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Feasibility of vestibulo-occular reflex gain measurement during large amplitude gaze shifts in acute unilateral vestibular dysfunction – a case series.

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ABSTRACT

Background: Head and eye movements orientate gaze during daily activities. In the case of vestibular disorders, these movements can lead to oscillopsia, avoidance behaviours or catch-up saccades. No specific test currently exists to assess large amplitude gaze shifts in clinical practice. **Objective**: The aim of the study was to determine the clinical feasibility of measuring the vestibulo-ocular reflex gain and the amplitude and velocity of the head during large amplitude gaze shifts in people with acute unilateral vestibular dysfunction. **Methods**: We retrospectively analysed recordings from a Frami-VCOR device in 4 individuals with acute unilateral vestibular dysfunction. Participants were asked to fix an illuminated diode and as soon as it switched off, to rotate their head as quickly as possible to fix a second illuminated diode. **Results**: The vestibular ocular reflex gain was smaller when the head turned towards the impaired side but head amplitude and velocity did not differ significantly between the impaired and healthy sides. Post-hoc analysis showed that the amplitude and the velocity of the first saccade (i.e. in the same direction as the head) differed significantly between the impaired and healthy sides. **Conclusions**: Evaluation of head and eye movements during a large gaze shift is feasible with a device available in clinical practice: this tool could be useful for clinicians who treat vestibular disorders.

KEYWORDS: large gaze shift, saccades, vestibular assessment, vestibular dysfunction

Background

The vestibular-ocular reflex (VOR) is usually assessed with a bithermal caloric test, rotatory chair test or video head impulse test (vHIT) [1, 2]. The first two tests are performed in darkness whereas the vHIT is performed in light with a target fixed in front. However, these tests do not assess large amplitude gaze shifts that are necessary in daily life. Indeed, daily activities require permanent changes of gaze amplitude and velocity depending on the context and environment. These changes involve well-defined structures and processes. For instance, for a gaze shift of less than 25°, a simple saccade is sufficient, however beyond this amplitude, head movements are necessary [3]. The head and eye coordination required to bring and then stabilize the gaze on the visual target is not evaluated in clinical practice.

Daemi & Crawford [4] have precisely modelled the kinematic processes of these movements: the head and the eyes start almost synchronously and in the same direction, however since the velocity of the eyeballs is greater than that of the head, the eyes arrive at the target before the head finishes turning. To compensate for this delay, the VOR triggers a movement of the eyes in the opposite direction at the same speed as the head. This coordination stabilizes the gaze on the target until the head stops [5]. The first phase of these movements is achieved by modulation of the VOR when the eyes and the head move in the same direction [3]. In healthy subjects, the gaze deviation caused by unexpected disturbance of head movement is corrected. These processes can be explained by the combination of a preset and constantly adapted motor control (feed-forward) and a vestibular feedback loop [6]. In subjects with vestibular impairment, performance during large amplitude gaze shift is highly variable and generally sub-optimal [7], leading to oscillopsia [8]

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and avoidance behaviours with reductions in head amplitude and head velocity [9, 10].

The aim of this study was to show the feasibility of measuring, in clinical practice, head and eye movements during an exercise involving gaze shifts to fix a target initially outside of the visual field in people with acute unilateral vestibular dysfunction. We hypothesised that gaze stabilization on the target would be suboptimal, as indicated by a reduction in the VOR gain, because of the vestibular dysfunction. We also hypothesised that the amplitude and velocity of the head during rotations towards the impaired side would be lower than for rotations towards the healthy side.

Methods

Participants

We retrospectively selected individuals with acute unilateral vestibular deficit, from the Paul Coste Floret rehabilitation hospital (France) between May 2019 and February 2020. According to French law for retrospective studies, we obtained the authorization to perform the study from the medical information department and the head of the department. We also informed the hospital correspondent for the National Commission for Data Protection and Liberties (CNIL-France) of the study. Participants received a letter informing them of the anonymous processing of their data and how to oppose the use of their data if they wished: none refused.

The inclusion criteria were adults (>18 years old) with a diagnosis of acute unilateral vestibular dysfunction, who had undergone evaluation with the Frami-VCOR device. The criteria for non-inclusion were: refusal of the use of their data, oculomotor paralysis, systematic closing of eyes during head rotation, comorbidity that prevented expression of refusal to participate and being under guardianship.

