



Dizziness and Vertigo

Part 1: Untangling the complexity of dizziness and vertigo requires a careful history.

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A stepwise and careful history helps identify the many causes of vertigo and dizziness. In the next issue, Part 2 covers the oculomotor and vestibular examinations.

First, Characterize Symptoms

Definitions

In 2009, the Barany Society published the first consensus classification of vestibular symptoms. *Internal vertigo* is a false or distorted sensation of self-motion including spinning, swaying, bobbing, tilting, bouncing, and sliding. *External vertigo* is a false or distorted sensation of the surroundings, excluding bidirectional motion, which is known as *oscillopsia*. The feeling of being unstable without a particular direction preference while sitting, standing, or walking is *unsteadiness*. *Dizziness* is a nonmotion sensation of disrupted spatial orientation. Purposefully, the definitions do not suggest a particular disease pathophysiology.¹ Orthostatic hypotension and benign paroxysmal positional vertigo (BPPV), for instance, can both induce vertigo or dizziness,^{2,3} although the term *vertigo* will be used throughout this article to describe either symptom. Patients may have more than a single symptom at a time. For example, a common combination of symptoms in vestibular neuritis includes vertigo (due to semicircular canal imbalance) and oscillopsia (due to horizontal jerk nystagmus). Patients may also have symptoms that transition from one to another over time; for example, acute vertigo to chronic unsteadiness.

Direction

In some vestibular disorders (eg, vestibular paroxysmia), patients have directionally specific spinning that may be better recognized in vertigo than in external vertigo.⁴ Spinning vertigo that changes direction during a single event, is unique to Ménière's disease and related to the phases of the attack—excitatory, inhibitory, or recovery.⁵ Understanding the direction of vertigo can occasionally help lateralize the disorder or better understand the pathophysiology.

Duration

Vertigo spells are brief, usually lasting seconds in patients with BPPV, vestibular paroxysmia, and cardiac arrhythmias.

Patients with Ménière's disease, vestibular migraine (VM), or transient ischemic attacks (TIAs) often present with vertigo spells lasting minutes to hours. In patients with vestibular neuritis or central vestibular lesions from stroke or demyelination, vertigo lasts days to weeks. Patients with bilateral vestibular loss (BVL), uncompensated unilateral vestibular loss (UVL), chronic intoxication, or persistent postural perceptual dizziness (PPPD) often have months to years of symptoms.

In episodic conditions, asking the patient how long the specific vestibular symptom lasted (ie, dizziness or vertigo), rather than how long an attack lasted, can give a better estimate of duration. For example, a patient with BPPV may overestimate attack duration (eg, 5 minutes) because of persisting vegetative symptoms (eg, nausea, vomiting, and sweating) even when the spinning associated with BPPV lasted less than 1 minute.⁶

Second, Categorize Symptoms

Vestibular disorders can be grouped by presentation into acute, episodic, and chronic vestibular syndromes (AVS, EVS, and CVS, respectively). Patients with AVS present with more than 24 hours of continuous vertigo (lasting days to weeks and monophasic) with nausea/vomiting, imbalance, head motion intolerance, spontaneous nystagmus (eg, stroke or vestibular neuritis). Patients with EVS have similar symptoms and signs as AVS, lasting seconds to hours (eg, Ménière's disease, VM). Patients with CVS have constant vestibular symptoms for weeks to years (eg, bilateral vestibular loss).

A convenient strategy is to employ a 2-layer approach to acute and episodic clinical syndromes (Figures 1-4).¹

1. First Layer: Are symptoms provoked or unprovoked?
2. Second Layer: Do vestibular symptoms occur in isolation or are there additional neurologic or audiologic symptoms?

