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## Vertigo and Dizziness in the Emergency Department

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### Abstract

Understanding three peripheral vestibular disorders – vestibular neuritis, benign paroxysmal positional vertigo, and Meniere’s disease - is the key to the evaluation and management of vertigo and dizziness presentations in the emergency department. Each of these benign disorders is a common cause of a broad category of dizziness presentation. In addition, each of these disorders has characteristic features that allow for a bedside diagnosis. An effective strategy for “ruling-out” a serious disorder – such as stroke – is “ruling-in” a peripheral vestibular disorder. In this review, a focus is on the key features of these disorders.

### Keywords

Vertigo; dizziness; emergency department; stroke; vestibular neuritis; benign paroxysmal positional vertigo

### Introduction

Dizziness can be a problematic presentation in the emergency department, both from a diagnostic and a management standpoint. Dizziness is among the most common reasons that patients present for an evaluation.<sup>1</sup> In terms of signs and symptoms, overlap exists among the many potential causes. The report of symptoms can be vague, inconsistent, or unreliable.<sup>2</sup> Life-threatening disorders can masquerade as benign disorders,<sup>3-7</sup> but tests ordered to screen for life-threatening disorders are often insensitive.<sup>8</sup>

Patients presenting with vertigo and dizziness in the emergency department typically fall into one of the following three categories: acute severe dizziness, recurrent attacks of dizziness, or recurrent positional dizziness. (Table) A benign peripheral vestibular disorder is the most common cause within each of these categories and fortunately each of these disorders – vestibular neuritis, benign paroxysmal positional vertigo, and Meniere’s disease - is characterized by unique features allowing for a bedside diagnosis. Often, the most effective way to “rule-out” a life-threatening disorder is to “rule-in” one of these peripheral vestibular disorders. Because of this, it is critical that physicians can identify the key features of these three common peripheral vestibular disorders. The time to consider a sinister disorder as the cause is when the presentation is atypical for a peripheral vestibular disorder or when other red flags are identified. (Figure 1)

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The focus of this review is the categories of vertigo and dizziness presentations and the peripheral vestibular disorder that corresponds to each category.

## Acute Severe Dizziness

The patient who presents with sudden onset severe dizziness - in the absence of prior similar episodes - has the “acute severe dizziness” presentation. Patients with acute severe dizziness appear ill due to the dizziness and accompanying nausea and vomiting. Impaired ability to walk is also common. Though rigorous epidemiological studies are lacking, the most common cause is an acute lesion – presumed viral in origin - of the vestibular nerve on one side, so-called vestibular neuritis.<sup>9</sup> The mechanism underlying vestibular neuritis is similar to that of Bell’s palsy. The seventh cranial nerve is affected in Bell’s palsy, whereas the eighth cranial nerve is affected in vestibular neuritis. Patients with vestibular neuritis nearly always report true vertigo, which is characteristically described as visualized spinning of the environment. The symptoms are typically severe for 1-2 days with gradual resolution over weeks to months. It is exceedingly rare to have more than one bout of vestibular neuritis, so an alternative diagnosis should be considered whenever more than one episode is reported.

It is now clear that a small stroke within the posterior fossa can present as acute severe dizziness, closely mimicking vestibular neuritis.<sup>3-5</sup> The first step to distinguishing vestibular neuritis from stroke is asking the patient about other neurological symptoms such as focal numbness, focal weakness, or slurred speech. Mild double vision can result from a peripheral vestibular lesion so this symptom is not a reliable discriminator. The next step is the physical examination. Patients with vestibular neuritis have highly characteristic exam features. Only in an extremely rare case can all of the vestibular neuritis exam features be mimicked by a stroke.

