Cervical Arterial Dissection — Cheat Sheet for Vestibular Physicians

The stroke not to miss in the young patient. In a continuous AVS, a normal head-impulse is the danger sign — image the vessel wall, not just the lumen.

► Why cervical arterial dissection matters

The leading cause of ischaemic stroke in young and middle-aged adults and the archetypal stroke not to miss. Population incidence ~2.6–3/100,000/yr (under-counted: painless or minor cases are missed). It accounts for up to ~25% of ischaemic stroke under age 50 (15% in the Helsinki Young Stroke Registry). Internal carotid dissection (ICAD) is ~twice as common as vertebral dissection (VAD); ICAD ~46 yr, male-predominant; VAD ~42 yr, sexes even. Outcomes are favourable — mortality ~2%, mRS 0–1 in ~80% — so the prize is recognition before the embolic stroke.

► When to apply this work-up

► *Young or middle-aged patient with acute head, face, orbital or neck pain — especially new, unilateral and unlike previous headaches.*
► *Painful partial Horner syndrome, pulsatile tinnitus, or lower cranial-nerve palsy on the painful side.*
► *Acute vertigo, posterior-circulation symptoms or any focal deficit after recent neck trauma, strain, or manipulation.*
► *Isolated acute vertigo with vascular risk for dissection — it can be the only sign of VAD.*

► Mechanism — the intramural haematoma and the vulnerable wall

Layer	Mechanism	Clinical relevance
Intramural haematoma	Blood splits the arterial wall, creating a false lumen between layers.	The defining lesion; trauma may be trivial or absent, pointing to an underlying wall predisposition.
Subintimal (inward)	False lumen bulges into the true lumen → stenosis or occlusion.	The ischaemic phenotype — thromboembolism is the dominant stroke mechanism, not pure hypoperfusion.
Subadventitial (outward)	Haematoma expands outward → pseudoaneurysm; intracranial vessels lack a strong adventitia.	Pseudoaneurysm usually benign; intracranial VAD risks SAH (~6% vs ~0.6% extracranial).
Vulnerable segments	Distal extracranial ICA (pharyngeal) and vertebral V2/V3 (atlas loop) — mobile, stretched over bone.	Mobility + tethering = the sites where rotation and stretch tear the wall.
Underlying arteriopathy	Connective-tissue ultrastructural abnormality in ~half to two-thirds; FMD ~20% (PHACTR1); overt CTD under 8%.	Explains spontaneous and minor-trauma dissection; screen for FMD and a connective-tissue background.

Pearl — Image the wall, not just the lumen — the haematoma sits inside the vessel wall. A T1 fat-saturated MRI crescent sign shows it directly when luminal imaging looks normal.

► Clinical features — carotid (ICAD) vs vertebral (VAD)

Domain	Carotid dissection (ICAD)	Vertebral dissection (VAD)
Pain	Head, face, orbital, anterolateral neck pain.	Posterior neck / occipital pain; may be thunderclap.
Local signs	Partial Horner (ptosis + miosis, sweating preserved); pulsatile tinnitus; CN XII palsy.	Fewer local signs; pain often the only warning before ischaemia.
Ischaemia	Anterior-circulation TIA/stroke; amaurosis fugax.	Posterior-circulation stroke — Wallenberg, PICA; AICA → audiovestibular loss.
Dizziness	Less prominent.	Vertigo in ~58%; isolated vertigo can be the only sign; stroke within 7 days in 82% (Biousse).

Pearl — Painful Horner plus recent neck strain in a young patient is dissection until excluded. Sweating fibres run with the external carotid, so the Horner is partial — ptosis and miosis without anhidrosis.

► Bedside examination — the AVS patient

Sign	Peripheral (reassuring)	Central / dissection (danger)
Head-impulse	Abnormal (corrective saccade) on the affected side.	NORMAL head-impulse in a continuous AVS — the single most useful danger sign.
Nystagmus	Unidirectional, horizontal, fixation-suppressed.	Direction-changing or vertical / torsional nystagmus.
Skew	Absent.	Vertical skew deviation on cover–uncover.

