Nonvestibular Dizziness

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KEYWORDS

• Non-vestibular dizziness • Autonomic disorders • Visual disturbances
• Cervicogenic disorders • Medications • Vertigo

KEY POINTS

- Dizziness is a common patient complaint that may be due to vestibular or nonvestibular etiologies.
- There are many causes of nonvestibular dizziness including visual disturbances, autonomic dizziness, cervicogenic dizziness, medication-induced dizziness, metabolic dysregulation, thyroid diseases, and cardiovascular conditions.
- A detailed understanding of these medical conditions will help aid diagnosis and appropriate management.

Video content accompanies this article at http://www.oto.theclinics.com.

INTRODUCTION

Dizziness is a common symptom, accounting for an estimated 5% of all primary care visits.[1] In fact, approximately 35% of Americans report a lifetime prevalence of dizziness.[2,3] Determining the root cause of a patient's dizziness, however, is a diagnostic challenge for many providers. During history taking, patients use the terms "dizziness" and "vertigo" interchangeably to describe a broad range of sensations.[4] Although

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typical American usage defines dizziness as an umbrella term with vertigo as a subset category, the Barany Society delineates these two terms to be nonhierarchical.[5] Dizziness refers to a distorted sense of spatial orientation without a false sense of motion, whereas vertigo describes the sensation of self-motion when there is no movement or the perception of distorted self-motion with normal head movement. Owing to the confusion patients may face with distinguishing between dizziness versus vertigo, some providers find it helpful to ask patients to describe what they are feeling to determine whether they are experiencing true vertigo, presyncope/syncope, unsteadiness, anxiety, lightheadedness, and so forth. Broadly speaking, the differential diagnosis for dizziness may be broken down into two categories: (1) vestibular (central or peripheral) versus (2) nonvestibular sources of dizziness. This article focuses on common causes of nonvestibular dizziness such as visual disturbances, autonomic dizziness, cervicogenic dizziness (CGD), medication-induced dizziness, and other miscellaneous medical conditions. Within each subsection, we will provide an overview of these disorders with the goal of increasing awareness, diagnosis, and management of nonvestibular dizziness.

DIZZINESS AND OSCILLOPSIA IN VISION DISORDERS

Superior Oblique Myokymia

Superior oblique myokymia (SOM) is a monocular disorder characterized by episodic involuntary contraction of the superior oblique muscle. As the primary action of the superior oblique muscle is intorsion, SOM results in torsional high-frequency low-amplitude eye movements (Video 1).[6] This may be experienced by the patient as episodes of oscillopsia (shifting, shimmering, jumping, or fluttering of vision), vertical or oblique diplopia (since the secondary action of the superior oblique muscle is depression), or occasionally vague symptoms of dizziness. The frequency and duration of symptoms can vary greatly between individual patients. Typically, a patient may report multiple daily occurrences over a period of weeks or months, with each episode lasting seconds.[7,8] Symptoms may occur more frequently with stress or fatigue and may spontaneously remit and then recur months or years later.[8]

Diagnosis of SOM can be challenging. Patients with SOM may end up in an otology clinic because the associated oscillopsia can be misinterpreted as a vestibular symptom. The monocular nature of the disorder is an important clinical clue, and SOM should be considered in any patient reporting dizziness or oscillopsia which resolves when the affected eye is covered. In most patients, there are no abnormal signs between episodes.[7,8] Episodes can sometimes be induced by downgaze or by a head tilt toward the affected side. In some patients, the high-frequency low-amplitude eye movements may be difficult to appreciate on bedside examination and may be better seen with binocular video-oculography or with the fundoscopic examination (to observe oscillation of the retinal structures).[7] Notably, monocular video-oculography may not capture SOM episodes if the camera is viewing the unaffected eye.

