Vestibular Emergencies:

What Not to Miss in the Emergency Department

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How to Use This Review

This review focuses on life-threatening conditions that present with acute dizziness or vertigo. Many of these conditions mimic benign peripheral vestibular disorders on initial presentation but carry high morbidity and mortality if not recognised. Recognition of red flags and timely imaging are essential to prevent catastrophic outcomes.

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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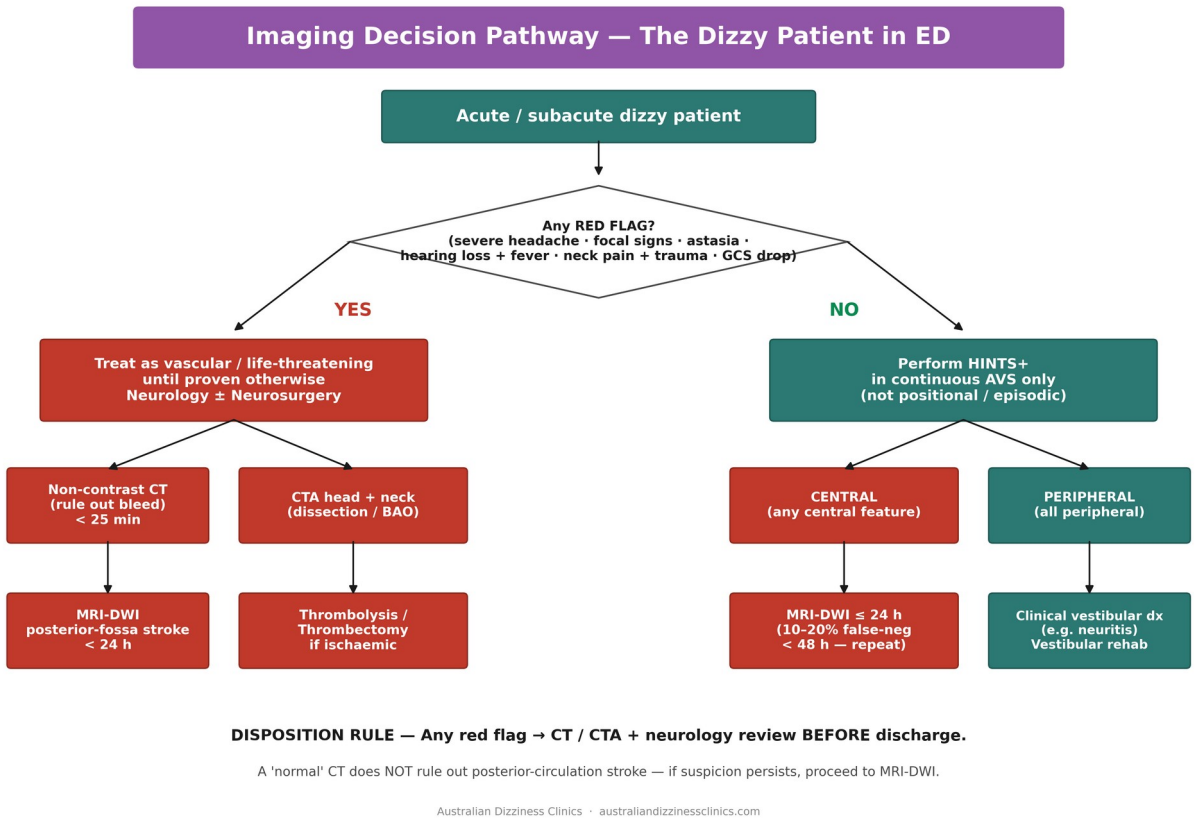

Figure 7. ED red-flag → imaging decision pathway for the dizzy patient. Any single red flag (severe headache, focal signs, astasia, neck pain + trauma, hearing loss + fever) mandates CT ± CTA + neurology review before discharge; in their absence, HINTS+ is performed only in continuous AVS.
Source: Australian Dizziness Clinics — original figure (2026).

Vestibular emergencies are life- or limb-threatening causes of dizziness that masquerade as benign peripheral vestibular disease [1,2]. They are uncommon in absolute terms but disproportionately represented in missed or delayed ED diagnoses because the dominant symptom — vertigo — is identical to that of vestibular neuritis and BPPV [2,3,11].

This review covers the most important ED vestibular emergencies: cerebellar haemorrhage, malignant cerebellar infarct, vertebral artery dissection, basilar artery occlusion, Wernicke's encephalopathy, carbon monoxide poisoning, bacterial labyrinthitis and meningitis, and temporal bone fracture [3,4,5]. Each is grouped by pathophysiology and each has a time-critical intervention that changes outcome [4,18,19] [16].

The common thread is that each of these emergencies can present with pure vertigo, nausea and gait unsteadiness before focal signs declare themselves [2,3,11]. Safe ED practice therefore requires a structured approach to every AVS presentation: vascular risk assessment, HINTS+, gait testing, targeted history for toxic and metabolic precipitants, and low threshold for neuroimaging [2,8,12].

Missed vestibular emergencies are a leading cause of medicolegal claims in emergency medicine [11,20]. Recognising the few patterns in this review — and escalating early when one fits — is arguably the highest-yield diagnostic discipline in ED vestibular assessment [11,20].

