

# **Visually Induced Dizziness (VID): A Vestibular Physician's Deep Review of Pathophysiology, Assessment, and Rehabilitation**

## **Vestibular Medicine for Vestibular Physicians**

Multisensory Vestibular Disorders — Module 4.2

Australian Dizziness Clinics | [www.AustralianDizzinessClinics.com](http://www.AustralianDizzinessClinics.com)

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## How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of multisensory vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30-40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

## 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, atypical presentations, and critical safety points requiring escalation or imaging.

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# I. Introduction, Definitions, and Epidemiology

Visually induced dizziness (VID) — also referred to in the literature as visual vertigo, visual dependency syndrome, space and motion discomfort (SMD), and visuospatial sensitivity — describes a clinical syndrome in which exposure to complex, moving, or optic-flow-rich visual environments triggers dizziness, unsteadiness, or spatial disorientation that is not attributable to primary oculomotor dysfunction [1,2,11]. The cardinal feature is a disproportionate reliance on visual input for spatial orientation, such that environments that would be unremarkable to a healthy individual — a busy supermarket, a scrolling screen, heavy traffic, or a cinema — become destabilising [1,2,3,43].

The terminology in this field has evolved substantially over four decades, and the absence of a single internationally agreed label remains a source of clinical and research confusion [12,42]. Bronstein's 'visual vertigo' (1995) emphasised the postural and symptomatic response to optically complex environments in patients with known vestibular disorders [1]. Bronstein and Golding's 'space and motion discomfort' (SMD) described a broader spectrum including those without identifiable peripheral vestibular pathology [13]. The Barany Society's International Classification of Vestibular Disorders (ICVD) now recognises visuospatial sensitivity as a cardinal feature of persistent postural-perceptual dizziness (PPPD) but does not define VID as a standalone diagnostic category [9,12,42]. For clinical practice, 'visually induced dizziness' is the preferred working label, encompassing both isolated visuospatial hypersensitivity and the visual-trigger phenotype within PPPD [4,41].

**Table 1. Terminology for visually induced dizziness across the literature.**

Term	Author(s) / Year	Context / Scope
Visual vertigo	Bronstein, 1995 [1]	Dizziness in optically complex environments; patients with known vestibular disorders
Space and motion discomfort (SMD)	Bronstein & Golding, 2013 [13]	Broader term; includes patients without peripheral vestibular diagnosis
Visual vestibular mismatch	Longridge & Mallinson, 2005 [14]	Conflict-based framing; often post-peripheral-disorder rehabilitation context
Visual dependency syndrome	Cousins et al., 2014 [3]	Quantified as percentage of visual weighting on posturography
Visuospatial sensitivity	Barany Society / Staab, 2017 [9]	Criterion C of PPPD — not a standalone category in ICVD
Visually induced dizziness (VID)	Dannenbaum et al., 2011 [4]	Operationally defined; measured by the VVAS — the preferred clinical label

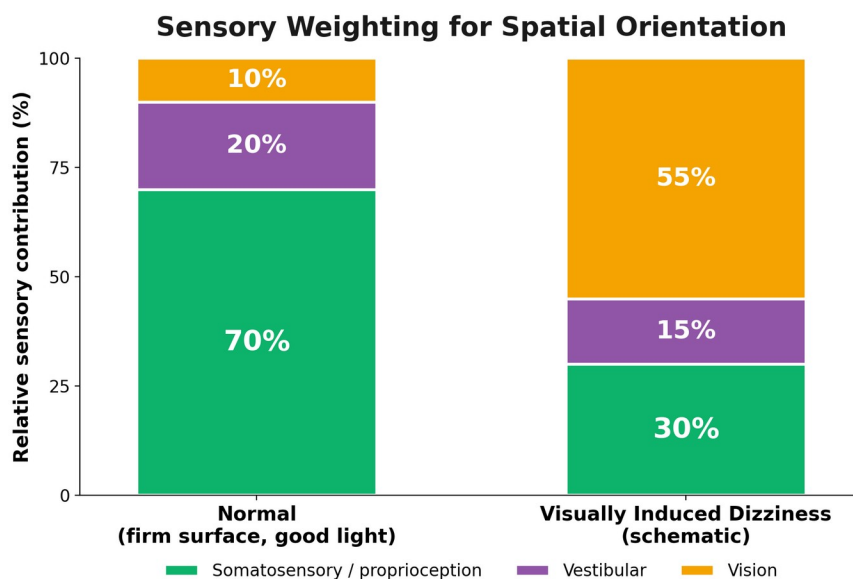
The epidemiology of VID is best understood within dizzy clinic populations. Cousins et al. (2017) quantified visual dependency in 255 consecutive patients presenting to a specialist neuro-otology clinic and found that 57% met criteria for clinically significant visual dependency [41]. Murdin and Schilder's systematic review of the community epidemiology of balance disorders found that self-reported dizziness provoked by visual environments was present in approximately 15-20% of adults who screened positive for chronic dizziness [20]. The prevalence is substantially higher in patients with prior peripheral vestibular disorders — particularly those with incomplete vestibular compensation — and in patients with anxiety disorders [3,19,27].

Female sex, pre-existing anxiety or panic disorder, migraine history, and a prior episode of acute vestibular neuritis or labyrinthitis are the most consistently identified risk factors across clinic-based series [3,19,26,27]. Onset is most common in the third to fifth decade. The condition can arise de novo (primary VID, without a preceding vestibular event) or as a post-vestibular complication (secondary VID), and this distinction carries prognostic and therapeutic implications [3,19,32]. A significant proportion of patients referred with 'chronic dizziness' or 'refractory vestibular disorder' have VID as the dominant — and often treatable — phenotype [2,39,43].

□ **Key Point:** VID is present in over half of patients presenting to specialist vestibular services. It is frequently overlooked because routine vestibular function testing is often normal — the diagnosis requires a history specifically directed at visual triggers and a validated measurement instrument such as the VVAS.

## II. Pathophysiology — Visual-Vestibular Weighting and Sensory Mismatch

Spatial orientation under normal conditions is achieved through the integration of three sensory streams: vestibular, visual, and somatosensory (proprioceptive). The central nervous system does not simply average these inputs; it assigns dynamic, context-sensitive weights to each modality based on their relative reliability — a process formalised in Peterka's sensorimotor integration model [10]. When one modality becomes unreliable, the brain upweights the remaining inputs, most commonly the visual system. In healthy individuals, this compensatory visual upweighting is adaptive and time-limited; in patients who develop VID, abnormal upweighting of visual input persists beyond the initial insult and becomes pathological [1,3,10,40].



