AUDIOMETRIC PROFILE AND EVOKED OTOACOUSTIC EMISSIONS PER PRODUCT OF DISTORTION IN TRANSIT MANAGERS, EXPOSED TO CARBON MONOXIDE AND NOISE

Perfil audiométrico e de emissões otoacústicas evocadas por produto de distorção em gestores de trânsito expostos a monóxido de carbono e ruído

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ABSTRACT

Purpose: to evaluate the hearing profile and otoacoustic emission evoked by distortion product in Traffic Managers exposed to noise and carbon monoxide, as well as to establish the presence of both agents at their work environment. **Method:** 37 workers were divided into two groups: G1 formed by 18 individuals with no history of concomitant exposure to carbon monoxide and noise, and G2 formed by 19 workers simultaneously exposed to both agents. To determine the presence of those agents, audio dosimeter and short period evaluation with instantaneous measurement were used. The variances of anamnesis were studied applying the Student t test and Mann-Whitney test. Otoacoustics emissions and auditory thresholds were analyzed by chi-square or Fisher exact and Wilcoxon test with 5% significance. **Results:** it was verified the presence of carbon monoxide and noise during the workers' activity. There was no significant difference in age and time of function. The average hearing threshold was greater on G2 for the right ear at 1KHz (p=0,050) and for the left at 3KHz (p=0,016) and 4KHz (p=0,028). The audiometric changes showed that G2 was significantly worse at 3KHz on the left ear (p=0,003) compared to G1. The Emissions showed worse results in G2 when compared to G1 for 2.730Hz and 3.218Hz (p=0.016) on the right ear. It was found significant impairment in both exams responses, in both ears, at the frequency ranges between 2.730Hz and 7.604Hz in G2 group. **Conclusion:** workers exposed to carbon monoxide and noise showed worst results in the auditory thresholds and Otoacoustic Emissions when compared to non-exposed group.

KEYWORDS: Hearing Loss; Carbon Monoxide; Drug Synergism; Asphyxiating Gases, Audiometry

INTRODUCTION

The transport activity, especially in the road sector, is responsible for much of the environmental degradation in urban areas[1]. Vehicles used by people emit 27.4 million tons of pollutants a year with their movement. The environmental degradation with respect to air quality, consists of a complex system involving the presence of natural or anthropogenic emission sources and topographical and meteorological conditions[2]. Vehicle emissions can be divided into two categories, one coming from the complete combustion resulting in the release of carbon dioxide (CO2) in the atmosphere, and another, incomplete, that releases hydrocarbons (HC), carbon monoxide (CO), nitrogen oxides (NOx) and particulate matter from fossil fuels in the atmosphere[1].

Carbon monoxide (CO) is a considerable chemical agent, one of the major contaminants in

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the atmosphere of the Earth. It is comparable in magnitude to the environmental problems in Latin America, and the vehicles and industrial processes are responsible for approximately 80 % of its emission in the atmosphere[3].

Noise is considered a physical agent which also has motor vehicles as its main source in urban areas, being responsible for about 80% of noise disturbance[4]. Given so much importance, noise pollution has been considered one of the three top ecological priorities for the next decade by the World Health Organization (WHO) [5].

Workers are commonly exposed to multiple agents that are toxic or aggressive to their ears. Physiological interactions with some mixed exposures can lead to an increase in the severity of harmful effects. This applies not only to the combination of chemicals substances, but also, in certain cases, to a joint action of chemical and physical factors [6]. Thus, it should be pointed out that all workers whose jobs involve the use of incomplete combustion engines have a potential exposure to carbon monoxide and noise. This category includes truck and bus drivers, mechanics, valet parking drivers in underground garages, police officers/ transit managers, street vendors and other salespeople [7,8]. Health problems caused by these types of pollutants are mainly related to the levels of agent concentration and exposure time in the environment. The exposure to both agents concomitantly presents a challenge to epidemiological studies in the understanding of the interactions between exposure and formulation of appropriate course of actions for this or that spectrum of occupational agents [9].

