ASYMMETRIC OPTOKINETIC AFTERRESPONSE IN PATIENTS WITH VESTIBULAR NEURITIS

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Abstract—The symmetry of primary and secondary optokinetic afternystagmus (OKAN I and OKAN II, respectively) was studied in 14 patients with vestibular neuritis, as well as in 50 normals. The patients were examined at onset of symptoms and at follow-up 3 and 12 months later. At onset, OKAN was found mainly to reflect the spontaneous nystagmus. Although the spontaneous nystagmus disappeared in all patients within 3 months, both OKAN I and OKAN II was asymmetric at the 3- and 12-month check-ups. OKAN beating toward the lesioned ear was weaker than the OKAN beating toward the healthy ear. Thus, the asymmetric vestibular function was reflected not only in the OKAN I, but also by an asymmetry in OKAN II. Between the 3- and 12-month check-ups, asymmetry in OKAN declined, even among those patients who showed no improvement in caloric response during that time. The decreasing asymmetry in OKAN with time after lesion was, however, related to the disappearance of a positional nystagmus. Hence, the results may be interpreted as suggesting OKAN not only to be affected by vestibular side-difference, but also to be modified by the process responsible for vestibular compensation following a peripheral vestibular lesion.

Keywords—optokinetic afternystagmus; primary; secondary; vestibular neuritis.

Introduction

In cases of unilateral sensorineural hearing loss, or in patients suffering from unsteadiness or vertigo, it is essential to evaluate the functional symmetry of the vestibular end-organs and nerves. Stimulus to the vestibular end-organ in the natural state consists of linear or angular acceleration, detected by both labyrinths simultaneously. However, rotatory tests have so far proved to be of limited success in indicating the extent of vestibular side-difference in man (1). In attempting to quantify vestibular side-difference, investigators mainly depend on the caloric test, though the magnitude of caloric response may only be loosely related to the severity of peripheral lesion. As caloric irrigation tests only the low frequency response, chiefly of the lateral canal and its afferents (2,3), complementary quantitative tests of vestibular side-difference would be useful.

Optokinetic nystagmus (OKN) is triggered by image slip on the retina. In man, two mechanisms evoke OKN: a cortical (or fast) mechanism, which may be identical with the pursuit system (4), and a subcortical (or slow) mechanism (5). The vestibular nuclei are important premotor structures in the subcortical component of OKN and receive input during retinal image slip (6–8). A summation of velocity information from the retina and from the vestibular nerves has been suggested to take place in the vestibular nuclei (9,10). Thus, the subcortical component of OKN is dependent on the status of received peripheral vestibular input and could, theoretically, be used to demonstrate vestibular side-difference.

Although, the subcortical component of OKN is only accessible to study if the cortical
component is absent, as it is in certain species (11), it is reflected in the primary optokinetic afternystagmus (OKAN I) (12). During optokinetic stimulation, activity related to slow phase eye-velocity in the subcortical pathways of OKN is stored by the “central velocity storage integrator” (12-14). When the optokinetic stimulus is abolished by darkness, the integrator discharges, generating OKAN I that moves in the same direction as the previous OKN.

OKAN I is sometimes followed by a nystagmus beating in the opposite direction (OKAN II). The mechanism responsible for OKAN II has been suggested to be a “central adaptation operator” (15-17). Although, the anatomical site of this operator is not known, in animal experiments it was found that OKAN II is mediated by the same vestibular nuclei neurons as OKAN I (18,19). Thus, the symmetry of OKAN II might also be affected by asymmetric peripheral vestibular activity.

The aims of the present study were to ascertain whether asymmetric peripheral vestibular function is reflected in an asymmetry of OKAN, and if so, whether asymmetry in OKAN changes when a patient compensates for the lateral vestibular difference. An additional aim was to evaluate the clinical significance of asymmetry in OKAN by comparing the findings in patients with those in controls.

Materials and Methods

Subjects

Those studied were 14 patients with vestibular neuritis (6 males and 8 females, mean age 40.4 years, range 15-65) recruited consecutively. Diagnosis was based on a sudden onset of severe vertigo with spontaneous nystagmus and unilaterally reduced caloric response (20,21). Patients with cardiovascular or metabolic disease, or on any form of continuous medication, were excluded from the study, as were patients who, either at onset or at follow-up, had histories or signs of cochlear or central nervous disorders, or who were more than 65 years old. None of the patients reported previous episodes of vertigo.

