

Original articles

Study of the neural plasticity in adults and older adults new hearing aid users

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ABSTRACT

Purpose: to monitor, with long-latency auditory evoked potentials, the plasticity of the central auditory pathways in adults and older adults, new users of hearing aids.

Methods: a total of 15 adults and older adults, aged 55 to 85 years, participated in the research. They had a symmetric bilateral mild to moderate sensorineural hearing loss, without previous experience with any type of hearing aid. The long-latency auditory evoked potentials were conducted with and without amplification, at 60 and 75 dBnHL, with speech stimulus in a sound field, in two assessment moments: up to one week after fitting the hearing aid and after six months of its use. The Student's t-test was used for statistical analysis, considering significant the p-value < 0.05.

Results: responses with lower latency values were observed for the right ear in the second assessment. Comparing the first with the second assessment, both with and without the hearing aid, an increase in the amplitude of P2-N2 was observed, as well as an increase in the latency of the P2 component at the intensity of 75 dBnHL. No statistically significant differences were observed at the intensity of 60 dBnHL.

Conclusion: the use of the hearing aid promoted the plasticity of the central auditory pathways, increasing the number of neurons responsive to the sound stimuli.

Keywords: Hearing Aids; Neuronal Plasticity; Evoked Potentials, Auditory; Hearing Loss; Sensorial Deprivation

INTRODUCTION

Neuroplasticity is the central nervous system's ability to be modified through a structural and/or functional reorganization. It is dependent on intrinsic and environmental mechanisms and takes place after the intervention, resulting from either exercising an ability or being frequently exposed to a stimulus1.

Stimulating the auditory pathways with the current technological resources - such as the cochlear implant and hearing aid (HA) – brings about noticeable changes in the neuronal connections, evidencing the existence of neural plasticity¹. The effects of plasticity resulting from the use of HA can be assessed with auditory electrophysiological tests, also known as auditory evoked potentials².

The auditory evoked potentials are objective procedures used in the field of clinical audiology to assess the neuroelectric activity of the auditory pathway, from the auditory nerve to the cerebral cortex, in response to an acoustic stimulus. Such activity can be picked up with surface electrodes attached to the earlobes, face, or scalp3.

The P1, N1, P2, and N2 components of the longlatency auditory evoked potentials (LLAEP) are being investigated in the specialized literature. It has proved to be quite promising in the assessment of HA users to verify modifications in the central auditory nervous system (CANS) following auditory stimulation with sound amplification^{4,5}. These components' possible generators encompass areas of the primary (superior temporal lobe) and secondary auditory cortex, and the limbic system, and are interfered by the neural plasticity process⁶.

Besides these, the results of the P3 (or P300) component of the LLAEP are also being researched in HA users, proving to be an effective method to verify how these people are objectively processing the acoustic signal. The possible P300 generators encompass the hippocampus, frontal lobe, and parietal lobe6.

There is a vast amount of published longitudinal studies in the scientific literature describing the effects of neural plasticity with LLAEP analysis in patients who use electronic devices. Nevertheless, most of the articles report the experiences of either children or cochlear implant users. Also, there are in the literature studies assessing older adults, but not longitudinally, and approaching the assessment of experienced HA users.

Hence, there is a need for scientific evidence of the benefit of amplifying neural plasticity in adult and older adult HA users following a certain period of stimulation throughout the rehabilitation process.

The present study's differential is the longitudinal assessment (before and after six months of experience) of adult and older adult new HA users to measure the effects of neural plasticity in their auditory pathway. The hypothesis is that adults and older adults with sensorineural hearing loss improve in the LLAEP due to their use of the HA.

This study aimed to monitor, with the LLAEP, the plasticity of the central auditory pathways in adults and older adults, new HA users.

METHODS

This is a prospective longitudinal study conducted with adults and older adults with a symmetric, bilateral, mild to moderate sensorineural hearing loss.

This research was approved by the research ethics committee of the Faculdade de Medicina da Universidade de São Paulo - FMUSP, São Paulo, Brazil, under number 228/15, having complied with the ethical precepts for human research. The collection began after the project had been approved by the ethics committee. All the participants were informed of its objectives and procedures and signed the informed consent form.

Altogether, 15 adult and older adult individuals, aged 55 to 85 years (72 ± 7.16 years), seven males and eight females, participated in the study. All of them were beginning to use the HA.

