

Chapter 146: Vestibular Function Tests

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Otolaryngologists often perceive vestibular function tests as being less clinically useful than functional tests of the auditory system and, as a result, underestimate their full potential. Several factors contribute to this perception. The anatomy and physiology of the peripheral and central vestibular system are at least as complex as those of the auditory system, but otolaryngologists in general possess less knowledge and interest in the vestibular system. For many years, otolaryngologists considered it sufficient to separate peripheral vestibular problems from those of CNS origin, treating peripheral lesions and referring nonperipheral lesions to neurologists. This no longer is sufficient; today knowledge of the visual and somatosensory systems also is necessary to complement that of the vestibular system if assessment of patients with dizziness or imbalance is to be complete. Proper understanding and interpretation of current tests designed to assess the contributions from all three sensory systems require detailed knowledge of the sensory and motor systems under investigation, as well as an appreciation of the strengths and limitations of each test.

The assessment of patients with balance disorders or dizziness has changed significantly over the past several years (Cyr, 1991). Technical improvements have resulted in the refinement of eye movement analysis (computerized electronystagmography (ENG) and led to the development of tests of rotation (rotary chair test) and standing balance / posture (computerized dynamic posturography). This has broadened the otolaryngologist's ability to assess not only the vestibular system, but also aspects of the visual and somatosensory systems. In some instances the clinical application has lagged behind the technical advances, which has made it somewhat difficult to detail the clinical usefulness of certain tests.

The purpose of this chapter is to familiarize the reader with the vestibular tests used most commonly in a clinical setting to assess patients with dizziness and imbalance. The chapter describes the subtests that constitute the traditional ENG test battery and discusses the perceived strengths and limitations of each. In addition, the chapter provides an overview of two of the more recent clinical tests, namely, computerized rotation (rotary chair test) and platform posturography (computerized dynamic posturography). Although general interpretation guidelines are provided for each test, the guidelines are not intended to be exhaustive, since the scope of this chapter is to describe the technical aspects of each test. The reader is referred to additional sources for more complete and specific information regarding the diagnostic implications of each test. Four excellent resources are the works of Baloh and Honrubia (1990), Barber and Stockwell (1980), Stockwell (1986, 1990), and Leigh and Zee (1991).

Basis for ENG Recordings

In the early 1800s, Flourens (1830) reported on a functional connection between the vestibular and ocular motor systems. Flourens noted that destruction of the inner ear semicircular canals of pigeons and rabbits created an uncontrollable movement of the eyes, as well as a significant change in the animal's body posture. Two decades later, Du Bois-Reymond (1849) detailed the existence of an electrical potential between the positively charged cornea and the negatively charged retina of the eye. This potential, later referred to

as the corneoretinal potential (CRP), continues to serve as the basis for monitoring eye movements in general and for recording eye movements generated by stimulation of the vestibular system in particular. Although several types of electrodes have been used to detect the CRP, including needles, contact lenses, and coils, surface electrodes taped in place at the outer and inner canthi continue to serve as the method of choice in most clinical settings.

The eye movement recording technique using the CRP is referred to as electrooculography (EOG). The technique was modified in 1939 by Jung for the specific purpose of evaluating vestibular function in patients with dizziness. With the electrodes placed strategically around the periphery of the eyes, Jung was able to use the corneoretinal potential for recording nystagmus and other eye movements created during and following stimulation of the vestibular end-organs. This noninvasive recording technique eventually led to the development of modern clinical electronystagmography.

Electronystagmography

Electronystagmography (ENG) is the diagnostic test used most commonly to assess patients with complaints of dizziness or vertigo. It also is used for the detection and measurement of several eye movement aberrations such as visual fixation and suppression abnormalities, eye movement accuracy, velocity and latency deficits, breakup in the smoothness of visual pursuit, disconjugate eye movement, limitations in the range of ocular motion, and various forms of nystagmus.

The ENG procedure includes a battery of neurologic and otologic tests that have been used by physicians for many years (Coats, 1975). The addition of ENG to these bedside tests provides objective documentation of the examination on a strip-chart recorder and allows for a more precise measurement of the response. The sensitivity and specificity of the various ENG subtests have been enhanced even further by the more recent computer-based ENG systems. In addition, the ENG procedure permits the clinician to record eye movements behind closed eyelids, thus enhancing the eye movement response by eliminating the effects of visual suppression.

The recording technique is rather straightforward. Surface electrodes are taped on the forehead, at the outer and inner canthi for horizontal recordings, and above and below one eye for vertical recordings (Fig. 146-1). The electrodes should be positioned so that a straight line can be drawn through the electrodes to intersect at the center of each pupil. When the eyes move from the center of the socket to the right, the positive pole (cornea) of each eye moves closer to the electrode positioned at the right outer canthus and further from the electrode at the left outer canthus. Since the negatively charged retina also moves in a direction opposite that of the cornea, the difference in electrical activity is detected by the various electrodes. The resultant change in polarity is transmitted through the electrodes, where it is amplified by a recording device, such as a strip-chart recorder. A pen attached to the strip-chart recorder is deflected in a direction that corresponds to the degree and direction of the eye displacement (Fig. 146-2). This procedure allows the recording device to monitor the movement of the eyes at all times (assuming that the eyes move conjugately) whether the eyes are open or closed. When the eyes are positioned in the center of the socket, such as with straight-ahead gaze, the position of the recorder pen remains in the center of the strip-chart paper. When the eyes move either to the right or left, the pen moves to either side

of the center position. Traditionally, for horizontal eye movements, upward pen deflections reflect rightward eye movements and downward pen deflections reflect leftward eye movements. For vertical eye movements the eye deviation and pen deviation are in the same direction.

The clinical usefulness of ENG depends on several factors. Both the otolaryngologist and the operator must recognize the limitations of each ENG subtest in order to understand the test's diagnostic value. For example, the ENG test procedure deals primarily with only one of the major vestibular tracts, namely, the vestibuloocular reflex (VOR). The overall ENG procedure fails to address the vestibulospinal tracts, which play an important role in the maintenance of balance and posture. In addition, stimulation of the vestibular end-organs, either by temperature change (caloric test) or by head/body acceleration (rotary chair test), is limited to the horizontal semicircular canals and superior branch of the vestibular nerves. The remaining semicircular canals (superior and posterior), otolith organs (utricle and saccule), and the inferior branch of the vestibular nerves are not stimulated directly with routine VOR test techniques. As a result, data generated from the various ENG subtests relate to only a portion of the peripheral vestibular system and do not address the vestibular system in its entirety. These limitations, accompanied by considerable variability in test administration and interpretation between clinics, have limited the overall effectiveness and general acceptance of ENG until recently, when more accurate and sensitive computer-based systems were introduced.

Although the information from the ENG battery is important, it must be evaluated in conjunction with the patient's history, symptoms, and other pertinent tests. The purpose of the ENG battery is not to diagnose or identify specific pathologic processes such as Ménière's disease, acoustic neuroma, or multiple sclerosis. Rather, the test is used (1) to detect the presence or absence of organic pathologic conditions within at least a portion of the vestibular system, (2) to monitor change in vestibular function, and (3) to identify a general site of lesion limited to the peripheral or central vestibuloocular pathways, to include the ocular motor system.

The standard ENG test battery consists of a number of subtests. They include (1) the gaze test, (2) the saccade test, (3) the ocular pursuit test, (4) the optokinetic test, (5) the fixation suppression test, (6) the static positional test, (7) the dynamic positioning test, and (8) the bithermal caloric test. Although subtests 1 to 5 primarily evaluate the central vestibular and ocular motor pathways and subtests 6 to 8 attempt to elicit responses from the peripheral vestibular structures, peripheral vestibular lesions can have a significant effect on the results obtained during the "central" ENG subtests and vice versa. As a result, caution must be exercised when identifying results obtained from a specific subtest as being the sole result of a peripheral or central lesion.

