Otolaryngology–Head and Neck Surgery (2006) 135, 286-290

ORIGINAL RESEARCH

# The application of vestibular-evoked myogenic potentials in otoneurosurgery

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**OBJECTIVE:** To evaluate the applicability of vestibular-evoked myogenic potentials (VEMPs) in the diagnostics, intraoperative monitoring, and postoperative follow-up of patients in otoneurosurgery.

**STUDY DESIGN:** A prospective study of patients who underwent either cochlear implantation (CI, n = 18) or were diagnosed with an acoustic neuroma (AN, n = 9) or with neuro(micro)vascular compression of the VIIIth nerve (NVC, n = 27) in the period 2002 to 2004. The follow-up was 1 year for all patients.

**SETTING:** A tertiary-referral unit.

**RESULTS:** VEMPs could be recorded in 64% of all patients before CI and in 22% after surgery. The patients with AN had normal VEMPs in 22% of all cases when first diagnosed. Normal VEMPs were found in 37% of those patients with NVC. From the 5 AN patients who had to be operated, only 1 had intact VEMPs after surgery. In contrast, after microvascular decompression all patients (4) had normal VEMPs.

**CONCLUSIONS:** VEMPs are helpful in diagnosing patients with vertigo to better identify saccular defects. They are highly sensitive in the early diagnosis of retrocochlear lesions.

**SIGNIFICANCE:** VEMPs can help to reliably identify patients with a retrocochlerar lesion at an early stage and can be used in intraoperative, neurophysiological monitoring.

**EBM rating: C-4**



The vestibular-evoked myogenic potentials (VEMPs) have been described to be electromyographic (EMG) responses of the sternocleidomastoid muscle (SCM) upon high-level acoustic stimulation of the saccule. This so-called vestibulocollic reflex can be used in vestibular testing of otolith (saccular) function.[1,2] It was shown that VEMPs can also be elicited in subjects with severe sensorineural hearing loss[1] because they are not associated with the cochlear (hearing) function.[3] VEMPs disappear after vestibular deafferentation.[1] The inferior vestibular nerve (IVN) carries the information from the saccule to the vestibular nuclei[2] so that either a saccular loss or any interference with the IVN (eg, compression by a tumor, a contacting artery, and any other disease process of the cerebello-pontine angle [CPA]) should possibly impair the VEMP response. It was therefore the aim of the present article to apply VEMP recordings in different types of labyrinthine and retrochlear pathology. Moreover, the IVN was electrically stimulated in otoneurosurgery of the CPA to compare the findings with those VEMPs elicited by acoustic stimulation. We examined to what extent VEMPS contribute to audiovestibular diagnostics of neurotological patients and in intraoperative monitoring during CPA surgery as suggested earlier.[4,5]

## MATERIALS AND METHODS

### Patients and Study Protocol

In a prospective study, 54 patients (26 women, 28 men; age range 18-62 years, mean 43) were included from 2002 to 2004. All patients gave their informed consent to participate in the study. The patients were diagnosed with (1) progressive hearing loss suitable for cochlear implantation (CI

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Supported by a grant from the Sonnenfeld Foundation, Berlin, Germany.

Presented at the Annual Meeting of the American Academy of Otolaryngology–Head and Neck Surgery, New York, NY, September 19-22, 2004.

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0194-5998/$32.00 
doi:10.1016/j.otohns.2006.03.006

**Table 1**
**Audiovestibular test battery (in addition to VEMP testing) applied in the AN group (IM, intrameatal location of the AN "watch-and-wait" subgroup; IM & EM, intra/extrameatal location of the AN surgery subgroup) and NVC group**

| Audiovestibular test | AN group (n = 9) IM (n = 4) | AN group (n = 9) IM & EM (n = 5) | NVC group (n = 27) |
|---|---|---|---|
| Pure-tone audiometry (mean hearing loss) | 5 | 15 | 10 |
| Stapedial reflex testing | | | |
| Normal | 1 | 1 | 9 |
| Lacking | 3 | 4 | 18 |
| TEOAE recording | | | |
| Normal | 0 | 1 | 13 |
| Lacking | 4 | 4 | 14 |
| ABR recording | | | |
| Normal | 0 | 2 | 14 |
| Pathological | 4 | 3 | 13 |
| VOR testing | | | |
| Normal | 3 | 3 | 23 |
| Pathological | 1 | 2 | 4 |

The mean hearing loss (in dB) is given as sum of hearing losses at 2, 3, 4 kHz divided by 3; the stapedial reflexes were "lacking" when absent in more than half of all frequencies tested; transiently evoked otoacoustic emissions (TEOAE) were "lacking" when absent in more than 3 frequencies; ABR recording was "pathological" when the interaural, interpeak difference of J5 exceeded 0.2 milliseconds; and VOR testing was "pathological" when cold-water irrigation showed a 25% interaural difference.

group); (2) unilateral acoustic neuroma by MRI scanning for the first time, with symptoms of tinnitus and unsteadiness, tumbling (AN group); or (3) neurovascular compression of the VIIIth nerve by magnetic resonance angiography of the brain (MRA) scanning with reports of attacks, typically of rotational vertigo (NVC group).[6,7] The patients were examined by an ENT specialist (including otoscopy) and underwent routine audiovestibular testing (Table 1) before CT scanning (CI group), MRI scanning (AN group), or MRA scanning (NVC group). All patients in the CI group (n = 18) were implanted with Clarion 90k (Adv Bionics, Sylmar, CA).

