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Original Article

The Effect of Intensity on the Speech Evoked Auditory Late Latency Response in Normal Hearing Individuals

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OBJECTIVE: Among the stimulus factors, the influence of presentation level is less studied in normal-hearing individuals when using speech stimuli withvarious presentation levels for the auditory late latency response (ALLR). Hence, the present study aimed to explore the Latency-Intensity (L-I) function, i.e., how the latency and amplitude change as a function of intensity using speech stimuli.

MATERIALS and METHODS: Speech-evoked ALLR was obtained from 15 normal-hearing individuals. The syllable/ta/ was used to record ALLR with an intensity of 30, 50, 70, and 90 dBSPL. Electroencephalography (EEG) from five channels was recorded and analyzed offline.

RESULTS: The overall results revealed that there is an influence of intensity on P1 and N1 latencies in a nonlinear fashion. The latency change is consistent at lower intensities than at moderate and high intensities. The amplitude changes did not reach significance, though a decrease with a reduction in intensity was obvious.

CONCLUSION: There is a significant effect of intensity on the latency and amplitude of ALLR in speech stimulus. However, this effect may vary for different speech stimuli.

KEYWORDS: Auditory late latency response, speech-evoked ALLR, latency-intensity function

INTRODUCTION

Auditory-evoked potentials (AEPs) are bioelectrical signals that are time locked to a particular event such as sound. AEPs can be recorded at various stages along this auditory pathway and have subsequently been divided into short, middle, and long latency-evoked potentials, depending upon the delay between the presentation of the stimulus and the resulting electrical signals. Cortical AEPs (CAEPs) are those potentials that are called long latency responses (LLRs) and include two broad classes of potentials, namely "obligatory" and "endogenous," where the former is a transient response to the stimulus and the latter, driven by a cognitive process of an individual, is also called a cognitive potential. Obligatory LLRs consist of a series of positive and negative peaks, namely P1-N1-P2-N2, typically observed at latencies of 60–80 ms, 90–100 ms, 100–160 ms, and 180–200 ms, respectively. While P1, N1, and P2 are predominantly exogenous potentials, N2 is not truly an exogenous potential as it is affected by intrinsic factors such as attention and sleep [1].

Each peak of the CAEP waveform appears to originate from multiple neural generators [2]. These generators are situated in the primary and secondary auditory cortices, including Herschel's gyrus, the superior temporal lobe, and the planumtemporale [3]. It was reported that late thalamic projections into the auditory cortex are the generators for the P1 potential [4]. It was also reported that the lateral frontal supra temporal auditory cortex and the nonspecific polysensory system are the generators for the P2 potential [5]. The last LLR potential, i.e., N2, has its generation in the supra temporal cortex and nonspecificpolysensory system [6].

Auditory late latency responses are affected by many factors, including arousal and type of attention; in addition, obligatory auditory LLRs (ALLRs) are influenced by stimulus factors since they are transient responses to external stimuli. The stimulus parameters that influence CAEP characteristics include the presentation rate [5, 7], stimulus duration [8–10], stimulus level [4, 11, 12], and type of speech sound [10, 13–16] or tonal stimulus frequency [17, 18]. In a previous study [19], non-speech vs. speech stimuli and natural vs. synthetic speech stimuli were compared. The results showed that P1-N1-P2 component latencies were significantly shorter when evoked with the tonal stimulus versus speech stimuli and for natural versus synthetic speech. These findings are consistent with the notion that spectro-temporal characteristics of non-speech and speech stimuli affect the P1-N1-P2 latency and amplitude components. CAEP differences between speech stimuli are an indication of different underlying neural representations of speech sounds and suggest that the information needed to differentiate the stimuli is available to the listener. There has been increasing interest in the use of cortical potentials to investigate the neural encoding of speech [20]. However, the influence of various parameters of speech stimulus on ALLR is less studied, especially stimulus intensity.

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N1 latency for tones with lower intensity is delayed in older adults compared with in younger adults. Some authors have studied the effect of intensity by means of interfacing with a hearing aid. A study showed that speech evoked a cortical response in normal-hearing individuals with and without amplification and found no significant effect of amplification (gain) in latency or amplitude [20]. In a previous study [17], intensity function in hearing impaired elderly in aided conditionswas investigated by varying the intensity of the pure tones through a hearing aid, and it was reported that a larger amplitude at P2 only in the aided condition. Some authors have studied the effect of the stimulus level by increasing the current levels in cochlear implant recipients, such as Firszt, Chambers, Kraus, Reeder and Kim, Brown, Abbas, Etler, O'Brien [21, 22] where they presented biphasic current pulses of varying magnitude as stimuli and found a decreased latency and increased amplitude of the N1 and P2 components as the stimulus level increased. Only very few studies have reported speech stimulus level effects in normal-hearing individuals [12].

