SPINE Volume 33, Number 11, pp 1180–1184

Vestibular-Evoked Myogenic Potential (VEMP) to Evaluate Cervical Myelopathy in Human T-Cell Lymphotropic Virus Type I Infection

Lilian Felipe, ScM,* Denise Utsch Gonçalves, MD, PhD,* Marco Aurélio Rocha Santos, MD, PhD,* Fernando Augusto Proietti, MD, ScD,* João Gabriel Ramos Ribas, MD,† Anna Bárbara Carneiro-Proietti, MD, PhD,‡ and José Roberto Lambertucci, MD, PhD*

Study Design. Cross-seccional analysis.

Objective. To define the clinical usefulness of vestibular-evoked myogenic potential (VEMP) in detecting cervical medullar involvement related to human T-cell lymphotropic virus type 1 (HTLV-1) associated myelopathy/tropical spastic paraparesis (HAM/TSP).

Summary of Background Data. VEMP is generated by acoustic or galvanic stimuli, passing through the vestibulo-spinal motor tract, the spinal nerves and recorded by means of surface electrodes on the sternocleidomastoid muscle. HAM/TSP is a progressive inflammatory myelopathy with predominant lesions at the thoracic spinal cord level, although the cervical spine can be affected. VEMP may be of value to investigate cervical myelopathy.

Methods. Seventy-two individuals were evaluated of whom 30 HTLV-1 were seronegative and 42 HTLV-1 seropositive (22 asymptomatic, 10 with complaints of walking difficulty without definite HAM/TSP and 10 with definite HAM/TSP). VEMP was recorded using monaural delivered short tone burst (linear rise-fall 1 millisecond, plateau 2 milliseconds, 1 KHz) 118 dB NA, stimulation rate of 5 Hz, analysis time of 60 milliseconds, 200 stimuli, band pass filtered between 10 and 1.500 Hz.

Results. VEMP was normal in the seronegative group (30 controls). In the seropositive, abnormal VEMP was seen in 11 of 22 (50%) of the HTLV-1 asymptomatic carriers, in 7 of 10 (70%) of those with complaints of walking difficulty and in 8 of 10 (80%) of the HAM/TSP patients. In this last group, the pattern of response was different. No VEMP response was more frequent when compared with the HTLV-1 asymptomatic group (2-tailed *P*-value = 0.001).

Conclusion. VEMP may possibly be useful to identify patients with cervical myelopathy and to distinguish variable degrees of functional damage. Minor injury would be related to latency prolongation and major injury to no potential-evoked response.

Key words: electrophysiology, viral infections, spinal cord infection, vestibular-evoked myogenic potential, cervical spine, HTLV-1 associated myelopathy. **Spine 2008; 33:1180–1184**

Human T-cell lymphotropic virus type 1 (HTLV-1) was the first retrovirus linked to human disease and is transmitted sexually, by blood and from mother to child, mainly through breastfeeding.[1] The 2 major HTLV-I-associated diseases are adult T-cell leukemia/lymphoma and HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP).[1] The majority of the HTLV-1 infected persons remain healthy asymptomatic lifelong carriers. Of those who have the related diseases, about 5% may develop adult T-cell leukemia/lymphoma[2] and 1% HAM/TSP.[3] The concomitant occurrence of both diseases in a same individual has rarely been described.[4] What defines the predisposition to disease development has been associated mainly to host genetic polymorphisms.[5]

HAM/TSP is a slowly progressive myelopathy with pyramidal tract signs, sphincter disturbances, and mild sensory deficit.[6] Lesions predominate at the thoracic spinal cord level.[7] However, cervical abnormalities have also been described.[8,9] Diagnosis is based on clinical criteria and the detection of antibodies to HTLV-I in serum and/or in cerebrospinal fluid.[10] Strategies related to early diagnosis have epidemiological and therapeutic implications.[10]

Several reports on electrophysiological tests related to HAM/TSP have been published.[11,12] Vestibular-evoked myogenic potential (VEMP) may be a test of value to investigate cervical myelopathy in HAM/TSP, because as it evaluates cervical medullar functional response, it may lead to early diagnosis.

VEMP is a middle-latency evoked potential generated by a vestibulo-spinal muscle reflex recorded in the ipsilateral muscle, more commonly the sternocleidomastoid (SCM), in response to intense acoustic or galvanic stimulation of the saccule in the internal ear.[13] VEMP explores the sacculo-collic pathway which goes from the saccular macula through its primary vestibulo-spinal

From the *Faculty of Medicine of Federal University of Minas Gerais; †Sarah Hospital of Rehabilitation, Belo Horizonte; and ‡Hemominas Foundation, Minas Gerais State Centre of Hematology and Blood Transfusion, Brazil.

