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Defining Dizziness

The definition of “dizzy,” according to Merriam-Webster, is “having a feeling of being whirled about and in danger of falling down” [1]. *Synonyms* for this specific term include, “aswoon, giddy, lightheaded, reeling, swimmy, vertiginous, whirling, and woozy.” *Related* words include, “faint, befuddled, confused, dazed, and groggy” [1]. This is *not* an all-encompassing list and may have cultural aspects of presentations as discussed later in this chapter [p. 833].

Patients may use the term “dizziness” to refer to various subjective experiences related to sensations of disturbed relation to one’s surroundings in space. It can be described as feelings of rotation or whirling, but also non-rotary swaying, weakness, or faintness [2]. Based on the ambiguous and subjective nature of the term dizziness, creating a differential for dizziness or operationalizing it as a useful diagnostic anchor is often not useful. Due to this lack of specificity, we recommend treating this term as a “complaint” rather than an actual “symptom.” This must be done without discounting the experience of the patient. Further exploring this complaint often yields evidence of more specific and clinically meaningful symptoms of vertigo, syncope/presyncope, and disequilibrium/lightheadedness [3].

To this end, the patient should be asked “*what do you feel when you experience these symptoms or what do you mean when you say that you are dizzy?*” The

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clinician should be empathic and allow the patient to describe their personal and subjective experience without interruptions to establish a shared mental model of understanding. More close-ended questions such as “*does the room spin?*” Or “*does the floor move?*” may help refine the complaint into one of the more specific categories. This is quite different from asking the patient if their vision became narrow, or if they are unable to recall events surrounding the event—questions that would indicate presyncopal and syncopal etiologies, respectively.

Separating Vertigo from Other Symptoms

As previously discussed in the introduction, vertigo is a possible symptom of a patient complaining of “dizziness.” Other possible etiologies of dizziness are syncope and disequilibrium. Refer to Table 5.1 for an overview.

Syncope

Syncope is defined as a transient loss of consciousness secondary to inadequate cerebral perfusion with oxygenated blood. When the patient experiences a presyncope, there are feelings of passing out without actual loss of consciousness. Most etiologies that cause syncope can cause presyncope [3, 4]. There are many causes of syncope as identified in Table 5.1. Unlike syncope, vertigo does not result in loss of consciousness, and the patient experiences the spinning sensation.

Disequilibrium

Disequilibrium is referred to unsteadiness or a sense of disconnect from the environment. Its pathophysiology is often related to a disconnect or miscommunication between the central nervous system (somatosensory cortex and the cerebellum), and the peripheral nervous system such as the visual system, sensory fibers, and proprioception. It is typically associated with aging and can be attributed to the aging vestibular system that shows a decrease in neural and sensory cells. Other implicated mechanisms include attrition of cerebellar Purkinje cells over time, neuronal and fiber loss in the extrapyramidal system, and a general decline in postural control and reaction time. Risk factors include visual impairments, muscle weakness, medications, and cognitive impairment among others. Patients may describe this as a “feeling like being on a boat” but deny spinning of the environment symptoms which differentiates it from vertigo [5]. The differential is very large as listed in Table 5.1.

Each of the aforementioned three symptoms of vertigo, syncope/presyncope, and disequilibrium/lightheadedness provide the much-needed organization to help operationalize the clinical encounter to yield a working diagnosis [6]. This allows for the determination of appropriate triage, ascertainment of clinical acuity, and accuracy of diagnosis. The three categories enable the clinician to more focally consider a more limited set of systemic medical and psychiatric differential diagnostic considerations. Table 5.1 and Fig. 5.1 provide the differential for these three symptoms. Each of these symptoms may also point to the underlying pathophysiology. For example, vertigo symptoms either occur due to a dysfunction in the vestibular apparatus (peripheral anatomy) or central nervous system lesions such as in multiple

Table 5.1 Common medical and psychiatric conditions that may present with the complaint of “dizziness.” Differentiation of this nonspecific term into either vertigo, syncope, or disequilibrium allows for a narrower medical and psychiatric differential diagnosis

Dizziness			
	Vertigo	Syncope	Disequilibrium
Description	False sense of motion or a spinning sensation	<i>Syncope</i> : loss of consciousness <i>Presyncope</i> : feelings of impending passing out <i>without</i> actual loss of consciousness	A feeling of disconnect from the environment or a sense of imbalance or unsteadiness
Medical conditions	<p><i>Peripheral:</i></p> <ul style="list-style-type: none"> • BPPV • Vestibular neuritis/labyrinthitis • Ménière’s disease • Acoustic neuroma <p><i>Central:</i></p> <ul style="list-style-type: none"> • Vestibular migraine • Cerebellar hemorrhage or ischemia • Vertebrobasilar insufficiency • Chiari I malformation • Head or neck trauma • Multiple sclerosis • Neoplasm • Postural-perceptual dizziness 	<ul style="list-style-type: none"> • Arrhythmia • Aortic stenosis • Pulmonary embolism • Cerebral stroke/bleed • Hypovolemia • Hemorrhage • Vasovagal • Hypoglycemia • Medications • Substance 	<ul style="list-style-type: none"> • TIA or stroke • Peripheral neuropathy • Demyelinating disease • Motor neuron disease • Vitamin B12 deficiency • Parkinsonism • Myelopathy • Normal pressure hydrocephalus • Infection • Medications • Substances
Psychiatric conditions in DSM-5-TR	<ul style="list-style-type: none"> • Substance/medication-induced anxiety disorder • Anxiety disorder due to another medical condition • Major or mild neurocognitive disorder due to: traumatic brain injury; substance-induced; vascular; due to another medical condition, and neurocognitive disorder not elsewhere classified • Cultural concepts of distress 	<ul style="list-style-type: none"> • Specific phobia • Functional neurological symptom disorder • Major or mild neurocognitive disorder with Lewy bodies • Cultural concepts of distress 	<ul style="list-style-type: none"> • Depersonalization/derealization disorder • Trauma spectrum disorders • Anxiety disorders • Depressive disorders • Somatic symptom disorder • Conversion disorder • Cultural concepts of distress • Major or mild neurocognitive disorder due to: vascular neurocognitive disorder, neurocognitive disorder due to another medical condition (any of the disorders listed in medical conditions), and neurocognitive disorder not elsewhere classified

BPPV benign paroxysmal peripheral vertigo

sclerosis [7, 8]. Similarly, the sudden loss of consciousness associated with syncope points to an underlying loss of blood flow to the brain which can be due to “pump” or “plumbing” issues, i.e., cardiac or vascular, respectively. A large number of

