Visual-Induced Dizziness:

A Clinical Approach to Visual Vertigo and Visual Dependence

Vestibular Medicine for General Clinicians

Topic 13 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 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 one-page 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. The Clinical Problem

Visual-induced dizziness (VID) is one of the most under-recognised dizziness presentations in primary care. Patients describe being made dizzy by looking — by walking down a supermarket aisle, by scrolling on a phone, by busy patterned floors, by crowds, or by sitting as a passenger on a highway. Between episodes the bedside neurological and vestibular examination is typically normal, and routine blood tests and imaging are unrevealing [1,2]. The mismatch between dramatic functional impact and unremarkable examination leads many patients to be reassured prematurely or referred for repeated structural investigation.

VID is best understood not as a single disease but as a sensitive cross-sectional marker of an underlying vestibular vulnerability. It dominates the clinical picture of persistent postural-perceptual dizziness (PPPD), is highly prevalent in vestibular migraine and mal-de-débarquement syndrome, and persists in roughly one third of patients in the months following acute peripheral vestibulopathy or concussion [3–5]. Recognising VID is therefore the entry point to a small set of treatable diagnoses that account for a substantial proportion of chronic dizziness in general practice.

□ **Key Point:** A patient who reports being made dizzy by visual environments — supermarkets, scrolling, patterned floors, crowds, highway driving — almost always has an underlying vestibular condition. Reassurance alone is rarely sufficient; targeted assessment and habituation rehabilitation usually help.

The functional impact is substantial. Patients reduce or stop driving, avoid shops and social venues, struggle with screen-based work, and frequently reduce paid employment. Many present after months of symptoms, having seen multiple clinicians and accumulated normal test results [4]. A structured five-feature trigger inventory and a single validated questionnaire — the Visual Vertigo Analogue Scale (VVAS) — allow VID to be confidently identified and triaged in a standard primary-care appointment.

II. Pathophysiology — Visual-Vestibular Mismatch and Visual Dependence

Spatial orientation depends on continuous integration of three sensory streams: vestibular (canals and otoliths), visual (especially peripheral optic flow), and somatosensory (proprioception, cutaneous pressure). Integration occurs in a network centred on the parieto-insular vestibular cortex (PIVC), the medial superior temporal area (MST), and posterior parietal cortex, with the cerebellum acting as a sensory recalibration hub [6,7]. When one stream becomes unreliable — for example after a vestibular insult — the brain reweights the remaining streams to maintain orientation. In most patients this reweighting is brief and corrects as vestibular function recovers.

In a substantial minority, sensory reweighting becomes maladaptive. The patient becomes persistently over-reliant on vision, a state termed visual dependence [6,8]. Once visual dependence is established, normal real-world visual environments — particularly those rich in optic flow or repetitive geometry — generate a sustained mismatch between the visual stream the brain is over-weighting and the vestibular and proprioceptive streams it is under-weighting. The conscious correlate of this mismatch is dizziness on looking. The mechanism cascade is summarised in Figure 1.

Visual Dependence as Trait and State

- Trait visual dependence: identified in healthy adults using the Rod-and-Disc test; predicts severity of dizziness after vestibular insult.
- State visual dependence: increases acutely after vestibular neuritis, concussion, or a vestibular migraine flare; normalises in many but persists in PPPD.
- Cortical correlate: increased blood-oxygen-level-dependent activity in MST and PIVC during optokinetic stimulation in visually dependent patients.
- Modifiable: graded optokinetic exposure (visual habituation) reduces visual dependence and dizziness — the central rationale for VID rehabilitation.

□ **Clinical Insight:** Visual dependence is the best-validated mechanism of visual-induced dizziness and is directly modifiable by rehabilitation. Bedside testing of dynamic visual acuity and Romberg with

eyes-closed-on-foam screens for it without dedicated equipment.

Visual-vestibular mismatch is therefore the unifying mechanism, and visual dependence is the physiological state that maintains it. Brief optokinetic stimulation in clinic reproduces the symptom and can serve as a powerful patient education tool.