Lesions involving the vestibular end-organs *and* the vestibular nerve are classified as "peripheral". Lesions beginning at the vestibular nuclei and proceeding centrally through the brain stem and cortex are classified as "central". For example, an internal auditory canal (IAC) acoustic neuroma would be considered "peripheral" for vestibular test purposes, although it would not be considered peripheral for audiologic purposes. With reference to ENG, test results obtained from a patient with an IAC acoustic neuroma might be no different from those of a patient with Ménière's disease; both are peripheral lesions although the

acoustic neuroma primarily affects the vestibular nerve whereas Ménière's disease has its primary effect in the vestibular end-organs. A cerebellopontine angle tumor would be an example of a "central" vestibular lesion if its primary impact and presence involved the vestibular pathways in the brain stem or cerebellum. More commonly, acoustic neuromas tend to create both peripheral (caloric weakness) and central (gaze nystagmus, poor pursuit, or saccadic dysmetria) vestibular test abnormalities depending primarily on the site and size of the lesion.

ENG subtests

Gaze (fixation) test

The gaze, or visual fixation system, functions to stabilize visual fixation of an object on the fovea during fixed, visual gaze. The fovea is the retinal area of most distinct vision. The purpose of the gaze test is to identify the presence of random or spontaneous eye motion, primarily nystagmus, which interferes with the ability of the eyes to maintain visual fixation. Persons with normal gaze control ability can maintain ocular fixation indefinitely when looking at a fixed target in their visual field. Patients with abnormal gaze stability cannot.

In conducting the gaze test, the patient is asked to fixate visually on stationary targets placed directly in front, 20 or 30 degrees to either side of center gaze, and 20 or 30 degrees above and below the central fixation point. If the patient's gaze fixation ability is compromised, either by an acute, unilateral, peripheral vestibular lesion or by a lesion in the brain stem or cerebellum, a spontaneous eye movement often results. This eye movement usually takes the form of nystagmus, although other ocular movements also may be seen. When nystagmus is detected, its direction always is determined by the fast phase of the nystagmus "beat", (for example, the nystagmus is said to be right beating when the fast phase of the nystagmus is to the patient's right). If a gaze nystagmus is present and its direction changes when the patient changes direction of gaze (for example, right-beating nystagmus with right-lateral gaze, left-beating nystagmus with left-lateral gaze, up-beating nystagmus with upward gaze), the lesion is likely to be isolated in the central nervous system (CNS), usually within the brain stem or cerebellum, and not in the peripheral vestibular system. This type of nystagmus is referred to as a *direction-changing gaze nystagmus* (Fig. 146-3), where the gaze nystagmus "beats" in the same direction as the patient's gaze.

Conversely, if the gaze nystagmus beats in only one horizontal direction (right beating or left beating), irrespective of the patient's direction of gaze, the nystagmus is probably caused by an acute, unilateral peripheral vestibular lesion. This is referred to as a *direction-fixed gaze nystagmus* (Fig. 146-4) and results from weaker neural output from one vestibular system compared with that of the opposite side. The asymmetry between the left and right vestibular systems creates a neural imbalance in the VOR that causes the eyes to be pulled (slow phase of the nystagmus) toward the weaker ear, followed by a rapid saccade (fast phase of the nystagmus) directed toward the stronger ear. As a result, a peripherally based vestibular nystagmus will beat horizontally, away from the weaker ear in most cases, and will not change direction even when the patient changes direction of gaze.

Although one of the strengths of the ENG procedure is to record and document the eye movement pattern without interference of visual suppression, it is imperative that the patient's eyes be visually inspected by the clinician during the gaze test. This is an essential task because nystagmus with a velocity of less than 2 or 3 degrees/sec is difficult to identify on most standard ENG strip-chart records, primarily because of the limited frequency response of the recorder. Also, many commercially available ENG recorders are designed with a limited frequency range to exclude extraneous neural and muscular activity, which could interfere significantly with detection of the EOG response obtained through the relatively small corneoretinal potential.

Even though recognition of low-velocity nystagmus is difficult with an ENG recording, nystagmus with a velocity of considerably less than 1 degree/sec can be visualized by the human eye. If detection of gaze nystagmus is limited to the strip-chart recording alone, without visual verification, low-velocity gaze nystagmus often will not be identified. In addition, true rotary gaze nystagmus (that is, nystagmus that rotates around its horizontal axis without lateral or vertical deviation) will not register on a strip-chart recorder, irrespective of the number of surface electrodes positioned around the eye. The reason rotary nystagmus cannot be identified on the strip-chart recorder is that the positively charged cornea does not move toward or away from any of the electrodes in the array. Without a polarity change on which to rely, the recorder pen will not move, thus preventing detection of the nystagmic activity on the recorder. However, rotary gaze nystagmus can be identified quite easily by close, visual inspection of the clinician.

Gaze-evoked nystagmus generally can be caused by central or peripheral vestibular lesions. Specifying the site of the lesion can be accomplished on the basis of the nystagmus being either (1) direction fixed or direction changing, (2) horizontal or vertical, or (3) enhanced or suppressed with eye closure. In cases of gaze nystagmus caused by lesions within the CNS, the exact site of the lesion is somewhat unclear, although a few generalizations can be considered.

Rotary gaze nystagmus usually is consistent with a brain stem lesion, often involving the vestibular nuclei (Cogan, 1977). It is observed in such disease processes as multiple sclerosis and large, space-occupying lesions that distort the floor of the fourth ventricle wherein the vestibular nuclei are housed. Rotary gaze nystagmus has been reported in cerebellar disease as well (Zee, 1987). It should be noted that in very early stages of an acute, unilateral *peripheral* vestibular lesion, a rotary component to the predominantly horizontal nystagmus may be present. This should be considered whenever a patient is in an acute stage of vertigo secondary to a unilateral peripheral vestibular lesion where a strong spontaneous nystagmus is present.

Vertical nystagmus observed during gaze fixation testing almost always is secondary to a central lesion. One type, down-beating gaze nystagmus, especially when present with lateral gaze, is observed frequently in lesions of the cervicomedullary junction (Barber and Stockwell, 1980). This type of nystagmus has been noted in base-of-brain lesions such as Chiari malformation (Dyste et al, 1989) and basilar impression (Barber and Stockwell, 1980). Down-beating nystagmus has been reported also with lesions in the vestibular nuclei (Cogan, 1977) and flocculus of the cerebellum. When cerebellar lesions cause down-beating nystagmus, the nystagmus is usually present when the eyes are in the primary gaze position,

as opposed to base-of-brain lesions, where the nystagmus is usually most obvious when the patient's eyes are deviated laterally.

Periodic alternating nystagmus (PAN) is a form of gaze nystagmus that usually is present in the primary gaze position (Kestenbaum, 1930). This type of nystagmus changes direction every 2 to 6 minutes within a single gaze position and includes a null period each half cycle. That is, without the patient's changing direction of gaze, the nystagmus beats in one direction (right or left beating) for several minutes, stops, and then beats in the opposite direction. This cycle is repeated indefinitely. PAN often is secondary to cerebellar disease although it can be seen in patients with space-occupying or vascular lesions of the brain stem and midbrain as well. More often than not, however, the exact site of the lesion in patients with isolated PAN is undetected.

Spontaneous ocular square waves observed during the gaze test often are caused by lesions of the brain stem/cerebellum, although this type of eye movement is sometimes characteristic of a tense or nervous patient as well. Fig. 146-5 is a recording from an anxious patient showing continuous side-to-side eye movements during attempts to maintain gaze fixation on a target. It should be noted that certain CNS lesions, specifically those in the brain stem and cerebellum, may show square wave patterns of higher amplitude and frequency than illustrated in Fig. 146-5. Anxious patients tend to exhibit square wave patterns with eyes open and closed, whereas eye closure tends to abolish or reduce the square wave activity in patients with CNS lesions.

When reviewing results obtained during the gaze test, the otolaryngologist should know whether that patient has been taking barbiturates, phenytoin, carbamazepine, or alcohol, because they are known to cause direction-changing gaze nystagmus as well as poor ocular fixation.

During the gaze test the patient's eyes should not be deviated more than 30 degrees from the center gaze position. Extending the eye deviation beyond this point, especially in elderly patients, can result in a "physiologic" end-point nystagmus that does not represent actual vestibular or ocular motor disease. In addition, patients should be tested under a condition of best corrected vision. Eye glasses are preferred over contact lenses since contact lenses can induce slippage resulting in excessive eye blinking, thus creating difficulty when attempting to detect subtle eye movements.

