Effects of Amplification on Neural Phase Locking, Amplitude, and Latency to a Speech Syllable

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Objective: Older adults often have trouble adjusting to hearing aids when they start wearing them for the first time. Probe microphone measurements verify appropriate levels of amplification up to the tympanic membrane. Little is known, however, about the effects of amplification on auditory-evoked responses to speech stimuli during initial hearing aid use. The present study assesses the effects of amplification on neural encoding of a speech signal in older adults using hearing aids for the first time. It was hypothesized that amplification results in improved stimulus encoding (higher amplitudes, improved phase locking, and earlier latencies), with greater effects for the regions of the signal that are less audible.

Design: Thirty-seven adults, aged 60 to 85 years with mild to severe sensorineural hearing loss and no prior hearing aid use, were bilaterally fit with Widex Dream 440 receiver-in-the-ear hearing aids. Probe microphone measures were used to adjust the gain of the hearing aids and verify the fitting. Unaided and aided frequency-following responses and cortical auditory-evoked potentials to the stimulus /ga/ were recorded in sound field over the course of 2 days for three conditions: 65 dB SPL and 80 dB SPL in quiet, and 80 dB SPL in six-talker babble (+10 signal to noise ratio).

Results: Responses from midbrain were analyzed in the time regions corresponding to the consonant transition (18 to 68 ms) and the steady state vowel (68 to 170 ms). Generally, amplification increased phase locking and amplitude and decreased latency for the region and presentation conditions that had lower stimulus amplitudes—the transition region and 65 dB SPL level. Responses from cortex showed decreased latency for P1, but an unexpected decrease in N1 amplitude. Previous studies have demonstrated an exaggerated cortical representation of speech in older adults compared to younger adults, possibly because of an increase in neural resources necessary to encode the signal. Therefore, a decrease in N1 amplitude with amplification and with increased presentation level may suggest that amplification decreases the neural resources necessary for cortical encoding.

Conclusion: Increased phase locking and amplitude and decreased latency in midbrain suggest that amplification may improve neural representation of the speech signal in new hearing aid users. The improvement with amplification was also found in cortex, and, in particular, decreased P1 latencies and lower N1 amplitudes may indicate greater neural efficiency. Further investigations will evaluate changes in subcortical and cortical responses during the first 6 months of hearing aid use.

Key words: Amplification, Cortical auditory-evoked potential, Frequency-following response, Hearing loss, Older adults, Phase locking

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INTRODUCTION

The primary treatment option for most people with mild to moderate sensorineural hearing loss is the use of hearing aids. However, the benefit received from hearing aids varies greatly from person to person, regardless of the degree and configuration of the hearing loss (Kochkin 2012). Hearing aid benefit may be reduced by age- and hearing-related central changes that affect the quality of the speech signal reaching the central auditory nervous system. Current amplification strategies do not compensate for auditory temporal processing deficits that have been demonstrated in behavioral (Fitzgibbons et al. 2006; Pichora-Fuller et al. 2007; Grose & Mamo 2010) and electrophysiological studies of aging (Tremblay et al. 2003; Harris et al. 2010; Presacco et al. 2015, 2016b). Hearing impairment may also lead to downstream changes in central auditory processing, including changes in tonotopicity (Willott 1991; Thai-Van et al. 2010) and in the balance of excitatory and inhibitory neurotransmission (Mossop et al. 2000; Felix & Portfors 2007; Dong et al. 2009). Changes in neural encoding associated with aging and hearing loss may affect speech perception. Thus, efforts to examine hearing aid benefit should consider taking into account amplification effects on speech encoding beyond the cochlea. The current method of hearing aid verification, probe microphone measurement, does not address effects of amplification beyond the tympanic membrane. An understanding of amplification effects at higher levels of the auditory system (both auditory cortex and midbrain) may inform amplification algorithms or strategies and, thus, improve the success of hearing aid fittings.

Few studies to date have assessed the effects of amplification on auditory-evoked responses. Billings et al. (2007) investigated the effects of amplification on cortical auditory-evoked potentials (CAEPs) of young normal-hearing listeners to tonal stimuli. Normal-hearing listeners were used to control for the effects of hearing loss on cortical responses. These listeners were fit with behind-the-ear hearing aids to the right ear, and probe microphone measurements were used to ensure that the hearing aids provided 20 dB of gain to input signals of varying intensities. Comparisons of intensity growth functions for both conditions revealed no differences in CAEP morphology for aided and unaided conditions. Further investigation into individual in-the-canal intensity measurements found that listeners with more favorable signal to noise ratios (SNRs) had larger CAEP amplitudes and shorter latencies than listeners with less favorable SNRs, suggesting that the SNR may have a greater effect on cortical potentials than amplitude only. A follow-up study investigated differences in aided and unaided CAEP intensity growth functions when hearing aid gain was 0, 10, 20, and 30 dB (Billings et al. 2011). Intensity of the input tonal signal was fixed at 40 dB SPL for aided conditions and was 40, 50, 60, and 70 dB SPL for unaided conditions to ensure equal in-the-canal intensities for aided and unaided conditions. Although in-the-canal intensities were equal for the aided and unaided conditions, aided responses had prolonged latencies and reduced amplitudes when compared to unaided responses, possibly because of lower in-the-canal SNRs in aided compared to unaided conditions as a result of the noise produced by the

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hearing aid. The Billings et al. (2011) study examined effects of hearing aid gain on CAEPs in young adults with normal hearing. In contrast, Van Dun et al. (2016) compared effects of amplification, audibility, and SNR on CAEPs of young normal-hearing listeners and older listeners with hearing loss to the speech stimuli /m/, /g/, and /t/ presented in sound field. Unaided stimulus levels were 55, 65, and 75 dB SPL, and the aided stimulus level was 55 dB SPL. Amplification resulted in increased CAEP amplitudes for listeners with hearing loss, but no effects were seen in normal-hearing listeners. Furthermore, in the listeners with hearing loss, CAEP amplitudes were positively correlated with audibility but were negatively correlated with SNR. The lower SNRs occurred for aided versus the unaided conditions, but the internal noise of the hearing aid was likely inaudible to the hearing-impaired listeners. Therefore, audibility is likely the most significant factor in the listeners with hearing loss. These studies indicate the importance of recruiting listeners with hearing loss when investigating the viability of using evoked potentials to verify amplification effects.

Evoked potentials may also be used to verify audibility in infants and other difficult-to-test populations and to evaluate the effects of hearing aid technology. Glista et al. (2012) investigated effects of hearing aid frequency compression technology on CAEPs to tonal stimuli in children with moderately severe high-frequency sensorineural hearing loss (SNHL). Frequency compression compresses high-frequency signals into a narrower bandwidth at lower frequencies with better hearing thresholds to increase audibility. They found that P1–N1–P2 responses to high-frequency tones were present when frequency compression was activated and were absent when it was turned off. These results verified that frequency compression improves detection of high-frequency sounds in the auditory cortex and that cortical-evoked potentials are sensitive to changes in signal processing of hearing aids.

Feasibility studies have also been conducted to investigate use of the frequency-following response (FFR) with hearing aids. The FFR is an evoked potential to periodic stimuli arising largely from brainstem and midbrain for modulation frequencies associated with the fundamental frequency of the human voice (Smith et al. 1975; Chandrasekaran & Kraus 2010) with possible cortical contributions (Coffey et al. 2016). As the FFR waveform closely resembles the stimulus waveform (Greenberg 1980; Galbraith et al. 1995), the FFR can be used to assess precision of midbrain encoding of temporal and spectral features of speech (Skoe & Kraus 2010). Evaluation of amplification effects on the FFR may provide insight into the perceptual changes that listeners with hearing loss experience when using hearing aids. Bellier et al. (2015) recorded aided and unaided FFR responses to the speech syllable /ba/ in four listeners with normal hearing. The signal was presented at 80 dB SPL via insert earphones (unaided), through wireless transmission to the hearing aids at two different gain levels and to three "muted" conditions (microphones turned off), resulting in a total of six listening conditions. Clear responses were collected for the insert earphone condition, and both hearing aid conditions and no responses were present in the muted conditions, verifying the feasibility of collecting viable FFRs when the signal is presented through wireless transmission.

Amplification effects on the FFR have also been investigated in individuals with hearing loss. In a 75-year-old individual with hearing loss, FFRs to a speech syllable /da/ presented through a speaker demonstrated changes in speech encoding in unaided versus aided conditions and with different hearing aid settings (Anderson & Kraus 2013). Easwar et al. (2015b) used direct audio input to investigate aided and unaided FFRs to a male-spoken token /susa ∫ i/ representing a wide range of frequencies in older listeners with hearing loss. Amplification resulted in increased detectability and amplitude of the response. Increasing hearing aid bandwidth to 4 kHz further increased detectability, suggesting that the FFR may be used to verify audibility and to evaluate the effects of manipulating hearing aid parameters. Taken together, these studies demonstrate the potential usefulness of the FFR for assessing hearing aid benefit on midbrain encoding of speech signals in individuals with hearing loss.

