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ORIGINAL ARTICLES

Prevalence of post-headshake nystagmus in patients with caloric deficits and vertigo

JOEL A. GOEBEL, MD, and PHILIP GARCIA, MD, St. Louis, Missouri

Post-headshake nystagmus (PHN) has recently been described as a clinically useful physical sign implying uncompensated asymmetric input from the vestibular end organs. A rapid 20-second headshake and sudden stop produces a jerk nystagmus of 5- to 20-second duration in certain individuals with symptoms suggestive of a peripheral vestibulopathy. This retrospective review of 214 patient evaluations was undertaken to study the associations between post-headshake nystagmus, caloric deficits after bithermal binaural irrigation, and the presence of vertigo. Both clinical observation of the nystagmus with eyes open (PHN-OBS) and routine EOG recording with eyes closed (PHN-EOG) were used. In patients with unilateral caloric deficits, 42% (18 of 43) had PHN-EOG, compared with 18% (3 of 17) in patients with bilateral dysfunction and 15% (23 of 154) in patients with normal calorics ($p < 0.001$). In similar fashion, 26% (32 of 124) of patients with vertigo (recent or past) had PHN-EOG compared to 13% (12 of 90) of patients without vertigo ($p < 0.03$). Finally, of 110 cases with both PHN-EOG and PHN-OBS performed, 45% (9 of 20) with PHN-EOG also had PHN-OBS, as opposed to only 4% (4 of 90) without PHN-EOG displaying PHN-OBS ($p < 0.0001$). We conclude that the prevalence of post-headshake nystagmus is increased in patients with either a unilateral caloric deficit or a history of true vertigo, and is best detected in the absence of vision.
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The diagnosis of unilateral vestibular dysfunction is suspected in patients with attacks of true vertigo and supported by the presence of unidirectional nystagmus on physical examination and a unilateral caloric deficit with bithermal binaural caloric stimulation and electro-oculographic (EOG) recording. In some cases, however, spontaneous nystagmus is absent as a result of central compensation. In such cases, the documentation of a peripheral vestibulopathy relies heavily on the finding of a unilateral deficit after caloric stimulation.

The present study investigated the use of an additional test, post-headshake nystagmus (PHN), as a clinical and laboratory tool for documenting peripheral vestibular dysfunction. Unidirectional or biphasic nystagmus on EOG recording after a high-frequency (1 to 2 Hz) 20-second headshake maneuver has been described in patients with known unilateral vestibular ablation[1] and in patients with caloric asymmetries.[2,3] In this study, the prevalence of either observed or EOG-

From the Vestibular and Oculomotor Laboratory, Department of Otolaryngology–Head and Neck Surgery, Washington University School of Medicine.
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Reprint requests: Joel A. Goebel, MD, 517 South Euclid, Box 8115, St. Louis, MO 63110.
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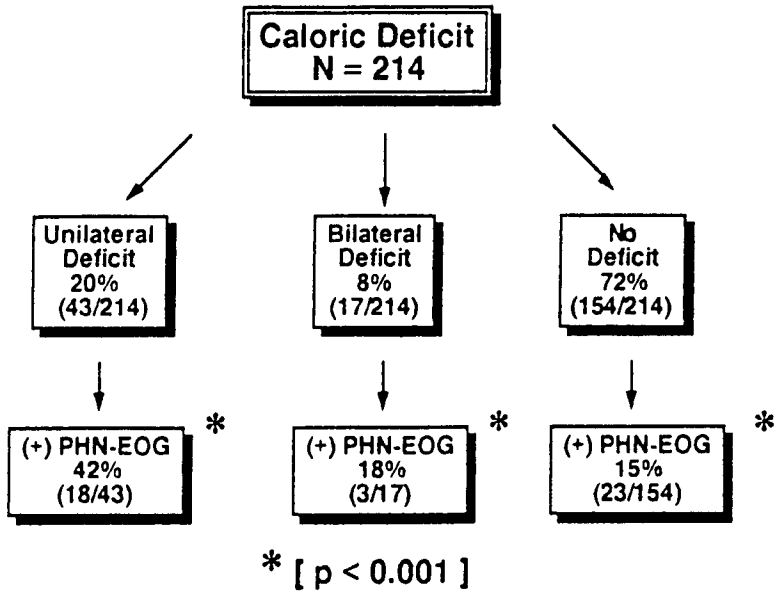

Fig. 1. Caloric deficits vs. EOG-recorded post-headshake nystagmus (PHN-EOG).

recorded post-headshake nystagmus in two patient populations—(1) patients with unilateral or bilateral caloric deficits and (2) patients with a history of vertigo—was investigated.

