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# Long-latency auditory evoked potentials-verbal and cortical gain in patients with tinnitus

## Potencial evocado auditivo de longa latência-verbal e ganho cortical em indivíduos com zumbido

## Potencial evocado auditivo verbal de latencia larga y ganancia cortical en individuos con tinnitus

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***Abstract***

**Objective:** To analyze whether the presence of tinnitus can alter the amplitudes and the P2/P1 wave ratio in the cortical Long-Latency Auditory Evoked Potential (LLAEP) with verbal stimulus in young adults. **Methodology:** Observational, analytical, cross-sectional study with a convenience sample consisting of educated, right-handed patients without hearing loss and without auditory complaints other than tinnitus. Patients undergoing pharmacological treatment for tinnitus or presenting conditions that could compromise the research were excluded. Twenty individuals participated, divided into two groups: study group (SG) [7 women/5 men aged 19–35 years (mean = 24 years); 11 right ears and 12 left ears were evaluated; 11 cases of bilateral tinnitus and 1 case of unilateral tinnitus in the left ear]; control group

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**Authors´contributions:**
HGM: study conception, methodology; data collection and article design.
LC, BRM, JHZ: data collection; article design.
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(CG) [5 women/3 men aged 19–35 years (mean = 25 years); 8 right ears and 8 left ears were evaluated]. All participants underwent basic audiological assessment, central auditory processing evaluation, neuropsychological assessment, transient otoacoustic emissions, brainstem auditory evoked potential, and the LLAEP as the research procedure. Data analysis was performed using the Mann-Whitney U test, adopting a significance level of $p \leq 0.05$. **Results:** Statistically significant differences were observed only for the P2 component in the left ear, with mean values of 4.42 for the control group and 6.39 for the study group ($p = 0.017$). **Conclusion:** The presence of tinnitus in young adults with normal audiometry was associated with higher amplitude of the P2 component in the LLAEP with verbal stimulus, specifically in the left ear. However, no significant changes were observed in the P2/P1 ratio between the groups.

**Keywords**: Tinnitus; Auditory Evoked Potentials; Adult; Central Nervous System; Auditory Cortex.

## *Resumo*

**Objetivo:** Analisar se a presença do zumbido pode alterar as amplitudes e a relação das ondas P2/P1 no Potencial Evocado Auditivo de Longa Latência (PEALL) cortical com estímulo verbal em adultos jovens. **Metodologia:** estudo observacional de corte transversal analítico, com amostra de conveniência composta por pacientes escolarizados, destros, sem perda auditiva e sem queixas auditivas além do zumbido. Foram excluídos pacientes em tratamento farmacológico para o zumbido ou com quadros que pudessem comprometer a pesquisa. Participaram 20 indivíduos divididos em dois grupos: grupo estudo (GE) [7 mulheres/ 5 homens de 19-35 anos (média= 24 anos), foram avaliadas 11 orelhas direita e 12 orelhas esquerdas; 11 casos de zumbido bilateral e 1 caso de zumbido unilateral na OE]; grupo controle (GC) [5 mulheres/ 3 homens de 19-35 anos (média= 25 anos); foram avaliadas 8 orelhas direitas e 8 orelhas esquerdas. Todos os indivíduos submeteram-se a avaliações: audiológica básica, processamento auditivo central, neuropsicológica, emissões otoacústicas transientes, potencial evocado auditivo de tronco encefálico e, como procedimento de pesquisa, o PEALL. A análise dos dados foi realizada por meio do teste U de Mann-Whitney, adotando p-valor ≤0,05. **Resultados:** Foram observadas diferenças estatisticamente significantes somente para componente P2 na orelha esquerda, com valores médios de 4,42 para o grupo controle e 6,39 para o grupo estudo (p-valor= 0,017). **Conclusão:** A presença do zumbido em adultos jovens com audiometria normal esteve associada a maior amplitude do componente P2 no PEALL com estímulo verbal, especificamente na orelha esquerda. Contudo, não foram observadas alterações significativas na relação P2/P1 entre os grupos.