Material

All participants underwent evaluation of the position of the head and the eye with the Frami-VCOR device (Framiral, France) (25 Hz). This device includes a helmet with a mask on the front with an infrared camera module positioned on one or the other of the eye holes, and an inertial measurement unit (Xsens Technologies B.V., Netherlands) located at the top to measure head velocity in real time (Figure 1). A bar (Regled Framiral, France) with 2 diodes located at each extremity was positioned horizontally, 1 m in front of the participants to orientate their gaze. The diodes lit independently and randomly.

Set-up and procedure

Participants were seated in front of the diode bar, with the Frami-VCOR device over their face and the infrared camera in front of their right eye (Figure 1). They were asked to fix the illuminated diode with the uncovered eye and as soon as it switched off, to rotate their head as quickly as possible keeping their eyes open to fix the other diode that had just been illuminated. The shift in gaze orientation was about 50°. The recording continued until at least 5 saccades had been recorded for head movements to each side.We then calculated the amplitude and peak velocity of the head movement and the gain after the first saccade (eye velocity/head velocity) from 720 ms of data, starting 80 ms before the beginning of the eye movements. Figure 2 shows the position and velocity data measured during the large gaze shift on the healthy and impaired sides.

Statistical analysis

The statistical analysis was performed with Jamovi software (version 1.8.4). Owing to the small sample size (n=4), we described the quantitative variables by the median and interquartile range (IQR). We used the non parametric Wilcoxon test to compare results between the healthy and impaired sides. We performed an additional post-hoc analysis to analyse differences between the velocity and amplitude of eye movement as a function of the direction of head rotation. Significance was set at 5% (two-tailed).

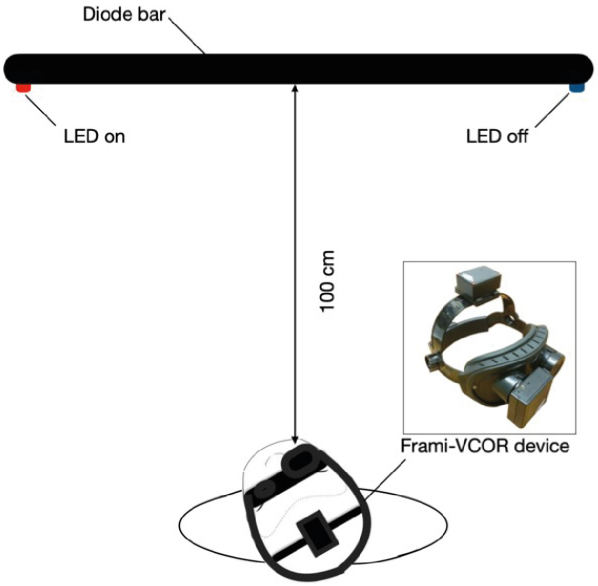

Figure 1 Illustration of the Frami-VCOR device and setup.

Results

We included data from 4 individuals with acute unilateral vestibular dysfunction, 2 women, age range 37 to 74 years, 3 with vestibular neuronitis and one following surgery for an VIII neuroma. All were assessed between 2 and 12 days after onset. The directional preponderance in the lateral semicircular canals measured with the vHIT ranged from 81 to 111%, the directional preponderance in the rotary chair ranged from 41 to 59% and the Dizziness Handicap Inventory [11] score ranged from 19 to 84 (Table 1).

The median gain after the first saccade was smaller on the impaired than the healthy side (0.62, IQR 0.13 vs 0.81, 0.12; p <0.001) (Figure 3A). Median rotation amplitude and peak head velocity did not differ between sides (43.97°, IQR 26.47° vs 37.17°, IQR 15.93°; p = 0.066 179.40°/s, IQR 98,56°/s vs 177.51°/s, IQR 104.00°/s; p = 1.000).

