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Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders

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ABSTRACT

Objectives: The function of the semicircular canal receptors and the pathway of the vestibulo-ocular-reflex (VOR) can be diagnosed with the clinical head impulse test (cHIT). Recently, the video head impulse test (vHIT) has been introduced but so far there is little clinical experience with the vHIT in patients with peripheral vestibular disorders. The aim of the study was to investigate the horizontal VOR (hVOR) by means of vHIT in peripheral vestibular disorders.
Methods: Using the vHIT, we examined the hVOR in a group of 117 patients and a control group of 20 healthy subjects. The group of patients included vestibular neuritis (VN) ($n = 52$), vestibular schwannoma (VS) ($n = 31$), Ménière's disease (MD) ($n = 22$) and bilateral vestibulopathy (BV) ($n = 12$).
Results: Normal hVOR gain was at 0.96 ± 0.08, while abnormal hVOR gain was at 0.44 ± 0.20 (79.1% of all cases). An abnormal vHIT was found in VN (94.2%), VS (61.3%), MD (54.5%) and BV (91.7%). Three conditions of refixation saccades occurred frequently in cases with abnormal hVOR: isolated covert saccades (13.7%), isolated overt saccades (34.3%) and the combination of overt and covert saccades (52.0%).
Conclusions: The vHIT detects abnormal hVOR changes in the combination of gain assessment and refixation saccades. Since isolated covert saccades in hVOR changes can only be seen with vHIT, peripheral vestibular disorders are likely to be diagnosed incorrectly with the cHIT to a certain amount.

1. Introduction

The clinical head impulse test (cHIT) by Halmagyi and Curthoys is a mandatory tool in the diagnosis of dizziness and balance disorders [1]. By means of the cHIT information of the vestibulo-ocular-reflex (VOR) is obtained and disturbances of gaze stabilization can be diagnosed by the identification of compensatory refixation saccades. The cHIT testing in all planes of semicircular canals (SCC) provides a side and receptor specific information of VOR disorders [2,3].

The video head impulse test (vHIT) is a new method to record eye and head velocity. Conducting the vHIT allows to visualize the VOR in its physiological range in a similar way as search-coil measurements [5–7]. VOR gain reduction and refixation saccades can therefore be analyzed quantitatively. Refixation saccades are thought to substitute VOR gain reduction. They are either invisible (catch-up covert) or visible (catch-up overt) since they occur during or after the head impulse [8–12].

With the cHIT catch-up overt saccades can be detected but catch-up covert saccades remain invisible to the simple visual inspection by the examiner. In contrast, the vHIT allows analyzing both types of refixation saccades of the induced VOR.

In peripheral vestibular disorders, as in patients with acute vestibular neuritis (VN), the vHIT of the horizontal VOR (hVOR) is equivalent to search coil techniques in identifying peripheral vestibular deficits, but more suitable in daily clinical practice because bedside use is possible [10]. In the literature there are only few reports on the clinical application of vHIT in several vestibular disorders [8–10]. The aim of this study was to evaluate the vHIT in patients with peripheral vestibular disorders and to analyze the occurrence of refixation saccades.

2. Patients and methods

2.1. Patients

117 patients (65 women and 52 men, with a mean age of 52.8 years, range 24–78) with four types of peripheral vestibulopathies were included: unilateral vestibular neuritis (VN) ($n = 52$), unilateral vestibular schwannoma (VS) ($n = 31$), unilateral Ménière's disease (MD) ($n = 22$) and bilateral vestibulopathy (BV) ($n = 12$).

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Diagnosis of unilateral VN was based on the following criteria: rotatory vertigo, horizontal-rotatory spontaneous nystagmus, gait imbalance and falling (ipsiversiv), vomiting, nausea, a pathological side difference (>25%) at bithermal caloric irrigation (44° and 30°, water, normal <25% asymmetry). The vHIT measurements in patients with VN lasted from 1 to 94 days after the onset of symptoms.

Diagnosis of unilateral VS was confirmed by cranial MRI. The clinical diagnosis unilateral MD was made according to the guidelines of the AAO-HNS [13]. In cases of BV an abnormal bithermal examination of the horizontal SCC occurred on both sides using cold and warm water.

Persons taking CNS depressants, vigilance-influencing drugs or with central vestibular disorders were excluded from the study. Acute and chronic disorders were not differentiated.

This study was conducted in conformity with the declaration of Helsinki and approved by the ethic committee of the University of Mannheim. Written informed consent was obtained from all participants after the experimental procedure was explained.

2.2. Control group

The control group for normative data consisted of 20 healthy volunteers (10 women and 10 men, mean age 51.3 years, range 18–72) who had no history of vestibular disorders, no intake of vigilance-affecting drugs, a normal vHIT for the horizontal SCC and a normal caloric irrigation.

2.3. Video head impulse testing

For vHIT measurements the EyeSeeCam™ System was used [6,7]. The vHIT-system consists of a light-weighting goggle with integrated videooculography (VOG)-camera and intertial sensors, tightly fixed to the head with rubber band. The sampling rate of the VOG-camera and the intertial sensors was up to 256 Hz.

Subjects were instructed to fixate a dot located on the wall 1.2 m straight ahead. A minimum of 10 head impulses in the horizontal plane (head rotation 15–20°, duration 150–200 ms, peak velocity > 150°/s) were performed manually to both sides, with unpredictable timing and direction to record hVOR.