The inclusion criteria were defined as:

- Having normal otoscopy;
- · Presenting mild to moderate symmetric bilateral sensorineural hearing loss, according to Lloyd and Kaplan's classification⁷;
- Having used the HA for up to a week before the first assessment;
- Not having a neurologic or psychiatric impairment that might hinder their understanding of the procedures to be followed. Using medications or having chronic diseases were not considered at this moment.

Conventional audiological assessments (measuring acoustic immittance, pure-tone threshold the audiometry, and speech audiometry) were performed to discard changes in the middle ear and determine the hearing thresholds.

All the procedures described below were performed in two separate moments: up to one week after fitting the HA and after six months using the HA. The selection, fitting, and follow-up of HA use, as well as the fine adjustments, when necessary, were done by the speech-language-hearing team of the hearing health service attended by the adults and older adults for follow-up before the audiological assessments.

For the LLAEP, the skin was cleaned and the electrodes attached with electrolytic paste in predetermined positions (vertex - Cz, and right and left mastoids - M2 and M1), following the norm in the International Electrode System IES 10-208. The electrodes' impedance values, which had to be below 5 kOhms, were verified.

The equipment used to pick up the LLAEP was the model Smart EP USB Jr manufactured by Intelligent Hearing Systems (IHS 5020). The acoustic speech stimuli (syllables /ba/ and /da/) were presented at the intensities of 60 dBnHL and 75 dBnHL in an acoustically treated room, in a previously calibrated sound field, and with the loudspeaker positioned at 0º azimuth one meter away from the patient.

The stimulus was presented at the rate of 1.1 per second, totaling 300 stimuli - 15% corresponding to the rare stimulus (syllable /ba/) and 85%, to the frequent stimulus (syllable /da/). The analysis window lasted 512 ms, and the high-pass and low-pass filters were set at 1 and 30 Hz.

Each person was assessed in two conditions: one of them with both devices turned on, and the other without the HA. Thus, the ears were simultaneously stimulated, and the responses of both left and right auditory pathways were picked up.

The individuals were instructed to pay attention to the rare stimuli, which were randomly presented amid a series of frequent stimuli. They were asked to count the number of times the rare event occurred9. Considering the oddball paradigm, each hearing condition (with and without the HA, and at the intensities of 60 dBnHL and 75 dBnHL) was collected only once.

After the LLAEP pick-up, the P1, N1, P2, N2, and P3 components were analyzed. The P3 component (an emerging positive peak at approximately 300 ms) was analyzed in relation to its latency and amplitude (N2-P3) in the tracing corresponding to the rare stimulus. In the tracing corresponding to the frequent stimulus, the P1 and P2 components were identified (emerging positive peaks at approximately 50 ms and 150 ms), as well as the corresponding N1 and N2 (emerging negative peaks at approximately 100 ms and 200 ms), whose latency and amplitude were analyzed (P1-N1 and P2-N2) (Figure 1).

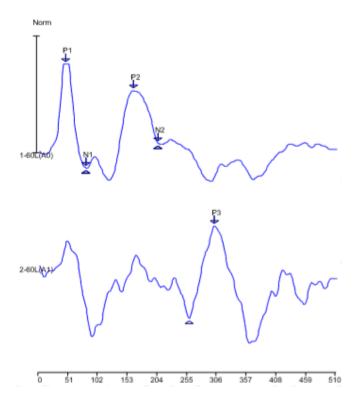


Figure 1. Example of the analysis of the tracing of long-latency auditory evoked potentials

To increase the accuracy of the result analysis, the electrophysiologic tracings were blindly analyzed by two judges (professionals experienced in LLAEP analysis). Whenever there were divergences, a third judge (who had not had access to the previous analyses) was consulted.

Each component's latency and amplitude results were analyzed with descriptive statistical measures concerning the mean and standard deviation values.

Afterward, the data were analyzed with inferential analysis to compare the results obtained in the right and left auditory pathway. In a second analysis stage, the results obtained in the two assessment moments (up to one week using the HA and after six months experiencing it) were compared.

Considering that the data obtained in the sample presented a normal distribution around the mean, the Student's paired t-test was used to analyze the comparisons. The statistically significant differences were considered based on p-values up to 0.05.

