

Dorsolateral Medullary Infarction: A Neurogenic Cause of a Contralateral, Large- Amplitude Vestibular Evoked Myogenic Potential

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Larry Lundy*
David Zapala*
Ketil Olsholt*

Abstract

The vestibular evoked myogenic potential (VEMP) has become a useful tool to assess the saccule and inferior vestibular nerve function. Vestibulopathies involving the saccule or inferior vestibular nerve typically result in VEMP responses that are diminished or absent on the involved side. Abnormally large VEMPs are rare. Large VEMPs have been associated with superior canal dehiscence, Ménière's disease, and labyrinthine fistula. In all of these cases, the abnormally large VEMP can be explained on the basis of labyrinthine hydromechanical changes that result in excessive saccular displacement in response to intense sound. In this report, a case is presented of a 74-year-old male with dorsal lateral medullary infarction (Wallenberg's syndrome) who presented with an enlarged VEMP—a finding that has not been reported to date as a result of a brain stem lesion. Particularly perplexing, the enlarged VEMP was on the contralesional side. A proposed mechanism of contralateral vestibular nuclei disinhibition secondary to the brain stem stroke is discussed.

Key Words: Lateral medullary infarction, vestibular evoked myogenic potentials, Wallenberg's syndrome

Abbreviations: CDP = computerized dynamic posturography; CN XI = cranial nerve XI (spinal accessory nerve); EMG = electromyogram; MRA = magnetic resonance angiography; MVST = medial vestibulospinal tract; VEMP = vestibular evoked myogenic potential; VNG = videonystamography

Sumario

El potencial miogénico vestibular evocado (VEMP) se ha convertido en una herramienta útil para evaluar el sáculo y la función del nervio vestibular inferior. Las vestibulopatías que involucran el sáculo y el nervio vestibular inferior típicamente generan respuestas del VEMP que están disminuidas o ausentes en lado involucrado. Los VEMP anormalmente grandes son

*Mayo Clinic Florida

raros. Los VEMP grandes se han asociado con dehiscencia del canal superior, con enfermedad de Ménière y con fistula del laberinto. En todos estos casos, el VEMP anormalmente grande puede explicarse sobre la base de cambios hidromecánicos del laberinto, que producen un desplazamiento excesivo del sáculo, en respuesta a un estímulo sonoro intenso. En este reporte, se presenta un caso de un hombre de 74 años de edad con un infarto medular dorsolateral (Síndrome de Wallenberg), quien mostró un VEMP grande—un hallazgo que a la fecha no ha sido reportado como resultado de una lesión del tallo cerebral. Sorprendentemente, el VEMP agrandado estaba en el lado contrario a la lesión. Se discute un mecanismo propuesto de desinhibición de los núcleos vestibulares contralaterales, producto de la apoplejía en el tallo cerebral.

Palabras Clave: Infarto medular lateral; potenciales miogénicos vestibulares evocados, Síndrome de Wallenberg

Abreviaturas: CDP = posturografía dinámica computarizada; CN XI = nervio craneal XI (nervio espinal accesorio); EMG = electromiografía; MRA = angiografía por resonancia magnética; MVST = tracto vestibuloespinal medial; VEMP = potencial miogénico vestibular evocado; VNG = videonistagmografía

The vestibular evoked myogenic potential (VEMP) has become a commonly accepted clinical test of vestibular function. The discovery of sound-induced changes in skeletal muscle tone can be attributed to the early reports of Bickford and colleagues (1964), Cody and Bickford (1969), and Townsend and Cody (1971). These researchers established that intense, transient acoustic stimuli provoke changes in myogenic field potentials. Their work implicated the saccule and descending vestibulospinal pathway as underpinning these “sonomotor” responses. However, sound-evoked myogenic potentials were not used clinically until Colebatch, Halmagyi, and Skuse (1994) established a reliable procedure for recording the potentials from the sternocleidomastoid muscle. Although not definitively established, it now seems likely that the VEMP recorded from the ipsilateral sternocleidomastoid muscle involves a pathway with a sensory limb that includes the ipsilateral saccule and inferior vestibular nerve (Kushiro et al, 1999; Colebatch and Rothwell, 2004; Basta et al, 2005). Within the brain stem, the medial vestibular nuclei and the medial vestibulospinal tract (MVST) have been implicated (Baloh

and Honrubia, 2001). At the level of the cervical spine, the descending MVST apparently interacts with the spinal accessory nucleus within the anterior horn cells of the cervical cord (C1 through C6). The final efferent limb includes the spinal component of cranial nerve XI, leading to the sternocleidomastoid muscle (Uchino et al, 1997).