MR imaging is often normal in patients with SOM,[7,8] although vascular contact with the 4th cranial nerve (which innervates the superior oblique muscle) has been identified and proposed as a pathophysiologic mechanism in some patients,[9] and other structural pathologies have been rarely described.[6] Where treatment of SOM is required, carbamazepine or carbamazepine derivatives are most commonly used.[10] Other medications that have also been used empirically include gabapentin, pregabalin, baclofen, memantine, phenytoin, clonazepam, and beta-blockers (topically or orally).[6,7] In some patients with refractory symptoms, extraocular surgery has been

undertaken.[11] In addition, microvascular decompression has been reported as a successful treatment in some individuals with neurovascular contact.[12]

Visual Vertigo

Dizziness in some patients may be triggered by visual motion or complex visual environments, for example, traffic on a street, a grocery store aisle, or watching fast-moving sports on television. These patients do not describe visual symptoms, but rather a sense of disequilibrium, dizziness, or vertigo in response to these triggers.[13] Patients with visual vertigo often have a preceding vestibular injury (such as vestibular neuritis), vestibular migraine, or an underlying disorder of balance, for example, cerebellar disease.[14,15] Increased visual dependence in response to the underlying disorder is a unifying feature in these patient groups and is thought to be an important pathophysiological trigger for the development of visual vertigo.[15] Similar to other patient groups with chronic dizziness, patients with visual vertigo often have comorbid anxiety or depression, and episodes of visual vertigo can sometimes be erroneously attributed to panic attacks.[13] The presence of an underlying balance disorder along with sensitivity to multiple types of visual motion can help differentiate visual vertigo from panic attacks or specific phobias. In addition, an ophthalmology assessment may be useful to rule out an ocular disorder.

Visual vertigo can be treated with a multifaceted approach. Vestibular physical therapy is an essential component of treatment and should focus on underlying balance or dizziness alongside visual desensitization approaches. Visual desensitization typically includes exposure to optokinetic stimuli while the patient is stationary, eventually progressing to exposure while in active motion.[13] Some of these exercises can now be completed by patients remotely, using appropriate online video material. Pharmacologic therapies may be helpful for the treatment of the underlying disorder (eg, vestibular migraine or Meniere's disease) or managing comorbid anxiety or depression that can exacerbate symptoms.

Medial Longitudinal Fasciculus Brainstem Syndromes

The medial longitudinal fasciculus (MLF) is a paired white matter tract close to the midline in the dorsal brainstem linking several brainstem nuclei, and injury to this tract can result in several clinical manifestations relevant to otologists, including an internuclear ophthalmoplegia (INO), skew deviation, spontaneous nystagmus, and abnormalities of the vertical vestibulo-ocular reflex (VOR).

The MLF links the horizontal gaze centers in the pons (sixth cranial nerve nucleus and the paramedian pontine reticular formation) and the third cranial nerve nucleus in the midbrain (including the medial rectus subnucleus).[16] In the normal state, horizontal gaze in one direction is triggered by the ipsilateral pons, and signals are transmitted through decussating fibers to the contralateral MLF, and in turn, the contralateral 3rd cranial nerve nucleus in the midbrain, allowing for the contraction of the ipsilateral lateral rectus muscle and contralateral medial rectus muscle, thus facilitating conjugate horizontal gaze.[17] A lesion of the MLF can cause disruption of this pathway, resulting in an INO—defined as an abnormality of adduction (slowed adduction, limited range of adduction, or complete failure of adduction) during horizontal gaze (Video 2). An INO is described as right or left according to the abnormal adducting eye (which is ipsilateral to the MLF lesion) and may be associated with nystagmus of the abducting eye. On bedside examination, a subtle INO may be most apparent as an adduction lag during large horizontal saccades (slowness of the adducting saccade relative to the abducting saccade).[16]

An INO can cause a variety of symptoms or may indeed be asymptomatic. Patients may describe double vision, blurred vision, oscillopsia, visual lag, and difficulty tracking moving objects.[18] In the clinical history, patients may find these symptoms hard to differentiate and simply describe dizziness when looking in a particular direction. Dizziness triggered by horizontal gaze can thus often be confused with head-motion-induced dizziness, for example, a patient with an INO may describe dizziness triggered when vehicles approach from the right while driving, making it important for the physician to try and differentiate head movement from eye movement triggers in the history. In some cases, an INO can actually be associated with typical vestibular dizziness/vertigo and spontaneous nystagmus (with head-movement-independent oscillopsia) acutely, and an abnormal vertical VOR (with head-movement dependent oscillopsia) which can persist over time, because the vertical semicircular canal pathways (linking the vertical semicircular canals to the vertical gaze centers in the midbrain) also travel within the MLF and can be affected by lesions in this tract.[19] A skew deviation can commonly occur in association with an INO because the utricle pathways (contributing to vertical alignment of the eyes through the physiologic ocular tilt reaction responding to head tilt) also travel within the MLF.[16]