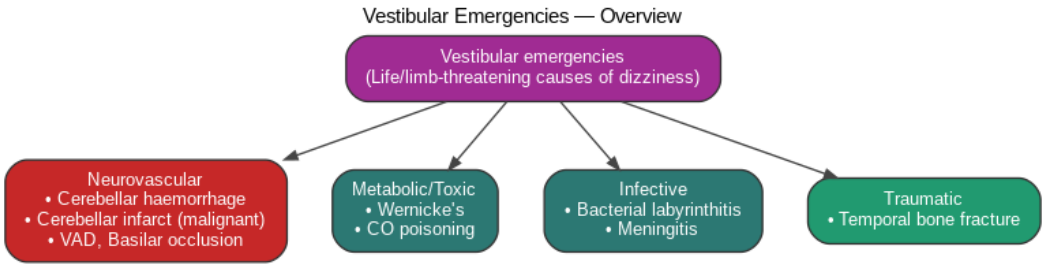

Figure 1. Vestibular emergencies — overview of neurovascular, metabolic, infective and traumatic causes.

Although individually uncommon, vestibular emergencies as a group account for a significant proportion of preventable death and disability in dizzy-patient cohorts [2,4]. The unifying clinical principle is that none of these conditions can be reliably excluded by bedside examination alone — appropriate imaging and admission are essential whenever the clinical pattern raises suspicion [3,4,11].

Key Point: *Not all acute vertigo is BPPV. Red flags include focal neurological signs, severe headache, acute hearing loss, rapid worsening, and systemic illness.*

II. Cerebellar Haemorrhage

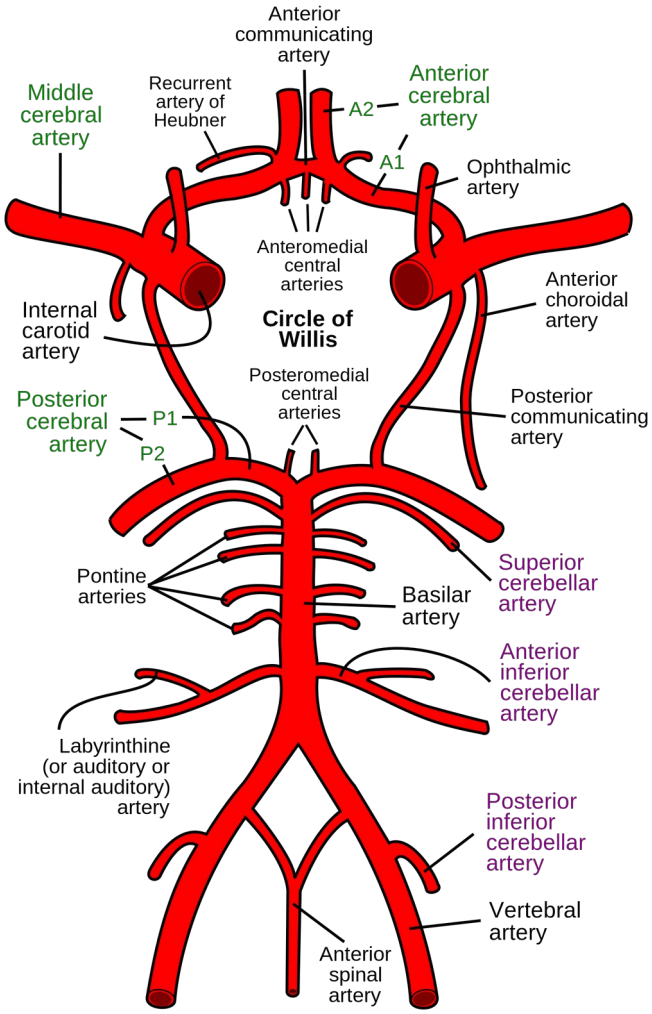

Figure 8. Posterior circulation anatomy showing the vertebrobasilar system and its cerebellar branches (PICA, AICA, SCA). Occlusion or dissection at any level of this system produces the stroke syndromes covered throughout this review.
Source: Wikimedia Commons — File:Circle of Willis en.svg (Rhcastilhos) — Public domain.

