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Original articles

Brainstem auditory evoked potentials in smokers

Potenciais evocados auditivos de tronco encefálico em fumantes

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ABSTRACT

Purpose: to perform a comparative study of brainstem evoked auditory potentials between smokers and non-smokers.

Methods: the group studied was composed of 40 individuals, being 20 non-smokers and 20 smokers within the range of 20 to 59 years of age. All participants had to present responses to tonal thresholds within normal range and tympanometry type A, with the presence of ipsilateral and contralateral acoustic reflexes. Both groups underwent brain stem auditory evoked potential (BAEP). The parameters used to compare the two groups were the absolute latencies of waves I, III and V, the inter-latency waves I-III, IV and III-V in both ears, the difference between the IV inter-peak latency between the two ears and the inter--aural difference of wave V absolute latency between the two ears.

Results: in our results, it was ascertained that the group of smokers showed latency I in the RE (p= 0.036), latency V in the RE (p= 0.007), latency V in the LE (p=0.014), inter-latency III-V in the RE (p=0.015) and LE (p= 0.016) significantly higher than the non-smokers. There was no significant difference in wave V latency between the two ears.

Conclusion: the results of the study led to the conclusion that tobacco is a risk factor for the central auditory nervous system, interfering with latencies and with BAEP inter-wave latencies in the group of smokers when compared to the group of non-smokers.

Keywords: Smoking; Evoked Potentials, Auditory, Brain Stem; Hearing

RESUMO

Objetivo: comparar os resultados dos exames de potenciais evocados auditivos de tronco encefálico em indivíduos não tabagistas e tabagistas.

Métodos: foram estudados 40 indivíduos, sendo 20 não tabagistas e 20 tabagistas, com idades entre 20 e 59 anos. Todos os participantes incluídos na pesquisa deveriam apresentar respostas de limiares tonais dentro dos padrões da normalidade e timpanometria tipo A com presença de reflexos acústicos contralaterais e ipsilaterais. Em ambos os grupos foram realizados os potenciais evocados auditivos de tronco encefálico (PEATE), por meio de cliques. Os parâmetros que foram utilizados na comparação dos dois grupos foram as latências absolutas das ondas I, III e V; as interlatências das ondas I-III, I-V e III-V em ambas as orelhas; a diferença da latência interpico I-V entre as duas orelhas e a diferença interaural da latência absoluta da onda V entre as duas orelhas.

Resultados: os resultados encontrados mostraram que o grupo de tabagistas apresentou latência I da Orelha Direita (p=0,036), latência V da Orelha Direita (p=0,007), latência V da Orelha Esquerda (p=0,014), interlatência III-V da Orelha Direita (p=0,015) e Orelha Esquerda (p=0,016) significantemente maior que o grupo de não tabagistas. Não houve diferença significante na latência da onda V entre as duas orelhas.

Conclusão: os resultados da pesquisa levaram à conclusão de que o tabaco é um fator de risco para o sistema nervoso auditivo central, que pode interferir nas latências e interlatências das ondas do PEATE no grupo de tabagistas quando comparado com o grupo de não tabagistas.

Descritores: Tabagismo; Potenciais Evocados Auditivos de Tronco Encefálico; Audição

INTRODUCTION

According to the World Health Organization (WHO, 2003)[1,2], the cigarette, in a general way, affects the health of the individual and it is responsible for cancer in the lungs, larynx, cervix (in female smokers), pancreas, bladder, esophagus, stomach and kidneys, besides cardiovascular and respiratory diseases, among others. These are fatal diseases, and the amount of cigarettes smoked per day is proportional to the risk of contracting them.

The cigarette can also be harmful for the hearing because of the antioxidant mechanism effect or because of the vascular suppression of the auditory system, as this may cause conductive, mixed, sensorineural or central hearing loss. The brainstem auditory evoked potentials (ABR) test allows one to check the electrophysiological activity of the auditory system at brainstem level, to map the synapses of the auditory pathways from the cochlear nerve, cochlear nuclei, superior olivary complex (bridge) to the inferior colliculus (midbrain)[3].