Research Ethic Committee of Federal University of Minas Gerais approved this study (approval number: ETIC 266/05).

Supported by CAPES/CNPq and Fapemig.

Acknowledgment date: August 8, 2007. Revision date: December 9, 2007. Acceptance date: December 18, 2007.

The manuscript submitted does not contain information about medical device(s)/drug(s).

No funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

Address correspondence and reprint requests to Denise Utsch Gonçalves, Tropical Medicine Post Graduation Program, Faculty of Medicine, Federal University of Minas Gerais, Av. Prof. Alfredo Balena, n° 190, sala 3005, Belo Horizonte, Minas Gerais, CEP 30100130, Brazil; E-mail: deniseg@medicina.ufmg.br

neurons to the lateral vestibular nucleus, the medial vestibular tract and finally to the motor neurons of the ipsilateral SCM.[13,14] The myogenic origin generates the biphasic morphology in a positive-negative wave form (P13-N23).[13] Damage to any of the structures related to the VEMP pathway results in abnormalities of these potential.[15,16] The VEMP applicability has been studied for many diseases of the internal ear and brain stem, including Meniere's disease,[17,18] vestibular neuritis[19], superior canal dehiscence syndrome,[20] basilar type migraine,[21] multiple sclerosis[16,22] spinocerebellar degeneration,[23] brain stem stroke,[24] cerebellopontine angle tumor,[25] acoustic neuroma,[26] benign paroxysmal positional vertigo,[27] and traumatic medullar lesion.[28] As regards VEMP parameters, the prolongation of latency has been related to neural damage.[15,16]

VEMP may evaluate the cervical spine, since the last synapsis of the arc-reflex from the ear to the muscle occurs in the second cervical spinal nerve.[28] The purpose of the present study was to evaluate VEMP in a group of HTLV-1 infected patients, with and without HAM/TSP to verify the clinical utility of this examination in detecting functional cervical vestibulo-spinal tract abnormality.

■ Materials and Methods

Subjects

The Interdisciplinary HTLV-1/2 Research Group (GIPH) was started in 1997 as an open prevalent cohort of HTLV infected individuals from Minas Gerais State, Brazil, aiming to study the natural history, clinical manifestations, and epidemiological aspects of this infection.[29] It has now over 700 individuals with HTLV-1 infection and negative controls who have been followed since 1997. The seroprevalence of HTLV-1 infection among eligible blood donors from Minas Gerais State is 0.32%.[30]

For this study, 72 participants from the GIPH cohort were submitted to VEMP. They were separated into 4 groups: (1) 30 HTLV-1 seronegative without neurologic complaints and with normal clinical examination (negative control); (2) 22 asymptomatic individuals infected by HTLV-1 (positive control); (3) 10 HTLV-1 seropositive individuals with walking complaints related to fatigue, but normal knee reflexes and extensor plantar responses; (4) 10 individuals with definite HAM/TSP.[10]

The inclusion criteria were vestibulo-coclear normality, defined by absence of complaints related to its function, normal hearing, normal external ear canal, and tympanic membrane and absence of head-shaking nystagmus.[31]

This research project was evaluated and approved by the Research Ethical Committee of Minas Gerais Federal University. Informed consent was obtained from all participants according to the Declaration of Helsinki.

Materials

The electromyographic activity was recorded from the upper half of the SCM muscle using surface electrodes, with a reference on the upper sternum and a ground electrode on the forehead. During each recording session, in the seated position, the subject was instructed to rotate the head towards the contralateral side of the tested ear to keep the SCM muscle under tension.[32]

The acoustic stimuli were short tone burst (1 KHz, 118 dBHL, rise-fall 1 millisecond, plateau 2 milliseconds) presented through a supra-aural earphone. The stimulation rate was 5 Hz and the analysis time for each response was 60 milliseconds; 200 responses were averaged for each run. Two consecutive runs were performed on the same ear to verify reproducibility. The electromyographic signals were amplified and band-pass filtered between 10 and 1.500 Hz (Bio-logic Program). The responses were recorded twice in both sides, to confirm replication.[33] The tests were conducted in a silent and acoustically treated environment.

