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Clinical Study

Compensatory saccade differences between outward *versus* inward head impulses in chronic unilateral vestibular hypofunction

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ABSTRACT

The horizontal head impulse test (HIT) is a valuable clinical tool that can help identify peripheral vestibular hypofunction by the refixation (compensatory) saccade that returns the eyes to the target of interest after the head has stopped. We asked if there were differences in the compensatory saccade responses during the HIT when the head was rotated away or toward straight ahead (outward *versus* inward). We also investigated the influence of a fixation target. Using scleral search-coils, we tested five patients with chronic unilateral vestibular hypofunction (UVH) and three healthy control subjects. In UVH patients, the latencies of both overt and covert saccades were longer when the head was rotated inward from an initially eccentric position, regardless of a visual target. The proportion of HIT with covert saccades was independent of a visual target. In control subjects no compensatory saccades were observed and there were no differences in either angular vestibulo-ocular reflex gain or latency between inward and outward HIT. Our data suggest that inward applied HIT in chronic UVH is more likely to include an overt compensatory saccade based on its lengthened latency. Neither latency nor the occurrence of covert compensatory saccades during HIT depended on a visual target, suggesting they have become a learned behavior in response to chronic UVH.

1. Introduction

The head impulse test (HIT) is an established way to test the angular vestibulo-ocular reflex (aVOR) at the bedside [1,2]. When the aVOR is normal, the eyes rotate opposite to the head movement through the angle required to keep images stable on the fovea. If the aVOR is impaired, the eyes move less than required and, at the end of the head rotation, the eyes are not directed at the intended target and the visual image is displaced from the fovea. A promptly-generated corrective saccade brings the image of the target back on the fovea. The identification of this corrective saccade is the signature feature of vestibular hypofunction and has greatly increased the utility of the bedside examination for identifying an aVOR deficit. The HIT has become an important part of the differential diagnosis of both acute and chronic vestibular disturbances [3–7].

Various terms have been used to identify the unique type of saccade that occurs in the direction of the aVOR and during the head rotation, including corrective saccade [8], gaze correcting saccade [9], refixation saccade [1], vestibular catch up saccades [10], and compensatory saccades [11]. More recently, two types of compensatory saccades have been named through the quantitative study of abnormal HIT – covert and overt [2]. Covert saccades occur during the head rotation, while overt saccades occur after the head has stopped moving.

From the original work of Halmagyi and Curthoys, the HIT was described as "…easiest to do this if the head was already positioned 20° or so away from the side to which it was about to be turned" [1]. The subject was instructed to fixate a target 1 meter away. Others have suggested that the head be moved from a neutral position (midline) to an eccentric position (lateral) [12]. Although both maneuvers would excite the vestibular afferents in the direction of rotation, and inhibit the vestibular afferents from the contralesional side, the initial eye position in the orbit or starting head position might, theoretically, affect the response, even though the amplitude and direction of head rotation are the

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same. We sought to determine if differences in the starting head position might influence the pattern of compensatory oculomotor behavior in a way that could affect the test's diagnostic sensitivity at the bedside.

2. Methods

We tested five patients with unilateral vestibular hypofunction (UVH) and three healthy controls. In all five patients, a corrective saccade was generated with the bedside ipsilesional HIT; the sign of a hypometric aVOR (Table 1). Three normal subjects (mean age = 41) had no history of vestibular or neurological disease and a normal bedside HIT. Participation in this study was voluntary and all subjects consented to participate in accordance with a protocol approved by the Johns Hopkins School of Medicine Institutional Review Board.

2.1. Scleral search coil technique

Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line in the earth-horizontal plane. Each subject fixed on a light-emitting diode (LED) target 124 cm directly in front of them. The room was otherwise completely dark. The movements of one eye were recorded around the three axes of rotation (horizontal, vertical, torsional) using a pair of search coils embedded in a silicone annulus. One was placed directly onto the sclera of the left eye to measure eye rotation [13]. The other search coil was embedded in a bite block and used to measure head rotation. Eye and head angular positions were sampled at 500 Hz at 16 bit resolution. Analog signals (pre-sampled) were low-pass filtered with a single-pole analog filter that had a 3 dB bandwidth of 100 Hz.