The patients were examined at onset of disease (mean 6.0 days, SD 4.6), and again both 3 months later (mean 100.4 days, SD 18.9) and 12 months later (mean 364.9 days, SD 53.6). In two of the 14 patients, the optokinetic test was not included in the electro-nystagmographic examination at onset of disease.

Normative values in optokinetic afterresponse were determined in 50 healthy subjects (24 females, 26 males), ranging in age from 24 to 65 years, none of whom had any history of cochlear, vestibular, or central nervous disorders. The 50 healthy subjects were divided into two groups by age: group 1 comprising those below the age of 40 (13 females, 13 males) and group 2 those over 40 (11 females, 13 males).

Test Procedure

Horizontal optokinetic stimulation was administered by means of a whole-field optokinetic drum (diameter 180 cm), with alternating black and white stripes, 10 cm broad. During the test, the subject was seated in a chair with the head fixed by an occipital support (eye-ear axis horizontal), and instructed to stare, but not to try to follow the moving stripes. Eye movements were recorded with DC electro-oculography (EOG) and transcribed simultaneously by ink-jet recorder (Mingograph 81, Siemens-Elema, Stockholm). The optokinetic drum was accelerated up to a constant velocity of 90°/s in complete darkness, after which light was turned on for 60 seconds, recordings being continued until 60 seconds after the light was extinguished.

The patients, but not the controls, were further investigated. Spontaneous and positional nystagmus in darkness were recorded with the subject in the upright position and after gently placing the patient in the supine position (ie, facing upward). The supine position was maintained for at least 30s. The patients were instructed to look straight ahead during the recordings for spontaneous and positional nystagmus, and conversation in sequences with the investigator was used to support maintenance of alertness throughout
the test. Voluntary eye movements and caloric response were also evaluated by EOG. In addition, pure tone audiometry and a full clinical otoneurological work-up were done in all patients.

**Analysis of Recordings**

Values for eye movement variables were obtained manually from the recordings. Persistent nystagmus with a slow phase eye velocity (SPEV) greater than 2°/s in the upright position was described as spontaneous nystagmus, and that in the supine position as positional nystagmus.

OKAN I was evaluated as the SPEV moving in the same direction as the previous OKN two seconds (OKAN₂ₙ) after the end of the optokinetic stimulus (ie, lights off), and as the response decay time (OKAN₃ₙ) for the SPEV to reach 2°/s (measured at the final fast phase of nystagmus to be preceded by a slow phase with a velocity exceeding 2°/s). Nystagmus (SPEV > 2°/s) opposite in direction to, and following, OKAN I was considered to be significant OKAN II, and was characterized by its maximal SPEV.

In the patient group, asymmetry of OKAN I was calculated as the quotient (Q) between OKAN I with the fast phase beating toward the healthy ear (HE) and OKAN I beating toward the lesioned ear (LE): Q = (OKAN₃ₙHE - OKAN₃ₙLE) / (OKAN₃ₙHE + OKAN₃ₙLE). In normals, Q was calculated as the absolute asymmetry between OKAN I beating towards right (R) and left (L): Q = ABS((OKAN₃ₙR - OKAN₃ₙL) / (OKAN₃ₙR + OKAN₃ₙL)). If OKAN I was absent in both directions, the 2-value was said to be zero.

The caloric ratio was calculated from the SPEV produced by irrigation: (44°R + 30°R) - (44°L + 30°L) / (44°R + 30°R + 44°L + 30°L), a caloric ratio > 0.20 being considered to be a significant caloric side-difference.

**Statistics**

Among the 50 normal subjects, the effect of age and sex, both on the symmetry and on the strength of OKAN I, was tested with the Mann–Whitney test and two-way analysis of variance. In the patient group, the difference between OKAN I beating toward the healthy and the lesioned ear, as well as difference in OKAN I between the 3- and 12-month check-up, were evaluated using Student’s t test for paired data, the t test being applied where normal distribution was not rejected by the Wilk–Shapiro test.

The Mann–Whitney test was used both in comparing absolute values for asymmetry in OKAN I between the patient and the normal group, and in comparing OKAN I between patients with versus without an OKAN II beating toward the healthy ear.