Core clinical syndromes commonly overlap and evolve as do symptoms (Figure 4, Table).⁷



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Symptoms may evolve from acute or episodic to chronic, or from episodic to acute, and acute and episodic symptoms may be concurrent

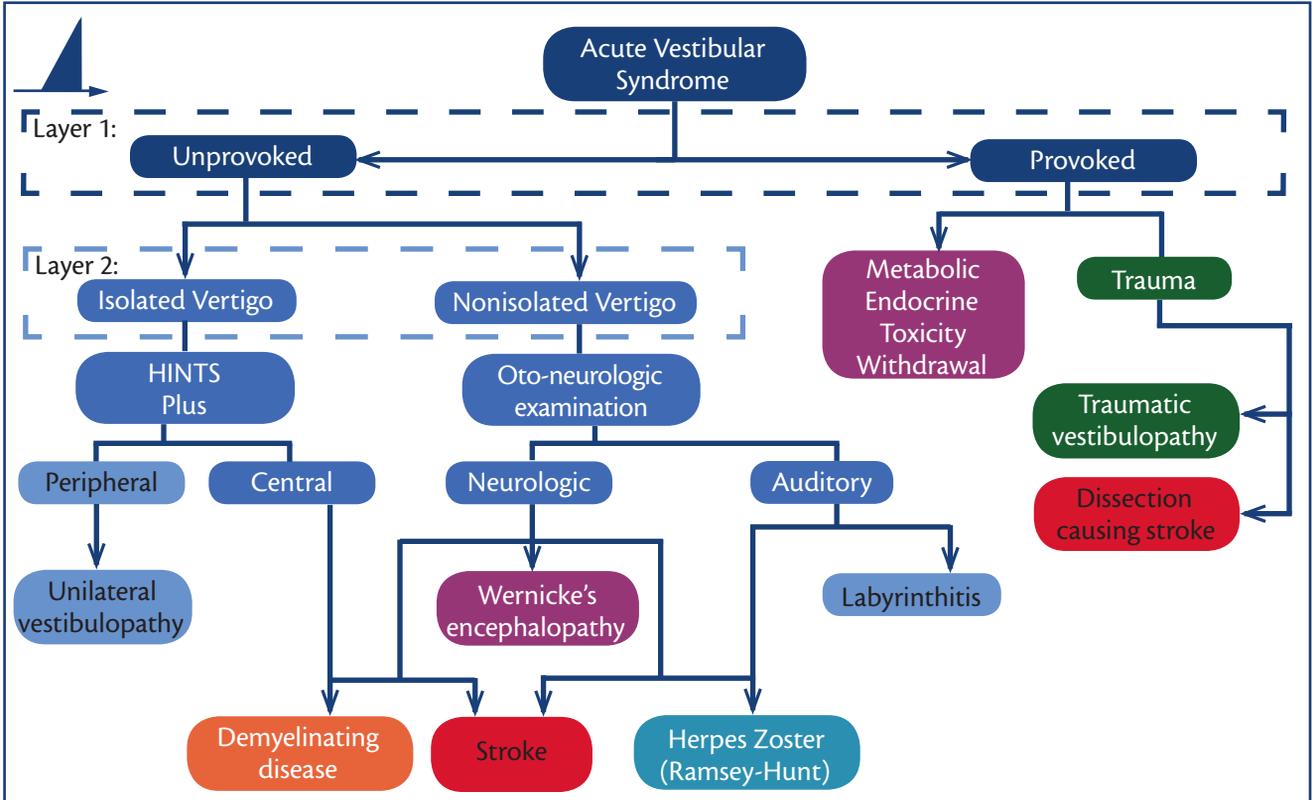


Figure 1. Acute vestibular syndromes (AVS). Note: HINTS Plus is a mnemonic for head impulse, nystagmus, test for skew, plus new unilateral hearing loss.

