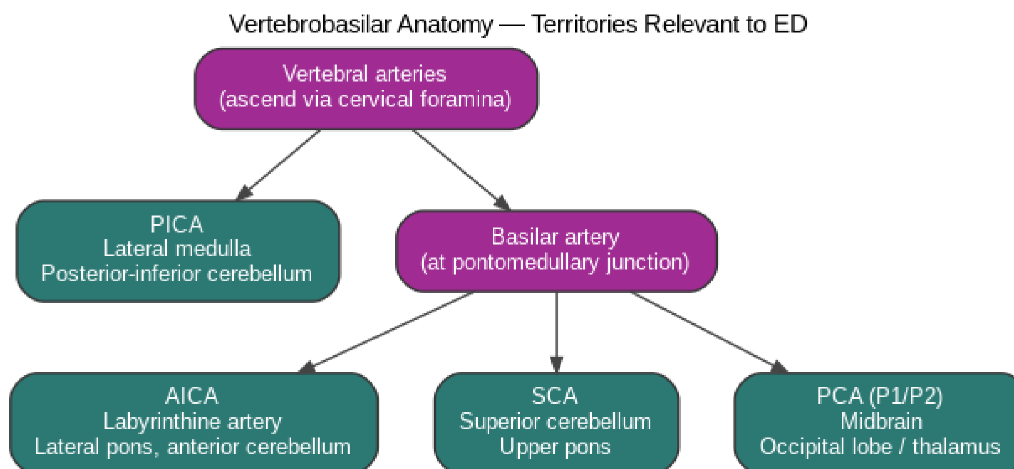

Figure 1. Vertebrobasilar arterial anatomy and territories.

Knowledge of brainstem vascular territories allows the clinician to predict the syndrome from the artery and vice versa [1,8]. PICA territory disease produces lateral medullary or cerebellar syndromes; AICA territory affects the lateral pons and labyrinth, producing combined vertigo and hearing loss; SCA territory causes upper-cerebellar and midbrain signs [7].

Watershed infarction at the boundary zones of these territories can produce mixed pictures, particularly in the setting of basilar stenosis or hypoperfusion [3,8]. CT angiography is the most useful first-line vascular study to define the calibre and patency of the entire posterior circulation [11,12] [16].

Artery	Key Branches	Territory
Vertebral Artery	PICA, medullary perforators	Lateral medulla, cerebellum
Basilar Artery	AICA, pontine perforators, SCA, PCA	Pons, midbrain, superior cerebellum, occipital lobes
AICA	Labyrinthine artery	Inner ear, labyrinth, lateral pons
PICA	Medullary, cerebellar branches	Lateral medulla, inferior cerebellum

