**Paparella: Volume II: Otology and Neuro-Otology**

**Section 1: Diagnosis of Disorders of the Ear**

# Chapter 4: Vestibular Function Tests

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Vestibular function tests are neurodiagnostic procedures used by otolaryngologists to evaluate patients who suffer from dizziness or unsteadiness. The following tests are in general clinical use:

1. *Electronystagmography,* in which the patient's eye movements are monitored as he or she undergoes a series of procedures designed to detect inappropriate eye movements and to determine whether the vestibular system generates appropriate eye movements when stimulated.

2. *The rotation test,* in which the patient's eye movements are monitored as he or she is oscillated slowly back and forth.

3. *Posturography,* in which the patient's body sway is monitored as he or she tries to maintain balance while posture control cues are systematically varied.

4. *The Hallpike maneuver,* in which the patient's eye movements are monitored after his or her head is quickly inverted in the plane of the posterior semicircular canals.

5. *The pressure test,* in which either the patient's eye movements or body sway is monitored as the external ear canal is subjected to pressure changes.

These five tests fall into two categories - *nonspecific* and *specific.* The three nonspecific tests are electronystagmography, the rotation test, and posturography. These tests often yield evidence of vestibular lesions, particularly lesions involving the labyrinth or vestibular nerve, and in some patients they provide the only evidence of such lesions. Sometimes they also specify the location of the lesion, discriminating labyrinthine or vestibular nerve lesions from central nervous system lesions and identifying the lesioned ear. The reason why these tests are nonspecific is that they generally do not identify specific etiologies. To be sure, there are strong correlations, both positive and negative, between certain test results and specific diseases. Nevertheless, an etiologic diagnosis depends on the physician's evaluation, not only of vestibular function test findings, but also of the patient's history, physical examination, and other laboratory tests.

The two specific tests are the Hallpike maneuver and the pressure test. These tests yield abnormalities that strongly suggest labyrinthine disorders. An abnormality on the Hallpike maneuver suggests benign positional vertigo; an abnormality on the pressure test suggests a perilymph fistula.

## Nonspecific Vestibular Function Tests

### Electronystagmography

Electronystagmography, as defined previously, is an examination of eye movements, with emphasis on vestibular nystagmus. The physician could detect vestibular nystagmus merely by looking at the patient's eyes if it were not for the fact that the patient's eyes must be open and permitted to fixate. Visual fixation strongly suppresses vestibular nystagmus; therefore, it is necessary to monitor eye movements electronically while denying fixation either by having the patient close the eyes or by placing him or her in total darkness.

Electronystagmography depends on the fact that there are steady direct-current potentials, called *corneoretinal potentials,* between the corneas and retinas of the eyeballs. These potentials create an electrical field in the front of the head that rotates as the eyes rotate in their orbits. Rotation of the electrical field produces a systematic voltage change between electrodes attached to the skin on either side of the eyes.

The standard placement of electrodes is shown. Horizontal eye position is monitored by a pair of electrodes placed on the patient's temples. Some examiners also monitor vertical eye position from a second pair of electrodes placed above and below one of the patient's eyes. A fifth electrode placed on the forehead serves as a ground. When placing the electrodes in this manner, the examiner assumes that the patient's eye movements are conjugate, since the horizontal electrodes record the average horizontal position of both eyes together and the vertical electrodes record the vertical position of only one eye. To verify this assumption, most examiners perform a brief evaluation of oculomotor function before testing. If disconjugate horizontal eye movements are seen, the examiner may elect to arrange the electrodes to monitor the horizontal movements of the two eyes separately and omit vertical eye movement recording.

The voltages picked up by the electrodes are fed to a *nystagmograph,* a special-purpose device that filters and amplifies the eye position signals. The output of the nystagmograph is fed either to a strip chart recorder, which writes the signals on a moving strip of chart paper, or to a computer, which displays them on a monitor and stores them for later analysis.

Electronystagmography usually consists of six tests:

1. Saccade test.
2. Tracking test.
3. Optokinetic test.
4. Gaze test.
5. Positional test.
6. Caloric test.

The first three tests (saccade, tracking, and optokinetic tests) are not tests of vestibular function. The saccade tests evaluates the saccade eye movement control system, and the tracking

and optokinetic tests together evaluate the pursuit eye movement control system. These eye movement control systems are of secondary interest to those who evaluate patients with dizziness or unsteadiness, but they are routinely evaluated because abnormalities are occasionally detected in such patients. Saccade defects are rare, but pursuit defects are sometimes found, because pursuit pathways are anatomically intermingled with central vestibular pathways.

The last three tests (gaze, positional, and caloric tests) are tests of vestibular function. The gaze and positional tests are designed to detect inappropriate vestibular nystagmus induced by various eye and head positions. The caloric test is designed to determine whether an appropriate nystagmus response is generated by thermal stimulation of the labyrinths.

## Saccade Test

The saccade test serves a dual purpose. First, it calibrates the nystagmograph. The examiner asks the patient to look between two visual targets separated by a known visual angle (usually 20 degrees) and adjusts the gain of the nystagmograph to yield a given signal deflection. This procedure is performed separately for the horizontal recording channel as the patient looks right and left and for the vertical channel as the patient looks up and down.

Second, it evaluates the saccade eye movement system. When the patient looks back and forth, he or she performs saccades and affords the examiner an opportunity to ascertain whether they are defective. A strip chart recording of horizontal saccadic eye movements of a normal individual is shown. The saccades are quick and precise, stopping exactly on the target each time.

Strip chart recordings are useful in screening for severe saccade defects, but careful evaluation of saccades requires the use of a computer. The results of a computerized saccadic test of a normal individual are shown. This individual has been asked to watch a computer-controlled visual target that jumps back and forth in a random sequence. A segment of the eye movement tracing, with stimulus position superimposed, is shown. The complete stimulus sequence consists of 80 target jumps (40 to the right and 40 to the left) with amplitudes ranging from 5 to 25 degrees. After testing, the computer calculates three values - peak velocity, accuracy, and latency - for each saccade and plots these data in graphic form. The saccades of this individual are within normal limits.

## Tracking Test

The patient is asked to follow a visual target moving back and forth following a sinusoidal waveform. A strip chart is shown tracing of the eye movements of a normal person following such a target. The eye movements are nearly perfectly sinusoidal, matching the motion.

