

Chapter 5 – The Dizzy Patient: A Clear-Headed Approach

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Introduction

The problem of dizziness is one of the most exasperating in the practice of medicine. Physicians all know that sinking feeling elicited by the patient who sits down and, when you ask, “What can I do for you?” says, “I’m dizzy.” The goal of this discussion is to offer practitioners a reasoned approach to dizziness that will lead expeditiously to diagnosis and effective therapy.

Before getting down to cases, a caveat: “Dizziness” may or may not mean vertigo. More often than not, if a patient proclaims that the problem is vertigo, it’s because he or she has heard the term and thinks it sounds medical. It’s really dizziness that brought this patient to you.

Case 1

A 61-year-old woman comes to the office complaining of dizziness. When asked to describe the sensation, she says that it is a feeling of violent motion, a sensation of being pulled to the right. It occurs in waves a moment after she lies down on her right side in bed. If she remains motionless, the sensation will pass in about 30 seconds. However, if she then sits up, the phenomenon recurs, although less severely, this time with the environment moving from left to right and a sensation of falling to the left. There is no history of hearing loss or tinnitus, nor is there an associated diplopia, dysarthria, or weakness. Many years before, she was thrown from a horse and struck her head, with resultant severe vertigo, which cleared gradually over a six-week period.

On examination, vital signs including orthostatic blood pressure and pulse determinations are normal. General examination and routine neurologic examination are normal. The findings on examination of cranial nerve VIII include mildly abnormal hearing in the right ear, equal air and bone conduction, and intact speech discrimination. There is no spontaneous nystagmus. However, with Bárány position testing (see **Figure 5-2**), rotatory nystagmus develops five seconds after the patient attains the right-ear-down position, with fast phase in the counter-clockwise direction and slow phase in the clockwise direction. The patient reports vertigo, with the environment spinning right to left, which she says is the same as her symptomatology at home. The nystagmus and vertigo stop after 30 seconds, but when she sits up, there are a few beats of nystagmus in the opposite direction with recurrence of vertigo but in the reverse direction. Head-hanging and left-ear-down positions fail to elicit vertigo or nystagmus. The tympanic membranes are intact, but when air is insufflated via the otoscope

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into the right external auditory meatus, the patient complains of vertigo, and a few beats of nystagmus develop.

Differential Diagnosis

The first principle to observe in evaluating a dizzy patient is not to put any words in the patient's mouth. This is, of course, a good rule in taking any medical history, but it is particularly applicable in this instance. When the patient says to you, "I am dizzy," sit back in your chair, slowly spin around, perhaps stare aimlessly out the window, and reply, "What do you mean, dizzy?" Then wait for the response. This may take what seems to be a long time; nonetheless, don't ask, "Does the room spin?" "Do your legs get weak?" "Do you feel as if you might stagger?" "Are you lightheaded?" because the answer to all these questions will nearly always be yes. If you're lucky enough to be the first physician to examine a patient complaining of dizziness, always take the undirected approach. Merely say to the patient, "What do you mean, dizzy?" and wait for the response. There are several responses that the patient may give (see **Figure 5-1**).