The most common causes of an INO are stroke (most frequently unilateral INO) and multiple sclerosis (most frequently bilateral INO).[20] A myriad of other causes have also been described including trauma, neoplasms, infection, neuroimmune disorders, iatrogenic injury, vasculitis, and vascular malformations.[20] There is no specific treatment for INO, so management should focus on identification and treatment of the etiologic cause. Some patients report that diplopia improves when wearing spectacles with thick frames, which can block the image from the abducting eye in horizontal gaze. This can also mitigate any oscillopsia associated with the abducting nystagmus.

AUTONOMIC DIZZINESS

Standing positions can be challenging for patients with cardiovascular autonomic dysregulation and may cause various symptoms including dizziness. Within a few minutes after standing up, gravity shifts about 500 mL of intravascular fluid (blood) down below the heart, mainly in the splanchnic and large muscular vasculatures. After approximately 20 to 30 minutes of standing, plasma volume decreases due to fluid shift into the interstitial spaces by about 450 mL, which intensifies orthostatic stress.[21] Orthostatic stress can cause various hemodynamic abnormalities with inadequate blood flow to the brain and other organs. The decreased perfusion results in dizziness, vision changes, tinnitus, near-fainting, and syncope.[22] Patients can also experience nausea, sweating, flushing, and palpitations due to compensatory sympathetic hyperactivation.

Orthostatic hypotension (OH) is defined as a drop in systolic blood pressure (BP) by at least 20 mm Hg (diastolic by 10 mm Hg) within 3 minutes of standing (**Fig. 1**A).[23] OH is not always symptomatic, and management of this condition should be based on symptoms, not BP measurements only[24]; there is no association between the severity of BP changes or absolute standing BP and clinical symptoms.[25] Nonetheless, a study involving patients with Parkinson's disease reported a standing mean arterial pressure of 75 mm Hg as an objective threshold for pharmacologic interventions.[26]

Postural orthostatic tachycardia syndrome (POTS) is a heterogeneous autonomic syndrome with exaggerated postural tachycardia and relevant orthostatic symptoms. An exaggerated postural tachycardia is defined as a sustained increase in heart rate of 30 or greater beats per minute (bpm) without OH within 10 minutes of standing (>40 bpm for 12- to 19-year-old populations) (**Fig. 1**B).[23] An exaggerated postural

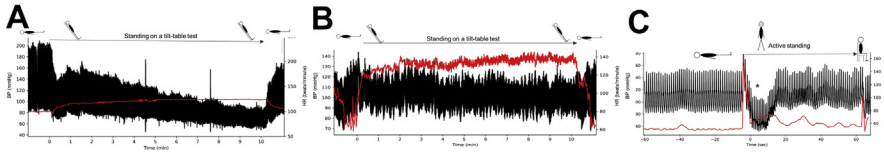

Fig. 1. Various hemodynamic changes to orthostatic stress measured by continuous beat-to-beat blood pressure and heart rate monitoring. A tilt-table test on a patient with a neurodegenerative disease revealed supine hypertension and orthostatic hypotension that persisted during the test (*A*). A patient with postural orthostatic tachycardia syndrome showed a sustained increase in heart rate without a significant drop in blood pressure during a 10-minute-long tilt-table test (*B*). A person with initial orthostatic hypotension may show a transient drop in systolic blood pressure by 40 mm Hg or more within 15 seconds of *active* standing (*C*) (marked with *). An active standing test is not sensitive, but specific, for initial orthostatic hypotension. Please note a different time scale on the x axis (*C*). Black lines show continuous beat-to-beat blood pressure measurements with upper and lower margins indicating systolic and diastolic blood pressure, respectively. Red lines indicate heart rate.

tachycardia alone is not diagnostic of POTS. Chronic orthostatic intolerance for at least 3 months (ideally 6 months) that improves significantly upon sitting/lying down should be present for the diagnosis.[27] Possible immunologic triggers such as infection often precede POTS. Patients may have autonomic symptoms of sudomotor (sweating), bladder, and/or gastrointestinal involvement. Other associated conditions include hypermobile joints syndrome, chronic fatigue syndrome, gastrointestinal dysmotility, and migraine. Their causal relationship with POTS remains unclear.