Schematic illustration only. Normal values follow the Peterka (2002) firm-surface model (somatosensory-dominant); the VID column shows pathological visual up-weighting and is illustrative / conjectural, not measured.

*Figure 1. Sensory weighting for spatial orientation - normal versus visually induced dizziness (schematic abstraction). Under normal conditions on a firm surface in good light, postural orientation is somatosensory-dominant (approximately 70% somatosensory, 20% vestibular, 10% visual; Peterka 2002). In VID, visual input is pathologically up-weighted. The VID column is an illustrative abstraction, not measured data, and visual dependence remains a proposed mechanism.*

*Source: Adapted from Peterka [10] and Cousins et al. [3].*

### The three-sensor conflict model

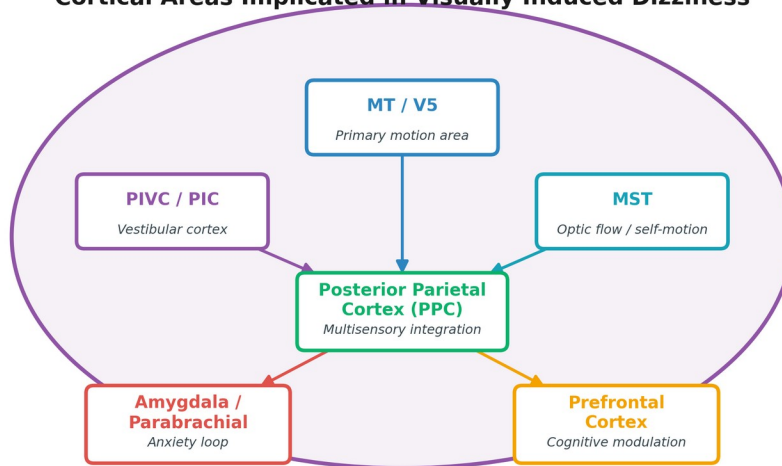
Brandt's foundational model of visual vertigo proposed that the underlying mechanism is a mismatch — or conflict — between a pathologically weighted visual motion signal and the accompanying vestibular and somatosensory signals [11]. In optic-flow-rich environments, the visual system signals movement while the vestibular and proprioceptive systems signal stillness. In a patient with normal visual weighting, this mismatch is modest and readily resolved. In a patient with pathological visual upweighting, the mismatch is amplified — generating the subjective experience of dizziness, unsteadiness, and spatial disorientation [1,2,11]. The conflict model explains why static visual environments are symptom-free in most VID patients and why symptoms are dose-dependent on the magnitude and complexity of the optic flow field [2,5,6,13]. Pavlou et al. demonstrated this dose-response by incrementally increasing optokinetic drum velocity and tracking symptom and postural sway responses in patients with peripheral vestibular disorders [6].

### Cortical visual motion processing

Neuroimaging studies have provided important insights into the cortical substrate of VID. The middle temporal area (MT/V5) and the medial superior temporal area (MST) are the primary cortical regions for visual motion and optic flow processing respectively [15,16,17]. In healthy controls, vestibular stimulation

deactivates MT/V5 and visual stimulation deactivates the vestibular cortex — a reciprocal inhibition phenomenon that provides the neural substrate by which the two modalities compete for dominance [15,16]. The parieto-insular vestibular cortex (PIVC) and posterior insular cortex (PIC) serve as the primary vestibular cortical areas and both show reciprocal relationships with MT/V5 [17,18]. In patients with vestibular disorders and evidence of visual dependency, fMRI studies show enhanced activation of MT/V5 and MST in response to optic flow stimuli alongside reduced activity in PIVC — the neuroimaging correlate of pathological upweighting [15,16,17]. Reduced vestibular cortical tone disinhibits the visual motion processing network, amplifying the cortical response to optic flow [15,16,18]. The posterior parietal cortex (PPC) serves as a critical multisensory integration hub; dysfunction at this level explains the difficulty some patients experience even in moderately complex environments [30].

### Cortical Areas Implicated in Visually Induced Dizziness



PIVC = Parieto-insular vestibular cortex | PIC = Posterior insular cortex | MST = Medial superior temporal area | PPC = Posterior parietal cortex

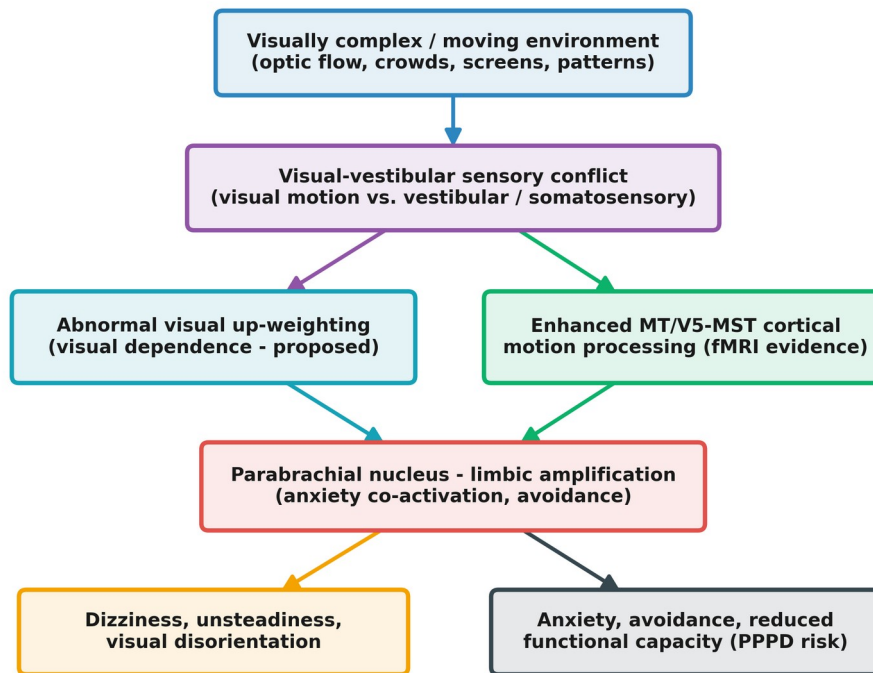
Figure 2. Cortical areas implicated in visually induced dizziness. Reduced vestibular cortical tone (PIVC/PIC) disinhibits MT/V5 and MST, amplifying the response to optic flow. Limbic projections via the parabrachial nucleus mediate anxiety co-activation.

