Abstract
Dizziness and vertigo are common reasons that patients present to the emergency department. Most patients have benign or self-limited causes, but a small proportion harbor a dangerous disorder such as ischemic stroke. In this chapter, we highlight the critical steps in evaluating and managing patients who present with dizziness symptoms in the emergency setting. We focus on the symptom of vertigo and distinguishing peripheral vestibular from central vestibular disorders.

Keywords
Dizziness • Vertigo • Vestibular neuritis • Benign paroxysmal positional vertigo • Stroke • Meniere’s disease

Introduction
Physicians have high levels of uncertainty when faced with the evaluation and management of dizziness presentations in the emergency setting. In a recent survey of emergency medicine physicians, “identification of central or serious causes of vertigo” was ranked as the #1 priority for clinical decision support research in adult emergency presentations [1]. Uncertainty also likely contributes to the dramatic increase in the use of imaging studies in emergency department dizziness presentations. In 1995, less than 10% of patients presenting to emergency departments (ED) with dizziness were evaluated with a head computerized tomography (CT) scan, but by 2004 the rate had doubled to greater than 25% [2]. Despite this increase in head CT use, the proportion of ED dizziness visits receiving a central nervous system diagnosis did not increase [2].

Most patients presenting with dizziness can be rapidly assessed and valid estimates can be made regarding diagnostic possibilities—thus informing management decisions. There already exist many effective treatments for dizziness symptoms and specific dizziness disorders. In fact, benign paroxysmal positional vertigo (BPPV) is
The three most common specific peripheral vestibular disorders

<table>
<thead>
<tr>
<th>Type of presentation</th>
<th>Symptoms</th>
<th>Exam findings</th>
<th>Red flags for central etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibular neuritis</td>
<td>Acute severe prolonged dizziness</td>
<td>Constant vertigo, nausea, and imbalance</td>
<td>Spontaneous unidirectional horizontal nystagmus, positive corresponding head thrust test</td>
</tr>
<tr>
<td>Meniere’s disease</td>
<td>Recurrent spontaneous attacks</td>
<td>Vertigo, nausea, imbalance last hours, unilateral fluctuating hearing loss</td>
<td>Peripheral pattern of nystagmus, unilateral hearing loss</td>
</tr>
<tr>
<td>Benign paroxysmal positional vertigo</td>
<td>Recurrent positionally triggered attacks</td>
<td>Recurrent positionally triggered attacks of vertigo</td>
<td>Duration &lt;1 min</td>
</tr>
</tbody>
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Red flags for all presentation type include other focal neurological signs or symptoms.

With left-sided vestibular neuritis, the spontaneous nystagmus beats to the right side and the head thrust test reveals a corrective saccade after movements to the left side.

Central patterns of nystagmus include spontaneous vertical (up or downbeating) nystagmus, bidirectional gaze-evoked nystagmus, and downbeating nystagmus triggered by positional testing.

Benign paroxysmal positional vertigo (BPPV) variant: with horizontal canal BPPV, horizontal nystagmus will be triggered by supine positional testing.

among the most common causes of dizziness and it can be cured by a simple repositioning maneuver (i.e., the Epley maneuver) at the bedside [3]. The goal in the management of dizziness presentations is to get the most effective treatments to the dizziness patients most likely to benefit from them, and to do so in an efficient manner. Achieving this goal depends on the bedside assessment and the formulation of the case.

Most of the uncertainty in dizziness presentations occurs when attempting to distinguish “peripheral” (and generally benign) from “central” (and potentially life-threatening) causes. The key to distinguishing between these is understanding the three most common peripheral vestibular disorders (i.e., vestibular neuritis, BPPV, and Meniere’s disease) (Table 3.1). Typically, the most effective way to “rule out” a life-threatening central disorder is to “rule in” a specific peripheral vestibular disorder. The peripheral vestibular disorders are important because they account for a large proportion of the causes of dizziness, present with highly stereotyped characteristics, and can be effectively treated. The time to consider a central etiology is when the presentation deviates from the stereotyped characteristics of the specific peripheral vestibular disorders.

In this chapter, we highlight the critical steps in evaluating and managing patients who present with dizziness symptoms in the emergency setting. We focus on the symptom of vertigo and distinguishing peripheral vestibular from central vestibular disorders.

**Evaluation of Emergency Presentations**

The effective clinical evaluation of patients presenting with dizziness in the emergency setting requires an organized approach that allows the physician to gather all of the most relevant information and then to formulate the case in a way that establishes the most likely cause and identifies any relevant “red flags” that could suggest a central disorder.

