The Egyptian Journal of Otolaryngology

ORIGINAL ARTICLE Open Access

Acoustic change complex findings in mild and moderate sensorineural hearing loss

Mona Hamdy[1], Amira El Shennawy[1], Noha Hosny[1], Aya Salah Ezz Elregal[2] and Hussein Sherif Hamdy[1*]

Abstract

Background Auditory electrophysiological tests of the cortex, which are processed in or close to the auditory cortex, are brain reactions to sound. A variation in a continuous stimulus causes the acoustic change complex potential (ACC), which is a wave following the P1-N1-P2 response.

Objective To measure the amplitude and latency of different components of ACC in normal subjects and across individuals with mild and moderate degrees of sensorineural hearing loss (SNHL).

Patients and methods The study includes 100 individuals with the age ranged from 10 to 50 years with different degrees of SNHL. The ACC was evoked by a change of second formant in the middle of ongoing steady-state synthetic, 3 formant vowels (ooee). The total duration was 500 ms. Changing occurred at 250 ms.

Results The SNHL subgroups showed statistically significantly longer P1 and N1 latencies. N1 and P2 amplitudes of ACC onset response were larger with a statistical significance as compared to controls. Post hoc analysis revealed no statistically significant difference between mild and moderate SNHL on ACC parameters. Age showed a significant negative correlation with ACC N1 and P2 latency, ACC P1 and N1 amplitude, and onset P2 latency. Onset response P1 latency was significantly higher in children than adults. Median ACC P1 amplitude significantly increased in children than adults.

Conclusion ACC is a reliable tool for testing the auditory cortex function of detecting difference in sounds presented that can be recorded readily in patients with mild and moderate SNHL.

Keywords Sensorineural, Acoustic change complex, Cortical evoked potentials, Hearing loss

Background

The acoustic change complex (ACC) is an electrophysiological cortical test produced by making changes within a continuous stimulus and it exhibits auditory cortex level discrimination [1].

Surface electrodes put on the scalp can capture the P1-N1-P2, a brief potential in response to a variety of stimuli. Typically, a quick stimulus (with a change within the stimulus) will elicit this potential. It is composed of P-N-P wave recorded between 50 and 200 ms latency. The ensuing waveform is referred to as the ACC (ACC P1N1P2) [2].

According to the findings of various research, changes in intensity, spectrum [3], and/or gap duration [4] can consistently activate the ACC. Martinez et al. [5] showed that the ACC can reliably be elicited in normal hearing and hearing-impaired individuals. The ACC also has a role in investigating the change that occurs in the auditory central pathways that occur with the aging process in normal hearing individuals as well as the hearing impaired as reported in Harris et al. [6] and how the auditory discrimination skill neuromaturation and decline occurs with age. They reported poor temporal processing

*Correspondence:
Hussein Sherif Hamdy
hussein-sherif@kasralainy.edu.eg
[1] Audiology Unit, Otolaryngology Department, Kasr Al Ainy Faculty of Medicine, Cairo University, Cairo, Egypt
[2] Audiology Unit, Otolaryngology Department, Faculty of Medicine, Benha University, Benha, Egypt

skill that was probably due to aging of the auditory pathway when older adults were compared to the young.

Strahm et al. [7] reported difference in the maturation of the ACC between the young infants and older adults when compared using different stimuli implying the objectiveness of the ACC as a tool to assess the age-related maturational effects that occur in the auditory pathway. They concluded that ACC could serve as an objective method for assessing age-related alterations in the brain's ability to process changes in auditory characteristics.

The effect of hearing loss on cortical auditory pathways was reported in various research but few reported the effect on the ACC complex. The aim of this study is to determine the differences in latencies and amplitudes of different components of the ACC complex in hearing loss as compared to normal hearing as well as within different degrees of hearing loss (mild and moderate) and whether the ACC is a valid tool in assessing auditory central pathways in the hearing impaired as compared to normal individuals.

Methods

This is a case-controlled study which was approved by the Otorhinolaryngology Department and Ethical Committee Council of Cairo University (number N-306-2023). It was conducted on 100 subjects, divided as follows.

The study group was composed of fifty patients with age range 10–50 years and mean age of 32 ± 13 years. Thirty females and twenty males with mild to moderate sensorineural hearing loss. The control group was composed of fifty normal hearing individuals age matched to the study group. The age range was from 10 to 50 years and the mean age was 28 ± 13 years. The group included 31 females and 19 males.

Inclusion criteria

- Mild to moderate sensorineural hearing loss.
- Cooperative, of good attention and average intelligence to be able to understand and perform the tests.

