Objective Verification of Full Recovery of Dynamic Vestibular Function After Superior Vestibular Neuritis

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INTRODUCTION

The following reports the value of new objective measures of the dynamic function of the semicircular canals and otoliths of a patient during acute unilateral superior vestibular neuritis (SVN), compared to the same objective measures 3 months later. This comparison showed that there was full recovery from the SVN. This report shows that it is possible to obtain fast, simple, accurate, objective evidence of the status of the major vestibular sense organs using published clinical tests that are firmly based on anatomical and physiological evidence. These tests can be carried out even in patients in the midst of a major vertigo attack without causing the patient distress, in contrast to a caloric test. The test results allow the clinician to distinguish between a vertigo attack due to vestibular neuritis as opposed to an attack due to Ménière's disease (MD), because the published evidence shows that the test results of patients in these two conditions have very different response profiles.

As we explain below, to our knowledge this is the first case of objectively verified recovery of dynamic utricular function reported in the literature.

If a patient is seen during the acute stage of a vestibular disease, a clinician can identify the classical signs and symptoms, which are dependent on the absent function of the affected vestibular sense organ. Such classical static clinical signs are: 1) spontaneous nystagmus with the quick phase of the horizontal component directed away from the affected ear, indicating reduced static horizontal semicircular canal function of the affected ear; 2) ocular torsion with both eyes rolled toward the affected ear, indicating unilaterally reduced otolithic function of the affected ear; 3) skew deviation that the ipsilesional eye shows a lower position in the orbit; and 4) postural instability with sensations of falling toward the lesioned side, related to reduced function of the affected ear projecting to ipsilateral vestibulo-spi
cal responses. The usual standard tests of dynamic vestibular function are: 1) the canal paresis measure from Fitzgerald-Hallpike caloric testing; and 2) the head impulse sign—during brief, passive, unpredictable, horizontal head turns toward the affected ear. The patient with reduced unilateral horizontal canal function fails to maintain fixation on an earth-fixed target, and so makes corrective (overt) saccades at the end of the head rotation to regain fixation. During similar rotations toward the healthy ear no overt saccades are evident. The overt saccade is a sign of reduced dynamic horizontal canal function; however, this sign is subjective in that there is no objective measure of the head velocity stimulus or the eye velocity response and therefore no objective measure of vestibulo-ocular reflex (VOR) gain.

More recently, vestibular-evoked myogenic potentials have become widely used. The cervical vestibular-evoked myogenic potential (cVEMP) to air-conducted sound (ACS) or bone-conducted vibration (BCV), stimuli that have been shown to activate otolithic neurons, indicate dynamic human saccular and inferior vestibular nerve function. In response to BCV or intense ACS stimulation, healthy subjects with surface electrodes...
over the tensed sternocleidomastoid (SCM) muscles show a short-latency positive (inhibitory) myogenic potential (p13-n23) at a latency of around 13 ms from stimulus onset, indicating dynamic function of the ipsilateral saccular macula because this is an uncrossed sacculo-colic response. The calorics, the head-impulse sign, and the cVEMP allow identification of whether it is the superior or the inferior vestibular nerve, or both, that are involved in the disease. To further clarify this matter, we can use the anatomical information from de Burlet and the more recent physiological evidence to show how each vestibular sense organ can be

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**Fig. 1.**

![Graphs and diagrams showing vHIT, Gain, Gain, oVEMPs, and cVEMPs for Acute (29 December 2010) and Recovered (21 March 2011).](image)

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independently tested. For the clinician, the other issues are the questions, can this patient fully recover from this disease and over what time course might this happen?

Two new tests of dynamic vestibular function have recently been reported. It has been shown that BCV and ACS activate otolith irregular afferents from the utricular macula. These neural results underpin a new test of dynamic utricular function—the ocular vestibular-evoked myogenic potential (oVEMP) to short tone bursts of BCV and ACS stimulation. The first negative (excitatory) component of this myogenic potential at 10 ms latency and so called n10, recorded by surface electrodes beneath the eyes to ACS or BCV while the subject looks vertically upward, indicates dynamic function of the contralateral utricular macula because this is a crossed utriculo-ocular response.

