Agreement between Functional and Electrophysiologic Measures in Patients with Unilateral Peripheral Vestibular System Impairment

Gary P. Jacobson*
Devin L. McCaslin*

Abstract
This investigation was conducted to determine whether there was congruence between “physiology-based” definitions of compensated and uncompensated unilateral peripheral vestibular system impairment and “functional” measures of self-perceived dizziness disability/handicap. A retrospective analysis was performed on data obtained from 122 patients evaluated in the Balance Function Laboratory at Henry Ford Hospital over a 4-year period. Both electronystagmography and rotational test data were tabulated. Additionally, results of a self-report measure of dizziness disability/handicap were tabulated. Patients were placed into four groups, with one group representing normal vestibulometric test results, one group representing compensated unilateral peripheral vestibular system impairment, and two groups representing increasing magnitudes of uncompensated unilateral peripheral vestibular system impairment. The total and subscale scores on the self-report measure served as the dependent variable. Results showed a lack of congruence between the physiologic and functional measures. We interpret these findings as evidence that factors other than semiobjective evidence of vestibular system compensation probably impact functional recovery following unilateral peripheral vestibular system impairment.

Key Words: Dizziness, Dizziness Handicap Inventory, electronystagmography, vestibular compensation

Abbreviations: CNS = central nervous system; DHI = Dizziness Handicap Inventory; DP = directional preponderance; ENG = electronystagmography; SN = spontaneous nystagmus; UW = unilateral weakness; VOR = vestibuloocular reflex

Sumario: Se condujo esta investigación para determinar si había congruencia entre las definiciones “fisiológicas” de trastornos periféricos, unilaterales, compensados o descompensados del sistema vestibular y las medidas “funcionales” de la auto-percepción del grado de impedimento o discapacidad a partir del mareo. Se realizó un análisis retrospectivo de la información obtenida de 122 pacientes evaluados en el Laboratorio de Balance del Hospital Henry Ford durante un periodo de 4 años. Se tabuló la información tanto de la electrónistagmografía como de las pruebas de rotación. Además, se tabularon los resultados de una medida de auto-reporte sobre el grado de impedimento o discapacidad a partir del mareo. Los pacientes fueron colocados en cuatro grupos: uno representando los resultados vestibulométricos normales, otro representando el trastorno vestibular periférico, unilateral, compensado, y dos grupos representando magnitudes crecientes de trastorno vestibular periférico, unilateral y descompensado. El puntaje total y el de las sub-escalas sobre la medida de auto-reporte constituyeron la variable independiente. Los resulta-
Acute, unilateral, permanent peripheral vestibular system impairment results in an asymmetry in the tonic resting discharge from the two end-organs. The result of this resting asymmetry is a horizontal-rotary spontaneous nystagmus (SN) with a horizontal component fast-phase beating toward the unimpaired end-organ. In addition to this “paretic” nystagmus, patients also demonstrate abnormal postural control (e.g., on Romberg testing, patients fall toward the ipsilesioned ear). Unimpeded reflexes during the acute phase of disease commonly result in vertigo, ataxia, blurred vision (i.e., owing to the nystagmus), nausea, and vomiting. Central nervous system (CNS) compensation for this unilateral deficit occurs rapidly for active, neurologically intact patients. Within days, the vertigo, nystagmus (i.e., with eyes open), nausea, and vomiting subside. Within weeks, patients are capable of ambulating normally and under normal conditions will be symptom free except during rapid head movements in which the peripheral asymmetry is exaggerated. Usually, these symptoms are short-lived because of the influence that the central vestibular system and, in particular, the cerebellar vermis have on the vestibular nuclei on the affected and nonaffected sides. The process of CNS compensation for unilateral loss of peripheral vestibular system function has been described in detail in a previous report (Jacobson and Calder, 2000).

