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Abnormal motion perception in vestibular migraine

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Introduction

Vertigo caused by migraine, referred to as vestibular migraine (VM), is a frequently diagnosed but poorly understood entity. No specific oculomotor, postural, or perceptual defect has been described in this disorder and its pathophysiology remains uncertain. In this study we used quantitative psychophysical methods to investigate VM because recent work in normal humans suggests that the brain uses different mechanisms to generate percepts of head motion and reflexive vestibular-mediated eye movements. In particular, perception appears to be more dependent on central interactions between semicircular canal cues (which sense head rotation) and otolith cues (which sense gravity and linear acceleration) than eye movements. Given the high incidence of positional vertigo and nystagmus in VM, we hypothesized that canal-otolith integration may be abnormal in these patients and that this abnormality may be reflected in perceptual responses.

Materials and Methods

Subjects

Eight patients with VM (defined with the Neuhauser criteria¹), eight migraine patients with no history of vestibular symptoms, and eight normal subjects² were tested. All migraine patients met the International Headache Society criteria, all subjects were tested at least two weeks after their most recent vertigo or headache episode, and no subject took prophylactic migraine medications. The mean age did not differ between groups (ANOVA on ranks, $p = 0.65$). Informed consent was obtained from all subjects and the study was approved by the institutional ethical committee (IRB).

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Motion Stimuli

Three paradigms were used, allowing us to stimulate the canals and otoliths simultaneously (*dynamic roll tilt*), stimulate the canals in isolation (*roll rotation*), and stimulate the otoliths in isolation (*quasi-static roll tilt*). Subjects were rotated about the nasal-occipital axis centered between the ears while seated upright (for dynamic and quasi-static roll tilt) or while supine (for roll rotation). For dynamic roll tilt and roll rotation, each trial consisted of a single-cycle of sinusoidal acceleration, which produced a unidirectional angular velocity and displacement. Quasi-static roll tilt thresholds were measured by tilting subjects at a very low velocity (0.125 deg/s). To minimize potential non-vestibular motion cues, trials were performed in the dark, skin surfaces were covered and the body was padded, and hearing was masked with sound provided via headphones.

Psychophysical task

We used a “forced-choice, direction-recognition task” to determine perceptual thresholds. A single motion stimulus was provided for each trial, the direction of motion was random, and after each motion the subject had to press one of two buttons (one in the right hand, one in the left hand) to indicate if they felt they had moved to the right or left. If they were uncertain they were required to guess. Perceptual thresholds were determined using a “three-down, one-up” staircase paradigm.³ Three consecutive correct responses were required before the acceleration of the sinusoid was reduced, while the acceleration was increased after one incorrect response. After five direction-reversals had occurred, the perceptual threshold was defined as the mean of the last two reversal values.³

Motion sickness and velocity storage

These were quantified with the revised Golding questionnaire and by calculating the dominant time constant of the horizontal vestibulo-ocular reflex (VOR).

Results

The mean threshold for VM subjects tested with *dynamic roll tilt* at 0.1 Hz (Figure) was substantially lower than the migraine (t-test: $p < 0.001$) or normal (t-test: $p = 0.001$) subjects, but the normal and migraine groups did not differ (t-test: $p = 0.69$). In contrast, at 1.0 Hz thresholds did not differ between groups. Overall, thresholds on the dynamic roll tilt test depended on subject group ($p = 0.02$) and motion frequency ($p < 0.001$), with a significant interaction (Holm-Sidak test: $p < 0.001$) between these factors. Thresholds during *roll rotation* (Figure) did not differ between VM and normal subjects at either 0.1 Hz (t-test: $p = 0.81$) or 1.0 Hz (t-test: $p = 0.53$), and thresholds measured during *quasi-static roll tilt* (Figure) did not differ between the three groups (ANOVA: $p = 0.99$). No significant difference in VOR time constant was observed between the normal (20.2 $\pm$ 1.1 sec) and VM (21.3 $\pm$ 1.6 sec) groups (t-test: $p = 0.55$), nor were there differences in motion sickness susceptibility scores between the three subject groups (ANOVA: $p = 0.44$).

Discussion

We found a large reduction in perceptual motion thresholds in VM patients, compared to normal and migraine subjects, when they were tilted dynamically in the roll plane at a mid-frequency but not when they were tilted at a high frequency, tilted quasi-statically or rotated in roll. These results suggest that canal and otolith cues, which are modulated in tandem during dynamic roll tilt, are synthesized differently in subjects with VM. No difference was evident at the higher tilt frequency (1.0 Hz), probably because canal cues dominate perception at this frequency (note that roll tilt and roll rotation thresholds at 1.0 Hz did not differ).

Since perceptual thresholds were reduced in VM, it appears that the signal encoding head motion in vestibular regions of the thalamus or cerebral cortex are enhanced (relative to the neural noise inherent in sensory transduction and subsequent central processing) during combined canal-otolith stimulation. This could reflect primary changes in thalamic or cerebral cortical processing or could be mediated by ascending inputs. Areas of particular interest include the cerebellar nodulus and uvula, since they are required for normal canal-otolith integration. Altered signal processing in this brain region could explain changes in dynamic tilt thresholds without changes in canal or otolith-mediated thresholds.

Conclusion

Given the multiple potential interactions between migraine and the vestibular system, it is likely that vertigo in VM is multi-factorial and could include labyrinthine as well as neurologic components. Despite this complexity, we have described a specific abnormality related to percepts of head motion that appears to be derived from changes in canal-otolith integration in the brain.

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Figure.

Perceptual thresholds (in deg) for the three motion paradigms. Icons represent mean values $\pm$ one SEM. Note the difference in y-axis scale in Fig. B compared to Figs. A and C.