The measured parameters were the peak latency (in ms) of the 2 early waves P13-N23; and the peak to peak amplitude of the P13-N23 waves with the asymmetry amplitude index below 34%, used to control muscular symmetry between sides.[34]

Data Analysis

The VEMP analysis was double-masked and done at the end of the study. The VEMP patterns of response to the acoustic stimulus considered in the analysis were "P13-N23 latency prolongation" or "no response". These parameters were compared among the groups. Statistical tests were performed using the statistical computer program EPI-INFO 6.04. Shapiro-Wilk, T-paired test, χ^2 test and Fisher's Exact Method were used for the statistical analysis. Differences or changes in P values less than 0.05 were considered to be statistically significant.

The accuracy of the examination was assessed calculating sensitivity and specificity. The true positives (disease present) were those with definite HAM/TSP[10] and the true negatives those from the negative control group, according to the inclusion criteria. In the analysis of the predictive value, the HTLV-1 prevalence considered was 0.32%.[30]

■ Results

The studied groups consisted of 13 men and 17 women not infected by HTLV-1 and 15 men and 27 women infected by HTLV-1 ($P = 0.9$). HTLV-1 seropositivity was defined by repeated reactive EIA tests (Ortho), with positive Western blot (Cambridge Biotech). The mean age was 48.9 among the HTLV-1 seropositive individuals and 45.2 among the seronegative individuals ($P = 0.7$). Table 1 presents P13-N23 latency comparison.

Regarding VEMP patterns of response among the groups, Table 2 shows that P13-N23 latency prolongation and no response were more frequent in the HTLV-1 groups, compared with the seronegative group. The same analysis using only the groups infected by HTLV-1 was done, considering the HTLV-1 asymptomatic carriers as the group with lower risk of VEMP abnormalities.

Table 1. VEMP P13-N23 Latencies (Means) in 30 HTLV-1 Seronegative and 42 Seropositive Individuals

	HTLV-1		
Variables	Negative (Mean ± SD) (N = 30)	Positive (Mean ± SD) (N = 42)	*P*
P13 latency (ms)	13.66 ± 0.52	14.84 ± 1.59	0.0002 (*t* test)
N23 latency (ms)	23.23 ± 0.95	24.60 ± 1.85	0.0004 (*t* test)

GIPH cohort study. Belo Horizonte, Brazil, 2007.

Table 2. VEMP P13-N23 Response in the HTLV-1 Seronegative and Seropositive Groups (N = 72)

	VEMP Response				
HTLV-1 Groups	Normal	P13-N23 Latency Prolongation	*P**	No Response	*P**
Seronegative (N = 30)	30	0	1	0	1
Asymptomatic (N = 22)	11 (50%)	10 (45%)	0.00003	1 (5%)	0.29
Complaint of walking difficulty (N = 10)	3 (30%)	3 (30%)	0.003	4 (40%)	0.0005
HAM/TSP (N = 10)	2 (20%)	3 (30%)	0.02	5 (50%)	0.0005

GIPH cohort study. Belo Horizonte, Brazil, 2007.
*2-tailed *P*-value.

It was seen that P13-N23 latency prolongation was more frequent among the asymptomatic carriers than among the patients complaining of walking difficulty or with definite HAM/TSP (Table 3). Conversely, no response after the acoustic stimulus was more frequent among the patients complaining of walking difficulty ($P = 0.04$) or with definite HAM/TSP ($P = 0.01$). Figure 1 displays the difference among the groups.

Considering the accuracy of VEMP to define cervical spinal cord neural dysfunction, the sensitivity was 80% (8 of 10) for definite HAM-TSP and the specificity was 100% (30 of 30).

■ Discussion

The lesion in the tissue of the central nervous system caused by HTLV-1 is associated to dense infiltrates of mononuclear cells, largely CD8+ lymphocyte response.[5] Thus, in terms of medullar injury, HAM/TSP is considered an inflammatory disease, in which cellular damage leads to demyelination.[5]

The increasing interest in VEMP is because of its property of eliciting neurogenic vestibular-evoked potentials in a noninvasive manner, it is easy to perform, to interpret and does not cause any discomfort to the patient.

