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Middle Latency Auditory Evoked Potential (MLAEP) in Workers with and without Tinnitus who are Exposed to Occupational Noise

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Background: Tinnitus is an important occupational health concern, but few studies have focused on the central auditory pathways of workers with a history of occupational noise exposure. Thus, we analyzed the central auditory pathways of workers with a history of occupational noise exposure who had normal hearing threshold, and compared middle latency auditory evoked potential in those with and without noise-induced tinnitus.

Material/Methods: Sixty individuals (30 with and 30 without tinnitus) underwent the following procedures: anamnesis, immittance measures, pure-tone air conduction thresholds at all frequencies between 0.25–8 kHz, and middle latency auditory evoked potentials.

Results: Quantitative analysis of latencies and amplitudes of middle latency auditory evoked potential showed no significant differences between the groups with and without tinnitus. In the qualitative analysis, we found that both groups showed increased middle latency auditory evoked potential latencies. The study group had more alterations of the "both" type regarding the Na-Pa amplitude, while the control group had more "electrode effect" alterations, but these alterations were not significantly different when compared to controls.

Conclusions: Individuals with normal hearing with or without tinnitus who are exposed to occupational noise have altered middle latency auditory evoked potential, suggesting impairment of the auditory pathways in cortical and subcortical regions. Although differences did not reach significance, individuals with tinnitus seemed to have more abnormalities in components of the middle latency auditory evoked potential when compared to individuals without tinnitus, suggesting alterations in the generation and transmission of neuroelectrical impulses along the auditory pathway.

MeSH Keywords: Evoked Potentials, Auditory • Electrophysiology • Noise, Occupational • Tinnitus

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Background

Tinnitus is the perception of sound without an external acoustic stimulus [1]. The mechanisms underlying tinnitus are still not fully understood and, although this symptom is often associated with hearing loss, many individuals may experience it in the presence of normal hearing [2–5]. Tinnitus may result from a pathological state of the auditory system, which triggers a series of events increasing neuronal activity at different levels of the central auditory pathway [5]. Other systems also take part in the generation and maintenance of this symptom, particularly the limbic and autonomic nervous systems ("neurophysiological concept of tinnitus generation") [5, 6]. Some authors have proposed that tinnitus originates in the cochlea; however, the maintenance of this abnormal activity is perpetuated by the central auditory pathways [6,7].

Among clinical procedures to assess the central auditory pathway, the most widely used involve auditory evoked potentials. Alterations in central auditory tests and electrophysiological abnormalities in brainstem auditory evoked potential (BAEP) [8–10], middle latency auditory evoked potential (MLAEP) [11,12], and long latency auditory evoked potential (LLAEP) [13–15] have been reported in individuals with tinnitus. Specifically, the MLAEP consists of an electrophysiological measure that has not often been applied to evaluate the central auditory pathways of individuals with tinnitus. The role of cortical and subcortical auditory structures in the generation and maintenance of tinnitus is still controversial. Nevertheless, some studies have investigated the MLAEP in individuals with this symptom [10,11,16], in an attempt to identify the structures involved in this process, assisting in medical diagnosis and, consequently, in the development of more effective treatments and therapies.

However, these previous studies using MLAEP [10,11,16,17] used heterogeneous groups of individuals with tinnitus and yielded controversial results that need to be reconciled. An important aspect to consider regarding tinnitus is that it may also be caused by various diseases that affect different structures along the auditory pathway. Therefore, studies should control for these variables by evaluating a population that is homogeneous in terms of possible causes of tinnitus, e.g., individuals exposed to occupational noise [5,13].

Therefore, the aim of this study was to analyze MLAEP in individuals with normal hearing exposed to occupational noise and compare results between those with and without tinnitus.

Material and Methods

The present research was a cross-sectional study conducted at the Laboratory for Hearing Research in Auditory Evoked Potentials of the Speech Therapy program of the School of Medicine of the University of São Paulo (FMUSP) and was approved by the Ethics Committee of the University Hospital of the University of São Paulo, as well as by the Ethics Committee for the Evaluation of Research Projects of the Hospital das Clínicas and of the School of Medicine of the University of São Paulo – CAPPesq, under protocols 712/06 and 1278/06, respectively. All procedures were conducted after participants signed the informed consent form.

