Medical Progress

Modern Vestibular Function Testing

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Current tests of vestibular function concentrate on the horizontal semicircular canal-ocular reflex because it is the easiest reflex to stimulate (calorically and rotationally) and record (using electro-oculography). Tests of the other vestibulo-ocular reflexes (vertical semicircular canal and otolith) and of the vestibulospinal reflexes have yet to be shown useful in the clinical setting. Digital video recording of eye movements and vestibular-evoked responses are promising new technologies that may affect clinical testing in the near future.

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As with other sensory systems, there are two general categories of tests of vestibular function: those relying on the subjective response of a patient and those relying on objective measurements of reflex activity. Unlike tests of the auditory and visual systems, however, quantifying the sensation of movement derived from excitation of the vestibular receptors has been a difficult task for clinicians.[1] It is often impossible for a patient to differentiate those sensations that are strictly vestibular from visual and proprioceptive sensations. Equally important, the subjective awareness of vestibular stimulation depends on a patient's general state of alertness and degree of cooperation. For these reasons, modern vestibular function testing has focused on objective measurements of vestibular reflex activity. The horizontal semicircular canal-ocular reflex has received the most attention to date because there are several relatively simple techniques for stimulating and recording it. In the future, the clinical assessment of vestibular function must include tests of the other suborgans of the vestibular apparatus, namely, the vertical semicircular canals and otolith organs.

Electronystagmography

Methods of Recording Eye Movements

Electro-oculography (EOG) is the simplest and most readily available method for recording eye movements.[2] With this technique, a voltage surrounding the orbit is measured whose magnitude is proportional to the amplitude of the eye movement. When used for evaluating vestibular function, the technique has been termed electronystagmography (ENG), and often the terms EOG and ENG are used interchangeably.[3-5] With EOG the velocity, frequency, and amplitude of spontaneous or induced nystagmus and the changes in these measurements brought about by a loss of fixation—either with the eyes closed or with the eyes open in darkness—can be quantified. Also, visually guided eye movements (saccadic and pursuit) can be quantitatively assessed.

The principle of electro-oculography is shown in Figure 1.[6] The pigmented layer of the retina maintains a negative potential with regard to the surrounding tissue by means of active ion transport. The potential difference between the cornea and the retina, known as the corneoretinal potential, acts as an electric dipole oriented in the direction of the long axis of the eye. In relation to a remote electrode, an electrode placed in the vicinity of the eye becomes more positive when the eye rotates towards it and less positive when it rotates in the opposite direction. With EOG, the measured voltage is proportional to the sine of the angle of motion.

The advantages of EOG are that it is relatively inexpensive, easily administered, noninvasive, does not interfere with vision, and does not require head restraint. It is reasonably accurate, even for the large horizontal eye movements that are encountered during routine vestibular and ocular motor testing. The disadvantages of EOG include the inability to accurately measure vertical eye movements, interference of eye-blink artifacts,[7] poor signal to noise ratio, susceptibility to changes in skin resistance caused by perspiration, and a dependence on lighting conditions in the test room.[8,9]

Table 1 compares the characteristics of EOG with those of three other eye movement recording techniques that are currently used, primarily in research.[10-14] These other techniques are more sensitive than EOG but at present are not practical for routine ENG testing. The direct video recording of eye movements is the newest and most promising of these techniques. A video camera is used that interfaces with a digital computer. At regular intervals images are stored by the computer for subsequent data analysis.[13] Specialized algorithms for digital signal processing are then used to determine horizontal and vertical eye positions. With the rapid advances occurring in this area, it is reasonable to expect that the sensitivity will improve and the costs will decrease in the near future.

Electronystagmography can be used to evaluate many types of eye movement disorders by adapting the testing procedure for specific abnormalities. It is useful, however, to have a standard test battery that screens all important areas. A typical test battery includes tests for pathologic nystagmus, vestibulo-ocular reflex function (usually the bithermal caloric test), and visual ocular control (saccades and smooth pursuit).

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ABBREVIATIONS USED IN TEXT

ENG = electronystagmography
EOG = electro-oculography
VOR = vestibulo-ocular reflex
VOR-Fix = VOR with fixation-suppression
VVOR = visual vestibulo-ocular reflex

Recording for Pathologic Nystagmus

Pathologic nystagmus may be spontaneous (present in the primary position with the patient seated), positional (induced by a change in head position), or gaze-evoked (induced by a change in eye position).[6]

Spontaneous nystagmus of peripheral vestibular origin is strongly suppressed with fixation and usually is not evident on routine examination unless a patient is seen within a week of the acute incident (Table 2). By contrast, peripheral vestibular nystagmus can be recorded with patients' eyes closed or with their eyes open in darkness and for as long as five to ten years after an acute peripheral vestibular lesion. About 10% to 20% of normal subjects will have a low-velocity spontaneous nystagmus with eyes closed or with eyes open in darkness—less than 3 degrees per second.[5,15] As a general rule, spontaneous nystagmus present with fixation—that is, seen on routine examination—that persists for more than a week is a central sign. Congenital spontaneous nystagmus is usually easily differentiated from acquired central vestibular nystagmus because the former has a high frequency and variable waveform and has been present since infancy.