Source: Adapted from Dieterich and Brandt [15], Bense et al. [16], and Brandt and Dieterich [18].

## The anxiety amplification pathway

A third mechanistic pathway — the anxiety or limbic amplification loop — is clinically and therapeutically central to VID. Balaban's work on the parabrachial nucleus demonstrated direct anatomical connections between brainstem vestibular nuclei and limbic structures, providing the neural basis for the well-documented comorbidity of vestibular disorders with anxiety and panic disorder [27,28]. When visually complex environments trigger dizziness in a patient who already has heightened autonomic arousal, the limbic response amplifies the sensory signal: sympathetic activation, hyperventilation, and attentional bias toward vestibular and visual symptoms compound the subjective severity and promote avoidance behaviour [19,27,44]. Avoidance is the clinical consequence most relevant to rehabilitation planning — patients who reduce exposure to trigger environments protect themselves from acute distress but deprive the central nervous system of the sensory input required for compensatory recalibration [20,26,38]. This creates the maintenance loop driving VID from acute symptom to chronic condition, mirroring the perpetuating mechanisms well characterised in PPPD [9,32]. The single most important therapeutic principle is graduated re-exposure to visual triggers, with anxiety management to reduce the amplification factor [5,7,25].

### Pathophysiology of Visually Induced Dizziness



*Schematic synthesis - 'visual dependence' is a proposed mechanism, not a measured pathway.*

Figure 3. Pathophysiology of visually induced dizziness (schematic synthesis). Three intersecting mechanisms - visual up-weighting, enhanced cortical motion processing, and limbic amplification - are proposed to converge to generate symptoms and drive avoidance. This is an abstract conceptual model; visual dependence is a proposed mechanism rather than a measured pathway.

Source: Adapted from Peterka [10], Dieterich and Brandt [15], and Balaban et al. [27,28].

□ **Clinical Insight:** The three-mechanism model directly predicts the treatment approach: optokinetic habituation addresses visual upweighting, graded exposure addresses cortical motion amplification, and anxiety management addresses limbic amplification. Treating only one mechanism produces partial, unstable improvement.

## III. Clinical Features and Symptom Characterisation

VID presents as dizziness, unsteadiness, visual disorientation, nausea, or spatial confusion triggered specifically by visually complex or motion-rich environments. The symptom quality is typically described as a floating, rocking, or swimming sensation rather than true rotational vertigo; this distinction is clinically important because patients and non-vestibular clinicians sometimes mistake the absence of spinning for the absence of vestibular pathology [2,12,39]. Symptoms arise within seconds to minutes of entering the trigger environment and typically subside over minutes to hours on removal from the trigger — or not at all in patients with severe chronicity and prominent anxiety overlay [1,2,13,43].

### Classic trigger environments

The canonical triggers map directly onto the nine items of the VVAS: walking through a supermarket aisle, walking through a shopping mall, watching traffic at a busy intersection, being a passenger in a car, going down an escalator, walking over a patterned floor, watching a movie at the cinema, watching action television, and being under fluorescent lights [4,1,13]. The common structural feature of all these environments is large-field visual motion or high spatial-frequency patterns that overwhelm the patient's reduced visual-vestibular integration capacity [1,6,11]. Triggers are hierarchical in severity: most patients are comfortable in simple environments and deteriorate as visual motion complexity increases. The patient's individual trigger hierarchy forms the foundation of graduated exposure rehabilitation [5,7,23,45]. Clinicians who fail to systematically identify this hierarchy will design programmes that are either

insufficiently challenging (no therapeutic habituation) or too aggressive (sympathetic overflow and avoidance reinforcement) [7,8].

Screen-related triggers deserve specific clinical attention in the contemporary patient population. Scrolling social media feeds, video calls, gaming environments, and cinema attendance are among the highest-rated triggers in working-age patients [4,41]. Screen avoidance is common and has occupational consequences not always captured by the DHI or standard balance-outcome measures [40,46]. The VVAS item 'screen scrolling' or 'reading in motion' is often the most functionally limiting item in younger adults, while supermarket and traffic environments dominate in older patients [4,41].

## Functional and psychological impact

The functional impact of VID is underestimated when outcome is measured solely by vestibular test battery. The DHI does capture visual symptom components through its physical and functional subscales, but only partially reflects VID-specific restriction [40,46]. Patients with significant VID progressively restrict vocational, social, and recreational participation as avoidance deepens: they stop driving, resign from work, withdraw from social events, and reduce physical activity — with secondary deconditioning compounding the primary vestibular impairment [44,20,19].

Yardley et al. demonstrated a specific relationship between balance system dysfunction and agoraphobic avoidance: dizziness triggered by visually complex public environments is one of the strongest predictors of avoidance behaviour and secondary anxiety in dizzy patients, independent of symptom severity [44]. VID left untreated commonly transitions to PPPD — the persistent postural-perceptual dizziness phenotype in which visual sensitivity is one of three core diagnostic criteria [9,32]. Early identification and intervention for VID may represent the most effective point for PPPD prevention [31,32].

**□ Important:** VID that is not identified and treated within 12 months of onset carries a high conversion risk to PPPD — with its associated chronicity, functional impairment, and resistance to rehabilitation. Early, targeted assessment and treatment represents the most effective prevention strategy.

## IV. Diagnostic Criteria, Assessment Tools, and the VVAS

Unlike BPPV or Meniere's disease, VID does not have a validated, standalone set of Barany Society diagnostic criteria. The closest relevant framework is the 2017 PPPD consensus criteria, in which visuospatial sensitivity (dizziness exacerbated by complex visual environments or moving visual stimuli) constitutes Criterion C [9]. For patients who do not meet full PPPD criteria — because symptoms are less than three months in duration, are situationally bounded, or lack pervasive non-positional dizziness — but who clearly have VID as a dominant complaint, a clinical operational definition based on the VVAS remains the practical standard [4,9,32,42].