Results of conventional laboratory tests, including the traditional electronystagmographic (ENG) test battery (e.g., tests of ocular motility, positional/positioning testing, bithermal caloric testing) and rotary chair testing, show stereotypical patterns when they are viewed from the acute period through the process of CNS compensation following a unilateral peripheral vestibular system impairment. In the acute period, on ENG testing, patients often demonstrate SN, impaired smooth pursuit for targets moving in the direction of the fast phase of the SN, impaired optokinetic nystagmus for targets moving in the direction of the fast phase of the SN, unilateral weakness (UW) on the side of the impaired end-organ, and a directional preponderance (DP) (i.e., caloric responses that result in nystagmus beating in the same direction as the SN are enhanced). Rotary chair testing for these patients shows phase leads at multiple frequencies and slow-phase asymmetries (i.e., the magnitude of the slow phase of nystagmus is greater when patients are rotated toward the unimpaired end-organ). Slow-phase gain usually is normal or near-normal. This pattern of electrodiagnostic test results has been described as representing an “uncompensated” unilateral, peripheral vestibular system impairment (Furman and Cass, 1996).

Within weeks, on ENG testing, many patients will demonstrate normal ocular motility. Some patients may still demonstrate residual SN when visual fixation is removed. Additionally, patients will demonstrate a UW on caloric testing. Often patients will demon-
strate a residual low-frequency residual phase lead (i.e., at 0.01 Hz) with normal slow-phase gain and symmetry. This constellation of findings has been described as evidence showing that patients “compensated” for unilateral, permanent, peripheral vestibular system impairments (Furman and Cass, 1996, Stockwell and Bojrab, 1997).

An intermediate patient profile has been described in which ENG findings are identical to those described for patients with unilateral, centrally “compensated” peripheral vestibular system impairments but in which rotary chair testing demonstrates multiple-frequency phase leads (e.g., through 0.16 Hz), with normal nystagmus slow-phase symmetry and gain (Bojrab and Stockwell, 1994). The authors reported that these abnormalities could persist for many years and that patients demonstrating these findings had compensated for vestibular system impairments (i.e., had they not compensated for the impairments, they would have demonstrated persistent gain asymmetries).

The purpose of the present investigation was to determine whether patient self-report measures of dizziness disability/handicap were congruent with physiologic subclassifications of vestibular system compensation. That is, it was our hypothesis that stepwise increases in the magnitude of vestibular system impairment would be associated with a statistically significant, stepwise increase in self-perceived dizziness disability/handicap. Accordingly, the purpose of the present project was to determine whether conventional physiologic definitions of “compensated” and “uncompensated” unilateral peripheral vestibular system impairment are congruent with measures of self-perceived dizziness disability/handicap.

**METHOD**

Subjects were culled from a retrospective analysis of all balance function tests conducted between 1997 and 2001 in the Balance Function Laboratory at Henry Ford Hospital. That search yielded 122 patients (41 male; mean age = 54 years [SD = 17 years]), who were classified into four groups:

- **Group 1 (normal)** (n = 21; 7 male; mean age = 48 years [SD = 13 years]): patients showing normal ENG and rotary chair test results
- **Group 2 (compensated unilateral)** (n = 45; 13 male; mean age = 50 years [SD = 17 years]): patients demonstrating significant UW on caloric testing and on rotational testing demonstrated a single-phase lead at 0.01 Hz as the only abnormality
- **Group 3 (uncompensated, resolving unilateral)** (n = 36; 14 male; mean age = 64 years [SD = 15 years]): patients demonstrating a statistically significant UW on caloric testing and on rotational testing showed phase leads at three or more adjacent frequencies from 0.01 to 0.32 Hz as the only abnormality
- **Group 4 (uncompensated unilateral)** (n = 20; 7 male; mean age = 54 years [SD = 17 years]): patients demonstrating a statistically significant UW on caloric testing and on rotational testing showed phase leads present at three or more adjacent frequencies from 0.01 to 0.32 Hz, and additionally demonstrated slow-phase gain asymmetries for three or more adjacent frequencies from 0.01 to 0.32 Hz

Although these groupings appear somewhat arbitrary, there is some evidence to support the contention that groups 2, 3, and 4 represent “compensated unilateral,” “resolving (uncompensated) unilateral peripheral vestibular system impairment,” and “uncompensated unilateral peripheral vestibular system impairment,” respectively (Furman and Cass, 1996; Stockwell and Bojrab, 1997). Based on our laboratory normative data, a statistically significant unilateral peripheral weakness on the alternate binaural bithermal caloric test was defined as ≥ 22 percent left- versus right-ear difference in average slow-phase velocity at the peak of the caloric response (≥ 28% difference between right- and left-beating responses for a statistically significant DP).

