Vestibular Rehabilitation SIG

American Physical Therapy Association/Neurology Section

In this Issue:

Message from the Editor

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The Vestibular Rehabilitation SIG is excited to bring this special publication to our members on the topic of Vestibular Function Testing. We hope these articles will provide you with a greater understanding of the multiple components of vestibular function testing, including what the tests are, what information they can provide and how to interpret the results of the various tests. The articles in this publication cover the basic elements of testing and more specialized tests and newer advancements in testing. There are also several case examples that will hopefully help you to interpret the results of testing within the context of actual patient presentations.

A very special thank you to Dr. Sherrie Davis, Dr. Neil Shepard, and Dr. Michael Schubert for contributing to this special publication. We cannot thank them enough for sharing their knowledge with us and being part of this publication. Their contributions to our SIG are greatly appreciated.

The Vestibular Rehabilitation SIG is looking forward to bringing our members more special publications such as this on special topics related to vestibular rehabilitation and vestibular pathology. If you have any comments or suggestions, we are interested to hear from you with your ideas. Please contact me at Elizabeth.grace@uphs.upenn.edu.

Vestibular Diagnostic Studies

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Purpose and Overview of Vestibular Diagnostic Testing

Regardless of condition, the ultimate goal of any diagnostic testing is to reveal the cause for the patient's presenting symptoms. Vestibular diagnostic testing is also intended to provide causal information regarding the patient's vertigo, dizziness, lightheadedness or imbalance. Testing can suggest that the patient's symptoms are related to a central vestibular deficit or that a peripheral site of lesion is more likely the cause for symptoms. Additionally, vestibular diagnostic studies can provide valuable information regarding the extent of a vestibular deficit and whether or not that deficit is bilateral or the result of a unilateral abnormality.

Providing information regarding potential site-of-lesion is only one aspect of balance function testing. Compensation is an extremely important piece of information that can be provided by a standard vestibular test battery and should be included in every patient's vestibular work-up.

To clinicians who routinely evaluate and treat patients with complaints of dizziness, the importance of compensation status is very evident. Why do two patients with the exact same vestibular abnormality have very different presenting symptoms or degrees of disability? The reason, often, is that one has compensated for the deficit and the other has not. The plasticity of the vestibular system is such that a person even with a complete loss of vestibular function on one side can be essentially symptom free once compensation has occurred.[1, 2] Vestibular rehabilitation is paramount in transforming patients with uncompensated deficits affecting quality of life, to patients who have compensated and are functioning at a normal or near normal capacity.

The maintenance of balance is complex, requiring integration of sensory information from the visual, somatosensory and vestibular systems. This information is organized and integrated by the central nervous system.[2, 3] When there is conflicting sensory information, the central nervous system must accurately prioritize and utilize the information to maintain stability.[4] Breakdown can occur in one or more sensory modalities or in the central integrators, resulting in a multitude of symptoms including dizziness, vertigo and unsteadiness. Comprehensive balance function testing should include assessment of the peripheral sensory systems, as well as the central nervous system structures involved in balance and equilibrium. These assessments are achieved by evaluating the integrity of the vestibular ocular reflex and postural control mechanisms through vestibular diagnostic testing.

Much of the standard vestibular diagnostic battery relates to evaluation of the vestibular ocular reflex (VOR). This reflex is a three neuron system, which begins with stimulation of the semicircular canals secondary to head movement. This stimulation results in increased activity in the afferent neurons on the side that the head moved toward. This increased signal firing is transferred to the vestibular nuclei in the brainstem and from there the signal is carried to the motor neurons resulting in movement of the eyes.[5, 6] The VOR is what allows us to keep an object of interest in focus while moving our head.[2] Essentially, head movement is sensed by the vestibular end organs of the inner ear. A compensatory eye movement that is equal and opposite of the head movement is elicited as a result, allowing for the maintenance of stable vision.[7] The eye movement resulting from head movement is known as nystagmus. When nystagmus occurs in the absence of VOR stimulation then an abnormality is likely suggested. The integrity of the VOR is evaluated in the clinical setting by stimulating the peripheral vestibular system and analyzing the resulting eye movements.

An assessment of postural control is also an imperative part of vestibular diagnostic testing. The maintenance of

upright stance is a complex task that is accomplished by using and integrating information from various sensory systems. The visual, somatosensory and vestibular systems are utilized for maintaining equilibrium and for providing precise assessment of one's position in space.[2,8] These systems alternate with regard to importance depending on the availability and accuracy of environmental cues.[1] When one system is unavailable or is receiving inaccurate sensory information, the others must be utilized to maintain upright stance.[9,10] The body's center of gravity must be effectively maintained over the base of support. The body and thus, the center of gravity can move either volitionally or unexpectedly, however there are limits to the permissible degree of movement. If the body movement exceeds this limit of stability then a reaction is necessary to avoid a fall.[10,11] The sensory information perceived for the preservation of equilibrium results in various muscle reactions that are necessary for postural control. [12,8] Postural control is evaluated in a clinical setting by assessing the patient's ability to use visual, somatosensory and vestibular inputs alone and in concert to maintain balance.

Two types of compensation should occur following an insult to the peripheral vestibular system. The first type is physiologic compensation and essentially relates to eye movements and the VOR. The second type is functional compensation, which is associated with the maintenance of balance and postural control. Physiologic compensation refers to the return of the necessary eye movements needed for gaze stabilization during head movement, despite the partial or complete loss of vestibular function on one side.[2] This type of compensation is assessed by stimulating the peripheral vestibular system and evaluating the VOR by monitoring the eye movements elicited as part of this reflex. This is achieved through videonystagmography and rotational studies. Functional compensation refers to the return of balance and stability following partial or complete loss of peripheral vestibular function. It essentially relates to the evaluation of maintenance of stance when vestibular information is the only sensory information provided. An assessment of postural control is an imperative part of a vestibular diagnostic battery. This assessment will not only provide invaluable information about one's functional balance ability, it will provide key information regarding functional compensation status. This is achieved through Computerized Dynamic Posturography.

Ocular motor Studies

The Ocular motor studies provide information regarding central vestibular integrity. Eye movements are recorded while the patient is instructed to either stare at or follow a target positioned at a set distance in front of them. Abnormal ocular motility occurs when there is dysfunction in the central neural pathways, specifically the brainstem and cerebellum.[13,14,15] Different patterns of abnormality suggest different central nervous system etiologies. Ocular motor studies can be affected by many non-central vestibular system issues, such as visual deficits, ocular muscle or ocular nerve abnormalities, thus information regarding the integrity of these structures is imperative for interpretation purposes.

Gaze Testing Gaze is the ability to keep the eyes fixated on an object of interest. Our ability to gaze allows us to stare at an object that is in the primary position, straight ahead, or that occurs eccentrically, to the right or left or above or below center, without the intrusion of extraneous eye movements.[13,16] The patient is asked to fixate on a target that is directly in front of them while the presence or absence of gaze-evoked nystagmus is determined. The target is displaced usually 30 degrees or less to the right and then to the left of center and the patient is asked to stare to determine if horizontal gaze-evoked nystagmus is present and to what degree and direction. The same procedure is also employed for vertical gaze using a target above and below the center position. The presence of nystagmus when they eyes are opened and fixated is always pathologic and is *usually* a sign of central vestibular involvement.[13, 5] However, unlike abnormalities with the other ocular motor studies, gaze evoked nystagmus can *sometimes* be the result of a peripheral abnormality. Nystagmus occurring in the absence of fixation that is elicited by a peripheral vestibular system bias can almost always be suppressed with visual fixation. Thus, when nystagmus is observed only with the eyes opened and fixated, a central vestibular abnormality is suspected.[13,14,17] The exception is with acute peripheral vestibular lesions, which produce strong spontaneous nystagmus. In some cases, nystagmus is observed with eyes opened and fixated because the spontaneous nystagmus is suppressed but not completely abolished because of the strength of the nystagmus or the lack of compensation associated with acute abnormalities. [2]

Random Saccades Saccades are the ability to move our eyes rapidly, in a single movement to re-fixate on an object of interest that has either moved from a previous position or has entered our visual field. [16] The patient is asked to follow a target with their eyes while keeping their head fixed. A target is then presented at randomized intervals and locations. The patient must quickly and accurately adjust their focus to stay on the target. Multiple parameters are then assessed for each eye. These parameters can provide insight to the integrity of the central neural pathways that are required to elicit a saccade. It is advantageous to assess each eye individually, (binocular recordings) because patterns of disconjugacy can suggest certain central nervous system etiologies. There are three test parameters measured for each eye during random saccade testing. The first refers to the accuracy of the random saccade. Essentially, it tells us whether the patient could accurately redirect their focus to stay on the target or did they overshoot (exceed the position of the target) or undershoot (fail to reach the target position). [16] The second measurement parameter is velocity. Velocity refers to the peak eye movement speed measured while the eyes are traveling to the target.[16] Finally, latency is the measurement of the momentary lapse in time that occurs between the target relocating and the eyes moving to follow it. [16,2] Random saccade abnormalities are always considered to be the result of central nervous system involvement. Different abnormality patterns can suggest different central sites of lesion.[5]

Smooth Pursuit Smooth pursuit refers to the ability to track an object of interest that is moving in a continuous fashion by using a single, smooth eye movement, as opposed to many small, jerky eye movements. [16,18] It is the eye movement that would be employed when following a moving pendulum with the eyes. The patient is asked to follow a moving target with their eyes while keeping their head fixed. A moving target is then presented at various frequencies of oscillation. The patient must use smooth, continuous eye movements to pursue the moving target. There are several parameters for analysis when it comes to interpretation of smooth pursuit. The first is gain, which refers to the speed at which the eyes moved compared to the target speed. [13] A gain of 1.0 would suggest that the patient's eyes moved at the same velocity as the target's velocity. The second parameter is asymmetry and refers to the percentage difference between the eye's velocities when tracking the target as it moves to the right compared to the left. [13] Finally, smooth pursuit phase measurement indicates whether the eye stayed right with the target or led in front or lagged behind the target.[13] In addition to the objective measurements described, a subjective opinion regarding smooth pursuit morphology should always be made. The judgment regarding smooth pursuit integrity is based on whether the patient was truly able to elicit smooth eye movements to track the target or did they require many small saccadic eye movements to stay with the target. Some degree of saccadic pursuit can be explained by increased patient age. Abnormal pursuit does suggest central pathway involvement, which can broadly be described as the vestibulocerebellum. [13]

Optokinetic Testing Optokinetic Testing involves the elicitation and recording of optokinetic nystagmus. Optokinetic nystagmus is essentially nystagmus elicited by visual stimulation as opposed to vestibular stimulation. It is the slow phase followed by the fast phase reflexive eye movement that would be created when visualizing something that fills at least 90% of the visual field and is moving in a regular or repetitive manner. [16,2,5] Optokinetic nystagmus can be generated when the head is in constant motion while looking at something that is not moving, such as, glancing at a series of telephone poles that one is passing while traveling in an automobile. Optokinetic nystagmus can also be generated by when the head is stationary and one is looking at something that is moving in a repetitive fashion, such as, sitting on a bench while looking at a train passing by. The patient is asked to look ahead as a full field visual pattern (such as long vertical stripes that are cast on a wall) moves either clockwise or counter-clockwise in front of them. Nystagmus is generated and recorded for each direction of optokinetic stimulation. A sinusoidal optokinetic paradigm can be used, where the stimuli moves in one direction and then the other at varying frequencies, similar to the test frequencies utilized in smooth pursuit testing. A fixed velocity optokinetic test can be used, where the stimuli is rotated a constant speed and direction for 60 seconds, during which time optokinetic nystagmus is recorded. The stimulus is then discontinued abruptly and the presence of residual nystagmus, known as optokinetic after nystagmus is measured.[16]

This is repeated in the opposite stimulation direction. In both the sinusoidal and fixed-direction test methods, the velocity of the optokinetic nystagmus is measured and compared to the velocity of the optokinetic stimuli to determine the gain for each direction of stimulation. In the sinusoidal technique, these gains should correlate with the smooth pursuit gains at the same test frequencies. This can be useful for confirming test validity in cases of significantly abnormal smooth pursuit.[13] Gain symmetry is also evaluated for both test methods. Asymmetries most commonly suggest disruption in the cerebellar or brainstem pathways and should also be evidenced in the other ocular motor tests.[13, 5] Optokinetic after nystagmus is evaluated by measuring the velocity of the nystagmus several seconds after the stimuli ceases and comparing the calculated velocity following clockwise and counter-clockwise fixed-direction stimulation. The time it takes the optokinetic after nystagmus to decline or decay is also evaluated following both directions of presentation. Abnormalities related to optokinetic after nystagmus can be suggestive of dysfunction of the velocity storage mechanism in the cerebellum.[5]

Videonystagmography (VNG)

Videonystagmography (VNG) is utilized to evaluate the integrity of both the peripheral and central vestibular systems. Commonly, the ocular motor studies are performed as part of VNG. The ocular motor portion of the VNG provides the majority of the information regarding central vestibular function. Most other portions of the test battery reveal information regarding the peripheral vestibular system. VNG is the only means to assess vestibular function on one side independent of input from the opposite side. Therefore, it is an invaluable tool for lateralizing a unilateral peripheral vestibular lesion.[19] VNG is used to primarily assess semi-circular canal integrity by evaluating the Vestibular Ocular Reflex (VOR); therefore eye movements are recorded and evaluated to obtain information regarding peripheral vestibular integrity. VNG provides information about the integrity of the peripheral and central vestibular systems and physiologic compensation status.

Spontaneous Nystagmus Test The afferent vestibular neurons have a baseline firing rate, even in the absence of head movement or semicircular canal stimulation. The brain or central mechanisms expect equal baseline firing from each side when there is no Vestibular Ocular Reflex (VOR) stimulation.[1] When equal baseline firing is not received, the central system interprets that the lack of symmetric input is the result one side being in an excitatory state and the other in an inhibitory state because of head movement toward the more excited side. The system then elicits the VOR response, which should occur with the presumed head movement. [1,20] That VOR response is a compensatory eye movement that is equal and opposite of the head movement. When asymmetric firing is the result one side being weaker than the other side the same compensatory eye movement will occur. This eye movement is observed and recorded as nystagmus with the fast phase beating toward the stronger or more stimulated ear.[1] To assess for the presence of spontaneous nystagmus, the patient is in a seated position with the eyes opened, while the presence or absence of nystagmus is determined. Vision needs to be denied because in most cases nystagmus that does occur will be abolished with visual fixation. If there is observed spontaneous nystagmus then the direction and velocity of the nystagmus is documented. With peripheral causes the spontaneous nystagmus should suppress or abolish with visual fixation. When spontaneous nystagmus does not suppress or is enhanced with visual fixation then a central etiology may be suggested.[1]

Spontaneous nystagmus is always clinically significant regardless of the degree. The direction of the fast phase of the nystagmus will provide insight into which side is more excited or firing at a stronger rate than the other side. For example, right beating spontaneous nystagmus suggests that the right peripheral vestibular system is being more stimulated than the left. This could be the result of a weakness on the left side or an overly excited state on the right side. When there is no spontaneous nystagmus observed, it does not necessarily mean that the peripheral vestibular mechanisms on both sides are normal and symmetric. Because of the process of physiologic compensation, central adaptive plasticity can occur resulting in the return of the neural firing to the weak side or regulating the over-firing of the irritative side.[1] This results in the improvement of the patient's subjective vertiginous symptoms and the cessation of the spontaneous nystagmus.

Positioning Tests / Dix-Hallpike Maneuvers Dix-Hallpike Maneuvers or testing to assess abnormality during the active process of changing position, are intended to identify patients with Benign Paroxysmal Positioning Vertigo (BPPV). BPPV is the most common cause for vertiginous symptoms in patients with vestibular abnormalities.[21,22]

The most common positioning technique employed for the purpose of eliciting BPPV is the Dix-Hallpike Maneuver.[23,24] Eye movements are observed either with direct observation or with eye movement video monitoring. The removal of vision is not necessary during these maneuvers allowing this to be included in a bedside assessment when there is suspicion of BPPV.

The patient is seated on an examination table with the examiner at their side or behind them. The patient is instructed to turn their head approximately 45 degrees toward the side being assessed for BPPV. The examiner then supports the patient's head and back while the patient reclines into a supine position. With continued support, the head is slightly hyper-extended off of the table, while the examiner watches the eyes for any resultant nystagmus. The position in maintained for 45 to 60 seconds and then the patient is instructed to rise to a seated position, again being supported throughout. The maneuver is then repeated with the head turned in the opposite direction. The downward ear or the direction the head is turned is the side being assessed.

