Characteristics and clinical applications of vestibular-evoked myogenic potentials

Miriam S. Welgampola, MBBS, PhD; and James G. Colebatch, FRACP, PhD

Abstract—A recent technique of assessing vestibular function, the vestibular-evoked myogenic potential (VEMP), is an otolith-mediated, short-latency reflex recorded from averaged sternocleidomastoid electromyography in response to intense auditory clicks delivered via headphones. Since their first description 10 years ago, VEMPs are now being used by investigators worldwide, and characteristic changes observed with aging and in a variety of peripheral and central vestibulopathies have been described. Additional methods of evoking VEMPs, which use air- and bone-conducted short-tone bursts, forehead taps, and short-duration transmastoid direct current (DC) stimulation, have been described, and these complement the original technique. Click-evoked VEMPs are attenuated or absent in a proportion of patients with vestibular neuritis, herpes zoster oticus, late Ménière disease, and vestibular schwannomas; their amplitudes are increased and thresholds are pathologically lowered in superior semicircular canal dehiscence presenting with the Tullio phenomenon. VEMPs evoked by clicks and DC are useful when monitoring the efficacy of intratympanic gentamicin therapy used for chemical vestibular ablation. Prolonged p13 and n23 peak latencies and decreased amplitudes have been observed in association with central vestibulopathy. VEMPs evoked by clicks are a robust, reproducible screening test of otolith function. DC stimulation enables differentiation of labyrinthine from retrolabyrinthine lesions; bone-conducted stimuli permit VEMP recording despite conductive hearing loss and deliver a relatively larger vestibular stimulus for a given level of auditory perception.

NEUROLOGY 2005;64:1682–1688

Background. Although primarily responsive to head acceleration, vestibular afferents are activated by nonphysiologic techniques using loud sound, vibration, and electrical stimulation applied over the mastoid processes. The click vestibular-evoked myogenic potential (VEMP) is a technique based on residual acoustic sensitivity of the sacculus, which, during the course of its evolution, functioned as an organ of hearing and still does so in primitive vertebrates.[1,2] Sound-evoked vestibular responses in humans were described by Von Békésy[3] who, using intense sounds of 128 to 134 db, evoked head movement toward the stimulated ear. Displacement of the stapes footplate, which lies in close proximity to the sacculus, was thought to lead to eddy current formation within the endolymph, hair cell displacement, and activation of primary afferents.

Vestibular-dependent short-latency electromyographic (EMG) responses to intense sound were initially recorded from the posterior neck muscles inserting at the inion ("inion response").[4] Responses were recordable only during activation of the relevant muscles. They were preserved despite sensorineural hearing loss and abolished in vestibulopathy. Studies performed on selective inner ear lesions identified the saccule to be the responsive end organ.[5] VEMPs are now recorded using symmetric sites over the sternocleidomastoid muscles (SCMs).[6] The response consists of an initial positivity or inhibition (p13) followed by a negativity or excitation (n23) (figure 1). Later components (n34, p44) (figure 2) have a lower stimulus threshold and are nonvestibular (probably cochlear) in origin. The short-onset latency of the VEMP (about 8 milliseconds) indicates that it is likely to be mediated by an oligosynaptic pathway, possibly disynaptic and consisting of primary vestibular afferents projecting to the vestibular nuclear complex and thence via the medial vestibulospinal tract to the accessory nucleus.

The VEMP arises from modulation of background EMG activity and differs from neural potentials in that it requires tonic contraction of the muscle. It is best observed in averaged unrectified EMG (see figure 2). In single-unit EMG recordings, intense clicks are followed by a 2- to 6-millisecond period of inhibition between 8 and 20 milliseconds following the

From the Institute of Neurological Sciences (Drs. Welgampola and Colebatch), Prince of Wales Hospital and School of Medicine, University of New South Wales, Sydney, Australia; and Sobell Department for Motor Neuroscience and Movement Disorders (Dr. Welgampola), National Hospital for Neurology and Neurosurgery, London, UK.

Supported by the Garnett Passe and Rodney Williams Memorial Foundation and the National Health and Medical Research Council of Australia.

Received June 23, 2004. Accepted in final form January 3, 2005.

Address correspondence and reprint requests to Dr. M.S. Welgampola, Sobell Department for Motor Neuroscience and Movement Disorders, National Hospital for Neurology and Neurosurgery, Queen Square, London WC 1N 3BG, UK; e-mail: m.welgampola@unsw.edu.au