For ENG and rotational testing, disposable silver/silver chloride surface electrodes were placed bitemporally (i.e., to record horizontal versional eye movements) and above and below the eye with better corrected vision (i.e., to record vertical versional eye movements). Electrode impedances were ≤ 5000 ohms and interelectrode impedances were ≤ 3000 ohms. ENG testing was conducted with an ICS Mastr system. Calibration was performed by having
patients follow a sinusoidal target moving ± 15 degrees from midline horizontally (i.e., for horizontal eye movement calibration) and then ± 15 degrees from midline vertically (i.e., for vertical eye movement calibration). The ENG test battery included conventional tests for gaze-evoked nystagmus; pursuit, saccadic, and optokinetic ocularmotor subsystem impairments; spontaneous, position-induced, and positioning nystagmus; and alternate binaural bithermal water caloric testing. Rotary chair testing (Neurokinetics 1010) was conducted over a frequency range including 0.01, 0.02, 0.04, 0.08, and 0.16 Hz (maximum velocity 100 degree/sec, maximum acceleration, 50 degrees/sec²). Measures of eye movement slow-phase velocity, gain, and symmetry were calculated on the data collected for each frequency.

In addition to undergoing ENG and rotary chair tests, the patients also completed a self-report measure of the impact of dizziness on quality of life. This metric, called the Dizziness Handicap Inventory (DHI; Jacobson and Newman 1990), consists of 25 self-evaluative statements that patients may answer in one of three ways: (1) “yes” (awarded a score of 4 points), (2) “sometimes” (awarded a score of 2 points), and (3) “no” (awarded a score of 0 points). The DHI consists of three content domains designed to evaluate the effects of dizziness and unsteadiness on the functional aspects of life, emotional well-being, and physical functioning. There are nine items in the Functional subscale (i.e., response range = 0–36 points), nine items in the Emotional subscale (i.e., response range = 0–36 points), and seven items in the Physical subscale (i.e., response range = 0–28 points). In addition to subscale scores, this metric yields a total score ranging from 0 (representing minimum self-perceived dizziness handicap) to 100 points (representing maximum self-perceived dizziness handicap). This scale was administered using a face-to-face format.

RESULTS

Caloric Test Results for Groups 1 to 4

The results of bithermal caloric testing for the four groups in this investigation are shown in Table 1. It can be seen that the mean percent UW was greater for groups 3 (uncompensated resolving) and 4 (uncompensated) compared with group 2 (compensated). In this regard, patients demonstrating phase leads at multiple frequencies demonstrated, on average, 71 percent greater UW than patients who demonstrated a single-phase lead at the lowest rotational frequency (i.e., 0.01 Hz). Patients who demonstrated both phase leads and asymmetries at multiple frequencies showed, on average, 89 percent greater UW than did patients demonstrating a single-phase lead at the lowest rotational frequency. Analysis of variance (ANOVA) was conducted with percent UW serving as the dependent variable and patient group (i.e., groups 1–4) serving as the grouping factor. The result of this analysis showed a significant main effect (F = 39.47, df = 3, 118, p < .001). Post hoc testing indicated that statistically significant differences in UW (p < .001 in each case) existed between group 1 (normal) and groups 2 (compensated), 3 (uncompensated resolving), and 4 (uncompensated). Post hoc testing also showed statistically significant differences (p < .001 for both post hoc comparisons) between group 2 (compensated) and groups 3 (uncompensated resolving) and 4 (uncompensated).

