Cervical Arterial Dissection:

A Vestibular Physician's Deep Review of Vascular Vertigo, Stroke Risk, and Management

Vestibular Medicine for Vestibular Physicians

Central Vestibular Pathology — Module 3.5

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of central vestibular practice, where a working command of mechanism, localisation, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

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

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Cervical arterial dissection (CAD) — dissection of the extracranial internal carotid artery (ICAD) or vertebral artery (VAD) — is the archetypal stroke not to miss in the dizzy patient. It is uncommon in absolute terms yet accounts for a strikingly large share of stroke in the young, and it frequently announces itself with pain, vertigo, or a partial Horner sign days before the disabling infarct arrives [1,2]. For the vestibular physician the lesson is unambiguous: dissection lives in the differential of every acute vestibular syndrome, every new unilateral neck or occipital pain, and every young patient with vertigo after trivial neck trauma or manipulation [1,16].

The population incidence of spontaneous CAD is approximately 2.6 to 3 per 100,000 person-years, and is almost certainly underestimated because painless or minor presentations never reach a vascular service [4,6,44]. Internal carotid dissection is roughly twice as common as vertebral dissection in most series. The two diverge in demography: in the largest direct comparison (668 spontaneous ICAD versus 302 spontaneous VAD), carotid patients were older (46.3 vs 42.0 years) and more often male (62.7% vs 53.0%), while vertebral dissection skewed younger and was more evenly distributed by sex [5]. A modest autumn peak in incidence has been described and remains unexplained [1].

The epidemiological signature that matters most clinically is the age distribution of stroke aetiology. In the Helsinki Young Stroke Registry of 1,008 consecutive patients aged 15 to 49 with a first-ever ischaemic stroke, cervicocerebral dissection was the single identified cause in 15% — second only to cardioembolism — making it one of the leading mechanisms of stroke in young adults [3]. Consensus reviews place dissection behind up to a quarter of ischaemic strokes in patients under 50, falling to only 2 to 3% across all ages as atherosclerotic mechanisms dominate later life [1,2,44]. Dissection is therefore a disease whose pre-test probability is dictated by the patient in front of you: high in the young, the post-traumatic, and the connective-tissue patient; low but never zero in the elderly [4].

Table 1. Cervical arterial dissection — epidemiology at a glance [1,3,4,5,6].

Measure	Value	Notes
Incidence (spontaneous CAD)	~2.6–3 / 100,000 / year	Likely underestimated; ICAD roughly 2× VAD [4,6]
Share of ischaemic stroke, age under 50	up to ~25% (15% in Helsinki registry)	Second commonest identified mechanism in the young [3]
Mean age	ICAD ~46 yr; VAD ~42 yr	ICAD older and more male than VAD [5]
Sex	ICAD male-predominant; VAD ~even	M 62.7% (ICAD) vs 53.0% (VAD) [5]
Seasonality	Modest autumn peak	Mechanism unknown [1]
Mortality	Low (~2%)	Good functional outcome in ~80% [11,12]

Outcomes are, in aggregate, favourable. Mortality is around 2%, and most survivors reach a modified Rankin score of 0 to 1; functional recovery does not differ materially from young stroke of other causes [11,12,13]. This benign statistical horizon, however, conceals the early hazard: the interval between the first warning symptom and completed stroke can be very short, and it is in that window that recognition changes the trajectory [7].

□ **Key Point:** Cervical arterial dissection is rare overall but causes up to a quarter of ischaemic strokes in adults under 50. In the dizzy patient it is the vascular diagnosis whose probability rises sharply with youth, recent neck trauma or manipulation, and a connective-tissue background.

II. Pathophysiology — Intramural Haematoma and the Vulnerable Arterial Wall

The defining lesion of dissection is an intramural haematoma: blood within the arterial wall, splitting its layers and creating a false lumen [1]. Two initiating mechanisms are recognised and probably coexist. In some patients an intimal tear admits luminal blood into the media under arterial pressure; in others the

primary event is rupture of the vasa vasorum, producing a haematoma that expands within the wall without any demonstrable intimal breach (primary medial haematoma) [1,43]. Either way, the wall is now structurally compromised and the lumen geometry distorted.

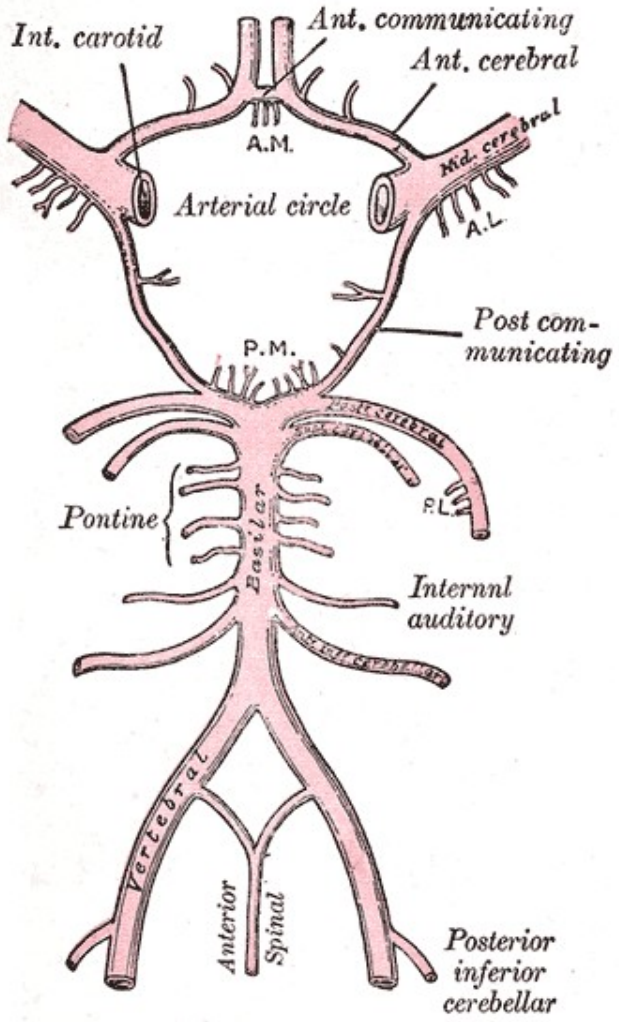

Figure 1. The internal carotid and vertebral arteries and the arterial circle — the cervical vessels at risk of dissection and the posterior-circulation territory they feed.

Source: Henry Vandyke Carter, Gray's Anatomy of the Human Body (1918). Public domain, via Wikimedia Commons.