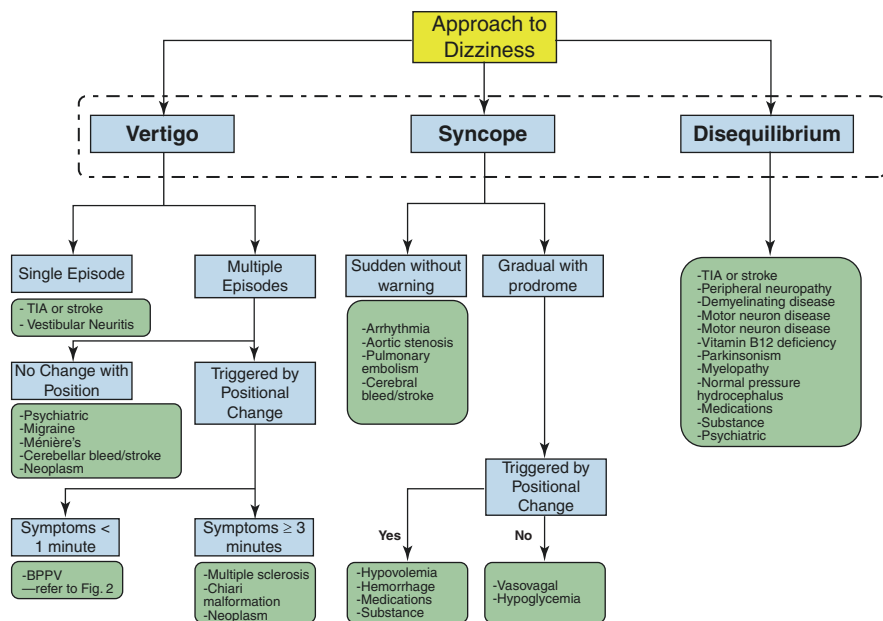


Fig. 5.1 Approach to dizziness involves clarification of the complaint into symptoms of syncope/presyncope; lightheadedness; and vertigo. Vertiginous symptoms are the most common presentation and can be approached using the algorithm based on the number of episodes, positional trigger, and duration of symptoms. Sudden syncopal episodes portend malignant etiology and immediate referral is needed. *TIA* transient ischemic attack, *BPPV* benign paroxysmal positional vertigo

etiologies such as mass, inflammatory, infectious, and metabolic etiologies can cause disruptions across these systems to result in disequilibrium.

Finally, as discussed later, the term “dizziness” may have other important clinical meanings when reviewed through a cultural lens. It is often a replacement term for various conditions such as depressive disorders, anxiety disorders, and trauma spectrum disorders.

As shown in Table 5.1, the differential for the three diagnostic categories is still broad; however, the majority of cases of dizziness end up with final diagnoses related to vertigo or psychiatric illness. One study showed that among 21,000 patients in Korea referred to a specialty dizziness clinic with dizziness complaints, 46.9% of patients were categorized into vertiginous etiologies with final diagnoses including benign paroxysmal positional vertigo (BPPV) 24.1%, vestibular migraine (10.2%), Ménière’s disease (7.2%), and vestibular neuritis (5.4%). In 20.4% of the patients, the symptoms were attributed to psychiatric illnesses [9]. We only focus on vertigo, including psychiatric and cultural etiologies of this complaint in this chapter, which covers the majority of causes of “dizziness” symptoms, but the reader is encouraged to refer to additional sources on approaches to syncope/presyncope and disequilibrium/lightheadedness symptoms [10, 11].

Vertigo

Vertigo is a false sense of motion or a spinning sensation. Pathology directly affects the vestibular apparatus or how the brain processes vestibular afferent information [7]. Both benign and more concerning etiologies, such as central nervous system (CNS) malignancies, ischemia, or hemorrhage, can result in vertigo [3]. When a patient complains of “dizziness,” the clinician’s challenge is to appraise the underlying quality of the symptoms. The feelings of “the world” or “the room” spinning during the episode qualify the presentation as vertiginous. In the following sections, we discuss the pathophysiology of vertigo. Following that, we review important systemic medical etiologies associated with vertigo. Next, we discuss psychiatric conditions that are associated with complaints of dizziness. Finally, we further highlight the challenge of dizziness complaints in the cultural context with specific cultural phenomena that have been described in the Diagnostic and Statistical Manual, 5th edition, text revision (DSM-5-TR) [p. 833]. Interestingly, other institutions have constructed a framework for dizziness including the National Institute of Mental Health’s (NIMH) Research Domain Criteria Initiative (RDoC), which categorizes multimodal perceptions such as a sense of self in space under the cognitive systems domain [12].

Pathophysiology

The vestibular apparatus is responsible for the sense of balance. This system detects head orientation and linear acceleration. The varying orientations of the hair cells that result from the different types of head movement lead to depolarization and hyperpolarization patterns that maintain equilibrium via afferent input to the vestibular nerve [7]. It also relies on input from the visual system and proprioceptive system of the head and neck. Mismatch of information from any of these systems, including integration of input in the CNS, can result in various vertigo and other dizziness symptoms. An in-depth discussion on the pathophysiology of balance and equilibrium can be found elsewhere [8].

Medical Specialty Approaches to Assessing Vertigo

Regardless of specialty, the goal of assessing a “vertiginous” complaint is to determine the correct diagnosis to establish acuity. Figure 5.1 provides an algorithm to help with identifying a specific diagnosis for symptoms of vertigo.

Medicine In medicine, including primary care, psychiatry, and the emergency department setting, the greatest emphasis is placed on identifying whether the presentation is due to a malignant etiology such as stroke, malignancy, or cardiac arrhythmia or a more benign condition such as benign paroxysmal positional vertigo (BPPV). The clinician may be less focused on the exact diagnosis, but rather ruling out conditions that require immediate interventions. Acute central dizziness variants would require acute evaluation for cerebellar stroke. Following ruling out acute concerns, primary care also typically focuses on common presentations such as BPPV.

Neurology Common conditions such as BPPV, and deadly etiologies such as strokes are often ruled out by primary care or emergency medicine physicians. Neurologists are therefore more concerned with exact neurological diagnosis of vertigo such as migraine or multiple sclerosis, or to determine the symptoms to be non-neurological.

Otolaryngology and Audiology Patients with hearing loss or tinnitus with vertigo symptoms are referred to otolaryngologists for diagnostic assessment if BPPV is ruled out by primary care. The otolaryngologists often focus on vestibular (peripheral) causes of vertigo or to determine the presentation as non-otological. This may result in a benign diagnosis such as vestibular neuritis or a more malignant process such as an acoustic schwannoma [8].

Differentiating Systemic Conditions that Present with Vertigo

When evaluating patients, it is important to consider medical, psychiatric, substance use, or medication causes. The most important first step is clarifying the ambiguous concern of *dizziness* and determining whether it is specifically *vertiginous* in nature. Most problems presenting with vertigo are episodic, and two symptoms associated with vertigo are *impulsion* and *oscillopsia*, which can help guide the physician from the vague complaint of dizziness to a more precise complaint of vertigo. *Impulsion* is defined as “a sensation that the body is being hurled or pulled in space,” while *oscillopsia* is “a visual illusion of moving back and forth” [13, p. 191]. Figure 5.1 outlines an algorithm to help the physician differentiate various etiologies that present with vertiginous symptoms. Conditions that cause vertigo are usually divided into *central* and *peripheral* causes. A *peripheral* etiology (88%) is most common and results from vestibular disease. The remaining 12% have vertigo related to a *central nervous system* problem [14]. In psychiatric practice, the presence of true vertigo should cause the psychiatrist to pursue evaluation for systemic medical causes in addition to traumatic brain injury, substance or medication use, or a withdrawal syndrome (Table 5.1).

