Episodic Vestibular Syndrome:
ED Triage of Recurrent Vertigo

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How to Use This Review

Episodic vestibular syndrome (EVS) is characterized by recurrent episodes of vertigo separated by symptom-free intervals. This review examines vestibular migraine, Meniere's disease, vertebrobasilar TIA, cardiac causes, and other conditions requiring ED triage and risk stratification.

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.

Contents

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I. Introduction

Episodic vestibular syndrome (EVS) describes recurrent vertigo separated by symptom-free intervals, in contrast to the continuous symptoms of acute vestibular syndrome [7]. The differential is broad and spans benign vestibular disorders, dangerous cerebrovascular disease and cardiac causes: vestibular migraine (most common overall), Ménière's disease, vertebrobasilar TIA, cardiac arrhythmia, orthostatic hypotension and vestibular paroxysmia [1,3,5] [18].

The emergency-department challenge is to separate benign recurrent causes from dangerous episodes that herald stroke or cardiac catastrophe [3,7,15]. A focused history that captures episode duration, triggers, associated features (headache, hearing loss, palpitations, syncope) and a targeted examination including ABCD2 scoring, ECG and orthostatic vital signs supports safe disposition in the majority of cases [4,7].

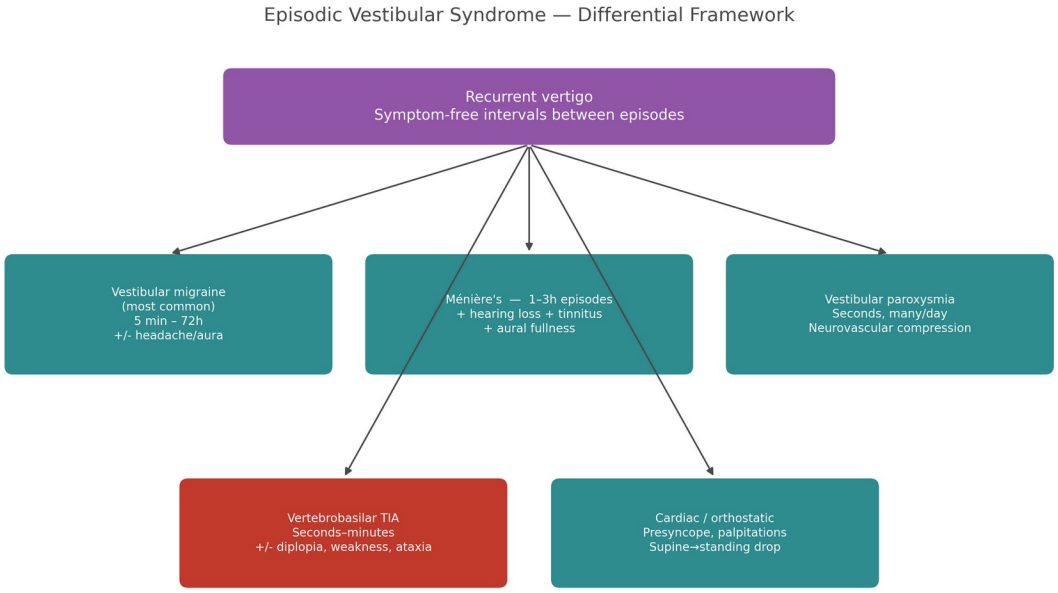


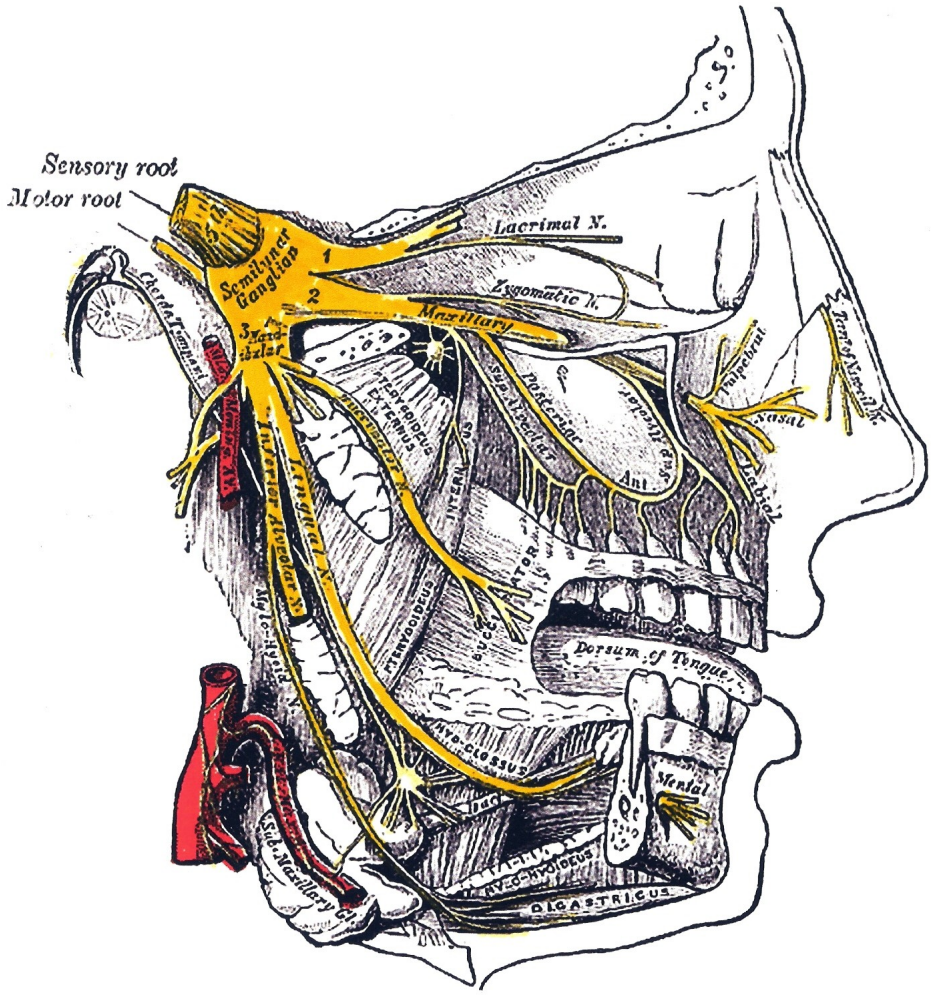
Figure 1. Differential framework for episodic vestibular syndrome.

