Persistent Postural-Perceptual Dizziness (PPPD):

Recognising and Managing Chronic Functional Dizziness in General Practice

Vestibular Medicine for General Clinicians

Topic 8 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 chronic 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 an 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 PPPD Matters to Every General Clinician

Persistent Postural-Perceptual Dizziness (PPPD) is the most common cause of chronic dizziness in adults under 65 and the second most common cause overall after benign paroxysmal positional vertigo [1,2]. Despite this, it is one of the most under-recognised vestibular diagnoses in primary care. Population-based studies from neuro-otology clinics suggest PPPD accounts for 15–25% of all referrals, yet fewer than one in five patients arrive with a suspected diagnosis [3,4].

The consequence for patients is substantial. Mean time from symptom onset to diagnosis exceeds four years in Australian and international cohorts [5]. Patients commonly undergo repeated neuroimaging, cardiac investigations, and empirical treatment with vestibular suppressants — all of which can worsen the condition. Many are ultimately labelled as having "functional", "psychogenic", or "anxiety-related" dizziness, which is both inaccurate and therapeutically unhelpful.

□ **Key Point:** PPPD is a positive diagnosis defined by Bárány Society consensus criteria — not a diagnosis of exclusion and not a synonym for anxiety. It is present in up to one in five patients presenting with chronic dizziness, and its recognition changes management fundamentally.

PPPD was formally defined by the Bárány Society Committee for the Classification of Vestibular Disorders in 2017, unifying four previously described conditions: phobic postural vertigo, space and motion discomfort, visual vertigo, and chronic subjective dizziness [6]. The ICD-11 now lists PPPD as a discrete disorder (AB32.0).

For GPs and allied health professionals, the practical question is not whether to manage PPPD single-handedly, but whether to recognise the pattern and initiate the correct care pathway — vestibular rehabilitation, selective serotonergic medication, and cognitive-behavioural support — rather than perpetuate a cycle of investigation and suppressive therapy.

II. Pathophysiology — A Functional Sensorimotor Disorder

PPPD is understood as a disorder of central vestibular processing rather than a structural inner-ear or brain disease. After an acute vestibular or balance-destabilising event, the central nervous system fails to reset its postural control strategies, leaving the patient locked in a high-risk, high-vigilance mode of balance [7]. Three interacting mechanisms have been consistently demonstrated:

- Stiffened, high-risk postural control. Patients use ankle-strategy co-contraction and reduced trunk mobility as if walking on ice, even on flat ground [8].
- Visual dependence. Reliance on vision for spatial orientation increases, so visually complex or moving environments provoke symptoms [9].
- Heightened threat processing. Functional imaging shows increased insular and amygdala activation with attenuated vestibular cortical engagement — a pattern of hypervigilance to motion [10].

