P3 Cognitive Potential in Cochlear Implant Users

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Int Arch Otorhinolaryngol 2018;22:408–414.

Abstract

Introduction The P3 cognitive evoked potential is recorded when a subject correctly identifies, evaluates and processes two different auditory stimuli.

Objective to evaluate the latency and amplitude of the P3 evoked potential in 26 cochlear implant users with post-lingual deafness with good or poor speech recognition scores as compared with normal hearing subjects matched for age and educational level.

Methods In this prospective cohort study, auditory cortical responses were recorded from 26 post-lingual deaf adult cochlear implant users (19 with good and 7 with poor speech recognition scores) and 26 control subjects.

Results There was a significant difference in the P3 latency between cochlear implant users with poor speech recognition scores (G-) and their control group (CG) ($p = 0.04$), and between G- and cochlear implant users with good speech discrimination (G+) ($p = 0.01$). We found no significant difference in the P3 latency between the CG and G+. In this study, all G- patients had deafness due to meningitis, which suggests that higher auditory function was impaired too.

Conclusion Post-lingual deaf adult cochlear implant users in the G- group had prolonged P3 latencies as compared with the CG and the cochlear implant users in the G+ group. The amplitudes were similar between patients and controls. All G- subjects were deaf due to meningitis. These findings suggest that meningitis may have deleterious effects not only on the peripheral auditory system but on the central auditory processing as well.

Keywords
- P3 event-related potentials
- cochlear implant
- speech discrimination

Introduction

Cognitive and linguistic skills, as well as social and emotional behavior are influenced by hearing loss. According to the Brazilian Institute of Geography and Statistics (IBGE, in the Portuguese acronym), 344,200 people have profound hearing loss and may benefit from cochlear implantation.[1] Even using careful selection criteria, not all cochlear implant (CI) recipients show the expected results of good speech and language skills. Results from CIs performed in 2002 at an institution in São Paulo, Brazil, indicate that among 10 implantees, 7 were able to have phone conversations.[2] Sensory deprivation, cause of deafness, incomplete electrode insertion and even psychosocial and personality factors have been related to poor outcome in CI recipients.[3–5]

received
February 22, 2017
accepted
September 3, 2017
published online
April 24, 2018

DOI https://doi.org/10.1055/s-0037-1613687.
ISSN 1809-9777.

All CI recipients go through postoperative speech tests that rely on subjective data. Objective testing of central nervous system (CNS) speech processing is not a routine procedure in postoperative CI protocols. The cognitive P3 potential is a powerful tool to evaluate auditory discrimination abilities and difficulties among CI recipients because it gives useful information about speech recognition, auditory maturation and functional integrity of the central auditory system as well as attention and memory status. Therefore, the plasticity of the central auditory system may be evaluated by the P3 cognitive potential in all CI recipients who understand the test conditions.[6,7]

The P3 event-related late potential is recorded as a positive peak following the late potentials N1, P2 and N2 with a peak latency of $\sim$ 300 to 350 milliseconds (ms) in young normal hearing subjects[6–10] after presentation of two contrasting auditory stimuli (oddball paradigm). The rare or target stimulus is presented with frequency, intensity or variety of speech contrasts and must be identified by the proband. The subject must recognize, categorize and process the auditory stimulus to produce the cognitive potential. The latency depends on the age, attention and memory status of the subject, but no significant differences between genders have been observed.[8] The P3 arises primarily from areas of the temporal lobe and hippocampus.[11,12]

The aim of this study was to evaluate the latency and amplitude of the P3 potential in adult CI recipients with post-lingual deafness with poor and good speech recognition scores, as compared with normal hearing subjects, matched for age and educational level.

Method

One hundred and eleven adults with post-lingual deafness were implanted at the Hospital das Clínicas, Universidade de São Paulo, School of Medicine, Cochlear Implant Center between April 1999 and January 2006. The study was performed according to the guidelines of the Ethics Committee of the Universidade de São Paulo and was approved under the protocol number 1059/07.

The inclusion criteria were: age $\geq$ 18 years on the day of implantation, complete electrode insertion, post-lingual deafness, device activation of at least 3 months, pure tone thresholds of 35 dB HL or better at frequencies between 500 and 8,000 Hz in free-field test condition. We excluded subjects who were not able to understand the test conditions and individuals with neurological or psychiatric disorders. Nine subjects were excluded due to incomplete electrode insertion; 70 because they lived too far away from the implant center, in other states, and 6 because they did not sign the informed consent form.

