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Caloric and Search-Coil Head-Impulse Testing in Patients after Vestibular Neuritis

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ABSTRACT

The objective of this study was to compare results of quantitative head-impulse testing using search coils with eye-movement responses to caloric irrigation in patients with unilateral vestibular hypofunction after vestibular neuritis. The study population consisted of an acute group (<3 days; $N = 10$; 5 male, 5 female; 26–89 years old) and a chronic group (>2 months; $N = 14$; 8 male, 6 female; 26–78 years old) of patients with unilateral vestibular hypofunction after vestibular neuritis. The testing battery included: (1) simultaneous measurement of eye and head rotations with search coils in a magnetic coil frame during passive Halmagyi–Curthoys head-impulse testing and (2) electronystagmography during bilateral monaural 44°C-warm and 30°C-cold caloric irrigation. The main outcome measures were (1) the gain of the horizontal vestibulo-ocular reflex during search-coil head-impulse testing and (2) the amount of canal paresis during caloric irrigation. All acute and chronic patients had a unilateral gain reduction during search-coil head-impulse testing. A pathological canal paresis factor was present in 100% of the acute patients but in only 64% of the chronic patients. The clinically suspected unilateral vestibular hypofunction resulting from vestibular neuritis was validated in all acute patients by both search-coil head-impulse and caloric testing. Hence, either of these tests is sufficient for diagnosis in the acute phase of vestibular neuritis. Chronic patients, however, were reliably identified only by search-coil head-impulse testing, which suggests that the low-frequency function of the labyrinths often becomes symmetrical, leading to a normal canal paresis factor.

Keywords: nystagmus, labyrinths, canal paresis, neurootology

INTRODUCTION

Caloric ear irrigation produces a unilateral low-frequency stimulation of the peripheral vestibular organ. According to Bárány's theory, the caloric response primarily is the result of endolymphatic convection currents resulting from local temperature and, hence, fluid-density changes in the presence of gravity (Bárány 1906). The fact that caloric nystagmus can also be elicited in a weightless environment indicates that direct thermal effects upon hair cells and nerve fibers also contribute to the caloric response (Scherer et al. 1986). There is evidence that all three semicircular canals play a part in the generation of caloric nystagmus, but the major input to the caloric response comes from the lateral semicircular canal (Bohmer et al. 1992, 1996; Fetter et al. 1998).

Caloric irrigation is the most widely used tool in routine vestibular testing to identify the presence and side of a peripheral vestibular hypofunction. The caloric response, i.e, the slow-phase velocity of nystagmus, is quite variable as it depends on the aural irrigation and eye-movement recording techniques (Becker 1978; Baloh et al. 1977), the morphology of the ear (Feldmann et al. 1991), the pneumatization of the mastoid cells (Zangemeister and Bock 1979), and the patient's alertness (Davis and Mann 1987).

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The major limitation of caloric irrigation is that it provides only a nonphysiological stimulation of the peripheral vestibular organ. In contrast, the assessment of the high-frequency vestibulo-ocular reflex (VOR) by passive head impulses allows a more physiological testing of the vestibular system (Halmagyi and Curthoys 1988). Moreover, using three-dimensional search coils and applying head impulses in different directions, the function of individual semicircular canals can be quantified precisely (Aw et al. 1996).

Harvey et al. (1997) showed that the head-impulse test, when used as a bedside screening tool for vestibular hypofunction, has a sensitivity of only 35% in reference to caloric testing; on the other hand, the specificity was 95%. Similar results were reported by Beynon and coworkers (1998). Thus, these data suggest that the clinical head-impulse test alone cannot be used to reliably identify patients with peripheral vestibular hypofunction. We asked whether the quantitative analysis of eye and head movements during head impulses would improve the sensitivity of this test in reference to caloric testing. For that reason, we mounted search coils on the right eye and the forehead to measure the vestibulo-ocular reflex (VOR) during head thrust. In patients with unilateral acute or chronic vestibular hypofunction after vestibular neuritis, we compared the gain of the horizontal VOR with caloric responses elicited by bilateral monaural 30°C-cold and 44°C-warm water irrigations (canal paresis factor, directional preponderance of nystagmus). The main finding was that all patients—chronic and acute—showed a unilateral gain reduction in search-coil head-impulse testing. A pathological canal paresis factor with relative ipsilateral caloric weakness was present in 100% of the acute patients but only in 64% of the chronic patients. Hence, it seems that the sensitivity of the quantitative head-impulse test with search coils is equal (acute patients) or even superior (chronic patients) to caloric testing.

