Visually Induced Dizziness (VID) — Cheat Sheet for Vestibular Physicians

Visual dependence and the visual–vestibular mismatch — assessment, the VVAS, and optokinetic rehabilitation.

► Why VID matters

One of the commonest and most treatable phenotypes in the dizzy clinic — about 57% of consecutive specialist neuro-otology patients show clinically significant visual dependency (Cousins 2017), and roughly 15–20% of community adults with chronic dizziness report visually provoked symptoms. VID is a disproportionate reliance on vision for spatial orientation, so optic-flow-rich environments (supermarkets, screens, traffic) become destabilising. The balance hardware works — the brain's control software stays locked in a vision-dependent mode after the precipitant has resolved. It is a positive, treatable diagnosis, and a normal vestibular test battery does not exclude it.

► When to suspect VID

► *Floating, rocking or swimming dizziness — not true spinning — coming on within seconds of entering a visually busy or moving environment and easing on leaving.*

► *Symptoms map onto the nine VVAS triggers: supermarket, shopping mall, busy traffic, car passenger, escalator, patterned floor, cinema, action TV, and fluorescent lighting.*

► *An identifiable precipitant in secondary VID — peripheral vestibular event, vestibular migraine, concussion or a stressful period — or de novo onset in primary VID.*

► *Normal or only well-compensated examination with a normal test battery: "every scan is normal but busy places make me dizzy."*

► Trigger environments — the visual-motion triad

Trigger category	What patients describe	Bedside / history pointer
Large-field optic flow	*Supermarkets, shopping malls, busy traffic, crowds — worse as visual-motion complexity rises*	*"Do busy or moving visual environments make you worse?" — the highest-yield screening question*
Self / passive visual motion	*Car passenger, escalators, scrolling screens and cinema*	*Provoked by visual motion in any direction; screens dominate in working-age patients*
High spatial-frequency pattern	*Patterned or shiny floors, stripes, fluorescent lighting*	*Static simple scenes are tolerated — symptoms are dose-dependent on optic-flow complexity*

Pearl — Two screening questions capture VID with high yield: "Do busy or moving visual environments make you worse?" and "Are screens or supermarkets a problem?" Consistent yes answers raise VID strongly.

► Diagnosis — no standalone criteria; anchor on the VVAS

Element	Requirement
Trigger pattern	*Dizziness, unsteadiness or disorientation provoked by complex, moving or optic-flow-rich visual environments*
Symptom quality	*Floating, rocking or swimming within seconds of a busy scene — not true rotational vertigo*
VVAS	*Nine-item Visual Vertigo Analogue Scale; mean item score over 2 (total over 18 of 90) supports clinically significant visual dependency*
PPPD overlap	*Daily dizziness over 3 months with postural/motion exacerbation likely meets PPPD criteria (visuospatial sensitivity is Criterion C)*
Exclusions	*No spontaneous/positional nystagmus, spinning attacks or vomiting — these point to active disease that may coexist*

Pearl — Probe avoidance, not just symptoms. A low VVAS score because the patient has stopped going to triggering environments is avoidance, not improvement — reframe it as the core therapeutic target.

► Bedside examination and diagnostic approach

Domain	Expected in VID	Interpretation
Neuro / vestibular exam	*Normal, or only an old well-compensated deficit*	*Deficit size does not predict VID severity — VID is central reweighting*
Spontaneous / positional nystagmus	*Absent*	*Its presence points away from isolated VID — seek active disease*
Optokinetic / optic-flow provocation	*Sway or symptoms on a rotating drum or screen-based optic flow*	*Positive provocation engages the mechanism; grade by velocity and complexity*
Spinning vertigo / vomiting	*Absent*	*If present, look for BPPV, Ménière's or vestibular migraine that may coexist and keep triggering VID*

Pearl — VFT-normal does not mean VID-absent. The magnitude of any peripheral deficit does not predict VID severity; visual dependency is a central reweighting phenomenon.