Thus, 26 adults (13 females) with post-lingual deafness and using unilateral CI met the inclusion criteria of the study. All were fitted with multi-channel cochlear implants, such as Nucleus 22 and Nucleus 24 (Cochlear Ltd., Sydney, Australia; MED-EL, Innsbruck, Austria). The mean age at implant activation was 42.7 years (20–64 years, SD 13.00).

The CI recipients were divided into two groups: G+ with good speech recognition scores ($\geq$ 80% speech recognition in open-set sentences) ($N = 19$ subjects), the "good performers," and G- with poor speech recognition ($<$ 80% speech recognition in open-set sentences) ($N = 7$ subjects), the "poor performers".

Complete electrode insertion was controlled by postoperative radiologic studies. For the G- group, the internal CI unit was tested and retested using the software provided by the manufacturer, Impedance check RA26 (Cochlear Corporation, Denver, CO, USA).

We selected a control subject for each CI recipient, matched for age and educational level. All control subjects had bilateral pure tone audiometric thresholds of 25 dB HL or better at frequencies between 500 and 8,000 Hz, no hearing or tinnitus complaints and no history of otological diseases. Among the 26 controls, there were 15 females (mean age: 44.69 years, from 23–68 years, SD: 14.75). The exclusion criteria were the same as for the study group.

All the participants were informed about the study and signed the informed consent form.

Procedures

Before testing, every subject completed a questionnaire about his or her medical history and had a complete ear, nose and throat examination. Pure tone thresholds and speech recognition scores were obtained from all the subjects.

The cortical auditory evoked responses were recorded with an Amplaid MK 12 equipment (Amplifon, Milan, Italy) in a sound treated, light attenuated room. The subjects were seated comfortably in a reclining chair. The stimulus was delivered by a loudspeaker placed at an angle of 45° on the side of the implant, at a distance of 4.26 feet. For the control subjects, the loudspeaker was placed at an angle of 45° on the right side. The loudspeaker was calibrated to deliver 70 dB HL tone bursts, generated by the Amplaid MK 12 equipment. All the subjects were asked to keep their eyes closed, to point out with the left hand index finger when they heard the rare stimulus and to count silently all the rare stimuli of each run.

Before testing, we explained the test session and introduced the stimuli to all the subjects, encouraging them to be attentive during the whole session and to count only the rare stimuli. Cochlear implant recipients were asked to use their usual device setting.

The subjects had to discriminate between two sounds with different frequencies. In the first test condition, the rare (target) stimulus was a 2,000 Hz tone burst, and the non-target (frequent) stimulus a 1,000 Hz tone burst. In the second condition, the target was a low-frequency tone burst at 1,000 Hz, and the non-target a 1,500 Hz tone burst. This condition is thought to be more difficult. At every test condition, 100 stimuli were presented at the rate of 0.5 per second, with 20 rare (target) stimuli randomly distributed among 80 frequent (non-target) ones, and every condition was run at least twice. The recordings were automatically suspended every time eye movements or other electrical sweeps of great amplitude (rejection level, 100 μV) interfered with the responses, and then a new run was started. Each test session took about 40 minutes.

For evaluation, we selected the best run at each test condition for each subject (waves with highest amplitudes). Only replicable responses were accepted.

The positive silver/silver chloride cup electrode was placed at Cz with reference at M1 or M2 and ground at Fpz. The reference electrode was placed on the non-implanted mastoid in CI recipients and on the right side in the case of controls. Evoked potentials for frequent and rare stimuli were averaged simultaneously, but separately.

The event-related potential P3 was identified at the rare stimulus recording from 230 to 750 ms after stimulus onset. The N1 potential was measured at 50 to 150 ms after stimulus onset and the P2 at 125 to 230 ms.

Data Analysis

Median latencies of N1, P2 and P3 of CI recipients were compared with those of controls (Wilcoxon non-parametric test). Cochlear implant recipients were further subdivided into two groups: G+ and G-. The results were considered significant if $p < 0.05$.

Results

The etiology and duration of deafness, age at implantation, duration of device activation and speech recognition scores in open-set sentences of G+ and G- are shown in ►**Table 1**.