MATERIAL AND METHODS

Subjects

Twenty-four consecutive patients with clinically suspected unilateral, peripheral, vestibular hypofunction resulting from vestibular neuritis participated in this study. Informed consent was obtained after full explanation of the experimental procedure.

The clinical examination was performed by either of two experienced neuro-otologists (A.B., D.S.), and the clinical diagnosis was based on the patient's history and bedside testing. The patients were partitioned according to the time elapsed since the beginning of vestibular symptoms: If the symptoms were present less than three days, patients were assigned to the acute group; if more than two months had passed since the initial symptoms, patients were assigned to the chronic group. While in all acute patients a spontaneous nystagmus was demonstrated in our laboratory using electro-oculography, the records of all chronic patients contained at least the description of a spontaneous nystagmus in the acute phase. Clinically, the chronic patients showed at least one of the following two signs: a head-shaking nystagmus or correcting saccades after head impulses to one side. This clinical head-impulse test was pathological in all acute patients and 71% of the chronic patients.

The acute group consisted of 10 patients (A1–A10; 5 male, 5 female) with a mean age of 50.6 years (range: 26–89); the chronic group consisted of 14 patients (C1–C14; 8 male, 6 female) with a mean age of 53.4 years (range: 26–78). Here we report on (1) search-coil recordings during horizontal head impulses and (2) electronystagmographic recordings during bithermal caloric irrigations. Both tests were done in all patients. For convenience, the data from patients with left-sided vestibular lesions were mirrored.

Quantitative head-impulse testing

Eye and head movements were recorded in a magnetic frame (Remmel-type system, modified by A. Lasker, Baltimore, MD) using scleral search coils (Skalar Instruments, Delft, Netherlands) (Remmel 1984; Robinson 1963; Ferman et al. 1987). One search coil was placed on the right eye around the cornea (after anesthetizing the conjuctiva with oxybuprocaine 0.4%), the other was tightly fixed on the forehead with adhesive tape. Digitized data were sampled with 1000 Hz and stored on the hard disk of a computer. Raw signals were calibrated and processed using interactive programs written in MATLAB™ Version 5.3 (MathWorks Inc, Natick, MA, USA).

During experiments, subjects were seated inside the magnetic frame (side length: 1.4 m). Care was taken to position the center of the interpupillary line in the center of the magnetic frame. While the subject was fixing a light dot 1.24 m straight ahead, horizontal head impulses (amplitude: 15°–25°; duration: 150–200 ms; peak velocity: approx 250°/s; peak acceleration approx 10,000°/s^2) were applied by the investigator standing behind the subject. To exclude skin artifacts onto the head coil, special care was taken that the experimenter's hands did not touch the subject's forehead. In a pilot study, we specifically checked that there were no signal differences between fixing the head coil on the forehead or a bitebar. The starting position of the head was always well inside a range of 5°. Directions of impulses were pseudorandomly intermingled; five head rotations were applied to each

side. Subjects were instructed to keep their gaze on the light dot straight ahead. For the head impulse test, however, it is irrelevant if performed in the light or in the dark because the accelerations are above the working range of the smooth pursuit system.