## Nystagmus in Acute Severe Dizziness Presentations

Nystagmus is a term used to describe alternating slow and fast movements of the eyes. These alternating movements give the appearance that the eyes are beating toward one or more directions. Patients with vestibular neuritis have a peripheral vestibular pattern of nystagmus. In this setting, the peripheral vestibular pattern is a unidirectional, principally horizontal pattern of nystagmus. This description means that the nystagmus beats is in only one direction (i.e., a left-beating nystagmus never converts to right-beating, or a right-beating nystagmus never converts to left-beating). Conversely, bi-directional gaze-evoked nystagmus (i.e., right beating nystagmus present with gaze toward the right, and left-beating nystagmus present with gaze toward the left side) is a central nervous system pattern of nystagmus.<sup>10</sup> Other central nervous system patterns are pure torsional nystagmus or spontaneous vertical (typically downbeat) nystagmus. With an acute peripheral vestibular lesion, the only pattern of nystagmus that can result is unidirectional nystagmus. In acute severe dizziness presentations, any other pattern should be considered a central nervous system sign. Patients often prefer to keep their eyes closed early on, but the eyes should be opened and the pattern of nystagmus defined.

Nystagmus in vestibular neuritis is spontaneous (i.e., present in primary gaze) for at least the first several hours of symptoms. Following this initial time period, the nystagmus may only be identified during gaze testing (i.e., having the patient look to each side) or if visual fixation is blocked. Patients can suppress peripheral vestibular nystagmus by visual fixation on a target, so removing the patient’s ability to fixate can bring out the spontaneous nystagmus. The simplest way to block fixation is to place a blank sheet of paper a few inches in front of the patient and then observe for spontaneous nystagmus from the side.

The reason for the characteristic pattern of nystagmus in vestibular neuritis is an imbalance in the peripheral vestibular signals to the brain. Normally, the peripheral vestibular system on each side has a baseline firing rate of action potentials that functions to drive the eyes toward

the other side. When the peripheral vestibular system on each side is intact, the input from each side is balanced so the eyes remain stationary. When an acute lesion occurs on one side, the input from the opposite side is unopposed. As a result, the eyes will be “pushed” toward the lesioned side. This movement of the eyes is the slow phase of nystagmus. When the eyes reach a critical point off center, the brain responds by generating a corrective eye movement to move the eyes back. This is the fast phase of nystagmus. Since the direction of the fast phase gives the appearance that the eyes are beating in that direction, an acute left peripheral vestibular lesion leads to spontaneous right-beating nystagmus. Over time, the asymmetry resolves or the brain compensates for the asymmetry.

### The Head-Thrust Test

A recently described bedside test, the “head-thrust test,” is now an important component of the bedside evaluation in acute severe dizziness presentations.<sup>11, 12</sup> The test allows the examiner to assess the vestibulo-ocular reflex (VOR) on each side. The VOR is the component of the vestibular system that triggers eye movements in response to stimulation. In different settings, the VOR has long been tested using the doll’s eye test of the coma exam and caloric stimulation (i.e., the laboratory caloric test or the bedside cold caloric test in a comatose patient). To test the VOR using the head thrust test, the examiner stands in front of the patient and grasps 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 5-10° movement of the patient’s head to one side. When there is a lesion of the VOR on one side – as occurs with vestibular neuritis – 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.<sup>12</sup> 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.<sup>10</sup> 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 by the side the head moved toward. Thus a patient with vestibular neuritis of the left side will present with right-beating unidirectional nystagmus and have a positive head thrust test with movements toward the left side.

### Vascular Causes of Acute Severe Dizziness

Although vestibular neuritis is the most common cause of the acute dizziness presentation, no laboratory or imaging test exists to confirm a viral etiology. A peripheral vestibular lesion can be caused by a vascular occlusion of the blood supply to the peripheral vestibular components, though presumably this cause is much less common.

Stroke should be a serious consideration in the patient who presents with the acute dizziness presentation. Dizziness is a symptom of stroke in 50% of stroke presentations.<sup>13</sup> Most stroke patients that report dizziness as a symptom have other prominent central nervous system features, but a small stroke of the cerebellum or brain stem can present with isolated dizziness (i.e., dizziness without other accompanying central nervous system signs or symptoms). In a population-based study, about 3% of patients with dizziness had a stroke etiology, but less than 1% of patients with isolated dizziness had stroke as the etiology.<sup>14</sup> However, a prospective study of 24 patients with acute severe dizziness reported 6 patients (25%) with stroke etiology.<sup>3</sup> Patients with stroke presenting as isolated dizziness may report imbalance, true vertigo, a more vague dizziness sensation, or a combination of these. Nausea and vomiting are also common, as they are with vestibular neuritis. Unfortunately, computerized tomography (CT) scans are an extremely insensitive test for acute stroke presentations in general,<sup>15</sup> and particularly so for infarction within the posterior fossa.<sup>16, 17</sup> A stroke within the posterior fossa may not appear on a CT scan for days or weeks because of artifacts or poor resolution.