The computer considerably increases the ability of the examiner to analyze tracking tests for a normal individual are shown. This individual has been asked to watch a computer-controlled target moving back and forth following a sinusoidal waveform at frequencies varying from 0.2 to 0.7 Hz. A sample of the eye movement tracing, with stimulus position superimposed, is shown.

After testing, the computer deletes any interpolated saccades, differentiates the eye position signal, calculates the gain of eye velocity re target velocity separately for rightward and leftward tracking at each target frequency, and plots these data in graphic form, as shown. The tracking of this individual is normal at all frequencies of target motion.

## Optokinetic Test

The patient is asked to follow a series of vertical stripes moving first to the right and then to the left at velocities ranging from about 10 to 40 degrees/second. The moving stripes provoke nystagmus, consisting of slow phases in the direction of the stripe motion periodically interrupted by fast phases in the opposite direction. In normal individuals, the velocities of the nystagmus slow phases roughly match stripe velocity for both rightward- and leftward-moving stripes, as shown.

## Gaze Test

The patient's eye movements are recorded with his eyes in various position of gaze. He is then asked to look at targets placed so that, as he fixates upon them, he gazes straight ahead, 30 degrees to his right, 30 degrees to his left, 30 degrees up, and 30 degrees down, holding each gaze position for at least 20 seconds. Then the patient closes his eyes for at least 20 seconds and performs a mental task that keeps him alert. The patient's eyes normally assume approximately a center gaze position when they are closed. Some examiners ask their patients also to hold eccentric eye positions (30 degrees to the right, 30 degrees to the left, 30 degrees up, and 30 degrees down) with their eyes closed, but most individuals have difficulty performing this task, and the tracings are hard to interpret.

The eye movement tracings of a normal individual during gaze testing is shown. This person, like many normal individuals, does not have nystagmus with eyes open under any of these test conditions, although some normal individuals do have nystagmus, known as *physiological end-point nystagmus*, with eyes open and gazing laterally. Physiological end-point nystagmus is always faint, with centripetal slow phases of equal intensity on right and left gaze.

With eyes closed, this individual has horizontal nystagmus, consisting of rightward slow phases periodically interrupted by leftward fast phases, that produces a characteristic sawtooth pattern on the eye movement tracing. Such nystagmus is quite common in normal persons but is nearly always quite weak. Most examiners use slow-phase eye velocity as a measure of nystagmus intensity. An example of such a measurement is shown for a representative nystagmus beat, where it can be seen that slow-phase eye velocity is approximately 4 degrees/second, which is within the widely accepted normal limit of 6 to 7 degrees per second (Coats, 1975; Barber & Stockwell, 1980).

If this patient's nystagmus had been stronger than 6 to 7 degrees/second, it would have been called *spontaneous nystagmus*, an abnormality that indicates a left-right asymmetry in the tonic level of activity within the vestibular system.

## Positional Test

The patient's eye movements are recorded both with eyes open at center gaze and with eyes closed while his head is in at least three positions - supine with nose upward, supine with right ear down, and supine with left ear down. If nystagmus is detected in either of the latter two positions, the patients is asked to lie on that side to determine if the nystagmus was due to neck rotation. Some examiners also test the patient with his head in other positions, such as supine with head hanging to the right, supine with head hanging to the left, or supine with head hanging straight backward.

The eye movement tracings are shown during positional testing of the same person whose gaze test results are shown in the other figure. He has the same nystagmus with eyes closed in all head positions, which is within normal limits.

## Caloric Test

The type of caloric test in most widespread use today is the *bithermal caloric test*, first described by Fitzgerald and Hallpike (1942). They irrigated each ear twice (once with water at 30 °C for 40 seconds and once with water at 44 °C for 40 seconds) and watched the patient's eyes to determine the duration of the nystagmus response. Aschan et al. (1956) described the use of electronystagmography to monitor the nystagmus response with eyes closed. Today we require that patients perform a mental task to maintain alertness while their nystagmus is being recorded, and we use peak slow-phase eye velocity rather than duration of nystagmus as the index of caloric response strength. Otherwise we perform the bithermal caloric test exactly as Aschan and colleagues described it.

Caloric stimulation produces nystagmus by modulating the firing rates of the afferent nerves of the horizontal semicircular canals. For many years, it was assumed that the mechanism of caloric stimulation was *endolymph convection,* first hypothesized by Barany (1906). According to Barany's hypothesis, the thermal stimulus causes convection currents within the endolymph of the horizontal canal that deflect the cupula and thus modulate the firing rates of the afferent nerves. The way in which the labyrinth is stimulated by this mechanism is shown. The patient is in the standard nose-up caloric test position and the right external ear canal has been irrigated with cool water. The coolness first reaches the most lateral part of the horizontal canal and cools the endolymph in that region. The cooled endolymph becomes more dense and sinks, putting pressure on the medial side of the cupula and causing it to bend laterally. Lateral bending of the cupula produces lateral bending of the imbedded hairs of the hair cells, causing the firing rates of their afferent nerves to decrease and creating an asymmetry between left and right horizontal canal inputs. The asymmetry causes rightward eye rotations, which are periodically interrupted by leftward fast phases - nystagmus with slow phase toward the irrigated ear.

Warm water irrigation of the right ear causes a nystagmus response in the opposite direction. The endolymph in the most lateral portion of the horizontal canal is heated, becomes less dense and rises, deflecting the cupula medially, increasing the firing rates of the hair cell

afferents, and producing leftward eye rotation periodically interrupted by rightward fast phases - nystagmus with slow phases away from the irrigated ear.

Coates and Smith (1967) showed that endolymph convection is not the only mechanism of caloric stimulation. They found that the caloric response was greater when the patient was in the standard nose-up position than when he was in the nose-down position and concluded that the caloric response is provoked by two mechanisms: (1) endolymph convection, which depends on head position relative to gravity and which accounts for approximately 80 percent of the response, and (2) a direct thermal effect, which is independent of head position and which accounts for approximately 20 percent of the response.

The caloric test is specifically a test of the horizontal semicircular canals, although inferences about the condition of the other labyrinthine receptors are often made on the basis of caloric test results. In theory, it should be possible to stimulate the vertical canals after appropriately positioning the head; however, in practice, vertical canal stimulation does not seem to work well. The vertical canals are deeply embedded in the temporal bone far from the caloric stimulus. Furthermore, nystagmus produced by vertical canal stimulation is primarily vertical-rotatory and difficult to monitor by the electronystagmographic method.