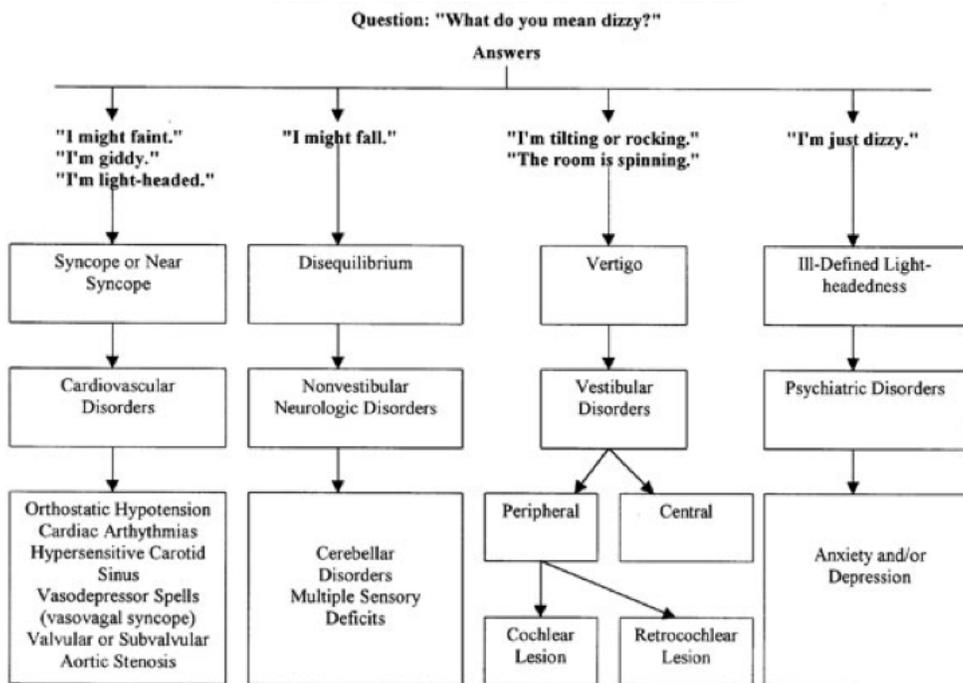


Figure 5-1: Differential Diagnosis of Dizziness

Syncope Or Near-Syncope

"I feel as if I might faint," or "I feel giddy or light-headed." Some patients do faint or report that they have done so; others have never actually fainted (near-syncope). Pathophysiologically, both syndromes suggest several cardiovascular disorders that produce a generalized decrease in cerebral blood flow; there is no qualitative difference between syncope and near-syncope with respect to the differential diagnosis.

Circulatory syndromes that should be considered in the differential include *orthostatic hypotension*, which may have a number of causes, most of

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them iatrogenic (e.g., antihypertensive agents and/or vasodilators). *Cardiac arrhythmias* are a very frequent cause of syncope and near-syncope. If the history suggests arrhythmic episodes, Holter monitoring may be required. *Hypersensitive carotid sinus* is relatively uncommon. *Vasovagal attacks* are otherwise known as the simple faint or the simple swoon. Neurocardiogenic syncope is probably due to over activity of the baroreceptor reflex such that brief periods of hypertension result in disproportionate bradycardia and hypotension resulting in decreased cerebral blood flow and consequent loss of consciousness. Any faintness that occurs during or immediately after exercise suggests a possible serious cardiac disorder, such as aortic stenosis, anginal equivalents, or asymmetric septal hypertrophy.

Disequilibrium

"I feel as if I might fall." This version of dizziness generally reflects one of two major categories of neurologic disease, apart from disorders of the vestibular system.

Cerebellar ataxia is due either to a primary disease of the cerebellum, e.g., cerebellum degeneration, or to a tumor in or near the cerebellum, e.g., in the cerebellopontine angle. Neurologic examination will ordinarily reveal such pathology.

The multiple sensory deficits syndrome reflects multiple abnormalities in the various sensory proprioceptive systems. When several of these systems fail in a given individual, the central nervous system receives conflicting proprioceptive input, with consequent dizziness. The typical patient is rather elderly, perhaps with some *visual disorder* due to cataracts, some *auditory disorder* due to presbycusis, and *peripheral neuropathy* due to diabetes and/or chronic use of alcohol. Such a patient typically complains of dizziness at night, for instance, when the lights are out or dim and he or she has to go to the bathroom. On occasion, the patient may fall.

The treatment of this extremely common syndrome is common sense: As many of the sensory abnormalities as can be corrected, should be. Cataracts and hearing disorders can be treated, and the progression of peripheral neuropathy can be prevented by abstinence from alcohol. You might also advise your patient to keep the lights on at night, which would help the visual system compensate for other sensory abnormalities. Such patients should not be treated with drugs that might sedate them, as antivertigo medications would do. Mistaking this syndrome for vertigo would, in fact, make matters worse.

Anxiety And/Or Depression

There are patients who when asked, "What do you mean, dizzy?" respond, usually after a pause, "Dizzy." If the physician persists with "Do you mean you might faint?" or "Do you mean that you might fall?" or "Do you mean that the room spins?" the patient repeats, "No, I mean I'm dizzy." This disorder can only be called true dizziness, and it generally arises from various psychological disorders, most commonly anxiety (with or without hyperventilation) and/or depression.

Affective disorders can often be recognized because of the effect that the patient has on the examiner. If you feel depressed or anxious yourself after spending time with a patient, it may well be because the patient is depressed

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or anxious. It is extremely important to recognize instances when dizziness represents a metaphor for depression, because treatment for vertigo is likely to exacerbate depression, whereas treatment for depression might dramatically relieve the dizziness.

Vertigo

The fourth and last category of disorder found in patients who complain of dizziness is true vertigo, or illusion of motion. Some patients insist that they themselves are moving, others—such as the one presented at the beginning of this chapter—that the environment is moving. In either case the patient says, “I feel as if I am tilting, rocking, or moving in some other way,” or “I feel as if the room is spinning.”

