Syncope vs Vertigo:

Sorting It Out in the Emergency Department

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How to Use This Review

Syncope and vertigo both cause acute loss of equilibrium and may present with similar chief complaints. However, they have distinct pathophysiologies, clinical features, and management strategies. This review provides a framework for rapid differentiation in the ED, reducing unnecessary testing and improving diagnostic accuracy.

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

Syncope and vertigo are frequently conflated by patients and — in poorly structured assessments — by clinicians [1,2]. Both are reported as ‘dizziness’ or ‘blackouts,’ yet they arise from entirely different physiological mechanisms and carry different prognoses [1,3].

Syncope is a transient cerebral hypoperfusion event producing loss of consciousness and postural tone with rapid spontaneous recovery [1,4]. Vertigo is an illusion of motion produced by asymmetric vestibular input without loss of consciousness [2,5]. Disequilibrium is unsteadiness without either, usually from multi-sensory or neurological impairment [2].

The ED challenge is rapid differentiation, because each category drives a different workup and disposition [1,6]. Syncope demands cardiac and autonomic assessment; vertigo demands a focused vestibular and neurological examination [1,6,9].

Confusion between the two is a common root cause of missed posterior-circulation stroke, missed arrhythmia and inappropriate discharge [1,11,14]. A clear first-pass classification — syncope, presyncope, vertigo or disequilibrium — is arguably the single highest-yield step in ED evaluation of ‘dizziness’ [1,2].

This review covers the diagnostic criteria for the common causes of syncope and vertigo, the distinguishing history and examination features, and the investigations and disposition decisions that flow from them [1,2,6].

 Key Point: *Key distinction: In syncope, the patient loses consciousness; in pure vertigo, consciousness is preserved but balance is disrupted.*

II. Defining Terms

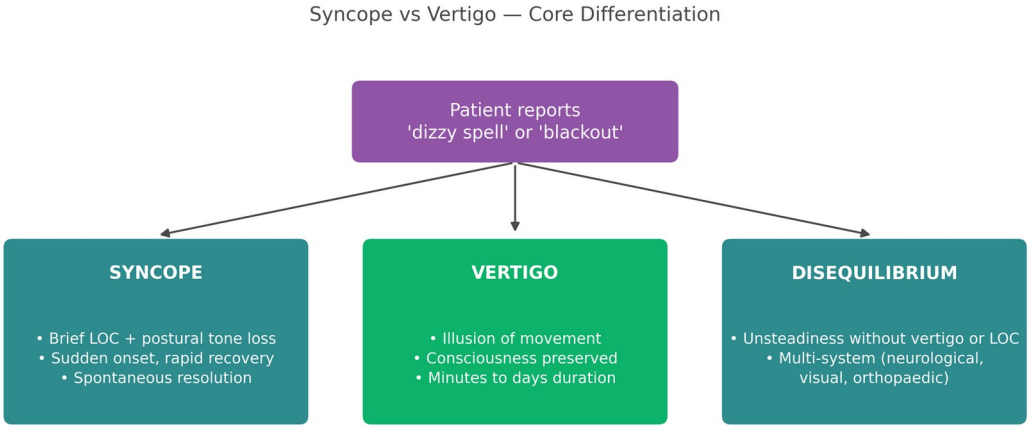

Figure 1. Syncope, vertigo and disequilibrium — core definitions.

Syncope

Syncope is defined as sudden, brief loss of consciousness and postural tone due to global cerebral hypoperfusion, followed by rapid and complete spontaneous recovery [1,4]. Common causes include vasovagal syncope, orthostatic hypotension, cardiac arrhythmia and structural cardiac disease [1,4] [2].