When disconjugate eye movement is present, each eye should be visualized and recorded separately. This can be accomplished by connecting the electrodes of one eye to the horizontal channel of a dual-channel strip-chart recorder and the electrodes of the other eye to the recorder's vertical channel. An additional electrode must be placed at the inner canthus of each eye for this procedure. In some cases (for example, unilateral alternating strabismus), recordings are taken from the dominant eye while the opposite is covered so it does not interfere with the tracking of the dominant eye.

Table 146-1 summarizes the more common gaze abnormalities and the suspected site of the lesion. Please refer to Baloh and Honrubia (1990), Barber and Stockwell (1980), Cogan (1977), Kestenbaum (1930), and Leigh and Zee (1991) for a more complete review of gaze nystagmus and its diagnostic implications.

Table 146-1. Common types of gaze nystagmus and suspected site of lesion

Type	Region of dysfunction
Direction-fixed, horizontal Peripheral vestibular (end-organ or nerve); weakened ear usually is away from fast component of nystagmus (that is, right-beating nystagmus suggests left ear weakness; left-beating suggests right ear weakness); nystagmus enhanced with eye closure	
Direction-changing, horizontal Brain stem; cerebellum (rule out physiologic end-point nystagmus and various CNS medications as noted in text)	
Vertical Upbeating gaze nystagmus suggests lesion in brain stem or cerebellum; down-beating gaze nystagmus suggests lesion in cerebellum or cervicomedullary junction	
Rotary Brain stem (vestibular nuclei); also seen in cerebellar disease	
Periodic alternating Cerebellum, brain stem, or cervicomedullary junction	
Ocular Pendular with center gaze; never vertical; suppresses with convergence; null point; present from early life; exact site of lesion unknown.	

Saccade test

The saccadic eye movement system has at least two distinct functions. The first is to redirect the eyes from one target to another in the shortest possible time. This may be involuntary, as in the case of the fast phase of a nystagmus beat, or voluntary, from the desire to look to either side. The second function is refixation, when saccades correct the retinal position error by bringing the object to the fovea as quickly as possible.

In the saccade test the patient is asked to look between targets (dots on the wall or lights mounted on a light bar) on either side, as well as above and below the center gaze position. The targets are usually spaced 10 degrees from center gaze although this can vary depending on the purpose of the test. The patient looks rapidly between the targets, and the examiner inspects for eye movement accuracy. Inaccurate eye movements, where the eyes either undershoot or overshoot the target, is referred to as ocular dysmetria. Consistent undershoot (hypometric saccades) are abnormal and are seen frequently in patients with cerebellar dysfunction (Leigh and Zee, 1991). In certain instances, hypometric saccades may be seen in normal subjects (Barber and Stockwell, 1980) although a consistent finding of hypometric or hypermetric saccadic eye movement deserves further consideration. In addition to noting saccadic dysmetria on the recording device, hypometric and hypermetric saccadic eye movements also can be detected early by watching the patient's eyes.

Recordings from a saccade test are illustrated in Fig. 146-6. A normal saccadic eye movement test should produce rapid and accurate eye movements that appear on the recorder as a square wave pattern (see Fig. 146-6, A). Consistent overshooting of the target, to the right in this case (Fig. 146-6, B), is seen commonly in patients with brain stem pathologic conditions, most often with concomitant involvement of the cerebellum. Another saccadic abnormality is small catch-up eye movements in both directions in order for the eyes to reach the target. This is referred to as hypometric saccadic eye movement and is seen also in brain stem/cerebellar disease.

The clinician should be alert for disconjugate eye movements. If such a condition exists, the eyes should be recorded individually as described previously.

The saccadic eye movement test has been improved and expanded considerably with the development of computer-driven test systems. In addition to identifying dysmetric eye movements (saccadic accuracy), the computer-based saccade systems are now able to measure saccadic latencies and velocities for eye movements of varying amplitudes (Fig. 146-7). The additional test parameters allow for a more sensitive and accurate measure of brain stem and cerebellar ocular motor function by comparing results to a large age- and sex-matched normative base. Fig. 146-8 illustrates results from a patient with marked velocity slowing bilaterally (16% and 0% normal velocity measurements for saccadic movements to the right and left, respectively) secondary to a brain stem lesion.

Table 146-2 summarizes some of the most common saccade test abnormalities associated with the suspected site of lesion. Please refer to Baloh (1984), Baloh and Honrubia (1990), Coats (1975), Leigh and Zee (1991), Robinson (1964) and Zee (1987) for a more complete review of saccadic eye movements.

Table 146-2. Common saccade test abnormalities and suspected causes for test abnormalities

Abnormality Suspected causes

Ipsilateral dysmetria

Cerebellopontine angle lesions on same side as dysmetria

Bilateral dysmetria

Cerebellum or brain stem lesions

Decreased saccadic velocity

Extraocular muscle weakness, peripheral nerve palsy, lethargic or sedated patient, various progressive neurologic and metabolic diseases

Internuclear ophthalmoplegia

Medial longitudinal fasciculus lesions, such as demyelinating disease.

Ocular pursuit test

Ocular pursuit is one of the more important ocular motor systems. Its function is to stabilize a slowly moving object on the fovea by matching the angular acceleration of the eye with that of the moving object. During the pursuit test the patient is asked to watch a target that moves in a slow, sinusoidal fashion. Fig. 146-9 shows a normal, smooth tracing. As in the case with all ocular motor tests, the examiner should watch the patient's eyes during this test in addition to recording the eye motion, and the patient should be tested under a condition of best corrected vision. Fig. 146-10 illustrates breakup of normal smooth pursuit in a patient with a vascular brain stem lesion.

When a patient's visual pursuit system is impaired, rapid corrective eye movements replace the smooth pursuit movement so the eye can "catch up" with the moving target. When the catch-up movements form a "stair-step" pattern consisting of small saccadic movements, the defect is referred to as *saccadic pursuit*. Saccadic pursuit is seen frequently in patients with cerebellar disease. As is the case with the saccade test described earlier, computer-controlled ocular pursuit measurements have increased the sensitivity and specificity of this test as well. A range of test frequencies within which the pursuit system is capable of operating (0.2 through 0.7 Hz) provides a more comprehensive evaluation of the pursuit system. Fig. 146-11 illustrates normal pursuit test results obtained with one of the many commercially available clinical test systems. Note that all test frequencies are normal from 0.2 through 0.7 Hz in both directions. Fig. 146-12 shows saccadic pursuit in a patient with a cerebellar tumor. Note the stair-step pattern of the eye movement as the patient attempts to pursue a target moving at low (Fig. 146-12, A) and high (Fig. 146-12, B) frequencies.

Tracking irregularities that are secondary to pursuit system abnormalities can usually be seen by monitoring the eyes during the test. The otolaryngologist should screen for pursuit system deficits along with gaze fixation and saccade abnormalities as part of the clinical eye movement evaluation.

The pursuit test, or pendular tracking test as it is also known, can be affected by a variety of conditions, including patient age (elderly patients may have difficulty with this task), mental alertness (drowsy patients tend to lag behind the stimulus), unclear examiner instructions, and various medications (CNS suppressants or stimulants can produce disjointed pursuit).

Visual pursuit abnormalities are usually caused by lesions in the brain stem, cerebellum, or cerebral cortex. However, acute peripheral vestibular lesions that cause a spontaneous nystagmus also may affect the smooth pursuit test unilaterally and must be considered. In this situation a unilateral pursuit tracing abnormality may be secondary to a peripheral lesion when there is no actual deficit within the pursuit per se.

It is important to note that breakup of the pursuit smoothness may result from visual clutter or distraction behind the pursuit target. Therefore a clear, clutter-free background should be present to eliminate any extraneous visual cues. Despite these test artifacts, ocular pursuit provides useful information for detection of ocular motor or central vestibular lesions and is one of the more sensitive tests for CNS dysfunction within the ENG battery.

Table 146-3 illustrates the basic types of abnormal pursuit patterns and the suspected site of lesion. Please refer to Baloh (1984), Baloh and Honrubia (1990), Leigh and Zee (1991), and Robinson (1965) for a more complete review of pursuit findings.

Table 146-3. Pursuit test abnormalities and suspected sites of lesion

Abnormality	Suspected site of lesion
General breakup, no pattern	
	Brain stem or cerebellum; must rule out intense spontaneous nystagmus, poor patient cooperation, and CNS suppressant medication
Saccadic pursuit	
	Cerebellum.