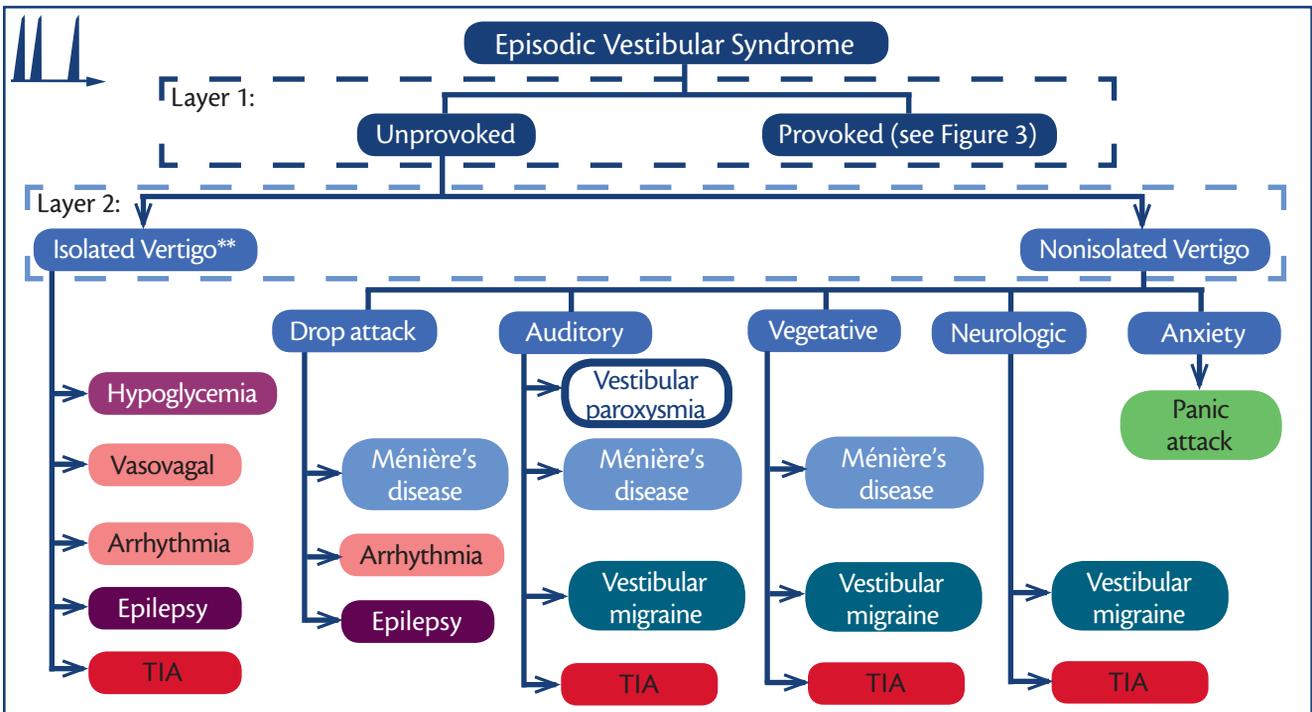


Figure 2. Episodic vestibular syndromes (EVS)—unprovoked. **Each of these disorders may or may not be isolated (eg, aura is common in seizure and diaphoresis in hypoglycemia.) Abbreviation: TIA, transient ischemic attack.