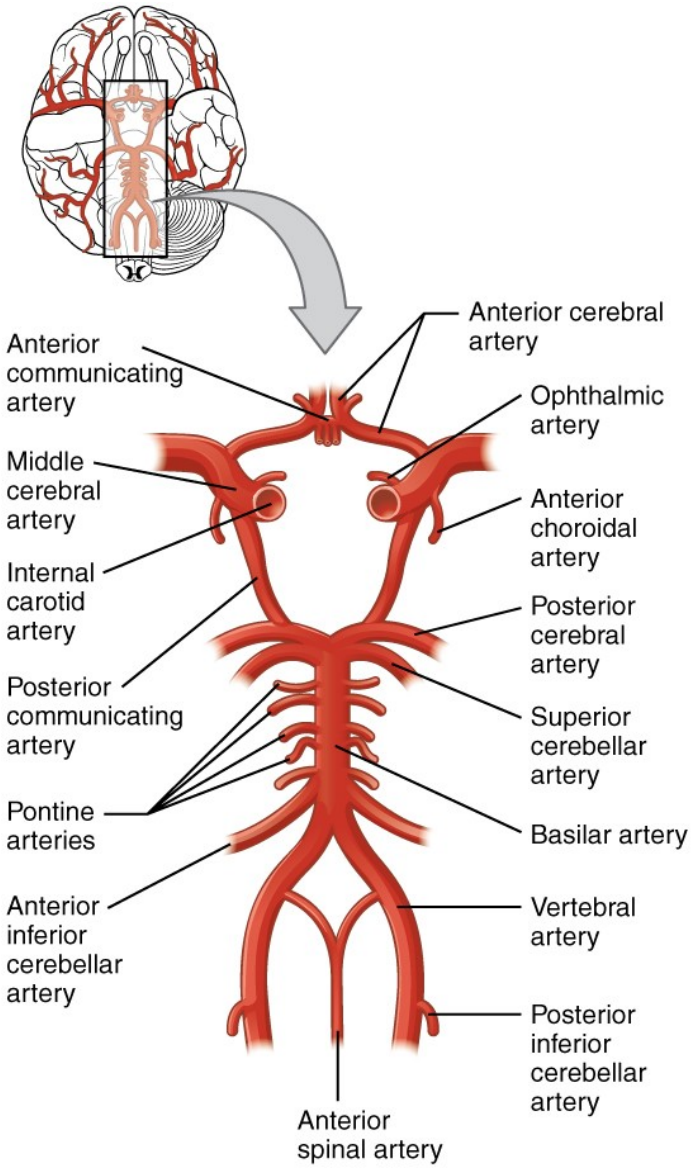

Figure A1. Circle of Willis — the cerebral arterial circle whose global hypoperfusion produces syncope.
Source: Wikimedia Commons — File:1314 Circle of WillisN.jpg — CC BY 4.0 (OpenStax Anatomy & Physiology).

Presyncope

Presyncope is the sensation of impending syncope — light-headedness, visual greying, diaphoresis, nausea — without loss of consciousness [1,4]. Its causes overlap with syncope, and high-risk presyncope (particularly exertional or with palpitations) should be worked up as syncope [1,4].

Vertigo

Vertigo is the illusion of movement — ‘the room spinning’ or ‘I’m spinning’ — generated by asymmetric vestibular input [2,5]. It is a symptom, not a diagnosis, and requires classification by time-course (acute vestibular syndrome, episodic vestibular syndrome, triggered) and accompanying features (hearing loss, focal neurology) [2,5,6].

Disequilibrium

Disequilibrium is a sense of unsteadiness without vertigo or loss of consciousness [2]. It is typically multifactorial in older patients — visual impairment, peripheral neuropathy, proprioceptive deficit, gait disorder and polypharmacy — and warrants structured falls assessment rather than vestibular-focused workup [2,18].

III. History-Taking Framework

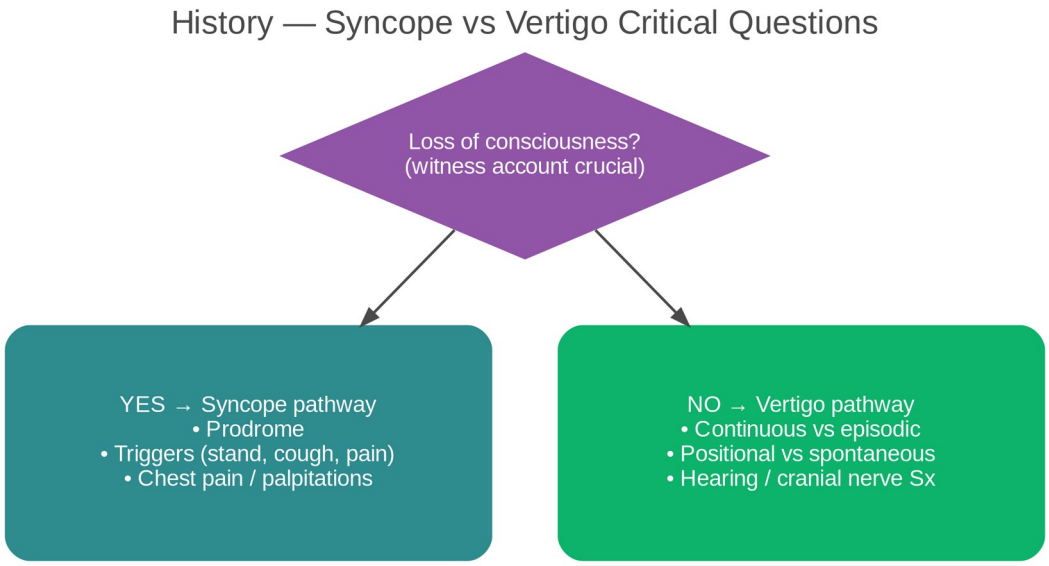

Figure 2. History-taking framework — syncope vs vertigo.

The Critical Questions

Obtain careful history from the patient and, wherever possible, a witness — witness accounts are the single most useful source of information in distinguishing syncope from non-syncopal events [1,4,6]. Document timing, prodrome, triggers, duration, recovery profile, injuries, associated symptoms and prior similar episodes [1,4].

- **Timing and onset: syncope is characteristically sudden and brief (seconds to <1 minute); vertigo often has a more gradual onset and lasts minutes to hours or longer [1,2,4]. Sudden severe vertigo of short duration should raise suspicion for posterior-circulation TIA [6,11].**
- **Prodrome: vasovagal and orthostatic syncope typically have a recognisable prodrome (warmth, nausea, tunnel vision, sweating); cardiac syncope is frequently prodrome-free [1,4]. Vertigo is usually abrupt and does not have autonomic prodromal features [2,6].** [6]
- **Did the patient lose consciousness completely? Witnessed pallor, loss of tone and rapid recovery support syncope [1,4]. Preserved consciousness with an illusion of movement and head-motion intolerance supports vertigo [2,5]. Eye-witness testimony is particularly important when the patient has amnesia for the event [1,4].**
- **Triggers: vasovagal syncope follows sudden standing, Valsalva, cough, micturition, pain or fear [1,4]. Vertigo is commonly triggered by head position change (BPPV), visual motion (vestibular migraine, PPPD) or occurs spontaneously (vestibular neuritis, Ménière's, stroke) [2,5,15].** [6]
- **Associated symptoms: chest pain, palpitations, dyspnoea or exertional onset direct attention to cardiac syncope [1,4,7]. Nausea, vomiting, nystagmus, hearing change, focal**

neurology, ataxia or head-motion intolerance direct attention to vestibular or central causes [2,6,11].