RESULTS

Table 1. Comparison between latencies (milliseconds) and amplitudes (microvolts) of the components of the long-latency auditory evoked potentials in the right and left auditory pathways, at the intensity of 60 dBnHL, without the hearing aid

| | | 1st Assessment | p-value | 2 nd Assessment | p-value |
|----------|---|--------------------|---------|----------------------------|---------|
| P1 | R | 86.0 (±14.9) | 0.410 | 84.7 (±16.8) | 0.591 |
| PI | L | 89.4 (±11.6) | | $83.7 (\pm 18.4)$ | |
| N1 | R | $132.7 (\pm 16.3)$ | 0.560 | $130.6 (\pm 26.8)$ | 0.819 |
| INI | L | $134.5 (\pm 16.5)$ | | $131.6 (\pm 29.2)$ | |
| P2 | R | $222.0 (\pm 26.4)$ | 0.740 | 231.4 (±51.0) | 0.341 |
| Γ2 | L | $220.0 (\pm 26.9)$ | | $225.4 (\pm 60.9)$ | |
| N2 | R | $277.7 (\pm 40.4)$ | 0.090 | $278.3 (\pm 57.8)$ | 0.673 |
| INZ | L | $268.0 (\pm 38.6)$ | | $282.5 (\pm 61.5)$ | |
| P3 | R | $363.3 (\pm 49.0)$ | 0.700 | 375.4 (±51.7) | 0.473 |
| FU | L | 367.1 (±55.0) | | $367.1 (\pm 43.5)$ | 0.473 |
| P1-N1 | R | $4.2 (\pm 2.4)$ | 0.210 | $3.3 (\pm 1.2)$ | 0.905 |
| F I-IN I | L | $4.6 (\pm 1.8)$ | | $3.2 (\pm 1.6)$ | |
| P2-N2 | R | $2.7 (\pm 3.2)$ | 0.517 | $2.4 (\pm 2.1)$ | 0.709 |
| 1 Z-1VZ | L | $2.4 (\pm 1.4)$ | | $2.3 (\pm 2.0)$ | |
| N2-P3 | R | $3.9 (\pm 2.9)$ | 0.454 | $4.6 (\pm 3.0)$ | 0.574 |
| 11/2-1 0 | L | 4.5 (±2.9) | | 5.1 (±3.2) | 0.017 |

Legend: R- right auditory pathway; L- left auditory pathway; Mean (± standard deviation) Student's t-test

Table 2. Comparison between latencies (milliseconds) and amplitudes (microvolts) of the components of the long-latency auditory evoked potentials in the right and left auditory pathways, at the intensity of 60 dBnHL, with the hearing aid

| | | 1 st Assessment | p-value | 2 nd Assessment | p-value |
|---------|---|----------------------------|---------|----------------------------|---------|
| P1 | R | $79.3 (\pm 13.9)$ | 0.260 | $82.3 (\pm 15.7)$ | 0.012* |
| FI | L | $86.7 (\pm 25.7)$ | | $96.2 (\pm 22.4)$ | |
| N1 | R | 121.4 (±21.5) | 0.258 | 122.9 (±19.8) | 0.038* |
| INI | L | $129.4 (\pm 19.4)$ | | 134.9 (±28.2) | |
| P2 | R | 202.1 (±50.9) | 0.372 | $208.2 (\pm 40.0)$ | 0.300 |
| Γ2 | L | $217.5 (\pm 68.4)$ | | 219.1 (±41.0) | |
| N2 | R | $243.1 (\pm 53.5)$ | 0.930 | $259.2 (\pm 46.1)$ | 0.822 |
| IVZ | L | $242.3 (\pm 52.9)$ | | $261.3 (\pm 48.1)$ | |
| P3 | R | $392.6 (\pm 46.3)$ | 0.509 | 353.7 (±57.3) | 0.129 |
| го | L | $385.5 (\pm 46.4)$ | | $375.7 (\pm 57.9)$ | 0.129 |
| P1-N1 | R | $3.2 (\pm 2.2)$ | 0.383 | $3.4 (\pm 2.0)$ | 0.900 |
| FI-IVI | L | $2.8 (\pm 1.6)$ | | $3.2 (\pm 2.0)$ | |
| P2-N2 | R | $2.0 (\pm 1.1)$ | 0.842 | $2.3 (\pm 1.8)$ | 1.000 |
| rz-INZ | L | $2.1 (\pm 1.4)$ | | $2.3 (\pm 1.4)$ | |
| N2-P3 | R | $4.5 (\pm 3.2)$ | 0.234 | $4.1 (\pm 3.4)$ | 0.700 |
| 11/2-53 | L | 5.1 (±2.9) | | 4.3 (±2.4) | 0.700 |

Legend: R- right auditory pathway; L- left auditory pathway; Mean (± standard deviation); *p-value with a statistically significant difference.