A new objective measure of horizontal canal dynamic function has been developed and validated, called the video head impulse test (vHIT). The vHIT test provides an objective measurement of the head velocity stimulus and the eye velocity response during the head impulse test, and so allows a measure of VOR gain and VOR gain asymmetry (analogous to canalic vestibular paresis).

The outcome of these developments is that now it is possible to obtain objective measurement of the dynamic function of all vestibular sense organs and understand how these dynamic functions change over time (e.g., does the function fully return after the disease?). Here we report the case of an apparently simple resolution of a clinical problem using these new objective measures to underscore the value of such objective measurements.

### CASE REPORT

A 70-year-old woman was referred for the first time to our tertiary referral neurotological center (MSA ENT Center, Cassino, Italy) on December 29, 2010, with acute vestibular symptoms (rotatory vertigo, nausea, pallor, and vomiting). There were no cochlear symptoms, such as tinnitus, fullness, or acute hearing loss either before, during, or after the onset of the acute vertigo. Therefore, it was presumed during collection of the history that she was not a patient with MD, although patients in the early stages of MD may present with only vestibular symptoms. The same patient was tested again 3 months later when she had recovered.

At the time of the first visit, all symptoms reported by the patient were evaluated with a standardized set of vestibular tests. The patient was submitted to instrumental audiovestibular tests: audiometry, tympanometry with stapedial reflexes, and auditory brainstem response, which were all normal for the patient’s age. However vestibular sense organ testing showed marked losses. First, Fitzgerald-Hallpike caloric testing showed hyporesponsiveness of the left horizontal canal of the affected ear with a canal paresis score of 67%. Bedside procedure in our clinic consists in evaluating eye movements (spontaneous nystagmus, gaze-evoked nystagmus in darkness and in light, and head impulse test), limb coordination Romberg test, and gait observation. Eye movements were evaluated and recorded with three-dimensional (3D) infrared video-oculography. The patient wore a mask with a camera fixed in front of the left eye, and the pattern of horizontal, torsional, and vertical eye movement components were measured in darkness with 3D infrared video-oculography (50 Hz sampling) (Torsio VNG Ulmer; Synapsys, Marseille, France) during spontaneous nystagmus, during head-shaking nystagmus, during the Dix-Hallpike maneuver, and during the head-roll maneuver for positional and positioning nystagmus. At the time of the acute attack, the patient showed spontaneous nystagmus in darkness with horizontal and torsional components; the horizontal component of the quick phase was directed away from the left ear toward the right (healthy) side. Gaze-evoked nystagmus at the time of the attack decreased in intensity when the gaze was directed away from the quick phase (Alexander’s law). There was no evidence of benign paroxysmal positional vertigo or positional nystagmus. In our patient, tandem walking revealed that the patient tended to veer toward the side of the lesion (left).

Dynamic horizontal canal function was tested by the vHIT, which revealed a clearly impaired horizontal VOR when her head was abruptly, unpredictably, or

