B-ENT, 2008, **4**, Suppl. 8, 39-43

Vestibular evoked myogenic potentials

S. Castelein, N. Deggouj, F. Wuyts and M. Gersdorff

Cliniques universitaires Saint-Luc, ENT Department, Bruxelles, Belgium

Key-words. Galvanic; tone burst; vestibular evoked myogenic potential; vestibulocollic

Abstract. *Vestibular evoked myogenic potentials.* The testing of Vestibular Evoked Myogenic Potentials has become a well-established approach for the exploration of sacculocollic pathways. Two types of tests have been described: VEMP induced by high intensity sound (click or tone burst) and VEMP induced by galvanic stimulation. We describe a method for recording the VEMP using both types of stimulation, and how to interpret them, and we report several clinical applications. Each of these techniques activates the vestibular afferents in different ways, and is a complementary tool for testing vestibular function at the level of the saccule or the inferior vestibular nerve.

Introduction

Patients suffering from vertigo are usually examined using caloric and rotation tests. These tests are essential, but they only inform the clinician about one of the five pairs of labyrinthic receptors, and they fail to establish a full picture of the functionality of the otolith receptors.

Vestibular evoked myogenic potential (VEMP) testing has become a well-established approach for the exploration of the sacculocollic pathways. This test has several advantages compared with other otolithic tests: it studies saccular function and the sacculocollic pathways selectively and never compensates. It requires the standard equipment for auditory evoked potentials to deliver the triggered sounds as well as EMG equipment.

We describe two types of test: VEMP induced by high-intensity sound (click or tone burst) and VEMP induced by galvanic stimulation. Both of these techniques activate the vestibular afferents in different ways and they are complementary.

VEMP induced by loud sound

Von Bekesy first discovered the sensitivity of the human vestibule to loud sound in 1935 and several studies since have confirmed this. It has been reported that a high-intensity sound induces an inhibitory impulse in the ipsilateral sternocleidomastoid (SCM) muscle.[1] Intense monaural clicks and tones give rise to short latency biphasic positive-negative responses (p13n23) in the ipsilateral SCM muscles if they are activated tonically.[2] These responses are preserved in subjects with severe sensorineural hearing loss and absent in patients who have undergone selective vestibular neurectomy and are therefore vestibular-dependent.[3] Several reports based on animal studies have suggested that these responses originate from the sacculus. Primary saccular afferents, due to their proximity to the footplate of the stapes, may be mechanically stimulated by loud sound[4] or stimulated by means of Eddy currents. It has been reported that the VEMP may travel along the hypothesised response pathway from the vestibular sacculus to the inferior vestibular nerve, vestibular nucleus, lateral (Deiter's) nucleus, and lateral vestibulospinal tract to the SCM muscle.[1]

Methods

VEMPs can be evoked by two kinds of auditory stimuli: clicks (0.1 ms, 100 dB nHL) and tone bursts (250, 500, 750 or even 1000 Hz, tone bursts of 500 Hz are often used, 95 dB nHL, rise/fall time = 2 ms, plateau time = 2 ms, repetition rate = 5.1 Hz). These stimuli are presented through a headphone, and the VEMPs are recorded on the SCM muscle ipsilateral to the stimulated ear.

Electromyographic activity in the SCM muscle is recorded using surface electrodes on the upper half of the SCM muscle with a reference electrode on the upper edge of the sternum and a ground electrode on the forehead. It is essential for the SCM to be in a strong contracted state during the recording. Several methods can be used for this purpose. The seated subject may be required to flex the

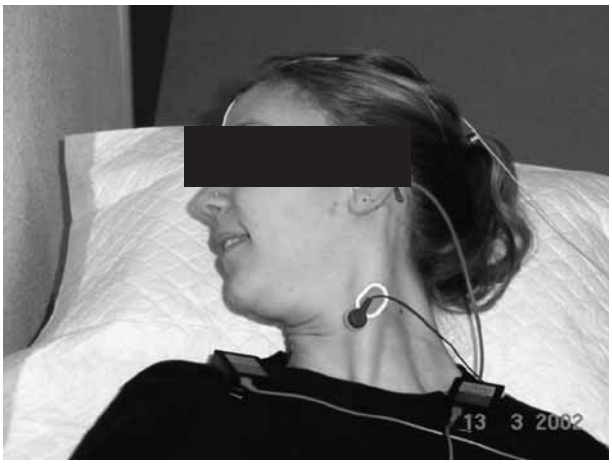

Figure 1
Electrode placement and contraction of the SCM muscle. Insert earphones are used. (Picture UZA).

head approximately 30 degrees forward and rotate it approximately 90 degrees to one side. Alternatively, patients can be placed in a supine position and be instructed to turn and hold their heads as far as possible toward the side contralateral to the stimulated ear to activate the ipsilateral SCM muscle (Figure 1). The vestibulospinal pathways have an inhibitory action on the SCM, and the VEMP records result from a diminution of the EMG activity of the SCM induced by the sound stimulation. EMG responses are amplified, band-pass filtered (20 Hz to 2 kHz), and averaged over a series of approximately 128 stimuli. Each recording should be repeated twice.