Three control subjects were excluded because their recordings were contaminated by electrical artifacts and despite of filtering and subtraction, no reliable waves could be identified. One subject of G- showed inconsistent responses in both test conditions and was excluded for this reason. The remaining six poor performers completed successfully the test conditions with the exception of one subject, who was not able to discriminate between 1,000 and 1,500 Hz, the second test condition, but nonetheless remained in the G- group.

The median latencies of N1, P2 and P3 of G- (all were deaf due to meningitis) and their controls are shown in ►**Table 2**. The median P3 latencies were significantly prolonged among poor performers in both test conditions as compared with their controls (1,000/2,000 Hz ($p = 0.028$), 1,500/1,000 Hz ($p = 0.042$).

All 19 subjects in the G+ group completed both test conditions. The results of median N1, P2 and P3 latencies for G+ and controls are presented in ►**Table 3**. There was no significant difference in median P3 latency between G+ and controls ($p > 0.05$). On the other hand, median N1 and P2 latencies were significantly longer among G+ as compared with their controls in both test conditions (N1: $p = 0.02$ and $p < 0.001$), (P2: $p = 0.028$ and $p = 0.038$).

When we compared the CI recipients among themselves, G+ outperformed G- with shorter median P3 latencies in both test conditions ($p = 0.009$) and ($p = 0.005$) (►**Table 4**).

The median amplitude of P3 showed no significant difference in either test condition between CI recipients and controls, or between G+ and G-.

The N1, P2 and P3 latencies of each subject are shown in ►**Appendices 1** to **3**.

Table 1 Clinical data of 16 CI users with post-lingual deafness

	G- ($N = 7$)	G+ ($N = 9$)
% of speech recognition in open-set sentences: Mean (Min–Max)	8.6 (0–60)	97.7 (80–100)*
Male: N (%)	4 (66.7%)	9 (56.3%)
Mean Age at CI surgery (years)	38.8 (±10.1)	42.8 (±13.1)
Duration of hearing loss (years): Median (Min–Max)	18.8 (1–36)	12.8 (1–43)
Cochlear implant activation (years): Median (percentile 25–75)	2.7 (1.5–4.2)	1.5 (0.8–3.3)
Nucleus 22 processing strategy: Speak	4	10
Nucleus 24 processing strategy: ACE	3	8
MED-EL Speech processing strategy: CIS	0	1
Etiology of deafness		
Meningitis	7	0
Head trauma	0	3
Otosclerosis	0	3
Ototoxicity	0	2
Chronic otitis media	0	1
Viral infection	0	1
Unknown	0	9

Abbreviations: %, percentage; ACE, Advanced Combination Encoder; CI, cochlear implant; CIS, continuous interleaved sampling; G-, cochlear implant users with poor hearing performance; G+, cochlear implant users with good hearing performance; Min-Max, minimum – maximum; N, sample size.
* ($p < 0.001$).

Table 2 N1, P2 and P3 latencies of G- and CG

	G-	CG	$p^{\dagger}$
	Latency (ms)	Latency (ms)	
1,000/2,000 Hz			
N1 ($n = 4$)	131 (119–224)	132 (113–140)	0.72
P2 ($n = 6$)	266 (180–305)	189 (174–255)	0.17
P3 ($n = 6$)	402 (387–449)	353 (293–369)	**0.028***
1,500/1,000 Hz			
N1 ($n = 3$)	117 (114–228)	135 (123–138)	1
P2 ($n = 5$)	288 (204–302)	195 (180–227)	0.14
P3 ($n = 5$)	453 (431–482)	363 (318–384)	**0.042***

Median (percentile 25–75). † Wilcoxon rank sum test.
*significant *p* value.

Table 3 N1, P2 and P3 latencies of G+ and CG

	G+	CG	$p^{\dagger}$
	Latencies (ms)	Latencies (ms)	
1,000/2,000 Hz			
N1 ($n = 15$)	150 (138–174)	126 (108–132)	**0.02***
P2 ($n = 16$)	230 (205–249)	203 (188–212)	**0.028***
P3 ($n = 19$)	360 (330–387)	360 (336–387)	0.67
1,500/1,000 Hz			
N1 ($n = 18$)	146 (132–173)	123 (108–133)	< **0.001***
P2 ($n = 19$)	228 (201–246)	201 (195–213)	**0.038***
P3 ($n = 19$)	354 (333–396)	384 (363–399)	0.41

Median (percentile 25–75). † Wilcoxon rank sum test.
*significant *p* value.