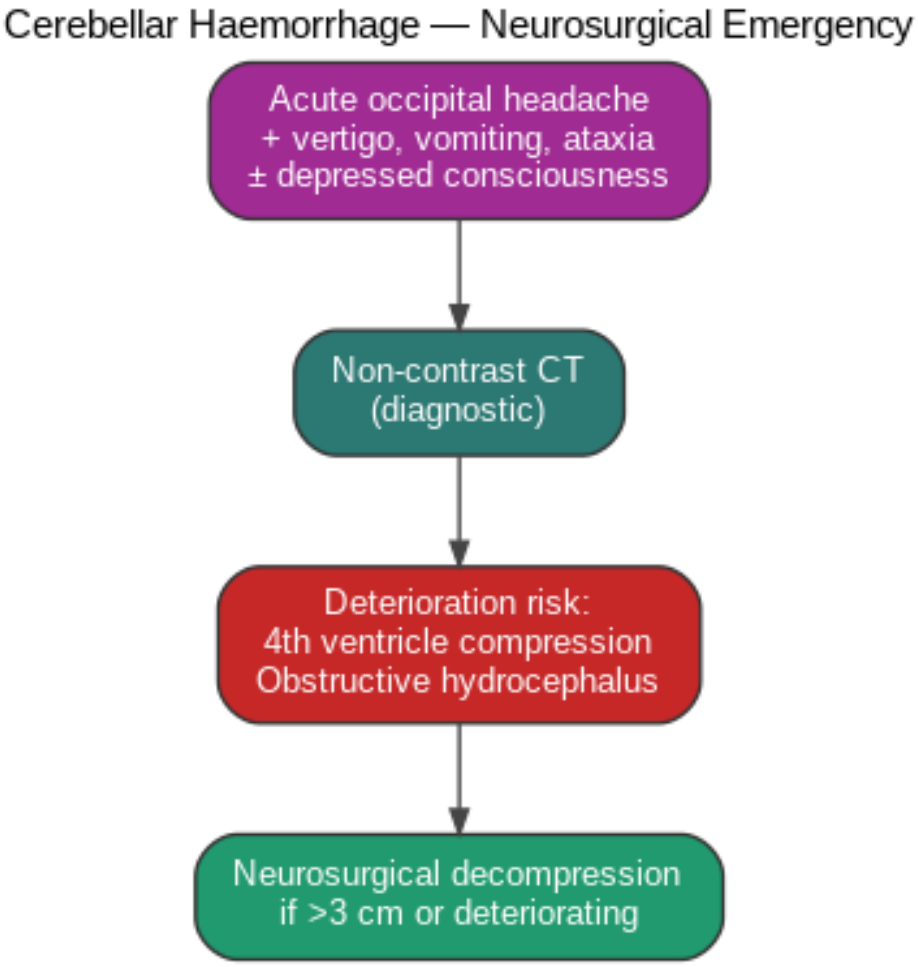

Figure 2. Cerebellar haemorrhage — neurosurgical emergency.

Presentation and Pathophysiology

Cerebellar haemorrhage is a neurosurgical emergency; mortality rises steeply once brainstem compression or hydrocephalus develops [3,4]. It typically occurs in patients with hypertension, anticoagulation or vascular malformations and presents with acute-onset severe occipital headache, vertigo, vomiting and truncal ataxia [3,4] [6].

The classic triad is acute occipital headache, vertigo with vomiting, and inability to stand unaided [3,4]. Depressed consciousness, cranial nerve palsies or new focal deficits signal brainstem compression and mandate immediate escalation [3,4].

Clinical Features and Red Flags

Examination findings include severe truncal ataxia, limb dysmetria ipsilateral to the haematoma, gaze-evoked nystagmus, and — in larger bleeds — cranial nerve deficits and reduced level of consciousness [3,4]. Astasia (inability to stand unaided) is a particularly strong red flag for cerebellar pathology [8,9].

Neuroimaging and Management

Non-contrast CT is the immediate investigation and is highly sensitive for acute haemorrhage [4]. Haematoma volume >3 cm in maximal diameter, compression of the fourth ventricle, or clinical deterioration are indications for emergency neurosurgical decompression — suboccipital craniectomy is potentially life-saving when performed early [3,4] [9].

In-hospital management requires ICU-level monitoring, reversal of anticoagulation, blood pressure control (systolic target typically 140–160 mmHg), and close neurological observation for deterioration [4]. Osmotic therapy, external ventricular drainage and definitive decompression are coordinated with neurosurgery [3,4].

⚠ **Important:** Any patient with acute vertigo + severe headache + inability to walk should undergo emergency CT and neurosurgery consultation.

III. Cerebellar Infarction with Malignant Oedema

Pathophysiology and Presentation

Large cerebellar infarcts — typically in PICA or SCA territory — can develop malignant vasogenic oedema over 24–72 hours, producing delayed deterioration that catches clinicians off guard [3,4]. Patients may appear well on admission and then decompensate rapidly as oedema peaks [3,4].

Recognition and Intervention

ED recognition depends on suspecting cerebellar infarct in any AVS patient with vascular risk, severe truncal ataxia, or central HINTS findings [2,8,11]. MRI-DWI is the imaging gold standard, recognising that small infarcts may be missed on early scans [19,20]. Any patient with a large cerebellar infarct requires ICU admission with hourly neuro observations for the first 72 hours [3,4].

Management includes ICU observation, osmotic therapy and, when oedema becomes life-threatening, posterior fossa decompression [3,4]. Outcomes with appropriate surgical intervention can be excellent — decompensation is preventable if recognised early [3,4].

 Clinical Pearl: Malignant cerebellar oedema can develop over 48–72 hours. Patients with large cerebellar infarction must be admitted to ICU, not discharged.

