Orthostatic & Medication-Related Dizziness:

Recognition and Management of Common Iatrogenic and Postural Causes of Dizziness in General Practice

Vestibular Medicine for General Clinicians

Topic 9 of 14

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How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with orthostatic or medication-related dizziness.

The review is designed to be read in a single 20-30 minute sitting, or used as a desktop reference. It is supported by a two-page A4 cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

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, emergencies, and critical safety points requiring immediate action.

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I. Why Orthostatic and Medication-Related Dizziness Matter to Every General Clinician

Orthostatic intolerance is one of the most common, most under-diagnosed, and most easily reversible causes of dizziness presenting to primary care. Population studies estimate orthostatic hypotension (OH) in 6–10% of community-dwelling adults, rising to 20–30% in those over 75 and to over 50% in nursing-home residents [1,2]. In Australian general practice cohorts, more than one in five patients presenting with "dizziness" has either classical OH, delayed OH, or a clinically significant medication-induced postural drop [3].

Despite this prevalence, the diagnosis is missed routinely. Active stand testing is performed in fewer than 30% of older patients presenting with dizziness or falls in primary care, and fewer still have a structured medication review at the same visit [4]. The cost of this miss is substantial: orthostatic dizziness is an independent predictor of falls, fracture, cognitive decline, and all-cause mortality [5,6].

□ **Key Point:** Orthostatic and medication-related dizziness are among the few causes of dizziness where a five-minute bedside test and a structured medication review can make the diagnosis and guide curative treatment in a single GP consultation.

Three features make these conditions particularly relevant for the general clinician. First, they overlap heavily with vestibular disease - many patients with BPPV, vestibular migraine, or PPPD also have orthostatic contributors that worsen their symptoms. Second, they are often iatrogenic; a careful medication review is curative in 30–40% of cases [7]. Third, the consequences of missing orthostatic dizziness in older adults - falls, fractures, hospital admission, and loss of independence - are disproportionate to the ease of detection.

This review is designed for the general clinician who has 10–15 minutes with a dizzy patient and needs a practical, evidence-based pathway: how to perform the bedside test, how to read it, how to identify and modify the culprit medications, and when first-line lifestyle measures should give way to pharmacotherapy or referral.

II. Cardiovascular Pathophysiology of Postural Symptoms

Standing transfers approximately 500–800 mL of blood from the central circulation into capacitance vessels of the lower extremities and abdomen within seconds. Cerebral perfusion is preserved through a tightly orchestrated neural reflex: aortic and carotid baroreceptors detect a fall in pressure, sympathetic outflow drives peripheral vasoconstriction and a rise in heart rate, and venous return is augmented by skeletal muscle pumping [8]. Failure of any step in this loop causes cerebral hypoperfusion and the hallmark presyncopal symptoms - lightheadedness, blurred vision, neck and shoulder ache (the "coathanger" sign), and occasionally syncope.