A member of the chemical asphyxiants, CO is a dangerous, colorless, odorless, tasteless and non-irritating gas. It can knock a person unconscious or even kill in a couple of minutes. This contaminant has similarities with the hemoglobin contained in red blood cells which carry oxygen (O2) to tissues of all body organs. Its main toxic action results in anoxia due to the conversion of oxyhemoglobin into carboxyhemoglobin (COHb) [10]. The chronic exposure to CO at low concentrations as observed in some work places, is related to symptoms such as headaches, anorexia, insomnia, behavioral disturbances, impairing in the carrier capacity of O2 with an increased cardiac output and acceleration in the process of atherosclerosis installation, as it happens in cases of smoking [11].

Occupational toxicology studies the harmful effects produced by the interaction of chemical agents foreign to the organism (toxicity) to which workers are exposed. If we consider the main routes of absorption or input ports that chemical agents use to reach the blood stream as three: Skin, gastrointestinal tract and lungs, the effects generated can be local (occurring at the contact surface between the body and the chemical agent) or systemic (when they are absorbed and distributed in the body by the blood stream, acting at a distant place from the route of entry, causing damage in tissues, target organs or body systems) [12]. When the individual is exposed simultaneously to two or more chemical substances, interactions between agents may occur, resulting in changes in the absorption rate and in the quality absorbed. This is what is called synergism, in which a substance in a dose or concentration that in itself does not have a harmful adverse effect leverages the damage caused by another substance or agent [13].

A series of studies on the toxic action of CO2 on the hearing system tends to show that CO may have a direct effect on the cochlear metabolism. The results show the most important effects on the potential for action (PA) generated by the auditory nerve fibers compared to the effects of endocochlear potential (EP), generated by the *stria vascularis* and the effects of the cochlear microphonic potential (CM), generated by the outer hair cells (OHC)[14,15].

In recent literature review, several studies on the auditory effects of acute exposure to CO have been found, even in the absence of excessive noise; in these cases it was observed a prevalence of varying degrees of sensory-neural hearing loss which confirms its ototoxic action. Only one study on the auditory effects of chronic exposure to CO in the presence of noise in occupational settings has been reported, pointing to a significant worsening of hearing thresholds in exposed workers, especially ones with an exposure of more than 20 years [15].

Hearing loss induced by high sound pressure level is a cumulative and insidious pathology that develops over the years of exposure. It is directly related to the amount of exposure to sound pressure levels (LPS), to the hours of work, to the time of exposure and individual susceptibility [16]. The initial damage occurs in the region of the first third of the cochlea or at 10mm from the base as it is a more sensitive area in regards to metabolic, anatomical and vascular damage [17]. Hearing loss induced by high sound pressure level histological changes are characterized by deviations in the cochlear flow, stereocilia alterations (softening, collapsing, merging, stretching) and an increase in the number of damaged hair cells over the years of exposure, thereby causing the reduction of active processes of the outer hair cells (such as the ability of quick contraction and degeneration of the organ of Corti). When these changes occur, it is impossible to recover the auditory thresholds [18].

The risk of acquiring hearing loss in the presence of moderate exposure to noise associated with the presence of asphyxiating chemical substances was evaluated, using mainly animal models. These studies demonstrated a potentiation of noise-induced hearing loss in which rats were simultaneously exposed to carbon monoxide (CO) and high levels of noise that alone could not produce any change in hearing thresholds [19]. The control of damaging agents for health for workers in Brazil is done through regulatory standards (NR's) of the Ministry of Labor and Employment. The NR no 15 deals with the unhealthy activities and operations and defines environmental hazards and their limits of tolerance. It considers as the tolerance threshold the concentration or intensity (maximum or minimum), related to the nature and time of exposure to the agent, which will not harm the worker's health. The NR no7 deals with the biological control of clinical manifestations caused by the working environment [20].

Given the fact above, this study aimed at evaluating the audiometric profile and distortion-produced evoked otoacoustic emissions in transit managers exposed to carbon monoxide and noise, as well as attesting the presence of these two agents in the workplace.

METHOD

We conducted a cross-sectional descriptive study with a sample initially composed of 103 individuals who underwent the anamneses protocol for investigation of excluding factors such as: Prior ear disease, diabetes, hypertension, smoking and extra-labor exposure to agents that are toxic or may injure the ear. 37 workers were eligible to take part in the study, all of them with previous exposure to impact noise (firearms), divided in two groups as described below:

G1 – The control group, consisting of 18 individuals who perform indoor work activities, with no history of concomitant exposure to carbon monoxide or high levels of sound pressure.

