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# BRAINSTEM AUDITORY EVOKED POTENTIALS AND FREOUENCY-FOLLOWING RESPONSES IN PATIENTS WITH TRAUMATIC BRAIN INJURY

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#### Abstract

Background: Traumatic brain injuries (TBIs) damage the peripheral and central auditory pathways, impairing the patient's sensory and cognitive processing with possible impacts on their quality of life. The objective of this study was to assess the integrity of neural processing in individuals with mild TBI.

Material and methods: A descriptive, quantitative study on 10 people who had suffered mild TBI. We used two procedures: brainstem auditory evoked potentials (BAEPs) using click stimuli at 80 dBSPL and frequency-following responses (FFRs) using a complex (speech) stimulus, the syllable /da/, presented monaurally to the right and left ears.

Results: Abnormal results in the BAEP assessment were characterized as prolonged latencies of waves I, III, and V and interpeak intervals I-III and I-V, bilaterally, whereas in the FFR analysis there were prolonged or absent V, A, D, E, F, and O, components bilaterally.

Conclusions: Mild TBI negatively impacts the neural processing of auditory information, as we observed longer latencies and/or absent components in the BAEP and FFR.

Key words: hearing • cognition • speech perception • electrophysiology • brain injury

# SŁUCHOWE POTENCJAŁY WYWOŁANE PNIA MÓZGU I POTENCJAŁY PODĄŻAJĄCE ZA CZĘSTOTLIWOŚCIĄ U PACJENTÓW Z URAZOWYM USZKODZENIEM MÓZGU

## Streszczenie

Wprowadzenie: Urazowe uszkodzenia mózgu powodują zmiany obwodowych i centralnych odcinków drogi słuchowej, upośledzając zdolności przetwarzania zmysłowego i poznawczego i mogą prowadzić do pogorszenia jakości życia pacjenta. Celem tego badania była ocena integralności przetwarzania informacji w układzie nerwowym u osób z lekkim urazowym uszkodzeniem mózgu.

Materiał i metody: Opisowe badania ilościowe 10 osobach po lekkim urazowym uszkodzeniu mózgu. Zastosowaliśmy dwie procedury badania: słuchowe potencjały wywołane pnia mózgu (BAEPs) z zastosowaniem trzasku o poziomie 80 dB SPL i rejestrację potencjałów podążających za częstotliwością (FFR) z użyciem złożonego bodźca (mowy), sylaby /da/, prezentowanego jednostronnie do lewego i prawego ucha.

Wyniki: Nieprawidłowe wyniki badania BAEP charakteryzowały się obustronnie wydłużonymi latencjami fali I, III, V i wydłużonymi interwałami między szczytami fal I-III i I-V. W analizie FFR zanotowano obustronnie wydłużone lub brakujące komponenty V, A, D, E, F i O.

Wnioski: Lekkie urazowe uszkodzenia mózgu wywierają negatywny wpływ na przetwarzanie informacji słuchowej, gdyż zaobserwowaliśmy wydłużone latencje i/lub brakujące komponenty w badaniu BAEP i FFR.

Słowa kluczowe: słuch • procesy poznawcze • percepcja mowy • elektrofizjologia • uraz mózgu

### Introduction

Traumatic brain injuries (TBIs) are caused by a violent impact on or movement within the cranium, affecting the brain. The pathophysiology of TBI involves two main components, namely primary and secondary brain lesions. A primary lesion occurs at impact, is irreversible, and results from two mechanisms: (i) a direct lesion on the brain tissue due to a (penetrating) projectile or bone fragment, or (ii) a closed brain lesion, in which the impact causes a rupture of the brain tissue and vascular structures. A secondary lesion either occurs in other parts of the brain or extends from the primary lesion. It takes place as the TBI evolves, due to intracranial changes triggered by the primary lesion, such as hematomas, edemas, hydrocephalus, brain inflammatory responses, or systemic changes [1].