The purpose of the present study was to assess the effects of amplification on FFRs and CAEPs to a speech syllable /ga/ presented in sound field in first-time hearing aid users at input levels that approximate normal listening conditions. This study addresses the first of two aims in a larger project that investigated amplification effects on central auditory processing and plasticity changes with hearing aid use over a 6-month period. While previous studies have focused on increasing detectability of the signal, this study investigated the effects of amplification on suprathreshold speech processing. It was hypothesized that amplification results in improved encoding of the speech signal in the midbrain (higher response amplitudes, improved phase locking, and earlier latencies) because of an increase in audibility. Based on the findings of Van Dun et al. (2016) for listeners with hearing loss, it was also expected that amplification would result in higher CAEP amplitudes and decreased latencies. These hypotheses will be tested by comparing FFRs and CAEPs to a speech syllable presented at different intensity levels in aided and unaided conditions. The interacting effects of noise and amplification will also be evaluated. The evaluation of amplification effects on central processing using ecologically valid listening conditions is a first step in determining the usefulness of these measures for improving hearing aid outcomes.

MATERIALS AND METHOD

Participants

Thirty-five older adults with sensorineural hearing loss (22 females, ages 60 to 88 years, mean ± SD 73.97 ± 5.79 years) were recruited from the Washington DC metro area through the use of flyers distributed across the University of Maryland campus, local senior living communities, and through Craigslist advertisements. Participants had hearing levels ranging from mild to severe, with pure-tone averages ≥25 dB HL from 500 to 4000 Hz, no pure-tone thresholds ≥90 dB HL at any one frequency, no air-bone gaps of 15 dB HL or greater at two or more adjacent frequencies, and no interaural asymmetries of 15 dB HL or greater at two or more frequencies. Figure 1 shows individual hearing thresholds in right and left ears and for average thresholds at each frequency. All subjects had normal click-evoked auditory brainstem response latencies for age and hearing loss (wave V < 6.8 ms; Otto & McCandless 1982), measured by a 100 μs click stimulus presented at 80 dB SPL (peak equivalent) at a rate of 21.1 Hz. In one participant, data were not obtained for the noise condition because of equipment difficulties.

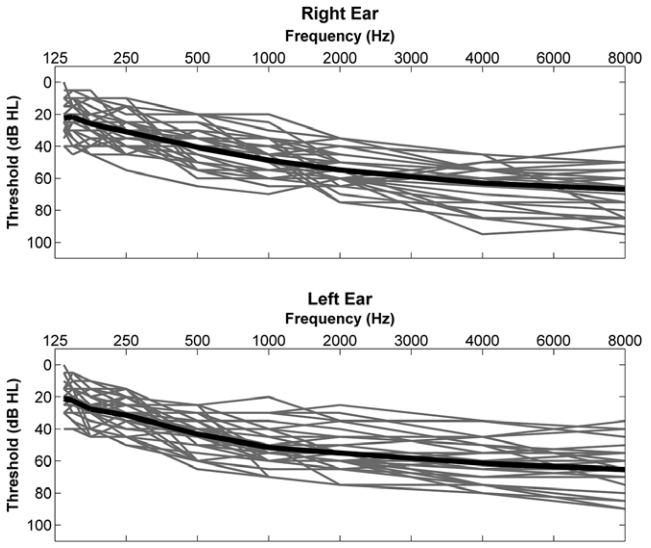

Fig. 1. Individual pure-tone air-conduction thresholds for participants (n = 35) from 125 to 8000 Hz for right and left ears, shown in gray. The solid black line indicates group average pure-tone thresholds.

Participants had normal IQs (≥85) as evaluated using the Wechsler Abbreviated Scale of Intelligence (mean ± SD 113.05 ± 14.76; Zhu & Garcia 1999) and were screened for dementia using a criterion score of 22/30 on the Montreal Cognitive Assessment (mean ± SD 25.74 ± 2.27; Nasreddine et al. 2005). All participants were native speakers of English, had no history of neurological disorders, and had no previous experience with hearing aid use. As music training may have an effect on subcortical auditory processing (Bidelman & Krishnan 2010; Parbery-Clark et al. 2012), professional musicians were excluded from the study. All procedures were approved by the Institutional Review Board of the University of Maryland. Participants provided informed consent and were compensated for their time.

Hearing Aid Fitting

Each participant was fit bilaterally with Widex Dream 440 receiver-in-the-ear hearing aids with size M receivers with open domes (individual thresholds for 250 to 500 Hz < 30 dB HL) or tulip domes (individual thresholds for 250 to 500 Hz ≥ 30 dB HL). The Widex Dream 440 Fusion hearing aids accommodate hearing losses up to 85 dB HL from 125 to 8000 Hz when coupled with M receivers. Although there is greater variability in the amount of low-frequency gain provided by the hearing aids when open domes are utilized, their use was necessary in terms of patient comfort and compliance. As this study was also part of a longer project to assess central plasticity associated with hearing aid use, it was imperative that the patients be comfortable enough with the hearing aids to wear them 8 hours per day over the course of 6 months. Although the hearing aids can be programmed with five manual programs, only one automatic program was used for the purposes of this study. This program had an extended input dynamic range of 113 dB SPL, 15 frequency channels, wide dynamic range compression, directional microphones, and noise reduction technology. The hearing aids were linked using ear-to-ear communication technology for compression, speech enhancer, and feedback cancellation.

Individual real-ear measurements were performed to verify the fitting. Real-ear-to-coupler differences were first obtained and then the hearing aids were adjusted to match NAL-NL2 prescriptive targets for International Speech Test Signal stimuli (Holube et al. 2010) presented at 55, 65, and 75 dB SPL. Each participant received a pair of hearing aids that were programmed to match his or her NAL-NL2 prescriptive targets based on each individual's audiogram. Table 1 reports the group average–aided SPLs obtained from real-ear testing and the differences between actual and target SPLs for 250 to 4000 Hz at 55, 65, and 75 dB SPL in each ear. A goodness of fit test was performed to determine how well measured outputs matched expected outputs based on NAL-NL2 prescriptive targets. As shown in Table 1, gain measures fit well to expected values, with the exception of 4000 Hz. The average thresholds at 4000 Hz were in the moderately severe range, and fitting to the target for this frequency was not possible without causing feedback or patient discomfort when listening to high-frequency sounds. Maximum power output measurements were performed to ensure that the hearing aids were not uncomfortably loud.

Hearing Aid Audibility

As the use of open domes results in variable gain values for low-frequency inputs, further investigation into the audibility of the signal was done using open and closed domes. After reviewing audiograms of each participant, we determined that each hearing loss fell into one of three configurations: gradually sloping mild to moderate SNHL, gradually sloping mild to severe SNHL, and mild sharply sloping to severe SNHL (Fig. 1). This information was used in the collection of KEMAR measurements to ensure the hearing aids were providing adequate audibility for the input signals used in the protocol. Hearing aids were programmed for each of the three categories of hearing losses described above, and experimental stimuli were presented to KEMAR at ear level at a distance of 2 m from the loudspeaker at 0° azimuth for all presentation levels, an identical collection paradigm to that used in the study. In-ear intensity levels were measured for frequencies from 125 to 8000 Hz. Table 2 reports the degree to which the aided in-ear levels of the /ga/ syllable exceed audiometric thresholds for each frequency for the 65 dB SPL, 80 dB SPL in quiet, and 80 dB SPL in noise presentation conditions for open and tulip (closed) dome fittings for the three types of sensorineural hearing losses. Positive values indicate that the aided levels exceed audiometric threshold, verifying adequate audibility through 2000 Hz for most conditions for both the tulip dome and open dome fitting configurations. Table 3 provides unaided sensation levels above thresholds as a reference for the three hearing loss groups.

Electrophysiology

Stimuli and Recording • A 170-ms speech syllable /ga/ (Fig. 2) synthesized with a Klatt-based synthesizer (Boersma & Weenink 2009) at 20 kHz was the chosen stimulus. The stimulus was characterized by a 10-ms onset burst followed by a 50-ms consonant–vowel transition and a steady state vowel region from 60 to 170 ms. Voicing was constant for the duration of the stimulus with a fundamental frequency (F_0) of 100 Hz. The transition region was characterized by rapidly changing formants: the first formant rose from 400 to 720 Hz, the second formant fell from 2480 to 1240 Hz, and the third formant fell from 2580 to 2500 Hz; all three formants stabilized for the

TABLE 1. Real-ear measurement values

		250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz	
dB SPL	Ear	Output	Target Diff.	Output	Target Diff.	Output	Target Diff.	Output	Target Diff.	Output	Target Diff.
55	Right	48.17 (2.47)	0.28 (4.26)	49.97 (4.49)	−1.59 (5.26)	55.48 (5.32)	1.83 (5.97)	62.79 (6.58)	−2.90 (5.16)	58.76 (5.47)	−8.97 (7.82)
	Left	48.21 (3.52)	0.07 (4.71)	50.26 (4.92)	−2.10 (4.59)	55.96 (8.19)	0.64 (5.72)	63.75 (8.17)	−3.07 (4.74)	60.52 (7.45)	−9.32 (7.65)
65	Right	56.50 (2.06)	0.89 (1.62)	58.89 (4.00)	0.29 (4.67)	63.21 (5.12)	3.86 (4.62)	69.39 (6.70)	0.61 (3.54)	65.86 (5.92)	−7.29 (5.27)
	Left	56.96 (3.57)	1.21 (3.99)	59.41 (3.82)	0.11 (3.28)	62.63 (6.25)	2.25 (4.04)	69.46 (7.10)	−1.89 (4.04)	68.11 (7.80)	−4.93 (7.27)
75	Right	55.77 (3.69)	−9.18 (3.80)	63 (3.95)	−4.18 (3.98)	70.55 (5.58)	5.95 (4.87)	77.59 (7.20)	4.05 (4.28)	74.23 (7.19)	−2.05 (6.02)
	Left	55.71 (4.04)	−9.38 (3.96)	62.10 (4.98)	−4.95 (4.93)	70.05 (6.23)	6.00 (5.20)	77.71 (6.51)	3.62 (4.62)	76.71 (7.70)	0.24 (7.75)

Mean output levels and mean differences from NAL-N2 targets in dB SPL are displayed along with standard deviations for the right and left ears at 55, 65, and 75 dB SPL input levels. The levels are generally within a few decibels of the target values except for the 55 and 65 dB levels at 4000 Hz, at which it was not possible to provide sufficient gain to meet the targets. The output values in regular font meet the goodness of fit test ($F > 6.0$, $p < 0.02$), and values in lighter font do not meet the goodness of fit test ($F < 4.0$, $p > 0.05$).

steady state region of the syllable. The fourth through sixth formants remained constant over the entire duration of the syllable at 3300, 3750, and 4900 Hz, respectively. This stimulus was chosen to investigate amplification effects on audibility of the higher frequency information present in the transition region of the syllable. In addition to frequency differences, we also note that the stimulus regions differ in relative power. A root mean square (RMS) power calculation revealed values of 0.08 V^2 for the transition region and 0.10 V^2 for the steady state region. The syllable's waveform and its spectral energy are represented in Figure 2.