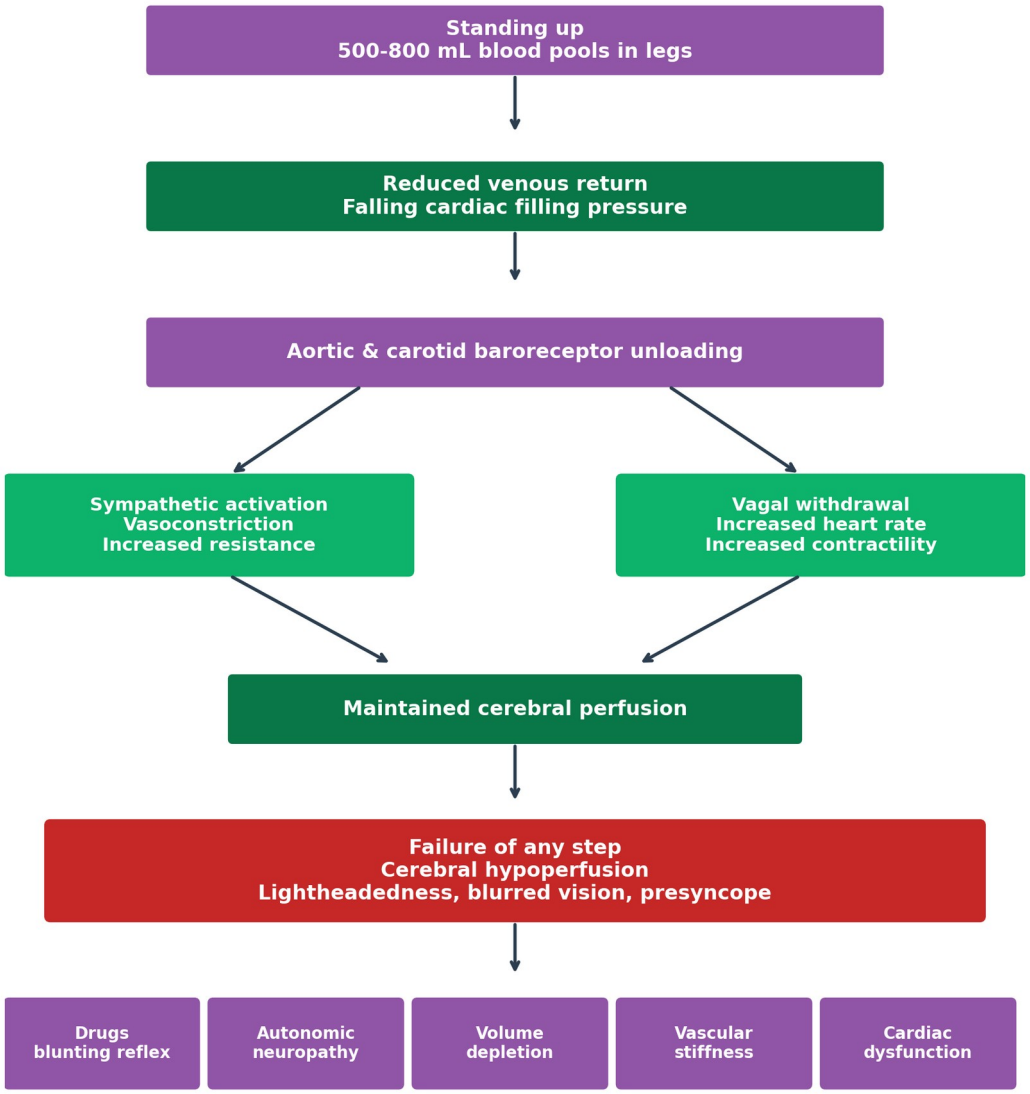

Figure 1. — Cardiovascular pathophysiology of orthostatic intolerance - the reflex loop that maintains cerebral perfusion on standing and the points at which it fails.
Source: Australian Dizziness Clinics - clinical flowchart.

Five mechanisms account for the great majority of clinical presentations:

- Reflex (autonomic) failure. Diabetic, amyloid, or Parkinsonian autonomic neuropathy disables the sympathetic vasoconstrictor response. The HR fails to rise on standing, the hallmark of neurogenic OH.
- Volume depletion. Dehydration, blood loss, diuretic use, or salt restriction reduces effective circulating volume. The reflex itself is intact but cannot compensate.
- Pharmacological blockade. Antihypertensives, alpha-blockers, vasodilators, antidepressants, and antipsychotics all blunt one or more limbs of the reflex.
- Vascular stiffness and venous pooling. Aging large arteries lose compliance; venous valvular incompetence and varicosities increase pooling. Standing tolerance falls.
- Cardiac dysfunction. Severe AS, restrictive cardiomyopathy, or arrhythmia can present as orthostatic dizziness when stroke volume cannot rise to meet upright demand.

□ **Clinical Insight:** The single most useful piece of physiology to remember at the bedside is that an inadequate HR rise on standing implicates the autonomic nervous system or a heart-rate controlling drug (most often a beta-blocker), whereas a marked HR rise of 30 bpm or more with a preserved BP suggests POTS - a quite different problem with quite different management.

III. The Clinical Spectrum - From Initial OH to Delayed OH and POTS

Orthostatic dizziness is not a single disorder. The Bedrosian-Freeman 2011 consensus and subsequent updates [9,10] identify five overlapping but clinically distinct syndromes, distinguished by the timing and pattern of BP and HR change after standing. Each has different management implications.

