ASYMMETRIC OPTOKINETIC AFTERRESPONSE IN PATIENTS WITH SMALL ACOUSTIC NEURINOMAS

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Abstract—Directional asymmetry of primary and secondary optokinetic afternystagmus (OKAN I and OKAN II, respectively) was studied in 20 patients with small acoustic neurinomas (≤20 mm), and results were compared to those for 24 normal controls. The optokinetic afterresponse was induced by 60 s of horizontal whole-field optokinetic stimulation in both directions. Among patients, the optokinetic afterresponse was asymmetric, OKAN I and OKAN II beating toward the lesioned ear being significantly weaker than the OKAN I and OKAN II beating toward the healthy ear. Hence, in these patients with gradual deterioration of vestibular function, the vestibular side-difference was reflected both in OKAN I and OKAN II. Although asymmetry in OKAN I was frequently observed among controls, it was significantly more pronounced among the patients. Moreover, patients could be distinguished by the occurrence of OKAN II, as it did not occur at all among controls exposed to the same stimulation.

Keywords—optokinetic afternystagmus; acoustic neurinoma, primary, secondary.

Introduction

Optokinetic nystagmus (OKN) is triggered by image slip on the retina. In man, OKN is evoked by way of two mechanisms, cortically and subcortically mediated (1,2). Although cortical response to an optokinetic stimulation generally conceals the response of the subcortical optokinetic mechanism, the latter is nonetheless accessible to study, as it is reflected in the primary optokinetic afternystagmus (OKAN I) (3). During optokinetic stimulation, activity related to slow phase eye-velocity in the subcortical pathways is stored by the “central velocity storage integrator” (3-5). When the optokinetic stimulus is terminated, the integrator discharges, generating OKAN I with fast phases beating in the same direction as the previous OKN. OKAN I is sometimes followed by a secondary optokinetic afternystagmus (OKAN II) moving in the opposite direction (6,7), a reversal phase that has been suggested to be caused by central-nervous adaptation to the optokinetic stimulus (8,9).

In man, asymmetric peripheral vestibular function has been reported to cause an asymmetry in OKAN I, with stronger responses after exposure to drum rotation generating nystagmus beating toward the healthy ear than after drum rotation causing nystagmus toward the lesioned ear (10-15). In these studies, most of the patients investigated had sudden unilateral vestibular lesions. Patients with gradual loss of vestibular function may, however, differ in OKAN from those with sudden lesions, as the symmetry of OKAN may be modified by the process responsible for vestibular compensation (15).

The presence of a cerebellopontine angle tumor enables the effect on OKAN of a gradual vestibular loss to be studied. An acoustic neurinoma, which generally arises from the
vestibular portion of the eighth cranial nerve within the internal auditory canal (16), will by virtue of its growth compress the nerves and vessels within the canal. As the tumor is slow to grow, a gradual destruction over years of the vestibular nerve-fibers may be expected. Further growth of the tumor may exert pressure on structures in the posterior cranial fossa, causing functional deficits within the central nervous system, in addition to the peripheral vestibular deficits. Disturbed pursuit eye movements and gaze nystagmus have been suggested to be early signs of brain stem and/or cerebellum involvement in patients with cerebellopontine angle tumors (17-19).

The aim of the present study was to investigate whether a gradual unilateral deterioration of peripheral vestibular function, as may be expected in patients with small acoustic neurinomas without signs of posterior fossa involvement, is reflected in asymmetry of OKAN.

Materials and Methods

Subjects

Those studied were 20 patients (10 females and 10 males), ranging in age from 35 to 65 years (mean age 50.6 years, SD 7.4) with small acoustic neurinomas. Diagnosis was based on a finding of a cerebellopontine angle or intracanalicular mass in neuroradiographic evaluation (MRI or CAT scan with air insufflation). The size of the tumor was judged from the largest diameter calculated from the computerized tomography. Patients with a tumor size exceeding 20 mm were excluded from the study, as were patients with disturbed pursuit eye movements (20) or gaze nystagmus (21), patients with a spontaneous nystagmus in darkness, and patients over the age of 65. The interval between the computerized tomography and the examination did not exceed 3 months in any case; and in all cases diagnosis was histopathologically verified after surgical removal of the tumor.