III. PICA Territory Infarction

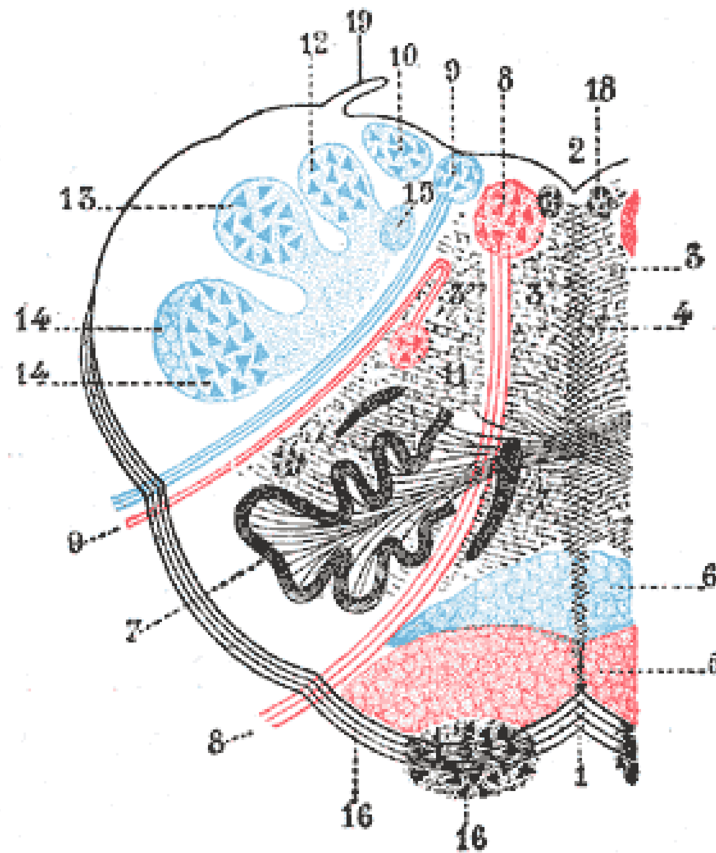

Figure 3. Transverse section through the medulla. The lateral medullary territory (supplied by PICA) contains the spinal trigeminal nucleus, vestibular nuclei, nucleus ambiguus, spinothalamic tract, and descending sympathetic fibres - accounting for the multifaceted clinical syndrome of Wallenberg.
Source: Wikimedia Commons

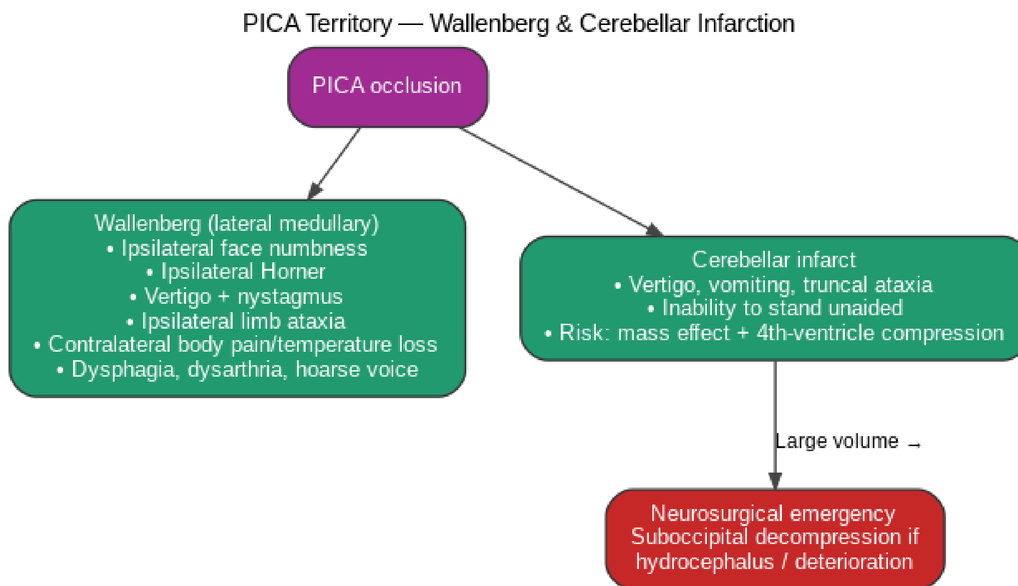

Figure 2. PICA-territory infarction — Wallenberg syndrome and cerebellar infarct [7].

Wallenberg Syndrome (Lateral Medullary Infarction)

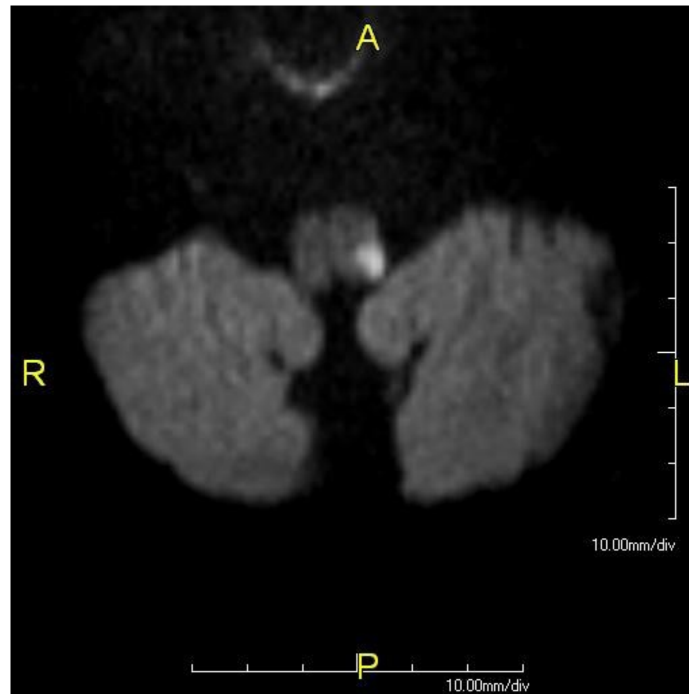

Figure 4. Diffusion-weighted MRI (axial) demonstrating restricted diffusion in the lateral medulla - the radiological signature of lateral medullary (Wallenberg) syndrome from PICA territory infarction.