One characteristic of the VEMP that led investigators to suspect a myogenic origin was the observation that the response amplitude increases with increased muscle tone. If the neck muscle from which the potential is recorded is relaxed, no measured response occurs. The VEMP recorded from the sternocleidomastoid muscle is actually an inhibitory potential, reflecting a transient decrease in underlying motor unit firing in response to the stimulus (Kushiro et al, 1999; Halmagyi and Curthoys, 2000; Colebatch and Rothwell, 2004; Welgampola and Colebatch 2005). The latency of the response varies little with stimulus intensity, but the amplitude varies depending on muscle tension and stimuli intensity. Therefore, given adequate muscle tension and stimulus intensity, abnormal VEMPs are either absent, reduced in amplitude, prolonged in latency, or excessively large in amplitude. Absent or low-amplitude

VEMPs are commonly attributed to a conductive hearing loss, saccule or inferior vestibular nerve pathology (vestibular neuronitis, labyrinthitis, acoustic neuroma), or brain stem lesions. A large-amplitude VEMP is a relatively uncommon finding, usually associated with superior semicircular canal dehiscence (Minor, 2005), Tullio phenomenon (Watson et al, 2000), enlarged vestibular aqueduct (Sheykholslami et al, 2004), or Ménière's disease (Young et al, 2002a; Young et al, 2002b). In all of these cases, the very large-amplitude VEMP can be explained on the basis of hydro-mechanical changes within the labyrinth.

This case report, however, involves an excessively large-amplitude VEMP in a patient with a lateral medullary infarct on the contralateral side. A hydromechanical explanation would not be plausible in this case. Rather, a brain stem neurogenic etiology of the large-amplitude VEMP is offered.

CASE REPORT

A 74-year-old male presented to a local emergency department with a complaint of suddenly developing imbalance with a tendency to fall to the right and mild dizziness. A computed tomography (CT) scan of the head and neurologic exam were normal, leading to discharge with a tentative diagnosis of an "inner ear problem." Over the next several days the patient experienced drooping of his right upper eyelid, double vision (vertical diplopia, especially on upward gaze), hoarseness, difficulty swallowing (dysphagia), and loss of ability to detect cold sensation on the left side of the body.

The patient came to Mayo Clinic Florida for a second opinion concerning his symptoms. His physical examination revealed right upper eyelid ptosis, right miosis, torsional lateral nystagmus on rightward gaze, and diminished right corneal reflex. The patient had normal facial nerve function and normal touch sensation on both sides of the face but had diminished perception to cold temperature on the left face. His right true vocal cord was immobile. He had decreased sensation on the entire left side of the body to light touch, pinprick, and cold (more prominent in the left upper extremity). He had slight dysmetria with finger to nose and heel to shin bilaterally and a markedly ataxic, wide-based gait. His left toe was up-going (positive Babinski's sign).

Formal testing included an audiologic examination, vestibular assessment, and magnetic resonance imaging (MRI) scanning. The audiogram, shown in Figure 1, revealed a symmetrically sloping mild to moderate high-frequency sensorineural hearing loss with 92 percent word-recognition scores bilaterally. Left ipsilateral acoustic reflex thresholds were elevated. Otherwise acoustic reflex and reflex decay studies were within normal limits.

The vestibular assessment consisted of videonystamography (VNG), slow harmonic acceleration rotational chair testing, computerized dynamic posturography (CDP), and VEMPs. The ocular motor subsection of the VNG test identified a gaze-evoked nystagmus that beat counterclockwise and to the right on rightward gaze and counterclockwise on leftward gaze. Saccadic testing demonstrated a right ocular lateral pulsion—that is, there was saccadic overshoot to the right in greater than 50 percent of trials, and there was saccadic undershoot to the left in greater than 50 percent of trials. This is shown in Figure 2. While smooth pursuit movements were only mildly saccadic, torsional eye movements were clearly evidenced when the eyes deviated toward the right.

With vision denied, there was a direction-changing oblique positional nystagmus that beat leftward (maximum slow-phase velocity of 12 degrees/sec) and upward (maximum slow-phase velocity of 18 degrees/sec). In some positions, only a horizontal or vertical component could be appreciated. Hyperventilation provoked the vertical component of this nystagmus but not the horizontal component. The nystagmus did suppress with visual fixation.

Caloric responses were essentially symmetrical (18% unilateral weakness, 8% directional preponderance) and strong at 240 degrees/sec total eye speed (95% limits = 37 degrees/sec to 274 degrees/sec). Slow harmonic acceleration demonstrated normal gain, phase, and symmetry. There was a modest failure of fixation suppression bilaterally (overall gain = 0.28, normal limits = 0.20), more so for leftward vestibular-induced eye movements.