### History-taking framework

A structured VID history has three essential components. First, trigger identification: systematically screen all nine VVAS domains (supermarket aisle, shopping mall, traffic at a busy intersection, car passenger, escalator, patterned floor, cinema, action television, and fluorescent lighting). Ask the patient to rate each on a 0-10 scale [4]. Second, avoidance assessment: for each endorsed trigger, determine whether the patient currently avoids the environment (scoring 0 because they no longer go there, not because it is symptom-free). Active avoidance masquerades as a reduced score and must be distinguished from genuine habituation [19,44]. Third, onset and trajectory: was there a precipitating vestibular event? Has the VID worsened, plateaued, or improved? What compensatory strategies is the patient already using? [2,39,43]

### The Visual Vertigo Analogue Scale (VVAS)

The VVAS, developed and validated by Dannenbaum, Chilingaryan and Fung (2011), is the gold-standard patient-reported outcome measure for VID [4]. It comprises nine visual trigger scenarios rated on a visual

analogue scale from 0 (no dizziness) to 10 (worst dizziness imaginable), giving a total of 0-90. Items were adapted from earlier visual-vertigo measures (Longridge and colleagues) and validated against the DHI in a cohort of 102 patients with vestibulopathy. Internal consistency was excellent (Cronbach's alpha 0.94) and the scale correlated strongly with the total DHI ( $r = 0.67$ ) in the original validation cohort [4].

The VVAS has several properties that make it particularly valuable in clinical practice and in research settings. Unlike the DHI, which measures general dizziness-related handicap, the VVAS specifically captures the visual trigger dimension - allowing differentiation of the VID component from other dizziness subtypes in complex patients [4,40,46]. A mean item score above 2 out of 10 (total above 18 on the nine-item scale) has been proposed as a clinically meaningful threshold for visual dependency in dizzy-clinic populations [41]. As a rehabilitation outcome measure, a minimum clinically important difference (MCID) of approximately 15-20% reduction in total score has been reported, though this requires further validation across different VID populations [4,7,37].

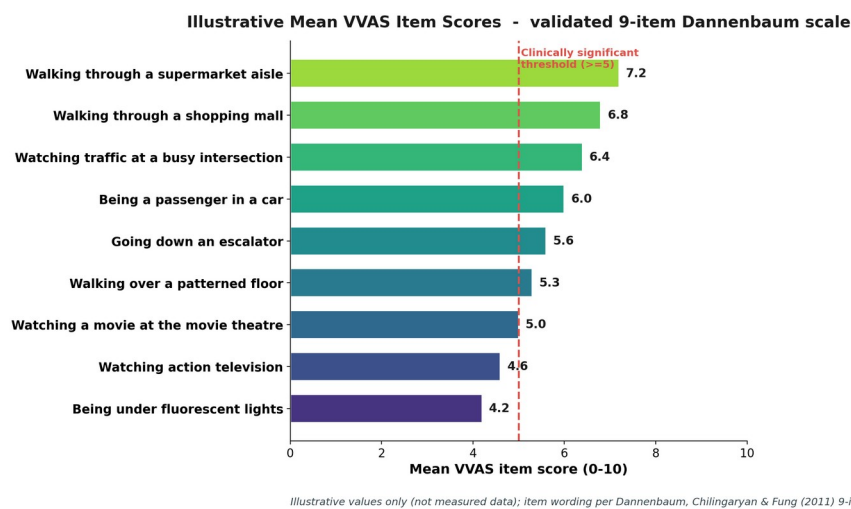


Figure 4. Illustrative mean VVAS item scores in a dizzy-clinic population - validated nine-item Dannenbaum scale. Supermarket, shopping mall, and busy-intersection traffic consistently score highest, reflecting their complex large-field optic-flow properties. Illustrative values only - not measured data.

Source: Item wording from Dannenbaum, Chilingaryan & Fung [4]; illustrative score pattern adapted from Cousins et al. [41].

**Table 2. VVAS structure, scoring, and clinical interpretation.**

Parameter	Detail
Items	9 visual trigger scenarios; VAS 0-10 per item
Total score range	0-90 (sum of all items)
Clinically significant threshold	Mean item score greater than 2 (total greater than 18); high dependency: mean greater than 5 (total greater than 45) [41]
Validated against	DHI ( $r = 0.67$ , $p$ less than 0.0001); internal consistency Cronbach alpha 0.94 ( $n = 102$ ) [4]
MCID (proposed)	15-20% total score reduction; correlates with functional improvement in VRT trials [4,7]

Beyond the VVAS, supplementary instruments add clinical depth. The DHI remains widely used and its visual subscale items partially capture VID impact [40,46]. Computerised dynamic posturography, where available, can objectively quantify visual dependency by comparing performance under visual-conflict versus non-visual conditions on the Sensory Organisation Test (SOT) [10,35,36]. A disproportionate performance improvement when vision is restored (high Condition 1:2 ratio) and failure under visual conflict (Conditions 3, 5) indicates significant visual dependency [10,36].

□ **Clinical Pearl:** When assessing a VVAS, always probe avoidance explicitly. A patient who scores 0 on 'supermarket' because they have not been in one for six months is not symptom-free — they are deeply avoidant. Avoidance masquerading as improvement is the principal source of under-treatment in VID rehabilitation.

## V. Investigations and Objective Measures

VID is primarily a clinical and history-based diagnosis. Investigations serve two purposes: to characterise any underlying peripheral vestibular deficit that may have precipitated the visual dependency, and to exclude structural or central pathology that could mimic or coexist with VID [2,12,39]. In the majority of patients, routine vestibular function testing will be normal or show the residual pattern of a resolved peripheral vestibular event — neither finding alters the immediate treatment plan, which is rehabilitation [1,2,22].

### Vestibulo-ocular reflex (VOR) testing

Video head impulse test (vHIT) assesses the integrity of the high-frequency VOR in each semicircular canal plane. In primary VID without a preceding vestibular event, vHIT is typically normal. In secondary VID following vestibular neuritis, BPPV, or labyrinthitis, vHIT may show residual gain reduction or catch-up saccades [24,39,40]. The critical insight is that the magnitude of the peripheral vestibular deficit as measured by VFT does not predict VID severity: patients with near-complete VOR recovery can have profound VID, and patients with significant caloric reduction may have minimal visual dependency [3,14,24]. This dissociation is explained by the primarily central nature of VID — visual dependency is a compensatory response, not a direct measure of the peripheral lesion [3,14].