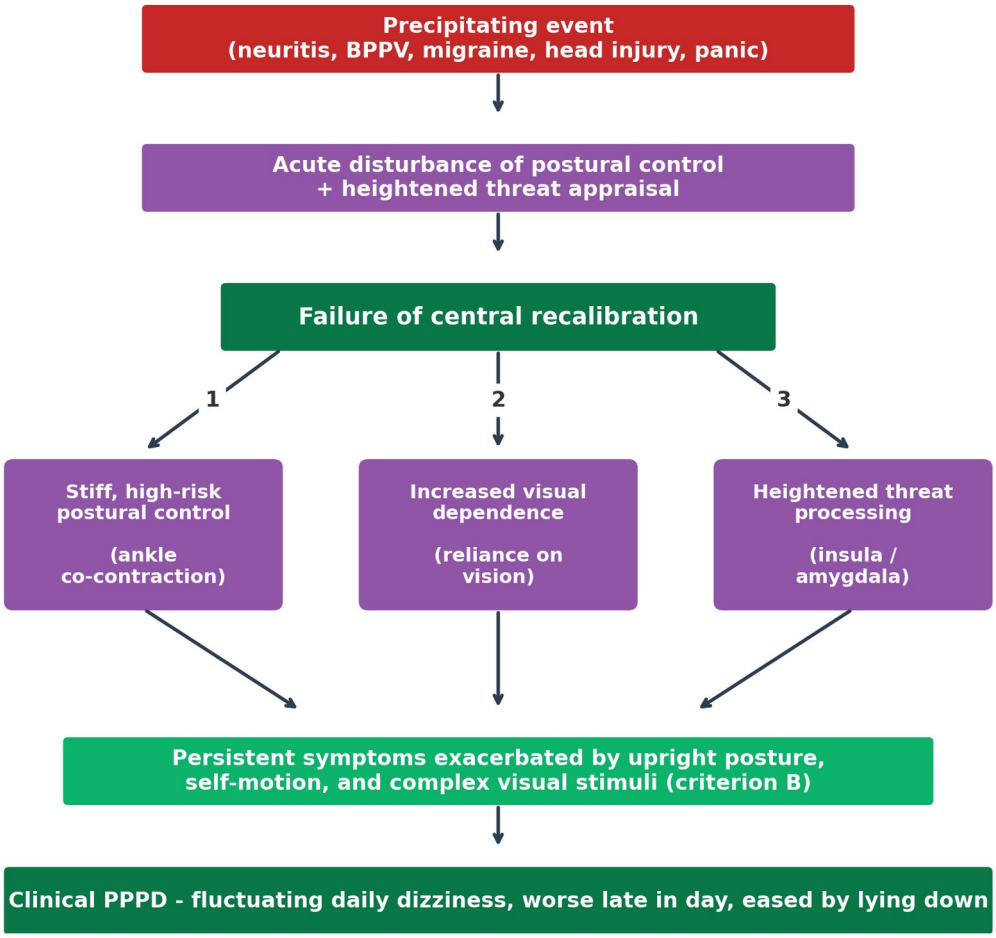

Figure 1. — PPPD pathophysiology — the maladaptive sensorimotor loop initiated by a precipitating event.
Source: Australian Dizziness Clinics — clinical flowchart.

□ **Clinical Insight:** PPPD is not "in the head" in a dismissive sense. It is a real, measurable reorganisation of sensorimotor control. Framing it for patients as a software rather than hardware problem — the inner ear and brain are structurally intact but the balance program has been miscalibrated — is both accurate and therapeutically useful.

Most cases (around 70–80%) are triggered by a precipitating event: vestibular neuritis, BPPV, vestibular migraine attack, head injury, panic attack with vertigo, or medical illness causing transient imbalance [11]. In a minority, no discrete trigger is identifiable. Premorbid anxiety traits and neuroticism are risk factors but are neither necessary nor sufficient for the diagnosis.

III. The Bárány 2017 Diagnostic Criteria

Diagnosis rests on the five criteria below, all of which must be met for at least three months [6]. These criteria are deliberately positive — they describe what PPPD looks like, not merely what it is not.

Table 1 — Bárány Society 2017 Diagnostic Criteria for PPPD

Criterion	Requirement
A	One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo present on most days for ≥3 months (symptoms typically last hours but may wax and wane).

B	Persistent symptoms occur without specific provocation but are exacerbated by three factors: (1) upright posture, (2) active or passive motion, and (3) exposure to moving or complex visual stimuli.
C	The disorder is precipitated by an event that caused vertigo, unsteadiness, dizziness, or balance problems — including peripheral or central vestibular disorders, medical illness, or psychological distress.
D	Symptoms cause significant distress or functional impairment.
E	Symptoms are not better accounted for by another disorder.

All five criteria A–E must be fulfilled. Note that criterion E does not require exhaustive exclusion — it requires that another disorder does not better explain the presentation.

□ **Clinical Pearl:** The three exacerbators in criterion B — upright posture, self-motion, and visual stimuli — are the single most reliable clinical fingerprint of PPPD. Asking about all three in every patient with chronic dizziness takes under a minute and is highly diagnostic.

A common misconception is that PPPD cannot coexist with another vestibular disorder. In fact, it frequently overlays residual vestibular migraine, treated BPPV, or compensated vestibular neuritis [12]. Treating the comorbidity alone rarely relieves the chronic dizziness; the PPPD must be addressed in parallel.