Vertigo indicates a disturbance in the vestibular system, which is responsible for keeping the central nervous system informed of the head’s position in space, its relation to the pull of gravity, and its acceleration in various planes. The question is whether the vertigo is due to a disorder in the *peripheral nervous system* (the end organ or the peripheral nerve) or in the *central nervous system* (the brainstem or its projections to parts of the cerebral cortex, particularly the temporal lobe). Each lesion has its own differential diagnosis and treatment.

Evaluation of Vertigo

The first step is to do a complete history and physical examination and a general neurologic examination with particular attention to the VIII cranial nerve. The VIII cranial nerve is in fact two separate cranial nerves, the vestibular and cochlear. (Unfortunately, it received only one number, and generations of medical students have been confused ever since.) These two nerves have closely juxtaposed end organs, run close to each other in the internal auditory meatus, and have two completely different pathways in the central nervous system. Because of the close proximity of these two nerves and their end organs, it is common for disease of one to affect the other. *The physician should therefore examine both aspects of cranial nerve VIII whenever there is a complaint of vertigo.*

Cochlear VIII Nerve Function

Pure Tone Hearing Loss

Auditory testing.

Examination of the cochlear system involves three steps whether or not the patient complains of hearing loss. The first is to test for pure tone hearing loss. This can be done quite reliably in the office by comparing the sensitivity of the patient’s ears or comparing the patient’s ears with your own, using a ticking watch or the sound of your fingers rubbing together.

Sensory Neural vs. Conductive Hearing Loss

If there is a pure tone hearing loss, the next step is to determine whether it is a sensory neural hearing loss, i.e., a neurologic problem, or a conductive hearing loss, i.e., a disorder in the middle ear interfering with the functions of the ossicles. These determinations are made by using two tests, the Weber and Rinne.

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1. The Weber test is performed by placing a vibrating tuning fork at the midline of the skull and asking the patient on which side the sound can be heard. If there is a definite lateralization to one side, you can determine whether there is sensory neural or conductive hearing loss. For example, if the Weber lateralizes to the left, this may be interpreted as either a left-sided conductive hearing loss or right-sided sensory neural hearing loss. Combining this information with the knowledge of which ear has the hearing loss, you can determine whether that loss is sensory neural on the right or conductive on the left.

However, as the reader probably knows, the Weber test is often difficult to use effectively in actual office circumstances. Frequently, when asked where the sound is heard, the patient points directly at the tuning fork and cannot appreciate a definite lateralization.

2. The Rinne test is of greater use in an office setting. Bone and air conduction are compared by placing the tuning fork: first over the mastoid bone, and then in front of the ear. Under normal circumstances, air conduction is better, because the ossicles in the middle ear amplify and intensify the sound as it passes through the middle ear to the inner ear. If the ossicles are not functioning because of otosclerosis, cholesteatoma, or perhaps fluid in the middle ear, air conduction may suffer, which leads to a situation in which air and bone conduction are equal or bone conduction is the better of the two. If, however, there is sensory neural hearing loss, air conduction remains better than bone conduction.

Unfortunately, generations of physicians have been taught to do the Rinne test by placing the vibrating tuning fork on the mastoid process and saying to the patient, "Tell me when that stops." Time passes, and passes, other patients line up in the waiting room, and this patient is still just sitting there quietly. Finally the physician asks, "Has it stopped yet?" and the patient replies, "Oh, yes, it stopped a long time ago." The frustrated physician repeats the test, this time interrupting every second or two to ask whether the patient still hears the sound. The talking-physician approach does tend to interfere with a hearing test—by drowning it out. It is easier simply to put the vibrating tuning fork over the mastoid process (number 1) and then in front of the ear (number 2) and ask the patient which is louder. If the patient says number 2, you know that air is better than bone conduction. End of test. This finding, along with hearing loss, tells you unequivocally that there is a sensory neural problem.

Cochlear vs. Retrocochlear Hearing Loss

The third step in the hearing examination, needed only if there is a sensory neural loss, is perhaps the most important of the differential procedures but paradoxically the one least well known to many physicians. The question being asked is whether the sensory neural deficit is due to end organ disease (cochlear) or to peripheral or central neural disease (retrocochlear).