Fig. 1. Objective measures of vestibular function for the two testing sessions—acute phase (December 29, 2010) and recovery phase (March 21, 2011). (A) Superimposed records of head velocity (red) and the corresponding eye velocity (black) versus time for the tests of horizontal canal dynamic function using video head impulse test (vHIT). The signs of head velocity for leftward impulses and of eye velocity for rightward impulses have been inverted for easier comparison. In a healthy subject the eye velocity trace closely matches head velocity (as shown by reduced peak eye velocity in the panel for leftward impulses), and a large number of corrective saccades, confirming the inadequate left canal function. In the recovery phase, the gain in the left ear has increased to be within normal range. During the acute phase, the gain for impulses to the affected (left) side is around 0.5, well below the normal value, whereas the gain for impulses to the healthy side is around 0.8, within the normal range. During the recovery phase, the average gains have increased to around 0.9 for impulses to the affected side and 1.0 for impulses to the healthy side. (B) Horizontal vestibulo-ocular reflex (VOR) gains versus peak head velocity for the head-impulse data shown in (A). Closed circles indicate leftward impulses, and open circles indicate rightward impulses. The mean and 95% confidence intervals for gains in leftward and rightward impulses are shown at the right of each plot. Normal horizontal VOR gains are about 0.7 to 1.0. During the acute phase, the gain for impulses to the affected (left) side is around 0.5, well below the normal value, whereas the gain for impulses to the healthy side is around 0.8, within the normal range. During the recovery phase, the average gains have increased to around 0.9 for impulses to the affected side and 1.0 for impulses to the healthy side. (C) Tests of utricular function: ocular vestibular-evoked myogenic potential (oVEMP) to 500 Hz, 7 ms (1 ms rise-fall), short-duration tone burst bone-conducted vibration (BCV) at Fz (the skull location of the midline of the forehead at the hairline) in the patient. The upper traces are for the right eye (due to the affected left ear because this is a crossed response), and the lower traces for the left eye (due to the healthy right ear). Two repeats are shown for each condition. The inverted triangles mark the approximate times of the n10 potentials. During the acute phase, the strong asymmetry in the amplitude of the n10 response between left and right sides is evident, showing the absent dynamic utricular function on the left, with the response beneath the contralateral right eye absent. By the recovery phase there is a clear oVEMP n10 beneath the right eye showing the return of dynamic left utricular function so that the n10 amplitudes have become symmetrical, as normal healthy subjects show. Cervical vestibulo-activated myogenic potential (cVEMP) to BCV were recorded over both sternocleidomastoid (SCM) muscles to the same 500 Hz Fz BCV stimulus on the two occasions. The upper traces are for the right SCM muscle, and the lower traces for the left SCM muscle. The small vertical lines mark the p13 and n23 potentials. In contrast to the asymmetric oVEMP data in (C), during the acute phase the p13-n23 amplitudes on both sides are similar, and within the normal range, showing that the left saccular function is normal. There is little change in the p13-n23 amplitudes between the two sessions. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]
passively turned to the left, whereas similar abrupt, unpredictable, passive head turns to the right had normal VOR gain. During the ipsilesional (leftward) passive head turns, the patient also showed a series of overt and covert saccades, symptomatic of inadequate left horizontal canal VOR dynamic function (Fig. 1A). The objective test of saccular function, the oVEMP p13-n23 (using a short tone burst of 500 Hz lasting 7 ms at 4/s for 50 presentations), delivered by a Bruel and Kjaer minishaker 4810 to the midline of the forehead at the hairline (a skull location called Fz), showed normal symmetrical cVEMP responses over tensed SCM muscles (Fig. 1D). However, the objective test of utricular function, the n10 component of the oVEMP, using the same 500 Hz Fz BCV stimulus, showed absent n10 responses beneath the right eye (the contralateral eye), with n10 responses to that stimulus beneath the left eye being of normal amplitude, resulting in an oVEMP n10 asymmetry ratio of 100%. This result indicates a left utricular dysfunction because the oVEMP n10 is a crossed response. Overall the results show reduced left horizontal canal function (the vHIT asymmetry of VOR gain) and reduced left utricular function (the oVEMP n10 asymmetry), whereas the left saccular function was normal (the cVEMP symmetry), and there was no auditory involvement. As a result of these objective tests the patient was diagnosed as having left SVN.

Because of her medically controlled hypertension the patient declined the methylprednisolone treatment as recommended by Strupp et al., and chose to receive only medication to alleviate the vomiting and nausea. She was instructed to undertake rehabilitation exercises at her home and to restart her regular daily activities including moving around during the day as much as possible. As an additional precaution, she was referred to a tertiary radiology center for a magnetic resonance imaging scan of the posterior cranial fossa using paramagnetic contrast enhancement. Radiologic evaluation revealed normal and symmetrical cranial nerve VIII and normal signals from her midbrain and posterior cranial fossa.