Patients with initial OH, defined as a transient drop in systolic BP by >40 mm HG and/or in diastolic BP by >20 mm Hg within 15 seconds after standing report short-lived symptoms right after standing (**Fig. 1**C).[28] Because the BP changes are transient, symptoms often present right after standing up. Initial OH is considered benign in adolescents and young adults. Patients with autonomic neuropathy or on BP-lowering agents may report similar symptoms.[29] Patients with mild or intermittent autonomic disorders may become symptomatic only in situations that amplify orthostatic stress or cause shifts and stasis of plasma volume. Common scenarios include prolonged standing, alcohol, BP-lowering medications, exposure to warm weather, postprandial period, and exercise.

CERVICOGENIC DIZZINESS

CGD, formerly referred to as cervical vertigo, is described as a nonspecific sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck, but not necessarily the illusion of motion.[30] It is characterized by the presence of disequilibrium, lightheadedness, imbalance, unsteadiness, disorientation, visual disturbances, and neck pain and may be accompanied by a headache.[31–35] Dizziness is described as lasting minutes to hours, lower in intensity, episodic, and rarely involves true vertigo compared with dizziness from the vestibular system.[35] Symptoms can result in anxiety, depression, and inability to perform activities of daily living and occupation duties, which contributes to decreased quality of life.[36]

The pathophysiology of CGD remains to be fully understood. In many cases of CGD, there is a diagnosis of whiplash-associated disorder, spondylosis, degenerative disc disease, inflammation, degeneration, or mechanical dysfunction of the cervical

spine.[31,33,37] Adverse changes in proprioceptors in the cervical spine may affect the sensorimotor control of gaze stabilization, eye-head movements, and postural stability.[37] These changes result in a sensory mismatch between the vestibular, somatosensory, and visual afferent inputs.[35,37] The VOR and cervico-ocular reflex work together in conjunction to stabilize the visual image on the retina creating clear vision with movement.[38]

CGD is often a diagnosis of exclusion secondary to a lack of appropriate diagnostic tests and the potential overlap into other etiologies.[31] A clinician must perform a proper examination and demonstrate sound clinical decision-making to lead to a diagnosis of CGD. The examination must assess for other causes of dizziness including the peripheral or central vestibular system and visual system.[35] A clinical cervical assessment includes ligament stability, vertebrobasilar blood flow, manual spinal examination of facet joint dysfunction, palpation for segmental tenderness, traction, strength, clinical tests, proprioception, and postural alignment (**Fig. 2**; **Table 1**).[33,39,40]

Treatment for CGD includes both orthopedic and vestibular physical therapy. Manual orthopedic techniques, including Maitland's passive mobilizations and Mulligan's sustained natural apophyseal glides, address hypomobility of the cervical spine and produce a significant improvement in the frequency of dizziness.[35,36,41,42] The performance of strengthening and neuromuscular recruitment of the deep cervical flexors combined with cervical proprioception enable fine motor movement patterns of the upper cervical spine which leads to an improvement in range of motion and activity with a decrease in dizziness and pain.[39]

Despite the paucity of studies performing vestibular physical therapy and orthopedic physical therapy, Wrisley and colleagues[43] and Lystad and colleagues[42] support the combination of both therapies to fully address all the patient's symptoms. Future research investigating the benefits of orthopedic and vestibular physical therapy is imperative for efficient diagnosis and treatment of CGD.

MEDICATION-INDUCED DIZZINESS

Dizziness is a common side effect of many medications, and misuse of these medications is associated with an increased risk of falls in older adults.[44] In an effort to