In Brazil, information in DATASUS encompassing TBI cases between 2008 and 2012 indicates approximately 125,000 hospitalizations a year due to TBI – an incidence of 65.7 hospitalizations per 100,000 inhabitants per year. According to hospital admission records, the most common causes of TBI from 2001 to 2007 were falls (35%) and accidents at work (31%), totaling 440,000 people. These data show a socioeconomic impact on the country, while other clinical studies report irreversible neurological sequelae resulting from TBI [2]. The more severe the TBI, the higher the risk of motor, cognitive, visual, behavioral, emotional, auditory, and other types of sequelae.

In audiology, it is reported that TBI can impair both the peripheral (outer, middle, and inner ears, and eighth cranial nerve) and central auditory systems (cortical and subcortical pathways), regardless of the severity of the lesion [3–8].

The integrity of the auditory pathways to the brainstem can be assessed with brainstem auditory evoked potentials (BAEPs). The great advantage of using this assessment instrument is that it can identify abnormalities in TBI patients at different degrees of the lesion, from the mildest to the most severe [9–11]. TBI lesions increase the conduction time along the neuronal network involving the structures that pick up this potential. More severe lesions are known to cause long delays in neuronal responses [12].

Since TBI can damage structures in higher areas of the central auditory nervous system, BAEPs need the capability of analyzing this trajectory. There is nowadays a model of one auditory evoked potential, the frequency-following response (FFR), which analyzes these higher regions and can quantify how a person processes verbal and linguistic auditory information without requiring their active participation The FFR is considered an objective method to verify impaired sound coding, reflecting with high precision auditory neurophysiological processes [13]. This is extremely relevant to cases of TBI, potentially benefitting both assessment and monitoring.

People affected with TBI often have a significant loss of verbal and nonverbal sound perception. There is evidence of major changes involving perception of speech sounds in environments with competing noise; these changes are associated with sensory and cognitive impairments, which negatively impact their communications and quality of life [11–12,14–16].

Hence, the objective of this study was to analyze both BAEP and FFR responses in individuals who had suffered a mild TBI.

#### Material and methods

This quantitative descriptive research was conducted at the clinical audiology outpatient center of the Federal University of São Paulo (UNIFESP) as part of a course on hearing disorders conducted by the department of speechlanguage-hearing sciences. It was approved by the Research Ethics Committee at UNIFESP (number 1.844.535).

The sample comprised 10 individuals – 2 females and 8 males, aged 16 to 64 years – with a medical diagnosis of mild closed TBI whose injury had occurred 4 to 12 months previously. We chose this period because spontaneous plasticity takes longer in cases of TBI.

Those who agreed to participate in the study signed an informed consent form. They were invited to an initial assessment to ensure they met the inclusion criteria, which were as follows: absence of previous or current complaints of an affected auditory system; normal auditory thresholds in both ears; good mobility of the tympanic–ossicular chain in both ears; absence of diagnosed and/or evident behavioral or psychiatric changes.

In the initial session, we surveyed their medical history and carried out meatoscopy and basic audiological assessment (pure-tone and speech audiometry and acoustic immittance). The medical history questionnaire was developed by the primary researcher to obtain information on their clinical history regarding hearing and TBI. We also consulted the patients' electronic medical records in the public health service of the university hospital.

The electrophysiological assessment of hearing consisted of BAEP and FFR examinations made with the Smart EP equipment (Intelligent Hearing Systems). The electrodes were positioned following the norms of the international 10-20 electrode system (IES) [17]: active electrode at Cz (vertex), reference electrodes at A1 and A2 (left and right earlobes), and ground electrode on the forehead. To elicit BAEPs, we used click stimuli at 80 dB presented monaurally to the left and right ears at a rate of 19.1 stimuli per second, averaging 2,024 stimuli, and using a 10.66 ms recording window, a 100 Hz high-pass filter, and a 1500 Hz low-pass filter. We made a second recording to reproduce and confirm the tracing, identifying and analyzing the absolute latencies of waves I, III, and V and interpeak intervals I-III, III-V, and I-V (see Figure 1). To assess the integrity of the auditory pathway, we applied the normality criteria used at the UNIFESP services (neurological protocol IHS/UNIFESP, 2017, as shown in Table 1). Values that exceeded 2 standard deviations from the standard for absolute latency and 1 standard deviation for the interpeak intervals were considered abnormal.