Source: Wikimedia Commons

Lateral medullary infarction from PICA or vertebral-artery occlusion produces Wallenberg syndrome: ipsilateral facial sensory loss, ipsilateral Horner syndrome (ptosis, miosis, anhidrosis), vertigo with spontaneous nystagmus, ipsilateral limb ataxia, dysphagia, dysarthria, hoarse voice, and contralateral loss of pain and temperature over the body [1,6,13] [7].

Patients are often misdiagnosed with vestibular neuritis when the focal deficits are subtle [3,11]. A targeted cranial nerve and sensory examination in every AVS patient reduces missed cases [2,8].

 Clinical Pearl: Wallenberg syndrome: ipsilateral facial numbness + contralateral body sensory loss + vertigo + ataxia = lateral medullary stroke.

Cerebellar Infarction

Large PICA-territory cerebellar infarcts can cause progressive swelling with mass effect on the fourth ventricle and obstructive hydrocephalus [3,4]. Severe truncal ataxia with inability to stand unaided (astasia) is one of the strongest bedside predictors of cerebellar stroke in AVS and mandates urgent neuro-imaging regardless of HINTS findings [9] [7].

Deterioration in conscious level, new brainstem signs or evidence of hydrocephalus on imaging is a neurosurgical emergency — suboccipital decompression is potentially life-saving when performed early [3,4] [15].

The classical descriptions of Wallenberg syndrome by Adolf Wallenberg in 1895 remain accurate, but partial syndromes are common and often diagnostically challenging [1]. Isolated facial numbness with vertigo, isolated dysphagia with hiccups, or isolated Horner syndrome with ataxia should all prompt consideration of lateral medullary infarction [1,3].

⚠ **Important:** Cerebellar infarction with severe ataxia and inability to sit upright is a neurosurgical emergency. Obtain urgent CT and neurosurgery consultation.

IV. AICA Territory Infarction

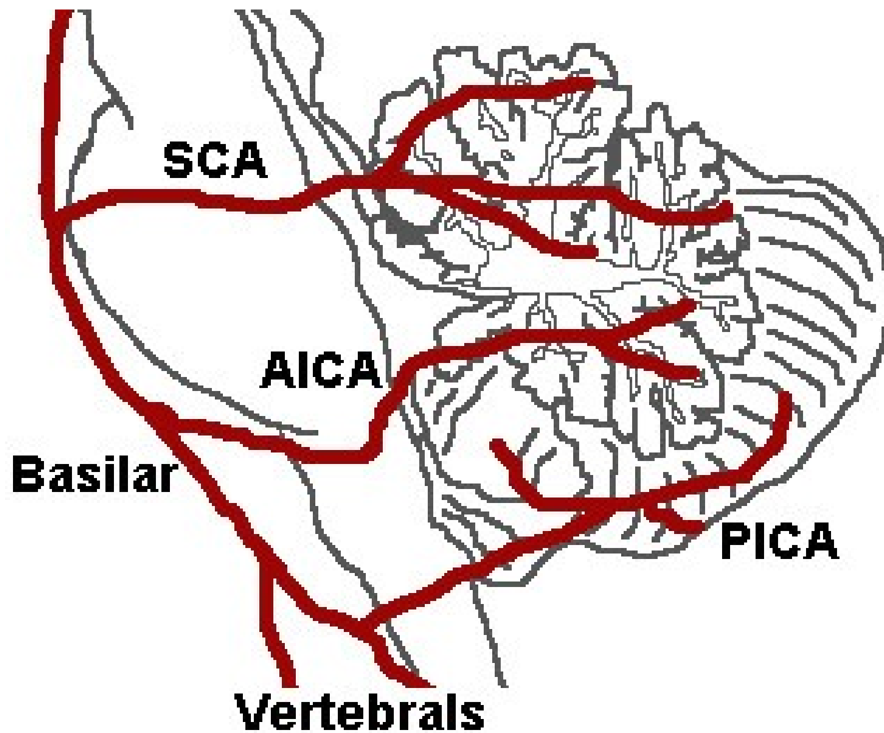

Figure 5. Posterior view of cerebellar arterial supply. The superior cerebellar artery (SCA) supplies the superior cerebellum, AICA the anterolateral cerebellum and flocculus (explaining co-occurrence of vertigo with hearing loss in AICA stroke), and PICA the posteroinferior cerebellum and lateral medulla.

