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Reply to the Commentary on Luis et al. "Spontaneous Plugging of the Horizontal Semicircular Canal With Reversible Canal Dysfunction and Recovery of Vestibular Evoked Myogenic Potentials"

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We reported on an unusual patient who presented in the emergency room with symptoms and signs of an exceptional hBPPV (1): acute spontaneous and spinning vertigo, nausea, vomiting, spontaneous nystagmus, and VOR gain deficit on the right side. After being treated with liberatory maneuvers, the patient was free of symptoms and nystagmus and was released from the emergency unit. This case was special in four respects: 1) gravity dependence of spontaneous nystagmus; 2) recovery of the horizontal VOR gain on the affected right side immediately after treatment; 3) asymmetry of VEMP (both cVEMP and oVEMP) to air-conducted sound (ACS) before and 2 days after treatment; and 4) VEMP recovery 30 days after treatment. We hypothesized that the most plausible explanation for all effects is a reversible horizontal canal dysfunction due to the presence of a plug.

The Commentary by … (this issue) on our case report (1) reveals the ongoing controversy surrounding the hypothesis of a canal origin of VEMP to ACS. At the heart of the controversy lies the challenge which this hypothesis poses to the widely accepted interpretation that VEMP responses to ACS have an otolithic origin. The Commentary raised a number of issues related to Luis et al. (1) and Zhu et al. (2). Therefore, this joint reply by the authors of both studies aims to address the concerns in the Commentary by … and to clarify several important issues related to the technical aspects of VEMP testing, the time course of VEMP and VOR gain recovery in the reported patient, the observed eye velocity saturation as a possible sign for a canal plug, and to the literature on human and animal VEMP neurophysiology.

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VEMP Testing

First, the Commentary by … raised doubts as to whether we met the minimum requirements for interpreting VEMPs to ACS.

Both VEMP and audiometry (pure tone and acoustic impedance, i.e., tympanometry with stapedial reflexes) are always evaluated as bundle tests in the lab of the first author. Both tests were performed on all occasions from day one on and were unremarkable. Otoscopy, which is mandatory in any patient presenting to an ENT department, was also unremarkable. The phones were correctly inserted by an experienced otologist under direct viewing conditions with headlight. Nevertheless, as in all cases of absent or asymmetric response, the patient was still asked if he could hear the stimulus; the examiner himself heard the stimulus, and the cables were double-checked and swapped. A technical failure can therefore be excluded as a possible explanation for the simultaneous absence and recovery of both cVEMP and oVEMP.

To avoid all this redundant information and to keep the paper, a Clinical Capsule Report, as short as possible, the information provided was formulated simply: “the neurootological examination was otherwise unremarkable”.

Association of VOR with VEMP recovery

In view of the apparent dissociation between VOR and VEMP recovery times in the reported patient, the authors of the Commentary raised the issue of a “logical problem” in our conclusion (1). The VOR gain recovered early after treatment, while the recovery of both oVEMP and cVEMP was delayed. In our Discussion (1) we explicitly emphasized that “this differentially delayed normalization (…) remains to be explained”. Therefore, we provide the following more detailed considerations:

A possible but admittedly highly speculative explanation might be the residual mild gain asymmetry of 15% (0.81 on the right versus 1.09 on the left) reported for Day 3, i.e., 48 hours after the treatment. As can be seen in the eye and head velocity regression diagram in FIG. 1, this mild gain asymmetry is the result of a right gain deficit occurring only at head velocities above 150 °/s. In the Discussion this was attributed to “continuing changes in biomechanical semicircular canal properties”. It might well be that such a mildly reduced gain is already a sufficient condition to have an effect on the VEMPs. The interpretation in the Commentary by … that “canal function and VEMPs returned independently” is therefore not supported by the data we presented (1). On the contrary: after complete recovery 80 days after treatment, a second episode with a right geotropic hBBPV, which occurred 8 months later, again showed a mild asymmetry of both VOR gain and cVEMP, which eventually recovered 30 days later. The recovery pattern in the two episodes shows that VEMPs and horizontal VOR gain are not independent, as suggested by the Commentary, but that they are associated: whenever the horizontal VOR was even mildly affected, this also had an effect on the VEMPs.