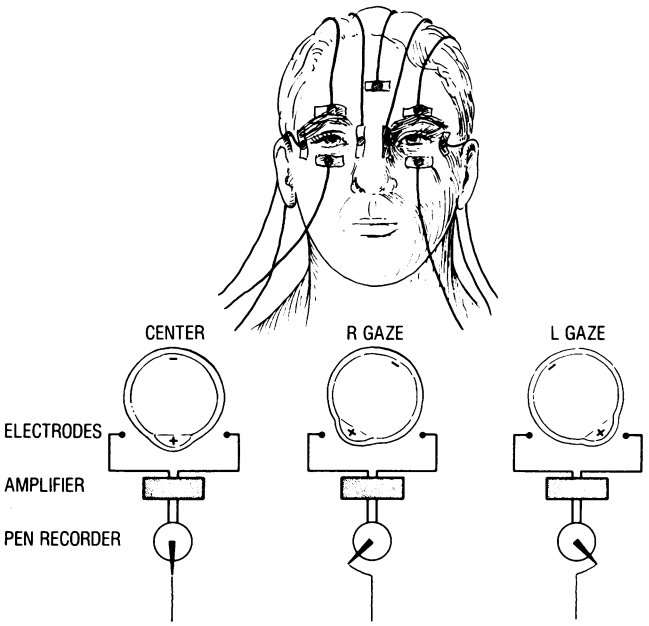

Figure 1.—The technique of electro-oculography is shown (from Baloh[6]).

Positional nystagmus can be static or paroxysmal (Table 3). Static positional nystagmus is induced by slowly placing the patient in the supine, right lateral, and left lateral positions. This type of positional nystagmus persists as long as the position is held. Paroxysmal positional nystagmus, on the other hand, is induced by a rapid change from the erect sitting to the supine head-hanging left, center, or right positions. It is initially high in frequency but rapidly dissipates within 30 seconds to 1 minute.

The most common variety of paroxysmal positional nystagmus—so-called benign positional nystagmus—usually has a 3- to 10-second latency before it begins and rarely lasts longer than 15 seconds. The nystagmus has linear and torsional components with the fast component directed upward—that is, towards the forehead.[16] A key feature is that patients experience severe vertigo with initial positioning but with repeated positioning, vertigo and nystagmus rapidly disappear. By contrast, paroxysmal positional nystagmus of central origin typically does not decrease in amplitude or duration with repeated positioning, does not have a clear latency, and usually lasts longer than 30 seconds.[17] The direction is unpredictable and may be different in different positions. It is often truly vertical with the fast component directed downward—that is, towards the cheeks.

Static positional nystagmus is a common finding both in normal subjects and patients when eye movements are recorded with their eyes open in darkness or with their eyes closed. As in the case of spontaneous nystagmus, however, the average slow-phase velocity of static positional nys-

TABLE 1.—*Important Features of Different Eye Movement Recording Techniques*

	Eye Movement Recording Techniques			
Feature	*Electro-oculography*	*Infrared Reflection*	*Scleral Search Coil*	*Video**
Recording device	Paste-on electrodes	Photovoltaic diodes on glasses	Coil inside contact lens	Video camera
Principle	Corneoretinal potential	Differential reflection of iris and sclera	Electric current induced in coil	Digital processing of video image
Range of horizontal eye movement, degree	±40	±10-15	Unlimited	Unlimited
Range of vertical eye movement, degree	±30	±5-10	Unlimited	Unlimited
Range of torsional eye movement	...	...	Unlimited	Unlimited
Approximate accuracy, degree	1-2	0.5	0.01	1
Approximate cost, $†	2,000	8,000	20,000	100,000
Will record when patient's eyes closed	Yes	No	Yes	No
Able to record normal vision	Yes	Yes	Yes	Yes
Able to record during head movement	Yes	No	Yes	Yes
Susceptible to eye-blink artifacts	Yes	Yes	Yes	Yes
Sensitive to changes in room lighting	Yes	No	No	No
Sensitive to electrical interference	Yes	Yes	Yes	No
Sensitive to electromyographic interference	Yes	No	No	No

*Computer analyzed video recordings.
†Including electronic amplifiers, if needed.

tagmus in normal subjects does not exceed 3 degrees per second.[15,18,19] The nystagmus may be unidirectional in all positions or direction-changing in different positions. Both direction-changing and direction-fixed types of static positional nystagmus occur most commonly with peripheral vestibular disorders, but both also occur with central lesions.[19] Their presence only indicates a dysfunction in the vestibular system without a localizing value. As with spontaneous nystagmus, however, a lack of suppression with fixation and signs of associated brain-stem dysfunction suggest a central lesion.