The clinical consequence depends on which way the haematoma expands [1]. A subintimal dissection bulges inward, narrowing or occluding the true lumen; this is the substrate of ischaemia, whether by artery-to-artery thromboembolism from the dissection flap or by haemodynamic low-flow distal to a tight stenosis [1,43]. A subadventitial dissection bulges outward, producing an aneurysmal out-pouching — a dissecting pseudoaneurysm. Subadventitial dissection of an intracranial segment carries the additional, feared possibility of subarachnoid haemorrhage when the thin intracranial adventitia ruptures; in the von Babo comparison, subarachnoid haemorrhage occurred in 6.0% of vertebral versus 0.6% of carotid dissections, essentially confined to intracranial disease [5,15].

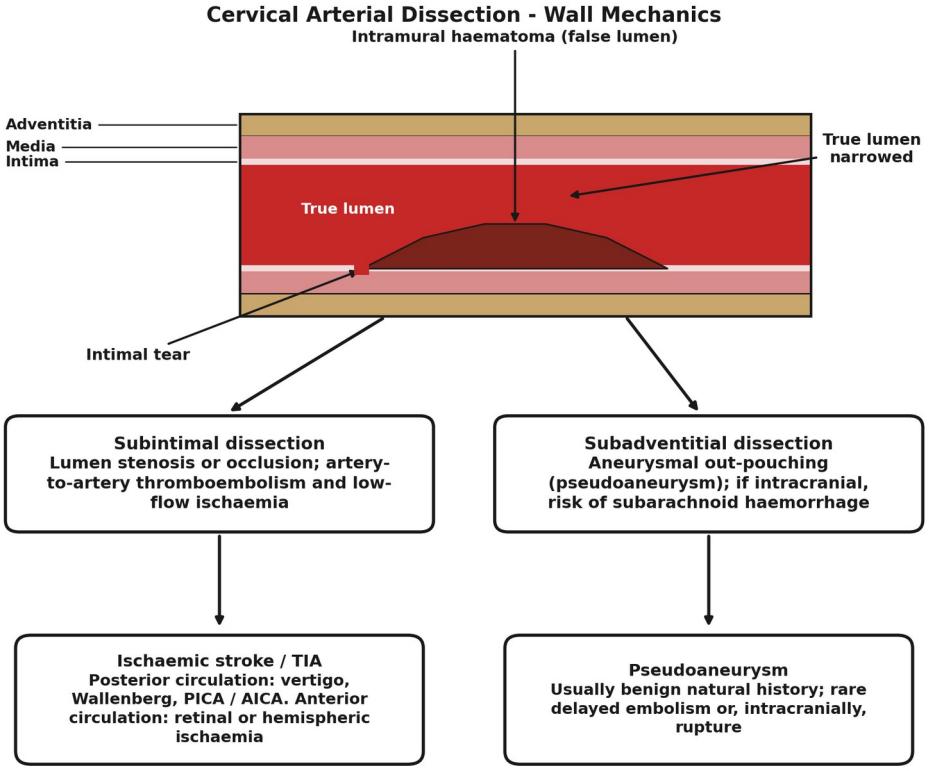

Figure 2. Wall mechanics of cervical arterial dissection — intramural haematoma and the divergent subintimal (ischaemic) and subadventitial (aneurysmal) consequences.

Source: Australian Dizziness Clinics. Adapted from Schievink [1] and Debette and Leys [2].

The vulnerable segments

Dissection is a disease of arterial mobility. The cervical vessels are tethered proximally and distally but run a mobile course in between, where stretching and shearing over bone concentrate mechanical stress [1]. For the internal carotid this is the distal extracranial pharyngeal segment, two to three centimetres above the bifurcation and up to the skull base, where the artery can be stretched over the upper cervical vertebrae [1,43]. For the vertebral artery the mobile V2 (pars transversaria) and V3 (atlas loop) segments dominate: in Arnold's series of 169 vertebral dissections, the lesion localised to V2 in 35% and V3 in 34%, far more than the relatively fixed V1 (20%) or intracranial V4 (11%) [12]. The V3 atlanto-axial loop, which must accommodate the full range of head rotation at C1–C2, is the classic stretch point and the commonest site in paediatric vertebral dissection [12,14].

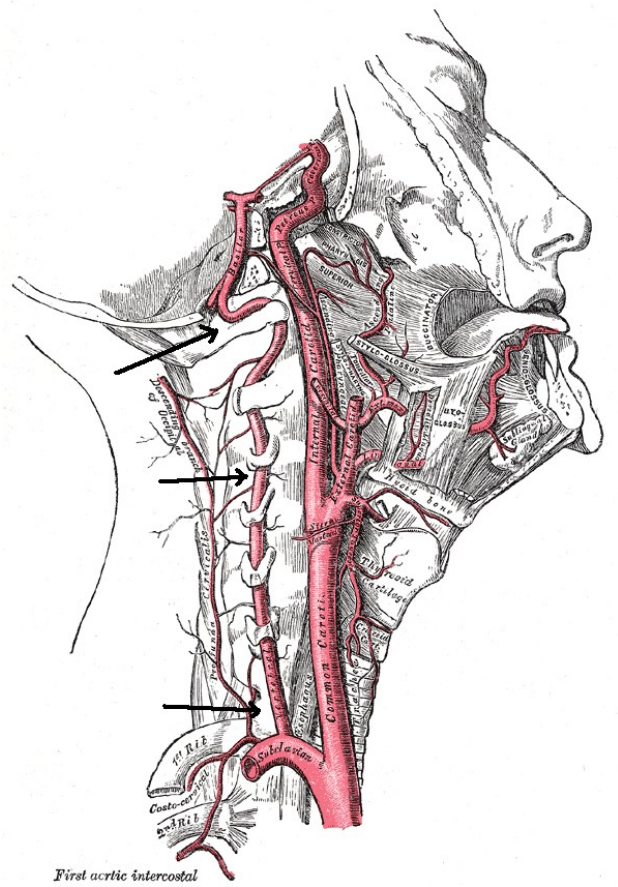

Figure 3. Lateral anatomy of the neck — the mobile distal extracranial internal carotid and the vertebral artery looping through the transverse foramina (arrows), the segments most prone to dissection.
Source: Gray's Anatomy of the Human Body (1918), annotated by M. Häggström. Public domain, via Wikimedia Commons.