CDP motor-control tests were essentially within normal limits, with the exception of a strong tendency to shift center mass to the right. A global (3, 4, 5, and 6) pattern

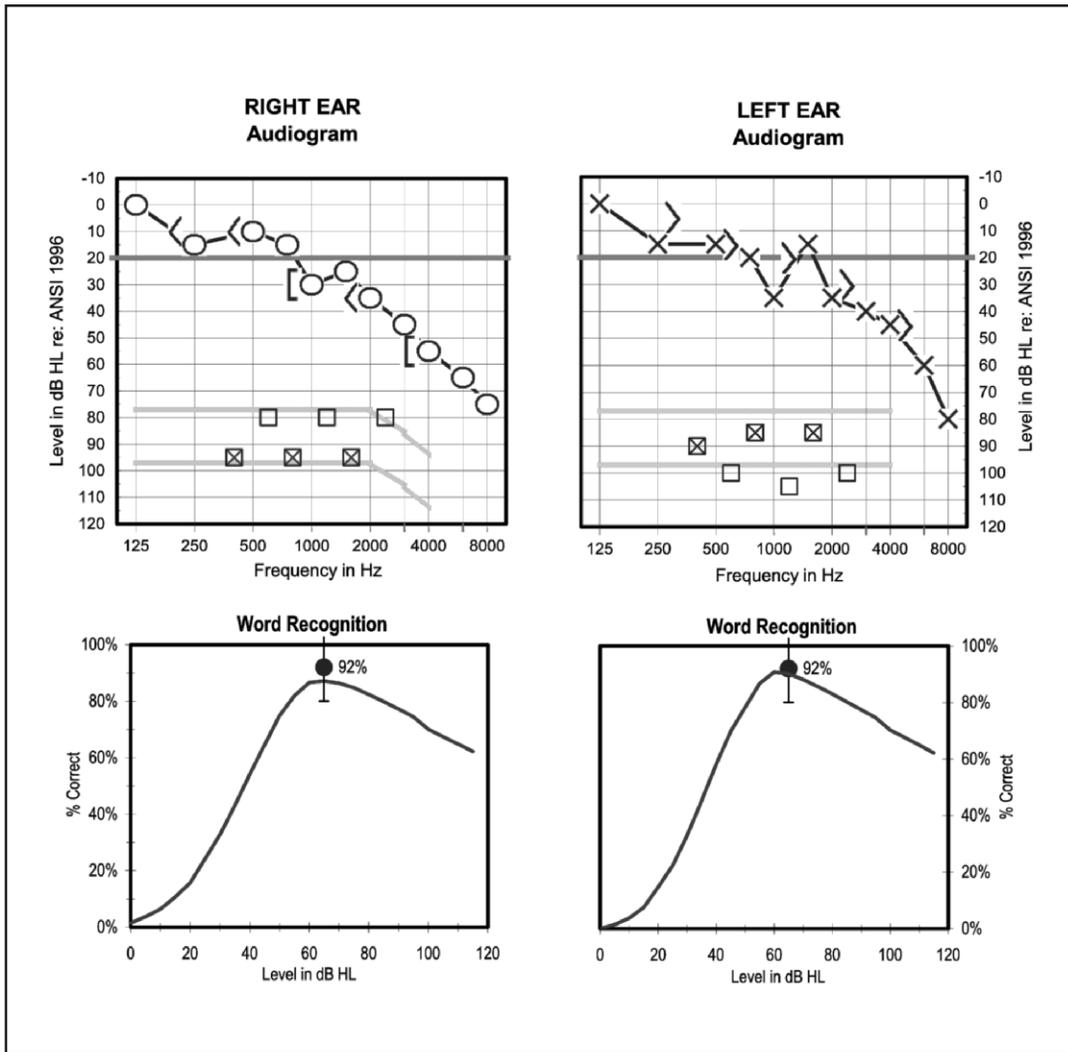


Figure 1. Audiogram showing pure-tone thresholds, word-recognition scores, and acoustic reflex thresholds. Ipsilateral acoustic reflexes are represented by the open squares on the audiogram, and contralateral acoustic reflexes are represented by the crossed squares. Expected normal limits for the acoustic reflexes (95% confidence limits) are shown as the light gray lines on the audiogram. The light gray line on the word-recognition performance intensity plot represents expected performance using a speech transmission index model.

