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OPTIMISING PARAMETERS FOR MEASURING THE ACOUSTIC CHANGE COMPLEX WITH AMPLITUDE-MODULATED STIMULI USING A CLINICAL EVOKED-POTENTIAL SYSTEM

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ABSTRACT

The Auditory Change Complex (ACC) is a transient cortical auditory-evoked potential, recorded via electroencephalography (EEG), and elicited by changes in an ongoing auditory stimulus. It provides insight into auditory discrimination, making it a relevant tool for evaluating cochlear implant (CI) performance. Previous studies have demonstrated that the ACC can be recorded in CI recipients, and many have reported a strong correlation with behavioural outcomes. However, these studies typically used equipment and analytical techniques not widely available in clinical settings.

As a first step toward developing a clinically feasible method for ACC recording, we adapted our EEG research protocol for amplitude-modulated (AM) rate discrimination for use with normal hearing listeners using the Eclipse system (Interacoustics). Our findings show that the ACC can be reliably recorded using this system, with good test-retest reliability. As expected, larger AM rate changes and lower carrier frequencies (CFs) elicited greater ACC amplitudes. Furthermore, increasing the presentation rate by reducing the inter-stimulus interval diminished the ACC response, and changing presentation level had minimal and inconsistent effects across participants. We are now exploring optimal AM rate changes and recording parameters for CI users, and will discuss preliminary results regarding the potential for routine clinical use in CI fittings.

Keywords: auditory change complex, cochlear implants, amplitude modulation, electroencephalography

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1. INTRODUCTION

Cochlear implants (CIs) are electronic hearing devices used to restore hearing in individuals with severe-to-profound deafness. They work by decomposing incoming sounds through a bank of bandpass filters, extracting the slowly-varying envelope cues within each filter band and using them to amplitude modulate fixed-rate electrical pulse trains presented to each electrode [1-2].

Envelope cues are crucial for speech understanding as they convey linguistic information across a range of modulation frequencies. Typical speech envelope modulations occur

within the 2-50 Hz range, with lower modulation rates (~2-5 Hz) providing syllabic and prosodic information and higher rates (~5-50 Hz) providing manner of articulation and voicing cues [3]. Modulation rates above 50 Hz provide pitch cues and the ability to discriminate periodic from aperiodic sounds. These modulation frequencies are associated with oscillatory activity in the auditory cortex. Delta (1-4 Hz), theta (4-8 Hz), and low gamma (25-50 Hz) oscillations are known to play crucial roles in processing the multi-timescale, rhythmic properties inherent in speech, facilitating the tracking of its dynamic nature [4]. These oscillatory patterns serve as foundational elements in speech and language processing, aiding in the segmentation of incoming information into units of appropriate temporal scales [4]. Slower oscillations, such as delta and theta, are implicated in phrasal and syllabic processing, while faster oscillations in the low gamma range are associated with phoneme-level processing.

Despite the success of cochlear implants, there remains significant variability in speech-perception outcomes among CI users [5-10]. One factor that contributes to this variability is the ability to accurately encode and process the temporal and spectral features of sound, which are essential for speech understanding, particularly in complex listening environments [11-13].

Although subjective speech-perception tests remain the standard method of assessing CI benefit, they are limited in scope. These behavioural measures are influenced by non-auditory factors such as attention, motivation, and cognitive ability, and are not suitable for individuals—such as young children or some adults—who cannot reliably complete listening tasks. This makes it challenging to determine whether a CI has been optimally programmed to provide access to the full range of relevant speech cues. Currently, however, there are no standardised, objective approaches to verify CI fitting. Tools such as electrically evoked compound action potentials (eCAPs) can assist in setting initial stimulation levels, but they provide only a rough indication of neural responsiveness and do not confirm functional access to speech information.

