



SIGNS AND SYMPTOMS OF CENTRAL VESTIBULAR DISORDERS

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Dizziness can come from many sources. Therefore, one of the goals of a healthcare provider is to start to rule in or out possible causes of a patient's symptoms of dizziness. One of the distinctions that may need to be made is if the dizziness the patient is reporting is coming from the **peripheral vestibular system** (the labyrinth of the inner ear, and the pathways/nerves connecting to the brainstem) or the **central vestibular system** (the brain and brainstem). Being able to find the vestibular system involved is key in helping the healthcare provider decide on further testing, determine the urgency of the symptoms, and develop treatment plans. This article will review the signs and symptoms that are associated with dizziness originating from the central vestibular system.

The symptoms being reported by the

patient can be very useful as a first filter to narrow in on a possible cause of their dizziness and assist with interpretation of findings from formal laboratory and clinical tests. While the patient's symptoms can be a clue into the origin of their dizziness, a provider often needs to further question the patient regarding their symptoms to fully understand what the patient is experiencing. The most common term used by a patient is that they are *dizzy* or having *dizziness*. The term *dizziness* is a general term that can encompass imbalance, lightheadedness, objective vertigo (objects in the room appear to move) and subjective vertigo (the sensation of spinning is within the patient's head, objects in the environment are stationary), or combinations of the above. In 2009 the International Classification for Vestibular Disorder (ICVD) committee of the Barany Society (an international society for the

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study of clinical and research aspects of dizziness and balance disorders) published a document to attempt to define the symptoms expressed by patients with 'dizziness' (see Suggested Resources). In this document the following major definitions were put forth:

- **Vertigo** - The sensation of self-motion or motion of the external environment when no such movement is occurring.
- **Unsteadiness** - The sensation of being unstable sitting, standing and walking; can include ataxia and falls (meant to include terms like imbalance and disequilibrium).
- **Dizziness** - A sensation of a distortion of the spatial orientation, but without any perception of self or environmental motion, and not unsteadiness. This can include sensations such as lightheadedness and disorientation.

When examining a patient's current and past symptoms, there are four areas of information that play a major role in helping to provide a first-pass judgment as to whether the symptoms would most likely be of peripheral or central origin.

1. Temporal course (timing) of the symptoms: If the symptoms are

paroxysmal (sudden onset of symptoms which then subsides), would the typical duration be measured in seconds, minutes, hours, or days, and what is the range from the shortest to longest? If continuous, are there exacerbations in the intensity of the symptoms, and what is the duration of those exacerbations?

2. Circumstances surrounding the onset of the symptoms: Are the symptoms occurring in a spontaneous manner or are the symptoms provoked by head or visual movement, visual complexity, or visual patterns?
3. The characteristics of the symptoms: Specifically, what does the patient mean when he or she uses the term *dizziness*? Is the patient experiencing true objective external vertigo, subjective (internal) vertigo, unsteadiness, lightheadedness, unexplained falls, or combinations of these symptoms? Also, are the symptoms accompanied by any of the following: nausea and vomiting, headaches, heart palpitations, feelings of panic, drop attacks (sudden falls with or without loss of consciousness), or any of the "Ds"

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(diplopia = double vision, dysphagia = difficulty swallowing, dysarthria = difficulty with speech, dysmetria = lack of coordination). The importance of the “Ds” is that any of these symptoms on a consistent, unexplained basis is an indicator of involvement of the posterior fossa of the brain (containing the brainstem and cerebellum), which can change urgency and course of treatment. The other associated symptoms can occur with either peripheral or central lesions, or damage.

4. Status of the patient’s hearing by their perception: Do they have unilateral (one-sided) or bilateral (both sides) perceived hearing loss? Is this slowly progressive and is one ear worse than the other? Do they have sudden changes in hearing or fluctuations in hearing? Are they experiencing tinnitus and/or aural fullness?

Before looking in more detail at the symptom characteristics that are typical for central versus peripheral, a brief discussion of the pathophysiology behind true vertigo will be useful.

Vertigo, independent of where it is coming

from, results from sudden, asymmetrical neural activity. The asymmetry in neural activity could be coming from anywhere in the vestibular system from the inner ear to the brain. This is why it is key to look at the other signs and symptoms that the patient is presenting with (such as the “Ds” mentioned above) to determine the involved structures. Even once the practitioner believes that symptoms may be originating from the brain, they can further drill down on location as not all locations of the brain will produce the true vertigo sensation.

One can make a broad generalization regarding the symptoms that are more likely to be of peripheral origin compared to those of central origin. Table 1 shows this generalized separation. As shown in Table 1, when a peripheral lesion is involved, onset is more often than not sudden and usually memorable as the patient will be able to tell you a specific date and in some cases a specific time. The most common initial symptom will be true vertigo (seeing objects moving in the room). And unless there is an acute vestibular crisis (e.g., vestibular neuronitis or labyrinthitis), the true vertigo should last less than 24 hours. In contrast, lesions of central origin are usually slow in development, with the patient unable to

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give you a time of onset. This can also be true for symptoms from non-vestibular involvement (e.g., peripheral neuropathy). If symptoms are of sudden onset with vertigo or imbalance and they do not involve the labyrinthine or eighth cranial nerve, then you usually have accompanying symptoms suggesting posterior fossa involvement ("Ds"). The principal symptom is more likely to be that of unsteadiness and lightheadedness with vertigo absent.