Kendall’s tau was used to test for correlation between the discrete variable, presence-absence of positional nystagmus, and variables of OKAN (Q for OKAN₂ₙ, Q for OKAN₃ₙ, and maximal SPEV of OKAN II beating toward the healthy ear). In addition, linear regression analysis and correlation analysis was used to test for correlation between OKAN₂ₙ and SPEV of spontaneous nystagmus at onset of the disease. A difference or regression of P < 0.05 was considered significant.

**Results**

**Normals**

In response to optokinetic stimulation, 5 normal subjects showed complete absence of OKAN, whereas in 8 subjects OKAN I symmetrically outlasted the recordings irrespective of the direction of drum rotation. There was no significant difference in values for OKAN I between subjects who were first exposed to drum rotation toward the right (n = 23) and those first exposed to drum rotation toward the left (n = 27). The overall mean (n = 50) for OKAN₂ₙ was 8.00°/s (SD 6.1) and that for OKAN₃ₙ 27.6 s (SD 21.6). Analysis of variance (ANOVA) using the variables age group and sex showed a significant relationship to exist between these variables and the strength of OKAN I among normals (OKAN₂ₙ; P < 0.05, OKAN₃ₙ; P < 0.01). Females under 40 years of age manifested sig-
nificantly higher mean OKAN_{2s} and longer mean OKAN_{dur} than any other subgroups, male or female. Absolute values for asymmetry in OKAN I among the 50 normal subjects (ie, Q for OKAN_{2s} and OKAN_{dur}) are shown in Figure 1. The 90th percentile values of Q for OKAN_{2s} and OKAN_{dur} were 0.39 and 0.70, respectively. Asymmetry of OKAN I did not increase with increasing age. OKAN II was not observed in any of the 50 normal subjects.

**Patients at Onset of Symptoms**

At onset of vestibular neuritis, all patients manifested a caloric ratio greater than 0.20 (mean 0.89, SD 0.12) and spontaneous nystagmus in darkness beating toward the healthy ear (mean SPEV 10.9°/s, SD 5.9). Following OKN with fast phases beating toward the lesioned ear, OKAN changed direction in all patients within 2 s after “lights off.” Hence, Q for OKAN was always 1.0 at onset of the disease, as all tested patients showed OKAN I beating toward the healthy ear but no OKAN I beating toward the lesioned ear. Since the duration of OKAN I (OKAN_{dur}) beating toward the healthy ear outlasted the recordings in all patients, the only “informative” parameter of OKAN I at onset of vestibular neuritis was the initial SPEV of OKAN I (OKAN_{2s}) beating toward the healthy ear (mean 20.6°/s, SD 7.9). This variable correlated with the SPEV of spontaneous nystagmus (r = 0.63, p < 0.05), though OKAN_{2s} was significantly greater (P < 0.001).

**Patients at Follow-up**

**Caloric ratio (CR).** At follow-up, 6 of the 14 patients were found to have recovered normal caloric side-difference (CR ≤ 0.20), all 6 of them having done so before the 3-month check-up. These 6 patients manifested a significant further decrease in CR between the 3- and 12-month check-ups (at 3 mon, CR = 0.11, SD 0.07; at 12 mon, CR = 0.01, SD 0.06, P < 0.05). However, there was no sig-

![Figure 1. Histogram of distribution for a) Q for OKAN_{2s} and b) Q for OKAN_{dur} among 50 normal subjects. Q is calculated from the difference in response to the two directions of drum rotation.](image)
significant difference in caloric ratios between the 3- and 12-month check-ups among the 8 patients who did not recover normal caloric side-difference ($CR = 0.70$, SD 0.31 and $CR = 0.71$, SD 0.27, respectively).

**Spontaneous and positional nystagmus.** None of the patients had spontaneous nystagmus in darkness at the 3- or 12-month follow-up. Nevertheless, a nystagmus beating toward the healthy ear was recorded in the supine position in 7 patients after 3 months (in 2/6 of the patients who recovered normal caloric side-difference, and in 5/8 among those who did not); and after one year, the positional nystagmus beating towards the healthy ear was still present in 5 patients (1/6 and 4/8, respectively). However, neither at the 3-month nor at the 12-month check-up were any cases of nystagmus beating toward the lesioned ear encountered with the patient in the supine position.