- **Injury: syncope patients often sustain falls from standing because postural tone is abruptly lost, so facial, head or dental injuries are common [1,4]. Vertigo patients tend to sit or lie down protectively; significant injury from 'just vertigo' should prompt reconsideration of the working diagnosis [2,6].**
- **Recovery: syncope resolves within minutes with prompt return to baseline cognition [1,4]. Prolonged post-event confusion, focal neurology or weakness should prompt assessment for seizure, stroke or metabolic cause [1,4,6].**

Red Flags in History

Cardiac syncope red flags are exertional syncope, syncope preceded by palpitations or chest pain, syncope while supine, family history of sudden cardiac death, and syncope with structural heart disease or known arrhythmia [4,7]. Any of these mandates cardiac monitoring and urgent cardiology assessment [4,7] [10].

Posterior-circulation stroke/TIA features include focal weakness, dysarthria, diplopia, severe occipital headache, crossed sensory loss or focal cranial nerve deficits accompanying vertigo [6,11]. Any such feature should trigger the stroke pathway including CT, MRI-DWI and stroke team input [11,19].

 Clinical Insight: A detailed witness account is invaluable. Ask witnesses: "Did the patient's eyes open throughout?" "How long were they unconscious?"

IV. Orthostatic Hypotension

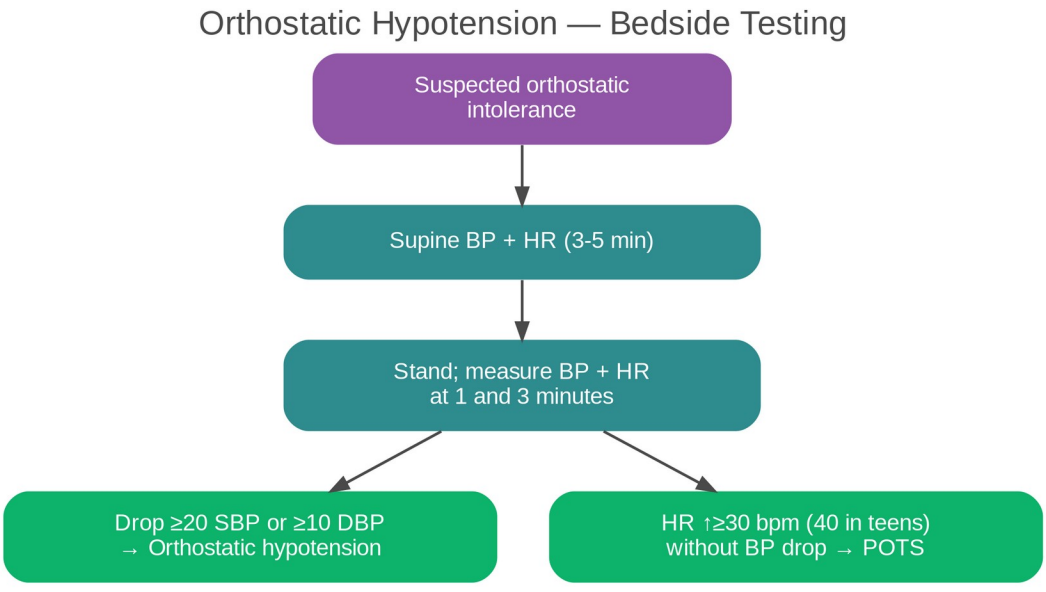

Figure 3. Orthostatic vitals — bedside protocol and interpretation.

Definition and Pathophysiology

Orthostatic hypotension (OH) is a sustained drop in systolic BP of ≥20 mmHg or diastolic BP of ≥10 mmHg within 3 minutes of standing [8]. It produces presyncope or syncope on assuming upright posture and is common in older adults, diabetics, and patients on antihypertensives or diuretics [8,18].