The most commonly performed and used analysis for the ABR test are waves I, III and V latency values and their interpeaks I-III, III-V and I-V, since these three waves have higher amplitude and stability. Absolute wave latency V, the interaural difference of wave V latency and interpeaks I-III, I-V, and III-V are good parameters for diagnostic purposes[4]. One of the qualities of the ABR test is the ability to evaluate the neurophysiological integrity of the brain stem auditory pathways. You can compare the speed of progress of the stimulus (latencies) in both ears[5].

For over 100 years it´s been suggested that there is a relation between smoking and hearing loss[6] , and recent research carried out by Belgian researchers offer convincing evidence on the subject. These researchers stated that smoking significantly increased high-frequency hearing loss and the dose dependent effect. According to Erik Fransen, one of the researchers of this study from the University of Antwerp (Belgium), hearing gets worse after smoking regularly for over a year. Once the damage is done, it is irreversible[7, 8].

In reviews of national and international papers, some studies indicate that smoking may be a risk factor for hearing loss. However, other papers have found no correlation between hearing loss and smoking[6, 7].

Research has shown that people who smoked more than a pack of cigarettes per day have worse thresholds of 250 to 1000 Hz than those who smoke little or do not smoke at all[9]. Smoking can significantly influence in the beating rate of hair cells lashes in the lining of the middle ear, reducing the frequency of its beating rate and leading to persistence of middle ear infections[10].

A study on the effects of smoking on brainstem auditory evoked potentials (ABR) in twelve smokers found that the latency and amplitude of wave peaks I, III and V were evaluated and analyzed, and there was no significant effect of peaks I and III. A significant effect for peak V with tobacco, resulting in higher latencies was observed[11].

The information above motivated this research, and its goal is to carry out a comparative study of the brainstem auditory evoked potentials in non-smokers and in smokers, observing absolute latencies of waves I, III and V; interpeak latencies of waves I-III, III-V, I-V in both ears; the difference in interpeak latency I-V between the two ears and the interaural difference of wave V latency between the two ears.

METHODS

This research was approved by the Research Ethics Committee (CEP) of the University Veiga de Almeida (UVA), under number 302/11. It is a cross-sectional, observational study, of descriptive and exploratory type.

The literature review was performed using the Regional Medical Library (BIREME), and we used the databases from LILACS and SCIELO. Articles and scientific publications were also reviewed.

The study was conducted in the Audiology Clinic of the University Hospital, Department of Audiology.

The study was divulged by the Program of Study and Treatment of Smoking - PROJETA, a project linked to the INCA (National Institute of Cancer). This project was developed in the Hospital, which recruits volunteers for the study.

Forty individuals were evaluated and placed in two different groups: G1 and G2. Group 1 (G1) was composed of twenty non-smokers and Group 2 (G2), was composed of 20 smokers.

As a criterion for inclusion in Group 1, we selected non-smokers, i.e., individuals who had never smoked; and in Group 2, smokers. We considered as smokers individuals who had smoked and/or were currently smoking at least one cigarette a day with prior use of more than 100 cigarettes or five packs of cigarettes throughout their lives[1]. In both groups, we selected individuals of both genders, with a minimum age of 20 and a maximum age of 59.

To select the sample, we initially carried out anamnesis, otoscopy, pure-tone audiometry threshold and immitanciometry exams.

The anamnesis was composed of questions in order to gather information on personal background, such as audiological history, general health and exposure to occupational noise. We excluded individuals who presented otalgia, otitis, ear surgery history, neurological disorders and exposure to occupational noise/acoustic trauma.

An ENT doctor performed the otoscopy, in order to detect any alterations that could affect the implementation of the remaining stages of the research. When the presence of earwax was detected, the participant was referred to earwax removal and advised to return to the study after that procedure. In cases where the otorhinolaryngology evaluation detected other ear abnormalities, the participant was excluded from the research.