Table 3. Comparison between latencies (milliseconds) and amplitudes (microvolts) of the components of the long-latency auditory evoked potentials in the right and left auditory pathways, at the intensity of 75 dBnHL, without the hearing aid

| | | 1st Assessment | p-value | 2 nd Assessment | p-value |
|--------|---|--------------------|---------|----------------------------|---------|
| P1 | R | 80.3 (±16.0) | 0.664 | 78.9 (±22.4) | 0.251 |
| гі | L | 81.7 (±8.1) | | $83.3 (\pm 15.0)$ | |
| N1 | R | 129.8 (±15.2) | 0.944 | 122.3 (±21.1) | 0.148 |
| INI | L | $130.0 (\pm 13.8)$ | | 128.3 (±18.1) | |
| P2 | R | $202.9 (\pm 40.0)$ | 0.284 | $224.6 (\pm 34.4)$ | 0.698 |
| ۲۷ | L | $214.3 (\pm 25.5)$ | | 226.1 (±35.1) | |
| N2 | R | 249.7 (±41.5) | 0.729 | $284.5 (\pm 48.9)$ | 0.958 |
| IVZ | L | 252.7 (±26.7) | | $285.0 (\pm 37.7)$ | |
| P3 | R | 359.6 (±37.3) | 0.660 | 348.1 (±38.7) | 0.813 |
| FJ | L | $355.6 (\pm 30.0)$ | | $346.8 (\pm 35.1)$ | |
| P1-N1 | R | $4.0 (\pm 1.3)$ | 0.280 | $4.3 (\pm 1.9)$ | 0.383 |
| F1-IVI | L | $4.3 (\pm 1.2)$ | | $4.5 (\pm 1.5)$ | |
| P2-N2 | R | $2.3 (\pm 1.2)$ | 0.181 | $3.2 (\pm 2.2)$ | 0.946 |
| | L | $2.1 (\pm 1.2)$ | | $3.2 (\pm 2.4)$ | |
| N2-P3 | R | $4.6 (\pm 3.1)$ | 0.306 | $5.2 (\pm 3.3)$ | 0.509 |
| | L | $5.6 (\pm 3.8)$ | | $4.8 (\pm 3.2)$ | 0.509 |

Legend: R- right auditory pathway; L- left auditory pathway; Mean (± standard deviation) Student's t-test

Table 4. Comparison between latencies (milliseconds) and amplitudes (microvolts) of the components of the long-latency auditory evoked potentials in the right and left auditory pathways, at the intensity of 75 dBnHL, with the hearing aid

| | | 1st Assessment | p-value | 2 nd Assessment | p-value |
|---------|---|--------------------|---------|----------------------------|---------|
| P1 | R | 74.7 (±9.8) | 0.283 | 75.9 (±9.7) | 0.046* |
| гі | L | $77.6 (\pm 10.9)$ | | 81.2 (±8.1) | |
| N1 | R | 123.1 (±15.4) | 0.559 | $126.3 (\pm 10.7)$ | 0.731 |
| IVI | L | $126.3 (\pm 12.4)$ | | $125.3 (\pm 10.4)$ | |
| P2 | R | $202.6 (\pm 28.6)$ | 0.449 | $216.0 (\pm 34.0)$ | 0.354 |
| ΓZ | L | 242.9 (±31.3) | | $220.7 (\pm 34.8)$ | |
| N2 | R | $244.3 (\pm 38.2)$ | 0.872 | $266.7 (\pm 52.5)$ | 0.354 |
| IVZ | L | 242.9 (±31.3) | | 277.1 (±45.5) | |
| P3 | R | $334.1 (\pm 48.3)$ | 0.138 | $349.0 (\pm 34.8)$ | 0.932 |
| гo | L | $347.8 (\pm 48.4)$ | | $348.3 (\pm 38.7)$ | 0.932 |
| P1-N1 | R | $4.2 (\pm 1.8)$ | 0.151 | $4.3 (\pm 1.9)$ | 0.982 |
| F 1-1V1 | L | $4.9 (\pm 1.3)$ | | $4.3 (\pm 1.6)$ | |
| P2-N2 | R | $2.0 (\pm 1.6)$ | 0.769 | $2.5 (\pm 1.7)$ | 0.077 |
| F Z=INZ | L | $1.9 (\pm 0.9)$ | | $3.2 (\pm 1.9)$ | |
| N2-P3 | R | $4.8 (\pm 3.8)$ | 0.354 | $4.5 (\pm 2.2)$ | 0.932 |
| | L | 4.2 (±2.4) | | 5.6 (±3.6) | 0.302 |