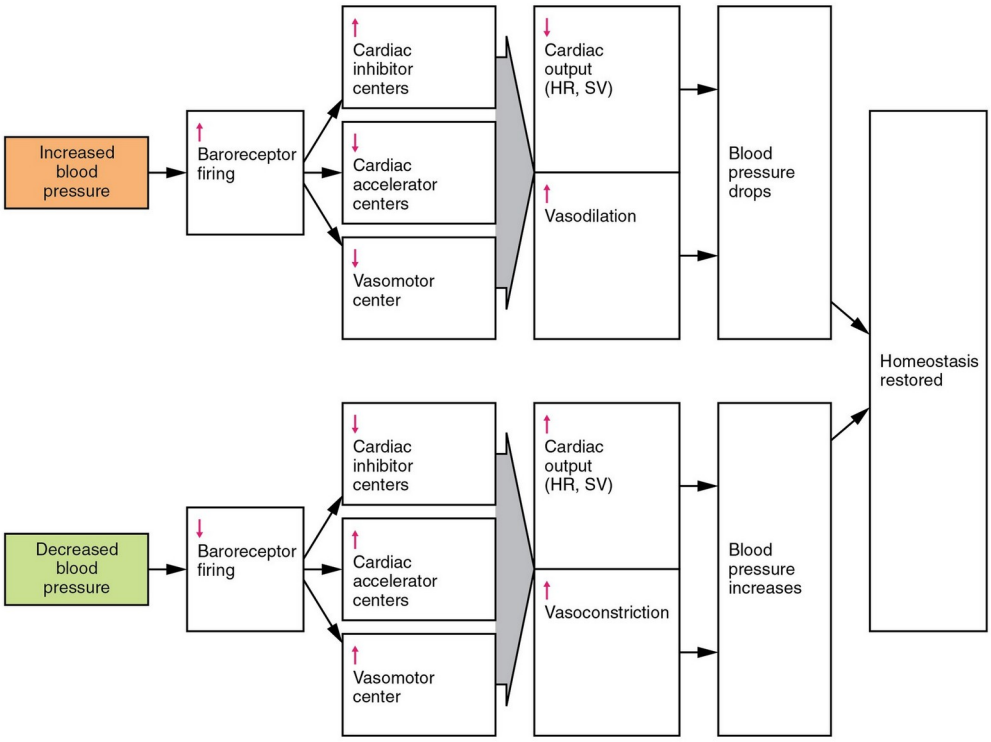

Figure A2. Baroreceptor reflex arc — the autonomic circuit whose failure produces orthostatic hypotension.
Source: Wikimedia Commons — File:2116 Baroreceptor Reflex Flow Art.jpg — CC BY 3.0 (OpenStax College).

Causes

Hypovolaemic causes include dehydration, acute blood loss, severe diarrhoea and excessive diuretic use [8]. These are usually reversible with rehydration and medication review [8].

Medication-related OH is common: diuretics, antihypertensives (especially alpha-blockers), tricyclic antidepressants, antipsychotics, nitrates, and PDE5 inhibitors are frequent culprits [8,18]. Medication review and rationalisation are first-line management, particularly in frail older adults [8,18].

Autonomic failure underlies OH in diabetes, Parkinson's disease, multiple system atrophy and pure autonomic failure [8]. Pattern recognition — OH alongside constipation, erectile dysfunction, urinary symptoms and thermoregulatory disturbance — suggests primary autonomic disease and warrants specialist referral [8].

Bedside Testing

Measure supine BP and HR after 3–5 minutes recumbency [8]. Have the patient stand and measure BP and HR at 1 and 3 minutes [8]. Symptomatic reproduction of presyncope during standing increases diagnostic confidence [8].

A drop ≥20 mmHg in SBP or ≥10 mmHg in DBP within 3 minutes defines orthostatic hypotension [8]. HR increase ≥30 bpm (≥40 in adolescents) without significant BP drop, sustained ≥10 minutes, suggests postural orthostatic tachycardia syndrome (POTS) [8,12] [4].

 Key Point: *Always measure orthostatic vitals in patients with syncope or presyncope. Dehydration is the most common, most easily treated cause.*

V. Cardiac Arrhythmia

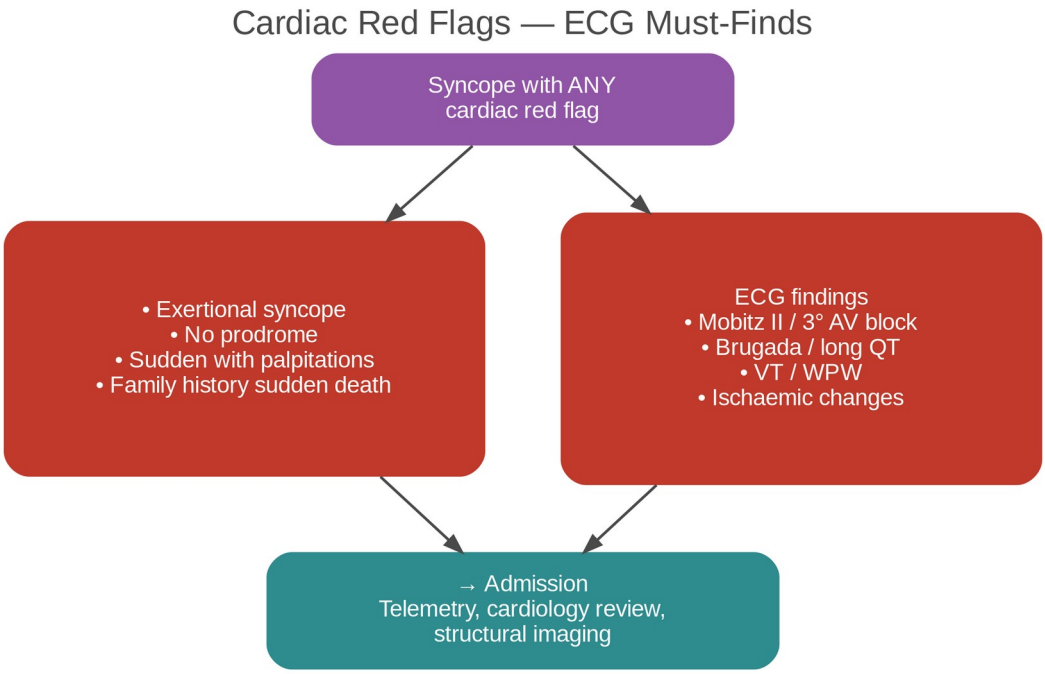

Figure 4. Cardiac red flags — ECG findings mandating admission [11].

Arrhythmia as a Cause of Syncope

Arrhythmias cause approximately 10–15% of syncope presentations and carry the highest short-term mortality of any syncope mechanism [4,7]. Syncope without prodrome, syncope during exertion, syncope in the supine position and syncope with palpitations are characteristic [4,7].