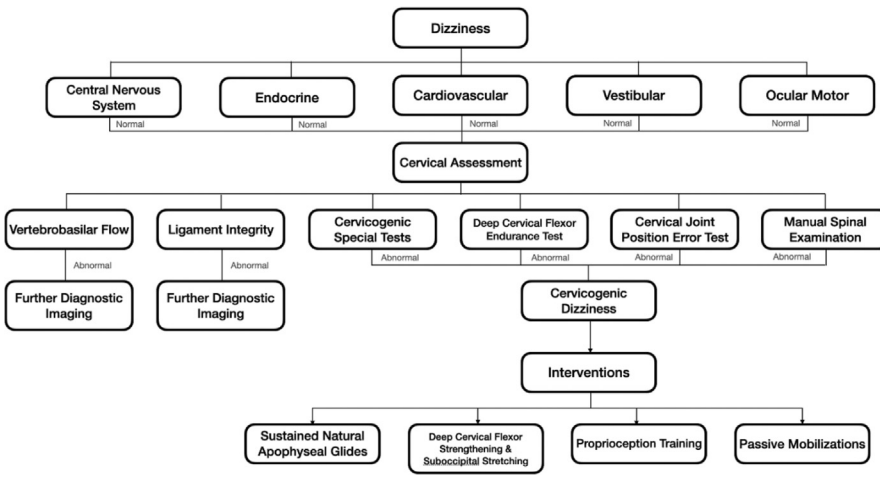

Fig. 2. Clinical assessment and management of cervicogenic dizziness.

Table 1
Clinical tests for cervicogenic dizziness

Test	Purpose	Performance	Abnormal	Specificity	Sensitivity
Alar Ligament Stress Test[69]	Alar ligament integrity at the atlas and axis.	Assess in the supine position. The clinician stabilizes the spinous process of C2 and passively performs side bending bilaterally.	The C2 spinous process has a lack of movement.	96%–100%	69%–72%
Sharp-Purser Test[39]	Transverse ligament integrity to maintain the position of the odontoid process on the atlas.	Assess is in the seated position. The clinician places their thumb on the C2 spinous process. Their other hand on the patient's forehead. The patient flexes their cervical spine while the clinician pushes on the forehead posteriorly.	A clunk or click with symptoms of dizziness.	96%	69%
Vertebrobasilar blood flow[39,70]	Assess blood flow in the vertebral artery	Assess in the seated position for signs of vertebrobasilar insufficiency during symptom provocation. Patient performs sustained end range rotation or end range rotation with extension. Can be performed passively in the supine position.	Patient complains of dizziness, diplopia, dysarthria, or drop attacks.	76%–100%	0%–57%
Smooth Pursuit Neck Torsion Test[71]	Cervicogenic dizziness	Patient is seated on a swivel chair with their neck in a neutral position. The clinician holds their head stable. The patient performs smooth pursuits in the neutral position, body rotation 45° right with neutral head, and body rotation 45° left with neutral head.	Abnormal pursuits or symptoms of dizziness.	91%	90%

(*continued on next page*)

Table 1
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Test	Purpose	Performance	Abnormal	Specificity	Sensitivity
Head-Neck Differentiation Test[72]	Cervicogenic dizziness	Patient is seated on a swivel chair with neck in a neutral position with their eyes closed. Clinician holds their head stable while the patient moves their body side to side 45° from center at 60 beats per minute.	Patient has symptoms of dizziness.	90%	N/A
Cervical Torsion Test[72]	Cervicogenic dizziness	Patient is seated on a swivel chair with neck in a neutral position with their eyes closed. Clinician holds their head stable while the patient moves their body side to right 90° for 30 s. Repeat with the body rotating to the left 90° for 30 s.	Patient has symptoms of dizziness.	98%	N/A
Cervical Joint Position Error Test[39]	Cervical proprioception	Patient is seated with a head laser 90 cm from a bullseye target. Patient will either rotate, extend, or flex their neck with their eyes closed and then return to the bullseye.	Patient has a difference of >4.5° from the bullseye.	N/A	N/A
Deep Cervical Flexor Endurance Test[73]	Strength of deep cervical flexor muscles (longus capitis and longus colli) during craniocervical flexion.	Patient is in the supine position. The clinician passively moves the patient into craniocervical flexion with the head lifted 2 cm from the surface. Patient is required to maintain this position independently as long as they are able.	<39.9 s for men <29.4 s for women	N/A	N/A

improve medication selection and to minimize adverse drug events among the elderly, the American Geriatrics Society (AGS) began publishing the Beers Criteria of Potentially Inappropriate Medication (PIM) use.[45] This is an evidence-based list of PIMs that should be avoided in most older adults, and incorporation of the AGS Beers Criteria into clinical prescribing patterns would likely help mitigate medication-induced dizziness and falls.