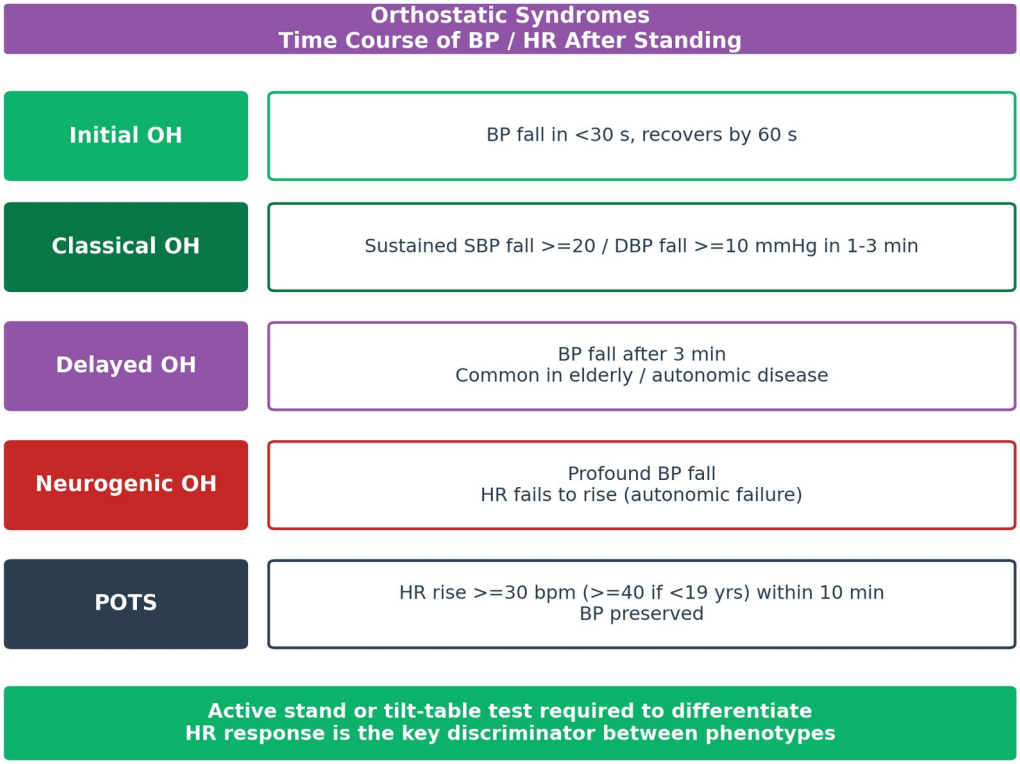

Figure 2. — The orthostatic syndrome spectrum - timing of BP and HR changes after standing, distinguishing initial, classical, delayed, and neurogenic OH from POTS.

Source: Australian Dizziness Clinics - clinical flowchart.

Table 1 - The Five Orthostatic Syndromes Distinguished by Active Stand Test

Syndrome	Definition	Typical age / context	Management priority
Initial OH	SBP fall >40 or DBP fall >20 mmHg within 30 s, recovers by 60 s	Young to middle-aged; rapid risers; dehydration	Reassurance; slow rising; counter-manoeuvres
Classical OH	Sustained SBP fall >=20 / DBP fall >=10 mmHg within 1-3 min	Older adults; medication-related; volume depletion	Medication review; salt and fluid; consider midodrine
Delayed OH	Sustained drop appearing only after 3 min standing	Elderly; early autonomic dysfunction	Often missed; needs full 10 min stand; treat as classical
Neurogenic OH	Profound BP fall with absent or blunted HR rise	Parkinsonism, MSA, diabetic neuropathy, amyloid	Expert input; droxidopa, fludrocortisone
POTS	HR rise >=30 bpm (>=40 if <19 yrs) within 10 min, BP preserved	Adolescents and young women; post-viral or post-COVID	Volume; exercise programme; refer for tilt

Initial and delayed OH are the two phenotypes most commonly missed by a single 1-minute standing measurement; document at 1, 3, 5, and 10 min for any patient with a strongly positive history but a normal 1-min reading.

□ **Clinical Pearl:** Initial OH and POTS are the two phenotypes most often misclassified as "anxiety"

or "functional dizziness" in younger patients. Both are real, measurable, and treatable. Both demand a full 10-minute active stand or tilt-table study to be confidently diagnosed.

IV. Bedside Assessment - Active Stand and the Practical Orthostatic Workup

The active stand test, performed correctly, is the most cost-effective single investigation in the dizzy patient. It takes ten minutes, requires only a sphygmomanometer, and answers the diagnostic question for the great majority of patients.

How to Perform an Active Stand Test

1. Patient supine for 5 minutes in a quiet room. Record baseline BP and HR.
2. Patient stands actively (not assisted to standing) and remains upright with feet uncrossed.
3. Measure BP and HR at 1 minute, 3 minutes, 5 minutes, and 10 minutes standing.
4. Record symptoms (dizziness, blurred vision, nausea, neck ache) at each interval.
5. If the patient feels presyncopal, sit them down immediately - terminate the test.