Peripheral Causes of Vertigo

Benign Paroxysmal Positional Vertigo (BPPV)

Among all the causes of nystagmus, benign paroxysmal positional vertigo (BPPV) is the most common etiology and accounts for one-quarter of all patients complaining of dizziness [9]. It is relatively easy to diagnose based on history and examination as is described at greater length below. Given its prevalence, all clinicians should be familiar with three special maneuvers to diagnose BPPV.

Incidence/Prevalence

BPPV is the most common cause of vertigo and has a lifetime prevalence of 2.4% [15, 16].

Typical Findings in the HPI

As the name implies, the patient complains of brief (usually less than 1 min) episodes of vertigo that are triggered by position changes such as turning over, moving the head, sitting up, or bending over.

Typical Physical Exam Findings

The goal of the exam is to confirm either spontaneous or positional nystagmus with an otherwise non-focal neurological exam. The positional nystagmus can be triggered by the Dix-Hallpike maneuver or the supine roll [17].

The Dix-Hallpike maneuver is used to evaluate for any free-floating calcium carbonate crystals in the vestibular system, which disrupt the sensing mechanisms, causing vertiginous symptoms associated with BPPV [18, 19]. The sensitivity and the specificity for the Dix-Hallpike maneuver are 79% and 75%, respectively. This makes it a useful test for *ruling out* BPPV. If positive, the probability of BPPV is only 50%, but when this test is negative, the probability of the patient having BPPV is dropped to 8% [20]. To perform the Dix-Hallpike, have the patient sit with their head turned 45°, quickly and safely lower them in a supine position until their head is about 20° below the examination table. Observe ocular movements for nystagmus; the test is positive if there are a few seconds of latency, followed by transient (about 15–60 s) of upbeat, torsional nystagmus [21]. A video illustrating the Dix-Hallpike maneuver can be reviewed here: <https://youtu.be/8RYB2QIO1N4>

If the Dix-Hallpike maneuver is negative, the canalith (a crystalline particle derived from otoliths in the utricle of the inner ear) may be lodged within the lateral canals. To assess for this, utilize the supine roll test [21, 22]. This test begins with the patient sitting and then is brought to a supine position but the head is turned 90° and brought to a positive 20° above the table. Evaluate for nystagmus for 30 s; if negative for nystagmus, then rotate to midline for another 30 s, and then proceed to rotate their head in the opposite 90° direction [23]. A video illustrating the supine roll test can be reviewed here: <https://youtu.be/ns8XZ4rKiJc>

The Head Impulse, Nystagmus, and Test of Skew or HINTS exam is a brief, three-part, effective evaluation for determining if the vertiginous pathology is a central or peripheral etiology. To perform, have the patient sit across from the physician and rapidly have them rotate their head 20° in both rotational directions while trying to focus their gaze on the physician. If their gaze moves in the direction of rotation and is quickly followed by a saccade back to the physician, this is consistent with a *peripheral* cause. If their gaze is fixed then the test is *normal or concerning for a central etiology* [21].

One major component of the HINTS exam is *nystagmus*, which is defined as “rhythmic oscillation of the eyes” [13, p. 15]. If nystagmus is unidirectional when not looking straight ahead or can be suppressed by fixating on a point, then this is more consistent with a *peripheral* cause. Alternatively, if nystagmus changes direction, or is not suppressed by gaze fixation, this is more supportive of a *central* etiology [21].

The last component of the HINTS exam is a test of *skew*. Have the patient focus on a fixed point and cover and uncover one eye and repeat on the other side. If there is any rapid movement of the eye, it may be concerning for a central cause [21]. A video illustrating the HINTS exam can be reviewed here: <https://youtu.be/VwmrjYuvqtQ>

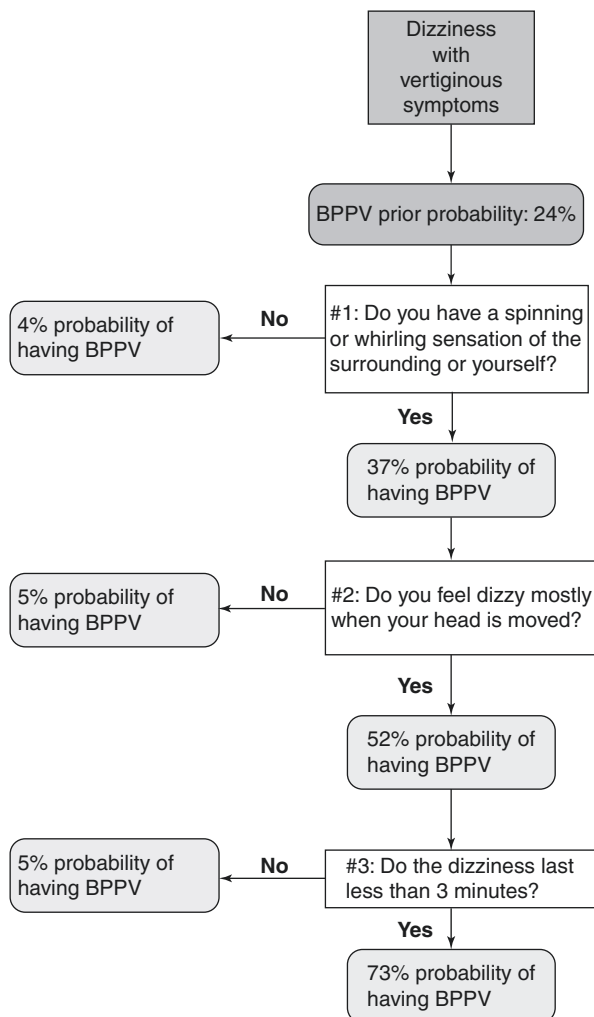
Typical Laboratory/Radiological Findings

There are no laboratory tests specific for vertigo. Magnetic resonance imaging (MRI) may be used to rule out vestibular lesions such as acoustic neuroma.

Screening Tools

An excellent screening questionnaire from Kim et al. can quickly help assess the presence or absence of BPPV and overall performs better than the Dix-Hallpike maneuver. It is based on three questions (see Fig. 5.2). A negative response to question #1 drops the probability of BPPV from 24% to just 4%. If the first question is positive, then the subsequent 2 questions may be used. Each additional positive question further improves the positive likelihood of BPPV. When all three questions are positive, there is a 73% probability of having BPPV [24]. Clinicians can utilize Fig. 5.2 to quickly conduct an assessment for BPPV.

Fig. 5.2 Three Question BPPV Questionnaire



Pitfalls/Differential Diagnosis

There is a broad differential of diagnoses to consider in addition to BPPV, but most are much rarer than BPPV. Peripheral conditions include otological disorders such as Ménière's disease, vestibular neuritis, posttraumatic vertigo, and inner ear lesions or masses. Central neurological conditions that may mimic BPPV include vestibular migraines, stroke, demyelinating diseases such as multiple sclerosis, vertebrobasilar insufficiency, or central positional vertigo. Stroke syndromes are nonrecurrent and may have other associated focal neurological findings on exam. Any central infection, as well as substance use or withdrawal may also mimic BPPV. The clue to diagnostic success is in the term “paroxysmal” in “BPPV,” where the symptoms are sudden, short-lived, and triggered by changes in the position of the head [25]. As outlined in Fig. 5.1, most of these conditions can be separated based on history and physical exam.