The same patient was re-evaluated on March 21, 2011. At that date, she was well and her vestibular symptoms had improved. She was submitted to the same clinical examinations as before. She noted that cochlear symptoms had not appeared during the interval. Audiometry, tympanometry with stapedial reflexes, and auditory brainstem response were again normal in relation to the patient’s age. Her hearing level remained symmetrical just as had been obtained on the first occasion. The spontaneous nystagmus had disappeared, and the caloric canal paresis (CP) score had improved so that it was a normal value (CP = 6%). The oVEMP n10 to 500 Hz Fz BCV under the contralateral (right) eye reappeared, and the n10 component was symmetric and of normal amplitude beneath the two eyes (asymmetry ratio of 3%) (Fig. 1C). The cVEMP remained symmetrical (Fig. 1D), although the morphology of the records was rather different compared to the test at attack. This difference may be due to differences in the configuration of the electrodes or the muscle tension exerted by the subject on these two very different testing occasions, but in this case it is important to emphasize that symmetry of the p13-n23 complex recorded on both sternocleidomas- tod muscles on both occasions, indicating intact function of the inferior vestibular nerve. vHIT also revealed normal and complete recovery of the VOR gain for rotations to the previously affected left side with no overt or covert saccades (Fig. 1A, B), in sharp contrast to the significantly reduced VOR gain at testing during the acute phase (Fig. 1A, B). It was concluded that the patient’s left superior vestibular neuritis had fully recovered.

DISCUSSION

There are two major reasons why this case is important. First, to our knowledge this is the first case of objectively verified recovery of dynamic utricular function reported in the literature. It clearly shows a fully documented recovery of peripheral vestibular dynamic function of the affected sense organs in only 3 months. Therefore, the vestibular system can fully recover quickly after neuritis. If this patient had been seen first on March 21, 2011, no clinician would have guessed that she had been through a major vestibular catastrophe just a few months before. A clinician might possibly infer it from a close case history, but he or she could not imagine that catastrophic situation of just 3 months before. Even patients who appear to have normal vestibular function at first testing may have had a major vestibular disturbance shortly beforehand. The neuritis was probably confined to the superior vestibular nerve, as it is difficult to imagine that receptor damage could recover so quickly and completely. The attack is unlikely to have been due to a unilateral labyrinthitis, because the saccular function (shown by the cVEMP) was normal, and there was no evidence of auditory involvement.

It is more likely that an inflammation, an edema, had compressed fibers of the superior branch of cranial nerve VIII, and as the inflammation resolved the nerve activity returned, in much the same way as Bell’s palsy may resolve over time. Second, it shows one advantage of these new tests, that of discriminating between an acute attack of vertigo due to vestibular neuritis as opposed to MD. The oVEMP n10 is totally different during an acute MD attack as opposed to an acute crisis of vestibular neuritis. During an acute MD attack, the oVEMP n10 beneath the eye opposite the affected ear is usually enhanced as we have previously published, whereas during an acute crisis of vestibular neuritis the oVEMP n10 beneath the eye opposite the affected ear is reduced or absent. In this present patient, the oVEMP n10 beneath the contralateral eye was absent. In addition, with an MD attack, horizontal canal function as measured by vHIT is either normal or enhanced, whereas in this patient horizontal canal function was reduced. We consider the combination of the absent contralateral oVEMP n10, the reduced horizontal VOR, together with the absence of any hearing difficulties or symptoms confirms the preliminary diagnosis that this patient did indeed have vestibular neuritis and not MD.

CONCLUSION

These results are in agreement with the hypothesis that the horizontal semicircular canal head impulse sign, caloric, and contralateral oVEMP n10 all have the same origin—the superior vestibular nerve. All were absent or reduced on the first occasion, and all recovered after only 3 months without any specific treatment. The cVEMP p13-n23 amplitude was unaffected on both occasions, further consistent with the idea that the saccular fibers course in the inferior vestibular nerve, which was apparently unaffected by the neuritis in this patient.
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