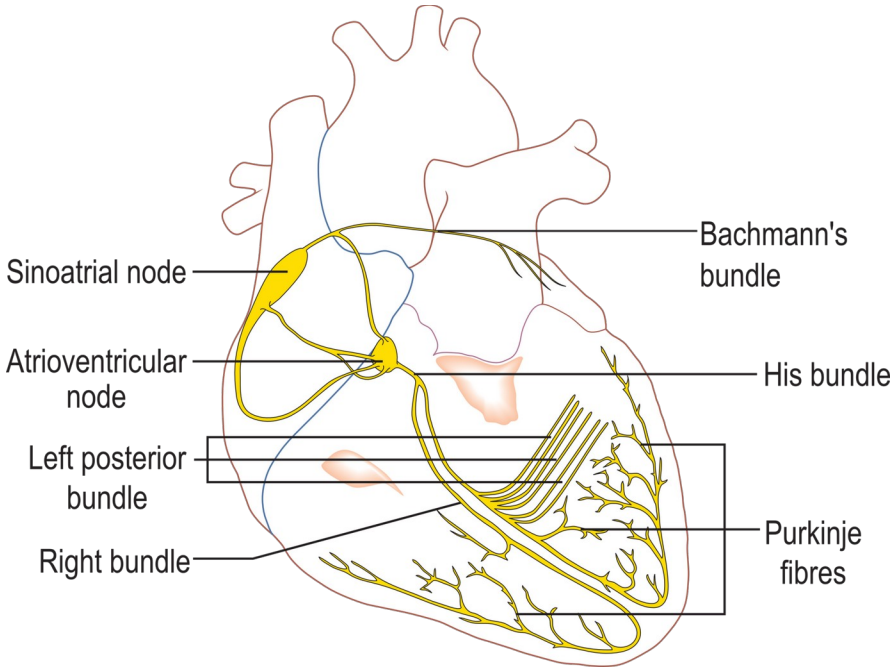

Figure A3. Cardiac conduction system — SA node, AV node, His–Purkinje pathway; sites of high-risk block.
Source: Wikimedia Commons — File:Conductionsystemoftheheart.png — CC BY 3.0 (OpenStax College).

Key arrhythmias include bradycardia (high-grade AV block, sinus arrest), tachyarrhythmias (SVT, ventricular tachycardia, atrial fibrillation with rapid ventricular response), and hereditary channelopathies (long QT, Brugada, CPVT) [4,7]. Any of these mandates admission to a monitored bed and cardiology review [4,7].

ECG Interpretation

The ECG is the primary ED tool in syncope evaluation [4,7]. Look for bradycardia (<40 bpm), prolonged QT interval (>500 ms), AV block, bundle branch block, pre-excitation, Brugada pattern, ARVD signs, and ischaemic changes [4,7]. A normal ECG does not exclude arrhythmia but lowers pre-test probability [4,7] [11].

High-risk ECG findings include Mobitz II or third-degree AV block, bifascicular block, sustained ventricular tachycardia, Brugada pattern, long or short QT, and new ischaemic changes — any of these mandates admission and cardiology input [4,7]. Prolonged monitoring (telemetry, Holter, implantable loop recorder) is appropriate when initial ECG is normal but suspicion remains [4,7] [11].

⚠ **Important:** Any patient with exertional syncope, syncope without prodrome, or high-risk ECG findings requires admission for cardiac monitoring. Do not discharge.

VI. Carotid Sinus Hypersensitivity

Pathophysiology and Population

Carotid sinus hypersensitivity (CSH) is an exaggerated baroreceptor response producing syncope through bradycardia (cardioinhibitory), hypotension (vasodepressor) or both (mixed) [9]. It is most common in older men and may be precipitated by head turning, tight collars or shaving [9].

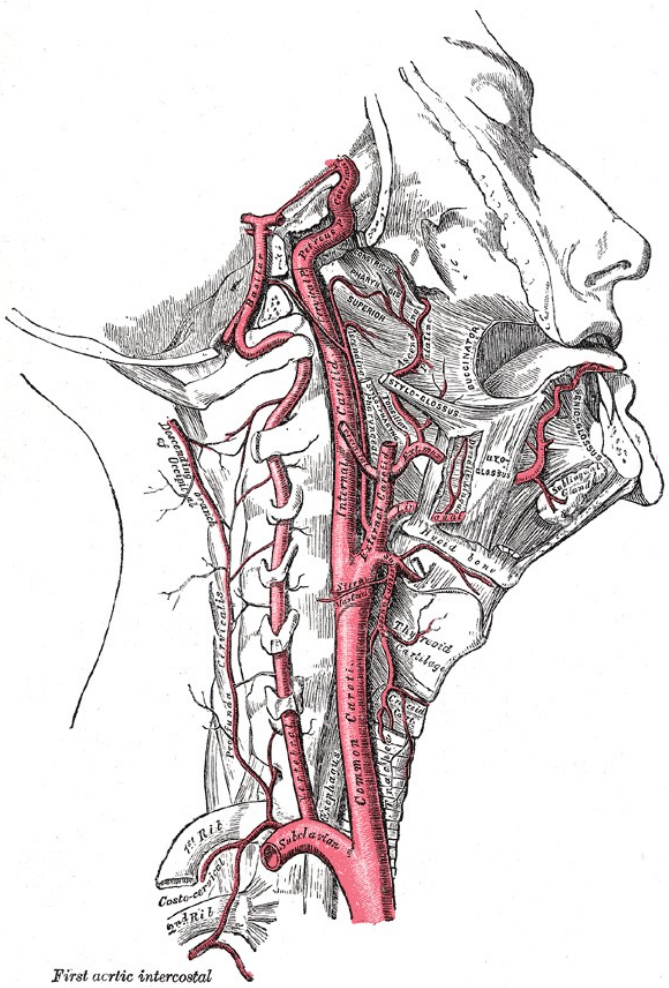

Figure A4. Carotid bifurcation and carotid sinus — mechanoreceptor site targeted by carotid sinus massage.
Source: Wikimedia Commons — File:Gray513.png — Public Domain (Gray's Anatomy, 1918).