The caloric stimulus used by most examiners consists of 250 mL of water irrigated into the external ear canal within 30 seconds. The temperature of the water is 30 °C for the cool irrigation and 44 °C for the warm irrigation. Some examiners use air (8 liters at 24 °C and 50 °C within 60 seconds) instead of water as caloric stimuli. Others use a "closed-loop" system, in which water continuously circulates within a watertight system that includes a small balloon that inflates in the external ear canal during the irrigation. All three irrigating methods - water, air, and "closed-loop" - produce approximately equivalent stimuli.

Caloric stimulation produces nystagmus responses that begin approximately 20 seconds after the onset of the irrigation, reach peak intensity approximately 40 seconds later, thereafter decline, and finally disappear after about 3 minutes. If a strip chart recorder is used to monitor the responses, the examiner inspects the nystagmus tracing, locates the point at which each response is most intense, and measures the slow-phase velocities of several nystagmus beats at that point. Figure shows strip chart tracings of peak caloric nystagmus responses for a normal individual. Measurements of peak slow phase velocities yield 18 degrees/second for the right warm response, 22 degrees/second for the left warm response, and 20 degrees/second for the left cool response.

If caloric responses are fed into a computer, it is feasible to measure the intensities of the entire responses, and not just the peaks. An example of computerized caloric test results for a normal individual is shown. The computer calculates slow-phase velocities at 1-second intervals and displays slow phase velocity versus time for all four responses.

The basic assumption of the caloric test is that both ears receive equal caloric stimuli; therefore, if both ears are normal, they should produce approximately equal caloric responses.

If not, the patient is said to have a *unilateral weakness,* which is evidence of a lesion of the labyrinth or vestibular nerve on the side of the weak response. To quantify the difference in caloric response strength of the two ears, most examiners use the following formula, proposed by Jongkees and Philipszoon (1964):

100 x ((RW+RC) - (LW+LC))/(RW+RC+LW+LC) = unilateral weakness,

where RW, RC, LW, and LC are peak slow-phase velocities of the responses to right warm, right cool, left warm, and left cool irrigation, respectively. The normal limit of ± 20 to 25 per cent is widely accepted (Barber and Stockwell, 1980). Calculation of unilateral weakness for the responses shown in Figure yields

100 x ((18+25) - (22+20))/(18+25+22+20) = 1 per cent

which is well within normal limits.

Sometimes the caloric responses of one ear are not only weak but entirely absent. In such cases, most examiners irrigate that ear again with ice water to confirm the absence of response. The usual procedure is to place the patient's head on its side so that the ear to be irrigated is facing upward. The examiner pours 2 ml of ice water into the external ear canal, leaves it there for 20 seconds, then turns the patient's head sot the water drains out, and finally positions the head nose upward. The patient's eyes are closed, and his eye movements are recorded for at least 2 minutes. The ice water produces a much stronger stimulus than the standard bithermal irrigations. In normal individuals, it provokes a vigorous nystagmus response, with peak slow-phase velocities of approximately 40 to 50 degrees/second. If the examiner elicits such a response after failing to provoke any response to the bithermal caloric irrigations, he would conclude that the labyrinth is normally responsive and that the lack of response to bithermal stimuli was due to inadequate irritations or to insufficient patient alertness. If the examiner elicits a weak response, he would conclude that the labyrinth or vestibular nerve is damaged but not totally destroyed. If he elicits no response, the examiner would conclude that the labyrinth or vestibular nerve is severely damaged, but he could not safely conclude that they are "dead", since caloric irrigation stimulates only the horizontal semicircular canal receptors.

If all patients who underwent caloric testing displayed only normal responses or unilateral weaknesses, it would not be necessary to irrigate each ear twice. One irrigation of each ear, either warm or cool, would suffice. However some patients have a *directional preponderance* of caloric responses; that is, the peak intensities of their caloric responses are stronger in one direction than in the other direction. Therefore, it is necessary to provoke responses in both directions from each ear in order to obtain a valid assessment of unilateral weakness. The formula of Jongkees and Philipszoon employs the sum of the warm and cool responses as a measure of caloric response strength for each ear and thus cancels the effect of any preponderance.

A directional preponderance of caloric responses is a manifestation of vestibular asymmetry, the same asymmetry that produces spontaneous nystagmus. Caloric nystagmus is

superimposed upon spontaneous nystagmus, so peak response intensity in the same direction is stronger than peak response intensity in the opposite direction; hence the preponderance.

To quantify directional preponderance, Jongkees and Philipszoon (1964) proposed the following formula:

100x ((RW+LC)-(LW+RC))/(RW+LC+LW+RC) = directional preponderance

where RW, LC, LW, and RC are the same as in the formula for unilateral weakness. This formula is intended to yield a comparison of rightward and leftward caloric response intensities in the same way that the formula for unilateral weakness yields a comparison of right ear and left ear response intensities. However the rationale here is faulty, because the underlying abnormality is a response asymmetry, and therefore it is inappropriate to normalize for peak response intensities. It would be more rational to express preponderance in terms of the asymmetry, which should roughly correspond to the slow-phase velocity of the patient's spontaneous nystagmus.

Although the caloric test is highly sensitive to unilateral labyrinth or vestibular nerve lesions, it is fairly insensitive to bilateral lesion. The reason is that the caloric stimulus is uncalibrated. Different patients receive different amounts of stimulation, depending on the size and shape of the external ear canal and middle ear. Bilateral lesions produce weak caloric responses from both ears, but these weaknesses must be severe to fall below normal limits. A *bilateral weakness* is said to exist if the sum of the peak warm and peak cool responses fail to reach 12 degrees/second for either ear (Barber and Stockwell, 1980). A bilateral caloric weakness should be confirmed by bilateral ice water irrigations. It should also be confirmed by rotation testing, if such testing is available, because rotation tests have demonstrated horizontal canal function even when ice water irrigations have failed to provoke a response from either ear (Baloh et al, 1984).

Pursuit function can be evaluated by the caloric test when the patient is asked to open his eyes and fixate during the caloric response. Fixation should reduce nystagmus slow-phase velocity by at least 40 per cent (Demnanez and Ledoux, 1970; Alpert, 1974). If not, the patient has *failure of fixation suppression.* The results of this test nearly always confirm the results of the tracking and optokinetic tests, because suppression of inappropriate vestibular nystagmus is performed by the pursuit system. However, some disagreement exists on this point, since there have been reports of dissociation between the results of tracking and fixation suppression tests (Dell'Osso et al, 1981; Chambers and Gresty, 1983; Mira et al, 1983). On the other hand, interpretation of fixation suppression test results is sometimes problematic, because the amount of nystagmus to be suppressed is uncontrolled, being dependent on the intensity of caloric nystagmus that happens to be present when the patient is asked to open his eyes. Even normal individuals have difficulty suppressing caloric nystagmus when it is intense, and even persons with defective pursuit can suppress it when it is weak.