The most common BPPV is a result of posterior semicircular canal involvement.[21,25] The expected nystagmus observed with posterior canal or anterior canal BPPV should be slightly latent, short lived and fatigable upon repeat of the maneuver. Additionally, the nystagmus will be torsional or rotary in nature. When the torsion deviates upward then the posterior canal is the suspected canal of origin, which is the most common observation. When the torsion deviates downward then the less likely anterior semicircular canal is the origin of the BPPV. When the nystagmus is not torsional and persists without fatigue then another causative entity is likely suggested. Another possibility when nystagmus is observed that does not meet the requisite criteria for vertical canal BPPV, is that the otoconia causing the symptoms and eye movements is in the horizontal semicircular canal. A different assessment method is employed to identify horizontal canal BPPV and different eye movements are expected to confirm this diagnosis.

Positional Tests When one is still and nystagmus is observed following a position change then an imbalance in neural activity is suggested. This post-position nystagmus is termed positional nystagmus. The presence of clinically significant positional nystagmus can suggest an uncompensated peripheral vestibular asymmetry.[2] Therefore it is an important indicator of one's physiologic compensation status. In some cases, the characteristics of the nystagmus observed as a result of a position change can provide supportive evidence that a more central vestibular cause may be suggested. The presence or absence of nystagmus that results following a change in position is assessed with vision denied because, again, when a peripheral etiology is the cause, then the positional nystagmus will be suppressed or abolished with visual fixation. A common test battery includes assessing the presence or absence of nystagmus with the patient lying in a supine position, with their head and/or body turned rightward and then leftward and finally in position with the head elevated 30 degrees, which is requisite for the caloric studies. The patient is placed in each position and the eyes monitored for 30 to 60 seconds following each position. The positions employed can be customized based on patient's report of which conditions make them symptomatic. The direction and velocity of any position-provoked nystagmus should be documented.

Position-provoked nystagmus can be described in various ways. The specific attributes associated with the positional nystagmus can suggest that the cause may be more peripheral or centrally mediated. Positional nystagmus can be direction fixed, indicating that it always beats in the same direction regardless of position. In this case, it will beat toward the more stimulated periphery, either because one side is abnormally weak or the other is abnormally strong. Positional nystagmus can be direction changing, indicating that the nystagmus changes direction relative to gravity of the patient's position. Direction changing positional nystagmus can be further described as geotropic, beating toward the ground or the undermost ear, or ageotropic, beating away from the ground or away from the undermost ear.[21,26] The terms geotropic and ageotropic are simply descriptors and do not suggest that one is more indicative of a central cause for the positional nystagmus than the other. That is not the case when the positional nystagmus changes direction within a body position, that is, it begins beating in one

direction and then changes to the other direction all while the same position is maintained. When this direction changing phenomenon occurs it is always deemed clinically significant and a cause related to a central vestibular abnormality is suggested. [2,21] Purely vertical, either up beating or down beating, positional nystagmus also is more commonly associated with a central nervous system involvement.[27] When positional nystagmus is observed, it is expected that the nystagmus will either be suppressed or abolished when the patient is asked to fixate. When visual fixation does not result in suppression of the positional nystagmus, central involvement also may be indicated. In addition to the direction of the position-provoked nystagmus and the correlated positions that it occurs in, degree and incidence of the nystagmus is of value in some vestibular labs. One accepted criteria is that in order to be deemed clinically significant, the positional nystagmus must have a velocity of at least 5 degrees/second. If the nystagmus is less intense, then it needs to be frequent, occurring in at least 50% of the tested positions.[3] Other vestibular diagnosticians feel that any positional nystagmus, regardless of degree or frequency, is clinically significant.[21] It is important that the presence or absence of positional nystagmus and one's criteria for clinical significance be considered concomitantly with the patient's symptoms and case history, as well as, with the results of the other vestibular diagnostic findings.

Caloric Studies Caloric studies provide information about the integrity of mechanisms within each peripheral vestibular end organ, independent of input from the opposite side. In essence, caloric studies allow the VOR to be engaged without participation from the contralateral or inhibited side.[19] As previously discussed, the plasticity of the vestibular system is such that one can have a completely normal VOR response even with a total loss of function on one side. When the intact side elicits the inhibitory response, decreased neural firing that occurs as a result of stimulation or excitation of the opposite side, the correct compensatory eye movement will occur.[3] Stimulation of the vestibular system by moving the head and observing the resulting eye movements is a natural form of stimulation. It is the way the VOR is intended to function. However, from a diagnostic standpoint, this mode of vestibular stimulation is limited because if physiologic compensation has occurred, the eye movement response will be normal even if there is no function on Caloric studies allow for each labyrinth, specifically the horizontal semicircular canal, to be stimulated and assessed without input from the other side.[28]

Caloric testing utilizes temperature change to stimulate the VOR. Fluids within the human body are essentially equal to body temperature. When the endolymph within the horizontal semicircular canal is sufficiently heated or cooled above or below body temperature, the same cupula deflection will occur that would be elicited when the head is moved. The direction of the deflection of the cupula is temperature dependent. That is, when the endolymph is sufficiently heated, the molecules become further apart, making the endolymph less dense. This change in density of the heated endolymph causes ampullopetal deflection of the cupula, or an excitatory responsible.[2, 29] This excitatory response is comparable to the response that occurs with VOR stimulation resulting from head movement. Rightward head movement causes ampullopetal cupula deflection resulting in excitation or increased neural firing on the right side, resulting in right beating nystagmus. Similarly, a right warm caloric stimulation also results in ampullopetal displacement of the cupula producing an excitatory reaction or increased neural firing on the right side, which also elicits right beating nystagmus. [29, 30] Conversely, the opposite occurs when the endolymph is sufficiently cooled, as with cool caloric stimulation. In this circumstance, the molecules within the endolymph become closer together, making the fluid heavier or denser. This increased density results in ampullofugal movement of the cupula.[29] This direction of cupula deflection is the same that results when the horizontal semicircular canal is in an inhibitory state because the head is being moved in the opposite direction.

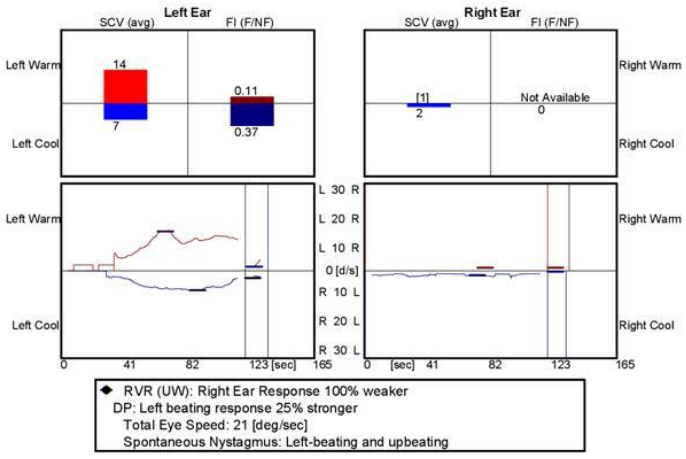
The caloric test is performed by stimulating the peripheral vestibular mechanisms by heating and cooling the endolymph sufficiently to elicit the VOR.[1] The resultant response is compared for right ear versus left ear stimulation and for right beating versus left beating responses. Caloric stimulation can be achieved via water or air irrigations. In most clinical settings, bithermal caloric irrigations are employed. That is, each ear is individually stimulated with each temperature resulting in a total of four irrigations. This technique allows for each ear to produce both an excitatory (warm stimulation) and inhibitory (cool stimulation) response. [19] The nystagmus that is elicited following each

irrigation is recorded, measured and then compared. The caloric response is observed in the absence of vision to prevent visual fixation, which would suppress or abolish the response. Regardless of the delivery method used, the temperature gradient stimulating the external auditory canal must effectively reach the labyrinth equally on each side and for each irrigation. Caloric response elicited from stimulation on one side is compared to the response that results from stimulation of the other side. Thus, equal symmetric stimulation is paramount. The nystagmus that results from caloric stimulation is measured by calculating the peak slow phase velocity for each of the four irrigations. Decisions regarding whether both vestibular labyrinths are weak, whether one is weaker than the other and whether there is a predominance of one direction of nystagmus can be determined by comparing the peak slow phase velocity responses for each of the irrigations.

Assessing unilateral caloric weakness provides interpretative information regarding whether there is a reduced vestibular response on one side. This measurement compares the response for caloric stimulation of the right ear to the response of caloric stimulation of the left ear. The result of this comparison is expressed in percentage. The criteria for clinical significance should be established for each clinic; however a unilateral weakness equal to or greater than 25% is commonly accepted as significant.[28] Essentially this means that if the caloric induced response is at least 25% weaker when one ear is stimulated compared to the other ear, and then an abnormally reduced vestibular response is indicated on the side with the weaker reaction.

Assessing directional preponderance provides interpretative information regarding whether there is a stronger response for one direction of nystagmus compared to the other direction. This measurement compares right beating caloric induced nystagmus that results from right warm and left cool stimulation to left beating caloric induced nystagmus that occur as a result of left warm and right cool stimulation. The result of this comparison is also expressed in percentage. Again, the criteria for clinical significance should be established for each clinic; however 25% is also commonly accepted as significant.[2] A directional preponderance that is equal to or greater than 25% suggests that with equally effective stimulation, there is an abnormal predominance of one direction of caloric induced nystagmus. This is commonly the result of pre-existing nystagmus that occurs spontaneously or in the pre-irrigation condition that is essentially added to the caloric induced nystagmus.[28] Unlike unilateral weakness, which definitively implicates one side as the weaker or paretic side, directional preponderances can occur because of a system bias in which one side may be abnormally weak or the other abnormally strong. Therefore, the finding of a directional preponderance is not a useful parameter for lateralizing a unilateral peripheral vestibular abnormality. Instead, it suggests a physiologically uncompensated bias within the vestibular system. [2]

A bilateral weakness is suggested when the response for all irrigations are lower than the clinically established norms. This threshold for abnormality is dependent upon stimulation method (air irrigations versus water irrigations) and consequently will vary from clinic to clinic. The criterion for bilateral weakness sometimes uses the sum of all irrigations to determine if this value is lower than expected, suggesting a weak response for both sides. For example, if adding all four caloric responses yield a sum less than 22° /second, a bilateral weakness is suggested.[35] In other centers the absolute values of each caloric response is compared to a set threshold for an expected normal response. In this case, a clinic may use an established norm of 10°/second as their lower limit of normal. In the event that the responses to all four irrigations are less than 10°/second, bilateral involvement may be suggested.[28] In cases of bilateral vestibular weakness, testing at higher frequencies is important for establishing the degree of bilateral involvement and the amount of residual function. Rotational Studies can prove to be very helpful in this regard.



Sample caloric results showing unilateral (right) reduced vestibular response

Rotational Chair

Rotational studies enhance the investigation of the peripheral vestibular mechanisms, providing further information regarding physiologic compensation status, and sometimes identifying vestibular deficits not evidenced by VNG studies.[2,33] The frequency range of the stimuli used for rotational studies is closer to the frequency range at which we move during every day activities. [2,34] Therefore, evaluating the VOR at multiple frequencies, closer to the frequencies employed in daily life can be valuable. Evaluation of the peripheral vestibular mechanisms using rotation can be an advantageous because the stimulation of the VOR is more controlled and potentially more precise than caloric stimulation, which can be influenced by examiner technique and patient anatomy. Additionally, it is a more natural, physiologic stimulation method than caloric studies. The innate limitation of rotational testing is that each side cannot be assessed without input from the opposite side, precluding lateralization of a unilateral abnormality.[34,35,36] Rotational studies can be an important adjunct to ENG/VNG when vestibular integrity and physiologic compensation status are being assessed. Rotational testing can provide information regarding the velocity storage mechanism in the cerebellum. In cases where caloric testing suggests bilaterally weak vestibular mechanisms, rotational studies are an invaluable tool to quantify the extent of this bilateral impairment. Knowing the degree of bilateral vestibular loss can be helpful from a rehabilitative perspective. This information can be useful in determining if vestibular therapy should focus on utilization of residual vestibular function or whether using sensory information from the visual and somatosensory systems to supplement for the loss of vestibular function is warranted.[37,38,39]

Rotational testing uses various types of controlled head movements with known velocities and frequencies to elicit the VOR. The stimuli are varied in terms of frequency and the resultant eye movements are recorded and measured. The head is fixed to the chair so that the frequency of the head movement can be inferred by the frequency of the chair movement. When the chair/head are moved in one direction the horizontal semicircular canal on the side the head is moved toward, is stimulated, while the contralateral horizontal semicircular canal is inhibited. [30,3] Clockwise motion of the rotational chair stimulates the right semi-circular canal, which results in an eye movement in the opposite direction of the head movement. This eye movement is characterized by a slow phase to the left (opposite to the head movement direction) and then a fast phase eye movement to reposition the eye back to baseline. This leftward slow phase followed by a right-ward fast phase is right beating nystagmus. When the chair, and head, are initially moved or accelerated, the inertia of the movement will result in endolymph movement and hence deflection of the cupula. In test situations where the head continues to rotate at a fixed velocity and a fixed direction, the movement of the fluid will catch up with the movement of the head, resulting in the return to baseline position of the cupula. This mechanical portion of the response takes approximately six seconds. [40] However, the nystagmus or compensatory eye movements will continue to be present for several seconds beyond the return of the cupula to its' resting position. This prolongation of the nystagmus is the result of the velocity storage integrator in the cerebellum. Centrally mediated velocity storage perseverates or sustains the vestibular signals produced by peripheral vestibular stimulation.[41] Information regarding velocity storage integrity can be achieved by looking at several rotational chair test parameters, specifically, phase and time constant.[2]

Sinusoidal harmonic acceleration testing consists of stimulating the VOR by oscillating the rotational chair at various frequencies while recording the eye movement response. Most test protocols consist of a multiple frequency paradigm from 0.01 Hz through 0.64 Hz, with sinusoidal oscillations performed in octave intervals. The patient is seated in a chair with seat belts for safety and a head strap to fix the head to the chair and eliminate unwanted head movements. Eye movements are recorded with infrared video cameras, similar to recording measures utilized in VNG. Vision must be eliminated so that the patient is unable to suppress the VOR response with visual fixation. This is achieved by housing the rotary chair in a light tight booth to eliminate vision or with the use of goggles that preclude vision. The movement of the chair is computer controlled to provide precise rotational stimulation while the eye movements are compared to the chair movement with regard to gain and phase.[37] The chair continues to be oscillated sinusoidally at varying frequencies, while the slow phase eye velocity of the

VOR response is recorded. Three test parameters are assessed for each plotted sinusoid. These parameters include phase, gain and symmetry.[2,34]

Gain is the comparison of the slow-phase eye velocity to the velocity of the head (or chair). The eye movement necessary to compensate for head movement is measured and assessed. It is a direct assessment of the VOR and the responsiveness of the peripheral vestibular system. When gains are low relative to norms then bilateral peripheral vestibular weakness is likely suggested. When this is the case, caloric responses should correlate and are expected to be reduced for all irrigations. [32] Because the gain value represents the degree of eye movement as a function of head movement for both directions of stimulation, gain can be below normal threshold when there is an uncompensated unilateral vestibular deficit resulting in reduced response when the chair is oscillated in one direction. When the slow phase eye velocity is averaged for both directions of oscillation, the result will be a lower than normal gain because of the asymmetric response pattern. In this case, the reduced gain will be accompanied by a clinically significant slow phase eye velocity asymmetry.

Asymmetry assesses whether stimulation to the right produces an adequate slow phase eye movement response when compared to leftward stimulation. In some cases of asymmetric function between the right and left peripheral vestibular mechanisms, stimulation or excitation of one side will produce a normal VOR response while the other produces a less than normal compensatory eye movement when stimulated. That is an adequate equal and opposite compensatory eye movement is elicited for one direction of rotation while a less than expected compensatory eye movement results when rotation occurs in the opposite direction. Asymmetric slow phase eye velocity responses suggest a directional preponderance for one direction of slow phase eye velocities or for one direction of VOR stimulation.[32] This VOR asymmetry can be the result of a paretic state on one side, resulting in lower slow phase eye velocity than expected or from an irritative state on one side, eliciting a greater than expected VOR response. Abnormality lateralization can be further defined by other diagnostic studies, such as caloric testing. In addition to suggesting a system bias, either paretic or irritative, asymmetry can also provide information regarding physiologic compensation status. When a clinically significant asymmetry is yielded during sinusoidal harmonic acceleration testing, the lack of physiologic compensation is indicated.