Four patients in the AN group (n = 9) were put on a "watch-and-wait" list (MRI scanning each year, no surgical intervention unless their clinical symptoms deteriorated or the AN showed rapid growth toward the brainstem). Over the period of the study, these tumors remained within the internal auditory canal (IAC). The tumors in the remaining 5 patients of the AN group had an intra/extrameatal portion and had to be resected because of their size and impingement on the brainstem. They were operated on by using a retrosigmoid approach.

Twenty-three of the patients in the NVC group (n = 27) were given drug treatment (beta-blockers, carbamazepine, ie, "medical decompression") after confirmation of the diagnosis by evidencing an anterior inferior cerebellar artery (AICA) loop around the VIIIth cranial nerve by MRA. Four patients failed to respond to drug therapy and therefore underwent microvascular decompression of the VIIIth nerve by using IVALON (M-Pact, Eudora, Kansas, USA).

The study protocol was approved by our institutional review board. The patients gave their written, informed consent to participate in the study.

## VEMP Testing and Data Evaluation

VEMP testing for saccular function was performed as described earlier[2] by bone-conducted acoustic stimuli. In brief, the stimuli (85-dB SPL, 0.1 millisecond, 520 clicks/cycle) were delivered through a bone conductor, and the electromyogram of the SCM was recorded ipsilaterally (surface EMG electrodes were placed on the upper half of each SCM and reference/ground electrodes at the upper sternum and the central forehead). VEMP recordings were performed during the patients' first visit and 1 year after remission (Fig 1).

In those patients (n = 9) who underwent surgery of the CPA for AN removal (n = 5) or microvascular decompression (n = 4), intraoperative VEMP recording was performed by direct electrical stimulation of the exposed IVN (Fig 1). Before surgery, subdermal needle electrodes (Xomed Inc, Nashville, TN) were placed in the middle of the ipsi- and contralateral SCM (active) of each patient. The upper sternum was used again (as described earlier) as a reference, and the ground electrode was attached at the forehead. The electromyograms recorded measured from both muscles were amplified (sensitivity 500 μV), filtered (bandpass 30-

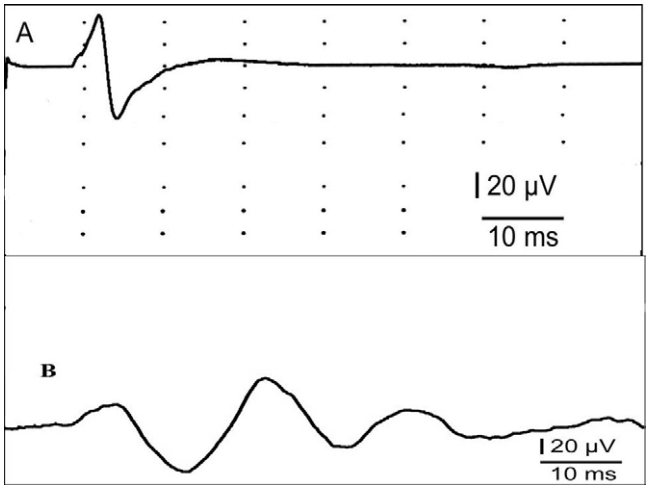


**Figure 1** (A) Averaged (90x) EMG reponse of the ipsilateral sternocleidomastoid muscle on electrical stimulation of the inferior vestibular nerve (threshold stimulus 0.4 mA). (B) Typical VEMP response on acoustic stimulation by bone conduction. (For details, see Materials and Methods and Results sections).

**Table 2**
**VEMP test results in all patients (CI, AN, NVC group) at the patient's first visit and after 1 year (acoustically elicited VEMPs by bone-conducted sound) and VEMPs by electrical stimulation of the IVN in operated patients (n = 9) (5 AN, 4 NVC patients)**

| VEMP test results (n = 54) | CI group (n = 18) | AN group (n = 9) | NVC group (n = 27) |
|---|---|---|---|
| First visit | | | |
| VEMPs elicitable | 12 | 2 | 17 |
| No VEMPs | 6 | 7 | 10 |
| One year follow-up | | | |
| VEMPs elicitable | 4 | 3 | 21 |
| No VEMPs | 14 | 16 | 6 |

For details, see Materials and Methods and Results sections.