Exclusion criteria

- History of neuro-otologic pathology, diabetes mellitus, or hypertension.
- Conductive and mixed hearing loss.
- Moderately severe and severe to profound sensorineural hearing loss.
- Patients older than 50 years old and younger than 10 years old.
- Uncooperative children who could not perform behavioral audiometry.
- Use of antipsychotics drugs, anti-depressants, or other treatments affecting cognition.

Instrumentation

- Two-channel audiometer: Grason-Stadler Inc, Milford (made in USA), New Hampshire (GSI 61) calibrated according to ISO standards.
- Acoustic immittancemeter (Otometrics, Zodiac model), made in USA with a probe tone 226 Hz, calibrated according to ISO standards.
- Biologic Pro-Navigator AEP (model 580_NVBOX1014), made in USA.

Test material

- Live voice speech audiometry including:
  - Arabic bi-syllabic words [8].
  - Arabic phonetically balanced words [9].
- The stimulus used in this study for acoustic change complex test was formed of short duration stimulus (500 ms) to be able to use it on the available instruments. Change occurred at 250-ms interval. It was evoked by a change of second formant in the middle of an ongoing steady-state synthetic 3 formant vowels (ooee) [8]. The fundamental frequency was 150 Hz, first formant was 300 Hz, second formant was 1050 Hz, and third formant was 3000 Hz. This stimulus was previously used in another investigation [10].

All cases undergone complete history taking including sociodemographic data: as age, sex, and occupation. Present history of hearing loss and medical history of drug intake or any current medical condition were recorded as well as family history of hearing loss. Otoscopic examination was performed to exclude wax occlusion.

A 226-Hz probe-tone tympanometry was done to assess middle ear function. Pure-tone behavioral thresholds were obtained from 250 Hz to 8 kHz by air conduction, and pure-tone bone conduction thresholds were obtained at 500 Hz, 1 kHz, 2 kHz, and 4 kHz with the participant seated in a sound-treated room. Speech audiometry including speech reception test (SRT) and word discrimination score (WDS) were done using Arabic spondees [8] and Arabic monosyllabic words [9] respectively presented by monitored live voice.

During acoustic change complex (ACC) recording procedure, participants were half seated comfortably. Electro-encephalogram (EEG) electrodes were applied after cleaning of skin with abrasive gel to minimize skin impedance. The inverting (active) electrode was placed on the high forehead, noninverting (reference) electrode was placed on test right ear mastoid (M2), and ground electrode was placed on the non-test left ear mastoid (M1). Electrode impedance was kept below 5 kΩ for each electrode.

The ACC was recorded using Biologic Pro-Navigator (model 580_NVBOX1014), USA-evoked potential system. The ACC was produced by presenting the stimuli monaurally to the right ear of subjects at 80-dB SPL, using headphones. The analysis window was 1080 ms. The stimulus was presented at repetition rates of 0.7/s with a band-pass filter of 0.1–100 Hz. Fifty sweeps were used or when a reliable response was repeatable as assessed by the examiner.

A response was considered present when it replicated the onset morphology and was expected following the change in the stimulus, i.e., after 250 ms following the onset response. ACC response waves were named as P1′, N1′, and P2′. Latency (in ms) was the time after stimulus onset (or change) which is taken by a given peak to occur. Amplitude (in μV) was the difference between the baseline and the maximum positive peak in case of positive waves or maximum negative peak in case of negative waves.

Statistical analysis

Data management and statistical analysis were done using SPSS vs.25 (IBM, Armonk, NY, USA). Numerical data were assessed for normality using the Shapiro-Wilk test, Kolmogorov-Smirnov test, and direct data visualization methods. Then, numerical data were summarized as means and standard deviations or medians and ranges. Categorical data were summarized as numbers and percentages. Two groups' comparisons were done using independent *t*-test or Mann-Whitney *U* test for normally and non-normally distributed numerical data, respectively. Categorical data were compared using the chi-square test. Different grades of severity were compared using one-way ANOVA or Kruskal-Wallis test for normally and non-normally distributed numerical data, respectively. Correlation analysis was done using Pearson's or Spearman's correlation. All *P* values were two-sided. *P* values less than 0.05 were considered significant [11].

Results

This case-control study was conducted on 50 patients with mild to moderate sensorineural hearing loss and 50 controls. There were non-significant differences between groups regarding age and gender. The age range was from 10 to 50 years (Table 1).

Pure tone audiometry showed that half of the cases (50.0%) showed bilateral symmetrical mild SNHL, and the other half (50.0%) showed bilateral symmetrical moderate SNHL for both ears. All patients had some sort of bilateral amplification from 2 years up to 5 years. They all had history of hearing loss diagnosed since childhood.

