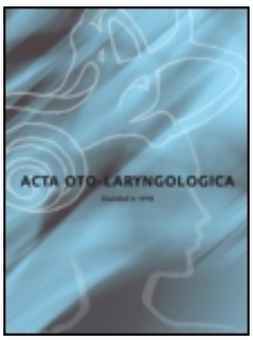

Acta Oto-Laryngologica

ISSN: 0001-6489 (Print) 1651-2251 (Online) Journal homepage: http://www.tandfonline.com/loi/ioto20

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To cite this article: S. T. Aw, G. M. Halmagyi, D. V. Pohl, I. S. Curthoys, R. A. Yavor & M. J. Todd (1995) The Effect of Unilateral Posterior Semicircular Canal Inactivation on the Human Vestibulo-Ocular Reflex, Acta Oto-Laryngologica, 115:sup520, 260-262, DOI: 10.3109/00016489509125243

To link to this article: http://dx.doi.org/10.3109/00016489509125243

Acta Otolaryngol (Stockh) 1995; Suppl 520: 260–262

The Effect of Unilateral Posterior Semicircular Canal Inactivation on the Human Vestibulo-Ocular Reflex

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Aw ST, Halmagyi GM, Pohl DV, Curthoys IS, Yavor RA, Todd MJ. *The effect of unilateral posterior semicircular canal inactivation on the human vestibulo-ocular reflex.* Acta Otolaryngol (Stockh) 1995; Suppl 520: 260–262.

The responses to rapid, passive, unpredictable, low amplitude (10–20°), high acceleration (3,000–4,000°/s²) head rotations were used to study the human vestibulo-ocular reflex (VOR) in pitch and yaw plane after unilateral posterior semicircular canal occlusion (uPCO) in 10 subjects. The results from these 10 uPCO subjects were compared with those from 18 normal subjects. The VOR gains at a head velocity of 200°/s in the uPCO subjects were: pitch upward = 0.62 ± 0.06, pitch downward = 0.87 ± 0.11, yaw ipsilesion = 0.78 ± 0.06, yaw contralesion = 0.79 ± 0.10 and in normal subjects were: pitch upward = 0.92 ± 0.06, pitch downward = 0.96 ± 0.04, yaw right = 0.88 ± 0.05, yaw left = 0.91 ± 0.12 (group means ± two-tailed 95% confidence intervals). The results showed that the pitch-vVOR gain was significantly ($p < 0.05$) decreased in response to upward head impulses whereas in response to downward, ipsilesion and contralesion head impulses were not significantly different ($p > 0.05$) from the normals. This study shows that there is 30% permanent residual deficit of the upward pitch-vVOR with an up-down asymmetry in pitch-vVOR gain following inactivation of a single posterior semicircular canal and that compensation of pitch-vVOR function is incomplete. *Key words: vertical vestibulo-ocular reflex, pitch, human unilateral posterior semicircular canal occlusion.*

INTRODUCTION

The human vestibulo-ocular reflex (VOR) generates smooth compensatory eye rotations which stabilize the direction of gaze during head rotations. Occlusion of a single semicircular canal mechanically inactivates that canal's response to angular acceleration without influencing its neural innervation or tonic vestibular activity (1). There were several animals studies on the VOR function after canal occlusions (2–5). This study measured the VOR function in human subjects following unilateral posterior semicircular canal occlusion (uPCO) using a rapid, passive, unpredictable, high acceleration, low amplitude head rotations ("head impulses"). The results showed a significant 30% deficit in upward pitch-vVOR following uPCO with an up-down asymmetry in pitch-vVOR gain.

METHODS AND MATERIALS

Subjects

We studied 10 subjects (33–72 years, mean = 57.9 years) who had undergone unilateral posterior semicircular canal occlusion as treatment for intractable benign paroxysmal positional vertigo and compared the results to 19 normal subjects (21–55 years, mean = 32.8 years). The 10 (6 right and 4 left) uPCO subjects were studied from 1 week to 15 months (mean ± 1 s.d. = 5.8 ± 4.2 months) following operation. All normal and uPCO subjects gave informed consent, and all protocols were approved by the RPA Hospital Human Ethics Committee.