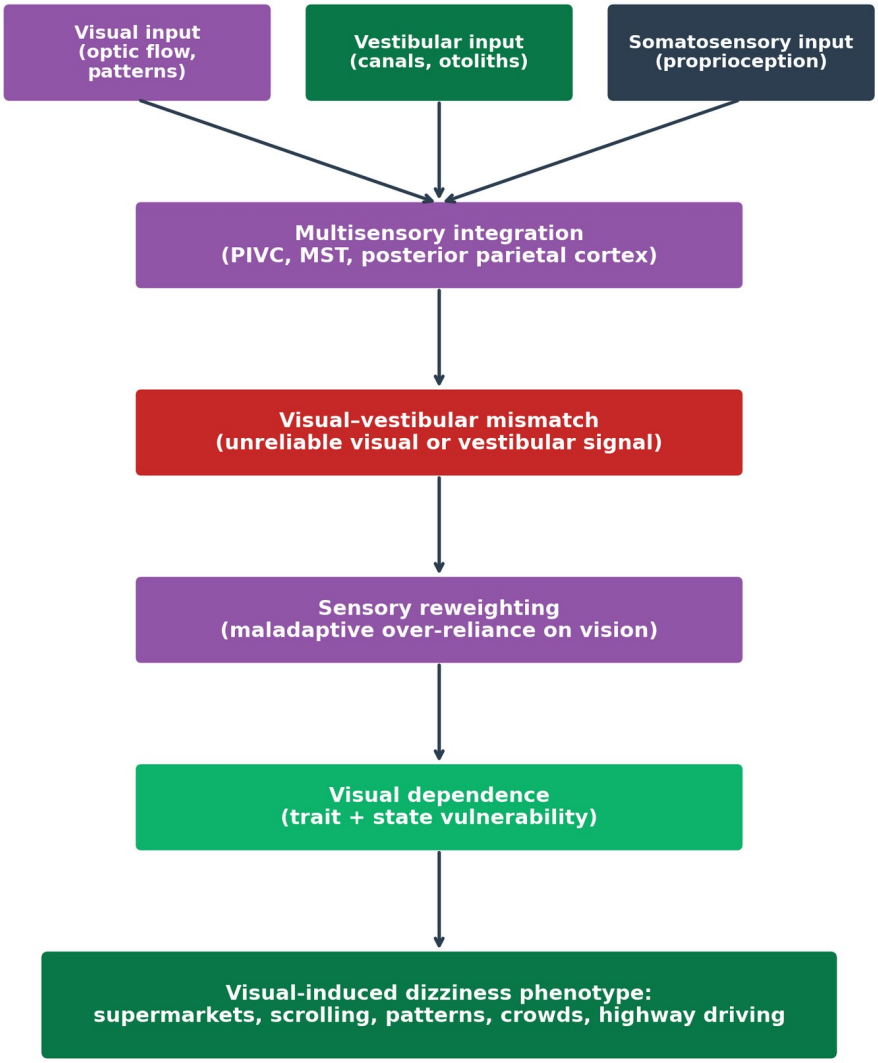

Figure 1. Mechanism of visual-induced dizziness — sensory mismatch, reweighting, visual dependence, and resulting clinical phenotype.

Source: Australian Dizziness Clinics — clinical flowchart.

III. Terminology and Definitions

Several overlapping terms are used in the literature, often interchangeably and sometimes incorrectly. The Bárány Society Classification of Vestibular Disorders and the International Classification of Vestibular Disorders give working definitions that should anchor primary care language [10,11].

Table 1. Working definitions of visual-induced dizziness and related terms (Bárány Society Classification of Vestibular Disorders).

Term	Working definition
Visually-induced dizziness (VID)	Dizziness or unsteadiness provoked or worsened by visual environments — recommended Bárány Society umbrella term.

Term	Working definition
Visual vertigo	Older term, often used synonymously with VID; preserved in the questionnaire name "Visual Vertigo Analogue Scale".
Visual dependence	Trait or state in which orientation relies disproportionately on visual cues; the underlying mechanism of VID.
Space-and-motion discomfort	Anxiety-related discomfort in visually rich, low-support environments (heights, supermarkets); overlaps with VID.
PPPD criterion	Bárány PPPD criteria require symptoms to be exacerbated by upright posture, motion, and complex visual stimuli — the third element being VID.