2000 Hz) (Madison, WI, USA), averaged (n = up to 130), and stored (Viking IV; Nicolet). Only electrodes with an impedance of <1.5 kΩ were used for the recordings. During surgery, bipolar electric stimuli were applied to the IVN and superior part of the vestibular nerve by a concentric bipolar electrode (Xomed Inc, Nashville, TN). The current pulses, which were generated by the Viking IV system, ranged in amplitude from 0.4 to 1 mA, with a duration of 0.2 millisecond and a frequency of 4.7 Hz.

The follow-up period of the patients was 1 year. In the AN and NVC groups, the audiovestibular and VEMP test results from the healthy, unaffected side of each patient were taken as individual controls. The VEMP results of the CI group were compared with age- and sex-related, healthy, normal-hearing controls. Statistical evaluation was performed by a paired *t* test ($P < 0.01$) (SPSS 10.0; SPSS Inc, Chicago, IL).

## RESULTS

### The CI Group

In the CI group (n = 18), VEMP responses were absent in 36% of the patients before implantation. This increased to 78% after the operation (Table 2). Despite this significant increase of VEMP loss as a result of CI electrode insertion, the caloric VOR responses were found to be normal in 82% of all patients (ie, compared with the nonoperated ear).

The subjective complaints of the patients, which remained unchanged for at least 6 months postoperatively, were typically of "slipping" and "tumbling" sensations. A systematic workup of these symptoms (eg, Dizziness Handicap Inventors (DHI) was not undertaken.

### The AN Group

The AN subgroup (n = 4) with small intrameatal tumors ("watch and wait") showed statistically significant changes (compared with their contralateral, nonaffected ears) in stapedial reflex, Transitory Evoked Otoacoustic Emissions (TEOAE), and Auditory Brainstem Response Audiometry (ABR) recordings (Table 1). VEMPs were absent in all patients. These test results had not changed after 1 year (Table 2).

Preoperative testing in the AN subgroup with intra/extrameatal tumor extension (n = 5), who subsequently underwent AN removal, yielded statistically significant, pathological test results in stapedial reflex and TEOAE recordings (Table 1). VEMPs could not be evidenced in 3 patients preoperatively, but 1 patient showed normal VEMP recordings 1 year after surgery (Table 3 and Fig 1).

### The NVC Group

The NVC group (n = 27) showed statistically significant, pathological results for stapedial reflex testing only (Table 1). VEMPs could not be elicited in 63% (Table 2). This is statistically significantly different from the contralateral, nonaffected ears of this group. The 1-year follow-up showed no changes in VEMP testing in that subgroup of NVC patients who were treated by drug combinations (n = 23) (Table 2). However, all patients who underwent microvascular decompression surgery (n = 4) had normal VEMPs after 1 year (Table 3).

### Direct Electrical Stimulation of the IVN

In all patients (n = 9), VEMPs could be elicited by intraoperative electrical stimulation of the IVN (Fig 1) even if preoperative acoustic stimulation failed to elicit a VEMP response (n = 6) (Table 2). The VEMP amplitudes were linearly related to stimulus intensity (Fig 1). The mean latencies were 9.1 (±2.2) milliseconds for P13 and 13.2 (±2.3) milliseconds for N23. The EMG responses to electrical stimulation of the IVN were similar in amplitude but not in latency to those elicited by acoustic stimulation (Figs 1 and 2). No responses were observed in the contralateral SCM derivation. No EMG responses were found, neither ipsilaterally nor contralaterally, during electrical stimulation of the superior part of the vestibular nerve. In those cases in which the IVN was preserved during otoneurosurgery, VEMPs could be elicited by electrical stimulation throughout the surgical procedure and subsequently during postoperative testing with acoustic stimulation (Tables 2 and 3).

## DISCUSSION

The use of VEMP responses as a test of saccular function has become established over the last decade.[1] Our results show that saccular defects and/or the compression of the IVN by acoustic neurinoma (which frequently originates from this nerve [branch]) or an artery (eg, AICA) can induce a loss of acoustically elicited VEMPs on the diseased side.