Speech discrimination testing can be done in your office to differentiate a cochlear from retrocochlear sensory neural hearing loss. (There are a number of

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ways to make this distinction, but many require the services of an audiologist.) The physician whispers words in the affected ear (e.g., hot dog, ice cream) loud enough for the patient to hear. At the same time, you make a sound in the other ear so that the patient cannot hear your words through that ear. Putting a finger in the patient's other ear and moving it around will serve the purpose. Do this on both sides five or 10 times, have the patient repeat your words each time, and compare the two ears.

In people with cochlear-type sensory neural hearing loss, such as occurs in Ménière's disease, speech discrimination is not perfect, but it is *relatively* preserved. On the other hand, in patients with retrocochlear hearing loss, such as accompanies a vestibular schwannoma, there is a disproportionate loss of speech discrimination. Thus a patient with a cochlear hearing loss should be able to understand 70 percent or more of the words heard, whereas a patient with a retrocochlear hearing loss might understand only two out of 10 words. *If there is any question of a retrocochlear hearing loss, one should order an audiogram.*

Vestibular VIII Nerve-Function

Testing for nystagmus.

The vestibular aspect of the VIII cranial nerve may be examined by testing for nystagmus. First, ask the patient to sit on the end of the examining table and to look about 45° to the right and to the left. (Asking the patient to look beyond 45° is not useful, since when asked to look too far in either direction, about 10 percent of the normal population show some degree of gaze-evoked end-point nystagmus.) If nystagmus develops when the gaze is directed to 45°, note the direction of the fast phase, the direction of the slow phase, and in what position of the eyes they occur.



Figure 5-2: To perform Nysten-Bárány maneuver, have patient lie back on examining table so that head hangs over edge at 30° below horizontal. Do not move patient into this position too quickly. Then have patient look straight ahead, and watch for 30 seconds. If nystagmus and vertigo develop, note directions of fast and slow phases and ask patient to describe sensations: In which direction does world seem to spin and toward which direction is the feeling of falling or being pulled? If vertigo lasts longer than 60 seconds in this position, it is persistent positional vertigo; if not, it is transient positional vertigo. Repeat position three or four times to see whether vertigo and nystagmus extinguish; also note whether they return when patient sits up and in which directions they occur. If head-hanging posture fails to elicit vertigo, repeat maneuver with right ear down; if this fails, repeat with left ear down.

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Next the patient should be put through a series of positions called the Dix-Hallpike maneuver (see **Figure 5-2**). All vertigo is positional to some extent, but if vertigo is positional only, there are specific pathogenetic and prognostic implications. Once position testing has been done, the physician knows in which direction the world seems to be spinning and in which direction the patient seems to be falling when the vertigo develops. The directions of the fast and slow phases of the nystagmus have been recorded. The question now is how to interpret these data.

Peripheral or Central Nervous System?

A brief review of the neuroanatomy and neurophysiology of the vestibular system may be helpful at this point. The text that follows should be considered as a unit with the illustrations above, which depict the normal structural components (**Figures 5-3A** and **5-3B**) and neurologic events pertinent to vertigo and nystagmus.

Vestibulo-Ocular Reflex

The end organ of the vestibular nerve is located in the semicircular ducts, utricle, and saccule. The lateral, or horizontal, semicircular duct is oriented in the inner ear so that it tilts at about 30° above the horizontal plane (see **Figure 5-3A**). When the head is held in the usual carrying position, this duct is approximately parallel to the ground. Thus turning the head right and left would be expressed almost entirely in a vector within the plane of the lateral semicircular duct.

The series of events that make up the active phase of the vestibulo-ocular reflex is schematized in **Figure 5-3D**. When the head turns to the left, a series of impulses is initiated (beginning with stimulation of the hair cells in the left lateral semicircular duct) that leads to contraction of the right lateral rectus muscle (right eye abductor). This sequence, taken no further, would of course lead to a situation in which the eyes are pointed in two different directions, which produces diplopia, an unacceptable situation for the nervous system. Therefore, a corresponding series of impulses must also reach the left medial rectus muscle in order for the left eye adductor to contract as well.

In a comatose patient with an intact brainstem but with cortical signals in abeyance, the vestibulo-ocular reflex can be elicited by turning the patient's head, which produces the *oculocephalic reflex*, or the so-called doll's eyes. In an awake patient the reflex may be demonstrated by having the patient fix his or her gaze on a distant object or by infusing the ears with warm or cold water (the *caloric reflex*). Although the caloric reflex should be a routine part of the evaluation of a comatose patient, in an awake patient it is a procedure perhaps best left to the otologist or neurologist.