We obtained the FFR using the complex speech stimulus /da/ presented monaurally to the right and left ears at 80 dBSPL. We used alternating polarity, presentation rate of 10.9 stimuli per second, 100 k gain, 100 Hz high-pass filter, 2000 Hz low-pass filter, 40 ms stimulation, and 60



**Figure 1.** Screenshots from Smart EP illustrating the way key measures were derived in a typical case. (**A**) Location of 0.00  $\mu$ V (blue arrow). (**B**) Wave V amplitude (blue arrow, 0.23  $\mu$ V). (**C**) Wave A amplitude (blue arrow, -0.13  $\mu$ V)

ms window [18]. During the examination, the subjects watched a video of their choice with no sound and subtitles. We calculated the mean value of two 3,000 stimulation sweeps and, at the end of the collection, we summed the traces and identified the components V, A, D, E, F, and O and the slope. We visually compared the individual traces and their sum to confirm the presence of the components and responses and the replicability of the peaks. We calculated the slope as: Slope = (wave V amplitude – wave A amplitude) / (Wave A latency – wave V latency).

The amplitude measure was established as follows. The wave V amplitude was the difference between the point corresponding to 0.0  $\mu$ V in the wave (Figure 1A) and the maximum positive value (Figure 1B), whereas the wave

A amplitude was the difference between the point corresponding to 0.0  $\mu V$  and the maximum negative value (Figure 1C).

In this study, we used the reference criteria proposed by Skoe et al. [18], set out in **Table 2**. Latency values with variations of up to 1 standard deviation per component were considered normal.

The data were analyzed descriptively, based on the absolute and percentage frequencies for the discrete variables and mean, standard deviation (mean  $\pm$  SD), and median for the numerical variables. We used the paired Student's t-test and Wilcoxon test for the inferential statistical analysis. The margin of error was set at p < 0.05% for the statistical tests. **Table 1.** Normality criteria for the absolute latencies of waves I, III, and V and interpeak intervals I–III, III–V, and I–V in adults (from IHS/UNIFESP, 2017)

Absolute latencies and interpeak intervals	Intensity (dBSPL)	Latency (ms)	Standard deviation (SD)	Standard deviation (+2SD)
I	80	1.65	0.06	1.77
III	80	3.80	0.15	4.10
V	80	5.67	0.16	5.99
I–III	80	2.15	0.16	2.47
III–V	80	1.86	0.12	2.10
I–V	80	4.01	0.17	4.35

Key: dB, decibels; ms, milliseconds; SD, standard deviation.

Table 2. Criteria for normality of the latencies of components V, A, D, E, F, and O (from Skoe et al., 2015)

Age		Latencies of the components of the frequency-following response													
(years)	V	SD	Α	SD	D	SD	Е	SD	F	SD	0	SD			
14–17	6.62	0.27	7.59	0.33	22.53	0.54	31.07	0.54	39.50	0.43	48.15	0.45			
17-21	6.58	0.23	7.53	0.31	22.41	0.40	31.02	0.44	39.50	0.46	48.26	0.34			
21–30	6.65	0.26	7.60	0.34	22.60	0.67	31.12	0.71	39.61	0.62	48.33	0.73			
30–40	6.61	0.33	7.53	0.43	22.52	0.56	31.09	0.50	39.54	0.42	48.21	0.46			
40–50	6.67	0.19	7.64	0.29	22.84	0.71	31.26	0.30	39.49	0.22	48.30	0.65			
50–60	6.86	0.32	7.89	0.44	23.08	0.71	31.57	0.70	39.92	0.77	48.72	1.00			
60–73	6.92	0.38	7.89	0.46	23.05	0.61	31.37	0.55	39.68	0.46	48.84	0.59			

Key: SD, standard deviation.