Two types of waves are recorded for the SCM muscle ipsilateral to the stimulated ear: short latency waves and long latency waves. The short latency waves (Figure 2) are composed of a first positive wave with a mean latency of 12 ms (p13), followed by a negative wave with a mean latency of 20ms (n23).

These waves originate from the activation of the sacculospinal pathways. Stimulus intensity largely determines the response, as can be seen in Figure 3. The long latency waves appear with mean latencies of 30 ms (n34) and 44 ms (p44) respectively. They originate from cochlear afferents. These last waves appear to be inconsistent, and therefore have no potential clinical application.[5]

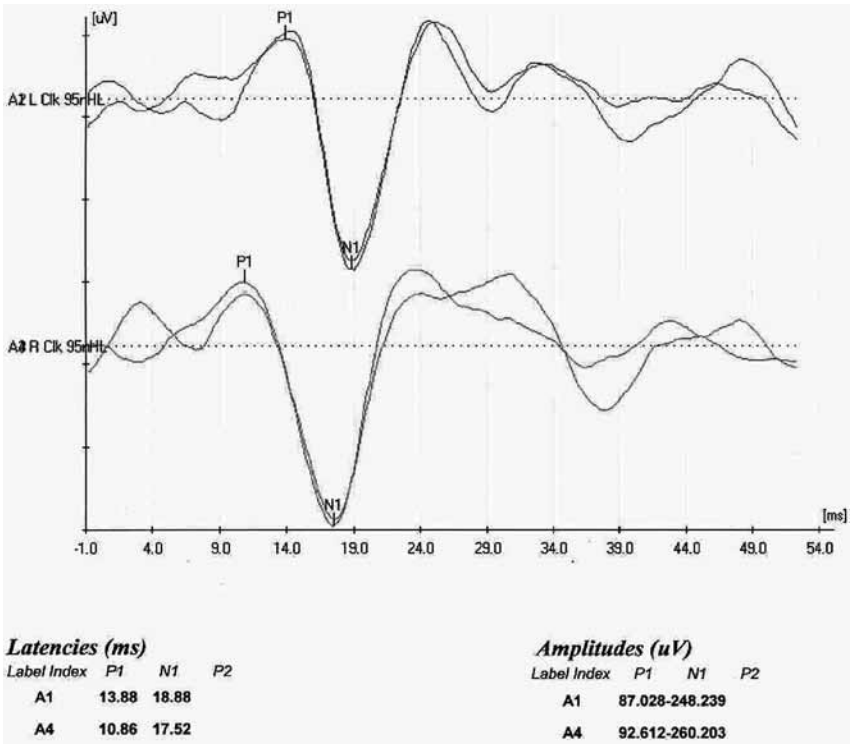

Figure 2
Click-induced VEMPs: the short latency waves are composed of a first positive wave p13 (p1), followed by a negative wave n23 (n1).

Results

VEMP responses are present in all normal subjects under the age of 60.[4] After the sixth decade the amplitude of the reflex decreases progressively by 25-30% per decade, but there are no differences in response amplitudes between the sexes. The absence of VEMP on one side indicates a disorder in the ipsilateral sacculospinal pathway and/or the mobility of the footplate.

It has been suggested that the most cautious interpretation of the potentials seems to be their ability to identify the asymmetry between left and right sides and