The ACC detectability showed non-significant differences between both groups (*P* value = 0.084) (Table 2). Regarding the onset response latency, only P1 and N1 latency were significantly longer in cases than in controls with *P* value 0.047 and 0.024, respectively (Table 3). The onset response amplitude, median N1 and P2 were significantly larger in cases than in

Table 1 General characteristics in both groups

			Cases (n = 50)	Controls (n = 50)	P value
Age (years)	Mean ± SD		32 ±13	28 ±13	0.122
Age categories	Child (10 - 18) Adult (19 - 50)	n (%) n (%)	10 (20.0) 40 (80.0)	17 (34.0) 33 (66.0)	0.115
Gender	Males	n(%)	20 (40.0)	19 (38.0)	0.838
	Females	n(%)	30 (60.0)	31 (62.0)	

Independent t-test was used for age. Chi-square test was used for gender

Table 2 ACC detectability in both groups

		Cases (n = 50)	Controls (n = 50)	P value
ACC	Present	40 (80.0)	46 (92.0)	0.084
	Absent	10 (20.0)	4 (8.0)	

Chi-square test was used

Table 3 Onset response (P1, N1, and P2) latencies and amplitudes in both groups

			Cases (n = 50)	Controls (n = 50)	P value
	Onset latency (msec.)				
P1		Mean ±SD	69.6 ±12	65.1 ±9.7	0.047*
N1		Mean ±SD	114.1 ±15.1	107.9 ±10.7	0.024*
P2		Mean ±SD	197.7 ±22.4	191.4 ±13.4	0.098
	Onset amplitude (μV)				
P1		Median (range)	3 (0.8 -8.2)	2.8 (0.6 - 8)	0.789
N1		Median (range)	-4.4 (-10.1 - - 0.5)	-3.5 (07.7 - - 0.4)	0.046*
P2		Median (range)	4.1 (0.5 - 7.5)	2.7 (0.9 - 8.3)	0.001

Independent t-test was used for latency. Mann Whitney U test was used for amplitude

controls with *P* value 0.046 and 0.001, respectively (Table 3) (Fig. 1).

Regarding ACC latency and amplitude, the values of P1, N1, and P2 showed a non-significant difference (Table 4). The ACC latency, the mean of P1 was 367.9 ms ± 37.4 SD. The mean of N1 was 429 ms ± 45.6 SD and the mean of P2 was 491 ms ± 47 SD. The ACC amplitude, the median for P1 was 2.5 μV with a range of 0.6–6.5 μV, the median for N1 was −2.6 μV with a range of −6.2 to −1 μV, and the median for P2 was 2.8 μV with a range of 1.1–6.6 μV.

ACC detectability showed a non-significant difference between controls and mild (*P* value = 0.067) to moderate (*P* value = 0.052) SNHL group where there was no change in the morphology of the ACC in all individuals, both in normal hearing subjects and in patients with mild or moderate SNHL. The ACC was consistently triphasic (Table 5). Post hoc analysis revealed significantly longer ACC P2 latency in the moderate SNHL group (531 ± 77.2 ms) than the control group (491 ± 47 ms). Non-significant differences were observed between control and mild groups and between mild and moderate SNHL groups. P1 and N1 of ACC latency showed non-significant differences between mild and moderate SNHL (Table 6). ACC P1, N1, and P2 amplitudes showed no significant differences between control and mild to moderate SNHL groups (Table 6).

Hearing loss showed a significant positive correlation with ACC P2 latency ($r = -0.239$ and *P* value = 0.026) which indicates a trend towards longer latency of ACC