As discussed, there is an extensive list of medications that could cause dizzy symptoms. In general, medications that are more prone to this side-effect are antidepressants, antiseizure drugs, antihypertensive medications, antibiotics, anti-inflammatory medications, and diuretics.[46,47] Antidepressants are a common offender. The abrupt discontinuation of selective serotonin reuptake inhibitors (SSRIs) frequently causes dizziness. Paradoxically, SSRIs have also been shown to relieve dizziness in patients with major or minor anxiety disorders, major depressions, and undifferentiated somatoform dizziness.[48] For a detailed table of common medications that may induce dizziness or vertigo as an adverse drug reaction, please refer to Chimirri and colleagues.[46] In order to minimize incidences of medication-induced dizziness, medication reconciliation should be performed regularly during provider outpatient visits, and drugs that do not offer benefit to the patient should be discontinued in a timely fashion.

OTHER FORMS OF NONVESTIBULAR DIZZINESS

Symptoms of metabolic dysregulation may include dizziness. Hyperventilation is defined as rapid, deep breathing exceeding metabolic needs.[49] The process of overbreathing increases serum pH and lowers the concentration of ionized calcium. Subsequent arterial vasoconstriction reduces both cerebral and inner ear circulation, thereby decreasing the tissue oxygenation and inducing lightheadedness. Another metabolic-related condition is hypoglycemia. Glycemic variability is common in people who use insulin or take certain tablets to reduce high blood sugar, and this patient population is more vulnerable to hypoglycemia.[50] Because the brain relies on blood sugar as its primary source of energy, hypoglycemia can cause neuronal death.[51] Signs of hypoglycemia include headache, fatigue, anxiety, confusion, cold sweats, pale face, and dizziness. Carbon monoxide has also been reported to cause dizziness. Lumio first described hearing loss and vertigo from chronic carbon monoxide poisoning in 1948, which may have arisen from neurotoxicity, lactate acidosis, and cochlear and vestibular nerve damage.[52] Although the exact pathophysiology remains unclear, carbon monoxide has also been shown to affect cochlear electrophysiology and blood flow.[53,54]

In addition to metabolic conditions, dizziness may also manifest from autoimmune thyroid diseases. Hypothyroidism may result in low BP and bradycardia leading to dizziness. Conversely, hyperthyroidism may cause heart palpitations, arrhythmia, and lightheadedness. These symptoms typically resolve with medical management of the underlying thyroid disease. Interestingly, thyroid diseases such as goiter, hypothyroidism, and hyperthyroidism are associated with Meniere's disease[55]—a medical condition clinically diagnosed by recurrent vertigo attacks and cochlear symptoms of fluctuating hearing loss, tinnitus, and/or aural fullness.[56] As such, patients with a thyroid disorder and dizziness complaints should also be evaluated for peripheral vestibular dysfunction.

During a clinic visit, an alcohol drinking history should be solicited. Alcohol consumption can cause dizziness through central, peripheral, and autonomic mechanisms. Ethanol induces cerebellar dysfunction and ataxia by disrupting molecular neurotransmission, inducing dendritic regression, provoking neuronal inflammation/

toxicity, and modifying brain input functionality.[57,58] Alcohol can also induce peripheral neuropathy, resulting in sensory ataxia and alcohol-related dizziness/imbalance.[59] Positional alcohol nystagmus can also occur when alcohol diffuses into the cupula and transforms the semicircular canal into gravity-sensitive receptors because of the difference of specific gravities of alcohol and the vestibular endolymph.[60] Last but not least, alcohol ingestion may induce dehydration and orthostatic dizziness by inhibiting the release of antidiuretic hormones.[61] This effect is even more pronounced and prolonged in chronic alcoholics.

Although dizziness resulting from a primary cardiovascular disorder (eg, arrhythmia, carotid sinus reflex, defective heart valve, embolism, myocardial infarction, vascular anomaly, anemia) could also be classified as a form of nonvestibular dizziness, it is more frequently framed as presyncope (impending faint) or syncope among clinicians.[62] Seminal syncope studies have explicitly excluded patients with dizziness or vertigo.[63–65] We did not delve into cardiovascular-associated dizziness in the present article; however, this should be kept on the differential diagnosis whenever a patient presents with dizziness.

