Effectiveness of Careful Bedside Examination in Assessment, Diagnosis, and Prognosis of Vestibular Neuritis

Marco Mandalà, MD; Daniele Nuti, MD; Aimee Teo Broman, MA; David Samuel Zee, MD

Objective: To determine whether the use of 4 bedside tests (head-impulse, head-heave, head-shake, and vibration tests) can be as effective as the caloric test, a widely accepted standard, in the diagnosis and prediction of the time to recovery from vestibular neuritis.

Design: Inception cohort (1-year follow-up), criterion standard study.

Setting: Primary referral center.

Patients: All patients had acute vertigo, and those having a diagnosis of vestibular neuritis were eligible for inclusion in the study. Sixty-eight patients (43 men and 25 women; mean age, 54.9 years) met this criterion, and 53 of them (77.9%) completed the study.

Main Outcome Measures: Spontaneous head-shaking and vibration-induced nystagmus elicited with a battery-powered device were tested wearing Frenzel goggles. The head-impulse and head-heave tests were performed manually. Caloric irrigation was administered with hot, cold, and ice water.

Results: At baseline, more than half of the patients exhibited positive signs with all 4 tests and all had caloric paralysis or paresis. Signs with the head-impulse and head-heave tests correlated highly (odds ratio, 24.9; \( P < .001 \)), as did those with the head-shake and vibration tests (odds ratio, 22.8; \( P < .001 \)). Patients with a positive sign with the head-impulse or vibration test were 70% less likely to recover than were those with a negative sign. Head-impulse (hazard ratio, 0.08; \( P = .002 \)) and head-shake (hazard ratio, 0.23; \( P = .01 \)) test results were associated with the outcome of the caloric test.

Conclusion: Careful bedside examination of patients with vestibular neuritis has both diagnostic value in the short term and prognostic value in the long term.


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A CUTE ONSET OF SEVERE VERTIGO with no other signs or symptoms is usually attributed to vestibular neuritis. Patients with vestibular neuritis also usually have disequilibrium, nausea, and vomiting but no auditory symptoms. Symptoms usually resolve in weeks, but there may be a more protracted course with persistent disequilibrium. The etiology is thought to be viral, though in rare cases, the cause may be labyrinthine ischemia.1 Caloric testing has been the traditional gold standard for detecting a peripheral vestibular deficit, but some recently developed bedside tests can also provide important information for the diagnosis and prognosis of vestibular neuritis.

In 2005, we reported use of the head-impulse test (a measure of function of the lateral semicircular canal) and the head-heave test (a measure of function of the utricle) as prognosticators of recovery from vestibular neuritis.2 We had the advantage of examining a large number of patients in the acute phase of the illness (within 3 days of onset of symptoms). Results were compared with those from the caloric test. Herein, we expand our previous study to determine whether the results of 4 bedside tests (head-impulse, head-heave, head-shake, and vibration tests) can better predict the time to recovery from vestibular neuritis.

METHODS

POPULATION DESCRIPTION

The cohort described is the same patient-based clinic population seen between January 1, 2002, and January 31, 2004, and whose results were reported previously.2 Patients were seen in the acute stage of the disease, 1 to 3 days after the onset of symptoms. All patients had vertigo, and those having a diagnosis of ves-
tibular neuritis were eligible for inclusion in the study. Inclusion criteria were as follows: (1) acute vertigo for at least 24 hours; (2) horizontal unidirectional spontaneous nystagmus for at least 24 hours; (3) no hearing loss; (4) no additional neurologic signs or symptoms and, when obtained, normal brain images; and (5) abnormal caloric test results (canal paralysis or paresis). Sixty-eight patients met these criteria. Patients were asked to return for follow-up evaluation after 1, 3, 6, and 12 months. Patients were considered to have recovered when results of both caloric testing and bedside examination were normal, after which they were not considered further for this study.

**CLINICAL TESTING**

Patients underwent a complete bedside clinical examination at each visit by one of us (D.N.). Spontaneous nystagmus, head-shaking nystagmus, and vibration-induced nystagmus were tested for with fixation removed wearing Frenzel goggles (Gordon N. Stowe & Associates, Inc, Wheeling, Illinois). Vibration-induced nystagmus was elicited with a battery-powered device; the technical details are reported elsewhere. Vibration was applied to the mastoid region for 10 seconds, first on one side and then on the other. Test results were considered positive if there was persistent nystagmus. Results of the head-impulse test were considered positive if there were at least 3 consecutive beats of nystagmus in patients without spontaneous nystagmus or if there was a clear increase in frequency of eyelid movements in patients with spontaneous nystagmus.

