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Steady-State Auditory Evoked Potentials in Workers Exposed to Occupational Noise

Mariana Keiko Kamita[1*], Clayton henrique Rocha[1], Liliane Aparecida Fagundes Silva[1], Alessandra Giannella Samelli[1], Carla Gentile Matas[1]

[1]Department of Physiotherapy, Speech-Language and Hearing Sciences and Occupation Therapy, Faculdade de Medicina FMUSP, Universidade de São Paulo, São Paulo, SP, Brazil

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Abstract

Background: *The present study aims to investigate neural synchrony, as measured by Auditory Steady State Response (ASSR), in individuals with normal hearing who are exposed and not exposed to occupational noise, thereby providing insights into hidden hearing loss within the central auditory nervous system, and justifying the importance of exploring auditory neural function in populations at risk.* **Methods:** *A cross-sectional study involved 30 noise-exposed individuals in the Study Group and 30 unexposed individuals in the Control Group, all paired by an average age of 35 years. The following procedures were performed on all individuals: clinical and occupational history, meatoscopy, immitanciometry, pure tone audiometry, speech audiometry, and ASSR (40Hz). We analyzed the audiometric hearing thresholds at frequencies of 1 kHz and 4 kHz, the electrophysiological thresholds estimated by ASSR, and the comparison of the differences between them: the thresholds estimated by ASSR and the audiometry thresholds. The data were analyzed using both descriptive and inferential statistics. P-values ≤ 0.05 were considered significant.* **Results:** *When comparing hearing thresholds at 1 kHz and 4 kHz between groups, we found significant differences, with the SG showing higher hearing thresholds than the CG bilaterally. No significant differences were seen in the electrophysiological thresholds estimated by ASSR, nor in the comparison between the ASSR-estimated threshold and the psychoacoustic hearing threshold groups.* **Conclusions:** *The results of this study suggest that workers exposed to occupational noise did not show detectable changes in neural synchrony in the midbrain, thalamus, or primary auditory cortex when compared to individuals without occupational noise exposure.*

1. Introduction

The initial studies on the pathophysiology of noise-induced hearing loss used animal models. They showed that noise exposure mainly causes mechanical damage to inner ear structures, ischemia, and ionic imbalances, with oxidative stress also playing a role. New prevention strategies include antioxidants [1-6].

Later animal studies revealed that noise can induce a temporary threshold shift (TTS) but cause permanent degeneration of presynaptic structures and spiral ganglion neurons. At the same time, hair cells remain intact—indicating synaptopathy [7]. This might involve high- and low-spontaneous-rate auditory fibers connecting with inner hair cells; low-spontaneous-rate fibers are more vulnerable, leading to synapse loss without affecting hearing thresholds,

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* Corresponding Author: Mariana Kamita; E-mail: marikeikokamita@gmail.com

as high-spontaneous-rate fibers remain functional [2, 4, 8, 9]. Such damage may result in difficulties understanding speech amidst noise, problems perceiving temporal cues, hyperacusis, or tinnitus [2, 4, 6, 9-11]. Still, the pathophysiology of hidden hearing loss (HHL) in humans remains unclear [12]; further research using audiological tests, especially those assessing the central auditory nervous system (CANS), is needed [2-4, 6].

Auditory evoked potentials (AEPs), widely used electrophysiological tools in neuroscience, can evaluate the CANS. Among these, the auditory steady state response (ASSR) is instrumental, as it relies on neural synchrony related to sound localization, pitch, and speech-in-noise perception, and can be recorded at various modulation frequencies to assess temporal processing [13-16]. ASSR helps analyze acoustic transmission and detect neural dyssynchrony, which may result from synaptopathy at the auditory nerve's first synapse or along the pathway, potentially causing dyssynchrony throughout [17].

A previous study found that noise-exposed individuals with normal hearing performed worse on speech perception tests than non-exposed controls, possibly due to noise-induced impairments in neural synchrony not reflected in tonal thresholds, suggesting a link to HHL. Therefore, we hypothesize that workers exposed to occupational noise, without hearing threshold impairment, show altered neural synchrony, which can be demonstrated in ASSR recordings. The objective of this study was to evaluate neural synchrony through ASSR in normal-hearing individuals exposed and not exposed to occupational noise, aiming to gain insights into HHL in the CANS.

2. Methods

This cross-sectional study was approved by the institutional research ethics committee (No. 2.435.259), and the research was conducted in accordance with the Declaration of Helsinki. All subjects included in the study received prior explanations about the research, and after agreeing to the terms, they signed the informed consent form.