**Palavras-chave:** Zumbido; Potenciais Evocados Auditivos; Adulto; Sistema Nervoso Central; Córtex auditivo.

## *Resumen*

**Objetivo:** Analizar si la presencia de acúfeno puede alterar las amplitudes y la relación de las ondas P2/P1 en el Potencial Evocado Auditivo de Larga Latencia (PEALL) cortical con estímulo verbal en adultos jóvenes. **Metodología:** Estudio observacional, analítico y transversal, con una muestra por conveniencia compuesta por pacientes escolarizados, diestros, sin pérdida auditiva y sin quejas auditivas además del acúfeno. Se excluyeron los pacientes que estaban en tratamiento farmacológico para el acúfeno o que presentaban condiciones que pudieran comprometer la investigación. Participaron 20 individuos, divididos en dos grupos: grupo de estudio (GE) [7 mujeres/5 hombres de 19 a 35 años (media = 24 años); se evaluaron 11 oídos derechos y 12 oídos izquierdos; 11 casos de acúfeno bilateral y 1 caso de acúfeno unilateral en el oído izquierdo]; grupo control (GC) [5 mujeres/3 hombres de 19 a 35 años (media = 25 años); se evaluaron 8 oídos derechos y 8 oídos izquierdos]. Todos los participantes se sometieron a evaluaciones audiológicas básicas, evaluación del procesamiento auditivo central, evaluación neuropsicológica, emisiones otoacústicas transitorias, potencial evocado auditivo de tronco encefálico y el PEALL como procedimiento de investigación. El análisis de los datos se realizó mediante la prueba U de Mann-Whitney, adoptando un valor de $p \leq 0,05$. **Resultados:** Se observaron diferencias estadísticamente significativas únicamente para el componente P2 en el oído izquierdo, con valores medios de 4,42 para el grupo control y 6,39 para el grupo de estudio ($p = 0,017$). **Conclusión:** La presencia de

acúfeno en adultos jóvenes con audiometría normal se asoció con una mayor amplitud del componente P2 en el PEALL con estímulo verbal, específicamente en el oído izquierdo. Sin embargo, no se observaron cambios significativos en la relación P2/P1 entre los grupos.

**Palabras clave:** Zumbido; Potenciales evocados auditivos; Adulto; Sistema nervioso central; Corteza auditiva.

## Introduction

Several theories seek to understand the neurophysiological mechanisms related to tinnitus perception[1]. Currently, it is known that 90% of cases are associated with reduced auditory input[2]. However, some individuals with tinnitus do not present abnormalities in conventional audiological evaluations[3]. Therefore, recent research has aimed to measure changes at the level of the Central Auditory Nervous System (CANS) resulting from the perception of this symptom[1,3,4].

Among the theories related to tinnitus perception, the central gain model stands out as the most widely accepted explanation for the pathophysiology of tinnitus[4]. This theory focuses on neural deafferentation mechanisms and suggests that minor changes in auditory pathway input result in multiple diffuse alterations in various brain areas, generating thalamocortical hyperactivity as well as increased activity in the primary and secondary auditory cortex, alongside enhanced neural synchrony[5]. In this context, neuroplastic reorganization occurs within the auditory pathway, increasing neural responsiveness to compensate for reductions, consequently leading to the perception and/or maintenance of the symptom. Other authors have also identified thalamocortical dysrhythmia as a key pathophysiological cause of tinnitus[6], which supports the central gain theory.

To investigate the underlying mechanisms of tinnitus in relation to neuroplasticity, researchers[3] use Auditory Evoked Potentials (AEPs). These are neuroelectric measures of the auditory pathway elicited by acoustic stimuli and recorded using surface electrodes[3,7]. These measures allow observation of neural recruitment and understanding of the changes occurring in the CANS. Additionally, AEPs enable visualization of the activation of structures involved in the symptom's pathophysiology, justifying the relevance of this evaluation.

Studies using Brainstem Auditory Evoked Potential (BAEP) with click stimuli have demonstrated that this test is promising for analyzing central gain by showing increased neural responsiveness, indicating neurobiological changes in brainstem structures[7]. One study suggests that the ratio between wave amplitudes in BAEP can serve as a reliable metric for objectively identifying tinnitus and as a biomarker of plasticity-related changes resulting from different treatments[8].