Vestibular Neuritis

Incidence/Prevalence

The annual incidence is 3.5 per 100,000 persons-year [26].

Typical Findings in the HPI

Patients typically present with sudden onset of severe vertigo with nausea, vomiting, and gait imbalance for hours to days that gradually improve over several weeks [27]. It can be preceded by a viral syndrome with a post-viral etiology being the leading theory for etiology [26]. Note that this differs from Ménière's disease since it is *not* associated with *hearing loss*, but can be uncommonly associated with a sense of aural “fullness” or tinnitus [27].

Typical Physical Exam Findings

With all peripheral etiologies of vertigo, the HINTS exam will display saccade back to a fixed point with head rotation or head impulse, unilateral nystagmus, or the resolution of nystagmus with gaze fixation, and fixed gaze with the test of skew [21]. While assessing gait and station, postural imbalance can be seen with the patient falling towards the affected ear [26]. Notably, focal limb weakness, sensory deficits, speech impairment, and memory loss are *not consistent* with vestibular neuritis [27].

Typical Laboratory/Radiological Findings

There are no specific laboratory test results. Magnetic resonance imaging (MRI) with contrast may be warranted if an acoustic neuroma or central etiology cannot be ruled out.

Screening Tools

There are no specific screening tools for vestibular neuritis.

Pitfalls/Differential Diagnosis

Differential diagnosis includes all peripheral etiologies for vertigo, including BPPV, Ménière's disease, or acoustic neuroma. CNS causes should be considered,

including transient ischemic attack, cerebellar or brainstem stroke, vestibular migraine, or demyelinating disease [26]. If symptoms are episodic in nature, associated with hearing loss, change in the level of consciousness or cognition, or have peripheral motor or sensory deficits, the etiology of vertigo is *unlikely* to be vestibular neuritis.

Ménière's Disease

Incidence/Prevalence

Ménière's disease has a lifetime prevalence of 0.2%, with women affected more than men [22].

Typical Findings in the HPI

The classic triad in Ménière's disease is *episodic vertigo* without triggers that last minutes to hours, *unilateral tinnitus* and aural fullness or pain, with reversible *sensorineural hearing loss*. About 25% of patients present with *all three* symptoms [15, 28]. Frequency of vertigo episodes is variable and symptoms may occur every few weeks to every few years. Ménière's disease is associated with progressive hearing loss, and as hearing declines, vertigo severity usually lessens [29].

Typical Physical Exam Findings

Head rotation/head impulse testing will be notable for saccades back to a fixed point, unilateral nystagmus with resolution of spontaneous nystagmus with gaze fixation, and fixed gaze with test of skew [21]. Hearing loss is usually unable to be assessed at the bedside if not advanced [13, p. 204].

Typical Laboratory/Radiological Findings

There are no specific laboratory tests. MRI with contrast may be needed to differentiate if there is a peripheral mass or central etiology that is contributing to the patient's vertigo. If an MRI is obtained, endolymphatic hydrops, an increase in the volume of labyrinth endolymph, can be seen [30]. Formal audiology consultation with audiometry may need to be obtained to fully assess and monitor hearing deficits.

Screening Tools

There are no specific screening tools for Ménière's disease.

Pitfalls/Differential Diagnosis

As discussed with vestibular neuritis, peripheral causes of vertigo should be at the forefront of the differential. These include BPPV or acoustic neuroma. Other etiologies that affect the inner ear such as temporal bone fractures, infection, and otosclerosis can have a similar presentation as well [30]. The classic triad of symptoms is important but only present in a quarter of patients with Ménière's disease.

Acoustic Neuroma/Vestibular Schwannoma

Incidence/Prevalence

Incidence is 1–20 per million persons-year with the mean age at diagnosis being 50–55 years old [31]. Vertigo eventually develops in 8% of patients with acoustic neuroma [32].

Typical Findings in the HPI

Initial presentation is primarily insidious unilateral hearing loss, but poor balance, vertigo, or tinnitus can be the presenting symptom. Most cases of vestibular schwannomas are sporadic but some are genetically related with two disorders being notable, including neurofibromatosis type 2 (NF2) and schwannomatosis. NF2 is known to cause bilateral vestibular schwannomas but also additional schwannomas located in cranial and spinal nerves as well as ocular involvement including cataracts. About 25% of vestibular schwannomas are incidentally found when imaging is obtained for unrelated symptoms [32].

Typical Physical Exam Findings

Unilateral sensorineural hearing loss is common; facial palsy or sensory loss, abnormal corneal reflex, imbalance, and/or ataxia can also be seen [32].

Typical Laboratory/Radiological Findings

Cerebrospinal fluid (CSF) protein is elevated in most patients with the range typically being 50–200 mg/dL [13, p. 205]. The tumor is typically found in the internal auditory canal and/or cerebellopontine angle [32]. Gadolinium-enhanced MRI is the imaging modality of choice with a sensitivity and specificity of 96% and 88.2%, respectively [33].

Screening Tools

There is no screening test for acoustic neuromas.

Pitfalls/Differential Diagnosis

If cranial nerve V or VII impairment, or ataxia is present on examination, then other etiologies should be evaluated including other types of schwannoma, meningioma, or metastatic cancer. The progressive, continuous quality of vertigo, if present, differentiates it from other peripheral causes [32].

Central Causes of Vertigo

Vestibular Migraine

Incidence/Prevalence

Lifetime prevalence is 1% and affects women more than men with women 3.3 times more likely to suffer from vestibular migraine [34]. Vertigo is a hallmark symptom.

Typical Findings in the HPI

Vestibular migraine usually presents as vertigo with headache, nausea, emesis, photophobia, and/or phonophobia. Notably, headaches may be absent or mild. Episode length is variable from seconds to days. To formally make the diagnosis, there need to be five or more attacks with episodic or fluctuating vestibular symptoms, a history of migraines, and other associated migraine symptoms with at least half of the attacks [15, 28]. Some notable features can include triggers of vertigo with moving visual scenery or susceptibility to motion sickness, but vestibular migraine is usually *not* associated with positional changes [35].

Typical Physical Exam Findings

Examination is usually unremarkable when the patient is asymptomatic. During an episode, the patient can have persistent bilateral horizontal positional nystagmus, as well as spontaneous nystagmus worsened by fixation of gaze. Other rare findings on examination may include imbalance or hearing loss [35].

Typical Laboratory/Radiological Findings

There are no typical laboratory or radiographic findings.

Screening Tools

Celebisoy et al. published the eight-question Vestibular Migraine Diagnosis Questionnaire, which is a useful screening tool [34]. For specifics regarding this questionnaire, please see our references. With certain answer choices, the sensitivity was 82.8% with a specificity of 83.9% [34]. With a prevalence of 1%, this questionnaire has a positive likelihood ratio of 5.19 and a negative likelihood ratio of 0.20. When the questionnaire result is negative, the chance of this condition is close to 0%. If positive, the posterior probability is only 5% and clinically not helpful. Therefore, this test is most helpful in ruling out vestibular migraine.