Phase may have the greatest clinical utility with regard to evaluating peripheral vestibular integrity. [2] Phase angle refers to the timing relationship between the head movement and the slow phase compensatory eye movement produced by the VOR. [34] Phase quantifies the degrees at which the compensatory eye movement of the VOR led ahead or lagged behind the head movement portion of the VOR. When the eye moves exactly equal and opposite to head movement, as seen with a normal VOR at functional test frequencies, then the eye is 180^{o} out of phase with the head, represented clinically as a phase angle of zero. For stimulation frequencies employed during rotational chair testing, the compensatory eye movement is not exactly equal resulting in phase angles greater than zero, described as phase leads. There are frequency specific expected phase angles based on normative data. When timing relationship between eye movement and head movement exceed this limit of normal it most commonly results in an increased phase lead. [41] Phase leads suggest a loss of velocity storage that is provided by the central vestibular mechanisms to enhance the VOR response, particularly for low frequency stimulation [41]. Clinically significant increased phase leads are often correlated with peripheral vestibular abnormalities.[34]

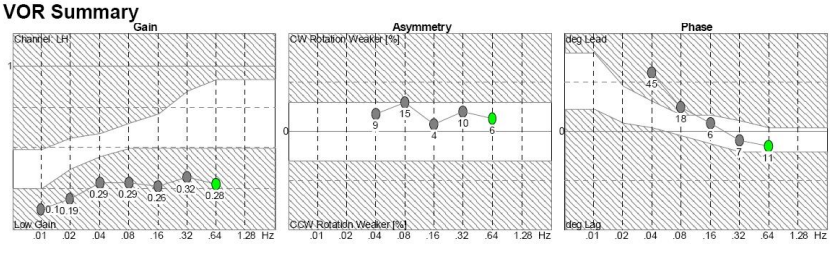
Velocity Step Testing is another form of rotational stimulation. It involves rotating the patient at a constant velocity in a fixed direction while recording the subsequent VOR eye movement response. The rotational stimulation is performed in a clockwise direction, exciting the neural afferents in the right semi-circular canal, while inhibiting neural firing of the peripheral vestibular system on the left. This is repeated in a counter clockwise direction resulting in the opposite excitatory / inhibitory pattern of neural stimulation. A commonly used protocol entails the chair being accelerated in a clockwise direction at an angular acceleration magnitude of 100^{o}/second^{2} until it reaches a fixed test velocity, of 100^{o}/second. The acceleration impulse lasts approximately one second.[40] The chair then continues to rotate at this set, constant velocity for 45 – 60 seconds while the resultant nystagmus is recorded. The nystagmus that is yielded from the active rotation portion of the velocity step is referred to as per-rotary nystagmus. The chair is then rapidly decelerated

to the same degree as the initial acceleration impulse and eye movement monitoring continues for the next 45 to 60 seconds. The eye movements that result when the motion of the chair is ceased is referred to as post-rotary nystagmus. [42]

Similar to sinusoidal stimulation, velocity step stimulation produces an excitatory or increase in neural firing in the direction of rotation. The slow phase eye velocity is utilized for interpretation purposes because it again, it is the compensatory eye movement generated by the peripheral portion of the VOR. The slow phase eye velocities are plotted over time for both the per-rotary and post-rotary conditions. The test parameters measured from this data are gain and time constant, with time constant being the most useful information resulting from the velocity step test when performed in combination with sinusoidal harmonic acceleration. [41,42] Time constant is defined as the time, in seconds, that it takes the rotationally induced nystagmus to go from its' peak slow phase eye velocity to 37% of that peak velocity.[41,42] This provides information regarding velocity storage. Abnormally reduced time constants can occur for both per and post rotary conditions and clockwise and counter clockwise directions of rotation when there is bilateral reduction of velocity storage associated with bilateral peripheral vestibular lesions. Reduced time constants can also be the result of unilateral peripheral vestibular compromise, affecting the conditions in which the impaired labyrinth should be in an excitatory state. This is the result of regulation by the velocity storage integrator in the cerebellum to reduce velocity storage, as a central compensation phenomenon when there is an insult to the peripheral mechanisms. [41,42] Conversely, when there are abnormally long time constants, which exceed the upper limits of normal, then cerebellar involvement may be indicated. This is felt to be the result of failure of the velocity storage integrator to attenuate the sustained eye movements in a normal time frame. [12, 21] This central finding should likely be evidenced during the other tests of central vestibular integrity, particularly the ocular motor studies.[41]

Tests of Visual / Vestibular Interaction In addition to sinusoidal harmonic acceleration and velocity step tests, the rotational chair can be utilized to perform various other measures for the purpose of assessing the interaction of the visual and vestibular systems. These are commonly incorporated into a standard rotational chair test battery. An assessment of one's ability to suppress their VOR with visual fixation can be easily performed by eliciting the VOR with rotational stimulation at a specific frequency of oscillation while asking the patient to stare at a target, which moves concurrently with the chair. It is expected that the rotational induced nystagmus will be abolished or central involvement may be suggested.[2] The second type of visual vestibular interaction assessment that can be employed as part of the rotational studies relates to enhancing the VOR by stimulating the visual system concurrently. This is performed by presenting fixed optokinetic stripes on the walls of the rotational chair booth while rotating the patient sinusoidally at a predetermined test frequency. It is expected that the optokinetic stripes will elicit visually mediated nystagmus at the same time that nystagmus is produced as a result of vestibular stimulation, resulting in an increase in gain compared to the same sinusoidal frequency performed in darkness. This test also can provide information regarding the integrity of the central vestibulo-ocular pathways. [2] Central involvement suggested by either of these measures should also be evidenced during the gaze and /or ocular motility tests.

Above: Test set up. Below: sample Rotational chair results



Video Head Impulse Testing (vHIT)

VNG and Rotational Studies provide information about peripheral vestibular integrity by assessing the Vestibular Ocular Reflex. However, all subtests, including caloric studies, stimulate the horizontal semicircular canals specifically. An assessment of vertical semicircular canal integrity can be achieved performing Video Head Impulse Testing (vHIT). This test again assesses the VOR, but stimulation of all six semicircular canals is employed and the reflexive responses recorded.

vHIT is performed by utilizing an unpredictable, high velocity thrust of the head to determine whether the eye can maintain fixation on a target or whether the eye moves with the head and an eye movement back to the target is necessary to maintain fixation.[43] The video version of this test is based on the Halmagyi-Curthoys Head-Thrust test first described in 1988.[43] Essentially, the head is thrust rightward to stimulate the right lateral semicircular canal and the examiner makes an observation regarding the presence of a catch-up, or an overt saccade, suggesting an abnormal VOR.[43,44] Overt Saccades occur after the thrust to re-direct the eye to the target, are easily observable by the examiner, and often resolve following physiologic compensation has occurred. The same head-thrust stimulation is employed during video assessment; however the high speed cameras utilized to record the VOR are often capable of recording covert saccades. Covert saccades occur during the thrust to keep the eye on the target and are not observable by the examiner. The presence of covert saccades suggests a reduced VOR response on the stimulated side. [43,44,45]

vHIT expands the investigation of the peripheral vestibular system by stimulating the vertical semicircular canals and recording the response. To assess the left anterior canal and its' functional partner the right posterior canal, the patient turns the head 35 to 45 degrees to the right to put those canals in the correct plane for stimulation . [45] The head is then thrust in the pitch plane. The opposite is then employed for right anterior and left posterior canal stimulation. The presence or absence of catch-up saccades, overt and covert, are then recorded to determine whether there is reduced reactivity for one or more semicircular canals. [45]

Postural Control Studies

Postural control studies are an important part of the vestibular work-up. They provide information regarding functional balance ability by assessing the utilization of the sensory inputs and motor responses employed to maintain balance.[46,47] The information obtained from measurement of postural control can determine whether functional compensation has occurred in cases of peripheral vestibular involvement. It can also be useful for creating individualized vestibular rehabilitation and balance retraining therapy programs.

Postural control can be assessed as part of a bedside examination using low-tech, subjective measures such as the Clinical Test for Sensory Interaction and Balance (CTSIB). This assessment involves observation of the patient while they attempt to maintain their balance on firm and compliant surfaces and with eyes opened and closed. This subjective measure is sometimes utilized as a screening tool to determine if formal measures, such as Computerized Dynamic Posturography (CDP), are warranted to objectify the patient's functional balance ability. Computerized Dynamic Posturography (CDP) is a quantitative method for evaluating one's ability to maintain their balance during various conditions that simulate conditions potentially encountered during every day activities.[48,49] It is a dynamic test in which the patient stands on a computerized platform that measures forces exerted by the patient. Postural sway activity can be inferred from these measurements. [49] CDP has multiple components, which can provide insight into one's ability to use various senses, together and in isolation, to determine center of gravity and make the appropriate movements to preclude the center of gravity from exceeding the limits of stability. Additionally, CDP evaluates the timeliness of the motoric reactions, which occur in response to unexpected disruptions of equilibrium.

The sensory organization (SOT) test evaluates one's ability to use the visual, somatosensory and vestibular systems to maintain balance. As in everyday activities, all three systems are not always available to utilize for equilibrium purposes. CDP can provide information regarding how balance is influenced when one or more of these senses is absent or cannot be utilized.[48] Sensory information can sometimes be conflicting, requiring one to be adept at ignoring the inaccurate cues while making

use of the correct cues to maintain balance. CDP can provide insight into a patient's ability to sustain equilibrium despite inaccuracies in certain sensory information. The SOT consists of six conditions, during which selective manipulation of the somatosensory and/or visual cues is executed and an assessment is made regarding the ability to maintain balance in the absence of these cues.[48] The patient's performance for each condition is characterized by an equilibrium score, which is compared to age based normative data and represented graphically. The equilibrium scores are then compared for each of the six sensory conditions to quantify balance performance when using somatosensory cues, visual cues and vestibular cues without concomitant information from the other sensory modalities.

Condition one of the SOT is an eyes opened on a firm support surface scenario that is used as a baseline condition. Condition two also consists of a fixed, firm support surface allowing for somatosensory information; however the eyes are closed precluding access to visual information. The equilibrium score ratios are compared between these two conditions to quantify how the patient uses somatosensory information to maintain balance. When patients have difficulty taking advantage of somatosensory information effectively, perhaps because of decreased sensation in the feet or distal lower limbs, then a somatosensory pattern will be observed. Condition three involves evaluation of stability with the support surface fixed and the visual surround sway referenced, moving anteriorally and posteriorally based on the patient's movement. The equilibrium score of condition three is compared to that of condition two, where vision is absent as opposed to inaccurate. If there is a clinically significant difference with performance on condition three being poorer than two, then a visual preference is suggested. A visual preference indicates that the patient has difficulty maintaining balance when in the presence of orientationally inaccurate visual stimuli.[46,48,50] Condition four consists of a fixed visual field with a sway - referenced surface, in which the platform moves anterior or posterior based on the patient's sway. The equilibrium score obtained for this condition is compared to the baseline. Decreased performance during condition four, when somatosensory cues cannot be reliably used because they are inaccurate, suggests the inability to effectively use visual information to maintain balance. Condition five and condition six both evaluate how the patient makes use of vestibular information alone, to maintain balance. During both conditions the support surface is sway referenced precluding use of somatosensory input. During condition five the patient maintains balance with eyes closed, preventing the utilization of visual information. During condition six, the support surface continues to be swayed, however the visual surround also becomes sway referenced. Both eliminate the ability to use visual information because either vision is absent (condition 5) or vision is inaccurate (condition six). The only available sensory cue during these conditions is that from the vestibular system. A vestibular pattern is one in which increased sway is elicited during conditions five and six, when the patient cannot make use of sensory information from the visual and somatosensory systems. This can occur as a result of a peripheral vestibular abnormality evidenced by the other vestibular diagnostic tests, indicating that the abnormality has not been functionally compensated for. Performance on condition five and six provides insight into functional compensation status in cases of peripheral vestibular deficits.[48] Cases of bilateral vestibular paresis will result in abnormal performance on the vestibular conditions, commonly characterized as immediate free-falls when vision and somatosensory inputs are eliminated.

The Motor Control Test (MCT) is an assessment of a patient's reaction to unexpected disruptions of equilibrium. Sudden center of mass perturbations are generated by translating the platform either anterior or posterior to varying degrees and measuring the response time.[48] This evaluation provides information regarding the integrity of the long-loop pathway that begins with tendon and muscle stretch receptors in the area of the ankle. The information ascertained from these receptors are sent to the motor cortex where a response to maintain equilibrium is generated and then executed to preserve upright stance. [4, 51] The entire afferent and efferent neural pathways are assessed when measuring the long-loop automatic response. Weight distribution between legs, response strength and latency of response are obtained for the MCT.[2]

The patient is asked to maintain their balance while the platform on which they are standing slides backward for three movement sizes or translations. A backward translation displaces the center of gravity forward, relative to the base of support. The center of

gravity is then automatically re-centered over the base with a corrective movement backward [49] The time, in milliseconds, for this response to occur is measured and averaged for the three trials for each size translation. Separate response times are obtained for each leg. The MCT perturbations are then performed for forward translations. The expected response for this direction of movement is a backward displacement of the center of gravity and then an automatic, compensatory correction forward to return the center of gravity over the base of support. [49] Response latencies are calculated for both directions of movement, both legs and for all three translation magnitudes. Abnormally prolonged response latencies suggest an abnormality in the long-loop automatic response pathway. This abnormality can be the result of disruption of the afferent or efferent neural pathways, however further localization cannot be made based on the MCT alone.[53] Abnormalities on MCT often warrant additional work-up to determine causality of the response prolongation.

Tests of Otolith Function

Tests to evaluate the integrity of the otolith organs have remained elusive for multiple reasons. One challenge relates to technical parameters and equipment needed to stimulate the otolith organs for assessment purposes. Producing linear acceleration necessary to stimulate the otoliths can be difficult. Additionally, dysfunction affecting the otoliths is often compensated for quite quickly resulting in normal test findings despite an organic abnormality. [52,53] The otoliths are similar to the semicircular canals in that they act as vestibular receptors transmitting information regarding linear acceleration, head tilt and gravity. The otolith organs are stimulated by multiple axes of linear acceleration and changes of head position. [52] The utricles are predominantly sensitive to accelerations in the horizontal plane. The saccule is primarily sensitive to sagittal plane or up/down acceleration.[52] The displacement of the otolithic membranes because of angular acceleration of the head, results in increased neuronal firing on one side and decreased neuronal firing on the opposite side. This produces CNS stimulation and subsequent eye movement reflexes that preserve equilibrium. [55] Several diagnostic studies are utilized to evaluate the integrity of the otolithic reflex response.

Vestibular Evoked Myogenic Potentials (VEMPs)

Cervical VEMPS or cVEMPS assess the integrity of the saccule and inferior vestibular nerve by stimulating those mechanisms with sound and recording the response via surface electrodes.[54,55] This otolith-mediated, short-latency reflex is recorded from averaged sternocleidomastoid electromyography in response to intense auditory stimulation, presumably of the saccule.[56] The saccule responds to sound allowing for auditory stimuli to be utilized for stimulation purposes. The surface electrodes placed on the neck measure the interruption of the SCM contraction that occurs when the saccule is stimulated by sound. [57] This is an inhibitory response, thus the SCM needs to be contracted during the cVEMP acquisition. [58] This contraction can be achieved by instructing the patient to lift their head while in the supine position, effecting both sides, or by having the patient rotate their head away from the stimulated ear and contracting the SCM by raising the head or providing resistance to contract the muscle unilaterally. Electrodes placed on the neck record this inhibition of contraction yielding an excepted cVEMP waveform response.

