
Ian S. Curthoys and Leonardo Manzari

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Ian S. Curthoys, PhD and Leonardo Manzari, MD

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Abstract

Welgampola and Carey have missed evidence that shows how utricular and saccular function can be differentiated, and here the authors note that evidence and report a new result that further substantiates the differentiation.

Keywords

vestibular, utricular, saccular, VEMP, ocular vestibular-evoked myogenic potential

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The recent commentary by Welgampola and Carey1 has missed evidence that has already been published. There is a wealth of evidence that the cervical vestibular-evoked myogenic potential (cVEMPs) to bone-conducted vibration (BCV) and air-conducted sound (ACS) is due to the ipsilateral saccular macula, afferents from which course predominantly in the inferior vestibular nerve (see Curthoys2 for a review of the anatomy, physiology, and clinical evidence and predictions of patient responses). Recently, we tested the origin of a new VEMP: the n10 component of the ocular vestibular-evoked myogenic potential (oVEMP), which is a crossed otolith-ocular response, by studying patients with deficits of the superior vestibular nerve (SVN).3 Is this new oVEMP n10 also due to saccular afferents that course mainly in the inferior vestibular nerve, or does it originate from afferents from the utricular macula that course exclusively in the SVN?4

To answer that question, we enrolled patients with superior vestibular neuritis who had evidence of intact saccular and inferior vestibular nerve function as shown by having normal, symmetrical cVEMPs to both ACS and BCV stimulation.5,29 But in these patients, there was evidence that the superior division of the vestibular nerve was dysfunctional because the patients had reduced or absent caloric nystagmus on the affected side and usually had a head impulse sign for ipsilesional head rotations. Using the same BCV stimulus on the same patients, the question we asked was, Are cVEMPs and oVEMPs equally affected? The stunningly clear answer was no; in these SVN patients, there is a very clear dissociation: the cVEMP p13 to 500-Hz BCV was of normal amplitude, but the oVEMP n10 to 500-Hz BCV was reduced or absent.3 It follows that the sense organ responsible for the cVEMP cannot be the sense organ responsible for the oVEMP because one response was normal whereas the other response was not. It must be the case that the oVEMP and cVEMP have different causes, that is, they are differentiated. That is the logical interpretation of the empirical result, and it does not need any additional evidence for interpretation. It is exactly in accord with a prediction made in Curthoys (figure 9c).2 This result3 was obtained on one of the largest patient cohorts in the VEMP literature with 133 SVN patients and 58 healthy subjects. Since all utricular afferents course in the superior vestibular nerve4 and there is strong evidence that BCV activates utricular afferents,5 we concluded that the oVEMP n10 is due to utricular function.

We ensured that patients had adequate tension in their neck muscles using a procedure that has been in use and accepted since the very first articles on the cVEMP: having the patients lift their head off the pillow as Welgampola et al6 had done. We agree that it would be advisable for future studies to use average rectified electromyography recordings, but there is no good reason to exclude data carefully obtained with a criterion that has been acceptable to now. Such a retrospective application of this new criterion would exclude most of the published VEMP literature.

1Vestibular Research Laboratory, School of Psychology, the University of Sydney, NSW, Australia
2Department of Experimental Medicine and Pathology, “La Sapienza” University–Rome, Italy
3MSA Academy ENT Center, Cassino, Italy

Corresponding Author: Ian S. Curthoys, PhD, Vestibular Research Laboratory, School of Psychology, A18, University of Sydney, Sydney, NSW, Australia
Email: ianc@psych.usyd.edu.au

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Wolgampola and Carey address the relationship of ACS to BCV. But that relationship is not relevant to the Manzari et al study since in that study the measures of oVEMPs and cVEMPs were based on just BCV alone. We showed that in response to BCV alone, cVEMPs were symmetrical whereas oVEMP n10s were not. The relative thresholds for ACS and BCV are just not an issue since we did not compare responses to ACS and BCV.

They state, “Although it is assumed that saccular afferents must also be sensitive to BCV, this assumption remains untested.” In fact, there is evidence in support of that. First, Young et al found that BCV activated saccular afferents. Second, Fernandez and Goldberg showed that pitch-sensitive otolithic afferents come predominantly from the saccular macula, and in the Curthoys et al study, there were 24 afferents that responded to static pitch, and 20 of the 24 were activated by BCV.

Our conclusion that utricular and saccular function can be differentiated by measuring the different responses to the same stimulus is further strengthened by new evidence we have recently published in which, in response to ACS or BCV, there was unilateral reduction of cVEMPs but the oVEMP n10 was not affected. This new result is precisely the converse of the result in Manzari et al in that saccular function was unilaterally reduced but the contralateral oVEMP n10 to either ACS or BCV was unaffected. Again, the most probable explanation is that oVEMP n10 to either ACS or BCV depends on utricular function, and this result further confirms the Curthoys hypothesis (figure 9d).

Of course, we agree with Wolgampola and Carey when they state that “clinicians must also be cautious and realize that while vestibular neuritis may, more often than not, be ‘superior’ vestibular neuritis, there will be cases with inferior nerve involvement, either exclusively or in addition to superior nerve involvement.” But the diagnostic accuracy of oVEMPs does not fall when it is applied to acute vestibular neuritis and inferior vestibular neuritis, as Wolgampola and Carey claim. Quite the contrary: oVEMPs and cVEMPs help to identify both conditions. In acute total vestibular neuritis with both vestibular nerve components involved, there is a unique response profile in which the oVEMP n10 beneath the contrallesional eye is absent or markedly reduced, and in addition, the ipsilateral p13 of the cVEMP is markedly reduced. Conversely, when the inferior vestibular nerve alone is damaged, oVEMPs n10s to both BCV and ACS are preserved beneath the contrallesional eye, while cVEMPs over the ipsilateral SCM to both ACS and BCV are absent, as we have reported.

We have shown that there is ample existing evidence showing that oVEMPs and cVEMPs differentiate utricular and saccular function, and that evidence continues to grow.

Author Contributions
Ian S. Curthoys, corresponding author, interpretation of the data, writing most of the article, final approval of the submitted version; Leonardo Manzari, design of the study, critical revision of the article, final approval of the submitted version.

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