The eye and head velocities (°/s) were captured at 40, 60, and 80 ms after the head impulse was initiated and averaged for both. The velocity gain of the hVOR is represented by the ratio of mean eye velocity (°/s) over mean head velocity (°/s). Refixation saccades were sampled within a time span of 700 ms after the onset of head impulses and with amplitudes up to 400°/s eye velocity.

The vHIT results were defined as normal, if they were within the calculated gain-reference range, $\text{mean}_{\text{normal}} \pm 2$ standard deviations (SD), incorporating 95% of population and if no refixation saccades occurred. The vHIT results were classified as abnormal if two conditions were met: abnormal gain according to the calculated normative data and presence of refixation saccades (revealed by visual inspection, cf. type 3 in Fig. 1). As a robust characteristic to define a refixation saccade, we used peak acceleration. Refixation saccades were classified as covert, if they occurred before the end of the head impulse and classified as overt afterwards. Amplitudes (°/s) and latencies (ms) of the identified refixation saccades and physiologically induced saccades were evaluated at peak eye velocity (°/s).

2.4. Statistical analysis

To analyze the hVOR with vHIT, data were numerically captured for left and right sided gain. The significance of side-specific differences of the gain was determined with the Student's *t*-test.

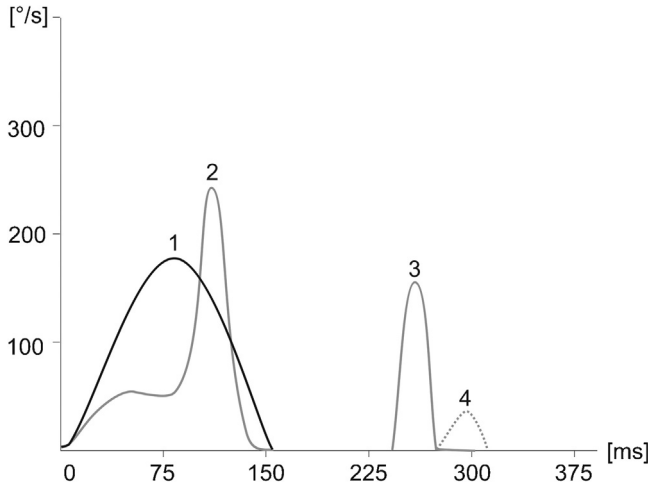

Fig. 1. Schematic occurrence of refixation and physiological saccades. (1) eye velocity, (2) covert saccade, (3) overt saccade, and (4) physiological saccade.

3. Results

For the healthy controls, the average hVOR gain was 0.96 ± 0.08 ($\text{mean}_{\text{normal}} \pm \text{SD}$) on the right side and 0.97 ± 0.09 on the left side. Testing revealed no significant difference ($p < 0.05$) between both sides. The hVOR gain was classified as abnormal if gain values were below $\text{mean}_{\text{normal}} - 2\text{SD}$. Thus, the gain threshold was set to <0.79.

Physiological saccades were identified based on their peak eye velocity ($60°/\text{s} \pm 19$) and occurred with a latency of $215\ \text{ms} \pm 38$ after onset of vHIT. Refixation saccades were measured at an average latency of $146\ \text{ms} \pm 28$ (covert saccades) and of $245\ \text{ms} \pm 48$ (overt saccades). Peak eye velocity of refixation saccades was $249°/\text{s} \pm 51$ for covert saccades and $148°/\text{s} \pm 81$ for overt saccades, respectively.

In all peripheral vestibular disorders an ipsilateral gain reduction of the hVOR was recorded (overall mean: 0.44 ± 0.20; VN: 0.43 ± 0.20; VS: 0.54 ± 0.25; MD: 0.60 ± 0.20; BV: 0.44 ± 0.21). In 16 cases with unilateral vestibulopathy (20.0%) (VN: $n = 11$, VS: $n = 4$ and MD: $n = 1$), an additional gain reduction of the unaffected side was recorded (0.73 ± 0.07). Abnormal saccades did not appear on this side. In these cases the gain on the affected side was further reduced to 0.38 ± 0.21.

In the patient group (in cases of BV we counted both sides individually), we found a normal vHIT in 20.9% ($n = 27$) and an abnormal vHIT in 79.1% ($n = 102$). In 5.8% ($n = 3$) of VN there was a normal vHIT. Two VN cases demonstrated a hVOR gain reduction but without the occurrence of refixation saccades, one VN case exhibited neither an abnormal hVOR gain nor refixation saccades. In one case of BV (8.3%) the vHIT revealed an abnormal hVOR gain on both sides, but no refixation saccades could be observed. In VS and MD a normal hVOR was exhibited in 38.7% ($n = 12$) and 45.5% ($n = 10$), respectively (Table 1).

Table 1
Patients with normal and abnormal vHIT (hVOR).

Diagnosis	Number of patients	Normal vHIT	Abnormal vHIT
Vestibular neuritis	52	3 (5.8%)	49 (94.2%)
Vestibular schwannoma	31	12 (38.7%)	19 (61.3%)
Ménière's disease	22	10 (45.5%)	12 (54.5%)
Bilateral vestibulopathy	12	2 (8.3%)[a]	22 (91.7%)[a]
All disorders	117	27 (20.9%)	102 (79.1%)

[a] Pathological ears.