Several common errors invalidate the test. The most frequent is taking only a 1-minute reading, which misses both initial OH (already recovered) and delayed OH (not yet present). The second is using a passive tilt or having the patient sit on the bed - blunting the gravitational stress. The third is ignoring symptoms that are not accompanied by a >=20/10 mmHg drop; symptomatic orthostatic intolerance with a preserved BP can still be treated as such, especially if HR rises markedly.

□ **Important:** A single normal 1-minute reading does not exclude orthostatic dizziness. Approximately 20-30% of older adults with symptomatic orthostatic intolerance have either initial or delayed OH and will be missed unless readings are taken at multiple time points across 10 minutes [11].

What Else to Examine

- General observation - hydration, peripheral oedema, signs of Parkinsonism, gait initiation.
- Neurological screen - cranial nerves, limb power and reflexes, cerebellar testing, autonomic stigmata (anhidrosis, fixed pupil).
- Cardiac examination - heart rate and rhythm, murmurs (especially AS), JVP, peripheral pulses.
- Bedside vestibular screen - pursuit, saccades, head impulse test, Dix-Hallpike if positional symptoms.
- Cognitive screen - especially in older adults; OH is a common driver of unmasked cognitive impairment.

V. The High-Yield Medication Review

A structured medication review is the single most important therapeutic intervention in orthostatic dizziness. In Australian primary care cohorts, simple deprescribing or substitution of one or more high-risk agents resolves symptoms in 30–40% of patients before any specific OH treatment is started [7,12]. Older patients on five or more medications carry a relative risk of OH of 2–3, and on ten or more, the risk approaches 60% [13].