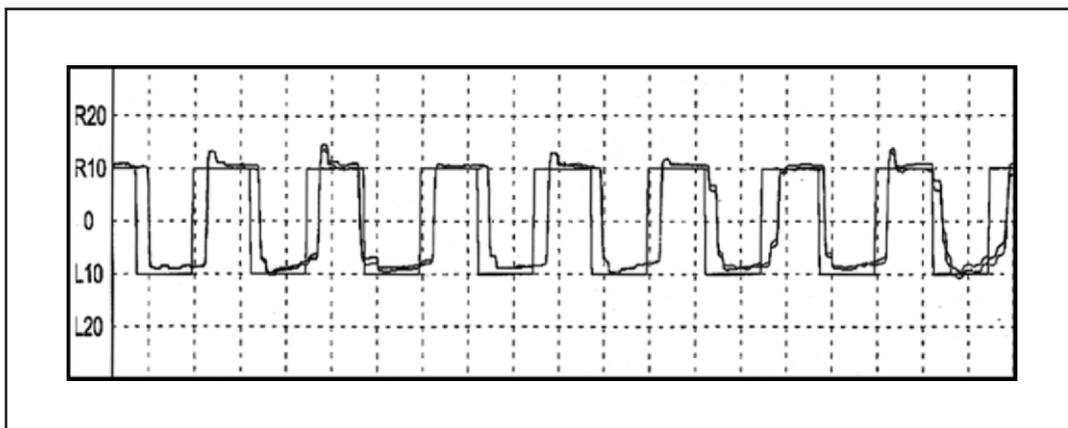


Figure 2. Ocular lateral pulsion on saccadic testing. The tracings show overshoots to the right (dark, solid arrows) and undershoots to the left (light, open arrows).

was evidenced, with falls on conditions 5 and 6. Again, there was a strong tendency to shift the center mass to the right.

VEMPs were obtained using methods described in earlier articles (Zapala and Brey, 2004; Zapala, 2007). Acquisition parameters are summarized in Figure 3. Briefly, stimuli consisted of 500 Hz tone pips (two-cycle rise and fall, no plateau—Blackman envelope), calibrated in dB nHL and presented at a rate of 5.1 bursts/sec through an ER-3A insert earphone. The noninverting lead consisted of a large 2" × 3" disposable surface electrode placed on the belly of the ipsilateral sternocleidomastoid muscle, located on the upper half of the muscle length from sternum to mastoid. An inverting lead and ground

electrode were placed on the forehead. The neck and forehead leads were amplified × 10,000, with bandpass filtered from 1 to 250 Hz, digitized (2560 Hz), and signal averaged (Nicolet Spirit evoked potential unit). Epochs consisted of a 20 msec prestimulus interval and an 80 msec poststimulus interval of electromyogram (EMG) activity.

To obtain a VEMP in our clinic, the patient is placed in a semirecumbent position on a custom examination table with the head and upper torso elevated 30 degrees (caloric position). During each average, the subject is instructed to rotate his or her head 45 degrees to the side (so that the test ear is up) and to lift his or her head an inch off the table. In this pose, the sternocleidomastoid

VEMP Acquisition Method	
Stimulus	
Frequency:	500 Hz tone burst (2-0-2 rise-plateau-fall)
Rate:	5.1 or 5.7 bursts/sec
Presentation:	Monaural through ER3-A inserts
Recording	
Montage:	Cz (+) to upper 1/3 of SCM m. (-); ground = Cz
Filter:	5-250 Hz bandpass
Amplification:	× 5000
Averaging	
Window:	100 msec
Prestimulus:	25 msec
Epochs/Average	60-120
Averaging Stop Rule:	120 epochs or when VEMP amplitude is three times larger than any wave in the prestimulus interval
Superaverages	Measurements taken from superaverage (3-6 averages summed together)
Measurements	
P1 Latency	Normal range: 15-20 msec
N1 Latency:	Normal range: 20-30 msec
P1-N1 Amplitude	Normal range in eighth decade = 60-250 microVolts
Threshold:	Normal range: 80 dB ±10 dB

Figure 3. Parameters for acquisition of the vestibular evoked myogenic potential (VEMP).

muscle is the primary flexor used to achieve this head position. The amplitude of the EMG is not systematically monitored during signal averaging. Rather, it is assumed that essentially equal muscle contractions are achieved across sides because the weight of the head does not change. Between 40 and 120 epochs (median = 60) are typically collected for each average with the subject so positioned. Between three and five such averages are added together for analysis purposes. For each average, recording stops when the VEMP is at least three times larger than any competing wavelet in the prestimulus period or when 120 epochs are collected. This subaveraging technique is used to minimize muscle fatigue. In this case, only two subaverages were collected for each trial at 100 dB nHL because the responses were so large and repeatable (note the relatively flat prestimulus intervals in Figure 4).