Unlike acute vestibular syndrome, where the diagnostic question is largely binary (peripheral neuritis versus central stroke), episodic vestibular syndrome (EVS) encompasses a broad differential where benign and dangerous causes can present identically at first contact [1,3,12]. The clinician's task is therefore stratification — identifying the small subset whose recurrent vertigo signals transient ischaemia, cardiac arrhythmia, or critical posterior-fossa pathology [4,11].

EVS is defined as recurrent, episodic dizziness lasting seconds to hours, with the patient typically asymptomatic between episodes [1,5]. The clinical history carries disproportionate weight because the examination between events is often normal — the diagnosis hinges on episode duration, triggers, associated features, and accompanying neurological symptoms [3,12,15].

 Clinical Pearl: Episodic vertigo with symptom-free intervals suggests migraine, Meniere's, TIA, or cardiac cause. Systematic history narrows the diagnosis.

II. Vestibular Migraine



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Vestibular migraine is the single commonest cause of recurrent vertigo, with a lifetime prevalence of approximately 1–4% and a female predominance [2,8,18]. Bárány Society and IHS criteria require at least five episodes of moderate-to-severe vestibular symptoms lasting 5 minutes to 72 hours, and migrainous features (headache, aura or photo/phonophobia) in at least half of the attacks [1,16].

Attacks are often precipitated by migraine triggers — sleep disturbance, stress, dehydration, menstruation, visual triggers and dietary factors [8,17]. Between attacks, patients commonly describe head-motion intolerance, visual-induced dizziness and a persistent sense of unsteadiness [17,19].

 Clinical Pearl: Vestibular migraine is most common in women, often has prior migraine history, episodes last hours, and respond to migraine abortives.

ED management combines symptom control with early, accurate diagnosis [16]. Acute pharmacotherapy includes NSAIDs, triptans in appropriate candidates, antiemetics such as ondansetron or prochlorperazine, and short-term vestibular suppressants for severe vertigo [8,16].

A quiet, low-stimulus environment, IV fluids and reassurance materially reduce symptom severity [16]. Definitive diagnosis and initiation of prophylaxis are made in the outpatient setting by a vestibular clinician/physician [18,19].