Visual analysis of the graphs (Figure 2) suggested a difference in velocity and amplitude of the first saccade. We therefore conducted a post-hoc analysis which showed that the median amplitude was lower on the impaired side than the healthy side (19.97, IQR 8.70° vs 29.70, 11.58°; p <0.001) (Figure 3D). Median peak velocity was lower on the impaired than the healthy side (214.40, 51.77 °/s vs 297.98, 70.40 °/s; p <0.001) (Figure 3E).

Discussion

This study demonstrated the feasibility of measuring head and eye movements during large gaze shifts in individuals with acute unilateral vestibular dysfunction in clinical practice. We found that VOR gain was significantly reduced during head rotations on the impaired side as compared with the healthy side. In contrast, head rotation amplitude and velocity did not differ between the impaired and healthy sides. The results for the VOR gain are consistent with previous work and show gaze instability during large amplitude gaze shifts [3, 6, 7, 9]. However, the results do not support our hypothesis that head rotation amplitude and velocity

Table 1 Description of the participants

	Age	Sex	Diagnostic	Time since onset (days)	DHI	vHIT (%)	VOR (%)
Participant 1	37	M	Left vestibular neuronitis	5	74	81	NA
Participant 2	56	F	Left vestibular neuronitis	2	84	103	59
Participant 3	67	M	Right vestibular neuronitis	2	44	111	50
Participant 4	74	F	Right acoustic neuroma	12	19	107	41

DHI: Dizziness Handicap Inventory, vHIT: Directional preponderance in the lateral semicircular canals measured during the video head impulse test, VOR: directional preponderance of the vestibulo-ocular reflex in the rotary chair test.