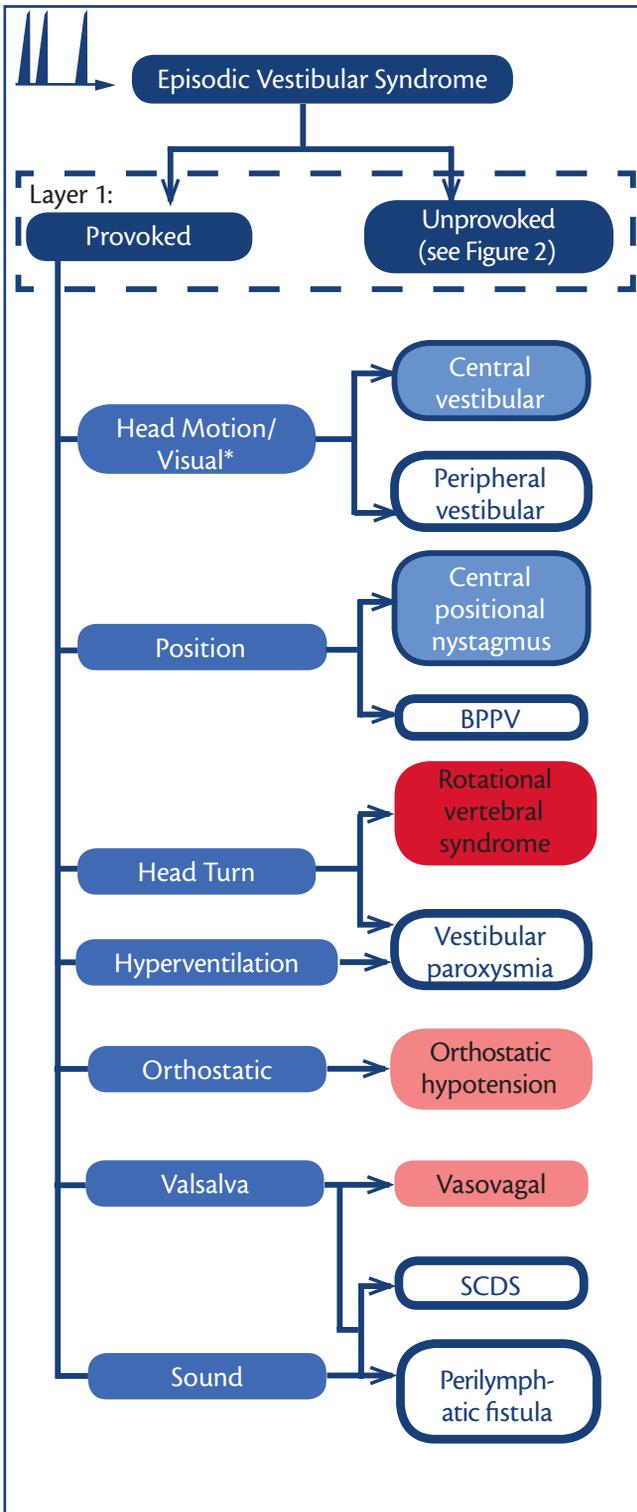


Figure 3. Episodic vestibular syndromes (EVS)—provoked. * Visual vertigo following any vestibular disorder. Abbreviations: BPPV, benign paroxysmal positional vertigo; SCDS, superior canal dehiscence syndrome.

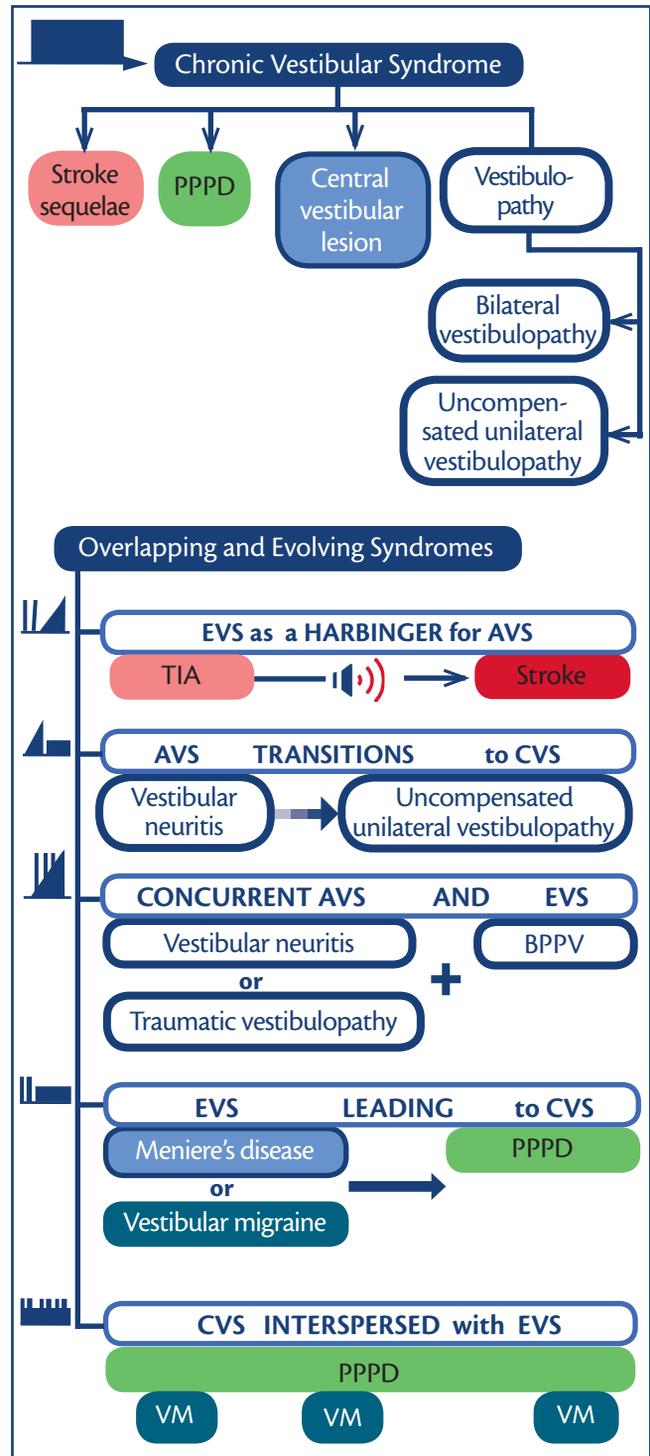


Figure 4. Chronic vestibular syndromes and overlapping/evolving syndromes. Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; CVS, chronic vestibular syndrome; EVS, episodic vestibular syndrome; PPPD, persistent postural perceptual dizziness; TIA, transient ischemic attack; VM, vestibular migraine.