**Step 1. Determine if the Dizziness Is the Principal Symptom as Opposed to a Minor Accompanying Symptom**

Dizziness is an incredibly common accompanying symptom. More than 60% of all patients in the emergency department will report having
dizziness when specifically queried about it [4], and in most cases it is a minor accompanying symptom rather than the principal symptom. One of the main problems with dizziness presentations is that the patient’s descriptions of dizziness can be very vague, inconsistent, and unreliable [4]. So prior to focusing all attention on the dizziness symptom, first consider if other symptoms are more prominent. For example, if chest pain is the principal symptom, then an initial focus on cardiac etiologies is probably more effective than a focus on vestibular system etiologies.

**Step 2. Define the Characteristics of the Dizziness Symptom**

If the dizziness symptom is the principal symptom, then the next step is to more clearly define it. However, defining the dizziness symptom is often not a simple task because it is a subjective experience and many patients with dizziness have difficulty describing what they are actually experiencing. There can also be problems with being overly reliant on the patient’s description of the symptom in informing the potential causes. For example, vertigo (i.e., visualized movement of the environment) is one common type of dizziness symptom, and it should localize to the vestibular system (either the peripheral vestibular system or the central vestibular system). But some patients with cardiac disorders unlikely to involve the vestibular system will report movement of the environment particularly if they are specifically queried about it [5]. An intense visualized room-spinning sensation is a much more valid indicator of a vestibular system disorder than either an “internal” spinning sensation (i.e., no actual spinning of the environment) or a very mild visualized spinning sensation. There are also patients who have a clear vestibular disorder, but who describe the symptom as a nonvertiginous vague type of dizziness, even in the setting of frank nystagmus.

Other types of dizziness symptoms to consider are light-headedness with presyncope, light-headedness (or similar “head” sensation) without presyncope, or imbalance. Some patients will use the label “dizziness” to describe anxiety-like symptoms, general fatigue or weakness, or just not feeling well.

Because of the problem with patient descriptions of dizziness symptoms, in many cases the characteristics of the symptom may be equally or even more important than defining the exact symptom itself. Defining the characteristics of the symptom starts with defining whether the symptom is episodic or constant. If the symptom is episodic, then one should probe regarding triggers of the symptom and the frequency and duration of the episodes. When the symptom is constant, one should determine the onset of the symptom and aggravating and alleviating factors. Determining accompanying symptoms is also a vital step, particularly gathering information about auditory symptoms or focal neurological symptoms.

The information from the history will eventually be a key aspect when formulating the case (see step 5). The details from the history help to categorize the patient into broad classifications of dizziness presentations which are relevant to determining potential etiologies. Helpful classifications of presentations include the following: acute severe prolonged dizziness, recurrent spontaneous dizziness attacks, and recurrent positionally triggered dizziness attacks. The type of presentation is determined based on the details of the history of present illness. Acute severe prolonged dizziness is the sudden onset of a constant symptom that is generally a debilitating symptom. Recurrent spontaneous dizziness attacks are reported by patients who have had at least several attacks that come on without any apparent inciting event. Recurrent positionally triggered dizziness attacks consist of the symptom triggered by certain head movements.

**Step 3. Perform a General Neurological Examination**

A general neurological examination is important because any relevant motor, sensory, or language deficits will likely warrant a workup for a central disorder regardless of the other characteristics of
the dizziness symptom. This is because peripheral vestibular disorders and general medical disorders will not cause focal neurological deficits. Unilateral hearing loss, on the other hand, will strongly suggest a peripheral etiology.

For similar reasons, a general medical examination can also be important when trying to exclude a general medical disorder such as a heart arrhythmia or orthostatic hypertension.

### Table 3.2 Patterns of nystagmus associated with the type of dizziness presentation

<table>
<thead>
<tr>
<th></th>
<th>Spontaneous nystagmus?</th>
<th>Peripheral vestibular patterns of nystagmus</th>
<th>Central vestibular patterns of nystagmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute severe prolonged dizziness</td>
<td>Yes</td>
<td>• Unidirectional, horizontal spontaneous&lt;sup&gt;a&lt;/sup&gt;</td>
<td>• Direction-changing, gaze-evoked&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Unidirectional, horizontal spontaneous&lt;sup&gt;c&lt;/sup&gt;</td>
<td>• Spontaneous vertical or pure torsional</td>
</tr>
<tr>
<td>Recurrent spontaneous attacks</td>
<td>Yes/No&lt;sup&gt;d&lt;/sup&gt;</td>
<td>• Dix–Hallpike test: burst of upbeat torsional&lt;sup&gt;e&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Dix–Hallpike test: burst of upbeat torsional&lt;sup&gt;d&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>Recurrent positionally triggered attacks</td>
<td>No</td>
<td>• Dix–Hallpike test: burst of upbeat torsional&lt;sup&gt;e&lt;/sup&gt;</td>
<td>• Downbeating persistent</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Dix–Hallpike test: burst of upbeat torsional&lt;sup&gt;d&lt;/sup&gt;</td>
<td>• Pure torsional</td>
</tr>
</tbody>
</table>