In the acoustic cabin, patients were previously instructed on the dynamics of the exam. We verified pure tone hearing thresholds in the frequencies of 0.25 kHz, 0.5 kHz, 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz and 8 kHz for air conduction, and 0.5 kHz, 1 kHz, 2 kHz, 3 kHz and 4 kHz for bone conduction. We excluded from the research subjects with alterations in the audiometric thresholds, i.e., those who obtained thresholds worse than 25 dBNA[12].

The individuals underwent tympanometry and research of acoustic reflexes in frequencies 0.5 kHz, 1 kHz, 2 kHz and 4 kHz. We also excluded individuals who presented type B or C curve and absence of contralateral acoustic reflex in two or more frequencies, among frequencies 0.5 kHz, 1 kHz, 2 kHz and 4 kHz.

All participants selected for the research in both groups had to present responses to tonal thresholds within the standards of normality and type A tympanometry, with the presence of contralateral and ipsilateral acoustic reflexes.

The register of the ABR test was obtained with the patient lying awake, in a quiet environment, with the use of insert earphones (Eartone), surface electrodes placed in the vertex and bilateral mastoid region, fixed with electrolytic paste. The sound stimulus used, issued on intensity of 85 dB SPL, consisted of 100 μs long clicks, alternating polarity at rate 27.7 Hz and promediation of 1024 stimuli, and the collected signal was filtered between 100 and 5000 Hz. The electric stimulus generated in the computer was turned into acoustic stimulus and transmitted through the auditory system to generate the tone-evoked potential[13].

Two records were performed, for each ear, in order to check the reproducibility of the tracing and confirm the presence or absence of the waves. For this research, between the two records we chose the one with the clearest morphology.

In the electrophysiological evaluation, we analyzed the latency values of waves I, III and V, as well as the interpeak intervals IIII, IV and IIIV, for both ears.

We studied interpeak latency I-III, which represents the activity between the auditory nerve and the lower brainstem, interpeak latency III-V, which reflects the activity of the higher brainstem and interpeak latency I-V, which is the most important one, because it represents all the activity from the auditory nerve to the nuclei and tracts of the brainstem[13].

We also studied the comparison of interpeak latency I-V between the two ears, granted that the interaural difference should not exceed 0.3 ms in normal individuals. In the absence of wave I, the interaural difference was calculated between absolute latencies of waves V and it should not exceed 0.3 ms in normal individuals either.

For the study of the absolute latencies and interlatencies, we adopted the normality criterion proposed by Hall (1992)[13], as shown in figure 1.

Waves	Correspondent (probable)	Latency in *ms* for adults (Hall, 1992)
I	Distal portion at brainstem of auditory nerve	1.5 to 1.9
II	Proximal portiom at brainstem of auditory nerve	2.5 to 3.0
III	Cochlear nucleus	3.5 to 4.1
IV	Superior olivary complex	4.3 to 5.2
V	Lateral lemniscus	5.0 to 5.9
VI	Inferior colliculus	-
VII	Medial geniculate body	-
Interpeaks	I - III	2.14
	III - V	1.89
	I - V	4.02

Figure 1. Normality standard of the latency and inter-latency values of the ABR (Hall, 1992)

Statistical analysis was done using the following methods: We applied the Mann-Whitney Test to ascertain if there was significant difference between the smoking and the non-smoking groups in latency and inter-latency measurements (at 85 dB NHL). We applied the Fisher's Exact Test to check the rate of change of these measurements. In order to check if there was significant variation in latency and inter-latency measurements from the right to the left ear we used the Wilcoxon signed-rank test.

Clinical variables were analyzed using Fisher's exact Test (sex) and Mann-Whitney Test.

Nonparametric tests were applied because latency and inter-latency measurements did not present Gaussian distribution, due to the rejection of the hypothesis of normality according the Kolmogorov-Smirnov test. The criteria for determining significance level was 5%. The Statistical analysis was processed by the software SAS 6.11 (SAS Institute Inc., Cary, NC).