METHODS

The charts of 214 patients, ages 9 to 83 years (median age, 55 years), seen in the Vestibular and Oculomotor Laboratory for evaluation of dizziness, were randomly selected for review. A thorough history, including a written questionnaire, was obtained on all patients. Emphasis was placed on determination of whether these patients had experienced vertigo (defined as a distinct illusion of motion within the visual surround) and, if so, whether such sensations of vertigo occurred recently (within 1 month) or in the past (greater than 1 month) preceding the examination. All patients had a full otologic and neurotologic examination, including cranial nerve, oculomotor, cerebellar, and gait testing performed by the senior author (J.A.G.) or Dr. Gary Paige. In 110 of the 214 cases, a 20-second high-frequency (1 to 2 Hz) low-amplitude (15 to 20 degrees each direction) headshake maneuver with eyes closed was performed passively on the patient by one of the authors (J.A.G.) and any post-headshake nystagmus (three or more consecutive beats) observed with eyes open after the maneuver was scored as a positive response.

After the clinical examination, eye movement recordings were performed (bitemporal EOG) after bithermal binaural irrigation (30° C and 44° C water) with the Brookler-Grams closed-loop irrigation system. Peak slow-component eye velocity was measured, and labyrinthine asymmetry (LA) was derived from the following formula:

$$\text{LA (\%)} = \frac{(RW + RC) - (LW + LC)}{(RW + RC + LW + LC)} \times 100\%$$

In this formula, *RW* = right warm, *RC* = right cool; *LW* = left warm; and *LC* = left cool. An LA of 25% or more was considered significant. A sum-total slow-component eye velocity of 10 degree/second or less from each ear (two irrigations) or 20 degrees/second for both ears (four irrigations) was considered a bilateral deficit.

In all cases EOG recording, a 20-second headshake maneuver (1 to 2 Hz, 15- to 20-degree amplitude each direction) with eyes closed was performed actively by the patient, and any subsequent nystagmus was recorded immediately upon cessation. A criterion described by Wei et al.[2] of three distinct consecutive nystagmus beats greater than 2.5 degrees/second (beginning within 5 seconds after cessation of the headshake maneuver) was used to identify a positive result. In addition, the initial direction and any reversal in direction of the PHN was noted.

For data interpretation, the following associations were analyzed:

1. The prevalence of EOG-recorded (PHN-EOG) nystagmus in patients with normal vs. abnormal calorics (unilateral and bilateral deficit groups analyzed separately).

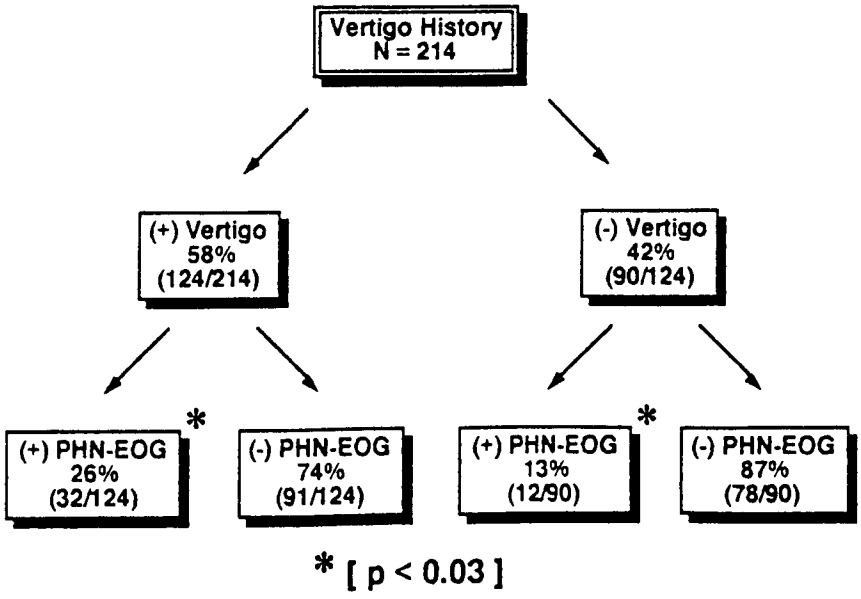

Fig. 2. History of vertigo vs. EOG-recorded post-headshaking nystagmus (PHN-EOG).