Furthermore, it is known that the Long-Latency Auditory Evoked Potential (LLAEP) plays an important role in analyzing patients with tinnitus, although little is known about neural responsiveness in this region [3,9]. This raises the question of whether LLAEP could also serve as a valuable diagnostic tool at the cortical level. LLAEP is used in clinical audiology to provide information about cortical structure functioning, reflecting the arrival of acoustic information to the auditory cortex and the beginning of cortical auditory processing, through the identification of peaks P1, N1, P2, N2, and P300 [10]. Given that tinnitus may cause alterations in thalamocortical regions and in the primary/secondary auditory cortex and that these regions are respectively associated with the P1 and P2 waves of LLAEP [11], it is possible to justify this research considering the importance of these structures and the need to understand their functioning, potentially introducing a new clinical analysis tool.

Additionally, verbal stimuli were used in the LLAEP in this study because tinnitus can cause changes in speech perception. It is also evident that verbal stimuli elicit greater neural recruitment, making this approach more relevant for this population[12].

The recording of cortical/endogenous auditory evoked potential (CAEPs) is relatively inexpensive, non-invasive, and a clinically feasible technique to objectively collect information about inhibitory and/or excitatory alterations in the CANS, and therefore about the nature of tinnitus. The study's hypothesis is that the presence of tinnitus in young adults is associated with significant alterations in wave amplitudes in cortical LLAEP, as diffuse disorganization may occur in multiple brain areas, especially in thalamocortical regions and the

primary/secondary auditory cortex, consequently altering neural responsiveness.

Thus, the objective of this study is to analyze whether the presence of tinnitus can alter the amplitudes and the P2/P1 wave ratio in cortical LLAEP with verbal stimuli in young adults.

## Method

This is an observational, analytical, cross-sectional study, conducted in accordance with Resolution No. 466/12 and approved by the Research Ethics Committee involving Human Subjects under protocol number 57700721.0.0000.5346. Participants were informed about the procedures to be performed and, upon agreement, signed the Informed Consent Form, authorizing their voluntary participation. This study followed the guidelines of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) tool.

Inclusion criteria for both groups were: individuals with a minimum of 12 years of formal education, right-handed, native Brazilian Portuguese speakers, with hearing thresholds within normal limits (up to 19 dBHL at all conventionally tested frequencies from 250 to 8000 Hz)[13], normal tympano-ossicular system mobility, normal contralateral acoustic stapedial reflexes, no self-reported hearing complaints, normal auditory and cognitive abilities, and intact cochlear and brainstem function.

For the study group, an additional inclusion criterion was the presence of unilateral or bilateral tinnitus for at least three months, with a score higher than 4 on the Visual Analogue Scale (VAS).

Exclusion criteria for both groups included: undergoing pharmacological treatment for tinnitus, noise exposure, dizziness complaints, objective tinnitus or pulsatile tinnitus suggesting vascular origin, neurological, psychiatric, or cognitive impairments (evident or diagnosed), or current infection with SARS-CoV-2.

The sample size was determined by convenience. Participants were recruited from the audiology outpatient clinic of a university clinic between July 2021 and May 2022.

A total of 77 individuals were assessed, with 57 excluded for not meeting inclusion criteria. Thus, 20 young adults of both sexes, matched by age and education, were included and divided into two groups:

- **Study Group (SG):** composed of 7 females and 5 males, aged 19–35 years (mean = 24 years ± 2.08). A total of 11 right ears (RE) and 12 left ears (LE) were evaluated. Eleven participants had bilateral tinnitus, and one had unilateral tinnitus in the left ear.
- **Control Group (CG):** composed of 5 females and 3 males, aged 19–35 years (mean = 25 years ± 4.08). Eight right ears and eight left ears were evaluated.