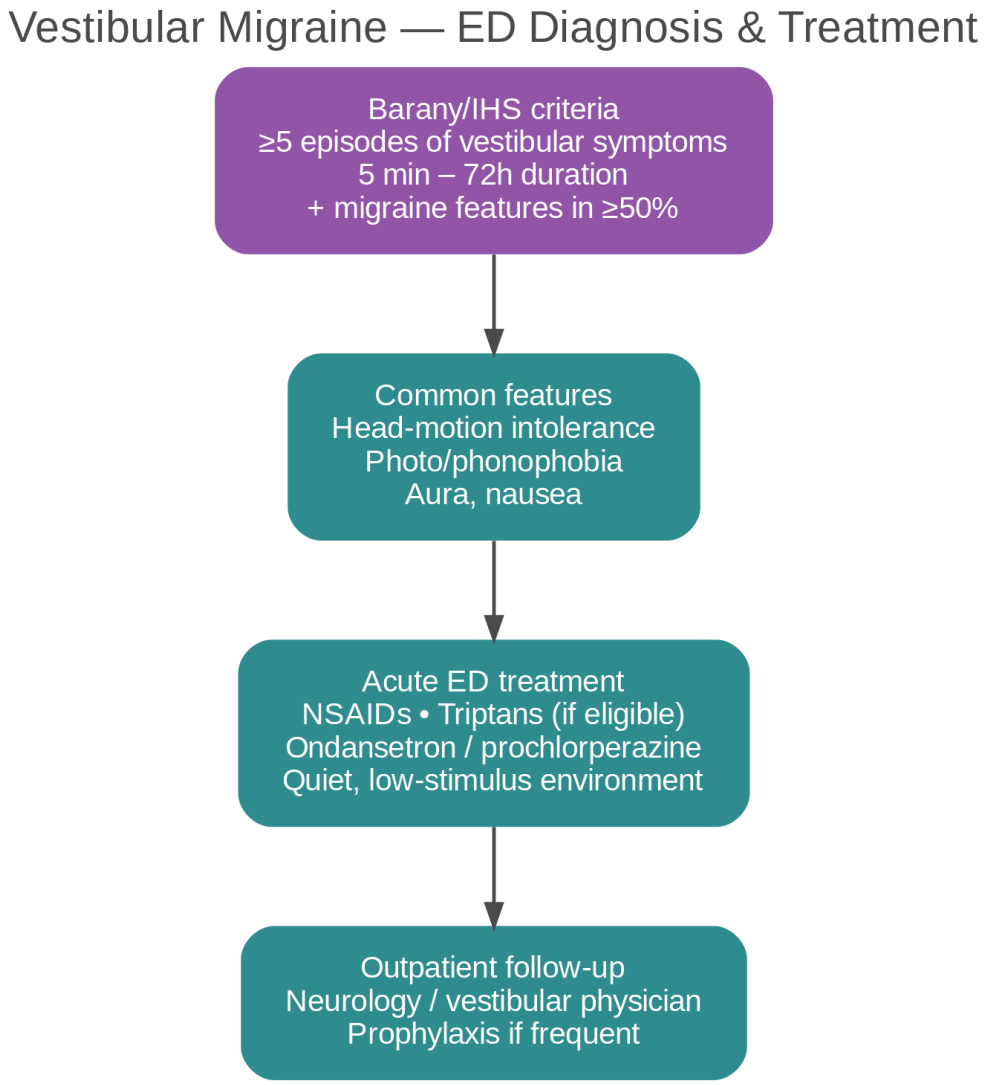
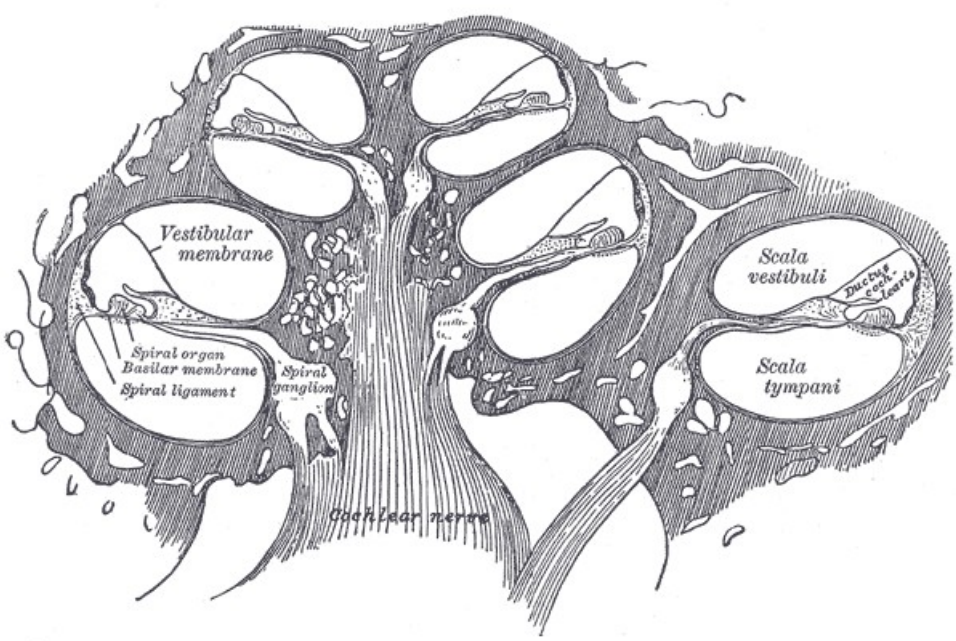


Figure 2. Vestibular migraine — ED diagnosis and treatment.

Triggers in vestibular migraine often mirror those of classical migraine: stress, sleep disturbance, dehydration, alcohol, hormonal cycling, and dietary precipitants such as aged cheeses or processed meats [13]. A symptom diary correlating attacks with triggers, sleep, and menstrual cycle is the single most useful diagnostic and therapeutic tool [13,15].

Acute attack management combines vestibular suppressants for short-term relief with migraine-specific agents (triptans, antiemetics) where photophobia or headache are present [13]. Preventive therapy — propranolol, amitriptyline, topiramate, or candesartan — is indicated when attacks exceed 4 per month or substantially interfere with function [13,15].

III. Meniere's Disease



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Ménière's disease is characterised by episodic vertigo lasting 1–3 hours, fluctuating low-frequency sensorineural hearing loss, tinnitus and aural fullness [5,6]. Episodes are sudden, disabling and may be clustered; long-standing disease may produce permanent hearing loss [5] [7].

The proposed mechanism is endolymphatic hydrops, although the trigger for acute attacks remains debated [5,6]. ED management centres on antiemetics, IV hydration, brief vestibular suppressants if severe, and referral to a vestibular clinician/physician for audiometry and long-term management [12] [8].