TABLE. THE MOST COMMON VESTIBULAR SYNDROMES

Vestibular Conditions	Symptoms & Historical Pearls
Acute Vestibular Syndromes (AVS) (>24 hours)	
Peripheral (vestibular neuritis [VN] and labyrinthitis)	Vertigo/dizziness, disequilibrium, nausea/vomiting, “sitting” oscillopsia at rest (not dependent on head motion, also referred to as “external vertigo”) from nystagmus; aggravated by head movements; hearing is spared in VN and lost in labyrinthitis; consider labyrinthine ischemia when new hearing loss is present
Central (stroke > demyelinating disease and other)	Symptoms can be indistinguishable from VN; may or may not have additional posterior fossa symptoms
Wernicke’s syndrome	History of alcoholism, malnutrition, hyperemesis gravidarum, gastric bypass or related surgeries; dizziness/vertigo, imbalance, confusion, diplopia are common
Spontaneous Episodic Vestibular Syndromes (EVS)	
Transient ischemic attack (TIA) (minutes to hours)	Similar to AVS, TIAs may present as isolated dizziness/vertigo or with additional posterior fossa symptoms; new headache with vestibular symptoms is concerning for dissection, or may be due to TIA
Vestibular migraine (seconds to days)	Vertigo, dizziness, imbalance; history of motion sickness; vestibular symptoms occur with or without headache; headache history may be remote and unrelated, but other migraine features (eg, photo- and phonophobia, nausea) will be present and patients may describe similar triggers for vestibular symptoms
Ménière’s disease (20 minutes to 12 hours)	Vertigo, aural fullness, hearing loss, and tinnitus
Vestibular paroxysmia (seconds to minutes)	Dizziness, vertigo, imbalance commonly associated with ipsilesional aural symptoms often spontaneous and many times each day; can be provoked by exercise or head movements
Triggered Episodic Vestibular Syndromes	
Benign paroxysmal positional vertigo (BPPV) (<1-2 min)	Vertigo or dizziness triggered by head movements (eg, rolling over in bed, lying to sitting, sitting to lying, looking up or down)
Superior canal dehiscence syndrome (SCDS) (sec to min)	Autophony (hearing internal noises that are not normally perceived (eg, heartbeat, eye movements); episodic vertigo/dizziness brought on by pressure changes (eg, sneezing) or loud noises; may have chronic disequilibrium
Chronic Vestibular Syndromes (CVS)	
Bilateral vestibular loss (BVL)	Oscillopsia provoked by head movements (“walking” oscillopsia [dependent on head motion]); imbalance
Persistent postural perceptual dizziness (PPPD)	After a vestibular, medical, or psychiatric event causing dizziness/vertigo or impaired balance, some patients (particularly those prone to anxiety or who had a particularly anxious response to the initial event) develop a maladaptive reaction including stiff/rigid posture that can lead to chronic dizziness (lasting > 3 months) with a postural component (worse when upright) and visual sensitivity
Mal de débarquement syndrome (MDDS)	Rocking or swaying, feeling of being on a boat usually experienced after a cruise, long car ride, or flight (although some cases are spontaneous); symptoms are minimal with passive motion as in a car; significant overlap with migraine
Cerebellar/brainstem syndromes	Oculopalatal tremor: oscillopsia due to vertical-torsional pendular nystagmus, worsening imbalance months after a posterior fossa injury (eg, pontine hemorrhage); flocculus/paraflocculus syndrome: oscillopsia from downbeat nystagmus, progressive ataxia (eg, spinocerebellar ataxia); uvula/nodulus syndrome: oscillopsia from periodic alternating nystagmus, central positional vertigo/nystagmus
Modified from: Neuro-Ophthalmology Virtual Education Library: NOVEL. https://collections.lib.utah.edu/ark:/87278/s64x9bq1.	