In an internal study, we have looked at the effect of age, gender, and side on VEMP responses in 458 patients who were found to be otologically normal. From these data, we have established probability density distributions for each of the reported VEMP parameters in this study. Following each reported parameter, the probability of observing the reported value is given as a p value.

In this case, the patient's VEMP study was remarkable in several respects (see Figure 4). First, a normal VEMP response was demonstrated on the ipsilesional right side. Specifically, N1 and P1 latencies were normal, VEMP amplitude was well within age-adjusted normal limits (183 μV , $p = .64$), and the VEMP threshold was a normal 90 dB nHL ($p = .75$). In contrast, on the contralateral left side, the age-adjusted VEMP amplitude was abnormally large (374 μV , $p = .025$), with a very low age-adjusted threshold

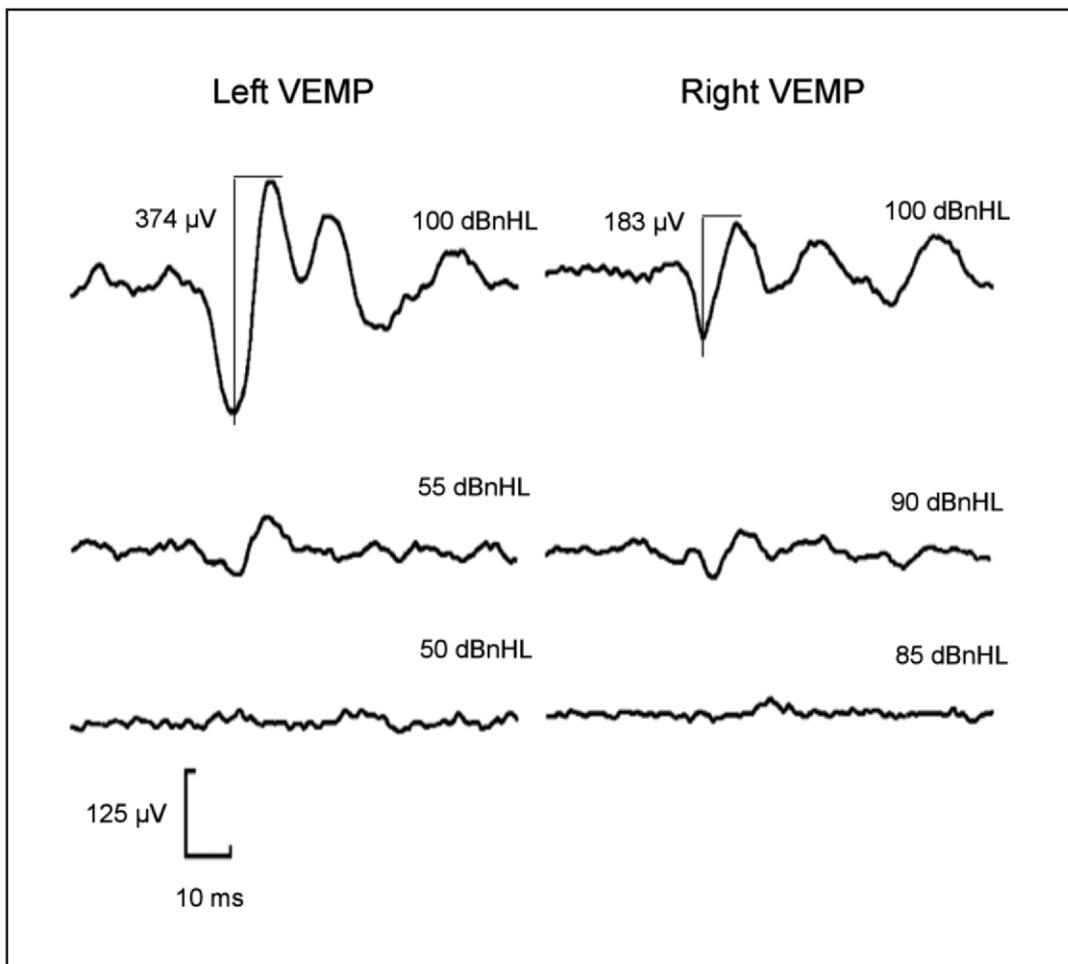


Figure 4. Vestibular evoked myogenic potential (VEMP) waveforms for right and left ear stimulation. All responses demonstrated latencies within normal limits. The threshold for the right VEMP was 90 dB nHL. The threshold for the left VEMP was 55 dB nHL. VEMP amplitude on the left (contralateral response) was 374 μV . The asymmetry ratio was 34 percent.

of 55 dB nHL ($p = .001$). Therefore, the asymmetry ratio is 34 percent.