IV. Vertebral Artery Dissection

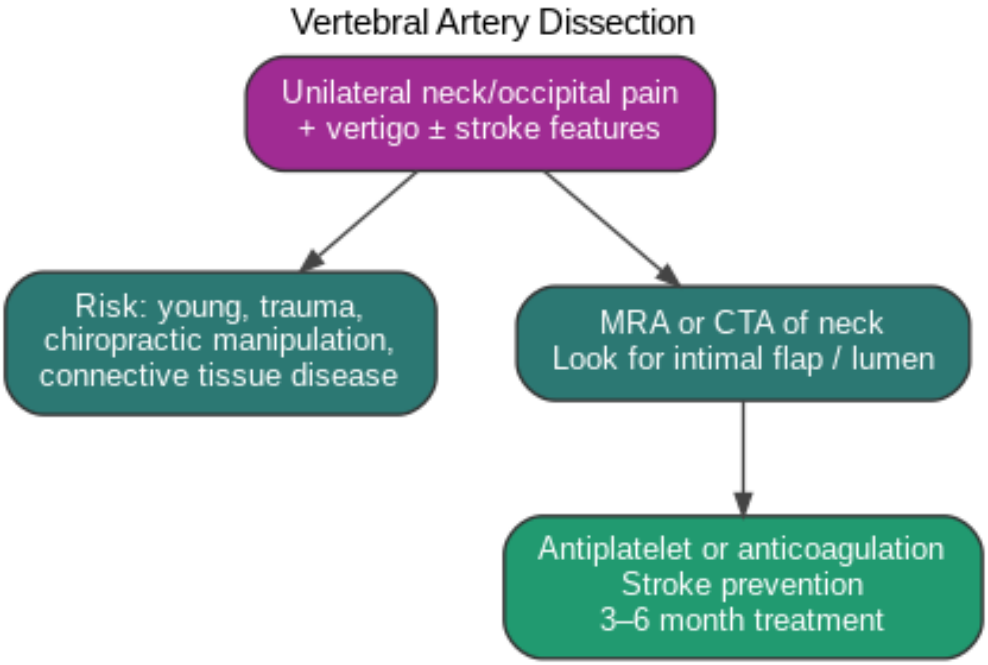

Figure 4. Vertebral artery dissection — diagnosis and management.

Presentation and Diagnosis

Vertebral artery dissection (VAD) typically presents with ipsilateral neck or occipital pain preceding vertigo and stroke features by hours to days [13]. Young patients, those with recent trauma or neck manipulation, and patients with connective tissue disorders are at increased risk [13].

Imaging and Risk Stratification

MRA of the neck is the investigation of choice; CTA is an acceptable alternative [13,19]. Findings include an intimal flap, mural haematoma, tapered or string-sign lumen, and associated posterior-circulation infarct [13]. Imaging should be obtained urgently whenever VAD is suspected [13].

Management

Acute management is antiplatelet therapy (aspirin) or anticoagulation for 3–6 months, guided by local stroke-service protocols; both strategies have comparable outcomes in contemporary trials [13,18]. Patients with associated ischaemic stroke are assessed for thrombolysis or thrombectomy on standard criteria [18,19] [13].

 Clinical Insight: Young patient with neck pain + acute vertigo = suspect VAD. The absence of Horner's does not exclude VAD. CTA/MRA should be obtained promptly.

