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Original articles

Differences and similarities in the long-latency auditory evoked potential recording of P1-N1 for different sound stimuli

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ABSTRACT

Purpose: this study aimed at illustrating the similarities and differences in the recording of components P1 and N1 for verbal and non-verbal stimuli, in an adult sample population, for reference purposes.

Methods: twenty-one adult, eutrophic individuals of both sexes were recruited for this study. The long-latency auditory evoked potential was detected by bilateral stimulation in both ears, using simultaneous recording, with non-verbal stimuli and the syllable /da/.

Results: for non-verbal and speech stimuli, N1 was identified in 100.0% of the participants, whereas P1 was observed in 85.7% and 95.2% individuals for non-verbal and speech stimuli, respectively. Significant differences were observed for the P1 and N1 amplitudes between the ears ($p < 0.05$); the P1 component, in the left ear, was higher than that in the right ear, whereas the N1 component was higher in the right one. Regarding the stimuli, the amplitude and latency values of N1 were higher for speech, whereas in P1, different results were obtained only in latency.

Conclusion: the N1 component was the most frequently detected one. Differences in latency and amplitude for each stimuli occurred only for N1, which can be justified by its role in the process of speech discrimination.

Keywords: Auditory Tests; Auditory Pathways; Auditory Perception; Adult

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INTRODUCTION

Auditory evoked potentials (AEPs) are characterized by the recording of resultant bioelectric activities after acoustic stimulation. The long-latency auditory evoked potential (LLAEP) is commonly studied for measuring neurophysiological changes during the maturational process, usually through components P1 and N1[1,2], which are individualized because they theoretically represent the first activity in the auditory cortex resulting from sound stimulation[3].

During development, modifications are observed in the latency and amplitude of these components, concurrent with increased myelination and synaptic efficiency[4]. These changes may reflect the refinement of the neural processes required for the acquisition and development of auditory processing skills[4,5]. Both P1 and N1 reach values similar to those of an adult in the second decade of life, at 17 and 16 years for P1 and N1, respectively[4].

The distinctions in the maturational course of these components reflect different neural generators for each of them. The P1 component, which is caused by the activity of the thalamic-cortical circuit[6], is obligatory in childhood[7,8] and present in all age groups from 5 to 78 years[9]. On the other hand, N1 occurs as a reliable waveform around the age of 6 to 7 years and becomes mandatory in adulthood[9] as a result of activities of the supratemporal auditory cortex, responsible for the initial decoding of the stimulus[10].

The recording of P1 and N1 occurs in accordance with the spectral characteristics of the stimulus employed[11], mainly duration and frequency. There are several possible modes of acoustic stimuli application including verbal stimuli, with simple and complex structures, and non-verbal stimuli[12,13] such as clicks, tone bursts, vowels, and syllables.

There are a variety of findings regarding the use of different stimuli in LLAEP, as well as different populations and goals. Swink and Stuart[14] compared the use of the vowel /a/, in its natural and synthetic form, to a non-verbal stimulus. The authors verified latency prolongation for verbal stimulus versus a pure tone of 0.723 kHz, and synthetic versus natural speech. Contrary to this finding, the latency and amplitude variables were not different when they were elicited in an oddball paradigm between the non - verbal stimuli (tone burst in frequencies of 1 kHz - frequent and 4 kHz - rare), and verbal stimuli (syllables /ba/ - frequent and /ga/, /da/ e /di/ - rare)[15]. Another study, when comparing a population of healthy individuals with mild cognitive impairment, found a prolonged latency for a tone burst of 1 kHz in relation to the speech stimulus /ba/ for the group with cognitive impairment[16].

The processing of verbal stimuli is a task of greater complexity in discrimination compared to the processing of non-verbal ones[15], allowing the acquisition of complementary information regarding the biological processes that are necessary for proper processing of speech[17].