Currently, the most important parameters considered in the VEMP analysis are P13-N23 latency and amplitude. However, amplitude may vary according to age,[35] muscle strength[34,36] and cochlear diseases.[17,18] In spite of having these variables controlled in the present study, possible bias related to amplitude, which could induce to false positive results, has discouraged its consideration as a parameter for defining abnormalities in neural conduction. Therefore, latency prolongation and no response were the parameters considered in the analysis of the VEMP. The quality of the test was guaranteed by the presence of a biphasic wave P13-N23 in VEMP of the 30 controls, which was validated by previously published literature[33–37] and by an amplitude asymmetry index below 34%, which controlled variations of muscular strength between the 2 tested sides.[34,36]

In terms of sensitivity, the HAM/TSP group was the gold standard for the presence of medullar injury.[10] A gradient of abnormal VEMP going from asymptomatic carriers (50%), patients with walking difficulty (70%) to patients with HAM/TSP (80%) was observed. Taking into account that VEMP is an objective test, this result identified variable degrees of functional abnormality (Table 2). Hence, this examination was showed to have sensitivity enough to detect early functional medullar damage. In case of therapeutic follow-up, VEMP may be of value to evaluate recovery.

Regarding VEMP parameters no response and latency prolongation, the most common finding in HAM/TSP patients was no response, while in those HTLV-1 infected individuals without myelopathy, this alteration was less frequent (Table 3). These data support the idea that a correlation between the degree of neural damage and a VEMP parameter may exist. Severe and massive neural lesions usually lead to a total failure of conduction, known as conduction block, with no response in VEMP.[15] Delayed-evoked response has been traditionally attributed to impaired focal conduction through focal demyelination, as in multiple sclerosis[16,22]

In terms of specificity, the accuracy of VEMP to define the absence of functional disease in the cervical spine was

Table 3. VEMP P13-N23 Response in HTLV-1 Asymptomatic Individuals, in HTLV-1 Infected Individuals With Complaint of Walking Difficulty Without HAM/TSP and in Individuals With HAM/TSP (N = 42)

	VEMP				
HTLV-1 Groups	Normal	P13-N23 Latency Prolongation	*P**	No Response	*P**
Asymptomatic (N = 22)	11 (50%)	10 (45%)	1	1 (5%)	1
Complaint of walking difficulty (N = 10)	3 (30%)	3 (30%)	1	4 (40%)	0.04
HAM/TSP (N = 10)	2 (20%)	3 (30%)	1	5 (50%)	0.01

GIPH cohort study. Belo Horizonte, Brazil, 2007.
*2-tailed *P*-value.

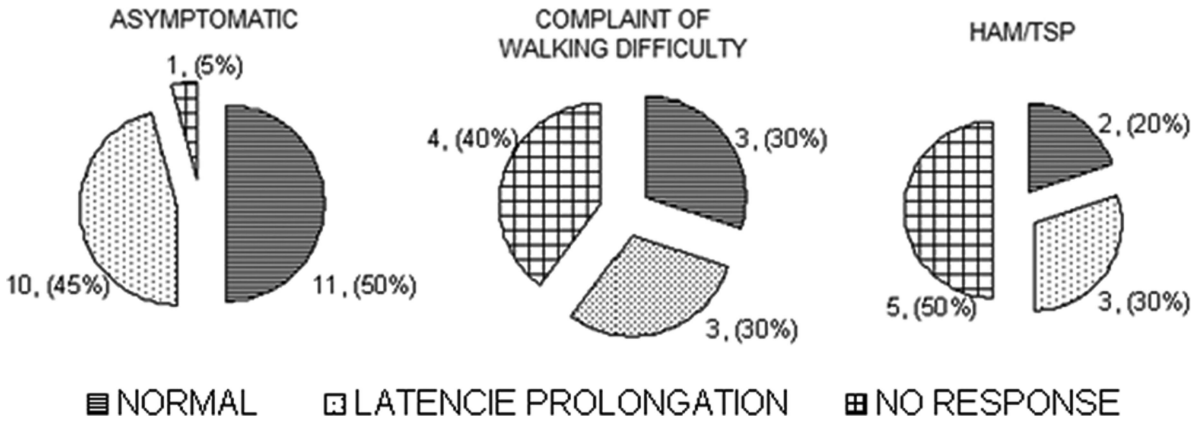

Figure 1. VEMP response distribution according to the HTLV-1 evaluated group. GIPH cohort study. N = 42. Belo Horizonte, Brazil, 2007.

considered satisfactory, being guaranteed by the evaluation of the seronegative control group (100% normal VEMP).