<sup>a</sup>Pattern can less commonly be caused by central lesions, increasing the importance of assessing risk for central lesion and the results of the head thrust test.

<sup>b</sup>Example of direction-changing gaze-evoked nystagmus: left-beating nystagmus with gaze to the left; then, right-beating nystagmus with gaze to the right.

<sup>c</sup>May not have nystagmus if evaluation takes place in between attacks.

<sup>d</sup>Upon sitting up from Dix–Hallpike test, a burst of downbeating torsional nystagmus will often be triggered. Thus, a direction-changing positionally evoked nystagmus.

<sup>e</sup>In rare circumstances, pattern can be caused by a central lesion.

### Step 4. Perform a Neuro-Otologic Assessment

If the source of the symptom is not clear after performing steps 1–3, then the neuro-otologic assessment becomes paramount. Subtle differences in eye movements or the vestibuloocular reflex can be highly localizing. The key neuro-otologic examination components are the following: an assessment of nystagmus, positional testing when applicable, and the head thrust test when applicable.

Pathological nystagmus occurs as the result of an acute imbalance of the vestibular system which can stem from a lesion (or aberrant stimulation) of peripheral or central vestibular structures. Most physicians notice when nystagmus is present, but it is the pattern of nystagmus—not the mere presence—that is important for discriminating a peripheral lesion from a central lesion. The localizing value of the pattern of nystagmus is also dependent on the type of presentation. Some general rules about the localizing value of nystagmus apply (Table 3.2). In patients with acute severe prolonged vertigo, a unidirectional spontaneous horizontal nystagmus is highly suggestive of a lesion of the vestibular nerve. The lesioned side is the side opposite the direction of the fast phase of nystagmus. Unidirectional spontaneous nystagmus implies that the nystagmus is present in primary gaze and that the nystagmus never changes direction. For example, if nystagmus beats to the left side, then it should never transition to beating to the right side. The left-beating nystagmus does increase in velocity when the patient looks to the left side and also decrease (or stop) when the patient looks to the right side, but it will not transit to a right-beating nystagmus if the lesion is on the vestibular nerve. A central
lesion is presumed in acute severe vertigo presentations whenever a pattern other than uni-directional horizontal nystagmus is observed. The most common central nervous system patterns of nystagmus in acute severe vertigo presentations are direction-changing gaze-evoked nystagmus (i.e., patient looks to the right and nystagmus beats to the right; then, patient looks to the left and nystagmus beats to the left) and spontaneous vertical (typically downbeating) nystagmus.

Positional testing is an important component of the bedside examination when the type of presentation is recurrent positional dizziness. It is important to note that the patterns of nystagmus that discriminate peripheral from central etiologies when the presentation type is recurrent positional dizziness are different from the patterns in acute severe dizziness presentations. Generally no spontaneous nystagmus is present in positionally triggered presentations. In positionally triggered attacks caused by BPPV, the nystagmus can change direction, which occurs with changes in head position. In addition, a principally vertical nystagmus is the characteristic pattern of the most common BPPV variant (i.e., posterior canal BPPV). In posterior canal BPPV, the Dix–Hallpike test (Fig. 3.1) [3, 6] triggers a burst of upbeating and torsional nystagmus which lasts less than 1 min. If the patient were to next sit back up from the Dix–Hallpike position, then a burst of downbeating and torsional nystagmus is triggered. The reason for the change in direction of the nystagmus after sitting up is that the particles move in the opposite direction after sitting up compared to the head-hanging (i.e., Dix–Hallpike) position. However, if persistent downbeating nystagmus is triggered by the Dix–Hallpike test, then a central nervous system lesion is presumed.