Carotid Sinus Massage Testing

Carotid sinus massage is performed under continuous ECG and BP monitoring: gentle pressure for 5–10 seconds, first supine then upright [9]. A pause >3 seconds (cardioinhibitory) or SBP drop >50 mmHg (vasodepressor) reproducing symptoms is diagnostic [9] [11].

Contraindications include carotid bruits, recent stroke or TIA within 3 months, and known carotid stenosis [9]. In appropriate patients, pacemaker implantation markedly reduces recurrent syncope in cardioinhibitory CSH [9].

 Clinical Pearl: CSH is easily missed in elderly patients. If history suggests positional trigger, consider CSM testing.

VII. POTS and Dysautonomia

Postural Orthostatic Tachycardia Syndrome

Postural orthostatic tachycardia syndrome (POTS) is a form of dysautonomia defined by sustained HR rise ≥30 bpm (≥40 in adolescents) within 10 minutes of standing, without significant orthostatic

hypotension, accompanied by orthostatic symptoms [8,12]. It is strongly associated with hypermobile EDS, long-COVID and post-viral syndromes [12] [4].

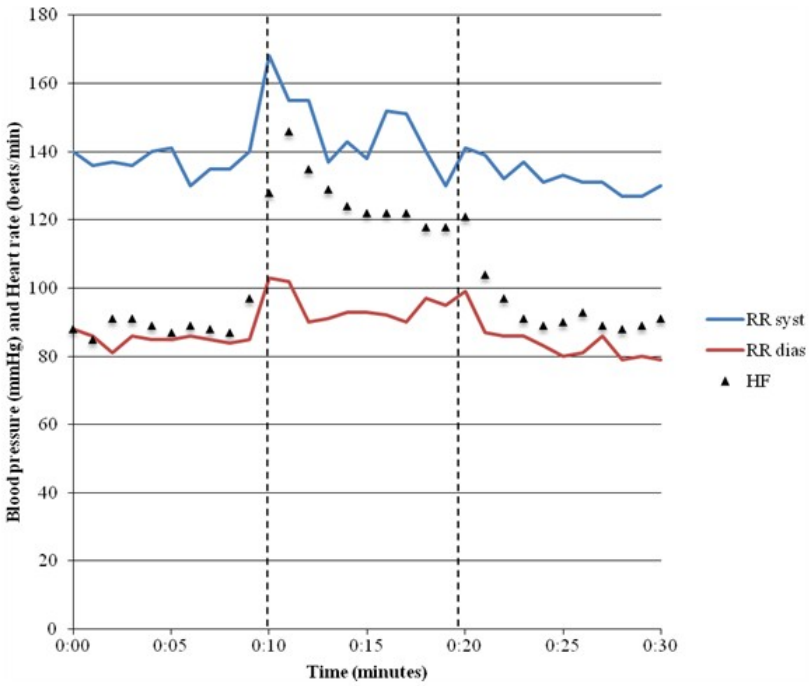

Figure A5. POTS tilt-table trace — sustained tachycardia without BP drop on upright tilt.
Source: Wikimedia Commons — File:Tilt table test showing POTS.webp — CC BY 4.0 (Furryscaly).

POTS predominantly affects women aged 15–50 (~75% female) [12]. Symptoms include light-headedness, palpitations, fatigue, cognitive fog and exercise intolerance [12]. Differential diagnosis includes hyperthyroidism, anaemia, phaeochromocytoma and inappropriate sinus tachycardia — these should be excluded before diagnosing POTS [12] [18].

Diagnosis and Management

Diagnosis is clinical, supported by active standing test or tilt-table study [12]. Management combines increased salt and fluid intake (target 3 L/day fluid, 10 g/day salt), compression garments, graded reconditioning, and — where required — pharmacotherapy with beta-blockers, ivabradine, midodrine or fludrocortisone [12].

 Key Point: *Young woman with presyncope and HR increase >30 bpm on standing, but no BP drop = POTS. First-line treatment is hydration and salt.*

VIII. Overlap Syndromes

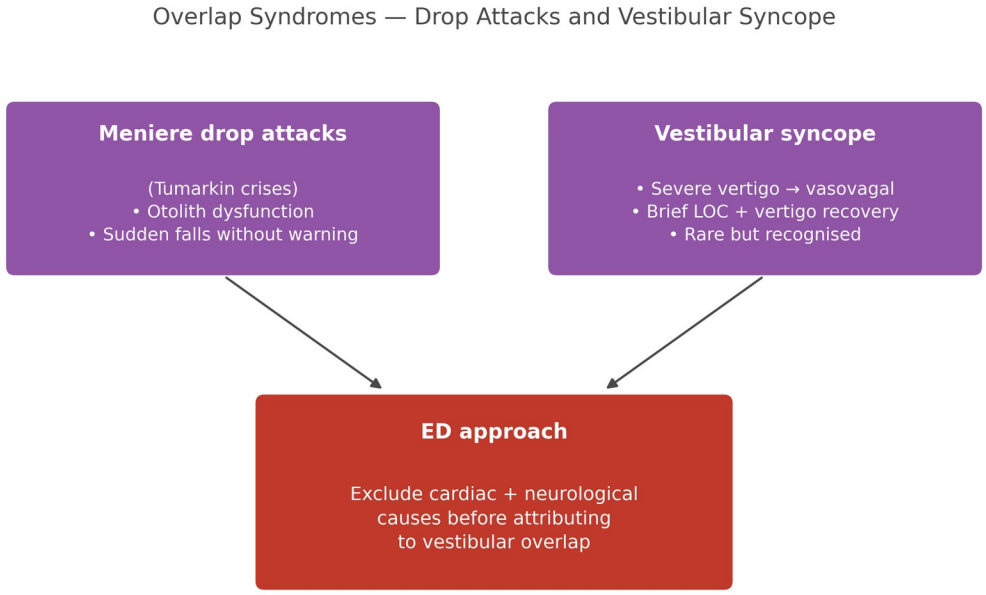

Figure 5. Overlap syndromes — Tumarkin drop attacks and vestibular syncope.