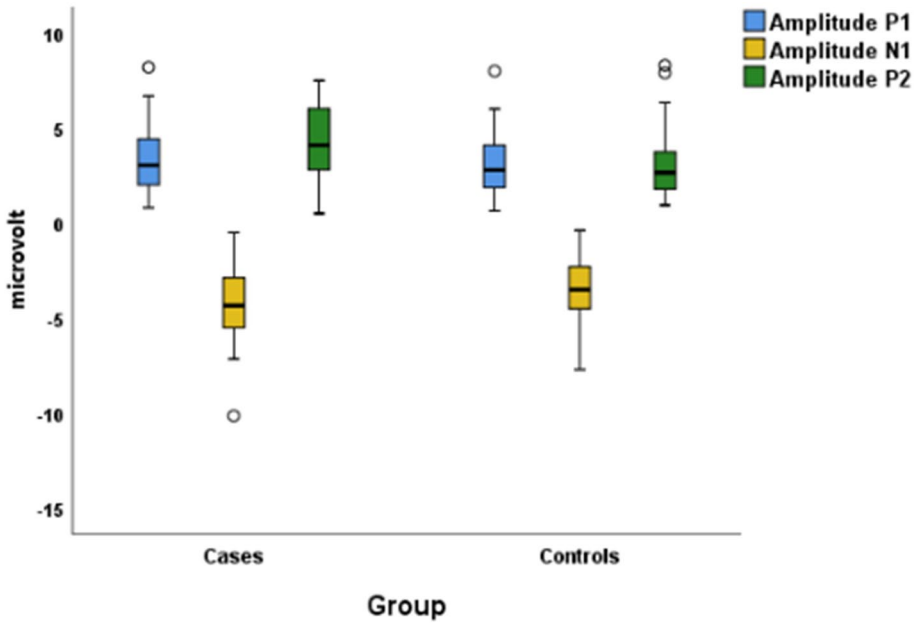

Fig. 1 Onset response amplitude (μV) in both groups

Table 4 ACC (P1, N1, and P2) latencies and amplitudes in both groups

		Cases (n = 50)	Controls (n = 50)	P value
ACC latency (msec.)				
P1	Mean ±SD	374.9 ±50.8	367.9 ±37.4	0.468
N1	Mean ±SD	437.7 ±64.5	429 ±45.6	0.48
P2	Mean ±SD	508.9 ±68.7	491 ±47	0.156
ACC amplitude (μV)				
P1	Median (range)	3 (1.3 - 4.7)	2.5 (0.6 - 6.5)	0.187
N1	Median (range)	-2.8 (-5.6 - -1.3)	-2.6 (-6.2 - -1)	0.588
P2	Median (range)	3.3 (1.2 -7.2)	2.8 (1.1 - 6.6)	0.719

Independent t-test was used for latency. Mann Whitney U test was used for amplitude

P2 with increasing degree of hearing loss (Table 7) (Fig. 2).

Age showed significant negative correlation with the cases group ACC N1 latency ($r = -313$ and P value = 0.049), ACC P2 latency ($r = -0.407$ and P value = 0.009), ACC P1 amplitude ($r = -0.0518$ and P value = 0.001), and N1 of ACC amplitude ($r = -0.486$ and P value = 0.001). This indicates a trend towards shorter latency of ACC N1, ACC P2, and smaller amplitude of ACC P1 and ACC N1 with increasing age in the cases group (Table 8) (Figs. 3 and 4).

Table 5 ACC detectability according to the degree of hearing loss

(A)				
		Normal ($n = 50$)	Mild ($n = 25$)	*P* value
ACC	Present	46 (92.0)	22 (88.0)	0.067
	Absent	4 (8.0)	3 (12.0)	
(B)				
		Normal ($n = 50$)	Moderate ($n = 25$)	*P* value
ACC	Present	46 (92.0)	18 (72.0)	0.052
	Absent	4 (8.0)	7 (28.0)	

Discussion

This case-control study was conducted on 50 patients with bilateral mild to moderate sensorineural hearing loss and 50 controls, all ranging in age from 10 to 50 years. There were non-significant differences in age and gender (Table 1).

The stimulus used in our study was 80-dB SPL speech stimulus. The change occurred at 250 ms. It was evoked by a change of second formant in the middle of ongoing steady-state synthetic, three formant vowels. The fundamental frequency (F0) was 150 Hz, first formant (F1) was 300 Hz, second formant (F2) was 1050 Hz, and third formant (F3) was 3000 Hz (oo-ee) [10].

The ACC was measured by various authors in response to variations in speech stimuli [12–14] and to intensity or variations in frequency amid continuous tones [15–17]. In accordance with the present study, Martin et al. [18] produced an ACC response by applying a 75-dB SPL stimulus. This stimulus had a perceived variation between /u/ and /i/ or from /i/ to /u/. The stimulus was generated with the following parameters: F0 was 100 Hz, F1 was 400 Hz, F2 was 1000 or 2000 Hz, F3 was 3000 Hz, and F4 was 4000 Hz. Tremblay et al. [19] applied speech stimuli that were generated naturally (/si/ and /ʃi/) to trigger the ACC in sensorineural hearing loss. Formant values for /si/ were F1 was 347, F2 was 2655, F3 was 3294, and F4 was 4129 at vowel midpoint.

In the present study, the ACC detectability showed a non-significant difference between both groups. ACC was present in all controls except 4 subjects and in all SNHL subjects except 10 patients (Table 2). Similarly, Jeon [20] reported 100% detectability of ACC in normal hearing children and adults aging from 3 to 19 years. In the study by Martinez et al. [5], in response to changes in speech stimuli, the ACC was produced in both adults and young children. All hearing-impaired children, except for the youngest, displayed ACC P1-N2 responses, while adults exhibited robust ACC P1-N1-P2 responses. They discovered that the evoked response was both influenced by the degree of hearing impairment and the intensity of the stimuli.

