MEDICAL SCIENCE MONITOR

CLINICAL RESEARCH

e-ISSN 1643-3750
© Med Sci Monit, 2015; 21: 3172-3178
DOI: 10.12659/MSM.894958

Received: 2015.06.10
Accepted: 2015.07.02
Published: 2015.10.20

Brainstem Auditory Evoked Potential in HIV-Positive Adults

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEFG 1 Carla Gentile Matas
CDEF 1 Alessandra Giannella Samelli
BCDE 1 Rosanna Giaffredo Angrisani
BCDE 1 Fernanda Cristina Leite Magliaro
ACDEF 2 Aluísio C. Segurado

1 Department of Physical Therapy, Speech-language Pathology and Audiology, and Occupational Therapy, School of Medicine (FMUSP), University of São Paulo, São Paulo, SP, Brazil
2 Department of Infectious Diseases, School of Medicine (FMUSP), University of São Paulo, São Paulo, SP, Brazil

Thesis presented to Faculdade de Medicina, Universidade de São Paulo, in fulfillment for the title of Full Professor in the Department of Physical Therapy, Speech and Hearing Sciences and Occupational Therapy (Speech and Hearing Sciences Course), 2010

Corresponding Author: Carla Gentile Matas, e-mail: cgmatas@usp.br

Source of support: This research was supported by a grant from FAPESP (Foundation for Research Support of the State of São Paulo)

Background: To characterize the findings of brainstem auditory evoked potential in HIV-positive individuals exposed and not exposed to antiretroviral treatment.

Material/Methods: This research was a cross-sectional, observational, and descriptive study. Forty-five HIV-positive individuals (18 not exposed and 27 exposed to the antiretroviral treatment – research groups I and II, respectively – and 30 control group individuals) were assessed through brainstem auditory evoked potential.

Results: There were no significant between-group differences regarding wave latencies. A higher percentage of altered brainstem auditory evoked potential was observed in the HIV-positive groups when compared to the control group. The most common alteration was in the low brainstem.

Conclusions: HIV-positive individuals have a higher percentage of altered brainstem auditory evoked potential that suggests central auditory pathway impairment when compared to HIV-negative individuals. There was no significant difference between individuals exposed and not exposed to antiretroviral treatment.

MeSH Keywords: Acquired Immunodeficiency Syndrome • Evoked Potentials, Auditory, Brain Stem • HIV

Full-text PDF: http://www.medscimonit.com/abstract/index/idArt/894958

2823 5 — 36

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Background

The human immunodeficiency virus (HIV) is the etiologic agent of the Acquired Immune Deficiency Syndrome (AIDS), a syndrome characterized by significant deterioration of the immune system. The virus mainly affects the T CD_{4+} lymphocytes, causing their progressive decrease and consequently leading to opportunistic infections [1].

The incidence of hearing impairment in patients with HIV/AIDS ranges from 20% to 40% [2,3]. The literature reports a diffuse and progressive impairment of the central auditory nervous system (CANS), either due to a direct action of the virus on its structures or due to opportunistic infections [4].

Besides alterations in behavioral tests of central auditory abilities, brainstem auditory evoked potential (BAEP) alterations can be found in this population before the onset of clinical symptoms such as neurological and cognitive deficits [3,5–8].

The AIDS treatment currently most commonly used is combination therapy, also known as highly active antiretroviral therapy (HAART). This therapy consists of the combination of at least 3 drugs and monitoring of viral burden in blood plasma [9]. HAART leads to better prognosis for patients, with improved survival rate and, consequently, reduced mortality [10,11].

Despite the significant improvement of infection and disease in HIV-infected individuals, antiretroviral therapy can cause various adverse effects, such as the ototoxicity recently reported in the literature by case reports, as well as in longitudinal and transversal studies, using clinical data and/or hearing and electrophysiological tests [12–16].

Thus, the purpose of this study was to characterize the electrophysiological profile of the auditory system in HIV-positive individuals exposed and not exposed to antiretroviral treatment, with the use of BAEP, hypothesizing that the clinical evolution of the disease and, consequently, the use of potentially ototoxic medications, affect their hearing and cause impairment in the central auditory system.