### Computerised dynamic posturography (CDP)

CDP provides the most direct objective measure of sensory weighting and visual dependency. The Sensory Organisation Test (SOT) systematically removes or conflicts visual and somatosensory inputs across six conditions. Visual dependency is indicated by disproportionate performance improvement when visual input is restored and by catastrophic failure under visual conflict conditions (3, 5) [10,35]. The sensory analysis score quantifies the relative contributions of vision, vestibular input, and somatosensation, providing a quantitative profile analogous to the sensory weighting model [10,36]. Serial SOT scores across a rehabilitation programme correlate meaningfully with functional improvement [35,36].

### Optokinetic assessment and neuroimaging

Optokinetic stimulation using a rotating drum, projector screen, or virtual reality environment displaying moving patterns is a provocative test that directly engages the VID mechanism. Abnormal postural sway or symptom provocation during optokinetic stimulation is a positive test for VID; response severity can be graded by increasing stimulus velocity or pattern complexity [5,6,7]. In clinical practice, a screen-based optic flow stimulus (rotating rings, expanding dots) serves adequately in any clinic with a large monitor [7,8]. Neuroimaging is not required in the majority of patients with straightforward VID and a plausible precipitating vestibular event. Indications for MRI include: progressive worsening without an identified trigger, additional neurological symptoms, onset after age 60 without precipitating event, or any feature suggesting a central vestibular disorder such as spontaneous vertical nystagmus, abnormal vHIT in a cerebellar canal distribution, or abnormal VEMPs [2,39,42].

**Table 3. Investigation summary for visually induced dizziness.**

Investigation	Typical finding in VID	Indication / Value
vHIT	Normal (primary VID) or residual gain reduction (secondary VID)	Characterises peripheral VOR integrity; does not quantify VID severity
Caloric testing	Normal or unilateral weakness in secondary VID	Useful if vHIT equivocal; rarely changes rehabilitation plan
VEMP (oVEMP/cVEMP)	Normal in most VID; may be reduced if otolith-pathway involved	Otolith function; screen if bilateral weakness or otolith disorder suspected
CDP / SOT	High visual dependency ratio; abnormal Conditions 3 and 5 scores	Direct objective measure of sensory weighting; rehabilitation outcome measure
MRI brain	Normal in isolated VID	Mandatory for atypical features, focal neurology, or progressive course

□ **Key Point:** A normal vHIT and caloric test do not exclude VID — they confirm the absence of a current peripheral VOR deficit. The diagnosis rests on history and the VVAS, not on vestibular function tests. Ordering a full VFT battery without taking a VID history will miss the diagnosis in the majority of patients.

## VI. Differential Diagnosis

Several conditions share visual trigger symptoms with VID, and overlap is the rule rather than the exception. Accurate differentiation requires a structured history, targeted provocation, and an understanding of the characteristic features of each differential. The most clinically challenging differential pairs are: VID versus PPPD, VID versus vestibular migraine (VM), and VID versus functional neurological disorder (FND) [9,29,31,32,34].

Persistent postural-perceptual dizziness (PPPD) is the condition most closely related to VID — visuospatial sensitivity is Criterion C of the PPPD diagnostic framework and VID is frequently the presenting phenotype of evolving PPPD [9,32]. The distinction is clinically meaningful: PPPD requires dizziness on most days for at least three months (Criterion A), exacerbated by upright posture and movement (Criterion B), and by complex visual environments (Criterion C) [9]. A patient with VID who meets Criterion C but not A or B, or whose symptoms have been present less than three months, should be coded as VID and treated with the same rehabilitation framework — early treatment may prevent transition to full PPPD [9,31,32].

Vestibular migraine frequently presents with visual sensitivity and complex visual aura, and VM patients have a well-characterised visual symptom burden spanning both episodic attacks and the interictal period [29,34]. The differential features are the episodic nature of VM attacks (minutes to hours), their association with headache, phonophobia, and visual aura, and the migraine history. In practice, VM and VID co-exist — a migrainous individual who develops VID after a precipitating vestibular event requires both migraine management and VID rehabilitation in parallel [29,34]. Mal de débarquement syndrome (MdDS) represents an important related condition: a persistent rocking/swaying sensation after sea, air, or land travel, with the paradoxical feature that patients typically improve in visually complex environments (moving cars, ships) and worsen at rest — the inverse of VID [37]. Functional neurological disorder with vestibular features can present with disproportionate and inconsistent visual sensitivity; hallmarks are symptom variability on distraction and high health anxiety [31].

**Table 4. Differential diagnosis of visually triggered dizziness.**

Condition	Visual trigger	Discriminating features	Management direction
VID (isolated)	Yes — principal feature	VVAS elevated; normal posture at rest; no persistent daily dizziness	Optokinetic rehabilitation + graded exposure
PPPD	Yes — Criterion C	Daily dizziness greater than 3 months; Criteria A, B, and C all met; often post-precipitant	VRT + SSRI/SNRI + CBT
Vestibular migraine	Yes — episodic and interictal	Episodic attacks; migraine history; headache/photophobia	Migraine prevention + VRT
Mal de débarquement	Inverse — improves with motion	Persistent rocking; post-travel onset; worsens at rest	VRT; transcranial stimulation under trial [37]
Anxiety/panic disorder	Phobic avoidance	Panic features; no vestibular precipitant; minimal VVAS vs. high health anxiety	CBT/exposure; anxiolytic if needed
Functional vestibular disorder	Variable, inconsistent	Distractibility; inconsistent provocation; major life event trigger	Multidisciplinary; neuro-psychiatry liaison

□ **Clinical Insight:** The VID-PPPD boundary is a clinical spectrum, not a binary. Treat the visual trigger phenotype regardless of whether formal PPPD criteria are met — the rehabilitation principles are identical and early treatment is the strongest predictor of preventing chronicity.

## VII. Vestibular Rehabilitation and Optokinetic Habituation

Vestibular rehabilitation therapy (VRT) with targeted optokinetic habituation is the evidence-based first-line treatment for VID. Three randomised controlled trials by Pavlou and colleagues have established that optokinetic-enriched VRT — incorporating graded exposure to moving visual stimuli — produces superior outcomes compared with standard VRT alone or no treatment [5,7,8]. The mechanism is habituation: repeated, subthreshold exposure to visual triggers progressively reduces the cortical motion response and promotes re-weighting of the sensory hierarchy toward more normal vestibular and somatosensory contributions [5,6,8].

