Vertical Component of the Caloric Response, Including a Caloric Second Phase Provoked by Positional Change: A Preliminary Report

Sachiko Aoki, Yasuko Arai, Natsumi Ide, Eisaku Sugiura, and Keisuke Miyajima

Department of Otolaryngology, Tokyo Women's Medical University, Medical Center East, Tokyo, Japan

Abstract: We measured the horizontal and vertical components of caloric nystagmus in 120 ears of 60 vertiginous patients who had moderate to vigorous caloric first-phase response in both ears, no spontaneous nystagmus, and no severe disorders in the central nervous system. We provoked a caloric first phase in the supine position with 5 ml of water at 20°C for 15 seconds. We provoked a caloric second phase by changing the position of a patient from supine to sitting after the end of the first phase. The horizontal component of the caloric second phase was recorded in 108 of 120 ears (90%). The vertical component was recorded in 57 of 120 ears (48%) during the caloric first phase and in 51 of 120 ears (43%) during the caloric second phase. We suspected that the vertical component of the caloric first phase was mainly due to the inhibition of the anterior semicircular canal.

Key Words: caloric nystagmus; human; positional change; vertical component; vertical semicircular canals

Use the caloric response, including the caloric second phase provoked by positional change.

PATIENTS AND METHODS

We investigated patients who from 2004 to 2005 visited Tokyo Women's Medical University, Medical Center East, with the complaint of vertigo. The caloric first phase was provoked by pouring 5 ml of water at 20°C into one ear of a supine patient for 15 seconds. After the caloric first-phase response declined and no nystagmic beat was seen for more than 10 seconds, the patient's upper body was lifted 90 degrees to place the lateral semicircular canal in the gravitational horizontal plane. We provoked the caloric second phase with this maneuver. All eye movements were recorded with eyes open in total darkness by electronystagmography through Ag/AgCl electrodes.

The data of those patients who fulfilled the following conditions were submitted to the study: (1) moderate to vigorous horizontal component provoked in the caloric first phase in both ears, (2) absence of severe disorders in the central nervous system, (3) absence of spontaneous nystagmus with eyes open in total darkness, (4) absence of positioning nystagmus with Stenger's maneuver, and (5) electronystagmography in good condition.

RESULTS

The data of 60 patients (17 male and 43 female; aged 21–69 years; mean age, 51.9 years) were qualified for the analysis. The horizontal component of caloric nystagmus was recorded in 108 of 120 ears (90%) in 60 patients. The vertical component was recorded in 57 of 120 ears (48%) during the caloric first phase and in 51 of 120 ears (43%) during the caloric second phase.

Reprint requests: Sachiko Aoki, MD, Tokyo Women's Medical University, Medical Center East, 2-1-10 Nishiogu, Arakawaku, Tokyo 116-8567 Japan. Phone: +81-3-3810-1111; Fax: +81-3-3894-7988; E-mail: yaot@dnh.twmu. ac.jp

In 53 of 57 ears (93%), the vertical component of the caloric first phase was directed to the upper eyelid, regardless of the irrigated side. In 50 of 51 ears (98%), the vertical component of the caloric second phase was directed to the lower eyelid, also regardless of the irrigated side.

DISCUSSION

We have reported elsewhere about the caloric second phase that is provoked by a subject's positional change [1]. The vertical component in the caloric first phase has been reported to beat mainly toward the upper eyelid in normal subjects [2-8]. Our findings confirmed the results in vertiginous patients.

What is the origin of the vertical component of the caloric first phase? Yagi et al. [2,3] reported that all semicircular canals were inhibited by cold calorization in the supine position. Suzuki et al. [9] and Cohen et al. [10] proved, in an animal experiment, that the vertical nystagmus toward the upper eyelid is generated by inhibition of the anterior canal or by excitation of the posterior canal. Aw et al. [11] declared that the contribution of the anterior canal to the caloric response is much greater than that of the posterior canal.

Therefore, we suspected that the vertical component toward the upper eyelid in the caloric first phase, which we observed most frequently in our study, appeared when inhibition of the anterior semicircular canal overwhelms that of the posterior semicircular canal. We considered that the vertical component toward the lower eyelid in the first phase, which we saw in a small number of our patients, was a result of inhibition of the posterior semicircular canal that surpassed inhibition of the anterior semicircular canal.

A vertical component was *not* recorded in 63 of 120 ears (52%) in our study during the caloric first phase. We believe that this was because inhibition of the anterior semicircular canal and the posterior semicircular canal had balanced and canceled each other out.

The origin of the vertical component in the caloric second phase is not clear and must be investigated further.

CONCLUSION

In the caloric first phase, we recorded the vertical component in 57 of 120 ears (48%), which was directed to the upper eyelid in most of the ears. We believed this was due mainly to the inhibitory effect of the anterior semicircular canal. The vertical component was recorded in 51 of 120 ears (43%) in the caloric second phase, provoked by positional change. The vertical component of the caloric second phase was mostly directed to the lower eyelid, and its origin remained unclear.

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