Table 3. Descriptive measures of the results of the brainstem auditory evoked potentials and comparison between ears

		Ab	solute latency (r	ns)	Interpeak interval (ms)					
Ear		I	III	V	I–III	III–V	I–V			
	Mean	1.71	3.94	5.83	2.23	1.89	4.12			
Right	Standard deviation	±0.11	±0.19	±0.32	±0.18	±0.16	±0.32			
	Median	1.68	3.96	5.83	2.29	1.89	4.19			
	Mean	1.69	4.00	5.86	2.31	1.86	4.17			
Left	Standard deviation	±0.10	±0.18	±0.28	±0.20	±0.16	±0.32			
-	Median	1.65	3.97	5.94	2.32	1.86	4.18			
	<i>p</i> -value	p(1) = 0.468	p(1) = 0.039*	p(1) = 0.264	p(1) = 0.043*	p(1) = 0.324	p(2) = 0.066			

Key: ms, milliseconds. (\*) Significant difference: p < 0.05; p(1) Using paired Student's t-test; p(2) Using paired Wilcoxon test.

## Results

The sample age ranged from 16 to 64 years (mean 44.5, standard deviation 18.3, median 53.0). Most of the patients (80%) were males. As for the age ranges in the FFR assessment, most of the subjects (60%) were over 50 years old.

There were different causes of TBI in the subjects of this study, namely: falling from a place higher than 2 m (40%), falling over from their own height (30%), and vehicle

accident (30%). The brain lesion affected the left side (40%), bilateral (40%), and right side (20%).

All the patients submitted to basic audiological assessment (pure-tone and speech audiometry and acoustic immittance) before the electrophysiological assessment of hearing. The audiometric thresholds were better than or equal to 25 dBnHL from 250 to 8000 Hz, and the tympanometric curves were type A, bilaterally. Thus, all the patients had normal responses in both ears. Table 4. Qualitative analysis of the absolute latencies and interpeak intervals of brainstem auditory evoked potentials for right and left ears

	Right ear –	Nor	rmal	Abn	ormal	Left	Normal		Abnormal	
		n	%	n	%	- ear	n	%	n	%
Absolute latencies <sup>-</sup> (ms) -	I	6	60	4	40	I	6	60	4	40
	Ш	7	70	3	30	Ш	7	70	3	30
	v	6	60	4	40	v	7	70	3	30
Interpeak intervals <sup>-</sup> (ms)	I–III	9	90	1	10	I–III	7	70	3	30
	III–V	10	100	0	0	III–V	10	100	0	0
	I–V	7	70	3	30	I-V	7	70	3	30

Key: ms, milliseconds.

Table 5. Descriptive measures of the results of frequency-following responses and comparison between ears

Ear		Slope (μV/ms)	VA complex (μV)						
		V	А	D	E	F	0		
Right	Mean	6.87	8.18	24.18	33.14	41.06	50.18	0.24	0.29
	Standard deviation	±0.61	±0.50	±1.14	±1.40	±1.24	±0.91	±0.09	±0.08
	Median	6.67	8.24	23.62	33.07	40.70	50.15	0.25	0.29
	Mean	6.98	8.04	23.97	32.36	40.73	50.09	0.23	0.25
Left	Standard deviation	±0.45	±0.52	±0.26	±1.05	±0.35	±2.49	±0.07	±0.08
	Median	6.82	7.80	23.93	32.14	40.65	50.09	0.26	0.25
	p-value	p(2) = 0.048*	p(2) = 0.296	p(1) = 0.317	p(1) = 0.285	p(2) = 0.371	p(1) = 0.655	p(2) = 0.963	p(2) = 0.354

Key: ms, milliseconds;  $\mu$ V, microvolts. (\*) Significant difference, p < 0.05; p(1) Using paired Student's *t*-test; p(2) Using paired Wilcoxon test.