If the Dix–Hallpike positional test does not trigger the nystagmus of BPPV, then supine positional testing is used to test for the less common horizontal canal variant of BPPV [7]. With this test the patient lies supine and the head is turned first to one side and held for at least 30 s and then to the other side and held for the same duration. A burst of horizontal nystagmus beating toward the ground is characteristic of the horizontal canal variant of BPPV. The side with stronger nystagmus is the abnormal side. More persistent nystagmus beating away from the ground can occur if the debris is stuck within the canal or is attached to the cupula [3, 7].

The head thrust test is an important bedside examination component when the type of dizziness presentation is acute severe dizziness (Fig. 3.2). The head thrust test allows a direct assessment of the vestibular-ocular reflex (VOR) and an abnormal result is highly suggestive of a vestibular nerve lesion [8, 9]. This test is different from the doll’s eye test because the doll’s eye test uses slow rotation of the head to either side, whereas the head thrust test uses quick movements which isolate the vestibular system function. The corresponding eye movements of the doll’s eye test can be generated by either the vestibular system or the smooth pursuit system in a conscious patient. But only the vestibular system generates the reflex movement of the eyes after the quick movement of the head thrust test (the smooth pursuit system only works at low stimulus velocities). To test the VOR using the head thrust test, the examiner stands in front of the patient and holds the patient’s head with both hands. The patient is instructed to focus on the examiner’s nose, and then the examiner initiates a quick 10–15° movement of the patient’s head to one side. When there is a lesion of the VOR on one side, a corrective eye movement (i.e., a corrective “saccade”) back to the examiner’s nose is seen after the head is moved toward the affected side. In contrast and serving as an internal control, the eyes will stay on target (i.e., the examiner’s nose) after the head thrust test toward the normal side because the VOR is intact on that side. These features can be appreciated even when spontaneous nystagmus is present. The reason for the corrective saccade with a peripheral vestibular lesion is rooted in the physiology of the vestibular system [10]. When the head is moved quickly in one direction, the reflex (i.e., the VOR) that moves the eyes toward the opposite direction is generated mostly by the side the head moved toward. Thus a patient with vestibular neuritis of the right side will present with a
left-beating unidirectional nystagmus and the head thrust test will be positive with movement toward the right side.

The neuro-otologic examination of the patient with dizziness caused by Meniere’s disease is less predictable because most of the symptoms have typically resolved by the time of the evaluation, the nystagmus can be either toward or away from the affected ear (since it can be a stimulatory or inhibitory lesion),

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**Fig. 3.1** The Dix–Hallpike test for the diagnosis of posterior canal benign paroxysmal positional vertigo affected the right ear, and the Epley maneuver for the treatment of posterior canal benign paroxysmal positional vertigo affecting the right ear. The procedure can be reversed for treating the left ear. The drawing of the labyrinth in the center shows the position of the debris as it moves around the posterior semicircular canal (PSC) and into the utricle (UT). The patient is seated upright, with head facing the examiner, who is standing on the right. (a) The patient is then rapidly moved to head-hanging right position (Dix–Hallpike test). This position is maintained until the nystagmus ceases. (b) The examiner moves to the head of the table, repositioning hands as shown. (c) The head is rotated quickly to the left with right ear upward. This position is maintained for 30 s. (d) The patient rolls onto the left side while the examiner rapidly rotates the head leftward until the nose is directed toward the floor. This position is then held for 30 s. (e) The patient is rapidly lifted into the sitting position, now facing left. The entire sequence should be repeated until no nystagmus can be elicited. Following the maneuver, the patient is instructed to avoid head hanging positions to prevent the debris from reentering the posterior canal. From: Rakel RE. Conn’s Current Therapy 1995, p. 839, WB Saunders, 1995. Used with kind permission of Elsevier. Video clips of the Dix–Hallpike test, Epley maneuver, and other positional test are available from the American Academy of Neurology at [http://www.neurology.org/cgi/content/full/70/22/2067/DC2](http://www.neurology.org/cgi/content/full/70/22/2067/DC2).
Fig. 3.2 The head thrust test. The head thrust test is a test of vestibular function that can be easily done during the bedside examination. This maneuver tests the vestibulo-ocular reflex (VOR). The patient sits in front of the examiner and the examiner holds the patient’s head steady in the midline. The patient is instructed to maintain gaze on the nose of the examiner. The examiner then quickly turns the patient’s head about 10–15 degrees to one side and observes the ability of the patient to keep the eyes locked on the examiner’s nose. If the patient’s eyes stay locked on the examiner’s nose (i.e., no corrective saccade) (picture a), then the peripheral vestibular system is assumed to be intact. Thus, in a patient with acute dizziness, this finding suggests a central nervous system localization. If, however, the patient’s eyes move with the head (picture b) and then the patient makes a voluntary eye movement back to the examiner’s nose (i.e., corrective saccade), then this suggests a lesion of the peripheral vestibular system and not the central nervous system. Thus when a patient presents with the acute vestibular syndrome, the test result shown in picture a would suggest a central nervous system lesion, whereas the test result in picture b would suggest a peripheral vestibular lesion (thus, vestibular neuritis). From: Edlow JA, et al. Lancet Neurology 2008; 7(10):951–964. Used with kind permission of Elsevier.
and the head thrust test is typically normal. Regardless, central patterns of nystagmus should be a red flag. Furthermore, a key feature of Meniere’s disease is fluctuating hearing loss; however, the auditory symptoms can be mild or unappreciated by the patient during the early phases of the disorder. By mid-to-late stages of the disorder, a persistent unilateral hearing loss will be present.