In primary care it is acceptable, and often clearer for the patient, to use the lay phrase "dizziness brought on by busy visual environments". In notes and referrals, the recommended precise term is visually-induced dizziness with a parenthetical note of the dominant underlying diagnosis (e.g. "VID with PPPD overlay" or "VID secondary to vestibular migraine").

Why Distinguish PPPD from VID?

PPPD is a defined syndrome — chronic dizziness lasting three or more months, exacerbated by upright posture, motion, and complex visual stimuli, often triggered by a precipitating vestibular event [3]. VID is a symptom that is present in PPPD but also occurs in vestibular migraine, mal-de-débarquement syndrome, post-concussive syndromes, and partially compensated peripheral vestibulopathies. Conflating them risks under-treating the broader condition or over-diagnosing PPPD before three months have elapsed.

A Note on Older Terms

- "Phobic postural vertigo" and "chronic subjective dizziness" are historical precursors to PPPD and should no longer be used in new documentation.
- "Visual vertigo" remains in clinical use, particularly in questionnaire names, but the recommended term in new documentation is visually-induced dizziness.
- "Motion sensitivity" describes a related but broader phenotype that includes susceptibility to passive whole-body motion (cars, boats), with substantial overlap with VID.
- Use of the term "psychogenic dizziness" is discouraged — it underplays the physiological mechanism and risks therapeutic nihilism.

□ **Clinical Pearl:** When writing referrals, name both the symptom (VID) and the suspected underlying syndrome (PPPD, vestibular migraine, post-vestibular insult, post-concussive). This clarifies the rehabilitation target for the receiving service.

IV. Conditions Where Visual-Induced Dizziness Dominates

A handful of conditions account for the majority of patients presenting with prominent VID. Recognising the typical clinical pattern of each allows the generalist to assign a working diagnosis and a treatment plan at the first visit.

Table 2. Conditions in which visual-induced dizziness is a dominant feature.

Condition	Typical pattern	First-line action
PPPD	Daily non-spinning dizziness ≥3 months; worse upright, on motion and with visual stimuli; often follows a vestibular event.	Educate; visual habituation; consider SSRI/SNRI; refer to vestibular physician if severe.
Vestibular migraine	Episodic vertigo with migrainous features; interictal VID and motion sensitivity common; family history of migraine.	Lifestyle and trigger management; migraine prophylaxis; treat VID with habituation.
Mal-de-	Persistent rocking or swaying after	Reassurance; vestibular

Condition	Typical pattern	First-line action
débarquement (MdDS)	travel; ameliorated by passive motion; female predominance.	rehabilitation; refer for MdDS-specific protocols if persistent.
Post-vestibular insult	VID emerging in weeks following vestibular neuritis, labyrinthitis, or treated BPPV; visual dependence persists.	Reinforce vestibular rehabilitation with explicit visual-habituation component.
Post-concussive	VID after mild traumatic brain injury; often combined with cervicogenic and oculomotor features.	Multidisciplinary concussion programme; vestibular and visual rehabilitation.
Anxiety / SMD	VID in visually rich, low-support environments (atria, supermarkets); panic features prominent.	CBT; treat any coexisting vestibular condition; SSRI if indicated.

PPPD and vestibular migraine together account for the large majority of chronic VID seen in primary care [3,12]. In any patient with new VID, a careful migraine history is mandatory — untreated migraine reliably maintains VID and undermines rehabilitation.

□ **Important:** New visual-induced dizziness in a patient over 50 with vascular risk factors, particularly if accompanied by oscillopsia or headache, requires consideration of a central cause and urgent imaging. VID is rarely the presentation of stroke but central vestibular lesions can produce similar symptoms.

V. The Targeted History

The history should establish three things: that the patient has VID rather than spinning vertigo or pre-syncope; which underlying condition is most likely; and what functional impact VID has on driving and work. The single most efficient screening tool is the five-feature visual-trigger inventory shown in Figure 2.

The Five-Feature Visual-Trigger Inventory

Ask the patient explicitly about each of the five environments listed in Figure 2: supermarket aisles, crowds and markets, scrolling and screens, patterned floors and carpets, and highway or passenger driving. Three or more positive features have a high positive predictive value for VID, particularly when paired with a normal interictal examination [13].