G2 – Formed by 19 transit managers with minimum length of 1 year in the function, exposed to high carbon monoxide levels and high sound pressure levels, 8 hours a day, 5 days a week.

In order to ascertain the presence of agents in the workplace, samples were collected e work stations, previously identified in qualitative analysis, as being the ones with highest risk. Criteria for qualitative analysis compared the working places mainly taking into consideration the flow and diversity or types of vehicles, as well as the highest intervention on the part of managers regarding the use of the whistle. All instruments used for sample collection had the calibration certificates within the validity period.

Samples of the physical agent noise were performed using individual audio dosimeters, model DOS 500. The device was programmed with a level of 85 dB criterion, duplicative dose factor equal to 5, curve A, circuit slow response and reading done near the auditory zone of the worker [21].

Environmental assessments in the workplace for chemical CO were performed in 3 short duration samples, within the period of 15 minutes each and 1 hour interval between them, using Multigas Monitor BW technologies.

To perform the hearing evaluations, a previous acoustic rest of 14 hours was followed with an inspection of the external auditory canal in both ears. As no constraint condition in the performance of the exams was observed, the tonal audiometry was performed with an Auditec device, model AD65, TDH 39 earphones and bone vibrator B71 Radioear. The audiometric booth was installed in acoustically suitable premises provided by the base division of the workers.

Pure tone thresholds were investigated by ascending/descending method at frequencies of 0.25K, 0.5K, 1K, 2K, 3K, 4K, 6K and 8KHz for airways, and 0.5K, 1K, 2K, 3K and 4K for bone conduction, when thresholds of airways above 20 dB [22] were found. The study excluded individual with air-gap bone.

The study of distortion-produced evoked otoacoustic emissions (DPOAE) was performed with the Bio-logic unit, AUDX Plus. Using the protocol of ototoxicity (1.305 to 9.071 Hz), levels of intensity equal to 55 to 65 dBNA and ratio frequency (F2/F1) equal to 1.22. To characterize the presence (normal) and absence (amended) of response we used the criteria recommended by Gorga (1996) in which the signal/noise with a result greater than 3 dB is considered normal.

This study was approved by the Ethics Committee for Research at CEFAC under nº 071/09. All subjects involved were informed about the voluntary aspects of the study, its benefits and impacts, and by means of signing a Term of Consent, agreeing to participate in research and allowing the disclosure of its results, according to Resolution MS/CNS/CNEP nº 196/96 of October 10, 1996.

The statistical analysis was conducted using the statistical software SAS® System version 6.11 (SAS Institute, Inc, Cary, North Carolina). Non-parametric methods were used because the variables did not present a normal distribution (Gaussian distribution) due to dispersion of data or lack of symmetry of the distribution. Student's t test and Mann-Whitney test respectively analyzed the factors of age and length of time in the job. Mann Whitney test was

used for statistical analysis of otoacoustic emission measures and pure tone audiometry between the groups; Chi-square test (x^2) or Fisher exact test checked the possibility of significant difference in the proportion of measure alteration in otoacoustic emissions and in pure tone audiometry; Wilcoxon signed rank test verified the measure of otoacoustic emissions and pure tone audiometry from the right to the left ear. The adopted criterion for determining significance in all tests was the 5% level.

RESULTS

Measurements

Table 1 shows the results of evaluations obtained with CO concentration. It was observed the presence of the chemical in question, being pointed out, on a corroborating level, the highest value found in particles per million (ppm) in each working station, i.e., 32, 46 and 14 ppm respectively [20].

Table 1 – Result of the samples of exposure to CO and noise

Volunteers	CO (ppm) (> value found)	Levels of Sound Pressure		
		Evaluation time (minutes)	Working hours (minutes)	NEN (dBA)
L.A.F.P	32	422	480	86.1
E.J.S.	46	397	480	90.6
M.C.N.	14	409	480	96.3

Regarding the result of the samples, two exceeded the Threshold Limit Value (TLV), according to the American Conference of Governmental Industrial Hygienists (ACGIH), which refers to the maximum instantaneous concentration permitted (25 ppm).