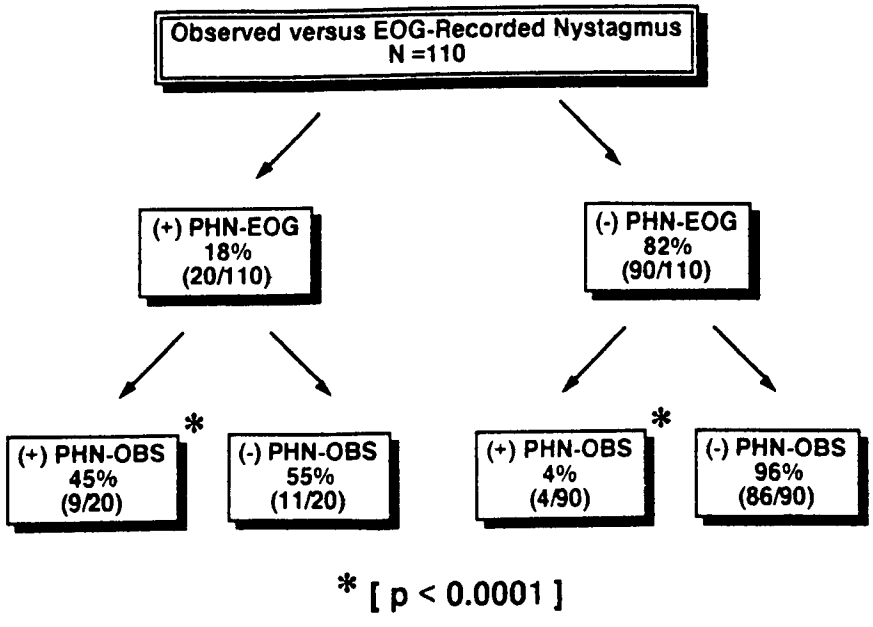

Fig. 3. Observed (PHN-OBS) vs. EOG-recorded (PHN-EOG) post-headshake nystagmus.

2. The prevalence of EOG-recorded (PHN-EOG) nystagmus in patients with and without a history of vertigo (recent or past).
3. The prevalence of observed post-headshake nystagmus (PHN-OBS) in subjects with and without PHN-EOG.

For each of the above associations, chi square analysis or Fisher's exact test was used to determine a p value, with $p < 0.05$ considered significant.

RESULTS

Headshake Nystagmus vs. Caloric Deficits

Of the 214 cases reviewed, 43 had a significant unilateral caloric deficit (LA) and 17 had bilateral deficits. In the group with unilateral deficits, 42% (18 of 43) had EOG-recorded post-headshake nystagmus (PHN-EOG). In contrast, only 18% (3 of 17) of cases with bilateral dysfunction and 15% (23 of 154) of cases with normal caloric tests exhibited PHN-EOG. These dif-

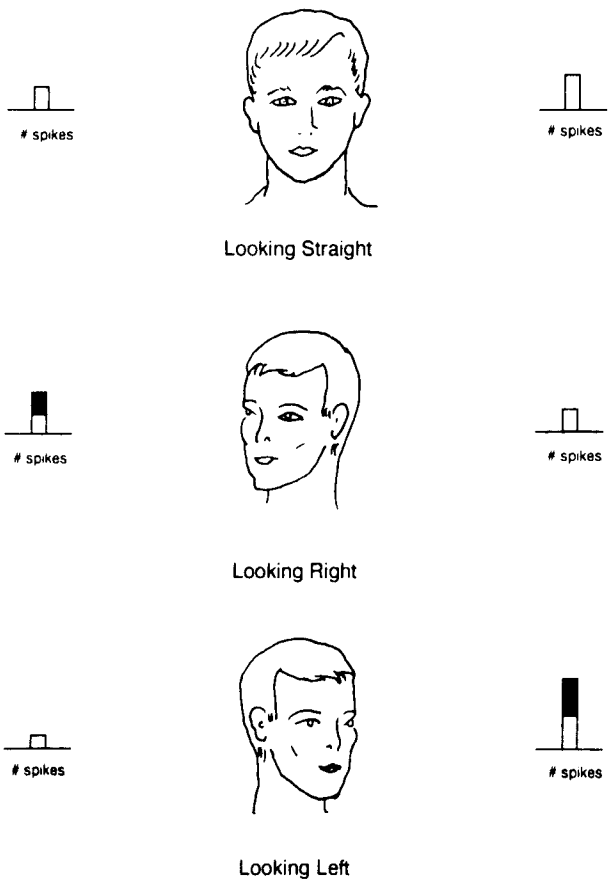

Fig. 4. Ewald's second law and post-headshake nystagmus. Right-sided labyrinthine injury leads to lower neural activity on the right at rest *(top figure)*. Head movement to the right produces limited ampullopetal stimulation and normal left-sided ampullofugal inhibition *(middle figure)*. Head movement to the left creates a strong ampullopetal response and diminished right ampullofugal response *(bottom figure)*. Repeated bidirectional headshake leads to repeated asymmetric right-sided input, producing nystagmus to the right.

ferences were highly significant ($p < 0.001$) (see Fig. 1).