All participants underwent a semi-structured anamnesis, pure-tone audiometry 250–8000 Hz[13], speech audiometry [13] and acoustic immittance measurements (tympanometry according to Jerger, Jerger & Mauldin[13], classification, and contralateral acoustic stapedial reflexes per Jerger & Jerger criteria[13]), neuropsychological assessment, behavioral assessment of central auditory processing skills, transient otoacoustic emissions (TOAEs), BAEP, and the LLAEP with verbal stimuli as the research procedure. The tinnitus group also completed the VAS.

Procedures were conducted over two sessions of approximately two hours each. The first session involved audiological diagnosis, cognitive assessment, and behavioral evaluation of central auditory processing. The second session (one week later) consisted of electroacoustic and electrophysiological procedures: BAEP-click, TOAEs, and LLAEP-verbal

The neuropsychological assessment used the *Neuropsychological Assessment Battery – NEUPSILIN*, validated for individuals aged 12 to 90 years. This instrument comprises 32 subtests covering nine cognitive domains: temporal-spatial orientation, attention, perception, memory, arithmetic skills, oral and written language, praxis, and executive functions. Its goal is to provide a brief neuropsychological profile, both quantitative and qualitative, identifying preserved or impaired neuropsychological abilities. For this study, only the attention and memory subtests were analyzed, given their influence on the LLAEP-verbal response. Normality criteria were based on standards for the studied population's mean age and education level[14].

The behavioral assessment of central auditory processing was conducted in an acoustically treated booth using supra-aural headphones (Telephonics TDH39) and a two-channel audiometer (Interacoustics AD629B) connected to a notebook. The

following tests were selected: Frequency Pattern Test (FPT-Auditec), Masking Level Difference (MLD)[15], Dichotic Digits Test (DDT), Speech in Noise Test (SN) (ipsilateral with competitive noise at +5 dB SNR[16]) and Gap-in-Noise (GIN) test (monaural, track 1, both ears)[17]. This battery complies with the minimum testing recommendations of the Brazilian Academy of Audiology[18]. All participants had to present normal results on these tests due to the influence of central auditory processing on LLAEP.

Electroacoustic (TOAEs) and electrophysiological (BAEP-click) procedures were performed using the Intelligent Hearing Systems (IHS) equipment. Participants were seated comfortably and instructed accordingly for each test. TOAEs were elicited using nonlinear click stimuli, with responses analyzed in a 20 ms window at 80 dB SPL. Up to 15% artifacts were allowed. Cochlear function was considered normal with responses present at 3 of 5 frequencies (1, 1.5, 2, 3, and 4 kHz) with a signal-to-noise ratio greater than 3 dB[19].

Prior to electrophysiological testing, the participants' skin was prepared with abrasive paste where electrodes were placed. Disposable electrodes were positioned, maintaining impedance values below 3 kΩ and inter-electrode impedance below 2 kΩ. ER-3A insert earphones were used.

BAEP was performed to assess auditory pathway integrity at the brainstem level. Electrodes were placed at Fpz, Fz, A1, and A2. The stimulus was a 100 ms rarefaction polarity click at 80 dBHL, with 2,048 stimuli presented at 27.7/s, a gain of 100 K, and a bandpass filter of 100–3,000 Hz, within a 12 ms recording window. Normative values were based on wave I, III, and V latencies, interpeak intervals (I–III, III–V, and I–V), interaural wave V differences, and V/I amplitude ratio, following Webster's standard[20], using two standard deviations. Participants remained relaxed with eyes closed during testing.