In cases where psychological conditions such as anxiety are a major portion of the

disorder, the symptoms may be very vague, with the patient struggling to articulate his or her experiences. Patients with symptoms steaming from a physiological condition are more likely to present with subjective (internal) sensation of movement that is a slow spinning within the head or a rocking that is present on a constant basis (at least > than 50% of the time) and exacerbated by visual motion and/or complex visual patterns as seen with Persistent Postural-Perceptual Dizziness (PPPD-see Suggested Resources).

Table 1: Generalized symptoms of peripheral and central origin.

Peripheral Origin	Central or non-vestibular Origin
Sudden, memorable onset	Sudden onset of vertigo, lightheadedness/imbalance with one of the Ds
Typically true vertigo at onset	Slow-onset of imbalance, standing, and walking
Paroxysmal spontaneous events <24 hours	Vague symptoms of any character
Head movement provoke symptoms <2 minutes	Slow, subjective vertigo (spinning within the patient's head) lasting 24/7
Vestibular crisis: sudden onset vertigo slowly improving from continuous to head movement provoked in days	
More likely to have auditory involvement	

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Unfortunately, not all patients present with a clearly defined central or peripheral cause of their symptoms. While patients will have a dominant group of symptoms that will be more closely tied to peripheral or central origin, there will be those who have a full mix of the two groups.

Therefore, the symptoms listed earlier can serve as a guide, but not necessarily lead to a final diagnosis. Just as we have done with symptoms, the signs (either direct office examination or formal vestibular and balance laboratory findings) that are presented, when mixed with the symptoms, begin in most patients to present a clearer picture of the origin of the dizziness. Table 2 presents a generalization of signs divided as was done for symptoms into peripheral and central origin.

One of the key signs that practitioners will examine is nystagmus. Nystagmus is a back and forth movement of the eyes with the eyes often moving one direction slowly and the other direction more quickly. As presented in Table 2, nystagmus can assist in the diagnosis of central versus peripheral origin. The lesion of peripheral origin is likely to present with direction-fixed (fast movement to the same direction all the time) or dominantly horizontal nystagmus (eyes move horizontally back and forth). The nystagmus, especially in

the subacute and chronic states, may only be seen when the patient is not able to fixate their vision on an object. In contrast, the lesion of central origin is more likely to present with pure vertical or torsional nystagmus, and if horizontal it is more likely to change direction based on the direction of the patient's gaze. Other contrasting features would be the absence of abnormalities on pursuit tracking (following an object with your eyes) and saccade testing (looking back and forth between two objects) in the peripheral lesion and the likelihood of seeing abnormalities on these tests in the patient with a central lesion.

Headshake testing in the horizontal or vertical direction, if nystagmus is produced, should be horizontal from either direction of shaking for the peripheral lesion and may well be vertical for the central lesion. Headshake testing is performed by the practitioner rotating the patient's head back and forth either horizontally or vertically while the patient's eyes are closed, and then asking the patient to open their eyes. The practitioner then watches for nystagmus. Although most persons with sudden onset of severe peripheral origin vertigo with nystagmus say they could not walk at onset of their symptoms, they are able to coordinate

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their legs to be able to walk even though they may well need assistance secondary to the severe unsteadiness. However, central vestibular lesions may produce a

situation where at the onset of symptoms, if they are sudden, the patient cannot coordinate their legs in a walking pattern and cannot walk even with assistance.

Table 2: Generalized signs for peripheral and central vestibular lesions

Peripheral Origin	Central or Non-Vestibular Origin
Direction-fixed, dominantly horizontal nystagmus	Direction-changing nystagmus
Abnormal vestibulo-ocular reflex, via head thrust or caloric testing	
Nystagmus more likely to be seen with fixation removed	Nystagmus more likely enhanced with fixation present
Nystagmus more likely to be exacerbated when gazing in the direction of the fast component of the jerk nystagmus (Alexander’s law)	Nystagmus more likely to be pure vertical or pure torsional
Nystagmus more likely to be exacerbated post horizontal headshake - horizontal nystagmus	Nystagmus post-headshake vertical
Pursuit tracking and saccade performance normal (or age dependent)	Likely to have abnormal performance on pursuit and/or saccades
If sudden onset, can stand and walk with assistance	If sudden onset, likely not to be able to stand and walk even with assistance

When considering the signs that represent possible central system involvement, abnormalities in pursuit tracking (following an object with your eyes) and in random saccade (looking back and forth between two objects) testing are such that they are specific to central system deficits. There

are no peripheral lesions that are known to produce abnormalities in either of these two tests, with the exception of spontaneous nystagmus appearing during pursuit or saccade testing. For further information on these two tests, please refer to the suggested resources at the end

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of the article. The two other principal indicators of central involvement are the type of nystagmus (pure vertical and pure torsional) and nystagmus provoked by eccentric (off-center) gaze. Finally, as nystagmus of peripheral origin behaves differently than nystagmus of central origin when the patient is able to clearly look at an object, the practitioner will also examine what happens to the patient's nystagmus when they are able to visually fixate on an object.

A caveat to the above discussion of central origins is that the signs and some of the symptoms that we would associate with central nervous system involvement can be produced by migraine headaches. Virtually all of the abnormal findings we have discussed for both central and peripheral lesions, as well as abnormal caloric and rotational chair findings, have been reported in patients where migraine headaches were the principal cause of their dizziness. In diagnosing a person with Vestibular Migraine as the cause for their dizziness, first the individual has to be determined to currently be or have evidence in the past of being a migraine sufferer. For more information regarding Vestibular Migraines, please refer to the suggested resources at the end of the article.

This article has provided a brief overview of what practitioners are looking for when determining if a patient's reports of dizziness are more of a central or peripheral origin. If you are interested in reading more on this subject, please refer to the suggested resources below.

Suggested Resources

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