Using speech as the stimulus, the above-mentioned studies used various presentation levels and mainly focused on clinical populations. Although each study provided valuable information regarding the mechanism of cortical processing, no systematic studies have been reported to explore the effect of stimulus across intensity levels. Studying this will help us to understand the changes of ALLR components for speech stimuli with changes in stimulus levels in normal-hearing individuals; in turn the findings could be applied to several clinical conditions. Hence, the current study is an attempt to explore the CEAP changes as a function of intensity using speech stimuli.

MATERIALS and METHODS

Participants

Participants comprised 15 young, healthy adult volunteers (sevenfemale and eightmales, mean age=21.03, SD=1.62). The study was approved by the institutional ethical committee and participants were explained about the procedures and written informed consent was taken prior to the study. Participants had hearing thresholds of less than 20 dB HL across the octave frequencies 250–8000 Hz, as tested using a Grason-Stadler Inc. (GSI-61; Milford, NH, USA) audiometer; normal type A tympanograms; and presented ipsilateral and contra lateral acoustic reflexes at 500, 1000, and 2000 Hz, as tested using a Grason-Stadler Inc. (GSI-33 Version II; Milford, NH, USA) middle ear analyzer. Participants had not reported any known history of otological or neurological disease, brain injury, or poor cognitive function.

The syllable/ta/was digitally recorded using a condenser microphone using STIM 2 software (Compumedics Neuroscan; Charlotte, NC, USA) at a sampling frequency of 44,000 Hz and 16 bit resolution. The duration of stimulus was 153 ms and stimuli were presented unilaterally by Etymotic ER3 (Etymotic Research, Inc; Elk Grove Village, IL, USA) insert earphones and foam tips placed in the participants' ears. The waveform and corresponding frequency spectra of the stimulus /ta/ are shown in Figure 1.

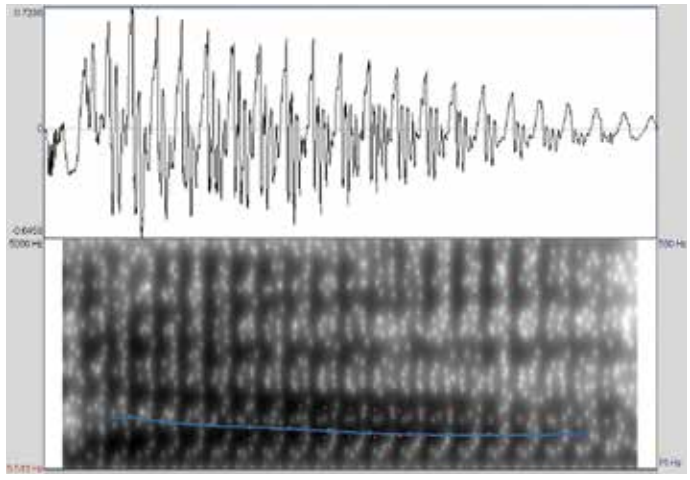

Figure 1. Top panel shows the waveform and corresponding frequency spectra in the bottom panel for the stimulus /ta/

AEP Recording

All the participants were made to recline comfortably on a reclining couch in a sound-treated room and an electrode cap (Eazycap™, Compumedics Neuroscan; Charlotte, NC, USA) was placed on the scalp. The participants were instructed to ignore the auditory stimulus and to watch a silent video. Electroencephalography (EEG) from five channels: Fz, Cz, C4, T7, and T8, were recorded, which were referenced to the right mastoid with the forehead as ground. The electrode sites maintained < 5kOhms impedance for all sites during the recording. Electro-ocular (EOG) activity was recorded with a horizontal and vertical electrode placement. Horizontal EOG was recorded from the lateral side of the outer canthus of each eye, with a bipolar EOG montage, and vertical EOG recorded with a bipolar montage placed above and below the left eye. The stimulus was played through the "SOUND" module in STIM2 software (Compumedics Neuroscan; Charlotte, NC, USA) connected to the insert receiver (Etymotic ER3 earphones). A total of 300 sweeps was presented to all participants in their left ear with an ISI of 1000 ms. Intensity was varied from 90 dB SPL to 30 dB SPL in 20 dB SPL steps. The continuous EEG was captured by the "Acquire" module Scan 4.5 software (Compumedics Neuroscan; Charlotte, NC, USA) with the analog band pass filter set between 1 and 30 Hz, and EEG was amplified with a gain of 2010× by SynAmps^2 (Compumedics Neuroscan; Charlotte, NC, USA) and sampled at a rate of 1000 Hz. All the EEG data was then offline analyzed using the "EDIT" module in Scan 4.5 software (Compumedics Neuroscan; Charlotte, NC, USA).