In the same table, you can see the standard exposure levels (NEN), corresponding to the exposure level (NE) converted to a standard working day of 8 hours. All dosimetry tests results were higher than the tolerance limit (85 dBA) for this physical agent, taking into account the given exposure time. The values found were 86.1, 90.6 and 96.3, respectively [20].

Anamnesis

When considering the factors of age and length of time in function, a statistically significant difference was not observed. (Student's t test and Mann-Whitney) between the two groups, given in the group of non-exposed workers (G1) the average age of 37.2 and the years of professional activity 13.5 and the group of exposed workers (G2) 41.2 and 10.1 years respectively.

In regard to history, complaints about tinnitus were reported in 16.7% in G1 and G2 in 10.5%.

Audiometric Assessment

In the analysis of pure tone thresholds between the groups (Table 2), it became evident that G2 had a significantly higher average threshold than the non-exposed group G1 in the right ear at a frequency of 1 KHz (p = 0.050) and left ear at the frequencies of 3KHz (p= 0.016) and 4 KHz (p= 0.028).

Table 2 – Comparative analysis between pure tone thresholds between groups

Frequency (KHz)		25		50		1		2		3		4		6		8	
Ears		RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
G1 - Non Exposed (n = 18)	Average	16.1	16.4	16.7	16.7	16.7	12.2	16.9	15.0	16.7	14.7	19.4	20.6	23.3	18.6	20.0	17,2
	SD	6.1	5.4	4.5	4.5	4.9	6.0	8.9	6.6	13.3	6.7	14.1	12.0	16.5	10.1	14.3	11
G2 - Exposed (n = 19)	Average	18.4	15.5	20.0	16.8	20.5	16.3	18.4	17.4	23.7	24.7	25.5	27.1	26.1	24.5	19.7	17,4
	SD	5.1	5.2	5.5	5.8	6.0	7.4	8.5	9.0	15.5	13.4	14.5	11.0	15.0	14.4	10.7	12
p value [a]		0,43	0.70	0.075	0.84	**0.050**	0.069	0.30	0.50	0.12	**0.016**	0.19	**0.028**	0.32	0.17	0.44	0.93

SD: Standard Deviation
Mann-Whitney Test
Value p=0,05

In quantitative analysis (table 3), related to audiometric thresholds, classified as amended compared to the two groups, it was found that G2 presented a statistically significant difference higher only for the frequency of 3 KHz in the left ear (36.7 %, p = 0.003) compared to G1 (5.6%)

Table 3 – Comparison of altered pure tone thresholds between groups

Frequency (KHz)		25		50		1		2		3		4		6		8	
Ears		RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
G1 - Non Exposed (n = 18)	n	0	0	0	0	0	0	1	1	1	1	3	2	5	3	4	3
	%	0	0	0	0	0	0	5.6	5.6	5.6	**5.6**	16.7	11.1	27.8	16.7	22.2	16,7
G2 - Exposed (n = 19)	n	1	0	1	0	2	1	2	2	5	7	7	6	8	6	3	4
	%	5.3	0	5.3	0	10.5	5.3	10.5	10.5	26.3	**36.8**	36.8	31.6	42.1	31.6	15.8	21,1
p value [a]		pc	pc	pc	pc	pc	pc	pc	pc	0.1	**0.0**	0.2	0.1	0.4	0.3	0.5	0.5

fc: few cases < 5 patients with presence or absence of variable.
χ^2 test or Fisher's exact.
Value p=0,05

Distortion-product evoked otoacoustic emissions

In the statistical analysis of the DPOAE measures in relation to missing results (amended), group G2 showed a significantly greater difference than G1 for the frequencies of 2.730 Hz and 3.218 Hz (47% and p = 0.016) only in the right ear. No significant difference was observed at 5% in proportion to changes in frequency between the other groups.

It was also observed that a higher incidence of abnormal responses in DPOAEs occurred from 2.730 Hz in both ears in G2, equivalent to the changes found in the pure tone thresholds for the same group.