### Assessment and treatment planning

Prior to commencing VRT, the clinician constructs a personalised trigger hierarchy using the VVAS — ranking environments from least to most provoking. This hierarchy forms the exposure ladder guiding treatment progression [7,23,45]. The most commonly used framework grades exposure from low-complexity optic flow in a controlled clinical setting, through intermediate stimuli (standardised optokinetic drum or screen-based optic flow), to real-world environments in graded complexity [5,7,8]. Each step is practised until the patient achieves a pre-defined symptom habituation threshold (typically two consecutive sessions at or below 3/10 on the VAS) before advancing. This prevents therapeutic overwhelm while ensuring sufficient stimulus intensity to drive habituation [5,7,45].

#### Graded Exposure Ladder for VID Vestibular Rehabilitation

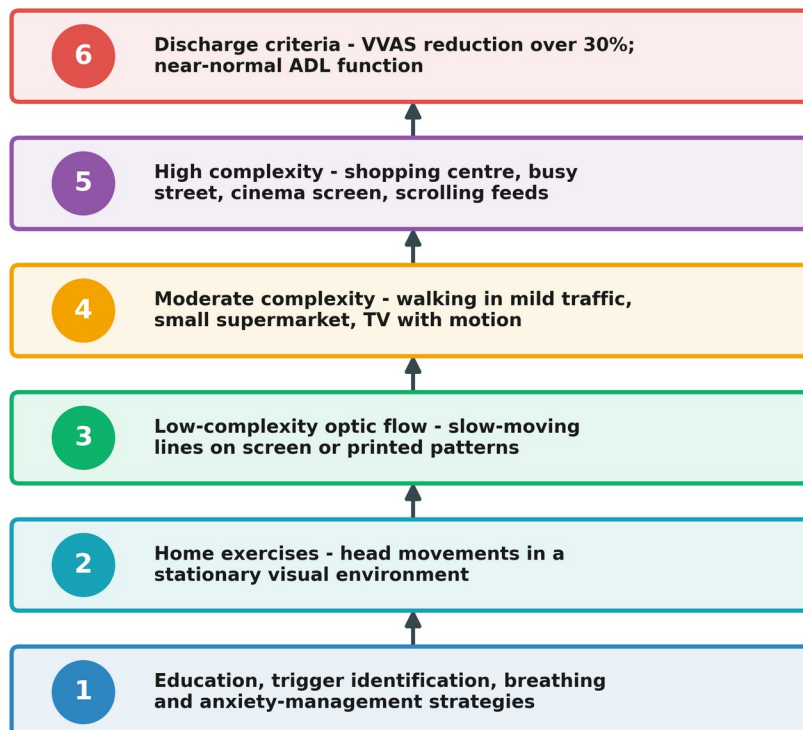


Figure 5. Graded exposure ladder for VID vestibular rehabilitation. Each step is practised to habituation (VAS 3/10 or less on two consecutive sessions) before advancement. Steps are personalised to the patient's VVAS trigger hierarchy.

Source: Adapted from Pavlou et al. [5,7,8] and Herdman [45].

## Optokinetic stimulation in clinic

Pavlou's optokinetic rehabilitation programme uses a full-field projection screen or large monitor displaying rotating or expanding optic flow patterns of incremental velocity and complexity [5,6,7]. Patients are initially seated, then progress to standing, tandem stance, and head movement tasks within the optic flow environment — incrementally loading the visual-vestibular interaction. The supervised programme typically comprises 6-12 clinic sessions over 4-8 weeks, supplemented by daily home exercises using a simplified optic flow stimulus [7,8]. Pavlou et al. (2015) demonstrated that supervised optokinetic VRT produced significantly greater VVAS reductions and DHI improvements than unsupervised standard VRT at 12 weeks, with effects maintained at 24 weeks [7]. The supervised advantage reflects the importance of clinician-guided progression through the trigger hierarchy [8].

## Real-world exposure and virtual reality

Clinic-based optokinetic training must be complemented by graded exposure in real-world environments to achieve generalisation of habituation [5,7,45]. A structured real-world exposure programme assigns weekly exposure tasks based on the VVAS hierarchy: for example, a 15-minute supervised supermarket visit using a pre-agreed anxiety management strategy. The principle is exposure without avoidance — the dizziness is allowed to habituate rather than being terminated by exit from the environment [7,25,45]. Clinicians should document exposure tasks assigned, adherence, and VVAS/VAS ratings at each session to track improvement and identify avoidance masquerading as low symptom scores [4,19,44].

Virtual reality (VR) platforms offer controlled, reproducible, and progressively graded optic flow environments without the logistical demands of real-world exposure assignments [47]. Meldrum et al. (2015) demonstrated equivalence between VR-based and conventional balance rehabilitation in unilateral peripheral vestibular loss [47], and emerging pilot data suggest specific benefit for the VID phenotype given the capacity to simulate high-complexity visual environments at calibrated stimulus levels. The principal limitation of consumer VR headsets in vestibular rehabilitation is VR-induced motion sickness — a paradox in a population already highly susceptible to visual motion sickness — which requires careful initial calibration and monitoring [48]. Dedicated clinical VR systems avoid this by using low-latency, high-field-of-view displays engineered specifically for vestibular use.

□ **Clinical Pearl:** The single strongest predictor of VRT success in VID is adherence to the home exposure programme. Patients who limit exposure to clinic sessions plateau early. Weekly real-world exposure tasks — documented, reviewed, and advanced at each session — drive the bulk of functional recovery outside the clinic.

# VIII. Pharmacotherapy, Psychological Interventions, and Adjuncts

## Pharmacotherapy

There is no pharmacological treatment with established efficacy for isolated VID as a primary endpoint. Vestibular suppressants (antihistamines, benzodiazepines, prochlorperazine) are contraindicated in VID management: they reduce central arousal, impair VOR plasticity and gaze stabilisation adaptation, and actively inhibit the habituation process that rehabilitation depends upon [21,38,39]. Their short-term symptom relief comes at the cost of prolonged central compensation failure and worsened long-term outcome. Clinicians who receive patients on chronic vestibular suppressants should initiate a supervised withdrawal programme as part of the overall treatment plan [21,38].