**Step 5: Formulate the Differential Diagnosis**

When formulating the case, an initial helpful step is to first determine which classification of dizziness presentation the patient falls under. Likely etiologies can then be determined by further considering the presentation features and the information gathered from the examination.

**Acute Severe Prolonged Vertigo**

Vestibular neuritis is the most common cause of acute severe prolonged vertigo [11]. It is caused by a viral inflammation of the eighth cranial nerve and vestibular end organs. Vertigo is accompanied by severe nausea, vomiting, and imbalance. Patients will often describe the need to hold onto objects when walking or may even need to crawl. Typically hearing is not affected, but if it is, then the virus likely involves both auditory and vestibular components, so-called labyrinthitis. As noted earlier, the hallmark examination signs of vestibular neuritis are a spontaneous unidirectional horizontal nystagmus and a positive head thrust test to the side opposite the fastiging beating component of the nystagmus.

Patients with vestibular neuritis are typically debilitated for the first day. Then, the natural history of the disorder is a gradual recovery over weeks to months. Vestibular physical therapy programs can help to speed the recovery [12]. In addition, the use of a burst and taper of oral corticosteroids can improve the recovery of the affected vestibular system as measured by the laboratory caloric response [13].

In any patient who presents with acute severe vertigo, stroke diagnosis should be considered. Stroke is an obvious concern when the patient reports other focal neurological symptoms or has other focal neurological signs. Though the likelihood of stroke diagnosis drops substantially when the patient presents with isolated vertigo (i.e., no symptoms other than vertigo, nausea, and imbalance) [14], case reports now demonstrate just how closely stroke can mimic vestibular neuritis [15–17]. Lacking is a formal validated tool to assess the probability of stroke in acute severe vertigo presentations. From epidemiological study designs, the risk of stroke etiology among patients presenting to the ED with dizziness symptoms is about 3% [14]. If the dizziness is an isolated symptom, then the risk of stroke etiology drops to less than 1% [14]. However, the population of this study was patients with any dizziness symptom presentation, not just the acute severe vertigo presentation. This distinction is important because the probability of stroke is highest for acute severe vertigo presentations compared to the other types of dizziness presentations. One series that looked at acute vertigo presentations found a 25% (6 out of 24 patients) rate of stroke diagnosis [18]. Another series found that 25 out of 33 patients (76%) with acute severe vertigo presentations had stroke etiology, though a higher rate of stroke was selected for since at least one stroke risk factor was required for inclusion and a recent viral infection was an exclusion criterion [16]. In addition, patients in this series were enrolled even if they had bidirectional gaze-evoked nystagmus (a central pattern of nystagmus) at the time of presentation [16]. Even in these series which showed very concerning rates of stroke among acute severe vertigo presentations, the rate of stroke etiology dropped substantially among patients with isolated dizziness, unidirectional spontaneous horizontal nystagmus, and a corresponding positive head thrust test. Despite this, we still need large studies of the patients with acute severe vertigo so that validated and clinically meaningful probabilities can inform decisions.
Recurrent Spontaneous Attacks of Vertigo

Meniere’s disease is the prototypical episodic otological disorder characterized by recurrent vertigo attacks (typically lasting hours). Overall, the prevalence of Meniere’s disease in the general population is low [19]. In addition, Meniere’s disease patients are probably less likely to present to the ED during acute attacks compared to those with the first ever acute severe vertigo attack. The reason may be that Meniere’s disease attacks are typically limited to a couple of hours and patients learn over time that the attacks resolve with rest. To make the diagnosis of Meniere’s disease requires the presence of a unilateral hearing loss which is typically a fluctuating symptom early in the course but then later becomes a fixed and progressive feature. Other auditory symptoms are also common, including unilateral tinnitus (typically a low roaring sound rather than a high-pitched sound) or bothersome pressure in one ear. The examination in patients with Meniere’s disease can be variable in the acute setting because nystagmus can be caused by either stimulation or inhibition of the affected side. But a central pattern of nystagmus (e.g., spontaneous downbeating nystagmus or bidirectional gaze-evoked nystagmus) would be a reason for a workup for a central disorder. Patients with Meniere’s disease do not typically have a positive head thrust test.