Material and Methods

The current research consisted on a cross-sectional, observational and descriptive study conducted at the Laboratory for Hearing Research in Auditory Evoked Potentials of the Speech and Hearing Sciences Course of the School of Medicine of the University of São Paulo (FMUSP). This is the second article in a series of 3 that investigated the peripheral and central auditory pathways of HIV-positive individuals. The first published article reported the audiological manifestations in this same population [17].

The study was approved by the Ethics Committee for Analysis of Research Projects (CAPPesq) of the Clinical Board of Hospital das Clínicas and Faculdade de Medicina da Universidade de São Paulo under protocol number 1026/04. All participants signed a consent form.

The sample consisted of 75 subjects aged between 20 and 60 years, divided into 3 groups: Research Group I (RGI), composed of 18 HIV-positive individuals, 4 women and 14 men (mean age of 39.4 years), who were not exposed to antiretroviral treatment; Research Group II (RGII), composed of 27 HIV-positive individuals, 9 women and 18 men (mean age of 40.3 years), who were receiving antiretroviral therapy; and Control Group (CG), composed of 30 healthy subjects, 14 women and 16 men (mean age of 25.6 years).

Inclusion criteria for the 3 groups included pure-tone audiometry from normal to bilateral symmetric moderate hearing loss (conductive and sensorineural). Specific inclusion criteria for the RGI was: HIV positive (confirmed by serology) without exposure to antiretroviral treatment. Inclusion criteria for RGII included HIV positive (confirmed by serology), with exposure to antiretroviral therapy (Highly Active Antiretroviral Therapy or combination therapy) consisting of at least 3 of the following drugs: lamivudine, zidovudine, efavirenz, didanosine, nevirapine, lopinavir-r, tenofovir, stavudine, indinavir, abacavir, amprenavir, ritonavir, atazanavir. Specific inclusion criteria for the control group (CG), consisted of: healthy, no HIV (report on questionnaire and seronegative), no history of psychiatric or neurological diseases, and no auditory, language, or auditory processing complaints.

The following exclusion criteria were considered for all 3 groups: pure-tone audiometry from moderately severe to profound hearing loss, pregnancy, presence of active opportunistic infections, presence of any clinical and/or cognitive impairment that would impair audiological testing, history of otologic surgery, or disease uncorrelated to HIV.

Individuals who composed RGI and RGII were referred by the House of AIDS - Zerbini Foundation (Casa da AIDS – Fundação Zerbini) and the City Health Services Specialized in Sexually Transmitted Diseases (STD/AIDS) of the City of São Paulo Health Department.

The first procedure consisted of reading the medical history of the individual in order to obtain data regarding eligibility criteria, followed by interview to obtain data on complaints and/or presence of risk indicators for hearing loss.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

The analysis of patients' records (RGI and RGII) aimed at obtaining data related to HIV infection, exposure category, as well as the history of the use of antiretroviral drugs and other non-antiretroviral drugs with ototoxic potential.

Visual inspection of the external ear canal was subsequently performed using a Heine otoscopy to check for possible obstructions by cerumen or foreign body that would possibly interfere in the electrophysiological assessments.

Pure-tone audiometry was conducted using the audiometer model GSI-61 from Grason Stadler with TDH-50 supra-aural headphones. The results were classified as normal or alteration, with the following considered as normal: hearing thresholds lower than or equal to 20 dB HL for the frequencies tested [18].

The equipment used for research of Brainstem Auditory Evoked Potential (BAEP) was the Express Traveler Portable System, from Bio-Logic, with supra-aural headphones model TDH-39.

The BAEP was performed in an acoustically- and electrically-treated room. The skin surface was cleaned with abrasive paste, and the electrodes were fixed with electrolyte paste and microporous tape, and placed on the forehead (Fpz) and left and right mastoids (M1 and M2), according to the standard International Electrode System (IES) [19]. Impedance values of the electrodes were maintained below 5 kOhms.

The click stimulus with rarefied polarity was monaurally presented at 80 dBHL in a speed of 19.1 stimuli per second, and duration of 0.1 millisecond (ms), totaling 2000 stimuli per trace. Two recordings were obtained for each side (ipsilateral) to allow the reproduction of traces.

The values of absolute latencies of waves I, III and V, and interpeaks I–III, III–V, IV were analyzed.