The head-impulse test was performed by rapidly rotating the patient's head (abrupt, high-acceleration rotations of about 20° amplitude) to the right and to the left. The head-heave test was performed by heaving the head of the patient rapidly (abrupt, high-acceleration interaural translations [heaves] of about 5-10 cm in excursion). For both tests, the examiner stood in front of the patient, who was instructed to fix on the examiner's nose. Results of the head-impulse and head-heave tests were considered abnormal if there was an obvious corrective saccade (deficit) with a lateral acceleration toward one (affected) side.

**LABORATORY TESTING**

Vestibular function was determined within 7 days after onset of symptoms using caloric irrigation with hot, cold, and ice water. Maximum slow-phase velocity of nystagmus evoked by irrigating each ear was analyzed for unilateral weakness and directional preponderance according to the Jongkees formulas. Caloric paresis was diagnosed when there was some response on both sides but the difference between the 2 ears was 30% or more. Caloric paralysis was diagnosed when there was no response to ice water irrigation on one side.

**TREATMENT**

After the baseline evaluation, all patients were offered treatment with corticosteroids (oral methylprednisolone sodium succinate, 60 mg/d, tapered during 2 1/2 weeks) and acyclovir (800 mg 4 times daily for 7 days). Twenty-two patients received no treatment, either because they refused it or because there was a contraindication (eg, peptic ulcer, hypertension, or diabetes mellitus). Information about treatment was missing for 3 patients; all had canal paralysis at the baseline examination.

**STATISTICAL ANALYSIS**

The strategy for analysis of these data has been described previously. The strength of correlation between bedside tests was determined with pairwise odds ratios. The Kaplan-Meier method was used to estimate time to recovery. Prediction factors were analyzed using multiple regression models to estimate relative risk of an event (recovery). Recovery was assumed to have occurred at some point between the visit when the patient had a less than 30% caloric deficit and the previous visit, when the patient had a 30% caloric deficit or higher. Data were analyzed with a logistic model with a complementary log-log link. The interpretation of the parameter estimates is similar to that of a proportional hazards model: estimates are raised to the exponential power and represent the percent increase in the chance of recovery per unit increase of the parameter.

Although relative risk analyses consider dropout of participants during the study, these analyses assume that the dropout rates are not influenced by the outcome measures. Dropout in this study, however, was likely owing to the patient feeling better. Therefore, we performed sensitivity analyses to determine the robustness of the predictor estimates. The first sensitivity analysis assumed that patients who dropped out of the study recovered by the time of their first nonattendance at a follow-up visit. The second analysis assumed that those who dropped out did not recover. Estimates and standard errors from this analysis were compared with the analysis using the original data.

The bedside tests were correlated with the caloric test using the same analysis, with time-dependent covariates. This model predicts recovery, and the bedside tests are allowed to be changed across time.

**RESULTS**

Sixty-eight patients participated in the study. Two patients were excluded from further analyses because caloric irrigation could not be performed owing to tympanic perforation. Baseline testing in the remaining 66 patients revealed deficits of 30% to 99% (paresis) in 10 and 100% in 54; baseline testing was not performed in 2 patients.

There were no differences between age and sex insofar as the severity of deficit at baseline (Table 1). Patients with positive head-heave or positive head-impulse signs were more likely to have a severe caloric deficit, but those with positive head-shake or positive vibration signs were not. Although all patients were offered treatment, those with less severe deficits were slightly more likely to accept it, but this difference was not significant, probably because of the small number of those with less severe deficits.

We previously reported the life tables estimating chance for recovery. In brief, the chance of not recovering by the end of 12 months was 49.3%, given that the subject remained in the study until recovery. Six patients (9.2%) did not return to the clinic for follow-up at 3 months, 5 (11.4%) did not return at 6 months, and 2 (7.1%) did not return at 12 months.

More than half of all patients had positive signs at baseline with the head-impulse, head-heave, head-shake, and vibration tests (Table 2). There were 3 patients (4.6%) with a negative head-shake sign at baseline compared with 12 (18.5%) with a negative head-impulse sign, 12 (18.5%) with a negative vibration sign, and 22 (34%) with a negative head-heave sign. All had caloric paralysis or paresis at baseline. When compared across time, head-impulse...
and head-heave signs correlated highly (odds ratio, 24.9; \(P < .001\)), as did head-shake and vibration test results (odds ratio, 22.8; \(P < .001\)) (Table 3). Results of the head-heave test did not correlate so highly as those with the head-shake or vibration tests.

Using information from the initial examination, bedside tests with negative signs independently predicted a higher chance for recovery (Figure). In a multiple regression model in which signs at all 4 bedside tests at baseline predicted recovery, patients with a positive sign with the head-impulse test were 70% less likely to recover than those with a negative sign (odds ratio, 24.9; \(P = .001\)) (Table 4). Results of the bedside test yielded a negative sign. In a multiple regression model, adjusting for month and treatment at baseline, across time, the head-impulse (hazard ratio, 0.08; \(P = .002\)) and head-shaking (hazard ratio, 0.23; \(P = .01\)) tests were associated with the outcome of the caloric test (Table 5) across time. In the presence of these 2 tests, the vibration test did not show any association with the results of the caloric test (hazard ratio, 0.36; \(P = .14\)).