IV. Clinical Presentation and History Cues

The typical PPPD patient describes non-spinning dizziness, unsteadiness, or a rocking/swaying sensation that is present "almost every day" for months. The language patients use is remarkably consistent and worth learning:

- "Like walking on a boat, or on a trampoline."
- "Head feels disconnected from the body."
- "Floor moves when I am still."
- "Supermarket aisles and computer screens make me worse."
- "Worse the longer I am on my feet; better when I sit or lie down."
- "Started after a bout of labyrinthitis / a bad migraine / a car accident months ago, and it has never fully settled."

Symptoms characteristically fluctuate through the day, typically worst in the late afternoon and evening after prolonged upright time, and ease with rest. They are rarely present on first waking. True rotational vertigo is absent in PPPD itself, although it may have featured in the triggering event.

Differentiating PPPD from Its Mimics

Table 2 — Distinguishing PPPD from Common Chronic Dizziness Differentials

Feature	PPPD	Vestibular migraine	Bilateral vestibular loss	Generalised anxiety
Duration per episode	Continuous, daily	Minutes to 72 hours	Continuous, worse with movement	Variable
Visual trigger	Prominent	Present	Absent or mild	Absent
Postural trigger	Worse upright	Variable	Worse with any motion	Unrelated
Oscillopsia walking	Absent	Absent	Present (bouncing vision)	Absent
Better when lying	Yes	Variable	Yes	No consistent pattern

Bilateral vestibular loss is the most important structural mimic to exclude; oscillopsia on walking is the key discriminator and mandates formal vestibular function testing.

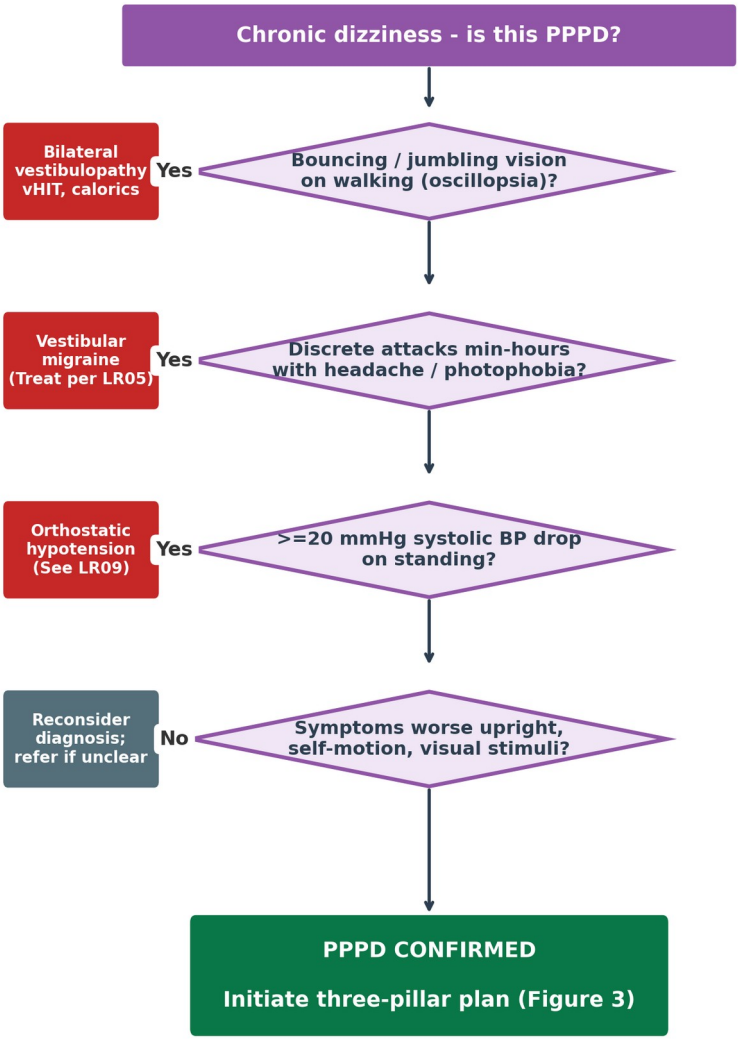

Figure 2. — A four-step decision tree for distinguishing PPPD from its commonest chronic-dizziness mimics in primary care.
Source: Australian Dizziness Clinics — clinical flowchart.