The present research is part of a larger study that conducts several peripheral and central audiological assessments in normal-hearing adults exposed to occupational noise, to investigate audiological findings and possible changes resulting from noise exposure.

The study included 60 normal-hearing individuals divided into two groups paired by age: the study group (SG) comprised 30 individuals exposed to occupational noise and with a mean age of 35.60 years, and the control group (CG) comprised 30 individuals not exposed to occupational noise and with a mean age of 35.37 years.

The inclusion criteria for both groups were: male individuals with normal-hearing thresholds bilaterally, absence of earwax and middle ear alterations, aged at least 18 years old and no more than 50 years, with no history of ear diseases or surgeries, no tinnitus, not taking medication or using potentially ototoxic treatments, and no exposure to chemical products. Furthermore, in addition to the aforementioned criteria, the SG required exposure to occupational noise above 85 dB HL for one year or more. In contrast, the CG required the absence of such exposure.

The workers were chosen based on the university's environmental risks prevention program, which outlines the risks each worker faces during their workday. The SG included individuals who worked in maintenance at the university and were exposed to intermittent noise (Lavg [average sound pressure level over a period of time]: 88 dBA; minimum: 75 dBA, maximum: 111 dBA; 69% of the daily dose) during their 8-hour workday for an average of 8.6 years (SD: 6.1 years) in their current position. All of them used Hearing Protection Devices (HPDs) during their shifts, with the plug type being most common (70%). The CG also consisted of workers from the same institution, but from departments without noise exposure, mostly from administrative sectors. The detailed characterization of the sample is shown in Table 1, and the history of occupational and non-occupational noise exposure is shown in Table 2.

Initially, the following procedures were performed: clinical and occupational history; meatoscopy (Mini 3000, Heine); acoustic immittance measurements (AT235, Interacoustics), including tympanometry and the assessment of ipsi- and contralateral acoustic reflexes; and pure tone audiometry (PTA)

Table 1. Characterization of the sample.

Variable		Study Group (n=30)	Control Group (n=30)
Age range (in years)	*Mean*	35.60	35.37
	SD	7.10	7.56
	Minimum	23.00	22.00
	Maximum	50.00	49.00
Educational level, n (%)	*High School*	14 (46.7%)	6 (20.0%)
	Technical Education	3 (10.0%)	1 (3.3%)
	Incomplete Higher Education	7 (23.3%)	2 (6.7%)
	Higher Education Complete	6 (20.0%)	21 (70.0%)
Complaints & otologic history n (%)	*Hyperacusis*	3 (10.0%)	0 (0.0%)
	Itching	3 (10.0%)	5 (17.0%)
	Difficulty listening in noise	3 (10.0%)	0 (0.0%)

Legend: n- Sample number; %- Percentage.

Table 2. History of occupational and non-occupational exposure to noise.

		Study Group (n=30)	Control Group (n=30)
Occupational exposure to noise (in years)	*Mean*	13.86	Not applicable
	SD	8.33	
	Minimum	1.00	
	Maximum	32.00	
Non-occupational exposure to noise, n (%)	*Headphones*	17 (56.7%)	14 (46.7%)
	Stadium/Autodrome	5 (16.7%)	5 (16.7%)
	Church	12 (40%)	7 (23.3%)
	Shows/Parties	5 (16.7%)	13 (43.3%)

Legend: n- Sample number; %- Percentage.

(MA42, MAICO), where hearing thresholds were considered within the normal range of ≤25 dB HL at 250 to 8000 Hz [19].

Once the individuals received normal results in the assessments mentioned above and met the inclusion criteria, they underwent an electrophysiological assessment using the ASSR with the intelligent hearing system (ANSI S3.7-1996) Smart EP model, conducted in an acoustically treated room. The individual was seated comfortably in a reclining chair.

First, the skin was cleaned with abrasive paste at the electrode placement sites, following international standard IES 10-20 [20]: on the vertex (Cz) for the active electrode, on the mastoids of the left (M 1) and right (M 2) ears for the reference electrodes, and on the forehead (Fpz) for the ground electrode. Electrodes were attached using electrolytic paste and microporous tape, ensuring impedance values of less than 5 kOhms. The patient received the acoustic stimulus through ER-3 A insert earphones, stimulating both ears simultaneously.