An abnormal caloric response (paresis) with normal results of bedside examination was noted in 2 patients at 1-month follow-up, 3 patients at 3 months, and 2 patients at 6 months. However, 6 patients had a normal caloric response and at least 1 positive sign with a bedside test, primarily with the head-shake or vibration tests. At follow-up, the head-shaking test in 3 patients and vibration test in the other 2 bedside tests accepted treatment seemed to have an almost 3-fold effect on the chance for recovery.

Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Paresis at Baseline</th>
<th>Paralysis at Baseline</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13-39</td>
<td>1 (10.0)</td>
<td>12 (21.8)</td>
<td>.55</td>
</tr>
<tr>
<td>40-54</td>
<td>4 (40.0)</td>
<td>17 (30.9)</td>
<td></td>
</tr>
<tr>
<td>55-69</td>
<td>4 (40.0)</td>
<td>13 (23.6)</td>
<td></td>
</tr>
<tr>
<td>70-84</td>
<td>1 (10.0)</td>
<td>13 (23.6)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>7 (70.0)</td>
<td>34 (61.8)</td>
<td>.73</td>
</tr>
<tr>
<td>Female</td>
<td>3 (30.0)</td>
<td>21 (38.2)</td>
<td></td>
</tr>
<tr>
<td>Positive sign at baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head heave</td>
<td>3 (30.0)</td>
<td>40 (72.7)</td>
<td>.02</td>
</tr>
<tr>
<td>Head thrust</td>
<td>4 (40.0)</td>
<td>50 (90.9)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Head shake</td>
<td>9 (90.0)</td>
<td>53 (96.4)</td>
<td>.40</td>
</tr>
<tr>
<td>Vibration</td>
<td>6 (60.0)</td>
<td>46 (82.5)</td>
<td>.08</td>
</tr>
<tr>
<td>Treatment given</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>8 (80.0)</td>
<td>33 (63.5)</td>
<td>.47</td>
</tr>
<tr>
<td>No</td>
<td>2 (20.0)</td>
<td>19 (36.5)</td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: HH, head-heave test; HS, head-shake test; HT, head-thrust test; Vib, vibration test. Value calculated using Fisher exact test.*

Table 2. Results of 4 Bedside Tests at Baseline

<table>
<thead>
<tr>
<th>Test Pair</th>
<th>OR</th>
<th>LCL a</th>
<th>UCL a</th>
<th>(P) Value a</th>
</tr>
</thead>
<tbody>
<tr>
<td>HT-HH</td>
<td>24.9</td>
<td>8.54</td>
<td>72.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HT-HS</td>
<td>19.5</td>
<td>7.60</td>
<td>49.9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HT-Vib</td>
<td>15.2</td>
<td>7.36</td>
<td>31.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HH-HS</td>
<td>13.8</td>
<td>7.29</td>
<td>28.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HH-Vib</td>
<td>7.91</td>
<td>3.47</td>
<td>18.1</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Abbreviations: HH, head-heave test; HS, head-shake test; HT, head-thrust test; Vib, vibration test. Value calculated using Fisher exact test.*

Table 3. Pairwise Odds Ratios for a Positive Test Predicting a Positive Test, All Persons, Across All Time Points

<table>
<thead>
<tr>
<th>Test Pair</th>
<th>OR</th>
<th>LCL a</th>
<th>UCL a</th>
<th>(P) Value a</th>
</tr>
</thead>
<tbody>
<tr>
<td>−HT/−HH</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>−HT/−HH</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>+HT/−HH</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>+HT/+HH</td>
<td>1</td>
<td>4</td>
<td>36</td>
<td>41</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>1</td>
<td>10</td>
<td>52</td>
</tr>
</tbody>
</table>

An abnormal caloric response (paresis) with normal results of bedside examination was noted in 2 patients at 1-month follow-up, 3 patients at 3 months, and 2 patients at 6 months. However, 6 patients had a normal caloric response and at least 1 positive sign with a bedside test, primarily with the head-shake or vibration tests. At follow-up, the head-shaking test in 3 patients and vibration test in the other 2 bedside tests showed a response in the opposite direction to that predicted on the basis of loss of function, that is, nystagmus beating toward the side of the lesion.

Four patients developed benign paroxysmal positional vertigo during follow-up. In all 4 patients, this disorder was of the posterior semicircular canal type on the same side as the vestibular neuritis.

