Letters to the Editor

On "The Influence of Otolith Dysfunction..." Murray et al. *Phys Ther.* 2007;87:143–152.

Murray and colleagues[1] are to be commended for their attempt to identify the role of the otolith organs in the clinical presentation of patients with unilateral vestibular disorders. With the advent of the subjective visual horizontal (SVH), subjective visual vertical (SVV), and vestibular evoked myogenic potential (VEMP) tests, it has become feasible to assess otolith function as a component of the vestibular function test battery. How the otolith organs influence symptoms, clinical signs, and recovery is not known and is a valid question.

Murray and colleagues attempted to address this question by assessing both physical performance measures and self-report measures of symptom intensity and the impact of those symptoms in patients with either combined semicircular canal and otolith disorders or semicircular canal–only disorders. As the authors noted, this study was predicated on the ability to correctly classify these 2 groups of patients, and this is where the study suffers.

First, with the exception of benign paroxysmal positional vertigo (BPPV) and surgical canal plugging procedures, it is not clear that it is possible to have vestibular pathology that does not involve *both* semicircular canals and otolith organs. The peripheral vestibular apparatus is innervated by the superior and inferior divisions of the vestibular nerve. The superior division innervates the cristae of the horizontal and anterior canals, the utricular macula, and the anterosuperior portion of the saccular macula, whereas the inferior division innervates the posterior canal crista and the majority of the saccular macula.[2] Likewise, the vascular supply to the peripheral vestibular system is provided by 2 arteries. The anterior vestibular artery perfuses the ampullae of the anterior and horizontal semicircular canals, the utricle, and a portion of the saccule. The posterior vestibular artery perfuses the posterior semicircular canal ampulla, and the majority of the sacculus.[3]

Based on the differential blood supply and innervation patterns, it is possible to have a vestibular disorder that involves only a portion of the peripheral vestibular apparatus. However, these disorders will have combined involvement of both semicircular canal and otolith structures. For example, studies have demonstrated that there is often sparing of posterior canal and saccule function in cases of vestibular neuronitis, which leads to the common finding of posterior canal BPPV ipsilateral to the unilateral vestibular hypofunction.[4–7]

For the sake of this discussion, let us assume that it is possible to have a deficit in vestibular function that affects only the semicircular canals. This, then, raises the second concern with the study: the sensitivity of the tests. As the authors noted, the sensitivity of the 2 tests used to identify otolith involvement is not good (43% for SVH, and 59% for VEMP testing). Consequently, one would expect that roughly half of the subjects classified as canal-only involvement were actually misidentified (false negatives) and had actual otolith involvement. This misclassification would blur any clinical distinctions that may have occurred between the 2 groups of patients.

As the authors noted in the discussion, one cannot predict the degree of disability secondary to a vestibular deficit based on electronystagmography (ENG)/caloric tests. One of the reasons for this is the fact that the ENG/caloric test measures static bias (or lack of static compensation) and the integrity of the peripheral vestibular system (horizontal semicircular canal). The ENG/caloric test does not measure the central, dynamic compensation process. Similar to the ENG/caloric test battery, SVH is a measure of static bias, and the VEMP test measures the integrity of the peripheral vestibular system (saccule). These tests do not assess the central, dynamic compensation process. Just as spontaneous nystagmus resolves in cases of unilateral vestibular loss, static measures of perceived orientation—in this case, SVV—have been shown to improve with time, even though the peripheral vestibular deficit persists.[8]

The clinical items assessed in this study, on the other hand, are measures of function and essentially reflect the status of the central, dynamic compensatory process. Because the tests (SVH and VEMP) used in this study do not assess the central compensation processes and the clinical symptoms are essentially a manifestation of the central compensation processes, the lack of a difference between the 2 groups is not unexpected (assuming there was a pathophysiologic mechanism that would result in a canal-only lesion and that the SVH and VEMP tests had adequate sensitivity).

In summary, the authors are to be commended for their detailed assessment of the range of symptoms and functional deficits seen in this patient population. Given the pathophysiology of the peripheral vestibular system, the lack of sensitivity of the tests, and the nature of the tests, one cannot reach any conclusions about the influence of

otolith dysfunction on the clinical presentation of patients with unilateral vestibular deficits based on the data presented in this study.

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Author Response

We thank Dr Clendaniel for his comments.

First, it is acknowledged that, because of the differential blood supply and innervation patterns of the peripheral vestibular apparatus, disorders in this area are likely to have combined involvement of both semicircular canal and otolith structures. However, this view may be somewhat simplistic, as it remains unknown whether the otolith organs and the semicircular canals are equally sensitive to vascular or neurally based inflammatory disorders.

Second, it is also acknowledged that the sensitivity with which caloric, vestibular evoked myogenic potential (VEMP), and static bias testing were able to identify vestibular dysfunction in the study participants was paramount in the group allocation process. The otolith function tests, in particular, have been reported to have a sensitivity in the range of 40% to 60%, and, for this reason, otolith dysfunction may not have been correctly identified across the study sample. It is important to recognize, however, that until recently otolith function could be measured only in research laboratories with elaborate and expensive equipment designed to measure horizontal, vertical, and torsional eye movements. The recent development of simple clinical tests, such as VEMP and static bias testing, has been an important step in the assessment of vestibular function. This study was the first of its kind to use these tools to advance our knowledge regarding the clinical presentation of individuals with peripheral vestibular dysfunction. As other, more sensitive, tests of otolith function are developed, this research can be progressed.

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