The ASSR was performed with modulation at 40 Hz, using sinusoidal acoustic stimulation with 100% amplitude modulation and 100% frequency modulation at frequencies of 1 and 4 kHz. Up to 400 stimuli were presented, divided into 20 sweeps of 20 stimuli each, with high-pass and low-pass filters set from 30 to 3000 Hz.

Evaluation began at 1 kHz, followed by 4 kHz. The initial intensity was set at 80 dBnHL, decreasing by 10 dBnHL steps until the electrophysiological threshold was found for each ear, especially if one ear responded and the other did not.

The electrophysiological thresholds (dB SPL) were converted into estimated thresholds (dB HL) based on ISO 389-2, with corrections of 0 dB at 1 kHz and -6 dB at 4 kHz, as used in other studies [21, 22]. The difference between the estimated electrophysiological threshold and the audiometric threshold was then calculated.

Data were analyzed using both descriptive and inferential statistical methods. The Shapiro-Wilk test was used to assess the distribution of the sample, complemented by visual inspection of histograms. Since the data followed a normal distribution pattern, parametric tests were used for further analysis. A repeated measures ANOVA was performed, with the ear as the repeated measure and the group as the between-subjects factor. Variance equality was checked with Levene's test, and sphericity was verified with Mauchly's test. If necessary, the Greenhouse-Geisser correction was applied. Effect sizes were evaluated using eta squared (η^2), following Cohen's (1988) guidelines: $\eta^2 = 0.01$ for a small effect; $\eta^2 = 0.06$ for a medium effect; and $\eta^2 = 0.14$ for a large effect.

3. Results

Initially, a descriptive analysis was carried out of the values obtained in the PTA at 1 and 4 kHz (Table 3) and the estimated thresholds obtained by the ASSR (Table 4).

The comparison analysis showed a statistically significant difference between the groups for the thresholds obtained in the PTA at both 1 kHz and 4 kHz, with a medium effect size in both cases, regardless of which ear was assessed (Table 5; Figure 1). As for the estimated thresholds achieved by ASSR, there were no significant differences either between the ears or between the groups (Table 5).

Regarding the difference between the threshold estimated in the ASSR (Table 4) and the threshold obtained through the ATL (Table 3), there was no statistically significant difference between the groups or between the ears evaluated (Table 6).

4. Discussion

Regarding the data described in scientific literature about HHL in humans, a source of uncertainty relies on indirect electrophysiological measurements, because unlike animal studies, cochlear synaptopathy cannot be directly measured in vivo. Additionally, some authors have suggested that these changes trigger a series of alterations in neural processing in regions posterior to these in the CANS [23]. Therefore, more recent studies have included, among other behavioral tests in noisy environments, measures of function using evoked potentials, mainly auditory brainstem response (ABR), as well as envelope following response (EFR). Currently, audiometry remains the gold standard clinical tool in audiology. However, it is known that this dysfunction can be

Table 3. Descriptive analysis of PTA thresholds for the 1 and 4 kHz frequencies by group and ear.

	Ear	Group	Mean	SD	Minimum	Maximum
1 kHz	Right	Control	7.67	5.68	0	20
		Study	10.33	4.54	0	20
	Left	Control	6.83	5.00	0	20
		Study	10.00	5.09	0	15
4 kHz	Right	Control	9.33	6.40	0	20
		Study	13.00	7.02	0	25
	Left	Control	9.03	6.31	0	20
		Study	12.50	7.63	0	25

Legend: kHz = kilo Hertz.

Table 4. Descriptive analysis of the estimated ASSR thresholds for the 1 and 4 kHz frequencies by group and ear.

	Ear	Group	Mean	Standard deviation	Minimum	Maximum
1 kHz	Right	Control	27.3	7.85	20	40
		Study	26.3	7.65	20	40
	Left	Control	26.0	6.75	20	40
		Study	27.3	10.15	20	60
4 kHz	Right	Control	23.0	13.73	14	54
		Study	25.0	11.55	14	54
	Left	Control	25.7	16.42	14	44
		Study	23.7	10.98	14	44

Legend: kHz- kilo Hertz.

Table 5. Comparison of the thresholds obtained in the PTA and the estimated ASSR threshold at each frequency according to group and ear.