Cerebral Cortex

In the hypothetical situation illustrated in **Figure 5-3D**, the eyes have deviated to the right. This information is transmitted to the cerebral cortex by more than one mechanism. The movement of images on the retina sends information to the occipital cortex through the usual visual pathways. However, it is presumed that information regarding the movement of the eyes may reach the cerebral cortex even in the absence of visual stimuli, since proprioceptive organs in the orbit probably convey information to the parietal cortex.

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The cerebral cortex, however, finds itself in a dilemma. It asks itself, “Have I, in fact, turned the eyes to the right?” The left frontal eye fields could, of course, turn the eyes under normal circumstances to produce a voluntary saccade (rapid conjugate eye movement) to the right. However, in this instance the left frontal eye fields have not fired. It is possible that the right parietal-occipital region could have turned the eyes to the right by producing a conjugate pursuit or tracking eye movement. But in this case, these areas have not fired either. Thus the cerebral cortex has received conflicting information.

On the one hand, it seems that the eyes have turned to the right. On the other hand, it seems as if the eyes have not *been moved* to the right. What conclusion can the cerebral cortex draw? It concludes not that the eyes have moved to the right but that the world has moved to the left! This arrogant conclusion is based on the cerebral cortex’s assumption that it alone is capable of moving the eyes, although of course the brainstem can also move them.

Frontal Lobe

Parts of the cerebral cortex, however, are not so egocentric. The frontal lobe, for example, knowing that the eyes have turned to the right, decides to make a correction. The corrective phase of the vestibulo-ocular reflex arises from the frontal eye fields and results in rapid turning of the eyes back to the left.

In the circumstance postulated, the stimulus has arisen from the left vestibular system and caused a slow conjugate eye movement to the right followed by intermittent rapid conjugate correction back to the left. It is associated with a vertigo in which the patient has a feeling that the world is spinning to the left while he or she is being pulled to the right.

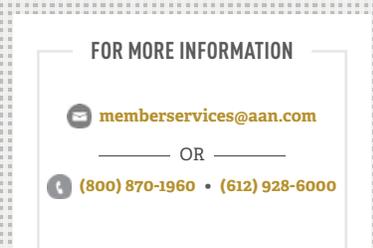
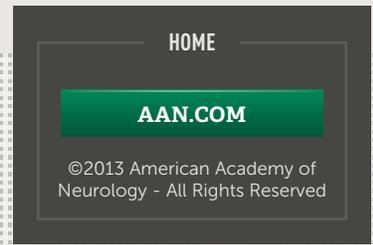
The patient’s feeling of being pulled may become worse when the eyes are closed, because closing the eyes removes another proprioceptive system that would help to compensate. Romberg’s sign, i.e., when a person’s balance becomes worse with the eyes closed, can be seen in any abnormality producing a proprioceptive disorder, including peripheral neuropathy and disease of the spinal cord as well as disease of the vestibular system.

The Two Phases of Nystagmus

Thus vestibular imbalance nystagmus consists of two components. *The first (active) phase* originates in the brainstem or vestibular system, is caused by different vestibular input from the ears, and is associated with slow eye movement. *The second (corrective) phase* is initiated by the cerebral cortex via the frontal eye field and is associated with fast eye movement. Both phases act through the final common pathway of the ocular motor system of the brainstem.

Under normal circumstances (see **Figure 5-3B**), the entire vestibular system functions bilaterally with all of its central connections. There is no vertigo or nystagmus with ordinary accelerations of the head. In fact, the situation depicted in **Figure 5-3D**, although useful as a model for understanding the mechanics of vertigo and nystagmus, hardly ever happens in real life. It is relatively rare for the pathology to produce an excess of stimuli from the affected side.

More likely is the pathologic situation illustrated in **Figure 5-3C**, which depicts a “lesion” in the right ear, perhaps functional, perhaps anatomic. In this situation, an imbalance develops between the two sets of vestibular apparatus in the ears.



With disruption of the vestibular impulses from the right ear, it is as if the left side has been stimulated or the head has been turned with acceleration to the left. What symptomatology does such a lesion produce? The eyes are driven conjugately *toward* the side of the lesion. This movement is interrupted by intermittent rapid corrective movement *away from* the side of the lesion. The patient has a sensation of vertigo, with the world spinning *away from* the lesion (or toward the fast phase) and a feeling of falling *toward* the side of the lesion (or toward the slow phase).

Criteria For Locating The Lesion

There are four criteria for a peripheral type of vertigo and nystagmus (see **Table 5-1**). If there are: 1) fast-phase nystagmus away from the lesion, 2) slow-phase nystagmus toward the lesion, 3) environment spinning away from the lesion, and 4) Romberg's sign toward the lesion, one can say with confidence that there is a lesion of the peripheral nervous system, probably in either the end organ or the peripheral nerve.