Also, in the study by Vonck et al. [21], ACC was recorded in 12 normal-hearing and 13 age-matched hearing-impaired subjects. ACC thresholds increased with hearing loss degree which matches the results

Table 6 ACC (P1, N1, and P2) latencies and amplitudes according to the degree of hearing loss

		Normal (n = 50)	Mild (n = 25)	Moderate (n = 25)	P value
ACC latency (msec.)					
P1	Mean ±SD	367.9 ±37.4	360.3 ±38.9	392.7 ±58.6	0.151
N1	Mean ±SD	429 ±45.6	421.2 ±48.9	457.9 ±76.2	0.229
P2	Mean ±SD	491 ±47 [a]	490.8 ±56.3 [a,b]	531.1 ±77.2 [b]	0.033*
ACC amplitude (μV)					
P1	Median (range)	2.5 (0.6 - 6.5)	3.1 (1.3 - 4.4)	2.8 (1.3 - 4.7)	0.306
N1	Median (range)	-2.6 (-6.2 - -1)	-2.8 (-5.6 - -1.5)	-2.9 (-4.8 - -1.3)	0.849
P2	Median (range)	2.8 (1.1 - 6.6)	3.6 (1.2 - 7.2)	3 (1.5 - 5.9)	0.436

One-way ANOVA test was used for latency. Kruskal Wallis test was used for amplitude. Post hoc was done using Bonferroni. Different letters indicate significant pair

Table 7 Correlation between hearing loss and ACC (P1, N1, and P2) latencies and amplitudes

	Hearing loss	
	r	P-value
ACC latency (msec.)		
P1	0.099	0.363
N1	0.081	0.458
P2	0.239	0.026*
ACC amplitude (μV)		
P1	0.111	0.310
N1	-0.076	0.486
P2	-0.003	0.976

Pearson's correlation was used for latency

Spearman's correlation was used for amplitude

r = Correlation coefficient

*Significant

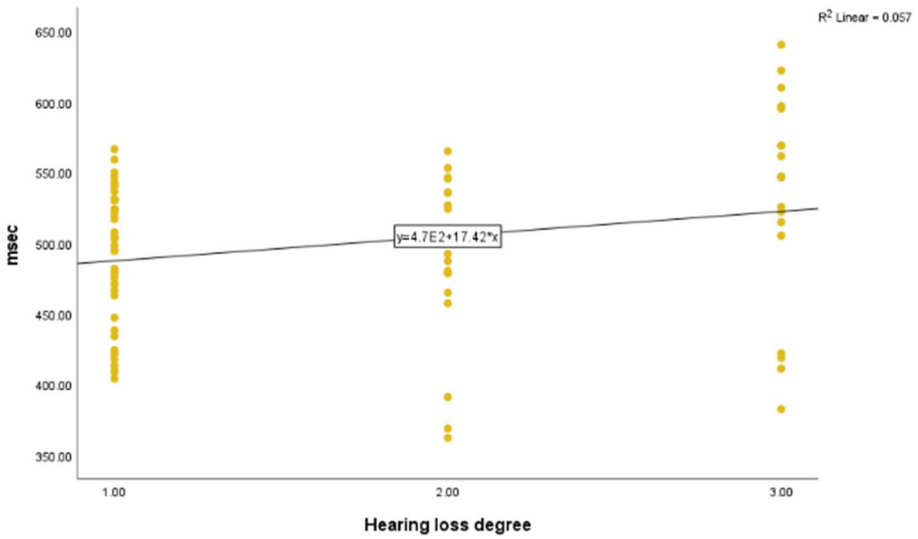

Fig. 2 Correlation between hearing loss and ACC P2 latency

Table 8 Correlation between age and ACC (P1, N1, and P2) latencies and amplitudes of cases group

	Age (years)	
	r	P-value
ACC latency (msec,)		
P1	-0.291	0.069
N1	-.313*	0.049*
P2	-.407*	0.009*
ACC amplitude (uv)		
P1	-.518*	0.001*
N1	-.486*	0.001*
P2	-0.176	0.277

Pearson's correlation was used for latency

Spearman's correlation was used for amplitude

r = Correlation coefficient

*Significant

of this study. Tremblay et al. [19] showed that acoustic changes within a syllable are normally represented in the auditory cortex in subjects with sensorineural hearing loss.

In our study, the ACC P1 latency (374.9 ms), N1 latency (437.7 ms), and P2 latency (508.9 ms) were significantly longer than onset latency P1 (69.6 ms), N1 (114.1 ms), and P2 (197.7 ms); *P* value was <0.001 (Tables 3 and 4).

Median of onset response N1 amplitude was significantly higher than median ACC N1 amplitude. The median of onset response P2 amplitude was also significantly higher than the median ACC P2 amplitude.