 Clinical Pearl: Meniere's disease: episodic vertigo + fluctuating hearing loss + tinnitus + aural fullness. Attacks are sudden and severe, lasting 1–3 hours.

Tumarkin crises (otolithic drop attacks) are sudden unprovoked falls without warning or loss of consciousness, typically in established Ménière's disease [5]. The mechanism is thought to be abrupt otolithic dysfunction producing a transient loss of postural control [5,6].

They must be distinguished from syncope (preceded by prodrome, often associated with cardiac or vasovagal triggers), seizures and vertebrobasilar TIA [3,10]. Tumarkin crises carry significant injury risk and should prompt urgent vestibular specialist review [5].

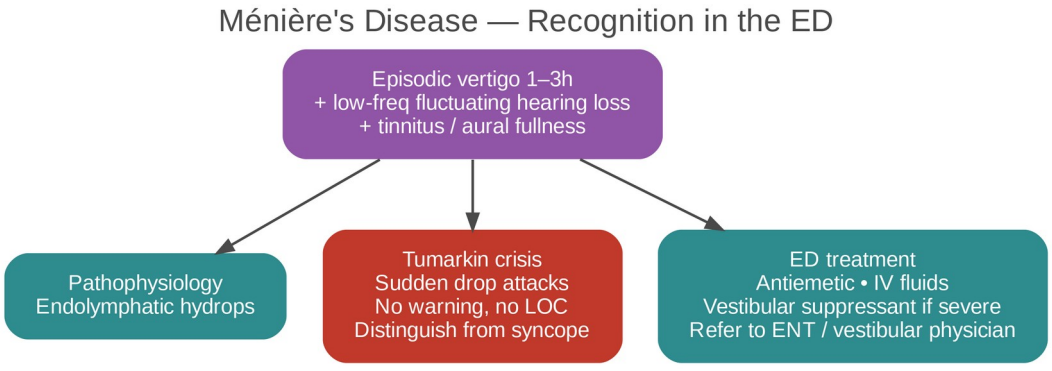
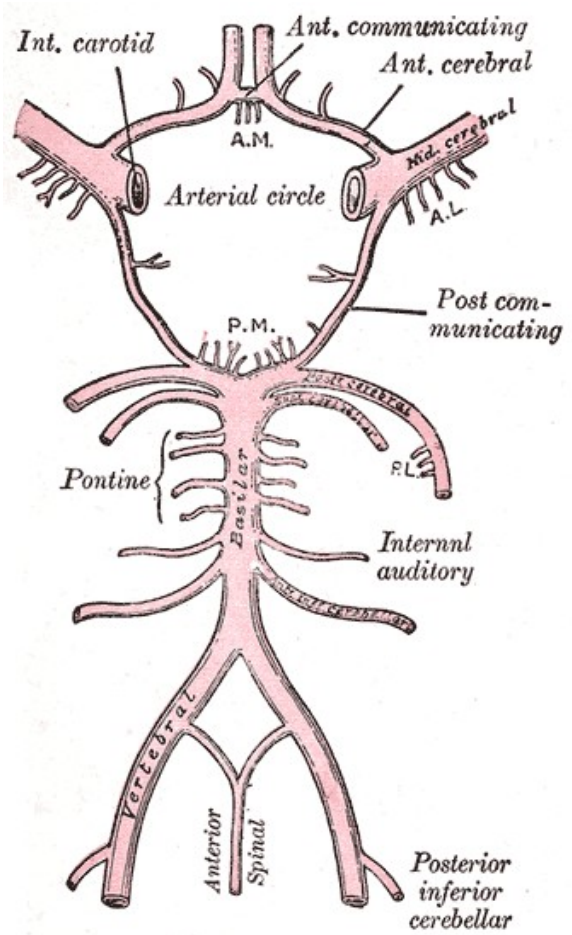


Figure 3. Ménière's disease — features and ED management.

The pathophysiology of Meniere's disease is endolymphatic hydrops — distension of the membranous labyrinth from impaired endolymph homeostasis [6]. The classical attack reflects rupture of Reissner's membrane with potassium-rich endolymph contacting hair-cell synapses, transiently silencing vestibular afferents [6,15].
First-line management is dietary sodium restriction (<1,500 mg/day) and avoidance of caffeine and alcohol, supplemented by thiazide or loop diuretics where attacks persist [6]. Second-line options include intratympanic steroids and, for refractory cases, intratympanic gentamicin or surgical intervention — decisions that should involve a vestibular clinician/physician [6,9].

⚠ **Important:** Tumarkin crisis is a sudden fall without LOC during Meniere's. Distinguish from syncope, which suggests cardiac or neurological causes.

IV. Vertebrobasilar TIA



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Vertebrobasilar TIA typically presents with brief vertigo lasting seconds to minutes, followed by complete resolution [3,10,14]. Associated symptoms — diplopia, dysarthria, hemiparesis, numbness, ataxia or visual field loss — are critical clues to a cerebrovascular cause [3,10].

Isolated vertigo is an uncommon presentation of TIA but is well described; unexplained brief vertigo in a vasculopath warrants posterior-circulation imaging [14,15]. TIA carries approximately 10–15% risk of stroke at 90 days, with the highest risk in the first 48 hours [4,10].