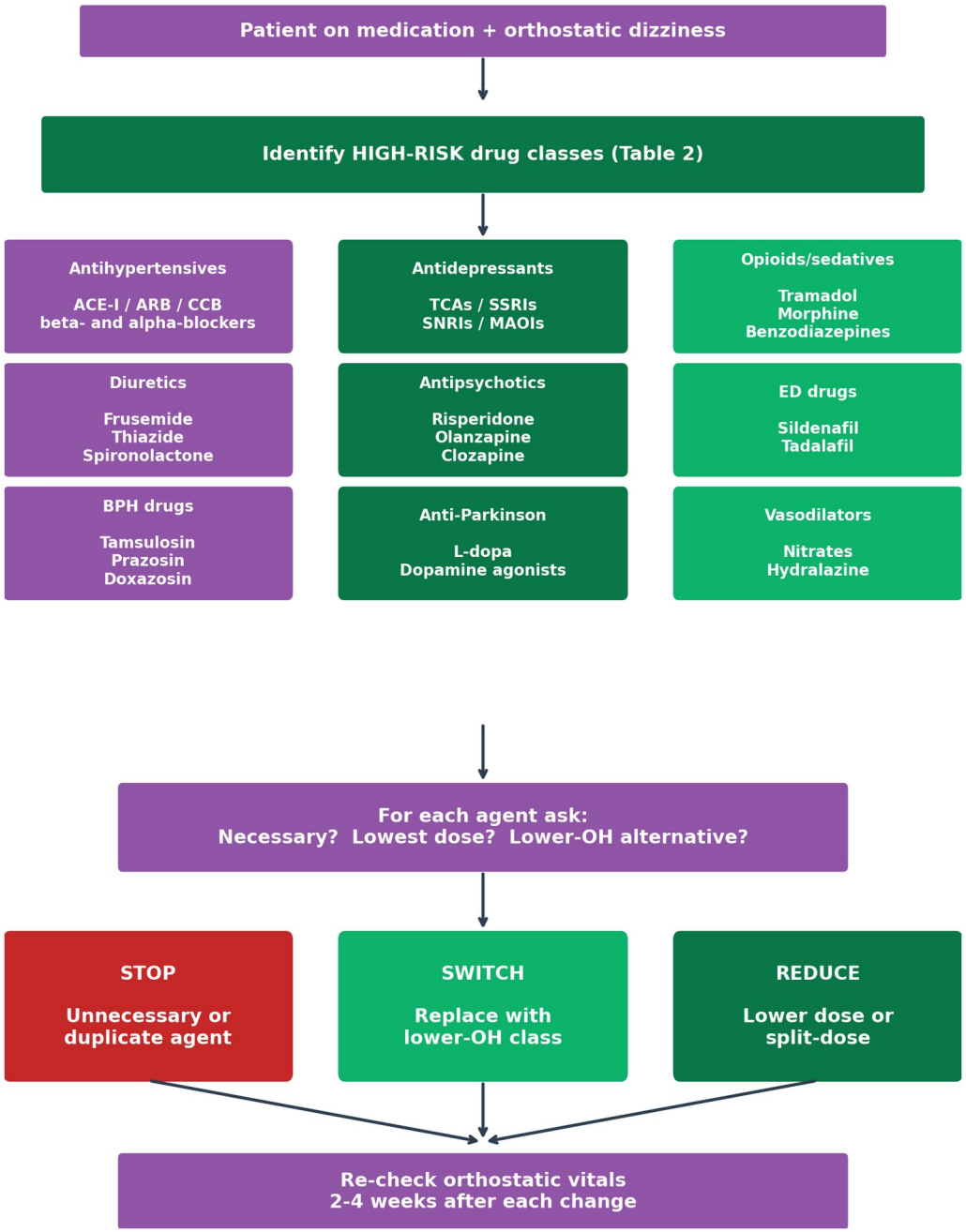

Figure 3. — High-yield medication review - the nine drug classes most likely to cause or worsen orthostatic dizziness, with a structured stop / switch / reduce framework.

Source: Australian Dizziness Clinics - clinical flowchart.

Table 2 - High-Risk Drug Classes for Orthostatic Dizziness and Practical Actions

Drug class	Examples	Mechanism	Practical action
Alpha-blockers	Tamsulosin, prazosin, doxazosin	Block peripheral vasoconstriction	Substitute / nocturnal dose / consider 5-alpha-reductase inhibitor for BPH
ACE-I / ARB	Perindopril, ramipril, telmisartan	Vasodilation; volume reduction	Halve dose; review BP target in elderly (140/90 acceptable)
Diuretics	Frusemide, indapamide, hydrochlorothiazide	Volume depletion	Reduce or stop if no clear indication; check Na, K

Beta-blockers	Metoprolol, atenolol, bisoprolol	Blunt HR response to standing	Substitute (CCB) where indication permits
Calcium channel blockers	Amlodipine, nifedipine	Vasodilation	Often well tolerated; halve dose if drop on standing
Antidepressants	TCAs > SNRIs > SSRIs	Alpha-blockade; volume effects	Switch from TCA to SSRI; SSRIs usually tolerable
Antipsychotics	Risperidone, olanzapine, clozapine	Alpha-1 blockade	Lowest effective dose; falls-risk MDT review
Anti-Parkinson	L-dopa, dopamine agonists	Disease + drug effect	Coordinate with neurology; do not stop abruptly
Opioids / sedatives	Tramadol, morphine, benzodiazepines	Central sympatholysis	Deprescribe where possible
PDE-5 inhibitors / nitrates	Sildenafil, GTN	Vasodilation	Educate on positional changes; avoid combination

Anticholinergic burden score (e.g. ACB) is also a useful heuristic in elderly polypharmacy. Aim to reduce total burden, not just discontinue single agents.

□ **Clinical Pearl:** For every dizzy patient on antihypertensives, it is reasonable to ask: "What is the evidence-based BP target for THIS patient?" In a frail 85-year-old, a target of 140/90 is supported by SPRINT-MIND and HYVET; pursuing 130/80 in this patient drives OH and falls without any cardiovascular benefit [14].

Withdrawal must be gradual where appropriate (beta-blockers, alpha-2 agonists, opioids, antidepressants) and recheck of symptoms and BP at 2–4 weeks is essential.

VI. Investigations - Targeted, Not Comprehensive

Investigation in orthostatic dizziness is targeted, not comprehensive. Most patients fitting a clear pattern of classical OH following a positive medication review need no further workup before initiating treatment. Investigation is directed at three questions: is volume or autonomic function the primary problem, are there contributing comorbidities, and does the pattern suggest cardiac syncope rather than OH?

Table 3 - Targeted Investigations in Orthostatic Dizziness

Investigation	Indication	Yield in primary care
FBC, EUC, glucose, HbA1c, B12, TSH	All patients - first visit	High; identifies anaemia, dehydration, diabetic autonomic risk
Morning cortisol +/- short Synacthen	Postural symptoms with hyperpigmentation, weight loss, hyponatraemia	Adrenal insufficiency - rare but unmissable
ECG +/- ambulatory monitor	Palpitations, syncope, structural heart disease	Detects arrhythmic syncope mimicking OH
Echocardiogram	Murmur, exertional symptoms, suspected AS or HCM	Identifies obstructive cardiac causes
Tilt-table test	Persistent symptoms despite negative active stand; suspected POTS / vasovagal	Definitive in equivocal cases
Autonomic testing (HRV, QSART, Valsalva)	Suspected neurogenic OH; Parkinson plus syndromes	Autonomic clinic only; not first-line

MRI brain has no routine role in OH. Reserve it for atypical neurology, suspected MSA, or progressive Parkinsonism with autonomic features.