Concerning its predictive value, VEMP is worthless in terms of myelopathy diagnosis. As an evoked potential, the abnormality is the same for demyelization,[16,22] ischemia,[24] compression[25] or trauma.[28] The information derived from an abnormal VEMP is related to a defect in the neural conduction itself. Therefore, the positive predictive value must be analyzed in terms of the prevalence of the evaluated disease in the population. Conversely, the negative predictive value is important, because in case of medullar disease suspicion, a normal VEMP result can be the assurance of a functionally normal cervical spinal cord.

The value of VEMP has also been studied using galvanic electrical stimulation and the examination could provide information about the level of the spinal cord injury.[28] VEMP using acoustic stimulus evaluates the vestibulo-spinal tract up to the second cervical spinal nerve, which is a disadvantage when compared with galvanic stimulation that can evaluate the spinal cord as a whole.[13,28] The advantages of using acoustic stimuli are that the examination is more comfortable and safe to the patient, the equipment is more accessible and the technique easier, when compared with galvanic electrical stimulation.

In terms of cervical spine, the great majority of HAM/TSP patients (80%) had abnormal VEMP when compared with none in the HTLV-1 seronegative control group. Recent studies have supported the hypothesis that cervical spinal cord in HAM/TSP could be more affected than it was previously believed and that this alteration can be related to the severity of the myelopathy prognosis.[8,9]

Concluding, according to the present research, accuracy enough was found to indicate the clinical use of VEMP in the cervical functional spinal cord analysis. VEMP, as any other evoked potential modality, allows the detection of silent lesions, not displayed by neuroimaging or other neurophysiologic tests, opening the possibility of an early diagnosis in nontraumatic cervical myelopathy. Perhaps, vague walking complaints in an otherwise considered HTLV-1 healthy carrier indicate a spine cord lesion (Figure 1). Prospective studies are necessary to answer this question. For this reason, the participants of the GIPH cohort will from now on be submitted to VEMP each semester in order to correlate neurologic progression and VEMP parameters.

■ Key Points

- Human T cell lymphotropic virus type 1 (HTLV-1) is associated with myelopathy/tropical spastic paraparesis (HAM/TSP).
- HTLV-1 causes cervical myelopathy.
- VEMP is useful in HTLV-1 associated cervical myelopathy precocious diagnosis.

References

1. Blattner WA. Human T lymphotropic viruses and diseases of long latency. *Ann Intern Med* 1989;111:4–6.
2. Murphy EL, Hanchard B, Figueroa JP, et al. Modelling the risk of adult T-cell leukemia/lymphoma in persons infected with human T-lymphotropic virus type I. *Int J Cancer* 1989;43:250–3.
3. Kaplan JE, Osame M, Kubota H, et al. The risk of development of HTLV-1 associated myelopathy/tropical spastic paraparesis among persons infected with HTLV-1. *J Acquir Immune Defic Syndr* 1990;3:1096–101.
4. Goncalves DU, Guedes AC, Carneiro-Proietti ABF, et al. Simultaneous occurrence of HTLV-I associated myelopathy, uveitis and smouldering adult T cell leukaemia. *Intern J STD & AIDS* 1999;10:336–7.
5. Bangham CRM, Osame M. Cellular immune response to HTLV-1. *Oncogene* 2005;24:6035–46.
6. Osame M, Matsumoto M, Usuku K, et al. Chronic progressive myelopathy associated with elevated antibodies to human T-lymphotropic virus type I and adult T-cell leukemia like cell. *Ann Neurol* 1987;21:117–22.
7. Akizuki S, Nakazato O, Higuchi Y, et al. Necropsy findings in HTLV-I associated myelopathy. *Lancet* 1987;1:156–7.
8. Umehara F, Nagatomo S, Yoshishige K, et al. Chronic progressive cervical myelopathy with HTLV-I infection: variant form of HAM/TSP? *Neurology* 2004;63:1276–80.
9. Umehara F, Tokunaga N, Hokezu Y, et al. Relapsing cervical cord lesions on MRI in patients with HTLV-I associated myelopathy. *Neurology* 2006;66:289.
10. Castro-Costa CM, Araújo AQ, Barreto MM, et al. Proposal for diagnostic criteria of tropical spastic paraparesis/HTLV-I-associated myelopathy (TSP/HAM). *AIDS Res Hum Retroviruses* 2006;22:931–5.
11. Kakigi R, Shibasaki H, Kuroda Y, et al. Multimodality evoked potentials in HTLV-I associated myelopathy. *J Neurol Neurosurg Psychiatry* 1988;51:1094–6.
12. Suga R, Tobimatsu S, Kira J, Kato M. Motor and somatosensory evoked