Disuse disequilibrium, otherwise known as deconditioning, is a common cause of nonvestibular dizziness.[66,67] Elderly adults are more prone to disuse disequilibrium because of a greater incidence of immobilizing surgeries, chronic illnesses, near-falls or falls, and fatigue.[68] Unfortunately, fear of falling and disuse disequilibrium can perpetuate a vicious cycle of a sedentary lifestyle and reduce a patient's willingness to participate in physical rehabilitation. Although there are no large controlled studies of the management of disuse disequilibrium, this condition is generally responsive to gait and balance physical therapy.[66]

SUMMARY

Dizziness is a common symptom that arises from various disorders. Determining whether a patient is experiencing central, peripheral, or nonvestibular dizziness is an important aspect of history taking and physical examination. Here, we described many forms of nonvestibular dizziness including visual disturbances, autonomic dysfunction, CGD, medication-induced side effects, metabolic dysregulation, autoimmune diseases, and alcohol consumption. An understanding of each of these medical conditions will improve the likelihood of diagnosis and appropriate management of these patients.

CLINICS CARE POINTS

- Patients who demonstrate monocular oscillopsia, vertical or oblique diplopia, and vague symptoms of dizziness should be evaluated for superior oblique myokymia via downgaze examination during a fundoscopic examination or binocular video-oculography (to observe oscillations of the retinal structure). Treatment options include medications (eg, carbamazepine) or extraocular surgery for those who are refractory to medical management.
- Visual vertigo often presents as dizziness triggered by visual motion or complex visual environments. Patients with visual vertigo often have had preceding vestibular injury, vestibular migraine, or an underlying balance disorder. Treatment should be multifaceted and include vestibular physical therapy, visual desensitization, and pharmacologic therapies (if appropriate) for the underlying or comorbid disorders.
- Medial longitudinal fasciculus (MLF) brainstem syndromes occur when there is injury to a paired white matter tract linking the horizontal gaze centers in the pons and the third cranial nerve nucleus in the midbrain. Disruption of the MLF pathway can result in internuclear ophthalmoplegia, which may be described as diplopia, blurred vision,

oscillopsia, visual lag, difficult tracking moving objects, or dizziness when looking in a particular direction. Differentiation between dizziness induced by head movements versus eye movements is important for diagnosis and treatment of the underlying cause.

- Orthostatic hypotension is defined as a drop in systolic blood pressure by at least 20 mm Hg (diastolic by 10 mm Hg) within 3 minutes of standing and should be treated if a patient is symptomatic. Medications such as fludrocortisone, midodrine, and pyridostigmine are considered in patients who do not respond to maximized lifestyle modifications.
- Postural orthostatic tachycardia syndrome (POTS) is a heterogeneous autonomic syndrome with exaggerated postural tachycardia and chronic orthostatic intolerance. Patients with POTS are generally treated with graded exercise program, hydration, modified high-sodium diet, medications, and compression garments.
- Initial orthostatic hypotension is defined by a transient drop in systolic blood pressure by >40 mm HG and/or in diastolic BP by >20 mm Hg within 15 seconds after standing. Patients are typically treated with lifestyle modifications.
- To patients with autonomic dizziness, it is recommended to avoid their own triggers such as abrupt/prolonged standing, alcohol, antihypertensive medications, warm weather, large meals, and strenuous exercise.
- Cervicogenic dizziness is often a diagnosis of exclusion due to the lack of appropriate diagnostic tests and is a nonspecific sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck but not necessarily the illusion of motion. Treatment involves orthopedic and vestibular physical therapy.
- Antidepressants, antiseizure drugs, anti-hypertensive medications, antibiotics, anti-inflammatory medications, and diuretics are medications that commonly induce dizziness as a side effect. Medication reconciliation should be regularly performed for potentially inappropriate use (Beers Criteria), particularly in the elderly population.
- Hyperventilation, hypoglycemia, carbon monoxide poisoning, and thyroid disorders may induce metabolic or homeostatic dysregulation that can result in dizziness. Treatment of these underlying metabolic conditions generally resolves symptoms. Hypothyroidism and hyperthyroidism are also associated with an increased incidence of Meniere's disease (a form of peripheral vestibular dizziness); therefore, clinicians should also assess for peripheral vestibulopathies.
- Disuse disequilibrium is associated with a fear of falling, resulting in deconditioning. This medical condition is common among the elderly population and generally responds well to physical therapy exercises.

DISCLOSURE

The authors have nothing to disclose.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.otc.2021.05.017.

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