□ **Clinical Pearl:** The single best screening question is "Are you made dizzy by busy visual environments — supermarkets, scrolling, busy patterns, crowds, or being a passenger?" A clear yes warrants a VVAS and a structured assessment.

VI. Bedside Assessment and the Visual Vertigo Analogue Scale

The interictal examination in VID is normal, which is itself diagnostically useful — it excludes most central and many peripheral vestibular causes. The clinician should still perform a structured screen and supplement it with deliberate provocation manoeuvres and the VVAS.

The Visual Vertigo Analogue Scale

The VVAS is a validated nine-item self-report questionnaire developed by Dannenbaum and colleagues [13]. Each item asks the patient to rate, on a 0–10 visual-analogue scale, the dizziness provoked by a specific visual environment (supermarket, busy shopping centre, screen-based action, patterned floor, escalator with eyes open, watching cars pass, busy street, looking up or down, and being a vehicle passenger). Item scores are summed for a total of 0–90. A total score above 15 is clinically significant and above 40 indicates severe disability. Score thresholds and recommended actions are summarised in Figure 3.

Bedside Provocation Manoeuvres

When equipment is available, brief optokinetic stimulation (full-field stripes or scrolling pattern) reliably provokes symptoms in VID and gives the patient a tangible demonstration of the mechanism. Without optokinetic equipment, a smartphone scrolling through a busy webpage for 30 seconds is a reasonable surrogate.

Examination Screen

A focused examination should include: smooth pursuit and saccades (often normal); head impulse test (normal or only mildly abnormal in long-standing post-vestibular VID); Romberg with eyes open and closed and Romberg on foam (instability with eyes closed-on-foam supports visual dependence); gait with head turns; and dynamic visual acuity if a Snellen chart is available. Cranial nerves and limb examination are normal. Any abnormality requires re-evaluation for an alternative or additional diagnosis.

Driving and Work Impact

Document driving status explicitly: many patients have stopped driving without telling their GP. Specifically ask about highway driving, driving through tunnels, lane changes, and being a passenger. Work impact (screen time, fluorescent-lit environments, supermarket or warehouse work) should be recorded — it informs rehabilitation goals and certification.

□ **Clinical Insight:** Use the VVAS at every visit. A pre- and post-rehabilitation score is the simplest objective measure of treatment response, and a falling score reassures the patient that intervention is working. Print copies are freely available — keep them in the consult room.

VII. Differential Diagnosis

Distinguishing the Common Conditions

The differential of chronic non-spinning dizziness with VID is narrow once history and examination are taken seriously. The main conditions are listed in Table 3 with the discriminating features that allow them to be separated at the bedside.

Table 3. Differential diagnosis of chronic dizziness with prominent visual-induced dizziness.

Condition	Key discriminating features
PPPD	Daily symptoms ≥3 months; precipitating vestibular event in 60–80%; VVAS often >40; normal interictal examination.
Vestibular migraine	Episodic vertigo with migrainous features (headache, photo/phonophobia, aura); family or personal migraine history; interictal motion sensitivity.
MdDS	Persistent rocking sensation after motion exposure; characteristically improves with passive re-motion; female predominance.
Post-vestibular insult	Clear preceding event (neuritis, labyrinthitis, BPPV); subjective recovery incomplete; head impulse may remain mildly abnormal.
Anxiety / SMD	Prominent panic features; symptoms strictly bound to specific environments; CBT alone may resolve.
Cervical / post-traumatic	Recent neck trauma or whiplash; reproducible by neck rotation; cervicogenic headache; oculomotor abnormalities common.
Central vestibular	Persistent abnormal pursuit or saccades; vertical or direction-changing nystagmus; gait or limb ataxia — refer urgently.

Two practical rules help in primary care. First, dizziness that is strictly episodic with discrete attacks lasting minutes to hours is migraine until proven otherwise; dizziness that is daily and constant is PPPD until proven otherwise. Second, any patient with persistent oscillopsia, vertical or direction-changing nystagmus, or new gait ataxia warrants central workup regardless of how unequivocal the visual triggers seem.