Headshake Nystagmus vs. History of Vertigo

A history of vertigo, either recent or past, was recorded in 58% of the 214 cases. Within this group, 26% (32 of 124) exhibited PHN-EOG. In the group of patients without vertigo, however, only 13% (12 of 90) showed PHN-EOG ($p < 0.03$). There was no significant difference of PHN-EOG prevalence in the patients with vertigo based solely on timing of their attacks before examination (see Fig. 2).

Observed vs. Recorded Headshake Nystagmus

In 110 cases, a passive headshake maneuver and visual observation for subsequent nystagmus was performed in addition to the active headshake maneuver with EOG recording. In 20 cases with PHN-EOG, 45% (9 of 20) also showed observed post-headshake nystagmus (always in the same direction). In the remaining 90 cases without PHN-EOG, however, only 4% (4 of 90) exhibited PHN-OBS ($p < 0.0001$) (see Fig. 3).

DISCUSSION

The first account of a headshake-induced nystagmus was described by Vogel[4] in 1932 as a form of provoked spontaneous nystagmus. In 1964, Kamei et al.[5] reported the headshake test as a means of identifying peripheral vestibular dysfunction. In a subsequent study, Kamei[3] described a biphasic post-headshake nystagmus seen in all 18 patients studied with unilateral vestibular dysfunction. With EOG recording, the initial phase of the nystagmus lasted from 5 to 15 seconds and always beat away from the paretic side (paretic nystagmus). A second phase nystagmus of lower amplitude but opposite direction followed and lasted up to 30 seconds (recovery nystagmus). In a later account, Demer[6] proposed the explanation of asymmetric central velocity storage as one possible mechanism for this biphasic nystagmus; that is, the perserverence of asymmetric peripheral vestibular input within the brainstem after rapid repeated bidirectional head movement produces nystagmus.[6] Hain et al.[1] expanded this explanation to include Ewald's second law.[7,8] In their argument, they proposed that ampullopetal excitation from the intact labyrinth was not counterbalanced by ampullopetal input from the damaged side during repeated to and fro motion and, therefore, the brainstem was repeatedly receiving asymmetric stimulation during the headshake (Fig. 4). The central brainstem mechanism responsible for prolonging the end-organ vestibular signal (central velocity storage mechanism) was perseverating this asymmetric input after the maneuver was complete, leading to the observed nystagmus beating initially in the direction of the intact ear. The second phase nystagmus, they explained, represented a compensatory change in the velocity storage, leading to a reversal of the orginal nystagmus. In their study of six subjects with surgically created unilateral vestibular lesions, they were able to record a low-amplitude post-headshake nystagmus with reversal in all subjects in the dark using a scleral search coil technique. One of seven normal subjects in their study, however, also exhibited a post-headshake nystagmus. They concluded that post-headshake nystagmus was a low-amplitude response to repeated head rotation, which was closely related to but not diagnostic of peripheral vestibular dysfunction. In a recent study, Wei et al.[2] examined 108 subjects with EOG recording (eyes closed), including 30 cases of unilateral dysfunction. Post-headshake nystagmus after active head rotation was recorded in 40% of these cases and three fourths of the time the nystagmus was directed toward