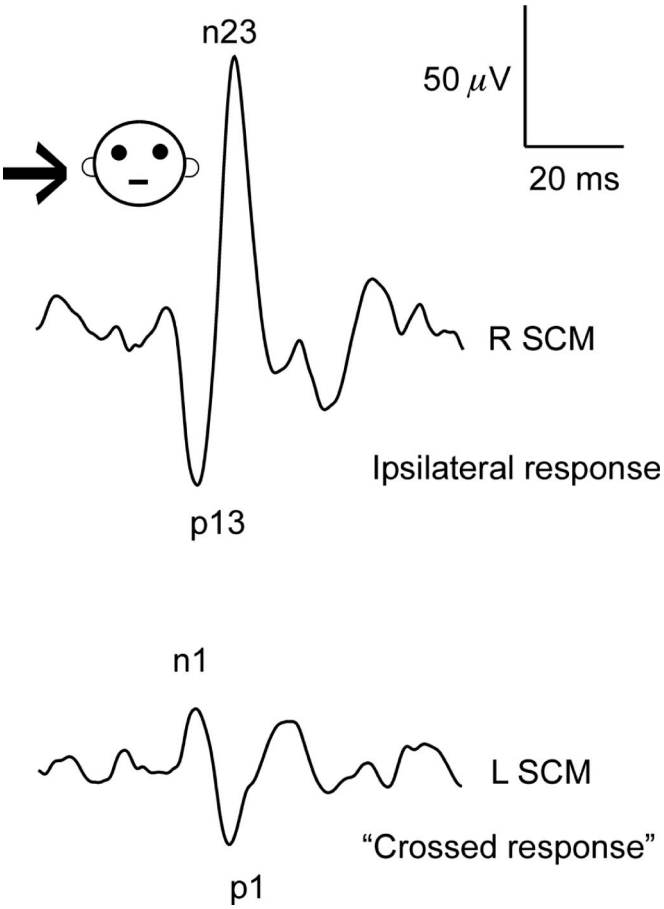

Figure 1. Vestibular-evoked myogenic potential evoked by a 100 db normal hearing level click delivered in the right ear via headphones. The traces consist of averaged unrectified electromyogram from both sternocleidomastoid muscles (SCMs). A large biphasic p13n23 response is seen ipsilateral to the stimulated ear, and a smaller response of opposite polarity (n1p1) is seen contralaterally.

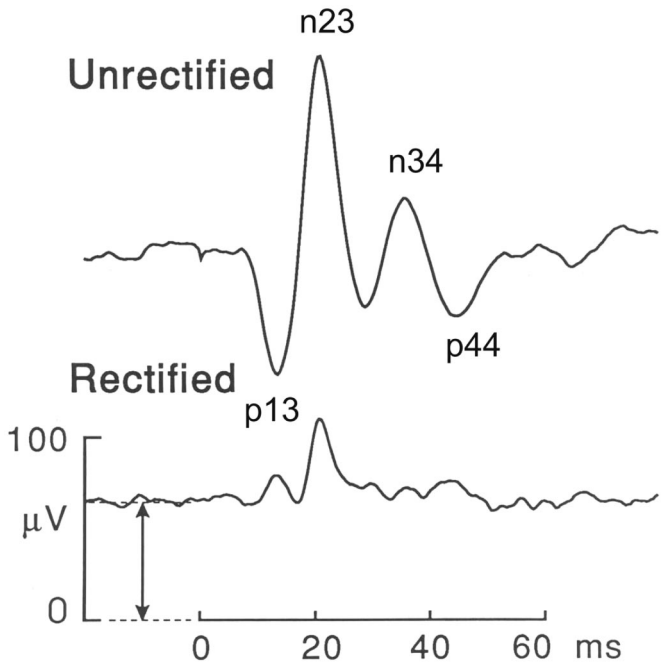

Figure 2. Simultaneous averages of rectified and unrectified sternocleidomastoid electromyograms recorded from a normal subject. The stimulus was delivered at time 0. Responses to 512 stimuli were averaged. The initial biphasic p13n23 response is larger on the unrectified trace. The late response (n34p44) is likely to represent cochlear stimulation. Modified from ref. 6.

stimulus that coincides with the surface positivity. Blockade of the motor point abolishes the response.[7] The reflex amplitude scales in proportion to tonic EMG activity and should therefore be normalized to the level of EMG activity ("corrected reflex amplitude" = peak-to-peak amplitude/prestimulus-rectified EMG activity). Side-to-side differences in reflex amplitude can be expressed as an asymmetry ratio (AR) using the following formula: $AR\% = 100 * (A_l - A_s)/(A_l + A_s)$, where A_l and A_s are the larger and smaller amplitudes obtained from stimulating each ear.

Morphologic and physiologic studies in experimental animals confirm that intense sound selectively activates otolith afferents.[8-12] Stimulation of the saccular nerve in cats results in inhibitory postsynaptic potentials in the ipsilateral SCM motor neurons, which travel in the medial vestibulospinal tract[13,14] with only weak effects on the contralateral neurons. Utricular nerve stimulation, in contrast, evokes excitatory postsynaptic potentials in about two-thirds of contralateral SCM neurons.[13] Thus, the predominantly ipsilateral, inhibitory SCM responses (e.g., click VEMPs) are likely to represent saccular activation, and prominent crossed responses (observed in direct current [DC]– and tap-evoked VEMPs) may indicate utricular stimulation.

VEMPs recorded from clicks and tones delivered via headphones. Intense clicks of about 95 to 100 db above normal hearing level (NHL) (equivalent to 140 to 145 db sound pressure level [SPL]) are required to evoke VEMPs and are at the limit of what is considered safe but are generally well tolerated. Stimuli of 95 db NHL and 0.1-millisecond duration are used in routine clinical tests performed on subjects below age 60. The presence of tinnitus is a relative contraindication to click and tone burst VEMP testing, and an alternate stimulus should be considered in this condition. A calibrated sound source is essential. An intact middle ear conductive apparatus is needed to convey the click to the end organ. The response is abolished or attenuated in conductive hearing loss with air–bone gaps as small as 8.75 db.[15]

The amplitude of the p13n23 response is largely determined by click intensity and the level of tonic SCM contraction.[16] Ideally, rectified EMG is averaged or, alternatively, feedback given to subjects to control for the levels of tonic muscle activation. Subjects must activate their SCMs, for example, by lying semirecumbent and lifting their heads, so that ade-

quate levels of tonic neck activation are maintained during the recording (corresponding to a mean rectified EMG of about 60 μV). Alternate methods of bilateral SCM activation by pushing the head forward against the resistance of a padded bar while sitting upright, causing isometric contraction of both SCMs, are less comfortable and cannot be sustained for prolonged periods. Unilateral activation by rotating the head against resistance permits recording from only a single SCM.