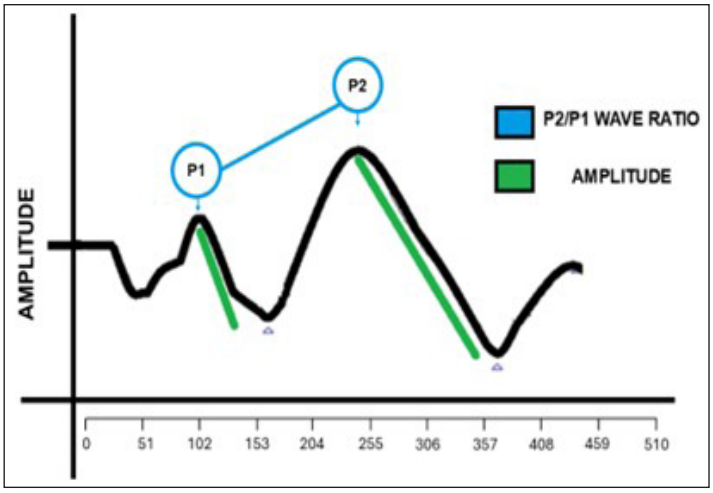
For group comparisons, the LLAEP-verbal was performed to assess CANS neural functioning and measure possible cortical gain by analyzing wave amplitudes (in microvolts) and the P2/P1 amplitude ratio.

Recording used the same equipment and impedance standards. Electrodes were positioned at Fpz, A1, A2, and Cz. A total of 150 verbal stimuli were presented at 80 dBHL using the oddball paradigm: the syllable /ba/ as the frequent stimulus (80%, 120 occurrences) and /di/ as the rare stimulus (20%, 30 occurrences). The stimulus rate was 1.1/s, with a 1–30 Hz filter, 100 K gain, and a 510 ms recording window [21].

Ears were tested binaurally but analyzed separately. Two waves were generated: one frequent and one rare. The P1 and P2 components were marked only in the frequent wave, as this reflects the cortical auditory processing (P1-N1-P2 complex), whereas the rare wave primarily elicits the P300, related to attention, discrimination, recognition, and memory processes[22]. However, for wave marking, replicability between the two traces was considered; that is, the morphology of both (frequent and rare) was compared to verify the presence or absence of the components.

For this study, amplitude marking was performed by measuring from the wave peak to the subsequent trough. Participants remained in an alert state, paying attention to the "rare" stimuli and mentally counting the number of rare stimuli perceived[21].

Below, Figure 1 illustrates the marking of the components used for the analysis.




Legend: P2/P1 = wave amplitude ratio.
Source: Adapted from Bruno et al.[21]

**Figure 1.** Graphic representation of amplitude marking and wave ratio analysis

The reference values adopted for component marking were those proposed by Bruno et al.[21], using two standard deviations. Wave marking was independently performed by two judges with expertise in auditory electrophysiology, and results were considered valid only when there was agreement between the markings.

Data were analyzed using the *Statistical Package for the Social Sciences* (SPSS), version 21.0 for Windows. After data collection, the data were entered into an Excel spreadsheet for statistical analysis. The normality of the variables was assessed using the Shapiro-Wilk test. Subsequently, the Mann-Whitney U test was applied to assess data homogeneity and to compare the amplitudes of the P1 and P2 waves, as well as the P2/P1 ratio, for the analysis and comparison of ears with and without tinnitus. A significance level of 5% ($p < 0.05$) was adopted for group comparisons and statistical differences.

## Results

Table 1 shows the amplitude values for LLAEP components by ear. A statistically significant difference was found only for the P2 component in the left ear, with a mean of 4.42 in the control group and 6.39 in the study group ($p = 0.017$).

**Table 1.** Amplitude analysis of the P1 and P2 components of the LLAEP-verbal, by ear

| COMPONENT | GROUP | N | MEAN | SD | P-VALUE |
|---|---|---|---|---|---|
| RE P1 | CG | 8 | 4.13 | 1.82 | 0.741 |
| | SG | 11 | 4.15 | 2.31 | |
| RE P2 | CG | 8 | 4.62 | 1.78 | 0.364 |
| | SG | 11 | 5.93 | 3.38 | |
| LE P1 | CG | 8 | 3.83 | 1.93 | 0.709 |
| | SG | 12 | 4.36 | 1.48 | |
| LE P2 | CG | 8 | 4.42 | 1.47 | 0.017* |
| | SG | 12 | 6.39 | 2.90 | |

Legend: RE = right ear; LE = left ear; CG = control group; SG = study group; N = number of ears; SD = standard deviation; * = statistically significant difference.
Source: Authors of the study.