□ **Important:** A patient reporting bouncing or jumbling vision while walking (oscillopsia) does NOT have PPPD. This suggests bilateral vestibular loss and requires formal vestibular function testing before a PPPD label is applied.

V. Bedside Examination

Examination in PPPD is most useful for what it does not show. There are no PPPD-specific signs, but the absence of significant abnormalities supports the diagnosis.

What to Check

- Spontaneous and gaze-evoked nystagmus. Should be absent. Any persistent nystagmus argues for another diagnosis.
- Head impulse test. Should be normal. A unilateral catch-up saccade suggests uncompensated peripheral loss; bilateral abnormality suggests bilateral vestibulopathy.
- Dix-Hallpike and supine roll test. Should be negative. Coexistent BPPV must be treated regardless of a PPPD diagnosis.
- Romberg, tandem stance, tandem gait. Usually normal or show functional inconsistency (worse when observed, better when distracted).
- Orthostatic vitals. A drop in systolic BP ≥20 mmHg on standing points to orthostatic dizziness rather than PPPD.
- Cranial nerves and cerebellar examination. Normal.

□ **Clinical Insight:** A patient with months of non-spinning dizziness, normal ear and neurological examination, no nystagmus, negative Dix-Hallpike and normal orthostatics has PPPD as a front-running diagnosis. Further imaging and testing are rarely required before initiating treatment.

Functional inconsistency during stance and gait testing — marked sway when observed that disappears with a distracting task (for example, counting backwards) — is common in PPPD and not evidence of feigning. It reflects the heightened self-monitoring characteristic of the condition [13].

VI. Investigations — Resisting the Urge to Over-Test

By the time PPPD is considered, most patients have already had MRI brain, audiometry, routine blood tests, and often cardiac investigation. Repeating these rarely changes management. The role of further investigation is to identify coexisting or alternative diagnoses suggested by the history and examination — not to "rule out" PPPD, which is a positive diagnosis.

Reasonable investigations in a patient fitting the Bárány criteria:

- Audiometry — if not already performed, to exclude Ménière's disease or vestibular schwannoma.
- MRI brain with internal auditory meati — only if neurological examination is abnormal, headaches are prominent, or hearing is asymmetric.
- Formal vestibular function testing (vHIT, calorics, VEMPs) — where bilateral vestibular loss is suspected (oscillopsia, poor balance in the dark) or where prior vestibular diagnosis is unclear.
- Orthostatic vitals — mandatory in every patient with chronic dizziness, especially those over 65 or on antihypertensives.

□ **Clinical Pearl:** Do NOT order repeat MRI, Holter monitors, or tilt-table studies on patients who clearly fit PPPD criteria in the hope of "finding something". Each additional negative test delays treatment and reinforces the patient's belief that something has been missed, which worsens the condition.

Standard laboratory testing (full blood count, electrolytes, thyroid, vitamin B12, glucose) is reasonable once and rarely needs repeating unless clinically indicated.

VII. Management — The Three-Pillar Approach

Evidence-based PPPD management rests on three pillars deployed in combination. Monotherapy is substantially less effective than combined treatment, which achieves meaningful symptom reduction in 60–80% of patients [14,15].