The optimal site for electrode placement is the middle third of the SCM.[17] The VEMP is best seen in averaged unrectified EMG; rectified EMG is also measured for monitoring background activation. A typical amplifier setting uses a gain of 5,000 (200 μV/V) and a band pass filter of 10 Hz to 2 kHz. Typically, 256 repetitions are averaged, and stimulus repetition rates of up to 5 Hz can be used without a decrement in amplitude.[18] The initial positive–negative response (p13n23) is vestibular dependent and strictly ipsilateral to the stimulus.[19] A contralateral response of opposite polarity, with an initial negativity (n1p1, crossed neural response), is present infrequently (see figure 1). These are small, becoming prominent only in the presence of vestibular hypersensitivity to sound.[20]

Click-evoked VEMPs are present in all subjects younger than 60 years. Large interindividual variability in VEMP amplitudes (e.g., corrected and uncorrected click VEMP amplitudes of 0.5 to 3.0 and 25 to 297 μV peak to peak in normal subjects age <60) limits the use of absolute amplitudes as measures of otolith function. Within-subject left-to-right asymmetry is likely to be a more sensitive method of detecting unilateral hypofunction. With use of corrected reflex amplitudes, ARs up to 35% have been calculated for subjects below age 60.[21] As between-experiment reproducibility is high ($r = 0.92$), click VEMPs are likely to be useful in longitudinal studies. Decreased amplitudes, increased thresholds, and increasing asymmetry are seen from the sixth decade. Mean stimulus thresholds rise from 85 db in the third decade to 96 db in the eighth and ninth decades.[21] Click VEMPs can be absent in normal subjects over age 60; therefore, these tests should be interpreted with caution in older subjects presenting with vertigo.

Short tone bursts of 250- to 2,000-Hz frequencies, 2- to 10-millisecond duration, and intensities of 120 db SPL evoke VEMPs similar to clicks[22,23] (figure 3B). Their optimum stimulus frequencies lie between 500 and 1,000 Hz.[22,23] VEMPs of opposite polarity to those from the SCM have been recorded from the splenius capitis during tonic activation.[24] Reflex amplitudes were shown to increase with increasing tone burst duration up to 7 to 10 milliseconds and decrease thereafter, probably owing to activation of the stapedius reflex.[23] Peak latencies increase with increasing stimulus duration.[23]

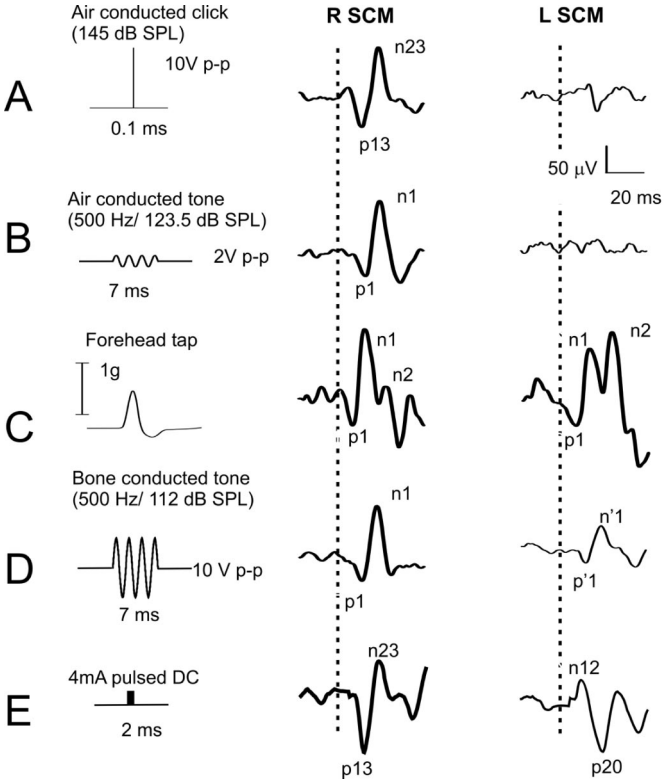

Figure 3. Myogenic potentials to five different stimuli (A through E). The traces consist of averaged unrectified electromyograms recorded from the left and right sternocleidomastoid muscles (SCMs). The thick traces represent responses ipsilateral to and the thin traces contralateral to the side of stimulation. (A) The 145 db sound pressure level (SPL) clicks delivered in the right ear evoked p13n23 responses only in the ipsilateral SCM. (B) Air-conducted tones (123.5 db SPL/500 Hz/7 ms), like clicks, give rise only to ipsilateral p1n1 responses. (C) Forehead taps produced bilateral symmetric vestibular-evoked myogenic potentials by activating both labyrinths; these consisted of an initial positivity followed by two negativities (n1, n2), of which the first is vestibular dependent. (D) Bone-conducted tones (112 db SPL/500 Hz/7 ms) also result in p1n1 responses bilaterally; the ipsilateral response is larger and occurs earlier in most subjects. (E) The 4-mA/2-ms transmastoid galvanic stimulation evokes an ipsilateral p13n23 response and a contralateral n12p20 response (crossed excitation or crossed neural response). Tone amplitudes are plotted to scale; head acceleration is shown for taps. DC = direct current.