The use of AEP is recommended in order to complement the diagnostic evaluation of auditory processing disorder[18], because it is regarded as a biological marker of the functional integrity of the neural pathways. However, there is still no evidence that one of these presents satisfactory sensitivity and specificity for the identification of this condition[19]. Understanding that the P1 component reflects the first record of sound signal processing in the primary auditory area[3] and N1 is involved in the decoding function[10]; it is necessary to know the parameters of differences and similarities of these components for different sound stimuli, to aid in the investigation of the neural bases responsible for speech processing at the cortical level[20].

Considering the assumptions presented, the comparison of the differences and similarities of components P1 and N1, for two distinct stimuli, in a young healthy adult population with complete auditory maturation, provides data for the evaluation of the functioning of the cortical input pathway, in response to sound stimuli, and allows the use of this information as a parameter in studying other populations and age groups, including people with auditory processing disorder, using the same protocol.

Thus, the present study aimed to characterize the registration of components P1 and N1 for verbal and nonverbal stimuli in an adult population, for reference purposes.

METHODS

This was a prospective, transversal, and observational study approved by the Research Ethics Committee of the Hospital das Clínicas of the Medical School of Ribeirão Preto – University of Sao Paulo (number 10482/2015). All subjects signed the terms of free and informed consent.

Casuistry

Twenty-one young healthy adults participated in the study: 8 (38.1%) males and 13 (61.9%) females.

Inclusion criteria were considered ages ranging from 18 to 30 years, complete secondary education as minimum level of education, absence of personal and/or family background of hearing loss of any nature, and current symptoms or preceding the evaluation period, suggestive Central Auditory Nervous System (CANS) disorders such as epilepsy, seizures, and migraine. Exclusion criteria were defined as the presence of altered results in tonal audiometry and/or in at least one of two auditory processing tests.

Procedures

Initially, the external auditory meatus was inspected with an otoscope, model MISSOURI 001, to confirm the absence of conditions that could influence the determination of tonal thresholds by air. The following procedures were performed: tonal audiometry; speech reception threshold measurement; acoustic immittance measurement; behavioral tests of auditory processing, including the pattern duration test and dichotic digit test; and AEP measurement, including short (SLAEP) and long latency AEP (LLAEP) measurement.

Auditory sensitivity was determined in a cabin acoustically treated with the Otometrics brand audiometer model MEDSEN Astera2, HDA 300 handset. The tonal thresholds by air conduction were searched in the frequencies 0.25 kHz to 8 kHz, in a descending-ascending technique. Those with ≤ 20 dB NA were considered normal. To confirm the veracity of the thresholds, the speech recognition threshold (SRT) was used with trisyllabic words; results equal or up to 10 dB NS of the tritonal mean (0.5, 1, and 2 kHz) were interpreted as adequate.

Acoustic immittance measurements were obtained using the Otometrics model, ZODIAC 901, with a 226 Hz probe. The presence of a tympanometric curve of types “A,” “As,” “Ad,” or “C” was considered an adequate result only if an acoustic reflex was present in the contralateral modality, in 0.5 to 2 kHz.

To exclude the possibility of Auditory Processing Disorder, two behavioral tests were applied. The Dichotic Digits Test (DDT), a Brazilian Portuguese version, was performed in the binaural integration stage, according to the application guidelines and the manual analysis[21]. Scores ≥ 95% in both ears were considered normal. The Duration Pattern Test (DPT)[22] was performed monaurally and applied in its naming stage - short or long - for each of the 30 tone sequences applied to each of the ears. The normal values adopted were scores ≥ 74.2% for the right ear and ≥ 72.7% for the left one[23].

Auditory evoked potentials were assessed with Intelligent Hearing Systems brand, SmartEP module, two-channel, with the ER3A model insertion handset. After cleansing the skin for the removal of epithelial scales and oil residues, the surface electrodes were set according to the international standard 10-20, arranged as follows: negative in A1 (left earlobe), A2 (right earlobe), positive in Cz (vertex) and the electrode ground in Fpz. The impedance level was maintained between 1-3 Kohms.