2.2. HIT

We measured the compensatory ocular motor response during passive (examiner-initiated) outward and passive inward HIT under two different viewing conditions: target on and target off. With the exception of the target on condition, the room was completely dark. For target off, the subjects were asked to imagine a target in front of them. The same examiner applied both inward and outward horizontal HIT with a mean 181 ± 36°/s head velocities and a mean 2518 ± 654°/s^2 head accelerations (Fig. 1). Subjects were instructed to relax their neck and keep their eyes on the LED (target on) or keep looking straight ahead (target off).

2.3. Data analysis

Angular positions for eye and head with respect to space (body) coordinates and eye with respect to head coordinates were represented by rotation vectors [14–16]. Results were calculated for only those trials in which HIT velocities were between 100°/s and 400°/s. Trials that included blinks or noise artifact were discarded. The onset of each HIT was identified with polynomial curve fitting [13]. Compensatory saccade latencies were determined by fitting a line to the eye velocity trace along the trajectory of the saccade and subtracting that point at which the fitted line crossed zero (ordinate) from the similarly determined point of head velocity onset. aVOR gain during HIT was calculated by dividing peak horizontal eye velocity by peak horizontal head velocity [17]. aVOR latency was defined as the difference in time (ms) for the head and eye velocities to reach 10°/s.

The frequencies of the compensatory saccades (overt or covert) from each HIT were determined. Each overt or covert saccade was visualized to insure accurate characterization. Overt saccades were defined as when peak eye acceleration was greater than 6000°/s^2, occurred in the opposite direction of the head rotation, and reached peak acceleration after the head had stopped moving. To distinguish overt saccades from incidental saccades, the corrective saccade had to begin within 170 milliseconds after the head stopped moving [18]. Covert saccades were defined as when peak eye acceleration was greater than 6000°/s^2, occurred in the opposite direction of the head rotation, and reached peak acceleration before the head had stopped moving [18].

2.4. Statistical analysis

We used the generalized estimating equation method and linear regression to analyze longitudinal data from repeated HIT. Data are presented as mean and one standard deviation. All reported p values were two-tailed, and the results were considered statistically significant at alpha <0.05 (StataCorp, College Station, TX, USA).

Table 1
Clinical characteristics of the five patients with chronic unilateral vestibular hypofunction

Age/Sex	Diagnosis	Onset (Test date)
51/F	Left VS	6 May 2010 (21 Sep 2010)
65/F	Left VS	8 April 2010 (6 Oct 2010)
59/F	Left VS	18 Nov 2010 (4 Jan 2011)
57/M	Left vestibular neuritis	6 Nov 2010 (16 March 11)
52/F	Right labyrinthectomy	16 March 2012 (8 May 2012)

F = female, M = male, VS = vestibular schwannoma.

3. Results

3.1. Normal subjects

There was no difference in peak head velocity (mean 148 ± 21°/s, $p = 0.34$), peak head acceleration (2751 ± 19.5°/s^2, $p = 0.3$), aVOR gain (mean 1.0 ± 0.1, $p = 0.89$), or aVOR latency (mean 9.1 ± 0.5 ms, $p = 0.22$) between outward HIT and inward HIT. Normal subjects did not make any compensatory saccades during HIT testing.

3.2. UVH

3.2.1. Ipsilesional HIT (inward versus outward)

We assessed 86 outward and 94 inward ipsilesional HIT from the five UVH patients with the target on (Table 2). There was no difference in stimulus applied. Each ipsilesional HIT had at least one compensatory saccade (Fig. 2). In this sample, the proportion of impulses with at least one overt saccade was greater for outward HIT (73% *versus* 55%) and the proportion with at least one covert saccade was greater for inward HIT (66% *versus* 82%), but neither difference was statistically significant. The latency of both overt and covert saccades was longer during inward HIT (Table 2). The latency of the aVOR was slightly shorter for the inward HIT (20.9 *versus* 19.1 ms, $p = 0.02$). Gain of the aVOR was slightly higher for the outward HIT (0.45 *versus* 0.42, $p = 0.001$). The percentage of HIT with a compensatory saccade increased with the greater extent of UVH (Fig. 3).

3.2.2. Target on versus target off

We assessed 92 outward and 85 inward ipsilesional impulses from the five UVH patients in complete darkness. In the target off condition, the proportion of impulses with at least one overt saccade was substantially reduced regardless of the direction of HIT (25% *versus* 66%, $p = 0.003$). In the target off condition we also found there was a large increase in the number of impulses that