The patient underwent MRI and magnetic resonance angiography (MRA) of the brain with and without contrast enhancement. The results are shown in Figure 5 and reveal a small right dorsolateral medullary infarction and a 1.3 cm dissecting pseudoaneurysm of the distal right vertebral artery. The final clinical diagnosis was dorsolateral medullary infarction, also known as Wallenberg's syndrome.

DISCUSSION

German neurologist and internist Adolf Wallenberg first described the clinical manifestations (Wallenberg, 1895) and autopsy findings (Wallenberg, 1901) of dorsolateral medullary infarction. The clinical signs and symptoms are the result of occlusion of the vertebral artery or the posterior inferior cerebellar artery or one of its branches. The severity of the signs and symptoms depends on the extent of the area of infarction and ischemia, at times resulting in death.

The neuroanatomical bases for the signs and symptoms of dorsolateral medullary infarction have been well established and correlated with MRI scans (Vuilleumier et al, 1995; Kim, 2003). In the patient reported here, the key areas of involvement were the right inferior cerebellar peduncle (ataxia), right spinothalamic tract (loss of pinprick and cold on contralateral side), right nucleus ambiguus (hoarseness due to right true vocal cord paralysis and dysphagia),

and right reticulospinal pathways (right upper eyelid ptosis and right miosis). The nystagmus can be explained on the basis of the ipsilateral vestibular nuclei, involvement of vestibulo-cerebellar pathways in the restiform body, or possibly posterior inferior cerebellar involvement (Baloh and Honrubia, 2001). Rambold and Helmchen (2005) have noted that in many cases, semicircular canal imbalance may account for spontaneous nystagmus in patients with Wallenberg's syndrome. However, in this case the anterior inferior cerebellar artery and labyrinthine artery were spared, saving horizontal semicircular canal tone and labyrinthine function overall. A central vestibular nuclei or vestibular cerebellum focus is supported by the vertical nystagmus that enhanced with hyperventilation and indirectly by the hyperactive caloric responses (suggesting loss of cerebellar modulation of vestibular output).

This patient's ataxia and wide-based gait, in view of the site of the lesion, were likely due to involvement of the ipsilateral inferior cerebellar peduncle (restiform body). He demonstrated a lateral pulsion phenomenon, where he felt pulled to the right, and this was evidenced in his center of mass measurements on CDP. He also demonstrated the ocular equivalent—ocular lateral pulsion—where ipsilesional saccades overshoot their target and contralesional saccades undershot the target. All of these phenomena are in keeping with a lateral medullary focus.

The unusual and unexpected finding in this patient was an abnormal, large-amplitude VEMP on the contralateral side from the lesion. The test was repeated to ensure there was no simple explanation such as operator error in labeling the sides. With an established set of normative data over three years and over 450 normal VEMP studies, it was clear that the amplitude of the response was well outside the normal range.

Our evidence for this assertion is shown in Figure 6. In this figure, the median and 95 percent amplitude limits for N1–P1 are shown by age (in decades). For his age group (70–79), the median amplitude is 109 μV , and the 95 percent limits are 36–326 μV . His VEMP amplitude on the ipsilesional side was 183 μV (dashed line). This amplitude is within the normal range, somewhat above the geometric mean. On

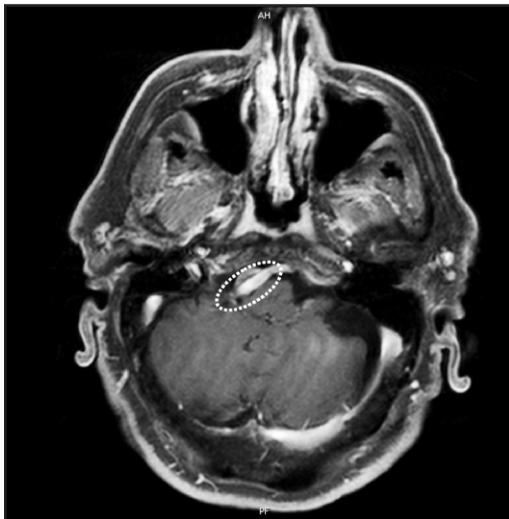


Figure 5. Gadolinium-enhanced MRI (T2 weight, high spin) showing the aneurysm on the right vertebral artery (dashed circle).

the contralesional side, the VEMP amplitude was 374 μ V, which clearly exceeds 95 percent limits (solid line). Although the 34 percent asymmetry ratio for this patient was within two standard deviations, the combination of the abnormally low threshold (55 dB nHL, $p = .001$) and large VEMP amplitude ($p = .025$) on the contralesional side is compelling. We therefore feel confident that the abnormally large VEMP was a true finding.