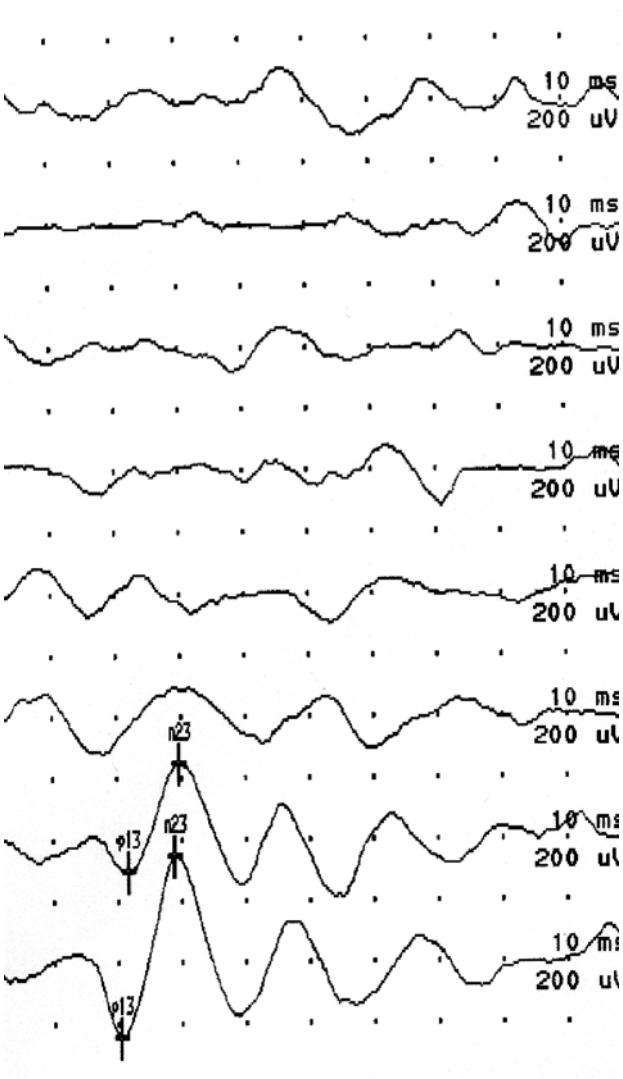

Figure 3
VEMP responses as a function of stimulus intensity (500 Hz tone burst). From top to bottom: 50, 60, 65, 70, 75, 80, 85 and 90 dB nHL. A clear p13-n23 waveform is seen only at intensities above 80 dB.

therefore to suggest the likely side of the pathology.[1] The tracings are too non-specific to allow for a discrimination of the underlying pathology or to suggest a diagnosis. Furthermore, the amplitude of the p13n23 waves varies widely between patients, making it difficult to use this parameter for clinical evaluation. The amplitude of the wave also depends on muscular tension during recording, increasing the difficulties for interpretation and comparison.

Welgampola *et al.*[4] provided reference values in 2001 for subjects below the age of 60.

Since the relationship between the reflex amplitude and the tonic EMG activity has been found to be linear, raw amplitudes were divided by the mean rectified EMG activity for 20 ms prior to stimulus onset to correct for differences in activation (corrected reflex amplitude). All subjects had corrected click-evoked reflex amplitudes of at least 0.5. Side-to-side differences in corrected reflex amplitudes were expressed as an "asymmetry ratio" (AR) calculated as AR = 100 (Al-Ar)/(Al+Ar), and a "side-to-side ratio" (SR), which was Al/Ar. In subjects under the age of 60 years, ARs of up to 35% and SRs of up to 2.1 were found to be normal for click-evoked responses.

The mean p13n23 peak latencies were 12.0 and 20.3 ms, respectively, for clicks. Side-to-side differences in the p13 latency were always less than 3.6 ms and less than 3.8 ms in the n23 latency. Given the strong dependency of amplitude on the SCM contraction state, it is highly advisable to measure the mean rectified voltage during the VEMP recording. It is only if this is done that the differences between left and right VEMP amplitudes can be attributed to a genuine sacculocollic pathway lesion on one side. Otherwise, the difference in VEMP amplitude may be induced by a difference in muscle contraction between the left and right SCM. Furthermore, there must be no air bone gap since this obviously does not deliver the appropriate stimulus to the saccule.

When comparing click and tone-burst evoked responses, Welgampola *et al.*[3] discovered that amplitudes of responses were not significantly different. On the contrary, the average p13 and n23 latencies of responses to an optimal tone burst stimulus were 13.1 and 22.8 ms, both occurring significantly later than the corresponding latencies for clicks.

The clinical application of VEMP has been reported for patients with acoustic tumours, Ménière's disease and vestibular neurolabyrinthitis. De Waele *et al.*[5]

showed that VEMPs were absent in 54% of 60 patients with Ménière's disease. However, it has also been demonstrated that VEMP can be augmented in cases of saccular hydrops.[6]

Since most acoustic tumours are thought to originate from the inferior vestibular nerve, VEMP might be the most useful test for the detection of these tumours.[1] Chen *et al.*[7] reported that 89% of patients with CPA tumours had no VEMP on the affected side. Matsuzaki *et al.*[8] reviewed the files of 33 patients with acoustic tumours, finding two cases with normal ABR and absent VEMP.

They assumed that VEMP might be more sensitive for detecting early acoustic tumours involving only the inferior vestibular nerve. De Waele *et al.*[5] also showed that, after surgery for these tumours, VEMPs were definitely abolished, thereby demonstrating the absence of central compensation for the reflex.

In the case of vestibular neuritis, Ochi *et al.*[1] showed that VEMPs were absent in two cases out of three. Similar results were obtained by De Waele *et al.*,[5] and they concluded that the viral lesion affects the inferior vestibular nerve in two-thirds of cases.