Sign	Peripheral (reassuring)	Central / dissection (danger)
Hearing / gait	Hearing intact; can stand and walk.	AICA pattern adds hearing loss (use HINTS-plus); inability to stand unaided.

Pearl — Normal head-impulse in a continuous AVS is the single most useful danger sign — HINTS outperforms early MRI for posterior-circulation stroke. Add hearing testing (HINTS-plus) because AICA territory can lie to a normal head-impulse.

► Imaging — confirm the wall lesion

Modality	Role	Practice points
T1 fat-sat MRI + DWI	Direct mural-haematoma imaging (crescent sign) plus infarct detection.	Crescent sign is the most specific finding; DWI confirms ischaemia. First-line wall imaging.
CTA / MRA	Luminal assessment — stenosis, occlusion, pseudoaneurysm.	Comparable diagnostic accuracy; CTA fast and accessible in the acute setting.
DSA	Reference standard.	String sign, flame-shaped occlusion, double lumen, pseudoaneurysm; reserved for equivocal or interventional cases.
Ultrasound	Screening adjunct.	~70% sensitivity; blind to the high cervical ICA and V3 — a normal scan does not exclude dissection.
Vessel-wall MRI	Emerging.	High-resolution mural imaging; growing role in equivocal cases at experienced centres.

Pearl — CTA and MRA have comparable accuracy — image with whatever you obtain fastest, then add T1 fat-sat for the wall. A normal carotid ultrasound never excludes a high-ICA or V3 dissection.

► Red flags — Isolated acute vertigo can be the only sign of vertebral dissection. A NORMAL head-impulse in a continuous AVS, direction-changing or vertical nystagmus, skew deviation, new hearing loss, or inability to stand unaided → image the vessels. Add neck/occipital pain, partial Horner, recent neck trauma or manipulation, age under 50, or a connective-tissue background and the threshold to image falls further.

► Management — antithrombotic first, intervention selectively

Tier	Intervention	Practice principles
Antithrombotic	Antiplatelet OR anticoagulation — both acceptable, 3–6 months, then re-image.	CADISS: equivalent, ~2% recurrence. TREAT-CAD did not show aspirin non-inferiority. IPD meta-analysis: anticoagulation fewer strokes but more bleeds.
Acute reperfusion	IV thrombolysis when otherwise eligible; thrombectomy for large-vessel occlusion.	Dissection is NOT a contraindication to thrombolysis. Treat the occlusion, not the dissection label.
Endovascular / surgery	Stenting only for refractory ischaemia or an enlarging pseudoaneurysm.	Most pseudoaneurysms are benign and medically managed; intervention is the exception.
Intracranial + general	Early intervention for intracranial VAD with SAH; avoid neck manipulation; control BP.	SAH changes the calculus — secure early. Counsel against high-velocity neck manipulation while healing.

Pearl — Dissection is not a contraindication to thrombolysis — eligible patients within the window should be treated. For prevention, antiplatelet and anticoagulation are broadly equivalent over 3–6 months; re-image before deciding to stop.

► Prognosis and follow-up

► *Recanalisation in ~70% (up to ~90% on serial MRI) by 3–6 months — re-image before stopping antithrombotic therapy.*
► *Recurrence ~1% per year; higher with a family history or a connective-tissue disorder.*
► *Pseudoaneurysms are generally benign and rarely a source of late embolism.*
► *Mortality ~2%; ~80% reach mRS 0–1 — recognition before the disabling stroke is the outcome that matters.*

Key references — Schievink. NEJM 2001 · Debette & Leys. Lancet Neurol 2009 · CADISS. Lancet Neurol 2015 · Engelter et al. TREAT-CAD. Lancet Neurol 2021 · Putaala et al. Stroke 2009.