Most reports of VEMP abnormality refer to absent, reduced, or delayed responses (de Waele et al, 1999; Matsuzaki et al, 1999; Chen et al, 2000; Itoh et al, 2001; Halmagyi et al, 2002; Chen and Young, 2003; Liao and Young, 2004; Node et al, 2005; Kingma, 2006). Abnormally large-amplitude VEMPs are clinically rare but have been reported in Ménière's disease, presumably with a dilated saccule (Young et al, 2002b), delayed endolymphatic hydrops (Young et al, 2002a), enlarged vestibular aqueduct (Sheykhosslami et al, 2004), labyrinthine fistula, or dehiscent superior semicircular canal (Brantberg et al, 1999; Minor et al, 2001). In all of these

instances, the large-amplitude VEMP could be explained on the basis of altered labyrinthine hydromechanics.

To understand how altered hydromechanical changes might lead to an enlarged VEMP, recall that the saccule is located directly medial to the stapes footplate. The distance from the bottom of the footplate to the saccule is fairly consistent and ranges from 0.38 mm to 1.4 mm (Anson et al, 1965). Under normal conditions, stapes movement produces a pressure gradient that travels down the cochlea, where it is shunted through the round window. Saccular distension (as might occur in acute Ménière's disease) or a relative decrease in perilymph pressure (as might occur in labyrinthine fistula or superior semicircular canal dehiscence) could change the relative isolation of the saccule from this pressure gradient and result in more compression/expansion of the saccule from movement of the stapes. This in turn can result in a larger saccular action potential volley and, hence, a larger-amplitude VEMP.

No such change in labyrinthine mechanics was observed in this case. Specifically,

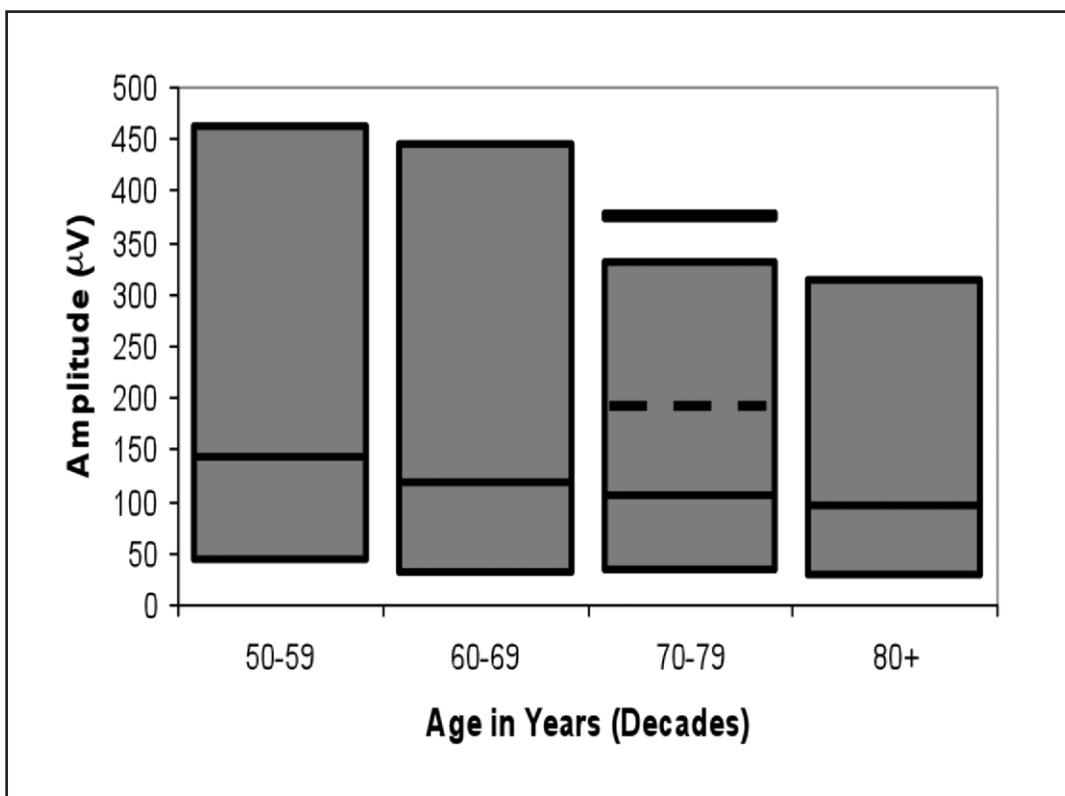


Figure 6. Normal vestibular evoked myogenic potential (VEMP) amplitude limits (95%) by patient age in decade. The solid line within each bar represents the geometric mean amplitude for each age group. The dashed line represents the right VEMP amplitude (70- to 80-year-old age range). The solid line above the range represents the amplitude of the left VEMP.

signs associated with semicircular canal dehiscence were absent (i.e., no Tullio or Hennebert effect, no low-frequency hearing loss, and no conductive component to the hearing loss). CT reports did not note enlarged vestibular aqueducts or semicircular canal dehiscence. The patient's presentation was entirely compatible with brain stem stroke. Consequently, a neurogenic explanation for the enlarged contralesional VEMP seems necessary. Unfortunately, current understanding of the neural pathways underpinning the VEMP and understanding of the functional organization of the vestibular nuclei are still primordial. Nevertheless, the following possibility seems intriguing and potentially explanatory.