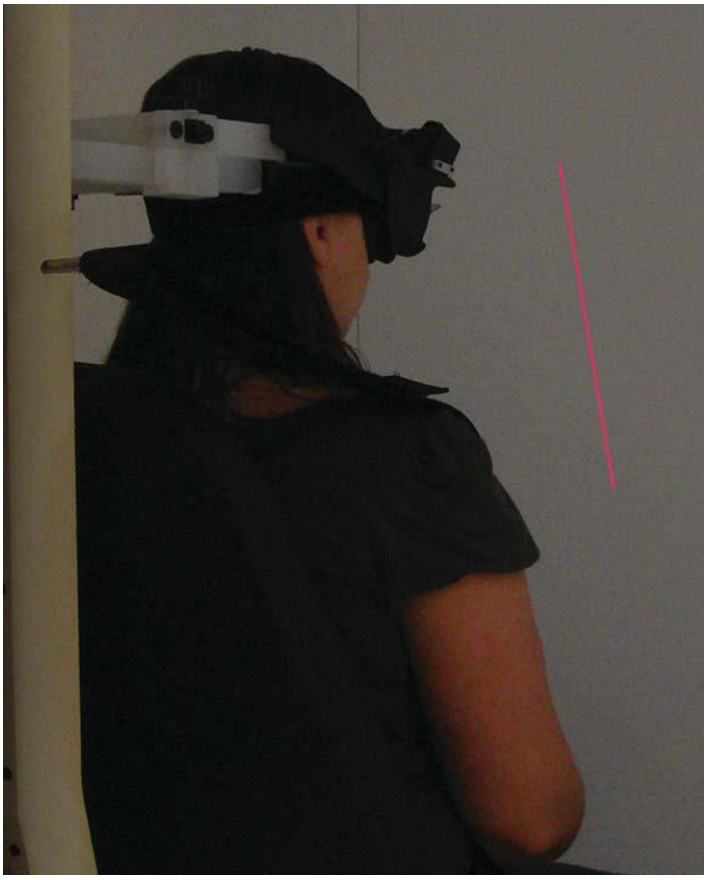
The cVEMP waveform can be analyzed in a multitude of ways using individual clinic protocols and established normative data. Analysis parameters include latency and amplitude of the response, threshold determination and asymmetry ratio. [54] Abnormal cVEMPs can be associated with a variety of vestibular disorders and it is important that each clinic establish utilization and interpretation parameters for cVEMPs. The most commonly utilized criteria for abnormality suggesting a vestibular disorder or otolith involvement is complete absence of a cVEMP response or amplitude asymmetries. [54] cVEMPs can be utilized in conjunction with other vestibular diagnostic tests to provide supplemental information. Assessment of cVEMP threshold is yet another test parameter used clinically. The intensity of the acoustic stimuli is decreased in an effort to determine the lowest intensity that a cVEMP can be elicited. cVEMPs elicited at abnormally low intensities have been correlated with superior semicircular canal (SSC) dehiscence. SSC dehiscence is a thinning or absence of the temporal bone between the apex of the superior semicircular canal and the middle cranial fossa. [63]

Evoked potentials related to the VOR are also utilized as another means to assess otolith integrity. This

diagnostic study is referred to as an ocular VEMP (oVEMP). The electrodes utilized for recording purposes are placed around the eyes to record extraocular muscle activity. Similar to cVEMPs, intense auditory stimulation is utilized to elicit or change muscle activity, in this case ocular muscles. The sound activates the vestibular afferents and evokes a short latency potential from the recorded eye movements. The response is not inhibitory and ipsilateral as with cVEMPs, but is excitatory and occurs predominantly contralateral to the auditory stimulation.[59] oVEMP test parameters are similar to cVEMPs in that absent responses can suggest vestibulopathy related to the otoliths and abnormally low response thresholds can suggest the presence of SSC dehiscence.[60] The oVEMP may complement the cVEMP by providing a comprehensive evaluation of the VOR pathways to the extraocular muscles.

Subjective Visual Vertical (SVV) testing is a measurement of primarily utricular function, in which the subject's perception of vertical and actual, true vertical are compared. The SVV can be measured with the patient in a stationary position or during various types of rotation.[61] During static SVV testing the patient is seated in front of an illuminated, adjustable line, with other cues eliminated by darkness or by a cue free background. The subject is asked to set the line to what they perceive as true vertical. Normal subjects can effectively perform this task within one to two degrees of actual verticality.[62] When patients are unable to accurately adjust the line to a vertical position, an otolith abnormality may be suggested.[52] Conversely, normal SVV does not rule out otolith or labyrinthine involvement because physiologic compensation may have occurred resulting in normal SVV despite the fact that a vestibular abnormality exists.[64] Therefore SVV in a static position may be most sensitive to acute lesions of the otoliths, prior to physiologic compensation completion.

The sensitivity of SVV testing may be increased with the addition of concurrent rotational stimulation. On-Axis Rotation consists of rotating an individual around the vertical axis at constant velocity to stimulate the otolith organs.[65] This type of rotation creates a centrifugal linear acceleration, bilateral stimulation that activates both utricles simultaneously in a way that they are exposed to equal and opposite centrifugal force. This equal and opposite stimulation should result in cancellation of the stimulus, and hence no perception of tilt, resulting in the ability to judge vertical accurately.[65,66] Similar to other rotational tests, this testing does not allow for abnormality lateralization. When test equipment allows, an attempt to further lateralize utricular dysfunction and / or identify more chronic, physiologically compensated disorders of the otoliths can be made by incorporating unilateral centrifugation into the SVV assessment. This rotation consists of rotating at a constant, high velocity with the test ear positioned off-axis and the non-test ear positioned on-axis.[67] With this rotation parameter, the VOR of the horizontal semicircular canals lessens, and the off-axis rotation creates a centrifugal force stimulating the utricle that is in the off-axis position only. [65] The result should be a SVV tilt in the opposite direction of the rotational tilt. The SVV should be symmetric for both directions of stimulation. When this response symmetry is not achieved, then utricle dysfunction on the side with less SVV tilt is suggested. [65]



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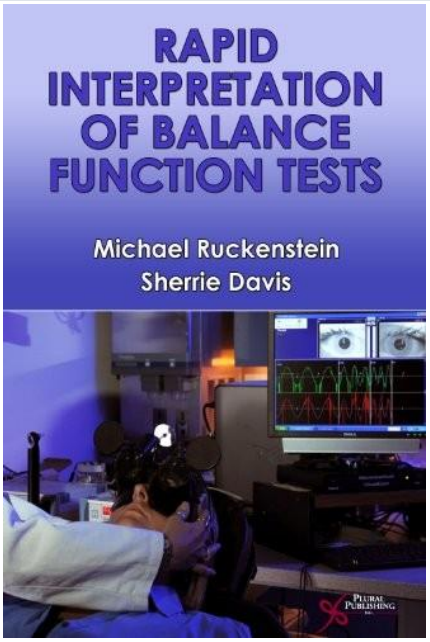

Interpretation and Usefulness of Computerized Dynamic Posturography

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The following article is a modification of Chapter 19, Neil T Shepard, Ph.D. from "Assessment and Management of the Balance Disorder Patient", 2nd edition, Jacobson & Shepard (eds), Plural Publishing 2015.

Introduction

The focus of this article is the clinical utility of the tests of postural control and their clinical interpretations. This is provided in a series of patient case studies that present the use of computerized dynamic posturography (CDP) within the construct of the patient's signs and symptoms represented by the patient history and results from the other clinical evaluations of VNG, rotational chair, and otolith function testing. The presentation of the material is provided in this manner as the principal use of CDP is not in isolation.

Clinical Utilization

When considering the clinical utility of formal postural control assessment, the primary discussion becomes focused on when specific tests should be performed and the reliability and validity of the tests.

Staged Protocol

In addressing when the tests should be performed, one can make the argument that some level of assessment should be used on all patients complaining of dizziness even if imbalance or falls are not part of the principal symptoms. This argument is supported by the increased likelihood of a fall at all ages with the identification of peripheral vestibular involvement (Herdman, Blatt, Schubert, & Tusa, 2000). Further support is the evidence that if identified as being at risk for a fall, even if no falls have yet occurred, the use of vestibular and balance rehabilitation therapy can reduce the risk of falls and specific programs have been successful in reducing the rate of falls with young and older populations (Gillespie et al., 2007; Hall, Schubert, & Herdman, 2004; McClure et al., 2007). It has been shown that tests other than those dealing with postural control can also predict a falls risk. These involve the use of functional evaluations of gaze stability (i.e., dynamic visual acuity and the gaze stabilization test) (Honaker & Shepard, 2012; Honaker, Lee, & Shepard, 2013). Within this context it can be questioned as to whether all patients require a full formal postural control analysis. Given the ability to make a reasonable prediction as to whether the sensory organization test (SOT) will be abnormal by first performing the clinical test of sensory interaction on balance (CTSIB or modified CTSIB) (el-Kashlan, Shepard, Asher, Smith-Wheelock, & Telian, 1998; Shumway-Cook & Horak, 1986; Wrisley & Whitney, 2004) a staged protocol can be used. By clinical experience only and not through an experimental clinical trial study, the author finds the motor control test (MCT) assistive in helping to interpret complex patterns of abnormality that can occur on the SOT and therefore recommends its use (if available) whenever formal SOT is performed.

As an example of the staged protocol concept, one used by the author is provided. The criteria were developed based on (1) a study comparing the CTSIB to SOT (el-Kashlan et al., 1998); (2) a large retrospective study of findings in over 2,000 patients when all tests were used on all patients (Shepard & Telian, 1996); and (3) a prospective study on false-positive findings of the MCT (Shepard, 2000). Indications for when to use SOT and MCT (both performed together) are given below. These criteria are applied in a parallel loose format such that if any one of the criteria is met, the patient goes on for a full postural control evaluation. SOT and MCT are indicated by the following:

1. Abnormal performance on the modified CTSIB. Normative data across age exist for this study performed in a semiqualitative manner or via the use of a fixed forceplate (el-Kashlan et al., 1998; Rose & Clark, 2000; Shumway-Cook & Horak, 1986; Weber & Cass, 1993).

2. A major complaint of symptoms of unsteadiness or imbalance in standing and/or walking (constant or episodic) in the absence of vertigo at the same time.
3. Known or suggested pathologic involvement of the pyramidal/extrapyramidal tracks, involvement in spinal tracks, or suggestion of sensory and/or motor neuropathy via the patient's presenting symptoms or past medical history.

Validity and Reliability

Validity of the SOT and MCT protocols has been partially approached in a study comparing normal young and older adult's performance on the clinical protocols to performance in a basic laboratory situation using an optoelectric, two-dimensional, three camera, motion analysis system (Shepard et al., 1993). The findings of this work showed that the impressions of hip versus ankle-dominant strategies and the ability to predict the movement of the upper body and whole body sway from the forceplate data were consistent with the detailed analysis used under similar conditions with the motion analysis system.

In the desire to use serial sensory organization testing to monitor patient progress, test-retest reliability becomes important. Studies looking at this issue in normal volunteers retested on different days had mixed results, some suggesting no learning effect with test repetition (Black, Pabski, Reschke, Calkins, & Shupert, 1993; Kubo & Wall, 1990) and another giving results implying that a learning effect is present (Ford-Smith, Wyman, Elswick, Fernandez, & Newton, 1995). An additional study investigating the test-retest reliability when testing was repeated within the same day on patients (Shepard & Boismier, 1992) suggested that a learning effect is present. In the study where interclass correlation coefficients (ICCs) were used as part of the outcome evaluation (Ford-Smith et al., 1995), the values for all six conditions were found to be less than the recommended value for routine clinical use (Portney & Watkins, 1993). Findings of this nature should not be interpreted as rendering the equipment inappropriate for serial monitoring. Given the novel nature of the task at hand, it would be surprising if there were not some minimal learning effect. In the work using the ICC statistic, the magnitude of the improvement suggesting a learning effect was within the known range for the variance given in the normative data used to develop clinical cutoff criteria for abnormal performance. Therefore, although a learning effect likely does exist for normal subjects, the magnitude of the effect is relatively small which introduces the issue of its clinical relevance.

Although the work on normal subjects is important in studying reliability, it is also important to know if the same result occurs in patients who start with abnormal performance. Clinical experience suggests that patients have an increased likelihood for improvement on a second test administration if they meet either of the following conditions: (1) show a pattern of improving performance across three trials of a given condition; or (2) suspect an unreliable test result secondary to significant anxiety noted by the examiner. A pilot study investigating this issue prospectively showed that greater than 50% of the patients meeting one or both of the above criteria changed to a normal pattern with repeat testing on the same day (Boismier & Shepard, 1991). To study this issue in the average balance disorder patient not suspected for retest improvement, a prospective, random, 20% sample of 650 consecutive patients were subjected to repeat dynamic posturography within 120 min of the original testing (Shepard & Boismier, 1992). The study did demonstrate statistically significant improvement in sensory organization test scores for several of the test conditions and for the overall composite score, suggesting a learning effect. However, only 10% of those with abnormal sensory organization test composite scores initially changed to a normal score with repeat testing. All of those changing to a normal score were initially outside the normal range by an amount less than the variance in normal subjects for the conditions in question. Therefore, our current clinical protocol suggests repeat testing of an abnormal sensory organization test on patients only if they meet one of the following three conditions:

- The patient shows a pattern of improving performance across three trials of a given condition.
  The test result is suspected to be unreliable secondary to significant anxiety noted by the examiner. (Typically this would involve a pattern that appears functional with improved performance on the more difficult tests. This is discussed further below.)

- The composite score is outside the normal range by 20 or less points.

The same concerns about test-retest reliability and validity are discussed elsewhere and are only summarized herein (Shepard, 2000; Shepard & Telian, 1996). To consider the MCT results being a reliable indication of abnormality, the following three conditions need to be considered:

- The latencies following the medium translation must be longer than those for the large translation, unless the latencies for both responses are greater than 190 ms.
- If the latencies are abnormal by 50 ms or less, the condition must be repeated and the results replicated. If the latencies remain in the abnormal range, the data can be considered abnormal. If the latencies fall within the normal limits on the repeat test, then the results should be considered as normal findings.
- Complaints of pain in the lower back, lower limb, or hip joints are a likely source of abnormality in latency on the backward translation studies.

Volitional Reduction in Limits of Stability

The equilibrium score of the SOT is a percentage representing the magnitude of sway in the sagittal plane for each trial of each condition. This score is based on a normal value of 12.5 degrees of anterior/posterior sway about the ankle joint, typically 8 degrees forward and 4.5 degrees backward. It is assumed that this range of sway is available to all patients during the test. Some patients may not have this normal range because of physical restrictions at the ankle, or because of limits the patient has adopted secondary to his or her sense of imbalance and fear of a potential fall. Recognizing the patient who has a reduction in his or her volitional limits of sway can be helpful in the interpretation of the SOT results and may be able to be addressed with a vestibular and balance rehabilitation program. Therefore, testing the limits of volitional limits of sway for each patient as part of the sensory organization test protocol can provide useful additional information.

Prior to testing under condition 1, patients are asked to lean as far forward onto their toes as possible without taking a step or reaching out, and then as far back on their heels as possible. This is done with the eyes open, stressing that the movements are to be done about the ankle joint rather than bending at the hip. Following this practice trial of allowing the patient to explore the limits of sway, the equipment is activated under condition 1. Rather than maintaining stable stance at first, the patient is asked to repeat the limits of sway task again during the 20-s trial. When completed, the equilibrium score on the screen can be interpreted as the percent reduction in limits of sway in the sagittal plane. A score of 35% or less is interpreted as no significant reduction, knowing that the range of movement only increases if repeated practice is allowed. Then, the actual condition 1 test is repeated, so that the limits of sway test results do not enter into the calculation of the cumulative equilibrium score at the end of the test. An example of the use of this procedure to help explain an inconsistent test result is given in Figure 1. The SOT results shown are from a patient with severe bilateral vestibular hypofunction. His average performances (the numeric average of the three trials) for conditions 1 through 3 are well within normal limits. This is also reflected in the raw data shown in the bottom portion of the slide with little or no calculated center-of-mass (COM) sway on any of the trials for conditions 1 through 3. On condition 4 in the summary plot giving the equilibrium score, he shows a fall reaction on trial 1, performs significantly better on trial 2, yet has another fall reaction on trial 3. Both conditions 5 and 6 show repeated fall reactions (indicated as free falls from inspection of the raw COM trace—no attempt to correct the falls) on all three trials of each condition. From the COM traces for condition 4 it is seen that he starts with a free fall on trial 1, maintains stance with increased sway for the entire 20 s on trial 2, and has a fall reaction within the last 4 s of trial 3. In testing his reduction in volitional limits of sway his result was 55%. Therefore, the 0 or fall line on the equilibrium score plot, it could be argued, should be at 55% equilibrium score (position of the dashed black line) as that represents his range of anterior-posterior movement. He has no physical limitations in range of motion or strength that would explain the limited range of motion. It is likely an artificial limit that makes him feel secure during stance. Given this reduction in limits of sway, it is easy to explain the apparent inconsistency in performance on condition 4. As his sway magnitude is

so close to his perceived limits of movement and when those limits are exceeded, he simply takes a step to prevent what he perceives as an impending fall risk. His performance on conditions 5 and 6 would be expected and explainable given the severity of his bilateral hypofunction. Yet, the hypofunction would not provide an explanation for the performance on condition 4. He was treated with a vestibular and balance rehabilitation program with no change in performance on conditions 5 and 6 (not unexpected given the bilateral loss) but within 3 weeks of the onset of the program condition 4, well within normal limits on all three trials, and his reduction in volitional limits of sway was reduced to 35%.