Study participants were 60 individuals exposed to occupational noise (above 85 dB (A)), 30 with tinnitus (study group) and 30 without tinnitus (control group), including four women (13.33%) and 26 men (86.67%) for each group. Study and control participants did not differ in terms of age (with tinnitus: mean age=41, range 27–50; without tinnitus: mean=41.6, range 27–50; p=0.563).

Patients were selected to participate in the study if they experienced constant or intermittent tinnitus that was either unilateral or bilateral (study group),were exposed to occupational noise, had hearing thresholds within normal limits (less than or equal to 25 dB HL at all frequencies – 0.25 kHz to 8 kHz) in both ears, had a type A tympanogram (pressure up to 100 Pa and volume between 0.3 and 1.6cc), and a contralateral acoustic reflex at frequencies of 0.5, 1, and 2kHz[18].

Individuals with neurological, psychiatric, and/or behavioral disorders (elucidated from workers' medical records) were excluded from the study.

The following procedures were performed: medical history; inspection of the external acoustic meatus using a Heine type otoscope; pure tone audiometry in the frequency range of 0.25 to 8 kHz by air conduction and 0.5 to 4 kHz by bone conduction bilaterally (when thresholds by air conduction were higher than 20 dBHL), using Grason Stadler (GSI-61 and GSI-68) audiometers; acoustic immittance measures (tympanometry with a tone of 226 Hz, and acoustic reflex analysis of the ipsilateral and contralateral stapedius muscle at frequencies of 0.5, 1, and 2 kHz) using the Grason Stadler (GSI-33) middle ear analyzer.

After the audiological evaluation, the selected individuals underwent an electrophysiological assessment of hearing (BAEP and MLAEP). The MLAEP was used to obtain the Na and Pa wave amplitudes and latencies both contralaterally (C3/A2, C4/A1) and ipsilaterally (C3/A1) [19,20]. It is noteworthy that 23.3% of subjects in the control group and 53.3% of subjects in the study group showed BAEP abnormalities [5].

Participants were seated in a reclining chair in a dimly lit room and were instructed to keep their eyes closed throughout the examination. We used the BioLogic Traveler Express

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portable model with the EP317 program. We first cleaned the skin with abrasive paste and then attached the electrodes to the skin in predetermined positions using electrolytic paste and tape (micropore).

Values of electrode impedance were verified to be below 5 kOhms.

To obtain the MLAEP, electrodes were positioned on the right and left ears (A2 and A1), at the right and left temporo-parietal junctions (C3 and C4), and at the vertex (Cz) site, according to the 10–20 International Electrode System (IES). The acoustic stimulus was a monaural click presented at 70 dB HL through supra-aural headphones (TDH39) at a display speed of 9.9 clicks per second, with a 10–300 Hz band pass filter, for a total of 1,000 stimuli. Participants were instructed to remain quiet and still, to pay attention to the sound, and not to sleep or talk. MLAEP results were analyzed from the Na and Pa wave latencies according to Pratt [19], and Na-Pa amplitudes, obtained contralaterally (C3/A2, C4/A1) and ipsilaterally (C3/A1, C4/A2), were analyzed according to Musiek and Lee [20].

The MLAEP was initially classified as normal or abnormal and then we assessed the types of alterations. An individual presented altered results when at least one ear (or one side) presented alterations. The Na and Pa wave latency results followed the criteria for normality proposed by Pratt [19], and the Na-Pa amplitude values, in the various modalities studied, followed the criteria of Musiek and Lee [20].

Na and Pa latencies were classified as "increased" when the wave latency was increased compared to normal values, "absent" when the wave was not detected, and "both" when the same individual displayed increased and absent waves.

For the Na-Pa amplitude analyses, a difference greater than 50% between the amplitudes obtained in the contralateral (C3/A2, C4/A1) and ipsilateral (C3/A1, C4/A2) modalities indicated dysfunction, which could be observed through the electrode effect (ELE) and the ear effect (EE). Thus, abnormal results were classified as "electrode effect (ELE)" when a difference greater than 50% was observed for the Pa wave amplitude between electrodes positioned on each temporo-parietal junction (between C3/A1 and C4/A1 or between C3/A2 and C4/A2); as "ear effect (EE)" when an ear, regardless of the electrode site (comparison between C3/A1 and C3/A2, and between C4/A1 and C4/A2) showed consistently reduced Pa wave amplitudes; and "Both" when ELE and EE alterations were observed in the same individual, one type for each ear.