The underlying arteriopathy

That trivial or even absent trauma can tear an artery points to an underlying structural predisposition. Skin-biopsy electron microscopy reveals subtle connective-tissue ultrastructural abnormalities — irregular collagen fibrils and elastic-fibre fragmentation reminiscent of a mild Ehlers-Danlos phenotype — in roughly half to two-thirds of spontaneous CAD patients, the great majority of whom have no clinically overt connective-tissue disease [34,35,36]. Rare copy-number variants enriched for extracellular-matrix and collagen genes (COL3A1, COL5A2) have been identified in the subset with abnormal dermal ultrastructure, supporting a genuinely heritable matrix defect [37]. At the common-variant level, a genome-wide association study identified the PHACTR1 locus (rs9349379) as a susceptibility allele shared with migraine and inversely with myocardial infarction [33]. Overt heritable disorders — vascular (type IV) Ehlers-Danlos syndrome, Marfan syndrome, and alpha-1-antitrypsin deficiency — are classically associated but explain only a small minority; a clinically apparent connective-tissue disease is documented in fewer than 8% of cases [1,11].

Fibromuscular dysplasia is the most frequently identified associated arteriopathy. In the United States FMD Registry of 447 patients, nearly one in five (19.7%) had experienced an arterial dissection, and the registry population was 91% female [9,10]. Other epidemiological associations — recent infection, migraine, hyperhomocysteinaemia, and mechanical neck trauma — are best understood as triggers acting on a predisposed wall rather than independent causes [2,16]. This two-hit model — a vulnerable matrix plus a mechanical or inflammatory trigger — reconciles the spectrum from spontaneous dissection during sleep to dissection after a roller-coaster ride or chiropractic manipulation [2,16,38].

□ **Clinical Insight:** Most spontaneous dissection occurs in a structurally predisposed but clinically normal-looking artery. Absence of a connective-tissue diagnosis or a history of major trauma does not lower the index of suspicion — the typical patient has neither.

III. Clinical Features — Local Signs, Ischaemia and the Dizzy Presentation

Dissection produces two families of symptoms: local symptoms from the wall lesion itself, and ischaemic symptoms from its embolic or haemodynamic consequences [1,2]. The local symptoms are the early-warning system, and recognising them is where vestibular and neuro-otology clinics add value, because they often precede the stroke by hours to days [7].

Local symptoms

Pain is the commonest and earliest symptom of dissection, present in the large majority of symptomatic patients [11,12]. Its character is diagnostic when the pattern is recognised: ipsilateral head, facial, orbital or anterolateral neck pain in carotid dissection, and posterior neck or occipital pain in vertebral dissection. Neck pain is far more frequent in vertebral than carotid disease (65.8% vs 33.5%), and thunderclap-onset headache, although uncommon, is likewise commoner in vertebral dissection (9.2% vs 3.6%) and should raise the possibility of intracranial extension and subarachnoid haemorrhage [5,11].

A partial Horner syndrome — ptosis and miosis with preserved facial sweating (oculosympathetic paresis) — is the classic local sign of carotid dissection, produced when the expanding wall stretches the sympathetic plexus running on the internal carotid. The painful Horner sign, with or without delayed cerebral or retinal ischaemia, is the textbook triad and is effectively pathognomonic in the right setting [1,16,43]. Pulsatile tinnitus is a further carotid-predominant local symptom (10.9% vs 3.4%), and lower cranial-nerve palsies — most often the hypoglossal — arise when a high cervical carotid pseudoaneurysm compresses the nerves near the skull base [1,5,16].

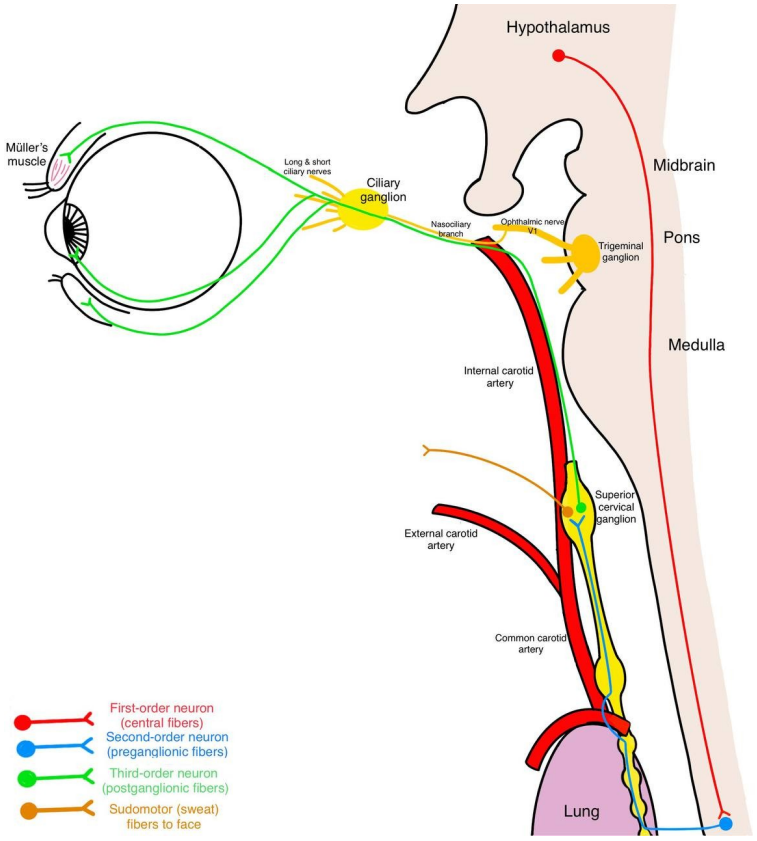

Figure 4. The oculosympathetic pathway. Carotid dissection interrupts the third-order (postganglionic) sympathetic fibres travelling on the internal carotid artery, producing a painful partial Horner syndrome with preserved facial sweating.

Source: Lina Khanna, via Wikimedia Commons (CC0 1.0).

Ischaemic symptoms and the dizzy presentation

Carotid dissection produces anterior-circulation ischaemia — transient or completed hemispheric stroke, amaurosis fugax and retinal infarction. The timing is the clinically decisive feature: in Biousse's series of 80 carotid dissections the interval from the first local symptom to ischaemic stroke was seven days or less in 82% of patients, though it could extend to a month [7]. This narrow window is the rationale for starting antithrombotic therapy urgently the moment dissection is suspected, and for continuing it even in patients who present late [7,15].