## Rotation Test

The type of rotation test in most widespread use today is called the *slow harmonic acceleration test* (Wolfe et al, 1978a, b), which employs a computer-controlled system to rotate the patient and monitor eye movements. The patient is seated in a chair in complete darkness with his head positioned so that the horizontal semicircular canals are in the plane of rotation and firmly restrained so that it follows chair motion. Typically the patient undergoes sinusoidal oscillation about a vertical axis at oscillation frequencies of 0.01, 0.02, 0.04, 0.08, 0.16, 0.32, and 0.64 Hz, with peak angular velocity of 50 degrees/second at all frequencies. The patient's horizontal eye movements are monitored by the same method used in electronystagmography, and the relationships between eye and head movement are examined. These relationships are illustrated in Figure for a normal person during a single cycle of oscillation. The oscillation frequency in this case is 0.16 Hz, which is near the middle of the test frequency range. The two upper tracings show head angular position and angular velocity, respectively. The third tracing shows horizontal eye position. The patient has nystagmus with leftward slow phases while moving rightward and nystagmus with rightward slow phases while moving leftward. The computer differentiates the eye position signal, removes the fast phases of nystagmus, and displays slow-phase eye velocity, shown in the bottom tracing.

Three features of this response are of clinical interest. (1) The response of this normal person is *symmetric;* that is, rightward and leftward slow phase velocities are roughly equal. (2) Eye velocity and head velocity are opposite in *phase;* that is, the direction of slow phase eye velocity is exactly opposite the direction of head velocity at all times, reflecting the fact that the vestibulo-ocular reflex functions to compensate for head movements. (3) The amplitude, or *gain,* of slow-phase eye velocity is about 60 per cent of head velocity, reflecting the fact that the compensation provided by the vestibulo-ocular reflex is imperfect, even in normal individuals. Nystagmus slow-phase velocities are not quite fast enough to compensate entirely for head velocities.

Figure summarizes relationships between slow-phase eye velocity and head velocity for the same normal individual at four different test frequencies. At the higher frequency of 0.64 Hz, these relationships are approximately the same as they are at 0.16 Hz. Slow-phase eye velocity is the mirror image of head velocity, although the gain of eye velocity is only about 60 per cent, and rightward and leftward velocities are roughly symmetric. When the person is oscillated at progressively lower frequencies of 0.04, and finally 0.01 Hz, these relationships show progressive change. Slow-phase velocities exhibit progressively lower gains, and they are no longer exactly opposite in phase, but rather display progressively larger phase leads; that is changes in slow phase eye velocity occur more and more in advance of changes in head velocity. The low-frequency gain reduction and phase lead reflect the fact that the horizontal semicircular canal system functions as an integrating angular accelerometer. It detects angular accelerations of the head and produces an output related to head angular velocity. Part of this integration is performed mechanically within the semicircular canal itself. Further integration is performed within the central vestibular system, extending the low-frequency sensitivity of the vestibulo-ocular system. However, even with this integration, the vestibulo-ocular system performs well only for relatively

fast head motions. The oscillatory frequencies used in the slow harmonic acceleration are at the lower end of its operating range, and therefore, as oscillation frequency decreases, the horizontal canal system becomes less sensitive to the head motions and fails to integrate the head motion signals completely, yielding lower gains and greater phase leads.

Figure shows graphic plots of the phase, gain, and asymmetry data over the entire range of test frequencies. Phase and gain values show the progressive phase lead and gain reduction as oscillation frequency decreases. Asymmetry values are nearly zero at all frequencies.

Some examiners also perform the slow harmonic acceleration test while the patient fixates on a small target light that rotates with him (Baloh et al, 1982). This procedure tests the patient's ability to suppress the pendular nystagmus induced by the rotation. It is analogous to the test for fixation suppression of caloric nystagmus, but offers the advantage that the amount of vestibular stimulation is precisely controlled. Most normal individuals can completely suppress the nystagmus generated by the stimuli used in the slow harmonic acceleration test.

Some examiners also perform the slow harmonic acceleration test while the patient rotates in a lighted room with striped walls (Baloh et al, 1092). This procedure stimulates not only the vestibular system but the pursuit and optokinetic systems as well. In most normal individuals, it produces nystagmus with a gain of approximately 1.0 and no phase advance at any oscillation frequency. The enhancement of nystagmus under these conditions is due to the contribution of the pursuit and optokinetic
systems.

## Posturography

Until recently, evaluation of vestibular function by measuring postural stability was of limited clinical value. The primary reason was that these measurements were made as the patient stood on a firm surface, in which case postural stability depended not only upon vestibular cues but also upon cues from the somatosensory system. Nashner (1970) analyzed postural control in the pitch plane during quiet standing, showing that either visual or somatosensory cues by themselves are sufficient for maintaining postural stability when the patient stands on a firm surface in a lighted room and that the vestibular system plays a secondary role under these circumstances. In order to demonstrate a vestibular deficit, one must force the patient to rely solely upon vestibular cues, and in order to do that, one must defeat the visual and somatosensory systems. Nashner and colleagues developed a computerized method of testing vestibular control of posture based on this concept (Nashner et al, 1982; Black, 1985; Black and Nashner, 1985). Their test is called the *sensory organization test.* The patient is asked to stand on a force plate that is capable of rotating in the pitch plane about an axis colinear with his ankle joints. He stands facing a visual scene subtending his entire field of view that is capable of rotating about the same axis. As the patient sways back and forth, the force plate measures the changes in the position of his body's center of pressure. These data are transmitted to the computer, which calculates the angle of body sway in the pitch plane.