VEMPs evoked by bone-conducted stimuli: skull taps and bone-conducted tones. Skull taps and bone-conducted tones are stimuli that bypass the middle ear conductive apparatus and can evoke VEMPs despite conductive hearing loss. A forehead tap, delivered at F_{pz} (International 10–20 System) via a tendon hammer, evokes a vestibular-dependent short-latency p1n1 response in both SCMs[25] (see figure 3C). The tap also evokes a second negativity ("n2"), which can sometimes be difficult to separate from n1 and thus precludes unambiguous analysis in some normal subjects. In unilateral ves-

tibular deafferentation, a prominent crossed response is seen on the SCM ipsilateral to the lesion, representing crossed excitation from the intact side.

The technique is operator dependent and does not deliver a calibrated stimulus. Taps delivered laterally above the ear evoke inverted VEMPs in the ipsilateral SCM and those of normal polarity in the contralateral SCM, suggesting that the contralateral vestibular apparatus is more effectively stimulated.[26] Tap-evoked VEMPs, owing to the magnitude of the stimulus, are 1.5 to 3 times as large as those evoked by clicks. They are relatively preserved in older subjects in whom stimulus thresholds are likely to be high.[21] The precise afferents activated by skull taps are not known, although based on the appearance of lateral skull taps in normal subjects and in unilateral vestibulopathy, utricular stimulation has been proposed.[26,27]

A bone-conducted tone burst delivered over the mastoid process, via a B71 clinical bone vibrator (Radioear Corp., Philadelphia, PA), routinely used in audiometric testing, evokes VEMPs despite conductive hearing loss.[28,29] Optimum stimulation is delivered with the conductor placed 3×2 cm posterosuperior to the external acoustic meatus, using frequencies of 200 to 250 Hz.[29,30] VEMPs are often bilateral (see figure 3D), as the stimulus is transmitted via bone and activates end organs on both sides. The ipsilateral VEMP is about 1.5 times larger and occurs approximately 1 millisecond earlier. Rarely, larger responses have been recorded contralateral to the stimulated ear, reflecting the vagaries of bone conduction. A mean AR of 17.3% with a range of 0 to 58% was recorded in a group of subjects ages 24 to 52.[29] The stimulus threshold for bone vibration is less than 50 dB above hearing level and this allows relatively stronger vestibular stimulation to be given without causing cochlear injury. Clinical bone vibrators used in conjunction with audiometers need additional amplification to produce stimuli intense enough for VEMP recording.

VEMPs evoked by galvanic stimulation. A short-duration (2-millisecond) pulsed current delivered via electrodes attached to the mastoid processes evokes a p13n23 response on the side ipsilateral to cathodal stimulation similar to that evoked by sound[31] (see figure 3E). Stimuli of 4 mA/2 ms, as used for clinical testing, are well tolerated by patients. Such a current, in close proximity to the recording site, causes a large stimulus artifact, and specific subtraction techniques are required to recover the response of interest.[31] DC stimulation acts proximal to the end organ, at the terminal part of the primary vestibular afferent, the spike trigger zone. Cathodal currents increase and anodal currents decrease spontaneous firing rates.[32] A monaural cathodal stimulus evokes an ipsilateral p13n23 response and a contralateral n12p20 crossed neural response in all subjects. The greater frequency of a crossed neural response when compared with clicks may indicate activation of utricular afferents.

This technique should help distinguish between end organ (labyrinthine) lesions and more proximal (retrolabyrinthine) lesions. In confirmation of this, all 10 subjects with Ménière disease and endolymphatic hydrops had well preserved galvanic VEMPs, whereas 16 of 18 subjects with cerebellopontine angle tumors had reduced or absent responses.[33]

ARs of 0 to 41.3% were recorded in normal subjects below age 60. Amplitudes were found to decrease from the seventh decade.[21] Responses can be absent in normal subjects over age 60.[21] Vestibular-evoked myogenic responses to galvanic stimulation have recently been recorded from the human masseter muscles.[34]

VEMPs in peripheral vestibulopathy. *Ménière disease.* Absent VEMPs were reported in 35 to 54% of affected ears in Ménière disease.[35,36] The absence correlated with low-frequency hearing loss and poor performance in posturography under vision-deprived sway-referenced conditions (situations in which vestibular dependence increases). VEMP amplitudes are increased in early Ménière disease and attenuated or absent in later stages.[37] Augmentation was attributed to saccular dilatation with pressure on the stapes footplate, leading to enhanced saccular sensitivity. Later depression of VEMPs was thought to be due to dilatation of the saccule and atrophied sensory epithelium. Absent VEMPs in advanced disease may represent collapse of the saccular membrane on the sensory epithelium.[37] These hypotheses are still to be proven histopathologically. In modified Klokhoff tests using IV furosemide or orally administered glycerol (1.3 mg/kg body wt), an increase in VEMP amplitude or reappearance of the VEMP has been demonstrated after diuresis.[36,38] The reversal of reflex attenuation was attributed to reduction of saccular hydrops following diuresis.