Because of this, CT should never be considered as a means of excluding stroke. Magnetic resonance imaging (MRI) is a much more sensitive test, but is not a practical test to screen for stroke in emergency department dizziness presentations. Like CT, the sensitivity of the test is the lowest for stroke of the posterior fossa.<sup>18</sup>

The key features discriminating stroke from vestibular neuritis are the pattern of nystagmus and the results of the head thrust test. Down-beating nystagmus or bidirectional gaze-evoked nystagmus are both immediate indications that the localization must be in the central nervous system. These patterns are not caused by lesions of the peripheral vestibular system. This is the reason that an examination of ocular movements is required before a diagnosis is even considered. Another highly suspicious pattern of nystagmus is a pure torsional pattern. There are now case reports of patients who have unidirectional horizontal nystagmus and a stroke etiology so the pattern of nystagmus should not be the sole criterion.<sup>3-5</sup> A patient with unidirectional nystagmus, a positive head thrust in the direction opposite the fast phase of nystagmus, and no other neurological features can be diagnosed with vestibular neuritis with a high level of certainty. It would take a well placed and small stroke to cause the peripheral vestibular pattern of nystagmus and a corresponding positive head thrust test without any other central nervous system features. Though all patients with vestibular neuritis are unsteady walking, the inability to walk is another red flag.<sup>5</sup> Finally, a person's risk for stroke based on stroke risk factors should be considered. Though no validated scale exists to grade stroke risk based on stroke risk factors in this population, a stroke work-up is reasonable in patients with a high risk for stroke. One should not be over reliant on stroke risk factors as discriminators, however, since other stroke mechanisms – such as arterial dissection – occur in the absence of stroke risk factors.

### Management of Acute Severe Dizziness

The management of the acute dizziness presentation begins with supportive care. If stroke is suspected then a neuro-imaging study should be considered. Though CT could serve as the initial study, a normal result on CT should provide little confidence that stroke can be excluded. In this situation, an MRI or hospital admission for close observation should be considered. If stroke is confirmed to be the cause and the patient presents within three hours of onset, thrombolytic treatment should be considered. A short course of corticosteroids should be considered for patients with vestibular neuritis. A randomized controlled trial showed that patients with vestibular neuritis treated with corticosteroids within three days of symptom onset had a higher likelihood of recovery of the peripheral vestibular caloric response at 12 months.<sup>19</sup> However this study did not test whether the patient's functional or symptomatic outcome improved, and corticosteroids are not without potential side effects. After the initial severe symptomatic time period, it is important that patients resume activities because this helps the brain to compensate for the asymmetry of vestibular signals. A formal vestibular therapy program has been shown in a randomized trial to improve outcomes in patients with vestibular neuritis.<sup>20</sup>

### Recurrent Positional Dizziness

Patients with positional dizziness have symptoms triggered by certain head positions. In acute presentations, patients are often more frightened by symptoms than debilitated by them.

Benign paroxysmal positional vertigo (BPPV) is the likely cause in patients reporting brief recurrent attacks of dizziness triggered by changes in head position. It is important to recognize this cause because it can be readily treated at the bedside and because identification of the key features is the most effective way to exclude a central nervous system cause of positional dizziness. Important points about BPPV are that the dizziness episodes last less than one minute and patients are normal in between episodes. Sometimes nausea or a mild lightheadedness can

persist longer than one minute, but any patient reporting positional dizziness lasting longer than one minute should be carefully scrutinized for other potential causes. A patient with dizziness from any cause will feel worse with certain position changes, but the patient with BPPV has dizziness that is triggered by positional changes and then returns to normal between attacks. Patients with vestibular neuritis are often misclassified as BPPV because the symptoms improve when the patient remains still and worsen with movement, but that is very different than the patient who returns to normal at rest.