There is growing interest in using objective, neurophysiological measures to inform and verify fitting based on auditory detection and discrimination abilities in CI users. One such measure with the potential to support this goal is the Auditory Change Complex (ACC), a cortical auditory-evoked potential elicited in response to a change in an ongoing auditory stimulus [14-15]. The ACC typically exhibits the standard obligatory cortical P1–N1–P2 morphology, similar to responses evoked by sound onset, but time-locked to the point of stimulus change. This makes it a valuable tool for assessing auditory discrimination in a way that is both objective and functionally meaningful.

Previous studies have demonstrated that the ACC can be reliably recorded in CI users in response to changes in place of stimulation [16-19], indicating electrode discrimination abilities; rate of amplitude modulation—typically for rates below 50 Hz [19-22], which relates to the ability of the listener to use envelope cues—and stimulation level [23], as well as speech tokens [24-26]. Several studies have also reported correlations between ACC responses and behavioural speech perception measures [27, 28] and the development of these responses over time following device activation [29]. However, these studies have often utilised recording equipment and analytical techniques that are not typically available in clinical settings.

The primary aim of this study was to evaluate the feasibility of recording the ACC to changes in the AM rate using a clinical evoked-potential system (Eclipse, Interacoustics). We performed an initial study with normal-hearing listeners to validate our protocol and ensure that responses could be obtained for stimuli that have previously been used with the Biosemi EEG system [4]. We wanted to investigate the effects of various stimulus parameters on the ACC response amplitude, similar to the approach taken by Calcus et al [15], but using the Eclipse system. Both the N1-P2 and Root Mean Square (RMS) amplitude were evaluated. The parameters investigated were the magnitude of the AM rate change, carrier frequency (CF; equivalent to a centre frequency of a CI channel), presentation rate (adjusted by increasing the inter-stimulus interval), presentation level and direction of AM change (low to high versus high to low). We also evaluated test-retest reliability across two test sessions.

In a second phase of testing we are exploring the optimal AM rate change to use and are evaluating whether the Eclipse can be used to measure ACC responses in CI users.

2. METHODS

Two experiments were conducted using a two-channel clinical evoked potential system (Eclipse, Interacoustics). Experiment 1 investigated the effects of CF, stimulus level, AM rate change, presentation rate (adjusted by changing inter-stimulus-interval) and direction of AM rate change

(low to high or high to low) on the ACC amplitude. Test-retest reliability across sessions was also examined. Experiment 2 explores the interaction between CF and AM rate on the ACC amplitude, and testing is still ongoing.

2.1 Experiment 1

2.1.1 Participants

Twelve participants (9 females, 3 males) aged 26 to 44 years (mean age = 34.8 years) were recruited. All had normal hearing thresholds. The full experiment took two sessions to complete. The first session took two to three hours, and the follow-up session took approximately 30 minutes.

2.1.2 Stimuli and paradigm

Stimuli were 900-ms sinusoidally amplitude-modulated (AM) pure tones, created using custom MATLAB scripts. Stimulus presentation was controlled using the Eclipse EP software (Interacoustics) connected to an SP90A active loudspeaker (RadioEar). All stimuli were calibrated at 65 dB SPL prior to testing.

A total of four CFs were tested, including 626, 1438, 3313, and 5688 Hz, which correspond to the centre frequencies of electrodes 19, 13, 7 and 3 in the Cochlear Ltd device (assuming all electrodes are active). Halfway through the stimulus (at 450 ms), the AM rate was changed from 12 to 114 Hz (or vice versa). The modulation depth was 100%, and the presentation rate was set to 0.5 Hz (i.e., 1100-ms inter-stimulus interval, the quiet period between consecutive stimulus presentations). At the CF that elicited the largest ACC amplitude (usually 626 Hz), the effects of the magnitude of the AM rate change (12-40, 12-80, 12-114 Hz), presentation level (55, 65, 75 dB SPL), direction of the change (12-114 (up; low-to-high), 114-12 (down; high-to-low), and presentation rate (0.5 & 0.8 Hz with an inter-stimulus interval of 1100 ms and 350 ms respectively) were also assessed. Test-retest reliability of the ACC was evaluated by repeating the recording at the best CF and at a 12-114Hz AM rate change on Session 2. Finally, we included a 12-Hz AM no-change condition at the best CF. An example stimulus with an AM rate change is shown in Figure 1.