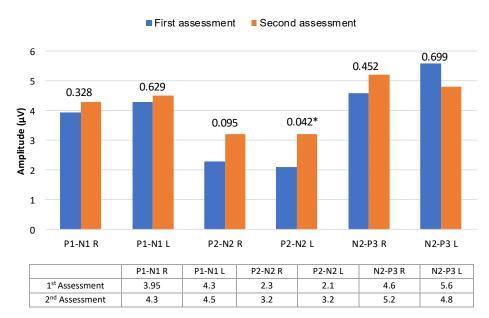
Legend: R- right auditory pathway; L- left auditory pathway; Mean (± standard deviation); *p-value with a statistically significant difference. Student's t-test

In the comparison between right and left auditory pathways, considering both intensities and both assessment moments, no significant differences were verified in any of the components assessed without the HA (Tables 1 and 3).

As for the assessments with the HA, there were statistically significant differences for the latencies of the P1 and N1 components in the second assessment at 60 dBnHL - with greater latencies in the left (Table 2). In the second assessment as well, at the intensity of 75 dBnHL, a statistically significant difference was

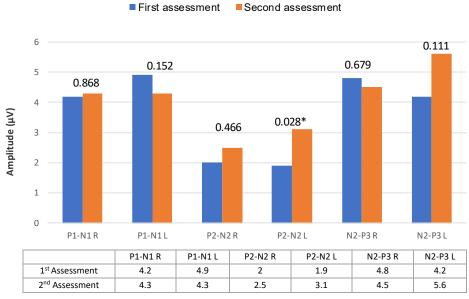
observed between the right and left auditory pathways for the latency of the P1 component using HA - the greatest values were also observed in the left (Table 4).

When each of the LLAEP components was analyzed for each intensity used, with and without HA, statistically significant differences were observed between the first and second assessment, in the left auditory pathway, for the P2-N2 amplitude (Figures 2 and 3). The amplitude was greater after six months using the HA.



Legend: R- right auditory pathway; L- left auditory pathway; *p-value with a statistically significant difference

Figure 2. Comparison of the amplitudes of the long-latency auditory evoked potentials between the 1st and 2nd assessments, without the hearing aid, at the intensity of 75 dBnHL

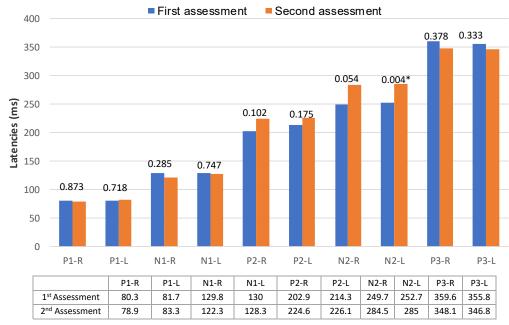


Legend: R- right auditory pathway; L- left auditory pathway; *p-value with a statistically significant difference

Figure 3. Comparison of the amplitudes of the long-latency auditory evoked potentials between the 1st and 2nd assessments, with the hearing aid, at the intensity of 75 dBnHL

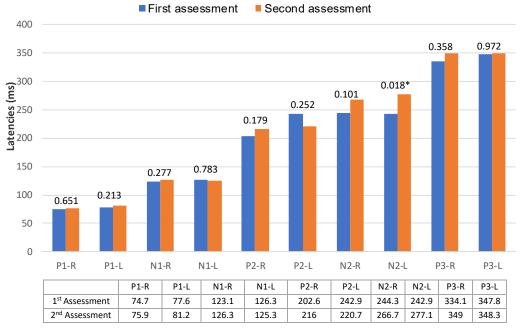
Differences were observed in the left auditory pathway latency results of the N2 component between both assessments, both without (Figure 4) and with HA (Figure 5) - the latency was greater in the second assessment.

Concerning the comparison between the first and second assessment at the intensity of 60 dBnHL, no statistically significant differences were observed neither with HA, nor without it (p-values > 0.05).



