Examination of the Patient with Dizziness or Imbalance

Gregory T. Whitman, MD

KEYWORDS

• Examination • Dizziness • Vertigo • Balance • Vestibular • Nystagmus • Gait

KEY POINTS

- Because of the multisensory nature of balance and vestibular function, the clinician seeing a dizzy patient, must perform a comprehensive neurological assessment.
- Altered mental status and abnormalities of vision may disturb balance and gait.
- The direction and duration of any nystagmus should be recorded, and one should note whether nystagmus is spontaneous, gaze evoked, or positional.
- Past pointing tests, Romberg tests, and the Fukuda stepping test may disclose imbalance.

INTRODUCTION

"Dizziness" may signify vertigo (an illusory sense of movement of the environment or self), lightheadedness (eg, a floating sensation), or other sensations that defy description. Many people with dizziness have an abnormality of gait or postural stability, necessitating inclusion of gait and balance in the clinical approach to dizziness.

Assessment of dizziness in US emergency rooms reportedly costs $4 billion per year.[1] Annual medical expenses due to falls in the United States were reported to be $50.0 billion.[2] Despite these high costs, many patients with dizziness or imbalance are discharged from the hospital without a clear diagnosis, suggesting the need for improved physical examination approaches.

One might reasonably ask: What is the rationale for combining dizziness and imbalance into one category? Some patients with dizziness report their symptoms are localized within or around the head and that their gait is normal. For others, the main problem is abnormality of gait, the head feels normal. In practice, though,

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Department of Otolaryngology, Harvard Medical School, Massachusetts Eye and Ear Infirmary, Massachusetts Eye and Ear Balance and Vestibular Center, 250 Pond Street, Braintree, MA 02184, USA
E-mail address: gtw3@cornell.edu

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most patients with dizziness or imbalance fall on a spectrum between the patient with head-related symptoms alone, and the patient with balance and gait symptoms alone.

The reader is assumed to be familiar with the basic neurologic examination. This topic will not be reviewed in detail. The routine neurologic examination, though, is critical, because of the extensive differential diagnosis. The differential diagnosis of dizziness is reviewed elsewhere.[3] The primary goals of this review are as follows:

1. To outline a systematic approach to physical examination of the patient with dizziness or imbalance,
2. To discuss useful and frequently overlooked neurologic signs that are useful in clinical practice for making a diagnosis.

First and foremost, one has to acknowledge the multisensory nature of balance and vestibular function. Balance function may remain surprisingly normal (compensated), until a large number of neurons and systems have been lost. One is surprised to encounter patients with substantially absent peripheral vestibular reflexes but near normal gait. Although the latter situation may be surprising, it is also common in otherwise young, healthy patients who have lost vestibular function.

At the same time, a diverse array of neurologic disturbances may contribute to imbalance. That is, gait disorders are commonly "multifactorial." Consider a set of 3 disorders, as follows:

1. Unilateral benign paroxysmal positional vertigo (BPPV),
2. Mild diabetic peripheral neuropathy,
3. Bilateral impairment of visual acuity, due to cataracts.

It is by no means clear that any of the formerly mentioned 3 problems in isolation would greatly disturb balance. Combined, however, the set of all 3 diagnoses would almost assuredly cause imbalance. A critical principle, then, is that one must maintain a high index of suspicion that imbalance is multifactorial.

As elsewhere in Neurology, the assessment of multiple coincident lesions is challenging and benefits from the experience of the examiner. For the nonspecialist, the best approach to the possibility of multiple lesions may be to carefully catalog as many abnormal neurological findings as possible, in as much detail as possible, acknowledging that in the end, the combination of objective abnormalities may add up to an explanation.