What “remains to be explained” in future work is the observation that the association of horizontal VOR with VEMPs does not seem to be a metrical one, i.e., the immediate partial recovery of VOR gain after treatment from 0.29 to 0.81 is not reflected in a comparable VEMP recovery. This might be due to a number of mechanisms, e.g., a threshold in semicircular canal biomechanics, central compensation, or frequency-dependent differences in response to 500 Hz ACS versus head impulses with a frequency content of 5 Hz. In addition, the two episodes are different in that the first event was characterized by an unusual horizontal canal plug and the second event by a more common hBPPV, which might be a reason for the dissociation of oVEMP and cVEMP between the two episodes.

A Clinical Capsule Report like ours (1) cannot provide conclusive explanations for all these observations. The observed association and its alternative explanations, however, demonstrate that the rigid assumption of an otolithic origin of VEMP responses as the only possible interpretation for the arguable independence of VOR gain and VEMP recovery does not justify a falsification of our conclusion. The controversy ensuing in both the Commentary by … and in this reply shows how important it is for the clinical interpretation of ACS-induced VEMPs to clarify these questions in future work.

The Commentary by … continues to cite three publications by Manzari et al. (3-5) in support of the view that a dissociation of VOR gains and BCV-induced VEMPs demonstrates the different vestibular origins of the two outcomes, with VOR gains reflecting canal and VEMPs reflecting otolith function.

The first difficulty that arises from comparing our VEMP results with those of Manzari et al. (3-5) is the difference between stimulation methods: We used ACS (1) and the latter used BCV.

The Commentary further claims that “all 59 patients in Manzari et al. (3) had normal horizontal canal function during both vHIT and caloric testing”. However, Manzari et al (3) did not report that ALL 59 patients were examined with vHIT, but only that “MANY patients were also tested by video recording of horizontal head impulses”, without specifying how many patients were tested by vHIT and what their VOR gains were. Apparently, only the presence of a corrective saccade was assessed by vHIT and not the gain. From the few vHIT details reported by Manzari et al. (3) it is difficult to compare their results with our quantitative vHIT analysis of VOR gain (1). Apart from VOR gain, another missing detail is head impulse velocity. In FIG. 1 of our case report (1), VEMP asymmetry on Day 3 was associated with a mild VOR gain asymmetry of 15%, which was unmasked only at head velocities between 150 °/s and 300 °/s. Whether the head impulses used by Manzari et al. (3,4) were too slow to unmask such a gain asymmetry is anyone's guess.

In the case report on a 4-year-old boy by Manzari et al. (5) head impulse velocity was indeed too slow to unmask even a more pronounced asymmetry. Head velocity on the affected right side reached its peak of about 80 °/s at about 90 ms after movement onset (Fig. 1). This corresponds to an estimated acceleration of roughly 1,400 °/s^2, which is well below the accelerations typically used in previous studies on head impulse testing. For example, Aw et al. (1996) (6) used accelerations of 3,000 to 4,000 °/s^2 and Schmid-Priscoveanu et al. (2001) (7) reported 10,000 °/s^2, with peak velocities of 250 °/s. In contrast, Manzari et al. (5) stimulated with less than a third of this peak velocity, although it is well known that only “rapid, … unlike slow” head movements demonstrate the VOR gain asymmetry “that is expressed by Ewald's 2nd Law”(Halmagyi et al. 1990) (8).

Since this law is fundamental to head impulse testing, a possible consequence of ignoring it is a false negative vHIT outcome, as recently demonstrated by Machner et al (9) in a patient with unstable gait and oscillopsia after left-side mastoidectomy for cholesteatoma. vHIT outcome in this patient was indeed negative as long as peak head velocities remained below 200 °/s. In accordance with Ewald's 2nd Law, gain asymmetry was only unmasked with head impulse velocities of 300 °/s. Therefore, the dissociation between VEMP and vHIT responses in Manzari et al. (3,5) might simply be due to false negative vHIT outcomes.