Eye-blink artifacts were corrected offline using the Neuroscan software. After blink correction, continuous EEG was epoched between - and 800 msec and baseline corrected for pre-stimulus duration after averaging the data. The waveform was visually analyzed. P1 was defined as a positive peak occurring in the range of 40–70 msec. N1 was the maximum negativity between 50 and 150 ms, P2 was the next positive response between 150 and 250 msec, and the N2 response was between 250 and 360 msec. Horizontal and vertical eye movements were monitored with electrodes located at the horizontal and vertical sites of both eyes.

A previous study reported that the Cz site has a higher amplitude and lower latency for CAEPs [16]. Hence, the current study also considered Cz for the statistical analysis of L-I function. All the statistical analyses were performed using Statistical Package for the Social Sciences version 16 (SPSS Inc.; Chicago, IL, USA). A one way repeated measures of ANOVA was performed and the changes in latency and amplitude were regarded as significant if the p value was less than 0.05.

RESULTS

Effect of Intensity on the Individual Peaks

Table 1 and Table 2 show the mean and standard deviations of the latencies and amplitudes at Cz for the various intensity levels. Figure 3 shows the latency changes across various intensity levels. The latency and amplitude change for each peak at various intensity levels was nonlinear, except for P1 and N1.

The results of statistical analysis revealed a significant difference between the intensities, ranging from 30dBHL to 90dBHL ($F(3, 69)=82.74$, $p<0.05$, $\eta^2=0.78$) for P1. Furthermore, a pairwise comparison with Bonferroni correction was performed, and the results revealed statically significant prolongation between all the intensities. N1 latencies too showed significance ($F(3, 69)=135.98$, $p<0.05$, $\eta^2=0.85$),and statistical significance was not seen only at 70 dBSPL and 90dBSPL. P2 latencies showed a statistical significance ($F(3, 69)=17.69$, $p<0.05$, $\eta^2=0.43$) across intensities; however, the pairwise comparison revealed significance only between 30 and 50 dBSPL. Statistical analysis revealed no significance for amplitude at P1 ($F(3, 69)=0.85$, $p>0.05$ $\eta^2=0.04$). However, N1 ($F(3, 69)=12.35$, $p>0.05$, $\eta^2=0.35$) and P2 ($F(3, 27)=12.15$, $p>0.05$, $\eta^2=0.35$) showed a significance for amplitude with intensity change,but the pairwise comparison revealed a significance only between 30 and 50 dBSPL.

DISCUSSION

The concept of amplitude and latency changes with intensity has been investigated in a number of studies [16, 23-26]. Research using tonal stimulus such as clickshas shown a linear increase in latency and reduction in amplitude of N1-P2 with decreasing stimulus intensity, and this effect is predominant in the range of 0 to 40 dB SL and saturated thereafter [16, 23]. Several other researchers have used tonal stimuli and also demonstrated similar findings. A previous study [24] revealed that a 1 kHz pure tone saturation was reached around 60–70 dB above threshold. Evidence from studies using MEG is also in agreement with the above findings [25, 26].

However, there is little known regarding the dynamics of speech-evoked ALLR, thus the current study employed a wide range of intensities on a reasonably large number of participants to explore this aspect. The current study showed a clear change in latency as a function of intensity with respect to all the measured components (P1-N1-P2) (Figure 2). Close observation revealed that the largest change was observed for P1 among all the ALLR components (60 msec), followed by N1 (42 msec), and P2 (37 msec). The amplitude of P1 was variable among participants and also intensity, thus it did not result in statistically significant differences. The grand mean average waveform clearly shows that the amplitude of P1 was comparable across

Table 1. The mean latency and standard deviations of P1, N1, P2, and N2 across intensities

	P1 lat		N1 lat		P2 lat	
Intensity (dB SPL)	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
30	80.00	9.736	143.42	16.368	232.00	17.490
50	70.50	6.627	109.83	8.676	207.42	21.274
70	48.67	13.130	97.08	7.569	191.92	19.244
90	38.42	10.677	100.58	10.065	196.83	29.090