Pitfalls/Differential Diagnosis

Ménière's disease has a significant overlap with vestibular migraine, which can make diagnosis challenging. Both conditions may present with transient vertigo symptoms with aural fullness; however, only Ménière's disease presents with hearing loss. Other considerations for differential diagnosis include BPPV, vertebrobasilar transient ischemic attack (TIA), vascular compression of the eighth cranial nerve, and acoustic schwannoma [35].

Transient Ischemic Attack

Incidence/Prevalence

The annual incidence rate of transient ischemic attacks (TIAs) in the United States is 1.1 per 1000 person-years [36]. While there are an estimated 400,000 individuals in the United States diagnosed with TIAs each year, the true number of cases is estimated to be around five million. This difference is due to under-reporting due to many patients not seeking medical attention [37]. Incidence or prevalence of vertigo with

TIA is unknown. A nonspecific dizziness symptom is present in 3.2% of patients with stroke or TIA. Isolated dizziness, i.e., without other focal findings, is much rarer and only present in 0.7% of patients with TIA or stroke. Patients presenting with vertigo symptoms do not have higher odds of having TIAs or stroke when compared to the nonspecific complaint of dizziness. Patients with imbalance (disequilibrium) symptoms are four times more likely to have TIAs or stroke when compared to patients without disequilibrium symptoms [38]. Disequilibrium symptoms result from motor impairment of the lower extremities or cerebellar lesions. The Rapid Arterial Occlusion Evaluation (RACE) scale, which assesses leg weakness or paralysis resulting in disequilibrium, is part of some prehospital stroke assessments [39].

Typical Findings in the HPI

TIAs can present with a wide range of symptoms, including but not limited to isolated vertigo, diplopia, aphasia, dysarthria, limb weakness or sensory loss, nausea, vomiting, or ataxia [15, 40]. To be deemed a transient ischemic attack, neurologic symptoms need to occur secondary to retinal and/or focal brain ischemia and resolve fully, which usually occurs within 1 h. Additionally, patients need to have no evidence of acute infarction [41].

Typical Physical Exam Findings

Examination findings are highly variable and are determined by specific vascular involvement. Cranial nerve palsies, sensorimotor deficits, alteration in level of consciousness, aphasia, and ataxia can be seen on physical exam [40, 41].

Typical Laboratory/Radiological Findings

There are no specific laboratory or radiographic findings with TIA. The main goal of brain imaging is to exclude acute ischemic or hemorrhagic stroke. Head Computer Tomography (CT) is helpful to rule out bleeding or subacute strokes. MRI is the most sensitive modality for excluding acute and subacute strokes. The diffusion-weighted imaging (DWI) of MR is 90% sensitive and 97% specific for acute ischemic strokes [42].

Screening Tools

There are many screening tests for TIA and stroke symptoms, with one of the most common being the Cincinnati Prehospital Stroke Severity Scale (CPSSS), which examines facial droop, arm drift, and speech. Using a cut-off of 2 on a scale of 1–4, CPSSS is 70.0% sensitive and 86.8% specific for predicting large vessel occlusion [43].

Pitfalls/Differential Diagnosis

Evaluation of serum glucose is paramount since hypoglycemia can mimic TIA symptoms [41]. If symptoms continue to evolve, then there should be a strong consideration of stroke. Additional differential diagnoses that should be considered are seizures, migraines, multiple sclerosis, neoplasm, central nervous system infection, or peripheral causes of vertigo [44].

Chiari Malformation Type I

Incidence/Prevalence

Chiari Malformation Type I (CM-I) occurs among 1 in a 1000 live births with a male to female ratio of 1.3:1. Vertigo is an unusual presentation of CM-I [45]. In one study of patients with various otological complaints (including vertigo) in an otolaryngology clinic, 0.9% of the patients were found to have Chiari malformation [46].

Typical Findings in the HPI

The most common symptom is pain in the occipital region that is exacerbated by the Valsalva maneuver. Patients with CM-I may also experience unspecified dizziness, vertigo, syncope, and disequilibrium. Patients may also complain of dysphagia and tinnitus. Additionally, patients may develop progressive muscular weakness in the extremities, as well as bowel and bladder dysfunction [47]. Patients can also be asymptomatic with incidental findings of CM-I on MRI [45].

Typical Physical Exam Findings

If there is autonomic dysfunction, bradycardia may be seen. Patients may display ataxia, dysmetria, impaired balance, limb sensorimotor dysfunction, and impaired swallowing [47]. Positional nystagmus is common. Vertigo may be induced by neck flexion, extension, or rotation similar to findings in cervical vertigo resulting in a delayed onset of positional nystagmus on the Dix-Hallpike maneuver [48].

Typical Laboratory/Radiological Findings

There are no standard laboratory findings for Chiari Malformation Type I. MRI shows greater than 5 mm tonsillar descent and herniation (6 mm in children) through the foramen magnum. These cut-offs are 92% sensitive and 100% specific [49].

Screening Tools

There are no routine screening tools for Chiari Malformation Type I.

Pitfalls/Differential Diagnosis

Differential diagnosis includes other Chiari malformations (Types II–IV), tonsillar ectopia, basilar invagination, and idiopathic intracranial hypertension [47].

Primary Central Nervous System Tumors

Incidence/Prevalence

The overall incidence of CNS tumors is 19 per 100,000 persons-years and was noted to have increased by about 3% between 2000 and 2012 [50]. Due to heterogeneity of CNS masses, incidence and prevalence of vertigo secondary to CNS tumors are unknown, though isolated vertigo or dizziness is rare.

Typical Findings in the HPI

History of present illness varies depending on the type of CNS tumor and its regional involvement, with symptoms typically being divided into general and focal. General

symptoms usually cause pathology from pressure on the cerebral or spinal tissue. This mass effect may manifest as morning headaches, nausea and emesis, seizures, cognitive and personality changes. Focal symptoms include ataxic gait, impaired balance, vertigo, unspecified dizziness, vision changes, cranial nerve deficits, aphasia, and agnosia [51].

Typical Physical Exam Findings

Neurologic examination is paramount, which is variable and dependent on tumor location. Papilledema can be seen due to the mass effect. Visual field defects, other cranial nerve deficits, and focal findings may be present [51].

Typical Laboratory/Radiological Findings

When CNS mass or tumor is suspected, the next step is gadolinium-enhanced MRI. The likelihood ratios depend on the type of underlying tumor type and size. 18F-fluorodeoxyglucose (18F-FDG) positron emission tomography (PET) may have a role when primary CNS lymphoma is suspected [52].

Screening Tools

Due to heterogeneity of CNS masses, there are no screening tools.

Pitfalls/Differential Diagnosis

There is a broad differential but may be narrowed based on a patient's age, immunocompetency, and risk of peripheral malignancies with avidity for CNS metastasis. The greatest branch point is malignancy vs. infection [53].

Cerebellar Hemorrhagic or Ischemic Infarction

Incidence/Prevalence

There are approximately 600,000 strokes in the United States each year. Of these, 2–3% are cerebellar ischemic or hemorrhagic strokes [54]. Vertigo presents in 48% of patients with posterior circulation ischemia [13, p. 373].