RESULTS

Using the descriptive level of the Mann-Whitney test it was ascertained that the smoking group showed latency I for the RE ($p = 0.036$), latency V for the RE ($p = 0.007$) and latency V for the LE ($p = 0.014$) significantly higher than those of the non-smoking group (Table 1).

Using the descriptive level of the Mann-Whitney test, it was ascertained that the smoking group presented inter-latencies III-V for the RE ($p = 0.015$), inter-latencies III-V for the LE ($p = 0.016$) significantly higher than those of the non-smoking group (Table 1). There was no significant difference, at the 5% level, in inter-latencies between the two groups.

In order to check if there was a significant difference in the change in the of latency measure between non-smoking groups (G1) and smoking groups (G2), table 2 provides the frequency (n) and percentage (%) of change in latency measurements of right and left ear. Table 2 also provides the corresponding descriptive level (p-value) of Fisher´s exact test.

It was ascertained that there was no significant difference, at 5% level, in the rate of change of latency measurements between the two groups. Table 2 also provides the interaural difference analysis (IA) between the groups.

In table 3, we can observe if there is significant difference in latency and inter-latency measurements (at 85 dB NHL) from the right to the left ear. Table 3 also provides the average, standard deviation (SD) and the median latencies (at 85 dB NHL) according to the ear (right and left) and the corresponding descriptive level (p-value) of the Wilcoxon signed-rank test for the total sample and per group (smokers and non-smokers). It was ascertained that there was no significant difference, at 5% level, in the rate of change of latency and inter-latency measurements between the two groups.

Conclusion: There is no significant difference, at 5% level, in wave V latency between the two ears.

Table 1. Latency values of waves I, III e V and inter-latencies of I-III, I-V e III-V, right and left ears, for the group of smokers and for the group of non-smokers at 85dB NHL

Ear	Variable	Smokers (n = 20)				Non-smokers (n = 20)				p value [a]
		Average	±	SD	Median	Average	±	SD	Median	
Right	Latency I	1,51	±	0,14	1,49	1,42	±	0,10	1,42	0,036
	Latency III	3,58	±	0,13	3,54	3,56	±	0,15	3,57	0,84
	Latency V	5,58	±	0,22	5,57	5,31	±	0,45	5,43	0,007
Left	Latency I	1,50	±	0,13	1,46	1,43	±	0,10	1,42	0,17
	Latency III	3,66	±	0,17	3,62	3,57	±	0,10	3,58	0,16
	Latency V	5,57	±	0,29	5,49	5,38	±	0,16	5,35	0,014
Right	Interlatency I-III	2,09	±	0,20	2,06	2,13	±	0,16	2,12	0,44
	Interlatency I-V	4,07	±	0,25	4,11	3,99	±	0,18	4,02	0,32
	InterlatencyIII-V	2,00	±	0,17	1,96	1,86	±	0,17	1,88	0,015
Left	Interlatency I-III	2,16	±	0,14	2,14	2,15	±	0,10	2,16	0,86
	Interlatency I-V	4,08	±	0,29	4,04	3,95	±	0,15	3,96	0,18
	InterlatencyIII-V	1,94	±	0,21	1,94	1,80	±	0,12	1,79	0,016

SD: Standard deviation
[a] descriptive level of Mann-Whitney test.

Table 2. Altered latency values, in the group of smokers, compared to the unaltered values, in the group of non-smokers, for waves I, III, V and for inter-latencies I-III,I-V, III-V

Ear	Altered Value	Smokers (n = 20)		Non- smokers (n = 20)		p value [a]
		n	%	n	%	
Right	Latency I	0	0,0	0	0,0	NA
	Latency III	0	0,0	0	0,0	NA
	Latency V	2	10,0	0	0,0	0,24
Left	Latency I	0	0,0	0	0,0	NA
	Latency III	0	0,0	0	0,0	NA
	Latency V	1	5,0	0	0,0	0,50
Right	Inter-latency I-III	1	5,0	0	0,0	0,50
	Inter-latency I-V	2	10,0	0	0,0	0,24
	Inter-latency III-V	1	5,0	0	0,0	0,50
Left	Inter-latency I-III	0	0,0	0	0,0	NA
	Inter-latency I-V	2	10,0	0	0,0	0,24
	Inter-latency III-V	1	5,0	0	0,0	0,50
Difference IA < 0,3ms		20	100,0	20	100,0	NA

[a] descriptive level of Fisher´s Exact test.