Legend: R- right auditory pathway; L- left auditory pathway; *p-value with a statistically significant difference

Figure 4. Comparison of the latencies of the long-latency auditory evoked potentials between the 1st and 2nd assessments, without the hearing aid, at the intensity of 75 dBnHL



Legend: R- right auditory pathway; L- left auditory pathway; *p-value with a statistically significant difference

Figure 5. Comparison of the latencies of the long-latency auditory evoked potentials between the 1st and 2nd assessments, with the hearing aid, at the intensity of 75 dBnHL

DISCUSSION

In this study, the LLAEP was used to verify modifications in the latencies and amplitudes associated with amplification in adults and older adults, new HA users, to monitor the auditory pathway plasticity.

In the first stage, the results obtained in the right and left auditory pathways were compared with and without HA. Greater latency time in the sound processing in the left auditory pathway was observed in the analysis of some components.

Considering that the neural pathways are crossed throughout the CANS and that the right hemisphere processes mainly nonverbal sounds, while the left processes mainly verbal sounds10-12, the better responses (lower latencies) observed in the right auditory pathway (left hemisphere) can be justified, as the acoustic stimulus used to elicit the neural responses in the present research was speech.

Regarding the longitudinal monitoring of the auditory pathway before and after six months using the HA, an increase in the P2-N2 amplitude was verified at the intensity of 75 dBnHL, both with and without the HA, in the left auditory pathway.

Few studies in the literature assessed the plasticity resulting from the use of hearing aids in adult and older adult new HA users, and they came to conflicting results. One of them assessed the impact that using HA for six months had on the P300 in individuals 16 to 60 years old with unilateral sensorineural hearing loss. They used the tone-burst stimulus presented at 70 to 100 dB, depending on the person's hearing threshold13. An increase in the amplitude of the P300 component was verified13, differing from the findings in this study; however, the methodology and sample differed considerably as well.

Another study, with a different methodology from the present one (using 500 and 3000 Hz tones, presented at 65, 75, and 85 dB), aimed to investigate changes in the central auditory processing in adult uni- and bilateral new HA users. After 12 weeks, no significant change was observed in either the N1 or P2 waves, although a significant improvement was noticed in speech recognition¹⁴.

Previous studies demonstrate that, with the use of the HA, there is a decrease in the latencies, an increase in the amplitudes, and an improvement in the morphology of the LLAEP waves when compared with the nonuse of the HA. Moreover, the increase in the amplitude of the LLAEP components reflects the plasticity following auditory treatment in adults and can reflect a possible increase in the neural synchrony. Hence, it is likely that the effects of amplification furnish similar changes in the neural synchrony for older hearers and that these effects are measurable using the amplitudes of cortical auditory evoked potentials¹⁵⁻¹⁷, as observed in the findings of the present study.

An increase was observed in the latency of the N2 component after six months using the HA. It is difficult to explain this finding since what is expected is a decrease in the latency due to the plasticity. Nonetheless, previous studies have been showing

situations similar to what was observed in the present study. The authors have related these findings to the influences determined by the algorithms of the HAs18-20. For instance, when the LLAEP was compared between using and not using HA, a delay in the latencies of the N1 and P2 waves was observed in the amplified ears¹⁹. Another study comparing analog and digital HAs with LLAEP verified latencies in the N1 and P2 waves, without significant differences between using and not using HA, as well as a significant delay in the latencies of N1 and P2 for both digital HAs18.

As for the intensity of the acoustic stimulus, greater latency and lower amplitude values were observed at the lowest intensity assessed (60 dBnHL). These findings were already expected, especially for the first LLAEP components (P1, N1, P2, and N2), which are considered exogenous due to their being interfered by the characteristics of the acoustic stimulus - i.e., the lower the intensity, the fewer neurons respond to the stimulation, which decreases the amplitude and increases the latency of the response³.

In this study, 75 dBnHL was the best intensity employed for the acoustic stimulus, as at this intensity the responses were registered with the lowest latencies and greatest amplitudes, making it easier to visualize the LLAEP components.

Hence, it is suggested that the increase in amplitude observed in the present study is related to the morphological and functional changes resulting from the increase in the number of responsive neurons, as well as the increase in the dendritic ramification and synaptic connections and synchronizations^{16,21}, which took place in the adults and older adults assessed after six months using the HA.