VEMP evoked by galvanic stimulation

Watson and Colebatch[9] showed in 1998 that galvanic stimulation applied to the mastoid process also evoked myogenic responses in the SCM, and that these responses disappeared after selective vestibular nerve section. They therefore seemed to be of vestibular origin, like those evoked by loud sound. Watson *et al* [9] suggested that galvanic stimulation stimulated the most distal portion of the inferior vestibular nerve ("spike trigger zone"), while the sound stimulation acted directly on the saccular receptor. Accordingly, Murofushi *et al.*[10] showed that galvanic-evoked myogenic responses in the SCM are useful in differentiating labyrinthine lesions from retrolabyrinthine lesions in patients with an absence of VEMP evoked by clicks.

Methods

A transmastoid galvanic stimulation of 4 mA and 2 ms is used, and the VEMPs are recorded on the ipsilateral SCM using the method described above for VEMPs evoked by loud sound (the SCM must be tonically activated). With this method, there is often a problem with stimulus artefact, and a technique of trace subtraction has to be used to minimise it:[9] the average EMG when stimulation is applied during relaxation consists of stimulus artefact only, while the average during tonic activation consists of stimulus artefact plus the reflex response; the first trace is subtracted from the second to cancel out the artefact, and to obtain the reflex response alone.

A biphasic response similar to the one observed with loud sound stimulation is observed on the SCM ipsilateral to the galvanic stimulation occurring at similar latency. In addition, a biphasic negative-positive (n12p20) response is observed in the other SCM. Late responses can also be seen, but these are not of vestibular origin.

Results

Welgampola *et al.*[4] have also studied VEMP induced by galvanic stimulation and have provided reference values. In this study, all patients below the age of 60 had detectable reflexes with corrected reflex amplitudes of at least 0.3 and a decrease in reflex amplitude was seen after the seventh decade. These authors propose the following reference values for side-to-side differences in corrected reflex amplitudes: for subjects below the age of 60 years, an AR of up to 41% and an SR of up to 2.4. The mean p13n23 peak latencies were 12.1 and 20.2 ms respectively.

Clinical application of VEMP induced by galvanic stimulation was reported to distinguish labyrinthine from retrolabyrinthine lesions in patients with an absence of VEMP evoked by loud sounds. Murofushi *et al.*[10] recorded galvanic-evoked VEMPs in patients with an absence of VEMP evoked by clicks. All patients who were diagnosed as having Ménière's disease or delayed endolymphatic hydrops showed normal galvanic-evoked responses on the affected side, while 88% of patients who were diagnosed as having cerebellopontine angle tumours showed no response or decreased responses, even to galvanic stimulation.

Conclusion

VEMP testing is a complementary tool for assessing vestibular function at the level of the saccule or the inferior vestibular nerve.

References

1. Ochi K, Ohashi T, Nishino H. Variance of vestibular-evoked myogenic potentials. *Laryngoscope*. 2001;111:522-527.
2. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic

reflex. *J Neurol Neurosurg Psychiatry*. 1994;57:190-197.
3. Welgampola MS, Colebatch JG. Characteristics of tone burst-evoked myogenic potentials in the sternocleidomastoid muscles. *Otol Neurotol*. 2001;22:796-802.
4. Welgampola MS, Colebatch JG. Vestibulocollic reflexes: normal values and the effect of age. *Clin Neurophysiol*. 2001;112:1971-1979.
5. de Waele C, Huy PT, Diard JP, Freyss G, Vidal PP. Saccular dysfunction in Meniere's disease. *Am J Otol*. 1999;20:223-232.
6. Young YH, Wu CC, Wu CH. Augmentation of vestibular evoked myogenic potentials: an indication for distended saccular hydrops. *Laryngoscope*. 2002;112:509-512.
7. Chen CW, Young YH, Tseng HM. Preoperative versus postoperative role of vestibular-evoked myogenic potentials in cerebellopontine angle tumor. *Laryngoscope*. 2002;112:267-271.
8. 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.
9. Watson SRD, Colebatch JG. Vestibulocollic reflexes evoked by short-duration galvanic stimulation in man. *J physiol*. 1998;513:587-597.
10. Murofushi T, Takegoshi H, Ohki M, Ozeki H. Galvanic-evoked myogenic responses in patients with an absence of click-evoked vestibulo-collic reflexes. *Clin Neurophysiol.* 2002; 113:305-309.

Sara Castelein, M.D.
Cliniques universitaires Saint-Luc
ENT Department
Avenue Hippocrate 10
B-1200 Bruxelles, Belgium
Tel.: 32.2.764.19.42
Fax: 32.2.764.89.35
E-mail: saracastelein@yahoo.com