Gaze-evoked nystagmus is induced by having a patient fixate on a target 30 degrees to the right, left, above, and below the center position. Eye position should be held for at least 30 seconds. A gaze deviation beyond 40 degrees should be avoided because it may result in nystagmus even in normal subjects ("end-point nystagmus"). Normal subjects can have gaze-evoked nystagmus in the dark or with eyes closed, but gaze-evoked nystagmus recorded with fixation is always an abnormal sign.

Symmetric gaze-evoked nystagmus is most commonly produced by the ingestion of drugs such as phenobarbital, phenytoin, alcohol, and diazepam. It can also occur in patients with such varied conditions as myasthenia gravis, multiple sclerosis, and cerebellar atrophy.[1] Asymmetric horizontal gaze-evoked nystagmus always indicates a structural brain-stem or cerebellar lesion, with the lesion usually on the side of the larger amplitude nystagmus ("Bruns's nystagmus").[20] Rebound nystagmus is a type of gaze-evoked nystagmus that either disappears or reverses direction as an eccentric gaze position is held. When the eyes are returned to the primary position, nystagmus occurs in the direction of the return saccade. Rebound nystagmus is the only variety of nystagmus thought to be specific for cerebellar involvement.[21,22]

Bithermal Caloric Testing

The caloric test uses a nonphysiologic stimulus to induce endolymphatic flow in the horizontal semicircular canal and thus horizontal nystagmus by creating a temperature gradient from one side of the canal to the other. Unfortunately, because of their position in the temporal bone, the vertical semicircular canals cannot be reliably activated by a caloric stimulus.

With bithermal caloric testing each ear is irrigated with a constant flow rate of water that is 7°C below body temperature (30°C [86°F]) and 7°C above body temperature (44°C [111°F]). The patient lies in the supine position with the head tilted 30 degrees forward so that the horizontal semicircular canals are in the vertical plane. Recordings are made with the patient's eyes open behind Frenzel glasses or in total darkness. Recording with patients' eyes closed is not recommended since eye closure can suppress vestibular nystagmus in some subjects; in others, lid artifacts are prominent.[23] From the ENG recordings, the maximum slow-phase velocity is calculated for a five- to ten-second interval at the peak of response. Slow-phase velocity is a much more sensitive indicator of vestibular function than either the duration or frequency of the caloric response.[24]

The four responses of a bithermal caloric test are routinely compared with two standard formulas. The vestibular paresis formula,

$$\frac{(L\ 30°C + L\ 44°C) - (R\ 30°C + R\ 44°C)}{L\ 30°C + L\ 44°C + R\ 30°C + R\ 44°C} \times 100,$$

compares the right-sided responses with the left-sided responses, and the directional preponderance formula,

$$\frac{(L\ 30°C + R\ 44°C) - (L\ 44°C + R\ 30°C)}{L\ 30°C + R\ 44°C + L\ 44°C + R\ 30°C} \times 100,$$

compares nystagmus to the right with nystagmus to the left in the same person. In both of these formulas, the difference in response is reported as a percentage of the total response. This is important because the absolute magnitude of caloric response is dependent on several factors, including age. Dividing by the total response normalizes the measurements to remove the large variability in absolute magnitude of normal caloric responses. In our laboratories, the upper normal value for vestibular paresis is 22%, while that for directional preponderance is 28% (using the maximum slow-phase velocity in the above equations).[15]

A caloric fixation-suppression index can be obtained by having a patient fixate on a target during the middle of the response. Because the slow-phase velocity of caloric-induced nystagmus is constantly changing, it is important that the fixation period occur near the time of maximum

TABLE 2.—*Types of Spontaneous Nystagmus*

Nystagmus Type	*Description*	*Location of Lesion*
Peripheral vestibular	Combined horizontal-torsional, inhibited with fixation except in acute stage (<1 week)	Labyrinth or vestibular nerve
Central vestibular	Pure horizontal, vertical, or torsional not well inhibited with fixation	Central vestibular pathways—brain stem and cerebellum
Congenital	High-frequency variable waveform present since infancy	Unknown, no structural correlate

TABLE 3.—*Types of Positional Nystagmus*

Nystagmus Type	*Description*	*Locating Lesion*
Paroxysmal Positional		
Peripheral	Torsional up-beat, brief (<30 sec), fatigability, usually prominent in only one position	Labyrinth, probably posterior semicircular canal
Central	Pure horizontal or vertical, persists (>30 sec), no fatigue, prominent in multiple positions	Fourth ventricular region
Static Positional		
Peripheral	Combined horizontal-torsional, inhibited with fixation except in acute stage	Labyrinth or vestibular nerve
Central	Pure horizontal, vertical, or torsional, not inhibited with fixation	Central vestibular pathways—brain stem and cerebellum

response to obtain the best estimate of fixation-suppression. The fixation-suppression index is defined as the average slow-phase velocity with fixation divided by the average slow-phase velocity without fixation × 100. In normal subjects, the average visual-suppression index is 48% ± 10%.[25]

Abnormal findings on the bithermal caloric test are summarized in Table 4. As a general rule, a substantial vestibular paresis indicates a peripheral vestibular lesion (including the nerve root entry zone), while a substantial directional preponderance is nonlocalizing—that is, it can occur with peripheral and central lesions. The caloric test is relatively insensitive for identifying bilateral vestibular lesions, such as those caused by ototoxic drugs. Because of the wide range of normal values for a maximal slow-phase velocity—5 to 100 degrees per second in our laboratory—a patient's value may decrease severalfold before falling below the normal range.[24] Lesions of the cerebellum occasionally can lead to bilateral increased caloric responses, but again because of the wide range of normal values, it is rare to find caloric responses that exceed the upper normal range. Impaired fixation-suppression of caloric-induced nystagmus indicates a central nervous system lesion, most commonly a lesion involving the midline cerebellum.