Table 5-1: Criteria for peripheral lesion of vestibular system

Rapid-phase nystagmus	away from lesion
Slow-phase nystagmus	toward lesion
Environment	spinning away from lesion
Romberg's sign	toward lesion

If any of these four rules fails to hold, one can assume by exclusion that the lesion is in the central nervous system. Central nervous system lesions can cause bilateral nystagmus in the same position of the head, vertical nystagmus of any kind, and any conditions in which the directions of the fast and slow phases, the Romberg's sign, and the spinning of the environment do not strictly fit the four criteria specified. Those criteria specify only the anatomic localization without implying anything about the severity or seriousness of the underlying disease. Peripheral diseases can be trivial (e.g., vestibular neuronitis) or very serious (e.g., acoustic schwannoma). Central diseases can range from the trivial complications of many drugs to vertebrobasilar insufficiency.

Synthesizing the Data

Thus by testing the auditory system and the vestibular system, one can divide all cases of vertigo into three categories:

1. Peripheral (by vestibular criteria) cochlear disease (by auditory criteria and signs)
2. Peripheral (by vestibular criteria) retrocochlear disease (by auditory criteria), and
3. Central disease

With this in mind, we can now consider the major diseases in each category.

Peripheral Cochlear Lesions

Labyrinthitis is thought to be a result of viral infection of the endolymph and perilymph affecting both the vestibular and cochlear components of the system. The usual history is viral illness followed by acute onset of severe spinning vertigo and sensory neural deafness with tinnitus. Examination shows a classic peripheral picture by vestibular criteria and a classic cochlear picture by auditory

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criteria. Paracentesis of the perilymph may show growth of common ubiquitous viruses, such as coxsackievirus or echovirus. Despite its severe onset, labyrinthitis is a benign illness, which resolves completely in three to six weeks. Patients regain normal hearing and vestibular function.

Vestibular neuritis, or acute vestibulopathy, is thought to be pathogenetically identical to labyrinthitis but without any hearing symptomatology. If the patient has vertigo unaccompanied by a hearing abnormality, it is strictly speaking impossible to be sure whether the disease is cochlear or retrocochlear. However, its natural history is also benign, and it clears up completely in three to six weeks, which makes a retrocochlear illness very unlikely.

Ménière disease is caused by a cryptogenic hydrops of the endolymph such that there is intermittent swelling of the semicircular ducts, with damage to the hair cells. An attack of Ménière's syndrome is classically characterized by a dull ache in the region of the mastoid process or around the ear associated with severe tinnitus, a cochlear kind of sensory neural hearing loss, and a classic peripheral type of vestibular syndrome with severe spinning vertigo. It is identical in almost every respect with an acute attack of labyrinthitis. However, it does not clear up completely in three to six weeks, and patients are left with residual hearing loss. Several months or years later a similar attack may occur, leaving the patient with even more severe hearing loss. Tinnitus, a nonspecific sign of auditory system disorder, is a major problem for these patients, who can be terribly disabled for weeks at a time by the vertigo that accompanies acute attacks.

Many therapies have been tried, including shunting of the perilymphatic system and diuretics, but none has proved effective. About 15 percent of these patients will have bilateral disease in subsequent years. Management of such cases is complex and often best entrusted to an otolaryngologist or otoneurologist.

Benign positional vertigo, or Bárány's vertigo, usually occurs in older patients and is characterized by the sudden onset of a peripheral vestibular syndrome with no auditory aspect. It is present only in certain positions, which are specific to the individual.

Typically, the patient reports that a few moments after attaining a certain position, perhaps in bed at night, a severe vertigo occurs in which the world spins in one direction while the patient has a sensation of falling in the other direction. If he or she does not move, the vertigo stops, which implies that it is transient in type. If the patient sits up, the vertigo recurs, but this time in reverse. If the patient repeats the posture several times, the tendency toward vertigo and nystagmus will fade. All the symptoms can be reproduced using the Dix-Hallpike maneuver. Benign positional vertigo has a natural course, which improves gradually over a six-month period and ends with complete recovery.

Etiology of Benign Vertigo

Cupulolithiasis

Two major theories have been proposed to explain benign vertigo. The theory of canalolithiasis maintains that bits of calcium break off from the otolithic apparatus in the ear, perhaps as a consequence of aging or minor head trauma. If these bits of calcium are floating in the endolymph, they can, in certain positions, put pressure on the end organ, which initiates an impulse arising from that ear. Since the calcium tends to fall into the most dependent of the

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three semicircular ducts, the canalolithiasis tends to affect the posterior vertical semicircular duct resulting in rotatory nystagmus in the dependent ear and vertical nystagmus in the other ear maximum when the affected ear is down.