All conditions were presented in three blocks until 100 sweeps were accepted for each block (total of 300 sweeps per condition). Participants sat in a comfortable chair and watched a subtitled video for the duration of the experiment.

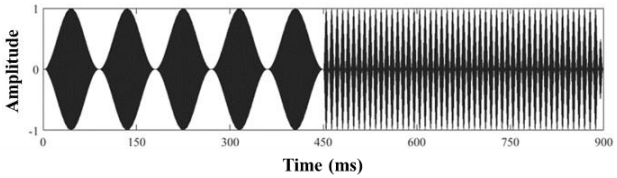

Figure 1. Example stimulus with an AM rate change. The CF for this stimulus is 1438 Hz, and the AM rate change is 12 to 114 Hz (change halfway at 450 ms).

2.1.3 EEG recording, EEG analysis, and statistical analysis

All EEG recordings were obtained using the Eclipse EP system (Interacoustics) at a sampling rate of 500 Hz. Pre-gelled disposable electrodes were placed on FPz (active), the low forehead (ground), and both mastoids (linked reference) in accordance with the standard clinical protocol. Impedances were checked prior to testing and were kept below 3 kΩ. The artefact rejection threshold was set to 40 or 80 µV depending on the participant, and the recording automatically stopped after 100 accepted sweeps. Each sweep (-90 to 810 ms post-stimulus onset) was saved for offline analysis with a 0.5-Hz high-pass hardware filter.

Individual sweeps were low-pass filtered at 30 Hz and baseline-corrected using the pre-stimulus quiet baseline period (-90 to 0 ms post-stimulus onset). For each participant and condition, all 300 sweeps were then averaged into one waveform. The amplitude of the ACC was quantified by determining the peak-to-peak amplitude for N1 and P2, which were defined as the lowest and highest amplitude values between 500-600 and 575-750 ms, respectively. The N1-P2 amplitude was chosen as the primary measure as it is the clinical standard. However, we also measured the root mean squared (RMS) amplitude within the 500-750-ms window post-stimulus onset for comparison. RMS amplitude provides an index of overall response magnitude that is less sensitive to individual differences in waveform morphology and avoids the subjectivity involved in identifying component peaks. This might make it a more suitable option for automated analysis.

Data were analysed with Repeated-Measures Analysis of Variance (RM-ANOVA) and Paired-Samples T-Tests, using jamovi (version 2.6) statistical software. Separate analyses were run to assess the effects of CF (626, 1438, 3313, 5688 Hz), magnitude of AM rate change (12-40, 12-80, 12-114 Hz), stimulus level (55, 65, 75 dB SPL), presentation rate (0.5, 0.8 Hz) and AM direction (12-114 (up), 114-12 (down)). Post-hoc pairwise comparisons were

corrected for multiple comparisons using a Bonferroni correction. Test-retest reliability (Session 1, Session 2) was assessed using a two-way random-effects intraclass correlation coefficient (ICC(2,k)) and Spearman's rank correlation.

2.2 Experiment 2

2.2.1 Participants

Four participants (2 females) aged 35 to 43 years (mean age = 39.75 years) were recruited to this pilot study. All had normal hearing thresholds. Two of the four participants had previously been tested in experiment 1.

2.2.2 Stimuli and paradigm

The stimuli were created, controlled and calibrated in the same way as in Experiment 1. Three CFs were tested (626, 1438 and 3313 Hz) at three AM rate changes (12-40, 12-80, 12-114 Hz) and a no-change condition at each CF.

2.2.3 EEG recording, EEG analysis, and statistical analysis

As per Experiment 1 (see 2.1.3).

3. RESULTS

3.1 Experiment 1

The coloured lines in Figure 2 show the group-grand-average waveforms (the average waveform computed across all participants, after first averaging each individual's responses across trials) for the largest AM rate change (i.e., 12-114 Hz) measured at four different CFs, as well as the group-grand-average waveform for the no-change condition in black. The waveforms show an initial typical onset response and an ACC after the AM change within the expected time windows.