Saccades and Smooth Pursuit

Along with the vestibulo-ocular reflexes, two visually controlled ocular stabilizing systems produce conjugate eye movements—the saccadic and smooth pursuit.[26] The saccadic system responds to a retinal position error to bring a peripheral target to the fovea in the shortest possible time. The smooth pursuit system maintains gaze on a moving target by generating a continuous match of eye and target velocity. Optokinetic nystagmus is a form of smooth pursuit in which eye tracking motion in one direction is periodically interrupted by corrective saccades in the opposite direction to relocate the gaze onto new targets coming into the visual field. Saccadic and pursuit eye movements are typically induced by having a patient follow a target moving in a stepwise and a sinusoidal pattern, respectively. For optokinetic testing, a striped pattern is moved across the patient's visual field in a clockwise and counterclockwise direction. With ENG, features of these visually controlled eye movements can be accurately measured and the results compared with normative data. Typically measured are the peak velocity, accuracy, and reaction time of saccadic eye movements.[27] For pursuit and optokinetic nystagmus, the tracking eye velocity is compared with the target or optokinetic drum velocity.[27]

Abnormalities of visual ocular control are helpful for localizing lesions of the central nervous system (Table 5).[28] With one exception, peripheral vestibular lesions do not impair visual ocular control. After an acute unilateral labyrinthine or vestibular nerve lesion, smooth pursuit and optokinetic nystagmus slow-phase velocity will be transiently decreased to the contralateral side—that is, in the direction of the spontaneous nystagmus. The asymmetry of smooth pursuit and optokinetic nystagmus disappears in a few weeks despite the persistence of the vestibular nystagmus in the dark.

Rotational Testing of the Horizontal Semicircular Canals

Rotational testing of the horizontal vestibulo-ocular reflex is becoming more widely used because modern motor-driven platforms can be precisely controlled and multiple graded stimuli can be delivered in a relatively short time. In addition, rotatory testing is often less bothersome to patients than caloric testing. Unlike caloric testing, rotatory testing depends only on the inner ear and is unrelated to the physical features of the external ear or temporal bone. Thus, rotational testing is a more reliable vestibular stimulus. A major disadvantage of rotational testing is that both ears are stimulated simultaneously so that it is less useful than caloric testing for identifying unilateral peripheral vestibular lesions.

TABLE 4.—*Interpreting the Results of a Bithermal Caloric Test*

Result	*Description*	*Location of Lesion*
Vestibular paresis	>22%* asymmetry between right- and left-sided responses	Unilateral labyrinth, vestibular nerve, including root entry zone
Directional preponderance	>28%* asymmetry between left-beating and right-beating nystagmus	Nonlocalizing (anywhere in peripheral and central vestibular pathways)
Bilateral hypoactive	Slow-component velocity <5 degrees/sec bilaterally	Bilateral labyrinth, vestibular nerves, including root entry zones
Impaired fixation-suppression	Fixation does not produce at least 50% decrease in maximum slow-component velocity	Central—brain stem and midline cerebellar

*Normal values for UCLA; each laboratory should establish normative data.

TABLE 5.—*Summary of Visual Ocular Control Abnormalities Produced by Focal Neurologic Lesions*

Location of Lesion	*Saccades*	*Smooth Pursuit and Optokinetic Nystagmus Slow Phase*
Cerebellopontine angle	Ipsilateral dysmetria*	Progressive ipsilateral impairment
Diffuse cerebellar	Bilateral dysmetria	Bilateral impairment
Intrinsic brain stem	Decreased maximum velocity, increased delay time	Ipsilateral or contralateral impairment
Basal ganglia	Hypometria,† increased delay time (bilateral)	Bilateral impairment
Frontoparietal cortex	Contralateral hypometria	Normal
Parieto-occipital cortex	Normal	Ipsilateral impairment

*Undershoots and overshoots.
†Undershoots only.

For rotational testing in our laboratories, a patient is seated in a chair mounted on a motorized rotating table placed inside a light-tight electrically shielded room.[29] For EOG calibration, an array of three light-emitting diodes spaced at the center and 15 degrees to the right and left is attached to a chair directly in front of the patient. Frequent calibrations are interspersed throughout the testing procedure to correct for any fluctuations in the corneoretinal potential. The rotatory chair, a surrounding optokinetic drum, and calibration lights are all controlled by the same microprocessor that analyzes the nystagmus response.