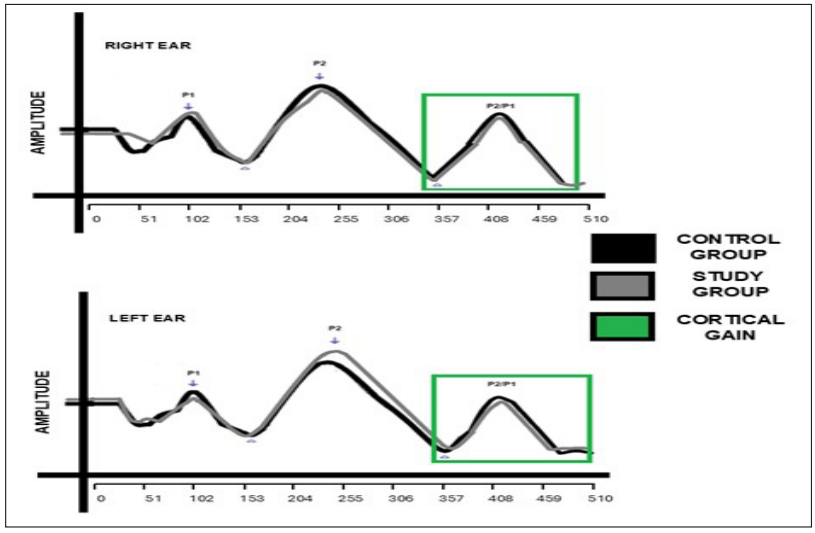
Table 2 presents the P2/P1 amplitude ratio values, showing no statistically significant differences for the right ear (p = 0.804) or left ear (p = 0.119).

**Table 2.** Analysis of the P2/P1 amplitude ratio of the LLAEP-verbal, by ear

| COMPONENT | GROUP | N | MEAN | SD | P-VALUE |
|---|---|---|---|---|---|
| RE P2/P1 | CG | 8 | 2.09 | 2.00 | 0.804 |
| | SG | 11 | 1.78 | 1.32 | |
| LE P2/P1 | CG | 8 | 1.56 | 1.13 | 0.119 |
| | SG | 12 | 1.17 | 1.35 | |

Legend: RE = right ear; LE = left ear; CG = control group; SG = study group; N = number of ears; P2/P1 = amplitude ratio between P2 and P1 waves; SD = standard deviation.
Source: Authors of the study.

In Figure 2, it is possible to observe the graphical representation of the mean amplitude and the wave ratio of the LLAEP components when comparing the CG and the SG, showing higher P2 component amplitudes in individuals with tinnitus perception in the left ear.




Legend: CG = control group; SG = study group; RE = right ear; LE = left ear; P2/P1 = wave amplitude ratio; SD = standard deviation.
Source: Authors of the study.

**Figure 2.** Graphical representation of the grand mean amplitude of the P1 and P2 components of the LLAEP and the P2/P1 wave ratio

## Discussion

The present study is consistent with tinnitus clinical practice and the specialized literature, considering that various perspectives and parameters related to the assessment of the LLAEP are currently reported, mainly focusing on measuring latency and the presence or absence of the components of this potential[23]. Thus, measuring wave amplitude and cortical gain in cortical/endogenous regions becomes extremely important for understanding neuroplastic mechanisms, since such analysis is still underexplored.

Additionally, recent studies aiming to measure neuroplastic changes in the central auditory pathway, especially in cortical auditory regions, often involve non-homogeneous populations or other associated variables, whether related to longevity,

pathologies, or sound perception disorders[24],[25]. Therefore, the present study aimed to apply rigorous methodology to exclude other variables (cognition, attention, central auditory processing, and auditory acuity) that could interfere with the electrophysiological findings, thereby allowing a specific observation of the influence of the tinnitus symptom in the cortical region, which justifies the smaller sample size.

Correlating this study's findings with the literature, a systematic review aimed to measure alterations in latency and amplitude of cortical and cognitive potential waves in individuals with tinnitus, concluding that event-related potentials can help determine the neurotransmitter involved in tinnitus generation[20]. The same review observed that the speed of cognitive processes in tinnitus patients is not affected. Therefore, the hypothesis of the present study is justified in focusing on measuring amplitude and cortical gain, rather than the speed/latency of the waves[26].