SPL: sound pressure level; lat: latency

Table 2. The mean amplitude and standard deviations of P1, N1, P2, and N2 across intensities

	P1 amp		N1 amp		P2 amp	
Intensity (dB SPL)	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
30	0.84	0.63	-2.33	0.642	1.544	0.31
50	0.68	0.82	-3.57	0.84	3.07	0.95
70	0.75	0.91	-3.8	0.88	2.28	1.01
90	0.52	0.51	-4.26	1.97	2.46	1.32

SPL: sound pressure level; amp: amplitude

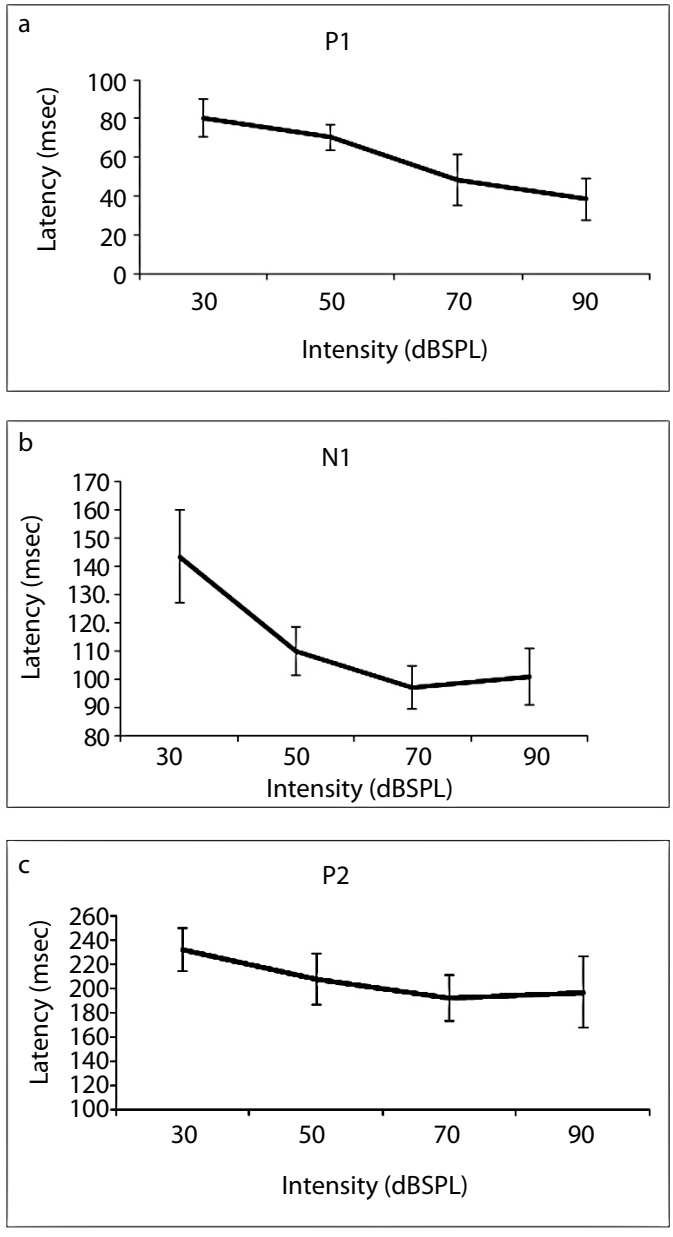

Figure 2. a-c. The figure shows the latency-intensity function for the P1 (a), N1 (b), and P2 (c) components

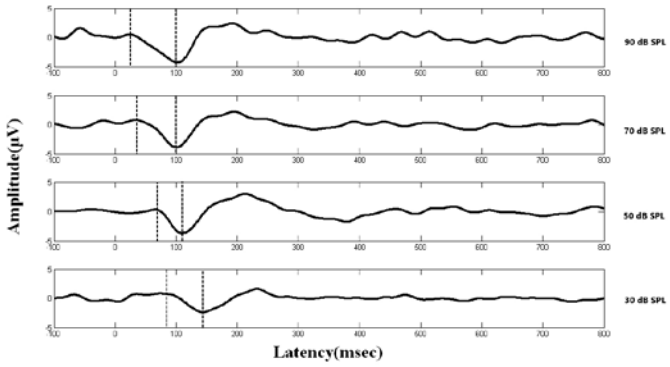

Figure 3. The figure shows the grand average waveform of all participants across intensities (90, 70, 50, and 30 dB SPL) at the Cz electrode site