BAEP results were classified as normal or alteration and the alteration were classified according to the location [20]: Low Brainstem (LB): increased latency values of waves III and V and/or interpeaks I-III and I-V; High Brainstem (HB): increased latency values of wave V and/or interpeaks I-V and III-V in the presence of normal absolute latencies for waves I and III; Low and High Brainstem (LB + HB): type LB and HB alterations are concurrently find on the same individual; Sensory Impairment (SI): absence of responses, compatible with hearing thresholds recorded in pure tone audiometry; Middle Ear Impairment (MEI): increased latency values of waves I, III, V and normal interpeaks I–III, III–V, I–V.

Component analysis of the BAEP was performed by the researcher and a second experienced researcher in the field of electrophysiology.

Results were referred to the home institution of individuals of RGI and RGII. In case of abnormal results, the individual was referred for ENT evaluation and was instructed to return for audiological reassessment after 3 months.

Statistical methods

BAEP findings were quantitatively and qualitatively analyzed. For quantitative data analysis (continuous data), mean and standard deviation were established for each assessment for each group. The Kruskal-Wallis test was used for comparisons of continuous data that did not follow a normal distribution. Chi-square (χ^2) and Fisher's exact test were used to compare categorical data (qualitative).

The probability (p) of less than 0.05 was considered to indicate statistical significance, except when a potential problem of multiple comparisons was identified. In this latter case, Bonferroni correction was used in analyses involving the comparison of scores between right and left ears and analyses that involved the between-group comparisons. In these cases, p was corrected to 0.001. When comparing the percentage of alterations in electrophysiological hearing assessments on the qualitative analysis, p was corrected to 0.005. When analyses were performed 2 by 2, p was corrected to 0.017.

All statistical analyses were calculated using SPSS 15 (Statistical Package for the Social Science) for Windows.

Results

In the quantitative data analysis, mean and standard deviation of latencies of waves I, III and V and interpeaks I–III, III–V and I–V obtained in the right and left ears in each group were separately calculated (Table 1). No statistically significant between-ears difference was observed.

Because no between-ears difference was found, the average latencies of waves I, III, V and interpeaks I–III, III–V, I–V of right and left ears were analyzed according to each group (CG, RGI and RGII). No statistically significant between-groups difference was observed, as seen in Table 2.

In the qualitative data analysis, a statistically significant between-groups difference ($p<0.001$) was found in the analysis of BAEP and audiometry alteration proportions of CG, RGI and RGII – with a higher percentage of alterations in groups of HIV-positive individuals not exposed (RGI) and exposed (RGII) to antiretroviral treatment (55.6% and 66.7%, respectively for BAEP and 27.7% and 48.1% for audiometry) when compared to the control group (13.3% for BAEP and 0% for audiometry) (Table 3).

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 1. Descriptive analysis of BAEP latencies of waves I, III, V and interpeaks I–III, III–V, I–V (in ms) for CG, RGI and RGII and right and left ears.

BAEP		I		III		V		I–III		III–V		I–V	
		RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
CG	M	1.6	1.5	3.8	3.7	5.7	5.7	2.2	2.2	2.0	2.0	4.1	4.1
	SD	0.1	0.1	0.2	0.1	0.2	0.2	0.2	0.1	0.4	0.4	0.4	0.4
	p	0.026		0.356		0.981		0.651		0.288		0.901	
RGI	M	1.7	1.7	4.0	3.9	6.0	5.9	2.3	2.2	2.0	2.0	4.3	4.2
	SD	0.3	0.1	0.3	0.3	0.4	0.3	0.2	0.2	0.2	0.1	0.3	0.2
	p	0.952		0.943		0.776		0.763		0.923		0.443	
RGII	M	1.7	1.6	3.8	3.9	6.0	5.9	2.1	2.2	2.1	2.0	4.2	4.2
	SD	0.2	0.2	0.3	0.3	0.4	0.4	0.2	0.2	0.2	0.2	0.2	0.3
	p	0.085		0.109		0.104		0.164		0.053		0.623	

M – mean; SD – standard deviation; p<0.001 was considered to indicate statistical significance according to Bonferroni correction; BAEP – brainstem auditory evoked potential.

Table 2. Mean (SD – standard deviation) by group and between-group (CG, RGI and RGII) comparison of BAEP latencies of waves I, III, V and interpeaks I–III, III–V, I–V according to Kruskal-Wallis test.