Vestibular neuritis. Absent click VEMPs were reported in 12 to 39% of subjects diagnosed with vestibular neuritis.[39-41] Subjects who subsequently develop benign positional vertigo (BPV) demonstrate preservation of the VEMP. An intact inferior vestibular nerve (which supplies the posterior semicircular canal as well as the saccule) thus seems necessary to generate BPV. In 11 subjects with vestibular neuritis studied within 1 month of symptom onset, in whom caloric responses and click VEMPs were absent, absence of both click and galvanic VEMPs was reported in 8 ("neuritis pattern").[42] The presence of a galvanic VEMP despite absence of click-evoked VEMP in the remaining three localized the lesion to the labyrinth in these cases ("labyrinthitis pattern").[42] Whether the loss of both galvanic and click-evoked responses represents combined lesions of the labyrinth and the vestibular nerve ("neurolabyrinthitis") or secondary changes in afferents after severe end organ injury is not known. A recent comparison of click- vs tap-evoked VEMPs in vestibular neuritis showed absent tap-evoked responses on the affected side in 56% and absent click-evoked responses in 22%. As vestibular neuritis more commonly affects

the superior vestibular nerve territory, greater prevalence of abnormal tap responses may indicate that taps excite more afferents from this territory (possibly utricular afferents) than clicks.[27] Absent click-evoked VEMPs have also been reported in five of eight subjects with the Ramsay Hunt syndrome, in whom absence of VEMPs correlated with vertigo.[43]

Bilateral vestibulopathy. VEMPs were absent bilaterally in two of three patients with idiopathic bilateral vestibulopathy.[44] The preservation of clicks in such subjects is a useful indicator of residual function. Documentation of bilaterally absent VEMPs (in addition to absent caloric responses) is essential to diagnose complete vestibulopathy.

Vestibular schwannomas. Click VEMPs were absent in 72.2 to 80% of subjects presenting with vestibular schwannomas.[45-48] VEMPs may complement the information obtained from brainstem auditory-evoked responses (BAERs) and caloric function tests in these patients, in whom absent VEMP and normal BAERs have sometimes been reported.[49] The utility of VEMPs in predicting the vestibular nerve division giving rise to the tumor is unclear. Absence of VEMPs has ranged from 35 to 100% in patients who had tumors arising from the inferior vestibular nerve.[50,51] The presence of postoperative VEMPs in 3 of 10 subjects with inferior vestibular schwannomas has been reported but cannot be easily explained in terms of current knowledge of this response.[52] In a large series of 170 patients presenting with vestibular schwannomas, 78.8% had absent or low-amplitude responses to clicks or 500-Hz short-tone bursts. Whereas 69.4% of all subjects had absent click-evoked VEMPs, 23.5% of the entire group had absent click VEMPs and normal or reduced VEMPs to short-tone bursts, suggesting that tone burst VEMPs may be of value in detecting residual inferior vestibular nerve function.[48]

Gentamicin therapy. VEMPs can be used to monitor the effects of low-dose intratympanic gentamicin injections used to achieve chemical labyrinthectomy, a procedure used to control debilitating vertigo in Ménière disease and other peripheral vestibulopathies. Post treatment, 92% of patients had absent VEMPs at 1 month and remained so at 2-year follow-up.[53] Of these, 32% had also lost VEMPs to DC stimulation at 1 month, with the percentage rising to 41% at 6 months and 1 year and 46% at 2 years. Those with absent click- and DC-evoked VEMPs did not develop recurrent vertigo. VEMPs evoked by DC stimulation could thus be a useful tool in monitoring adequacy of intratympanic gentamicin. Serial evaluations using click VEMPs may also have a potential application in monitoring subjects who require prolonged systemic gentamicin therapy.

Tullio phenomenon. The superior canal dehiscence (SCD) syndrome is characterized by vertigo and oscillopsia in response to loud sounds (Tullio phenomenon).[54] The dehiscence is thought to create a low-impedance pathway ("third window"), resulting in enhanced vestibular sensitivity. The diagnosis is made on the basis of high-resolution CT scans, which demonstrate the dehiscence, sound-evoked ocular movements, and pathologically lowered VEMP thresholds of 55 to 70 db NHL (100 to 115 db SPL).[20,55-57] Click VEMP amplitudes are larger on the affected side. Subjects with unilateral symptoms may have the CT appearance of bilateral SCD but lowered VEMP thresholds only in symptomatic ears.[55,56] A subgroup with SCD have low-frequency "air–bone gaps" (better hearing for bone-conducted sound than for air) in the affected ears yet retain VEMPs evoked from stimulation of the affected ear, a finding uncharacteristic of a middle ear conductive disorder.[57,58] Enhanced sensitivity to bone-conducted sound ("conductive hyperacusis") is likely to be the basis for the air–bone gap. VEMP testing in subjects with apparent conductive hearing loss may help identify those with undiagnosed SCD, even in the absence of a history of vertigo.[59] VEMPs to bone-conducted tones also show significantly larger amplitudes and lowered or low normal thresholds in SCD.[29,60] It is not known whether low bone-conducted VEMP thresholds correlate with the clinical observation of conductive hyperacusis and air–bone gaps in audiometry.

DC VEMPs are normal in SCD, indicating that the increase in excitability occurs at receptor level. Tap VEMPs, although larger than click VEMPs in normal subjects, are not enlarged in parallel with clicks in SCD.[60,61] Comparison of the tap- and click-evoked response amplitudes will help differentiate between a large click VEMP due to dehiscence and a nonpathologically enlarged response.

Otosclerosis. As conduction across the middle ear ossicular chain is defective, VEMPs are attenuated or absent in subjects with otosclerosis.[62,63] Attenuation of the VEMP occurs early; therefore, apparent conductive hearing loss without abolition of VEMPs warrants careful investigation for an alternate diagnosis such as SCD with enhanced bone conduction. VEMP testing may be of potential value in monitoring the efficacy of stapes mobilization procedures.

VEMPs in central vestibulopathy. VEMP latency prolongation is an abnormality characteristic of central vestibulopathy. Prolonged p13 and n23 peak latencies were observed in subjects with multiple sclerosis (in 5.8 to 100% of the sides tested for p13 and 1.4 to 44.4% for n23). The abnormal latencies were attributed to slowing of conduction along the vestibulospinal pathways consequent to demyelination.[64-67] Absent or attenuated VEMPs were reported in 19 to 25% of sides tested.[64,66] The prevalence of symptoms of vestibular dysfunction in these patients ranged from 31 to 65%.