Typical Findings in the HPI

With hemorrhagic stroke, there is usually a sudden headache with nausea, vomiting, or vertigo. This is followed by an ataxic gait and decreased level of consciousness. Ischemic infarction has an array of symptoms including nystagmus, limb ataxia, and cranial nerve deficits, but there is a significant overlap with hemorrhagic stroke symptoms, which also include vertigo, nausea, and emesis [55].

Typical Physical Exam Findings

The neurologic exam is imperative. Findings may include small and sluggish pupils, and/or gaze palsy towards the affected side. Patients can have ipsilateral limb ataxia and fall towards the side with pathology. Additionally, dysarthria, nystagmus, and sensory deficits can occur. It is crucial to have the patient ambulate, since impairments in gait are usually the earliest neurologic sign, especially with a hemorrhagic etiology [55].

Typical Laboratory/Radiological Findings

There are no specific laboratory findings, but if cerebrospinal fluid is obtained, it is usually bloody with hemorrhagic stroke [13, p. 209]. Head CT can help with assessing for hemorrhage but its sensitivity diminishes when a posterior fossa stroke is a concern. Brain MRI with DWI sequences is the imaging of choice to exclude posterior fossa strokes [56].

Screening Tools

The HINTS exam should be the primary screening tool used to assess patients presenting with vertigo where there is a concern for posterior circulation infarction. If there is a normal head impulse test, a positive test of skew, or direction-changing nystagmus then there is a 100% sensitivity and 96% specificity for infarction [57]. The Cincinnati Prehospital Stroke Scale (discussed in section “Transient Ischemic Attack” above) is also valuable for cerebral strokes, although it is insensitive for posterior fossa strokes, including cerebellar.

Pitfalls/Differential Diagnosis

All causes of central etiologies of vertigo should be considered on the differential when the HINTS exam is positive. These include vestibular migraine, multiple sclerosis, midbrain or cerebral strokes, or vertebrobasilar insufficiency [21]. Indolent symptoms that develop over weeks to months suggest a cause other than stroke since it is an acute illness. It is important to assess for gait impairment since this is usually one of the initial signs of cerebellar pathology, which would warrant neuroimaging [55].

Multiple Sclerosis

Incidence/Prevalence

Approximately 2.8 million people worldwide live with multiple sclerosis (MS) with a 2:1 ratio of females to males. The global incidence is 2.1 per 100,000 persons-year [58]. Vertigo is a rare presenting symptom, only accounting for 8% of presenting symptoms of MS [59].

Typical Findings in the HPI

Clinical course is variable with insidious or acute symptoms. Some hallmark signs and symptoms are optic neuritis, worsening of symptoms with heat (Uhthoff phenomenon), and Lhermitte’s phenomenon, which is a radiating electric sensation after neck flexion. Other associated symptoms include sensory loss, weakness, bladder dysfunction, and fatigue. The clinical course is usually relapsing and remitting but can be primary or secondary progressive [60, 61].

Typical Physical Exam Findings

Physical exam findings are highly variable. Some notable findings include cranial nerve palsies, optic disc swelling, sensory impairments, limb and facial weakness, spasticity, and ataxia [61].

Typical Laboratory/Radiological Findings

Cerebrospinal fluid is notable for oligoclonal immunoglobulin IgG bands. MRI findings would include multifocal white matter lesions that are disseminated in time and space and can be seen within the brain, brainstem, and/or spinal cord [60, 61].

Screening Tools

While there are no screening assessments for the diagnosis of multiple sclerosis, there are screening assessments for *cognitive impairment* in people affected by MS. The Brief International Cognitive Assessment for Multiple Sclerosis (BICAMS) is a validated tool that evaluates information processing speed, immediate verbal recall memory, and immediate visual recall memory [62].

Pitfalls/Differential Diagnosis

The differential is wide due to the variable nature of MS as well as the extensive symptomatology that can occur. This includes stroke, neuromyelitis optica, sarcoidosis, antiphospholipid syndrome, vasculitis, B12 deficiency, and space-occupying lesions [60].

Traumatic Brain Injury

Incidence/Prevalence

There were 27 million cases of traumatic brain injury (TBI) globally in 2016. The global incidence of TBI was 369 per 100,000/year [63]. The prevalence of vertigo symptoms depends on the severity of the TBI and the time since injury. The greater the TBI severity, the higher the likelihood of vertigo. In one study of 321 patients, 23.8% of those with mild TBI reported symptoms of vertigo, 65% of those with moderate TBI reported symptoms of vertigo, and 78% with severe TBI had vertigo. The overall prevalence of vertigo dropped to 20% among the entire cohort after 6 months [64]. In another study ($n = 1255$) of subjects with mild TBI, the prevalence of nonspecific symptoms of dizziness was 28% at 2 weeks and 9% at 6 weeks [65]. Of note, the DSM classifies TBI as a mild or major neurocognitive disorder due to TBI [p. 624].

Typical Findings in the HPI

Traumatic brain injuries occur after trauma to the brain such as motor vehicle accidents, sports injuries, concussive forces (e.g., blast trauma), or rapid acceleration or deceleration. Patients usually endorse a history of loss of consciousness, amnesia, or disorientation following trauma. Headaches, vertigo, photo/phonophobia, irritability, and cognitive impairments in executive function, learning, and memory are common [pp. 625–627]. About 65% of patients with moderate to severe TBI report cognitive problems while up to 15% of mild TBI patients have persistent complaints of neurocognitive deficits [66]. Depending on the severity of the injury, other neurologic sequelae may include seizures, vision deficits, cranial nerve impairments, focal motor or peripheral sensory deficits, and sleep disturbances. Comorbid psychiatric symptoms may be present as well including depressed mood, poor concentration, sleep disturbance, and apathy [pp. 625–627].

Typical Physical and Mental Status Exam Findings

Physical exam findings vary depending on severity. Visual field defects can be seen, as well as cranial nerve abnormalities, weakness, and peripheral sensory deficits. Mental status exam findings vary as well. They include labile, inappropriate, apathetic, or dysphoric affect. Cognitive assessment can show executive function, language, attention, and/or memory impairment [pp. 626–627].

Typical Laboratory/Radiological Findings

Computed tomography and magnetic resonance imaging can be used to identify cerebral contusions or petechial, subarachnoid, and/or epidural and subdural hemorrhages depending on the location and severity of trauma [p. 627].

The Canadian CT Head Injury/Trauma Rule is a helpful tool to assess the need for head imaging. It applies to patients aged 16 and older, not on anticoagulation, with the absence of seizure after injury. There are seven criteria and if all are negative then it rules out the need for computed tomography or neurosurgical intervention [67].

Several candidate biomarkers of intracranial injury are under consideration. The Center for Disease Control and Prevention (CDC) mild traumatic brain injury (mTBI) guidance mentions the serum S-100B biomarkers with a possible role in assessing the need for head CT though it is currently not approved for clinical use in the United States [68]. The Food and Drug Administration (FDA) has approved a blood test for marketing in the United States. This test looks at the combination of two biomarkers including glial fibrillary acidic protein (GFAP) and ubiquitin C-terminal hydrolase-L1 (UCH-L1) [69]. The sensitivity and negative predictive value of the combined biomarkers are 97.6% and 99.6%, respectively, for the detection of acute intracranial injuries on head CT scan [70]. The impact of these tests on clinical practice is currently unclear but has great potential for resource constraint environments and reducing radiation exposure by eliminating unnecessary head CT scans.