The ABCD2 score stratifies short-term stroke risk after TIA: Age ≥60 (1), BP ≥140/90 (1), Clinical features (unilateral weakness 2; speech disturbance without weakness 1), Diabetes (1) and Duration (≥60 min 2; 10–59 min 1) [4] [11].

Scores of 0–3 are low risk and can usually be managed via a rapid outpatient TIA clinic, whereas scores of 4–7 warrant admission, urgent MRI-DWI and CTA, antiplatelet initiation and statin therapy pending definitive imaging [4,10]. Posterior-circulation TIA may be under-scored by ABCD2, and clinical suspicion should override a low score [3,15].

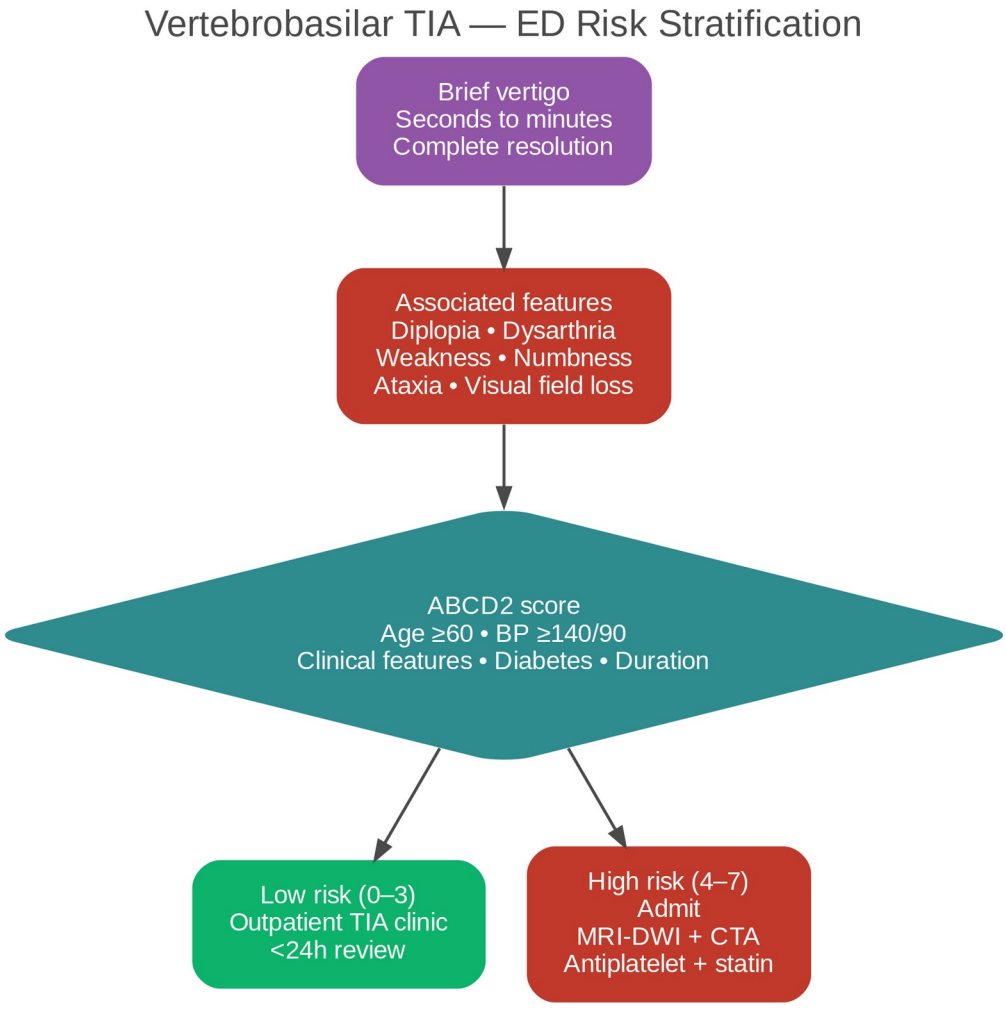
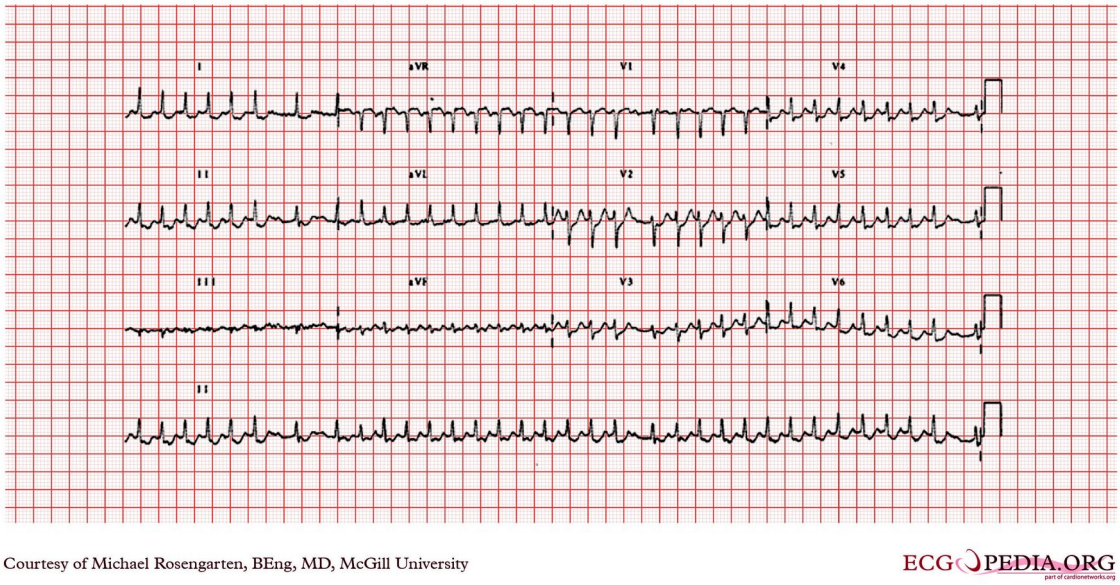