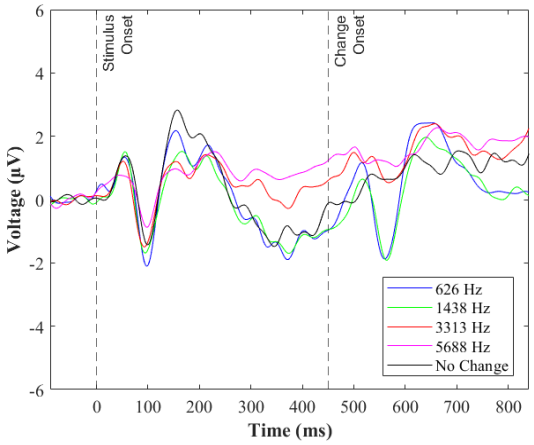

Figure 2. Group-grand-average waveforms for the change (coloured) and no-change conditions (black lines). Different colours represent different CFs. The 'change onset' at 450 ms indicates a change from 12- to 114-Hz AM rate. The group grand average refers to the average waveform computed across all participants, after first averaging each individual's responses across trials.

Figure 3 shows the mean ACC N1-P2 amplitude and standard deviation for all conditions tested in this study. Grey circles show single-participant data. Coloured bars show change conditions, and the no-change condition is shown in black. Change conditions generally evoked larger ACCs compared to the no-change condition.

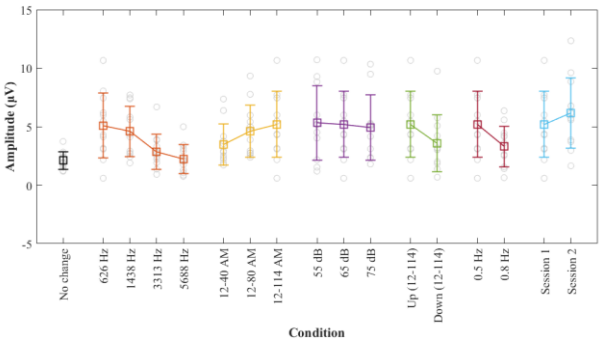

Figure 3. Mean ACC N1-P2 amplitude and standard deviations (± 1) for all conditions tested in this study. Coloured bars show change conditions grouped by their manipulation, and the no-change condition is

shown in black. Grey circles show single-participant data.

3.1.1 N1-P2 amplitude

RM-ANOVA showed a significant main effect of CF ($F(3,33) = 16.7$, $p < .001$) and AM rate change ($F(2,22) = 13.9$, $p < .001$) but no significant main effect of stimulus level ($F(2,20) = 0.512$, $p = 0.607$) on N1-P2 amplitude.

Post-hoc comparisons revealed significantly greater N1–P2 amplitudes at lower CFs: 626 Hz elicited larger responses than 3313 Hz ($p = .018$) and 5688 Hz ($p = .002$); and responses at 1438 Hz were significantly larger than those at 3313 Hz ($p = .015$) and 5688 Hz ($p = .002$). For AM rate change, amplitudes were significantly smaller at 12–40 Hz compared to both 12–80 Hz ($p = .045$) and 12–114 Hz ($p = .012$). Presentation rate also significantly affected amplitude, with larger responses at 0.5 Hz ($M = 5.20$; $SD = 2.82$) versus those at 0.8 Hz ($M = 3.30$; $SD = 1.70$), $t(11) = 2.76$, $p = .019$, Cohen's $d = 0.80$. There was no statistically significant difference in N1-P2 amplitude between upward (12-114 Hz) ($M = 5.20$; $SD = 2.82$) and downward (114-12 Hz) ($M = 3.60$; $SD = 2.43$); $t(11) = 1.95$, $p = 0.077$. direction of AM rate change. There was good test-retest reliability of N1-P2 amplitude (ICC = 0.889) and a significant positive correlation (Spearman's $\rho = 0.874$, $p < 0.001$) between sessions 1 and 2.