V. Basilar Artery Occlusion

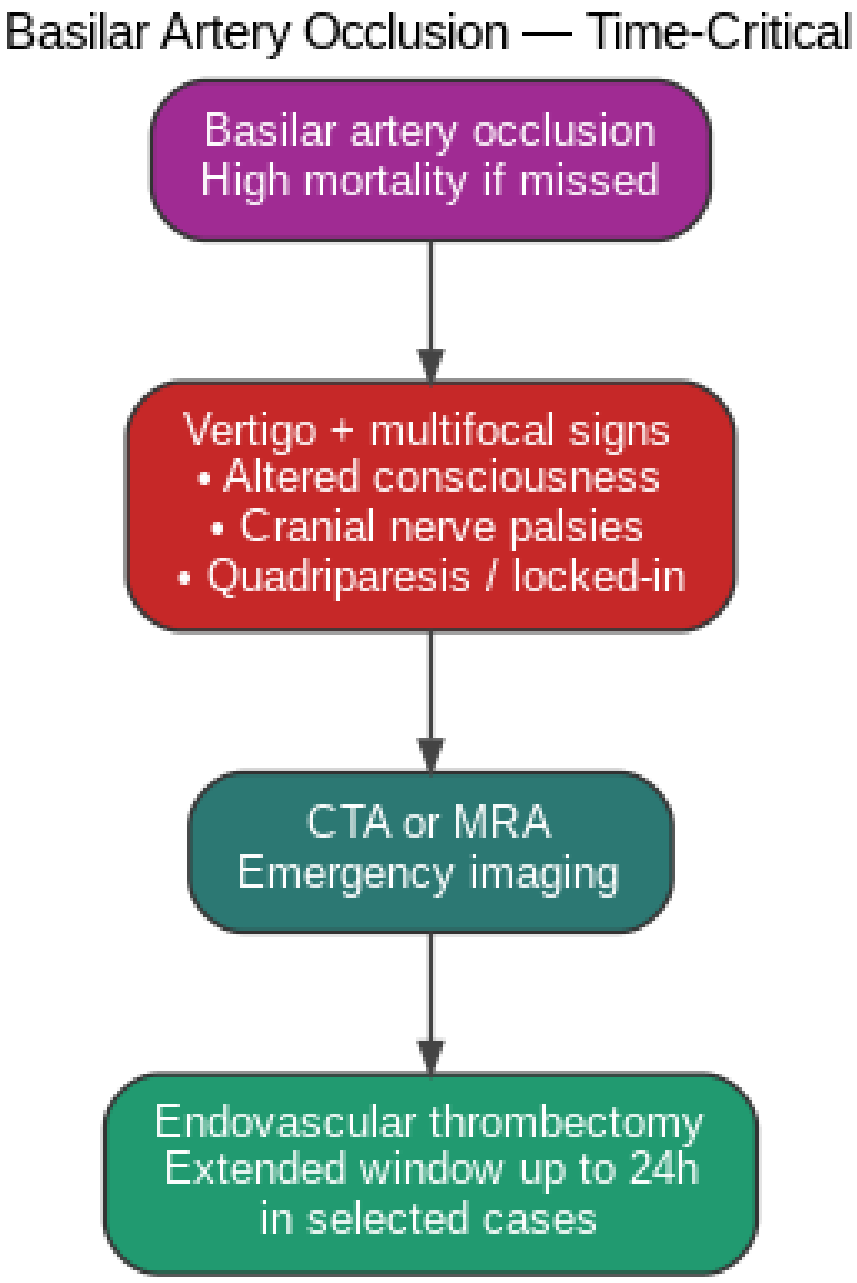

Figure 3. Basilar artery occlusion — time-critical thrombectomy pathway [15].

Presentation and Severity

Basilar artery occlusion (BAO) is among the most lethal stroke syndromes, with untreated mortality approaching 80% [5,19]. Presentation ranges from vertigo with multifocal neurological features to catastrophic locked-in syndrome [5,6,19].

Recognition and Urgent Imaging

Key red flags are vertigo with multifocal brainstem signs: altered consciousness, cranial nerve palsies, bilateral motor or sensory deficits, dysarthria, dysphagia, or vertical gaze palsy [5,6,19]. Rostral BAO produces top-of-basilar syndrome with midbrain, thalamic and occipital involvement [6,17].

Endovascular Thrombectomy

BAO is a strong indication for emergency endovascular thrombectomy, with extended windows up to 24 hours in selected cases based on imaging mismatch or clinical profile [19,20]. CTA or MRA is the investigation of choice; early activation of the stroke pathway and interventional neuroradiology is critical [19,20] [15].

⚠ **Important:** Basilar artery occlusion presents with vertigo + multifocal neurological signs. Any patient with acute vertigo + altered mental status warrants urgent CTA and stroke team activation.

VI. Wernicke's Encephalopathy

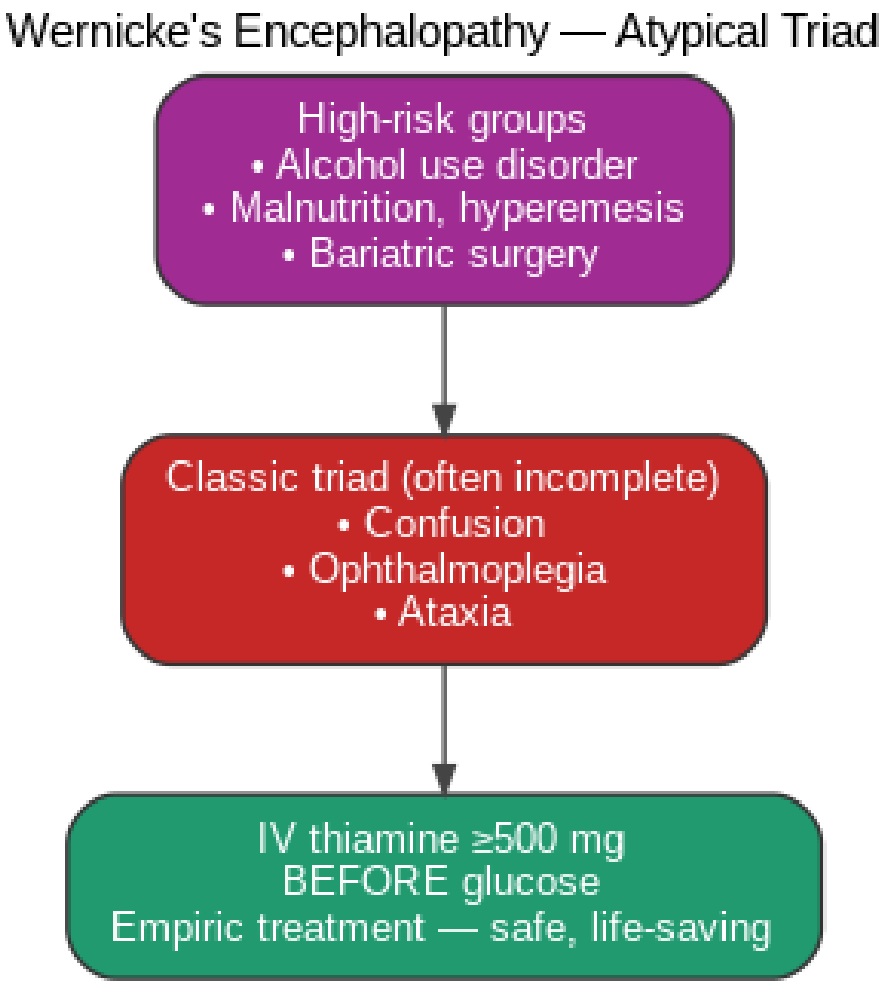

Figure 5. Wernicke's encephalopathy — empirical thiamine saves lives [16].