Optokinetic test

The purpose of the optokinetic test (OKN) system is to maintain visual fixation when the head is in motion. The OKN system complements the vestibular system in this regard but functions primarily at frequencies lower than those of the vestibular system. It also functions during more sustained head movements whereas the vestibular system, through the VOR, functions during the quick, short-lived head movements. The OKN test is performed by having the patient fixate visually on horizontal and vertical moving stripes or objects, usually from an OKN drum (Fig. 146-13) or a light bar. This task creates a nystagmus similar to that obtained during head rotation or following caloric irrigation. OKN abnormalities may include an asymmetric nystagmus response, a low amplitude response, or poor nystagmus wave-form morphology.

OKN abnormalities are noted in lesions affecting the ocular motor pathways of the brain stem and cerebral cortex. Although most OKN abnormalities are secondary to slow-phase (pursuit) deficits, they can also be caused by fast-phase (saccade system) pathologic conditions.

Isolated OKN abnormalities are thought to reflect cerebral cortex disease, and when they appear in conjunction with a direction-changing gaze nystagmus, they are thought to represent brain stem or cerebellar dysfunction (Coats, 1975). When the lesion is in the brain stem, an abnormal OKN response is more commonly asymmetric not because of dysfunction within the OKN system, but because of the presence of the gaze nystagmus. Similarly, a spontaneous nystagmus generated from an acute, unilateral peripheral vestibular lesion also can cause an OKN asymmetry that is dominant in the direction of the fast phase of the spontaneous nystagmus. Fig. 146-14 illustrates a normal OKN tracing, whereas Fig. 146-15 shows an OKN asymmetry secondary to an acute, unilateral (left) peripheral vestibular lesion. In Fig. 146-15 the OKN stimulus adds to the right-beating spontaneous nystagmus in one direction and subtracts from it in the opposite direction, thus creating or contributing to the asymmetry. However, this finding is not always consistent, and simple mathematic manipulation of the spontaneous nystagmus velocity is not always helpful.

A problem with OKN testing is the type of stimulus used to elicit the OKN response. Tape-measure OKN strips and the small, hand-held, commercially available OKN drums do not actually evaluate the OKN system. For a true evaluation of the OKN system, all or a majority of the visual field must be filled. If not, the patient will pursue the stimulus relative to a fixed reference point located around the periphery of the target. In that situation the OKN test actually evaluates the ocular pursuit system and not the OKN system.

If small lighted stimuli are used, as in the case of a digital light bar, the room should be darkened so that the only stimulus available in the patient's visual field is the OKN stimulus itself. This method seems to produce stronger and better-formed nystagmus responses when compared to the tape-measure OKN strip or the hand-held OKN drum. Based in part on the stimulus controversy, many clinics have eliminated the OKN test from the ENG battery, when a thorough assessment of the saccade and pursuit systems can be obtained.

For a more thorough review of computerized ocular motor testing, see Stockwell (1988), and for additional information on nystagmus and ocular motor systems and tests in general, the reader is referred to Cogan (1977), Leigh and Zee (1991), Robinson (1964), and Zee (1985).

Static positional test

The purpose of static positional testing is to determine if changes in head position create nystagmus or modify already existing nystagmus. The test is conducted with the patient's head and body placed in several positions and with eyes closed. Eye closure is necessary to eliminate the effects of visual suppression on potential nystagmus. Nystagmus that is present with eyes closed during static head positions (sitting, supine, supine-head right, supine-head left, and supine-head hanging) indicates organic disease although the site of lesion could be central or peripheral. In other words, persistent nystagmus that is present behind closed eyes in any or all head positions is considered "nonlocalizing" with reference to site of lesion. When a nystagmus direction remains unchanged, even when the patient's head position changes, this is referred to as *direction-fixed* positional nystagmus (Fig. 146-16). When the nystagmus direction changes as the patient's head position changes, this is referred to as *direction-changing* positional nystagmus (Fig. 146-17). Both types are nonlocalizing although static positional nystagmus in general is seen more commonly in peripheral vestibular disease than in central vestibular disease.

If a nystagmus is present behind closed eyes only in the sitting position, it is referred to as a spontaneous nystagmus. If the spontaneous nystagmus changes (intensifies or changes direction) when the head is moved into other positions, the patient is said to have a spontaneous *and* positional nystagmus. If the nystagmus is present only in head positions other than sitting, the patient is said to have a positional nystagmus alone.

When a spontaneous nystagmus is present, the direction of the fast phase and the intensity of the slow phase should be noted. This allows the clinician to monitor changes over time. Nystagmus that beats toward the undermost ear is called *geotropic*, and nystagmus that beats toward the uppermost ear is referred to as *ageotropic*. It has been suggested that ageotropic nystagmus is seen more commonly in central vestibular disease, and geotropic is seen more commonly in peripheral vestibular disease. In reality, both types are caused by

peripheral vestibular disease more often than central disease.

True spontaneous or positional nystagmus is persistent and remains present for as long as the patient's head remains within each position. A few intermittent or isolated beats of nystagmus over a 30- or 60-second recording period are more likely an artifact, such as eye blinks, provided the patient is kept alert throughout the test. It is important to keep the patient's head in each position for at least 30 seconds. If a positional nystagmus is present, it should be noticeable over the entire 30-second period.

If a head-lateral position (supine-head right or supine-head left) causes a nystagmus, the patient's entire body should be turned to the side without twisting the neck. This is to rule out neck involvement (so-called cervical vertigo), which, although rare, should not be overlooked.

The use of vertical electrodes might be considered during static position tests although vertical nystagmus behind closed eyes has little apparent localizing value. On occasion, this phenomenon has appeared as an initial test finding in early multiple sclerosis and other CNS pathologic conditions, although most of the time a specific diagnosis is not made when this is the only objective test abnormality. Vertical electrodes used for the detection of eye blinks are helpful because blinks often resemble nystagmus beats on the horizontal channel of the strip-chart recorder, particularly if the recorder is set in the AC mode.

In all positional testing, as well as during caloric irrigation, the patient must be kept alert throughout. If not, absent or poorly formed nystagmic responses may be recorded, making accurate measurement of the response difficult. The patient's level of alertness should be controlled as well. Mental tasks that are too difficult may be as ineffective as no mental alerting at all. Overly difficult tasks may cause facial tension and eye blinking, thereby disrupting the nystagmus waveform smoothness and creating measurement errors.

Low-velocity spontaneous or positional nystagmus has been reported in a significant percentage of the "normal" population (Barber and Stockwell, 1980; Coats, 1975). As a result, some clinics choose not to report spontaneous or positional nystagmus with slow-phase velocities of less than 6 or 7 degrees/sec. Although low-velocity nystagmus might be clinically insignificant in some patients, nystagmus should not be present if the patient's vestibular systems are truly normal. The presence of low-velocity spontaneous or positional nystagmus provides objective evidence that an asymmetry or other organic vestibular system abnormality is present. When this occurs, we suggest that the nystagmus direction and velocity be recorded and that the test be repeated at a later date to determine the stability of the nystagmus.

Dynamic positioning test

The most common type of true vertigo in adult patients is probably benign paroxysmal positional vertigo (BPPV). After the history, the test used to diagnose this condition is a dynamic positioning maneuver, the Dix-Hallpike test. This test should always be performed as part of the ENG test battery, especially when patients complain of motion-related vertigo. Although head position is important, this subtest is considered to be a *positioning* test rather than a *positional* test. The final position of the head is important; however, it is the maneuver

that places the patient's head into the position that creates the response.

The phenomenon of BPPV was first described by Barany (1921) and popularized years later by Dix and Hallpike (1952). These investigators discovered that by moving certain patients rapidly from a sitting to a head-hanging lateral position (Fig. 146-18), they observed a burst of nystagmus accompanied by vertigo following a brief latency period after the final head position was reached. Repeat positioning maneuvers lessened the response until there was no response after several repetitions. When the investigators noted a positive response, the patient was said to have benign paroxysmal vertigo provided certain criteria were met.