All testing was conducted in a sound-treated, electrically shielded booth with the lights off to reduce electrical interference. The /ga/ stimulus was presented through a speaker placed 2 m from the participants at 0° azimuth via Presentation software (Neurobehavioral Systems, Inc.). The stimulus was presented through sound field rather than via direct input to allow processing through hearing aid microphones and to simulate situations that were ecologically valid. The /ga/ was presented in three listening conditions: (1) 65 dB SPL in quiet; (2) 80 dB SPL in quiet; and (3) 80 dB SPL in the presence of 70 dB SPL six-talker babble (herein referred to as 80 dB SPL in noise). The six-talker babble was taken from the Words-in-Noise (WIN) sentence lists (Wilson et al. 2003) and was continually played on a 4.6 sec loop. Before recording, the /ga/ and noise stimuli were calibrated to within ±1 dB of the desired presentation level using a Larson Davis System 824 sound-level meter at ear level.

Frequency-Following Response

Recording • The /ga/ stimulus was presented with alternating polarities at a rate of 4 Hz. A standard vertical montage of five electrodes (Cz active, unlinked earlobe references) was used with all offsets <50 μV. Responses were recorded using the Biosemi ActiABR-200 acquisition system (BioSemi B.V., Amsterdam, Netherlands) with a sampling frequency of 16,384 Hz. A single run of 2300 sweeps was collected for each condition. During recording, participants were seated in an upright position so that the microphones of the hearing aids were in the same plane as the speaker at a relative angle elevation of 0°. They watched a silent movie with subtitles playing on a projector screen to promote relaxation and a state of calm wakefulness and to minimize head movement. All three conditions were recorded consecutively during one test session in both aided and unaided conditions, resulting in a total of six listening conditions per participant. Order of condition presentation was randomized.

Data Reduction • The sweeps were averaged and processed off-line using MATLAB (MathWorks, version R2011b). The time window for each sweep was −50 to 185 ms referenced to the stimulus onset. The stimulus onset in the aided conditions was adjusted by 2 ms to allow for hearing aid processing time (based on frequency-specific values for hearing aid processing delays provided by Widex USA). Responses were digitally band pass–filtered from 70 to 2000 Hz using a 4th order Butterworth filter to minimize the effects of low-frequency signals originating from cortex (Dinse et al. 1997). A criterion of ±30 μV was used for off-line artifact rejection. A final average response was created by averaging the first 2000 artifact-free sweeps of the two polarities (1000 per polarity) to minimize the influence of cochlear microphonic and stimulus artifact on the response and to maximize the envelope response (Gorga et al. 1985; Aiken &

TABLE 2. Aided sensation levels above thresholds based on one-third octave band measurements obtained from KEMAR to simulate electrophysiological recordings.

	Open Dome								Tulip Dome							
Gradually sloping mild to severe SNHL																
SPL	125	250	500	1000	2000	3000	4000	6000	8000	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	22	37	28	17	–4	–11	–27	–37	–55	21	30	33	13	5	–7	–15	–34	–55
80 dB	38	48	32	25	11	8	–2	–26	–47	35	43	44	22	16	10	–7	–35	–50
Noise	38	49	31	24	14	9	16	–14	–40	37	45	40	22	16	4	–6	–27	–51
Gradually sloping mild to moderate SNHL																		
SPL	125	250	500	1000	2000	3000	4000	6000	8000	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	32	36	18	21	8	–1	–2	–27	–49	22	37	32	20	10	1	0	–22	–49
80 dB	40	50	32	31	19	9	5	–20	–48	36	48	42	35	19	15	5	–19	–42
Noise	43	50	31	28	19	10	3	–21	–42	40	58	37	29	20	9	7	–18	–40
Mild sharply sloping to severe SNHL																		
SPL	125	250	500	1000	2000	3000	4000	6000	8000	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	31	38	33	39	19	–1	–26	–37	–57	22	36	46	48	17	–1	–25	–34	–59
80 dB	41	50	46	51	33	10	–15	–30	–53	42	50	50	30	29	11	–19	–31	–55
Noise	42	49	47	48	37	11	–16	–30	–53	32	49	58	47	35	26	–15	–31	–54

Stimuli were presented from a loudspeaker placed at 0° azimuth at ear level at 2 m distance from the mannequin. A Widex 440 dream hearing aid was programmed with three different hearing loss configurations representing the average hearing loss types in the study (gradually sloping mild to severe SNHL, gradually sloping mild to moderate SNHL, and mild sharply sloping to severe SNHL) with ear open dome or tulip dome tips. As denoted by positive values, audibility was achieved through 2000 Hz for all listening conditions and hearing aid configurations.
SNHL, sensorineural hearing loss

Picton 2008; Campbell et al. 2012). SNR in decibel was calculated using the following formula:

$$20 \times \log10 \text{ (RMS Post-Stimulus/RMS Noise)}$$

where the poststimulus period is defined as 5 to 190 ms, and the prestimulus period (noise) is defined as −40 to 0 ms. All unaided and aided responses had SNR decibel values >1.

Response Amplitude and Latency • RMS amplitude was calculated for the transition (18 to 68 ms) and steady state (68 to 170 ms) regions for each condition (aided and unaided, for a total of six conditions). An automatic peak-picking algorithm was run in MATLAB that identified the peak that was closest to the expected latency (within 2 ms), based on average latencies obtained in previous studies (Anderson et al. 2012, 2013b; Presacco et al. 2015). A trained peak picker confirmed each peak identification and made changes where appropriate. The first consistently identifiable peak of the consonant transition (≈31 ms) was used in the analysis.

Phase Locking Factor • The complex Morlet wavelets were used to decompose the signal between 80 and 800 Hz and to analyze the phase locking factor (PLF) of single trials in the time–frequency domain (Tallon-Baudry et al. 1996). The PLF evaluates the inter-trial phase consistency by extracting the phase from each of the N sweeps recorded and then averaging the N phases. The phase is extracted for each frequency bin (1 Hz) at each point in time. The normalized energy was calculated for each sweep by dividing the convolution of the complex wavelet with the signal by its absolute value: $P_i(t,f_0) = \frac{w(t,f_0) \times s(t)}{|w(t,f_0) \times s(t)|}$, leading to a complex value that describes the phase distribution at each frequency and point in time. The final PLF was represented by the modulus of the average across sweeps of this complex value, which ranges from 1 (phase-locked) to 0 (nonphase locked). The mean values for the fundamental frequency (F_0) were averaged in a 10 Hz bin across the transition (18 to 68 ms) and steady state (68 to 170 ms) regions.

Cortical Response

Recording • The /ga/ stimulus was presented at a rate of 1 Hz, and responses were recorded at a sampling frequency of 2048 Hz using a 32-channel electrode cap that incorporated a subset of the International 10–20 system (Jasper 1958), with average earlobes (A1 and A2) serving as references. A single run of 600 sweeps was collected for each condition.

Data Processing and Analyses • Responses were off-line band pass–filtered from 1 to 30 Hz with a 4th order Butterworth filter. Eye movements were removed from filtered data using a regression-based electrooculography reduction method (Romero et al. 2006; Schlögl et al. 2007). The time window for each sweep was −100 to 400 ms referenced to the stimulus onset. A final average response was extracted with the first 500 artifact-free sweeps.

Denoising Source Separation • Artifact-free data from each of the 32 channels recorded were decomposed into N signal components (where $N \leq 32$) using the denoising source separation (DSS) algorithm (Särelä & Valpola 2005; de Cheveigne & Simon 2008). The first DSS, which accounts for the highest variability in our data and, therefore, the best SNR for our ERPs, was then used for the final analysis. Amplitude was calculated for the expected time region for each of the prominent cortical peaks: P1 (35 to

TABLE 3. Unaided sensation levels above thresholds based on one-third octave band measurements obtained to stimuli presented from a loudspeaker placed at 0° azimuth at ear level at 2 m distance from the KEMAR mannequin to simulate electrophysiological recordings

Gradually sloping mild to severe SNHL									
SPL	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	20	30	21	–4	–20	–31	–27	–43	–75
80 dB	36	44	35	25	8	–5	–2	–30	–75
Gradually sloping mild to moderate SNHL									
SPL	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	22	34	20	0	–11	–21	–27	–52	–60
80 dB	35	45	35	13	–5	–16	–19	–38	–53
Mild sharply sloping to severe SNHL									
SPL	125	250	500	1000	2000	3000	4000	6000	8000
65 dB	21	35	38	30	7	–23	–55	–67	–75
80 dB	42	51	44	44	20	–8	–40	–54	–73

Values are provided for each of the three hearing loss groups (gradually sloping mild to severe SNHL, gradually sloping mild to moderate SNHL, and mild sharply sloping to severe SNHL). SNHL, sensorineural hearing loss.