Significant prolongation of p13 and n23 peak latencies was present only in the subgroup with clinical evidence of brainstem involvement.[67] Peak latencies correlated significantly with disability scores and disease duration.[67]

Absence of VEMPs was reported in two of four subjects with the Wallenberg syndrome, those with

abnormal results having been studied more acutely.[68] Of seven subjects presenting with brainstem infarcts or hemorrhage, VEMPs were absent bilaterally in three and unilaterally in two. Age-related effects may account for a proportion of the abnormalities, as most of the patients tested were over age 60.[69]

Subjects with large acoustic neuromas compressing the brainstem have been reported to show p13 and n23 latency prolongation.[64] In survivors of irradiated nasopharyngeal carcinoma, p13 and n23 latencies were delayed in 50% of the ears in subjects irradiated once and 100% of the ears in those irradiated twice.[70] These abnormalities were attributed to brainstem pathology.

As expected, VEMPs were preserved in midbrain and upper pontine lesions likely to have been above the level of the vestibular nuclei.[68,71] Whereas VEMP abnormalities were present in medullary and mid to lower pontine lesions, BAER abnormalities were more common in upper brainstem (midbrain and pons) lesions.[71]

Conclusion. VEMPs are a new and relatively innocuous technique for assessing otolith function. Of the techniques currently in use, clicks are the most reproducible, symmetric, and technically easy to perform. Normal values for their thresholds, latencies, amplitudes, and side-to-side differences have been defined. They best fit the role of a screening test of otolith function. The remaining techniques should be used when specifically indicated. When click or tone burst-evoked VEMPs are absent, technical and physiologic causes (inadequate stimulus intensity, inadequate SCM activation, conductive hearing loss, and age effects) should first be sought. Audiometry should be performed to determine whether conductive hearing loss is present, and an alternate stimulus that bypasses the middle ear (taps, bone vibration) should be used. Bone-conducted VEMPs are less well lateralized than those evoked by clicks. They deliver a relatively mild acoustic stimulus. Unlike air-conducted sound, there is the potential to deliver an intense bone-conducted vestibular stimulus with little risk of acoustic trauma. In comparison with the techniques currently used to elicit bone vibration, the tap still delivers a larger stimulus; thus, it has a potential role for testing otolith function in subjects with absent VEMPs to all other stimulus modalities. Galvanic-evoked VEMPs should be used when VEMPs are absent or attenuated in response to both air- and bone-conducted stimuli.

Collectively, these techniques represent a clinically useful addition to existing vestibular function tests, which are currently biased toward the assessment of canal rather than otolith function.

Note added in proof. Bone conduction stimuli should be given in units of dB FL (force level), as a ratio to a reference force of 1 uN, and these values are (10–20 dB) higher than previously given "SPL" values (depending on the stimulus frequency).