Source: Wikimedia Commons

The anterior inferior cerebellar artery supplies the inner ear through the labyrinthine artery, together with the lateral pons and anterior cerebellum [3,5,7]. AICA-territory infarcts classically combine acute vertigo with ipsilateral sensorineural hearing loss — a combination that should never be attributed to vestibular neuritis without imaging [2,16] [9].

Additional features may include ipsilateral facial weakness or sensory loss, Horner syndrome, ipsilateral limb ataxia, and contralateral spinothalamic sensory loss [5,6]. AICA strokes represent roughly 40–50% of posterior-fossa infarcts seen in AVS cohorts [10,11] [9].

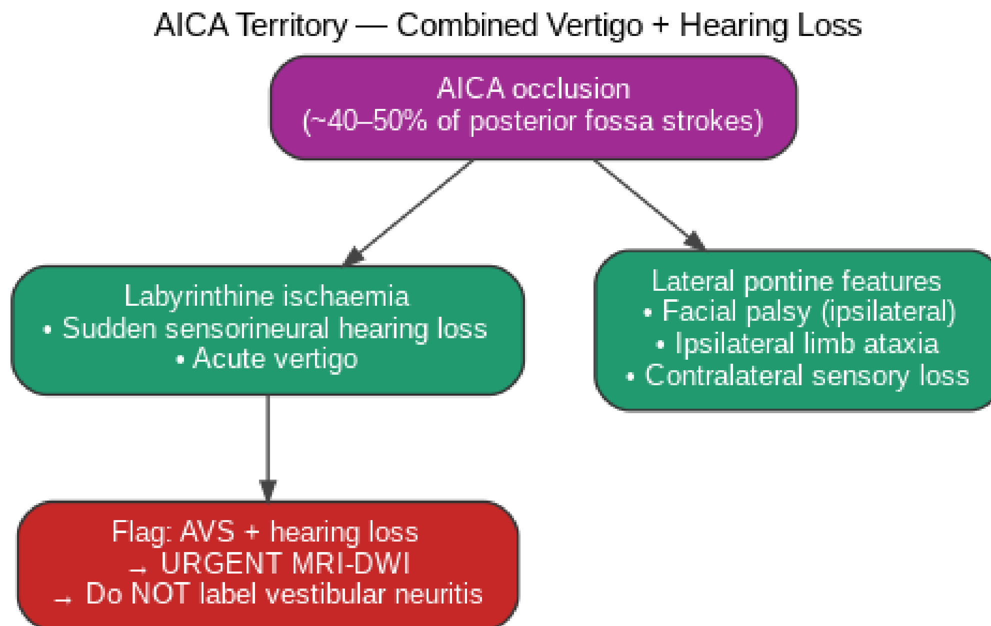

Figure 3. AICA-territory infarction — combined vertigo and hearing loss [9].

Acute audiovestibular loss — sudden vertigo with sensorineural hearing loss — should be regarded as AICA-territory stroke until vascular imaging excludes it [3,9]. The traditional teaching that hearing loss localises to the inner ear is unsafe in the emergency department: AICA infarcts that involve the labyrinthine artery present identically and have markedly worse prognosis [3,9,11] [9].

Audiometry, when available, can demonstrate the sensorineural pattern, but the immediate priority is vascular imaging and admission [9]. False reassurance from a normal HINTS examination is possible if the AICA infarct also disrupts the central vestibular pathways, producing a 'central HINTS' pattern with abnormal head impulse — a known limitation of the test [3,7].

> □ **Clinical Insight:** Acute hearing loss + acute vertigo in the same ear = assume AICA stroke; obtain urgent MRI.