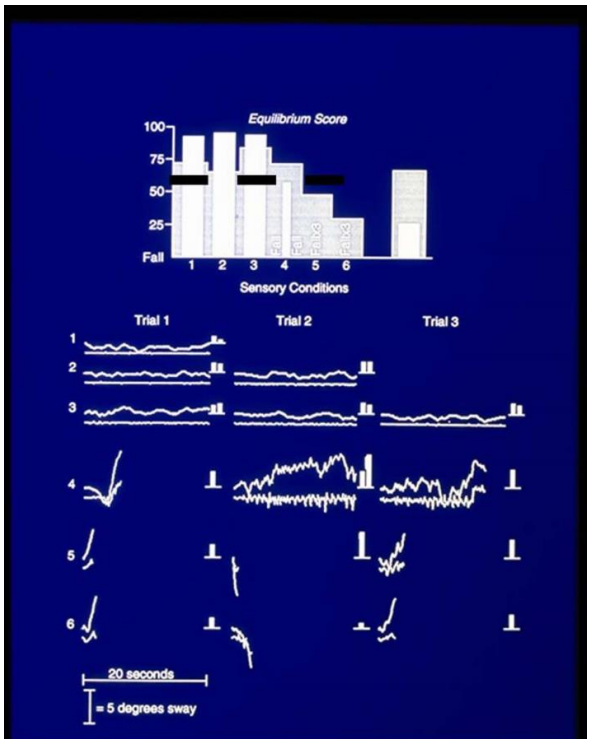


Figure 1. The SOT EquiTest summary of the equilibrium scores and the corresponding raw data results for each of the trials for each of the six SOT conditions tested. For conditions 1 through 3, the single bar on the Equilibrium Score graph represents the average of the three individual trials. For conditions 4 through 6 the individual results for each trial are shown as a fall reaction (word Fall) or the equilibrium score by the single bar on condition 4. In the lower section of the figure the thicker line represents the calculated COM anterior/ posterior excursion over the 20-s trials. The thinner line represents the force activity on the shear force transducer sensing horizontal force applied to the dual platforms. See text for further explanations of the figure.

An additional measurement that may also be important in explaining results that appear to be inconsistent is that of the average position of the center of mass during each trial. Normal distribution would have the weight positioned 2 to 3 degrees in front of the ankle joint. The interactive display can give the examiner an indication as to whether the patient appears to be standing with his or her weight distributed significantly over the heel of the foot. When this situation is noted during condition 1 testing, correct patient foot placement and a normal comfortable posture should be confirmed prior to continuing. If the COM continues positioned far to the rear with correct foot position and the patient reporting what he or she perceives as his or her normal posture, the range of motion in the posterior direction is severely limited. Therefore, if they start to sway backward from their neutral position, they are very likely to have to quickly take a step or make another fall reaction to prevent further posterior sway. This type of performance can result in an appearance of inconsistency, especially on the more difficult conditions 4 through 6, though they may be able to maintain their COM in their neutral or a forward position on one of the trials within normal limits, but on another trial a slight sway backward causes the fall reaction.

Determination of Exaggerated (Aphysiologic) Performance

Dynamic posturography is useful for identification of patients who may be, for whatever reason, exaggerating their condition. Works by several investigators (not all with EquiTest) have attempted to quantify the use of this tool to identify these patients and list qualitative factors that would raise questions in this dimension (Allum, Huwiler, & Honegger, 1994; Cevette, Puetz, Marion, Wertz, & Muenter, 1995; Goebel et al., 1997). Among the most common factors is the improvement of performance as the person being tested proceeds from condition 1 through condition 6. In a situation of this nature the equilibrium score is outside the normative range for the age of the patient on the easier conditions (1 through 3), yet as the task significantly increases in difficulty the performance returns to normal or near normal. Another common feature is that of a regular sway pattern seen in the raw COM traces. This typically is sinusoidal in nature at a closely maintained frequency across the conditions with only the amplitude of the sway varying. Both of these conditions are indications of influences beyond a peripheral or central vestibular system lesion. However, the term *aphysiologic* may be inappropriate as some of these features are seen regularly in patients with anxiety disorders. Therefore, these types of performance may be a physiologic reaction to the anxiety disorder.

Cases

The remainder of this article presents a group of cases that illustrate many of the interpretation points and clinical uses of dynamic posturography together with the other studies including the office exam, ENG/VNG, and rotational chair testing. It is important for the clinician to understand the interaction of the tests on the final interpretation of the laboratory studies in the context of the patient's presenting signs and symptoms for final determination of the patient's condition. The majority of these cases are presented here, with permission from Shepard (2007).

Case 1

Case 1 is a male, who was 35 years of age, seen for complaints of sudden-onset vertigo, 6 months prior, with nausea and vomiting in a crisis event (acute vestibular syndrome) with continuous symptoms lasting 3 days, steadily showing slow improvement and no accompanying auditory symptoms. The continuous vertigo resolved into head movement provoked spells of light-headedness with imbalance and occasional vertigo lasting seconds to a minute after a movement. All planes of motion were provocative. Symptoms had continued to improve but still occurred on an infrequent daily basis. He presented with no neurologic focal complaints and past medical history was noncontributory. Audiometric evaluation was completely normal bilaterally as was his contrasted magnetic resonance imaging (MRI) study of the head. His detailed vestibular examination both with and without visual fixation was remarkable for a positive head thrust test to the left, right-beating post-head-shake nystagmus, and spontaneous right-beating nystagmus with visual fixation removed. The full neurologic and ocular motor components of the examination together with Hallpike testing were normal. His CTSIB was well within normal limits, and together with his history formal postural control testing was not needed in this case. The history with the examination was strongly suggestive of uncompensated left peripheral vestibular hypofunction, secondary to vestibular neuritis. Laboratory vestibular function testing revealed spontaneous right-beating nystagmus with visual fixation removed and a 76% left reduced vestibular response with ocular motor testing and postural control assessment normal. In this case the tests were, as is typical in most cases, confirmatory of the clinic suspicions from the history and direct examination. Management decisions made at the time of the office visit to initiate treatment with vestibular and balance rehabilitation therapy (VBRT) and to discontinue vestibular suppressive medication were not in any manner altered with obtaining the laboratory findings. The vestibular function and balance tests were well justified given the length of symptoms and the fact that the testing has better sensitivity for some ocular motor findings than the direct examination, specifically saccade velocity testing and quantification of smooth pursuit. Sensitivity to mild peripheral vestibular function asymmetry is also better with the laboratory testing. In this case the magnitude of the peripheral asymmetry made it detectable by both the direct examination and the caloric irrigation studies. This patient's vestibular rehabilitation program consisted primarily of exercises focused on improvement of the vestibulo-ocular reflex (VOR exercises, X1 and X2) and habituation exercises to reduce symptom production with head movements in the horizontal and vertical planes. No specific balance or gait activities were needed for this patient; however, he was put on a general walking program with casual head movements to increase his overall level of activity. Given that the primary reason for his lack of compensation was avoidance of head movements, he responded rapidly to the use of the rehabilitation program becoming virtually asymptomatic with a 6- to 8-week interval.

Case 2

A 31-year-old male presented with onset of head motion-provoked vertigo with more or less constant imbalance with standing and walking. He denied any vestibular crisis event or auditory complaints. His symptoms were more concentrated in sagittal plane movement and when rolling left or right from a supine position. These symptoms had been ongoing for several years with intervals when the vertigo was resolved and the imbalance was reduced but not absent. He reported an MRI from several years prior to this evaluation that was normal with a cervical MRI positive for mild disk abnormalities. Audiologic examination was normal. Other than the development of mild paresthesia of the right hand and arm over the last year, he had no other neurologic complaints and his past medical history was noncontributory. His direct office examination was remarkable for anterior semicircular canal benign paroxysmal positional vertigo (BPPV) canalithiasis, and inability to maintain quiet stance on foam with his eyes

closed during the CTSIB. The remainder of the examination was normal. He was treated in the office with a canalith repositioning procedure and referred for a formal VBRT program. Secondary to the length of time of the symptoms and the complaints of persistent imbalance (although this is a common report with BPPV), vestibular and balance function testing was requested. The laboratory studies continued to show anterior canal BPPV with no other indications of peripheral vestibular system involvement. Pursuit tracking tests were normal, but saccade testing was positive for mild right internuclear ophthalmoplegia (INO). Postural control abnormalities were consistent with that seen in demyelinating disorders, increased latency on the MCT test. The MCT test abnormality, although nonspecific to the cause of his symptoms was completely unexpected and difficult to explain on the basis of BPPV alone. However, given the ocular motor results, the prolonged latencies to active recovery fit with the overall suspicion of central nervous system involvement. His SOT results showed an increase in sway under conditions 5 and 6 without fall reactions. The SOT results are nonspecific to the disorder underlying the condition but reflective of his functional ability to maintain quiet stance when challenged. The SOT results simply reflect the fact that he was having difficulty using vestibular system information when visual and proprioceptive/ somatosensory cues were absent or disrupted. Secondary to these findings and his report of paresthesia starting in the left foot a new MRI was obtained that showed multiple hyperintense spots throughout the brainstem region. He was referred on to neurology and is being followed with a diagnosis of probable multiple sclerosis with BPPV. Unlike Case 1, the management of this case was driven strongly by the results of the ocular motor and collective results of the dynamic postural control tests. The test results revealed abnormalities too subtle to be detected in a direct examination, the ocular motor abnormality of INO. Although the CTSIB and the SOT results were consistent, they were nonspecific and could have realistically been caused by the ongoing BPPV. It was the MCT test that reinforced the ocular motor findings with strong presumptive diagnosis of MS. This is an exception to the impact that the testing has on a more routine basis in the decisions regarding management of the dizzy patient where confirmation is the more common roll of the laboratory testing of the dizzy patient.

Case 3

A 44-year-old female presented with spontaneous spells of light-headedness and imbalance lasting hours. Frequency of occurrence was 1 per every 8 weeks but increasing. Typically, she would return to her normal baseline between events. The core studies involving ENG and ocular motor tests were normal. Her CTSIB was normal, but as her history was that of unsteadiness, episodic without vertigo, full SOT/ MCT were indicated. The results of these evaluations are given in Figure 2. This figure demonstrates normal SOT with significantly prolonged latencies on the MCT. Secondary to the prolonged latency for the forward support surface translation, the protocol indicated the use of Postural Evoked Responses (PER). These are surface recordings of select muscles of the lower limbs. Results of the PER are given in Figure 3. This demonstrates a pattern of absent middle latency component and, when taken with the abnormally long MCT latencies for both limbs, is suggestive of possible demyelinating disease. Therefore, this patient's care was directed to neurology from otolaryngology, and she was followed for possible demyelinating disease as a result of the test findings guiding the management.

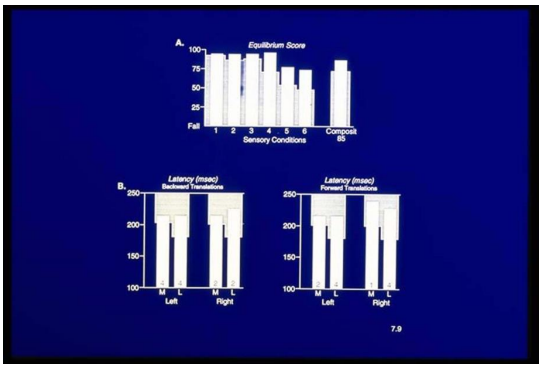


Figure 2. SOT and MCT results, respectively, for the patient in Case 3.

Figure 3. Postural-evoked response (PER) study for Case 3 is shown. CH1 and CH3 show the response for the gastrocnemius of the left and right legs, respectively, with CH2 and CH4 showing the response from the tibialis anterior from the left and right legs, respectively. *SL1*—onset time for the short latency response; *SL2*—offset time for the short latency response; *LL1*—onset time for the long latency response; and *LL2*—offset time for the long latency response. Latencies for onset and offset times are given in the grids at the right of each trace in milliseconds. The grids also provide for the absolute amplitude of the trace at the time of the onset and offset marks and peak amplitude of the short, medium, and long responses in microvolts. Integrated amplitude (IEMG) is given in microvolt-seconds for each response. The trace in the panel at the top left shows the timing of the toe-up rotation of the support surface. Zero time indicates the time of actual start of the platform movement. Note the striking absence of a medium latency response bilaterally.

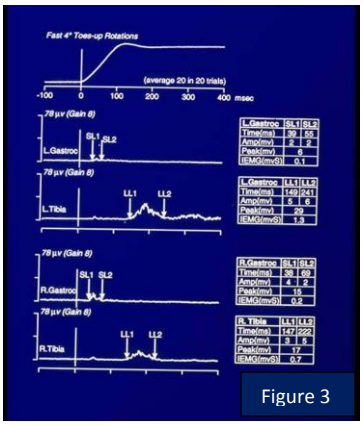


Figure 3

Case 4

A 70-year-old male reported with a working diagnosis of right-side Ménière's disease. Laboratory testing was to be used to establish a baseline against which to compare for monitoring his disorder and possible treatment. His history was classic with regard to Ménière's disease with spontaneous spells of true vertigo with nausea and emesis production lasting 1 to 4 hour spells had been ongoing for a year beginning with one event every 2 months increasing in frequency to one to two times per week at the time of his evaluation. Conservative treatment with a low-sodium diet and diuretic were being used with no effect. He reported fluctuant hearing on the right with bilateral tinnitus and significant past noise exposure (Figure 4). Between the events he was free of dizziness symptoms. He did admit to increasing falls with his events but not between. The remainder of his past medical/surgical history was noncontributory. Results from his VNG showed ocular motor findings that were normal or consistent with his age. Spontaneous right-beating nystagmus with a slow component velocity of 1 to 3 deg/s was noted in sitting with fixation removed. No exacerbation with head-shake testing and no positional nystagmus were seen. His caloric irrigation test revealed a surprising bilateral reduction with warm, cool, and ice water irrigations producing nystagmus with slow component velocity less than 4 deg/s for both right and left stimulations. The immediate question that required an answer was what was the degree of his bilateral involvement? If significant, it could limit more aggressive treatment options.

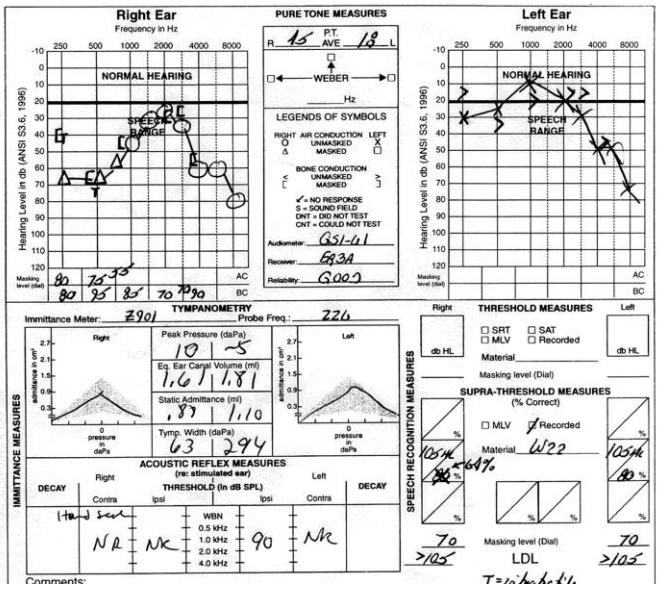


Figure 4. Audiometric results for the 70-year-old male in Case 4.

To attempt an answer to the issue of the degree of bilateral involvement rotational chair, the SOT of dynamic posturography and dynamic visual acuity testing (Herdman et al., 1998; Hillman, Bloomberg, McDonald, & Cohen, 1999; Peters & Bloomberg, 2005) were combined to provide a collective estimate of involvement. Rotational chair results to sinusoidal harmonic acceleration testing and the SOT results of posturography are given in Figures 5 and 6, respectively.