The SLAEP was performed with a click stimulus in a monoaural condition, at 80-dB NA intensity, with 1024 averages at a rate of 21.1 stimuli per second, with rarefied polarity. Band filter of 100-1500 Hz, gain of 100 μV, and analysis window of 12 ms were used. At least two consecutive stimulations were performed in order to verify the reproducibility of the components.

As a criterion for the analysis of components I, III, and V, their identification was considered in at least two tracings of the same ear, and the mean values of each component, absolute latencies, as well as their respective interpeak latencies, and the difference of the V wave were evaluated as well.

For the realization of LLAEP, two stimuli, click and the synthetic syllable /da/, were used in a binaural presentation with an intensity of 70 dB NA, 300 averages at a rate of 1.1 stimuli per second, and an inter-stimulus interval (ISI) of 810 ms, with alternating polarity. A bandpass filter of 1-30 Hz, 50 μV gain, and analysis window from -25 to 256 ms were applied as well. Two consecutive stimulations were performed for each stimuli in order to verify the reproducibility of the components.

Two criteria were adopted to identify the P1-N1 complex. The first one was the occurrence of a positive deflection (P1), around 40 to 50 ms, followed by a counter deflection (N1) of around 100 ms[16-24]. As a second criterion, P1 was considered present only on the condition of its amplitude having positive values, with the baseline as a reference; the same criterion was applied to N1 but with negative values. Thus, the visual presence of positive deflection, but with values lower than 0.1 μV, qualified P1 as absent.

Statistical analysis

Statistical analysis was performed using the non-parametric Wilcoxon test for paired samples, considering the comparison between the variables studied, ear and/

or stimulus, for each of the components. The level of significance was set at 5%.

RESULTS

The 21 individuals evaluated presented results within the values established as adequate for the psychoacoustic and electroacoustic auditory tests, enabling the registration of LLAEP in all of them. Descriptive analyses related to age, behavioral tests, and SLAEP are given in Table 1.

Initially, the occurrence of the components studied was verified. When the click stimulus was used, the presence of P1 was identified in 85.7% (n = 18) of the individuals and in 100% (n = 21) of the individuals for N1. For the speech stimulus, the presence of the P1 component was identified 95.2% (n = 20) of the individuals and N1 was present in 100% (n = 21) of them.

Table 1. Distribution of independent variables (n = 21)

Variables			Average	Min. – Max.
Age (years)			22	18 – 29
DDT (%)	RE		99.8	97.5 – 100.0
	LE		99.4	97.5 – 100.0
PDT (%)	RE		94.8	80.0 – 100.0
	LE		96.4	73.3 – 100.0
BAEP* (ms)	RE	I	1.85	1.66 – 2.00
		III	3.88	3.49 – 4.21
		V	5.95	5.55 – 6.20
		I-III	2.01	1.50 – 2.38
		III-V	2.05	1.56 – 2.31
		I-V	4.10	3.63 – 4.30
	LE	I	1.83	1.64 – 1.94
		III	3.88	3.58 – 4.21
		V	5.91	5.44 – 6.20
		I-III	2.04	1.71 – 2.41
		III-V	2.01	1.56 – 2.23
		I-V	4.08	3.51 – 4.35
	I.D.V		0.10	0.01 – 0.23

Caption: Min. = Minimum; Max. = Maximum; DDT = Dichotic Digits Test; PDT = Pattern Duration Test; RE = Right Ear; LE = Left Ear; BAEP = Brainstem Auditory Evoked Potential; IDV = Interaural Difference of Wave V.
*Reference values considering 30 adult eutrophic individuals, the average +/- 2 standard deviations: I –1.76 +/- 0.13; III– 3.84 +/- 0.22; V – 5.84 +/- 0.21; I –III= 2.34 +/- 0.23; I – V = 2.18 +/- 0.21; I – V = 4.08 +/- 0.24.