The human brain's capacity is known to change much more effectively with the auditory experience in younger people. However, such plasticity is also present throughout life¹⁷ and the findings of the present study reinforce the idea that even in a degenerating aging pathway, the effects of neural plasticity are present. Therefore, it is essential to perform interventions in them.

CONCLUSION

The results revealed that using a HA for six months could intensify the CANS plasticity in the sample studied, increasing the number of neurons responsive to sound stimuli. This reinforces the need for adults and older adults presented with mild to moderate sensorineural hearing loss to use hearing aids.

REFERENCES

- 1. Cardon G, Campbell J, Sharma A. Plasticity in the developing auditory cortex: evidence from children with sensorineural hearing loss and auditory neuropathy spectrum disorder. J Am Acad Audiol. 2012;23(6):396-495.
- 2. Billings CJ. Uses and limitations of electrophysiology with hearing aid. Semin Hear. 2013;34(4):257-69.
- 3. Hall JW III. eHandbook of auditory evoked response: principles, procedures & protocols. Kindle Direct Publishing; 2015.
- 4. Souza PE, Tremblay KL. New perspectives on assessing amplification effects. Trends in Amplification. 2006:10(3):119-41.
- 5. Tremblay KL, Kalstein L, Billings CJ, Souza PE. The neural representation of consonant-vowel transitions in adults who wear hearing aids. Trends in Amplification. 2006;10(3):155-62.
- 6. Mc Pherson DL. Late potentials of the auditory system. San Diego: Singular Publishing Group, 1996.
- 7. Lloyd LL, Kaplan H. Audiometric interpretation: a manual of basic audiometry. Baltimore: University Park Press: 1978.
- 8. Jasper HH. The ten-twenty electrode system of the International Federation. Electroenceph. Clin. Neurophysiol. 1958;10:371-5.
- 9. Musiek FE, Lee WW. Potenciais auditivos de média e longa latência. In: Musiek FE, Rintelmann WF (orgs). Perspectivas atuais em avaliação auditiva. 1ª ed. Barueri- SP: Manole; 2001. p. 239-67.
- 10. Kimura D. Cerebral dominance and the perception of verbal stimuli. Can J Psychol. 1961;15(3):167-71.
- 11. Zatorre RJ. Neural specializations for tonal processing. Ann NY Acad Sci. 2001;930:193-210.
- 12. Oppitz SJ, Bruno RS, Didoné DD, Garcia MV. Temporal resolution and cortical potential in different levels of English proficiency. Rev. CEFAC. 2017;19(1):27-40.
- 13. Fonseca CBF. Estudo dos potenciais evocados auditivos de Longa Latência (P300), em indivíduos com perda auditiva neurossensorial unilateral, antes e após a adaptação de prótese auditiva [Dissertação]. São Paulo (SP): UNIFESP - Escola Paulista de Medicina; 2006.
- 14. Dawes P, Munro KJ, Kalluri S, Edwards B. Auditory acclimatization and hearing aids: late auditory evoked potentials and speech recognition following unilateral and bilateral amplification. J Acoust Soc Am. 2014;135(6):3560-9.

- 15. Krishnamurti S, Wise L. Effects of amplification on cortical electrophysiological function. Hear Rev. 2018;26(7):26-32.
- 16. Jang JH, Jang HK, Kim SE, Oh SH, Chang SO, Lee JH. Analysis of P1 latency in normal hearing and profound sensorineural hearing loss. Clin Exp Otorhinolaryngol. 2010;3(4):194-8.
- 17. Ponton CW, Vasama JP, Tremblay K, Khosla D, Kwong B, Don M. Plasticity in the adult human central auditory system: evidence from late-onset profound unilateral deafness. Hear Res. 2001;154(1-2):32-44.
- 18. Marynewich S, Jenstad L, Stapells D. Slow cortical potentials and amplification - Part I: N1-P2 Measures. Int J Otolaryngol. 2012;2012:921513.
- 19. Chun I, Billings CJ, Miller CW, Tremblay KL. Aided electrophysiology using direct audio input: effects of amplification and absolute signal level. Am J Audiol. 2016;25(1):14-24.
- 20. Billings CJ, Tremblay KL, Miller CW. Aided cortical auditory evoked potentials in response to changes in hearing aid gain. Intl J Audiol. 2011;50(7):459-67.
- 21. Fallon JB, Irvine DRP, Shepherd RK. Neural prosthesis and brain plasticity. J Neural Eng. 2009;6(6):065008.