Figure 4. Vertebrobasilar TIA — ED risk stratification.

Risk-stratification scores such as ABCD2 are less reliable in posterior-circulation TIA than in anterior circulation [3,12]. A high index of suspicion, urgent vascular imaging (CTA or MRA of the head and neck), and admission for monitoring with antiplatelet therapy is the safer pathway when isolated vertigo is the only feature [3,4,11].

 Clinical Insight: Brief episodes with complete resolution + age >60 + vascular risk factors = TIA. Requires ABCD2 scoring and urgent neuro consultation.

V. Cardiac Causes



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Paroxysmal arrhythmias — atrial fibrillation, supraventricular tachycardia and ventricular tachycardia — can cause episodic presyncope and dizziness, typically with palpitations, dyspnoea or chest discomfort rather than true spinning vertigo [5].

Diagnosis relies on ECG, telemetry and in some cases Holter or implantable loop recorder, with cardiology input for management [5]. Structural heart disease (aortic stenosis, hypertrophic cardiomyopathy, pulmonary embolism) must be considered in the dizzy patient with exertional symptoms or a new murmur [5,6].

Orthostatic hypotension is defined by a fall in systolic BP of ≥20 mmHg or diastolic BP of ≥10 mmHg on standing and typically presents with lightheadedness rather than true rotational vertigo, relieved by lying down [5].

Causes include volume depletion, autonomic failure, medications (diuretics, antihypertensives, alpha blockers, antidepressants) and Parkinson's disease [5,6]. Orthostatic vital signs — supine then after 1 and 3 minutes of standing — are a cheap, high-yield bedside test [5].

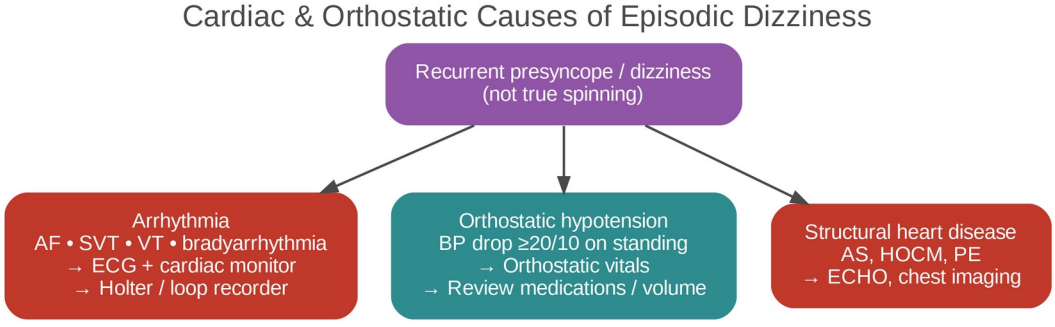

Figure 5. Cardiac and orthostatic causes of episodic dizziness.

Recurrent dizziness with palpitations, exertional triggers, family history of sudden cardiac death, or syncope warrants a cardiology consult and prolonged ambulatory monitoring [10,17]. Implantable loop recorders capture arrhythmias missed by 24–72-hour Holter monitoring and are increasingly used in unexplained recurrent dizziness with syncope [17].

 Key Point: *Cardiac causes present with palpitations, dyspnea, or chest discomfort—not true spinning vertigo. ECG and monitoring help identify arrhythmias.*

VI. Vestibular Paroxysmia

Vestibular paroxysmia presents with very brief episodes of vertigo lasting seconds to 1–2 minutes, occurring multiple times per day and often triggered by changes in head position but not solely positional [12].

The mechanism is thought to be neurovascular compression of the vestibulocochlear nerve, analogous to trigeminal neuralgia, and response to low-dose carbamazepine or oxcarbazepine is both therapeutic and diagnostic [12] [19].

Vestibular paroxysmia is a rare but treatable cause of brief recurrent vertigo, typically lasting seconds, attributed to neurovascular compression of the vestibulocochlear nerve [18]. Diagnosis is supported by short attacks (often dozens per day), response to low-dose carbamazepine or oxcarbazepine, and MRI evidence of vascular loop contact with the eighth nerve [18,19]. The condition is frequently misattributed to BPPV or vestibular migraine for years before correct diagnosis [12,18].