BAEP	CG M (SD)	RGI M (SD)	RGII M (SD)	p
I	1.55 (0.10)	1.70 (0.20)	1.65 (0.20)	0.140
III	3.75 (0.15)	3.95 (0.30)	3.85 (0.30)	0.061
V	5.70 (0.20)	5.95 (0.35)	5.95 (0.40)	0.006
I–III	2.20 (0.15)	2.25 (0.20)	2.15 (0.20)	0.183
I–V	4.10 (0.40)	4.25 (0.25)	4.20 (0.25)	0.124
III–V	2.00 (0.40)	2.00 (0.15)	2.05 (0.20)	0.487

M – mean; SD – standard deviation; p<0.001 was considered to indicate statistical significance according to Bonferroni correction; BAEP – brainstem auditory evoked potential.

Table 3. Comparison of BAEP and audiometry alterations for CG, RGI and RGII according to Chi-square or Fisher's exact test.

Groups	BAEP	p	Audiometry	p
CG	13.3%		0.0%	
RGI	55.6%	<0.001*	27.7%	<0.001*
RGII	66.7%		48.1%	

p<0.005 was considered to indicate statistical significance according to Bonferroni correction. BAEP: brainstem auditory evoked potential.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Table 4. Two by two comparisons of CG, RGI and RGII concerning proportions of BAEP and audiometry alterations according to the Chi-square or Fisher's exact test.

Between-groups comparison	p
BAEP	
CG X RGI	0.003*
CG X RGII	0.000*
RGI X RGII	0.537
Audiometry	
CG X RGI	0.005*
CG X RGII	0.000*
RGI X RGII	0.190

p<0.017 was considered to indicate statistical significance according to Bonferroni correction. BAEP – brainstem auditory evoked potential.

In order to further investigate this effect, chi-square test or Fisher's exact test followed by Bonferroni corrections were applied to compare the groups 2 by 2. It was observed that the proportions of BAEP and audiometry alterations were significantly lower in CG than in the other groups. However, there was no statistically significant difference between groups exposed (RGII) and not exposed (RGI) to antiretroviral treatment (p=0.537; p=0.190). These analyses are described in Table 4.

Of the individuals from CG who had altered BAEP, 100% had low brainstem alterations. As for RGI individuals, the type of alterations varied – 20% middle ear impairment, 70% low brainstem, and 10% high and low brainstem. In RGII, 27.8% of individuals with alterations presented middle ear impairment, 22.2% sensory impairment, 11.1% high brainstem, 33.3% low brainstem, and 5.6% high and low brainstem.

Table 5 describes the percentage of BAEP alterations types according to group (CG, RGI, and RGII).

Discussion

No difference between right and left ears regarding latency of BAEP waves was found for the studied groups (Table 1). Although latency values obtained for right and left ears were not statistically compared between groups, mean latency for the control group was predominantly smaller than for RGI and RGII. Larger latency values were visualized mainly for waves III (4.0 ms) and V (6.0 ms) in the right ear of RGI and wave V (6.0 ms) in the right ear of RGII when compared to values obtained in the control group (3.8 ms and 5.7 ms, respectively, for waves III and V). Similar data were also found in a study [21] that evaluated 30 patients with AIDS and 9 HIV-positive patients without AIDS symptoms. The author concluded that, although not statistically significant, there was a tendency to increased BAEP wave V latency in the HIV-asymptomatic group when compared to the control group (HIV-negative).

No statistically significant differences were observed when comparing BAEP latencies of waves I, III, V and interpeaks I–III, III–V, I–V among CG, RGI, and RGII (Table 2). This finding corroborates the results obtained by Lima and Fukuda (1999) in a study that compared 30 asymptomatic HIV-positive individuals and 30 healthy HIV-negative individuals. In turn, the findings of the current study differed from those obtained by others authors [22–24] who investigated auditory evoked potentials of HIV-positive individuals and observed a significant increase in latencies of waves III and V when compared to the control group. The current results are also not in agreement with findings by Mata Castro et al. [3], who concluded that an increased latency of wave III was the most common BAEP alteration in HIV-infected individuals.

Table 5. Percentage of BAEP alteration types according to group (CG, RGI, and RGII).