Screening Tools

The Veterans Affairs Traumatic Brain Injury Clinical Reminder Screen is a four question self-assessment to screen for prior traumatic brain injuries [71]. In diagnosing head injuries in the past, the sensitivity is 72% and specificity is 54% when all four questions are answered positively. The screening questionnaire is helpful for clinicians to rule out TBI with a negative predictive value of 91–94% [72].

Pitfalls/Differential Diagnoses

Differential diagnosis includes somatic symptom disorder, factitious disorder, acute stress disorder, and posttraumatic stress disorder, which can overlap with TBI [p. 627]. Vertebrobasilar insufficiency should also be on the differential, which may present after reduced cerebral perfusion from prolonged standing or hypovolemia [21]. Another consideration is persistent postural-perceptual dizziness, which is

associated with waxing and waning dizziness spells and can be from a medical or psychiatric etiology [73].

Psychiatric Conditions Presenting with Vertigo

In the DSM-5-TR, the term “dizziness” is used 26 times (with “dizzy” having two additional mentions), “vertigo” and “syncope” are each mentioned six times, “light-headedness” is mentioned once, and there is no mention of “disequilibrium.” It is worth highlighting that there are only a very small number of psychiatric diagnoses from the DSM-5-TR that have dizzy or dizziness as part of their diagnostic description or criteria: panic disorder, panic attack specifier, and inhalant intoxication, although “dizziness” is not required to diagnose these disorders. Table 5.1 highlights various conditions where dizziness, vertigo, and other associated complaints and symptoms may present.

In the DSM-5-TR, “vertigo,” is more narrowly mentioned. It is essentially restricted to association with substance/medication-induced anxiety disorder and major or mild neurocognitive disorder due to traumatic brain injury. Vertigo is not part of either condition’s diagnostic criteria but may be part of a patient’s presentation. Onset of panic disorder after age 45 or atypical panic attack symptoms including vertigo should lead the clinician in the direction of a potential substance/medication-induced etiology. In regard to major or mild neurocognitive disorder due to traumatic brain injury, vertigo (and dizziness) are mentioned as a potential associated physical disturbance [74].

Anxiety disorder due to another medical condition in DSM-5-TR does not have vertigo or dizziness as part of the presentation, but vertigo due to another medical condition may result in this diagnosis. Any medical condition can create significant suffering and stress for a patient. Conditions with chronic vertigo can be distressing if unresponsive to therapy [75].

It is important to recognize, that most “classic” psychiatric conditions are diagnoses of exclusion, so all behavioral health clinicians need to consider a full work-up to rule out malignant etiologies.

Substance/Medication-Induced Anxiety Disorder

Incidence/Prevalence

Prevalence is unclear. General data suggests it is rare, with a 12-month prevalence of roughly 0.002% (likely to be higher in clinical populations) [p. 229]. The prevalence of vertigo is not known.

Typical Findings of the HPI

Anxiety, including panic attacks, is predominant in the clinical picture and there is evidence from the history, physical exam, or laboratory tests that the anxiety developed during or soon after starting a medication, or a substance intoxication or

withdrawal. The substance or medication involved must also be capable of producing panic or anxiety symptoms. In general, intoxication with central nervous system stimulants or cannabis and withdrawal from central nervous system depressants can precipitate a panic attack but there are many different classes of specific drugs, that can precipitate the spectrum of anxiety presentations [p. 217, 228, 229]. Patients can present with concerns for feelings of choking, chest pain, nausea, dizziness or lightheadedness, palpitations, paresthesias/cold extremities, fear of losing control, or that they may die during a panic attack. For a more general anxiety presentation, there may be fatigue, restlessness, irritability, sleep disturbance, muscle tension, or concentration deficits, and may also include tinnitus and neck soreness [pp. 222–223].

Typical Findings on Physical and Mental Status Exam

The presentation depends on the substance or medication and its associated effects on the autonomic nervous system. Physical exam may reveal atypical panic attack symptoms like slurred speech, loss of bladder control, loss of balance, and loss of consciousness [p. 229]. Familiarity with classic toxidromes is important. Patient's presenting with acute toxicity with anticholinergic, sympathomimetic, and serotonergic compounds (serotonin syndrome) have tachycardia, hyperthermia, and mydriasis on examination [76]. Hyperreflexia may be present in anticholinergic and sympathomimetic toxidromes, with extreme hyperreflexia and clonus unique to serotonin syndrome [77]. Withdrawal from sedative-hypnotics and alcohol may result in anxiety and panic symptoms. Patients in withdrawal may present with mental status changes, tachycardia, hypertension, mydriasis, as well as hyperreflexia. In Wernicke's encephalopathy, the classic triad of delirium, ataxia, and oculomotor abnormalities may be seen [78].

Typical Laboratory/Radiological Findings

Laboratory findings depend on the suspected substance or medication involved. Urine drug screens and blood alcohol level (BAL) may be positive for illicit substances. For alcohol, there are numerous biomarkers that may be helpful in assessing recent or excessive drinking. One such marker is the carbohydrate-deficient transferrin (CDT) that has a sensitivity of 46–90% and a specificity of 70–100% based on alcohol consumption in the prior 2-week period [79]. Combining CDT with gamma-glutamyltransferase (GGT) and mean corpuscular volume (MCV) improves the sensitivity and specificity by 88% and 95% respectively for chronic excessive drinking [80]. Other laboratory tests may be abnormal due to secondary end-organ damage such as acid-base abnormalities, acute kidney injury, liver injury, or rhabdomyolysis [76]. In case of specific syndromes such as Wernicke's encephalopathy, serum thiamine levels or red blood cell transketolase activity can confirm the diagnosis. MRI has a sensitivity and specificity of 53% and 93%, respectively. In the T2 and FLAIR sequences, MRI can show hyperintensities in the medial thalami, hypothalamus, mammillary bodies, periaqueductal area, and tectal plate [78].

Screening Tools

Laboratory testing such as urine and serum toxicology may be helpful to measure intoxication as discussed above. There are no other singular screening tools given the broad possibilities of substances and drugs.

Cultural Concepts Associated with Dizziness and Vertigo

In considering the full differential for dizziness and its subtypes, one must also consider the cultural aperture through which there can be numerous—and often incongruent—meanings attached to these terms. According to the DSM-5-TR, mental disorders are defined in relation to cultural and social norms which provide the interpretive context for the symptoms, signs, and behaviors used to make a diagnosis. Therefore, clinical assessment must consider whether an individual's presentation differs from sociocultural norms [81 p. 833].

Historically, previous versions of the DSM utilized the term, “culture-bound syndrome,” when speaking about cultural psychiatry, but the DSM-5-TR has clarified and refined cultural considerations with three concepts, hoping to increase clinical utility: cultural syndrome, cultural idiom of distress, and cultural explanation or perceived cause. Readers are referred to the DSM-5-TR pages 16–17 and pages 871–874 for a lengthier explanation of the concepts in the cultural issues section of the introduction and the glossary of cultural concepts of distress. The important takeaway is that the ability to classify symptoms and conditions culturally impacts many important aspects of the clinical encounter, including symptomatology, help-seeking, treatment expectations, illness adaptation, and treatment response.