**Primary OKAN.** At the 3-month check-up, OKAN I was asymmetric (Tables 1 and 2). The $OKAN_{dur}$ was significantly shorter after an OKN beating toward the lesioned ear than after an OKN beating toward the healthy ear. Among the patients who had not recovered normal caloric side-difference, values for $OKAN_{2s}$ toward the lesioned ear were lower than those for $OKAN_{2s}$ toward the healthy ear. The absolute values for $Q$ among the patients with vestibular neuritis 3 months after onset of disease were significantly higher than those among normals, the difference being significant both for $OKAN_{2s}$ and for $OKAN_{dur}$, both among the patients who recovered normal caloric side-difference ($P < 0.05$ and $P < 0.05$, respectively) and among those who did not ($P < 0.05$ and $P < 0.01$, respectively).

At the 12-month check-up (Tables 1 and 2), no significant asymmetry in OKAN was found among the patients whose caloric side-difference had recovered. Among the patients who had not recovered normal caloric response, OKAN I was still asymmetric. However, in neither of these two subgroups were absolute $Q$ values significantly different from those among normals.

Between the 3- and 12-month check-ups, $Q$ for $OKAN_{2s}$ and $Q$ for $OKAN_{dur}$ decreased (Figure 2). Among the patients who did not recover normal caloric response, a significant decrease in $Q$ for $OKAN_{dur}$ was noted between the 3- and 12-month follow-ups, although there was no sign of improved caloric response in the lesioned ear during the 9-month interval.

**Secondary OKAN.** OKAN II beating toward the healthy ear was observed in 9/14 patients after 3 months (in 4/6 patients who had recovered normal caloric response and in 5/8

| Table 1. Disparity between $OKAN_{dur}$ Beating toward the Healthy versus the Lesioned Ear (HE and LE, Respectively) at the 3- and 12-Month Checkups in Patients with Vestibular Neuritis (Means and SDs Are Given) |
|-----------------|-----------------|-----------------|
| **After 3 months** | **Not recovered** | **Recovered** |
| **(n = 14)** | **(n = 8)** | **(n = 6)** |
| **HE** | 33.1 ± 18.2 | 38.5 ± 20.7 | 26.0 ± 12.5 |
| **LE** | 9.2 ± 12.1 | 10.5 ± 13.4 | 7.5 ± 11.2 |
| **P** | < 0.001 | < 0.01 | < 0.05 |
| **After 12 months** | **(n = 14)** | **(n = 8)** | **(n = 6)** |
| **HE** | 26.9 ± 18.9 | 32.3 ± 17.5 | 19.8 ± 19.9 |
| **LE** | 15.9 ± 16.3 | 15.9 ± 12.3 | 15.8 ± 21.9 |
| **P** | < 0.05 * | < 0.05 | NS * |

*indicates nonnormally distributed differences and the use of the Wilcoxon signed rank test.
patients who had not), and in 4/14 patients after 12 months (1/6 and 3/8, respectively). The max SPEV of the observed OKAN II was in the range of 3° to 8°/s after 3 months and 3° to 5°/s after 12 months. The patients with OKAN II beating toward the healthy ear manifested more pronounced asymmetry in OKAN I, as reflected in greater values for $Q$ both at the 3- and 12-month check-ups (Figure 3). The duration of OKAN I preceding an OKAN II was significantly shorter than that of OKAN I among those who manifested no OKAN II, both at the 3- and 12-month check-ups ($P < 0.01$ and $P < 0.05$, respectively). No case of OKAN II beating toward the lesioned ear was observed.

### Table 2. Disparity between OKAN$^2$S Beating toward the Healthy versus the Lesioned Ear (HE and LE, Respectively) at Onset of Vestibular Neuritis and at 3- and 12-Month Checkups (Means and SDs Are Given)