Table 6. Qualitative analysis of latencies in the frequency-following responses for right and left ears

	Right	Nor	mal	Abno	ormal	Left	No	rmal	Abno	ormal	Total
	ear	n	%	n	%	ear	n	%	n	%	(n)
Latency (ms)	V	4	40	6	60	V	5	50	5	50	10
	А	3	30	7	70	А	5	50	5	50	10
	D	0	0	10	100	D	0	0	10	100	10
	E	2	20	8	80	E	3	30	7	70	10
	F	1	10	9	90	F	1	10	9	90	10
	0	2	20	8	80	0	1	10	9	90	10

Key: ms, milliseconds.

The descriptive measures of the BAEP test are shown in **Table 3**. In this table, there was a statistically significant difference between the right and left ears for absolute latency of wave III and interpeak interval I–III, with better results in the right ear.

The qualitative analysis of the BAEP results is shown in **Table 4**. In this table, we observed prolonged latencies of waves I, III, and V and of interpeak intervals I–III and I–V, bilaterally. For waves I and III, we observed similar responses between the ears, where 40% and 30% of the responses were abnormal, respectively.

The results for the interpeak interval I–III were particularly altered in the left ear, where 30% of the responses were abnormal. For the interpeak interval I–V, the responses were similar between the ears, where 30% of the responses were abnormal. For the interpeak interval III–V the whole sample had normal results in both ears.

The FFR quantitative analysis is shown in **Table 5**. There was a statistically significant difference between the right and left ears for wave V, with better results (i.e., lower latency) in the right ear.

The qualitative FFR results are shown in **Table 6**. Here there are abnormal results, characterized as prolonged and/or absent FFR components V, A, D, E, F, and O, bilaterally. In all the subjects, component D was altered (100%), followed by 90% having component F altered in both ears.

# Discussion

TBI can cause focal and/or diffuse lesions involving subcortical and/or cortical regions of the brainstem. Hence, the electrophysiological assessment of hearing is an important instrument to identify changes in the central auditory nervous system.

There was a predominance of males (80%) in the study sample. Studies have shown that men are more susceptible to brain lesions because they are more often involved in intense sports and ride motorcycles and cars [16,19,20]. The literature does not present any correlation between sex and mild TBI, neither was it possible to establish one in this study due to the small sample size.

The literature has discrepant data regarding BAEP responses in TBI. For instance, Munjal et al. [12] conducted a comparative analysis of the absolute latency values of the waves (I, III, and V) and interpeak intervals (I–III, III–V, and I–V), relating them to the degree of severity of TBI. The authors concluded that there is a relationship between the increase in latency values and the severity of the lesion. However, Werff and Rieger [14] compared BAEP responses in normal subjects and mild TBI patients but did not find any difference between the groups.

The qualitative analysis of the BAEP results revealed, in both ears, prolongation of all the absolute latencies and interpeak intervals (except for the interpeak interval III-V). We found a similar percentage of abnormal results between the ears in the analysis of waves I and III and interpeak interval I-V. This may indicate an impairment in the distal portion of the auditory nerve to the brainstem, cochlear nucleus, and lateral lemniscus. For the interpeak interval I-III, we observed better performance in the right ear. These findings corroborate a study [15] that demonstrated a prolongation in the interpeak intervals I-III and I-V in individuals with mild TBI. Other authors [12] observed a prolongation in the interpeak intervals I-III, III-V, and I-V in people with mild TBI, revealing an increase in conduction time in the neural network, especially in the upper brainstem. Atcherson and Steele [11] found abnormal BAEP results in individuals with mild TBI. Even though they did not describe them in terms of absolute latencies and interpeak intervals, the authors pointed out that the result of the electrophysiological assessment could identify small abnormalities in people with mild TBI, as in the present study.

There are few studies involving BAEP responses in mild TBI, and the data they present are not definitive, as they employ different ways of analyzing the sample. The lack of data hinders the generalization of the findings to establish a standard of expected responses in mild TBI [11,12,14,15]. However, the present study can contribute towards this.