Table 1 shows the levels of DP for patients in the four groups. Patients with
phase leads and asymmetries at multiple frequencies demonstrated, on average, greater-magnitude DP than the other two patient groups. A second ANOVA was conducted to determine whether there were significant group differences in DP. The results of this analysis revealed a significant main effect ($F = 10.31, df = 3, 118, p < .001$). Post hoc testing showed that statistically significant differences ($p < .001$ for all three post hoc comparisons) in DP occurred only between group 4 (uncompensated) and groups 1 (normal), 2 (compensated), and 3 (uncompensated resolving).

**Assessment of Balance Function Test Result on DHI**

Table 2 illustrates mean data from the four groups for the DHI total and subscale scores. A series of four ANOVA were conducted to determine whether DHI total and subscale scores (i.e., Functional, Emotional, and Physical subscales) differed as a function of patient groupings. Post hoc testing was conducted in the presence of a significant main effect.

Group differences were statistically significant for the Functional subscale ($F = 9.79, df = 3, 117, p < .001$). Post hoc testing showed that only the differences between group 1 (normal) and groups 2 (compensated), 3 (uncompensated resolving), and 4 (uncompensated) were statistically significant ($p < .001$ in each case).

Statistically significant group differences were observed for the Emotional subscale ($F = 3.35, df = 3, 117, p = .02$). Post hoc testing showed a significant difference ($p = .04$) only between group 1 (normal) and group 4 (uncompensated).

Statistically significant group differences were observed for the Physical subscale ($F = 5.85, df = 3, 117, p = .001$). Post hoc testing revealed statistically significant differences between group 1 (normal) and group 2 (compensated; $p = .02$), group 1 (normal) and group 3 (uncompensated resolving; $p = .007$), and group 1 (normal) and group 4 (uncompensated; $p = .001$).

Finally, statistically significant group differences were observed for the total score ($F = 8.41, df = 3, 117; p < .001$). Post hoc testing revealed statistically significant differences between group 1 (normal) and group 2 (compensated; $p = .005$), group 1 (normal) and group 3 (uncompensated resolving; $p < .001$), and group 1 (normal) and group 4 (uncompensated; $p < .001$).

**DISCUSSION**

Sudden unilateral peripheral vestibular system impairment results in vertigo and a contralesional beating SN. If connections between the vestibular nuclei and midline cerebellar structures (e.g., the cerebellar vermis) are intact, this SN resolves over time (Jacobson et al, 1998). In most instances, within a month, the patient is asymptomatic for vertigo.

During the acute (i.e., uncompensated) phase of a unilateral peripheral vestibular system impairment, caloric testing shows an ipsilesional UW and a contralesional DP. On rotational testing, a patient will demonstrate both slow-phase velocity phase leads and contralesional vestibulococular reflex (VOR) asymmetries at multiple frequencies. The asymmetries occur owing to the bias imposed by the spontaneous nystagmus on slow-phase eye movements that are generated toward the ipsilesional ear. The phase leads occur owing to the ipsilesional loss of the velocity storage function (Barin and Durrant, 2000). The concept of velocity storage is complex, and clear descriptions of this concept are difficult to find (see Barin and

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**Table 2** Mean DHI Data (SD) for Each of the Four Groups in the Present Investigation

<table>
<thead>
<tr>
<th>Scale</th>
<th>Group 1 (Normal)</th>
<th>Group 2 (Compensated Unilateral)</th>
<th>Group 3 (Uncompensated Resolving Unilateral)</th>
<th>Group 4 (Uncompensated Unilateral)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional (points)</td>
<td>6.00 (5.31)</td>
<td>15.07 (8.92)</td>
<td>16.61 (8.21)</td>
<td>18.90 (9.14)</td>
</tr>
<tr>
<td>Emotional (points)</td>
<td>7.60 (8.22)</td>
<td>11.58 (8.70)</td>
<td>14.11 (9.21)</td>
<td>15.60 (9.53)</td>
</tr>
<tr>
<td>Physical (points)</td>
<td>8.60 (7.92)</td>
<td>15.20 (9.01)</td>
<td>16.05 (6.80)</td>
<td>18.70 (7.84)</td>
</tr>
<tr>
<td>Total (points)</td>
<td>22.00 (19.93)</td>
<td>41.84 (23.12)</td>
<td>46.77 (20.34)</td>
<td>53.70 (20.58)</td>
</tr>
</tbody>
</table>