Pathophysiology and Risk Groups

Wernicke's encephalopathy is acute thiamine (vitamin B1) deficiency affecting the medial thalamus, mammillary bodies and periaqueductal grey, producing vertigo, ataxia and oculomotor abnormalities [10,14]. It is under-recognised in the ED and routinely misdiagnosed as intoxication or peripheral vestibular disease [10,14] [16].

High-risk groups include chronic alcohol use disorder, malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, eating disorders and post-bariatric surgery [10,14]. Any ED patient from these groups presenting with new ataxia, confusion or oculomotor dysfunction should be considered for empirical thiamine [10,14] [17].

Clinical Triad and Atypical Presentations

The classic triad of confusion, ophthalmoplegia and ataxia is present in fewer than a third of cases [10,14]. Isolated vertigo, unsteadiness or nystagmus can be the sole feature [10,14]. Empirical IV thiamine is safe and potentially life-saving — give it before glucose in suspected cases to avoid precipitating Wernicke's in a thiamine-deplete patient [10,14] [16].

Diagnosis and Management

Diagnosis is clinical; MRI may show symmetrical T2 hyperintensity in the medial thalami and periaqueductal grey but is neither sensitive nor specific and must not delay treatment [10,14]. Treatment is IV thiamine 500 mg three times daily for 2–3 days, followed by oral replacement and correction of underlying nutritional deficits [10,14] [17].

 Clinical Pearl: Do not wait for imaging or lab testing. If clinical suspicion is high, give IV thiamine immediately. Delay can result in permanent cognitive impairment.

VII. Carbon Monoxide Poisoning

Pathophysiology and Presentation

Carbon monoxide (CO) binds haemoglobin with >200-fold the affinity of oxygen, producing tissue hypoxia that preferentially affects high-metabolic tissues including the central vestibular pathways [15]. Patients present with dizziness, headache, nausea and confusion — symptoms frequently attributed to vestibular disease before the environmental history is taken [15].

Diagnosis and Management

Diagnosis relies on carboxyhaemoglobin (COHb) level measured by co-oximetry on venous or arterial blood gas — standard pulse oximetry is falsely normal [15]. Management is immediate removal from the source and high-flow 100% oxygen; hyperbaric oxygen is considered for severe poisoning (neurological features, COHb >25%, pregnancy, loss of consciousness) [15] [19].

 Key Point: *Obtain COHb level in any patient with dizziness, headache, or altered mental status if environmental exposure is possible.*

VIII. Bacterial Labyrinthitis and Meningitis

Bacterial Labyrinthitis

Bacterial labyrinthitis is infection of the inner ear, most often secondary to otitis media or meningitis, presenting with acute vertigo, sensorineural hearing loss, ear pain, fever and systemic toxicity [16]. It is an otological emergency that can progress to meningitis and permanent deafness if untreated [16].

Management is immediate IV broad-spectrum antibiotics covering Streptococcus pneumoniae and Haemophilus influenzae (ceftriaxone 2 g IV 12-hourly is standard), plus urgent ENT review for possible mastoidectomy or labyrinthectomy if mastoiditis or intracranial complications are present [16].

Bacterial Meningitis with Vestibular Involvement

Bacterial meningitis can present with acute vertigo and nystagmus, particularly when the inflammatory process involves the cerebellopontine angle or causes labyrinthitis [16]. Any patient with vertigo accompanied by fever, headache, neck stiffness, photophobia or altered consciousness requires urgent lumbar puncture after neuroimaging and empirical antibiotics [16].

Do not delay antibiotics: give ceftriaxone 2 g IV 12-hourly plus vancomycin for penicillin resistance (and acyclovir if herpes encephalitis is a differential) as soon as meningitis is suspected — delays increase mortality [16]. Dexamethasone 10 mg IV given with or before the first dose of antibiotics reduces neurological sequelae in pneumococcal meningitis [16].

⚠ **Important:** Acute vertigo + fever + headache = bacterial meningitis until proven otherwise. Do not delay antibiotics.

IX. Temporal Bone Fracture

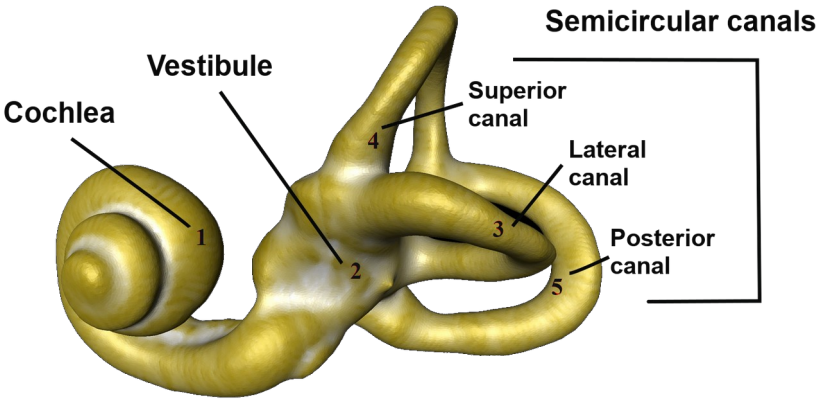

Figure 9. The bony labyrinth — cochlea, vestibule, and three semicircular canals. Transverse temporal bone fractures cross the otic capsule and typically cause profound SNHL with vertigo; longitudinal fractures spare the otic capsule but often produce conductive loss and facial palsy.
Source: Wikimedia Commons — File:Bony labyrinth labelled.png — CC BY-SA 4.0.