The criteria used today for a "classic" Dix-Hallpike response include (1) subjective vertigo, (2) a transient nystagmus burst, (3) a gradual lessening in the severity of the nystagmus and vertigo when the maneuver was repeated (fatigue), and (4) the response begins following a latency period of 1 to 10 seconds once the head reaches the final position. If the response is determined to be classic, a benign peripheral vestibular lesion (BPPV) in the undermost ear is suspected. If the response is "nonclassic" (one or more of the above criteria are absent), the lesion is nonspecific as to site. The lesion can be either peripheral or central in this case. Although the pathophysiology remains in question, one common theory suggests that loose otoconia become dislodged and fall from the utricle into the ampulla of the posterior semicircular canal. This action creates a mechanical stimulation of the posterior semicircular canal cupula when the patient performs a specific head movement such as rolling over in bed, looking upward, or bending down. As noted above, this vertigo is arguably the most common type observed in patients from 40 to 70 years old.

The hallmark of a classic Hallpike response is a rotary nystagmus without horizontal or vertical components. Keeping in mind the fact that a true rotary nystagmus cannot be detected on a strip-chart recorder, even with electrodes surrounding the eyes, the clinician should visually monitor the patient's eyes during the Dix-Hallpike maneuver (Cyr and Brookhouser, 1984). The optimal method is to observe the eyes behind lighted, 20 diopter Frenzel's lenses (Fig. 146-19). This eliminates any possible although unlikely effects of visual suppression of the response. If Frenzel lenses are not available, the next favorable option is to perform the test with the patient's eyes open and fixed. Even using this latter procedure, a positive response will almost always occur with little or no effect of visual suppression, if the patient indeed has BPPV. The least favorable method is to perform the test with the patient's eyes closed while relying on the recorder device to determine if a nystagmic response is present, since this method will fail to identify those patients with pure rotary nystagmus. Conversely, if the nystagmic response consists of a horizontal, vertical, or torsional component, the abnormality can be recorded behind closed eyes (Fig. 146-20). A burst of nystagmus in the reverse direction may occur when the patient is moved back to the sitting position although the nystagmus burst may be somewhat less intense.

When performing the Dix-Hallpike maneuver, the patient's head should be turned to the side before movement into the down-left or down-right position. This modification eliminates the several beats of righting nystagmus that are seen even in normal patients when the head is being turned and guards against false-positive Hallpike responses caused by the righting nystagmus. This modification places less strain on the patient's neck as well. In most cases, BPPV is usually present in only one head-hanging direction although bilateral responses are noted periodically.

Before initiating the Hallpike maneuver, it is suggested that the patient be warned about the brief sensation of vertigo that may appear in a positive response. Motion-induced vertigo can be frightening to many patients, and better patient cooperation might be achieved if the patient is not surprised by the maneuver-induced symptoms. Reassuring the patient that the response will dissipate quickly often helps to alleviate strong patient reaction as well. The speed with which the maneuver is conducted appears to be only mildly important. A moderate speed of positioning should be sufficient, especially in elderly patients and those with neck or spine problems.

Table 146-4 summarizes static and dynamic positional test results and provides an indication of the suspected site of lesion. Please refer to Baloh (1984), Barber and Stockwell (1980) and Coats (1975) for a more complete review of the diagnostic significance of both static and dynamic positional tests.

Table 146-4. Summary of positional test (static and dynamic) results and suspected site of lesion

Type of abnormality	Suspected site of lesion
Direction-fixed, static, positional nystagmus Nonlocalizing although more commonly peripheral vestibular	
Ageotropic, static, positional nystagmus Nonlocalizing	
Geotropic, static, positional nystagmus Nonlocalizing, although more commonly peripheral vestibular; rule out use of alcohol within past 48 hours	
Dix-Hallpike (classic response) Peripheral vestibular in undermost ear	
Dix-Hallpike (nonclassic response) Nonlocalizing.	

Caloric test

The bithermal caloric test is the oldest and most specific test within the ENG battery. The caloric test is a nonphysiologic procedure used to induce endolymph flow in the semicircular canals (primarily the horizontal canal) by creating a temperature gradient from the lateral to the medial part of the canal. The standard procedure stimulates only one labyrinth at a time, and the test results can be compared only within the same patient (one ear compared to the other). Several types of caloric stimulation are used clinically (Fig. 146-21), including water (the most common), air, and closed loop (water within a closed silicon balloon).

The test consists of having the patient lie on a table with the head anteroflexed 30 degrees (Fig. 146-22). This position places the horizontal semicircular canals in a vertical plane. A thermal stimulus, usually water, flows into the external ear canal. Water temperatures of 30°C and 44°C are used with approximately 250 mL delivered over a 30-second period. When cold water is used, the temperature is transmitted to the wall of the horizontal canal, cooling the endolymph and causing the fluid to become more dense as it falls in the vertically positioned canal. This sets up a fluid motion in the canal that deflects the cupula away from the utricle. The deflected cupula causes the resting neural discharge to drop relative to the opposite ear, creating a neural imbalance and resulting in a nystagmus with the slow phase toward the side of irrigation and the fast phase away from the irrigated side. The opposite occurs when warm water is used, causing the nystagmus to beat toward rather than away from the irrigated ear. As described in the section on positional tests, controlled patient alertness must be maintained throughout the caloric test. Vestibular and CNS suppressants should be discontinued at least 24 hours, and preferably 48 hours, before the test, depending on the half-life of the medication in question.

The period of maximum nystagmus activity is identified (usually occurring approximately 60 to 90 seconds after the irrigation begins) and measured. The right ear warm and right ear cool responses are summed and compared to the left ear warm plus left ear cool responses to determine if a unilateral weakness (UW) exists. Right-beating (right warm + left cool) responses are compared to left-beating (left warm + right cool) responses to determine if a directional preponderance (DP) is present. Interear differences of 20% and greater are considered abnormal for UW, and differences of 30% or greater are considered abnormal for DP. Please refer to Coats (1975), Stockwell (1986), and Teter (1983) for detailed instructions on measuring the slow-phase velocity of the caloric-induced nystagmus.

Although water caloric delivery systems today are improved over systems 20 years ago, the caloric test remains highly variable between and within patients for several reasons. The rather large normal range for UW (20% or greater) and DP (30% or greater) may be the result of several factors, including small but significant stimulus temperature and duration changes, mental alerting procedures, size and shape of the external ear canal, aeration pattern of the mastoid, type of stimulus (water, air, closed loop), and operator competence. As a result, the variability of the caloric test makes it difficult to monitor subtle changes in vestibular function.

Caloric abnormalities (unilateral or bilateral weakness) usually reflect peripheral lesions. In a small number of patients, a caloric abnormality may also reflect a brain stem lesion, although this is rare. DP is considered a nonspecific finding. In fact, most cases of DP simply represent either a spontaneous nystagmus or a positional nystagmus that is present in the caloric head position. Fig. 146-23 is an example of a normal and symmetric caloric response (2% left weakness only) with no significance DP (4%). Fig. 146-24 illustrates results from a patient with a right peripheral vestibular weakness and a right-beating nystagmus that is present in the caloric head position (supine with 30 degree anterior neck flexion). The caloric results show a 66% right weakness and a 29% right-beating DP.

Failure of fixation suppression. During the caloric test, normal patients and those with peripheral vestibular disease should be able to suppress caloric-induced nystagmus by opening the eyes and fixating visually on a target. Patients with CNS pathologic conditions show little difference in the velocity of caloric-induced nystagmus whether their eyes are open or closed.

After the peak portion of the caloric-induced nystagmus is reached, the patient is instructed to open the eyes and fixate on a target. Fig. 146-25 illustrates results from a patient with normal fixation suppression of a caloric-induced nystagmus. Fig. 146-26 is a recording from a patient with CNS disease. Fig. 146-26, A, shows an eye-closed nystagmic velocity of approximately 17 degrees/sec. After the patient's eyes are opened and fixed (Fig. 146-26, B), the nystagmic amplitude drops significantly although the slow-phase nystagmic velocity remains at approximately 12 degrees/sec. A reduction in the nystagmus velocity of at least 40% should be achieved when the patient's eyes are open and fixed before fixation suppression is considered normal. Failure of fixation suppression (FFS) is a strong indication of CNS disease, usually within the cerebellum. Patients with FFS frequently have pursuit system deficits as well, most often showing saccadic pursuit. The clinician must ensure that the patient has sufficient visual acuity to clearly visualize the fixation target, so that each patient should be tested under best corrected vision with sufficient light available when the fixation target appears.