Caloric testing

Patients were placed in a supine position. To bring the horizontal semicircular canals approximately parallel to the earth-vertical axis, the head was tilted 30° forward. Horizontal eye movements were recorded using direct-current electro-oculography (EOG). In sequence, unilateral 30°C-cold and 44°C-warm water irrigations during 20 s with 20 mL of water were performed on either side ($30°C_{right}$, $30°C_{left}$, $44°C_{left}$, $44°C_{right}$). The eye movement response to the caloric stimulation was recorded for 180 s while the eyes were closed and the patient had to perform mental tasks. Resting periods of 5 minutes or more were observed between recordings. The maximal slow phase velocity of caloric nystagmus was determined during the time interval between 30 and 90 s from the beginning of the irrigation. To quantify the asymmetry of peripheral vestibular function in percent, the following formula of canal paresis (CP) was used (Jongkees 1966):

$$CP = [(W_L + C_L) - (W_R + C_R)]/ [W_L + C_L + W_R + C_R] \cdot 100$$

where W_R, W_L, C_R, and C_L are the maximal slow-phase velocities of nystagmus elicited by 44°C (W) or 30°C (C) irrigations on the right (R) or left (L) side, respectively. The directional preponderance (DP) of the caloric response in percent was determined by (Jongkees 1966)

$$DP = [(C_L + W_R) - (C_R + W_L)]/ [W_L + C_L + W_R + C_R] \cdot 100$$

To determine the significance of the correlation between two sets of data (Pearson's R), we applied the t-test.

RESULTS

Figure 1 shows examples of calibrated search-coil data during horizontal head impulses. The left panels (Fig. 1A, C) depict data from a healthy subject, the right panels (Fig. 1B, D) from a patient (A6) with an acute, right-sided, vestibular hypofunction. The traces on the upper panels (Fig. 1A, B) represent head-in-space ("head") and eye-in-head ("eye") movements during head impulses to the right; on the lower panels (Fig. 1C, D), the corresponding movements elicited by head impulses to the left (normal side) are shown.

The following definition of the vestibulo-ocular gain during head impulses (VOR_{HI}) was applied: At peak head velocity, we determined the position change of both head-in-space (Δh) and eye-in-head (Δe) from the beginning of the impulse (Okinaka et al. 1993). The gain (g) was computed by:

$$g = \Delta e / \Delta h$$

For each set of five head impulses in one direction, the average gain G was determined ($G_R = G$ during head impulses to the right; $G_L = G$ during head impulses to the left). The healthy subject (Fig. 1A, C) showed normal gains to both sides ($G_R = 0.95$; $G_L = 0.81$), while in the patient (Fig. 1B, D) there was a clear gain reduction during head impulses to the right ($G_R = 0.5$) but normal gain values during head impulses to the left ($G_L = 0.85$). Notice in Figure 1B the correcting saccade, by which the patient compensated the decreased gain of the VOR_{HI} to refix the light straight ahead (arrow).

For the acute (<3 days) group of patients, Figure 2 represents the scatterplot of the average vestibulo-ocular gains (G) during horizontal head impulses against the amount of canal paresis (CP). The average gains obtained during rightward (ipsilesional) head impulses (G_R) (open circles) and the average gains obtained during leftward (contralesional) head impulses (G_L) (asterisks) are plotted against the corresponding CP values. Thus, for an individual patient, the two plotted symbols (open circle and asterisk connected by a dashed line) correspond to the same CP value. (Note again that the data from patients with left-sided vestibular lesions were mirrored.) The dotted lines parallel to the ordinate mark the normal gain values of the horizontal head impulse test (average gain = 0.85 ± 2 SD: $0.74 < G_{norm} < 0.96$), and the dotted line parallel to the abscissa mark the limit of normal CP values ($CP_{norm} < 25\%$), as determined from a database of healthy subjects.

Pathological gain reductions of the horizontal vestibulo-ocular reflex (VOR) during head impulses to the affected side (G_R, open circles) were found in all acute patients. Gains during head impulses to the normal side (G_L, asterisks), however, were within normal limits (50%) or slightly reduced (50%). For individual patients, the gains during ipsilesional head impulses (G_R) were always lower than during contralesional impulses (G_L). The caloric response on the pathological side was reduced in all acute patients; CP values ranged between 33% and 100%. The amount of canal paresis did not correlate with either the ipsilesional or the contralesional gain during horizontal head impulses ($p > 0.05$). Yet the indicated side of the vestibular lesion was in agreement between the two testing methods, and the results from both head-impulse and caloric testing allowed us to confirm the