□ **Clinical Insight:** A 24-hour ambulatory blood pressure monitor is often more revealing than a tilt-

table test in older patients - particularly in detecting supine hypertension paired with orthostatic hypotension, the classic "non-dipper" or "reverse dipper" pattern of autonomic failure that has prognostic significance.

VII. Management - Non-Pharmacological First

Non-pharmacological measures are the foundation of management and are sufficient in isolation for many patients with mild to moderate OH. They should be initiated in every patient and reinforced before pharmacotherapy is considered [15].

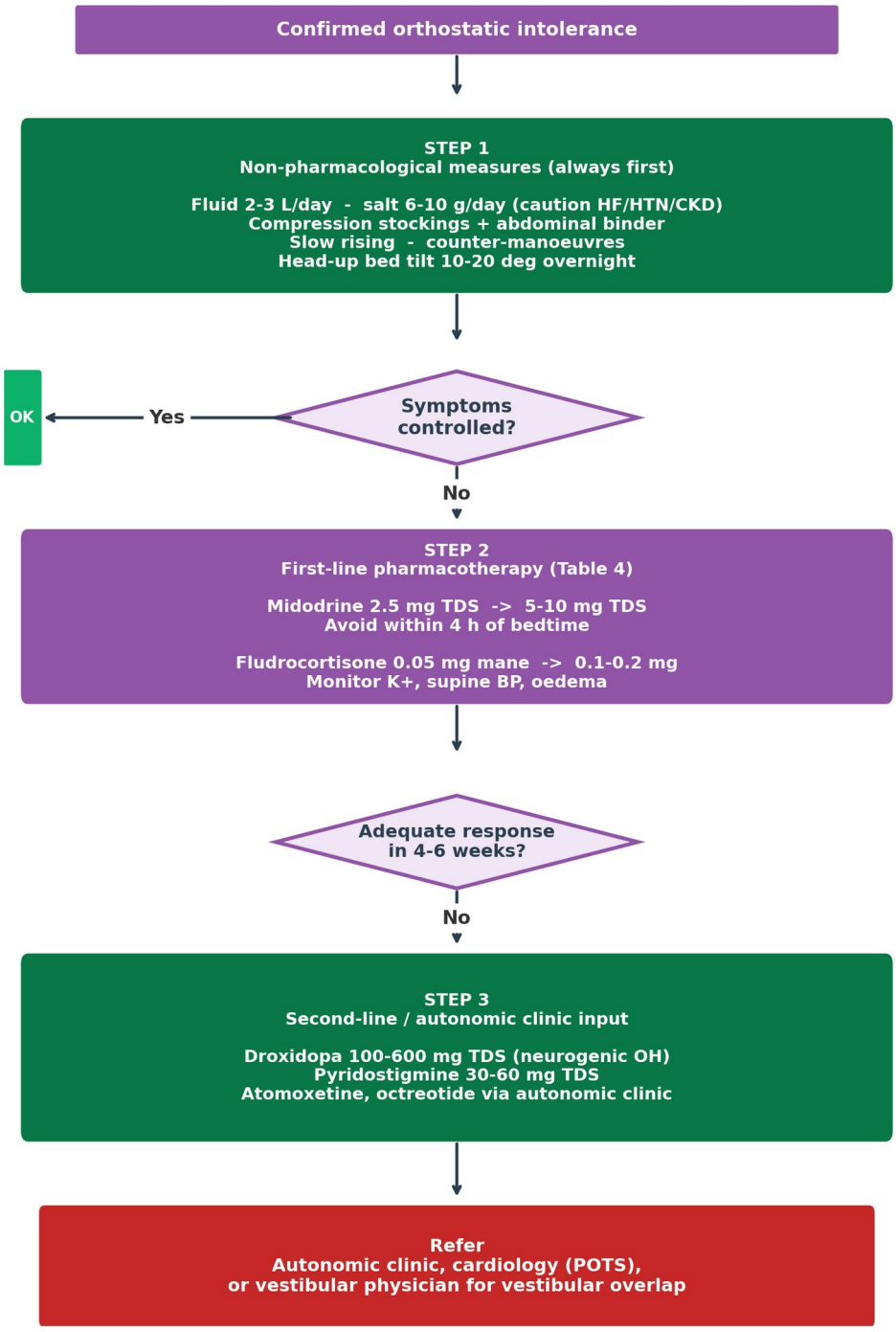

Figure 4. — Stepwise management algorithm for orthostatic intolerance - non-pharmacological first, single-agent pharmacotherapy second, specialised therapy third.
Source: Australian Dizziness Clinics - clinical flowchart.