Table 146-5 summarizes the caloric and FFS test results and the suspected site of lesion for each type of abnormality. This table and Tables 146-1 and 146-4 are meant to be general guidelines only since exceptions may be noted for each category. Please refer to Baloh (1984), Barber and Stockwell (1980), Coats (1975), and Stockwell (1986) for a more complete review of caloric test results and various modifications to the standard test, including the Kobrak, monothermal, and simultaneous binaural bithermal tests.

Table 146-5. Summary of caloric test and fixation suppression test findings and suspected region causing test abnormalities

Type of abnormality	Suspected site of lesion
Unilateral or bilateral weakness Almost always peripheral vestibular disease; rule out vestibular suppressants in cases of bilateral vestibular weakness	
Directional preponderance Nonlocalizing; usually secondary to a positional nystagmus when nystagmus is present in caloric head position	
Failure of fixation suppression (FFS) Cerebellum; ensure that patient has sufficient visual acuity to allow fixation on target.	

Rotational Tests

In addition to stimulation of the vestibular system by temperature change within the inner ear fluid (caloric test), a similar nystagmic response can be elicited by rotation of the head. In 1907 Barany described a manually driven rotary chair that produced observable nystagmic eye movements. Barany turned the patient, seated in the chair, from side to side and observed the patient's eye movements after the rotation stopped (post-rotary nystagmus). Numerous problems were noted during the early development of the test, including difficulty controlling the manually driven stimulus and the inconsistent effects of visual suppression and enhancement whenever post-rotation nystagmus was assessed in light with open eyes. Both problems contributed to excessively high variability between and within subjects.

The clinical use of the rotation test fell from grace somewhat because of its inability to produce repeatable results based on the stimulus/response problems (Brown et al, 1983) and also because of the emergence of the ENG/caloric procedure. Renewed interest appeared in the 1970s (Mathog, 1972; Reder et al, 1977; Wolfe et al, 1978a, 1978b) based in part on the emergence of microprocessors and improved hardware. The torque-driven motors used to control the rotary chair improved dramatically as did the computer software that controlled the stimulus and measured the per-rotary nystagmus, a more sensitive parameter than the post-rotary nystagmus used earlier. Because the acceleration and deceleration of a chair could be controlled more accurately, the subsequent compensatory eye movements could be measured more effectively. In addition, the strong accurate motors were able to compensate for weight differences between patients, a problem with earlier spring-driven swing chairs.

The rotational chair system has several advantages over the standard caloric test:

1. The rotational stimulus is less bothersome to the patient because it does not create the vertigo and nausea often associated with the caloric test.
2. The mechanical artifacts associated with delivering the caloric stimulus to the inner ear (size and shape of the external ear canal, scarring, thickness, and cross-sectional area of the tympanic membrane; aeration of the mastoid; thermal transmission through bone and soft tissue; small but significant temperature changes in the caloric stimulus; and operator experience and skill) are not factors with the rotation stimulus. As a result, a more accurate stimulus response relationship is possible.
3. The rotational stimulus is more natural than caloric irrigation because it attempts to simulate natural environmental motion. In addition, the nystagmus can be measured at various acceleration levels, enabling the vestibular system to be tested over a wider portion of its operating range.
4. Multiple gradations of the stimulus can be presented in a short time period.
5. Because the rotational stimulus is controlled more accurately and the response measured irrespective of the external ear canal and middle ear geometry, small changes within the VOR can be monitored more effectively.

The primary disadvantage of the rotary chair test is that it stimulates both labyrinths (horizontal semicircular canals and superior vestibular nerves) simultaneously. To determine the relative strength of the right and left horizontal semicircular canals, respectively, a caloric test must be performed.

The low frequency rotary chair test is the most commonly used rotational test in the USA. It is performed with the patient seated in a computer-driven chair that rotates around the earth's vertical axis (Fig. 146-27). The chair is turned by a torque motor capable of accurate sinusoidal acceleration and deceleration. The patient's head is tilted 30 degrees forward so that rotation occurs in the plane of both horizontal semicircular canals. The chair is situated in an enclosure that becomes completely dark when the door is closed. Standard electrooculographic (EOG) recordings are made of the compensatory nystagmus eye movements during rotation at a range of acceleration frequencies. Since the semicircular canal systems act as angular accelerometers, a range of acceleration frequencies is used to evaluate vestibular output over a wider portion of its operating range. An infrared camera mounted to the chair allows monitoring of eye movement, head position, and patient status during the test.

As the chair and patient begin to rotate, a slow, compensatory eye movement is observed in the direction opposite the rotation. The saccadic (fast) eye movement used to return the eye to the central position is eliminated via a fast Fourier transform. The slow-phase compensatory eye movement velocity is then averaged over all test cycles and compared to the average velocity of the rotating chair for rightward and leftward chair oscillations.

Three test parameters (phase, gain, and symmetry) are considered on most commercially available rotary chair systems. *Phase* of the rotation-induced compensatory eye movement is the temporal relationship between the velocity of the head (chair) and that of the slow-phase component of the rotation-induced nystagmus. At high frequencies of head movement, the eyes move in a precise compensatory manner, although 180 degrees out of phase. At lower acceleration frequencies, the eye movement velocity leads or lags in reference to the chair velocity.

Phase leads are inversely related to rotational frequency. As the acceleration frequency decreases, the phase lead increases. A low-frequency phase lead is exaggerated in many patients with central or peripheral vestibular pathologic conditions, and this phase lead will likely remain, even following compensation of unilateral vestibular lesions (Baloh et al, 1979; Hirsch, 1986). Phase is probably the most stable and repeatable parameter measured during the low-frequency rotary chair test provided gain is adequate (see below), although its clinical significance is still somewhat obscure.

With rotation to the right, a leftward compensatory eye movement occurs. A rightward eye movement occurs when the chair turns to the left. The *symmetry* parameter of the rotary chair test reflects the peak slow-phase eye velocity when the patient turns to the right, versus the peak slow-phase velocity when the patient turns to the left. This parameter is usually expressed as a percentage although it may be expressed in degrees.

It must be stressed that the symmetry measure is not a definite indication of laterality (side of the lesion); rather, it is similar to directional preponderance on the caloric test. In both cases the cause is often secondary to a spontaneous or positional nystagmus and, as a result, is nonlocalizing. In acute, unilateral peripheral vestibular disease, the fast phase of the spontaneous nystagmus usually beats away from the weaker ear. In this situation the rotary chair symmetry will indicate accurately a weakened response on the impaired side. In unilateral peripheral vestibular lesions where the spontaneous nystagmus beats toward the side of the disease (so-called irritative lesions), the rotary chair symmetry measure mistakenly will indicate that the normal ear is the involved side. Lesions such as early Ménière's disease, serous labyrinthitis, labyrinthine fistulas, and occasionally, acoustic neuromas can cause irritative spontaneous nystagmus.

Because the symmetry parameter seems to reflect central compensation to some degree, it is often normal in compensated, unilateral peripheral vestibular lesions. This observation is based on clinical evidence that shows the degree of asymmetry decreases as central compensation occurs. The improvement in symmetry in these cases likely reflects a decrease in the intensity of the spontaneous nystagmus, resulting in a decreased directional preponderance, another possible indication of central physiologic compensation following unilateral peripheral vestibular disease.

The *gain* parameter of the rotary chair test is the amplitude of maximum slow-phase velocity of the nystagmus compared with the amplitude of the maximum velocity of the rotating chair. In other words, the gain response is a ratio between the maximum eye velocity and the maximum chair velocity. At "natural" head movement frequencies (1 to 5 Hz), a gain of "1" indicates that the compensatory slow-phase eye movement velocity is equal and opposite the chair/head movement velocity. At lower chair/head velocities, the compensatory eye movement gain is generally less than the chair/head movement velocity. In other words, slow-phase eye movement velocity (in darkness) decreases when the head velocity decreases.

Because gain is a performance parameter, abnormally low gain may indicate a pathologic condition. Of all the parameters measured during the low-frequency rotary chair test, however, gain appears to be the most unstable since it is related closely to the patient's state of mental alertness. Because adequate gain is needed for the analysis algorithm used to calculate phase and symmetry, every effort must be made to keep the patient mentally alert.