Presentation and Complications

Temporal bone fractures result from blunt head trauma and can cause acute vertigo through labyrinthine concussion, perilymph fistula, or direct eighth-nerve injury [17]. Facial nerve palsy, haemotympanum, CSF otorrhoea or Battle's sign should be sought in any trauma patient with new dizziness [17].

Imaging and Management

HRCT of the temporal bones is the gold-standard investigation: look for fracture lines crossing the otic capsule or involving the facial nerve canal, and for pneumolabyrinth [17]. Otic-capsule-violating fractures carry higher risk of sensorineural hearing loss and facial palsy [17].

Management is primarily conservative — bed rest, vestibular rehabilitation and observation — with ENT review for persistent CSF leak, facial palsy or progressive hearing loss [17]. Surgical exploration is reserved for complications such as perilymph fistula or decompression of an injured facial nerve [17].

Table 1 summarises the principal vestibular emergencies and their time-critical interventions; the operative message is that ED vigilance — not sophisticated tests — is the dominant determinant of outcome [1,2,11].

Condition	Key Features	Urgent Action
Cerebellar Haem.	Vertigo + severe headache + ataxia → deterioration	CT; neurosurgery; ICU
Cerebellar Infarc.	Cerebellar stroke + oedema risk 48–72 hrs	MRI DWI; ICU; neuro
VAD	Neck pain + vertigo + possible Horner's	CTA/MRA; anticoagulation
BAO	Vertigo + multifocal signs	CTA stat; stroke alert
Wernicke's	Confusion + ophthalmoplegia + ataxia	IV thiamine immediately
CO Poisoning	Dizziness + headache + confusion	COHb level; 100% O2
Bact. Lab.	Severe vertigo + fever + hearing loss	Blood cx; CT; antibiotics
Meningitis	Vertigo + fever + headache + stiff neck	Blood cx; LP; antibiotics
Temporal Bone	Post-trauma vertigo + hearing loss ± CSF	HRCT; ENT

X. Conclusions

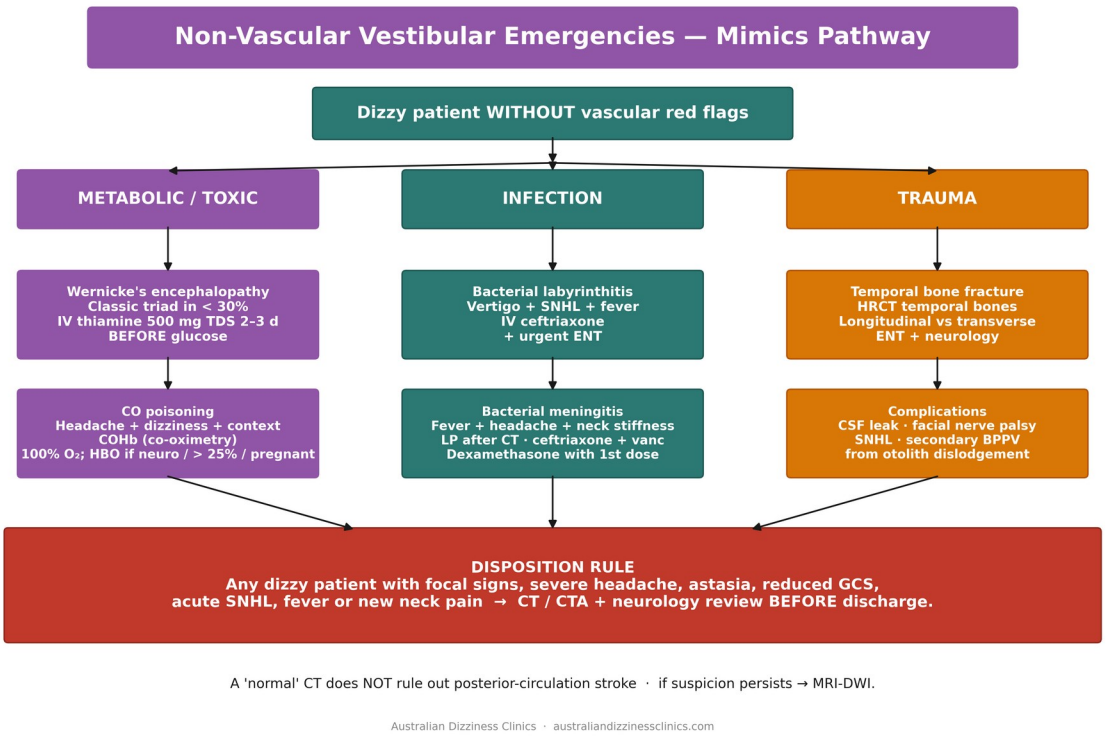

Figure 10. Non-vascular emergency pathway — metabolic (Wernicke's, CO), infective (bacterial labyrinthitis, meningitis) and traumatic (temporal bone fracture) causes are distinct but share a single disposition rule: any red flag triggers CT/CTA and neurology/neurosurgery review before discharge.
Source: Australian Dizziness Clinics — original figure (2026).