This agreed with Elkholy et al. [22] study which concluded that ACC had the same morphology of the onset response in most subjects, with longer latency and smaller amplitude. Jeon [20] also reported the same as well as Lister et al. [23], who also found the same results as regards P2 latency in young adults with normal hearing. On the other hand, He et al. [24] reported that ACC had a delayed latency and larger amplitude in most subjects.

Post hoc analysis revealed significantly longer ACC P2 latency in the cases with moderate SNHL than the control group (Tables 5 and 6). Regarding ACC amplitude,

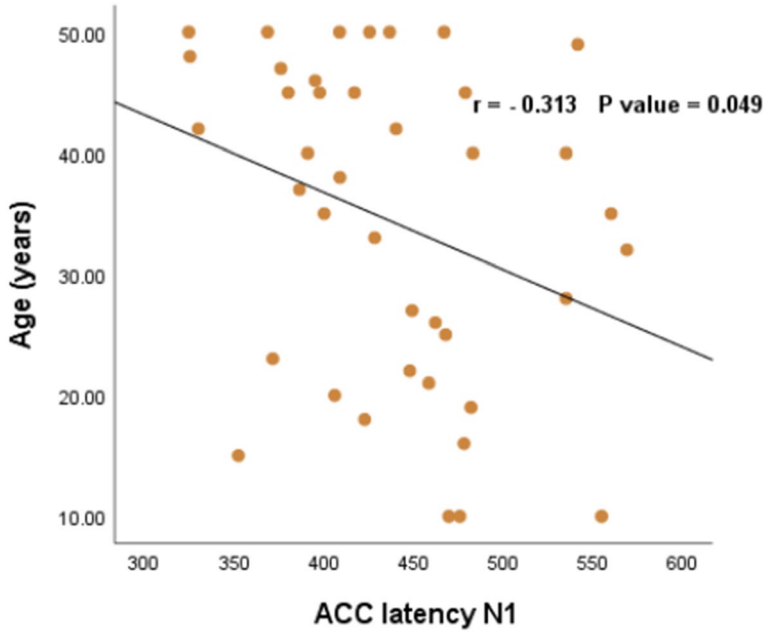

Fig. 3 Correlation between age and ACC N1 latency of cases group

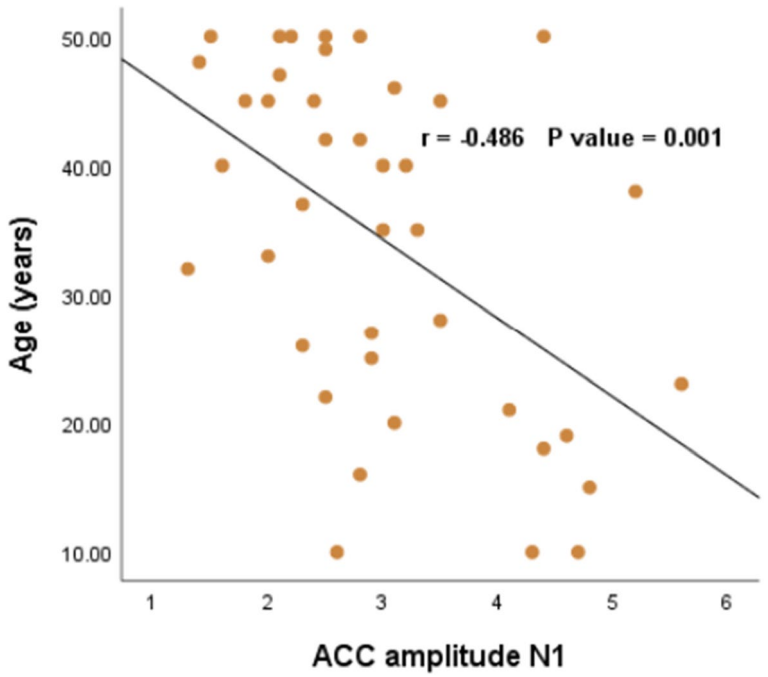

Fig. 4 Correlation between age and ACC N1 amplitude of cases group

the median amplitude of the ACC P1, ACC N1, and ACC P2 were larger in the cases of SNHL when compared to normal subjects; however, there was a non-statistical difference. ACC P1, N1, and P2 amplitude showed a non-statistically significant difference between the control group and mild to moderate SNHL group (Tables 5 and 6).

Wall et al. [25] studied ACC response in hearing loss and reported non-significant differences in the latencies and amplitudes when compared to normal hearing except for ACC N1 amplitude which was reduced in SNHL patients. The present study disagreed with the results of Vonck et al. [21] which reported that amplitudes of the

ACC varied widely amongst patients, and even within the NH group. ACC amplitudes in subjects with normal hearing appeared to be higher than those in subjects with SNHL. The ACC amplitudes of the more severe SNHL participants were typically lower than those of the less SNHL subjects.