For statistical analyses, we used the following tests: Wilcoxon, Mann-Whitney, equality of two proportions, and chi-square tests. Statistical significance was defined as $p \leq 0.05$ with 95%confidence intervals.

Results

Regarding the quantitative MLAEP analyses (Table 1), there were no statistically significant differences between the study and control groups in terms of normal and abnormal Na and Pa wave amplitudes and latencies.

Regarding qualitative analyses (Table 2), there were no statistically significant differences between the study and control groups in terms of normal and abnormal Na and Pa wave latencies. However, there was a statistically significant difference within each group between the occurrence of normal and abnormal Na and Pa wave latencies, with both groups obtaining a higher percentage of normal results relative to abnormal results. Both groups showed increased latencies for the Na and Pa waves.

Table 3 shows that there were no significant differences in Na-Pa amplitude alterations within or between groups. However, the control group displayed a higher percentage of normal results, while the study group had a higher percentage of abnormal results.

Groups did not differ in terms of Na-Pa amplitude (Table 4). The most frequent alteration in the control group was the electrode effect (46%), while in the study group it was the "both" effect (41%).

Discussion

Groups did not differ significantly in terms of mean Na and Pa latency and amplitude values for C3/A1, C4/A1, C3/A2, or C4/A2. This lack of significant difference may be explained, in part, by the intra- and intergroup variability for mean latency and amplitude values of the MLAEP components, even though all individuals in the present study had hearing thresholds within normal limits. This large intra- and/or inter-subject variability in mean MLAEP values has previously been described [19,21].

The study group showed a greater percentage of abnormal MLAEP results for the Na and Pa wave latencies (26.7% and 20%, respectively) compared with the control group (16.7% and 13.3%, respectively), but this difference did not reach statistical significance.

The only alteration observed in both groups was the increased latency for Na and Pa waves, and these abnormal results may be explained by changes in several subcortical [22] and cortical [23]

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Table 1. Mean MLAEP latency (in ms) and amplitude (in μv) values for the Na-Pa wave for modalities C3/A1, C4/A1, C3/A2 and C4/A2 for control and study groups (n=30).

Na latency	C3/A1		C4/A1		C3/A2		C4/A2	
	CG	SG	CG	SG	CG	SG	CG	SG
Mean	20.15	19.99	19.56	20.40	19.99	20.26	20.37	20.36
Median	19.31	19.89	19.50	20.28	19.41	19.50	19.50	19.40
Standard deviation	2.89	3.26	2.46	3.14	2.59	3.90	3.22	3.23
p-value	0.544		0.132		0.756		0.929	
Pa latency	**C3/A1**		**C4/A1**		**C3/A2**		**C4/A2**	
	CG	SG	CG	SG	CG	SG	CG	SG
Mean	33.55	32.80	33.55	32.50	33.56	32.09	33.57	31.81
Median	33.54	32.96	33.15	32.37	33.54	32.18	33.35	31.59
Standard deviation	3.61	5.77	3.91	4.95	4.10	6.27	4.37	5.50
p-value	0.631		0.251		0.280		0.183	
Na-Pa amplitude	**C3/A1**		**C4/A1**		**C3/A2**		**C4/A2**	
	CG	SG	CG	SG	CG	SG	CG	SG
Mean	3.16	2.33	2.32	1.74	2.50	2.42	1.94	1.94
Median	1.61	1.63	1.53	1.51	1.31	1.75	1.52	1.46
Standard deviation	4.85	1.88	2.11	1.08	3.56	2.01	1.28	1.88
p-value	0.717		0.641		0.204		0.784	

CG – control group; SG – study group.

Table 2. Normal and altered MLAEP values for Na and Pa wave latencies for control and study groups.

Na latency	Control		Study		p-value
	n	%	n	%	
Normal	25	83.3%	22	73.3%	0.347
Altered	5	16.7%	8	26.7%	
p-value	<0.001*		<0.001*		
Pa latency	**Control**		**Study**		**p-value**
	n	%	n	%	
Normal	26	86.7%	24	80.0%	0.488
Altered	4	13.3%	6	20.0%	
p-value	<0.001*		<0.001*		

* Statistically significant differences.

auditory structures of the CNS, which would cause an increase in the processing speed of acoustic information and hence an increase in the latency of the MLAEP components.