Perilymphatic fistula

The second theory for benign positional vertigo is the development of a perilymphatic fistula. Normally the middle ear and inner ear are separated by the oval and round windows, which are completely sealed. If for some reason (e.g., head trauma) a crack develops in the oval or round window, some of the perilymph may leak from the inner ear into the middle ear. Such patients may have intermittent episodes of conductive hearing loss superimposed on a sensory neural hearing loss. This pathology is established by an audiogram.

The presence of the fistula can be detected by placing the otoscope in the ear and closing the glass window, which produces an air-tight space. Air is then pumped into the external ear using the ordinary balloon attachment to the otoscope. This air distorts the tympanic membrane, which briefly increases the pressure in the middle ear. Under normal circumstances, a mild sensation in the ear is produced but no vertigo. If, however, there is a pathologic connection between the middle ear and the inner ear, increased pressure in the middle ear will be transmitted to the perilymphatic space in the inner ear, which produces an abnormal stimulus and causes vertigo and nystagmus.

Case Diagnosis and Treatment

The 61-year-old woman described at the beginning of this discussion was found to have perilymphatic fistula. This diagnosis—established by the findings of benign positional vertigo, a syndrome of conductive and sensory neural hearing loss, and a positive fistula test—may be less rare than was once thought. It is important to recognize it, because it is completely treatable surgically. Microsurgery, performed in this patient, repaired the defect in the oval window by filling it with fat from the earlobe, with complete relief of her vertigo.

Peripheral Retrocochlear Syndromes

Vestibular Schwannoma

A second category of disease is a peripheral type of vertigo but with retrocochlear hearing loss, i.e., patients are found to have poor speech discrimination. Such patients should always have an audiogram; if the audiogram confirms retrocochlear hearing loss, a CT scan with special views of the internal auditory meatus is indicated. If a CT scanner is not available, conventional tomography of the internal auditory meatus should be done.

It is important to recognize the presence of a tumor while it is still contained within the internal auditory meatus and thus surgically resectable. Vestibular schwannomas (often incorrectly called acoustic neuromas) are histologically benign tumors, but they can become quite dangerous. If a vestibular schwannoma is allowed to grow into the brainstem, treatment requires a posterior fossa craniotomy, with significant morbidity and even some mortality. *Any patient with a history of progressive hearing loss should at some time during the evaluation have a careful audiogram, and if any retrocochlear characteristics are found, a CT scan with careful views of the internal auditory meatus should be ordered.*

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Central Lesions

The last category of vertigo is central disease, i.e., patients with vestibular symptomatology that does not meet the criteria for peripheral disease. This group includes patients with vertical nystagmus or bilateral nystagmus in the same position of the head.

Drugs

All drugs that act by intoxicating the reticular activating system in the core of the brainstem—including all anticonvulsants, all sedatives, and some sleeping pills—will by their nature produce nystagmus in two different directions in the same position of the head. When the patient looks to the right, the nystagmus beats to the right; when the patient looks to the left, it beats to the left. Overdosage can produce vertigo.

The fact that the lesion is central does not necessarily mean that it is serious. In fact, the appearance of this form of nystagmus may prove that a given drug is in the therapeutic range. Such patients should be asked specifically about their use of drugs, including alcohol; before any invasive studies are performed, it is useful to order blood and urine toxic screening.

Demyelinating Illness

Demyelinating illnesses, such as multiple sclerosis, can and often do produce vertigo, presumably because there are lesions somewhere in the vestibular system in the brainstem. Although such vertigo usually has characteristics that indicate a central lesion, occasionally it can resemble peripheral vertigo and be misdiagnosed as vestibular neuronitis. If the same patient returns a year later with optic neuritis, it would be clear in retrospect that the first disorder was due to multiple sclerosis. However, nothing has been lost in the interim, because multiple sclerosis of this mild degree would not be treated.

Vascular Disease Affecting the Brainstem

In approaching vascular disease affecting the brainstem, it should be remembered that the most common manifestation of vertebrobasilar insufficiency is vertigo, but vertigo is almost never the only manifestation. Such patients can also be expected to complain of double vision, weakness of the limbs, sensory loss, dysarthria, and dysphagia. It might be possible for disease of the small branch of the vertebral artery to produce vertigo as its only symptom, but in such instances there is no specific therapy anyway.