Two separate analyses were conducted for LLAEP. In the first analysis, the neural synchrony between the two ears for each stimuli used was studied, and in the second, the stimuli were compared with each other for each ear.

In the first analysis, it was verified that for the binaural presentation, there were significant differences in the amplitudes of P1 and N1 but not in their latencies. This result was observed for the click stimulus (Table 2) and the speech stimulus (Table 3). On presentation of both stimuli, P1 presented higher values to the left than to the right, whereas for N1, the inverse occurred, the right register was larger than the left ($p < 0.05$).

Table 2. Latency and amplitude values for click stimuli in the right and left ears

Stimulus	Comp.	Variable	Ear	Average (SD)	Median	Minimum	Maximum	p#
Click	P1	Latency (ms)	OD	42.8 (11.6)	36.2	30.5	66.0	0.148
			OE	41.5 (11.2)	36.5	31.0	66.5	
		Amplitude (μV)	OD	0.82 (0.50)	0.76	0.13	1.58	0.000*
			OE	1.23 (0.60)	1.14	0.18	2.46	
	N1	Latency (ms)	OD	89.0 (11.0)	90.5	73.0	105	0.051
			OE	90.1 (11.5)	90.5	70.0	106	
		Amplitude (μV)	OD	2.99 (0.97)	3.19	1.04	4.66	0.004*
			OE	2.65 (1.03)	2.63	0.53	4.47	
	P1-N1	Amplitude (μV)	OD	3.88 (1.08)	3.73	1.31	5.89	0.435
			OE	3.73 (1.40)	3.80	0.45	6.43	

Wilcoxon Test value of p
Caption: Comp. = Component; P1 = Positive Peak; N1 = Negative Peak; SD = Standard Deviation; ms = Milliseconds; μV = Microvolts; RE = Right Ear; LE = Left Ear; p# = value of P.
* Significant difference.

Table 3. Latency and amplitude values for speech stimuli in the right and left ears

Stimulus	Comp.	Variable	Ear	Average (SD)	Median	Minimum	Maximum	p#
Speech	P1	Latency (ms)	RE	49.6 (10.0)	51.5	35.0	67.0	0.492
			LE	49.4 (10.6)	53.0	35.0	67.5	
		Amplitude (μV)	RE	0.59 (0.67)	0.46	0.10	3.1	0.002*
			LE	1.05 (0.50)	1.11	0.17	2.24	
	N1	Latency (ms)	RE	98.7 (11.1)	99.0	75.0	131.5	0.146
			LE	97.8 (17.2)	101.5	43.5	131	
		Amplitude (μV)	RE	4.46 (1.19)	4.69	1.31	6.33	0.031*
			LE	4.05 (1.08)	3.71	2.02	5.57	
	P1-N1	Amplitude (μV)	RE	4.70 (1.27)	4.84	2.48	7.29	0.162
			LE	4.91 (1.53)	4.82	1.93	7.35	

Wilcoxon Test value of p
Caption: Comp. = Component; P1 = Positive Peak; N1 = Negative Peak; SD = Standard Deviation; ms = Milliseconds; μV = Microvolts; RE = Right Ear; LE = Left Ear; p# = value of P.
* Significant difference.

In the second analysis, the verbal and non-verbal stimuli for the right ear (table 4) and left (table 5) were compared. The results were similar for both ears. P1 latency was significantly higher for speech (p <0.00) in both ears, but for the amplitude variable, no differences were observed. For N1, the values of the two variables, latency and amplitude, were significantly higher for speech in both ears (p <0.05).