The vestibular system is tested by rotating the chair sinusoidally—in the dark (vestibulo-ocular reflex, or VOR), in the light with the optokinetic drum stationary (visual vestibulo-ocular reflex, or VVOR), and in the dark with the center light-emitting diode lit (VOR with fixation-suppression, or VOR-Fix). In the first instance, the vestibular system is tested without visual influence, whereas in the second, the vestibular and optokinetic systems are stimulated in a synergistic fashion; in the third, the pursuit system is used to suppress vestibular-induced nystagmus. The computer generates stimulus signals at frequencies ranging from 0.0125 to 1.6 Hz and peak velocities of 15 to 100 degrees per second. For screening purposes, we routinely use 0.05 Hz and a peak velocity of 60 degrees per second.

Typical responses of a normal subject to optokinetic and the three standard rotational tests are shown in Figure 2, left panel. In each case, the peak stimulus velocity is 60 degrees per second. All responses are symmetric. The mean gain (defined as peak slow-phase velocity divided by peak stimulus velocity) $\pm$ 1 standard deviation for similar testing in 20 normal subjects is as follows: optokinetic nystagmus, 0.83 $\pm$ 0.13; VOR, 0.50 $\pm$ 0.15; VVOR, 0.99 $\pm$ 0.05; VOR-Fix, 0.03 $\pm$ 0.02.

Patients with unilateral lesions of the labyrinth or eighth nerve have two characteristic abnormalities on VOR testing; they show an asymmetric gain—that is, a decreased slow-phase velocity with rotation towards the side of the lesion—and an increased phase lead (abnormal timing) of eye velocity relative to stimulus velocity at low frequencies of rotation, 0.05 Hz and lower (Figure 2, center panel).[30] The asymmetry of response is most pronounced with acute lesions and often disappears as recovery occurs, whereas the abnormal timing at low frequencies remains indefinitely.[31] With bilateral peripheral vestibular lesions, the VOR responses are symmetrically decreased or absent at low frequencies (Figure 2, right panel), although VOR responses may be preserved at higher frequencies. As a general rule, optokinetic nystagmus and visual-vestibular interaction tests are normal in patients with peripheral vestibular lesions although a transient asymmetry can occur if there is a strong spontaneous nystagmus.

As opposed to patients with peripheral vestibular lesions, patients with central nervous system lesions usually have abnormalities of visual vestibular interaction. Three characteristic abnormal patterns on the standard rotational test battery are shown in Figure 3. Lesions of the lateral medulla involving the vestibular nuclei characteristically affect both visual and vestibulo-ocular responses.[32] Often the optokinetic nystagmus gain is greater towards the side of the lesion, whereas the VOR gain is greater away from the side of the lesion. Fixation-suppression of the VOR is uniformly impaired, particularly slow phases towards the side of the lesion. Patients with lesions involving the vestibulocerebellum have severely impaired optokinetic responses.[33] The gains of their VOR, VVOR, and VOR-Fix responses are approximately the same—that is, these patients are unable to modulate their vestibulo-ocular reflex with vision. Finally, lesions of the visual motor pathways from the parietal-occipital cortex to the horizontal gaze center in the pons impair ipsilateral visual following responses—smooth pursuit and optokinetic nystagmus slow phases—without affecting VOR responses.[34] Fixation-suppression of contralateral VOR slow phases is impaired because pursuit in one direction is used to inhibit VOR slow phases in the opposite direction.

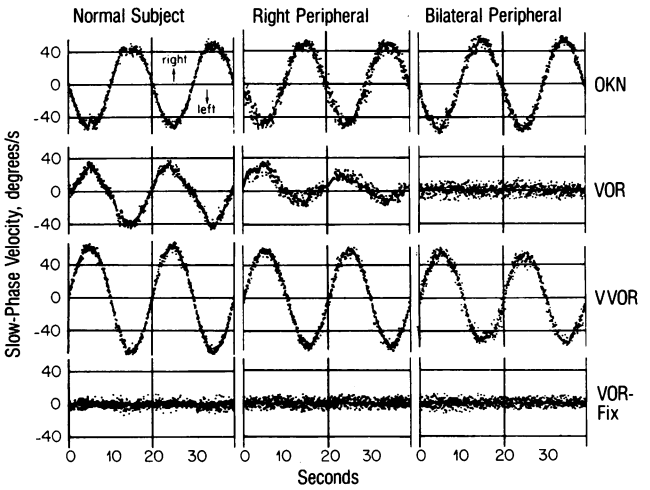

Figure 2.—The plots show slow-phase velocity versus time for optokinetic nystagmus (OKN) and sinusoidal rotational tests (0.05 Hz, peak velocity 60 degrees per second) in a normal subject **(left)**, a patient who underwent right labyrinthectomy **(center)**, and a patient with bilateral vestibulopathy due to using ototoxic drugs **(right)** (from Baloh et al[29]). VOR = vestibulo-ocular reflex, VVOR = visual VOR, VOR-Fix = VOR with fixation-suppression