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Not recovered</th>
<th>Recovered</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At onset</strong></td>
<td>($n = 12$)</td>
<td>($n = 6$)</td>
<td>($n = 6$)</td>
</tr>
<tr>
<td>HE</td>
<td>20.6 ± 7.9</td>
<td>19.2 ± 9.1</td>
<td>22.0 ± 7.0</td>
</tr>
<tr>
<td>LE</td>
<td>0.0 ± 0.0</td>
<td>0.0 ± 0.0</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td><strong>After 3 months</strong></td>
<td>($n = 14$)</td>
<td>($n = 8$)</td>
<td>($n = 6$)</td>
</tr>
<tr>
<td>HE</td>
<td>11.9 ± 4.1</td>
<td>11.8 ± 4.5</td>
<td>12.0 ± 4.0</td>
</tr>
<tr>
<td>LE</td>
<td>6.7 ± 6.9</td>
<td>6.5 ± 7.2</td>
<td>7.0 ± 7.2</td>
</tr>
<tr>
<td>$P$</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>NS</td>
</tr>
<tr>
<td><strong>After 12 months</strong></td>
<td>($n = 14$)</td>
<td>($n = 8$)</td>
<td>($n = 6$)</td>
</tr>
<tr>
<td>HE</td>
<td>12.9 ± 7.5</td>
<td>14.3 ± 9.1</td>
<td>11.2 ± 4.9</td>
</tr>
<tr>
<td>LE</td>
<td>10.1 ± 7.7</td>
<td>11.0 ± 9.7</td>
<td>9.0 ± 4.3</td>
</tr>
<tr>
<td>$P$</td>
<td>&lt; 0.05</td>
<td>&lt; 0.01</td>
<td>NS</td>
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*Figure 2. Quotients a) $Q$ for OKAN$^2$s and b) $Q$ for OKANdur at 3- and 12-month follow-up, showing decreasing asymmetry in OKAN I with time after onset of vestibular neuritis for all 14 patients together ("all"), for patients who recovered normal caloric side-difference ("recoy.") and for those who did not ("not recoy."). Means, SEM, and levels of significance (Student's $t$ test for paired data) are given.
Figure 3. The difference in caloric ratio (CR), Q for OKAN\textsubscript{2s} and Q for OKAN\textsubscript{dur} between patients with versus without OKAN II beating toward the healthy ear. Patients with OKAN II demonstrate a preponderance of OKAN I beating toward the healthy ear, which is reflected in their greater values for Q, both at the 3- and 12-month check-ups (Figures 3a and 3b, respectively). Means, SEM, and levels of significance (Mann-Whitney test) are given.

Relationship between OKAN and positional nystagmus. Rank correlation showed a significant correlation to exist between positional nystagmus and variables Q for OKAN\textsubscript{2s} and maximal SPEV of OKAN II at the 3-month check-up (correlation coefficient \( r = 0.44 \) and \( r = 0.51 \), respectively). At the 12-month check-up, the presence of a positional nystagmus was correlated to Q for OKAN\textsubscript{2s}, Q for OKAN\textsubscript{dur}, and maximal SPEV of OKAN II (\( r = 0.51 \), \( r = 0.61 \), and \( r = 0.80 \), respectively). From values of Q for OKAN\textsubscript{dur}, it was possible to predict the presence or absence of a positional nystagmus correctly in 13/14 patients at the 12-month check-up. No significant correlation was found between the presence of a positional nystagmus and the magnitude of the caloric ratio.

Discussion

An asymmetry in horizontal optokinetic afterresponse was demonstrated in patients with vestibular neuritis. Asymmetric peripheral vestibular function was reflected, not only in the OKAN I, but also in asymmetry of OKAN II. The asymmetry of OKAN II was, however, merely a representation of the peripheral vestibular function, as reflected in the caloric side-difference, but also during follow-up showed changes corresponding to the process of vestibular compensation.

A sudden onset of severe vertigo with spontaneous nystagmus and unilaterally reduced caloric response, in the absence of cochlear symptoms, suggests a diagnosis of vestibular neuritis. Neurological signs indicating lesion to the central nervous system are found in one-third of such cases (22–24). Therefore, we excluded from the study all patients with any sign or history of metabolic, cardiac or vascular disease, or with ocular-motor abnormalities consistent with the possibility of a central nervous lesion (22).

Different instructions to a subject exposed to an optokinetic stimulation may effect the
generation of OKN and OKAN. An instruction to “stare” at the optokinetic stimulus may favor the subcortical optokinetic mechanism (5), as retinal image slip is considered to constitute the stimulus for this mechanism (4,5,12,25), while the cortical optokinetic mechanism has been suggested to be identical with the pursuit eye movement mechanism (4,5). Since OKAN I is associated with the subcortical mechanism (5,11,12,25) one would assume a stimulus favoring this mechanism to be appropriate when investigating OKAN. Nevertheless, if subjects are instructed to “look” at the optokinetic stimulus, most subjects will still have an adequate retinal slip to evoke OKAN (26,27,30). However, when a subject is instructed to “look,” the retinal slip will depend on the subjects’ capacity for pursuit eye movements. Subjects with a high capacity may reduce the retinal slip more prominently than others. To minimize such variations, the subjects in the present study were instructed to focus on the stimulus but not to try to follow the moving stripes during the test.