Effectiveness of posterior semicircular canal occlusion

All the uPCO subjects in this study had pre- and post-operative baseline audiometry. The mean difference in hearing threshold at 2 kHz following uPCO was −4 ± 7.3 dB. Only 1 subject had a significant hearing loss of 30 dB at 2 kHz following uPCO. All the remaining subjects had a drop of only 5 dB or less.

Recording system

Horizontal and vertical angular displacement of the head and the eye were recorded using the search coil technique with two search coils (Skalar, Delft), one fixed to the nose-piece of a spectacle frame worn by the subject and the other on the subject's left eye. Signals were recorded from the search coils while the subject was seated upright in the centre of a 2 m³ transmitter coil field (CNC Engineering, Seattle) and fixated on a target at a distance of 1 m. The search coil signals were recorded at 1,000 Hz sampling frequency with 16-bit resolution and a bandwidth of 0–100 Hz by a PDP-11/73 computer. Data acquisition programs were written in DAOS running under the TSX-plus operating system. The head coil was calibrated before the testing on a 25 cm diameter 3-axis plexiglass gimbal in 5° steps over the range ±20° in pitch and yaw.

Protocol

The "head impulse" stimulus was a rapid, passive, unpredictable (in timing and direction), low ampli-

 ISSN 0365-5237

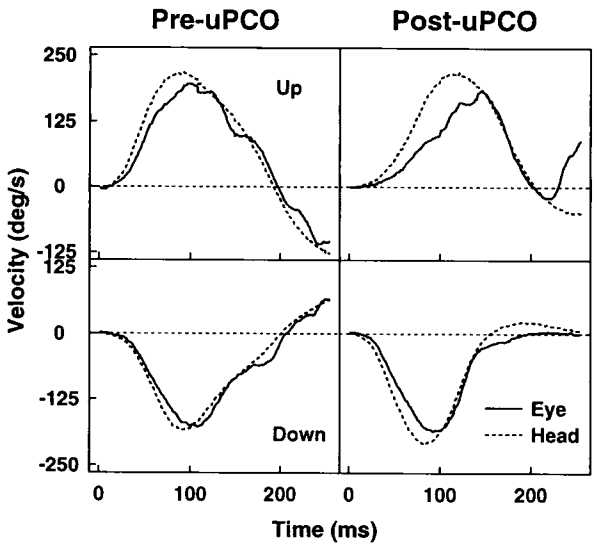

Fig. 1. Head and compensatory eye velocity responses during pitch head impulses before and after unilateral posterior semicircular canal occlusion. The eye signals are inverted for illustration.

tude (10–20°), high-acceleration (3,000–4,000°/s^2) angular head rotation in pitch: upwards and downwards; and in yaw: rightwards and leftwards were tested in all subjects. *In vivo* eye coil calibration was performed with the subject fixating on target while it was moved through a range of ±20° at 5° intervals in pitch or yaw. A linear regression analysis was carried out on-line to measure the gain, offset and linear correlation coefficient of the head and eye calibration. The head impulses in pitch (10 up and 10 down) or yaw (10 right and 10 left) were delivered by an operator standing behind the subject, when cued by the data acquisition software, while the subject fixated on the target.

RESULTS

Responses to impulses before and after uPCO

Fig. 1 shows pre- and post-operative pitch head and compensatory eye velocities from one subject before and after uPCO. The eye signals are inverted for illustration. The pre-operative horizontal and pitch-vVOR gains were within the two-tailed 95% confidence intervals of normals. Following uPCO, compensatory eye position and velocity lagged during upward head impulses; during downward pitch impulses it was similar to the pre-operative response. There was a clear up-down asymmetry in the pitch-vVOR gain.

VOR gains

Group means and 95% confidence intervals of eye velocity as a function of head velocity were used to compare the differences in pitch vertical and yaw