Vertebral dissection is the form most likely to reach a vestibular clinic, because the posterior circulation it supplies governs the vestibular pathways. Dizziness or vertigo is the single most common symptom of vertebral dissection, reported in 58% of patients in a systematic review of nearly 2,000 cases [11]. The territorial syndromes are familiar: lateral medullary (Wallenberg) syndrome from posterior inferior cerebellar artery (PICA) or distal vertebral involvement, PICA-territory cerebellar infarction, and — when the dissection propagates to compromise the anterior inferior cerebellar artery (AICA) — a combined audiovestibular syndrome [8,11,18]. In a consecutive series of 130 pure lateral medullary infarcts, dissection was the underlying mechanism in 15% [8].

The presentation that most often deceives is isolated vertigo. A cerebellar infarct can mimic vestibular neuritis exactly — acute prolonged vertigo, spontaneous nystagmus, gait unsteadiness and vomiting, with no other localising sign. In Lee's study of 240 isolated cerebellar infarctions, 25 (10.4%) presented as such an isolated vestibular syndrome, and 96% of these pseudo-neuritis cases lay in the medial branch of the PICA territory [17,19]. AICA-territory infarction adds a cochlear dimension: in a prospective series of 82 AICA infarcts, acute prolonged vertigo occurred in 98% and combined auditory plus vestibular loss was the commonest pattern, because AICA supplies the labyrinth through the internal auditory artery [18]. Sudden sensorineural hearing loss can therefore be the heralding symptom of vertebrobasilar — including dissection-related — ischaemia rather than a benign cochlear event [42].

Table 2. Internal carotid versus vertebral dissection — comparative clinical profile [5,7,11,12].

Feature	Internal carotid dissection	Vertebral dissection
Typical age / sex	Older (~46 yr), male-predominant	Younger (~42 yr), sexes even
Pain	Head / face / orbit / anterolateral neck	Posterior neck / occipital; thunderclap commoner
Hallmark local sign	Partial Horner, pulsatile tinnitus, CN XII palsy	Neck pain; few local signs
Ischaemic territory	Anterior circulation; retinal ischaemia	Posterior circulation; Wallenberg, PICA, AICA
Dizziness / vertigo	Uncommon as presenting symptom	Common (~58%); may be isolated vertigo
Subarachnoid haemorrhage	Rare (0.6%)	6% — intracranial (V4) disease [5]

□ **Important:** Isolated acute vertigo can be the only manifestation of vertebral dissection. A normal head-impulse test, direction-changing or vertical nystagmus, skew deviation, or inability to stand unaided in a patient who otherwise looks like vestibular neuritis are red flags for a posterior-circulation stroke and mandate vessel imaging.

IV. Recognising Dissection at the Bedside — HINTS and Red Flags

Because dissection so often hides inside an acute vestibular syndrome, the decisive skill is separating a peripheral from a central cause at the bedside. The HINTS battery — Head-Impulse, Nystagmus, Test-of-Skew — was designed for exactly this and, applied by trained examiners to patients with a continuous acute vestibular syndrome, outperforms early diffusion-weighted MRI [20,41]. In the original study a 'dangerous' HINTS pattern was 100% sensitive and 96% specific for central pathology, while early DWI was falsely negative in 12% of strokes [20]. The counter-intuitive core is that a normal, non-corrective

head-impulse test in a patient with spontaneous nystagmus points toward the brainstem or cerebellum, not the labyrinth [19,20].

HINTS is a rule for the right patient, not a universal screen. It applies only to the continuous acute vestibular syndrome with nystagmus, not to brief positional or episodic vertigo, and it demands competence in head-impulse interpretation [41]. Adding hearing ('HINTS plus') captures the AICA-territory infarcts that produce acute audiovestibular loss; in the small-stroke study HINTS plus was 100% sensitive against 47% for early MRI, with 53% of initial scans falsely negative and non-lacunar mechanisms — including vertebral occlusion and dissection — in 47% [21]. The practical message is that an early negative MRI never excludes posterior-circulation stroke in the first 24 to 48 hours [21,24].

HINTS sits within a wider diagnostic frame. The timing-and-triggers approach (TiTrATE) first sorts the dizzy patient by the temporal pattern — a continuous acute vestibular syndrome, recurrent spontaneous episodes, or brief triggered spells — because each category carries a different differential and a different role for the bedside examination [45]. Dissection can present in two of these categories: as a continuous acute vestibular syndrome from established infarction, where HINTS is the decisive tool, and as recurrent spontaneous episodes of vertebrobasilar transient ischaemia that precede the completed stroke, where the history and the dissection red flags carry the weight because the examination between episodes may be normal [45,25]. Recognising that a painful, stuttering, episodic vertigo in a young patient may be vertebrobasilar transient ischaemia rather than vestibular migraine is one of the highest-yield judgements in the clinic [7,45].

HINTS interprets the vestibular syndrome; the dissection-specific red flags interpret the patient around it. Severe or unusual neck or occipital pain, a partial Horner sign, recent neck trauma or manipulation, age under 50, a connective-tissue background, and any focal neurological sign each raise the dissection probability independently and should lower the threshold to image [1,2,16]. Inability to sit or stand unaided, truncal ataxia disproportionate to the vertigo, and a direction-changing gaze-evoked nystagmus are the cerebellar signs most easily missed when attention is fixed on the eyes [19,24].