Third, Identify Symptom Triggers
Head Motion, Position, Orthostatic Change, or Exertion

Head Motion. In a patient with a vestibular disorder (peripheral or central), a normal head movement can lead to a faulty estimation of movement. Symptoms are experienced during or time-locked with the head movement.

Position. Attacks of vertigo and dizziness in patients with BPPV or central positional nystagmus occur in response to changes in the gravitational vector during movements such as looking down-then-up, bending over, or rolling over in bed. Symptoms are triggered by the head movement.



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In patients with acute-onset severe vomiting, who cannot stand for evaluation, consider cerebellar stroke as well as gastroenteritis. Shaking the head will significantly aggravate cerebellar stroke symptoms but should not effect a nonvestibular condition⁸

Orthostatic Change. Triggered by changing from a seated to standing position or from lying to seated, orthostatic vertigo and may be related to a neurologic condition (eg, multiple system atrophy), medications (eg, antihypertensives), hypovolemia, or presyncope. If symptoms are triggered by standing up from sitting when there is no change in spatial orientation of the head with respect to gravity (eg, standing from a chair without moving the head), orthostatic hypotension is favored. Symptoms triggered by going from sitting to lying or rolling over in bed (both cause a change in gravitational vector), in contrast, favor BPPV.

Exertion. Exertion-related vertigo may be caused simply by activities leading to head movements that trigger positional or head motion-induced symptoms. If vertigo occurs with exertion when the head is stationary, however, cardiopulmonary disorders should be considered. Additionally, hyperventilation and changes in cerebrospinal fluid (CSF) pH that occur with strenuous activity may also lead to 8th cranial nerve hyperexcitability in vestibular paroxysmia or with acoustic neuroma.

Head Turning and Eye Position

Head Turning. Some patients with vestibular paroxysmia may experience short spells of vertigo with head turn (to the right or left) in the upright position, because they may have more neurovascular contact with certain head positions. Rotational vertebral artery occlusion syndrome should be considered when protracted head turn induces stereotypical vertigo attacks.⁴

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Patients who are symptomatic from any vestibular disorders (eg, vestibular neuritis) will have worsening of symptoms with the Dix-Hallpike maneuver; however, this should not be considered a positive Dix-Hallpike test for BPPV

Eye Position. A particular eye position may bring on symptoms. For example, monocular oscillopsia can be induced by down and medial gaze in superior oblique myokymia.

Sound, Valsalva, and External Ear Pressure Changes

Patients with a third-window syndrome (superior canal dehiscence syndrome [SCDS], perilymph fistula, or enlarged

vestibular aqueduct syndrome) have inner ear bony structure deficits. Changes in intracranial or middle ear pressure or loud sound (Tulio phenomenon) often lead to inappropriate excitation or inhibition of a semicircular canal, causing vertigo and nystagmus. In SCDS, excitatory stimuli for the anterior (also known as superior) canal include Valsalva against pinched nostrils (eg, blowing the nose), positive pressure in the external auditory canal (EAC) (eg, inserting a wet finger into the EAC) or a loud sound. The resultant nystagmus will be downbeat-torsional (top poles beating toward the affected ear). Inhibitory stimuli include Valsalva against a closed glottis (eg, heavy objects lifting, coughing, straining, and laughing) or negative pressure in the EAC (eg, pulling out a wet finger from the EAC). The resultant nystagmus will be upbeat-torsional (top poles beating toward the unaffected ear).⁹ A history of head trauma including barotrauma and blast injury is a known risk factor.¹⁰ In comparison to patients with third-window syndromes, vertigo spells in Chiari malformation or situational and vasovagal syncope may be driven by closed-glottis Valsalva maneuvers but are not triggered by loud sound or middle ear pressure changes.