ORTHOSTATIC VITAL SIGNS

Orthostatic vital signs are most useful when viewed as a stress test. The basic routine of checking vitals lying down and then sitting up is insufficient. Prolonged action of gravity may be necessary to cause enough pooling of blood below the heart to elicit orthostatic intolerance, except in cases where orthostatic vital sign measurements are hardly necessary. The patient who says, "When I stand up, I get lightheaded," should be encouraged to stand, for a few minutes, for example, while history taking is continued. The examiner should ask about symptoms (including blurred vision and dizziness) while the patient stands. Note should be made of any deterioration in cognitive function while upright, and serial sets of standing vital signs may be informative. It is common knowledge that a sustained fall in systolic blood pressure of at least 20 mm Hg, after moving from the sitting (or lying down) position to the standing position, is evidence for orthostatic

hypotension; however, one must not overlook the patient with possible orthostatic cerebral hypoperfusion, and marked clinical symptoms in the standing position, but who may not manifest overt hypotension. Although many approaches to orthostatic vital signs would be acceptable, it is wise to routinely observe dizzy patients, in the standing position, for at least 3 minutes, while monitoring for symptoms and/or changes of vital signs. If overwhelming dizziness develops during such a test, the patient should, for safety, be placed back into a sitting or lying down position and allowed to recover.

CERVICAL SPINE

The neck provides proprioceptive information to the brain and is thus a potential substrate for dizziness. Pain specialists are familiar with the dizziness occasionally elicited with performance of bilateral cervical spine injection procedures.[4] It is likely that application of a combination of tests involving error in position sense of the neck and nystagmus upon turning of the neck would improve diagnosis of patients with cervical spine disorders plus dizziness.[5,6] The best approach to the use of such methods, in practice, remains to be clarified. At present, the best the busy clinician can hope for with respect to diagnosing cervicogenic dizziness may be to perform range-of-motion testing in the yaw (horizontal), pitch (sagittal), and roll tilt (coronal) planes and ask the patient whether any of the "dizziness" symptoms are reproduced with certain neck positions or motions. If so, the clinician can then look for treatable cervical spine disorders. One should remember to consider the differential diagnosis of cervical artery dissection, of which neck pain is a common early feature. For that reason, along with the possibility of cervical myelopathy, the clinician considering a diagnosis of cervicogenic dizziness must be sure to look for abnormal findings on the basic neurologic examination that might lead one in a different direction.

MENTAL STATUS

Although the central vestibular system is often conceptualized in terms of posterior fossa structures, widespread brain areas are needed to integrate multisensory information in such a way as to create cues and impressions pertaining to space and motion. Such integrative function inevitably involves the cerebral cortex.

One should at least consider in passing the possibility that a chief complaint of "dizziness" may be localized to the cerebral cortex. Indeed, it is not unusual for a patient with Alzheimer disease to report "dizziness" that defies explication, even in the best of hands. This being the case, one should identify and record abnormal cognitive behaviors such as repetitive questioning. If present, these suggest short-term memory impairment. Cognitive function can be screened for by asking the patient to spell the word "world" backward. If there is any doubt about the patient's cognition, the clinician can assess further with a brief cognitive test battery such as the Montreal Cognitive Assessment.[7]

As part of the mental status examination, one should assess for anxiety. A common clinical conundrum is anxiety, or more properly an anxiety disorder, may be formulated as a cause of dizziness, a consequence of an inner ear disorder that causes dizziness, or a product of aggravation of an underlying anxiety disorder by an inner ear disorder.[8] In some cases, the anxiety is an important therapeutic target. A qualitative notation on the chart about the examiner's impressions of the patient's mood and affect contributes to tracking of this component.

VISION

Isolated loss of vision seldom causes a marked disturbance of balance. Like other sensory channels, however, vision does contribute to balance, and visual impairment contributes to imbalance. In particular, visual field cuts may be unobvious to the patient and may aggravate imbalance and increase fall risk.[9] The standard confrontation tests of visual fields are familiar to most physicians. In addition, visual fields can be assessed in detail at the bedside, using a laser pointer and a wall.[10] For patients who are users of bifocal, trifocal, or progressive lenses, walking should be assessed with and without the use of glasses. Declines in performance with the use of certain lenses should be noted.