all the intensities tested. The effect of intensity on auditory N1 latency was found to be nonlinear up to 70 dBSPL and saturatedthereafter. A greater change was seen at lower intensities than higher intensities. A 20 dB reduction from 90 dBSPL led to a very negligible 2 msec prolongation; however, a similar reduction from 50 to 30 dBSPL resulted in an approximately 30 msec prolongation. The amplitude characteristics of N1 mimicked the latency measure, except for the negative correlation. It can be considered that the dynamic range of P2 latency is larger than N1, as evident from the grand mean average. On the other hand, P2 amplitude grew non-monotonically, with the largest amplitude observed for 50 dBSPL, compared to both high and low intensity sound. P2 showed a broadening of the peak at higher intensities, while at the lowest intensity level, the peak was sharp.

First, the findings of the current study are in overall agreement with the previous investigations, which report a nonlinear change in latency with respect to intensity. Several studies have reported a reduction in latency and an increase in amplitude with an increase in stimulus level for P1-N1-P2 in adults, with the extent of the reduction being comparable with the current study [11, 23, 24, 27]. However, contradictory findings were reported in a previous study [12], where negligible changes were observed in P1 latency for /ta/ with the change in intensity level butan increase in amplitude was observed with increased stimulus level in infants. This resistance in change in latency in infants might be due to their immature auditory system and higher centers [28, 29].

Second, many studies have reported that CAEPs saturates at moderate to high intensities predominantly using non-speech stimuli [11, 16, 24]. Similarly, higher intensity levels showed negligible changes at N1 and P2 latency. The amplitude changes for N1 and P2 were nonlinear, even in the current study where above moderate intensities (50 dB SPL) produced negligible amplitude growth. The reasons for the differences in latency, amplitude, and morphology of ALLR can be several. These changes can be due to the nonlinearity of the auditory system, stimulus parameters, and diversity in cortical activation due to the stimulus. The linearity in amplitude for the auditory pathway is reported to be different for the auditory nerve, which shows the highest dynamic range compared to the brain stem and cortex [30, 31].

Furthermore, N1-P2 are "obligatory" ERPs, where the latency and amplitude are largely determined by the stimulus parameters, such as frequency, duration, and timing of the primary auditory pathway [11, 16, 32-35] where the high, mid, and low frequency stimuli follow different neural activation at different intensity levels in the auditory pathway. Speech stimuli vary in amplitude, duration, and frequency, which might cause a different activation of the auditory pathway compared to frequency and duration specific stimuli. In addition, the voice onset time and burst duration varies across speech stimuli, which might also cause a change in latency and amplitude with respect to non-speech stimuli [12].

Third, it is possible to postulate from the findings of the present study that each component of ALLR could be influenced differentially by intensity. In the current study, the extent of latency change could be arranged in descending order from P1 to P2. ALLR components are supposedly of distinct origin in the cortex [2, 6, 15], and as ERP measures the phase canceled average activity of the cortex. It is possible to assume that each component could be individually influenced by the change in intensity which is failed to reflect in the averaged responses. For example, an increase in amplitude of N1 could possibly cancel nearby peaks (P1-P2) and cause a reduced amplitude, or the detection of the sound may be controlled by a specific area while the processing of the signal involves several other areas. Furthermore, speech stimulus introduces larger temporal and spectral modulation in the steady-state portion when compared to tonal stimulus and could influence the number of components arising from the cortex. This notion needs further empirical support using various stimulus manipulations.

Previously reported investigations targeting the same research question have mostly focused on non-speech stimulus. However, when we changed the stimulus type to speech sounds (consonants,vowels), the influence of intensity was slightly different in that the latency and amplitude were altered when compared to tonal and particularly click stimuli [12, 16, 23]. Thus, these findings suggest that corticalevoked potential studies employing speech stimulus should take adequate caution in terms of the intensity used. It might be possible that speech-evoked CEAPs could saturate and show no variation across experimental manipulation, particularly at moderate- to high-intensity stimulus. The results of the present study could be greatly influenced by ISI used; the present study used longer ISI (1000 msec), which allow neurons to recover adequately and greatly influence morphology [36-38].

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of School of Allied Health Science, Manipal University.

Informed Consent: Written informed consent was obtained from all the participants who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - H.P., A.A.; Design - H.P., A.A.; Data Collection and/or Processing - H.P., A.A.; Analysis and/or Interpretation - H.P., A.A.; Literature Search - B.R., K.Y.; Writing Manuscript - H.P., A.A., B.R., K.Y.; Critical Review - B.R., K.Y.

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