The patient is tested under six different conditions, as shown in Figure. Under conditions 1 to 3, the supporting surface is fixed so that the patient has correct somatosensory cues, and visual cues are varied. Under condition 1, the visual scene is fixed so that visual cues are correct. Under condition 2, the patient closes his eyes, denying visual cues. Under condition 3, the visual scene is rotated in proportion to the angle of body sway so that visual and somatosensory cues are in conflict, with somatosensory cues being correct and visual cues erroneously indicating that the subject is motionless. Under conditions 4 to 6, the supporting surface is rotated in proportion to the angle of body sway, and visual cues are varied as before. Under condition 4, the visual scene is fixed so that visual and somatosensory cues are again in conflict, with visual cues being correct and somatosensory cues erroneously indicating that the subject is motionless. Under this condition, the only correct cue regarding the direction of gravity comes from the vestibular system. Under condition 6, the visual scene is rotated in proportion to the angle of body sway so that both visual and somatosensory cues indicate that the patient is motionless, and again the only correct cue regarding the direction of gravity comes from the vestibular system.

Figure shows the calculated body sways of a normal individual under these six test conditions. This individual underwent three 20-second trials under each condition. He was able to stand virtually motionless under conditions 1 to 3, when the supporting surface was fixed, regardless of the visual conditions. His stability was somewhat worse under condition 4 when the supporting surface rotated in proportion to body sway, and still worse under conditions 5 and 6 when visual information was absent or incorrect. These body sway data are used to calculate a performance index, shown in Figure. This performance index is the measured peak-to-peak body sway during the 20-second trial divided by the total amount of body sway that would be possible without actually falling - assumed to be 12 degrees.

The method developed by Nashner and colleagues includes another test, the *motor coordination test*, in which the patient undergoes a series of translations of the supporting surface in the backward direction followed by a series of backward rotations about an axis colinear with the ankle joints. This series is followed by an identical series of translations in the forward direction followed by a series of forward rotations. The patient's body sway is monitored by the force plate, and various response indices are calculated from these data by the computer, including response latencies, amplitudes, and left-right asymmetries. In addition, the ability of the patient to adapt to support surface rotation following repeated exposures to support surface translation is calculated. The motor coordination test is intended primarily to evaluate patients with disorders of movement coordination caused by head injuries and by metabolic and degenerative disorders of the brain and neuromuscular system (Nashner, 1983). It rarely yields abnormal findings in patients with the primary complaint of dizziness.

## Abnormalities Detected by Nonspecific Tests

Electronystagmography, the rotation test, and posturography do not yield diagnoses, but they often detect vestibular dysfunction and sometimes localize the site of dysfunction in patients with dizziness or unsteadiness. Abnormalities nearly always fall into one of seven categories. The first three categories indicate labyrinth or vestibular nerve lesions - an *acute unilateral peripheral*

*vestibular lesion,* a *chronic unilateral peripheral vestibular lesion,* or *bilateral peripheral vestibular lesions.* The next two categories indicate vestibular dysfunction, but are nonlocalizing - either a *nonlocalizing asymmetry* or *nonlocalizing positional nystagmus.* The final two categories indicate lesions involving vestibular pathways within the central nervous system - either a *symmetric central vestibular lesion* or an *asymmetric central vestibular lesion.*

It cannot be claimed that this list of categories is exhaustive. Occasionally, dizzy or unsteady patients show other types of abnormality, although such patients are rare. Neither are these categories mutually exclusive. Occasionally dizzy or unsteady patients show abnormalities in more than one category.

Typical examples of test findings in each category follow.

**Acute Unilateral Peripheral Vestibular Lesion**

The patient was a young man who suffered a sudden onset of severe vertigo with nausea and vomiting. His symptoms gradually abated over a period of several days but were greatly exacerbated when he moved his head. His hearing was normal. The diagnosis was vestibular neuritis. Vestibular function testing was performed on the day following the onset of symptoms.

Abnormalities found by electronystagmography, rotation testing, and posturography are shown in Figure. Gaze and positional testing revealed spontaneous nystagmus, indicating a left-right asymmetry in the tonic level of vestibular activity. This nystagmus had rightward slow phases with eyes open on leftward gaze and was greatly enhanced with eyes closed, with slow-phase velocities of approximately 16 degrees/second. The intensity of the nystagmus was not measurably altered by changes in head position.

The tracking test showed a pursuit defect for leftward-moving targets. Defective pursuit generally indicates central nervous system dysfunction, but here it did not. It was merely secondary to the presence of nystagmus with eyes open. The optokinetic test confirmed the result of the tracking test, showing poor nystagmus for leftward-moving stripes.

The caloric test showed a unilateral weakness of caloric responses from the right ear. Calculation of unilateral weakness by the formula of Jongkees and Philipszoon yielded:

100 x ((-16+16)-(37+2))/(-16+16+37+2)=100 per cent

indicating an absence of response from the right ear. This unilateral weakness was the key finding in this patient, since it demonstrated a severe lesion of the right labyrinth or vestibular nerve and explained both the patient's symptoms and his spontaneous nystagmus. The lesion suddenly abolished the resting input coming from the right ear, thereby producing an asymmetry of resting inputs coming from the two labyrinths. This asymmetry mimicked the asymmetry that would be produced by head movement away from the side of the lesion, and the result was tonic nystagmus that would be appropriate for such movement; that is, nystagmus with slow phases

toward the damaged side. The caloric responses were superimposed on the spontaneous nystagmus. The slow-phase velocity of this nystagmus formed a new baseline of approximately 16 degrees/second, and the caloric responses were roughly symmetric about this new baseline. Irrigation with ice water also failed to provoke any response from the right ear. The saccade test showed no abnormalities.

The rotation test showed greater-than-normal phase leads at the lower oscillation frequencies and an abnormally low gain at the lowest frequency of 0.01 Hz. The cause of such a low-frequency phase lead and gain reduction is not completely understood but is presumably due to the fact that the central vestibular system is largely responsible for the low-frequency response of the vestibulo-ocular system. Following severe vestibular injury, the central nervous system contribution is suppressed, thus somewhat ameliorating the severity of the symptoms. In addition to the increased low-frequency phase lead and gain reduction, there was a strong asymmetry to the right, which roughly corresponded to the slow-phase velocity of the patient's spontaneous nystagmus at low frequencies and increased at higher frequencies.

Posturography showed that postural stability was normal under conditions 1 to 4, but abnormal under conditions 5 and 6, when the patient was required to rely solely upon vestibular cues. In fact, he was unable to stand at all under these conditions and went into free fall immediately after the trials started. Such behavior is similar to that exhibited by a person with no vestibular function, which is remarkable because this patient still had vestibular input coming from his left ear. Presumably it reflects central suppression of vestibulo-spinal signals akin to the suppression of vestibulo-ocular responses to low-frequency rotation.