		Factor	Sum of squares	*F*	p	η^2
PTA threshold	1 kHz	Group	255.208	5.904	0.018*	0.078
		Ear	10.208	1.183	0.281	0,003
		Group x Ear	1.875	0.217	0.643	<0.001
	4 kHz	Group	381.633	4.818	0.032*	0.065
		Ear	4.800	0.323	0.572	<0.001
		Group x Ear	0.300	0.020	0.888	<0.001
ASSR threshold	1 kHz	Group	0.833	0.008	0.931	<0.001
		Ear	0.834	0.034	0.854	<0.001
		Group x Ear	40.833	1.682	0.200	0.005
	4 kHz	Group	<0.001	<0.001	>0.999	<0.001
		Ear	13.333	0.270	0.605	<0.001
		Group x Ear	120.000	2.428	0.125	0.006

*Legend: kHz- kilo Hertz; * statistically significant difference;* η2*- Eta squared refers to the size of the effect.*

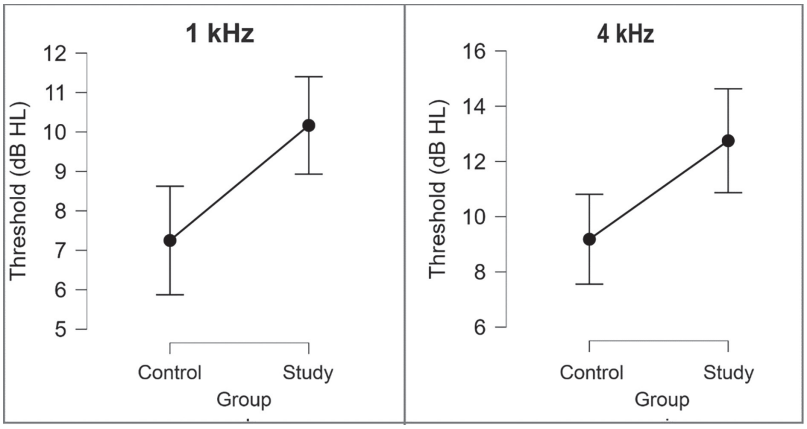

Figure 1. Comparison (in decibel Hearing Level – dB HL) of ATL thresholds between both groups in 1 kHz e 4 kHz (kilo Hertz).

Table 6. Comparison of the difference between the thresholds estimated by ASSR and the PTA threshold for the 1 and 4 kHz frequencies by group and ear.

	Factor	Sum of squares	*F*	*P*	η^2
1 kHz	Group	226,875	2,298	0,135	0,029
	Ear	5,208	0,156	0,694	<0,001
	Group x Ear	25,208	0,757	0,388	0,003
4 kHz	Group	381,633	1,208	0,276	0,017
	Ear	34,133	0,524	0,472	0,002
	Group x Ear	108,300	1,664	0,202	0,005

Legend: kHz- kilo Hertz; η*2- Eta squared refers to the size of the effect.*

"hidden" in the presence of a normal audiogram [12]. Following this trend, and aiming to clarify some of the gaps surrounding the topic, this study aimed to assess neural synchrony through ASSR in normal-hearing individuals exposed and not exposed to occupational noise, in order to identify possible insights into HHL in the CANS, since individuals with central impairment may have a lower agreement between psychoacoustic and electrophysiological estimated thresholds [24].

The study found no significant differences in electrophysiological thresholds between workers exposed and not exposed to occupational noise. However, the exposed group showed higher audiometric thresholds at 1 kHz and 4 kHz compared to the control group. Contrary to the initial hypothesis, which predicted poorer neural synchrony and greater discrepancies between audiometric and electrophysiological thresholds in the exposed group, both thresholds were similar across groups. These findings support what was observed by Grose et al. [25], who conducted ABR, ASSR, and behavioral assessments with speech tests in individuals who frequently attended noisy environments. They found that noise exposure did not lead to changes in any of the behavioral or electrophysiological assessments (ABR and ASSR). The authors emphasized that it was not possible to detect any findings consistent with HHL in their study. However, they suggested that musical experience might have influenced the responses, as many participants in the study group were members of rock bands, and the results could have been affected by the benefits of musical training.

Guest et al. [26], who evaluated individuals with speech perception difficulties in noise using behavioral tests, a questionnaire on previous noise exposure, ABR, and ASSR, observed no correlation between lifetime noise exposure and difficulty listening in noise, as well as the findings of ABR and ASSR. It should be noted that, although HHL likely occurs in humans, no changes in ASSR were observed in the studies mentioned earlier [25,26], including the present one. Guest et al. [26] proposed that potential changes—such as a reduction in the amplitude of wave I of the ABR, which may happen in individuals exposed to noise—do not necessarily result in other detectable alterations along the auditory pathway. Alternatively, ASSR might not be the most sensitive audiological measure for detecting HHL in humans.