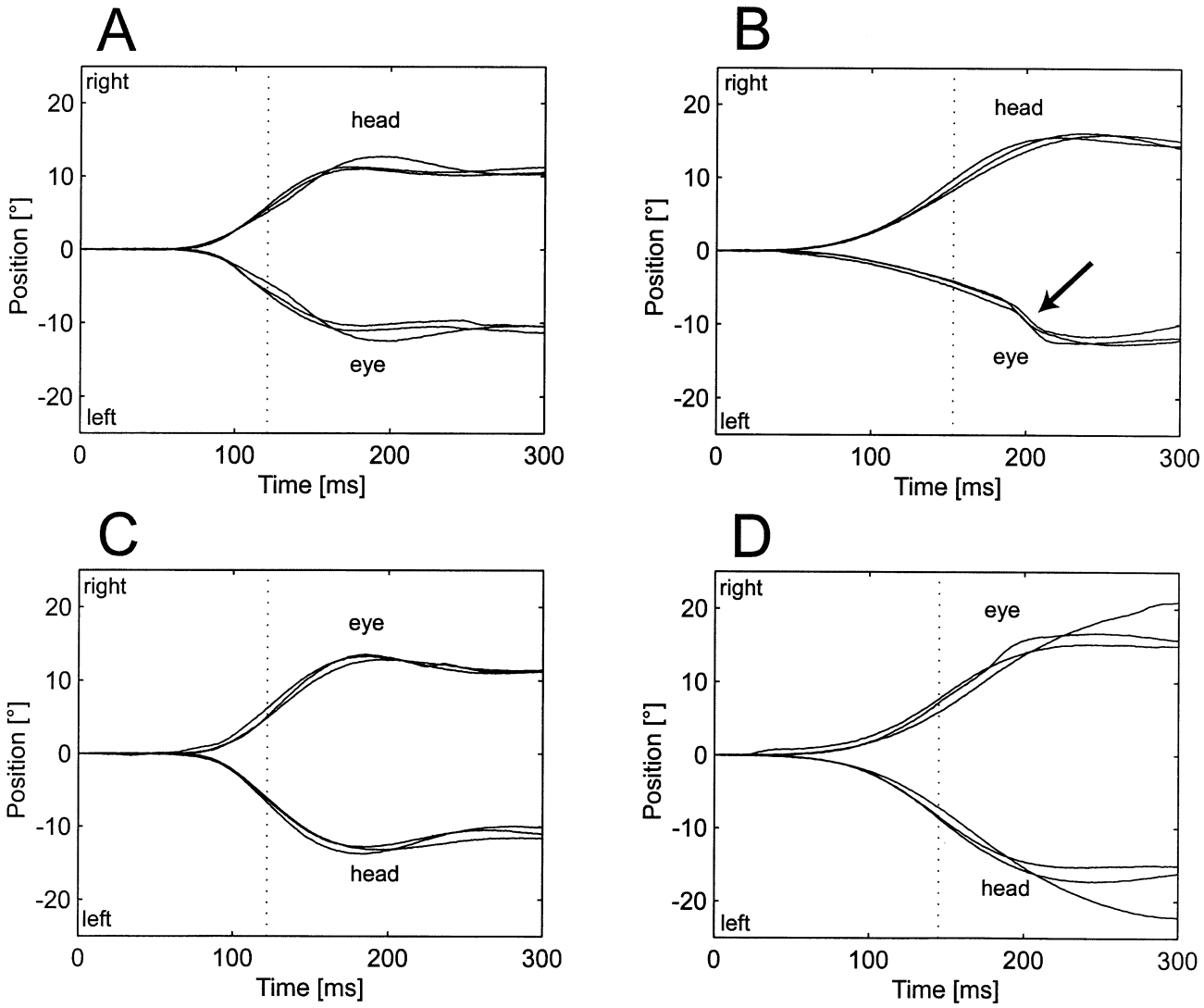

FIG. 1. Examples of horizontal head-impulse testing. Head-in-space and eye-in-head position traces measured with the search-coil technique. **A,C**. Healthy subject. **B,D**. Patient with an acute peripheral vestibular hypofunction on the right side. **A,B**. Head impulses to the right. **C,D**. Head impulses to the left. Arrow: correcting saccade.

clinical diagnosis of a unilateral vestibular hypofunction in all acute patients.