Recall that the VEMP recorded from the sternocleidomastoid muscle is an inhibitory response that likely involves the medial vestibulospinal tract. It is known that the medial vestibular nucleus, the largest of the four primary vestibular nuclei, receives significant afferent input from the saccule and utricle. Interestingly, the medial vestibular nucleus receives only small-diameter fibers, which are, in general, inhibitory in nature (Baloh and Honrubia, 2001). It also receives a large commissural projection from the contralateral medial vestibular nucleus. These commissural projections in mammals are thought to provide a signal used in central compensation after unilateral loss of vestibular function (Precht et al, 1966; Shimazu and Precht, 1966; Guyot et al, 1995). Uchino and colleagues (2001) studied otolith-specific commissural fibers and found that only 10 percent of saccular-activated second-order neurons received commissural inhibition, 16 percent received commissural facilitation, and 74 percent showed no response to stimulation of the contralateral saccular nerve.

In this patient, the vestibular commissural pathways would be anatomically located at the superior medial aspect of the infarct. It is hypothesized that some of the commissural inhibitory fibers were damaged. The loss of commissural inhibitory fibers from the right, ipsilateral medial vestibular nucleus to the left, contralateral medial vestibular nucleus would result in a disinhibition (loss of inhibitory influence) of the left medial vestibular nucleus. Since the VEMP is an inhibitory reflex from the

medial vestibular nuclei via the MVST, then loss of inhibition (from the infarct involving the commissural fibers from the right medial vestibular nucleus) to the left medial vestibular nucleus would result in a larger response to saccular stimulation and a larger-amplitude VEMP.

In support of this idea, consider that it is unlikely that the ipsilateral medial or lateral vestibular nuclei were affected by the infarct, as the ipsilateral VEMP was normal. Also, it is unlikely that the contralateral medial vestibular nuclei were affected by the infarct, as the contralateral VEMP was present with a large amplitude. Additionally, there is independent evidence that commissural projections from the medial vestibular nucleus may be selectively impaired in Wallenberg's syndrome. Dieterich and colleagues (2005) show that cortical representation of caloric stimuli during positron-emission tomography scanning was altered in three cases of Wallenberg's syndrome, with the pattern consistent with loss of ipsilesional medial vestibular nuclei output to the contralateral medial longitudinal fasciculus. This damage spared ipsilaterally ascending vestibulo-cortical pathways.

There are other potential influences on vestibulospinal outflow that may have been affected in this case. For example, the strong lateral propulsion, positional nystagmus, and hyperactive caloric responses implicate ipsilesional changes in central vestibular function. Cerebellar projections onto the vestibular nuclei are numerous, reciprocal, and known to play a role in calibrating vestibular reflexes. Further, the primary pathway carrying cerebellar control over the vestibular nuclei would be the restiform body (inferior cerebellar peduncle). However, it is difficult to explain how ipsilateral damage to this pathway would result in only a contralateral disinhibition.

Similarly, there are vaguely defined pathways from the cervical spine to the vestibular nuclei that have been shown to be sensitive to otolith stimulation (Buttner-Ennever, 1999). These are thought to reflect a feedback loop carrying information about the effectiveness of vestibular spinal stimulation back to vestibular nuclei. Some reports suggest that these ascending pathways may be bilateral. However, it still

remains unclear how unilateral damage to such a feedback loop could selectively disinhibit the contralateral pathway.

CONCLUSION

A large-amplitude VEMP has been attributed to several types of end-organ pathologies (Ménière's disease, delayed endolymphatic hydrops, enlarged vestibular aqueduct, and dehiscence superior semicircular canal syndrome). This case is the first report of a large-amplitude VEMP associated with a central, brain stem etiology—dorsolateral medullary stroke (Wallenberg's syndrome). It is hypothesized that loss of medial vestibular commissural inhibitory fibers resulted in disinhibition of the contralateral medial vestibular nucleus, causing a large-amplitude VEMP on the side contralateral to the brain stem stroke.

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