Our primary goal was to determine whether a complete and careful bedside clinical examination could be as ef-
fective as the caloric test (the gold standard) in the diagnosis of vestibular neuritis. Our results confirm that, with careful bedside clinical examination, one can almost always diagnose a unilateral peripheral vestibular deficit. This is especially important in those patients evaluated in the emergency department because quantitative caloric testing is rarely immediately available. Careful anamnesis for vascular risk factors and a general neurologic examination are required to rule out the common finding of a vertebrobasilar infarction as an acute peripheral vestibular deficit.9,10 Acute spontaneous prolonged vertigo without any other accompanying neurologic or audiologic signs and symptoms may also occur in patients with cerebellar stroke (pseudovestibular neuritis). Magnetic resonance imaging should be performed to rule out a central lesion in the presence of (1) nystagmus with central features, such as a directional changing gaze-evoked nystagmus, or (2) unidirectional spontaneous nystagmus with normal head-impulse test and normal caloric test results.

As is usually the case, at about 1 month after treatment, most patients showed no spontaneous nystagmus, implying either peripheral recovery or central com-

<table>
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<th>Table 4. Multivariate Results of Hazard Model, Adjusted for Month</th>
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<tbody>
<tr>
<td>Baseline Characteristic</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Positive head-thrust sign</td>
</tr>
<tr>
<td>Positive head-heave sign</td>
</tr>
<tr>
<td>Positive head-shake sign</td>
</tr>
<tr>
<td>Positive results of vibration test</td>
</tr>
<tr>
<td>Treatment at baseline</td>
</tr>
</tbody>
</table>

Abbreviations: CL, confidence limits; HR, hazard ratio.
high-acceleration head heaves. though this has not been studied with high-frequency, may be more easily compensated than rotational VOR,11,12 retic side. There is also evidence that translational VOR cades between the response on the normal and the pa-
jects with normal findings, who typically exhibit small corrective saccades in both directions. Thus, one must appreciate an asymmetry in corrective saccades to con-
trols may also account for differences between the 2 tests. The ability to store activity in the central velocity storage mechanism may differ among patients and in a single pa-
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result from the 4 bedside tests varied considerably among patients, though the vibration test and head-shake test were the most sensitive. Results of the head-impulse and head-
heave tests correlated highly across time, though the head-
P Baseline Characteristic HR (95% CL) Hazard Ratio, Assume Patients Who Dropped Out Did Not Recover Hazard Ratio, Assume Patients Who Dropped Out Recovered P Value P Value
Positive head-thrust sign 0.32 .02 0.26 .009
Positive head-heave sign 0.38 .01 0.48 .11
Positive head-shake sign 0.21 .21 0.82 .87
Positive results of vibration test 0.30 .02 0.37 .05
Treatment at baseline 2.34 .03 2.56 .96

Table 6. Association of Bedside Tests With Caloric Test (Time to Event Analysis With Time-Dependent Covariates), Adjusted for Month and Treatment at Baseline

Baseline Characteristic HR (95% CL) P Value
Positive head-thrust sign 0.08 (0.009, 0.68) .002
Positive head-shake sign 0.23 (0.06, 0.82) .01
Positive results of vibration test 0.36 (0.08, 1.54) .14

Abbreviations: CL, confidence limit; HR, hazard ratio.

Abnormal results with the head-shake test depend on (1) asymmetry in peripheral vestibular input, which is best elicited with high-speed head shaking owing to Ewald’s second law; (2) storage of this asymmetric activity in central structures during head shaking; and (3) decay of the stored activity after the head stops moving. In the acute phase, central velocity storage may be impaired; thus, head-shaking nystagmus may be absent or even in the wrong direction.13,14 Vibration-induced nystagmus, however, probably depends on direct stimulation of the vestibular end organ on both sides because the stimulus is transmitted through the bone to both labyrinths; thus, if one side is less responsive, the other will predominate.13 Vibration-induced nystagmus also de-

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vere caloric deficit. Milder degrees of paresis, as reflected in milder caloric response abnormalities, are still revealed by the relatively sensitive head-shake and vibration tests. Especially in the chronic phases, however, head-shaking or vibration-induced nystagmus may be in the wrong direction, perhaps reflecting mechanisms similar to those that underlie spontaneous nystagmus that is in the wrong direction during recovery (so-called recovery nystagmus). Thus, these tests alone cannot reliably be used to identify the side of the lesion.

As described in our previous study, both the head-impulse and head-heave tests have good prognostic value if these signs were absent. We found that negative results of the vibration test in the acute phase of disease is a strong predictor of a high chance for recovery. The absence of head-shaking nystagmus does not seem to be of prognostic value, possibly because velocity storage may be severely depressed, with severe acute unilateral loss. Accepting treatment seemed to have an almost 3-fold effect on chance for recovery. We emphasize, however, that this assumption of tied or discrete data with the LOGISTIC, PROBIT, and GENMOD procedures. In: Survival Analysis Using the SAS system: A Practical Guide. Cary, NC: SAS Institute Inc; 1998.


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