Table 4. Latency and amplitude values for click and speech stimuli in the right ear

Ear	Comp.	Variable	Stimulus	Average (SD)	Median	Minimum	Maximum	p#
RE	P1	Latency (ms)	Click	42.8 (11.6)	36.2	30.5	66.0	0.007*
			Speech	49.6 (10.0)	51.5	35.0	67.0	
		Amplitude (μV)	Click	0.82 (0.50)	0.76	0.13	1.58	0.143
			Speech	0.63 (0.72)	0.46	0.1	3.1	
	N1	Latency (ms)	Click	89.0 (11.0)	90.5	73	105	0.001*
			Speech	98.7 (11.1)	99.0	75	131	
		Amplitude (μV)	Click	2.99 (0.97)	3.19	1.04	4.66	0.000*
			Speech	4.46 (1.19)	4.69	1.31	6.33	
	P1-N1	Amplitude (μV)	Click	3.88 (1.0)	3.73	1.31	5.89	0.010*
			Speech	4.70 (1.27)	4.84	2.48	7.29	

Wilcoxon Test value of p
Caption: Comp. = Component; P1 = Positive Peak; N1 = Negative Peak; SD = Standard Deviation; ms = Milliseconds; μV = Microvolts; RE = Right Ear; p# = value of P.
* Significant difference.

Table 5. Latency and amplitude values for the click and speech stimuli in the left ear

Ear	Comp.	Variable	Stimulus	Average (SD)	Median	Minimum	Maximum	p#
LE	P1	Latency (ms)	Click	41.5 (11.2)	36.5	31	66.5	0.000*
			Speech	49.4 (10.6)	53.0	35.0	67.5	
		Amplitude (μV)	Click	1.23 (0.60)	1.39	0.18	6.5	0.465
			Speech	1.05 (0.50)	1.11	0.17	2.24	
	N1	Latency (ms)	Click	90.1 (11.5)	90.5	70.0	106	0.001*
			Speech	97.8 (17.2)	101.5	43.5	131.5	
		Amplitude (μV)	Click	2.65 (1.03)	2.63	0.53	4.47	0.000*
			Speech	4.05 (1.08)	3.71	2.02	5.57	
	P1-N1	Amplitude (μV)	Click	3.73 (1.40)	3.80	0.45	6.43	0.005*
			Speech	4.91 (1.53)	4.82	1.93	7.35	

Wilcoxon Test value of p
Caption: Comp. = Component; P1 = Positive Peak; N1 = Negative Peak; SD = Standard Deviation; ms = Milliseconds; μV = Microvolts; LE = Left Ear; p# = value of P.
* Significant difference.

DISCUSSION

The tests that preceded the PEALL evaluation provide information regarding the functional integrity of the middle ear, auditory sensitivity, and the neural pathways in the brainstem level, as well as the absence of auditory processing disorder. Thus, these results provide support for the conduction of the LLAEP evaluation in conditions of functional integrity of the peripheral and central subcortical auditory pathways in each individual.

Identification of the P1 and N1 components

The P1 component was not identified in all individuals in the present study, a finding supported by the available literature. The component can be identified in all age groups, from 5 to 78 years[9]; however, its presence is described as mandatory in childhood[7,8]. As in this one, two other studies[15,25] did not identify it in approximately one third of its population, using the active Cz electrode and stimuli with frequency and speech specificity as reference. Results similar to the aforementioned ones were presented by other authors[26,27]. Cone et al[26] studied P1 at different intensities; at 60 dB NPS, its occurrence was 86.0% in adults, which is similar to that of the study by Fitzoy et al[27], which reported that for a population aged 17 years, at which age the component is expected to mature completely, it was not observed in all individuals.

Regarding the presence of N1 in all individuals, this has been predicted by previous studies and with different protocols, evidencing its mandatory presence in adulthood[4,9,14-16,25,26].

Right versus left ears

Regarding the results of the analysis between the ears for the click and speech stimuli, P1 presented higher values of amplitude in the left ear for both stimuli, whereas N1 presented higher values in the right ear. These results should be discussed in two aspects.