DHI = Dizziness Handicap Inventory.
In brief, the purpose of this function (that is believed to reside in the rostral medulla and/or caudal pons at the level of the vestibular nuclei; Holstein et al., 1999) is to increase the vestibular time constant by gating the outflow of activity from the vestibular nuclei and, in so doing, increase the low-frequency sensitivity of the vestibular system. Without velocity storage, the best sensitivity of the vestibular system to angular acceleration/deceleration would be for the frequency range from approximately 0.3 to 10 Hz. For this range of frequencies, the gain of the compensatory eye movements will approximate 1.0 (i.e., head velocity will be equal to eye velocity) and the phase difference in the eye position versus head position will be perfectly opposite (i.e., a 180-degree difference). The addition of the velocity storage mechanism has the effect of extending the lower-frequency response of this best frequency range to approximately 0.1 Hz (i.e., from 0.1–10 Hz; Barin and Durrant, 2000). Loss or impairment in velocity storage results in reductions in low-frequency gain and phase abnormalities (e.g., phase leads) at lower frequencies.

During the postacute phase of a unilateral peripheral vestibular system impairment, usually patients will show neither a contralesional beating SN with eyes open nor a contralesional DP on caloric testing. However, they do continue to demonstrate an ipsilesional UW on caloric testing and phase leads at multiple frequencies on rotational testing.

Assuming that pathways subserving central vestibular system compensation are intact, patients with unilateral, permanent reductions in afferent peripheral vestibular “tone” continue to demonstrate an ipsilesional UW on caloric testing and may or may not demonstrate a single-phase lead for low-frequency rotations (e.g., 0.01 Hz). This residual abnormality in the timing relationship between head and eye movement for low-frequency oscillations represents a permanent reduction in the velocity storage mechanism.

This investigation was conducted in an effort to determine whether conventional operational definitions of the terms “compensated” and “uncompensated” unilateral peripheral vestibular system impairment are supported by self-report measures of dizziness disability/handicap. That is, we were interested in knowing whether there was congruence between physiologic measures of unilateral vestibular system impairment and behavioral measures of dizziness disability/handicap. The results of this investigation have suggested that, although there was a trend for patients with “uncompensated” unilateral impairments to demonstrate greater self-perceived dizziness handicap, these differences were not statistically significant.

The lack of congruence between physiologic and functional measures of balance is not surprising. That is, generally poor agreement has been reported between modality-specific self-report measures of disability/handicap and measures of impairment. For example, although it has been reported that > 90 percent of the time, tinnitus loudness matches (i.e., at the tinnitus pitch matching frequency) range between 3 and 15 dB SL (Vernon and Meikle, 1988); the range of self-report measures of tinnitus disability/handicap can range from none to severe (Newman et al., 1996). In the same regard, there is only a modest relationship between the magnitude of hearing impairment and self-report hearing disability/handicap until hearing impairment reaches the severe to profound magnitude (e.g., Newman et al., 1990). It might then be expected that only large-magnitude bilateral reductions in peripheral vestibular system function would be associated consistently with the greatest levels of dizziness disability/handicap. This, in fact, has been our observation in previous research (Jacobson and Calder, 2000).