75 ms), N1 (75 to 130 ms), and P2 (130 to 250 ms) in the quiet conditions (65 and 80 dB SPL) and P1 (35 to 75 ms), N1 (125 to 175 ms), and P2 (225 to 275 ms) in the noise condition. Latency was calculated for the highest peak in each of these time ranges.

Statistical Analyses

Frequency-Following Response • To test effects of amplification, individual two-way repeated-measures (RM) analysis of variances (ANOVAs) (stimulus region, two levels: transition and steady state; amplification, two levels: aided, unaided) were performed for each presentation condition (65 dB SPL, 80 dB SPL in quiet, and 80 dB SPL in noise) for the FFR PLF using SPSS version 21.0. RM ANOVAs were also performed with two within-subjects independent variables (stimulus region and amplification) for RMS and one within-subject variable (amplification) for latency for each presentation condition (65 dB SPL, 80 dB SPL in quiet, and 80 dB SPL in noise). In addition, level effects were tested with a three-way RM ANOVA (65 dB SPL in quiet versus 80 dB SPL in quiet, transition versus steady-state regions, aided versus unaided), and noise effects were tested with a three-way RM ANOVA (80 dB SPL in quiet versus 80 dB SPL in noise, transition versus steady state regions, aided versus unaided) for the PLF, RMS, and latency analyses.

Cortical • To test amplification effects on DSS amplitude and latency, individual two-way RM ANOVAs (peak, three levels: P1, N1, and P2; and amplification, two levels: aided, unaided) were performed for each presentation condition (65 dB SPL in quiet, 80 dB SPL in quiet, and 80 dB SPL in noise). In addition, three-way RM ANOVAs were used to evaluate effects of level (65 dB SPL in quiet versus 80 dB SPL in quiet; P1, N1, and P2; aided versus unaided) and noise (80 dB SPL in quiet versus 80 dB SPL in noise; P1, N1, and P2; aided versus unaided). Post hoc paired *t* tests were used to evaluate

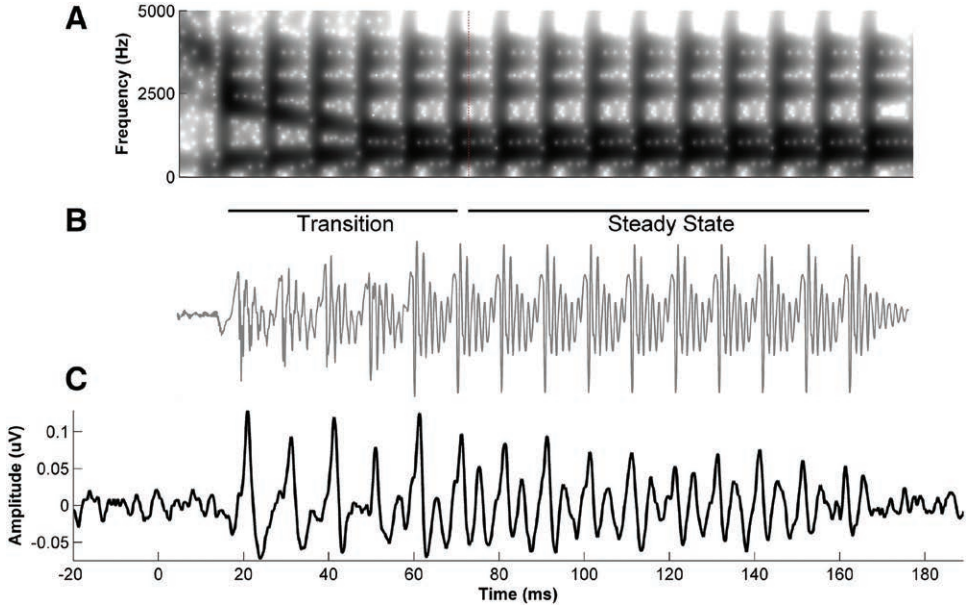

Fig. 2. A, Spectrogram of the stimulus /ga/. B, Stimulus waveform with horizontal lines marking the transition (18–68 ms) and the steady state (68–170 ms) regions. The onsets of the waveform and spectrogram are temporally aligned with the response. C, Grand average response waveform to the unaided /ga/ syllable presented at 80 dB SPL in sound field.

within-subject differences when interactions were noted for single variables (FFR RMS and latency and cortical analyses). The false discovery rate procedure (Benjamini & Hochberg 1995) was applied to control for multiple comparisons for main effects.

RESULTS

Frequency-Following Response

Table 4 provides mean and standard deviation data of PLF, RMS, and latency values and *F* statistics for main effects and interactions for the different presentation conditions. The specific details are as follows:

PLF

Amplification Effects • In response to the 65 dB SPL presentation level, there was a main effect of amplification [$F(1,34) = 6.052$, $p = 0.019$, $\eta^2 = 0.151$]. The Region × Aided interaction was not significant [$F(1,34) = 2.234$, $p = 0.144$, $\eta^2 = 0.062$] (Fig. 3). In response to 80 dB SPL in quiet and 80 dB SPL in noise, there were no main effects of amplification [80 quiet: $F(1,34) = 1.546$, $p = 0.222$, $\eta^2 = 0.044$; 80 noise: $F(1,34) = 1.074$, $p = 0.307$, $\eta^2 = 0.031$] (Figs. 4 and 5).

Level Effects • The PLF was significantly higher in response to the 80 dB SPL level compared to the 65 dB SPL level [$F(1,34) = 7645$, $p = 0.009$, $\eta^2 = 0.184$], and there was no Level × Aided interaction [$F(1,34) = 0.145$, $p = 0.706$, $\eta^2 = 0.004$] or Level × Region interaction [$F(1,34) = 2.068$ $p = 0.160$, $\eta^2 = 0.057$].

Noise Effects • The effect of noise on the PLF of the envelope was not significant [$F(1,34) = 1.544$, $p = 0.223$, $\eta^2 = 0.043$].

RMS Amplitude

Amplification Effects • In response to the 65 dB SPL level, there was a main effect of amplification [$F(1,34) = 4.343$, $p = 0.045$, $\eta^2 = 0.113$] and a significant Aided × Region interaction [$F(1,34) = 7.407$, $p = 0.010$, $\eta^2 = 0.179$], driven by a significant increase in amplitude in the transition but not the steady state regions [transition: $t(34) = 3.079$, $p = 0.004$, $\eta^2 = 0.218$; steady state: $t(34) = 0.527$, $p = 0.601$, $\eta^2 = 0.008$]. There was no main effect of amplification in response to 80 dB SPL in quiet or noise [80 dB quiet: $F(1,34) = 1.698$, $p = 0.201$, $\eta^2 = 0.048$; 80 dB noise: $F(1,34) = 1.212$, $p = 0.279$, $\eta^2 = 0.035$]. Figure 6 displays unaided and aided time domain waveforms and bar graphs representing RMS values at different presentation conditions.

Level Effects • Response amplitude was significantly higher in response to the 80 dB SPL level compared to the 65 dB SPL level [$F(1,34) = 8.342$, $p = 0.007$, $\eta^2 = 0.197$)], and there was a Level × Region interaction [$F(1,34) = 5.040$, $p = 0.031$, $\eta^2 = 0.129$)]. Amplitude was larger for the 80 dB SPL level in quiet in both regions across amplification conditions, but the effects were larger for the steady state [transition: $F(1,34) = 7.106$, $p = 0.012$, $\eta^2 = 0.173$; steady state: $F(1,34) = 10.695$, $p = 0.002$, $\eta^2 = 0.239$]. The Level × Aided interaction was not significant [$F(1,34) = 0.713$, $p = 0.404$, $\eta^2 = 0.021$].

Noise Effects • Noise had no effect on response amplitude [$F(1,33) = 0.00$, $p = 0.994$, $\eta^2 = 0.000$].

Latency

Amplification Effects • Amplification resulted in significant latency decreases at 65 dB SPL [$t(34) = 5.187$, $p < 0.001$, $\eta^2 = 0.442$] and at 80 dB in quiet [$t(34) = 3.717$, $p = 0.001$, $\eta^2 = 0.289$] but not at 80 dB in noise [$t(33) = 1.582$, $p = 0.123$, $\eta^2 = 0.070$].