BPPV occurs when calcium carbonate debris dislodge from the otoconial membrane in the inner ear and then inadvertently enter a semicircular canal.<sup>21</sup> The debris is typically free-floating in the canal so that head movements will trigger the symptom. The most common semicircular canal affected is the posterior canal because of its anatomic location. However, the particles can also enter the horizontal canal, or very rarely the anterior canal. It is important to be aware of the different variants of BPPV since each has unique examination features.<sup>22</sup> The most common triggers for BPPV episodes are extending the head back to look up (top shelf vertigo), turning over in bed, or getting in and out of bed. Posterior canal BPPV is the most important type to be able to identify because it is the most common type.

### Positional Testing and Particle Re-Positioning

When the patient with posterior canal BPPV is placed in the head-hanging position (Dix-Hallpike test) with the head turned toward the affected side, a burst of upbeat and torsional nystagmus is seen. (Figure 2) Turning the head toward one side for the Dix-Hallpike test lines the plane of the posterior canal on that side up with the movement of the test. The duration of nystagmus is typically 15-25 seconds. The Epley maneuver – a curative bedside maneuver - can then be used to reposition the debris.<sup>21, 23</sup> Success of the maneuver can be confirmed by retesting. If nystagmus continues to be triggered by the Dix Hallpike test, the Epley maneuver can be repeated.

When the particles are in the horizontal canal, the nystagmus triggered by head movement is a horizontal nystagmus rather than the vertical-torsional nystagmus seen with BPPV of the posterior canal.<sup>22</sup> If the patient lies supine and turns the head to either side - thus in the plane of the horizontal canal - nystagmus will be triggered. The two potential patterns of nystagmus in horizontal canal BPPV are 1) right beating nystagmus after head turns toward the right side, then left beating nystagmus after head turns toward the left side, and 2) left beating nystagmus after head turns toward the right side and right beating nystagmus after head turns toward the left side. Which pattern occurs depends on where the debris is located within the horizontal canal. The nystagmus of horizontal canal BPPV typically last longer than nystagmus triggered by posterior canal BPPV.<sup>22</sup> BPPV from the horizontal canal can be more difficult to treat than posterior canal BPPV. A common re-positioning strategy is to have the patient roll toward the unaffected side (360 degrees) in 90 degree increments.<sup>24</sup> The unaffected side is generally the side that triggers less severe nystagmus. Another approach is simply to instruct the patient to lie on the unaffected side for hours, which can be done at home rather than in the emergency department.<sup>25</sup>

BPPV of the anterior canal is quite rare. When present the Dix-Hallpike test will trigger a short burst of down-beating nystagmus.<sup>22</sup> This cause will also respond to the Epley maneuver.

### Central Positional Dizziness

Central positional vertigo stems from a lesion of the cerebellum or the brainstem. Positional vertigo and nystagmus are common features of a Chiari malformation, cerebellar tumor, multiple sclerosis, migraine vertigo, and degenerative ataxia disorders. As with the acute dizziness presentations, the key to distinguishing a central nervous system disorder from a

peripheral vestibular disorder is the pattern of nystagmus. The most common pattern of central positional nystagmus is pure down-beating nystagmus that lasts as long as the position is held. Pure torsional nystagmus is another type of central positional nystagmus. The pattern of nystagmus seen with horizontal canal BPPV can also be caused by a central lesion. A general rule is that a central nervous system cause of positional nystagmus should be considered whenever the pattern of nystagmus is a persistent downbeating nystagmus, pure-torsional nystagmus, or whenever the nystagmus is refractory to repositioning maneuvers.

## Recurrent Attacks of Dizziness

Patients with recurrent attacks of dizziness will report prior episodes that were similar to the current attack. The duration of the attacks is highly variable but can be helpful in discriminating among the potential causes. Patients may present during an attack or after the attack has already ended.