Migraine is a more common cause of recurrent attacks of vertigo (so-called migrainous vertigo). Attacks can last from minutes to hours and during attacks patients may exhibit features of both peripheral and central spontaneous and positional vertigo [20]. In between episodes, the exam is normal. The diagnosis rests on identifying other migraine symptoms (headache, aura, photophobia, phonophobia) with at least some attacks [21], but if a patient has recurrent vertigo attacks without hearing loss over time, then the most likely diagnosis remains migraine even if other migraine symptoms are not reported.

Transient ischemic attacks should be considered when brief vertigo attacks (minutes) occur in a patient with vascular risk factors. Usually at least some attacks are accompanied by other neurological symptoms, and they may have a crescendo-like presentation. Sometimes patients who eventually suffer a posterior circulation stroke can have isolated transient vertigo episodes preceding the stroke [22]. As with stroke in general, auditory symptoms can accompany the vertigo symptoms if the anterior inferior cerebellar artery is involved.

Recurrent Positionally Triggered Attacks of Vertigo

BPPV is the most common cause of positionally triggered vertigo and in fact is also believed to be the most common cause of vertigo in general [23]. BPPV can be cured at the bedside with a simple repositioning maneuver [24]. Thus, the ability to identify and treat BPPV is a major step not only for improving patient outcomes, but also for reducing unnecessary tests. The key feature of the history is that the episodes are triggered by head movements, not simply worsened by head movements. It is important to know that dizziness of any cause can worsen after certain position changes. But for patients with BPPV, the vertigo attacks are triggered by position changes. The patient with constant vertigo who reports that the symptom is better in certain positions and worse with movement should be classified as having acute severe prolonged vertigo rather than recurrent positionally triggered vertigo. The history of the patient with BPPV is vertigo triggered by head tilts (reaching for something on a high shelf), rolling over in bed, or getting in/out of bed. The vertigo attacks last less than 1 min, followed by a return to the normal state. Some patients will report attacks only in the morning or evening when getting in or out of bed, but others will report attacks throughout the day.

BPPV is caused by calcium carbonate crystals which are free floating in a semicircular canal, typically the posterior canal. The debris breaks from the otolith membrane for reasons that are not clear. This can occur as the result of head trauma, but typically occurs spontaneously (particularly with aging). When the particles enter the posterior canal they can become trapped and move back and
forth with position changes. Since the particles settle quickly after the movement, the symptoms and nystagmus last for only a brief period of time (<1 min). The particles can less commonly enter the horizontal canal and rarely even the anterior canal. The pattern of nystagmus is different depending on which canal is affected [25]. When the particles are in the posterior canal, a burst of upbeat and torsional nystagmus is seen after the patient is placed in the Dix–Hallpike position with the head turned toward the affected side (see Fig. 3.1) [6]. The nystagmus typically lasts only about 20–30 s. When the particles are in the horizontal canal, the nystagmus is horizontal and typically beats toward the ground after turning the head to either side while the patient is supine. The horizontal canal nystagmus lasts longer than the posterior canal nystagmus (as long as a minute) and can persist when the patient returns to the sitting position.

Central disorders can cause positional vertigo attacks, but the attacks typically have features that distinguish them from attacks in BPPV. A downbeating positional nystagmus is the most common pattern of nystagmus indicating a central localization—typically a midline cerebellar lesion. Downbeating positional nystagmus can be caused by the anterior canal variant of BPPV, but this variant is rare. Multiple sclerosis can also cause various types of positional nystagmus as can any other lesion involving central vestibular pathways in the brainstem or cerebellum. Importantly, central lesions do not cause the characteristic vertical torsional nystagmus pattern of posterior canal BPPV. However, central lesions—particularly lesions around the fourth ventricle—may cause a pattern of nystagmus similar to the pattern seen with horizontal canal BPPV [26]. Thus, a central lesion should be considered when a patient with the horizontal canal BPPV pattern of nystagmus has atypical features or is refractory to repositioning.