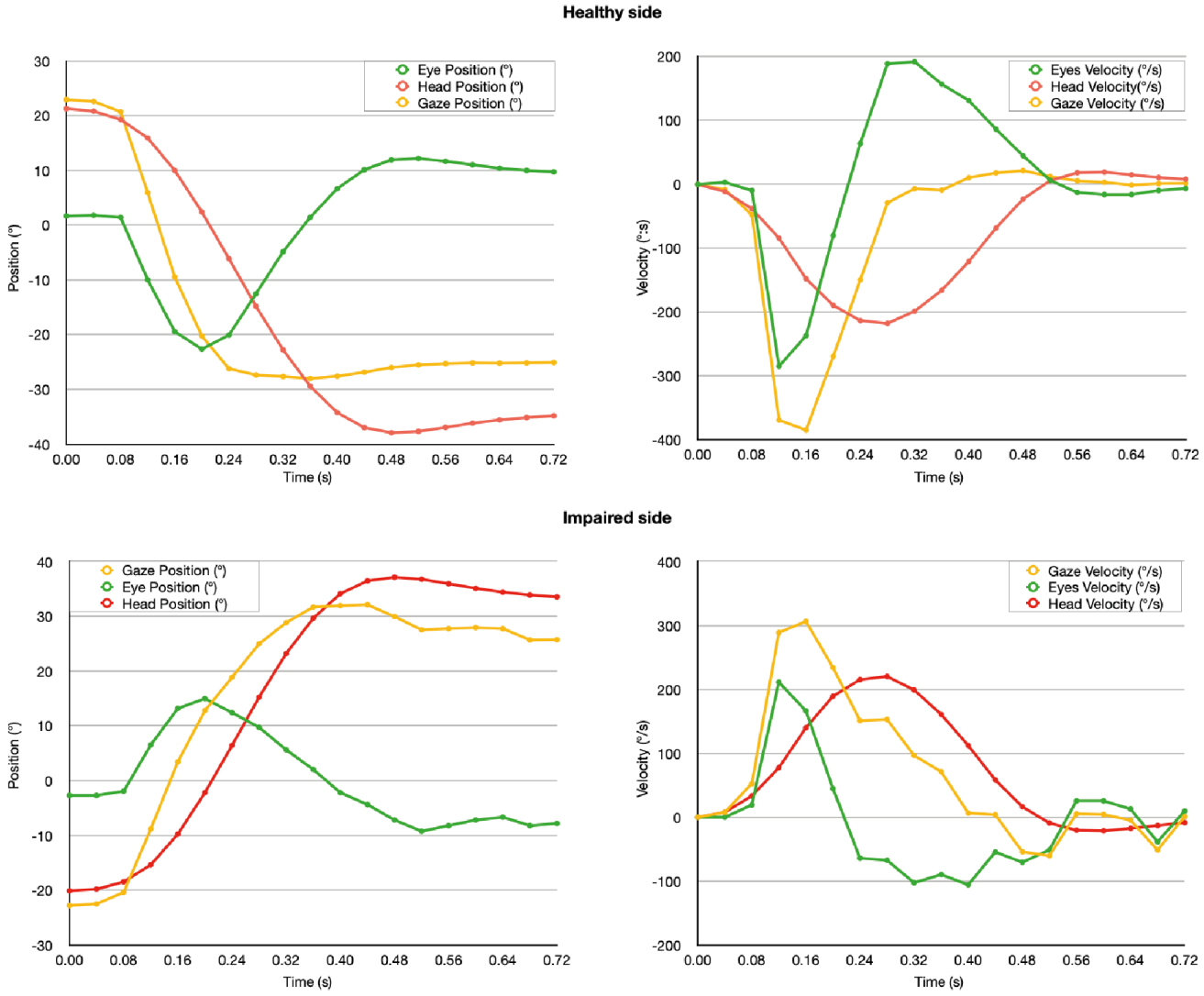

Figure 2 Data recorded by the Frami-VCOR device. The red line indicates head movement, the green line indicates eye movement and the yellow line the gaze shift (sum of head and eye movement). On the healthy side (top graphs), the head and eyes initially turn in the same direction but the velocity of the eyes is greater (first saccade). The gaze therefore reaches the target before the head is sufficiently rotated. After this first saccade, the gaze stabilizes on the target until the head stops rotating. On the impaired side (bottom graphs), peak eye velocity is lower and the gaze takes longer to stabilize (stabilization = gaze velocity around 0).