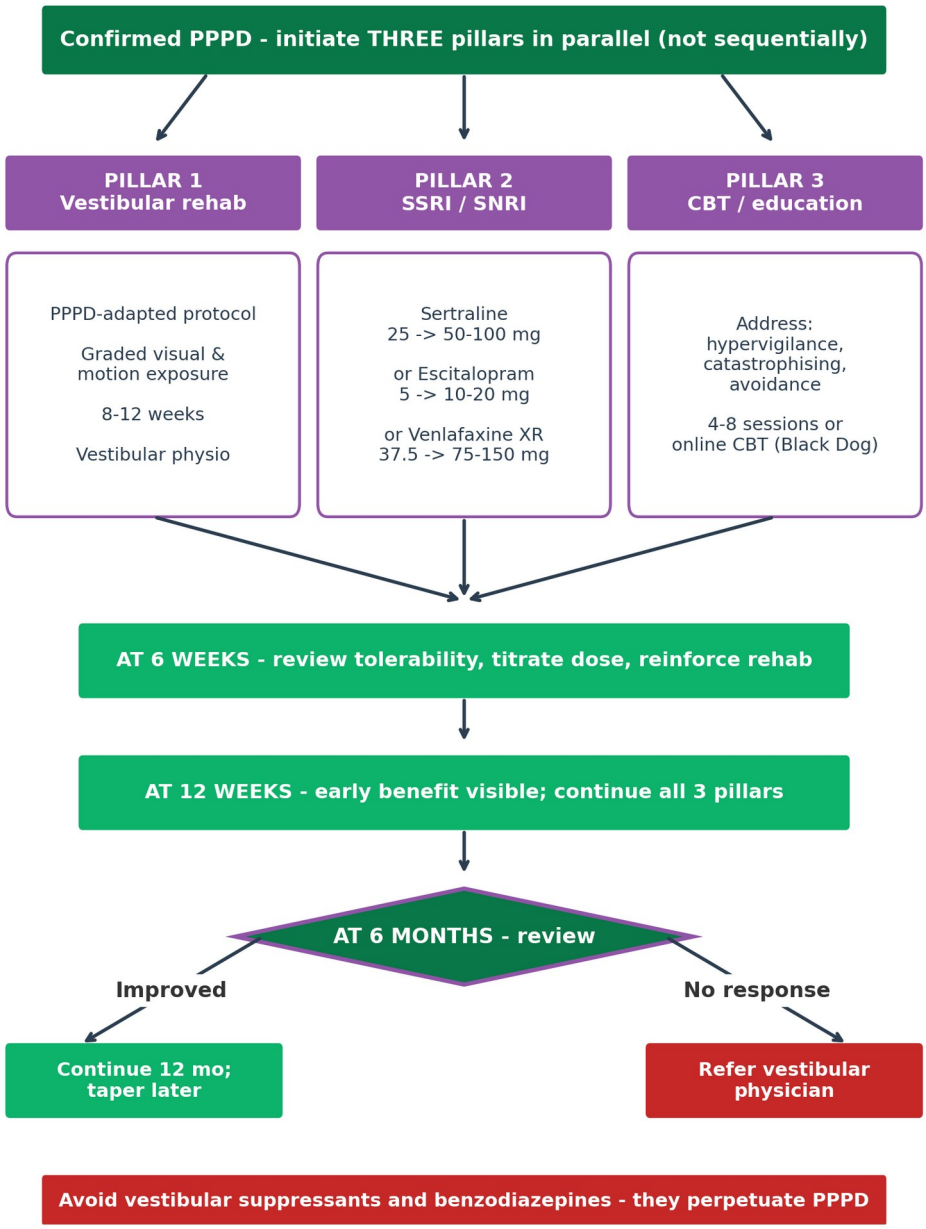

Figure 3. — The three-pillar treatment algorithm — vestibular rehabilitation, serotonergic pharmacotherapy, and CBT initiated in parallel with structured review at 6 weeks, 12 weeks, and 6 months.

Source: Australian Dizziness Clinics — clinical flowchart.

Pillar 1 — Vestibular Rehabilitation

A PPPD-adapted program of graded exposure is the cornerstone of treatment. Standard peripheral vestibular rehabilitation protocols must be modified: progression is slower, visual motion exposure is deliberate and graded, and symptom provocation is used therapeutically rather than avoided [16]. Typical duration is 8–12 weeks with a trained vestibular physiotherapist.

Pillar 2 — SSRI or SNRI Pharmacotherapy

Serotonergic antidepressants are effective in PPPD regardless of whether overt anxiety or depression is present [17]. Response rates of 60–70% are reported. Starting doses must be low to avoid early destabilisation. Counsel patients that initial side effects (transient increase in dizziness, nausea, sleep disturbance) typically settle within 1–2 weeks and that benefit takes 6–12 weeks to emerge. A minimum 6-month trial at therapeutic dose is recommended before considering the medication ineffective.