Meniere's Drop Attacks

Ménière's disease can present with drop attacks (Tumarkin otolithic crises) — sudden loss of postural tone without warning or loss of consciousness, thought to result from transient otolith dysfunction [13]. These carry a significant injury risk and are an indication for aggressive Ménière's management [13].

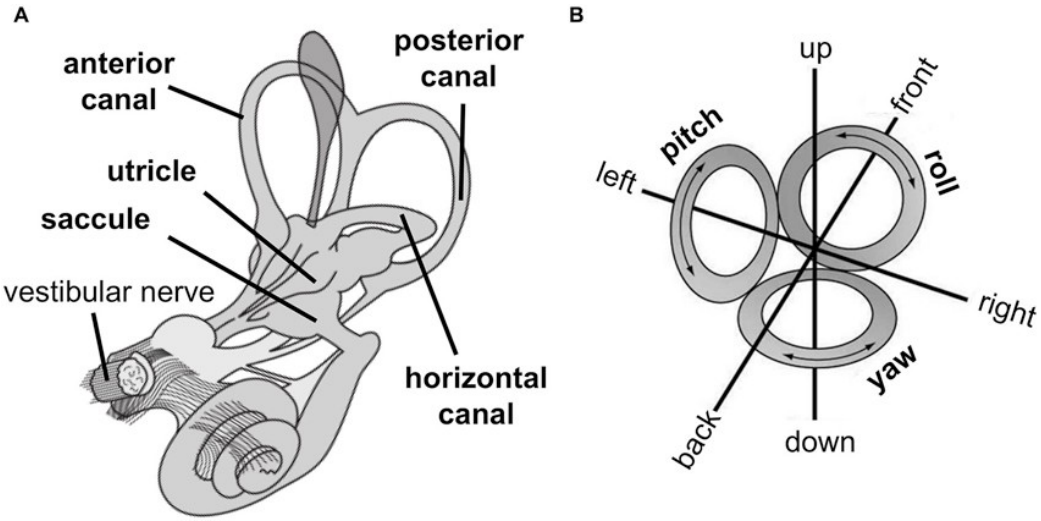

Figure A6. Peripheral vestibular organs — otolith organs whose dysfunction underlies Tumarkin drop attacks.
Source: Wikimedia Commons — File:Peripheral vestibular organs in the inner ear.jpg — CC BY 3.0 (de Waele et al.).

Vestibular Syncope

Rarely, severe acute vertigo can trigger a vasovagal response producing secondary syncope — so-called vestibular syncope [15]. Recognition requires consistent temporal sequence (vertigo first, LOC second) and exclusion of cardiac and primary neurological causes [15] [6].

 Clinical Insight: If patient reports "sudden loss of balance with consciousness preserved," think vertigo, not syncope.

IX. Investigation Algorithm

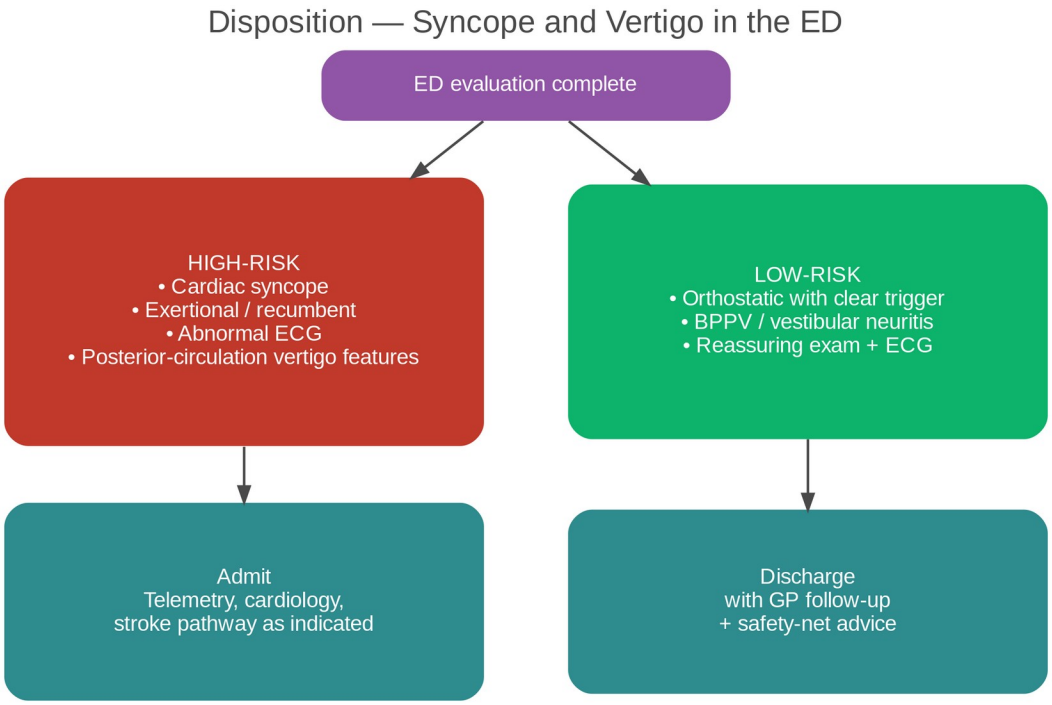

Figure 6. Disposition — high- vs low-risk syncope and vertigo.