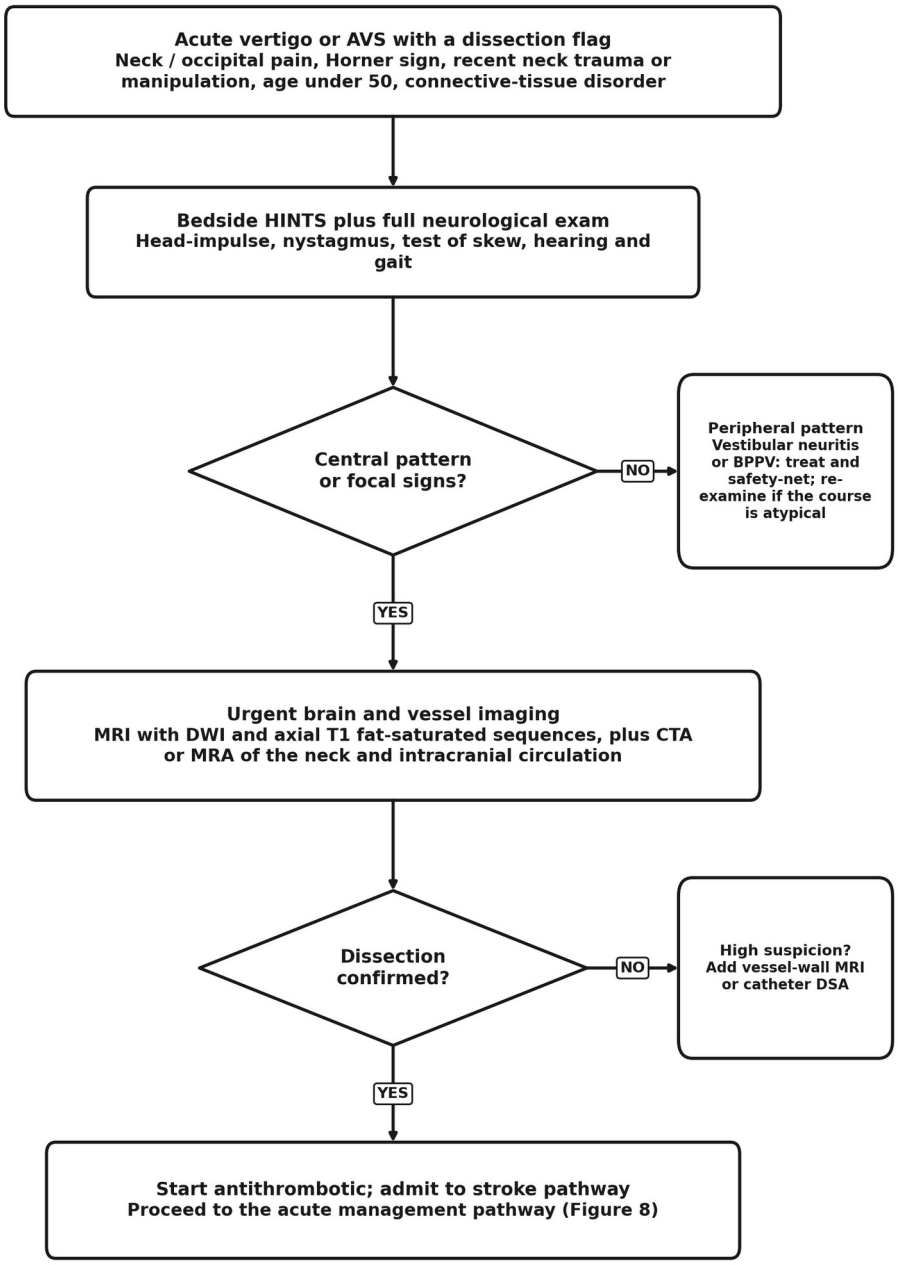

Figure 5. A bedside-to-imaging pathway for the dizzy patient with a possible dissection. HINTS and the dissection red flags drive the decision to image; an early negative MRI does not exclude stroke.

Source: Australian Dizziness Clinics. Adapted from Kattah et al. [20], Tarnutzer et al. [41] and the ESO guideline [15].

Table 3. Peripheral versus central HINTS pattern in the acute vestibular syndrome [19,20,21].

Component	Peripheral (e.g. neuritis)	Central (stroke / dissection)
Head-impulse test	Abnormal — corrective saccade	Normal — no corrective saccade
Nystagmus	Unidirectional, horizontal, fixation-suppressed	Direction-changing or vertical / torsional
Test of skew	Absent	Present (vertical ocular misalignment)
Hearing (HINTS plus)	Usually intact	New unilateral loss suggests AICA
Gait	Unsteady but able to walk	Often unable to stand unaided

□ **Clinical Pearl:** In a continuous acute vestibular syndrome, a NORMAL head-impulse test is the single most useful danger sign. Combined with new hearing loss, direction-changing nystagmus, or an inability to stand, it should trigger vessel imaging regardless of an early negative MRI.

V. Investigations and the Role of Imaging

No single modality is the universal gold standard for dissection; carotid and vertebral imaging is a matter of choosing the test that best demonstrates the lesion in a given segment [16,22]. The direct sign is the intramural haematoma, and the sequence that shows it best is axial T1-weighted fat-saturated MRI, on which the methaemoglobin of a subacute mural haematoma appears as a bright crescent surrounding the narrowed flow void — the crescent sign [1,22].

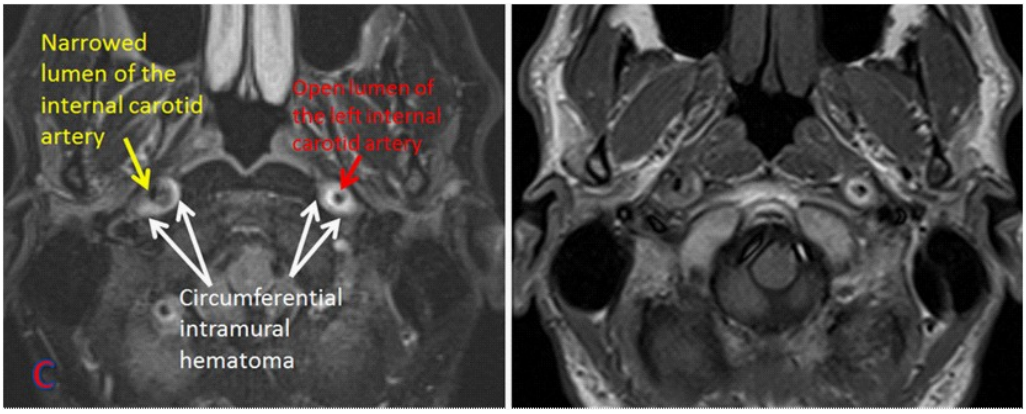

Figure 6. Bilateral internal carotid dissection. Axial fat-saturated imaging shows the eccentric intramural haematoma (crescent) narrowing the true lumen; MR angiography shows the corresponding tapered luminal narrowing.

Source: Mirza S and Gokhale S, via Wikimedia Commons (CC BY 4.0).

CT angiography and MR angiography have broadly comparable test characteristics, and in practice the choice is driven by availability, the need to image the lumen versus the wall, and contraindications [22]. CTA resolves the luminal signs superbly and is fast and widely available in the acute setting; MRI with fat-saturated sequences uniquely visualises the mural haematoma and simultaneously stages the brain parenchyma with DWI [16,22]. Catheter digital subtraction angiography remains the historical reference standard and best demonstrates the luminal signatures — the tapered string sign, the flame-shaped occlusion, the double lumen, the intimal flap and the dissecting pseudoaneurysm — but it is invasive and, crucially, can miss the mural haematoma that fat-saturated MRI shows directly [1,16,22].