In the study performed by Martin et al. [26], the ACC was reliably produced in sensorineural hearing loss; the ACC decreased in amplitude and increased in latency as the degree of second formant frequency change decreased. Elkholy et al. [22] also concluded that ACC amplitude, which is consistently influenced by magnitude of change, is a better predictor of cortical detection than latency. In accordance with the present study, Tremblay et al. [19] found that the ACC amplitudes were reduced in the hearing-impaired group. Jerger and Jerger [27] observed that the behavioral audiometry differences in both intensity and frequency were matched in evoked potentials amplitude response which agrees with the present study.

In the present study, the age showed significant negative correlation with ACC N1 latency, ACC P2 latency, ACC P1 amplitude, and ACC N1 amplitude of the hearing loss group. This indicates a trend towards shorter latency of ACC N1, ACC P2, and smaller amplitude of ACC P1 and ACC N1 with increasing age. Median ACC P1 amplitude was significantly larger in children than adults; *P* value was 0.001 (Tables 7 and 8). In the study by Jeon [20], they stated that as age increases, the ACC P1 latency is considerably reduced. Only around 10% of the variability in the amplitude of ACC responses was predicted by age in terms of amplitude (Tables 7 and 8) which agrees with our study.

In the study done by Elkholy et al. [22] which is in accordance with the results of the present study, a correlation was done between subject's age and ACC response parameters using different stimuli. It was found that age was negatively correlated to ACC P1 latency and amplitude evoked by 25% change, /a-i/ and /a-u/ stimuli. As regards ACC N2 latencies, those evoked by /a-i/ and /a-u/ stimuli were negatively correlated with age and there was no correlation between age and ACC N2 amplitude. However, Strahm et al. [7] disagreed with the present study reporting that adults showed an increase in ACC N1 amplitude when compared to the young.

Conclusion

In conclusion, these findings suggest that the ACC detectability, latency, and amplitude are highly variable in individuals with SNHL, based on the methodology used, the stimulus parameters, subjects' age, and the degree of hearing loss.

ACC response can be recorded reliably in patients with mild and moderate SNHL at the auditory cortical level in normal hearing and in patients with SNHL. However, normal hearing and SNHL cannot be differentiated by ACC parameters. Moreover, age is inversely related to ACC N1 and P2 latency. ACC P1 and N1 amplitudes are inversely correlated to age.

There are numerous advantages of recording ACC over other similar potentials (MMN, P300), including the fact that it does not require attention and can be elicited even in the absence of attention. It is also a potential clinical tool for investigating the brain processing of speech in people with hearing loss especially difficult-to-test groups.

Recommendation

Further research is required to study ACC in different degrees of hearing loss using different stimuli. Also, the effect of hearing aid use and cochlear implants on ACC parameters should be further investigated.

Limitations

All patients had some sort of amplification bilaterally for at least 2 years up to 5 years of regular use. It is reported in the literature that hearing aid use is a confounding factor that might have contaminated our results as regards ACC latencies and amplitudes, however the main aim of this study was to compare hearing loss patients with normal individuals and hearing aid use was out of the scope of the present study. Also, the results of the study were confounded by the limited number of patients in the children age group affecting the maturation of the ACC.

Acknowledgements
Not applicable.

Authors' contributions
HH and NH contributed by sharing in writing the paper and submission and correspondence. AS and AE contributed to the application of the idea, steps of the methods, and supervising the study work. AS and MH supervised the work. AE and NH contributed to data collection and writing the paper. All authors have read and approved the manuscript.

Funding
Not applicable.

Availability of data and materials
The datasets during and/or analyzed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study has been approved by the Research Ethics Committee of Kasr Al-Aini Hospital, Cairo University (number N-306-2023). An informed written consent to participate in the study was provided by all participants including parents/guardians of patients under 16 years old.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests

Received: 29 September 2023 Accepted: 2 February 2024
Published online: 14 February 2024