**Other Dizziness Symptoms and Presentations**

In the ED setting, the symptom of imbalance is associated with a higher odds of stroke diagnosis compared to the symptom of “dizziness” [14]. In stroke patients with imbalance, the lesion is typically in the midline or superior cerebellum and often the patient requires assistance to ambulate, if ambulation is possible at all [15, 27]. Since the lesions are often in the midline cerebellum, appendicular ataxia may be lacking. Some patients with dizziness in the emergency room will present with a chronic constant dizziness presentation rather than one of the three common presentation types described previously. If the neurological exam is normal in the patient with chronic dizziness, then the chance of a structural neurological disorder is very low. Migraine is the great mimicker of all causes of dizziness [20]. Symptoms in migraine can present as an acute severe attack, positional episodes, recurrent spontaneous attacks, or chronic constant symptoms. An accompanying headache occurs in less than 50% of the presentations, although a personal history of migraine headaches or a strong family history of migraine is common. Suggested diagnostic criteria require migraine symptoms with at least some attacks of vertigo [21, 28]. Unfortunately, the diagnosis of migraine remains a diagnosis of exclusion. Thus, if the symptom is new in onset and does not fit the features of a specific peripheral vestibular disorder, then serious central causes should be considered. However, if the symptoms have been present for at least a couple of months and the neurological exam is nonfocal, then the chance of uncovering a causative structural lesion of the central nervous system is very low.

Panic disorder and anxiety disorder often have dizziness or even vertigo as a symptom. Common accompaniments of these psychiatric disorders are a sense of doom or fear, heart palpitations, shortness of breath, and nonfocal numbness and tingling.

General medical disorders can cause various types of dizziness presentations. The dizziness is typically described as light-headedness. Processes that result in transient drops in blood pressure are probably the most common general medical causes of dizziness. Medication side effects or metabolic derangements should also be considered in the differential diagnosis.
Management of Emergency Presentations

The goal in the emergency setting is to stabilize symptoms and identify treatable disorders or monitor those patients at risk for worsening. Proceeding through the above steps will help to identify the most likely causes and red flags. Simply classifying the presentation as a “peripheral” cause or “dizziness not otherwise specified,” without proceeding through the above steps, probably leaves too much room for error [29]. BPPV is not only readily identifiable at the time of the clinical presentation, but the most common type (i.e., posterior canal BPPV) can be effectively treated with the Epley maneuver (see Fig. 3.1) [3, 6, 30]. BPPV is unique in clinical medicine because not only can an accurate assessment of the likelihood of the diagnosis be made, but also you can take this one step further and actually prove the diagnosis at the bedside by treating it in a matter of minutes. If the features are atypical for BPPV or the patient does not respond to repositioning maneuvers, then central disorders can be considered (see Table 3.1).

If the patient presents with acute severe vertigo, then the history and the examination are the key elements. The patient with isolated vertigo has a very low probability of stroke [14], but this may still be a concerning probability. The probability of stroke drops further when isolated vertigo is accompanied by a unidirectional horizontal spontaneous nystagmus [15, 16]. And the probability of stroke drops even further when the patient has isolated vertigo, unidirectional horizontal spontaneous nystagmus, and a corresponding positive head thrust test [15, 16].

If a patient presents with recurrent spontaneous episodes of vertigo, the chance of transient ischemic attack as the cause is low if the symptom lasts for hours, the attacks date back more than a couple of months, and prominent unilateral auditory features are reported. These features are highly suggestive of Meniere’s disease. On the other hand, if the attacks are new in onset, brief in duration (minutes rather than hours), and not accompanied by prominent unilateral auditory features, then TIA should be a strong consideration.

Vestibular physical therapy is recommended for patients with acute vestibular neuritis [12]. Regarding medication treatments in patients with vestibular neuritis, a randomized placebo-controlled trial of oral corticosteroids and valacyclovir (2 by 2 factorial design) found that on average patients treated with corticosteroids within 3 days of onset had a superior improvement in vestibular recovery as measured by the surrogate outcome of caloric response at 12 months compared to placebo [13]. Valacyclovir did not demonstrate an average beneficial effect. It is not known if corticosteroids improve functional outcome since functional outcome was not assessed in this trial and many patients with a chronic caloric asymmetry are asymptomatic. If the patient presenting with acute vertigo actually has the Ramsay Hunt syndrome (additional features of vesicles in the external auditory canal and facial palsy, with or without hearing loss) [31], then antiviral treatment is often added since Ramsey Hunt syndrome is of presumed varicella zoster virus (VZV) origin [32]. However, there remains uncertainty because of a lack of adequate trials in Ramsey Hunt syndrome [33, 34], and also because the beneficial effect of antiviral use in the most common VZV disorder (i.e., shingles), is the time to pain recovery and time to skin lesion healing, without evidence of a beneficial long term outcome [32, 35, 36]. Another neurotologic cranial nerve neuritis syndrome, “sudden sensorineural hearing loss,” also lacks adequate trial data [37] but generally oral corticosteroids are recommended [38]. Interest has increased in the use of intratympanic (IT) corticosteroids injection in sudden sensorineural hearing loss, particularly when used as a “salvage” therapy following failed oral treatment [39, 40], but large and rigorous trials are still necessary to establish the effect.