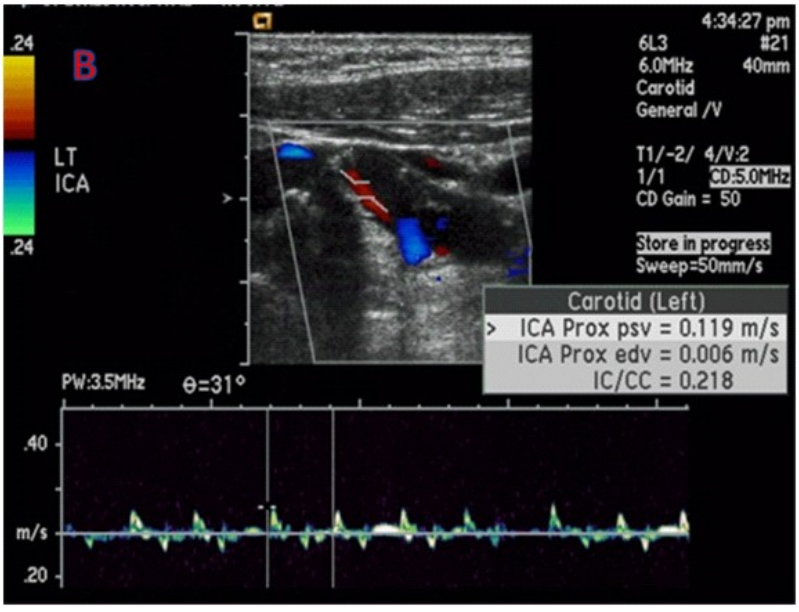

Figure 7. The string sign of the internal carotid artery on duplex ultrasonography — a long, tapered, markedly narrowed residual lumen. Ultrasound is operator-dependent and insensitive to high cervical and non-occlusive dissection, so a normal scan cannot exclude the diagnosis.

Source: Mirza S and Gokhale S, via Wikimedia Commons (CC BY 4.0).

Duplex ultrasonography is useful for following flow and may show indirect signs, but its sensitivity is limited — around 70% for non-ischaemic carotid dissection and somewhat higher for vertebral disease — and it is blind to the high distal carotid near the skull base and the V3 loop, exactly the segments dissection favours [40]. A negative ultrasound therefore never excludes dissection and must be confirmed by cross-sectional angiography or MRI [40]. High-resolution vessel-wall MRI is the most promising recent advance, directly characterising the mural haematoma and intimal flap and helping to separate dissection from vasculitis and atherosclerosis when conventional angiography is ambiguous [39].

Table 4. Imaging modalities and the radiological signs of dissection [1,16,22,39,40].

Modality	Strengths	Limitations / signs
T1 fat-saturated MRI	Direct mural haematoma — crescent sign; stages brain with DWI	Subacute window; access; motion sensitive
CT angiography	Fast, available; excellent luminal detail	Contrast and radiation; wall less well seen
MR angiography	Lumen plus wall; no radiation	May overestimate stenosis; slower
Catheter DSA	Reference for luminal signs: string, flame, double lumen	Invasive; may miss mural haematoma
Duplex ultrasound	Bedside, follow-up of flow; string-flow sign	~70% sensitive; blind to high ICA and V3 [40]
Vessel-wall MRI	Direct wall characterisation; mimics	Limited availability; expertise [39]

□ **Clinical Insight:** Image the wall, not just the lumen. Axial T1 fat-saturated MRI shows the intramural haematoma directly and is the most specific single test; pair it with DWI so the brain and the vessel are staged in one study.

VI. Differential Diagnosis

The differential of dissection-related dizziness is the differential of the acute and episodic vestibular syndromes, with the constant question of which presentations carry a vascular tail. Vestibular neuritis is the principal mimic of vertebral dissection with PICA-territory infarction; the discriminators are the HINTS pattern, new hearing loss, and the inability to stand, not the severity of the vertigo [19,20]. Reassuringly, population data show that isolated dizziness, vertigo or imbalance in the emergency department carries only a 0.7% rate of stroke or transient ischaemic attack overall — but the subgroup with imbalance and with vascular risk factors concentrates the risk, which is precisely where dissection hides [25].

Vestibular migraine is the commonest cause of recurrent spontaneous vertigo and is diagnosed on the Barany Society and International Headache Society criteria — recurrent vestibular symptoms lasting five minutes to 72 hours, a history of migraine, a temporal association with migrainous features, and exclusion of other causes [23]. It overlaps with dissection in two ways: migraine is itself a risk factor for dissection and shares the PHACTR1 susceptibility locus, and a first, abrupt, painful episode in a migraineur should not be assumed benign [23,33]. Benign paroxysmal positional vertigo is distinguished by its brief, positionally triggered, fatigable nystagmus and is not a diagnostic trap for dissection except when a clinician stops at it without examining the rest of the patient.

Among the dangerous differentials, posterior-circulation atherosclerotic stroke occupies the same territories as vertebral dissection and is distinguished chiefly by age and vascular risk profile; large-artery atherosclerosis caused half of the lateral medullary infarcts in Kim's series against 15% from dissection [8,24]. Subarachnoid haemorrhage must be considered with thunderclap headache, particularly where an intracranial vertebral (V4) dissection has ruptured [5]. Reversible cerebral vasoconstriction syndrome enters the differential of recurrent thunderclap headache with segmental arterial narrowing that resolves within three months, often post-partum or drug-related, and can be mistaken for — and rarely coexists with — dissection [26].

Table 5. Differential diagnosis of the acutely or recurrently dizzy patient with possible dissection [8,23,24,25,26].

Diagnosis	Discriminating features	Vascular risk

Vestibular neuritis	Peripheral HINTS; intact hearing; can stand	Low; mimic of medial-PICA infarct
Cerebellar infarct (PICA)	Central HINTS; truncal ataxia; cannot stand	High — dissection / atheroma
AICA infarct	Acute combined audiovestibular loss	High — vertebrobasilar / dissection
Vestibular migraine	Recurrent 5 min–72 h; migraine history	Shared PHACTR1; migraine a risk factor
Atherosclerotic PCA stroke	Older, vascular risk factors	High — large-artery disease
RCVS / SAH	Thunderclap headache; segmental narrowing	Consider intracranial V4 dissection

□ **Important:** The safest discriminator is not the diagnosis you reach but the examination you complete. Never close on a peripheral label without a head-impulse test, a hearing check, and an attempt to stand the patient up.