□ **Clinical Insight:** PPPD and vestibular migraine commonly coexist. Treat both: trigger and prophylactic management for migraine, and structured visual habituation for the dizziness. Address only one and the patient is unlikely to improve substantially.

Differentiating from Pre-Syncope

Pre-syncope is a distinct symptom — the sensation of impending faint, often with visual greying, sweating, and nausea. It is reproducible by orthostatic challenge, not by visual environments. If the trigger of the patient's dizziness is standing rather than looking, consider orthostatic hypotension and reflex syncope rather than VID.

Differentiating from Vertigo

True rotational vertigo — the room or the patient is spinning — is an attack-based symptom of BPPV, vestibular migraine, Ménière's disease, or vestibular neuritis. Spinning is not a feature of VID. A patient who consistently uses the word "spinning" is unlikely to have isolated VID.

VIII. Investigations

Investigation in primary care is light. The diagnosis is almost always clinical, and excessive investigation reinforces the patient's perception of an undiagnosed structural problem and undermines rehabilitation. Investigations should be targeted to specific clinical questions.

Audiometry

Pure tone audiometry should be requested only if hearing symptoms are present. A normal audiogram supports a non-otologic VID. New asymmetric sensorineural loss requires ENT referral and consideration of MRI.

- Vestibular function testing (vHIT, VEMPs, calorics): not routine; request when the underlying diagnosis is uncertain or when planning vestibular physician input.
- Imaging: MRI brain only when central red flags are present (vertical nystagmus, persistent oscillopsia, focal signs) or when symptoms are progressive in an older patient.
- Bloods: no routine panel diagnoses VID; targeted testing (thyroid, B12, ferritin) is reasonable in fatigue-dominant presentations.
- VVAS: this is the practical baseline measurement — repeat it at 6, 12, and 24 weeks to track response.

□ **Clinical Pearl:** Avoid open-ended "rule everything out" investigation pathways. They reinforce illness identity, generate incidental findings, and delay the only intervention proven to help — visual habituation rehabilitation.

When to Order vHIT and VEMPs

Video head impulse testing and vestibular-evoked myogenic potentials are requested through the vestibular physician or audiology service, not routinely from primary care. They are most useful when the clinical picture is mixed — new VID in a patient with historical vestibular events, suspected bilateral vestibulopathy, or pre-rehabilitation characterisation in severe cases.

□ **Clinical Insight:** A normal vHIT does not exclude VID — it simply confirms an intact peripheral vestibular response. The pathology of VID lies in central reweighting, which vHIT does not measure.