FACIAL NERVE FUNCTION

The Ramsay Hunt syndrome consists of facial nerve palsy, combined with a painful rash involving the pinna or external auditory canal, plus vestibular and hearing deficits in varying proportions. This syndrome is presumed to often be due to latent virus reactivation. Treatment, which is beyond the scope of this review, is modeled after that used in the setting of Bell palsy. It is important to recognize the facial palsy, which may be relatively subtle in the context of severe dizziness. On examination, eye closure is less vigorous on the involved side, and the ipsilesional nasolabial fold is relatively flat. After some recovery, synkinesis may be noted, such that closure of the eye on the affected side is associated with aberrant twitching of facial muscles.

OTOSCOPY AND HEARING

Although a detailed ear examination is beyond the scope of this review, a few points warrant brief discussion. Applying positive and negative pneumatic pressure during otoscopy may elicit dizziness in pressure-sensitive disorders. These pressure-sensitive disorders, including the superior semicircular canal dehiscence syndrome[11] and occasionally Meniere syndrome.[12] In the instance of Ramsay Hunt syndrome (see earlier), otoscopy may disclose vesicles in the external auditory canal.

Hearing loss is a key feature of some disorders that cause dizziness. One must not, however, immediately assume that hearing loss is sensorineural. For example, cerumen blocking the external auditory canal may cause hearing impairment. At the same time, one should try to make a diagnosis of sensorineural hearing loss as early as possible, because of the potential need to treat sudden sensorineural hearing loss with corticosteroid medication. Sensorineural hearing loss that involves loss of acuity for low-frequency sound is a component of the diagnostic criteria for Meniere disease. Interestingly, there are also emerging data suggesting that hearing loss of any form may contribute to imbalance due to loss of sound cues about the spatial environment.[13,14]

The well-known Rinne test is typically performed with a 512-Hz tuning fork. If the patient reports that sound is heard louder through bone than air, the result suggests either a component of conductive hearing loss or a pseudoconductive hearing loss, due to a third mobile window, such as superior semicircular canal dehiscence.[15] It is helpful to have a low-frequency (128 or 64 Hz) tuning fork, because hearing asymmetries tend to be marked for this frequency when testing patients with Meniere. If whispered speech is poorly understood in either ear, a retrocochlear lesion, for example, vestibular schwannoma, should be considered.

NYSTAGMUS

The most common type of nystagmus, called jerk nystagmus, is a rhythmic eye movement having well-defined slow and fast phases. One should observe for the presence of spontaneous nystagmus, while the patient is sitting, looking straight ahead. In the case of acute unilateral peripheral vestibulopathy, and/or vestibular neuritis, and/or labyrinthitis, spontaneous nystagmus is predominantly horizontal and unidirectional with fast phases away from the lesion (a smaller torsional component may be evident). The amplitude of spontaneous, unidirectional nystagmus typically increases with gaze in the direction of fast phases.

The nystagmus of acute peripheral vestibulopathy becomes coarser when one eliminates fixation, using Frenzel video goggles. Alternatively, to eliminate fixation, one of the patient's eyes can be covered while the other eye is viewed with an ophthalmoscope and observed for nystagmus.

Smooth pursuit should be tested by asking the patient to slowly follow a finger to the right and left, and up and down, noting smoothness of eye movements. Saccadic smooth pursuit localizes to the brain. The specificity of the latter sign, however, is greater in younger patients.[16] Despite the high prevalence of saccadic smooth pursuit with increasing age, excessive degeneration of smooth pursuit function is reported to be associated with neurodegenerative brain diseases.[17]

Gaze-evoked and/or gaze-holding nystagmus may be defined as nystagmus that is absent when looking straight but is present when looking (maintaining gaze) in some directions. Gaze-evoked nystagmus may be seen in normal people with extreme eye deviation, for example, all the way to the right, but there is no widely accepted consensus on a definition of "extreme." Suffice it to say that the specificity of gaze-evoked nystagmus is highest when small deviations of eye position away from straight ahead elicit nystagmus.