References

1. Moffat AJM, Capranica RR. Auditory sensitivity of the saccule in the American toad (*Bufo americanus*). J Comp Physiol 1976;105:1–8.
2. Lowenstein O, Roberts TD. The localization and analysis of the responses to vibration from the isolated elasmobranch labyrinth; a contribution to the problem of the evolution of hearing in vertebrates. J Physiol 1951;114:471–489.
3. Békésy G von. Über akustische Reizung des Vestibularapparates. Pflugers Arch 1935;236:59–76.
4. Bickford RG, Jacobson JL, Cody DT. Nature of average evoked potentials to sound and other stimuli. Ann NY Acad Sci 1964;112:204–223.
5. Townsend GL, Cody DT. The averaged inion response evoked by acoustic stimulation: its relation to the saccule. Ann Otol Rhinol Laryngol 1971;80:121–131.
6. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 1994;57:190–197.
7. Colebatch JG, Rothwell JC. Motor unit excitability changes mediating vestibulocollic reflexes in the sternocleidomastoid muscle. Clin Neurophysiol 2004;115:2567–2573.
8. McCue MP, Guinan JJ. Acoustically responsive fibers in the vestibular nerve of the cat. J Neurosci 1994;14:6058–6070.
9. McCue MP, Guinan JJ. Spontaneous activity and frequency selectivity of acoustically responsive vestibular afferents in the cat. J Neurophysiol 1995;74:1563–1572.
10. Murofushi T, Curthoys IS, Topple AN, Colebatch JG, Halmagyi GM. Responses of guinea pig primary vestibular neurons to clicks. Exp Brain Res 1995;103:174–178.
11. Murofushi T, Curthoys IS. Physiological and anatomical study of click-sensitive primary vestibular afferents in the guinea pig. Acta Otolaryngol (Stockh) 1997;117:66–72.
12. Murofushi T, Curthoys IS, Gilchrist DP. Response of guinea pig vestibular nucleus neurons to clicks. Exp Brain Res 1996;111:149–152.
13. Uchino Y, Sato H, Sasaki M, et al. Sacculocollic reflex arcs in cats. J Neurophysiol 1997;77:3003–3012.
14. Kushiro K, Zakir M, Ogawa Y, Sato H, Uchino Y. Saccular and utricular inputs to sternocleidomastoid motoneurons of decerebrate cats. Exp Brain Res 1999;126:410–416.
15. Bath AP, Harris N, McEwan J. Effect of conductive hearing loss on the vestibulocollic reflex. Clin Otolaryngol 1999;24:181–183.
16. Lim CL, Clouston P, Sheean G, Yiannikas C. The influence of voluntary EMG activity and click intensity on the vestibular click evoked myogenic potential. Muscle Nerve 1995;18:1210–1213.
17. Sheykholeslami K, Murofushi T, Kaga K. The effect of sternocleidomastoid electrode location on vestibular evoked myogenic potential. Auris Nasus Larynx 2001;28:41–43.
18. Wu CH, Murofushi T. The effect of click repetition rate on vestibular evoked myogenic potential. Acta Otolaryngol 1999;119:29–32.
19. Li MW, Houlden D, Tomlinson RD. Click evoked responses in sternocleidomastoid muscles: characteristics in normal subjects. J Vestib Res 1999;9:327–334.
20. Colebatch JG, Day BL, Bronstein AM, et al. Vestibular hypersensitivity to clicks is characteristic of the Tullio phenomenon. J Neurol Neurosurg Psychiatry 1998;65:670–678.
21. Welgampola MS, Colebatch JG. Vestibulocollic reflexes: normal values and the effect of age. Clin Neurophysiol 2001;112:1971–1979.
22. Murofushi T, Matsuzaki M, Wu CH. Short tone burst-evoked myogenic potentials on the sternocleidomastoid muscle: are these potentials also of vestibular origin? Arch Otolaryngol Head Neck Surg 1999;125:660–664.
23. Welgampola MS, Colebatch JG. Characteristics of tone burst-evoked myogenic potentials in the sternocleidomastoid muscles. Otol Neurootol 2001;22:796–802.
24. Wu CH, Young YH, Murofushi T. Tone burst-evoked myogenic potentials in human neck flexor and extensor. Acta Otolaryngol. 1999;119:741–744.
25. Halmagyi GM, Yavor RA, Colebatch JG. Tapping the head activates the vestibular system: a new use for the clinical reflex hammer. Neurology 1995;45:1927–1929.
26. Brantberg K, Tribukait A. Vestibular evoked myogenic potentials in response to laterally directed skull taps. J Vestib Res 2002;12:35–45.
27. Brantberg K, Tribukait A, Fransson PA. Vestibular evoked myogenic potentials in response to skull taps for patients with vestibular neuritis. J Vestib Res 2003;13:121–130.
28. Sheykholeslami K, Murofushi T, Kermany MH, Kaga K. Bone-conducted evoked myogenic potentials from the sternocleidomastoid muscle. Acta Otolaryngol 2000;120:731–734.
29. Welgampola MS, Rosengren SM, Halmagyi GM, Colebatch JG. Vestibular activation by bone conducted sound. J Neurol Neurosurg Psychiatry 2003;74:771–778.
30. Sheykholeslami K, Habiby Kermany M, Kaga K. Frequency sensitivity range of the saccule to bone-conducted stimuli measured by vestibular evoked myogenic potentials. Hear Res 2001;160:58–62.
31. Watson SR, Colebatch JG. Vestibulocollic reflexes evoked by short-duration galvanic stimulation in man. J Physiol 1998;513:587–597.