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In VM, spontaneous vertigo and phonophobia (discomfort from loud sounds) co-occur. In contrast, in SCDS, the sound (eg, alarm, siren) triggers the vertigo⁹

Complex Visual Environments, Passive Self-Motion, or Illusions of Passive Self-Motion

Loss or distortion of vestibular input may cause increased reliance on visual information for balance, which is thought to be the genesis of most visual vertigo. Complex visual environments include patterned wallpaper or carpets or a busy grocery store. In patients who overrely on visual input, there is commonly impaired compensation for moving scenes, (eg, passive self-motion on a car, bus, train, or plane) or illusions of passive self-motion (eg, video games on large screens, 3D movies, virtual reality headsets or looking at traffic) that can cause spatial disorientation. Abnormal visual dependency can cause anxiety specific to open spaces or shopping centers and ultimately agoraphobia. Visual tasks that require fixation on small target (eg, mobile devices or reading a book) may be a trigger in patients with PPPD.¹¹

Walking on Uneven Surfaces or in the Dark

Understanding that normal balance relies upon visual, proprioceptive, and vestibular inputs can help the clinician establish which system(s) are impaired depending on the specific condition(s) that worsen balance. Asking about situations with down-regulation of visual cues (walking in the dark) and dis-



rupted somatosensory cues (walking on uneven surfaces) can have localizing value.

A history of oscillopsia while walking (ie, head movement dependent) will favor BVL,¹² whereas an abnormal general neurologic exam in a patient with imbalance may suggest a nonvestibular etiology (eg, diminished vibration/proprioception sensation and hyporeflexia in polyneuropathy). In elderly patients, imbalance is often multifactorial, and related to a combination of orthopedic, neuropathic, visual, proprioceptive, and vestibular impairments. Recovery from vestibular neuritis may be suboptimal because of combined abnormal visual dependency and anxiety, despite objective vestibular function tests showing recovery of function.¹³

Fourth, Assess Associated Symptoms

Vegetative Symptoms: Nausea and Vomiting

When it's not clear whether a patient with acute prolonged or episodic symptoms has a vestibular or nonvestibular etiology, the presence of vegetative symptoms suggest a vestibular disorder.

Auditory Symptoms: Deafness, Tinnitus and Aural Fullness

Ischemic auditory symptoms. New unilateral hearing loss in a patient with AVS is concerning for anterior inferior cerebellar artery (AICA) ischemia. The inner ear is particularly susceptible to ischemic injury because it is supplied by an end artery, the internal auditory artery (IAA). The most common mechanism of IAA territory infarction is thrombotic stenosis of the parent vessel, usually the AICA, or the origin of the AICA in the basilar artery. Spells of vertigo associated with auditory symptoms (eg, tinnitus or unilateral hearing loss) that last for minutes can represent AICA TIAs that precede stroke. Patients with transient vestibular or auditory symptoms and vascular risk factors should have head and neck vascular imaging and brain MRI.¹⁴



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Isolated labyrinthine infarction is undetectable on neuroimaging—even diffusion-weighted MRI—this clinical diagnosis must be considered, especially in patients with vascular risk factors¹⁴

Nonischemic auditory symptoms. Clinically, labyrinthitis resembles vestibular neuritis but can be differentiated by the presence of acute unilateral hearing loss. In patients with acute or chronic middle ear infection or meningitis, abrupt audiovestibular symptoms are concerning for bacterial labyrinthitis. Abrupt-onset audiovestibular symptoms with ipsilesional peripheral facial paresis and vesicular rash (may involve the auricle, EAC, and tympanic membrane) is concerning for herpes zoster (Ramsay-Hunt syndrome).

Head injuries, especially those including temporal bone trauma, can cause hearing loss with or without vestibular symptoms. In patients with a recent temporal bone fracture, barotrauma, or stapes surgery, co-occurrence of episodic vertigo and unilateral hearing loss may suggest a perilymphatic fistula.¹⁰ Supranormal bone thresholds and a low-frequency conductive hearing loss in the presence of normal tympanometry are associated audiologic findings of SCDS.⁹

Progressive peripheral facial nerve weakness with auditory symptoms (eg, tinnitus and hearing loss) suggests a neoplastic process in the middle ear (eg, glomus body tumor), internal auditory canal, or cerebellopontine angle (eg, vestibular and facial schwannoma or metastasis).^{15,16}

Fluctuating sensorineural hearing loss in the low-to-medium frequency (> 30 dB, < 2000 Hz) range with tinnitus and aural fullness occur in association with spontaneous episodic vertigo in patients with Ménière's disease. With recurrent attacks, hearing loss is often progressive.⁵



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Recovery of low to mid frequency sensorineural hearing loss on a follow-up audiogram suggests Ménière's disease