Direction switching nystagmus (eg, right beating with right gaze, and left beating with left gaze) is a red flag for a possible brain lesion, as is pure vertical nystagmus, or vertical nystagmus with only a subtle torsional component. If direction switching nystagmus is asymmetric, that is, coarser with gaze in one particular direction, this suggests a focal, structural brain lesion.

Persistent downbeat nystagmus may be spontaneous, but it tends to be coarser on gaze to the right or left. It can be due to lesions of the cerebellum or at the craniocervical junction, as in the case of Arnold Chiari malformations. Rebound nystagmus localizes to the cerebellum. It is recognized as follows: the patient first is asked to follow a target such as the examiner's finger to an eccentric position, for example, all the way to the right, and then to make a saccade back to the midline, at which point a burst of nystagmus is seen, in the direction of the saccade back toward the midline.

An important type of nystagmus is that observed in patients with BPPV. As implied by the name of this condition, the nystagmus that is the hallmark of BPPV is paroxysmal and occurs in certain positions. The nystagmus occurs within seconds after moving into a provocative position. The latency between adopting a provocative position and the onset of nystagmus is typically less than 15 seconds, but it can be longer.

If all evidence points to BPPV, but nystagmus cannot be easily elicited, it may be worth a try to maintain the patient in each provocative position for a longer period, for example, 30 seconds, and observe for nystagmus. The latency between movement of the head and onset of nystagmus is presumably due to a combination of the time needed for freely floating particles to move around a semicircular canal and then subsequently into and through the ampulla.

The best single test for BPPV is the Dix-Hallpike test,[18] which was originally described by Dix and Hallpike.[19] In brief, while the patient is seated, the head is turned 30° to 45° in the horizontal plane toward the ear to be tested. The patient next lies down with the neck extended about 30° below the level of the examining surface. The examiner observes for a paroxysmal burst of nystagmus. With the onset of nystagmus, most patients feel anxious, some more than others. If the clinician anticipates such reactions, anticipation of such reactions may facilitate counseling of the patient and talking the patient through particle repositioning maneuvers, in a calm and reassuring manner.

In the most common form of BPPV, where debris is trapped in a posterior semicircular canal, the nystagmus is a combination of upbeat and torsional with the upper poles of the eyes beating toward the ground. The technical method of performance of the Dix-Hallpike test has been described elsewhere in great detail.[20,21]

It is important to note that the Dix-Hallpike test will only be positive if there is debris in one or more of the semicircular canals. If debris is instead primarily within the utricle, the Dix-Hallpike test may be negative. The patient may, however, experience vertigo again within a short timeframe, if the debris is transported from the utricle into a semicircular canal. The moral is that if BPPV is strongly suspected, but the Dix-Hallpike test is negative, it may be advisable to repeat the test on a different day. Selected nystgamus types and associated abnormalities may be found in (**Table 1**).

VERTICAL OCULAR MISALIGNMENT

The search for skew deviation is becoming a routine part of the neurologic examination in some emergency departments.[22] The key point for the nonspecialist is that any new vertical misalignment of the eyes should be considered a potential central sign, for example, due to involvement of the brainstem. To probe for ocular misalignment, one can simply ask the patient whether there is a history of double vision. Ocular misalignment can be revealed on examination through use of the alternate cover test, in which a patient fixates on a target, such as the examiner's nose, while each of the patient's eyes is alternately covered. This procedure is repeated several times, alternating between the right and left eye. The examiner should observe for readjustment of eye position when either eye is uncovered.