In 36% of the examined CI group, preoperative absence of VEMPs was observed. Some of these patients also suf-

**Table 3**
**VEMP test result comparison of operated/nonoperated patients of the AN ("watch-and-wait" and "resection" subgroup) and NVC ("drug therapy" and "microvascular decompression, MVD" subgroup) groups on acoustic stimulation (preoperatively/postoperatively) on direct intraoperative stimulation of the IVN (n = number of patients)**

| | AN group | | NVC group | |
|---|---|---|---|---|
| VEMP test results | "Watch and wait" (n = 4) | "Resection" (n = 5) | "Drug therapy" (n = 23) | "MVD" (n = 4) |
| Acoustically elicited VEMPs | | | | |
| First visit (preoperative) | | | | |
| VEMPs elicitable | 4 | 2 | 17 | 4 |
| No VEMPs | 0 | 3 | 6 | 0 |
| One-year follow-up (postoperative) | | | | |
| VEMPs elicitable | 4 | 3 | 17 | 4 |
| No VEMPs | 0 | 2 | 6 | 0 |
| Electrically elicited VEMPs (intraoperatively) | | | | |
| VEMPs elicitable | | 5 | | 4 |
| No VEMPs | | 0 | | 0 |

For details, see Materials and Methods and Results sections.

fered from connexion 26–associated progressive, sensorineural hearing loss. A cochleosaccular defect has already been described by temporal bone histopathology.[8] The postoperative VEMP loss in the majority of the implanted patients is probably because of the surgical trauma after cochleostomy and electrode insertion. This finding could explain why some of the (elderly) patients have a longstanding history of "unsteadiness" and/or "tumbling sensations," which, despite having been reported in the literature,[9,10] has not yet been adequately explained. One group suspected the development of a delayed endolymphatic hydrops[11] and pointed out that the preoperative Vestibulo-Ocular Reflex (VOR) recordings have a limited relevance for predicting the occurrence of postoperative vertigo. Another group reported normal utricular function (the otolith organ consists of saccule and utricle) after cochlear implantation[12] and concluded that the primary origin of postoperative vertigo would be the preoperative meningitis that had destroyed the labyrinths of both sides per se. However, these authors did not investigate saccular function. Loss of saccular function appears difficult to rehabilitate, even after 1 year of training.[13] This could possibly explain the long-term vertiginous complaints of some implantees.[11]

Acoustic neuromas frequently originate from the IVN and have been reported to be accompanied by a preoperative VEMP loss.[5,14] Our small AN subgroup with purely intrameatal tumors of the IAC still had normal hearing but significantly changed VEMPs (4 pathological, 1 normal); in addition, audiovestibular test results (stapedial reflex, TEOAE and ABR) proved pathological. In contrast, the subgroup with intra/extrameatal tumors had complete VEMP loss but normal ABR recordings in 2 of the 5 cases. This finding is not in line with that of Tsutsumi et al,[15] who reported a close correlation of VEMP and ABR recordings in AN patients. However, the authors did not indicate the size and location of the tumors under investigation. The postoperative reappearance of VEMPs in 1 of our operated patients is indicative of a surgically preserved IVN. Besides the benefit to the patient, this is of particular interest as an intraoperative, neurophysiological monitoring method to guide the surgeon (eg, definition of the plane of dissection within the VIIIth nerve), similar to the technique described for the facial nerve.[16]

Neurovascular compression of the VIIIth nerve was confirmed by MRA scanning.[6] In brief, a definite contacting artery (AICA) had to be identified, either at the root entry zone of the brain stem or along the VIIIth nerve in the CPA or along (into) the nerve into the IAC.[7] The most interesting finding in this group was the lack of stapedial reflex response in 66% of the patients and the absence of VEMP responses in 63%. Nerve compression (as in the subgroup of intrameatal ANs above) seems to affect primarily the acustico-facial fibres and the IVN,[17] but the site of compression depends on the localization of the neurovascular contact.[7] The VEMPs of the patients operated on in the present study (microvascular decompression) appeared to have recovered completely. Only the VEMP responses of the subgroup of NVC patients with drug therapy ("medical decompression") remained unchanged after 1 year.

Intraoperatively, direct electrical stimulation of the IVN was performed successfully in all patients who were operated on (4 with AN resection and 5 with microvascular decompression). Interestingly, electrically evoked VEMPs could be observed in those patients who did not show VEMP responses to acoustic stimulation before surgery.[18] Similar to a previous report,[19] VEMP responses recovered postoperatively in 1 AN patient.

The electrical stimulus threshold for VEMP response was aproximately 0.4 mA. The intraoperative VEMP responses correlated well in shape but not in latencies (for

P13 and N23) with the responses to acoustic stimulation. The electrical stimulation of the IVN resulted in significantly decreased latencies because of the direct suprathreshold stimulation without the contributions of the vestibular-collic reflex arc (receptor excitation, synaptic transmission, and so on).

By using this approach, preservation of the IVN can be monitored successfully in neuroma resection[18] to prevent unintended postoperative sequelae, such as unsteadiness, dizziness in the dark, and other symptoms of otolith disorders[13] and to increase the surgeon's and patient's safety. Moreover, the cleavage plane and dissection strategy can be optimized[19] by this additional tool in intraoperative, neurophysiological monitoring.[16]

This article is dedicated to Vittorio Colletti, MD (Verona, Italy), a brilliant surgeon and pioneer in intraoperative neurophysiological monitoring.

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