Disorders of The Temporal Lobe

Temporal lobe seizures arising from trauma, tumors, or prior strokes can, as one of their manifestations, produce vertigo.

Treatment of Vertigo

Anticholinergic- and Antihistamine-Type

There are three categories of drugs for treating true vertigo. Anticholinergic- and antihistamine-type drugs include dimenhydrinate, diphenhydramine, meclizine, and cyclizine. All of these drugs are effective if the dosage is adequate—about 50 mg every six hours (see **Table 5-2**). They produce major sedation as their side effect, but this is usually of no concern: Patients who have been dizzy and

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vomiting for hours tend to be more than happy to go to sleep.

Table 5-2. Drugs useful in symptomatic treatment of vertigo

	Duration of Activity	Useful Adult Oral Dosage	Sedative Effects	Other Modes of Administration
Ethanolamines				
Dimenhydrinate	4–6 hr	50 mg ev. 6 hr	++	Rectal IM, IV, IM, IV
Diphenhydramine	4–6 hr	50 mg ev. 6 hr	++	
Piperazines				
Meclizine	12–24 hr	25–50 mg ev. 6 hr 50 mg ev. 6 hr	+	Rectal, IM
Cyclizine	4–6 hr		+	
Phenothiazine				
Promethazine	4–6 hr	25 mg ev. 6 hr	++	Rectal, IM, IV
Belladonna alkaloid				
Scopolamine	4 hr	0-6 mg ev. 6 hr	+	SC, IV

+Mild ++Moderate

The Phenothiazines: Promethazine

Promethazine is the only phenothiazine that works against the nausea associated with vestibular imbalance and vertigo. Other phenothiazines, useful for chemical nausea, are of no help whatsoever in this setting. Promethazine may be effective primarily because it is an anticholinergic, not because it is a phenothiazine; it is useful also because it can be given together with the antihistamines or the antiserotonin drugs. A combination of promethazine and antihistamine is particularly effective for acute vertigo.

Belladonna Alkaloids

A belladonna alkaloid, usually scopolamine, is used only for severe recurrent vertigo (e.g., in difficult cases of Ménière's disease) because it is a dangerous drug with many cardiovascular and psychiatric side effects. Transdermally absorbed scopolamine, although helpful for motion sickness, is of inadequate dosage for use in treating an acute vestibular syndrome. Benzodiazepines are often useful in treating the anxiety components of dizziness, but are not particularly useful for true vertigo.

Summary

To evaluate dizziness, you must first decide whether it can be categorized as near-syncope, disequilibrium, ill-defined light-headedness, or vertigo. If it is vertigo, vestibular and auditory testing will allow you to place the patient in one of three categories: peripheral cochlear disease, peripheral retrocochlear disease, or central disease. When this distinction is made, you can create a reasonable differential diagnosis and arrive at the likely diagnosis. Some of these disorders (e.g., vestibular schwannoma) require specific evaluation and treatment, whereas others have a benign natural history and require only symptomatic relief for the duration. Symptomatic therapy is straightforward and makes use of the three categories of drugs discussed.

This approach should allow you to diagnose and treat your dizzy patients quickly and effectively.

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Self-Assessment Questions

1. Endolymph
 - A. is the intracellular fluid
 - B. is continuous with the subarachnoid space
 - C. surrounds the utricle and saccule
 - D. passes through the perilymphatic duct
2. The following are all true statements about the vestibular system EXCEPT:
 - A. the semicircular ducts sense angular acceleration
 - B. the cells of origin of the vestibular hair cells are bipolar neurons in Scarpa's ganglion
 - C. the utricle and saccule sense linear acceleration
 - D. the vestibular nerve travels in the internal auditory meatus
 - E. impulses conveying vestibular sensation travel in the vestibular portion of the cranial nerve VIII
3. The vestibular nuclei connect the ocular-motor system via the:
 - A. posterior columns
 - B. Clark's column
 - C. the medial lemniscus
 - D. the medial longitudinal fasciculus
 - E. the lateral lemniscus

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4. The vestibulo-ocular reflexes
 - A. utilize the frontal eye fields as a command center
 - B. bypass the pontine paramedial reticular formation
 - C. utilize the lateral lemniscus to transmit impulses to the oculomotor nuclei
 - D. may be utilized to distinguish a nuclear from an infranuclear gaze palsy
 - E. may be utilized to distinguish a supranuclear from a nuclear or infranuclear gaze palsy

Answers:

1. A
2. B
3. D
4. E



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