This example illustrates the abnormalities typically seen in patients with severe lesions. Patients with milder lesions show less severe unilateral weaknesses and weaker spontaneous nystagmus. In fact their nystagmus may be so weak that it is completely suppressed with eyes open, in which case the results of the tracking and optokinetic tests would be normal. In addition, the rotation test may not show abnormal gain reductions and phase leads at low oscillation frequencies, and posturography may also fail to show abnormalities.

## Chronic Unilateral Peripheral Vestibular Lesions

The patient was a middle-aged woman with a progressive right sensorineural hearing loss and no complaint of dizziness, although upon close questioning, she did admit to occasional mild unsteadiness. Imaging studies revealed a right acoustic neuroma.

Abnormalities are shown in Figure. Electronystagmography showed only a severe right unilateral weakness of caloric responses. The rotation test showed greater-than-normal phase leads at the lower oscillation frequencies. Gain and asymmetry values were within normal limits. The patient did have nystagmus with rightward slow phases with eyes closed, but it was not strong enough to be significant. Posturography showed no abnormalities.

This case illustrates the profound plasticity of the vestibular system. If a peripheral vestibular system lesion develops slowly, as is generally the case with acoustic neuromas, the compensation process is able to continuously rebalance the asymmetry and therefore prevent the vertigo and tonic nystagmus that would otherwise occur. Even when the lesion develops suddenly, as it did in the previous case, compensation would gradually rebalance the asymmetry over a period of weeks and months. Thus, the acute lesion of a patient with test findings like those shown in Figure would evolve into a chronic lesion and yield test findings like those shown in Figure if the patient were tested after compensation was complete.

## Bilateral Peripheral Vestibular Lesions

The patient was a middle-aged man who complained of persistent unsteadiness following a course of gentamicin therapy. The diagnosis was ototoxicity.

Abnormalities are shown in Figure. Bithermal caloric irrigations provoked no response from either ear. This result was confirmed by ice water irrigations, which also failed to provoke any response from either ear, and by rotation testing, which provoked no measurable response at any oscillation frequency. Posturography provided additional confirmation of the caloric test result, since the patient showed normal postural stability under conditions 5 and 6, when he was required to rely solely upon vestibular cues. The other vestibular function tests showed no abnormalities.

## Nonlocalizing Asymmetry

This patient's history was almost identical to that of the patient whose test results are shown in Figure. He suffered an acute onset of severe vertigo with nausea and vomiting, with gradual abatement of symptoms. No diagnosis was made. Vestibular function testing was performed several days following the onset of symptoms.

Abnormalities are shown in Figure. Gaze and positional testing showed spontaneous nystagmus with rightward slow phases with eyes closed, indicating an asymmetry of tonic vestibular activity. There was no nystagmus with eyes open. Saccades, tracking, and optokinetic nystagmus were normal.

Caloric responses were normal and approximately equal bilaterally - a key finding. Since there was no unilateral weakness of the right ear, the patient's spontaneous nystagmus (and his symptoms) could not be explained by a sudden loss of resting input coming from the right ear. Thus, while electronystagmography did yield evidence of vestibular dysfunction, it did not provide localization.

Rotation testing showed a greater-than-normal phase lead at the lowest oscillation frequency, which was further evidence of vestibular dysfunction, but which also did not provide localization. Both caloric and rotation tests showed a marked rightward asymmetry, consistent with the patient's spontaneous nystagmus. Posturography showed no abnormalities.

## Nonlocalizing Positional Nystagmus

The patient was a middle-aged woman who complained of vertigo while lying on her side and persistent lightheadedness at other times.

As shown in Figure, the position test revealed nystagmus with leftward slow phases when the patient was lying supine with right ear undermost, and nystagmus with rightward slow phases when she was lying supine with left undermost. This nystagmus was totally suppressed when the patient's eyes were open. It was unchanged when the patient was asked to lie on her right and left sides, proving that it was not caused by neck rotation. The remainder of the electronystagmographic examination showed no abnormalities. Rotation testing and posturography also yielded normal results.

his result provided evidence of vestibular dysfunction, supporting the patient's complaint, but it did not localize the site of dysfunction. Such nystagmus could be caused by a variety of vestibular disorders, either peripheral or central (Barber, 1978).

Patients exhibit many varieties of positional nystagmus, but generally the nystagmus is horizontal with right ear undermost and/or with left ear undermost. The nystagmus may beat in either direction. If these varieties of positional nystagmus are adequately suppressed by visual fixation, then they are nonlocalizing. The exception is nystagmus that changes direction in a single head position. Such nystagmus indicates a central nervous system lesion (Barber and Stockwell, 1980).

## Symmetric Central Vestibular Lesion

The patient was an elderly woman with progressive unsteadiness. The diagnosis was cerebellar degeneration.

Vestibular function tests showed multiple abnormalities, shown in Figure. The gaze test showed gaze-evoked nystagmus with centripetal slow phases with eyes open, and the tracking test showed defective tracking bilaterally. These two findings together usually indicate a disorder of the cerebellar system (Zee et al, 1976).

The caloric test showed a bilateral weakness, and this result was confirmed by ice water caloric irrigations and by rotation testing, which showed a virtual absence of response to all oscillation frequencies. The results of caloric and rotation testing were the same as those found in the patient with bilateral peripheral vestibular lesions; however, in this case, the absence of vestibular function was presumed to be due to a central nervous system disorder, since clear evidence of such a disorder was provided by the gaze and tracking tests.

Posturography have additional evidence of central nervous system dysfunction. The patient was markedly unsteady under all testing conditions. In fact, she was able to stand at all only under condition 1. Under all other conditions, she was not able to stand long enough to complete

a 20-second trial. This deficit was much more severe than those displayed by patients with purely peripheral vestibular lesions, implying that non-vestibular balance control pathways were also involved.

Saccades were normal. There was no nystagmus with eyes closed. The results of the optokinetic test were consistent with tracking test results.

In this case, the central vestibular involvement was severe, resulting in bilateral absence of nystagmus responses to caloric and rotation stimuli. In cases of less severe involvement, nystagmus induced by such stimuli may be normal or it may be unusually intense, in which case the patient has *hyperresponsiveness;* it may beat in opposite the expected direction, in which case he has *inversion;* or it may beat vertically, in which case the patient has *perversion.* Hyperresponsiveness, inversion, and perversion of nystagmus are extremely rare.