VII. Acute and Secondary-Prevention Management

Management has two goals: prevent the early ischaemic stroke that the dissection threatens, and treat any stroke that has already occurred. The cornerstone of secondary prevention is antithrombotic therapy, and the long-standing debate over antiplatelet versus anticoagulation has now been settled, in practical terms, by two randomised trials [27,29,30].

The Cervical Artery Dissection in Stroke Study (CADISS) randomised 250 patients with symptomatic carotid or vertebral dissection to antiplatelet or anticoagulant therapy for three months. Ipsilateral stroke or death occurred in just 2% of the antiplatelet group and 1% of the anticoagulant group — no significant difference — and the overall recurrent-stroke rate of 2% was far lower than the figures from older observational series that had driven aggressive anticoagulation [27]. The one-year final results confirmed a recurrent-stroke rate of 2.4% with no difference between strategies and no difference in recanalisation; notably, central review failed to confirm dissection in 52 of the randomised patients, underscoring how often the diagnosis is over-called [28].

TREAT-CAD then tested whether aspirin is non-inferior to a vitamin-K antagonist over 90 days using a stringent composite of clinical and MRI endpoints in 194 patients. Non-inferiority of aspirin was not shown: the primary endpoint occurred in 23% on aspirin versus 15% on anticoagulation, and seven ischaemic strokes occurred in the aspirin arm against none with anticoagulation [29]. The individual patient-data meta-analysis of both trials (444 patients) reconciles the two results: no significant difference in the composite early endpoint, but anticoagulation associated with fewer strokes (0.5% vs 4.0%) at the cost of more bleeding [30]. The defensible reading is that either agent is acceptable, absolute event rates are low, and the choice should be individualised — favouring anticoagulation where the ischaemic burden is high and the bleeding risk low, and antiplatelet therapy where the reverse holds [15,30,32].

Table 6. The randomised antithrombotic evidence in cervical arterial dissection [27,28,29,30].

Trial	Design	Key result
CADISS (2015)	250 pts; antiplatelet vs anticoagulant, 3 months	Stroke/death 2% vs 1%, no difference; overall recurrence 2%
CADISS final (2019)	1-year follow-up	Recurrent stroke 2.4%; no difference; 52 had no confirmed dissection
TREAT-CAD (2021)	194 pts; aspirin vs VKA, 90 days	Aspirin non-inferiority NOT shown; 7 strokes on aspirin vs 0
IPD meta-analysis (2024)	444 pts pooled	Fewer strokes with anticoagulation (0.5% vs 4.0%), more bleeding

Antithrombotic therapy is typically continued for three to six months and then reviewed against follow-up vessel imaging, stepping down to an antiplatelet agent or stopping once the artery has healed; the European Stroke Organisation guideline endorses either antithrombotic strategy as a strong

recommendation on the basis of these trials [15,32]. Supportive measures — avoidance of further neck strain and manipulation, sensible blood-pressure control, and a graded return to activity — accompany pharmacological treatment [1,16].

□ **Key Point:** Antiplatelet and anticoagulant therapy give broadly equivalent, low recurrent-stroke rates in cervical dissection (CADISS, TREAT-CAD). Either is acceptable for three to six months; individualise by ischaemic burden and bleeding risk, then re-image before stepping down.

VIII. Reperfusion, Endovascular Therapy and Refractory Disease

When a patient arrives within the reperfusion window with a disabling stroke, dissection should not deny them acute treatment. Intravenous thrombolysis in dissection-related stroke carries no excess of intracranial haemorrhage compared with stroke of other causes: in Engelter's comparison of 55 thrombolysed dissection patients with over a thousand non-dissection controls, symptomatic intracranial haemorrhage rates were equivalent, and dissection should therefore not be treated as a contraindication to alteplase when standard criteria are met [13]. The European Stroke Organisation guideline accordingly recommends intravenous thrombolysis within the licensed window and mechanical thrombectomy for anterior-circulation large-vessel occlusion in dissection [15].