BAEP	CG (%)	RGI (%)	RGII (%)
MEI	0.0	20.0	27.8
SI	0.0	0.0	22.2
HB	0.0	0.0	11.1
LB	100.0	70.0	33.3
LB+HB	0.0	10.0	5.6
Total of BAEP alterations% (n)	100.0 (4)	100.0 (10)	100.0 (18)

BAEP – brainstem auditory evoked potential; MEI – middle ear impairment; SI – sensory impairment; HB – high brainstem; LB – low brainstem; HB+LB – high and low brainstem.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

According to Bankaitis et al. [25], the central conduction of auditory information in patients with several degrees of HIV infection may be compromised, usually with an increase in latency of BAEP waves III and V. Other studies in the literature have also revealed BAEP alterations in HIV-infected individuals when compared to the control group, especially in latency of wave V and interpeaks III–V and/or I–V [26–29].

Statistically significant differences were observed ($p<0.001$) when comparing the percentage of BAEP alterations among CG, RGI, and RGII (Table 3). A higher percentage of alterations in groups of HIV-positive individuals exposed and not exposed to antiretroviral treatment (55.6% and 66.7%, respectively) was found when compared to the control group (13.3%). A similar situation was observed in audiometry alterations, with higher percentage of alterations in groups of HIV-positive individuals (RGI 27.7% and RGII 48.1%) when compared to CG, which presented no alterations in audiometry.

As in the current study, several other studies have observed high incidence of BAEP alterations in HIV-positive patients [5,24,29,30], emphasizing that these alterations may be present beginning from the earliest stages of the disease, even in the absence of clinical manifestations. This aspect could be observed in this study, as the group of HIV-positive individuals not exposed to antiretroviral treatment (RGI) also exhibited a large percentage of BAEP alterations (55.6%), as well as the group of individuals exposed to treatment (66.7%). The different percentage of BAEP and audiometry alterations among RGI and RGII also show that this central auditory pathway impairment detected by BAEP may be present before the clinical manifestations are detected by audiometry.

The BAEP results also corroborated findings by other authors [31], who reported BAEP alteration as one of the electrophysiological manifestations directly attributed to HIV infection.

In the present study, the percentage of BAEP alterations was significantly lower in CG than in other groups (Tables 3, 4). Despite no statistically significant difference between the groups exposed and not exposed to antiretroviral therapy ($p=0.537$), the group exposed to antiretroviral therapy showed a higher percentage of abnormal results (66.7%) than the group not exposed to treatment (55.6%). Several explanations for the BAEP electrophysiological manifestations observed in groups with HIV-positive individuals may exist. For the group not exposed to treatment, the impairment in the auditory pathway at the brainstem level possibly occurs due to the direct action of HIV on this structure [10,31,32].

In contrast, for the group exposed to antiretroviral therapy, several possible etiologic factors – beyond the direct action of HIV in the brainstem – can be considered, such as higher exposure to ototoxic drugs, greater occurrence of opportunistic infections due to immunosuppression, or use of antiretroviral therapy [4,12–14,21].

The types of BAEP alterations observed in RGI and RGII (Table 5) reflected peripheral (mainly middle ear disorders) and central compromises (auditory pathway alterations in the low brainstem). The most frequently observed alteration in both groups of HIV-positive individuals was suggestive of involvement of the lower brainstem, followed by abnormalities suggestive of involvement of the middle ear, corroborating findings presented in recent studies [30,33].

The central alterations became evident through the increased latencies of waves III and/or V and interpeaks I–III and I–V (characteristic of low brainstem impairment). These data are in agreement with those reported by Bankaitis et al. [25], who stated that the central conduction of auditory information in patients with several degrees of HIV infection may be compromised, as reflected by changes in wave form, usually through increased latency of waves III and V. The results of the present study also corroborate the findings of Pierelli et al. [34], who observed a significant increase in latency of interpeaks I–III, III–V, and I–V in HIV-positive patients.

Assessing the auditory pathway at the brainstem level in HIV-seropositive individuals, authors [3,22,30,33] predominantly found an increase in latencies of waves III and V and interpeaks I-V and/or III-V, suggesting lower brainstem impairment in this population.

The present study reports findings suggestive of middle ear impairment, especially in RGI and RGII. This finding supports the literature that emphasizes immunosuppression as one of the responsible factors for susceptibility to middle-ear infections in individuals with HIV/AIDS [32,35,36].