The data of the chronic (>2 months) patients are shown in Figure 3. Again vestibulo-ocular gains during horizontal head impulses toward the lesioned side (G_R, open circles) and the other side (G_L, asterisks) are plotted versus the CP values obtained from caloric testing. The dotted lines parellel to the ordinate or abscissa are placed identically as in Figure 2 and indicate the limits of normal values.

All chronic patients showed clear gain reductions during horizontal head impulses to the affected side (G_R). In only 64% of the patients, however, the CP values were in the pathological range. Similar to the acute group, half of the chronic patients had slightly reduced (50%) or normal (50%) gains during horizontal head impulses to the normal side (G_L).

Contrary to the acute group, a (weak) significant correlation between the vestibulo-ocular gains during head impulses to the lesioned side (G_R) and CP values was present in the chronic patients ($R^2 = 0.3$; $p = 0.047$; offset = 95; slope = −15.6). None of the chronic patients, however, had a pathological caloric response together with a normal horizontal head-impulse test. Maximal slow-phase eye velocities averaged 10.38 deg/s (±6.74 SD) during warm irrigation of the affected side, 20.88 deg/s (±12.89 SD) during warm irrigation of the healthy side, 13.88 deg/s (±15.34 SD) during cold irrigation of the affected side, and 23.13 deg/s (±12.31 SD) during cold irrigation of the healthy side.

Similar to the canal paresis (CP) factor, we computed the coefficient of gain asymmetry (GA) between head-impulse tests to the lesioned and healthy side (GA):

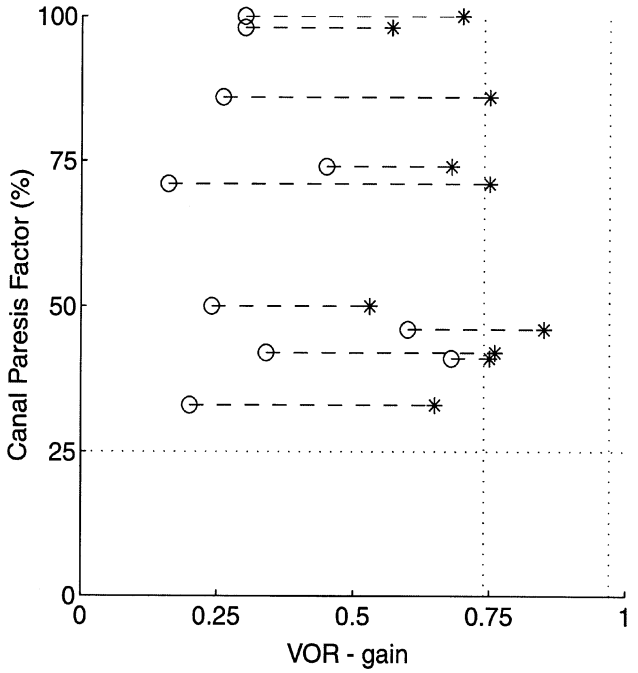

FIG. 2. Patients with acute vestibular hypofunction (data in patients with left-sided vestibular hypofunction are mirrored to the right side): Gains of the horizontal vestibulo-ocular reflex (VOR) during head impulses to the right (G_R, open circles) and the left side (G_L, asterisks) are plotted against the percentage of canal paresis (CP). The two symbols are connected by a dashed line in each patient. Dotted lines parallel to the ordinate: range of horizontal VOR gains in the healthy subjects; dotted line parallel to the abscissa: upper limit of normal CP values.