A significant increase was observed only for the P2 component in the left ear. This finding corroborates the results of a study conducted on individuals with chronic tinnitus and hearing thresholds within normal limits[2], which emphasized that there are impairments that can manifest in functional aspects related to the processing of acoustic information and in cognitive aspects, negatively impacting individuals' quality of life[3]. Thus, the greater responsiveness of the P2 component in the left ear may be associated with cerebral perceptual asymmetry and disorganization of neural functioning in the primary/secondary auditory cortex and the reticular formation, acting as a compensatory mechanism for auditory discrimination and attention performance[3],[26].

Two other studies used the LLAEP in the cortical analysis of individuals with tinnitus and found a reduction in the amplitude of the P2 wave[25],[27]. This reduction in neural responsiveness of the component, without changes in neural firing speed, may be justified by a decrease in the number of neurons responding, a reduction in neural activity, and/or greater desynchronization in the firing of the involved neurons. Therefore, this fact may not be consistent with the present study because the participants only presented the perception of the symptom, with other variables that could interfere with the potential findings being controlled.

Conversely, Morse et al.[28] observed an increase in P2 responsiveness, without changes in the other components. This amplification of neural responsiveness related to the primary/secondary auditory cortex and the reticular formation, which are responsible for auditory discrimination[22], was also evidenced in the present study. This may be justified by the hypothesis of reduced central inhibition and/or increased excitation in this region.

Analyzing the other parameters of the LLAEP, such as the P1 component in both ears, P2 in the right ear, and the P2/P1 ratio (cortical/endogenous gain), it was found that there were no differences between the groups (Tables 1 and 2). This demonstrates the possibility that tinnitus perception alone does not modify the neural responsiveness of the entire cortical region but, when associated with other pathologies, leads to greater alterations due to the diffuse changes caused by the cumulative effect of events in the brain region[29].

The amplitude of P2 in individuals with tinnitus complaints in the left ear shows that individuals with a worse perception of the symptom in one ear have greater neural activation in the ipsilateral primary/secondary auditory cortex. This finding does not align with most studies conducted in the studied population[25], but it is a promising finding, as it demonstrates that the severity of symptom perception is also an influential factor in the disorganization of the central auditory pathway.

As previously mentioned, the central gain theory combined with the chaos theory appears to be the most accepted explanation for tinnitus perception. Therefore, to objectively measure the increase in neural responsiveness, BAEP is suggested; however, considering the functional impairments, the LLAEP-verbal can also assist in measuring these alterations, as well as assessing the impacts on quality of life and understanding neurobiological capacity.

Although no statistically significant difference was observed in the other parameters in the present study, it is noteworthy that the large variability in amplitude values and the sample size may have contributed to these results. It is believed that the change in amplitude may reflect the brain's adaptive response to tinnitus and point to central auditory involvement in the perception of the symptom.

A limitation of the present study is the small sample size. Therefore, further studies using this methodology with larger samples are needed so that

the results are representative of the population of interest, avoiding bias and providing a solid foundation for valid conclusions. These findings should be considered with caution when generalizing the data. Measuring other parameters may yield interesting findings for understanding the neurobiological mechanisms related to tinnitus perception, such as measuring the N1 and N2 troughs. In the present study, only cortical/endogenous components were analyzed due to the need to initially exclude the influence of cognitive aspects related to attention and memory (N2-P3). Thus, further research is still needed, given the relevance of this content for a comprehensive analysis of the auditory pathway in individuals with tinnitus and for understanding the neuroelectrical functioning in higher regions of the auditory pathway, with the LLAEP being an important clinical tool and promising for tinnitus treatment. In this regard, the exam may be a useful tool for the population studied.

## Conclusion

Individuals with tinnitus symptoms and normal audiometry showed increased values and amplitude of the P2 component in the LLAEP with verbal stimuli, specifically in the left ear; however, no significant differences were observed in the P1/P3 wave ratio between the groups.

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