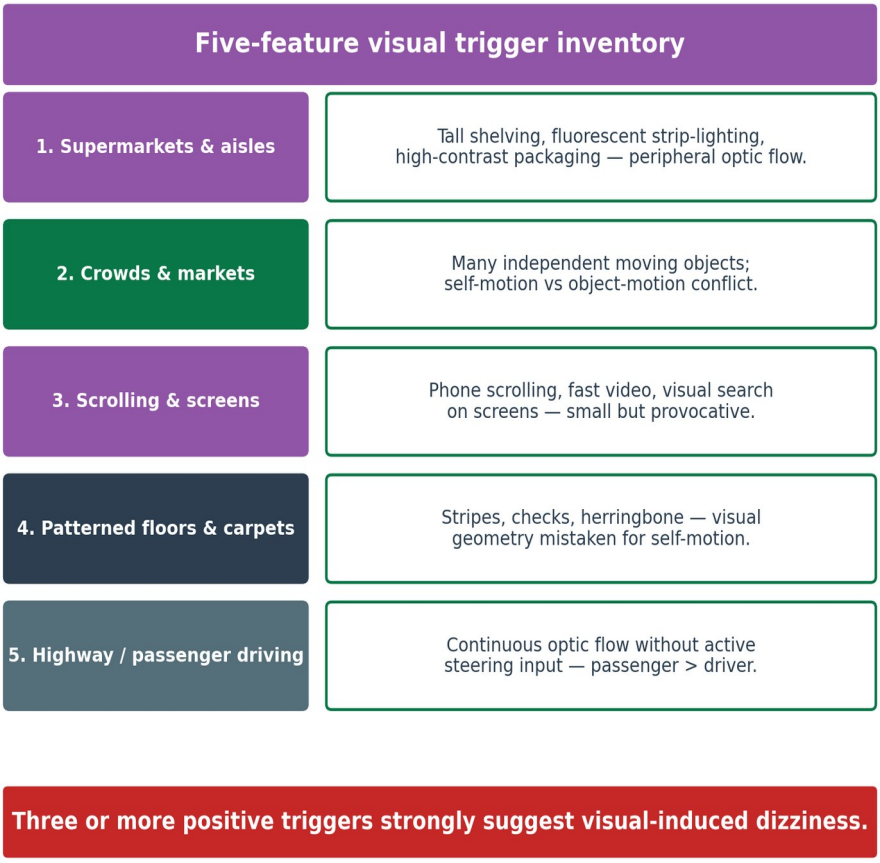

Figure 2. Five-feature visual trigger inventory — the five environments to ask about explicitly when VID is suspected.

Source: Australian Dizziness Clinics — clinical flowchart.

IX. Management — Visual Habituation, Pharmacotherapy, and Lifestyle

Management of VID rests on three pillars: structured visual habituation, treatment of any underlying condition (vestibular migraine, anxiety, residual peripheral deficit), and pharmacotherapy where indicated. Driving and work scripts complete the package.

Visual Habituation Rehabilitation

- Phase 1 — Preparation: education, expectation setting, baseline VVAS, identification of underlying conditions to treat in parallel.
- Phase 2 — Optokinetic provocation: 1–2 minute sessions with stripes, rotating dot fields, or scrolling video, twice daily; symptom intensity should reach 2–3/10 only.
- Phase 3 — Real-world graded exposure: progress through quiet supermarket aisles, busy aisles, patterned carpets, escalators, and finally highway passenger driving.
- Phase 4 — Maintenance: weekly self-graded exposure, three-monthly VVAS, prompt resumption of rehabilitation after any flare. The ladder is shown in Figure 4.
- Pavlou and colleagues have shown that structured optokinetic habituation reduces VVAS scores by approximately 50% over 12 weeks compared with generic vestibular rehabilitation [14,15].
- VR-based and at-home app-based habituation regimens are an emerging adjunct and are particularly useful where in-person rehabilitation access is limited [16].

Pharmacotherapy and Adjuncts

- SSRIs and SNRIs: first-line pharmacotherapy where PPPD is the dominant condition; sertraline 25–50 mg/day or venlafaxine 37.5–75 mg/day are commonly used; effect builds over 8–12 weeks [3,17].
- Migraine prophylaxis: when vestibular migraine is contributing, treat per migraine guidelines — propranolol, amitriptyline, topiramate, or candesartan are reasonable starting agents.
- Vestibular suppressants: avoid. Prochlorperazine, betahistine, and benzodiazepines maintain visual dependence by blunting central recalibration and are the dominant modifiable factor in poor recovery [3,15].
- CBT: a useful adjunct, particularly when anxiety, avoidance, or panic is prominent [20].

□ **Key Point:** Three rules for VID management: (1) name the underlying condition and treat it in parallel; (2) prescribe structured visual habituation, not generic vestibular rehabilitation; (3) avoid vestibular suppressants and benzodiazepines, which prolong symptoms.

X. Clinical Decision Pathway and Referral

Most patients with VID can be successfully managed in primary care. Clear gates for referral, captured in the VVAS and the underlying diagnosis, prevent delay where dedicated input is needed.