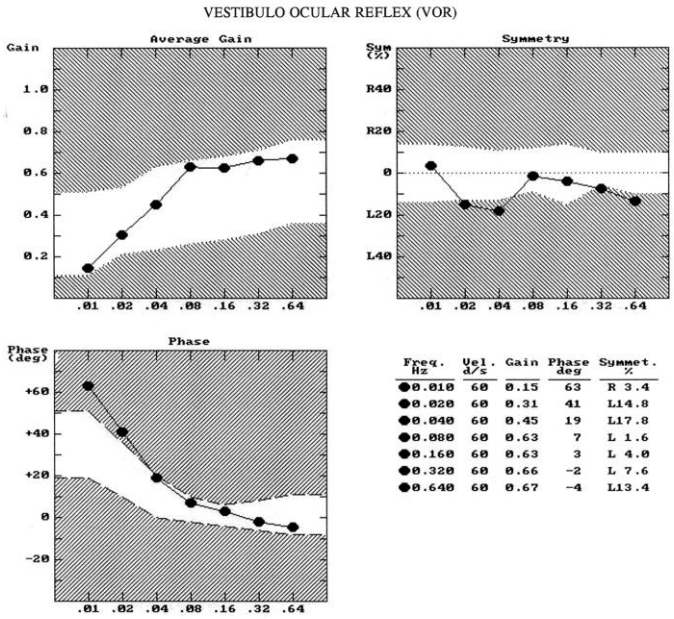


Figure 5. Rotational chair results from the sinusoidal harmonic acceleration test for the male in Case 4. The graph in the upper left shows overall gain values of the slow component velocity of the eye referenced to the velocity of the head as a function of test frequency. The graph in the upper right provides the percentage difference in the average strength of the slow component eye velocity stimulated during rotations to the right versus rotations to the left as a function of frequency. At several frequencies the slow component velocities to the left (produced by rotation to the right) were significantly greater then the slow component eye velocities to the right (produced by rotation to the left). The resulting asymmetry could therefore be a result of a left paretic horizontal canal system or an irritative right horizontal peripheral system (an irritative status is not unusual in Ménière's disease). The graphical result in the lower left of the figure presents the phase angle (timing relationship of eye velocity versus head velocity) as a function of test frequency. This graph shows phase angle to be within normal limits progressing to an abnormal phase angle lead as the test frequency is lowered to 0.01 Hz. The table in the figure provides for the numerical results of gain, phase angle, and asymmetry at each of the frequencies tested.

In summary, the chair results demonstrated an abnormally high phase lead in the lower frequencies with a left greater than right slow component velocity asymmetry. These findings, given the negative ocular motor testing, would suggest peripheral involvement of

either a left paretic or right irritative style lesion. Given his spontaneous right-beating nystagmus and documented asymmetric hearing loss worse on the right, the right irritative lesion would be considered more likely. Of importance were the overall gain values within normal limits, although trending to the lower limit of normal as the test frequencies approached 0.01 Hz. This suggested that the extent of the bilateral involvement was minimal and restricted to the very low-frequency region of the peripheral system. The magnitude of the phase lead at 0.01 Hz was also supportive of this impression. If this impression from rotary chair is correct, then the functional impact of his bilateral involvement should also be minimal regarding maintenance of quiet upright stance and his ability to maintain visual clarity with his head in motion.

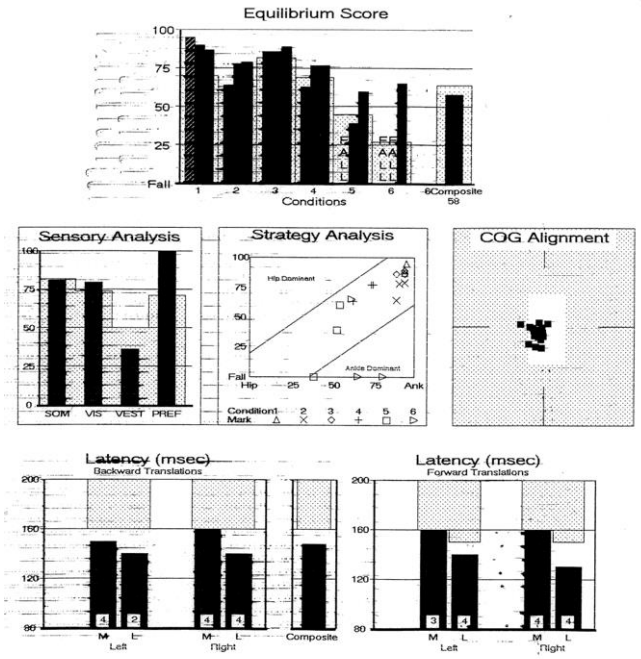


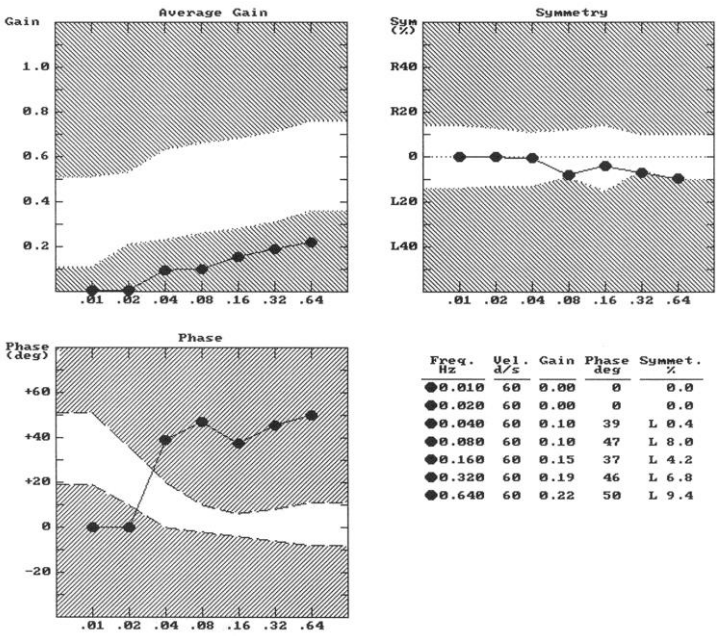
Figure 6. The graphical summary of the SOT and MCT tests for the gentleman in Case 4. The six conditions of the SOT test are shown in the graph at the top of the figure. This shows performance on conditions 1 through 4 to be within a normal range given the patient's age. Conditions 5 and 6 demonstrate fall reactions on the first trial with performance normal and improving on trials 2 and 3 for condition 5 and normal on trial 3 for condition 6. The graphs at the bottom of the figure demonstrate normal MCT findings for posterior (graph on the left) and anterior (graph on the right) translations of the surface on which the patient was standing.

The results of the SOT shown in Figure 6, although showing difficulty when he was forced to rely on vestibular system cues alone, demonstrates his ability with practice to maintain stance within a normal range for his age by the second or third trial of test conditions 5 and 6. This SOT result would be consistent functionally with minimal bilateral vestibular involvement. Last, the Dynamic Visual Acuity (DVA) test performed using the clinical office technique with horizontal reciprocal head movements at 2 Hz was within normal limits. Overall, the collective results of the laboratory studies demonstrated peripheral vestibular system involvement bilaterally, but were mild in degree and restricted to the very low-frequency region of function of the peripheral system with greater involvement on the right than the left. These findings including the SOT and DVA provided a firm baseline physiologically and functionally for monitoring of the patient's slowly titrated transtympanic gentamicin treatment for his right-side Ménière's disease. This was successful in stopping the spontaneous events without causing him to experience any further functional deficits of significance related to his bilateral peripheral system involvement.

Case 5

This 55-year-old female was referred for evaluation with complaints of oscillopsia and imbalance with ambulation especially in darkened environments or on walking surfaces that were uneven or soft. Her history was that of diagnosis of non-Hodgkin's lymphoma and started on chemotherapeutic agents including cisplatin 1.5 years prior to her laboratory evaluation. Shortly after starting treatment she experienced a vestibular crisis event (acute vestibular syndrome) with sudden onset true vertigo, nausea, and vomiting without hearing change. Symptoms were continuous over a 3-day interval, improving into head movement–provoked symptoms and resolving completely within 2 weeks. She was without dizziness until 1 year later when she experienced another vestibular crisis event, equal in intensity to the first and again without auditory symptoms. This began after a second chemotherapy treatment with cisplatin. This event had a similar time course to the first crisis event; however, she developed left-side benign paroxysmal positional vertigo (BPPV) of the posterior semicircular canal that finally responded to treatment maneuvers and resolved after 1 month. Since the resolution of the BPPV, she has had the complaints of oscillopsia and imbalance that she presented with for evaluation. The physicians working with the patient strongly suspected that she was now a bilateral

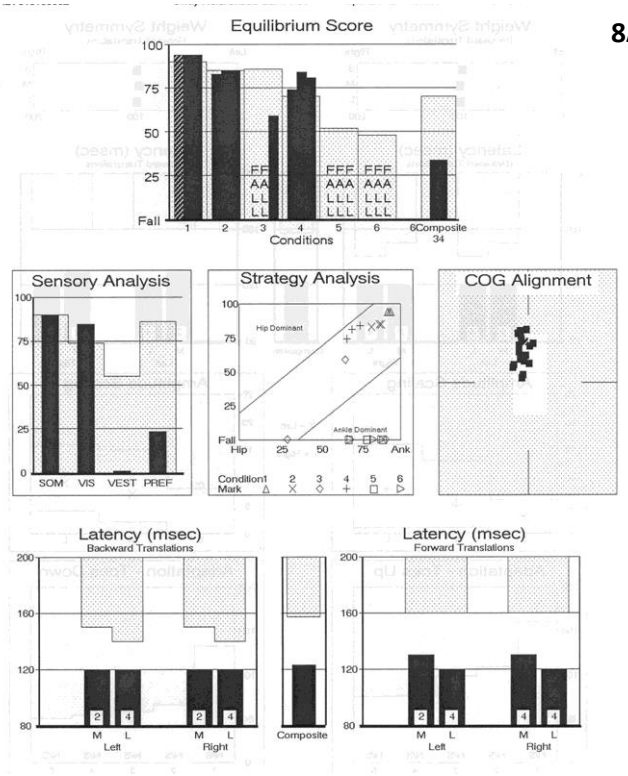
peripheral paresis patient but wanted to know if there was any evidence that could suggest whether this was as a result of the use of the cisplatin. Her history would suggest otherwise given the two sequential crisis events, but ototoxic drugs have been known to produce other than symmetric effects on the vestibular system. Of importance in the history is the identification of left-side BPPV after the second crisis event. This verified report tells us that the posterior semicircular canal on the left was indeed functioning normally as to the neuro-ephithelial tissues but had abnormal mechanical reaction to changes in a gravitational field. The possibility of differential damage to a labyrinth would not be the pattern of damage expected if caused by cisplatin. The laboratory challenge was to objectively investigate this issue. The VNG study demonstrated normal ocular motor results; no spontaneous, hyperventilation, or post-head-shake nystagmus; but clinically significant left-beating positional nystagmus with slow component velocity ranging from 3 to 6 deg/s was noted. Caloric irrigation with warm, cool, and ice water produced right- and left-side responses less than 4 deg/s. Formal hearing evaluation was well within normal limits through 8 kHz bilaterally. Her rotational chair and SOT/MCT findings are given in Figures 7 and 8A–B.



Freq. Hz	Vel. d/s	Gain	Phase deg	Symmet. %
0.010	60	0.00	0	0.0
0.020	60	0.00	0	0.0
0.040	60	0.10	39	L 0.4
0.080	60	0.10	47	L 8.0
0.160	60	0.15	37	L 4.2
0.320	60	0.19	46	L 6.8
0.640	60	0.22	50	L 9.4

Figure 7. Shown are the rotational chair results for Case 5. Details of the figure are the same as given in Figure 4.

8A.



8B.

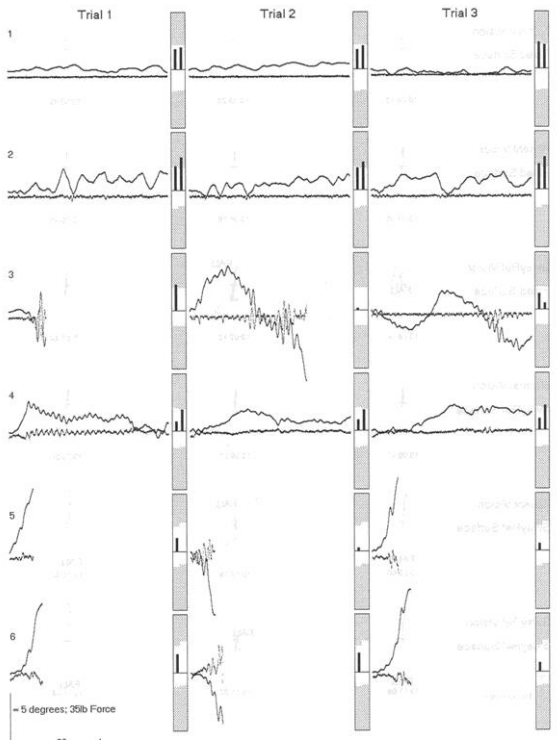
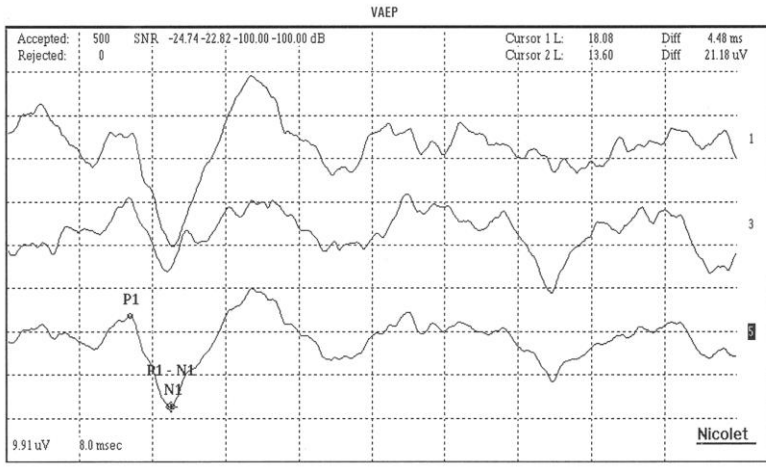


Figure 8. A. For Case 5 results of the sensory organization test and motor control tests are given. **B.** The raw data tracings obtained during the sensory organization test. See the text for explanation of the findings

In contrast to those of Case 4, these findings show none to minimal vestibulo-ocular reflex (VOR) responses to chair rotations across the entire frequency range tested and a profound functional impact on postural control when forced to rely on vestibular system cues alone with repeated true free falls (see tracings in Figure 8B) on all trials of conditions 5 and 6 with normal MCT findings. The functional impact of the loss of horizontal VOR was reflected in a dramatic DVA result of a five-line loss of visual acuity with reciprocal head movements at 2 Hz in the horizontal plane. The collective findings to this point demonstrated, as suspected, bilateral peripheral horizontal canal paresis of a moderate to severe degree. However, nothing has been done to directly evaluate components of the peripheral vestibular system other than the horizontal canal.

To investigate the vertical semicircular canals clinical head thrusts were performed with video-oculography in the planes of the right anterior/left posterior canals (called the RALP thrust), and the left anterior/right posterior (called the LARP thrust) (Aw et al., 1999). In both of these thrust tests, the patient was able to maintain her vision steady on the target during a thrust upward (assessing posterior canals) but made repeated corrective saccades for downward (assessing anterior canals) movements. For assessment of the utricular otolith organs, video-oculography was used to assess the ocular counter-roll (static position of the eye as an orienting response relative to the pull of gravity mediated by the utricular organs causing the eye to make a static torsional position change away from the ear that is down) when the head was tilted in the coronal plane from upright toward the left or right shoulders (Raphan & Cohen, 2002). For movements in toward either shoulder no counter-roll of the eyes was observed. Collectively, these clinical findings suggest involvement in both anterior canals and the utricular systems but preservation of function in the posterior canals, a pattern seen commonly in vestibular neuronitis (Aw, Fetter, Cremer, Karlberg, & Halmagyi, 2001).