TABLE 4. Aided and unaided means and standard deviations for PLF (μV), RMS (μV), and latency (ms) are displayed for three presentation conditions (65 dB SPL, 80 dB SPL in quiet, and 80 dB SPL in noise)

dB SPL		PLF (μV), Mean (SD)					Amplitude (μV), Mean (SD)			Latency (ms), Mean (SD)		
		Transition		Steady State		*F* Statistic	Transition	Steady State	*F* Statistic			*F* statistic
		100 Hz	200 Hz	100 Hz	200 Hz							
65 dB	Unaided	0.041 (0.018)	0.027 (0.009)	0.046 (0.022)	0.025 (0.005)		0.076 (0.027)	0.074 (0.025)		32.05 (0.90)		
	Aided	0.061 (0.039)	0.037 (0.022)	0.058 (0.038)	0.031 (0.016)		0.086 (0.031)	0.076 (0.027)		31.03 (0.74)		
				Aided		8.043*		Aided	4.343		Aided	12.91*
				Region × Aided		4.985		Region × Aided	7.407†			
				Region × Harmonic		1.886						
80 dB	Unaided	0.061 (0.027)	0.040 (0.017)	0.058 (0.024)	0.037 (0.014)		0.096 (0.029)	0.085 (0.026)		31.39 (0.68)		
	Aided	0.077 (0.065)	0.047 (0.033)	0.067 (0.077)	0.034 (0.018)		0.131 (0.141)	0.085 (0.033)		30.77 (0.84)		
				Aided		1.391		Aided	1.698		Aided	13.819*
				Level		12.510*		Level	8.342*		Level	13.098*
				Aided × Level		0.622		Aided × Level	0.713		Aided × Level	2.459
				Region × Level		2.673		Region × Level	5.040			
Noise	Unaided	0.065 (0.029)	0.038 (0.016)	0.068 (0.044)	0.037 (0.016)		0.098 (0.034)	0.092 (0.033)		31.34 (0.60)		
	Aided	0.078 (0.058)	0.038 (0.026)	0.068 (0.048)	0.029 (0.010)		0.121 (0.101)	0.088 (0.031)		31.06 (0.94)		
				Aided		0.115		Aided	1.212		Aided	2.502
				Noise		0.014		Noise	0.000		Noise	0.507

In addition, the F statistic is provided for main effects of amplification, level, and noise, and Region × Aided, Aided × Level, and Region × Level interactions.
**p < 0.004; †p < 0.02 (corrected α levels using the FDR procedure).*
FDR, false discovery rate; PLF, phase locking factor; RMS, root mean square.

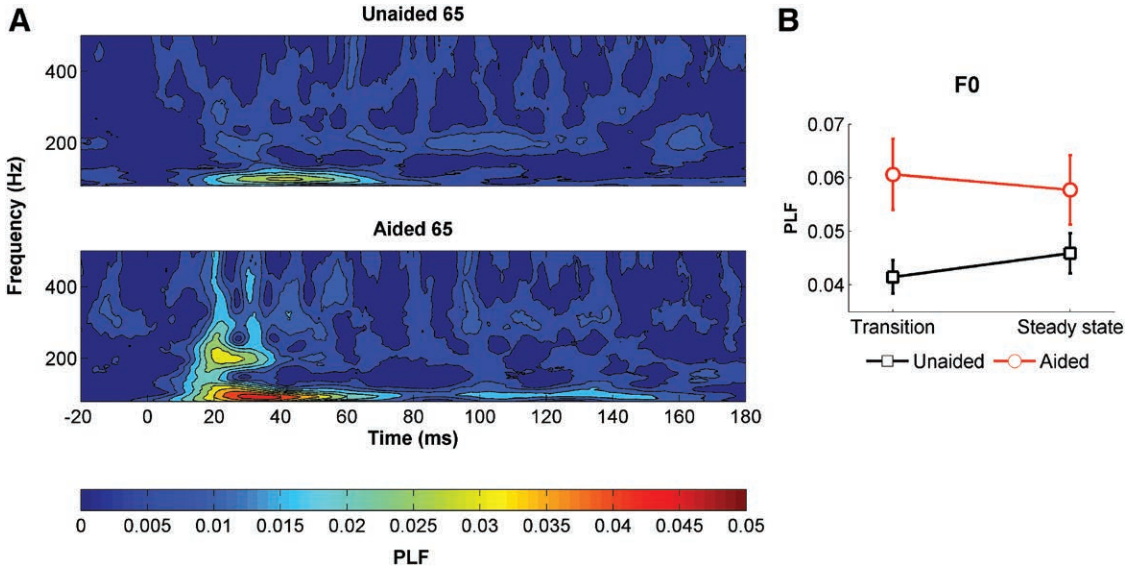

Fig. 3. Amplification increased phase locking to the speech syllable /ga/ at 65 dB SPL. A, Phase locking factor (PLF) in the time–frequency domain for group average unaided and aided responses. B, Unaided (black) and aided (red) PLF at the F_0. We note that phase cancellation occurred as a result of averaging across subjects, so that the color intensity is less than that shown in the means displayed in the line graphs. The scale of the color map in A is reduced compared to the line graphs to enhance the color contrasts in the PLF. Error bars = 1 SE.

Level Effects • Latencies were earlier at 80 dB SPL in quiet than at 65 dB SPL [$F(1,34) = 13.098$, $p = 0.001$, $\eta^2 = 0.278$], and the Latency × Aided interaction was not significant [$F(1,34) = 2.459$ $p = 0.126$, $\eta^2 = 0.067$].

Noise Effects • Noise had no effect on response latency [$F(1,33) = 0.507$, $p = 0.481$, $\eta^2 = 0.015$].

Cortical

Table 5 provides means and standard deviations of amplitude and latency values and F statistics for main effects and interactions for the different presentation conditions. The specific details are as follows:

Amplitude

Amplification Effects • In response to the 65 dB SPL in quiet presentation level, there was no main effect of amplification [$F(1,34) = 0.782$, $p = 0.383$, $\eta^2 = 0.022$]. At 80 dB SPL in quiet, there was a main effect of amplification [$F(1,34) = 4.767$, $p = 0.007$, $\eta^2 = 0.309$] and a significant Aided × Peak interaction [$F(2,33) = 7.305$, $p = 0.002$, $\eta^2 = 0.027$], driven by an increase in amplitude for P1, a decrease in amplitude for N1, and no change in P2 [P1: $t(34) = 2.952$, $p = 0.006$, $\eta^2 = 0.204$; N2: $t(34) = 2.794$, $p = 0.008$, $\eta^2 = 0.187$; P2: $t(34) = 0.423$, $p = 0.675$, $\eta^2 = 0.027$]. In the noise condition, there was no main effect of amplification [$F(1,34) = 0.109$, $p = 0.743$, $\eta^2 = 0.003$].

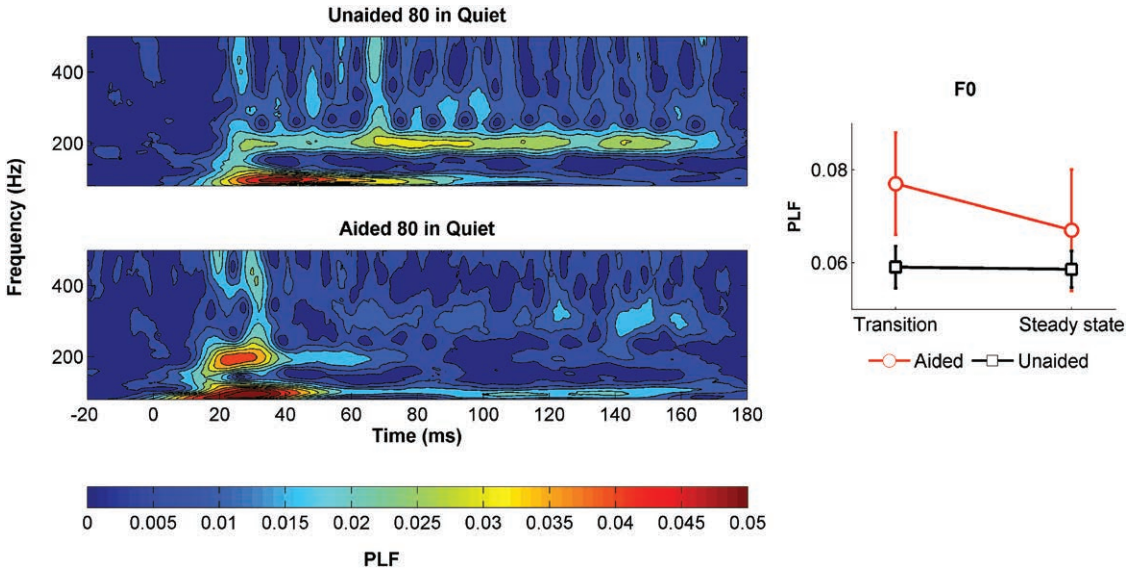

Fig. 4. There were no effects of amplification on phase locking at 80 dB SPL in quiet. A, Phase locking factor (PLF) in the time–frequency domain for group average unaided and aided responses. B, Unaided (black) and aided (red) PLF at the F_0. We note that phase cancellation occurred as a result of averaging across subjects, so that the color intensity is less than that shown in the means displayed in the line graphs. The scale of the color map in A is reduced compared to the line graphs to enhance the color contrasts in the PLF. Error bars = 1 SE.