Table 3. Latency values for waves I, III, V and inter-latencies of I-III, I-V, III-V, according to right and left ears, in all the sample per groups – smokers and non-smokers, at 85 dB NHL

Sample	Variable	Right Ear				Left Ear				p value a
		Average	±	SD	Median	Average	±	SD	Median	
Total (n = 40)	Latency I	1,47	±	0,13	1,45	1,46	±	0,12	1,42	0,58
	Latency III	3,57	±	0,14	3,56	3,62	±	0,15	3,59	0,010
	Latency V	5,45	±	0,38	5,51	5,47	±	0,25	5,41	0,81
	Inter-latency I-III	2,11	±	0,18	2,10	2,16	±	0,12	2,15	0,10
	Inter-latency I-V	4,03	±	0,22	4,05	4,01	±	0,24	3,99	0,97
	Inter-latency III-V	1,93	±	0,19	1,92	1,87	±	0,18	1,84	0,063
Group of smokers (n = 20)	Latency I	1,51	±	0,14	1,49	1,50	±	0,13	1,46	0,52
	Latency III	3,58	±	0,13	3,54	3,66	±	0,17	3,62	0,010
	Latency V	5,58	±	0,22	5,57	5,57	±	0,29	5,49	0,90
	Inter-latency I-III	2,09	±	0,20	2,06	2,16	±	0,14	2,14	0,22
	Inter-latency I-V	4,07	±	0,25	4,11	4,08	±	0,29	4,04	0,54
	Inter-latency III-V	2,00	±	0,17	1,96	1,94	±	0,21	1,94	0,23
Group of non-smokers (n = 20)	Latency I	1,42	±	0,10	1,42	1,43	±	0,10	1,42	0,93
	Latency III	3,56	±	0,15	3,57	3,57	±	0,10	3,58	0,33
	Latency V	5,31	±	0,45	5,43	5,38	±	0,16	5,35	0,66
	Inter-latency I-III	2,13	±	0,16	2,12	2,15	±	0,10	2,16	0,47
	Inter-latency I-V	3,99	±	0,18	4,02	3,95	±	0,15	3,96	0,42
	Inter-latency III-V	1,86	±	0,17	1,88	1,80	±	0,12	1,79	0,13

SD: Standard Deviation
[a] descriptive level of Wilcoxon *signed-rank* test

DISCUSSION

The ABR test stands out for being an effective method in measuring the brainstem electrophysiological profile, considering the ascending auditory pathways, which occupy the segment of this structure in the central nervous system. The brainstem runs various functions of the human organism, from the simplest, such as primitive reflexes, to integrated reflexes, like the reflexes responsible for heart rate, breathing and blood pressure control[13-17].

The deterioration of the nervous system function, for the most part, takes place in a rostro-caudal direction, i.e., it starts at the cortex, passing through subcortical regions until it reaches the brainstem[15].

The assessment of neurophysiological integrity of the brainstem by the ABR test is given by the synchrony of the neural element, which can be observed by the overlapping of waves, proper morphology, latency of the waves and interpeak intervals in normal individuals, ipsilateral comparison of some waves and interaural comparison of other recorded waves[13,14].

When studying latencies I, III and V (table 1), better latency measurements value I were found in the right ear (RE) and left ear (LE) best for the non-smoking group (G1). However, statistically significant difference was found only in the right ear (RE).