32. Goldberg JM, Fernandez C, Smith CE. Responses of vestibular-nerve afferents in the squirrel monkey to externally applied galvanic currents. Brain Res 1982;252:156–160.
33. Murofushi T, Takegoshi H, Ohki M, Ozeki H. Galvanic-evoked myogenic responses in patients with an absence of click-evoked vestibulocollic reflexes. Clin Neurophysiol 2002;113:305–309.
34. Deriu F, Tolu E, Rothwell JC. A short latency vestibulomasseteric reflex evoked by electrical stimulation over the mastoid in healthy humans. J Physiol 2003;553:267–279.
35. De Waele C, Tran Ba Huy P, Diard JP, Freyss G, Vidal PP. Saccular dysfunction in Meniere's patients. A vestibular-evoked myogenic potential study. Ann NY Acad Sci 1999;871:392–397.
36. Murofushi T, Matsuzaki M, Takegoshi H. Glycerol affects vestibular evoked myogenic potentials in Meniere's disease. Auris Nasus Larynx 2001;28:205–208.
37. Young YH, Huang TW, Cheng PW. Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Arch Otolaryngol Head Neck Surg 2003;129:815–818.
38. Seo T, Node M, Yukimasa A, Sakagami M. Furosemide loading vestibular evoked myogenic potential for unilateral Meniere's disease. Otol Neurootol 2003;24:283–288.
39. Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG. Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis. An indicator of inferior vestibular nerve involvement? Arch Otolaryngol Head Neck Surg 1996;122:845–849.
40. Chen CW, Young YH, Wu CH. Vestibular neuritis: three-dimensional videonystagmography and vestibular evoked myogenic potential results. Acta Otolaryngol 2000;120:845–848.
41. Ochi K, Ohashi T, Watanabe S. Vestibular-evoked myogenic potential in patients with unilateral vestibular neuritis: abnormal VEMP and its recovery. J Laryngol Otol 2003;117:104–108.
42. Murofushi T, Monobe H, Ochiai A, Ozeki H. The site of lesion in "vestibular neuritis": study by galvanic VEMP. Neurology 2003;61:417–418.
43. Lu YC, Young YH. Vertigo from herpes zoster oticus: superior or inferior vestibular nerve origin? Laryngoscope 2003;113:307–311.
44. Matsuzaki M, Murofushi T. Vestibular evoked myogenic potentials in patients with idiopathic bilateral vestibulopathy. Report of three cases. J Otorhinolaryngol 2001;63:349–352.
45. Murofushi T, Matsuzaki M, Mizuno M. Vestibular evoked myogenic potentials in patients with acoustic neuromas. Arch Otolaryngol Head Neck Surg 1998;124:509–512.
46. Takeichi N, Sakamoto T, Fukuda S, Inuyama Y. Vestibular evoked myogenic potential (VEMP) in patients with acoustic neuromas. Auris Nasus Larynx 2001;28(suppl):39–41.
47. Ushio M, Matsuzaki M, Takegoshi H, Murofushi T. Click-and short tone burst evoked myogenic potentials in cerebellopontine angle tumors. Acta Otolaryngol (Suppl) 2001;545:133–135.
48. Patko T, Vidal PP, Vibert N, Tran Ba Huy P, de Waele C. Vestibular evoked myogenic potentials in patients suffering from an unilateral acoustic neuroma: a study of 170 patients. Clin Neurophysiol 2003;114:1344–1350.
49. Matsuzaki M, Murofushi T, Mizuno M. Vestibular evoked myogenic potentials in acoustic tumor patients with normal auditory brainstem responses. Eur Arch Otorhinolaryngol 1999;256:1–4.
50. Chen CW, Young YH, Tseng HM. Preoperative versus postoperative role of vestibular-evoked myogenic potentials in cerebellopontine angle tumor. Laryngoscope 2002;112:267–271.
51. Tsutsumi T, Tsunoda A, Noguchi Y, Komatsuzaki A. Prediction of the nerves of origin of vestibular schwannomas with vestibular evoked myogenic potentials. Am J Otol 2000;21:712–715.
52. Tsutsumi T, Komatsuzaki A, Noguchi Y, Tokano H, Kitamura K. Postoperative vestibular-evoked myogenic potentials in cases with vestibular schwannomas. Acta Otolaryngol 2001;121:490–493.
53. De Waele C, Meguenni R, Freyss X, et al. Intratympanic gentamicin injections for Meniere disease: vestibular hair cell impairment and regeneration. Neurology 2002;59:1442–1444.
54. Minor LB, Solomon D, Zinreich JS, Zee DS. Sound-and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. Arch Otolaryngol Head Neck Surg 1998;124:249–258.
55. Watson SR, Halmagyi GM, Colebatch JG. Vestibular hypersensitivity to sound (Tullio phenomenon): structural and functional assessment. Neurology 2000;54:722–728.
56. Minor LB, Cremer PD, Carey JP, Della Santina CC, Streubel SO, Weg N. Symptoms and signs in superior canal dehiscence syndrome. Ann NY Acad Sci 2001;942:259–273.
57. Streubel SO, Cremer PD, Carey JP, Weg N, Minor LB. Vestibular-evoked myogenic potentials in the diagnosis of superior canal dehiscence syndrome. Acta Otolaryngol (Suppl) 2001;545:41–49.
58. Minor LB, Carey JP, Cremer PD, Lustig LR, Streubel SO, Ruckenstein MJ. Dehiscence of bone overlying the superior canal as a cause of apparent conductive hearing loss. Otol Neurootol 2003;24:270–278.
59. Mikulec AA, McKenna MJ, Ramsey X, et al. Superior semicircular canal dehiscence presenting as conductive hearing loss without vertigo. Otol Neurootol 2004;25:121–129.
60. Brantberg K, Lofqvist L, Fransson PA. Large vestibular evoked myogenic potentials in response to bone-conducted sounds in patients with superior canal dehiscence syndrome. Audiol Neurootol 2004;9:173–182.
61. Brantberg K, Bergenius J, Tribukait A. Vestibular-evoked myogenic potentials in patients with dehiscence of the superior semicircular canal. Acta Otolaryngol 1999;119:633–640.
62. Halmagyi GM, Colebatch JG, Curthoys IS. New tests of vestibular function. Baillieres Clin Neurol 1994;3:485–500.
63. Ochi K, Ohashi T, Kinoshita H. Acoustic tensor tympani response and vestibular evoked myogenic potential. Laryngoscope 2002;112:2225–2229.
64. Murofushi T, Shimizu K, Takegoshi H, Cheng PW. Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. Arch Otolaryngol Head Neck Surg 2001;127:1069–1072.
65. Sartucci F, Logi F. Vestibular-evoked myogenic potentials: a method to assess vestibulo-spinal conduction in multiple sclerosis patients. Brain Res Bull 2002;59:59–63.
66. Versino M, Colnaghi S, Callieco R, Bergamaschi R, Romani A, Cosi V. Vestibular evoked myogenic potentials in multiple sclerosis patients. Clin Neurophysiol 2002;113:1464–1469.
67. Bandini F, Beronio A, Ghiglione E, Solaro C, Parodi RC, Mazzella L. The diagnostic value of vestibular evoked myogenic potentials in multiple sclerosis. J Neurol 2004;251:617–621.
68. Dillmann U, Heide G, Wollenberg I, et al. Der Klick-evozierte vestibulokolläre Reflex bei topologisch definierten Hirnstammläsionen. Z Elektroencephalogr Elektromyogr 1997;28:146–150.
69. Chen CH, Young YH. Vestibular evoked myogenic potentials in brainstem stroke. Laryngoscope 2003;113:990–993.
70. Wu CC, Young YH, Ko JY. Effect of irradiation on vestibular evoked myogenic potentials in nasopharyngeal carcinoma survivors. Head Neck 2003;25:482–487.
71. Itoh A, Kim YS, Yoshioka K, et al. Clinical study of vestibular-evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. Acta Otolaryngol (Suppl) 2001;545:116–119.