A total of 56.7% of individuals in the study group and 43.3% of the control participants presented abnormal results for Na-Pa amplitude, but this difference did not reach significance. The MLAEP is sensitive to dysfunctions in the thalamo cortical

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Table 3. Normal and altered MLAEP values for Na and Pa wave amplitudes for control and study groups.

Na-Pa amplitude	Control		Study		p-value
	n	%	n	%	
Normal	17	56.7%	13	43.3%	0.302
Altered	13	43.3%	17	56.7%	
p-value	0.302		0.302		

Table 4. Alterations in Na and Pa wave amplitudes for control and study groups.

Na-Pa amplitude	Ear effect		Electrode effect		Both	
	n	%	n	%	n	%
Control	4	31%	6	46%	3	23%
Study	4	24%	6	35%	7	41%
p-value	0.657		0.547		0.297	

pathway, since the responses of its generators are interpreted according to the presence or absence of response, or in terms of the ear and/or electrode effect [24].

The type of alteration most frequently found in the control group was the ELE (46%), followed by the EE (31%), and then both (23%), while in the study group the both type was the most common (41%), followed by the ELE (35%) and the EE (24%), but the difference in types of alteration between groups was not statistically different.

Based on our findings, we suggest that the study group (individuals with tinnitus exposed to occupational noise) may have subtle abnormalities in the central auditory pathway at cortical/subcortical levels, due to a greater number of alterations in both MLAEP latency and amplitude, although differences between groups did not reach significance. This hypothesis is consistent with that proposed by Singh et al. [7].

Few studies have investigated the auditory pathway of individuals with tinnitus through the use of MLAEP alone or combined with other electrophysiological measurements, e.g., BAEP or P300. Furthermore, the results of these studies are controversial. Kadlec and Mendel [11] did not find effects of tinnitus on MLAEP results, while Gerken et al. [10] observed an increase in MLAEP wave latencies, but only in some individuals with tinnitus, suggesting a selective alteration of the generators of this potential by different forms of tinnitus. Similar alterations were reported by Rybalko and Syka [25] when studying noise-induced tinnitus in rats. On the other hand, Gudwani et al. [17] found that Na and Pa wave amplitudes were more than 90% normal in individuals with tinnitus. Interestingly, Theodoroff et al. [16] suggested that the MLAEP protocol used is not specific enough to detect neurophysiological abnormalities associated with tinnitus, which may explain some of the different findings between these studies.

It is noteworthy that the control group, including individuals with hearing thresholds within normal limits and no tinnitus, also showed considerable alterations in the MLAEP, a result that we did not anticipate. Indeed, some authors have reported that exposure to noise can affect not only peripheral, but also the central portion of the auditory system. In studies with guinea pigs, MLAEP was altered in animals that were exposed to noise [26,27].

Some authors have emphasized that the cell density and structures of the central auditory pathway (central nucleus of the inferior colliculus, medial geniculate body, and primary auditory cortex) are also affected by exposure to noise, due to the overstimulation of the neurons and intense calcium influx [28,29]. These changes lead to toxic neurodegenerative processes and an enhanced neurotransmitter release that may induce glutamate excitotoxicity due to ionic homeostasis changes [28–30]. Thus, we could hypothesize that abnormalities in MLAEP observed in the two groups are due to the impact of noise exposure to the central auditory pathways.

Other authors did not employ electrophysiological procedures to study the possible site of origin of tinnitus, but reported dysfunctions in the inferior colliculus [31], the geniculate body [32], and the primary auditory cortex and temporal lobe [33]. The MLAEP has multiple generators that contribute to the formation of the positive and negative waves that make up the potential, and is therefore considered an effective procedure for identifying dysfunctions in cortical and subcortical regions [22].

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Therefore, further research should be conducted to clarify whether the cortical and subcortical structures that participate in the generation of MLAEP also contribute to the generation of tinnitus.

Conclusions

Based on our results, we conclude that:

– Individuals with and without tinnitus and normal hearing thresholds who are exposed to occupational noise present altered MLAEP, suggesting impairment of transmission of neuroelectrical impulses along the auditory pathways in cortical and subcortical regions.
– Individuals with occupational noise-induced tinnitus and normal hearing thresholds (study group) present more alterations (although not statistically significant) in MLAEP than individuals without tinnitus (control group).

Conflicts of interest

No conflicts of interest.

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