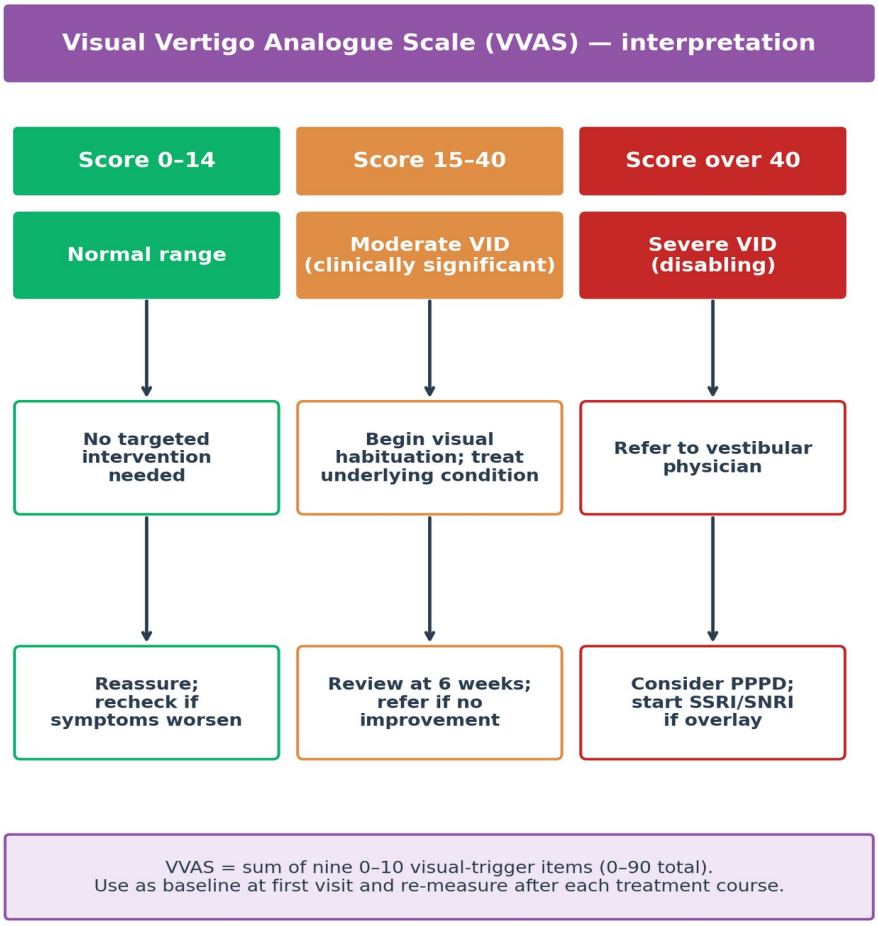

Figure 3. Visual Vertigo Analogue Scale interpretation — three score bands and recommended primary-care actions.

Source: Australian Dizziness Clinics — clinical algorithm.

□ **Clinical Insight:** Treat VID like an exposure-based intervention: short, frequent, slightly uncomfortable doses. Patients who say "I felt worse the first week of habituation" are usually doing it correctly — reassure and continue the regimen.

Figure 4. Visual habituation graded progression ladder — four phases from preparation to maintenance.
Source: Australian Dizziness Clinics — clinical flowchart.

When to Refer to a Vestibular Physician

Indications for Referral

- VVAS above 15 with no improvement after six weeks of structured rehabilitation.
- Suspected PPPD (≥3 months daily symptoms with VID dominance) requiring confirmation and pharmacotherapy planning.
- Vestibular migraine with prominent VID not responding to standard prophylaxis.
- Mal-de-débarquement syndrome persisting beyond three months.
- Failure of habituation rehabilitation despite adherence at 12 weeks.
- Coexisting hearing loss requiring combined audiological and vestibular workup.
- Diagnostic uncertainty or atypical features.
- Patient request for second opinion or for vestibular function testing.

Red Flags Requiring Urgent Imaging or Vestibular Physician Review

- Vertical nystagmus, direction-changing horizontal nystagmus, or skew deviation.
- New persistent oscillopsia (the world appears to bounce with head movement).
- Any focal neurological sign — diplopia, dysarthria, limb weakness or sensory change.
- Gait or limb ataxia disproportionate to the dizziness.
- New severe headache or progressive headache with dizziness.

- Asymmetric sensorineural hearing loss developing alongside VID.
- Vascular risk factors plus new dizziness in a patient over 50 with abnormal central signs.

□ **Clinical Insight:** A patient labelled "anxiety-related dizziness" who improves with structured visual habituation almost always had VID with secondary anxiety — not the reverse. Rehabilitation is the test of mechanism.

□ **Important:** Two messages to emphasise at every review: (1) avoidance of visual environments worsens VID in the long term — graded re-engagement is the cure; (2) symptom severity often spikes transiently when rehabilitation begins — this is expected and the regimen should continue.

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