V. Basilar Artery Pathology

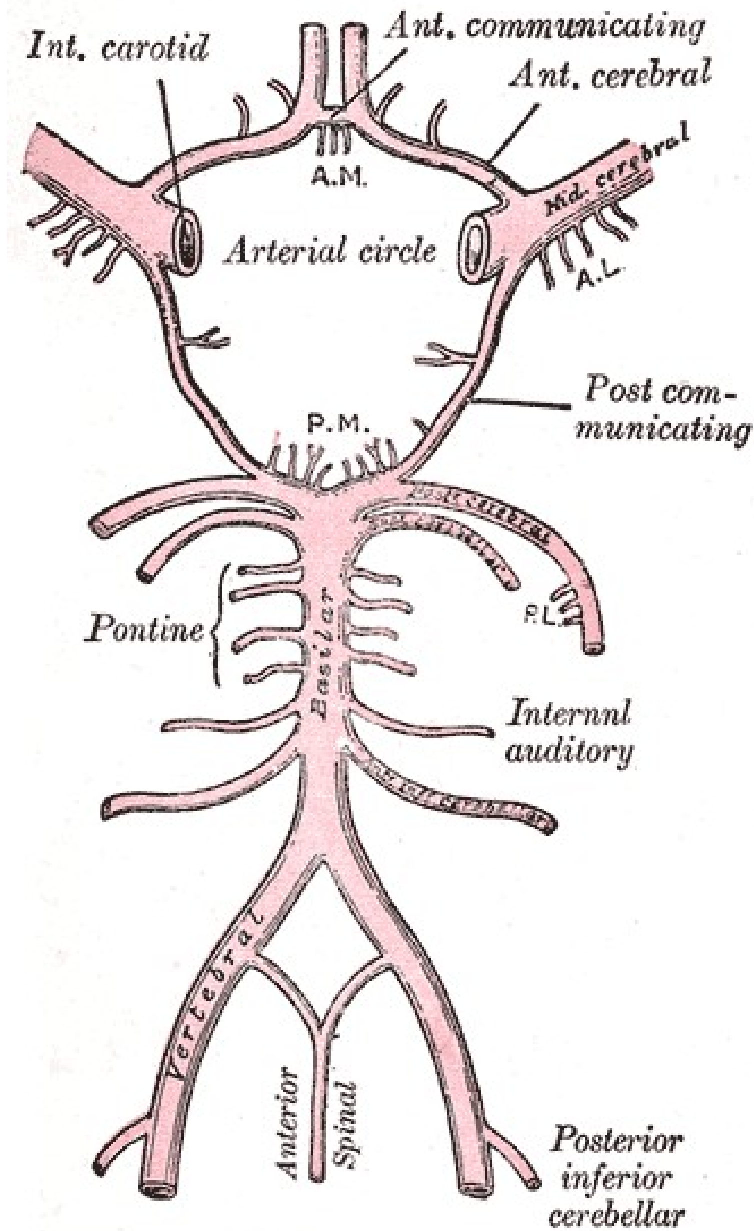

Figure 6. Classical anatomical plate of the arterial supply at the base of the brain. The basilar artery occupies the midline ventral to the pons and gives rise to pontine perforators before terminating in the PCAs - the vascular territory at risk in basilar occlusion.