In the case report by Manzari et al. (4) the vHIT results in Fig. 2 A do not support the conclusion in the Commentary by … that the patient had normal horizontal canal function. On the contrary: The gain on the affected right side (mean=1.14, SD=0.08, N=24) was significantly smaller ($p=7*10^{-9}$, one-tailed t-test) than the gain on the left side (mean=1.35,

SD=0.08, N=12). Both gains were above the normative range of 0.78 to 1.1 (8). The gain asymmetry of 8.4% was beyond the range of normal values of <5.6% (7).

Velocity Saturation by a Canal Plug

The most puzzling remark in the Commentary by … concerns the "final error" that we (1) are said to have made by not citing Manzari et al. (10) as the first to "describe" the velocity saturation. This comment is puzzling because in the whole text there is indeed no description of any velocity saturation. The authors of the Commentary point to a velocity saturation in Figure 1, which, however, is characterized by the occurrence of many saccades, by slow phase eye velocities that are difficult to distinguish from saccades and "bump artefacts" (11), by a considerable noise content, by a low image resolution, and by image compression artifacts. A velocity saturation is therefore difficult to detect. Only on the basis of this figure and without the raw data it is impossible to assess the claim in the Commentary that this figure shows a velocity saturation. If there was a velocity saturation, it went unnoticed.

Interestingly, a similar velocity saturation also went unnoticed in a surgical canal plugging (See figure S1 in MacDougall et al., 2013 [11]), which clearly supports our conclusion that this particular velocity profile might be a specific sign of a canal plug. This profile was documented both with search coil and with vHIT. The search coil recording, however, showed a clearer image of this saturation than did the vHIT recording. The difference might be due to the recognized "bump artifact"(11) present in the vHIT device that the authors used.

Could the contralateral inhibitory saturation be responsible for the saturation in the eye velocity profile? Evidence from vHIT testing in unilateral vestibular loss after schwannoma surgery contradicts this, as there is clearly no velocity saturation (see Figure 4 in MacDougall et al (11)). The reason for this probably relies on the fact that each contralateral firing cell has its own firing discharge and velocity-firing characteristic. Therefore each would hit the firing rate saturation boundary at 0 Hz at another velocity. vHIT velocity saturation in vestibular neuritis seems unlikely. Instead, for us (1) the most plausible explanation for the saturation of the eye velocity response (and the nystagmus pattern) observed in the reported BPPV patient was the presence of an otoconial canal plug, since such a plug would cause a negative cupular pressure and block endolymph flow, thus modifying the cupular-endolymph biomechanical dynamics.

VEMP Physiology

Curthoys' group was the first to specifically examine the neural basis of VEMP testing by studying the responses of vestibular afferents to clinical VEMP stimuli. They reported that sound primarily activated the saccule (12-14) and utricle (15) (Curthoys et al., 2012), but not the canals, even at intensities of 80 or 90 dB SL re ABR threshold. These seminal works have been widely cited to support the current saccular theory of cervical VEMP. While the simplicity of the saccular theory has played an important role in the rapid development of the field, it has been challenged by accumulating evidence that shows sound activation of the semicircular canals (for literature review, see Zhu et al., 2011 [2]). To address the neural basis of sound activation of the vestibular system, which is essential for interpreting clinical VEMP testing results, Zhu and Zhou at the University of Mississippi Medical Center have conducted a series of studies over the past decade to further characterize the responses of the vestibular system to clinical VEMP stimuli in monkeys (16-19) and rats (2, 20-23). Their efforts are motivated by three aims. The first aim is to develop a quantitative measurement of sound sensitivity of an individual vestibular neuron. This is achieved by computing the cumulative probability of evoking a spike (CPE) that measures how a transient stimulus (e.g., a brief click) induces a change of firing probability of a neuron (24). Instead of simply

classifying an afferent as sound sensitive or non-sound sensitive, the CPE analysis provides a quantitative assessment of sound sensitivity of a vestibular neuron. The second aim is to employ the CPE approach to record a large number of vestibular afferents from all the five vestibular end organs to test the saccular theory of VEMPs. The third aim is to seek sound parameters that can selectively activate certain vestibular end organs, which will serve as the neural basis of discriminative VEMP testing protocols and interpretation guidelines.