Lifestyle and Behavioural Measures

- Fluid intake 2-3 litres per day. A rapid 500 mL water bolus before known triggers (e.g. before getting out of bed) acutely raises BP via the osmopressor reflex.
- Salt 6-10 g per day - relative contraindication in severe heart failure or hypertension; otherwise well tolerated.
- Compression - thigh-high or waist-high stockings (>=20-30 mmHg) and an abdominal binder are more effective than knee-highs.
- Slow rising - sit up for 30 seconds before standing; flex calves and abdomen briefly before rising.
- Physical counter-manoeuvres - leg crossing, squatting, toe raises, abdominal contraction during prolonged standing.
- Head-up tilt of the bed at night by 10-20 degrees - reduces nocturnal pressure natriuresis and supine hypertension.
- Avoid hot baths, large meals, alcohol, and prolonged motionless standing.
- Graded exercise - particularly recumbent (cycling, rowing, swimming) builds calf muscle pump capacity.

□ **Important:** Salt and fluid loading must NOT be advised in patients with congestive cardiac failure, severe hypertension, or stage 3-5 chronic kidney disease without autonomic clinic input. A modest fluid increase to 1.5-2 L/day with stockings and counter-manoeuvres is the safer starting point in these patients.

VIII. Pharmacological Therapy When Lifestyle Fails

When non-pharmacological measures are insufficient after 4-6 weeks, single-agent pharmacotherapy is added. Two first-line agents - midodrine and fludrocortisone - have acceptable evidence in classical and neurogenic OH. Specialised second-line agents (droxidopa, pyridostigmine, atomoxetine, octreotide) are usually initiated by an autonomic or vestibular physician.

Table 4 - First and Second-Line Pharmacotherapy for Orthostatic Hypotension

Agent	Starting dose	Target dose	Mechanism	Key cautions
Midodrine	2.5 mg TDS (mane, midday, late afternoon)	5-10 mg TDS	Alpha-1 agonist - peripheral vasoconstriction	Avoid within 4 hours of bedtime; supine hypertension; urinary retention
Fludrocortisone	0.05 mg mane	0.1-0.2 mg mane	Mineralocorticoid - salt and water retention	Hypokalaemia; oedema; supine hypertension; CCF
Droxidopa	100 mg TDS	300-600 mg TDS	Norepinephrine pro-drug - augments sympathetic outflow	Initiated by autonomic clinic; supine hypertension; nausea
Pyridostigmine	30 mg TDS	60 mg TDS	Acetylcholinesterase inhibitor - augments ganglionic transmission	Modest BP rise only; cholinergic side effects
Atomoxetine	10-18 mg mane	18-40 mg mane	NET inhibitor - augments noradrenergic tone	Vestibular physician or autonomic clinic use in neurogenic OH; off-label

Combination therapy (midodrine + fludrocortisone) is often required in moderate to severe disease but raises supine hypertension risk substantially - check supine BP at every review.

□ **Clinical Pearl:** Supine hypertension is the major adverse effect of all OH pharmacotherapy and is

present in over half of patients with neurogenic OH at baseline. Always measure supine BP when titrating; if supine SBP exceeds 160 mmHg, consider head-up sleeping, dose reduction, or evening avoidance of midodrine.