In DSM-5-TR, vertigo is only mentioned within the cultural context of *nervios*, or “nerves,” which is a common idiom of distress among Latinos that refers to vulnerability to stressful life experiences and circumstances [82]. Although this chapter focuses specifically on vertigo, there are cultural phenomena that may present with the complaint of “dizziness.” *Kufungisisa* (“thinking too much” in Shona) is an idiom of distress and a cultural explanation among the Shona of Zimbabwe. As a cultural explanation, it may be the underlying cause of anxiety, depression, and somatic complaints. In the context of it being an idiom of distress, *kufungisisa* may represent social and interpersonal difficulties. “*Khyâl attacks*” (*khyâl cap*) or “wind attacks,” is a syndrome found among Cambodian people. It is defined by thoughts centered on the worry that *khyâl* (a windlike substance) may increase in the body—along with blood—and cause a range of symptoms such as compressing the lungs to cause shortness of breath and asphyxia; entering the cranium to cause tinnitus, dizziness, blurry vision, and a fatal syncope. Of note, there are no diagnostic criteria per se for each of these cultural concepts. The goal is to broaden the clinician's differential by highlighting the important consideration of culture in the clinical encounter. The additional cultural elements add a richer layer to the biopsychosocial formulation of our patients [83, pp. 833–837].

Nervios “Nerves”

Incidence/Prevalence

There is a prevalence of 15.5% in the general Mexican population. Women have a significantly higher prevalence (20.8%) than men (9.5%). Somatic and psychological symptoms associated with nervios have a higher prevalence among women than men [84]. Incidence and prevalence of Nervios “nerves” presenting with dizziness or vertigo is unknown [p. 835].

Typical Findings of the HPI

Findings may include emotional distress, somatic issues, and functional concerns. The most common presentation includes headaches and “brain aches” (occipital neck tension), tingling, stomach issues, sleep concerns, trembling, easy tearfulness, concentration difficulty, nervousness, irritability, and mareos (dizziness with occasional vertigo-like exacerbations) [p. 835].

Typical Findings on Physical and Mental Status Exam

Physical exam may reveal trembling and occipital muscle tension [p. 835]. The mental status exam can range in affective changes to include anxiety and depression. There may be dissociative and psychotic features expressed [p. 835].

Typical Laboratory/Radiological Findings

None.

Screening Tools

No known screening tools exist for nervios, but the patient should be screened for depressive and anxiety disorders.

Pitfalls/Differential Diagnoses

Differential diagnoses include major depressive disorder, persistent depressive disorder (dysthymia), generalized anxiety disorder, social anxiety disorder, other specified or unspecified dissociative disorder, somatic symptom disorder, and schizophrenia. There are related conditions in other cultures in North American Greeks and Sicilians, as well as Whites in Appalachia and Newfoundland [p. 835].

Kufungisisa “Thinking Too Much”

Incidence/Prevalence

Kufungisisa is experienced by up to 80% of Zimbabweans who present with a common mental disorder (mainly depressive and anxiety disorders). The estimated lifetime prevalence of common mental disorders in low- and middle-income countries is roughly 22% [85]. Incidence and prevalence of Kufungisisa presenting with dizziness or vertigo are unknown.

Typical Findings of the HPI

Kufungisisa is associated with anxiety symptoms, worry, panic, depressive symptoms, irritability, and somatic complaints. In many cultures, “thinking too much” is damaging to the mind and body and can cause specific symptoms like headache and dizziness [p. 834, 835].

Typical Findings on Physical and Mental Status Exam

Physical exam may reveal panic attack symptoms such as palpitations, tachycardia, sweating, trembling, shaking, and dyspnea [p. 834, 835]. Mental status exam may reveal ruminating on upsetting thoughts, particularly worries. Affect can present as anxious or depressed [p. 834, 835].

Typical Laboratory/Radiological Findings

None.

Screening Tools

No known screening tools exist for nervios, but the patient should be screened for depressive and anxiety disorders.

Pitfalls/Differential Diagnoses

Differential diagnoses include major depressive disorder, persistent depressive disorder (dysthymia), generalized anxiety disorder, posttraumatic stress disorder, obsessive-compulsive disorder, and persistent complex bereavement disorder. There are related conditions in other cultures described in Africa, the Caribbean, Latin America, and East Asian and Native American groups [p. 834, 835].

Khyâl Cap “Wind Attacks”**Incidence/Prevalence**

Unknown.

Incidence/Prevalence of Khyâl Cap “Wind Attacks” Presenting with Dizziness

Unknown.

Typical Findings of the HPI

Common symptoms include panic attacks to include dizziness. The attacks may occur suddenly but are commonly triggered by worrisome thoughts, orthostasis from standing, odors with negative associations, and agoraphobic cues like crowded spaces as one example. These attacks usually meet panic attack criteria [p. 834].

Typical Findings on Physical and Mental Status Exam

Physical exam symptoms include those of panic attacks, such as palpitations, shortness of breath, and cold extremities [p. 834]. The mental status exam can reveal anxious affect.

Typical Laboratory/Radiological Findings

None.

Screening Tools

No known screening tools exist for Khyâl cap, but the patient should be screened for depressive disorders, anxiety disorders, and trauma spectrum disorders.

Pitfalls/Differential Diagnoses

Differential diagnoses include panic attacks, panic disorder, generalized anxiety disorder, agoraphobia, posttraumatic stress disorder, and illness anxiety disorder. There are related conditions in other cultures of Laos, Tibet, Sri Lanka, and Korea as well [p. 834].

Conclusion

As the reader can grasp by now, “dizziness” is a broad and difficult to quantify presenting symptom across medical specialties, cultures, and individual patients. For culturally discordant clinicians, the task of negotiating subjective complaints of dizziness and associated symptoms become even more challenging. While we briefly mention some of the phenomena, the prudent approach is openness and curiosity on the part of the clinician to understand the individual patient’s full breadth of experiences and suffering.

To conclude, obtaining clinical utility from a chief concern of “dizziness,” a term shown in this chapter to be vague and unclear in its meaning (not only from person to person, but also culture to culture, and specialty to specialty), requires differentiation into the subcategories of vertigo, syncope, or disequilibrium. As discussed, this is best accomplished by transitioning from an open-ended inquisition to more closed-ended follow-up questions that all take place in an empathic environment. Once a subcategory is identified, it allows for a narrower medical and psychiatric differential with implications for acuity of presentation that can impact patient outcomes.

Although understanding disequilibrium/lightheadedness and syncope/presyncope are crucial for a comprehensive approach to “dizziness,” this chapter focused on vertigo, given its representation as the major etiological cause. As highlighted, vertigo results from direct vestibular apparatus pathology or from how the brain processes information from it, resulting in a false sense of environmental spinning/motion. Key points worth remembering are that most vertiginous cases are the result of peripheral etiologies as opposed to central, and that evaluation of vertigo by psychiatric providers should focus on ruling out medical causes including TBI, substances and medications use or withdrawal.

Although, “dizziness” can be a scary experience for patients and a daunting assessment for clinicians, following the mental model laid out in this chapter provides a framework to ensure that is not the case.

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