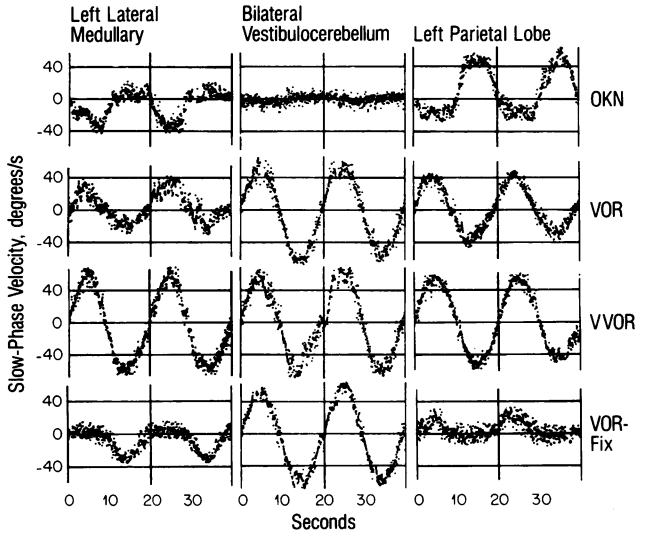

Figure 3.—The plots show slow-phase velocity versus time for optokinetic nystagmus (OKN) and the four standard sinusoidal rotational tests (0.05 Hz, peak velocity 60 degrees per second) in a patient with infarction of the left lateral medullary region **(left)**, a patient with caudal midline cerebellar atrophy **(center)**, and a patient with glioma in the deep parietal lobe on the left side **(right)**. VOR = vestibulo-ocular reflex, VVOR = visual VOR, VOR-Fix = VOR with fixation-suppression

TABLE 6.—*Vestibular Tests and the Suborgans They Stimulate**

Test	Semicircular Canals		Otoliths
	Horizontal	Vertical	
Conventional rotatory chair	×	...	...
Pitch rotation			
Upright	...	×	×
On-side	...	×	...
Ocular counterrolling			
Static	...	...	×
Dynamic	...	×	×
Eccentric rotation	×	...	×
Off-vertical rotation	×	...	×
Linear track	...	...	×
Parallel swing	...	...	×

*× denotes stimulation.

Tests of the Vertical Semicircular Canals and Otoliths

As noted, vestibular tests that are in current use evaluate only the horizontal semicircular canals. Future tests, designed to evaluate other vestibular suborgans—the vertical semicircular canals and otolith organs—will require movements that stimulate these receptors singly or in combination.[35] The determination of which vestibular suborgans are stimulated by a particular movement is often complex and requires a knowledge of three factors: whether the stimulus is an angular or a linear acceleration, the orientation of the skull (and thus the labyrinth) with respect to the movement, and the orientation of the movement with respect to gravity. Table 6 lists the tests we will describe and indicates which suborgans the various tests are designed to evaluate.

Pitch Rotation

Rotating the head in the sagittal plane—like nodding the head "yes"—stimulates all four vertical semicircular canals. If the pitch stimulus is delivered with the subject seated in the upright position (upright pitch), the head changes its orientation with respect to gravity as it tips forward and backward. This change in orientation with respect to gravity stimulates the otolith organs in addition to the vertical semicircular canals. If, however, a subject is seated in a conventional rotatory chair with the head tilted towards the shoulder so that one ear is down, the orientation of the subject with respect to gravity will not change during rotation and the otoliths will not be stimulated. This stimulus, called "on-side" pitch, is thus comparable to conventional rotation described above, except that for on-side pitch rotation, the vertical rather than the horizontal semicircular canals are stimulated.

There are major limitations to the use of pitch rotation clinically because of problems with delivering the stimulus and measuring the response. Although on-side pitch can be delivered with only minor modifications of a conventional rotatory chair, upright pitch requires cumbersome and costly equipment. Moreover, the vertical eye movements that are induced by pitch rotation cannot be measured accurately with EOG; rather, either the magnetic scleral search coil or a video recording system is used. Preliminary studies in normal animals and humans indicate that there is a difference in the vestibulo-ocular responses to pitch between the upright and the on-side positions.[36–38] The gain was decreased and asymmetries were present in the latter compared with the former.

Ocular Counterrolling

The otolith-ocular reflex produces torsional eye movements during static head tilts. Rotating the head towards the right shoulder causes the eyes to counterrotate to the left, and rotating the head towards the left shoulder causes the eyes to counterrotate to the right. Such rotation of the head in the coronal plane is called roll and the counterrotation of the eyes is called ocular counterrolling.[39–41] To complicate matters, dynamic roll movements also stimulate the vertical semicircular canals because of the angular acceleration of the movement. Thus, when using roll stimulation, a distinction should be made between static and dynamic ocular counterrolling.