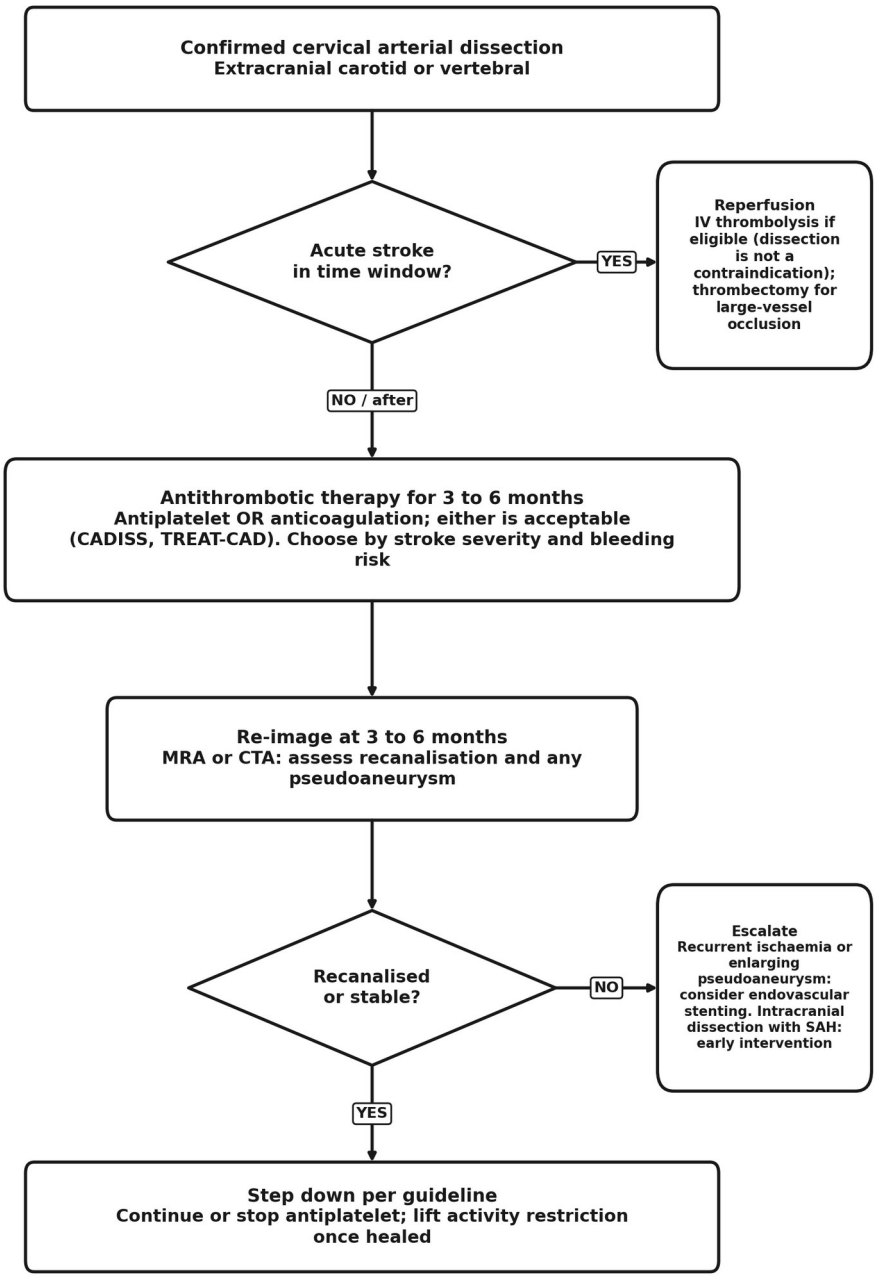

Figure 8. An acute and secondary-prevention pathway for confirmed cervical arterial dissection, from reperfusion eligibility through antithrombotic choice, re-imaging, and escalation.

Source: Australian Dizziness Clinics. Adapted from the ESO guideline [15], CADISS [27] and TREAT-CAD [29].

Endovascular stenting has a narrow, well-defined role. It is reserved for recurrent ischaemia despite optimal medical therapy, an enlarging or symptomatic pseudoaneurysm, or haemodynamic compromise from a critical stenosis; the ESO guideline found insufficient evidence to recommend routine endovascular or surgical treatment of residual stenosis or stable dissecting aneurysms [15]. The exception that demands early intervention is intracranial dissection presenting with subarachnoid haemorrhage, where the rebleeding risk justifies early endovascular or surgical securing of the lesion [15]. For the great majority of extracranial dissections, however, the artery heals on medical therapy alone and the interventional questions never arise [15,31].

□ **Clinical Pearl:** Dissection is not a contraindication to thrombolysis. A disabling anterior-circulation stroke from carotid dissection within the window should be treated like any other — withholding alteplase causes more harm than the theoretical bleeding risk.

IX. Prognosis, Recurrence and Special Populations

The natural history of treated cervical dissection is reassuring. Stenotic lesions resolve, usually without angiographic sequel, in around 70% of patients within a few months, and recanalisation approaches 90% where serial MRI is used to track the mural haematoma; the bulk of healing occurs in the first three to six months, which is why follow-up imaging is timed to that window [13,31]. Functional outcomes are good — a modified Rankin score of 0 to 1 in roughly 80% of vertebral dissection strokes — and do not differ from young stroke of other aetiologies [11,12].

Recurrence is low. After the first month the annual risk of a further dissection is approximately 1%, and the annual recurrent-stroke risk lies between about 0.3% and 3.4%, concentrated in the earliest period [6,31]. The recurrence hazard is meaningfully higher in two groups: those with a family history of dissection and those with demonstrable connective-tissue abnormality, in whom younger age at first event also predicts recurrence [6,35,37]. Dissecting pseudoaneurysms, a common source of anxiety on follow-up imaging, have a benign natural history — carotid aneurysms commonly persist while vertebral aneurysms often resolve, and delayed rupture or embolism from a stable pseudoaneurysm is exceptional [1,31].

Special populations refine the picture. Paediatric dissection shows a marked male predominance not explained by trauma, a higher proportion of intracranial disease in anterior-circulation cases, and the vertebral artery at C1–C2 as the commonest posterior site (53%); recurrent ischaemia is higher in posterior than anterior childhood dissection [14]. The post-partum period carries an elevated risk of both dissection and the overlapping reversible cerebral vasoconstriction syndrome, a consideration when counselling women on activity and future pregnancy [26]. Across all groups, return to contact sport and heavy neck loading is generally deferred until vessel healing is confirmed on follow-up imaging [1,16].

□ **Clinical Insight:** Reserve heightened surveillance for the patients who actually recur — those with a family history of dissection or a connective-tissue phenotype, and the young. For everyone else, a single dissection that heals carries a low long-term recurrence risk.

X. Controversies, Guidelines and Future Directions

The most contested question in cervical dissection is its relationship to cervical manipulative therapy. The American Heart Association and American Stroke Association scientific statement is the authoritative synthesis: current biomechanical evidence is insufficient to establish that manipulation causes dissection, yet most controlled studies find an association between manipulation and vertebral dissection stroke in young patients [16]. The unresolved issue is reverse causation — whether patients with an already-symptomatic dissection seek manipulation for the resulting neck pain and headache, and then stroke regardless. The case-control and case-crossover data of Cassidy and colleagues support this interpretation: vertebrobasilar stroke was equally associated with prior visits to a chiropractor and to a primary-care physician, implying that the consultation marks the prodrome rather than the cause [38]. The statement's practical recommendation stands: consider dissection as a presenting symptom and inform patients of the statistical association before cervical manipulation [16].

Two further questions remain open. The first is whom to screen for an underlying arteriopathy: only a minority of patients have a clinically apparent connective-tissue disease, yet fibromuscular dysplasia is found in around a fifth when actively sought, and the yield and cost-effectiveness of systematic screening and of imaging other vascular beds are not settled [9,10,11]. The second is genetic: an underlying, partly heritable arteriopathy is firmly established in principle through the PHACTR1 association and the connective-tissue ultrastructural studies, but translating this into individual risk prediction is not yet possible [33,34,37].

The clearest near-term advance is imaging. High-resolution vessel-wall MRI directly visualises the mural haematoma and intimal flap, distinguishes dissection from vasculitis and atherosclerosis, and allows serial monitoring of healing in a way luminal angiography cannot; as availability and expertise spread it is likely to become the reference test for the ambiguous case and the standard tool for follow-up [39]. For the vestibular physician, the enduring message is older and simpler: the diagnosis is made not by the

scanner but by the clinician who keeps dissection in mind for the young, painful, or atypically dizzy patient, examines them completely, and images the wall when the story does not fit a benign label [1,2,20].

□ **Key Point:** The dissection–manipulation debate turns on reverse causation: neck pain and headache are the prodrome of dissection, so a manipulation that precedes a stroke may be a marker of the lesion, not its cause. Counsel patients of the association and treat new neck pain in the young as a possible dissection.

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