When VID coexists with PPPD (i.e., when full PPPD criteria are met), SSRIs and SNRIs have established efficacy for the PPPD phenotype and should be prescribed alongside rehabilitation [9,32]. Venlafaxine 37.5-75 mg daily has the strongest evidence base in PPPD, with sertraline and fluoxetine representing reasonable alternatives [32]. The mechanism is primarily anxiolytic and partially involves central serotonergic modulation of the vestibular-limbic pathway [27,32]. Pharmacotherapy does not replace VRT — the two modalities are additive, and patients receiving both show superior outcomes compared with

either alone [9,32]. Betahistine has no evidence base in VID or PPPD and should not be prescribed for this indication [33].

## Cognitive-behavioural therapy and acceptance-based approaches

Psychological intervention is a core component of VID management when anxiety, avoidance behaviour, or health anxiety is identified as a significant perpetuating factor [19,25,26,44]. CBT delivered by a vestibular-aware psychologist targets cognitive appraisal of vestibular symptoms (catastrophising, hypervigilance), behavioural patterns that maintain avoidance, and autonomic arousal that amplifies the visual-vestibular conflict [19,20,25]. Naber et al. (2011) demonstrated that interdisciplinary VRT incorporating mindfulness and CBT produced significantly greater DHI reductions than VRT alone in a mixed vestibular population [25]. Acceptance and commitment therapy (ACT) is theoretically well-suited to VID: rather than targeting symptom elimination, ACT facilitates psychological flexibility and willingness to engage with triggering environments despite dizziness — directly addressing the experiential avoidance that drives VID chronicity [25,26].

## Multidisciplinary management

Optimal VID management integrates the vestibular physician, vestibular physiotherapist, and psychologist into a coordinated treatment programme [9,25,31]. The vestibular physician's role spans the diagnostic phase — characterising the vestibular substrate, identifying PPPD comorbidity, prescribing where appropriate — and the supervision of the VRT programme, particularly in managing optokinetic dose escalation for patients with severe visual dependency or high anxiety at presentation [9,31,32]. Communication between team members regarding the patient's current trigger hierarchy position, avoidance patterns, and psychological co-activators is critical to programme coherence.

□ **Clinical Insight:** For patients with VVAS scores above 45 (mean item score greater than 5) and significant avoidance, a coordinated vestibular physiotherapy and psychology programme delivers better outcomes than VRT alone. Referring the 'refractory VID' patient to a psychologist is not a diversion — it is evidence-based co-treatment.

# IX. Prognosis, Predisposing Factors, and Special Populations

The prognosis of VID with targeted rehabilitation is generally favourable: Pavlou's randomised controlled trials report significant VVAS score reductions (typically 30-50%) and meaningful functional improvement in the majority of patients completing a 6-12 session VRT programme [7,8,22]. Improvement is generally maintained at 24-week follow-up, though some patients experience symptom relapse on re-exposure to high-complexity trigger environments after a prolonged avoidance period [7,8,37]. Key prognostic determinants are: duration of symptoms before treatment (shorter is better), anxiety severity (higher anxiety predicts slower response), degree of avoidance at presentation (more avoidance predicts greater initial optokinetic sensitivity and a longer exposure ladder), and rehabilitation adherence [5,7,19,26].

**Table 5. Prognostic factors in visually induced dizziness.**

Factor	Direction	Notes
Symptom duration less than 6 months	Favourable	Earlier treatment prevents chronicity and PPPD transition [9,32]
Isolated VID without PPPD	Favourable	Simpler treatment target; no pharmacotherapy required in most cases [9]
High anxiety / panic disorder comorbidity	Unfavourable	Requires psychological co-treatment; longer rehabilitation course [19,25,27]
High avoidance at presentation	Unfavourable initially	Longer exposure ladder; increased risk of drop-out [7,44]
Good rehabilitation adherence	Strongly favourable	Home exposure programme completion is the dominant outcome predictor [7,8]

Secondary VID post-vestibular neuritis	Favourable	Clear precipitant; resolves well with VRT in majority [3,22]
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## Predisposing and perpetuating factors

Pre-existing anxiety disorder, panic disorder, or health anxiety is the most consistently identified risk factor for developing VID after a precipitating vestibular event [19,26,27]. Individuals with these traits have an amplified limbic response to vestibular symptoms, more profound avoidance behaviour, and slower rates of spontaneous vestibular compensation [19,27,44]. Migraine biology independently elevates VID risk: VM patients have pre-existing central motion-processing amplification — a low threshold for visual motion discomfort — and a higher baseline VVAS even in the absence of acute vestibular pathology [29,34]. Perpetuating factors that maintain VID once established include: persistent avoidance of trigger environments, ongoing vestibular suppressant use (which prevents central recalibration), inadequate VRT (failing to include optokinetic elements), and continued uncontrolled exposure to high-stakes visual environments without compensatory graduated exposure [19,21,38,44].

## Special populations

Older adults with VID present additional challenges. Multisensory decline — reduced proprioceptive acuity, impaired visual acuity, and age-related vestibular peripheral loss — creates a platform of reduced sensory redundancy rendering the elderly especially vulnerable to pathological visual upweighting [20,39]. Falls risk is substantially elevated in older patients with VID, and addressing the visual dependency dimension in falls-prevention programmes represents an underutilised intervention point [20,23]. VRT for older patients should incorporate slower exposure progression, balance training on unstable surfaces, and functional mobility goals aligned with activities of daily living [22,23,45].

Working-age patients with screen-intensive jobs (programmers, data analysts, media professionals) represent a growing VID subpopulation in whom occupational screen exposure directly drives symptom perpetuation [4,41]. The clinical challenge is that absolute screen avoidance is not operationally possible, while unrestricted use may prevent habituation. A graduated, time-limited screen exposure programme — beginning with brief intervals on a static screen and building to sustained scrolling content — with ergonomic optimisation (screen size, room lighting, refresh rate) produces the best occupational outcomes in this group [4,41,45]. Post-COVID dizziness has introduced a new cohort of VID patients, in whom persistent visuospatial sensitivity is described as a component of the post-COVID vestibular syndrome, with central sensitisation and PPPD-like mechanisms likely implicated [31].