The first aspect covers the comparison with other studies. Regaçone et al[10] did not observe differences in

the N1 component between ears in their control group, aged from 7 to 14 years. Similarly, Ismail et al.[28] found no distinctions between the ears in a control population aged 8 to 18 years. Oppitz et al.[29] reported differences between the ears for the amplitudes of P1 and N1 and latency of N1, with higher values on the left, for an adult population. However, it is noteworthy that these differences were found in certain groups due to their level of proficiency in speaking English language. It is essential to point out that the three studies referenced above had different populations, as well as different stimulus parameters and records of evoked potentials, which makes it impossible to accurately compare the results.

The second aspect concerns the complexity of the auditory neural pathways. When entering the brainstem, the stimuli coming from the two cochleae have the cochlear nuclei as the first point of processing of acoustic information. From this point, bundles of neurons project simultaneously to several structures, the upper olivary complex being the first binaural convergence point; from there, the representation of the sounds of the two ears is shared in all neural structures ascending to the complex[30]. The complexity of the auditory pathways in the cortex makes a simplistic conclusion of the results impossible; based on the findings and previous studies, there is certainly a differentiation of neural pathways from the right and left ears at the level of the P1 and N1 components in terms of amplitude.

Speech stimulation versus click stimulation

The latency and amplitude differences found between the sound stimuli was expected, with higher values for speech compared to the click, since the processing of sound stimulus by the CANS is related to the complexity of the same. Verbal stimuli are a more complex task of discrimination than non-verbal ones[15]. Another aspect concerns the duration of the stimulus[31]; since the extension is proportional to the decoding time by the structures of the auditory cortex, this may promote prolongation of latency due to acoustic characteristics[11].

Skink and Stuart[14] found similar results as those of the present study when comparing the use of smaller and more complex stimuli. They compared the use of vowels of the same duration, synthetic and natural, and verified higher latency for synthetic stimuli as well. In their study, the duration of the stimulus was not an influential factor, but the complexity of the stimulus was considered. However, in another study that compared verbal and non-verbal stimuli in the oddball paradigm, differences were not observed for P1 and N1[15]. These distinct findings may be due to the methodology adopted, mainly regarding the stimuli; it has already been stated that the diversity of results in the study of the AEP results from methodological variety[32].

As for the amplitude, there were no differences for P1; however, the speech stimulus promoted greater absolute amplitude for N1 as well as for peak-to-peak P1-N1. These results are not corroborated by previous studies[14,15] but are attributed once again to methodological differences; this makes a comparative discussion unfeasible, yet some considerations as to the results found can be realized.

The initial decoding role of the stimulus is attributed to the N1 component; the available literature refers to its role in the investigation of speech perception and discrimination[20,33]. Its developmental maturation is related to the structural refinement of auditory cortical maturation, the development of auditory processing, and improved auditory abilities[4,5]. Thus, the greater amplitude resulting from the complexity of the speech stimulus in front of the non-verbal stimulus may have been promoted by the discriminatory role of the component, which recruited a larger number of neurons in order to decode it.

The present study confirms the hypothesis that acoustic signal processing occurs differently according to the type of stimulus and provides information regarding the differences and similarities of the P1 and N1 record, which can be used as a reference in the study of a population with auditory processing disorder. The latter result supports the relevance of the use of speech for studies of the neural bases responsible for detection and discrimination at the level of CANS. The development of electrophysiological tests that are related to speech discrimination is a challenge; however, these are ideal for evaluations in which individuals do not have the cognitive prerequisites for behavioral tests of speech perception[20].

CONCLUSION

For an adult population, N1 was identified in 100.0% of the individuals, whereas the same did not occur for P1, regardless of the ear and the stimulus used. Although the two components reflected the differences in the processing of acoustic information as a function of the type of stimulus, N1 results evidenced their role in the process of speech discrimination. The reproduction of the present method in a population presented with

Auditory Processing Disorder may contribute to a better understanding of the condition.

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