3.1.2 RMS amplitude

There was a significant main effect of CF ($F(3,33) = 19.5$, $p < .001$), stimulus level ($F(2,20) = 5.50$, $p < .012$) and AM rate change ($F(2,22) = 13.9$, $p < .001$) on RMS amplitude.

Post-hoc comparisons indicated significant differences in RMS amplitude between CFs 626 Hz and 3313 Hz ($p = 0.016$), 626 Hz and 5688 Hz ($p = < .001$) and 1438 Hz and 5688 Hz ($p = < .001$), AM rate changes 12-40 Hz and 12-114 Hz ($p = < .001$) and 12-80 Hz and 12-114 Hz ($p = 0.037$), and stimulus levels 55 dB SPL and 75 dB SPL ($p = 0.035$). There was also a significant effect of presentation rate 0.5 Hz ($M = 3.39$; $SD = 4.00$) and 0.8 Hz ($M = -0.36$; $SD = 3.77$); $t(11) = 2.79$, $p = 0.018$, and direction of AM change from upward (12-114 Hz) ($M = 3.39$; $SD = 4.00$) to downward (114-12 Hz) ($M = 0.038$; $SD = 4.80$); $t(11) = 2.77$, $p = 0.018$. There was good test-retest reliability of RMS amplitude (ICC = 0.877) and a significant positive correlation (Spearman's $\rho = 0.797$, $p = .0019$) between sessions 1 and 2.

3.2 Experiment 2

As testing is still ongoing (currently N = 4), statistical analyses were not performed. However, as shown in Figure 4, the general trend indicates the ACC N1-P2 amplitude diminishes as CF increases and AM rate change decreases. The ACC appears to be recordable using the clinical system at all AM rate changes tested at CF 626 Hz and 1438 Hz, although responses to the 12-40 Hz AM rate change are weak. At CF 3313 Hz it does not look possible to record the ACC for smaller AM rate change differences, despite each participant reporting that they could clearly hear the change.

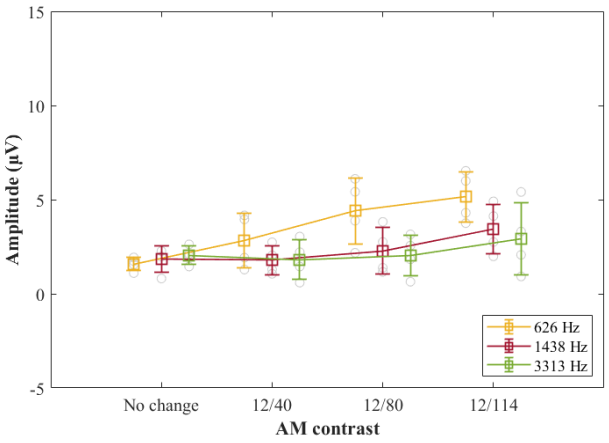

Figure 4. Mean N1–P2 amplitude (μV) as a function of AM rate change at three carrier frequencies (626 Hz, 1438 Hz, 3313 Hz). Error bars show ±1 SEM.

4. DISCUSSION

The primary aim of this study was to evaluate whether the ACC to AM rate changes can be reliably recorded using a clinical evoked-potential system (Eclipse, Interacoustics), as a step toward developing an objective measure for verifying CI fitting and informing mapping. In addition, we aimed to investigate how various stimulus parameters—including CF, AM rate change, presentation level, presentation rate, and direction of change—affect the amplitude (N1-P2 and rms) of the ACC response. In normal-hearing listeners, we found that the ACC could be robustly recorded using this system, with good test-retest reliability. These findings support the feasibility of adapting ACC protocols for clinical use, using equipment that is already available in many audiology departments.