ABNORMAL SACCADIC EYE MOVEMENTS

Fixation may be derailed by inappropriate saccades referred to as saccadic intrusions. One common saccadic intrusion is the square wave jerk. The square wave jerk is an

Table 1
Selected abnormal examination findings and the differential diagnosis

Examination Finding	Differential Diagnosis
Dix-Hallpike test shows paroxysmal upbeat torsional (or horizontal or downbeat) nystagmus	BPPV involving the posterior (or horizontal or anterior) semicircular canal, respectively
Spontaneous, unidirectional, predominantly horizontal nystagmus	Acute peripheral vestibulopathy and/or vestibular neuritis and/or labyrinthitis
Gaze evoked, direction switching nystagmus	Posterior fossa lesion
Skew deviation	Posterior fossa lesion
Facial palsy and vesicles in the external auditory canal	Ramsay Hunt syndrome
Asymmetric loss of hearing acuity	Meniere syndrome, vestibular schwannoma

eye movement in which a small, single saccade, in random direction, is followed by a saccade back to the direction of fixation. Square wave jerks are nonspecific. Their presence may signify basal ganglia or cerebellar disease, but they are also seen in older people without evident neurologic disease. Unlike jerk nystagmus, saccades may be distinguished by the absence of well defined, rhythmic fast and slow phases.

VESTIBULO-OCULAR REFLEX TESTS

A focused assessment of the semicircular canal function, at least for the horizontal canal, can be performed at the bedside. The head impulse test was developed to assess the horizontal angular vestibulo-ocular reflex. The technical method of the head impulse test has been described in detail elsewhere.[23] In brief, the patient fixates on the examiner's nose, typically starting with the patient's head turned about 10° to the right or left of midline. The head is then abruptly, passively, and unpredictably turned by the examiner, toward one side or the other, through about 20°, using caution to avoid injury. The examiner watches for catch up saccades back toward the midline. In the case of peripheral vestibulopathy, the eyes move passively with the head during head impulses, resulting in the need for a catch up saccade, away from the weak ear, toward the examiner's nose. An assumption of this test, and a fairly good one, is that in some disorders, such as acute vestibular neuritis, peripheral vestibular involvement is substantially diffuse and would thus be expected to involve the horizontal semicircular canals.

Like the head impulse test, dynamic visual acuity tests assess the vestibulo-ocular reflex. First, the patient's best near vision is determined. Then, the examiner passively oscillates the patient's head to the right and left in the yaw (horizontal) plane at about 2 Hz. If the patient loses more than 2 lines of vision, vestibular weakness should be suspected. It is best to use a near card, because the demands on the vestibulo-ocular reflex are greater with near viewing.[24]

LIMB MOTOR TESTS

The title of this section is deliberately noncommittal and meant to encompass what physicians typically call motor and cerebellar tests. Labeling tests as "motor" (by which many neurologists would mean muscle bulk, tone, and strength) or "cerebellar," as is often done in practice, belies the inherent multisystem and poorly localized nature of the functions being tested.

Moreover, many techniques involving limbs depend on substantial sensory inputs for which any test interpretation must (but usually does not) account. Thus, it is wise to choose tests that are as simple as possible, on both the afferent and efferent sides, in order to limit the amount of information given to the patient and increase the sensitivity of the test.

The finger-to-nose tests familiar to every medical student are flawed. They give the patient abundant sensory information, potentially disguising deficits. More sensitive for detection of peripheral vestibulopathy are past pointing tests. These past pointing tests that require a patient (with eyes closed) to extend both arms and repeatedly touch the examiner's fingers approaching the examiner's fingers either from above or below. Past pointing tests were studied by Róbert Bárány,[25] who described his method as follows[26]:

> *The patient is asked to close his eyes and touch the doctor's finger with…index finger, then…keeping the arm outstretched…lower it to knee-level, then raise it again and touch the doctor's finger once more…The direction of…deviation will*

differ according to the direction of the nystagmus…always in an opposite direction to the nystagmus…

The muscle power examination has obvious relationships to balance and gait. Particularly important is to test both foot dorsiflexor[27] and plantarflexor[28] power. Deficits of either may cause critical gait and balance impairment. Strength can be assessed either with manual muscle power examination, or at times more revealingly, by asking the patient to perform tasks using the patient's own body weight, such as standing on the toes, while the examiner observes the vigor with which this maneuver is completed.