► Targeted investigations — proportionate, not reflexive

Investigation	Purpose	When to order
VVAS questionnaire	*Quantify visual-trigger severity and track rehabilitation*	*Every suspected VID patient — the core measure*
Dynamic posturography (SOT)	*Objectively quantify visual dependency (fail conditions 3 and 5; gain on vision restoration)*	*Where available; for severity and serial tracking*
vHIT, VNG with calorics	*Characterise any precipitating peripheral deficit (usually old/compensated)*	*Most patients, to confirm no active loss*
Optokinetic provocation	*Engage and grade the VID mechanism*	*In clinic via drum or large-screen optic flow*
MRI brain	*Exclude central or structural disease*	*Red flags only: progressive course, focal signs, onset over 60 without precipitant*

Pearl — Over-investigation is anti-therapeutic. In the right pattern, a normal battery supports VID — reflexive serial imaging delays rehabilitation and entrenches health anxiety.

► Red flags — these are NOT isolated VID: new spontaneous or positional nystagmus, fresh focal neurological signs, acute hearing loss, spontaneous vertical (down-beat) nystagmus, or a cerebellar-pattern vHIT. Their presence demands a search for active vestibular or central pathology — image and/or refer before attributing symptoms to VID.

► Key differentials — and what distinguishes them

Condition	Distinguishing feature from VID
PPPD	Daily dizziness over 3 months with postural and motion exacerbation; VID (visuospatial sensitivity) is its Criterion C — a continuum
Vestibular migraine	Discrete vertigo episodes with headache, photophobia and aura; migraine history — frequently coexists with VID
Mal de débarquement (MdDS)	Rocking after travel that improves in motion (driving) and worsens at rest — the inverse of VID
Functional neurological disorder	Inconsistent, distractible visual sensitivity with high health anxiety
Uncompensated vestibular hypofunction	Active deficit on vHIT/calorics with directional symptoms; VID is central and disproportionate to the lesion

Pearl — Coexistence is the rule. VID frequently overlaps with vestibular migraine or a compensated neuritis — treat both layers rather than forcing one label, and screen for PPPD if dizziness is daily for over 3 months.

► Management — coordinated, multimodal stepped care

Tier	Intervention	Practice principles
1 · Education	Name VID; explain the brain over-relies on vision and that graded visual challenge is the fix	Therapeutic in itself and the indispensable first step — drives adherence
2 · Optokinetic VRT	Optokinetic-enriched rehabilitation up a VVAS-based exposure ladder; real-world and VR exposure	First-line (Pavlou RCTs); advance each rung at VAS 3/10 or less on two sessions; pace to avoid flare
3 · Psychology	CBT or ACT for catastrophising, hypervigilance and avoidance	Add where anxiety or avoidance perpetuates; best combined with VRT
4 · Pharmacology	No drug for isolated VID; SSRI/SNRI (venlafaxine) only if full PPPD criteria are met	Vestibular suppressants are contraindicated — they block habituation; withdraw if chronic

Pearl — Avoid vestibular suppressants. Antihistamines, benzodiazepines and prochlorperazine blunt arousal and block the habituation rehabilitation depends on — withdraw chronic users under supervision.

► Prognosis, relapse and perpetuating factors

► With optokinetic-enriched rehabilitation the prognosis is good — typically 30–50% VVAS reduction and meaningful functional gain in most completing a 6–12 session programme, largely maintained at 24 weeks.

► Act early — VID presenting within 6 months responds faster and more completely; untreated VID commonly transitions to PPPD, so early treatment is the best PPPD-prevention point.

► Favourable: short duration before treatment, low anxiety, limited avoidance, good adherence. Unfavourable: high trait anxiety, entrenched avoidance, untreated migraine, ongoing suppressant use.

► Relapse follows a new vestibular insult, high stress or a long avoidance period — discharge with a maintenance plan: continued graded exposure, retained strategies, and early re-engagement.

Key references — Dannenbaum et al. (VVAS). J Vestib Res 2011 · Cousins et al. PLoS One 2014 · Pavlou et al. Otol Neurotol 2015 · Bronstein. JNNP 1995 · Staab et al. (PPPD criteria). J Vestib Res 2017.