Table 4 – Percentage of alterations in the measurement of otoemissions per frequency

Otoemissions (qualitative)		1305 Hz		1562 Hz		1866 Hz		2285 Hz		2730 Hz		3218 Hz		3825 Hz		4549 Hz		5434 Hz		6367 Hz		7604 Hz		9071 Hz	
		RE a4 b1	LE a2 b2	RE a2	LE a1 b1	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
G1 - Non Exposed (n = 18)	n	0	0	2	2	3	2	5	3	2	1	2	3	5	2	6	5	7	5	8	4	9	7	7	2
	%	0.0	0.0	12.5	11.8	16.7	11.1	27.8	16.7	**11.1**	5.6	**11.1**	16.7	27.8	11.1	33.3	27.8	38.9	27.8	44.4	22.2	50.0	38.9	38.9	11,1
G2 - Exposed (n = 19)	n	1	1	2	0	2	1	5	3	9	2	9	4	8	3	8	7	10	9	7	9	10	10	7	4
	%	5.6	5.9	10.5	0.0	10.5	5.3	26.3	15.8	**47.4**	10.5	**47.4**	21.1	42.1	15.8	42.1	36.8	52.6	47.4	36.8	47.4	52.6	52.6	36.8	21,1
p value [a]		*pc*	*pc*	*pc*	*pc*	*0.47*	*pc*	*0.60*	*0.6*	***0***	*pc*	***0***	*0.5*	*0.36*	*0.52*	*0.58*	*0.55*	*0.40*	*0.21*	*0.63*	*0.10*	*0.87*	*0.40*	*0.89*	*0.35*

fc: few cases < 5 patients with presence or absence of variable.
[a1] loss of one patient in G1, [a2] loss of two patients in G1, [a4] loss of four patients in G1.
[b1] loss of one patient in G2, [b2] loss of two patients in G2.
χ^2 test or Fisher's exact.
Value p=0,05

DISCUSSION

Measurements

In this paper, evaluations of exposure to noise proved higher than the tolerance limit (85dBA) established by Standard Regulating Norm nº 15 [20,21]. It was also found that maximum NPS peaks between 4 to 7 p.m. were registered in the histogram, even reaching 122 dBA. It should be highlighted that the Threshold Limit Value of Exposure (TLV) for continuous or intermittent noise is 115 dB, this is, the maximum value found is elevated, above, this exposure is not permitted at any time during the workday for individuals who are not adequately protected, in spite of the values obtained for the daily dose or for the level of exposure [21].

Finding from scanning electronic microscopy showed that the most damaged areas by this type of exposure were between 7 and 13 mm from the apex of the cochlea, corresponding to the regions of frequencies between 0.8 and 5 KHz. These studies reveal that after a four-second exposure, the cochlea presents lesions restricted to Deiters and Hensen cells and, after 24 hours after the end of exposure, large portions of the cochlea did not recover from mechanical trauma and began to degenerate [17].

Concerning the results obtained from samples of CO concentration, it was primarily found the presence of this chemical agent in the working environment and variations that lead us to reflect upon the discussion that remains: How much smaller should the concentration of CO be so as to produce the potentialization, particularly in human beings. This answer would partly depend on the selection of criteria to determine the reference between concentration and time of exposure, for the results of these surveys were collected within four weeks of experimental exposure [23].

Another important data to be discussed is that studies show that hearing rest intervals are important to avoid permanent damage, as they provide the "recovery" of the cochlea. Thus, it seems that intermittent noise causes fewer changes in the hearing threshold than the continuous noise of equal intensity; however, when the exposure to noise happens in combination with CO, there are no differences between the effects of intermittent or continuous noise [24].

It is believed that it is necessary to carry out a longitudinal study where the results of systematic collection of samples of the gradients of exposure to these agents, with concomitant biological monitoring. In both cases, methodologies used in scientific research should be obeyed in order to facilitate the understanding of the relations between dose and emergence or extension of hearing damage; a crucial tool for control and prevention measures.

Anamnesis

In this study, the absence of a significant difference between the age ranges registered between the groups provided a more unbiased analysis of results in the auditory aspects related to natural aging of the ear in the two proposed audiological tests, mainly because of the presence of controversy about the possible influence of age in otoacoustic emissions, due to changes in cochlear biomechanics and/or loss of outer hair cells, observed throughout life [25].