It is possible that what constitutes “compensated” and “uncompensated” unilateral peripheral vestibular impairment extends beyond the limited scope of the VOR. How well patients recover from a unilateral peripheral vestibular system impairment is probably influenced by the intactness of the other supporting senses such as vision and somesthesia and the intactness of the motor system that helps restabilize a patient who has become destabilized by a postural challenge. For example, an elderly sedentary patient with a significant unilateral peripheral vestibular system impairment who has poor vision and a diabetic peripheral neuropathy affecting sensation in the lower extremities is not going to compensate functionally as
well as an active young patient with intact vision and somesthesia. In fact, it has been our clinical experience that those patients with unilateral peripheral vestibular system impairments who demonstrate the greatest-magnitude self-report disability/handicap also demonstrate concomitant impairments of supporting senses (Jacobson, 2002). It is our suggestion that the value of self-report measures of dizziness disability/handicap may be to help identify patients whose central vestibular systems are incapable of compensating completely for unilateral impairments or whose other supporting senses (e.g., vision and somesthesia) are not sufficiently intact to compensate for unilateral reductions in function. That is, we would suggest that patients who demonstrate vestibulometric patterns suggestive of “compensated” peripheral vestibular system impairment (e.g., UW on caloric testing and normal results on rotational testing) but who show signs of significant dizziness disability/handicap should be suspected of having central balance system impairments and/or impairments of the other supporting senses.

The relationships between ENG and rotational test results also were revealing. That is, we found that the results of ENG testing are predictive of the results of rotational testing and vice versa. For example, investigations have suggested that a phase lead at 0.01 Hz on rotational testing greatly increases the likelihood that there will be a significant UW on caloric testing. The magnitude of that UW increases significantly in the presence of multiple-frequency phase leads on rotational testing (Stockwell and Bojrab, 1997) (Furman and Cass, 1996). In the same regard, the presence of a significant DP on caloric testing greatly increases the likelihood of significant gain asymmetries on rotational testing.

**Limitations of the Present Investigation**

Our “field of view” for this investigation was limited to the results of those tests that quantify characteristics of the horizontal VOR. Vestibular autorotation testing, which permits an evaluation of the characteristics of the VOR for frequencies > 1 Hz, does not exist at our center and was not performed on these patients. Likewise, computerized dynamic posturography, which could have been helpful for evaluating to what extent vestibular system input and visual and proprioceptive inputs contributed to postural stability and self-perceived dizziness disability/handicap, was not performed on all of the patients, and, accordingly, these data were not used in the analyses. Additionally, no instrumentation to assess the otolith organs (e.g., off-vertical axis rotation) was available at our center for the period of time inclusive from 1997 to 2001. Thus, we do not know to what extent coexisting impairments of the utricular and saccular maculae influenced these findings.

Age was a partially uncontrolled variable in this investigation. That is, there was a statistically significant difference in the ages of group 3 (uncompensated resolving) compared with groups 1 (normal; p = .003) and 2 (compensated; p = .001) but not compared with group 4 (uncompensated; p = .198). However, group 3 (uncompensated resolving) consisted of every patient evaluated between 1997 and 2001 who demonstrated this unique pattern of results (i.e., UW and phase leads at three or more phase leads, beginning with 0.01 Hz). The age of this subpopulation appears to have been weighted (by approximately 14 years) toward more elderly patients. It may be that this subpopulation of elderly patients with unilateral peripheral vestibular system impairments, because of their age, were less able to compensate physiologically for these impairments. This could have occurred if the intactness of the central vestibular system was compromised in this subset of patients. If true, then these findings would be representative of another variation of “uncompensated” (instead of “uncompensated resolving”) unilateral peripheral vestibular system impairment.

Despite these limitations, we feel that the present data are useful for helping clinicians understand the complex relationships that exist between physiologic and functional measures. The results suggest that the relationship between these two types of measures is complex. For example, even patients with test results suggesting “compensated” unilateral peripheral system impairments demonstrated significant self-perceived dizziness disability/handicap (mean total DHI score = 42 points). The results of the present investigation suggest
that the conventional physiology-based definitions of what constitutes a "compensated" or "uncompensated" peripheral vestibular system deficit may be augmented with scores on self-report measures of dizziness disability/handicap.

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REFERENCES