Considering the increasing development of antiretroviral drugs that allow the delay of the disease, the knowledge of possible adverse effects associated with antiretroviral therapy (specifically ototoxicity) becomes even more relevant, so one can tailor the medication regimen also considering a better quality of life.

Therefore, due to increased life expectancy, early identification of peripheral and central auditory disorders – with the application of behavioral, electroacoustic, and electrophysiological hearing procedures – can contribute to a better prognosis and rehabilitation process in these patients. This would, in turn, aid in a better definition of the degree and evolution rate of the impairment and, consequently, monitoring the evolution of the disease caused by HIV.

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]

Conclusions

HIV-positive individuals have a higher percentage of altered brainstem auditory evoked potential that suggests central auditory pathway impairment when compared to HIV-negative individuals. These alterations suggest impairment in the low brainstem, mainly related to changes in synchrony in the generation and transmission of neuroelectric impulses along the auditory pathway in the brainstem. There was no significant difference between individuals exposed and not exposed to antiretroviral treatment.

BAEP can detect neurological dysfunction in still-asymptomatic individuals, and thus is useful in early detection of neuropathology associated with HIV infection.

References:

1. Pahwa S: Human immunodeficiency virus infection in children: nature of immunodeficiency, clinical spectrum and management. Pediatr Infect Dis J, 1988; 7(5 Suppl.): S61–71
2. Khoza K, Ross E: Auditory function in a group of adults infected with HIV/AIDS in Gauteng, South Africa. S Afr J Commun Disord, 2002; 49: 17–27
3. Mata Castro N, Yebra Bango M, Tutor de Ureta P et al: [Hearing loss and human immunodeficiency virus infection. Study of 30 patients]. Rev Clínica Española, 2000; 200(5): 271–74 [in Portuguese]
4. Chow KY, Ang LW, Verghesse I et al: Measurable predictive factors for progression to AIDS among HIV-infected patients in Singapore. Ann Acad Med Singapore, 2005; 34(1): 84–89
5. Reyes-Contreras L, Silva-Rojas A, Ysunza-Rivera A et al: Brainstem auditory evoked response in HIV-infected patients with and without AIDS. Arch Med Res, 2002; 33(1): 25–28
6. Polich J, Basho S: P3a and P3b auditory ERPs in HIV patients receiving anti-viral medication. Clin Electroencephalogr, 2002; 33(3): 97–101
7. Chao LL, Lindgren JA, Flenniken DL, Weiner MW: ERP evidence of impaired central nervous system function in virally suppressed HIV patients on antiretroviral therapy. Clin Neurophysiol, 2004; 115(7): 1583–91
8. Tartar JL, Sheehan CM, Nash AJ et al: ERPs differ from neurometric tests in assessing HIV-associated cognitive deficit. Neuroreport, 2004; 15(10): 1675–78
9. Volberding PA, Deeks SG: Antiretroviral therapy and management of HIV infection. Lancet. 2010; 376(9734): 49–62
10. Reis AC, Santos EM dos, Cruz MM da: A mortalidade por aids no Brasil: um estudo exploratório de sua evolução temporal. Epidemiol e Serviços Saúde. Coordenação-Geral de Desenvolvimento da Epidemiologia em Serviços / Secretaria de Vigilância em Saúde / Ministério da Saúde, 2007; 16(3): 195–205 [in Portuguese]
11. Tsibris AMN, Hirsch MS: Antiretroviral therapy in the clinic. J Virol, 2010; 84(11): 5458–64
12. Vogeser M, Colebunders R, Depraetere K et al: Deafness caused by didanosine. Eur J Clin Microbiol Infect Dis, 1998; 17(3): 214–15
13. Williams B: Ototoxicity may be associated with protease inhibitor therapy. Clin Infect Dis, 2001; 33(12): 2100–2
14. Rey D, L'Héritier A, Lang JM: Severe ototoxicity in a health care worker who received postexposure prophylaxis with stavudine, lamivudine, and nevirapine after occupational exposure to HIV. Clin Infect Dis, 2002; 34(3): 418–19