Meniere's disease is the prototypical disorder characterized by recurrent spontaneous episodes of dizziness. Patients with this disorder have severe episodes of dizziness – generally true vertigo – with nausea, vomiting, and imbalance.<sup>26</sup> The episodes are accompanied by unilateral auditory features, either hearing loss, a loud 'roaring' tinnitus, or severe ear fullness. Episodes are variable in duration but generally will last for hours. The type of tinnitus experienced by patients with Meniere's disease is typically very different from the more common constant bilateral high pitched tinnitus or the fleeting mild tinnitus that most people experience at some time. The tinnitus in Meniere's disease is usually a very loud roaring sound in one ear. Though the nystagmus may not follow all the rules of peripheral vestibular nystagmus described in vestibular neuritis, the same red flags for central causes (i.e., down-beat, pure-torsional, or bi-directional gaze-evoked nystagmus) apply. The head thrust test is generally normal in patients with Meniere's disease since the peripheral vestibular system is intact.

Transient ischemic attacks (TIA) should be a concern in the patient who presents with new onset recurrent spontaneous attacks of dizziness. TIA generally lasts for minutes, less than is typical for Meniere's disease. Recurrent spontaneous attacks of dizziness is often the initial symptom of an impending basilar artery occlusion.<sup>7</sup> Transient ischemia should be a leading concern when the patient reports recent onset brief attacks, particularly if the attacks are increasing in frequency (i.e., a crescendo pattern). Auditory symptoms can also accompany an ischemic etiology since the anterior inferior cerebellar artery can be involved. As with TIA in general, a CT scan is not helpful for ruling out this cause. CT angiography (CTA) or MR angiography (MRA) are the tests to consider ordered when the integrity of the posterior circulation needs to be assessed.

## Other potential causes

Migraine is the great mimicker of all causes of dizziness. Symptoms can present as an acute severe attack, positional episodes, or recurrent spontaneous attacks.<sup>27, 28</sup> The exam features can suggest a peripheral vestibular or central nervous system localization. As with migraine in general, a strong genetic component is felt to play a role in addition to numerous environmental, food, or lifestyle factors. Patients frequently report that stimuli - such as light, sound, or motion - can trigger or aggravate the symptom. The diagnosis of migraine vertigo, unfortunately, remains a diagnosis of exclusion. Thus, if the symptom is new in onset, diagnoses such as stroke or TIA should still be considered if the features do not fit for a peripheral vestibular disorder. When these causes are excluded, migraine becomes a leading candidate. The main supportive features of this diagnosis are a lack of the key features of the other common disorders, and an onset at least several months prior to presentation. A headache around the time of the dizziness is frequently reported but is not required. Triptan medicines do not generally improve migraine dizziness symptoms.<sup>29</sup>

Panic disorder is another common cause of dizziness symptoms. Most patients with panic disorder will have the other typical symptoms of panic disorder but the dizziness symptom may be the most bothersome. If a diagnosis of panic disorder is not clear based on the history and physical examination, then a work-up may be warranted to exclude the other potential causes.

General medical causes are also common causes of dizziness, though typically true vertigo is not reported. In addition, the finding of nystagmus means that either peripheral or central components of the vestibular system are involved. Thus, nystagmus generally rules out most general medical disorders. A cardiac arrhythmia or myocardial infarction should be considered in the appropriate setting.

## Misconceptions

A recent physician survey highlights some common misconceptions that exist regarding dizziness presentations.<sup>30</sup> Some physicians feel that the report of 'isolated dizziness' can discriminate a stroke etiology from a benign peripheral vestibular disorder. While it is true that the lack of other associated neurological symptoms reduces the likelihood of stroke diagnosis, numerous reports in literature demonstrate how closely stroke can mimic vestibular neuritis.<sup>3-5</sup> A second misconception is that defining the type of dizziness sensation can be used to discriminate benign from sinister disorders. Some feel that lightheadedness or other vague dizziness sensations make a stroke diagnosis less likely than a report of true vertigo. However, a recent population based study showed that patients reporting vertigo do not have a higher odds of stroke diagnosis than patients reporting "dizziness."<sup>14</sup> A third misconception is that dizziness exacerbated by head motion indicates a benign disorder. The fact is that dizziness from any cause can worsen with head movements. The characteristic of BPPV is that the dizziness symptom is triggered by a head movement, not simply worsened by the movement. Finally, some physicians report that a negative CT scan of the brain rules out stroke. However, a CT scan is an insensitive test for acute stroke, particularly stroke within the posterior fossa.<sup>15-17</sup> Thus a negative CT scan should not be considered adequate for excluding stroke.