 Clinical Pearl: Vestibular paroxysmia: very brief episodes (seconds), multiple daily, not position-triggered, responsive to carbamazepine.

VII. Risk Stratification

Table 1. Episodic Vertigo: Red Flags

Practical risk stratification in EVS combines history (episode duration, neurological accompaniments, vascular risk), examination (interictal neurological deficits, orthostatic vitals, cardiac auscultation), and selective investigation (ECG, electrolytes, glucose, vascular imaging where indicated) [4,12,18]. Reliance on examination alone systematically under-detects TIA and arrhythmia [14].
Patients with red flags — focal neurological signs, syncope, abnormal ECG, age >60 with vascular risk factors, or sudden severe headache — require admission and urgent investigation [3,4,11]. Lower-risk patients with classical vestibular migraine or Meniere's patterns can typically be managed as outpatients with appropriate follow-up [13,15].

Feature	Concern	ED Action
Brief episodes, complete resolution, age >60, vascular risk factors	TIA	ABCD2 score; urgent neuro consult
Palpitations, dyspnea, chest pain, syncope	Arrhythmia/ Cardiac	ECG, monitoring
Dizziness with standing, relief lying down	Orthostatic hypotension	Check orthostatic vitals
Multiple daily episodes, not position-triggered	Paroxysmia or migraine	Reassure; outpatient referral

VIII. ED Management Algorithm

A structured initial assessment captures supine and standing blood pressure, heart rate and rhythm, a focused history of episode duration, frequency, positional triggers, associated headache, hearing loss, palpitations, dyspnoea and features of focal neurology [4,5,7].

An ECG should be obtained in every patient with recurrent episodic dizziness; orthostatic vitals and bedside glucose are high-yield and low-cost [5]. The HINTS+ examination retains limited utility outside the acute vestibular syndrome but directed bedside testing of nystagmus, head-impulse and gait still informs risk assessment [11,13] [14].

Patients with a normal examination, normal ECG, no features of TIA, no cardiac symptoms and stable orthostatic vitals can generally be discharged with structured outpatient follow-up — typically a vestibular physician, neurology or cardiology depending on the leading differential [7,16] [14].

Clear written safety-net advice, a symptom diary and rapid access to review shortly after discharge materially reduce representation rates [7].

Admission and urgent workup are indicated for high ABCD2 scores, ECG abnormalities suggesting arrhythmia or ischaemia, syncopal episodes, severe orthostatic hypotension refractory to volume replacement, any new focal neurology or red-flag features pointing to cerebellar or brainstem pathology [3,4,10,13] [11].

These patients warrant MRI-DWI and CTA/MRA, telemetry, and early discussion with neurology and/or cardiology [10,14,15].

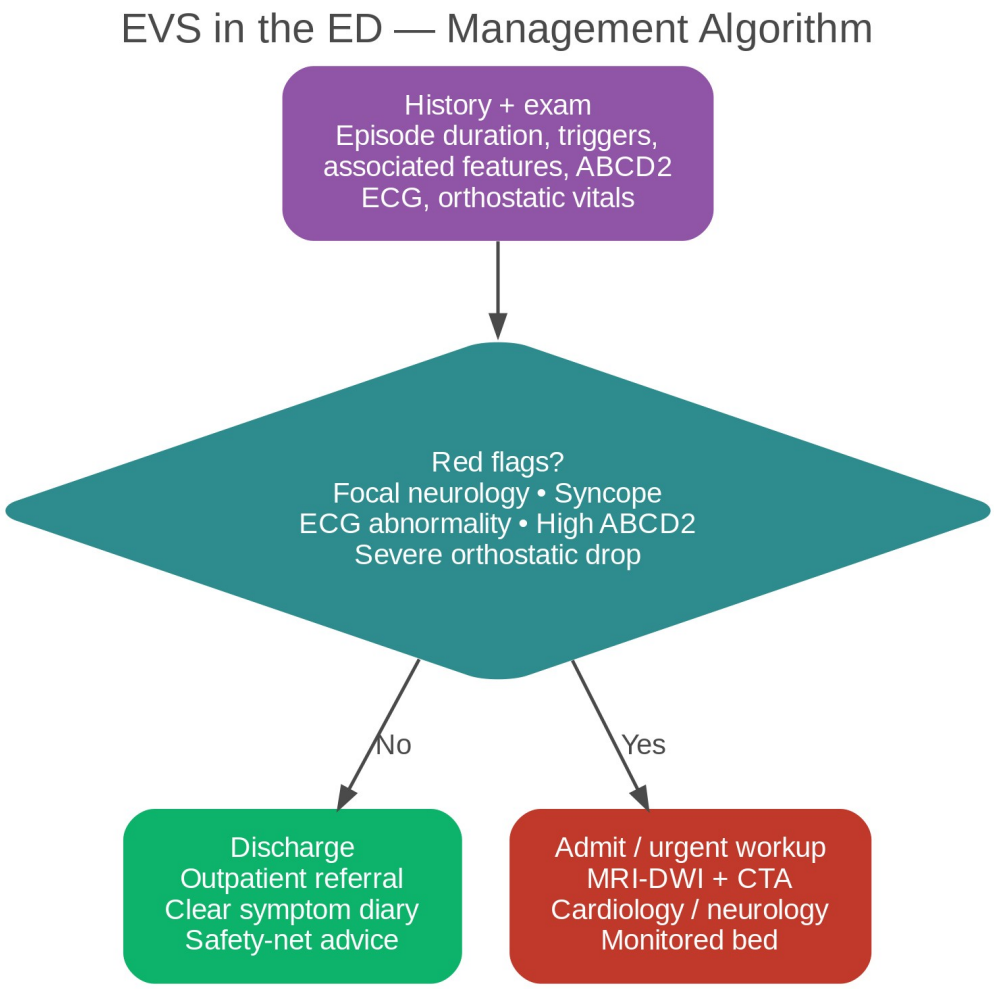

Figure 6. EVS in the ED — management algorithm.