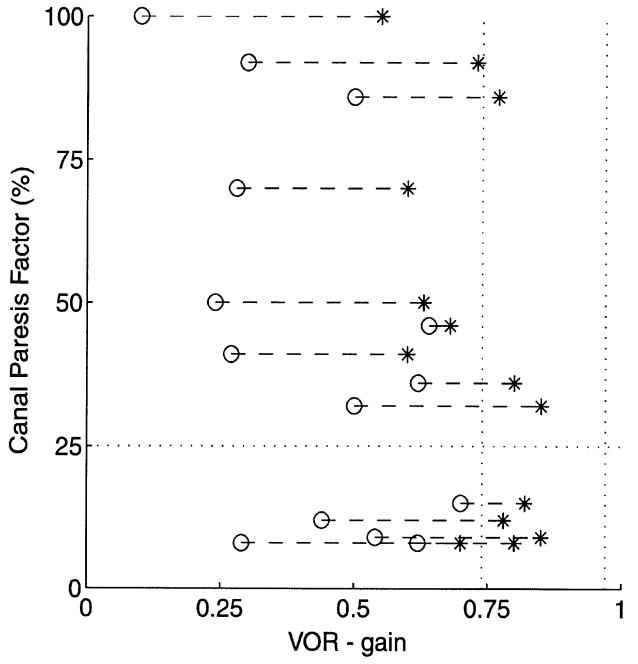

FIG. 3. Patients with chronic peripheral vestibular hypofunction: Symbols, axes, and lines are defined in Fig. 2.

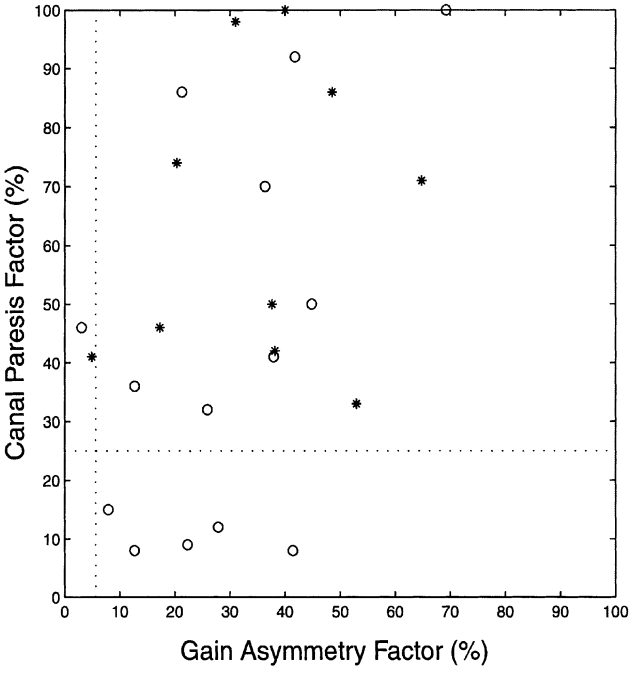

FIG. 4. Patients with acute (asterisks) or chronic (open circles) vestibular hypofunction: The percentage of gain asymmetry (GA) is plotted versus the percentage of canal paresis (CP). Dotted line parallel to the ordinate: upper limit of normal GA values; dotted line parallel to the abscissa: upper limit of normal CP values.

$$GA = [(G_L - G_R)/(G_L + G_R)] \cdot 100$$

In Figure 4 we plotted GA vs. CP-factor in acute (asterisks) and chronic (open circles) patients. The dotted lines indicate the normal values from our database of healthy subjects ($GA_{norm} < 5.6\%$; $CP_{norm} < 25\%$). There was no correlation between the two factors in both the acute and chronic patients. Note that only two patients (one acute, one chronic) were within normal limits of the GA-coefficient, while the CP-factor was normal in five chronic patients (see Fig. 3).

The head-impulse data were also correlated with the directional preponderance (DP) of caloric nystagmus (definition given in Methods section). When we used this formula to describe labyrinthine asymmetry, only 60% of the acute and 64% of the chronic patients showed pathological DP-values (normal <31%), which were always biased to the healthy side. Hence, for the patients enrolled in this study, the sensitivity of DP was inferior to both the gains of horizontal search-coil head-impulse testing and the factors of canal paresis (CP).