On the other hand, Shaheen et al. [27] reported that ASSR has a high sensitivity in detecting HHL in animal models. They assessed distortion product otoacoustic emissions, ABR, and ASSR in mice exposed to noise and noted that only the ASSR test showed altered results, with a reduction in amplitude values. However, it should be noted that the study was conducted on mice living in a controlled environment exposed to noise, and its results were compared with those of mice without noise exposure, who also lived in a controlled environment. This comparison is not possible to carry out on humans.

Furthermore, previous studies have shown that non-human primates are less vulnerable to noise-induced hair cell loss than rodents, although susceptibility to synaptopathy has not yet been investigated [28]. However, this indicates that different species

respond differently to noise exposure and, therefore, more subtle effects of damage to the auditory pathway may not be as easily detected in assessments across all species.

In this way, it is suggested that it is not possible to observe altered ASSR results in humans in the same way as in rodent animals, since occupational and non-occupational noise exposure cannot be measured equivalently. There is also the possibility that humans have auditory system structures that are less susceptible to possible alterations resulting from noise exposure [29].

Despite this, research on humans has yielded findings that differ from those in the present study. Bharadwaj et al. [30], aiming to find an association between hearing assessments and HHL, observed a strong correlation between ASSR and the behavioral performance of individuals exposed to noise with normal hearing. They suggested that the measures obtained by ASSR are sensitive and promising for assessing HHL [30]. Similarly, a study by Mepani et al. [31], involving normal-hearing individuals aged 18 to 63 years with no complaints or history of hearing disorders, found that ASSR has good sensitivity across a range of responses for assessing HHL and that these findings correlate well with behavioral assessments. This suggests that these individuals may have a cochlear or neural deficit and a limited ability to decode words in challenging listening environments.

This difference in findings between ASSR studies may be due to the methods used and the characteristics of the populations studied. Unlike our research, which used an ASSR with a modulation frequency of 40 Hz, Bharadwaj et al. [30] employed a modulation frequency of 100 Hz, and Mepani et al. [31] used frequencies of 128 Hz or 750 Hz, assessing different parts of the CANS.

Along with neural desynchronization, previous research has also identified difficulties in speech understanding in noisy settings within this group. Neural signal transmission from inner hair cells to the auditory nerve occurs across synapses. It is now known that intense noise exposure can selectively harm these synapses, often without a corresponding loss of hair cells. Clinically, this condition exhibits normal audiometric thresholds; however, it presents significant problems in speech perception, particularly in noisy environments. The authors suggest that these issues may be caused by noise exposure, which results in neurodegeneration of spiral ganglion neurons, thereby impairing speech comprehension in such conditions [7, 11, 30, 31].

In the present study, we did not conduct a behavioral assessment, so such a comparison is not possible. However, we observed that 10% of individuals exposed to occupational noise reported difficulty listening in noise, while none of the individuals in the CG did. This suggests that these workers may have some dysfunction in the auditory system. Future studies that incorporate electrophysiological assessments, such as ASSR, alongside behavioral assessments, could help clarify this topic.

Several limitations must be considered in our study, including the lack of detailed information about the duration and level of exposure to extra-occupational noise, as these factors can influence the results for both the control and study groups. Additionally, it is essential to note that the workers participating in this study wore HPDs at work, which may have helped preserve or reduce the risk of noise exposure to the CANS. However, it is known that the use of HPDs is not always practical or consistent across all workplaces or occupational settings (Morata et al, 2024) [32]. Therefore, further research that includes this variable is necessary. Furthermore, the results related to neural synchrony among noise-exposed workers who wear HPDs cannot be generalized to individuals without hearing protection, such as drivers or those exposed to non-occupational noise, nor to those using different types of HPDs. Future studies should examine these specific groups and explore how various types of HPDs might prevent neural synchrony damage, even in cases of lifelong noise exposure. Additionally, the duration of occupational noise exposure should be investigated, as longitudinal studies are essential to assess potential injury to the CANS and its compensatory mechanisms over time.

5. Conclusion

The results obtained in this study suggested that workers exposed to occupational noise did not show detectable changes by ASSR in neural synchrony in

the midbrain, thalamus or primary auditory cortex when compared to individuals without exposure to occupational noise.

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