The controls comprised 24 healthy subjects (11 females, 13 males) ranging in age from 40 to 65 years (mean age 52.7 years, SD 7.7), none of whom had any history of cochlear, vestibular, or central nervous disorders.

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 wide. 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 at, but not try to follow the moving stripes of the surrounding drum. Eye movements were recorded with DC electro-oculography (EOG) and transcribed simultaneously by an 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 s, recordings being continued until 60 s after the light was extinguished.

The patients but not controls, were further investigated. Spontaneous and positional nystagmus in darkness were recorded—with the patient in the upright position and after he or she was gently placed in the supine position (ie, facing upward). The supine position was maintained for at least 30 s. Voluntary eye movements, gaze nystagmus, and caloric response were also evaluated using EOG (21,22). In addition, audiological tests and a full clinical otoneurological work-up was done in all cases.

Analysis of Recordings

Records were digitized by manually selecting the starting and end-point of slow phases of nystagmus on a graphics tablet (Hipad Plus, Houston Instrument, Austin, Texas), and fed into an IBM-compatible AT computer for analysis. 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 1 was evaluated as the SPEV 2 s after turning off the light (OKAN 2s) and as the
OKAN decay time ('duration') for the OKAN I response \( \text{OKAN}_{\text{dur}} \) (23). OKAN I was further evaluated as the cumulative eye displacement (24) during the period from the 3rd to the 60th second after 'light-off.' This variable \( \text{OKAN}_{\text{cum}} \) was assessed as the aggregate of all slow phase amplitudes, once any slow phases with velocities not exceeding \( 2^\circ/s \) had been excluded. The strength of OKAN I was represented by the mean response to left and right drum rotation. Nystagmus (SPEV > \( 2^\circ/s \)) opposite in direction to, and following an OKAN I, was considered to be significant OKAN II, and was characterized by its maximal SPEV. Any OKAN was described by the direction in which the fast phases of OKAN I and OKAN II beat, and not by the stimulus direction that caused the response. Thus, a drum rotation toward right caused an OKAN I toward the left ear followed by an OKAN II toward the right ear, and vice versa.

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 \( \text{HE} \) and the OKAN I beating toward the lesioned ear \( \text{LE} \), ie, \( Q = (\text{OKAN}_{\text{HE}} - \text{OKAN}_{\text{LE}})/(\text{OKAN}_{\text{HE}} + \text{OKAN}_{\text{LE}}) \). In controls, \( Q \) was calculated from differences between OKAN I beating toward the right \( R \) and left \( L \), ie, \( Q = (\text{OKAN}_{R} - \text{OKAN}_{L})/(\text{OKAN}_{R} + \text{OKAN}_{L}) \). Therefore, absolute \( Q \)-values were used when comparing directional asymmetry among patients with that among controls. Moreover, if OKAN I was absent in both directions, the \( Q \)-value was said to be zero.

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

Statistics

In analyzing data for the patient-group, the Wilcoxon signed rank test was used to evaluate the differences between OKAN I beating toward the healthy ear and that toward the lesioned ear ('paired sample'), as well as between \( Q \)-values and zero ('one-sample'). The Mann–Whitney test was used in comparing strength of OKAN I between the patient and control groups, and to evaluate the differences in absolute \( Q \)-values between patients and controls, as well as between those patients with versus those without an OKAN II beating toward the healthy ear. In addition, linear regression analysis and correlation analysis was used to test for correlation between tumor size and \( Q \)-values, tumor size and caloric ratio, as well as between caloric ratio and \( Q \)-values. A difference or regression of \( P < 0.05 \) was considered significant.

