

cervical muscles (cVEMPs). The oVEMPs are considered to represent vestibular function mediated by crossed vestibulo-ocular pathways, because they are present in patients without hearing, but absent on the contralateral side in those with unilateral vestibular loss [2].

The oVEMPs in response to BCV have been suggested to reflect the function of the otolith organs, especially the utricle. Physiological studies using guinea pigs showed that moderate BCV selectively activates irregular otolithic primary vestibular neurons including some that originate in the utricular maculae [1]. Clinically, oVEMPs to BCV are reduced or absent in patients with unilateral superior vestibular neuritis, affecting the nerves from the horizontal and anterior canals and the utricular macula, but sparing the saccule and inferior vestibular function [3].

On the other hand, the origin of the oVEMPs to ACS is still unclear, while several studies suggested that oVEMPs to ACS are saccular in origin.

Objective: To compare the oVEMPs to ACS and BCV in patients with superior vestibular neuritis, to determine the role of the saccule in generating oVEMPs.

Methods: The n10 component of the oVEMP to BCV and ACS was measured in 12 patients (age 23–70 years) with unilateral superior vestibular neuritis.

For measuring oVEMPs, the active EMG electrodes were placed just below the eyelid and the reference electrodes were placed 3 cm below the active electrode. The ACS stimuli were 500 Hz tone burst (95 dBnHL, 4 ms in duration) delivered monaurally by a calibrated headphone. BCV stimuli were 6ms 500 Hz tone bursts delivered by a stimulator (Bruel and Kjaer minishaker 4810) to the midline forehead of the patient at the hairline (Fz).

Asymmetry ratio (AR) for n10 amplitude of the oVEMP was calculated using a following formula: Asymmetry Ratio (AR) = ((larger n10 – smaller n10) / (larger n10 + smaller n10))* 100

Results: All patients had normal cVEMPs on both sides. The average AR for cVEMPs to ACS was $9.1 \pm 1.1\%$ (SE here and below). In most patients with superior vestibular neuritis, the contralateral oVEMPs in response to ACS of the affected ear were reduced or absent. The average AR for oVEMPs to BCV was $65.2 \pm 8.9\%$, and that for oVEMPs to ACS was $61.4 \pm 11.5\%$. There was no significant difference between the mean value of the oVEMPs to BCV and ACS.

There was a very close relationship between the size of AR for oVEMPs to ACS and that for oVEMPs to BCV ($r = 0.87, p < 0.001$).

Conclusion: The n10 component of the oVEMP to ACS is probably mediated predominantly by the contralateral superior vestibular nerve and so most likely by the utricular receptors and afferents.

References

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About correlation between centre of pressure, trunk, and head sways during quiet upright stance

Angelo Buizza, Paola Ferraris, Roberto Gandolfi, Remo Lombardi, Eleonora Pilato
University of Pavia, Pavia, Italy

In order to better understand the mechanisms of orthostatic balance, centre of pressure (CoP), trunk, and head sways were simultaneously measured during quiet upright stance, and compared to each other, looking for possible correlation.

Methods: A total of 16 healthy young adults served as subjects. They were asked to keep orthostatic position for 120 s, once with open and once with closed eyes. COP sway was measured by force platform, trunk oscillation by inclinometers placed at the sternum level, trunk and head angular velocities by miniature gyroscopes, placed at the sternum and the skull vertex, respectively. Cross-correlation functions were used to compare sways measured at different levels to each other. In particular, antero-posterior (AP) and medio-lateral (ML) components of CoP sway were correlated to the homologous components of trunk oscillation, and AP and ML trunk oscillation velocities to the homologous head oscillation velocities.

Results: Clear and consistent positive correlation was found between trunk and head sways. In 83% of cases, cross-correlation functions presented one sharp

peak near the origin, and much lower values elsewhere. Mean correlation delays in the different conditions (open or closed eyes) and directions (AP and ML) ranged 17 to 47 ms, and weren't significantly different from 0 at $p = 0.05$ (t -test). Cross-correlation functions computed inside a 20 s sliding window showed that sharp and steady correlation was kept all along the test duration (120s).