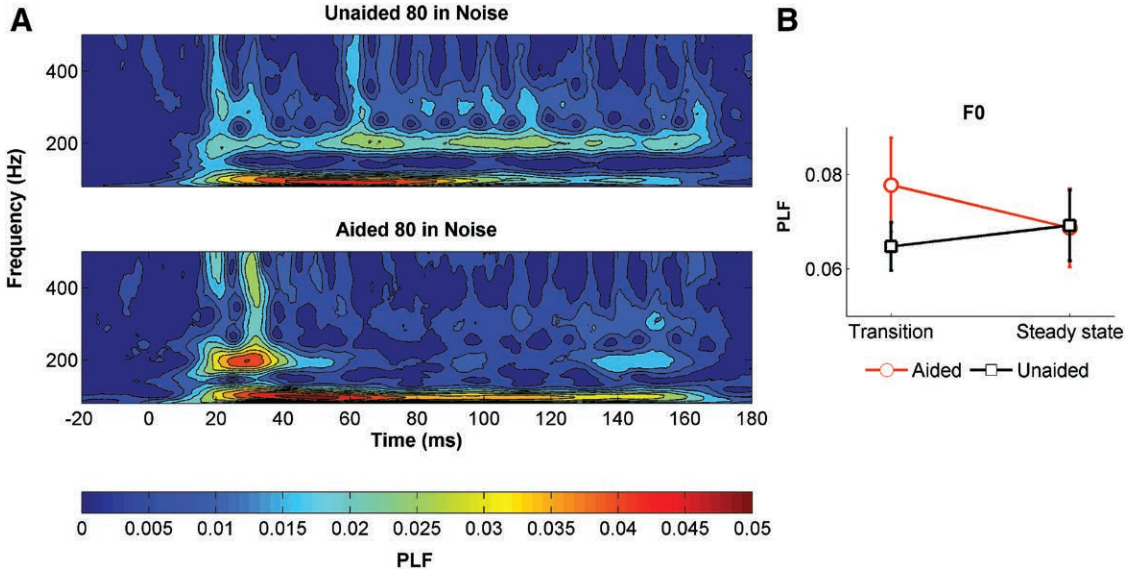

Fig. 5. There were no effects of amplification on phase locking at 80 dB SPL in noise. A, Phase locking factor (PLF) in the time–frequency domain for group average unaided and aided responses. B, Unaided (black) and aided (red) PLF at the F_0. We note that phase cancellation occurred as a result of averaging across subjects, so that the color intensity is less than that shown in the means displayed in the line graphs. The scale of the color map in A is reduced compared to the line graphs to enhance the color contrasts in the PLF. Error bars = 1 SE.

Figure 6 displays unaided and aided time domain waveforms and bar graphs representing amplitude levels at different presentation conditions.

Level Effects • There was no main effect of level [$F(1,34)=0.369$, $p = 0.547$, $\eta^2 = 0.011$], but there was a Level × Peak interaction [$F(1,34) = 9.148$, $p = 0.001$, $\eta^2 = 0.357$]. P1 amplitude increased significantly with presentation level [$F(1,34) = 6.137$, $p = 0.018$, $\eta^2 = 0.153$], but in contrast, N1 amplitude decreased significantly with presentation level [$F(1,34) = 16.102$, $p < 0.001$, $\eta^2 = 0.321$]. *P2.* There was no main effect of level for P2 [$F(1,34) = 0.263$, $p = 0.611$, $\eta^2 = 0.008$]. The Aided × Level interaction was not significant [$F(1,34) = 0.245$, $p = 0.624$, $\eta^2 = 0.007$].

Noise Effects • There was a main effect of noise [$F(1,34) = 57.469$, $p < 0.001$, $\eta^2 = 0.628$] but no Peak × Noise

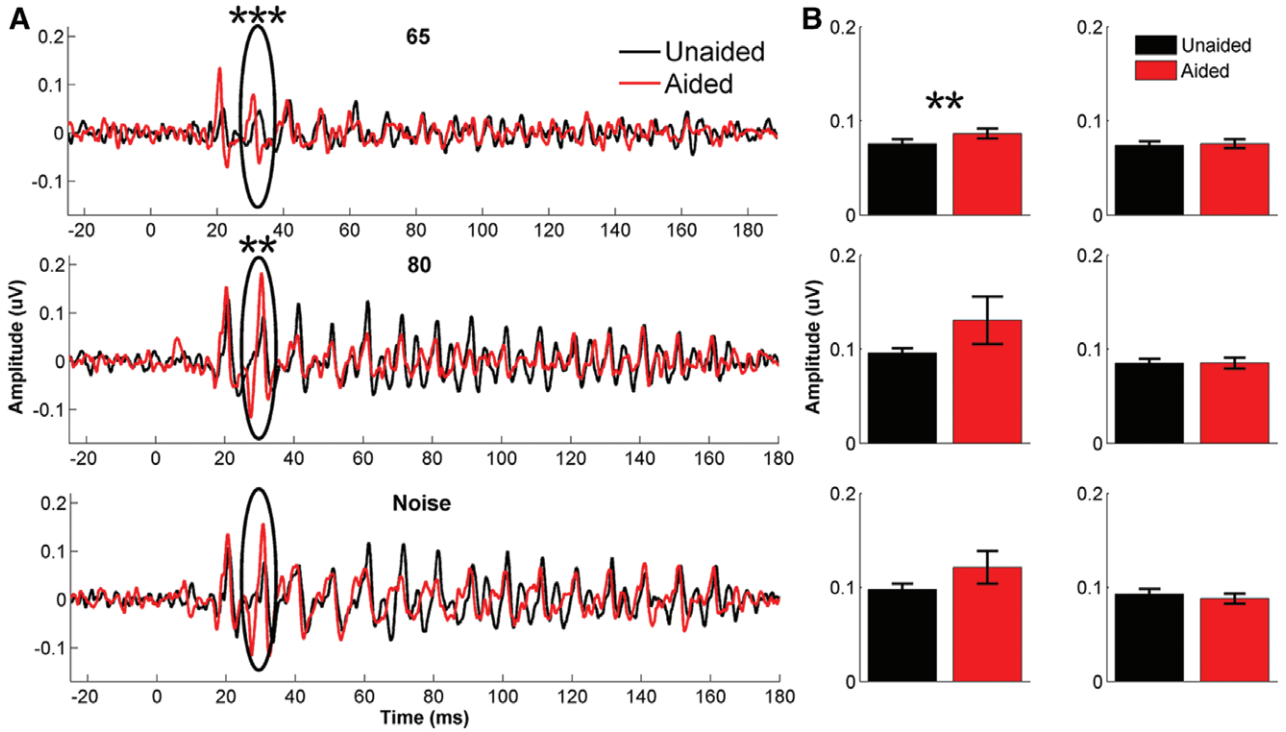

Fig. 6. Amplification increased response amplitude in the consonant transition region at 65 dB SPL, but not at the other presentation conditions. Latency decreased for 65 and 85 dB SPL in quiet but not for 85 dB SPL in noise. A, Time domain waveforms for unaided (black) and aided (red) responses. The asterisks indicate significant latency decreases. B, Bar graphs demonstrating root mean square (RMS) increases in the consonant transition region at 65 dB SPL (top) but no changes for other presentation levels or for the steady state vowel. The asterisks indicate amplitude changes. $^{**}p < 0.01$, $^{***}p < 0.001$. Error bars = 1 SE.

TABLE 5. Aided and unaided means and standard deviations for cortical DSS amplitude (μV), and latency (ms) values are displayed for three presentation conditions (65 dB SPL, 80 dB SPL in quiet, and 80 dB SPL in noise)

		Amplitude (μV), Mean (SD)				Latency (ms), Mean (SD)			
dB SPL		P1	N1	P2	*F* statistic	P1	N1	P2	*F* statistic
Amplification effects									
65	Unaided	3.08 (1.39)	3.42 (1.10)	3.09 (0.59)		59.41 (6.58)	107.79 (13.82)	213.83 (19.98)	
	Aided	3.09 (1.29)	3.36 (1.14)	2.90 (0.68)		55.51 (6.56)	108.21(12.72)	210.59 (12.70)	
		Aided			0.782		Aided		8.994*
		Peak × Aided					Peak × Aided		2.519
80	Unaided	3.13 (1.40)	3.16 (1.06)	2.94 (0.70)		56.22 (9.18)	99.33 (13.43)	211.89 (12.05)	
	Aided	3.63 (1.43)	2.80 (1.01)	2.90 (0.64)		49.66 (8.13)	100.84 (16.17)	217.48 (15.94)	
		Aided			4.767†		Aided		9.332*
		Peak × Aided			7.305*		Peak × Aided		8.338*
Noise	Unaided	2.59 (1.12)	3.01 (0.93)	1.68 (0.85)		56.28 (10.89)	150.02(30.68)	251.27 (18.37)	
	Aided	2.41 (1.10)	3.08 (1.02)	1.72 (0.87)		55.11 (8.56)	156.66(25.06)	252.78 (16.95)	
		Aided			0.109		Aided		0.573
Level effects									
F statistic		6.137†	16.102*	0.263		15.303*	14.792*	2.523	
Noise effects									
F statistic		13.193*	0.292	91.067†		1.609	131.171*	79.130*	

In addition, the F statistic is provided for main effects of amplification, level, and noise, and Peak × Aided interactions.
**p < 0.005, †p < 0.03 (corrected α levels using the FDR procedure).*
FDR, false discovery rate; PLF, phase locking factor.

interaction [$F(1,34) = 2.905$, $p = 0.069$, $\eta^2 = 0.150$] or Aided × Noise interaction [$F(1,34) = 0.491$, $p = 0.488$, $\eta^2 = 0.014$].

Latency

Amplification Effects • In response to the 65 dB SPL presentation level, amplification resulted in earlier latencies [$F(1,34) = 8.994$, $p < 0.001$, $\eta^2 = 0.428$], and there was no Aided × Peak interaction [$F(1,34) = 2.519$, $p = 0.096$, $\eta^2 = 0.132$]. There was a main effect of amplification at 80 dB SPL in quiet [$F(1,34) = 9.323$, $p < 0.001$, $\eta^2 = 0.466$], and there was also a significant Aided × Peak interaction [$F(1,34) = 8.338$, $p = 0.001$, $\eta^2 = 0.336$], driven by an amplification-related latency decrease for P1 that was not present for N1 or P2 [P1: $t(34) = 5.029$, $p < 0.001$, $\eta^2 = 0.427$; N1: $t(34) = 0.612$, $p = 0.545$, $\eta^2 = 0.011$; P2: $t(34) = 2.034$, $p = 0.095$, $\eta^2 = 0.080$]. Amplification did not affect peak latencies in noise [$F(1,34) = 0.573$, $p = 0.637$, $\eta^2 = 0.051$].