Results

The overall means for strength and asymmetry of OKAN I among patients and controls are given in Table 1, and the distribution of individual \( Q \)-values is shown in Figure 1. The variable \( \text{OKAN}_{\text{cum}} \) was found to elicit larger differences between the patient and control groups than the variables \( \text{OKAN}_{\text{dur}} \) and \( \text{OKAN}_{\text{cum}} \).

Asymmetry in OKAN I

Among patients, \( \text{OKAN}_{\text{cum}} \) beating toward the healthy ear was significantly greater than \( \text{OKAN}_{\text{cum}} \) beating toward the lesioned ear \( (P < 0.05) \) (Table 2). The asymmetry in the relative response to drum rotation in the two directions was reflected in \( Q \) for \( \text{OKAN}_{\text{cum}} \), which among patients was significantly different from zero \( (P < 0.01) \). No significant correlation was found, however, between \( Q \)-values and caloric ratio, nor between \( Q \)-values and tumor size. Moreover, in 5/20 patients there was a negative \( Q \) (Table 3), the \( \text{OKAN}_{\text{cum}} \) beating toward the lesioned ear being stronger than the \( \text{OKAN}_{\text{cum}} \) beating toward the healthy ear.

Asymmetry in OKAN II

No OKAN II was observed in any of the 24 controls. One patient manifested reversal
Table 1. Means and Standard Deviations of OKAN I in 20 Patients with Small Acoustic Neurinomas ("Patients") and in 24 Healthy Controls ("Normals")

<table>
<thead>
<tr>
<th></th>
<th>Strength of OKAN I</th>
<th>Q for OKAN I</th>
<th>Absolute Q for OKAN I</th>
</tr>
</thead>
<tbody>
<tr>
<td>OKAN&lt;sub&gt;cum&lt;/sub&gt; (°)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>patients</td>
<td>45.6 ± 72.3</td>
<td>0.34 ± 0.55</td>
<td>0.58 ± 0.26</td>
</tr>
<tr>
<td>normals</td>
<td>72.3 ± 56.5</td>
<td>0.05 ± 0.49</td>
<td>0.38 ± 0.29</td>
</tr>
<tr>
<td>P*</td>
<td>&lt; 0.05</td>
<td>not applicable</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>OKAN&lt;sub&gt;dur&lt;/sub&gt; (s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>patients</td>
<td>13.5 ± 11.3</td>
<td>0.26 ± 0.40</td>
<td>0.42 ± 0.22</td>
</tr>
<tr>
<td>normals</td>
<td>24.7 ± 16.4</td>
<td>-0.15 ± 0.39</td>
<td>0.29 ± 0.30</td>
</tr>
<tr>
<td>P*</td>
<td>&lt; 0.05</td>
<td>not applicable</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>OKAN&lt;sub&gt;2s&lt;/sub&gt; (°/s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>patients</td>
<td>8.7 ± 5.1</td>
<td>0.12 ± 0.39</td>
<td>0.31 ± 0.26</td>
</tr>
<tr>
<td>normals</td>
<td>8.2 ± 4.7</td>
<td>-0.04 ± 0.22</td>
<td>0.17 ± 0.15</td>
</tr>
<tr>
<td>P*</td>
<td>NS</td>
<td>not applicable</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

* Mann-Whitney test (NS = not significant). Strength of OKAN I is calculated from the mean of OKAN I beating toward the right (R) and toward the left (L); ([OKAN<sub>R</sub> + OKAN<sub>L</sub>]/2). Among patients, Q is calculated from the relative side-difference between OKAN I beating toward the healthy and toward the lesioned ear (HE and LE, respectively): ([OKAN<sub>HE</sub> - OKAN<sub>LE</sub>]/OKAN<sub>HE</sub> + OKAN<sub>LE</sub>); and among controls, Q is calculated from the relative side-difference between OKAN I beating toward the right and toward the left: ([OKAN<sub>R</sub> - OKAN<sub>L</sub>]/OKAN<sub>R</sub> + OKAN<sub>L</sub>).

