Persistent Postural-Perceptual Dizziness (PPPD) — Cheat Sheet for Vestibular Physicians

The commonest cause of chronic vestibular symptoms in working-age adults. A positive clinical diagnosis — normal tests support it, they do not exclude it.

► Why PPPD matters

The most common cause of chronic vestibular symptoms in working-age adults and the single most frequent diagnosis in many tertiary dizziness clinics (~15–25% of all diagnoses). A chronic functional vestibular disorder codified by the Bárány Society in 2017 (ICD-11 AB32.0); it unifies and replaces phobic postural vertigo, visual vertigo and chronic subjective dizziness. The balance hardware works — the control software stays locked in a high-alert, vision-dependent mode that no longer matches the patient's actual risk. It is a positive diagnosis, not a diagnosis of exclusion and not a synonym for anxiety.

► When to suspect PPPD

► *Near-constant dizziness, unsteadiness, swaying or rocking — often described as being on a boat — on most days for ≥ 3 months, rather than discrete spinning vertigo.*
► *Symptoms build through the day: relative ease on waking, worse by afternoon/evening as sensory and cognitive demands accumulate.*
► *An identifiable precipitant — peripheral vestibular event, vestibular migraine, concussion, dysautonomia, medical illness or a panic attack — even if remote and forgotten.*
► *Normal or only well-compensated examination, with years of normal investigations: "every test has been normal but I am still dizzy every day."*

► The three exacerbating factors — the defining triad

Exacerbating factor	What patients describe	Bedside / history pointer
Upright posture	Worse on standing or walking; eases on lying down	"Are you better lying down than standing or walking?" — a high-yield screen
Active or passive motion	Own movements and passive transport (car, escalator, lift) aggravate, regardless of direction	Symptoms provoked by motion in any direction, not a single position
Moving or complex visual stimuli	Supermarkets, scrolling screens, patterned floors, traffic provoke a spike	"Do busy or moving visual environments make you worse?" — visual dependence made visible

Pearl — Two screening questions capture the syndrome with high yield — the supermarket/screen question and the lying-down question. Consistent "yes" answers should raise PPPD strongly.

► Diagnosis — Bárány Society criteria (all five A–E required)

Criterion	Requirement
A. Persistent symptoms	Dizziness, unsteadiness or non-spinning vertigo on most days for ≥ 3 months; prolonged (hours) but fluctuating.
B. Provoking factors	Present without specific provocation but exacerbated by upright posture, active/passive motion, and moving or complex visual stimuli.
C. Precipitant	Triggered by a condition causing vertigo, unsteadiness or balance problems — vestibular, neurological, medical or psychological (acute, episodic or chronic).
D. Distress or impairment	Symptoms cause significant distress or functional impairment.
E. Not better explained	Symptoms are not better accounted for by another active disease or disorder.

Pearl — Criterion C is the one most often missed — patients and prior clinicians forget or normalise the original trigger. A timeline linking a remote vestibular event, concussion or period of intense anxiety to the onset of daily symptoms frequently unlocks the diagnosis. Criterion E permits PPPD alongside a compensated neuritis or controlled migraine.

► Bedside examination and diagnostic approach

Domain	Expected in PPPD	Interpretation
Neuro / vestibular exam	Normal, or only an old well-compensated deficit	Does not explain the ongoing daily symptoms
Spontaneous / positional nystagmus	Absent	Its presence points away from PPPD — seek active disease
Gait	Cautious, co-contracted, "walking on ice"; improves with distraction, worse with self-observation	The distraction paradox is a useful bedside pointer to a functional mechanism
True spinning vertigo / vomiting	Absent	If present, look for BPPV, Ménière's or vestibular migraine (which may coexist and trigger PPPD)

Pearl — When a patient says "every test has been normal but I am still dizzy every day," do not reach for another scan — reach for the PPPD criteria. Normal investigations in the right symptom pattern support, rather than undermine, the diagnosis.

► Targeted investigations — proportionate, not reflexive

Investigation	Purpose	When to order
Audiometry	Exclude Ménière's and other cochleovestibular pathology	Most patients, especially with any aural symptoms
vHIT, VNG with calorics	Detect or characterise a peripheral deficit (usually old and compensated)	Most patients, to confirm no active loss
VEMP	Assess otolith pathways; supports characterisation	Selected patients where otolith disease is considered
MRI brain	Exclude central or structural disease	Red flags, atypical course or focal signs only
Orthostatic / autonomic testing	Identify orthostatic intolerance contributing to symptoms	Lightheadedness on standing or syncope in the history

Pearl — The battery is typically normal or shows only a stable, compensated unilateral deficit. Over-investigation is anti-therapeutic — reflexive serial imaging delays treatment, escalates health anxiety and entrenches the threat appraisal that drives the disorder.