Initial Assessment

All syncope patients warrant structured assessment: history, witness account, vital signs including orthostatic measurements, full cardiovascular and neurological examination, and 12-lead ECG [1,4,6]. Further investigations are guided by risk stratification [1,4] [11].

Risk Stratification

High-risk features mandating admission include exertional syncope, syncope with recumbent onset, abnormal or high-risk ECG, severe structural heart disease, heart failure, syncope causing significant injury, family history of sudden cardiac death, and age >65 with syncope of unclear cause [4,7]. Validated tools such as the Canadian Syncope Risk Score aid disposition [4,7] [10].

Low-risk syncope suitable for discharge includes orthostatic hypotension with identified reversible trigger, vasovagal syncope with clear prodrome and trigger, and young patients with normal ECG, normal examination and no family history of sudden death [4,7]. Safety-netting and GP follow-up are essential; repeated or unexplained syncope warrants specialist review [4,7] [4].

Table 1 summarises the key distinguishing features of syncope, presyncope, vertigo and disequilibrium and maps each to first-line investigation and disposition [1,2,4,6].

Feature	Syncope	Vertigo	Presyncope
Consciousne	Lost (brief)	Preserved	Preserved

Feature	Syncope	Vertigo	Presyncope
ss			
Prodrome	Often (lightheadedness)	Absent or abrupt	Yes
Nystagmus	Absent	Present	Absent
Vertigo (spinning)	No	Yes (hallmark)	No
Duration	Seconds to 1–2 min	Hours to days	Seconds to minutes
Recovery	Rapid, alert	Slower, nausea	Rapid
Triggers	Stand, Valsalva, pain	Head movement	Stand quickly
Injury risk	High (LOC)	Moderate	Low
First-line test	ECG, orthostatic vitals	Vestibular exam	ECG, orthostatic vitals

X. Conclusions

Syncope and vertigo are distinct diagnoses with different mechanisms, workups and dispositions [1,2]. The key historical elements — loss of consciousness, prodrome, triggers, associated symptoms, and recovery profile — reliably separate the two in the large majority of ED presentations [1,4,6].

A systematic history, orthostatic vital signs, a 12-lead ECG and a focused neurological examination rapidly stratify risk [1,4,7]. High-risk features — cardiac red flags, exertional onset, abnormal ECG, posterior-circulation features — mandate admission; low-risk cases with reassuring workup can be safely discharged with GP follow-up and clear safety-netting [4,7] [11].

References

1. Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for syncope. Eur Heart J. 2018;39(21):1883–1948.

2. Low PA, Sandroni P, Joyner M, Shen WK. Postural tachycardia syndrome. J Cardiovasc Electrophysiol. 2009;20(3):352–358.

3. Freeman R, Abuzinadah AR, Gibbons C, et al. Orthostatic hypotension. J Am Coll Cardiol. 2018;72(11):1294–1309.

4. Ganzeboom KS, Colman N, Reitsma JB, et al. Prevalence and triggers of arrhythmias. J Am Coll Cardiol. 2003;41(2):280–286.

5. Brignole M, Auricchio A, Baron-Esquivias G, et al. 2013 ESC guidelines on syncope. Eur Heart J. 2013;34(30):2381–2429.

6. Newman-Toker DE, Edlow JA. TiTrATE approach. Ann Emerg Med. 2016;68(2):175–186.

7. Grubb BP. Postural tachycardia syndrome. Circulation. 2008;117(21):2814–2817.

8. Raj SR. POTS: pathophysiology, diagnosis & management. Indian Pacing Electrophysiol J. 2006;6(2):84–99 [18].

9. Kenny RA, Travers B, Unsworth A, et al. Dropped attacks in older adults. Age Ageing. 2007;36(4):405–410.

10. Baron-Esquivias G, García-Alberola A. Syncope diagnosis and management. Rev Esp Cardiol. 2012;65(4):327–337.

11. Cheshire WP Jr, Fealey RD, Boes CJ, Cortese NA. Autonomic dysfunction in Ehlers-Danlos. Clin Auton Res. 2006;16(4):280–286.

12. Fleg JL, Kennedy HL. Cardiac arrhythmias in healthy elderly. Chest. 1982;81(3):294–299.

13. Calkins H, Shyr Y, Frumin H, et al. Clinical history in syncope differentiation. Am J Med. 1995;98(4):365–373.

14. Serletis A, Florea VG, Sheldon RS, Wieland JM. Paroxysmal atrial fibrillation. J Am Coll Cardiol. 1994;23(3):749–755.

15. Benditt DG, Ferguson DW, Grubb BP, et al. Tilt table testing. J Am Coll Cardiol. 1996;28(2):263–275.

16. Disertori M, Brugada J, Brugada R. Arrhythmia management. Eur Heart J. 2011;32(14):1999–2009.

17. Zimetbaum P. Arrhythmias in athletes. Curr Treat Options Cardiovasc Med. 2014;16(12):345.

18. Strickberger SA, Benson DW, Buss BF, et al. Syncope evaluation. J Am Coll Cardiol. 2006;47(2):473–484.

19. Albright KC, Abdelhak T, Barreto AD, et al. TIA rapid evaluation trial. Stroke. 2014;45(1):107–112.

20. Shen WK, Sheldon RS, Benditt DG, et al. 2017 ACC/AHA Atrial Fibrillation Guideline. J Am Coll Cardiol. 2019;74(1):104–132.

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