in OKAN, irrespective of the direction of optokinetic stimulus; and in 12 patients no OKAN II was observed irrespective of the direction of drum rotation. OKAN II was asymmetric (occurring with drum rotation in one direction, but not in the other) in 7 patients, of whom 6 manifested OKAN II beating toward the healthy ear, while in the 7th patient the fast phase of OKAN II beat toward the tumor side (Table 3). Maximal SPEV of OKAN II ranged from 3 to 6°/s. The cumulative eye displacement (OKAN<sub>cum</sub>) preceding a unilateral OKAN II beating toward the healthy ear was depressed compared with corresponding OKAN I among those who manifested no OKAN II toward the healthy ear (P < 0.05). Moreover, as reflected in their greater values for Q, the patients with unilateral OKAN II beating toward the healthy ear manifested more pronounced asymmetry in OKAN I than did the other patients (Figure 2).

Caloric Ratio

The overall mean caloric ratio for the patients was 0.65 (SD 0.32). In 2 patients, response to caloric stimulation revealed no significant side-difference (CR ≤ 0.20), and complete canal paresis was found in 4 patients (Table 3). No significant correlation was found between caloric ratio and tumor size.

Positional Nystagmus

According to the inclusion criteria, none of the patients had spontaneous nystagmus in darkness, nevertheless 3 patients manifested positional nystagmus beating toward the healthy ear in the supine position (Table 3). No cases of nystagmus beating toward the lesioned ear in the supine position were observed.
Figure 1. Histogram of distribution of absolute Q values among 24 healthy controls ("normals") and 20 patients with small acoustic neurinomas ("patients"). Numbers of subjects within five different ranges of Q for variables OKAN$_{2s}$, OKAN$_{dur}$, and OKAN$_{cum}$ are given. Q is calculated from the difference in response to the two directions of drum rotation.
Table 3. The Relationship between Tumor Size and Different Indicators of Asymmetric Vestibular Function in 20 Patients with Small Acoustic Neurinomas

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Tumor size (mm)</th>
<th>Caloric ratio</th>
<th>Positional nystagmus*</th>
<th>Q for OKANcum</th>
<th>OKAN II*</th>
</tr>
</thead>
<tbody>
<tr>
<td>46/M</td>
<td>5</td>
<td>0.32</td>
<td></td>
<td>0.57</td>
<td>LE</td>
</tr>
<tr>
<td>58/F</td>
<td>10</td>
<td>0.38</td>
<td></td>
<td>-0.10</td>
<td>HE</td>
</tr>
<tr>
<td>45/M</td>
<td>10</td>
<td>0.92</td>
<td></td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td>49/F</td>
<td>11</td>
<td>0.48</td>
<td>HE</td>
<td>0.86</td>
<td>HE</td>
</tr>
<tr>
<td>46/F</td>
<td>15</td>
<td>0.13</td>
<td></td>
<td>-0.64</td>
<td></td>
</tr>
<tr>
<td>53/M</td>
<td>15</td>
<td>0.79</td>
<td></td>
<td>0.26</td>
<td></td>
</tr>
<tr>
<td>59/F</td>
<td>15</td>
<td>1.00</td>
<td></td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>43/F</td>
<td>12</td>
<td>0.33</td>
<td></td>
<td>0.79</td>
<td></td>
</tr>
<tr>
<td>54/M</td>
<td>16</td>
<td>0.49</td>
<td></td>
<td>-0.63</td>
<td></td>
</tr>
<tr>
<td>45/M</td>
<td>16</td>
<td>1.00</td>
<td>HE</td>
<td>0.90</td>
<td>HE</td>
</tr>
<tr>
<td>43/F</td>
<td>18</td>
<td>0.39</td>
<td>HE</td>
<td>0.54</td>
<td></td>
</tr>
<tr>
<td>59/M</td>
<td>20</td>
<td>0.06</td>
<td></td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>49/M</td>
<td>20</td>
<td>0.43</td>
<td></td>
<td>-0.45</td>
<td></td>
</tr>
<tr>
<td>35/F</td>
<td>20</td>
<td>0.79</td>
<td></td>
<td>-0.62</td>
<td></td>
</tr>
<tr>
<td>49/F</td>
<td>20</td>
<td>0.86</td>
<td></td>
<td>1.00</td>
<td>HE</td>
</tr>
<tr>
<td>41/M</td>
<td>20</td>
<td>0.88</td>
<td></td>
<td>0.20</td>
<td>HE, LE</td>
</tr>
<tr>
<td>56/F</td>
<td>20</td>
<td>0.88</td>
<td></td>
<td>0.46</td>
<td></td>
</tr>
<tr>
<td>58/M</td>
<td>20</td>
<td>0.95</td>
<td></td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>65/M</td>
<td>20</td>
<td>1.00</td>
<td>HE</td>
<td>0.61</td>
<td>HE</td>
</tr>
<tr>
<td>54/F</td>
<td>20</td>
<td>1.00</td>
<td></td>
<td>0.37</td>
<td></td>
</tr>
</tbody>
</table>