Disposition should be tailored to underlying diagnosis and risk profile [4,18]. Vestibular migraine and Meniere's disease can typically be managed as outpatients with appropriate primary-care or neurology follow-up [13,15]. Vertebrobasilar TIA, suspected cardiac arrhythmia, and unexplained recurrent dizziness with red flags warrant admission [3,11,17].

Patient education at discharge is essential: explain the diagnosis, expected course, trigger avoidance, when to return urgently, and when to seek non-urgent specialist review [13]. Written information improves adherence and reduces re-presentation rates [18,20].

Vestibular rehabilitation is increasingly recognised as effective for chronic compensatory dizziness following episodic vestibular events, particularly persistent postural-perceptual dizziness (PPPD) that may follow vestibular migraine or Meniere's attacks [15,20].

⚠ **Important:** When uncertain about TIA or arrhythmia, be conservative: obtain ECG, consider monitoring, consult neurology. Early TIA and arrhythmia identification saves lives.

IX. Conclusions

Episodic vertigo spans a wide differential from benign peripheral disease to life-threatening cerebrovascular and cardiac causes [3,5,15]. A systematic bedside approach — structured history, ABCD2 scoring, ECG, orthostatic vitals and a low threshold for posterior-circulation imaging — allows

emergency clinicians to identify the dangerous minority while safely discharging the benign majority [4,7,10] [14].

Outpatient follow-up with a vestibular physician, neurology or cardiology closes the diagnostic loop and supports definitive treatment, including migraine prophylaxis, Ménière's disease management and secondary stroke prevention [16,18,19].

 Clinical Insight: Episodic vertigo is common and often benign, but always consider TIA in older patients with brief episodes and risk factors. Early TIA identification prevents stroke.

References

1. Lempert T, Olesen J, Furman J, et al. Vestibular migraine diagnostic criteria. J Vestib Res. 2012;22(4):167–172.

2. Neuhauser H, Leopold M, von Brevern M, et al. Interrelations of migraine and vertigo. Neurology. 2001;56(4):436–441.

3. Savitz SI, Caplan LR, Edlow JA. Ischemia of vertebrobasilar circulation. Neurology. 2012;78(1):23–30.

4. Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. ABCD2 score validation. Lancet. 2007;369(9558):283–292 [11].

5. Brandt T, Dieterich M, Strupp M. Vertigo and Dizziness. 2nd ed. Springer; 2013.

6. Baloh RW, Honrubia V, Kerber KA. Clinical Neurophysiology. 4th ed. Oxford; 2011.

7. Newman-Toker DE, Edlow JA. TiTrATE approach to acute dizziness. Neurol Clin. 2015;33(3):577–599.

8. Reploeg MD, Goebel JA. Migraine-associated dizziness. Otol Neurotol. 2002;23(3):364–371.

9. von Brevern M, Radtke A, Lezius F, et al. BPPV epidemiology. J Neurol Neurosurg Psychiatry. 2007;78(7):710–715.

10. Adams HP, Love BB, Sicks JD, et al. Warning signs of stroke. JAMA. 1998;280(14):1236–1240.

11. Halmagyi GM, Curthoys IS. Clinical sign of canal paresis. Arch Neurol. 1988;45(7):737–739.

12. Strupp M, Brandt T. Peripheral vestibular disorders. Curr Opin Neurol. 2006;19(1):47–52.

13. Saber Tehrani AS, Kattah JC, Kerber KA, et al. Astasia predicts stroke in AVS. Stroke. 2013;44(6):1659–1665.

14. Navi BB, Kaizer AM, Iadecola C, et al. Ischemic stroke in AVS. Stroke. 2012;43(10):2697–2702.

15. Kerber KA, Meurer WJ, West SL, et al. Stroke mimics of vertigo. Neurology. 2015;85(17):1490–1497.

16. Hughes E, Spiers M, De Lusignan S. Migrainous vertigo. Prim Care. 2015;42(3):371–382.

17. Balaban CD. Central vestibular network and stress responses. Front Neurol. 2018;9:340.

18. Furman JM, Balaban CD. Vestibular migraine state of the art. Headache. 2019;59(Suppl 1):3–4.

19. Von Brevern M, Clarke AH, Lempert T. Migrainous vertigo follow-up. Neurology. 2004;62(9):1623–1625.

20. Adams HP, Bendixen BH, Kappelle LJ, et al. Classification of stroke subtypes. Stroke. 1993;24(1):35–41.

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