In patients with acute, unilateral peripheral vestibular lesions, gain is likely to be depressed. This finding has been noted following disease and labyrinthectomy (Wolfe and Kos, 1977). Following central compensation, gain appears to recover somewhat, provided appropriate mental alertness is maintained.

In bilateral peripheral vestibular lesions secondary to ototoxicity, gain decreases as the drug-induced damage increases, although changes in phase may be a more sensitive parameter than gain for monitoring purposes (Cyr et al, 1989). See text related to Figs. 146-30 to 146-32.

Baloh et al (1979) noted excessively high gain associated with cerebellar atrophy. Since the flocculonodular lobe of the cerebellum is involved in the visual suppression of vestibular nystagmus, it has been suggested that the high gain may be secondary to decreased cerebellar inhibition, possibly involving the vestibular nuclei. In these patients, slow-phase nystagmus velocity from caloric stimulation may be normal or even depressed. Fig. 146-28 illustrates excessively high gain in a patient with cerebellar degeneration and a long-standing history of severe motion sensitivity. Phase and symmetry measures are normal.

Fig. 146-29 shows the graphed results of the phase/gain symmetry pattern from a normal patient. Responses are plotted for rotation frequencies ranging from 0.01 to 0.64 Hz. The shaded area represents the 95th percentile. Phase, gain, and symmetry are all normal.

Clinical utility

Attempts have been made to identify the location of vestibular lesions based on patterns of phase, symmetry, and gain relationships. Although certain trends may appear, low-frequency phase leads, asymmetries, and depressed gain can be seen both in peripheral and central vestibular lesions (Baloh et al, 1982). As a result, it is difficult to determine the exact site of lesion on a consistent basis from the parameter pattern generated during the low-frequency rotary chair test. However, a series of patterns have been reported that appear to fairly accurately represent specific types of lesions. Those patterns are illustrated in Figs. 146-30 to 146-35.

The primary strength of the computerized rotary chair test appears to be its sensitivity to monitor change within the vestibular system, particularly in the early detection of bilateral peripheral vestibular disease (Cyr et al, 1989). Fig. 146-30 illustrates test results from a patient with profound loss of vestibular function bilaterally secondary to meningitis. Phase and symmetry measures are invalid in this case because there are no slow-phase velocity measurements identified because of the absence of nystagmic activity. Figs. 146-31 and 146-32 show test results from two patients treated with gentamicin. Fig. 146-31 shows a gradual decrease in gain over a 2-week period, which is a typical test finding associated with patients treated with gentamicin who exhibit toxicity. Fig. 146-32 shows an increased phase lead abnormality before any significant decrease in gain as the earliest indication of damage. As noted earlier, increased phase lead abnormalities may occur before gain changes in many patients exhibiting early vestibulo-toxic signs. Consequently, the monitoring procedure used with aminoglycoside patients should include evaluation of both gain and phase parameters, remembering to keep the patient as alert as possible so as to not reduce gain artificially.

Although disagreement continues about the low-frequency rotary chair test's ability to monitor physiologic compensation following a sudden, unilateral vestibular insult, some points are worth reviewing. For example, in an acute, unilateral vestibular weakness, an asymmetry will usually be present in the direction of the spontaneous nystagmus. In addition, gain may be depressed (primarily in the low frequencies) and a large phase lead should be seen (Fig. 146-33). As central compensation occurs, the degree of asymmetry will usually decrease or resolve completely, and gain may improve to a degree as well. In contrast, the low-frequency phase lead appears to persist (Fig. 146-34) in most but not all patients (Hirsch, 1986; Jenkins et al, 1982). Recall that abnormalities in the symmetry parameter (Fig. 146-35) are affected by spontaneous nystagmus and the gain parameter can be affected by patient alertness and

other extravestibular factors during low-frequency acceleration (Larsby et al, 1984). For these reasons the clinical significance of gain and symmetry (as well as phase) during low-frequency rotation remains somewhat unclear.

Another effective application of the rotary chair test is its use with special populations, especially infants and young children in whom caloric testing cannot be completed effectively (Cyr et al, 1985). The test is easy to administer and takes a minimal amount of time, and the gentle rocking motion is usually enjoyable to infants and young children.

Even though the low-frequency rotary chair test has specific limitations (for example, inability to consistently identify site of lesion or side of involvement), it has demonstrated a significant clinical usefulness. Stockwell (1990) reported a 15% improvement in the detection of vestibular abnormalities using the rotary chair when compared to ENG.

It must be stressed, however, that the rotary chair results should be treated with a degree of caution. Our knowledge of the visual/vestibular system is limited to date, and the low-frequency rotary chair test monitors only a fraction of the VOR. We must take into account other factors such as the otolith organs (that may influence the response), the remaining semicircular canals, the brain stem integration of the response, and the cognitive processes, such as prediction, that may also affect the test results. As is appropriate with all clinical assessments, the rotary chair results, as well as those obtained with the standard ENG, should always be interpreted in conjunction with patient history, symptoms, and other test findings.

Posturography

As noted earlier in this chapter, balance and equilibrium constitute a complex reflexive response initiated by three primary sensory systems (vestibular, visual, and somatosensation) and coordinated by the central nervous system. Up to this point, we have discussed objective measures of vestibuloocular and ocular motor function alone. These VOR tests permit us to examine only a portion of the mechanism used for the maintenance of posture and both static (standing) and dynamic (walking) balance. In addition, although the VOR tests provide the clinician with diagnostic information related to the physiologic status of the vestibuloocular system, they do not provide a complete indication of the functional impact a vestibular lesion may have on a patient's sense of equilibrium. That is, one patient with a profound, bilateral peripheral vestibular weakness may have much greater difficulty ambulating when compared to a second patient with a similar condition. Circumstances such as age, visual acuity, overall strength, interactive or suppressive medications, and status of the patient's somatosensory system represent but a few of the reasons for this difference. It would seem logical to conclude that a functional measure of a patient's balance is important if the otolaryngologist is to obtain knowledge into the effects of various vestibular system lesions, and, at the same time, to provide the vestibular- or balance-disordered patient the most effective care and treatment possible.

Although many of the patients referred for "dizziness" have straightforward vestibular disease, others have multisystem lesions. Standard vestibular tests such as electronystagmography (ENG) and computerized rotation may be insufficient for these patients since these tests deal almost exclusively with the vestibuloocular reflex (VOR). The

effects of vision and proprioception are not effectively considered with VOR tests.

Over the years, several attempts have been made to evaluate the somatosensory and visual influences on posture and equilibrium, commonly using some type of force platform that measures body sway with and without visual awareness (often referred to as static posturography or stabilometry). Because the effects of sensory input referable to balance usually are measured through a motor reaction, the patient's coordinated muscle response also must be considered. That is, the brain must not only receive and integrate the input from the sensory systems subserving balance, but also generate an appropriate motor response. Therefore both sensory and motor components of balance should be evaluated when assessing postural stability. Normal motor responses imply normal function of both sensory and motor arcs, but abnormal motor responses can reflect abnormalities in the sensory or motor arcs or both.

Recently computerized force vector analysis has been added to improve the precision of platform posturography. Nashner (1978, 1987) has carried this evaluation a step further. This system (Equitest), known generically as computerized dynamic posturography (CDP), attempts to ferret out the effects of various sensory inputs to the brain and relate them to overall on-feet balance and stability.

CDP (Fig. 146-36) employs a computer-controlled, menu-driven moveable platform and a moveable background that surrounds the patient almost completely and fills the patient's visual field. The background, attached to a moveable surround, contains colored patterns simulating an abstract landscape and provides the surface for visual orientation and fixation. Both the platform and background are "sway referenced". That is, the platform and background move to track the anteroposterior (AP) sway of the patient, who is asked to stand on the platform surface with feet shoulder width apart. The patient's body sway is monitored by pressure-sensitive gauges located in each quadrant of the platform. As the patient sways around his or her center of balance, the platform and background track the patient's motion, providing an objective measure of actual AP sway. The surface platform is able to operate independently of the background. The test is conducted with eyes open or closed, which eliminates all visual cues, and because of sway referencing, can provide conditions of visual distortion and somatosensory distortion. In addition, the platform can be jerked suddenly front-to-back and back-to-front to measure the patient's motor response to sudden loss of balance. The platform also performs a "toes-up" and "toes-down" movement. These latter computer-induced platform movements evaluate motor responses that include the strength (force), symmetry, and latency of muscle response. In addition, movement adaptation is evaluated with repeat platform rotations. A safety harness is attached to the patient to prevent injury should a fall occur.