Zhu et al., cited by Luis et al. (1) as reference 15 and in the Commentary (2) as reference 2, surveyed the sound sensitivity of over 900 vestibular afferents in anesthetized rats. In addition to activating 81% of irregular otolith afferents, acoustic clicks [80dB SL re ABR threshold (∼130dB pSPL)] activate a substantial number of irregular anterior canal afferents (AC, 59%) and horizontal canal afferents (HC, 47%). Among them, ∼ 50% of sound sensitive AC afferents and ∼20% of sound-sensitive HC afferents are high sound-sensitive afferents (i.e., CPE>0.5; Figures 2 and 3 in Zhu et al., [2]), which are considered to contribute to generating VEMPs. It should be noted that the canal afferents with lower CPE values may also contribute to VEMPs because summation of synchronous activation of a population of sound-sensitive afferents may result in measurable VEMP responses.

In addition to the neurophysiological evidence of sound activation of the canals, a recent intraaxonal recording/labeling study shows that click sensitive afferents innervate the HC and AC cristae as well as the saccular and utricular maculae (22), therefore, providing direct anatomical evidence for sound activation of both the canals and otoliths. Since motoneurons of the sternocleidomastoid muscles (SCM) receive inputs from both the canals and the otoliths (for reviews, Wilson and Schor, 1999 [25]; Uchino et al, 2005 [26]), these new data suggest that the contribution of canal afferents to VEMPs should not be ruled out in clinical VEMP testing. However, given the distinct physical and geometrical properties of the otoliths and the canals, it is possible to achieve selective activation of a set of vestibular end organs by employing appropriate sound parameters (20,21,27-30) (Lewis et al., 2010; Donnellan et al., 2010, Wei et al., 2012; Ashford et al., 2013; Zhu et al., 2010, 2011). Their ongoing experiments have this aim.

The Commentary also mentioned an issue related to identifying the end organ innervated by an otolith afferent. In intact animals, otolith afferents can be reliably identified by their responses to static head tilts, because canal afferents do not respond to changes in head orientation with respect to gravity. In animals that undergo surgical procedures for vestibular nerve recording, however, Goldberg and Fernandez (1975) (31) showed that the vertical canal afferents are sensitive to static head tilts because removal of the brain tissue overlying the vestibular nerves and ganglion exposes the bony labyrinth to room temperature. This results in a thermal gradient across the labyrinths, which makes the vertical canals sensitive to gravitational changes. To avoid this ambiguity, it is important to employ turntables that provide adequate rotational stimulation to the vertical canals.

Conclusion

In summary, we addressed the arguments of the Commentary by … in the following points:

1. We have demonstrated that a technical failure can be excluded as a possible explanation for the simultaneous absence and recovery of both cVEMP and oVEMP. Very basic technical aspects, such as the possible presence of wax in the ear canal, are not the most plausible causes for our findings;
2. vHIT and VEMP responses did not return independently but were associated. Whenever the horizontal VOR was even mildly affected, this also had an effect on VEMPs;

3. To the best of our knowledge vHIT was used for the first time to document a high-frequency VOR hypofunction during BPPV. Moreover, it documented an eye velocity saturation profile as was later demonstrated in a surgical canal plug (11);
4. Our case proved that a patient with BPPV may present with spontaneous nystagmus. BPPV must be ruled out in acute vestibular syndrome patients. Not only the direction but also the intensity of the nystagmus position dependency should be tested in every patient with spontaneous nystagmus, just as the vHIT velocity profile;
5. As there is solid and growing evidence of sound canal activation, canal contributions to VEMPs should not be ruled out before the neurophysiological basis of sound activation of the vestibular system is understood.

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Abbreviations

cVEMPs	cervical vestibular-evoked myogenic potentials
oVEMPs	ocular vestibular-evoked myogenic potentials
VOR	vestibulo-ocular reflex
ACS	air-conducted sound
BCV	bone-conducted vibration
hBPPV	horizontal benign paroxysmal positioning vertigo
vHIT	video head impulse test