Table 3 — First-Line SSRI / SNRI Dosing Schedule for PPPD

Agent	Starting dose	Target dose	Titration	When to choose
Sertraline (SSRI)	25 mg mane	50–100 mg mane	↑ 25 mg every 1–2 weeks	First-line in Australian practice; well tolerated
Escitalopram (SSRI)	5 mg mane	10–20 mg mane	↑ 5 mg after 2 weeks	Alternative if sertraline poorly tolerated
Venlafaxine XR (SNRI)	37.5 mg mane	75–150 mg mane	↑ 37.5 mg every 1–2 weeks	Useful where vestibular migraine coexists
Fluoxetine (SSRI)	10 mg mane	20–40 mg mane	↑ 10 mg every 2 weeks	Long half-life — gentler discontinuation

Initial flare in dizziness, nausea, or sleep disturbance is expected for 1–2 weeks; counsel patients in advance and persist. Minimum 6-month trial at therapeutic dose before declaring failure.

Pillar 3 — Cognitive-Behavioural Therapy

CBT addresses the hypervigilance, catastrophisation, and avoidance behaviours that maintain PPPD. Even brief CBT (4–8 sessions) delivered by a psychologist familiar with chronic illness produces meaningful benefit [18]. Patient self-help resources and online CBT programs (such as those via the Australian Black Dog Institute) are reasonable adjuncts where formal therapy is not accessible.

□ **Important:** Vestibular suppressants (prochlorperazine, betahistine, cinnarizine, benzodiazepines) should NOT be used in PPPD. They blunt central adaptation, perpetuate symptoms, and expose the patient to dependence, falls, and sedation. Deprescribing existing suppressant therapy is often the single most impactful first step.

VIII. Prognosis, Pitfalls, and Patient Communication

With structured three-pillar treatment, 60–80% of patients achieve substantial symptom improvement and 30–50% achieve remission within 6–12 months [14,15]. Untreated, PPPD tends to persist for years with fluctuating severity. Relapse can be triggered by further vestibular events, significant stress, or life changes, and is generally responsive to resumption of treatment.

Common Management Pitfalls

- Labelling the condition as "anxiety" or "functional dizziness" without offering the specific PPPD treatment pathway.
- Prescribing ongoing vestibular suppressants or benzodiazepines.
- Recommending avoidance of triggers rather than graded exposure.
- Stopping SSRI/SNRI treatment early because of initial symptom flare.
- Failing to treat coexisting BPPV, vestibular migraine, or orthostatic hypotension.
- Delaying vestibular rehabilitation while awaiting further investigations.

Framing the Diagnosis for the Patient

The explanation offered to the patient materially affects outcome. A useful framing acknowledges that the original precipitating event was real, that the current dizziness is a consequence of a brain recalibration problem rather than ongoing damage, and that specific, evidence-based treatment exists. Avoid language implying the condition is psychological, imagined, or less real than structural disease.

□ **Key Point:** The explanation patients find most useful: "Your inner ears and brain are structurally fine, but after what you went through, your balance system is running in high-alert mode — like a car alarm that keeps going off. We have a three-part plan — exercises, medication, and a way of thinking about it — that re-teaches the system to switch off."

IX. When to Refer

Most patients with PPPD can be managed in general practice with allied health input. Referral to a vestibular physician or dedicated dizziness service is appropriate where the diagnosis is uncertain, where standard management has failed, or where complexity warrants comprehensive assessment.

- Diagnostic uncertainty — particularly if oscillopsia, progressive hearing loss, or central features are present.
- Failed first-line treatment — no meaningful improvement after 3–6 months of combined rehabilitation and pharmacotherapy.
- Complex comorbidity — coexisting vestibular migraine, Ménière's disease, bilateral vestibular loss, or significant mental health illness.
- Intolerance of SSRI/SNRI requiring alternative pharmacotherapy.
- Patients requesting expert confirmation of diagnosis before committing to treatment.