The results of Experiment 1 suggest that several stimulus parameters significantly influenced ACC amplitude. ACCs were largest at lower CFs, with the strongest responses typically observed at 626 Hz. This is consistent with previous findings suggesting greater cortical sensitivity to lower-frequency modulation cues [20, 30, 31]. As expected, larger AM rate changes also evoked greater ACC amplitudes (N1-P2 and RMS), reflecting the increased neural response to more salient acoustic changes. This pattern aligns with prior work using research-grade EEG recording systems [15, 30]

The direction and speed of stimulus presentation also impacted response amplitude. Upward changes in AM rate (12–114 Hz) elicited stronger ACCs than downward changes (114–12 Hz), potentially reflecting perceptual asymmetries or differences in adaptation across modulation frequencies [31-33]. Faster presentation rates reduced ACC amplitude, likely due to reduced neural recovery time or attentional effects [14, 24, 34].

Notably, presentation level had only a modest and inconsistent influence on the ACC, which may be advantageous in clinical contexts where stimulus level cannot always be precisely controlled.

Importantly, ACCs were recordable across most test conditions, and test-retest reliability was high, indicating that the response is stable over time. This is a key requirement for clinical implementation, where repeatable and interpretable results are essential for guiding decisions. The ability to record robust ACCs with the Eclipse system using stimuli modelled after previous research protocols demonstrates the potential for clinical translation without the need for complex analysis pipelines or specialised hardware.

The preliminary data from Experiment 2 suggest that ACC responses are weaker at higher CFs, especially when paired with smaller AM rate changes. This trend is notable, as it may limit the clinical utility of the ACC in assessing the perception of fine-grained modulation changes in higher-frequency channels. However, responses at 626 Hz and 1438 Hz were reliably present even at smaller AM rate changes, suggesting that lower-frequency channels may provide a more consistent window into temporal processing abilities. Further work is needed to determine whether these trends hold in CI users and how best to adapt parameters for individual implant configurations.

Together, these findings suggest that the ACC has promise as a clinically feasible, objective measure to support CI programming. Unlike eCAPs, which provide only an estimate of neural excitability, the ACC reflects discrimination at the cortical level via the detection of a change in an acoustic stimulus, and it may offer more direct insight into a CI user's ability to access critical AM cues that are so important for perceiving distinctive speech features. This is especially relevant in cases where behavioural testing is not possible, such as in young children or adults with additional needs. While ACCs have been widely studied in research contexts, our study contributes to the growing evidence that they can be measured using accessible clinical systems and under test conditions that align with practical constraints.

There are several limitations to this study. First, the experiments were conducted in normal-hearing listeners, and while this allowed for controlled testing of stimulus parameters, recordings in CI users are needed to fully assess clinical applicability. Second, data collection for Experiment 2 is ongoing and currently does not allow for statistical analysis, though observed trends provide useful guidance for ongoing work. Finally, although the ACC is promising as an objective measure of auditory discrimination, further research is required to understand how it relates to real-world speech perception and functional benefit in CI users.

In the next phase of this work, we are recording ACCs in CI listeners using the Eclipse system, focusing on optimising AM rate changes and exploring the extent to which these responses may support objective verification of CI fitting. So far, we have collected data from a single CI user under two conditions, both using a 1438-Hz carrier that was amplitude-modulated at 25 Hz before switching to 80 Hz at 550 ms. First, we used the participant's clinical MAP, but with all audibility and noise reduction features disabled to ensure the processor did not alter the stimulus. Second, we set the parameters below the participant's hearing thresholds (confirmed as inaudible), enabling us to observe the CI-related artefact in the EEG without a neural response. The recording protocol extended to 900 ms, providing a 350-ms window after the modulation change (25 Hz to 80 Hz) to capture ACC. We leveraged the second (inaudible) condition to locate and characterise the CI-related artefact in the EEG. The data were first low-pass filtered below 25 Hz, then we applied a frequency-domain algorithm that detects and suppresses narrowband peaks (e.g., residual CI noise) by replacing them with the surrounding baseline in the frequency domain (calculated

via FFT). This procedure preserves the low-frequency cortical responses while minimising contamination from artefact.

If validated in this population, the ACC may offer a valuable addition to current programming tools, providing clinicians with a neurophysiological measure of auditory discrimination that is both practical and clinically meaningful.

5. ACKNOWLEDGMENTS

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