DISCUSSION

Patients with clinically suspected acute (<3 days) or chronic (>2 months) unilateral vestibular hypofunction resulting from vestibular neuritis were quantitatively tested. The gains of the vestibulo-ocular reflex

(VOR) during horizontal head impulses as measured with search coils were compared with responses to bithermal caloric irrigation. In all patients, the search-coil head-impulse test revealed clear gain reductions of the horizontal VOR during head rotation to the side assumed to be affected. A slight gain reduction during head impulses toward the normal side was present in about half of the patients, which is in line with previous reports in patients after unilateral vestibular neuritis or vestibular deafferentation (Schmid–Priscoveanu et al. 1999; Aw et al. 1996a). Applying the canal paresis formula of caloric nystagmus, we found pathological asymmetries in all acute patients but in only 64% of the chronic patients. Hence, in the caloric tests, about 40% of the chronic patients showed a symmetrical peripheral vestibular function, which is similar to the percentages reported by others (Imate and Sekitani 1993; Okinaka et al. 1993; Herzog et al. 1997). For the head-impulse test, only two patients showed an asymmetry that was still within normal limits; in both of these patients, however, the gain of the head-impulse test toward the lesioned side was in the pathological range. It seems that the low-frequency function of the peripheral vestibular system often recovers or becomes symmetrical. In contrast, deficits of the high-frequency horizontal VOR to the lesioned side are permanent in patients after unilateral vestibular deafferentation (Aw et al. 1996b) or neuritis (Schmid-Priscoveanu et al. 1999). This might explain the higher sensitivity of the search-coil head-impulse test in reference to caloric testing in the chronic group. The different frequency range of the vestibular system measured by the head-impulse test (high frequencies up to 5 Hz) and the caloric test (low frequencies up to 0.003 Hz) is probably the reason why there is no (acute group) or only a weak (chronic group) correlation between the head impulse and the caloric data. In other words, it seems that in individual patients the peripheral vestibular system can be affected differentially over the frequency range.

Our data seem to contradict the data published by (Harvey et al. 1997) and (Beynon et al. 1998). Both groups found a low sensitivity of the head-impulse test in reference to caloric testing in patients with unilateral vestibular hypofunction. The studies, however, relied on the bedside observation of the head impulse. In this situation, the clinician cannot track the eye movement *during* the head impulse because it lasts only about 150 ms; he/she has to estimate the amplitude of the correcting saccade *after* the head impulse. This measure seems to underestimate the presence of an abnormally low gain of the vestibulo-ocular reflex because, for example, the patient can make multiple correcting saccades or can generate saccades at very short latencies (already started during the head impulse). In addition, since even in healthy subjects the vestibulo-ocular gain during horizontal head impulses is usually below 1.0, a small correcting saccade is frequently observed, despite intact labyrinths on both sides. Quantitative head-impulse testing with search coils, however, does not rely on the amplitude of the correcting saccade but measures the actual performance of the vestibulo-ocular reflex *during* the head impulse. Another advantage of eye and head search-coil measurements during head impulses is the possibility of testing the VOR about different axes, provided one uses dual search coils (Aw et al. 1996a), which allows the examination of individual semicircular canals (Cremer et al. 1998).

If one has to make a choice between the two tests, we prefer the search-coil head-impulse test over the caloric test because of a higher sensitivity in the stage of chronic vestibular hypofunction following vestibular neuritis. Also, the results of search-coil head-impulse testing are less variable, i.e., do not have all the disadvantages that influence the responses to caloric testing, such as anatomical variability of the ear, pneumatization of the mastoid, irrigation, and recording technique. Ideally, both methods, the search-coil head-impulse *and* caloric testing, should be applied for a more complete assessment of frequency-dependent deficits of the peripheral vestibular organ. If required, eye-movement recordings during rotations on a turntable can give additional information about the performance of the VOR during midband frequency stimulation; however, with this method the side of the lesion usually cannot be determined.

In conclusion, the sensitivity of search-coil head-impulse and caloric testing is equal in patients with clinically diagnosed acute peripheral vestibular hypofunction. In chronic patients, however, search-coil head-impulse testing has a higher sensitivity than caloric testing.

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