Level Effects • There was no main effect of level [$F(1,34) = 1.304$, $p = 0.261$, $\eta^2 = 0.037$], but there was a Level × Peak interaction [$F(1,34) = 8.880$, $p = 0.001$, $\eta^2 = 0.350$]. There was a significant latency decrease in the 80 dB SPL in quiet versus the 65 dB SPL conditions for P1 [$F(1,34) = 15.303$, $p < 0.001$, $\eta^2 = 0.310$] and N1 [$F(1,34) = 14.792$, $p = 0.001$, $\eta^2 = 0.303$] but not for P2 [$F(1,34) = 1.063$, $p = 0.310$, $\eta^2 = 0.030$]. The Aided × Level interaction was not significant [$F(1,34) = 2.236$, $p = 0.144$, $\eta^2 = 0.062$].

Noise Effects • There was a main effect of noise on peak latencies [$F(1,34) = 254.001$, $p < 0.001$, $\eta^2 = 0.882$] and a significant Noise × Peak interaction [$F(1,34) = 63.864$, $p < 0.001$, $\eta^2 = 0.795$]. Noise had no effect on the latency of the P1 peak [$F(1,34) = 1.609$, $p = 0.213$, $\eta^2 = 0.045$] but significantly delayed the latency of N1 [$F(1,34) = 131.171$, $p < 0.001$, $\eta^2 = 0.795$] and P2 [$F(1,34) = 79.130$, $p < 0.001$, $\eta^2 = 0.699$]. There was no Noise × Aided interaction [$F(1,34) = 0.754$, $p = 0.391$, $\eta^2 = 0.022$].

DISCUSSION

This study investigated hearing aid amplification effects on FFRs and CAEPs to a speech syllable in first-time hearing aid users at levels which approximated typical listening conditions. Overall results suggest that amplification may improve subcortical representation of the speech syllable /ga/. More notably, the findings throughout the study suggest that this improvement may, in part, be because of increased audibility. While previous studies found minimal amplification effects on CAEPs in individuals with normal hearing (Billings et al. 2007, 2011), the current investigation found differences in CAEP responses between aided and unaided conditions, consistent with Van Dun et al. (2016). These results suggest the importance of using participants with sensorineural hearing loss when investigating the efficacy of incorporating evoked potentials in the hearing aid fitting.

Frequency-Following Response

Phase Locking Factor and RMS Amplitude • Amplification effects on the FFR were similar for phase locking factor and RMS amplitude. More consistent phase locking and increased amplitudes to the speech syllable were observed between aided and unaided responses, but only for the 65 dB SPL presentation level and not for the 80 dB SPL level in quiet or in noise (Figs. 3–5). Furthermore, the increase in RMS amplitude was

stronger in the transition region than in the steady state region (Fig. 6). The relatively higher frequency transition region has lower RMS power compared to the steady-state region (Fig. 2).

Level effects were also observed in the form of improved phase locking and increased RMS amplitudes at higher presentation levels for both the transition and steady-state regions. The increases in phase locking and amplitude from 65 to 80 dB SPL in quiet suggest, in part, improved midbrain processing because of increased audibility of the signal. We found no significant Aided × Level interactions for either RMS or PLF, suggesting that level effects were similar for unaided and aided conditions. These results are similar to the level effects found by Easwar et al. (2015a), who noted that increasing the test level from 50 to 65 dB SPL resulted in an increase in response amplitudes. In their follow-up study, Easwar et al. (2015b) found a Level × Aided interaction for response amplitude. In that study, the differences between aided and unaided responses to the vowel /a/ first formant were greater for the 50 dB SPL condition than for the 65 dB SPL condition; although amplification increased response amplitude, there was a main effect of amplification at both levels. In our study, we presented stimuli at 65 and 80 dB SPL, and the amplification effects may have been more pronounced if we included lower level stimuli of less 65 dB SPL.

Latency • Increases in audibility may also explain the reductions seen in FFR latencies (Fig. 6). Decreased latencies were seen in aided compared to unaided responses for both 65 and 80 dB SPL in quiet presentation levels. We used a 2 ms correction for latency to account for hearing aid processing time, corresponding to the delay noted for the low-frequency components of the signal (frequency-specific delays provided by Widex USA). If the latency reduction was attributable to this correction, we would have expected a more uniform latency decrease across conditions. However, we found that the latency decrease was greatest for 65 dB SPL in quiet, was smaller for 80 dB SPL in quiet, and was not significant for 80 dB SPL in noise. The latency results may provide support for the idea that improved midbrain processing is the result of increased audibility. However, differences in latency changes between listening conditions may also be influenced by smaller changes in sensation level at the 80 dB SPL presentation level because of the compression in the hearing aids or to increased audibility of the stimulus at 80 dB SPL in the unaided condition.

Noise Effects • A number of factors may account for the lack of significant noise effects on any of the FFR variables. In this study, we used a relatively favorable SNR of +10 dB, which may not have resulted in sufficient degradation of neural synchrony to affect midbrain processing (Easwar et al. 2015b). Li and Jeng (2011) evaluated the effects of noise on FFRs to lexical tones in young adults and found that midbrain processing was relatively unaffected at SNRs of 6 and 12 dB but the F_0 amplitude and other measures of neural fidelity were significantly decreased at SNRs of −6 and −12 dB. These results suggest the need to use more unfavorable SNRs when evaluating effects of noise on midbrain processing.

Minimal effects of noise might also be attributed to hearing loss. Noise-related reductions of amplitude and latency shifts in the auditory brainstem response have been demonstrated in young adults (Hecox et al. 1989; Burkard & Sims 2002), but these effects are not as pronounced in older adults with hearing loss. Even older adults with normal hearing have reduced effects of noise compared to younger adults. In a comparison of FFRs to a speech syllable presented in one-talker babble at SNRs varying from −6 to +3 dB, Presacco et al. (2016b) found that noise minimally affected the response amplitudes of older adults with normal hearing compared to younger adults with normal hearing. These differences may arise from age-related cochlear synaptopathy. A study investigating the feasibility of assessing Wave V clicks in noise as a measure of cochlear synaptopathy in humans found that Wave V latency shifts in noise mirrored changes in Wave I amplitude (Mehraei et al. 2016). The study also found that greater Wave V latency shifts correlated with better performance on a temporal processing task (discrimination of interaural time differences). Taken together, these results suggest that larger Wave V shifts in noise are an indication of healthier auditory nerve function. Therefore, the lack of effects of noise on any of the FFR measures in this study may be because of reduced auditory nerve function associated with hearing loss.

Cortical Response

Effects of Amplification • The results of our cortical analyses only partially supported our hypothesis. Changes in the P1 peak between aided and unaided responses suggest effects of increased audibility associated with amplification, specifically increased P1 amplitude and decreased P1 latency. The P1 component likely reflects a nonspecific sensory response to an acoustic stimulus (Shtyrov et al. 1998; Sharma et al. 2002; Ceponiene et al. 2005); therefore, increased amplitude/decreased latency with amplification may indicate increased detection (Fig. 7). These results are consistent with those of Billings et al. (2012) who found that CAEPs may reflect physiological detection of hearing aid–processed signals.

The results for the N1 peak did not support our hypothesis of larger amplitudes with amplification. N1 amplitudes were smaller for aided than unaided responses at 80 dB SPL in quiet and decreased with louder presentation levels. These results contrast with those of Van Dun et al. (2016), who found that the N1 amplitude increased with amplification. Our study differed from that of Van Dun et al. in that we assessed overall cortical activity, and the Van Dun study focused analyses on the Cz electrode only. We only found the amplitude decrease when we analyzed overall electrode activity using the DSS analysis but did not find it when we compared amplitudes for the Cz electrode only. The results in the unaided condition may reflect aging and hearing loss effects that lead to exaggerated amplitudes of the N1 component because of inefficient resource allocation. Using CAEPs, Billings et al. (2015) found that the older group with hearing loss had larger N1 amplitudes than either the younger or older groups with normal hearing, while this effect was not seen for other components. Using magnetoencephalography, several studies have found over-representation of the N1 and P2 components in older adults, both with normal hearing and with hearing loss, compared to young adults with normal hearing (Sörös et al. 2009; Alain et al. 2014; Presacco et al. 2016a, b). Imaging studies have suggested that cortical network connectivity is reduced in older adults, resulting in redundant processing of the same stimulus by neighboring cortical areas (Peelle et al. 2010). This redundant processing may be a contributing factor to over-representation of the N1 component. A reduction in N1 amplitude may, therefore, indicate that amplification results in a decrease in redundant processing.