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This review covers the most important ED vestibular emergencies: cerebellar haemorrhage, malignant cerebellar infarct, vertebral artery dissection, basilar artery occlusion, Wernicke's encephalopathy, carbon monoxide poisoning, bacterial labyrinthitis and meningitis, and temporal bone fracture [3,4,5]. Each is grouped by pathophysiology and each has a time-critical intervention that changes outcome [4,18,19] [16].

A systematic approach to every AVS presentation — full neurological examination, HINTS+, gait testing, focused history for toxic, metabolic and traumatic precipitants, and early liaison with neurology, neurosurgery, interventional radiology and ICU — allows recognition and treatment of the small number of patients whose outcome depends on it [2,3,4,5,18,19]. Early empirical treatment for Wernicke's, meningitis or CO poisoning is often appropriate before definitive confirmation [10,14,15,16] [16].

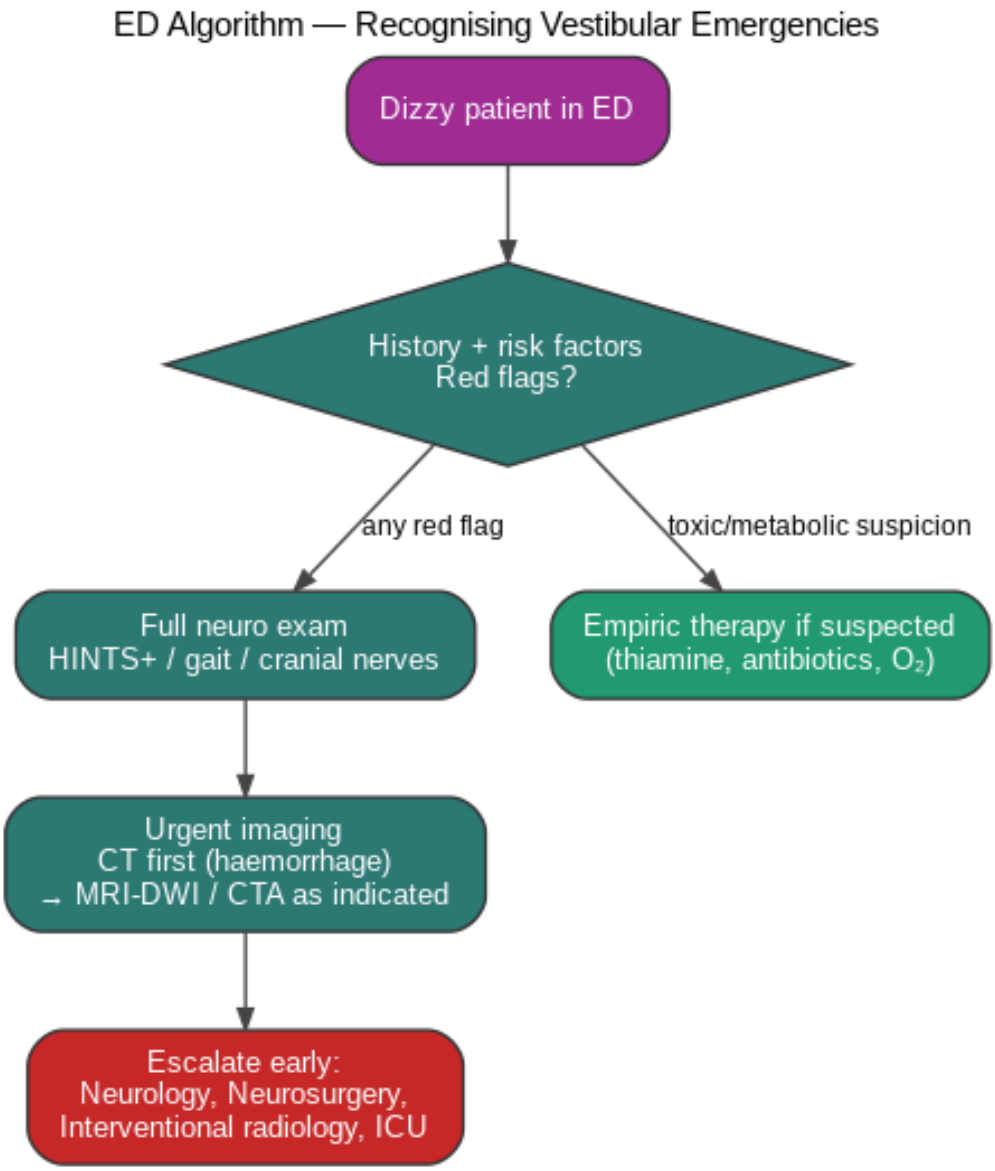

Figure 6. ED algorithm for recognising vestibular emergencies.

A practical operational rule is that any dizzy patient with depressed consciousness, focal neurological signs, severe headache, or inability to stand unsupported requires urgent neuroimaging and senior review before discharge can be considered [3,4,11].

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