References

1. Chen KH, Small SA (2015) Elicitation of the acoustic change complex to long-duration speech stimuli in four-month-old infants. Int. J, Otolaryngol, p 562030
2. Kim J (2015) Acoustic change complex: clinical implications. J. Audio. Otol 19(3):120–124
3. Dimitrijevic A, Starr A, Bhatt S et al (2011) Auditory cortical N100 in pre- and post-synaptic auditory neuropathy to frequency or intensity changes of continuous tones. Clin. Neurophysiol 122(3):594–604
4. Michalewski HJ, Starr A, Nguyen TT, Kong YY, Zeng FG (2005) Auditory temporal processes in normal-hearing individuals and in patients with auditory neuropathy. Clin. Neurophysiol 116(3):669–80
5. Martinez AS, Eisenberg LS, Boothroyd A (2013) The acoustic change complex in young children with hearing loss: a preliminary study. Semin. Hear 34(4):278–87
6. Harris KC, Mills JH, Dubno JR (2007) Electrophysiologic correlates of intensity discrimination in cortical evoked potentials of younger and older adults. Hear Res 228(1–2):58–68
7. Strahm S, Small SA, Chan S, Tian DY, Sharma M (2022) The maturation of the acoustic change complex in response to iterated ripple noise in 'normal'-hearing infants, toddlers, and adults. J Am Acad Audiol. 33(5):301–310. https://doi.org/10.1055/a-1862-0198. (Epub 2022 May 25 PMID: 35613945)
8. Soliman S, El-Mahalawi T (1984) Simple speech test as a predictor for speech reception threshold (SRT) in preschool children. Unpublished master thesis, Ain Shams University, Cairo
9. Soliman S (1976) Speech discrimination audiometry using Arabic phonetically balanced words. Ain Shams Medical Journal 27:27–30
10. Boothroyd A (2004) Measuring auditory speech perception capacity in very young children. International Congress Series 1273:292–295
11. Levesque R (2005) SPSS programming and data management: a guide for SPSS and SAS users. SPSS, Fourth edition book
12. Martin BA, Boothroyd A (2000) Cortical, auditory, evoked potentials in response to changes of spectrum and amplitude. J. Acoust. Soc. Am 107(4):2155–61
13. Tremblay K, Friesen L, Martin B, Wright R (2003) Test-retest reliability of cortical evoked potentials using naturally produced speech sounds. Ear Hear 24(3):225–232
14. Friesen L, Tremblay K (2006) Acoustic change complexes recorded in adult cochlear implant listeners. Ear Hear 27(6):678–685
15. Pratt H, Starr A, Michalewski HJ, Dimitrijevic A, Bleich N, Mittelman N (2009) Auditory-evoked potentials to frequency increase and decrease of high- and low-frequency tones. Clin. Neurophysiol 120(2):360–373
16. Presacco A, Middlebrooks JC (2018) Tone-evoked acoustic change complex (ACC) recorded in a sedated animal model. J. Assoc. Res. Otolaryngol 19(4):451–466
17. Vonck BMD, Lammers MJW, de Waals MV, Zanten GAV, Versnel H (2019) Cortical auditory evoked potentials in response to frequency changes with varied magnitude, rate, and direction. J Assoc Res Otolaryngol 20(5):489–498
18. Martin BA, Boothroyd A, Ali D, Leach-Berth T (2010) Stimulus presentation strategies for eliciting the acoustic change complex: increasing efficiency. Ear and Hearing 31(3):356–366
19. Tremblay K, Billings C, Friesen L, Souza P (2006) Neural representation of amplified speech sounds. Ear and Hearing 27(2):93–103
20. Jeon E (2016) The effect of development on cortical auditory evoked potentials in normal hearing listeners and cochlear implant users. PhD (Doctor of Philosophy) thesis, University of Iowa, Iowa, p 38–39
21. Vonck BMD, Lammers MJW, Schaakea WAA, Zanten GAV, Stokroos RJ, Versnela H (2021) Cortical potentials evoked by tone frequency changes compared to frequency discrimination and speech perception: thresholds in normal-hearing and hearing- impaired subjects. Hearing Research 401:108154
22. Elkholy WA, Hassan DM, Shafik NA, Eltoukhy YEK (2020) Acoustic Change Complex (ACC) using Speech and Non-Speech Stimuli in Normal Hearing Children. QJM: Int J Med 113(Supplement_1):hcaa047.008. https://doi.org/10.1093/qjmed/hcaa047.008
23. Lister JJ, Maxfield ND, Pitt GJ (2007) Cortical evoked response to gaps in noise: within channel and across channel conditions. Ear Hear 28(6):862–78
24. He S, Grose J.H, Buchman C.A (2012) Auditory discrimination: the relationship between psychophysical and electrophysiological measures. Int. J. Audiol 51(10):771–82.27
25. Wall L, Dalebout S, Davidson S, Fox R (1991) Effect of hearing impairment on event-related potentials for tone and speech distinctions. Folia Phoniatrica 43(6):265–74
26. Martin BA, Tremblay KL, Korczak P (2008) Speech evoked potentials: from the laboratory to the clinic. Ear Hear 29(3):285–313
27. Jerger J, Jerger S (1970) Evoked response to intensity and frequency change. Arch Otolaryngol 91(05):433–436

Publisher's Note