Cogan's syndrome has similar presentation to Ménière's disease (episodic vertigo with fluctuating hearing loss) with a more fulminant course and interstitial keratitis.¹⁷ Other inflammatory disorders that may involve the inner ear include polyarteritis nodosa, systemic lupus, Sjögren syndrome, Vogt-Koyanagi-Harada syndrome, granulomatosis with polyangiitis, and relapsing polychondritis.¹⁸ Patients with presyncope may experience muffled sounds. Patients with VM may have a mild feeling of aural fullness and tinnitus.¹⁹

Migraine Headache

Episodes of VM typically last 5 minutes to 72 hours and are accompanied by a variety of sensations including dizziness, vertigo, and/or unsteadiness that are often aggravated by head movements and visual stimulation. Associated migraine features may include headache (unilateral onset, throbbing or pounding in quality, moderate to severe in intensity and worsening by routine physical activity), phonophobia (bilateral sound discomfort), photophobia and visual aura, and nausea.¹⁹ Patients with migraine may experience associated migraine features before, during, or after vestibular symptoms. Commonly, patients are overwhelmed by vestibular symptoms and devalue the associated headache or non-headache migraine features during the acute phase.¹⁹

Neurologic Symptoms

Neurologic symptoms of diplopia, dysarthria, numbness, weakness, hiccups, vision loss, or clouded consciousness should



be assessed. Although stroke is the most common cause of central AVS, multiple sclerosis and Wernicke's encephalopathy also should be considered.

In addition to nonischemic causes of a lower motor neuron facial palsy discussed, the presence of vertigo and a 7th nerve palsy should also raise suspicion for an AICA territory infarction involving the root entry zone and 7th nerve fascicle.¹⁴

Inflammatory disorders (eg, granulomatosis with polyangiitis, lupus erythematosus, or sarcoidosis) and infectious diseases (eg, tuberculosis, fungal infection, or syphilis) are often subacute and may present as focal (inflammatory infiltrate or abscess) or diffuse (multiple cranial neuropathy) neurologic deficits. Autoimmune encephalitis (paraneoplastic or non-paraneoplastic) also has a subacute temporal profile.

Autophony

A patient with SCDS may report hearing an echo of their own voice, footsteps, or movements of the jaws and eyes while chewing or looking around, respectively. Autophony is also a core clinical symptom in patients with a patulous eustachian tube; however, these patients will note an echo of their own breath sounds that patients with SCDS will not.⁹

Anxiety

Anxiety is a common comorbidity of all vestibular disorders and may adversely influence recovery. Of note, spells of dizziness or vertigo may result from a panic attack, agoraphobia, traumatic stress disorders, or generalized anxiety. Patients with anxiety-related dizziness are less likely to report external vertigo or significant vegetative symptoms.

Last, in Cases of Isolated Vertigo

The most common diagnoses of AVS are vestibular neuritis and stroke (about 5%-10% of cases), which can be indistinguishable with history and general neurologic examination. Even MR with diffusion-weighted imaging (DWI) in the first 24 to 48 hours of an attack may be falsely negative in 6% to 21% of strokes.²⁰ Approximately 29% of patients with vertigo caused by a posterior circulation stroke reported a preceding isolated episode of vertigo that lasted minutes.²¹ Transient isolated vertigo attacks are potentially a warning sign for strokes, and like any other transient neurologic attack, vascular imaging (CT angiography or MRA) should be considered.²² Because there is potential for acute MRI results to be false negative, CT-perfusion may be helpful in some cases if posterior fossa ischemia is suspected.²⁰

Conclusion

By approaching the vestibular history methodically, an accurate diagnosis can usually be made, or the differential diagnosis at least narrowed substantially. Using symptom type alone to diagnose dizzy or vertiginous patients is an ineffective

approach and often leads to the wrong diagnosis and inappropriate testing. Once timing and triggers are understood, categorization into acute, episodic, or chronic vestibular syndromes can be done and a targeted exam chosen. That examination will then confirm the diagnosis and/or inform further audiovestibular testing or neuroimaging. The approach to the vestibular and ocular motor examination will be discussed in Part 2 in the next issue of *Practical Neurology*. ■

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Disclosure

The authors have no financial or other relationships relevant to this content to disclose.