A common theme among these misconceptions is an over-reliance on the patient's description of symptoms and an over-reliance on CT scans. In this review, most emphasis is placed on the critical components of the examination.

## Symptomatic Treatment

Patients who present with severe nausea and vomiting typically require intravenous fluids during the emergency department stay. When drug therapy is necessary to reduce symptoms in the acute setting, generally two different categories of drugs are used: vestibular suppressants and antiemetics. An important point is that medicines to symptomatically reduce dizziness can be effective for acute attacks, but are generally not effective as prophylactic agents. Thus, these medicines are best used during the emergency department visit and not as new daily medicines. When taken on a daily basis, the medicines are more likely to result in side effects or reduce the brain's ability to compensate (as with vestibular neuritis).

The major classes of vestibular suppressants include antihistamines, benzodiazepines, and anticholinergics. Although the exact mechanism of action of these drugs is unclear, most appear to act at the level of the neurotransmitters involved in propagation of impulses from primary to secondary vestibular neurons and in maintenance of tone in the vestibular nuclei. Antiemetic drugs are directed against the areas in the brain controlling vomiting. Dopamine, histamine, acetylcholine, and serotonin are transmitters thought to act on these sites to produce vomiting. Most of the vestibular suppressants have anticholinergics or antihistamine qualities, giving them antiemetic properties in addition to the effects on vertigo. When nausea and vomiting are

prominent, a mild vestibular suppressant (such as meclizine) can be combined with an antiemetic (such as prochlorperazine) to control symptoms. These medicines typically have central dopamine antagonist properties and are believed to prevent emesis by inhibition at the chemoreceptor trigger zone. A major side effect of both medicine categories is drowsiness, though this effect probably contributes to the therapeutic effect as well.

Few randomized controlled trials have been conducted on the symptomatic treatment of acute dizziness. In one study, 74 patients were randomized to treatment with either 2mg of intravenous lorazepam or 50mg of intravenous dimenhydrinate.<sup>31</sup> The results suggested that dimenhydrinate was more effective for reducing symptoms and improving the ability to ambulate. Dimenhydrinate also resulted in less drowsiness.

## Conclusions

The ability to identify the key features of the three most common benign peripheral vestibular disorders allows the evaluating physician to sort through the most common types of dizziness presentations in the emergency department. The most effective way to “rule-out” a serious cause is to “rule-in” a benign inner ear disorder. When the features are atypical or other red flags appear, sinister causes should be considered. The two presentations with the most at stake are the following: 1) acute severe dizziness when the presentation is atypical for vestibular neuritis, and 2) recurrent attacks of dizziness when the attacks are recent in onset and last only minutes. For these two presentations, an ischemic etiology should be strongly considered even if dizziness is the only symptom and the CT scan is normal. For recurrent positional dizziness, a sinister disorder – such as a structural posterior fossa lesion – should be considered when a central positional pattern of nystagmus is seen or when the patient does not respond to particle repositioning techniques. However, generally central positional nystagmus is caused by disorders that require a less urgent evaluation than acute severe dizziness or recurrent attacks of dizziness.