*HE = beating toward the healthy ear; LE = beating toward the lesioned ear.

Discussion

Asymmetry in OKAN was demonstrated in patients with a gradual deterioration of vestibular function due to small acoustic neurinomas. In response to optokinetic stimulation causing OKN beating toward the healthy ear, the OKAN I was stronger and the OKAN II was weaker, as compared with optokinetic stimulation in the opposite direction. Hence, the asymmetric peripheral vestibular function was reflected by a predominance of OKAN I and OKAN II beating away from the tumor side. Moreover, the strength of OKAN I, calculated from the individual mean response to optokinetic stimulation in both directions, was decreased in patients compared with controls.

A cerebellopontine angle tumor compressing the brain stem may have a direct effect on the functional integrity of the "central velocity storage mechanism" and the generation of OKAN, as well as on the eye motor control mechanism. Disturbed pursuit eye movements and gaze nystagmus have been suggested to be early signs of an acoustic neurinoma affecting structures in the posterior fossa, the frequency increasing with increasing tumor size (17), especially over 20 mm in diameter (18,19). As the study was designed to investigate OKAN in patients with peripheral vestibular lesions, all patients with a tumor size exceeding 20 mm, as well as any patients with oculomotor dysfunction, were excluded. However, although there were no clinically apparent oculomotor abnormalities, it is likely that tumors protruding by more than 15 mm into the cerebellopontine angle contact the pons and the cerebellar hemisphere (17). Therefore, we also tested for differences between the patients with a tumor size not exceeding 15 mm (n = 7) and those with larger tumors (n = 13), but found no significant differences with respect to caloric ratio or Q-values (Mann-Whitney test).

A further aim was to study cases of gradual loss of vestibular function, as may be expected in patients with acoustic neurinomas. However, a few of these patients may present with symptoms indicating sudden changes in the functional status of the inner ear. This may be due to a more rapid rate of tumor
growth, owing to degenerative mechanisms, such as edema, cyst formation, or hemorrhages into tumor tissue (25), or to thrombosis of vessels supplying the inner ear (26). Thus, the assumption of a gradual increase of tumor size causing gradual destruction of vestibular nerve fibers may not be justified in each patient with an acoustic neurinoma. However, none of the patients in the present study had spontaneous nystagmus in darkness. This may indicate that they had not suffered from a sudden change in the vestibular function to which they had not yet compensated.

Since the first reports on OKAN, different variables have been used for its characterization. The decay of SPEV in OKAN I 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 (8,9) may be involved. However, identification of specific variables of these systems is beset with practical difficulties. Two readily accessible variables of OKAN, initial velocity and decay duration (OKAN_{2s} and OKAN_{dur}, respectively), have been demonstrated to reflect a galvanically induced asymmetric vestibular activity in man (23); however, these earlier data have since been re-assessed, and we found the galvanically induced changes in cumulative slow phase amplitudes of OKAN (OKAN_{cum}) to be significantly more pronounced than corresponding changes in OKAN_{2s} or OKAN_{dur}.