CoP-trunk correlation was much less clear. Cross-correlation peaks as sharp as those observed between trunk and head velocities could never be observed. However, in many instances the two sways did show almost parallel time courses. This could either extend to almost the whole test duration or be limited to more or less shorter periods, and was much more frequent in ML than in AP plane, where, in general, the two sways appeared to be uncorrelated. However, in a few instances, in the AP plane they seemed to be phase opposed.

Discussion: Sharp cross-correlation peaks with almost no delay show that trunk and head moved almost synchronously, like one rigid body. This was consistent with the fact that in either plane (AP or ML) and vision condition (eyes open or closed) the sway velocity distributions of trunk and head were similar to each other, and the corresponding average velocity ranges (5th to 95th percentile of sway velocity distribution) were not significantly different from one another at $p = 0.05$ (t -test).

Poorer correlation between CoP and trunk sways is a likely consequence of the presence of the hip joint, and the inertia of the trunk-arms-head system, linked to this joint. This makes the trunk over the hip intrinsically less steady, and more moveable, than the head over the neck. Interestingly enough, CoP-trunk correlation was better in ML plane, meaning a stronger tendency to move together in this plane, than in AP one, and this may easily be explained by the different stiffness of the hip joint in the two planes, due to the different geometry.

Different degrees and modes of correlation between CoP and trunk sway, from almost parallel patterns throughout the whole test to seemingly phase opposed patterns, suggest the use of different balance strategies, that further investigation might possibly elucidate. It is also possible that the two sways convey different, maybe complementary, information about balance control, and it could possibly make sense to consider also trunk movements in the diagnostic approach to balance control. This conclusion is consistent with previous suggestions by different authors (e.g.: Kamen et

al. 1998; Allum et al. 2001; Mayagoitia et al. 2002; Moe-Nilssen and Helbostad 2002).

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3D analysis of benign positional nystagmus due to cupulolithiasis in posterior semicircular canal

Takao Imai¹, Atsuhiko Uno¹, Suetaka Nishiike¹, Tadashi Kitahara², Arata Horii³, Noriaki Takeda⁴, Hidenori Inohara¹

¹Osaka University Graduate School of Medicine, Osaka, Japan

²Department of Otolaryngology, Osaka Rosai Hospital, Osaka, Japan

³Department of Otolaryngology, Suita Municipal Hospital, Osaka, Japan

⁴Department of Otolaryngology, Tokushima University Graduate School of Medicine, Tokushima, Japan

Objective: Recently, it is recognized that the pathophysiology of benign paroxysmal positional vertigo (BPPV) is cupulolithiasis or canalolithiasis in either posterior semicircular canal (PSCC) or horizontal semicircular canal (HSCC). Patients with cupulolithiasis in HSCC show apogeotropic positional nystagmus, while those with canalolithiasis in HSCC show geotropic positional nystagmus. Thus, the differential diagnosis of cupulolithiasis from canalolithiasis can be made by the direction of the positional nystagmus in patients with the HSCC type of BPPV (H-BPPV). On the other hand, in patients with the PSCC type of BPPV (P-BPPV), cupulolithiasis in PSCC induced the vertical-torsional positional nystagmus, of which direction is the same as that induced by canalolithiasis. In the present study, an attempt was made to diagnose cupulolithiasis in patients with the posterior semicircular canal (PSCC) type of benign paroxysmal positional vertigo (P-BPPV). **Methods and Results:** We first, three-dimensionally analyzed the vertical-torsional positional nystagmus in 111 patients with P-BPPV and evaluated its time constant. This parameter showed a wide variation that could be divided into two groups: one lasting more than 40 sec in 8 patients and another below 20 sec in 103 patients. Since the time constant of the positional nystagmus induced by cupulolithiasis was much longer than that induced by canalolithiasis, this finding suggests that cupulolithiasis in the PSCC induced the vertical-torsional positional nystagmus with a long time constant in the group of 8 patients. We also found that the vertical-torsional positional nystagmus disappeared in these patients at the neutral head position, where the axis of the cupula of affected PSCC