IX. When to Refer

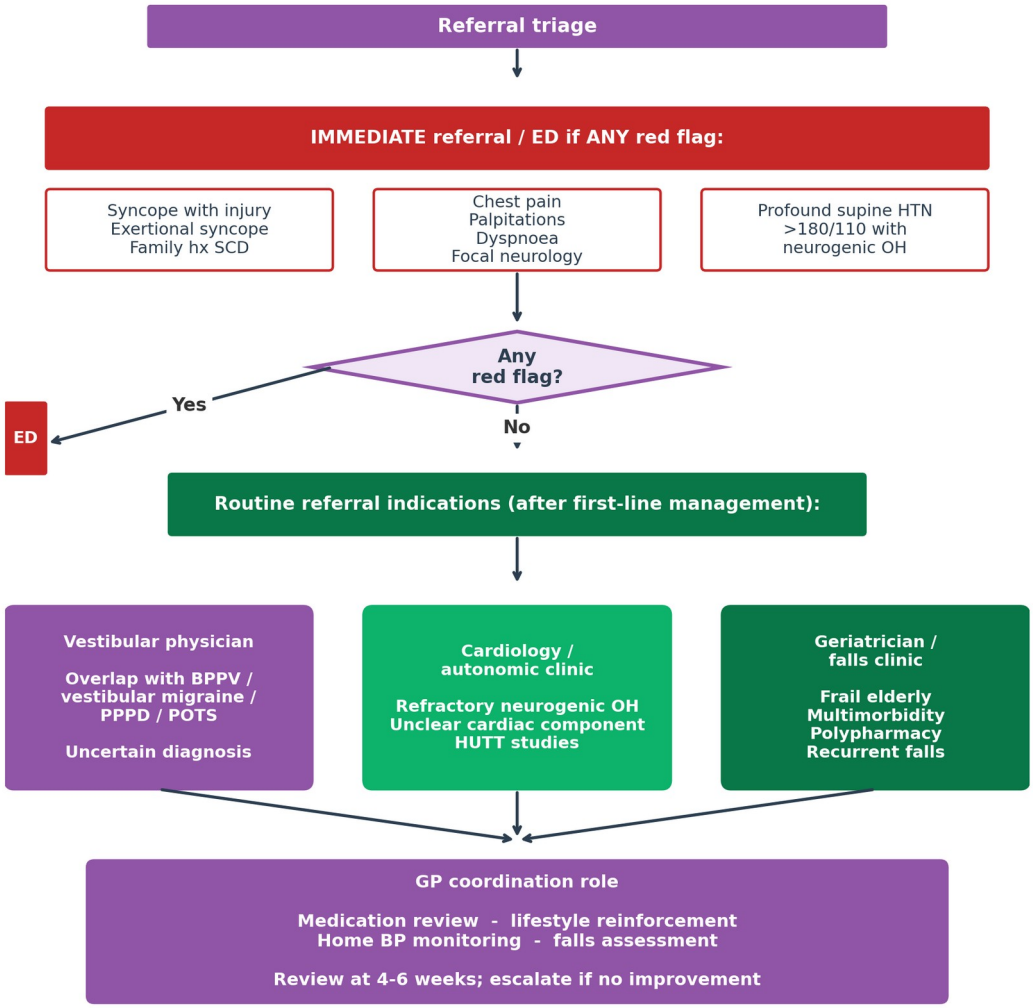

Figure 5. — Referral and red-flag triage for orthostatic dizziness - immediate ED, cardiology, vestibular physician, or geriatric pathways.

Source: Australian Dizziness Clinics - clinical flowchart.

Most patients with orthostatic dizziness can be managed entirely in primary care. Referral is appropriate when red-flag features are present, when first-line management has failed, or when complexity warrants expert input.

Immediate (ED or cardiology) referral

- Syncope with injury, exertional syncope, or family history of sudden cardiac death.
- Chest pain, palpitations, dyspnoea on exertion, or focal neurology accompanying the dizziness.
- Profound supine hypertension >180/110 alongside OH, suggesting marked autonomic failure.
- Suspected arrhythmia or structural cardiac cause on initial assessment.

Routine expert referral

- Vestibular physician - overlap of orthostatic and vestibular features (BPPV, vestibular migraine, PPPD, POTS), uncertain diagnosis, or persistent dizziness with normal active stand and head-up tilt-table test

(HUTT - a passive 60 to 80 degree tilt-table study performed in autonomic or cardiology labs to confirm orthostatic intolerance, POTS, or vasovagal syncope when bedside testing is equivocal).

- Cardiology / autonomic clinic - suspected POTS, refractory neurogenic OH, or patients requiring tilt-table study and autonomic function tests.
- Geriatrician / falls clinic - frail elderly with multimorbidity, polypharmacy, recurrent falls, and complex deprescribing.
- Movement disorders / neurology - suspected multiple system atrophy, Parkinsonian autonomic failure, or pure autonomic failure.

□ **Clinical Insight:** In the patient with overlapping vestibular and orthostatic features, a vestibular physician review is often more efficient than separate cardiology and ENT referrals. A single integrated assessment can clarify which contributor is dominant and whether vestibular rehabilitation, autonomic input, or both are required.

X. Diagnostic and Management Pathway

The pathway below consolidates the diagnostic and management approach for use in a standard GP consultation. It assumes a 10-15 minute encounter and integrates Tables 1-4.

Figure 6. — Integrated diagnostic and management pathway for orthostatic and medication-related dizziness in general practice.

Source: Australian Dizziness Clinics - clinical flowchart.

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