In history data, the percentage of complaint about tinnitus, 16.7% in G1 and 10.5% in G2 called our attention. This symptom still features scarce epidemiological data; yet, it is considered the third worst symptom that can affect human beings, surpassed only by incurable pains and intense dizziness [26].

A study that sought to associate hearing loss with high sound pressure levels to tinnitus complaints reported that about 25% of the individuals mentioned this problem [27]. The prevalence of 48% in a population of workers was found in another research. In the latter, a statistic model used associated hearing loss to the occurrence of tinnitus [26]. In this study, apart from the relation Hearing loss induced by high sound pressure level and tinnitus, it was also observed a dose-response relation between hearing loss and tinnitus [26]. These studies report important and differentiated percentages, and it is important to remember that in this research, other factors that could interfere in the etiology of tinnitus were excluded [28], and previous exposure to impact noise for both groups was considered a factor that could be related to such claim.

Audiometric Assessment

Concerning the study of pure tone thresholds, similarities in the results obtained at frequency of 1 KHz in the right ear for group G2 were not to be found in other research papers. However, in studies on combined exposure to noise and CO in rats exposed to 105 dB/1 hour, it was observed that there was an impairment of both low and high frequency ranges [23], which denotes the diffusion length of the cochlea lesion from its base to its apex, including the involvement of medium frequencies.

Research has shown that the exposure to carbon monoxide alone does not cause changes in hearing thresholds, though it was observed that in the combined exposure to noise and carbon monoxide a linear relationship between the increase in CO concentration and extension of potentiation of noise-induced hearing loss when noise level were maintained. The same did not occur when the levels of noise and maintained CO (non-linear relationship) were increased. Studies have shown that the higher the CO dose, keeping the same level of noise, the auditory damage proved equally and gradually higher, extending the low frequencies even after a single exposure [29].

Still in the statistical analysis of hearing thresholds, significant differences in the left ear with respect to group G2, the frequencies of 3 KHz ($p = 0.016$) and 4 KHz ($p = 0.028$) can be observed. These results are supported by research that showed significant differences ($p < 0.01$) in auditory thresholds of groups exposed to CO and noise, with respect to the non-exposed group, precisely at the sharp frequencies 3K, 4K and 6 KHz [30].

Similar findings in another recent study made evident that workers exposed to CO and noise had significant differences ($p < 0.05$) in hearing thresholds, particularly at high frequencies (3K, 4K and 6K) [31].

In the audiometric qualitative analysis, the present study clearly showed a statistically significant higher difference for changed results changed only for frequency 3 KHz in the left ear (36.8%, $p = 0.003$) for G2 in relation to G1 (5.6%). This feature tends to a fact which is exclusively related to noise:

Most of the exposure to occupational noise has a broad spectrum of frequencies and the resonance of the external auditory canal is located at around 3000 Hz, so these signals would be amplified, causing the basal region of the cochlea the most vulnerable to intense sounds [32].

It should be pointed out that in none of the studies aforementioned so far there is explicit reference to the predominance of occurrence in only one of the ears, as it can be found in the results of this paper. However, in another study, asymmetries between the ears in hearing losses related to noise exposure were found, and they sought ground either in the position between the emission source and the worker, or in a likely susceptibility of the left ear, yet with little evidence, the possible physiological mechanisms for this difference appear to be unknown [33].

Although in this paper we did not ascertain significant differences in the level 5% in proportion to chances in the remaining frequencies of the tone thresholds, there was a higher incidence of hearing loss in the group exposed to carbon monoxide and noise, especially at the frequencies 3K, 4K and 6 KHz in both ears. This alteration profile is expected in hearing loss induced by isolated exposure to high sound pressure levels and also in concomitant exposure to asphyxiating gases, attributing higher sensitivity in the first third of the cochlea to metabolic, anatomical and vascular damage, as aforementioned.

Distortion-product Evoked Otoacoustic Emissions

This profile of a higher incidence of altered responses in DPOAEs in the groups studied here, occurred from 2.730 Hz in both ears to the members of group G2, equating to what was observed in the changes of hearing thresholds.