Finally, to investigate the saccular system, vestibular-evoked myogenic potential (VEMP) testing was performed using a click stimulus for the auditory signal. Figure 9A–B shows the potential recorded from the left and right sternocleidomastoid muscles resulting from stimulation to the left and right ears, respectively. The presence of these responses confirmed functioning on at least a partial basis of both saccular organs and the integrity of the inferior division of the vestibular portion of the VIIIth cranial nerve.



A. Above . B Below

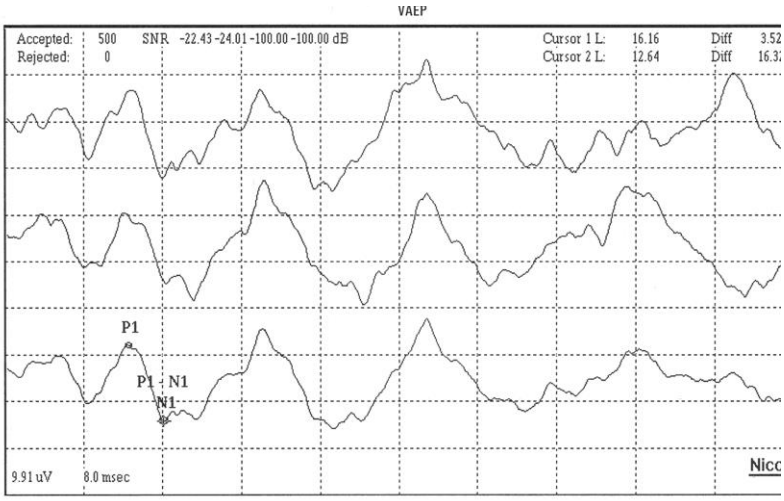


Figure 9. A. Results of the vestibular-evoked myogenic potential test are provided for stimulation to the left saccule. **B.** Stimulation to the right saccule in. The acoustic stimulus was a click presented at 105 dB nHL to each ear individually. In each panel the top two average responses are for two different recording trials. The third trace is the average of the two individual trials. The response of interest is marked in each panel as Pl. N l.

In summary, the overall results for the patient in Case 5 clearly demonstrated differential damage to the vestibular labyrinthine systems bilaterally in a pattern of lesion site and by history of events most consistent with sequential vestibular neuronitis not damaged as a result of the use of the ototoxic agent cisplatin. This cleared the way for a third round of chemotherapy that would include cisplatin.

Case 6

A 42-year-old female reported for evaluation after multiple episodes of 1 to 3 days of head movement sensitivity. These intervals of time had been going on for about 6 months prior to the evaluation. She denied any changes in hearing and denies any spontaneous events of vertigo. On the days when she was sensitive to head movements, it was dominantly movements in the pitch plane or when rolling in bed that would provoke vertigo that would last for less than 1 min after a provocative movement. Her direct examination and all of her laboratory studies including SOT and MCT were all normal other than a positive Hallpike maneuver on the left. She also scored high on the Hospital Anxiety and Depression Scale for anxiety. She was treated with a left Canalith repositioning maneuver and symptoms resolved completely. She was instructed as to how she could treat herself if the symptoms were to return. A year later she returned reporting she had several recurrences of the symptoms but was able to manage the symptoms with the home maneuver she had been taught up until about 5 months ago. She reports that about 5 months ago she had a severe recurrence and the home exercise had reduced the symptoms as in the past but had not resolved the symptoms. She reported that repeated use of the treatment at home and by her local physical therapist had not helped. She was now having symptoms that were a constant sensation of vague movement in the head with unsteadiness that worsened with head movements. Symptoms were best when lying down and increased with sitting and going to standing and walking. She also reported that her symptoms would be exacerbated with visual motion, visual complexity such as stores, walking over or past visual patterns, and reading.

On re-evaluation she was again high on the Anxiety scale but all of her VNG, rotary chair, VEMP both ocular and cervical testing were normal. Her Hallpike to the left and right provoked equal symptoms, but there were was no nystagmus. The Hallpike was repeated a second time after about 30 min with the same results. She also reported that the symptoms with the Hallpike were that of unsteadiness, vague motion in her head but no true vertigo that had been present at the start of this recurrence of symptoms. Her MCT was well with in normal limits, but her SOT showed abnormally increase sway magnitude without fall reactions on conditions 1, 2, and 3 with normal performance on conditions 4 to 6. While this pattern has been reported as aphysiologic per the discussion above, we interpreted the pattern as showing an anxiety reaction to the testing situation. The combination of her persistent symptoms and the development of the visual sensitivities along with the anxiety reaction on SOT collectively supported the diagnosis of Persistent Postural-Perceptual Dizziness (3PD - former Chronic Subjective Dizziness Syndrome-CSD). Typical treatment was to start a medication for anxiety and depression and to start on habituation therapy for the sensitivities to visual and head movement stimuli that exacerbate her symptoms. She had developed 3PD secondary to the severity of the recurrence of the BPPV and to the length of time that the symptoms lasted (1 to 2 weeks) compared to the other times with symptoms for only 1 to 4 days. At follow-up in 3 months she was virtually free of symptoms and had not had any recurrence of the BPPV. At the second follow-up she called to cancel informing us that she was now fully without any of the symptoms. She was slowly tapered off the medication and told she could stop the therapy exercises.

In this case the findings on SOT had changed from the initial symptoms to those consistent with 3PD. The findings that are seen on SOT that imply an "aphysiologic pattern" should be interpreted in the context of the overall presentation as in more cases than not this appears as an anxiety reaction to the testing situation.

Conclusion

This article aimed to discuss the clinical utilization of dynamic posturography by presenting a series of illustrative cases. It is important, as shown in the cases, to understand how posturography can be used in the overall assessment of the patient with dizziness and balance complaints with other studies for a full picture of the patient's status.

As computerized dynamic postural control assessment moves into its next phase of technology with the use of virtual reality as substitute for the mechanical visual surround, it is hoped that new uses of the device clinically will become apparent. These may focus on using the device for recognizing and performing therapy for visual motion sensitivity that is seen in migraine patients and in those with Persistent Postural-Perceptual Dizziness (3PD - former Chronic Subjective Dizziness Syndrome-CSD).

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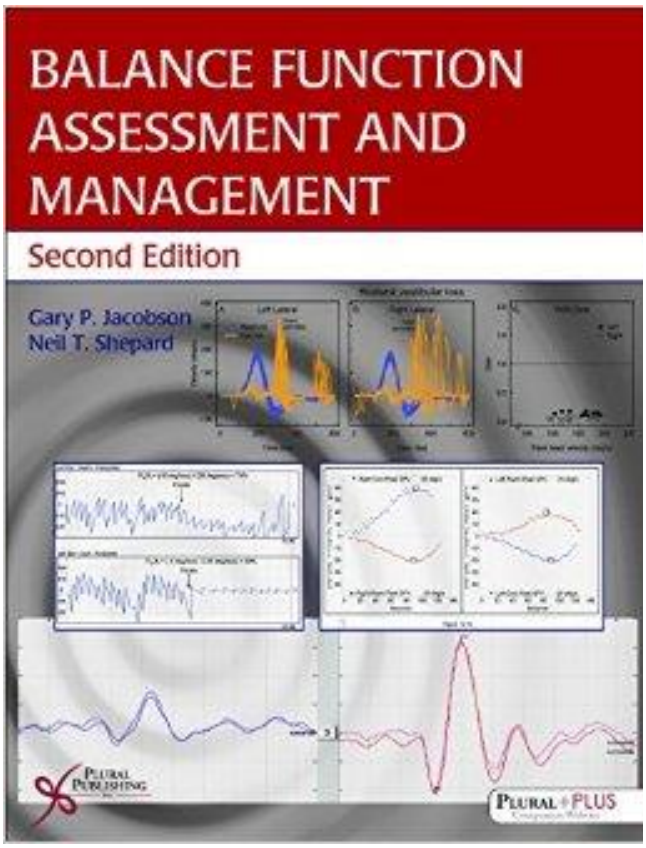

For more detail on this article and more information on Balance Function, please refer to Balance Function Assessment and Management, 2nd edition, Editors Gary P. Jacobson, Neil T. Shepard. Copyright © 2015 Plural Publishing, Inc.).

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Figure 1

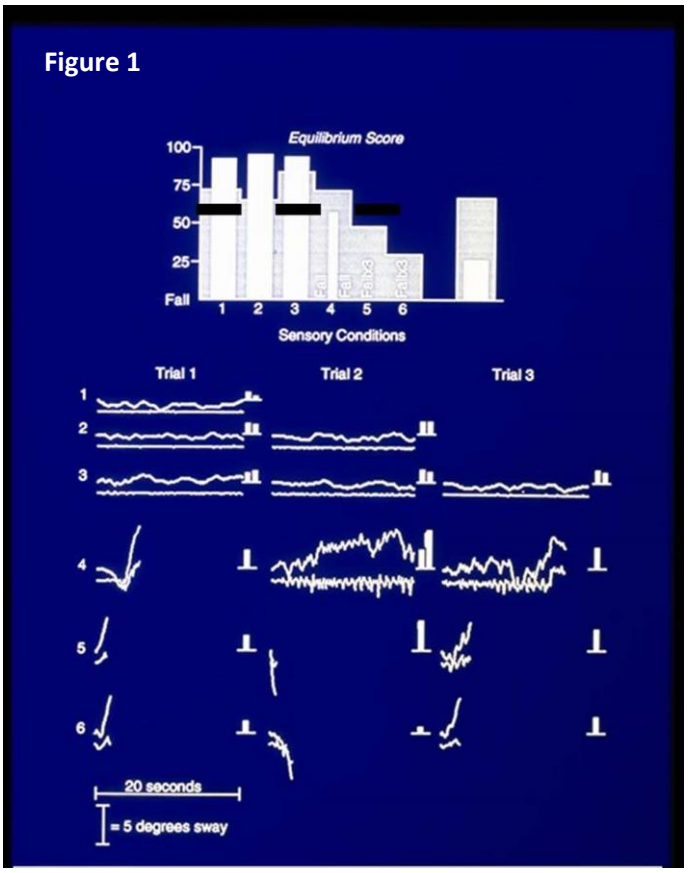
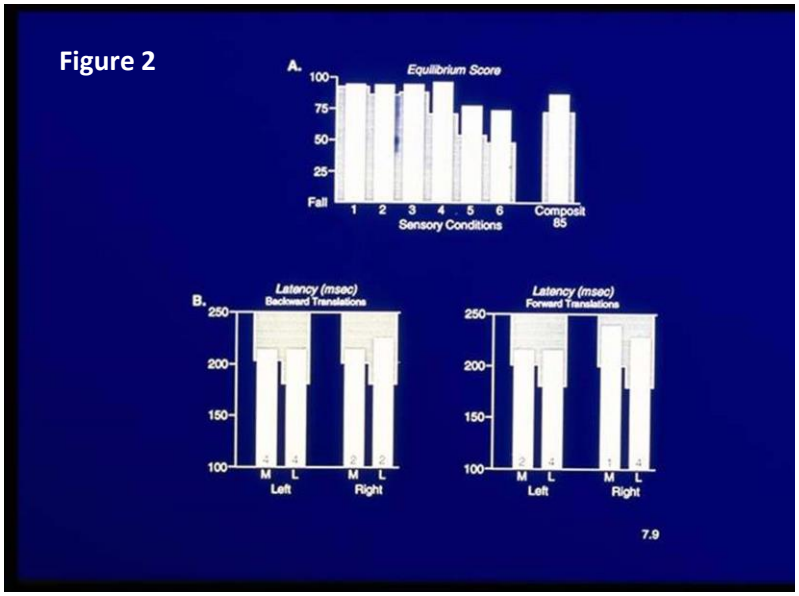


Figure 2



Advances in Vestibular Diagnostics: VEMPS and vHIT

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Advances in vestibular diagnostic testing have extended the region of identifiable pathology to include the otolith organs and the vertical semicircular canals. In particular, the development of two relatively new tests has broadened our capability to measure function from the vestibular labyrinth; the Vestibular-Evoked Myogenic Potentials (VEMP) test and the video head impulse test.

VEMP

The VEMP test is broadly categorized in two unique applications with evidence of its origin from the otolith end organ; the cervical and ocular VEMP. Both tests may use air conducted sound, bone conducted sound, or skull vibration in order to generate an electromyographical potential. Results are averaged over many trials, the stimulus level (decibels of volume (dB)) is linearly related with the response amplitude, and it appears that the best frequency response occurs between 500 and 1000Hz (Akin et al 2003, Nguyen et al 2010). Recordings from otolith afferents suggest that sound and skull vibration stimuli excite both utricular and saccular neurons. Thus, both superior and inferior divisions of the vestibular nerve may contribute to both cVEMP and oVEMP responses (Curthoys 2006). However, it is the final motor neuron synapse (and related behavior) that suggest the unique anatomical origin between the cervical and ocular forms of the VEMP test. **Table 1**.

The cVEMP test exposes patients to a series of graduated, and loud (70-105 dB) clicks. During the sound application, the ipsilateral sternocleidomastoid (SCM) muscle must be contracted. At the same time it is assessed for a release or *inhibition* of its myogenic potential. In healthy vestibular function, subjects generate an initial inhibitory potential (occurring at a latency of 13 msec after the click) followed by an excitatory potential (occurring at a latency of 23 msec after the click), **Figure 1**. The pathway of the cVEMP is believed to be associated with the head-neck reflex that maintains verticality of the head in relation to gravity (the vestibulocollic reflex). The saccule has been implicated as the originating site stimulation during cVEMP testing because saccular afferents provide ipsilateral inhibitory disynaptic input to the SCM muscle, (Kushiro et al 1999) are responsive to click noise (Young et al 1977; Murofushi et al 1995; Murofushi et al 1996) and are positioned close to the footplate of the stapes and, therefore, are subject to mechanical stimulation (Young et al 1977; Halmagyi et al 1995). Additionally, fewer saccular projections exist to oculomotor nuclei/muscle but instead have strong projections to cervical sternocleidomastoid muscles (Uchino and Kushiro 2011).

Table 1: Cervical and Ocular VEMPS

VEMP	Pathway	Stimuli	Latency (msec)	Patient Demand	Origin
Cervical	Vestibulo-spinal: Inhibitory, ipsilateral, descending	Tone bursts or tone clicks	P13 N23	Contracted ipsilateral SCM	Primarily Saccular
Ocular	Vestibulo-ocular: Excitatory, contralateral, ascending	Skull vibration or tone bursts	N10 P16	Look up to bring inferior oblique closer to the skin electrode	Primarily Utricular

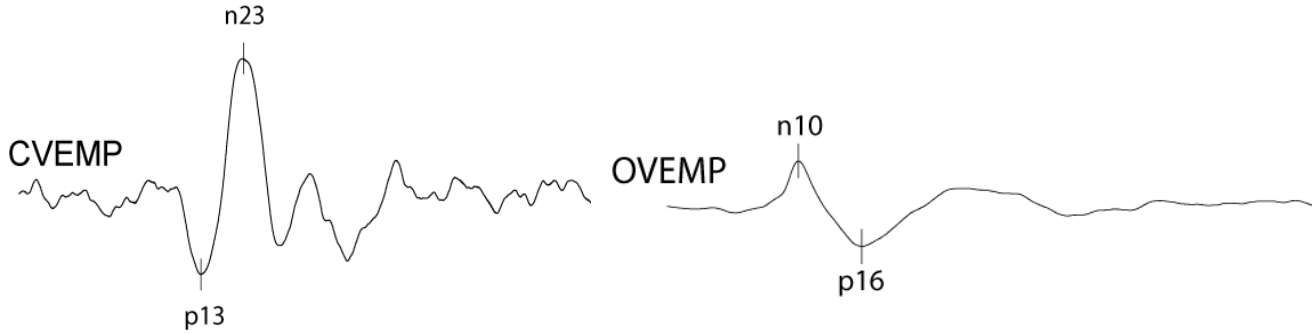


Figure 1. Cervical and ocular VEMP waveforms with the expected positive and negative peak latencies.