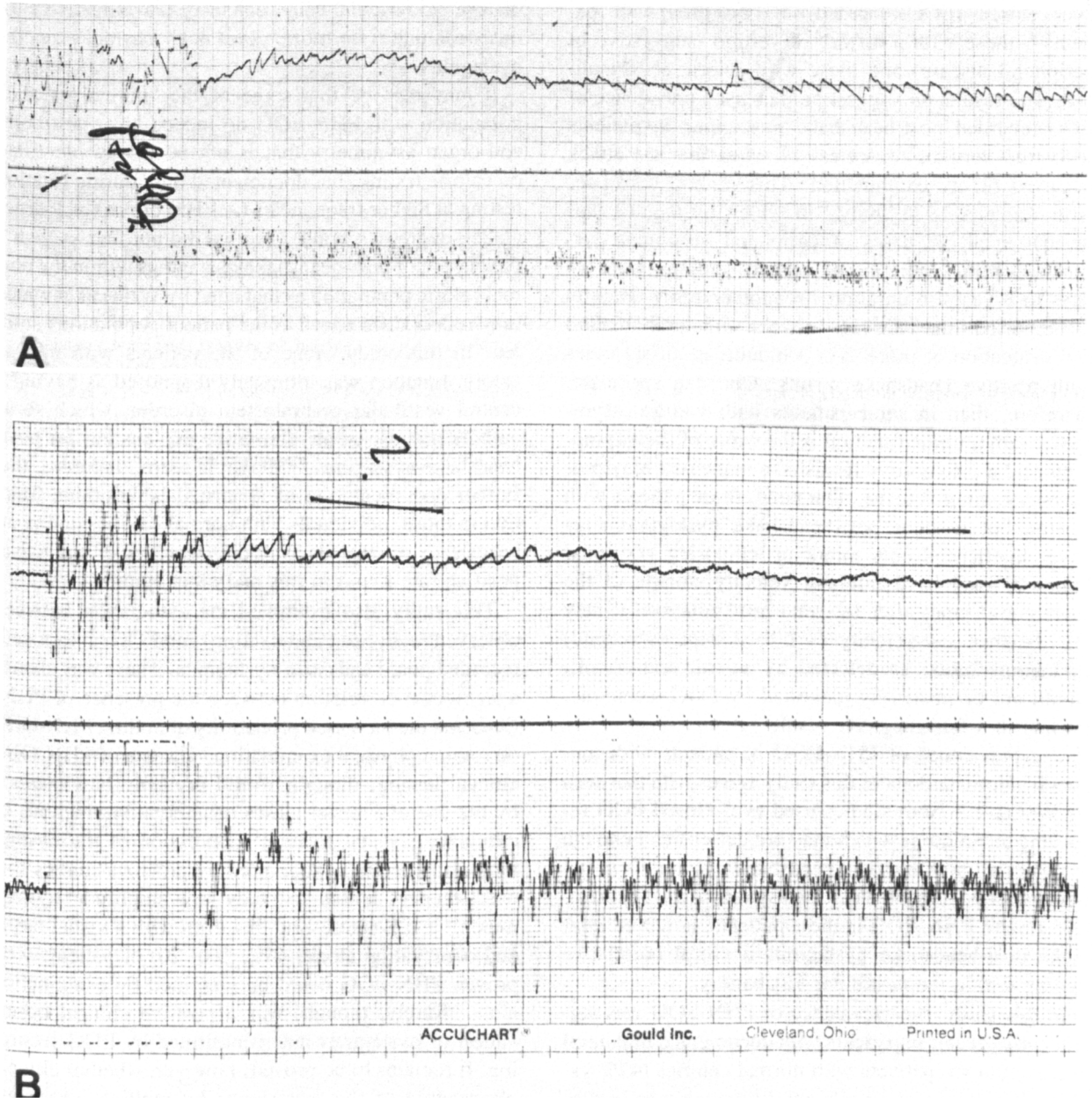

Fig. 5. EOG recordings in two patients with post-headshake nystagmus. **A,** Biphasic nystagmus beating initially to the left; **B,** monophasic nystagmus beating to the left. *Top trace,* eye position; *bottom trace,* eye velocity, standard EOG convention.

the intact ear withour reversal. In their population with normal calorics, however, they recorded 32% (24 of 75) with PHN-EOG; in addition, one of three central cases had a biphasic post-headshake nystagmus using passive (rather than active) head rotation with Frenzel glasses and scleral search coil technique to better evaluate the nature of the nystagmus.

Although most reports have emphasized the association of post-headshake nystagmus with peripheral vestibular disease, in 1974 Kamei and Kornhuber[9] identified nystagmus after headshake in 25% of patients with central lesions, as seen with Frenzel lenses. Unfortunately, there was no reference to caloric responses in these patients to rule out concurrent peripheral vestibular dysfunction.