Discussion

All CI recipients with poor speech recognition scores (G-) presented longer P3 latencies when compared with good performers (G+) and controls (CG), as shown in previous studies.[10] Whereas G+ subjects scored mostly between 90 and 100% in open-set sentences (mean: 97.7%), G- subjects had a mean score of 8.6% of speech recognition with a wide distribution from 0 to 60%. Thus, their performance was significantly poorer. The cause of deafness was meningitis in all, whereas no G+ subject had this etiology. Remaining auditory neuron population is thought to be related to CI

Table 4 N1, P2 and P3 latencies of G- and G+

	G-	G+	$p^{\dagger}$
	Latencies (ms)	Latencies (ms)	
1,000/2,000 Hz			
N1 ($n = 3$)	123 (117–138)	132 (123–138)	0.32
P2 ($n = 4$)	219 (174–271)	170 (143–200)	0.14
P3 ($n = 6$)	402 (387–449)	321 (300–340)	**0.027***
1,500/1,000 Hz			
N1 ($n = 3$)	117 (114–228)	171 (123–183)	0.59
P2 ($n = 5$)	288 (204–302)	204 (200–242)	0.1
P3 ($n = 5$)	453 (431–482)	354 (335–386)	**0.043***

Median (percentile 25–75). † Wilcoxon rank sum test.
*significant *p* value.

performance.[13] Most studies have found the cochlea to be the major site of hearing loss after bacterial meningitis,[14] with support from histological studies,[15–17] due to hair cell destruction and decreased number of spiral ganglion cells. But audiological and neuropsychological assessment of meningitis survivors with valuable hearing suggests that lesions of the central auditory system may also be an important cause of hearing dysfunction, including abnormalities of auditory memory and poor short-term memory,[18–20] which could be responsible for longer P3 latencies among our meningitis patients. These alterations may be subtle, not causing psychological or social problems,[18] so they may be overlooked. The P3 test is a valuable tool to evaluate cognitive function and gives us an insight on how the subject processes auditory information.

The task to detect differences between tonal stimuli is rather simple and may be accomplished easily by CI recipients if the tones are detected by different electrodes. One subject amongst the poor performers did not discriminate between 1,000 and 1,500 Hz, as both frequencies were encoded by the same electrode. In this case the test condition could not be performed. In all other CI recipients P3 was recorded, so the individual speech processor settings or coding strategies did not interfere in the results, differing from Mühler et al.[21]

The P3 potential elicited by this simple task correlated well with speech recognition scores, indicating, like other studies,[22] that it reflects real cognitive activity, and is not just a function of perceived stimulus differences. Therefore, it is not surprising that post-meningitis subjects had a poorer outcome than those who became deaf due to other causes, suggesting discrete CNS dysfunction.

It is interesting, that three patients, deaf due to head trauma, also performed better than the post-meningitis deaf subjects. One possible explanation for that is that central bacterial infection is more devastating, or central nervous plasticity may be more effective to overcome traumatic injury. Recently, it was shown that postural recovery was reduced in CI recipients with poor hearing performance as compared with good CI recipients.[23]

The N1–P2 waves indicate that all subjects detected either or both stimuli; in other words, sound had reached the auditory cortex. This finding suggests functional integrity of auditory pathways, including brainstem pathways up to cortical levels. Not sound perception, but auditory processing was affected, which is important for speech discrimination.[5,8,14] We do not have an explanation for the longer N1 and P2 latencies among G+ as compared with normal subjects. Similar results were found by Beynon et al,[4] probably an effect of our small sample size. The amplitudes of all waves were similar among CI recipients and controls, like in other studies.

Auditory deprivation is thought to be a cause of poor speech discrimination and prolonged P3 latencies among CI users.[8] Although the mean duration of deafness was slightly longer among G- than G+, this difference was not significant. So it could not have accounted for prolonged P3 latencies among this group, contrasting with the findings of Blamey et al.[8] Duration of deafness does not mean auditory deprivation, since all CI candidates at our institution use hearing aids prior to surgery and receive auditory rehabilitation. Furthermore, post-lingual individuals must have preserved speech abilities to be suitable for implantation.

The subjects in G+ had similar P3 latencies as normal hearing subjects, even after long duration of deafness, in agreement with the findings of Kubo et al.[10] These results suggest that in deaf adults, auditory pathways may remain functional over a long period of time and plasticity of the central auditory system is preserved, even when hearing aids do not provide optimal auditory stimulation.