□ **Clinical Insight:** A confident primary-care PPPD diagnosis with early initiation of rehabilitation and an SSRI, followed by expert review if needed, produces better outcomes than referral-and-wait. Every month of delay is a month of maladaptive postural learning.

X. PPPD Diagnostic and Management Pathway

The following pathway consolidates the diagnostic and management approach suitable for use in a standard GP consultation. Use it alongside the Bárány criteria (Table 1), the differentiating features (Table 2), and the SSRI/SNRI dosing guide (Table 3).

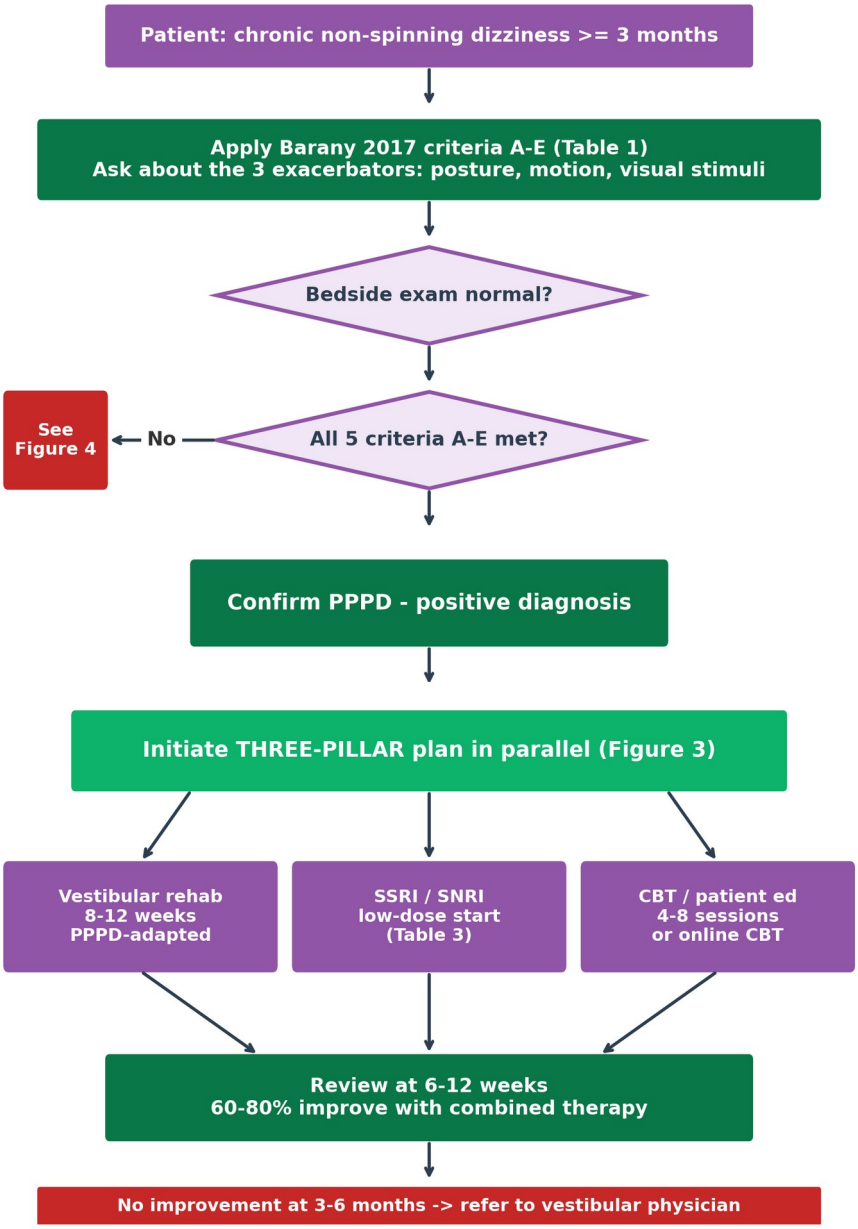

Figure 4. — PPPD recognition and management pathway for general clinicians — from pattern recognition through three-pillar treatment to review and onward referral.

Source: Australian Dizziness Clinics — clinical flowchart.

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