The oVEMP commonly exposes subjects to either loud clicks separately in each ear (as does the cVEMP) or bone vibration at the center of the forehead (Fz). During the sound or vibratory application, the contralateral inferior oblique muscle is measured for an *excitatory* potential. The subject is asked to look up (25 - 30°) in order to bring the inferior oblique closer to the skin. In healthy vestibular function, an initial contralateral excitatory potential (occurring at a latency of 10msec) is followed by an inhibitory potential (occurring at a latency of 16msec), **Figure 1**. The oVEMP pathway is functionally relevant for maintaining verticality of eye position in relation to gravity or tilt. The utricle is implicated as the site of afferent stimulation during oVEMP testing based on afferent recordings showing strong projections to the oculomotor system (Uchino and Kushiro 2011), and patient studies establishing abnormal oVEMP test along with abnormal caloric and horizontal head impulse tests (similarly innervated by the superior vestibular nerve), yet normal saccular function (cVEMP). Patients with vestibular neuritis that primarily affected the superior vestibular nerve were found to have a mean n10 amplitude asymmetry ratio of 67%, suggesting the superior vestibular nerve contributed to the oVEMP response (Iwasaki et al 2009).

VEMP INTERPRETATION

The interpretation of the VEMP test considers the threshold of sound intensity needed to recruit the response, the latency of the positive and negative potentials, the peak to peak amplitude, and the asymmetry ratio of the response amplitude. Normal subjects have an expected early (10msec, 13msec) and later (16 msec, 23 msec) latency with symmetric amplitudes at thresholds ~ 100 dB sound pressure level. VEMPS can be recorded in children and infants as long as the clinician can appropriately recruit a strong SCM contraction (cVEMP) and upward gaze (oVEMP) (Chen et al., 2007). Healthy subject above 50 years of age appear to have reduced amplitudes in response to clicks, tones, and skull vibration but normal response latencies and asymmetry ratios (Welgampola and Colebatch 2001; Nguyen et al 2010). Both VEMP tests can be used in individuals with sensorineural hearing loss, but the cVEMP test requires normal conductive hearing (Halmagyi et al., 1994).

VEMP PATHOLOGY

For patients with vestibular hypofunction, typically the cVEMPs may be absent or abnormal on the side of the lesion (ipsilesional) when the saccule or inferior vestibular nerve is affected (Brantberg and Mathiesen, 2004; Iwasaki et al 2007). In contrast, the oVEMP will typically be abnormal at the contralateral inferior oblique when the utricle or superior vestibular nerve is affected. Therefore, one would expect that in the case of a complete loss of left unilateral vestibular function, the cVEMP would be absent from the left SCM (and normal in the right SCM) and the oVEMP absent from the right inferior oblique muscle (and normal from the left inferior oblique muscle). Patients that have third mobile window syndrome (i.e. superior canal dehiscence syndrome (SCDS), or a perilymphatic fistula) may have a lowered threshold for waveform generation (70-80 dB vs 100 dB), and often a large asymmetry ratio from the asymmetrically large waveforms on the affected side (**Figure 2**).

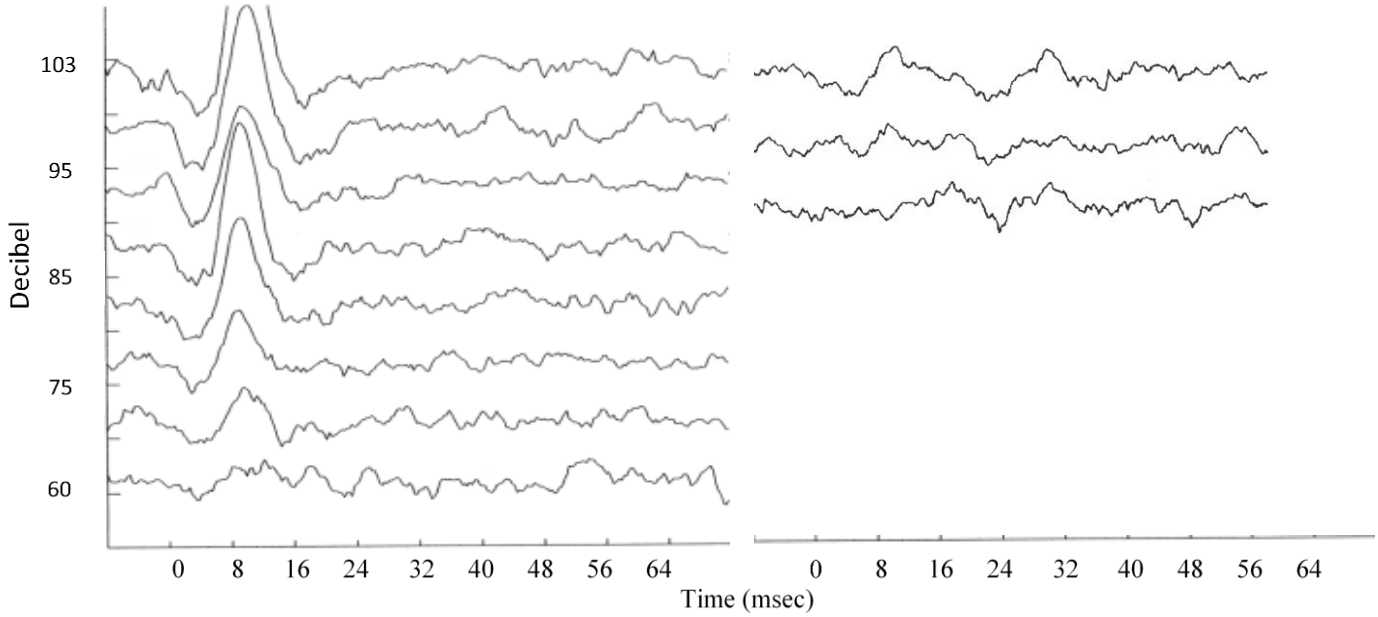


Figure 2. Abnormally low threshold cVEMP response starting ~ 70 dB in a patient with left ear SCDS (left panel). Right panel illustrates normal VEMP response threshold ~ 100dB.

Video Head Impulse Test

The clinical head impulse test is a widely accepted measure of semicircular canal function. Patients are asked to keep their eyes focused on a target while their head is manually rotated in an unpredictable direction using a small amplitude (5°–15°), moderate velocity (200 – 300d/s), and high-acceleration (3,000– 4,000°/s2) angular rotation. The clinical HIT can be applied to each of the six semicircular canals in order to discern their unique function. When the vestibulo-ocular reflex (VOR) is functioning normally, the eyes move in the direction opposite to the head movement and through the exact angle required to keep images stable on the fovea. In the case of vestibular hypofunction, the eyes move less than the required amount. Thus, at the end of the head movement the eyes are not looking at the intended target and images have shifted on the fovea. As a result, the brain recruits a saccade to bring the target back on the fovea. The appearance of these compensatory saccades indicates vestibular hypofunction as evaluated by the head impulse test. The clinical version of this test is subjective and the examiner may not be able to identify a compensatory saccade if it occurs during the head rotation, positioning the eye on the intended target by the time the head has stopped rotating.

Recently, several 2D (pitch and yaw) video-oculography (VOG) systems have been developed that enables the clinician to quantitatively measure the VOR at the bedside with non-invasive, portable, and lightweight technology. The portable VOG goggle systems consist of high-speed digital cameras that track the pupil and inertial measurement devices that measure head velocity. The first video head impulse to obtain FDA approval for use in patients was the ICS video head impulse test made by Otometrics (**Figure 3**). This device has been validated with scleral search coils and provides a graphic representation of the head and eye velocity (both vestibular slow phase and saccades) with metrics including VOR gain, peak head and eye velocity, and compensatory saccade latencies (MacDougall 2009). Software also includes ability for the user to record the oculomotor exam using tests such as spontaneous nystagmus, positioning, and saccades. Aside from the incredible ease the device now affords in objectifying the VOR, its graphical output enables the clinician to identify covert compensatory saccades. Compensatory saccades likely reflect a spectrum of saccades that occur related to a head rotation and have been dichotomized into occurring during (covert) or after (overt) the head rotation (Weber 2009; Schubert 2010). Thus, in the clinical version of the HIT, it is the overt compensatory saccade that identifies the pathology. However, if the compensatory saccade occurs during the head rotation, the clinician will not be able to see it. The vHIT can identify these covert saccades (**Figure 4**).

Figure 3. The video head impulse test goggles as developed by Otometrics. The unit includes one high speed camera for the right eye only, which tracks pupil motion as reflected from the transparent 'hot mirror'.

Report Date: 1/22/2015 Report Operator: Michael Schubert PhD

Head Impulse

Lateral Impulse Test: 1/22/2015 1:48 PM
Test Operator: Michael Schubert PhD
Analysis Left: 19, Right: 20, Rejects: 7
Collection Left: 20, Right: 21, Rejects: 5
Mean Frame Rate: 244

Left Mean: 0.4, σ: 0.04 Right Mean: 0.69, σ: 0.04
Asymmetry: 42%

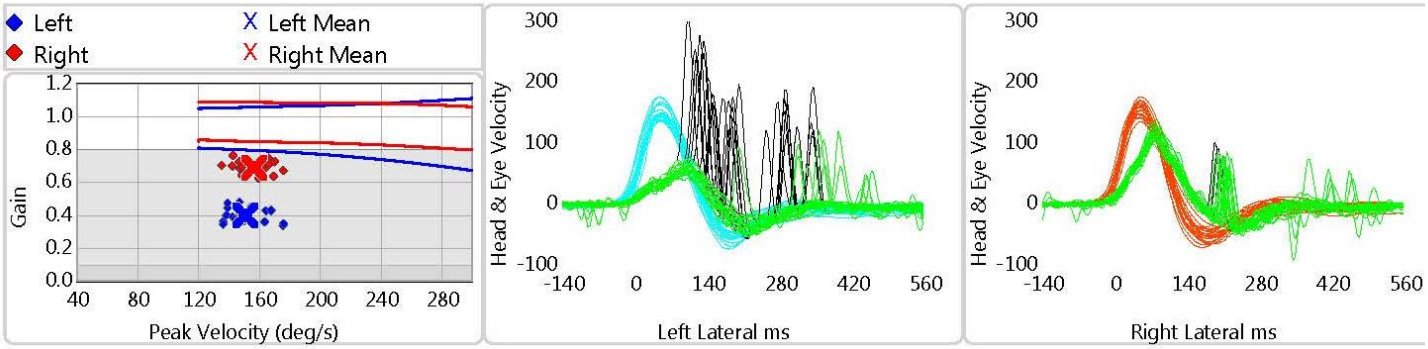


Figure 4. Three panel display of vHIT data. Left panel is the mean (blue and red X) and individual (blue and red dots) VOR gain values; Red and Blue lines denote normal VOR gain over increasing velocity for age-matched healthy controls. The middle and right panel shows raw head and eye velocity traces: leftward head rotation is represented by blue traces, rightward head rotation in red traces, and eye velocity is green. Overt and covert compensatory saccades (black spikes) for leftward head rotation, with bilaterally reduced VOR gain in a subject with asymmetrical bilateral vestibular hypofunction. The compensatory covert saccades occur near the peak slow eye velocity, while the overt saccades occur after the head velocity crosses zero. VOR gain denoted above the respected HIT.

vHIT interpretation

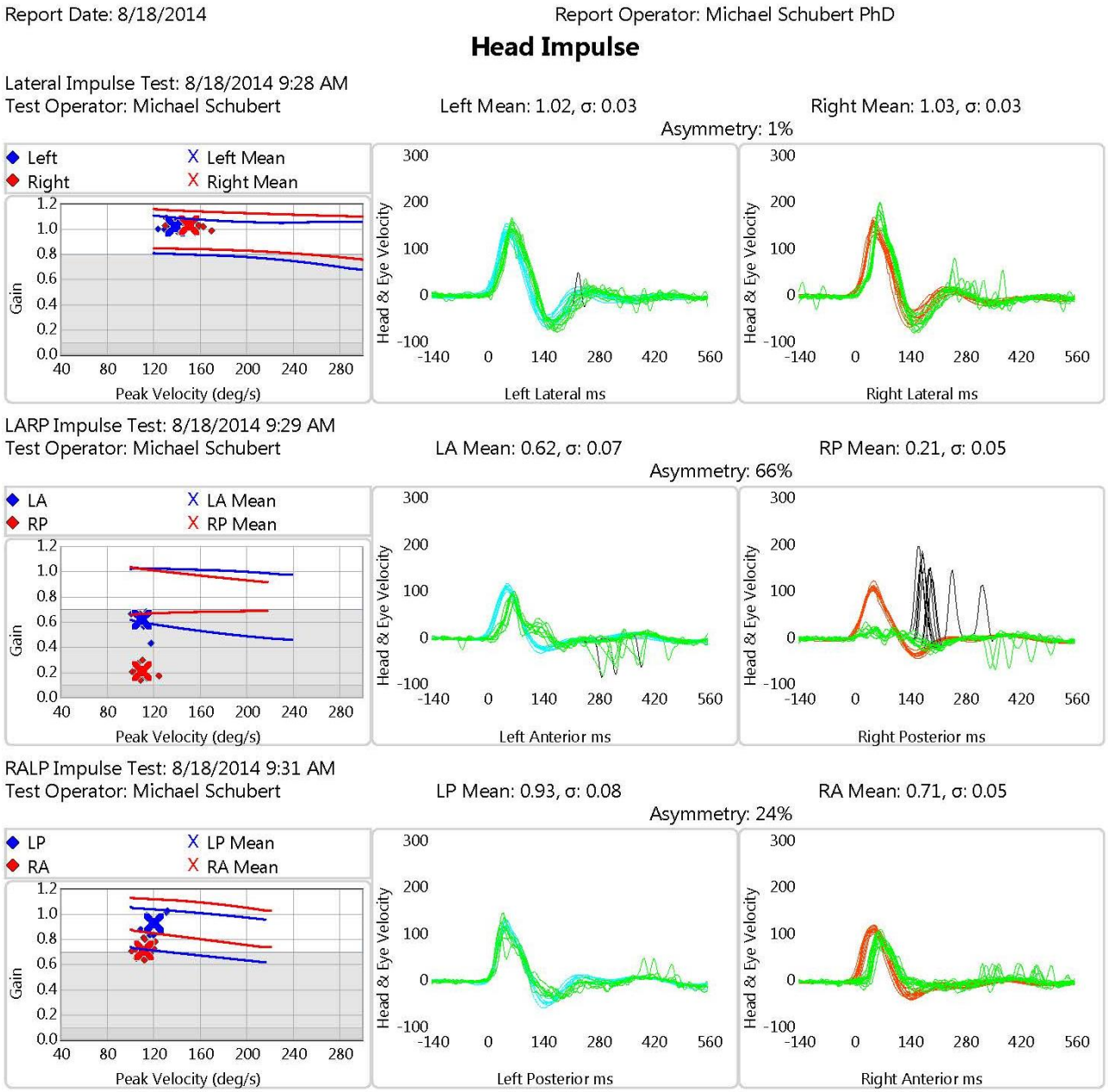
Current software does not enable the measurement of torsion, a significant component of the slow phase vestibular response generated from the vertical semicircular canals. Thus, the test is applied by first rotating the head 45deg to one side and moving the head in pitch (up for pSCC and down for aSCC). In doing so, the torsional component is nullified and only the vertical eye rotation is recorded. The software comes with modifiers the clinician can adjust as minimum values to identify thresholds of VOR gain pathology.

Recently, the ICS version of the vHIT now offers age-matched control normative VOR gains for each of the six semicircular canals. Evidence is mounting that both VOR gain and/or the presence of compensatory saccades may be useful to identify pathology. As these devices become more common, we expect to learn more about vestibular physiology and pathophysiology as well as compensation.

Figure 5. Video head impulse test data identifying presumed inferior vestibular nerve hypofunction based on pathologic right posterior semicircular canal VOR gain (flat green trace) and presence of compensatory overt saccades (black spikes in the waveform). LA – left anterior semicircular canal; RP- right posterior semicircular canal; LP – left posterior semicircular canal; RA – right anterior semicircular canal.

vHIT Pathology

The vHIT enables the user to identify reduced VOR gain, increase VOR latency, or the presence of compensatory saccades in each semicircular canal. This information is critical and can help identify a superior vs. inferior vestibular nerve lesion (**Figure 5**), a high frequency VOR deficit when caloric exams may be normal, and whether compensation is occurring (presence of compensatory saccades). Studies are investigating how the VOR may change over time, with rehabilitation, or across unique diseases.



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The VR SIG sincerely congratulates Dr. Michael Schubert on his SERVICE TO THE SIG award.

Thank you, Dr. Schubert, for all you do for us and the field of vestibular rehabilitation!!

Congratulations to the Vestibular Rehabilitation SIG and all our members for 20 years of supporting the practice of Vestibular Rehabilitation.

Thank you to all those who have been a part of the SIG, and especially those who have been part of SIG leadership since 1996!