The other results of statistical significance with respect to the absence of responses for frequencies only in the right ear at 2.730 Hz and 3.218 Hz (47%, p = 0.016), differ from expected results when compared to audiometric findings, which showed a higher statistically significant difference only at frequency 3 KHz in the left ear (36.8%, p = 0.003) for G2.

A group of studies on the toxic action of CO on the auditory system show that CO may have a direct effect on the cochlear metabolism which would account for the potentiation of hearing impairment by metabolic exhaustion of the enzyme succinate dehydrogenase involved in the Krebs Cycle (breathing) of sensory cells, particularly the CCE [34,35] and the marginal cells of stria vascularis [34], the excitotoxicity attributed to excessive relaxation of the neurotransmitter glutamate from the inner hair cells (ICC) towards the auditory nerve fibers [34,36,37]; and the oxidation of nervous structures by the production of free radicals [37,38].

The results presented in Tables 3 and 4 (qualitative results of the audiometry and otoacoustic emissions, respectively) allow us to observe a higher occurrence of altered responses in otoacoustic emissions, both in G1 and in G2, when comparing the number of recorded changes in hearing thresholds in both groups, with a especially higher prevalence in G2. Thus, it seems to us it would be correct to affirm that the DPOAEs are more sensitive in detecting cochlear dysfunctions, since the outer hair cells are the first structure of the inner ear to be damaged by external agents, even before the record of changes in audiometric thresholds [39,40].

The data analyzed here lead us to believe that the injuries in the audiometric profiles and in the otoacoustic emissions found in the group of workers exposed may be correlated to the concomitant exposure to carbon monoxide and noise. The data also reveal a need for studies in human beings, making use of both technological advances in the field of audiology (aiming at deeper and more precise analysis in the auditory function), as well as in the field of occupational hygiene (for consistent information gathering about the gradients of exposures), in search for information that, when correlated, may support reflection and revision of the limits defined as safe for simultaneous exposure of the agents studied here.

CONCLUSION

It was ascertained the presence of CO and noise in the working environment of the transit managers assessed.

The group of workers exposed to carbon monoxide and noise showed significant differences (worsening) in both the pure tone thresholds and the responses in distortion-product Evoked otoacoustic emissions when compared to workers non-exposed to the two agents.

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RESUMO

Objetivo: avaliar o perfil audiométrico e de emissões otoacústicas evocadas por produto de distorção em gestores de trânsito, expostos a monóxido de carbono e ruído, bem como constatar a presença de ambos agentes nos postos de trabalho. **Método:** estudo transversal, descritivo, com 37 gestores do trânsito, submetidos a anamnese, meatoscopia, audiometria tonal e emissões otoacústicas, distribuídos em: G1, composto por 18 indivíduos sem histórico de exposição concomitante a monóxido de carbono e ruído; e, G2, formado por 19 trabalhadores expostos simultaneamente aos dois agentes. Para pesquisa da presença dos agentes no ambiente foram utilizadas audiodosímetrias e avaliações de curta duração com medidor instantâneo. As variáveis de anamnese foram analisadas segundo o teste *t Student* e Mann-Whitney. Para as medidas de otoemissões acústicas e de limiares tonais utilizou-se testes de qui-quadrado ($\chi 2$) ou exato de Fisher e dos postos sinalizados de Wilcoxon com significância de 5%. **Resultados:** foi constatada presença de monóxido de carbono e ruído durante a atividade dos trabalhadores. Não foi observada diferença significante na idade e tempo de função. O G2 obteve média de limiares tonais maior que G1, para orelha direita, em 1KHz (p=0,050) e para orelha esquerda em 3KHz (p=0,016) e 4KHz (p=0,028); e, comparados os limiares tonais alterados G2 apresentou diferença maior em 3KHz na orelha esquerda (p=0,003). Nas emissões otoacústicas, G2 apresentou maior ausência de respostas que G1 em 2.730Hz e 3.218Hz (p=0.016) para orelha direita. **Conclusão:** trabalhadores expostos a monóxido de carbono e ruído apresentaram piores resultados audiométricos e nas emissões otoacústicas quando comparado ao grupo de não expostos.

DESCRITORES: Perda Auditiva; Monóxido de Carbono; Sinergismo Farmacológico; Gases Asfixiantes; Audiometria

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