## Vestibular compensation and hippocampal considerations

A growing body of evidence, including Brandt's demonstration that vestibular loss causes hippocampal atrophy and impaired spatial memory in humans, suggests that inadequately compensated vestibular disorders carry cognitive sequelae beyond the vestibular symptom burden [49]. VID — by perpetuating incomplete compensation and sustaining avoidance of spatially rich environments — may compound this hippocampal stress through reduced spatial navigation. This provides an additional rationale for early, comprehensive VID rehabilitation: beyond symptomatic benefit, restoring engagement with varied spatial environments may protect hippocampal function in older patients [49,50]. This remains an active area of investigation.

**Key Point:** VID in the elderly is a falls risk, not just a comfort issue. Falls-prevention services should screen for visual dependency using the VVAS or SOT and incorporate optokinetic rehabilitation into balance programmes for older patients with unexplained falls in visually complex environments.

## X. Guidelines, Controversies and Future Directions

No dedicated international clinical practice guidelines exist specifically for VID. The Barany Society PPPD consensus (2017) provides the nearest recognised framework and applies to patients who meet full PPPD criteria [9]. The International Classification of Vestibular Disorders (ICVD) includes visuospatial sensitivity as a defined symptom category but does not specify management [12,42]. Clinical practice should default

to the PPPD treatment principles where applicable and to Pavlou's optokinetic VRT programme for isolated VID [5,7,8,9].

## Controversies

- **Nosological debate — VID vs. PPPD vs. SMD:** The clinical and mechanistic overlap between VID, PPPD, and space-and-motion discomfort has generated debate about whether these are distinct entities or phenotypic variants on a single continuum of central vestibular dysfunction [9,13,32,42]. The practical consensus is that the treatment approach is sufficiently similar to allow clinicians to treat the symptom complex without resolving the nosological debate.
- **Pharmacotherapy threshold:** At what PPPD comorbidity threshold does SSRI/SNRI prescribing become indicated? Current evidence supports pharmacotherapy when full PPPD criteria are met, but there is no RCT evidence specifically for partial-PPPD or isolated VID. Shared decision-making is best practice [9,32].
- **Optokinetic vs. standard VRT:** Pavlou's trials demonstrate optokinetic superiority, but involve supervised programmes using specialised equipment. Real-world generalisability to community VRT settings where optokinetic stimulators may not be available remains uncertain. Screen-based home programmes show promise but are under-resourced in most settings [5,7,8].
- **VVAS as sole diagnostic criterion:** The VVAS is the best available validated instrument for VID severity, but its use as a diagnostic threshold is based on a single validation cohort. A multi-site, prospective validation study is needed to establish a formal diagnostic cut-point [4,41].
- **VR rehabilitation:** Virtual reality VRT is promising but no RCT specifically in VID exists to date. The motion sickness paradox in VR headsets remains a practical barrier. Expanded trials are in progress [47,48].

## Patient education and self-management

Patient education is a treatment modality in VID, not merely a supplement. Patients who understand the mechanism — that their brain has learned to over-rely on visual input and that deliberate, graduated visual challenge is the fix — are substantially more adherent to exposure programmes than those who perceive the exercises as arbitrary [5,19,25]. A simple mechanistic explanation using the sensory weighting model is effective in clinic: 'Your balance system is currently using your eyes as its primary reference. The treatment is to practise in environments that challenge that reliance — gradually, so your brain recalibrates.' Framing the anticipated dizziness during exposure as evidence that the treatment is working (habituating) rather than evidence of harm is critical to adherence [5,7,19].

## Future directions

Active research areas include: standardised VVAS diagnostic cut-points from large multi-site cohorts [4,41]; randomised trials of VR-based optokinetic rehabilitation with active comparator [47]; head-mounted optic flow stimulators for home use that avoid the motion-sickness limitations of consumer VR [47,48]; transcranial non-invasive brain stimulation (tDCS/rTMS) targeting MT/V5 or PIVC as an adjunct to habituation [30]; and machine-learning analysis of optokinetic postural sway responses to auto-grade exposure programmes. From the nosological side, a Barany Society working group has been proposed to develop standalone diagnostic criteria for VID distinct from PPPD — a development that would substantially advance both clinical research and coding [42]. At the health-system level, scalable, clinician-supervised digital rehabilitation programmes combining screen-based optokinetic stimulation with tele-supervised exposure assignments represent the most pragmatic model for expanding VID treatment access at the population level [7,8,25,50].

□ **Key Point:** VID is under-diagnosed, under-treated, and potentially preventable. Every vestibular service should routinely screen with the VVAS, deliver targeted optokinetic rehabilitation, and identify PPPD comorbidity requiring pharmacotherapy. The treatment works — the barrier is awareness and access, not evidence.

## Summary

Visually induced dizziness is a mechanistically well-characterised, evidence-based treatable condition at the intersection of vestibular physiology, cortical sensory processing, and behavioural psychology. The vestibular physician's value in VID management is: at the diagnostic phase, to characterise the vestibular substrate, quantify VID severity with the VVAS, identify PPPD comorbidity, and initiate appropriate pharmacotherapy; in the rehabilitation phase, to supervise the optokinetic VRT programme and coordinate multidisciplinary care; and in the long term, to prevent transition to chronic PPPD by early, comprehensive treatment. The sensory weighting shift that drives VID is reversible: with targeted graduated exposure, the brain recalibrates its reliance on visual input, and the majority of patients achieve clinically meaningful, durable improvement.

Five practice points consolidate this review:

- **Screen every dizzy patient for VID** — ask specifically about supermarket, traffic, scrolling screens, and crowds. Administer the VVAS if any item is endorsed. VFT-normal does not mean VID-absent.
- **Probe avoidance, not just symptoms** — a low VVAS score because the patient has stopped going to triggering environments is not improvement; it is avoidance. Reframe it as the core therapeutic target.
- **Prescribe optokinetic rehabilitation, not standard VRT alone** — the evidence base for VID requires graded visual stimulation; gaze-stabilisation exercises without optic flow challenge are insufficient.
- **Screen for PPPD** — if VID has been present for more than three months with daily dizziness and positional/movement exacerbation, PPPD criteria are likely met and SSRI/SNRI plus psychology referral are indicated alongside VRT.
- **Act early** — VID presenting within 6 months responds faster and more completely than established PPPD. Prevention of chronicity is the single highest-yield outcome in VID management.

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