Source: Wikimedia Commons

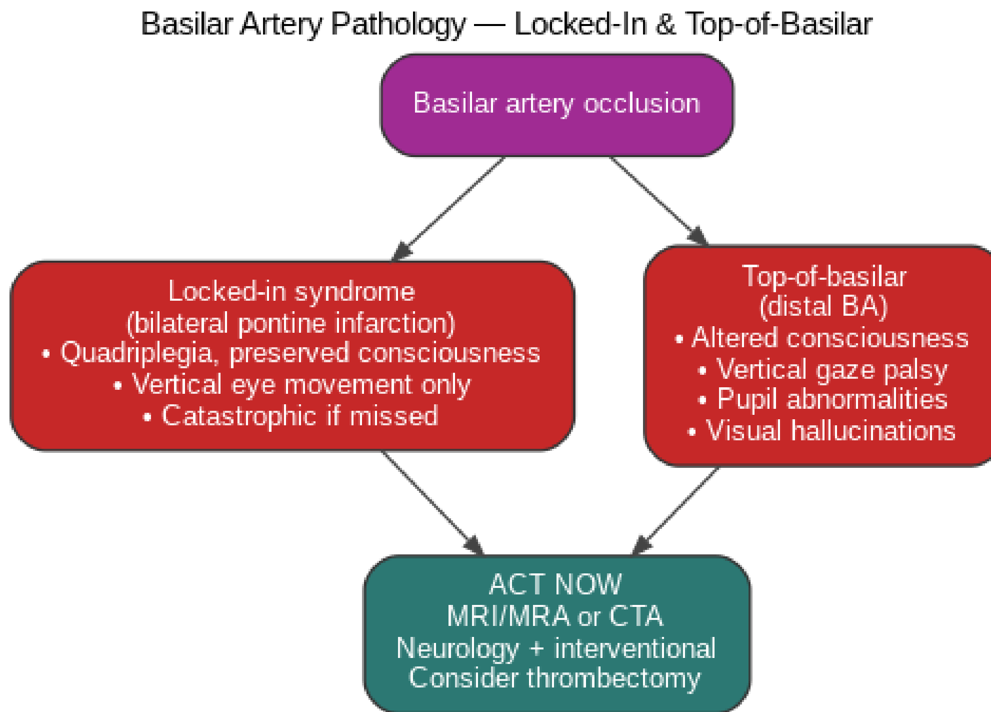

Figure 4. Basilar artery pathology — locked-in syndrome and top-of-basilar [13].

Locked-In Syndrome

Bilateral pontine infarction from basilar-artery occlusion causes locked-in syndrome: quadriplegia with preserved consciousness, where only vertical eye movements and blinking are volitionally controlled [5,6]. The patient may appear comatose but is fully aware, making early recognition critical [5,11] [13].

Any patient with sudden quadriparesis, cranial nerve palsies and preserved vigilance should be assumed to have basilar occlusion until proven otherwise — urgent MRA or CTA and immediate discussion with interventional neurology is mandatory [19,20].

⚠ **Important:** Locked-in syndrome: appearance of unresponsiveness with vertical eye movements preserved. This is massive brainstem stroke.

Top-of-Basilar Syndrome

Top-of-basilar syndrome results from distal basilar or bilateral posterior cerebral artery occlusion and produces midbrain, thalamic and occipital dysfunction [5,6,17]. Clinical features include altered consciousness, vertical gaze palsy, skew deviation, pupillary abnormalities, visual field loss, and vivid visual hallucinations (peduncular hallucinosis) [6,17].

These patients are often mislabelled as delirium, psychosis or intoxication if the ocular and brainstem signs are missed [11]. Early CTA or MRA and thrombectomy assessment are the priorities [19,20].

Time-to-recanalisation is the single most important determinant of outcome in basilar occlusion [3,18]. Delay of even one hour in initiating thrombectomy substantially worsens functional outcome at 90 days [18,20].

VI. Cerebellar Hemorrhage

Cerebellar haemorrhage is a neurosurgical emergency [3,4]. The haematoma causes posterior-fossa mass effect, compression of the fourth ventricle and obstructive hydrocephalus — deterioration can be rapid and catastrophic [3,4] [15].

Typical presentation is acute severe posterior headache, vomiting, vertigo, truncal ataxia and inability to stand, followed by depressed consciousness if untreated [3,4]. Non-contrast CT is usually diagnostic; neurosurgical referral and early decompression markedly improve outcome in patients with haematoma volume >3 cm or clinical deterioration [4].

Cerebellar haematomas exceeding 3 cm in maximum diameter, or any haematoma causing brainstem compression or hydrocephalus, are a neurosurgical emergency requiring urgent suboccipital decompression [3,4]. Mortality without surgery exceeds 80% once brainstem compression develops; with timely surgery, functional recovery can be excellent [3,4,11] [15].

> ⚠ **Important:** Cerebellar hemorrhage: acute posterior headache + vertigo + rapid deterioration = neurosurgical emergency.

VII. Clinical Presentation Patterns

Small posterior-fossa infarcts commonly mimic vestibular neuritis: acute sustained vertigo, vomiting, and head-motion intolerance without obvious focal neurology [2,3,11]. This is the classic missed-stroke scenario and the main reason AVS warrants a structured bedside examination in every case [2,8].

Larger strokes tend to present with ‘vertigo-plus’ syndromes — vertigo accompanied by facial numbness, hearing loss, dysphagia, hemisensory loss or gait-disabling ataxia [5,11]. Any extra-vestibular sign in an AVS patient should be treated as a central red flag [1,2].