15. Campanini A, Marani M, Mastroianni A et al: Human immunodeficiency virus infection: personal experience in changes in head and neck manifestations due to recent antiretroviral therapies. Acta Otorhinolaryngol Ital, 2005; 25(1): 30–35
16. Vieira A, Mancini P, Gonçalves D: Doenças infecciosas e perda auditiva. Rev Med Minas Gerais. 2010; 20(1): 102–6 [in Portuguese]
17. Matas CG, Angrisani RG, Magliaro FCL, Segurado AAC: Audiological manifestations in HIV-positive adults. Clinics (Sao Paulo), 2014; 69(7): 469–75
18. British Society of Audiology. Recommendation. Descriptors for pure-tone audiograms. Br J Audiol, 1988; 22(2): 123
19. Jasper H: The ten-twenty electrode system of the International Federation. Electroencephalogr Clin Neurophysiol, 1958; 10: 371–75
20. Matas CG: Medidas Eletrofisiológicas da Audição. Audiometria de Tronco Cerebral. In: Carvallo R, editor. Fonoaudiologia Informação para a formação: Procedimentos em Audiologia. São Paulo: Guanabara Koogan, 2003; 43–57 [in Portuguese]
21. Bankaitis A: The effects of click rate on the auditory brain stem response (ABR) in patients with varying degrees of HIV-infection: a pilot study. Ear Hear, 1995; 16(3): 321–24
22. Hart CW, Cokely CG, Schupbach J et al: Neurotologic findings of a patient with acquired immune deficiency syndrome. Ear Hear, 1989; 10(1): 68–76
23. Castello E, Baroni N, Pallestrini E: Neurotological auditory brain stem response findings in human immunodeficiency virus-positive patients without neurologic manifestations. Ann Otol Rhinol Laryngol, 1998; 107(12): 1054–60
24. Serafini G, Stagni G, Chiarella G et al: ABR and HIV-induced impairment of the central nervous system. Rev Laryngol Otol Rhinol (Bord), 1998; 119(2): 87–90
25. Bankaitis A, Christensen L, Murphy G, Morehouse C: HIV/AIDS and auditory evoked potentials. Semin Hear, 1998; 19(2): 177–93
26. Somma-Mauvais H, Farnarier G: [Evoked potentials in HIV infection]. Neurophysiol Clin, 1992; 22(5): 369–84 [in French]
27. Boccellari AA, Dilley JW, Yingling CD et al: Relationship of CD4 counts to neurophysiological function in HIV-1 – infected homosexual men. Arch Neurol, 1993; 50(5): 517–21
28. Grimaldi LM, Luzi L, Martino G V et al: Bilateral eighth cranial nerve neuropathy in human immunodeficiency virus infection. J Neurol, 1993; 240(6): 363–66
29. Reyes A, Ysunza A, Silva A et al: Potenciales provocados auditivos de latencia corta en pacientes infectados por virus de inmunodeficiencia humana. Resultados preliminares. An Otorrinolaringol Mex, 1999; 44(1): 3–7 [in Spanish]
30. Juan K: Avaliação audiológica, eletroacústica e eletrofisiológica da audição em adultos com HIV/AIDS. Universidade de São Paulo; 2009 [in Portuguese]
31. Bankaitis AE, Keith RW: Audiological changes associated with HIV infection. Ear Nose Throat J, 1995; 74(5): 353–59
32. Prasad HKC, Bhojwani KM, Shenoy V, Prasad SC: HIV manifestations in otolaryngology. Am J Otolaryngol, 2006; 27(3): 179–85
33. Matas CG, Silva SM, Marcon B de A, Gonçalves IC: Manifestações eletrofisiológicas em adultos com HIV/AIDS submetidos e não-submetidos à terapia anti-retroviral. Pró-Fono Rev Atualização Científica, 2010; 22(2): 107–12 [in Portuguese]
34. Pierelli F, Garrubba C, Tilia G et al: Multimodal evoked potentials in HIV-1-seropositive patients: relationship between the immune impairment and the neurophysiological function. Acta Neurol Scand, 1996; 93(4): 266–71
35. Soucek S, Michaels L: The ear in the acquired immunodeficiency syndrome: II. Clinical and audiologic investigation. Am J Otol, 1996; 17(1): 35–39
36. Gold S, Tami T: Otolaryngological manifestations of HIV/AIDS. Semin Hear, 1998; 19(2): 167–75

Indexed in: [Current Contents/Clinical Medicine] [SCI Expanded] [ISI Alerting System] [ISI Journals Master List] [Index Medicus/MEDLINE] [EMBASE/Excerpta Medica] [Chemical Abstracts/CAS] [Index Copernicus]