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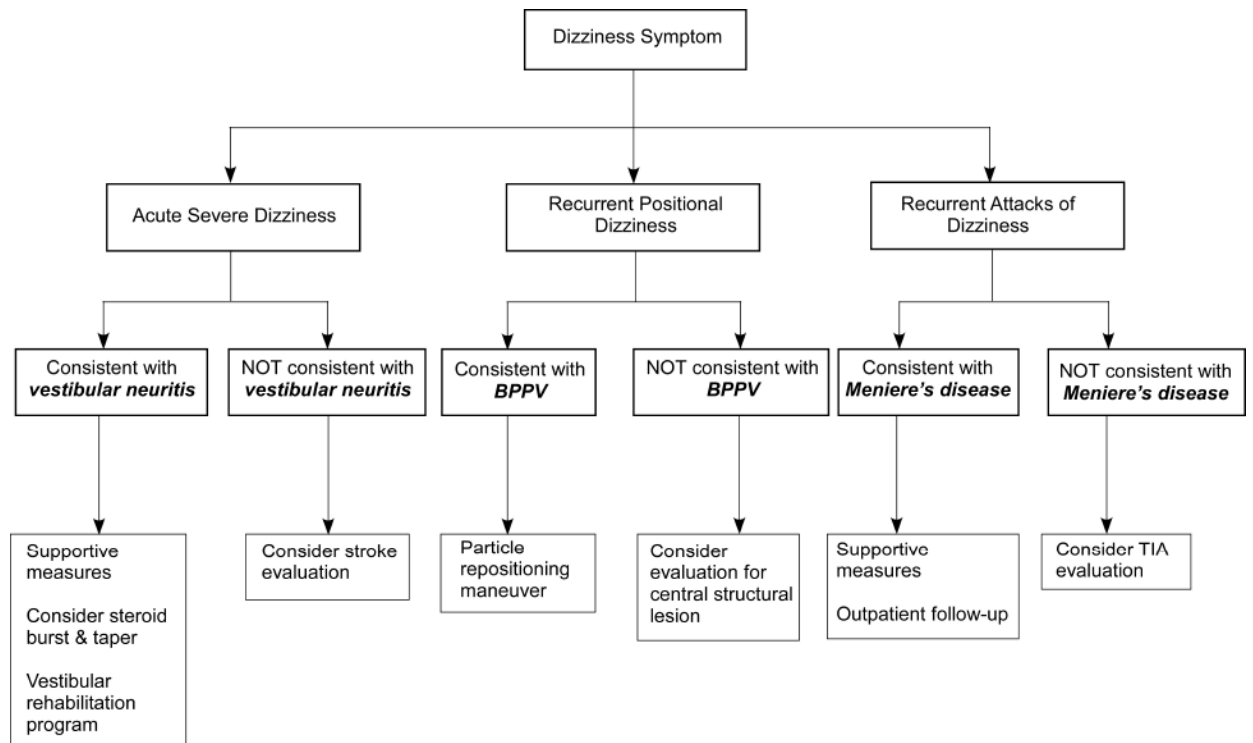
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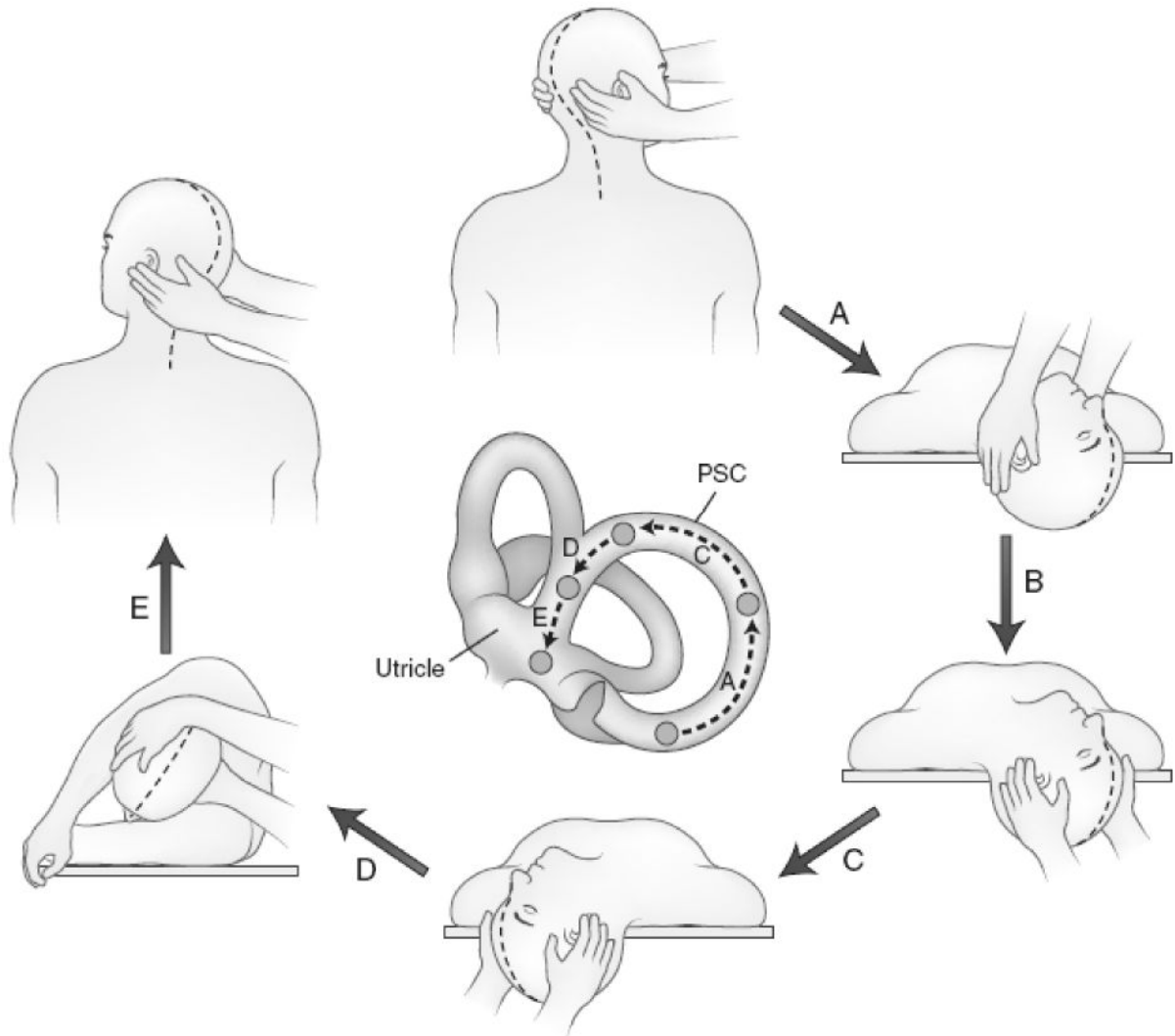