Recognising 'vertigo-plus' syndromes is the single most important skill for emergency clinicians: vertigo accompanied by any other neurological symptom — diplopia, dysarthria, dysphagia, weakness, numbness, ataxia — is a posterior-circulation stroke until proven otherwise [3,11,12].
Equally important is recognising that small posterior-fossa infarcts can mimic vestibular neuritis with no additional features [2,11]. The HINTS examination, gait assessment, and a low threshold for MRI in patients with vascular risk factors are the operational safety net [2,5,7].
Beyond classical brainstem syndromes, partial or evolving deficits are common and easily underweighted at first contact [3,4]. A patient with mild dysarthria and ataxia at presentation may deteriorate over hours as a basilar thrombus extends — repeat focused examination at 30-minute intervals during the early ED phase is the safest practice in dizzy patients with any concerning feature [3,11,12].

> **Key Point:** *Posterior circulation stroke often presents with vertigo, but additional neurological signs (facial numbness, hearing loss, sensory loss, severe ataxia) indicate central pathology.*

VIII. Diagnostic Approach

Non-contrast CT brain is insensitive for acute posterior-fossa ischaemia, with sensitivities of only 10–30% in the first 24 hours and well-described false negatives for small cerebellar and brainstem infarcts [2,5]. CT remains useful to exclude haemorrhage but should not be used to rule out stroke in AVS [4,5].

MRI with diffusion-weighted imaging is the gold standard, with sensitivity of approximately 90–95% for acute infarction, although false-negative DWI within the first 24–48 hours is recognised in small posterior-fossa lesions [2,5,11]. The National Institutes of Health Stroke Scale (NIHSS) is weighted toward anterior-circulation deficits and can be low or zero in significant posterior-circulation stroke — it should not be used alone to triage the dizzy patient [5,15].

Table 2. Imaging for Posterior Circulation Stroke

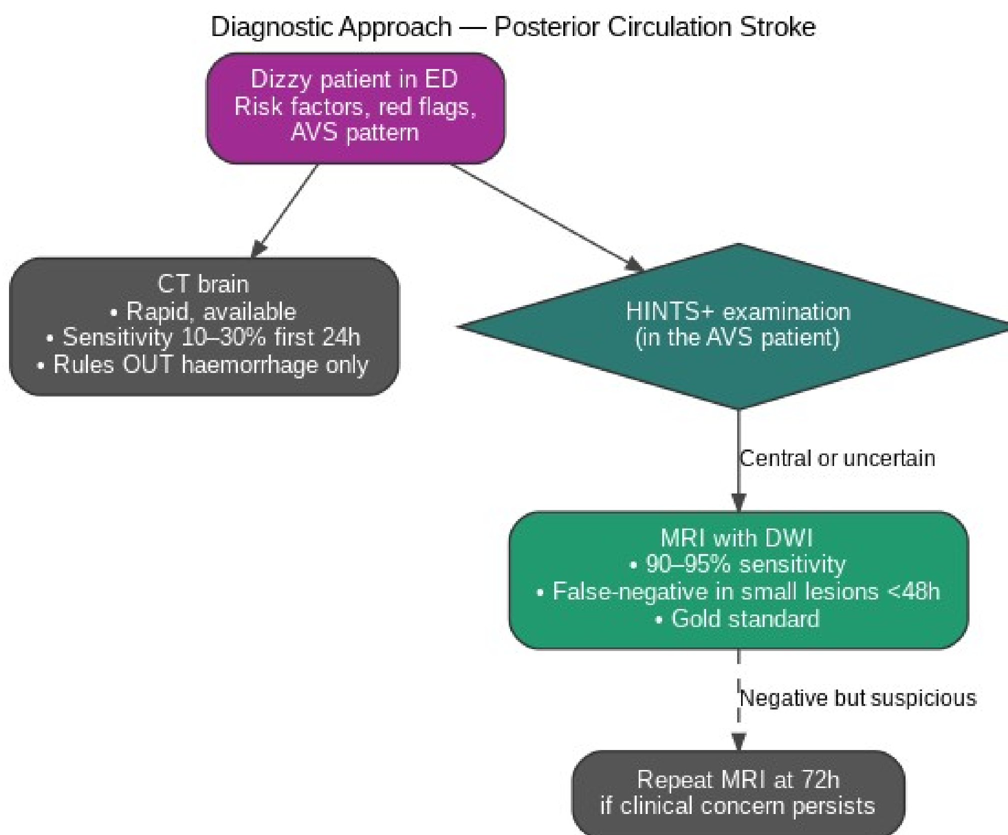

Figure 5. Diagnostic pathway for suspected posterior-circulation stroke.