BALANCE AND GAIT TESTS

The Romberg sign is a well-known indicator of visual dependence that implies loss of normal proprioception or loss of peripheral vestibular function.[29] Originally developed for diagnosis of myelopathy, namely tabes dorsalis, the Romberg sign is also encountered in clinical practice when testing patients with bilateral peripheral vestibular hypofunction,[30] and those with vitamin B12 deficiency, copper deficiency, or hyperzincemia.[31]

The Romberg test can be made more difficult and thus more sensitive to balance-related issues by asking the patient to stand in tandem with eyes closed. The examiner should note whether the patient can confidently maintain this posture for several seconds. There are many variations of the Romberg test. In general, these tests have been somewhat unduly criticized, as a result of their less than excellent sensitivity (due in part to vestibular compensation) and specificity (many different types of disorders and aging affect balance).

As is the case with respect to other clinical neurologic tests, any one isolated abnormality must be interpreted with caution. Combined with other tests, though, the Romberg test contributes sensitivity to the neurologic examination. For example, a patient who presents after an acute vertiginous illness, and who falls strongly to the left, has mild left-sided hearing loss and has right beating nystagmus on extreme rightward gaze, might be suspected to have had left-sided labyrinthitis. Any one of these findings alone, in isolation, might be discounted, but together, they form an important pattern.

Similar comments might be made about the Fukuda stepping test. In this test, a patient is asked to march in place with arms outstretched and eyes closed for about 1 minute, and the examiner observes for abnormal turning toward the right or left greater than 20°.[32] If used as a stand-alone test, it is not likely to have high sensitivity or specificity for any particular disorder. However, when combined with other tests described in this review, the Fukuda test increases the likelihood of recognition of a diagnostically useful pattern.

In the examination of gait, one should ask the patient to walk outside the examination room and look for potential signs of central nervous system dysfunction, for example, small step length, irregular step length, and asymmetric arm swing. The "walking Romberg" test gauges postural stability during a 5-m walk with the patient's eyes closed. Most patients with clinically significant cervical myelopathy were reported to manifest either a Romberg sign or a walking Romberg sign.[33]

Recently, there has been increasing interest in dual-task paradigms that simultaneously assess cognition and gait.[34] Balance places phenomenal demands on attention. If a patient has an underlying cognitive disorder, an abnormality of gait, or both, the gait and balance dysfunction can in some cases be unmasked by placing cognitive demands on the patient while simultaneously also asking the patient to perform a balance or gait task. The breakdown of gait function during a cognitively challenging task

may be expressed in terms of the dual task cost (DTC) of walking while performing a cognitively demanding task, for example:

DTC = ([single-task gait velocity – dual-task gait velocity]/single-task gait velocity) × 100.[35]

One of the first reported clinical tests in this category was the "Stops walking when talking" test that was reported to predict fall risk.[36] The clinician can glean helpful information through careful observation of the patient's gait in the hallway during conversation, in comparison to that when the patient is concentrating on walking. One definition of high DTC that might reasonably be used in practice would be a decrease in gait velocity of greater than 20% when performing a cognitive task, such as counting backward from 100 while walking.[37] If a patient's performance declines markedly during conversation or performance of a cognitive task such as counting backward (reflecting a high DTC), then cognitive impairment and a risk for falls[38] should both be suspected.

SUMMARY

Through performance of a neurologic and ear examination, objective data can be gleaned that narrow the differential diagnosis of "dizziness." The multisensory and integrative nature of balance places distinct demands on the diagnostic process. Accordingly, the combination of multiple types of physical examination methods may be expected to increase the sensitivity of one's approach to detection of disorders.

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