## Asymmetric Central Vestibular Lesion

The patient was a middle-aged woman who suffered an acute onset of severe vertigo with nausea and vomiting, with gradual abatement of symptoms over the next several days. No diagnosis was made. Vestibular function testing was performed 1 week after the onset of symptoms.

Abnormalities are shown in Figure. Gaze and positional testing showed strong spontaneous nystagmus with rightward slow phases. A key finding was that her nystagmus was not significantly suppressed by visual fixation, which is evidence of central nervous system dysfunction. Tracking was defective for leftward-moving targets. Caloric responses were normal and equal bilaterally. Rotation testing showed marked phase leads, especially at the lower oscillation frequencies. Both caloric and rotation responses showed marked rightward asymmetries, consistent with the patient's spontaneous nystagmus. Posturography showed marked instability under conditions 4 to 6, when the supporting surface was servodriven to deny somatosensory cues. This deficit was more severe than those displayed by patients with purely vestibular lesions, since it included condition 4, implying that nonvestibular balance control pathways were also involved. Saccades were normal. The results of the optokinetic test were consistent with tracing test results.

This patient's history and test results were similar to those of the patient with nonlocalizing asymmetry; however, the asymmetry in this case was presumed to be caused by central vestibular dysfunction because of concomitant central findings on the gaze and tracking tests and posturography.

## Other Central Lesions

These vestibular function tests occasionally reveal other evidence of nonvestibular central nervous system dysfunction.

The saccade test detects abnormalities caused by a variety of disorders. Reduced peak velocity can occur in paresis of ocular muscles or ocular motor nerves, in lesions of the medial longitudinal fasciculus, and in various types of degenerative and metabolic diseases of the central nervous system. Prolonged saccadic latency is the hallmark of Parkinson's disease, but it can occur in various other degenerative and metabolic diseases. Impaired saccadic accuracy, known as saccadic dysmetria, is the hallmark of cerebellar system disease, but it also can occur in brain stem disease, ocular motor disorders, and visual defects. A thorough review of saccadic disorders is provided by Leigh and Zee (1983).

The pursuit test also detects abnormalities caused by a variety of disorders. Defective pursuit may simply be due to aging, inattentiveness, or drugs, particularly tranquilizers and anticonvulsants. Pursuit is usually defective if the patient has nystagmus with eyes open. Otherwise, defective pursuit is evidence of central nervous system dysfunction, most commonly of the vestibulo-cerebellum. Pursuit defects generally are asymmetric; that is, they are more severe for targets moving in one direction than in the other. Leigh and Zee (1983) provide a thorough discussion of pursuit function and pathology.

The gaze and positional tests occasionally also yield ocular abnormalities caused by central nervous system disorders. Electronystagmography may be helpful in documenting these abnormalities but generally is not required, because they are present with eyes open and therefore are rapidly apparent on physical examination. A concise description of nonvestibular nystagmus and other abnormal eye movements seen in the gaze and positional tests is given by Daroff and co-workers (1978). Electronystagmographic tracings of many of these are illustrated by Barber and Stockwell (1980).

## Specific Vestibular Function Tests

### The Hallpike Maneuver

The Hallpike Maneuver is designed to provoke a nystagmus response in patients who have benign positional vertigo. The maneuver begins with the patient in the sitting position, as shown in Figure. The patient's head is turned 45 degrees toward the ear that is suspected of dysfunction. Then the examiner, standing behind the patient, grasps the patient's head and pulls him briskly backward, without rotating his neck, so that he is lying supine with his head hanging over the end of the examining table. After the movement, the examiner, still holding the patient's head, asks him to look straight ahead and observes the patient's eyes for at least 20 seconds. The patient is then returned to the sitting position. If a response was elicited, the examiner repeats the same maneuver as many times as is necessary to determine if the response fatigues. Then the maneuver is performed with the patient's head turned 45 degrees to the other side and, if a response is elicited, the maneuver is repeated to determine if it fatigues.

Normal individuals have a few beats of nystagmus during the backward movement, but none after the movement has been completed. Some patients with benign positional vertigo display, after the movement has been completed, a burst of intense nystagmus - *benign positional*

*nystagmus* - that is the hallmark of this disorder (Baloh et al, 1987). In nearly all patients, only one ear is involved, and the burst of nystagmus appears when the Hallpike maneuver places the involved ear undermost. Rarely, both ears are involved, and then the nystagmus appears when both ears are undermost.

This nystagmus response usually has four distinct features. (1) The nystagmus is *delayed in onset;* that is, there is an interval of at least a few seconds after the patient reaches the head-hanging position before the nystagmus begins. (2) The nystagmus is *transient;* that is, it rapidly builds in intensity, reaches a crescendo, slowly abates, and finally disappears as the head remains in position. (3) The nystagmus is *accompanied by vertigo,* usually intense vertigo, that follows the same time course as the nystagmus. (4) The nystagmus is *fatigable;* that is, it becomes progressively weaker in intensity when the Hallpike maneuver is repeated.

A tracing of benign positional nystagmus is shown in Figure. The patient was a middle-aged man suffering from attacks of vertigo when lying down on his left side. The Hallpike maneuver provoked a nystagmus response with left ear undermost that possessed all of the features described above. The horizontal tracing showed rightward-beating nystagmus, and the vertical tracing showed upward-beating nystagmus. This nystagmus response also included a prominent rotatory component, which could be readily appreciated when visually observing the patient's eyes, but which did not appear in the electronystagmographic tracing. No response was elicited by the Hallpike maneuver with the right ear undermost, and the other vestibular function tests showed no abnormalities.

Available evidence strongly suggests that benign positional vertigo is caused by a malfunction of the posterior semicircular canal. Benign positional nystagmus is identical to nystagmus that would be produced by a burst of posterior canal excitation (Baloh et al, 1979), and section of the posterior canal afferent nerve abolishes this nystagmus (Gacek, 1978). The reason for this malfunction is not completely understood, but a plausible explanation has been offered by Schuknecht (1969). He proposed that otoconia become dislodged from the utricular macula and settle upon the posterior canal cupula, making it heavier than the surrounding endolymph and therefore sensitive to gravity. As a result, any head movement that reorients the posterior canal relative to gravity deflects its cupula, with resulting vertigo and nystagmus. The Hallpike maneuver completely inverts the posterior canal and produces benign positional nystagmus, which is an extreme manifestation of this disorder.