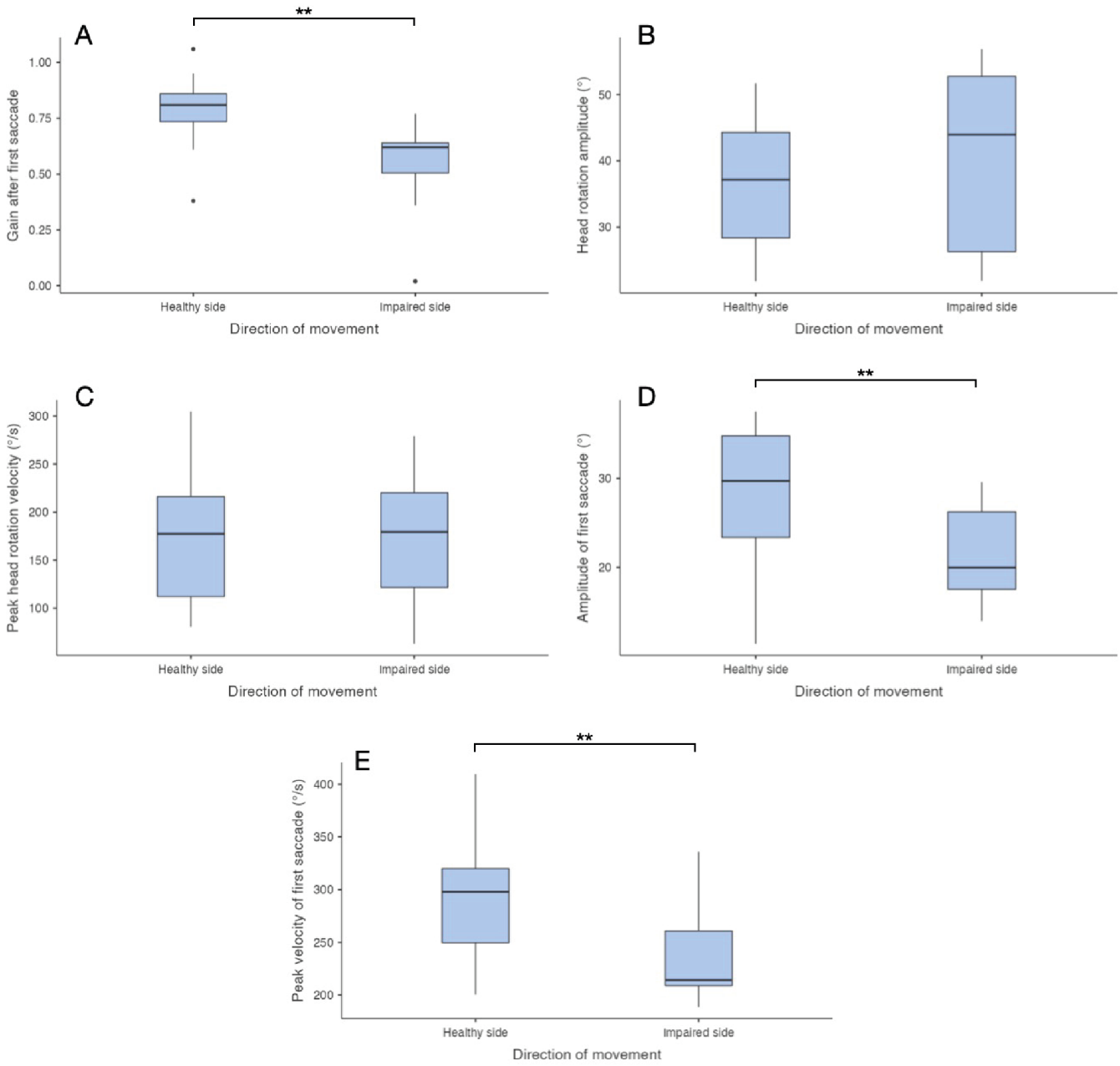

Figure 3 Box plot of VOR gain (A), head rotation amplitude (B), peak head rotation velocity (C), amplitude of first saccade (D) and peak velocity of first saccade (E). (*p <0.05, ** p <0.01)

would be reduced towards the impaired side, as found previously [9, 10]. We suggest that this may result from the fact the camera fixed on the Frami-VCOR obstructed the vision of one eye, and the vision of the other eye was reduced because of the small size of the eye hole; the visual field was reduced to about 50° which might have led to increased head rotations [11].

The results of post-hoc analyses showed for the first time that during a rotational movement of the head towards the impaired side, the amplitude and velocity of the first saccade in the same direction as the head, was significantly lower than towards the healthy side. These results can be interpreted in the light of motor control framework. According to this widely recognized theory, motor control uses predictions made from internal models and sensory inputs [12, 13]. It has been suggested that gaze stabilization during active head movement is possible owing to an internal model which includes an efference copy of head movement and coordination between the VOR prediction and the pre-programmed eye movements (PPEM) [14]. According to this theory, the internal model programs the necessary eye movements based on prediction of the PPEM and the counter-rotational movement of the eyes by the VOR. If this is correct, impairment of the VOR could have affected eye movement prediction in our gaze shift task. The update of the prediction of the impaired VOR in the internal model could decrease the amplitude and the velocity of the first saccade during large gaze shifts.

Another hypothesis is that the decrease in velocity and amplitude

of the first saccade reduces the variance of the final gaze stabilization. Indeed, Harris and Wolpert proposed that, in the presence of biological noise, the temporal profile of ocular motor control is selected to minimize the variance of the final position [15]. In our case, the loss of vestibular function leads to an unstable gaze, thus slower eye movement could reduce the motor control noise and facilitate gaze stabilization on the final target.

Further studies should determine whether the asymmetries in amplitude and velocity of the first saccade during large gaze shifts could be indicators of vestibular dysfunction and compensation [16, 17] in a large sample, with robust methodology, before and after vestibular compensation.

The main limitations of our study are the retrospective design and the absence of a control group of healthy subjects. Moreover, the small number of participants does not allow conclusions to be drawn.

Conclusion

In conclusion, our study showed that measurement of the amplitude and velocity of the head and eyes during large amplitude gaze shift exercises is feasible with the Frami-VCOR device in clinical practice. Our results also showed that the first saccade could be an indicator of vestibular dysfunction in subjects with acute unilateral dysfunction. However, this will need to be confirmed by prospective studies in a larger cohort of individuals with vestibular dysfunction and compared to healthy subjects.

Disclosure of interest

The authors declare they have no conflicting interests with the content of the article.

Authors' contribution

GLP collected and analyzed the data. GLP and GT participated in the publication writing.

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