FFR data in cases of mild TBI are less common. In our FFR quantitative analysis, we observed a statistically significant

difference between the right and left ears in component V, with a better performance in the right ear. Components V and A represent the transient portion (onset) of the stimulus, reflecting the decoding of quick temporal changes inherent to consonants. That is, in this study we found that the temporal processing of auditory information was faster in the subjects' right ear, which can be explained by the left hemisphere dominance effect for linguistic stimuli. For this very reason, the FFR is conducted exclusively in the right ear.

Nonetheless, in cases of lesions such as TBI, picking up responses from both ears separately may furnish important data to analyses of the FFR. For example, extreme acceleration of the brain and sudden deceleration of the head at the moment of trauma may cause one injured area to consequently affect another [21]. This mechanism reinforces the usefulness of obtaining potentials from each ear (as performed here), particularly in terms of the effects of brain neuroplasticity.

Abnormal FFR results in the onset portion have also been described in mild TBI patients by Werff and Rieger [14]. They suggest inefficient neural coding of auditory information in the brainstem of those with a mild TBI, possibly resulting in neuronal changes in the subcortical auditory pathway, thus reducing speech comprehension. Another study [22] indicates that mild TBI can cause cognitive deficits affecting memory, attention, information processing speed, visual-spatial skills, and verbal fluency. Other authors [23] observed impairment in speech-in-noise comprehension and attention performance over time in individuals with brain concussion, even without a hearing loss.

We observed no statistically significant differences between the ears in the analysis of slope measures and VA complex amplitude. The slope ( $\mu$ V/ms) can be considered a temporal indicator of the synchronized response generators, as VA duration represents the temporal progression of the generation and/or transmission of neural activity, while the VA amplitude represents the synchronicity of electrophysiological activity. In the present study, we observed higher VA complex amplitude values in the right ear, which indicates better neural synchronicity due to the excitation of a larger neuronal network.

The FFR components V, A, D, E, F, and O were considered abnormal in the qualitative analysis when there was a prolonged latency and/or absent component. In this study, we observed prolonged latencies of the components that make up the sustained portion of the FFR (components D, E, and F), especially in the right ear. This may represent inefficient coding of the verbal stimulus related to temporal aspects of auditory information processing in mild TBI patients, giving rise to impaired speech comprehension and, possibly, language skills. Such changes coincide with the clinical manifestations of TBI.

This study contributes to the combined BAEP and FFR assessment in mild TBI patients who may have lesions of the brainstem and cortex. Procedures to assess these regions could help identify dysfunction arising from trauma. The BAEP has been commonly used in studies with TBI. The inclusion in this study of the FFR as an electrophysiological assessment is justified because it is an electrophysiological measure picked up in response to verbal stimuli [24]. The BAEP and FFR responses are obtained with two different stimuli and reflect distinct neural processes [25].

The findings of the present study, showing abnormal BAEP and FFR results, have revealed a greater impairment of the central auditory nervous system in our studied individuals. Taken together, our findings could contribute in an important way to planning these patients' rehabilitation.

# Limitations and future research

It was not possible to include a control group without lesions in this study because of the difficulty in matching them for sex and age. The study was also limited regarding the sample size – our selection criteria prevented a wider and larger sample being recruited. Furthermore, due to equipment limitations, we did not calculate the area of the VA complex amplitude in the FFR.

The findings point to the need for future research in this area, such as: other forms of FFR pick-up (in both silence and noise), correlated studies, results of behavioral

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assessments of central auditory processing, more electrophysiological assessments in individuals with mild TBI, and studies with therapeutic language intervention combined with auditory training. These would provide fresh scientific evidence on the rehabilitation of people with acquired neurological lesions, and confirm the usefulness of the FFR in monitoring central auditory function.

#### Conclusions

Our results have revealed certain abnormal BAEP and FFR responses in patients with mild TBI. This reflects an impairment of neuronal processing of auditory information in these subjects, producing delayed latencies and/or absent components in the electrophysiological responses of the auditory pathway. This study contributes to a widening awareness of the usefulness of combined BAEP and FFR in assessing mild TBI patients.

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