When MRI is unavailable or contraindicated, a strategy of CTA combined with prolonged observation, repeat clinical examination, and delayed MRI offers the next-best safety profile [11,12]. Discharge of an AVS patient without MRI should only occur where bedside testing is unequivocally peripheral, performed by an experienced examiner, and where vascular risk is low [2,4,7].

MR angiography of the head and neck (MRA) provides additional information on vessel patency, plaque morphology and dissection, and should be combined with diffusion-weighted MRI in suspected posterior-circulation stroke [11,16,17]. CT perfusion can help identify salvageable penumbra in candidates for endovascular intervention beyond the standard window.

Modality	Sensitivity	Advantages/Disadvantages
CT Brain	10–30%	Fast; low sensitivity for posterior fossa
CTA	70–90% large vessel occlusion (LVO)	Identifies large vessel occlusion
MRI/DWI	90–95%	Gold standard; slower
HINTS Exam	100%*	Rapid bedside triage tool

IX. Time-Critical Management

Intravenous thrombolysis with tPA is indicated within 4.5 hours of onset in eligible patients without contraindications [18]. Mechanical thrombectomy is the treatment of choice for large-vessel occlusion, including basilar-artery occlusion, and extended windows up to 24 hours apply in selected cases [19,20].

Concurrent priorities include close neurological observation for deterioration, blood-pressure and glycaemic control, and early neurosurgical input when cerebellar haematoma, large cerebellar infarct or hydrocephalus is present [3,4]. Admission to a stroke unit or ICU is appropriate for most confirmed posterior-circulation strokes [18] [15].

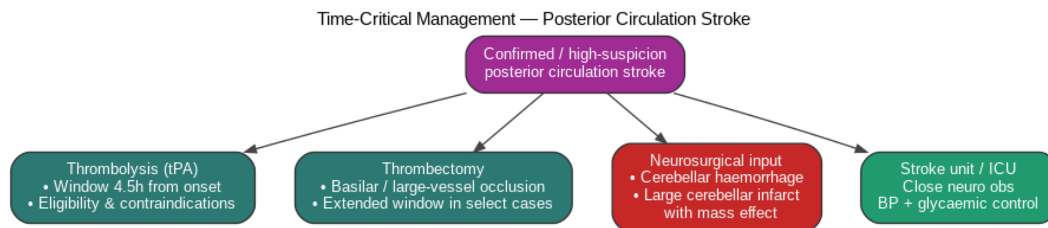

Figure 6. Time-critical management of posterior-circulation stroke.

Posterior-circulation thrombectomy windows have lengthened with the BASICS and BAOCHE trials supporting intervention up to 24 hours from onset in selected patients with basilar occlusion [18,20]. Clinicians should not assume that a delayed presentation excludes intervention — urgent neurology and neurointervention review is warranted [18,20] [19].
Adjunctive priorities include strict blood-pressure parameters (typically <180/105 mmHg post-thrombolysis, individualised for collateral-dependent territories), normothermia, normoglycaemia, and aspiration precautions for patients with bulbar involvement [4,18].

> ⚠ **Important:** Posterior circulation stroke is time-sensitive. Early recognition and thrombolysis/thrombectomy can be life-saving.

X. Conclusions

Posterior-circulation stroke is common and frequently missed at first contact because small infarcts mimic benign vestibular disease [2,3,11]. A high index of suspicion, structured HINTS+ bedside examination, gait testing and liberal use of MRI-DWI narrow the diagnostic gap and support appropriate disposition [2,8,9].

Early recognition enables time-critical reperfusion therapy and, where indicated, neurosurgical intervention — both of which substantially change outcome [4,18,19,20].

> □ **Clinical Insight:** Posterior circulation stroke accounts for 15–20% of strokes but is often missed because it presents with vertigo. Early recognition is critical.

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