► **Red flags — these are NOT PPPD: new spontaneous or positional nystagmus, fresh focal neurological signs, acute hearing loss, or a first severe ("worst-ever") headache. Their presence demands a search for active vestibular or central pathology — image and/or refer before attributing symptoms to PPPD.**

► Key differentials — and what distinguishes them

Condition	Distinguishing feature from PPPD
Vestibular migraine	Discrete episodes of vertigo with migrainous features and headache; PPPD is continuous and non-episodic — though the two frequently coexist (the most important companion).
BPPV	Brief positional spinning vertigo with diagnostic positional nystagmus; PPPD lacks true vertigo and positional nystagmus.
Ménière's disease	Episodic vertigo with fluctuating low-frequency hearing loss, tinnitus and aural fullness; PPPD has no cochlear progression.
Uncompensated vestibular hypofunction	Active deficit on vHIT/calorics with directional symptoms; PPPD shows compensated or normal testing.
Orthostatic intolerance (OH / POTS)	Symptoms tied to standing with measurable BP or heart-rate change; PPPD is provoked by motion and visual load too.

Pearl — Coexistence is the rule, not the exception. A patient can have well-controlled Ménière's, a compensated neuritis or active vestibular migraine and still meet PPPD criteria — treat both layers rather than forcing a single label.

► Management — coordinated, multimodal stepped care

Tier	Intervention	Practice principles
1 · Education	Name the diagnosis; validate symptoms as real; frame as a reversible fault in balance control, not a hidden catastrophe	Therapeutic in itself and the indispensable first step — directly counteracts the threat appraisal; improves engagement with active therapy
2 · Rehabilitation + CBT	Graded habituation and motion/visual desensitisation (VRT) plus disorder-specific CBT for catastrophising, hypervigilance and avoidance	Pace carefully — too aggressive a start flares symptoms; best delivered by a PPPD-familiar physiotherapist; CBT most effective early
3 · Pharmacotherapy	SSRI (sertraline, paroxetine, escitalopram, fluoxetine) or SNRI (venlafaxine, duloxetine) for the vestibular-limbic network	Start low, titrate slowly (patients are activation-sensitive); 8–12 week trial before judging; ~60–70% respond; alongside — not instead of — VRT and CBT
4 · Refractory	Reconfirm diagnosis and audit delivery; switch/combine antidepressants; intensify MDT; treat coexisting migraine and anxiety	Genuine resistance is far less common than incomplete or uncoordinated treatment; neuromodulation remains experimental

Pearl — Before labelling PPPD refractory, confirm the diagnosis and the dose: an under-dosed or short antidepressant trial, mis-paced rehabilitation, or an untreated coexisting vestibular migraine accounts for most apparent treatment failures. Avoid maintenance vestibular suppressants — they impede central adaptation.

► Prognosis, relapse and the three-P framing

► **With coordinated treatment the prognosis is good — most patients achieve substantial symptom reduction and recover function; spontaneous remission is uncommon, so confident early diagnosis beats watchful waiting.**

► **Three-P framing: predisposing traits (anxious/introspective personality, visual dependence) + a precipitating event + perpetuating mechanisms (avoidance, hypervigilance, deconditioning). You cannot change the first two — treatment dismantles the third.**

► **Favourable: short symptom duration before diagnosis, early coordinated multimodal treatment, resilient coping. Unfavourable: high trait anxiety, entrenched avoidance, untreated comorbid migraine, medication non-adherence.**

► **Relapse follows a new vestibular insult, high stress or intercurrent illness — discharge every patient with a relapse-prevention plan: maintained habituation, retained cognitive strategies, and a low threshold for early re-engagement.**

Key references — Staab et al. (Bárány criteria). J Vestib Res 2017 · Popkirov, Staab & Stone. Pract Neurol 2018 · Dieterich & Staab. Curr Opin Neurol 2017 · Axer et al. Brain Behav 2020 · Trinidade et al. JNNP 2023.