In the present study, patients with acoustic neurinomas manifested weaker OKAN I beating toward the lesioned than toward the healthy ear. This finding is in accord with those obtained in patients with sudden peripheral vestibular lesions (10–15). Since velocity information from the subcortical optokinetic mechanism and the activity of the vestibular nucleus are considered to have an aggregate net effect (28,29), the asymmetry in OKAN I may be due to the loss of spontaneous activity of secondary vestibular neurons on the lesioned side (30). Moreover, the conception of an abatement in OKAN I performance causing the asymmetry in OKAN I among patients with vestibular lesions is sup-

Figure 2. The difference in quotient (Q) between patients with a unilateral OKAN II beating toward the healthy ear ("OKAN II HE") versus patients without a unilateral OKAN II beating toward the healthy ear ("No OKAN II HE"). Means, SEM, and levels of significance (Mann-Whitney test) are given.
ported by the present observation of a reduction in strength of OKAN I among patients compared with controls. Although, the asymmetric vestibular function in these patients with acoustic neuromas was reflected in an asymmetry in OKAN I, the clinical value of OKAN I as an indicator of a vestibular side-difference is not clearcut. Stronger OKAN I beating toward the healthy ear was not a consistent finding, as 4 patients manifested substantial asymmetry with a predominance of OKAN I beating toward the lesioned ear. Moreover, asymmetry was also substantial in controls, which may be a reflection of a difference in gain in two separate “velocity storage integrators,” one for either side of the brain stem, as suggested (3,4); however it may also be due to individual fluctuations in attention and alertness during the test, and the importance of repeated measurements has been emphasized (31).

No significant correlations were seen between Q-values and tumor size in the present study. A correlation may have been masked, however, by intersubject variability in OKAN I, or the parameter Q may simply not be sensitive enough for assessment of tumor size in patients with acoustic neuromas. Furthermore, no significant correlation was found between caloric ratio and tumor size. The absence of such a significant correlation may be due to correlation, if existing, being weak and the study comprising insufficient number of patients, as a linear relationship has been demonstrated between tumor size and caloric side-difference in a group of 41 acoustic neurina patients with normal oculomotor function (19).

The mechanism responsible for OKAN II has been suggested to be a ‘central adaptation operator,’ responsible not only for OKAN II but also for the reversal phase of postrotatory nystagmus (PRN II) (9). 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 (32,33).

The frequency of asymmetry in OKAN II among patients with small acoustic neuromas is consistent with the suggestion that OKAN II is as dependent as OKAN I on an intact peripheral vestibular apparatus (34). OKAN II was mainly observed after an OKAN I beating toward the lesioned ear. OKAN I was decreased when it preceded an OKAN II, compared with patients who manifested no OKAN II. This finding is consistent both with the suggestion that the duration of OKAN I is partly determined by the development of OKAN II (35), and with the proposal that the “velocity storage integrator” and the storage element responsible for OKAN II are interconnected in a negative feedback loop (8,9). Moreover, the present observation, that patients with OKAN II show more pronounced asymmetries in OKAN I (Q-values) than patients without OKAN II, is in agreement with observations among patients with vestibular neuritis (15).

The finding of an asymmetry in OKAN II in a patient with a suspected peripheral vestibular lesion may be of clinical significance. Although, OKAN II is generally seen in normal subjects after optokinetic stimulation with long duration (7), the short stimulation time in this study (60 s) did not generate OKAN II in any of the controls. Nevertheless, asymmetric OKAN II was frequent among the patients with acoustic neuromas. Hence, a patient who develops an asymmetric OKAN II during an observation period should be further investigated with respect to an acoustic neuroma causing gradual deterioration of vestibular function. Moreover, during follow-up of patients with vestibular neuritis, OKAN II was observed only when beating toward the healthy ear (15), and the same pattern was now found in 6 of 7 patients with acoustic neuromas and unilateral OKAN II.

To sum up, although vestibular compensation was able to suppress any spontaneous nystagmus among these patients with small acoustic neuromas, asymmetry in vestibular function was reflected in the OKAN.

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