Few studies have been made of OKAN in peripheral vestibular disorders in man, perhaps due to the problem of quantifying OKAN. The decay of SPEV in OKAN may be due to the output from three separate systems in each of which eye-velocity information is stored. In addition to the central velocity storage integrator, discharges from the smooth pursuit system (27) and the mechanism responsible for OKAN II (16,17) may be involved. Identification of individual variables of these systems leads to practical difficulties, however. In this study were used two readily accessible variables of OKAN (OKAN₂ₙ and OKANₐₙ) that have been demonstrated to reflect asymmetric vestibular activity in man (28). However, evaluation of cumulative slow phase eye displacement or area under the decay curve may add further information.

Normals in this study manifested considerable asymmetry of OKAN I in their response to drum rotation toward right and left. Such asymmetry may be consistent with the suggestion of two separate velocity storage integrators, one for either side of the brain stem (12,13). However, changes in alertness during the test might also affect the symmetry of OKAN (29). Moreover, large intrasubject variability in OKAN I has been reported in man (30).

Asymmetry in OKAN I did not increase with age among the 50 normals in this study. However, the strength of OKAN I is known to decrease with increasing age (30,31), a relationship also noted in this study, where OKAN I was more prominent among females under the age of 40 than in any other subgroup, male or female.

Among the patients with vestibular neuritis, OKAN I beating toward the lesioned ear was weaker than OKAN I beating toward the healthy ear, a finding in accord with those of other studies on unilateral peripheral vestibular disorders in man (32-36). The asymmetry in OKAN I has been interpreted as being due to the loss of spontaneous activity of secondary vestibular neurons on the lesioned side (25). In animal experiments, recovery of tonic activity in the ipsilateral vestibular nucleus has been demonstrated to occur with time after a unilateral peripheral lesion (37). Such a recovery after a lesion may cause a decrease in the relative side-difference between the tonic activity in two vestibular nuclei. As velocity information from the subcortical optokinetic mechanism and the activity of the vestibular nucleus are considered to have an aggregate net effect (9,10), a decrease in asymmetry of OKAN I with time after a unilateral peripheral lesion may be expected. Indeed, in the monkey after unilateral labyrinthectomy, the initial asymmetric OKAN I was found to have become nearly symmetrical at long-term follow-up (38,39). However, additional mechanisms may also be involved in resolving the side-differences in OKAN after a vestibular lesion.

During the one-year follow-up of the patients in the present study, asymmetry of OKAN declined, not only among those who recovered normal caloric response, but also in those with unchanging asymmetry of caloric
Among patients with vestibular neuritis, asymmetries in OKAN II were common, a finding in accord with the suggestion that both OKAN I and OKAN II are dependent on an intact peripheral vestibular apparatus (41). OKAN II was observed only after drum rotation generating OKAN I beating toward the lesioned ear. The OKAN I toward the lesioned ear was of shorter duration when it preceded an OKAN II than it was in patients who manifested no OKAN II. This finding is in agreement with the suggestion that the “velocity storage integrator” and the storage element responsible for OKAN II are interconnected in a negative feedback loop (16,17).

Taken together, the asymmetry of OKAN II in patients with vestibular neuritis and the absence of asymmetry in 50 normal subjects indicate that clinically valuable information can be obtained by investigating OKAN II in cases of suspected peripheral vestibular disorders. The magnitude of asymmetry in OKAN I among normals in this study suggests testing OKAN I with the variables used here to be of limited clinical value. In any individual, however, asymmetry may be subject to fluctuation, and the importance of repeated measurements has been emphasized (30).

The finding of asymmetry in optokinetic afterresponse does not indicate whether there is a lesion to the peripheral or central vestibular pathways (42), but may be considered to reflect asymmetry in the performance of vestibular reflexes. Consequently, testing OKAN should not be the sole test used when investigating a suspected vestibular lesion. However, as a complementary quantitative test of vestibular asymmetry, it may well prove useful, particularly at follow-up after a lesion or during the course of Meniere’s disease.

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