We carefully selected the control subjects, mainly among relatives or friends of the CI recipients, with similar social conditions and educational level to avoid other cognitive or linguistic skills to interfere with P3 results.

In our study, we found that the main variable for poor speech performance and prolonged P3 latencies of CI users was meningitis as cause of deafness. There were no subjects with auditory neuropathy spectrum disorder in our study. As P3 and speech perception scores measure auditory processing and cognitive abilities, these may be impaired in deaf meningitis survivors, even without evident clinical symptoms. We suggest including more specific psychological test batteries in preoperative evaluation of post-meningitis CI candidates to better estimate their performance after surgery. This could prevent them from having unrealistic expectations on the device. The P3 test has proven to be a useful test to evaluate the CI recipient who did not reach the expected speech performance and could be used, among other tests, to reconsider if this patient may benefit from bilateral CI or even a brainstem implant.

Conclusion

In this study, post-lingual deaf adult CI users in the G- group had prolonged P3 latencies as compared with normal CG subjects and CI users in the G+ group. Amplitudes were similar among patients and controls. All CI users with poor recognition scores were deaf due to meningitis. These findings suggest that meningitis may have deleterious effects not only on the peripheral auditory system but on central auditory processing as well.

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Appendix 1 N1, P2, P3 latencies of G+ in both tests

		Latencies (ms) 1,000/2,000			Latencies (ms) 1,500/1,000		
Patient	AGE	N1	P2	P3	N1	P2	P3
AJS	60	138	240	378	135	237	378
AMFC	38	147	237	360	120	249	324
AEN	45	150	207	330	141	237	339
CLNL	53	123	222	450	114	228	474
EGS	30	123	159	333	150	201	342
EPC	50	195	222	327	138	186	330
FJC	35	165	204	357	132	192	378
FRCJ	39	NA	NA	282	183	246	396
GM	39	NA	NA	360	132	195	375
GRS	22	138	189	312	171	204	330
JE	60	159	264	381	132	237	348
LCM	47	132	180	306	123	204	354
MHR	34	NA	273	387	NA	351	486
MFV	19	174	249	405	153	192	306
MFFA	63	126	288	423	183	246	450
MFSP	49	NA	NA	351	180	225	333
RR	60	138	246	333	150	252	345
FFC	67	177	249	390	165	255	402
MHRA	63	165	219	378	177	228	381

Abbreviation: NA, data not available (no reliable potential).

Appendix 2 N1, P2, P3 latencies of G- in both tests

		Latencies (ms) 1,000/2,000			Latencies (ms) 1,500/1,000		
Patient	AGE	N1	P2	P3	N1	P2	P3
AAS	42	252	312	492	228	297	501
JGC	25	123	255	405	NA	NA	NA
LAF	44	177	276	369	NA	288	447
MFCA	42	117	171	393	117	177	414
REC	36	NA	303	435	NA	306	453
RMB (*)	52	NA	NA	NA	NA	NA	NA
VES	20	138	183	399	114	231	462

Abbreviation: NA, data not available (no reliable potential).
*patient excluded from analysis due to inconsistent responses in both tests.

Appendix 3 Latencies of the CG in both tests

		Latencies (ms) 1,000/2,000			Latencies (ms) 1,500/1,000		
	AGE	N1	P2	P3	N1	P2	P3
AFAS	23	141	249	360	135	204	363
NA	68	120	162	375	114	162	390
BBS	49	129	213	360	132	195	393
DC	51	132	198	375	117	255	375
HZE	63	126	201	363	123	291	393
IZS	42	108	177	300	123	195	378
JSS	27	129	198	363	141	210	384
LJJ	35	132	213	339	123	195	390
LS	30	126	225	333	138	204	357
MA	45	126	213	351	135	225	393
MAF	61	129	213	390	111	228	369
MAL	45	96	204	387	108	177	399
MBC	41	150	273	387	105	165	330
MHC	26	135	207	351	123	216	345
MEG	49	126	180	345	126	195	390
MAC	40	99	207	336	99	270	378
NR	31	96	213	261	114	219	339
OV	47	132	192	297	150	213	423
RNC	67	117	204	339	168	240	351
SRS	56	126	186	402	114	210	438
IDN	65	120	186	351	126	180	345
BR	24	156	195	303	141	204	303
TPL	24	135	165	273	138	249	306