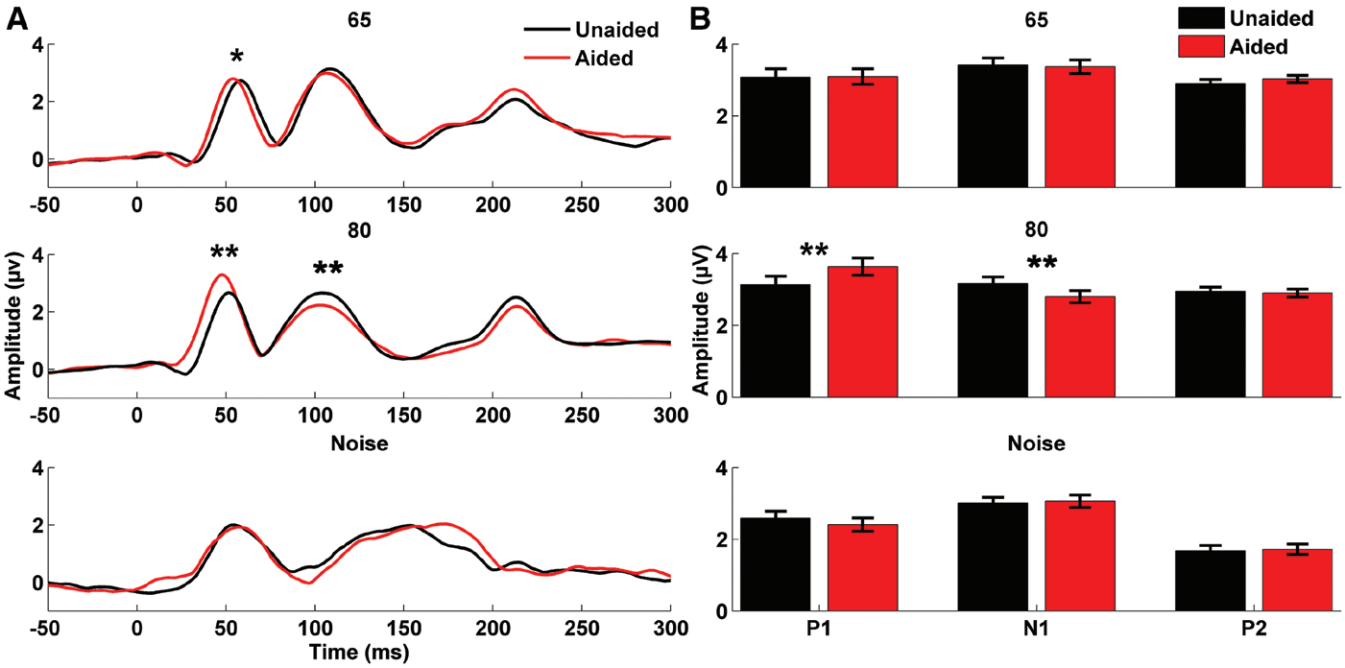

Fig. 7. Different effects of amplification were noted for different cortical components. A, denoising source separation (DSS) rectified waveforms for unaided (black) and aided (red) responses at three presentation levels with the P1, N1, and P2 peaks. The asterisks indicate significant latency decreases. B, Amplitude of the cortical peaks for unaided (black) and aided (red) response for three presentation levels. A significant increase in P1 amplitude and a significant decrease in N1 amplitude from unaided to aided responses were noted in the 80 dB SPL condition. The asterisks indicate significant amplitude changes. *$p < 0.05$, **$p < 0.01$, Error bars = 1 SE.

Effects of Level • Increasing the presentation level resulted in increased P1 amplitudes and decreased N1 amplitudes and decreased P1 and N1 latencies for both aided and unaided conditions. These results are consistent with those of Billings et al. (2015), who found effects of level in older participants with hearing loss but not in the normal hearing younger or older groups. Previous studies revealed minimal effects of level on the CAEPs of individuals with normal hearing (Billings et al. 2009, 2013), but the fact that increased level results in changes in individuals with hearing loss suggests that audibility is a key factor in these changes. The direction of the amplitude change for P1 and N1 is consistent with the interpretation of amplification effects. Larger P1 amplitudes suggest that increased level leads to increased detectability for this sensory component. On the other hand, the N1 component, which is believed to reflect early triggering of attention to auditory signals (Näätänen 1990; Ceponiene et al. 2002), decreases in amplitude with level, suggesting that less neural activity is required to trigger attention to the signal at louder input levels. The P2 component was unaffected by level. This component may represent a later stage of auditory processing than signal detection, possibly auditory object formation (Ross et al. 2013), and may be minimally affected by level.

Effects of Noise • In contrast to the FFR, significant effects of noise were noted for aided and unaided CAEPs (Fig. 7). Significant increases in latency were noted for the N1 and P2 components, and the P2 component had a significant reduction in amplitude, consistent with the findings of previous studies (Sharma et al. 2014; Billings et al. 2015; Kuruvilla-Mathew et al. 2015). The differences in noise effects between midbrain and cortex in individuals with hearing loss may arise from how these signals are processed. Precise synchrony is required to accurately represent signals in the brainstem and midbrain, even when those signals are presented in quiet (Kraus et al. 2000), but cortical responses may be present in cases of complete desynchronization of the auditory nerve or lower levels of the auditory system (Kraus et al. 2000; Chambers et al. 2016). Nevertheless, cortical responses are more vulnerable to the effects of noise in individuals with auditory neuropathy compared to individuals with normal hearing (Michalewski et al. 2009). In our study, the FFRs of the participants (who all had hearing loss) may be affected by desynchronization to some degree, and the addition of noise (at least at a favorable SNR) did not significantly affect an already degraded system, yet their cortical responses remain vulnerable to noise effects. As noted by Sharma et al. (2014), however, it is important to include multiple stimulation levels and SNRs to make any reasoned interpretations about noise effects on cortical processing.

Limitations

Speech stimuli were presented in sound field to simulate an ecologically valid listening situation. When using direct audio input, as was done in the Easwar et al. (2015b) study, the hearing aid microphone is bypassed. However, the use of sound field presentation introduces the possibility of jitter contaminating the response through slight movements on the part of the participants. The participants were encouraged to remain still while watching a subtitled movie, and their movements were monitored through observation of electrical activity and through a webcam. However, slight movements may have reduced the temporal precision of the responses. Nevertheless, this possibility likely does not change interpretation of the results as robust responses above the noise floor were obtained in all of the participants (SNR decibel values >1).

Another aspect of the method that may limit the interpretation of the findings is the use of open-fit hearing aids. The effect of this fitting is that the unaided speech signal enters the ear canal through the open dome, along with an aided signal that has been processed through the hearing aid circuitry. The aided signal has a processing delay that varies depending on the frequency content, and this delay results in a degree of temporal smearing that will also affect the recording. This temporal smearing may minimize amplification effects on evoked potentials. Nevertheless, the resulting somewhat distorted signal represents what the hearing aid listener becomes accustomed to during every day listening.

Participants were part of a larger plasticity study; therefore, to mimic the typical clinic fitting, we used traditional real-ear measures to verify that the hearing aids appropriately met NAL-N2 targets. However, controlled comparisons of aided and unaided responses using in situ measures during EEG testing in each individual participant would have allowed us to make more definitive interpretations of our data.

Although the hearing aids are designed to amplify stimuli from 100 to 7300 Hz, there was a significant roll-off of hearing aid output above 2000 Hz. When gain was increased to match the higher frequency targets, the participants experienced feedback or discomfort because of the level of the high-frequency sounds. As indicated by Tables 1 and 2, adequate audibility above 2000 Hz was not achieved.

The listeners had never worn hearing aids before participating in this experiment; therefore, these results may not apply to individuals who are experienced hearing aid users. As mentioned previously, work is underway to determine if and when these effects change with hearing aid use over time. Twelve weeks of hearing aid use did not result in changes in the click-evoked auditory brainstem response (Dawes et al. 2013), but it may be possible to see neuroplasticity using a more complex stimulus, as has been demonstrated in auditory training studies (Anderson et al. 2013a, b).

CONCLUSIONS

As hearing aid technology advances, so too must the assessment of the hearing aid fitting. As speech perception is influenced by both peripheral and central components of the auditory system, it is important that verification of hearing aid benefit be confirmed beyond the tympanic membrane at higher levels of auditory processing. The present study demonstrates that it is feasible to collect aided sound field responses to ecologically valid signals in listeners with sensorineural hearing loss and to identify key differences between aided and unaided responses that may indicate increased audibility with amplification. This information contributes to the understanding of the central effects of amplification and may lead to the development of improved verification measures and hearing aid algorithms to improve speech intelligibility for listeners with sensorineural hearing loss. However, future studies should consider study design and stimuli parameters that might provide better control of amplification contrasts, such as the use of direct audio input or wireless transmission, real-ear measures during EEG testing, and a greater range of stimulus level and frequency differences. Furthermore, substantial intersubject variability was noted between subjects, possibly related to different degrees or etiologies of hearing loss, and it would be important to identify sources of individual variability in responses. Hearing aid manufacturers use different strategies to maximize speech clarity, and evoked potentials may provide a useful tool for evaluating the effects of these strategies on neural speech processing, taking into consideration the likelihood of large variability in this population.

In this study, clear differences were noted on both FFRs and CAEPs between aided and unaided responses, at least at suprathreshold levels. The comparison of aided and unaided FFRs and CAEPs may provide information, in part, regarding audibility of the speech signal at central levels of the auditory system. This study also verifies the importance of testing participants with hearing loss when determining amplification effects on central processing. Therefore, it provides important first steps into determining how auditory-evoked responses can be utilized to enhance hearing aid outcomes, as well as improve methods of investigation into the possible uses of auditory-evoked potentials in future aural rehabilitation treatment plans.

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S.A. designed the experiment; K.J., C.F., A.P., and S.A. collected and analyzed the data; and K.J., C.F., A.P., and S.A. wrote the article.

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