potential findings in HTLV-1 associated myelopathy. *J Neurol Sci* 1999;167: 102–6.
13. Colebatch JG, Halmagy GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 1994;57: 190–7.
14. Akin FW, Murnane OD. Vestibular evoked myogenic potentials: preliminary report. *J Am Acad Audiol* 2001;12:445–52.
15. 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;127:1069–72.
16. Shimizu K, Murofushi T, Sakurai M, Halmagyi M. Vestibular evoked myogenic potentials in multiple sclerosis. *J Neurol Neurosurg Psychiatry* 2000; 69:276–7.
17. de Waele C, Tran Ba Huy P, Diart JP, Freyss G, Vidal PP. Saccular dysfunction in Meniere's disease. *Am J Otol* 1999;20:223–32.
18. Young YH, Huang TW, Cheng PW. Assessing the stage of Meniere′s disease using vestibular evoked myogenic potentials. *Arch Otolaryngol Head Neck Surg* 2003;129:815–8.
19. Cheng CW, Young YH, Wu CH. Vestibular neuritis: three-dimensional videonystagmography and vestibular evoked myogenic potential results. *Acta Otolaryngol* 2000;120:845–8.
20. Brantberg K, Bergenius J, Tribukait A. Vestibular-evoked myogenic potentials in patients with dehiscence of the superior semicircular canal. *Acta Otolaryngol* 1999;119:633–40.
21. Liao LJ, Young YH. Vestibular evoked myogenic potentials in basilar artery migraine. *Laryngoscope* 2004;114:1305–9.
22. 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.
23. Takegosh H, Murofushi T. Vestibular evoked myogenic potentials in patients with spinocerebellar degeneration. *Acta Otolaryngol* 2000;120: 821–4.
24. Chen CH, Young YH. Vestibular evoked myogenic potentials in brainstem stroke. *Laryngoscope* 2003;113:990–3.
25. Chen CW, Young YH, Tseng HM. Preoperative versus postoperative role of vestibular-evoked myogenic potentials in cerebellopontine angle tumor. *Laryngoscope* 2002;112:267–71.
26. 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;114:1344–50.
27. Akkuzu G, Akkuzu B, Ozluoglu LN. Vestibular evoked myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. *Eur Arch Otorhinolaryngol* 2006;263:510–7.
28. Iles JF, Ali AS, Savic G. Vestibular-evoked muscle responses in patients with spinal cord injury. *Brain* 2004;127:1584–92.
29. Proietti FA, Carneiro-Proietti ANF, Catalan-Soares BC, Murphy EL. Global epidemiology of HTLV-I infections and associated diseases. *Oncogene* 2005; 24:6058–68.
30. Proietti FA, Lima-Martins MV, Passos VM, Brener S, Carneiro-Proietti AB. HTLV-I/II seropositivity among eligible blood donors from Minas Gerais State, Brasil. *Vox Sang* 1994;67:77.
31. Perez VG, Gonzales EG, Garcia AP, Piqueral ADR, Morera CP, Perez HG. Vestibular evoked myogenic potential: a contribution to the vestibular physiology and pathology knowledge. Quantitative patterns in healthy subjects. *Acta Otorrinolaringol Esp* 2005;56:349–53.
32. Wang CT, Young YH. Comparison of the head elevation and rotation methods in eliciting vestibular evoked myogenic potentials. *Ear Hear* 2006;27: 376–81.
33. Wu CH, Murofushi T. The effect of click repetition rate on vestibular evoked myogenic potential. *Acta Otolaryngol* 1999;119:29–32.
34. Lim CL, Clouston P, Sheean G, Yiannikos C. Influence of voluntary EMG activity and click intensity on the vestibular click evoked myogenic potential. *Muscle Nerve* 1995;18:1210–3.
35. Ochi K, Ohashi T. Age-related changes in the vestibular-evoked myogenic potentials. *Otolaryngol Head Neck Surg* 2003;129:655–9.
36. Akin FW, Murnane OD, Panus PC, Caruthers SK, Wilkinson AE, Proffitt TM. The influence of voluntary tonic EMG level on the vestibular-evoked myogenic potential. *J Rehabil Res Dev* 2004;41:473–80.
37. Basta D, Todt I, Ernst A. Normative data for P1/N1-latencies of vestibular evoked myogenic potentials induced by air- or bone- conducted tone bursts. *Clin Neurophysiol* 2005;116:2216–9.