Regardless of cause, symptoms can be managed with either oral or intravenous medications (Table 3.3). Few randomized controlled trials have been conducted on the symptomatic treatment of acute dizziness. In one study of 74 patients with acute dizziness, the average effect
of 50 mg of intravenous dimenhydrinate was superior to that of 2 mg of intravenous lorazepam [41]. Antinausea medications (e.g., promethazine, prochlorperazine) can be considered when nausea and vomiting are prominent symptoms. One typically begins with less sedating medications. If the effect is not adequate, then more sedating medications are indicated. Patients should be instructed to only use these medicines during the acute phase because use beyond this period is more likely to cause bothersome side effects than any benefit.

**Imaging Studies in Emergency Presentations**

The use of neuroimaging studies in ED presentations of dizziness has risen dramatically. As noted earlier, in a population of “vertigo-dizziness” ED presentations, more than 25% of patients had a CT scan in 2004 compared with less than 10% of patients in 1995 [2]. In some subgroups, the percent evaluated with a CT scan was nearly 40% [2]. But the sensitivity of CT scans for identifying stroke in the acute setting is a dismal 26% [42], meaning that a negative test does not change the probability of stroke diagnosis in a meaningful way. In addition, CT scans are associated with important downsides, including radiation exposure, increased cost, and increased time in the emergency room. MRI is a much more sensitive test, but is not generally available in the acute setting, takes more time, and has much greater expense. In addition, the sensitivity of MRI is lowest (thus can miss a stroke) when the test is performed within the first 24 h of symptom onset and when the lesion is in the brainstem or cerebellum [16, 42, 43]. A validated clinical decision rule to determine which patients are likely to benefit from neuroimaging could go a long way in optimizing patient outcomes and health-care utilization. But such a rule would need to be supported by policy to have an impact. In the absence of such a rule, clinical judgment must be used when evaluating individual patients. The vertigo patients at highest risk for stroke etiology are those with other focal neurological symptoms or signs (including central patterns of nystagmus), stroke risk factors, acute severe vertigo or imbalance type of presentation, and acute severe vertigo with a negative head thrust test [14–16, 18].

### Conclusion

The evaluation of emergency department dizziness patients is facilitated by an organized approach. An accurate assessment of the most likely diagnosis can be made by categorizing the type of presentation and considering the examination findings. The optimal way to “rule out” a central disorder is to “rule in” a specific peripheral vestibular disorder (i.e., vestibular neuritis, BPPV, or Meniere’s disease). When the key

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing</th>
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<tbody>
<tr>
<td><strong>Less sedating</strong></td>
<td></td>
</tr>
<tr>
<td>Dimenhydrinate</td>
<td>50–100 mg PO/IV every 4–6 h</td>
</tr>
<tr>
<td>Meclizine</td>
<td>25–50 mg PO every 4–6 h</td>
</tr>
<tr>
<td>Scopolamine</td>
<td>0.4 mg PO every 8 h; 1.5 mg topical disc every 3 days</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>25–50 mg PO/IV every 4–6 h</td>
</tr>
<tr>
<td><strong>More sedating</strong></td>
<td></td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>10 mg PO every 4–6 h</td>
</tr>
<tr>
<td>Promethazine</td>
<td>25 mg PO or suppository every 4–6 h</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.5–2 mg PO/IV every 6–8 h</td>
</tr>
<tr>
<td>Diazepam</td>
<td>2–10 mg PO every 6–8 h</td>
</tr>
</tbody>
</table>

*PO* by mouth, *IV* intravenous.
clinical features fit with a specific peripheral vestibular disorder, then the likelihood of a serious central disorder is extremely low. CT scans are not a valid discriminator of central versus peripheral vertigo presentations in the emergency department. More research is needed to determine which patients are likely to benefit from neuroimaging in the acute setting.

References