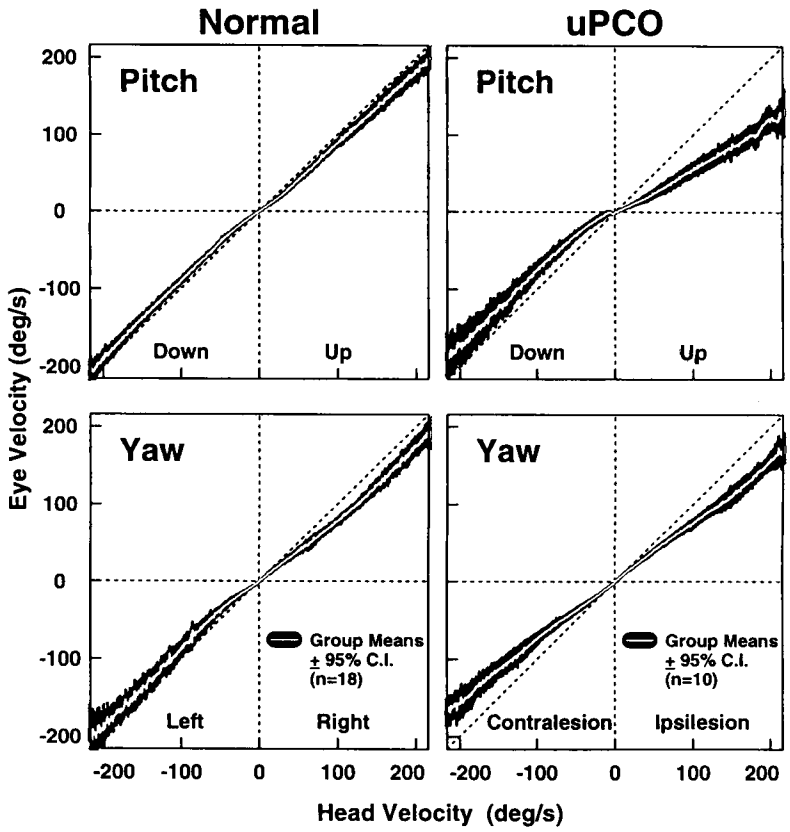

Fig. 2. Group means and two-tailed 95% confidence intervals of eye velocity as a function of head velocity in normal and unilateral posterior semicircular canal occlusion subjects during pitch and yaw head impulses.

horizontal VOR gains during head impulses between the 10 uPCO subjects and the 18 normal subjects (Fig. 2). The mean VOR gains ±95% confidence intervals at head velocity of 200°/s in uPCO subjects were: pitch upward = 0.61 ± 0.06, pitch downward = 0.85 ± 0.11, yaw ipsilesion = 0.78 ± 0.11, yaw contralesion = 0.78 ± 0.11 and in normal subjects were: pitch upward = 0.92 ± 0.06, pitch downward = 0.96 ± 0.04, yaw rightward = 0.88 ± 0.05, yaw leftward = 0.91 ± 0.12.

The mean VOR gain in normal subjects during pitch head impulses was close to unity. Inactivation of a single posterior semicircular canal in uPCO subjects reduced the mean pitch-vVOR gain during upward head impulses to about 70% of that in normal subjects and the difference is significant ($p < 0.05$). During downward head impulses, the mean downward pitch-vVOR gain in the uPCO subjects was not significantly different ($p > 0.05$) from normals. The mean VOR gains during ipsilesion and contralesion impulses in uPCO subjects was not significantly different ($p > 0.05$) from that in normals. The results show that unilateral loss of posterior semicircular canal function produced a permanent deficit in upward pitch-vVOR gain of about 30% and an up-down asymmetry in pitch-vVOR.

DISCUSSION

Upward pitch-vVOR deficit following uPCO

Since occlusion of posterior semicircular canal modifies canal dynamics without disrupting spontaneous primary afferent activity, the deficit in the upward pitch-vVOR is due to absence of excitatory input from the cupula of the inactivated canal during head rotation. Barmack (5) reported a reduced vertical VOR gain following bilateral anterior semicircular canal occlusions in rabbits and Halmagyi et al. (6) reported a reduced upward pitch-vVOR gain following unilateral vestibular deafferentation in humans. In this study, 5 subjects were tested more than 6 months after uPCO. These subjects would have been exposed to retinal slip velocities greater than 30°/s during locomotion and this should provide a strong stimulus for VOR adaptive plasticity. However the uPCO subjects showed a permanent 30% deficit in upward pitch-vVOR gain following unilateral inactivation of the posterior semicircular canal. This shows that bilateral excitatory canal inputs are essential for a normal upward pitch-vVOR. This data is contrary to the study by Paige (4) which demonstrated adaptive recalibration in freely moving monkeys following unilateral horizontal semicircular canal occlusion, in response to low-frequency, low-velocity sinusoidal stimulus. The discrepancy may be due to differences between the stimuli used in these experiments.

ACKNOWLEDGEMENT

Supported by the National Health and Medical Research Council.

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