The present study was performed to further evaluate the use of the post-headshake test performed passively with eyes open (clinical observation) and actively with

eyes closed (EOG-recorded) in two groups of patients—those with a history of vertigo (suggestive of peripheral disease) and those with caloric abnormalities. The results *do* suggest an increased prevalence of EOG-recorded post-headshake nystagmus in patients with true vertigo, regardless of when the last attack occurred before examination. This finding correlates with Kamei's 1975 study,[3] in which nystagmus was present in all 22 cases of peripheral vestibular dysfunction with symptoms of vertigo, ranging from 14 days to 8 years in duration. In another study, Hain et al.[1] reported time intervals between unilateral ablation and evaluation of more than 3 months in all six cases with positive headshake results. One can speculate, therefore, than in some patients with vertigo, asymmetric labyrinthine function is the cause of their symptoms and a provocative headshake maneuver may produce nystagmus. On the other hand, those patients with vertigo but without post-headshake nystagmus may have experienced only minor or temporary vestibular injury or may have minimal velocity storage of the asymmetric input after repeated head rotation. It may also be that low-velocity (<1 to 2 degrees/second) nystagmus beats, as detected by scleral search coil, would not be positively identified by EOG technique, leading to a false-negative result.

The prevalence of PHN-EOG in patients with unilateral caloric deficits in this study agrees with that seen by Wei et al.[2] Both studies used eyes closed EOG recording techniques, which may not be sensitive enough to detect all possible post-headshake nystagmus in patients with documented caloric asymmetries. The identification of PHN-EOG in these patients, however, *does* make post-headshake nystagmus a useful correlative finding within the vestibular test battery.

In this study, the prevalence of PHN-EOG was significantly greater in patients with documented unilateral dysfunction vs. patients with normal calorics (42% vs. 15%). There was no significant difference seen in subjects with bilateral dysfunction. Furthermore, in fifteen of the eighteen caloric deficits, the nystagmus was directed toward the side with the stronger caloric response. This apparent localizing feature of post-headshake nystagmus agrees with that seen in the majority of previous reports.[2,3,6] Spindler and Schiff,[10] however, reported a number of cases of biphasic headshake nystagmus directed initially toward the paretic ear in contrast to the more frequently observed pattern. Regarding the biphasic nature of post-headshake nystagmus, the majority of tracings in this study failed to show a clear biphasic response, except in isolated instances (Fig. 5, *A* and *B*). Further evaluation of this technique with eyes open in darkness and scleral search coil recording of eye movement may enhance the sensitivity of this technique to more accurately characterize the nystagmus.

It may also be that some of the "normal caloric" population with PHN-EOG do indeed have vestibular end-organ asymmetry that is not positively identified on caloric testing, but documented only during rotation testing at higher frequencies (>1 Hz) or peak velocities (>250 degrees/second). But we did not find as high a prevalence of post-headshake nystagmus within the normal caloric population as that cited by Wei et al.,[2] which may reflect differences in the patient populations studied. In this study, none of the patients with normal caloric function was ultimately diagnosed as having a central vestibular or brainstem disorder, which some authors propose as an alternative explanation for post-headshake nystagmus.[9,10] It would seem, therefore, that further documentation of abnormal labyrinthine function in those individuals with normal calorics and post-headshake nystagmus would indeed lower the false-positive rate found in this and other studies.

This study also examined the association between observed (eyes open and fixating) and EOG (eye closed) recorded post-headshake nystagmus. There was clearly a significant correlation between the presence of PHN-EOG and the increased probability of finding PHN-OBS on routine physical examination. The use of PHN-OBS clinical testing, though, would probably be enhanced by the use of Frenzel lenses or alternative methods of eliminating visual fixation suppression of the nystagmus. This notion is supported by previous studies that used Frenzel lenses to study low-amplitude nystagmus.[2,10,11] It is interesting that even with a visual target and observation alone, 45% (9 of 20) of patients with proven PHN-EOG could be identified by examination alone. Rarely, though, was a positive clinical observation of nystagmus not reconfirmed by EOG recording. It remains to be proved, however, whether clinical observation of this nystagmus by multiple observers yields reproducible positive results.

The question of whether active vs. passive head rotation is the ideal stimulus for this examination was not addressed in this study. In fact, the clinical examination was performed using passive rotation by the examiner and the EOG-recorded protocol used active head rotation by the patient. Further prospective evaluation of this issue is needed to determine whether active or passive rotation makes any difference.

We therefore conclude that the prevalence of post-headshake nystagmus is increased in patients with unilateral caloric deficits or symptoms of vertigo suggestive of peripheral labyrinthine dysfunction. This simple maneuver may enhance the clinical and laboratory eval-

uation of patients with suspected labyrinthine dysfunction and is probably best performed in the absence of visual fixation.

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