Not all patients with benign positional vertigo display a nystagmus response following the Hallpike maneuver, perhaps because they have fatigued the response by their own natural head movements before testing. In any case, the lack of a response does not rule out the disorder. Also, patients with other disorders sometimes display nystagmus after the Hallpike maneuver (Harrison and Ozsahinoglu, 1975), although nearly always their responses fail to meet all of the criteria for benign positional nystagmus.

## Pressure Test

The pressure test is another test designed to detect a specific disorder - a perilymph fistula. Either the patient's eye movements or his body sway are monitored as pressure changes are induced in the external ear.

When eye movements are monitored, electrodes are placed to record horizontal eye movements by electronystagmography with the patient's eyes closed (Daspit et al, 1980). Then, using an impedance bridge, the examiner rapidly increases the pressure in the external ear canal to +200 mm of water, maintains it at this level for 15 to 20 seconds and then rapidly decreases the pressure to -400 mm of water, maintains it at this level for 15 to 20 seconds, and finally returns it to atmospheric pressure. This sequence is repeated several times.

The pressure test does not provoke nystagmus or vertigo in normal individuals, but some patients with perilymph fistulas do display these signs when the test is performed on the involved ear. An example of pressure-induced nystagmus is shown in Figure. The patient was a middle-aged woman with persistent dizziness following stapedectomy of the left ear. The other vestibular function tests showed no abnormalities. The presence of a left perilymph fistula was confirmed at surgery.

When the pressure test provokes nystagmus, its direction may be either toward or away from the involved ear, and it is generally the same for both positive and negative pressure changes. The nystagmus and vertigo usually persist for as long as the pressure change is held.

When body sway is recorded, the patient is asked to stand on the posturography platform with eyes closed and the supporting surface servodriven to follow body sway, thus denying both visual and somatosensory cues (Black et al, 1987). Then, using a computer-controlled impedance bridge, the pressure in the external canal of the suspected ear is rapidly increased to +300 mm of water and returned to atmospheric pressure. Ten trials are performed. Body sway in both the anteroposterior and lateral directions is monitored during each trial and the computer calculates average body sway over the 10 trials. This 10-trial sequence is then repeated with pressure decreased to -300 mm of water.

This procedure does not produce body sways that are time-locked to the pressure changes in normal individuals, but it does produce such time-locked body sways in some patients with perilymph fistulas. An example is shown in Figure. The patient was a young woman with chronic infection of the right middle ear and dizziness related to abrupt atmospheric pressure changes. The other vestibular function tests showed no abnormalities. A right perilymph fistula was found at surgery.

## Summary

Vestibular function tests are used by otolaryngologists to evaluate patients who suffer from dizziness and unsteadiness. Five tests are in general clinical use - ENG (which includes the saccade, tracking, optokinetic, gaze, positional, and caloric tests), the rotation test, posturography, the Hallpike maneuver, and the pressure test. Three of these tests (electronystagmography, the rotation test, and posturography) are nonspecific in the sense that they generally do not identify specific etiologies. The two remaining tests (the Hallpike maneuver and the pressure test) are specific. The Hallpike maneuver strongly suggest benign positional vertigo, and the pressure test suggests a perilymph fistula.

Common types of test results are summarized in Table 1. This list is neither exhaustive nor mutually exclusive, but it does include most of the abnormalities seen in patients being evaluated by otolaryngologists for dizziness and unsteadiness.

Patients with severe acute unilateral peripheral vestibular lesions show a unilateral weakness and asymmetry of caloric responses and spontaneous nystagmus with slow phases toward the side of the lesion that is adequately suppressed by visual fixation. On rotation testing, such patients show asymmetry toward the side of the lesion as well as greater-than-normal phase leads and gain reductions at the lower oscillation frequencies. On posturography, they show marked instability when required to rely solely upon vestibular cues. If their spontaneous nystagmus is incompletely suppressed by visual fixation, these patients usually show defective tracking and optokinetic nystagmus for visual targets moving toward the side opposite the lesion.

Patients with milder lesions show a unilateral caloric weakness, spontaneous nystagmus that is adequately suppressed by visual fixation, and asymmetry on the caloric and rotation tests, but usually show no abnormalities on the tracking and optokinetic tests and posturography, and no abnormal phase lead or gain reduction at low oscillation frequencies on the rotation test. These patients usually show no abnormalities on the other vestibular function tests.

Patients with chronic unilateral peripheral vestibular lesions show a unilateral caloric weakness and, if the lesions is severe, greater-than-normal phase leads at the lower oscillation frequencies on rotation testing. Such patients usually show no abnormalities on the other vestibular function tests.

Patients with bilateral peripheral vestibular lesions show a bilateral caloric weakness, lower-than-normal gains on rotation testing, and marked instability on posturography when required to rely solely upon vestibular cues. They usually show no abnormalities on the other vestibular function tests.

Patients with nonlocalizing asymmetry show spontaneous nystagmus that is adequately suppressed by visual fixation on gaze and positional tests, and they usually show no abnormalities on the other vestibular function tests.

Patients with nonlocalizing positional nystagmus show nystagmus that is adequately suppressed by visual fixation on the positional test, usually with right ear undermost and/or with left ear undermost, and they usually show no abnormalities on the other tests.

Patients with symmetric central vestibular lesions usually show signs of concomitant nonvestibular involvement, such as gaze-evoked nystagmus and defective tracking and optokinetic nystagmus. On posturography, they usually also show evidence of nonvestibular involvement. If vestibular involvement is severe, such patients usually show bilateral weakness on the caloric test, and lower-than-normal gains on the rotation test. If vestibular involvement is less severe, they may show normal responses on the caloric and rotation tests, or they may occasionally show hyperresponsiveness, inversion, or perversion of induced vestibular responses. On the rotation test, they may show much greater-than-normal phase leads and reduced gains at the lower oscillation frequencies.

Patients with asymmetric central vestibular lesions show spontaneous nystagmus that is inadequately suppressed by visual fixation and usually show other signs of concomitant nonvestibular involvement. They may show normal responses on the caloric and rotation tests or may occasionally show hyperresponsiveness, inversion, or perversion. On posturography, they usually show signs of nonvestibular involvement. On the rotation tests, they may show greater-than-normal phase leads and reduced gains at the lower oscillation frequencies.

Patients with benign positional vertigo sometimes show benign positional nystagmus on the Hallpike maneuver with the involved ear undermost and usually show no abnormalities on the other vestibular function tests.

Patients with a perilymph fistula sometimes show a positive response on the pressure test administered to the involved ear and usually show no abnormalities on the other tests.