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**Figure 1.** Diagnostic and management algorithm for common dizziness presentation categories. BPPV = benign paroxysmal positional vertigo; TIA = transient ischemic attack.



**Figure 2.**

Epley maneuver for 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 seconds. (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 seconds. (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. Permission granted by Elsevier Ltd.

**Table**  
Summary of the features of the most common categories of dizziness presentations.

Dizziness Presentation Category	Main symptoms	Peripheral vestibular signs	Central nervous system signs <sup>***</sup>	Potential causes
<b>Acute Severe Dizziness</b>	Sudden onset, severe & constant dizziness, nausea and vomiting, and imbalance	Unidirectional Spontaneous nystagmus, positive head thrust test	Down-beat or bidirectional gaze-evoked nystagmus, severe imbalance	PV: Vestibular neuritis CNS: Stroke
<b>Recurrent Positional Dizziness</b>	Dizziness attacks triggered by head movements	-Attacks last less than 1 minute. Normal in-between attacks. -Dix-Hallpike test: Burst of upbeat torsional nystagmus. -Epley maneuver: Resolution of signs & symptoms.*	-Attacks can be of short or long duration. Less severe dizziness symptoms may persist between attacks. -Dix-Hallpike test: Persistent down-beating nystagmus or pure torsional nystagmus. -Epley maneuver: No effect.	PV: BPPV CNS: Chiari malformation, cerebellar tumor, degenerative ataxia.
<b>Recurrent Attacks of Dizziness</b>	Spontaneous attacks of dizziness	Duration: >20 minutes to hours. Associated unilateral hearing loss, roaring tinnitus, or ear fullness	Duration: Minutes. New onset and crescendo pattern.	PV: Meniere's disease CNS: TIA

\* See text for details regarding less common types of BPPV

\*\* Any other CNS symptom as well (speech alteration, focal weakness, focal sensory features).

PV = peripheral vestibular; CNS = central nervous system; TIA = transient ischemic attack