The standard, clinical CDP test battery includes an assessment of automatic postural responses to platform perturbations, the so-called motor control (MC) test, and the sensory organization (SO) test. The sensory organization portion of the test battery manipulates visual and proprioceptive inputs while determining the effects on a patient's standing balance.

Motor control test

As noted above, the motor control portion of the test is concerned with the measurement of automatic postural responses. During this part of the test, the platform performs sudden front-to-back and back-to-front perturbations. Three small, medium, and large forward and backward perturbations are made, followed by a series of five "toes-up" and five "toes-down" platform rotations. The reaction time and the amount of force a patient applies to the platform surface in response to the platform movement are detected by the strain gauges and then measured to determine the following factors:

1. *Weight symmetry*: Is the patient's lateral weight distribution during the platform perturbations equally distributed over both feet, or is the weight shifted to one side more than the other?

2. *Amplitude scaling*: Does the patient apply equal force through both legs during the platform movement, and is the amount of force applied commensurate with the size of the platform perturbation?

3. *Latency*: Is the patient able to apply active force to the platform within a short period of time following the platform perturbation, and does the response time within both legs decrease as the size of the platform perturbation increases?

4. *Adaptation*: Does the patient gradually apply less force to the platform with each platform rotation?

Fig. 146-37 illustrates a normal motor control test showing normal weight and strength symmetry, normal long motor loop latencies, and normal amplitude scaling. Fig. 146-38 illustrates results from a patient with abnormal weight and strength symmetry as well as poor adaptation. This pattern can be observed in patients with unilateral neurologic or orthopedic abnormalities. Fig. 146-39 illustrates prolonged latencies through the long motor arc bilaterally. This patient was found to have diabetic neuropathy with numbness in his feet and lower legs. The information obtained from this portion of the test battery is used to examine the efficiency of the long motor arc in patients with proprioceptive system disease, as well as weight or strength asymmetries that may cause postural alignment problems in patients with certain orthopedic or neurologic diseases, both of which would tend to cause imbalance when standing or walking.

Sensory organization

Six 20-second subtests comprise the SO portion of the test battery. Those six conditions are shown in Fig. 146-40. Under the first three test conditions, the platform remains fixed. This situation provides the patient with a stable reference for somatosensory information. In condition 1 the visual surround also remains fixed so that the patient has a stable visual reference as well. A majority of patients should be able to complete this test condition with only minimal difficulty. In condition 2 the patient's eyes are closed, eliminating all visual cues to the brain so that the amount of the patient's AP sway is determined by vestibular and somatosensory information. In this test condition, standing balance is controlled primarily by the somatosensory system. Therefore, even though the visual input is eliminated

by eye closure, patients with absent or distorted vestibular function show only minimal unsteadiness on this condition, provided somatosensory function is normal. In contrast, patients with normal vestibular function but abnormal somatosensory function tend to sway considerably on this test condition. It appears that most patients use somatosensory input for standing balance more than vestibular input, particularly when standing with feet shoulder width apart. In condition 3 the visual surround moves to track the AP sway of the patient. This technique is referred to as "sway referencing" of the visual surround. Under this test condition the visual input to the brain is distorted. The brain is forced to suppress the orientationally inaccurate visual input and select vestibular and somatosensory cues for balance. Patients with absent or distorted vestibular input have little difficulty with this test condition although patients with poor somatosensation do poorly.

In conditions 4, 5, and 6, the platform moves to follow AP sway of the patient (sway referencing of the platform), thus distorting somatosensory input to the brain. The brain must suppress the inaccurate surface cues in this case and select vestibular and visual input to maintain a stable posture. In condition 4 the visual surround remains fixed so that the patient's balance is maintained by visual and vestibular inputs. Patients with vestibular disease show only minimal sway on this condition, provided they have intact visual systems. During condition 5 the patient is asked to close his or her eyes, thus eliminating all visual cues. Since the platform is free moving (sway reference) as well, on-feet balance must be maintained by vestibular cues alone. Patients with bilateral or uncompensated unilateral vestibular disease sway excessively on this condition. In condition 6, both the platform and the visual surround are sway referenced to track the AP sway of the patient. The brain must now suppress both orientationally inaccurate visual and somatosensory cues and select vestibular input in order to remain standing. Once again, if vestibular function is abnormal, patient sway is excessive in this test condition. Perfect stability (no sway) is given a score of "100" whereas maximum sway or a patient fall is given a score of zero (0).

In addition to the normal pattern (Fig. 146-41), several abnormal patterns have been identified and they are illustrated in Fig. 146-42 to 146-47.

In addition to obtaining equilibrium scores, balance strategies are measured. This information is used by the physical therapist to teach the patient appropriate angle, hip, or upper body movements in order to maintain static and dynamic balance and posture. Under normal conditions, most individuals move about the ankle joint as a fixed point. This is referred to ankle strategy, and it is the strategy of choice provided the degree of body sway does not exceed certain limits. When body sway is increased beyond what the ankle movement can handle or if the frequency of the body sway increases, the individual must incorporate corrective movements by bending at the hip or using upper body and arm movements. This strategy would be normal for a large amount of body sway; however, it is usually considered inappropriate for the degree of body sway induced by the six SO conditions.

Clinical uses of computerized dynamic posturography

CDP is suggested for (1) patients with histories of imbalance and on-feet unsteadiness, as opposed to patients with rather straightforward vertigo for which ENG would likely be given first thought; (2) children with delayed motor development or "clumsiness" in order to

evaluate body alignment and disorganization of sensory inputs to the brain; (3) neurologically impaired patients to evaluate weight and strength symmetry, long motor loop reaction time between left and right legs, and adaptation ability; (4) patients with suspected organic vestibular system disease where the standard VOR tests are noncontributory (Cyr et al, 1988); and (5) patients who need a determination of the functional impact a lesion (or feigned lesion in the case of a malingerer) has on a patient's standing balance and posture. The test results can assist in planning remediation strategies for balance-disordered patients, and the effects of various therapies (medical, surgical, and physical) can be monitored in patients with known or suspected vestibular disease by repeating the test at specific intervals.

The primary value of this test appears to lie in its ability to describe the functional impact various lesions have on static and dynamic balance. Although certain patterns of dysfunction have been noted and are illustrated in the preceding figures, this test does not yet possess the diagnostic usefulness of the ENG battery, at least as far as vestibular lesions are concerned. This is not meant to criticize the test procedure. In fact, we believe CDP can add significantly to the evaluation of patients with dizziness and various forms of dysequilibrium and imbalance. The test is easy to administer, requires very little time (approximately 15 to 20 minutes), and does not create discomfort. The test results appear to be repeatable, when conducted properly, and improvement in on-feet stability can be monitored over time with considerable reliability. Because of the repeatability of testing, the effect of physical therapy and improvement noted following medical or surgical intervention can be quantified. On the negative side, the dynamic posturography equipment is expensive, and at this time, reimbursement by third parties is poor, which may restrict placement of CDP to larger clinical settings.

Summary

This chapter has attempted to familiarize the practicing otolaryngologist with three of the most common test methods used to evaluate dizziness and balance-disordered patients. The purpose of these tests (ENG, rotary chair, and dynamic posturography) is not to diagnose per se and certainly not to label patients with specific diagnoses. Rather, the tests are used to identify organic causes for the patient's symptoms in many cases and, in the case of posturography, to evaluate the functional impact of the lesion on standing balance. In doing so, various patterns and test findings can provide the otolaryngologist with information related to which sensory or motor system may be involved and information as to the general site of lesion within that sensory system or motor system. The tests are not meant to replace a careful review of patient history and symptoms. They are meant to augment the medical evaluation. When that is done, the otolaryngologist should find that these clinical tests provide useful information when evaluating many patients with a variety of complaints related to dizziness or imbalance. Like any test of structure or function, however, ENG, rotary chair, and dynamic posturography may be of little assistance in certain pathologic conditions and of significant assistance in certain other types of disease. The tests are not appropriate for all patients presenting with dizziness, vertigo, or imbalance, although one or more might be considered when the history and physical examination fail to show a definitive cause for the patient's symptoms.