As with pitch rotation, the use of ocular counterrolling clinically is hampered by difficulties both in delivering the stimulus and in measuring the response. For patients to be rotated in their coronal plane, they must be securely fastened to a cumbersome and costly device. In addition, the amount of torsional eye movement produced by a static tilt in the coronal plane is relatively small. For example, if the head is tilted 45 degrees, the eyes counterroll only about 7 degrees. Electro-oculography and infrared reflection techniques are insensitive to this type of movement so that photographic or video recording or the magnetic scleral search coil must be used.

To date, several studies have indicated that unilateral peripheral vestibular lesions produce asymmetries in static ocular counterrolling.[42–44] Further research is needed, however, to determine the reliability of this technique in clinical vestibular laboratories.

Eccentric Rotation

Eccentric (off-center) rotation is delivered by seating a subject upright in a conventional rotatory chair so that the head is away from the axis of rotation, as if the head were placed at the end of the arm of a centrifuge. During angular acceleration with the head eccentric, the labyrinth is exposed to both a rotational and a linear acceleration and thus both the otolith organs and the horizontal semicircular canals are stimulated. Once a constant angular velocity is achieved, only the otoliths are stimulated. A complexity in fully describing this vestibular stimulus arises from the fact that the net linear acceleration delivered to a subject is the vector summation of the linear acceleration produced by the movement itself and the linear acceleration produced by gravity. The advantages of eccentric rotation are that conventional rotatory chairs (with minor modifications) and EOG methods can be used for this test.

With sinusoidal angular acceleration, the eye movements induced with the head at the center of rotation are compared with those induced during eccentric rotation, the difference being caused by the otolith organs.[45] Preliminary studies suggest that this might be a useful clinical test of the otolith-ocular reflex.[46] An even simpler test of otolith function is to have the patient estimate the subjective vertical—using a vertical light bar—during constant-velocity eccentric rotation.[47] Unlike other tests of subjective vestibular sensation, the sensation of tilt experienced during eccentric rotation appears to be highly reproducible. Patients with unilateral peripheral

vestibular lesions experience less of a sensation of tilt when the damaged ear is outermost.[47]

Linear Acceleration on Sleds

Another technique that has been used to study otolithic function in research laboratories is to deliver a pure linear acceleration on a linear track.[48,49] The device typically consists of a roller-coasterlike sled that runs along a track. As with eccentric rotation, the otolith organs sense the net linear acceleration—that is, the vector summation of the linear acceleration induced by the sled itself and that induced by gravity. For a relatively simple case in which the subject is placed on the sled facing the side as if looking out of the side window of an automobile moving forward, a consistent horizontal eye movement (including nystagmus) can be recorded with EOG. For other head orientations, vertical or torsional eye movements are induced, requiring other eye movement recording techniques such as the magnetic scleral search coil or a video system. The use of linear sleds clinically would be severely limited by the expense and size of the equipment.

Parallel Swing

The parallel swing is a simple technique for inducing linear acceleration that may be practical in a clinical laboratory.[50,51] It consists of a platform suspended from the ceiling by supporting cables at each of its four corners. For small-amplitude displacements, almost pure horizontal linear acceleration is experienced by a subject seated on the platform. As with a linear sled, the eye movement response on a parallel swing depends on the orientation of the subject's head relative to the linear acceleration of the swing and gravity. Thus, various combinations of horizontal, vertical, and torsional eye movements may be induced. Preliminary studies indicate that horizontal eye movements are reliably induced when normal human subjects are seated in the dark facing the side so that the linear acceleration occurs along the interaural axis.[50,51] Patients with bilateral peripheral vestibular lesions have diminished or absent responses.[51]

Off-Vertical Rotation

The off-vertical rotation test is done by seating the subject in a conventional rotatory chair and then tilting the entire apparatus, including the chair and subject.[52,53] In this way, as the subject rotates, the head is continually changing its orientation with respect to gravity. In the extreme case, in which the chair is tipped completely on its side (earth horizontal axis or "barbecue rotation"), the subject is rotated from supine to lateral to prone to lateral, and so on. Once a constant velocity is achieved, only the otolith organs are stimulated as the canals respond only to angular acceleration.

A major advantage of this type of otolith test is that a conventional rotatory chair can be used if the angle of the inclination is kept small. Subjects can be placed in or removed from the apparatus easily, and conventional EOG can record the eye movements because they are largely horizontal. A disadvantage is that the stimulus often produces nausea.

Off-vertical rotation testing using a constant velocity in normal subjects induces two horizontal eye movement components, a bias and a modulation component. In patients with unilateral peripheral vestibular lesions, the bias component is diminished when a patient rotates towards the involved ear, while the modulation component remains unchanged.[54,55]

Summary of Vertical Semicircular Canal and Otolith Tests

The tests of the vertical semicircular canal and otolith function briefly described are all based on the knowledge that the vertical semicircular canals sense angular acceleration in the coronal and sagittal planes and the otoliths sense linear acceleration in all directions. Each test induces a combination of horizontal, vertical, and torsional eye movements depending on the characteristics of the movement, the orientation of the subject with respect to the movement, and the orientation of the movement with respect to gravity. Further research is needed to determine the potential clinical usefulness of these investigational techniques.