In latency III, no significant change between the two groups was observed. However, significantly higher change in latency V in the RE ($p = 0.007$) and LE ($p = 0.014$) was found for the smoking group (G2) when compared to the non-smoking group (G1). These results are consistent with a study on the effects of smoking on the latencies of the ABR where a significant effect for wave V, with higher latency values was found. This study did not describe the findings of waves I and III[11].

There was also an agreement with the results obtained by another study[18], stating that nicotine interferes with the neural transmission of hearing information, because the amplitude and latency of the ABR waves are altered in smokers [19]. Such data were also observed in research[20] in which the latency of wave III showed higher values in the group of smokers when

compared to the group of non-smokers, and this difference is significant. There was no similarity found in studies that presented statistically significant values only in the right ear (RE); perhaps, this fact can be justified because of the small size of the sample.

According to Table 1, in the analysis of inter-latencies I-III and I-V, it was observed that there was no statistically significant difference between the two groups, at 5% level. It was observed that the group of smokers (G2) presented inter-latencies III-V in the RE ($p = 0.015$) and inter-latencies III-V in the LE ($p = 0.016$) significantly higher than those of the group of non-smokers (G1). This increase in III-V inter-latencies found in the group of smokers (G2) may represent an abnormality of neural transmission, possibly located between the cochlear nuclei (wave III) and lateral lemniscus (wave V) 15. Due to the size of the sample, it was not possible to carry out a study considering the time of smoking, the minimum amount of cigarettes smoked per day and inter-latency III-V. However, we suggest studies with a larger population, with a view to the identification of the influence of the quantity of tobacco in neural transmission of auditory information, reflected in the latencies and inter-latencies of the ABR.

To analyze the criterion of normality in table 2, we adopted the values suggested by Hall (1992)[13]. In studies of the latencies according to groups, in agreement with the proposal of normality, individuals with altered latencies were found only in the group of smokers (G2), where the latencies of wave V were altered in two individuals, granted that one individual had it in both ears (RE and LE) and the other in his left ear (LE). In consulted literature, we did not find other studies using individual values of latency, inter-latency and interpeak and these criteria of normality, which makes it impossible to compare the findings with other studies.

Still based on the normality criteria of Hall (1992)[13], an analysis of individuals of inter-latencies I-V was presented in table 2. It was verified that there were more alterations in the group of smokers (G2.) We observed three individuals with altered values, considering that the participant had alterations in both ears (RE and LE), one participant (RE) and another participant (LE). However, statistical analysis only pointed difference in inter-latencies III-V.

In the analysis per individual, we observed more alterations in three participants, perhaps because they presented greater sensitivity to regular exposure to tobacco, which may be signaling future complications.

For the group of non-smokers (G1) we found that 100% of the sample did not present any alterations in the items studied (latency, inter-latency and interpeak).

Thus, in the total of the group of smokers (G2), three individuals were found (15%) with alterations in one or more of the items studied (latency, inter-latency and interpeak).

In the analysis of interaural difference, it was observed that there was no significant difference at the level of 5% in wave V latency between the two ears.

Given the importance of the study of the interaural difference of wave V latency for diagnostic purposes[13], we decided to conduct this study according to the groups, but no alterations were found.

CONCLUSION

There are significant differences in the absolute latencies of wave I, electric impulse transmission as far as the auditory nerve, in the right ear, and of wave V, electrical impulse to the lateral lemniscus, in both ears, for the groups of smokers when compared with the group of non-smokers.

There is an increase in inter-latencies III-V, which may indicate impairment of the higher brainstem, in both ears, for the group of smokers.

There is no interaural difference of wave V between the ears, in both groups.

The nicotine found in tobacco, which interferes with the neural transmission of auditory information, is a risk factor for the central auditory nervous system. It can affect the latencies and inter-latencies of the auditory brainstem response test in the group of smokers, when compared to the group of non-smokers, when we consider that this test is able to evaluate the neurophysiological integrity of the brainstem auditory pathways.

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