Vestibulospinal Testing

The neural pathways that underlie the vestibular contribution to the control of head, body, and limb positions are collectively called the vestibulospinal system.[56] As noted, current vestibular laboratory tests concentrate on the vestibulo-ocular system; the vestibulospinal system has been relatively neglected. A major reason for this neglect is that it is difficult to accurately assess the role of the vestibulospinal system in isolation from other sensory systems, namely, vision and somatosensation.

Static Force Plates

The simplest method of recording human postural sway uses a so-called force plate. There are several devices of this type, each designed with the basic idea of recording the position of a subject's center of mass when upright. In fact, these devices measure the position of the center of force, which is a good estimate of the position of the center of mass if a body is moving slowly. The major limitation of such devices relates to the following: the nervous system uses a combination of sensory modalities during the maintenance of an upright stance, and static force plates do not yield controlled stimulus-response measures of vestibulospinal function and thus must rely on spontaneous movements of the body. This latter consideration is analogous to assessing the vestibulo-ocular system by simply monitoring eye position in the absence of vestibular stimulation. Measuring postural sway might be useful as a screening test for imbalance, but the information it provides is nonspecific and probably not helpful for identifying vestibular lesions.[57,58]

Moving Platform Posturography

Moving force platforms have been designed to overcome the limitations of static force platforms discussed above both by controlling the relative contributions of the visual, somatosensory, and vestibular inputs that are normally used to maintain an upright posture and by incorporating stimulus-response measures. With such a device, the platform on which a subject stands can be moved simultaneously with the visual surround. By coupling the platform to the sway of the subject, it is possible to maintain a constant angle between the foot and lower leg, thereby reducing a major source of somatosensory input to the postural control system.[59] If the subject closes his or her eyes or if the movement of the visual enclosure is coupled to body sway, the subject is also deprived of visual information about postural sway. In this way, the influence of the labyrinth on the upright posture through the vestibulospinal system can be studied in a more or less isolated manner.[60] A disadvantage of this technique is that during postural sway, many of the suborgans of the vestibular

labyrinth are stimulated simultaneously, including the vertical semicircular canals and the otolith organs. For this reason, moving platform studies are incapable of providing an assessment of the individual suborgans of the vestibular labyrinth. To date, moving force platform studies have indicated that a bilateral reduction in peripheral vestibular function results in abnormal postural sway when the device is operated to isolate the vestibulospinal system.[61] More research is required, however, to determine the usefulness of moving force platforms for routinely assessing the vestibulospinal system.

Vestibular Evoked Potentials

The ability to record a human vestibular evoked potential has obvious merits as it would provide an objective measure of peripheral vestibular function that would be independent of either the ocular motor or postural control systems. Despite the fact that sensory evoked potentials using auditory, visual, and somatosensory inputs have been developed and are in routine clinical use, short-latency vestibular evoked potentials have been recorded successfully only in small laboratory animals.[62,63] This lack of development is related to the difficulty in delivering a vestibular stimulus that is capable of triggering a coordinated volley of neural activity, a requirement for eliciting a measurable evoked potential. The vestibular equivalent of an auditory click, visual flash, or somatosensory prick is a brief, abrupt, high-intensity rotation equivalent to the rotation encountered during a blow to the face—an angular acceleration in the range of 7,000 degrees per second squared.

Prior research regarding human vestibular evoked potentials has focused on recording long-latency cortical potentials rather than brain-stem evoked potentials.[64-67] The results of these studies are conflicting; it is still unclear whether the recorded potentials are specific for the vestibular stimulus. Considering their possible clinical usefulness, research on vestibular evoked potentials will undoubtedly continue.

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Travelers' Diarrhea

IF YOU TAKE A GROUP OF TRAVELERS FROM THE UNITED STATES TO MEXICO, you can show that a prophylactic antibiotic such as trimethoprim-sulfamethoxazole will prevent gastroenteritis in the treated group versus the placebo group. I don't think there's any doubt about that. (Studies show that Pepto-Bismol does the same thing.) The problem with making the recommendation generically and having all travelers carrying around a 7-day, a 10-day, or 2-week supply of trimethoprim-sulfamethoxazole is that it will lead to resistance and lessen the use of a very valuable drug combination. Most gram-negative rods are sensitive to trimethoprim-sulfamethoxazole, including the *E coli* in your own GI tract, and you may run into a *Salmonella* or a *Shigella* which is resistant to the trimethoprim-sulfamethoxazole. So now you've ingested a salad that has a *Shigella sonnei* or a *Shigella dysenteriae,* worse yet. And you are taking an antibiotic that is wiping out your own normal flora, which does have some counterbalancing effect in your fight against invading pathogens. And not only that, the antibiotic that you're taking is not effective against the organism that you've run into because it is resistant to the trimethoprim-sulfamethoxazole. This is the one that bothers me most, personally, when I'm tempted to take Bactrim if I'm going to Mexico.

—JOHN E. CONTE, Jr, MD

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